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Commentary

Clinical Diagnosis and Symptoms of Systematic Lupus Erythematosus

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DESCRIPTION

Systematic Lupus Erythematosus (SLE) is a disease with an unknown aetiology that manifests itself in a variety of ways. SLE is an autoimmune disorder that causes multisystemic microvascular inflammation as well as the production of numerous autoantibodies, particularly antinuclear antibodies. Patients clinical features range from minor joint and skin involvement to life-threatening kidney, hematologic, or central nervous system involvement. The clinical heterogeneity of SLE the lack of pathognomonic features or tests makes diagnosis difficult. SLE is a chronic disease that causes inflammation in connective tissues, such as cartilage and the lining of blood vessels, which provide strength and flexibility to structures throughout the body.

SYMPTOMS

The signs and symptoms of SLE vary from person to person and can affect many organs and systems, including the skin, joints, kidneys, lungs, central nervous system, and blood-forming (hematopoietic) system. SLE is one of a large group of conditions known as autoimmune disorders, which occur when the immune system attacks the body's own tissues and organs. SLE symptoms include extreme tiredness (fatigue), a vague feeling of discomfort or illness (malaise), fever, loss of appetite, and weight loss. Most affected people also have joint pain, which usually affects the same joints on both sides of the body, as well as muscle pain and weakness. Skin problems are common among SLE patients. A flat, red rash across the cheeks and bridge of the nose, dubbed a "butterfly rash" due to its shape, is a distinguishing feature. Normal variations (polymorphisms) in many genes can influence the risk of developing SLE, and multiple genetic factors are thought to be involved in most cases. SLE is caused in rare cases by variants known as mutations in single genes. The majority of the genes linked to SLE are involved in immune system function, and changes in these genes are likely to affect proper immune response targeting and control. Sex hormones as well as a number of environmental factors such as viral infections, diet, stress, chemical exposures, and sunlight, are thought to play a role in the onset of this complex disorder.

DIAGNOSIS AND TREATMENT

A health care provider will use symptom assessments, physical examinations, X-rays, and lab tests to diagnose SLE. SLE can be difficult to diagnose because its early signs and symptoms are vague and can mimic those of other diseases. If only a blood test is used for diagnosis, one case of SLE may be misdiagnosed. Several dermatologic entities that can mimic LE-specific lesions should be considered in patients who have atypical features and/or are resistant to standard therapy. Acne rosacea can cause a red face and is frequently misdiagnosed as acute cutaneous LE. Photosensitive psoriasis can mimic papulosquamous SCLE, and erythema multiforme can occasionally be confused with annular SCLE. Other common dermatoses, such as contact dermatitis, eczema, and seborrheic dermatitis, may coexist in SLE patients. Because of the number of organs that can be affected, treating SLE frequently necessitates a collaborative approach.

SLE treatment primarily consists of immunosuppressive drugs that inhibit immune system activity. SLE is frequently treated with hydroxychloroquine and corticosteroids. SLE can coexist with other autoimmune diseases that necessitate additional treatment, such as Sjogren's syndrome, antiphospholipid syndrome, thyroiditis, hemolytic anaemia, and idiopathic thrombocytopenia purpura. The purpose of treatment is to alleviate symptoms. Treatment options vary depending on the severity of symptoms and the parts of body affected by SLE. Anti-inflammatory medications for joint pain and stiffness steroid creams for rashes, corticosteroids to reduce the immune response, antimalarial drugs for skin and joint problems, disease-modifying drugs, or targeted immune system agents for more severe cases, are among the treatments that may be used.

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