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Recent updates of BKV treatment

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BK virus nephropathy (BKVN) is a severe complication that can occur after kidney transplantation, caused by the reactivation of the BK virus in the transplanted kidney. It poses a significant threat to graft function and can lead to transplant failure if left untreated. The primary goal of treatment is to control viral replication and prevent further damage to the transplanted kidney while preserving its function. The first step in managing BKV infection is close routine monitoring for plasma BK viral load. Kidney allograft biopsy is the gold standard for diagnosing BKVN, assessing its severity, and evaluating concomitant processes. However, because of its invasiveness and the potential for sampling errors, a presumptive diagnosis is often made based on the presence of significant viremia (plasma BKV virus levels >10,000 copies/mL). The cornerstone of treatment involves reducing immunosuppression. The immunosuppressive regimen is tailored based on the patient's risk factors, and doses of medications like calcineurin inhibitors, mycophenolate mofetil, or mTOR inhibitors are often reduced or temporarily discontinued. Several agents have shown anti-BKV activity in vitro, including intravenous immunoglobulin, leflunomide, cidofovir, and quinolone antibiotics. However, the efficacy of these agents has not been conclusively established, and it remains unclear whether their use is superior to reducing immunosuppression alone. Due to the critical role of cellular and humoral immune mechanisms in controlling BKV infection and the lack of proven therapies, several immune-based treatments, such as virus-specific T cells and BK virus-specific monoclonal antibodies against the BK virus, are actively being evaluated in human clinical trials. In summary, the established treatment for BKVN after kidney transplantation currently involves reducing immunosuppression and closely monitoring kidney function and viral load. Novel agents may become available in the near future.