LUPUS NEPHRITIS

Kidney Disease in Lupus, RA, and Scleroderma

© Elaine Moore

Lupus nephritis is a kidney disorder with symptoms ranging from mild hypertension to renal failure that can occur in patients with systemic lupus and related disorders.

What is Lupus Nephritis?

Lupus nephritis is a potentially serious condition of kidney inflammation that can occur as a manifestation of systemic lupus erythematosus (SLE) and occasionally rheumatoid arthritis and scleroderma. Lupus nephritis is characterized by damage to the glomeruli, the kidney’s functional units. Kidney disease in lupus typically emerges within the first five years after diagnosis, and it rarely develops in people who have had SLE for as long as 10 years. While changes in kidney tissue are seen to some degree microscopically in nearly all patients with lupus, about 50 percent of patients with SLE show evidence of lupus nephritis.

Although lupus nephritis is one of the most serious complications that can occur in SLE, improvements in the treatment of lupus have dramatically improved renal (kidney) involvement and overall survival. During the 1950’s patients with lupus nephritis had a very poor prognosis. Currently, the survival rates for lupus nephritis are documented to be as high as 85 percent at 5 years and 73 percent at 10 years.

Who is Affected?

Systemic lupus erythematosus is more common in African-Americans, African-Caribbeans, and Asians, which accounts for a higher prevalence of lupus nephritis in these ethnic groups. However, although SLE is more common in women, men with SLE have an increased prevalence of kidney disease with a worse prognosis. The primary age affected is 20-40 years.

Symptoms

Symptoms of lupus nephritis range from the presence of protein in urine (proteinuria) and mild hypertension (elevated blood pressure) to acute or chronic renal failure. Signs and symptoms of lupus nephritis include blood-tinged urine, weight gain, proteinuria, foamy urine, fatigue, fever, fluid retention (edema), hypertension, and swelling around the eyes, legs, ankles and fingers. Swelling is usually absent in the morning and increases as the day progresses.

Depending on the clinical symptoms and the number of glomeruli damaged (as seen in tissue biopsy studies), patients with lupus nephritis may have conditions of interstitial
nephritis, mesangial glomerulonephritis, membranous glomerulonephritis, diffuse proliferative glomerulonephritis, focal proliferative glomerulonephritis or chronic glomerulosclerosis ranging from Class I through Class VI. An activity index is also used along with the disease classification to determine disease prognosis and treatment response.

**Morbidity**

Renal failure and mortality in patients with lupus nephritis are related to kidney disease as well as to treatment-related complications including heart disease and blood clotting disturbances. Renal failure can cause anemia, uremia, hypertension, metabolic disturbances, and it may lead to edema, ascites (abdominal fluid excess), elevated lipid levels, and stroke.

**Diagnosis**

Lupus nephritis is diagnosed in lupus patients with either histological (tissue studies) or clinical signs of kidney disease. Patients with lupus nephritis usually have high titers of double-stranded DNA antibodies and low levels of complement, especially C3. Imaging tests such as ultrasound and intravenous pyelogram are used to detect kidney damage.

Clinical signs of active lupus nephritis include the presence of protein, white blood cells, red blood cells, and casts in the urinalysis exam. Blood tests for albumin are usually low, whereas blood tests for blood urea nitrogen (BUN), creatinine, phosphorus, and cholesterol are usually elevated. Renal biopsy is useful in evaluating the severity of kidney disease and it is helpful in determining optimal treatment.

**Causes**

Lupus nephritis occurs in people with certain immune system genes and risk factors. Autoimmunity, primarily immune complexes consisting of linked antigens and antibodies and complement, are responsible for lupus nephritis. These lattice-like complexes lodge into kidney tissue causing an inflammatory response and activating the complement cascade, which involves the production of immune system chemicals.

**Treatment**

Treatment consists of immunosuppressant agents such as cyclophosphamide, azathioprine and cyclosporine. Glucocorticoid steroids are often used, but because of associated toxicity, they are not recommend for long-term use. Depending on the results of the renal biopsy, glucocorticoid steroids may be used on alternate days or as pulses of intravenous medication.

Patients with antiphospholipid syndrome, who are at risk for glomerular thrombosis, or who have autoimmune hemolytic anemia or thrombocytopenia (condition of low platelets) are often treated with plasmapheresis (removal of blood plasma and
replacement with transfusions of donor plasma) to remove circulating immune complexes. Intravenous immunoglobulin therapy is sometimes used although its expense and potential side effects cause its use to be limited.

Dialysis may be necessary to control symptoms of acute or chronic renal failure. The 5-year prognosis for patients requiring dialysis is greater than 80 percent. In cases of severe disease, a kidney transplant may be required although transplants are not recommended or patients with active lupus.

**Diet and Lifestyle**

Dietary recommendations include limiting protein, sodium, dietary fats, and potassium. Drugs that affect kidney function, particularly non-steroidal anti-inflammatory drugs such as ibuprofen should also be avoided. Statins are often recommended to reduce blood limits and to prevent nephritic syndrome. Calcium supplements are recommended for preventing osteoporosis in patients using glucocorticoid steroids.

**Resources:**

H. Michael Belmont, Lupus Clinical Overview, New York University Medical Center, Hospital for Joint Diseases, accessed March 1, 2007.


The copyright of the article Lupus Nephritis is owned by Elaine Moore. Permission to republish Lupus Nephritis in print or online must be granted by the author in writing.