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Short Communication

Screening for BK Virus Infection in Kidney Transplant Recipients at Mohamed V Military Teaching Hospital

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Abstract

After kidney transplantation, BK polyomavirus reactivation can manifest as nephropathy in 1% to 10% of patients. PCR testing of urine and blood is commonly used to screen for BK polyomavirus nephropathy. The study aims to detect BK virus infection in kidney transplant patients to prevent tubulointerstitial nephropathy and graft loss. This retrospective study includes 26 patients who underwent kidney transplants between January 2019 and December 2023. We diagnosed BK virus infection by performing real-time PCR on blood and urine samples. BKV DNA was detected in 3 patients. Reducing immunosuppressive therapy led to negative PCR results and favorable clinical and biological outcomes in these 3 patients.

More Information

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Keywords: BK Virus; Kidney transplant; PCR

Introduction

BK polyomavirus (BKV), a member of the Polyomavirus family, was first identified in 1971 from the urine of a kidney transplant patient whose initials were B and K. BKV infection can remain latent for years in renal tubular and urothelial cells, with potential reactivation occurring under immunosuppression, such as during post-renal transplantation immunosuppressive therapy. This reactivation can lead to nephropathy, which may cause graft rejection [1].

In this study, we highlight the crucial role of the virology laboratory in detecting and monitoring BKV infection in kidney transplant patients.

Methods

This is a retrospective study carried out over five years (January 2019 to December 2023). The patients' virological data were obtained from the laboratory's information system.

BKV testing was conducted on blood and urine samples. Viral DNA extraction was carried out using the Qiagen EZ1 automated system. Real-time quantitative amplification was performed on a ROTOR GENE using the RealStar® BKV PCR Kit 1.0.

The RealStar® BKV PCR Kit 1.0 is a reagent system, based on real-time PCR technology, for the detection and quantification of BK virus (BKV) specific DNA. The Kit consists of two Master reagents (Master A and Master B), Internal Control (IC), four qualification standards (QS1 - QS4), and PCR-grade water.

The Quantification Standards contain standardized concentrations of BKV-specific DNA. These Quantification Standards were calibrated against the 1st World Health Organization International Standard for BK Virus for Nucleic Acid Amplification Techniques (NAT) (NIBSC code 14/212). The Quantification Standards can be used individually as positive controls, or together to generate a standard curve, which can be used to determine the concentration of BKV-specific DNA in a sample.

Results

The study enrolled 26 patients, with a male-to-female sex ratio of 1.6. The average age was 48, ranging from 17 to 66. Parents contributed as donors in 52% of cases.

BKV DNA was identified in 3 kidney transplant recipients (11%). BKV viremia occurred within the first post-transplant year in two patients. One patient was also found to have cytomegalovirus (CMV) co-activation. Renal function in the 1st patient was correct after renal transplantation during episodes where viremia was positive and also outside these periods. In the 2nd patient, renal function was slightly impaired at the time of viremia, then returned normal after adjustment of treatment and negativation of viremia in the 3rd patient, renal function was not impaired.

Prompt adjustment of immunosuppressive therapy upon detection of the initial positive viral load resulted in favorable clinical and biological outcomes for all three patients.

Discussion

BK virus infection can commence as early as childhood through the respiratory route. Following an often asymptomatic primary infection accompanied by transient viremia, the virus establishes a latent presence in renal tubular and urothelial cells. Reactivation of the virus is facilitated by situations of immunosuppression, such as the administration of immunosuppressive therapy post-renal transplantation, increasing the risk of nephropathy and graft rejection [2].

The high incidence of BKV viremia during the first year post-transplant has led to the development of standard screening protocols by transplantation centers. Given the low specificity of urinary viral loads and the higher positive predictive value of plasma levels, screening for BKV infection via PCR in blood remains the preferred method [3].

After transplantation, the activation of BKV usually is not associated with the co-activation of CMV, and BK nephropathy is not associated with other graft infections [4]. Rarely, BKV nephritis has been reported in patients with co-existing CMV infection of other sites [5]. A synergistic effect of the two viruses in the rejection process has been documented [6].

Currently, there is no effective antiviral prophylaxis or treatment available; only reduction of immunosuppression and regular monitoring of viremia via PCR are recommended. Multiple protocols have been developed to reduce immunosuppression, although trials comparing their efficacy have not yet been conducted [6].

Conclusion

To date, no antiviral treatment has proven effective in eradicating the BK virus. Nevertheless, ongoing research into new therapeutic approaches holds promise for delivering the efficacy that has remained elusive for the transplant community.

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