Sonographic Appearance of Pediatric Kidney Parenchyma: 
A spectrum of Normal Variants and Parenchymal 
Pathologies

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number as shown above.
Learning objectives

• To acquaint oneself with the accurate renal sonographic examination technique.
• To identify the appearance of normal renal parenchyma in various age groups of pediatric patients.
• To familiarize oneself with the normal variants that may mimic pathological processes.
• To review the most common renal parenchymal pathologies in the pediatric population.

Background and purpose

Ultrasonography is the primary imaging modality utilized in the diagnostic assessment of kidneys. It is a noninvasive, reliable, affordable and radiation-free modality that offers detailed assessment of the renal parenchyma and its vasculature.

The normal renal parenchymal sonographic appearances in different pediatric age groups are illustrated along with the normal variations that mimic pathology.

We illustrate the numerous common and rare parenchymal pathologic changes associated with disease process.

Content, imaging findings or procedure details

Findings and procedure details:

Technique:

The examination starts with the use of a convex array transducer (9-2 MHz) which provides a general assessment of the kidneys and its surrounding structures. A higher frequency-linear array transducer (up to 17 MHz) is then used which provides greater
resolution required to delineate the parenchymal details and assess the cortico-medullary differentiation.

Magnification of selected parts of each kidney is essential to depict smaller structures in the parenchyma, by limiting the field of view to the half of the kidney closest to the transducer and focused evaluation of only one or two pyramids and the surrounding cortex is done. An examination without these focused and magnified images of the kidney is considered incomplete (Fig. 1 on page 10 and Fig. 2 on page 10).

**Normal Kidney ultrasonic appearance:**

In the longitudinal plane, the newborn's kidney has a characteristic oval bean-shape, with lobulated margins (called fetal lobulations). The parenchyma is composed of the:

1. Cortex, which becomes less echoic as the patient grows.
2. Medullary pyramids, which appear hypoechoic and are heart-shaped, with their tip pointing towards the renal sinus and an arcuate artery runs at their base. Normally, this cortico-medullary differentiation should be observed at least till the age of 4 years. (Fig. 3 on page 11).

The length of the kidneys is mainly used to assess their size and growth. This is achieved by comparing their lengths to standardized charts and with each other. The difference between the kidney length should be <1.5 cm.

**Normal Variants:**

1. **Renal malrotation:**

   The renal hilum is normally directed medially. The renal hilum is initially oriented anteriorly, but during its ascent from the pelvis the kidney rotates 90° along its longitudinal axis to its more typical orientation. When the renal hilum is more anteriorly oriented, the kidney is malrotated (Fig. 4 on page 11).

2. **Junctional parenchymal defect or line:**
Represents incomplete embryologic fusion of two primary renal lobes, more commonly seen on the right kidney as a triangular echogenic cortical defect or as a hyperechoic line. The defect is an extension of sinus fat into the cortex, usually at the border of the upper pole and interpolar region of the kidney. Synonyms: Interrenicular septum and Oddono sulcus (Fig. 5 on page 13).

3. **Fetal Lobulation:**

Develop from fusion of embryonic parenchymal masses termed "renunculi" resulting in lobulated margins, indentations lie between renal pyramids or calyces, no cortical loss.

*Note:* Fetal lobulation, renal malrotation and renal parenchymal junction defect or lines maybe mistaken for parenchymal scarring.

Parenchymal scarring can be differentiated by focal thinning of renal cortex above the renal pyramid (Fig. 6 on page 13).

4. **Column of Bertin:**

The adjacent cortices of the contiguous renunculi fuse to form a thick layer of cortex, termed a "column of Bertin" The fused parenchyma usually resorbs during in utero development. If resorption is incomplete, the column of Bertin can hypertrophy and cause splaying of the calyces, mimicking a mass (Fig. 7 on page 14).

At sonography, it appears as a round or oval mass with an echogenicity equal to or slightly greater than that of normal adjacent cortex. It extends from the renal cortex to the renal sinus and is located between two medullary pyramids, and it is frequently bordered by a junctional parenchymal defect and/or line. The preserved arcuate arteries with absence of renal contour abnormality and vascular distortion are useful features in differentiating a hypertrophied column of Bertin from a true mass.

5. **Dromedary hump:**

It is a bulge in the lateral margin of the mid-pole of the kidney, most commonly the left one. The echotexture of the hump is similar to that of the surrounding normal parenchyma (Fig. 7 on page 14).

*Congenital Anomalies:*
Congenital anomalies can be classified as abnormalities in number (renal agenesis), position (ptosis or ectopia), and fusion (horseshoe kidney, crossed fused ectopia, and renal duplication).

**Renal Agenesis:**

Defined as complete absence of renal tissue. It results when there is absence of the metanephric blastema, absence of ureteral bud development, or failure of the ureteric bud to induce metanephric differentiation.

**Renal hypoplasia:**

Refers to a congenitally small kidney with normal parenchyma. (Fig. 8 on page 15).

**Renal Ectopia:**

Absence of kidney in its expected renal fossa, can range in location from pelvic to thoracic.

Renal ptosis: the kidney is located lower than its normal location overlying the 12th rib.

Simple renal ectopia: kidney located ipsilateral to its ureteral insertion (Fig. 9 on page 16).

Crossed fused ectopia: kidney is malrotated with fusion of the upper pole of the ectopic kidney to the lower pole of normally positioned kidney, the ectopic kidney is located contralateral to its ureteral insertion (Fig. 10 on page 17).

**Horseshoe kidney:**

Kidneys on opposite sides are fused at their lower poles in midline, anterior to spine and aorta but posterior to inferior mesenteric artery (Fig. 11 on page 18).

**Renal Parenchymal Pathologies:**
Consists of three main categories, including nephrocalcinosis, parenchymal hyperechogenicity and cystic renal diseases.

**Nephrocalcinosis:**

Represents calcification within the renal parenchyma, either cortical or medullary in location, seen as hyperechogenicity without acoustic shadowing. Medullary nephrocalcinosis is the entity most commonly encountered in pediatric age group.

**Medullary nephrocalcinosis:**

Variations in distribution may range from hyperechoic foci sparsely distributed, to outlining the periphery of the pyramid to diffusely involving the entire pyramid (Fig. 12 on page 19 and Fig. 13 on page 20).

**Cortical nephrocalcinosis:**

Can exhibit 3 sonographic patterns:

- Thin peripheral rim of calcification.
- Parallel lines of calcification known as tram track appearance.
- Punctate calcification distributed randomly in the cortex.

(Fig. 14 on page 21).

**Hyperechoic renal parenchyma:**

It is defined by a hyperechoic cortex in comparison with the adjacent liver or spleen with possible loss of cortico-medullary differentiation. Hyperechogenicity is a nonspecific finding and is probably complex; and may involve glomerular, tubular, interstitial, or vascular abnormalities. (Fig. 15 on page 21, Fig. 16 on page 22, Fig. 17 on page 23, Fig. 18 on page 24)

**Cystic Renal diseases:**
Simple cysts are rare in children, with an incidence of less than 1%, therefore any cyst noticed in the pediatric renal parenchyma is a significant finding, unlike cysts in adult kidneys.

Cystic diseases of the kidney can be unilateral or bilateral, symptomatic at birth or detected later in life and can be inherited or sporadic.

The Potter classification of renal cystic diseases (Fig. 19 on page 24) has been replaced by a classification based on the genetic or non-genetic origin of the renal cystic diseases (Fig. 20 on page 25).

Genetically transmitted diseases exhibit a defect in the structure or function of primary cilia. Primary cilia are microtubular antenna-like cellular organelles that extend outward from the surface of many cells of the renal tubular epithelium. Cilia regulate cell proliferation and differentiation in developing and mature kidneys.

**Multicystic Dysplastic Kidney Disease:**

It is a type of non-heritable developmental anomaly due to in Utero atresia of the proximal ureter and renal pelvis, resulting in multiple rounded or oval shaped cysts of various sizes, without connections between them, with no or little remnant renal parenchyma. Most cases are unilateral, but can be bilateral too.

It regresses in size over time spontaneously, but it may also progress in size.

May affect the entire kidney or a segment of the kidney, most commonly in crossed fused ectopia or in the upper pole moiety of a duplicated collecting system. (Fig. 21 on page 26).

**Autosomal recessive polycystic kidney disease:**

(ARPKD) is the most common inherited renal cystic disease that manifests in childhood. It is characterized by enlarged echogenic kidneys, poor cortico-medullary differentiation with cylindrical and saccular dilatation of the collecting tubules.
Increased parenchymal echogenicity is produced by the innumerable fluid-tubular wall interfaces. Bilateral whole kidneys are often affected, although focal and unilateral renal involvement has also been described. (Fig. 22 on page 27 and Fig. 23 on page 28).

**Autosomal dominant polycystic kidney disease:**

In the neonate, sonography demonstrates enlarged, diffusely echogenic kidneys with multiple small cysts. The sonographic findings are similar to those of autosomal recessive polycystic disease, and the diagnosis is based on family history or renal biopsy.

In older children and adolescents, the more classic changes develop. The kidneys may be normal size or enlarged and contain multiple bilateral cortical cysts, causing calyceal distortion. Hemorrhage into the renal cysts can result in low-level internal echoes, fluid-blood levels, septations, and thick walls. Calcification of the cyst walls may be seen later in life. Cysts can also be seen in other organs, particularly the liver.

Sonography is used to screen children in affected families to determine whether they have the disease. Criteria for diagnosing the disease include the presence of at least two cysts in one kidney or one cyst in each kidney.

(Fig. 24 on page 29 and Fig. 25 on page 30).

**Renal parenchymal abnormalities in syndromes:**

**Zellweger Syndrome:**

Also, known as the cerebrohepatorenal syndrome, is a multisystem metabolic abnormality. It primarily affects the central nervous system (CNS), liver and kidneys. It results from an abnormality in peroxisomal metabolism. They tend to have bilateral hyperechoic renal parenchyma with subcapsular cysts (Fig. 26 on page 31).

**Joubert’s Syndrome:**

Also, known as vermian aplasia or molar tooth midbrain-hindbrain malformation. It is an autosomal recessive disorder where there is a variable degree of cerebellar vermal agenesis. They are known to have renal cysts and cystic dysplastic kidneys (Fig. 27 on page 32).
**Tuberous sclerosis:**

Also, known as tuberous sclerosis complex or Bourneville disease, is a neurocutaneous disorder (phakomatosis) characterised by the development of multiple benign tumors of the embryonic ectoderm (e.g. skin, eyes, and nervous system). It was classically described as presenting in childhood with a triad (Vogt triad) of: seizures, mental retardation, adenoma sebaceum. The full triad is only seen in a minority of patients (~30%).

Their kidneys may show multiple small cysts at birth and numerous angiomyolipoma, that increase in number and size with increasing age. (Fig. 28 on page 33 and Fig. 29 on page 34).

**Denys-Drash Syndrome:**

A rare congenital syndrome caused by WT1 gene mutation. It is characterized by the presence of congenital nephropathy (nephritic syndrome), 90% risk of developing Wilms tumor, and intersex disorders (Pseudohermaphroditism). Also, Meacham syndrome and Frasier syndrome, allelic disorders with similar clinical features.

**DiGeorge Syndrome:**

Also, called velocardiofacial syndrome, is a syndrome where a small portion of the chromosome 22 is lost.

CATCH 22: cardiac defects, abnormal facial features, thymic underdevelopment, cleft palate and hypocalcemia. Therefore, they are treated with calcium and tend to have medullary nephrocalcinosis. (Fig. 30 on page 35).

**Turner's syndrome:**

Caused by single X chromosome or by absence of a set of genes from the short arm of one X chromosome. It affects girls only and they present with short stature, delayed puberty, coarctation of the aorta and horseshoe kidney. (Fig. 31 on page 36).
**Fig. 1:** Longitudinal US images of the right kidney of the same patient obtained with (A) convex transducer; good for general assessment of the renal contour, size and echogenicity compared to the liver. (B) Linear transducer; better delineates the corticomedullary differentiation and parenchymal details. Here the cortex is hyperechoic to the liver, which is pathological for this age. (C) A magnified, focused image obtained with high resolution transducer. By limiting the field of view to the part of the kidney closest to the transducer, punctate foci of medullary nephrocalcinosis are demonstrated.

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**Fig. 2:** (A) Longitudinal kidney ultrasound scan in a 2-month-old child. The pyramids appear anechoic, as the machine settings weren't well adjusted. This can be easily misinterpreted as dilated calyces. (B) Companion case of hydronephrosis to compare.
Fig. 3: Demonstrates the normal ultrasonographic appearance of kidney in various Pediatric age groups. (A) Renal cortex is hyperechoic compared to the liver in a 2 days old baby, renal pyramids are hypoechoic with hyperechogenicity at the tip of the pyramids, which is transient. (B) Renal cortex is isoechoic to the liver in a 4 months old baby. The fetal lobulations are well appreciated and the cortico-medullary differentiation is maintained.

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Fig. 4: Longitudinal ultrasound image of a malrotated left kidney in an 11-year-old child and an axial T2 fat saturated MR image of the same patient for correlation. (A) Left kidney shows false impression of smooth parenchymal thinning of its mid-lower pole, with the renal hilum facing anteriorly. (B) MRI image provided to correlate.
Fig. 5: Images demonstrate a right kidney parenchymal junctional defect and line. (A) Longitudinal US image of the right kidney shows a wedge shaped mid-polar, hyperechoic, well-defined parenchymal defect is seen (red arrows) without associated parenchymal loss. (B) Oblique coronal and (C) oblique axial unenhanced CT sections of the same patient (done to look for ureteric stone) show fat density extending from the perirenal fat till the renal sinus at the corresponding hyperechoic defect seen on ultrasound. (D) Illustrates a case of Junctional parenchymal line of kidney (yellow arrow), seen as an echogenic thin oblique line at the upper-mid pole junction, extending from the renal sinus into the cortex.
Fig. 6: (A & B) Two cases of fetal lobulations. (C & D) Are companion cases of renal scaring due to vesico-ureteric reflux. (A) Longitudinal US scan of the kidney of a 1 week old newborn, show indentations between renal pyramids, no cortical loss, consistent with fetal lobulation (arrows) and (B) in a 15-month-old baby's kidney shows persistence of fetal lobulations. (C) Shows focal cortical thinning above the pyramid (arrowhead). (D) Shows two areas of mid-polar parenchymal thinning (arrowhead), which is replaced with an irregular hyperechoic tissue, ongoing with scar formation.

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**Fig. 7:** (A & B) Demonstrate column of Bertin in a 4 and 14-year-old child respectively. This is seen as a hypertrophied band of normal renal tissue continuous with renal cortex and extending into or indenting the renal sinus, outer renal contour is normal. (C) Longitudinal US image shows a mid polar contour bulge isoechoic to the cortex, consistent with Dromedary hump.

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Fig. 8: A case of left kidney hypoplasia. Longitudinal US images of the right kidney (A) and the left kidney (B) show normal parenchymal thickness and echogenicity, however, the left kidney is more than 1.5 cm smaller than the right kidney.

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Fig. 9: Two cases of ectopic kidney. (A) Left kidney is seen superior to the urinary bladder (asterisks), consistent with pelvic kidney. (B) right kidney is malrotated, found in the right iliac fossa.

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**Fig. 10:** An 11-month-old baby boy, a case of crossed fused ectopia on the right side. The right kidney is malrotated and elongated. (B) The left adrenal gland is elongated (Lying down adrenal gland sign) with empty left renal fossa. (C) The left kidney is seen fused to the lower pole of the right kidney, overlying the aorta and spine. (D) Tc 99m DMSA Static Renal Cortical Scan, anterior and posterior views were acquired for the kidneys 2 hours after IV injection of the radiotracer. The right kidney is seen in its anatomical place at the right loin, posteriorly oriented and the left one represents crossed fused ectopia. The left kidney is more anteriorly situated, crossing the mid line and fusing with the medial border of the right kidney.

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Fig. 11: Horseshoe kidney. Longitudinal scan of (A) right kidney, (B) left kidney and (C) fused portion of the lower poles in the midline. (D) Transverse scan with linear transducer shows an isthmus of tissue anterior to the spine and aorta connecting the lower poles of the kidneys.

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### Differential Diagnosis of Medullary Nephrocalcinosis

<table>
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<td>Hypothyroidism</td>
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<td>Corticotropin therapy</td>
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**Fig. 12:** Differential Diagnosis of Medullary Nephrocalcinosis.


**Fig. 13:** Medullary nephrocalcinosis in a patient with distal renal tubular acidosis. (A) is a longitudinal image obtained with convex transducer & (B) is a longitudinal ultrasound
image obtained with high resolution transducer, both show abundant echogenic foci (arrows) within the renal pyramids.

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**Cortical nephrocalcinosis**

- Renal tubular acidosis.
- Acute cortical necrosis.
- Chronic glomerulonephritis.
- Chronic hypercalcemia.
- Ethylene glycol poisoning,
- Primary oxalosis.
- Sickle cell disease.

**Fig. 14:** Cortical nephrocalcinosis in a patient with renal tubular acidosis. Longitudinal ultrasound image in a patient with Tuberous Sclerosis, shows echogenic punctate (arrow) and linear (arrowhead) foci in the cortex of the left kidney.

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### Differential Diagnosis of Increased Renal Parenchymal Echogenicity

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<th>Large kidney</th>
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<td>X</td>
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<tr>
<td>Acute Glomerulonephritis</td>
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<td>X</td>
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<td>Glycogen Storage disease</td>
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<td>Hemolytic uremic syndrome</td>
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<tr>
<td>Acute pyelonephritis</td>
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</tr>
<tr>
<td>Sickle cell anemia</td>
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<td>Renal dysplasia</td>
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<td>Chronic renal failure</td>
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<td>Acquired Immunodeficiency syn.</td>
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**Fig. 15:** Differential Diagnosis of Increased Renal Parenchymal Echogenicity.

Fig. 16: A 12-year-old patient with dysplastic kidney. (A) Longitudinal ultrasound image of the right kidney obtained with convex transducer shows hyperechoic renal parenchyma in a small for age (7 cm) kidney. (B) Longitudinal image obtained with linear transducer shows poor cortico-medullary differentiation and small subcapsular cyst (arrow).

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**Fig. 17:** Longitudinal ultrasound images of the right kidney in a 3-year-old male patient with Leukemia and hypocalcemia, obtained with linear transducer (A) shows hyperechoic renal parenchyma. (B) Focused high resolution imaging with linear transducer shows few punctate foci of medullary nephrocalcinosis (arrows).

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**Fig. 18:** Reflux Nephropathy due to posterior urethral valve in a 7-year-old boy. (A & B): Longitudinal ultrasound images obtained with convex (A) and linear (B) transducers reveal hyperechoic parenchyma with loss of cortico-medullary differentiation along with moderate hydronephrosis secondary to reflux nephropathy.

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### Potter’s Classification of Renal Cystic Diseases

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<td>I</td>
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<td>ARPCKD</td>
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<td>II</td>
<td>Cystic dysplastic kidney disease</td>
<td>MCDK</td>
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<td>III</td>
<td>Adult polycystic kidney disease</td>
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<td>IV</td>
<td>Partial or intermittent urinary outflow</td>
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**Fig. 19:** Potter's Classification of Renal Cystic Diseases.

© Fred E. Avni, Imaging and classification of congenital cystic renal disease. AJR. 2012;198: 1004-1013. 10.2214/AJR.11.8083
Genetic or Non-Genetic Classification of Renal Cystic Disease

<table>
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**Fig. 20:** Genetic or Non-Genetic Classification of Renal Cystic Disease

© Fred E. Avni, Imaging and classification of congenital cystic renal disease. AJR. 2012;198: 1004-1013. 10.2214/AJR.11.8083
Fig. 21: Longitudinal ultrasound images in 3 different cases of MCDK. (A) Shows multiple rounded non-communicating cysts that enlarged from 10 cm at birth to 15 cm at the age of 2 years. (B) Shows non-communicating cysts with intervening echogenic parenchyma (arrow), which showed no function on DMSA scan. MCDK can be associated with crossed fused ectopia as seen in (C) rounded cysts seen at the lower pole of an ultrasonographically normal left kidney. No kidney was seen in the right renal fossa.

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Fig. 22: ARPCKD. (A) High-resolution longitudinal ultrasound images in a 9-month-old baby show enlarged hyperechoic kidneys with numerous small rounded cysts (arrows) and loss of cortico-medullary differentiation. (B) Longitudinal ultrasound images in a 13-month-old baby show severely enlarged kidneys that they don't fit in the field of a convex array transducer (C 1-5 MHz) and their lower poles abut each other reaching the pelvis. In this case, cysts are larger, cylindrical and more widely spread than in case (A).

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Fig. 23: ARPCKD in a 3-year-old child. Longitudinal ultrasound image of the kidney with the use of convex (A) show enlarged hyperechoic kidney with well-defined anechoic renal cysts better visualized with high-resolution linear transducer, show numerous cylindrical cysts in the medulla and cortex, which represent ectatic collecting ducts. (C) Transverse US image of the liver of the same patient, show intrahepatic biliary ductal dilation consistent with Caroli’s disease in association with ADPCD (arrows).

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Fig. 24: ADPKD in an 11-year-old boy, screening US was done, as his father has ADPKD. (A) Longitudinal ultrasound images of the right kidney, show a mid-polar and another lower polar simple cyst (arrow). B: Longitudinal ultrasound image of the left kidney shows an upper polar septated cyst. There are bilateral parenchymal calcifications. The son fulfills the criteria of ADPKD.

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Fig. 25: A case of Chronic kidney disease patient, due to Infantile nephrotic syndrome, on peritoneal dialysis. (A): Longitudinal US image of the right kidney at age of 1 year old, shows small kidney with thinned out parenchyma and loss of cortico-medullary differentiation. (B & C): Longitudinal ultrasound images of both kidneys at the age of 9 years show bilateral development of renal cysts consistent with acquired cysts in uremia.

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**Fig. 26:** Zellweger syndrome. Longitudinal scans of both kidneys show increased parenchymal echogenicity and multiple various sized subcapsular cortical cysts.

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Fig. 27: Joubert's syndrome in a 5-year-old child having chronic kidney disease. Longitudinal scans of both kidneys show hyperechoic parenchyma, upper polar complex small cyst and parenchymal calcifications.

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Fig. 28: Tuberous sclerosis in (A) A 1-week-old baby and (B) a 10-year-old child. Longitudinal and transverse US images for the kidney show multiple small cortical cysts (arrow) and a linear hyperechogenic lesion (arrow heads) consistent with angiomyolipoma. Notice that the cortico-medullary differentiation is maintained.

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Fig. 29: Tuberous sclerosis in a 5 year old child, show bilateral tiny cysts, multiple punctate and linear hyperechogenicities in keeping with small angiomyolipomas. Cortico-medullary differentiation is poor.

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**Fig. 30:** (A) A 2-month-old baby diagnosed with congenital nephrotic syndrome (Denys-Drash Syndrome), longitudinal images show enlarged hyperechoic kidneys with loss of cortico-medullary differentiation. (B) DiGeorge Syndrome, longitudinal US images in a 13-year-old child show medullary nephrocalcinosis.

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**Fig. 31:** A case of Turner syndrome. Longitudinal US images of both kidneys, show bilateral few cortical cysts and cortical nephrocalcinosis.

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Conclusion

Ultrasound is a useful tool for the assessment of the pediatric renal parenchymal diseases. In knowing the significance of specifically using the high resolution linear array transducers and being well-versed with the various ultrasound appearances of diseased renal parenchymas, one can provide a wholesome evaluation of the renal parenchyma and obtain an accurate diagnosis of the ongoing pathologic process.

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