Pediatric Kidney Transplant and BK Viral Infection: A Single-Center Retrospective Analysis of Interventions and Outcomes

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Background

•BK virus-associated nephropathy (BKVAN) is an important cause of graft loss in kidney transplant recipients.

•The successful advent of increasingly efficacious immunosuppression has been accompanied by high rates of BK viremia (BKV) in up to 30% of kidney transplant recipients.

•A reduction in the intensity of immunosuppression is the overarching principle for the treatment of BK viremia and BKVAN.

•There is no therapeutic agent available to treat this virus-associated disease, with many agents lacking conclusive efficacy in the reduction in viral loads. Multiple protocols have been developed for a reduction in immunosuppression, albeit trials are yet to be conducted to compare their effectiveness.

•In a single-center cohort of pediatric renal transplant patients, we investigated BK virus-associated nephropathy (BKVN) incidence, management strategies, and clinical outcomes.

Methods

We analyzed kidney transplant patients from Jan 2009 to Dec 2022. Ureteral stents placed during transplantation removed in 4-6 weeks. Recipients had monthly urine and plasma PCR screening for BK virus for the first 12 months and during rejection treatment.

Results

•Among 101 patients, 17 (16.8%) had BK viruria, 15 (14.9%) had presumptive BKVN (DNAemia >10,000 copies/mL), and 15 (14.9%) had probable BKVN (DNAemia 1,000-10,000 copies/mL), with 4 showing blip DNAemia.

• Median time to BK viruria was 48 days post-transplant and 141 days for BK viremia.

•Of the 15 patients with presumptive BKVN, 11 (73.3%) were male; 10 (66.6%) were white, 4 (26.7%) African American, and 1 (6.7%) other. Mean age at transplantation was 11.6 years (range 2.4-18.4); 11 (73.3%) received kidneys from deceased donors.

•10 patients had IVIg monthly at 500 mg/kg for 6 months. Before IVIg, mean urinary BKV DNA was 232 million ± 574 million copies/mL, and plasma load was 171,945 ± 428,810 copies/ml. At diagnosis, mean serum creatinine peaked at 1.3 mg/dL (baseline 0.85 mg/dL).

• Post-treatment, mean serum creatinine improved to 0.95 mg/dL, and plasma BKV DNA load decreased to 312 ± 788 copies/mL. 7/10 had non-quantifiable plasma loads.

• One IVIg-allergic patient received leflunomide alone, clearing BK viremia from 160,000 to non-quantifiable levels, with creatinine improving from 1.6 to 1 mg/dL.

•4 patients received 10 monthly doses of IVIg but did not clear DNAemia and then received leflunomide. Two cleared BK virus (mean 27,900 copies/mL to undetected) with creatinine improving (2.02 to 1.14 mg/dL).

•Two non-responsive patients received low-dose IV cidofovir (0.5-0.75 mg/kg/dose) for six doses. Despite BK viral level improvements (28,000 to 15,000 copies/mL), viremia clearance was unsuccessful, with minimal creatinine improvement (1.45 to 1.2 mg/dL).

Conclusions

•At our center, IVIg seems to be an effective treatment for persistent BKVN after failed reduction in immunosuppression. IVIG was effective in 70% of cases, while leflunomide alone or following IVIG was effective in 60%. The addition of cidofovir for those resistant to IVIG and leflunomide was not successful.