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TOPIC PAPER

# Urinary tract infection associated with conditions causing urinary tract obstruction and stasis, excluding urolithiasis and neuropathic bladder

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**Abstract**
*Purpose* The aim of this study was to examine urinary tract infection (UTI) associated with conditions causing urinary tract obstruction and stasis, excluding urolithiasis and neuropathic bladder dysfunction.
*Methods* An electronic literature search was performed using the key words *urinary tract infection (UTI), benign prostatic hyperplasia (BPH), hydronephrosis, obstruction, reflux, diverticulum, urethra,* and *stricture*. In total, 520 abstracts were reviewed, 210 articles were studied in detail, and 36 were included as references.
*Results* It is one of the axioms of Urological practice that urinary tract obstruction and stasis predispose to UTI. Experimental studies indicate that, whereas transurethral inoculates of bacteria are rapidly eliminated from the normal bladder, urethral obstruction leads to cystitis, pyelonephritis, and bacteremia. BPH is, next to urolithiasis, the most common cause of urinary tract obstruction predisposing to UTI. Urethral stricture remains a common cause of UTI in many parts of the world. Urinary stasis in diverticula of the urethra or bladder predisposes to UTI. Experimental studies have shown that, whereas the normal kidney is relatively resistant to infection by organisms injected intravenously, ureteric obstruction predisposes to pyelonephritis. It also causes renal dysfunction which impairs the excretion of antibiotics in the urine, making eradication of bacteria difficult.
*Conclusions* In patients with UTI and urinary tract obstruction, targeted antibiotic treatment according to urine culture should be complemented with urgent drainage (bladder catheterization, percutaneous nephrostomy or ureteric stenting) followed by definitive surgery to remove the cause of obstruction or stasis once infection is under control.

**Keywords** Urinary tract · Infection · Obstruction · Prostate · Urethra · Stricture · Hydronephrosis · Diverticulum

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## Introduction

The aim of this paper is to focus on urinary tract infection (UTI) associated with conditions causing urinary tract obstruction and stasis, other than urolithiasis and neuropathic bladder dysfunction. The prevalence and causes of urinary tract obstruction in patients with UTI vary considerably in different reports in the literature. The most common causes are benign prostatic hyperplasia (BPH) in up to 45% of cases, prostatic cancer in 2–8%, urethral stricture in 3–8%, and pelviureteric junction obstruction (PUJO) in 2% (Fig. 1) [1].

## Methods

An electronic literature search was performed on PubMed using the key words *urinary tract infection (UTI)* alone and in combination with the following terms: *benign prostatic hyperplasia, BPH, hydronephrosis, obstruction, reflux, diverticulum, urethra,* and *stricture*. Abstracts were reviewed, relevant articles were studied in full-length

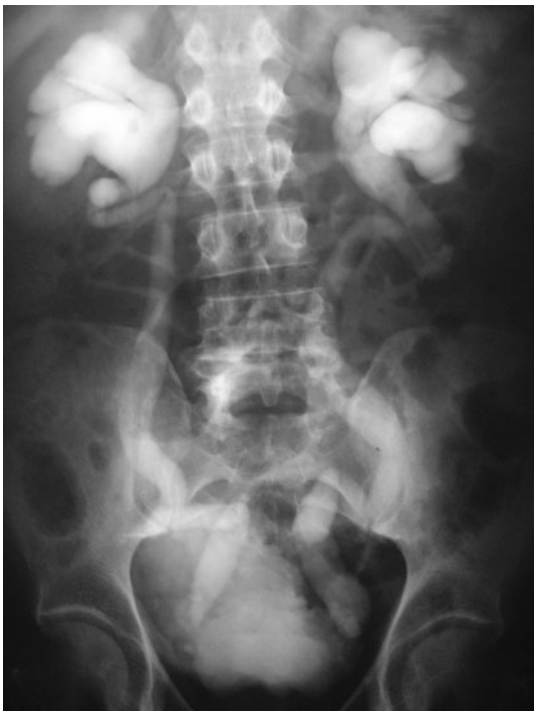

**Fig. 1** Excretory urogram shows bilateral hydro-ureteronephrosis with large postvoid residual urine volume due to bladder outlet obstruction

version, and further references were obtained from these articles. In total, 520 abstracts were reviewed, 210 articles were studied in detail, and 36 were included as references in this paper.

A more extensive version of this work was published as a chapter titled "Urinary tract infection in patients with underlying urological abnormalities" in [37].

## Bladder outlet obstruction (BOO)

An experimental animal model in mice has shown that after transurethral inoculation with *E coli,* bacteria will be rapidly eliminated by urination in normal mice, but will cause bacteremia and pyelonephritis in mice with reversible urethral obstruction for 1–6 h. Urethral obstruction enhances the uropathogenicity of nonpathogenic *E coli* and causes more severe renal lesions in mice challenged with uropathogenic *E coli* [2].

A large postvoid residual (PVR) urine volume may be caused by BOO, but may also result from inadequate detrusor contraction, a large bladder diverticulum or even high-grade vesico-ureteric reflux. It seems logical that a large PVR should predispose to UTI, because bacteria are not mechanically washed out by normal urinary flow, and stasis allows more time for bacterial adherence and multiplication. However, there is little evidence in the literature that the occurrence of UTI in the aging male population is associated with either PVR or BOO. In elderly men, bacteriuria does not appear to correlate with age, PVR, or symptoms of BOO [3]. In elderly women, an increased PVR does not appear to be associated with bacteriuria. However, the incidence of bacteriuria after urodynamic studies is higher in men (36%) compared with women (15%), possibly due to larger PVR urine volumes, or to less effective washout of inoculated bacteria [4].

## Benign prostatic hyperplasia (BPH)

A review of more than 200 studies published between 1954 and 1988 concluded that most men with UTI had a functional or anatomic abnormality of the genitourinary tract. BPH and genitourinary instrumentation were the major predispositions to UTI in men [1]. In older surgical series, UTI was the indication for surgical intervention in about 12% of men with BPH [5]. However, among placebo-treated patients in the Medical Therapy of Prostatic Symptoms (MTOPS) study, the cumulative incidence of UTI at 4 years was <1% [6].

Bacteriuria prior to prostatectomy in men with BPH has been reported in 28–54% of cases. The incidence is higher in those catheterized before operation (44–57%) than in those not catheterized (18%). Several studies have indicated a correlation between the incidence of preoperative bacteriuria and the duration of catheterization as well as PVR urine volume [7].

Bacteria can be cultured from prostatic tissue obtained at prostatectomy in 21–44% of cases (67% in catheterized and 28% in uncatheterized patients) [8]. Bacteremia after transurethral resection of the prostate (TURP) has been reported in 23–29% of patients despite antibiotic prophylaxis, in 54% of patients with preoperative bacteriuria and 8% without bacteriuria. In 70–81% of cases, an identical species was isolated from preoperative urine cultures, and in 54% the organism causing bacteremia was identical to that cultured from prostatic tissue. Bacteremia was significantly more common in patients with preoperative UTI [9].

## Prostate cancer and prostatitis

In men with febrile UTI, the prostate is frequently involved, causing PSA elevation. The increased total PSA and decreased free/total PSA ratio in prostatitis may be falsely interpreted as a sign of cancer. The use of empiric antibiotic treatment to decrease the PSA and to avoid unnecessary prostate biopsy is controversial, but increases the risk of bacteremia caused by resistant bacteria after transrectal prostate biopsy [10].

There appears to be a correlation between bacterial colonization or chronic inflammation of the prostate and the development of prostate cancer. It is postulated that during acute or chronic inflammation in the prostate, various cells are activated by chemokines via different chemotaxin receptors which then trigger processes in angiogenesis, cellular growth and neoplasia which may lead to prostate cancer [11].

## Urethral stricture

In men with UTI, in some developing countries, the second most frequent underlying abnormality, after BPH, is urethral stricture. Men with urethral strictures present with UTI in up to 44% of cases [12].

## Diverticula

Bladder diverticula are most often acquired (secondary to BOO or neuropathic bladder dysfunction) but may also be congenital. They are much more common in men than women and are most often associated with BPH, prostate cancer or urethral stricture, although obstruction is not present in all cases [13].

Congenital bladder diverticula most often present with UTI [14]. Urinary stasis in a bladder diverticulum may predispose to UTI and may impede the eradication of established infection. Relapsing or persistent UTI unresponsive to antibiotic therapy may be an indication for bladder diverticulectomy.

A study comparing patients with BPH plus a bladder diverticulum and patients with BPH only found that the group with BPH plus a diverticulum had a higher rate of acute urinary retention and UTI (22% vs. 3%), a larger mean PVR and greater urethral resistance [15]. In patients who underwent TURP with or without diverticulectomy, urethral resistance parameters and PVR decreased in both groups. Diverticulectomy showed an improvement of bladder contractility with longer detrusor contraction duration, supporting its use in cases of BPH-associated diverticula [15].

Urethral diverticula in women are most probably caused by infection of the periurethral glands, possibly by *Neisseria gonorrhoeae*, although the initial infection and especially subsequent reinfections may be caused by *E coli* and other coliform bacteria or vaginal flora. Recurrent infection of the periurethral glands leads to obstruction, suburethral abscess formation and subsequent rupture of the infected glands into the urethral lumen [16].

UTI is the mode of presentation in about 30% of women with urethral diverticula. *E coli* is the most common organism isolated, but other gram-negative enteric flora as well as *N. gonorrhoeae, chlamydia, streptococci*, and *staphylococci* are often present [17].

The management of female urethral diverticulum is surgical excision of the diverticulum and closure of the tract communicating with the urethra. In women with recurrent UTI and a large PVR, dilatation of the urethra to improve bladder emptying is often performed, although there is no evidence to support its efficacy [18].

## Upper tract obstruction

It is generally accepted that urinary stasis provides the time and opportunity for bacteria to adhere to the urothelium, multiply, and infect the host [19]. A contributing factor may be that ureteric obstruction causes renal dysfunction, so the kidneys are less effective in concentrating antibiotics in the urine, making eradication of bacteria difficult and predisposing to bacterial resistance. The most important determinants of UTI caused by resistant strains are previous use of antibiotics and the presence of underlying urological diseases [18].

Hematogenous infection of the kidney is rare in normal individuals, but experimental data indicate that infection is enhanced when the kidney is obstructed [20]. In an animal model of hematogenous pyelonephritis, the kidney is relatively resistant to infection by organisms injected intravenously, but if a ureter is ligated, the obstructed kidney becomes infected [21].

In an experimental model in rats, transient ureteric obstruction (24 h) with inoculation of *E coli* in the bladder after release of ureteric obstruction predisposes to ascending pyelonephritis. It is postulated that acute ureteric obstruction induces foci of medullary necrosis in the papillae, which may provide a nidus for bacterial invasion, while altered urodynamics after the release of ureteric obstruction may promote ascending infection [22].

There are few clinical studies on the risk of UTI associated with upper tract obstruction. In a study of children prenatally diagnosed with severe hydronephrosis due to PUJO or ureterovesical junction obstruction and not on antibiotic prophylaxis, the incidence of UTI during the first 12 months postnatally was 36%. Most cases of UTI (93%) occurred in the first 6 months postnatally [23].

In another study of children with prenatally detected renal pelvic dilatation, the estimated cumulative incidence of UTI was 8% at age 12 months, 13% at 24 months, and 21% at 36 months. Independent predictors of UTI during follow-up were female gender and the presence of vesicoureteric reflux (VUR) or urinary tract obstruction [24].

In a study of children with prenatally diagnosed nonrefluxing hydronephrosis, UTI developed in 19% during

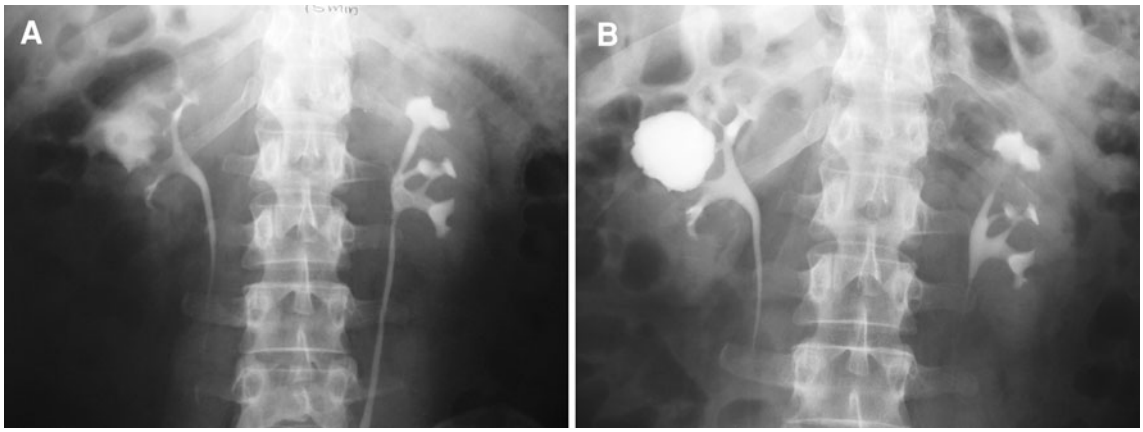


**Fig. 2** **a** and **b** Intravenous pyelogram showing calyceal diverticulum containing a stone (seen as a filling defect in the early pyelogram phase)

12 months of follow-up—in 84% of those cases within the first 6 months. UTI developed in 39% of children with and in 11% without obstructive uropathy. High-grade hydronephrosis was associated with an increased incidence of UTI: 40% in grade IV hydronephrosis, 33% in grade III, 14% in grade II, and 4% in grade I. The authors recommended antibiotic prophylaxis in neonates with obstructive uropathy, severe hydronephrosis or hydroureteronephrosis without VUR [25].

However, another study of children with antenatally diagnosed severe hydronephrosis secondary to obstructive megaureter or PUJO not maintained on prophylactic antibiotics reported an overall rate of UTI of only 4.3%. No statistically significant difference in the infection rate was noted according to sex, obstruction level, hydronephrosis grade, or circumcision status. The authors concluded that antibiotic prophylaxis is unlikely to benefit most children with hydronephrosis secondary to upper tract obstruction [26].

Despite a surprising lack of relevant clinical studies, it is generally accepted practice that in patients with UTI and upper urinary tract obstruction, empiric antibiotic treatment should be complemented with urgent intervention to drain the urinary tract (e.g., percutaneous nephrostomy or double-J ureteric stenting). This should be followed with targeted antibiotic treatment (according to urine culture), and definitive surgery to remove the cause of obstruction once infection is under control.

### Renal papillary necrosis (RPN)

Although there are several factors that on their own may cause RPN, the coexistence of multiple factors (e.g., diabetes mellitus or obstruction and UTI) increases the risk of developing RPN [18]. Therefore, in ureteric obstruction, it is important to prevent UTI and, if UTI is present, to drain the kidney by ureteric catheter or percutaneous nephrostomy [27]. A necrotic renal papilla may cause obstruction, and the medullary cavity caused by RPN may cause urinary stasis, both conditions predisposing to UTI.

### Renal calyceal diverticula

Renal calyceal diverticula are often asymptomatic, but may be associated with UTI and stone formation in up to 39% of cases (Fig. 2). The management is usually conservative, but indications for surgery include persistent pain, UTI, and nephrolithiasis. Open surgery has been largely replaced by percutaneous, uretero-renoscopic or laparoscopic approaches to obliterate the diverticulum or to dilate the connection between the collecting system and the diverticulum to improve drainage [28].

## Bacteriology

Urinary tract abnormalities may predispose to infection with organisms other than *E coli*, and long-term antibiotic therapy often leads to bacterial resistance or fungal superinfection with *Candida albicans* [29]. Urinary tract obstruction may permit ascending infection of *E coli* strains with lower adhesive ability. P-fimbriae are mannose-resistant adhesins of uropathogenic *E coli* which cause acute pyelonephritis (APN). The pap gene cluster (PapGI, -II, and -III) encodes the proteins required for P-fimbrial biogenesis [30]. The incidence of the PapG class II allele in *E coli* infecting adult patients with hydronephrosis is lower than in *E coli* infecting patients without urinary tract abnormalities (69% vs. 93%). This indicates that structural or functional abnormalities of the urinary tract may permit infection with *E coli* strains that would not be pathogenic in a normal urinary tract [31].

**Table 1** Indications for imaging of patients with UTI [18, 29, 32]

| |
|---|
| Babies and children |
| Men (see text for exceptions) |
| Patients with history of: |
| Voiding difficulties |
| Urolithiasis |
| Previous renal disease |
| Previous urinary tract surgery |
| Neurological disease/neuropathic bladder |
| Poor response to appropriate antibiotic treatment (after 3–6 days) |
| Recurrent or unusually severe symptoms |
| Diabetes mellitus or immunocompromised states |
| Renal failure |
| Hematuria (macroscopic, or microscopic >1 month after UTI) |
| Acute urinary retention |
| Infection with urea-splitting bacteria |

## Special investigations

The aim of imaging in UTI is to detect conditions that must be corrected to avoid rapid deterioration of renal function or systemic sepsis and to prevent recurrent infections and long-term kidney damage. Indications for imaging in patients with UTI are shown in the Table 1 [18, 29, 32].

Intravenous urography (IVU) and ultrasound (US) have traditionally been used in the assessment of complicated UTI. US with abdominal radiography (AXR/KUB) is as accurate as IVU in detecting important urological abnormalities in men presenting with UTI.

Computerized tomography (CT) has become accepted as a more sensitive and specific modality. CT urography is increasingly performed as a comprehensive urinary tract imaging study. Magnetic resonance (MR) imaging is particularly useful in those with iodinated contrast allergies. Nuclear medicine has a limited role but is useful in the assessment of renal function prior to surgery [18]. Imaging with microbubble ultrasonographic contrast agents can overcome the limitations of conventional B-mode imaging and may become the modality of choice in the future [33].

It is generally accepted that urological evaluation should be carried out in men with febrile UTI, pyelonephritis or recurrent infection, or whenever a complicating factor is suspected. However, imaging may not be necessary in all men with febrile UTI. It has been suggested that in men younger than 45 years with a first acute UTI no radiologic, endoscopic or urodynamic investigation is required, provided a urethral stricture has been excluded [34].

In patients with complicated APN, urine cultures are positive in 90–98% of cases and bacteremia may occur in 21–42%, but in only a small minory (around 1%), the pathogens found in blood cultures are different from those in the urine [35]. Some authors have suggested that blood cultures should be reserved for patients with an uncertain diagnosis, those who are immunocompromised, and those who do not respond promptly to treatment. However, others recommend that blood cultures should be done in all patients with complicated APN, because bacteremia indicates severe disease, which is more likely to recur within 6 months in patients with non-*E coli* bacteremia and those with urolithiasis or hydronephrosis, especially men [35].

## Antibiotic treatment

The treatment of UTI in the presence of urinary tract obstruction requires effective antibiotic therapy as well as appropriate Urological intervention to remove predisposing factors and to restore as far as possible the normal anatomy and function of the urinary tract in order to prevent septicemia and recurrent UTI [18].

Severely ill patients with possible urosepsis should be hospitalized, and empirical treatment may include intravenous ampicillin and gentamicin or alternatives such as ciprofloxacin, levofloxacin, ceftriaxone, aztreonam, ticarcillin-clavulanate or imipenem-cilastin. The choice of empirical antibiotic treatment should be according to local guidance or policy. Indiscriminate use of quinolones and cephalosporins is strongly discouraged because of increasing bacterial resistance. Every effort should be made to correct any underlying urinary tract abnormalities that may compromise treatment efficacy. Therapy is usually switched from parenteral to oral as soon as possible [18].

Empirical therapy of serious UTI should usually include an intravenous antipseudomonal agent. Targeted therapy should be initiated once susceptibility data are known. Agents commonly prescribed include aminoglycosides, beta-lactamase inhibitor combinations, imipenem, advanced-generation cephalosporins, and fluoroquinolones. Several pivotal clinical trials support the use of fluoroquinolones for serious UTIs, with ciprofloxacin the most frequently studied drug [36].

A 7-day course of therapy is recommended for women with symptoms of 1 week or more (i.e., complicated UTI), men (even those with apparently uncomplicated cystitis), and individuals with possible complicating factors. For patients with fever or more severe systemic infection, therapy for 10–14 days is recommended. Urine cultures should be performed prior to initiation of antibiotic therapy, during treatment if clinical response is unsatisfactory, and 7–14 days after cessation of therapy to determine whether treatment has been effective [1, 18, 34].

## Conclusions

Despite the lack of evidence based on prospective randomized controlled clinical trials, logic dictates that in patients with UTI and obstruction, e.g., pyocystis or pyonephrosis, targeted antibiotic treatment should be complemented with emergency drainage by means of transurethral bladder catheterization, ureteric stenting or percutaneous nephrostomy, followed by definitive surgery to remove the cause of obstruction or stasis once infection is under control.

Further study is required to define the role, if any, for antibiotic prophylaxis in urinary tract obstruction, dilatation or stasis without infection, taking into consideration the risk of bacterial resistance and superinfection with highly resistant organisms. Further study is indicated to better define, in different populations and countries, the optimal type and duration of drainage of the infected, obstructed urinary tract. Given the increasing incidence of bacterial resistance due to indiscriminate use of antibiotics, further research is required to develop new antibacterial agents.

**Conflict of interest** None.

## References


1. Lipsky BA (1989) Urinary tract infections in men. Epidemiology, pathophysiology, diagnosis, and treatment. Ann Intern Med 110(2):138–150
2. Johnson DE, Russell RG, Lockatell CV, Zulty JC, Warren JW (1993) Urethral obstruction of 6 hours or less causes bacteriuria, bacteremia, and pyelonephritis in mice challenged with "nonuropathogenic" *Escherichia coli*. Infect Immun 61(8):3422–3428
3. Riehmann M, Goetzman B, Langer E, Drinka PJ, Rhodes PR, Bruskewitz RC (1994) Risk factors for bacteriuria in men. Urology 43(5):617–620
4. Payne SR, Timoney AG, McKenning ST, den Hollander D, Pead LJ, Maskell RM (1988) Microbiological look at urodynamic studies. Lancet 2:1123–1126
5. Holtgrewe HL, Mebust WK, Dowd JB, Cockett AT, Peters PC, Proctor C (1989) Transurethral prostatectomy: practice aspects of the dominant operation in American urology. J Urol 141:248–253
6. McConnell JD, Roehrborn CG, Bautista OM, Andriole GL Jr, Dixon CM, Kusek JW, Lepor H, McVary KT, Nyberg LM Jr, Clarke HS, Crawford ED, Diokno A, Foley JP, Foster HE, Jacobs SC, Kaplan SA, Kreder KJ, Lieber MM, Lucia MS, Miller GJ, Menon M, Milam DF, Ramsdell JW, Schenkman NS, Slawin KM, Smith JA, Medical Therapy of Prostatic Symptoms (MTOPS) Research Group (2003) The long-term effect of doxazosin, finasteride and the combination on the clinical progression of benign prostatic hyperplasia. N Engl J Med 349(25):2387–2398
7. Miller J, Ludwig M, Schroeder-Printzen I, Schiefer HG, Weidner W (1996) Transurethral laser therapy and urinary tract infections. Ann Urol (Paris) 30(3):131–138
8. Nickel JC, Downey J, Young I, Boag S (1999) Asymptomatic inflammation and/or infection in benign prostatic hyperplasia. BJU Int 84(9):976–981. Comment in: BJU Int 2000;85(9):1155–1156
9. Ohkawa M, Shimamura M, Tokunaga S, Nakashima T, Orito M (1993) Bacteremia resulting from prostatic surgery in patients with or without preoperative bacteriuria under perioperative antibiotic use. Chemotherapy 39(2):140–146
10. Hochreiter WW (2008) The issue of prostate cancer evaluation in men with elevated prostate-specific antigen and chronic prostatitis. Andrologia 40(2):130–133
11. König JE, Senge T, Allhoff EP, König W (2005) Analysis of the inflammatory network in benign prostate hyperplasia and prostate cancer. Prostate 2004;58(2):121–129. Comment in: J Urol 173(1):161
12. Santucci RA, Joyce GF, Wise M (2007) Male urethral stricture disease. J Urol 177(5):1667–1674
13. Blacklock AR, Geddes JR, Shaw RE (1983) The treatment of large bladder diverticula. Br J Urol 55:17–20
14. Pieretti RV, Pieretti-Vanmarcke RV (1999) Congenital bladder diverticula in children. J Pediatr Surg 34:468–473
15. Adot Zurbano JM, Salinas Casado J, Dambros M, Virseda Chamorro M, Ramírez Fernández JC, Silmi Moyano A, Marcos Díaz J (2005) Urodynamics of the bladder diverticulum in the adult male [Article in Spanish] Arch Esp Urol 58(7):641–649
16. Daneshgari F, Zimmern PE, Jacomides L (1999) Magnetic resonance imaging detection of symptomatic noncommunicating intraurethral wall diverticula in women. J Urol 161:1259–1261
17. Romanzi LJ, Groutz A, Blaivas JG (2000) Urethral diverticulum in women: diverse presentations resulting in diagnostic delay and mismanagement. J Urol 164:428–433
18. Schaeffer AJ, Schaeffer EM (2007) Infections of the urinary tract. In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA (eds) Campbell-Walsh Urology, Vol 1, 9th edn. Elsevier, Philadelphia, Chapter 8, pp 221–303
19. Cox CE, Hinman F Jr (1961) Experiments with induced bacteriuria, vesical emptying and bacterial growth on the mechanism of bladder defense to infection. J Urol 86:739–748
20. Smellie J, Edwards D, Hunter N, Normand IC, Prescod N (1975) Vesico-ureteric reflux and renal scarring. Kidney Int Suppl 4:S65–S72
21. Beeson PB, Guze LB (1956) Experimental pyelonephritis: I. Effect of ureteral ligation on the course of bacterial infection in the kidney of the rat. J Exp Med 104:803–815
22. Bitz H, Darmon D, Goldfarb M, Shina A, Block C, Rosen S, Brezis M, Heyman SN (2001) Transient urethral obstruction predisposes to ascending pyelonephritis and tubulo-interstitial disease: studies in rats. Urol Res 29(1):67–73
23. Song SH, Lee SB, Park YS, Kim KS (2007) Is antibiotic prophylaxis necessary in infants with obstructive hydronephrosis? J Urol 177(3):1098–1101; discussion 1101
24. Coelho GM, Bouzada MC, Lemos GS, Pereira AK, Lima BP, Oliveira EA (2008) Risk factors for urinary tract infection in children with prenatal renal pelvic dilatation. J Urol 179(1):284–289
25. Lee JH, Choi HS, Kim JK, Won HS, Kim KS, Moon DH, Cho KS, Park YS (2008) Nonrefluxing neonatal hydronephrosis and the risk of urinary tract infection. J Urol 179(4):1524–1528
26. Roth CC, Hubanks JM, Bright BC, Heinlen JE, Donovan BO, Kropp BP, Frimberger D (2009) Occurrence of urinary tract infection in children with significant upper urinary tract obstruction. Urology 73(1):74–78
27. Eknoyan G, Qunibi WY, Grissom RT, Tuma SN, Ayus JC (1982) Renal papillary necrosis: An update. Medicine (Baltimore) 61:55–73
28. Gross AJ, Herrmann TR (2007) Management of stones in calyceal diverticulum. Curr Opin Urol 17(2):136–140
29. Roberts JA (1999) Management of pyelonephritis and upper urinary tract infections. Urol Clin North Am 26(4):753–763
30. Lane MC, Mobley HL (2007) Role of P-fimbrial-mediated adherence in pyelonephritis and persistence of uropathogenic *Escherichia coli* (UPEC) in the mammalian kidney. Kidney Int 72(1):19–25

31. Tseng CC, Huang JJ, Ko WC, Yan JJ, Wu JJ (2001) Decreased predominance of papG class II allele in *Escherichia coli* strains isolated from adults with acute pyelonephritis and urinary tract abnormalities. J Urol 166(5):1643–1646
32. Johansen TE (2004) The role of imaging in urinary tract infections. World J Urol 22(5):392–398
33. Mitterberger M, Pinggera GM, Colleselli D, Bartsch G, Strasser H, Steppan I, Pallwein L, Friedrich A, Gradl J, Frauscher F (2008) Acute pyelonephritis: comparison of diagnosis with computed tomography and contrast-enhanced ultrasonography. BJU Int 101(3):341–344
34. Abarbanel J, Engelstein D, Lask D, Livne PM (2003) Urinary tract infection in men younger than 45 years of age: is there a need for urologic investigation? Urology 62(1):27–29
35. Hsu CY, Fang HC, Chou KJ, Chen CL, Lee PT, Chung HM (2006) The clinical impact of bacteremia in complicated acute pyelonephritis. Am J Med Sci 332(4):175–180
36. Wagenlehner FM, Naber KG (2006) Current challenges in the treatment of complicated urinary tract infections and prostatitis. Clin Microbiol Infect 12(Suppl 3):67–80
37. Naber KG, Schaeffer AJ, Heyns CF, Matsumoto T, Shoskes DA, Bjerklund Johansen TE (eds) (2010) Urogenital infections. European Association of Urology–International Consultation on Urological Diseases, 1st edn. Arnhem, The Netherlands, ISBN: 978-90-79754-41-0