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Pediatric urolithiasis: what can pediatricians expect from radiologists?

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Abstract

The incidence of urolithiasis in children has increased over the two last decades. Urolithiasis formation results from urine oversaturation following insufficient water intake, urinary obstruction (notably in cases of congenital uropathies), excess production of an insoluble compound, or imbalance between crystallization promoters and inhibitors. Whereas most urolithiases in adults occur secondary to environmental factors, in children, secondary causes are far more frequent, and 15% are related to genetic causes, most often monogenic. This is especially true in recurrent forms, with early and rapid progression and bilateral stones, and in cases of familial history or consanguinity. Because of differing clinical management, one should rule out cystinuria, primary hyperoxaluria and renal tubular acidosis, among other causes of urolithiasis. As such, a complete biochemical evaluation must be performed in all cases of pediatric urolithiasis, even in cases of an underlying uropathy. Ultrasound examination is the first-line modality for imaging pediatric urolithiasis, allowing both diagnosis (urolithiasis and its complications) and follow-up. US examination should also explore clues to an underlying cause of urolithiasis. This review is focused on the role of imaging in the management and etiological assessment of pediatric urolithiasis. Radiologists play an important role in pediatric urolithiasis, facilitating diagnosis, follow-up and surgical management. A trio of clinicians (pediatric nephrologist, pediatric surgeon, pediatric radiologist) is thus necessary in the care of these pediatric patients.

Keywords Child · Congenital · Kidney · Stone · Ultrasound · Urinary tract · Urolithiasis

Introduction

Urolithiasis formation is generally multifactorial and results from urine oversaturation following insufficient water intake, urinary obstruction (notably in cases of congenital uropathies), excess production of an insoluble compound, or imbalance between crystallization promoters and inhibitors. In 2012, the lifetime risk of urolithiasis was approximately 23% for men and 15% for women [1]. The incidence of urolithiasis is greater among adults (estimated to be 2,054 per 100,000 adults per year in 2018 in the United States [2]) than in children (estimated at 54 per 100,000 per year in 2016

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in the United States [3]). However, the pediatric incidence has increased; for instance in children from South Carolina the incidence of urolithiasis was reported to be 7.9 per 100,000 children in 1997 and 18.5 per 100,000 children in 2012 [4]. This might be explained by changes in living conditions, dietary habits and the increasing prevalence of obesity [5].

Children of all ages are affected, but the median age of presentation is 4.4 years in boys and 7.3 years in girls [6]. Clinical features vary according to age and are mostly non-specific [7]. For example, Alpay et al. [8] reported that urinary tract infection occurs in 27% of children with urolithiasis younger than 1 year and that this frequency decreases progressively with increasing age. Between 1 year and 5 years of age, the occurrence of macroscopic hematuria leads to the diagnosis of urolithiasis in a third of children, and after 5 years of age the classic signs of renal colic are found in the majority of patients [8]. Regardless of age, 24% of urolithiasis cases are incidentally diagnosed on imaging [8].

The frequency of underlying metabolic abnormalities is high in children (62–87%) [8, 9]. However, the etiology of urolithiasis in children is most frequently secondary; 15% of these cases are related to genetic causes, most often monogenic [10]. These genetic diseases are associated with familial history of urolithiasis or consanguinity, and recurrent forms of urolithiasis that are characterized by early and rapid progression and bilateral stones [11]. Among the genetic etiologies, primary hyperoxaluria type 1 is the most severe disease and can be found during the assessment of acute or chronic kidney failure [12]. A complete biochemical evaluation in all cases of pediatric urolithiasis, even in cases of an underlying uropathy, is therefore important to diagnose primary hyperoxaluria, but also to exclude other potential causes of urolithiasis, for example cystinuria, renal tubular acidosis and vitamin-D-related hypercalciuric lithiasis. In this paper, after detailing the pathophysiology and first-line biochemical assessment, we focus on the role of imaging in the management and etiological assessment of pediatric urolithiasis. Adult imaging benefits from the development of techniques such as dual-energy CT, and we also discuss the role of these imaging modalities in pediatric urolithiasis.

Pathophysiology of pediatric urolithiasis

Urolithiasis consists of an agglomeration of crystals bound to an organic matrix that is driven by a combination of several factors [11]. Nucleation, corresponding to the initial formation of a crystal nidus, is caused by an imbalance between the promoters and inhibitors of crystallization [11, 13]. Among the promoters, urinary tract retention leads to urinary oversaturation, which is an increased concentration of crystallization promoters (e.g., calcium, oxalate, phosphate, uric acids/urate, dibasic amino-acids, notably cystine and purines such as xanthine) that leads to crystal growth and stone formation; in addition, low urine volume and intrinsic metabolic abnormalities might be related to an increased urinary excretion of crystallization promoters [14]. In children, urolithiases are most often located in the kidneys and the upper urinary tract, more rarely in the bladder (except in cases of an underlying uropathy), and are bilateral in a quarter of cases [8]. Poor/inadequate diets such as excess protein intake, high-fat products, salty food and excessive vitamin C and D intake can increase urinary calcium [15]. In contrast, a low-calcium diet might promote enteric hyperoxaluria. Monogenic diseases can be responsible for excessive crystallization promoters and should be investigated according to the context. Conversely, a lack of citrate and magnesium, and high or low urinary pH can inhibit urolithiasis growth, but the mechanisms involved remain incompletely understood [16]. Taken together, the competition between urinary oversaturation and crystallization inhibitors determines crystal formation and ultimately urolithiasis [17]. In terms of pathophysiology of stone growth, there are two hypotheses: it occurs from either a tubular agglomeration of crystals or an agglomeration of crystals from sub-epithelial interstitial Randall plaques in the renal medulla [18].

Imaging in pediatric urolithiasis

Imaging impacts management by specifying the size and the location of the urolithiasis, the presence of urinary tract obstruction or other complications, and possibly a potential underlying etiology.

Ultrasound examination

The widespread availability and absence of radiation exposure makes US the first-line imaging modality for the diagnosis and follow-up of urolithiasis [19]. Indeed, US meets the ALARA (as low as reasonably achievable) principle, which is the guiding principle of radiation safety and is even more important in pediatric imaging because children's tissues are highly radiosensitive and cumulative doses across years can be significant in a patient with imaging early in life. US can detect urolithiases regardless of their chemical nature or radiodensity. A urolithiasis is more readily visible if it is large and located in the dilated renal pelvis, the ureteral meatus or full bladder (if intravesical). Lumbar ureteral locations are more difficult to access because of interposition of bowel and other anatomical structures.

On US, urolithiasis appears as an echogenic focus in the urinary tract with posterior acoustic shadowing on two-dimensional (2-D) images (Fig. 1). The twinkling artifact is

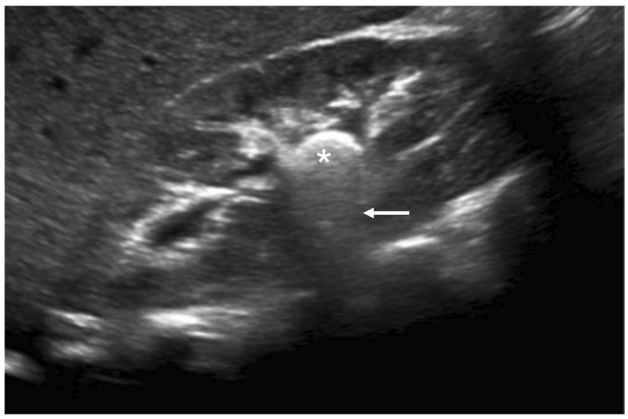

Fig. 1 US examination in a 5-year-old girl with urolithiasis. Longitudinal image shows urolithiasis in the right kidney. Urolithiasis appears as a round echogenic formation (*) in the urinary tract with posterior acoustic shadowing (*arrow*)

a useful sensitive and specific sign, particularly for detecting small intrarenal urolithiases [19]. Twinkling artifact is observed on color Doppler examination, corresponding to a rapidly changing mixture of colors deep to a strong reflector with an acoustically efficient rough surface (Fig. 2) [20]. Revelation of a twinkling artifact is dependent on color Doppler settings: when looking for this artifact to detect intrarenal urolithiasis, one should remove background color Doppler blood flow in the kidney using the highest pulse repetition frequency (PRF) [20]. Although the results of US are operator-dependent, measurements of kidney size and pelvicalyceal dilatation should be reproducible between operators.

Radiography of the abdomen

Radiography of the abdomen is less frequently coupled with US to confirm the diagnosis and to follow radiopaque urolithiasis. Radiographs show a density projecting over the kidneys, ureters or bladder, sometimes better seen in the decubitus position. The distinction between radiopaque and radiolucent urolithiasis is defined on standard radiography, with radiolucent stones not being visible on radiographs of the abdomen irrespective of their size. Calcium urolithiases are radiopaque, cystine and struvite lithiases appear weakly radiopaque, while uric acid and drug-induced lithiases appear radiolucent. Note that intravenous urography is no longer used in pediatrics [21].

Cross-sectional imaging

Children undergoing management for urolithiasis are at risk of significant radiation exposure [19]. CT being a radiating imaging modality, its use is more limited in pediatric

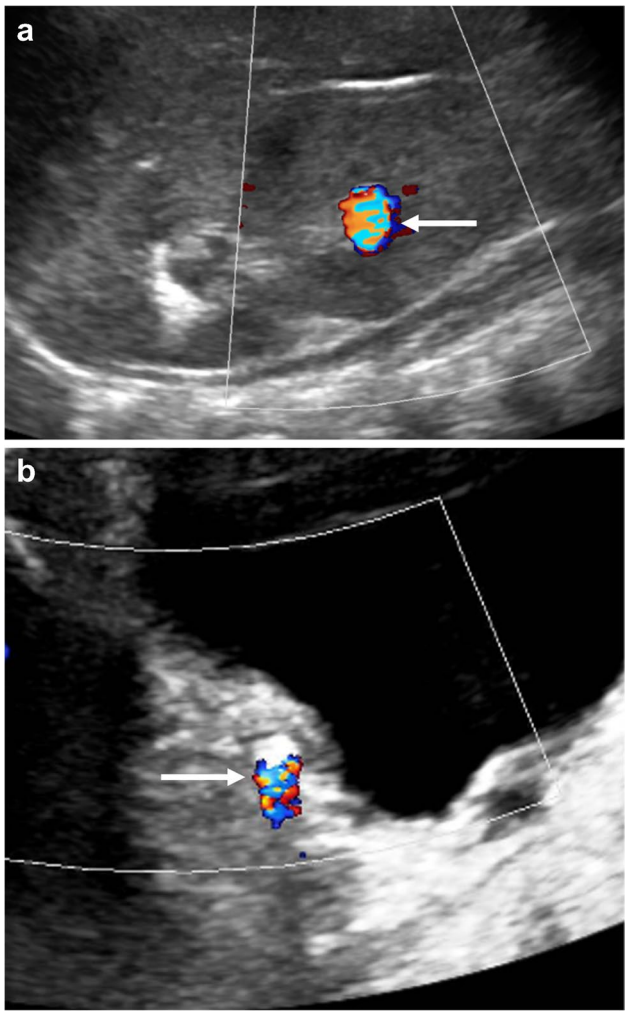

Fig. 2 Twinkling artifact at US examination in a 5-year-old girl with urolithiasis. **a, b** Longitudinal images show twinkling artifact in an intrarenal (**a**) and a meatal (**b**) urolithiasis on color Doppler examination (*arrow*). It appears as a mixture of colors deep to the urolithiasis

urolithiasis diagnosis compared to its use in adults, and CT is not recommended for initial imaging in this context. CT is sometimes indicated after US in cases with positive indirect signs (such as urinary tract dilatation in an acute pain context) without urolithiasis detected on US; in cases of negative US with discordant clinical and imaging findings; for surgical planning allowing an accurate anatomical assessment, particularly in cases of multiple urolithiases; or for assessing complications [5, 22]. If CT is needed, use of low-dose protocols adapted to the age and weight of the child allow for reduced radiation exposure without impacting the sensitivity or specificity for urolithiasis detection compared to standard CT [19]. Prone CT, when possible, is superior to supine CT for distinguishing intramural ureterovesical junction lithiasis from stones that have already passed into

the bladder [23]. CT for urolithiasis is a rapidly performed examination without contrast agent and is possible in most cases without sedation, unlike MRI. On the other hand, MR urography, another non-irradiating technique, can be useful when information about the anatomy of the renal collecting system is required for surgical planning. CT urography is not usually used in pediatrics because of hazards of ionizing radiation associated with multiphasic acquisition.

On non-contrast CT, urolithiasis appears as a focal hyperdensity in the urinary tract, and CT has high sensitivity (91–100%) for its detection and diagnosis [24]. The soft-tissue rim sign can be used to distinguish a ureteral lithiasis from a phlebolith because the edematous ureteral wall is visible surrounding the lithiasis [25]. Urolithiasis attenuation on non-contrast CT can be used to differentiate several stone compositions (Hounsfield units) [26]. For example, struvite, cystine and uric acid urolithiases are less dense than calcium urolithiases. Urolithiasis composition can be specifically assessed through the use of dual-energy CT [27], and dual-energy CT has been shown to be useful for predicting extracorporeal shock wave lithotripsy in pediatrics [28]. If a CT is performed, signs of obstruction (Fig. 3) include:

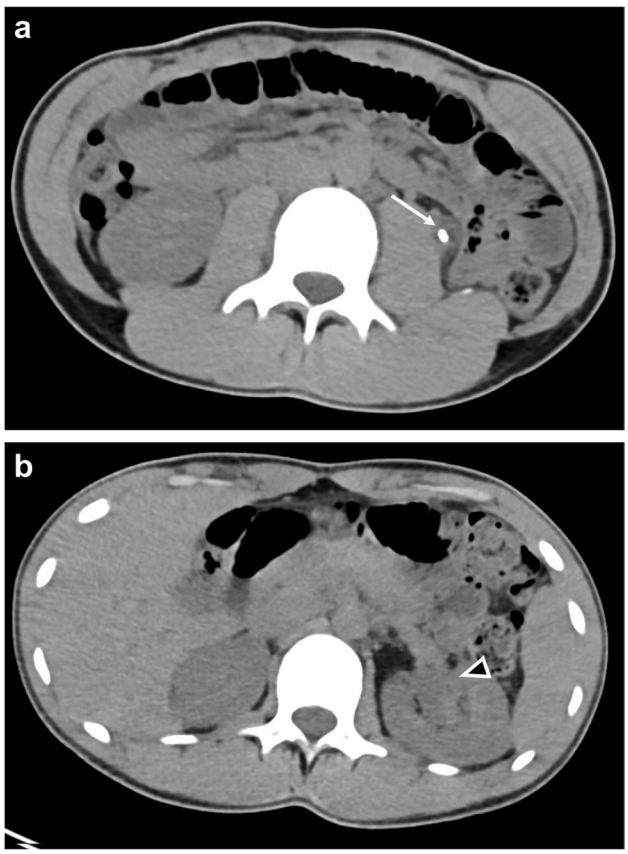

Fig. 3 CT in an 18-year-old man with obstructive urolithiasis. **a, b** Axial non-contrast CT images show an obstructive urolithiasis in the left ureter (*arrow* in **a**) with upstream dilatation of the urinary tract (*arrowhead* in **b**). Note that the radiodense focus on the left quadratus lumborum muscle (**a**) corresponds to surgical material following a left partial nephrectomy

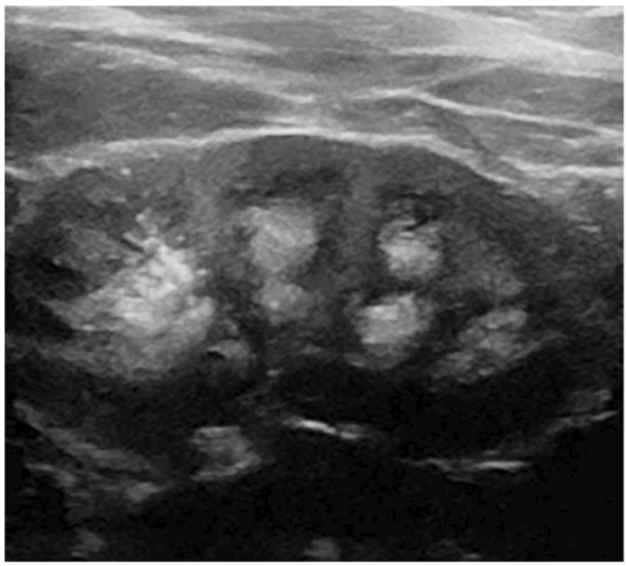

Fig. 4 Nephrocalcinosis on longitudinal US examination in a 5-month-old boy. Nephrocalcinosis appears as hyperechogenicity of the renal parenchyma (pyramids), and is responsible for an inverted cortico-medullary differentiation

- dilatation of the urinary tract upstream of the urolithiasis,
- increased size of the kidney on the pathological side,
- fluid infiltration around the ipsilateral kidney or ureter and
- moderate decrease in pyramid density.

Differential diagnoses

The main differential diagnoses of urolithiasis on imaging include nephrocalcinosis, transient renal medullary hyperechogenicity in newborns, fungus balls, pelvi-ureteric junction syndrome, vesicoureteral reflux, dense feces, renal microcysts, sickle cell disease, intrarenal gas and other calcifications of the urinary tract [20, 29].

Nephrocalcinosis corresponds to calcifications in the renal parenchyma, usually of the pyramids, and is responsible for an inverted cortico-medullary differentiation. Nephrocalcinosis can be associated with urolithiasis, especially in cases of monogenic disorders such as primary hyperoxaluria, distal renal tubular acidosis and vitamin D hypersensitivity (Fig. 4).

Transient renal medullary hyperechogenicity in newborns is a physiological observation on US during the first weeks after birth.

Fungus balls are caused by renal fungal infection such as *Candida albicans*. Fungus balls can be obstructive or non-obstructive (Fig. 5) and require long-term management.

Pelvi-ureteric junction syndrome corresponds to a chronic dilatation without urolithiasis. Vesicoureteral reflux can cause

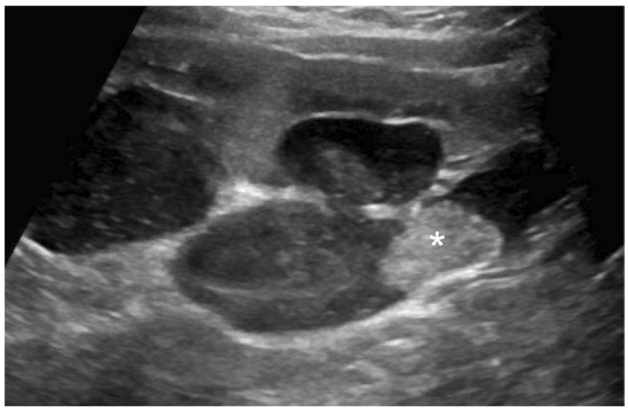

Fig. 5 Fungus ball in a 7-month-old boy. Longitudinal US examination shows an obstructive fungus ball in the context of *Candida albicans* infection, visible as a round mass (*) in the inferior calyx

urinary tract dilatation without obstruction. Dense feces can also mimic urolithiasis on abdominal standard radiography.

Renal microcysts appear as focal hyperechogenicity because of their numerous acoustic interfaces. Interrogation with a superficial probe is an essential feature of US examination to identify microcysts.

Sickle cell disease is also in the differential diagnosis because it is sometimes associated with medullary hyperechogenicity not to be confused with nephrocalcinosis [30] (Fig. 6).

Intrarenal gas on US is often more mobile than urolithiasis. Finally, other calcifications of the urinary tract such as renal tuberculosis, schistosomiasis-associated kidney disease and calcified renal cyst should be considered in the differential diagnosis for urolithiasis.

Purpose and impact of imaging in pediatric urolithiasis

The first step after detecting urolithiasis on imaging is to determine whether the lithiasis is obstructive and whether

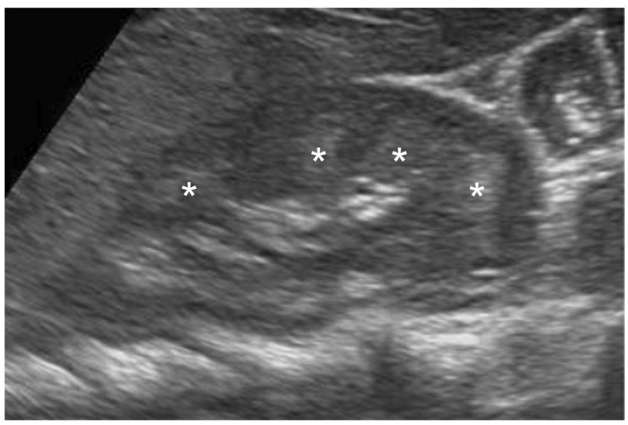

Fig. 6 Sickle cell disease in a 5-year-old boy. Longitudinal US examination shows medullary hyperechogenicities (*) associated with sickle cell disease

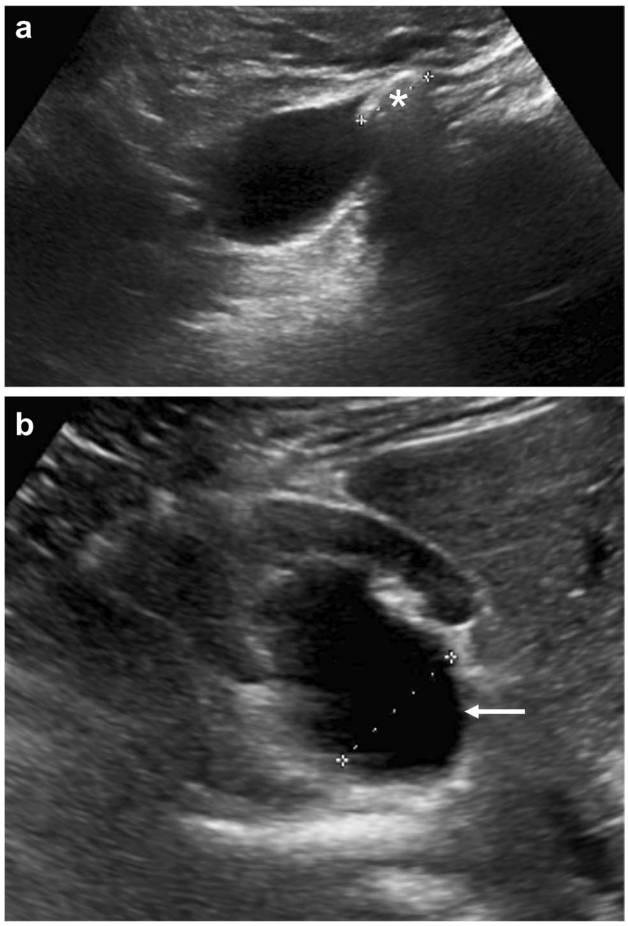

Fig. 7 Obstructive urolithiasis on US. **a, b** Axial US images in a 5-year-old girl show obstructive urolithiasis of the upper urinary tract (* in **a**) with upstream dilatation (*arrow* in **b**)

emergency urinary decompression is needed, especially in cases of associated pyelonephritis, acute kidney failure or drug-resistant pain [5]. In these cases, percutaneous nephrostomy under US and radiologic guidance by an interventional radiologist might be necessary to allow urine flow before managing the urolithiasis. To determine whether the urolithiasis is obstructive, look for the following signs on US [29]:

- dilatation of the urinary tract upstream of the urolithiasis (Fig. 7),
- increased size of the kidney on the pathological side,
- fluid infiltration around the kidney or the ureter, which might correspond to a urinoma (signifying urinary tract rupture),
- absence of ureteral jet into the bladder using color Doppler and
- high-resistance renal arterial flow using pulsed wave Doppler, which may precede the dilatation.

Both size and location of the urolithiasis are important points to assess on imaging because urolithiases smaller

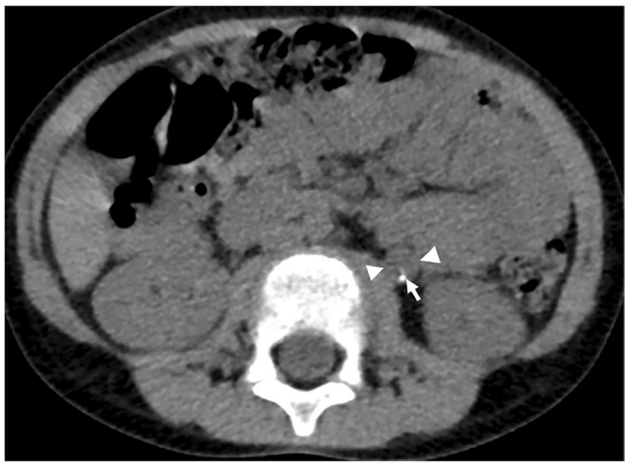

Fig. 8 Urolithiasis in association with urinary tract malformation. Axial non-contrast CT in a 5-year-old girl shows a non-obstructive round urolithiasis (*arrow*) in a left megaureter (*arrowheads*)

than 5 mm are usually evacuated spontaneously. In cases of urolithiases located in the upper urinary tract that persist after medical treatment, extracorporeal lithotripsy is proposed, while ureteral lithiasis is accessible to ureteroscopy. After treatment, the search for a residual urolithiasis is also important because it may favor recurrent urolithiasis formation (see prior section on pathophysiology). Imaging may show signs of chronic kidney disease which may either be a complication of untreated obstructive urolithiasis or related to an underlying disease [31].

In addition to detecting urolithiasis and its complications, US must be used to search for clues to an underlying disease promoting the formation of urolithiases. An underlying cause should be suspected on imaging in cases of nephrocalcinosis and in radiolucent, bilateral or multiple urolithiases [29].

A urinary tract malformation such as megaureter, horseshoe kidney, pelvi-ureteric junction syndrome or ureteral duplicity is present in a third of urolithiasis patients [32]. Consider such urolithiases in the presence of mobile, multiple, round and weakly dense lithiasis (Fig. 8) [32]. Detection of an associated urinary tract malformation may modify the treatment the child receives.

Following physical and radiologic evaluation, children should be referred to a pediatric urologist in the following select situations. First, a urologist might be involved in emergency cases — such as when renal colic is complicated by acute pyelonephritis or there is acute renal failure or resistant flank pain — to consider ureteral stent placement prior to percutaneous nephrostomy. Second, children might need scheduled intervention to remove persistent stones over 5 mm not naturally flushed out despite medical observational management of several weeks. Finally, endoscopic and percutaneous techniques, known as minimally invasive approaches, have emerged as standards over the last decades [5, 7, 33]; however, in a recent review of the American pediatric urolithiasis population, only 22% of children required surgery to achieve a stone-free state [34].

Biochemical assessment

When a child presents a first urolithiasis episode, a consultation with a pediatric nephrologist is highly recommended. Even when there is an underlying uropathy or the composition of the urinary stone is known, a complete biochemical assessment including blood and urine tests should be performed. Family history, early onset, consanguinity and recurrent or bilateral stones are strong pointers in favor of a genetic etiology [7]; urological abnormalities, drugs, infectious conditions and intestinal malabsorption increasing intestinal oxalate absorption (for example in cases of short bowel syndrome, bariatric surgery, exocrine pancreatic insufficiency and Crohn disease) are potential secondary causes of urolithiasis and should also be investigated.

Where possible, the morpho-constitutional analysis provides a better understanding of lithogenesis and allows the medical management to be adapted to the cause [35]; it is based on Fourier-transform infrared spectrometry or X-ray diffraction [14]. According to the mineral composition, five types of kidney stones are described: calcium oxalate, carbapatite, uric acid, struvite (magnesium ammonium phosphate) and brushite [36]. Crystallography of fresh first morning urine, is used to detect the presence of crystals in the urine (crystalluria) and to assess the effects of preventive measures against recurrence [37]. This method can also be useful for diagnosing underlying genetic diseases and for detecting urine crystallization

Table 1 First-line blood and urine tests performed at first urolithiasis in children

Source	Biochemical tests
Blood	- Urea, creatinine, uric acid - Sodium, potassium, chloride, calcium, phosphate, magnesium, bicarbonate - Parathyroid hormone; 25-hydroxy vitamin D - Tubular maximum phosphate reabsorption per glomerular filtration rate (TmP/GFR) [39]
Urine	- Oxalates and amino acids - Cystine - Proteins - Beta-2 microglobulin - Sodium, potassium, chloride, calcium, phosphate, magnesium - Citrate - Uric acid

Table 2 Etiologies of secondary pediatric urolithiasis with imaging findings

Secondary urolithiasis	Etiologies	Stone contents	Imaging findings
With hypercalciuria	Dietary factors - High intake of sodium - Animal protein-rich diet - High fructose intake	Calcium oxalate	Radiopaque urolithiasis
With hyperoxaluria	Increasing intestinal oxalate absorption - Short bowel syndrome - Bariatric surgery - Exocrine pancreatic insufficiency - Crohn disease	Calcium oxalate	Radiopaque urolithiasis
Infections	Bacteria with urease activity - *Proteus mirabilis* - *Klebsiella pneumoniae* - *Pseudomonas* - *Staphylococcus aureus* - *Streptococcus pneumoniae*	Struvite or Carbapatite	Coral struvite lithiasis (Fig. 9)
Iatrogenic	Loop diuretics Vitamin D Vitamin C	Carbapatite	Radiopaque urolithiasis
Congenital abnormalities of kidney and urinary tract	- Ureteropelvic junction obstruction - Neurogenic bladder - Horseshoe kidney - Obstructive renal dysplasia	Struvite or Carbapatite	Coral struvite lithiasis Bladder stones

secondary to drugs [38]. This test is not available everywhere, and its results should be interpreted with caution, and considering urinary pH.

Blood and urine tests (24-h urine collection for a toilet-trained child; single voided urine sample in others; Table 1 [39]), performed at the same time, can be used to calculate the fraction excretions, modulated and interpreted according to the age of the child, clinical features and under normal dietary conditions, for each urolithiasis episode and after any urological intervention [14].

Table 3 Monogenic etiologies of pediatric urolithiasis with imaging findings

Monogenic causes	Etiologies	Stone contents	Imaging findings
With hypercalciuria	Primary hyperparathyroidism	Calcium oxalate	Radiopaque urolithiasis Nephrocalcinosis
	- Variants in 24 hydroxylase gene - Variants in renal phosphate transporter NPT2a - Variants in renal phosphate transporter NPT2c	Calcium oxalate	Radiopaque urolithiasis Nephrocalcinosis Prenatal hyperechogenic kidneys
	Tubular disorders - Renal Fanconi syndrome - Dent disease - Lowe disease - Fanconi–Bickel syndrome - Types I-II-IV-V Bartter syndrome	Calcium oxalate	Radiopaque urolithiasis Nephrocalcinosis
Cystinuria	- Type 1 - Type 2	Cystine	Radiopaque urolithiasis (Fig. 10) Antenatal hyperechoic colon
Distal renal tubular acidosis	Genetic abnormality: 80% of cases	Calcium phosphate	Radiopaque urolithiasis (Fig. 11) Nephrocalcinosis Renal cysts
Hyperoxaluria	- Type 1 - Type 2 - Type 3	Calcium oxalate	Radiopaque urolithiasis (Fig. 12) Nephrocalcinosis
Purine metabolism disorders	- Hypoxanthine-guanine phosphoribosyltransferase deficiency - Phosphoribosylpyrophosphate synthetase hyperactivity - Hereditary xanthinuria	Uric acid	Radiolucent urolithiasis

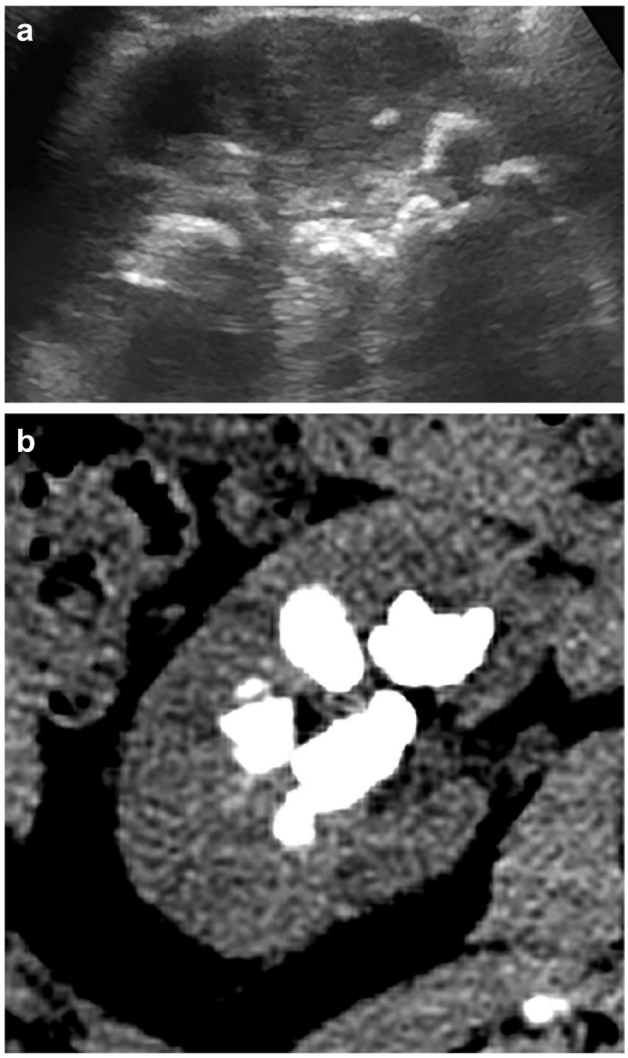

Fig. 9 Coral urolithiasis in an 18-year-old man. **a**, **b** Longitudinal US (**a**) and axial non-contrast CT (**b**) images show coral (staghorn) urolithiasis molding the renal pelvis and calyces

The level of 1.25-hydroxy vitamin D is not systematically determined in the initial biochemical assessment. However, in cases of hypercalciuria or low parathyroid hormone levels, this is required to investigate genetic conditions such as hypersensitivity to vitamin D (*CYP24A1* mutations) or secondary increase in 1.25-hydroxy vitamin D (*SLC34A1*, *SLC34A3* and *NHERF1* mutations) [40].

According to the results of the initial biochemical assessment, dynamic tests might be proposed, for example using oral calcium loads [41]. A nutritional evaluation also needs to be performed, ideally with the help of a pediatric renal dietician,

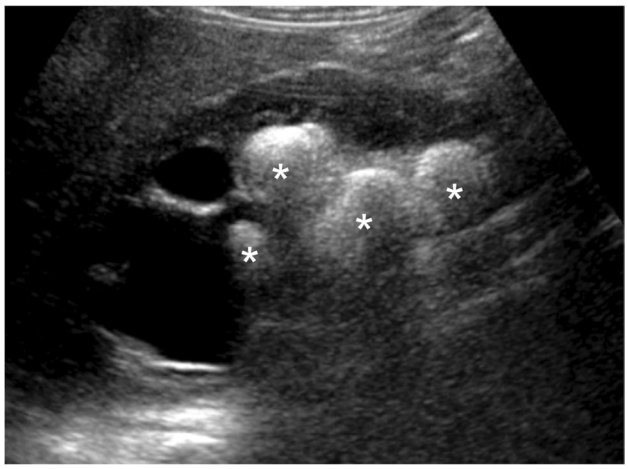

Fig. 10 Cystinuria in a 4-year-old boy. Longitudinal US image shows multiple urolithiases (*) that are both obstructive and non-obstructive

with a special focus on hydration, calcium, protein and sodium intakes. It is important to keep in mind that even in cases of overt hypercalciuria, nutritional calcium intake should be within the normal target for age in the majority of children [42].

Underlying diseases promoting the formation of urolithiasis

Rauturier et al. [43] reported that the main components of stones in children treated in a French tertiary center were calcium oxalate (weddellite 31% of stones, whewellite 21%), calcium phosphate (carbapatite 29%, brushite 5%, amorphous calcium phosphate 3%), struvite (5%), cystine (4%), uric acid (2%) and ammonium acid urate (2%). The authors also reported that one-component stones represented 18% of stones, while 22% of stones were associated with urinary infection; carbapatite stones were the most frequent in children younger than 2 years and calcium oxalate stones in children age 2 years and older. Metabolic abnormalities (most frequently hypercalciuria) were found in 50% of all tested patients and in 54% of patients with infectious stones [43].

Secondary urolithiasis is more common, occurring secondary to dietary factors, increased intestinal oxalate absorption or infection [5]. Infectious urolithiasis remains the most frequent in children younger than 5 years, with a male predominance [14, 15]; infection increases urinary pH, leading to the transformation of urea to ammonium, which enhances

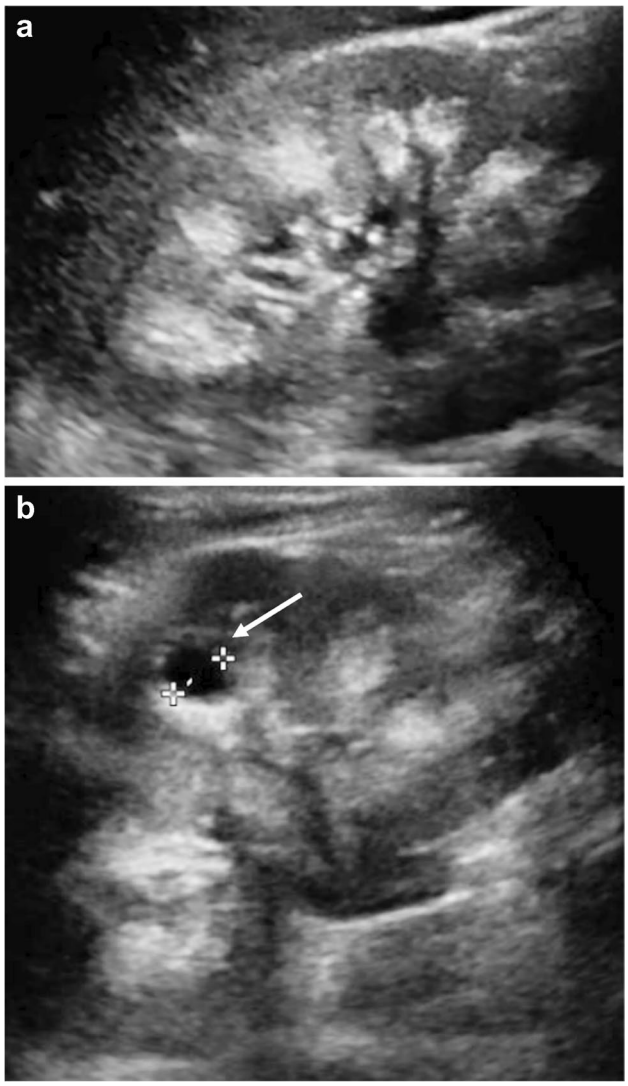

Fig. 11 Distal renal tubular acidosis in a 2-year-old girl. **a, b** Longitudinal US images show nephrocalcinosis (medullary hyperechogenicity in **a** and **b**) and renal cyst (*arrow* in **b**)

crystallization [15] — congenital abnormalities of kidney and urinary tract are risk factors for such urolithiases [15]. Tables 2 and 3 summarize the etiology and imaging findings of pediatric urolithiasis (Figs. 9, 10, 11 and 12).

Conclusion

This review not only underlines the fact that pediatricians (and ideally pediatric nephrologists) should prescribe a complete biochemical evaluation in all cases of pediatric urolithiasis, even in the context of an underlying uropathy, but also that radiologists play an important role in

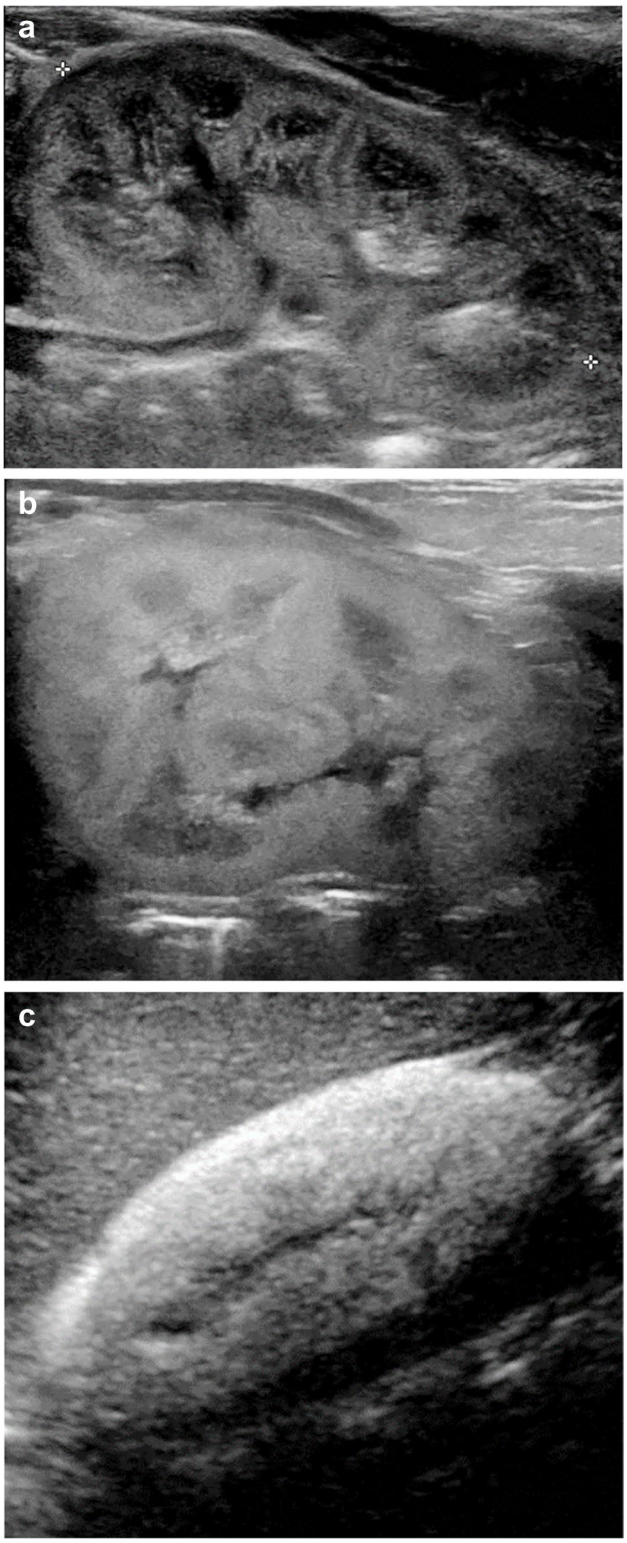

Fig. 12 Longitudinal US examination of the right kidney in a girl with primary hyperoxaluria. **a**–**c** US at 25 days old shows medullary nephrocalcinosis (**a**), at 5 weeks old, diffuse cortical hyperechogenicity (**b**), at 2 years old, progressive diffuse cortical hypherechogenicity and acoustic shadowing (**c**). Although only the right kidney is shown, multiple bilateral radiopaque urolithiases were evident

urolithiasis management. Pediatric nephrologists, pediatric surgeons and pediatric radiologists should therefore work as a trio for the care of these pediatric patients.

Declarations

Conflicts of interest None

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