Many glomerular diseases may be associated with acute and chronic infectious diseases of bacterial, viral, fungal, or parasitic origin. In many instances, the glomerular activators are transient and of little clinical consequence. In other instances, distinct clinical syndromes such as acute nephritis or nephrotic syndrome may be provoked. Some of the more important infection-related glomerular diseases are illustrated here. Others diseases, including human immunodeficiency virus and hepatitis, are also discussed in Volume IV.
Light, immunofluorescent, and electron microscopy of poststreptococcal (postinfectious) glomerulonephritis. Glomerulonephritis may follow in the wake of cutaneous or pharyngeal infection with a limited number of "nephritogenic" serotypes of group A β-hemolytic streptococcus. Typically, patients with glomerulonephritis exhibit hematuria, edema, proteinuria, and hypertension. Renal function frequently is depressed, sometimes severely. Most patients recover spontaneously, and a few go on to rapidly progressive or chronic indolent disease. A, On light microscopy the glomeruli are enlarged and hypercellular, with numerous leukocytes in the capillary lumina and a variable increase in mesangial cellularity. The leukocytes are neutrophils and monocytes. The capillary walls are single-contoured, and crescents may be present. B, On immunofluorescence, granular capillary wall and mesangial deposits of immunoglobulin G and complement C3 are observed (starry-sky pattern). Three predominant patterns occur depending on the location of the deposits; these include garlandlike, mesangial, and starry-sky patterns. C, The ultrastructural findings are those of electron-dense deposits, characteristically but not solely in the subepithelial aspects of the capillary walls, in the form of large gumdrop or hump-shaped deposits (arrow). However, electron-dense deposits also are found in the mesangial regions and occasionally subendothelial locations. Endothelial cells often are swollen, and leukocytes are not only found in the capillary lumina but occasionally in direct contact with basement membranes in capillary walls with deposits. Similar findings may be observed in glomerulonephritis after infectious diseases other than certain strains of Streptococci.
Infection-Associated Glomerulopathies

**FIGURE 4-2**

Infective endocarditis and shunt nephritis. The glomerulonephritis accompanying infective endocarditis or infected ventriculocavitary shunts or other indwelling devices is that of a postinfectious glomerulonephritis or membranoproliferative glomerulonephritis type I pattern, or both (see Fig. 2-18). In reality, the changes often are a combination of both. As shown here, this glomerulopathy is characterized by increased mesangial cellularity, with slight lobular architecture; occasionally thickened capillary walls, with double contours (arrow); and leukocytes in some capillary lumina. This glomerulus also has a small crescent.

**FIGURE 4-3** (see Color Plate)

Human immunodeficiency virus (HIV) infection. Many forms of renal disease have been described in patients infected with HIV. Various immune complex-mediated glomerulonephritides associated with complicating infections are known; however, several disorders appear to be directly or indirectly related to HIV itself. Perhaps the more common of these is known as HIV-associated nephropathy (HIVAN). This disease is a form of the collapsing (focal segmental) glomerulosclerosis with significant tubular and interstitial abnormalities. **A**, In HIVAN, many visceral epithelial cells are enlarged, coarsely vacuolated, contain protein reabsorption droplets, and overlay capillaries with varying degrees of wrinkling and collapse of the walls (arrows). **B**, In HIVAN, the tubules are dilated and filled with a precipitate of plasma protein, and the tubular epithelial cells display various degenerative features (arrow). Ultrastructural findings are a combination of those expected for the glomerulopathy as well as those common to HIV infection. Thus, the foot processes of visceral epithelial cells are effaced and often detached from the capillary basement membranes. **C**, Common in HIV infection are tubuloreticular structures, modifications of the cytoplasm of endothelial cells in which clusters of microtubular arrays are in many cells (arrow). Some evidence suggests that HIV or viral proteins localize in renal epithelial cells and perhaps are directly or indirectly responsible for the cellular and functional damage. HIVAN often has a rapidly progressive downhill course, culminating in end-stage renal disease in as few as 4 months. HIVAN has a striking racial predilection; over 90% of patients are black.

The other glomerulopathy that may be an integral feature of HIV infection is immunoglobulin A nephropathy. In this setting, HIV antigen may be part of the glomerular immune complexes and circulating immune complexes. The morphology and clinical course generally are the same as in immunoglobulin A nephropathy occurring in the non-HIV setting.