# INTRODUCTION

The preferred modality for renal replacement is renal transplantation, and its superiority in prolonging the longevity of patients with end-stage renal disease is well established.[1] Economically, renal transplant is a more feasible method of renal replacement therapy, an important factor when considering the increasing health care costs and the government's continued commitment to provide coverage of end stage renal disease therapies. In terms of quality of life, patient surveys have consistently indicated a clear preference for transplantation over other replacement therapies.[2]

Survival rates have improved because of refined surgical techniques, more effective immunosuppression with medications such as cyclosporine and muromonab-CD3( OKT3) and improved availability of human leukocyte antigen typing for donor-recipient matching.[3] Despite improvements in diagnostic and surgical techniques, surgical complications (SCs) following kidney transplantation remain an important clinical problem that may increase morbidity, hospitalization and costs.[4] Surgical complications remain an important potential cause of graft loss after kidney transplants. They may also result in graft dysfunction that must be differentiated from that result from medical and immunologic causes.[5] For all of these reasons, it is crucial that all medical persons involved in the postoperative care of kidney transplant recipients be aware of the potential surgical complications that may occur, thus allowing for rapid diagnosis and initiation of treatment.[6]

Depending on the stage of the transplant process these complications may have an origin in back-table work to prepare the allograft, the dissection of the graft bed and vascular anastomosis or in the ureterovesical anastomosis. However, SCs do not necessarily imply a surgical procedure-related technical problem. Several risk factors such as older donor and recipients with atherosclerosis and obesity are observed among kidney transplant candidates.[7] This may lead to both vascular thrombotic events and ureteral complications after surgery. Moreover, potential kidney recipients have unfavorable cardiovascular profiles and frequently they have to be treated with antiplatelet agents which may predispose to perioperative bleeding complications. On the other hand, newer and more potent immunosuppressive drugs, such as mycophenolate mofetil (MMF) and sirolimus (SRL) have contributed to decrease the acute rejection rate and improve kidney graft function. However, recent reports show a strong association between these agents and increased incidence of wound complications and lymphocoele formation.[8-10] So, an additive effect between these newer agents and risk factors related either to donor or recipient characteristics may magnify the development of SCs.

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%.[3] Vascular complications include renal artery stenosis, infarction, arteriovenous fistulae, pseuodaneurysm, and renal vascular thrombosis. Nonvascular complications include ureteral obstruction, urine leak, peritransplant fluid collections (hematomas, lymphoceles, abscesses), neoplasms, gastrointestinal and herniation complications, and post transplantation lymphoproliferative disorder. Advancements in percutaneous diagnostic and

interventional techniques have been well established and invasive surgical intervention to treat complications may be obviated in many cases.[11]

## Post-transplant hemorrhage

Bleeding is uncommon after kidney transplants; it usually results from unligated vessels in the graft hilum or from the vascular bed preparation of the recipient. Risk factors include recipient obesity, the presence of antiplatelet agents, and the need for anticoagulation.[3]

A falling hematocrit level, hypotension or tachycardia, and significant iliac pain should all raise concern regarding the possibility of bleeding. Surgical exploration is seldom required, because the bleeding often stops spontaneously. However, ongoing transfusion requirements and hemodynamic instability are all indications for surgical re-exploration.[3]

## Transplant renal artery stenosis

Transplant renal artery stenosis (TRAS) is a very common vascular complication following kidney transplantations. It is reported in a wide range of frequency occurring in 3-12% of patients in some reports even up to 20% depending on the awareness and the available imaging means especially MDCT-angiography.[12] It can be classified into two main categories; TRAS and proximal or pseudo-TRAS. Also, TRAS can be categorized by the level of stenoses and this includes anastomotic stenoses, stenoses of the proper transplant renal artery and finally segmental renal artery stenoses. Proximal-TRAS refers to pre-existing or developing atherosclerotic inflow stenoses in the native iliac arteries of the transplant recipient.[13] Predisposing factors for transplant renal artery stenosis are cadaveric transplant, end-to-end anastomosis, surgical clamp injury, intimal dissection, and inadequate suturing technique, long or kinking artery, prolonged cold ischemia time, acute cellular rejection, and cytomegalovirus (CMV) infection.[14, 15]

Clinically, patients with TRAS in the immediate post-transplant period, present with oliguria or anuria and are dialysis dependent. After the first week, patients with TRAS usually present with severe renovascular hypertension.[16] Even though severe renovascular hypertension could be attributed to TRAS, all other causes like chronic rejection, steroid use, cyclosporine toxicity, recurrent glomerulonephritis and disease of native kidneys should be kept in mind.[17] If TRAS is not managed properly in the proper time, it could lead to renal impairment and graft ddysfunction. Non-invasive imaging is mandatory in the immediate post-transplant period to evaluate possible transplant renal artery stenosis.[18]

## Diagnosis

Doppler ultrasound should be the initial diagnostic modality used because of its ability to reveal the location, length, and gross appearance of the stenosis. In addition, it is widely available, cost-effective, and does not use ionizing radiation and the nature of the transplanted renal arteries makes Doppler ultrasound an ideal screening modality.[18, 19]

Categorisation and better characterisation of TRAS can be demonstrated using magnetic resonance angiography with or without contrast medium (especially gadolinium-enhanced), MDCTangiography. Radionuclide imaging that includes the administration of an angiotensin converting enzyme inhibitor (captopril scan) shows

findings similar to those of renovascular hypertension in native kidneys.[13, 17] Catheter-based angiography is the gold standard technique for diagnosing TRAS. The use of low- or iso-osmolar contrast material is recommended to reduce the risk of contrast material–induced nephropathy.

Doppler findings in TRAS include peak systolic velocity 2.0-2.5 m/s, low pulsatility index, and a parvus et tardus waveform with a systolic acceleration time of ≥ to 0.1 seconds.[20, 21] A velocity ratio of the stenotic to pre-stenotic segments of greater than 2:1 is considered supportive of the diagnosis.

Multidetectors helical CT gives accurate assessment of the site and degree of TRAS and provides accurate and valuable imaging, requires less volume of iodinated contrast medium than digital subtraction angiography (DSA).[13]

The alternative is to perform MRI with gadolinium, a non-iodinated contrast medium which can provide accurate information about the anatomy of the urinary tract and vasculature of the kidney, and can be used to accurately estimate the selective GFR of each kidney.[22]

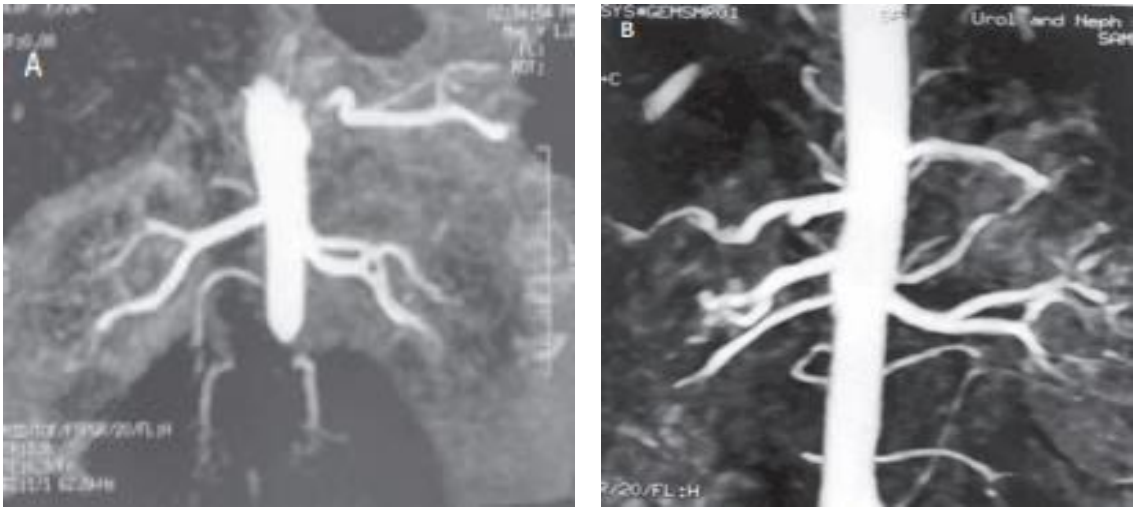



**Figure (1):** MRA in the identification of arterial supply. (A) Double left renal arteries with closed ostia on reformatted coronal oblique MIP image. (B) Triple left renal arteries and double right renal arteries on coronal MIP MRA.[22]

## Treatment

Treatment options for TRAS include both surgical and endoluminal options. Primary treatment for TRAS involves percutaneous transluminal angioplasty (PTA) with or without stent placement.[23, 24] The technical success rate of PTA has been reported to be as high as 94%, with a clinical success rate of 82%. [15] Recurrent stenosis may occur in more than 10%, and allograft loss has been reported in up to 30% of cases.[25]

There have been reports correlating TRAS with acute cellular rejection and that long term survival is significantly higher in non- TRAS patients compared with those had TRAS. Surgical revascularization is now considered rescue therapy and generally has been reserved for patients with disease unsuitable for PTA.[24, 26]

## Endoluminal Interventions

Since the introduction and the evolution of the endovascular interventions, there has been a shift in TRAS treatment option with Percutaneous Transluminal Angioplasty (PTA) with or without the use of stent being now the gold standard and the initial option of treatment.[27]

The type of arterial anastomosis that is present is the deciding factor in determining the angiographic approach utilized. If there is an end-to-end anastomosis with the internal iliac artery, done in living donor allografts, then a contralateral femoral approach is utilized to make access to the downward sloping artery as easy as possible. However, if there is an end-to-side anastomosis with the external iliac artery, then an ipsilateral femoral approach is preferred to access the cephalad sloping artery.[23, 27]

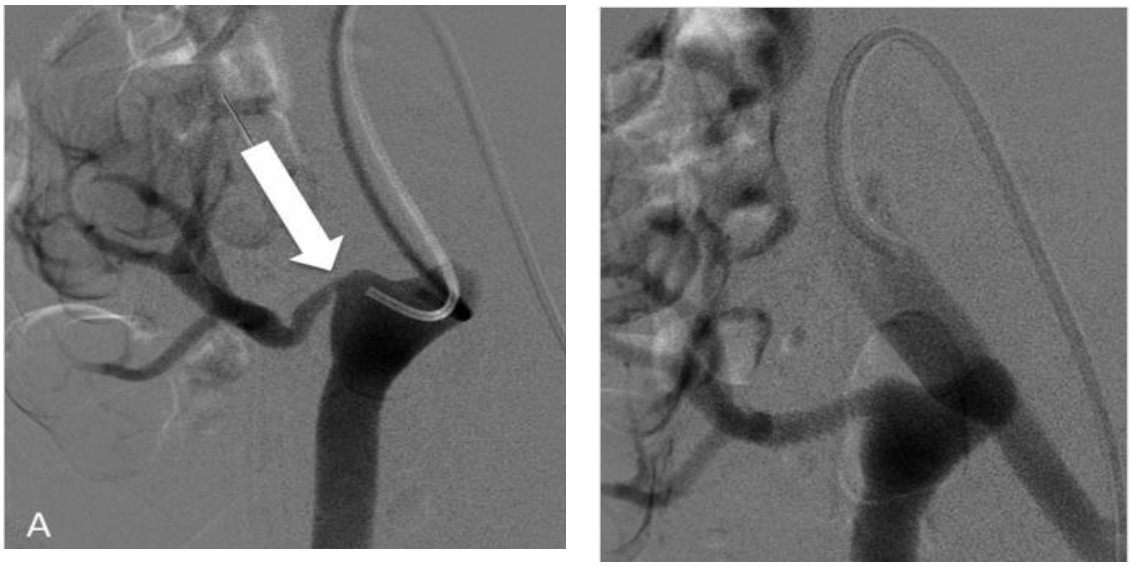



**Figure (2):** Percutaneous Transluminal Angioplasty
A-Arteriogram showing 75-80% anastomotic stenosis of the transplant renal artery at its junction with the external iliac artery (block arrow)
B-Arteriogram following stent deployment shows minimal residual stenosis
(https://www..intechopen.com)

Most of the complications are related to puncture site, but there could be also more severe complications like hemorrhage, rupture of transplant renal artery, iliac artery and loss of the allograft, in those patients there could be a need for "salvage" operation. Evolution in endovascular technology, with newer pre-mounted stents, has minimised complications especially the life threatening ones and the risk of allograft loss. Rate of re-stenosis are reported to be 10% to 50% and depends on the primary cause of the stenosis, length of follow-up, and use of stents.[28]

## Surgical Repair

Surgical correction of TRAS is regarded as a difficult operation with graft loss rates exceeding 20%.[23] A couple of risks existing to the recipient and to the allograft, the latter is not a contraindication to surgery, since severe TRAS could deteriorate the transplanted kidney, the patient proceeds to renal failure, and finally may end up in hemodialysis.[28]

In general, indications for surgery include: TRAS caused by kinking, anastomotic strictures and complex atherosclerotic disease. There are several options to treat TRAS; mostly excision of the stenosis with direct anastomosis to the external iliac artery and grafting with saphenous vein, recipient internal iliac artery. Reported surgical success rates range from 63% to 92%, with recurrence in 12% of patients.[26]

Limitations of surgical procedure are access to the artery and most importantly the subsequent warm ischemia time. A warm ischemia of 60 minutes might be tolerated by a kidney allograft that has been heparinized even though the risk of Acute Tubular Necrosis (ATN) and cortical necrosis is increased due to diminished blood flow. An alternative option even though rarely used, is back table reconstruction of a complex arterial problem and auto transplantation of the allograft.[28]

## Renal vascular thrombosis

The reported incidence of these rare complications that often results in graft loss ranges from 0.4% to 6%.[29, 30] Bakir et al reported that thrombosis represented 45% and 37% of renal allograft loss at 3 and 12 months.[31]

## Renal vein thrombosis

The causes that may lead to this serious complication include compression due to hematomas or lymphoceles, angulation or kinking of the vein, anastomotic strictures, or an underlying state of deep venous thrombosis or hypercoagulability.[32] Renal vein thrombosis (RVT) usually occurs suddenly and towards the end of the first week of an otherwise uncomplicated kidney transplantation.[33] Clinical presentation is initiated by oliguria and hematuria with a tender swollen graft, which if ruptured, is accompanied by life-threatening bleeding.[32, 33]

## Renal artery thrombosis

This uncommon complication which may occur most often as an early but also as a late event after kidney transplantation consists a devastating clinical condition leading frequently to graft loss.[32] Renal artery thrombosis (RAT) onset most often follows a technical problem such as intimal dissection, kinking or torsion of the vessels. Risk factors include poor cardiac output, hyper acute rejection, unresponsive acute rejection, and a hypercoagulable state.[34]

It presents with a rapid onset of oliguria. In cases of segmental infarct, the patient may be asymptomatic or presents with manifestations of uremia and hypertension. When (RAT) occurs as a late event, it could be attributed to renal artery stricture or its manipulation post-operatively during angiography, or usually due to graft rejection.[32]

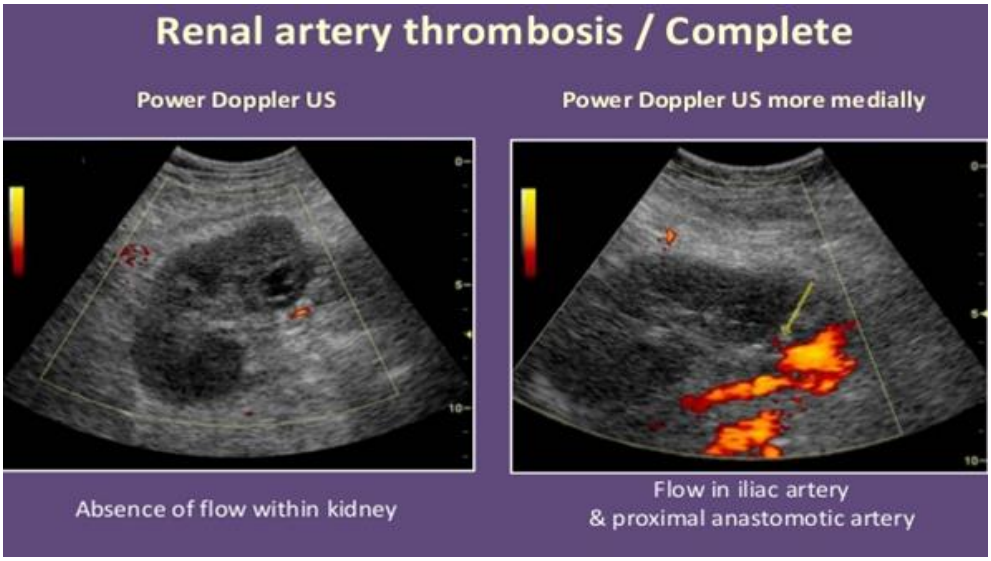



**Figure (3):** Color Doppler ultrasound showing renal artery thrombosis that is absence of blood flow to the kidney.[18]

## Diagnosis

An early clinical diagnosis is very important for both (RVT) and (RAT). Diagnosis of these complications is established by color flow Doppler studies, demonstrating in (RVT) a swollen graft with a crescent of clot along the convex margin of the kidney. In this case it is essential that the patient is taken immediately to the operative theatre.[20]

On the contrary of normal clinical conditions, lack of flow in the renal artery is demonstrated in (RAT), with the presence of intraluminal filling defects. In (RAT), diagnosis is set by Doppler studies or at time of surgical exploration, however by that time it is not possible for the graft to be saved due to the kidney's low tolerance to warm ischemia.[29]

## Treatment

Following establishment of diagnosis for RVT, the treatment of choice is urgent thrombectomy.[24] However graft salvage may not be possible, in such situation graft nephrectomy is usually required. In case thrombectomy is applied early, within 1 hour following the event, graft salvage can be achieved. The increased risk of swelling, edema and also a possible rupture of the kidney graft in such a condition, makes urgent exploration essential. Systemic anticoagulants can be applied as treatment only in cases of partial vein thrombosis.[35, 36]

The surgical treatment for renal graft thrombosis includes laparotomy, thrombectomy and ultimately a possible graft nephrectomy. Several authors describe endoluminal therapy for renal graft thrombosis; however the exact role of interventional radiologic treatment is not yet well-defined.[37] Trans catheter thrombolysis should be limited to low clot burden, segmental artery thrombosis, or high-risk surgical candidates.[24]

Additionally catheter-directed thrombolytics should be avoided in the first 2 weeks following kidney transplant due to the immature anastomotic suture line.[29]

## 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%. [3, 38] 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.[3, 38]

## Urinary fistulae and Urinoma

It can occur at the level of the bladder, ureter or renal calices. 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.[39] The leakage of urine can be collected around the graft, extends to the retro peritoneum, scrotum or may manifest through the incision.[40]

The average prevalence in many studies is around 5.7%. In general, most urinary leaks are the results of ureteral problems, failure of ureterovesical anastomosis or ischemia and necrosis of the distal ureteral stump.[41]

## Clinical presentation:

In most cases, there is constant discharge of clear liquid through the drain, in the immediate postoperative period. When later, after removal of the drain, there may be iliac swelling with extension into the perineum and scrotum or decreased urine output with maintenance of renal function. Unexplained graft dysfunction, pelvic fluid collection, fever, graft tenderness, lower limb edema can also occur.[39]

Early urinary leaks can be divided into two types: the first usually occurs within the first 1 to 4 days and is almost always related to technical problems with the implantation. In this case, the ureter has usually pulled out of a tunnel caused by excessive tension at the anastomosis. This complication appears to be more common with the extravesical ureteroneocystostomies.[42]

The second type of early ureteral leak, usually presents between 5 and 10 days, is associated with distal ureteral ischemia, which may be a consequence of injury during the donor nephrectomy, technical causes such as tunnel hematoma or distal stripping of the blood supply.[43]

## Diagnosis:

For being the most common surgical complication of kidney transplantation, urinary fistula is easily diagnosed.[43] In doubtful cases, where there is need to exclude the lymphocele as main differential diagnosis, biochemical analysis of the liquid is characterized by having elevated levels of creatinine, urea and potassium. In the lymphocele, creatinine should be similar of blood. Urinary leak are often suspected because of increased drainage from the wound.[39]

Radiographic tests of help include an abdominal ultrasound and nuclear renal scan. The ultrasound is nonspecific for evaluating patients with suspected urinary fistula after kidney transplantation. It will only reveal a fluid collection (anechoic image) around the graft.(44)

A renal scan demonstrating extravasation is the most sensitive method to differentiate a urine leak from other fluid collections such lymphoceles or hematomas.(45)

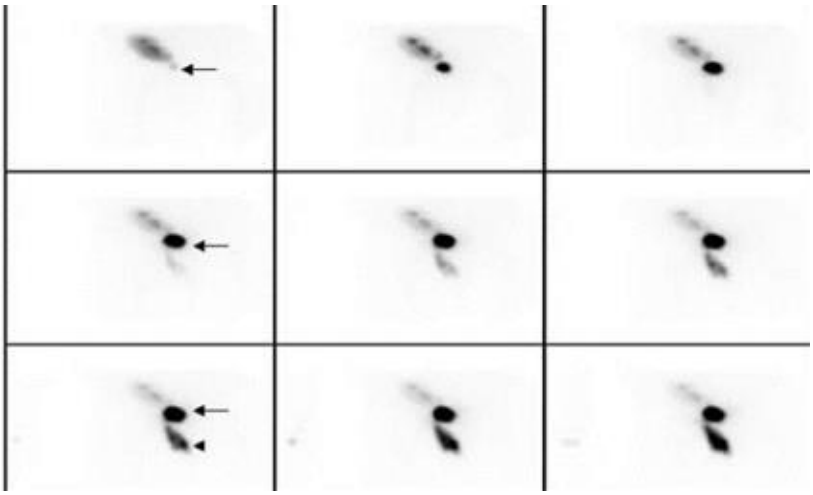


**Figure (4)**:**Renal isotope scan of transplanted kidney**; normal perfusion and function with excretion of radiotracer into the renal collecting system and a focal collection mimicking the urinary bladder (arrows). There is a vertically oriented accumulation of radioactivity inferior to the pelvis (arrow head). (www.biomedcentral.com)

## Management of the urinary fistulae:

Urinoma in a renal transplant patient requires rapid diagnosis and treatment. Ureteral leakage needs careful and accurate diagnosis of the exact cause and site. It is important to know if the problem has a surgical cause or associated with an acute rejection episode that required specific treatment.(39)

Surgical treatment has to be performed in all patients except those presenting with minimal extravasation at the ureteral reimplantation site and clinically stable. This group was initially treated by urinary drainage.(40)

Surgery is the initial approach for sever extravasation or when mid or upper ureteral leaks were suspected. The type of surgical reconstruction is based on the intraoperative evaluation of the extent of the ureteral necrosis and local and systemic condition of the patient at the time of surgery.(46)

Primary reconstruction with the ureter of the recipient or a new ureteral reimplantation are performed preferentially when local and systemic conditions allowed.(46)

All patients received prophylactic or therapeutic antibiotic according to the antibiogram of the collected fluid.(46)

The need for immediate open surgical intervention has been replaced, to a large extent, by early endourologic intervention.[47] The placement of a percutaneous nephrostomy can divert a leak or relieve obstruction and allow more definitive diagnosis. Percutaneous techniques like nephrostomy and ante grade ureteral stenting works in 40% of a much selected group of patients presenting with small fistulae from the distal ureter.[40, 48]

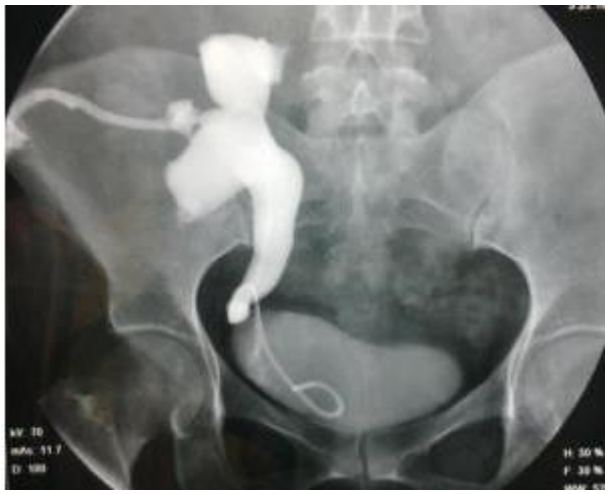


**Figure (5): Nephrostogram one month after transplant** revealed obstruction at the ureteroneocystostomy level inspite of the presence of JJ stent (https://www.wjnu.elmerpress.com)

## Urinary Obstruction

Obstruction may occur during the early postoperative course due to blood clots, ureteral malrotation or kinking, tight sub mucosal tunnel, unsuspected donor calculus[49] or perigraft fluid collection.[50,51] Late ureteral obstructions generally after the first month or even at years post-transplant are secondary to chronic ischemia which leads to chronic fibrosis and strictures.[52] Other causes include compressive lymphoceles or pelvic masses, ureteral calculi and rarely obstruction by ureteral carcinoma[53] or fungus ball.[54]

The clinical presentation includes pain over the surgical site, decreased urine volume leading to oliguria and rise in blood pressure secondary to impaired renal function with gradual rise in serum creatinine. The ultrasound demonstrates hydronephrosis or hydroureteronephrosis in most of cases.[55] Nuclear scintigraphy is less sensitive because the obstructed kidney also displays impaired radionuclide uptake, a sign often present in allograft rejection.[45] When the diagnosis is unclear, the ante grade pyelogram must be performed, because it is an accurate method to define anatomically the site, degree of obstruction.[48]

The treatment must be instituted as early as possible to avoid loss of renal graft function. Initially the percutaneous nephrostomy must be done to ensure the urinary drainage and restore renal function to normal.[47, 48] The definitive treatment of the obstruction is planned according to the etiology. Ureteric stricture at the site of reimplantation is more common and can be addressed by several endourologic techniques such as ureteral meatotomy or ureteral dilation with balloon. Such techniques are at acceptable levels of success especially when treat small lesions.[47, 48] However, open surgery with reconstruction is still considered the gold standard. In distal ureteral obstructions or when there is redundant ureter, we can review the ureteroneocystostomy by either extravesical or intravesical techniques. [46, 56]

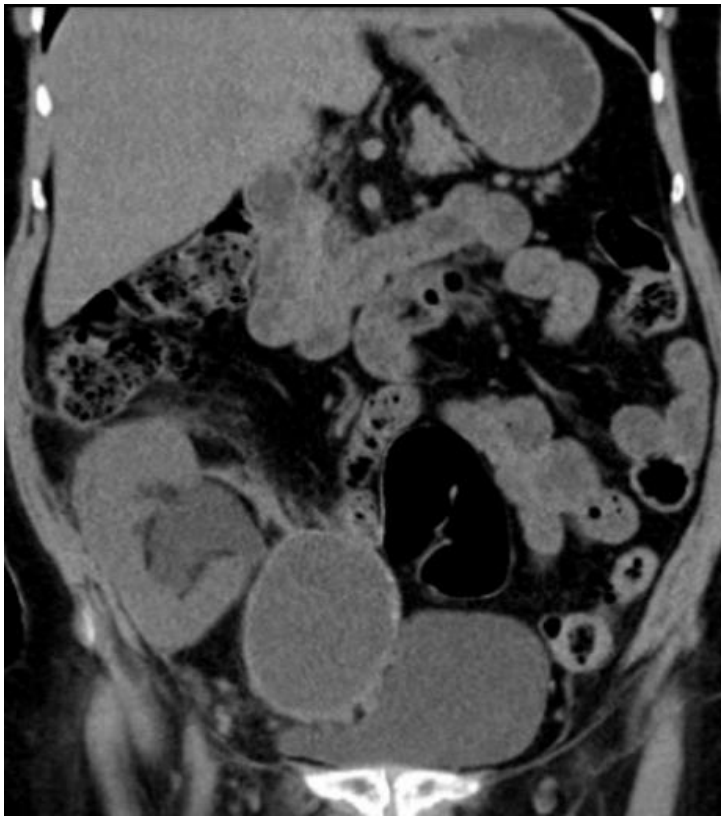
When there are multiple, long stenosis of the ureter or even poor vascularization, it is necessary to perform the anastomosis of the renal pelvis with the native ureter (ureteropyelostomy) or the ureter with the native ureter (ureteroureterostomy).[46, 57] However, the last technique has a higher rate of stenosis. When the native ureters cannot be used, "Boari flap" should be done joining the short ureteral stump or the renal donor pelvis, allowing an adequate distance to the bladder.[51] This allows tunneling the flap under the ureter, decreasing reflux and bacterial contamination during episodes of infection at the lower urinary tract.[58] Extreme situations may require a pyelovesicostomy with anastomosis the donor urinary pelvis directly to the bladder.[51] In this circumstance there is direct transmission of voiding pressure to the urinary collecting system as well as any urinary infection, leading to chronic pyelonephritis and deteriorating renal graft.[59]

## Lymphocele

Lymphocele is a lymph collection from the iliac lymphatic vessels of recipient or graft hilum that accumulates between the transplanted kidney and bladder. The average incidence of lymphocele in the literature ranges from 0.6 to 16%.[60-62] The etiology has been attributed to inadequate ligation of the delicate lymph vessels overlying the iliac vessels or present in the graft hilum. The method of donor nephrectomy also appears to influence the appearance of lymphatic complications. The laparoscopic nephrectomy may prolong the lymphatic leak requiring drain for a longer period.[63]

The small lymphoceles are more frequent but usually asymptomatic.[64] However, large ones are manifested clinically in a few weeks to months after transplantation, bulging can occur in the surgical wound with or without cutaneous extravasation of lymph. In severe cases, there may be edema of ipsilateral lower limb, frequent urination due to bladder compression and ureteral obstruction leading to hydronephrosis and loss of renal graft function.[62]

The diagnosis is confirmed by ultrasound which may show hydronephrosis, altered vascular flow by Doppler and quantify the lymphocele or the presence of other collections such as hematoma or urinoma.[64] In doubtful cases, a computerized axial tomography (CT) can be performed following puncture of the collection guided by CT or ultrasound (US) with biochemical analysis of the liquid obtained.[61]



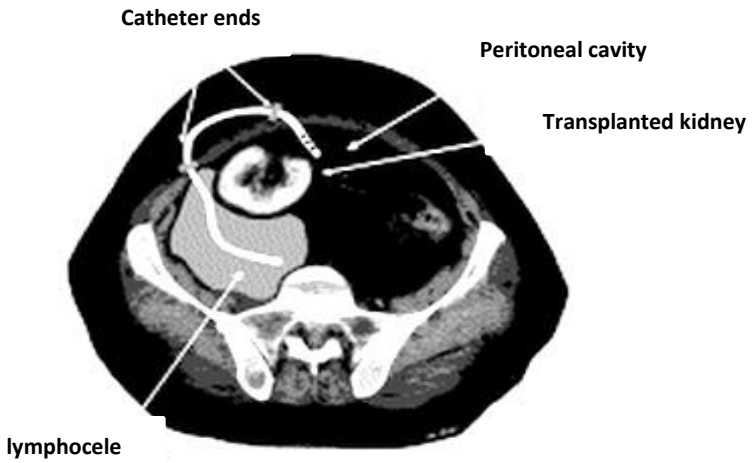
**Figure(6):** Non-contrast CT abdomen and pelvis shows lymphocele compressing both bladder and the graft causing moderate hydronephrosis. (www.thirdeye-radiologysite.blogspot.com)

The treatment can be divided into observation, puncture and drainage or surgery. The small volumes of lymphocele with less than 140ml and asymptomatic, tend to resolve spontaneously without impact on graft function. Larger collections or lymphoceles with clinical manifestations can be punctured and drained, under strictly aseptic techniques and guided by US or CT.[61, 64] If there is clinically significant recurrence, a sclerotherapy with povidone-iodine, 5% ethanol or antibiotics can be performed.[65, 66]

In lymphoceles larger than 500 ml, punctures, drainages and sclerotherapy are usually not effective. In refractory or complicated cases, the laparoscopic lymphocele fenestration (Marsupialization) is the procedure of choice in many centers.[67] The laparoscopic technique reduces the risk of injury to the ureter or infection and demonstrates high rate of success. It is important to create a window sufficiently large to ensure complete resolution.[67]

When the location is not favorable to laparoscopy or in recurrent cases, the approach by open surgery should be performed, especially when the lymphocele is located posterior and lower to the transplanted kidney or behind the bladder.[66, 67] In both techniques it is recommended to perform large peritoneostomy with an oval window of at least 2.5 x 5.0 cm in width associated with interposition of short segment of omentum, allowing a good peritoneal absorption of lymphocele and avoiding internal hernias of the bowel segments.[67]

Recently was reported the treatment of recurrent and symptomatic lymphocele by inserting a Tenckhoff catheter at the site of lymphocele tunneled to the abdomen, allowing the intraperitoneal drainage.[60] This procedure offers an advantage of being performed in outpatient clinic, without general anesthesia, with good efficacy and safety, however the number of cases reported is still small.[68]




**Figure(7):** Intraperitoneal Tenckhoff catheter for the treatment of recurrent lymphoceles after kidney transplantation[68]

# OTHERS

## Abscesses and Infection

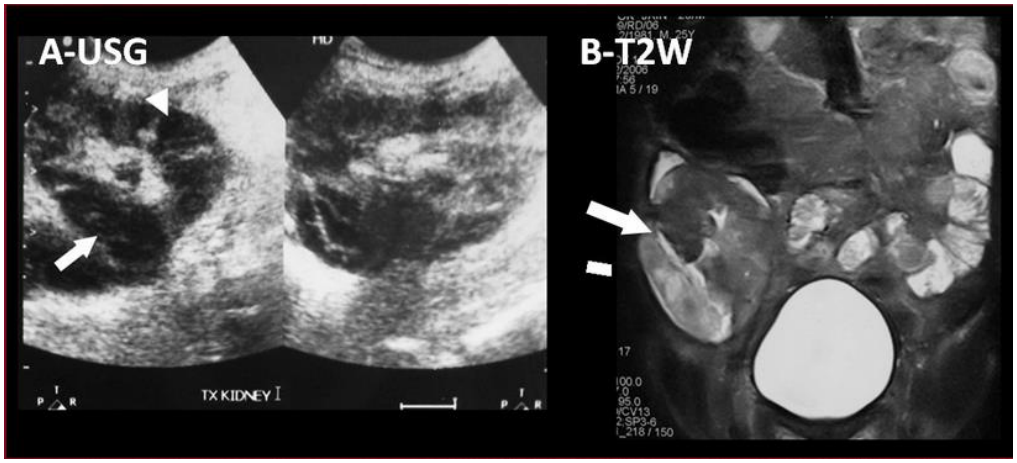
More than 80% of renal transplant recipients suffer at least one episode of infection during the first year after transplantation. Early diagnosis and intervention for infectious diseases can prevent loss of graft function and improve patient outcome.[69-71]

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 non immunocompromised patients who have undergone surgery.[72] Infections with opportunistic pathogens and cytomegalovirus often develop 1–6 months after surgery. [70, 73]

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 contamination of a lymphocele, hematoma, or urinoma. Acute bacterial nephritis, renal abscess, or perinephric abscess may occur.[58]

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 kidney.[59] In a febrile transplant recipient, any peritransplant fluid collection must be presumed to be infected.[74]

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. Abscesses have a complex, cystic, nonspecific appearance at US.[75, 76] 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.[77]




**Figure(8):Diagnostic modalities of post-transplant abscesses and infections**

(A)USG of a 25 year old transplant patient shows multiple hypoechoic lesions within the cortex(arrowhead A) with one large lesion laterally (arrow A)

(B)MR shows multiple hyper intensities in the renal cortex and a large well defined abscess laterally (arrow B).

(Department of Radiology, All India Institute of Medical Sciences, New Delhi, 2012)

Any echogenicity within a dilated collecting system is usually clinically significant. The presence of low-level echoes in a dilated pyelocaliceal system in a febrile patient suggests pyonephrosis.(78)

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.(79)

Herein this study will address all kidney transplant recipients and their living donors evaluating the pre-operative characteristics, intra-operative findings and post-operative period to report surgical complications and their impact on patients outcomes.