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Brief note on hypereosinophilic syndromes

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DESCRIPTION

Eosinophilia syndrome is associated with organ system involvement or dysfunction symptoms that are directly associated with eosinophilia, without the parasitism, allergies, or other secondary causes of eosinophilia. It is a condition characterized by peripheral blood eosinophilia. Symptoms depend on which organ is disturbed. Diagnosis includes excluding eosinophilia and other causes of bone marrow and cytogenetic testing. Treatment may include prednisone, and in some cases imatinib, depending on the particular subtype of hypereosinophilic syndrome. Eosinophil syndrome (HES) is a group of disorders characterized by sustained overproduction of eosinophils, in which eosinophil infiltration and mediator release damage multiple organs. Although these disorders have long been considered idiopathic (for example, “idiopathic hypereosinophil syndrome” IHES), the etiology of several forms of HES has been described. Hypereosinophilia is traditionally defined as peripheral blood eosinophilia> 1500 / mcL (>1.5 × 109/L) that lasts for more than 6 months. (See also Eosinophil production and function.)

Hypereosinophil syndrome was previously thought to be idiopathic, but molecular features indicate that many cases have specific clonal disorders. Limitations of the conventional definition are known causes of hypereosinophilia syndrome, but do not meet the conventional definition of hypereosinophilia syndrome in the degree or duration of eosinophilia, the same abnormalities. Do not include patients with some of the chromosomal abnormalities. Another limitation is that patients with organ damage characteristic of eosinophilia and hypereosinophilia require treatment before the 6 months required to confirm conventional diagnostic criteria. There are times when you do. Eosinophilia of any cause can cause the same type of tissue damage.

Researchers do not know all the factors that cause the dramatic increase in eosinophils that causes most cases of eosinophilia syndrome. However, we have identified some conditions or situations that may be the cause.

Myeloproliferative disorders these disorders are characterized by overproduction of blood cells in the bone marrow. High levels of interleukin 5 this is a protein produced by white blood cells. Genetic abnormalities that promote cell proliferation. The most common symptom of HES is a rash. Symptoms of hypereosinophilia correlate with areas of the body affected by high levels of eosinophils. For example, if the heart has abnormally high levels of eosinophils, symptoms of congestive heart failure, cardiomyopathy, myocarditis, and pericardial effusion may occur. Accumulation of eosinophils in the lungs can cause recurrent (repeated) upper respiratory tract infections, coughing, and dyspnea. Symptoms of HES are also present in many other medical problems, making initial diagnosis difficult. The first step is to rule out other conditions with similar symptoms. These include parasitic infections, allergic diseases, cancers, autoimmune diseases, and drug reactions. Allergists / immunologists are professionally trained to effectively diagnose the problem and, if HES is present, to work with other specialists such as hematologists and cardiologists to treat and monitor HES. The tests are personalized according to symptoms, stool tests to detect parasite infections, allergy tests to diagnose environmental or food allergies, biopsies of skin or other organs, blood to screen for autoimmunity. Testing or CT imaging of affected organs, and genetic

testing to detect FIP1L1-PDGFRA or other mutations that may include molecules to assist in identification, diagnosis, prognosis, and treatment. When diagnosing HES, it is important to determine the extent of organ damage. Chest x-rays and echocardiography are routinely done to evaluate the heart and lungs. Other tests commonly performed in HES patients include liver and kidney function, serum vitamin B12 levels, ESR, and serum tryptase levels.

Treatment goals include reducing the number of eosinophils in the blood, preventing organ damage, and slowing the progression of the disease. Treatment depends on the affected organ, the truth of the disease, and the presence of other medical problems that the patient may have. Treatment of hypereosinophil syndrome requires careful discussion with your healthcare provider about the risks and benefits of treatment for the involvement of certain HES-related organs. HES treatment may include glucocorticoids (i.e., prednisone) and chemotherapeutic agents such as chlorambucil, hydroxyurea, and vincristine. Frequent injections of interferon alpha may also be prescribed.

HES are on-going research areas, including the use of tyrosine kinase inhibitors (i.e., Gleevec, dasatinib, and nilotinib) and anti-interleukin-5 (IL-5) monoclonal antibodies (mepolizumab and reslizumab). Systemic steroids are often needed to treat HES with systemic symptoms such as organ involvement or severe rash, fluid retention. Steroids are drugs that fight (suppress) many types of inflammation. They are not specific for eosinophil suppression, but eosinophils are particularly sensitive to them. Systemic steroids that are taken into the bloodstream (orally or intravenously) are very effective in treating many eosinophil disorders. Steroids are very effective in controlling blood eosinophil counts, and most HES patients can be treated with oral steroids (called prednisone) for a long period of time with good disease control. However, withdrawal of steroids generally causes blood eosinophils and symptoms of the disease to recur. Unfortunately, long-term use of steroids (especially at high doses) is associated with certain side effects. Serious side effects include osteoporosis (fragile bone caused by weak bones), infections, adrenal insufficiency (the body does not respond properly to disease and stress), and avascular necrosis (joints, usually hip bones). Common side effects include fluid retention (swelling), increased appetite, “moon face”, and hypersensitivity. Interferon alpha (IFNa) is used for a variety of diseases, including infectious diseases (such as hepatitis) and malignant tumors (such as certain types of leukemia). IFNa has been shown to be effective in HES by suppressing disease-related symptoms. However, toxicity is a major barrier to the use of this treatment. IFNa is usually injected into the subcutaneous adipose tissue 35 times a week. At the beginning of treatment, most patients experience flu-like symptoms such as fever, chills, muscle aches, headaches, and arthralgias. Other side effects of IFNA are increased liver enzymes that require hypocytic counting and careful monitoring. These side effects are usually reduced with time, but after long treatment can appear in different forms of different toxicity. The entire experience with IFNA in myeloproliferative tumors is that about 2530% of the patient is required for the robbery of the treatment. In recent years, new years of IFNA (pegylated interferons) has been developed and is currently approved as a treatment of hepatitis. These drugs are administered only once a week and thus may be better acceptable.

Cyclosporin is a stronger drug that compresses the immune system, which is mainly used to prevent organ transplant to organ transplant. Some patients with HES have evidence that immune cells play a role in supporting the disease, and there is evidence that cyclosporin can play a role in such a role in such a case. Antine-biologically active ingredients (chemotherapy) provide an alternative approach to the treatment of HES travel cases. These are chemotherapeutic agents that can control the disease. They are used to treat many malignancies and are not specific for eosinophilic diseases. They are a strong drug that kills strong cells, which may have a harmful side effect (Eosonophiles in HES) and only heavy cases are reserved. Careful monitoring when taking these drugs is essential. Chemotherapeutic agents used in HES include hydroxyurea, methotrexate, etoposide, cyclophosphamide, vincurostin and cladribine. Gleevec (iMatinib Mesylat) is a tyrosine kinase inhibitor. As a result of cell proliferation studies, scientists could develop a group of therapeutic agents known as tyrosinin case inhibitors (TKI). Tyrosinase is an enzyme in cells having various functions. By blocking the ability of tyrosine kinase to function, TKI provides valuable tools to control malicious cell proliferation. A few years ago, some discoveries of the genetic abnormal HES patients involved as PDGFFRα were born. In these cases, an abnormality in PDGFRα seems to be involved in disease diseases. Gleevec is TKI and inhibits PDGFFRα and eliminates the disease in HES patients with PDGFRα abnormalities. In our case, we saw the participation of various systems. Initiation of treatment with corticosteroids is essential and urgent, especially if the patient suffers from severe or life-threatening organ failure. Some cases are resistant to corticosteroids and may require alternative therapy. Finally, thromboembolic disease associated with HES is particularly difficult to control.