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Introduction

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Introduction

Nasimul Ahsan

Although the incidence of infectious complications following organ transplantation has declined over the past two decades, nephropathy associated with BK virus has emerged in recent years as an important cause of allograft failure in renal transplant recipients. First isolated in 1971 from a recipient of a kidney transplant, BK virus is a DNA virus and is a member of the polyomavirus family. BK virus-associated illnesses occur primarily in the urinary tract, causing ureteral ulceration and stenosis, hemorrhagic cystitis, and tubulo-interstitial nephritis.

The risk factors for BK virus nephropathy in renal transplant recipients are unknown and may or may not be related to specific immunosuppressive agents. In this regard, a relative absence of BK virus nephropathy in recipients of nonrenal transplants receiving similar immunosuppressions suggests a “second hit” hypothesis, a necessary cofactor for the pathogenesis of invasive infection. These second hits may include allograft injuries due to acute rejections, acute tubular necrosis, ischemia-reperfusion, and ureteral trauma. Although virological assays may allow early screening for the infection, the diagnosis of BK virus nephropathy is confirmed by histopathological evaluation. At present, there is no effective treatment for BK virus nephropathy, but investigators have applied several conservative approaches and novel antiviral therapies to preserve renal allograft function.

This special supplement explores, in great detail, the epidemiology, discovery, biology, autoimmunity, neoplasm, and molecular genetics of polyoma virus, particularly BK virus. The authors in this volume have also shared their clinical experiences with BK virus-associated infections in renal and nonrenal transplant situations. Some have examined the relationships between BK virus infection and various immunosuppressive agents currently in use,

whereas others have provided guidelines for diagnosis and treatment.

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