

# sonography of medical renal disease

BY FAYE TEMPLE AMS, CHIEF SONOGRAPHER, ST VINCENT'S HOSPITAL, VIC.

**“Medical renal disease” is a commonly, and perhaps poorly used term in imaging to encompass a broad range of renal parenchymal pathologies which have the potential to cause renal failure. The term alerts the renal physician to the possibility of renal failure, but as an imaging diagnosis, it provides few clues as to the precise cause or chronicity of the failure. This paper will discuss the signs and symptoms of renal impairment, the main sources of acute and chronic renal failure and particularly focus on the role of ultrasound in this context. The key sonographic features of renal parenchymal disease will be described and discussed.**

## Signs and Symptoms of Renal Impairment

Patients may be referred for renal ultrasound with various clinical presentations. They may be asymptomatic with abnormal blood or urine tests, have an acute deterioration of their renal function with no prior history of renal disease or they may have known renal impairment with sudden exacerbation of their condition. It is worth noting that many renal diseases are insidious in their onset with many of the symptoms being so vague or trivial that they are ignored until the renal impairment is very advanced. Many cases of renal disease are now detected by routine screening leading to earlier diagnosis and appropriate treatment.

Signs and symptoms that are commonly seen on a request for renal ultrasound include:

### Pain

Renal pain can be poorly localized but is usually in the loin, flank or hypochondrium. Renal pain is thought to be due to

distension of the fibrous renal capsule or the renal pelvis [1]. Pain can be caused by obstruction, infection, infarction, tumour, glomerulonephritis or renal cysts [1].

### Haematuria

Haematuria is the presence of blood in the urine. It may be microscopic where the blood is not visible to the naked eye or macroscopic where there is visible blood. It may include clots, which can be associated with stones, trauma or tumour [1]. Blood seen at the beginning or end of the urine stream usually indicates bleeding from the urethra or bladder [1]. Blood mixed throughout the stream is usually of renal or ureteral origin [1]. Painless haematuria is suggestive of glomerular bleeding or tumour anywhere in the renal tract [1]. Pain with haematuria is usually associated with stones, bleeding into cysts or severe infection [1].

### Polyuria

Polyuria is the passage of abnormally large quantities of urine. The normal volume is 0.7-2 litres/day. Three litres or more is considered polyuria [2].

### Oliguria

The passage of less than 400ml/day of urine [2].

### Anuria

The passage of no urine. It is most commonly related to obstruction [3].

### Hesitancy/poor stream

This suggests a bladder outlet obstruction (often caused by prostatomegaly in males) [1].

### Dysuria

Pain or burning during or after voiding. Most commonly occurs with infection [1].

### Proteinuria

Proteinuria is the presence of excess protein in the urine. Testing for proteinuria

is a part of routine physical examinations and has become a common presenting abnormality in asymptomatic patients with renal disease [1]. Persistent proteinuria is one of the earliest and most constant signs of damage to the kidney; however the absence of protein does not exclude renal disease [2]. The only direct symptom of proteinuria is urine frothing in the toilet because the protein lowers the surface tension of the water [1]. As does bile and toilet cleaner! Proteinuria can be due to increased permeability of the glomerulus or due to failed resorption of the proteins at the tubule [1]. Levels of protein in the urine are usually normal in the presence of obstruction [4].

### Reduced glomerular filtration rate (GFR)

GFR assesses the function of the kidney by measuring the amount of a given substance cleared from the blood by the kidney [2].

### Increased creatinine

This is an indirect and imprecise measure of the GFR as creatinine is mainly removed by glomerular filtration. Serum creatinine is not raised until the GFR is reduced by at least 50% [2].

### Sonographic Appearances Associated with Medical Renal Disease

Ultrasound is a commonly used first line investigation of renal impairment. It demonstrates the presence, size and appearance of the kidneys but most importantly it can demonstrate the presence of hydronephrosis, which may be due to obstruction. Obstruction is the cause of acute renal failure in about 10% of cases, but it must be identified as early as possible to allow timely intervention and treatment [5].

In the majority of cases where hydronephrosis is not demonstrated, useful

information is obtained about renal size, thickness and echogenicity of the cortex and the appearance of the pyramids. Changes in these appearances are generally non-specific but in combination with the clinical picture may provide valuable information. The specificity and sensitivity of ultrasound (and other forms of radiology) in distinguishing between the various causes of acute and chronic renal failure is generally low [5].

**Renal Size**

Renal volume is the best measure of renal size and it has been demonstrated to correlate well to GFR [3]. The formula used is that of the volume of an ellipsoid. (length x width x depth x 0.5) [3]. Adult kidneys demonstrate a mean volume of 134cm<sup>3</sup> on the right and 146cm<sup>3</sup> on the left [3]. Volume measurement is, however, notoriously inaccurate because three measurements are involved [3]. In practice renal length measurement has become the standard because it is simple, more accurate and correlates well with renal volume [3]. A measurement of 10-12cm is a useful range for normal renal size with the average renal length on the right side being demonstrated to be 10.9cm and on the left 11.2cm [3,6]. Discrepancy between the two kidneys is normally seen but the smaller kidney should not be less than 37% of the total renal mass [3]. Kidney size, however, varies with height so allowance should be made for this when assessing renal size. It has also been demonstrated that normal kidneys can vary in size by 5% so that differences of up to 1cm may not be significant [3].

Acute renal failure may demonstrate swelling of the parenchyma due to oedema or inflammatory change [5]. However, in the majority of cases, the size remains within normal limits. In chronic renal failure (CRF) the kidneys tend to reduce in size but the degree of reduction is very variable and correlation with the degree of impairment or the specific disease process is poor [5].

**Cortical Changes**

Normal ranges for the thickness of the cortex are not well established however 9.3 +/- 1.1mm has been suggested with a value of <7mm probably being abnormal [3]. The cortical thickness can be measured from the arcuate arteries to the capsule or from the outer border of the pyramids to the capsule [3]. The measurement from the arcuate arteries to the capsule is

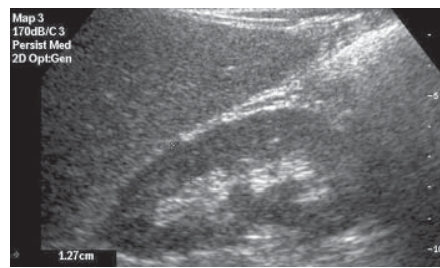


Fig 1. Measurement of cortical thickness from capsule to sinus fat.

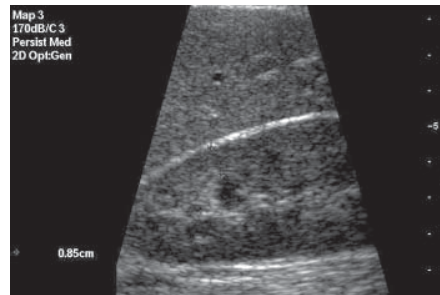


Fig 2. Measurement of cortical thickness from capsule to outer edge of pyramid.

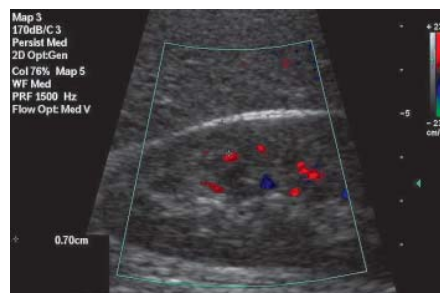


Fig 3. Measurement of cortical thickness from capsule to arcuate artery.

significantly less than the measurement from the pyramid to the capsule. The combined thickness of the cortex and the medulla (the distance between the sinus fat and the capsule) has been found to be 15-16mm but a normal range has not been established [3]. A measurement of <12mm is probably abnormal [3]. Figures 1 -3 show the appropriate way to measure renal cortex.

In acute renal failure (ARF) the cortex may increase in thickness and the echogenicity may be reduced or markedly increased. Echogenicity is assessed by comparison with the liver or spleen (provided they are normal) (fig 4). Comparison with the echogenicity of the renal pelvis is also useful. The cortical echogenicity may demonstrate no change even in the presence of severe impairment [5]. The cortex may demonstrate thinning in chronic renal failure (CRF) but again is not consistent with the degree of impairment or the causative pathology [5]. Platt et

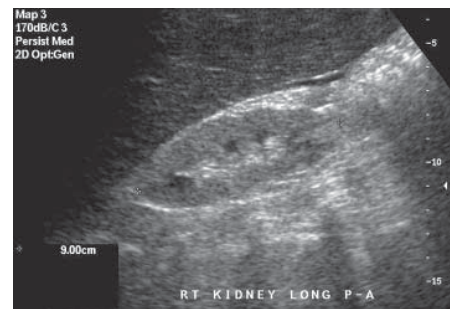


Fig 4. Increased cortical echogenicity when compared to the liver.



Fig 5. Increased cortical echogenicity with normal echogenicity seen in the pyramids.



Fig 6. Increased echogenicity demonstrated in medulla associated with acute renal failure.

al found that using the criterion of renal echogenicity greater than that of the liver gave a specificity of 96%; however the sensitivity was only 20% [7].

**Medullary Changes**

The medulla is easily seen and well defined in neonates with visibility declining with age. Initially in childhood, it is because the cortical echogenicity gradually reduces and then in adulthood, it is because the echogenicity of the pyramids increases probably due to medullary fibrosis [3].

The pyramids may demonstrate an apparent decrease in echogenicity when compared to increased cortical echogenicity (fig 5). The echogenicity of the pyramids may also increase so that they are indistinguishable from the cortex (fig 6). Again this is a non-specific finding

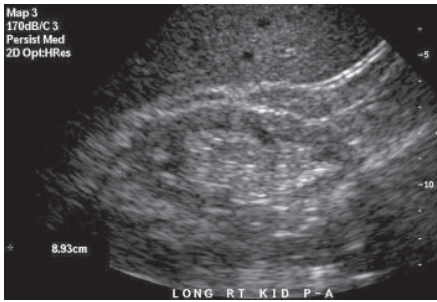


Fig 7. Decreased cortical thickness and overall renal size.

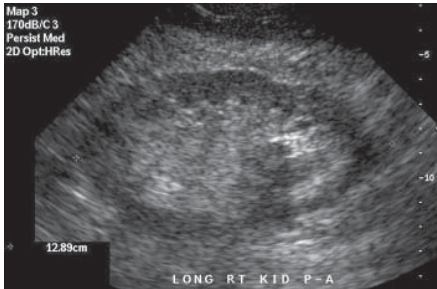


Fig 8. Decreased cortical thickness with maintenance of overall renal size.

with no correlation to type of pathology [5]. In acute conditions primarily affecting the glomeruli the oedematous pyramids can be increased in size [3]. The diuretic status of the kidney also affects the ultrasound appearance of the pyramids. In the presence of diuresis the pyramids may be more hypochoic and more prominent [3].

### Spectral Doppler

There is commonly an increase in resistive indices (RIs > 0.7) in parenchymal renal disease; again these changes are non-specific although acute tubular necrosis (ATN) has been demonstrated to produce significantly higher RIs than other disease processes [5]. A normal RI does not exclude intrinsic renal disease [5].

### Renal Failure: Acute and Chronic

Acute renal failure is characterized by a sudden decrease in renal function. It is usually, but not always, reversible [1]. In general, renal size is not affected in ARF [4]. In chronic renal failure there is persistent and irreversible reduction in renal function usually due to cortical atrophy and fibrosis [1,3]. In CRF the kidneys are usually reduced in size [4] (fig 7). However, it is important to recognize that in some circumstances renal size is relatively preserved in the presence of CRF [4] (fig 8). Acute on chronic renal failure is characterized by a sudden reduction in renal function in a patient with previously stable CRF [6].

If we accept the definition of medical renal disease that I have used in the introduction then many pathologies (including renal artery stenosis, renal vein thrombosis, infection and some cystic diseases) should be described here. However to simplify things I have chosen to describe only those processes that directly affect the parenchyma of the kidney.

### Acute Tubular Necrosis (ATN)

ATN is the most common cause of reversible ARF [1,5]. It can be due to nephrotoxins (drugs or poisons), or tubular ischaemia [1]. ATN usually causes acute oliguric renal failure of one to three weeks duration [1]. The ultrasound appearance of ATN depends on the precipitating cause. Ischaemic ATN will often demonstrate normal kidneys on ultrasound, although a thickened, hypochoic cortex may be seen [5,7]. ATN caused by toxins however, may demonstrate enlarged echogenic kidneys with enlarged, prominent pyramids [5,7]. Kidneys affected by ATN tend to display a significantly higher RI than other pathologies [5].

### Glomerulonephritis

Glomerulonephritis is the inflammation of the glomerulus that may be caused by an autoimmune reaction, infection, exposure to toxins or drugs or by infiltration of inflammatory cells in the renal parenchyma [6]. Patients usually present with haematuria, hypotension and abnormally high levels of urea in the blood [6]. Glomerulonephritis is the most common cause of CRF but only a small number of those affected by the disease progress to CRF [2].

Ultrasound appearances are variable. Renal size may be normal or enlarged and the echogenicity decreased, increased or normal [7]. The kidney may be rounded with the cortex appearing thickened and possibly compressing the sinus fat [3]. These varying appearances are of no value in determining cause or prognosis [5]. Acute glomerulonephritis may progress to chronic glomerulonephritis over a period of weeks or months after the acute attack. In chronic glomerulonephritis severe, symmetric parenchymal loss may be seen on ultrasound, sometimes with a loss of cortico-medullary differentiation demonstrated [1].

### Papillary Necrosis

This condition is caused when one of many possible agents damages the pyramids

leading to necrosis [5]. The pyramids have a poor blood supply which, if interrupted, may result in necrosis and subsequent sloughing of the damaged papillae [2].

Initially swollen pyramids may be present but are not commonly seen on ultrasound. If the papilla sloughs the affected calyx will have a clubbed appearance with cystic collections within the pyramid [5]. Subsequent calcifications of affected papillae may be visualized [5]. These sloughed papillae can cause ureteric obstruction and may be mistaken for calculi [5]. Initially renal size is unchanged but may reduce over time [5].

### Acute Interstitial Nephritis

Acute interstitial nephritis is caused by an acute inflammatory reaction to some drugs or various infections. It is treatable and usually reversible [3]. On ultrasound the kidneys may be normal or enlarged in size and the echogenicity may be normal or increased [5].

### Acute Cortical Necrosis

As the name implies this condition causes necrosis of all cell types within the cortex with sparing of the medulla [8]. It is associated with acute ischaemia of various causes. Interestingly there is sparing of a thin rim of cortex that is supplied by the capsular vessels [5]. On ultrasound the renal cortex is initially hypochoic with loss of cortico-medullary differentiation [5,7]. The rim of perfused cortex may be visualized on ultrasound [5].

### Amyloidosis

Amyloid is a disease that results in abnormal deposition of protein in various organs including the kidneys [6]. Renal amyloidosis is associated with extensive deposition of amyloid in the glomeruli, arterioles and interstitium of the kidney causing complete obliteration of the glomerulus [6]. Amyloidosis may be primary or secondary. Causes of secondary amyloidosis include tuberculosis, renal cell carcinoma, Hodgkin's disease, rheumatoid and multiple myeloma [7].

Ultrasound in the acute stage demonstrates symmetric enlargement with progressive reduction in size over time [5]. Enlarged amyloid kidneys may demonstrate increased echogenicity which is usually uniform but may be patchy [5]. The protein is primarily deposited in the cortex leaving the medulla relatively untouched [5]. Perirenal masses, pelvic



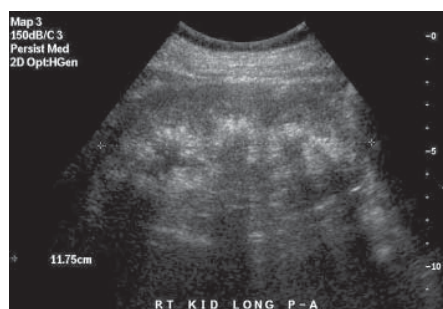


Fig 9. Medullary nephrocalcinosis seen in medullary sponge kidney.

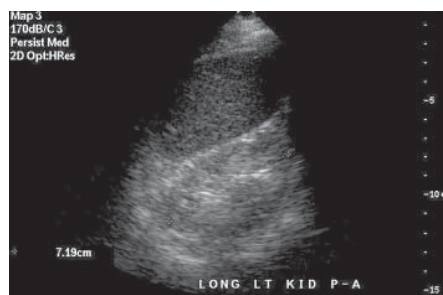


Fig 10. Small echogenic kidney seen in CRF.

masses and focal renal masses may be seen [7]. Amyloid may also affect the ureters and bladder where wall thickening or masses may be seen [7].

#### Diabetes Mellitus

Diabetes affects the kidneys primarily through vascular changes but also as a result of recurrent infections [1]. The ultrasound appearances are again variable with size being normal, increased or decreased although with chronic changes the kidney is reduced in size with normal or slightly increased echogenicity [7]. As the renal damage progresses the pyramids may become prominent probably due to increasing echogenicity of the cortex but the thickness of the cortex is usually maintained [3].

#### Connective Tissue Disorders (systemic lupus erythematosus (SLE), scleroderma and rheumatoid arthritis)

Connective tissue disorders may affect the kidney directly or as a result of drug therapy [5]. Sixty to 70% of patients with SLE develop renal involvement [8]. Glomerulonephritis and vasculitis may occur with non-specific changes in ultrasound appearance. Renal length may be normal or decreased and a variable increase in reflectivity is seen [7].

#### Medullary Sponge Kidney

Medullary sponge kidney is a disease of uncertain aetiology which causes the dilatation of the distal collecting tubules in the papillae [2]. Ultrasound may demonstrate nephrocalcinosis (within the pyramids) before it is demonstrated radiographically [5] (fig 9). The process usually involves the majority of pyramids but occasionally only two or three are affected [5].

#### Hypertensive Renal Disease

Hypertension can cause renal disease by damaging the interlobar arteries and afferent arterioles [5]. These changes cause minimal signs on ultrasound although the kidney size may be slightly reduced bilaterally [5].

These diseases and others may progress to CRF with the destruction of nephrons, resulting in the kidney being unable to maintain adequate function. As the kidneys become contracted they can be difficult to recognize on ultrasound as their echogenicity becomes very similar to perirenal fat. A careful examination in the appropriate area however can usually demonstrate the remaining renal tissue (fig 10).

#### Conclusion

In summary, ultrasound is generally sensitive to "medical renal disease" but while specificity is low it can offer a significant amount of information that may direct further investigation. It is important to remember that an absence of ultrasound findings does not exclude renal disease.

#### References

- Whitworth JA, Kincaid-Smith PS, Becker GJ. Clinical Nephrology in Medical Practice. Cambridge: Blackwell Scientific Publications; 1992.
  - Gabriel R. Renal Medicine. 3<sup>rd</sup> ed. Cambridge: Bailliere Tindall; 1988.
  - O'Neill WC. Atlas of Renal Sonography. Philadelphia: W. B. Saunders Co; 2001.
  - Freidland GW. Uroradiology, Vol 2. New York: Churchill Livingstone; 1983.
  - Meire HB, Dewbury KC, Cosgrove DO, Lowe, Foster, Shafi et al. Abdominal and General Sonography. 2<sup>nd</sup> ed. Vol 2. Exeter: Churchill Livingstone; 2001.
  - Kawamura DM. Diagnostic Medical Sonography. 2<sup>nd</sup> ed. Philadelphia: Lippincott; 1997.
  - Rumack CM, Wilson SR, Charboneau JW. Diagnostic Ultrasound. 2<sup>nd</sup> ed. Vol 1. St Louis (Missouri): Mosby; 1998.
  - Elkin, M. Radiology of the Urinary System. Boston: Little, Brown and Co; 1980.
- #### Additional Resources
- Middleton WD. Ultrasound: The Requisites. 2<sup>nd</sup> ed. New York: Mosby; 2004.
  - Bluth EI. Ultrasound, A Practical Approach to Clinical Problems. St Louis (Missouri): Thieme; 2000.

## 2006 RURAL AND REMOTE SONOGRAPHER SCHOLARSHIP

Are you a rural or remote Sonographer? AND

Have you conducted a research project recently? OR

Have you encountered an interesting/unusual case?

If the answer is YES . . . You are eligible to apply for the

### ASA 2006 Rural and Remote Sonographer Scholarship

Further information, conditions and an application form can be downloaded from the ASA website: [www.A-S-A.com.au](http://www.A-S-A.com.au), or obtained from the ASA National Office: 03 9585 2996.

Applications close Friday 28 October 2005.

