Journal of NeuroVirology, 10(suppl. 2): 35, 2004

ISSN: 1355-0284 print / 1538-2443 online
DOI: 10.1080/13550280490469789

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Polyomavirus replication and renal disease

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Polyomavirus-associated nephropathy (PVAN) is an emerging disease in renal transplant patients with a prevalence of 1–10% and graft loss reaching >80% in some centers. BK virus is most frequently involved, but JC virus may account for some cases. The recent emergence of PVAN is not well understood, but coincides with the increasing use of potent immunosuppressive drugs like tacrolimus and mycophenolate mofetil. Their different mechanism of action suggests that the overall immunosuppressive effect may be the common denominator. However, drug-specific machanisms have not been investigated. The preferential manifestation in renal transplants as compared to other allografts, or to autologous kidneys of other transplant patients suggests that organ-specific determinants and immunologic factors synergize: Tubular cell injury e.g. from rejection, and compensatory proliferation to restore tubular integrity may provide signals/enzymes favoring polyomavirus replication while immunologic control is impaired due to immunosuppression, antirejection therapy and HLA-mismatches. Patient determinants (older age, male gender, seronegative recipient), and viral factors (new genotype, serotype) may have a contributory role. Since PVAN determinants differ in individual patients and are difficult to quantitatively assess, screening for polyomavirus replication has become an important surrogate marker in clinical practice. Quantification of polyomavirus DNA in urine and plasma allows for an early diagnosis of PVAN at a stage of limited damage. Moreover, the response to intervention can be monitored using viral load. Although the definitive diagnosis of PVAN is made histologically, the focality of earlier stages gives rise to biopsy sampling error limiting diagnostic sensitivity. In some centers, intervention is considered in cases of presumptive PVAN e.g. with plasma viremia >10'000 copies/mL.