Renal failure is a serious, acute or chronic, reversible or irreversible, total or partial loss of excretory renal function with potentially deleterious sequelae, that unresolved may lead to dialysis or the need for renal transplantation. Pathophysiologically, there are three major causes:

- Intrinsic RF with a disease process affecting the kidney itself
- Postrenal RF, usually secondary to obstructive uropathy
- Prerenal RF secondary to a systemic or extrarenal disease without an essential intrinsic kidney problem

There are some situations in which more than one aspect is present or otherwise cannot be strictly classified according to these categories: systemic lupus erythematosus (SLE) with renal involvement and RF; hemolytic uremic syndrome (HUS) with RF secondary to bacterial or viral toxins and tubular congestion by hemolysis; hepatorenal syndrome; renal perfusion restriction in severe (bilateral) renal artery stenosis (RAS); traumatic renal vascular injury (tear, dissection, embolus); or crush kidneys with RF caused by tubular obstruction by myoglobin.

Clinically one can observe increased blood pressure and oliguria or anuria. Polyuria may precede or follow these stages, producing so-called “polyuric RF.” Additionally there may be more or less generalized edema secondary to fluid overload or protein deficiency. Diagnosis is based on laboratory findings with elevated serum creatinine levels, electrolyte disturbances, low protein or albumin levels, and metabolic disturbances (such as acidosis). Additional symptoms or findings may be secondary to fluid overload (eg, hypertension, cardiomegaly and cardiac insufficiency, ascites, or pleural effusion) and metabolic problems (eg, tachypnea in severe acidosis).

Treatment options vary depending on the underlying condition, with supportive measures such as fluid and electrolyte balancing and hemofiltration being common. Treating systemic conditions and restoring circulation is necessary to overcome prerenal RF, and relief of obstruction is the treatment in postrenal RF. Intrinsic RF in general offers fewer
treatment options, such as diet and fluid balance, antibiotics (in post- or parainfectious GN), steroids in some GN entities or nephritic and nephrotic syndrome (NS), and cytotoxic or immunosuppressive medication (eg, cyclophosphamide or cyclosporine in some forms of GN or NS).

In RF the role of imaging (and of US in particular) is initially to help differentiate acute from chronic and prerenal from postrenal or intrinsic RF. In some conditions, US can offer a treatment approach (eg, PCN, placing central lines for monitoring and dialysis), can assess the kidney during follow-up (eg, assessment of the amount of dilatation or by serial evaluation of renal perfusion using Doppler sonography [DS]), and can help establish the histologic diagnosis by offering a safe biopsy approach. US may also be helpful in evaluation of complications during the course of renal disease, to follow renal development and growth after recovery, or to check for sequelae and progression of a chronic disease under long-term treatment.

As this review focuses on pediatric US, several specific aspects important for diagnosis in neonates, infants, and children have to be mentioned. The neonatal kidney is still immature and thus on US appears different from a normal adult kidney. Normal creatinine levels change with age (normal in newborns, <0.5 mg/dL) because of increasing renal maturation, different metabolism, and growing body size. Different diseases occur than in adults: vascular disease is rare, although congenital malformations with renal hypoplasia or autosomal recessive polycystic kidney disease (ARPKD) with early progression are more important in this age group. Sensitivity to radiation is higher in children, thus radiation-sparing imaging is even more important than in adults. Communication is different and there may be reduced patient cooperation. The different physiology and the different tissue composition (less fat and fibrous components) and the small size, different anatomic relations, and higher heart and respiration rates need technically different imaging equipment (high temporal and spatial resolution, different transducers) and cause different imaging appearances. All of these factors necessitate adapted imaging algorithms. This requires special knowledge and training and a dedicated infrastructure, because infants require different handling than do adults (eg, swaddling facilities, breastfeeding room, pacifiers, heating, and place for accompanying or assisting persons).

The aim of this article is to briefly list the various imaging modalities applicable in pediatric RF, to reflect on the task of imaging in infants and children with RF, and then to discuss the potential of US in pediatric RF with special regard to effective use of imaging.

### Imaging modalities

Imaging is primarily based on US, supplemented by plain film, fluoroscopy, scintigraphy, and MR imaging in specific conditions and queries.

Ultrasound is the generally accepted basic imaging tool in RF. For a long time, however, US was seen as a “roughly orienting” method that allowed for depiction of collecting system dilatation or urinary tract malformations, but it did not offer any more detailed information. This started to change initially with the advent of DS, then color Doppler sonography (CDS) and particularly amplitude-coded CDS (aCDS). These tools enabled assessment of renal perfusion and helped in differentiating various conditions, such as prerenal failure, focal renal lesions, or renal vein thrombosis [1–3]. Gray-scale imaging was then improved by the introduction of high-resolution US, image compounding, and harmonic imaging [3–6]. Upcoming techniques, such as intravenous US contrast agents, together with refined sonographic contrast depiction capabilities, will further enhance US potential, making US a comprehensive, noninvasive, portable imaging tool that allows for anatomic and functional (ie, perfusion, vesicoureteral reflux [VUR]) evaluation of the urinary tract [3,7–12]. US thus has become the major imaging tool for pediatric RF.

Plain films are rarely useful or indicated in patients who have RF. Evaluation of systemic sequelae, assessment of central line position, or the initial diagnostic evaluation, however, often require a chest film, and diagnosing some underlying conditions such as urolithiasis still may require a plain film [13]. Note that film speed and exposure need to be adapted to age and weight and that digital radiography may lack sufficient resolution, particularly in neonates and infants. Fluoroscopy is applied in VCUG for diagnosing PUV or VUR with associated renal dysplasia and in angiography or intervention-al procedures, such as PCN [14–17].

The value of CT is limited in the pediatric urinary tract and in particular in RF, because RF is a contra-indication for the use of intravenous contrast material required in most pediatric urinary tract queries [18–20]. The primary indication for CT in the pediatric urinary tract is major trauma or work-up of renal tumors and tumorlike lesions, usually not presenting with RF and as such beyond the scope of this article.

The two other major imaging players in RF are scintigraphy and, increasingly, MR imaging. Static renal scintigraphy (Tc⁹⁹m DMSA) is used for evaluating overall and split renal function. Diuretic dynamic renal scintigraphy (Tc⁹⁹m MAG3) is considered the gold standard for assessment and grading of obstructing uropathy. MRI has successfully
been introduced into pediatric uroradiology [21–28]. New technical refinements and sequence modifications promise to widen the potential of MR urography (MRU) to become a comprehensive “all in one” imaging tool, offering not only anatomic assessment but additionally providing detailed and quantitative functional information. MR contrast material may be given in RF because of its reduced nephrotoxicity, allowing assessment of renal perfusion and residual function. Diffusion-weighted imaging and new intracellular contrast materials promise to open yet unexplored diagnostic fields for imaging diagnosis and prognostic assessment.

The use of imaging in typical pediatric diseases and common clinical queries

Severe pediatric RF is a rare but serious and life-threatening condition that needs urgent diagnosis and treatment. The differential diagnoses of neonatal RF include [29–35]:

- Intrinsic renal diseases, such as severe urinary tract infection, renal vein thrombosis, hypoxic and toxic renal parenchymal damage (including treatment- or drug-induced), congenital hypodysplasia, bilateral renal agenesis, ARPKD, congenital NS, syndromic nephropathies, and neonatal GN
- Postrenal problems caused by bilateral severe obstructing uropathy (ie, PUV, megaureter with uretero-vesicle junction obstruction, and uretero-pelvic junction obstruction)
- Prerenal RF caused by systemic problems, such as sepsis and multiorgan system failure, heart disease (particularly PDA and aortic coarctation), hypotension and hypovolemia (eg, after placental bleeding or uterine rupture), or dehydration and hyperviscosity

In older children, RF may evolve because of an underlying chronic condition, some of them syndromal or hereditary (eg, reflux nephropathy, [RNP]), juvenile nephronophthisis, or Alport syndrome), and subsequent chronic RF is more common. Additional intrinsic renal diseases that may cause acute RF entities have to be considered: HUS, NS, para- or postinfectious GN, tubular renal acidosis (or oxalosis and cystinosis) with consecutive progressive nephrocalcinosis, various tubulopathies, renal involvement in systemic diseases such as in Henoch-Schönlein disease or SLE, or toxic- and drug-induced RF (ie, herbs and fungus, chemotherapy, antibiotics, or intravenous iodinated contrast agents) [20,36–42]. Furthermore, acute obstruction caused by urinary tract calculus or an abdominal tumor, renal destruction by tumors, or chronic infections, such as xanthogranulomatous pyelonephritis, and traumatic RF (eg, bladder rupture, crush kidney, or vascular injury) need to be mentioned as rare causes for pediatric RF to complete the list. Looking at all these entities, it becomes obvious that a thorough and detailed discussion of these diseases would fill a book and thus is beyond the scope of this brief overview. Most nephropathies, however, have similar or even identical imaging appearances. The author focuses only on modern US features in typical neonatal and pediatric conditions.

Intrinsic renal conditions

Neonatal presentation of RF is often suggested prenatally; the correct diagnosis of the most common renal causes, however, is eventually established after birth. ARPKD and congenital NS (“of the Finnish type”) or—less often—neonatal GN are the most common causes, with some more or less typical imaging features. In general, ARPKD exhibits the typical “pepper and salt” appearance of the parenchyma of an enlarged and hyperechoic kidney, usually with no or just a few tiny cysts seen (Fig. 1) [32–35]. Neonatal GN and NS also present with an enlarged kidney and some alteration of the parenchymal echo pattern; no specific features are known, although some differences in corticomedullary

Fig. 1. (A) Transverse sonogram of neonatal ARPKD shows echogenic parenchyma without any corticomedullary differentiation. (B) Transverse sonogram in an infant who had syndromic cystic renal parenchymal disease shows multiple parenchymal cysts and absent corticomedullary differentiation.
Differentiation and in the amount and pattern of echogenicity changes have been noted to be more common in some diseases [34,35,43]. Even DS findings (reduced peripheral vasculature on aCDS, increased or decreased RI or velocities on DDS) are nonspecific and correspond better with renal function (degree of renal failure, modulated by therapeutic aspects such as fluid load, drugs, blood pressure modulation, and heart rate) than with the underlying disease entity. This is also valid in other forms of GN or NS as seen in older children, such as para- and postinfectious GN, Henoch-Schönlein nephritis and IgA-nephropathy, lupus nephritis, or (familial) NS (Fig. 2) [3,38,40,41]. Usually kidney size is enlarged or normal, and US appearance depends on the relative echogenicity of the cortex and the parenchyma. Corticomedullary differentiation thus depends on whether both or only one of these structures is affected, how much each of them are affected and may be increased or decreased, or sometimes are even normal in mild disease. The US appearances are thus rather nonspecific, and the value of US in these patients is not to specify a certain disease (although some suggestions concerning the underlying disease may be achievable), but rather to (1) rule out other potential causes for RF; (2) assess the amount of pleural effusion or ascites and intravascular fluid load for balancing supportive measures, and (3) assess extrarenal disease aspects.

Fig. 2. (A) Unspecific renal parenchymal changes in severe Lupus nephritis. (B) Significantly reduced peripheral renal perfusion in aRF shown by aCDS. (C) Reduced perfusion of the renal cortex (cursors) becomes even more obvious when compared with the aCDS vascularity of the adjacent spleen. (D) Transverse sonogram through a right kidney in NS. (E) Peripherally reduced vasculature on aCDS (halo sign) indicating restricted peripheral renal perfusion.
Renal vein thrombosis

Renal vein thrombosis mostly occurs in neonates who have adrenal gland hemorrhage, coagulopathies, femoral central lines, or dehydration and polyclonal syndrome [3,44–46]. Note that unilateral renal vein thrombosis usually presents with hypertension and hematuria and should not lead to global RF except for single systems or coexisting impairment of the other kidney. In neonates, thrombosis usually starts in the peripheral veins, only gradually growing into the central veins, behaving similarly to tumor thromb (Fig. 3A). In older children, extrarenal origin of thrombosis or a primary thrombus of the major renal vein is more common. The basic and often single imaging tool is US, demonstrating a large, swollen, and hypechoic kidney with undifferentiated parenchyma (Fig. 3B). On CDS, renal vein color signals are missing in the affected veins, although they may be demonstrable in patent central veins. DDS shows increased

![Fig. 3](image)

(A) Transverse sonogram showing renal vein thrombus (between cursors) reaching into the IVC. (B) Longitudinal image showing a swollen kidney with hazy corticomedullary differentiation in neonatal renal vein thrombosis. Note the secondary ascites (A). (C) Longitudinal aCDS image demonstrates regionally diminished perfusion in partial/peripheral neonatal renal vein thrombosis. CDS with DDS trace depicts the (D) increasingly reduced and then (E) reversed diastolic flow with elevated RI.
arterial resistance with high resistive indices (RI) and low or missing diastolic flow (Fig. 3C–E). On follow-up—even with some treatment success—one often observes shrinkage of the kidney with diffusely abnormal echo pattern of the parenchyma and reduced perfusion. These changes sometimes only become apparent over time, as a lack of age-concordant renal growth with contralateral compensatory renal hypertrophy. Usually no other imaging is needed except for tumor conditions, in which MR imaging (or CT) and MR angiography (MRA) are performed for overall assessment and staging and preoperative planning.

**Congenital hypodysplasia**

Congenital hypodysplasia (with RF) may be somewhat difficult to diagnose. Only the most severe forms (eg, bilateral multicystic dysplastic kidney) are easily and often prenatally recognizable, though sometimes not compatible with extrauterine life. As described by the name, the affected kidneys usually are small and present an altered echotexture with reduced corticomedullary differentiation and increased echogenicity; cysts of varying size may be present (Fig. 4A). The amount of these changes depends on the degree of dysplasia, however, and pure hypoplasia may initially even exhibit normal US features, with the kidney size initially ranging within normal limits [32–34]. Only over time does the size deficit perhaps become obvious and then may eventually correspond to developing and progressive chronic RF. This entity may be difficult to be distinguished from RNP (a term that is increasingly under discussion). The underlying renal pathology in RNP usually is a combination of coexisting congenital renal dysplasia with renal growth retardation and acquired renal damage caused by infection and scarring associated with VUR that may have vanished by the time of diagnosis (Fig. 4B). The “water hammer theory” for RNP is (postnata!lly) probably only valid in some patients who have severe, long lasting, high pressure VUR usually associated with bladder dysfunction.

**Hemolytic uremic syndrome**

HUS is a condition in which acute RF is caused by toxins of a certain *Escherichia coli* strain affecting the renal cortical and glomerular capillaries causing vasculopathy, hemolysis, and thrombosis. It is a disease that may occur as familial or endemic (in certain areas and populations) and incidentally, preferably affecting young patients during early summer. Diagnosis is assumed in case of a recent, sometimes hemorrhagic enterocolitis with acute RF; thrombocytopenia, anemia, hypertension, and typical erythrocyte morphology on microscopy (fragmented cells called Burr cells or schistocytes), and is confirmed by culture results. Treatment options are restricted to symptomatic measures and renal replacement therapy. Prognosis varies; in a single event a good outcome is common. In prolonged, chronic, and recurrent disease, prognosis concerning renal functional outcome is poorer, though mortality has been nearly eradicated. US demonstrates bilaterally enlarged kidneys with a large, hyperechoic cortex and increased corticomedullary differentiation (Fig. 5A,B). RI is markedly increased (Fig. 5C), and aCDS exhibits diffusely reduced cortical perfusion with a marked uncolored peripheral halo [34,39–41]. Additionally, US examination may demonstrate regional bowel wall thickening and secondary signs of acute RF and bowel inflammation. During the course of the disease, improvement of DDS with gradually improving RI values precedes clinical improvement of renal function and thus may be useful for monitoring and for making therapy decisions.

Eventually US can help to establish the histologic diagnosis in intrinsic renal conditions producing RF, which may be essential for treatment decisions and prognostic estimation, by providing safe biopsy guidance [14–17,47–49]. Here US not only helps to guide the biopsy, thus increasing safety and
enabling a satisfactory specimen harvest with a minimum of needle passes, but is also essential for postbiopsy monitoring and identification of postbiopsy complications, such as hemorrhage, urinary tract obstruction by clots, or postbiopsy arteriovenous fistula (AVF) (Fig. 6).

**Postrenal conditions**

In obstructing uropathy, all features of acute or chronic urinary tract obstruction may be observed. Acute obstruction (eg, by a ureteral calculus) usually exhibits little dilatation, as may severe obstruction

*Fig. 5.* (A) Longitudinal sonogram of an enlarged right kidney demonstrates the typical US appearance of an acutely affected kidney in HUS with increased cortical echogenicity and corticomedullary differentiation. (B) Transverse CDS with duplex tracing demonstrates the markedly reduced perfusion with reversed diastolic flow and increased RI.

*Fig. 6.* (A) US-guided biopsy with the needle (arrowheads) passing into the lower pole of the left kidney as predefined by the dotted needle trace of the guiding device. (B) Postbiopsy hematoma on the lower pole of the left kidney (arrowheads), superiorly delineated by aCDS. (C) CDS depicts a postbiopsy AVF as shown by focal aliasing in the lower pole and elevated diastolic arterial flow.
with subsequently deteriorated renal function. US demonstration of dilatation of the renal collecting system or the ureter does not necessarily equal obstruction. To exhibit a severely dilated system, good urine production is needed and thus this finding may indirectly indicate persisting renal function [31,32]. Polyuria (polyuric phase of RF, stimulated diuresis, volume overload) and laxity of a collecting system (eg, in neonates or in infection) may cause some dilatation that should not be misinterpreted as urinary tract “obstruction.” In these conditions RF only occurs in single obstructed systems or in bilateral disease (such as bilateral UPJO or PUV with bilateral obstructing or refluxing megaureter and renal dysplasia) (Fig. 7).

The role of US is to detect collecting system dilatation and to suggest the cause and the level of obstruction (urinary calculus, UPJO, accessory renal artery) (Fig. 8A,B). US may help to differentiate acute from chronic obstruction; in acute obstruction DDS may reveal an asymmetrically elevated RI at usually little dilatation and diffusely increased echogenicity of the enlarged and swollen kidney [3,44–46,50–53]. In this setting, modern US tools such as aCDS, DDS, and 3D US may become helpful. For example, the depiction of an ostial ureteral jet into the urinary bladder by CDS may demonstrate ureteral patency and the UVJ (see Fig. 7A); diffusely decreased (peripheral) renal perfusion on aCDS (halo sign) may hint toward more severe

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**Fig. 7.** (A) Transverse CDS through the lower pelvis and urinary bladder demonstrates a right ostial ureteric jet, with lack of depictable urine inflow into the bladder on the left side caused by a distal ureteral calculus. (B) The "twinkling sign": CDS demonstrates twinkling color signals within the distal ureter at the ureterovesical junction caused by a small distal ureteral calculus in the same patient as seen in (A). (C) Echo-enhanced urosonography (A). Initial longitudinal sonogram shows a dilated ureter (U) posterior to the urinary bladder (B). After instillation of Levovist into the prefilled urinary bladder, VUR is seen as echogenic material in the dilated distal ureter (U). Furthermore, the initially narrow renal pelvis of the corresponding kidney (C) becomes markedly dilated and filled with US contrast material (D), indicating high-grade VUR. (D) Longitudinal perineal US demonstrating PUV (arrow) with the typical dilated bladder neck appearance. B, bladder; A, anal canal. (E) 3D US in hydronephrosis: outlining of outer renal contour (green line) for volume calculation in three orthogonal sections, with deduction of the segmented dilated collecting system as visualized in the right lower box for estimation of renal parenchymal volume.
conditions; the “twinkling sign” on CDS improves detection of ureteral calculi (see Fig. 7B); perineal US may enable detection of a PUV (see Fig. 7C); echo-enhanced urosonography may enable differentiation of refluxing versus obstructing uropathy (see Fig. 7D); and relative renal parenchymal volume assessment by 3D US may help estimate split renal parenchymal size (see Fig. 7E) [3,4,6–12].

In severe obstruction or pyohydronephrosis, US is the ideal imaging modality to guide percutaneous nephrostomy (PCN), potentially complemented by fluoroscopy for comprehensive visualization of the entire collecting system anatomy and for detection of potential extravasations [10,14,15,17]. Note that indications for operation and PCN have changed over the past decade; now only severe conditions with acute threat to renal function or intractable urinary tract infection are considered indications for these invasive treatment options.

Pre-renal conditions

Pre-renal RF is defined by an extrarenal condition causing renal hypoperfusion and hypoxia. The most common causes are trauma with blood loss and shock or other causes of prolonged hypovolemia and hypoxia (eg, drowning, complicated operation, asphyxia, vascular injury) and cardiac disease or malformations, such as persisting duct of Botallo (patent ductus arteriosus) with left-to-right shunt, aortic coarctation, and heart failure. The US findings are nonspecific, demonstrating normal kidneys (particularly in early stages) or bilaterally swollen, often diffusely hypechoic kidneys. Perfusion alterations on DS, such as decreased flow velocities with “pseudonormal” or elevated RI values, flattened systolic flow curves, and pathologically delayed systolic flow acceleration can be seen (Fig. 9). US is used to assess the degree of perfusion disturbance, to find or rule out other renal conditions, to evaluate the systemic changes of the underlying condition, such as free peritoneal or retroperitoneal fluid (in trauma), cardiac function (by echocardiography), brain perfusion (by transfontanellar or transtemporal DDS), and to monitor renal perfusion as a prognostic indicator or for treatment guidance.

Renal transplantation

Increasingly, even small children who have terminal RF can now be treated by renal transplantation, with an improved long-term outcome and a better life quality compared with chronic dialysis, which may still be needed until a compatible transplant organ is available. Imaging in these children is primarily based on US. Initially, donor organs are evaluated by US before explantation. Then early postoperative surveillance uses serial US and DDS/CDS, particularly in evaluating transplant malfunction (eg, tubular necrosis, early rejection, vascular problems, and obstructed urinary drainage). Assessment of kidney size, renal parenchymal structure, and potential dilatation of the collecting system is mandatory, as is visualization of the main supplying vessels, including a DDS and aCDS assessment of renal perfusion. Furthermore, evaluation of the vascular anastomosis is desirable to depict stenoses or aneurysm formation (Fig. 10).

During later post-transplant phases, routine US investigations are performed to monitor renal growth and to detect early stages of potential rejection or (Cyclosporine-induced) vasculopathy, particularly if clinical and laboratory findings indicate a potential problem. In this scenario, transplant organ size changes are one of the most sensitive, though unspecific, signs to detect transplant disease. Additional findings in transplant disease are changed parenchymal echogenicity and corticomedullary differentiation, dilatation of the collecting system (in stenosis of ureteral anastomosis), increased RI values (in rejection and vasculopathy), flattened systolic flow acceleration (in renal artery stenosis, usually located near the anastomosis), regional venous flow turbulences (more likely in
vasculopathy or partial renal vein thrombosis), AVF (after biopsies), and impaired peripheral perfusion demonstrated by reduced peripheral color signals on aCDS (usually in significant functional impairment) or regional lack of aCDS-depictable perfusion (in segmental infarction). US, however, (and scintigraphy and MR imaging) often remains nonspecific as to the disease entity, and US-guided transplant biopsy may become necessary for definitive diagnosis and further management decisions. Extrarenal complications, such as post-transplant lymphoproliferative disease, have to also be considered in long-term imaging follow-up of transplant patients.

**Follow-up evaluation**

Finally, follow-up is a critical aspect of managing patients who have acute or chronic RF. US serves as a follow-up tool, allowing for noninvasive monitoring of renal perfusion, evaluation of collecting system dilatation, and assessment of renal parenchymal growth. Even after renal transplantation or during dialysis, the original kidneys, the urinary bladder, and the renal transplant may develop disease that needs imaging, such as secondary cystic changes, malignancy, infection, or transplant malfunction. Other imaging modalities, such as VCUG, IVU, plain film, scintigraphy, or MRU, may be indicated for a complete work-up of the underlying condition, particularly preoperatively and in complex urogenital malformations. Similarly, following patients who have chronic RF also may require the use of other imaging modalities, such as plain film or MR imaging, for diagnosis of systemic sequelae or treatment complications, such as hypertension and cardiomyopathy or osteonecrosis during steroid treatment.

**Summary**

US is the ideal and often single imaging modality in infants and children who have acute or chronic RF. Modern US tools and US-guided renal biopsy offer extended diagnostic capabilities, eventually enabling a safe histologic definition of a nephropathy.
that cannot be sufficiently diagnosed by clinical, laboratory, and imaging findings. The imaging approach for children who have RF thus heavily relies on US as the primary imaging modality. It always starts with an initial US study (including DDS and CDS) to differentiate pre-, post-, or intrarenal origin of RF and to assess the severity of renal perfusion (and thus functional) impairment. Uncommonly, other imaging techniques, such as plain film, scintigraphy, or MR imaging are necessary for initial assessment and therapeutic decisions, depending on the initial US results, the general patient condition, and the underlying disease. Follow-up imaging assessment is primarily by US (focused on renal size, parenchyma, and perfusion), often complemented by scintigraphy (or, in the future, increasingly by MR imaging) to assess overall renal function and parenchymal scarring. Occasionally, specific diseases or complications may indicate additional imaging; some conditions may be diagnosed or managed by US-guided interventions.

Modern US techniques, a US facility and equipment adapted and suited for pediatric needs, specific knowledge, and education are compulsory to make the utmost use of the great potential of US. Children are not just small adults, but present specific diseases and different imaging appearances, and deserve special, focused, and skilled care.

References


