

hematuria. A high prevalence of many of these infectious diseases in undeveloped countries results in infection-associated renal disease being the most common cause of glomerulonephritis in many parts of the world.

**Poststreptococcal Glomerulonephritis** This form of glomerulonephritis is one of the classic complications of streptococcal infection. The discussion of this disease can be found earlier, in the section “Acute Nephritic Syndromes.”

**Subacute Bacterial Endocarditis** Renal injury from persistent bacteremia absent the continued presence of a foreign body, regardless of cause, is treated presumptively as if the patient has endocarditis. The discussion of this disease can be found earlier, in the section “Acute Nephritic Syndromes.”

**Human Immunodeficiency Virus** Renal disease is an important complication of HIV disease. The risk of development of end-stage renal disease is much higher in HIV-infected African Americans than in HIV-infected whites. About 50% of HIV-infected patients with kidney disease have HIV-associated nephropathy (HIVAN) on biopsy. The lesion in HIVAN is FSGS, characteristically revealing a collapsing glomerulopathy (see Fig. 62e-3) with visceral epithelial cell swelling, microcystic dilatation of renal tubules, and tubuloreticular inclusion. Renal epithelial cells express replicating HIV virus, but host immune responses also play a role in the pathogenesis. MPGN and DPGN have also been reported but more commonly in HIV-infected whites and in patients coinfecting with hepatitis B or C. HIV-associated TTP has also been reported. Other renal lesions include DPGN, IgA nephropathy, and MCD. Renal biopsy may be indicated to distinguish between these lesions.

HIV patients with FSGS typically present with nephrotic-range proteinuria and hypoalbuminemia, but unlike patients with other etiologies for nephrotic syndrome, they do not commonly have hypertension, edema, or hyperlipidemia. Renal ultrasound also reveals large, echogenic kidneys despite the finding that renal function in some patients declines rapidly. Treatment with inhibitors of the renin-angiotensin system decreases the proteinuria. Effective antiretroviral therapy benefits both the patient and the kidney and improves survival of HIV-infected patients with chronic kidney disease (CKD) or end-stage renal disease. In HIV-infected patients not yet on therapy, the presence of HIVAN is an indication to initiate therapy. Following the introduction of antiretroviral therapy, survival on dialysis for the HIV-infected patient has improved dramatically. Renal transplantations in HIV-infected patients without detectable viral loads or histories of opportunistic infections provide a better survival benefit over dialysis. Following transplantation, patient and graft survival are similar to the general transplant population despite frequent rejections.

**Hepatitis B and C** Typically infected patients present with microscopic hematuria, nonnephrotic or nephrotic-range proteinuria, and hypertension. There is a close association between hepatitis B infection and polyarteritis nodosa with vasculitis appearing generally in the first 6 months following infection. Renal manifestations include renal artery aneurysms, renal infarction, and ischemic scars. Alternatively, the hepatitis B carrier state can produce a MGN that is more common in children than adults, or MPGN that is more common in adults than in children. Renal histology is indistinguishable from idiopathic MGN or type I MPGN. Viral antigens are found in the renal deposits. There are no good treatment guidelines, but interferon  $\alpha$ -2b and lamivudine have been used to some effect in small studies. Children have a good prognosis, with 60–65% achieving spontaneous remission within 4 years. In contrast, 30% of adults have renal insufficiency and 10% have renal failure 5 years after diagnosis.

Up to 30% of patients with chronic hepatitis C infection have some renal manifestations. Patients often present with type II mixed cryoglobulinemia, nephrotic syndrome, microscopic hematuria, abnormal liver function tests, depressed C3 levels, anti-hepatitis C virus (HCV) antibodies, and viral RNA in the blood. The renal lesions most commonly seen, in order of decreasing frequency, are *cryoglobulinemic glomerulonephritis*, MGN, and *type I MPGN*. Treatment with pegylated interferon and ribavirin is typical to reduce the viral load.

**Other Viruses** Other viral infections are occasionally associated with glomerular lesions, but cause and effect are not well established. These viral infections and their respective glomerular lesions include: cytomegalovirus producing MPGN; influenza and anti-GBM disease; measles-associated endocapillary proliferative glomerulonephritis, with measles antigen in the capillary loops and mesangium; parvovirus causing mild proliferative or mesangioproliferative glomerulonephritis or FSGS; mumps and mesangioproliferative glomerulonephritis; Epstein-Barr virus producing MPGN, diffuse proliferative nephritis, or IgA nephropathy; dengue hemorrhagic fever causing endocapillary proliferative glomerulonephritis; and coxsackievirus producing *focal glomerulonephritis* or DPGN.

**Syphilis** Secondary syphilis, with rash and constitutional symptoms, develops weeks to months after the chancre first appears and occasionally presents with the nephrotic syndrome from MGN caused by subepithelial immune deposits containing treponemal antigens. Other lesions have also rarely been described including interstitial syphilitic nephritis. The diagnosis is confirmed with nontreponemal and treponemal tests for *Treponema pallidum*. The renal lesion responds to treatment with penicillin or an alternative drug, if allergic. Additional testing for other sexually transmitted diseases is an important part of disease management.

**Leprosy** Despite aggressive eradication programs, approximately 400,000 new cases of leprosy appear annually worldwide. The diagnosis is best made in patients with multiple skin lesions accompanied by sensory loss in affected areas, using skin smears showing paucibacillary or multibacillary infection (WHO criteria). Leprosy is caused by infection with *Mycobacterium leprae* and can be classified by Ridley-Jopling criteria into various types: tuberculoid, borderline tuberculoid, mid-borderline and borderline lepromatous, and lepromatous. Renal involvement in leprosy is related to the quantity of bacilli in the body, and the kidney is one of the target organs during splanchnic localization. In some series, all cases with borderline lepromatous and lepromatous types of leprosy have various forms of renal involvement including FSGS, mesangioproliferative glomerulonephritis, or renal amyloidosis; much less common are the renal lesions of DPGN and MPGN. Treatment of the infection can cause remission of the renal disease.

**Malaria** There are 300–500 million incident cases of malaria each year worldwide, and the kidney is commonly involved. Glomerulonephritis is due to immune complexes containing malarial antigens that are implanted in the glomerulus. In malaria from *P. falciparum*, mild proteinuria is associated with subendothelial deposits, mesangial deposits, and mesangioproliferative glomerulonephritis that usually resolve with treatment. In quartan malaria from infection with *Plasmodium malariae*, children are more commonly affected and renal involvement is more severe. Transient proteinuria and microscopic hematuria can resolve with treatment of the infection. However, resistant nephrotic syndrome with progression to renal failure over 3–5 years does happen, as <50% of patients respond to steroid therapy. Affected patients with nephrotic syndrome have thickening of the glomerular capillary walls, with subendothelial deposits of IgG, IgM, and C<sub>3</sub> associated with a sparse membranoproliferative lesion. The rare mesangioproliferative glomerulonephritis reported with *Plasmodium vivax* or *Plasmodium ovale* typically has a benign course.

**Schistosomiasis** Schistosomiasis affects more than 300 million people worldwide and primarily involves the urinary and gastrointestinal tracts. Glomerular involvement varies with the specific strain of schistosomiasis; *Schistosoma mansoni* is most commonly associated with clinical renal disease, and the glomerular lesions can be classified: Class I is a *mesangioproliferative glomerulonephritis*; class II is an *extracapillary proliferative glomerulonephritis*; class III is a *membranoproliferative glomerulonephritis*; class IV is a *focal segmental glomerulonephritis*; and class V is *amyloidosis*. Classes I–II often remit with treatment of the infection, but classes III and IV lesions are associated with IgA immune deposits and progress despite antiparasitic and/or immunosuppressive therapy.

**Other Parasites** Renal involvement with toxoplasmosis infections is rare. When it occurs, patients present with nephrotic syndrome