BK Virus in Hematopoietic Stem Cell Transplantation Recipients

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BK virus has become a serious issue in hematopoietic stem cell transplantation recipients, commonly manifesting as hemorrhagic cystitis, which can last from a matter of days to months and, if severe enough, may result in death. Patients with BK virus-associated hemorrhagic cystitis often experience poor quality of life, severe pain and discomfort, and prolonged hospitalizations. Despite numerous advances in stem cell transplantation methods, BK virus-associated hemorrhagic cystitis is difficult to control and treatment options are few. This article provides an overview of BK virus along with risk factors, current treatment modalities, and nursing considerations.

At a Glance

- BK virus can cause life-threatening hemorrhagic cystitis in stem cell transplantation recipients.
- Risk factors for the development of BK virus-associated hemorrhagic cystitis may include conditioning regimen, donor type, graft-versus-host disease, and the patient’s BK viral load.
- Despite many advances in stem cell transplantation methods, BK virus remains difficult to treat.

Polyomavirus hominis-type 1, commonly referred to as BK virus, infects up to 90% of the world’s population (Hirsch & Steiger 2003) but does not usually cause symptoms in immunocompetent individuals. However, BK virus can be a very serious issue in immunocompromised patients.

BK virus, which derives its name from the initials of the first infected patient, is troubling to renal and hematopoietic stem cell transplantation recipients. Allograft dysfunction and failure may occur secondary to Polyoma-associated nephropathy in kidney transplantation recipients (Hirsch, 2002). BK virus commonly manifests in hematopoietic stem cell transplantation recipients as hemorrhagic cystitis, characterized by painful hematuria secondary to inflammation and breakdown of epithelial cells of the bladder mucosa (Leung, Yuen, & Kwong, 2005). If severe, hemorrhagic cystitis may be life threatening. BK virus also has been linked to ureteric stenosis, vasculopathy pneumonitis, encephalitis, retinitis, and multi-organ failure (Galan, Rauch, & Otis, 2005; Hirsch & Steiger, 2003). BK virus has proven to be a challenge for patients and healthcare professionals alike. This article provides an overview of BK virus infection in hematopoietic stem cell transplantation recipients, along with risk factors, treatment modalities, and nursing considerations.

BK Virus: An Overview

Virology

BK is a double-stranded, nonenveloped virus that contains icosahedral capsids. The icosahedral capsids contain the DNA genome (Hirsch & Steiger, 2003). The BK virus genome is very similar to that of Polyomavirus hominis-type 2 (known as JC Virus), which also is found in immunocompromised patients. The BK virus genome is comprised of regulatory, early, and late regions. The regulatory region is referred to as the noncoding region and is the site of replication, containing promoter elements of early and late genes (Hirsch & Steiger). Early genes are responsible for encoding the small tumor antigen and the large tumor antigen. Large tumor antigen plays a pivotal role in BK virus transcription and replication (Hirsch & Steiger). BK virus replication is very dependent on the amenities of host cell enzymes, and the large tumor antigen identifies host cell proteins necessary for replication. Late genes are responsible for encoding viral capsid proteins and the agnoprotein, which serves in viron assembly (Hirsch & Steiger). Late gene expression only occurs after BK virus replication has occurred (see Figure 1).

BK Virus Disease

Infection, replication, and disease are three terms key to understanding BK virus (see Table 1). BK virus infection is

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