A case of primary JC polyomavirus infection-associated nephropathy

ID 4390956
Author(s) Lautenschlager, Irmeli T.; Jahnukainen, Timo; Kardas, Piotr; Lohi, J.; Auvinen, Eeva; Mannonen, Laura; Dumoulin, A.; Hirsch, Hans H.; Jalanko, H.
Author(s) at UniBasel Hirsch, Hans H. ;
Year 2014
Title A case of primary JC polyomavirus infection-associated nephropathy
Journal American Journal of Transplantation
Volume 14
Number 12
Pages 2887-2892
Keywords Adolescent; DNA, Viral/genetics; Graft Rejection/diagnosis/*etiology; Humans; Immunosuppressive Agents/therapeutic use; JC Virus/pathogenicity; Kidney Failure, Chronic/complications/*surgery/virology; *Kidney Transplantation; Male; Nephritis, Interstitial/diagnosis/*virology; Polyomavirus Infections/complications/*virology; Postoperative Complications; Prognosis; Renal Dialysis; Tumor Virus Infections/complications/*virology; Viral Load; biopsy; clinical research/practice; infection and infectious agents; infectious disease; kidney transplantation/nephrology; rejection: acute; rejection: antibody-mediated (ABMR); viral: BK/JC/polyoma
A 15-year-old boy with a posterior urethral valve received a deceased donor kidney transplant (KT) in March 2011. Basiliximab induction followed by tacrolimus-based triple medication was used as immunosuppression. Eleven months after KT, the graft function deteriorated and the biopsy demonstrated interstitial nephritis suggestive of acute rejection. BK polyomavirus (BKPyV) surveillance in urine and plasma was negative. The patient received methylprednisolone pulses and anti-thymocyte globulin. Immunohistochemistry was positive for simian virus 40 (SV40) large T-antigen (LTag) in the biopsies, and quantitative polymerase chain reaction for JC polyomavirus (JCPyV) indicated high viral loads in urine and borderline levels in plasma. Immunosuppression was reduced and follow-up biopsies showed tubular atrophy and interstitial fibrosis. Two years after KT, antibody-mediated rejection resulted in graft loss and return to hemodialysis. Retrospective serologic work-up indicated a primary JCPyV infection with seroconversion first for IgM, followed by IgG, but no indication of BKPyV infection. In the SV40 LTag positive biopsies, JCPyV deoxyribonucleic acid (DNA) with archetype noncoding control region was detected, while BKPyV DNA was undetectable. To the best of our knowledge, this is the first reported case of primary JCPyV infection as the cause of PyV-associated nephropathy in KT.
Publisher Wiley
ISSN/ISBN 1600-6135 ; 1600-6143
edoc-URL https://edoc.unibas.ch/61914/
Full Text on edoc No;
Digital Object Identifier DOI 10.1111/ajt.12945
PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/25359127
ISI-Number WOS:000345294300029
Document type (ISI) Article