

David A. Schulsinger
Editor

Kidney Stone Disease

Say NO to Stones!

 Springer

Kidney Stone Disease

David A. Schulsinger
Editor

Kidney Stone Disease

Say NO to Stones!

 Springer

Editor

David A. Schulsinger, MD
Stony Brook School of Medicine
Stony Brook, NY
USA

ISBN 978-3-319-12104-8 ISBN 978-3-319-12105-5 (eBook)
DOI 10.1007/978-3-319-12105-5
Springer Cham Heidelberg New York Dordrecht London

Library of Congress Control Number: 2014958180

© Springer International Publishing Switzerland 2015

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

Foreword I

About one person in ten will have a urological stone over their lifetime. Also about 60–70 % of those individuals will have recurrent stones! This well written, clearly articulated and comprehensive book about urolithiasis, urological stones, by an expert in the field, David Schulsinger, and his co-authors discusses all aspects of stone disease, directed to patients with this condition and their families.

In my 40 years in the practice of urology there is no area that has changed as much as the management of patients with stone disease. When I began practice, most kidney and ureteral stones were removed by open, invasive, surgical procedures requiring incisions through several layers of muscles, often with the removal of a rib and incisions into the kidney or ureter. Patients could easily have spent days in the hospital while recovering from these surgeries. In addition, often we could not remove all the stones requiring additional treatment! The first change was when we discovered we could insert small tubes into the kidney, enlarge the tract and look directly at the stone and destroy it with newly invented forms of energy, a procedure called percutaneous lithotripsy. Then small tubes, called ureteroscopes, were developed so we could look into the ureter and kidney and again fragment and remove ureteral stones. Before these advances, we would blindly insert a small tube into the ureter, open a basket, and move the basket up and down “trolling” for the stone, when we felt the resistance we would close the basket, pull down the tube with our fingers crossed and hope to remove the stone! The third advance came from “thinking out of the box” and Dr. Christian Chaussy in Munich discovered that focused shock waves on a stone without an incision could fragment stones to small pieces, which would pass. Extracorporeal shock wave lithotripsy was born. As a consequence, open stone surgery is rarely needed today and “blind” basketry is only of historical interest, all to the benefit of the patient. At the same time we learned more about stone prevention, developed new medical drugs to prevent the recurrence of stones and finally developed drug regimens for enhancing the spontaneous passage of small ureteral stones, known as medical expulsive treatment.

This book illustrates all aspects of stone disease from inherited factors and personal habits, especially dietary factors, including medical treatments for prevention of both stone formation and stone recurrences. The relevant anatomy is clearly shown, and the different types of stones described and complimented by excellent illustrations. The book continues with clear descriptions of the various interventional forms of treatment including the pros and cons

of each. The chapters are often punctuated with relevant comments from patients who unfortunately have suffered from urological stones. Medical management and necessary life style changes are then reviewed for patients with different types of stones. Finally, in a unique chapter the authors give advice as to who should treat your stone and where. Additional changes that have taken place since I began my practice has been the development of “sub-specialists.” Thus, across the country there are experts, such as Dr. Schulsinger, who specialize in the area of stone disease. Moreover, we now know the success of various procedures with lower complications occurring in hospitals with high volume of care for a specific problem performed by “high volume” surgeons. Finally, although this book is primarily directed to patients and their families, I feel it will be an excellent reference book for primary care physicians who without a doubt will take care of patients with stone disease. In addition, urologists will also benefit from the book, a great deal of highly valuable informative information in one concise package.

New York, NY, USA

E. Darracott Vaughan Jr.

Foreword II

What would a patient want in a book on stone disease? Nothing more than an understanding of the multiple different clinical issues that arise in dealing with this clinical entity. This patient-centric compendium presents in impressive detail the nuances of stone disease whether it be medical, surgical or nutritional. In this text, Dr. Schulsinger presents a highly personal and comprehensive approach to clinical problems and solutions that arise in the management of patients with stone disease. The patient will see this unique field through the eyes of a practitioner who has lived in this world for almost two decades. A skilled endourologist, he shares a broad knowledge of dealing with the various complex situations that arise in treating patients with stone disease. Endourology is one of the numerous miracles coming out of twentieth-century medicine, having evolved over a relatively short period of time. In these days of cost containment, endourologic procedures are now preferred as they allow the patient a shorter hospital stay and more rapid return to work. These methods have minimized patient morbidity and reduced the fear of more extensive surgical procedures. The results have significantly enhanced urologic care by improving outcomes, reducing costs and enhancing patient satisfaction. I am certain any patient would find great interest and solace in this guide to treatment of stone disease.

Stony Brook, NY, USA

Wayne C. Waltzer

Preface

As a fellowship- and residency-trained urologist with a background and major in nutritional biochemistry, I had the good fortune of combining these concentrations into a profession to best aid and guide my patients. I am an endourologist with a concentration in treating urolithiasis, or urinary tract stones. In my professional career, I have surgically treated over 8,000 patients. Surgically, we have methods to treat both large and small stones. Medically and nutritionally, we have resources to treat all stone types and prophylactically prevent future stones. My motto in treating patients with kidney stones is “I leave *no stones unturned!*”

I share with you my own personal encounter with pain, diagnosis, and surgeries. The first two chapters identify a view through patient medicine and physician medicine. I look at situations through the eyes of a physician and the interpretation as a patient and how these perspectives differ from each other. It was through this journey that I became compelled to share with you and prospective patients the questions and skills needed to be an empowered patient. I provide you with the appropriate questions to ask, the methods to make the diagnosis, the understanding of different treatment options, and the means of selecting the physician who is best suited for you. I share with you the stories from my patients and a workable and sensible approach to manage, treat, and ideally prevent future stones.

I am grateful and wish to thank my patients whom I meet, treat, and learn from each and every day and can appreciate the adage “no two patients are alike,” just as no two surgeries are equivalent. For their untold patience and incredible understanding, I dedicate this book to my loving wife Kari and my endearing children Ariel and Hailey.

Stony Brook, NY, USA

David A. Schulsinger, MD

Acknowledgements

The author would like to acknowledge the following individuals:

T. Colin Campbell, my academic advisor, mentor, and my honors advisor at Cornell University who provided me with the skills and vision to combine nutrition and cancer, and the academic resources of applying research principles and how to go about answering the questions to a scientific hypothesis. I am indebted to him for allowing me to take my scientific curiosity and applying it to a research investigation and honors thesis in nutritional biochemistry.

E. Daracott Vaughan, my fellowship advisor and urology mentor who gave me the second chance to pursue my academic dreams in urology. A Doctor's Doctor who showed me the necessary and appropriate approach to challenging urologic procedures.

R. Ernest Sosa, my endourology mentor, who I am grateful for allowing me to refine my endourology skills and enforcing within me the importance of paying attention to detail. I carry those words with me both inside and outside of the operating room. Thank you for showing me the epitome of superb bedside manners, validating for me that surgical care and bedside manners do co-exist!

Wayne C. Waltzer, my chairman, who I am grateful and very appreciative for creating an academic environment to pursue my urologic endeavors.

Philip Li my best friend and colleague who has inspired me to be a "starter" and "finisher" and maintains a clear perspective, "life is beautiful."

Marc Goldstein a friend, colleague, and running partner who has been there in times of need and has provided the insight and understanding of a world class urologist.

Andrew Schneider, a friend, marathon running partner, and colleague since medical school who I am grateful for always providing the moral clarity and understanding of life.

Hong Kim, my residency chairman, who I am grateful for his incredible patience and who has taught me the critical fundamentals and basic urological principles to be a good clinical urologist, which I will carry throughout my professional career.

Urology staff who I work with as a team each and every day, providing the best care for our patients. I am greatly appreciative for their daily commitment to patient care and managing patient's expectations.

My parents and sister who provided the nurturing, love, resources and support for me to pursue my dreams; your advice of "doing the things that make you happy" has greater meaning everyday.

My patients who make each day new and exciting and each surgical case unique and adventurous. I am grateful for your questions, which manage to keep me on my toes and to stay one step ahead. Thank you for bringing interest, new excitement, and keeping my job interesting everyday.

Contents

Part I My Personal Encounter with the Medical Profession, Surgery and PAIN!

- 1 Room #2: *The Test I Could Not Study For!* 3**
David A. Schulsinger
- 2 Visiting the Other Side: *The Roller Coaster Ride Continues!* 11**
David A. Schulsinger

Part II The Significance of Stone Disease

- 3 The Urinary Tract: *The Inside Story!* 19**
David A. Schulsinger
- 4 The Rock of Ages: *Stones Have Stood the Test of Time!* 27**
Abe D'Amato, Yefim Sheynkin, and David A. Schulsinger
- 5 Facts and Figures: *Stones by the Numbers!* 31**
Scott Herfel and David A. Schulsinger
- 6 Urinary Tract Stones: *From the Invisible to the Clearly Distinct and Discernible Stone!* 35**
David A. Schulsinger
- 7 Stone Characteristics: *Not All Stones Are Created Equal!* 43**
David A. Schulsinger

Part III Risk Factors, Risk Groups

- 8 Metabolic and Hereditary Factors: *Are You Stone Prone?* . . . 51**
Merrit Debartolo and David A. Schulsinger
- 9 The Stone Belt 55**
David A. Schulsinger
- 10 Sex and Stones: *Sex and Stones May Break Your Bones, but Water Will Not Harm You!* 69**
David A. Schulsinger

11	Pregnancy and Stones: <i>Stubborn Stone Situation</i>	75
	Heather N. Di Carlo and David A. Schulsinger	
12	Children and Elderly with Stones: <i>Stones for the Ages!</i>	79
	Heather N. Di Carlo and David A. Schulsinger	
13	Obesity and Stones: <i>Losing the Waist Is More Than Weight!</i>	85
	David A. Schulsinger	
Part IV The Work-Up		
14	Symptoms: <i>Listen to How Your Stone Is Communicating with You!</i>	91
	David A. Schulsinger	
15	The Incidental Stone: <i>It May Not Be So Insignificant!</i>	97
	David A. Schulsinger	
16	Stone Disease Imaging: <i>There Is More to X-Rays Than What We See!</i>	103
	Andres Pena and John A. Ferretti	
Part V Treatment		
17	Who, What, Where and When to Treat!	113
	David A. Schulsinger	
18	Meeting Your Expectations: <i>What to Anticipate Before, During and After Treatment?</i>	119
	David A. Schulsinger	
19	Medical Therapy: <i>Mind Your Medicines</i>	123
	Merrit Debartolo and David A. Schulsinger	
20	The Surgical Procedure: <i>No Doc, No Shock!</i>	129
	Jonathan J. Melquist and David A. Schulsinger	
21	Treatment of Complex Stones: <i>Location, Location, Location!</i>	143
	David A. Schulsinger	
22	Ureteral Stents: <i>To Stent or Not to Stent, That Is a Great Question!</i>	153
	David A. Schulsinger	
23	Managing Your Pre-operative and Post-operative Pain	159
	Marco Palmieri and Siddharth K. Dave	

Part VI Prevention

24 Stop the Stones!: Necessary Tests to Determine Your Risk for Stones 169
 David A. Schulsinger

25 Hydration: Why We Drink, When to Drink, What to Drink, and How Much to Drink, That Is the Question! 175
 David A. Schulsinger

26 Complimentary Medicine: A Natural and Healthy Approach to Stone Prevention 181
 Tiffany Graham

27 Nutrition Recommendations to Prevent Kidney Stones: Realistic Dietary Goals and Expectations! 187
 Kristina L. Penniston

28 Diet Fads and Stones: Is Your Diet All Cracked Up to What It Is Supposed to Be? 201
 David A. Schulsinger and Kristina L. Penniston

29 Maintain Control: Managing Your Expectations! 207
 David A. Schulsinger

Part VII Bonus

30 Update: What Is New on the Horizon? 215
 Brian Sninsky and Stephen Y. Nakada

31 Special Stone Stories 221
 David A. Schulsinger

32 Billing, Coding and Your Stone: What the Patient Should Know! 229
 David A. Schulsinger and Michael A. Ferragamo

33 ObamaCare and Your Stone: Good News, Bad News! 235
 David A. Schulsinger

Index 241

Contributors

Abe D’Amato, MD Department of Urology, Stony Brook Medicine, Stony Brook, NY, USA

Siddharth K. Dave, MD Department of Anesthesia and Pain Medicine, Stony Brook Medicine, Stony Brook, NY, USA

Merrit Debartolo, MD Department of Urology, Stony Brook Medicine, Stony Brook, NY, USA

Heather N. Di Carlo, MD Division of Pediatric Urology, Johns Hopkins School of Medicine, The James Buchanan Brady Urological Institute, Baltimore, MD, USA

Michael A. Ferragamo, MD, FACS Department of Urology, Stony Brook Medicine, Stony Brook, NY, USA

John A. Ferretti, MD Department of Radiology, Stony Brook Medicine, Stony Brook, NY, USA

Tiffany Graham, MPH, RD, LD Theralogix, Rockville, MD, USA

Scott Herfel, PA Department of Urology, Stony Brook Medicine, Stony Brook, NY, USA

Jonathan J. Melquist, MD MD Anderson Cancer Center, Houston, TX, USA

Stephen Y. Nakada, MD Department of Urology, University of Wisconsin School of Medicine and Public Health, Madison, WI, USA

Marco Palmieri, DO Department of Anesthesia and Pain Medicine, Stony Brook Medicine, Stony Brook, NY, USA

Andres Pena, MD Department of Radiology, Stony Brook Medicine, Stony Brook, NY, USA

Kristina L. Penniston, PhD, RD Department of Clinical Nutrition Services, University of Wisconsin Hospital and Clinics, Madison, WI, USA

David A. Schulsinger, MD Department of Urology, Stony Brook
Medicine, Stony Brook, NY, USA

Yefim Sheynkin, MD Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA

Brian Sninsky, MD Department of Urology, University of Wisconsin
School of Medicine and Public Health, Madison, WI, USA

Part I

**My Personal Encounter with the Medical
Profession, Surgery and PAIN!**

Room #2: The Test I Could Not Study For!

1

David A. Schulsinger

My pain was in the upper right hand quadrant and it was quite significant. The insight I gained from my days of preparation for clinical medicine, a second year medical school class, and my clinical training as a general surgery resident, allowed me to consider the differential diagnosis for this pain. The thought of the pain being caused by a renal stone was not out of the question; it was in the differential for the cause of this throbbing discomfort I sustained. In that the sharp and stabbing sensation appeared shortly after eating, other possibilities were to be considered, including cholecystitis (inflammation of the gallbladder from a stone), a gastric or duodenal ulcer, or even appendicitis. The pain exacerbated after eating food supported this potential diagnosis. In my differential diagnosis, was it abdominal discomfort from a pulled muscle following a long run as I had been training for my 13th New York City Marathon? At the time, I was also one week away from an important examination, so the possibility that I was having some gastritis was a possible explanation. I chose to stop eating food that day hoping the gallbladder pain, my initial diagnosis, would resolve or at least improve. Unfortunately, the pain intensity only got worse. My family and I journeyed out to a Bar Mitzvah that day, as I did not want to spoil the festivities, but the increasing

level of agony prevented me from staying beyond the welcome reception.

Hours later, a visit from the local fire department after my wife could not get a hold of me, escalated the level of concern. As most physicians, being our own worst patient, I was able to convince the firemen visiting me in my bedroom that I was just fine and they did not need to continue watching Saturday college football with me any longer.

“A happy wife is a happy life” had taken on a whole new level of concern. As if the fire department visit was not enough, she pulled out all the stops. “It’s not just for me, it’s for our children.” I realized that it was not only just a good decision for me to go to the ER to verify that I was well, but also that it would be healthy for future of our marriage! To amplify the situation, these events were occurring 1 week before my Urology recertification examination. This is an exam Urologists take every 10 years to recertify that their knowledge and practice of Urology is updated to current standards.

Three feet from where I stood, down the hall, was ER Room #2. This was the nebulous room in the ER that seemed to always have the door closed. Over the course of my tenure at this hospital, I have never known who was on the other side, what their diagnosis was or what they were being treated for. Now the site of this closed door represented a whole other world of personal possibilities and threats. Once or twice, I had a patient of mine there who was being treated for a

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

kidney stone. Now I was the patient in this room, being worked up for my inexplicable abdominal pain!

Dr. G entered the room. He was a well-respected ER physician who was not only a colleague of mine, but his mom was also a patient who I had treated. Our initial conversation was physician to physician. I was discussing my symptoms as if I was the physician describing my patient. Only this time, I was both the physician and the patient! With my wife and dad present, he discussed the working diagnosis and the battery of tests that would be completed. With the location of my discomfort and with the onset of pain following food, I was being worked up for cholelithiasis and pancreatitis. I was going to have an ultrasound to evaluate the gallbladder and pancreas and additional blood tests to evaluate bilirubin, pancreatic enzymes, etc. It was now, 7 pm. The night was young!

Several hours later, I received the report that all tests were negative. I was pleased to get this news. While I was feeling somewhat better since I arrived to the ER, the pain was not completely resolved, but the negative test results suppressed my mental distress even more. As a patient, I was delighted to hear the good news. As a physician, I was cautious to interpret these results that the patient was still having pain and that we still needed to find the source. While certain disease states were ruled out, the true diagnosed needed to be ruled in. As a Urologist, I thought, could it be a kidney stone or even a renal mass, despite the fact there was no blood in my urine?

It was now 2 am. I was pleased that all the tests to this point were negative. I was feeling physically and psychologically a bit better. Dr. G entered the room with a poker face that was challenging to read. I lay on the ER stretcher sandwiched between my emotionally drained wife and my concerned dad. He told me that all of my numbers looked good and that all the tests were negative. "Before you go home", he stated, "let's get a CT scan of your abdomen." "I am sure it will be negative", he affirmed, "but let's verify this concern." We spoke to your Internist, Dr. L, who suggested as a colleague and fellow physician that we go over the top to rule out even the

most remote possibilities. "You have already spent most of the night here, what's another two hours?", he said. This seemed like a reasonable option. My physician cap went back on. I quickly questioned myself whether I needed this CT scan, realizing that this may not have been my medical recommendation. I recalled a recent journal article that suggested the lifetime risk of cancer following a single CT scan was 1:2,000. "Did I need this risk?", I thought. My patient cap went back on. Unless I was going to sign out against medical advice (AMA), I was going to have this test.

Throughout my life, I was always overly prepared for my examinations. I had developed a knack for excelling beyond high school pop quizzes; college prelims, midterms and finals; taking the Kaplan course for the MCAT's, urology in-service exams, and board examinations; these all conditioned me to become a tenured successful test taker. I now faced the "pop quiz" of my life, *the exam that I could not study for!*

For certain, this was the test I could not study for! Preparation for this test included the awful contrast that I always heard about from my patients, but now I had first hand knowledge and confirmation of how it feels to be outside of the white coat. I drank the oral contrast required for this test. Surprisingly, the flatulence that ensued gave me complete resolution of my abdominal pain. It was as if the solution that was intended in making the diagnosis was also the one that provided therapeutic relief. As a physician, I did not lose site of the big picture. The test still needed to be done.

It was now 4 am. I was completely fatigued and ready to go home. It was the type of full day that I usually encountered after being on-call with multiple urologic emergencies, admissions and surgeries. A new ER physician came into the room to report the results. I did not know him directly. He acknowledged me by Dr. Schulsinger, so the thought immediately passed that he knew me or at the very least, he was aware that I was a fellow colleague. His first words spoken were, "do you want me to give these results to you alone or in front of your family?" Wow, what a heavy and loaded question. This response posed

some possible answers with that single question. At the very least, I knew they found something. The new question, was, “What was it?”

It was important for my family to be there. They were my support system, my second ear and of course, my family. The news was not favorable. I had a 12 cm retroperitoneal mass. It was the size of a uterus at 12 week of pregnancy! I was told that this lesion was either a lymphoma, sarcoma or GIST (gastrointestinal stromal tumor) tumor. The list in the differential diagnosis was not good. I then wished that it was a kidney stone because unlike the diagnoses they posed, a kidney stone was not life threatening. I knew that this was only the beginning of additional tests and eventually, surgery.

Now for the first time in my life, I was the startled patient. As a marathoner, the race had begun. As a physician, the choices for choosing words came as a second nature for me. As a patient, all I could think of was recapturing the words stated to me and replaying them over and over in my head; like a YouTube video, the refresh button was on perpetual reset without having to tap it. I wondered if I heard things correctly. I tried to identify any possible hope or solution. These words were very cut and dry, “you have a mass.” Afterwards, the ER attending turned and left the room.

I became the doctor again. I saw the report myself. There were several questions that rushed through my head. Was it a preliminary report read by the resident? Did the report still need an attending physician’s interpretation and seal of approval? My hope changed into sorrow. It was read by the attending, a Radiologist well known to me. Searching for additional answers, I called the Radiologist at home even though it was 4:30 am. She was surprised to hear my voice having just read my films from her home and not making the connection between the patient’s name on the report and me, a colleague, who she had just passed in the hallway days previously. “Is there any chance it could be anything else?” I asked. She said she would review the films and get back to me. After 30 min, I grew tired waiting for the call, emotionally drained, but not yet defeated. It was time to go home.

The short ride home seemed like an eternity! The thoughts driving in my head were “screaming” for answers, but the sound in the car was unusually quiet. I sat in the passenger seat while my dad drove and my wife rode in the back. I could not see her face, but her facial expressions were quite clear. Suddenly, I could hear tears fall and the sounds of her labored breathing and all I could do was attempt to control my emotions within. There were the thoughts of my 3 and 6 year old girls sleeping at home, unaware of our absence. My attention was deflected from me to them. I resigned myself to believing that I had not had a chance to father them. They did not yet even know their father. How much longer would they know me or how much longer would I be there for them? Having lost my biological father on my first day of 6th grade, I only wanted to have a family with children that I could offer a lifetime of parenthood, or at the very least, raise my daughters during those formidable years. For the short 11 years I had with my biological father, the thoughts of time spent with my dad may supersede the period I would live to see my children, an unconscionable and difficult thought to swallow.

The following morning, I met with the hematologist/oncologist, Dr. W. This was the physician I usually send my patients to when they required additional therapy before surgery (neoadjuvant chemotherapy) or after surgery (adjuvant chemotherapy). As a physician, I was able to communicate to Dr. W, as I was talking about another patient: “a 1 day history of mid-abdominal pain exacerbated 1 hour after food; work up included a CT scan demonstrating a 12 cm solid retroperitoneal mass.” As the patient, I sat in silence feeling him palpate and press my belly in silence with an occasional “hmmm.” Next, the crackle of the CT scan images came out of the envelope packet. I could see the sunlight passing through the back of the film with an absence of light blocked by the large circular lesion created by the mass. It was as clear to see as the sun, but solid as any other planet. This was the first time I saw the mass. It punctuated the last 24 hour of events.

“It looks like a GIST tumor to me”, Dr. W replied. “Either way”, he said, “we will need to

begin treatment after the surgery". GIST is a **Gastro-Intestinal Stromal Tumor**, a rare, but aggressive form of an abdominal tumor. Certain that this was cancer, he told me that I would begin chemotherapy immediately after the mass was removed. He would start me on Gleevec, an oral chemotherapeutic agent known to treat GIST tumors. However, it had its known side effects, including congestive heart failure, gastrointestinal bleeding, bleeding from the brain, anemia, liver toxicity, nausea, vomiting, fluid retention, muscle cramps, diarrhea, fatigue, fever, headache, fluid around the lungs, just to name a few! He was a colleague and friend, yet now I saw him in a different light. There was the cold, callous, straight to point approach to these sessions. As a physician, I realized we still did not have a tissue diagnosis. Yet from his professional opinion, he knew what we were dealing with. I took him at his word. As a patient, it was easy to be intimidated by the tone and quickness of our initial visit. I was then quickly rushed from the office to the blood drawing station of the office to have additional blood tests for baseline measures.

The next journey was the colonoscopy. It was explained that 50 % of sarcomas originate from the GI tract, so a colonoscopy was required. As a physician, I was pleased that the test was set up for the next day. Most patients have their first colonoscopy at age 50. I was beginning mine five years earlier. I guess you can never be too early! As a patient, you need to first make an appointment with your GI physician, and then schedule your colonoscopy-the wait of the unknown can be physically and emotionally draining.

My first procedure! I came to the hospital that I work at, care for and treat patients. I had completed close to 5,000 surgeries by this time, but now this became the most important procedure. I met with the Anesthesiologist, a respected physician in the hospital. I came to the OR as a patient in street clothes, not the usual green scrubs I am used to. My morning greeting, however, was like that to a fellow staff member. "How are you today?", I asked. The anesthesiologist replied, "a lot better than you!" As a physician, this was a sarcastic remark. As a patient, it was another wound to endure. I was once again reminded how

critical it is to speak to patients the way you would want to be spoken to.

The first bit of good news arrived. The colonoscopy was negative. While this did not eliminate the possibility that it was a sarcoma, it did rule out where it was originating from.

I visited the surgeon the following day, a fellow colleague at SBUH. It was no longer colleague-to-colleague, but he was the surgeon and I was the patient. The conversation was short, but not sweet. The mass needed to come out and that was final. I enquired about the mass close to the superior mesenteric artery (SMA). He said that we would remove as much of the tumor without sacrificing the SMA. The surgeon in me was not satisfied as that meant, he would save the artery at the expense of leaving tumor behind. Subconsciously, as a surgeon, I knew that all of the tumor needed to be removed. As a patient, I asked the physician, "What was next?" He told me that, "surgery was scheduled tomorrow to remove the mass". I told him this would need to be put off. It was not denial that shadowed my decision, but it was about *the test I could study for!* "I have my recertification exam coming up in less than a week", I replied. I was reminded that, "you have cancer and it needs to come out." Respectfully, I told him it would have to wait until after the exam.

While I was a fellow at NYPH, I spent the first year in the laboratory. I remember saying hello to a gentleman and new surgeon there, Dr. M. He was unknown to me, but always friendly, and our conversations were limited to "hello." Little did I know, that one day this would be the person to potentially save my life. It was now 13 years later, and I transitioned from a fellow, to attending, and now I was a patient at NYPH. This was not the trifecta I wished to brag about. I brought my films in for Dr. M to review. He was my second opinion. The mass was close to my SMA, the major vessel supplying blood to the colon. Without it, one would lose most of their colon, and possibly not survive. He told me that the SMA would need to be sacrificed and a bypass would need to be performed by a vascular surgeon, using an internal jugular vein from my neck or saphenous vein from my thigh. A laparoscopic procedure could

not be considered here, as the mass was too large to be removed by this technique. Dr. M told me that he would meet with the radiologist the following week to review my films. There was still the uncertainty as to what this lesion was, nothing on the diagnosis list that was very promising. The physician in me wanted answers and sooner than later. The patient in me knew I must be patient. The physician in me dominated and I needed more; I needed answers to hold onto. I asked Dr. M, "if you were a betting person, what would you say this mass was?" He replied, "I do not bet." I left his office satisfied that Dr. M was my surgeon, but dissatisfied that I still had no answers. As a patient, I learned that you capture and hold onto any words that the surgeon tells you. The only words I heard repeated in my mind were "I don't bet." I would later realize that this experience would make me a better physician, knowing that you cannot mislead or provide false hope to patients. Honesty is the best virtue, but there are multiple ways to deliver news.

I became the physician again. My recertification examination was now 5 days away and I needed to begin studying again, after a 3 day hiatus. Our Urology recertification exam is only offered once per year and must be completed and passed by the 10th year after completing your board examination. Now I was the patient again. "You have cancer and you need the surgery now", recycled in my mind. The answer became apparent, right or wrong. The surgery will have to wait until after the examination.

The next 5 days became paradoxical. How could you possibly study for a test when a diagnosis of cancer festers in your every thought? This was compounded when reviewing the urologic oncology sections for the test related to the treatment of prostate, kidney, bladder and testis cancers. The solution became very clear to me. If I was thinking about my own diagnosis and treatment, then I was not studying sufficiently. If my thoughts were limited to my review, I was studying firm enough. The study preparation needed to be my distraction. I made certain the intensity was there.

The day before the exam, my wife and I did a trial run to the exam center. I wanted to verify the

location and site to park my vehicle for the test. The mission was a success! The morning after, the exam began at 8 am. I left for the test at 7 am to allow plenty of time to spare. I came to a garage that we identified 1 day earlier, only to find out that they do not accept utility vehicles. With now 15 min until the exam, I began my search through city streets to find a parking spot. With alternate side parking in effect and the exam ending at 12 noon, finding the ideal street parking spot was rather remote. Ultimately, I found a garage to accept my vehicle, only it was 1 mile away with 8 min until the exam began. While I canceled my NY marathon entry a day earlier, I was able to get one last run in, a sprint to the testing center with 1 min to spare.

The test was a success, and now the next journey was to begin. Most of my fellow physicians were going out for a drink to celebrate. As a patient, I was going home to begin my 1 pm bowel prep for my ultimate surgery the next day!

I woke up promptly the morning of surgery. We planned the early journey into NYC as I was the first case that day. I put on the clothes that I usually change into after running the marathon. Subconsciously, I may have thought the race was over, but in actuality, the race had yet to begin. They were comfortable and relaxing. I knew that today was going to be the marathon of my life! I went into my daughters' rooms admiring the way they slept so soundly. I kissed them on their foreheads hoping there would more days I would do the same. My older daughter, Ariel, was 6 at the time, awoke from her sleep. She asked me how I was doing. Trying to remain the role of the physician and parent, I spoke to her as if she was the patient. Sometimes people have a boo boo in their belly that the physician needs to take care of. She reminded me that I was the patient, when she replied, "I know daddy, I know.....they will take good care of you!"

I had the A-Team in place for my surgery. Dr. M was my GI surgeon and Dr. U was my plastic surgeon. Dr. M was going to be removing the abdominal mass and performing the possible bowel resection. The vascular surgeon was on standby, to perform the vascular bypass with a graft from my leg (saphenous vein) or from my

neck (internal jugular vein). Dr. U was performing the abdominal wound closure. My colleagues and friends, including my best friend and best-man at my wedding, Philip, also a staff member from NYH Urology department, made themselves available to check on the surgery and to report to my wife and family waiting cautiously in the family waiting area. The anesthesiologist, not known to me, began the IV and the IV sedation, a protocol used before they begin the general anesthesia during an elective surgery. They asked me to count backwards from 100. The last number I remembered was 99.

Five hours later, I awoke in the OR. I was extubated so I knew the case was over. I could not see anyone around me. I knew sometimes when I completed a surgery, the recovery room was full and the OR was in a holding pattern, retaining the patient until a bed spot becomes free. I was conscious enough to know that I was alert, awake and in the OR. I was still under the influence of the anesthesia so my muscles were weak and I was limited in my movements. I needed answers but there was no one around. I made due with resources in my reach. I palpated my neck to check the dressing covering my anticipated neck wound for the internal jugular vein they would have harvested. Astonishingly, there was no dressing there. The curiosity continued to grow. I then reached toward my groin to feel the dressing over the location where my anticipated wound for the harvested saphenous vein would be. Unexpectedly, there was no bandage there either. The curiosity peaked. How could they salvage the superior mesenteric artery engrossed within such a large mass?

I was then wheeled to the RR. I was more awake, but my vision was somewhat fuzzy. I could see the silhouette of my wife but it was difficult to make out her facial expression, a barometer that allows me to gauge the situation. I could not see if she was happy or sad. "Did you hear what happened?", she cried. I was somewhat puzzled as she was the first person I have seen and heard from since the surgery. "No," I replied. "It was a hematoma....a blood clot", she sighed. "A what?", I whispered. I could not process this information, as this was not part of the differential diagnosis. There was no discussion for this lesion

being anything but cancerous. As a patient or physician, this news was pleasing, yet confusing.

The doctor came into my area of the RR, he pulled the curtain away. So "when did you get kicked by a horse?" he said. He continued, "The hematoma was large, solid and old." To generate a hematoma that size, a significant force must have generated it. While an animal did not kick me, I told him that I had a couple of fender benders with my car. "That would have not done it," he believed. The actual cause remains a mystery. Looking back, I do recall a ski accident during my freshman year of college. While night skiing one Friday after classes, my abdomen landed over my ski poles after landing from a 20 ft jump, knocking the wind out of me. This was now 27 years later, however, the true cause remains unknown.

Hours later, I was still in the RR, awaiting a bed on the floor. Family, friends and most of the staff had gone home. The phlebotomist came by to make their evening blood drawing. They came to draw blood from my arm on the same side as the hand with the IV. I told the phlebotomist that you might want to draw blood from the opposite arm. They told me that it was not important and that they can proceed. I realized that I just survived a cancer scare and nothing else in world mattered at the time. It was 2 hours later when the resident ran into my room, he came with a red top tube and a syringe. The expression on his face was one of fear and with great concern. He told me that he needed to draw blood. "What is this for?", I questioned. "It appears that you lost blood and you will require a blood transfusion", he said. He was unaware that I just had blood drawn from my arm with the IV, resulting in a hemodiluted blood drawing. Following this, they re-drew the blood from the opposite arm demonstrating a normal hematocrit and all is well. The moral of the story is to ask questions when blood is being drawn and do not assume or take anything for granted. If you are a patient and tests are being performed or medication is being administered, do not feel embarrassed to ask the appropriate question: Why are we doing that? What is it?

I returned home from the hospital after several days in NYH. I called several people to share the good news, including Dr. W, the physician who

told me that I would start chemotherapy after the surgery. Once telling him the lesion was benign, I heard a thump to the floor. To this day, I am not certain if it was the phone or the doctor falling to the floor. When Dr. W returned to the phone, he told me “I hear a story like this 1:10,000 cases...I am glad I got it wrong.”

I thought by now, the worst was behind me. I was cancer-free. I had a new lease and a new appreciation for life. Now I could potentially see my children on the second day of their 6th grade of school. Little did I know that there was another medical adventure awaiting me just around the corner.

Visiting the Other Side: *The Roller Coaster Ride Continues!*

2

David A. Schulsinger

It was 2 months before my wife's 40th birthday and the planning had begun. In general, I have always tried to sequence the planning of important events with enough cushion time in between to avoid facing any conflicts and to also avoid missing out on the opportunity to celebrate life's most precious milestones. For instance, to the best we could, we planned our daughter's birth the month before my 6th New York City Marathon, this allowed for enough time to gracefully welcome her into the world and still train for the run and have family by my side to cheer me on the side lines. It was not atypical then for me to concomitantly plan what would be my next surgery and a surprise party for my wife in a sequence where I could still plot out training runs for my 14th New York City Marathon.

It was now 1.5 years from my original surgery. Since that procedure, I had developed an incisional hernia, which became larger over time. Incisional hernias are not unusual after abdominal surgery. The surgical site and repair represents the weakest location of the abdominal wall, and intestinal contents that fill that area can present with a rather large bulge. Prior to the procedure, the plastic surgeon took a photo of my abdomen. Even with deep inhalation, my

abdomen protruded far enough out that I could not see my belly button or my urologic anatomy. I was eager to have this treated.

I chose to have my wife's surprise 40th birthday, the day before my surgery knowing that all the planning would occur before the party behind the scenes. After the joyous celebration, I went in for the surgery the following day. In the rare event that my recovery was longer than normal, I felt that I had better control of events if I made her surprise party first. Additionally, planning for the party and the "elective" surgery well in advance would provide enough time for the recovery and time for marathon training. Low and behold, the surprise for me would be far greater than the one my wife experienced on her birthday.

Having survived the threat of cancer leading to my first procedure, I had no idea that my second surgery was potentially more life threatening. After I left for the hospital for my first surgery a little more than a year before, there was a slight doubt as to whether I would return home. In anticipation of the second surgery, I left my house on the day of surgery already knowing the projects to do around the backyard and inside the house. I kissed my kids who were going to school, knowing that I would be at their weekend softball and tennis commitments. My wife double checked with me on our way into the car saying, "hernia repairs are benign and low risk procedures right?" My response was "Of course, many people go home the next day. There's nothing to worry about."

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

For the first time, I became exclusively the patient, separating my thoughts of being a physician and knowing the rules that patients play by. As a surgeon, I always thought being on the other side meant being on the other side of the OR table. This was the side of the OR table, separated by a surgical drape, where the anesthesiologist worked. Surgeons treated the patient with surgery while anesthesiologists “passed the gas!” My interpretation, however, of *visiting the other side* had taken on a whole new meaning. At this venture, it meant being the patient and asking questions of my doctor. I became a patient that put my hands in the trust of a surgeon, the same way a passenger directs his confidence to the pilot of an aircraft. Good or bad, I was naive about any risks in the situation; possibly this was a result of the trust I developed earlier considering I survived a great scare and believed that not much could be worse than the previous situation. Needless to say, I kept my A-Team for this procedure as they did right by me for the first procedure. Dr. M would place the mesh for the hernia repair and Dr. U would do the abdominal wound closure.

When getting authorization from my patients for minor or major surgeries, one must get an informed consent. The consent identifies your surgeon, the technical name of your procedure and well as the procedure described in laymen terminology. It will detail the risks of the procedure, including issues like infection, bleeding and other items that are specific for that procedure. While the outcome is quite rare for most elective procedures, it is not inappropriate to tell a patient that death, is always a possibility.

Following my elective hernia procedure, I returned to the recovery room without any delay. It was the same RR and bed spot that I remember bringing my patients to and the place that I visited as a patient only 1.5 years earlier. The surgeon had come to the bedside once again. Last time, he asked me “when was I kicked by a horse?” Ironically, now I felt like I was just kicked by a horse. On a pain scale of 1–10, where 1 was minimal pain and 10 was severe pain, my agony level was an 11. The surgeon, who thought I initially had an abdominal bulge, confirmed that I

had multiple hernias. He stated, “Yes, you did have a hernia, but not 1 or 2 or even 3, but 5 of them.” A hernia of this nature is nicknamed a Swiss cheese hernia, based on the visual of multiple holes representing each hernia. The surgical team did not need to enter my abdominal cavity; they were able to perform the procedure from a superficial approach utilizing the mesh.

Exiting my abdomen, there were 2 tubes, called Jackson Pratt drains. These drains were connected to bulbs, which were football-shaped containers, draining extra fluid and blood between the muscle layers of my abdominal wall, each holding 100 cc of liquid. I recall the aid coming by to drain the blood. It appeared that she came quite frequently, but at this point following my surgery, my memory was not acute. With 2 bulbs, the collected volume was 200 cc. They drained them while I was in the recovery room and they continued draining blood from them while I was sent to the floor.

While blood was collected and drained from each bulb, I was not aware as of the amount. My mind began to race as to the source of bleeding from a superficial and relatively avascular surgery. This was not a primary vascular procedure or open heart surgery. However, anytime a patient has surgery, the risk of bleeding is always there. When a patient signs consent prior to a surgical procedure, the risk of bleeding is always one of the first potential complications described.

I recalled from my first year anatomy class of medical school, the blood supply to the superficial abdominal wall muscles included the inferior epigastric artery. In the human body, inferior epigastric artery arises from the external iliac artery in the groin area and anastomoses or attaches with the superior epigastric artery.

Our abdominal wall is a multilayered sandwich of multiple muscle layers, including the rectus abdominis layer, the external oblique, the internal oblique and the transversus abdominus. The inferior epigastric artery pierces through these muscle layers. A tear or injury to this vessel could result in significant bleeding. As a laparoscopic surgeon, this is a vessel we attempt to avoid when placing trocars, a conduit to the abdominal cavity, to place our working instruments during the surgical procedure.

There was the pain from the surgery, the drowsiness from the narcotics to control the pain, and there was the lightheadedness from the bleeding, all converging at the same time. It was the perfect storm, except my health was in question. The pain was able to neutralize the drowsiness, but the lightheadedness from the bleeding continued to grow. I knew my time to respond was limited. I wanted to be efficient about my time and make sure that I could reach out to the people that mattered.

Once again the aid came by to drain the bulbs. I was more aware of the frequency in which they were drained, but incredibly fatigued to maintain my concentration. I realized another perfect storm had occurred. The nurse in the RR had changed shifts while I was sent to the floor. The floor nurse had not seen me as she was still dispensing medication to her patients on the ward. The total volume of blood collected in the RR and on the hospital floor was not common knowledge. The aids, assisting the nurses, continued to drain the bulbs. I was awake enough to know that she was coming frequently; not coherent enough to sum up the number of times. I asked the floor aid how many times. She told me at least ten times. Now I could do the arithmetic. I searched for my phone. I found his number and called my surgeon immediately. "Mike, where are you?", I shouted. He was home with his family. I told him that he needed to bring me back to the OR, immediately. "Why?", he asked. "I am hemorrhaging", I shouted. Without questioning me, he would have me in the OR within the hour.

I awoke from the second surgery of the day. I was now in the recovery room for the 3rd time in 18 months and the second time within 11 hours. I could hear the surgeon's walk as he approached my area of the RR once again, footstep sounds I can still hear and was anxiously waiting for. Although I knew the sounds of his steps, I did not know what he was going to say. I took for granted, as a surgeon, the time I spoke to my patients after their procedure. Oftentimes, I would speak with them, but the effects of the Versed (Midazolam), the anesthetic received in the OR, would cause retrograde amnesia, whereby patients would not remember situations from the immediate past. It

took a long time for me to learn as an attending physician the rationale for not telling patients too much in the RR immediately after surgery because of how typically it was not remembered minutes later. Patients who would typically ask very precise questions about their procedure would later say "doctor, I have not seen you since the surgery, how did things go?"

My memory was very acute, but the pain was not as sharp as I experienced earlier. Dr. M would always make the astonishing revelation in the RR following my procedure. This time it would be about the numbers. "We identified the source of blood and removed ½ liter of blood from below the mesh", he shared. He mentioned that the pressure exerted from my abdomen must have been great as I blew out 3 sutures holding the mesh in place. They repaired the mesh that was torn. Finally, they repaired the wound closure from the previous surgery. Everything combined, the surgery took another 4–5 hours.

"Did I get a blood transfusion?" I asked. "No, I did not give you any blood. Your young enough to handle it. Being anemic would help to slow you down a bit", he advised. Suggesting that I was very active, he said, "I would rather you be a little bit slow and tired as this will allow you to recover." With a hemoglobin and hematocrit of 6.8 and 17, I was not planning on running races anytime soon.

Slow was no exaggeration! The anemia that I experienced was more than just a number verifying that my blood count was low. My capillary refill was quite delayed. This is a test to grossly measure the blood level in our capillaries. When pressing your finger against your skin, the blood "refill" may take a second or two. My capillary refill was like a snail's pace, taking approximately 6 seconds.

The ability to raise my self in the bed was a tremendous task. I would become exhausted. I could not even raise my head without feeling lightheaded and watching the room spin around me.

Taking iron supplements would make me constipated, the last thing I would want to do to compromise the second wound closure. I suggested that I receive Venofer, a form of IV iron. This

treatment, combined with the Ensure milk shakes, would put my bone marrow into overdrive. It took 5 days for me to get out of bed and 7 days until I could leave the hospital. It was now the middle of April, knowing that my marathon training was to begin in 6 weeks. It was hard to imagine the link between crawling out of bed and the idea of running 26.2 miles, only months away. I did not, nor did I anticipate 2 surgeries that would set me back. This required a longer recovery period. Among my personal goals in 2010 was to run the marathon. I was determined and I knew that this would come to fruition.

Following my discharge, I slowly began feeling stronger and my endurance made my days longer. I returned to work 4 weeks later. I began to reflect on my patient experience each time I saw a patient. I felt that I had taken a course on “a physician’s perspective on how it is to be a patient, 101.” Whether it was the patient I examined in the office, the patient I was preparing for surgery in the ER or the patient I spoke with about their surgery in the recovery room, the reflection of my personal experience became transparent.

It was now November 3, 2010, the day of the New York City Marathon. It was my 14th NYC marathon. As always, I would travel to Staten Island with my belongings for the day. I would take my 6 power gels for the race; a Tylenol before and 1 for after the race; a fresh pair of dry warm clothes to change into after the race, a fresh pair of socks and a dry pair of sneakers. For this marathon, however, I would require one additional piece of attire. My abdominal binder! For now, I was complete and had gone full circle.

You may ask yourself, “What do the first 2 chapters of this book have to do with kidney stones?” These chapters were written to share with you my personal encounter as a patient with a deeper understanding that situations can happen, even unexpectedly in circumstances that appear less than risky. Literally and figuratively, it was a crash course on becoming a patient. It was not until I became a patient, that I was able to understand the mindset of the patient. Medical school, residency, and fellowship teach you how to think to be a well skilled and compassionate

physician. This training does not educate you how to be a patient and what you can learn from being a patient. You cannot always think as a physician, and you cannot always take things for granted as a patient. It is important to be vigilant, thoughtful and uninhibited enough to ask the most thoughtful questions.

I want for my patients and for you to realize that your personal health and the health care system should not be taken for granted. As you would make it your homework assignment to educate yourself before the purchase of a new computer, a new TV, a new car, or a new home, your health also deserves, at the very least, the same level of attention to details and decisions. The goal is to be able to become an informed decision maker. This book is designed to allow you to ask the appropriate questions, to be insightful and to be diligent and cautious with respect to receiving not only the best quality care, but also the most appropriate care.

After I had an opportunity to digest and absorb the series of events that had taken place, I found it not unusual that things can happen. These situations can happen to people who are patients and they can happen to physicians who become patients too. The only difference was that I had a heightened degree of awareness of what I thought was right and what was wrong. With all said, I was a victim of the circumstances that can happen, and which happen every day in a medical environment regardless of the patient’s profession. My elective procedure became a potentially life threatening event. It goes to show you, that at any time, no matter the simplicity or complexity of the procedure, any surgery can result in an adverse event or potentially death. It is paramount for you to have at least, a basic understanding of your scheduled procedure and have knowledge of many dimensions of thought. Understand your options and whether the procedure you are considering is required. Try to determine the risks and benefits and if there are alternative options before you opt for one specific procedure. Learn to do due diligence to the phrase, “doing your homework,” as this may be the most important assignment you have been given in life.

Just as any baseball team can beat another team on any given day, a similar situation can happen during any surgery. You take the appropriate precautions to avoid infections with antibiotics and minimize bleeding by discontinuation of blood thinners. Any type of surgery, no matter how simple or complex can result in a good outcome or potential complications. This is why it is paramount to be certain that when scheduled for surgery, be certain that the surgery is required and that all alternative treatments have been discussed and that all conservative approaches for treatment have been considered.

I decided to write this book, with you, the current patient, the future patient, or the friend or relative of a patient in mind. As an Academic Urologist and as a specialist in stone disease and Endourology, I wish to share with you what you need to know about stone disease; if you require treatment, the different dietary, medical and surgical treatment options of stones; identify the risk factor for stones; how to choose your urologist; how to ask the appropriate questions and how to make educated decisions on stone prevention to minimize your risk for future stones. Let's begin the journey!

Part II

The Significance of Stone Disease

David A. Schulsinger

The Short and the Long

- The urinary system consists of kidneys, ureters, urinary bladder and urethra.
- The urinary tract is the body's drainage system for removing wastes and extra water.
- The ureter has three regions of narrowing, including the UPJ, crossing iliac vessels and the UVJ, which are the most common sites where stones can obstruct.

The urinary tract system is a group of organs in the body, which are responsible for the excretion of urine. These organs include the kidneys, ureters, urinary bladder and urethra (See Fig. 3.1). In humans, the urinary system includes two kidneys, a pair of tube-like structures, called the ureters, which connect the kidneys to the bladder. The bladder is connected to the urethra, which delivers the urine to the outside of the body. The primary function of the urinary tract system is to remove substances from the blood, to form urine and help to regulate various metabolic processes, including water, ions, pH, and blood pressure. The urinary tract is the plumbing system of the body that works to drain urine produced by the kidneys, store it and then release it during voiding.

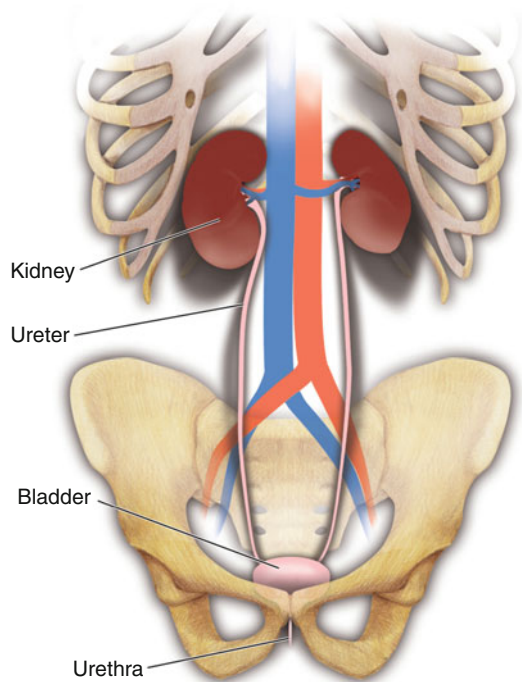


Fig. 3.1 The urinary tract demonstrating the location of the kidneys, ureters, bladder and urethra

What Is the Urinary Tract?

The kidneys are two bean-shaped organs, each about the size of a fist. They are located near the middle of the back, just below the rib cage, one on each side of the spine. The urine flows from the kidneys to the bladder through tubes called ureters.

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

The bladder stores urine until it is released. When the bladder empties, urine flows out of the body through a tube called the urethra at the bottom of the bladder, a process called *urination*. This medical terminology for this process is known as *micturition*.

The Kidneys: *The Stone Factory!*

Function

The kidney is responsible for two primary functions. First, the kidneys work by extracting water, urea, mineral salts, toxins and other waste products from the blood to form urine, with filtering units called nephrons. From the nephrons, the urine is collected into the pelvis of the kidney. Both kidneys process about 200 quarts of blood to produce about 1–2 quarts of urine/day, primarily composed of wastes and water. Secondly, the kidneys regulate the production of red blood cells, blood pressure, blood volume, blood composition and pH of the blood.

Renin is an enzyme secreted by blood vessels of the kidney, called afferent arterioles. It converts angiotensinogen, produced by the liver, to

produce angiotensin I, which is further converted into angiotensin II, a potent vasoconstrictor, by angiotensin converting enzyme (ACE). The primary function of renin is to increase the blood pressure, leading to restoration of perfusion pressure within the kidneys.

Structure and Location

The kidney is one of a pair of small bean-shaped organs of the urinary system, located near the spine. They lie on either side of the spinal column in a depression high on the posterior wall of the abdominal cavity, and are positioned behind the parietal peritoneum, called the retroperitoneum, against the deep muscles of the back. The right kidney is located slightly lower than the left kidney because the right side of the liver is much larger than the left side, pushing the kidney down to a lower position. The kidneys are surrounded by a layer of adipose tissue, called Gerota's Fascia, which holds the kidneys in place and protects them from physical damage.

The meat of the kidney is called the parenchyma. This tissue consists of many nephrons, which represent the functional unit of the kidney (See Fig. 3.2). The nephron consists of a renal

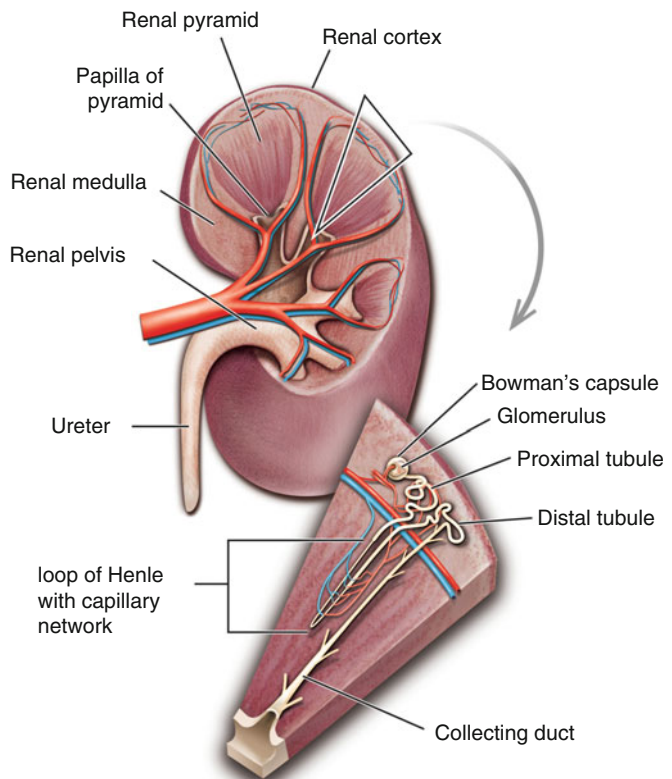


Fig. 3.2 The kidney and its relationship to the nephron

corpuscle made up of a glomerulus and Bowman's capsule, and a renal tubule made up of a proximal convoluted tubule, loop of Henle, distal convoluted tubule and collecting duct. The kidney contains a hollow chamber, called the renal sinus. Through the renal sinus passes various blood vessels, nerves, lymphatic vessels and the ureter. The superior end of the ureter is expanded to form a funnel-shaped sac called the renal pelvis, which is located inside the renal sinus. The renal pelvis is subdivided into several tubules or major calyces. The major calyces are then subdivided into minor calyces.

The majority of stones form within the kidney. The development of stones begins with formation of crystals in the concentrated urine, which adhere to the urothelium, generating a nidus for stone growth. Many calcium oxalate stones develop Randall's plaques, which are composed of calcium phosphate crystals. As these small stones grow, they erode through the urothelium, forming a nucleus for calcium oxalate stone formation.

The Ureters: *The Rate-Limiting Step!*

Function

The ureters are a pair of thick-walled tubes leading from each kidney, which carries urine to the urinary bladder. Urine flows down partly by gravity, but mainly by a wave of contractions, called peristalsis, which pass several times per minute through the muscle layers of the ureteral walls.

Structure and Location

Each ureter is a tubular organ measuring about 25–30 cm (10–12 in.) in length and approximately 3–4 mm in diameter. The ureter begins at the funnel-shaped renal pelvis, called the ureteropelvic junction (UPJ), and extends along the left and right sides of the body, on top of (anterior) the psoas muscle, parallel to the vertebral column as they descent toward the pelvis of the body, called the pelvic brim. At this point, the ureters cross over (anterior) to common iliac arteries, which are blood vessels to the lower extremities (left and right legs). As the ureters pass along each side of the pelvis, they then curve and enter the bladder from each side as they enter the back of

the bladder, a location called the ureterovesical junction (UVJ).

Within the wall of the ureter are three layers. The inner layer, or mucous coat, is continuous with the linings of the renal tubules above and the urinary bladder below. The middle layer, or the muscular coat, is composed of largely smooth muscle fibers. The outer layer, or the fibrous coat, is primarily composed of connective tissue. Each ureter enters the bladder through a tunnel in the bladder wall, which is angled to prevent the urine from running back into the ureter, known as reflux, when the bladder contracts.

The ureters have three anatomical regions where the ureter narrows. This includes the proximal or beginning portion of the ureter, called the UPJ. The second anatomical narrowed area is where the ureter crosses in front of the common iliac vessels. Finally, the last narrowed area is the lower end of the ureter, or distal end, where the ureter enters the bladder, called the UVJ. These are the three structural regions where stones leaving the kidney can get stuck within the ureter.

The Bladder: *No Other Place to Go but Out!*

Function

Urine enters the bladder from the ureters above. The urinary bladder is responsible for the storage where the average bladder capacity is approximately 400 ml of urine. Finally, the urine is delivered into the urethra for excretion from the body.

Structure and Location

The urinary bladder is a hollow, distensible, muscular organ, which is a reservoir to store urine. The bladder is located along the body's midline at the inferior side of the **pelvis**. It is spherical in shape, however, the pressure of the surrounding organs causes its shape to become altered. When the bladder is empty, the inner wall retracts into many folds, but as it is filled with urine, the bladder wall expands and becomes smoother. The wall of the urinary bladder consists of four layers. The inner layer, or mucous coat, is composed of epithelial cells. The second layer, or submucous layer, is made up of connective tissue and contains many elastic fibers. The third layer, or muscular coat, is

primarily made up of coarse bundles of smooth muscle fibers. These muscles in the muscular coat are interlaced to form the detrusor muscle. The portion of the detrusor muscle around the neck of the bladder forms an internal urethral sphincter, which controls the excretion of urine. The outer layer, or serous coat, is made of the parietal peritoneum. However, this layer occurs only on the upper surface of the urinary bladder, or bladder dome. Elsewhere, the outer coat is composed of fibrous connective tissue.

The Urethra: *Go with the Flow!*

Function

In females, the urethra is a tube that transports urine from the urinary bladder to the outside of the body. In males, the urethra also functions as a urinary canal and as a passageway for sperm and secretions from various reproductive organs.

Structure and Location

The wall of the urethra is lined with mucous membranes and contains a relatively thick layer of smooth muscle tissue. It also contains numerous mucous glands, called “urethral glands” that secrete mucous into the urethral canal. The smooth muscle fibers within the urethra run longitudinally. In females, the urethra is about 4–5 cm (2 in.) long. It passes forward from the bladder, descends below the symphysis pubis, and empties into the labia minor, inferior to the clitoris and superior to the vaginal canal. In males, the urethra is approximately 20–25 cm (8–10 in.) in length and can be divided into four sections: the prostatic urethra (3 cm), the membranous urethra (1 cm), the bulbar urethra and the penile urethra (16 cm). The prostatic and membranous urethra represents the posterior portion of the urethra; the bulbar and penile urethra are referred to as the anterior portion. The end of the urethra, called the urethral meatus, is located on the head (glans) of the penis.

The urine passes through the urethra and is controlled by the internal and external urethral sphincter muscles. The internal urethral sphincter is made of smooth muscle and opens involuntarily when the bladder reaches a given level of

fullness. The opening of the internal sphincter results in the sensation of needing to void. The external urethral sphincter is made of skeletal muscle and may be opened voluntarily to allow urine to pass through the urethra or may be held closed to delay or interrupt the urination.

Anatomical Variations

The previous section describes the normal function and structure of the urinary tract. In some patients, there can be a variation of the urinary anatomy, called congenital anomalies.

Kidney

Renal Fusion Anomaly

Horseshoe Kidney

In this congenital anomaly, there is a fusion of the lower poles of the kidney to form a horseshoe-shaped kidney (See Fig. 3.3). Horseshoe kidney is the most common renal fusion anomaly with a prevalence of 1:1,500. Most cases of horseshoe kidney are asymptomatic, but this anomaly may be associated with risk of renal obstruction, renal infection, stone disease and renal tumors.



Fig. 3.3 Coronal CT image of horseshoe shaped kidney

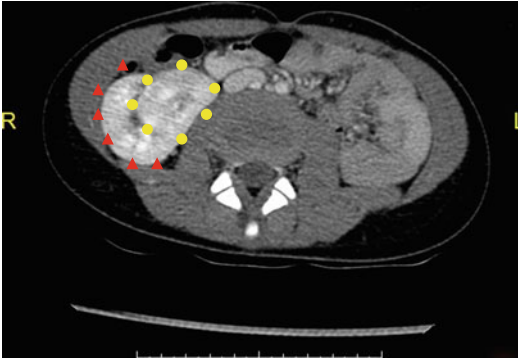


Fig. 3.4 Axial CT image of crossed fused renal ectopia with both kidneys (*red triangles and yellow circles*) on the right side

Renal Ectopia

Renal Ectopia or ectopic kidney refers to a kidney that is not located in the normal anatomical location.

Crossed fused renal ectopia is an anomaly where the kidney from one side is fused and located with kidney on the same side of the midline (See Fig. 3.4). The incidence of this is 1 in 1,000 births [1] with a 2:1 male to female ratio. Approximately 85 % of crossed renal ectopia present as fused kidneys.

Ureter

Ureteral Duplication

A duplicated collecting system is the most common congenital variations of the urinary tract [2, 3], occurring in 1 % of the population. The collecting system divides the kidney into an upper pole moiety and a lower pole moiety, each draining into a separate ureter. Ureteral duplication can present as complete or incomplete duplication of the collecting system. Duplex systems can exist on one side (unilateral) or both sides (bilateral, 10 %).

Complete

In complete ureteral duplication, there are two ureters originating from the kidney that drain separately into the bladder (See Fig. 3.5). The

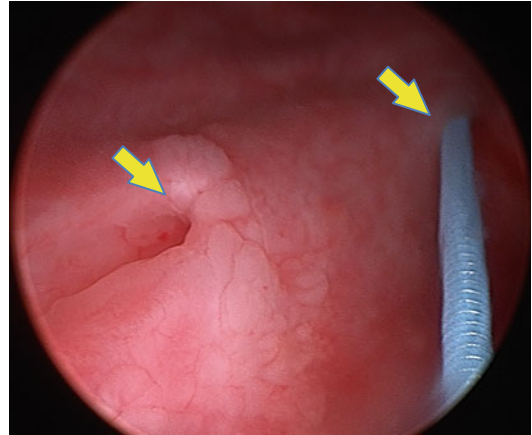


Fig. 3.5 Cystoscopic view of the bladder demonstrating two separate orifices (*yellow arrows*) from the left kidney draining into the bladder. A wire was placed in the lateral ureteral orifice

lower pole ureter will drain normally into the bladder. The upper pole ureter can drain into the bladder or ectopically into other areas of urinary or reproductive tract, including the urethra, vagina, vulvar vestibule or prostate.

Incomplete

An incomplete ureteral duplication, or bifid ureter, is where a duplex kidney drains into separate ureters, but the tubes will unite into a single ureter before draining into the bladder.

Bladder

Bladder Exstrophy

Bladder exstrophy is a rare congenital anomaly, which involves the protrusion of the urinary bladder through a defect in the abdominal wall (See Fig. 3.6). The incidence of this anomaly is 1 in 10,000–50,000.

Urethra

Hypospadias

Hypospadias is a birth defect that results in an abnormally positioned urethral meatus, positioned in a location separate from the glans of the

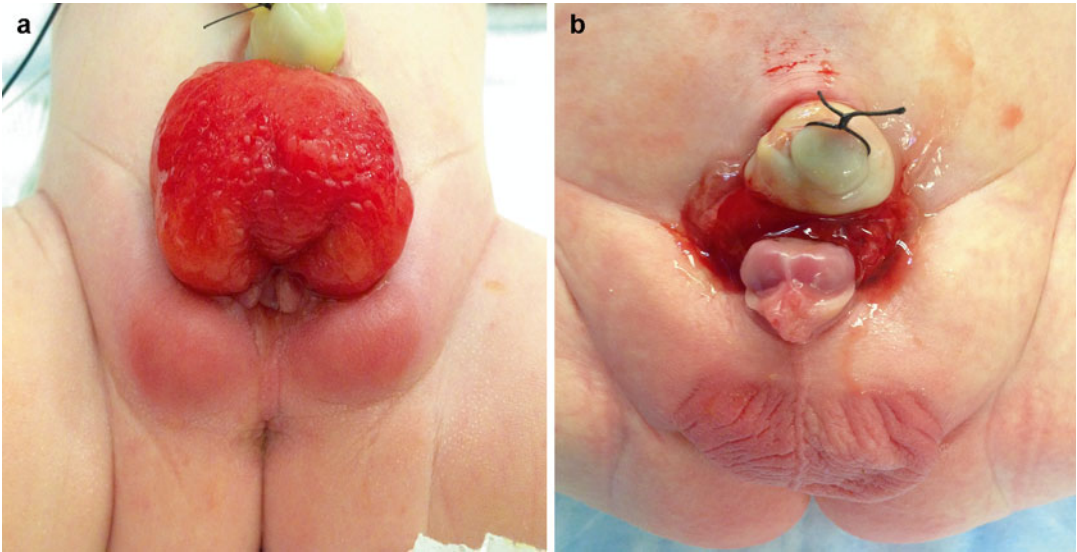


Fig. 3.6 Bladder exstrophy with bladder visible on the abdominal wall of female (a) and male (b) newborns

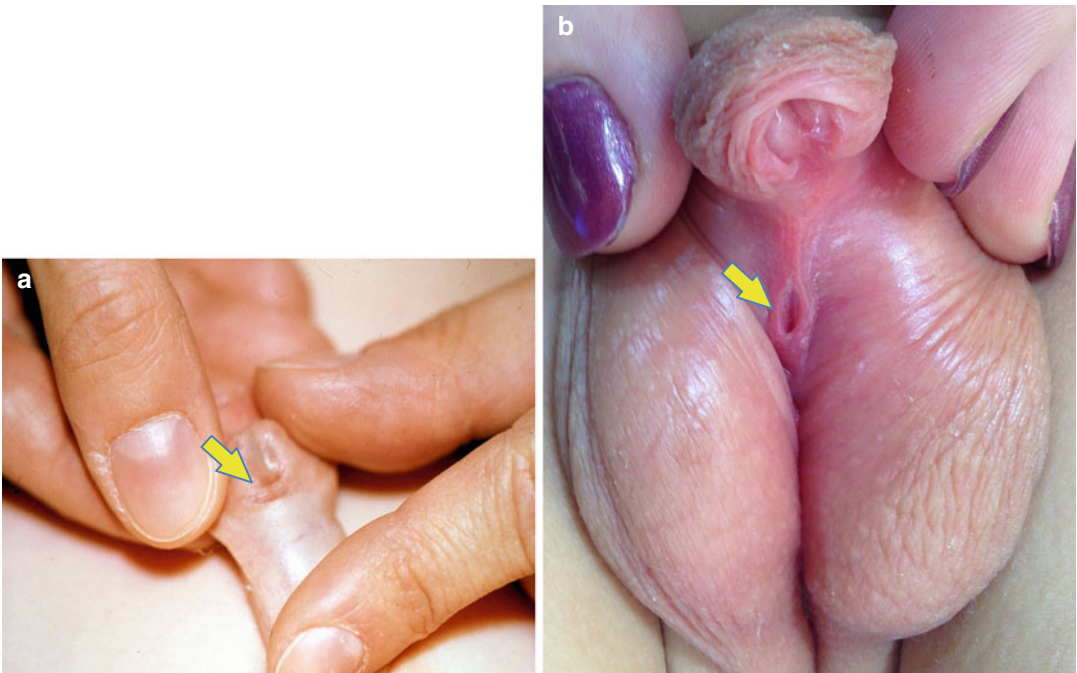


Fig. 3.7 (a) Hypospadias demonstrating the meatal opening (yellow arrow) on the ventral surface (underneath portion) of the glans penis; (b) penoscrotal hypospadias

penis (Fig. 3.7). The urethral meatus can open anywhere along the course of the urethra on the ventral (underside) side of the penis, including

the shaft of the penis, scrotum or perineum. The incidence of hypospadias is approximately 1:125 newborn males [4].



Fig. 3.8 Epispadias demonstrating urethral opening (yellow arrow) of the penis on the dorsal (top) side

Epispadias

Epispadias is urethral malformation in which the opening of the urethra is on the dorsal (top side) aspect of the penis (Fig. 3.8). The incidence of epispadias is approximately 1 in 200,000–500,000 births.

Conclusion

In summary, the urinary tract is the plumbing of the body that is responsible for removing waste and excess water. The urinary tract is composed of the filtering system, called the kidney, tubes to drain the system called ureters and urethra and a reservoir to store the urine called the bladder. The urinary tract can also form stones where they can become trapped and obstruct the urinary tract in both normal anatomy and in patients with congenital anomalies.

Ms. LP was a 35 year old female with new onset of right flank pain. During her ER visit, a renal US demonstrated a “solitary enlarged right kidney” with hydronephrosis

but no stones were identified. Due to persistent pain and vomiting, there remained a high degree of suspicion that a stone was present. The patient had a spiral CT scan demonstrating a crossed fused renal ectopia and mid ureteral stone in the crossed ectopic kidney was present. The patient underwent a ureteroscopy with laser lithotripsy with basket and removal of the stone. Tommy was standing among a group of other patients to get his release from the psychiatric day facility. The physician asked each patient to identify body positions as he pointed to different body parts. He asked, “point to your left knee”, “point to your right elbow”, “point to your right leg”, “point to your stomach”, etc. Tommy watched very carefully as the questions were asked, especially the answers that were correct. When it was his turn to be asked the body parts, he answered the entire list of questions correctly. The doctor was surprised and curious that he knew all the answers. When Tommy was asked, “how did you know the answers to the questions?” Tommy replied, pointing to his head, “the kidneys, man! The kidneys!”

References

1. Dunnick NR. Textbook of uro radiology. Philadelphia: Lippincott Williams & Wilkins; 2001.
2. Croitoru S, Gross M, Barmeir E. Duplicated ectopic ureter with vaginal insertion: 3D CT urography with i.v. and percutaneous contrast administration. *AJR Am J Roentgenol.* 2007;189(5):W272–4.
3. Fernbach SK, Zawin JK, Lebowitz RL. Complete duplication of the ureter with ureteropelvic junction obstruction of the lower pole of the kidney: imaging findings. *AJR Am J Roentgenol.* 1995;164(3):701–4.
4. Paulozzi LJ, Erickson JD, Jackson RJ. Hypospadias trends in two US surveillance systems. *Pediatrics.* 1997;100:831–4.

The Rock of Ages: Stones Have Stood the Test of Time!

4

Abe D'Amato, Yefim Sheynkin,
and David A. Schulsinger

MileSTONES of the Disease

The first emperor of the Roman Empire Caesar Augustus, James I of England, King Louis XIV of France, Russian Czar, Peter the Great and George IV had them. Martin Luther, Michelangelo, Oliver Cromwell, Sir Isaac Newton endured them. Benjamin Franklin, President James K. Polk, Lyndon B. Johnson experienced them. Actor William Schatner sold them. Even the fictional character from *Seinfeld*, Cosmo Kramer, suffered with them. What if Napoleon Bonaparte had not had his stone during the Russian campaign in 1812? All of world history might have changed!

Rocky Road to the Treatment of Urinary Stones

The history of urinary stones is as old as civilization itself. Urinary stones are an ancient health problem and have afflicted humans since the dawn of history. In 1901, the English Archeologist E. Smith found a bladder stone from a 4,500 to 5,000-year-old mummy in El Amrah, Egypt. The removal of kidney and bladder stones is one of

the earliest known surgical procedures. Treatment for stones was mentioned in ancient Egyptian medical writings from 1500 BC. The first descriptions of the surgical treatment of bladder stones are found in Hindu and Greek writings. Sushruta, physician who lived in ancient India around 600 BC, provided detailed description of surgical removal of the bladder stone through the perineum (area between genitals and anus).

In ancient Greece, Hippocrates (460–377 BC) defined symptoms of bladder stones as follows: pain in urination, passage of urine drop by drop, blood-stained urine, inflammation of the bladder, and passage of sand in the urine. He opposed surgical treatment of stones as a risky and dangerous procedure, which has to be performed only by specialists: “I will not cut to the stone, but will leave this to be done by practitioners of this work.” Hippocrates’ authoritative statement could be one of the strongest obstacles to the development of new surgical approaches up until sixteenth century, with one notable exception. In the third century BC, Ammonius of Alexandria suggested crushing the stone to facilitate its removal. He designed a hook or crotchet to hold the stone and blunt ended iron instrument to cleave the stone. Ammonius used the word “lithotomus” in relation to cutting the stone (“lithos”-stone and “tomos”-cut). Unfortunately, this idea was not popular and the principle of stone fragmentation was revived only about 2,000 years later.

In the first century AD Roman physician and philosopher, Cornelius Celsus, provided a written

A. D'Amato, MD (✉) • Y. Sheynkin, MD •
D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: Abe.damato@gmail.com;
ysheyn@yahoo.com; endourology@yahoo.com

description of how to remove a bladder stone with a hook or fingers. He recommended it only for boys between ages of 9 and 14 years old. While he never performed the procedure himself, his book remained a landmark in the stone surgery for many centuries ahead. For almost 16 centuries this technique was never questioned or even modified despite high surgical mortality.

During early medieval times in Europe, the practice of surgery was relegated to craft status with training by apprenticeship. Most bladder stone surgeries were not performed by university educated physicians but by the lowest group of surgical practitioners, the barbers. They traveled around Europe with special tables called "lithotomy tables" on which they placed patients to cut out stones. Some of those traveling barber/surgeons became top specialists after operating on a large number of patients. The procedure was generally performed in public and in the best hands lasted only a few minutes. Many of these "surgeons" were just dishonest showmen. The fear of inexperienced "stonecutter" made Jan de Door, Dutch blacksmith, to remove the stone from his bladder by himself after two previous unsuccessful and painful procedures by a "specialist". While malpractice was not yet introduced to the medical profession, the travelling "stonecutters" were held responsible for their bad results and fined accordingly. The punishment did fit the crime. In one case, the fine was a sum of money sufficient to build a stonewall about the local churchyard.

In 1556, Pierre Franco removed a stone from the bladder of a child via a low abdominal incision. It was done in despair since he could not remove the bladder stone with the traditional approach. The practice was not accepted at that time due to multiple complications, but slowly became the main surgical approach to bladder stones.

In the early seventeenth century, the idea of crushing stones without opening the bladder gained popularity. Many new instruments were designed to be inserted into the bladder via the

urethra blindly in order to grasp and fragment the stone. Stones were crushed, broke and drilled but fragments still had to be evacuated through a bladder incision. The mortality of the procedure remained very high. The problem was not solved until 1878 when Maximilian Carl-Friedrich Nitze, a German urologist, presented the first working cystoscope, a hollow tube equipped with a lens and light. Now bladder stones can be seen and fragmented under the direct vision with complete evacuation of fragments via the cystoscope.

At the same time, significant progress was achieved in the surgery of kidney stones. While symptoms of kidney stones were also known as an existing problem since the time of Hippocrates, real surgical treatment was not attempted until nineteenth century when anesthesia was introduced to clinical practice. However, one of the first surgical attempts is the mythical vivisection case of 1474 in France. French Archer was condemned to death because of several larcenies. Unfortunately, Archer suffered with stone disease. Physician and surgeons of Paris were permitted by the King "to cut open the body of living man...to look at the sites where the disease ...formed." The opening and incision were done and, supposedly the stone was removed from the kidney. Archer "... was sewed up and his entrails placed back within him. Within fifteen days he was perfectly cured and his sentence was commuted, and with this he was given money."

Just like bladder surgery, renal stone surgery was condemned for centuries as uncertain, unlawful and unsuccessful, which halted its progress for a long time. The first planned open renal surgery to remove a stone was performed on October 8, 1872, by Dr. William Ingalls, a member of the surgical staff of Boston city hospital. Since then, multiple modifications of surgical stone removals were introduced to clinical practice. Surgery remained the only options for kidney and ureter stones until late twentieth century.

Modern Urinary Stone Surgery: Past, Present and No Suture!

The development and fast advances of minimally invasive procedures in the second half of twentieth century completely changed the treatment of both bladder and kidney stones. Electrohydraulic lithotripter, a device to break the stone inside the body, was invented by Lev Yutkin from USSR in 1955. While still a student in 1933, Yutkin was doing experiments with electricity under the water that once resulted in a small explosion and splitting of the bowl of water. The lightning under water for the first time demonstrated its ability to work.

Later Yutkin designed the first clinical device to break bladder stones called Urat-1. It was based on a creation of strong underwater spark that produces a shock wave to “hammer” the stone. The procedure was later modified with better and safer energy delivery systems. Subsequently, ultrasound and different type of lasers were introduced to fragment bladder, kidney and ureter stones.

Treatment of kidney stones was significantly advanced with the introduction of minimally invasive procedure to break kidney stones without surgical incision and prolonged hospital stay, called the percutaneous nephrolithotomy (PCNL). The first PCNL procedure was performed in 1974 by Fernstrom (radiologist) and Johansson (urologist). This procedure represents amazing convergence of several ideas throughout the history regarding access to the stone, breaking the stone and evacuation of the fragments. Coupled with multiple new devices including laser, ultrasound, pneumatic impactor, minimally invasive PCNL is the most commonly performed procedure for large kidney stone, also called staghorn calculi.

ESWL: Blast from the Past, the Wave of the Future!

Currently, large proportion of kidney and ureteral stones in the United States are managed with

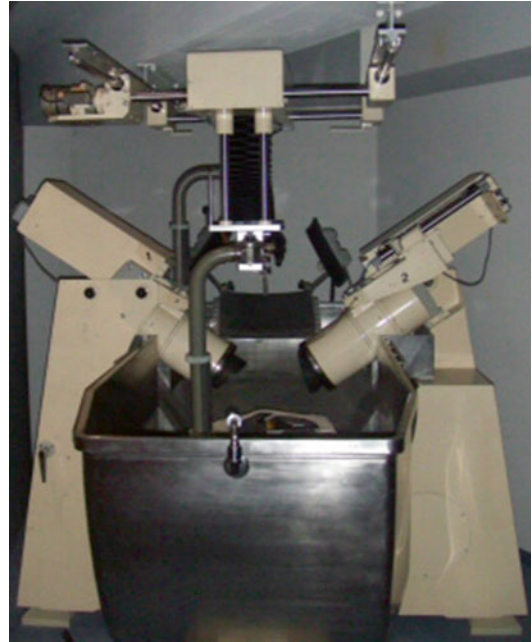


Fig. 4.1 Dornier HM1 (Courtesy of Dornier MedTech)

Extracorporeal Shock Wave Lithotripsy (ESWL). ESWL was a real breakthrough in the history of urinary stone treatment, a first true noninvasive procedure. The ESWL machine was produced by the German aerospace firm, Dornier. The company embarked on a program to develop a system of reproducible focused shock wave for the aircraft industry. This idea was then applied to the concept of kidney stone fragmentation (reportedly by suggestion of one of the engineer’s wife). In 1970, an intensive research program showed that focused shock waves sufficient for stone fragmentation could be created. In 1980, the first patient with a kidney stone was treated in Munich with a prototype machine called Dornier HM1 (Human Model 1, see Fig. 4.1) Lithotripter. In 1984, Dornier introduced the first commercially available machine, Dornier HM3 (Human Model 3). The Dornier HM-3 required the use of a water bath and an elaborate overhead gurney to position patients. Currently, ESWL machines instead use fluid filled “balloons” and gel placed

against the patient's body to allow transmission of shockwaves for treatment. Following Dornier's success, the lithotripter market expanded quickly with many different types and models of ESWL machines.

Conclusion

Treatment of urinary stones changed drastically over the centuries, from ancient medical treatment and primitive dangerous surgery to the sophisticated open surgery and latest minimally invasive procedures. New technological advances, targeted research and inquisitive minds

will certainly lead to the future discovery of new and even less invasive methods of stone treatment.

Selected Readings

1. Tefeki A, Cezayirli F. The history of urinary stones: in parallel with civilization. *Scientific World Journal*. 2013;2013:1–5.
2. Shah I, Whitfield HN. Urolithiasis through the ages. *BJU Int*. 2002;89:801–10.
3. Riches EW. Some landmark in surgery of stones. *BJU*. 1935;7:140–7.
4. Lingeman JE. Extracorporeal shock wave lithotripsy: development, instrumentation and status. *Urol Clin North Am*. 1997;24:185–213.

Scott Herfel and David A. Schulsinger

Simple Stone Facts

- **1 %** of all stones in adult patients are cystine.
- **2** liters of urine production per day is recommended for stone prevention.
- **3rd** most common disorder of the urinary tract is stones.
- **4** most common stones are calcium, uric acid, cystine and struvite.
- **5–10 %** of all stones are uric acid stones.
- **6** weeks is adequate time for most stones that will pass to do so.
- **7 %** of women will have a stone.
- **8mm** stones have a **20 %** chance of spontaneous passage.
- **9** out of **10** patients will be stone free after having ureteroscopy for a ureteral stone.
- **10–12 %** lifetime risk for stones in industrialized countries.

When it comes to kidney stones numbers play an important role. We are often faced with difficult questions like, “will my stone pass”? When will my stone pass? What happens if I do nothing? Am I a time bomb waiting for my stone to pass? Why am I making stones? What surgery is best for me?

The truth is we do not have all the answers to these questions. Even though some of these

questions may be difficult to answer, to tell patients that we do not have a crystal ball and that we cannot predict the future are not acceptable answers. What we can offer patients is the knowledge gathered from years of research, clinical studies, personal experience, and anecdotal evidence to best answer these difficult questions.

With all of our current knowledge about stone, we still have more to learn. The prevalence of stones does appear to be increasing and the gender gap is narrowing. What is the reason for this? Among other factors, environmental changes, diet, and obesity rates will all continue to be studied to guide patients and medical professionals in the future.

Like snowflakes, no two kidney stones are alike and therefore may behave differently. If you are unlucky enough to be a repeat stone former then personal experience is an important factor in determining how to best manage your stone and should always be taken into consideration by your physician. Other factors that play a major role in stone decision making are size, location, shape, texture and composition of the stone.

In this chapter, you will find facts and figures to help guide you along your personal stone experience. In addition, you will be presented with options to consider to prevent stone recurrences and the means to modify your entire stone experience. Remember if you have had a stone you are not alone. Millions of others have felt your pain. Their experiences are what will help you to

S. Herfel, PA • D.A. Schulsinger, MD (✉)
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

make your difficult decisions and potentially prevent stone formation in the future. Hopefully you will find solace knowing that your experience may help others in the future with their decision making.

Stone Facts

- 1 – 1 stone passage is all you need to have to know you don't want another.
- 1 – Stones account for 1 % of all hospital admissions.
- 1 – 1–2 % of all stones are cystine stones.
- 2 – Producing 2 liters of urine per day will decrease stone recurrence.
- 2 – Stones account for over two billion in medical cost annually.
- 2 – Almost two million out-patient visits for stones annually.
- 2 – PCNL is recommended for stones >2 cm.
- 3 – Stones are the third most common disorder of the urinary tract, behind urinary tract infections and prostate conditions.
- 4 – 4 most common stone types are calcium, uric acid, struvite and cystine.
- 4 – Stones <4 mm have and 80 % chance of spontaneous passage.
- 5 – Most stones <5 mm will pass spontaneously.
- 5 – Urine Ph<5.5 often associated with uric acid stones.
- 5 – 5 % of all stones in children are cystine.
- 5 – 5–10 % of all stones are uric acid stones.
- 6 – Vitamin B6 can be used to treat hyperoxaluria.
- 6 – Most stones that pass do so within 6 weeks of symptoms.
- 7 – Urine Ph>7 often associated with calcium phosphate and struvite stones.
- 7 – 7 % of women will have a stone.
- 8 – 8 mm stones have a 20 % chance of spontaneous passage.
- 8 – 8 % of stones contain uric acid.
- 10 – Stones larger than 10 mm are unlikely to pass spontaneously.
- 10 – Stones in the upper ureter at time of symptoms have a 10 % chance of spontaneous passage.
- 10 – 10–20 % of all kidney stones require surgical removal.
- 10 – 10–25 % of children with stones have an anatomical anomaly.
- 10 – 10–15 % of stones are struvite stones.
- 13 – Largest kidney stone ever recorded was 13 cm.
- 13 – Stones will affect 13 % of men.
- 16 – Up to 16 % of the population is affected by kidney stones in their lifetime.
- 20 – 8 mm stones have a 20 % chance of spontaneous passage.
- 20 – 20 % of patients with acute renal colic will require hospital admission.
- 20 – 20–25 % of children with a stone have or have had a urinary tract infection.
- 22 – Heaviest kidney stone ever recorded was nearly 22 oz.
- 24 – 24 hour urine collection is an important test to help determine risk factors for stone disease and decrease recurrence.
- 24 – 24 and 26 cm are the most commonly used stent lengths.
- 25 – William Shatner sold a kidney stone that he had passed for 25,000 with proceeds going to charity.
- 25 – 25 % of patients with uric acid stones have gout.
- 25 – Stones in the mid ureter at time of symptoms have a 25 % chance of spontaneous passage.
- 25 – 25 % of patients with kidney stones have a family history.
- 28 – 28 % of patients with a staghorn stone may have deterioration in their renal function.
- 35 – 35–45 years of age is the peak incidence of stones.
- 40 – Up to 40 % of stone formers will develop a second stone in 5 years.
- 40 – 40–50 % of children with stones have a metabolic abnormality.
- 49 – 49 % chance of an asymptomatic kidney stone becoming symptomatic in 5 years.
- 50 – 50 % of patients with an acute stone attack will have nausea and vomiting.
- 50 – Stones in lower ureter at the time of symptoms have a 50 % chance of spontaneous passage.
- 55 – 55 % of patients with recurrent stones have a family history.

- 60** – More than 60 % of patients presenting with renal colic will have stones within 3 cm of where the ureter enters the bladder.
- 60** – 60–90 % success with ESWL.
- 65** – Medical expulsive therapy accounts for 65 % greater chance of spontaneous stone passage.
- 75** – 75–85 % of children with stone have an identifiable underlying risk factor.
- 80** – 80 % of all stones are calcium stones, usually calcium oxalate.
- 80** – Stones <4 mm have and 80 % chance of spontaneous passage.
- 80** – 80 % lifetime risk of forming a second stone.
- 85** – 85 % of patients with renal colic will have microscopic or gross blood in the urine.
- 85** – Stones <3 mm have 85 % chance of stone passage.
- 90** – 90 % of patients presenting to an emergency department with a stone will have acute, unilateral flank pain, hematuria, and a positive plain film of the abdomen.
- 90** – 90 % stone free rates for ureteral stones treated with ureteroscopy.
- 90** – 90 % of symptomatic stones during pregnancy will present in the second and third trimester.
- 95** – Stone analysis combined with 24 hour urine can find a cause of stones in 95 % of patients.
- 95** – CT scan has 95–100 % sensitivity in detecting stones.

The Rule of 50!

When dealing with odds a 50/50 chance may sound great. Sure we would all like those odds in a casino. It turns out that when dealing with our health those odds may or may not sound so great. Below are 5 stone facts that have an approximate 50 % chance of occurring. You be the judge and see if you like these odds.

- 50 %** chance of stone recurrence in 5–10 years.
- 50 %** chance of spontaneous passage for a 5 mm stone.
- 50 %** chance of an asymptomatic kidney stone becoming symptomatic in 5 years.
- 50 %** of patients with an acute stone attack will have nausea and vomiting.
- 50 %** chance of spontaneous passage for stones in the lower ureter at time of symptoms.

Conclusion

In summary, personal experiences with stones will vary from patient to patient. One patient may be able to pass an 8 mm stone in the upper ureter. Another individual may require multiple trips to the ER for a 2 mm stone in the distal ureter and they may eventually require surgery. The reality is, the majority of stones will pass spontaneously and a smaller percentage will require surgical management. Let the facts and figures from this chapter guide you on your journey, which may at times be a “rocky” road. Remember that facts and figures are not etched in “stone” and statistics do not always tell the whole story.

Your urologist will convey with you a very important statistic after your stone passes or is treated. “Once you get a stone, you have a 50 % chance of getting another stone in 5–10 years and an 80 % risk in your lifetime.” Several questions travel through a patient’s head: What does this mean for me? If I flip the coin, how do I know if I wind up heads for stones and tails for no stones? As urologists, we do our best to beat the odds. We run a structured program to identify patients at risk for stones and provide the appropriate dietary, hydration and medical recommendations to minimize their risks for future stones. There are some patients who play by the rules of stone prevention. They hydrate well, they minimize their salt intake and consume reasonable levels of calcium. These patients had a stone more than 10 years ago and continue to remain stone free. These are the patients who beat the odds! These are the stone “outliers”. There are those patients who had an emergency treatment for their stone. The stone analysis reported a calcium stone. The plan is for the patient to follow up, do a 24 hour urine, and return to discuss the stone prevention protocol. However, these patients never return for their follow up

tests or visits. These patients wind up in the ER 1 year later with a new stone. These are the patients who are referred to as the “frequent flyers”! Then there is that small group of patients who had a stone who are completely responsible and compliant. They come to all their appointments, they obtain all their tests in a timely fashion, and they even request additional tests that were not originally ordered. Despite their best efforts, these individuals come back less than a year later with another stone. These are the “unfortunate”. Statistics help us provide information about the population, but may not always represent you, the individual. There are patients who do everything correct and reliably and still form stones; there are patients who do everything wrong, and never form another stone (assuming they come back to you)! Like many things in life, there are no guarantees. However, doing the “right thing” can aid in minimizing your risk of developing future stones. Keep in mind when it comes to stones we have outliers, frequent fliers and the unfortunate. The choices you make can help put you in the more favorable category. *Remember, statistics don’t always tell the entire story!*

Suggested Reading

1. Coe FL, Parks JH, Asplin JR. The pathogenesis and treatment of kidney stones. *N Engl J Med*. 1992;327:1141.
2. Teichman JM. Clinical practice. Acute renal colic from ureteral calculus. *N Engl J Med*. 2004;350:684.
3. Elton TJ, Roth CS, Berquist TH, Silverstein MD. A clinical prediction rule for the diagnosis of ureteral calculi in emergency departments. *J Gen Intern Med*. 1993;8:57.
4. Pearle MS, Calhoun EA, Curhan GC, Urologic Diseases of America Project. Urologic diseases in America project: urolithiasis. *J Urol*. 2005;173:848.
5. Scales Jr CD, Smith AC, Hanley JM, et al. Prevalence of kidney stones in the United States. *Eur Urol*. 2012;62:160.
6. Stamatelou KK, Francis ME, Jones CA, et al. Time trends in reported prevalence of kidney stones in the United States: 1976–1994. *Kidney Int*. 2003;63:1817.
7. Uribarri J, Oh MS, Carroll HJ. The first kidney stone. *Ann Intern Med*. 1989;111:1006.
8. Glowacki LS, Beecroft ML, Cook RJ, et al. The natural history of asymptomatic urolithiasis. *J Urol*. 1992;147:319.
9. Chandhoke PS. Evaluation of the recurrent stone former. *Urol Clin North Am*. 2007;34(3):315–22.
10. Hollingsworth JM, Rogers MA, Kaufman SR, Bradford TJ, Saint S, Wei JT, et al. Medical therapy to facilitate urinary stone passage: a meta-analysis. *Lancet*. 2006;368(9542):1171–9.
11. Milliner DS, Murphy ME. Urolithiasis in pediatric patients. *Mayo Clin Proc*. 1993;68:241.
12. Roudakova K, Monga M. The evolving epidemiology of stone disease. *Indian J Urol*. 2014;30:44–8.
13. Smith, J, Stapleton, FB. Epidemiology of risk factors for nephrolithiasis in children. In: UpToDate, Basow, DS (Ed), UpToDate, Waltham, MA, 2011.
14. www.guinnessworldrecords.com/world-records/4000/largest-kidney-stone

Urinary Tract Stones: *From the Invisible to the Clearly Distinct and Discernible Stone!*

6

David A. Schulsinger

Types of Stones: *The Good, the Bad and the Ugly!*

The human body can manufacture a variety of different stone types (See Fig. 6.1). The more common types of stones include calcium, uric acid, struvite and cystine. There are a variety of other stones that while unique, are rarely seen in clinical practice. This chapter will address some of the more common and unusual stone types.

Randall's Plaques: *Big Stones Have Small Beginnings!*

Dr. Alexander Randal demonstrated that stones begin from small calcifications that are present in the papilla of the kidney (Fig. 6.2). He referred to these small calcifications as Randall's Plaques. During modern stone surgery these are often seen but are too small to be treated. Over time as they grow they detach from the kidney and become true kidney stones. It is from this that we know that large stones have small beginnings.

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Calcium Stones

Simple Stone Facts

- Calcium oxalate is the most common type of urinary stone.
- Can be treated with surgical therapy.

Calcium containing stones are the most common types of stones formed within the urinary tract. They are most commonly composed of a mixture of calcium phosphate and calcium oxalate. Among the different calcium stones are calcium oxalate monohydrate, calcium oxalate dihydrate and calcium phosphate (Fig. 6.3). The etiology of calcium stone disease is very diverse.

The majority of patients with calcium stone disease will demonstrate hypercalciuria (40–75 %), which may be subdivided into one of three categories. These include absorptive hypercalciuria, renal hypercalciuria, or resorptive hypercalciuria. Other causes for calcium stones include hyperuricosuria (10–50 %), hyperoxaluria (8 %), and hypocitraturia (10–50 %). For a further detailed description of the metabolic factors associated with calcium stones, please refer to Chap. 8.

In 15 % of patients, calcium stones are secondary to a known underlying disorder. Primary hyperparathyroidism accounts for approximately 3 % of the calcium stone forming population. Less commonly, hereditary hyperoxaluria, renal

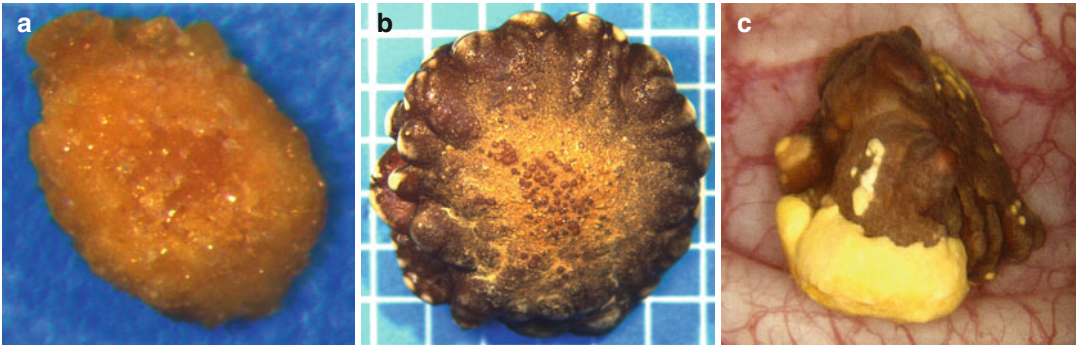


Fig. 6.1 (a) Uric Acid, (b) calcium oxalate monohydrate and a (c) mixed stone (50 % uric acid and 50 % calcium oxalate monohydrate)

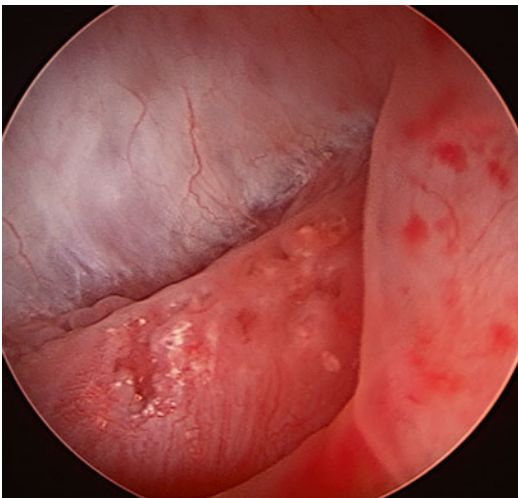


Fig. 6.2 Randall's Plaques in a renal papilla

tubular acidosis, sarcoidosis, Cushing syndrome, steroid treatment, Vitamin D intoxication, immobilization and medullary sponge kidney disease may be present. In the majority of patients, however, none of these disease processes are present and the stone disease is referred to as idiopathic or primary.

Treatment for calcium stones is surgical if the stones are too large to pass. A specific metabolic condition contributing toward calcium stone formation must be properly diagnosed so as to tailor the appropriate treatment for stone prevention.

Uric Acid Stones

Simple Stone Facts

- Most common cause of these stones is low urine pH.
- Associated with obesity, diabetes and metabolic syndrome.
- Treatment is medical therapy by alkalization of the urine.
- Radiolucent (not visible on plain x-ray) stones.

The incidence of uric acid calculi is approximately 5–10 % of all renal stones (Fig. 6.4). Men are affected four times more often than women. Approximately 25 % of patients with gout will form uric acid stones, however, the majority of patients who form uric acid calculi have no detectable abnormalities in uric acid metabolism. Factors that may contribute or predispose to uric acid stone formation are divided into three categories: (1) high urinary uric acid levels or hyperuricosuria; (2) low urinary volume; and (3) low urinary pH. Low urine pH is the hallmark of uric acid stone formers. This will be described in more detail in Chap. 8.

Treatment is tailored toward increasing urine volume and raising urinary pH. If hyperuricosuria is present, it can be corrected with appropriate dietary management and/or administration of medical therapy (see Chap. 19).

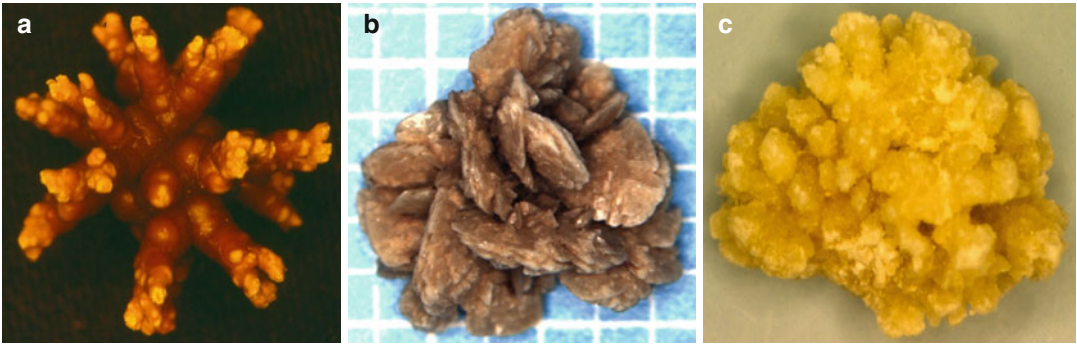


Fig. 6.3 (a) Calcium oxalate monohydrate, (b) calcium oxalate dihydrate and (c) calcium phosphate stones (Courtesy of Louis C. Herring & Co., Orlando, FL)

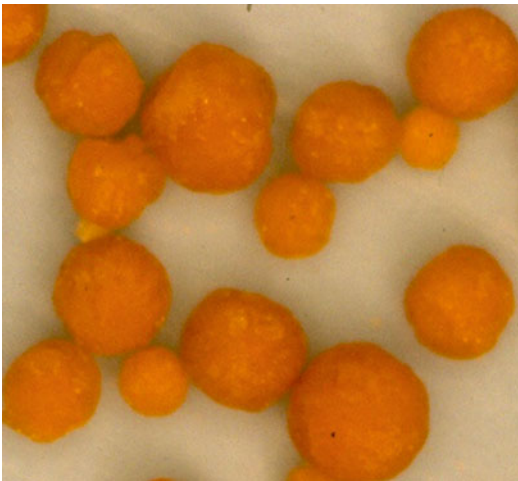


Fig. 6.4 Uric acid stones (Courtesy of Louis C. Herring & Co., Orlando, FL)



Fig. 6.5 Struvite stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

Struvite Stones

Simple Stone Facts

- Also called infectious stones or Magnesium Ammonium Phosphate stone.
- Stones are caused by urease splitting organisms.
- Primary treatment of stones is complete removal of stones and eradication of infection.
- Medical management is recommended in patients who have incomplete removal of stones or poor surgical candidates.

Struvite stones, commonly called infectious stones, are composed of magnesium ammonium and phosphate (Fig. 6.5). Struvite stones represent 10–15 % of all urinary tract stones. Historically, these stones made up the majority of staghorn calculi. Presently, 56 % of staghorn calculi are metabolic and 44 % are infectious stones. These stones occur more commonly in females with a female to male ratio of 2:1. This female gender predominance is believed to be due to a higher susceptibility to infection.

These stones are always associated with urinary tract infections with urease-splitting bacteria

(*Proteus mirabilis*, *Klebsiella Pneumonia*, *Pseudomonas* and less commonly, *Staphylococcus aureus* and *Staphylococcus epidermidis*). *E.coli* does not have urease and is does not cause struvite stones. Risk factors for these stones include neurogenic bladder, urinary diversion, urinary tract obstruction and chronic indwelling catheter.

Diagnosis of these stone are made by patients with recurrent UTI's, alkaline urine and demonstration of coffin-lid shaped crystals.

The mainstay of treatment for these stones is surgical. There is a 28 %, 10-year mortality with conservative care of these stones. Elimination of stones is essential for eradication of bacteria. The indications for stone removal include urinary tract infection, progressive renal damage, urinary tract obstruction and persistent pain. For large stones, percutaneous nephrolithotomy is the recommended treatment. Antibiotic suppression is indicated after stone clearance. Medical management with antimicrobial therapy, chemolysis (hemiacidrin), urease inhibition (acetohydroxamic acid) and urinary acidification helps to prevent stone recurrence. However, stone dissolution is rare and should be reserved for patients who are not surgical candidates. Medical therapy is only an adjunctive roll in the management of these patients and is directed primarily at controlling the urinary tract infection prior to, during, and subsequent to surgical removal in association with residual stone fragments after surgery. Close follow up of patients is recommended with frequent urine cultures.

Cystine Stones

Simple Stone Facts

- Autosomal recessive disorder.
- Impaired kidney and intestinal tubular transport of cystine.
- Goal of treatment is to reduce concentration of urinary cystine by increasing fluid intake, Alkalinization of urine and medication (chelating agents).

Cystine in the urine, or cystinuria, is a relatively rare autosomal recessive inborn error of metabo-



Fig. 6.6 Cystine stones (Courtesy of Louis C. Herring & Co., Orlando, FL)

lism, which is characterized by impaired reabsorption of four dibasic amino acids, Cystine, Ornithine, Lysine and Arginine (COLA), from the renal tubules as well as the gastrointestinal tract. Only the basic amino acid, cystine, is associated with stone formation due to its poor solubility. Patients with cystinuria (>250 mg/24 hour) produce supersaturated urine and are at risk for crystallization and stone formation. There is no clinical consequence of this disorder for ornithine, lysine and arginine.

Cystine stones represent 1–2 % of all urinary calculi (Fig. 6.6). The prevalence of cystinuria in the United States is 1 in 7,000 persons. It accounts for 6–8 % of pediatric stones. The age of onset is between 2 and 40 years. The median age for these stones to present in males and females is 12 and 15 years, respectively. Men average 0.42 stone events/year; women average 0.21 stone events/year.

Successful medical therapy in treatment of patients with cystine stones is based on three factors:

1. Decreasing total urinary cystine concentration
2. Increasing the solubility of cystine
3. Decreasing urinary cystine excretion. The chemoprevention of cystine stones includes hydration and urinary alkalinization, cystine-binding agents (D-penicillamine, alpha-mercaptopropionylglycine, captopril), and irrigation chemolytic therapy (tromethamin E, acetylcysteine). Successful medical manage-

ment can be achieved by a combination of these therapeutic measures. This includes adequate fluid hydration and fluid diuresis. Further details of the medical management of cystine stones are detailed in Chap. 19.

Xanthine Stones

Simple Stone Facts

- Caused by an inborn defect of xanthine oxidase.
- Can be caused by patients taking Allopurinol to treat uric acid stones.
- Stones are radiolucent.

Xanthine stones are very rare (Fig. 6.7). These stones may result from a genetic disorder in patients who have an enzyme defect of xanthine oxidase.

Normally, xanthine oxidase is necessary to oxidize xanthine to uric acid. With a deficiency of this enzyme, there is an increase excretion of hypoxanthine and xanthine. With xanthine being poorly soluble, xanthine stones can form.

Urinary calculi occur in about a third of patients with this enzyme deficiency. These calculi may also develop in patients taking allopurinol, a xanthine-oxidase inhibitor, used to treat patients with uric acid stones. Pure xanthine stones are radiolucent, but approximately a third of patients with xanthinuria, there may be a calcium salt mixture to render these stones slightly radio-opaque.



Fig. 6.7 Xanthine stone (Courtesy of Louis C. Herring & Co., Orlando, FL)



Fig. 6.8 2, 8-Dihydroxyadenine stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

2,8-Dihydroxyadenine (DHA) Stones

Simple Stone Facts

- An autosomal recessive disorder
- These stones do not appear KUB but will appear on renal US or CT scan

This is an extremely uncommon metabolic condition. This is an autosomal recessive disorder, in which a deficiency of the enzyme adenine

phosphoribosyltransferase (APRT) results in the conversion of adenine 2,8-dihydroxyadenine, a poorly soluble substance that readily crystallizes in urine. This results in DHA renal calculi (Fig. 6.8), which have been identified primarily in children who would appear to have no other manifestations of their disease. Similar to uric acid stones they are radiolucent. However, unlike uric acid calculi, they are much more soluble in acidic pH than in an alkaline pH.

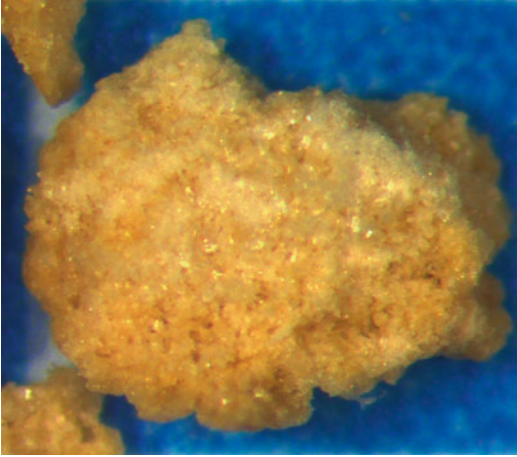


Fig. 6.9 Indinavir stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

Crixivan® Stones

Simple Stone Facts

- Caused by medication used to treat patients with HIV.
- These stones do not appear on renal US or CT scan without contrast, only present on CT scan with contrast.

Protease inhibitors are a class of medication used to treat patients with HIV disease. Crixivan® (indinavir sulfate), a protease inhibitor, is widely used to treat patients with HIV infections. Urinary nephrolithiasis has been associated with the use of Indinavir (Fig. 6.9). The risk of stone formation in patients on indinavir therapy resulting is 3–9 %. Radiographically, Indinavir stones are typically radiolucent. Abdominal CT scan demonstrated hydronephrosis without calcifications. CT scan with contrast may demonstrate the presence of Crixivan® stones.

The radiolucent-gelatinous nature of such stones makes lithotripsy a poor choice of treatment. Hyperhydration, acidification of urine and change of HIV medication are usually successful.



Fig. 6.10 Reyataz stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

Reyataz® Stones

Simple Stone Facts

- Reyataz® has an increased risk of stones, especially when prescribed with Norvir.

Reyataz® (atazanavir) is an HIV protease inhibitor, which is associated with the risk of kidney stones. Patients using Norvir (ritonavir)-boosted Reyataz® were ten times more likely to develop stones compared with patients taking other Norvir-boosted protease inhibitor based regimens [1]. The risk of stones was 7 % among individuals using Norvir-boosted Reyataz® (Fig. 6.10). This combination of medication should be carefully to individuals with risk factors for renal stones. Switching this medication to another antiviral treatment for patients with these stones is recommended due to the high risk of recurrence.

Topamax® Stones

Simple Stone Facts

- Topamax® used to treat seizures and migraines.
- Can increase the risk of calcium phosphate stones.

Topamax® (topiramate) is a medication used to treat seizure disorders and is a prophylactic treatment for migraine headaches. Topamax® can induce a systemic metabolic acidosis, lower urinary citrate and increased urinary pH. These changes increase the risk of calcium phosphate stones [2].

Conclusion

In summary, this chapter described the different types of stones that are manufactured by our body. These stones not only look different, but they are caused by different reason. As you will find in future chapters, the treatment for various stones is distinct and the prevention of these stones will differ. Stay tuned!

Recently, I saw a patient who returned to my office to review his stone analysis. Interestingly, the stone analysis was reported as “gypsum.” In my professional career, I had never seen a stone type with this composition before. I asked the patient

to detail for me how he collected the stone. He went into the graphic details of how the stone passed into the toilet and how he retrieved it. I asked him if there were any technical issues in retrieving the stone? He replied, “in fact there was. The stone had fallen onto the floor.” I asked him if there was any construction going on in his house? He replied, “Yes, we were installing dry-wall in the bathroom.” And there is your answer! The moral of the story is, keep your rocks to yourself and make sure you pick up the right rock!

References

1. Hamada Y, Nishijima T, Watanabe K, Komatsu H, Tsukada K, et al. High incidence of renal stones among HIV-infected patients on ritonavir-boosted atazanavir than in those receiving other protease inhibitor-containing antiretroviral therapy. *Clin Infect Dis*. 2012;55(9):1262–9.
2. Welch BJ, Graybeal D, Moe OW, Maalouf NM, Sakhaee K. Biochemical and stone-risk profiles with topiramate treatment. *Am J Kidney Dis*. 2006;48(4): 555–63.

Stone Characteristics: Not All Stones Are Created Equal!

7

David A. Schulsinger

Simple Stone Facts

- Different stone groups have a variety of stone characteristics.
- Variability also exists in stone subtypes.
- These characteristics are helpful in determining treatment strategies.

Like people, no two stones are the same. The color of the stone depends on the chemicals that make up the calculus. Most stones are yellow or brown, but they can also appear to be tan, gold or black. Stone shapes can be round, jagged or branched. Stones can also vary in size as small as crystals and as large as a tangerine. There are different characteristic among different stone types. This chapter will discuss stone composition and differences in fragility, stone size and symptoms, stone location and ability to pass and other characteristics.

Stone Characteristics (Shapes, Color and Sizes)

Calcium Oxalate

While calcium oxalate is the most common stone, it may present as calcium oxalate dihydrate,

calcium oxalate monohydrate or mixture of both stone types. While these two types of stones are both calcium oxalate, they vary significantly in their hardness and appearance. Just as coal and diamonds are both made of carbon, they vary in their hardness, shape and color. These two types of calcium oxalate stones also vary with their chemical and physical properties.

- **Calcium Oxalate Dihydrate**

These stones are also called Weddellite. These stones are typically very crystalline in nature. Also referred to as “jackstones”, these stones tend to be rough, uneven and jagged, similar to the seed of a sandspur (see Fig. 7.1). While

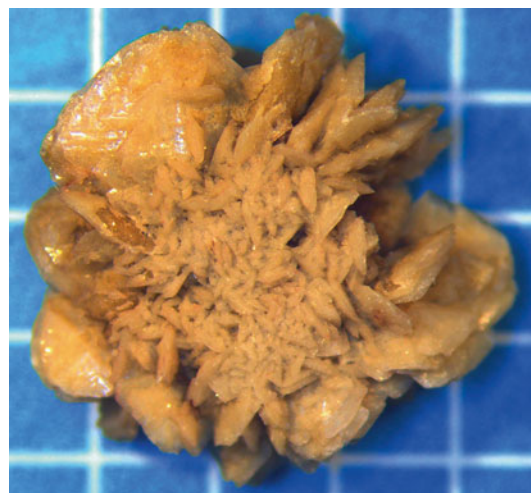


Fig. 7.1 Calcium oxalate dihydrate stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

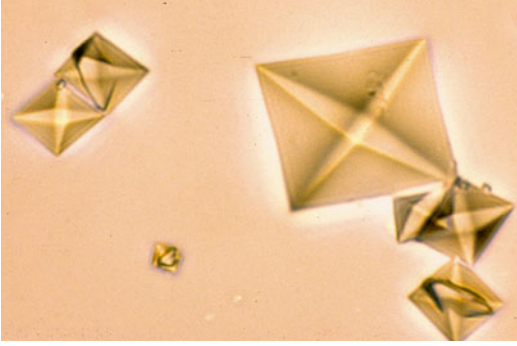


Fig. 7.2 Urinary calcium oxalate dihydrate crystals under polarized light microscopy (M. Daudon, CRISTAL Laboratory)

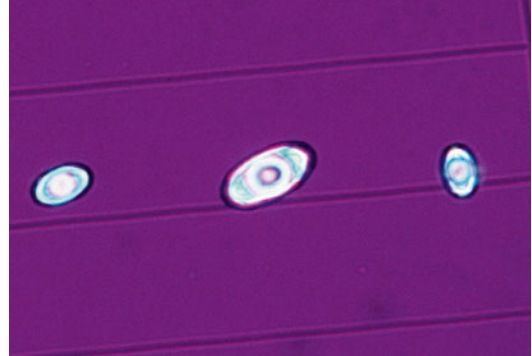


Fig. 7.4 Urinary calcium oxalate monohydrate crystals under polarized light microscopy (M. Daudon, CRISTAL Laboratory)



Fig. 7.3 Calcium oxalate monohydrate stone (Courtesy of Louis C. Herring & Co., Orlando, FL)



Fig. 7.5 Calcium phosphate stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

may have a spindle, oval or dumbbell shape (Fig. 7.4).

these stones are dense, they also break easily with lithotripsy. These stones are light tan in color. Calcium oxalate dihydrate crystalluria are envelope shaped (bipyramidal) on microscopic examination (Fig. 7.2).

- **Calcium Oxalate Monohydrate**

These stones are also called Whewellite. These stones are usually knobby shaped and have few to no spiked crystals on their surface (Fig. 7.3). These stones are typically dark brown in color. Similar to diamonds, these stones are very tough and do not break up with lithotripsy. Calcium oxalate monohydrate crystalluria vary in size and

Calcium Phosphate

These stones are also known as Hydroxyapatite. These stones are usually smooth and round (Fig. 7.5). The urine crystals of these stones are amorphous shaped found on microscopic examination (Fig. 7.6).

Uric Acid

These stones are usually orange-colored (Fig. 7.7). The urine crystals of these stones are diamond or rhomboid shaped discovered on microscopic examination (Fig. 7.8).

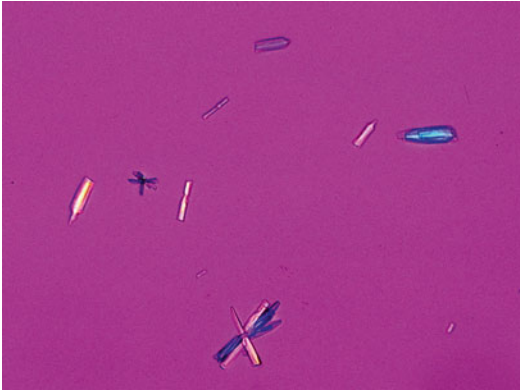


Fig. 7.6 Urinary calcium phosphate crystals under polarized light microscopy (M. Daudon, CRISTAL Laboratory)



Fig. 7.9 Struvite stone (Courtesy of Louis C. Herring & Co., Orlando, FL)



Fig. 7.7 Uric acid stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

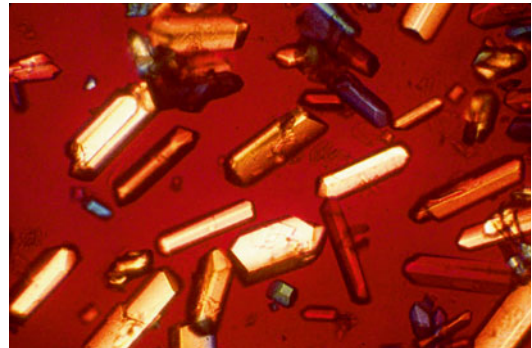


Fig. 7.10 Urinary struvite crystals under polarized light microscopy (M. Daudon, CRISTAL Laboratory)



Fig. 7.8 Urinary uric acid crystals under polarized light microscopy (M. Daudon, CRISTAL Laboratory)

Struvite

These stones are also known as magnesium ammonium phosphate or infection stones. These stones can become very large, called staghorn cal-

culi. These stones are tan in color (Fig. 7.9). The urine crystals of these stones are coffin-lid shaped identified on microscopic examination (Fig. 7.10).

Cystine

These stone are amber in color (Fig. 7.11). The urine crystals of these stones are hexagon “stop sign” shaped seen on microscopic examination (Fig. 7.12).



Fig. 7.11 Cystine stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

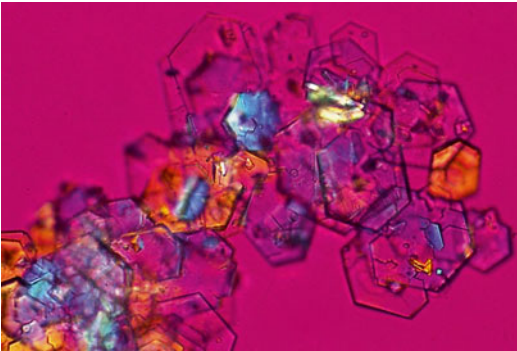


Fig. 7.12 Urinary cystine crystals under polarized light microscopy (M. Daudon, CRISTAL Laboratory)

Stone Composition and Fragility

Of the six major stone types (calcium oxalate monohydrate, calcium oxalate dihydrate, calcium phosphate, uric acid, cystine and struvite), not all stones are easily broken. Fragility refers to the ability of a stone to fragment by various treatment techniques. Different types of stones are thought to have characteristic fragilities and variations in the fragility exist within some types of stones. Stones that are the most to least ESWL-resistant in descending order are cystine, brushite, calcium oxalate monohydrate (COM),

hydroxyapatite, struvite, calcium oxalate dihydrate (COD), and uric acid. Because of these differences, treatment is tailored to the type of stone. See Chap. 20 on additional treatment options of stone disease.

Stone Etiology

The etiology of stone formation can be classified into those produced by infection, no infection and those arising from genetic defects. Stones caused by infection include magnesium ammonium phosphate, apatite and ammonium urate. Stone not caused by infection include calcium oxalate, calcium phosphate and uric acid. Finally, stones caused by genetic factors include cystine, xanthine and 2,8-dihydroxyadenine.

X-ray Characteristics of Stones

Some stones are radiolucent, meaning they are not visible on an abdominal x-ray. These include uric acid, ammonium urate, xanthine and 2,8 dihydroxyadenine stones. Some stones are radiopaque, in which they are visible on x-ray. These stones include calcium oxalate dihydrate, calcium oxalate monohydrate and calcium phosphate. Some stones are poorly radiopaque and these include magnesium ammonium phosphate, apatite and cystine stones. The majority of stones are visible on renal ultrasound and spiral CT scans (CT scan without contrast), with the exception of Crixivan stones, which are only apparent on CT scan with contrast.

CT scan is also used to help differentiate between different stone types based on Hounsfield units (HU), a quantitative scale to measure radiodensity. Several studies have shown that the HU of calcium stones are much greater than the HU of other stone types. Studies showed that calcium phosphate stones have the highest HU [1, 2]. Due to the difference in HU, calcium phosphate stones can be distinguished from struvite [2]. Other studies showed difference in HU between different groups of stones (calcium oxalate > struvite > uric acid) [3]. Differences in HU

between the various subtypes of calcium stones were also appreciated (calcium phosphate > calcium oxalate monohydrate > calcium oxalate dihydrate) [1]. For additional information on stones and imaging, refer to Chap. 16.

Urinary pH and Stone Formation

The formation of various types of kidney stones is strongly influenced by urinary pH. An alkaline pH promotes the crystallization of calcium phosphate stones ($\text{pH} > 6.3$). Acidic urine pH favors uric acid ($\text{pH} < 5.3$) or cystine stones ($\text{pH} < 7$). Struvite stones require a unique set of urinary conditions to grow: a high urinary pH and high levels of ammonium. This occurs only when the urine is infected with urease-producing bacteria such as a proteus species. Some stones, however, are pH independent, such as calcium oxalate stones. *Remember, pH can be a slippery slope for stone formation!*

Stone and Gender

The prevalence of stones favors men with up to 12 % of males and 6 % of females developing stones in their lifetime. The one stone type that favors women, are struvite stones by 2:1 ratio. See Chap. 10 for more differences between men and women related to stones.

Symptoms Associated with Stones

Location

Different types of calculi can present with similar symptoms, while similar stones can present with different symptoms. The location and quality of pain is characteristic to the position of the stone within the urinary tract. For example, pain associated with a calculus in the proximal (upper) ureter (Fig. 7.13) tends to radiate to the flank and lumbar regions. Mid-ureteral stones cause pain that radiates anteriorly (front side) and caudally



Fig. 7.13 Ureteroscopic view of stone in the upper ureter

(lower). Distal ureteral stones cause pain that radiates into the groin or testicle in men or the labia majora in women as the pain is referred from the stimulation of ilioinguinal or genitofemoral nerves in that region.

Size

The size stones can vary and the symptoms associated with these stones can differ too. Very small stones made in the kidney may be small enough to not obstruct the urinary tract and may pass without any symptoms.

For stones that are large enough to obstruct the urinary tract, patients can present with the characteristic symptoms. The classic presentation for a patient with acute renal colic is the sudden onset of severe pain originating in the flank and radiating inferiorly and anteriorly. At least half of patients will also have nausea and vomiting. Patients may also present with hematuria.

For the stones that are very large (Fig. 7.14), referred to as staghorn calculi, they occupy most of the renal pelvis and ironically, may be asymptomatic. These staghorn calculi are too large to move, obstruct or block the urinary

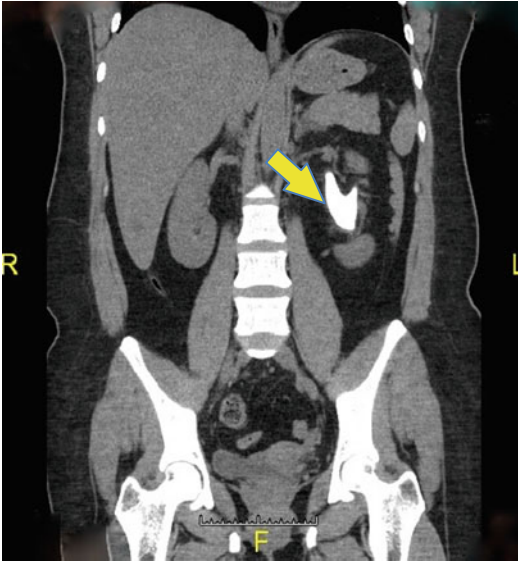


Fig. 7.14 Coronal CT scan image demonstrating a stag-horn calculus (yellow arrow) occupying much of the left renal pelvis

tract. Just as water can get into our basement, urine will seek it's lowest level too. Urine typically finds a way to move between the stone and walls of the kidney to make its way down the urinary tract. Small stones, on the other hand, are more likely to block the pipes resulting in a back up of urine within the kidney. The resulting swollen kidney, or hydronephrosis, causes the kidney to dilate, resulting in flank pain. Please refer to Chap. 14 for additional information on stones and symptoms.

Stone-Passage Rates: Size Does Matter!

The narrowest portion of the urinary tract is the ureter. Smaller stones have a better chance of spontaneous passage than larger stones. There is 68 % stone passage rate for stones less than 5 mm compared to 47 % if the stone is greater than 5 mm [4].

In addition, smaller stones are more likely to pass sooner than larger stones. For stones that are <2 mm, 2–4 mm and >4–6 mm, 50 % of these stones will take 8.2 days, 12.2 days and 22 days, respectively to pass. For 95 % of these stones to pass, it will take 31 days, 40 days and 39 days, respectively [5].

Conclusion

In summary, there are many characteristics that separate one stone subtype or group from another. Differences exist between stone types and even among the same type of stone. Your urologist will work with you to determine your stone type, and ultimately, options for treatment and stone prevention. Remember, not all stones are the same!

Ms. SM is a morbidly obese patient (BMI=41) who presented with recurrent UTI over many years. Her primary care physician treated her with multiple therapeutic courses of antibiotics, treating 6–12 UTI's/year. Undergoing a CXR for a pre-admission testing work up prior to a gynecologic procedure, a large right renal stone was identified. After that procedure she followed to urology clinic and a CT scan revealed a 5.1 cm right renal stone within the pelvis of the kidney. She never presented with symptoms of nausea, vomiting, fever, chills or flank pain. Imaging studies demonstrated no evidence of obstruction. She ultimately underwent definitive therapy of her stone and now remains stone free. Remember that stone size does not always correlate with stone symptoms!

References

1. Patel SR, Haleblan G, Zabba A, Pareek G. Hounsfield units on computed tomography predict calcium stone subtype composition. *Urol Int.* 2009;83(2):175–80.
2. Aticia I, Voyvodab N, Tokgoza O, Tokgoz H. The efficiency of non-contrast computed tomography in the estimation of urinary stone composition. *World J Nephrol Urol.* 2012;1(1):23–8.
3. Demirel A, Suma S. The efficacy of non-contrast helical computed tomography in the prediction of urinary stone composition in vivo. *J Int Med Res.* 2003;31(1):1–5.
4. Preminger GM, Tiselius HG, Assimos DG, et al.; American Urological Association Education and Research, Inc; European Association of Urology. 2007 guideline for the management of ureteral calculi. *Eur Urol.* 2007;52(6):1610–31.
5. Miller OF, Kane CJ. Time to stone passage for observed ureteral calculi: a guide for patient education. *J Urol.* 1999;162(3 Pt 1):688–90; discussion 690–1.

Part III

Risk Factors, Risk Groups

Metabolic and Hereditary Factors: Are You Stone Prone?

8

Merrit Debartolo and David A. Schulsinger

Simple Stone Facts

- Majority of patients with stone disease have hypercalciuria.
- Hypocitraturia is the second most common metabolic defect.
- Causes for hyperoxaluria are congenital, dietary consumption and gastrointestinal factors.
- Remember that dehydration is the most common factor for all stones.

Does the adage, “the apple doesn’t fall far from the tree,” apply to kidney stones falling from the kidney? It does, but the reason for this actually has much more to do with environmental than genetic factors. After all, children in families share a home environment with their parents, and they tend to eat and drink the foods and fluids that their parents have available. This is particularly important because **dehydration** is the most common cause of urinary tract stones. Fluid consumption (or the lack thereof) is the main determinant in stone-forming tendencies. Some innate genetic factors do influence metabolism to make stone formation more likely. These include, in order of decreasing prevalence: excess calcium in the urine (**hypercal-**

ciuria, 62 %), deficient urinary citrate (**hypocitraturia**, 41 %), excess urinary urate (**hyperuricosuria**, 35 %), excess urinary oxalate (**hyperoxaluria**, 8 %), decreased urinary magnesium (**hypomagnesiuria**, 1 %), and increased urine levels of the amino acid **cystine** (**cystinuria**, 1 %). If it looks like the math doesn’t add up, that is because these conditions can and do coexist in stone-formers. In this chapter, the metabolic conditions and hereditary factors contributing to stone formation are discussed in detail.

Hypercalciuria

Hypercalciuria is the most common metabolic factor associated with stone disease, accounting for 62 % of all cases. The three types of hypercalciuria are absorptive, renal and resorptive. In *absorptive* hypercalciuria, where gastrointestinal absorption is the root cause, sometimes the urinary calcium can normalize by limiting dietary calcium and avoiding such high-calcium indulgences as milk, cheese, yogurt, and leafy green vegetables. Limiting dietary calcium, however, may not be enough to normalize the urinary calcium concentration, in which case a healthcare provider may prescribe the medication **hydrochlorothiazide**, a medication that inhibits secretion of calcium into the urinary tract. Medications will be discussed further in Chap. 19.

In *renal* hypercalciuria (also called “renal leak”), the kidney is intrinsically unable to

M. Debartolo, MD • D.A. Schulsinger, MD (✉)
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: merritmd@gmail.com;
endourology@yahoo.com

reabsorb filtered calcium; therefore, calcium filtered by the kidneys into the urine stays in the urine instead of being reabsorbed. This results from mutations in genes coding for the channels through which the kidney reclaims calcium. Because it addresses the underlying issue of calcium transport out of the urine back into the kidney, hydrochlorothiazide is particularly effective in cases of renal hypercalciuria.

In *resorptive* hypercalciuria (also called primary hyperparathyroidism”), the excess urinary calcium is the result of another disease – most commonly, disorders of the **parathyroid glands** that regulate calcium concentration in the blood by effecting breakdown of bones for minerals. Certain hormonal syndromes called **Multiple Endocrine Neoplasias (MEN)** can lead to increased activity of the parathyroid glands, which in turn increases calcium concentration by increasing bone breakdown. In addition, other diseases marked by widespread inflammation (**systemic lupus erythematosus, sarcoidosis**) can increase urinary calcium concentration. Such catabolic steroids as **prednisone** can increase urinary calcium as well. Lastly, certain **cancers** such as those of the breast and lung can increase the body’s calcium by bone breakdown. In these cases, targeting and treating the underlying condition represents the surest way to normalize urinary calcium and stave off calcium stone formation.

Hypocitraturia

The next most common identifiable abnormality is a deficiency of the buffering ion **citrate**, which is naturally found in citrus juices (hence the name). Normally, citrate binds to calcium to form a soluble complex, thus rendering calcium unable to form urinary tract stones. Patients with low urinary citrate, however, do not enjoy this protective effect: their urinary calcium is free to form insoluble complexes with oxalate and phosphate, which in turn precipitate to form urinary tract stones.

An important cause of low urine citrate is a low body pH (**acidosis**), which can result from

the kidney’s intrinsic inability to eliminate acid in the urine – a condition called **renal tubular acidosis**. The body restores its pH in these conditions by retaining citrate as a buffer, preventing its elimination in the urine. The lack of citrate in the urine, in turn, leads to urinary tract stones in the manner previously described. Patients with these conditions can still enjoy the protective effects of citrate, thanks to the medication **potassium citrate (Urocit-K)**, described more comprehensively in Chap. 19.

Hyperuricosuria

Hyperuricosuria, or high urinary uric acid levels are seen in 35 % of patients with stones. In patients with gout, blood cell cancers (*e.g.* lymphoma), disorders of amino acid metabolism (such as **Lesch-Nyhan Syndrome**), or overindulgence in purine-rich products (*e.g.* beer, red meat, poultry) an excess of urate in the urine leads to urinary tract stone formation.

Urate is a “double-whammy” in terms of urinary tract stone formation, in that it not only precipitates out of the urine to form **uric acid** stones directly, but also decreases the pH of the urine to increase the likelihood of *calcium-based* stone formation. Additionally, through a process called **heterologous nucleation** in which insoluble precipitates propagate a chain reaction of urinary solute crystallization, urate causes stone formation even independently of pH. Thus, in these ways hyperuricosuria fosters a very stone-friendly environment in the urinary tract. Luckily, through mechanisms discussed further in Chap. 19, **potassium citrate** targets both of these causes to decrease stone formation. If this proves insufficient, the medication **allopurinol** can, as described in Chap. 19, decrease uric acid formation.

Hyperoxaluria

Oxalate is an organic anion naturally found in nuts, tea, chocolate, spinach, rhubarb, and dry beans. In the urine, it binds with urinary calcium

to form **calcium oxalate** stones – the component most commonly found in urinary tract stones. Urinary oxalate may reflect an inability of the gastrointestinal tract to absorb fats (as in Crohn’s Disease, or other inflammatory bowel diseases), an inability of the liver to metabolize particular amino acids, dietary overindulgence in oxalate-rich products, or modification of oxalate-consuming bacteria that normally live in the colon.

In situations where gastrointestinal inflammation impairs the absorption of dietary fat, the fat binds calcium in a process called **saponification**. Since calcium now binds to the unabsorbed fat rather than the oxalate, this leaves dietary oxalate un-complexed by calcium and, since oxalate no longer has a “dance partner,” it is now available to be absorbed into the blood from the gastrointestinal tract. From there, the oxalate is filtered by the kidneys into the urine, where it may form urinary tract stones. Aside from treating the underlying inflammatory bowel disease, the treatment for this condition is **calcium supplementation**, as discussed in Chap. 19, to reunite oxalate with its “dance partner.”

When excess urinary oxalate reflects the liver’s intrinsic inability to metabolize particular amino acids, a congenital disorder, it is called **primary hyperoxaluria**. Unfortunately, the only definitive way to treat this congenital liver enzyme deficiency is with a combined liver and kidney transplantation.

When urinary oxalate reflects overindulgence in the aforementioned oxalate-rich products, dietary modification to reduce oxalate intake normalizes the urine’s oxalate concentration. Lastly, in patients on chronic oral antibiotic therapy (*e.g.* patients with cystic fibrosis), the oxalate-consuming bacteria endemic to the gastrointestinal tract (*Oxalibacter formigenes*) are killed, increasing the amount of absorbed oxalate that eventually reaches the urine. In these cases, modification of antibiotic therapy and/or addition of probiotic therapy may need to be considered if feasible. These options are also discussed in Chap. 19.

Hypomagnesiuria

Identified in 1 % of stone formers, low urinary magnesium leads to stone formation because magnesium normally competes with calcium for binding with organic anions; without magnesium, oxalate and other ions are free to form insoluble complexes with calcium that precipitate into urinary tract stones. Not surprisingly, the treatment is increased magnesium intake either in the form of medicine (**magnesium oxide**, **magnesium citrate**, described in Chap. 19) or magnesium-rich foods (bananas, apricots, soy products; described in Chap. 27).

Cystinuria

Another rare (1 %) cause of recurrent urinary tract stones, particularly in children, is **cystinuria**. In this condition, the kidney is intrinsically unable to reabsorb dibasic amino acids. Specifically, the amino acid **cystine** from dietary proteins accumulates within the urine until it eventually precipitates into hexagonal crystals (Fig. 8.1). This tends to occur at a threshold concentration of >300 mg/L.

The first line of treatment for this condition (or, for that matter, ANY affliction of a urinary tract stone former) is **copious hydration** and **dietary sodium limitation** so as to minimize the concentration of urinary solutes that might precipitate into stones. Also, dietary protein limitation will reduce the amount of

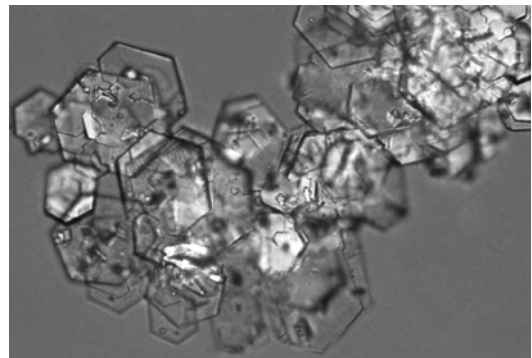


Fig. 8.1 Urinary cystine stone crystals (M. Daudon, CRISTAL Laboratory)

substrate for urinary cystine, thus reducing urinary cystine. In some of the more fortunate cystinurics, these measures may suffice. If hydration alone fails to reduce urinary cystine concentration below its solubility threshold, raising the urine pH to at least 7.5 with **potassium citrate (Urocit-K)** can dissolve the cystine by bringing the urine's pH closer to its solubility point pK.

Patients who remain cystinuric despite the above measures may need a **chelating agent** to solubilize their urinary cystine. These are discussed further in Chap. 19.

Summary

Urinary tract stones represent the quintessential “nature and nurture” duality in that stone formers can and do have conditions predisposing to stones, but the most important factor in stone formation, dehydration, is a predominantly environmental consideration. Nevertheless, an individualized approach to stone treatment that addresses patients' individual metabolic and genetic nuances is the most likely to be successful. *Remember, some people are stone prone!*

Ms. UA is a 65-year-old woman with a history of gout and urinary tract stones. She stated that she had passed many “golden yellow colored stones” for several years, the last of which was found to be uric acid. Two years ago, she learned that her daughter was pregnant with her first grandchild. However, shortly thereafter she received the also life-changing but less joyous news that she had breast cancer. Wanting to be there for her soon-to-be grandchild, Ms. UA took a more proactive approach to her health: she underwent a left mastectomy and chemotherapy for breast cancer, started taking her gout medicine and potassium citrate prescribed by her primary doctor and urologist respectively, then through vigorous diet and exercise lost 50 lb of excess weight. Now, having beaten cancer and remained stone-free for 2 years, she triumphantly declares she is in “the best shape of my life.” She can't help but smile and have a positive outlook as she gets a high-five from her adorable 15 month-old grandson. Now when life presents her with adversity, her response, influenced no doubt by her struggle with stones, is: “This too shall pass!”

David A. Schulsinger

Summary Stone Facts

- There are risk factors for stones that we can control: dehydration, diet, obesity, medicines that cause stones.
- There are risk factors for stones that we cannot control: age and gender, family history of stones, history of UTI's, diseases (i.e., Inflammatory Bowel Disease, hyperparathyroidism), Insulin resistance from diabetes.

There are a host of factors that contribute to stone formation. They include environmental, occupational, lifestyle, gastrointestinal, medical disease, hormone, surgical, medication, family history and ethnicity factors. This chapter will discuss climate and other factors favoring stone formation. The following factors will be discussed in this chapter:

Climate Factors: *The Stone Belt*

People who live in dry, hot climates are at a higher risk of developing kidney stones. The hot environment results in low urine volume either as

a result of inadequate fluid going into the body or the body losing fluid too quickly, resulting in dehydration. This, in turn, increases the stone risk by increasing the concentration of stone-forming mineral salts. Stones are more common in the South than in the Northern United States. These high risk areas are referred to as the Stone belt or kidney stone belt. In the U.S., the kidney stone belt includes Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, South Carolina and Tennessee. It is believed that the stone belt zone encompassed 40 % of Americans in 2000 and will expand to include 56 and 70 % of American population in 2050 and 2095, respectively [1]. Other studies also have documented a positive relationship between high ambient temperature and the development of renal stones [2, 3].

In New York, we see more stones in the summer time. In the northeast, where I practice Urology, I tell my patients that summer is kidney stone season. During the summer months, the temperatures soar, people are more active, they perspire more and dehydrate frequently. As a result, the seasonality of stones becomes more prevalent.

In summary, kidney stones vary by season and region with a greater prevalence in warmer temperatures. Stones are more common in warmer regions and geographic locations during hotter times of the year. For summers in New York, it must be stone season!

D.A. Schulsinger, MD
Department of Urology, Stony Brook
Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Occupational Factors

Workers exposed to high temperatures demonstrated a ninefold increase risk of nephrolithiasis. Risk factors include daily fluid intake, stress level, activity level, medical condition and family history. The metabolic alterations contributing to stone disease were hypocitraturia and low urine volumes. Health care professionals demonstrated a 14.6 % and physicians working in the operating room had a 17.4 % prevalence of stones [4]. Other occupations associated with risk for stone disease include steel workers, painters and outdoor workers.

Lifestyle Factors

Immobilization

Any medical condition or physical circumstance that keeps an individual bedridden or immobilized for extended periods of time can cause demineralization of bones. Ultimately, this increases blood levels of calcium in the blood and urine which poses an increased risk for stone formation.

Stress

Patients who have had a major, stressful life experience (either positive or negative) during a 2 year period prior to diagnosis of a kidney stone were more likely to develop stones than those who had not had a stressful experience [5]. Some investigators speculate that the increased risk may be due to vasopressin, which is released in response to stressful conditions. Vasopressin also increases the concentration of urine by reducing the urine volume, thereby allowing chemicals in the urine to form crystals and stones.

Gastrointestinal Factors

Inflammatory Bowel Disease (*Crohn's Disease and Ulcerative Colitis*)

Ulcerative colitis causes chronic inflammation in the inner lining of the large intestines. Crohn's

disease causes chronic inflammation that extends into deeper areas of the intestinal wall and can affect any part of the gastrointestinal tract. Patients with irritable bowel disease have a 10–100 times risk of forming stones compared to the general population, forming calcium oxalate or uric acid stones [6]. These patients have fat malabsorption. Calcium binds with fat, leaving oxalate free to be absorbed resulting in hyperoxaluria and calcium oxalate stones are formed. Treatment includes increased fluid intake, low oxalate diet and calcium supplementation to bind oxalate in the intestinal tract. Patients with irritable bowel disease develop uric acid stones as a result of low urine volume and decreased urinary citrate levels and acidic urine. Treatment includes correction of diarrhea, hydration and potassium citrate.

Diarrhea

Large losses of fluid from the gastrointestinal tract can lead to dehydration along with losses of bicarbonate and potassium. This leads to smaller urine volumes and low urinary citrate levels and as a result, both calcium and uric acid stones can form. Therapy is directed to decrease gastrointestinal fluid losses, increase urine volume and supplement with oral citrate therapy. These stones are often very difficult to prevent without decreases in diarrhea losses.

Medical Disease Factors

Sarcoidosis

Sarcoidosis is an autoimmune disorder, which results in abnormal growth of lymph nodes particularly in the chest around the lungs. This is most common in African Americans, but does occur in all ethnic groups. People with this disorder produce vitamin D in the lymph nodes resulting in increased absorption of calcium from the diet. The increased calcium absorbed from food is excreted in the urine. This raises urine calcium levels causing calcium crystals and ultimately, kidney stones to precipitate and form. Treatment for this disorder often requires prednisone.

Diabetes

Individuals with type 2 diabetes have a low urine pH (acidic urine) that can lead to kidney stones, including uric acid stones.

Kidney Disease

Medullary Sponge Kidney

Medullary sponge kidney (MSK) is a renal malformation that is associated with nephrocalcinosis (calcifications within the parenchyma of kidney) and nephrolithiasis (renal stones in the collecting system). MSK affects 1 in 5,000–20,000 individuals in the United States [7]. Up to 20 % of individuals who have stones also have MSK [8]. These patients may also develop hematuria and urinary tract infections. In medullary sponge kidney, these tubules become dilated. Radiographically, the kidney takes on a spongy appearance. Crystals form in these dilated tubules, which lead to the formation of kidney stones. In some, the medulla contains an abundance of calcium crystals. The reason for crystal formation may be due to stasis of urine in the tubules. In addition, the changes in the tubules may lead to increased calcium excretion into the urine (hypercalciuria) and renal tubular acidosis. Medullary sponge kidney can affect one kidney more than the other. In some patients stones seem to only occur on one side.

Treatment for patients with MSK is to maintain adequate fluid hydration to reduce the risk of stone formation. In patients with low citrate levels, treatment with potassium citrate is important. Finally, patients with UTIs should be treated with the appropriate antibiotics.

Cancer

Leukemia

Leukemia is tumor of white blood cells, resulting in an increase of abnormal cells. This tumor is associated with high levels of urinary uric acid, which form into stones, typically uric acid stones. Treatment includes decreasing the levels of white blood cells, maximizing water/fluid consumption, and possibly allopurinol and bicarbonate/citrate therapy.

Lymphoma

Lymphoma is cancer affecting the lymphatic system. The two types of lymphoma are the rare, Hodgkin's lymphoma and the more common, Non-Hodgkin's lymphoma. Patients with lymphoma have an increased risk of kidney stones.

Hypertension

Individuals with high blood pressure are up to three times more likely to develop nephrolithiasis. It is not completely clear as to whether having high blood pressure increases the risk for a stone, whether having stones lead to high blood pressure, or whether there is a factor linking both conditions.

Chronic Urinary Tract Infections

Urinary tract infections (UTI) are almost always the cause of struvite stones. Women are more likely to develop struvite stones than men.

Urinary Stasis

The incidence of urinary tract stones is greater in patients with an anatomical abnormality of urinary system, which may result in urinary stasis [9]. Urinary tract obstruction increases the risk of urinary tract stones. Bladder outlet obstruction secondary to an enlarged prostate increases the risk of bladder stones. Obstruction of the ureteral pelvic junction increases the risk of renal calculi. Other conditions associated with urinary stasis include neurogenic voiding dysfunction and urinary diversion. The risk of urinary stone formation is 4–48 % among patients with urinary diversion. Urinary stones occur three times more commonly in continent than in non-continent urinary diversion. Risk factors are urinary stasis, mucus production, urinary tract infections, clips, non-absorbable suture material, need for catheterization and metabolic disorders. Patients with urinary diversion are more likely to form magnesium ammonium phosphate stones (infection stones).

Gout

Gout is a condition that results in high uric acid levels in the blood. Gout is a painful form of

arthritis that occurs when uric acid in the blood precipitate into crystals in one or more joints. This leads to intense pain and inflammation. In patients without kidney disease or gastric ulcers, Gout is typically treated with non-steroidal anti-inflammatory (NSAID's) medications such as Motrin (ibuprofen), Indocin (indomethacin), or Celebrex (celecoxib). If NSAID's are contraindicated, people can sometimes take colchicine or steroids (i.e., prednisone). With very high uric acid levels or recurrent gout attacks, patients can be treated with allopurinol (Zyloprim), lowering uric acid levels.

Uric acid and other calcium stones develop in up to 25 % of patients with primary gout. The most common cause for these stones is a low urine pH (i.e., acidic urine), resulting in uric acid to precipitate in the urine. This can occur even if the uric acid levels in the urine are not elevated. Other causes for these stones are dehydration and over indulgence of purine containing foods. The basis of treatment is similar to patients without gout. The goal of treatment is to raise the pH of the urine (i.e., basic pH) to dissolve these stones. This can be accomplished with citrate. Gout patients may also require allopurinol and a low purine diet to lower uric acid levels. As with all stones, hydration is important too.

Renal Tubular Acidosis

This disorder can develop as a consequence of certain syndrome diseases such as Sjogren's Syndrome. There are also genetic causes (See Chap. 8). Distal Renal Tubular Acidosis, or RTA Type I, causes an acid and alkaline imbalance. People with this disorder cannot pump acid, which builds up in the body. This results in a hypokalemic, metabolic acidosis. This results in increases of calcium levels in the bloodstream. This also reduces the protective citrate levels in the urine. These patients cannot acidify their urine, resulting in a high urine pH (i.e., basic), increasing the risk of calcium phosphate stones to form. Treatment for this disorder includes bicarbonate or potassium citrate therapy to reduce the acidity in the body.

Hormones

Hyperparathyroidism

The parathyroid glands produce parathyroid hormone, which regulates the calcium levels in the blood. Overactive parathyroid glands cause about 5 % of calcium stones. Individuals with hyperparathyroidism have at least a 20 % chance of developing kidney stones. Patients typically develop calcium phosphate stones. Women are more likely to have this disorder than men. Surgery to remove the hyperactive parathyroid gland in such patients reduces the risk for stone formation. Unfortunately, even after removing the parathyroid gland many individuals can continue to develop stones.

Estrogen

Estrogen can lower the risk of hyperoxaluria. This female hormone may help to prevent the calcium oxalate stone formation by maintaining alkaline urine, and by increasing the protective citrate levels.

Cystinuria

Cystinuria is an autosomal recessive disorder of the transport of an amino acid called cystine resulting in Cystinuria, resulting in the formation of cystine stone formation. Cystinuria is one of the more common genetic disorders with an overall prevalence is 1 in 7,000. Cystinuria is the most common defect known in the transport of amino acids. The goal of treatment is to prevent the formation of cystine stones. Patients are encouraged to hydrate with a minimum of 4 L/day. Alkalinization of urine to pH > 7.5 can prevent cystine stones. However, at this pH the formation of calcium phosphate can occur. Chelating agents such as D-Penicillamine is used to complex with cystine to make it 50 times more soluble than cystine alone. Alpha-mercaptopropionylglycine (Alpha-MPG), Captopril and Bucillamine (Rimatil) are additional chelating agents to treat patients with cystinuria. These stones are hard and often require surgical intervention by ureteroscopy and laser lithotripsy or percutaneous nephrolithomy.

Hypocitraturia

Hypocitraturia (<320 mg/day), a low level of urinary citrate, is an important factor for kidney stone formation. Citrate is a major inhibitor of calcium salt crystallization in the urine. Low levels of citrate in the urine is a significant risk factor for calcium stones. The causes for hypocitraturia include the 6 D's: patients using **d**iuretics (thiazide or acetazolamide), patients with **d**istal RTA Type I, **d**iets high in animal protein, gouty **d**iathesis, chronic kidney **d**isease and **d**iarrhea. Other causes include the use of topiramate (inhibits renal carbonic anhydrase), corticosteroids, aluminum containing ant-acids and vitamin D. Hypocitraturia also increases the risk for uric acid stones. This condition most likely contributes to about a third of all kidney stones.

The majority (80–90 %) of patients are successfully treated with potassium citrate. Correction of the hypocitraturia reduces the risk of recurrent stone formation [10, 11].

Hyperuricosuria

Hyperuricosuria is a condition in which there are high levels of uric acid in the urine. Hyperuricosuria plays a major role in uric acid stone formation. It also occurs in 15–20 % of people (mostly men) with calcium oxalate stones. When sodium combines with uric acid, it forms Sodium urate, is the salt formed from uric acid combined with sodium, creates the center of a crystal (*nidus*), around which calcium oxalate crystals generate, forming calcium oxalate stones. Such stones tend to be severe and recurrent. They appear to be strongly related to a high intake of protein.

Anatomical Factors

There are multiple anatomical anomalies that increase the risk for nephrolithiasis. These include:

- Horseshoe Kidney
- Medullary Sponge Kidney
- Hydronephrotic renal pelvis or calices
- Calyceal diverticulum
- Ureteral Pelvic Junction Obstruction

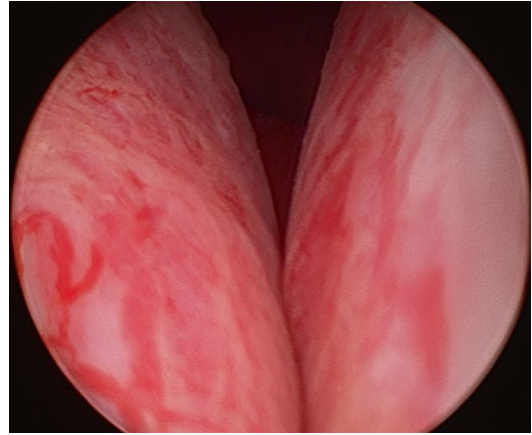


Fig. 9.1 Cystoscopic view demonstrating enlarged lateral lobes of the prostate

- Ureteral stricture
- Vesicoureteral reflux
- Ureterocele
- Benign Prostatic Hyperplasia (Fig. 9.1).

Surgical Factors

Summary Stone Facts

- Not all weight surgical procedures are the same and the risk of stones can be quite different.
- Patients with resection of their gastrointestinal tract have an increased risk for stones.
- The portion of the intestinal tract resected will influence the type of stone the patient will form.

Gastric Bypass Versus Gastric Banding: Does Bariatric Surgery Increase Your Risk of Stones: Fact or Fiction?

Patients who undergo gastric bypass procedures develop changes in their urinary chemistry that increase their risk for developing nephrolithiasis. Patients who undergo the most common type of gastric bypass surgery, the Roux-en-Y (RYGB), whereby creation of a small gastric pouch so food

bypasses part of the small intestines, are at increased risk for calcium oxalate kidney stones beginning 6 months after surgery. In one study, the risk of stones was 7.65 % compared to 4.63 % in the control group [12]. The added kidney stone risk is thought to be due to changes in the urinary chemistry. Patients who have undergone Roux-en-Y gastric bypass surgery have excess oxalate and low levels of citrate in their urine after the procedure.

Other types of weight loss surgery, including gastric banding, is not associated with an increased risk for kidney stones or kidney stone surgeries [13].

Recommendations to reduce an individual's risk for stones following gastric bypass is to increase fluid intake, limit oxalate containing foods, reduce sodium intake and red meat consumption.

Small Bowel Resection

Short bowel syndrome, is a condition that generally occurs when portions of the small intestines are surgically removed. It causes an inability to properly absorb fat and nutrients. These patients may develop steatorrhea, a condition whereby there is excess fat in the stool. Calcium may bind to the unabsorbed fatty acids, resulting in a condition referred to as saponification. This allows increased concentration of free oxalate in the bowl lumen to be absorbed in the colon resulting in hyperoxaluria. As a result of low urine volume, Hypocitraturia, hypomagnesuria and hyperoxaluria, these patients are at an increased risk of developing calcium oxalate stones. Prevention of stones involves a low fat, low oxalate diet, increased fluid consumption to increase urine volume, oral calcium to bind oxalate in the gut lumen and citrate and magnesium replacement [14].

Ileostomy and Colon Resection

Patients with an ileostomy and individuals with a colon resection have a tendency to chronic volume contraction due to loss of water, bicarbonate and potassium in diarrheal stool, leading to reduced urine volumes. These individuals also

have decreased absorption of citrate and magnesium, resulting in less inhibitory action on calcium oxalate crystallization. As a result, these patients have a tendency to produce calcium oxalate stones. These patients also form uric acid stones as a result of bicarbonate loss in the ileostomy resulting in acidic urine combined with watery stools and low urine volume. Therapy is directed by alkalization of the urine to a pH of 6.5, increasing urine volume to increase the solubility of uric acid to prevent crystallization [14]. These stones are often very difficult to prevent without decreases in diarrhea/ileostomy losses.

Genetic Factors

Please see Chap. 8.

Dietary Factors

Please see Chaps. 27 and 28.

Medication Factors: *Drug Induced Renal Calculi*

Summary Stone Facts

- Urinary tract stones can be produced by a number of medications used to treat a variety of medical conditions.
- Some medication can treat one type of stone but produce a different stone type.
- Medication may produce a metabolic abnormality or the drug itself may crystallize in the urine to produce a stone. Correcting this metabolic abnormality may prevent the stone from forming.
- Other drugs may form stones by crystallizing in the urine. Preventing these stones usually involves discontinuation of the medication and initiation of an alternative therapy.

Nephrolithiasis may be produced by a number of prescription and nonprescription drugs used to treat a variety of medical conditions.

These drugs may lead to metabolic abnormalities that initiate the formation of urinary tract stones. Approximately 1–2 % of renal calculi are drug-induced. Drugs that induce metabolic calculi include diuretics, carbonic anhydrase inhibitors, and abuse of laxatives. Correcting the underlying metabolic abnormality may reverse or eliminate the stone formation process.

Urinary calculi can also be produced by medications when the drugs crystallize (i.e., urinary supersaturation) and become the primary component of the stone. Medications that initiate this process include magnesium trisilicate, Ciprofloxacin, sulfa medications, triamterene, indinavir, and ephedrine with or without guaifenesin. When this situation occurs, cessation of the medication is usually necessary to reverse this process.

Loop Diuretics

Bumetanide and Furosemide

Bumetanide and Furoside are used to treat hypertension, edema and hypercalcemia. They inhibit both sodium and calcium resorption in the thick ascending limb of the loop of Henle. In addition to exerting a diuretic effect, this mechanism of action produces a hypercalciuric state, resulting in an increased risk of calcium oxalate stones. In low birth weight infants receiving furosemide therapy, stones have been identified in up to 64 % of infants. These stones are radiopaque on plain x-ray studies.

Carbonic Anhydrase Inhibitors

Carbonic anhydrase inhibitors, such as acetazolamide (Diamox), is used to treat patients with glaucoma, altitude sickness, congestive heart failure, seizure disorder, pseudotumor cerebri and urinary alkalization. This medication acts in the proximal tubule where they block resorption of sodium bicarbonate. Extended use of carbonic anhydrase inhibitors may lead to a hyperchloremic metabolic acidosis, in which urinary pH is increased and urinary citrate is decreased. Consequently, long-term use of this drug can increase the risk for the production of calcium phosphate stones.

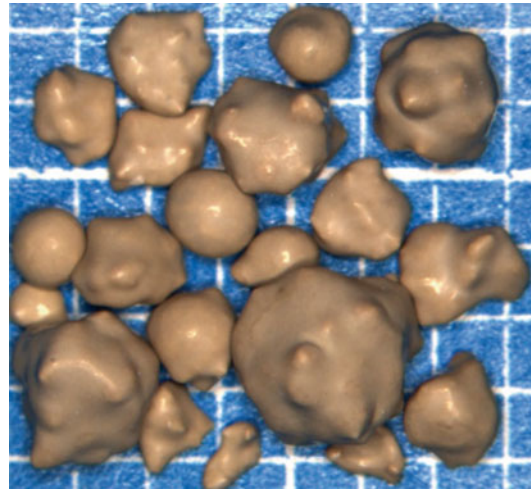


Fig. 9.2 Ammonium acid urate stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

Laxatives

Laxatives are used primarily to treat constipation. Abuse of laxatives can result in diarrhea, nausea and vomiting. Significant water loss can result in low blood pressure, cardiac problems and kidney related issues, including stones. Patients with chronic diarrhea secondary to laxative abuse often have low urine volumes and acidic urine pH. Ammonium acid urate calculi (Fig. 9.2) are more frequent among patients with persistent diarrhea associated with laxative abuse.

Ammonium acid urate calculi are radiolucent on plain X-rays unless mixed with calcium. Cessation of laxatives may lead to the correction of the urinary abnormalities. In addition, counseling by an internist, psychiatrist or psychologist and a registered dietician is recommended to treat the patient's eating disorder.

Magnesium Trisilicate

Silica is a universally distributed element and frequently found in foods such as vegetables, whole grains, seafood and drinking water. Magnesium trisilicate is an antacid medication that is available without a prescription for the treatment of symptoms of gastroesophageal reflux disease. Although dietary silicate is readily excreted in the urine, the consumption of excessive amounts of magnesium trisilicate can produce silicate stone

formation. Silicate calculi are poorly radiopaque and easily treated with conventional lithotripsy methods. Prevention of Stone recurrence can be prevented if the patient discontinues the use of magnesium trisilicate antacids.

Antibiotics

Ciprofloxacin

Ciprofloxacin is a fluoroquinolone antibiotic used to treat complicated and uncomplicated urinary tract infections. It is nearly insoluble at neutral or alkaline pH and crystallizes in excreted alkaline urine of animal models. In patients, ciprofloxacin crystalluria can form when dosages are greater than 1,000 mg and the urine pH is greater than 7.3. Ciprofloxacin calculi are radiolucent on plain radiography and are best identified with contrast imaging studies.

Sulfa Medications: Sulfonamides

The administration of sulfonamides can be complicated by the development of sulfa crystalline aggregates of these drugs in the urine; stones are radiolucent.

Sulfamethoxazole-Trimethoprim

Sulfamethoxazole-trimethoprim (Bactrim) is a commonly employed antibiotic to treat infections.

Sulfadiazine

Sulfadiazine, a medication commonly used to treat acquired immunodeficiency syndrome and toxoplasmic encephalitis, has particularly low urine solubility and can produce sulfonamide-induced calculi. These stones may be avoided with adequate hydration and urinary pH manipulation. Sulfa-induced calculi are radiolucent on plain radiography.

Indinavir

The protease inhibitor, indinavir (Crixivan), is a medication used to commonly treat patients with human immunodeficiency virus (HIV). More than 10 % of AIDS patients who take the medicine indinavir develop stones (Fig. 9.3). The risk is even higher in people with AIDS

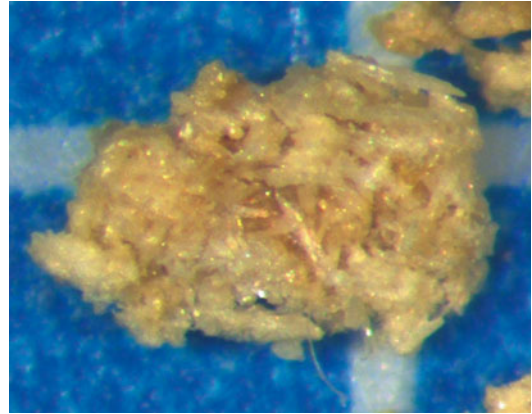


Fig. 9.3 Crixivan stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

who also have hepatitis B, hepatitis C, or hemophilia, as well as those who are very thin or who take the antibiotic combination TMP-SMX. Indinavir stones are not visible with spiral CT scan or renal ultrasound. CT with contrast allows the stones to be detected. Initial therapy of indinavir stones includes discontinuation of medication (with alternative protease inhibitor prescribed), hydration and analgesic support. Surgical intervention is required as would be for other symptomatic stones, including intractable pain, nausea, vomiting, fever, chills or signs of sepsis.

Uricosuric Agents

These medications include such drugs as colchicine and probenecid, causing hyperuricosuria and thus, can increase the risk for uric acid as well as calcium stones.

Decongestants

Ephedrine

Ephedrine, can be derived from certain types of evergreen plants (genus *Ephedra*), is an easily obtainable drug that is claimed to promote euphoria, increase energy, heighten sexual sensation, increase muscle mass, and effect weight loss.



Fig. 9.4 Guaifenesin stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

Ephedrine is also found in some drugs used to treat asthma and congestion and can produce ephedrine-based stones as they are excreted into the urine. These stones are radiolucent

Guaifenesin

Guaifenesin is known for its bronchodilation and expectorant properties, may result in stones composed of these products as they are excreted into the urine.

Preparations containing ephedrine and guaifenesin are easily attainable in the United States on an over-the-counter basis. These calculi derived from Ephedrine and Guaifenesin (Fig. 9.4) should be treated in a similar way to other types of stones. Counseling for substance abuse is recommended after calculi treatment to help limit stone recurrence. These stones are radiolucent on plain radiographic imaging and may require renal ultrasound or spiral CT scan to identify these stones.

Chemotherapeutic Agents

Certain cancer chemotherapies lead to cell breakdown and can cause uric acid stones.

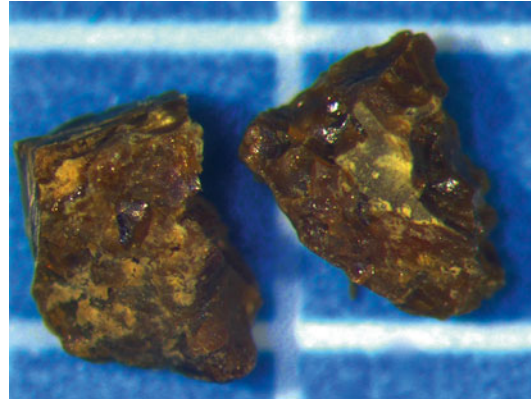


Fig. 9.5 Triamterene stone (Courtesy of Louis C. Herring & Co., Orlando, FL)

Diuretics

Triamterene

Triamterene is a potassium-sparing diuretic used to treat edema and hypertension, is associated with the formation of triamterene stones. The mechanism by which these stones form is not clear. Among patients with triamterene-induced stones (Fig. 9.5), 21 % of the stones are pure triamterene, with the remainder being mixed stones. Triamterene calculi cannot be dissolved by pH manipulation and, therefore, must be treated with conventional lithotripsy procedures. The formation of triamterene calculi can be avoided by discontinuation of the medication. Triamterene is poorly radiopaque on plain radiography, although it does not have the density of stones consistent with calcium oxalate.

Salicylates: Can Aspirin Cause Kidney Stones?

The best known example of salicylate is Aspirin (acetylsalicylic acid). Aspirin can be used as an analgesic to relieve aches and pain, as an anti-pyretic to reduce fever, as an anti-inflammatory agent and as antiplatelet by inhibiting the production of thromboxane. Side effects of aspirin include gastrointestinal ulcers, gastric bleeding, ringing in ears and Reye's Syndrome (acute encephalopathy and fatty liver). Salicylates have been demonstrated to cause stones.

Anti-epileptic Drugs (AEDs)

Topiramate

Topiramate (Topamax) is an anticonvulsant used to treat seizures and migraine headaches. Topiramate can lead to high urine pH and very low citrate levels, increasing the risk of stones. Studies show that 1.5 % of patients using topiramate reported urinary calculi. Patients develop calcium phosphate calculi, supporting the hypothesis that topiramate induces a metabolic acidosis, resulting in hypocitraturia and an alkaline urine. Preventive measures of these stones include high fluid intake, limited sodium intake, and consumption of citrate-containing fluids. Moreover, patients receiving long-term topiramate therapy may require bone densitometry testing to rule out early calcium loss secondary to acid buffering by bone.

Zonisamide

Zonisamide (Zonegran) is a sulfonamide drug used as an anti-epileptic agent. Zonisamide also has weak carbonic anhydrase activity. Studies demonstrate that this medication has a 4 % incidence of renal calculi. Use of this medication is associated with an alkaline urine, hypercalciuria, and calcium phosphate stones. Urinary calculi resolved with cessation of Zonisamide and supportive therapy.

Allopurinol

Allopurinol is a medication that decreases the amount of uric acid in the urine, reducing the risk of uric acid stone formation. In patients taking Allopurinol for the treatment of gout, xanthine stones may develop.

Potassium Citrate

Potassium citrate (Urocit K) is used to prevent the formation of calcium, uric acid and cystine stone. The medication can prevent calcium oxalate stones in individuals who have hypocitraturia and uric acid stones or cystine stones in patients who have acidic urine. Common side effects of

this medicine include diarrhea and nausea. In patients taking potassium citrate and there was an increase in urine pH (>8), patients were at risk for the formation of calcium phosphate stones.

Steroids

Long-term corticosteroid use can increase gastrointestinal absorption of calcium, leading to hypercalciuria and an increased risk for calcium-containing stones.

Other medications that result in drug-induced stones include phenytoin, cephalexin, felbamate, phenazopyridine and naproxen (see Fig. 9.6).

In summary, medications have a purpose for treating patients for a specific medical condition, however, all medications have potential side effects. There are many different medications used to treat common conditions that can lead to kidney stones. Fortunately, the risk of these medications causing stones is rare. Patients should remain aware and know the potential risks of these medications prescribed. Review with your physician that a specific medication is best for you. Be certain that the benefits of the medication outweigh the potential risks. Even medications that are used to treat one type of stone, as with Allopurinol and Potassium Citrate, may put you at risk for forming another stone type. Always remember, that too much of a good thing is not always better!

Other Risk Factors for Stones

Gender

Males are two times more likely to develop stones than women. See Chap. 10 for additional information.

Obesity

Obesity and weight gain are both risk factors for nephrolithiasis. An elevated body mass index (BMI) and wider waist circumferences are both

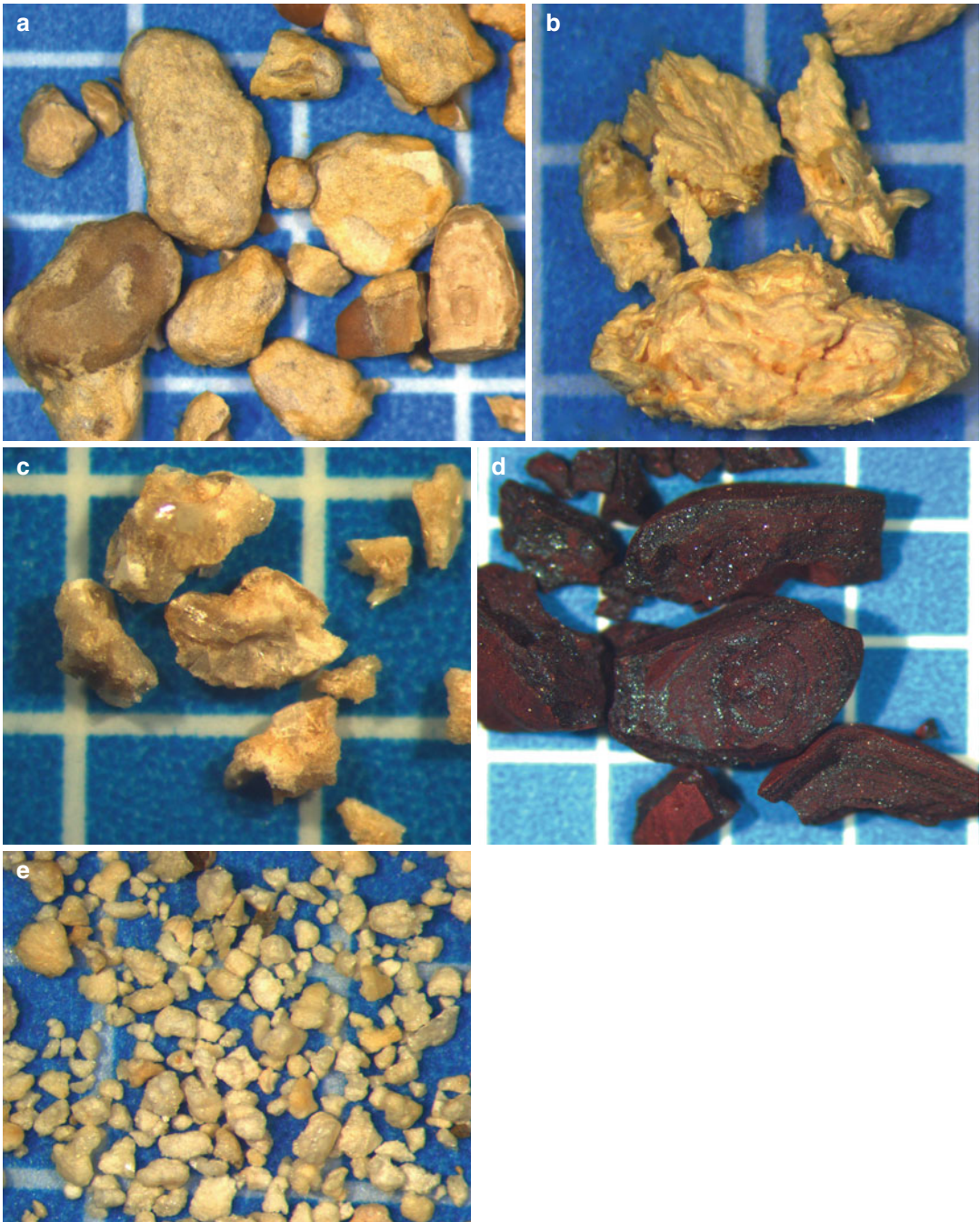


Fig. 9.6 Drug-induced stones include (a) phenytoin, (b) cephalixin, (c) felbamate, (d) phenazopyridine, and (e) naproxen (Courtesy of Louis C. Herring & Co., Orlando, FL)

risk factors for stone disease. Compared with individuals at or near ideal body weight (BMI=21–23), obese men (BMI \geq 30) have a 33 % greater risk for stone formation, while

obese women have a 200 % greater risk [15]. A relationship may exist between fat tissue, insulin resistance, and urine composition. Individuals with larger body sizes may excrete more calcium

and uric acid into the urine, increasing the risk for stone formation.

Personal History

People with a personal history of stones have a 50 % risk of developing stones over the next 5 years. Additionally, Individuals who developed a kidney stone have an 80 % chance of recurrence over their lifetime.

Family History

A family history of stones is associated with an increased risk. Patients with a family history of kidney stones have a two to three times higher risk. In rare situations, certain disease situations, including Renal Tubular Acidosis Type I, associated with calcium phosphate stones, is an autosomal recessive trait. Both parents would need to be a carrier for this trait to pass it on to their child. Likewise, patients forming cystine stones are due to genetic defects. A family history of gout may also put an individual at risk to developing kidney stones.

Nationality

Developing countries have a much lower risk of nephrolithiasis, compared with developed countries. This is presumably due to dietary factors, specifically the absence of a Western-style, meat-based diet (see Chap. 28 for additional information on diets and stones).

Geography

Areas of elevated temperatures and high humidity appear to have an increased incidence of stone disease. Food factors, chemicals and minerals in the drinking water may contribute to in the presence of stones in different regions. Nephrolithiasis is reported to be highest in the southern regions and lowest in the western regions of the United States.

Ethnicity

Caucasians have the highest incidence of nephrolithiasis, followed by Mexican Americans. African-Americans have the lowest stone risk for kidney stones. Caucasians are three times more likely to develop stone disease than African-Americans. Dietary factors, however, can minimize the protective effects of ethnicity.

Conclusion

In summary, there are multiple factors that contribute to nephrolithiasis. The risks for stones include environmental, occupational, lifestyle, gastrointestinal, medical disease, hormone, surgical, medication, family history and ethnicity factors. Individuals who are risk for stones should be aware of these factors. Address these risks with your physician and discuss options for reducing your chances for stone formation.

August and September are my two least favorite months of the year! This dislike has nothing to do with the fact that I am a college professor and those months mark the beginning of the fall semester, but rather that nearly half of my approximately 25 kidney stones over the last 30 years have began their journey of passing during those 2 months. I have been told that summer is known as “kidney stone season” and for me this is very much the case! The beginning of one of my worst kidney stone incidents occurred after a full day in the Florida sun at the amusement park in mid August. I make a conscious effort to hydrate during the summer and yet I still have an uptick in stone frequency in late summer/early fall. As a Marine and Atmosphere scientist who researches climate change, I am the expert as a researcher and patient of climate on human health, a career that I am eager to teach and as a patient that *I am enthusiastic to pass down!*

References

1. Brikowski TH, Lotan Y, Pearle MS. Climate-related increase in the prevalence of urolithiasis in the United States. *Proc Natl Acad Sci U S A*. 2008;105(28):9841–6.
2. Lo SS, Johnston R, Al Sameraai A, et al. Seasonal variation in the acute presentation of urinary calculi over 8 years in Auckland, New Zealand. *BJU Int*. 2010;106(1):96–101.
3. Chen YK, Lin HC, Chen CS, et al. Seasonal variations in urinary calculi attacks and their association with climate: a population based study. *J Urol*. 2008;179(2):564–9.
4. Linder BJ, Rangel LJ, Krambeck AE. The effect of work location on urolithiasis in health care professionals. *Urolithiasis*. 2013 Aug;41(4):327–31.
5. Najem GR, Seebode JJ, Samady AJ, Feuerman M, Friedman L. Stressful life events and risk of symptomatic kidney stones. *Int J Epidemiol*. 1997;26(5):1017–23.
6. Katsanos KH, Tsianos EV. The kidneys in inflammatory bowel disease. *Ann Gastroenterol*. 2002;15(1):41–52.
7. Glassberg KI. Renal dysgenesis and cystic disease of the kidney. In: Walsh PC, Retik AB, Vaughan ED, Wein AJ, editors. *Campbell's urology*, vol. 3. 8th ed. Philadelphia: WB Saunders Company; 2002. p. 1925–94.
8. Grantham JJ, Nair V, Winklhofer F. Cystic diseases of the kidney. In: Brenner BM, editor. *Brenner & Rector's the kidney*, vol. 2. 6th ed. Philadelphia: WB Saunders Company; 2000. p. 1699–730.
9. Parmar MS. Kidney stones. *BMJ*. 2004;328(7453):1420–4.
10. Barcelo P, Wuhl O, Servitge E, Rousaud A, Pak CY. Randomized double-blind study of potassium citrate in idiopathic hypocitraturic calcium nephrolithiasis. *J Urol*. 1993;150(6):1761–4.
11. Pak CY, Fuller C, Sakhaee K. Long-term treatment of calcium nephrolithiasis with potassium citrate. *J Urol*. 1985;134(1):11–9.
12. Matlaga BR, Shore AD, Magnuson T, Clark JM, Johns R, Makary MA. Effect of gastric bypass surgery on kidney stone disease. *J Urol*. 2009;181(6):2573–7.
13. Semins MJ, Matlaga BR, Shore AD, Steele K, Magnuson T, Johns R, Makary MA. The effect of gastric banding on kidney stone disease. *Urology*. 2009;74(4):746–9.
14. Worcester EM. Stones from bowel disease. *Endocrinol Metab Clin North Am*. 2002;31(4):979–99.
15. Taylor EN, Stampfer MJ, Curhan GC. Obesity, weight gain, and the risk of kidney stones. *JAMA*. 2005;293:455–62.

Sex and Stones: Sex and Stones May Break Your Bones, but Water Will Not Harm You!

10

David A. Schulsinger

Summary Stone Facts

- Gender is a risk factor for certain disease states that may increase an individual's risk for renal stones.
- Stones are more common in men, but the gender gap is declining.
- Struvite stones are more common in women; uric acid stones are more prevalent in men.

While stones are common among men and women there are stone-related factors that vary between the sexes. Certain disease states are gender specific and gender neutral. Various disease states are associated with nephrolithiasis. This chapter addresses some of the gender barriers that exist between men and woman regarding nephrolithiasis.

Gender Statistics and Stones

Kidney stones represent an important health care issue, affecting up to 15 % of the United States population [1]. Stones form twice as often in men as they do form in women. The lifetime risk for

stone disease exceeds 12 % in males and 6 % of females [1–3].

While previous reports suggested a male to female predominance of stones, there seems to be growing evidence that the differences are narrowing and that stones may be an “equal opportunity disease.” In a study using The Nationwide Inpatient Sample from 1997 to 2002, the change in prevalence by gender (male to female ratio) for treated stone disease changed from 1.7:1 to 1.3:1 [4]. The increased prevalence of stones in women may be accounted for by lifestyle risk factors, such as obesity, in women as compared to men.

Annual incidence rates are approximately three cases per 1,000 for men and one to two per 1,000 in women [5–8].

A seasonal variation is also seen, with high urinary calcium oxalate saturation in men during summer and in women during early winter [9].

Gender Age and Stones

The average age and peak age at which patients are most likely to develop a stone varies between men and women. Men are more likely to present with nephrolithiasis between the ages of 40 and 70 years. The peak age in men is 50 years. Women have a bimodal age distribution, with peaks at 35 and 55 years. In younger women, nephrolithiasis are likely to develop during the late stages of pregnancy.

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Gender and Stone Types

Urinary tract infections are more common in women. Struvite stones are also called magnesium ammonium phosphate or infectious stones. These stones are more common in women. In fact, women are twice as likely to develop struvite stones as men. Gout, on the other hand is seen more commonly in men. Men are more likely to form uric acid stones.

Gender and Stone Symptoms

Both men and women can experience back or flank pain associated with a stone in the kidney or proximal ureter. When the stone nears the bladder (i.e., stone in the distal ureter), both genders may feel urgency, frequency, burning or painful urination. In this location, men may also have pain that radiates down to the testis and complain of scrotal pain on the same side as the stone. Women, on the other hand, may have pain that radiates down to the labia or vagina on the same side as the stone.

Gender Diseases and Stones

Despite the narrowing gender gap for stone formation, the reason for stone creation is different between men and women. This following section looks at some of the gender barriers for stones regarding different disease states.

Laxative Abuse

Laxative abuse is a disorder commonly seen in women. These patients are very thin, malnourished and even anorectic. Patients who abuse laxatives will develop a chronic acidosis due to a loss of bicarbonate from the gastrointestinal tract. The pH of the urine favors an acidotic state, which can lead to demineralization of the bones, resulting in osteopenia or osteoporosis.

These patients described above have low urine volume secondary to water loss. The loss of



Fig. 10.1 Ammonium acid urate stones derived from laxative abuse

potassium produces hypokalemia resulting in hypocitraturia (low urinary citrate). This causes in an increase of ammonium by the kidneys resulting in the production of ammonium acid urate (Fig. 10.1). These patients also develop calcium oxalate stones. These are the most common types of kidney stones reported with laxative abuse [10].

Eating Disorders

Eating disorders, such as anorexia nervosa and bulimia nervosa are very common. These eating disorders are known to be associated with kidney stones.

In anorexia, the person has an intense fear of gaining weight and limits the food they eat. Eighty-five to ninety-five percent of these individuals are female and 1–4.2 % of women suffer from anorexia in their lifetime [11]. As a result of this disorder, fasting, vomiting and purging result in loss of fluids and minerals. The chronic dehydration and low potassium levels can lead to kidney stones and even renal failure. In anorexia nervosa, nephrolithiasis are composed most frequently of ammonium urate due to low urinary output and increased urinary ammonium output secondary to low urinary phosphate and diarrhea with hyperchloremic metabolic acidosis. Low urine output leading to high urinary uric acid,

oxalate and calcium can lead to uric acid and calcium stones as well as nephrocalcinosis [12].

Bulimia is characterized by enormous consumption of food, followed by self-induced vomiting. Eighty-five to ninety percent of individuals with bulimia are women. Up to 4 % of females in the United States will have bulimia in their lifetime [13]. The individuals alternate between eating and purging. Urinary Calcium and Oxalate levels rise during eating and drop during the fasting phase.

In treating patients with eating disorders who make stones, the first issue is to address the underlying disease. These patients first need to address their eating disorder. Treating this may correct the risk of stones. Since eating disorders may be a life threatening condition, patients are encouraged to address this issue first. The psychological issues of this disorder are beyond the scope of this discussion.

Crohn's Disease

Crohn's disease is an inflammatory bowel disease that results in inflammation of the lining of the digestive tract. It affects men and women equally. In this disease, fat binds to calcium, leaving oxalate free to be absorbed and deposited in the kidney, where it can bind with calcium to form a stone. Their urine is more concentrated, a condition that is more likely to lead to stone formation. The risk of stones is greater than the general population and these individuals usually produce calcium oxalate stones [14]. Kidney stone treatment calls for an increased fluid intake together with a low-oxalate diet.

Patients with Crohn's disease are also at risk for forming uric acid stones. This type of stone develops in acidic urine and this is caused by increased uric acid absorption in the injured colon. These patients can be treated with potassium citrate.

Osteoporosis

There is a higher prevalence of osteoporosis and a greater incidence of fractures in postmenopausal women than older men [15]. Osteoporosis

is often referred to as a "woman's disease". Gender is a risk factor for osteoporosis and may be a risk factor for stones.

Cardiovascular Disease

Women with a history of kidney stones may be at a greater risk of developing coronary heart disease [16]. This association or increased risk was not found in men. A possible explanation for this distinction may be hormonal differences between men and women.

Gender and Anatomical Barriers for Stones

The ureter has three anatomical narrowed areas along its course from the kidney to the bladder. First is the ureteropelvic juncture (or UPJ); the second area is the crossing iliac vessels; the third area is the ureterovesical juncture (or UVJ). These anatomical barriers for blocking the passage of a stone exist in both men and women.

In men with enlarging prostates, they may have an additional barrier that may prevent the passage of stones.

Bladder Stones

Gender and age are the two highest risk factors for bladder stones. Ninety-five percent of the individuals who develop bladder stones are men. Middle-aged men over the age of 50 have the greatest risk of developing bladder calculi. An enlarged prostate, referred to as benign prostatic hyperplasia (BPH), is a major risk factor for bladder stones in men. As the prostate grows, it interferes with the flow of urine resulting urine to be retained within the bladder.

Five percent of all bladder stones occur in women. These stones are usually associated with calcifications of foreign bodies (synthetic material) within the bladder, such as sutures, synthetic tapes or meshes [17].

Gender Hormones and Stones

Testosterone

Male stone former were found to have higher serum total testosterone than men without stones [18]. This suggests that testosterone may be a lithogenic factor for stone disease.

Estrogen

Female hormones (estrogens) actually lower the risk of hyperoxaluria. Estrogen may help prevent the formation of calcium oxalate stones by keeping urine alkaline, and by raising protective citrate levels.

Gender and Stone Risk Factors

Obesity

Obesity and weight gain can increase the risk of stone formation. Men and Women weighing more than 220 lb were 44 and 90 %, respectively, more likely to develop stone disease than women weighing less than 150 lb. Both men and women who gained more than 35 lb after 21 years of age were 39 and 82 %, respectively, more likely to develop calculi than individuals who did not gain weight. The magnitude of this increased stone risk may be greater in women than in men [19].

Obesity is one of the major factors that increase the risk of nephrolithiasis in postmenopausal women. Eating more than 2,200 cal per day could increase the risk for nephrolithiasis by up to 42 % [20]. Exercise was shown to reduce the risk of stones. See Chap. 13 for more information on obesity and stones.

Gender Factors and Stones

Pregnancy

During pregnancy, there are physiological changes that occur which may influence a woman's chances of developing stones. Overall, the

risk of developing stones during pregnancy is 1 in 1,500, which appear to be a similar occurrence to non-pregnant women [21].

Factors that increase the risk of stones:

1. The intestines absorb additional calcium and more is released into the urine.
2. The kidney and ureters become dilated due to increased levels of progesterone, especially on the right side due to compression fetus against the upper urinary tract. This results in slower delivery of urine and a greater risk of infection and stone formation.

Factors that decrease the risk of stones:

1. There is an increase of urinary citrate, which is an inhibitor of stone formation.
2. There is an increase in the filtration activity by the kidneys.

Stones formed during pregnancy are more commonly of the calcium phosphate [22] variety compared to the more common calcium oxalate stones formed by the general population. This may be indicative of the physiologic changes taking place during pregnancy. See Chap. 11 for additional information on pregnancy and stones.

Post-menopause

The incidence of stones increases after menopause in women. Menopause is associated with an increased excretion of urinary calcium [23, 24], which may enhance the risk for calcium stones.

There are several conflicting studies on the use of estrogen replacement therapy in healthy postmenopausal women increasing [25] or decreasing [26] the risk of kidney stones.

Vasectomy

Studies demonstrate that men younger than their mid forties who underwent vasectomies had twice the risk for nephrolithiasis than their peers who did not have a vasectomy [27]. The risk for stones persisted for up to 14 years after the procedure. Men who undergo vasectomies are encouraged to drink fluids to minimize this risk.

Summary

In summary, while stones are common in both men and women, between the sexes, differences definitely do exist (see Table 10.1). The frequency of stones, the symptoms associated with stones, the types of stones, the prevalence of stones, anatomical barriers associated with stones, and the timing of stones are different between men and women. Remember, certain disease states are gender specific which may put an individual at risk for stones. The means by which we treat patients with stones is similar.

Nineteen year old college student and athlete who was gaining weight, turned to abusing laxatives for 12 months and had a 35 lbs. weight loss over the same period of time. She presented to the ER with right flank pain. She also complained of nausea, vomiting and chills but no fever. CT Scan revealed 8 mm right UPJ stone and moderate right hydronephrosis. Patient received a DJ stent and was discharged to follow up in the office and to determine definitive care of stone. Patient was given various treatment options including conservative care, ESWL and ureteroscopy. Because the patient was still in school, she wanted to undergo surgical intervention and do a procedure so as to minimize her trips to the ER and potentially jeopardize her time away from school. She decided on ureteroscopy, laser lithotripsy and stone manipulation. She underwent this procedure and due to the dilation of the ureter from her previously placed stent, the stone was manipulated intact without any stone fragments. Patient was stone free. Stone analysis revealed ammonium acid urate. Twenty-four-hour urine revealed low urine pH. The laboratory tests were consistent with a stone secondary to laxative abuse. While the patient was started on medical therapy, she was also referred to a psychiatrist for counseling. A week later she called

Table 10.1 The chart below summarizes some of gender differences that exist with stone-related factors

Factors	Male	Female
Prevalence	12 %	6 %
Incidence	3/1,000	1–2/1,000
Peak age of stones	30 years	35 and 55 years
Most common season for stones	Summer	Winter
Most common stone type	Calcium oxalate and uric acid	Calcium oxalate
Radiation of pain for pain in distal ureter	Testis	Labia/vagina
Obesity risk	44 %	90 %

the office to report, “Crap, I passed another stone.” That was an interesting way of phrasing that!

References

1. Stamatelou KK, Francis ME, Jones CA, Nyberg LM, Curhan GC. Time trends in reported prevalence of kidney stones in the United States: 1976–1994. *Kidney Int.* 2003;63:1817–23.
2. Gillen DL, Worcester EM, Coe FL. Decreased renal function among adults with a history of nephrolithiasis: a study of NHANES III. *Kidney Int.* 2005;67:685–90.
3. Johnson CM, Wilson DM, O’Fallon WM, Malek RS, Kurland LT. Renal stone epidemiology: a 25-year study in Rochester, Minnesota. *Kidney Int.* 1979;16:624–31.
4. Scales Jr CD, Curtis LH, Norris RD, Springhart WP, Sur RL, Schulman KA, Preminger GM. Changing gender prevalence of stone disease. *J Urol.* 2007;177(3):979–82.
5. Hiatt RA, Dales LG, Friedman GD, Hunkeler EM. Frequency of urolithiasis in a prepaid medical care program. *Am J Epidemiol.* 1982;115:255–65.
6. Curhan GC, Willett WC, Rimm EB, Stampfer MJ. A prospective study of dietary calcium and other nutrients and the risk of symptomatic kidney stones. *N Engl J Med.* 1993;328:833–8.
7. Curhan GC, Willett WC, Speizer FE, Spiegelman D, Stampfer MJ. Comparison of dietary calcium with supplemental calcium and other nutrients as factors affecting the risk for kidney stones in women. *Ann Intern Med.* 1997;126:497–504.
8. Curhan GC, Willett WC, Knight EL, Stampfer MJ. Dietary factors and the risk of incident kidney stones in younger women: Nurses’ Health Study II. *Arch Intern Med.* 2004;164:885–91.

9. Parks JH, Barsky R, Coe FL. Gender differences in seasonal variation of urine stone risk factors. *J Urol.* 2003;170:384–8.
10. Leaf DE, Bukberg PR, Goldfarb DS. Laxative abuse, eating disorders, and kidney stones: a case report and review of the literature. *Am J Kidney Dis.* 2012;60(2):295–8.
11. The Renfrew Center Foundation for Eating Disorders, “Eating Disorders 101 Guide: A Summary of Issues, Statistics and Resources,” published September 2002, revised October 2003, <http://www.renfrew.org>.
12. Jonat LM, Birmingham CL. Kidney stones in anorexia nervosa: a case report and review of the literature. *Eat Weight Disord.* 2003;8(4):332–5.
13. The National Institute of Mental Health. Eating disorders: facts about eating disorders and the search for solutions. Pub No. 01-4901. Accessed Feb 2014
14. Viana ML, Pontes RM, Garcia WE, Fávero ME, Prete DC, Matsuo T. Crohn’s disease and kidney stones: much more than coincidence? *Arq Gastroenterol.* 2007;44(3):210–4.
15. Cawthon PM. Gender differences in osteoporosis and fractures. *Clin Orthop Relat Res.* 2011;469(7):1900–5.
16. Ferraro PM, Taylor EN, Eisner BS, Gambaro G, Rimm EB, Mukamal KJ, Curhan GC. History of kidney stones and the risk of coronary heart disease. *JAMA.* 2013;310(4):408–15.
17. Stav K, Dwyer PL. Urinary bladder stones in women. *Obstet Gynecol Surv.* 2012;67(11):715–25.
18. Watson JM, Shrewsbury AB, Taghechian S, Goodman M, Pattaras JG, Ritenour CW, Ogan K. Serum testosterone may be associated with calcium oxalate urolithogenesis. *J Endourol.* 2010;24(7):1183–7.
19. Taylor EN, Stampfer MJ, Curhan GC. Obesity, weight gain, and the risk of kidney stones. *JAMA.* 2005;293(4):455–62.
20. Sorensen MD, Chi T, Shara NM, Wang H, Hsi RS, Orchard T, Kahn AJ, Jackson RD, Miller J, Reiner AP, Stoller ML. Activity, energy intake, obesity, and the risk of incident kidney stones in postmenopausal women: a report from the Women’s Health Initiative. *J Am Soc Nephrol.* 2014;25:362–9.
21. Drago JR, Rohner TJ, Chez RA. Management of urinary calculi in pregnancy. *Urology.* 1982;20(6):578–81.
22. Meria P, Hadjadj H, Jungers P, Daudon M. Stone formation and pregnancy: pathophysiological insights gained from morphoconstitutional stone analysis. *J Urol.* 2010;183:1412–6.
23. Nordin BEC, Need AG, Morris HA, Horowitz M, Robertson WG. Evidence for a renal calcium leak in postmenopausal women. *J Clin Endocrinol Metab.* 1991;72:401–7.
24. Adami S, Gatti D, Bertoldo F, Rossini M, Frattapasini A, Zamberlan N, Facci E, LoCascio V. The effects of menopause and estrogen replacement therapy on the renal handling of calcium. *Osteoporosis Int.* 1992;2:180–5.
25. Maalouf NM, Sato AH, Welch BJ, Howard BV, Cochrane BB, Sakhaee K, Robbins JA. Postmenopausal hormone use and the risk of nephrolithiasis: results from the Women’s Health Initiative hormone therapy trials. *Arch Intern Med.* 2010;170(18):1678.
26. Heller HJ, Sakhaee K, Moe OW, Pak CY. Etiological role of estrogen status in renal stone formation. *J Urol.* 2002;168(5):1923–7.
27. Roberts HJ. Is vasectomy worth the risk? A Physician’s Case Against Vasectomania. West Balm Beach, FL: Sunshine Sentinel Press; 1993.

Heather N. Di Carlo and David A. Schulsinger

Simple Stone Facts

- 1:1,500 pregnant women present with stones, however the risk of stone disease is not increased due to pregnancy.
- The majority of stones (66 %) will pass spontaneously during pregnancy.
- Evaluation and management of a pregnant woman with urolithiasis can be extremely challenging.
- Radiation exposure to the fetus should be minimized.
- Urinary tract infections must be treated promptly to prevent pre-term labor.
- Drainage of the obstructed kidney is the primary goal when infection, intractable pain, and/or intolerance to oral hydration and nutrition are present.
- PCNL and ESWL are contraindicated during pregnancy.

H.N. Di Carlo, MD
Division of Pediatric Urology,
Johns Hopkins School of Medicine,
The James Buchanan Brady
Urological Institute, Baltimore, MD, USA
e-mail: hdicar11@jhmi.edu

D.A. Schulsinger, MD (✉)
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Introduction

When a pregnant patient presents with a stone, many will declare that it feels like a double delivery! Some women will swear that the “delivery was easier and less painful than passing my stone.” Fortunately, the incidence of pregnancy and stones is no greater than the general population, with a 1:1,500 risk. Pregnancy and nephrolithiasis can pose diagnostic and therapeutic challenges during different stages of the pregnancy. The purpose of this chapter is to review urolithiasis and pregnancy, with an understanding of the symptoms, different types of work up and various treatment options to manage you and your fetus during this period.

Anatomic Factors

The dynamic anatomy and physiology of a pregnant woman along the course of her pregnancy can make the diagnosis of urolithiasis more difficult than her non-pregnant counterpart. The physiological dilation of the ureter begins at 6–10 weeks of gestation and continues until 4–6 weeks following delivery. Pregnant women often have swelling of the kidney and ureter, called hydroureteronephrosis, due to compression of the ureter by the uterus. This is more common on the right, as the uterus tends to lean to the right side. Additionally, increased circulating levels of progesterone reduce the peristalsis (contractions)

and relax the smooth muscles of the ureter. Increased urine production can also cause dilation of the ureters and renal pelvis. If obstruction of the kidney occurs due to compression of the ureter by the gravid uterus, renal colic symptoms can develop. It can be challenging to distinguish renal colic from a passing stone.

Metabolic Factors

An elevated level of vitamin D during pregnancy increases urinary calcium excretion causing hypercalciuria, seemingly increasing the risk of urolithiasis. However, this increase in urinary calcium is counteracted by an increase in urinary magnesium and citrate, inhibitors of calcium stone formation. In addition, an increase in glycosaminoglycans and acidic glycoproteins inhibit oxalate stone formation. There is also an increase in the glomerular filtration rate (GFR) and renal plasma flow (RPF), increasing the urine fluid output.

These opposing factors explain why pregnancy is not associated with an increase risk of stone formation when compared to non-pregnant patients.

Presentation and Work-Up

Symptoms of renal colic during pregnancy can be a result of ureteral obstruction from the gravid uterus, urinary stasis, or a ureteral stone. Renal colic is the most common nonobstetrical cause of abdominal pain in pregnant patients requiring hospitalization. A pregnant woman with renal colic should be evaluated with a urinalysis, urine culture and blood work. It is extremely important that a suspected urinary tract infection or any degree of bacteria in the urine, called bacteriuria, be promptly treated as this is a cause of premature labor and maternal pyelonephritis. Appropriate antibiotics that are safe to the mother as well as the fetus are necessary. Antibiotics that can be given during pregnancy include penicillin, cephalosporin and erythromycin.

Renal and pelvic ultrasound is the recommended gold-standard initial diagnostic imaging study for evaluation of the urinary tract and to rule out stones in the pregnant woman. The advantage of a renal ultrasound is the absence of radiation exposure to mother and fetus, no pain, and avoidance of intravenous contrast, which is potentially toxic to the kidneys. Hydroureteronephrosis will be seen, if present. Stones can be identified in the kidney, but can be difficult to visualize in the ureter.

Evaluation of urine drainage from the ureter into the bladder, called ureteral jets, can identify obstruction if they are not present. Radiologic imaging modalities with radiation exposure such as radiographs and CT should be limited to complex cases. Preferably, ionizing radiation should be avoided, particularly in the first and second trimesters of pregnancy. Radiation exposure in the third trimester is lower risk to the fetus. An MRI, while it does not pose a risk to the pregnancy, has limited value in stone disease.

Treatment Options During Pregnancy

The most ideal initial treatment option for pregnant women with stones is conservative care, taking advantage of bed rest, hydration and analgesia. A conservative approach to treatment is encouraged, especially if the stone is non-obstructing and the individual is asymptomatic. Conservative management permits the pregnant woman a trial of passage and pain management. There is a high rate of stone passage in patients undergoing conservative care with approximately two-thirds (64–84 %) of stones passing during the pregnancy period. Medical expulsive therapy (MET) can also be used as a conservative approach to manage stones in the ureter. The most common choice of MET to promote stone passage is the use of an alpha-1-blocker. These are medications used to treat men with benign prostatic hypertrophy (BPH), however, off label use of this medication has emerged as a safe and efficacious option for initial management of ureteral stones.

Studies have addressed the use of alpha-1 blockers in pregnant woman, and I have personally used

this form of MET therapy successfully in pregnant women with ureteral stones in coordination with my obstetrical colleagues. In pregnancy, alpha-1 blocking agents are considered Class B, with no 1st trimester studies available and no evidence of 2nd or 3rd trimester risk. Consult with your obstetrician and urologist to determine if this medication is best for you.

If a pregnant woman develops intractable pain, or inability to tolerate oral nutrition (food or water), then intervention is indicated. Ureteral stenting may be performed cystoscopically if the patient is not demonstrating signs of sepsis, but this typically requires sedation, regional or general anesthesia. It is recommended that the ureteral stents be exchanged every 3–4 weeks, as there is a higher rate of encrustation of these foreign bodies during pregnancy. If a patient requires drainage, a stent may be a good choice during the later stages of the pregnancy where fewer exchanges are required.

Drainage of the collecting system is recommended percutaneously with a nephrostomy tube using ultrasound guidance under local anesthesia, especially when there are signs of infection, fever, or sepsis. Nephrostomy tubes are recommended when a stone becomes symptomatic during early pregnancy, as the tubes require fewer exchange procedures compared to stent changes. Nephrostomy tubes are changed every 4–8 weeks.

There is a preference towards percutaneous nephrostomy placement by many urologists for this patient population, as the nephrostomy tube tends to encrust and occlude less and exchanges do not require anesthesia as are performed with ureteral stents.

If conservative therapy fails, patients are unable to tolerate a stent or nephrostomy tube and no infection is present, then surgical management of the stone may be necessary prior to delivery. Prior to the procedure, there is a coordinated effort between the obstetrical, urology, radiology and anesthesia services to plan the case and to have all services in the operating room during the procedure. Fetal monitoring will be required during the procedure. Radiation exposure to the fetus can be avoided with ultrasound guidance. Ureteroscopy and stone extraction, with holmium

laser lithotripsy, if necessary, is the preferred method. Other forms of lithotripsy utilizing ultrasonic and electrohydrolic forces are contraindicated. The advantage of this procedure is that the stone is potentially removed and the disadvantage is that the patient will require anesthesia. Other surgical treatment options, including shockwave lithotripsy (ESWL) and percutaneous nephrolithotomy (PCNL), are contraindicated during pregnancy. *The urologist is cognizant that while they are operating on 1 stone, they are treating 2 patients!*

There are rare circumstances where the woman makes it to the third trimester but is unable to tolerate the stent or nephrostomy tubes to term. If deemed appropriate by the obstetrician, labor may be induced and the stone surgery performed after delivery.

Once drainage of the collecting system has been achieved, definitive management of the stone is deferred until after the woman delivers. A CT can be obtained to evaluate for stone burden and location, guiding further management options. Commonly, the stone procedure is performed under elective circumstances. The urologist will typically wait a minimum of 6 weeks to allow circulating hormone levels to return to baseline, minimizing the risk of bleeding. Ureteroscopy with laser lithotripsy, PCNL or ESWL can all be performed once the mother recovers from childbirth.

Follow Up

After the delivery and definitive management of the stone, patients will require the same appropriate testing (see Chap. 24), medication (Chap. 19) and dietary management (Chap. 27), as other nonpregnant patients, to prevent future stones. The 24 hour urine test, for example, is used to understand risk factors for stones. This test should be postponed until after the delivery as physiologic and metabolic changes associated with pregnancy may affect the results. Your goal is to enjoy your new child as your urologist will care for your stone prevention!

Summary

Stone disease in pregnancy can be a diagnostic and therapeutic challenge. Fortunately, the majority of stones that present in pregnant women will pass spontaneously. If conservative treatment does not work, in some cases minimally invasive procedures can be utilized to treat the stone, maintaining the safety to the mother and fetus. With an accurate diagnosis, counseling and treatment strategy, the outcome for the mother and fetus is excellent. In treating this situation, the urologist is mindful that while they are treating 1 stone, they are caring for 2 patients!

I am a 39-year old nurse with a history of stones. At 10 weeks gestation, I was pregnant with my third child, when I went to hospital with fever and flank pain. The pain was relentless! An ultrasound showed I had a 17 mm right renal stone. Because it was early in my pregnancy, the physician gave me the various treatment options, including removal of the stone with ureteroscopy, stent placement or nephrostomy tube placement. Because I was in the first trimester, I felt it was best for me and my

fetus to minimize the risk during my pregnancy and plan for a definitive procedure after I delivered. I elected to have a nephrostomy tube placement with additional tube changes required, but less frequently than if I had a stent. I had a total of six nephrostomy tubes during that pregnancy with the last tube exchanged 4 weeks before my delivery. It was a long and trying pregnancy, but grateful that my daughter delivered uneventfully and is healthy. While my delivery was a walk in the park, the pain from my kidney stone was not!

Further Reading

1. Gupta N, Ko J, Matlaga BR, Wang MH. Ureteroscopy for treatment of upper urinary tract stones in children: technical considerations. *Curr Urol Rep.* 2014;15:407.
2. Moe OW. Kidney stones: pathophysiology and medical management. *Lancet.* 2006;367:333.
3. Riddell JVB, Denstedt JD. Management of urolithiasis during pregnancy. *Contemp Urol.* 2000;12(3):12.
4. Stothers L, Lee LM. Renal colic in pregnancy. *J Urol.* 1992;148:1383.
5. Waltzer WC. The urinary tract in pregnancy. *J Urol.* 1981;125(3):271.

Heather N. Di Carlo and David A. Schulsinger

Stone Summary Facts

- Stone incidence is increasing in the pediatric population.
- Western dietary changes coupled with metabolic predispositions increase the risk of stone formation in children.
- Kidney and bladder ultrasound is the first line imaging choice to assess the urinary tract in children.
- A multidisciplinary team approach is vital in managing the pediatric patient with stone disease.

The management of patients with urinary tract stones (urolithiasis) can be quite challenging, especially in the pediatric population, in the elderly and medically fragile. This chapter aims to discuss stone disease in this challenging group of patients.

H.N. Di Carlo, MD
Division of Pediatric Urology, Johns Hopkins School of Medicine, The James Buchanan Brady Urological Institute, Baltimore, MD, USA
e-mail: hdicar11@jhmi.edu

D.A. Schulsinger, MD (✉)
Department of Urology, Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Pediatric Patients and Stones

There has been a substantial increase in the number of children diagnosed and treated for urinary tract stone disease in the United States in the past decade. Adult stone disease has also been on the rise, but to a lesser extent. Although there are many reasons for children to form stones in their urinary tract, it is believed that lifestyle changes such as increased salt intake, inadequate calcium intake, and reduced fluid intake are mostly responsible for this rise. Stones in children, similar to adults, are more common in Caucasians than those individuals of Hispanic and African American descent.

Types of Stones

Anatomic Considerations

Stones (calculi) can be present anywhere along the urinary tract. The renal pelvis collects urine excreted by the collecting ducts via the calyces, which then moves down the ureter and into the bladder. The urine then passes through the urethra in the anatomically normal child.

Kidney stones (nephrolithiasis) are formed in the collecting system of the kidney. They form for many different reasons, some of which will be discussed later in this chapter. Stones that form in the kidney may stay in the calyces or renal pelvis without causing any symptoms. Conversely, these stones may pass into the ureter. When a

stone passes into the ureter, it may cause pain in the flank that radiates into the groin or genitalia on the affected side as it intermittently blocks the urine flow. This symptomatology is known as renal colic. Once a stone passes into the bladder, the pain usually improves and the stone may pass via the urethra without any other symptoms.

A *bladder stone* may be the result of a passed kidney stone that hasn't made its way out the urethra, or it could result from other causes. Less commonly in children, obstruction of the urinary tract at the level of the bladder neck or urethra can cause stasis leading to stone formation. This may be from an acquired obstruction from surgery to correct a urethral abnormality such as hypospadias, or from a congenital obstruction such as posterior urethral valves with a hypertrophic bladder neck. This may also be the result of a bladder that does not empty well, known as a neurogenic bladder. These are all potential causes of urinary stasis predisposing the child to the formation of stones.

Patients who have had reconstructive surgery of their bladder using segments of bowel, known as augmentation enterocystoplasty, form mucus in the urine, which can act as a nidus on which stones can form. If these patients do not adequately irrigate their bladders to remove the mucus, they are at a much higher risk for stone formation.

Another reason for a child or adolescent to have bladder stones is from a foreign body in the urinary tract. These will act as a nidus for stone formation as crystals form on the surface, even in patients that have no metabolic predisposition to forming calculi. Foreign bodies can include ureteral stents or urethral catheters placed in the urinary tract by a physician for a prolonged period of time, or other objects that are not intended to be inside the body. If an object is forced up the urethra, it can become lodged in the urethra or bladder, requiring surgical interventions to remove. This pelvic radiograph (Fig. 12.1) is that of a teenager with metallic beads in his bladder. They were a nidus for stone formation and became calcified over a short period of time requiring open surgical removal.

Stone Composition

Renal, endocrine, and metabolic disorders can lead to the development of crystallized material



Fig. 12.1 Abdominal X-ray demonstrating metallic beads within the bladder

in the urinary system ultimately causing stones (See Chapter 8). Stones are classified based on their chemical components. Most stones in children contain calcium, most commonly calcium oxalate, calcium phosphate, or a combination thereof. Uric acid, magnesium ammonium phosphate (struvite), and cystine are some of the lesser common causes of stones in the pediatric population. Struvite stones are associated with infection and are much less common in the pediatric population than they were a decade ago. Cystine stones form in patients with cystinuria, an autosomal recessive defect that impairs renal reabsorption of the amino acids cystine, ornithine, lysine, and arginine. Elevated urinary cystine leads to the formation of cystine stones.

Inborn errors of metabolism can cause significant but variable stone production in pediatric patients. Patients with the rare diagnosis of primary hyperoxaluria form calcium oxalate monohydrate stones while those with xanthinuria produce xanthine stones (which, interestingly, are not able to be seen on a radiograph – they are radiolucent). Another stone-forming syndrome is Lesch-Nyhan disease (juvenile gout). These patients have a deficiency of an enzyme called hypoxanthine-guanine phosphoribosyltransferase (HGPRT) and produce uric acid stones.

Risk Factors

As we have explored, the formation of urolithiasis is a multifactorial process influenced by the

patient's metabolic background/predisposition along with environmental conditions/exposures (See Chapter 9).

Stones form via super-saturation of the urine and crystallization of stone-forming (lithogenic) components, which is followed by growth of the crystals. A nidus for crystal precipitation (i.e., the urothelial surface properties that affect crystal retention) occurs when the microscopic crystalline structure of one crystal is similar to another crystal and the second crystal grows on the first.

Important risk factors for pediatric urolithiasis include habitually low urine volumes and high urine content of stone-forming components (e.g. calcium, uric acid and oxalate). Urine pH also affects stone formation – uric acid and cystine are less soluble in acidic urine while struvite and calcium phosphate are less soluble in alkaline urine.

In addition to lithogenic components, urine also contains substances that are inhibitory to stone formation, including citrate, magnesium, glycosaminoglycans, and osteopontin. Citrate is the only inhibitor that can be increased in the urine, having therapeutic applications.

More than 50 % of children diagnosed with stones have a metabolic risk factor. The most common risk factor is hypercalciuria, followed by hypocitraturia, hyperuricosuria, and hyperoxaluria, respectively.

Dietary influences are hypothesized to be a large contributor to the increase in the incidence of urolithiasis in the pediatric population. Inadequate fluid intake leading to low urine volume is the most significant cause coupled with an increased salt intake, which increases calcium excretion in the urine.

Congenital anomalies of the urinary tract causing obstruction and stasis include ureteropelvic or ureterovesical junction obstruction and sometimes horseshoe kidney. The stasis of urine can predispose the child to stone formation.

Urinary tract infections with urea-splitting microorganisms are a decreasing cause of stones (namely struvite as discussed earlier) in children as the diagnosis and management has greatly improved over the past decade.

Exposure to particular drugs including topiramate, phenytoin, loop diuretics, acetazolamide,

and high dose vitamin C and D increase calcium stone formation. Excessive consumption of calcium (milk alkali syndrome) as well as melamine-contaminated powdered formula (seen in China in 2008) have also been shown to be risk factors.

Presentation

The classic symptoms of renal colic are less common in younger children, who often present with irritability, abdominal pain and/or hematuria. Older children and adolescents present with more localizing symptomatology, as in adults. Urolithiasis should always be on the physician's radar when evaluating a child with abdominal pain.

Work-Up

During the acute episode, assessment with urinalysis, urine culture, blood work and imaging are necessary. In the pediatric patient, radiation exposure should always be limited. Evaluation with kidney and bladder ultrasound is the first line imaging choice to assess the urinary tract in children. This modality will demonstrate any evidence of swelling of the collecting system and/or ureter (hydroureteronephrosis, hydroureter, or hydronephrosis) indicating an acute obstruction. Urine flow from the ureters into the bladder can also be evaluated with ultrasonography, which will show ureteral jets as urine enters the bladder from each ureter in an unobstructed system. Urolithiasis may be visualized utilizing ultrasonography, although helical computed tomography (CT) without intravenous or oral contrast, called a spiral CT scan, is a much more sensitive study compared to ultrasound. There is no radiation exposure with ultrasound, as compared to CT. When CT is necessary, the imaging protocol should always be designed to decrease the amount of radiation exposure while ensuring adequate imaging quality, a technique known as ALARA ("As Low As Reasonably Achievable"). Abdominal and pelvic radiograph can be limited for detecting calculi in the pediatric patient due to overlying bowel gas.

Treatment

There are many options for the treatment of urinary tract stones in the pediatric patient. The choice for management is determined based on the characteristics of the stone, the chance for spontaneous passage, potential complications of intervention versus observation, and the chance for stone-free success.

Watchful waiting or observation can be used to manage asymptomatic or small calculi in the kidney, ureter, or bladder. Periodic imaging with renal and bladder ultrasound to ensure that the stone(s) are not enlarging is important. Additionally, small ureteral stones may be observed as they pass in the acute phase. Adequate hydration and pain control may necessitate an inpatient hospital admission. Medical expulsive therapy utilizing alpha-blockers such as tamsulosin dilate the distal ureter and can facilitate passage of the stone into the bladder. Persistent pain, intractable nausea and emesis, and/or fever prompt intervention for an obstructing ureteral calculus. This intervention in the acute setting is a drainage procedure to allow the kidney to be unobstructed – using a ureteral stent placed endoscopically under anesthesia, or a percutaneous nephrostomy tube.

Ureteroscopy is a technique using a tiny camera with fiberoptics to visualize the inside of the ureter and kidney, localizing the stone(s). A laser is then used to break apart the stone(s) too large to be retrieved from the kidney or ureter. Fragments are then removed from the urinary tract with small instruments inserted through the ureteroscope.

Extracorporeal shock wave lithotripsy (ESWL) is a technique using shock waves to fragment the stone(s) into small pieces for passage. It's safety and efficacy have been demonstrated in the pediatric stone disease population.

Percutaneous nephrolithotomy (PCNL) is a surgical technique where the kidney is accessed percutaneously usually via a flank approach under ultrasound and/or fluoroscopic guidance. A nephroscope is passed into the kidney to visualize, fragment, and remove stones. This is a par-

ticularly useful method for patients with a large stone burden in the kidney. Refer to Chapter 20 for additional information on surgical treatment of stones.

Medical/metabolic therapy is targeted at shrinking the size of existing stones without surgical intervention. The type of therapeutic option chosen, including medications to increase urinary citrate (e.g. potassium citrate) and decrease calcium excretion into the urine, depends upon the etiology of the stone disease. Chapter 19 provides a detail account of medical therapy for stone disease.

Prevention

Once a stone is captured, either by spontaneous passage or surgical intervention, it should be analyzed to determine its composition. After the acute phase and definitive treatment of the stone, a 24 hour urine collection should be performed at least twice to determine if the child has predisposing risk factors for stone production.

Dietary modifications are important preventative measures. Increasing fluid intake to get a target urine output of greater than 30 mL/kg/day can significantly decrease the recurrence of urolithiasis. A low sodium diet (<2 g/day) reduces urinary calcium excretion by enhancing sodium and calcium reabsorption in the tubules of the kidney. See Chapter 27 for more information on diet and stones.

Most pediatric centers have developed a multidisciplinary team approach to the pediatric patient with stone disease. This team consists of urologists, nephrologists, nutritionists, and specialized nursing staff to optimize outcomes and urinary parameters with the goal of decreasing the recurrence of urolithiasis.

Conclusion

In summary, while children and adults both form stones, the etiology for stones in children, the presenting symptoms, the choice of imaging studies

and the treatment options are different. The most common cause of kidney stones in children is an inherited abnormality. Increased salt intake coupled with low urine volumes (low fluid intake) increases the risk of stone formation in all comers, *particularly* in children. Unlike the classic colicky, flank pain seen in adults, diffuse abdominal pain may be seen in children. Minimizing radiation exposure is crucial in the evaluation of pediatric patients with urolithiasis. Abdominal ultrasound is the most reliable initial test in younger children. A multidisciplinary team approach to the pediatric patient with stone disease is essential for stone treatment and stone prevention.

The Elderly and Stones: Does Aging Impact Stones?

Summary Stone Facts

- Elderly patients with urolithiasis pose different challenges due to multiple medical comorbidities.
- Dehydration is a major risk factor for stones, especially in the elderly.

Overview

There is an increasing prevalence of urolithiasis in the aging population. When stratified by age, there is a rise-and-fall pattern with increasing age. The incidence rates by age groups peak at age 40–49 and then shows a steady decline for men in the United States, Japan and Iran into their 70's [1]. Despite this decline, the care for senior patients with stones is critical in this potentially medical fragile group. Elderly patients often have multiple medical comorbidities, which can make successful management of stones quite challenging. Chronic kidney disease (CKD), hypertension, diabetes, metabolic syndrome and coronary artery disease all pose serious challenges in the management of these patients. Additionally, the risk of CKD is greater in stone formers.

Risk Factors: Are Elderly Prone to Stones?

Dehydration is the most common risk factor for stones in adults, especially in the elderly. The elderly are prone to dehydration for several reasons. Their sense for thirst is less; seniors take medications (diuretics and laxatives) that increase the risk of fluid loss; elderly have higher risk for reduced mobility and limiting their ability to obtain sufficient fluids; seniors have a greater fear of drinking due to incontinence and inability to achieve access to the bathroom; medical comorbidities (Parkinson's Disease) may limit their ability to handle a cup of fluids. Seniors should be aware of signs and symptoms of dehydration and consult with their physician to identify individual needs for fluids. Refer to Chapter 9 for additional risk factors for stones.

Diagnosis and Management

Urolithiasis should always be on the differential diagnosis list of the physician caring for an elderly patient with abdominal pain. A thorough evaluation by a urologist is the same for the elderly patient as the adult patient with suspected stone disease. In the acute setting, urinalysis, urine culture, blood work, imaging including renal ultrasound and CT are useful in establishing the diagnosis.

Management strategies are similar to those utilized in the adult stone disease patient. Awareness of the patient's comorbidities and risks of anesthetics must be balanced so the patient can be optimized by their physician for possible surgical intervention.

Close medical management in addition to surgical therapies is critical, especially in patients with osteoporosis. These patients should have a high dietary intake of calcium and vitamin D, which lowers the risk of stone formation compared to those taking calcium and vitamin D supplements (i.e. vitamins purchased at the store). A low salt diet is also crucial to decrease urinary calcium excretion. Refer to Chapter 27 for additional information on diet, osteoporosis and stones.

Finally, hydration is critical factor in maintaining a healthy balance of fluids. An adequate volume of fluids aids in promoting a healthy cardiovascular system (to maintain blood pressure and heart rate) as well as minimizing the risk of future stones. Consult with your physician to determine the appropriate amount of fluids.

Conclusion

In summary, elderly patients with stones have unique challenges for proper management. These patients will make stones and are treated for stones similar to other adults. Senior patients tend to have more medical co-morbidities and take a greater number of medications (including anticoagulants) that put them at a high surgical risk. These patients need to be optimized by their primary care physician to ensure the best chances for success when they require surgical intervention for their stone disease.

Further Reading

1. Romero V, Akpınar H, Assimos DG. Kidney stones: a global picture of prevalence, incidence, and associated risk factors. *Rev Urol.* 2010;12(2–3):e86–96.
2. Gentle DL, Stoller ML, Bruce JE, et al. Geriatric urolithiasis. *J Urol.* 1997;158(6):2221.
3. Gupta N, Ko J, Matlaga BR, Wang MH. Ureterscopy for treatment of upper urinary tract stones in children: technical considerations. *Curr Urol Rep.* 2014; 15:407.
4. Moe OW. Kidney stones: pathophysiology and medical management. *Lancet.* 2006;367:333.
5. Nakamon T, Kitiratrakarn P, Lojanapiwat B. Outcomes of percutaneous nephrolithotomy: comparison of elderly to younger patients. *Int Braz J Urol.* 2013; 39(5):692.
6. Routh JC, Graham DA, Nelson CP. Epidemiological trends in pediatric nephrolithiasis at United States freestanding pediatric hospitals. *J Urol.* 2010;184(3): 1100.
7. Rule AD, Bergstralh EJ, Melton 3rd LJ, et al. Kidney stones and the risk for chronic kidney disease. *Clin J Am Soc Nephrol.* 2009;4(4):804.
8. Sas DJ. An update on the changing epidemiology and metabolic risk factors in pediatric kidney stone disease. *Clin J Am Soc Nephrol.* 2011; 6(8):2062.

David A. Schulsinger

Simple Stone Facts

- Obese individuals have a higher risk of stones disease.
- Individuals gaining weight as adults have a higher risk of stones.
- Higher BMI and waist size are associated with higher risk of stones.
- Obese and overweight patients with nephrolithiasis mainly produce stones composed of calcium and uric acid.

Obesity is a medical condition in which the accumulation of excess body fat may have a negative impact on an individual's health that increases the risk of various diseases or potentially reduces life expectancy. Worldwide there are 1.5 billion overweight or obese adults, a number that is expected to reach three billion by 2030. More than 34.9 % of adults (age ≥ 20 years) and 17 % of youths (age 2–19 years) and 8.1 % of infants and toddlers in the United States are obese. The prevalence of obesity in United States is high and has remained stable [1]. The health risk of obesity in United States has been associated with many diseases. Obesity-related conditions include, type 2 diabetes, hypertension, heart disease, high cholesterol, hypertension, stroke and cancer. The costs associated with obesity in the

United States are exponential. In 2008, the estimated annual medical cost of obesity in the United States was \$147 billion. The average medical costs for people who are obese are \$1,429 higher than those individuals of normal weight in 2006 [2].

On the other hand, approximately 10 % of men and 5 % of women developed kidney stones in their lifetime and more than \$2 billion are spent annually on treating stone disease. Among other health related issues, there is a strong association between obesity and stone disease. Many studies have shown a direct correlation between obesity and stone disease. This chapter will examine the Association between obesity and the risk for developing nephrolithiasis.

Obesity, Weight Gain and Stone Disease

Obesity and weight gain are both associated with an increased risk of kidney stones. Men weighing more than 220 lb and women weighing more than 150 lb were 44 and 90 %, respectively, more likely to develop stone disease than individuals weighing less than these body weights. Both men and women who gained more than 35 lb after the after 21 years of age were 39 and 82 %, respectively, more likely to develop calculi than individuals who did not gain weight. Among women, younger and older women who gained 35 lb had an 82 and 70 %, respectively, higher risk of forming stones than those whose weight did not change. These

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, New York, USA
e-mail: endourology@yahoo.com

studies demonstrate the strong link between obesity and formation of stone disease [3].

BMI and Stone Disease

Body mass index (BMI) is used to measure weight and height to indicate obesity. For example, for adult men and women with a BMI between 18.5 and 24.9 is considered healthy; an adult with a BMI between 25 and 29.9 is considered overweight and a BMI of ≥ 30 is considered obese.

Higher BMI's and larger waist circumferences are both risk factors for kidney stones. Researchers think that there may be a link between fat tissue, insulin resistance, and urine composition. People with larger body sizes may excrete more calcium and uric acid, which increase the risk of kidney stone formation. BMI is shown to be associated with both an increased prevalence of nephrolithiasis and with larger stone sizes [4].

Waist Size and Stone Disease: Look Great, Lose the Waist!

Waist circumference is one of the simplest and most often used measure of abdominal obesity. Women with a waist size ≥ 35 in. and men with a waist size ≥ 40 in. are considered to have abdominal obesity.

Waist circumference was correlated with an increase risk of stone formation in men and women. The relative risk (RR) of forming stones in men with larger waist (>43 in.) compared to smaller waist (<34 in.) was 1.48 [3]. In older and younger women, the RR to form stones with larger waist (>40 in.) compared to smaller waist (<31 in.) was 1.71 and 1.94, respectively [3].

The Link Between Obesity and Types of Stones

Obesity and weight gain is associated with insulin resistance. In type 2 diabetes mellitus, insulin resistance has been shown to lower urine pH due to a defect in ammonium production and acid excretion [5], thereby increasing the risk of uric acid stones.

In addition, a defect in renal acid excretion may lead to hypocitraturia, thus increasing the risk of calcium stones [6, 7]. Increased serum insulin, or hyper-insulinemia, is associated with obesity and increases in urinary calcium, thereby, increasing the risk of calcium stones [8–10].

Several studies have shown that an inverse relationship exists between weight and urine pH. Among patients with nephrolithiasis, higher weight is associated with lower urinary pH [11, 12]. While lower urinary pH is associated with uric acid stones, the inability to excrete acid can result in hypocitraturia, thereby increasing the risk of calcium stones.

Stones associated with obesity are mainly composed of calcium oxalate (94.4 %) and uric acid (55.5 %) [13]. These percentages are much greater than the general population of stone-forming patients where the distribution of stones is 80 % calcium oxalate and 10–15 % uric acid [3, 14].

Summary

In summary, there is strong evidence to demonstrate that stone disease is closely associated with obesity. Weight gain, high body mass index and large waist size are shown to increase an individual's risk for renal stones. Obesity is a recognized health problem that affects many organs systems and diseases including nephrolithiasis. In addition to your Cardiologist and Endocrinologist, your Urologist also has good reason for you to maintain a healthy weight and to avoid gaining weight. Remember, *the weight is over, lighten the load!*

Mrs. BI is a 57-year-old morbidly obese female with a BMI of 47. Her medical history is complicated by type II diabetes and hyperlipidemia. She also has a 5 year history of uric acid stone disease. 24 hour urine demonstrated a high urinary uric acid and low urine pH. She has undergone two lithotripsy procedures, passed several

painful stones and countless trips to the emergency room. She was under medical management for her stone disease using potassium citrate for alkalization of her urine and prevention of her stone disease. Unfortunately, she was not able to tolerate the medication and continued to generate uric acid stones. After a heart-to-heart talk with her cardiologist and endocrinologist, she was told about her obesity, health risks and longevity. She realized that if she did not make significant changes in her health that she might die. Considering all her treatment options, she underwent a diet modification under the guidance of her dietitian. After losing 120 lb, her serum triglycerides and glucose have normalized. She is no longer taking cholesterol medication and her diabetes is diet controlled. On the stone front, I was pleased to see that her 24 hour urine chemistry demonstrates normal urinary uric acid levels and pH. In addition, she no longer requires medical therapy for her stone disease and has remained stone free for 2 years. Remember, **lose the weight and give your kidneys a break!**

References

1. Cynthia L, Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA*. 2014;311(8):806–14.
2. Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer and service specific estimates. *Health Aff*. 2009;5:w822–31.
3. Taylor EN, Stampfer MJ, Curhan GC. Obesity, weight gain, and the risk of kidney stones. *JAMA*. 2005; 293(4):455–62.
4. Mosli HA, Mosli HH. Increased body mass index is associated with larger renal calculi. *Urology*. 2012;80(5):974–9.
5. Krivosikova Z, Spustova V, Dzurik R. Participation of P-dependent and P-independent glutaminases in rat kidney ammoniogenesis and their modulation by metabolic acidosis, hippurate and insulin. *Physiol Res*. 1998;47:177–83.
6. Coe FL, Parks JH, Asplin JR. The pathogenesis and treatment of kidney stones. *N Engl J Med*. 1992; 327:1141–52.
7. Hamm LL. Renal handling of citrate. *Kidney Int*. 1990;38:728–35.
8. Kerstetter J, Caballero B, O'Brien K, Wurtman R, Allen L. Mineral homeostasis in obesity: effects of euglycemic hyperinsulinemia. *Metabolism*. 1991;40:707–13.
9. Shimamoto K, Higashiura K, Nakagawa M, et al. Effects of hyperinsulinemia under the euglycemic condition on calcium and phosphate metabolism in non-obese normotensive subjects. *Tohoku J Exp Med*. 1995;177:271–8.
10. Nowicki M, Kokot F, Surdacki A. The influence of hyperinsulinaemia on calcium-phosphate metabolism in renal failure. *Nephrol Dial Transplant*. 1998;13: 2566–71.
11. Maalouf NM, Sakhaee K, Parks JH, Coe FL, Adams-Huet B, Pak CY. Association of urinary pH with body weight in nephrolithiasis. *Kidney Int*. 2004;65:1422–5.
12. Powell CR, Stoller ML, Schwartz BF, et al. Impact of body weight on urinary electrolytes in urinary stone formers. *Urology*. 2000;55:825–30.
13. Mosli HA, Mosli HH, Kamal WK. Kidney stone composition in overweight and obese patients: a preliminary report. *Res Rep Urol*. 2013;5:11–5.
14. Knoll T. Epidemiology, pathogenesis, and pathophysiology of urolithiasis. *Eur Urol Suppl*. 2010;9(12):802–6.

Part IV

The Work-Up

David A. Schulsinger

Summary Stone Facts

- There is a full spectrum of symptoms associated with stones that range from asymptomatic to symptomatic.
- Stones not blocking the ureter are usually asymptomatic.
- Symptomatic stones are typically associated with flank pain with/without nausea, vomiting, fever and/or chills.
- Be aware that the size of stone does not predict the severity of pain.

become larger over time. Resting in the kidney, stones can oftentimes remain asymptomatic. They are like bee bees in a glass jar circulating round and round. Imagine that there is a small hole in the bottom of that glass jar, similar to the labyrinth game that you may have played as a child. The ball would travel through the maze in hopes of avoiding the ball the fall into the hole.

This hole in the urinary tract is where the pelvis of the kidney joins the ureter, called the UPJ, or *ureteral pelvic juncture* (Fig. 14.2). This is the first anatomical narrowing of the urinary tract. Here, stones can get trapped and block the flow of urine from the renal pelvis into the ureter. This

Introduction: Why the Stone-Prone Groan

We all know someone who had a stone, or you may have had a stone yourself. We hear about that relentless pain that takes someone down to his or her knees, sometimes crying like an infant. Female patients describe that it was easier to give birth to a child than it was to deliver a stone. So why is it that patients get this unrelenting and sometimes unforgiving pain?

We know that stones begin as Randall's plaques (Fig. 14.1) in the papilla and mature their way into the pelvis of the kidney where they can

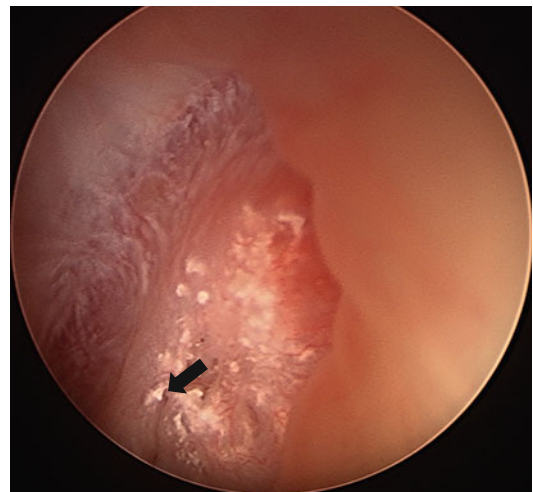


Fig. 14.1 Multiple Randall's plaques in the papilla (black arrow)

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

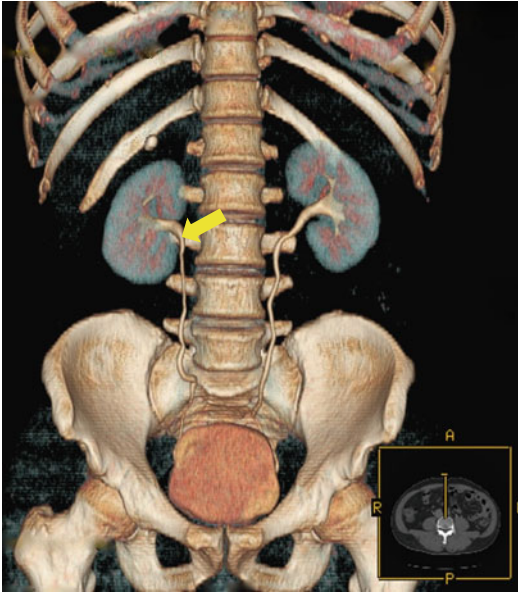


Fig. 14.2 3D CT Scan reconstruction of the urinary tract. The ureteropelvic junction (UPJ) is identified (yellow arrow)

interruption of flow results in the back up of urine into the kidney. This causes the kidney to swell, or properly termed *hydronephrosis*. Hydronephrosis can be graded as mild, moderate or severe, depending on how swollen the kidney becomes. Ultimately, the back of urine in the kidney can cause this intractable pain, referred to as *flank pain or renal colic*. This is the typical pain that brings a patient to the ER for treatment. Left untreated, the back up of urine may become infected, resulting in a kidney infection, also referred to as *pyelonephritis*.

Straight to the Point

Now that this stone is in the UPJ, the stone has several fates:

1. It can remain where it is. This is the typical scenario that if the stone does not move and it continues to obstruct the ureter and there is progressive hydronephrosis, surgical intervention is typically required. This is the typical patient that goes to the ER where the patient may require urinary diversion with a stent or nephrostomy tube.
2. It can continue forward into a deeper portion of the ureter toward the bladder. If the stone is small enough, it may advance further down the ureter,

also referred to *antegrade* progression. If fortunate enough, the stone may advance down to the bladder and voided out with urination. The ureter has two other narrowed points where the stone can become blocked. The stone may get stuck at the level where the ureter crosses the iliac vessels or in the lowest point of the ureter, referred to as the UVJ, or *ureteral pelvic juncture*.

3. It can propel backwards into the renal pelvis. The stone moving back into the kidney is called *retrograde* movement. In this last scenario, the stone has the potential to bounce back and forth between the kidney and ureter. This is what I refer to as the “ping pong” effect. The stone bounces back and forth from the renal pelvis to the ureter, and then back again. When the stone bounces back into the renal pelvis, the pain typically resolves as the obstruction is relieved and urine is able to pass the stone down the ureter into the bladder and ultimately through the urethra. Upon entering and obstructing the ureter again, there is back of urine within the kidney, causing hydronephrosis and the flank pain can return.

The Point of No Return

There is a point of no return. The stone has migrated down the ureter where the stone is distal enough not to bounce back to kidney. As the stone moves down the ureter, the flank pain can begin to radiate down to the groin. As the stone approaches the bladder, the symptoms begin to change. The stone moves into the ureter that traverses the bladder, called the *intramural ureter*. The stone in this location results in irritation to bladder muscle, resulting in irritative voiding symptoms, including frequency or urination, urgency of urination and even urge incontinence. Patients may also have burning with urination, or *dysuria*. These symptoms are very similar to symptoms of a UTI, however, the urine culture is typically negative for an infection.

Point Well Taken

Stone in the ureteral portion of the bladder presents differently in men and women. In men, the

pain in the bladder can radiate to the testicle on the same side, referred to as the *ipsilateral testicle*. In women, the pain may be referred to the ipsilateral labia or possible pain within the vagina. I tell my patients that the objective interpretation of pain in this location is that this is a pending stone for passage since the stone is in it's most distal location before entering the bladder.

In both men and women, a stone in the distal ureter needs to be differentiated from other disease processes, also called the *differential diagnosis*. Pain presenting in the right lower quadrant of the abdomen needs to be differentiated from appendicitis. Pain in the left lower quadrant should be differentiated from diverticulitis. Refer to your primary care physician or urologist who can do the appropriate examination and testing to make this differentiation and appropriate diagnosis.

The Point of the Matter

Most episodes of acute flank pain will be related to the kidney in origin. However, one must be aware of factors that can cause colic-like symptoms. This pain may be muscle-skeletal in origin. Pre-dating the onset of pain, patients may recall lifting or pulling something heavy that results in sharp flank pain that mimics pain associated with a stone. To differentiate stone pain from muscle skeletal pain, I ask patients to do a simple test. Placing your hands on your flank, rotate from side to side and from front to back. If the pain proves to be worse in one position and relieved in another, there is a good chance that this pain is muscle-skeletal in origin. If this pain displayed presents with the same intensity on the affected side regardless of the position they are in, this could represent pain that is renal in nature.

In addition, a patient's pain may be neurologic in nature. Shingles, caused by the varicella-zoster virus, the same virus that causes chickenpox, should be ruled out. Patients may present with pain, described "piercing needles in skin" or "a match", that is persistent. However, patients may have vesicles and surrounding erythema on the flank or torso, described as "tear drops on a rose

petal." The presence of a rash and blisters helps to differentiate shingles pain from renal colic. Shingles may present with pain without a rash, and differentiating this from renal colic maybe more difficult. Consult with your physician to make the diagnosis and proper treatment plan.

Alternatively, the source of this pain may be gastrointestinal in nature. If you recognize that the timing of this pain is coordinated with changes in your bowel habits, have a history of irritable bowel or constipation, let your primary care physician or your urologist know as this may require additional tests or investigation.

In the differential diagnosis, be aware that there are many other medical conditions that may mimic symptoms that are similar to nephrolithiasis. Urinary tract infections, while less intense, can cause a similar type of pain. Conversely, individuals with a urinary tract infection may also present with a stone. While the most common urinary tract infection is *E. coli*, this bacterium does not cause stones. Stones that are caused by infection are also known as *magnesium ammonium phosphate or struvite stones*. One of the more common infections associated with struvite stones is *Proteus mirabilis*. If you present with this type of infection, you have a high degree of suspicion that there may be a stone. Other bacteria that can cause struvite stones are *Pseudomonas*, *Klebsiella*, *Providencia*, *Serratia* and *staphylococci*. Other conditions that may have similar symptoms to nephrolithiasis include: intestinal obstruction, blood clots, Crohn's Disease or ulcerative colitis, gastric or duodenal ulcers, pancreatitis, hepatitis, pelvic inflammatory disease, irritable bowel syndrome and heart attack.

In summary, depending on the stone location and side of the body in which it is located (laterality), stone pain must be differentiated from other sources of discomfort. For proximal ureteral pain, on the right side, this can be confused with cholecystitis or cholelithiasis; on the left side, the differential diagnoses include acute pancreatitis, peptic ulcer disease, and gastritis. Mid to lower ureteral pain in particular can easily mimic appendicitis on the right or acute diverticulitis on the left. Consult with your physician when you present with abdominal pain.

What Is the Time for Pain?

Pain associated with a stone can occur at any-time. Stone attacks most commonly occur late in the evening or early in the morning. This may be related to the lack of hydration and low urine output during these hours. Kidney stone attacks are least common in the afternoon.

Can Stones Be Pain-Free?

We talked about the typical presentation about the patient who presents with flank pain. What about the patient that does not have any symptoms of back pain?

On occasion, there is the patient who obtains an imaging study for non-urological reasons. For example, a patient is being worked up for a gall-bladder stone. The individual obtains a renal ultrasound or a CT scan of the abdomen and pelvis, which reveals a kidney stone. This asymptomatic stone is referred to as the *incidental* stone (see Chap. 15).

On the other hand, there is the patient who is being worked-up for a known personal history of nephrolithiasis and the patient is found to have a stone on imaging studies. The typical scenario here is the person who has a known history of stones and comes to the office for their annual follow up with a urologist who ordered a renal ultrasound to rule out new stones. Here, the patient is found to have an incidental 5 mm renal stone, for example, and the patient is pain free.

Finally, there is the patient with a staghorn calculus. These are large stones that are limited to the renal pelvis and calyces. While the stone is large and occupies most of the area within the renal pelvis and/or calyces, it is too large to block or obstruct the renal pelvis. This is similar to water entering a person's basement; water seeks its lowest level. Similarly, urine finds its way around the stone and eventually passing down through to the ureter. Since there is no obstruction, the patient does not usually demonstrate any pain. Small stones on the other hand, have the potential to block the pipe and produce hydronephrosis and ultimately, flank pain. One might say

that *stone pain is inversely proportional to stone size!*

I have pain or blood in my urine, what should I do? (This section discusses the work up for patients with stones; symptomatic)

Hematuria, or blood in the urine, occurs in 90 % of patients. This hematuria may be visible to the naked eye, referred to *gross hematuria*. If the blood in the urine is "invisible" to the naked eye, but only visible under a microscope, this is called *microscopic hematuria*. Most cases of blood in the urine present as microscopic hematuria, however, gross hematuria can be reported. Blood in the urinary tract may be from the stone rubbing stone against the walls of the urinary tract. On the other hand, an obstructing stone may cause the kidney to swell, resulting in the stretching of small vessels along the lining of the kidney. This in turn, can result in bleeding once the blockage is relieved and the stretched vessels begin to leak as the vessels refill with blood. To satisfy your curiosity, *yes, you can get blood from a stone!*

While a patient with a history of stones presents with new onset of blood, this may very well be from a new stone. However, one should not lose site of the fact that the hematuria may be from a completely different source. Similarly, if it looks like a duck and quacks like a duck, don't forget that there may be a hippopotamus! Blood in the urine may have several different origins. Here, the differential diagnosis includes a renal mass, tumor of the ureter or even a lesion within the bladder or urethra. Other causes of hematuria include a renal cyst, urinary tract infection, pyelonephritis, glomerulonephritis (inflammation of kidney filtering system), sickle cell anemia, medication (aspirin, penicillin, heparin, cyclophosphamide and phenazopyridine), renal trauma and strenuous exercise (i.e., runners hematuria). In women, blood in the urine may be the result of endometriosis or contamination from menstrual bleeding. In this situation, a woman should have her urine retested following her menstrual period. In men, bleeding can originate from prostate cancer, prostatitis or the enlargement of the prostate, referred to as benign prostatic hyperplasia. It is paramount that you

refer with your urologist when presenting with hematuria so that the appropriate testing can be performed.

Other Symptoms Associated with Your Stone

While flank pain is the most recognized symptom associated with your stone, you should be aware of other symptoms. This includes nausea, vomiting, fever and/or chills. These latter symptoms may be related to a more serious consequence of your stone. Obstructing stones in the ureter, for example, may result in a stagnant flow of urine, resulting in *pyelonephritis*. If you recognize symptoms of fever and/or chills, please notify your physician immediately. These symptoms are critical, especially in cases of patients with immunosuppression. If you are taking medication to suppress your immune system (i.e., steroids or chemotherapy), diagnosed with diabetes or have HIV, symptoms associated with pyelonephritis should be recognized and treated immediately.

Patients may present with dysuria, or burning with urination. An individual who is straining to void slow and painful, small volumes of urine is referred to as *strangury*. Symptoms of dysuria and strangury can be associated with a stone within the lower urinary tract. However, be aware that other causes for these symptoms may include a urinary tract infection, sexually transmitted disease, urethritis, interstitial cystitis or bladder tumor. Consult with your urologist to do the appropriate testing to determine the etiology of these symptoms.

Conclusion: Got Stone?

In summary, your stone can present in a variety of ways. In one sense, you may present with the typical scenario of flank pain, with or without nausea, vomiting, fever or chills. On occasion, there may be the situation where the patient presents with hematuria with or without a UTI. On the other extreme, there may be the situation

where the stone is completely silent without any symptoms at all. There is an entire spectrum of symptoms associated with stones. Your urologist will work with you to decipher and make sense of your symptoms. Remember that your stone is talking to you; make sure you listen very carefully! Also, note however, it's the quiet ones that you need to be concerned about. It can be the *silent stones that are most deafening!*

Mr. JM is a 49-year-old female with a history recurrent UTI's for approximately 8 years. Her primary care physician had treated each of her UTI's with a therapeutic course of antibiotics. Shortly after completing the antibiotics, she repeated a urine culture demonstrating a new infection. Sometimes the infection was similar to the one before, but other times, it was different. On occasion, the urine culture demonstrated *Proteus Mirabilis*. The patient had occasional blood in the urine. She denied any history of flank pain, nausea, vomiting, fever or chills. It was not until the patient moved to a new town and was referred to a new primary care physician, that she had a new UTI infection. Only this time, the patient was referred to a urologist where she underwent a work up for her UTI and hematuria. The CT scan revealed a 5 cm right renal stone, the size of a tennis ball. The patient ultimately underwent a PCNL and her stone was successfully removed. She was surprised that she had no symptoms related to the stone. The patient was also pleasantly surprised that she weighed less after her stone was removed!

As we stopped for what seemed like the hundredth time on the way to the emergency room, I cursed the traffic lights for conspiring against me. My husband asked, as he did at every red light, "what hurts, exactly?" I replied the same way I had each

time he had asked. I eked out, through clenched teeth and waves of nausea, in a shaky, staccato voice, “My back. Or my stomach. I can’t tell. It just hurts.” My husband jokingly asked, “Are we going to be on that show ‘I Didn’t Know I was Pregnant?’” I shot him a look that confirmed that the question was ridiculous. If I weren’t wincing in pain, I would have rolled my eyes. Of course I wasn’t in labor. I had given birth twice before, and knew what that felt like. I began to think about my son, who was born at the same hospital that we were heading to at the moment. I did yoga and meditated through the entire labor and delivery. I declined all pain medication. The pain was intense, but manageable. I didn’t

cry. This pain, however, made me cry. It was unrelenting and no yoga pose or breathing technique quelled it even slightly. It felt worse than any stage of labor ever had. I began to wonder if maybe my husband’s question was not so ridiculous. Then, I panicked. If this pain is worse than an 8 lb 11 oz baby making his way through the birth canal, then what could possibly be going on in there? Upon arriving to the hospital, the reminiscences of my labor flashed before my eyes. The memories of my delivery were so similar to the throbbing pain I was experiencing. After my CT scan, it became clear to me how different things actually were. The doctor told me I was “expecting an 8 mm stone!”

David A. Schulsinger

Summary Stone Facts

- A stone identified during a work up for a separate medical condition is referred to as an *incidental stone*.
- Incidental stones can be managed on an elective basis.
- Be certain not to forget about the incidental stone.

This chapter discusses the work up of a patient with the incidental, asymptomatic stone.

The classic scenario for a patient with a kidney stone presents with symptoms that lead to a work up by their doctor or by the emergency room physician. For the patient with minimal complaints, the patient will see their physician in the office who will order the appropriate tests and imaging studies. For other patients, their symptoms of blood, flank pain, nausea and/or vomiting may require the individual to seek immediate medical attention in an urgent care center or the emergency area of the local hospital. Here, the patient will undergo a series of tests, including a physical examination, imaging studies and laboratory tests. The goal of these tests is to determine the size and location of the stone in the urinary tract. Additionally, imaging studies can aid in differentiating whether the

stone is calcium vs. radiolucent (uric acid). This will help in the treatment strategy for this patient as to whether medical vs. surgical intervention is required. These tests will also aid the physician as to whether there is any blockage in the urinary tract, referred to **obstructive uropathy**, and whether the patient is at risk for infection in the urine, or **UTI**; infection in the kidney, known as **pyelonephritis**.

On the other hand, there is also the group of patients without any urinary tract symptoms. These patients are without flank pain, nausea, vomiting, fever or chills. There is no gross blood in the urine. These are the patients whose urinary tract stone are identified **serendipitously**, also known as the **incidental** stone. Similar to the patient with symptomatic stones, these stones present in different shapes, sizes, composition and locations within the urinary tract.

The goal of treatment in these patients is similar to the individual with the symptomatic stone. The difference is, however, the lack of urgency to remedy this medical or surgical condition. These patients do not need to be treated ASAP, or at the time of the ER visit. These patients will generally follow up with a urologist on an elective basis for a scheduled appointment. There, the patient will meet with the urologist to discuss the various treatment options.

In my experience, some patients may experience complacency and not seek immediate or short-term solution to the stone. It is not unusual for these patients to follow up with me several

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

months or even a year after they were told about their stone. Patients admit to the syndrome “if it is not broke, don’t fix it.” Unfortunately, there are some patients who will “role the dice” and wait indefinitely to see a urologist. These are the patients who eventually go to the ER when their stone becomes symptomatic. These are the patients referred to as the “*stone procrastinators*”. These patients had an opportunity to address their asymptomatic stone, but needed to go to the ER once they became symptomatic with intractable flank pain, nausea, vomiting, fever and/or chills.

Indications to Treat

When a patient has a symptomatic stone, the situation to treat is better defined. In medical school and in residency, patients who present with nausea, vomiting, fever, chills and intractable pain are all absolute indications that a patient needs a procedure. As a Urology attending, I added an additional factor, called the rule of 2! If the patient has two or more stones that are sizeable and the stone burden (combined stone size) is greater than 10 mm or if the patient has had two or more visits to the ER, this is usually a relative indication of the need to treat. The number of kidney stones the patient has, becomes the issue. Murphy’s Law states that if it can go wrong, it will. In stone language that translates to if a patient has two stones that two stones will pass at the same time, as opposed to individually at separate times, potentially obstructing the ureter. The number of ER visits has become a quality of life issue where the patient has spent endless hours in the ER (sometimes 7–9 hours) having a work up for their stone.

For the patient with the asymptomatic stone, the indication for treatment is strictly elective (see section “[Treatment options](#)”).

Treatment Options

Treatment for the incidental stone can be done from an elective, medical or surgical approach. The key here is that treatment can be achieved electively. For the patient with the symptomatic stone, treatment many times is done emergently, especially if the patient cannot be managed with pain medication

and fluids in an emergency situation. This is referred to as **intractable pain**, when patients do not respond to conservative care management.

Small Stones

For patients with the small incidental stone (≤ 4 mm), patient can be managed by conservative care. Patients can continue with aggressive fluid hydration to pass their stone. These patients are followed on an interval basis determined by you and your urologist, typically with an imaging study (i.e., renal US with jets, KUB or CT scan) and possibly a urine analysis to rule out blood or infection. If your incidental stone is in the ureter, your urologist may give medication to help your stone pass. This is referred to medical expulsive therapy (MET).

Medium Sized Stones: 3 Procedures vs. 1 Scenario

What do you do with that asymptomatic, 8 mm stone? For the patient who has no stone-related symptoms and has a reasonable size stone, I always give patients several options. We can watch this stone and treat it conservatively. If the stone passes, we hit the homerun. If it gets bigger, we can pull the trigger and do a “pre-emptive strike” and perform an ESWL or ureteroscopy and stone manipulation procedure to remove the stone in a single or double procedure (i.e., remove a stent if stent is required). However, if you role the dice, and the stone becomes obstructed in the ureter, then a different scenario arises. Through the ER, your urologist will initially perform a stent placement procedure to unobstruct the kidney, a second and definitive procedure to treat the stone (i.e., ESWL or ureteroscopy), and the final and third procedure to remove the stent (usually in the office). This is what we refer to as the **3 vs. 1 rule**. Explained like this, most patients choose the elective, single procedure option!

Staghorn Stones

For patients with staghorn calculi (Fig. 15.1), the treatment options are more limited and better

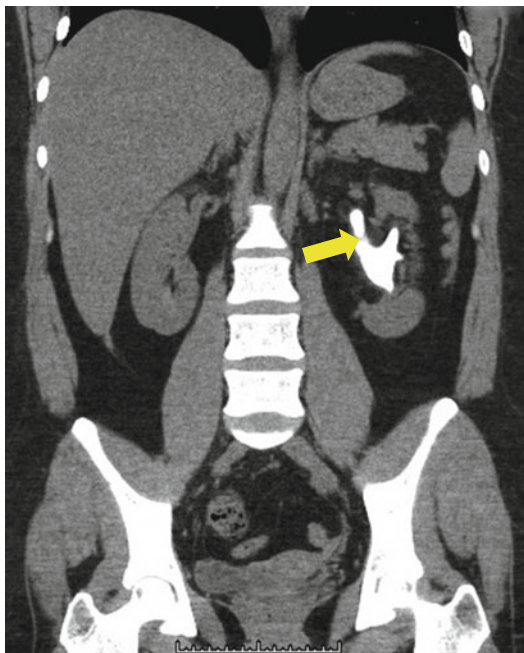


Fig. 15.1 Coronal CT scan image of a left renal staghorn calculus (yellow arrow)

defined. For the patient with an asymptomatic stone, the individual can plan for an elective percutaneous nephrolithotomy (PCNL) procedure where a nephrostomy tube and removal of the stone are performed during the same hospitalization. For the patient who presents to the ER with a symptomatic staghorn calculus, this patient may require a nephrostomy tube at the time of their ER visit (i.e., if they present with intractable pain or infection). Compared to the patient with the symptomatic stone who may require two or more procedures, the patient with the incidental stone may only require a single procedure during a single hospitalization.

Work Up for the Incidental Stone

Unlike the symptomatic patient who is getting an imaging study because of their stone, the incidental stone patient is identified due to a disease process being worked up for something other than their stone. These patient have stones found due to abdominal US working up a patient with a presumed gallbladder stone; a patient who had a chest CT who is being worked up for a respiratory condition; a kidney stone found on

lumbar sacral films when evaluating a patient's spine; a bladder stone found on a pelvic US during a work up for a patient with BPH; a renal stone found on an abdominal CT scan during a work up abdominal pathology.

Incidental Treatment: Planning Your Procedure from the Office vs. ER

The command center for treating your stone should be from your urologist's office, not from the ER. The ideal location to plan for your stone procedure is from the physician's office as compared to the ER or the holding area of the operating room. Once you are in the ER, your treatment options are limited. If you have an obstructing stone, you may require an initial procedure with a stone placement. If your obstructing stone is associated with fever, it may require that you have nephrostomy tube placement.

From the office, you can speak with your urologist from a position of strength. This elective office visit allows you to prepare your questions in advance. Your sensorium is not clouded, as it might be with taking pain medication in the ER, and questions can be properly addressed and understood. Without pain, you make an objective decision for choosing the best surgical treatment options for you. Planning your procedure allows the patient to have greater options of stone procedures for incidental stone, a greater chance of less invasive stone procedures for an asymptomatic stone and greater chance of less number of stone procedures for asymptomatic stones.

Remember, planning your procedure from the office, allows the patient time on their side. Like a football game, this is first and 10 with all your time outs left to plan your stone procedure; not fourth and 1 with no time outs left where your treatment options are limited.

Incidental Stone Prevention

Your incidental stone was treated either conservatively or possibly, electively. While your stone was identified incidentally, the risk for a recurrent stone still remains. Nevertheless, it is imperative for you to pursue preventive care and minimize

Table 15.1 Summary comparing symptomatic vs. incidental stones

Factors	Symptomatic stone	Incidental stone
Symptoms	Hematuria, flank pain, nausea, vomiting, fever or chills	None (not stone related)
Work up	CT scan, renal US or KUB	Determined by work up for other complaints
Initial treatment options	Stent or nephrostomy tube	All treatment options
Potential number of procedures	3	1 or 2
Type of surgery	Emergency, elective	Elective
Prevention	24 hour urine, stone analysis	24 hour urine, stone analysis
ER visits	Usually	No
Planning surgery	From the ER	From the office

your risk for future stones. It is still important for you to collect your stone and obtain a 24 hour urine (See stone prevention, Chap. 24).

You have completed the marathon and you are on the last leg of your journey. Your stone is gone, the course and timing of stone prevention is in the same.

Conclusion

In summary, incidental stones can come in all shapes and sizes. These stones present as asymptomatic calcifications. Incidental stones present much differently than symptomatic stones (see Table 15.1). Once an incidental stone becomes identified, do not make this the *forgotten stone*. While these stones are treated the same way as symptomatic stones, time is not of the essence. Over time, stones can become larger, the change in size depends on the etiology of the stone. Sometimes these incidental stones behave like a ticking time bomb, and if left forgotten, they can set off unexpectedly. Making a plan with your urologist to treat this stone before it becomes symptomatic, is the best medicine of all. Having the time and ability to plan the appropriate treatment option for your stone will give you the luxury to sit down with your urologist and allow you to make the best

decision. The planning and timing of your treatment options are the ingredients for success. Remember, incidental stones may still need to be treated, but time is on your side!

Story #1

Mr. TR was a 56-year old white male when he went into renal failure secondary to complications associated with diabetes, a condition known as **diabetic nephropathy**. He began hemodialysis 3 days/week. He was placed on the transplant list and was told the average waiting time for a kidney was 2 or more years. He was optimistic, but at the same time discouraged that he would be attached to a machine every other day for 6 hours at a time awaiting a kidney. During the work up to identify parameters necessary to find a good match for his renal transplant, he was pleasantly surprised to find out that his wife was a 4-antigen match (i.e., good match). Typically, the donor undergoes a CT angiogram to better understand the blood supply of the kidney, especially if there are multiple arteries to that need to be recognized when harvesting the kidney. The CT angiogram demonstrated a single right renal artery and vein, the ideal anatomy for the transplant surgeon harvesting the kidney from the donor. However, there was another surprise the couple was told about during the screening office visit. The wife had an incident 5 mm stone in the midpole of the right kidney. Ideally, one would not want to transplant a kidney packaged with a stone to a recipient receiving immunosuppressive medication, putting the patient at risk for an infection, and potentially jeopardizing the transplanted kidney. The patient, therefore, underwent a ureteroscopy and stone manipulation, making the patient stone free, and the kidney optimized for transplant. 6 weeks later, the stone-free kidney was transplanted into the recipient, as he remains free from dialysis. And, oh, yes.... he remains stone free!

Story # 2

I knew that I had serendipitous stone. It was discovered several years ago when my doctor ordered an ultrasound of my liver for elevation of my liver function tests/lumbar spine series for lower back pain. It demonstrated small renal stones. My doctor told me that they were small and did not need surgery since they were not causing me pain. Several months later, I wound up having a DVT and my physician started me on Xarelto and Aspirin. One week later, I began experiencing gross blood in the urine. It frightened me to see of the “Hawaiian punch”-colored urine. Needless to say, I had the million-dollar work for hematuria, which included a series of urine tests, CT scan and an office cystoscopy. All of these tests confirmed that I had a stone and that was not any sources for my stone. Needless to say, had I listened to my Urologist I could have avoided all these tests.

Story # 3: Oral Board Exam Question

The oral examination in urology is the final exam that you take to become board certified in Urology. The requirements are that you passed part 1 examination, a written exam after successful completion of your urology residency, you have been in practice for 18 months, submitted a log of all patients you operated on and a review of complications you encountered. If that is not enough, a committee meets to review your application prior to the exam to review the number of surgical

cases and variety of cases performed. You receive a letter determining your eligibility. If denied, you wait another year to go through the application process again. On the day of the exam, usually in Dallas, Texas, you and your fellow colleagues are sequestered in a large room waiting for your name to be called. An escort will take you and a group of others to a floor in the hotel where you report down the hall in single file. As you pass a room, a name is read for the eligible examinee. On the door of the room is the name of your examiner. You enter the room where the examiner is sitting at a table with two chairs; one made especially available to you. A 6" cardboard barrier separates you and examiner as he reads the questions to you. There are only two questions and you have 15 min. They read a single sentence scenario and you describe the appropriate tests, work up and history and physical exam you want to do. Anything you say, right or wrong, he has an answer for. My first question was a 23-year old male with history of blood in the urine. After doing the PE, history and physical and appropriate tests, it was determined that my patient had an asymptomatic 3 mm right renal stone. Given its small size, the correct initial response is to do conservative care and watch the stone. If you operated, given its size, you would probably fail that question. They actually wanted you to watch this two stone for a couple of rounds, before the stone became symptomatic and the examiner pulled your hand and ultimately forced you to operate on this fictitious patient.

Andres Pena and John A. Ferretti

Imaging Summary Facts

- Non-contrast CT is the most rapid and accurate technique for evaluating kidney stones.
- In pregnant patients with flank pain, renal ultrasound (renal US) is the best initial study.
- An X-ray of the Kidneys, Ureters and Bladder (KUB) is a follow-up method to see if the stone has passed or advanced. A KUB can be used in combination with an US to determine if the kidney stones are radiopaque vs. radiolucent.
- MAG-3 Renal Scan can be used to evaluate renal function.
- MRI has a limited role in the evaluation of kidney stones and in general is not used for this purpose.

also dictate the most appropriate treatment to follow [1].

There are different ways to work up kidney stones; some of these methods involve the use of x-rays such as CT scan, KUB (an X-Ray of the Kidneys, Ureters and Bladder), and Intravenous Pyelogram (IVP) [2]; others use sound waves, such as Ultrasonography [3]; MRI is another method, which uses a big magnet in order measure the water density in tissues and produce an image.

After a stone is found, depending on its specific characteristics, it may be treated medically with Shock Wave Lithotripsy (SWL), that uses shock waves to break a kidney stone into small pieces that can more easily travel through the urinary tract and pass from the body, or removed by Percutaneous Nephrolithotomy (PCNL), where the surgeon makes a small incision in your back to remove kidney stones. He or she then puts a hollow tube into your kidney and a probe through the tube.

Initially patients are followed with KUB and ultrasound. The KUB can detect larger stones but clinically significant fragments may not be detected and a non-contrast CT will find them [4]. If the physician is worried about recurrent or residual stones as stated earlier a non-contrast CT followed by a second set of images obtained after intravenous dye (contrast) may be performed to determine the best treatment.

The diagnosis and initial management of Urolithiasis have undergone considerable evolution in recent years. Technological advancements have greatly facilitated the diagnosis of

Introduction

Urolithiasis is the condition where kidney stones are formed anywhere along the course of the urinary tract. Diagnostic imaging is used in patients with symptoms suggestive of renal stones to confirm that Urolithiasis is the source of the patient's pain and to identify the location, size and possible complications of the kidney stones. It will

A. Pena, MD (✉) • J.A. Ferretti, MD
Department of Radiology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: John.Ferretti@stonybrookmedicine.edu

stone disease. The purpose of this chapter is to summarize the different types of imaging available to diagnose stone disease, including the advantages and disadvantages of each of them.

Computed Tomography

Computed tomography (CT scan), is a technology that combines a series of X-ray views taken from many different angles and computer processing to create cross-sectional images of the bones and soft tissues inside your body. The

images can be viewed as a horizontal cut (i.e., axial); from front to back (i.e., coronal); or from the side (i.e., sagittal) view (see Fig. 16.1). The resulting images appear as virtual slices that can be compared to looking down at single slices of bread from a loaf.

Non-contrast CT is the preferred initial study in patients with flank pain with a clinical suspicion of a ureteral calculus. Other indications include a Patient with ureteral calculus who is successfully treated with medical expulsion therapy and continues to have hydronephrosis (swollen kidney) by ultrasound. A third indication is

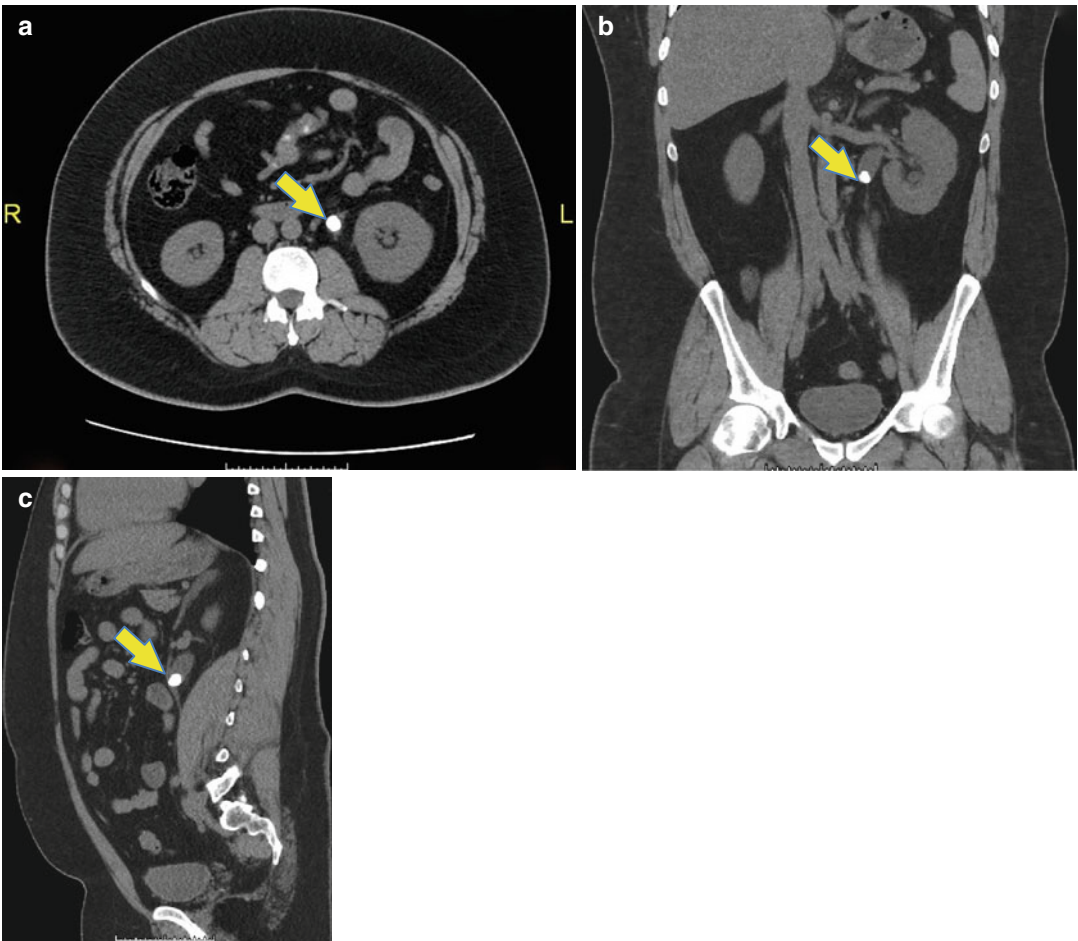


Fig. 16.1 Multiple non-contrast renal protocol CT scan images of a patient with a ureteral stone in the proximal (*upper*) third of the left ureter (*yellow arrows*) in the (a) axial, (b) coronal and (c) sagittal views

for patient's who have ultrasound evidence of hydronephrosis and who have recently passed a kidney stone [5].

A CT with IV dye (contrast) is performed after a stone has been removed surgically and the patient still has pain. It is also performed in patients who have an abnormal ultrasound (hydronephrosis) at the follow up after stone removal, even if the patient does not have pain [5]. The contrast dye CT is only performed if the patient's overall kidney function is near normal and if the patient is not allergic to the dye itself.

The advantage of non contrast stone protocol CT is that it is very accurate [6] and it does not take long to perform or interpret. The patient will receive the appropriate treatment in a timely manner, ultimately reducing cost [2].

The disadvantage of non-contrast CT scan is the radiation exposure that although relatively low, can accumulate in patients who are recurrent stone formers. A low dose CT in an adult has an effective dose of approximately 4 mSv [5] and a standard non contrast CT has an effective dose of 10–30 mSv [7]. It is not ideal to use this method in young children and pregnant woman because there is ionizing radiation, which is a concern [1, 5]. CT without contrast will detect the most common types of stones, although kidney stones are composed of different substances and the X-ray does not detect a minority of these.

Renal Ultrasound

Renal ultrasound – Renal US, is an oscillating sound pressure wave produced by a probe with a frequency greater than the upper limit of the human hearing range. A transducer collects the sounds that bounce back and a computer generates images.

Renal ultrasonography is the preferred initial imaging modality for children with suspected renal stones, pregnant women and recurrent stone formers.



Fig. 16.2 Longitudinal gray scale (renal ultrasound) image of the left kidney shows hydronephrosis (yellow arrow) within the kidney (+)

The advantage of ultrasound is that it does not use ionizing radiation, which is ideal for imaging pregnant women, children and recurrent stone formers. The sound waves detect kidney stones and indirect findings such as a swollen kidney, called *hydronephrosis* (Fig. 16.2), that will let the doctor know if there is obstruction or not.

The ultrasound can let us know if a stone is obstructing the flow of urine even if the stone is not seen, by detecting the urine flow within the urinary bladder, referred to as ureteral jets (Fig. 16.3). For example, if there is strong flow from the right kidney but only minimal flow from the left kidney, there is likely a stone on the left [3].

The disadvantage of ultrasound is that it is machine and Operator dependent which can lead to different results (sensitivity of 19–93 % and specificity of 84–100 %) [8]. Ultrasound will miss over 30 % of acute obstructions due to kidney stones in patients who are not specifically hydrated for the study [7]. Another disadvantage is that ultrasound usually cannot detect stones that are not close to the kidney or the urinary bladder.

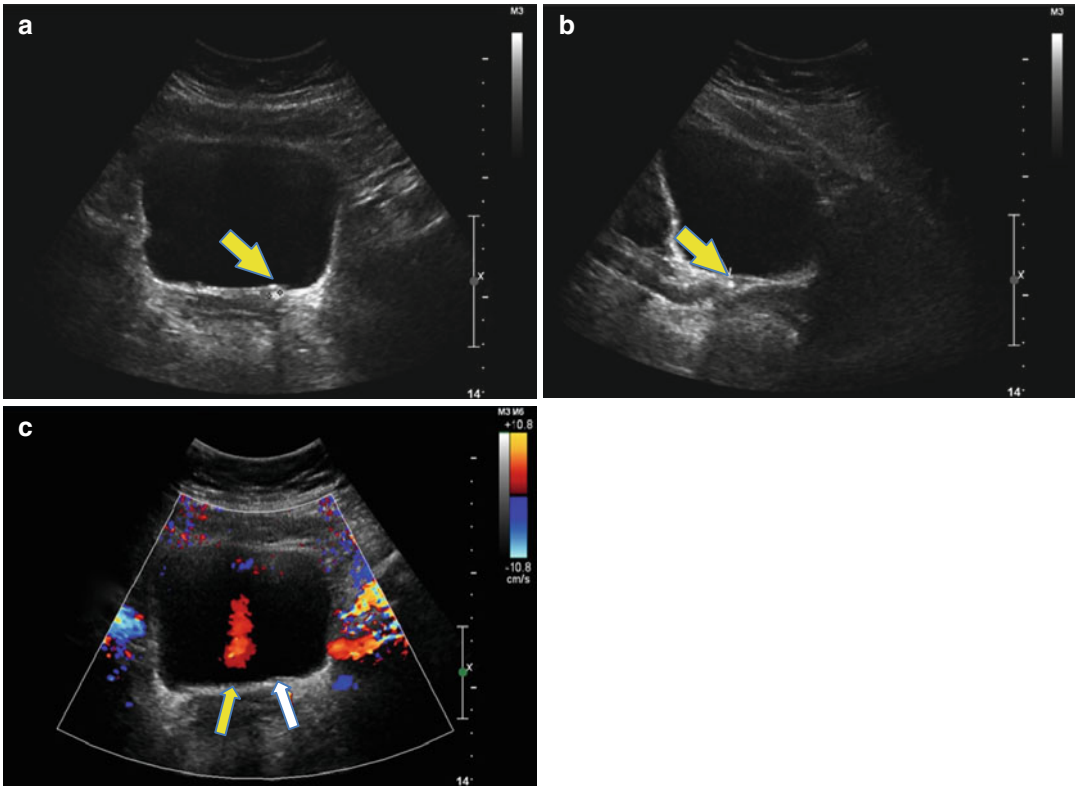


Fig. 16.3 Urinary bladder ultrasound image demonstrating left UVJ stone (*yellow arrow*) in the (a) transverse and (b) longitudinal view. (c) Ureteral jet (urine flow)

demonstrated in red from the right ureter (*yellow arrow*) and the absence of a jet from the left ureter (*white arrow*)

Intravenous Urography

Intravenous Urography (IVU), also called intravenous pyelogram (IVP), uses intravenous contrast (a dye), that will be excreted or removed from the bloodstream via the kidneys, and allows the visualization of kidney stones in the X-rays (Fig. 16.4). Currently this procedure is rarely performed or indicated, but it is the best investigation tool if “non-contrast CT” is not available [7].

A disadvantage of an IVP is that to have good images, the bowel needs to be prepared before the study can be performed and this significantly delays care. Also, IVP may tell you if you have a kidney stone or not but will not help make an alternative diagnosis that may actually be the source of pain [9]. These facts added to the

potential of adverse reactions to the dye and the widespread availability of CT has made IVP a study that is now rarely performed [10].

KUB

KUB is a plain-film radiography of the abdominal area and stands for **k**idneys, **u**reters and **b**ladder. KUB may be sufficient to document the size and location of radiopaque urinary calculi, which are stones that contain calcium, such as calcium oxalate and calcium phosphate stones (Fig. 16.4a).

The radiation dose of a KUB 0.1–1 mSv [7] is much lower than that of a stone protocol CT 4–30 mSv [5, 7]. The low radiation dose of a KUB makes it a good study that can be performed

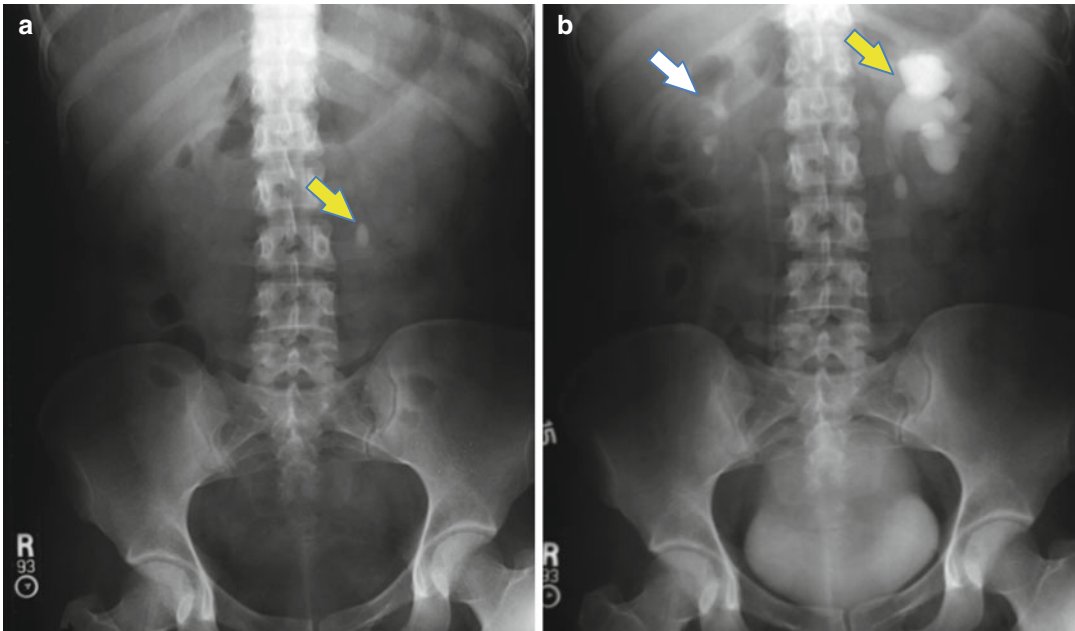


Fig. 16.4 (a) Scout film demonstrating a radiopaque (visible) stone (*yellow arrow*) seen in the area of the left ureter; (b) IVP demonstrating obstructed left kidney (*yellow arrow*) and normal drainage of right kidney (*white arrow*)

after a stone was found on CT and it is of a size that the body can eliminate without surgery with medical expulsive therapy (MET) [5, 11, 12]. The KUB is also used as a follow-up method to see if the stone has passed or advanced. One of its advantages is that it is easy to perform.

A KUB can also be used in combination with ultrasound in chronic stone formers or to follow up patients that have received surgical treatment for stone removal. The combination of KUB with ultrasound will help identify the stone and determine if the stone is radiopaque or not. This may eliminate the need for a CT; however it is only performed if the stones have been radiopaque (detected by x-ray) in the past.

A disadvantage of KUB is that a significant amount of stones are too faint to be detected by this modality. Less radiopaque calculi, known as Radiolucent stones, such as pure uric acid stones and stones composed mainly of cystine or magnesium ammonium phosphate, may be difficult, if not impossible, to detect using a KUB. Also, stool or air may be along the ureter in the expected region of the stone and thus further limit the visualization of significant stones.

MRI

The MRI uses a big magnet in order to measure the water density in tissues and produces an image. It doesn't produce ionizing radiation.

In rare cases the MRI is the imaging modality of choice for kidney stones as the stones are difficult to visualize using this technique [10]; An advantage of the MRI is that unlike x-rays (CT, KUB) that create the risk of injuring the fetus [5], the MRI does not use ionizing radiation to produce images, so it can be used in the first trimester of pregnancy, when the ultrasound has failed to detect a stone and the clinical suspicion is high.

MRI has also been shown to be useful in differentiating normal physiologic dilatation of the kidney's collecting system associated with pregnancy from pathologic dilatation due to a kidney stone [13]. MRI can help in an alternate diagnosis when the source of the patient's pain is not due to a kidney stone. It has excellent depiction of other organ systems such as liver, gallbladder, biliary system and pancreas. MRI images have good soft tissue contrast and organs can be viewed in different imaging planes.

Retrograde Pyelogram

Retrograde Pyelogram (RP) is a procedure where the doctor injects contrast into the ureter (Fig. 16.5). This then flows, as the name states, in a retrograde fashion up the ureters until it reaches the kidneys. During the administration of this dye a type of x-ray, called fluoroscopy, is used to produce images. This allows for the visualization of stones. It will also show if there is a scar in the ureter, called a *stricture*, which may be impeding the normal flow of urine.

A RP is also useful in the following cases: If there is suspicion of a tumor that may cause narrowing and there is a need to know if the urethra is obstructed; to evaluate hematuria; to assist in percutaneous access by delineating the entire collecting system (nonhydronephrotic kidneys) and to aid in stent placement and ureteroscopy.

An advantage of RP is that it does not depend on the patient producing urine to opacify the urinary system. With a RP, you can evaluate the length of the structure. A disadvantage is that RP is an invasive procedure performed under anesthesia and uses x-rays with a dose of approximately 1–10 mSv.

MAG-3 Renal Scan

It is a nuclear medicine study that involves administering small amounts of radioactive substances, called tracers. When imaging modalities such as CT or ultrasound show a swollen kidney (i.e., hydronephrosis) but it is not clear if this kidney is still producing urine and doing its job of filtering the blood, a nuclear medicine study can be performed using Tc-99 m MAG3 to allow the doctor to know if the kidney is functioning or not.

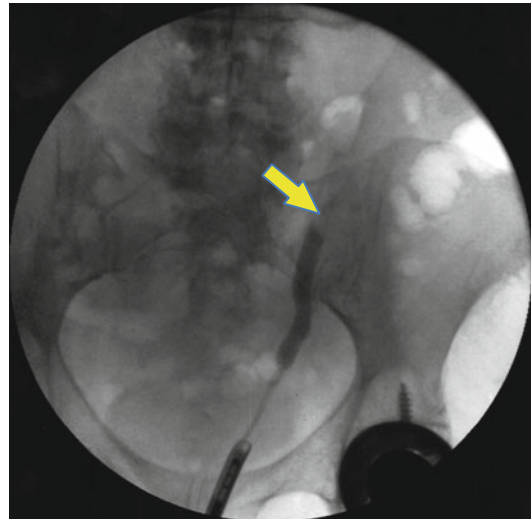


Fig. 16.5 Retrograde Pyelogram showing contrast injected into the left ureter. There is a filling defect (absence of contrast) in the ureter (yellow arrow) due to the presence of a ureteral stone

Before this test is done the patient is given fluids to hydrate and then injected with the tracer. Usually a diuretic will be given to see how the kidney responds. This tracer can be detected by the camera and thus give functional information that may change the treatment plan.

A nuclear renogram is useful when questions arise about obstruction of a kidney (hydronephrosis) (Fig. 16.6), compromise of a kidney's blood flow, or relative function of a kidney. The disadvantage is that it has very low resolution. They are really just a collection of black dots. A nuclear renogram may detect obstruction of a kidney, but the films would not indicate the specific anatomy of the kidney, renal pelvis or ureter. An IVP would better show such anatomy in a kidney with relatively good renal function.

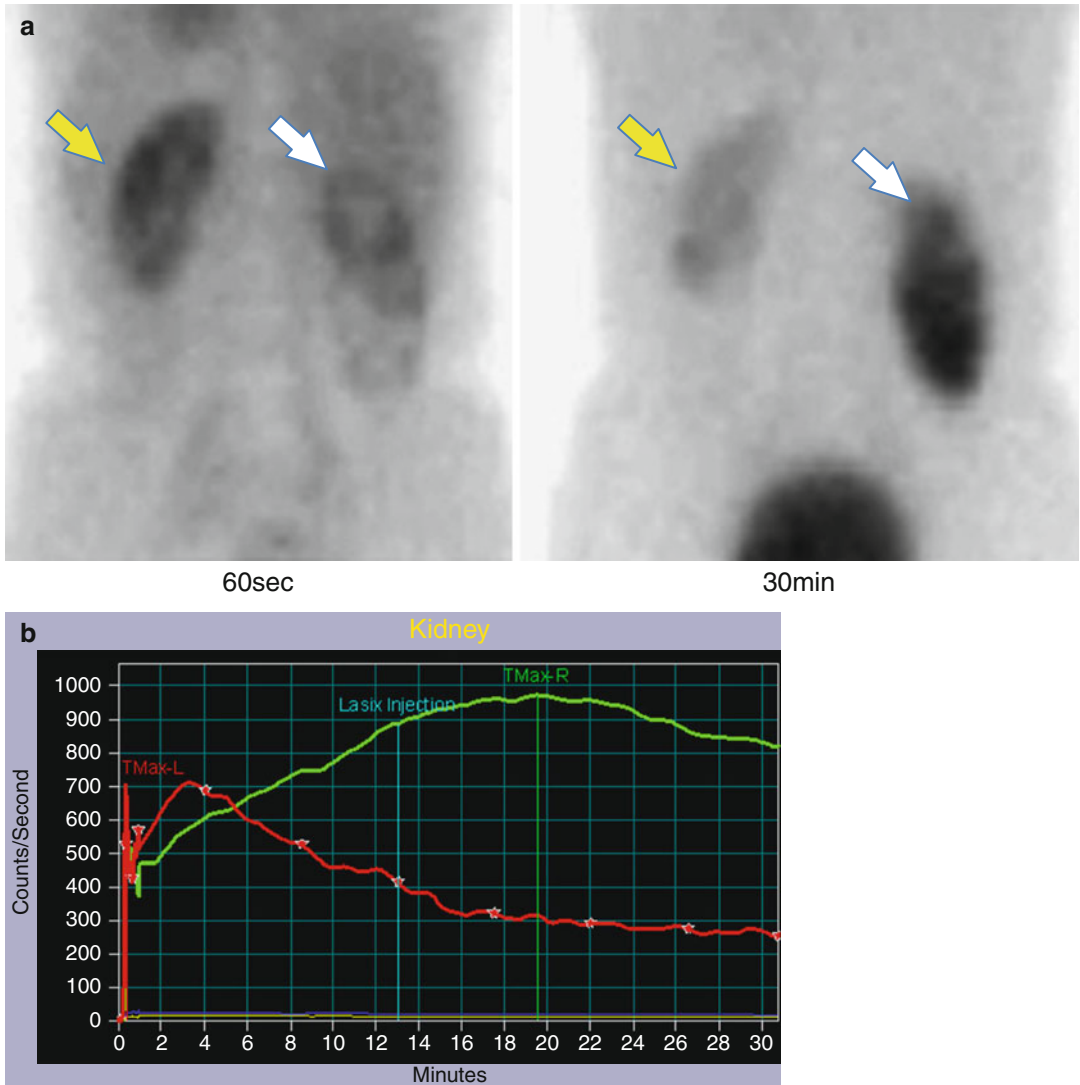


Fig. 16.6 (a) Nuclear Renal Scan. The *yellow arrow* points to the right kidney and the *white arrow* points to the left kidney. The left kidney is taking up and filtering the tracer normally between 60 s and 30 min. The right kidney demonstrates a faint shadow at 60 s and strong shadow at 30 min due to the obstruction of kidney from a stone.

(b) Nuclear Renal Scan report. The graph can be used to evaluate split kidney function by comparing the right and left kidneys. The *red line* represents the function of the left kidney; it goes up and down as expected from a normal kidney. The *green line* represents the right kidney; it shows a rise in the curve; this means that the kidney is obstructed

Conclusion

Physicians can now conclusively identify stone disease within minutes of considering the diagnosis. In recent years, technological advancements such as the ones described in this document have greatly facilitated the diagnosis of stone disease. The management of Urolithiasis is also becoming increasingly well defined. Your Urologist and Radiologist will work with you taking into account your specific needs.

References

1. Cheng PM, et al. What the radiologist needs to know about urolithiasis: part 2 – CT findings, reporting, and treatment. *AJR Am J Roentgenol.* 2012;198(6):W548–54.
2. Pfister SA, et al. Unenhanced helical computed tomography vs intravenous urography in patients with acute flank pain: accuracy and economic impact in a randomized prospective trial. *Eur Radiol.* 2003;13(11):2513–20.
3. Webb JA. Ultrasonography and Doppler studies in the diagnosis of renal obstruction. *BJU Int.* 2000;86 Suppl 1:25–32.
4. Park J, et al. Effectiveness of noncontrast computed tomography in evaluation of residual stones after percutaneous nephrolithotomy. *J Endourol.* 2007;21(7):684–7.
5. Fulgham PF, et al. Clinical effectiveness protocols for imaging in the management of ureteral calculous disease: AUA technology assessment. *J Urol.* 2013;189(4):1203–13.
6. Heneghan JP, et al. Soft-tissue “rim” sign in the diagnosis of ureteral calculi with use of unenhanced helical CT. *Radiology.* 1997;202(3):709–11.
7. Coursey CA, et al. ACR Appropriateness Criteria(R) acute onset flank pain – suspicion of stone disease. *Ultrasound Q.* 2012;28(3):227–33.
8. Ray AA, et al. Limitations to ultrasound in the detection and measurement of urinary tract calculi. *Urology.* 2010;76(2):295–300.
9. Smith RC, et al. Acute flank pain: a modern approach to diagnosis and management. *Semin Ultrasound CT MR.* 1999;20(2):108–35.
10. Cheng PM, et al. What the radiologist needs to know about urolithiasis: part 1 – pathogenesis, types, assessment, and variant anatomy. *AJR Am J Roentgenol.* 2012;198(6):W540–7.
11. Ege G, et al. Can computed tomography scout radiography replace plain film in the evaluation of patients with acute urinary tract colic? *Acta Radiol.* 2004;45(4):469–73.
12. Fielding JR, et al. Unenhanced helical CT of ureteral stones: a replacement for excretory urography in planning treatment. *AJR Am J Roentgenol.* 1998;171(4):1051–3.
13. Kalb B, et al. Acute abdominal pain: is there a potential role for MRI in the setting of the emergency department in a patient with renal calculi? *J Magn Reson Imaging.* 2010;32(5):1012–23.

Part V

Treatment

David A. Schulsinger

Who Should Be My Urologist? Pick Your Doc to Break Your Rock!

Just as all stones are not created equally, not all Urologists are trained the same! One should know that in 2013 there were 29,171 first-year residency positions offered to graduating medical students [1] and that there are only 239 urology resident spots each year. This number accounts for only 0.82 % of graduating medical students who go on to do a urology residency. With the few number of urology residencies, typically in university programs, urologists are generally well trained. They are trained to do general urology after completing 1–2 years of general surgery and 3–4 years of urology residency. If a graduating urology resident wants to pursue further training in a particular subspecialty of urology, then they would pursue a fellowship. The fellowship for stone disease is an Endourology fellowship. In summary, a Urologist who has been training longer will generally have greater experience.

You will see that there are some very important steps that you are going to need to follow when it comes to choosing the best urologist. Taking the time, if time allows for it, to choose the best urologist will be critical for your long term well-being.

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

In order to find the best doctor you are going to need to start by making a list of all of the different qualities you want in a doctor. You can also make a list of questions that you can ask the doctor to help you find out who is going to be the best. Some of the most important questions that you are going to want to ask will be related to how long the doctor can spend with you when you are in their office, if you can contact them by email if you need to, as well as how long you must wait to see them for an appointment. All of these answers will help you determine who is going to be the best doctor for you.

You may have your stone diagnosed in the ER, by your PMD or nephrologist and now it is time to choose your urologist. You want to feel confident in your decision as choosing a urologist will be critical in making the right decision on treating your stone, getting you stone free and keeping you stone free.

Unlike the circumstances of choosing your primary care physician, choosing a urologist is not always an elective situation. Circumstances prevail that in an emergency situation, you may only get to see the urologist on call for treating an obstructing stone. If a stent is placed or a nephrostomy tube inserted, then you have an elective period for which to choose your ideal urologist for the definitive procedure.

Finally, you will always want to write down notes about the doctor and the office when you are finished meeting with them. Keeping a diary is a great way to remember which experiences were positive and which ones may have been less than positive.

One of the most important things that you are going to want to look for when you carry out a doctor search will be the type of insurance that the doctor will accept. This is important because you are going to see that your visit may not be covered, if the doctor does not participate with your insurance company. In this situation, you will likely be faced with paying for your visit out of your own pocket. Other times the doctor may not accept you as a patient if you do not have a certain type of insurance.

Consider next, the amount of time that you might find yourself waiting on the doctor when you go in for the visits. There are some doctors that will make you wait in the waiting room for an extended length of time. Most of the searches that you are doing are going to give you reviews that will also mention the average waiting time that you can expect when you go to the office.

How to Pick Your Urologist?

Questions you should ask:

1. Where is the urologist located? Is his office accessible?
2. Which hospital is the urologist associated with? Are you comfortable with the possibility that this facility may be the location of your future surgery?
3. Is there a significant period of time for you to get an appointment with your urologist? If you are having pain, waiting any period of time may not be acceptable.
4. Is the office conscientious of your needs?
5. When you have questions regarding your care, does the staff (secretary, nurse, PA, physician) respond to your calls in a timely fashion?
6. If your urologist is away, is there another urologist in the group or another group that will be covering for him. If your urologist is not there, are you comfortable with an associate of your physician taking care of you? If outside coverage occurs, are you agreeable to having another urologist take care of you?
7. Does your Urologist practice preventive care of stones?
8. Does he have good outcomes?
9. Does the Urologist offer all treatment options for kidney stones? Some Urologists will not do PCNL's and may refer this procedure to another Urologist. Will you be OK with this if this happens?
10. What is his position's level of expertise? Is he a general urologist? Or does he have a specialty in kidney stones? There is a sub specialty of urology referred to as Endourology, which means treating urologic conditions by minimally invasive approach.

Helpful facts when choosing a Urologist:

1. Seek the opinion of friends, family and physicians about a Urologist who they know or used as a physician. This may be your best source of referral.
2. Choose a Urologist who has a good personality. A good bedside manner is critical.
3. Choose a Urologist who is technically good.
4. Recommendations from coworkers and neighbors.
5. Level of training.
6. Level of expertise with stones.
7. Fellowship trained in Endourology.
8. Preventive care of stones. It is important that you be cognitive and understand that in choosing your urologist, you have the team in place to help reduce the risk of future stones. Choosing your "A"-Team or Dream Team: (Urologist, Nephrologist and Nutritionist). In a perfect world, you would ideally have your urologist for treating your stone, your nephrologist for treating the medical aspect of your stone disease and finally the nutritionist, who could recommend a dietary plan that would reduce your risk for future stones. There are urologists, however, in an Endourology practice that specialize in stones in urology office that will implement your care your.

Most of the stone procedures that are performed are done from a noninvasive or minimally invasive access. The next important factor is preventive care. There are some

urologists that will treat your stones disease by the necessary surgical or medical approach. However, it is important that your stone is not only treated on the front end by the surgical or medical procedure, but that preventive care is utilized to help prevent your risk factors for stone disease. It's important to know that stone recurrence is approximately 50 % and the risk of stone recurrence can be minimized if workup is implemented to identify what your risk factor for treatment but the preventive care as well.

9. Choosing the appropriate stone doc now will pay dividends in the future:

Choosing the appropriate stone physician, or Endourologist, will pay dividends in the future. This is important if you do make future stones that you have an established relationship with that urologist so you can be treated in a manner that is comfortable and will reduce your fear and anxiety since you'll have an understanding and relationship with that urologist. In addition, there may be other urologic conditions in your future that may require additional urologic care. For example for a female who may develop overactive bladder, may require medical or behavioral therapy that could be treated by the urologist. On the other hand, a male patient that may develop prostate cancer would like access to the urologist to see what the various treatment options are for treating his prostate cancer. Therefore establishing a good relationship with your urologist now will allow the treatment of your stone disease and future potential urologic conditions.

In the Heart of the Matter: Get a Second Opinion

If your physician says that you need a surgical procedure, first things, first is to get a second opinion.

Do not be intimidated to question your urologist and say you want a second opinion. Many patients do not speak up because they worry that

they will offend their surgeon. In fact, most physicians welcome second opinions, especially when it pertains to surgical procedures.

A reasonable approach to initiate the process is to ask your urologist for a referral. You can also ask friends and family who have had similar conditions requiring treatment. If you decide to seek a second opinion, have your physician send all of your records to the new urologist. Be sure to ask the second opinion doctor to send the diagnosis to your original doctor.

Seeking a Second Opinion

Finally, it's important to understand that if you choose a urologist, that this is not a permanent relationship. It is ideal to have a "marriage" with your urologist that is a good long-lasting and "healthy" relationship. However, if your relationship does not work out, you do have the opportunity to divorce your urologist. If you, in fact, fire your urologist, and are looking to seek another, it's not unusual or uncommon to seek a second opinion. If you feel uncomfortable approaching the first urologist, his first urologist (take this out) to obtain your records, you can simply send a letter or fax stating that you would like to get a second approach and request that your records be forwarded to urologist #2. This is certainly an acceptable approach and there should be no hard feelings.

It's most important to realize that your health and your well-being are the primary condition in choosing a good urologist. Well established for your future urologic needs.

Begin by telling your first opinion doctor that you'd like a second opinion. Any doctor worthy of the practice of medicine will be supportive. If he is not supportive, then this is a red flag and this means that you should most definitely need a second opinion.

Why? Your goal is to get the most objective opinion about your urologic condition and treatment options as possible. Doctors tend to refer patients to their friends and acquaintances, even to a colleague in their same practice. They may

play golf together, belong to the same clubs or associations, have offices down the hall from each other, eat lunch together; there are many opportunities for them to build relationships.

That means you'll need to begin from scratch. You'll need to find the names of doctors who fit your needs (probably specialists), research their qualities and capabilities, and determine whether or not they will take you on as a patient.

Talk to other patients, including family, friends or colleagues who have been diagnosed with similar types of medical problems and ask about their experiences. Ask other healthcare workers – non-physicians – for their opinions. Keep in mind this one caveat: Just because a doctor is nice or has a good bedside manner, doesn't make that doctor a good clinician. It's great if you can have both! But be prepared to settle for the Urologist who is the better medical practitioner.

What Type of Procedure Will You Have?

There are several procedures that you may have to treat your stone. This depends on the type of stone, where it is located and the size of the stone. The treatment options include medication, ESWL, ureteroscopy and PCNL. Please see Chap. 20 for a description of the various surgical treatment options.

Where Will Your Stone Be Treated?

There are several options for where your stone will be treated. However, depending upon where your urologist is affiliated, what technology you have available, may dictate where your procedure will be performed. Stone procedures can be performed in the hospital, the ambulatory center, dedicated stone facility or even a truck. Some health care facilities that do not own a lithotripter will lease a shock wave lithotripsy machine that is transported by truck to the facility. In these

cases, the patients sometimes will have the procedure performed on the vehicle.

When Your Stone Should Be Treated?

There are two scenarios in which your stone will be treated. Most procedures are scheduled electively, however, some stone cases are performed emergently.

Most stones are treated electively. These are patients who have visited the urologist in the office, in the Emergency Room or even both places, and now are scheduled for an elective procedure. Typically, these are patients without pain or their pain is managed with pain medication. The urologist has discussed the various treatment options with you and now you are scheduled for the elective procedure.

In some situations, the case cannot be performed electively. The patient is seen in the emergency room. They are started on IV fluids and receive pain medication. Despite conservative methods, the patient continues to have intractable pain, nausea, vomiting, fever or chills. In this scenario, the patient will require an emergency procedure. In this situation, the patient may receive a stent or nephrostomy tube to decompress the kidney or reduce the pressure on the kidney so that urine can be drained from the kidney that was blocked from the stone. The patient may wind up with a staged procedure. This implies that the system is decompressed with a stent and then they have a definitive procedure to eliminate the stone.

Emily is a 56 year old teacher and mother of two boys who had an established history of stones. She had passed multiple stones and underwent several stone procedures. Some procedures were done electively and others were done through the emergency room. When it came to stones, she was a "stone veteran!" During a routine follow

up visit, she had a renal ultrasound demonstrating a 5 mm stone in the mid pole of her left kidney. She had some intermittent colic but was currently asymptomatic. She was petite, and has passed only small stones in the past. I discussed with her the various treatment options for this stone, including watchful waiting versus ESWL versus ureteroscopy and stone manipulation. We discussed the risks and benefits of all the treatment options. Knowing she was conservative by nature, I thought that she would lean toward conservative therapy in hopes of passing the stone with fluid hydration and medical expulsive therapy. When it came to stones, however, she was much

more aggressive. I will go with the ESWL now instead of an ESWL later. I would rather have a single ESWL now rather than waiting until I have pain to have an ER visit with a stent placement, followed by an elective ESWL and a third procedure to remove the stent. "Let me see: 1 procedure versus 3 procedures, it's a no brainer!" Let's go with 1 ESWL!

Reference

1. Ricks D. Medical students get 'Match Day' assignments. Newsday. 15 Mar 2013.

Meeting Your Expectations: *What to Anticipate Before, During and After Treatment?*

18

David A. Schulsinger

Preparation for Your Stone Procedure

Prior to surgery, preparation for your procedure is critical. The following recommendations are suggested to make your procedure less stressful and more uneventful. When considering these options, think of **MEDICALLY SAFE**:

1. **Medication:** bring a list of all medications you take with you the day of the your pre-admission testing and procedure. This includes prescribed medication, over the counter (aspirin, ibuprofen) and nutritional supplements/vitamins (Vitamin E).
2. **Emergency:** be prepared for delays on the day of your procedure. You are having an electively scheduled procedure. Emergencies and extended periods of waiting can occur resulting in a potential delay of your procedure.
3. **Driver:** Always have a family member, friend, significant other, co-worker or acquaintance to be available to drive you home and stay with you after the procedure, as you may have impaired judgment or fatigue as a result of the anesthesia.
4. **Instructions:** Follow all medication instructions given to you by your physician and/or pre-surgical testing center, the morning of your procedure with a minimal amount of water.
5. **Complimentary Medication:** Please review any herbal supplements at your pre-operative visit. Stop taking Vitamin E, Fish Oils, Ginkgo biloba, 7 days prior.
6. **Alcohol:** do not drink any alcohol 24 hour prior to surgery. Abstain from smoking too.
7. **Legal:** Make sure your physician reviews with you an informed consent. Know the risks, benefits and alternatives of your procedure, which should be discussed with you by your physician before you go for any procedure/surgery.
8. **Loot:** leave all valuables, money, cell phones and iPads at home. Remove all jewelry and body piercings to prevent potential burns and for safety reasons.
9. **Yield:** if you are diabetic, do not take any oral medications for diabetes the morning of surgery. If you take Insulin, contact your health care provider for specific instructions. Do not take any anti-inflammatory medications such as Advil, Aleve, Motrin, Ibuprofen, and Naprosyn for 7 days prior to the procedure/surgery. Tylenol (Acetaminophen) is OK.
10. **Shower:** shower or bathe the night before and/or morning of procedure. Use a prep solution if provided to you by your urologist or pre-surgical testing department. Remember to cut your nails.

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

11. **Anticoagulants:** If you take Aspirin, Plavix, Coumadin, Pradaxa or Effient, please contact your health care provider/surgeon for specific instructions.
12. **Fluids:** No fluids after midnight. Your physician may give you instructions to take morning medication with sips of water prior to arriving to the hospital/ambulatory center.
13. **Eating:** No solid food or non-clear fluids such as milk or orange juice after midnight in order to prevent respiratory complications.

Postoperative Instructions

After your surgery is completed, you will go to the recovery room to wake up from your surgery. Most stone procedures are ambulatory procedures and you will likely go home the same day. Patients undergoing a PCNL procedure will most likely remain in the hospital overnight or stay for several days. For patients undergoing ESWL, ureteroscopy or other endoscopic procedures, you will go home the same day. The exception to this will be if you are having significant pain (intractable pain), nausea, vomiting, fever or chills or you do not fully awake from the anesthetic. Then, in this situation, you will remain in the hospital.

Postoperatively the name of the game does not need to be pain! Stay well hydrated.

Once you go home, there are several things that you should consider. Patients should consider the following (**HAVE MORE FLUIDS**):

Hurt: If your doctor did not give you a prescription for pain relievers, take two Tylenol every 4 hours, as needed. If you received a prescription for pain medication, take the medication as directed. If the pain does not get better, **call your doctor**.

Alcohol: Do not drink alcohol after surgery for 24 hour.

Vomiting: If you received a prescription for treating nausea or vomiting, take the medication as directed.

Exercise: It will take several hours for the medications to wear off, so patients are instructed

to rest when they get home. In addition, wait until you feel comfortable to increase your activity or exercise. In the mean time, it is important for you to be out of bed and ambulating. This will minimize your risk of deep vein thrombosis (DVT).

Medication: If you received a prescription for antibiotics, take all of the medication as directed. Do not discontinue this medication until all the pills are gone. Return to your usual medications, unless otherwise directed by your doctor. Do not take aspirin or aspirin-like medication until your urine begins to clear.

Ouch! You may have some bruising, redness or slight swelling at your treatment site. Apply an ice pack to the affected area for any swelling you may have today. After 24 hour, use warm heat, along with your pain medication for relief.

Relief: Placing a pillow behind the lower area of your back while sitting or driving may help relieve any ache or pain you may have.

Emergency: In case of an emergency, you should be instructed to contact your urologist. If you are not able to reach your physician, come to the emergency room.

Food: After your procedure you may be hungry. Start off light. Sip fluids and progress to a regular diet. You want to prevent nausea and vomiting. As your appetite returns, start with fluids and then respect your appetite. Return to your usual diet, unless otherwise directed by your doctor. Do not plan a diet immediately after surgery. You need the calories during the healing and recovery period.

Lifting: Avoid heavy lifting (over 25–30 lb) for long periods of time during the first week after Lithotripsy.

Urine: Your urine will be bloody and should begin to clear a few days after your treatment. Increase fluids until your urine becomes clear.

Impaired judgment: Do not drive, operate any dangerous machinery, do any banking or sign important documents due to the sedation given, for 24 hour.

Doctor: Call your doctor if you:

1. Develop a fever (above 101 °F) or chills.
2. Have thick blood clotted urine.
3. Severe pain not relieved by pain medication.

4. Persistent vomiting or nausea.
5. Excessive swelling, redness or numbness on or around the wound site.

Strain: Be sure to strain your urine as directed for 1–2 weeks following Lithotripsy. Save all gravel/sand in the container. Please bring these fragments with you to your next appointment so that they will be sent for stone analysis.

Conclusion

In summary, it is important for you to be equally prepared before and after your surgical procedure. It is normal for you to feel anxious and you may have difficulty understanding or even remembering pre- and post operative instructions. Your Urologist will most likely have prepared written instruction for you. Be certain to have written questions in advance and have the questions answered to your satisfaction. Have your spouse, family member or friend join you. Having a greater knowledge and understanding of your procedure and most of all, meeting your expectations will help to minimize your fears and anxiety. The goal is to make your surgical experience a rewarding and uneventful one.

Larry was a gambler, but he knew when not roll the dice with his stone. He had a history of stones before with frequent trips to the emergency room. He knew that when he was diagnosed with his 8 mm right renal

stone, that he would take care of it as soon as possible. He did not want to chance the possibility of additional visits to the ER with more lost time from work and time away from the family. I scheduled Larry for an elective ESWL procedure for his renal stone. We gave him 2,500 pulses and we monitored the stone radiographically during the procedure. The stone broke into many fragments. Following the procedure he recovered well. He voided twice with a rosé colored urine, quite typical after this procedure. He had a couple of glasses of juice and a slice of toast. He was ready to leave. Prior to discharge, the nurse went over the discharge instructions with the patient. Unlike most of my patients who have a spouse, significant other or a friend to drive them home, this patient did not. There was a limousine outside the ambulatory center to drive him home. While walking him to the car, I admired the fact that the patient had a stretch limousine to drive him back home. I saw the patient 1-week later in the office for his postoperative visit. His follow up x-ray confirmed a good outcome without any residual stone fragments. I commented to him about how he used good judgment in carefully planning his trip home with a limo. “Dr. Schulsinger”, he said, “I did not go home. I went to Atlantic City to a party at the casino”. And “yes,” he said, “I took your advice. I did not do anything that required thinking. So instead of playing black jack, I just put money down on the roulette table!”

Merrit Debartolo and David A. Schulsinger

Medical Management Facts

- Medical management of stone disease has been shown to reduce stone recurrence.
- Hydrochlorothiazide (HCTZ) decreases urinary calcium.
- Potassium Citrate replenishes urinary citrate, increases urine pH and complexes urinary calcium.
- Thiopronin makes stone-forming cystine into soluble cysteine.

High-resolution fiber-optic scopes, powerful Holmium yttrium aluminum garnet (Ho:YAG) lasers, tandem and broad-focused shock wave lithotripters, intricately versatile surgical and endoscopic instruments, and other advances offer minimally invasive and efficacious treatment of urinary tract stones. Even these interventions have a modicum of invasiveness that subjects patients to possible complications. An ideal method for achieving and maintaining stone-free status would rely more on hydration, medicine, and nutrition than on state-of-the-art surgical instrumentation. In this chapter, tailored medical strategies for treatment and prevention of stone disease are discussed.

To develop a kidney stone management strategy that is best tailored to a patient's individual meta-

bolic nuances, tests of the blood, urine, and any previous kidney stones are extremely helpful. Depending on the results of these tests, the urine pH and the concentration of certain electrolytes – most notably calcium, oxalate, citrate, and urate – can be determined. Based on these concentrations, the metabolic cause for stone precipitation can be identified and targeted with specific medical and nutritional therapy. The most common identifiable abnormality in urinary tract calcium stone formers is excess calcium in the urine (**hypercalciuria**, 62 %), followed by deficient urinary citrate (**hypocitraturia**, 41 %), excess urinary urate (**hyperuricosuria**, 35 %), excess urinary oxalate (**hyperoxaluria**, 8 %) and decreased urinary magnesium (**hypomagnesiuria**, <5 %). For non-calcium stone formers, the common causes for these stones are infection (struvite, 15 %), low urine pH (uric acid stones, 5 %) and finally increased urine levels of the amino acid **cystine** (**cystinuria**, 1 %) (These factors are expressed as percentage of the total, and since some patients have multiple factors, the percentage total exceeds 100 %).

Medical management for stone disease is a critical approach in the treatment process. New guidelines for the Medical management of renal stones have just been established and presented at the American Urological Association Meeting AUA (2014); these strategies were developed jointly between the American Society of Nephrology and the Endourological Society. There is good evidence that dietary and medical therapy of stone disease can significantly reduce

M. Debartolo, MD • D.A. Schulsinger, MD (✉)
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: merritmd@gmail.com;
endourology@yahoo.com

the risk of stone recurrence. This chapter will address the medical management of stone disease to make the urine's composition less conducive to stone formation.

Hypercalciuria

As discussed in Chap. 8, there are three types of hypercalciuria: absorptive, renal and resorptive hypercalciuria. Chapter 27 discussed the dietary management of hypercalciuria, including maintaining adequate hydration, maintaining normal calcium intake in most patients (restrict calcium with absorptive hypercalciuria), limit dietary sodium and animal protein intake. Medical therapy for hypercalciuria include:

- **Hydrochlorothiazide**
Hydrochlorothiazide (HCTZ) (25 mg twice/day) decreases calcium excretion into urine by increasing the kidney's ability to reabsorb calcium. By helping the kidneys reabsorb calcium, HCTZ keeps calcium out of the urine and prevents it from forming urinary tract stones. As such, HCTZ is most useful in cases of hypercalciuria. When taken over a period of many years, HCTZ may become less effective. In addition, excess sodium can blunt the effects of HCTZ. Side effects of HCTZ include dizziness, upset stomach, lightheadedness, headache, and muscle cramps.
- **Chlorthaladone**
Chlorthaladone (25 mg/day) is similar the HCTZ and has a longer half-life.

Hypocitraturia

Citrate is the most commonly recognized inhibitor of calcium oxalate stone formation and the lack of citrate in the urine can produce hypocitraturia. As a solitary abnormality, hypocitraturia is responsible for 5–10 % of stones, however, as a mixed abnormality, it is accountable for up to 50 % of stones. Some of the causes for hypocitraturia include distal renal tubular acidosis (RTA Type I), chronic diarrhea, physical exercise (i.e., lactic acidosis), acid-ash rich diet and depletion

of potassium (excess use of HCTZ). The treatment for hypocitraturia is alkalinization therapy.

- **Potassium Citrate (Urocit-K)**
Potassium Citrate (Urocit-K) increases urinary pH, restores urinary citrate concentration, and forms soluble complexes with urinary calcium so that the latter does not form urinary tract stones. Due to these effects of potassium citrate, it is arguably one of the most useful and versatile medications available for stone prevention. Its ability to raise urinary pH makes it useful for **acidosis**, including **renal tubular acidosis (RTA)**, as well as **hyperuricosuria** and even **cystinuria**. Also, the citrate component is necessary and sufficient to treat **hypocitraturia**, and important and often-overlooked cause of recurrent stone episodes. Side effects include nausea, vomiting, diarrhea, and abdominal discomfort, but taking potassium citrate with meals minimizes these effects.

Hyperuricosuria

Uric acid is normally found in the urine. Excess uric acid in the urine, called hyperuricosuria, is due to endogenous causes (produced by the body) or exogenous factors (high intake of animal protein). The hallmark for uric acid stone formation is a low urine pH. Treatment of these stones is dietary modification and medical therapy. The cornerstone of medical therapy is alkalinization of the urine to increase the urine pH and to increase the solubility of uric acid.

- **Potassium Citrate (Urocit-K)**
As described above.
- **Allopurinol**
Allopurinol inhibits the enzyme xanthine oxidase to decrease body's production of uric acid, thus making it a useful medication to treat **hyperuricosuria** and **gout** as causes for recurrent stone episodes. Side effects include upset stomach, diarrhea, and drowsiness. In addition, use of allopurinol can result in the accumulation and urinary excretion of xanthine, resulting in xanthine stones. Allopurinol should be used with caution in patients with renal insufficiency.

Hyperoxaluria

Hyperoxaluria is a metabolic factor responsible for 8 % of stones. The pathogenesis of this disorder can be due congenital factors (primary hyperoxaluria), dietary factors (excessive dietary consumption of oxalate rich foods) or enteric hyperoxaluria (inflammatory bowel disease, gastrointestinal surgery). Oxalate can become too abundant in the urinary tract for the reasons described in Chap. 8, and the pharmacologic strategy depends on the reason for the oxalate excess. If a gastrointestinal disease is the culprit, the treatment is **calcium supplementation** – as counterintuitive as it may seem, calcium supplementation prevents dietary oxalate absorption by complexing with oxalate, making “free” oxalate available for intestinal absorption, and thus reducing the urinary oxalate concentration. However, if the oxalate excess reflects an inability of the liver to process amino acids (i.e., primary hyperoxaluria), the only solution is a liver/kidney combined transplant. Lastly, if oxalate excess results from antibiotics decreasing the intestines’ good bacteria, the treatment is to modify the antibiotics and/or add probiotic therapy. In any case, however, dietary modification to decrease oxalate intake may help (See Chap. 27).

- **Calcium + Vitamin D₃ (OsCal)**
Calcium + Vitamin D₃ complexes with oxalate to prevent the latter from forming stones, thus making it useful for the treatment calcium oxalate stones that form in **hyperoxaluria**. Side effects include upset stomach and constipation.

Hypomagnesiuria

Low urinary magnesium, or hypomagnesiuria, is identified in 1 % of stone formers. Magnesium is a known inhibitor of stone formation as it competes with calcium for binding oxalate and other ions to form soluble complexes to prevent stone formation. Treatment of hypomagnesiuria is consumption of magnesium-rich foods (bananas, apricots, soy products) or increased magnesium in the form of medicine (**magnesium oxide, magnesium citrate**).

- **Magnesium Oxide**

Magnesium Oxide replenishes bodily magnesium, making it the treatment of choice for **hypomagnesiuria**. Side effects include upset stomach and diarrhea.

Cystinuria

Cystinuria is an autosomal-recessive disorder leading to defective renal and intestinal transport of cystine and the other dibasic amino acids ornithine, lysine and arginine (COLA). The main clinical manifestation of cystinuria is cystine stones, which is a lifetime issue. Cystine represents 1 % of all stone formers. The mainstay of treatment is surgical intervention, however, stone prevention will require monitored dietary and medical management.

- **Potassium Citrate (Urocit K)**

Potassium citrate is the first-line alkalinizing drug. The goal of therapy is to maintain a urine pH level within the range of 7–7.5 for dissolution to occur. Paradoxically, with a urine pH >7.5, there is an increase risk of calcium phosphate stone formation. Urine pH must be closely monitored to maintain this small window for treatment of cystine stones and prevention of calcium phosphate stone formation.

- **D-Penicillamine**

D-Penicillamine is an option to treat **cystinuria**. D-Penicillamine converts stone-forming cystine into soluble cysteine, thus making it 50 times more soluble than cystine. Unfortunately, as in Wheel of Fortune, the extra “E” comes with a price. The potential side effects are severe, and the most egregious of these include suppression of the bone marrow’s blood cell-producing activity, diffuse skin inflammation, and protein in the urine (proteinuria). Mild side effects include rash, fever, joint pain, diarrhea, nausea or vomiting. Side effects of this medication is the main factor that influences compliance.

- **Thiopronin (Thiola)**

Alpha-Mercaptopropionylglycine (Thiopronin) is similar to D-penicillamine in that it turns

stone-forming cystine into soluble, non-stone-forming cysteine, but with fewer side effects. This makes Thiopronin a “kinder, gentler option” for treating **cystinuria**. Nevertheless, only 15 % of patients started on these chelating agents (*i.e.* D-penicillamine, Thiola) actually remain on these faithfully enough to maintain a stone-free therapeutic state because of the side effects. One reason for this poor compliance rate is that patients can take up to 16 pills/day (Tiopronin #10 and Potassium citrate #6).

- ***Captopril***

Captopril is a medication normally to treat patients with hypertension. Captopril is also shown to treat patients with cystinuria by forming a thiol-disulfide bond, which is 200 times more soluble than cystine. This treatment option is indicated in patients who cannot tolerate the other agents previously described. This drug is contraindicated in patients with high levels of potassium in the blood (hyperkalemia).

- ***L-cystine dimethylester (L-CDME)***

L-cystine dimethylester works by binding to L-cystine and inhibits the normal crystallization process for stone formation [1]. This medication is not approved.

Infection Stones

Infection stones are also referred to as magnesium ammonium phosphate stones or struvite stones. They represent 10–15 % of all stones. The cornerstone of treatment is surgical. Eradication of stones is essential for eradication of bacteria producing these stones. However, medical therapy to dissolve the stone is indicated in a smaller number of these patients. While dissolution is not common, it is used in patients who are not surgical candidates.

- ***Acetohydroxamic acid:***

Acetohydroxamic Acid (AHA) is a potent irreversible inhibitor of urease, the enzyme found in bacteria that produce these infection stones. AHA works by reducing the urine pH and ammonia. AHA is oftentimes taken in conjunction with suppressive antibiotics. AHA had limited compliance due to side effects: GI symptoms include nausea, vomiting and loss of appetite; neurological symptoms include headache; hematologic symptoms include anemia and thrombocytopenia; deep vein thrombosis and pulmonary embolus and alopecia.

Conclusion

Targeted medical and nutritional approaches tailored to patients’ specific metabolic nuances as evidenced on 24 hour urine tests have been discussed. Some dietary and nutritional aspects to stone prevention and treatment are generalizable to any patient, regardless of urine composition. These include **copious water intake, controlling dietary sodium, avoiding protein excess, and eating enough calcium**. Though the importance of these interventions has been and will continue to be stressed in other chapters, it is worth repeating that **the above interventions have been demonstrated to reduce stone episodes in up to 50 % of patients, regardless of stone composition or 24 hour urine analysis**.

Applying generalized nutritional stone management principles with specific medical adjustments based on the individual urine composition represents a comprehensive, targeted, and truly minimally-invasive strategy for helping urinary tract stone formers reach their stone-free “happy place.” *Remember, be mindful of your medication!*

A decorated combat veteran having served several tours of duty overseas, TJP had experienced his share of harrowing pains. He had also experienced a number of kidney stone attacks, which he remembers to be “just as bad, if not worse” than his combat-related injuries. For these, he had undergone ureteral stent placements as well as lithotripsy by ureteroscopic, shock wave, and percutaneous approaches. These yielded calcium oxalate mono- and dihydrate stones. Having undergone a “pound of cure” and now wishing for the “ounce of prevention,” he underwent a urinalysis and a 24 hour urine analysis that ultimately revealed him to be dehydrated, with urine that was acidic due to hypocitraturia. TJP’s urologist therefore prescribed potassium citrate and offered TJP, a self-proclaimed “meat-and-potatoes guy,” suggestions on

how to increase his water intake, mind his sodium intake, and enjoy meat in moderation. These were not easy changes to make, but remembering the pain of his stone attacks and procedures provided TJP with all the motivation he needed. Now, after a year of medical and dietary therapy, he “feels great” and imaging confirms he’s stone-free. TJP is so grateful that at his follow-up appointment, he invites his urologist to a cookout at his house and offers to grill him the “best Portobello burger in town.”

Reference

1. Rimer JD, An Z, Zhu Z, Lee MH, Goldfarb DS, Wesson JA, Ward MD. Crystal growth inhibitors for the prevention of L-cystine kidney stones through molecular design. *Science*. 2010;15(6002):337–41.

Jonathan J. Melquist and David A. Schulsinger

Summary Surgical Facts

- The majority of stone procedures can be treated by noninvasive or minimally invasive procedures.
- Depending on the stone size and stone location will determine which procedure is ideal for you.
- As there may be several procedure options, discuss with your physician about the best procedure for you and know the risks, benefits and alternatives for treating your stone.

Introduction

The management of urinary stones has revolutionized in the past 30 years. It is principally attributed to advances in technology and medical therapy. Prior to the invention of very small telescopic instruments called ureteroscopes, many patients had their stones removed by open surgery. Imagine having a 10-in. incision to remove a ¼-in. stone! In this chapter, we will discuss the advances in stone management while paying spe-

cial attention to the benefits and disadvantages of common technologies.

For ease of read, this chapter is broken into overview sections. We begin by discussing: Stone Prevention, Using Medicine to Pass a Stone, Prior to Surgery to Treat Urinary Stones and Preparation for your Procedure. It is followed by a breakdown of how various technologies can be used depending on the location of the urinary stone: kidney, ureter and bladder. Techniques are described, and significant advantages and disadvantages are discussed in a consistent format.

This chapter will provide an overview, the advantages and disadvantages of each procedure depending on where your stone is located. Ultimately close consultation with your Urologist will be necessary to make the most informed decision possible. The goal is to provide the stepping-stones to choosing a successful procedure!

Stone Prevention

Although not a technological advancement, our evolving understanding of how urinary stones form has lead to one of the best ways to treat stones – *never to get them in the first place!*

We find that long-term adequate hydration remains one of the most important means to preventing stones. Stones are formed from various compounds in the urine, and keeping their concentration low through good hydration reduces

J.J. Melquist, MD
MD Anderson Cancer Center, Houston, TX, USA
e-mail: melquist@gmail.com

D.A. Schulsinger, MD (✉)
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

the risk of stone formation. See Chap. 25 on additional information on hydration.

You may remember from chemistry class in high school that “*dilution is the solution.*” A better understanding of which compounds leading to stone formation that did not exist in the past will allow your doctor to make better dietary recommendations. With research advancements, your doctor will be able to provide better dietary recommendations. These recommendations can vary and will be discussed further in Chap. 27.

Using Medicine to Pass a Stone

A revolution in medical therapy for urinary stones has occurred. A newer concept involves the use of medicine to dramatically improve your chances of passing a stone.

This treatment is called “Medical Expulsive Therapy” (MET). MET is the use of a medication (e.g. Flomax®), which is typically given to older men with enlarged prostates and difficulty urinating. Urologists found that this category of medication, called an alpha-blocker, helps to dilate the ureter so that a stone may pass more easily. Multiple studies have shown a significantly higher rate of spontaneous stone passage when using this medication, thus foregoing the need for surgical intervention. If one can pass a stone on their own, it is surely the preferred method. It is safe to use in both men and women.

Considerations Prior to Surgery

Urinary stones, for the most part, are formed and grow to their final size in the kidney. Most stones in the kidney DO NOT CAUSE pain. It is only when a stone slips into the ureter and causes an obstruction of normal outflow urine will a patient feel pain. See Chap. 14 for more information on symptoms related to your stone.

Many patients find that they need relief for their pain before the stone can be safely removed. A urologist may place a small hollow plastic tube, called a ureteral stent, in a ureter that is obstructed by a stone. It is coiled on each end to

prevent it from migrating out of the ureter. This remains completely within the body and, thus, is not seen by the patient. Refer to Chap. 22 for additional information on stents.

In certain circumstances, the urologist will recommend a placement of a small plastic tube that enters directly from the back into the kidney, called a nephrostomy tube. It is typically half the width of a pencil and placed by interventional radiologists. Part of the tube remains inside while the remainder exits the body and is connected to a small drainage bag to collect urine.

Preparation for Your Procedure

Prior to your procedure, you will meet with health professionals (nurses, PA’s), phlebotomists and potentially anesthesiologists. This meeting is referred to as the pre-admission testing, or PAT’s. You will meet with a nurse, PA or even a physician, who will do a history and physical exam for your surgical procedure. Depending upon your health and other co-morbidities, you may be required to obtain medical clearance from one of your other physicians. For example, if you have any cardiac history, such as previous heart attack (e.g., MI, placement of cardiac stents or valves, you may be required to obtain a letter of medical clearance from your cardiologist for this procedure. Similarly, if you have any lung conditions or neurologic issues, you may need to obtain a letter from your pulmonologist or neurologist, respectively.

In addition, as your urologist plans to do this procedure, he will want to minimize your risk of bleeding. Therefore, if you are on any blood thinners, this will need to be discontinued for a period of time prior to your surgery. These include medication like aspirin or aspirin-like medication, Plavix or Coumadin (Warfarin). For example, if you are on Coumadin, your PMD or your cardiologist may agree to stop this for 5 days or more prior to procedure to allow your blood not to be as thin. On the other hand, if your physician feels that this may be an extended period of time to be off anticoagulants, you may need to be “bridged” with a short-acting medication up until the procedure, which would be discontinued at the time of the procedure.

There are also non-pharmaceutical grade products that may have an effect on your body's ability to bleed. This includes substances like Vitamin E and Omega-3, substances that can extend your bleeding time. These substances, too, will need to be discontinued prior to your procedure. Ask your physician when and how long they should be stopped.

Surgery Day

On the day of your procedure, you will be meeting with a series of health professionals to get you ready for your stone procedure. A nurse will come to your room to verbally confirm and identify you. They will ask you your name, date of birth and the procedure you will undergo. They provide you with your wristband identification, confirm any allergies, and initiate any medications prior to the procedure. This would include pre-operative antibiotics, deep vein thrombosis (DVT) prophylaxis or Beta-blockade.

Site and Side Verification

Prior to the procedure, you will meet with your physician. Your Urologist will also confirm your name, date of birth and wristband check. S/he will obtain or confirm your consent done at pre-admission testing (PAT's) and will ask you which side of your body the procedure is on. For example, if the patient has a stone in the right kidney, the patient will confirm that the stone procedure is on the correct location, not just the "right" side! The Urologist will need to also mark the patient's side with an indelible pen confirming the "site and side" of the procedure.

Anesthesia

You will also meet with an anesthesia care provider who will also confirm your name, date of birth, procedure and a wristband check. They will also confirm that your consent is complete and signed by you and your physician before you are transferred to the OR. They will confirm that the operative site

was marked and your antibiotics, beta-blockers, DVT prophylaxis were administered.

Time Out

Finally, the procedure is ready to be done! Not just yet! Yet again, before the procedure begins, there will be a "time out" before the procedure is initiated. The anesthesiologist will confirm the patient's name, date of birth, consent of procedure, allergies, antibiotics given, medications on the operative field and the patient's weight. It is imperative that your Urologist is present in the OR room for the time out to be discussed. *Without your doc, they cannot break your rock!* After there is agreement between the anesthesiologist, the OR nurse and the surgeon, your procedure is ready to commence.

In summary, you will be well prepared for your procedure on the day of your surgery. It will be potentially irritating to you that you will be asked repetitive question frequently by the same and different individuals. This is **ONLY** done with good intention to maintain your well-being and most of all, your safety! This should also alleviate any anxiety whether your Urologist is doing the procedure, is he running late, am I going to be under anesthesia for a long time while they are waiting for the urologist, or are they operating on the correct side? Remember, *if there is no doc, there is no shock!*

Surgical Procedures: *Let's Rock!*

In this section, details of the open, minimally invasive and non-invasive surgical procedures are discussed.

Stones in the Kidney

Depending on the size of a stone, a urologist might recommend treating it. There are many options at a Urologist's disposal to best treat the stone. They can range from open surgery to more minimally invasive techniques that utilize ultra-

sound or laser technology to fragment the stone. Fortunately even very large kidney stones can be removed without open surgery because of advances in technology. There are a variety of treatment options. For each section of the urinary tract, the individual treatment options are discussed, including a description of the procedure, indications, advantages, and disadvantages.

Treatment

Open Surgery (Pyelolithotomy, Anatomic Nephrolithotomy)

Indications

Large Stones of the kidney (greater than 2 cm)

Description

This was once very common but is now a rare procedure! It can be recommended for certain cases in which the stone encompasses the entire kidney (i.e. staghorn calculus) and other procedures (described below) cannot be performed or are unsuccessful. This approach requires a large incision – up to 8–10 in. in length! A urologist would remove the stone by making an incision in the renal pelvis, called a pyelolithotomy. The urologist may need to divide the kidney in half along its length to remove the stone. This procedure is called an anatomic nephrolithotomy.

Advantages

This procedure treats large stone burden. One can also treat other kidney problems at the same time. This procedure can also be approached by open, laparoscopically or robotically.

Disadvantages

This is very invasive and requires a large incision. Significant blood-loss is possible, including the potential loss of kidney function or the kidney itself. A multi-day hospital stay is almost certain.

Minimally Invasive Procedures

The next set of procedures to treat renal stones are performed by minimally or non-invasive techniques.

Treatment

Percutaneous Nephrolithotomy (PCNL)

Indications

Large stones of the kidney (greater than 1.5 cm; Fig. 20.1).

Description

Access to the kidney is gained by using x-rays to direct a tube from the skin on your back to the kidney. The size of the tube placed can be up to 1 cm in width. Stones are extracted with a rigid telescope (Nephroscope or Renoscope; Fig. 20.2). Placed through the scope is a probe that has the dual advantage of using ultrasonic and pneumatic energy to fragment the stone (See Fig. 20.3). The ultrasound emitter at the end is typically used to drill holes and break the stone in to small pieces (Fig. 20.4). A pneumatic hammer, which acts like a jackhammer, can also be employed to break the stone (Fig. 20.5). After the stone is broken, one extracts the stones fragments through a tube. After the procedure is complete, the large 1 cm access tube is exchanged for a smaller drainage tube (typical 0.4 cm in width), called a nephrostomy tube.

Advantages

Able to remove very large stones, multiple small stones, even stones that encompass the entire kidney.

Disadvantages

Usually requires hospitalization. After the procedure, the nephrostomy tube coming out the patient's back might need to remain for several days. This can be uncomfortable, especially when sleeping. With the nephrostomy tube in close proximity to the lungs, rarely patients can have air (pneumothorax) or fluid (hydrothorax) build between the lung and the chest wall. This may require further procedures to rectify.

Treatment

Ureteroscopy: *Going to great lengths to treat your stone!*

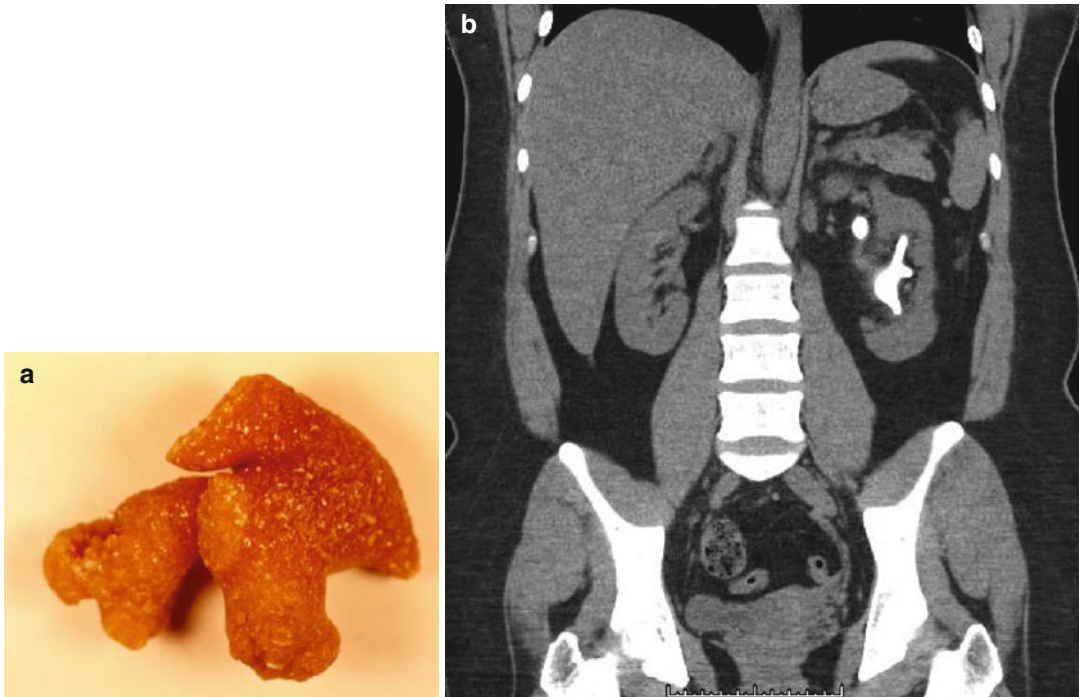


Fig. 20.1 Large cystine stone (a) and coronal CT scan image (b) demonstrating a left staghorn calculus and a smaller left upper pole stone



Fig. 20.2 Image of a nephroscope used to look inside the kidney



Fig. 20.3 Image of Lithoclast

Indications

Smaller stones of the kidney. The widths of all the stones when summed together should be less than 1.5 cm.

Description

Technological improvements have allowed for the manufacture of very small telescopic instruments, called ureteroscopes (See

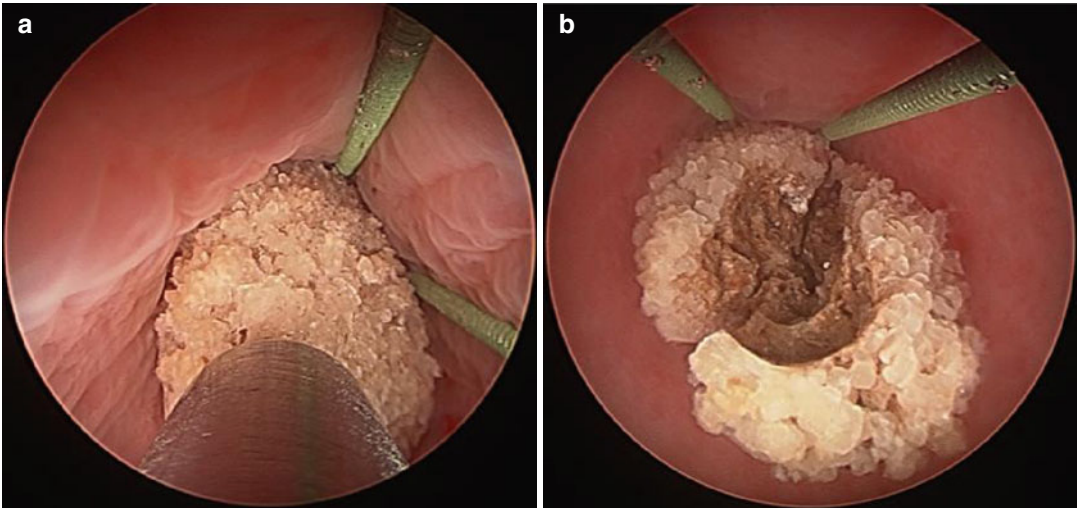


Fig. 20.4 Nephroscopic view of the renal pelvis demonstrating large renal stone. Lithoclast using the ultrasonic lithotripter (a) and the resulting large stone cavity created (b)

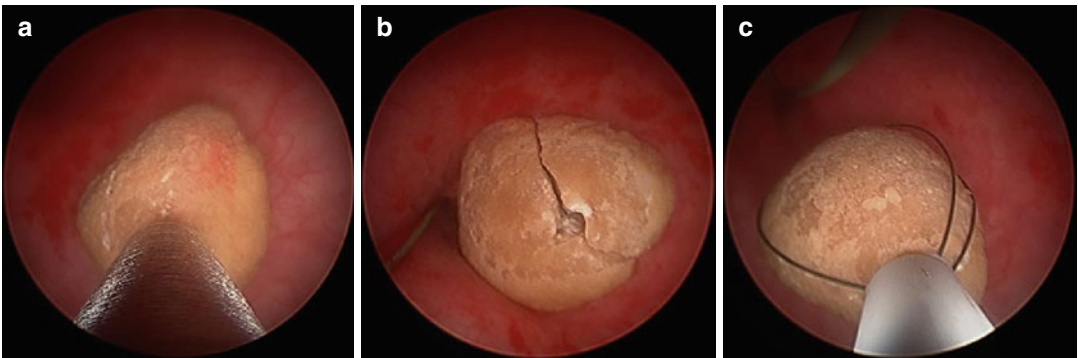


Fig. 20.5 Lithoclast using the pneumatic lithotripter used to fragment stone (a); Stone cracked (b) and fragment removed (c)



Fig. 20.6 Image of flexible ureteroscope

Fig. 20.6). A Urologist will advance the scope through the urethra and bladder, up through the ureter and extending into the kidney. Once in the kidney, the stone is typically pulverized with a powerful laser to manageable-sized pieces. These pieces must be small (<0.3 cm) so that they can be extracted with a basket deployed through the ureterscope (Fig. 20.7). Several passes are often required to remove the entire stone burden. A ureteral stent is placed after the procedure to allow the ureter to heal from the trauma of the ureterscope. The stent is usually removed 1–3 weeks later in the office.

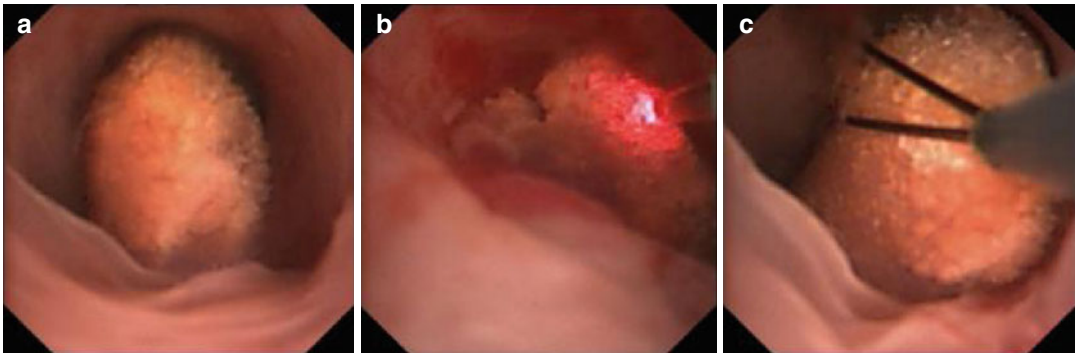


Fig. 20.7 Ureteroscopic view of stone within the renal pelvis (a); laser ablation of stone (b); stone in basket (c)

Advantages

This is a minimally invasive approach. There are no incisions as the approach is via the urethra. A good amount of stone can be treated. This is an ambulatory procedure, and most patients go home the same day of surgery.

Disadvantages

It is impractical for a large amount of stones.

Treatment

Extracorporeal Shockwave Therapy (ESWL): *The Stone Breaker!*

Indications

Stones up to 1.5 cm in the kidney

Description

ESWL is a very appealing way to treat kidney stones by utilizing shockwaves generated outside the body. Technology is such that an ultrasound wave can be targeted so that its energy is focused on a target stone (Fig. 20.8). There are no incisions. A patient will be sedated for up to 3,000 shockwaves to be delivered. This procedure is usually <45 min and can be performed under ultrasound or fluoroscopy (x-ray) guidance to localize and monitor the stone. The stone fragments into small pieces, which the patient subsequently urinates out over the following several days and weeks.

Advantages

No incisions! No telescopes (ureteroscopes) in the body. Same-day surgery.



Fig. 20.8 Dornier Compact Delta II lithotripter (Courtesy of Dornier MedTech)

Disadvantages

This procedure does not work well for treating hard stones, like cystine, calcium oxalate monohydrate and calcium phosphate stones. The stone may not fragment into small enough pieces, which may necessitate a Urologist placing a stent and extracting the stones with a ureteroscope. This procedure is contraindicated in pregnancy. One retrospective study in the literature describes the increased risk of diabetes and hypertension following ESWL [1]. This report remains controversial, as other reports do not support these findings.

Stones in the Ureter

Urinary stones lodged in the ureter can often be painful! Remember that discomfort from stones is most often caused by the blockage of urine

flow from the kidney to the bladder. Historically, these stones required open surgical extraction if they failed to pass. For a period of time, Urologist may have attempted to retrieve a stone by blindly placing a small basket in the ureter to snare it. This was associated with particular risk as it had a much higher rate of injuring the ureter compared to more modern techniques. With modern techniques described below, this major complication is exceedingly rare.

The use of ureteroscopy has really become the main stay for urologists to help a patient remove a ureteral stone that will not pass without surgical intervention.

Treatment

Open Surgical Stone Extraction

Indications

Simultaneous abdominal or pelvic surgery

Description

Once the standard of care many years ago, this invasive method of removing stones from the ureter is rarely indicated in the era of modern ureteroscopy. It treatment option is infrequently utilized today unless it is combined with another operation (e.g. open surgical removal of the uterus). Even in this scenario, the urologist may recommend to do a staged procedure in which the stone is removed ureteroscopically prior to the abdominal operation.

Advantages

When combined with another abdominal procedure, the stone extraction portion can be rather quick.

Disadvantages

This represents the most invasive means to remove a stone from the ureter (unless the patient is already undergoing a major abdominal incision for another reason). In spite of affording the urologist direct visualization of the stone, they may still have difficulty locating it.

Treatment

Ureteroscopy with Stone Extraction

Indications

Urinary stones of any size in the ureter that fail to pass spontaneously.

Description

Similar to stones in the kidney, ureteroscopes can be used to remove ureteral stones. Depending on the patient's anatomy, either flexible and/or semi-rigid ureteroscopes will be used.

The scope is advanced through the urethra and into the bladder. From there, it is guided by a direct visualization with a camera into the appropriate ureter and to the level of the stone. Uncommonly, the Urologist may need to use a small balloon and dilate the ureter to accommodate the ureteroscope. More commonly, an open sheath, called an access sheath (Fig. 20.9), can be advanced into the ureter (i.e., a tube within a tube approach), which aids to protect the ureter before the ureteroscope is inserted.

Once the stone is identified (Fig. 20.10a), it is engaged. If it is small, one can directly snare it with a basket and remove it, all the while keeping it in view the camera lens. If the stone is too large for immediate removal, it can be broken (Fig. 20.10b) with a powerful laser (LASER = Light Amplification by Stimulated Emission of Radiation) using a fiber (Fig. 20.10c) not much thicker than a single hair. The fragments are then removed in piecemeal with a basket (Fig. 20.10d) until the ureter is free from major particles.



Fig. 20.9 Access sheath helps to protect the ureter when removing stones

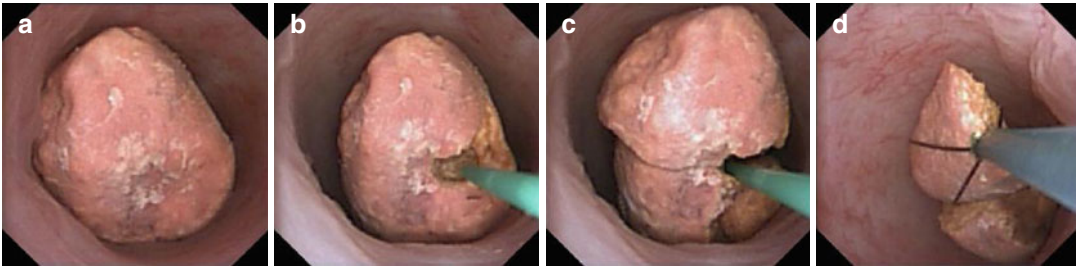


Fig. 20.10 Ureteroscopic view of a ureteral stone (a); laser lithotripsy of ureteral stone (b); stone fragmented into several pieces (c); first of several stones engaged and removed (d)

Advantages

This procedure is minimally invasive with no incisions! Patients invariably are discharged without need for a hospital stay. According to several studies, risk of creating an abnormal narrowing of the ureter (stricture) is no higher than letting the stone pass spontaneously. The ability to directly visualize the ureter allows the urologist to verify success of the procedure in removing all stone burden.

Disadvantages

Very large stones (or many stones) can lead to long procedure times. Rarely, damage to the ureter can occur. For example, part of the wall may become overly bruised or lacerated from manipulation. In these circumstances, a ureteral stent may be left in longer than normal to assure proper healing.

Treatment

Cystoscopy with Ureteral Stent Placement

Indications

To relieve urinary obstruction of the ureter on a temporary basis.

Description

The placement of a stent that assures proper urinary drainage from the kidney to the bladder when a stone is obstructing the ureter is common practice. A cystoscope is used to enter the bladder via the urethra. From there, the stent is advanced into the ureter, passing the obstructing stone to the level of the kidney using x-ray technology as a guide.

Invariably, when someone undergoes ureteroscopy with stone extraction, a ureteral stent is placed to ensure proper healing. There are, however, several circumstances in which a stent may need to be placed prior to actually removing the stone. Then, the stone is treated as a separate and staged procedure.

There are several reasons to place a ureteral stent as a separate procedure without attempting to remove the stone in a single surgery:

- **Infection:** The principle reason for placing a ureteral stent to unblock a ureter without removing the stone is infection. Excessive manipulation of the stone when the urine has bacteria can be dangerous and serious. When a pool has a clogged filtering system, after a period of time, the pool would often be filled with algae and grime for lack of flow. Similarly, if has urine has been blocked by a stone, it can become infected quite readily. Placing a stent to relieve the obstruction may only require a few minutes of working in “dirty” urine, compared to removing a stone over a longer period of time. The prolonged work in the latter situation substantially increases the chance of pushing bacteria into the blood and causing system-wide infection, also known as sepsis. This can have real consequences, and in it’s most severe form, can be life threatening! Based on various clinical factors (e.g. white blood cell count, body temperature, pulse rate, and urinalysis), your urologist will assess the risk of infection and decide if clinically, it is appropriate to remove your stone or perform a stent placement and stage removal of your stone.

- **Resource Availability:** A stent may be placed emergently if a patient shows signs of urinary tract infections, intractable pain, nausea or vomiting. This may be at a hospital or during a time when a stone extraction procedure cannot be performed. There are limited staffing and operating rooms overnight. Beyond, smaller hospitals may not have the appropriate equipment to perform stone extraction. The facility, for example, may not have the laser power generator or lithotripter to fragment a large stone.
- **Expertise of the Urologist:** Just as other professionals subspecialize, so do Urologists. While some urologists practice estate planning and others focus on criminal law, not all Urologists are experts in Endourology, the field that focuses on stone treatment.
- **Small-size Ureter Size:** If attempts to remove a stone with the ureteroscope fail because a ureter is too small, a stent can be placed. This will serve to dilate the ureter over the course of days to better accommodate a ureteroscope for later stone extraction.

Advantages

The procedure is a minimally-invasive procedure with no incisions. It is quick and often provides dramatic symptomatic relief.

Disadvantages

Placement of a stent alone is *not* a permanent solution. It buys the patient additional time until a definitive procedure is performed to treat and remove the ureteral stone. See Chap. 22 for additional information on stents and stones. Sometimes, patients suffer from “stent colic” or the pain associated with having a foreign body in the ureter. This can often be relieved with medication. See Chap. 23 for more information on pain management.

A second issue arises with non-compliant patients. If a stent is not removed, stone material can form on it over the course of months. This can present a real challenge to treat as the new stone that forms can be much larger than the original stone the stent was meant to temporize! In



Fig. 20.11 Axial CT scan image demonstrating calcified stent (yellow arrow) in the bladder

fact, in some instances, the entire stent can calcify resulting in a calcified stent (See Fig. 20.11). This may require multiple staged procedures, including percutaneous nephrolithotomy, ureteroscopy and cystolitholapaxy, to remove these stone(s).

Treatment

Extracorporeal Shockwave Therapy (ESWL):
Stone Treatment comes in waves!

Indications

Urinary stones within the ureter that fail to pass spontaneously.

Description

A very appealing way to treat ureteral stones is by utilizing shockwaves generated outside the body. This technology utilizes ultrasound waves targeted at the stone. A patient will be sedated for up to 3,000 shockwaves to be delivered, without any incisions required. Given the location of the stone within the ureter, these stones are imaged with fluoroscopy (not ultrasound). The stone fragments into small pieces, which the patient subsequently urinate out over the following several days. If the stone is large, your urologists may want to place a stent during this procedure to minimize the risk of obstructing stone fragments.

Advantages

No incisions! No telescopes (ureteroscopes) in the body. Same-day surgery.

Disadvantages

Given the location of the stone, the success rate for treating ureteral stones is less than the same size stone in the kidney. Similar to stones in the kidney, this procedure does not work well for treating hard stones, like cystine, calcium oxalate monohydrate and calcium phosphate stones. In addition, the stone may not fragment into small enough pieces, which may necessitate a Urologist placing a stent and extracting the stones with a ureteroscope.

Stones in the Bladder

Bladder stones are most commonly associated with men with obstructing prostates (See Fig. 20.12). They typically arise because of inadequate urine drainage from the bladder. In this regard, their formation differs greatly from stones that form in the kidney and may migrate to the ureter.

Treatment of a bladder stones often includes management of the underlying condition that caused it, an enlarged prostate. If the latter is not managed appropriately, a bladder stone may return in short order! An obstructed prostate represents the most common reason for bladder stone formation.

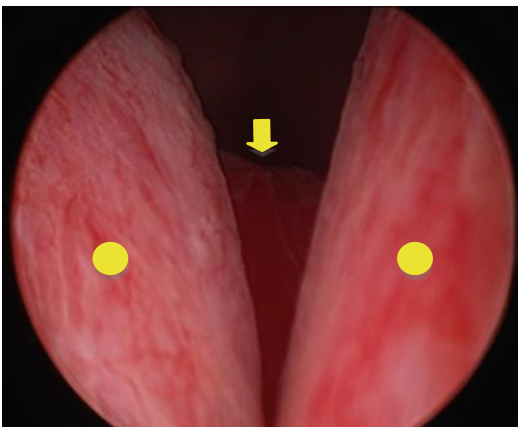


Fig. 20.12 Cystoscopic view demonstrating enlarged lateral (*yellow circles*) lobes and median lobe (*yellow arrow*) of prostate

There are other much less common causes of bladder stones including diverticula, which is beyond the scope of this chapter.

Treatment

Transurethral Stone Removal (Cystolitholapaxy)

Indications

Having a stone in the bladder that is too large to pass

Description

A small telescope (cystoscope; Fig. 20.13) is inserted through the urethra into the bladder, and the stone is fragmented with mechanical disruption, lasers, or other electrical energy sources (Fig. 20.14). The stone fragments are then evacuated.



Fig. 20.13 Cystoscope used to look inside bladder

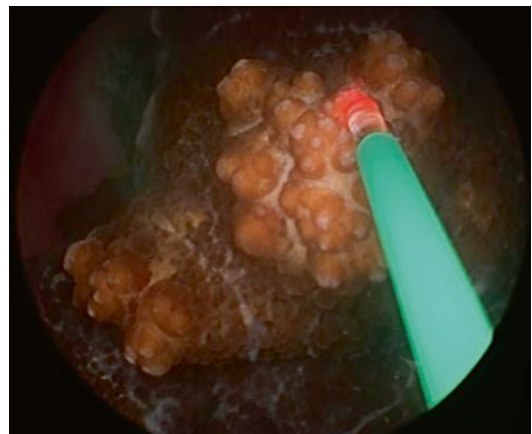


Fig. 20.14 Holmium:YAG laser ablation of large bladder stone using a 1,000 μ laser fiber

Advantages

This procedure is minimally invasive and can even be used in patients with a large stone burden. In the same operation, the surgeon can remove part of your prostate to improve urine flow in those with symptomatically enlarged prostate. *You are only required to make the decision for no incision!*

Disadvantages

At times, the stone burden can be too cumbersome to manage by this minimally invasive technique. Permanent damage to the bladder is very rare but can occur. Infrequently, there could be a small injury to the bladder or the ureteral orifices (the end of the ureters) that may require extended catheterization with a Foley catheter or stent, respectively. In rare circumstances, additional or open procedure may be required.

Treatment

Minimally Invasive Resection and Ablation of the Prostate

Indications

Symptomatic BPH is the principle cause of bladder stones in males. To remove the stone and treat the obstructing prostate. To improve the outflow of urine is essential to preventing reformation of bladder stones.

Description

The longest standing method to reduce the size of the prostate without an incision is by trans-

urethral resection of the prostate (TURP). Some individuals lovingly call it the “Roto Rooter” procedure. Using the small telescope (cystourethroscope), a metal loop is charged with electricity to literally cut away swaths of prostate tissue. When enough tissue has been resected to ensure adequate urine flow, a Foley catheter is typically placed for a short period of time.

A second method of removing excess prostate tissue is through ablation and vaporization. The tissue is “melted” away with high energy. The energy is usually delivered in the form of a laser. Holmium and KTP (i.e., Greenlight™) laser technology is the most prevalent type in the United States (Fig. 20.15). The cystourethroscope is used to guide the laser energy to vaporize the prostate tissue appropriately. When vaporization is complete, a Foley catheter is typically placed for a short period of time.

Advantages

This procedure for treating an obstructing prostate is minimally invasive! There are no incisions, and patients typically do not need to be observed in the hospital overnight.

Well-published literature has shown that patients can continue on blood thinners (e.g. Coumadin) when receiving prostate vaporization with the KTP (Greenlight™) laser. This can be a major advantage for those patients for whom it would be too risky to cease blood thinners even for a short period of time.

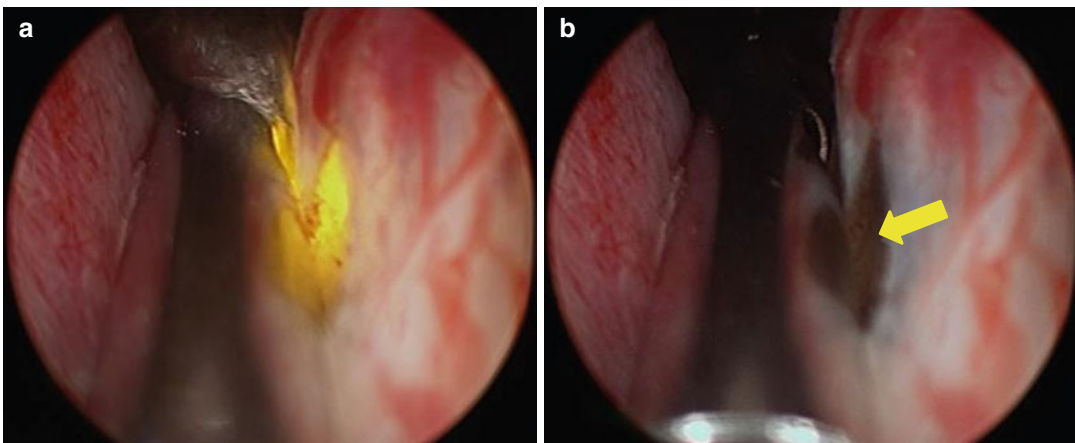


Fig. 20.15 Greenlight™ laser ablation of prostate (a); cavity (yellow arrow) created by laser (b)

Disadvantages

Large amounts of tissue can be resected; however, these minimally invasive techniques should not be used for excessively large prostates. Great debate exists about what the size cut-off should be. Some have argued that an open procedure (see next section) should be performed on anyone with a prostate gland greater than 70 g. Others say greater than 200 g. Either way, close consultation with the urologic surgeon is essential.

Rarely, the traditional TURP can cause excessive amounts of water might be absorbed by the patient throughout the case. This can dangerously low levels of sodium in the blood. The vast majority of the time, this can be managed conservatively with no long-term effects. In very rare circumstances, it can cause paralysis and even death. Vaporization techniques using the Holmium and KTP (Greenlight™) typically do not have this rare complication because free water is not used as the fluid to irrigate the device.

Treatment

Open Surgical Cystotomy with Stone Removal (and open simple prostatectomy)

Description

Sometimes the stone burden in the bladder is too large to manage by a minimally invasive technique. A male patient may also benefit from an open stone removal procedure if his prostate is very large from BPH and needs to be removed as well. In fact, concurrent simple prostatectomy for a large prostate is the main reason to remove stones by an open procedure.

As mentioned, debate exists about what size prostate an open simple prostatectomy should be performed. Some say any prostate that is greater than 70 g while others say 200 g should be the cut-off. Even others have argued for larger cut-offs with advancing minimally-invasive technologies.

A smaller incision, approximately 8 cm, is made below the belly button. Another incision is made into the bladder at which point the stone/s are identified and removed. If the size of the prostate needs to be reduced by open simple prostatectomy, the surgeon will “core” out the prostate either through the same incision in the

bladder or through a separate incision directly over the prostate. Please note, a simple prostatectomy is different from a radical prostatectomy. In the latter, a whole prostate is removed for cancer instead of part of it as is the case with simple prostatectomy.

Advantages

Quick and definitive. All the stones can be thoroughly removed. Importantly, for those with a very large prostate, open prostatectomy provides superior long-term results.

Disadvantages

It’s an open surgical procedure. It has longer recovery times due incision made under the belly button. Removing stones from the bladder incurs minimal blood loss. However, a simple prostatectomy, if performed can be associated with moderate blood loss given the prostate’s rich blood supply. In a minority of cases, patients may require a blood transfusion.

Conclusion: *Cutting Edge Technology in Stone Therapy Is Not Cutting at All!*

We live an exciting time. Technology continues to improve our lives and in ways that we do not always anticipate. Management of urinary stones is no exception. We have seen a rapid evolution in surgical technique. Whereas 30 years ago a large portion of stone removal was accomplished through open surgery with relatively large incisions, most stones are treated today by minimal invasive techniques and with no incisions at all! Advances in endoscopy (small telescope) have made this revolution in stone treatment possible. The endoscopes today can traverse a ureter that is only 7 mm thick. To put things in perspective, it is about the thickness of a pencil. It remains a marvel that we can use these endoscopes to both visualize a stone and place tools (e.g. lasers and baskets) to remove it!

The years to come will surely bring more advances that are both unforeseen and foreseen. We can expect endoscopy equipment to be

more robust and laser technology to further improve. Regardless, of the technology, you will always need a Urologist to manage the equipment and the overall disease condition, *treating one stone at a time!* Close consultation with your Urologist will remain essential for the years to come.

The nurse was sweet and sympathetic. “You are going to have to go for surgery. You need a stent because the stone is blocking your ureter.” Perhaps it was the morphine talking, but my response was firm. “No, thank you.” “Your kidney is not draining. You can go into renal failure if you do not get the stent.” It was at this point that I completely understood the expression, *looking like a deer in the headlights!* I felt helpless. I was confused at what was heading towards me. I felt imminent danger, but at the same time, staying still was the only thing I could do. While I was frozen in fear, my thoughts were racing. I had never had anesthesia before and feared the experience. Isn’t there a chance I could die? I have two babies. I was not taking any chances. But what if my kidney shuts down? I have

another one. No that’s crazy. Millions of people have anesthesia without incident. People get anesthesia for elective surgery. They elect to go under and they live to tell the tale. The nurse must have sensed my wavering conviction and took complete advantage of the opportunity. “You will be fine. I promise. Dr. S does this procedure all the time. It’s a piece of cake and the pain will be gone when you wake up.” I would like to think that my decision to head up to surgery was due to my complete faith in the safety of the procedure, but I am certain it was the promise of alleviating the pain that won me over. As promised, the procedure was a piece of cake and the pain was completely gone when I woke up. I will be eternally grateful to Dr. S and that sweet nurse for dimming the headlights and guiding me to safety.

Reference

1. Krambeck AE, Gettman MT, Rohlinger AL, Lohse CM, Patterson DE, Segura JW. Diabetes mellitus and hypertension associated with shock wave lithotripsy of renal and proximal ureteral stones at 19 years of followup. *J Urol.* 2006;175(5):1742–7.

David A. Schulsinger

Simple Stone Facts

- When selecting the optimal surgical approach for treating urinary tract stones, multiple factors require consideration.
- When considering treatment options, it is critical to consider diagnostic factors, stone-related factors and urinary tract anatomical factors.

Some patients with urinary tract stones may also have anomalies of the urinary tract. These anomalies may be anatomical factors or surgical in nature. When these anomalies coexist with renal stones, the location of the stone will determine the surgical approach that is required. Like a real estate agent finding the right home, a stone in the urinary tract associated with an anatomic variation is all about location, location, location! This chapter will focus on the diagnosis and treatment options of these complex stones.

Difficult Stones to Diagnose

Radiolucent Stones

Renal calculus detection continues to be refined by advances in imaging technology. Helical and

spiral CT scans can identify renal and ureteral calculi in an accurate and cost-effective manner. Three-dimensional reconstruction images of renal calculi are highly useful in planning stone procedures.

Radiolucent calculi such as uric acid, Crixivan, xanthine, hypoxanthine and 2,8-dihydroxyadenine calculi, are difficult to visualize on plain abdominal imaging studies (KUB). Spiral CT scan and renal ultrasounds are helpful imaging studies in visualizing stones that are not visualized on KUB (Fig. 21.1). The exception to this, is Crixivan stones, which are also not visualized on Spiral CT scan or renal US. These stones are visualized when CT scan is performed with contrast.



Fig. 21.1 Axial CT scan image demonstrating 2 cm right renal stone (*yellow arrow*) that was not previously visualized on KUB. Following surgery, the stone analysis revealed a uric acid stone

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Ureteral Stones

Ultrasound imaging is helpful in finding renal stones, bladder stones and stones at the ureterovesical junction. In the remainder of the ureter, ultrasound is a poor imaging modality for stone localization. Without contrast, Spiral CT scan is the best imaging choice to visualize stones along

the course of the ureter (Fig. 21.2). With contrast, the use of intravenous contrast (IVP) or retrograde pyelogram (RUG) can help to identify ureteral stones, especially if the stones are radiolucent. In addition, lithotriptors using fluoroscopy as a sole imaging device cannot be used to localize radiolucent ureteral stones. IVP and RUG during ESWL can also provide useful information for stone localization.

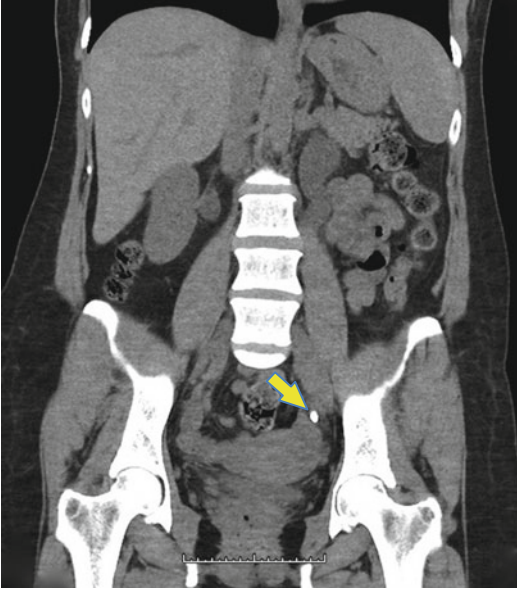


Fig. 21.2 Coronal CT scan image of a left ureteral stone (yellow arrow)

Difficult Stones to Fragment

Some renal calculi are refractory to ESWL therapy. The fragility of stones in descending order is: Cystine, brushite, calcium oxalate monohydrate (COM), hydroxyapatite, struvite, calcium oxalate dihydrate (COD), and uric acid. There is poor fragmentation of Cystine and brushite as these are the most ESWL resistant stones (Fig. 21.3). As a general rule, ESWL resistant stones (i.e., brushite, cystine, COM) should only be treated with ESWL when they are small (<1.5 cm) and larger stones preferentially treated with PCNL or ureteroscopy with Holmium:YAG (Ho:YAG) laser lithotripsy.

In rare circumstances, the very soft matrix calculi are also ESWL resistant. These radiolucent stones, often associated with urea splitting bacteriuria, are composed of as much as 65 %

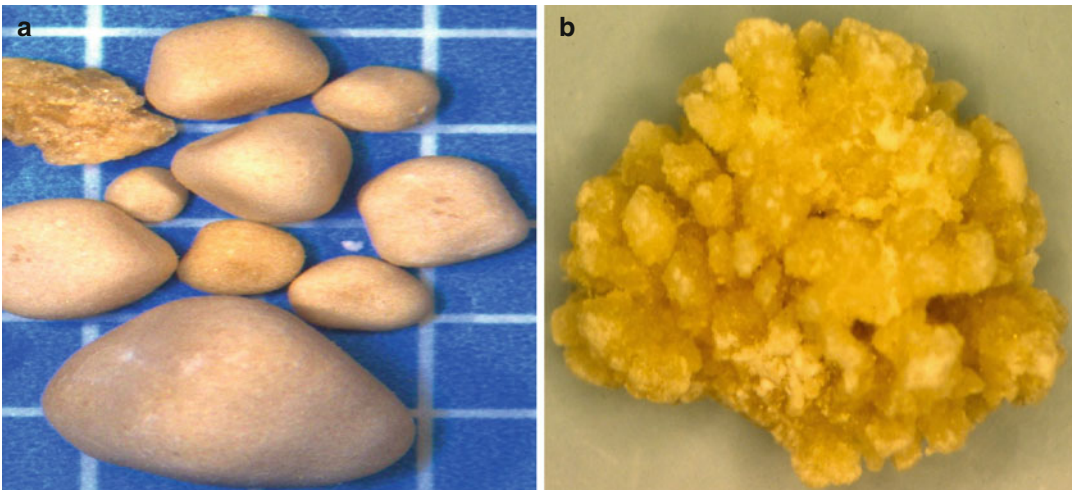


Fig. 21.3 Cystine (a) and brushite (b) stones (Courtesy of Louis C. Herring & Co., Orlando, FL)

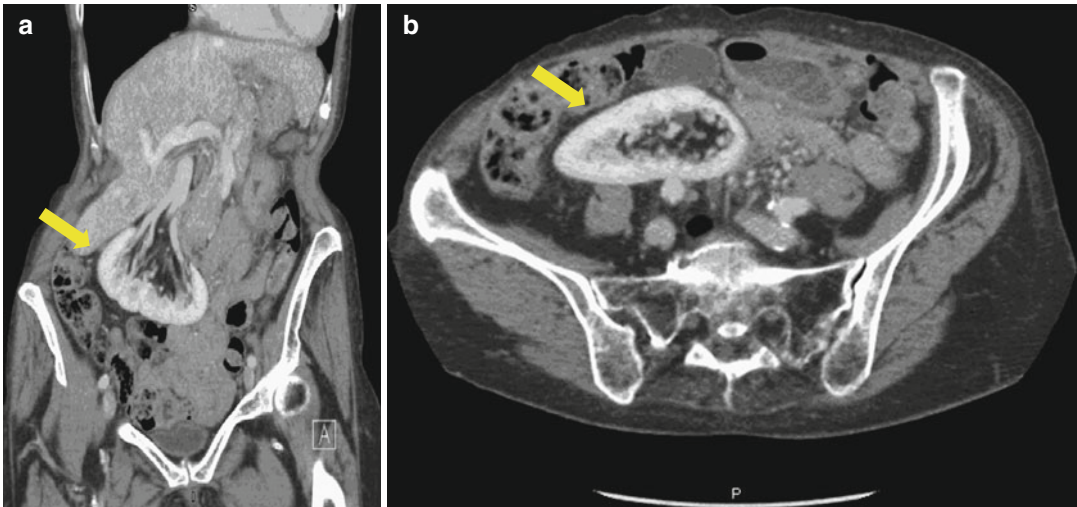


Fig. 21.4 Coronal (a) and (b) axial (b) CT scan images demonstrating a malrotated pelvic kidney (yellow arrow)

organic matter (compared to 2–3 % organic matrix in most non-infected urinary calculi). ESWL is not effective in treating matrix stones which are ideally treated with PCNL.

Difficult Stones to Access

Ectopic Kidney

An ectopic kidney is a congenital anomaly where the kidney is located above (thoracic), below (pelvis) (Fig. 21.4) or even the opposite side (crossed) of its normal position (See Fig. 21.5). The occurrence of ectopic kidney is approximately 1:2,200–3,000 people. Although these kidneys are not in the usual position, an ectopic kidney does not usually present with any symptoms. An ectopic kidney is usually discovered when imaging studies are done for other reasons. The diagnosis of an ectopic kidney is identified on renal ultrasound, CT scan, MRI, renal scan, VGUG or IVP. The location of the kidney and positioning problems of the patient on the operating room table may make ESWL a difficult choice for treatment. Alternative modalities, including ureteroscopy, PCNL and even open surgery may be required.

Ureteropelvic Junction Obstruction

The section of the urinary tract where the pelvis of the kidney meets the ureter is called the ureteropelvic junction. A blockage at this location is called a ureteropelvic junction obstruction (UPJO), where there is an interruption in the urine flow from the renal pelvis to the ureter (Fig. 21.6). The blockage seen in UPJO is thought to be caused by a congenital narrowing of the ureteropelvic junction or less commonly by compression of the ureteropelvic junction by a crossing blood vessel to the kidney. The blockage can be partial or complete with varying degrees of severity. The incidence of UPJO occurs in approximately one in 1,500 children. The male-to-female predominance is greater than 2:1, with the left kidney effected about twice as often as the right. Bilateral UPJO are seen in 10–40 % of cases. Ureteropelvic junction obstruction (UPJO) in adults is commonly associated with urinary stones. The treatment strategies for UPJ obstruction have seen a significant shift in the last several years. The majority of surgical treatments for UPJ obstruction include laparoscopic pyeloplasty, open pyeloplasty, endopyelotomy, endopyeloplasty, and robotic-assisted laparoscopic pyeloplasty. Percutaneous approach is a good alternative to render a patient stone-free

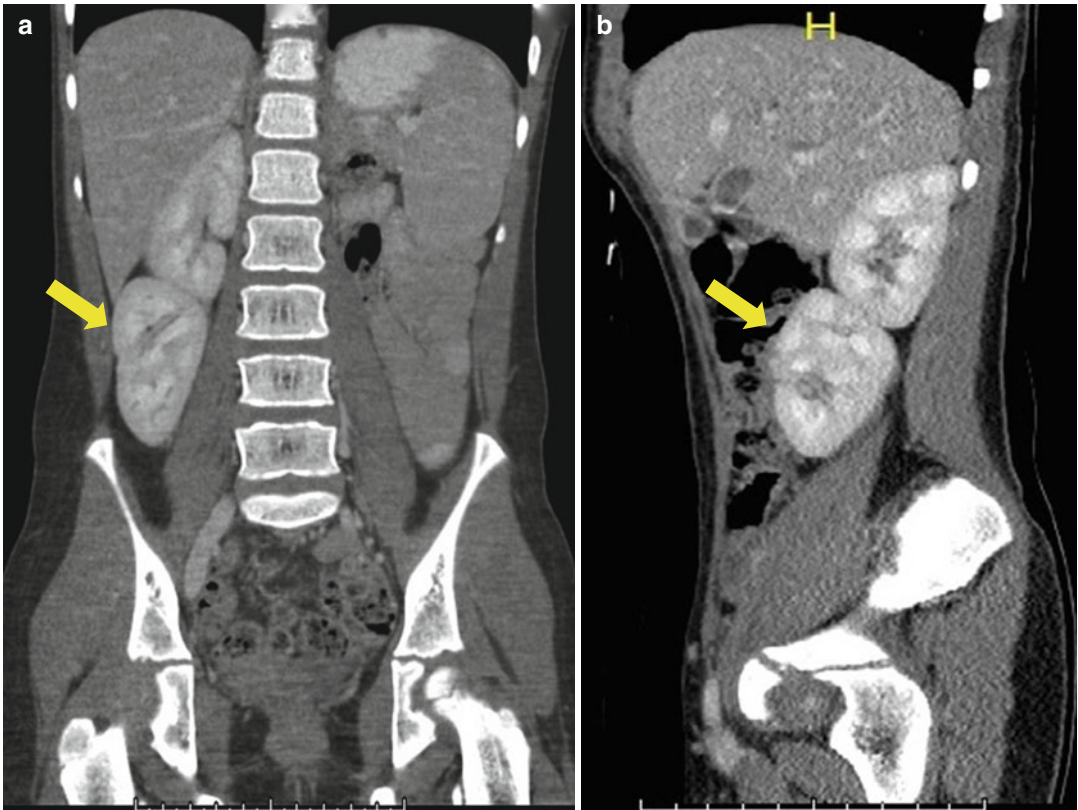


Fig. 21.5 CT scan coronal (a) and sagittal (b) views of a crossed fused renal ectopia. The crossed kidney (yellow arrow) lies underneath the native right kidney

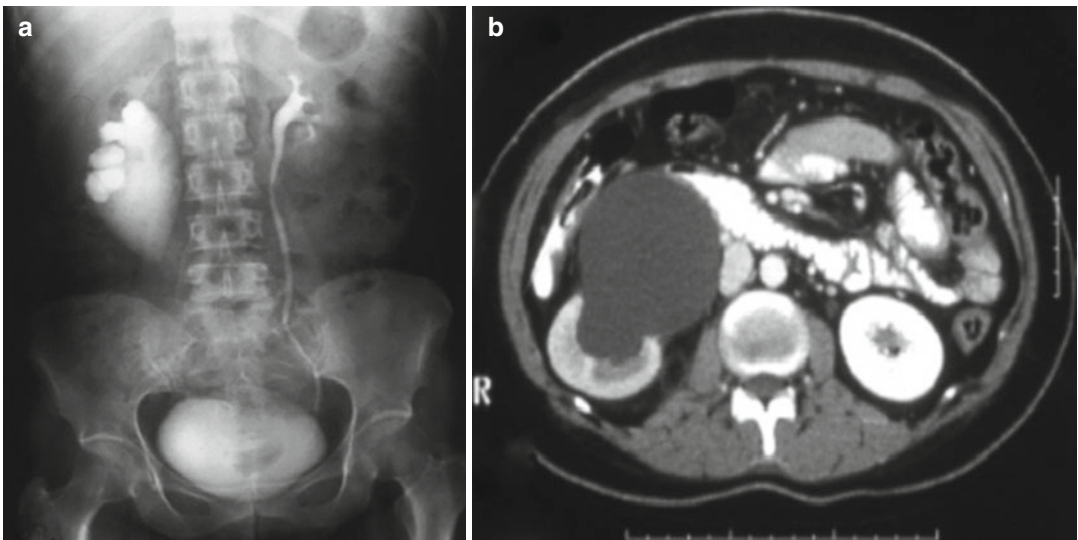


Fig. 21.6 IVP (a) and axial CT scan (b) images demonstrating a right UPJO and normal left kidney

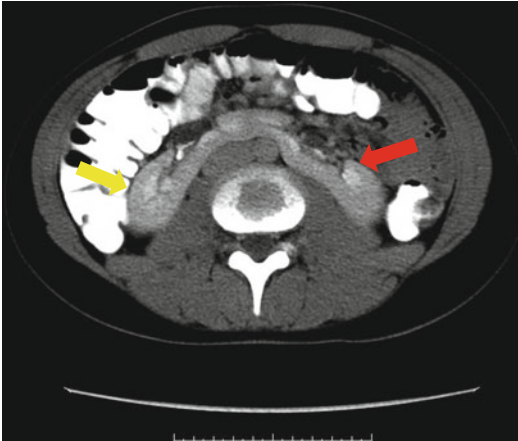


Fig. 21.7 Axial CT scan view of horseshoe kidney demonstrating the right (yellow arrow) and left (red arrow) kidneys connected in the middle

and treat ureteropelvic junction obstruction. The nephrostomy tube access for these stones allows simultaneous removal of stones and repair of the UPJ obstruction.

Horseshoe Kidney

A horseshoe kidney is the most common congenital fusion anomaly in the urinary tract (See Fig. 21.7). Typically, the ureter is attached to the kidney at a higher point, called a high insertion, and is anteriorly placed (in front of the kidney) instead of the normal medial location (toward the midline). Approximately up to two-thirds of patients with horseshoe kidney may experience hydronephrosis, infection or kidney stones. The results with ESWL are poor. Alternatively, small stones (<1.5 cm) can be approached with ureteroscopy and laser lithotripsy. Larger stones (>1.5 cm) can be treated by PCNL.

Renal Calyceal Diverticula

Calyceal diverticula are thin-walled cystic cavities located in the peripheral to an otherwise normal calyx, which communicate through a

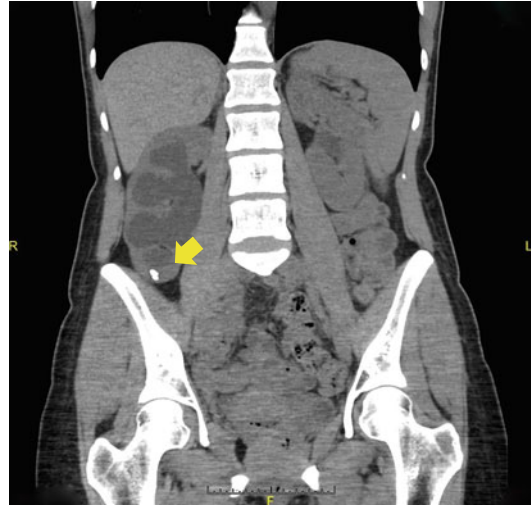


Fig. 21.8 Coronal CT scan image demonstrating a right renal stone (yellow arrow) in a lower pole diverticulum

narrow channel. They are lined by transitional epithelium, which is the same tissue that lines the urinary tract. These cysts may originate from embryogenesis, blunt renal trauma or obstruction. They occur in any part of the kidney, but usually originate from an upper pole calyx. Calyceal diverticula are found in both males and females. Renal diverticula may present with a renal stone (Fig. 21.8). The incidence of stone formation in renal calyceal diverticula present in 9.5–39 % and approximately one-third of cases become symptomatic. These stones may include calcium oxalate, calcium phosphate or carbonate apatite. Treatment of these diverticular stone with ESWL is controversial and it can be done when the stones are small and the diverticular neck is patent. Otherwise, ureteroscopy and laser lithotripsy and PCNL are better treatments of choice.

Difficult Stones to Clear

Lower Pole Stones

The kidney is divided into upper, middle and lower pole regions. Stones in the lower pole region are more difficult to pass (Fig. 21.9). The

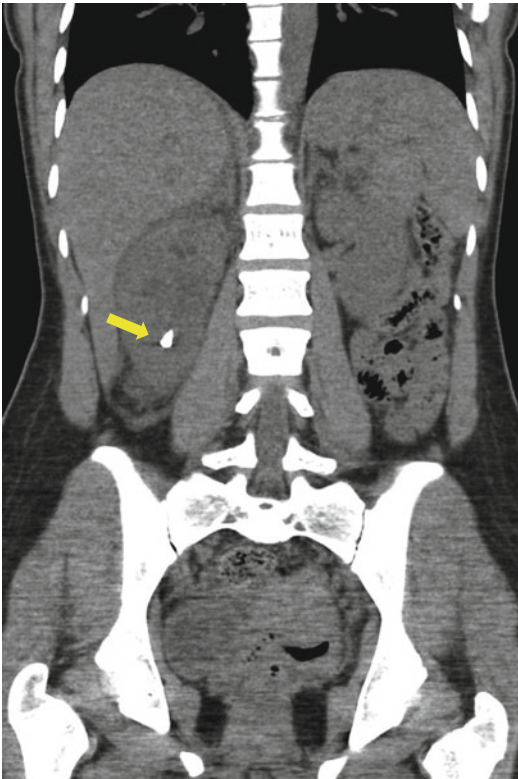


Fig. 21.9 Coronal CT scan image demonstrating a right lower pole renal stone (yellow arrow)

ability for stones to pass, or stone-free rates, is determined by stone location. The clearance of stone fragments for the lower pole after ESWL are more difficult to pass and is influenced by the anatomy of lower pole collecting system. Upper and middle calyceal calculi, for example, have a 70–90 % stone-free rate, whereas, lower pole stones range between 20 and 70 % following ESWL. ESWL is the preferred initial treatment for most individual with a lower pole stone <1 cm, whereas PCNL is the treatment of choice for stones >2 cm. For stones between 1 and 2 cm depends on other factors including stone composition and renal anatomy of the lower pole.

Steinstrasse

A complication of ESWL for urinary tract stones is stone fragments becoming blocked in the ureter. The collection of these stones form a “street of

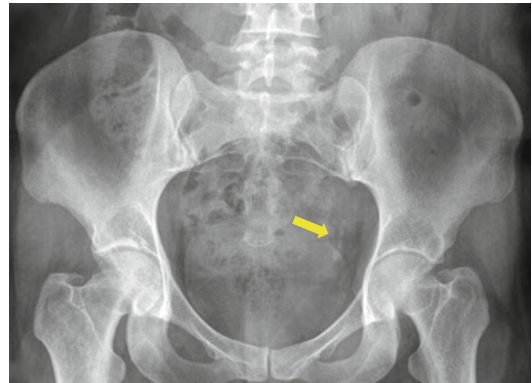


Fig. 21.10 KUB demonstrating steinstrasse (yellow arrow) in the region of the lower left ureter

stone”, referred to as steinstrasse. There may be either a single large fragment or accumulation of multiple small fragments that obstruct the ureter (see Fig. 21.10). The incidence of Steinstrasse secondary to SWL has been reported in up to 31 % of patients. Obstruction requiring intervention occurs in 6 and 12 % of cases. Ureteroscopic management or less frequently, open ureterolithotomy is necessary in the treatment of steinstrasse. Although preoperative ureteral stenting is controversial, stenting may be wisely indicated in patients with solitary kidneys, unusual renal anatomy or to aid in stone visualization.

Stones in Patients with Bladder Augmentation Cystoplasty

For patients with small contracted bladders, a patch of bowel segment on the existing bladder can be used to increase the capacity for storage purposes. One of the major complications of bladder augmentation is the formation of bladder calculi (Fig. 21.11). The incidence of calculus formation in augmentative bladders is between 18 and 50 %. Possible metabolic causes implicated in the formation of stones in these patients include intestinal mucus as a lithogenic agent and altered levels of inhibitors. Citrate, a known inhibitor of crystallization of stone formation, works by its ability to chelate calcium, thereby reducing calcium stone formation. Therefore, reduced citrate

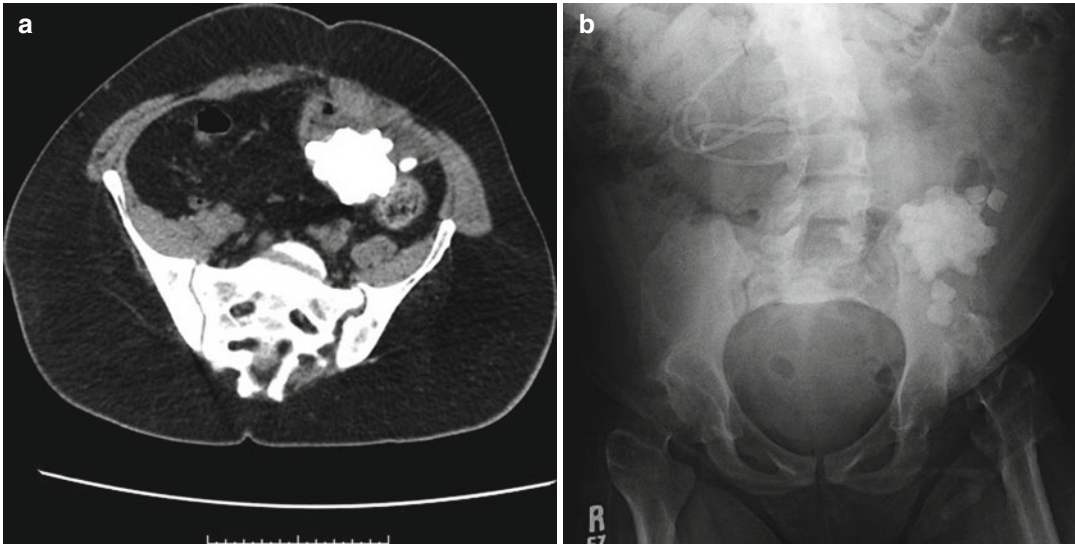


Fig. 21.11 Axial CT scan image (a) demonstrating multiple stones within an augmented bladder; KUB (b) demonstrates that the stone is radiopaque. Following removal

of the stone, the pathology report revealed a mixed calcium oxalate monohydrate and calcium oxalate dihydrate stone

levels in the urine are implicated in bladder stone formation. Placement of a scope through the urethra, called cystoscopy, will allow the use of laser lithotripsy for the break up and removal of these stones without any incisions or use of stitches.

Stones in Patients with Urinary Diversion

Urinary diversion is when a bladder is completely replaced with a section of small or large intestine that has been reconfigured into a reservoir to store urine. These reservoirs end with a stoma that can be non-continent (drain into a bag) or continent (requiring catheterization, to drain the pouch). Stones can form with the reservoir (Fig. 21.12). The prevalence of urolithiasis in patients with urinary diversions varies from 3 to 43 %. Stones produced by Infection may occur after creation of a uretero-intestinal diversion. These stones are most commonly seen between 5 and 10 years after urinary diversion surgery. The etiology of these stones may be due to infection and urea-splitting organisms, foreign body in the reservoir (i.e., staple or suture), mucus produced by the bowel and metabolic disturbances specific

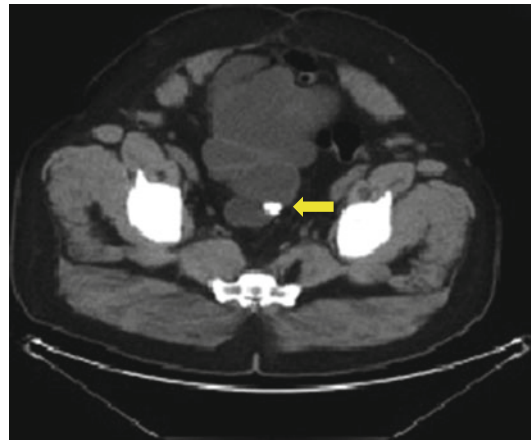


Fig. 21.12 Stone (yellow arrow) within an ileal conduit

to that type of intestine used (i.e., hyperchloremic acidosis). The stones that form may be composed of a mixture of magnesium ammonium phosphate, carbonate apatite, calcium phosphate and calcium oxalate.

Treatment of these stones can be performed through the stoma with a flexible scope, referred to as pouchoscopy. For medium sized stones, they can be managed with laser lithotripsy and residual small stones can be grasped and removed

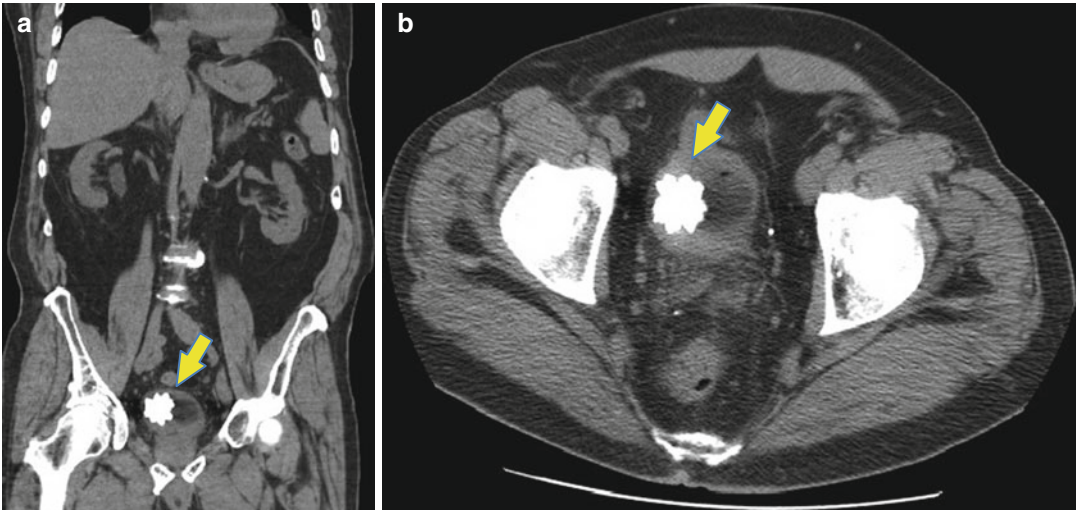


Fig. 21.13 Coronal (a) and axial (b) CT scan images of a large bladder stone (yellow arrow) in a patient with urinary retention secondary to an enlarged prostate

with a basket. For larger stones (several centimeters), a more invasive procedure may be required with either open surgical management or percutaneous pouch access to the reservoir allowing for stone entrapment or intracorporeal lithotripsy. Laparoscopic management can be performed in a highly selected group of patients.

Bladder Stones

Bladder stone formation is predominantly a disease of men. In the United States, bladder stones occur usually in men over the age of 50 and are usually associated with bladder outlet obstruction. The diagnosis of a bladder stone should result in a complete urologic evaluation for factors that result in retention of urine, including stricture of the urethra, benign prostatic hyperplasia, diverticulum of the bladder, and neurogenic bladder. In contrast to renal stones, bladder stones are usually composed of uric acid stones in non-infected urine or struvite stones in infected urine.

In patients with prostatic enlargement and residual urine, the complaints may be those of prostatic obstruction, with the calculus being found incidentally. Typical symptoms of bladder stone are painful voiding with terminal hematuria (blood at the end of the urinary stream). Besides

pain, there may be an interruption of the urinary stream from impaction of the stone at the bladder neck or urethra.

Bladder stones are not often seen on plain films because of the presence of uric acid in many of the calculi and because of overlying prostatic tissue. However, struvite stones, which are radiopaque, may be seen on plain film. All types of bladder stones can be visualized on CT scan (See Fig. 21.13). Ultrasonography is useful for detecting radiolucent calculi. Cystoscopic examination is the best method for detecting bladder stones (See Fig. 21.14).

For large bladder stones, open removal of the bladder stone, called open cystolithotomy, can be performed. If the patient has an enlarged prostate, simultaneous treatment of the bladder stone (cystolitholapaxy) and removal (TURP, open simple prostatectomy) or ablation (GLL or HOLAP) of the prostate can be performed, especially in patients with recurrent stones.

Conclusion

In summary, not all stones are made of calcium, which are simply seen on an abdominal x-ray (KUB) and will pass uneventfully following an



Fig. 21.14 Large bladder stone between lobes of the prostate

ESWL procedure. Diagnostic considerations are important if it is a non-calcium stone, where it is not visualized on a KUB, but instead identified on a CT scan or renal US. In some instances, it may not be seen on a KUB, renal US or even a Spiral CT scan! Also, consider that not all patients have “normal” anatomy of the urinary tract and that anatomical variations should be considered when planning the best treatment options. Finally, not all stones will easily pass through the urinary tract, even when it is normal anatomy, so the correct treatment approach should be considered in these patients. Remember, allowing your Urologist to take the additional time to make the correct diagnosis and the appropriate treatment choices will be worth its “wait in stone!”

Ms. M.E.S is a 36 year old female with a known history of recurrent stones. She had seen several urologists who performed a KUB each time she presented with right flank pain. She had multiple ESWL procedures, but never collected any stones. Follow up KUB studies would confirm broken stone fragments in a confined region within the region of the original stone. Success was measured on the fact that she felt better and the stones were broken up.

She was told to follow up as necessary. A year or two later, she would present with similar pain, and the same work up and treatment choice would be repeated. When she came to us, I was “suspicious” of the fact that she had multiple ESWL procedures for a stone that always presented in the upper part of her kidney, broken or not, and that fragments were never collected. We obtained a spiral CT scan confirming that the patient had a renal diverticulum with multiple small stones in the form of a circular pattern. We discussed the treatment options for this condition, including ureteroscopy and PCNL. She considered a ureteroscopy procedure. URS revealed a tiny opening communicating between the diverticulum and the pelvis of the kidney. A laser was used to create a larger opening between these two locations, allowing a larger passage area. All of the stone fragments were basketed and removed. Stone analysis revealed a calcium oxalate stone. The goal to making the right choices is that patient circumstances dictate the appropriate treatment rendered!

Suggested Reading

1. Paterson RF, Lifshitz DA, KUO RL, Siqueira TM, Lingman JE. Shock wave lithotripsy monotherapy for renal calculi. *Int Braz J Urol.* 2002;28: 291–301.
2. Bhatta KM, Prien EL, Dretler SP. Cystine calculi—rough and smooth: a new clinical distinction. *J Urol.* 1989;142:937–40.
3. Sakamoto W, Kishimoto T, Takegaki Y, Sugimoto T, Wada S, Yamamoto K, et al. Stone fragility—measurement of stone mineral content by dual photon absorptiometry. *Eur Urol.* 1991;20:150–3.
4. Wu TT, Hsu TH, Chen MT, Chang LS. Efficacy of in vitro fragmentation by extracorporeal, electrohydraulic, and pulsed-dye laser lithotripsy. *J Endourol.* 1993;7:391–3.
5. Wang YH, Grenabo L, Hedelin H, Pettersson S, Wikhol G, Zachrisson BF. Analysis of stone fragility in vitro and in vivo with piezoelectric shock waves using the EDAP LT 01. *J Urol.* 1993;149:699–702.

6. Pittomvils G, Vandeursen H, Wevers M, Lafaut JP, De Ridder D, De Meester P, et al. The influence of internal stone structure upon the fracture behavior of urinary calculi. *Ultrasound Med Biol.* 1994;20:803–10.
7. Renner CH, Rassweiler J. Treatment of renal stones by extracorporeal shock wave lithotripsy. *Nephron.* 1999;81 Suppl 1:71–81.
8. Lingeman JE, Siegel YI, Steele B, Nyhuis AW, Woods JR. Management of lower pole nephrolithiasis: a critical analysis. *J Urol.* 1994;151:663–7.
9. Michel W, Funke PJ, Tunn UW, Senge T. Pyelocalyceal diverticula. *Int Urol Nephrol.* 1985;17:225–30.
10. Timmons JW, Malek RS, Hattery RR, Deweerd JH. Calyceal diverticulum. *J Urol.* 1975;114:6–9.
11. Middleton AW, Pfister RC. Stone-containing pyelocalyceal diverticulum: embryogenic, anatomic, radiologic and clinical characteristics. *J Urol.* 1974;111:2–6.
12. Bauer SB. Anomalies of the kidney and ureteropelvic junction. In: Walsh PC, Retik AB, Vaughan Jr ED, Wein AJ, editors. *Campbell's urology.* Philadelphia: WB Saunders; 1998. p. 1708–56.

David A. Schulsinger

Simple Stent Facts

- Unlike cardiac stents that are permanent, straight and metallic, ureteral stents are temporary, curled on both ends and usually made of plastic.
- The purpose of a stent is to provide urinary drainage and dilation of the ureter.
- Stents come in different material and sizes and are used in a variety of circumstances.
- One of the potential consequences of stent placement is stent colic. Patients may experience significant irritative voiding symptoms including urinary frequency, urgency, nocturia and dysuria.

Some stones will move from the kidney and lodge within the ureter causing a blockage, referred to as ureteral obstruction. The back up of urine that ensues will cause the kidney to swell, known as hydronephrosis. This hydronephrosis may translate in significant flank pain, which can eventually bring a patient to the ER. To unblock the ureter, a ureteral stent is placed. The purpose of this chapter is to discuss conditions for which stent placement is indicated and what to anticipate while you have a stent.

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

What Is a Stent?

A ureteral stent is a specially designed hollow tube, made of a flexible material that is easily placed in the ureter. Small perforations along the outside of the stent (Fig. 22.1) allow urine to travel in and out of the tube the same way fish can move in and out of coral reef. Most ureteral stents are referred to as double J stents (i.e., DJ or JJ stent) as demonstrated by the J-shaped curls in the upper and lower portions of the tube (see Fig. 22.2). Unlike a cardiac stent placed in the coronary blood vessels, ureteral stents are temporary, bridging the kidney to the bladder with a tube within the ureter.

Stents are designed in a variety of different materials, different rigidity, different lengths, and different diameters to provide various benefits depending on the situation. Most stents are made of a polyethylene plastic-like material, which is used for ureteral stone or stricture treatment procedures. Some stents are also made of titanium that can withstand obstruction or blockage of the ureter. These stents are used in cancer patients that may experience obstruction or blockage of the tube.

The length of the stents used in adult patients varies between 22 and 30 cm. For children and shorter patients, there are smaller length and diameter stents. These tubes are also used in transplant patients where the stent bridges a short segment of ureter connecting the transplanted kidney to the native bladder.

Fig. 22.1 Small openings in the stent allow urine to drain in and out of the stent



Fig. 22.2 Ureteral stent demonstrating the curls on each end



What Are the Advantages of Having a Ureteral Stent?

A Urologist will place a ureteral stent in a patient for several reasons. These include:

- **Obstruction:** If there is a blockage in the ureter, called an obstruction, this may result in hydronephrosis. A ureteral stent will allow urine to flow from the kidney to the bladder even when the ureter is blocked due to a stone, ureteral stricture, etc. In this situation, the ureteral stent keeps the ureter patent and allows urine produced by the kidney to drain, avoiding potential renal damage, called acute kidney injury (AKI). Severe pain is avoided with the ureteral stent in place, as the kidney is able to drain properly.
- **Infection:** The risk of infection in the urinary tract is reduced with the stent in place.
- **Injury:** On occasion, there can be an injury to the ureter. This may result from blunt (motor vehicle accident) or penetrating trauma (gun-shot wound). Ureteral injuries can occur due to a surgical procedure, called an iatrogenic injury during a urological procedure (i.e., ureteroscopy) or non-urological procedure (i.e., hysterectomy or colon surgery). In this situation, a ureteral stent is inserted to protect the ureter and to insure proper healing. Without the placement of a stent, a severe narrowing of the ureter, referred to as a stricture, may develop resulting in poor drainage of the kidney and possible kidney injury.
- **Swollen ureter:** The irritation of a stone against the walls of the ureter can result in edema and narrowing of the ureter. Likewise, multiple trips with a ureteroscope to remove stone fragments, called ureteral manipulation, may result in irritation and swelling of the ureter, resulting in a smaller lumen. The presence of a ureteral stent allows the swelling to resolve and the ureter to open up to its normal diameter which may take up to 1–2 weeks.
- **Narrowed ureter:** If a stone is severely obstructing the ureter or the ureter is naturally small, it may be too tight for a ureteroscope or

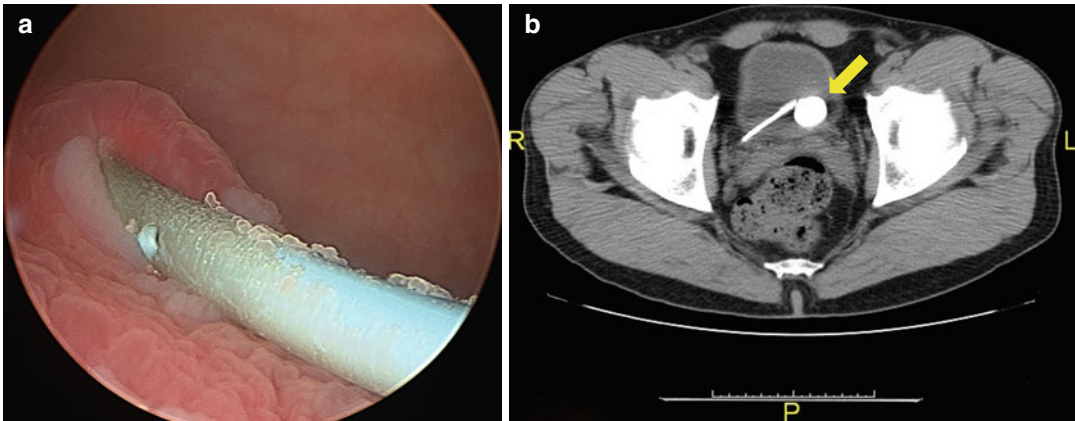


Fig. 22.3 (a) Cystoscopic view from the bladder demonstrating calcifications along the outside of a stent protruding through the ureteral opening (orifice); (b) Axial CT

scan image of a calcified stent (yellow arrow) within the bladder from a different patient

other instruments to pass. If passing a ureteroscope is unsuccessful, a stent is placed to aid in the stretching of the ureter. This dilation may allow the stone to pass. Otherwise, the distention of the ureter over time may aid the urologist to successfully pass a ureteroscope and remove the stone during a future procedure.

- **Prophylaxis:** A patient may undergo a stone procedure (ureteroscopy, ESWL or PCNL) where there are multiple stones in the kidney, referred to as high stone burden. During this procedure, the stone fragments that are generated can get stuck in the ureter forming a column of stones, called *steinstrasse* (German for street of stone). In anticipation of stone fragments resulting in *steinstrasse*, a stent can be placed to avoid a future obstruction.

What Are the Disadvantages of Having a Ureteral Stent?

Most patients will do fine with the inserted stent, some however, may have some symptoms related to the stent. Most individuals are able to endure the ureteral stent without any event. Some patients are unable to accept the stent. It is not possible to forecast which individuals will have side effects from the stent and those that will not. Some of the issues secondary to the stents include:

- **Blood:** Stents can cause **blood** to appear in the urine intermittently or continuously. During physical exertion, the movement of the stent and rubbing of the stent against the bladder can produce blood in the urine.
- **Pain:** Individuals may experience pain in the back (loin), bladder area, groin, penis or testicles in men or urethra in women. This discomfort or pain may be more noticeable after physical exertion and after voiding. This can occur when urine refluxes up to the kidney when passing urine.
- **Irritative voiding symptoms:** The stent can cause irritation to the base of the bladder resulting in irritative voiding symptoms. This includes greater frequency, urgency and getting up at night to void. These symptoms can sometimes be improved by medication.
- **Calcified Stents:** Most ureteral stents can stay in for up to a year. Rarely, do stents become calcified in this period of time. If a stent stays in longer than that, referred to as the “retained” or the “forgotten stent”, the consequence of which is it may become encrusted (Fig. 22.3) and difficult to remove, requiring additional procedure(s) to decalcify and remove the stent.
- **Displaced Stents:** Stents can move from their initially placed position. When this movement occurs, the stent usually migrates into the

bladder causing irritative voiding symptoms (i.e., frequency, urgency), pressure in the bladder and/or blood in the urine.

- **Stent colic:** Typically, the ureter is a one-way street with the flow of urine going in a forward direction from the kidney to the bladder. Occasionally, stents may irritate the bladder generating spasm and causing symptoms of frequency and urgency to urinate. As a result, urine can reflux within the tube to the level of the kidney. This retrograde flow of urine may result in colic of the kidney, or stent colic. Medication can be given to alleviate these symptoms, including narcotic analgesics, NSAID's, phenazopyridine, anticholinergics, beta-agonists and alpha blockers (see Chap. 23).

It is important to re-emphasize that these problems with the stent occur in some patients. Like a prescribed medication, most patients who take a drug are getting the favorable benefits of the medication. Some, however, will have a reaction. This is similar to the stent. Such issues may be short lived only after the stent is first placed and resolve several days later. Rarely, individuals may experience persistent symptoms throughout the treatment course and until the stent is removed.

Are There Any Limitations While I Have a Ureteral Stent?

With a stent in your urinary tract, you are able to do your regular activities. Individuals can go to work and are able to participate in sports with a stent. Individuals may be limited to the extent to which they perform these activities. While there may be greater frequency, urgency and getting up at night to void, this can be curtailed with medications described above.

Individuals are typically concerned about traveling with a stent in place. When an individual with an obstructing ureteral stone, the advantage of traveling with a stent, compared to without, is that the stone is unable to obstruct the ureter and kidney. If a patient has a stone procedure close to the time of travel, my preference is to err on the side of leaving the stent in place until after they return, especially in remote areas where access to a urologist or an emergency room is

limited. With regard to sexual activity, there are no restrictions with a stent in place; however, pleasure may be limited due to the adverse events described above. In summary, the best advice is to listen to your body—*it will tell you what you can and cannot do!*

When Should You Contact Your Urologist?

Following your stent placement, you will most likely have a follow up date with your Urologist to have your stent removed or to discuss your staged procedure to have your stone surgery. However, there may be situations that require you to contact your Urologist sooner:

- **Intractable pain:** If there is constant and unbearable pain related to the stent.
- **Symptoms of infection:** If there are symptoms of infected urine, including fever, rigors, and painful urination.
- **Protruding stent:** If the stent has fallen out or seen protruding at the level of the urethra.
- **Bleeding:** If there is a significant amount of blood in the urine with clots or the blood is thick like ketchup.

How Is a Ureteral Stent Inserted?

I always tell the patient that the short answer for how a stent is placed is “carefully!” A ureteral stent is inserted usually under a general anesthesia either by itself (i.e., stent placement for an obstructing stone) or in combination with another procedure. After advancing the cystoscope is through the urethra and into the bladder, a stent is passed through the cystoscope and into the ureter. The placement and positioning of the stent is performed under direct vision and verified with fluoroscopic x-rays.

Is There an Alternative to a Ureteral Stent?

In some circumstances, there may be a ureteral stone that was removed or ureteroscopy procedure that was uneventful, where there is little to

no irritation to the ureter. Under these circumstances, the urologist may decide that it is not necessary to have a stent placed. This is typically the exception rather than the rule. In managing a patient's expectations, the patient should assume that a stent will be placed during their urologic procedure.

There may be circumstances, however, where a tube is required, but it does not necessarily mean it's a stent. There are two other options to consider:

- **Open-ended catheter:** This is a straight catheter, rather than a catheter with a curl, that extends from the kidney and extends through the ureter and bladder like a ureteral stent, but differs by extending out the urethra. This is a tube that is placed, for example, when contrast needs to be injected into the kidney when a stone is difficult to visualize during an ESWL procedure. These tubes can be simply removed by gently pulling them out without the need for a cystoscopic procedure.
- **Nephrostomy tube:** This is a tube that goes directly into the kidney from the back. The nephrostomy tube is typically inserted by an interventional radiologist in the x-ray suite (rather than the operating room) under local anesthesia. This is the preferred means of draining the kidney in a patient with an obstructing stone with signs of sepsis (i.e., fever or elevated white count). While this tube has a larger diameter and drains better, this type of tube is less desirable than a stent as it extends out the back and drains to a leg bag.

How Is a Stent Removed?

A ureteral stent is removed with a cystoscope performed in the office or in the operating room, under local or general anesthesia, respectively. A special flexible telescope, called a cystoscope, is passed through the urethra. A grasper, called a 3 prong grasper, is passed through the scope and the stent is grabbed and removed under vision. The entire procedure is performed in a few minutes.

Alternatively, the stent is manufactured with a string attached to one end. The stent can be

placed with a string attached. This string will traverse your urethra and will be taped to the inner thigh in women or glans penis in men. This will allow the individual to avoid a cystoscopic procedure when the stent is removed. Discuss with your Urologist prior to your original procedure if there should be a string or *no strings attached!*

Conclusion

In summary, a stent is tube that can be a friend and a foe! The main objective for ureteral stent placement is to divert the urine that is blocked by the ureter. By unblocking the ureter, urine can drain from the kidney to the bladder. Unfortunately, the stent can sometimes have some less desirable side effects that may irritate your bladder. Your Urologist can work with you and even provide some medication that will help relax your bladder and make it less susceptible to stent colic. The saying, "*no pain, no gain*" need not apply!

I was 26 years old when I had my first foreign body placed within my body! The doctor told me it was a double-J stent or DJ stent. My urologist told me the tube was to bypass my stone lodged and stuck in my ureter. Well, the "Disc Jockey" of a stent was making much louder music than I could possibly stand to hear! It was irritating my bladder so, that I had a constant urgency, frequency and burning to urinate. I also had flank pain, something he called stent colic. I thought colic was something only babies had. Well, I must be a big baby, as these symptoms rushed me straight back to the ER. My urologist put me on a medication, an anticholinergic, which relaxed my bladder so that it will not be irritated by the stent. My symptoms completely resolved. This was music to my ears! Now that DJ stent is playing music I can appreciate!

Marco Palmieri and Siddharth K. Dave

Prescription for Pain Facts

- The pain of renal colic is highly variable in severity and nature depending upon the location of the stone.
- Renal colic pain can be very non-specific, mimicking the pain of other more serious conditions, therefore patients should seek medical attention early.
- Pain medications are not curative, but are intended to alleviate the symptoms of renal colic until definitive treatment can be performed.

Objectives of Chapter

1. Identify the common pain pattern and presentation of patients with renal colic
2. Identify commonly used medications to treat the acute pain associated with renal colic
3. Note the characteristics, strengths and limitations of each medication class
4. Identify some frequent misconceptions associated with commonly used medications to treat renal colic

M. Palmieri, DO (✉) • S.K. Dave, MD
Department of Anesthesia and Pain Medicine, Stony Brook Medicine, Stony Brook, NY, USA
e-mail: marco.palmieri@stonybrookmedicine.edu;
siddharth.k.dave@stonybrookmedicine.edu

Introduction

Renal colic associated with a urologic stone is typically episodic and highly variable in intensity. The pain waxes and wanes in severity as the stone travels through the urinary system and is associated with ureteral spasm.

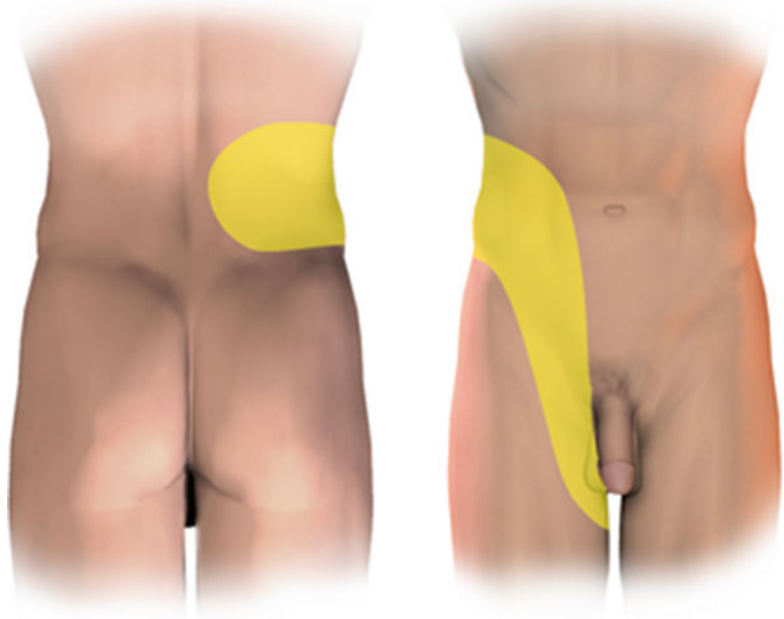
The location of the pain also varies depending on the location of the stone. Stones in the upper part of the genitourinary tract will cause flank pain and tenderness, while obstructions lower down will cause pain radiating to the groin (See Fig. 23.1). It is important to differentiate this pain from other causes of flank and groin pain such as aneurysms, gastrointestinal disease as well as other genitourinary and gynecological pathology.

The classic presentation of a patient with renal colic is writhing in pain and unable to remain in a still position. This is in contrast to a patient with abdominal pain from another origin that typically remains motionless in order to minimize discomfort.

Pain control in renal colic can be achieved with a variety of medications. After the diagnosis of obstructive renal colic is made, the severity of pain will determine the type of medication that the patient should receive. Intravenous pain medication will be needed for severe pain related to renal colic while oral medications should be reserved for patients without the need for surgical intervention as an outpatient.

The ultimate goal of the pain therapy is to allow the patient to tolerate the symptoms until

Fig. 23.1 Referred pain of renal colic from posterior and anterior views



definitive treatment can be performed. Several types of medications can be utilized before and after surgery to help alleviate the pain.

In the following pages we have a first-hand account from a patient that suffered with renal colic and what their experience entailed when they required urgent care. We will then review the various medications that are used to treat the pain associated with renal colic noting the strengths as well as limitations of each. We will also review some common misconceptions associated with these pain medications. Lastly, we will review some commonly asked questions with regard to pain associated with renal colic and the medications used to treat the pain.

Patient Interview

Q What were your first symptoms?

A For a long time I'd noticed small streaks of blood in my urine. Then 1 day I felt a sharp pain in my side and my belly.

Q Describe the pain you felt.

A It was really sharp, it felt like someone cutting me on the inside with a sharp knife. It

was mostly in my side but also went down to my lower belly and my groin.

Q Did you try anything to treat the pain? Did any of it help?

A I tried antacids, aspirin, but none of it helped. I tried drinking lots of water also, but that only made the pain more frequent because I had to go to the bathroom more often. The pain would last for a while then go away on it's own, but would come back later.

Q What made you seek medical attention?

A When the pain persisted for days and I couldn't control it anymore I felt I had to have it treated. It was the worst pain I have ever felt.

Q When you came to the hospital, what happened? How did you feel at that time?

A The first thing they did in the emergency room was place an intravenous line and give me fluids. They also gave me some morphine for the pain, it helped take some of the pain away, but it came back very soon. They also told me that I couldn't have anything to eat or drink.

Q What tests did they run?

A They did blood tests and urine tests, my urine had small amounts of blood in it, so

they did an ultrasound of my side and my bladder. Then they sent me for a CT scan which showed that I have a stone in my urinary system. The doctors said I needed a stent to help urine bypass the stone.

Q What were your concerns at that time?

A My biggest concern was finding relief from my pain, but I was also nervous about the procedure because it sounded like it would be painful.

Q What was your experience during the procedure? Was it painful?

A I don't remember the procedure very much, I was given sedative medication prior to the procedure and was sleeping for most of it. I don't remember any pain during the procedure.

Q How did you feel immediately after and a few days after the procedure? Were you still in pain?

A Immediately after I was very sleepy from the anesthesia, I didn't feel any pain at that time. Once the anesthesia wore off I felt fine. After I left the hospital I had more pain in my side, it was crampy and came in waves. My urologist gave me a pain pill call Percocet as well as Myrbetriq which helped, but it only completely went away after the stent was removed.

Opioids

Opioid medications provide very effective and reliable pain relief for patients experiencing acute to pain (See Table 1). Pain experienced by patients with renal colic is classified in this manner. Chronic opiate use remains a controversial topic however with very little to no available medical evidence. These medications work on opioid receptors located predominantly in the brain and spinal cord to decrease pain intensity. Opioids are an attractive treatment option for a practitioner due to their clinical predictability, multiple routes of administration (i.e. oral, intravenous, rectal, etc.), and effectiveness in the acute pain setting. Side effects include pruritus, nausea, constipation, urinary retention, sedation, and respiratory depression among others. Other considerations when prescribing opioids include

Table 1 Common opiates used in renal colic

Generic name	Brand name	Typical dosing
Morphine	MSIR, MS	5–30 mg 4×/day orally
	Contin, etc.	Variable IV dosing
Hydromorphone	Dilaudid	2–4 mg 4×/day
		Variable IV dosing
Codeine		15–60 mg 6×/day
Meperidine	Demerol	50–150 mg 6×/day
Hydrocodone	Norco, Vicodin	5–10 mg 3–4×/day
Tramadol	Ultram, Ryzolt	50–100 mg 3×/day
Oxycodone	Percocet,	5–10 mg 3–4×/day
	Roxicodone	

the risk of dependence, abuse, withdrawal and diversion. For this reason, it is important to counsel the patient on opioid safety and risks when initiating opioid therapy for outpatient use. Inpatient opioid administration should be done with careful monitoring, especially in opioid naïve patients. We will explore some of the more commonly used opiates for use in the management of renal colic below.

Morphine

Morphine is the “gold standard” with regard to opioid medications. All other opioid potencies are compared to that of morphine. It is available in a variety of formulations including intravenous and oral formulations which are the most commonly used for renal colic. For intravenous morphine dosing is typically 2–5 mg to start every 5 min to achieve adequate analgesia. The onset of action is typically 5–10 min. Metabolism is via the liver resulting in active metabolites that are renally excreted. This can result in unwanted accumulation and significant side effects in patients who have impaired renal function due to their renal disease [1].

Hydromorphone

Hydromorphone (Dilaudid) is a more potent opioid than morphine with equianalgesic ratios of approximately 1.5 mg of hydromorphone being equivalent to approximately 10 mg of morphine.

Dosing is started at 0.2–1 mg intravenously every 2–3 hours. Hydromorphone typically starts working in 5 min but takes 10–20 min to take full effect. Metabolism is via the liver with non-active metabolites excreted by the kidneys. Oral formulations are available for outpatient use but should be reserved for patients who are opioid tolerant and are already on oral hydromorphone [2].

Meperidine

Meperidine (Demerol) was the first synthetically produced opioid analgesic. Its use has been falling out of favor in recent years due to its significant side effects. Meperidine is metabolized by the liver to normeperidine. This active metabolite has approximately half of the analgesic effect of meperidine but two to three times the central nervous system effects. In addition, normeperidine has a half-life of 15–30 hours, much longer than the 2.5–4 hours half-life of meperidine. Accumulation of this metabolite can occur with doses greater than 600 mg per day as well as in patients with renal dysfunction. The central nervous system effects can range from irritability and muscle twitching to hallucinations and seizures. For these reasons, along with high abuse potential, it is not recommended that meperidine be used for pain control in the setting of renal colic [3].

Oxycodone

Oxycodone (ex. Roxicodone) commonly prescribed for management of pain and is available in

both a pill and as a liquid either by itself, or in combination with acetaminophen (Percocet, Endocet, Roxicet). Onset of action is within 15 min and lasts 3–6 hours. Dosing is typically 5–10 mg every 4–6 hours as needed. It is important to note that patients may want to take higher or more frequent doses but limitations on the maximum amount of acetaminophen (3 g per day in healthy adults) should be taken into account [4].

Tramadol

Tramadol (Ultram) exhibits weak opioid receptor agonist properties as well as inhibition of the reuptake of norepinephrine and serotonin. Onset of action is within an hour and lasts approximately 9 hours. Typical doses are 50–100 mg every 4–6 hours. Metabolism is via the liver and excreted by the kidneys. For this reason, it is important to remember to reduce the dose in patients with significant renal disease [5].

NSAIDs

Non-steroidal anti-inflammatory drugs (NSAID) are used to treat a wide variety of pain to conditions (See Table 2). By inhibiting cyclooxygenase (COX) and therefore the synthesis of prostaglandin from arachidonic acid, NSAIDs offer several advantages as well as significant side effects. NSAIDs, via inhibition of the COX-2 enzyme subtype, possess potent anti-inflammatory properties in addition to reducing pain and fevers. However, COX-1 is responsible for regu-

Table 2 Common NSAIDs used in renal colic

Generic name	Brand name	Adult daily dosing/frequency	Usual 24 hour adult dose range
Aspirin	Bayer	325–1,500 mg 4×/day	2.4–6 g
Naproxen	Naprosyn	250–500 mg 2×/day	750 mg–1 g
Ibuprofen	Motrin, Advil	200–800 mg 4×/day	1.2–2.4 g
Diclofenac	Voltaren	50–100 mg 2–3×/day	150–200 mg
Ketorolac	Toradol	15–60 mg 4×/day	60 mg (no more than 5 days)
Meloxicam	Mobic	7.5–15 mg once daily	15 mg
Celecoxib	Celebrex	100–200 mg 2×/day	400 mg
Acetaminophen	Tylenol, Ofirmev	500–1,000 mg 3×/day orally Up to 1 g 3×/day IV	3,000 mg

lating renal blood flow and maintaining gastric mucosa. As a result, the use of non-selective NSAIDs may cause patients to have decreased renal perfusion and impairment. In addition, patients may have significant and varying gastrointestinal side effects ranging from irritation to ulcerations, perforations and gastrointestinal bleeding. Other important side effects to take into consideration when administering NSAIDs are the risk of bleeding due to decreased platelet aggregation and increased risk of cardiovascular thrombotic events. Despite this, however, NSAIDs are the most commonly used pharmacologic therapy in the treatment of pain. Several different NSAID formulations exist and are offered in both intravenous and oral forms.

A 2004 Cochrane review found that patients receiving NSAIDs, as compared to opioids, achieved better pain scores and were less likely to require additional pain medication in the short term. It was concluded that NSAIDs were recommended over opioids for single dose administration and where titration of opioids might be difficult. It is worth mentioning that several of the included studies compared NSAIDs to single dose opioids versus in titrated doses which might result in a differing conclusion.

Ibuprofen

Ibuprofen (Advil, Motrin, Caldolor) is available over the counter in oral form or as a parenteral medication. Oral ibuprofen dosing is generally 200–400 mg every 4–6 hours up to 1.5–2 g per day. Onset of action is between 30 and 60 min with peak effect taking 1–2 hours. Half-life is approximately 2–4 hours. Caldolor available as an intravenous medication has a faster onset of action and dosing is recommended at 400–800 mg every 6 hours up to 3.2 g per day [6].

Indomethacin

Indomethacin is a non-selective COX inhibitor used for the treatment of pain. Onset of action is in approximately 30 min with peak effect taking

up to 2 hours. Analgesic benefit generally lasts 4–6 hours. Dosing for oral indomethacin adults is 20 mg up to three times a day or 40 mg two to three times a day [6].

Ketorolac

Ketorolac (Toradol) is also a non-selective COX inhibitor with both oral and intravenous formulations available. However, the oral form is generally considered to be used as a continuation of the intravenous form for up to 5 days. Dosing for parenteral ketorolac is 30 mg every 6 hours. Ketorolac can also be used intramuscularly in patients without intravenous access. Dosing in this case is the same with a maximum daily dose of 120 mg. Onset of action is approximately 30 min and 2 hours until full effect. Duration of analgesia is 4–6 hours [6].

Acetaminophen

Although not fully elucidated it is thought that Acetaminophen (Tylenol, Ofirmev) works via inhibition of prostaglandin synthesis. Available over the counter as an oral pill in 325 and 500 mg tablets, dosing is 325–650 mg every 4–6 hours or 1,000 mg three times per day with a maximum daily dose of 3 g. Ofirmev is an injectable intravenous solution of acetaminophen and dosing is 1,000 mg every 6 hours with maximum daily dosing also at 3 g. Onset of action for oral acetaminophen is less than an hour and approximately 5–10 min for the intravenous formulation. Duration of action is both 4–6 hours. Acetaminophen is extensively metabolized by the liver and strict adherence to maximum daily dosing is paramount to prevent hepatic cell necrosis. Acetaminophen should also be used with caution in patients with preexisting liver disease [7].

Anticholinergic

Muscarinic acetylcholine receptors mediate several important functions in the body. Of The bladder detrusor muscle, responsible for contracting

the bladder, works by way of muscarinic acetylcholine receptor subtype M3. Anticholinergics and newer more targeted drug therapy to inhibit these receptors have been developed to reduce the renal colic pain associated with stent placement and resultant bladder muscle spasm and pain. It is worth noting that conflicting results regarding anticholinergic therapy and pain reduction have been reported. Although it has not been shown to facilitate stone passage at the intramural junction of the bladder, the anticholinergic effect has been shown in some studies to decrease pain, particularly tolterodine.

As a whole antimuscarinic medications tend to have a wide range of side effects due to the ubiquity of effect sites. These include dry mouth, constipation, headache, nausea, confusion, palpitations and syncope. In addition, anticholinergic medications are contraindicated in patients suffering from uncontrolled narrow angle glaucoma as well as urinary and bowel retention. However, more targeted therapy to the M3 subtype has decreased the incidence of the complications, the M3 subtype is responsible for gastric smooth muscle, bladder detrusor muscle and pupillary ciliary control.

Solifenacin (Vesicare)

Solifenacin is an antimuscarinic with strong M3 receptor activity. Dosing is 5 mg once daily which can be increased to 10 mg if tolerated. However, due to its significant hepatic metabolism and renal excretion, patients with preexisting hepatic disease and renal impairment should be carefully dosed [8].

Darifenacin (Enablex)

Darifenacin is a competitive antimuscarinic with strong affinity for the M3 receptor subtype. Initial dosing should be started at 7.5 mg daily which may be increased to 15 mg daily. Hepatic metabolism should be taken into account particularly with medications that inhibit the activity of CYP 3A4 [9].

Tolterodine (Detrol/Detrol LA)

Tolterodine also has high affinity for the M3 muscarinic subtype and is available in both immediate and extended release formulations. The immediate release tablet is dosed at 2 mg twice daily whereas the extended release capsule can be given at 4 mg daily. Metabolism is via hepatic enzymes and care should be taken with patients already on inhibitors. Half-life is highly dependent on genetic variables and concurrent interactive therapies ranging from 2 to 10 hours for the immediate release and 7–18 hours for the extended release [10].

Oxybutynin (Ditropan, Oxytrol)

Oxybutynin is a non-selective antimuscarinic receptor blocker particularly the M1, M2 and M3 subtypes. Several formulations exist including immediate and extended release tablets as well as topical gels and transdermal patches. The immediate release dosing of oxybutynin is 5 mg two to three times daily with a maximum daily dose of 20 mg. The extended release can be given 5–10 mg once daily and titrated upwards to a maximum of 30 mg daily [11].

Trospium (Sanctura/Sanctura XR)

Trospium has the highest affinity for the muscarinic receptor subtypes but does so with the least selectivity. Dosing is generally 20 mg twice daily for the immediate release formulation whereas the extended release is prescribed 60 mg once daily. Metabolism is by the liver however, unlike other anticholinergics previously described, trospium displays minimal metabolism by the cytochrome P450 system resulting in less interaction with medications [12].

Beta Agonists

Beta adrenergic receptors (B1, B2, B3) are found in human detrusor muscle and urothelium with dominance of the B3 subtype. In addition to smooth muscle of the urinary tract, B3 receptor

subtypes can also be found in the gall bladder, adipose tissue, cardiac tissue, the brain as well as the myometrium. Activation of the B3 subtype is responsible for smooth muscle relaxation of the urinary tract. This allows for targeted treatment in bladder relaxation in the treatment of overactive bladder.

Mirabegron (Myrbetriq)

Mirabegron is a selective B3 adrenergic receptor agonist with little to no activity on the other receptor subtypes.

Following stent placement mirabegron can be used to reduce ureter spasm for better tolerance of the stent reducing pain medication requirements. Mirabegron undergoes hepatic metabolism and renal excretion. Adverse reactions include hypertension, tachycardia, headache, arthralgia, constipation and upper respiratory tract infections. Care should be taken when patients are at risk for hypertensive episodes and other cardiac events.

Dosing of mirabegron starts at 25 mg one a day to assess tolerance and may be increased to 50 mg once daily as needed. It should be noted however that steady state levels of the drug took 7 days to develop [13].

Frequently Asked Questions

How does the pain of renal colic typically present?

Pain associated with renal colic usually comes on *acutely* or abruptly. Patients with renal colic usually describe pain in the flank area that often radiates to the groin. It is frequently described as the worst pain a patient has ever experienced in their lifetime.

Why do patients get pain with kidney stones?

The pain associated can be attributed to many causes including obstruction of the urinary flow, increased pressure on the urinary tract wall, muscle spasm in the wall of the ureter, and inflammation near the stone.

What treatment options are available for the pain associated with kidney stones?

Pain due to kidney stones is often treated with intravenous and oral medications. Some of the medication classes typically used are opioids, NSAIDs, and acetaminophen (Tylenol).

What is an opioid?

Opioid is a term that is used for a medication which has similar pain relieving effects and properties of morphine. Opioids work by attaching to opioid receptors which are located throughout the body. The opioid receptors which are most responsible for pain relief are located in the brain and spinal cord.

What is an NSAID?

NSAID stands for non-steroidal anti-inflammatory drug. In addition to their anti-inflammatory effects these drugs have analgesic (pain relieving) and anti-pyretic (fever reducing) effects. These medications work by stopping the production of certain chemicals in the body which are responsible for inflammation.

What are some of the risks associated with opioids?

The most common side effects associated with opioids are constipation, nausea and vomiting, drowsiness, itching, and dry mouth. There are other more serious side effects such as respiratory (breathing) depression, tolerance, and addiction among others.

Will I get addicted to opioid pain medication if I take them for pain associated with renal colic?

The term addiction is misunderstood among patients and providers. According to the American Pain Society, American Society of Addiction Medicine, and American Academy of Pain Medicine addiction is a complex condition that is characterized by various behaviors that include one or more of the following: impaired control over drug use, compulsive use, continued use despite harm, and craving. The risk of addiction in the acute pain setting (ex. renal colic) while present, is exceedingly low.

What is tolerance?

Tolerance is a term used to describe decreased effect from drug despite stable doses or increasing doses required to provide equal effect. Tolerance is distinct from addiction and one does not imply the other

What are the risks associated with the use of NSAIDs?

The most common side effects with NSAIDs are related to the GI (gastrointestinal), cardiovascular and renal (kidney) systems. These adverse effects range from minor (ex. stomach upset, nausea) to major (ex. Heart attack, kidney damage/failure, excessive bleeding).

Can acetaminophen be used for renal colic pain?

Acetaminophen is a safe and effective pain medication with fewer side effects than both NSAIDs and opioids. These medications can certainly be used for the pain associated with renal colic either alone or in combination with other analgesics.

Are there side effects associated with Tylenol? Is there a maximum recommended dosage?

As with almost any medication there is a risk of allergic reaction. The most concerning side effect associated with acetaminophen however is related to liver toxicity (damage). The maximum recommended dosage is 3,000 mg/day.

Points to Remember

The pain of renal colic can vary from one episode to the next, and from one patient to another. Because the symptoms can be very similar to other conditions, some serious, some not, contact your physician sooner rather than later.

There are many medications available to alleviate the pain of renal colic most notably opioids and anti-inflammatories. Because improper or excess use of pain medications can be as dangerous as any other medical conditions, your physician should help you decide which medications are safest and most appropriate for you. Do not rely on these medications to mask your symptoms; instead use them to relieve your pain while your urologist treats the underlying problem.

While tolerance can occur with anyone taking opiate pain medications over a period of time, it should not be confused with addiction; nor should it be a reason to shy away from necessary treatment of your pain.

References

1. Lvovschi V, Aubrun F, Bonnet P, et al. Intravenous morphine titration to treat severe pain in the ED. *Am J Emerg Med.* 2008;26(6):676–82.
2. Drugs for pain. *Med Lett Drugs Ther.* 2000;42(1085):73–8.
3. Clark RF, Wei EM, Anderson PO. Meperidine: therapeutic use and toxicity. *J Emerg Med.* 1995;13(6):797–802.
4. Kalso E, Vainio A. Morphine and oxycodone hydrochloride in the management of cancer pain. *Clin Pharmacol Ther.* 1990;47(5):639–46.
5. Grond S, Sablotzki A. Clinical pharmacology of tramadol. *Clin Pharmacokinet.* 2004;43(13):879–923.
6. Brooks PM, Day RO. Nonsteroidal anti-inflammatory drugs – differences and similarities. *N Engl J Med.* 1991;324(24):1716–25.
7. Holdgate A, Pollock T. Nonsteroidal anti-inflammatory drugs (NSAIDs) versus opioids for acute renal colic. *Cochrane Database Syst Rev.* 2004;(1):CD004137.
8. Cardozo L, Lisec M, Millard R, et al. Randomized, double-blind placebo controlled trial of the once daily antimuscarinic agent solifenacin succinate in patients with overactive bladder. *J Urol.* 2004;172(5 Pt 1):1919–24.
9. Croom KF, Keating GM. Darifenacin in the treatment of overactive bladder. *Drugs Aging.* 2004;21(13):885–92.
10. Chancellor M, Freedman S, Mitcheson HD, et al. Tolterodine, an effective and well tolerated treatment for urge incontinence and other overactive bladder symptoms. *Clin Drug Invest.* 2000;19:83–91.
11. Chapple CR. Muscarinic receptor antagonists in the treatment of overactive bladder. *Urology.* 2000;55(5 Supp 1):33–46.
12. Pak RW, Petrou SP, Staskin DR. Trospium chloride: a quaternary amine with unique pharmacologic properties. *Curr Urol Rep.* 2003;4:436–40.
13. Bridgeman MB, Friia NJ, Taft C, Shah M. Mirabegron: B3-adrenergic receptor agonist for the treatment of overactive bladder. *Ann Pharmacother.* 2013;47:1029–38.

Part VI
Prevention

David A. Schulsinger

Summary Stone Facts

- Stone analysis is an essential test to determine the type of stone and aid in options for treatment and prevention.
- 24 hour urine test can determine excess stone-forming minerals or deficiency in stone prevention substances.

The risk of stone recurrence is 50 % over 5 years (approximately 10 %/year) or 80 % over your lifetime. Identifying risk factors associated with a patient's stone can help minimize their risk for future stone disease. This chapter discusses the appropriate tests necessary for treatment of a stone and ultimately, stone prevention.

Stone Analysis: *Strain and Retain Your Stone!*

The stone analysis is an essential test to evaluate the type of stone and aid in treatment options for treatment and prevention. Patients have the ability to collect their stones in various scenarios. The stone may pass on it's own; the physician may break the stone up and the patient subsequently col-

lects these fragments; or the physician may collect stones obtained during the surgical procedure.

When an individual has a small, asymptomatic stone, they are typically managed by conservative care, straining their urine in an attempt to capture their stone in stone cup (See Fig. 24.1). When a patient undergoes shock wave lithotripsy, they will strain their urine for broken stone fragments. Patients with large stones may undergo a PCNL procedure by which the physician removes stones during the procedure and subsequently sends them to pathology for evaluation. A stone former should always be mindful that one of their goals is to collect stone fragments for stone analysis to assist in stone assessment and potentially future stone prevention.



Fig. 24.1 Stone Cup Strainer (Courtesy of Advanced MedConnection, Inc.)

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Depending on the size and shape of the stone, this may take a few hours, to several days or even as long as a few weeks. Drinking plenty of fluids enhances the chances for the stone's passage from the kidneys to the urethra. It is important to continue to drink fluids and maintain hydration. The more fluids you consume, the more urine you will make and the more likely the stone will pass quickly out of your urinary tract system.

Once the stone is collected, your Urologist will send the stone for testing. This analysis reveals the composition of the kidney stone and enables your doctor to institute a plan to possibly prevent the formation of future stones. While straining the urine can be inconvenient, it is important to complete this task to make the appropriate diagnosis and aid in the treatment strategy. All stones fragments, no matter how large or small, should be collected and sent for stone analysis. This evaluation is essential and will shed light onto the etiology and pathogenesis of the renal stone so that therapy can be optimized. *Remember....No strain, no gain!*

24 hour Urine Test: The "World Series" of Urine Tests!

Obtaining a urine analysis for your physician is a test that will check for blood, bacteria, leukocytes and specific gravity of the urine. This is the equivalent of a regular baseball game. The 24 hour urine, however, is the grand test, where urine is collected for a continuous 24 hour period to determine all the factors responsible to determine potential causes of the patient's stone. This, some may argue, is the World Series of urine tests!

Sometimes your urologist may need to know how much urine your body is producing in a day or how much of a particular substance (for example, calcium, sodium, potassium, creatinine or blood urea nitrogen) is eliminated in 24 hour. A single specimen urinalysis cannot provide this information, so patients are instructed to collect all of their urine produced in a 24 hour period. A special container is provided for this purpose, which is returned to the laboratory after you have finished the urine collection at home (see Fig. 24.2).

The 24 hour urine will help your Urologist determine if your stone is related to diet, metabolic factors, or dehydration. Dehydration, for



Fig. 24.2 24 hour urine container (Courtesy of Therapak)

example is not how much fluid goes into the tank or body, but how the body mobilizes the fluid. So, for example, a patient who drinks ample amounts of fluids but sweats or has loose bowel movements that exceed his/her hydration, can become dehydrated and may be at risk for stones.

When Is the Appropriate Time for 24 hour Urine Test

Your urologist will determine when is the appropriate time for you to collect your 24 hour urine. Many times, if you are presenting with a symptomatic stone, your urologist will want to treat your stone first, followed by obtaining a 24 hour urine. This is done namely out of convenience for the symptomatic patient. For the patient with the asymptomatic stone, the 24 hour urine can be done right away. Likewise, for the patient with a known history of stones but not a current stone, who is undergoing preventive care, the urologist will determine the appropriate time for doing this test.

I always urge my patients to do their 24 hour urine test on a non-working day as they do not want to carry an orange jug around the office!

Preparation for the 24 hour Urine Test

Certain medications that you are taking may affect the test results of your 24 hour urine study. Your Urologist will determine which drugs to

discontinue and which to continue during the test. Never discontinue medication without authorization from your physician.

A patient's urine is a reflection of their eating habits. Therefore, individuals are encouraged to maintain their regular eating and drinking habits and not to artificially change their diet at the time of the study. If a patient is on a diet for specific health reasons, I ask the patient to continue these food choices, especially if this diet will be maintained. While this may represent a diet quite different from the regime contributing to their previous stone, it will help us learn if their current food choices are lithogenic and a potential cause for a future stone.

There are some factors that may influence the results of the 24 hour urine that may not be part of your normal daily activities or appropriate situations. These include heavy exercise, urinary tract infection, X-ray examination with contrast within 3 days prior to urine test and dehydration. Be certain to do this test at the right time and under the right circumstances.

How Do You Do the 24 hour Urine Test?

Urine is collected over a 24 hour period beginning with the second urine on the first day and ending with the first urine on the second day. After discarding the first urine of the day, all your further urine should be collected in the same container, including the first urine of the morning of the next day.

The 24 hour urine is certainly a way to keep you around the house. Being housebound, allows patients to obtain a complete specimen. Female patients can use a "graduated hat" for the collection of the urine.

Results of the 24 hour Urine Test

This 24 hour urine collection will be studied for chemicals in the urine that promote and inhibit stone formation. The specimen is analyzed for total urine volume, calcium, phosphorous, uric acid, creatinine, oxalate, magnesium, sodium, citrate, sulfur, urine pH, calcium oxalate supersaturation

(SS CaOx) and uric acid supersaturation (SS UA). The urine test is helpful in identifying risk factor(s) for kidney stones. The most common cause for stones and the most common factor identified on the 24 hour urine test, for example, is a low total urine volume. Ideally, the test will verify that the patient is consuming enough fluid to minimize their risk for future stones. Patients must achieve a urine volume of 2 l/day.

The Poor Man's 24 hour Urine Test

If a patient demonstrates only a single risk factor for stones, namely a low total urine volume, I will not subject this patient to multiple 24 hour urine tests to verify adequate hydration. I will, however, offer them an alternative, the "*poor man's 24 hour urine test*." Monitoring the urine color during voiding is what we refer to a "poor man's 24 hour urine." The color of the urine can help determine a patient's hydration status. Recall that the kidneys regulate the fluid balance in our bodies. When a patient is dehydrated, the kidneys respond by sequestering water, causing the urine to become more concentrated and darker. Thus, the golden color of urine can indicate dehydration. If the urine is concentrated, the patient knows that he should drink more. Alternatively, when a patient is adequately hydrated, the kidneys respond by opening the "flood gates" to allow excretion of excess fluids. If the urine is clear as water, then the patient is well hydrated. Use your last urine as an indicator for what you will do for the next 4 hours. The goal for stones patients is to maintain clear urine.

Is It Necessary to Repeat a 24 hour Urine Test?

For some patients, there may be multiple factors on their 24 hour urine test that puts that individual at risk for future stones. Besides hydration, there may be dietary implications and metabolic factors contributing to stone disease. If your stone is due to dietary factors, the 24 hour urine can determine which food products you can continue to eat and which that you should avoid. For metabolic factors, there are supplements, medications and vitamins to

choose from. Once a treatment regimen is instituted, follow up and repeat 24 hour urine tests will be required to be certain the stone risk factor(s) has been addressed and the risk for future stones has been minimized. In addition, each time a patient forms a stone, we repeat a 24 hour urine test. Your Urologist will determine the interval and number of 24 hour urine tests required.

In summary, stone prevention is like a pendulum on a clock. You identify certain risk factors and you treat them accordingly. The next time when the pendulum swings to the opposite side, there is a whole new set of risk factors to address and treat. The goal with stone prevention is to stay ahead of the curve and not to play catch-up!

Serum Chemistry (BUN, Cr)

Serum levels of blood urea nitrogen (BUN) and creatinine (Cr) are useful tests in assessing any individual's kidney function. These tests are usually ordered as part of a basic or comprehensive metabolic panel. Elevated BUN and creatinine are typically seen in patients with renal insufficiency or renal failure. Increased BUN and creatinine may also be seen in patients with urinary tract obstruction secondary to ureteral stone or other causes. Your urologist may request a basic or comprehensive metabolic panel to evaluate your kidney function.

Serum Uric Acid Test

Serum uric acid is a test to determine how much uric acid is in your blood. Uric acid is a chemical produced when the body breaks down food products called purines. Hyperuricemia, or elevated uric acid levels can occur when the body overproduces uric acid. With respect to stones, hyperuricemia may suggest an acute attack of gout, acute kidney failure, kidney stones, bone marrow disorder (leukemia), metastatic cancer or a diet high in purines. This test is used to evaluate patients with gout, monitor patients who were undergoing treatment for cancer (i.e., chemotherapy or radiation) and to determine the etiology of a patient's kidney

stone (i.e., uric acid stones). Alcohol, certain medication (i.e., aspirin, Ibuprofen, Motrin), excess vitamin C, and x-ray dyes may interfere with the serum uric acid test results. Consult with your physician about prescription medication and supplements that you are taking.

Serum Calcium Test

Serum calcium is a test to measure the calcium level in your blood. The total calcium test is the test most frequently ordered to measure the calcium status. This test reflects the calcium bound to protein and the ionized (free) calcium. In most situations, the total calcium is a good reflection of the amount of free calcium present in the blood since the balance between bound and free calcium is stable and predictable. In certain circumstances, however, the balance between bound and free calcium is disturbed and it may not a good reflection of the calcium status. In these situations, ionized calcium may be more appropriate.

A serum calcium test is ordered when patients are worked up for calcium stones, especially if the urinary calcium is elevated (hypercalciuria). Hypercalcemia, or elevated serum calcium, is suspected in patients with hyperparathyroidism, sarcoidosis, thiazide diuretics use, excess vitamin D intake and certain cancers, and a serum calcium test is ordered in these circumstances.

In summary, when serum calcium test is ordered in patients with calcium stone disease, total and ionized calcium are usually obtained.

Serum Parathyroid Hormone Test

Parathyroid hormone is a chemical produced by the parathyroid gland. The parathyroid hormone helps to regulate the calcium in the blood. This test may be useful in determining which patients may be at risk for calcium phosphate stones. When serum levels of parathyroid hormone are elevated, referred to as hyperparathyroidism, these patients are at risk for forming calcium phosphate stones. Your urologist will determine if this test is necessary and appropriate for you.

Serum Vitamin D

Serum Vitamin D levels are used to screen patients who are at high risk of deficiency and vitamin D excess. High levels of vitamin D can be seen in patients with sarcoidosis and lymphoma. Patients with Vitamin D deficiency can be seen in patients with kidney disease; patients who experience fat malabsorption, such as patients with gastric bypass or Crohn's Disease. Up to 80 % of stones formers showed Vitamin D deficiency or insufficiency [1, 2]. Your urologist will determine if serum Vitamin D levels are required during your work up.

Urine Analysis, Culture and Sensitivity (UA, C & S)

A single sample of urine is normally used to determine whether there are increased amounts of specific substances in your urine, such as glucose, red blood cells, white blood cells, or protein. This test will also tell you the presence or absence of bacteria in the urine, urinary crystals and the urine pH. The urinalysis is a "snapshot" assessment of your urine at one point in time. The urinalysis is a valuable screening tool for urinary tract infections, kidney disease, and other conditions.

Urine Dipstick

A urine dipstick is a quick way checking factors in the urine. For example, it may check for blood and urine pH. This test is sometimes performed in the emergency room when a patient shows symptoms associated with a kidney stone (i.e., flank pain). Approximately 2/3 of kidney stone patients will present with blood and urine, called hematuria. While this is not a definitive test for the work up of stones, it can be used as an initial screening test.

The dipstick urine pH test is important in assessing whether an individual's urine puts them at risk for a certain stone types (i.e., uric acid or calcium phosphate stones). This test can also be used to determine if a patient is being

managed appropriately with certain medication to treat a stone.

Conclusion

In summary, stone prevention begins with two important tests, the stone analysis and 24 hour urine. The stones analysis will determine the type of stone that was formed and type of stone to prevent. The 24 hour, at-home urine collection allows urologists to determine the cause of your kidney stones and provide appropriate treatments. The results of 24 hour urine combined with a patient's stone analysis can determine the factors for stone disease in as much as 97 % of cases. The results can also guide our recommendations for dietary changes and medications to help prevent future occurrences. Additional tests my help to further define the cause of the stone. Collectively, these tests will determine the cause of stone and establish the treatment regimen for stone prevention. Understanding the patients' stone and urinary parameter will aid in determining what medical therapy and/or dietary therapy to prevent future stones.

Mr. HM presented with a 4 mm ureteral stone and was asymptomatic now that he was consuming generous amounts of fluids. He was informed about the importance of straining his urine so the collected stone can be analyzed and provide information necessary to prevent future stones. Every day as well as each and every place he went, he religiously strained his urine for the "chunk of gold." It was not until he went to a major league base ball game to watch the NY Mets at their former home, Shea Stadium. There, in the top of the second inning, he went to the lavatory. Miraculously, he passed his stone. Unfortunately, he was poorly prepared for the encounter. He stared at the stone sitting still at the bottom of the toilet, in a public

rest room, used by hundreds of people each day. He knew that his urologist requested the collection of this “anatomical prize”, but he knew there was NO way he could collect that stone with only his bare hands with a line of people waiting behind him, in hopes to see their Mets batting in the bottom of the inning. Needless to say, the stone never left Shea Stadium. *Remember, there is no good time or bad time to pass a stone!*

Dr. TM is a podiatrist patient and frequent stone former. He made multiple uric acid stones for over 30 years and now presented to me with a 3.5 cm left renal staghorn calculus. While this stone was identified by CT scan, the Hounsfield units being high (>900) and the presence of the stone on the KUB suggested that it was calcium-based.

He underwent an elective PCNL procedure and was discharged on POD#1 and underwent an uneventful postoperative course with his nephrostomy tube removed on POD#5.

Following his nephrostomy tube removal, he underwent a metabolic work up. His stone analysis revealed a calcium oxalate monohydrate stone; his 24 hour urine revealed a high urinary oxalate (51 mg/day), low total urine volume of 1.7 L and low urinary citrate (400 mg/day). I counseled the patient on choosing foods that were low in oxalate (see Chap. 27). In addition, I explained to the patient that we needed to increase his fluid consumption to raise his urinary output and to increase his consumption of liquids that would raise his urinary citrate level. I told him that treatment of his stone was simple! By drinking more lemonade to supply a rich source for fluids and citrate, he would be **killing 2 birds with one stone!**

References

1. Elkoushy MA, Sabbagh R, Unikowsky B, Andonian S. Prevalence and metabolic abnormalities of vitamin D-inadequate patients presenting with urolithiasis to a tertiary stone clinic. *Urology*. 2012;79:781–5.
2. Eisner BH, Thavaseelan S, Sheth S, et al. Relationship between serum vitamin D and 24 hours urine calcium in patients with nephrolithiasis. *Urology*. 2012;80:1007–10.

Hydration: Why We Drink, When to Drink, What to Drink, and How Much to Drink, That Is the Question!

25

David A. Schulsinger

Favorable Fluid Facts

- Low urine volume is the most common abnormality associated with stone disease.
- The most important action a person can take to prevent kidney stones is drinking enough fluid.
- People with a history of kidney stones should consume enough water and other fluids to make at least 2 L of urine per day.
- Not all fluids are equally beneficial in reducing your risk of stone disease.

Water is the most quintessential nutrient in our body and to our life as it represents 60–70 % of our adult body weight. Water is the fluid in which all life processes occur. Water is essential to maintain the structure of our cells, aids in regulation of our body temperature, maintains our blood volume, aids in body metabolism, assists in all digestion and absorption functions, lubricates mucous membranes in the gastrointestinal and respiratory tracts, assists in excretion of waste from bowel and kidney, the major component to digestive juices. Without it, we are not able to survive. Consuming too much water can result in water intoxication, an event that is very rare.

D.A. Schulsinger, MD
Department of Urology,
Stony Brook Medicine, Stony Brook, NY, USA
e-mail: endourology@yahoo.com

When we do not drink enough water, dehydration may result. Dehydration is the most common cause for stone formation. Dehydration comes in different shapes and forms. It does not always imply that there is inadequate water going into our system, it also refers to how we manages these fluids. Indispensable water losses refer to water lost from our body in ways other than from the urine. These alternative water losses include breathing, sweating and diarrhea. In addition, hydration is the key to stone prevention. The purpose of this chapter is to focus on who, what, where, when, why and how we should consume these fluids. Be mindful that a *glass of water a day keeps the Urologist away!*

WHO Should Hydrate?

All adults should aim to drink between 2 and 3 L of water per day. Individuals with a history of stones are encouraged to maintain their fluid intake, as hydration is the mainstay of all treatment programs aimed at stone prevention.

Dehydration is recognized as the most common cause of stones. Patients who are at greatest risk for dehydration are also encouraged to stay well hydrated. Individuals who live in hot climate conditions, work in warm environments and lose excess water due to medical conditions are also recommended to stay well hydrated.

WHAT Type of Fluid Should You Drink?

Water

Water is the best fluid and friend you can rely on for stone prevention. Though water is best, other fluids and foods may also help prevent kidney stones.

Broccoli	Peaches raw
Cabbage raw	Peas raw
Carrots raw	Pineapple raw
Cauliflower raw	Potatoes boil
Celery	Squash boiled
Cherries raw	Strawberries raw
Cucumbers raw	Tomatoes raw
Eggplant raw	Watercress raw
Fruit cocktail	Watermelon
Grapes	

Citrus Based Fluids (Lemonade and Orange Juice)

Consumption of citrus drinks, like lemonade, will offer you a dual advantage. Lemonade will provide you with the necessary volume for stone prevention. In addition, lemonade contains large amounts of citrate, a chemical necessary to inhibit stone formation. If you prefer to make your own lemonade, this is a simple approach. From fresh squeezed lemonade or lemon from concentrate, use one part lemon (i.e., 1 oz.), seven parts water (i.e., 7 oz.) and sweeten the combination (8 oz.) to taste. In summary, lemonade is a double bonus for stone prevention!

Orange juice is also a rich source of citrate. A recent study demonstrated that orange juice had a 12 % lower risk of developing kidney stones [1]. Unlike lemonade, however, orange juice is also a good source of vitamin C which can be metabolized the oxalate. This may negate the beneficial effects of orange juice. Consult with your dietician and urologist to see which fluids are the best source for your stone prevention.

Food

If you want an alternative to liquid drinks, there are several types of foods that contain a high fluid content [2]. These foods include:

Apples	Lettuce head
Apricots	Onions
Bananas	Oranges
Bean sprouts	Papaya raw

There may be reasons why these food choices may or may not be adequate for your stone prevention. While you may be getting the extra volume, these foods may have other nutrients that may favor stone formation. For example, if your 24 hour urine test demonstrates low urine volume and low urinary oxalate, then grapes may be a good choice of foods with extra fluid content. On the other hand, the high oxalate content in grapes may enhance their stone formation and, therefore, may not be a good choice. Remember, not all foods are right and not all foods are wrong for each individual. You need to individualize the treatment options.

Alcohol

Alcohol is a fluid that has a diuretic affect allowing the kidneys to produce more urine. This may be helpful in preventing stones. Individuals who drink a beer a day had a 41 % lower chance of developing kidney stones while wine drinkers had a 33 % lower chance of developing stones [1]. Other studies demonstrated that for each 8 oz. serving of wine decreased the risk for stone formation by 39–59 % [3, 4].

The potent diuretic effects of excess alcohol can lead towards potential dehydration. Dehydration produces more concentrated urine, and increases the risk of stones if adequate consumption of other fluids is not maintained. In addition, certain alcoholic beverages, especially beer and grain alcohol are high in purines that may increase the risk of stones, particularly uric acid stones.

Coffee and Tea

The association of coffee and tea intake with risk for stone formation appears to be less controversial, with findings of an inverse association with risk [1, 3, 4]. A recent study indicated that caffeinated Coffee leads to a 26 % lower risk, decaffeinated coffee leads to a 16 % lower risk and tea an 11 % lower risk of stone formation [1].

Coffee and tea are sources of caffeine and increased diuresis. Excessive consumptions of these liquids can lead towards dehydration and may increase the risk of stone formation.

Soda

Although further research is required, evidence indicates that cola consumption significantly increases urinary calcium [5] and oxalate excretion [6]. Patients who avoid colas and other phosphoric acid-containing beverages have been found to have a 15 % lower rate of stone recurrence than those who continue to consume these beverages [7]. A recent study comparing sugar-sweetened soda and artificially sweetened soda demonstrated a 23 % higher risk and a trend to lower risk, respectively, suggesting the higher fructose content in the cola, which is known to increase urinary calcium, oxalate and uric acid, will increase the risk of stones [1].

Soda should not be the major source of your fluid intake to achieve your daily goal for hydration. Soda may provide you with appropriate volume, but it also has some negative effects. Caffeinated sodas act as a diuretic and can reduce your ability to achieve an adequate balance of fluid. In addition, soda also contains phosphoric acid. This can reduce the amount of citrate in your urine, which is necessary to prevent new stone formation. The more citrate you lose in your urine, the greater risk you have for generating new stones.

In summary, the key to avoiding kidney stones is moderation of your hydration. The occasional glass of wine, beer, soda, coffee or tea won't

mean you are going to start producing stones, but you should balance your alcohol intake with plenty of hydrating substances like water, decaffeinated tea and diluted juices. *Remember, not all fluids are the same!*

Are There Circumstances WHERE Consumption of More Fluids Is Required?

The amount of fluids you drink is not always equal to the volume of urine eliminated. Some fluids may be eliminated by sweat, loose bowel movements and even by breathing. This is what we refer to as *indispensable* water losses. Therefore, when we drink, we need to compensate for these losses. Previously, we stated that you want to produce at least 2 l of urine per/day. Even as a “couch potato”, with minimal energy expenditure, additional fluids are required to compensate for these losses.

There are multiple factors that determine an individual's requirement for fluids. When a patient exercises, the sweat that is produced is fluid that would have normally been eliminated as urine. Therefore, this fluid loss needs to be replaced. Likewise, patients with irritable bowel, such as Ulcerative Colitis, Crohn's Disease, Diverticulitis; and surgical treatment of the gastrointestinal tract, such as bowel resection, gastric bypass, can result in significant water losses. These patients need to replenish these losses too. Finally, if you live or work in a hot and dry environment or in the summer months spending time outside when you perspire, additional and adequate fluid consumption is paramount (see Chap. 9 for additional information on risk factors of stones).

There are some job related hazards that may contribute to kidney stone formation. Individuals who work in drycleaners, as carpenters or as roofers may be at risk for stones when working in dry, hot environments. Individuals who were frequent fliers including pilots, flight attendants, and business travelers are at great risk for stones when flying at high altitudes. In addition, people who consume larger quantities of salt should

increase their fluid hydration. Salt consumption increases the risk of water retention and additional fluids are required to flush the salt from our bodies.

Overactive bladder, or OAB, affects approximately one-third of individuals over the age of 45. This condition is associated with urinary urgency, urinary frequency, nocturia, and possible urge incontinence. Individuals with OAB tend to reduce their fluid intake to compensate for their irritative voiding symptoms. For individuals with overactive bladders who are also stone formers, this becomes a Catch-22. In one sense, patients need to drink plenty of fluids to avoid stones; on the other hand, individuals with OAB reduce their fluid intake to prevent these irritative voiding symptoms. Dehydration will put this same patient at an increased risk for stone formation. This patient has two separate medical conditions that need to be managed individually. It is paramount that these stone patients remain adequately hydrated for their stone prevention. The OAB symptoms are treated by biofeedback with or without medication, including anti-cholinergic and beta agonists to relax their bladder.

It is important to remember not to wait until you're thirsty to satisfy your sense of thirst. It is important to be proactive and to maintain good hydration and not wait until you are thirsty. Once you are thirsty you are probably already dehydrated. *Remember, don't wait to hydrate!*

WHEN Should You Drink Fluids?

Pace yourself. Drinking fluids throughout the day is like running a race. Don't burst out and drink as fast as you can in the first hour. Pace yourself as the day goes along. If the goal is to drink 8–12, 8 oz. glasses of water/day, spread it out throughout the day. Most individuals are awake 16 hours per day. Therefore, consuming 1 glass of water every 2 hours will allow you to achieve that goal.

Can I drink at night? The short answer is “yes”, however, you may be exhausted the next day from lack of sleep after being in the bathroom all night long! The goal to hydration is not to consume your daily load of fluid within a short

period of time, but to spread your drinking throughout the day. Like running a marathon, it is important to pace yourself with your fluids. Likewise, hydration is not to be done Monday through Friday and take a holiday from drinking on weekends. *Remember, stone prevention is a 24–7 job!*

WHY Do We Hydrate?

There are two goals to hydration. A sufficient amount of fluid is necessary to reduce the concentration of chemicals in the urine involved in stone formation. An adequate amount of hydration is quintessential (fundamental) for stone prevention. In addition, for a patient who presents with a stone, hydration is essential to help pass an existing stone located in the urinary tract. *Remember, you need fluid intake to make your stone evacuate!*

HOW to Manage Your Drinking to Prevent Future Stones?

We all contain the chemicals in our urine that will allow us to make kidney stones. When the urine is more concentrated, there is a better opportunity for these particles of calcium, oxalate, uric acid and/or phosphate, for example, to unite, called *crystallization*, and ultimately, form a stone. *When* the urine is diluted from drinking large amounts of fluids, there is a smaller likelihood that these particles will bond together to form a kidney stone.

There are two basic ways that you calibrate your fluid consumption to estimate that you are consuming the right amount of fluids. Think of colors and numbers!

Urine by the colors: Earlier, we discussed that a 24 hour urine will verify that fluid consumption is adequate to minimize risk of future stones (See Chap. 24). It is critical for patients to maintain adequate hydration to maintain 2 L of urine per day. I tell my patients, however, that we do not want to be doing daily 24 hour urine tests

to verify that they are drinking an adequate amount of fluids. A “poor man’s 24 hour urine”, as I explain, is looking at the color of your urine. As long as the color of the urine is as clear as water, the patient is adequately hydrated. On the other hand, if the urine is yellow, amber or dark, signifying a concentrated urine, then they need to increase their fluid consumption. An exception to this, for example, is if a patient takes vitamins in the morning, this may result in concentrated urine. This is especially true for individuals consuming water soluble vitamins, such as Vitamin B and Vitamin C, which are excreted in the urine. In this case, a patient is encouraged to evaluate their afternoon voided urine specimens. In summary, a patient should use the color of their last voided urine as an indicator for what they will do for the next several hours before their subsequent void.

Fluid by the numbers: In my practice, I encourage my patients to drink as much fluids as they can. The goal to hydration, is for patients to drink 8–12, 8 oz. glasses of water per day. When I have a 51-year-old female in my office that drinks two cups of coffee at breakfast, one glass of soda at lunchtime and a glass of wine at dinner, she is certainly at risk for dehydration and potential stone formation. I attempt not to change 51-year-old eating and drinking habits, however, I try to work with this patient to compensate for their drinking habits. While it is certainly appropriate to minimize the fluids that increase the risk for stones, I try to manage their expectations by working with their drinking habits. Therefore for every patient who drinks a fluid associated with a diuretic property, i.e. caffeinated soda, alcohol, coffee, or tea, I assign this drink a score of “-1”. In the example above, this patient had a score of “-4” for the day. Therefore, I would encourage her to drink four additional glasses of water per day added to her base of 8–12 glasses of water maintenance. In this case, this person should drink between 12 and 16 glasses of water per day. The goal of therapy here is to manage their hydration and to work with their current drinking habits. In summary, for each “minus one drink” a person consumes, the patient should chase those fluids with an equal volume of water.

What about the decaffeinated beverages? Decaffeinated coffee or tea is a misnomer. It does not mean that this drink is caffeine free, it simply means less caffeine. Therefore, in making an allowance for cups of decaffeinated tea or coffee a patient consumes, I usually assign a score of “-0.5” for these beverages. Therefore, if a patient had two cups of decaffeinated coffee per day, this individual should chase these drinks with one extra glass of water to their daily water maintenance. Remember, *good hydration for the right migration!*

HOW Much Fluid Should You Drink to Prevent Kidney Stone Formation?

Individuals with a history of kidney stones should drink enough water and other fluids to produce at least 2 l of urine per day. People who are cystine stone formers may need to drink even more.

A 24 hour urine collection may be used to determine the volume of urine produced during a single full day. If the volume of urine produced is too low, the person can be advised to increase their fluid intake. Drinking enough fluids is the most important factor a person can achieve to prevent most types of kidney stones. Overall fluid consumption appears to be most important. People who consume the highest amount of fluids each day (~2.6 L) have a 30–40 % lower risk for stone formation than those individuals consuming the least amount (~1.4 L) of fluids [3].

Remember, the right volume of fluids is necessary to help achieve the right “exit” strategy for your stone!

Summary

In summary, hydration is a critical factor in stone management. Water to the kidneys is like oil to the car engine. Water is necessary to allow the kidneys to filter and manage the water balance in our bodies. In addition, fluid intake is paramount not only in helping stones to pass, but in stone prevention. *Remember, you are what you drink!*

The pain was intense and beyond my imagination! They hydrated me well with IV fluid in the ER. That and the pain medication gave me some relief. My Urologist and his team of residents encouraged me to go home and hydrate. They gave me the confidence that I would pass that 3 mm stone located in my ureter. So, I went home, and had a beer. Thirty minutes later, that pebble shot into the toilet the same way I threw rocks into the pond when I was a kid. Only the splash was not as great! I will always recall that you have to hydrate to make it migrate!

References

1. Ferraro PM, Taylor EN, Gambaro G, Curhan GC. Soda and other beverages and the risk of kidney stones. *Clin J Am Soc Nephrol.* 2013;8:1–7.
2. From Litholink©: Your Guide to Increasing Fluid Intake. A Litholink patient resource guide, 2005.
3. Curhan GC, Willett WC, Speizer FE, et al. Beverage use and risk for kidney stones in women. *Ann Intern Med.* 1998;128:534–40.
4. Curhan GC, Willett WC, Rimm EB, et al. Prospective study of beverage use and the risk of kidney stones. *Am J Epidemiol.* 1996;143:240–7.
5. Iguchi M, Umekawa T, Takamura C, et al. Glucose metabolism in renal stone patients. *Urol Int.* 1993;51:185–90.
6. Rodgers A. Effect of cola consumption on urinary biochemical and physicochemical risk factors associated with calcium oxalate urolithiasis. *Urol Res.* 1999;27:77–81.
7. Shuster J, Jenkins A, Logan C, et al. Soft drink consumption and urinary stone recurrence: a randomized prevention trial. *J Clin Epidemiol.* 1992;45:911–6.

Tiffany Graham

Summary Stone Facts

- Diet and certain nutrients play a role in decreasing kidney stone formation.
- Magnesium, potassium, citrate, and vitamin B₆ have shown the most benefit.
- Not all dietary supplements are the same, so choose wisely.
- Look for supplements with the NSF® or USP® mark.

Introduction

Interest in complimentary health approaches has increased significantly over the last few decades. Complimentary medicine includes natural products (dietary supplements such as vitamins, minerals, herbals and botanicals) as well as various mind and body practices (acupuncture, chiropractic manipulation, massage, tai chi, yoga, and other therapies). This chapter will focus on dietary supplements, including vitamins, minerals, and other nutrients that, along with a healthful diet and adequate fluid intake, may help to decrease the risk of kidney stones in susceptible individuals.

Kidney stones can form when substances (such as calcium, oxalate, and phosphorus)

become too concentrated in the urine. Diet and certain nutrients play a role in decreasing the risk of kidney stone formation by inhibiting the crystallization of these compounds. The nutrients that have shown the most promise are magnesium, potassium, citrate, and vitamin B₆.

Magnesium

Magnesium is an essential mineral, meaning that it is not produced by the body but instead has to be consumed through food or supplements. Magnesium is involved in many reactions and pathways in the body, such as energy production, protein synthesis, muscle and nerve function, blood glucose control, and blood pressure regulation. Magnesium deficiency has been linked to an increased risk of heart disease, osteoporosis, high blood pressure, and type 2 diabetes.

Research indicates that magnesium decreases the risk of forming calcium oxalate kidney stones because it reduces oxalate absorption. Magnesium competes with calcium to bind with oxalate in the gut and the urine. Magnesium is therefore considered a “stone inhibitor” because it decreases the ability of calcium oxalate crystals to form into larger kidney stones.

Magnesium is naturally found in green leafy vegetables such as spinach, whole grains (bran and shredded wheat cereals, brown rice, whole wheat bread), beans, legumes, nuts, and seeds. While most magnesium rich foods are considered

T. Graham, MPH, RD, LD
Theralogix, Rockville, MD, USA
e-mail: mtknight@theralogix.com

healthful, increasing how much you eat of these foods may conflict with other recommendations. Foods that are good sources of magnesium are often rich in oxalate, which your doctor may have advised you to limit. For this reason, taking a magnesium supplement is a better option for most people that have a history of calcium oxalate kidney stones.

Magnesium is available in dietary supplements as magnesium oxide, citrate, chloride, or hydroxide, and is also found in some prescription medications. Even though magnesium is found in many foods, a large U.S. survey found that nearly 50 % of adults had inadequate magnesium intake, with adults over 50 consuming less magnesium than younger adults. These findings indicate that slight magnesium deficiency may be relatively common in the U.S.

Researchers are still trying to determine the best form of magnesium to take. Magnesium citrate is known to be well absorbed, and it has the added benefit of citrate (also discussed in this chapter), another stone inhibitor. Other forms of magnesium have also shown benefit. Both magnesium citrate and magnesium oxide have been shown to decrease oxalate absorption, increase the levels of magnesium and citrate in the urine, and decrease the saturation of calcium oxalate in the urine, which are all positive effects for someone who is prone to forming calcium oxalate stones. Both forms of magnesium worked best when taken with food.

Magnesium from food is well tolerated, but high-dose supplements (>400 mg) can cause stomach upset, nausea, diarrhea, and vomiting. It is important to always take magnesium supplements with food, stay within the recommended doses, and choose an extended release formulation to decrease the risk of these gastrointestinal symptoms.

Potassium

Potassium is an essential mineral and electrolyte that plays a role in many bodily functions including acid-base balance, nerve transmission, muscle contraction, and many others. The richest food

sources of potassium are fruits and vegetables. Potassium can also be found in dietary supplements in low doses (up to 99 mg per serving). Higher doses of potassium exist in the form of prescription medications (for more information, see Chap. 19). Potassium citrate is the form recommended for people who have had kidney stones.

Studies have found that men and women with high potassium diets are less likely to form kidney stones than those whose diets are low in potassium. Potassium citrate (from a supplement or prescription medication) increases levels of citrate in the urine and helps decrease the risk of calcium stone formation. High-dose potassium citrate is also beneficial for uric acid stone formers because it alkalinizes (increases the pH of) the urine, helping to decrease the recurrence of stones.

Potassium citrate is generally well tolerated at low doses, but higher doses may cause minor gastrointestinal side effects such as stomach upset, nausea, vomiting, and diarrhea. Potassium citrate should always be taken with food, and extended release formulations are best to decrease the risk of these gastrointestinal symptoms. Speak to your urologist about how much potassium citrate you should take.

Citric Acid (Citrate)

Citrate is an organic acid found in many fruits and vegetables. Citrus fruits (oranges, lemons, limes, and grapefruit) contain the most citrate. Lemon juice, or “lemonade therapy” has been used for people with kidney stones due to the citrate content of lemon, and the fluid content for hydration. Studies have found that drinking four ounces of lemon juice mixed into about eight cups of water throughout the day, providing just less than 6 g of citrate, increases urinary citrate levels and decreases urinary calcium levels in people with hypocitrauria (low urinary citrate levels) [1, 2].

Oranges and orange juice also contain citrate, along with potassium. Oranges contain more oxalate than lemons, however, so for those trying to limit their intake of oxalate, lemonade is a

better choice. Citrate can also be found in dietary supplements (as calcium citrate, magnesium citrate, or potassium citrate, for example) and prescription medications (as potassium citrate in Urocit[®]-K).

The main reason that citrate is helpful is because it increases the level of citrate in the urine, which is especially beneficial for people with low urinary citrate levels. Citrate is also thought to work by binding with calcium in the urine, thus reducing the amount of calcium available to bind with oxalate and form a kidney stone.

The recommended dosage of citrate varies and depends on the chemical form. The amount of actual citrate is usually not listed on a dietary supplement label. For example, if the supplement contains magnesium in the form of magnesium citrate, the dose listed is generally for the magnesium content, not the citrate. Speak to your urologist about what form and how much citrate you should take.

Vitamin B₆ (Pyridoxine)

Vitamin B₆ is a water-soluble vitamin that is involved in over 100 enzyme reactions in the body. It is involved in the metabolism of protein, and is important for immune and cognitive function.

Vitamin B₆ deficiency is associated with higher levels of oxalate in the body, as well as better absorption of oxalate. Vitamin B₆ decreases the body's production of oxalate, and therefore vitamin B₆ supplements are thought to help decrease the risk of forming calcium oxalate kidney stones. In a large study of women, those consuming more vitamin B₆ from foods were found to have a lower risk of forming kidney stones than those who took in low amounts of B₆ [3].

Other studies have shown that the combination of vitamin B₆ and magnesium increases urinary citrate and magnesium levels, and decreases urinary oxalate levels; these are all positive changes for a stone former [4]. The combination of potassium magnesium citrate has been shown to

increase urinary citrate levels and decrease stone recurrence rates [5].

Vitamin B₆ is naturally found in a variety of food, including fish, poultry, beef liver, chickpeas, potatoes and other starchy vegetables, and fruits (other than citrus fruits). Vitamin B₆ is also available as a dietary supplement, and small amounts are found in most multivitamins. A daily dose as low as 10–15 mg has shown benefit for stone formers. It is important to stay within the safe “upper limit” of 100 mg when taking a vitamin B₆ supplement. Higher doses taken for longer than a year may cause nerve damage (sensory neuropathy). This is reversible when the high-dose of B₆ is stopped. High amounts of vitamin B₆ from food have not been reported to have any adverse effects.

Other Nutrients

There are other nutrients and natural ingredients that could potentially decrease the risk of kidney stone formation, but more research needs to be done to recommend them specifically for that purpose. For example, studies indicate that the omega-3 fats found in fish oil decrease oxalate levels in the urine and may decrease the risk of calcium oxalate crystallization and thus risk of calcium stone formation. Probiotics are also of interest because by changing the bacteria in the gastrointestinal tract they have the potential to lower oxalate absorption. Although more research is needed before omega-3 fish oil and probiotics can be recommended for stone prevention, these supplements have many other health benefits and are generally safe to take. Pumpkin seeds and rose hips are two other ingredients that may favorably change the chemistry of the urine, although they have not been studied extensively in stone patients.

Dietary Supplement Safety

More than half of adults in the U.S. report using dietary supplements, but at least a third of them do not inform their physician about what they are taking. Many people assume that because a

supplement is “natural,” it must be safe. Because of this, or because they may think a doctor will not understand or will disapprove, many people do not discuss dietary supplements with their doctors. This increases the risk for interactions with medications, other supplements, or problems with certain health conditions.

Not all supplements are safe. Although supplement companies must follow Good Manufacturing Practices (GMPs) to control the basic quality of their products, the Food and Drug Administration (FDA) has limited ability to assure compliance, and the safety and effectiveness of products and individual ingredients are not reviewed by FDA. It is therefore important to make sure that the ingredients in the products you take are supported by scientific research, the content and purity of the final product is tested by an independent certification program (look for the NSF[®] International or USP[®] seal on the bottle’s label), and the supplement is appropriate for you and your health.

It is important to discuss all the dietary supplements you take with each of your doctors. There are certain supplement ingredients that you may need to be cautious about if you have had kidney stones, such as:

Aloe Vera- The overuse of aloe may decrease your potassium levels, especially if you already take a diuretic medication that causes you to lose potassium. Aloe has also been linked to kidney failure and may worsen kidney disorders.

Calcium is generally taken for bone health. Calcium-rich foods, such as low-fat milk and yogurt, are not a problem for calcium kidney stone formers. Healthful diets rich in calcium-containing foods such as low-fat dairy products actually protect against stone formation because the calcium binds to dietary oxalate, reducing oxalate levels in the urine. Taking high doses of some calcium supplements, however, is not recommended. If your health-care provider has recommended that you take a calcium supplement for bone health or other reasons, choose one with calcium citrate instead of calcium carbonate. Because citrate helps inhibit stone formation, this form of calcium is the preferred choice for individuals who form kidney stones.

Cinnamon is generally used to help control diabetes, although the evidence of effectiveness is mixed. Large doses may increase liver enzymes, and it is important to stop taking cinnamon before having surgery.

Cranberry is generally taken to decrease the risk of urinary tract infections. Although it is effective for many people, there is concern that cranberry juice and some cranberry supplements may increase the risk of kidney stones because of their oxalate content. Large amounts of cranberry juice are not recommended. Some supplements contain oxalate, and others do not, so consult with your urologist before taking a cranberry supplement.

Turmeric is generally taken because of its anti-inflammatory effects. It is usually well-tolerated but can have some gastrointestinal effects, may worsen acid reflux conditions, and should not be taken by someone with gallbladder disease.

Vitamin C (ascorbic acid) is safe to take in low doses, such as the amount typically found in a daily multivitamin. Higher doses (>500 mg), however, may increase the risk of kidney stones by increasing oxalate levels, so avoid high-dose vitamin C supplements unless otherwise recommended by your doctor.

While these and other supplements may be recommended to you, be cautious. If you have kidney stones, stop taking supplements if your oxalate level remains high and other dietary measures to reduce oxalate have not been successful. If your 24 hour urine demonstrates consistently high oxalate and no other sources can be identified, take a closer look at the supplements you are taking, and discuss them with your urologist.

Conclusion

Dietary supplements, as well as the amounts of certain nutrients in your diet, can impact your risk of kidney stone formation and recurrence. It is important to choose your supplements carefully, however, since some nutrients can inhibit kidney stones, while other nutrients can promote

them. Also, be sure to choose high-quality dietary supplements that have undergone independent testing and certification (through NSF® or USP®), so that you know what you are getting. Speak to your urologist or other health care provider about the best product options for you.

Mr. CM is a 47 year old male with a history of ulcerative colitis and resulting ileostomy. His first bout of kidney stones was in 1998. Stone analysis revealed uric acid stones, and his doctor prescribed the medication allopurinol, which Mr. CM began taking. He continued to suffer with multiple bouts of kidney stones over the next 10 years. In 2008, Mr. CM had a CT scan, which showed a large uric acid stone, and revealed a cancerous kidney tumor. After the cancer was treated, he had lithotripsy to blast the stone. In 2009, Mr. CM's urologist recommended that he start TheraLith XR, a nutritional supplement containing

magnesium, potassium, citrate, and vitamin B₆. He started taking TheraLith XR right away, and also continued taking allopurinol. Mr. CM has not had any further kidney stone "attacks" since starting TheraLith XR, and his 2013 CT scan was clear.

References

1. Seltzer MA, et al. Dietary manipulation with lemonade to treat hypocitraturic calcium nephrolithiasis. *J Urol.* 1996;156(3):907–9.
2. Penniston KL, et al. Lemonade therapy increases urinary citrate and urine volumes in patients with recurrent calcium oxalate stone formation. *Urology.* 2007; 70(5):856–60.
3. Curhan GC, et al. Intake of vitamins B6 and C and the risk of kidney stones in women. *J Am Soc Nephrol.* 1999;10(4):840–5.
4. Rattan V, et al. Effect of combined supplementation of magnesium oxide and pyridoxine in calcium oxalate stone formers. *Urol Res.* 1994;22(3):161–5.
5. Reddy SV, et al. Effect of potassium magnesium citrate and vitamin B-6 prophylaxis for recurrent and multiple calcium oxalate and phosphate urolithiasis. *Korean J Urol.* 2014;55(6):411–6.

Nutrition Recommendations to Prevent Kidney Stones: *Realistic Dietary Goals and Expectations!*

27

Kristina L. Penniston

Summary of Dietary Facts

- Diet is an important factor that can promote or inhibit kidney stone formation.
- Since kidney stones form for different reasons, including genetic factors, sometimes there is nothing in the diet that is causing stones.
- There is no “one-size-fits-all” approach to using diet to prevent kidney stones; dietary recommendations should be made individually to patients based on their specific kidney stone risk factors.
- A dietitian is an important part of the health-care team who can help a patient plan meals that lower the risk of forming stones based on the type of stone the person formed in the past.

ally some dietary influence that, if controlled, can reduce stone formation and recurrence.

This chapter will address dietary factors that contribute to kidney stone formation and strategies to change them. The strategies presented here are general; not all of them may apply to you, depending on your stone history, the type(s) of stones you form, in addition to other individual factors. It is very unlikely that you would need to implement all of the strategies addressed herein and are strongly encouraged to consult with a registered dietitian-nutritionist to help identify the dietary factors that will impact you most. The chapter outline is as follows: (I) General goals for nutrition therapy; (II) calcium oxalate stones; (III) calcium phosphate stones; (IV) uric acid stones; (V) cystine stones; and (VI) special medical situations.

Introduction

Diet can influence whether or not a person forms kidney stones, either in concert with or apart from inherited or genetic factors. Factors in the diet can influence whether crystals form and how large they potentially grow. Sometimes there is no dietary cause for kidney stones. In this case, there may be little one can do with their diet to control stone formation, There is, however, usu-

General Goals for Nutrition Therapy

First and foremost, your dietary risk factors for stones should be identified. This includes knowing what your kidney stones have been composed of, if possible, and what your 24 hour urinary risk factors are (See Chap. 24). A spot urine collection, similar to the one you get when you go to a doctor’s office for a check-up, is not sufficient for assessing your kidney stone risk factors. A 24 hour urine collection is the optimal way to identify whether your urine has an imbalance of stone promoters and inhibitors. Such an imbalance can sometimes be controlled by specific dietary

K.L. Penniston, PhD, RD
Department of Clinical Nutrition Services,
University of Wisconsin Hospital and Clinics,
Madison, WI, USA
e-mail: kpenniston@uwhealth.org

changes; in other cases, medication may be required.

The second part of determining your dietary risk factors involves linking the findings from the stone composition analysis and the 24 hour urine analysis to your diet. A registered dietitian can obtain a complete diet assessment from you, which involves documenting how you usually eat. The key to a good diet assessment is being as inclusive as possible about all the things you eat and drink, how much, and how often. Your intake of over-the-counter supplements is also part of the assessment. As with many of our daily habits, our diet is not the same on a day-to-day basis. There are usually patterns that can be identified, and these are helpful in making the assessment of whether or not your diet is contributory in any way to your kidney stones. From your assessment, which may be obtained verbally in an interview with you or by you documenting what you eat over a period of several days, connections can be made with specific dietary influences and your individual stone history. Be mindful as well that there may be no dietary contributor to your stones. If there is dietary factor, a good diet assessment can be the means by which this connection can be determined.

Urine Volume

See Chap. 25.

Calcium Oxalate Stones

If your diet is determined to be contributory to calcium oxalate stone formation, it could be for one or a combination of the following reasons:

- High urinary calcium excretion
- High urinary oxalate excretion
- Low urinary citrate excretion
- Low urinary magnesium excretion
- Low urine volume

High Urinary Calcium Excretion

While everyone normally excretes some calcium in their urine, usually between 100 and 150 mg

per day, reasons for excessive urinary excretion of calcium varies. The most common contributors to high urinary calcium excretion include sodium intake and the relative balance of foods providing acid ash vs. those providing alkaline ash. When sodium is consumed in an amount higher than the body's requirements, the kidneys response is to excrete the excess, however, when excess sodium is excreted in urine, calcium travels along with it, leaving less calcium available for body stores and for bone. The human body only needs between 500 and 1,500 mg of sodium daily, depending on individual and environmental conditions. However, in most developed countries, where the prevalence of convenience foods, processed foods, packaged foods, and salty snacks is high, individuals typically consume 4,000 or more mg of sodium daily. If urinary calcium excretion is high, and if a high intake of sodium is deemed to be contributory, then reducing sodium intake may help to decrease the amount of calcium in the urine. In fact, a reduction of sodium intake of 100 meq/day may reduce urinary calcium by 50 mg/day, depending on the individual and on other dietary and metabolic factors. If urinary calcium excretion is not high, or if it is high for some other reason, then reducing your sodium intake may be wise for other reasons but may not influence your calcium oxalate stone risk. Table 27.1 lists some of the foods most likely to contribute to high sodium intake. The Centers for Disease Control reported in 2012 that breads and rolls were the #1 contributor to sodium in the American diet. In its national nutrient database, the U.S. Department of Agriculture makes nutrient lists available online at <http://ndb.nal.usda.gov/>. Reports can be generated for the amounts of specific nutrients, such as sodium, in foods under the "nutrient lists" section.

In the case of the acid-base balance of the diet, a diet high in acid ash may cause calcium to be released from bone, which is then excreted in urine. Foods with an acid ash are those that have sulfur in their amino acid structure, specifically from the amino acids methionine and cysteine. These amino acids are mostly found in foods of animal and marine origin but are also richly expressed in some plant foods. While these amino acids are essential – needed for growth

Table 27.1 Foods and food groups with especially high sodium concentration

Food item	Measure	Sodium per measure (mg)
Table salt	1 teaspoon	2,325
Salted fish (cod, mackerel), canned anchovy	1 oz	1,030–1,970
Pizza, frozen entrees/meals	1 slice, 1 meal	300–1,660
Soy sauce, fish sauce	1 tablespoon	1,000–1,415
Pretzel twists	10 twists	1,000
Cold cuts, luncheon meats	2 oz	600–1,000
Canned vegetables, bottled/canned tomato sauce	½ cup	500–960
Cured meats (ham, sausage, kielbasa, hot dog)	2 oz	400–800
Salted pumpkin seeds	1 oz	710
Bacon, cooked	2 slices	600
Breads, rolls, bagels, muffins	1 slice	120–500
Condiments (ketchup, barbecue sauce)	1 tablespoon	190–450
Cheeses (American process, Blue, cottage)	1 oz	265–420
Certain ready-to-consume breakfast cereals	¾ to 1 cup	250–350
Salad dressings	1 tablespoon	120–240
Cheeses (Swiss, parmesan, Monterey Jack, ricotta, mozzarella, cheddar, brie)	1 oz	55–180

Information compiled from the USDA nutrient database. Available at <http://ndb.nal.usda.gov/ndb/nutrients/index>. Accessed 4 July 2014
 Restaurant food items are not included in the table

and maintenance of health – if not balanced with a sufficient amount of foods with an alkaline ash, the diet becomes high in acid load. If urinary calcium excretion is high, and if a diet with a high acid load is deemed to be contributory, then reducing the acid load of your diet may help to decrease the amount of calcium in your urine. If urinary calcium excretion is not high, or if it is high for some other reason, then reducing the

Table 27.2 Food groups and their average potential renal acid load (PRAL)

Food group	Serving size used for PRAL calculation	Average calculated PRAL/serving (mEq)
Meat, poultry	About 4 oz	9.5
Fish	About 4 oz	7.9
Flour, from various grains	1 cup, dry	7.0
Spaghetti, noodles	About ¾ cup, cooked	6.7
Cheeses with high protein content ^a	1 oz	5.9
Breads, bagels, muffins, rolls	About ¾ ounce	3.5
Cheeses with lower protein content ^b	1 oz	2.0
Yogurt, milk	1 cup	1.0–2.4
Vegetables	About ½ cup	-2.8
Fruits	1 piece or about ¾ cup	-3.1

Data adapted to table from Remer T, Manz F. Potential renal acid load of foods and its influence on urine pH. *J Am Diet Assoc.* 1995;95:791–7. Note that the PRAL values for individual foods within groups vary greatly; values in the table are averages per group
 The higher (more positive) the number for PRAL, the higher its acid load; negative values represent alkaline potential

^aExamples of high-protein cheeses, providing 7–10 g protein per 1 oz serving, include: parmesan, Edam, Romano, cheddar, mozzarella (part skim milk), Swiss, Colby
^bLower-protein cheeses include: blue, camembert, feta, cottage cheese, cream cheese

acid load of your diet may not influence your calcium oxalate stone risk. Table 27.2 lists some of the foods most likely to contribute to high dietary acid load if consumed in excess and/or without a sufficient amount of foods to counter the acid load, i.e., those that confer an alkaline load (Table 27.2).

Another potential contributor to high urinary calcium excretion, though less common, is excessive calcium intake, usually from calcium supplements. Calcium intake, even in individuals with premature bone loss, is usually not required to be more than 1,500 mg per day. Unfortunately, many over-the-counter calcium supplements are formulated to provide a high amount of calcium,

including 1,000 and 2,000 mg per day. A person who takes this much calcium from a supplement is very likely to have a high calcium intake as their diet, even without eating dairy foods, also provides some calcium. Too much calcium in the diet and/or from supplements may increase the amount of calcium in the urine, contributing to calcium oxalate stone disease. Adults typically need between 1,000 and 1,500 mg of calcium daily. A registered dietitian may be helpful in identifying how much calcium you usually get from your diet – from foods and beverages – and then the dietician can suggest the appropriate amount of supplemental calcium to take, if needed. Ideally, calcium intake should be from foods and beverages alone. Vegetarians who do not use dairy and people with lactose-intolerance can very easily achieve their ideal calcium intake from foods and beverages alone, as many calcium-fortified products are available. Additionally, there are some foods that are naturally rich in calcium that are non-dairy. Table 27.3 lists foods that are rich in calcium.

High Urinary Oxalate Excretion

It is estimated that only about 20 % of people who form calcium oxalate stones have high urinary oxalate excretion (hyperoxaluria). In other words, for the vast majority of calcium oxalate stone formers, high urinary oxalate excretion is not the cause. Yet, many people with calcium oxalate stones have been advised to avoid long lists of healthy foods, purportedly because of their oxalate content. The truth about high urinary oxalate excretion has more to do with factors other than oxalate intake. There are two sources of urine oxalate: endogenous, referring to oxalate that the human body makes; and exogenous, referring to oxalate that is absorbed from the digestive tract. The relative contribution of these sources to urine oxalate varies greatly between individuals for a variety of reasons both genetic and nutritional. Individuals with an overly high production of oxalate are usually those with an underlying disorder having nothing to do with their diet. Primary hyperoxaluria, a genetic disorder that results in extremely high

Table 27.3 Foods rich in calcium^{a, b}

Food item	Measure	Calcium per measure (mg)
Calcium-fortified non-dairy milks (soy, rice, coconut, almond)	8 fluid ounces	350–450
Tofu, prepared with calcium sulfate	½ cup	430
Calcium-fortified orange juice	8 fluid ounces	350
Buttermilk	8 fluid ounces	350
Yogurt (brands vary greatly)	6 oz	200–350
Eggnog	8 fluid ounces	330
Mustard spinach, raw, chopped	1 cup	315
Calcium-fortified vegetable juice	8 fluid ounces	300
Milk	8 fluid ounces	300
Sesame seeds, whole, roasted	1 oz	280
Almonds, whole	½ cup	190
Sesame seeds, whole, roasted	1 oz	280
Collards, chopped, cooked	1 cup	270
Spinach, turnip greens, cooked	1 cup	250
Salmon, canned, with bones	3 oz	240
Figs, dried	1 cup	240

^aInformation compiled from the USDA nutrient database, available at <http://ndb.nal.usda.gov/ndb/nutrients/index> (accessed July 4, 2014)

^bMost of the foods in the table are relatively low in sodium, making these calcium choices good for patients who need to maintain a low intake of sodium. But some of the items, such as vegetable juice, nuts and seeds, and canned fish, may have higher sodium content and should therefore be eaten in moderation or exchanged for no- or low-sodium formulations

oxalate production in the body, will thus not be addressed in this chapter (see section “[Special medical situations](#)” for a brief discussion of this).

How Does Oxalate Get into the Digestive Tract?

Oxalate is produced by many plants and is also picked up by plants from the soil. Most all plant foods contain some oxalate; some more than

others. When we eat these plants, we eat their oxalate. It is difficult, if not impossible, to follow recommendations for a healthy, plant-based diet without consuming some oxalate. In fact, oxalate restriction is controversial as (a) oxalate restriction also requires a calcium restriction in order to maintain a suitably low oxalate-to-calcium ratio in the urine; (b) high-oxalate foods are frequently those that provide stone inhibitors, such as fiber, magnesium, and phytate; and (c) high-oxalate foods are mostly fruits, vegetables, and whole grains, which are recommended in high amounts for cancer prevention, cardiovascular disease prevention, and overall good health. It is worth noting that vegetarians, whose oxalate intake can be as much as three times higher than the typical omnivore's, appear to have a lower incidence of calcium oxalate kidney stones (See Chap. 28).

What Happens to Oxalate in the Digestive Tract?

Oxalate in the digestive tract has three possible fates: (1) it can either be complexed with calcium or another mineral and excreted harmlessly in the stool; (2) it can be eaten (degraded and destroyed) by bacteria that live in the human digestive tract; or (3) it can be absorbed into the bloodstream. Once absorbed, oxalate must be excreted into the urine by the kidneys, as there is no use for oxalate by humans.

A low or sub-optimal calcium intake is a major contributor for high oxalate absorption in the digestive tract. When there is too little calcium within the digestive tract, especially to match with the amount of oxalate in the digestive tract, oxalate is freely absorbed. This knowledge has led to cessation of calcium restrictions in people with high urinary oxalate, instead focusing on normalizing calcium intake. Most recommendations to control high urinary oxalate excretion are to include calcium-containing foods or beverages at each meal in order to provide sufficient calcium in the digestive tract to bind any oxalate that has come from the meal. Magnesium is also capable of complexing oxalate in the digestive tract and thereby reducing its absorption, but our need for

magnesium is about three-fold lower than for calcium. Thus, the use of magnesium as a therapy for binding oxalate is not as clinically relevant as the use of calcium. Usually, foods and beverages providing about 300 mg of calcium at each meal will be sufficient. People with certain bowel disorders, such as malabsorption, may need supra-physiologic doses of calcium at meals to bind oxalate and prevent its absorption. Table 27.3 lists foods and beverages rich in calcium.

A low or sub-optimal amount of healthy bacteria in the digestive tract, commonly referred to as the "gut microbiome," may contribute to high oxalate absorption from the digestive tract and, thus, high urinary oxalate excretion. This is an area of current research, so evidence-based information to guide therapy is not sufficient. However, studies show that diets low in fruits and vegetables, which provide food (known as prebiotics) for good bacteria (known as probiotics), lead to a different profile of the gut microbiome. This could potentially lead to a reduced number of bacteria that can degrade oxalate and remove it from the digestive tract, thereby reducing its absorption. Aside from not eating enough prebiotic material to grow and sustain a healthy gut microbiome, antibiotic therapy may also alter the gut bacterial profile. Research in this area is increasing, and there is a strong potential for evidence to guide clinical therapy for individuals suspected to have low or sub-optimal bacterial oxalate degradation capacity in their digestive tracts.

Finally, certain amino acids we eat, such as from animal flesh, can provide ingredients (precursors) in the body to form oxalate. Another way people get oxalate is from high doses of vitamin C (ascorbic acid), which, when consumed in amounts greater than the body can use, is broken down or metabolized to oxalate. There is some thought, too, that fructose in high amounts – such as from drinking a lot of soda or fruit juice or from eating foods processed with high amounts of fructose – can contribute to the formation of oxalate in the body. Once oxalate is formed in the body, by whatever means, it is excreted into urine by the kidneys where it can bind with calcium to form stones. Table 27.4 lists some of the foods highest in oxalate per portion.

Table 27.4 Foods high in oxalate per serving

Food item	Measure	Oxalate per measure (mg)
Spinach	1 C if raw; ½ C if cooked	340–755
Rhubarb, fresh	½ C	200–540
Almonds	1 oz. or about 22 kernels	120–130
Potato, baked, with skin	1 medium	50–100
Miso soup	1 C	40–90
Bulgur, cooked	1 C	25–85
Beets, raw	½ C	25–85
Navy beans, canned	½ C	50–75
Cashews	1 oz. or about 18 kernels	50–75
Cocoa powder	4 teaspoons	20–70
Hot chocolate, homemade	1 C	30–65
Bran flakes cereal, with raisins	1 C	50–60
Okra, cooked	½ C	30–60
French fries	4 oz.	40–50
Shredded wheat & bran cereal	1 C	30–50
Raspberries	1 C	20–50

The oxalate content of foods varies depending on the source of the information, which may be due to differences in analytical technologies and other factors. The two sources used to compile the ranges for oxalate content in the table were the USDA nutrient database, available at <http://ndb.nal.usda.gov/ndb/nutrients/index>. Accessed 16 July 2014, and the Harvard School of Public Health Nutrition Department file download site, available at <https://regepi.bwh.harvard.edu/health/nutrition.html>. Accessed 16 July, 2014

Oxalate restriction is controversial. Many of the foods highest in oxalate are also foods rich in antioxidants – such as lutein, beta-carotene, and zeaxanthin – magnesium, and fiber, all of which inhibit stone formation in various ways

Low Urinary Citrate Excretion

Citrate is normally excreted in the urine and combines with calcium to prevent it from binding with oxalate to form stones. The official cut-offs for low urinary citrate excretion (hypocitraturia) are typically 340 mg per day or less. Many experts agree that the target for urinary citrate excretion should be at least 600 mg per day, as this is the

average amount excreted by healthy non-stone formers. Low urine citrate can be caused by several non-dietary factors, including certain medications and some underlying conditions (such as renal tubular acidosis). It is doubtful that very low urine citrate is caused by a dietary “indiscretion.” Nonetheless, urinary citrate excretion can be increased with specific dietary measures, namely, maximizing the intake of fruits and vegetables (all kinds) and using beverages flavored with citric acid sources. Most evidence suggests that the increase in urinary citrate excretion from these measures may be maximized at about 200 mg per day. This may be sufficient in some cases to raise urine citrate to or near therapeutic levels. Pharmacologic therapy is usually required in the most severe cases of hypocitraturia. Natural sources of citric acid include citrus fruits and their juices, particularly lemons and limes. Citric acid is also used as a flavoring agent and as a preservative in some commercially-prepared beverages. Table 27.5 lists concentrated sources of citric acid. It should be noted that if the overall diet is deficient or low for potassium, a mineral for which the adult requirement is nearly 5,000 mg per day (making it the mineral with the highest requirement for humans), citric acid alone might not raise the urine citrate concentration. Mostly all fruits and vegetables, milk and yogurt, and some whole grains contain appreciable amounts of potassium. Many Americans fail to reach the recommended daily amount. Table 27.6 lists foods particularly rich in potassium. If using citric acid sources in an attempt to raise urine citrate, it is recommended to also consume an adequate amount of potassium daily.

Low Urinary Magnesium Excretion

Some magnesium is normally excreted in urine and combines with oxalate there to form a soluble complex. This means that it stays in liquid form instead of forming a crystal. It also means that there is less oxalate available to bind with calcium to form a stone. In other words, magnesium competes with calcium to bind oxalate. Urinary

Table 27.5 Citric acid content (mg) of some juices and prepared juice drinks

Juice	Form	Per liter	Per oz.	Calories per 8 oz. serving
Lemon juice	Fresh, squeezed from fruit	48.0	1.42	54
Lime juice	Fresh, squeezed from fruit	45.8	1.35	62
Lemon juice	From juice concentrate	36.6	1.08	51
Lime juice	From juice concentrate	35.4	1.05	52
Grapefruit juice	Ready-to-consume	25.0	0.74	96
Orange juice	Ready-to-consume	16.8	0.50	122
Orange juice	Fresh, squeezed from fruit	9.1	0.27	112
Lemonade, regular	Ready-to-consume	5.6	0.17	110
Lemonade, diet/light	Ready-to-consume	4.6	0.14	20
Lemonade, sugarfree	Made from dry mix	3.2	0.09	5

Data in table adapted from Penniston KL, Nakada SY, Holmes RP, Assimos DG. Quantitative assessment of citric acid in lemon juice, lime juice, and commercially-available fruit juice products. *J Endourol.* 2008;22:567–70

Calorie information from the USDA nutrient database. Available at <http://ndb.nal.usda.gov/ndb/nutrients/index>. Accessed 24 June 2014

Table 27.6 Foods rich in potassium^a

Food item	Measure	Potassium per measure (mg)
Tomato puree, sauce, juice, or paste (canned)	½ cup	530–1,330
Beans, cooked (white, soy, lima, pinto, kidney, navy, split pea, great northern, refried)	1 cup	690–1,180
Potato, baked, flesh and skin	1 medium	1,081
Plantain, raw	1 medium	893
Sweet potato, baked in skin	1 medium	694
Beet greens, cooked	1 cup	655
Cabbage, Chinese, cooked	1 cup	631
Fish, cooked (halibut, salmon, Pacific rockfish, haddock, cod, swordfish, tuna)	4 oz	530–610
Yogurt, various brands	8 oz	400–575
Papaya, raw	1 medium	553
Prune juice, carrot juice, canned	6 oz	515–530
Beets, cooked	1 cup	519
Orange juice, raw or chilled ready-to-consume	8 oz	450–495
Vegetables, cooked (Brussels sprouts, squash, artichokes, broccoli)	1 cup	450–495
Banana, raw	1 medium	422

^aInformation compiled from the USDA nutrient database, available at <http://ndb.nal.usda.gov/ndb/nutrients/index> (accessed June 9, 2014)

magnesium excretion could be low because of a magnesium deficiency or low magnesium intake or because of frequent diarrhea and/or vomiting. Adults need between 310 and 420 mg of magnesium per day, depending on gender and age. Studies show that magnesium intake in the U.S. is lower, on average, than recommended. Magnesium is found to varying degrees in many plant foods. Spinach, almonds, cashews, peanuts, shredded wheat cereal, soymilk, black beans, and edamame all provide 50 mg or more of magnesium per typical household serving. It is notable that many of these foods are also purportedly ample for oxalate, putting into question the efficacy of widespread oxalate restriction. In cases of magnesium deficiency or very low urinary magnesium excretion, supplemental sources of magnesium, available over-the-counter, may be advised. Table 27.7 lists some foods high in magnesium.

Calcium Phosphate Stones

If diet is determined to be contributory to calcium phosphate stone formation, it could be for one or a combination of any of the following reasons:

- High urinary calcium excretion
- Low urinary citrate excretion
- High urine pH
- Low urine volume

Table 27.7 Foods rich in magnesium

Food item	Measure	Magnesium per measure (mg)
Beans, raw (mungo, soy, yellow, lima, small white, navy, pinto, great northern, black)	1 C	330–550
Pumpkin seeds, dried	½ C	382
Quinoa, uncooked	1 C	335
Oats	1 C	276
Brown rice, raw	1 C	260–270
Brazil nuts, dried, whole	½ C	250
Barley, hulled	1 C	245
Spelt, uncooked	1 C	237
Almonds, oil roasted, whole kernels	½ C	215
Molasses	¼ C	204
Wheat germ, crude	½ C	138
Sesame seed kernels, dried	¼ C	130
Cashews, dry roasted, whole	½ C	125
Peanuts, all types, dry roasted	½ C	125
Walnuts, black, dried, chopped	½ C	125
Seaweed, spirulina, dried	½ C	109

Information compiled from the USDA nutrient database. Available at <http://ndb.nal.usda.gov/ndb/nutrients/index>. Accessed 15 July 2014

Of these, high urinary calcium and low urinary citrate excretion may be influenced by diet. An overly high urine pH is not thought to be a problem caused by diet and is not addressed here.

High Urinary Calcium Excretion

Urinary calcium excretion can be high in the calcium phosphate stone former for the same reasons as in calcium oxalate stone formers. It is worth noting that calcium intake in the normal range, 1,000–1,500 mg per day, does not normally contribute to high urinary calcium excretion. To date, studies show that dietary calcium intake is significantly lower in brushite stone formers (a type of calcium phosphate stone) than in non-stone formers. High urinary calcium excretion should be strictly controlled in the calcium phosphate

stone former. If diet is indicated as contributing to high urinary calcium excretion, refer to the previous section for suggestions on reducing it.

Low Urinary Citrate Excretion

Urinary citrate excretion can be low in the calcium phosphate stone former for the same reasons as in calcium oxalate stone formers. If diet is indicated as contributing to low urinary citrate excretion, refer to the previous section for suggestions on increasing it.

It is worth noting that urinary phosphate excretion has not been associated with kidney stone formation in some studies. High urinary phosphate excretion, variably considered to be >1,100 mg per 24 hour urine collection, is frequently observed in calcium oxalate and uric acid stone formers; thus, its ability to predict or cause calcium phosphate stones is questionable. While most Americans easily obtain and even exceed the recommended daily requirement for phosphorus (700 mg for adults; 1,250 mg for adolescents), most Americans do not form calcium phosphate stones. Phosphorus is widely distributed in the food supply, with sources including most all foods except for fruits and vegetables. It is difficult to limit in the diet and may have more to do with overall excessive calorie intake than with an excessive intake of any particularly phosphorus-rich food(s). Many experts agree that urinary phosphate excretion may merit little attention except in those in whom renal phosphate wasting may be suspected, and this should be treated medically.

Uric Acid Stones

Some people who form uric acid stones do so because of a genetic or inherited problem. Certain diseases can also cause the formation of uric acid stones, and they include, leukemia, lymphoma, multiple myeloma, sickle cell disease, polycythemia vera, and in some conditions involving ineffective erythropoiesis (red blood cell formation). Additionally, tumor lysis

syndrome caused by chemotherapy can result in high uric acid production and concomitant high urinary uric acid excretion. Dietary changes in the scenarios above are not likely to reduce stone recurrence. However, if diet is determined to be contributory to uric acid stone formation, it could be for one or a combination of the following reasons:

- High urinary uric acid excretion
- Low (acid) urine pH
- Low urine volume

High Urinary Uric Acid Excretion (Hyperuricosuria)

Although uric acid is normally found in urine, if its concentration is too high, uric acid crystals can form. As with oxalate, urinary uric acid has two sources: endogenous (the body's own production) and exogenous (from outside the body). Uric acid is produced in the body from the normal breakdown of cells. This is the major source of urinary uric acid in a normal, healthy individual. Uric acid is also produced as a byproduct of protein digestion. As foods containing protein are eaten and digested, they are broken down into purines. Protein intake is not normally a cause of high urine uric acid. Rather, it is the uric acid produced from purine metabolism that is particularly problematic, especially if the intake of foods containing purines is very high. Foods containing purines may be consumed in balance with energy needs without inducing hyperuricosuria. Consumed in excess, however, purine-rich foods may contribute to elevated uric acid production and, hence, excretion in urine, however, not all purines are equal. Seafood and fish, the organ tissue of any animal (including fowl), and the flesh of all mammals contain purines that are associated with increased risk for high uric acid production. The consumption of purine-rich plant foods, such as leafy green vegetables, appears not to contribute; nor does the normal consumption of milk and yogurt. Patients with high urinary uric acid excretion whose diets are thought to be contributory should pay attention to eating seafood, fish, organ meats, and the flesh of all mam-

mals in moderation and not to excess. A registered dietitian can help to assess energy needs and the amount of these foods that may be included in a stone prevention diet.

Two other dietary components may also contribute to excessive urine uric acid. These are fructose and alcohol. Fructose is found naturally in sucrose (table sugar), which is 50 % glucose and 50 % fructose. While fructose is concentrated in certain plants – such as tree and vine fruits, berries, squash and some other root vegetables, and honey – it is fructose added to foods that may be a problem. These foods include soda and other sweetened beverages, canned and juice- or syrup-packed fruits, condiments (such as ketchup, barbecue, and sweet & sour sauce), and prepared salad dressings and pasta sauces. Prepared baby foods also usually contain ample fructose. If fructose intake is high, and if urinary uric acid excretion is also high, reducing fructose intake may help to reduce uric acid production. The metabolism of ethanol in alcoholic beverages increases the breakdown of purines and thus may lead to uric acid overproduction. Some alcoholic beverages, some beers and ales, for example, are high in purines and thus may directly contribute to uric acid overproduction. If alcohol intake is excessive and thought to be contributory to hyperuricosuria, then reducing alcohol intake may be recommended.

Low (Acid) Urine pH

The typical uric acid stone former has a low urine pH. This means the urine, while normally slightly acidic (<pH 7.0), is very acidic (<pH 5.5). Uric acid comes out of solution in an acid urine condition, leading to the formation of uric acid crystals. Obesity or insulin resistance, such as in type 2 diabetes or metabolic syndrome, is associated with lower urine pH. Diet is also known to influence urine pH. The “Atkins diet”, a popular diet fad of the 1990s that advocated restrictions on fruits and most vegetables, was shown to significantly reduce urine pH over a period of 6 weeks, thereby increasing the risk for uric acid precipitation in urine (See Chap. 28).

Conversely, studies confirm that diets with lower acid load, containing a preponderance of foods that induce more bicarbonate formation in the body, lead to higher urine pH in a matter of days. Medication may be required for those with very low urine pH. Increasing alkaline-ash foods may help increase urine pH and promote the intake of fruits and vegetables, which are recommended to increase urinary citrate excretion and for various other health reasons. Table 27.2 provides information about the relative ability of foods to influence urine pH.

Cystine Stones

Cystinuria is a genetic condition resulting in the inability of the kidneys to reabsorb filtered cystine, which is not soluble in urine with a pH of <7. People who form cystine stones require medication. While not a cause of cystine stones per se, diet may contribute to cystine formation. A high sodium intake, for example, increases the amount of cystine in the urine. Cystine stone formers whose sodium intake is excessive may be required to reduce or normalize their intake. In addition a higher-than-normal fluid intake is usually required in effort to reduce the supersaturation of cystine in urine, keeping it more soluble and less likely to form cystine stones. Cystine stone formers are usually advised higher goals than other stone formers for urine volume, sometimes recommended to aim for as much as 5 l (quarts) of urine output daily. Finally, while not recommended for children or pregnant women, limits on the amount of methionine consumed in the diet may be recommended. Methionine is an essential amino acid, meaning that our body does not produce it and that it must be obtained from the diet. It is essential for growth. While a “low-methionine diet” should not be recommended for anyone, efforts to avoid an excessive intake of methionine may be recommended. A registered dietitian can be particularly useful in assessing whether methionine intake is indeed excessive and whether and how it could be controlled.

Special Medical Situations

Nutrition therapy to prevent stones should always be individualized to the patient and to his/her risk factors for kidney stones. Not everyone who forms calcium oxalate stones, for example, forms them for the same reasons. Moreover, people follow various diets for various reasons. Factors influencing how people eat include cultural, social, environmental, physical, philosophical, and economic considerations. Medical factors also influence peoples’ diets. Nutrition therapy for stones should be integrated with these factors as much as possible. While this chapter cannot address all of the above, there are a few specific medical conditions that warrant attention.

Diabetes

Patients with kidney stones who have insulin resistance or diabetes should follow general diabetes meal planning concepts. Usually, patients meet with a registered dietitian upon diagnosis or shortly after to discuss this in detail. There is no reason for stone prevention not to be integrated into diabetes meal planning strategies. Because people with diabetes are at higher risk for uric acid stones, due largely to the tendency for lower (more acidic) urine pH, greater attention may be required for the inclusion of fruits and vegetables as a way to reduce the overall acid load of the diet. Patients with diabetes are sometimes under the impression, erroneously, that they cannot eat fruits because of their carbohydrate content. Nothing could be further from the truth. In fact, fruits and vegetables are highly recommended for people with diabetes. It is the timing of their ingestion, particularly for fruits and the higher-carbohydrate vegetables (e.g., starchy vegetables, such as potatoes, squash, peas, and beans), that is of importance. In order to blunt the effect of the healthy carbohydrates provided by fruits and some vegetables, it is best for the person with diabetes to consume these with meals that provide other macronutrients, namely, protein, fat, and fiber. The co-consumption of all of these nutri-

ents will attenuate the effect of carbohydrates on blood glucose, reducing the potential for a high spike in blood glucose.

Patients with diabetes are also commonly advised to use foods such as peanut butter and nuts and seeds as snacks because they are relatively rich in protein and fiber while providing minimal carbohydrates. While it's true that nuts and seeds can contain significant oxalate, unless the patient has high urinary oxalate or forms calcium oxalate stones, there may be no reason to alter this common diabetes recommendation. On the other hand, if high urine oxalate is a concern, the incorporation of calcium into the snack can allow the patient to continue to enjoy it and still maintain good blood glucose control. Registered dietitians are experts at integrating nutrition recommendations for multiple conditions, and the case of the stone former with diabetes is a good example of where a dietitian is particularly useful.

Malabsorption

Patients with malabsorption sometimes form stones primarily because of the malabsorption; others form stones for other reason(s) but have coincidental malabsorption. Medical conditions such as Crohn's disease, Celiac disease or gluten intolerance, ulcerative colitis, cystic fibrosis, short bowel, or idiopathic chronic diarrhea are frequently associated with malabsorption. Certain contemporary weight loss surgeries, such as the gastric bypass and the duodenal switch (a modified form of the biliopancreatic diversion), may also result in malabsorption. Stone formers with malabsorption may form any type of stone(s); commonly, they form calcium oxalate stones due to the malabsorption of calcium and magnesium, which leaves less of both to bind oxalate in the digestive tract. Typically, patients with malabsorption who have high urine oxalate must consume supraphysiological doses of calcium—usually from a combination of foods and supplements—with each meal in order to ensure there is sufficient calcium available in the digestive tract to bind with oxalate and prevent its absorption. Attention may also be required to patients' uri-

nary citrate excretion, as sometimes malabsorption leads to acidosis, which can result in low urine citrate. In this scenario, the same recommendations as described earlier for low urinary citrate excretion would apply. It may not be possible to increase fruits and vegetables enough to raise urine citrate to therapeutic concentrations. Thus, pharmacologic therapy is often an adjunct.

Another concern in patients with malabsorption and stones is fluid intake. Due to chronic and sometimes prolific diarrhea, stool fluid losses may be very large. It may be difficult to compensate for this. Special strategies may be required to help patients achieve fluid intake goals. In some cases, fluid intake may never be sufficient to compensate completely. In this event, nutrition recommendations aimed at controlling other existing risk factors will require more attention and higher priority.

Osteoporosis: Don't Break a Leg, Break a Stone!

There is no conflict between nutrition therapy to prevent calcium stones and treatment aimed at preventing or slowing premature bone loss. The individual with osteoporosis who also forms calcium kidney stones should neither be restricted for calcium nor supplemented unnecessarily with it. In the absence of malabsorption, calcium intake should be between 1,000–1,500 mg/day and should be distributed at meals. Distribution of calcium at meals accomplishes two things: (1) it ensures that the optimal amount of calcium is absorbed from the digestive tract (typically about 30 % of what is consumed) and made available to bone; and (2) it leaves some calcium available to bind with oxalate to form an insoluble complex that is removed via stool. Calcium can come from foods/diet alone, from supplements alone, or from a combination thereof. Even in the individual with severe osteoporosis, there is no evidence to suggest that excessive calcium intake somehow preserves bone.

Another concern for stone formers with osteoporosis may relate to vitamin D. Many individuals with osteoporosis and in earlier stages of premature

bone loss may have been advised to supplement with vitamin D. Vitamin D deficiency and insufficiency is indeed a contributor to premature bone loss and is a fairly common problem in the U.S. As such, it is not uncommon for patients to be on high doses of vitamin D for several weeks or even months (repletion) until their vitamin D status is normal, at which time their daily dose can be adjusted lower (maintenance). Because one of the actions of vitamin D is to increase calcium absorption from the digestive tract, increasing the amount of calcium in the blood and available to bone, some have suggested that it contributes to or causes hypercalciuria (high urine calcium). While it may be true that chronic, long-term, excessive vitamin D intake could contribute to hypercalciuria, there is no evidence that vitamin D intake by adults in the range between the RDA and the tolerable upper intake level (600–4,000 International Units per day) induces hypercalciuria. Urine calcium may not even rise appreciably in patients who are being repleted for vitamin D with temporary high doses. In short, nutrition therapy for stone prevention may be easily integrated with the nutrition plan to preserve bone mass.

Macular Degeneration

Evidence is amassing that certain fruits and vegetables may protect the macula, located in the center of the retina and responsible for sharp, clear vision. Patients with or at risk for macular degeneration, the leading cause of vision loss in individuals >55 years, are often advised to consume fruits and vegetables containing the carotenoids lutein and zeaxanthin. The highest concentrations of lutein and zeaxanthin in the human body are in the eye, and these carotenoids (also called pigments) are thought to play a positive role there. Foods richest in lutein and zeaxanthin are those with red, orange, and yellow skins, peels, and/or flesh, such as corn, peaches, oranges, papayas, squash, and dark leafy vegetables (in which the red-yellow pigment is overshadowed by chlorophyll). Interestingly, most of these foods are also ample for oxalate; and patients with calcium oxalate kidney stones may have been advised against eating them. This would appear to

present the patient with macular degeneration with a problem: how to preserve vision while preventing kidney stones? Once again, as stated earlier in this chapter, there may be no reason for an oxalate restriction in the first place. As not all calcium oxalate stone formers form stones because of high urinary oxalate, other risk factor(s) could be the cause and should be addressed. Secondly, it may well be possible to enjoy lutein- and zeaxanthin-rich foods that happen to contain oxalate, even if there is a concern for high urinary oxalate excretion. The strategy of coupling calcium-containing foods and beverages with meals and/or when eating these fruits and vegetables may be sufficient to reduce oxalate absorption in the digestive tract and, therefore, its excretion in urine. In summary, the dietary advice for the prevention of macular degeneration and that for preventing kidney stones need not be in conflict; a registered dietitian can be helpful in integrating these two sets of nutrition recommendations.

Conclusion

As there are multiple types of kidney stones and multiple reasons (risk factors) for forming them, there are multiple dietary approaches to prevent them. Thus, there will never be a “one-size-fits-all” diet for all stone formers. It is important to follow nutrition recommendations, if they are indicated at all, that are aimed specifically at correcting nutrition-related contributors to kidney stones. Nutrition recommendations are thus made after assessing a patient’s kidney stone history, 24 hour urine parameters, medications and medical history, and his/her overall dietary pattern. As with any therapy, recommendations aimed at non-existent problems or misguided recommendations aimed at real problems are not helpful and may confuse patients or dampen their enthusiasm for adherence and compliance. Dietary regimens and changes that will have the most desirable effects on stone prevention are those that should receive attention, and these must be individualized to each patient and integrated with any other medically-necessary nutrition regimens in place.

A fit and healthy-appearing elderly patient came to me for nutrition evaluation and education. He had no major medical conditions and was on no prescription medications. However, a small renal stone was incidentally detected on a CT scan he recently had for another reason. He and his wife were soon traveling to Europe, and he was worried about stone-related problems that might impact the trip. He said he would do “whatever it takes” to prevent his stone from growing bigger and from forming new ones. His first words to me were, “Don’t worry, I’ve already cut out all dairy and stopped grinding my own whole wheat.” He continued, “But now I’m confused about what I’m supposed to eat.” “Whoa,” I said, “let’s start from a clean slate: forget everything you’ve ever heard about kidney stones and diet.” He looked at me for a moment and then pulled a 3-ring binder out of his backpack. “You mean all of this?” Inside the binder were dozens and dozens of pages, lists actually, of foods and their oxalate content organized by food groups with tabs. There were some penciled-in calculations in the margins. “What are these?” I asked. He explained that he found so many different oxalate values for the same foods from different places on the World Wide Web and from other sources that he started calculating means and standard deviations for oxalate content. There was a mixture of reporting methods, I noted, with some oxalate reported as mg oxalate per mg of food and some reported as mg oxalate per serving; that was part of the problem. But the other truth was that there really IS divergence in the reported oxalate content of foods. I put down the binder. “Let’s go back to square one,” I suggested... There are several problems with this scenario, which isn’t all that uncommon. Even though the composi-

tion of the patient’s stone was not known, and even though his 24 hour urine was not assessed for stone risk factors, someone had seen fit to tell this man to reduce his calcium intake – hence the dairy embargo – and to avoid oxalate – hence the 3-ring binder. As I assessed the patient’s current diet as well as the one he followed prior to the detection of the stone, it became clear that the new diet was deficient for calcium, deficient for fiber (which might explain, I told him, his new-onset constipation problem), low in prebiotic potential, and low in antioxidants due to low plant food intake. His previous diet, in contrast, was ample for calcium, rich in fiber and prebiotics, and high in antioxidants. Long story short, I recommended he return immediately to his previous diet. “But what about the stone?” he asked. In light of his family history (turns out both his father and a brother had stones), I suggested that his life-long good diet might well have prevented him from having kidney stone problems earlier in his life, like his father and brother. Ironically, I pointed out, his current diet, based in part on recommendations he received at some medical appointment, was more stone-promoting than his usual diet. I saw the patient once more a few months later. He had results from a 24 hour urine collection, which showed no risk factors other than low urine volume. He remains asymptomatic for his stone, which apparently quietly resides in one of his kidneys. As for therapy, we discussed the need to increase fluid intake, to increase urine volume, to maintain a normal calcium intake, timed with meals when possible, and for a high intake of fruits and vegetables. When he stood up to leave, I realized how different he looked than at our first appointment; his face had more color and his eyes were brighter. He said he felt “great” now that he had returned

to his usual diet and happily reported no further problems with constipation. “What did you do with the binder?” I asked. “In the trash,” he said, “along with the white flour and rice I bought when I stopped eating my home-ground

whole grains.” While some patients require much more in the way of targeted nutrition therapy, I was struck by how equally important it may be to avoid and remove therapy that is wrongly applied.

Diet Fads and Stones: *Is Your Diet All Cracked Up to What It Is Supposed to Be?*

28

David A. Schulsinger and Kristina L. Penniston

Summary of Dietary Facts

- Not all diet plans are the same.
- Some dietary patterns are lithogenic and favor stone formation, while others do not.
- In diets that are rich in protein and low in carbohydrates, there is a greater risk to form both uric acid and calcium oxalate stones.
- If on a weight loss diet, be aware that you may be forming stones, depending on the diet.

You are ready to begin your new diet. Your desire is to lose weight or to initiate a diet for other health-related reasons. You may have been recently diagnosed with diabetes, hypertension, hyperlipidemia or coronary artery disease and your physician suggested dietary modification as an initial therapy. You may also have stress incontinence and your physician suggested that it would be ideal to lose weight. Whichever the reason, you were recommended a new diet plan. Are you aware of the risks of this diet? As you would talk with your physician about a newly prescribed

medication or an anticipated surgical treatment, you want to know what the risks and benefits of these treatment options. Likewise, you should know what the risks and benefits are of your new diet. Would you be surprised to know that your weight reduction diet might also increase your risk for kidney stones?

Some dietary patterns can be stone promoting, referred to as *lithogenic*. Other diets may not be related to kidney stones, or they may even help to reduce your risk of stone disease. This chapter will discuss some of the more popular diets, both past and present, and their relationship to nephrolithiasis. The objective of this chapter is not to provide you with a “how to” for implementing each diet but to alert you to any risk for kidney stones that are associated with the diet. If you are planning to initiate any of these or other dietary regimens, a registered dietitian can help you identify which one would be most appropriate for your needs and what type of information you will need in order to implement the right diet plan for you.

D.A. Schulsinger, MD (✉)
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

K.L. Penniston, PhD, RD
Department of Clinical Nutrition Services,
University of Wisconsin Hospital and Clinics,
Madison, WI, USA
e-mail: kpenniston@uwhealth.org

Diets with Lower Risk of Stone Disease

Dash Diet: An Apple a Day Can Keep the Urologist Away!

The Dietary Approaches to Stop Hypertension, or DASH diet, is recommended to help patients maintain normal blood pressure. In addition to

controlling hypertension, the DASH diet is recommended for patients with diabetes mellitus and other chronic diseases because of its many favorable effects. The DASH diet emphasizes a high intake, approaching or exceeding eight servings per day, of fruits and vegetables. It also particularly includes legumes, nuts, and foods made with whole grains. It includes low-fat dairy and a moderate amount of fish, meat, and poultry. The DASH diet is overall low for fat (especially saturated fat), salt, sugar, and refined grains.

One study compared recurrent calcium stone formers and stone formers with hyperoxaluria on either a low oxalate diet, a frequently prescribed recommendation for calcium oxalate kidney stone prevention and treatment, or the DASH diet. The DASH diet was modified for the study to minimize the use of nuts, which contain more oxalate than many other foods. Study results suggested that the DASH diet might be more effective than a diet restricted for oxalate at reducing urinary risk factors for calcium oxalate kidney stone formation [1]. The DASH diet may therefore be a novel approach to prevent kidney stones and a superior alternative to the traditional recommendation to restrict dietary oxalate. Other studies have shown that the DASH diet reduces the risk of stones by 40–45 %.

While the DASH diet in the study was modified by restricting nuts, which are a regular component of the DASH diet, nuts could be consumed concomitantly with calcium containing foods, such as milk or yogurt, to maintain suitably lower risk of stones.

In summary, as hypertension and stone disease are more common in patients over the age of 40, the DASH diet may be a good approach to managing both high blood pressure and the risk of kidney stone development.

Mediterranean Diet

The Mediterranean diet is similar to the DASH diet. It features foods frequently consumed in Greece, Spain, Southern Italy and France, and other countries that border the Mediterranean Sea. This dietary pattern advocates foods such

as fish, fruits, vegetables, legumes, whole grains, nuts, and olive oil. A moderate amount of low-fat dairy, particularly yogurt, is encouraged in this plan. Foods such as meat, cheese, and sweets are very limited. The Mediterranean diet is rich in monounsaturated fats, fiber, and omega-3 fatty acids, and encourages eating plenty of fruits, vegetables, and high-fiber grains. The Mediterranean diet, unlike other heart-healthy diets (which recommend less than 35 % of your calories from fat), recommends an average of 35–40 % of calories originating from fat. The fats encouraged in the Mediterranean diet are mainly from unsaturated sources such as fish and fish oils, olive oil, certain nuts (such as walnuts, hazelnuts, and almonds) and the oils of nuts and seeds (such as canola, soybean, or flaxseed) and from nuts.

A Mediterranean-style dietary pattern may especially protect individuals with high BMI against the risk of nephrolithiasis [2].

Weight Watchers Diet

Consistently ranked the #1 best plan for weight loss (by U.S. News and World Report), the Weight Watchers dietary plan is essentially a balanced diet. It can be manipulated to promote weight loss by reducing portion sizes to reduce calorie intake, especially limiting foods highest in energy (calorie) density. While the weight loss success of the plan may have as much or more to do with the group support it provides, its “point” system framework is associated with ease of use and acceptance. There are virtually no foods that are restricted, but emphasis is placed on those lowest in fat. Dairy consumption is allowed. Fruits and vegetables of all types are encouraged. The Weight Watchers diet can be made to be consistent with vegetarianism. The Weight Watchers diet can also be inappropriately followed, if adherents were to over-emphasize certain foods to the exclusion of others even while remaining within the points system.

Neither the Weight Watchers diet nor weight loss per se has been studied with respect to kidney stone risk. But both would theoretically be

associated with low lithogenic risk factors if followed appropriately in a balanced way.

Vegetarian Diet

Vegetarian diets span the gamut from vegan – which is the absence of any animal- or insect-derived foods and food products – to those that include dairy, eggs, and/or fish. People following vegan diets avoid honey and any food dyes or additives that are animal or insect-derived. Some ethnic diets, such as Asian and Mexican, are largely plant-based and thus may be considered vegetarian diets, depending on the individual. Surveys have shown that the risk for stone disease is 40–60 % lower in persons consuming vegetarian diets. People consuming vegetarian diets compared to omnivorous diets, particularly the Western style, have a decreased risk of uric acid crystallization [3]. Vegetarian diets can provide an excellent supply of whole grains that are high in phytic acid, a plant constituent associated with a 40 % reduced risk of calcium stone formation in individuals eating higher compared to lower levels [4]. It is important to note, however, that vegetarian diets can be just as unhealthy as non-vegetarian diets. Just because a person is “vegetarian” does not necessarily mean that he/she consumes adequate fruits and vegetables or eats a suitably low amount of saturated fat. Even the intake of whole grains can be suboptimal in a vegetarian diet if a preponderance of foods that are more ample for refined grains are consumed instead.

Vegan diets are associated with a lower urinary excretion of calcium and uric acid and higher urine pH. Vegan diets can be deficient for several micronutrients, such as vitamin B12 and vitamin D, unless supplemented.

It is recommended that individuals at risk to form renal stone should consider a vegetarian-style diet [5]. The American Academy of Family Physicians reports that diets high in non-dairy animal protein are associated with a higher prevalence of kidney stones in the United States.

In summary, a vegetarian-style diet of any type can offer significant protection against stone disease if implemented appropriately. A regis-

tered dietitian can be very helpful in assessing whether your vegetarian diet is both meeting your needs and maintaining a low risk for kidney stones.

Raw Food Diets

Raw food diets promote an eating plan of uncooked and unprocessed foods. The relative percentage of raw foods in the diet may vary, depending on the “strictness” of the adherent, from 50 to 100 % raw. A raw foods diet is not necessary vegetarian. Foods may not even be completely “raw,” as heating up to around 115 °F is commonly practiced. Meats, fish, and even raw milk products may be consumed by some, though more frequently the majority of foods consumed are fruits, vegetables, nuts, seeds and herbs.

There are no direct studies examining raw food diets and stone disease. Studies addressing this topic require additional investigation.

Lithogenic Diets

Atkins Diet

The Atkins diet is a well-known weight loss diet, developed over 40 years ago by Dr. Robert Atkins. The goal of this diet is to intensely restrict carbohydrates from all sources, to stimulate the use of body fat for fuel, resulting in weight loss. The diet at first excludes all fruits, fruit juices, starchy vegetables (including legumes), breads, rice, cereals, pasta, and other grain products, and then slowly re-introduces small amounts of these foods over time. The diet eliminates alcohol and caffeine. The diet consists largely of fat and protein from foods such as meats, fish, seafood, eggs, cheese, and non-starchy vegetables. As the individual progresses with the diet and as the carbohydrate restriction relaxes, fatty, high-protein foods continue to be the primary source of diet.

The Atkins diet is difficult to follow for most vegetarians. Diets that contain foods that are high

in protein and low in carbohydrates, especially when limiting fruits and vegetables, can increase the risk of kidney stones. Diets high in animal flesh foods produce a marked acid load to the kidney intake, reducing urinary citrate and increasing the risk for kidney stones [6].

Zone Diet

The Zone diet was developed by Dr. Barry Sears. The Zone diet typically caps daily calories for women at 1,200 and 1,500 for men. The diet program involves eating five times a day, which includes three meals and two snacks. Meals and snacks are to be consumed ideally at specific times during the day, e.g., within an hour of waking and just before bedtime. The plan recommends going no more than 5 hours without eating. Each meal and snack should contain a distribution of 40 % carbohydrates, 30 % protein, and 30 % fat; referred to by some as the 40:30:30 ratio. Overall, this represents a fairly high protein and fairly low carbohydrate diet, though it is not nearly as restrictive as the Atkins diet. Even so, many of the same lithogenic risk factors that apply to the Atkins diet theoretically apply with the Zone diet. However, if practiced in the context of a vegetarian-style diet ample for fruits and vegetables, the Zone diet may not confer as great a risk for kidney stones.

South Beach Diet

The South Beach diet is a low carbohydrate, high protein diet originally created by Dr. Arthur Agatston and dietitian Marie Almon to help individuals lower their risk for heart disease and eventually became popular for fast weight loss. The South Beach diet emphasizes certain carbohydrates, such as whole grains and specific fruits and vegetables, over others. The South Beach diet eventually includes dairy, though it initially restricts it. Similar to the Atkins diet, there are three phases, the first of which eliminates breads, rice, pasta, fruit, dairy, alcohol, and starchy vegetables (such as legumes and potatoes). Fruits and whole-grain breads, rice, and pasta are re-introduced in the second phase. Weight is

expected to be lost during these first two phases. The third phase is considered non-restrictive with the goal of maintaining the desired weight with a moderate intake of most all low-fat foods.

As with any type of diet, inappropriate restrictions and/or inclusions may promote complications. Kidney stone formation is such a potential complication with the South Beach diet, particularly during the first two phases, which are primarily low carbohydrate and high protein. Uric acid and calcium stones could both form if there is a higher acid load delivered to the kidneys.

Seizure Diet (Ketogenic Diet)

A ketogenic diet is prescribed to patients who have epilepsy and intractable seizures [7, 8]. The ketogenic diet includes foods that are high in fat and protein and low in carbohydrates. Without carbohydrates for conversion to glucose – the primary energy source for brain, red blood cells, and other body tissues – are ketones, which are derived from the amply available fat. The elevation of ketones in the blood, referred to as ketosis, is what is thought to be responsible for the management of epileptic seizures [9].

This diet is also associated with weight loss, due to the energy restriction conferred by the virtual removal of carbohydrates, and has been adopted by some for this purpose. Ketogenic diets have a similar effect to the Atkins diet in increasing the risk of stones. In one study, the risk of stones was 5 % in children on ketogenic diets [8], which is higher than the risk for children on non-ketogenic diets. Approximately half of these stones were composed of uric acid, and the remaining stones were calcium oxalate mixed with calcium phosphate or uric acid. Ketogenic diets are 500 times more likely to develop uric acid stones and 50 times more like to develop calcium oxalate stones but can be prevented with the prescription of potassium citrate.

Western Diet

The so-called “Western” dietary pattern is also referred to the “meat-sweet diet” or the “standard” or “typical” American diet. There are

many versions of the so-called Western dietary pattern, including some vegetarian diets. Additionally, although associated with the U.S., dietary patterns in many other countries, especially more developed countries, have a Western-style pattern. The pattern is typically characterized by high protein intake (especially from red meat), sugary deserts, high fat dairy and refined grains.

Diets that are high in protein, especially animal-derived, tend to promote the excessive excretion of urinary calcium, oxalate and uric acid, increasing the risk of kidney stones. Epidemiologic studies support that Western style diets are associated with a greater risk of calcium and uric acid kidney stones than the risks associated with the more prudent diets.

Dukan Diet

This diet was developed by Dr. Pierre Dukan. This diet consists of four phases:

- **Attack Phase:** Unlimited lean protein for up to 10 days plus at least 6 cups of water and 1½ tablespoons of oat bran daily.
- **Cruise Phase:** For up to a month as needed for desired weight loss, alternate the “Attack” phase regimen – in an every other day fashion – with the addition of non-starchy vegetables and a total of 2 tablespoons of oat bran daily.
- **Consolidation Phase:** Unlimited lean protein and vegetables of any kind daily with one piece of fruit, up to two slices whole-grain bread, and 1 oz of cheese; small amounts of starchy foods can also be included.
- **Stabilization Phase:** Anything is allowed, while maintaining desired body weight, on 6 days of the week with the 7th day limited to the “Attack” phase regimen.

Similar to the Atkins diet, the meal plan during the first two phases is high in protein and relatively low for fruits and vegetables. Diets high in protein can increase the risk of calcium stones as these diets increase the acid load in the urine, reducing urinary pH and urinary citrate while increasing urinary calcium. No studies to date have looked at the Dukan diet directly and the risk for kidney stone formation.

Paleo Diet: Does a Stone Age Diet Increase Your Risk for Kidney Stones?

This diet is based on the primary dietary pattern thought to be followed during the Paleolithic or “caveman” period, when sustenance was obtained via hunting animals and gathering plant foods. Although the Paleo diet is commonly referred to as low carbohydrate, it is not necessarily so. Foods associated with this dietary pattern are lean meats of any kind (especially those that are grass fed), fish and seafood, insects, eggs, fresh fruits and vegetables of any kind (variably including legumes), and unsalted nuts and seeds of any kind. Missing from this diet are dairy foods (including cheese) and processed grains and cereals. There is some debate about whether “wild” grains – such as wild rice, quinoa, amaranth, and buckwheat – are allowable. Sodium and fat are limited in a Paleo dietary pattern to that which occurs naturally in foods.

There have been no studies to date looking at stone disease and patients on the Paleo diet.

Conclusion

In summary, several protein rich diets (Atkins, Zone, South Beach, ketogenic) can increase the acid load delivered to the kidneys, resulting in bone loss, increasing urinary calcium, decreasing urinary citrate levels and increasing the risk of both uric acid and calcium stones. Consult with a registered dietitian and your urologist if you are a stone former and considering a weight reduction diet plan. Remember, *not all diets are the same!*

Mrs. MO is a 37-year-old obese patient with a BMI of 41. She has a known history of Gout and uric acid stones for which she takes a prophylactic dose of potassium citrate with good results. She has been able to manage her stones with medical therapy. Concerned about her weight, she decided to initiate a new diet recommended by some friends. This was a low carbohydrate, high

protein diet. Six months later, she had lost 45 lbs. and began to experience some right flank pain without nausea, vomiting, fever or chills. Thinking this was a recurrence of her uric acid stones, she recalled that her urologist put her on 40 meq of potassium citrate when she had colic attacks. With additional medication at home, she took it upon herself to take the additional potassium citrate. After several days, there was no improvement in her symptoms. She wound up in the ER with intractable flank pain, nausea, vomiting without fever or chills. Her Urologist performed an emergency ureteroscopy and stone manipulation for her 5 mm obstructing mid ureteral stone. Stone analysis revealed a Calcium Oxalate stone. Still on her weight reduction diet, her postoperative 24 hour urine revealed high urinary calcium, low urinary citrate and low urinary pH, consistent with her protein rich diet. Following a consultation with her urologist and establishing a relationship with a registered dietician, modifications were made to her diet with reduction in protein and a maintenance dose of potassium citrate so that she could get the combined benefit of weight reduction without the expense of additional stones.

References

1. Noori N, Honarkar E, Goldfarb DS, Kalantar-Zadeh K, Taheri M, Shakhssalim N, Parvin M, Basiri A. Urinary lithogenic risk profile in recurrent stone formers with hyperoxaluria: a randomized controlled trial comparing DASH (Dietary Approaches to Stop Hypertension)-style and low-oxalate diets. *Am J Kidney Dis.* 2014;63(3):456–63.
2. Soldati L, Bertoli S, Terranegra A, Brasacchio C, Mingione A, Dogliotti E, Raspini B, Leone A, Frau F, Vignati L, Spadafranca A, Vezzoli G, Cusi D, Battezzati A. Relevance of Mediterranean diet and glucose metabolism for nephrolithiasis in obese subjects. *J Transl Med.* 2014;12(34):1–6.
3. Robertson WG, Peacock M, Marshall DH. Prevalence of urinary stone disease in vegetarians. *Eur Urol.* 1982;8:334–9.
4. Curhan GC, Willett WC, Knight EL, et al. Dietary factors and the risk of incident kidney stones in younger women: Nurses' Health Study II. *Arch Intern Med.* 2004;164:885–91.
5. Robertson WG, Peacock M, Heyburn PJ. Should recurrent calcium oxalate stone formers become vegetarians? *Br J Urol.* 1979;51:427–31.
6. Siener R, Hesse A. The effect of a vegetarian and different omnivorous diets on urinary risk factors for uric acid stone formation. *Eur J Nutr.* 2003;42:332–7.
7. Furth SL, Casey JC, Pyzik PL, Neu AM, Docimo SG, Vining EP, et al. Risk factors for urolithiasis in children on the ketogenic diet. *Pediatr Nephrol.* 2000;15:125–8.
8. Sampath A, Kossoff EH, Furth SL, Pyzik PL, Vining EP. Kidney stones and the ketogenic diet: risk factors and prevention. *J Child Neurol.* 2007;22:375–8.
9. Freeman JM, Kossoff EH, Hartman AL. The ketogenic diet: one decade later. *Pediatrics.* 2007;119(3):535–43.

David A. Schulsinger

Summary Stone Facts

- If you are having symptoms of a stone it may be a new stone, however, do not lose sight that this may be a separate and distinct diagnosis.
- Be aware that other diseases may present the same way as a stone, referred to as the *differential diagnosis*.
- Tell your physician or Urologist about these symptoms so that they can initiate the appropriate tests and make the correct diagnosis.

Now that you have finished the reading passages up to this chapter, it is important for us to review many of the factors surrounding your stone formation and stone prevention. You may be stone free at this time and it is important to review some of the facts as to how you enhance your likelihood of remaining stone free. It is important to manage your expectations on stone prevention and steps required to achieve this goal. This chapter discusses tips allowing patients to manage their expectations and inspiring individuals to minimize their risk of future stones.

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

Maintain Awareness of Your Symptoms

First, you may recall the pain that you had during your last stone attack. You may have had flank pain associated with nausea, vomiting, fever and/or chills. The pain may have radiated to the groin associated with discomfort in the testis in men or in the labia or vaginal area in women. On the other hand, you may have blood in the urine as the only presenting sign of your stone.

If you have these symptoms again, it may be a new stone or a persistent recurrence of a stone. It is important not to lose sight that these symptoms may be an indication that another disease process is present and unrelated to your stone. It maybe a sign related to the urinary tract, or one completely unrelated to the urinary tract.

In the case of the flank pain, it may be a stone blocking the flow of urine, resulting in renal swelling, known as *hydronephrosis*. On the other hand, this pain may be something within the gastrointestinal (GI) tract or muscle skeletal system. Think about your symptoms of your GI tract. Do you have a history of constipation? When was your last bowel movement? Did you have previous abdominal surgery? With previous abdominal surgery, this may be small bowel obstruction secondary to adhesions.

On the other hand, if you are having right lower quadrant pain, it is important to entertain appendicitis or rule out a ruptured ovarian cyst in the differential diagnosis. If the pain is in the left

lower quadrant, the differential diagnosis should include diverticulitis.

If the pain is in the scrotum, the differential diagnosis may be an infection of the epididymis, known as *epididymitis*. More importantly, it may be a twisting of the testis, often referred to as a *testicular torsion*. Testicular torsion represents a urological emergency that requires surgical intervention.

Finally, blood in the urine may be a stone irritating the lining of urinary tract within the kidney, ureter or bladder. On the other hand, it may be something as simple as a urinary tract infection. Alternatively, it may be something more serious, such as a renal tumor, ureteral tumor or bladder tumor. It is important to make your Urologist aware of these symptoms so that s/he can completely evaluate your urinary tract and make certain that *no stones are left unturned!*

Remember, when it looks like a duck, quacks like a duck, moves like a duck, it is usually is a duck! However, do not lose sight that these symptoms may be a “red herring” and may be the unexpected elephant in the room! It is critical that when you have any of these symptoms previously described, that you don’t simply attribute them as being associated with a stone. It is paramount that you consult your urologist, primary care physician and/or Gynecologist to initiate the work up and make a definitive diagnosis.

In my experience over the years, I have found that over time, patients have become quite sophisticated about understanding their bodies and appreciating their symptoms associated with stones. Once a patient has had a stone, or what I have described as a “stone veteran”, they have become quite in tune with their body and know exactly what their symptoms are like for a stone recurrence.

Not all tremors result in earthquakes. If you have a small “twinge” or pressure feeling in your flank, don’t automatically assume that it is a stone. Remember, stone pain can be relentless and sometimes persistent. The presence of flank pain for a few seconds may not be stone related. However, if that pain is persistent, consider that this may in fact be a stone.

My experience with stone patients has taught me that individuals have a heightened degree of suspicion once they had a stone attack. Every patient recalls that first time they experienced flank pain associated with a stone. While it may have taken several days for it to crescendo into “full blown” flank pain, patients often recall several days or even a week of suspected “gas pain” or even “muscle ache” that the individual self-diagnosed and nursed on their own before they ended up in the ER.

Moving forward, after that first bout of pain, patients have a heightened degree of suspicion about what stone pain is all about. Their threshold for suspected stone pain is much less for making that “self” diagnosis and calling the urologist. While this is ideal for not allowing the pain to linger into a full-blown flank pain, the room for misdiagnosis and “the boy who cried wolf” potentially greater. Remember, not all flank pain is stone related. Not all stones present with pain. Be aware and be vigilant!

Maintain Your Relationship and Follow Up with Your Urologist

You are stone free and you just completed your first 24 hour urine and your Urologist has made some recommendations to you for stone prevention-this is great news! Do not believe after reviewing your stone analysis and 24 hour urine that you are done with your urologist. The same way that your Ophthalmologist tells you to return in 2 years after an eye exam or your Gastroenterologist, who tells you to come back in 5 years after a negative colonoscopy, you will need to follow up with your urologist. He or she will determine the interval for the follow up visit. If the stone analysis and 24 hour urine report demonstrate minimal or no risk factors, they may offer you a follow up in 1 year. On the other hand, if there are many risk factors for your stone requiring both dietary and medical therapy, a follow up with a repeat 24 hour urine may be necessary in just 3 months. Be prepared for a range of situations!

Stone prevention, as previously described in a continuum of tests that will make certain that your dietary practices, hydration practices, medication dosages are in check and that your urinary chemistries are optimized. This may mean that you see your urologist once or twice a year to be certain that you remain stone free. I usually see my patients twice a year with alternating 6 month periods of renal ultrasounds and 24 hour urine. Once we have the patient on track with negative renal ultrasound and normal 24 hour urine testing, I put the patient on a once a year tract where they will return to the office for annual renal ultrasound and 24 hour urine testing.

On the flip side, if a patient has abnormal 24 hour urine testing that requires medical intervention or dietary modification, we maybe do more frequent interval testing of 24 hour urines until a more “normal” 24 hour urine test is achieved. The same approach is true is persistent stones are seen on your imaging studies.

Finally, be demanding of your urologist to practice stone prevention. If he/she does not, find a Urologist who does.

In summary, be prepared to establish a lasting relationship with your urologist. I tell patients that they are “stuck” with me, for better or for worse, to keep them stone free!

Despite following all of your urologists’ recommendations and maintaining your stone prevention protocol, you may still manufacture a kidney stone. There are certain situations and parameters beyond our control, and despite your best efforts, a stone is born! The importance, however, of identifying this stone earlier and potentially before this stone becomes symptomatic, is that you can optimize your approach to passing this stone while it’s small. Hydration is typically your best friend and ideal medicine.

Maintain Your Relationship with Your Dietician

A dietician is an essential asset to your health care team. Based on your stone history and 24 hour urine test results, the dietician can

formulate a diet plan to lower your risk of forming future stones. While there are dieticians who may specialize in cardiac care, endocrine, weight reduction, make sure that you choose a registered dietician who specializes in nutritional support for patients with kidney stone disease.

There are a number of disease states that dieticians treat that are associated with stone disease. Dietary plans for patients with cardiac/hypertension, obesity, diabetes or endocrine disorders and stone disease are NOT mutually exclusive problems. Studies have shown, for example, that obesity increases your risk of stone disease, therefore, a dietician treating a patient with obesity and stones can address both conditions together. Likewise, patients with hypertension and stone disease can be treated together. A DASH (Dietary Approaches to Stop Hypertension) diet high in fruits and vegetables, low in animal protein and moderate in low-fat dairy products has been shown to not only be ideal for the patient with hypertension, this dietary approach can also reduce the risk of kidney stones.

In summary, a dietician is an essential part for your stone prevention. Certain disease states in addition to stones can be managed with the appropriate dietary regimen recommended by your dietician. Oftentimes, these conditions can be treated together with the same diet. *This is the equivalent of killing two birds with one stone!*

Maintain Your Stone Prevention Therapy

Maintain Your Hydration Status

Adequate hydration is the quintessential factor in stone prevention (review Chap. 25). Patients are encouraged to drink enough water and other fluids (citrus drinks) to produce a minimum of 2 L of urine/day. Drinking 2–3 L/day is recommended. Your level of activity, environment you reside, occupation and the type of stone you make are critical determinants as to the volume of fluid you consume. Patients who do excessive

exercise, live in hot weather areas, work in hot environments and have a history of cystine stones may need to drink more fluids than other patients.

Maintain Your Dietary Recommendations

Your Urologist may have made some recommendations as to modifying your diet for stone prevention (see Chap. 27). Remember, there is no one diet that is right for all stone patients. You may be a calcium oxalate stone former but your 24 hour urine demonstrates that you have high urinary sodium and high urinary calcium. It is your high urinary sodium that drives the high levels of urinary calcium. In this case, you may lower your dietary intake of sodium, switching it to sea salt or potassium chloride while maintaining your dietary intake of calcium.

Alternatively, you may make a calcium oxalate stone but your 24 hour urine demonstrates high urinary oxalate levels. In this case, reducing your dietary intake of oxalate may be prudent. In addition, and somewhat paradoxical, but **INCREASING** your dietary calcium in this scenario will help the binding of calcium and oxalate in the GI tract resulting in its elimination.

We frequently see the patient with calcium oxalate stones who has a 24 hour urine test result demonstrating a normal oxalate level and a low total urine volume. This patient needs to increase their oral consumption of fluids, but if they desire to drink ice tea, known to be rich in oxalate, they can have some of this as their fluid without jeopardizing their risk for stone formation.

In summary, there is no single food group that is right or wrong for all. Allow your dietician and/or urologist to guide you on dietary recommendations. These suggestions stem from the results of your stone analysis and 24 hour urine results. Different patients with the same stone may require opposite dietary recommendations. Likewise, patients with the different stones may require similar dietary counseling for their stone prevention.

Maintain Your Medical Therapy

Your Urologist may have put you on medication for treatment and prevention of your stone (see Chap. 19). The medication you take today most likely will not be the same medication or dosage that you take in the future, and it is plausible that you may not require medication in the future! The rate-limiting step here is what your metabolic work up shows. For example, you may have hypercalciuria today that requires you to be on Hydrochlorothiazide. However, your 24 hour urine 6 months from now may show normal calcium levels but a low citrate level suggesting that you should be taking potassium citrate. The bottom line: follow up with your urologist so he/she can determine the best regimen of medication and nutritional supplementation.

Please remember that your Urologist's recommendation for you to take medication is an attempt to reduce your risk for developing future stones. Despite these recommendations, even taking medication may not guarantee or prevent you from forming stones again!

Maintain Your Complementary Medication Therapy

Not all patients require or prefer taking pharmaceutical grade medication for their stone prevention. Stone prevention with complementary medication can and often times is utilized for these purposes (see Chap. 26). For example, for a patient with a uric acid stone and modest reduced levels of citrate determined by 24 hour urine testing, may require a nutritional supplement containing oral potassium citrate to minimize their risk of future stone formation.

Maintain Your Follow Up Tests

The first steps in stone prevention is knowing what type of stone you make and understanding what causes the stone to form. This is accom-

plished with a stone analysis and 24 hour urine, respectively (review Chap. 24). The stone that you pass or surgically remove will be sent for stone analysis. The 24 hour urine test identifies the chemicals in the urine that may contribute to that stone.

Your Urologist will determine at what interval the 24 hour urine tests needs to be repeated. For patients that have no risk factors or minimal risk factors (i.e., low urine volume), the need to repeat the 24 hour urine test would be less frequent than a patient who presented with multiple abnormal chemical factors in the urine.

You may be required to do additional laboratory testing such as a urine analysis and urine culture to rule out hematuria or infection, respectively; serum chemistries to rule out obstruction or renal insufficiency; serum calcium levels or serum parathyroid hormone test.

Diagnosis

Interval imaging studies are required to see if new stones have formed since your last procedure or metabolic work up. I attempt to limit the amount of radiation exposure patients receive who are being worked up for stones and those patients managed for stone prevention. I will obtain a renal ultrasound in patients being screened for stones and obtain a KUB to rule out a radiopaque stone (calcium vs. uric acid) if the renal ultra sound confirms its presence.

Maintain Your Relationship with Yoursel!

Finally, my best advice would be to you, the patient. It is the month of December and you are making your list of annual goals and New Years' resolutions. Stone prevention should be there right on top of the list. Patients should look at their current eating habits and ask themselves, am I following my urologist's recommendations to my best ability? Is there anything I can do to

improve my eating and drinking recommendations during the day, especially during work or while in school? Review your dietary, drinking and medication habits and come up with a refined plan to maintaining these therapy recommendations. Don't forget to include a visit to the urologist and dietician on that list too!

Is Another Stone in Your Future?

Notwithstanding your best efforts to maintain a stone prevention course, it remains possible that you may develop a new kidney stone. Despite following the urologist's treatment recommendations for your stone disease, this will not ensure a 100 % guarantee that you will remain stone free. There will be a reduction in your risk for future stones, but a possibility for future stones remains.

Your Urologist will work with you to maintain your preventive care, but a new kidney stone remains a potential and realistic possibility. Your urologist will discuss with you the various treatment options. They will manage your expectations about what the various procedure options are and what the potential complications of these procedures will be. Be open-minded and ask the appropriate questions:

1. What is involved with the procedure?
2. What are the risks of the procedure?
3. What is the preparation for this procedure?
4. Will I be in the hospital for this procedure?
5. What is my postoperative course?

Conclusion: *Don't Be a Rock Star!*

In summary, treating your stone and practicing stone prevention are the ingredients to a successful relationship with your Urologist. Knowing the right questions to ask and getting successful advice from your urologist and dietician will allow you to manage your expectations and to minimize your risk for future stones. *Remember to maintain your "A" team!*

I had a new stone and my physician performed a ureteroscopic procedure that involved laser lithotripsy and removing my stone. He put a stent in at the end of the procedure for which I was told would keep my ureter patent and allow small residual stone fragments to pass. Those were the good things! However, I was told that a stent could “tickle” my bladder, which may cause me to urinate more

frequently, may produce some burning, and even cause some flank discomfort from urine refluxing up the tube during voiding. For these less desirable side effects, I received some medication to relax my bladder. I am grateful that my urologist managed my expectations regarding the stent. Knowing this in advance, made it much easier to deal with until my stent was removed.

Part VII

Bonus

Brian Sninsky and Stephen Y. Nakada

Introduction

The treatment options for kidney stones have expanded immensely over the past 30 years, and new concepts in diagnosis and treatment continue to evolve from the research bench to the patient bedside. Advances in imaging capabilities, increased power, durability, and maneuverability of laser stone fragmentation technology, and novel tools to improve patient involvement in surgical decision-making are at the forefront of stone management. In addition, animal models, including rodent, swine, and even fruit flies, hold future promise for advancing our understanding of the progression and management of stone disease. New drug treatments with innovative mechanisms may provide tomorrow's urologist with new strategies for both therapy and prevention. Similarly, probiotics have received increased focus as a potential approach to the breakdown of oxalate in the digestive tract, a component in nearly 80 % of kidney stones. Perhaps the most unique development is the recent application of robotic-assisted surgery for both basic and complex stone management. A multitude of exciting new therapies, techniques, and technologies are on the horizon, and the future is bright for the management of kidney stones. In closing, we review the most

recent American Urological Association (AUA) guidelines for the medical management of stone disease.

New Technology

Imaging

Patients presenting to the emergency room with classic symptoms of kidney stones including flank pain, pain that spreads to the lower abdomen and groin, pain with urination, blood in the urine, and/or nausea and vomiting are typically diagnosed using a CT scan. This imaging test is more sensitive than x-ray, and can reveal more reliable information on the exact size and position of stones. Though these are both key factors in determining treatment, the chemical composition of a stone is also beneficial for providers to know. Different types of stones have different degrees of hardness (measured in "Hounsfield Units"), and are best treated with different medical or surgical modalities. In the past, the only way to determine stone composition was by sending an actual sample of a patient's stone, collected via spontaneous passage or surgical means, to a laboratory. However, new dual-energy CT (DECT) is a promising new technology that may help clinicians more accurately distinguish stone type at the time of presentation and diagnosis. Though this technology is still in the early stages of development, a recent study [1] found DECT

B. Sninsky, MD • S.Y. Nakada, MD (✉)
Department of Urology, University of Wisconsin
School of Medicine and Public Health,
Madison, WI, USA
e-mail: nakada@urology.wisc.edu

imaging correctly identified stone composition in 74 % of cases, as opposed to only 52 % with standard CT. Even more impressively, DECT was able to correctly differentiate non-uric acid stones from uric acid stones in 93 %, versus only 40 % using CT. Though further improvement and testing is needed, DECT may help providers better select the most effective and safe treatment for kidney stones based on size, location, and chemical composition.

Though traditionally used as an imaging (US) or fragmenting tool (SWL), ultrasound technology has advanced greatly over the last 5 years to the point where ultrasonic propulsion can be used to move stones within the urinary tract [2]. Though minimally invasive techniques for stone fragmentation, including SWL, ureteroscopy (URS), and percutaneous nephrolithotomy (PNL), have improved considerably in the last decade, residual fragments may still cause recurrent symptoms and may require future intervention. In these cases ultrasonic propulsion may be an effective tool in the urologist's arsenal for clearing residual stones. With this technology, stones could be repositioned at a clinic visit with the patient awake, in a procedure similar to standard ultrasound imaging. Additionally, ultrasonic propulsion could supplement medical expulsive therapy in patients presenting with small stones. Similarly, painful obstructing stones may be able to be moved back into a non-obstructing position in the renal pelvis, thereby reducing pain and avoiding an emergent procedure and narcotic analgesic medications [3].

Shared Decision Making (SDM)

There has been a considerable shift in healthcare over the last decade to increase focus on patient centered care [4]. In contrast to the traditional view with the clinician making decisions on behalf of the patient, shared decision making with greater patient input has become a key emphasis for providers, legislators, and patients themselves. The shared decision making model consists of three steps: (1) introducing choice, (2)

describing options, often with the use of an integrated informative handout, and (3) helping patients explore options and make decisions [5]. The use of a shared decision-making aid is supported by extensive research, including over 80 randomized trials that demonstrate increased knowledge gain for patients, as well as improved patient confidence and involvement with decisions made [6].

Currently, shared decision making aids exist in multiple areas of urology, including BPH [7] and prostate cancer [8]. However, until recently the use of a shared decision making aid in patients with kidney stones had not been explored. Patients with kidney stones requiring surgical treatment pose an ideal opportunity for the implementation of a shared decision making aid, as multiple reasonable treatment options exist. Depending on various patient and stone factors, surgical options may include shockwave lithotripsy, ureteroscopy, and/or percutaneous nephrolithotomy. Early development and testing of a shared decision making aid that compared shockwave lithotripsy and ureteroscopy showed that 86 % of patients found the aid helpful, and 79 % preferred the shared decision process compared to a typical office discussion [9]. With the addition of further patient input and validation, kidney-stone-focused shared decision aids will soon become widely available to physicians and patients. As urologists continue to search for strategies to improve patient outcomes and satisfaction in stone disease, shared decision aids will play a key role in both the transfer of information and collective treatment choice.

Animal Models to Understand Stone Disease

Animal models have long been used in medicine to better understand the mechanisms of disease and potential application of new therapies. Kidney stone formation is a complex series of events that occurs through multiple pathways, ultimately causing crystal formation and growth that becomes a kidney stone. Animal models

are used to simplify this complicated process into a series of distinct steps that can be individually studied and used to evaluate new therapies. In particular, rodent, swine, and even fruit fly models of stone formation are at the cutting edge of new research exploring the various types and treatments of kidney stones.

As discussed previously, the majority of kidney stones are composed primarily of calcium oxalate (CaOx), and appropriately the majority of animal models are based on formation of these stones. In both animals and humans, high concentrations of oxalate in the urine (hyperoxaluria), causes crystal formation with calcium ions to eventually form kidney stones. The initial CaOx model in rats was achieved by adding ethylene glycol (a chemical found in antifreeze) to the animal's drinking water [10]. Ethylene glycol is converted to oxalate by the liver then absorbed through the gastrointestinal tract, resulting in hyperoxaluria and subsequent stone formation within the kidney. However, ethylene glycol is toxic to multiple organ systems, including the kidney, making it difficult to differentiate if cellular damage is secondary to the initial ethylene glycol, or the crystallization and formation of stones [11]. The more recent rat model of nephrolithiasis is achieved by feeding the animal high concentrations of a different oxalate precursor, hydroxyproline (a derivative of the common amino acid proline found in many Western diets) [12]. This model more accurately models the increase in oxalate secondary to diet, and avoids the renal toxicity seen in ethylene glycol models.

One of the interesting new rat models is the formation of CaOx stones in obese rats that have undergone gastric bypass surgery [13]. In spite of the many benefits of gastric bypass surgery for weight loss, it is now known that in humans this surgery increases risk for kidney stones secondary to hyperoxaluria [14]. Normally, free oxalate and calcium in the gut lumen bind and are passed harmlessly out of the gastrointestinal tract via stool. However, patients with gastric bypass have an increase in fatty acids that bind calcium, leading to an increase in unbound oxalate. This

increased oxalate is absorbed in the bloodstream, where it eventually makes it to the kidney causing hyperoxaluria [15].

Though rats have historically served as the primary animal metabolic model for stone disease, the last decade has seen the development and advancement of a hydroxyproline induced swine model [16, 17]. Swine have been extensively studied in biomedical research, and are remarkably similar to humans in both genitourinary anatomy and physiology [18]. In addition, they have comparable nutritional needs as omnivores, and similar gastrointestinal functioning. The anatomical parallels to humans make swine an ideal surgical model, and as techniques to encourage stone formation improve, this model will be a major player in testing new minimally invasive stone treatments and medications.

Finally, one of the newer and more innovative animal models has been the emergence of *Drosophila melanogaster* (fruit fly) as a translational model of stone disease [19]. Though anatomically and physiologically different than humans, *drosophila* fed high-oxalate diets form stones in 2–3 days, in sharp contrast to the weeks required for the previously mentioned models [20]. This short formation period allows researchers to quickly and economically study the mechanistic cycle of stone development. Another advantage over other models is low financial cost, and the minimized constraints from ethical and institutional review boards. Additionally, another *drosophila* model has been constructed based on the genetic removal of gut oxalate transporters, resulting in a build-up of oxalate and resulting hyperoxaluria [21]. Though still in the early phases of development, this research may provide the foundation for isolating human genes responsible for kidney stone disease.

New Medications and Probiotics

Most symptomatic kidney stones are treated with increased fluids, pain control, and an alpha-blocker (a medication that relaxes the ureteral

smooth muscle, improving passage of stones or fragments), or surgically managed. Though there have been no major pharmacologic breakthroughs for the treatment of stone disease in the twenty-first century, two new therapies have shown promise in initial laboratory testing.

First, a promising compound known as cystine dimethyl ester (CDME) has been studied for its ability to inhibit cystine crystal growth. Cystine stones are rare, and formed by patients with an autosomal recessive trait that causes them to excrete high levels of cystine in the urine. Precipitation of cystine crystals in the urine result in large recurrent stones, as well as functional damage to the kidney. CDME is a compound with similar structure to cystine that essentially blocks aggregation of cystine molecules, thereby decreasing both the overall size and speed of stone formation [22]. However, CDME can accumulate intracellularly and cause toxicity and even kidney failure, so future studies are needed to identify safe dosing levels prior to starting human trials [23].

Secondly, oxalate decarboxylase (Oxazyme), is an enzyme that has been shown *in vitro* to degrade oxalate in simulated gastric and intestinal conditions [24]. Theoretically, if taken regularly with meals, the enzyme could have the potential to breakdown oxalate before it could be absorbed from the gut lumen, causing hyperoxaluria and stone formation. Similarly, many bacteria have the ability to degrade oxalate, and hold promise as probiotics for the prevention of kidney stones. The human digestive tract is colonized with innumerable unique bacteria, although very few have the ability to degrade oxalate. *Oxalobacter formigenes* is found in the colon of 60–80 % of adults, and degrades oxalate, as its sole energy source. Studies have been mixed, but it is suggested that patients without this bacteria excrete increased urinary oxalate and are at higher risk for stones [25]. The majority of oxalate may already be absorbed by the time it reaches *O. formigenes* in the colon, creating the opportunity for an oxalate degrading probiotic that works in the gut and small intestine. Research has been encouraging

thus far, with the greatest benefit in patients with initial hyperoxaluria secondary to increased gut absorption [26].

Robotic Approaches to Stone Disease

The robotic platform was introduced to the field of urology in the early 2000s, and has emerged as a popular and effective method for prostatectomy. The last few years have seen the evolution of this technology to other applications in urology, including the treatment of stone disease. In 2011, robotic flexible ureteroscopy was implemented for the first time, with encouraging initial results [27]. Eighteen patients were treated with no major complications noted and a 3 month stone free rate of 89 %. In this study, the ureteroscope was manually introduced into the urethra and advanced to the collecting system of the kidney, at which point the robotic actuator was fixed to the scope. Another application of the robot in stone disease is the surgical removal of large, impacted lower ureteral stones. These cases are extremely challenging, as shockwave lithotripsy has a high failure rate, and long operative times and complications, including ureteral perforation, are often seen with ureteroscopy. Here, robotic surgery has been suggested as an alternative to laparoscopic stone removal due to ease of suturing and a reduction in operative time [28].

Advantages of the robotic platform include improved surgical precision and increased range of motion, with the ultimate goal of safer outcomes and less surgeon-to-surgeon variability. Similar to the adjustable tracking speed for a computer mouse, the robotic platform allows motion scaling to enhance fine motor tasks, especially useful for visualizing and treating small, hard to reach stone fragments. Despite these advantages, drawbacks include limitations of current software and hardware, and most importantly the high cost of the robotic platform versus standard manual ureteroscopy and laparoscopy [29]. In the future, randomized controlled trials that directly compare outcomes, efficiency, and

cost of robotic versus standard techniques will determine the direction of surgical stone management.

New AUA Guidelines for Medical Management of Kidney Stones

In 2014, the AUA released new guidelines on the medical management of kidney stones [30]. This framework of these guidelines provides a clinical guide for providers to evaluate, diagnose, prevent, and monitor patients with kidney stones. Nearly 50 recently published studies on medical management were reviewed by top stone experts, and used to create these evidence-based guidelines. Below, we review key concepts from the new guidelines:

Evaluation

One of the most important sections of the guidelines is the work-up of new patients presenting with kidney stones for the first time. The panel recommends all patients should undergo a detailed medical and dietary history, as well as basic blood work and urinalysis. The history can reveal important risk factors associated with varying degrees of stone disease, and the laboratory testing may uncover an underlying medical condition that accentuates stone risk. The second key in evaluation is obtaining a stone analysis when available, as stone composition may guide further management. Lastly, the panel suggests a 24 hour urine collection to further identify and manage risk factors in all high-risk patients, as well as first-time and recurrent stone formers who are interested.

Diet Therapies

Since prevention and treatment can vary widely depending on stone type and individual risk factors, it is challenging to offer global advice to stone formers. However, some basic theories do

apply, and the new guidelines highlight these. First, regardless of stone type, all stone formers should aim to increase fluid intake sufficient to produce a minimum of 2.5 l of urine per day. Though this can be a difficult goal to meet, dilution of urine through increased fluid intake is the single most important component of stone prevention. Historically, patients with calcium stones were encouraged to reduce their calcium intake, however it is now known that this practice actually increases stone risk. Patients should consume a diet with the recommended daily allowance of calcium (1,000–1,200 mg/day) while limiting sodium intake ($\leq 2,300$ mg), as high sodium is associated with increased calcium excretion in urine. Last of all, calcium stone formers with low urinary citrate (a key inhibitor of stone formation) on 24 hour urine collection should increase their intake of fruits and vegetables and limit non-dairy animal protein.

Pharmacologic Therapies

The panel recommends offering thiazide diuretics and citrate in patients with recurrent stones with or without high urine calcium or low citrate. Thiazide diuretics, commonly prescribed for high blood pressure, are effective in reducing the concentration of calcium in the urine, and are generally well tolerated. If the addition of fruits and vegetables is not adequate to increase the levels of stone-inhibiting citrate, potassium citrate should be prescribed to reduce risk of both calcium oxalate and phosphate stones. Additionally, potassium citrate should be recommended in patients with cystine and uric acid stones as a means of raising urinary pH and reducing stone recurrence.

Follow Up

A number of strategies to monitor stone formers are recommended by the committee. First, a 24 hour urine collection within 6 months of starting therapy, as well as an annual 24 hour urine

collection thereafter to assess response to dietary and medical therapy. Second, in patients receiving pharmacologic therapy, intermittent blood testing is endorsed to screen for laboratory abnormalities (especially low or high potassium). Also, if stones recur, a repeat stone analysis should be obtained as a change in composition may require a shift in therapy. Finally, periodic imaging (x-ray, ultrasound, or low-dose CT) should be obtained to evaluate stone growth or recurrence, with interval based on severity of disease and clinical course. These approaches to follow-up enable providers to tailor and improve therapy, thereby reducing the overall risk for stone growth and/or recurrence.

In closing, new technology, the development of innovative animal models, and revolutionary medical and surgical strategies for stone treatment continue to advance the management of kidney stones, and the future holds many exciting new breakthroughs for both providers and patients.

References

1. Wisenbaugh ES, et al. Dual-energy vs conventional computed tomography in determining stone composition. *Urology*. 2014;83(6):1243–7.
2. Shah A, et al. Novel ultrasound method to reposition kidney stones. *Urol Res*. 2010;38(6):491–5.
3. Harper JD, et al. Focused ultrasound to expel calculi from the kidney: safety and efficacy of a clinical prototype device. *J Urol*. 2013;190(3):1090–5.
4. O'Connor AM, et al. Toward the 'tipping point': decision aids and informed patient choice. *Health Aff (Millwood)*. 2007;26(3):716–25.
5. Elwyn G, et al. Shared decision making: a model for clinical practice. *J Gen Intern Med*. 2012;27(10):1361–7.
6. Stacey D, et al. Decision aids for people facing health treatment or screening decisions. *Cochrane Database Syst Rev*. 2011;(10):CD001431.
7. Wills CE, et al. Treatment preference patterns during a videotape decision aid for benign prostatic hyperplasia (BPH). *Patient Educ Couns*. 2006;61(1):16–22.
8. Wilkes M, et al. Discussing uncertainty and risk in primary care: recommendations of a multi-disciplinary panel regarding communication around prostate cancer screening. *J Gen Intern Med*. 2013;28(11):1410–9.
9. Streeper N, et al. Patients prefer the use of a patient decision-making aid when discussing surgical options for nephrolithiasis. University of Wisconsin School of Medicine and Public Health: AUA meeting. Orlando: Florida. 2014.
10. Khan SR. Experimental calcium oxalate nephrolithiasis and the formation of human urinary stones. *Scanning Microsc*. 1995;9(1):89–100; discussion 100–1.
11. Poldelski V, et al. Ethylene glycol-mediated tubular injury: identification of critical metabolites and injury pathways. *Am J Kidney Dis*. 2001;38(2):339–48.
12. Khan SR, Glenton PA, Byer KJ. Modeling of hyperoxaluric calcium oxalate nephrolithiasis: experimental induction of hyperoxaluria by hydroxy-L-proline. *Kidney Int*. 2006;70(5):914–23.
13. Canales BK, et al. Steatorrhea and hyperoxaluria occur after gastric bypass surgery in obese rats regardless of dietary fat or oxalate. *J Urol*. 2013;190(3):1102–9.
14. Matlaga BR, et al. Effect of gastric bypass surgery on kidney stone disease. *J Urol*. 2009;181(6):2573–7.
15. Hofmann AF, et al. Complex pathogenesis of hyperoxaluria after jejunoileal bypass surgery. Oxalogenic substances in diet contribute to urinary oxalate. *Gastroenterology*. 1983;84(2):293–300.
16. Mandel NS, et al. A porcine model of calcium oxalate kidney stone disease. *J Urol*. 2004;171(3):1301–3.
17. Sivalingam S, et al. Dietary hydroxyproline induced calcium oxalate lithiasis and associated renal injury in the porcine model. *J Endourol*. 2013;27(12):1493–8.
18. Terris JM. Swine as a model in renal physiology and nephrology: an overview. In: Tumbleson ME, editor. *Swine in biological research*. New York: Plenum Press; 1986. p. 1673–89.
19. Miller J, et al. *Drosophila melanogaster* as an emerging translational model of human nephrolithiasis. *J Urol*. 2013;190(5):1648–56.
20. Hirata T, et al. In vivo *Drosophila* genetic model for calcium oxalate nephrolithiasis. *Am J Physiol Renal Physiol*. 2012;303(11):F1555–62.
21. Hirata T, et al. Ion and solute transport by Prestin in *Drosophila* and *Anopheles*. *J Insect Physiol*. 2012;58(4):563–9.
22. Rimer JD, et al. Crystal growth inhibitors for the prevention of L-cystine kidney stones through molecular design. *Science*. 2010;330(6002):337–41.
23. Sumorok N, Goldfarb DS. Update on cystinuria. *Curr Opin Nephrol Hypertens*. 2013;22(4):427–31.
24. Mufarrij PW, et al. The effects of oxazyme on oxalate degradation: results and implications of in vitro experiments. *J Endourol*. 2013;27(3):284–7.
25. Liebman M, Al-Wahsh IA. Probiotics and other key determinants of dietary oxalate absorption. *Adv Nutr*. 2011;2(3):254–60.
26. Al-Wahsh I, Wu Y, Liebman M. Acute probiotic ingestion reduces gastrointestinal oxalate absorption in healthy subjects. *Urol Res*. 2012;40(3):191–6.
27. Desai MM, et al. Robotic flexible ureteroscopy for renal calculi: initial clinical experience. *J Urol*. 2011;186(2):563–8.
28. Dogra PN, et al. Lower ureteral stones revisited: expanding the horizons of robotics. *Urology*. 2013;82(1):95–9.
29. Rosa M, et al. Recent finding and new technologies in nephrolithiasis: a review of the recent literature. *BMC Urol*. 2013;13:10.
30. Pearle MS, et al. Medical management of kidney stones: AUA guideline. *J Urol*. 2014;192(2):316–24.

David A. Schulsinger

Over my 16 years of practicing Urology, I have encountered, managed and treated many stone patients, each with their own unique stone experience. In this chapter, I share with you some of these special stone scenarios.

1. ***How I missed my son's wedding!***

I would like to share my story about my stone that has taught me more than 1 lesson in life. I have always been a procrastinator. Never did I think that this personal trait would impact my ability to make it on the big day.

I had a stone that was small, but too large to pass. My urologist told me that it needed to be treated with surgical intervention. Like many things in life, I took it under advisement, but was not quick to act on it. I had many things on my mind including my son's wedding. There was much to do and prepare for the big day. Unfortunately, I was working on other things at the expense of my own health.

On the weekend of my son's wedding, I had a severe colic attack, unfortunately, with fever and chills. The emergency treatment for this stone was NOT the same for the elective procedure. The elective procedure was to have a lithotripsy procedure without a

stent. The emergency procedure was to have a tube in my back, called a nephrostomy tube, followed by a second procedure to remove the stone. The time between these procedures were spent in the hospital receiving IV antibiotics. This, unfortunately, included the day of my son's wedding. On that day, I participated in the wedding from my hospital bed listening to the ceremony service from my phone. The flowers in my room did not match the arrangement of flowers at the wedding.

I learned a very valuable lesson that day. I may be a procrastinator, but I will never be late again.

2. ***How stones may fly!***

I was 73 years old when I visited Germany. Looking back, I should have "listened" to my body's symptoms of flank pain, recurrent infections, occasional nausea and vomiting.

My flight to Germany was uneventful. There, I began to experience fever, tiredness and fatigue. By the time I got to my hotel room, I was in septic shock. That was the last memory of my trip. I was told that I was sent by ambulance to the local hospital where I was placed on a respirator, IV fluids, IV antibiotics and medication to maintain my blood pressure.

After several days, I was more stable. I was then airlifted from Germany back to the States. I remained on a respirator for almost 1 month before I could breathe on my own.

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

After additional antibiotics, Dr. S then treated me for my stones. In retrospect, it was hard for me to believe that my kidney stone cut my trip short, let alone almost cutting my life short. My stone, however, racked up some frequent flyer miles!

3. ***Stones and My best golf game!***

My most satisfying round of golf came from a kidney stone. While on a golf trip with my three best friends, I awoke to the pain of a kidney stone in the middle of the night. I was taken to the ER and diagnosed with an obstructing kidney stone. Surgery was scheduled the next morning. I was discharged on Sunday and just made it to my tee time. The pain still emanating from my flank was enough of a distraction to help me shoot one of my best rounds of golf ever. Normally my head is full of a dozen swing thoughts while driving the ball, however, this day it was different. There was no swing thought in my head, I was only focused on my pain. I swung the clubs with no thought at all, I just “let it fly” as they say. When I play today, I still try and remember that day. I tell myself to clear my head and let it fly. If all else fails, I could always pray to the golf gods for another kidney stone!

4. ***Choosing the right treatment!***

I am a 51-year old male with a well-established history of stones. I also own a lithotripsy company that treats these stones. As a lithotripsy machine owner, I move machines to places where patients need to be treated for stones. As a stone patient, my stones move to remind me that they can cause pain. Over the years, I have passed many stones, both smaller and large. Recently, however, I passed a stone that was too great in size to pass. I knew that this stone required treatment by surgical intervention. The question to me was “by what method?” I know that there are interventions that are more invasive than ESWL, however, I was trying to avoid this procedure. Nonetheless, I treated myself with one of my machines. After multiple minimally invasive procedures, multiple trips to the ER, pain medication and lost work time, I knew the

best treatment option for me was a more invasive procedure. As a lithotripsy company owner, you need to respect that at certain times you are a patient too. I am not only the stone club president, but I am a patient too!

5. ***Stones and pregnancy should not be used in the same sentence.***

It was a surprise and a joy to find out that we were pregnant with our first at age 43. It was also a surprise but not so joyous when I needed a nephrostomy tube at 3½ months into the pregnancy. To be honest, I have dealt with kidney stones for many years prior to pregnancy, and while most were very uncomfortable, they were actually manageable. However, there were some situations that entailed curling up into a ball on the floor of the bathroom and praying to g-d to take the demon out of me, throwing up in the emergency room and taking Vicodin for a couple of weeks until it passed. Thank g-d for pain meds! Being pregnant with kidney stones is a whole different ball game. The option of a tube inserted into your back, snaked down into your kidney draining into a bag filled with urine attached to your thigh, sounds better than pain. My Louis Vuitton bag is hanging over my right shoulder while my lemonade colored “satchel” is filling up on my left thigh; very fashionable! From what I understand, kidney stones and pregnancy are not uncommon. They are still studying this phenomenon, but if you have them already and plan on getting pregnant, fasten your seat belt and get ready for the ride! Normally a nephrostomy tube needs changing about every 6–8 weeks, but in my case it was every 2 weeks and I had 10 of them to date. Lucky me!! While I have no intention of sounding frightened, one tube change was one too many!

With all said and done, I still am fortunate to have had a child at 43, survived the pain of stones through pregnancy, dealt with the litany of tears from tube changes and have grown to appreciate the benefits and limitations of pain medication; I now only look forward to the giggles and days and nights filled with numerous diaper changes ahead.

Life is full of surprises, from the pain of changing tubes to the baby cries and reminders of lactating boobs!

6. ***My first stone at 4 years old and why I became a nurse!***

I am a frequent stone maker, having made stones since the age of 4. As a child, I made almost 1 stone every year. By the age of 14, I had 14 stones. The first time I was treated for a stone, I was very nervous.

Over the years, I became very comfortable with my procedure and my physician.

Entering college, I thought about what I wanted to be when I grew up. Instinctively, I decided on nursing and chose a career to help others the same way that they helped me.

7. ***The forgotten stent!***

Five years ago I had a stent placed for an obstructing 5 mm stone in my ureter. The pain was relentless and stone was unforgiving. I was told to follow up with my urologist so that I could plan the next and final part of my procedure. After the stent was placed, I felt 100 %. Because my stone was no longer bothering me, I figured I would not bother with it. Unlike cardiac stents that are permanent, urinary tracts are not. I was later told they should be removed in a year or less.

Needless to say, 5 years later I began to experience pain, bleeding and recurrent infections, a follow up X-ray revealed that I had a 3 cm stone in my bladder attached to my stent, a 4.2 cm stone attached to my stent in the kidney. The stent was calcified in the ureter and the 5 mm stone in the ureter was now 7 mm. I need to have one procedure to remove the stone in my bladder, another procedure to remove the stone in my kidney and a third procedure to remove the stent. In retrospect, it was very clear to me that three procedures is not better than 1. I will not forget about my stent next time.

8. ***I have prostate cancer and a kidney stone-what do I do?***

At age 60, a routine physical exam and lab testing revealed hematuria and an elevated PSA test. After some additional tests and a biopsy, my physician told me that I had prostate cancer and a kidney stone.

While I was uncertain as to which would be treated first or together, my physician explained to me that one condition was potentially life threatening and one was a nuisance. I always thought that a kidney stone was life threatening, but my Urologist informed me that was not the case. Needless to say, I had my prostate surgery first and subsequently had my stone removed.

9. ***Stone for thought!***

Dr. Maurice Gonder was the founding Chairman of Urology at Stony Brook University Hospital and a pioneer in the treatment of prostate cancer using cryosurgery. He had seen many patients over his career and treated many urologic diseases, some life threatening. He was a man of many quotes and words of wisdom. He often said, "50 % of knowing urology was to use KY jelly!" Over the years, he treated many patients with kidney stones and knew of the pain that patients suffered. His most memorable and profound words of wisdom, however, was, "*I do not wish my enemies harm, just a kidney stone!*"

10. ***My kidney stones: There is no magic pill, but there is philosophical "food for thought!"***

Before I became a kidney stone patient, my experience with physicians and medicine was limited to that of most young people. An occasional well visit, some routine blood work to check to make sure nothing was outside of the "norm", an antibiotic for one thing or another, and chicken pox and a fractured leg from falling out of a tree when I was a kid. Like most, I went to the doctor because he or she was the definitive healer. The doctor always seemed to know exactly what pill or treatment was going to fix my problem and what I needed to do to avoid the problem in the future.

Then I had the misfortune of experiencing my first kidney stone. After the trip to the emergency room and the passing of that horrible little chunk of calcium and oxalate, I made my first of what has now been countless trips to the urologist with the delusion that he would give me the magic pill that would assure that this would never happen again.

What I've learned now though, as a middle-aged person with a recurring disease, is that some medical problems are easier to deal with and better understood than others. As an individual who has recently graduated from youth to middle age (at least in my own mind) this has come as quite a revelation to me. It is hard to make the mental transition that some medical conditions can be cured while other can only be managed. I have also learned that although the practice of medicine has come a long way in the last 100 years, many patients and diseases are treated on the basis of statistical probability rather than absolute certainty, and the treatment of kidney stones is a disease that best exemplifies the uncertainty that exists in medicine even in this modern era of medicine.

Since I work in the healthcare industry, I have had the good fortune of interacting each day with doctors and nurses who come from varying walks of life and come from diverse backgrounds both medically and culturally. One of the doctors with whom I have regular dealings with is from far eastern dissent. Although he has been in practice for almost 40 years, and in the United States for nearly 30 of those years, I have learned a lot from his philosophical approach to his profession which I believe is rooted in his cultural beliefs and experiences. During a recent conversation he asked me how I was doing with a medication I had just begun taking for the treatment of my stones. I told him I had discontinued taking it because of an uncommon side effect. His response to me was a much a philosophical commentary on the practice of medicine as it was a practical guide to the way people unknowingly live their lives each and every day. This long practicing physician said, "*I've learned in my practice, over the years, never to use the word never because even the most time tested medicines and treatments can have an uncommon result in certain patients.*" He then went on to offer me a philosophical thought that I think came more from his subconscious and was more a commentary on his dealings with today's

young doctors in contrast to him as an older, wiser and more traditional doctor. He said, "*I can always tell a young inexperienced doctor from an older student of the profession. The inexperienced physician speaks in more absolute terms while we older guys know there is always the possibility of some unexpected outcome.*"

My conversation with this doctor, which lasted only 3 min, opened the door to a world in which most patients only acknowledge in the deepest part of their subconscious. That is, as it is also in life itself, that most parts of the world of medicine are nothing more than an exercise in statistical probability. The better the research and understanding of the disease and the better and more competent the physician whose care you are under is trained, the better your statistical chances are for successful treatment. Of course, *a little luck never hurts anyone either!*

11. ***Stones and mother-in-laws!***

My kidneys and I have always had an adversarial relationship. I first became aware of my kidneys at age 11. I was involved in a sledding accident and taken to the hospital. The next thing I know, I am in a hospital bed with bandages all over me and tubes coming out of every part of my body. I was told my left kidney had been removed. My first question, what is a kidney? I would soon come to learn of its many functions and uses.

Fast forward 15 years later. A horrible pain awakened me. I immediately knew the pain was imitating from the kidney. I had only experienced that type of pain once before and I had not forgotten it. My wife drove me to the ER where I was informed I had a kidney stone. Many hours later I passed the stone. I could not believe how something so little could cause so much pain. Thankfully it passed, as I would soon learn, that is not always the case.

In the last 20 years since my first stone, I have a dozens more. Some stones have passed on their own, but may have needed a little help. I have had stents placed, lithotripsy and a nephrostomy tube placed. On

the bright side, I have managed to maintain normal daily activities and work. I remind myself, kidney stones are not fatal, just a horrible and painful inconvenience. I tell people that stone are like in laws: they arrive unexpectedly, stay too long, are a pain to get rid of and it is great relief when they finally leave!

12. ***As a Fireman, I not only put out fires, but kidney stones too!***

I am an 83-year old softball player and a volunteer fireman. As a fire fighter, I have put out many fires over the years. Water was our best friend.

Over the years I have encountered several stone. Like fires, they never happen during opportune times. Fortunately, all but one of my stones passed with hydration alone. One even passed while rounding second base during a game. The key to passing these stones was hydration, hydration and hydration.

I learned from my stone experiences that water is used to “put out more than just fires!”

13. ***Don't let a stone stop you from your dream!***

I am a tri-athlete, but my adventures are much greater than that. I received a heart transplant several years ago. I received a 24-year old heart and a new lease on life. I was back to doing triathlons 1 year later. Prior to doing the NY Triathlon I presented with a symptomatic stone. Dr. S treated my stone and I was on my path doing another triathlon. *Don't let a stone get between you and your dream!*

14. ***Father like daughter!***

I had stones most my adult life. On the day that my daughter was born, I could see that she looked a lot like me, but never knew that we would have more than looks in common. We would often joke about each other's stones. “My stone is bigger than your stone” or “I had more stones than you this year” were common phrases in our house.

Four years after my daughter passed a stone during her 9th month of pregnancy, she experienced that intense, labor-like pain once again. Only this time, she was not preg-

nant. This time was unique, as I had another stone myself, the first time we had stones together. I told my daughter that “I caught it from her.”

One day, however, the unthinkable happened. Both my daughter and I not only had stone attacks at the same time, but Dr. S did our surgeries on the same day. There was only one issue; we had to find a third person to drive us home that day!

15. ***Can Stones Break your Bones?***

Ms. OP is a 69 year old female with known history of a broken toe and femur on separate occasions. A bone density scan revealed that she had osteoporosis. Her history is complicated by numerous passed renal calculi. Stone analysis revealed calcium oxalate stones. The patient was puzzled by the fact that the rheumatologist insists that she begin calcium supplements at the same time she is producing calcium oxalate stones. Fortunately, her 24 hour urine revealed normal urinary calcium levels and low urinary citrate. She was put on Calcium citrate to help minimize her risk for osteoporosis and renal stones. Repeat 24 hour urine 6 months later revealed normal urinary calcium and citrate. Remember, when comparing osteoporosis and stone disease, brittle bones can be life threatening and stones can be a nescience. It is much easier for your urologist to handle your nescience over your brittle bones!

16. ***Differences between stones and childbirth!***

As a woman of three children and having recently passed my 3rd stone, I had some time to reflect on my “Brady Bunch!” I learned to appreciate that while there are some obvious similarities between kidney stones and delivering a child, there are some very important differences. Both kidney stones and labor can bring you to the hospital. Both stones and delivery can be done “naturally” (conservatively) or by surgery. Stones and childbirth can both be painful, however, many of us women would argue that stone pain trumps childbirth pain.

However there are some very important differences. If you pass a stone in the ER, as I have, you want nothing to do with that stone. In fact, I had no issues leaving the stone behind at the hospital. I encouraged my physician to take it away. In addition, it may also take several weeks before that stone ever gets a name. The names are usually very similar like calcium oxalate or calcium phosphate. Also, mind you, it's your physician that usually provides the name and tells the mother.

On the other hand, when you give birth, you are overwhelmed with excitement on bringing your bundle of joy home. There are many different names to choose from. You usually come up with a name in the hospital and the mother typically tells the physician.

The most important difference between the birth of a stone and the birth of your child is what happens over the months and years to follow. A year or 2 after you deliver, I could say to myself, "I believe I would like to have another one." After passing a stone, however, I could honestly say that I cannot say the same!

17. ***Stones and Rushing a Fraternity***

I was a freshman in college and there were lots of firsts: my first semester of college, my first time away from home and excitedly, it was also first time that I pledged a Fraternity. I first became symptomatic with this stone several weeks before and the doctor encouraged me to drink beer to pass the stone. The timing was perfect! It was now Hell Week and the brothers insisted on pledges wearing only burlap sacs for under garments without showering or shaving. It then happened. I was keeled over with the worst pain of my life. I was rushed to the ER where I was greeted by my mother. She looked at me in a disheveled state noticing my new attire and unshaved and poignant odor state, and said, "what are they doing to you at school?" The next day was my scheduled surgery. That night I lay in my Hospital bed under some heavy medication as I contemplated my fate. I questioned whether I wasted a semester? Would I have to pledge again? Would I be a freshman again? I

prayed to the Stone G-ds if in any way this stone could pass.

It was 6 am and I felt the scud missile exiting my body hitting the porcelain toilet bowl. My trophy was a stone the size of half my index finger, multi-colored with jagged edged borders. There it was in all its glory. A stone the size of half my index finger! The Stone G-ds had heard my plea! I quickly rushed into the hall, shouting to the entire hospital, "I am Stone free!" I now had additional questions: Would I have to go through Hell night alone? Would I have to pledge in the spring?

The Fraternity brothers had a vote and determined that my Hell night, while different from what they planned, was successful and I became a Fraternity brother. I was the first pledge to skip Hell Week, the first pledge to skip Hell night and the first to become a brother passing a stone. This was my first stone and an experience I would never forget. After passing my stone, I had a new appreciation for the word "rushing!"

18. ***A Mars rock it was not!***

I love rocks, don't get me wrong. I study rocks. I am a geologist. I study how rocks form on the Earth, on the Moon, on Mars, and on asteroids, secure in my position as an observer, never as a creator. But now I have had the ultimate experience: I not only created my own rock, but felt the pain of birthing it. This is my story.

This wondrous event occurred in Pasadena, California, about as far from my lovely home and adoring family in New York as I could get. But before this, like the foreshocks of an earthquake, I felt something was amiss. It started innocuously enough – a bit of discomfort in the bladder area, a bit of urinary urgency. But it precipitated more panic than it should have as it was a mere 3 days before I was to leave for a conference about Mars, a conference where eager planetary scientists would be assembled to hear about the rocks that the Curiosity Rover was sampling. So I rushed first to my medicine cabinet grabbing the last few amoxicillin capsules left over from my dog's last infection in case I could not get an appointment. But to my infinite relief, my primary care doctor was able to fit

me in, and found only some blood in the urine, no bacteria. I was perfectly happy, relegating the discomfort to one of the oddities of the human condition that would resolve in time. So I motored on blissfully unaware of what this discomfort portended, and got to Pasadena. But that Monday, about 5 days after the first discomfort, I woke up with what felt like a back spasm in the left flank. I remember mainly feeling puzzled and chalked it off to the long plane ride and it did go away. Other than noticing that the seats were particularly uncomfortable for my back that day, I gave my talk and all was fine until late afternoon. I was sitting at a table discussing the origin of Martian rocks with a colleague when suddenly I was hit by breathtaking pain in that same left flank in which I had had the “muscle spasm” that morning. I tried to keep a calm face and gingerly made my excuses to catch the first shuttle bus back to the hotel. By the time I got into the bus, I was wondering how I would stand the ride. Since walking upright was brutally painful, I sat in the closest seat to the door and to my great chagrin a reporter sat down to interview me about my talk. I was able only to croak that I study Martian rocks – as if he didn’t know that -but that was all I could muster before retreating into a silent trance with a mantra running over and over in my mind...let it be quick...let it be quick...let it be quick...feeling the need to deal with this pain alone, like a dog crawling under a table to lick his wounds.

In the hotel room I downed 6 Advil’s and stood hunched over in the hottest shower I could stand, baking that flank. When it went on for 2 hour, I started thinking about an ER, but the pain was so bad that I didn’t think I could handle the ambulance trip. Once the ibuprofen kicked in, however, I was lucid enough to give myself an ultimatum...if Pasadena had a local hospital I would get an ambulance, but if my only choice was that I had to go to some inner city LA hospital and sit in a waiting room for hours as the gunshot victims came in, I would rather drown, baked red in the shower. Fortunately there was a community hospital right there in Pasadena.

Step 1 was done. The second step was self-diagnosis and assessment if I needed an ER. So I then did a google search and figured out that either it was a kidney stone or adrenal apoplexy, neither of which I could handle with the few amoxicillin tablets I stole from my dog. So bent over in pain and tossing my cookies several times en route, I made my way downstairs to the concierge desk and croaked out my request for an ambulance – the absolute terror on the man’s face is something I will never forget. I then lay down on the nearest couch to just wait. Through a haze of pain, I remember, the stretcher and the ride across the “craters of the Moon” otherwise known as the roads in Pasadena. I got to the ER and was asked right away whether I was able to have morphine. Since I cannot, they asked ME what I wished for to control the pain. So I said what any reasoning person at that stage would, “Euthanasia please, but make it quick.” Instead they gave me more NSAIDS and left me to moan in peace. By the time I had an ultrasound, however, I was feeling better, and a couple of hours later I was pain free! The sweetest words I ever heard...“you have passed it, a lovely new kidney stone!”

So here I am, fully recovered, with only post-traumatic stress syndrome to deal with, as I think about the next airplane ride, the next conference, the next time when I am somewhere where there is no medical help, and the cold sweat appears on my brow as I imagine... another kidney stone is born.

Summary

In summary, each time I see a patient in the emergency room or in the clinic, I am enthused with delight to counsel on managing their stone disease. No 2 stone patients or their stones are the same. Treating these patients has been a learning experience and a process in evolution for both them and me. I thank those of you who have shared their stories and experiences to help others recognize and relate to their stone disease. Remember, *behind every kidney stone, there is a story!*

David A. Schulsinger and Michael A. Ferragamo

When it comes to billing and coding, it's all about YOU, the patient! The physician does the billing for your office visit, your procedure, and post-operative visits. However, there are important points for you, as the patient, to understand and facts that you should be aware of in the billing process.

There are several terms that you should be aware of in the billing and coding process:

ICD-9: *Playing by the Numbers!*

International Classifications of Diseases (ICD). ICD codes are alphanumeric designations given to every diagnosis, description of symptoms and in some instances, cause of death attributed to human beings. Currently, the codes we see in the United States today are version 9, called **ICD-9** codes. The paperwork we receive when we leave a doctor's office will contain ICD-9 codes to describe why that service was provided.

For example, if you present to the ER with flank pain, you may undergo testing that also determines that you have a right kidney stone with hydronephrosis. When you leave the hospital, you will receive a summary sheet that will have

diagnostic codes for renal colic (788.0), renal stone (592.0) and hydronephrosis (591).

New ICD-10 Coding System

ICD-10 is on the horizon to replace the current ICD-9-CM diagnosis code reporting. The numeric ICD-9-CM codes for renal or ureteral stones (592.0 or 592.1) with which you are currently familiar will be replaced by an alphanumeric system. Implementation for ICD-10 is underway and is in the testing stage in Australia at this time. The new diagnosis coding system will require more specific documentation from practitioners and further clinical knowledge for the coding/billing staff.

CPT: *The Numbers Tell the Story!*

Current Procedural Terminology or CPT® (registered trademark of the American Medical Association), is the listing of descriptive terms and identifying codes for reporting medical services and procedures performed by physicians. These codes are numbers that describe every task and service a medical practitioner may provide to a patient, including medical, surgical and diagnostic services that a patient receives. The information collected ensures uniformity of medical services and procedures among physicians, coders, patients, accreditation organizations, and payers for administrative, financial and analytical purposes.

D.A. Schulsinger, MD (✉) • M.A. Ferragamo, MD, FACS
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com; liqgold2@aol.com

For example, a patient is undergoing a percutaneous nephrolithotomy procedure for a stag-horn calculus. If the stone is smaller than 2 cm, you will label this as 50080, and if it is a larger stone (>2 cm), it will be categorized with CPT code 50081.

Global Period: *The Numbers Are In!*

All procedures on the Medicare Physician Fee Schedule are assigned a Global period. A “global fee period”, also known as a surgical aftercare period, is defined as the period during which office visits for the postoperative period following surgery are included in the fee for surgery and not separately billable.

There are three types of global periods based on the number of postoperative days:

- **0-Day postoperative period:** 0 days for endoscopies and certain minor procedure for which the physician visits the patient on the same day as the procedure.
Example: Ureteroscopy has a 0 day global period.
- **10- Minor Surgery:** 10 days for most minor surgeries.
Example: a circumcision has a 10 global period.
- **90-Major Surgery:** 90 days for major surgeries. To determine the global period for major surgeries, count 1 day immediately before the day of surgery, the day of surgery, and the 90 days immediately following the day of surgery.
Example: PCNL or ESWL procedures have a 90 day global period.

Modifiers: *It's All About the Numbers!*

A modifier provides a way to report that a service or procedure has been altered by some specific circumstance but not changed in its definition or code. Modifiers are used to indicate that services were performed bilaterally, professional or technical in nature or performed by more than one surgeon. Also, that a service or procedure was unusual, i.e. increased or reduced by specific circumstances.

Documentation is also important when appending Modifiers to CPT codes whether they

are surgical or medical. The following are modifier examples:

Modifier 22- Unusual Procedural Service

This modifier should be used when the service(s) provided is greater than that usually required for the listed procedure.

With this modifier, the physician would need to document, for example, that extra work during a procedure was provided. For instance, Procedures that take an unusually long time to perform due to obesity, dense adhesions or anatomical anomalies would require modifier 22 for proper payment.

Modifier 24- unrelated evaluation and management service by the same physician during a postoperative period

A physician may need to indicate that an E & M service was performed during a postoperative period for a reason(s) unrelated to the original procedure.

For example, a patient in a global period for an ESWL comes into the office for a penile lesion. Modifier 24 would be appended to the E & M code and it would be linked with the diagnosis with the reason for the visit, penile lesion.

Modifier 25- Significant, separately identifiable evaluation and management service by the same physician on the same day of the procedure or other service

A physician may need to indicate that on a day a procedure or service is performed, the patient's condition required a significant, separately identifiable E & M service above and beyond the usual pre-op and post-op care associated with the procedure that was performed.

A patient has a vasectomy in the office. This patient returns the same day with chief complaint of hematuria associated with renal stones. The vasectomy would be reported as the procedure and the E & M service code would be reported with modifier appended to it.

Modifier 26- Professional Component-

Certain procedures have both a professional and a technical component.

A surgeon performs retrograde pyelogram during a stone procedure. The radiological interpretation of the imaging study would be reported with a modifier-26 for the professional

interpretation. The physician would also bill for the technical component of the procedure.

Modifier 50- bilateral procedure

A urologist does a surgery that involves procedures on both sides (ie, kidney, testis, etc.).

A bilateral ureteroscopy and laser lithotripsy with stone manipulation would be reported with the modifier-50.

Modifier 52- Reduced services

In some circumstances a service or procedure is partially reduced or eliminated at the physician's election. The service may be identified by its usual procedure code with the addition of modifier 52 to signify that the service is reduced. This provides a means for reporting reduced services without disturbing the identification of the basic service.

A surgeon attempts to perform a cystoscopic ureteral stent insertion in a patient with an obstructing ureteral stone. After multiple unsuccessful attempts, the surgeon discontinues the procedure. This partially incomplete service would be reported with a modifier-52.

Modifier 53- Discontinued procedure

Under certain circumstances a service or procedure is terminated at the surgeons discretion due to extenuating circumstances or those that threaten the well-being of the patient. It may be necessary to indicate that a surgical procedure or diagnostic procedure was started but discontinued.

A surgeon is performing a percutaneous nephrolithotomy procedure for a staghorn calculus. During the procedure, the patient develops cardiac arrhythmia and the procedure is terminated. The procedure is reported with a modifier-53. The fee should be reduced at the discretion of the surgeon.

Please note that modifier-53 differs from -52 in that the patient's life threatening circumstance precipitated the terminated procedure.

Modifier 57- Decision for surgery

Modifier 57 is used to indicate that an E & M service performed that resulted in the decision to take the patient to surgery.

The urologist is called to the Emergency Room to consult on a patient who sustained multi-trauma from an MVA. The urologist decides

to take the patient to the OR for repair of a ruptured bladder. The E & M service level would be reported with modifier-57 appended to it.

Modifier 58- Staged or related procedure or service by the same physician during the postoperative period

This modifier may be reported if a procedure performed in the postoperative period was:

- Planned prospectively at the time of the original procedure (staged)
- More extensive than the original procedure
- For therapy following a diagnostic surgical procedure

An example of a staged procedure is removal of an indwelling ureteral stent (using modifier-58) within the global period of an ESWL/stent insertion.

A similar scenario would be the ureteral stent insertion for a patient with renal stones. The surgeon performs an ESWL, which fails and then at later date within the global period performs a PCNL, which finally resolves the stones.

Modifier 59- Distinct Procedure Service

This indicates that a service was distinct or independent from other services performed on the same day.

A urologist performs an emergent stricture dilation in the morning and then performs a more extensive surgery in the afternoon. The modifier -59 would show that these were two distinct procedures. Otherwise, the dilation would be bundled into the more extensive surgery.

Modifier 76- Repeat Procedure by Same Physician

This modifier is used when the urologist needs to indicate that a procedure or service was repeated subsequent to the original procedure or service.

Example: A urologist performs a ureteroscopic, laser lithotripsy and stone extraction for a ureteral stone. Patient later that day develops intractable pain and a second stone was found on CT scan. Patient requires a second identical procedure later that day. Coding for that second identical procedure requires a Modifier 76.

Modifier 78- Return to the operating room for a related procedure during the postoperative period

The surgeon may have to indicate that another procedure was performed during the postoperative period (global period) and related to the initial procedure. Most often, this represents a return to the operating room within a global period of an initial surgery to treat a complication of this procedure.

The patient's surgical site bleeds after the initial PCNL surgery and returns to the operating room to control the hemorrhage. The procedure would be reported with modifier-78, for exploration and control of the postoperative hemorrhage.

Modifier 79- Unrelated procedure or service by the same physician during the postoperative period

The surgeon may have to indicate that a procedure provided during the postoperative period was unrelated to the original procedure.

The surgeon performs a laparoscopic right cyst aspiration and within the surgical global period performs a left cyst aspiration. This would be reported using modifier-79 to indicate that the procedure was unrelated to the initial procedure.

Another example, a patient returns to the operating room for an ureteroscopy, laser lithotripsy and stone manipulation within the postoperative period for a radical prostatectomy. The ureteroscopy, laser lithotripsy and stone manipulation would be reported with modifier-79.

E & M Services: The Numbers Speak for Themselves!

Evaluation and management (E & M) services **those services provided by physicians and non-physician (i.e., NP, PA, etc.) practitioners to evaluate patients and to manage their care. The code is chosen based on where the service is performed (i.e., Physician Office, Outpatient Hospital, Inpatient Hospital, Outpatient Ambulatory Surgical Center, Emergency Room-Hospital), the extent of history taken, the**

extent of the examination and the level of medical decision-making process.

Deductible: The Numbers Are Fixed!

A deductible is the amount money you will pay for health care services before your health insurance begins to pay.

For example, if your deductible is \$5,000, you would pay 100 % of your health care charges until the amount you paid reaches \$5,000. After that, some services you receive may be covered at 100 %, or you may have to pay a coinsurance.

These are some other terms you may see associated with deductibles:

- After deductible: This lets you know that the insurance provider will start sharing costs with you for a service after you have met your deductible.
- No deductible: You don't have to pay toward your deductible for this service. You'll still have to pay any copays.
- Before deductible: We cover this service before you've met your deductible. You'll still have to pay any copays.

Coinsurance: Crunch the Numbers!

Coinsurance is your share of the costs of a health care service. It's usually figured as a percentage of the total charge for the service. You pay coinsurance plus any deductibles you still owe.

For example, you met your \$5,000 deductible and your coinsurance is 20 %. For a \$100 health care bill (i.e., follow up office visit), you would pay \$20 and your insurance company would pay \$80.

Copay: The Numbers Don't Lie!

A copay is a fixed amount that a patient pays for a health care service, usually when you receive the service. The amount can vary by the type of service. You may also have a copay when you get a prescription filled.

For example, a doctor's office visit might have a copay of \$30. The copay for an emergency room visit will usually cost more, such as \$150. However, there is a maximum amount you will pay for coinsurance and copays, called the coinsurance and copay maximum.

Important Questions and Answers that All Patients Should Know

How does the global period apply to me?

Depending on the type of stone procedure that you have will determine the type of global period that you will endure. For example, if you had a PCNL for a large stone or ESWL for a small stone, there will be a 90-day global period. That means that if you see your physician during a 90-day period following your surgery for a follow up visit, this would fall within the global period and there would be no charge for an E&M visit. On the other hand, if the patient underwent a ureteroscopy and stone manipulation procedure, there is a 0 day global period and the patient would be charged for any postoperative period visit.

How does the deductible apply to me?

A deductible is the money that the patient pays which is separate from the premium fee. The patient will have a deductible for their office visit, laboratory tests and imaging studies; a separate deductible for your stone procedure. When signing up with your insurance policy or exchange, know what your deductible fees will be.

How does the copay apply to me for my office visit?

Depending on your insurance carrier or your exchange in the affordable care act, following complete payment of your deductible, you may have a copay to pay for your office visit. All patients are required to pay this copay.

How does the copay apply to me for my procedure?

Depending on your insurance program or the exchange you signed up with the affordable care act, there will be a separate copay associated with your procedure and/or medication. You are required to pay this.

How does cost sharing apply to me?

Cost sharing applies to your deductibles, coinsurance and copay. How they work together will allow the patient to make the correct choice when selecting a health insurance plan.

How do I decide on a health care plan?

To establish a strategy on choosing a health care plan, it is important to evaluate the premiums, deductibles and coinsurance. At this point, you can decide which deductible, premium and coinsurance mix is the best option for you and your finances.

For example, if you visit your physician often, you might want to choose a plan that has low copays. For instance, you might choose a plan that has a higher monthly premium but smaller copay for doctor's office visits.

On the other hand, if you don't visit the doctor often, copays will probably be less of a concern when you are choosing the best coverage plan.

How does my patient information remain protected and secure?

HIPAA (Health Insurance Portability and Accountability Act of 1996) Privacy Rules and HIPAA Security work in tandem and manage how we handle patient information. HIPAA Privacy Rules cover how we can use and disclose patient information. The HIPAA Privacy Rule establishes national standards to protect individuals' medical records and other personal health information.

HIPAA Security Rules provide standards for safeguarding and protecting electronic patient information. HIPAA Security Rules are designed to protect electronic patient information and at the same time permit the appropriate access and use of that information by the people who need it for treatment, payment, and health care operations.

Conclusion

In summary, billing and coding is a complex process with a continuum of changing codes, modifiers and other parameters that requires coder specialists to understand and interpret as it

continues to evolve and change. It is important for you to know that these terms and information exist. When you leave a health care facility, the paperwork you receive will contain both CPT codes to describe the service that was rendered for billing purposes, and ICD-9-CM codes to describe why that service was provided. The patient should be aware that there are different costs for different part of the work up, surgery and postoperative appointments. Be aware that certain procedures include a global period that when a patient is seen during this time, there will be no charges for office visits.

There will be a new set of codes, called ICD-10, scheduled to be released on October 1st, 2015. These codes will provide much greater detail than the existing codes with ICD-9. ICD-10 will provide diagnostic details along with laterality (i.e., side) and location within the urinary tract. Codes will be very specific in describing well-known illnesses in details. Currently, in the ICD-9 system, there is a code for

renal stones. The new coding system will tell you not only that there is a stone, but also the location in the urinary tract where the stone is located and the side. There are not only specific codes for stones in the kidney, ureter and bladder; you can also find a code for a spacecraft crash injuring an occupant (V9542XA). For patients who get hurt walking into a lamppost (W2202XA), possibly concentrating on their cellphone, there is a code to cover that. Also, if you happened to be bitten by a turtle (W5921XS), sea lion (W5611XD) or especially attacked by an Orca whale (W5621XA)...**there are codes for that too!**

Recommended Reading

1. Kurac JH. The building blocks of evaluation and management coding. 6th ed. E & M Coding and Documentation Guide, Ingenix, Inc.; 2003. p. 7–29.
2. Center for Medicare and Medicaid Services. www.cms.hhs.gov/medlearn/qrfc.asp#edu.
3. Current procedural terminology. Professional ed. American Medical Association; 2004.

David A. Schulsinger

The Affordable Health Care Act and Your Kidney Stone: *The “Hard” Facts!*

The Affordable Health Care Act (ACA), commonly known as ObamaCare, is the new national health care law that was signed into law on March, 2010. Since becoming the law, the Patient Protection and Affordable Care Act (ACA) has altered insurance industry practices, expanded certain Medicare benefits, and initiated new payment and service delivery models. This law went into full effect on January 1, 2014. As of October 1, 2013 consumers looking for health insurance were able to turn to state-based health care exchanges. The exchanges are the centerpiece of the Affordable Care Act intended to help the uninsured and small businesses find affordable coverage. Many people who have had to put off seeking medical care because they could not afford it may finally get the care they need. Additionally, doctors may get paid for services that they have provided for free to uninsured patients. On the other hand, ObamaCare has resulted in the increase in Insurance premiums, an increase hiring of part-time workers and incentivizing businesses to scale back benefits.

D.A. Schulsinger, MD
Department of Urology, Stony Brook Medicine,
Stony Brook, NY, USA
e-mail: endourology@yahoo.com

While it is debatable whether this law is positive or negative, Obama Care is the future of health-care and it is here to stay. The healthcare law is over 2,500 pages and the complete details of this law are beyond the scope of this chapter. This chapter, will however, tell you the pluses and minuses of this law and how this affects you and your kidney stone.

There are many questions regarding the ACA. What is the impact of ObamaCare on you and your family? How will the ACA influence the health insurance industry over the next few years? What are the different plans? What kind of health care does a patient receive? Should individuals comply with the new federal mandate to purchase health insurance starting January 1, 2014, or pay the tax instead? This chapter will provide insight to help to answer some of these important questions.

There are ten health services that must be covered by every plan sold to patients. This includes:

1. Care at doctors office
2. Emergency services
3. Hospital care
4. Pregnant mother and baby care
5. Mental health and addiction treatment
6. Prescription drugs
7. Rehabilitation and skill development services and devices
8. Lab services
9. Prevention and wellness services and Chronic disease management
10. Dental and vision care for children

Most health insurers will offer plans for individuals in five separate categories: This includes catastrophic coverage and four metal tier programs. The metal tiers are associated with the costs of how much a patient will pay in out of pocket expense vs. monthly premiums.

1. Catastrophic Coverage: the least expensive premium (\$171/month), however, the deductibles are high (\$6,350). After the deductible is paid, your only costs are the monthly premiums. This insurance plan is used to cover medical emergencies. This plan is open to patients 30 years and younger and not available to small businesses.
2. Bronze: least costly premium of the metal tier program (\$285/month) and the highest out of pocket costs, for individuals working in small businesses (\$334/month). Individuals will be responsible for 40 % of costs, including deductibles and co-pays, of all covered benefits.
3. Silver: The monthly premium of this program will cost \$360/month and for individuals working in small businesses \$408/month. Individuals will be responsible for 30 % of costs, including deductibles and co-pays, of all covered benefits.
4. Gold: The monthly premium of this program will cost \$439/month. For individuals working in small businesses it will cost \$477/month. Individuals will be responsible for 20 % of costs, including deductibles and co-pays, of all covered benefits.
5. Platinum: most expensive premiums but the least expensive out of pocket costs \$516/month. For individuals working in small businesses it will cost \$561/month. Individuals will be responsible for 10 % of costs, including deductibles and co-pays, of all covered benefits.

Choosing a tier that best suits your medical needs depends on what the individual can afford as well as the patient's medical needs. If a patient is relatively healthy, the bronze plan with lower monthly premiums and higher out of pocket cost may make the most sense. However, for a patient with recurrent *kidney stones* requiring multiple procedures and preventive care, then the platinum plan with the high premiums but least costly deductibles may make the most logic.

In addition, individuals may qualify for a tax credit and/or cost-sharing reduction. Depending on the earnings of individuals and family incomes, one may qualify for a tax credit to reduce the monthly premiums, lower co-payments and deductibles.

Advantages of the ACA

1. Patients without insurance will get access to affordable quality health insurance.
2. Young adults can stay on their parents plan until age 26. Under ObamaCare, individuals under the age of 26, who have *a kidney stone*, for example, can remain insured as a dependent under their parent's or legal guardian's existing health insurance. Since the prevalence of stones is 12 %, patients in their 20s are at a high risk of stones. With the past health care situation, one was covered under his/her parent's health care plan until age 21. With the new Obama care plan, a patient will have coverage with the national health care plan until the age of 26. The cost of lithotripsy can be expensive, especially if this patient does not have healthcare coverage. Patients at this age may still be in school or working part time where health care coverage for this patient does not exist. With the government health care system, a student or part time worker will have peace of mind since they will have health care and coverage for the *treatment of their kidney stone*.
3. After age 26, if you are unemployed, you may apply for the Medicaid healthcare program.
4. Young adults employed by a business that does not offer health insurance will have cost-effective health insurance through the Affordable Insurance Exchange.
5. For senior patients on Medicare, the ACA will make senior drug coverage more affordable by eliminating the "Donut Hole". Therefore, patients on medication for the *treatment of their stone or stone prevention*, the ObamaCare plan will allow drug coverage which has adversely affected older

- patients requiring necessary prescription medication.
6. Senior patients aged 65 years or older who currently have Medicare may qualify for annual well visits and preventive services not requiring any charges (i.e., flu shots, pneumonia and hepatitis B vaccines).
 7. Counseling preventive services, such as tobacco-use cessation, will be free, unless a diagnosis has already been received for a tobacco-related illness.
 8. Patient screenings for medical conditions, such as cervical cancer, HIV, colorectal cancer, diabetes, cardiovascular disease, cholesterol and bone mass measurement will no longer require Medicare Part B deductibles or co-payments.
 9. Mammograms will typically no longer require Medicare Part B deductibles or co-payments.
 10. With the ACA, an individual's insurance can be taken from job to job.
 11. Under ACA, children cannot be denied health coverage.
 12. No patients with a pre-existing condition or chronic disease of any kind can be denied health care coverage under ObamaCare. Therefore, patients with a *history of stones or who currently have stones cannot be denied care for their stone*. With many health care plans, when you have an active health care problem, it is difficult to obtain a new health care plan to cover a specific diagnosis. If you have a kidney stone and you require surgery for that stone, with many current health care plans, you cannot receive health care to include a pre-existing condition. With the new Obama health care plan, if you have a kidney stone and this stone will require surgical intervention, you will receive coverage for this issue.
 13. Some workers may find their coverage includes new benefits and protection. For instance, as of 2014, employer-based insurance packages will be prohibited from excluding individuals based on pre-existing health conditions. Lifetime and annual limits on health benefits will also be banned.
 14. ObamaCare will not replace Medicare, Medicaid or private insurance and provides a way for millions of uninsured Americans to access health care.
 15. You can also keep or go to any physician within your plan's network.
 16. There are no lifetime limits on health insurance coverage or on most benefits.
 17. Preventive care including screenings is fully covered.
 18. If you require emergency care, you can access it at any hospital, even those outside of your plan's network. Most patients with an *acute kidney stone* attack will require emergency care. ObamaCare will provide coverage for patients in these circumstances.
-
- ### Disadvantages of the ACA
1. To pay for the ACA, there are new taxes on high-earners.
 2. Older patients and patients of higher incomes will pay more for their healthcare plan. As patients get older, the *risk of stones* is less. While individuals may be paying more for their stone care, the higher premiums will be offsets costs for their other healthcare needs.
 3. To pay for the ACA, there will be a home sale tax of 3.6 %.
 4. To obtain health care coverage by January 2014, patients must get an exemption or pay fee if able to afford it.
 5. Employers may find ways to shift costs to their workers:
 - (a) Some employers plan to increase the amount employees need to contribute to premiums, and the migration to high-deductible plans is likely to continue.
 - (b) A proportion of employers intend to cut worker hours to reduce the number of employees eligible for health care benefits.
 6. The Obama administration is counting on signing up seven million Americans, including 2.7 million younger and healthier consumers who are needed to offset the costs

of sicker members, in the first full year of reform through the state exchanges. Younger individuals tend to be healthy and not to need coverage as often as older Americans; however, they will be required to pay into the ACA.

7. ObamaCare focuses more on making sure that a greater percentage of patients are covered than it does on addressing the cost of care.
8. Patients who live in different locations will pay different rates for their health care. ***Therefore, a patient with a stone, for example, who moves from one state or location to another, may pay different premiums.***
9. The ObamaCare plan takes into account community rating. Community rating requires that all patients pay the same rates for their level of coverage regardless of their age or medical condition.
 - (a) As a result, the ACA forces young people, who typically have lower incomes than older workers, to pay far more than their actual cost, and gives older workers, who can afford to pay more, a significant discount.
 - (b) Second, the bills would ban insurers from charging differing premiums based on the health of their customers. Again, that's understandable for individuals with diabetes or cancer. But the bills would bar rewarding people who pursue a healthy lifestyle of exercise or a cholesterol-conscious diet. That's hardly a formula for lower costs.
10. **Freedom to choose your doctors:** Individuals buying through the exchanges must get their care through a "medical home." Medical home is similar to an HMO. You are assigned a primary care doctor, and the doctor controls your access to specialists. The primary care physicians will decide which services, like MRIs and other diagnostic scans, are best for you, and will decide when you really need to see a Urologist.
11. Primary care physician would be the "gatekeepers" and would theoretically guide patients to tests and treatments that have proven to be most cost-effective. The danger is that doctors will be financially rewarded for denying care, as were HMO physicians more than a decade ago.
12. Freedom to keep your existing plan: Individuals who got their personal coverage before the law went into effect can keep their plans, but there is a catch. If the plan changes in any way (i.e., altering co-pays, deductibles, or even switching formulary coverage for a drug) the individual must drop out and shop through the exchange. Since these plans generally change their policies every year, it is likely that many individuals will lose their plans within 12 months.
13. The bill gives Employee Retirement Income Security Act of 1974 (ERISA) employers a 5-year grace period when they can keep offering plans free from the restrictions of the "qualified" policies offered on the exchanges. But after 5 years, they would have to offer only approved plans, with the myriad rules we've already discussed. Therefore, for individuals working for large corporations, "keeping your own plan" has a strict deadline. In 5 years, you will be placed into the exchange.
14. The Affordable Care Act requires most people who can afford health insurance to buy coverage. This provision is known as the individual mandate. If an individual does not buy coverage, then you will pay a penalty based on their taxable annual income and household size.

How Should You Decide on a Health Care Plan That Is Best for You?

To establish a plan on choosing a health care plan, it is important to evaluate the premiums, deductibles and coinsurance. At this point, you can decide which deductible, premium and coinsurance mix is the best option for you and your finances.

For example, if you visit your physician often, you might want to choose a plan that has low copays. For instance, you might choose a plan

that has a higher monthly premium but smaller copay for doctor's office visits.

On the other hand, if you don't visit the doctor often, copays will probably be less of a concern when you are choosing the best coverage plan.

Conclusion

In summary, the Affordable Care Act will result in dramatic changes for America's uninsured, but the health care reform law will also have an impact on individuals with employer-based

coverage. The Affordable care act is anticipated to help make health care choices more predictable and available to all Americans. The reality of the health care bill may result in greater costs and taxes to many individuals. If anything is clear, however, the affordable health care act will be like *a renal stone traveling through the urinary tract-it will take on many twists and turns!*

Recommended Reading

1. www.obamacarefacts.com.
2. Ochs R. New York's health benefit exchange: lots of questions, some answers. Newsday, 28 Sept 2013.

Index

A

Absorptive hypercalciuria, 35, 51
ACA. *See* Affordable Health Care Act (ACA)
Acetaminophen, 163, 165
Acetazolamide (Diamox), 61, 81
Acetohydroxamic acid (AHA), 38, 126
Acute kidney injury (AKI), 154
Adenine phosphoribosyl transferase (APRT), 39
Adult stone disease, 79
Afferent arterioles, 20
Affordable Health Care Act (ACA)
 advantages, 236–237
 catastrophic coverage plan, 236
 disadvantages, 237–238
 four metal tiers plans, 236
 health care law, 235
 health services, 235
 impact of, 235
AHA. *See* Acetohydroxamic acid (AHA)
ALARA (“As Low As Reasonably Achievable”), 81
Allopurinol (Zyloprim), 52, 58, 64, 124
Aloe Vera, 184
Alpha-1 blockers, 76–77
Alpha-mercaptopyronylglycine (Alpha-MPG), 58
American Society of Nephrology and the Endourological Society, 123
American Urological Association (AUA) guidelines
 diet therapies, 219
 evaluation, 219
 follow up, 219–220
 pharmacologic therapies, 219
American Urological Association Meeting AUA (2014), 123
Ammonium acid urate calculi, 61, 70
Angiotensin converting enzyme (ACE), 20
Antegrade progression, 92
Antibiotics, 38, 62, 120, 211
Anticholinergics, 156, 163–164
Anticoagulants, 120
Anti-epileptic drugs (AEDs), 64
Antimicrobial therapy, 38
Appendicitis, 3, 93, 207
Aspirin, 63, 94, 119, 120, 130, 160, 162, 172
Asymptomatic stone. *See* Incidental stones
Atkins diet, 195, 203–204

Autosomal recessive disorder. *See* Cystine stones;
 Cystinuria; 2,8-Dihydroxyadenine (DHA)
 stones

B

Bacteriuria, 76
Benign prostatic hypertrophy (BPH), 59, 71, 76, 94, 99,
 140, 141, 150, 216
Beta agonists, 164–165
Billing and coding process
 coinsurance, 232
 copay, 232–233
 CPT®, 229–230
 E & M services, 232
 global period fee, 230
 ICD-9 codes, 229
 ICD-10 coding system, 229
 modifier, 230–232
 questionnaires, 233
Bladder, 20
 exstrophy, 23, 24
 function, 21
 overactive, 165, 178
 stones
 augmentation cystoplasty, 148–149
 in children, 80
 gender, 71
 between prostate, 151
 symptoms, 27
 treatment, 27, 150
 Urat-1, clinical device, 29
 structure and location, 21–22
 surgical procedure
 Greenlight™ laser ablation of prostate, 140–141
 open surgical cystotomy with stone
 removal, 141
 transurethral stone removal (cystolitholapaxy),
 139–140
 treatment, 139
Blood urea nitrogen (BUN), 170, 172
Body mass index (BMI), 64, 86
BPH. *See* Benign prostatic hypertrophy (BPH)
Bucillamine (Rimatil), 58
Bumetanide, 61

C

- Calcium, 35, 184
- Calcium oxalate (CaOx) stones, 188
- Calcium oxalate dihydrate (COD)
 - composition and fragility, 46
 - fragility of stones, 144
 - radiopaque, 46
 - shape, size and color, 37, 43–44
 - urinary, 44
- Calcium oxalate monohydrate (COM), 36
 - composition and fragility, 46
 - radiopaque, 46
 - shape, size and color, 44
- Calcium phosphate stones, 37
 - radiopaque, 46
 - shape, size and color, 44, 45
- Calcium stones. *See also* Calcium oxalate dihydrate (COD); Calcium oxalate monohydrate (COM)
 - associated with obesity, 86
 - hypercalciuria, 35
 - metabolic factors, 35
 - oxalate dihydrate, 35, 37
 - phosphate, 35, 37
 - surgical therapy, 35
 - treatment, 36
- Calcium + Vitamin D³ (OsCal), 125
- Cancer, 52
 - leukemia, 57
 - lymphoma, 57
 - metastatic, 172
 - obesity-related conditions, 85
 - patient, ureteral stent in, 153
 - prostate, 115
- Captopril, 58, 126
- Carbonic anhydrase inhibitors, 61
- Cardiovascular disease, 71
- Celebrex (celecoxib), 58
- Chelating agent, 54, 58
- Chemolysis, 38
- Chemotherapeutic agents, 63
- Children with stones
 - composition, 80
 - inherited abnormality, 83
 - prevention, 82
 - risk factors, 80–81
 - symptomatology, 81
 - treatment, 82
 - types of, 79–80
 - work-up, 81
- Chlorthaladone, 124
- Cholecystitis, 3
- Cinnamon, 184
- Ciprofloxacin, 62
- Citric acid (Citrate), 182–183
- Colchicine, 58
- Complete ureteral duplication, 23
- Computed tomography (CT scan)
 - IV dye (contrast), 105
 - multiple non-contrast renal protocol, 104
 - non contrast
 - advantage and disadvantage, 105
 - patient indications, 104–105
 - right lower pole renal stone, 148
- Congenital anomalies, 81
- Cranberry, 184
- Creatinine (Cr), 172
- Crixivan[®] stones, 40, 62
- Crohn's disease, 71
- Crystallization, 178
- Current Procedural Terminology (CPT[®]), 229–230
- Cyclooxygenase (COX), 162
- Cystine dimethyl ester (CDME), 218
- Cystine, Ornithine, Lysine and Arginine (COLA), 38
- Cystine stones, 35
 - autosomal recessive inborn error, 38
 - composition and fragility, 46
 - poorly radiopaque, 46
 - prevalence of, 38
 - risk factor, 38
 - shape, size and color, 45–46
 - treatment, 38–39
- Cystinuria
 - metabolic and hereditary factors, 53–54
 - stone formation, 58
- Cystoscope, 157

D

- Darifenacin (Enablex), 164
- DASH (Dietary Approaches to Stop Hypertension)
 - diet, 201–202, 209
- Dehydration, 51, 83, 175–178
- Diabetes, 57
- Diabetic nephropathy, 100
- Diarrhea, 6, 56, 59, 61, 64, 70, 124, 125, 175, 182, 197
- Diet. *See also* Dietary patterns
 - kidney stones, influence of, 187
- Dietary factors
 - calcium oxalate stones, 188
 - condition in digestive tract, 191–192
 - into digestive tract, 190–191
 - high urinary calcium excretion, 188–190
 - high urinary oxalate excretion, 190
 - low urinary citrate excretion, 192
 - low urinary magnesium excretion, 192–193
 - calcium phosphate stones
 - high urinary calcium excretion, 195
 - low urinary citrate excretion, 194
 - case study, 199
 - cystine stones, 196
 - diabetes, 196–197
 - macular degeneration, 198
 - malabsorption, 197
 - nutrition therapy, goals for, 187–188
 - osteoporosis, 197–198

special medical situations, 196
 uric acid stones
 high urinary uric acid excretion, 195
 low (acid) urine pH, 195–196
 urine volume (*see* Hydration)

Dietary patterns
 lithogenic diets (*see* Lithogenic diets)
 with lower risk of stones
 DASH diet, 201–202
 Mediterranean diet, 202
 raw food diets, 203
 vegetarian diet, 203
 Weight Watchers diet, 202–203

Dietary supplements
 cautious about certain, 184
 citric acid, 182–183
 magnesium, 181–182
 NSF® International seal, 184
 Omega-3 fats, 183
 potassium, 182
 pumpkin seeds, 183
 rose hips, 183
 safety, 183–184
 USP® seal, 184
 vitamin B₆ (pyridoxine), 183

2,8-Dihydroxyadenine (DHA) stones, 39
 radiolucent calculi, 46, 143

Diuretics, 63
 Dornier Compact Delta II lithotripter, 135
 Dornier HM1, 29
 D-Penicillamine, 125
Drosophila melanogaster, 217
 Dual-energy CT (DECT), 215–216
 Dukan diet, 205

E
 Ectopic kidney, 145, 146
 Elderly with stones
 diagnosis, 83
 management, 83–84
 prevalence, 83
 risk factors, 83

Electrohydraulic lithotripter, 29–30
 Electrohydrolic force, 77
 Endoscopy equipment, 141–142
 Endourology fellowship, 113, 114
 Ephedrine, 62–63
 Epididymitis, 208
 Epispadias, 25
 Estrogen, 58, 72
 Ethylene glycol, 217
 Evaluation and management (E & M) services, 232
 Extracorporeal shock wave lithotripsy (ESWL) technique, 77
 complication, 148
 lower pole stone, 147
 stone(s) fragment, 82
 surgical procedure

kidney stone, 135
 ureter stones, 138–139
 urinary stone treatment, 29–30

F

Flank pain/renal colic, 92
 Flomax®, 130
Fluids
 consumption
 kidney stone formation, prevention of, 179
 requirement for, 177–178
 timings, 178
 types of
 citrus based, 176
 coffee, 177
 food, 176
 soda, 177
 tea, 177
 water, 176

Food and Drug Administration (FDA), 184
 Furosemide, 61

G

Gastric/duodenal ulcer, 3
 Gastro-Intestinal Stromal Tumor (GIST), 5–6
Gender, 73
 age, 69
 anatomical barrier, 71
 bladder stones, 71
 case study, 73
 diseases
 cardiovascular disease, 71
 Crohn's disease, 71
 eating disorders, 70–71
 laxative abuse, 70
 osteoporosis, 71
 factors
 post-menopause, 72
 during pregnancy, 72
 vasectomy, 72
 hormones, 72
 risk factors, 72
 statistics, 69
 stone types, 70
 symptoms, 70

Gerota's Fascia, 20
 Good Manufacturing Practices (GMPs), 184
 Gout, 57–58
 Gross hematuria, 94
 Guaifenesin stone, 63

H

Health care plan, 238–239
 Hematuria, 173
 Heterologous nucleation, 52

- Horeshoe kidney, 147
 Hounsfield units (HU), 46
 24 hours urine test
 appropriate time for collecting, 170
 determination of urine volume, 179
 method, 171
 multiple factors on, 171–172
 poor man's, 171
 preparation, 170–171
 results, 171
 urine container, 170
 “World Series” of urine tests, 170
 Hydration, 129, 179
 fluids intake (*see* Fluids)
 goals, 178
 management
 fluid by numbers, 179
 urine by colors, 178–179
 Hydrochlorothiazide (HCTZ), 51, 124
 Hydromorphone (Dilaudid), 161–162
 Hydronephrosis, 92, 104, 105, 207
 Hydroureteronephrosis, 75, 76, 81
 Hydroxyapatite. *See* Calcium phosphate stones
 Hyperoxaluria, 35, 52–53, 125
 Hyperparathyroidism, 58, 172
 Hypertension, 57, 61, 63, 83, 85, 126, 135, 165, 201, 202, 209
 Hyperuricosuria, 35, 36
 metabolic and hereditary factors, 52
 stone formation, 59
 Hypocitraturia, 35, 51
 medical therapy, 124
 metabolic and hereditary factors, 52
 stone formation, 59
 Hypospadias, 23–24
 Hypoxanthine-guanine phosphoribosyl transferase (HGPRT), 80
- I**
- Ibuprofen, 58, 119, 162, 163, 170, 227
 Immobilization, 56
 Incidental stones
 indications to treat, 98
 medium sized stones, 98
 prevention, 99–100
 small stones, 98
 treatment
 option, 98
 planning your procedure from office vs. ER, 99
 vs. symptomatic stone, 100
 work up, 99
 Incisional hernia, 11
 Incomplete ureteral duplication, 23
 Indinavir (Crixivan), 62
 Indinavir sulfate, 40
 Indispensable water losses, 177
 Indocin (indomethacin), 58
 Indomethacin, 163
 International Classifications of Diseases (ICD), 229
- Intractable pain, 62, 77, 92, 98, 116, 120, 156
 Intramural ureter, 92
 Intravenous Urography (IVU), 106, 107
- J**
- Jackson Pratt drains, 12
- K**
- Ketorolac, 163
 Kidney
 congenital anomalies
 horseshoe kidney, 22
 renal ectopia, 23
 function, 20
 retroperitoneum, 20
 structure and location, 20–21
 Kidney stones. *See also* Stones
 in animal models, 216–217
 in children, 79–80
 management of
 AUA guidelines for, 219–220
 imaging technology, 215–216
 new medications and probiotics, 217–218
 SDM, 216
 preventing formation of
 complimentary medicine (*see* Dietary supplements)
 nutrition recommendations (*see* Dietary factors)
 robotic approaches, 218–219
 treatment options, 215
Klebsiella, 93
K. pneumonia, 38
 KUB (Kidneys, Ureters and Bladder)
 advantages, 107
 disadvantage, 107
 plain-film radiography, 106
 radiation dose, 106–107
 radiopaque stone, 106, 107
 steinstrasse, 148
 with ultrasound combination, 107
- L**
- Laxatives, 61
 L-cystine dimethylester (L-CDME), 126
 Lesch-Nyhan syndrome, 52, 80
 Leukemia, 57
 Lithogenic diets
 Atkins diet, 203–204
 Dukan diet, 205
 ketogenic diet, 204
 Paleo diet, 205
 South Beach diet, 204
 Western diet, 204–205
 Zone diet, 204
 Loop diuretics, 61–62, 81
 Lymphoma, 57

M

- Magnesium, 181–182
 Magnesium ammonium phosphate stone. *See* Struvite stones
 Magnesium citrate, 53
 Magnesium oxide, 53, 125
 Magnesium trisilicate, 61–62
 MAG-3 renal scan
 disadvantage, 108
 nuclear renogram, 108–109
 Tc-99 m MAG3, 108
 tracers, 108
 vs. IVP, 108
 Medical expulsive therapy (MET), 76–77, 98, 107, 130
 Medical therapy, 38, 82
 calcium stone formers
 cystinuria, 125–126
 hypercalciuria, 124
 hyperoxaluria, 125
 hyperuricosuria, 124
 hypocitraturia, 124
 hypomagnesiuria, 125
 future stones maintenance/management, 210
 non-calcium stone formers
 infection stones, 126
 Mediterranean diet, 202
 Medullary sponge kidney (MSK), 57
 Meperidine (Demerol), 162
 Metabolic and hereditary factors
 cystinuria, 53–54
 hypercalciuria, 51–52
 hyperoxaluria, 52–53
 hyperuricosuria, 52
 hypocitraturia, 52
 hypomagnesiuria, 53
 Metabolic therapy, 82
 Microscopic hematuria, 94
 Micturition, 20
 Mirabegron (Myrbetriq), 165
 Morphine, 161
 Motrin (ibuprofen), 58
 MRI, 103, 107, 145, 238
 Multiple endocrine neoplasias (MEN) syndrome, 52

N

- Nephrostomy tube, 157
 Non-steroidal anti-inflammatory drugs (NSAID), 162–163
 definition, 165
 side effects, 166

O

- ObamaCare. *See* Affordable Health Care Act (ACA)
 Obesity
 case study, 86–87
 causes, 85
 description, 85

- gender and stone risk factors, 72
 prevalence of, 85
 and stone disease
 body mass index, 86
 waist circumference, 86
 weight gain, 85–86
 in United States, 85
 Obstructive uropathy, 97
 Omega-3 fats, 183
 Open cystolithotomy, 150
 Open-ended catheter, 157
 Opioids, 161
 definition, 165
 side effects, 165
 Oral antibiotic therapy, 53
 Osteoporosis, 71
 Oxalate decarboxylase (Oxazyme), 218
Oxalobacter formigenes, 53, 218
 Oxybutynin (Ditropan, Oxytrol), 164
 Oxycodone (ex. Roxicodone), 162
- P**
- Paleo diet, 205
 Parenchyma, 20
 Patients
 asymptomatic stone (*see* Incidental stones)
 ER room #2 (nebulous room)
 blood test, 4
 colonoscopy, 6
 CT scan in abdomen, 4
 diagnosis and, 4
 gastrointestinal stromal tumor, 5
 hematologist/oncologist investigation, 5–6
 preparation for surgery, 7–8
 raising appropriate questions, 6, 8
 ultrasound evaluation, 4
 future stones maintenance/management
 CM therapy, 210
 diagnosis, 211
 dietary recommendations, 210
 with dietician, 209
 follow up tests, 210–211
 follow up with Urologist, 208–209
 hydration status, 209–210
 medical therapy, 210
 questionnaires, 211
 symptoms awareness, 207–208
 with yourself, 211
 staghorn calculi, treatment for, 98–99
 stone procrastinators, 98
 with symptomatic stone (*see* Symptomatic stone)
 Pelvic brim, 21
 Percutaneous nephrolithotomy (PCNL), 29, 77, 82, 103, 216
 surgical procedure, kidney stone
 advantages and disadvantages, 132
 description, 132–134
 indication, 132, 133

Phenytoin, 81
 Potassium, 182
 Potassium citrate (Urocit-K), 52, 54, 64, 124, 125
 Pre-admission testing (PAT), 130, 131
 Prednisone, 52
 Pregnancy and urolithiasis
 anatomic factors, 75–76
 case study, 78
 follow up, 77
 incidence, 75
 metabolic factors, 76
 symptoms, 76
 treatment of, 76–77
 work-up, 76
 Primary hyperoxaluria, 53, 80, 125, 190
 Primary hyperparathyroidism. *See* Resorptive hypercalciuria
 Probiotics, 183
Proteus mirabilis, 38
Providencia, 93
Pseudomonas, 38, 93
 Pumpkin seeds, 183
 Pyelolithotomy, 132
 Pyelonephritis, 92, 97

R

Randall's plaques, 21, 35, 36
 Raw food diets, 203
 Reflux, 21
 Renal calyceal diverticula, 147
 Renal colic, 79
 drug induced, 60–64
 pain associated with
 control, 159
 frequently asked questions, 165–166
 location of, 159
 medication, 161–165
 therapy, 159–160
 patient interview, 160–161
 Renal hypercalciuria (renal leak), 35, 51–52
 Renal pelvis, 21
 Renal sinus, 21
 Renal tubular acidosis (RTA), 52, 58, 124
 Renin, 20
 Resorptive hypercalciuria, 35, 51, 52
 Retrograde movement, 92
 Retrograde Pyelogram (RP), 108
 Reyataz® (atazanavir) stones, 40
 Rose hips, 183
 Roux-en-Y (RYGB), 59

S

Saponification process, 53
 Sarcoidosis, 52, 56
 Seizure diet (Ketogenic diet), 204
 Seizure disorders
 acetazolamide, 61
 topamax (topiramate), 40–41

Serratia, 93
 Serum calcium test, 172
 Serum chemistry (BUN, Cr) test, 172
 Serum parathyroid hormone test, 172
 Serum uric acid test, 172
 Serum Vitamin D, 173
 Shared decision making (SDM), 216
 Shock Wave Lithotripsy (SWL), 103
 Solifenacin (Vesicare), 164
 South Beach diet, 204
 Staghorn calculi, 29
 treatment for, 98–99
Staphylococcus, 93
 S. aureus, 38
 S. epidermidis, 38
 Steinstrasse, 148, 155
 Steroids, 64
 Stone Cup Strainer, 169
 Stones. *See also specific stones*
 to access, 145–147
 composition and fragility, 46
 diagnosis
 radiolucent stones, 143
 ureteral stones, 144
 etiology, 46
 facts, 32–33
 formation
 age and gender, 64
 anatomical factors, 59
 cancer, 57
 climate factors, 55
 diabetes, 57
 diarrhea, 56
 dietary factors (*see* Dietary factors)
 ethnicity, 66
 family history, 66
 genetic factors (*see* Metabolic and hereditary factors)
 hormones, 58–59
 inflammatory bowel disease, 56
 lifestyle factors, 56
 medication factors, 60–64
 medullary sponge kidney, 57
 occupational factors, 56
 sarcoidosis, 56
 surgical factors (*see* Surgical procedures)
 and urinary pH, 47
 urinary tract infections, 57–58
 fragility of, 144–145
 and gender, 47
 location, 47
 lower pole, 147–148
 passage rates, 48
 in patient
 with bladder augmentation cystoplasty, 148–149
 urinary diversion, 149–150
 personal experiences with, 31–33
 prevalence of, 31
 prevention, 129–130

Randall's plaques in papilla, 91
 rule of 50, 33
 size, 47–48
 special stone scenarios, 221–227
 symptoms
 antegrade progression, 92
 dysuria, 92, 95
 flank pain/renal colic, 92
 hydronephrosis, 92
 intramural ureter, 92
 ipsilateral testicle, 93
 pain, 92–95
 pyelonephritis, 95
 retrograde movement, 92
 strangury, 95
 in UPJ, 91, 92
 treatment and prevention
 analysis, 169–170
 24 hours urine test (*see* 24 hours urine test)
 types of, 35, 36
 X-ray characteristics of, 46–47

Stress, 56

Stricture, 108

Struvite stones, 93
 causes, 37
 composition and fragility, 46
 diagnosis, 38
 infectious stones, 37–38
 medical management, 37, 38
 poorly radiopaque, 46
 risk factors, 38
 shape, size and color, 45
 treatment, 37
 urinary pH, 47
 urinary tract infections, 37–38

Sulfadiazine, 62

Sulfamethoxazole-trimethoprim (Bactrim), 62

Sulfonamides, 62

Superior mesenteric artery (SMA), 6

Surgical procedures
 anesthesia, 131
 appropriate questions, 14
 bladder
 GreenlightT laser ablation of prostate, 140–141
 open surgical cystotomy with stone removal, 141
 transurethral stone removal (cystolitholapaxy),
 139–140
 treatment, 139
 elective hernia procedure, 12–14
 gastric bypass procedures, 59–60
 ileostomy and colon resection, 60
 kidney stone
 ESWL, 135
 open surgery, 132
 PCNL, 132, 133
 ureteroscopy, 132–135
 postoperative instruction, 120–121
 preparation, 119–120, 130–131
 site and side verification, 131

small bowel resection, 60
 on surgery day, 131
 time out, 131
 ureter stones
 cystoscopy with ureteral stent
 placement, 137–138
 ESWL, 138–139
 open surgery, 136
 ureteroscopy, 136–137

Symptomatic stone
 composition and location, 97
 imaging studies, 97
 medical attention in local hospital, 97
 series of tests, 97
 shapes and size, 97
 symptoms, 97
 treatment
 indications to, 98
 options, 98
 vs. incidental stones, 100

Systemic lupus erythematosus, 52

T

Tamsulosin dilate, 130

Testicular torsion, 208

Testosterone, 72

Thiazide diuretics, 219

Thiopronin (Thiola), 125–126

Tolerance, definition, 165

Tolterodine (Detrol/Detrol LA), 164

Topamax® stones, 40–41

Topiramate (Topamax), 64, 81

Tramadol (Ultram), 162

Triamterene stone, 63

Trospium (Sanctura/Sanctura XR), 164

Tumor lysis syndrome, 194–195

Turmeric, 184

Tylenol, 119, 120

U

Ultrasonic propulsion, 216

Ultrasound, 4, 29, 46, 77, 135, 144, 216
 abdominal, 83
 kidney and bladder, 81
 KUB with, 107
 renal, 83, 94, 105–106, 211

United States
 bladder stones, 150
 children with urinary tract stone, 79
 codes in, 229
 cystinuria prevalence, 38
 elderly with stones, 83
 kidney and ureteral stones, 29
 medullary sponge kidney in, 57
 obesity, prevalence of, 85

Urat-1, 29

Urease inhibition, 38

- Ureter, 19
 function, 21
 structure and location, 21
 ureteral duplication, 23
- Ureteral jets, 76, 81, 105, 106
- Ureteral manipulation, 154
- Ureteral pelvic junction (UPJ), 91
- Ureteral stent
 advantages
 infection, 154
 injury, 154
 narrowed ureter, 154–155
 obstruction, 154
 prophylaxis, 155
 swollen ureter, 154
 alternatives, 156–157
 contact with Urologist, 156
 disadvantages
 blood, 155
 calcified stents, 155
 displaced stents, 155–156
 irritative voiding symptoms, 155
 pain, 155
 stent colic, 156
 insertion of, 156
 length, 153
 limitations, 156
 materials, 153
 removal, 157
 structure, 153, 154
- Ureteropelvic junction (UPJ), 21, 71
- Ureteropelvic junction obstruction (UPJO), 145–147
- Ureteroscopy, 129
 kidney stone
 advantages and disadvantages, 135
 description, 133–135
 indications, 133
 ureter stones
 advantages and disadvantages, 137
 description, 136–137
 indications, 136
- Ureteroscopy (URS) technique, 82, 216
- Ureterovesical junction (UVJ), 21
- Urethra
 epispadias, 25
 function, 22
 hypospadias, 23–24
 structure and location, 22
- Urethral glands, 22
- Urethral meatus, 22
- Uric acid stones, 35
 associated with obesity, 86
 composition and fragility, 46
 diabetes, 57
 factors, 36
 incidence of, 36
 low urine pH, 36
 radiolucent, 46
 shape, size and color, 44–45
 treatment, 36
- Uricosuric agents, 62
- Urinary calculi, 61
- Urinary stasis, 57
- Urinary stones
 history of, 27
 milestones of disease, 27
 symptoms, 27
 treatment
 crushing stone, 27, 28
 cystoscope, 28
 electrohydraulic lithotripter, 29
 ESWL, 29–30
 lithotomous, 27–28
 PCNL, 29
 surgical procedure, 27
 Urat-1, 29
- Urinary tract infections (UTI), 57
- Urinary tract stones
 management strategy, 123
 medical therapy (*see* Medical therapy)
 metabolic cause, 123
 treatment, 123
- Urinary tract system
 bladder, 20
 functions, 19
 kidneys, two bean-shaped organ, 19
 ureters, 19
 urine excretion, 19
- Urination, 20
- Urine
 dipstick, 173
 low and high volumes, 81
 pH, 81
 volume (*See* Hydration)
- Urine Analysis, Culture and Sensitivity (UA, C & S), 173
- Urolithiasis
 diagnosis, 103–104
 diagnostic imaging
 MRI, 103, 107
 ultrasonography, 103
 x-rays (*see* Computed tomography (CT scan);
 Intravenous Urography (IVU); KUB)
 initial management, 103–104
 treatment, 103
- Urologist selection
 different qualities in, 113
 helpful facts, 114–115
 list of questions, 113, 114
 second opinion, seeking for, 115–116
 treatment
 electively and emergency room, 116
 option, 116
 performance place, 116
 type of insurance, 114
- V**
- Vasopressin, 56
- Vegetarian diet, 203

Venofen, 13
Vitamin B₆ (Pyridoxine), 183
Vitamin C, 81
Vitamin C (ascorbic acid), 184
Vitamin D, 76, 81

W

Water, 179
 dehydration, 175
 intoxication, 175
 significance of, 175
Weddellite. *See* Calcium oxalate dihydrate
Weight Watchers diet, 202–203

Western diet, 204–205
Whewellite. *See* Calcium oxalate monohydrate

X

Xanthine oxidase, 39
Xanthine stones, 39
 radiolucent, 46

Z

Zone diet, 204
Zonisamide (Zonegran), 64