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doi:10.1093/ndt/gfl672

Advance Access publication 12 December 2006

A case of ureteral lesions in a renal transplant recipient with a co-infection of BK virus and JC virus

Sir,

Most cases of Polyomavirus-associated nephropathy are due to BKV, although Randhawa [1] demonstrated the co-infection of JCV and BKV in renal transplant recipients with interstitial nephritis and Kazory [2] and Wen [3] described two cases of interstitial nephritis associated with JCV in the absence of BKV. We report a 65-year-old female who developed ureteral lesions with a co-infection of BKV and JCV. This patient underwent cadaver renal transplantation in November 2005, after four years of haemodialysis for chronic renal failure secondary to diabetes mellitus. The patient received immunosuppressive therapy with tacrolimus, mycophenolate mofetil and corticosteroids. On day 9 post-transplant, cystography showed a urinary fistula, for which a conservative management was initially chosen. On day 12, PCR assay showed a negative result for serum BKV-DNA and JCV-DNA and urine BKV-DNA and demonstrated a viral load of 10^2 JCV-DNA copies/ml on urine. On day 51 a pyeloureteral anastomosis was performed, as no definitive resolution of the leakage was achieved. During surgery, ureteral stricture and detachment were observed. Histological investigation showed cystic ureteritis and mild chronic inflammation of the wall. Neither nuclear findings consistent with viral infection nor positive reaction for SV40 antibody (Lee Biomolecular Res. Labs, CA) were present. PCR on ureteral specimen showed a viral load of 10^3 and 10^8 BKV- and JCV-DNA copies/μg of extracted DNA, respectively. The patient's clinical conditions and allograft function worsened; a transplantectomy was performed on day 65. Histological investigation of the explanted kidney revealed an acute microabscessual pyelonephritis due to *Candida albicans*, a small infarction and cystic pyelitis. Notwithstanding immunosuppression withdrawal, transplantectomy, and anti-fungal therapy, clinical conditions did not improve and the patient died on day 83. No autopsy was performed. Some authors have suggested that BKV and JCV reactivation are controlled by distinct mechanisms, hypothesising a protective effect of BKV with regard to JCV [4]. In our patient, both BKV- and JCV-DNA were detected on ureteral specimen, with JCV load being higher than BKV load. However, a histological examination of the explanted kidney did not show the presence of viral inclusions in uroepithelial cells. The discrepancy between morphological and molecular findings could be due to sampling, although it cannot be excluded that the affected cells were detached in the ulcerated areas. However, we could not unequivocally prove a cause and effect relationship between ureteral damage, PCR positivity and allograft dysfunction and failure. Based on these observations, we think a close monitoring of polyomaviruses DNA serum and urine levels should be recommended, to diagnose a viral reactivation in its early stages in kidney graft recipients and to perform a quantitative PCR on tissue samples when a biopsy is executed, even if serum and urine have previously tested negative.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfl725