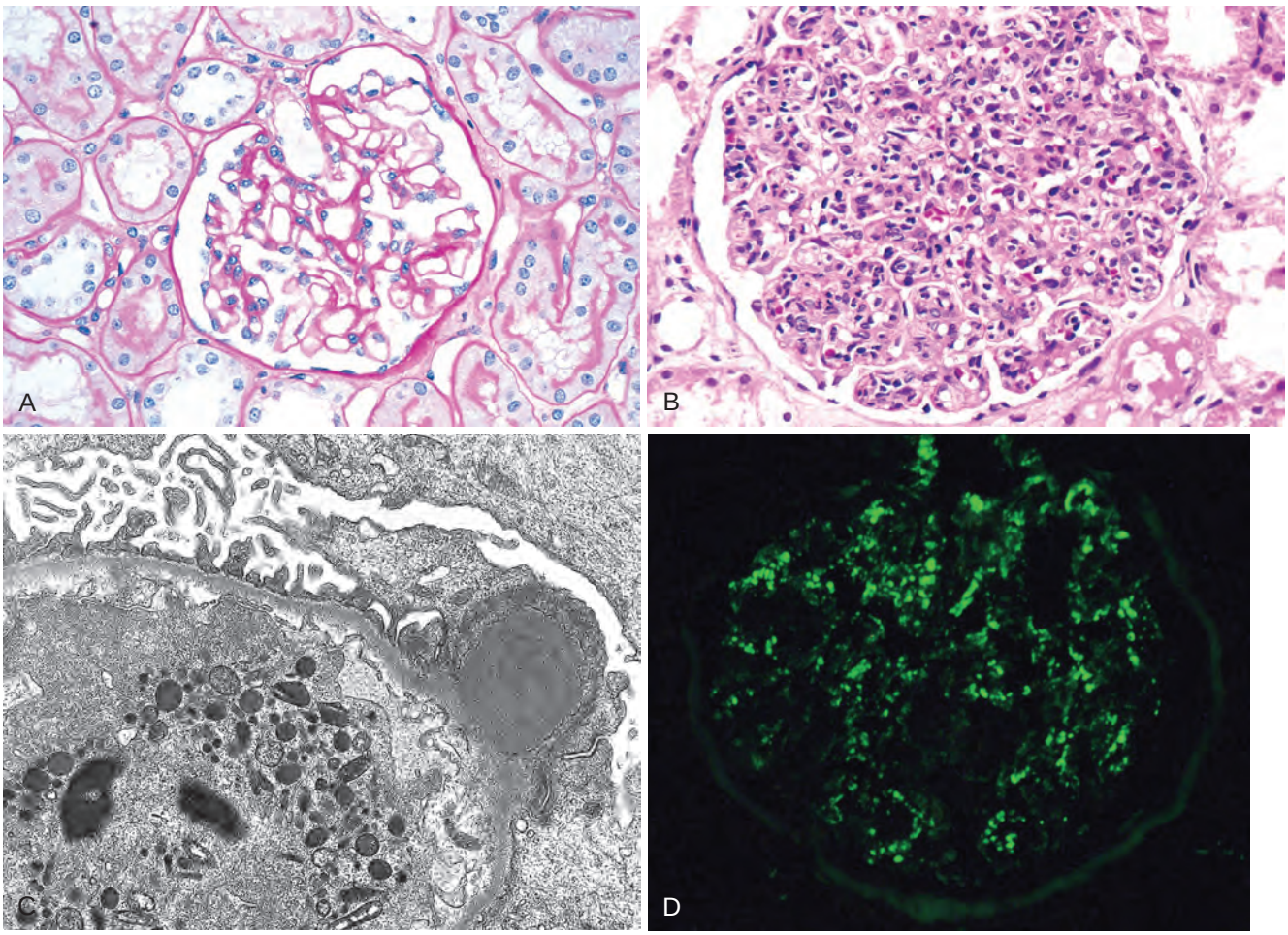


**Figure 20-9** Acute proliferative glomerulonephritis. **A,** Normal glomerulus. **B,** Glomerular hypercellularity is due to intracapillary leukocytes and proliferation of intrinsic glomerular cells. **C,** Typical electron-dense subepithelial "hump" and a neutrophil in the lumen. **D,** Immunofluorescent stain demonstrates discrete, coarsely granular deposits of complement protein C3 (stain for IgG was similar), corresponding to "humps" illustrated in part **C.** (**A-C,** Courtesy Dr. H. Rennke, Brigham and Women's Hospital, Boston, Mass. **D,** Courtesy D. J. Kowaleska, Cedars-Sinai Medical Center, Los Angeles, Calif.)

interstitial edema and inflammation, and the tubules often contain red cell casts.

By **immunofluorescence microscopy,** there are granular deposits of IgG, and C3, and sometimes IgM in the mesangium and along the GBM (Fig. 20-9*D*). Although immune complex deposits are almost universally present, they are often focal and sparse. The characteristic **electron microscopic findings** are discrete, amorphous, electron-dense deposits on the epithelial side of the membrane, often having the appearance of "humps" (Fig. 20-9*C*), presumably representing the antigen-antibody complexes at the subepithelial cell surface. Subendothelial deposits are also commonly seen, typically early in the disease course, and mesangial and intramembranous deposits may be present.

***Clinical Course.*** In the typical case, a young child abruptly develops malaise, fever, nausea, oliguria, and hematuria (smoky or cola-colored urine) 1 to 2 weeks after recovery from a sore throat. The patients have dysmorphic red cells or red cell casts in the urine, mild proteinuria (usually less than 1 gm/day), periorbital edema, and mild to moderate hypertension. In adults the onset is more likely to be atypical, such as the sudden appearance of hypertension or edema, frequently with elevation of BUN. The glomerulonephritis is subclinical in some infected individuals, and is discovered only on screening for microscopic hematuria carried out during epidemic outbreaks. Important laboratory findings include elevations of antistreptococcal antibody titers and a decline in the serum concentration of C3 and other components of the complement cascade.

More than 95% of affected children eventually recover renal function with conservative therapy aimed at maintaining sodium and water balance. A small minority of children (perhaps fewer than 1%) do not improve, become severely oliguric, and develop a rapidly progressive form of glomerulonephritis (described later). Some of the remaining patients may undergo slow progression to chronic glomerulonephritis with or without recurrence of an active nephritic picture. Prolonged and persistent heavy proteinuria and abnormal GFR mark patients with an unfavorable prognosis.

In adults the disease is less benign. Although the overall prognosis in epidemics is good, in only about 60% of sporadic cases do the patients recover promptly. In the remainder the glomerular lesions fail to resolve quickly, as manifested by persistent proteinuria, hematuria, and hypertension. In some of these patients, the lesions