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Commentary

A Brief Note on Hypereosinophilic Syndromes

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DESCRIPTION

Hypereosinophil Syndrome (HES) is a group of blood disorders that occur when there are a large number of eosinophil leukocytes that play an important role in the immune system. Over time, excess eosinophils invade various tissues and ultimately damage organs. If, despite a thorough diagnostic evaluation, the underlying cause of persistent hypereosinophil exacerbation is not identified, clinicians may refer to Hypereosinophil Syndrome (HES), which includes a heterogeneous group of rare diseases. You should consider diagnosing. The original definition of HES proposed in 1975 is known to cause blood eosinophilia of 1.5 × 10^9/L or more lasting 6 months or more, and hypereosinophilia. There is no evidence of underlying disease, including the presence of eosinophil-mediated organs.

These criteria no longer reflect clinical practice that integrates the latest diagnostic and therapeutic facilities with recent advances in pathogenic mechanisms. In short, persistent eosinophil exacerbations are no longer defined as a 6-month period, but can be shorter if other causes of eosinophil exacerbations are excluded. Several eosinophil-related diseases that were previously thought to be separate from HES have been integrated into the more recent HES classification scheme. These include Churg-Strauss syndrome and organ-specific eosinophilic disorders such as chronic eosinophilic pneumonia and eosinophilic gastrointestinal disorders. The target organ disorder does not need to be present first to make a diagnosis of HES. Clinically, HES is a potentially fatal multisystem disease characterized primarily by eosinophil infiltration of various target organs such as the skin, heart, lungs, gastrointestinal tract, central and peripheral nervous systems includes. Microvascular thrombotic phenomena associated with the involvement of other organs, liver and/or splenomegaly and endothelial damage occur at varying frequencies.

The type and severity of organ damage varies widely from patient to patient and is often unpredictable. With certain levels of eosinophilia in the blood, some patients have relatively mild clinical features such as isolated skin symptoms that may not require treatment. On the other side of the spectrum, on the other hand, some patients show rapidly progressing heart failure or thrombotic complications that require urgent medical attention. In some forms of HES, disease progression in a small number of patients is due to the development of malignant tumors involving either myelogenous (acute myelogenous or eosinophil leukemia) or lymphoid cells (peripheral T-cell lymphoma). Recent studies have led to the identification of two major pathogenically identifiable variants of HES.

Myeloproliferative variant HES (M-HES) is characterized by features that are typically encountered in other myeloproliferative diseases, including increased serum vitamin B12, hepatomegaly, splenomegaly, anemia, thrombocytopenia, circulating myeloid precursors, and increased BM cellularity with a left-shift in maturation. The majority of patients with M-HES have a cryptic interstitial deletion on chromosome 4q12 that results in expression of a FIP1L1-PDGFRA (FIP1-like 1/ Platelet derived growth factor receptor alpha) fusion protein with autonomous tyrosine kinase activity (F/P-associated HES or chronic eosinophilic leukemia). Although this appears to be a stem-cell mutation, clonal eosinophil expansion dominates over other lineages. Clinical characteristics of this variant include strong male predominance, increased serum vitamin B12 and tryptase levels, mucosal ulcers, splenomegaly, endomyocardial fibrosis as well as other organ-based fibrotic complications, and possible progression towards acute eosinophilic leukemia or blast crisis. Much less commonly translocations on chromosomes 5q33 and 8p11 lead, respectively, to Platelet-Derived Growth Factor Receptor Beta (PDGFRB) and Fibroblast Growth Factor Receptor 1 (FGFR1) -rearranged clonal forms of M-HES.

The Lymphocytic HES (L-HES) variant is characterized by polyclonal eosinophil expansion in response to marked over-production of IL-5 by deregulated T-cells *in vivo* . These T-cells can be detected on the basis of abnormal surface phenotypes, including CD3-CD4+ and CD3+CD4-CD8-, and are sometimes monoclonal. Published case reports indicate that these T cell clones may be prone to malignant transformation (T-cell lymphoma). Clinically, patients often present with predominant cutaneous manifestations, although other organs may be targeted as well, increased serum IgE levels and hypergammaglobulinemia.

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