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Endovascular Treatment of Large Vessel Arteritis

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Large vessel vasculitis is very uncommon, particularly in Western countries, but can cause serious sequelae. Large vessel vasculitis is usually due to either Takayasu arteritis or giant cell arteritis. The available laboratory tests are nonspecific, so the diagnosis is often dependent on imaging findings. The location and pattern of vessel narrowing will usually define the type of vasculitis. Symptomatic vascular stenoses and occlusions can frequently be managed using balloon angioplasty. Familiarity with the indications for treatment and techniques and outcomes associated with angioplasty and surgical alternatives is essential when treating these rare disorders.
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Clinical Evaluation

Most cases of large vessel vasculitis are due to either Takayasu arteritis (TA) or giant cell arteritis (GCA). Both of these disorders cause vessel wall inflammation but have slightly different distributions.[1] Involvement of the aorta and the proximal brachiocephalic arteries indicates TA, although other systemic disorders such as Behçet disease can rarely cause an aortitis. GCA more typically involves the temporal or axillobrachial arteries. Involvement of the renal arteries or mesenteric is more characteristic of TA. There are no specific biochemical or immunologic tests for either GCA or TA, but inflammatory markers such as the erythrocyte sedimentation rate (ESR) and C-reactive protein tend to be elevated during the acute phase of the disease.[2] The age of the patient at the time of clinical presentation is helpful in differentiating between these 2 entities as most patients with TA present during their second to fourth decades of life, whereas those with GCA typically present after the age of 50 years.

The incidence of TA is approximately 2.6 per million, but TA is more common in women and in Asia. GCA has geographic variability with an estimated incidence ranging from 1-18.8 per 100,000. It is 2-3 times more common in women and much more common in whites. Presenting symptoms in both disorders are variable, with headache and malaise being most common, and neurologic symptoms, including stroke, are additional common presentations.[3,4] Computed tomography angiography (CTA) and magnetic resonance angiography are essential tools in making the clinical diagnosis.[5] The relatively unique distribution of TA and GCA can often be demonstrated with cross-sectional imaging. Arterial wall enhancement, if present, helps differentiate large vessel vasculitis from atherosclerosis. Positron emission tomography has been recommended for determining the extent of the disease and for assessing the activity of the disease.[6]

Indications for Invasive Treatment

Angiography is rarely required for the clinical diagnosis with refinements in CTA and magnetic resonance angiography and is reserved for most patients in lieu of a potential percutaneous intervention. The mainstay of treatment is steroid administration during the acute inflammatory phase of the disease process and indeed, percutaneous therapy should not be done during the acute phase of the disease process (as suggested by an elevated ESR and C-reactive protein, in associated with systemic symptoms of myalgias, arthralgias, or low-grade fevers). Resistant cases may also receive additional immunosuppressive agents.[7] Medical management can arrest the process, but vessel stenoses or occlusions very rarely resolve.

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Percutaneous transluminal angioplasty (PTA) or surgical bypass are the 2 main considerations for revascularization of symptomatic lesions, both which are preferably performed during the quiescent stage of the disease.[8,9] Symptomatic stenoses and occlusions are indications for invasive treatment. Carotid or vertebral interventions may also be considered in asymptomatic patients in whom the critical cerebral circulation is compromised. It is less clear when prophylactic treatment is indicated for asymptomatic mesenteric, renal, brachiocephalic, or axillobrachial artery lesions. TA may have associated aneurysms: small aneurysms (<2 cm and not enlarging) can be watched, but larger aneurysms may need treatment with a covered stent, an endograft, or open repair.

The timing of endovascular or open therapy is important. As noted before, invasive treatment should be avoided during the acute phase of the disease.[10,11] Technical success and long-term patency rates are lower when procedures are performed during the active phase of the disease. After a course of steroids and when the ESR returns to reference range, outcomes with revascularization therapy are better. Significant or refractory symptoms or high-risk lesions (critical lesion of a common carotid artery) may necessitate therapy in patients during the acute

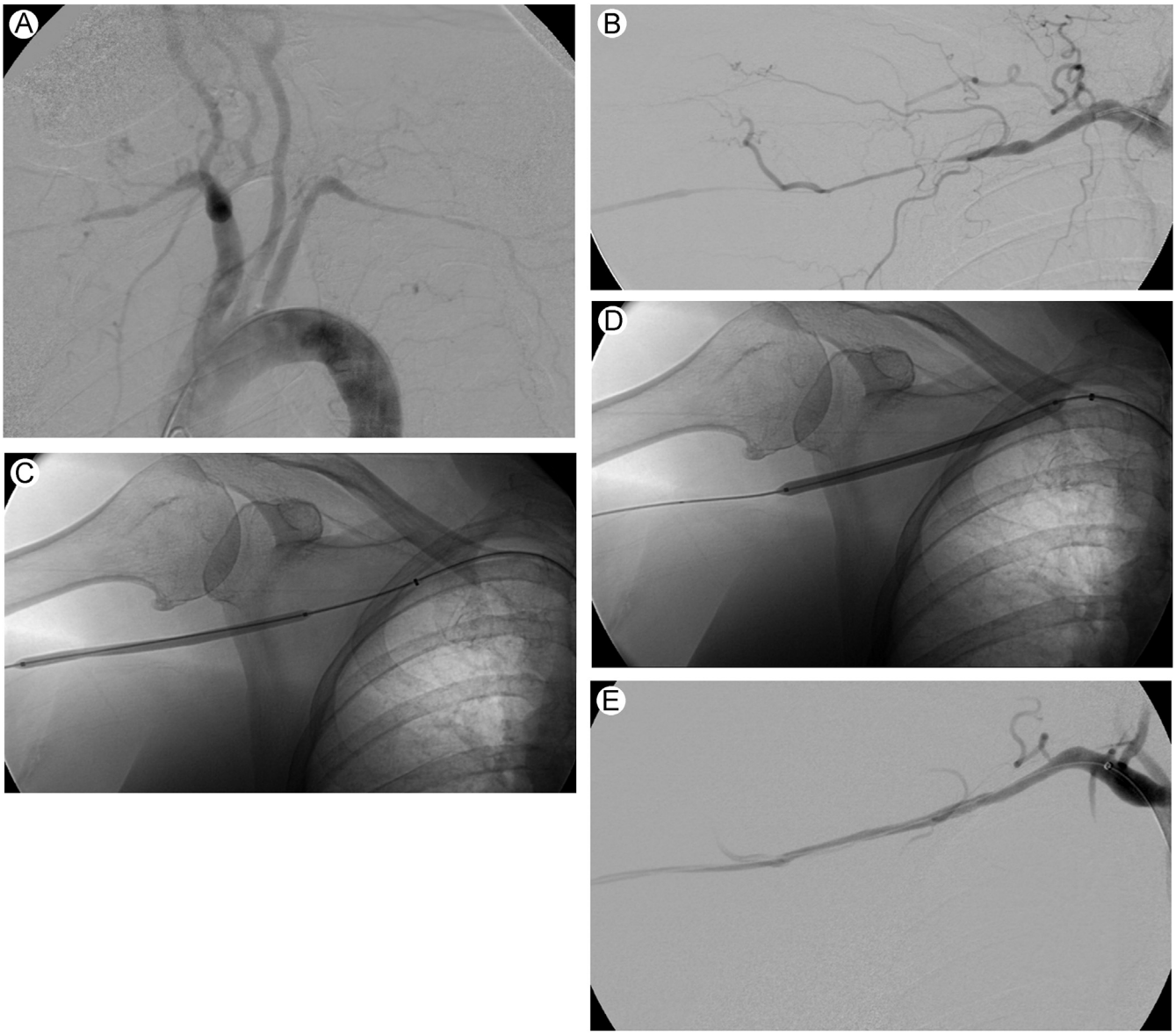

Figure 1 A 65-year-old woman presented with severe bilateral claudication. She had diminished pulses in both arms. Initially, she was managed with oral steroids. Although her erythrocyte sedimentation rate returned to reference range, her arm symptoms persisted. (A) An arch aortogram demonstrates normal proximal brachiocephalic vessels. (B) Selective right subclavian angiogram demonstrates a long smooth narrowing of the axillobrachial artery, consistent with giant cell arteritis. (C and D) The long segment of narrowing was dilated with 2 inflations of a noncompliant PTA balloon. The left brachial artery had similar findings and was treated similarly (not shown). (E) After PTA, there is some residual narrowing, but the patient had resolution of her claudication symptoms and remained symptom free at 1-year follow-up.

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