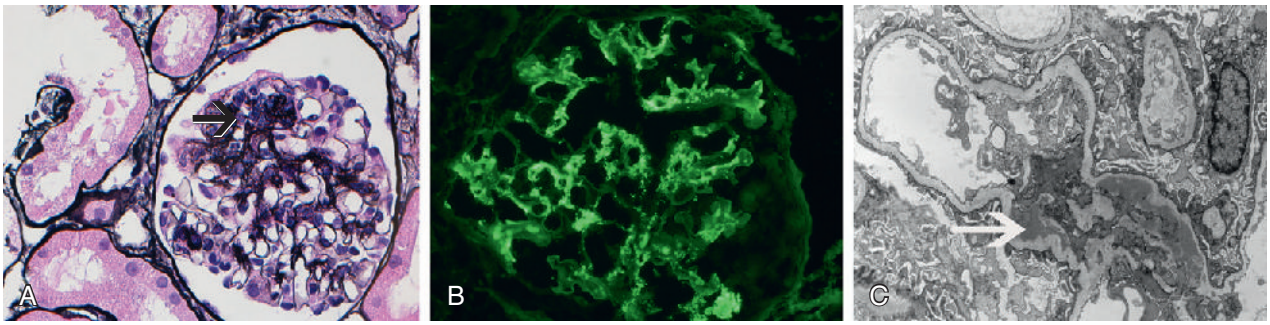

FIGURE 28-6 Immunoglobulin A (IgA) nephropathy. **A,** Light microscopy shows mesangial hypercellularity *(black arrow)* (silver methenamine, ×40). **B,** Immunofluorescence microscopy shows bright mesangial IgA staining. **C,** Electron microscopy shows large mesangial electron-dense deposits *(arrow)* (×7860)

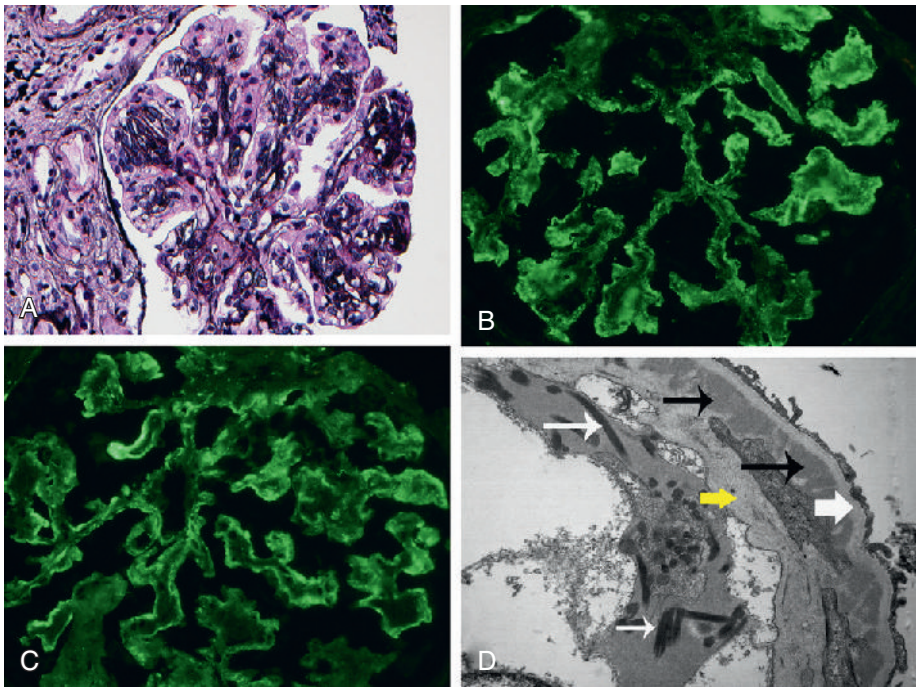

FIGURE 28-7 Immune complex–mediated membranoproliferative glomerulonephritis due to hepatitis C virus infection. **A,** Light microscopy shows a membranoproliferative pattern of injury with mesangial expansion, endocapillary proliferation, double-contour formation along the capillary walls, and lobular accentuation of the glomerular tufts (silver methenamine, ×40). **B** and **C,** Immunofluorescence microscopy shows bright capillary wall staining for immunoglobulin M (**B,** ×40) and for C3 (**C,** ×40). **D,** Electron microscopy shows capillary wall thickening and a double-contour formation due to accumulation of subendothelial electron-dense deposits *(black arrows)*, cellular elements, and new basement membrane formation (i.e., duplication) *(yellow arrow)* that produces the double contour. The *thick white arrow* indicates the old basement membrane, and fibrin tactoids *(white arrows)* in glomerular capillary loops indicate a prothrombotic state (×1350).

In patients with normal renal function, treatment is supportive only. Patients with persistent proteinuria >1 g/24h and/or progressive renal failure should be considered for treatment with high-dose corticosteroids with or without cytotoxic medication.

Membranoproliferative Glomerulonephritis

Membranoproliferative glomerulonephritis (MPGN) is a pattern of glomerular injury resulting from predominantly subendothelial and mesangial deposition of immune complexes or complement factors and their products. On light microscopy, mesangial hypercellularity, endocapillary proliferation, and capillary wall remodeling with double-contour formation are characteristic, and they result in a lobular accentuation of the glomerular tufts. Immunofluorescence microscopy shows immunoglobulins or complement factors, depending on the underlying cause of MPGN. Electron microscopy typically shows mesangial and subendothelial deposits, and, less commonly, intramembranous and subepithelial deposits (Fig. 28-7).

Based on a recent proposal, MPGN can be classified as immune complex mediated or complement mediated. Immune complex–mediated MPGN shows immunoglobulin and complement factors on immunofluorescence microscopy. Complement-mediated MPGN shows complement factors and a lack of significant immunoglobulin on immunofluorescence microscopy (Fig. 28-8). Immune complex–mediated MPGN results from chronic infections, autoimmune diseases, and monoclonal gammopathies. Complement-mediated MPGN is caused by genetic