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Posttransplant lymphoproliferative disorders (PTLD) in kidney transplant recipients: Case report

Julia Paravizo Lello Santos

José do Rosário Vellano University, Brazil

Abstract

Case 1: A 45-year-old male underwent a livinghaploidentical donor renal transplantation and received immunosuppression therapy (cyclosporine, mycophenolate mofetil, and prednisolone). Nine years after transplantation his graft function was stable (creatinine 1.1 mg/dL), when presented with diffuse abdominal pain with perforation intestine signals. The pathological finding were compatible with post-transplant lymproliferative monomorphic disorder, high-grade lymphocytes of B-cell origin by their CD20 positivity and co-expression of CD10 and negative for BCL 2.In situ-hybridization for EBV-encoded RNA (EBER) was strongly positive (Figure 1). Patient had mTOR conversion, and sirolimus was included in the immunosuppression schedule.

Case 2: A 35-year-old female was submitted to a renal transplant with HLA-distinct living related donor, receiving prednisone, cyclosporine and azathioprine. After 13 years with stable clinical/laboratorial outcomes, she was attended with small bowel obstruction symptoms. The cells in this tumour were confirmed immunohistochemically as lymphocytes of B-cell (Figures 2) origin by their CD20 (Figure 3) and CD79a, with a proliferation rate of 80% (Ki67). Sirolimus was initiated with CNI suspension.

Discussion: PTLD has become an important comorbidity in kidney transplant recipients. Some studies focus on the differences between early-onset and late-onset PTLD, that is, the segregation between EBV-driven diseases versus later coincidental lymphomas expected in the general immune competent population. The disease can occur in a wide range of locations. Minimization of immune suppression is the mainstay of treatment after PTLD diagnosis and a multidisciplinary oncological approach is essential.

Biography:

Julia Paravizo Lello Santos is an 18-year-old medical student at José de Rosário Vellano University (UNIFENAS) in Brazil.

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