What Not To Miss When Imaging The Transplanted Kidney: An Imaging Guide

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Learning objectives

- To describe the most common complications of renal transplantation in the acute, subacute and chronic settings.
- To illustrate the imaging findings of these complications with a variety of imaging modalities.
- To demonstrate the role of doppler ultrasound including the 'resistive index' in the evaluation of the transplant kidney.

Background

Over the years renal transplantation has become the treatment of choice for the majority of individuals with end-stage renal failure. Improved surgical techniques and developments in immunosuppression and tissue typing have been at the forefront of better outcomes in these patients.[1]

Kidney transplants are prone to a variety of complications ranging from the vascular complications of the immediate post-operative period to the long-term sequelae associated with chronic immunosuppression. These complications have a substantial impact on morbidity and mortality. Urologic complications occur in 4%-8% of patients, and vascular complications in approximately 1%-2%.[2] Ultrasound is the principal means of imaging to evaluate the transplanted kidney. Its availability, lack of ionising radiation, ability to assess perfusion and non-nephrotoxicity make it the most effective primary imaging tool.[3,4] In addition to sonographic techniques, alternative imaging modalities sometimes provide valuable additional information.

This exhibit provides an overview of the variety of complications that can be encountered in the transplanted kidney. The salient radiological features will be described. Parallel to this, the value of the resistive index will be demonstrated.

Imaging findings OR Procedure details

RESISTIVE INDEX

Colour doppler imaging allows rapid assessment of global perfusion to the transplanted kidney (See Figure 1). The transplant kidney normally has a low resistance arterial vascular bed. On Doppler ultrasound imaging, this is characterised by streamlined systolic flow and continuous forward flow in diastole (See Figure 2). [9]
The resistive index (RI) is a measure of the renal vascular resistance to flow within the vascular bed. The RI is calculated from the Doppler arterial waveform. RI = peak systolic velocity - end diastolic velocity / peak systolic velocity. RIs <0.7-0.8 are considered normal, whereas RIs >0.9 are a strong indicator of a failing renal transplant. [9,10]

The Doppler arterial waveform depends on the interaction of a variety of factors. It therefore follows that the differential diagnosis for a raised RI includes transplant rejection, obstruction, ATN, pyelonephritis, severe hypotension, or an acute vascular event. RIs must therefore be taken in the context of the clinical scenario as, in isolation, it represents a non-specific marker of renal transplant dysfunction.[9]

**PERINEPHRIC TRANSPLANT FLUID COLLECTIONS**

These occur in up to 50% of kidney transplantations.

**Lymphcoele**

Lymphoceles occur due to leakage of lymph from surgically disrupted lymphatic channels along the iliac chain or from the lymphatics of the transplanted kidney. They have a reported prevalence of 0.5-20%[1,4]. Their onset is usually within 4-8 weeks post-transplantation but can occur at any time. Lymphoceles are largely asymptomatic but are the most common perinephric collection to cause hydronephrosis due to pressure effect which can also cause lower limb, scrotal or labial oedema.

Lymphoceles are usually anechoic collections and may have septations. As with other fluid collections, they have the potential to become infected - when they do, they are more likely to contain thick septations and internal debris. Symptomatic lymphoceles can be percutaneously drained although they have a tendency to recur.

**Haematoma**

These are common in the immediate postoperative period. They can also be seen in the context of trauma to the post transplant kidney, and this includes biopsy. They have a variable appearance on ultrasound, ranging from echogenic collections acutely to anechoic collections several days or weeks, and may contain septations. They usually resolve within a few weeks. See Figure 3.

**Urinoma**
Urine extravasation occurs due to distal ureteral necrosis, leakage from the ureteroneocystostomy or segmental renal infarction. Although relatively rare, urinomas carry relatively high morbidity and mortality. They may present with reduced urine output, pain, swelling, wound discharge or local pressure effects in the early postoperative period. They are usually well-defined anechoic collections that tend to be smaller than lymphoceles but these can be difficult to distinguish. A rapid increase in size on serial ultrasound examinations points to the diagnosis of urinoma and progressive radiotracer activity on radionuclide imaging is diagnostic. Alternatively, a high creatinine level in the collection will also prove the diagnosis.[1,4]

Infections & Abscesses

Peritransplant abscesses are an uncommon complication and usually develop within the first few weeks postoperatively. They can be caused by pyelonephritis or bacterial translocation from an infected peritransplant fluid collection. Due to immunosuppression, these patients may have few clinical features of infection. They may present with pyrexia, pain, or symptoms related to pressure effects of the abscess on the transplanted system.[1] The sonographic appearances of infections and abscesses are quite variable. Focal pyelonephritis may appear as focal areas of non-specific increased or decreased echogenicity. Abscesses have a cystic appearance on ultrasound with variable amounts of echogenic debris and stranding. Gas, if present, differentiates abscesses from other collections. As in native kidneys, echogenic "balls" within the collecting system which exhibit little or no acoustic shadowing are suggestive of fungal infection. See Figure 4.

REDUCED RENAL FUNCTION

Causes of reduced renal function include acute tubular necrosis, rejection and drug nephrotoxicity.

Acute Tubular Necrosis (ATN)

ATN is the most common cause of delayed graft function. It is the primary non-function of the graft within 72 hours of transplantation followed by improvement within a few days to a month.[4,5] ATN is caused by prolonged ischaemia and reperfusion injury and is more common in cadaveric than living-related donor transplants, as well as in transplants with two or more renal arteries. Ultrasound may show an enlarged, oedematous kidney with loss of corticomedullary differentiation. Doppler will usually demonstrate an increase in the resistive index (RI), although this is a non-specific finding (See Figure 5). MAG-3 studies may demonstrate delayed, decreased or absent excretion of radiotracer with parenchymal retention (See Figure 6).[5]

Rejection
Hyperacute rejection is a humeral type of rejection and occurs within minutes after transplantation, requiring immediate reoperation.[5,6] Accelerated acute rejection occurs due to a combination of antibody and cell-mediated rejection and occurs within the first week following transplantation. This type of rejection carries a poor prognosis with graft loss rates of up to 60%.[5,6] Acute rejection is predominantly dependent on cellular immunity and typically occurs at 2-5 weeks following transplantation. On ultrasound, the graft appears enlarged and oedematous with cortical thickening and decreased cortical echogenicity. There may be oedema and reduced echogenicity of the renal sinus fat. There is usually an increase in the RI (>0.8) although this may be preceded by a transient decrease in the RI. However patients with mild but clinically significant acute rejection can have normal ultrasound and Doppler appearances. Furthermore, a rise in the RI is not specific and could be due to ATN or rejection, both of which could coexist.[5,6] Chronic rejection is a slow, progressive process which results in interstitial scarring and insidious decline in renal function, occurring months to years post-transplantation. The ultrasound appearances are of a small graft with a thinned echogenic cortex and mild hydronephrosis although the diagnosis is histological.[4,5,6]

Drug Nephrotoxicity

This is usually due to cyclosporine which has a direct vasoconstrictive effect. The ultrasound findings are non-specific and include a rise in RI, although the appearances are frequently normal.[4,5]

VASCULAR COMPLICATIONS

Vascular complications include renal artery stenosis, renal artery thrombosis, renal vein thrombosis and arterio-venous fistulae.

Renal Artery Stenosis (1-4%)

Usually occur within the first 3 years post transplant and have an increased incidence in cadaveric donors and end to end anastomoses. 50% occur as short segment stenosis at the anastamotic site.¹ Long segment stenoses of the proximal artery may also occur. Causes include poor operative technique, clamping, trauma, ischaemia, atherosclerosis, rejection and vascular kinking.[5]

Clinically patients can present with refractory hypertension, bruit over the graft site or hypertension with associated graft dysfunction.

On Doppler ultrasound arterial stenosis can result in increased peak systolic velocity (2 m/s), marked post stenotic disturbance or a velocity gradient >2:1 between the systolic
and post stenotic velocities. [1,5] Dampened arterial waveforms may be seen within the renal parenchyma, with increased resistive index values (tardus-parvus). See Figures 7 & 8. Scintigraphy will show decreased renal perfusion, which in itself is non-specific. MR or conventional angiography can be used for more accurate delineation.

**Renal Artery Thrombosis (1-5%)**

Usually occurs within the first week, and may be secondary to rejection or poor surgical technique. [5] Other causes include trauma, intimal flap or vascular kinking. Clinically patients present with sudden onset anuria with graft swelling and tenderness.

Ultrasound may show global or segmental changes. If global the kidney can appear enlarged and hypoechoic with widespread loss of arterial and venous flow. With segmental thrombosis a wedge shaped hypoechoic area with absent Doppler flow is seen; however these findings may also be seen in infection. [1] Conventional or MR angiography again can be used for more accurate assessment.

**Arterio-venous Fistula and Pseudoaneurysm (2-18%)**

The majority of cases occur secondary to percutaneous renal biopsy. Most are small and resolve spontaneously. Clinically patients have haematuria, high output cardiac failure (if large) or hypertension. [1,5].

Doppler ultrasound will show high velocity low impedance wave forms with associated arterialisation of the venous waveforms (See Figure 9). [8] Pseudo-aneurysms are seen as renal cyst like lesions that demonstrate high vascularity. Conventional angiography is the gold standard imaging modality and allows treatment at time of assessment.

**Renal Vein Thrombosis (4-5%)**

Usually occurs in the first week post transplant and may be due to damage at the anastamotic site or compression by a peri-nephric collection. Late presentation is usually secondary to rejection or hypovolaemia. Patients present with abrupt onset of oliguria and a painful enlarged kidney. [1,5]

Ultrasound features include an enlarged hypoechoic kidney (See Figure 10). Venous flow will be reduced or absent. Increased vascular resistance will cause dampening of the diastolic flow velocities, causing increased resistive index values, and ultimately reversed diastolic flow (See Figure 11). Early recognition is important as prompt treatment can restore function.
UROLOGICAL COMPLICATIONS

Urological complications occur in 4-8% of patients and are associated with low mortality rates.[1]

Urinary Obstruction

This can occur in up to 2% of patients and will usually present in the first 6 months as a rise in creatinine. The most common site of blockage is the distal 1/3 of the transplanted ureter with the most common causes being ureteral kinking, technical error at re-implantation or stenosis from scarring due to ischaemia or rejection (see Figure 12). Less commonly obstruction may be secondary to extrinsic compression from perinephric fluid collections, as previously discussed, or intrinsic blockage secondary to calculi, blood clots or infection.[1]

The initial imaging modality of choice is ultrasound which may show hydronephrosis (See Figure 13). It can however be difficult to differentiate obstructive hydronephrosis from pelvicalyceal dilatation secondary to vesico-ureteric reflux or non obstructive dilatation due to rejection. If hydronephrosis is confirmed functional radionuclide imaging (MAG-3) is useful to assess renal uptake of tracer and subsequent drainage into the ureter and bladder. In an obstructed system tracer will be readily taken by the kidney, but there may be no significant drainage. Delayed imaging or diuretic/postural changes may be used to promote drainage in a non obstructed system.

Calculus Disease

Stone formation can occur in 1-2% of renal transplant patients. This is slightly higher than the native population and may be associated with altered calcium states due to tertiary hyperparathyroidism in some patients. Patients present with a sudden deterioration post transplant and rising creatinine. Unlike native kidney stone disease these patients do not experience renal colic.[1,5] Imaging options are similar to those used for urinary obstruction. Calculi may be visible on ultrasound as echogenic foci producing acoustic shadowing. Radionuclide imaging may be used to differentiate obstruction from dilatation. In equivocal cases low dose CT is of use to determine the presence and location of calculi.

NEOPLASIA

The reported prevalence of neoplasms in renal transplant patients is 6%.

Post-Transplant Lymphoproliferative Disorder (PTLD)
PTLD is a recognized complication of renal transplantation occurring in 1-8% of patients. [1,3] It occurs as a consequence of impaired immunity due to transplant related immunosuppressant treatment, with a mean time to onset of 46 months, although this is influenced by the type of immunosuppressant and length of treatment. The majority of cases are linked to Ebstein Barr Virus infection, with subsequent induction of B Cell proliferation unopposed by the suppressed immune system. Manifestation of disease ranges from plasma cell hyperplasia to malignant monoclonal lymphoma, contributing to mortality in over 50% of affected patients.[3]

In up to 50% of cases disease will be confined to the abdomen and will be predominantly extra-nodal. Although almost any organ can be affected the liver is the most common and disease may manifest as diffuse or focal intraparenchymal lesions. Small bowel involvement includes bowel wall thickening, focal masses and small bowel intussusception. Pathologically enlarged lymph nodes may be seen in the 20% with extra-nodal involvement. See Figures 14,15 & 16.

**Renal Cell Carcinoma (RCC)**

Immunosuppressive drugs are associated with an increased risk of RCC. The development of RCC in transplant kidneys per se is relatively rare, and in transplant recipient patients, approximately 90% of RCCs develop in the native kidneys. This is because of chronic renal failure patients undergoing haemodialysis acquiring renal cystic disease which is associated with the development of RCC.[1] See Figure 17.

**Images for this section:**
Fig. 1: Colour doppler imaging demonstrating normal flow within the transplant kidney
Fig. 2: Normal resistive index in transplanted kidney
**Fig. 3:** Hypoechoic collection 1 day post operatively lying superficial to mid pole of transplanted kidney consistent with haematoma

**Fig. 4:** Echogenic material within dilated collecting system consistent with fungal balls.
Fig. 5: Doppler of transplant kidney demonstrating absent diastolic flow in ATN
Fig. 6: MAG 3 rising renogram curve and no filtrate production in ATN
**Fig. 7:** High velocity in renal artery stenosis of a transplanted kidney
Fig. 8: Tardus parvus waveform in renal artery stenosis of transplanted kidney
Fig. 9: Colour doppler demonstrating focal area consistent with arteriovenous fistula in a transplant kidney following a biopsy

Fig. 10: Inhomogeneous swollen transplant kidney in renal vein thrombosis
Fig. 11: Reversal of diastolic flow in renal vein thrombosis
Fig. 12: Nephrostogram demonstrating distal ureteric stricture and hydroureter in transplant hydronephrotic kidney
Fig. 13: Hydronephrotic transplant kidney due to ureteric stricture
Fig. 14: Lymph node mass in right iliac fossa in patient with histologically proven PTLD.
**Fig. 15:** Same patient as in Patient 14 showing abnormal soft tissue infiltration of transplant kidney in left iliac fossa;
**Fig. 16:** Same patient as in Figure 14 and 15 showing abnormal soft tissue infiltration in transplant kidney in left iliac fossa, right iliac fossa nodal mass and low density lesions in liver.
Fig. 17: Mixed echogenicity solid lesion in transplanted kidney, proven to be RCC on biopsy.
Conclusion

Doppler ultrasound is the primary imaging technique for assessing the transplanted kidney and its complications. In particular the resistive index and its interpretation is of great value in a variety of settings. Recognising acute vascular complications as causes of graft failure which may be potentially reversible is of paramount importance. The value of other imaging modalities, usually as complimentary techniques, should be used where appropriate. Each case of renal transplant dysfunction should be individually considered and the imaging workup tailored to that specific clinical scenario.

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References


