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VIRAL FOES OF KIDNEY TRANSPLANTS

ABSTRACT

Viruses are the most important and common causes of opportunistic infection after renal transplantation. Viral infection produces the damage either by "direct effect" of invasive disease or by "indirect effect" of modulating the immune response resulting in predisposition to rejection, other opportunistic infections and oncogenesis. Mechanisms of latency and re-activation are also important. Currently, viral infections are diagnosed by estimation of viral copy numbers in body fluids or by using specific immunohistochemistry and/or in-situ hybridization techniques in biopsies.

Important viral infections of the renal allograft include Polyoma virus BK/JC, Cytomegalovirus (CMV), Hepatitis B and C, Epstein-Barr virus (EBV), HHV 6,7,8 and Adenovirus. Our studies indicate that the incidence of Polyoma virus nephropathy (PVN) in India is 9.4%, which is higher than reported from the West. This is related to a higher prevalence of detectable virus in the plasma of recipients (28%) and donors (44%), the highest incidence of which occurs at 1-3 months after transplantation. Dual positivity in both recipients and donors confers a high risk of development of PVN. Infection by CMV has a wide pathologic spectrum, causing tubulo-interstitial nephritis, cytopathy in the glomeruli and thrombotic microangiopathy (TMA). Both Hepatitis viruses cause glomerulonephritis; in addition, Hepatitis C is an important cause of TMA. The pathology of HHV infection is similar to PVN and can be diagnosed only by molecular techniques. Nephritis by Adenoviruses is generally secondary and devastating. Post Transplant Lympho-proliferative Disease (PTLD), occurs early when caused by EBV.

Viral nephropathies of transplanted kidney are an important cause of renal allograft dysfunction; careful diagnosis is important to distinguish from rejection and appropriate treatment