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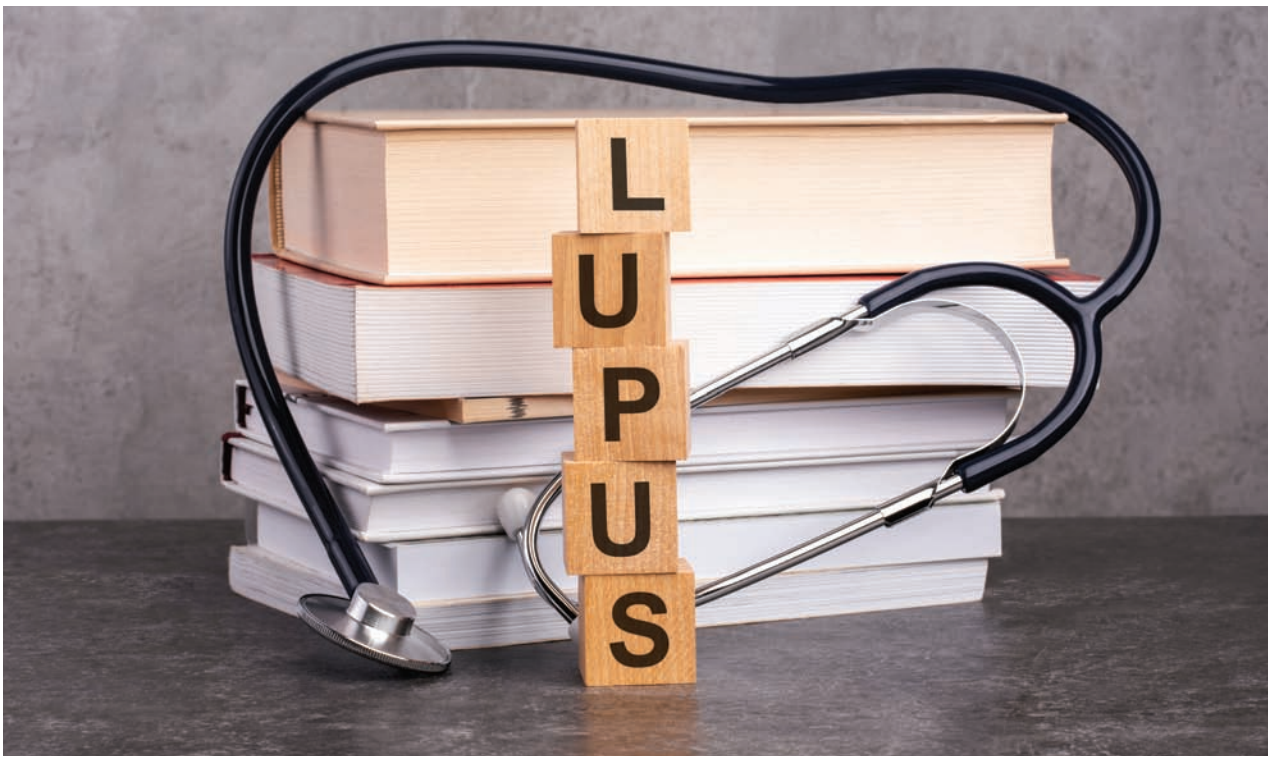

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Selected nursing interventions for systemic lupus erythematosus

This article presents the pathogenesis, signs, symptoms, and management of systemic lupus erythematosus.

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Systemic lupus erythematosus (SLE), an inflammatory autoimmune disease, results from immune system dysregulation that can cause multiorgan damage, variable signs and symptoms, and periods of flares and remission.[1,2] In autoimmune diseases, a person's immune system mistakenly determines their body organs and tissues to be foreign and attacks them. SLE symptoms can range from mild to life-threatening and its diversity and complexity of presentation can create challenges in diagnosis and treatment.[1,2] It's often called "the great imitator" because it mimics many diseases.[3] This article presents the pathogenesis, signs, symptoms, and management of SLE.

Epidemiology and risk factors

Thirteenth-century physicians used the word "lupus," meaning "wolf" in Latin,

to describe a facial rash pattern that was often noted in young women, thinking it resembled the facial features of a wolf.[3] "Erythematosus" means "redness" of the skin, a misnomer, because SLE can affect any body organ or tissue.[3] The degree of erythema varies among individuals. SLE affects 5 million people globally, including 1.5 million people in the US.[4-6] Global health disparities impacting SLE outcomes include poverty, access to insurance, medications, food, housing, transportation, and mental health resources.[6,7] People with SLE may find it challenging to cope with chronic pain, fatigue, and disability.[1,5] Today, the major causes of death in patients with SLE are renal disease, infections, central nervous system complications, thrombosis, and cardiovascular disease.[8,9] More than 80% of people with SLE are expected to live an average lifespan with treatment, close surveillance, and lifestyle modification, compared with 50% of patients living less than 5 years after SLE diagnosis in the 1950s.[3,9,10]

People of Black, Asian, Pacific Islander, Latino, and Native American descent are affected by SLE two to three times more often than White people.[4,6,7,11] Multiple genes are implicated in the pathogenic origins of SLE, particularly the major histocompatibility complex, where the human leukocyte antigen region and the toll-like receptor 7 gene are located.[1,12] Some medications cause an increase in oxidative stress, including isoniazid, procainamide, hydralazine, and minocycline, and may trigger immune system dysregulation which can lead to an SLE-like syndrome. Smoking; physical injury; emotional stress; and infections such as Epstein-Barr virus, cytomegalovirus, and varicella-zoster virus may predispose a person to the disease.

People of all skin tones are at risk for skin damage from UV light from the sun, tanning salons, and indoor halogen lighting.[4-6] UV light penetrates deep into the skin and can damage a person's DNA, which can cause an immune system response.[9,13]

The female-to-male ratio of SLE is 9:1 during childbearing years; however, the ratio is 3:1 during childhood and after menopause.[4,6,14] Estrogen levels during childbearing years are implicated in disease development and flares. Estrogen stimulates T-lymphocyte and B-lymphocyte activity, giving females a more robust immune system than males and with that, a propensity for autoimmune diseases.[1,4,12] On the other hand, testosterone has an immunosuppressive effect on T-lymphocyte and B-lymphocyte activity, possibly protecting males from autoimmune disease.[15,16] Research shows that males with SLE don't have abnormal estrogen levels and are just as sexually active and fertile as males without the disease.[9] Males with SLE are typically diagnosed after age 40 and may present with more severe renal and cardiovascular manifestations.[9]

Pathogenesis

The pathogenesis of SLE involves an interplay of genetic, hormonal, environmental, and immunologic factors that can lead to tissue injury and DNA damage.[1,12,17] DNA damage causes the cell to undergo programmed cell death (apoptosis) and die.[1,12,17] Small apoptotic bodies circulate and expose the inside of a cell and nucleus to the rest of the body. The immune system incorrectly identifies the nucleus components of the cell such as DNA, histones, cardiolipin, and other proteins as antigens and begins an immune attack. Because these components originate from the nucleus, they are called nuclear antigens. Inefficient clearance of apoptotic bodies also results in an abundance of nuclear antigens. B-lymphocytes begin to produce antibodies against components of the nucleus, called antinuclear antibodies (ANAs), and mediate inflammation with proinflammatory cytokines (proteins), predominantly interferon-alpha (IF-alpha).[1,12,17] The antibodies

produced incorrectly identify normal proteins (self-proteins or autoantibodies) as being foreign and harmful to the person. ANAs are present in 95% of people with SLE.[18,19] T-lymphocytes regulate B-lymphocyte responses and potentiate inflammation by dysregulation of proinflammatory interleukins (ILs), including IL-2, IL-17, and IL-21.[1,12,17-19] These nuclear antibodies bind to nuclear antigens, forming immune complexes (one antibody + one antigen together) that deposit into blood vessels and body tissues, initiating local inflammation and damage by activating the complement system. The complement system is made up of more than 30 proteins released by the liver in abundance with acute inflammation and infection to rid the body of antigens.[1,9,12,17-19] Abnormally low complement levels, particularly complement-3 (C3) and complement-4 (C4), indicate that chronic inflammation is consuming proteins. Low C3 and C4 are markers of active disease.[20]

Clinical presentation

The clinical presentation of SLE differs dramatically among patients. Some have mild disease, whereas others rapidly progress to life-threatening stages.[21-23] The most common initial presentations are fatigue, fever, nonerosive arthritis, arthralgia, weight loss, and cutaneous manifestations. The most common arthritis symptoms in patients with SLE are aching and stiffness.[21-23] Deforming joint abnormalities manifested in rheumatoid arthritis rarely occur in patients with SLE.[9,21] A decreased red blood cell (RBC) count may indicate anemia secondary to chronic inflammation.[9,21] Hemolytic anemia may occur when the immune system attacks RBCs. Decreased white blood cells, particularly neutrophils (neutropenia), results from treatment with immunosuppressive medications or an immune-mediated attack on the bone marrow. Reduction in platelets (thrombocytopenia) may indicate an immune-mediated attack on the bone marrow. Renal studies may be abnormal.[9,21]

Antibodies in SLE and related autoimmune diseases

Selected nuclear antigen (specific ANA)	Selected clinical association
Double-strand DNA (ds-DNA)	Systemic lupus erythematosus Lupus nephritis
Ribonuclear protein (RNP)	Systemic lupus erythematosus Mixed connective tissue disease Scleroderma
Smith (SM)	Systemic lupus erythematosus
Sjogren Syndrome Type A (SSA) (also known as "Ro")	Sjogren syndrome Systemic lupus erythematosus Subacute cutaneous lupus erythematosus (SCLE) (rash and photosensitivity) Neonatal lupus erythematosus and congenital heart block Rheumatoid arthritis
Sjogren Syndrome Type B (SSB)	Sjogren syndrome Systemic lupus erythematosus
Scleroderma-70 (Scl-70)	Systemic sclerosis
Anti Jo-1	Dermatomyositis Polymyositis
Histone	Drug-induced lupus
Chromatin	Systemic lupus erythematosus Lupus nephritis

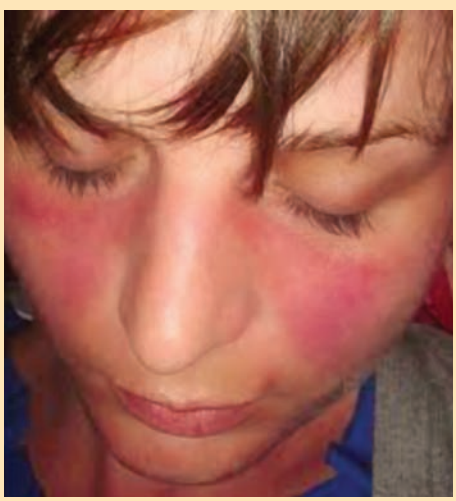
For example, a patient with high serum creatinine, low estimated glomerular filtration rate, proteinuria, low serum proteins, edema, and hypertension may indicate lupus nephritis (LN), a life-threatening complication of SLE. A renal biopsy is required to confirm a diagnosis of LN. Decreased C3 and C4 levels, elevated erythrocyte sedimentation rate, and elevated C-reactive protein reflect active inflammation and should be correlated with antibodies associated with SLE and other autoimmune diseases.[9,20,21] Combining broad ANA detection with specific antigenic ANA testing is a common practice.[18,19,24] For example, patients with a positive ANA may be tested for antibodies against histones and DNA and extractable nuclear antigens. Individual antibodies may indicate a specific clinical presentation (see *Antibodies in SLE and related autoimmune diseases*).[18,19,24]

Mucocutaneous manifestations are common SLE features encompassing lupus-specific and nonspecific rashes and lesions. **The first image features** a lupus-specific malar rash sparing the nasolabial folds that may be flat or raised.[13,21,25]

The second sidebar illustrates a lupus-specific papulosquamous subacute cutaneous lupus erythematosus, sometimes called "psoriasiform" because it resembles psoriasis.[13,21,25] The rashes in acute cutaneous lupus erythematosus (ACLE) malar rash and lupus-specific papulosquamous subacute cutaneous occur secondary to exposure to the sun and indoor lighting. Other specific lesions include chronic cutaneous lupus erythematosus and discoid lupus erythematosus (DLE), which may exist without systemic manifestations. Nonspecific mucosal and skin lesions, including nonscarring alopecia, vasculitis (inflammation of blood vessels in the skin), and urticaria, are common in SLE. Rashes and lesions cause pain, discomfort, and body image disturbances and may indicate inflammation in body organs and tissues.[13,21,25]

Neuropsychiatric syndromes in SLE include headache, cognitive dysfunction, anxiety, depression, mood disorder, aseptic meningitis, stroke, seizure, polyneuropathy, Guillain-Barre syndrome, and myasthenia gravis.[9,21] Abdominal pain, anorexia, nausea, vomiting, and diarrhea may occur secondary to medications to treat SLE or complications of SLE, including peritonitis, pancreatitis, and vasculitis of the mesentery and bowel.[9,21] Elevated liver enzymes are rarely caused directly by SLE, but likely result from the medications to treat the disease or autoimmune hepatitis. SLE can affect the eye in many ways. The most common eye symptom is keratoconjunctivitis sicca, manifested by itching, burning, and blurring eyes.[9,21] A chest X-ray may show pleural effusion. An echocardiogram may reveal valvular disease and pericardial effusion and may reveal conduction disturbances causing dysrhythmias.[9,21]

Acute cutaneous lupus erythematosus malar rash (also known as butterfly rash)



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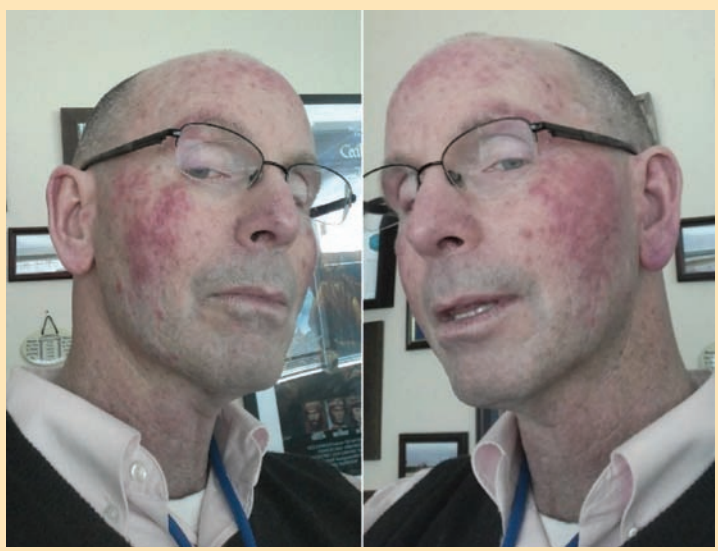
Subacute cutaneous lupus erythematosus (papulosquamous subtype)



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Revised criteria for the classification of SLE

Criteria	Description
Malar rash	Raised or flat erythema over the malar eminences sparing nasolabial folds.
Discoid rash	Erythematosus raised patches with keratotic scale and follicular plugging with or without scarring.
Photosensitivity	Rash from an unusual reaction to sunlight.
Oral ulcers	Oral or nasopharyngeal ulcers or lesions.
Arthritis	Nonerosive arthritis in two or more peripheral joints.
Serositis	Pleuritis OR pericarditis.
Renal disorder	Persistent proteinuria ≥0.5 g/day OR cellular casts and blood in the urine.
Neurologic disorders	Seizures OR psychosis in the absence of offending drug or metabolic imbalances.
Hematologic disorder	Hemolytic anemia OR leukopenia, lymphopenia, and thrombocytopenia.
Immunologic disorder	Abnormal levels of anti-ds DNA OR anti-Smith OR antiphospholipid antibodies: anticardiolipin and lupus anticoagulant.
Positive ANA	Abnormal levels of antinuclear antibody in the absence of drugs known to cause positivity.

Adapted from the American College of Rheumatology

The American College of Rheumatology (ACR) revised the 1982 criteria for the classification of SLE in 1997 (see *Revised criteria for the classification of SLE*). A person meeting 4 of the 11 criteria simultaneously or serially indicates SLE.[21,26,27] The ACR and European League for Rheumatism developed criteria to identify early-stage SLE (see *2019 Classification for SLE*). Patients must have a positive ANA to be eligible to use the classification system. Each criterion is assigned a number. Only the highest numbered criterion in a domain is counted toward the total score. A total score of 10 or more plus one criterion met is classified as SLE. A person must meet criteria simultaneously or serially. Both classification systems are tools that providers use to correlate complex clinical features in SLE, exclude differential diagnoses, and manage the patient.[21,26,27]

Management

Medication treatment is the standard intervention to manage inflammation in SLE. Medication classifications include corticosteroids (CS), disease-modifying antirheumatic drugs (DMARDs), and biological response modifiers (BRMs).[9,28-34] (See *Common SLE medications and priority assessments*.)[28-34]

Selected nursing interventions

1. Physical assessment. Conduct a head-to-toe physical assessment. Mood changes may be related to fatigue, medications, and depression from chronic illness. Changes in cognition, speech, and motor movement may indicate inflammation of the central nervous system. Assess joints for redness, swelling, pain, and limitation in range of motion. Erythema, rashes, and mucosal lesions may indicate inflammation in the body's vital organs. Assess respiratory rate, depth, rhythm, and oxygen saturation. Auscultate the lungs for pleural friction rubs and crackles. Assess cardiac rate and rhythm. Auscultate the heart valves for murmurs from valvular dysfunction and pericardial friction rubs. Assess the abdomen for bowel sounds, pain, or

2019 Classification for SLE

Domain	Criterion	Points
Constitutional	Fever	2
Hematologic	Leukopenia	3
	Hemolytic anemia	4
	Thrombocytopenia	4
Neuropsychiatric	Delirium	2
	Psychosis	3
	Seizure	5
Mucocutaneous	Nonscarring alopecia	2
	Oral ulcers	2
	SCLE or DLE	4
	ACLE	6
Serosal	Pleural or pericardial effusion	5
	Acute pericarditis	6
Musculoskeletal	Joint involvement	6
Renal	Persistent proteinuria ≥0.5 g/day	4
	Renal biopsy class II or V lupus nephritis	8
	Renal biopsy class II or IV lupus nephritis	10
Antiphospholipid antibodies	Anticardiolipin OR Anti-beta$_2$ glycoprotein 1 OR lupus anticoagulant	2
Complement proteins	Low C3 OR C4	3
	Low C3 AND C4	4
SLE-specific antibodies	Anti-ds-DNA OR anti-Smith	6

Adapted from the 2019 ACR-EULAR Classification Criteria

distension. Assess for infections, pain level, and quality of life.[9,21,29]

2. Lab and imaging. Correlate any physical assessment findings with lab indicators of inflammation. For example, elevated C-reactive protein and erythrocyte sedimentation rate and low RBCs, C3, and C4. Imaging studies also indicate inflammation. For example, ultrasound and MRI show inflammation and damage to joints and soft tissues without synovitis and bone erosion.[9,21,29]

3. Medication responses. Systemic medications control inflammation. The efficacy of these medications is measured with improved symptoms and minimal to no adverse reactions (see *Common SLE medications and priority assessments*). Nurses should teach patients to report adverse reactions to medications, including but not limited to an increase in pain or discomfort, the onset of fever (infection and increased inflammation), bleeding from anywhere in the body, weight gain or loss, changes in mental status, abdominal pain or discomfort, and skin rashes and lesions. Nurses should teach patients to apply a sun protection factor greater than 30 to protect the skin from harmful light sources when taking azathioprine and have sufficient hydration when taking cyclophosphamide to avoid hemorrhagic cystitis.[28-34]

4. Nutrition. Foods with anti-inflammatory properties should be encouraged, including fish, vegetables, fruit, beans, legumes, seeds, whole grain bread, oats, olive oil, and tofu. Overweight and obesity proinflammatory cytokines that can worsen SLE. Patients should consult a dietitian to help them with healthy food choices.[9,29,35]

5. Health promotion. Patients with SLE may have challenges coping with chronic illness and comorbidities unrelated to SLE. Strategies to promote quality of life include (1) adhering to the treatment plan, (2) receiving vaccinations as indicated, (3) avoiding people who are sick, (4) wearing an N95 or KN95 mask if receiving immunosuppressive medications, (5) sun protection such as wide-brimmed hats, wearing sunglasses, and applying sun protection factor greater than or equal to 30, (6) wearing comfortable clothing, (7) getting plenty of sleep, (8) resting at intervals, (9) being physically active, (10) getting a hobby, (11) avoiding smoking and excessive alcohol consumption, and (12) engaging in support groups.[9,29,35]

Conclusions

The primary goals in managing SLE are controlling inflammation to protect body organs and tissues from damage, reducing pain and discomfort, and promoting quality of life. Research continues to result in new medications to treat SLE. Thankfully, medication therapy and

Common SLE medications and priority assessments

Medication	Therapeutic action	Priority assessments
CS • Prednisone • Prednisolone • Methylprednisolone • Dexamethasone • Hydrocortisone • Triamcinolone • Budesonide	Suppresses migration of leukocytes and reversal of increased capillary permeability to decrease inflammation and causes immunosuppression	• Mood changes: Medication alters neurotransmitters in the brain • Hypertension • Dysrhythmias • Bleeding • Capillary fragility with easy bruising • Hypokalemia and hypernatremia • Weight gain: Increased appetite, sodium retention, edema • Increased blood glucose, alanine transaminase, aspartate aminotransferase, alkaline phosphatase, and cholesterol • Reduced platelet count and prolonged partial thromboplastin time • Medication masks infection: Potential for severe infection • Fractures: Osteoporosis • Visual changes: Cataracts
DMARD • Hydroxychloroquine	Inhibits proinflammatory cytokine release, slows movement of neutrophils, and impairs antibody-antigen responses to reduce inflammation. Mild immunosuppression.	• Elevated ALT, AST, and ALP: Hepatic toxicity • Elevated creatinine: Renal toxicity • Neutropenia: Infection • Dysrhythmias: Baseline and periodic ECG • Visual problems: Retinal damage (reversible) • Bleeding, bruising, and skin eruptions • Hypoglycemia: Medication increases insulin • Skin discoloration and rashes
DMARD • Azathioprine	Inhibits purine metabolism and T-lymphocyte expression causing immunosuppression to reduce inflammation	• Elevated ALT, AST, and ALP: Hepatic toxicity • Elevated creatinine: Renal toxicity • Reduced PLTs and prolonged PTT: Bruising and bleeding • Reduced white blood cell (WBC) and fever: Infection • Nausea and vomiting: Take with food • Fatigue and arthralgia
DMARD • Mycophenolate mofetil	Inhibits inosine monophosphate dehydrogenase, which involves purine synthesis by inhibiting T lymphocytes and B lymphocytes reducing inflammation. Immunosuppressant.	• Elevated ALT, AST, and ALP: Hepatic toxicity • Elevated creatinine: Renal toxicity • Hyperkalemia or hypokalemia • Hypocalcemia, hypomagnesemia, hypoglycemia • Elevated cholesterol and triglycerides • Reduced WBC and fever: Infection • Reduced PLTs and prolonged PTT: Bruising and bleeding • Reduced RBCs: Anemia • Nausea, vomiting, headache, anxiety, tremor • Increases susceptibility to malignancy by altering DNA
DMARD • Voclosporin	Reduces proinflammatory cytokines including IL-2, thereby inhibiting production of T lymphocytes to reduce inflammation. This medication is specifically for the treatment of active lupus nephritis. Immunosuppressant.	• Elevated creatinine and decreased estimated glomerular filtration rate: Renal toxicity • Elevated ALT, AST, and ALP: Hepatic toxicity • Seizures, tremors, mental status changes: Neurotoxicity • Reduction in RBCs: Anemia • Changes in cardiac conduction • Hypertension • Hyperkalemia • Reduced WBC and fever: Infection • Fatigue, mouth ulcers, abdominal pain, diarrhea, headache • Increases susceptibility to malignancy by altering DNA • Combined with mycophenolate mofetil

(Continues)

Common SLE medications and priority assessments *(Continued)*

Medication	Therapeutic action	Priority assessments
DMARD • Cyclophosphamide	Inhibits immune activity by blocking DNA synthesis, causing profound immunosuppression to decrease inflammation	• Marked reduction in WBC (neutropenia): Infection • Reduced RBCs and PLTs: Anemia, bruising, and bleeding • Elevated ALT, AST, and ALP: Hepatic toxicity • Dysrhythmias, pericarditis, heart failure: Cardiotoxicity • Pneumonitis and pulmonary fibrosis: Pulmonary toxicity • Dilutional hyponatremia from overhydration to prevent hemorrhagic cystitis • Hematuria: Hemorrhage cystitis from medication metabolite • Nausea, vomiting, diarrhea, abdominal pain, and mucositis • Skin rashes and alopecia • Increases susceptibility to malignancy by altering DNA • Administered with Mesna to protect the bladder from bleeding
BRMs • Anifrolumab • Belimumab • Rituximab	Inhibits proinflammatory cytokines to reduce inflammation. Immunosuppressant.	• Elevated ALT, AST, and ALP: Hepatic toxicity • Reduced WBC and fever: Infection • Changes in respiratory rate and depth (might indicate anaphylaxis) • Cough and rhinitis • Changes in BP: Hypotension or hypertension • Reduced RBCs: Anemia • Reduced PLTs and prolonged PTT: Bleeding • Fatigue, anorexia, nausea, vomiting, diarrhea, headache, insomnia • Skin rashes • Increased risk for malignancy

lifestyle modification have substantially improved morbidity and mortality in patients with SLE since the 1950s. In all, SLE is a complex illness that requires the nurse to use critical thinking and clinical reasoning to manage the patient safely. ■

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