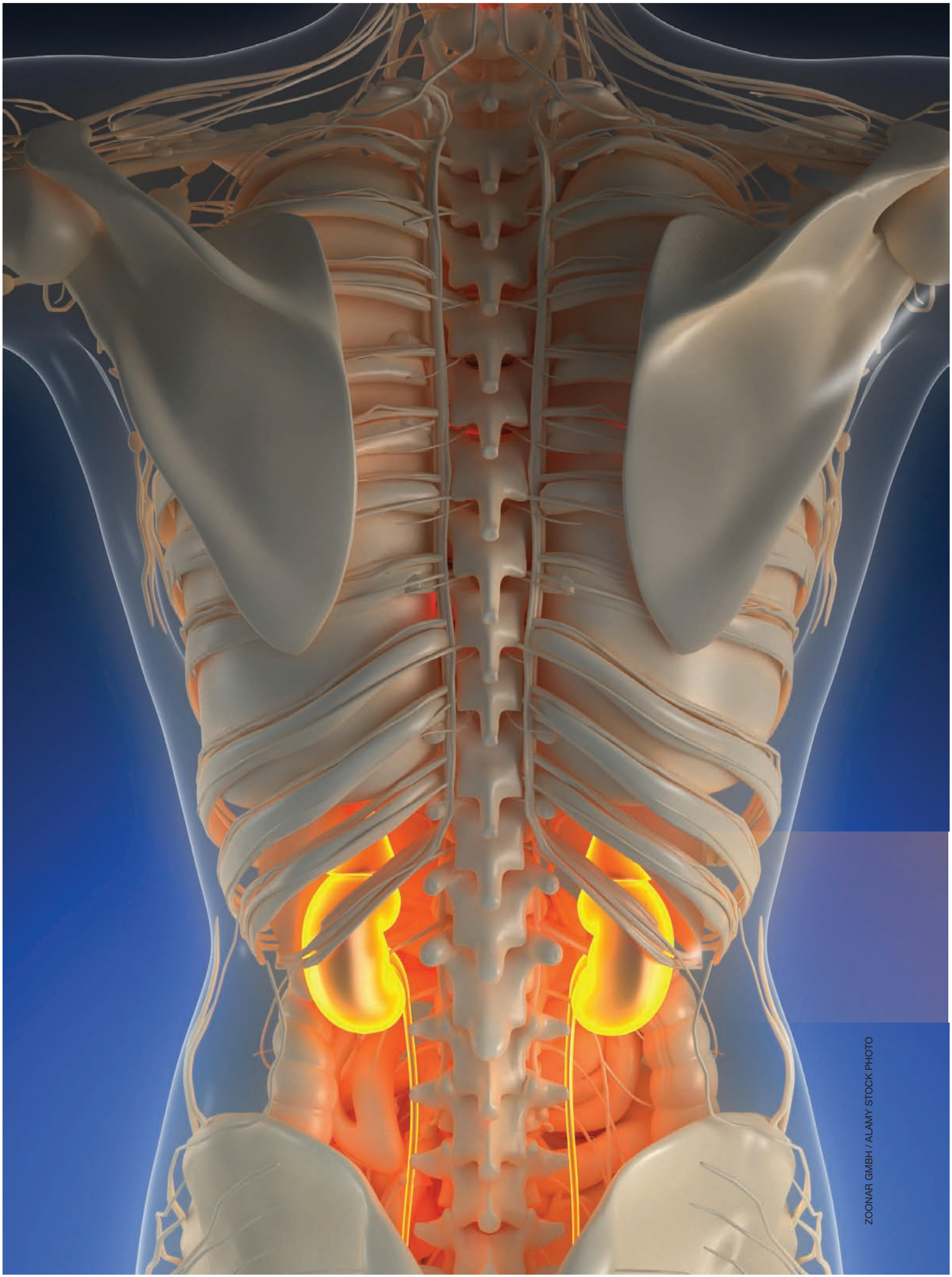

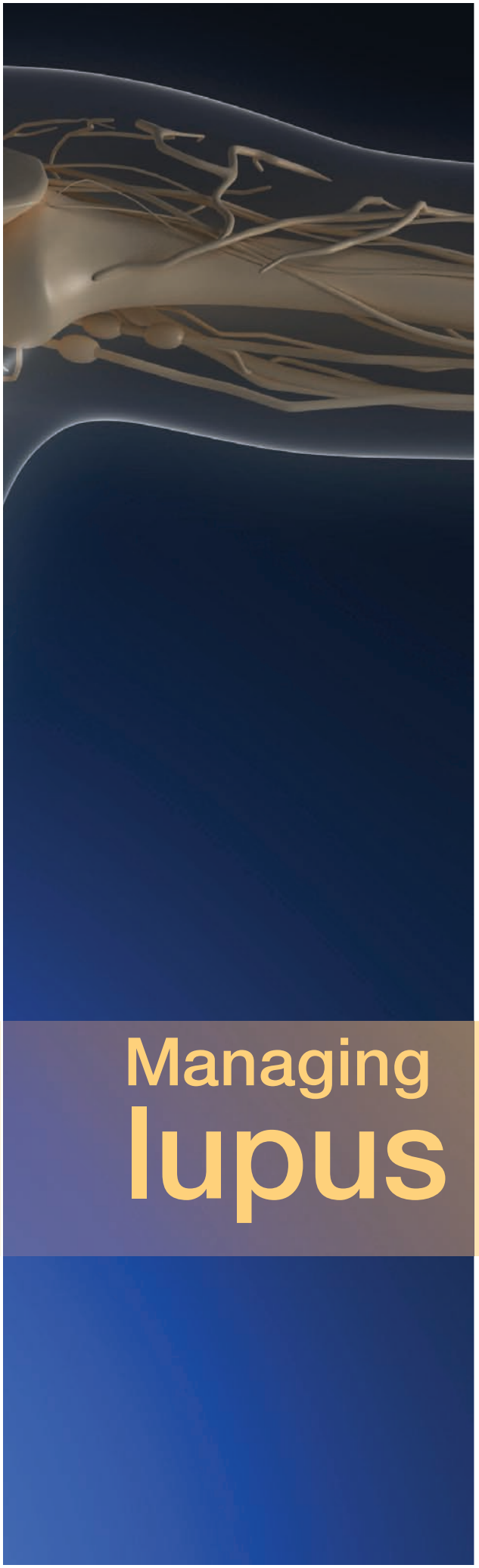

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Approximately 40% of all patients with systemic lupus erythematosus develop this life-threatening condition.

By Richard L. Pullen, Jr., EdD, MSN, RN, CMSRN

Lupus nephritis, sometimes referred to as lupus glomerulonephritis, is a complication of systemic lupus erythematosus (SLE) that results from inflammation in the kidneys. Lupus nephritis usually occurs within the first several years of being diagnosed with SLE; incidence is higher in children and ethnic minorities. Once a diagnosis of lupus nephritis is established, Black, Hispanic, and Asian patients tend to have more severe symptoms than White patients, which may be related to disparities in accessing healthcare and the presence of comorbidities such as hypertension and diabetes mellitus.

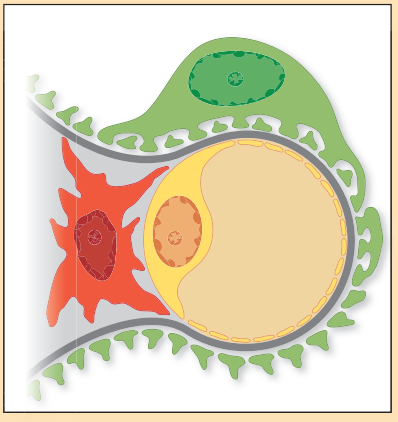
Managing lupus nephritis

Overview of SLE

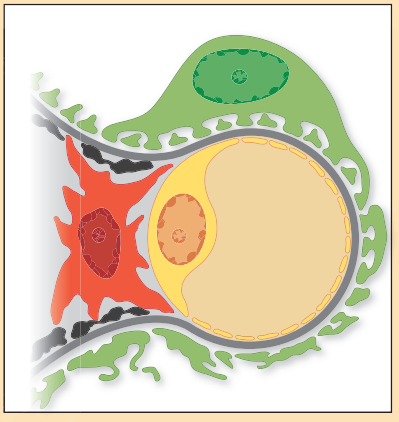
Lupus is a chronic inflammatory autoimmune disease that affects approximately 1.5 million people in the United States and 5 million people worldwide, according to the Lupus Foundation of America. Lupus is sometimes confined only to the skin, referred to as discoid or cutaneous lupus

Picturing lupus nephritis

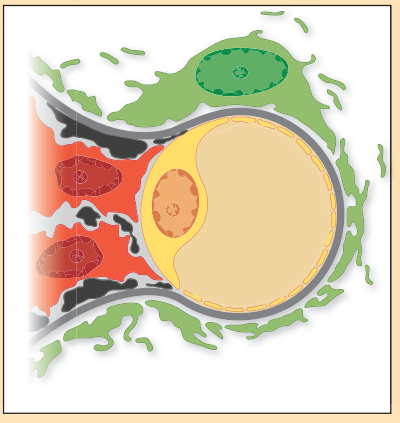
Normal
Normal capillary, with intact podocyte foot processes and no electron-dense deposits.



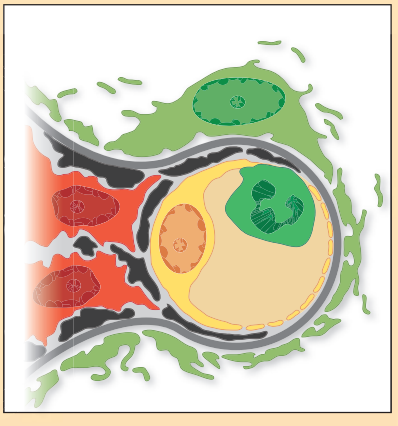
Lupus nephritis I
Class I lupus nephritis, with minimal mesangial deposits, focal foot process effacement, and no mesangial hypercellularity.



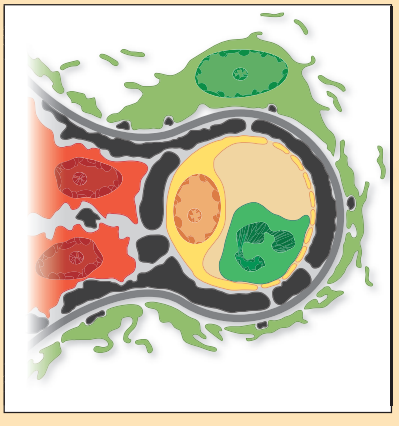
Lupus nephritis II
Class II lupus nephritis, with substantial mesangial deposits, mesangial hyperplasia, and more extensive foot process effacement.



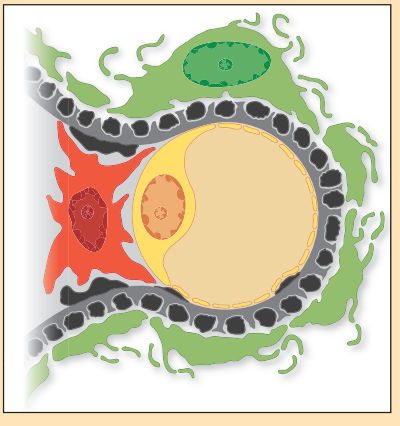
Lupus nephritis III
Class III lupus nephritis, with scanty subendothelial deposits, mesangial hyperplasia, endocapillary leukocytes, and extensive foot process effacement.



Lupus nephritis IV-G
Class IV-G lupus nephritis, with numerous subendothelial and a few subepithelial deposits, mesangial hyperplasia, endocapillary leukocytes, and extensive foot process effacement.



Lupus nephritis V
Class V lupus nephritis, with numerous subepithelial and a few subendothelial and mesangial deposits, and extensive foot process effacement.



Source: Jennette JC, D'Agati, VD, Olson JL, Silva FG. *Heptinstall's Pathology of the Kidney*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014.

erythematosus. The term SLE is used when many organs are affected by the disease. The American College of Rheumatology established criteria for a diagnosis of SLE, which is made when a patient meets at least 4 of the 11 criteria (see *American College of Rheumatology SLE diagnostic criteria*).

In a normally functioning immune system, antibodies are produced when antigens, such as bacteria and viruses, enter the body to protect a person from infection. Antigens and antibodies join together to become what's known as an immune complex. In SLE, the immune system is overactive and produces excessive antibodies (autoantibodies or self-antibodies) that attack a person's healthy bodily tissues. Essentially, the immune system sees a person's healthy tissues as being foreign and not belonging to him or her. This causes inflammation as a way for the immune system to remove excessive antibodies from the body. The autoantibody anti-dsDNA plays a major role in the pathogenesis of lupus nephritis.

Kidney function 101

The primary purposes of the kidneys are to regulate fluid volume; excrete metabolic waste products; eliminate toxins;

and secrete the hormones renin, erythropoietin, and 1,25-dihydroxyvitamin D3 for regulation of BP, erythrocyte production, and calcium metabolism. The nephron is the functional unit of the kidney that's divided into the glomerulus and tubules.

The glomerulus is comprised of a series of blood vessels that filter blood. Formed from endothelial and mesangial cells, a series of capillaries known as a glomerular tuft begins the filtration process (see *Picturing lupus nephritis*). Mesangial cells, which are similar to monocytes, and the mesangial matrix lie between and support the capillaries. Mesangial cells have phagocytic ability (engulf and absorb bacteria) and contract to regulate glomerular capillary blood flow. The glomerular basement membrane is positioned between the endothelial cells of the renal capillaries and visceral epithelial cells of the glomeruli to filter large molecules. Some of the filtered blood in the glomeruli is reabsorbed, whereas the rest is drained into the tubules.

The tubules are responsible for reabsorption of water and substances, such as glucose and sodium, back into the systemic circulation. Following the filtration and reabsorption process, urine is secreted that contains 95% water and other substances, such as electrolytes, urea, creatinine, and uric acid.

What's going on?

The process of lupus nephritis begins when an antigen is directed against the autoantibody anti-dsDNA. The antigen and anti-dsDNA become a circulating immune complex, which is deposited into the glomeruli. An immune complex may also develop when circulating anti-dsDNA binds to an antigen that's already located in the glomeruli. This is called immune complex in situ.

The presence of anti-dsDNA is strongly associated with lupus nephritis, especially the subclasses of immunoglobulin G1 and G3. Immune complexes cause an inflammatory response in the glomeruli by activating the complement system pathway and attracting B and T lymphocytes, macrophages, and neutrophils.

Complement is a system of proteins produced by the liver to destroy bacteria and remove immune complexes from the body. An indicator of the severity of disease activity in SLE and lupus nephritis, complement is consumed during inflammation. For example, a decreasing serum complement level may correlate with a disease flare in lupus nephritis. The most commonly measured complement proteins are complement 3 (C3) and 4 (C4). The C3 nephritic factor is another autoantibody that plays a major role in the inflammatory process in lupus nephritis.

Assessment

Perform a head-to-toe assessment and health history interview for the patient with SLE suspected of developing lupus nephritis. Ask the following questions during your assessment: 1) Does the patient have any skin lesions or photosensitive rashes? 2) Does the patient have joint

American College of Rheumatology SLE diagnostic criteria

Malar rash (butterfly rash)
Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds

Discoid rash
Erythematous raised disk-like patches that scar, usually on light-exposed areas

Photosensitivity
Skin rash as a result of an unusual reaction to sunlight (newest evidence also includes indoor lighting)

Oral ulcerations
Oral or nasopharyngeal ulcerations

Nonerosive arthritis
Inflammation of two or more peripheral joints, with tenderness or swelling

Pleuritis or pericarditis
Pleuritic pain assessed by a physician; evidence of pericardial effusion and ECG changes

Renal disorder
Persistent proteinuria or abnormal sediment in the urine

Neurologic disorder
Seizures or psychoses not related to other causes

Hematologic disorder
Hemolytic anemia, leukopenia, or thrombocytopenia

Immunologic disorder
Anti-dsDNA, anti-Sm, lupus anticoagulant, or anticardiolipin antibody

Positive antinuclear antibody
An abnormal titer for antinuclear antibody by immunofluorescence

pain or stiffness? 3) Does the patient have chest pain, discomfort, or dyspnea? 4) Does the patient have pericardial or pleural friction rubs? 5) Does the patient have changes in mental status or neurologic function? 6) Does the patient have positive antibody titers, particularly to anti-dsDNA? Assessing energy level and appetite is also important.

Lab markers of systemic inflammation and renal function must also be correlated with the patient's signs and symptoms. An elevated erythrocyte sedimentation rate, an increase in C-reactive protein, a decrease in C3 and C4, and the presence of anti-dsDNA indicate active and chronic inflammation. An elevated serum creatinine level, a decrease in urine creatinine through a 24-hour urine specimen, a decrease in the serum estimated glomerular filtration rate (eGFR), and an elevated blood urea nitrogen level indicate serious renal compromise. Note that a spot urine analysis for protein is a quick and simple way to determine if the patient has proteinuria; however, the 24-hour urine specimen is the gold standard to quantify protein excretion.

The inflammatory effects of lupus nephritis cause the glomerulus to leak protein, leading to proteinuria, which occurs when a person excretes more than 150 mg of protein in a 24-hour urine specimen. Proteinuria and hypertension often coexist in lupus nephritis. The inflammatory effects of immune complexes cause damage in the glomeruli that leads to ischemia in the renal arteries. The renin-angiotensin system is stimulated, causing an increase in BP. The amount of proteinuria is often in direct proportion to the degree of hypertension due to vasoconstriction within the glomeruli.

Hypertension may be the earliest symptom of lupus nephritis even when the patient doesn't manifest other symptoms associated with SLE. In other words, damage can be done to the kidneys long before the underlying cause of the patient's hypertension is determined. Total proteinuria may or may not equate with the severity of disease activity in lupus nephritis. For example, we can't assume that lupus nephritis is more active in a patient who excretes 800 mg of protein than in a patient who excretes 450 mg.

The amount of proteinuria must be correlated with serum albumin and proteins, serum creatinine, creatinine clearance, complement level (particularly C3), the presence of urine sediment and hematuria, and a titer for anti-dsDNA (see *A comparison of two patients with lupus nephritis*).

Nephritic syndrome occurs when a patient with lupus nephritis has 3,000 mg or more of protein and blood (proteinuria and hematuria) in a 24-hour urine specimen. Nephritic syndrome is related to

A comparison of two patients with lupus nephritis

Lab test	Patient 1	Patient 2
24-hour protein (30 to 150 mg/24 hours)	800	450
Albumin (3.5 to 5.0 g/dL)	3.7	3.6
Creatinine (0.6 to 1.1 mg/dL)	1.1	1.8
Creatinine clearance (85 to 125 mL/min)	94	77
C3 (83 to 177 mg/dL)	88	74
Anti-dsDNA Negative at less than 5 international units	Negative	36

Patient 1 has significantly more proteinuria than Patient 2. However, other lab data for Patient 1 indicate normal renal function and less active lupus nephritis than in Patient 2; lupus nephritis may be in remission. Patient 1's C3 warrants close observation. The current C3 is 88, which is within normal range, but 2 months ago it was 97 and 6 months ago, 106. This indicates that complement is being consumed through the inflammatory process and an exacerbation may be unfolding.

Patient 2 has active lupus nephritis. Renal function is compromised as indicated by an abnormally high creatinine, abnormally low creatinine clearance, abnormally low C3, and a positive titer for anti-dsDNA. In the past 3 months before the current lab specimen, Patient 2 has undergone aggressive immunosuppressive therapy and shown improvement. Three months ago, Patient 2's creatinine was 3.1; creatinine clearance, 44; C3, 62; and anti-dsDNA, 80.

inflammation secondary to immune complex deposition in the glomerulus. Inflammation leads to the destruction of red blood cells that are excreted in the urine. In comparison, nephrotic syndrome leads to proteinuria but not hematuria and occurs in other pathologies involving the glomerular basement membrane and kidney podocytes. What happens when a patient loses a massive amount of protein in nephritic syndrome that causes fluid volume overload?

Albumin is the main protein in the blood that prevents leakage of body fluids into the extravascular tissues. With massive proteinuria, there's a reduction of oncotic pressure that causes fluids to move from the vascular compartment to the extracellular compartment. The accumulation of sodium and water causes edema in the hands, feet, lower legs, and eyelids. Weight gain, headache, blurred vision, and oliguria are common symptoms. Urine is often foamy and frothy due to the massive amount of protein that's present. Edema may become generalized. The patient may experience shortness of breath and jugular vein distension secondary to cardiopulmonary involvement.

Possible complications of nephritic syndrome include thrombosis secondary to a loss of clotting factors in the urine and infection due to a loss of immunoglobulins in the urine. Because of protein loss, the liver compensates and produces additional proteins, alpha-2 macroglobulin, and lipoproteins, which increases serum triglycerides and cholesterol, accelerating the development of atherosclerosis. Sodium and fluid retention in nephritic syndrome may exacerbate already existing hypertension in the patient with lupus nephritis.

Diagnosis

Considered the gold standard for diagnosing lupus nephritis, a renal biopsy may be performed to establish a treatment plan and prognosis. There isn't a clear consensus among researchers about when to perform a renal biopsy. However, general themes have emerged that suggest when a renal biopsy may be performed: acute renal failure indicated by an increasing serum creatinine level, consistent proteinuria of 500 mg or greater, hematuria in the presence of proteinuria, presence of red and/or white cell casts in the urine, and failure to respond to current therapy or an exacerbation of lupus nephritis.

The International Society of Nephrology and the Renal Pathology Society (ISN/RPS) utilizes an immunologic and histologic approach to confirming lupus nephritis through renal biopsy. The

key points

Nursing Interventions

- Perform a head-to-toe assessment. Pay close attention to any fluid volume overload symptoms, including edema, shortness of breath, and jugular vein distension.
- Assess for the onset of new lesions, rashes, or other symptoms suggesting that other organs aside from the kidneys are being affected by SLE. An immune attack may be underway that can affect lupus nephritis status.
- Assess for the presence of any pain or discomfort. For example, a patient with lupus nephritis may also have arthritis or painful skin lesions, rashes, or other symptoms of SLE that need to be addressed.
- Assess trended lab data. Correlate lab data with the patient's signs and symptoms. Teach the patient about lab values and what they mean.
- Assess the patient's responses to medication therapy. Teach the patient about the purposes and adverse reactions of medications.
- Assess the patient's vital signs, especially BP. Teach the patient to take his or her BP daily.
- Assess the patient's weight. Teach him or her to measure weight daily.
- Teach the patient to be as physically active as possible. Walking is often effective to keep joints mobile and bones strong.
- Teach the patient about the importance of proper nutrition. The amount of protein in the diet may vary according to disease severity and renal function. A low-protein diet is generally recommended when renal function is decreased in active disease to reduce fluid volume overload and maintain a therapeutic BP. Monitor the amount of calories because an increase in body weight can occur secondary to an increase in appetite from corticosteroid therapy. Body weight can also increase from fluid retention secondary to corticosteroid therapy.
- Teach the patient to limit fat intake due to hyperlipidemia.
- Teach the patient to take calcium supplements as prescribed to prevent osteoporosis.
- Teach the patient to wear sunscreen with an SPF of 30 or greater to protect the skin from indoor lighting and the sun. Too much light can trigger a skin flare in SLE and lead to systemic involvement, including the kidneys.
- Actively listen to the patient. Encourage him or her to verbalize concerns and feelings. Help the patient identify support groups in his or her local area and/or online.

pathologist evaluates the renal specimen for any structural abnormalities in the kidney(s) using light microscopy, the presence of antibodies to immunoglobulins using immunofluorescence, and deposition of immune complexes using electron microscopy (see *ISN/RPS lupus nephritis classification*).

Treatment

Treatment goals include normalizing renal function, decreasing proteinuria, preventing the progression of lupus nephritis, and minimizing medication adverse reactions. Medications are used to reduce inflammation in the kidney and control BP and fluid volume. The overall goal is to address the patient's physical and psychosocial symptoms to add quality of life.

Treatment depends on the class of lupus nephritis as determined by renal biopsy. Class I is considered mild lupus nephritis and doesn't require immunosuppressive medications. Class II is also considered mild and doesn't generally require immunosuppressive medications; however, it may require low-dose prednisone, especially when proteinuria exceeds 500 mg in a 24-hour period. Both classes require the use of renin-angiotensin system blocking agents to control BP. The long-term prognosis for Classes I and II is favorable as long as they don't progress.

Classes III and IV include lesions that have a risk of progressing to kidney function loss. Aggressive induction (short-term) and maintenance (long-term) immunosuppressive medications are warranted. There isn't consensus among researchers about when to use renin-angiotensin system blockers in Classes III and IV; however, these medications can be considered when proteinuria exceeds 500 mg in a 24-hour period and to maintain a BP no higher than 120/80.

In Class V, if the patient has stable kidney function and no proliferative lesions, then treatment with renin-angiotensin system blocking agents may be the only treatment necessary. However, in severe forms of Class V, aggressive immunosuppression is warranted. There may be an overlap of Classes III and IV with Class V.

Treatment for Class VI isn't indicated because the kidney is nonfunctional. When both kidneys are affected and nonfunctional, the patient must undergo dialysis (also known as renal replacement therapy) or renal transplantation.

Immunosuppressive medications are the mainstay of therapy for severe lupus nephritis. These medications decrease inflammation to reduce clinical and histologic activity (see *Examples of immunosuppressive agents used to manage lupus nephritis*). The adverse reactions of these medications tend to create additional challenges, especially in the prevention of infection due to immunosuppression, and physical and psychological sequelae of corticosteroid therapy.

Hypertension should be aggressively treated to maintain a BP that's appropriate for the patient's age. Loop diuretics, such as

ISN/RPS lupus nephritis classification

Classification	Description
Class I: minimal mesangial lupus nephritis	Normally functioning glomeruli; however, there are small mesangial immune complex deposits without any involvement of the peripheral glomerular capillary walls
Class II: mesangial proliferative lupus nephritis	Mesangial hypercellular activity from immune complex accumulation and expansion of mesangial matrix; doesn't compromise glomerular capillary lumens
Class III: focal lupus nephritis	Focal segmental or global endocapillary and/or extracapillary glomerulonephritis involving less than 50% of the glomerular tuft
Class IV: diffuse lupus nephritis	Diffuse or global endocapillary and/or extracapillary glomerulonephritis involving greater than 50% of the glomerular tuft
Class V: membranous lupus nephritis	Immune complex deposits in the glomerular basement membrane, with or without mesangial hyperactivity; membranous lupus nephritis may also occur at the same time that a patient meets the criteria for Class III and/or Class IV
Class VI: advanced sclerosing lupus nephritis	Greater than 90% of glomeruli globally sclerosed without residual activity

Examples of immunosuppressive agents used to manage lupus nephritis

Medication	Priority nursing implications
Corticosteroids • Prednisone and methylprednisolone: Suppress humoral immune response and leukocyte infiltration at the site of inflammation. These medications are effective in acute flares to quickly reduce inflammation. One example is to administer "pulse corticosteroids" that may include high-dose I.V. methylprednisolone daily for 3 days, followed by a maintenance dose of prednisone.	• Assess for bruising, bleeding, and infection. • Assess complete blood cell count, blood glucose levels, and cardiovascular status. • Assess daily weight and BP. • Assess mental status. Explain to the patient that feelings of uneasiness, agitation, irritability, distraction, and insomnia may occur because corticosteroids cause a decrease in serotonin and gamma-aminobutyric acid, and an increase in norepinephrine. • Teach the patient not to stop taking medication or change the dose. • Teach the patient to report a fever, eat a low-sodium diet, and measure his or her weight daily. • Teach the patient to have regular eye exams. • Teach the patient to take vitamin D supplements, as prescribed, to strengthen bones.
Antirejection agents • Azathioprine: Purine antagonist that inhibits T cell activation to induce immune system suppression to prevent organ rejection. Effective as an induction and maintenance agent to maintain remission in lupus nephritis. • Mycophenolate mofetil: Inhibits inosine monophosphate dehydrogenase to inhibit T and B cell activation to induce immune system suppression to prevent organ rejection. Effective as an induction and maintenance agent to maintain remission in lupus nephritis. • Tacrolimus: Inhibits helper T lymphocytes by selectively inhibiting interleukin-2, interleukin-3, and interferon-gamma to suppress the immune system and prevent organ rejection.	• Assess for fever, sore throat, or other signs of infection. • Assess for bruising and bleeding. • Assess complete blood cell count and liver and renal function tests. • Assess neurologic status, especially with tacrolimus. • Teach the patient to have good body and hand hygiene, and avoid anyone who has a contagious illness. • Teach the patient to wear a mask when conducting activities of daily living, especially when the white blood cell count is low. • Teach the patient the importance of influenza and pneumococcal immunization after consulting with the healthcare provider.
Antineoplastic agents • Cyclophosphamide: Alkylating agent that interferes with cell DNA and inhibits neoplastic growth. Inhibits T and B cell production, and depletes these lymphocytes to reduce inflammation. This agent is highly effective in lupus nephritis, but causes profound immunosuppression. Effective for induction of remission in combination with mycophenolate mofetil or azathioprine.	• Teach the patient taking cyclophosphamide to drink 2 to 3 quarts of water every 24 hours and void frequently. A metabolite of the drug can irritate the lining of the bladder, causing bleeding. • Profound immunosuppression occurs within 7 days of an I.V. dose of cyclophosphamide. Expect the highest point of infection risk to occur between 10 and 14 days of the dose when the white blood cell count is lowest.
Biological response modifiers • Belimumab and rituximab: Inhibit the survival of B cells, which are mediators of inflammation.	• Monitor for signs and symptoms of infection. • Monitor complete blood cell count. • Ensure that the patient is current with influenza and pneumococcal vaccines. • Instruct the patient to avoid crowds and people with infection.

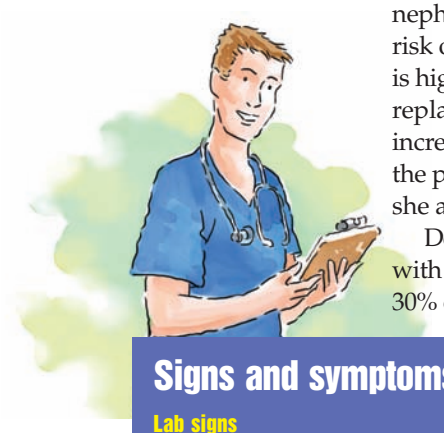
furosemide or torsemide, and thiazide diuretics, such as hydrochlorothiazide, are used to reduce edema and BP. Thiazide diuretics have an advantage over loop diuretics because they reduce calcium excretion in the urine (calciuria) and help prevent osteopenia and osteoporosis, which is a complication of long-term corticosteroid use. Cholesterol-lowering agents should also be considered secondary to nephritic syndrome and long-term use of corticosteroids.

Renal thrombosis may complicate lupus nephritis in some patients secondary to the presence of antiphospholipid antibodies. These antibodies, particularly anticardiolipin antibody, predispose the patient to a

hypercoagulable state that requires anticoagulant therapy. Antimalarial agents, such as hydroxychloroquine, have mild anticoagulant and cholesterol-lowering properties, and may also improve the patient's response to immunosuppressive therapy by reducing inflammation. Antimalarial agents are commonly used to manage inflammation in cutaneous lupus. Aspirin and nonsteroidal anti-inflammatory drugs should be avoided because they reduce renal function.

Contraception, fertility, and pregnancy should be discussed. Pregnancy during active nephritis should be discouraged due to the risk of renal failure. Morbidity and mortality is high for the mother and fetus during renal replacement therapy. The mother is at increased risk for thrombosis, including in the placenta, which can lead to fetal death if she also has antiphospholipid antibodies.

Despite advances in the care of patients with lupus nephritis, approximately 10% to 30% of patients may progress to end-stage renal disease (ESRD). Renal replacement therapy is an option in ESRD, but the process does pose significant risks, such as infection and vascular access thrombosis. Renal transplantation is the best treatment for ESRD.



Signs and symptoms cheat sheet

Lab signs

- Positive antinuclear antibody
- Positive anti-dsDNA
- High erythrocyte sedimentation rate
- High C-reactive protein
- Low C3 and C4
- Proteinuria
- Low serum albumin
- High serum triglycerides
- High serum cholesterol
- Hematuria
- Anemia
- Low eGFR
- Low creatinine clearance
- High serum creatinine

Physical symptoms

- Hypertension
- Edema
- Foamy, frothy urine
- Dark urine
- Increase in urination, especially at night
- Weight gain
- Fatigue
- Headache

A positive outlook

The prognosis for patients with lupus nephritis has evolved from a terminal condition to one in which a fairly normal quality of life can be achieved. Early diagnosis and proper identification of the disease stage according to the ISN/RPS classification are important. Aggressive therapy must be instituted as soon as possible, with the healthcare team and patient working collaboratively to manage disease symptoms and adverse reactions of medications. ■

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The author and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NME.0000521803.46775.15

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