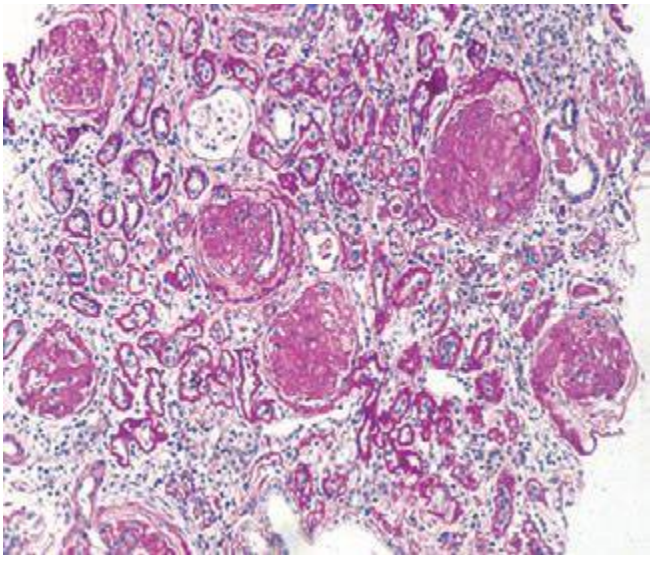
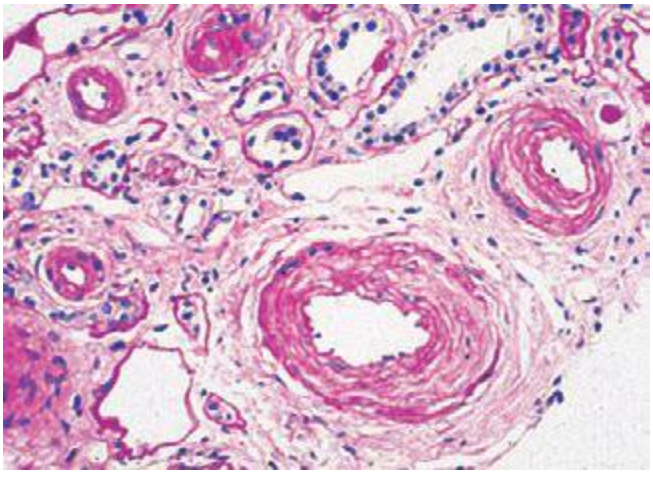


A



B

FIGURE 62e-24 Arterionephrosclerosis. Hypertension-associated injury often manifests extensive global sclerosis of glomeruli, with accompanying and proportional tubulointerstitial fibrosis and pericapsular fibrosis, and there may be segmental sclerosis (***A***). The vessels show disproportionately severe changes of intimal fibrosis, medial hypertrophy, and arteriolar hyaline deposits (***B***). *(ABF/Vanderbilt Collection.)*

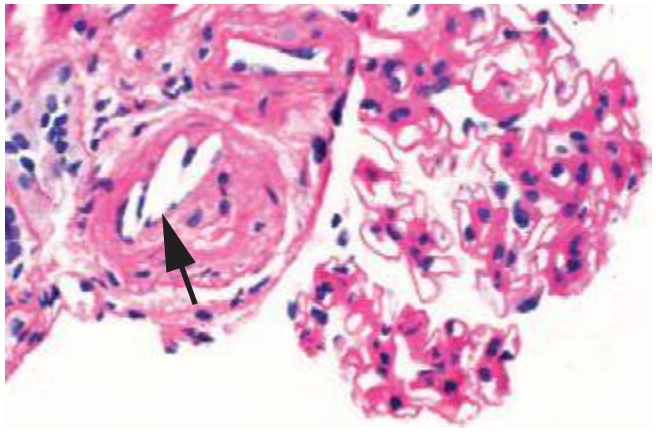


FIGURE 62e-25 Cholesterol emboli. Cholesterol emboli cause cleft-like spaces (*arrow*) where the lipid has been extracted during processing, with smooth outer contours and surrounding fibrotic and mononuclear cell reaction in these arterioles. *(ABF/Vanderbilt Collection.)*

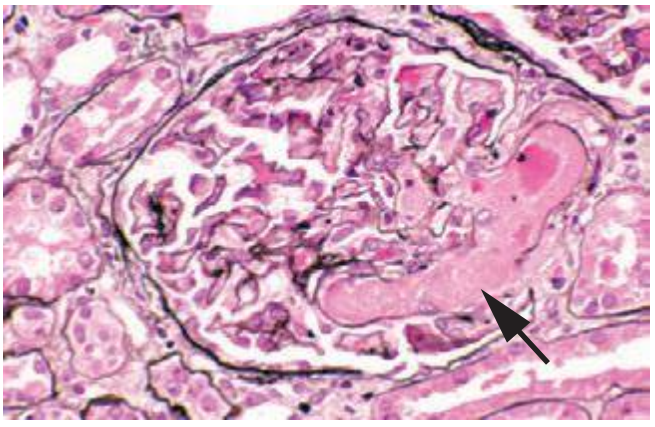
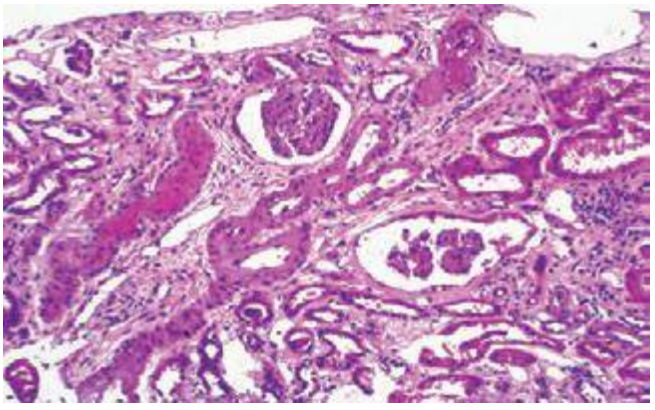
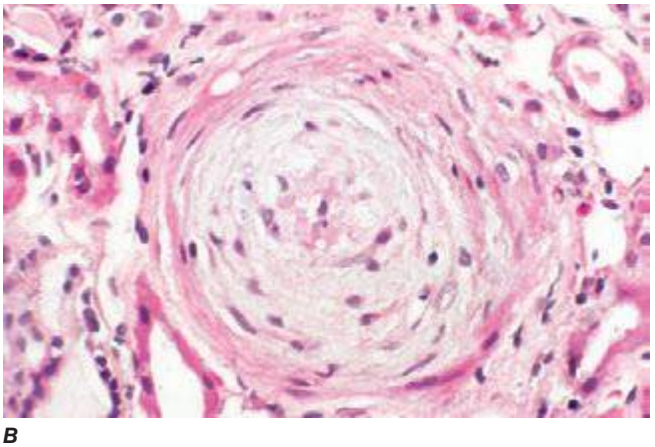


FIGURE 62e-26 Hemolytic-uremic syndrome. There are characteristic intraglomerular fibrin thrombi, with a chunky pink appearance (thrombotic microangiopathy) (*arrow*). The remaining portion of the capillary tuft shows corrugation of the glomerular basement membrane due to ischemia. *(ABF/Vanderbilt Collection.)*



A



B

FIGURE 62e-27 Progressive systemic sclerosis. Acutely, there is fibrinoid necrosis of interlobular and larger vessels, with intervening normal vessels and ischemic change in the glomeruli (***A***). Chronically, this injury leads to intimal proliferation, the so-called onion-skinning appearance (***B***). *(ABF/Vanderbilt Collection.)*