

variable and includes proteinuria, microscopic hematuria, acute renal failure, and hypertension. Serum complement levels are low, and there may be elevated levels of C-reactive proteins, rheumatoid factor, antinuclear antibodies, and cryoglobulins. Renal lesions include MPGN, diffuse proliferative and exudative glomerulonephritis (DPGN), or mesangioproliferative glomerulonephritis, sometimes leading to RPGN. Treatment focuses on eradicating the infection, with most patients treated as if they have endocarditis. The prognosis is guarded.

■ LUPUS NEPHRITIS

Lupus nephritis is a common and serious complication of systemic lupus erythematosus (SLE). Clinical manifestations of renal disease are present in 30% of patients at the time of diagnosis, and the majority will develop renal abnormalities in the course of their disease; it is more common in blacks, Asians, and Hispanics than it is in whites. Lupus nephritis results from the deposition of circulating immune complexes composed of primarily DNA and anti-DNA, which activate the complement cascade, leading to complement-mediated damage, leukocyte infiltration, activation of procoagulant factors, and release of various cytokines. In situ immune complex formation also plays a role in renal injury. These immune deposits may occur in the mesangial, subendothelial, and/or subepithelial spaces.

The clinical manifestations, course of disease, and treatment of lupus nephritis are closely linked to renal pathology. The most common clinical sign of renal disease is proteinuria, but hematuria, hypertension, varying degrees of renal failure, and active urine sediment with red blood cell casts can all be present. Anti-dsDNA antibodies that fix complement correlate best with the presence of renal disease. Hypocomplementemia is common in patients with acute lupus nephritis (70–90%), and declining complement levels may herald a flare. A kidney biopsy should be performed in most patients with renal involvement to establish the histologic subtype, which guides therapy.

The World Health Organization (WHO) workshop in 1974 first outlined several distinct patterns of lupus-related glomerular injury, and this classification was modified in 2004. This latest version of