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OPINION ARTICLE

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Pathophysiology of Epstein-Barr Virus Infection

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Description

Epstein-Barr Virus (EBV) primarily affects children and adolescents as its host. The majority of them are self-limiting, asymptomatic infections. However, EBV infection poses a serious threat to life for those patients who have immune system abnormalities. One of the serious consequences is called EBV-HLH, or hemophagocytic lymphohistocytosis, or EBV. This paper discusses the diagnosis and differential diagnosis of EBV-HLH and other EBV infectious illnesses. EBV-HLH problems and their molecular biology mechanism are briefly addressed. Additionally, it offers medical professionals a useful technique for the genetic identification of these illnesses and the differential diagnosis with other human immunodeficiency diseases.

There are two types of Hemo Phagocytic Syndrome (HPS), often referred to as Hemophagocytic Lympho Histocytosis (HLH). Primary HLH, also known as family HLH (FLH), typically manifests in childhood and is frequently accompanied with genetic flaws in T lymphocytes and Natural Killer (NK) cells. Infections, malignancies, autoimmune disorders, immunodeficiency states, and other conditions are frequent causes of secondary HLH. A non-neoplastic condition called Epstein-Barr Virus-associated Hemophagocytic Lympho-Histocytosis (EBV-HLH) is brought on by immune system problems brought on by Epstein-Barr Virus (EBV) infection. It is the most common form of secondary HLH and is mostly present in Asian nations like China and Japan. Its pathophysiological mechanism is that EBV-infected CTL and NK cells are functionally defective and proliferate abnormally, causing hypercytokinemia and the activation of macrophages, which in turn causes significant tissue damage. Fever, blood cytopenia, hepatosplenomegaly, coagulopathy, abnormalities of the central nervous system, and other uncommon consequences are among the symptoms of EBV-HLH. Although some EBV-HLH patients have been shown to have mutations in primary HLH-related genes like perforin and SH2D1A, EBV-HLH is not always secondary. These patients truly fall within the category of main HLH, and EBV infection is a major contributing factor to these individuals' trigger status. Even though some EBV-HLH cases lack known HLH-associated gene alterations, the potential of primary HLH cannot be completely ruled out because there may be undiscovered genetic mutations.

HLH is a rare but fatal condition brought on by unchecked immunological activation, which results in increased macrophage activity and cytokine production. EBV infection causes deficiencies in SAP protein function, which raise Th1 cytokine secretion levels and cause high quantities of IFN-, IL-10, and other cytokines to be released. This results in cytokine storms, which cause HLH. The exact pathophysiology of HLH is not well understood, despite the fact that EBV is one of the most common HLH triggers. Therefore, a careful analysis of HLH cases linked to EBV may shed light on how HLH develops. One the one hand, gene mutations have been related to main HLH in humans. Because of this, contaminated B lymphocytes cannot be removed by CD8-T/NK cells. The antigen continues to exist, which causes human lymphocytes and tissue cells to multiply and secrete a significant number of cytokines, creating a cytokine storm that causes sickness.

Genetic abnormalities are present in a significant portion of EBV-HLH patients; however they do not always have an impact on the clinical presentation or course of treatment. Though most EBV-HLH cases are sporadic, a small number of them might be the X-linked lymph proliferative syndrome's initial symptom.

EBV-HLH is a serious outcome following EBV infection, and humans are susceptible to the virus. The two types of EBV-HLH are primary EBV-HLH and EBV-HLH subsequent to EBV infection. As a result of abnormal hyperplasia and defunctionalisation of CTL and NK cells caused by EBV infection, excessive cytokine release and macrophage activation occur, causing extensive tissue damage. Recent research has shown that r/r EBV-HLH and r/r EBV-HLH reacted effectively to the treatment with nivolumab. There are few clinical data, but raxolitinib has also been shown to be highly effective in treating steroid-refractory acute GVDH. In conclusion, extensive research on cytogenetic, treatment, and relapse prevention should be done for patients with EBV-HLH.

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