BK Virus Infection after Renal Transplantation

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Abstract
Polyomavirus is one of the important causes of graft dysfunction. The incidence of polyomavirus nephropathies is increasing coinciding with the use of new immunosuppressive medications. It is usually associated with BK virus and frequently affects up to 8% of recipients often resulting in graft loss or permanent dysfunction. More recently, early detection, prompt diagnosis, and therapies including preventive measures have resulted in better outcomes.

The patient described was a 50 year old man on long-term haemodialysis for renal failure as a result of hypertension. He received a single renal transplant from a deceased donor. Initial doppler studies demonstrated good graft function as did a renogram but within a few weeks there was a gradual deterioration of graft function. He was suffering from general malaise and was found to be infected with BK virus. The initial treatment was based on reduction in dose of immunosuppressive therapy, he later underwent reimplantation of the ureter and the function of the transplanted kidney began to improve.

Of interest is that the recipient of the other kidney from the same deceased donor experienced graft rejection but was not found to have BK virus on serology.

Early diagnosis, prevention, and prompt treatment of BK virus infection have improved short- and long-term graft survival. The recommendation is that screening for BK virus should be performed monthly for the first six months then every three months when allograft dysfunction occurs and when a transplant kidney biopsy is performed.

INTRODUCTION
Polyomavirus is one of the important causes of graft dysfunction. The incidence of polyomavirus nephropathies is increasing coinciding with the use of new immunosuppressive medications [1]. It is usually associated with BK virus and frequently affects up to 8% of recipients often resulting in graft loss or permanent dysfunction [2]. The term “BK” is from a patient’s initials, in whom it was first described in 1971 [3]. Patients with this infection usually have asymptomatic viraemia and/or nephritis with or without worsening of renal function. Initially, approximately 30-60% of patients with BK virus nephritis developed graft failure. More recently, early detection, prompt diagnosis, and therapies including preventive measures have resulted in better outcomes. BK infection is an important clinical problem in kidney transplant recipients due to the enhanced immunosuppressive state and BK-specific immune deficiency with alloimmune activation. BK DNA in urine or plasma and renal histology are used for diagnosis [2].

CASE PRESENTATION
The patient was a 50 year old man on long-term haemodialysis for renal failure as a result of hypertension. He received a single renal transplant from a deceased donor. Initial doppler studies demonstrated good graft function (Figure 1) as did a renogram (Figure 2) but within a few weeks there was a gradual deterioration of graft function. He was suffering from general malaise and was found to be infected with BK virus. BK virus DNA was found in the plasma and urine tested on the same days with >10^4 copies/ml plasma and >10^7 copies/ml urine. Evidence of BK viraemia and viruria had cleared by 120 days post-transplant. The aim in treating BK virus was to eliminate the virus while preserving renal functions and preventing acute or chronic rejection. The initial treatment was based on reduction in dose of immunosuppressive therapy with calcineurin inhibitor and steroid avoidance. Serial renal biopsies showed evidence of BK virus infection with viral cytopathic changes in the tubular epithelium, which were resolving by day 79 post-transplant. He later underwent reimplantation of the ureter because of hydronephrosis caused by obstructive uropathy associated with stenosis of the distal urethra (Figure 3).

Initially post-operatively after transplantation he had required haemodialysis. Following surgery for reimplantation of the ureter the function of the transplanted kidney began to improve.
Of interest is that the recipient of the other kidney from the same deceased donor experienced graft rejection but was not found to have BK virus on serology.

DISCUSSION

The presentation of BK virus infection is often asymptomatic with a gradual rise in creatinine and a tubulointerstitial nephritis mimicking rejection. Mean BK viral load increases with intensity of infection as a sustained viraemia and BK specific IgG titres are correlated with the intensity of infection. The principle treatment for BK virus nephropathy is reduction in immunosuppression. Various strategies include reduction or discontinuation of...
calcineurin inhibitor, changing from mycophenolate mofetil to azathioprine or from tacrolimus to ciclosporin [4]. This causes a treatment dilemma as the decrease in immunosuppression needed to treat infection is opposite to the increase needed to treat rejection. In this case reduction in immunosuppression was successful in eliminating viraemia while preserving renal graft function as BK virus was detected early. There does not seem to be a graft survival benefit of adding antivirals such as cidofovir or leflunomide or intravenous immunoglobulin. There is no established treatment other than prompt reduction of immunosuppression to aid viral clearance which can precipitate rejection [5]. However, increased immunosuppression can lead to persistent or worsened viral cytopathic effects caused by reduction in viral clearance. BK nephropathy in solid organ transplants other than kidney recipients is extremely rare and supports BK infection being donor derived.

Usually stenosis of the distal urethra subsides spontaneously. In severe cases percutaneous nephrostomy may be necessary.

More important, therapy with reduction in immunosuppression and/or antiviral therapy with careful monitoring of patients with BK is of paramount importance to prevent progressive renal graft failure. Early diagnosis, prevention, and prompt treatment of BK virus infection have improved short- and long-term graft survival.

The recommendation is that screening for BK virus should be performed monthly for the first six months then every three months when allograft dysfunction occurs and when a transplant kidney biopsy is performed [5].

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REFERENCES