Surgical complications of renal transplantation

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Haemorrhage A haematoma may develop in the transplant bed in the first few postoperative days. This is often due to bleeding from small unsecured vessels in the renal hilum that were not apparent at the time of surgery . In this situation the patient is haemody namically stable and can be investigated by CT angiography and subsequently re-explored to remove the haematoma. Anastomotic haemorrhage can also occur but is very rare. It presents as haemorrhagic shock associated with a fall in the haemoglobin to <50 /uni00A0 g/L. This is a life-thr eatening situation and the patient should be returned to the operating theatre immediately . It may be possible to repair a defect in an arte rial or venous anastomosis but transplant nephrectomy is often required. Late anastomotic haemorrhage can also occur after some weeks or months owing to the development of a m ycotic aneu rysm. The commonest causative or ganism is donor-derived Candida albicans . A ruptured mycotic aneurysm requires an immediate graft nephrectomy and has a significant mortality . - Although this occurs in <1% of renal transplants, the most common outcome is loss of the allograft. Most cases are due to technical complications at the arterial anastomosis. The usual presentation of renal artery thrombosis is sudden anuria and a rapid decline in renal function. The diagnosis may be missed in patients who have DGF or a normal urine output from their native kidneys. An urgent Doppler ultrasound scan will show an absence of perfusion to the graft. The patient must - be returned to the operating theatre immediately to attempt thrombectomy , but most grafts will already be infarcted and transplant nephrectomy will be required. Renal vein thrombosis Renal vein thrombosis occurs in 1–5% of transplants. It may be due to a technical error such as kinking or torsion of the venous anastomosis, but many cases are idiopathic. The peak incidence occurs at postoperative days 3–7. The presentation is distinctive with sudden pain over the renal transplant associated with frank haematuria. In early cases with partial thrombosis, Doppler ultrasound scanning may demonstrate reversal of arterial blood flow in diastole. In an established renal vein thrombosis Doppler scanning will demonstrate in situ marked swelling of the renal allograft, often with a significant surrounding haematoma due to rupture of the renal capsule. Re-exploration to attempt a thrombectomy is rarely successful and the vast majority of cases lead to graft loss. Urological complications Urological complications occur in approximately 5% of renal transplants but they rarely result in graft loss. After dissection of the ureters at organ retrieval, only the ureteric blood supply from the renal artery is preserved. In consequence, the blood supply of the distal transplant ureter can be poor. Urinary leak Urinary leaks can occur in any part of the urinary drainage system but are commonly due to ischaemic necrosis of the distal ureter. The peak incidence is at the time of urinary catheter removal (day 5) but leaks can be delayed for a few weeks. In early leaks clear fluid discharges through the wound or collects in the drains. Biochemical analysis will show that the fluid is urine (creatinine in the millimolar range) rather than - lymph (creatinine in the micromolar range and equal to the serum

level). Leaks presenting later on when the wound is fully healed present as a peritransplant fluid collection. Initial management is bladder catheterisation and cystography to confirm the diagnosis. The anatomical site and extent of the leak can be deter mined by inserting a percutaneous nephrostomy and performing antegrade pyelography . It - is sometimes possible to place a double-J stent across the leakage point using an antegrade approach. If there has been significant ischaemic necrosis of the distal transplant ureter the leak will not resolve and surgery will be required. There are a - number of alternatives for reconstructing the urinary drainage system. If there is a su ffi cient length of transplant ureter after excision of the necrotic segment, the ureter can be reimplanted directly into the bladder over a new double-J stent. If the to-transplant ureteroureterostomy or ureteropyelostomy can be performed. Finally , if the ipsilateral native ureter is absent, because of a previous nephrectomy , a Boari bladder flap can be fashioned to drain the transplant kidney . Ureteric obstruction This can occur at any time after transplantation (days to years). Early obstruction is usually due to a technical error in the bladder anastomosis. Obstruction occurring after 3 months is invariably due to an ischaemic stricture. BK polyomavirus infection is also a cause of ureteric stricture. Patients present with renal dysfunction and investigation by ultrasound scanning reveals hydronephrosis. The initial management of choice is to place a percutaneous nephrostomy . An antegrade pyelogram can then be performed to define the site and extent of the stricture. Short strictures may be treated by interventional radiology with antegrade balloon dilatation and placement of a double-J stent across the stricture. Long, tight strictures require surgery and the options for reconstruction are the same as those for a urine leak. Lymphocele The incidence of significant lymphoceles (>3 /uni00A0 cm) that cause complications and need treatment is around 5%. Small lymphoceles (<3 /uni00A0 cm) are more common but asymptomatic and resolve spontaneously . The usual source of a lymph leak is from lymphatics that are divided during dissection of the iliac vessels. Lymph accumulates as a collection because of the extraperitoneal position of the kidney . Large lymphoceles can compress surrounding structures and, in some cases, cause renal vein thrombosis or ureteric obstruction. All large peritransplant collections should be aspirated under ultrasound to exclude a urinary leak. Lymph is charac terised by having the same biochemical profile as serum. Percu taneous drainage, sometimes on a number of occasions, leads to resolution of most lymphoceles. If this fails surgical drain age is required. This is usually performed laparoscopically and involves creating a fenestra tion in the wall of the lymphocele so that it drains into the peritoneal cavity . Transplant renal artery stenosis The incidence of transplant renal artery stenosis (TRAS) is approximately 5%. The usual presentation is refractory hyper tension associated with allograft dysfunction at 3–6 months post transplant. TRAS activates the renin–angiotensin–aldosterone system and may become evident by a sudden increase in serum creatinine after prescribing an angiotensin-conv erting enzyme inhibitor. Doppler scanning is suggestive of TRAS when the arterial peak systolic velocity is >250 /uni00A0 cm/s and the waveform may have the characteristic tardus parvus (Latin for late and small) pattern. MRA or CT angiograph y are useful for anatomical definition. Percutaneous transluminal angioplasty with or without stenting is the preferred treatment. Achille Boari , nineteenth century Italian urological surgeon from Ferrara, described the technique of a bladder flap in dogs in 1894; it was first performed in a patient in 1936. This can be defined as a rise in serum creatinine of >10% from baseline or an absolute rise of ≥ 20 /uni00A0 µmol/L. Common causes are listed in Summary box 88.2 . Allograft dysfunction should be investigated by urinalysis and culture to exclude urinary tract infection, CNI blood levels for drug-mediated nephro - toxicity and Doppler ultrasound scanning to check for arterial perfusion, venous drainage and hydronephrosis due to urinary obstruction. If there is uncertainty about the cause of graft d

ysfunction, a percutaneous needle-core transplant biopsy should be performed to establish whether allograft rejection is present. Summary box 88.2 Causes of early graft dysfunction /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Long-term allograft dysfunction There are immunological and non-immunological causes for a progressive decline in renal allograft function over a period - of months or years (Summary box 88.3). The commonest - cause of long-term renal allograft damage and subsequent loss is chronic AMR. This is often related to inadequate compli - - ance with immunosuppressive medication. There is no proven treatment for chronic AMR. Non-immunological causes are also significant risk factors for chronic allograft damage. Irrespective of the cause , chronic transplant injury leads to interstitial fibrosis and tubular atr o - phy . Once established these changes are irreversible and will eventually lead to failure of the transplant. - The two most important factors in maintaining good long - term allograft function are meticulous adherence to immuno - suppressive medication and good b lood pressure control (<130/80 /uni00A0 mmHg).

Any rise in serum creatinine of >10% of baseline or ≥ 20 /uni00A0 µmol/L should be considered as acute allograft dysfunction that requires investigation. Possible causes are: Acute rejection (antibody mediated or cell mediated) Calcineurin inhibitor toxicity Dehydration Urinary tract infection or pyelonephritis Any other source of sepsis Renal vein or renal artery thrombosis Ureteric obstruction or urine leak

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