

Ultrasonography of the kidney and the renal vessels
Part I: Normal findings, inherited and renoparenchymatous diseases

Prof. Dr. med. Jörg Radermacher
Dept. of Nephrology
Klinikum Minden
Friedrichstrasse 17
32427 Minden
Tel: 0571/ 801 – 3021
FAX: 0571/ 801 – 3076
E-Mail: Joerg.radermacher@klinikum-minden.de

Abstract

Renal ultrasonography has become the standard imaging modality in the investigation of kidneys because it offers excellent anatomic detail, requires no special preparation of patients is readily available and does not expose the patient to radiation or contrast agents.

Ultrasonography is used to determine the site and size of the kidney and to detect focal lesions like tumors, cysts and renal stones. Furthermore the presence and urodynamic relevance of hydronephrosis can reliably be found.

The presence of renoparenchymatous disease as such is also discernible to the experienced investigator, however most glomerular diseases cannot be further sub classified. Exceptions are primarily renovascular disorders like hypertensive nephrosclerosis, diabetic nephropathy or renal vasculitis which can be suspected if the intrarenal resistance index value is increased.

Color Doppler sonography in experienced hand allows the reliable detection and quantification of renal artery stenosis and increased resistance index values may indicate irreversible disease.

Ultrasonography has also been found of value in the evaluation of renal transplant kidneys. Especially in the early transplant course potentially fatal but reversible diseases like renal vein thrombosis or urinomas are detected with high sensitivity. In the long term course an increased resistance index value may also predict allograft failure.

Introduction

Renal ultrasonography has become the standard imaging modality in the investigation of kidneys. Renal size and location can be determined. Solid tumors can be detected and can be distinguished from renal cysts. Ultrasonography can detect nephrolithiasis and hydronephrosis. Dilated ureters can frequently be followed up to the location of the occluding concrement. Different renoparenchymatous diseases, especially the glomerulonephritic diseases cannot be differentiated by ultrasonography. However, the experienced investigator can almost always distinguish normal from diseased kidneys. Detection of renal arteries is reliably possible with Color Doppler sonography. A separate discussion of B-mode and Color Doppler sonography does not always make sense, because frequently only the combined use of both techniques (urinary tract obstruction, renal tumors, renal cystic disease, diabetic nephropathy...) enables the investigator to make a diagnosis. The first part of this review article will focus on normal findings and renoparenchymatous disease. The second part will

deal with focal findings including renal tumors. For more in depth information the reader is referred to books and review articles.¹⁻⁶

Patient preparation and ultrasonographic normal findings

Preparation of the patient (fasting, carminatives) is rarely necessary. The investigation should be performed in a warm, dark room with the patient lying on his back. For relaxation of the abdominal rectus muscle the investigation should be performed with the upper body elevated by 30° and possible with slightly bend knees (knee roll). This will cause a slightly more sub costal position of the kidneys which facilitates the investigation. In rare cases placement of the patient on his left or right side may become necessary to overcome poor visualization due to interposition of bowel or to have a better view on the origin of the renal arteries. Changing patient position however, may greatly prolong investigation time, especially in older patients. A primary approach with the patient in the prone position is not advised – unless one wants to perform a renal biopsy - because of poorer kidney visualization through the psoas muscle. In case of renal biopsy a more dorsal position of the kidneys is achieved by placing the patient on a bed roll. A 2-5 MHz convex array scanner is usually used in adult sonography. Scanners with higher frequencies have a lesser penetration depth but allow better visualization of kidney stones. Massive meteorism may be a lesser problem with the use of a sector scanner. Harmonic imaging modalities, which are available in most modern machines, allow better visualization of kidneys (see below). If available a Color Doppler machine with at least a pw-Doppler should be used.



Figure 1: Better visualization of the right kidney with harmonic imaging and B-mode contrast enhancement (photopic)

Examination procedure: To compare kidney with liver echogenicity a transhepatic depiction of the right kidney from a ventrolateral approach is advisable. The caudal pole of the kidney however is frequently not visible due to interposing bowel gas. Depiction of the whole kidney in longitudinal and transverse sections is usually possible from a dorsolateral sub costal approach. A valsalva maneuver will frequently be necessary to see the upper pole of the kidney. Mobility of a normal kidney should be 3-7 cm. Since deep inspiration may lead to elongation of kidneys with a concomitant reduction in depth and width all dimensions should

be measured in the same breath hold position. Otherwise false renal volume calculations may result.

Normal findings



Fig. 2 Normal kidney

Renal shape and -position

The kidneys are located retroperitoneally and slide on the musculus quadratus lumborum and musculus psoas during in- and expiration. Due to loin lordosis the lower pole of both kidneys is located more ventrally than the upper pole. The upper pole of the right kidney is transversed by the 12th and the upper pole of the left kidney by the 11th rib. This means, the right kidney is usually positioned 1 to 2 cm more caudally than the left kidney. The right kidney surface is adjacent to the lower liver boarder, so the liver can be used as an ultrasound window for the right kidney. The kidneys are shaped like beans, convex on the lateral und concave on the medial sight.

Renal surface and capsule

The kidneys are enclosed by an adipose capsule the thickness of which varies depending on the general constitution of the patient. This adipose capsule can have high or low echogenicity

and may be mistaken for the renal parenchyma in patients with shrunken kidneys. However, the missing mobility during in- and expiration usually allows clear differentiation of surrounding fatty tissue and renal parenchyma. Kidneys usually have a smooth surface. A normal variant are renal renculi. These are the remaining signs of fetal lobulation and are more easily recognizable in the right kidney. Two renculi always surround a medullary pyramid.

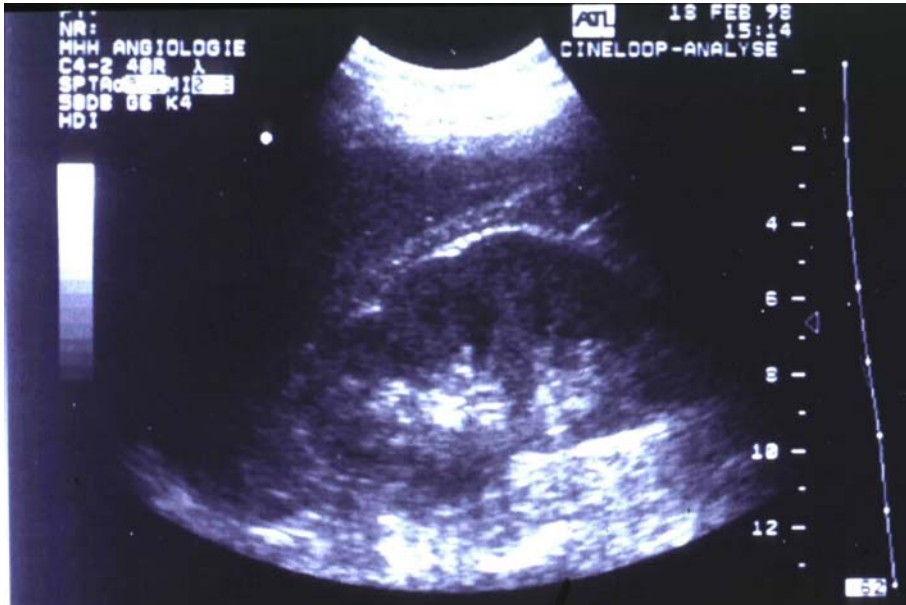


Figure 3. Persisting fetal lobulation of the right kidney (renculation)

Renal parenchyma

An inexperienced investigator should assess the renal parenchyma only in comparison to the adjacent liver and spleen. The normal renal parenchyma in children age 6 and older and in adults should be slightly less echogenic than that of liver and spleen. Normal renal parenchyma from birth until 6 months of age is slightly brighter than that of the liver. Increased echogenicity compared to liver and spleen in adults is a sensitive but unspecific sign of renal disease. The normal parenchymal width of 15-25 mm can be measured most reliably from the basis of a medullary pyramid to the kidneys surface. The renal hilum should be visible when measuring parenchymal width to avoid underestimation. If such a depiction is not technically possible a normal parenchymal width can be assumed if the parenchyma-pelvis relation is normal. The relation of ventral and dorsal parenchymal width to pelvic width should be 2 : 1. Medullary pyramids are usually less echogenic than the surrounding parenchyma. Columns of Bertini (columnae renales) are extensions of the renal parenchyma in the renal pelvis and hypertrophied columns of Bertini should not be confused with pelvic tumors. The left kidney sometimes has a very wide parenchyma in the medial portion, a so called splenic notch or dromedary hump. This also is a normal finding and should not be confused with renal masses. The echogenicity in the area of the hump will be the same as in the surrounding parenchyma and the medullary pyramids will still be present and the vessel architecture will be unaffected.

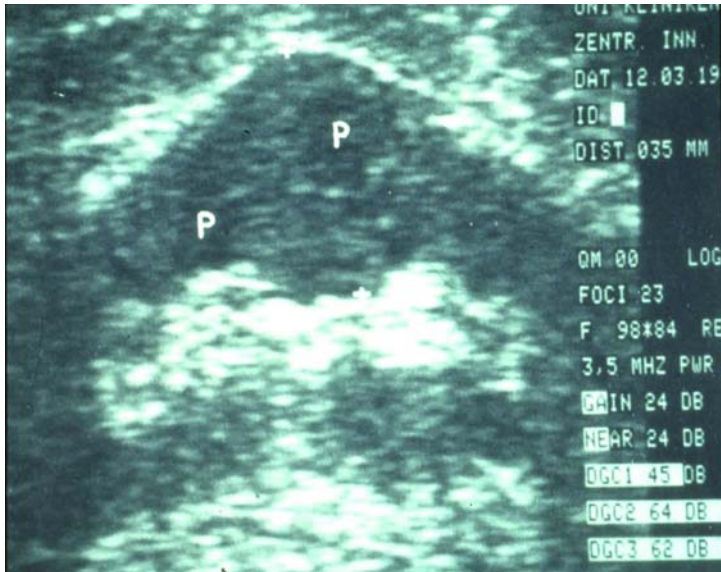


Abb. 4 Splenic notch (dromedary hump), P denotes medullary pyramids

Renal sinus

The central echogenic part of the kidney (sinus) is composed of the pelvis and the calyces, of blood and lymphoid vessels and interposed adipose tissue. The normal renal pelvis should not be fluid filled except in pregnant women. Anechoic areas are frequently due to dilated veins as can be easily proven by Color Doppler ultrasound. The ureter is located dorsally to the renal vessels and also should not be visible normally.

Renal vessels

The renal artery usually branches within the renal sinus or extrarenally in 2 to 3 segmental arteries of first order and these 1st order segmental arteries branch another 2 to 3 times into segmental arteries of 2nd and 3rd order. When entering the renal parenchyma the vessels are called interlobar arteries. From these the arcuate arteries branch off in a 90° angle, running parallel to the kidney surface and the interlobular arteries, which run towards the renal capsule, originate from the arcuate arteries. The right renal vein is relatively short (4 cm) and rarely visible from a ventral approach due to overlying bowel gas. The left renal vein runs between aorta and superior mesenteric artery and can rarely be compressed between these two vessels (so called nutcracker phenomenon). A retro aortal course of the left renal vein is also possible.

Estimation of normal renal size

Chronic renal disease frequently leads to renal shrinking. Reliable criteria depicting a shrunken or enlarged kidney have not been published for adults. A renal length of 9-12 cm is considered normal, renal length correlating to body length. The correlation is poor however. Frequently the right kidney is shorter than the left kidney whereas renal function estimated by scintigraphy and renal volume estimated by CT are equal. A better correlation can be found between renal volume and body weight or body surface area. Renal volume is frequently estimated as $\text{length} \times \text{depth} \times \text{width} \text{ (cm)} / 2$. In children with healthy kidneys normal renal volume (ml) could be estimated as $\text{body weight (kg)} \times 2$. The few available data in adult

populations – which were not evaluated for normal renal function - do not contradict these findings. A normal renal volume can therefore be defined as body weight (kg) x 2 ± 20%.

Congenital renal diseases

Renal agenesis or hypoplasia: Usually a chance finding. Unilateral small or missing kidney with normal architecture and echogenicity. Color Doppler and Doppler ultrasonography also show normal findings. The contralateral kidney shows compensatory hypertrophy. Caveat: Unilateral small kidneys due to renal artery stenosis will also result in – age dependent – hypertrophy of the contralateral kidney. Here altered Doppler signals in the small kidney will lead to the correct diagnosis. Renal agenesis cannot be proven by ultrasonography (see ectopy).

Ectopic kidneys: Usually small and malrotated. Located between the urinary bladder and the normal position. In crossed ectopy both kidneys are located on the same side and are frequently fused and therefore enlarged. Frequently two or more renal pelvices are present. Sometimes the kidneys can only be found with CT or MRT.

Horseshoe kidney: Fusion of both lower kidney poles with a parenchymal or connective tissue bridge (isthmus) ventral to the aorta. Here the fused part may be mistaken for enlarged lymph nodes. The lower poles of the kidneys are misplaced medially. This malformation is frequently accompanied by vesicoureteral reflux, stone formation and urinary tract obstruction.

Double kidney: Most frequent malformation (incidence 0,5-10%). B-mode ultrasonography shows a parenchymal bridge which completely separates the cranial from the caudal part of the kidney. Frequently these kidneys are enlarged compared to the contralateral kidney. The parenchymal bridge can be confused with hypertrophied columns of Bertini. Ultrasonography cannot prove the presence of double kidneys; this can only be done by an iv-urogramm showing two renal pelvices and two ureters. Reflux or renal pelvis obstruction occurs more frequently in double kidneys. Urinary tract obstruction may lead to dilatation of the renal pelvices in which case a double kidney can be proven by ultrasonography alone.



Figure 5: Enlarged left kidney with a parenchymal bridge which completely traverses the renal sinus. Due to crossed ectopy a further small renal parenchymal area and sinus can be seen in the lower third. This is the hypoplastic fused part of the right kidney.

Renoparenchymatous diseases

Renoparenchymatous diseases can be classified as uni- or bilateral diseases with small or large kidneys (see table 1). The method to evaluate kidney size as normal or not has been shown above. A general rule is that small kidneys depict chronic renal disease and enlarged or at least normal sized kidneys depict acute and therefore potentially reversible disease. A further distinguishing feature is parenchymal echogenicity. Diseased kidneys in general show increased echogenicity, however in the early stages echogenicity may still be normal. Increasing echogenicity is directly correlated to histopathological findings like global sclerosis, tubular atrophy, leukocyte infiltration and the number of hyaline casts per glomerulum. Decreased echogenicity is correlated to the magnitude of interstitial edema^{7, 8}. The normal renal parenchyma should be slightly less echogenic than that of the liver. This is problematic insofar, that it assumes liver parenchyma to be of normal echogenicity. Many patients, however, have fatty liver disease. Objective and easily applicable methods for quantification of renal parenchymal echogenicity are missing. Therefore the classification of renal echogenicity depends to a large amount on investigator experience. A further criterion separating normal from abnormal kidneys is renal perfusion. The renal resistive index (RI = decrease of minimal diastolic in relation to maximal systolic Doppler flow velocity, e.g. 0.6 = 60% decrease) has been best evaluated so far. The RI is increased in hypertensive nephrosclerosis⁴ and correlates with the histological severity of glomerulosclerosis and arterio- and arteriolosclerosis. Table 1 summarizes the major diseases associated with small or enlarged kidneys.

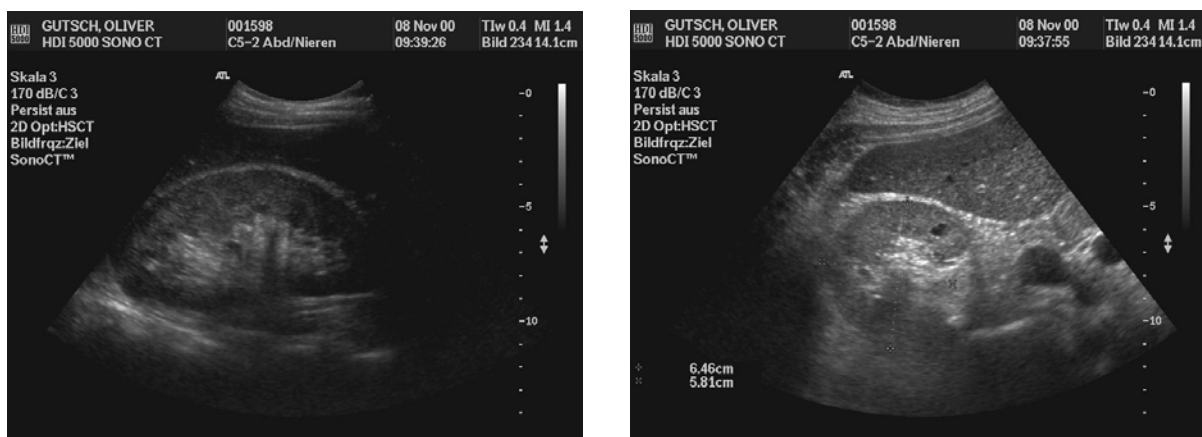


Figure 6: Right kidney with increased echogenicity as a sensitive but unspecific sign of renal disease. Compare the echogenicity of the kidney with that of the overlying liver

Diabetic nephropathy

Diabetic nephropathy is the most frequent renoparenchymatous disease, accounting for 40% of cases of incident terminal renal failure. Diabetic nephropathy is almost always associated with enlarged kidneys prior to terminal renal failure. Echogenicity increases with increasing stages of renal failure; however in earlier stages of disease echogenicity is frequently normal. A further diagnostic criterion is an increased RI (see below) however, the increased RI occurs relatively late in the course of diabetic nephropathy when other signs like microalbuminuria are also already present. Even when terminal renal failure is present diabetic kidneys remain relatively enlarged as opposed to kidneys from patients with glomerulonephritic or renal interstitial disease. Diminished parenchymal width or a kidney with a low volume in a patient with diabetes can hint at superimposed hypertensive nephrosclerosis. Renal scars, abscesses and papillary necrosis can also occur in diabetic nephropathy.

Glomerulonephritis and vasculitis

There is no specific ultrasonographic sign for glomerulonephritic disease. Depending on the degree of renal functional impairment kidneys frequently lose volume and almost always show increased echogenicity but no renal scarring. Increased echogenicity is less notable in IgA nephropathy, minimal change disease and membranous glomerulonephritis and corticomedullary differentiation is better preserved than in proliferative and interstitial glomerulonephritis. In acute glomerulonephritis kidneys are enlarged or of normal size, the parenchyma is frequently of increased depth and echogenicity but normal or even decreased echogenicity can also occur. A renal segmental artery RI value of 0.80 or greater is a bad prognostic sign⁹. About 3-5 years after initiation of dialysis treatment secondary cyst formation frequently occurs. These cysts may undergo malign transformation. For this reason yearly control ultrasonography is indicated

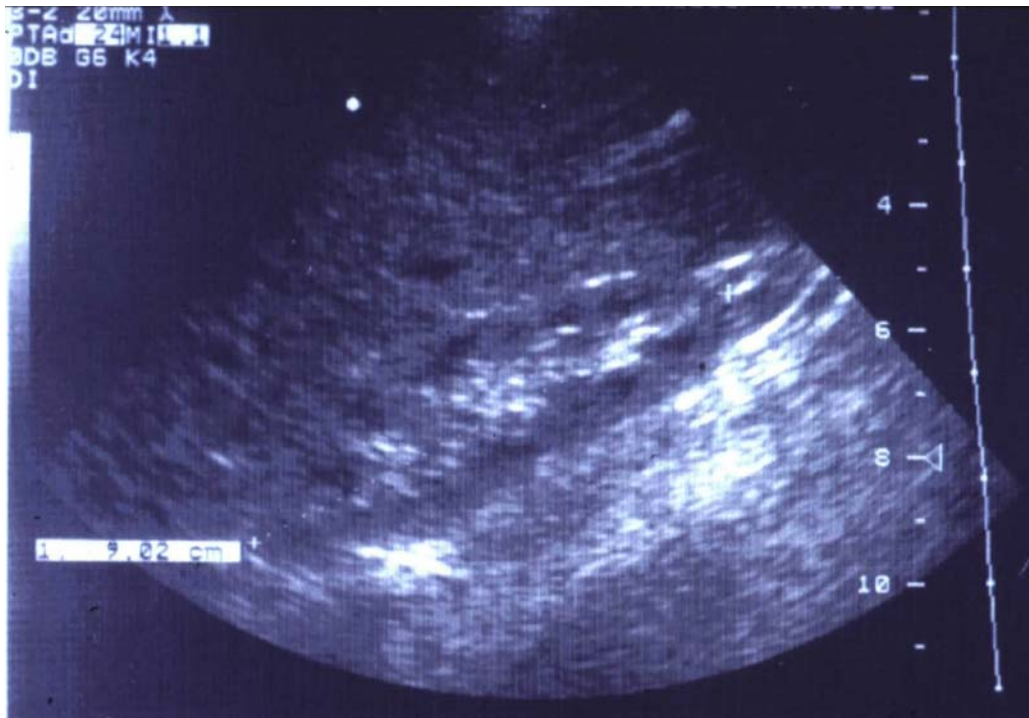


Figure 7: Condensed and small right kidney in preterminal renal failure due to chronic IgA nephropathy

Hypertensive nephrosclerosis

Hypertensive nephrosclerosis is frequently associated with small and echo dense kidneys and parenchyma of diminished thickness. The renal resistive index is frequently elevated above 0.80 and this is a bad prognostic sign.

Amyloidosis

Enlarged kidneys with thick parenchyma, greatly increased echogenicity, preserved corticomedullary differentiation, medullary pyramids with low echogenicity and an increased renal resistive index ($> 0.7-0.8$) are the unspecific hallmarks of renal amyloidosis.

Acute renal failure

In acute intrarenal failure kidneys are frequently enlarged, parenchymal echogenicity is increased, medullary pyramids appear to have low echogenicity and renal RI is severely increased. In acute prerenal failure renal echogenicity and renal RI is frequently normal. Patients with severely increased echogenicity appear to have a worse prognosis.

Hemolytic uremic syndrome

Due to endothelial damage occlusion of small intrarenal vessels is the hallmark of this disease. This process is accompanied by an increased renal RI value (> 0.80). In addition to the clinical presentation and laboratory parameters like elevated LDH, thrombocytopenia and fragmentocytes the increased RI can be used as a diagnostic clue.

Hepatorenal syndrome

According to B-mode ultrasonography the kidneys appear completely normal. However, a renal RI ≥ 0.70 in patients with liver cirrhosis and ascites is associated with a 20-30 fold increased likelihood for the development of hepatorenal syndrome.

Tubulointerstitial diseases

Pyelonephritis

Rarely the renal pelvis is filled with echogenic material. As in all acute diseases the kidneys are enlarged. Due to affection of pararenal tissues renal motility is frequently impaired. Chronic pyelonephritis with impaired renal function is frequently associated with reduced renal volume and renal scars. The scars traverse the parenchyma and pyelectasia is frequently seen next to a scar. Renal echogenicity is increased in a patchy pattern and the corticomedullary differentiation is lost. Renal parenchymal width is diminished locally or generalized. Xantogranulomatous pyelonephritis is a variant, which in 2/3 of cases occurs due to pelvic obstruction with infected renal stones (struvite stones = magnesium-ammonium-phosphate). This variant results in a chronic, putrid and fatty inflammation with complete destruction of the renal architecture. The destructed area may be confused with renal tumors or cysts. Renal stones or calcifications can frequently be found in affected areas. Definitive diagnosis relies on a CT-investigation

(Medullary) nephrocalcinosis

Nephrocalcinosis is defined by deposits of calcium and phosphate in renal tubules. Ultrasonography can only depict calcification in the region of the terminal collecting ducts (medullary pyramids). Medullary pyramids normally have lower echogenicity in comparison to the surrounding parenchyma and develop a hyper echoic ring or become completely hyper echoic sometimes even with acoustic shadowing. Classification in a 3 stage system has been suggested. Stage 1 is depicted by a barely visible hyper echoic ring, stage 2 by a complete ring; both stages do not show acoustic shadowing. Stage 3 then is a complete ring form or a completely hyper echoic pyramid with acoustic shadowing. Nephrocalcinosis is not specific to one disease. All diseases that can cause precipitation of minerals in the urinary collecting tract may cause nephrocalcinosis. Hypercalcemia or hypercalciuria but also pathological intrarenal structures serving as crystallization points may cause nephrocalcinosis. Diseases associated with nephrocalcinosis are shown in table 1.

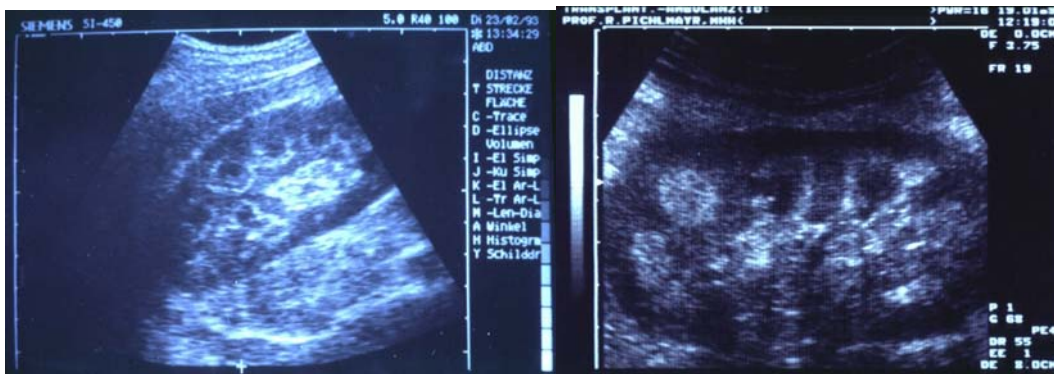


Fig. 8 Nephrocalcinosis with complete ring formation

Medullary sponge kidney: This is a congenital disease causing widening of collecting ducts in the area of the pyramids. Urolithiasis with urinary tract obstruction and nephrocalcinosis in more than 50% of affected patients are frequent findings. The medullary cysts usually are not seen by ultrasonography due to their small size (1-7 mm). Nephrocalcinosis however is easily seen. The kidneys are of normal size or slightly enlarged and show increased echogenicity. The gold standard for diagnosis is i.v. urography.

Table 1 Diseases associated with nephrocalcinosis

| | Frequency | Definitive diagnosis |
|---|-----------|--|
| Primary (metastatic) nephrocalcinosis | | Hypercalcemia, hypercalciuria |
| Hyperparathyroidism, primary or secondary | 30-40% | Lab: intact parathyroid hormone, calcium, phosphorus |
| Sarcoidosis | 2 % | |
| Malign tumors / bone metastases | 2 % | |
| Paraneoplastic increased PTH-related protein production | | |
| Increased bone turnover: M.Paget, progressive osteoporosis, Hyperthyroidism | 2 % | |
| Vitamin D intoxication | 2% | Anamnesis |
| Milk alkali syndrome | | Anamnesis |
| Idiopathic hypercalciuria | 6 % | Urinary calcium |
| | | |
| Secondary nephrocalcinosis (without hypercalcemia) | | |
| Renal tubular acidosis | 20% | astrup, normal anion-gap, urinary-pH, hypophosphatemia, glucosuria |
| Medullary sponge kidney | 10-15% | i.v. urogramm |
| Papillary necrosis | 2-3 % | |
| Primary hyperoxaluria | 4% | Oxalic acid in urine and serum |
| End stage of renoparenchymatous disease | 5% | |

Papillary necrosis, papillary calcification

Like nephrocalcinosis an unspecific sign. Calcification usually with acoustic shadowing due to necrotic tissue can be found at the tip of medullary pyramids. Possible underlying diseases are: analgesic nephropathy, urinary tract infection, chronic urinary tract obstruction, renal vein thrombosis, prerenal renal failure, sickle cell anemia and hemophilia. The initial sign is swelling of the medullary pyramid which is rarely detected with ultrasonography. The consecutive papillary calcification or the detachment of the papilla with consecutive urinary tract obstruction allows the ultrasonographic diagnosis. The detached papillae can be found as echogenic structures with or without acoustic shadowing in the renal pelvis. The differential diagnosis includes blood clots, urothelial carcinoma or a sponge ball. I.v. urography or CT allows a more definitive diagnosis.

Analgesic nephropathy

The kidneys are smaller than expected from the degree of renal insufficiency and show scars as in chronic pyelonephritis. Symmetric calcifications at the cortico-medullary border, due to papillary necrosis are characteristic for the disease. Initially papillary necrosis can be seen as a hyper echoic area at the tip of a pyramid; later on the necrotic papillae calcify and then show acoustic shadowing. Urinary tract obstruction with hydronephrosis can occur as a complication of a detached papilla.

Table 2. Classification of renoparenchymatous disease

| Unilateral small kidney | Additional ultrasonographic criteria | Definitive diagnosis |
|---|---|--|
| Renal hypoplasia | Normal echogenicity of the affected kidney, contralateral kidney enlarged. Normal perfusion in Color Doppler sonography | i.v. urography, CT |
| Renal agenesis | Contralateral kidney enlarged | CT |
| Renal irradiation | Shrinking of renal parenchyma only within the field of radiation | Anamnesis |
| Pyelonephritis | Renal scarring, possibly bilateral | Anamnesis |
| Renal artery stenosis | Normal echogenicity, small parenchyma, prolonged acceleration time in segmental renal arteries and flow acceleration in the renal artery | Spiral-CT, Angio-MRT, ia.-DSA |
| Tuberculosis | Strictures due to scars, calcifications, tumor-like lesions, perirenal abscesses, sometimes urinary bladder wall edema, Terminal stage: autonephrectomy with small completely calcified kidney, DD oxalosis but here kidneys usually of larger size | Clinic: dysuria, nycturia, however 20% asymptomatic, Acid-proof rods in urine „sterile“ leukocyturia and hematuria |
| Bilateral small kidney | | |
| Terminal stage of almost all renal diseases | | |
| Chronic glomerulonephritis | Smooth surface, hyper echoic parenchyma | Urinary finding |
| Chronic pyelonephritis | Scars, calyectasia, can be confused with renal tumor in xanthogranulomatous pyelonephritis | CT |
| Analgesic nephropathy | Similar to chronic pyelonephritis plus (symmetrical) papillary calcifications. Medullary pyramids frequently show increased echogenicity. In contrast to nephrocalcinosis acoustic shadowing is rarely seen | Anamnesis |
| Diabetic nephropathy | Like chronic glomerulonephritis but relatively large kidneys in comparison to the degree of renal impairment. Renal resistance index frequently > 0.80 | Diabetes duration at least 10 years Additional associated diseases (diabetic retinopathy, polyneuropathy...) |

| | | |
|-----------------------------------|--|--|
| | | Urine – frequently microalbuminuria and proteinuria |
| Bilateral renal artery stenosis | See unilateral renal artery stenosis | |
| | | |
| Bilateral enlarged kidneys | | |
| Acute urinary tract obstruction | If functionally relevant RI > 0.7-0.75 or RI difference ≥ 0.06 Always look for the cause of obstruction. In unilateral disease look prevesical (stone, tumor...) in bilateral disease look postvesical (prostate, iatrogenic...) Hydronephrosis \geq III/IV no complete reversibility possible | i.v. urography CT |
| Double kidney | Complete parenchymal bridge, 2 pelvices, 2 ureters (only visible if urinary tract obstruction is present) | i.v. urography when symptomatic |
| Acute pyelonephritis | Variable parenchymal echogenicity, frequently inhomogeneous swelling. Loss of corticomedullary differentiation. In case of abscess formation: area with low or absent echogenicity, sometimes with inclusion of air. Sometimes echogenic material can be found in the pelvic area (pyonephrosis) | Clinic (fever, loin pain) Urinary findings |
| Renal vein thrombosis | In acute thrombosis low parenchymal echogenicity, in chronic disease increased echogenicity. Pendular flow can only be seen in transplant kidneys. In native kidneys RI increases by >0.10 compared to the contralateral unaffected side. Sometimes thrombus visible in B-mode. Color Doppler shows absent flow signals in the renal vein | Digital subtraction phlebography Angio-CT Angio MRT |
| | | |
| Bilateral enlarged kidney | | |
| Almost all acute renal diseases | | Renal biopsy frequently necessary |
| Acute renal failure | In prerenal acute renal failure parenchymal echogenicity and RI frequently normal. In intrarenal ARF echo dense parenchyma and high RI. Normal sized kidneys are also possible, especially in prerenal | Anamnesis, Urinary findings Serology (Lupus, Wegeners granulomatosis, Goodpasture) |

| | | |
|------------------------------|--|--|
| | ARF. Very echogenic kidneys in toxic ARF | Urinary sodium before application of diuretics differentiates between pre- and intrarenal causes Renal biopsy frequently necessary |
| Acute glomerulonephritis | In most cases increased echogenicity but sometimes also decreased echogenicity and wide parenchyma. Enlarged medullary pyramids with low echogenicity. Ultrasonography cannot distinguish between the different glomerulonephritic diseases. A very dense parenchyma and high RI (>0.8) are bad prognostic signs ⁹ | Anamnesis (creatinine course) Urinary finding (hematuria, proteinuria) CXR (Wegeners granulomatosis /Goodpasture) Serology: Lupus nephritis, Wegeners granulomatosis, Goodpasture Renal biopsy |
| Acute interstitial nephritis | Frequently echogenic parenchyma | Drug history (NSAID, penicillin, rifampicin...) Urinary sediment Eosinophilia Renal biopsy |
| Urinary tract obstruction | Usually postvesical obstruction (prostate) or Ormond's disease with ureteral entrapment | |
| Diabetic nephropathy | When renal function is normal kidneys are frequently enlarged. Echogenicity is initially normal and increases with decreasing renal function. Increased intrarenal resistance index > 0.80 is a bad prognostic sign ⁹ | History of longstanding diabetes Diabetic retinopathy or other diseases associated with diabetes If anamnesis remains unclear or proteinuria worsens rapidly renal biopsy may be an option |
| Amyloidosis | Very large kidneys with very high echogenicity Resistance Index frequently > 0.80 | Urinary protein and Bence-Jones Protein, bone marrow biopsy to exclude myeloma, Deep rectal biopsy or abdominal fat aspiration to look for amyloid. Renal biopsy possible |

| | | |
|--------------------------|---|--|
| Adult polycystic disease | Diagnosis can be made with ultrasound. At least two cysts in one kidney at age 14-30 in the presence of a positive family history is definitive proof | Family history, polycystin-Gen 1 and 2 |
| Pregnancy | Normal echogenicity, thickened parenchyma, sometimes hydronephrosis (right > left). Renal size returns to normal 3 months after pregnancy | |

Pearls for practice

1. Special preparation of a patient for an ultrasound examination is not necessary.
2. New ultrasound methods like harmonic imaging improve kidney visualization
3. Normal renal size can best be estimated as renal volume (ml), which should be twice body weight (kg)
4. Renoparenchymatous disease can be detected with high sensitivity by increased echogenicity. This finding however is unspecific and does not allow classification of renal disease.
5. Nephrocalcinosis with echo dense medullary pyramids is easily detectable by ultrasonography. However, a multitude of diseases can cause nephrocalcinosis.
6. Ultrasonography will only lead to definitive diagnosis when the information from a patient's anamnesis and from B-mode, Color and Doppler-sonography are pooled.

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Ultrasonography of the kidney and the renal vessels

Part II: Focal lesions and Color Doppler Ultrasonography

Abstract

Whereas the diagnostic accuracy of ultrasonography is limited in renoparenchymatous disease it has become the method of choice in the detection and characterization of focal renal lesions. Even small renal cysts can be detected reliably and can be differentiated from malignant lesions according to fixed criteria. In the presence of a positive family history ultrasonography allows the definitive diagnosis of autosomal dominant polycystic kidney disease. Benign angiomyolipomas can be detected and differentiated from malignant renal lesions. However, small malignant lesions (< 2 cm) may be missed by ultrasonography.

Color Doppler sonography has become a standard method to screen for the presence of renal artery stenosis because it allows a fast, non-invasive and cost effective diagnosis. In experienced hands the diagnosis can be made with a sensitivity and specificity approaching 98%. However, color Doppler sonography is not only useful in vascular processes. A raised renal resistance index allows the differentiation of urodynamically significant from non-significant hydronephrosis. Furthermore the renal resistance index can be used as a reliable prognostic marker for renal survival. This could be shown in patients with a variety of renal diseases, in patients with renal artery stenosis and in renal transplant patients. The prognostic accuracy has to be tested in further clinical studies.

Introduction

This second part deals with renal focal lesions. Ultrasonography is the standard method for detection of renal cysts, usually a benign finding. Also for detection of renal stones in patients with renal colic or hydronephrosis ultrasonography has become the most frequently used first method due to its ready availability. Post renal failure can usually be easily differentiated from pre- or intrarenal acute renal failure. Renal tumors, from a certain size upwards are also readily detectable. Ultrasonography will detect most asymptomatic malign lesions during screening investigations. Definitive exclusion of malign lesions – e.g. to clarify the cause of macrohematuria or a pathological urine cytology – is not possible with ultrasonography. Lesions smaller than 2 cm will frequently be missed because they cannot be delimited from the surrounding parenchyma.

Color Doppler sonography is of value not only for diagnosis of renal artery stenosis. Color Doppler will give additional answers in almost all kinds of kidney lesions. Enlarged kidneys with increased resistance index value in a diabetic patient suggest a diagnosis of diabetic nephropathy. An echo-free lesion in the kidney showing perfusion in Color mode most certainly is not a benign cyst. Hydronephrosis in the presence of unilaterally increased resistance index values – even if hardly noticeable – suggest urodynamic relevance and should prompt quick referral to an urologist.

Ultrasonography today is an established method for the initial evaluation of kidneys. The ready availability of this method allows rapid diagnosis and therapeutic decisions, which is of extreme importance to keep in hospital time low.

Pearls for practice

1. Cysts Bosniak III and higher (irregular or thickened septa, thickened cyst wall, calcifications, solid intracystic tumors) should be evaluated by an additional method (CT,
2. Patients with terminal renal failure and renal cysts should have an ultrasound investigation once per year to exclude malign cyst transformation
3. Renal lesions with a differential diagnosis of cyst or malign tumor should be investigated by Color sonography. Perfusion in cystic lesions excludes the presence of a normal cyst.
4. When looking for renal stones a transducer with a high frequency (4-7 MHz) should be chosen, because this facilitates detection of smaller stones
5. When hydronephrosis has been detected not only the degree of pelvic dilatation should be evaluated but also parenchyma thickness and renal resistance index. This may allow distinction between urodynamically relevant and irrelevant hydronephrosis

Focal findings

Renal cysts

Most frequently observed renal lesion. The prevalence increases with age and reaches 20% at age 50. Autopsy studies found cysts in as many as 50%. Cysts are observed twice as frequent in men¹.

Characteristic cyst criteria

1. Relative distal enhancement
2. Smooth surface and round or oval shape
3. Anechoic
4. Enhanced proximal and distal wall
5. No measurable wall thickness

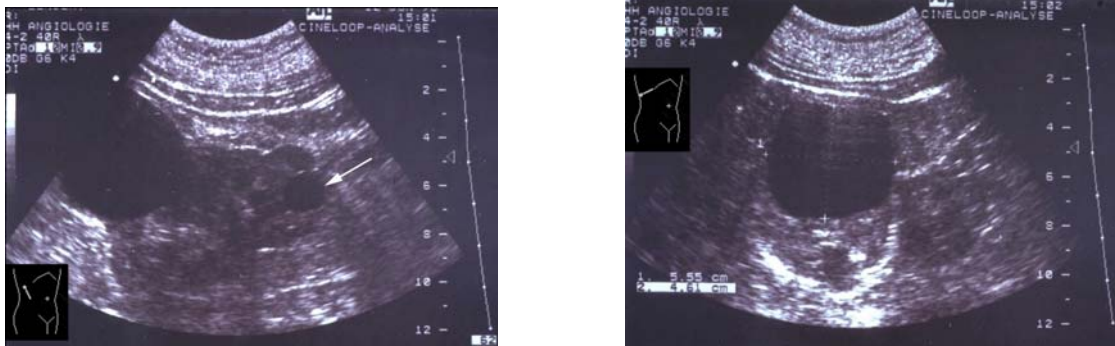


Figure 1. Uncomplicated renal cyst (Bosniak I) in a longitudinal and oblique view. A further cyst (→) is located at the lower pole

Cysts are sub classified as pararenal (perirenal and sub capsular), cortical and parapelveine. The association of a pararenal cyst to the kidney can be proven by the identical respiratory motility. Parapelvic cysts frequently have an oval shape and can be confused with fluid filled calyces. Careful investigation however will show that no enlarged renal pelvis is present.

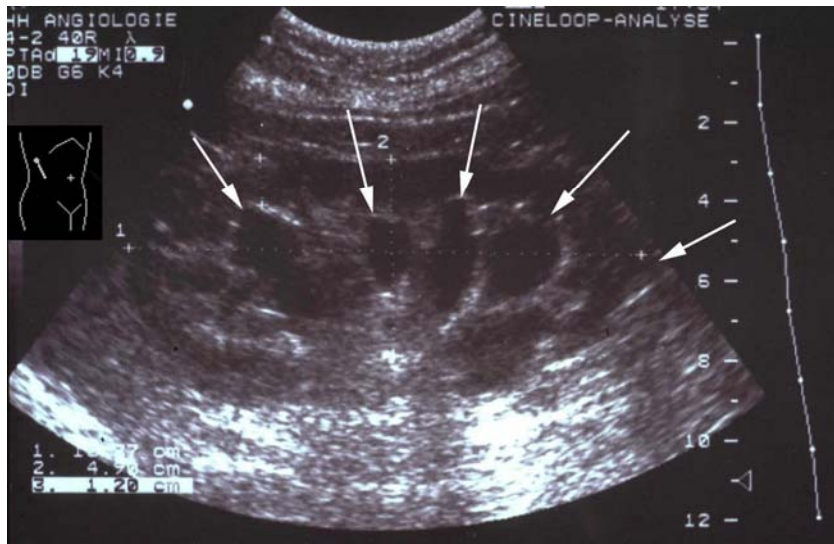


Figure 2: Parapelvic cysts (arrows)

Cysts fulfilling all of the above mentioned criteria are benign and do not need follow up investigations (Bosniak I). When cysts do not fulfill these criteria they are called complicated cysts. These cysts are categorized according to the Bosniak CT morphologic criteria²

Table 1. Classification of cysts according to the Bosniak criteria

| | |
|-------------|---|
| Bosniak I | All criteria mentioned above are fulfilled |
| Bosniak II | Thin septae with a wall thickness of ≤ 1 mm Minor calcifications Echogenic cyst content |
| Bosniak III | Irregular septae or septal thickness > 1 mm or multiple septae Irregular or large calcifications |
| Bosniak IV | Irregularly thickened septae or cyst wall Solid tumor within a cyst |

Stages III and higher are suspicious for malign transformation. If malignancy is suspected even grade II cysts should be evaluated with computed tomography. A frequent reason for echogenic cyst content is spontaneous – frequently painful - cyst bleeding. Abscess formation can also present as echogenic cysts. Sometimes air artifacts can be seen in such abscesses.

Multiple cyst formation can occur in some congenital diseases. The most frequent such disease is **autosomal dominant adult polycystic kidney disease (APKD)**. Sonographic criteria for diagnosis are dependant on age³. The diagnosis is proven in patients < 30 years of age with a positive family history and at least 2 unilateral or bilateral cysts. In patients between age 30 and 59 at least 2 cysts are required per kidney for a definitive diagnosis. Patients older than 60 require 4 or more cysts per kidney³. Cysts in APKD are found both in the parenchyma and sinus area. They touch and deform each other so typical cyst criteria are not always fulfilled. The multitude of cysts and the greatly enlarged kidney volume (> 10 kg) usually allows easy diagnosis.

In up to 50% of patients liver cysts can also be found and rarely also pancreatic or splenic cysts. Frequently sudden pain over a cystic kidney is caused by cyst hemorrhage. If these cysts invade the renal pelvis macrohematuria may occur. Hemorrhaged cysts are found by looking for echogenic cysts at the area of maximal pain. In autosomal recessive cystic kidney

disease, which frequently leads to dialysis dependence in early childhood the kidneys are greatly enlarged, very echogenic and the corticomedullary border is not well defined. Only occasionally cysts can be found.

3 to 5 years after the start of dialysis treatment secondary formation of multiple cysts measuring 0.5 – 3 cm frequently occurs. These cysts can be found in the parenchyma as well as in the renal sinus. Cyst formation is not as frequent in terminal diabetic nephropathy. Finding these secondary cysts sometimes is the only way to find the severely shrunken kidneys in patients with end stage renal disease. Because secondary cysts have the potential for malign transformation regular yearly ultrasonographical investigation should be performed.

Echinococcal cysts rarely occur solely within the kidney (3-4%). A cyst with a thickened wall, septae, echogenic contents and sometimes calcification should arouse suspicion. Also the area of the liver and the bladder should be investigated for such cysts. Because of the danger of metastatic spread biopsy of those cysts is contraindicated. Normal echinococcal serology can make the diagnosis less likely. Therapy of such cysts is by complete surgical resection under antibiotic cover.

Scars

Defects in the parenchyma which should not be confused with reniculi. Scars can occur due to pyelonephritis or due to a renal infarction (atherosclerotic, embolic). Typical pyelonephritic scars traverse the parenchyma and reach the renal sinus and are usually irregular formed and broad. Vascular scars frequently have a triangular shape and do not reach the pelvic area. New scars are of low echogenicity but echogenicity is increased in older scars.

Abscess

Abscesses frequently look like hypo echoic tumors or cysts with a thickened wall. The tumor contains echogenic material and septae are frequently found. Color Doppler frequently shows increased perfusion of the wall but no central perfusion which distinguishes abscesses from malign tumors which have perfusion within the borders even if the central section has become necrotic. The clinical picture is characterized by intermittent fever and high markers of inflammation. Diagnosis is by aspiration of material and drainage may be considered as a therapeutic measure. Contrast-CT can be performed to find other abscesses and to differentiate abscesses from malign tumors.

Malign tumors

Sensitivity of ultrasonography to detect renal tumors is dependant on size. Tumors ≥ 3 cm will be found in almost 100% of investigations because they almost always change kidney contour. Tumors of < 2 cm will be found in only 50%. Angiomyolipoma, due to their high echogenicity, will be found in almost all cases.

Angiomyolipoma is the most prevalent benign renal tumor. Autopsy studies show it to occur in 0,3%^{4,5}. Ultrasonography detects Angiomyolipoma in 0,25% of patients⁶ and this tumor appears 3 times as frequently in women than in men⁷. The shape is roughly round and the tumor is very echogenic (white tumor).

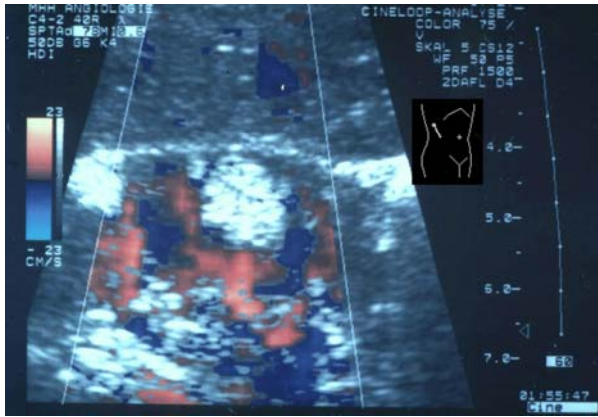


Fig. 3. . Angiomyolipoma. No visible perfusion in color mode

The renal contour is usually not altered by an angiomyolipoma and due to the very low flow rates within the tumor Color Mode will not detect perfusion within an angiomyolipoma. One third of these tumors show acoustic shadowing. If all of these criteria are present no further diagnostic method has to be applied and the tumor should be reinvestigated after 3 months time. If tumor size has not changed at that time the diagnosis is established. Hemorrhaged angiomyolipoma (occurring in angiomyolipoma > 4 cm) can also appear iso- or hypoechoic. If one or more of the above mentioned criteria are not fulfilled, if the tumor is not hyper echoic, is much larger than 1 cm or increases in size a CT or MRT should be performed which allows detection of the increased fat mass of this tumor. Tumors < 1 cm of size, which can be detected by ultrasonography, cannot be investigated further by CT or MRT. Here ultrasonographic follow up is the only possibility. If multiple Angiomyolipoma are found in both kidneys the possibility of Bourneville-Pringle disease should be considered.

Renal cell carcinoma accounts for 80% of all malign renal tumors. Echogenicity within the tumor is frequently increased, but iso- or hypoechoic tumors are also possible. The renal contour is frequently altered by the tumor. Tumors 25 mm and bigger can be detected reliably whereas tumors of size 20-25 mm will be detected in only 80% of cases and even smaller tumors cannot be reliably detected.

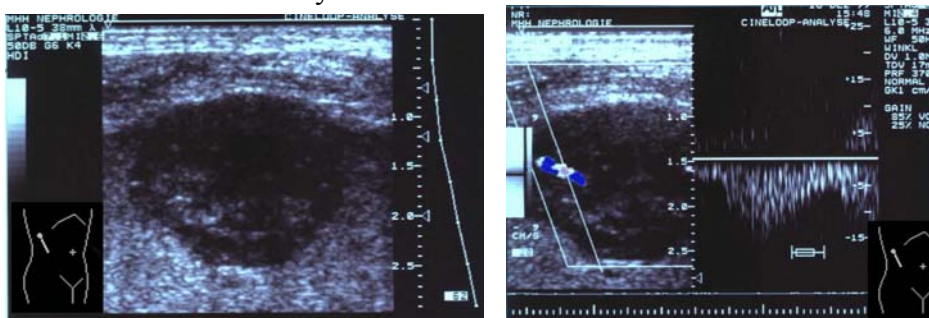


Fig. 4: Hypoechoic cyst like lesion with interior echoes. The picture on the right side shows central perfusion as a sign of malignancy

Color Doppler sonography frequently shows hypervascularisation of malign tumors with decreased RI values. Larger tumors may show central necrosis and can be confused with abscesses or cysts and the necrotic part can be super infected. 5% of all renal cell carcinoma have multilocular cystic parts. In these tumors too, the vital border area shows increased perfusion. Calcification of tumor areas adds to the complexity of presentation. 20% of renal cell carcinoma show invasion of the renal vein and V. cava^{8,9}. If a malign renal tumor is suspected the dedicated lymph node stations (renal hilus, paraaortal, iliac) should be investigated. Lymph node invasion and liver metastasis occur in a third of all cases. A splenic notch (dromedary hump) may be confused with renal cell carcinoma. The normal medullary

pyramids and the normal perfusion pattern help to differentiate this benign change from a malign lesion. Definitive diagnosis can be made with either biopsy or – more frequently – by surgical exploration and rapid section histology and total or partial nephrectomy if necessary. To enhance the diagnostic accuracy and to exclude distal metastasis ultrasonographic detection of a tumor should prompt a CT investigation. **Oncocytoma** (7% of renal malign tumors) and **renal sarcoma** (1%) cannot be distinguished from renal cell carcinoma by either ultrasonography or CT or MRT. Large oncocytoma (> 3 cm) may show a characteristic central star shaped scar ¹⁰.

Intrarenal **urothelial carcinoma** (about 5-9% of malign renal tumors) will not be found by ultrasonography unless the pelvic lumen is obstructed by a larger mass. The diagnosis of urothelial carcinoma can be suspected, when the tumor is located within the renal pelvis. In contrast to the renal sinus urothelial carcinomas appear hypoechoic. Urothelial carcinoma is found within the bladder 50 times more frequently than in the kidney. The diagnosis is verified by CT or MRT.

Rare renal neoplasias

Renal metastases occur in 5-8% of primary lung, breast and renal cell carcinoma (ipsi- and contralateral), less frequently in the course of liver, bone or adrenal malignancy.

Ultrasonographic appearance is that of one or more hypoechoic lesions of the kidney. The primary tumor will be known in most cases. Juxtaglomerular renal tumors are small, hyper echoic and may induce hypertension due to increased renin production. Leiomyomata usually have their origin in the renal capsule. Hemangioma or lipoma which are tumors frequently found in the liver rarely occur in the kidneys.

Lymphoma, due to absent intrarenal lymphoid tissue never originate from the kidney. Computertomographic studies showed, however, that 5% of lymphoma of other origin can metastase via the blood stream or can invade the kidney from the retroperitoneal space ^{11, 12}. The kidney may be focally or diffusely infiltrated. In focal disease one finds very hypoechoic lesions which sometimes even appear cyst like but have very little or no dorsal enhancement. Color Doppler will frequently find intralesional perfusion as the distinguishing feature from cysts. With diffuse infiltration the kidneys appear enlarged, hypoechoic with absent corticomedullary differentiation. The contour is usually preserved by the renal capsule. Lymphoma can affect one or both kidneys.

Renal stones

Renal stones appear as hyper echoic reflexes within the hyper echoic renal sinus. Dorsal shadowing usually attracts attention. The diagnosis is only secured if the circumscribed hyper echoic reflex can be found also in the second dimension. Transducers with high frequencies (4-7 MHz) facilitate detection of smaller stones. A further criterion differentiating a renal stone from a calcified vessel is the proximal widening of a renal calyx. In some stones (those with a rough contour) Color mode may show a shower of Color signals in the vicinity of a stone (twinkling artifact) which can also help to find smaller stones. Ultrasonography has a sensitivity of 60% and a specificity of 90% for the detection of renal stones. CT (without contrast agents) remains the gold standard for diagnosis. Obtruding stones within the ureter are frequently not found. The combination of hydronephrosis, typical clinic and intrarenal stones however is highly suggestive of such a stone.

Urinary tract obstruction

Urinary tract obstruction can usually be easily detected by ultrasonography. Acute obstruction, however, must not lead to immediate dilatation of the renal pelvis. This may take

from a few to 24 hours. Ultrasonographic evaluation of hydronephrosis should include naming the degree of hydronephrosis (see below), the location of the obtruding structure (usually the bladder and more distal parts of the urinary tract in bilateral hydronephrosis) and the cause of obstruction (stone, tumor...). There are many possible causes for urinary tract obstruction and ultrasonography will detect the reason with certainty in only 20% of cases. Table 2 gives the most frequent reasons.

Table 2: Causes of pyelocaliectasia

| |
|---|
| |
| Unilateral pyelocaliectasia |
| Ureteral stone |
| Ureteropelvic stenosis |
| Tumor of the renal pelvis or ureter |
| Adhesions |
| Inflammatory ureteral stenosis |
| Outside ureteral obstruction (tumor, pregnancy, M. Ormond) |
| Inflammatory ostial stenosis |
| Pregnancy (occurs physiologically in 80% of pregnancies; unilateral in 60%, right kidney most frequently affected. Max. sagittal width should be less than 2 cm) |
| Bilateral pyelocaliectasia |
| Carcinoma of the prostate |
| Bladder carcinoma or -adenoma |
| Stenosis of the bladder sphincter |
| Urethral valves or -strictures |
| Meatic stenosis |
| Phimosis |

The degree of hydronephrosis is quantifiable, the grading system of the „Society for Fetal Urology“ being the best established system¹³. The final stage of hydronephrosis which is not included in this system is complete loss of parenchyma, a so called “sacculated kidney”

Table 3 Grades of Pyelocaliectasia according to¹³

| | |
|----------|---|
| Grad 0 | Normal finding |
| Grad I | Minor separation of the central echo reflex (pure pyelectasia) No ectasia of calyces, parenchyma of normal width |
| Grad II | Medium grade separation of the central reflex Major calyces dilated with well defined fornix edges; parenchyma of normal width |
| Grad III | Renal pelvis clearly dilated Major and minor calyces uniformly dilated; parenchyma of normal width |
| Grad IV | Like grade 3 and parenchyma thin |

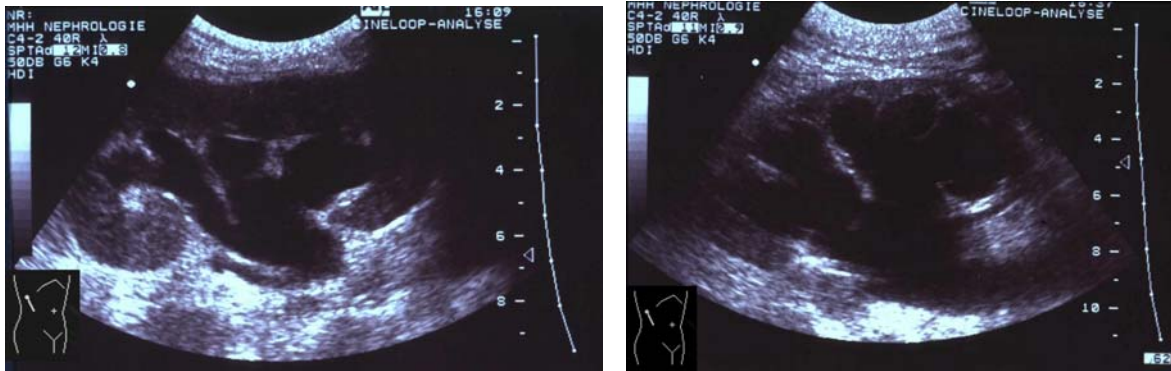


Figure 5. Hydronephrosis grade III (left picture) and IV (thin parenchyma-right picture)

Parapelvic renal cysts or sinus lipomatosis can occasionally be confused with calyectasia. However, the central reflex (renal pelvis) will not be widened in the latter two conditions. Extended renal veins may also be confused with calyectasia, however, Color sonography will show flow within renal veins. Urodynamically relevant hydronephrosis is present for sure in grade IV hydronephrosis. However, lower grades of hydronephrosis may also be relevant and B-mode ultrasonography cannot tell. Measuring of the renal resistance index may be of help here. A RI value ≥ 0.80 or a RI which is 0.07 – 0.10 units higher on the affected than on the unaffected side will provide additional diagnostic information. Renal scintigraphy is the standard method for evaluation of urodynamic relevance of hydronephrosis.

Perirenal findings

Seroma, urinoma, abscesses or hematoma may occur postoperatively. There is no characteristic ultrasonographic sign which clearly distinguishes these four entities. Lymphoceles with seroma formation show septae more frequently than the other diseases and abscess formation with gas forming bacteria may be associated with air entrapment which can be easily detected by the associated ultrasound artifacts (reverberation). If renal function deteriorates acutely or if the liquid structure is painful (urinoma) puncture of the structure can exclude urinoma or abscess formation which require rapid intervention. Large seroma or hematoma can cause secondary urinary tract obstruction. If this is of urodynamic relevance seroma or hematoma should be treated by drainage.

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Resistive index: an ideal test of renovascular disease or ischemic nephropathy?

Corresponding author:

Prof. Dr. med. Jörg Radermacher;
Abteilung Nephrologie
Klinikum Minden
Friedrichstrasse 17
32427 Minden
Tel: 0571/ 801 – 3021; FAX: 0571/ 801 – 3076
E-Mail: Joerg.Radermacher@klinikum-minden.de

Resistive index (RI) (synonym: resistance index) is a simple parameter derived from an ultrasound Doppler spectrum. It describes the percentage reduction of enddiastolic flow (Vmin) in relation to maximal systolic flow (Vmax): $RI = (V_{max} - V_{min}) / V_{max}$. RI can be measured in any vessel. However, for this review only renal derived RI values are considered. Even here a wide variety of RI measurements is possible, depending on the location of intrarenal vessel chosen. In general, the RI value declines, the more distal the measurement is made, i.e. it declines from the distal renal artery to the segmental, interlobar, arcuate and interlobular arteries. Unfortunately intrarenal measurements have never been standardized to a specific intrarenal region.

Furthermore RI is not a specific parameter and can be influenced by intra- and extrarenal factors. So, to give a short answer to the question made in the title: No, RI is certainly not an **ideal** test for either renovascular disease or ischemic nephropathy. RI increases with age, especially in hypertensive patients¹. It is increased in a variety of renal diseases other than ischemic nephropathy like diabetic glomerulosclerosis, haemolytic uremic syndrome, acute intrarenal failure, hepatorenal syndrome, urinary tract obstruction, high-grade reflux, lupus nephritis with high chronicity index, renal vein thrombosis and acute vascular rejection in renal transplant patients¹. Furthermore extrinsic factors like kidney compression and breath hold with the valsalva manoeuvre can cause an increase in RI and extreme bradycardia does too. RI values measured in the kidney are also correlated to extrarenal markers of vascular stiffness like intima-media-thickness and carotid stiffness².

Having said all this, RI, although not an ideal test is certainly useful in the diagnosis of renovascular diseases and in the diagnosis of ischemic nephropathy. Any experienced investigator has little difficulty in excluding most of the above-mentioned diseases and circumstances under which altered RI values can be found.

RI and diagnosis of renovascular disease (renal artery stenosis)

There is general agreement that the absolute value of RI is of little value to diagnose renal artery stenosis. A RI difference of 5% or more between the two kidneys however, suggests renal artery stenosis on the site with the lower RI value³. RI is so called indirect parameter (parvus phenomenon) of renal artery stenosis. Another indirect sign of stenosis which does not depend on the presence of both kidneys is a prolonged acceleration time > 70 msec¹. The definitive diagnosis of renal artery stenosis however does not rely solely on indirect parameters but relies more on direct parameters like an increased intrastenotic flow velocity above 180-200 cm/sec or a renal-aortic ratio of > 3.5 or an intrastenotic/poststenotic ratio > 4¹. Only if direct signs of renal artery stenosis cannot be obtained, one should rely on indirect signs alone. Indirect signs only depict higher degrees of stenosis usually exceeding 60% diameter reduction.

RI and diagnosis of renovascular hypertension and renovascular azotemia

We have shown, that an increased resistance index (> 0.80) measured in renal segmental arteries is associated with a lesser likelihood of therapeutic effect both regarding improvement of blood pressure (i.e. renovascular hypertension) and improvement of renal function (i.e. renovascular azotemia)⁴. These patients are suspected to suffer from ischemic nephropathy in addition to renal artery stenosis. This has been confirmed by some groups however, Zeller et al., in a retrospective study could not confirm this finding⁵. Voiculescu et al. suggested that a very low RI value (< 0.55) may suggest a higher therapeutic efficacy of correction of renal artery stenosis⁶.

RI and ischemic nephropathy

Ischemic nephropathy (i.e. hypertensive nephrosclerosis) is difficult to diagnose on clinical grounds alone. What is usually required is longstanding hypertension preceding the development of proteinuria and renal insufficiency and proven hypertensive end-organ damage in organs apart from the kidney. This can be a hypertensive retinopathy or left ventricular hypertrophy. Kidneys in ischemic nephropathy are usually small, hematuria does not occur and proteinuria is usually < 1 g/day in younger patients. Biopsy studies in very old patients (> 80 years) however, have shown that ischemic nephropathy is the major cause of nephrotic range proteinuria (> 3.5 g/day) in this age group. Renal biopsy may not be the best test to diagnose ischemic nephropathy due to sampling error. The intrarenal vessels frequently involved in this disease are the large and medium sized intrarenal arteries, which are frequently not sampled during biopsy. An additional parameter like an increased resistance index can therefore be of great help to establish the diagnosis of ischemic nephropathy. Derchi et al. found a twofold increased risk of mild renal dysfunction in patients with essential hypertension and RI values ≥ 0.63 after adjustment of RI for age, pulse pressure and LDL-cholesterol². In patients with untreated essential hypertension Pontremoli et al. found an association of early markers of target organ damage (microalbuminuria, IMT) with RI⁷. We found that a RI value > 0.80 is generally associated with advanced and rapidly progressive renal disease¹.

The increase of RI with age, which occurs more in patients with hypertension than in those without¹ can also be seen as a sign of progressive nephrosclerosis. Ischemic nephropathy may be the reason, why renal function declines with age in most patients. This age dependent decline in renal function is so common, that all formulas estimating glomerular filtration rate (Gault-Cockcroft, MDRD) include age as the main denominator. This decline however, should not be considered as natural but should be considered as a sign of renal disease, which is more common in elderly patients just as hypertension is more common in elderly patients. Noone today would say that increased blood pressure in elderly patients is a normal sign of aging and should not be called hypertension. A decline in RI has been shown with ACE inhibitors but not with calcium antagonists⁸ and this may be the reason why ACE inhibitors are nephroprotective and calcium antagonists are not. RI is lower in patients with early nonproteinuric diabetic nephropathy and hyperfiltrating enlarged kidneys, but increases when the kidneys start to shrink and when microalbuminuria occurs⁹. Diabetic nephropathy with impaired renal function is more closely related to renal arterial disease than to glomerular disease. The increased RI value in diabetic disease could also be a sign of renal vascular involvement. ACE inhibitor treatment of normotensive diabetic patients but not of nondiabetic patients causes a decline in RI¹⁰.

In summary a RI-difference between the two kidneys can be a sign of renal artery stenosis on the site with the lower RI value but should not be the only parameter used. Increased resistance index values in nondiabetic, non-hematuric patients with slowly evolving renal dysfunction and hypertension suggest the presence of hypertensive nephrosclerosis.

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