Vascular disorders of the kidney comprise a very heterogeneous array of lesions and abnormalities, depending on the site of the lesion and underlying pathogenesis. Here, three common disorders are the focus: thrombotic microangiopathies, benign and malignant nephrosclerosis, and vascular occlusive disease (atheroembolism). Vasculitis and renovascular hypertension are discussed in other chapters.
FIGURE 5-1
Light microscopy of thrombotic microangiopathies. This group of disorders includes hemolytic-uremic syndrome and thrombotic thrombocytopenic purpura, malignant hypertension, and renal disease in progressive systemic sclerosis (scleroderma renal crises). 

A. These lesions are characterized primarily by fibrin deposition in the walls of the glomeruli (fibrin). B. This fibrin deposition is associated with endothelial cell swelling (arrow) and thickened capillary walls, sometimes with a double contour. Variable capillary wall wrinkling and luminal narrowing occur. Mesangiolyis (dissolution of the mesangial matrix and cells) is not uncommon and may be associated with microaneurysm formation. With further endothelial cell damage, capillary thrombi ensue. C. Arteriolar thrombi also may be present. In arterioles, fibrin deposits in the walls and lumina are known as thrombocytocotic lesions, with extension of this process into the glomeruli on occasion (arrow). The arterial walls are thickened, with loose concentric intimal proliferation. D. On electron microscopy, the subendothelial zones of the glomerular capillary wall are widened (arrows). Flocculent material accumulates, corresponding to mural fibrin, with associated endothelial cell swelling. E. With widespread arterial thrombosis, cortical necrosis is a common complicating feature. The necrotic cortex consists of pale confluent multifocal zones throughout the cortex.
Microangiopathic hemolytic anemia. Bizarrely shaped and fragmented erythrocytes are commonly seen in Wright's stained peripheral blood smears from patients with active lesions of thrombotic microangiopathy. These abnormally shaped erythrocytes presumably arise when the fibrin strands within small blood vessels shear the cell membrane, with imperfect resolution of the biconcave disk shape. The resultant intravascular hemolysis causes anemia, reticulocytosis, and reduced plasma haptoglobin level.

Disseminated intravascular coagulation. In disseminated intravascular coagulation, fibrin thrombi are typically found in many glomerular capillary lumina. In contrast to the thrombotic microangiopathies, in disseminated intravascular coagulation, fibrin is not primarily in vessel walls but in the lumina. Consequently, the capillary wall thickening, endothelial cell swelling, and fibrin accumulation in subendothelial locations are not features of this lesion. In the glomerulus illustrated, the fibrin is in many capillary lumina and appears as bright fuchsin positive (red) masses.

Benign and malignant nephrosclerosis. In benign nephrosclerosis the artery walls are thickened with intimal fibrosis and luminal narrowing. Arteriolar walls are thickened with insudative lesions, a process affecting afferent arterioles almost exclusively. Both of these processes, which can be quite patchy, result in chronic ischemia. In glomeruli, chronic ischemia is manifested by gradual capillary wall wrinkling, luminal narrowing, and shrinkage and solidification of the tufts. As these processes progress, collagen forms internal to Bowman's capsule, beginning at the vascular pole and growing as a collar around the wrinkled ischemic tufts. This collagen formation ultimately is associated with tubular atrophy and interstitial fibrosis.

In malignant nephrosclerosis the changes are virtually identical to those of thrombotic microangiopathies (Fig. 5-1 C). Malignant nephrosclerosis may be seen in essential hypertension, scleroderma, unilateral renovascular hypertension (with a contralateral or “unprotected” kidney), and as a complicating event in many chronic renal parenchymal diseases.
Vascular occlusive disease and thrombosis. Atheroemboli (cholesterol emboli) are most commonly associated with intravascular instrumentation of patients with severe arteriosclerosis. Most commonly, aortic plaques are complicated with ulceration and often adherent fibrin, A. Portions of plaques are dislodged and travel distally in the aorta. Because the kidneys receive a disproportionately large share of the cardiac output, they are a favored site of emboli. Typically, the emboli are in small arteries and arterioles, although glomerular involvement with a few cholesterol crystals in capillaries is not uncommon. Because of the size of the crystals, it is sometimes difficult if not impossible to identify them in glomerular capillaries in paraffin-embedded sections. In plastic-embedded sections prepared for electron microscopy, however, the crystals are quite easy to detect. On light microscopy, cholesterol is represented by empty crystalline spaces. In the early stages of the disease the crystals lie free in the vascular lumina. In time, the crystals are engulfed by multinucleated foreign body giant cells. B, In this light microscopic photograph, a few crystals are evident in the glomerular capillary lumina and in an arteriole (arrows). C, In the electron micrograph the elongated empty space represents dissolved cholesterol. Note that no cellular reaction is evident.