Renal transplant ultrasound: the nephrologist’s perspective

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.

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.

**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 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                                                  |
| Haematomata                                                  |
| **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                                                  |
| **Parenchymal Disorder**                                     |
| Calcineurin inhibitor (CNI) toxicity                         |
| Acute Rejection                                              |
| **3. Subacute (3 months–1 year)**                            |
| Vascular                                                     |
| As above (not as common)                                     |
| **Urological**                                               |
| Obstruction                                                  |
| Collections                                                  |
| Lymphocele                                                   |
| **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                                    |
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.

Secondary criteria such as a peak systolic velocity (PSV) ratio between the transplant renal artery to external iliac artery (EIA) of > 2.0, markedly reduced resistive index (RI) and abnormal intrarenal waveforms (acceleration time > 0.1) have all been reported to support this diagnosis.

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. 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

| Common problems          | Possible Ultrasound Findings                                                                 |
|--------------------------|---------------------------------------------------------------------------------------------|
| Vascular Problems        |                                                                                             |
| Arterial Stenosis        | RI < 0.5                                                                                    |
| Venous Stenosis          | PSV > 300 cm/s                                                                              |
| Thrombosis               | Renal artery: Iliac artery ratio > 2.0                                                      |
| Infarction               |                                                                                             |
| AVM & Pseudoaneurysms    | Turbulent flow on Colour Doppler                                                            |
| Urological Disorders     | Dilated ureter or pelviccalceal system                                                       |
| Hydrenephrosis / Obstruction |                                                                                     |
| Strictures               | Narrowing of collecting system                                                              |
| Renal stones             | Hyperechoic lesion                                                                          |
| Leaks / Urinoma          | Well define anechoic collection                                                             |
| Collections              |                                                                                             |
| Haematoma                | Chronic – hypoechoic collection and varying areas of echogenicity                            |
| Lymphocele               | 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                                                                     |
| Routine biopsies         | N/A (anatomical guidance for procedure)                                                    |

Table 2:

Summary of Ultrasound Findings and Applications in Renal Transplantation

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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. 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). 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.

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. Ultrasound will demonstrate no venous Doppler flows, with mostly enlarged and oedematous kidney parenchyma. 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.

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...
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. 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. Frequently, this is due to strictures or stenosis at the anastomosis to the bladder. Ninety percent occur at the distal third of the ureter. 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. 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, however they are most
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.\(^5,11\) There have been several studies evaluating the importance of elevated RI or PSV in predicting long-term graft
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, amongst the other risk factors such as smoking, obesity, and immunosuppressive use. Screening of transplanted and native kidneys by 1–3 yearly post transplant surveillance ultrasound has therefore been widely recommended.

The reported incidence of both de novo allograft and native renal cell carcinoma varies in the literature. Melchior 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. 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, 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.

**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
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; 0.02% associated with death. AVM and pseudoaneurysms (as mentioned above) are usually post-biopsy complications arising in as many as 10–20% of cases. 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; hydrenephrosis; and AVM or pseudoaneurysms. 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.

References

1. Schwenger V, Hinkel UP, Nahm AM, Morath C, Zeier M. Real-time contrast-enhanced sonography in renal transplant recipients. *Clin Transplant* 2006; 20 (s17): 51–54. (doi:10.1111/j.1399-0012.2006.00600.x).
2. He WY, Jin YJ, Wang WP, Li CL, Ji ZB, Yang C. Tissue elasticity quantification by acoustic radiation force impulse for the assessment of renal allograft function. *Ultrasound Med Biol* 2014; 40 (2): 322–29. (doi:10.1016/j.ultrasmedbio.2013.10.003).
3. Yoursurgery.com. Available at: http://www.yoursurgery.com. Accessed June 12, 2015.
4. Manning M, Wong-You-Cheong J. Imaging of the Renal Transplant Recipient. In: Weir MR, Lerma EV, eds. *Kidney Transplantation*. New York, NY: Springer New York; 2014: 377–400. doi:10.1007/978-1-4939-0342-9_32.
5. Friedewald SM, Molmenti EP, Friedewald JJ, Dejong MR, Hamper UM. Vascular and nonvascular complications of renal transplants: sonographic evaluation and correlation with other imaging modalities, surgery, and pathology. *J Clin Ultrasound* 2005; 33 (3): 127–39. (doi:10.1002/jcu.20105).
6. Patel U, Khaw KK, Hughes NC. Doppler ultrasound for detection of renal transplant artery stenosis-threshold peak systolic velocity needs to be higher in a low-risk or surveillance population. *Clin Radiol* 2003; 58 (10): 772–77.
7. Horrow MM, Parsiakia A, Zaki R, Ortiz J. Immediate postoperative sonography of renal transplants: vascular findings and outcomes.
AJR Am J Roentgenol 2013; 201 (3): W479–86. (doi:10.2214/AJR.12.10310).

8 Sharfuddin A. Renal Relevant Radiology: Imaging in Kidney Transplantation. CJASN 2014; 9 (2): 416–29. (doi:10.2215/CJN.02960313).

9 Jakobsen JÅ, Brabrand K, Egge TS, Hartmann A. Doppler Examination of the Allografted Kidney. Acta Radiol 2003; 44 (1): 3–12. (doi:10.1034/j.1600-0455.2003.00015.x).

10 Lockhart ME, Wells CG, Morgan DE, Fineberg NS, Robbin ML. Reversed diastolic flow in the renal transplant: perioperative implications versus transplants older than 1 month. AJR Am J Roentgenol 2008; 190 (3): 650–55. (doi:10.2214/AJR.07.2666).

11 Radermacher J, Mengel M, Ellis S, Stuht S, Hiss M, Schwarz A, et al. The renal arterial resistance index and renal allograft survival. N Engl J Med 2003; 349 (2): 115–24.

12 Hevia V, Gomez V, Diez Nicolas V, Álvarez S, Gómez del Cañizo C, et al. Development of urologic de novo malignancies after renal transplantation. Transplant Proc 2014; 46 (1): 170–75. (doi:10.1016/j.transproceed.2013.12.004).

13 Hunt JD, van der Hel OL, McMillan GP, Boffetta P, Brennan P. Renal cell carcinoma in relation to cigarette smoking: meta-analysis of 24 studies. Int J Cancer 2005; 114 (1): 101–08. (doi:10.1002/ijc.20618).

14 Argani P, Loe M, Ballard ET, Amin M, Manivel C, Hutchinson B et al. Translocation carcinomas of the kidney after chemotherapy in childhood. J Clin Oncol 2006; 24 (10): 1529–34. (doi:10.1200/JCO.2005.04.4693).

15 Kalble T, Lucan M, Nicita G, Sells R, Burgos Revilla FI, Wiesel M. EAU guidelines on renal transplantation. Eur Urol 2005; 47 (2): 156–66. (doi:10.1016/j.euro.2004.02.009).

16 Melchior S, Franzaring L, Sharden A, Schwenke S, Plümpe A, Schnell R, Dreikorn K Urological de novo malignancy after kidney transplantation: a case for the urologist. J Urol 2011; 185 (2): 428–32. (doi:10.1016/j.juro.2010.09.091).

17 Richter F, Kasabian NG, Irwin RJ, Watson RA, Lang EK. Accuracy of diagnosis by guided biopsy of renal mass lesions classified indeterminate by imaging studies. Urology 2000; 55 (3): 348–52.

18 Israel GM, Hindman N, Bosniak MA. Evaluation of cystic renal masses: comparison of CT and MR imaging by using the Bosniak classification system. Radiology 2004; 231 (2): 365–71.

19 Israel GM, Bosniak MA. An update of the Bosniak renal cyst classification system. Urology 2005; 66 (3): 484–88. (doi:10.1016/j.urology.2005.04.003).

20 Liao C-H, Chueh S-C, Lai M-K, Chen J. Transitional cell carcinoma in renal transplant recipients. Transplant Proc 2004; 36 (7): 2152–53. (doi:10.1016/j.transproceed.2004.08.017).

21 Corapi KM, Chen JL, Balk EM, Gordon CE. Bleeding complications of native kidney biopsy: a systematic review and meta-analysis. Am J Kidney Dis 2012; 60 (1): 62–73. (doi:10.1053/j.ajkd.2012.02.330).