Renal transplant ultrasound: the nephrologist's perspective

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Abstract

One of the principal roles of a nephrologist is to closely monitor renal transplant allograft function and promptly evaluate any dysfunction. Renal transplant sonography has a major role in this assessment process given its ability to easily define renal transplant anatomy and surrounding structures. Abnormalities can be extrarenal or involve vascular, parenchymal and urological components of the graft and these can acutely or chronically influence graft function and survival. Procedural guidance as is required during allograft biopsy, as well as routine surveillance and screening for post transplant complications such as malignancy are also important applications of ultrasound in the management of renal transplant recipients. This article outlines key ultrasound findings and applications in renal transplantation from the clinician's perspective.

Keywords: allograft dysfunction, renal transplant, ultrasound.

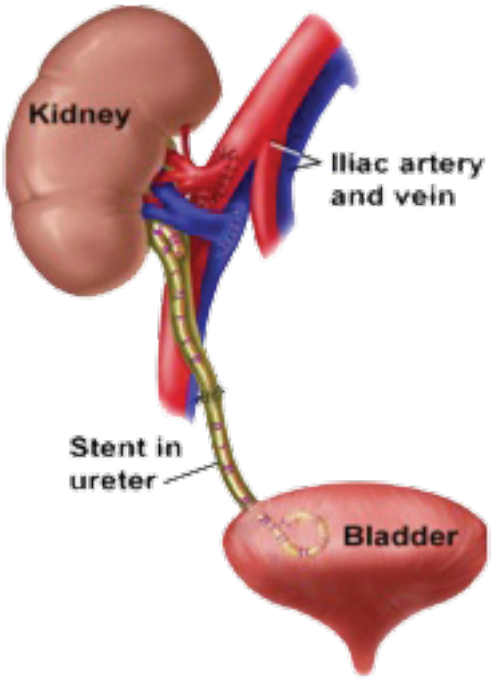

Figure 1: Basic anatomy of the kidney transplant: end-to-side anastamosis of donor renal artery with recipient external iliac artery; ureter of allograft attached to the patient's bladder; and ureteric stent insertion.[3]

Introduction

Renal Transplantation is the renal replacement therapy of choice for individuals with end stage kidney disease. It provides a much-improved quality of life, and morbidity and mortality benefit. Post-transplantation monitoring of allograft function is important, as detection of graft dysfunction requires prompt evaluation and management. Routine surveillance of graft anatomy and screening for post transplant malignancies are also important considerations in the long-term care of these patients.

Renal transplant ultrasound

Ultrasound is commonly used in the setting of renal transplantation. Particularly useful applications of this technology include: in the first few hours or days post renal transplantation; in routine post transplant surveillance; and very importantly when there is a need for evaluation of graft dysfunction.

Ultrasound is very convenient, rapid and relatively cheap. It can be done in real-time, provides multi-planar views, is non-invasive and has no ionising radiation involved. It avoids the need for iodinated contrast, thus avoiding the further risk of contrast nephropathy. Ultrasound evaluation of the renal allograft requires the use of B-mode, Colour Doppler (CDUS) and spectral Doppler technologies. B-mode ultrasound provides a morphologic evaluation, while colour

and spectral Doppler assesses blood flow.

Newer applications such as Contrast Enhanced Sonography (CES) and Shear Wave Elastography (SWE) are being investigated in the renal transplant setting. CES has been shown to be a useful technique that can provide improved quantitative analysis of the kidney allograft perfusion, and early prediction of chronic allograft nephropathy and loss of graft function.[1] SWE is in the earlier stages of investigation however there is some evidence that it may also be a predictor of transplant dysfunction.[2]

For these various beneficial reasons, ultrasound is often the first-line of evaluation of graft dysfunction.

Anatomy of renal transplantation

The basic anatomy[3] of a kidney transplant needs to be understood in order to appreciate the findings and importance of a renal transplant ultrasound (Figure 1). Traditionally, the kidney allograft lies in the extra-peritoneal space either in the right or left iliac fossa. Allograft renal arteries and veins are commonly anastomosed to their corresponding iliac arteries and veins. Either an end-to side or end-to-end anastomosis approach can be done, where donor renal artery is anastomosed to patient external iliac artery or internal iliac artery respectively. Donor vessels are occasionally duplex or may have natural non-pathological variations. The ureters of the allograft are then anastomosed to the anterolateral aspect of the bladder, with its delicate vascular supply originating from the renal hilum. Routinely in many transplant centres, a temporary ureteric stent is inserted at implantation to reduce the risk of leaks, obstruction or bleeding.

Renal transplant disorders by time of presentation

The main causes of renal allograft dysfunction are classically divided into time periods post transplantation (see Table 1): immediate (0–1 week post transplant), acute (1–12 weeks post transplant), subacute (3 months–1 year post transplant), and chronic (> 1 year post transplant). The main causes for allograft dysfunction can differ over this time course post transplantation. These are certainly not mutually exclusive at other time periods. The common causes of allograft dysfunction are further outlined in Table 1.

As will be highlighted, ultrasound is mainly helpful with the assessment of anatomical or gross structural causes of graft dysfunction. While there may be subtle changes on ultrasound in parenchymal renal disorders, these are usually non-specific.[4] Thus the value of performing an ultrasound often lies in the ability to exclude potentially reversible anatomical disorders, despite other underlying or contributory factors being present.

Table 1:

Common Renal Transplant Abnormalities by Time of Presentation	
1. Immediate (0–1 week)	**Vascular** Arterial stenosis Arterial thrombosis Venous stenosis Venous thrombosis **Urological** Leak Obstruction Collections Haematoma Urinoma **Parenchymal Disorder** Acute tubular necrosis (ATN) Calcineurin inhibitor (CNI) toxicity Acute rejection
2. Acute (1–12 weeks)	**Vascular** Arterial stenosis Arterial thrombosis Venous stenosis Venous thrombosis **Urological** Leak Obstruction Collections Urinoma Lymphocoele **Parenchymal Disorder** Calcineurin inhibitor (CNI) toxicity Acute Rejection
3. Subacute (3 months-1 year)	**Vascular** As above (not as common) **Urological** Obstruction Collections Lymphocoele **Parenchymal Disorder** Calcineurin inhibitor (CNI) toxicity Acute rejection Recurrence of primary disease Polyoma virus nephropathy
4. Chronic (>1 year)	**Vascular** As above (rare) **Urological** Obstruction **Collections** (rare) **Parenchymal Disorder** Calcineurin inhibitor (CNI) toxicity Chronic allograft nephropathy Recurrence of primary disease Polyoma virus nephropathy

Clinical applications for ultrasound in renal transplantation

A summary of the clinical applications for ultrasound in renal transplantation is presented in Table 2. A more detailed discussion of each of these follows.

Assessment of graft dysfunction

Broadly, graft dysfunction can be divided into non-structural and structural causes (see Table 2). Non-structural causes (or intrinsic parenchymal abnormalities) usually have non-specific findings on ultrasound.[5] When there is primary non-function or a change in allograft function, prompt evaluation is needed to aid in the management that will hopefully enable reduction and/or avoidance of graft failure or dysfunction.

Vascular assessment

Renal Artery Stenosis

Renal transplant artery stenosis (RTAS) is a common complication occurring especially during the first three years (most during the first year), with the incidence rate of 1–12%[5] (see Figure 2 and Clinical Case Correlation). This occurs mostly around the site of

Table 2:

Summary of Ultrasound Findings and Applications in Renal Transplantation		
	Common problems	**Possible Ultrasound Findings**
Vascular Problems	*Arterial Stenosis*	RI < 0.5 PSV > 300 cm/sec Renal artery: Iliac artery ratio > 2.0
	Venous Stenosis	Flow velocity increased / turbulence, Observed narrowing
	Thrombosis	Absence of Colour Flow Imaging
	Infarction	Often hypoechoic wedged area
	AVM & Pseudoaneurysms	Turbulent flow on Colour Doppler
Urological Disorders	*Hydronephrosis / Obstruction*	Dilated ureter or pelvicalyceal system
	Strictures	Narrowing of collecting system
	Renal stones	Hyperechoic lesion
	Leaks / Urinoma	Well define anechoic collection
Collections	*Haematoma*	Acute – hypoechoic collection Chronic – collection with varying areas of echogenicity
	Lymphocoele	Similar to haematoma, Wedge shaped, Consider clinical history
	Abscess	Variable from simple to complex collection
Parenchymal Disorders (non-specific)	*CNI toxicity*	Non-specific RI>0.8
	Rejection	
Surveillance for Malignancies	*Renal Cell Carcinoma (RCC)*	Complex cysts/ Solid lesions. Vascularity/ poor margins increase index of suspicion.
	Post Transplant Lymphoproliferative Disorder (PTLD)	
	Complex Cysts	
Renal Transplant biopsy	*Allograft dysfunction*	N/A (anatomical guidance for procedure)
	Routine biopsies	N/A (anatomical guidance for procedure)

anastomosis. Some factors that can contribute to this complication are: clamp-reperfusion injuries, suture technique, prolonged ischemia time, or rejection causing inflammatory fibrotic changes. Donor or recipient atherosclerosis may also play a role in this. The established criteria for making the diagnosis of RTAS is a renal artery peak systolic velocity (RA PSV) of > 300 cm/s.[6] Secondary criteria such as a peak systolic velocity (PSV) ratio between the transplant renal artery to external iliac artery (EIA) of > 2.0,[4] markedly reduced resistive index (RI) and abnormal intrarenal waveforms (acceleration time > 0.1) have all been reported to support this diagnosis.[7]

Early Transplant Period: An important role of the initial ultrasound examination is to identify anastomotic narrowing or kinking caused by the surgery. The experience of the authors is that a PSV > 300 cm/s in combination with a reduced RI (< 0.5) will increase the index of suspicion especially if there the early renal function is decreased.

RTAS surveillance: PSV values between 200–300 cm/s in this setting are a marker for RTAS and should be identified for ongoing surveillance. A PSV of > 300 cm/s will have a more acceptable specificity and should be used as the diagnostic threshold.[6] Additional findings such as: an increase in PSV since the previous examination; marked spectral broadening; significant PSV changes within the RA; and an abnormal distal tardus parvus waveform may increase the index of suspicion.

Careful examination with a B-mode and colour with higher frequency transducers is often the best method to appreciate the significance of these spectral Doppler changes.

Findings in this context should be carefully communicated in the report so that managing clinicians can correlate with clinical scenarios. If there are true suspicions of renal artery stenosis such as poor graft function, resistant hypertension or a bruit on auscultation, confirmation can be made based on other imaging modalities such as computer tomography (CT) or magnetic resonance (MR) angiography.

External iliac artery stenosis

The spectral Doppler waveform varies in the External Iliac Artery (EIA). Proximal to the anastomosis, it is low resistance with persisting flow through diastole as this part of the artery supplies the transplant. The distal artery has the typical peripheral artery triphasic waveform. The EIA proximal and distal to the anastomosis site can become stenosed secondary to clamp injury or atherosclerosis. A PSV> 200 cm/s or a focal increase in the

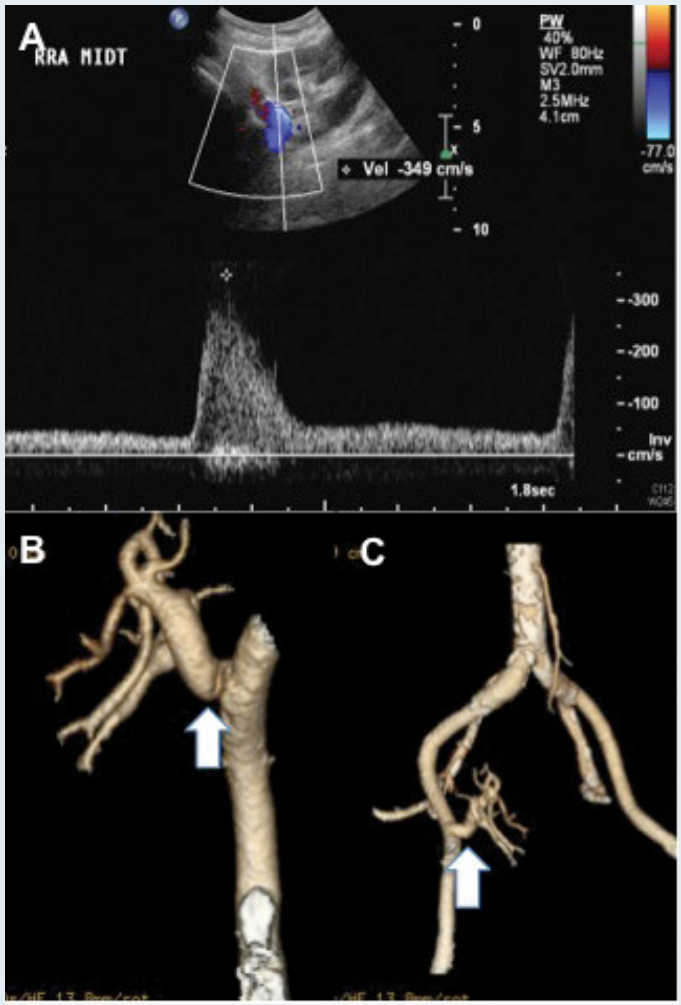

Figure 2: (A) Ultrasound examination at 15 months post transplantation showing a raised PSV of 349 cm/s at the origin of the renal artery. (B & C) CT angiography with 3-dimensional reconstruction showing a moderate kink at the origin/ proximal renal transplant artery. Despite the other findings the intrarenal resistive indices were relatively normal (0.76 at the midpole, 0.7 at the lower pole and 0.8 at the upper pole) (B).

Clinical case correlation

Two months following renal transplantation, a 68-year-old female was noted to be severely hypertensive, with systolic blood pressure measure of 188 mmHg. Renal allograft function was stable with a serum creatinine 80 mmol/L. A renal transplant ultrasound showed a PSV of 352 cm/s, consistent with significant renal transplant artery stenosis. Given the velocity was within the moderate (rather than severe) range, the patient's blood pressure was monitored for another 12 months and she was managed with additional antihypertensive medications. Repeat ultrasound at month 15 post renal transplant again demonstrated high velocities (349 cm/s) (see Figure 2A). Given the persistent hypertension, CT angiogram was performed showing a 50% post surgical kink of the renal transplant artery (see Figure 2B and C). The stenosis was not considered severe enough for intervention, and serial monitoring with renal transplant ultrasound continued.

artery of 2:1 are evidence of a >50% stenosis in the EIA however because of the changed anatomy and additional haemodynamics of the transplant, these are less reliable than the non-transplant leg arterial examination. Again, careful examination with a B-mode and colour with higher frequency transducers is often the best method to appreciate the significance of these spectral Doppler changes.

Renal vein stenosis

The renal vein typically has low-velocity phasic flow and is very uncommonly affected by marked luminal reduction or stenosis. This can be due to external compression by perinephric fluid collections or masses, but could also occur due to luminal fibrosis or anatomic kinking. It can be a transient finding at the day 1 examination due to compression from haematoma. A focal marked increase in velocity with associated marked turbulence and supporting B-mode and colour Doppler evidence is suggestive of this finding.[5] An increase in the intrarenal RI may be detected if this is severe. CT or MR venography could be further utilised to evaluate this ultrasound finding if it is highly suspicious, enabling venous angioplasty or stenting of luminal fibrosis.

Thrombosis

Thrombosis of the main renal artery is relatively uncommon but has a reported incidence of 0.5–2%, especially occurring during the early post transplant period (first week).[8] A more common finding is the thrombosis of a small accessory renal artery that has been difficult to anastomose during surgery. Major and minor branches of the renal artery can be similarly but less commonly affected (see Figure 3 and Clinical Case Correlation). Colour Doppler ultrasound will show no colour flows in areas of the infarcted kidney. These are classically wedged shaped, involve the upper or lower pole and in the acute phase, are hypoechoic when compared to the adjacent cortex.[5]

Renal vein thrombosis is also mostly an early (during the first week) complication that occurs infrequently (but more commonly compared to renal artery thrombosis) in 0.3-3% of cases.[4] Ultrasound will demonstrate no venous Doppler flows, with mostly enlarged and oedematous kidney parenchyma.[9] Occasionally, a reversed diastolic flow can be observed in the intrarenal arteries and the main renal artery, which can be suggestive of renal vein thrombosis however is non-specific among a range of anomalies.[10]

Detection of vascular thrombosis by ultrasound usually does not need other confirmatory imaging modalities. Prompt salvation of the allograft may be possible in some circumstances by either thrombectomy or thrombolysis / anticoagulation. In the majority of cases however, the allograft is unfortunately lost.

Other vascular complications

Other findings that can be detected by ultrasound are vascular

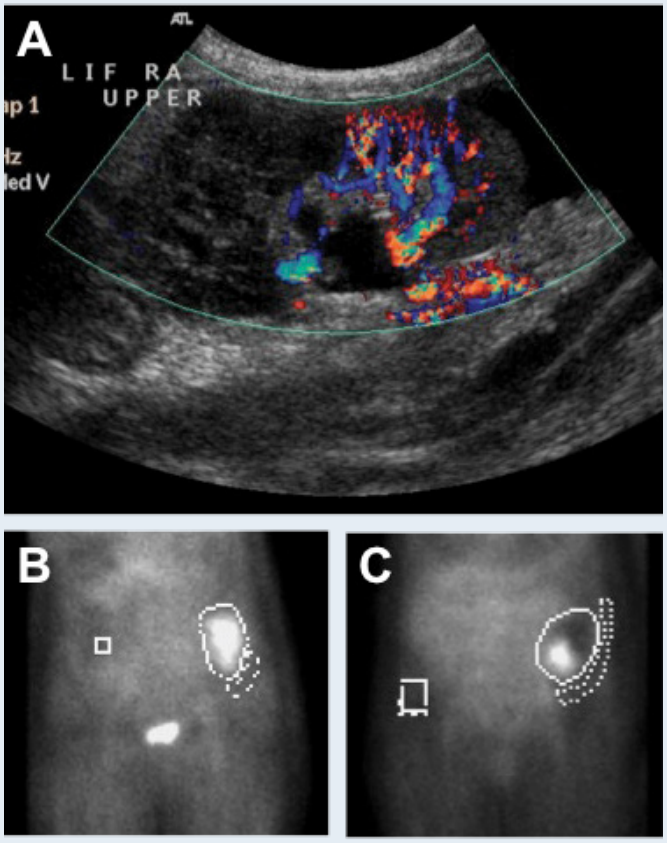

Figure 3: (A) Day 5 renal transplant ultrasound showing absent flow at the upper pole of the transplanted kidney. (B) Day 1 renal MAG3 scan (performed prior to the thrombosis of the arterial branch) with uptake of tracer throughout all poles of the transplanted kidney. (C) Day 5 Renal MAG3 scan showing absent uptake of tracer at the upper pole of the transplanted kidney.

Clinical case correlation

A 50-year-old lady with end stage kidney disease due to diabetic nephropathy received a deceased-donor renal transplant. The donor was noted to have dual renal arteries, with a single vein and ureter. The serum creatinine quickly improved from 605 µmol/L on Day 1 to 109 µmol/L on Day 4. However on Day 5, it was noted that the serum creatinine suddenly increased to 225 µmol/L. The patient was haemodynamically stable and clinically euvolaemic with a good urine output. The serum CNI (tacrolimus) level was appropriate and not excessively elevated. There was some mild tenderness over the allograft site. Renal transplant ultrasound was organised to further evaluate the graft dysfunction. This revealed absent flow at the upper pole of the transplanted kidney suggestive of an area of infarction secondary to a post surgical occlusion of an accessory renal artery (see Figure 3A). A renal MAG3 study (see Figure 3B and C demonstrated no uptake in the region of infarction.

kinking, dissection, arteriovenous malformations (AVM) and pseudoaneurysms. AVM and pseudoaneurysms are typically acquired post biopsy (see Figure 4 and Clinical Case Correlation) although they can present spontaneously.[8] They appear as high-velocity, markedly turbulent flow on colour Doppler imaging. The focal mosaic intrarenal colour pattern is pathognomonic. These can resolve without intervention however may occasionally require embolisation.

Various vascular complications can arise in donor kidneys with multiple renal arteries. Most often a single branch of this vascular supply may be stenosed, thrombosed or ligated. This will lead to infarction of the area that the affected polar branch supplies.

Urological Considerations

The ureter of the renal allograft is anastomosed to the anterolateral portion of the bladder. The vascular supply to the ureter originates from the renal hilum, and is therefore very delicate. The complications that can occur here are obstruction and urinary leaks. As noted, at surgery a stent is often inserted to protect the ureter for the first few weeks. This will be easily identified by ultrasound in the renal hilum and the bladder.

Ureteric obstruction occurs in 2–5% of cases.[4] Frequently, this is due to strictures or stenosis at the anastomosis to the bladder. Ninety percent occur at the distal third of the ureter.[8] Other causes of obstruction are ureteric calculi, or external luminal compression by collections. Hydronephrosis on ultrasound appears as a dilated urine-filled renal pelvis, occasionally with findings of dilated calyces and visible dilated ureter. With careful imaging, the normal transplant ureter can often be imaged throughout its length arising from the kidney and followed to the neo-ureterostomy site. When the ureter is dilated, it can be tracked to the point of obstruction. This may appear as a narrowing of the ureter with or without the cause being evident (calculi, kinking, compression, stenosis, or strictures) (see Figure 5 and Clinical Case Correlation). A mid-frequency linear transducer can often assist with defining the source of obstruction. The ultrasound finding of hydronephrosis is important although not always due to true obstruction. Non-obstructive mild dilatation of the collecting system is noted in a number of transplanted kidneys. Correlation with the clinical scenario (e.g. change in graft function) and comparison with further investigations such as nuclear diethylene-triamine-penta-acetic acid (DTPA) or mercaptoacetyltriglycine (MAG3) scan with diuretic challenge is often warranted to better demonstrate the functional significance of the findings.

Leaks originating from the ureter leading to urinoma are mostly an early complication post renal transplant surgery.[8] Rarely, leaks can occur at the calyceal or upper proximal ureter when ischaemia / infarction or ligation of an accessory renal artery, polar artery or post-biopsy complication occurs (see Figure 6 and Clinical Case Correlation). Ultrasound reveals a well-defined anechoic collection that has a high concentration of creatinine when drained.

Collections

Haematoma/ Seroma

During the immediate post-surgical period or following a procedural intervention such as a biopsy or drainage procedure, haematoma is the most common collection. These may be clinically significant in up to 8% of cases,[4] however they are most

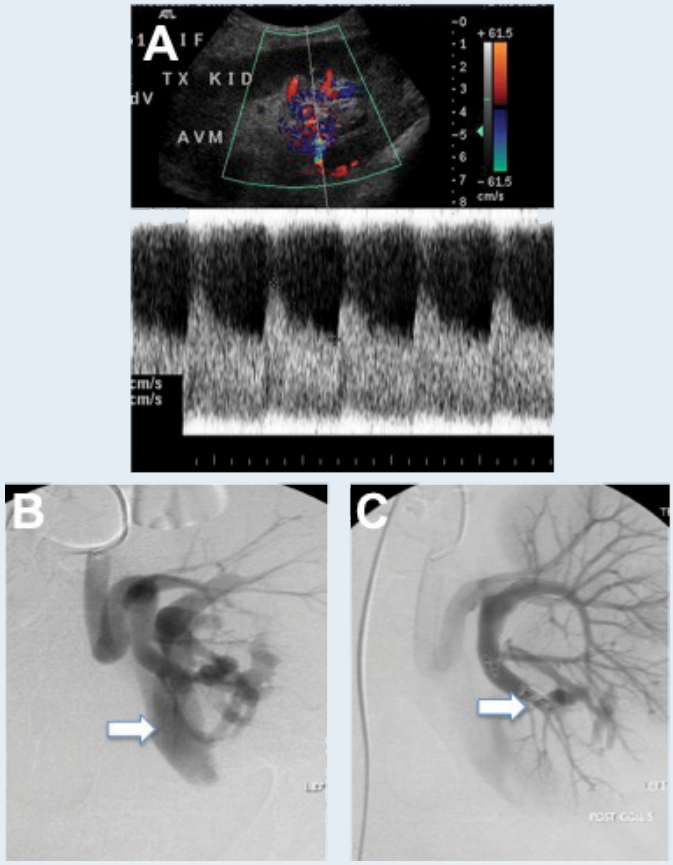

Figure 4: (A) Renal doppler ultrasound with an area of abnormal vascularity in the lower pole of the transplanted kidney with high velocity waveforms measuring between 240 and 340 cm/sec. (B) CT Angiogram showing arteriovenous malformation (AVM). (C) CT Angiogram post embolisation of AVM.

Clinical case correlation

A 28-year-old gentleman received a living donor kidney transplant from his father. The creatinine improved quickly from 734 µmol/L on day 1 to a baseline of 100 µol/L from day 3 onwards. He was reviewed on day 22 noting a creatinine rise to 170 µol/L. A biopsy was performed which revealed mild acute rejection which was treated with pulse intravenous steroids. Unfortunately he suffered frank haematuria immediately following the biopsy. Urgent renal transplant ultrasound was performed and this revealed an area of abnormal vascularity at the lower pole of the allograft consistent with an AVM arising as a complication of the renal biopsy (see Figure 4A). Colour flow imaging demonstrated aliasing at the arterial venous connection. CT angiogram with embolisation of the AVM was performed (see Figure 4B and C) with resolution of the haematuria and improvement in allograft function. Creatinine subsequently fell to 120 µmol/L.

often small and benign. When large and significant, they can cause pathological external compression to structures nearby or acute blood loss needing surgical intervention. In the acute phase, colour Doppler ultrasound should be performed quickly to try and identify active bleeding as a turbulent jet flow. The acute bleed usually appears hypoechoic. Haematomas that are more chronic usually form septa with both hypoechoic and echoic areas. Haematomas tend to follow tissue planes.

Lymphocoele

Lymphocoeles can occur anytime during the post-transplantation course. They often present with graft tenderness, swelling or dysfunction, but are also frequently asymptomatic. A lymphocoele may appear similar to a haematoma at the ultrasound examination. They can be both anechoic and have septa. They both have no blood flow on colour Doppler imaging. Lymphocoeles can sometimes be differentiated by their tendency to occur in the region of the major vessels. They also tend to accumulate, enabling detection when comparison is made with the baseline imaging. Fluid drainage from the collection is ultimately necessary to make a definitive diagnosis. Although potentially drainable percutaneously, lymphocoeles are notorious for reaccumulating whereby surgical intervention with marsupialisation is often needed.[4]

Abscess

Infection in an immunosuppressed patient is one of the main challenges a clinician faces throughout the course of managing a post-transplant recipient. Abscess formation surrounding the transplanted kidney (subcapsular or perinephric) can be evaluated by ultrasound, especially when a patient presents with signs of sepsis. Abscesses commonly appear as a complex collection on grayscale ultrasound. Similar to other collections mentioned, a definitive diagnosis by fluid drainage from the collection, either by ultrasound or CT guidance is often needed.

Urinoma

Urinomas have been described in the urologic complications. They are typically anechoic and located in the region of the ureter or bladder. They can be painful especially when the urine makes contact with the peritoneal membrane.

Rejection and parenchymal disorders

Ultrasound findings of parenchymal abnormalities may be subtle and non-specific. Usually the ultrasound appearance is actually quite normal. B-mode markers for dysfunction include increased / decreased echogenicity, cortical thinning or swelling / oedema, increased peri-medullary echogenicity, loss of corticomedullary differentiation and urothelial wall thickening. Colour Doppler ultrasound may show focal decrease in colour flow. Spectral Doppler has long been thought to be a better predictor. High quality waveforms with fast sweep speeds, no venous overlay and limited transducer pressure are essential to ensure reproducibility. An elevated RI (> 0.8) or an increasing RI over consecutive examinations in the setting of acute allograft rejection, chronic allograft nephropathy, CNI toxicity or ATN can be useful triggers for additional surveillance or further investigations such as a renal biopsy.[8,11] There have been several studies evaluating the importance of elevated RI or PSV in predicting long-term graft

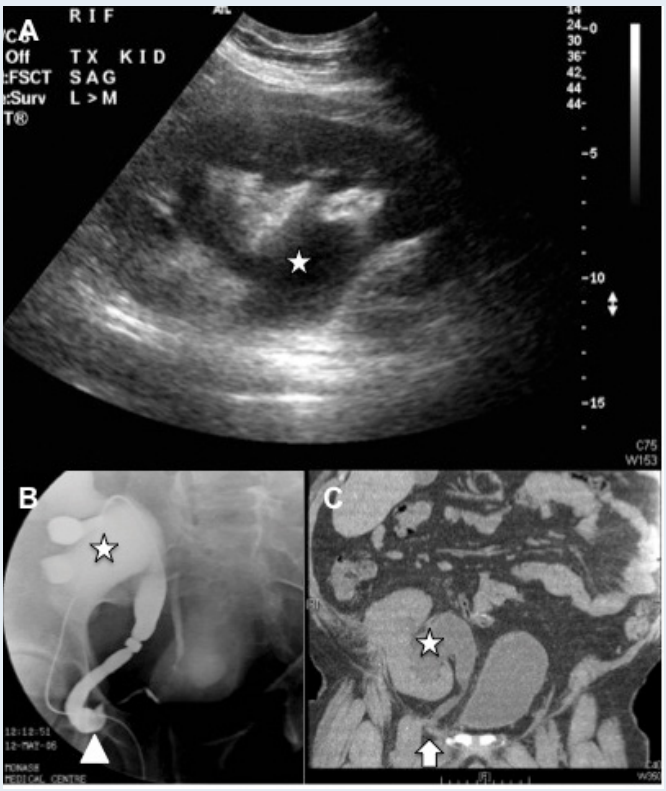

Figure 5: (A) B mode ultrasound showing an enlarged kidney with moderate hydronephrosis. (B) Percutaneous nephrostogram showing dilatation of the proximal half of the ureter terminating at the region of the right groin. (C) Coronal CT showing herniation of the ureter into the inguinal canal.

Clinical case correlation

A 38-year-old obese gentleman presented with symptoms of a urinary tract infection and had tenderness over the renal allograft site, three years following renal transplantation. Serum creatinine rose from 131 mmol/L to 223 mmol/L. Ultrasound examination showed an enlarged kidney measuring 16.6 cm with moderate hydronephrosis (see Figure 5a). The distal ureter was tracked to the lower pelvis, however a source of stricture could not be identified. Percutaneous nephrostogram was performed however this also showed a stricture of the ureter without a defined cause (see Figure 5b). On CT, the ureter was seen to herniate into the inguinal canal, causing obstruction (see Figure 5c).

survival and outcome, however this remains controversial. While nephrologists often refer to the RI they will also place significant weight on the clinical context.

Parenchymal abnormality will also be seen when there is pyelonephritis of the transplant. Focal increase in echogenicity with loss of colour flow are the most specific signs on ultrasound. Intrarenal gas is occasionally seen especially post transplant surgery. This is usually secondary to bladder catheterisation however may rarely be due to pyelonephritis.

Renal calculi are unusually encountered in the parenchymal examination of a renal allograft. Twinkle artefact demonstrated as a focal area of aliasing may help to confirm this finding.

Surveillance for malignancy

The incidence of renal cell carcinoma (RCC) and urothelial malignancy in the allograft or native kidney is much higher compared to that of the general population. It most often occurs in the native kidney. The high incidence of native RCC is likely contributed to by time on dialysis and acquired cystic kidney disease,[12] amongst the other risk factors such as smoking,[13] obesity, and immunosuppressive use.[14] Screening of transplanted and native kidneys by 1–3 yearly post transplant surveillance ultrasound has therefore been widely recommended.[15]

The reported incidence of both *de novo* allograft and native renal cell carcinoma varies in the literature. Melchior[16] and colleagues reported a prevalence of native RCC developing in 31% of 802 patients who underwent renal transplantation and regular screening post transplantation for urological malignancies. Three patients (10.3%) of their cohort developed RCC in the allograft kidney. Median time to the development of *de novo* native and allograft RCC post kidney transplantation was 47 months (range 24–112 months) and 36 months (range 3–38 months) respectively. This observation warrants long-term validation by others as this high incidence of asymptomatic malignancy may ultimately lead to increased morbidity and mortality, as has been suggested in multiple other reports. On ultrasound, malignant neoplasia will present as complex cysts or solid lesions and require further evaluation.[17,18] Vascularity within the lesion and poorly defined margins will increase the level of suspicion. An additional clue to the presence of a urothelial lesion may be the presence of hydronephrosis in the native kidney secondary to malignant obstruction. Identification of these lesions can be difficult because the kidneys will be highly affected by other pathology that can obscure smaller lesions. For e.g. patients with adult polycystic kidney disease who have multiple complex cysts making assessment almost impossible. Nonetheless, all surveillance examinations should include a careful examination for these lesions.

As indicated, renal cysts can also develop ranging from simple to complex based on the Bosniak classification,[19] where higher grade lesions suggest a greater likelihood of malignant transformation.

Surveillance should include the bladder as the incidence of bladder transitional cell carcinoma is also higher.[20]

Renal transplant biopsy

In many cases, renal transplant biopsy is needed for further evaluation of graft dysfunction. Some centres practice routine surveillance renal biopsies for example at 3 months and 12 months post-renal transplant.

Renal transplant and native renal biopsies are usually done under ultrasound guidance using local anaesthetic. Transplant biopsies are mostly done in a supine position, due to anatomy of the renal allograft. The benefit is that it can be done in real

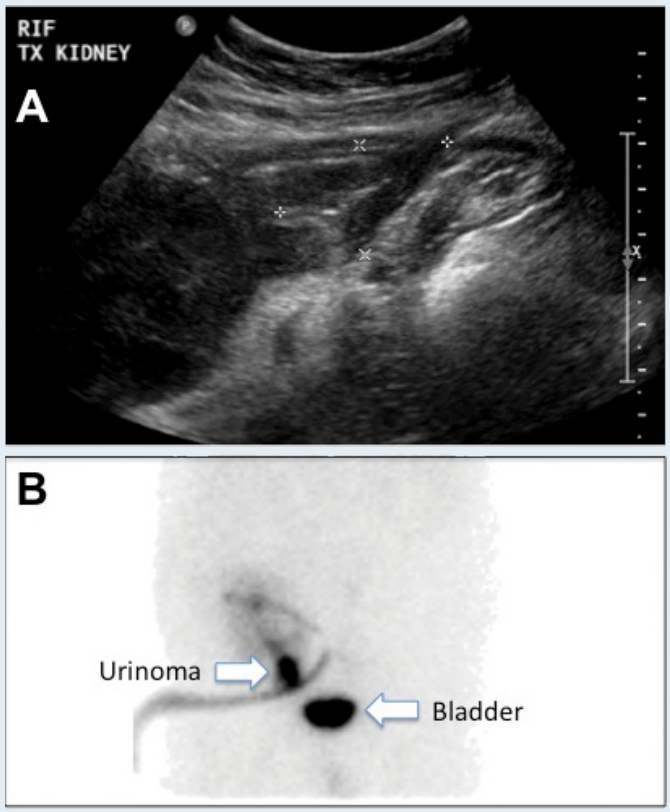

Figure 6: (A) Renal transplant ultrasound revealing a complex perinephric collection. (B) Renal MAG3 scan showing an active connection between the perinephric collection which is secondary to a urine leak and the urinary tract with the tracer seen through the inserted drain tube catheter.

Clinical Case Correlation

A 19-year old gentleman with end stage kidney disease due to reflux nephropathy received a living donor kidney transplant from his father. Allograft function was stable with a serum creatinine of 85 µmol/L as soon as 4 days post-renal transplant. Protocol renal biopsy was performed 3 months post-renal transplant. At Day 1 after the renal biopsy, his serum creatinine increased to 110 µmol/L. On examination, there was a fluctuant mass measuring approximately 2 x 3 cm over his allograft site at the right iliac fossa. Renal transplant ultrasound was performed which revealed new complex echogenic perinephric collection measuring 3.9 x 2.4 x 4.2 cm (see Figure 6A). A renal MAG3 scan showed an active connection between the perinephric collection and the urinary tract, suggesting the presence of a urinoma. A pigtail catheter was inserted to drain the collection (see Figure 6B) and this confirmed the presence of urine in the collection. Further complex surgical management was required to correct this abnormality.

time, and assists the operator in localisation of the biopsy site. The ultrasound transducer can be placed transversely or longitudinally, with the renal biopsy needle angled orthogonal to this plane to allow for optimal visualisation of the kidney and position / localisation of the biopsy needle.

Potential complications that may arise from an ultrasound guided renal biopsy are estimated to be 3.5% and include macroscopic haematuria with 0.9% requiring blood transfusion; 0.6% intervention with angiography for embolisation of bleeding point; 0.01% bleeding needing nephrectomy; and 0.02% associated with death.[21] AVM and pseudoaneuryms (as mentioned above) are usually post-biopsy complications arising in as many as 10–20% of cases.[4,9] Very rarely, perforation of structures nearby such as liver, bowel, pancreas, spleen and ureters can occur.

Occasionally, technical difficulties arise while performing the renal biopsy where the procedure will need to be postponed or cancelled for further evaluation or management. These are for example: multiple or large renal cysts; hydronephrosis; and AVM or pseudoaneuryms. Obese patients are often difficult to biopsy under ultrasound guidance as the biopsy needle is often difficult to visualise. CT guided renal biopsy is an option when there are technical difficulties. Of note, areas of infarction due to vessel thrombosis will need to be avoided when performing a renal transplant biopsy to obtain the appropriate renal parenchyma for pathological evaluation.

In conclusion, from the nephrologist's perspective, renal transplant ultrasonography is crucial for the ongoing care and management of the renal transplant recipient. The typical applications of this procedure in the renal transplant setting have been described, from the perspective of both the time period post-transplant as well as by the spectrum of specific underlying disorders. Renal transplantation is the renal replacement therapy of choice when feasible in patients with end stage kidney disease. Regular monitoring and prompt evaluation of allograft dysfunction is essential in order to optimise patient and allograft outcomes.

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