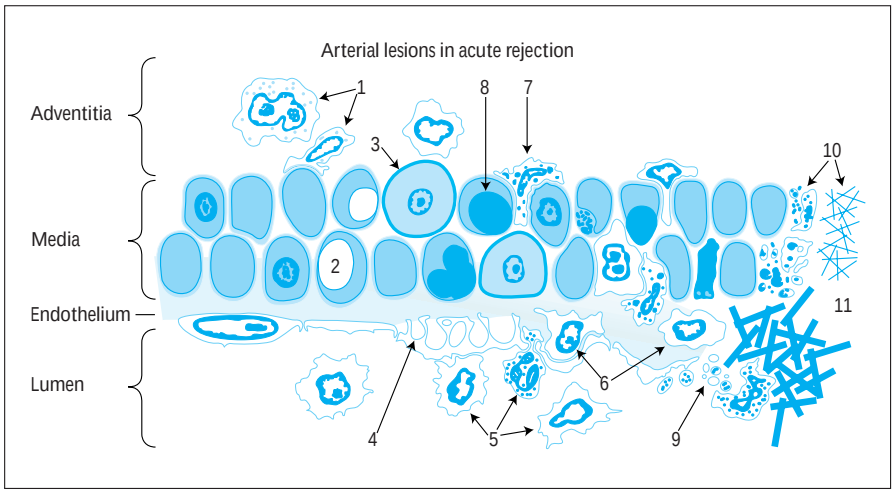

FIGURE 10-8
Diagram of arterial lesions of acute rejection. The initial changes (1–5) before intimal arteritis (6) occurs are completely nonspecific. These early changes are probably mechanistically related to the diagnostic lesions but can occur as a completely self-limiting phenomenon unrelated to clinical rejection. Lesions 7 to 10 are those characteristic of "transmural" rejection. Lesion 1 is perivascular inflammation; lesion 2, myocyte vacuolization; lesion 3, apoptosis; lesion 4, endothelial activation and prominence; lesion 5, leukocyte adherence to the endothelium; lesion 6 (specific), penetration of inflammatory cells under the endothelium (intimal arteritis); lesion 7, inflammatory cell penetration of the media; lesion 8, necrosis of medial smooth muscle cells; lesion 9, platelet aggregation; lesion 10, fibrinoid change; and lesion 11 is thrombosis.

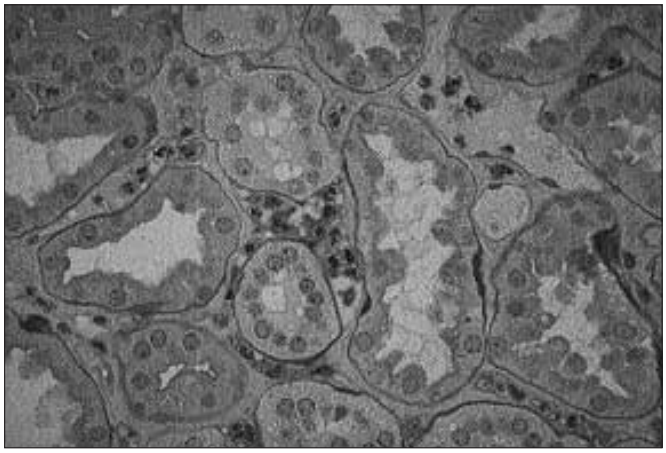

FIGURE 10-9 (see Color Plate)
Antibody-mediated rejection with aggregates of polymorphonuclear leukocytes (polymorphs) in peritubular capillaries. This lesion is a feature of both classic hyperacute rejection and of later appearing antibody-mediated rejection, which is by far the more common entity. Antibody- and cell-mediated rejection can coexist, so one may find both tubulitis and intimal arteritis along with this lesion; however many cases of antibody-mediated rejection have a paucity of tubulitis [2]. The polymorph aggregates can be subtle, another reason for looking with care at the biopsy that appears to show "nothing."

Acute Tubular Necrosis

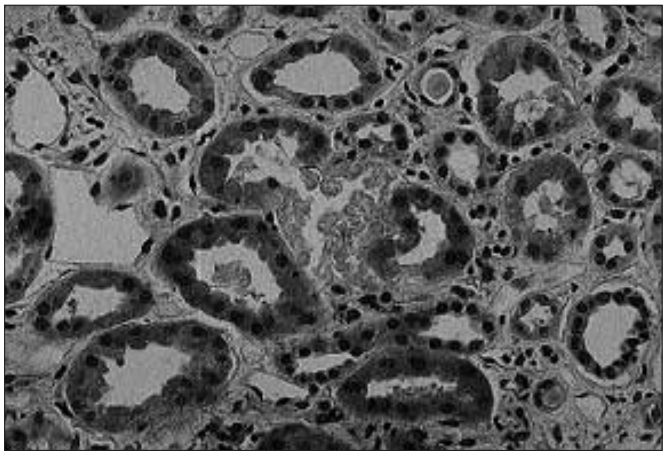

FIGURE 10-10 (see Color Plate)
Acute tubular necrosis in the allograft. Unlike "acute tubule necrosis" in native kidney, in this condition actual necrosis appears in the transplanted kidney but in a very small proportion of tubules, often less than one in 300 tubule cross sections. Where the necrosis does occur it tends to affect the entire tubule cross section, as in the center of this field [3].

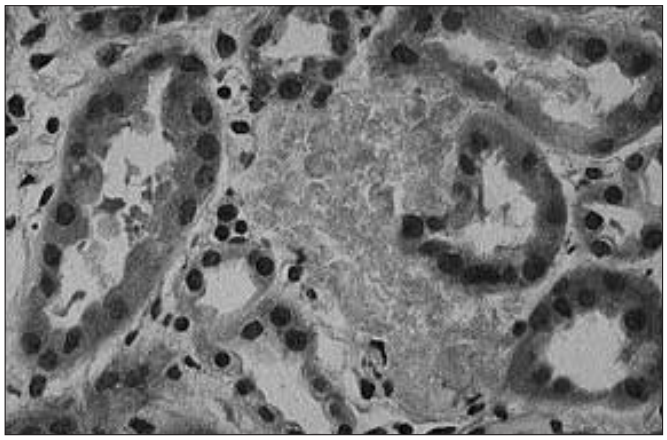

FIGURE 10-11 (see Color Plate)
A completely necrotic tubule in the center of the picture in a case of acute tubular necrosis (ATN) in an allograft. The tubule is difficult to identify because, in contrast to the appearance in native kidney ATN, no residual tubular cells survive; the epithelium is 100% necrotic.

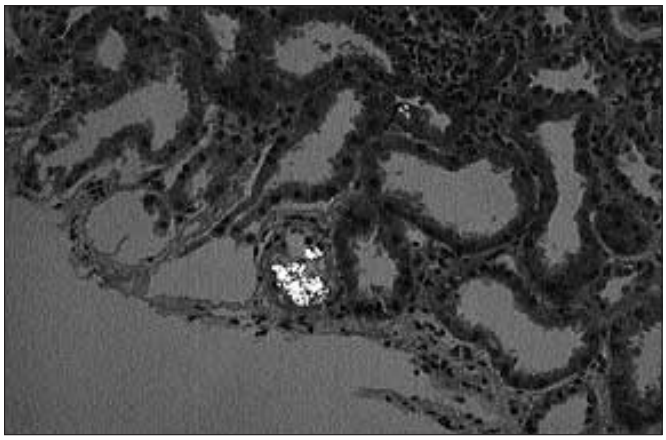

FIGURE 10-12 (see Color Plate)
Calcium oxalate crystals seen under polarized light. These are very characteristic of transplant acute tubular necrosis (ATN), probably because they relate to some degree to the duration of uremia, which is often much longer in transplant ATN (counting the period of uremia before transplantation) than in native ATN. With prolonged uremia elevation of plasma oxalate is greater and more persistent and consequently tissue deposition is greater [4].

FIGURE 10-13
Calcium oxalate crystals seen by electron microscopy in transplant acute tubular necrosis.

FEATURES OF TRANSPLANT ACUTE TUBULAR NECROSIS (ATN) WHICH DIFFERENTIATE IT FROM NATIVE KIDNEY ATN

1. Apparently intact proximal tubular brush border
2. Occasional foci of necrosis of entire tubular cross sections
3. More extensive calcium oxalate deposition
4. Significantly fewer tubular casts
5. Significantly more interstitial inflammation
6. Less cell-to-cell variation in size and shape (“tubular cell unrest”)

FIGURE 10-14
Features of transplant acute tubular necrosis that differentiate it from the same condition in native kidney [3].

Cyclosporine Toxicity

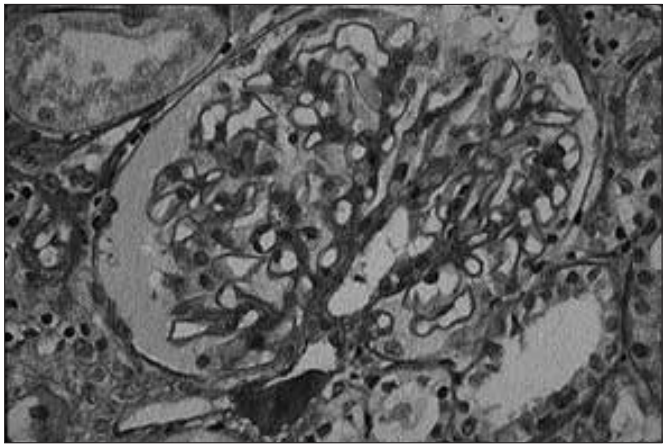

FIGURE 10-15
Cyclosporine nephrotoxicity with new-onset hyaline arteriolar thickening in the renin-producing portion of the afferent arteriole [5]. This lesion can be highly variable in extent and severity from section to section of the biopsy specimen, and it represents one of the strong arguments for examining multiple sections. The lesion is reversible if cyclosporine levels are reduced. Tacrolimus (FK506) produces an identical picture.

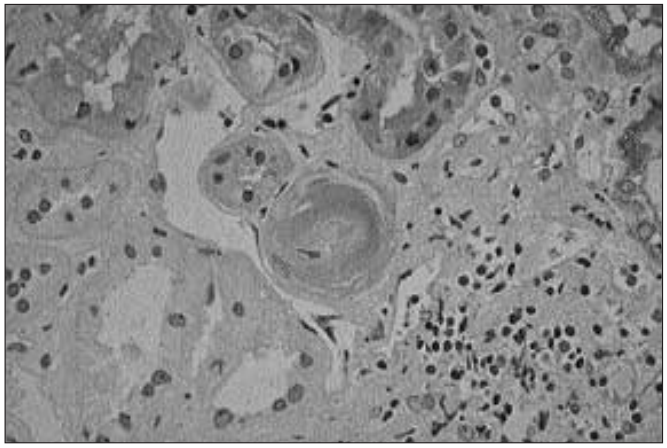

FIGURE 10-16 (see Color Plate)
Bland hyaline arteriolar thickening of donor origin in a renal allograft recipient never treated with cyclosporine. This phenomenon provides a strong argument for doing implantation biopsies; otherwise, donor changes can be mistaken for cyclosporine toxicity.

Posttransplant Lymphoproliferative Disorder

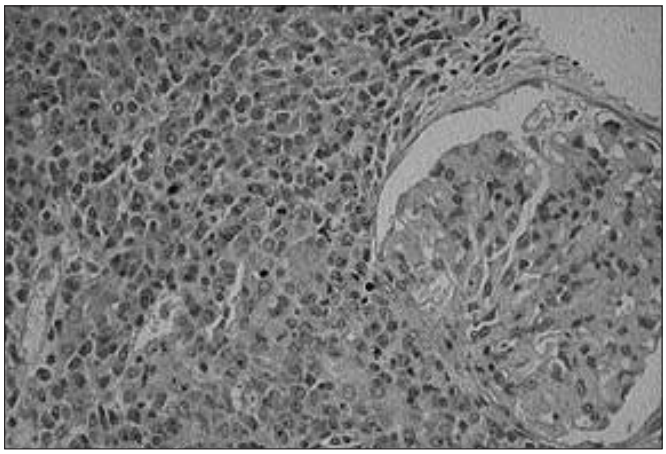

FIGURE 10-17
Posttransplant lymphoproliferative disorder (PTLD). The least satisfying facet of the 1997 Fourth Banff Conference on Allograft Pathology was the continued lack of good tools for the renal pathologist trying to distinguish the more subtle forms of PTLD from rejection. PTLD is rare, but, if misdiagnosed and treated with increased (rather than decreased) immunosuppression, it can quickly lead to death. The fact that both rejection and PTLD can occur simultaneously makes the challenge even greater [6]. It is hoped that newer techniques will make the diagnosis of this important condition more accurate in the future [7–9]. This figure shows an expansile plasmacytic infiltrate in a case of PTLD. However, most cases of PTLD are the result of Epstein-Barr virus–induced lymphoid proliferation.