Journal of Ultrasound (2019) 22:381–393
https://doi.org/10.1007/s40477-018-0347-9

PICTORIAL ESSAY

Pediatric cystic diseases of the kidney

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Received: 17 September 2018 / Accepted: 4 December 2018 / Published online: 1 January 2019

Abstract
Pediatric renal cystic diseases include a variety of hereditary or non-hereditary conditions. Numerous classifications exist and new data are continuously published. Ultrasound is the primary technique for evaluating kidneys in children: conventional and high-resolution US allows a detailed visualization of renal parenchyma and of number, size and location of the cysts, hence representing the most important diagnostic imaging technique for the first diagnosis and follow-up of these young patients. The purpose of this pictorial essay is to review the spectrum of renal cystic lesions in children from simple, complex or malignant single cysts to the several poly/multicystic kidney diseases.

Keywords Cystic renal disease · Kidney · Neonatal · Pediatric · Ultrasound

Sommario
La patologia cistica renale in età pediatrica è costituita da svariate condizioni ereditarie e non. La letteratura propone diverse classificazioni, in continua evoluzione. L'ecografia rappresenta la prima metodica per la valutazione del rene nel neonato e nel bambino: l'esame con metodica tradizionale e con sonde ad alta risoluzione consente uno studio dettagliato del parenchima renale e delle cisti per numero, dimensioni e localizzazione. L'ecografia è pertanto il cardine nell'iter diagnostico e nel *follow-up* di questi piccoli pazienti. Obiettivo di questo lavoro è rivedere le peculiarità delle lesioni cistiche renali in età pediatrica: dalla cisti singola, semplice o complessa, allo spettro delle patologie multi/policistiche.

Introduction

Neonatal and pediatric renal cystic diseases include a variety of hereditary or non-hereditary conditions, mono or bilateral, benign but also with a worse prognosis.

Several classifications of cystic kidney pathology have been proposed ranging from the historical classification of Potter to more recent ones, the latter mainly based on the distinction between hereditary and non-hereditary diseases (Table 1) [1]. Some diseases are part of the group of ciliopathies, hereditary disorders caused by mutation or the absence of genes that alter the structure and function of cilia: autosomal dominant polycystic kidney disease (ADPKD), autosomal recessive polycystic kidney disease (ARPKD), nephronophthisis (NPHP), and glomerulocystic kidney disease (GCKD). Others belong to the phakomatoses: a group of inherited conditions, mainly autosomal dominant, that affect structures derived from neuroectoderm, with distinctive skin and nervous stigmata, but also non-ectodermal involvement (liver, kidneys and pancreas) occurs especially in individuals with tuberous sclerosis (TS) and Von Hippel–Lindau syndrome (VHL) [1, 2].

The role of imaging, and especially of ultrasound (US), is to contribute to establishing or ruling out a diagnosis in the least invasive way for young patients.

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Table 1 Cystic renal diseases Adapted from Riccabona [1]

Non-hereditary cystic renal diseases
Simple renal cyst
Congenital
Acquired
Complex renal cyst
Cystic renal tumor
Multicystic dysplastic kidney (MCDK)
Obstructive cystic displasia
Medullary sponge kidney
Hereditary cystic renal diseases
Ciliopathies
Autosomal recessive polycystic kidney disease (ARPKD)
Autosomal dominant polycystic kidney disease (ADPKD)
Nephronophthisis
Glomerulocystic kidney disease (GCKD) HNF1B/TCF2-associated disease
Syndromic diseases
Tuberous sclerosis
Von Hippel–Lindau syndrome

An accurate echographic assessment of neonatal and pediatric renal cystic pathology examines both cystic characteristics and the general appearance of kidneys such as morphology, size, and echostructure (Fig. 1).

It is important to consider the size of kidneys (regular, reduced, and increased) according to the age, weight, and height of the child. The echogenicity of the cortex should be assessed, and attention should be paid to the corticomedullary differentiation by describing whether it is regular, increased, reduced, absent or even reversed due to the presence of hyperechogenicity of the medulla with respect to the cortex (Fig. 2).

At this point, the appearance of cysts should be considered, analyzing the number, size, localization, and extension [3]. Single and multiple cysts can have varying dimensions ranging from several centimeters to a few millimeters up to sub-millimetric cysts, bearing in mind that, in some cases, those are not detectable as they are smaller than the resolution of standard US probes (medullary sponge kidney, neonatal ADPKD or ARPKD) [4]. The second step is the assessment of their location in renal parenchyma noticing whether they are subcapsular/cortical at the cortico-medullary junction or in the medulla (Fig. 3). This is fundamental as some specific pathological forms can be distinguished according to the anatomical location of the cyst (ADPKD, ARPKD). Finally, it is important to establish whether cysts are diffusely or segmentally spread and renal involvement is mono or bilateral.

US of children affected by renal cystic diseases should not focus only on kidneys, but should include all abdominal organs, namely liver and pancreas that may be affected in some syndromes (VHL, TS, ARPKD). In some cases, it is necessary to extend US examination also to close relatives to confirm or exclude the diagnostic suspicion of some hereditary forms (ADPKD, NPHP) [5].

Simple cysts

Simple cysts are very rare in childhood, being found in a small percentage of cases (0.2–2% prevalence, 0.2–0.5% incidence) [6, 7], usually solitary and arising in the renal cortex. Sonographic findings are a well-defined anechoic formation with smooth, thin walls and posterior acoustic reinforcement, without signal at color and power Doppler (Fig. 4).

They are distinguished in asymptomatic and symptomatic forms (abdominal pain, hematuria etc.), but mainly asymptomatic cysts tend to grow slowly, though complications can arise from bleeding, infection or rupture, especially if large in younger children [7, 8].

Medical literature suggests a conservative management approach for those cases of asymptomatic simple renal cysts in children, running biannual clinical/ultrasound checks during the first year from diagnosis and annual checks for the following 10 years [6–8].

Cysts may disappear or show slight dimensional increase, approximately 0.3–1.6 mm/year in about 1–4% of the cases.

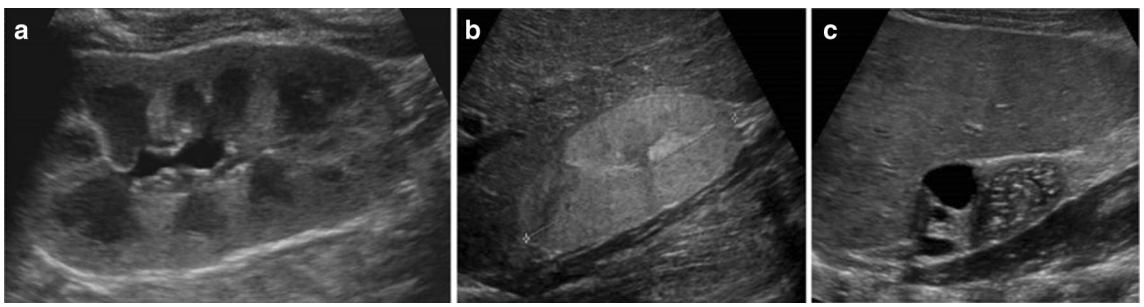

Fig. 1 Normal kidney (**a**) and two different aspects of dysplastic kidney (**b**, **c**)

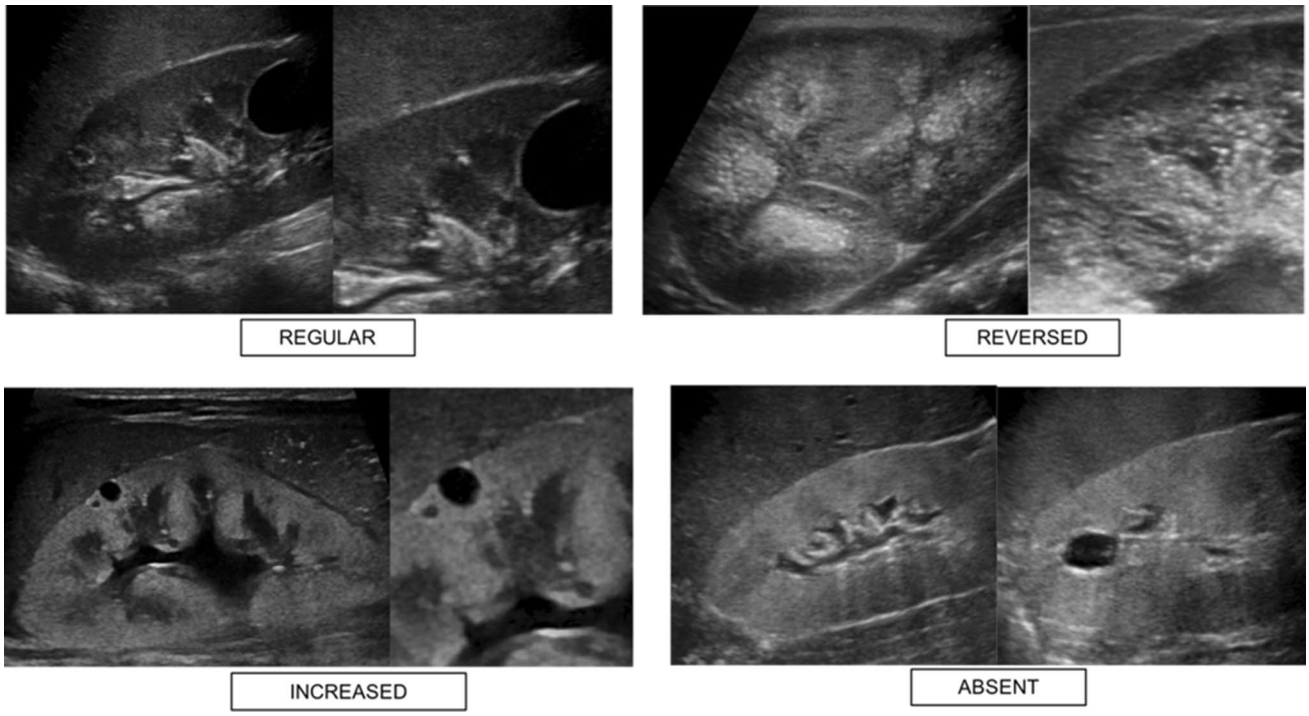

Fig. 2 Examples of various cortico-medullary differentiations in cystic renal diseases

Single cysts should be followed to make sure they remain anechoic without septa and/or nodules. They also should not increase in number, as this could indicate the presence of polycystic kidney disease, in which case family investigation is proposed.

Caliceal diverticulum

This is a extroflection of the collecting system covered with a transitional non-secreting epithelium. The connection to the caliceal system allows the passive flow of urine from the calyx to the cavity which can progressively enlarge keeping a tight neck, setting the conditions for development of calculi and infections.

We find two different types of caliceal diverticula: the first, more common, originates from a minor calyx and it is usually located at the kidney poles, especially in the upper pole, and often asymptomatic. The second type originates directly from the renal pelvis, and is usually larger and often symptomatic [9].

Caliceal diverticulum is very rare in childhood. Interestingly, the diverticulum can initially appear as a simple cyst suspicion of a diverticulum should arise when calculi are found in the 'cyst' or when scans show progressive abnormal increase in its size. Magnetic resonance (MR) without contrast and sometimes even US examination can assume a diagnosis of both forms through the evidence of a morphological connection with a calyx (Fig. 5); a definitive diagnosis is obtained by documenting, in delayed phase of contrast-enhanced computed tomography (CT) or MR, the filling of the 'cyst' with contrasted urine (iodine or gadolinium) (Fig. 6) [1, 6, 10].

Complex cysts and cystic tumors

A complex cyst is any cystic formation that does not meet the criteria of a simple cyst, presenting alterations of the wall or its contours, septa or solid nodules or internal echoes.

In adult population, the Bosniak Classification System, based on the CT criteria, identifies five distinct categories of cystic lesions according to cyst wall characteristics, septa, nodules and their contrast material enhancement; it is employed to determine the risk of malignancy and to guide the management of complex cysts. While types I and II are considered benign, III and IV type are classified as potentially malignant or malignant (Fig. 7). The IIF category includes cysts of intermediate characteristics for which image follow-up is recommended [11].

Since this classification is based on CT characteristics, for a radio-protectionist measure, a modified Bosniak classification has been introduced by some Authors for the pediatric population. The substantial difference between these two systems is that the modified version lacks a IIF category. This is because ultrasound examination has proven to be

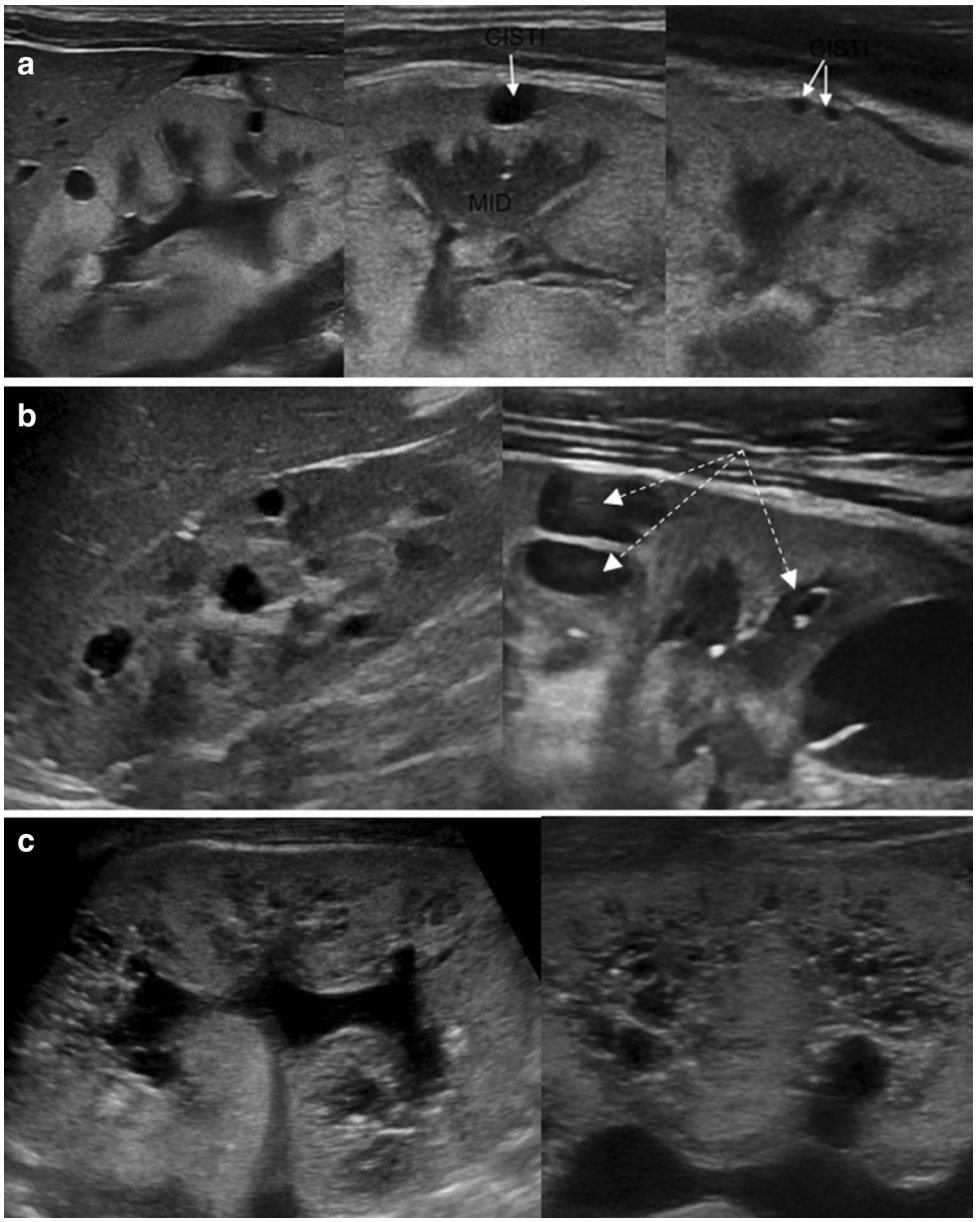

Fig. 3 Different localization of cysts: subcortical (**a**), cortico-medullary (**b**) and medullary (**c**)

more sensitive than CT in the detection of septa and small nodules; it is not uncommon for a cyst to appear clearly septate under US and not under CT/MR (Fig. 8). Authors suggest a US follow-up in children with class II cysts at 3–6-month intervals for the first year and then annually once the cysts are deemed to be stable [6, 12].

Contrast-enhanced CT or MR should be reserved for cystic lesions showing thickened wall, parietal nodules or irregular septa. If the report is positive, surgical excision is required [11, 12].

It is also worth mentioning that the use of contrast-enhanced ultrasound (CEUS) in adult population could be very useful because of its ability to show complete lack of signal (flow) into the cyst, in septa and nodules [13]. Nevertheless, the usefulness of CEUS studying a complex renal cystic lesion in childhood is not reported as a potential or useful application of CEUS by the European Federation of Societies for Ultrasound in Medicine and Biology in their guidelines and position statements [14].

Finally, malignant tumors which are usually solid in pediatric age can sometimes be partially cystic with a multilocular and pluri-septate aspect with flow signals at Doppler analysis. Cystic pediatric renal tumors vary from benign forms (cystic nephroma and cystic partially differentiated nephroblastoma, Fig. 9) to the cystic variant of Wilms' tumor and the rare multicystic variant of clear-cell renal carcinoma.

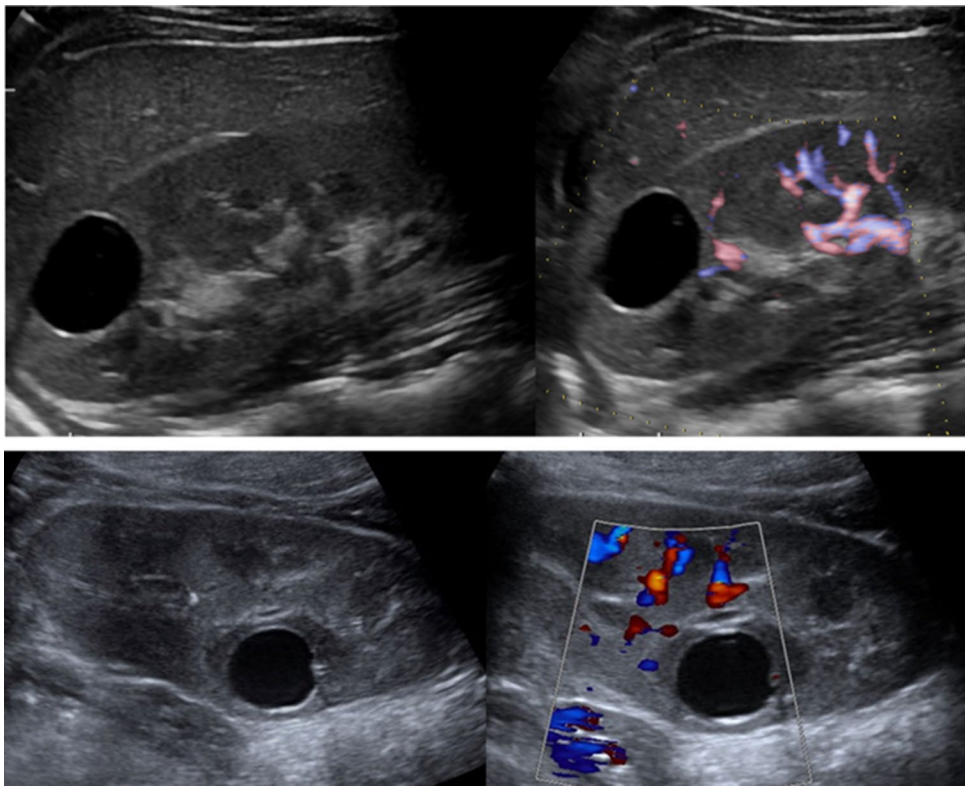

Fig. 4 Simple cysts: US and color Doppler typical aspects

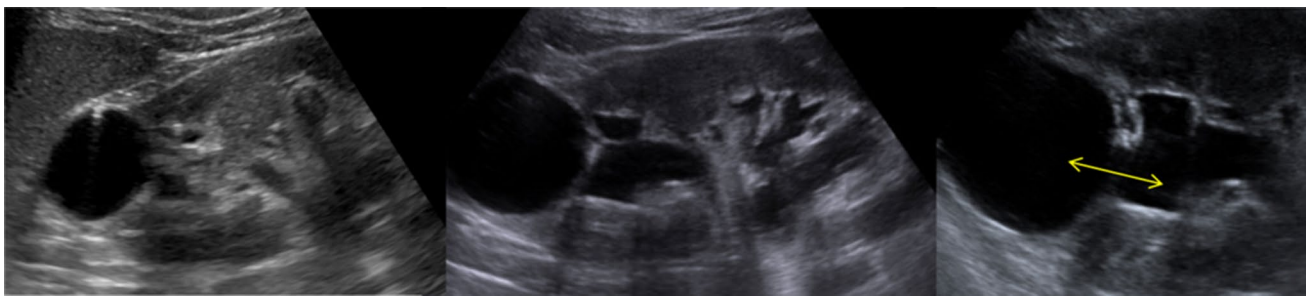

Fig. 5 Caliceal diverticulum: anechoic round lesion at the upper pole of the kidney with fortuitous evidence of a connection with a calyx (yellow double arrow)

Multicystic dysplastic kidney (MCDK)

Another non-hereditary form is the multicystic dysplasia, which is only found as unilateral (1:4300 live births) as its bilateral form is not compatible with life [2, 15, 16]. There are two predominant theories about its etiology: one proposes that ureteral atresia leads to severe obstructive hydronephrosis and consequent to multicystic dysplastic kidney, and the other suggests that an abnormal interaction between the ureteric bud and the metanephric blastema causes a failure of differentiation of this structure. The affected kidney is not functioning and multiple spread out cysts of variable size take the place of the parenchyma. Pathologists have compared its macroscopic appearance to that of a bunch of grapes (Fig. 10). A kidney affected by MCDK generally tends to involve till disappearing, especially when the size at onset was below 4 cm. In these cases, regular ultrasound checks—recommended till patients reach puberty—are meant to confirm the prenatal diagnosis, to verify the involution of the affected kidney as well as to monitor the evolution of the healthy contralateral kidney (Fig. 11). The latter should in fact develop a compensatory hypertrophy and may result in a variable percentage of cases (20–50%) being affected by vesicoureteral reflux or ureteropelvic junction obstruction [16].

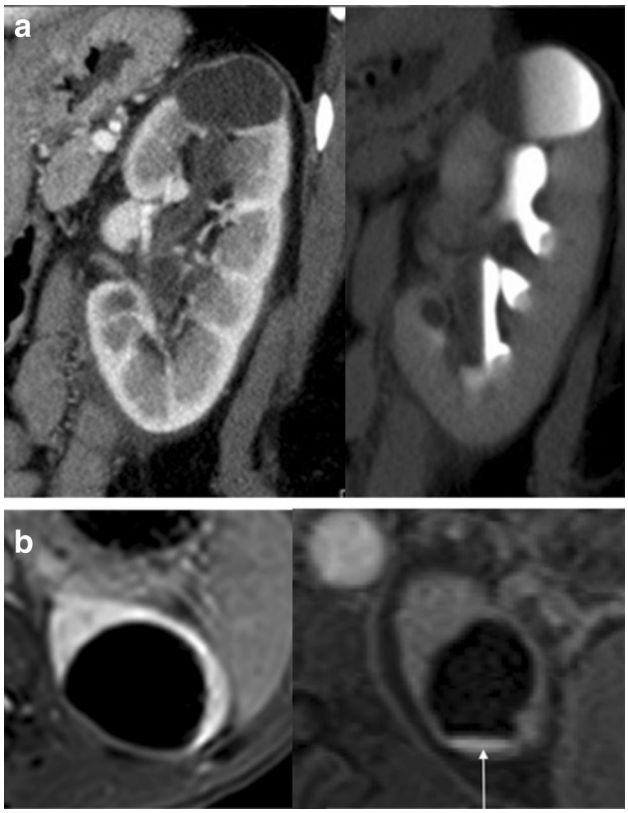

Fig. 6 Venous and delayed CT phases (**a**): presence of iodinated urine inside the 'cystic lesion' allows the diagnosis of caliceal diverticulum at the upper pole. Delayed MR phase of two different patients (**b**): a simple cyst homogenously hypodense and a caliceal diverticulum partially filled with enhancing urine (white arrow)

The MCDK shows lack of renal function through scintigraphy, when a residual function is found, a hydronephrosis, due to a ureteropelvic junction obstruction, should be suspected, since this condition can depict a similar radionephrogram [3]. In these cases, the distinction between the two forms is sonographically possible by demonstrating the presence of a portion of the dysplastic residual parenchyma in a central position, between the cysts, in the multicystic dysplastic form, while in the severe obstructive form, the residual parenchyma is always placed on the periphery of the kidney. A further possibility of distinguishing between the two forms is the peripheral or central position of the larger cystic formation: in the multicystic dysplastic kidney, larger cysts are generally placed on the periphery, while in the obstructive form the larger cysts are located in the central position and correspond to the dilated pelvis [1].

Multicystic dysplasia generally involves the whole kidney, though it can sometimes affect only a part of it (e.g., a duplicated collecting system with upper ureter atresia, Fig. 12) [17, 18].

Obstructive cystic dysplasia

Some cortical small cysts may occur in children affected by congenital anomalies of urinary tract (particularly severe obstructive hydronephrosis, Figs. 13 and 14). Obstructive cystic dysplasia is the most common cause of irregular, thinned, hyperechoic cortex associated with small peripheral cysts [19].

Medullary sponge kidney

Worth mentioning is the extremely rare medullary sponge kidney, very occasionally detected during an intravenous urography, therefore, even rarer in pediatric age.

The diagnosis, historically with urography, is characterized by medullary radial striping due to contrast stagnation in the linear dilatation of the pre-papillar distal tubes, giving it an appearance to bouquet of flowers [20].

With US, the disease shows substantial diffuse hyperechogenicity of the medulla to the point of showing medullary nephrocalcinosis (inversion of the normal corticomedullary differentiation).

Ciliopathies

This is a group of clinically and genetically overlapping disorders, whose etiologies lie in the defects in the structure and function of cilia. These antenna-like organelles are present on the surface of nearly every cell in the body justifying the multiorgan manifestations of these diseases.

Autosomal recessive polycystic kidney disease (ARPKD)

This is a hereditary disease characterized by renal and hepatic involvement with dilatation of collecting renal ducts, periportal fibrosis and bile ducts ectasia. It is transmitted through an autosomal recessive mechanism and tends to manifest in children and teens. ARPKD is much rarer than ADPKD (1:20,000 live births) and is usually diagnosed at birth as confirmation of prenatal sonographic suspicion. In severe cases, extensive involvement of the kidneys leads to oligohydramnios and pulmonary hypoplasia during the fetal period: these patients, may die rapidly after birth due to severe respiratory distress (Fig. 15).

Kidneys appear enlarged, regular shaped, and the cystic dilatations originate from the medulla and expand to the cortical region. Renal cysts are kept within the collecting

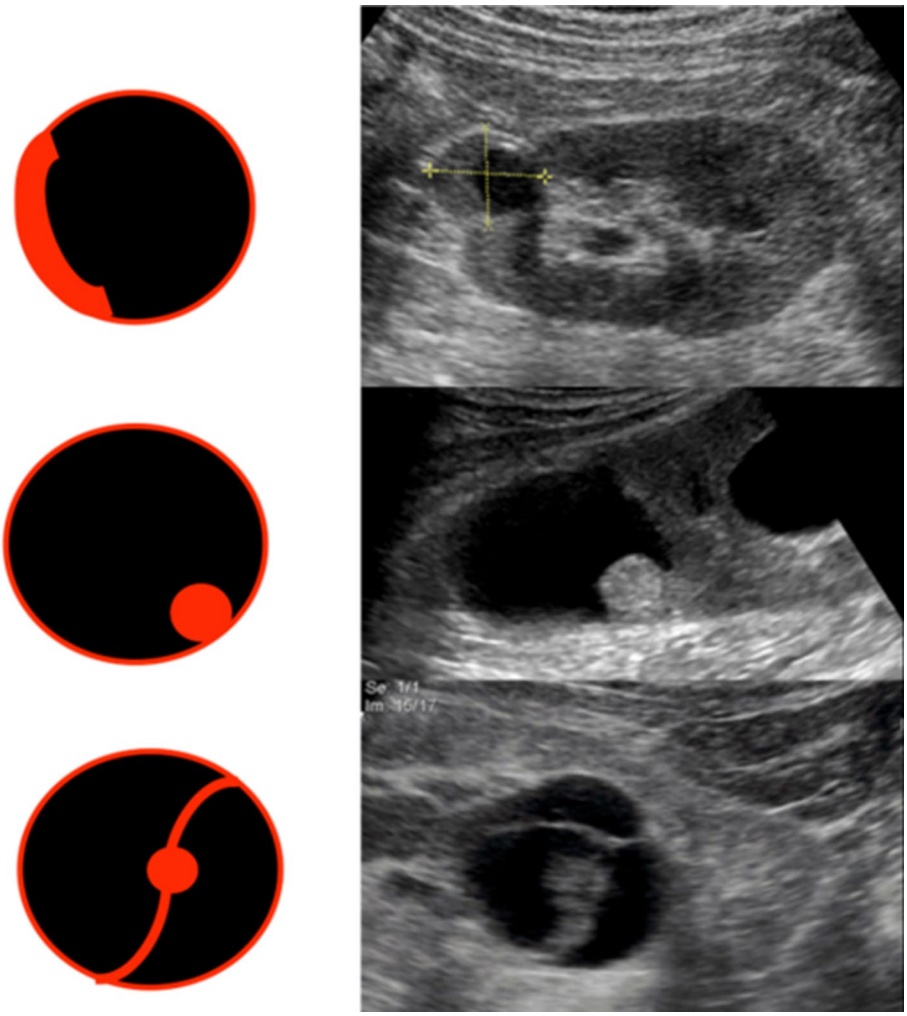

Fig. 7 Bosniak III–IV cystic lesions: wall thickening, parietal nodule and a septum with nodule

ducts and are usually too small to be detected under low-frequency evaluation [4]. The cystic dilatation of the ducts is fan shaped and can be visualized with high-frequency probes.

Sonographically, kidneys are enlarged (more than 4 standard deviations above the average), with thickened renal parenchyma and altered cortico-medullary differentiation (usually poor or reversed) [4, 15].

High-frequency probes allow the identification of micro-cysts often associated with hyperechogenic dots, without posterior shadow cone, sometimes with "comet-tail" reverb and with countless linear cystic microdilatations (Fig. 16) [21]. The disease is characterized by a saccular dilatation of the collecting ducts between 1 and 2 mm, with a radial distribution from the medullary to the cortical which leaves the subcapsular space unaffected as no collecting tubes are present on this layer. This translates into a hypoechoic band fading toward the margins, usually found in children surviving a less severe form [22, 23].

Extrarenal manifestations characterized by congenital hepatic fibrosis, bile duct ectasia, and biliary segmental dilatations (Caroli-like, Fig. 17) are common: these anomalies are generally associated with normal hepatic function. US should, therefore, evaluate the entire abdomen carefully to the liver, to exclude/confirm a diffuse/segmental dilatation of the bile ducts [15, 19, 22].

Autosomal dominant polycystic kidney disease (ADPKD)

This hereditary autosomal dominant disease affects the kidneys as well as have other systemic manifestations. Its incidence varies between 1:400 and 1:1000 live births, and it is among the most common hereditary pathology [15]. It generally becomes symptomatic in adults, though there is some report of cases in children and adolescents.

It is characterized by multiple small cysts of variable size, unevenly distributed along the whole renal parenchyma on both cortical and medullary levels. Such cysts overlap parenchyma with normal sonographic appearance

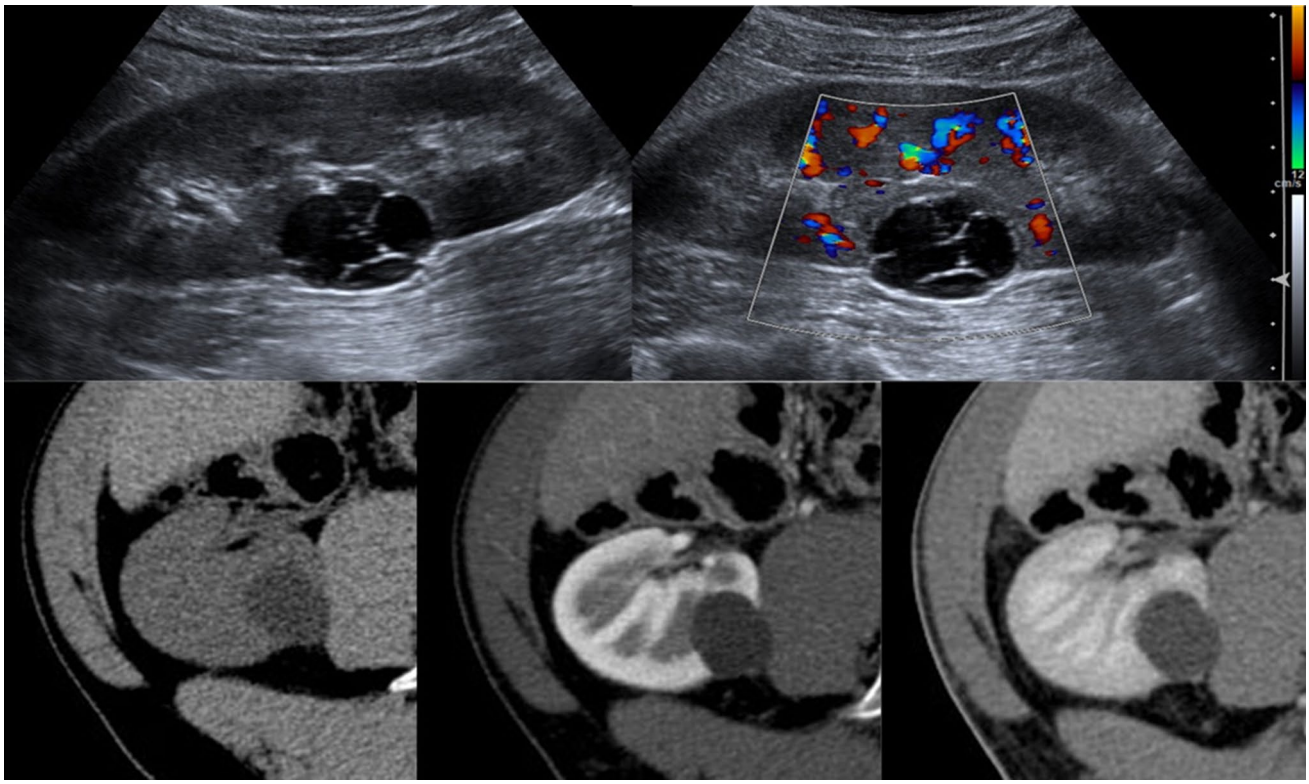

Fig. 8 A septate renal cyst at US evaluation and its simple appearance at multiphasic CT

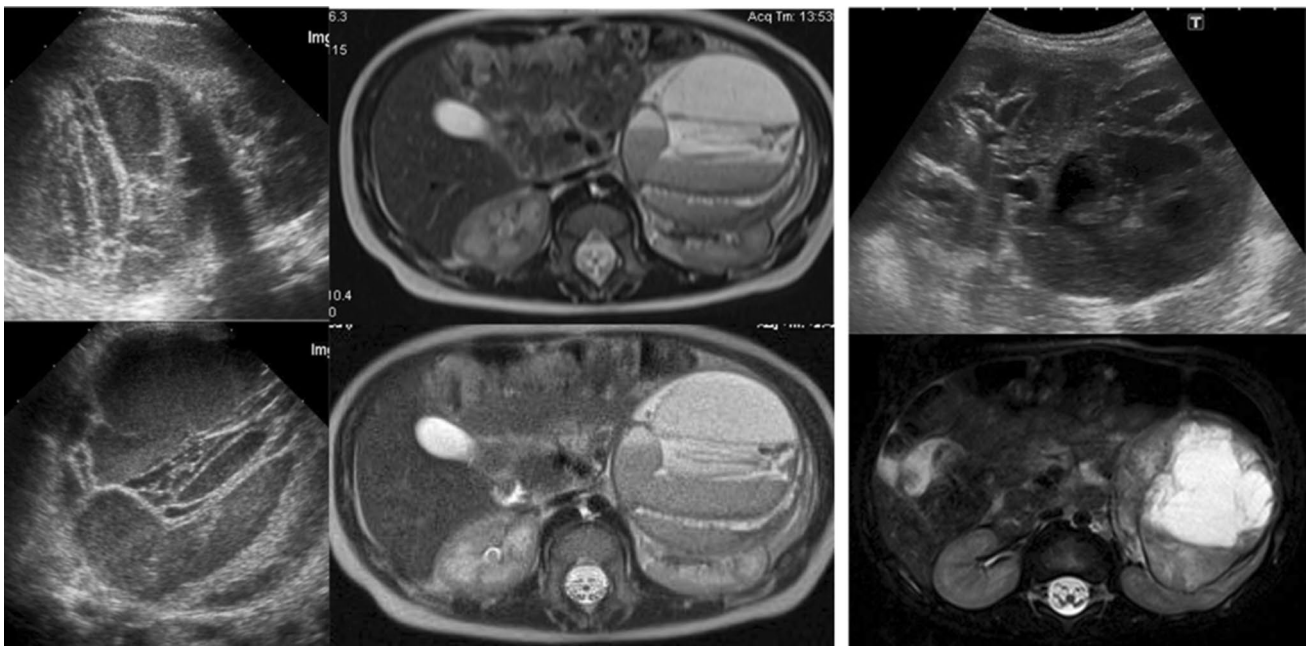

Fig. 9 Two examples of cystic pediatric renal tumors: US and MR appearance

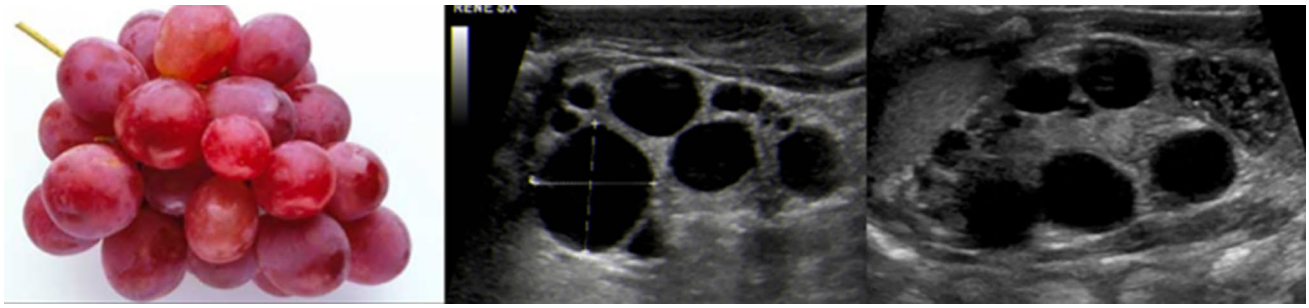

Fig. 10 MCDK: multiple cysts of variable size and dysplastic residual parenchyma in a central position

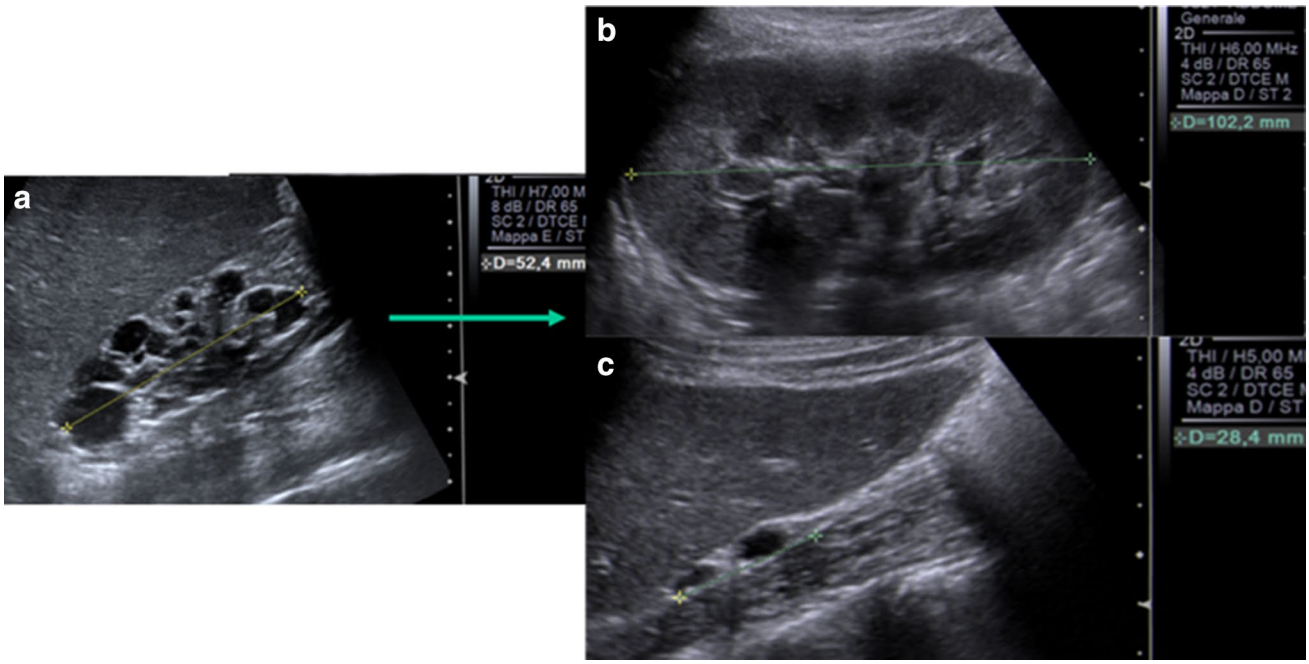

Fig. 11 MCDK: US scan at diagnosis (**a**), check scans demonstrating compensatory hypertrophy of contralateral kidney (**b**) and involution of the affected kidney (**c**)

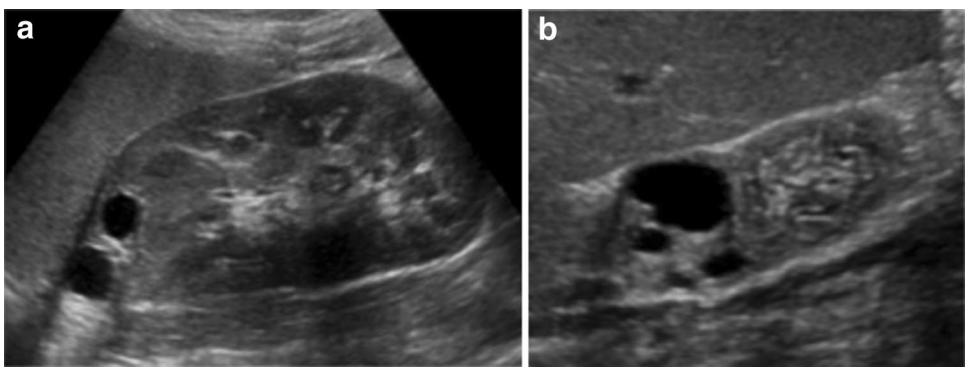

Fig. 12 Two cases of segmental MCDK associated with duplicated collecting system: isolated multicystic dysplasia in upper pole (**a**) and multicystic dysplasia in upper pole associated with dysplastic kidney (**b**)

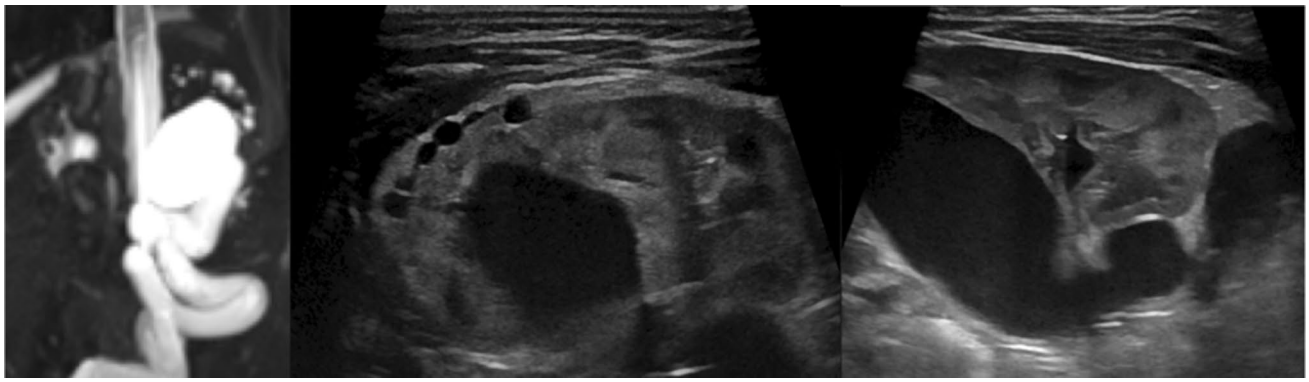

Fig. 13 Acquired renal cysts. Patient with left-sided duplicated collecting system and ureteral ectopic insertion in vagina: coronal T2w image demonstrates severe upper ureteral dilatation associated with multiple tiny cysts with peripheral distribution at upper moiety. US depicts hydronephrosis and thinned dysplastic renal parenchyma with small peripheral cysts

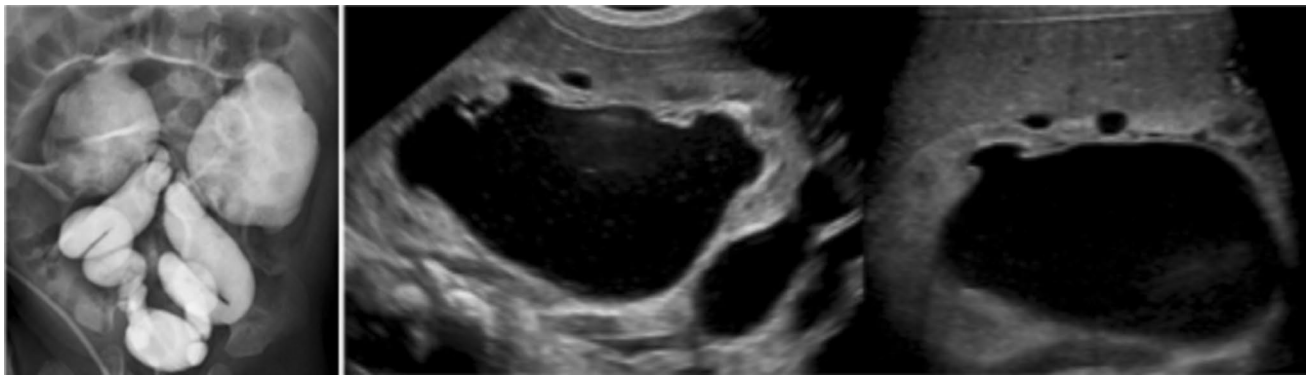

Fig. 14 Acquired renal cysts. Retrograde cystography demonstrates bilateral refluent megaureter. US depicts severe pyelic dilatation and thinned dysplastic hyperechoic renal parenchyma with small peripheral cysts

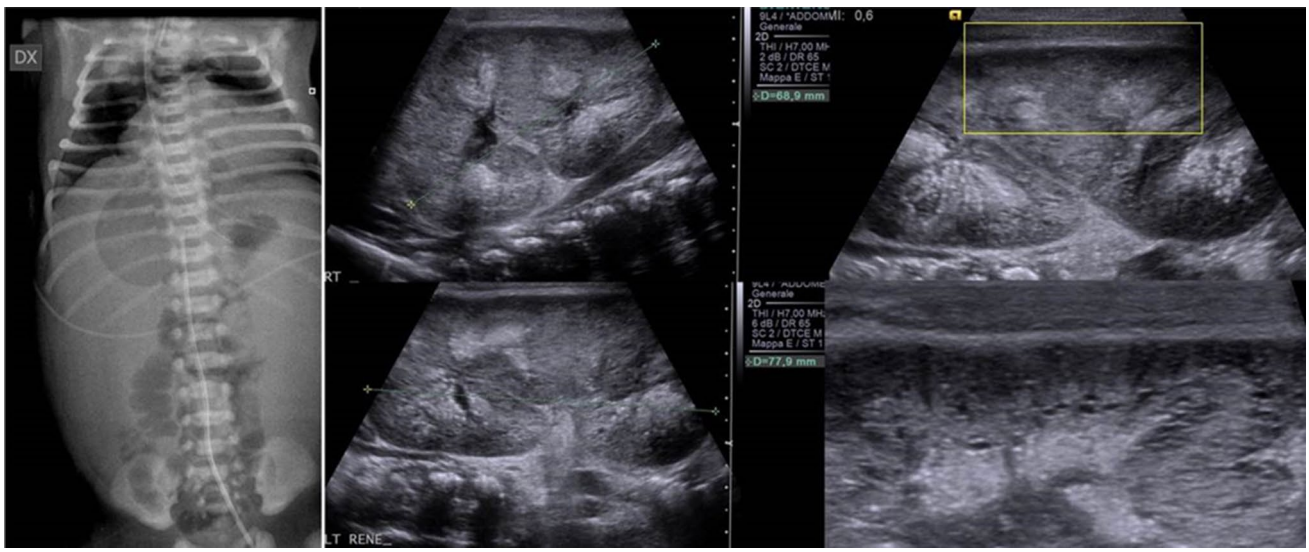

Fig. 15 ARPKD: anteroposterior *x*-ray depicts pulmonary hypo/dysplasia, centralization of bowel associated with bulging flanks due to enlarged kidneys. US confirms nephromegaly, and high-resolution US allows the identification of linear cystic cortico-medullary microdilatations

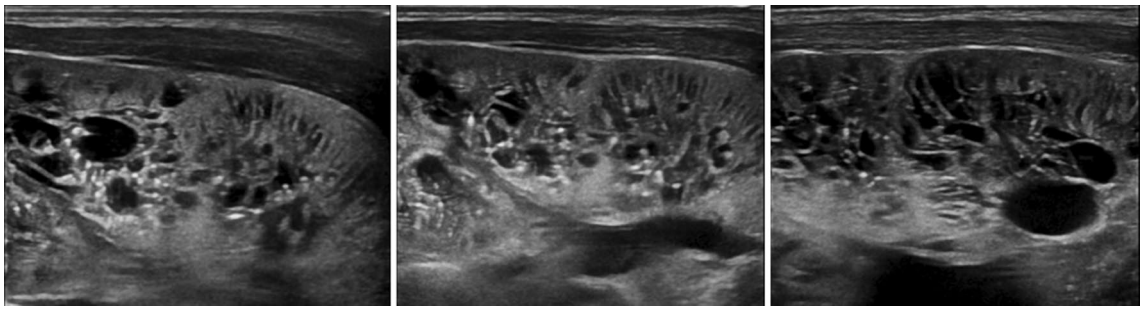

Fig. 16 High-frequency US in ARPKD: multiple hyperechogenic dots with "comet-tail" reverb and some macroscopic cysts between countless tiny microcysts in a radial distribution representing dilated tubules

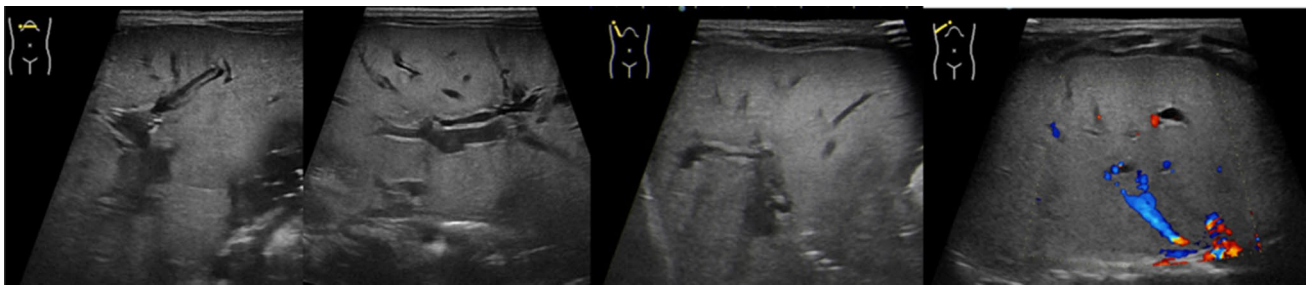

Fig. 17 US image of the liver in a child affected by ARPKD reveals hepatopathy and Caroli disease with extensive cystic biliary dilatation

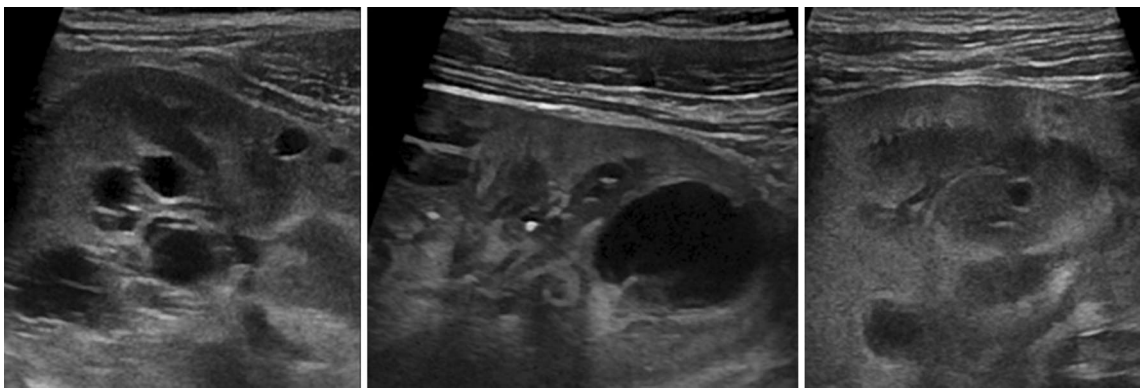

Fig. 18 ADPKD: small cortical and medullary cysts overlapping parenchyma with normal US appearance

(Fig. 18). The involvement can appear asymmetric although bilateral; typically some cysts develop under the subcapsular cortex.

Since the incidence of simple cyst is quite low in children, the presence of multiple/different cysts in both kidneys is highly suspicious of ADPKD, even in the absence of a positive family history.

Warning: the presence of two cysts in children with a positive family history of the disease is diagnostic for ADPKD [4, 5, 19]. For this reason, a small renal cyst in a newborn must be sonographically monitored to identify any additional or contralateral cysts. In those patients with positive family history, the diagnosis is based on the ultrasound evaluation as well as on genetical analysis.

In most cases, the natural history of ADPKD goes through a long period of stability followed by a progressive decline in renal function; however, the evolution of the disease can vary among members of the same family.

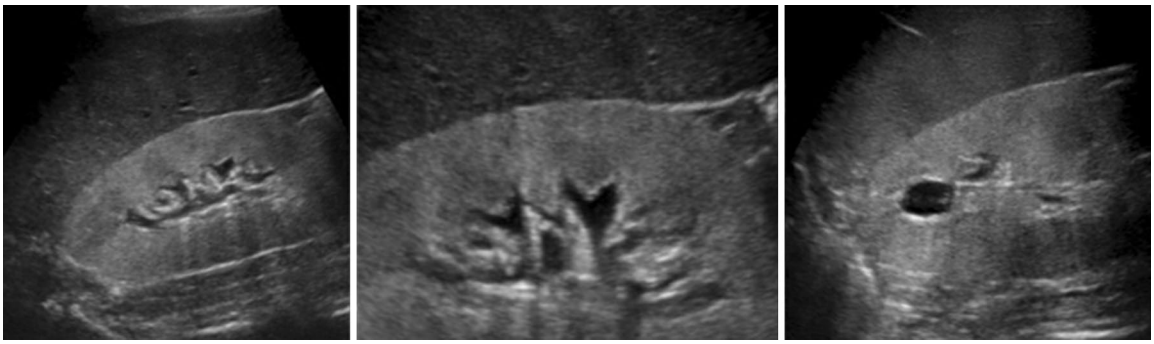

Fig. 19 NPHP: US depicts diffusely increased echogenicity of the renal parenchyma with loss/poor cortico-medullary differentiation, cysts, if present, affect the medulla as well as the cortico-medullary junction

Nephronophtisis (NPHP)

NPHP is a rare autosomal recessive disease responsible for renal insufficiency within the second decade. In its early stages, the disease may appear aspecific under US examination, leaving the normal appearance of the kidney unaffected. Successively, kidneys may appear smaller than usual, with a hyperechoic cortex and a poor cortico-medullary differentiation (Fig. 19). Renal cysts are initially not present, while in later stages they will appear segmentally distributed and affect the medulla as well as the cortico-medullary junction. However, quite frequent atypical forms will show no cysts [4, 5, 24].

Glomerulocystic kidney disease (GCKD)

GCKD is a rare ciliopathy with autosomal dominant transmission characterized by cystic dilatation of the first portion of the convoluted tubule. Consequentially, these cysts are localized in the subcapsular cortex and the medulla appears normal, as collecting ducts and loops of Henle are unaffected. US evaluation demonstrates the presence of multiple subcapsular tiny cysts that correspond to distention of the Bowman spaces kidneys are enlarged, and the cortex is diffusely hyperechoic with variable appearing medulla.

In some cases, neonatal forms of ARPKD, ADPKD, and GCKD may have a similar, virtually indiscernible, US appearance showing hyperechoic-enlarged kidneys.

Syndromic diseases

Renal cysts can also represent a clinical manifestation of some syndromic diseases such as TS and VHL, belonging to a heterogenic group of hereditary syndromes characterized by multisystemic neurocutaneous disorders.

Tuberous sclerosis

Tuberous sclerosis is a dominant autosomal disease (1:7000 live births) characterized by multiple hamartoma. In the kidney, the pathology can manifest through angiomyolipoma, cysts and carcinoma.

Renal cysts are found in 47% of the cases, generating from any portion of the nephron, they are bilateral and distributed through both medulla and cortex, and their size

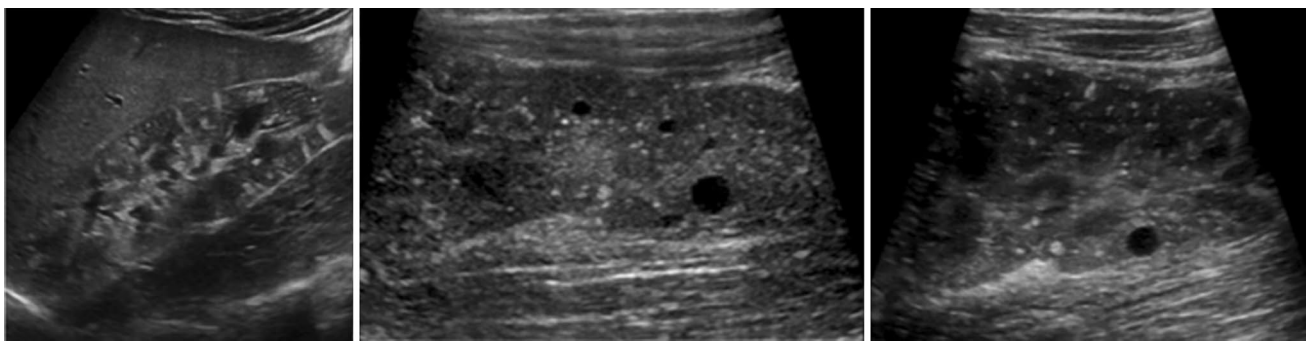

Fig. 20 Tuberous sclerosis: US shows combination of multiple hyperechoic angiomyolipomas and isolated small cysts

varies from few millimeters to some centimeter (Fig. 20) [25].

The coexistence of multiple angiomyolipoma and bilateral renal cysts is virtually pathognomonic of tuberous sclerosis.

Von Hippel–Lindau syndrome

Von Hippel–Lindau syndrome is a dominant autosomal phakomatoses (1:36,000 live births) characterized by the development of a variety of benign and malignant tumors in different organs such as retinal and central nervous system hemangioblastoma, pancreatic cysts, pheochromocytoma, and ependymal cystadenoma.

Renal cysts are a common manifestation of the disease (76% of patients), they are generally cortical, multiple, bilateral, and small (0.5–3 cm). They can sometimes mimic a ADPKD, but are not responsible for acute renal failure.

Compliance with ethical standards

Conflict of interest Author Federica Ferro declares that she has no conflict of interest. Author Norberto Vezzali declares that he has no conflict of interest. Author Evi Comploj declares that she has no conflict of interest. Author Elena Pedron declares that she has no conflict of interest. Author Marco Di Serafino declares that he has no conflict of interest. Author Francesco Esposito declares that he has no conflict of interest. Author Piernicola Pelliccia declares that he has no conflict of interest. Author Eugenio Rossi declares that he has no conflict of interest. Author Massimo Zeccolini declares that he has no conflict of interest. Author Gianfranco Vallone declares that he has no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent For this type of study formal consent is not required.

References

1. Riccabona M, Ring E, Avni F (2018) Imaging in renal agenesis, dysplasia, hypoplasia, and cystic diseases of the kidney. In: Riccabona M (ed) Pediatric urogenital radiology. Springer, Berlin, pp 553–577
2. Gupta I, Bitzan M (2104) Cystic renal disease. In: Manual of pediatric nephrology. Springer, Berlin, pp 249–262
3. Paliwalla M, Park K (2014) A practical guide to urinary tract ultrasound in a child: pearls and pitfalls. Ultrasound 22:213–222
4. Chung E, Conran R, Schroder JW, Rohena-Quinquilla I, Rooks V (2014) Pediatric polycystic kidney disease and other ciliopathies: radiologic-pathologic correlation. Radiographics 34:155–178
5. MacRae K, Chisthi A (2014) Cystic renal disease. In: Chisti AS et al (eds) Kidney and urinary tract disease in the newborn. Springer, Berlin, pp 263–274
6. Karmazyn B, Tawadros A, Delaney L, Marine M, Cain M, Rink R (2015) Ultrasound classification of solitary renal cysts in children. J Pediatr Urol 11:149 e1–149 e6
7. Koutlidis N, Joyeux L, Mejean N, Sapin E (2015) Management of simple renal cyst in children: French multicenter experience of 36 cases and review of the literature. J Pediatr Urol 11:113–137
8. Bayram MT, Alaygut D, Soylu A, Serdaroglu E, Cakmakci H, Kavukcu S (2014) Clinical and radiological course of simple renal cysts in children. Urology 83(2):433–437
9. Wulfsohn MA (1980) Pyelocaliceal diverticula. J Urol 123:1–8
10. Karmazyn B, Kaefer M, Jennings G, Nirmala R, Raske ME (2010) Caliceal diverticulum in pediatric patients: the spectrum of imaging findings. Pediatr Radiol 41:1369–1373
11. Bosniak AM (2012) The Bosniak renal cyst classification: 25 years later. Radiology 262(3):781–785
12. Wallis C, Lorenzo Afarhat W, Bagli D, Khoury A, Pippi Salle J (2008) Risk assessment of incidentally detected complex renal cysts in children: potential role for a modification of the Bosniak classification. J Urol 189:317–321
13. Sidhu P, Cantisani V, Dietrich C et al (2017) The EFSUMB Guidelines and recommendations for the clinical practice of contrast-enhanced ultrasound (CEUS) in non-hepatic applications: update 2017. Ultraschall Med 2018(39):e2–e44
14. Riccabona M, Lobo ML, Augdal TA et al (2018) European Society of Paediatric Radiology Abdominal Imaging Task Force recommendations in paediatric uroradiology, part X: how to perform paediatric gastrointestinal ultrasonography, use gadolinium as a contrast agent in children, follow up paediatric testicular microlithiasis, and an update on paediatric contrast-enhanced ultrasound. Pediatr Radiol 48:1528–1536
15. Dillman J, Trout A, Smith E, Towbin A (2017) Hereditary renal cystic disorders: imaging of the kidney and beyond. Radiographics 37:924–946
16. Dermentzoglou V, Grigoraki V, Zarifi M (2018) Contemporary concepts and imaging findings in paediatric cystic Kidney disease. Hell J Radiol 3(2):65–79
17. Lin C, Tsai J, Sheu J, Lu H, Chang B (2010) Segmental multicystic dysplastic kidney in children: clinical presentation, imaging finding, management, and outcome. J Pediatr Surg 45:1856–1862
18. Jeon A, Cramer B, Walsch E, Pushpanathan C (1999) A spectrum of segmental multicystic renal dysplasia. Pediatr Radiol 29:309–315
19. Verghese P, Miyashita Y (2014) Neonatal polycistic kidney disease. Clin Perinat 41:543–560
20. Patriquin H (1985) Medullary sponge kidney in childhood. AJR 145:315–319
21. Lucaya J, Enriquez G, Nieto J (1993) Renal calcification in patient with ARPKD: prevalence and cause. AJR 160:359–362
22. Turkbey B, Ocak I, Daryanani K, Font-Montgomery E, Lukose L, Byrant J (2009) Autosomal recessive polycistic kidney disease and congenital hepatic fibrosis (ARPKD/CHF). Pediatr Radiol 39:100–111
23. Traubici J, Daneman A (2005) High-resolution renal sonography in children with autosomal recessive polycystic kidney disease. AJR 184:1630–1633
24. Vester U, Kranz B, Hoyer PF (2010) The diagnostic value of ultrasound in cystic kidney diseases. Pediatr Nephrol 25:231–240
25. Casper K, Donnelly L, Chen B, Bissler J (2002) Tuberous sclerosis complex: renal imaging findings. Radiology 225:452–456