Endovascular Management of Aortoiliac Occlusive Disease

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ABSTRACT

Patients with aortoiliac occlusive disease (AIOD) may be asymptomatic or may have intermittent claudication or critical limb ischemia. Treatment options for AIOD include management of risk factors, endovascular intervention, and/or surgical revascularization.

KEYWORDS: Aortoiliac occlusive disease, peripheral arterial disease, intermittent claudication, critical limb ischemia, balloon angioplasty, stent, bypass graft

Objectives: Upon completion of this article, the reader should be able to identify the diagnostic and therapeutic options available for aortoiliac occlusive disease.

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CLINICAL PRESENTATION

Peripheral arterial disease (PAD) affects 8 to 10 million people in the United States per year.[1] One subset of PAD is aortoiliac occlusive disease (AIOD). AIOD can occur anywhere from the distal aorta to the common femoral arteries and is therefore called an "inflow lesion." Stenoses may be short- or long-segment, calcified, ulcerated, concentric or eccentric, single or multiple, unilateral or bilateral and may involve the aorta or iliac arteries alone or together. Focal infrarenal aortic stenoses that exclude the aortic bifurcation are fairly rare. This is most often seen in younger patients with less atherosclerotic disease.[2] More commonly, distal aortic occlusive disease extends into the common iliac arteries. Hypoplastic aortoiliac syndrome is seen most commonly in female smokers, in which the atherosclerotic effects of smoking narrow an already small aorta and iliac arteries. The Rutherford and Fontaine scoring systems for PAD are compared in Table 1, and essentially divide patients with AIOD into those with no symptoms, intermittent claudication (IC), or critical limb ischemia (CLI). Asymptomatic patients with PAD would be unlikely to benefit from endovascular or surgical treatment and may experience treatment-related complications, and, therefore, intervention should be limited to those with lifestyle-limiting claudication or limb-threatening ischemia.

IC

IC of the lower extremity is classically muscular pain brought on by activity and relieved by short rest. The prevalence of IC is 3% in those less than 40 and 6% in those older than 60.[1] Intermittent claudication caused by AIOD typically involves the thigh/buttock and may cause male impotence. Patients with IC should have lifestyle-limiting symptoms and be expected to have

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Table 1 Fontaine's Stages and Rutherford's Categories

Fontaine		Rutherford		
Stage	**Clinical**	**Grade**	**Category**	**Clinical**
Claudication				
I	Asymptomatic	0	0	Asymptomatic
IIa	Mild claudication	I	1	Mild claudication
IIb	Moderate to severe claudication	I	2	Moderate claudication
		I	3	Severe claudication
Critical limb ischemia				
III	Ischemic rest pain	II	4	Ischemic rest pain
IV	Ulceration or gangrene	III	5	Minor tissue loss
		III	6	Major tissue loss

reasonable symptom improvement from either endovascular or surgical revascularization prior to undergoing evaluation for revascularization.[3] Of those patients who present with typical IC, after 5 years, 70 to 80% will have stable claudication, 10 to 20% will have worsening claudication, and 5 to 10% will develop CLI.[1] Less than 2% of patients with IC go on to major amputation.[4,5] Other causes of aortoiliac IC include arteritis, congenital and acquired coarctation of aorta, endofibrosis of the external iliac artery (EIA; iliac artery syndrome in cyclists), fibromuscular dysplasia, peripheral emboli, primary vascular tumors, pseudoxanthoma elasticum, remote trauma and radiation injury (Fig. 1), Takayasu's disease, thromboangiitis obliterans (Buerger's disease), and thrombosis of a persistent sciatic artery.[1] Mimics for iliac artery IC include spinal stenosis and hip arthritis. Leriche syndrome is atheromatous occlusion of the distal abdominal aorta extending into the common iliac artery (CIA) origins with a triad of claudication, absent or weak femoral pulses, and erectile dysfunction.

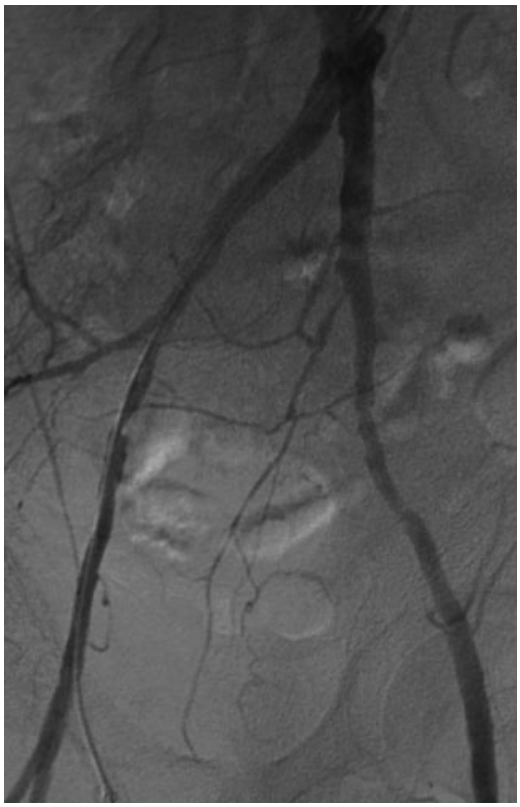

Figure 1 Changes of pelvic radiation for anal cancer. Pelvic oblique digital subtraction angiogram shows bilateral external iliac artery long-segment stenoses and left internal iliac artery origin stenosis. Patient was treated with primary stent placement in right external iliac artery and angioplasty with provisional stent placement in the left external iliac artery (not shown).

CLI

CLI is defined as pain at rest and/or tissue loss. Of those with CLI, at 1 year 45% are alive with two lower extremities, 30% undergo amputation, and 25% have died.[1] The differential diagnosis for rest pain includes diabetic neuropathy, complex regional pain syndrome, nerve root compression, and musculoskeletal disorders. In blue-toe syndrome, patients embolize distally, and this may occur even in the absence of claudication or significant stenosis. Symptoms must be present for more than 2 weeks to be considered chronic.

PATHOGENESIS OF AIOD

Risk factors for development of AIOD include nonwhite ethnicity, smoking, diabetes mellitus, dyslipidemia, hypertension, age, male gender, C-reactive protein elevation, hyperhomocystinemia, hyperviscosity/hypercoagulability, and chronic renal insufficiency.[1,6] Optimization of modifiable risk factors is an important component of successful management of patients with AIOD.

DIAGNOSIS

Noninvasive Methods

The noninvasive methods for the evaluation for lower-extremity PAD include a focused history and physical examination, physiological ultrasound, computed tomographic angiography and magnetic resonance (MR) angiography. According to the American College of Cardiology (ACC)/American Heart Association (AHA) Guidelines for the Management of Patients with Peripheral Arterial Disease,[3] individuals with risk factors for lower-extremity PAD should undergo a vascular review of systems to assess for claudication, rest pain, and/or wounds and should undergo a comprehensive pulse examination and foot inspection. Patients with CLI should undergo an expedited vascular evaluation.[3] The Society of Interventional Radiology standard of practice regarding physiological evaluation of extremity arteries suggests the use of segmental blood pressures, continuous Doppler waveforms, and/or volume plethysmography to evaluate for PAD.[7] Baseline bilateral ABI measurements should be obtained. An ankle brachial index (ABI) of $\leq$0.90 is indicative of PAD. An exercise ABI may be useful for those with or without IC who have a normal resting ABI, and measurement of toe-brachial index or pulse volume recording may be useful in asymptomatic patients if the resting ABI is greater than 1.30.[3] Color flow duplex ultrasonography may also be helpful to characterize stenoses, although in the aortoiliac system, this may be difficult based on patient body habitus. Computed tomographic angiography may be used to localize and diagnose severity of stenoses in individuals with PAD, especially in those with a contraindication to MR angiography. MR angiography should be performed with gadolinium. Impaired renal function prevents using gadolinium due to the association with nephrogenic systemic fibrosis.

Catheter Angiography

Because of the usefulness and ready availability of noninvasive imaging modalities, catheter angiography is rarely used now as a primary diagnostic modality but rather as a problem-solving tool. When necessary, digital subtraction angiography should be used when evaluating the aorta, iliac, and runoff vessels.[3] Digital subtraction angiography is most often combined with simultaneous endovascular treatment, when appropriate. Of note, a useful diagnostic technique is carbon dioxide angiography, especially in those patients with marginal renal function or severe contrast allergy. Consideration should also be given to treatment with sodium bicarbonate in patients with marginal renal function prior to and following administration of intravenous contrast.

Trans-stenotic Pressure Measurement

In cases in which the severity of a stenosis is uncertain, trans-stenotic intra-arterial pressure measurements should be obtained.[3] A peak systolic pressure gradient of >10 mm Hg or a 5 mm Hg mean pressure gradient is considered significant. A 10 to 15 mm Hg peak systolic pressure gradient after injection of intra-arterial nitroglycerine or other vasodilator to simulate exercise is also considered significant. ACC/AHA guidelines suggest that when iliac arterial stenoses of 50 to 75% are identified, significance needs to be determined with intra-arterial pressure gradient measurement prior to intervening[3] (Fig. 2).

TRANSATLANTIC INTER-SOCIETY CONSENSUS GUIDELINES

The TransAtlantic Inter-Society Consensus (TASC) guidelines, first published in 2000 and then revised in 2007, classify aortic and iliac lesions by lesion morphology. TASC A lesions include unilateral or bilateral CIA stenoses or unilateral or bilateral single short stenosis ($\leq$3 cm) of EIA. TASC B lesions include short segment stenosis ($\leq$3 cm) of infrarenal aorta, unilateral CIA occlusion, single or multiple stenosis totaling 3–10 cm involving the EIA and not extending into the common femoral artery (CFA), unilateral EIA occlusion not involving the origins of the internal iliac artery or CFA. TASC C lesions include bilateral CIA occlusions, bilateral EIA stenoses 3 to 10 cm long not extending into the CFA, unilateral EIA stenosis extending into the CFA, unilateral EIA occlusion that involves the origins of the internal iliac and/or CFA, heavily calcified unilateral EIA occlusion with or without internal iliac and/or CFA origin involvement. TASC D lesions involve infrarenal aortoiliac occlusion; diffuse disease involving the aorta and both iliac arteries requiring treatment; diffuse multiple stenoses involving the unilateral CIA, EIA, and CFA; unilateral occlusions of both the CIA and EIA; bilateral occlusions of EIA; iliac stenoses in those requiring treatment for AAA who are poor candidates for endovascular treatment or who have other lesions requiring open surgical repair of aorta or iliac arteries. TASC II recommends endovascular treatment for TASC A and B lesions and surgical therapy for TASC C and D lesions.[1] Several studies have reported success with endovascular treatment of TASC C and D lesions,[8–10] and patient presentation and comorbidities should be taken into account when planning treatment.

NONENDOVASCULAR THERAPY

Medical Therapy

ACC/AHA guidelines suggest smoking cessation and hyperlipidemia, diabetes, and hypertension management

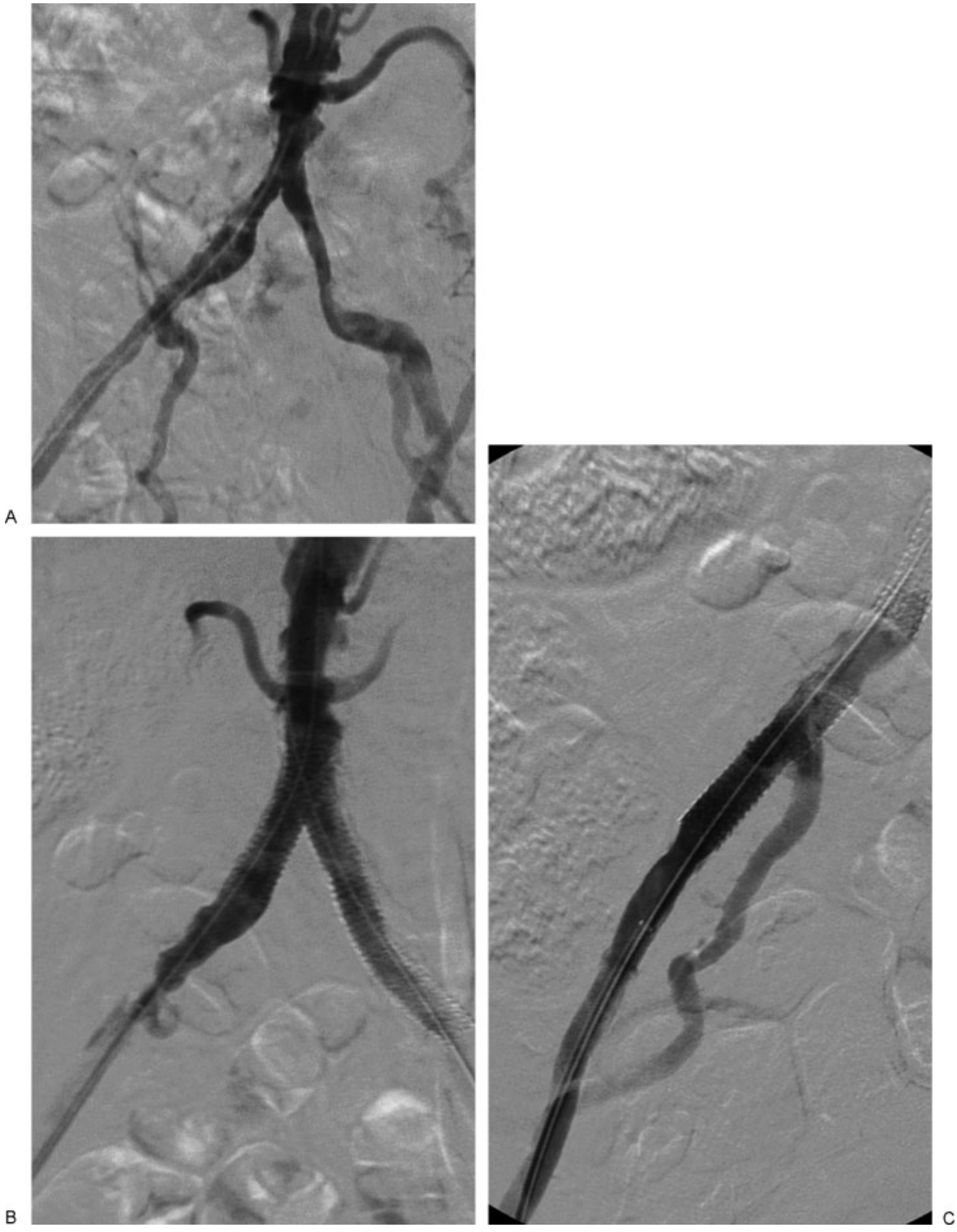

Figure 2 Trans-stenotic pressure measurement. (A) Distal abdominal aortic, bilateral common iliac artery (CIA) and right external iliac artery (EIA) stenoses. Intra-arterial pressure measurement confirms lesion significance with gradient of 18 mm Hg at right CIA origin. (B) ''Kissing'' bilateral CIA primary stent deployment to treat right CIA origin stenosis and mid left CIA stenosis. Right CIA pressure gradient has resolved after stenting, but right EIA stenosis persists. (C) Right EIA stenosis resolved after primary stent deployment.

as well as antiplatelet therapy according to current treatment guidelines for asymptomatic patients.[3] Aspirin and other antiplatelet agents (clopidogrel) are important to reduce the risk of cardiovascular events in those with PAD, but they have not been shown to reduce claudication.[11] Also to be considered is the use of angiotensin-converting enzyme inhibiting medication for cardiovascular risk reduction in asymptomatic individuals with PAD. Smoking cessation and successful management of hypertension and dyslipidemia can slow the progression of PAD.[1] Supervised exercise therapy may also be effective in some, especially older male patients.[12] Cilostazol has been shown to be effective in randomized controlled trials to reduce claudication.[13]

Surgical Therapy

TASC II suggests surgical therapy for type C and D lesions.[1] Surgical options for AIOD are anatomic versus extra-anatomic bypass graft or endarterectomy. Patency of aortic bifurcation grafts have been shown to be 90% and 75% at 5 and 10 years, respectively.[2,14] Extra-anatomic bypass graft 5-year patency ranges from 51% (44 to 79%) for axillary-unifemoral bypass graft to 71% (50 to 76%) for axillary-bifemoral bypass to 75% (55 to 92%) for femoral-femoral crossover graft.[1] Patient comorbidities should be taken into account when considering surgery.

ENDOVASCULAR THERAPY

Severe lifestyle-limiting claudication; rest pain, nonhealing ulcer, gangrene, or tissue loss; stenosis of the iliac system preventing other endovascular treatment; and decreasing renal function or hypertension in patients with renal transplant are indications for aortoiliac endovascular intervention. Contraindications include lack of symptoms, uncorrected anticoagulation, or operator inexperience with lack of appropriate surgical backup in the event of complication. Endovascular treatment options include angioplasty and/or stenting. After endovascular intervention, primary patency is defined as patency without any additional treatment. Primary assisted patency is defined as reintervention when the treated segment has not occluded, and secondary patency is defined as reintervention when the treated segment has occluded.

Aortic Lesions

As mentioned, isolated infrarenal aortic occlusive disease is rare. In an article comparing patients included in the SCVIR Transluminal Angioplasty and Revascularization (STAR) Registry with infrarenal aortic occlusive disease treated percutaneously with either angioplasty (13 patients) or angioplasty plus Palmaz stent placement (12 patients), no significant difference was found in procedural success or major or minor complication rate.[15]

Iliac Lesions

Several studies have found iliac artery angioplasty and stenting to be effective procedures.[16–18] As of yet, no definitive superiority of primary stent placement versus stenting after angioplasty failure has been documented. Provisional stent placement in the iliac arteries secondary to failed angioplasty with flow-limiting dissection or persistent translesional gradient is indicated.[3] Primary stenting is also effective in the common or external iliac arteries.[3] The Dutch Iliac Stent Trial found that angioplasty with provisional stenting of the iliac artery for angioplasty failure yielded similar technical results and would avoid stent placement in 63% of cases of IC.[19] In one study, 162 iliac artery stenoses were stented after failed angioplasty, and 141 iliac artery occlusions were stented primarily. The study found primary cumulative patency rates of $70 \pm 4\%$ after 5 years and $65 \pm 5\%$ after 7 years and secondary patency rates of $92 \pm 2\%$ after 5 years and $87 \pm 4\%$ after 9 years.[20] Tetteroo et al also did not show any clinical or technical difference in outcome with primary iliac stent placement versus dilatation and stent placement.[21] Stents may be balloon-expandable or self-expanding, covered or uncovered.[22] Houston et al evaluated the long-term patency of "kissing" self-expanding common iliac stent placement and found a primary patency rate of 89%, 82%, and 68% at 2, 5, and 10 years, respectively. Secondary patency rates were 93%, 93%, and 86% at 2, 5, and 10 years, respectively.[23] In another study, iliac recanalization and stent placement in 138 patients had a technical success of 99% and primary patency rates of 90%, 85%, 80%, and 68% at 3, 5, 7, and 10 years, respectively.[17] Figure 3 demonstrates bilateral CIA occlusions treated with primary stent deployment. Other studies have found a 5-year patency of 70 to 80% for primary stenting of the CIA.[20,24] A "reentry device" may be used with success to open an iliac artery occlusion.[25] Brachial access can also be considered in the event of unfavorable anatomy, such as in the setting of prior kissing iliac artery stent deployment or abdominal aortic stent-graft placement.

COMBINED SURGICAL AND ENDOVASCULAR THERAPY

CFA endarterectomy with ipsilateral iliac artery stenting or stent grafting demonstrated primary, primary assisted, and secondary patency rates of 60%, 97%, and 98%, respectively. Improved primary patency was seen when combining endarterectomy with covered stent grafts (87%) versus bare stents (53%).[26]

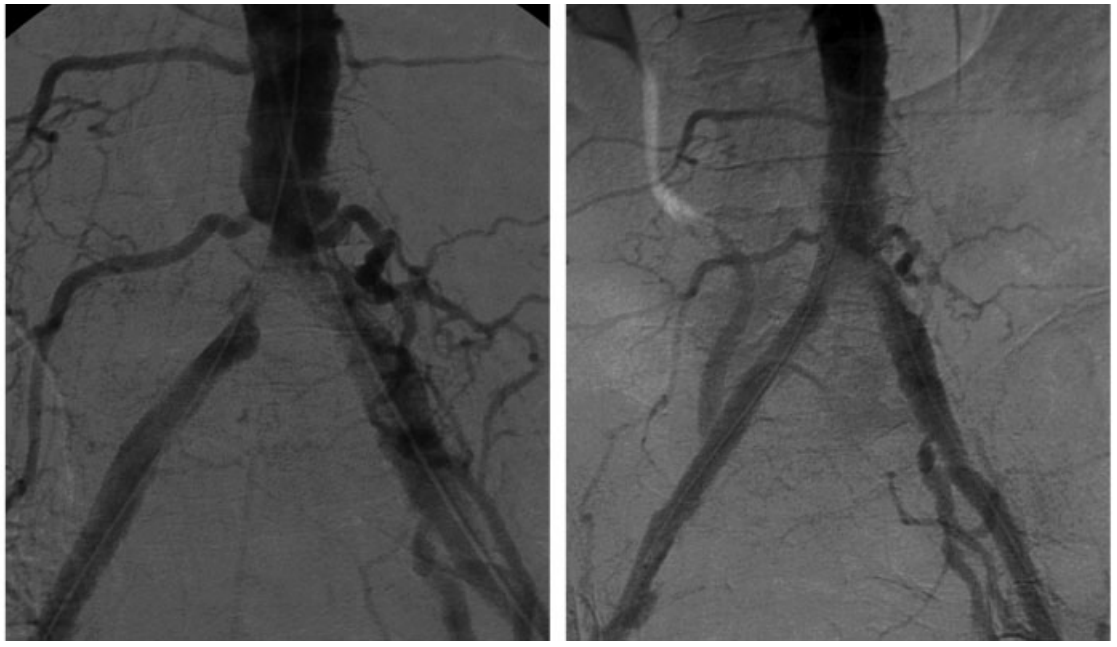

Figure 3 Recanalization of bilateral common iliac artery (CIA) occlusions. (A) Chronic total occlusion of bilateral CIAs with wire recanalization via bilateral common femoral arteries. (B) Successful primary CIA stent deployment.

COMPLICATIONS OF ENDOVASCULAR THERAPY

Many studies have shown that both angioplasty and stenting of infrarenal abdominal aortic stenoses are safe treatments.[27–33] One case report cites an arterioureteric fistula formation after EIA angioplasty and stenting.[34] Immediate complications include thrombosis, distal embolization, pseudoaneurysm, and arterial rupture. Most complications involve the access site. Often, careful attention to choice of appropriate puncture site and adequate hemostasis at procedure completion can prevent access site complications. Long-term complications include stent fracture, intimal hyperplasia, and thrombosis.

ADJUNCTIVE THERAPY

Antiplatelet therapy with aspirin or clopidogrel may prevent formation of thrombus within stented arteries.[20,35] TASC II guidelines recommend initiation of antiplatelet therapy at the time of endovascular intervention and continuation indefinitely.[1]

SURVEILLANCE

The first clinical follow-up after an endovascular intervention includes evaluation of the arterial puncture site for complication as well as vascular ultrasound and ABI calculation to establish the new baseline. Although ultrasound surveillance schedules vary, a plausible schedule is at 1 month after intervention, at 6 months, and then yearly and as needed. Presence or absence of symptoms should be documented in the ultrasound report.

CONCLUSION

The high prevalence of AIOD and potential for severe lifestyle-limiting claudication and/or tissue loss necessitates appropriate evaluation and management of this disease. With a combination of medical management of modifiable risk factors for PAD, accurate noninvasive and/or invasive diagnostic evaluation for patients with claudication or CLI, and endovascular and/or surgical revascularization when appropriate, symptomatic aortoiliac disease is often improved.

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