**TITLE:** **PERSISTENT BK POLYOMAVIRUS DNAEMIA IN A KIDNEY TRANSPLANT PATIENT DESPITE AGGRESSIVE TREATMENT: A CASE REPORT**

Nishanthi WAAGN1, Ranaweera ME2, Illeperuma PB3, Asmir WM1

1Teaching Hospital Anuradhapura, 2National STD/AIDS Control Programme, 3District General Hospital Matara

### ****Introduction****

Kidney transplantation, a transformative intervention for end-stage kidney disease, can be complicated by BK polyomavirus (BKPyV), leading to allograft failure. This case of presumptive BKPyV nephropathy underscores the complex challenges in its management.

### ****Case Report****

A 51-year-old male underwent a live donor kidney transplant (HLA mismatch 2:1:1) and was managed with tacrolimus, mycophenolate mofetil (MMF), and prednisolone after antithymocyte globulin (ATG) induction. Early post-transplant, he developed acute kidney injury treated with pulse methylprednisolone. The allograft biopsy showed early interstitial fibrosis but no evidence of rejection. He also experienced tacrolimus toxicity. His serum creatinine initially stabilized at 130 µmol/L, then gradually rose to 160 µmol/L. BKPyV-DNAemia (1.55x104 IU/mL) was detected at 7 months after transplant and monthly monitoring was initiated. Immunosuppression was tapered: MMF by 50%, then 25%, and eventually stopped. He received two courses of 5 doses of IVIG, ciprofloxacin, and leflunomide. Tacrolimus was reduced maintaining a target trough of 5 ng/mL, everolimus 0.5mg was added, but the DNAemia persisted in the 4-log range, with a 0.5-log drop on one occasion.

### ****Discussion****

Our patient had multiple risk factors for BKPyV-nephropathy: male gender, ATG use, high-dose corticosteroids, tacrolimus, and a ureteric stent. Donor or recipient BKPyV serostatus or genotypes were not assessed. While routine screening for BKPyV-DNAemia is recommended monthly for the first 9 months and then every 3 months until 2 years, this was not feasible in our resource-limited setting. This case highlights the importance of initiating BKPyV monitoring early and the urgent need for more effective therapeutic options.

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