perfusion in the setting of aortic dissection, but it is not recommended for the initial investigation of aortic dissection.

Aortic dissection is a hypertensive emergency that requires aggressive reduction of blood pressure; systolic blood pressure should be maintained between 100 and 120 mm Hg. Antihypertensive medications that reduce the rate of increase in blood pressure during the cardiac cycle (dP/dt), such as β-adrenergic receptor blockers, have a theoretical benefit in managing aortic dissection by reducing the rate of progression.

Surgical treatment options for renal involvement due to aortic dissection depend on individual circumstances, and careful evaluation by an experienced vascular surgeon is recommended. Thoracic aortic dissection requires surgical repair due to the high mortality rate if left untreated, but isolated abdominal aortic disease may be medically managed.

Thromboembolic Disease

Systemic arterial emboli, typically originating from the left atrium or left ventricle in patients with atrial fibrillation, infectious endocarditis, cardiac valvular disease, or atrial myxoma, may cause acute obstruction of the renal arteries. Rarely, a paradoxical embolus may occur from the venous system through an atrial septal defect.

Symptoms of acute renal ischemia and infarction include flank pain, gross hematuria, and fever. Laboratory findings are nonspecific but include an elevated level of lactate dehydrogenase (LDH), hematuria, and leukocytosis. A definitive diagnosis can be based on the finding of a focal nonenhancing region on contrast-enhanced CT. Imaging studies are necessary to differentiate renal artery embolic disease from renal artery dissection.

The renal mass affected by a renal artery embolus is usually not large enough to necessitate dialysis, although some worsening of kidney function may be observed. The diagnosis of renal infarction is rarely made early enough to initiate treatment with intra-arterial thrombolysis or thrombectomy, and it is questionable whether the risks and marginal benefit of these procedures warrant aggressive treatment. Therapy should instead address the underlying source of renal emboli with symptomatic treatment of pain as necessary. Systemic anticoagulation may be indicated to reduce the risk of further thromboembolic events.

Large and Medium-Sized Vessel Vasculitis

Systemic vasculitides such as temporal (giant cell) arteritis and Takayasu's arteritis affect primarily large and medium-sized arteries. Polyarteritis nodosa and Kawasaki disease affect primarily medium-sized and smaller arteries. These vasculitides are not associated with antineutrophil cytoplasmic antibodies (ANCAs) and do not typically cause glomerulonephritis. They are distinguished from ANCA-associated vasculitides that involve smaller blood vessels and more commonly cause glomerulonephritis.

Takayasu's arteritis and giant cell arteritis are typically associated with a granulomatous vasculitis of the aorta and its branches. Giant cell arteritis typically involves the carotid, vertebral, and temporal arteries, and renal involvement is rare. Both occur much more commonly in women than men. Takayasu's arteritis is usually diagnosed in patients younger than 50 years of age, whereas giant cell arteritis is diagnosed in those 50 years of age or older.

Involvement of the main renal arteries occurs in about 40% of patients with Takayasu's arteritis, producing areas of stenosis with renal ischemia or renal infarction. Common clinical features are constitutional symptoms, claudication, bruits, and hypertension. Pulses are often diminished or absent in one or more extremities, and a blood pressure discrepancy more than 10 mm Hg in the limbs is common. The diagnosis of Takayasu's arteritis is most often made on clinical grounds along with typical angiographic or other imaging findings. Corticosteroids are the primary treatment modality.

Polyarteritis nodosa is a medium-sized or small vessel vasculitis with no gender predilection that predominantly occurs in patients between 40 and 60 years of age. It affects the main renal arteries and renal interlobar arteries (less commonly, the arcuate and interlobular arteries) with a necrotizing vasculitis that typically produces microaneurysms of the intrarenal arteries. They can be seen on arteriograms in 40% to 90% of patients with renal involvement. Renal ischemia leads to loss of kidney function and renin-mediated hypertension. Low-grade proteinuria and hematuria may be seen, but the finding of acute glomerulonephritis indicates some other disorder. Renal infarction may occur, and rarely, a renal artery aneurysm may cause renal artery dissection or rupture.

The diagnosis is made on clinical grounds and by arteriography. There are no confirmatory serologic tests; polyarteritis nodosa is not an ANCA-associated vasculitis. Arteriography appears to be superior for diagnosis compared with CT and MRA. Progressive renal disease is not typical but may occur. Treatment with corticosteroids and immunosuppressive drugs is effective in reducing disease severity and mortality.

Kawasaki disease is an arteritis associated with the mucocutaneous lymph node syndrome that affects mostly medium-sized and small arteries, although the aorta may also be involved. It is primarily a self-limited disease of infants and young children. Renal involvement is extremely rare.

Hypertensive Nephrosclerosis

Chronic hypertension in susceptible individuals may lead to development of proteinuria, CKD, and ESRD. Hypertensive nephrosclerosis is cited as a cause of CKD and ESRD in African Americans at a much higher rate than whites, even with similar levels of blood pressure control and despite good control.

The renal manifestations of chronic hypertension include renal arterial and arteriolar intimal thickening and luminal narrowing with medial hypertrophy and fibroblastic intimal thickening of arteries and deposition of hyaline-like material in the walls of arterioles. Glomeruli show global and focal glomerulosclerosis; the former likely results from glomerular ischemia and the latter from increased intracapillary pressure and compensatory hypertrophy and injury in response to nephron loss. Wrinkled glomerular basement membranes due to glomerular ischemia are seen on electron microscopy. Chronic interstitial nephritis with tubular atrophy and interstitial fibrosis also occurs. These histopathologic changes of hypertensive nephrosclerosis are often found on renal biopsy for patients with other disorders, such as diabetes mellitus, atheroembolic disease, and RAS.

The overall risk of hypertensive nephrosclerosis with progressive CKD is low in the general hypertensive population, and most