Transplant International

LETTER TO THE EDITOR

Aggressive post-transplant monitoring of more importance to successful outcome following re-transplantation for BK virus nephropathy than absence of pretransplant viremia

doi:10.1111/j.1432-2277.2008.00721.x

We read with interest the article by Mindlova *et al.* [1] (April, 2008) addressing the issue of re-transplantation following renal allograft loss because of BK virus nephropathy (BKVN) in simultaneous pancreas and kidney (SPK) recipients. Two of the four patients in this report were re-transplanted before losing their graft function, with graft nephrectomy performed at the time of re-transplantation. Patient 1 lost the re-transplanted kidney to BKVN, as described in a previous publication [2]. Patient 4 is reported to have low-level viremia with impaired graft function at a follow-up of 55 months, although no BKVN could be documented on biopsies at 6 and 30 months after re-transplantation. The authors conclude from these two cases that the absence of viral replication should be confirmed prior to re-transplantation. We believe a few points are worth mentioning about these data. In their original report of Patient 1, the authors reported that no PCR monitoring for BKV was available [2]. Although not specifically mentioned for Patient 4, the lengthy follow-up suggests re-transplantation also occurred during an era prior to routine application of this technology. Studies in more recent years have emphasized the importance of aggressive post-transplant monitoring with pre-emptive immunosuppression reduction for the prevention of BKVN [3,4] and also the difficulties of such an approach after established disease [5]. Thus, the development of BKVN in Patient 1 and persistent viremia in Patient 4 may reflect the absence of monitoring and pre-emptive reduction early post-transplant rather than any influence of transient viremia at the time of re-transplantation. Furthermore, evidence exists to support donor origin for early BKV infection, further questioning the relevance of transient pre-existing viremia in the recipient [6]. As cited in the discussion section, we previously reported two pre-emptive re-transplants during active BK viremia, including one SPK recipient [7]. Although initially reported only to a follow-up of 21 and 12 months, respectively, Patient 1 of this study maintained normal graft function (serum creatinine – 0.9 mg/dl) and the absence of any BK viremia at the time of her death from intracranial hemorrhage after 3 years post re-transplantation. Patient 2 continues to enjoy normal graft function (serum creatinine – 1.0 mg/dl), with no evidence for active viral replication 3 years after re-transplantation. Although Patient 1 did have low-level BK viremia 4 months after re-transplantation, reduction of immunosuppression resulted in resolution without any detection of graft involvement by biopsy. Studies consistently demonstrate that BK viremia develops in at least 10% of *de novo* kidney transplants [3,8]. Given the probable donor origin of early BK viremia, the development of viremia after re-transplantation is likely no different, and in the absence of randomized trials indicating otherwise, should not dissuade centers from performing pre-emptive re-transplantation for graft failure because of BKVN if other aspects of the case favor such an approach. We agree with the authors that active surveillance is mandatory, however, to detect early recurrence and for timely intervention, and to prevent the development of BKVN.

Karl L. Womer,[1] Pamela R. Patton[2] and Bruce Kaplan[3]
1 Department of Medicine, Johns Hopkins University, Baltimore, MD, USA
2 Department of Surgery, University of Florida, Gainesville, FL, USA
3 Departments of Surgery and Medicine, University of Illinois-Chicago, Chicago, IL, USA

References

1. Mindlova M, Boucek P, Saudek F, *et al.* Kidney retransplantation following graft loss to polyoma virus-associated nephropathy: an effective treatment option in simultaneous pancreas and kidney transplant recipients. *Transpl Int* 2008; **21**: 353.
2. Boucek P, Voska L, Saudek F. Successful retransplantation after renal allograft loss to polyoma virus interstitial nephritis. *Transplantation* 2002; **74**: 1478.
3. Brennan DC, Agha I, Bohl DL, *et al.* Incidence of BK with tacrolimus versus cyclosporine and impact of preemptive

immunosuppression reduction. *Am J Transplant* 2005; **5**: 582.
4. Ginevri F, Azzi A, Hirsch HH, *et al.* Prospective monitoring of polyomavirus BK replication and impact of pre-emptive intervention in pediatric kidney recipients. *Am J Transplant* 2007; **7**: 2727.
5. Womer KL, Guerra G, Dibadj K, *et al.* Immunosuppression reduction for BK virus nephropathy: a case for caution. *Transpl Infect Dis* 2007; **9**: 244.
6. Bohl DL, Storch GA, Ryschkewitsch C, *et al.* Donor origin of BK virus in renal transplantation and role of HLA C7 in susceptibility to sustained BK viremia. *Am J Transplant* 2005; **5**: 2213.
7. Womer KL, Meier-Kriesche HU, Patton PR, *et al.* Preemptive retransplantation for BK virus nephropathy: successful outcome despite active viremia. *Am J Transplant* 2006; **6**: 209.
8. Hirsch HH, Knowles W, Dickenmann M, *et al.* Prospective study of polyomavirus type BK replication and nephropathy in renal-transplant recipients. *N Engl J Med* 2002; **347**: 488.