

FIGURE 9-15 (see Color Plate)

Fine-needle aspirate from patient with intrarenal cytomegalovirus (CMV) infection. **A**, There are activated and transformed lymphocytes with immature nuclear chromatin and abundant blue cytoplasm that infiltrate the kidney in response to the infection; large granular lymphocytes (NK cells) may be seen as well, but few neutrophils. Similar activated lymphocytes, NK cells, and atypical monocytes can be observed within the peripheral blood. The tubule epithelial cells are virtually never seen to contain CMV inclusions in aspirate material, in contrast to core biopsy specimens. All intrarenal

viral infections have a similar appearance, and immunostaining or in situ hybridization is required to identify specific viruses (May-Grunwald Giemsa, original magnification $\times 80$). **B**, Tubular epithelial cells stained with antibody to CMV immediate and early nuclear proteins in active intrarenal CMV infection. With an immunalkaline phosphatase method, cytoplasmic and prominent nuclear staining for these early proteins are observed in the tubular epithelium. In very early infection, neutrophils also may have cytoplasmic staining for these proteins (original magnification $\times 240$).

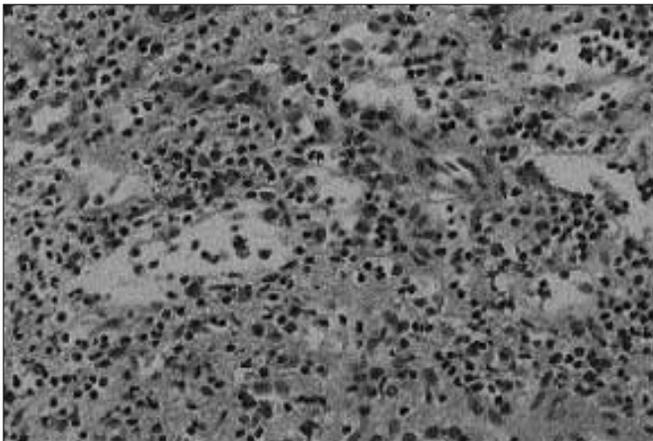


FIGURE 9-16 (see Color Plate)

Numerous eosinophils in an interstitial inflammatory infiltrate. Eosinophils may be diffuse within the infiltrate, but may also be clustered, forming "eosinophilic abscesses," as in this area (hematoxylin and eosin, original magnification $\times 400$). Eosinophils may also be demonstrated in the urine sediment. Drugs most commonly producing acute interstitial nephritis as part of a hypersensitivity reaction include: penicillins, sulfonamides, and nonsteroidal anti-inflammatory drugs [6]. The patient had recently undergone a course of therapy with methicillin. The interstitial nephritis may be part of a systemic reaction which includes fever, rash, and eosinophilia.

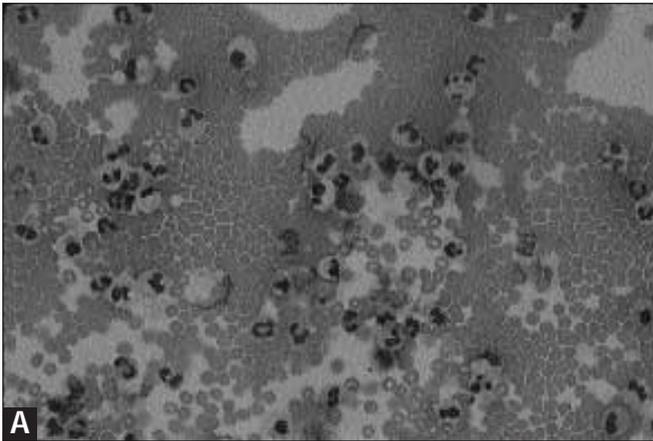
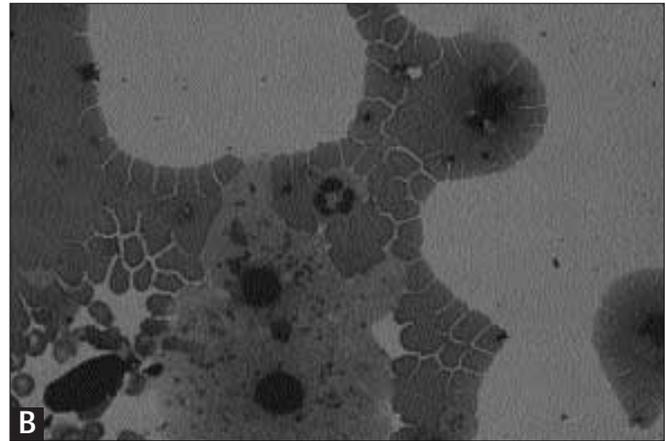


FIGURE 9-17 (see Color Plate)

Fine-needle aspirate of acute allergic interstitial nephritis. **A**, The aspirate contains numerous lymphocytes, occasional activated lymphocytes, and eosinophils without fully transformed lymphocytes, corresponding to the inflammatory component within the tubulointerstitium observed on routine renal biopsy. Monocytes often are



present (May-Grunwald Giemsa, original magnification $\times 80$). **B**, Higher magnification showing the typical infiltrating cells, including a monocyte, activated lymphocyte, and an eosinophil. A neutrophil is present, likely owing to blood contamination (May-Grunwald Giemsa, original magnification $\times 160$).

Tubular Diseases

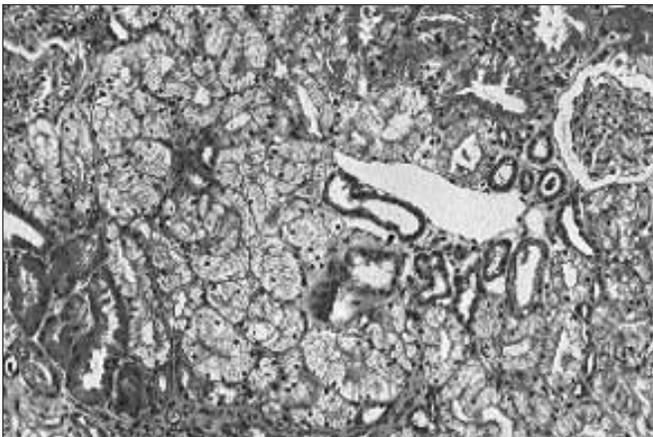


FIGURE 9-18 (see Color Plate)

Severe vacuolization of tubular cells in injured tubular epithelium (hematoxylin and eosin, original magnification $\times 400$). The vacuoles reflect cell injury and derangement of homeostatic mechanisms that maintain the normal intracellular milieu. In this case, the vacuoles developed on exposure to intravenous immunoglobulin in a sucrose vehicle; the morphology is reminiscent of the severe changes produced by osmotic agents. While generally a nonspecific marker of cell injury, a distinctive pattern of “isometric” vacuolization, in which there are numerous intracellular vacuoles of uniform size (not shown here) is very typical of cyclosporine/FK506 effect [6].

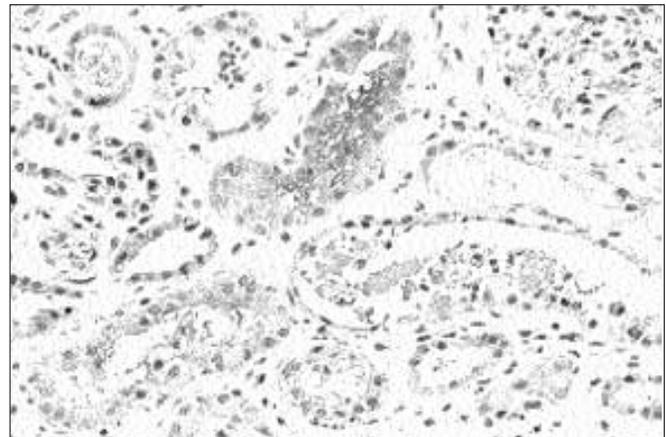
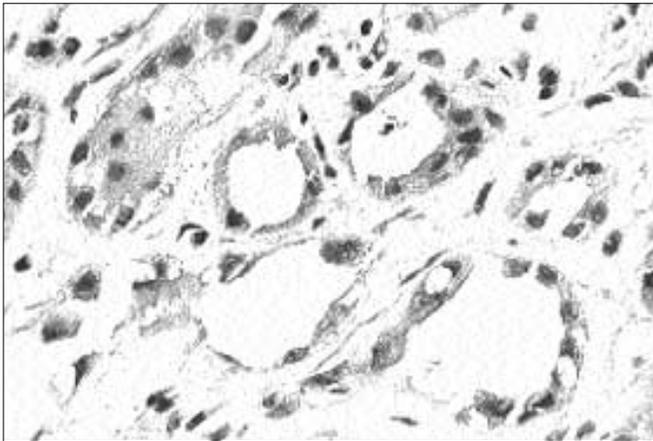
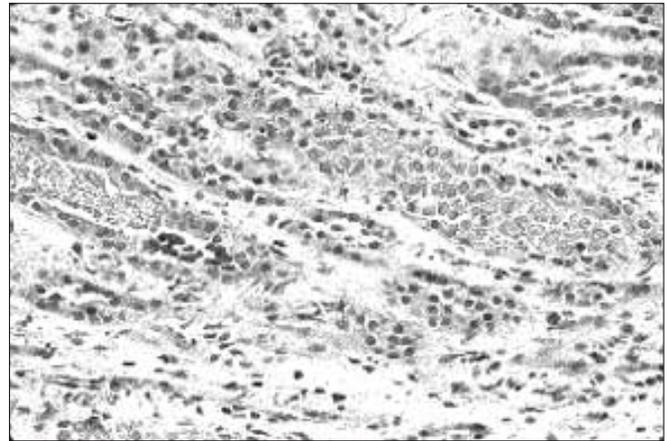


FIGURE 9-19 (see Color Plate)

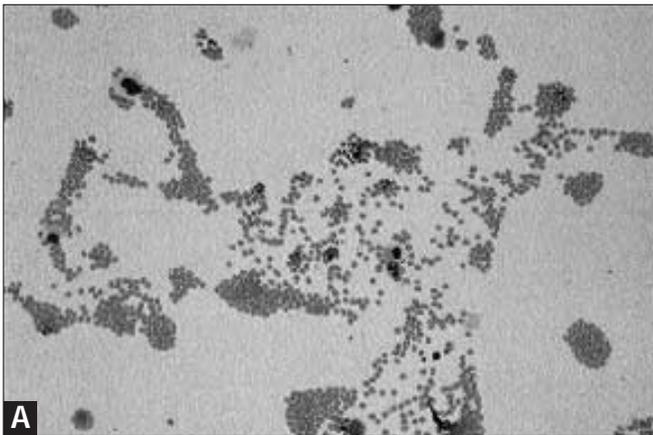
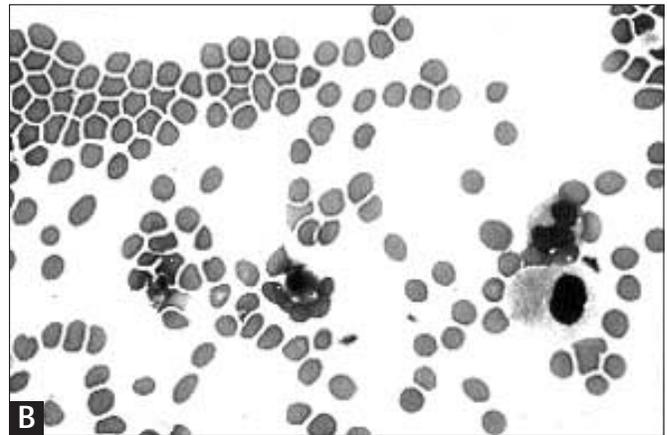
Necrotic tubular cells and cell debris in tubular lumina. One tubule shows extensive cell loss, with tubular epithelium lined only by a very flattened layer of cytoplasm. The dilated lumen contains numerous necrotic tubular cells with pyknotic nuclei. Several tubules contain cell debris and one contains red blood cells (hematoxylin and eosin, original magnification $\times 250$). Such changes are more often seen with toxic than with ischemic injury [6], unless the latter is very severe.

**FIGURE 9-20** (see Color Plate)

This micrograph shows sites of cell exfoliation, attenuation of remaining cells, and reactive and regenerative changes (hematoxylin and eosin, original magnification $\times 400$). Exfoliation occurs with disruption of cell-cell and cell-substrate adhesion, and may involve viable as well as non-viable cells [7]. Reactive and regenerative changes may include basophilia of cell cytoplasm, increased nuclear:cytoplasmic ratio, heterogeneity of nuclear size and appearance, hyperchromatic nuclei and mitotic figures.

**FIGURE 9-21** (see Color Plate)

Outer medulla shows in situ cell necrosis and loss in medullary thick ascending limb (hematoxylin and eosin, original magnification $\times 250$). Tubules contain cells and cell debris. Changes reflect ischemic injury. Impaction of cells and cast material may lead to tubular obstruction, especially in narrow regions of the nephron. Adhesion molecules on the surface of exfoliated cells may contribute to aggregation of cells within the tubule and adhesion of detached cells to in situ tubular cells [8].

**A****B****FIGURE 9-22** (see Color Plate)

Fine-needle aspirate showing acute tubular cell injury and necrosis. **A**, The aspirate shows scattered tubular epithelial cells with swelling and focal degenerative changes, and a minimal associated inflammatory infiltrate. There is no significant background cell debris (May-Grunwald Giemsa, original magnification $\times 40$). **B**, One tubular cell is degenerated with reduction in cell size, condensed gray-blue

cytoplasm, and a pyknotic nucleus. Another cell has more advanced necrosis with additional cytoplasmic disruption and a very small pyknotic nucleus. Compare the adjacent swollen damaged tubular cell which has not yet undergone necrosis (May-Grunwald Giemsa, original magnification $\times 160$).