Complications of Renal Transplantation

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Continued improvements in graft survival have led to widespread acceptance of renal transplantation as the preferred treatment for the majority of patients with end-stage renal disease. The long-term care of these patients is often provided away from transplantation centers. This article presents both the clinical and imaging features of renal transplantation complications and their interventional management. Urologic and vascular complications may occur. Vascular complications include renal artery stenosis and renal artery and vein thrombosis. Ultrasound can accurately depict and characterize many of the potential complications of renal transplantation and increasingly magnetic resonance imaging also facilitates this role. In addition, interventional radiologic techniques allow nonsurgical treatment.

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Introduction

The treatment of choice for patients with end-stage renal disease is renal transplantation. Despite improvements in peritoneal dialysis and hemodialysis, these patients survive much longer after receiving a kidney transplant. Survival rates have improved because of refined surgical techniques, more effective immunosuppression with medications such as cyclosporine and OKT3, improved availability of human leukocyte antigen typing for donor-recipient matching, and establishment of a nationwide coordinating network. These advances have enabled a better quality of life for these patients.

The transplanted kidney is usually placed extraperitoneally in the patient’s right iliac fossa, with end-to-side anastomosis to the external iliac vasculature. This placement allows easier dissection to locate these more superficially placed vessels. The type of arterial anastomosis created is dictated largely by the type of graft available. Cadaveric kidneys are typically harvested with an intact main renal artery and an attached portion of aorta. The piece of aorta is trimmed to an oval configuration and then anastomosed in an end-to-side fashion to the recipient external iliac artery. With kidneys from living donors, a portion of the aorta cannot be harvested; in these cases, either an end-to-side anastomosis of the donor renal artery to the recipient external iliac artery or an end-to-end anastomosis to the recipient internal iliac artery is performed. The donor renal vein is always sutured in an end-to-side fashion to the recipient external iliac vein. The currently preferred method for restoring urinary drainage is ureteroneocystostomy, a procedure by which the ureter is implanted directly into the dome of the bladder. However, ureteroureterostomy or peloureterostomy may also be performed.

Although the preceding renal transplantation procedures are most common, there is considerable technical variation. The transplanted kidney may be placed either extraperitoneally or intraperitoneally in the left or right iliac fossa with anastomosis to the external or internal iliac vasculature. The ureter may also be implanted into an interposed bowel segment. The imaging appearances and postoperative complications encountered vary depending on the surgical procedures used, and radiologists must become familiar with the techniques employed in their institutions (1).

Urologic and vascular complications do occur and have a substantial impact on morbidity and mortality. Urologic complications occur in 4%–8% of patients, and vascular complications occur in approximately 1%–2% (2). Vascular complications include renal artery stenosis, infarction, arteriovenous fistulas, pseudaneurysm, and renal vein thrombosis. Nonvascular complications include ureteral obstruction, urine leak, peritransplant fluid collections (hematomas, lymphoceles, abscesses, infection), neoplasms, gastrointestinal and herniation complications, and posttransplantation lymphoproliferative disorder. Advancements in percutaneous diagnostic and interventional techniques have been steady, and today surgical intervention to treat complications may be obviated in many cases (3).

The increasing number of kidney transplant recipients returning to their communities with functioning allografts suggests that much of the long-term care of these patients will be provided away from transplantation centers. The community hospital radiologist may be called on to diagnose and manage transplantation complications. In this article, the applications of multimodality imaging and interventional radiology to transplantation complications are considered, with emphasis on the clinical and radiologic features.

Urologic Complications

In early reports of renal transplantation, the prevalence of urologic complications varied from 10% to 25%, with a mortality rate ranging from 20% to 30% (2,4). In these patients, ureteroureterostomy or peloureterostomy was used to restore urinary tract continuity. Patients who undergo ureteroneocystostomy, the reconstructive technique used in most active kidney transplantation programs today, have a lower incidence of urine leak or obstruction, compared with patients who underwent the older procedures. Approximately two-thirds of the early urologic complications (urine leak or obstruction) are apparent in the first month after transplantation and are treated by the transplantation team. Currently, urologic complication rates are 4%–8% with very low patient mortality (2,4).
Urine leaks and urinomas are relatively rare complications of transplantation and usually constitute an early postoperative problem. Extravasation of urine may occur from the renal pelvis, ureter, or ureteroneocystostomy site due to ureteral necrosis caused by vascular insufficiency or increased urinary pressures caused by obstruction. Caliceal leakage is an uncommon cause and occurs secondary to segmental infarction in patients with accessory renal arteries or due to ligation of a polar artery. Urinomas vary in size and are usually found in the first 2 postoperative weeks between the transplanted kidney and the bladder. They may occur, however, in unexpected locations such as the scrotum or thigh.

A decrease in urine output suggests that a urine leak may have occurred, and clinical signs may include fullness and tenderness around the graft, ipsilateral leg swelling, or scrotal or labial edema. Early detection and repair have been instrumental in reducing patient mortality.

At ultrasonography (US), a urine leak or urinoma appears as a well-defined, anechoic fluid collection with no septations that increases in size rapidly (Fig 1). Drainage may be performed with US guidance, and the higher creatinine level of the fluid compared with its serum concentration differentiates a urine leak from a seroma or lymphocele. Large urinomas can rupture to produce urinary ascites. They can also become infected and eventually form abscesses. Radionuclide studies show extravasation of radiotracer into an area that was initially “cold.” Delayed scintigrams should be obtained, since accumulation of radiotracer may be slow. Antegrade pyelography is necessary to provide detailed information about the site of origin of the urinoma and in planning appropriate intervention (6).
Small urine leaks may be treated with percutaneous nephrostomy and stent placement. Caliceal leakage caused by infarction is treated with percutaneous nephrostomy alone. Ureteral stents must be kept in place for 6–8 weeks after cessation of leakage to allow complete healing of the ureter and to preserve long-term patency. If healing is unsuccessful, reimplantation of the ureter may be required; if distal necrosis is present, pyelocystotomy or pyeloureterostomy with the native ureter is performed (6).

**Urinary Obstruction**

Urinary obstruction occurs in approximately 2% of transplantations and almost always within the first 6 months after the procedure. Obstruction of the transplanted kidney may occur at any location but is most frequent at the site of implantation of the ureter into the bladder. More than 90% of ureteral stenoses occur within the distal third of the ureter (Fig 2). Narrowing at the ureterovesical junction may be caused by scarring secondary to ischemia or rejection, by technical error during the ureteroneocystostomy, or by kinking. These events account for more than 50% of obstructions that cause ureteral stricture. Less common causes include pelvic fibrosis, calculi, papillary necrosis, fungus ball, clots, and compression from an extrinsic mass such as adjacent peritransplant collections. Occasionally, obstruction that develops years after transplantation—especially in patients who have undergone multiple procedures—may be related to adhesions, vascular insufficiency, or fibrosis (3,7).

The transplanted kidney is denervated; thus, the patient will not complain of typical renal colic when obstruction occurs. Urinary obstruction manifests by a rising level of serum creatinine. Obstruction may be difficult to differentiate from chronic rejection, since both cause rising creatinine levels. In addition, mild dilatation of the collecting system may occasionally be seen in cases of chronic rejection.

US can be used to confirm the diagnosis of hydronephrosis; however, intrarenal edema and fibrosis associated with rejection may prevent the normal hydronephrotic response. Some transplant recipients may have substantial obstruction but little or no renal dilatation. Mild to moderate dilatation of the transplanted pyelocaliceal system and ureter may occur secondary to a full bladder. In all cases, when the patient’s bladder is full, the
bladder should be emptied and the transplant imaged again. In addition, a denervated, transplanted collecting system has no tone; thus, any transient episode of hydronephrosis may cause a persistent appearance of mild dilatation. The single renal graft is responsible for excreting the entire urine output from the transplant recipient’s body, and, over time, increasing output may exacerbate pelvocaliectasis. Studies have attempted to differentiate obstructive from nonobstructive pyelocaliectasis by using the resistive index. Extrapolation of these results from native to transplanted kidneys has not, however, been entirely successful. US also demonstrates lymphoceles, hematomas, abscesses, and urinomas that may cause ureteral obstruction. Any echogenicity within a dilated collecting system is usually clinically significant (8,9).

Scintigraphy may demonstrate urinary obstruction. In a patient with early partial obstruction, good perfusion and prompt uptake of the radiotracer may be seen; however, in a patient with functionally significant hydronephrosis, radioactivity is retained in the collecting system. Delayed images (obtained 2–4 hours after injection) are useful for differentiating an obstructed ureter from a dilated but unobstructed ureter, since an unobstructed system shows clearance into the bladder. Diuretic renography has proved useful for determining the functional significance of dilated collecting systems. Conventional clearance times (furosemide clearance t1/2 > 20 minutes [10]) can be used in the assessment of urinary tract patency.

Excretory urography is only infrequently successful for locating a urinary obstruction, and the site is usually best determined with an antegrade contrast material examination. The superficial anterior position of the allograft allows easy access for antegrade pyelography performed with real-time US guidance. The Whitaker test is helpful in equivocal cases (11). The combination of normal results from the Whitaker test and antegrade pyelography virtually excludes the presence of obstruction.

Percutaneous nephrostomy is used to relieve obstruction and allow the deployment of other radiologic interventions such as ureteral stent placement and balloon ureteroplasty (Fig 3). Balloon dilation of posttransplantation ureteral strictures produces an overall success rate of ureteral dilatation in 90% of cases, with the best results obtained in fresh surgical strictures and the poorest in chronic ischemic strictures or areas of periureteral fibrosis (12). Strictures of the middle and upper ureters result most commonly from ischemic or periureteral fibrosis and may require surgical intervention. High balloon pressures of

Figure 3. Hydronephrosis relieved by stent placement. (a) Gray-scale sonogram reveals severe hydronephrosis resulting from narrowing at the ureterovesical anastomosis. Obstruction was relieved by stent placement. (b) Sonogram obtained after placement of a retrograde stent (arrow) demonstrates complete resolution of the hydronephrosis.
10–12 atm or greater are usually needed for successful dilation, and the balloon is left inflated for 20 minutes or until balloon “waisting” has disappeared (Fig 4). After dilation, a 10-F stent is left in place for only 10 days. In the immunocompromised transplant recipient, the risk of infection precludes long-term stent placement unless a totally internal, double pigtail stent is used. Recurrences most commonly develop within the first year after transplantation (13).

**Peritransplant Fluid Collections**

Peritransplant fluid collections have been reported in up to 50% of renal transplantations and include urinomas, hematomas, lymphoceles, and abscesses. The clinical significance of these collections is largely determined by their size, location, and possible growth. In the immediate postoperative period, small hematomas or seromas manifesting as crescentic peritransplant collections are almost expected. Their size should be documented at baseline examination, since any increase in size may warrant intervention. Growing collections may be indicative of urine leak, abscess, or vascular injury. Different types of peritransplant fluid collections can be partially differentiated based on the time interval after transplantation. Urinomas and hematomas are most likely to develop immediately after transplantation, whereas lymphoceles generally occur 4–8 weeks after the surgical procedure. The sonographic characteristics of peritransplant fluid collections, however, are entirely nonspecific, and ultimately, diagnosis may be made only with percutaneous aspiration (14,15).
Hematomas
Hematomas are common in the immediate postoperative period, but they may also develop spontaneously or as a consequence of trauma or biopsy. They are usually small and resolve spontaneously. Large hematomas can displace the transplanted kidney and produce hydronephrosis.

At US, hematomas demonstrate a complex appearance. Acute hematomas are echogenic and become less echogenic with time. Older hematomas even appear anechoic, more closely resembling fluid, and septations may develop. Similarly, at computed tomography (CT), the appearance of a hematoma also is time dependent. An acute hematoma has high-attenuation components, and older hematomas contain liquefied and serous portions of intermediate attenuation (Fig 5). At magnetic resonance (MR) imaging, an acute hematoma can show high signal intensity with both T1-weighted and T2-weighted pulse sequences. At scintigraphy, hematomas demonstrate a “cold defect.”

Diagnostic aspiration can be performed to rule out abscess formation. However, percutaneous drainage of the entire fluid collection is often neither efficacious (due to its multiloculated nature) nor advisable because of the self-limited nature of the complication and risk of infection.

Lymphoceles
Lymphoceles are the most common peritransplant fluid collections, with a prevalence of 0.5%–20%. They may develop at any time, from weeks to years after transplantation. However, they are usually an early complication, occurring within 1–2 months after transplantation. Lymphoceles are caused by leakage of lymph from surgically disrupted lymphatic channels along the iliac vessels or from the lymphatics of the transplanted kidney. Risk factors include inadequate ligation of the lymphatic channels across the iliac vessels, administration of heparin, and possibly increased lymphatic flow secondary to edema of the lower extremities. These fluid collections usually occur medial to the transplant, between the graft and the bladder (16).

Lymphoceles are the most common fluid collection that causes transplant hydronephrosis. Patients with a failing allograft may develop ipsilateral lower extremity edema caused by compression of the femoral vein. In rare cases, lymphoceles may develop in the scrotum and lymphatic drainage may occur through the wound.
At US, lymphoceles are anechoic (Figs 6, 7) and may have septations. Similar to other peri-transplant fluid collections, they can become infected and can develop a more complex appearance (Fig 8). At CT, lymphoceles have variable characteristics and are usually sharply circumscribed. Their CT attenuation values are typical of those of water and usually lower than those of recent hematomas and abscesses. Radionuclide and MR imaging studies are helpful for excluding the presence of urine and blood, respectively.

Small lymphoceles are monitored sonographically, and large ones, if they grow or cause hydronephrosis, should be drained. Aspirated fluid should have a creatinine content equal to that of serum, and the cell count and differential may show relatively few white blood cells with a predominance of lymphocytes. Results of Gram stain and culture of the fluid should be negative.

Lymphoceles commonly recur after simple percutaneous or surgical drainage. Permanent resolution of lymphoceles may require prolonged catheter drainage and transcatheter instillation of sclerosing agents such as povidine-iodine, absolute alcohol, or doxycycline (17). Repeated aspirations run the risk of infecting an otherwise sterile fluid collection. However, success rates of up to 97% have been achieved safely and effectively with percutaneous transcatheter sclerotherapy (17). If a lymphocele becomes infected, external...
drainage and antibiotic treatment are needed. Surgical treatment of a sterile fluid collection involves removal of a generous window of peritoneum for marsupialization. The transplanted kidney effectively becomes an intraperitoneal structure. Future leakage is therefore resorbed in the peritoneal cavity. Retrograde pyelography helps identify the course of the transplanted ureter before marsupialization, since the ureter could be displaced anteromedially and injured when the peritoneal window is constructed (18–20).

**Abscesses and Infection**

More than 80% of renal transplant recipients suffer at least one case of infection during the first year after transplantation. Early diagnosis of and intervention for infectious diseases can help prevent loss of graft function and improve patient outcome.

Infections that occur in the first weeks after transplantation, such as pneumonia, surgical wound infections, and urinary tract infections, are similar to those that typically develop in nonimmunocompromised patients who have undergone surgery. Infections with opportunistic pathogens and cytomegalovirus often develop 1–6 months after surgery, and infections common in the general population are seen after 6 months (21,22). Patients frequently have multiple infections, and immunosuppressive medications, indwelling catheters, and frequent glycosuria are all risk factors. Peritransplant abscesses are an uncommon complication and usually develop within the first few weeks after transplantation. These abscesses may be caused by pyelonephritis or bacterial seeding of a lymphocele, hematoma, or urinoma. Acute bacterial nephritis, renal abscess, or perinephric abscess may occur.

Patients may have few signs or symptoms of infection because of their immunosuppressed state. They may present with fever of unknown origin, pain, or symptoms related to the pressure of the abscess on the transplanted system. In a febrile transplant recipient, any peritransplant fluid collection must be presumed to be infected. Acute pyelonephritis can mimic acute graft rejection, and their imaging appearances may be the same.

The sonographic appearances of infections and abscesses are quite variable. Focal pyelonephritis may appear as focal areas of increased or decreased echogenicity. These findings are nonspecific and can represent infarction or rejection. Intrarenal masses are infrequently visualized and are also sonographically nonspecific. They may represent primary renal cell carcinoma or posttransplantation lymphoproliferative disorder. Abscesses have a complex, cystic, nonspecific appearance at US (Figs 8, 9); however, at CT, they manifest with gas, which serves to differentiate them from other collections. In emphysematous pyelonephritis, gas in the parenchyma of the renal graft produces an echogenic line with distal reverberation artifacts. This finding can be confirmed with abdominal radiography or CT. Any echogenicity within a dilated collecting system is usually clinically significant. Highly echogenic, weakly shadowing masses within a transplanted collecting system are fairly specific for fungus balls (Figs 10, 11). The presence of low-level echoes in a dilated pyelocaliceal system in a febrile patient suggests pyonephrosis. Papillary necrosis results
in sonographic changes in the calices, and intravenous or retrograde pyelography is the definitive study for diagnosing this condition. Retrograde pyelography may also demonstrate distortion of the calices, a feature seen in tuberculosis of the renal transplant (Fig 12). Gallium-67 citrate and indium-111–tagged leukocyte studies are helpful for diagnosing infections that develop 1 month or more after graft healing is complete (23).

Abscesses may be treated with either US- or CT-guided percutaneous drainage. Both intrarenal and extrarenal abscesses usually respond to external drainage and systemic antibiotics (24).

**Vascular Complications**

**Renal Artery Stenosis**
Renal artery stenosis occurs usually in the first year after transplantation. The stenosis may be located before the anastomosis (because of atherosclerotic disease in the donor vessel), at the anastomosis (secondary to vessel perfusion injury, faulty suture technique, or reaction to suture material), or after the anastomosis (due to rejection, turbulent flow from kidney malposition, or arterial twisting, kinking, or compression). Approximately half of renal artery stenoses occur at the anastomosis, and end-to-end anastomoses have a threefold greater risk of stenosis than end-to-side anastomoses (25,26).

About 80% of patients with end-stage renal disease are hypertensive, and after renal transplantation two-thirds of this group experience a reduction in hypertension. In patients with persistent hypertension, renal artery stenosis may not be considered as a cause owing to concomitant graft failure. Several clinical scenarios should prompt a search for stenosis: (a) severe hypertension refractory to medical therapy, (b) hypertension and the presence of an audible bruit over the graft, and (c) hypertension associated with unexplained graft dysfunction (27).

Color Doppler US demonstrates stenotic segments as focal areas of color aliasing due to increased flow velocity. These areas can be selectively evaluated with duplex Doppler techniques to characterize and grade the flow disturbance. Doppler criteria for significant stenoses include (a) focal frequency shifts greater than 7.5 KHz (when a 3-MHz transducer is used) or velocities greater than 2 m/sec, (b) a velocity gradient between stenotic and prestenotic segments of more than 2:1, and (c) marked distal disturbance (spectral broadening). In the renal parenchyma, tardus-parvus waveform abnormalities can be observed.
MR angiography can be used to diagnose renal artery stenosis in renal transplants (Fig 13). MR angiography has the advantage of requiring either no contrast material or a gadolinium chelate that is not nephrotoxic. Renal scintigraphy and time activity curves show reduced perfusion in transplants with complete vascular obstruction and renal artery stenosis. This finding is nonspecific, however, as it may be seen with other causes of parenchymal failure, including graft rejection and urinary obstruction.

Surgical correction of graft renal artery stenosis is usually successful, but it is associated with substantial morbidity. Primary treatment of graft renal artery stenosis by means of percutaneous transluminal angioplasty with or without stent placement results in good intermediate-term patency and is associated with significant early improvement in blood pressure and creatinine level (28) (Fig 14). Because of its low level of morbidity, relatively modest cost, and effectiveness, percutaneous transluminal angioplasty is accepted as the initial treatment of choice. Clinical success rates resulting in substantial initial improvement or cure have been reported in 73% of patients.
Decrease in hypertension may be seen in 1 day, and the prevalence of periprocedural complications is substantially lower than that associated with surgical repair. Up to 20% of stenoses may require repeat dilation for maximum success (29).

Infarction
Renal artery thrombosis may result from hyperacute rejection, anastomotic occlusion, arterial kinking, or intimal flap. Segmental infarcts in the renal transplant may be focal or diffuse and may occur as part of rejection or as a result of an unassociated vascular thrombosis. Vasculitis may induce small segmental infarcts.

Patients with renal transplant infarction present with absence of urinary output and often with swelling and tenderness over the graft and anuria. Although the graft itself is denervated, the inflammation within the transplanted kidney may incite an inflammatory response in the adjacent visceral peritoneum, with local pain in this location.

At US, a segmental infarct appears as a poorly marginated, hypoechoic mass or a hypoechoic mass with a well-defined echogenic wall. If the infarction is global, the kidney will appear hypoechoic and be diffusely enlarged. At color or power Doppler imaging, segmental infarcts appear as wedge-shaped areas without color flow in (Fig 15), although these findings may also be seen in severe pyelonephritis or transplant rupture. In total vascular obstruction, there is no perfusion to the transplanted kidney, and no arterial or venous blood flow is seen in the graft at US. At technetium-99m dynamic radionuclide studies, a photopenic region may be seen. However, these findings are not specific, since hyperacute or accelerated acute rejection can have similar clinical, Doppler US, and scintigraphic features.

CT can be used to detect perfusion deficits in the renal graft parenchyma, but it is generally not employed in patients with elevated creatinine values because of the risk of compromising renal function (Fig 16). If any doubt exists as to the nature of the US findings, angiography or MR angiography may be performed. MR angiography is increasingly used to screen for vascular abnormalities in renal transplants. Phased-array surface coils provide excellent signal-to-noise information that permits rapid acquisition of high-quality images without the use of potentially nephrotoxic agents. Dynamic enhanced MR imaging can be useful for diagnosing both segmental and global infarctions (Fig 17) as well as renal artery
Figures 15, 16. Infarct of a renal graft. (15) Power Doppler US image demonstrates segmental loss of perfusion in the transplanted kidney (arrows), a finding compatible with infarct. (16) CT scan demonstrates segmental hypodensity (arrow) in the transplanted kidney, a finding that proved to represent an infarct.

Figure 17. Occlusion of a graft renal artery with lower pole infarction in a 38-year-old patient. (a) Nontargeted MIP image reveals one graft renal artery that is normal. However, a total of three graft renal arteries were anastomosed to the external iliac artery, and nonvisualization of two of the three arteries indicates perioperative thrombosis. This vascular anatomy could not be ascertained with Doppler US. (b) Another MIP image demonstrates the venous anatomy. (c) Contrast material–enhanced three-dimensional MIP image demonstrates perfusion to the upper renal pole with lack of perfusion to the lower pole, a finding that indicates lower pole infarction from arterial thrombosis.
thrombosis (Fig 18). In addition, MR imaging has been helpful for demonstrating small infarcts caused by iatrogenic drug-induced vasculitis (Fig 19).

Percutaneous angiographic interventional techniques may be valuable in treating infarctions and renal artery thrombosis. Early diagnosis and treatment are vital for allograft salvage. Depending on the cause and duration of the occlusion, thrombolytic therapy may prolong survival (30).

**Arteriovenous Fistulas and Pseudoaneurysms**

Percutaneous biopsy is commonly performed in transplant recipients when rejection is suspected. Arteriovenous fistulas and pseudoaneurysms are
occasionally seen after graft biopsies. Gross hematuria is seen after 5%–7% of biopsies and is usually self-limited; however, massive or persistent hematuria may occur (31).

Color and duplex Doppler US easily demonstrate arteriovenous fistulas. Arteriovenous fistulas and pseudoaneurysms appear as localized areas of disorganized color that extend outside the confines of the normal vessel. This appearance is caused by perifistula vibration and is detected with the more sensitive color Doppler units. Arteriovenous fistulas also appear as abnormal high-velocity turbulent flow isolated to a single segmental or interlobar artery and paired vein that produces aliasing on color Doppler images. The feeding artery shows a high-velocity low-resistance waveform (Fig 20), and the draining vein demonstrates arterialization (Fig 21).
On gray-scale US images, pseudoaneurysms appear as simple or complex renal cysts, but with color imaging, highly vascular intracystic flow is seen. Pseudoaneurysms with a narrow neck and no venous communication demonstrate a classic machinelike to-and-fro Doppler spectrum at their necks. Those associated with arteriovenous fistulas exhibit a high-velocity low-resistance spectrum at their necks, with minimally pulsatile high-velocity flow in the draining vein. When the degree of perifistula vibration limits US assessment of the vascular anatomy, MR imaging enables evaluation (Figs 22, 23).

Most complications after biopsy are treated conservatively, and most pseudoaneurysms resolve spontaneously. If, however, a pseudoaneurysm demonstrates progressive enlargement or unusual size (more than 2 cm in diameter), intervention is warranted. Selective transvascular catheterization may be used. The size and location of the arteriovenous fistula or pseudoaneurysm determine which embolic agent to use. For example, a large arteriovenous malformation is more easily treated with steel coils, since particulate matter such as absorbable gelatin sponge (Gelfoam) may pass through the fistulous communication and embolize the systemic circulation. Peripheral lesions may be difficult to catheterize selectively,

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**Figure 21.** Arteriovenous fistula. (a) Duplex Doppler US image of the lower pole segmental artery shows increased velocity and decreased resistive index. (b) Duplex Doppler US image of the adjacent vein shows arterialization of flow, a finding consistent with arteriovenous fistula.

**Figures 22, 23.** (22) Arteriovenous fistula following transplant biopsy. A color Doppler study in a 41-year-old hypertensive patient with a bruit audible over the renal allograft revealed tissue vibration. Because of marked tissue vibration, it was difficult to interrogate the transplant vasculature. Non-targeted MIP images during the arterial phase of contrast-enhanced three-dimensional MR angiography reveal a focal severe stenosis of the graft renal artery beyond its origin (arrow). The transplanted renal venous system is enhancing during the early arterial phase along with the iliac vasculature and inferior vena cava. The cortical nephrogram is very faint due to the marked arteriovenous shunting. (23) Arteriovenous fistula. Contrast-enhanced three-dimensional MR angiogram demonstrates the renal artery and veins simultaneously, an appearance suggestive of fistula formation. The diagnosis was confirmed at angiography (not shown).
but a flow-directed detachable balloon can be delivered to the site. Microcatheter systems may also be helpful for superselective embolization of small, peripheral feeding vessels. In treatment of transplant-related arteriovenous fistulas (unlike that of peripheral arteriovenous fistulas), occlusion of the proximal bleeding vessel is sufficient (27,32).

**Renal Vein Thrombosis**

Renal vein thrombosis is an unusual complication of transplantation; it occurs in less than 5% of patients and usually in the first postoperative week. Renal vein thrombosis is heralded by an abrupt cessation of urinary function and swelling and tenderness over the graft. Hypovolemia, venous compression from a peritransplant fluid collection, dysfunctional anastomosis, and slow flow secondary to rejection or other allograft disease can also precipitate renal vein thrombosis. An increased prevalence of renal vein thrombosis in left lower quadrant allografts has also been attributed to compression of the left common iliac vein between the sacrum and the left common iliac artery (silent iliac artery compression syndrome) (26).

At gray-scale US, renal vein thrombosis may manifest with an enlarged kidney. Venous flow is reduced or absent, and there is increased resistance on the arterial side, often resulting in reversed diastolic flow on Doppler images (Fig 24). MR venography helps confirm this complication in transplants (Fig 25) (33,34).
Early recognition of renal vein thrombosis is crucial because the allograft may sometimes be salvaged by prompt thrombectomy. Even with prompt diagnosis, however, graft infarction may develop, and a transplant nephrectomy is usually performed to prevent secondary infection (23).

Calculous Disease

Kidney transplant recipients, compared with the general population, are at increased risk for developing urinary calculi (35) and approximately 1%-2% develop a clinically significant stone (31). Persistent secondary hyperparathyroidism is seen in 38%-77% of end-stage renal disease patients after transplantation (31). In the first year after transplantation, 15% of patients may be hypercalcemic, which increases the risk of renal stones (36).

The diagnosis of urinary calculi is suspected when renal function suddenly deteriorates. The patient does not experience typical renal colic because the transplanted kidney is denervated. US, radionuclide imaging, and contrast material studies are all useful diagnostic methods (Fig 26). Percutaneous nephrostomy tube placement is valuable because it provides low-pressure drainage and allows renal function to stabilize. Antegrade pyelography can then be used to give exact definition of the collecting system and position of the stone (37).

Most stones can be removed with endourologic techniques. Upper tract stones are best treated by means of percutaneous nephrostolithotomy. Cystoscopy with electrohydraulic lithotripsy is appropriate for treating bladder stones.
Neoplasms

Prolonged immunosuppression following renal transplantation places the transplant recipient at about 100 times the normal risk for developing cancer. The average reported prevalence of malignancy in these patients is 6% (38), with cases consisting predominantly of skin cancers and lymphomas. The degree of immunosuppression and its duration are both important factors in the development of malignancy. The frequency of urologic tumors such as prostatic adenocarcinoma and germ cell tumors of the testis does not appear to be increased in this population. However, the prevalence of renal adenocarcinoma may be increased, with 90% of the tumors occurring in the native kidney and 10% occurring in the transplanted kidney (39). One reason for the increased risk of renal adenocarcinoma is that approximately half of the patients who undergo hemodialysis because of chronic renal failure develop acquired renal cystic disease and 9% develop tumors (40). Patients with longer use of hemodialysis are at greater risk for developing acquired cystic disease. Fortunately, less than 1% of these patients develop malignant tumors that metastasize. In addition, patients with a significant past exposure to cyclophosphamide have an increased risk for developing urothelial tumors (41); although cyclophosphamide is less commonly used now because of the availability of cyclosporin A, there are many renal transplant patients with a history of cyclophosphamide exposure.

A complete urologic evaluation for gross hematuria is recommended in all renal transplant recipients. Intravenous pyelography may be useful if there is good transplant function, although it will not provide information about the native kidney. US or CT of the native and transplanted kidneys provides more complete information, and a solid heterogeneous enhancing mass that may be cystic is often identified (Fig 27). Cystoscopy with retrograde pyelography of all present kidneys and ureters is advisable.

Transplant glomerulopathy and recurrence of preexisting glomerulonephritis frequently cause microhematuria often associated with proteinuria. However, since these patients have an increased risk of developing neoplasms, a complete urologic work-up is advisable.

Development of renal adenocarcinoma in the transplanted kidney is uncommon, with the majority of malignancies related to skin cancers and lymphomas. Malignancy in the transplanted kidney is treated by total removal of the renal graft and cessation of immunosuppression. Urothelial malignancies can likewise be treated in a standard fashion. In the presence of immunosuppression, intravesical instillation of Calmette-Guérin bacillus is not recommended.

Gastrointestinal and Herniation Complications

Gastrointestinal complications may occur in transplantation patients. The most common of these complications is gastrointestinal hemorrhage due to peptic ulcer (42). Prevalence of the complications may vary with use of an intraperitoneal or extraperitoneal approach in placement of the allograft (43). Postoperative adhesions may lead to intestinal obstruction. Herniation of bowel through a transplant peritoneal defect may lead to compromise of the intestine or of the transplant itself (Fig 28). Urinary obstruction from obturator herniation of the ureter may also occur (44).
Pseudomembranous colitis secondary to overgrowth of *Clostridium difficile* in patients receiving antibiotic therapy is another complication that can be seen in renal transplant recipients. A typical “accordion sign” secondary to massive edema of the intestinal wall is highly suggestive of the diagnosis on CT images of the abdomen.

**Posttransplantation Lymphoproliferative Disorder**

Impaired immune surveillance and the oncogenic effects of immunosuppressive therapy itself may be causative factors of the posttransplantation lymphoproliferative disorder (PLTD). An association with Epstein Barr virus is usually demonstrated. PLTD complicates 8% of transplantations and is diagnosed at a median of 80 months after transplantation. The liver, brain, and lung are more common sites of involvement than the gastrointestinal tract. PLTD lesions are polymorphic collections of B cells but may evolve into forms indistinguishable from non-Hodgkin lymphoma. If there is involvement of the small intestines, patients often have generalized disease (Figs 29, 30) (45).

**Conclusions**

Transplantation is currently one of the accepted treatments of irreversible kidney disease. Improvements in surgical techniques and more sophisticated, potent immunosuppressive drugs have resulted in remarkable advances in survival of patients and renal grafts. Nevertheless, substantial complications occur in both the immediate postoperative period and later. Imaging has a critical role in the evaluation of these complications, and interventional radiologic techniques are often successfully applied to their treatment.
References