EFSUMB – European Course Book

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Genitourinary ultrasound

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Content

Kidney

Topography

All urogenital system is retroperitoneally located. Kidneys have to be searched next to liver and spleen, close to musculus psoas and the large abdominal vessels. Dorsally of the kidney lies the musculus ileopsoas, ventrally are either liver or spleen, medial and ventral are the large abdominal vessels. The longitudinal axis is pointing from cranial medial to caudal lateral as well as from cranial dorsal to caudal ventral, which is especially important for the

accurate measurement of the renal length. In its short axis, the renal hilus is pointing towards ventral medial. [Fig. 1-4].

Figure 1 Normal position of kidneys and ureters.

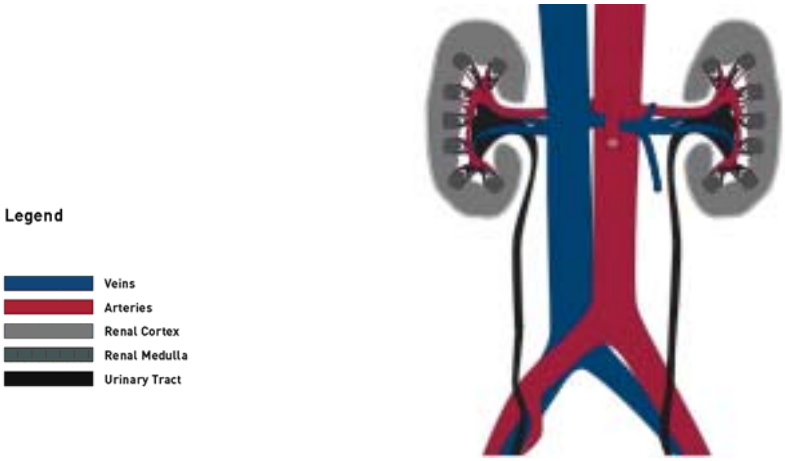


Figure 2 On the sagital cut, the longitudinal axis is pointed from dorsal cranial towards ventral caudal.

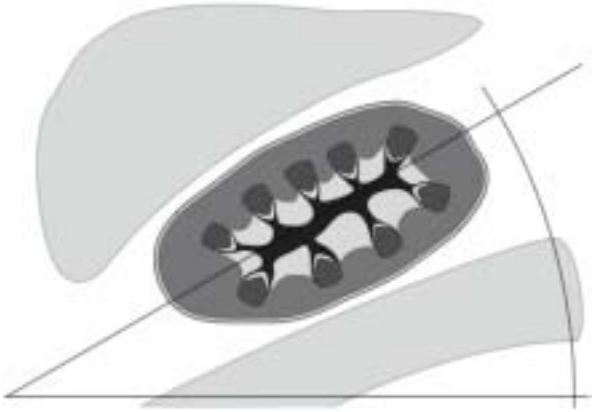


Figure 3 **In the frontal plane, the longitudinal axis is pointed from medial cranial towards lateral caudal.**

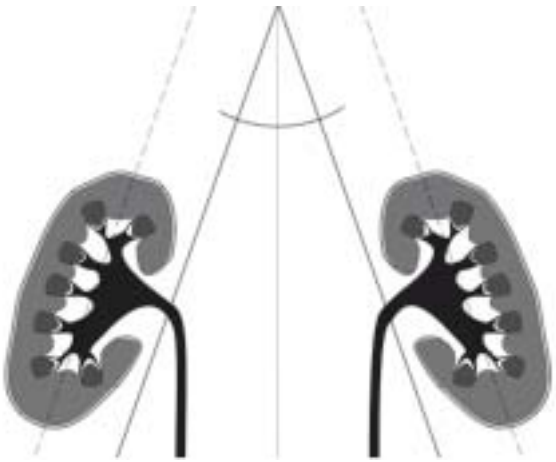
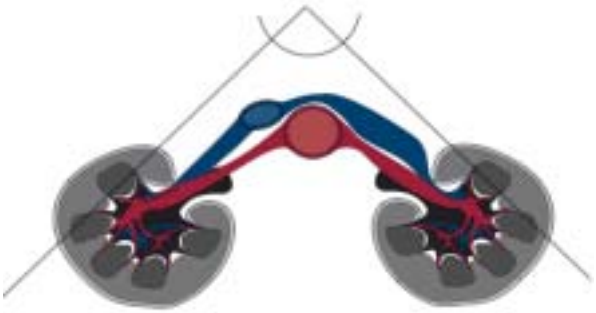


Figure 4 **The short axis is pointed from dorsal lateral towards ventral medial.**



Anatomy

The macroscopic structure of the kidney can be well depicted by the means of sonography. In case of obese patients, the outer kidney capsule will be limiting the perirenal fat body and the inner kidney echogenic capsule will represent a kidney contour. The parenchyma can be differentiated in echogenic renal cortex as well as echopoor mark pyramids, that are 12-15 and point conically towards the limit of the kidney sinus. Between pyramids the kidney cortex reaches up to the limit of the sinus. The basis of the pyramid is being limited by light reflexes of the arteria arcuata. In longitudal axis, the more echogenic and inhomogeneous kidney sinus can be seen ovally in the middle of the parenchyma, in cross section it is being contained by it semi-circular form. [Fig. 5-6].
Renal artery and vein can be observed coming square out of the renal hilus, whereas the right kidney vene is going straight into the Vena cava and the left one first ventrally crosses the

Aorta. The right Arteria renalis is pointing from behind Vena cava towards the renal hilus [Fig. 1,7].
The ureters turn out of the renal hilus directly towards caudal. Later on they proceed along the Musculus psoas and then cross the Iliac vessels to finally reach the ureteral orifices of the bladder retroperitoneally [Fig. 1]. This anatomical course of the ureters is crucially important when searching for ureter stones.
The urinary collecting system can be especially well recognised during urinary congestion. With a patient lying on the belly then, the pyelon which had not been jammed will be easily visible in most cases, and here, the exit of the ureter can be searched for as well [Fig. 8].
The basic functional unit of the kidney is a lobulus [Fig. 9, 10]. It consists out of a mark pyramid with surrounding kidney cortex and corresponds the whole of a rats kidney. In a human kidney, 8-14 of such lobules melt together. Sometimes traces of such mergings can be found (so-called lobulation) [Fig. 11]. Especially during childhood, this is being observed as a norm variation [Fig. 12].

Figure 5 Longitudinal section.

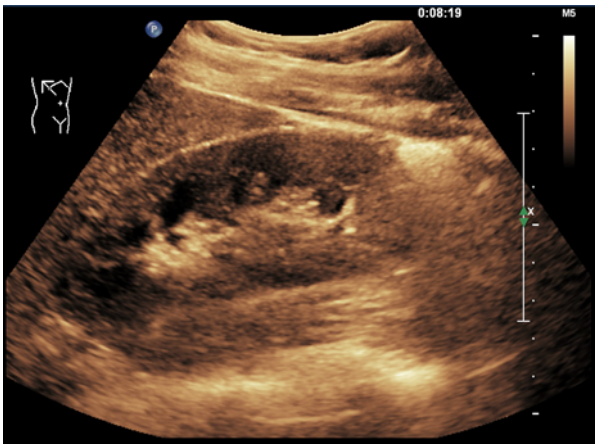


Figure 6 Cross section.

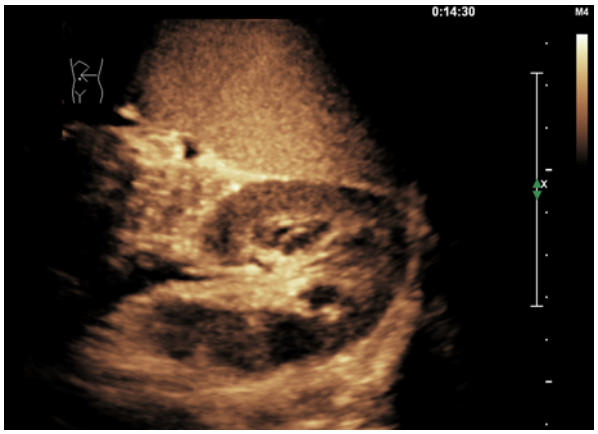


Figure 7 **Renal vessels: RRA (= right renal artery) and LRA (=left renal artery).**

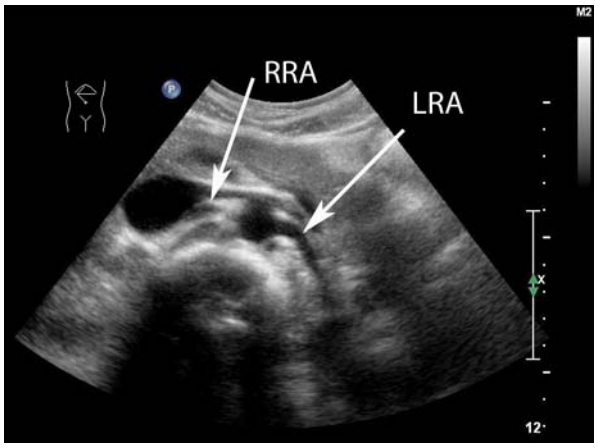


Figure 8 **Ureter outlet in prone position.**

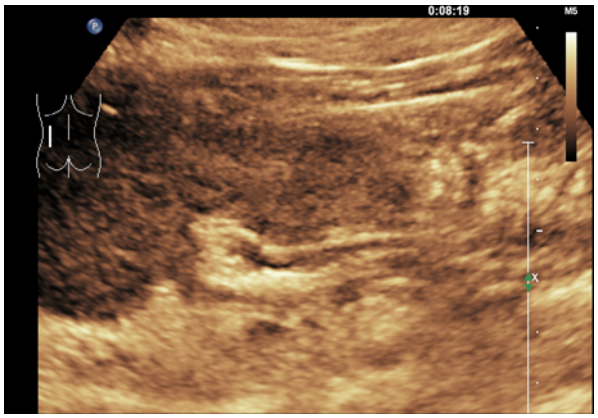


Figure 9 **A renal lobulus includes a single mark pyramid, surrounded by the cortex.**

Figure 10 Two adjacent lobules of a calf kidney. Clearly visible is the lighter kidney cortex and darker pyramids:

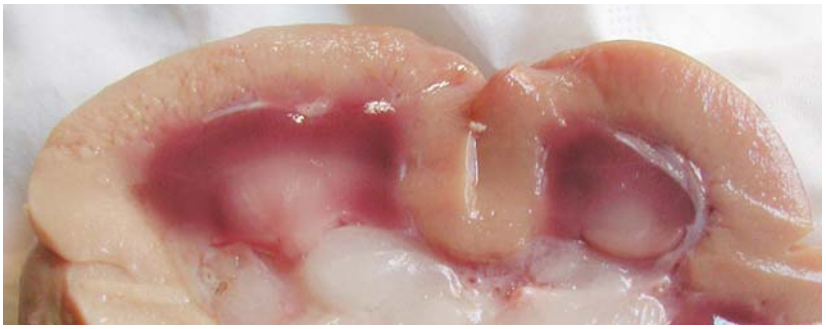
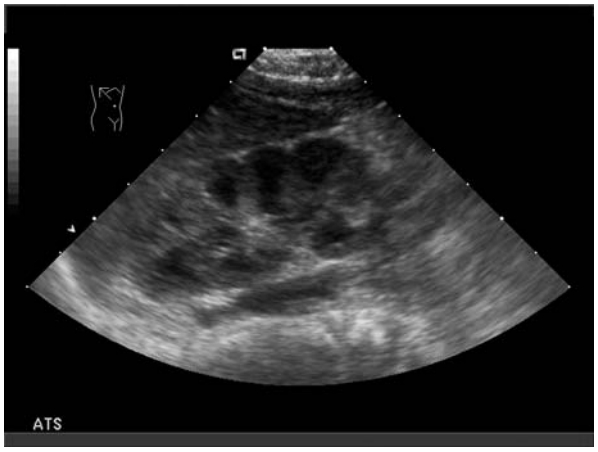


Figure 11 In case of lobulation, places of renal lobules fusion are easily recognizable.

Figure 12 Fetal lobulation in 8 years old child.



Normal findings

Size

Length (A): 9-14cm (measured in longitudinal section)

Width (B): 4-6cm (measured in cross section)
Depth (C): 4-6cm (measured in cross section)

Using the Ellipsoid formula, the kidneys volume is being calculated by these measurements:
Volume ml = A.B.C. $\pi/6$

The normal kidney volume correlates with the body surface:
Normal kidney volume: 100-170ml/1,73m^2 BSA (=body surface area).

For correct measurement of the kidney volumes it is crucial to pay attention to both longitudal and cross axis of the kidney [(11;14;45)]. Kidneys with a volume > 200ml/1,73m^2 BSA count as enlarged, kidneys with a volume < 80ml/1,73m^2 BSA count as reduced [(45)].

Parenchymal and cortical thickness

The parenchymal thickness is being measured from the tip of the mark pyramid unto the surface of the kidney [(45)].

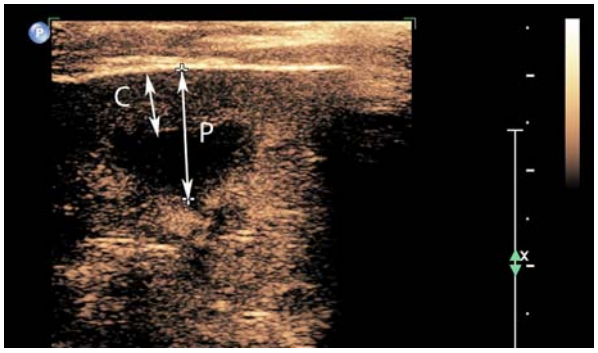
The normal parenchymal thickness consists of 14-18mm.

The parenchymal thickness can be used as a parameter of course, but the measurement should always be carried out on the same place, at the same papilla [Fig. 13]. This is particularly important while monitoring a transplanted kidney, but should be also being taken into account while controlling the process of chronically diffuse diseases of the parenchyma.
The cortical thickness is being measured from the border of kidney mark to kidney cortex surface [Fig. 13].

The normal cortical thickness consists of 8-10mm.

Narrowings can be found during chronical diseases of the parenchyma with kidney insufficiency. Due to this, they correlate with the degree of the kidney's insufficiency [(1,4)].

Figure 13 Measurement of parenchymal (P) and cortical (C) thickness.



The vascular supply of the kidneys is divided into five segments: They are called upper pole, lower pole, upper anterior, lower anterior and posterior segment. Only in 60% all five segments originate from one single renal artery. 8% show an artery originating directly from the Aorta for the apical segment, 6% show an artery for the lower segment, in 5% exists a separation of upper and lower pole and/or other segment arteries. Segment arteries divide themselves shortly before entering the kidney parenchyma. First, they give rise to the interlobar arteries, later branch into the arch arteries. Further diversions of the arch arteries

consist on one hand of the vasa recta leading into the mark pyramids, on the other hand of the interlobular arteries, leading into the kidney cortex.

Examination technique

In the beginning, the patient is being examined while lying on the back. The longitudinal axis is being searched for in the so-called edge-cut, meaning a section which runs from dorsal cranial to ventral caudal as well as from medial cranial to caudal lateral. The kidney is first being measured in longitudinal cuts and is being subsequently examined in the short axis or in cross cuts. The ribs can sometimes be in the way of showing a clean cross section. In such a case, it is recommended to find a space between the ribs and to let the patient breathe deeply. Thereby, the whole kidney can be examined properly and in detail. The kidney sinus is being examined most easily while patients lie prone, which is valid for both children and adults. This position proves in most cases successful to show both the renal pelvis and the outlet of the ureter. Sometimes the kidneys are positioned quite high, the left kidney can be directly subphrenical. In such a case, the examination in a standing position proves most sensible. This can also lead to the observation of a floating kidney (decrease of the kidney of > 5cm while standing). It is important to observe the respiratory displaceability of the kidney and to compare it with the respiratory displaceability of the liver (and the spleen on the left side) and the musculus psoas. This way, single focal space demands can be distinguished from each other, eg cysts in kidney or spleen. A lack of displaceability in comparison with the musculus psoas allude towards a paranephritical abscess or the kidney infiltration by a retroperitoneal tumor.
For an assessment of renal perfusion, a spectral analysis of the kidney parenchymal artery is being derivated while patients lie prone [Fig. 14]. Such spectral curves are evaluated from arteries, which are just about to dip into the kidney parenchyma. The renal arteries and venes however, are being assessed while patients lie on their back. Outlets of the renal arteries can be observed in longitudinal axis [Fig. 15-16] as well as in cross section [Fig. 8]. In the cross section, one tries to show the entire lenght of the arteries in colour duplex ultrasonography (CDUS). The spectral analysis can be derivated from cross cuts as well as from longitudinal cuts.

Figure 14 Spectral curves from the interlobar artery (representing renal parenchyma).

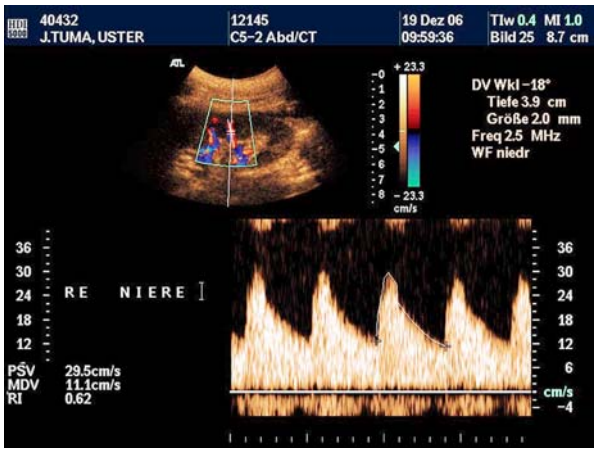


Figure 15 Longitudinal cut for renal artery (RA) outlets.

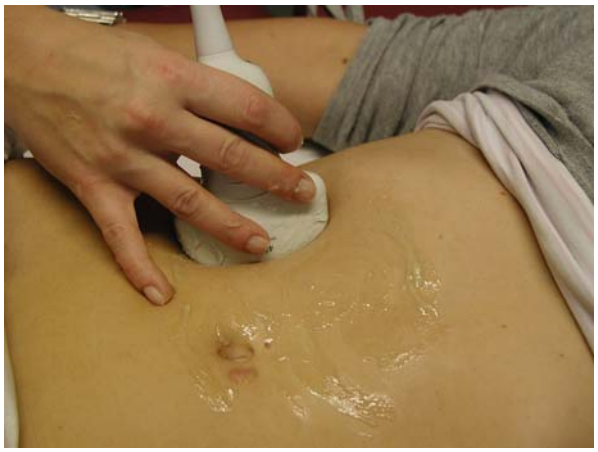
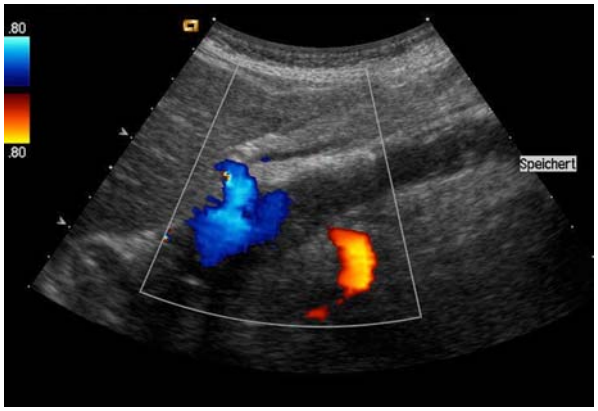


Figure 16 Longitudinal cut for renal artery (RA) outlets. Left RA dorsal, right ventral.



Sonographic assessment criteria

Deviations from the normal shape of the kidney can be used individually or in combination with each other to carry out an analysis of pathological states. Following criteria are being taken into account in the course of this:

- position and form
- size
- contour
- echo pattern
- architecture
- perfusion

Position and shape

Due to its complex genesis, kidneys show many norm variations. On one hand, there are ectopic kidneys with abnormal localisations (eg pelvis kidney) or an anomaly of rotation with a ventrally pointed kidney pelvis [Fig. 17]. A changed form of the kidney can show in various fusion anomalies (eg, horseshoe kidney, cake kidney etc.).

Figure 17Normal and malrotated right kidney (right in the image) in short axis.

Size

During acute diseases of the kidney (f.ex. acute Glomerulonephritis, acute Pyelonephritis) the main observation is in general an enlargement of the kidney's size (length > (13) - 14cm, volume > 200ml/1,73m^2 BSA). However, during chronic diseases (f.ex. chronic Glomerulonephritis, Pyelonephritis or others) the kidney's size shows diminution (length < 9 cm, volume < 80ml/1,73m^2 BSA). Cortical thickness correlates with the degree of kidney's insufficiency

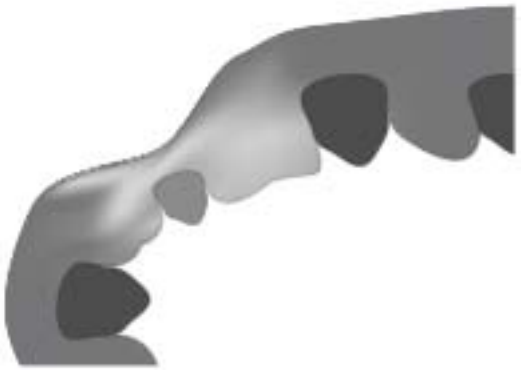
Contour

During the norm variation of lobulation [Fig. 11], the usually smooth contour is characterisied by a wavy outline with fine drafts. These are the traces of a fusion of single lobuli. Lobulations occur often with small children and babies, more rarely with adults and sometimes with adult patients that suffer from a chronic kidney disease. Somentimes between individual lobules junctional fusion defects are observed [Fig. 18].
Similar to junctional fusion defects and lobulations, vascular scars are located between the mark pyramids. Vascular scars are cuneiform and mostly acute angle shaped defects of the countour [Fig. 19]. In contrast, pyelonephritic scars show a flat and concave outline. In the epicenter of the scar lies the mark pyramid. This is being caused by processes of shrinkage in single renal lobuli [Fig. 20].

Figure 18 Junctional fusion defect.

Figure 19 Vascular scar.

Figure 20 Pyelonephritic scar.



Echo pattern

The echo pattern of the normal kidney is fine-grained and homogeneous. In adults, the kidney cortex shows slightly less echo than liver and spleen, the mark pyramids are usually easily recognizable and are very hypoechoic. The kidney cortex is complexly built, consisting of interlobular arteries, glomerula and convoluted tubules, so that it shows a lot of ultrasound interface [Fig. 21]. With an increase of interstitial water (edema, inflammation, congestion), amount of interface arise (similar to liver hemangioma) and therefore, the kidney cortex seems richer in echo [Fig. 22]. Only when reaching a certain amount of water, a decreasement of echo in the cortex can be observed [Fig. 23]. It can be observed in acute right heart failure and in severe pyelonephritis.

In contrast to renal cortex, the medullary pyramids contain parallely progressing tubular structures, which hardly influence the sonographic behavior with changes in the interstitial water. Thus, there are many chronic as well as acute diseases of the kidney parenchyma, which are characterized by a more intensified echo in the cortex of the kidney and easily recognizable mark pyramids.

Another modification of the echo pattern can show as an enriched echo pattern in the mark pyramids, which is in most cases caused by storages of either calcium or uric acid [Fig. 24].

Figure 21 **Renal vessels are filled with red Silastic®, arcuate and interlobular arteries and glomeruli are visible. A micro-punctured tubule (with convoluted tubules, Henle loop and collecting duct) is filled with white Silastic®.**

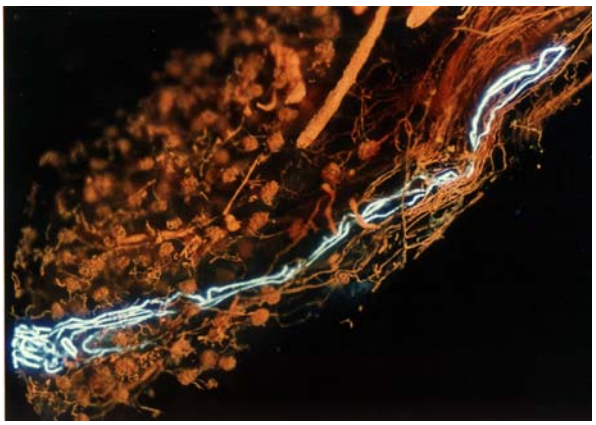


Figure 22 **Large kidney with hyperechoic cortex.**

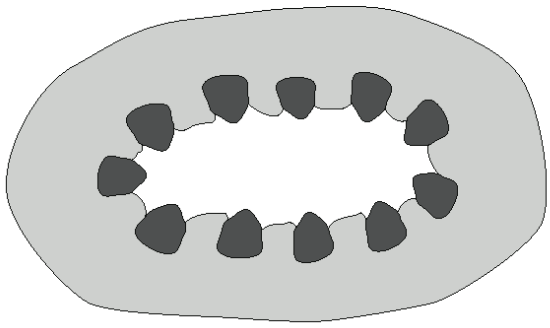


Figure 23 Large kidney with hypoechoic cortex.

Figure 24 Kidney with hyperechoic mark pyramids.

Architecture

The architecture is defined by the outer structure of the kidney in all its complexity. Deviations of the normal architecture form a set of modifications, which can't be described by the already mentioned criteria. In cases of polycystic kidney disease (PCKD) or advanced chronic pyelonephritis, a generalized destruction of the normal architecture is usually being observed. On the basis of destroyed architecture, it becomes possible to recognize neoplastic modifications.

Perfusion

The evaluation of the kidney perfusion is an important sonographic criterion. It can be assessed by CDUS (=color duplex ultrasonography) [Fig. 14], PWDS (=power doppler sonography), spectral analysis and CEUS (= contrast-enhanced ultrasonography). PWDS serves for a rough estimation of the overall perfusion in the kidney parenchyma [Fig. 25]. It should be spread uniformly within the parenchyma. A more subtle assessement is enabled by CEUS, which is important for questions considering renal infarction [(5;8;15;33)] or acute

pyelonephritic changes [Fig. 26a,b]. In accordance with the settings of Pulse Repetition Frequency (PRF) values, not only a representation of renal arteries becomes possible, but also a depiction of its branches - the segmental, interlobar, arcuate and interlobular arteries – succeeds [Fig. 14].
A quantitative evaluation of the kidney parenchyma perfusion is enabled by a spectral analysis of the interlobar arteries [(23;24)]. Maximum systolic velocity V_{max} as well as minimum end-diastolic velocity V_{min} are being detected and the resistive index RI (Vmax - Vmin/ Vmax) is being calculated. These values play an important role in depicting routine controls of transplanted kidneys and also for the evaluation of diffuse renal parenchymatous disease. Normal RI values lie between 0,55 – 0,75 and are age-dependent: For example a patient of 80 years, RI 0,75 is still normal, whereas with a 30 year old patient, RI 0,70 is already too high.

Figure 25 PWDS (power doppler sonography).

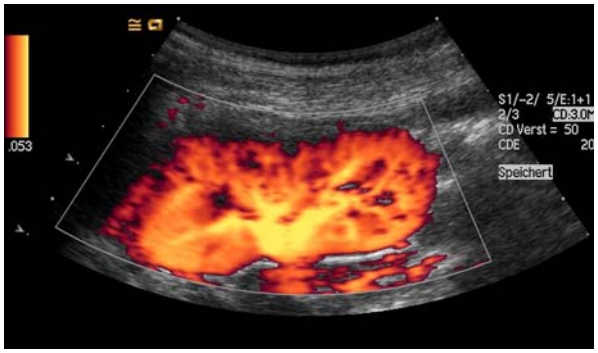
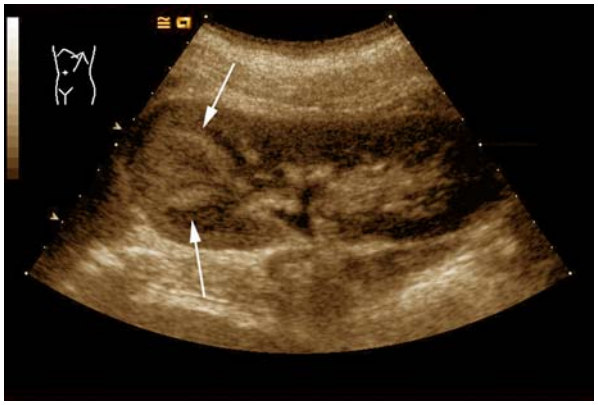
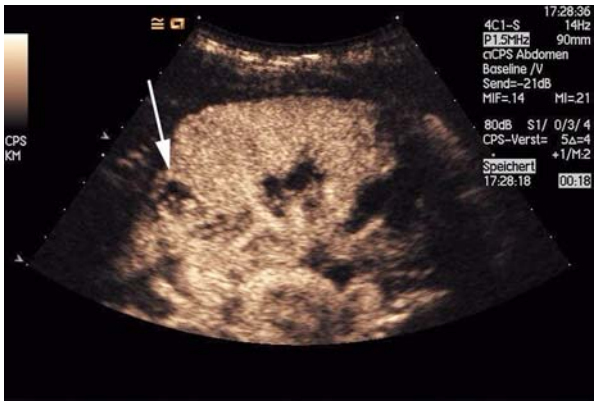


Figure 26 Patient with acute lobar pyelonephritis on upper pole (a). CEUS (contrast-enhanced ultrasonography, same patient): visible perfusion defect 18 sec. after contrast injection, typical for acute pyelonephritis (b).

a



b



Specific sonographic findings

Modifications of the kidney are primarily divided into different groups of classification by following sonomorphological criteria. In a single disease, often more than one criterion is abnormal. In such cases, the dominant criterion is being used for the classification and additional criteria complement the findings.

Diffuse changes of renal parenchyma

Kidney with abnormal position and shape

The kidney goes back to a complex genesis. The human kidney develops, similarily to other mammals, in three steps, or likewise, generations: the pronephros, which corresponds to the primitive kidneys of fish and basically fulfills the function of the glomeruli and the proximal tubules. The stucture of the pronephros appears at the human embryo already in the fourth week of pregnancy. They never transgress the rudimentary tubular stadium and, in opposition to the kidney of fish, they never produce any urine and shortly after, they regress. The largest part of the pronephros persists and becomes the structure for urinary collecting system of the following kidney generation.
The development of the mesonephros can be observed towards the end of the fourth week, immediately caudal of the rudimentary pronephros. They consist of large and elongated organs with fully formed glomerula. They function as interim kidneys for a period of approximately four weeks untill the permanent kidneys are fully developed. The tubules of the mesonephros open in the Wolffian duct, which is the caudal continuation of the pronephros duct and which more distal ends in the cloaca. While the mesonephros regresses towards the end of the first trimester, some of the tubules regularly persist to develop into the efferent ducts of the epididymis. The development of the metanephros or the definitive kidney starts at the beginning of the fifth week. Approximately four weeks later, they commence to function. The permanent kidney develops from two main sources: the ureteric bud and the metanephric blastema. The ureteric bud forms a protuberance of the Wolffian duct close to its connection to the cloaca, the metanephric blastema is derived from the caudal portion of the nephrogenic cord. Ureteric bud later on evolves into the ureter and the extended middle section into renal pelvis, its top branch several times and evolves into the calices and the collecting duct. The ampullary end of the arch-like collecting duct induces the development of glomeruli and tubules in the metanephric blastema. Each collecting duct is therefore linked to

proximal and distal tubules as well as to the loop of Henle and to the glomerulus. It forms the functional unit of the kidney, the nephron. The development of the glomeruli is already completed in the 32nd week of pregnancy, when the final number of glomeruli is reached. In cases of mislead or disturbed development, the ureteric bud plays a central role due to its impact onto all complex modifications of the architecture of the kidney. In some cases, disturbances of fusional processes of single renal lobuli occur, too. This leads to a series of anomalies considering structure, position and form of kidneys.

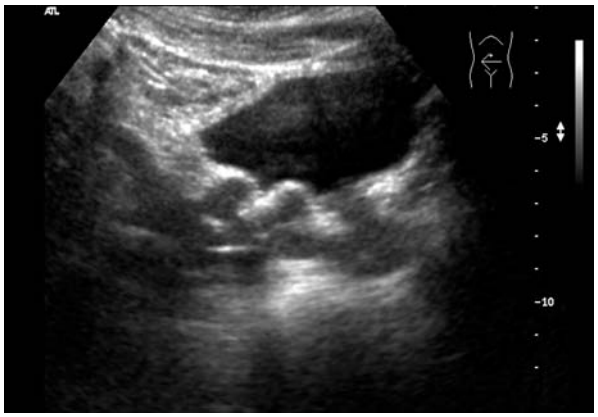
Agenesis

If there is no occurance of interaction between the ureteric bud and metanephric blastema, no kidney develops on that side. Simultaneously, ureters and collecting duct are missing and abnormalities in the area of the epididymis are being observed. If no kidney is found in the typical position, one can assume a case of agenesia.

Aplasia

In case of a faulty interaction between Ureteric bud and metanephric blastema, the kidney's Aplasy can occur. Sometimes, minimal traces of the kidney on a scale of a few millimeters can be discovered and occasionally, anechoic tubular malformations in the area of the falsly developed ureters appear [Fig. 27].

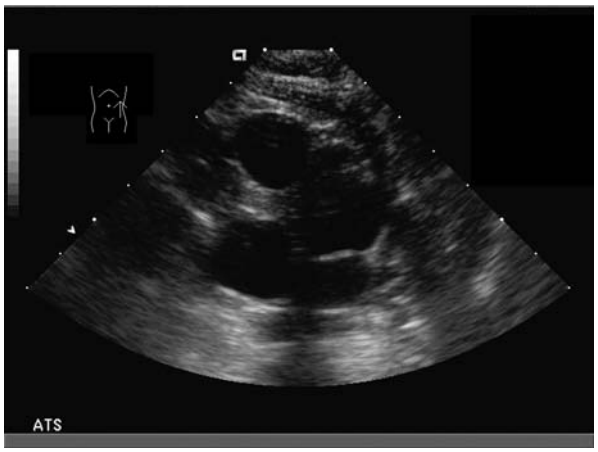
Figure 27 Tubular anechoic ureteric malformation in case of renal aplasia.



Dysplasia

A multicystic organ with mostly degenerated tubular structures results from a slightly better interaction between ureteric bud and metanephric blastema. Only in a few cases, intact nephrones are developed. They fulfill excretory functions, but usually this gets lost shortly after birth. The multicystic dysplastic kidney (MCDK) shows mostly on one side only [Fig. 28). Sometimes only a part of the whole kidney manifests such a degeneration.

Figure 28 Multicystic dysplastic kidney (MCDK) in 8 years old child.



Rotation anomaly

A failed or exzessive rotation can be taken into consideration, when the renal hilum leaves the kidney, which happens mostly towards ventral [Fig. 17]. Anomalies of rotation are often associated with kidney dystopia.
Rotation anomaly can cause limited and unusual sections of the parenchyma which generate images easily mistaken for solide mass.

Double kidney

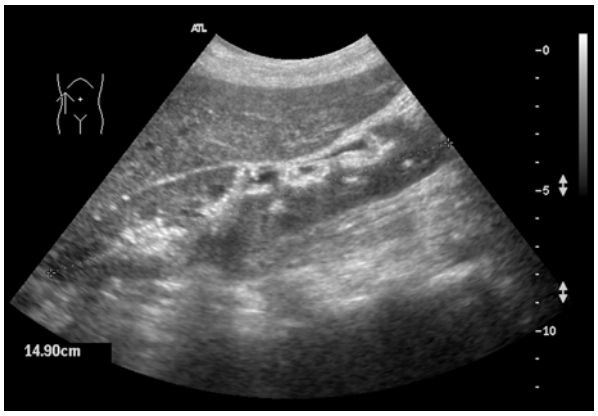
In case of a doubled system, a particularly long kidney can be found, usually with an incision in the parenchyma and with a parenchyma bridge [Fig. 29]. The verification of arteries and two ureters proves a double kidney.

Figure 29 Double kidney with two renal arteries.

Sigmoid kidney

The Sigmoid kidney is a special case of the double kidney. Caudal part is malrotated, the other shows an inconspicuous renal hilum [Fig. 30].

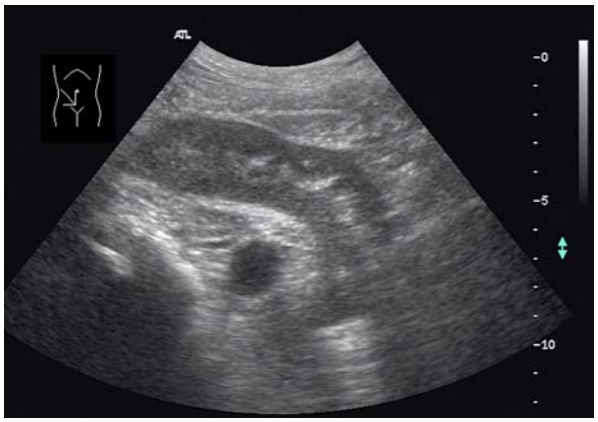
Figure 30 Sigmoid kidney with normal upper and malrotated caudal part.



Horseshoe kidney

The kidneys are fused at the lower pole, the parenchymal bridge is located in front of the aorta and should not be confused with a solid mass [Fig. 31].

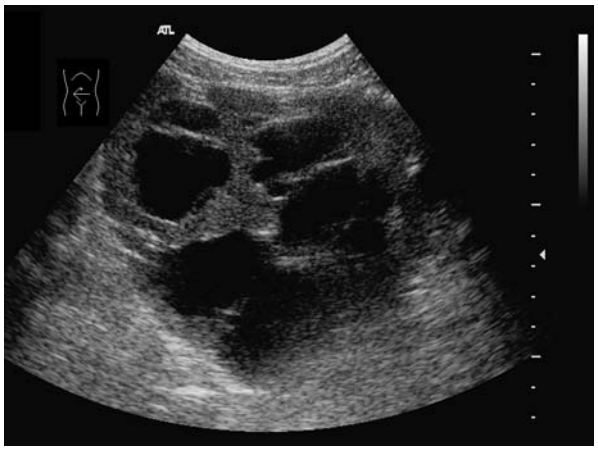
Figure 31 Horseshoe kidney:



Cake kidney

The cake kidney is located in front of the sacrum. It consists of a totally merged kidney with one single ureter [Fig. 32].

Figure 32 Cake kidney.



Hypoplastic kidney

The hypoplastic kidney consists of an overall reduced but otherwise normally developed kidney. In most cases, the width of the parenchyma amounts to < 13mm, the total volume amounts to < 80ml/1,73m^2 BSA.

Ectopic kidney

Ectopic kidneys are kidneys, which are away from their normal positions. Failure to show the kidney in a normal position doesn't necessarily mean a case of either aplasia or agenesis, and the kidneys should necessarily be sought in other places in the retroperitoneum. Deep in the belly is found a pelvic kidney, these ectopic kidney is often supplied from the iliac artery (similar to the kidney transplant).

Nephroptosis

This is a dynamic anomaly of position. While in a lying position, the kidney is to be found in the normal location, while in a standing position, a dropping by more than 5 cm is being observed.

Enlarged kidneys

Kidneys count as enlarged when their longitudinal diameter accounts to more than 13-14cm or, alternatively, to a volume over 200ml/1,73m^2 BSA. The width of the parenchyma lies usually beyond 18mm, the cortical width over 10mm. Enlarged kidneys are further subdivided into kidneys with normal cortex echointensity, with hyperechoic cortex and with hypoechoic cortex.

Enlarged kidneys with echo norm cortex

A *compensatory renal hypertrophy* or a *solitary kidney* lead to an increase of volume as well as of parenchymal thickness, which reaches in most cases over 20mm. The cortex usually shows echo norm or slightly hyperechoic. The mark pyramids show inconspicuous in these cases.

Enlarged kidneys with hyperechoic cortex

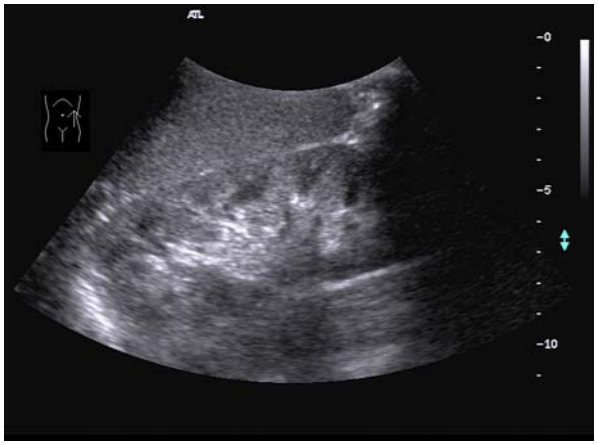
The finding of an enlarged kidney with hyperechoic cortex [(25;26)] and conspicuous medullary pyramids is nonspecific [(45)]. The increase of echogenicity is basically caused by an interstitial edema and/or by infiltrates [(32;42)]. Often, these are acute diseases. Normalisation of the kidney size and the echogenicity of the kidney cortex can be observed after these diseases have been successfully treated or after they fade away (acute and quickly progressive glomerulonephritis, acute interstitial nephritis, acute pyelonephritis). In case of bilateral disease a normalisation of the kidney function takes place. Because findings are nonspecific, a further assignment of the disease is possible only by clinical criteria:

- Acute nepritis
- Nephrotic syndrom
- Acute renal failure
- Acute pyelonephritis
- Infiltrative nephropathies
- Acute urinary tract obstruction

Acute nephritis

Acute nephritis syndrome is defined clinically by glomerular hematuria and/or erythrocytes casts and hypertension and/or renal failure and/or edema. This could be caused by acute glomerulonephritis or systemic disease (panaerteriitis nodosa, Wegener granulomatosis etc.) The definite diagnosis of acute glomerulonephritis is made through a renal biopsy [Fig. 33]. The kidneys with acute nephritis show enlargement, hyperechoic cortex and hypoechic, clearly visible mark pyramids.

Figure 33 Acute glomerulonephritis (IgA Nephropathy).



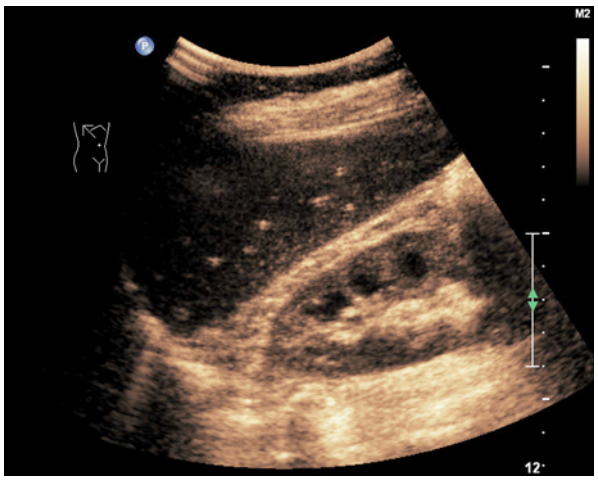
Nephrotic Syndrom

The nephrotic syndrom is defined by a proteinuria > 3,5g/24h/1,73m^2 BSA. The kidney cortex is hyperechoic. In most cases, they don't show much enlargement, instead they are often surrounded by an anechoic sharp edge due to hypoproteinemia. In this case, too, a more precise classification of the single diseases is only possible after renal biopsy.

Diabetic nephropathy

also manifests itself often with a nephrotic syndrom. Alredy in early stages, an enlargement of the kidney can be observed. In the beginning, the kidney cortex is shows descrete, but later on it becomes more hyperechoic. Even with increasing renal failure, the kidney show enlarged and even normal size in dialysis stage [Fig. 34].

Figure 34 Diabetic nephropathy with nephrotic syndrome, plasma-creatinine 190 µmol/l.



Acute renal failure

Acute renal failure is defined by its existence in a time period shorter than three months, where there is no renal anemia yet and the level of serum creatinine is increasing rapidly, weekly or even daily.

Acute interstitial Nephritis

Acute interstitial nephritis is a disease which arises from the reactions onto various medications (eg penicilline, non-steroidal anti-inflammatory drugs etc.) or infections (eg Leptospirosis). Its sonomorphology is characterised by enlarged kidneys, by a hyperechoic cortex and clearly visible hypoechoic mark pyramids [(45)]. This disease can be assumed on the basis of a characteristic case history, its progress, the sonomorphology and the proof of eosinophile leukocytes in the urine, although a definitive diagnosis can only be guaranteed by renal biopsy.

Acute tubular necrosis

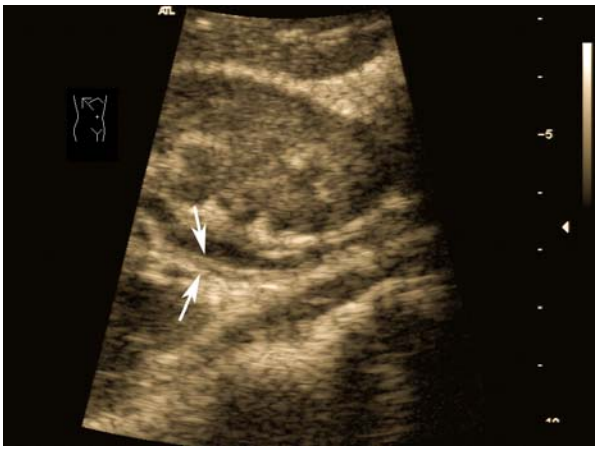
If an acute tubular necrosis emerges during a circulatory shock, the kidneys usually don't show any enlargement and the cortex isn't more echoic than normally. In many cases, the kidneys sonomorphically show completely inconspicious. This differs from the case of a crash kidney, where inhomogeneous, hyperechoic and hypoechoic areas of the cortex on one hand, and an overall enlargement of the kidney are to be found. Similar results are also found in cases of toxically caused tubular necrosis, eg due to aminoglycosides [(17;41;42)].

Acute Pyelonephritis

Severe cases of pyelonephritis show a diffuse enlargement of the organ whith a hyperechoic cortex and prominent hypoechic pyramids. A thickening of the renal pelvis [(12)] wall is in most cases detectable ($\geq$ 3 mm; norm < 2mm). Due to a disturbance of the renal pelvis motor activity, a slight dilatation can be observed [Fig. 35]. Of course, even typical signs (fever,

lumbar pain and high CRP) make acute pyelonephritis propably. The destinction between an acute pyelonephritis and a banal urinary tract infection becomes conclusive with the use of CEUS. It allows the representation of less perfused areas in the kidney parenchyma and allows therefore to place the diagnosis of an pyelonephritis, similarily to MRT [(15;46)]. Pyelonephritis can also lead to focal modifications, where often only single lobulus fall ill (see chapter: focal changes of the kidney parenchyma).

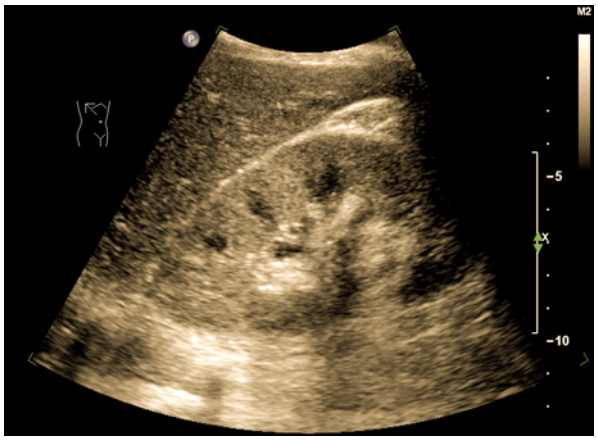
Figure 35 Acute pyelonephritis in a 16 years old girl: Cortex and medulla are hyperechoic, pelvis wall thickened (3 mm).



Infiltrative Nephropathies

The category of infiltrative Nephropathies includes diseases, which also show large hyperechoic kidneys, like storage diseases, lymphomas and leukemias. Of all storage diseases, clinically the most relevant one is **Amyloidosis**, which manifests through a nephrotic syndrom. The Echo compaction of the kidney cortex proves to be very strong in these cases, and the kidney cortex itself is very strong. The whole kidney is diffusely infiltrated by Amyloid [Fig. 36]. The very rare cases of another storage disease called **Nieman-Pick- disease**, an lipid storage disorder, looks similar. A diffuse infiltration of the kidney by a **Non-Hodgkin-Lymphom** or by **Leukemia** can also result in the manifestation of a large, kidney with hyperechoic cortex and with even more echoic and therefore less prominent mark pyramids.

Figure 36 Amyloidosis with nephrotic syndrome and normal creatinine: large kidney with hyperechoic cortex.



Acute urinary tract obstruction

In cases of a fresh renal colic with an acute urinary obstruction, an enlargement of the ill kidney as well as an hyperechoic cortex can be observed. These are the first reactions before the kidney pelvis is being extended. At this stage, a significantly rised resistive index (RI) can be determined for the ill kidney ($\Delta RI \geq 0{,}10$ on the ill side).

Enlarged kidneys with hypoechoic cortex

This configuration is rare. It has been observed during cases of severe acute *right heart failure* as well as in cases of fresh *renal vein thrombosis* and also during *Hanta virus infection,* an attenuated echogenicity of the cortex with an overall enlarged kidney has been described. A part of the inflammated kidney parenchyma can also appear hypoechoic in cases of *focal pyelonephritis* (see chapter focal changes of the kidney parenchyma).

Small kidneys

Small kidneys are defined as kidneys with a longitudinal diameter below 9 cm or alternativ with a kidney volume below 80ml/1,73m2 BSA (=body surface area). Reduced kidneys appear during kidney hypoplasia, vascular diseases of the kidney and in cases of chronic diseases of the kidney parenchyma. As enlarged kidneys, also small kidneys are further subdivided into kidneys with normal cortex echointensity, with hyperechoic cortex and with hypoechoic cortex.

Small kidney with normal cortex echointensity

Kidney hypoplasia

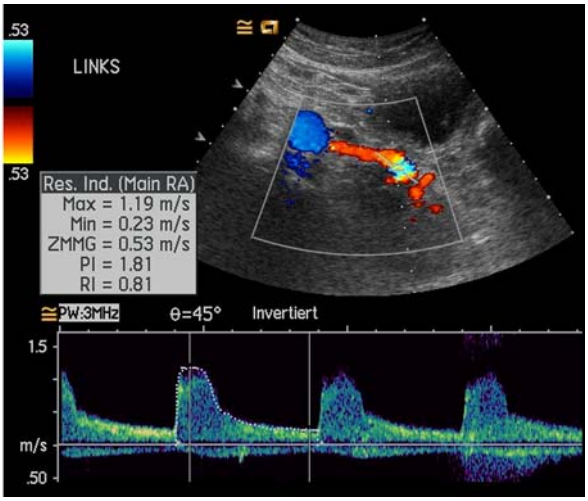
Hypoplasia goes back to a disturbance of the kidney's development and ist characterized by a size reduction of the organ and an otherwise inconspicuous look. Combinations of Hypoplasia with Dystopia and Malrotation can occur (see chapter: kidney with abnormal position and shape).

Nephrosclerosis

The kidneys of patients suffering from general arteriosclerosis and/or longer-existing hypertension show in most cases only discretely smaller. They have an inconspicuous echogenicity of the kidney cortex and often enough, characteristic vascular scars can be found

(see chapter: kidney with contour changes). The resitive index RI increases often with values of RI more than 0,80. Prognostically, this has to be considered inconvenient, since it corresponds to a rapid progession towards renal failure [Fig. 37].

Figure 37 Nephrosclerosis with high RI (= 0.80).



Small kidney with hyperechoic cortex

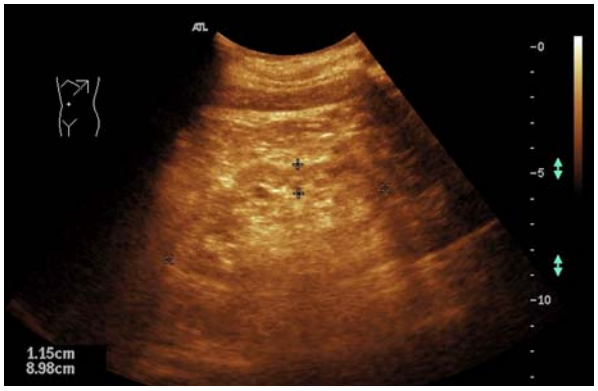
It makes sense to comine further clinical features and syndroms into the differential diagnosis when dealing with this group of diseases. If the kidney shows small in size on both sides, then chronic renal failure appears regularly. This isn't the case during a one-sided disease (pyelonephritis). Chronic renal failure is characterized by an existence of at last three months as well as renal anemia and/or renal osteopathy. Small kidneys on both sides means the chronic character of renal failure [Fig. 38].

Figure 38 Small kidney with hyperechoic cortex.

Chronic glomerulonephritis and chronic not destructive interstitial nephritis

In both cases, the sonomorphology looks very similar. An hyperechoic cortex can be observed as well as prominent mark pyramids, fine granular contour of the kidney and a well-preserved architecture [Fig. 39]. Again, obvious lobulation shows in many cases. A definitive diagnosis can be placed only within a clinical context (eg glomerular erythrocyturia during chronic glomerulonephritis) or on the basis of renal biopsy, which also holds often prognostic value [(25;26)].

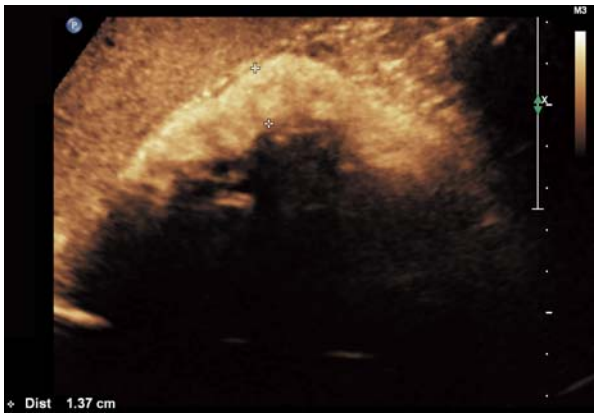
Figure 39 Chronic glomerulonephitis.



Primary oxalosis and hereditary hyperoxaluria

In both cases are renal cortex and medullary pyramids extremely hyperechoic. The oxalate crystals are diffusely distributed in the parenchyma. The normal parenchymal structures are partly no longer regcognizable [Fig. 40].

Figure 40 Hereditary hyperoxaluria.

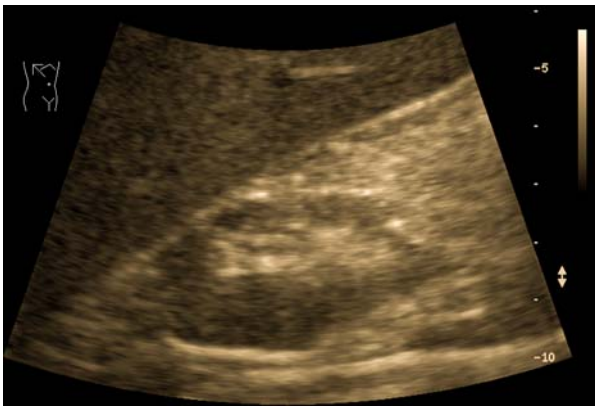


Chronic Pyelonephritis

Is often a unilateral disease, may of course be on both sides and then also lead to chronic renal failure. Observations include typical pyelonephritic scars (see chapter kidney with contour changes) and a hyperechoic cortex, while the architecture is disturbed. Large areas of the kidneys have shrunken, others are locally hypertrophic [Fig. 41]. In combination of urinary

tract obstruction and infection, a special case of pyelonephritis called *xanthogranulomatous pyelonephritis* mimic tumorous diseases [(22)]. *Renal tuberculosis* shows often parenchymal scarring and calcifications and congestion and stenosis in urinary collecting system.

Figure 41 Chronic pyelonephritis with irregular destructed kidney.



End-stage renal disease (ESRD)

All chronic diseases can lead to a hardly recognizable and completely shrunken kidney. Even histologically, with ESRD it is to difficult to differentiate between chronic pyelonephritis and chronic glomerulonephritis. The tuberculous pyonephrosis with extensive caseous necrosis is known as the end-stage tuberculosis. It usually doesn't show any traces of a regular structure at all.

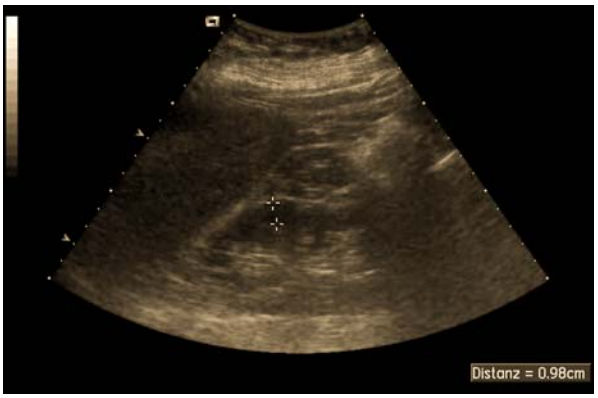
Small kidneys with hypoechoic cortex

Renal artery stenosis

Renal artery stenosis can lead to a a hypoperfusion of the whole organ and to an atrophy of the tubules, which move closer together. There are no infiltrates or edema [(41;45)]. Therefore, the kidney cortex seems more hypoechoic [Fig. 42,43]. In case of a corresponding clinic with hypertension, a systematic search for renal artery stenosis would be sensible (see chapter changes of the renal arteries).

Figure 42 Small kidney with hypoechoic cortex with stenosis of renal artery.

Figure 43 Small kidney with hypoechoic cortex with stenosis of renal artery.



Kidney with contour changes

The normal kidney contour is smooth. It forms the regular demarcation of the bean-like organ. During numerous norm variations and diseases, we can find typical deviations of this contour[(41;42;45)].

Dromedary hump

A hump of the outline outwards, the so-called Dromedary hump, is a form variation of only the left kidney. It is presumed, that due to a large ammount of space caudally of the spleen, the kidney can grow here very well during organogenesis which is opposed on the other side, where the kidney lies close to the large liver.

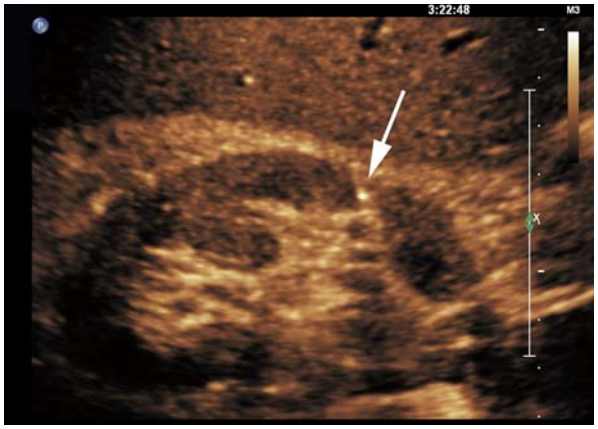
Lobulation

The kidney originates out of the merging of 12-15 lobuli. During childhood and sometimes also in adulthood, this fusion shows incomplete. Such an incomplete fusion or merging is called lobulation. Each mark pyramid close to it form a lobulated kidney contour. At that, the mark pyramid always shows in the epi center of the lobulus, and neighboring lobuli connect with each other in an arch way. Sometimes, fusion grooves appear slightly deeper. Even with depth it forms a sharp angle, but the hump still is clearly recognizable [Fig. 12).

Vascular scar

Opposed to lobulation, vascular scars originate out of the arterial flow curcuit of renal arterial branches, which lead to the infarction of the tissue. The vascular scar is mostly triangular, an acute angle shaped defect, but sometimes it can be also trapezoid [Fig. 44]. Trapezoid scars often lie between single mark pyramids, where the interlobar arteries plunge into the parenchyma. Such scars can often be found with patients that suffer from severe arteriosclerosis, morbus embolicus during endocarditis or atrial fibrillation. Also in cases of longer-existing hypertension, such scars can be often observed.

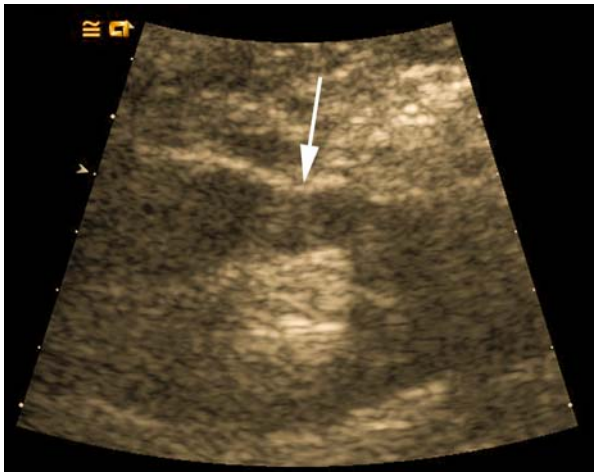
Figure 44 Vascular scar.



Pyelonephritic scar

The pyelonephritic scar is part of a chronic inflammatory changes, taking place in either one or more lobuli. The affected kidney cortex shrinks, it is located just above a mark pyramid and in most cases, it shows more hyperechoic than the surrounding cortex. Therefore, a shallow and concave scar with a mark pyramid at its epi-center develops under these circumstances [Fig. 45].

Figure 45 Pyelonephritic scar.



Kidney with abnormal echo pattern

The normal echo pattern of the kidney shows homogenuous and the mark pyramid is mostly well recognizable as a triangular shaped and nearly anechoic formation. The peak of the pyramid respectively the renal papilla is pointing towards the hyperechoic renal sinus. The base is limited by arcuate arteries. The ***kidney cortex*** can show both ***hyperechoic and hypoechoic*** (see chapters: large kidneys, small kidneys). Changes of the medullary pyramids appear additionally to that.

Kidney with hypoechoic medullary pyramids

In cases of slight right heart failure, in patients with diuretics and with IgA- nephropathy, the medullary pyramids seem particularly hypoechoic. After precisely measuring the kidney cortex' and the medullary pyramids' echogenicity before and after having prescribed diuretics, it becomes evident, that one the effect of particuarly hypoechic medullary pyramids had been evoked by an echo densification of the cortex. Other conditions, like an IgA-nephropathy or the early stage of a transplant rejection, can also show the image of such particularly prominent and hypoechoic medullary pyramids [(17; 32)].

Kidney with hyperechoic medullary pyramids

There is a series of diseases showing the opposite of the usually common echo pattern, meaning the medullary pyramids show less echo than the kidney cortex[(45)].

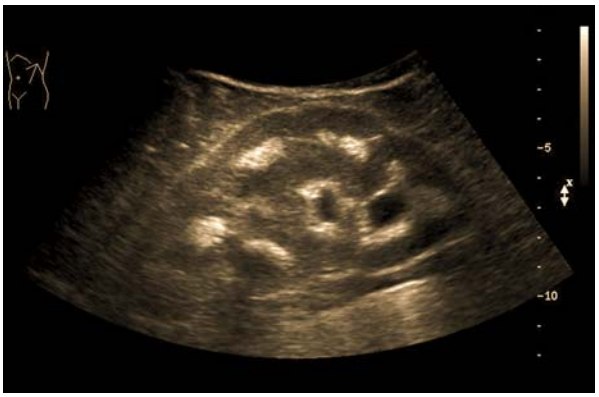
Renal failure in newborns

Newborns that have been born with oxygen lack show a renal failure which lasts only a couple of days. Characteristically, the medullary pyramids appear hyperechoic. At the origin of this, a breakdown of the Tamm-Horsfall protein in the collecting ducts is being presumed.

Medullary nephrocalcinosis

There are different conditions leading to hypercalciuria and precipitation of calcium in the medullary pyramids. One of them is primary hyperparathyroidism, others are sarcoidosis or multiple myeloma. Hyperechoic medullary pyramids are also being observed during Conn-syndrom, with some forms of Bartter-Syndrom [Fig. 46] and during furosemide abuse. Under all these conditions, the medullary pyramids gradually show hyperechoic, beginning on the lateral side and pulling slowly into the middle of the papilla.

Figure 46 Medullary nephrocalcinosis in Bartter Syndrome with hyperechoic pyramids.



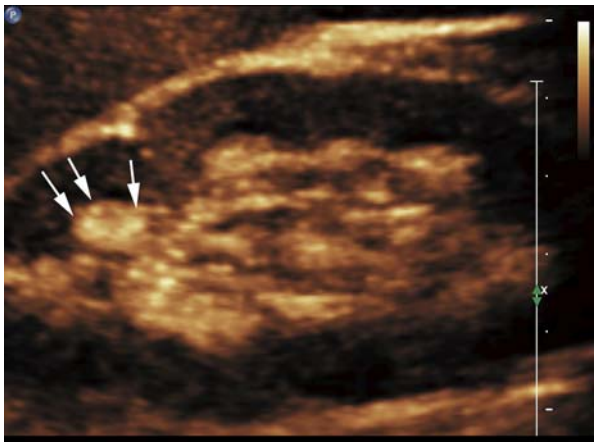
Urate nephropathy

In cases of acute urate nephropathy, caused by a sudden arse of a large amount of uric acid, a significant increase of volume as well as an echogenic kidney can be observed. These modifications are a consequence of the precipitation of uric acid crystals in the tubules and the thereby caused urinary obstruction. Sometimes, the crystallization can be localized in the collecting ducts, in particular during a longer occouring hyperuricosuria. Here as well, hyperechoic medullary pyramids are being found.

Medullary sponge kidney

Medullary sponge kidneys form a congenital malformation of the collecting duct, which shows cystic extension and is often combined with distal renal tubular acidosis. The medullary pyramids are hyperechoic. Single little stones are being observed inside the cystic extensions. The whole of the mark pyramids is of an overall crumbly character with individual hyperechic spots, showing very fine posterior acoustic shadow [Fig. 47].

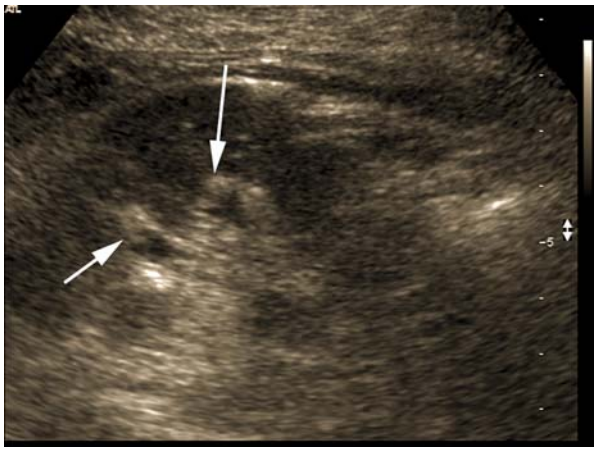
Figure 47 Medullary sponge kidney with very small stones (echogenic spots) in cystic extensions of collecting ducts (arrows).



Analgesic nephropathy

Due to the abuse of phenacetine, this has been a very common disease in Switzerland. Up to a third the whole dialysis population was affected by this illness. The formation mechanism comes from a capillary sclerosis in the vasa recta of the medullary pyramids, which leads to a papillary necrosis at the point of the same papilla [Fig. 48]. During an early state, hyperechoic spots are recognizable at the point of the papillas, later on almost whole medullary pyramids become calcified and necrotic. A small shrunken kidney with hyperechic calcified medullary pyramids can be found during advanced stages.

Figure 48 Analgetic nephropathy (early stage) with calcified tips of the papillae (arrows).



Kidney with abnormal architecture

To understand the most essential abnormalities of the kidney, it is necessary to be aware of the complex genesis of the human kidney (see chapter: kidney with abnormal position and shape).

Multicystic Dysplastic Kidney Disease (MCDKD)

Multicystic Dysplastic Kidney Disease goes back to a missing connection between Metanephros and Wolffian duct. This can take place either partially, where multicystic dysplastic focal modifications appear, or generally. In that case, the whole system of the kidney shows cystic degeneration (MCDKD=multicystic dysplastic kidney disease).

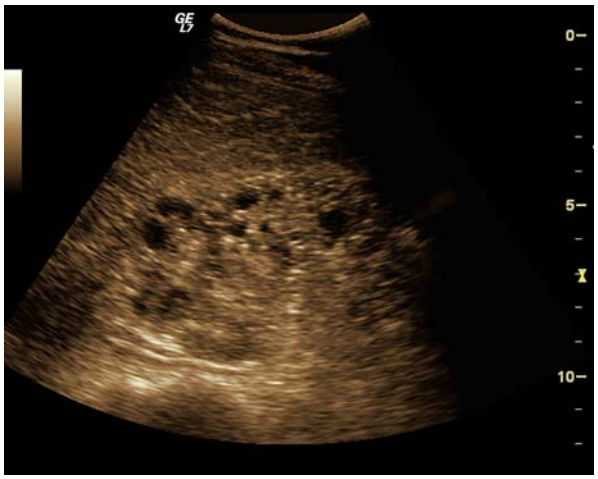
Polycystic kidney disease (PKD)

PKD is a familiar disease. The cysts form out of pre-existing tubular structures. Its appearances are being classified into different forms: the juvenile, the autosomal recessive and the adult, autosomall dominant varation of this kidney disease.

Autosomal recessive polycystic kidney disease (ARPKD)

In cases of ARPKD, numerous very small cysts are being observed [Fig. 49]. With newborns, their appearance recalls *the pattern of „salt and pepper*". Later on, multiple calcifications in the cysts' walls can be observed, which is being depicted and represented very well by twinkling.

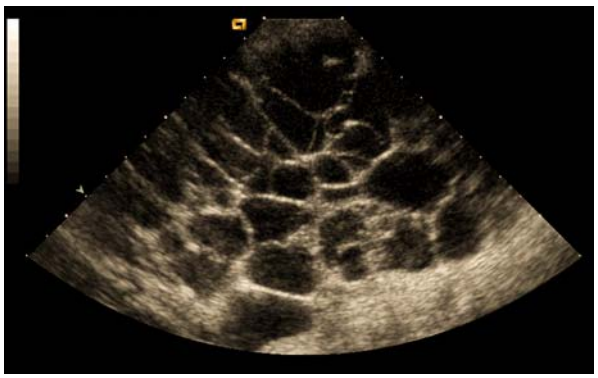
Figure 49 ARPKD with multiple, small cysts (salt and pepper pattern) in 27 years old woman.



Autosomal dominant polycystic kidney disease (ADPKD)

This variations consists of a large number of big cysts. Clinically, this disease manifests during adulthood. Next to the very big cysts, also numerous very small cysts are being observed [Fig. 50]. Many calcifications, represented by twinkling, sum up the image. The volume of such a kidney can reach up to 1000 ml.

Figure 50 ADPKD with multiple large cysts.



Secondary cystic degenerations with terminal renal failure (CRF)

With chronic renal failure, a number of secondary cystic degenerations comes into play which doesn't show any specific cause. The kidneys show multiple cysts, but they never reach the scale of variety of cysts or the volume of kidney during forms of ADPCKD. This is mostly being observed with long term dialysis patients.

Cystic renal cell carcinoma (RCC)

A cystic renal cell carcinoma can show great resemblance to multicystic dysplastic kidneys. Its image consists of a proof of calcifications and cystic as well as solid parts are visible [Fig. 51, 52]. In cases of very large tumours, the usually recognizable kidney parenchyma could be overlooked, which as a consequence would produce misinterpretations.

Figure 51 Cystic renal cell carcinoma.

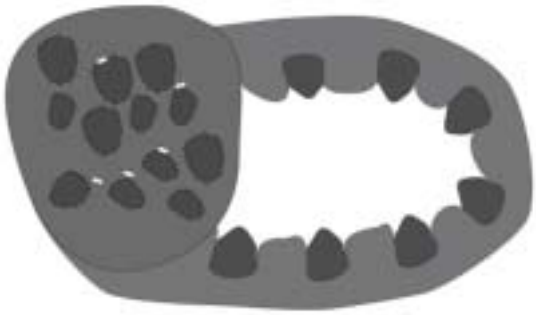
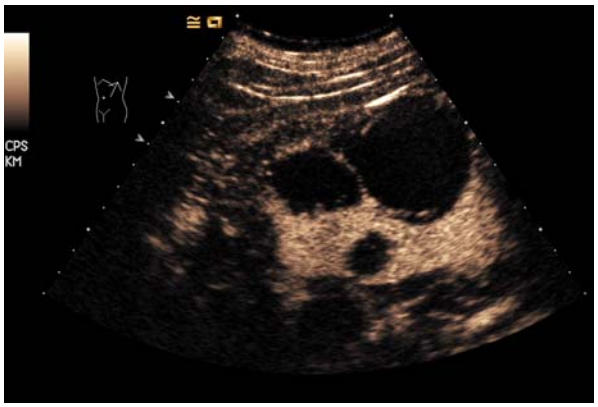


Figure 52 Cystic renal cell carcinoma: CEUS showing cystic and solide parts of tumor.



Focal changes of the kidney parenchyma

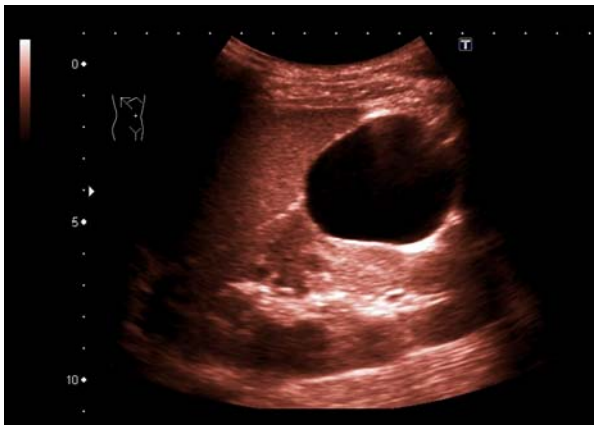
A change of architecture appearing in an otherwise inconspicuous kidney parenchyma is called a focal modification of the kidney parenchyma. Anechoic, hypoechic, hyperechoic and mixed focal forms of modification are being differentiated.

Anechoic focal changes of the kidney parenchyma

Simple renal cyst

A simple renal cyst forms about 65-70% of focal changes of the kidney. Its image consists of a smooth wall, posterior echo enhancement and tangential shadow [Fig. 53]. With ageing, the cysts usually grow. Up to 40% of adult patient show at least one simple kidney cyst.

Figure 53 Simple renal cyst (Bosniak I).



Calyx diverticulum

In differential diagnosis, one has to make a difference between kidney cysts and calyx diverticulum. They usually show a narrowing towards the kidney pelvis and some of them can hold stones [Fig. 54, 55].

Figure 54 Calyx diverticulum.

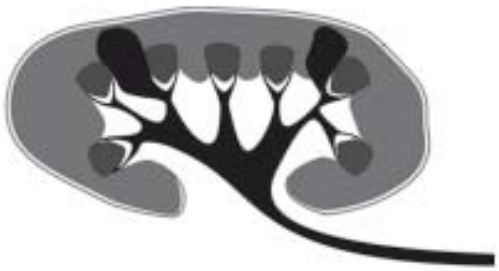
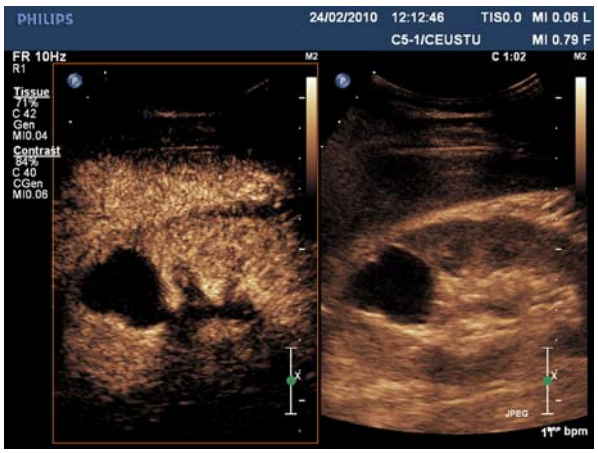


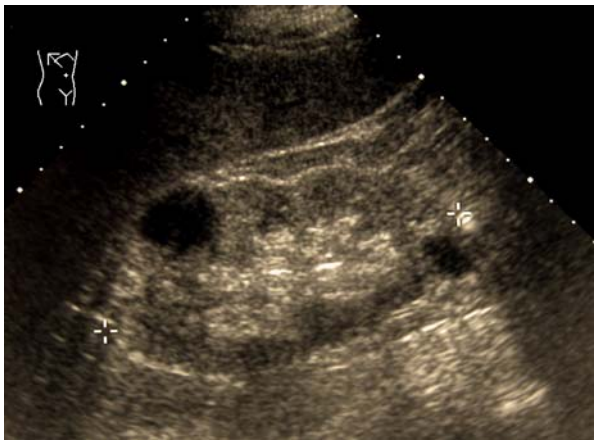
Figure 55 Calyx diverticulum: CEUS helps in differentiation from cyst; diverticulum has a clear connection to the collecting system:



Lymphoma

Sometimes, cyst-like formations appear during illnesses of the kidney caused by Non-Hodgkin-Lymphomas [Fig. 56]. For clarification, CEUS proves to be helfpul. Opposed to cysts, lymphomas are very well perfused.

Figure 56 Focal change in the kidney: cyst-like NHL.



Complex kidney cyst

Some cysts show thin septa and wall calcifications. But there are also renal cell carcinomas, which consist of cystic as well as solid parts. In the beginning, computer tomography helped for a better differentiation of cysts and mixed cystic-solid space demands [(6;21)], later on sonography using Bosniak classification was introduced [(2;19)]:

- **Bosniak I:** simple cyst without septations or other irregularities.
- **Bosniak II:** cyst with thin wall, septa and possible wall calcifications [Fig. 57].
- **Bosniak IIF** (=follow): echogenic cyst or, via CT hyperdensic cyst, which should be controlled in a regular time period of six months to not oversee a malignoma. CEUS is very helpful here [(18)]. An echogenic cyst (with bleedings) is being represented with this method as a cyst or non-perfused space [Fig. 58]. Therefore, it can be clearly

differentiated from a partly perfused space which will probably correspond to a cyst holding a carcinoma. Perfusion of even the smallest solid part of a cyst is always to be taken as a possible malignoma [Fig. 59].

- **Bosniak III:** cysts with thick and perfused septa call for further clarification. 40-60% of such changes prove to be maligne. Also here is CEUS very usefull [Fig. 52, 61, 62].
- **Bosniak IV:** cysts with solid parts, 85-100% maligne. Usually, such a case proves to be a clear cell renal cell carcinoma (RCC) with secondary regressive modifications and pseudocystic transformation. More rarely it can be a multilocular (cystic) clear cell renal cell carcinoma [Fig. 63]. Since the image of a ***benign cystic nephroma*** can also show solid parts, cystic parts and always shows calcifications, it is difficult to differentiate here.

Figure 57 Bosniak II: cyst with thin septa.

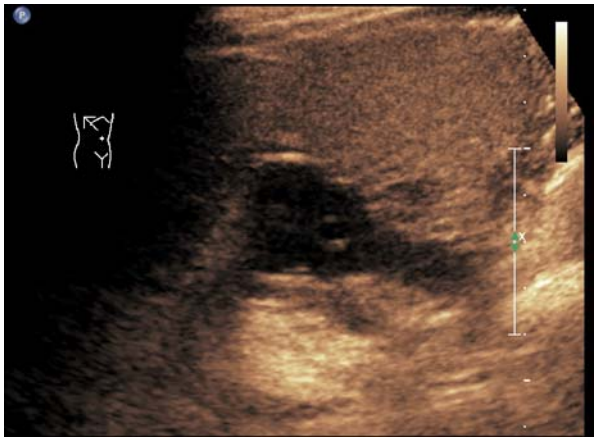


Figure 58 Bosniak IIF: echogenic cyst.

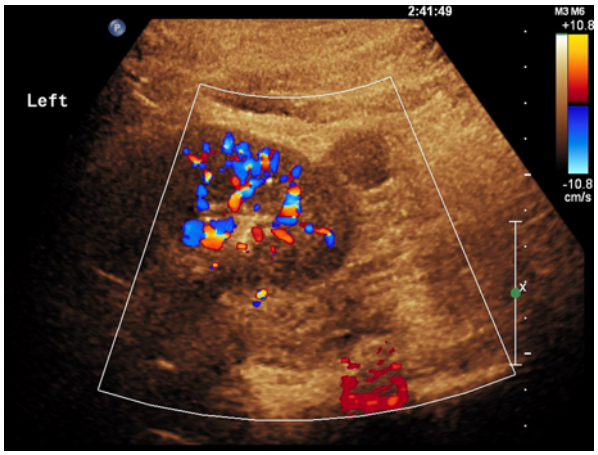


Figure 59 CEUS in Bosniak IIF: cyst is not perfused; simple renal cyst with bleeding.

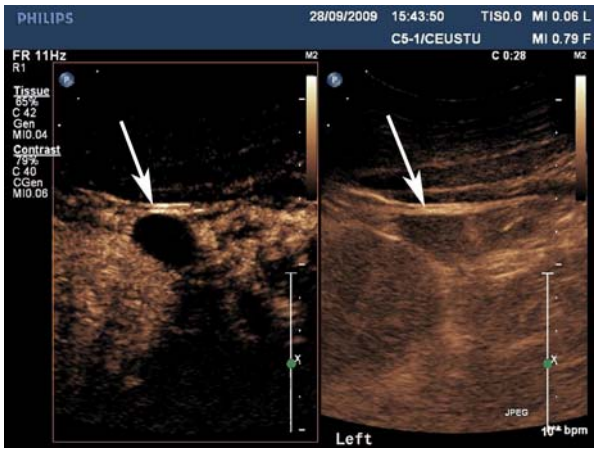


Figure 60 CEUS in Bosniak IIF cyst with perfused solid part; RCC.

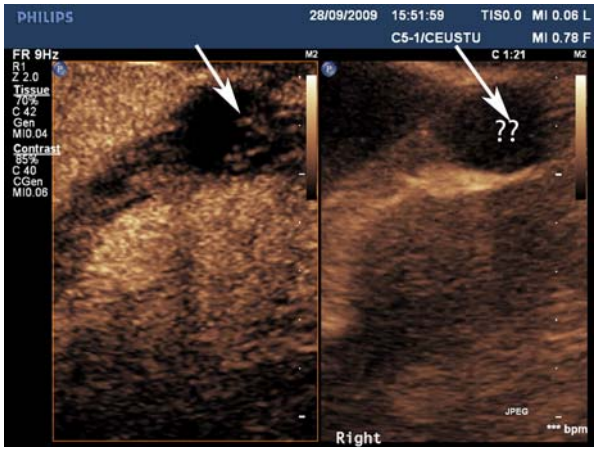


Figure 61 Bosniak III: cyst with thick septa.

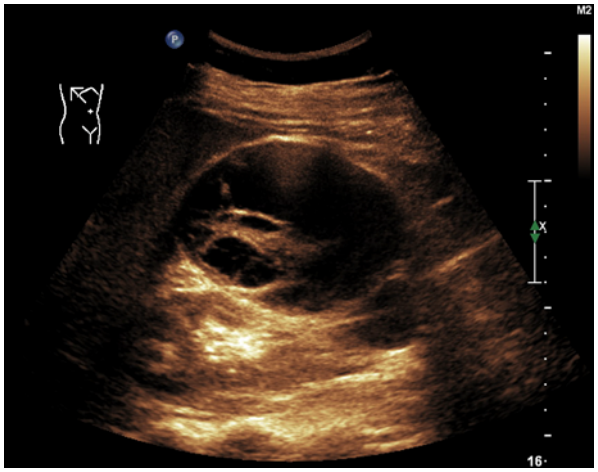


Figure 62 Bosniak III after CEUS [same case as in Fig 61): simple cysts without any perfusion, with bleeding.

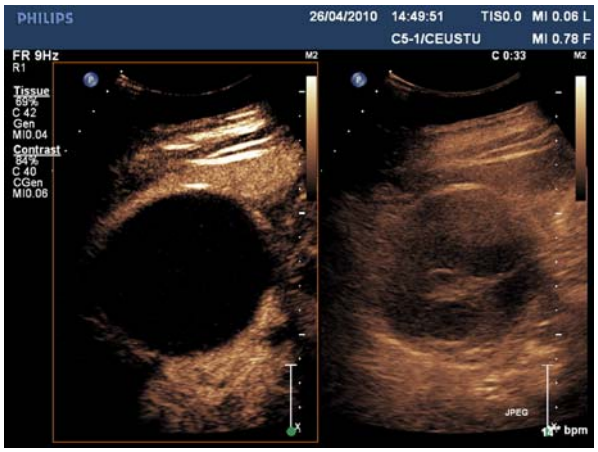
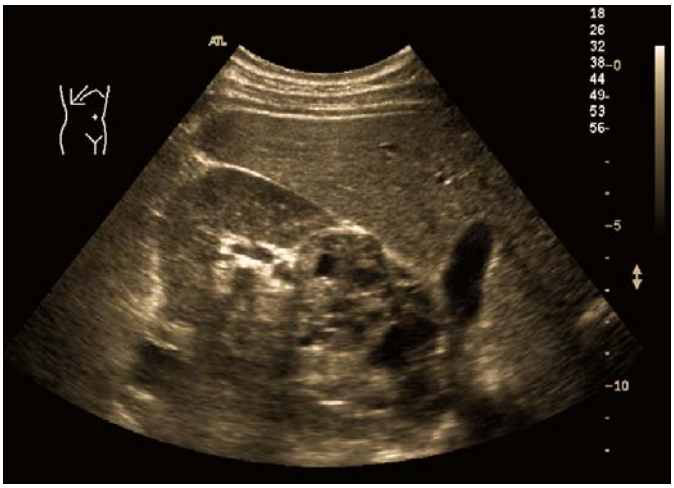


Figure 63 Bosniak IV: cysts and solid parts (RCC).



Hypoechic focal changes of the kidney parenchyma

Non-tumurous hypoechic focal changes of the kidney parenchyma

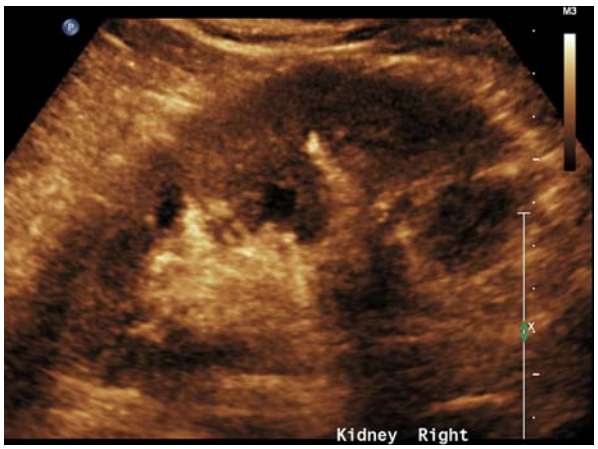
These are to be differentiated primarily from the genuine tumors [(10;27)].

Renal parenchymal tap (hypertrophied column of Bertin)

As a rule, this phenomenon is made from a isoechoic formation leading into the renal sinus. It was referred to as hypertrophied column of Bertin. But renal parenchymal tap is not a true hypertrophy of the cortex, but parts of the adjacent lobules reach deeper into the renal sinus [Fig. 10, 64]. Sometimes falls into the renal sinus an entire lobule including mark pyramid [Fig. 65]. Due to the inhomogenity of the result, those cases produce insecurity. With this accidental result, additional investigation is recommended. With atypical and ambiguous results, CEUS should be the examination of choice.

Figure 64 Renal parenchymal tap (“Parenchymzapfen”).

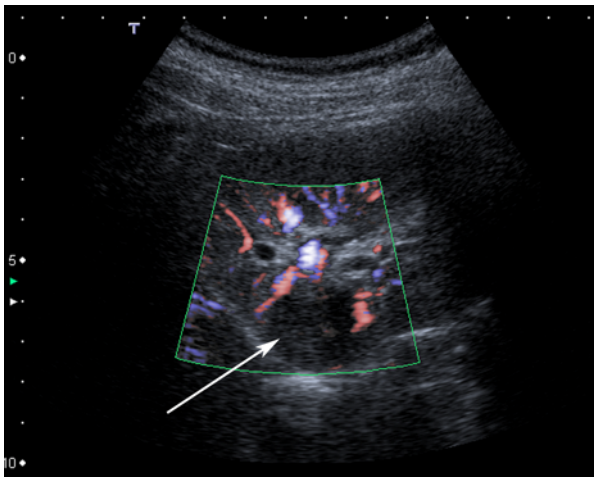
Figure 65 Renal parenchymal tap (“Parenchymzapfen”) with whole lobulus including medulla in renal sinus.



Lobar pyelonephritis

Lobar Pyelonephritis shows hypoechoic, meaning the infested lobulus is hypoechic. In the image, it shows edematously enlarged and, during PWDS- examination, its perfusion is reduced [Fig. 66]. This can lead to the suspicion of a real tumor, so the CEUS examination becomes crucial. If with pyelonephritis, aleady in the B-mode hypoechic areas become visible, one has to take into account a renal abcess. Due to the representation of non-perfused areas, CEUS can reveal the existence of an abcess already at its beginning, which would stay undiscovered in the B-mode of an otherwise unconspicuous kidney parenchyma.

Figure 66 Lobar pyelonephritis.



Renal cell carcinoma

The most important malignant tumor in the kidney is the renal cell carcinoma (RCC). It makes approximately 3% of all malignancies. Out of the primary kidney tumors, approximately 90% are formed by RCC, the remaining tumors are made from embryonal carcinomas, nephroblastomas, embryonal carcinomas and sarcomas, neuroblastomas and some very rare tumors. RCC are further classified and divided into clear cell carcinoma, papillary carcinoma, chromophobe carcinoma and collecting duct carcinoma [(18;45)]. The differentiation of benign and malignant kidney tumors is a major issue and the distinction is unsatisfactory both CEUS and CT, as well as an increasing number of tumor biopsy is also discussed [(35;36;47)].

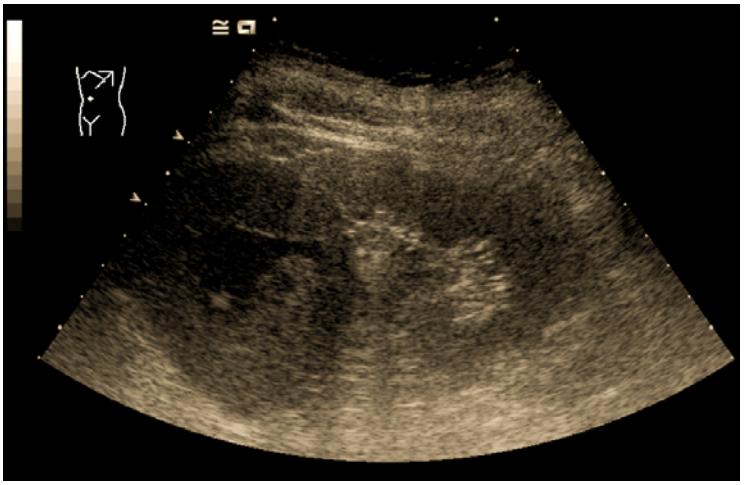
Clear cell renal cell carcinoma

This group is often hypoechoic and belongs to the most important hypoechoic focal modifications [Fig. 67-69]. These tumors are very good perfused, which can be represented by both CDUS and CEUS. With such tumors, the influx during CEUS is much steeper than in the surrounding kidney cortex. With larger tumors, both marginal increasement of the perfusion and inhomogeneity of perfusion inside the tumor with necrotic areas is characteristic.

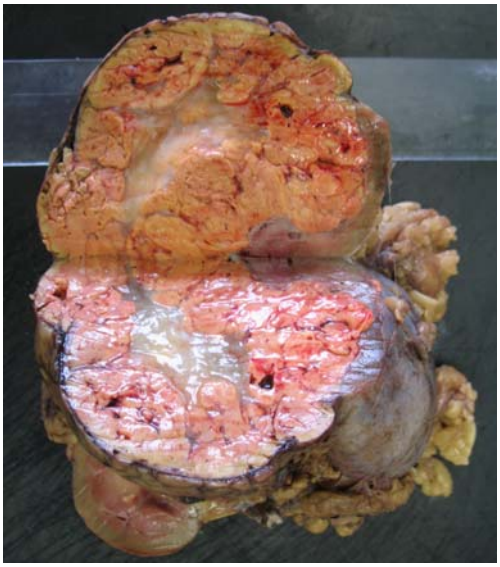
Figure 67 Renal clear cell carcinoma (RCCC).

Figure 68 RCCC with central hypoechoic zone (a). The same tumor as a surgical specimen with the same central zone (b).

a



b



Papillary renal cell carcinoma

This is also a hypoechoic tumor, which usually shows less inhomogeneity. A differentiation by the means of sonography only is impossible.

Renal cell adenoma

Usually this mass shows hypoechoic and it mostly occurs in the cortex. In most cases it shows as a homogeneous tumor without necrotic areas, which is not distinguishable from small renal cell carcinoma.

Hypoechoic angiomyolipoma

Although angiomyolipomas occurs in the field of hyperechoic focal changes, there is one variation of a tumor without any content of fat. It shows hypoechoic and can therefore not be distinguished from other hypoechoic masses.

Renal metastases

Renal metastases occurs with advanced disease. Metastases have been found in many tumors including lung cancer, ovarian cancer, leiomyosarcoma or neuroendocrine tumors. Since the introduction of CEUS, the existence of renal metastases has been occuring more frequently than it had been presumed before, which corresponds to pathological-anatomical studies.

Hyperechoic focal changes of the kidney parenchyma

Non-tumorous hyperechoic focal changes of the kidney parenchyma

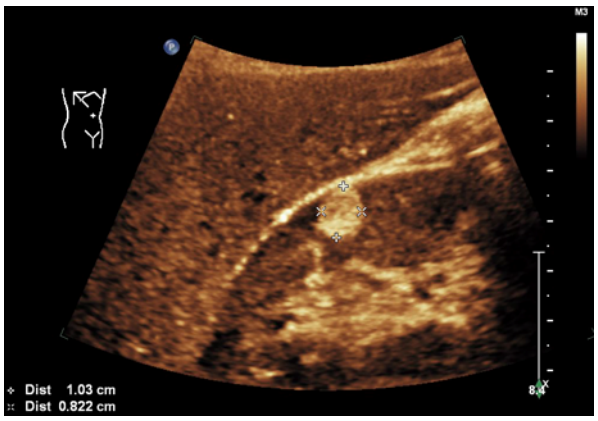
In some cases, *medullary nephrocalcinosis* can lead to the misdiagnosis of multiple renal tumors. Sometimes *focal pyelonephritis* can show more echo than the surrounding and this diagnosis therefore belongs to the differential diagnosis of hyperehcoic renal tumors. Distinction is usually possible with a synthesis of clinic and B-mode sonography, and the use of CEUS is necessary very rarely.

Angiomyolipoma

This hyperechoic tumor consists partly of fat, partly of vassels and partly of fibroid and proves to be relatively frequent with an occurance of 1% [(37)]. Mostly it is being discovered accidentally. Tumors are often smaller than 1cm and shows hardly any growth over the years. Such a small tumors are very well perfused. The influx during CEUS is almost as fast as that of the the surrounding kidney cortex [(39;47)]. Larger tumors have slower influx. The assessment of larger angiomyolipomas can sometimes prove to be somewhat difficult, because large tumors can show bleedings. As a rule, the clearly hyperechoic structure of the tumor proves to be of diagnosic importance [Fig. 69,70].

Figure 69 Hyperechoic angiomyolipoma.

Figure 70 Hyperechoic angiomyolipoma.



Renal cell carcinoma

Besides hypoechoic tumors, there are also hyperechic forms of renal cell carcinoma. In contrary to a case of angiomyolipoma, the use of Power Doppler sonography already shows strong perfusion. The very rapid influx of the tumor during CEUS is of further help.

Oncocytoma

This is a benign tumor which usually shows more echo than the kidney cortex and is of a mostly homogenuous structure. The occurance of scars located at the centre is quite common [Fig. 71, 72]. Using CEUS, a slowed down influx from periphery to centre is visible [Fig. 73, 74]. Because it is hardly distinguishable from chromophobic carcinoma, this tumor, altough benign, is often operated.

Figure 71 Oncocytoma.

Figure 72 Oncocytoma in B-mode without specific changes.

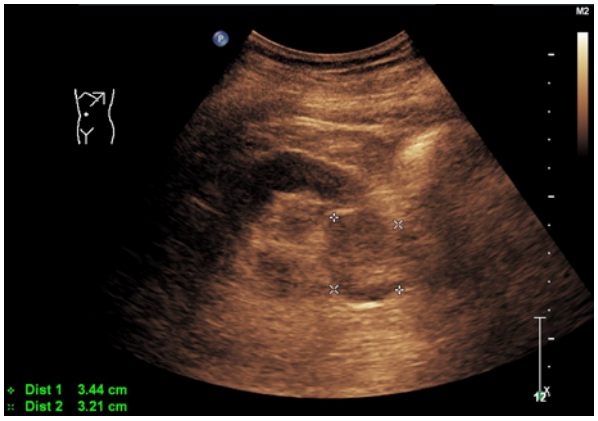


Figure 73 CEUS: Oncocytoma with typical central zone and perfusion from periphery to the center.

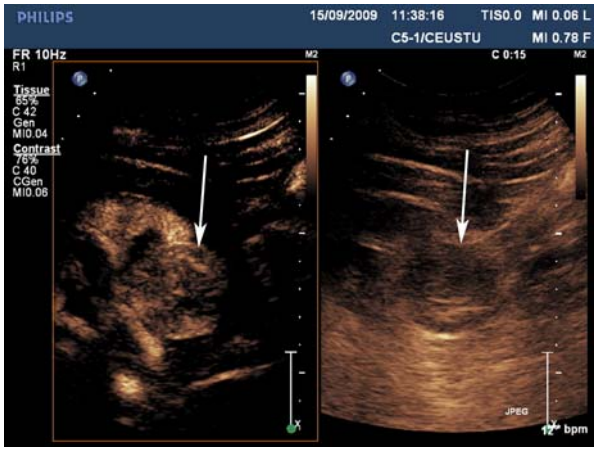
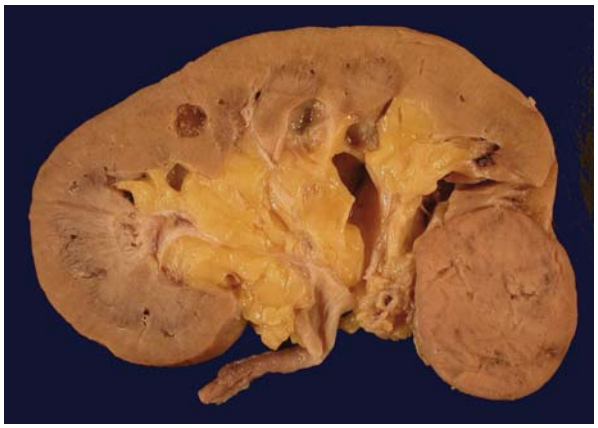


Figure 74 The same tumor as a surgical specimen.



Chromophobic renal cell carcinoma

The sonographic characteristics of this tumor are similar to the ones of an oncocytoma. Even the histological differentiation of these two tumors isn't always easy.

Bellini duct carcinoma (collecting duct carcinoma)

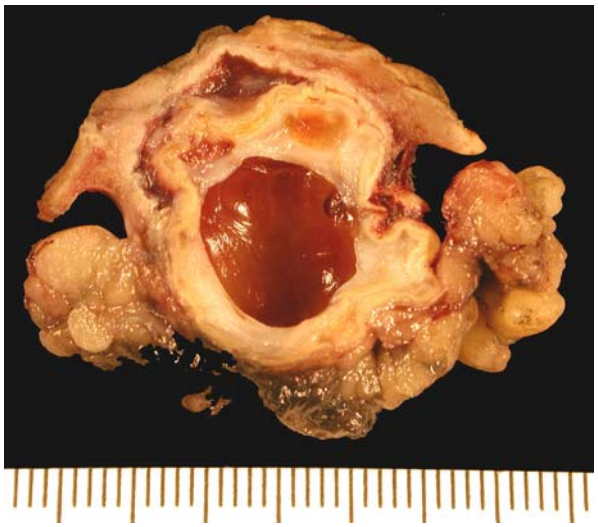
This tumor is very rare. It shows slightly more echo than the kidney cortex and isn't distinguishable from other forms by the means of B-mode sonography.

Complex focal changes of the kidney parenchyma

It can be problematic to differentiate between multicystic-dysplastic kidneys and tumors like the ***clear cell renal cell carcinoma*** or the ***multilocular cystic renal cell carcinoma***, as both of them can show solid and cystic or pseudocystic parts. With carcinoma, there is always a part of an intact kidney, as small as it might be, which is one of the main differences to ***multicystic-dysplastic kidneys***. Another change with a highly complex structure is the ***benign cystic nephroma***, a benignant tumor which consist of both cystic and solid parts and of calcifications, too [Fig. 75]. Even though the ***Wilms tumor*** is usually structured solidly, with very little cystic parts, variations of this tumor are found with almost only cystic parts. Even very large ***angiomyolipoma*** can show big bleedings and therefore consist of a complex structure with areas that are hypoechoic.

As a rule, tumors with a complex structure of partly cystic and partly solid shares prove to be large. The diagnostical task here is problematic and even combined imaging (CEUS, CT, MRT) doesn't always lead to a definitive diagnosis. Therefore most cases, benign or not, are being operated.

Figure 75 Benign cystic nephroma.



Changes of the kidney vessels

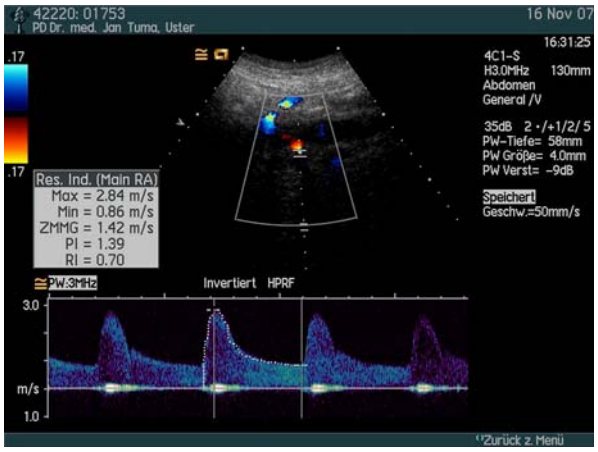
Changes in renal arteries

The kidney is an organ with very strong perfusion. A flow circuit of approximately 1500 litres of blood daily passes through them, which leads to a production of about 150 litres of primary urine and of approximately 1-2 litres of urine per day. The most crucial function of the kidney is to rinse blood, the glomerular ultrafiltration of blood. The glomerular filtration rate (GFR) or rather the production of primary urine by ultrafiltration, serves as an important parameter in analysing the kidneys' function. Normally, this value is around 150liter/ day or more than 90ml/min/1,73m^2 BSA. Therefore, the kidneys show a very high perfusion rate. While resting, the kidney is supplied by 20% of the heart volume and 100gr kidney tissue has perfusion of 400ml/min. Similarly to cerebral vessels, renal arteries show very low resistancy. By the means of color duplex ultrasonography (CDUS), renal arteries can be depicted both in longitudinal and profile sections. The spectral curves of renal arteries are monophasic. Due to the curve, an evaluation of following values becomes possible: Maximum systolic velocity V_{max} as well as minimum end-diastolic velocity V_{min} are being detected and the resistive index RI (Vmax - Vmin/ Vmax) is being calculated. Only 60% of all kidneys prove to have one main artery, the rest has 1-2 polar ateries. On average, renal arteries consist of a diameter of 3,5-6,8mm[(3)]. Cases of accessory arteries show a smaller diameter of around 3,0-5,0mm. A reduced diameter can be helpful in the diagnosis of accessory arteries. In case of a diameter less than 4,1mm it is highly probable the case of an accessory artery (with sensitivity and spccificity of 95%). With a diameter of more than 5,5mm it is hardly probable to be a case of accessory artery. Normal doppler values are as followed: Vmax = 70-180cm/sec , Vmin = 25-65cm/sec, RI 0,60-0,80. For a diagnosis of renal artery stenosis it is recommended to use color duplex sonography on one hand (with corresponding aliasing in cases of high degree stenosis), and to take advantage of the data derived from spectral curves by measuring systolic and diastolic velocities. Also systolic velocity in the aorta counts as another help, using the data and outcome of the reno-aortal ratio (RAR).

Renal artery stenosis

With hemodynamically significant stenois (> 50%), following values [(38)] apply as relevant limits: Vmax > 200cm/sec (sensitivity 92%, specificity 81%), RAR > 2,5 (sensitivity 92% , specificity 79%) and the quotient between renal artery and interlobar artery RIR > 5 (reno-interlobar ratio) as well as the Vmax of interlobar artery < 15cm/sec (sensitivity 91%, specificity 87%). A relevant stenosis can be treated with catheter dilatation. At high RI baseline (RI > 0.80), the success is unlikely [(20; 23; 30)] [Fig. 76].

Figure 76 Renal artery stenosis at the vessel origin in longitudinal section. Vmax = 2.84 m/sec.



Arteriovenous fistula and aneurysms

Besides detecting renal artery stenosis and accessory renal arteries, the proof of arteriovenous fistula (after renal biopsy) as well as aneurysmal broadened arteriovenous fistulas and aneurysmas of renal arteries are of great importance. The latter changes primarily consist of cystic changes, but could show a B-mode of a pulsating changes, which should be clarified by CDUS.

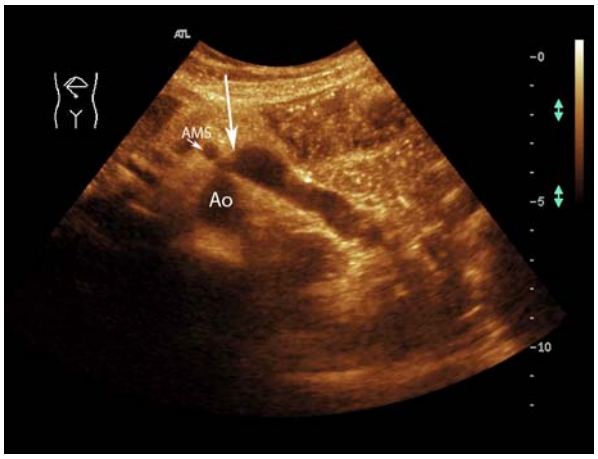
Changes in renal veins

With adolescents or very slim people, renal veins show as anechoic and broad tubular structures, which can sometimes take the look of a broadened kidney pelvis. CDUS provides clarity also here.

Nutcracker-phenomenon

The left renal vein draws over the aorta towards the vena cava. Sometimes, it can be compressed by the outgoing mesenteric superior artery in the sense of a nutcracker-phenomenon. This is followed by stasis of the left kidney and orthostatic proteinuria and can in extreme cases lead to renal vein thrombosis [(45)] [Fig. 77].

Figure 77 Nutcracker-phenomenon with dilated left renal vein:



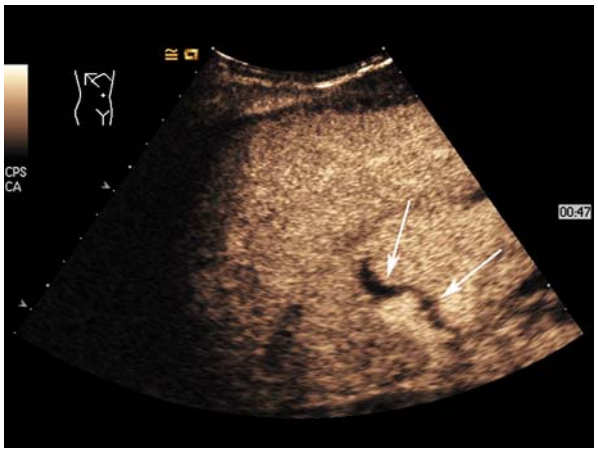
Renal vein thrombosis

With newborns, dehydration can leads to renal vein thrombosis. In cases of both-sided renal vein thrombosis, irreversible renal failure can occour. Due to the loss of antithrombotic proteins, a nephrotic syndrom can secondarily lead to renal vein thrombosis, so that 65% of all patients suffering from renal vein thrombosis also show a nephrotic syndrom. Thrombi in the renal veins are also found in malignant kidney diseases. Differentiation between a tumor thrombus and a blood clot can be very well distinguished by CEUS: A tumor is perfused, a clot isn't [(19; 46)].

Changes in renal trauma

Perirenal and intrarenal hematoma as well as renal pelvis hematoma are being observed. A rapid detection of traumatic **kidney ruptures** is crucial and very important [Fig. 78]. If the situation after using B-mode and CDUS still stays ambiguous, either a computer tomography or an CEUS should be carried out [(46)]. By these means, the rather indistinct changes of the kidney rupture can be safely detected. With blunt abdominal traumata, it is important to assess both the parenchymatous organs and the free liquid in the abdominal region. Besides kidney ruptures, ruptures of spleen and pancreatic contusion are quite common. Taking into account the high exposure to radiation during computertomography (a full investigation with contrast corresponds up to about 400 chest images!), CEUS should be prefered, particularly with children and adolescents - provided that the relevant departments are familiar with this method.

Figure 78 Kidney rupture in CEUS.



Upper urinary tract

Indications, examination technique, normal results

The examination of the upper urinary tracts consists of an assessment of renal sinus and ureter as it progresses from renal pelvis to bladder. This area is being superimposed ventrally by intestines and is, looked at it sonographically, therefore often covered by air. An prone position or a side position can help the examination to be successfull, especially the first few centimeters are being represented much clearer in an prone position. The outlet of the ureter can usually be made visible in this position, too, and in most cases of only slightly broadened ureters, a longer section up to the iliac can be represented. The examinaiton is being carried out either in cases of a corresponding clinic, i.e. renal colics, when suspecting urinary obstruction, or, when accidentally the proof of a change in the renal sinus has been found. It is important to asses the renal pelvis during the assessment of urinary tract infections. After the representation of the first section between renal pelvis and iliac in a prone position, the furter progression up to the crossing of pelvis vessels can be carried out in a side position or, in case of a lack of flatulence, in a back position. The last section of the ureter is often difficult to see. The best outcome is the representation of the ureteral orifice and a chase of the ureter retrogardly back to the crossing with the pelvis vessels.
The normal findings of the renal sinus contains mostly hyperechoic structures, the perirenal fat and the renal vessels and the non-extended pelvis. In children and slim people
echo-free dilated renal veins are observed and can lead to confusion with extensions of the collecting system [(13;31;45)]. The collecting system is assessed well in prone position and it can be depicted clearly in most cases. The ureter outlet is also clearly visible in this position. Only when a ureter is broadened 2cm after pyeloureteral transition by an enlargement of more than 5mm, we speak about a dilatation of the ureter.

Modifications of renal sinus

Anechoic renal sinus

Dialatation of the collecting system occur during urinary obstruction, with vesico-ureteral reflux and during pregnancy. All these forms show an anechoic representation of the collecting system. In case of an only slightly enlarged renal pelvis, it is particularly important to depict the outgoing ureter.

Norm variations

The renal sinus sonomorphologically shows a large variety of almost inhomogenuous up to homogenous occurence. The amount of sinus fat and how it behaves is hereby crucial. With children and slim adolescents holding little sinus fat, anechoic tubular structures (mostly broad renal veins), shares of collecting system and vessels appear. With elderly people, the sinus fat can also appear highly hypoechoic and therefore lead to a confusion with dilatation of collecting system (hydronephrosis).

Urinary obstruction

In case of a diatation of the collecting system form anechoic spaces which intertwine into each other and that dissociate from the remaning sinus. Depending on the level of obstruction, following classifications are being made:

- Intrarenal obstruction within tubuli and collecting ducts (eg with acute gouty kidney)
- Calyceal obstruction (eg with calyceal stenosis in tuberculosis)
- Obstruction at the pyeloureteral transition (eg with congenital stenosis)
- Ureteral obstruction (eg during passage of stones, necrotic papillae or blood clots)
- Infravesical obstruction (prostatic hyperplasia, urethral stenosis)

Furthermore, we distinguish between acute (hours to days), subacute (days to weeks) and chronic obstructions (months to years). An obstruction can occour either totally or partially. A total obstruction poses an urgent danger for the kidney. Obstructions located at the pyeloureteral transition or distally from it lead to dilatation of collecting system or hydronephrosis. A sonographical classification of hydronephrosis or urinary obstruction takes following parameters into account: changes of the renal sinus, changes of the parenchyma thickness and changes in the spectral curve of interlobar artery.

Acute congestion

With acute congestion, the volume of the affected kidney increases. There is a densification of echo in the kidney cortex and a significant increasement of the resistive index ($\Delta RI \geq 0{,}10$ compared to other kidney).

Chronic congestion

Chronic congestion leads to a gradual dilatation of the collecting system without any difference of the RI values. Following stages of hydronephrosis are being distinguished.

Figure 79 Stages of hydronephrosis: above: left normal, right degree I; second line: left degree II, right degree III; third line: left degree IV, right end-stage hydronephrosis.

Degree I: Slight expansion of pyelon while both renal pelvis and calices appear anechoic. 2cm from the pyeloureteral transition, the outgoing ureter should be at least 5mm wide.
Differential diagnosis for congestion stage I: A congestion of degree I can be simulated by **widened renal veins**. These tubular structures resemble the image of expanded collecting system [Fig. 80]. But renal veins don't leave caudally, but directly towards the large abdominal vessels. Including the use of CDUS clarifies the situation.
Another form variation of the renal pelvis is the **ampullary renal pelvis** which seems slightly more robust while calyceal necks and ureters don't appear expandend, so that it can be clearly distinguished form a degree I hydronephrosis [Fig. 81].

Figure 80 Prominent renal veins:

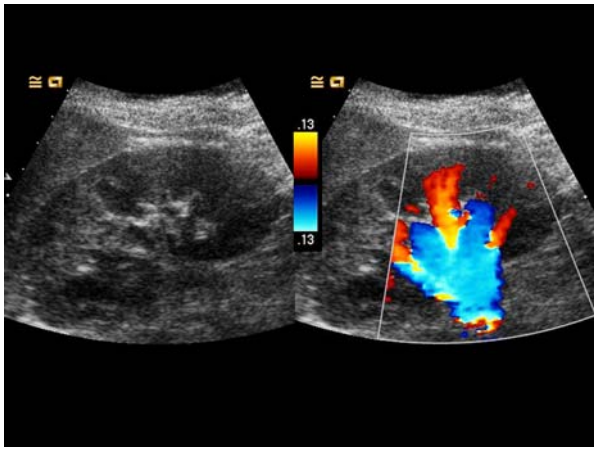
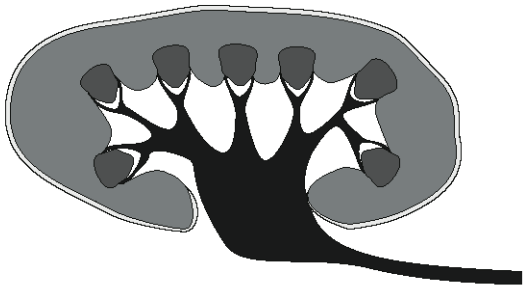


Figure 81 Ampullary renal pelvis.



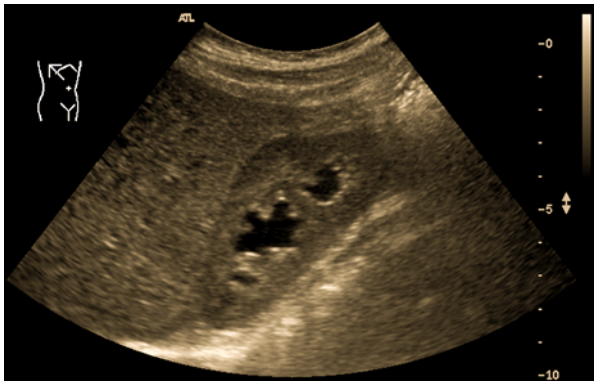
Degree II: Obvious expansion of pelvis, calyces including calyceal necks . The fornix angle can still lie below 90 degree, the kidney parenchyma shows normal thickness.
Degree III: pelvis, calyces including calyceal necks show obvious expansion and practically fill out the whole of the renal sinus. Often, parts of the pelvis already lie extrarenally. The parenchyma doesn't show any narrowing and the fornix angle lies above 90 degree.
Degree IV: Significant narrowing of the parenchyma with a fornix angle mostly above 120 degree. At the end of this progression lies the final stage without recognizable parenchyma.

Differential diagnosis of high grade obstruction

Megapolycalicosis

is being characterized by multiple calyces. This malformation goes back to extremely short mark pyramids that cannot be rendered visible in such images. Both the absence of mark pyramids and very many and enlarged calyces are highly specific for this diagnosis [Fig. 82]. There isn't any expansions of neither renal pelvis nor outgoing ureter.

Figure 82 Megapolycalicosis.



Parapelvic cysts

Another similar case of high grade hydronephrosis is formed by parapelvic cysts, which can often simulate hydronephrosis. While carefully analysing the image, following criteria lead to

a correct diagnosis: Absence of caliectasis with a missing connection of single cysts to a renal pelvis and absence of an expanded ureter [Fig. 83].

Figure 83 Parapelvic cysts .

Hypoechoic renal sinus

Opposed to the quite frequent depiction of hydronephrosis, hypoechoic changes of the renal sinus occur rather rarely.

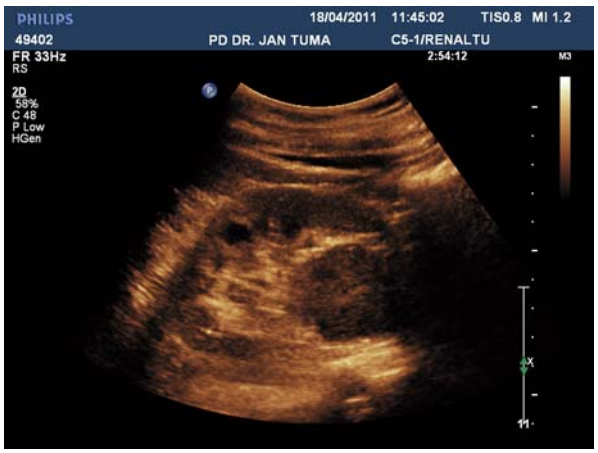
Hematoma

These hematoma, either post traumatic or as a complication after renal biopsy or lithotripsy, are being observed in the collecting system.

Urothelial carcinoma

Some tumors are located in the renal sinus. The far most frequent one is the urothelial carcinoma. It can be observed as a hypoechic formation in the renal sinus [Fig. 84]. Sometimes diagnosis is possible only after additional examinations like CEUS, CT or MRT-imaging.

Figure 84 Urothelial carcinoma:



Hyperechoic renal sinus

Kidney stones

From a certain size on, these strongly hyperechoic formations can be depicted already by means of B-mode. It is much more convenient to use CDUS while searching though. During examination, PRF should be set high and gain just below the artefact limit. Like this, a color artefact arises, the so-called twinkling: It reveals hard reflectors in the area of the sinus. These artefacts occur in more than 80% of even tiniest kidney stones. Yet the composition of the stones is of no importance and the artefacts are being produced by both calcium oxalate and calcium phosphate or uric acid. Twinkling artefacts aren't specific for a certain kind of stone and they are being observed as calcifications of vascular walls, of necrotic papillas in analgesic nephropathy, chronic pyelonephritis or during medullary nephrocalcinosis. Besides, one has to pay attention to the fact that twinkling can originate from intestinary gas, too.

Changes of ureter

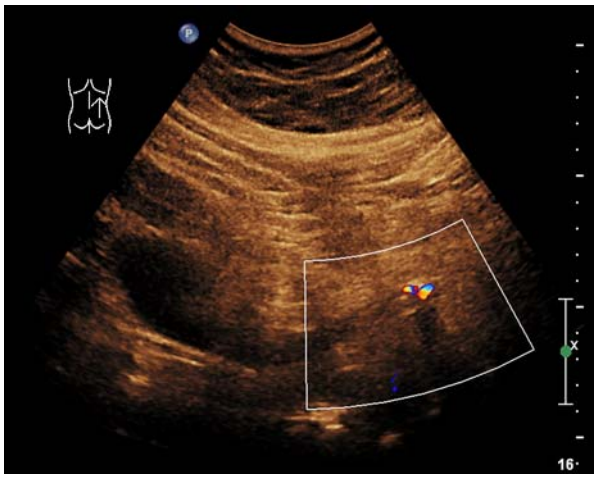
Anechoic changes of ureter

Hydroureter as a result of urinary obstruction

Renal colic

Kidney stones, blood clots during and pappilary necrosis passage are the most important causes for a renal colic. They provoke urinary obstruction and an expansion of ureter, renal pelvis and calyces. In many cases, the cause for urinary obstruction can be depicted sonographically. With typical renal colics, the existence of kidney stones can be proved with over 90% sensitivity [(7;16;28;29;34;40;43)]. Doing this, the phenomenon of twinkling is very useful, because tiny stones in the progression of the ureter are very difficult to depict. It is convenient to start examining in an prone position to scan the first few centimeters up to the iliac and then proceed to the prevesical spaces as well as the space at the crossing with the large vessels. The stone is often found due to the twinkling artefact and it is only afterwards, that the typical hyperechoic reflex gets represented, sometimes showing a very fine acoustic shadow or none at all.

Figure 85 Hydroureter with stone and twinkling artifact.



Differential diagnosis renal colic

In suspicion of a fresh embolism, it is sensible to immediatly proceed to the means of CEUS. The clinic resembles a renal colic. In case of a corresponding anamnesis (fresh atrial fibrillation, morbus embolicus) fresh embolism can be proved using CEUS and fibrinolytic therapy could be immediately started [(5;8;33)].

Painless (chronic) urinary obstruction with hydroureter

Next to the acute renal colic there are other conditions showing gradual narrowing of the ureter drains with both chronic inflammatory ureter modifications (ureter tuberculosis) and ureter tumors. Another cause of gradual axpansion can be very large myomas, exersizing outside pressure on the ureter. Tumors lying retroperitoneally can slowly lead to an obstruction of the ureter. As all these conditions proceed slowly, the hydroureter occurs mostly painless. In most cases, it is discovered accidental during systematic abdominal examinations.

Hydroureter with non-obstructive condition

Megaureter

This malformation of the ureter occours with primarily dilated ureters.

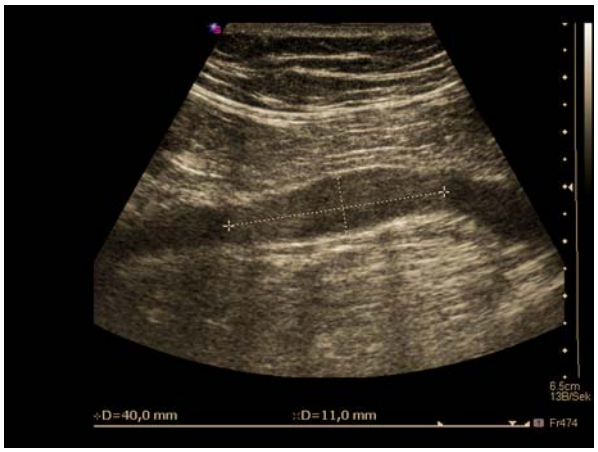
Vesicoureteral reflux

This is a malformation of the vesicoureteral transition with refluxing ureter. In cases of higher grade reflux not only the terminal but the whole of the ureter shows dilated. The diagnosis of vesicoureteral reflux is particularly important in children with recurrent urinary tract infections. Next to the method of voiding cystourethogram (VCUG) with the disadvantage of radiation exposure, sonographic voiding cystourethogram (SVCUG) with echo contrast agents has today become possible method. So far it is only carried out in a small number of clinic, but it should spread over the next years. Both sensitivity and specificity of this examination method is at least as precise as the method of VCUG [(9)].

Hypoechoic ureter changes

An expanded ureter can show anechoic with strong purulent infections. This case is called a Pyoureter, in which ureter catheters in the ureter can be followed as hypoechoic formations, too. Urothelal carcinoma can also be identified as hypoechoic and obstructive formations in the ureter [Fig. 86].

Figure 86 Ureter cancer.



Hyperechoic ureter changes

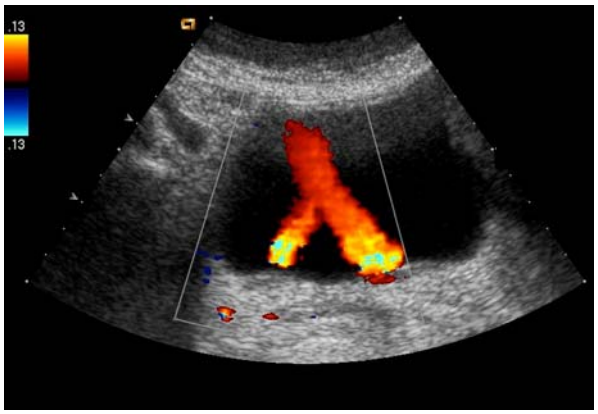
Ureter stones

The most important hyperechoic modification in the ureter are ureter stones. The procedure of following the outgoing ureter in prone position and in back position while crossing the pelvic vessels as well as the retrograde following of the ureter from ureteral orifice back to the crossing with the pelvis vessels has ben already described. With dilated ureters, the search is being carried out along the visible anechoic ureter. In case of unclear visibility, it is recommended to directly use CDUS for twinkling with high PRF setting and gain setting just below the artifact limit. Whether twinkling comes out or not is not depending on the composition of the stones, but obviously there is a correlation with the surface of reflection. It has been determined during examinations that the same, freely floating stone in the bladder can show a very strong twinkling artifact once and then a very weak one or none at all – depending on the position of the stone or the radiating Doppler signal, respectively.
Twinklings are being produced in more than 80% of ureteral stones, which is of great diagnostic significance. But twinkling artifacts are nonspecific. Twinkling caused by intestinal gas has to be distinguished from stones and passed papillar necrosis. Papillary necrosis keep a triangular form and are calcified only partially, so the distinction from a stone can succeed with B-mode, too.

Urinary jet

The diagnosis of urinary tract obstructions can be quite demanding – smaller kidney stones cause partly cases of only descrete hydronephrosis and no differences of RI, so further diagnostic methods come in helpful. Urinary jet forms a phenomenon of iradiating urine in the bladder. With normal drinking habits of approximately 2-3 litres a day, an occurence of ca two urinary jets/minute or ten urinary jets during five minutes has been observed, on both sides. A jet asymmetry is defined by < 2 jets / 5min on the ill side and > 5 jets / 5min on the other side. Next to the number of jets, the quality of jets can be assessed, too. A spectral analysis can give results on both maximal velocity Vmax and duration of the jets in sec. With ureters that are not completely obstructed, jets appear to run slower and to last longer, while shorter jets are being observed from time to time.

Figure 87 Urinary jet.



Summary of ureter colic diagnosis

With renal colics, a systematic procedure is recommended. First, the patient is examined in prone, assessing both collecting system and the outgoing ureters. In case of a successful verification of a stone in the area of the renal pelvis or calyx and therefore verification of the ureter stone as a cause of renal colic, the situation is under control. With an dilated ureter though and without any verification of a stone up to the iliac, a further examination takes place, while the patient is lying on his/her back. Here, the pelvic vessels are visited and the dilatation of the ureter gets controlled. The twinkling artifact of the stone, often placed just before the crossing, is searched for using CDUS. If this location doesn't show any direct results, the terminal ureter is examined retrogradually back from the bladder. As a rule, this is being done without CDUS in case of dilated ureters and using CDUS with rather slim ureters. With an adequate examination technique, the verification of a stone is successful in over 80% of all cases. The twinkling artefact is depicted often beforehand and it is then only later on, that a representation of the kidney stone is possible. Next to this very direkt verification of a stone, there are also some indirect signs for ureterolithiasis:

- non-glomerular hematuria
- hydronephrosis, requiring also minimal ureteral dilatation of 5mm, 2cm from the pyeloureteral transition
- Δ RI $\geq$ 0,10 on the affected side

A combination of two to three of these indirect signs define a probable stone. In a prospective study, probable and directly verified stones by means of sonography amounted to 98% sensitivity for ureter stones. As a further supplementing possibility, the asymmetry of urinary jets hints towards one-sided obstructions of the urinary tract. An appropriate examination technique can hereby help to reduce the number of stone clarifications carried out by the means of computed tomography, a very effective method holding the strenuous disadvantage of radiation exposure.

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