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BURKITT'S LYMPHOMA

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Burkitt's lymphoma (BL) is a highly aggressive B-cell non-Hodgkin lymphoma characterized by rapid tumor growth and a propensity for extranodal sites, such as the jaw, abdomen, or central nervous system. First described by Denis Burkitt in 1958 in African children, BL is now recognized globally with three epidemiological variants: endemic, sporadic, and immunodeficiency-associated. Its association with Epstein-Barr virus (EBV) and genetic translocations makes it a critical subject for studying lymphomagenesis and targeted therapies.

The etiology of Burkitt's lymphoma is multifactorial, with EBV infection playing a central role in endemic cases, present in nearly 95% of patients. Sporadic and immunodeficiency-associated forms show lower EBV association (20-30%). A hallmark genetic alteration is the chromosomal translocation t(8;14), which juxtaposes the MYC oncogene with immunoglobulin heavy-chain loci, leading to MYC overexpression. Other contributing factors include malaria co-infection in endemic regions, which may exacerbate B-cell proliferation, and immunosuppression, particularly in HIV-positive individuals. Environmental and genetic predispositions remain under investigation, but their roles are less defined.

Burkitt's lymphoma pathophysiology hinges on MYC-driven lymphomagenesis. The t(8;14) translocation dysregulates MYC, promoting uncontrolled B-cell proliferation and inhibiting apoptosis. EBV-encoded proteins, such as LMP1, further enhance B-cell survival by mimicking CD40 signaling. The tumor microenvironment, enriched with inflammatory cytokines, supports rapid cell turnover, contributing to Burkitt's lymphoma aggressive nature. Histologically, Burkitt's lymphoma exhibits a "starry-sky" pattern due to interspersed macrophages engulfing apoptotic cells. Metabolic reprogramming, driven by MYC, fuels high glycolytic rates, enabling tumor sustenance. These mechanisms collectively underpin Burkitt's lymphoma rapid progression and chemosensitivity.

Thus, Burkitt's lymphoma exemplifies how genetic and environmental factors converge to drive malignancy. Its MYC translocation and EBV association highlight unique pathophysiological mechanisms, offering insights into targeted therapies like rituximab and MYC inhibitors.