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Recent insights in the pathogenesis of post-transplantation lymphoproliferative disorders

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Abstract

Post-transplant lymphoproliferative disorder (PTLD) is an aggressive complication of solid organ and hematopoietic stem cell transplantation that arises in up to 20% of transplant recipients. Infection or reactivation of the Epstein-Barr virus (EBV), a ubiquitous human herpesvirus, in combination with chronic immunosuppression are considered as the main predisposing factors, however insight in PTLD biology is fragmentary. The study of PTLD is complicated by its morphological heterogeneity and the lack of prospective trials, which also impede treatment optimization. Furthermore, the broad spectrum of underlying disorders and the graft type represent important confounding factors. PTLD encompasses different malignant subtypes that resemble histologically similar lymphomas in the general population. Post-transplant diffuse large B-cell lymphoma (PT-DLBCL), Burkitt lymphoma (PT-BL) and plasmablastic lymphoma (PT-PBL) occur most frequently. However, in many studies various EBV+ and EBV- PTLD subtypes are pooled, complicating the interpretation of the results. In this review, studies of the gene expression pattern, the microenvironment and the genetic profile of PT-DLBCL, PT-BL and PT-PBL are summarized to better understand the mechanisms underlying post-transplantation lymphomagenesis. Based on

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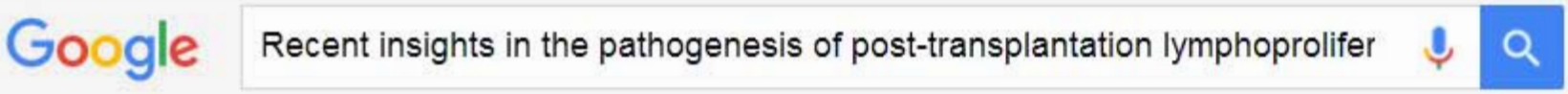

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