

# Management of the patient who has had a kidney transplant in the medical assessment unit

## Abstract

The number of people with kidney transplants has increased rapidly over the last 20 years. They are often medically complex and have a significant need for both routine and urgent care. Patients who have received a kidney transplant can be challenging to manage in the medical assessment unit. They are vulnerable to infections and acute kidney injury; disease presentation and course may be atypical and they are at risk of rapid deterioration. This review describes a systematic approach to their assessment and management and highlights specific considerations to be borne in mind.

**Key words:** Acute kidney injury; Emergency; Infection; Kidney; Transplant

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## Background

There are nearly 36 000 recipients with functioning kidney transplants in the UK, representing 55% of the population with end-stage kidney disease (UK Renal Registry, 2020). Prevalent kidney transplant recipients in the UK have a median age of 54 years and 60% are male (Pyart et al, 2018). Transplantation is a treatment modality rather than a cure – recipients are required to take immunosuppression to prevent rejection and remain under lifelong follow up. They may have significant comorbidities, some of which may be related to the legacy effect of previous end-stage kidney disease or treatment of their underlying diagnosis. Transplantation does not normalise kidney function, and 16% recipients have a baseline estimated glomerular filtration rate <30 ml/minute (chronic kidney disease stage 4 or 5) (Pyart et al, 2018). An estimated 1 in 4 recipients have diabetes (Peev et al, 2014; Schold et al, 2016).

In the USA and Europe, transplant recipients have been shown to have a significant requirement for emergency care (Li et al, 2016; King et al, 2017; Arms et al, 2018). The organisation of follow up and supervision of recipients in the UK reduces the requirement for attendance at emergency departments, but the symptoms leading to presentation and high admission rates are likely to be similar. Transplant recipients typically have a close relationship with their renal unit and ready access to advice via telephone, email or face-to-face consultation. They are well informed about their health, their usual level of kidney function and treatment, including the importance of adherence to and timing of immunosuppression. Anxiety about their transplant at the time of acute illness often colours their consultation.

This article focuses on the most common diagnostic dilemmas and specific considerations to be made when reviewing patients with a kidney transplant who present to the medical assessment unit (Table 1). Liaison with the local team responsible for ongoing care is essential for all transplant recipients.

## Infection

Infection is the leading cause of emergency presentation in transplant recipients, and the third commonest cause of death in this group (19%, after malignancy (26%) and cardiac disease (21%); Fishman, 2017). Up to 80% of recipients may experience a significant infection in the first year following transplantation (Zhong and Liang, 2018). These include both infections which may also occur in immunocompetent individuals and opportunistic infections consequent to immunosuppression (Table 2). Recipients are most vulnerable

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**Table 1. General assessment of the recipient of a kidney transplant**

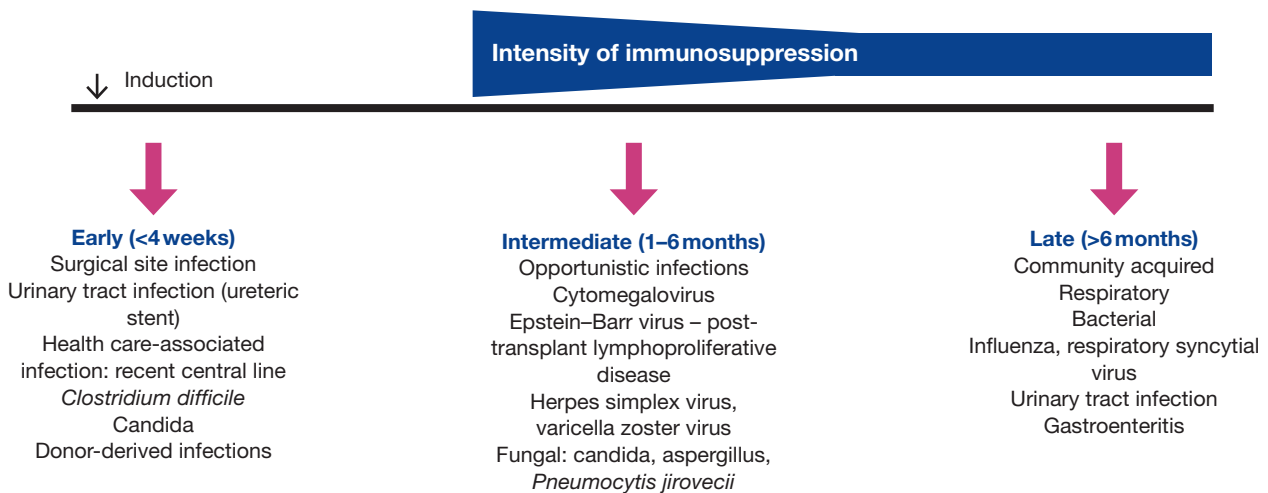
History	Symptoms of intercurrent illness	Infection Lower urinary tract symptoms Pain over graft
	Fluid status	Intake, loss (eg diarrhoea)
	Drugs	Changes – especially antibiotics (macrolides), antifungals, over the counter medication including non-steroidal anti-inflammatory drugs and St John’s wort  Adherence
	Transplant	Primary renal diagnosis When and where was the transplant Previous complications Surgical Rejection Infection Recurrent disease
	Other	Foreign travel
Examination	General	Temperature, lymphadenopathy
	Fluid status	Under- or over-hydration Urine output
	Transplant	Tenderness, masses, bladder distension
Investigations	Blood tests	Routine admission blood tests – full blood count, urea and electrolytes, bicarbonate, liver function tests, bone function, C-reactive protein  Calcineurin inhibitor levels (trough level taken just before morning dose – ethylenediaminetetracetic acid (EDTA) sample. Do not delay giving morning dose)
	Urine	Urinalysis
	Infection screen (guided by localising symptoms)	Blood, urine, sputum, stool cultures Viral throat swab, consider <i>Pneumocystis jirovecii</i> Additional blood tests: beta-D-glucan, polymerase chain reaction for cytomegalovirus and Epstein–Barr virus
	Imaging (as indicated)	Chest X-ray  Ultrasound transplant kidney and bladder (+/- Doppler studies of the transplant artery where appropriate expertise is available)

to opportunistic infections in the first 6–12 months following transplantation, when immunosuppression is most intense, but these infections may also develop at later time points (Fishman, 2017; Fishman et al, 2019) (Figure 1). The inflammatory response may be impaired by immunosuppression, resulting in an atypical presentation with absence of fever and leucocytosis. Early recognition of infection is crucial, with appropriate diagnostic testing and empirical antimicrobial therapy. There is the potential for rapid deterioration as a result of infection and a low threshold for admission is generally recommended (Fishman, 2017). Transplant recipients will have had significant exposure to medical environments and antimicrobials, therefore consideration should be given to resistant infections including vancomycin-resistant Enterococci, carbapenemase-producing Enterobacteriaceae, extended-spectrum beta lactamase *Escherichia coli* and meticillin-resistant *Staphylococcus aureus*.

Although fever in a transplant recipient is most likely a result of infection, there are mimics. A hectic fever frequently accompanies post-transplant lymphoproliferative disease (discussed below), and may occur in the context of graft rejection.

**Table 2. Common infections in patients who are immunosuppressed**

System affected	Pathogen	Symptoms and clinical features	Diagnosis
Constitutional illness	Cytomegalovirus	Fever, weight loss, diarrhoea, dyspnoea  Usually occurs in a cytomegalovirus-naive recipient of organ from a cytomegalovirus-positive donor after completion of antiviral prophylaxis	Cytomegalovirus polymerase chain reaction (whole blood)
	<i>Candida</i> spp.	Fever, weight loss, organ-specific symptoms such as dysphagia or retrosternal pain	Blood cultures Beta-D-glucan
	<i>Mycobacterium tuberculosis</i>	Fever, cough, weight loss, lymphadenopathy, culture-negative pyuria	Chest X-ray (+/-computed tomography)  Sputum – smear for acid-fast bacilli, culture, polymerase chain reaction  Interferon gamma release assay
	Epstein–Barr virus	Hectic fever, lymphadenopathy	Epstein–Barr virus polymerase chain reaction (whole blood)
Respiratory	Viral – including influenza, respiratory syncytial virus, adenovirus, coronavirus (including severe acute respiratory syndrome coronavirus 2)	Fever, dyspnoea, flu-like symptoms. Risk of rapid deterioration and requirement for respiratory support	Chest X-ray Viral throat swab
	<i>Pneumocystis jirovecii</i> pneumonia	May have abrupt or more indolent onset. Fever, general malaise, progressive dyspnoea, hypoxia, extensive changes on chest X-ray	Chest X-ray <i>Pneumocystis jirovecii</i> pneumonia throat swab  Blood beta-D-glucan levels Bronchoalveolar lavage
	Aspergillus	Fever, chest pain, dyspnoea, haemoptysis  Rhinosinusitis  (CNS involvement, endophthalmitis, endocarditis)	Chest X-ray (+/-computed tomography)  Aspergillus polymerase chain reaction
Urinary tract	Bacterial	May be associated with ascending infection and graft pyelonephritis	Culture mandatory – resistant organisms common
Gastrointestinal	Oropharyngeal candida	Fever, weight loss, source specific symptoms such as dysphagia or retrosternal pain	Examination of mucosal surfaces, endoscopy
	<i>Clostridium difficile</i>	Abrupt onset diarrhoea, prior history of antibiotic exposure	Stool culture
	Cryptosporidium	Abrupt onset diarrhoea	Stool culture
	Norovirus	Diarrhoea, may become chronic with fluctuating severity	Stool culture
	Hepatitis E	Diarrhoea, may be preceded by acute transient jaundice or derangement in liver function tests	Serology (immunoglobulin G and immunoglobulin M) and hepatitis E polymerase chain reaction (stool)



**Figure 1.** Time course of infections following transplantation.

A reduction in immunosuppression may be considered for patients with severe infection but this should only be done in conjunction with the transplant team, as a break in treatment carries a risk of rejection. The usual reduction is the dose of antiproliferative drug (mycophenolate mofetil or azathioprine). Calcineurin inhibitor (tacrolimus or ciclosporin) levels should be monitored (trough level taken pre-dose, ethylenediaminetetraacetic acid (EDTA) blood sample), particularly if the patient has diarrhoea, as this can result in an increase in calcineurin inhibitor levels. Steroids should never be stopped abruptly. The dose is often doubled in the acute setting to compensate for adrenal suppression following long-term treatment.

### Respiratory infection

Immunosuppression results in impaired cell-mediated immunity, conferring a vulnerability to viral and fungal opportunistic infections in addition to more conventional bacterial causes. Evidence is accumulating that infection with the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in transplant recipients is associated with high mortality (Ju et al, 2020).

### Urinary tract infection

Urinary tract infections are very common in transplant recipients, who are most vulnerable in the first year following transplantation (Lee et al, 2013). The potential causative organisms include Gram-negative bacilli, Gram-positive cocci and *Candida* spp. There is a high prevalence of antibiotic resistance and review of previous culture results may help guide treatment choice. The transplant ureter is very short and ascending infection with graft pyelonephritis is also common. This may present as pain over the transplant with minimal lower urinary tract symptoms. A ureteric stent may have been placed at the time of transplantation. These are usually removed after 6 weeks, but in the early post-transplant period may contribute to the risk of ascending infection.

Acute rejection is usually asymptomatic but may mimic pyelonephritis with pain over the graft.

All urinary tract infections in transplant recipients are considered ‘complex’ and clearance after a short (3–5-day) course of antibiotics is unlikely. A treatment course of 10–14 days is recommended in the first instance (Fishman, 2017; Fishman et al, 2019). The antibiotic of choice is guided by renal function and the availability of previous urine culture results. For example, trimethoprim may be appropriate unless there are concerns about the potential to cause hyperkalaemia and an increase in creatinine levels. Nitrofurantoin can only be used if the estimated glomerular filtration rate is >60ml/minute.

## Gastrointestinal infection

Post-transplant diarrhoea is common and can lead to dehydration and pre-renal acute kidney injury. Frequent causes in transplant recipients include cytomegalovirus, norovirus, *C. difficile*, Cryptosporidium and, less commonly, chronic hepatitis E infection. Norovirus may persist and lead to a chronic infection and symptoms. Diarrhoea is a frequent side effect of mycophenolate mofetil and may slow resolution of symptoms after an acute infection.

## Viral infections

Infection caused by cytomegalovirus or Epstein–Barr virus should be considered in patients with constitutional symptoms such as fever, fatigue, weight loss and myalgia. These viral infections may be associated with leucopenia and hepatitis. Cytomegalovirus can also cause pneumonitis. Transplant recipients typically receive prophylaxis against cytomegalovirus infection with valganciclovir for the first 3–6 months following transplantation. Their greatest risk of infection is following cessation of prophylaxis, particularly if a cytomegalovirus-naïve recipient has received a transplant from a cytomegalovirus-positive donor.

Varicella zoster and herpes simplex virus infections may result in severe symptoms in immunosuppressed individuals. Transplant patients are also more prone to influenza and annual vaccination is recommended.

## Acute kidney injury

Graft dysfunction is very common in the presence of any intercurrent illness. Recipients have a reduced functional reserve and limited capacity for autoregulation at times of physiological stress (Ronco et al, 2019). They may also have impaired urine concentrating ability, further increasing their risk of pre-renal acute kidney injury. The initial assessment is similar to that of any patient presenting with acute kidney injury, with additional consideration of transplant-specific causes (Table 3). A review of medications is particularly important, including any changes, compliance with immunosuppression and any evidence of infection.

**Table 3. Causes of acute graft dysfunction**

Pre-renal disease	Hypovolaemia	Denervated kidneys have a fixed urine output. Inadequate hydration may lead to acute kidney injury
	Hypotension	May be caused by dehydration, sepsis, heart failure or medication (over-treatment of high blood pressure)
	Hypoperfusion	Transplant renal artery stenosis
Intrinsic renal disease	Transplant specific	Acute or chronic rejection. Acute rejection may present with symptoms such as pain over graft and fever, especially in the context of poor adherence to immunosuppression. More commonly rejection is asymptomatic with an acute deterioration in function
	Recurrence of underlying disease	This may occur with certain forms of glomerulonephritis, for example IgA nephropathy, focal segmental glomerulosclerosis and membranoproliferative glomerulonephritis
	De novo glomerular disease	Glomerulonephritis may unusually develop in the transplant despite no prior history of this in the native kidneys
	Interstitial nephritis	Related to medications such as antibiotics, proton-pump inhibitors and non-steroidal anti-inflammatory drugs
Post-renal	Obstructive	Blocked stent, stenosis of the transplant ureter or ureteric anastomosis to bladder, bladder outflow obstruction
		Calculi, extrinsic compression, for example postoperative fluid collection, urinoma, lymphoma

The immediate management of acute kidney injury in these patients involves:

- Assess fluid status
- Give replacement fluids if indicated – oral or intravenous crystalloid
- Monitor urine output
- Treat any underlying infection
- Reassess – fluid status and renal function
- Exclude obstruction – ultrasound
- Consider transplant-related complication (vascular, obstruction, rejection).

## Cardiac disease

Dialysis patients have a markedly increased risk of developing cardiovascular disease compared to the general population (Foley et al, 1998). Although this risk is reduced by transplantation, a kidney transplant recipient aged 25–44 years has a 10-fold increase in cardiovascular mortality compared to the general population. The risk is influenced by conventional cardiovascular risk factors, which may themselves be modulated by the chronic effects of calcineurin inhibitors and steroids, previous duration of dialysis and ongoing chronic kidney disease (Stoumpos et al, 2015; Sharif and Cohn, 2016). This means that a high index of suspicion must be used in assessing symptoms suggestive of coronary, peripheral or cerebral vascular ischaemia even if the patient is young.

## Malignancy

Renal transplantation substantially increases the risk of certain cancers, particularly those driven by viral infection, which may occasionally present acutely. This increased incidence rises with intensity and duration of immunosuppression. A high index of suspicion for potential malignancy should be maintained, even in young patients. Post-transplant lymphoproliferative disease is commonly associated with Epstein–Barr virus infection. Post-transplant lymphoproliferative disease should be suspected if symptoms such as night sweats, weight loss and lymphadenopathy are present. Epstein–Barr virus replication is detected by viral PCR on whole blood, although lymphoma may also develop without an association with Epstein–Barr virus. The course of Epstein–Barr virus-driven disease can vary significantly from a relatively indolent presentation which responds to a reduction in immunosuppression to a more fulminant disease requiring chemotherapy. Treatment decisions are also informed by the histology of a lymph node biopsy.

## Drugs: complications of immunosuppression and common interactions

The intensity of immunosuppression is greatest immediately following transplantation, because of the administration of induction treatment. This usually includes inhibition of the interleukin 2 receptor with basiliximab, or T cell depletion with anti-thymocyte globulin or alemtuzumab. Recipients continue lifelong maintenance with a combination of drugs, although the dose of these may reduce in the longer term. The most common maintenance regimen for patients who have had a kidney transplant includes a calcineurin inhibitor (usually tacrolimus) and an anti-proliferative agent (usually mycophenolate mofetil), with or without steroids (prednisolone). The common side effects are listed in [Table 4](#). Immunosuppressants also have important interactions with other drugs. Calcineurin inhibitors are metabolised by the oxidative cytochrome P450 enzymes and any drugs that inhibit or induce those proteins will have an impact on tacrolimus levels ([Table 5](#)), so advice from the transplant team and pharmacist should be sought before considering starting a patient on such medications.

## Surgical complications

Surgical complications are usually seen within the first few days to months post-transplantation. Any presentation of these will need prompt discussion with the transplant team. Wound infection is more common in patients who are obese and such infections

**Table 4. Common side effects of immunosuppression**

Class of drug	Drug	Side effects
Calcineurin inhibitor	Tacrolimus	Nephrotoxicity Neurotoxicity (tremor, disturbed sleep, headaches) Hair loss Post-transplant diabetes ↑K <sup>+</sup> , ↓PO <sub>4</sub>
	Ciclosporin	Nephrotoxicity Hirsutism Gingival hypertrophy Hypertension
mTOR inhibitor	Sirolimus	Mouth ulceration Pneumonitis Impaired wound healing Increased risk of lymphocele
Antiproliferative	Mycophenolate	Leucopenia Gastrointestinal effects – particularly diarrhoea (causation as well as prolongation of symptoms) Hair loss
	Azathioprine	Leucopenia Pancreatitis Hepatitis
Corticosteroid	Prednisolone	Cushingoid features (buffalo hump, moon face, centripetal obesity, proximal myopathy, bruises, diabetes, hypertension, poor wound healing)

**Table 5. Commonly prescribed drugs affecting tacrolimus bioavailability and levels**

Increases tacrolimus level	Macrolide antibiotics (eg erythromycin, clarithromycin)
	Antifungals (eg fluconazole)
	Grapefruit juice
Decreases tacrolimus level	Rifampicin
	Carbamazepine
	Phenytoin

may be superficial or related to a deeper collection. Patients may present with erythema or tenderness over the wound, with or without discharge, and fever. Ultrasound is helpful to determine any underlying collections. Treatment is antibiotics and drainage of any collections. If there is discharge, a microbiology swab must be sent. Clear discharge from the wound or in the drain from any collection may be a sign of urine leak (often a result of leakage at site of ureteric anastomosis to the bladder). If this is suspected, a urinary catheter is recommended with prompt referral to the transplant team. The patient will need further evaluation to identify the potential site of leak and further management is likely to be ureteric re-implantation. A more common cause of a collection and/or clear discharge is a lymphocele, which occurs in as many as 20% of patients post-transplant. The majority of these are asymptomatic and resolve spontaneously. Occasionally they

### Key points

- Involve the patient's transplant team in management.
- Do not omit immunosuppression without specific advice from the transplant team.
- Ensure the patient has adequate hydration.
- Immunosuppression may mask signs of infection, there is a risk of rapid deterioration.
- Consider the acute presentation in the context of underlying diagnosis and immunosuppression.
- The patient usually knows what they are talking about.

require percutaneous drainage or surgical fenestration. Both urine leak and lymphocoeles may present very similarly and the urea and electrolytes will distinguish between them (lymphocoele – urea and creatinine similar to serum value; urine leak – urea and creatinine much higher than serum value).

Ureteric obstruction usually presents with a decline in kidney function and hydronephrosis is seen on the ultrasound. Causes include extrinsic compression from a collection or a ureteric stricture. These patients require urinary catheter and percutaneous nephrostomy to relieve the obstruction. Following this, a nephrostogram will demonstrate the site and size of stricture and guide further management (either radiological dilatation or surgical intervention).

Renal artery thrombosis is fortunately rare (<1%), as is renal vein thrombosis (2–3%). These usually happen during the index admission of the transplant. Patients develop oliguria or anuria with a decline in kidney function and pain over the transplant. Diagnosis is confirmed by ultrasound Doppler and often it is too late to salvage the kidney. Transplant renal artery stenosis is more common (1–27%) (Fervenza et al, 1998) and typically presents 6 months post transplantation with hypertension and decline in kidney function (especially after introduction of an angiotensin-converting enzyme inhibitor). It can be diagnosed by ultrasound Doppler or computed tomography angiogram, and initial treatment involves radiological angioplasty.

Although transplant recipients will present with general surgical problems, for example right iliac fossa pain suggestive of acute appendicitis, or left iliac fossa pain suggestive of acute diverticular disease, the location of the transplant must be borne in mind when considering the correct diagnosis. An urgent transplant ultrasound with Doppler studies must be organised in addition to the standard blood tests and urinalysis, followed by a prompt referral to the transplant surgical team. Biliary disease, including biliary colic, acute cholecystitis or pancreatitis, must also be managed jointly by the transplant and hepatopancreatobiliary surgeons. Transplant patients who present with bowel obstruction should be managed with nasogastric tube decompression, urinary catheterisation, careful fluid balance monitoring, and an early computed tomography scan (to look for pathology such as post-transplant lymphoproliferative disease). When arranging computed tomography imaging, there is often concern about the use of intravenous contrast in patients with impaired renal function. The risk of contrast-induced nephropathy causing acute kidney injury has previously been over-stated (van der Molen et al, 2018), and contrast should be administered if required to clarify diagnosis. Caution is also required when considering blood transfusion, to avoid the potential development of HLA antibodies which can increase the risk of rejection (Weinstock and Schnaidt, 2019). The need for blood transfusion should be advised by the clinical scenario and given if required.

### Conclusions

The assessment of a patient who has received a kidney transplant is done in the context of the time since transplant, renal function and immunosuppression, all of which will have an impact on their disease presentation and course. Atypical presentations are common, both

in terms of the constellation of symptoms and age of the patient. Transplant patients are vulnerable to acute kidney injury and at risk of rapid deterioration if infection is present. Hospital readmissions are common and have been reported to be associated with adverse long-term outcomes. Close liaison with the transplant team is essential – although the patient will often beat you to it and call them first.

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#### Conflicts of interest

The authors declare no conflicts of interest.

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