Systemic Lupus Erythematosus (SLE) Assessment

Systemic lupus erythematosus (SLE) is an autoimmune disease that activates the inflammatory response and damages healthy tissue and organs. This multisystem inflammatory disorder especially affects the skin, joints, and kidneys. Symptoms include a butterfly rash, red lesions, arrhythmias, hemolytic anemia, seizures, and glomerulonephritis. An anti-nuclear antibody assay (ANA) is used to diagnosed SLE. Since patients with SLE will experience periods of exacerbations and remissions, supportive therapy is critical for maintaining self-esteem (refer to the Picmonic on “Systemic Lupus Erythematosus (SLE) Interventions”).



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Mechanism

Multisystem Inflammatory Disorder
Multiple-systems In-flames
Systemic lupus erythematosus is a type III immune-complex reaction that causes multisystem inflammation. In SLE, the cell membrane is injured and damaged DNA enters the bloodstream. The DNA antigen reacts with an antibody and activates the complement system. Although phagocytes usually remove foreign bodies, these complexes are too small to be effectively removed. Instead, they are deposited in the basement membranes of capillaries in the kidneys, heart, skin, brain, and joints. The activation of the inflammatory response triggers the release of chemotactic factors and cause tissue inflammation and destruction. Fever and fatigue are common findings with the a fever being a sign of a “flare” or exacerbation.

Assessment

Malar Rash
Monarch-face-butterfly
A malar, or butterfly rash over the cheeks and nose is a classic symptom of SLE. Nearly 50% of patients diagnosed with SLE develop a butterfly rash.

Discoid Rash
Disco Rash
Patients with SLE may experience a discoid rash, described as erythematous lesions in any location of the body. The cutaneous vascular lesions typically develop in sun-exposed areas such as the neck, face, scalp, and ears. Some patients may develop coin-shaped discoid lesions on their body, which leave scars.

Arrhythmias
Broken Arrhythmia-drum
Systemic lupus erythematosus may cause stenosis of sinoatrial and atrioventricular nodes. SLE-related cardiac involvement suggests advanced disease process and significantly increases the risk of mortality. The inflammatory process of SLE may also cause pericarditis with symptoms of hypertension and hypercholesterolemia.

Arthritis
King-Arthur
Since the antibody complexes tend to deposit in the joints, the majority of patients with SLE will develop arthritis. Morning joint stiffness caused by polyarthralgia may precede the onset of SLE for years before diagnosis. Symptoms include diffuse swelling, stiffness, and severe joint and muscle pain. Although arthritis caused by SLE is nonerosive, it may cause deformities and increases the risk of bone loss and fracture.

Hemolytic Anemia
Hemolysing-RBCs from Anemone
SLE may lead to the formation of antibodies against blood cells. Some patients may develop hemolytic anemia related to the destruction of erythrocytes. The destruction of leukocytes increases the patient's risk of infection. Since infection is the major cause of death in patients with SLE, instruct the patient to report any fever that may indicate an underlying infectious process.

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Seizures

Caesar

Since the immune complexes may deposit into the brain, SLE may cause brain involvement. Patients with SLE may experience generalized or focal seizures. Drug therapy may include corticosteroids or anti-seizure medications.

Glomerulonephritis

Glow-mare

The inflammatory process associated with SLE causes kidney damage and may result in glomerulonephritis. Nearly 40% of patients with SLE will develop lupus nephritis within 5 years after initial SLE symptoms appear. Patients who develop rapidly progressing glomerulonephritis will lose renal function within a few weeks. Drug therapy may include corticosteroids, cytotoxic agents, and immunosuppressive agents.

Considerations

Periods of Exacerbation and Remission

Timeline of Attacks and Remission

Patients with SLE will experience chronic unpredictable periods of disease exacerbation and remission. Since hormones may exacerbate symptoms of SLE, symptoms may worsen after the onset of menarche, during pregnancy, and in the immediate postpartum period. Oral contraceptives may also trigger symptoms. Other triggers include sun exposure, infections, and certain medications.

Diagnosed by ANA (Anti-nuclear antibody assay)

Diagnostic-computer with Anti-nuclear Ant-tie-body

Since there is no specific diagnostic test, diagnosis of SLE is based on patient history, physical examination, and laboratory findings. The anti-nuclear antibody assay (ANA) may be used to determine the presence of antibodies that may damage healthy tissue and lead to problems associated with SLE.

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