Perioperative Management of Lower Extremity Revascularization

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KEYWORDS

• Peripheral arterial disease • Atherosclerosis • Revascularization of lower extremity
• Neuraxial blockade • General anesthesia • Cardiovascular morbidity • Graft patency

KEY POINTS

- Patients presenting for lower extremity revascularization often have multiple systemic comorbidities, making them high-risk surgical candidates.
- Neuraxial anesthesia and general anesthesia are equivocal in their effect on perioperative cardiac morbidity and improved graft patency.
- Postoperative epidural analgesia may improve perioperative cardiac morbidity.
- Systemic antithrombotic and anticoagulation therapy is common among this patient population and may affect anesthetic techniques.

INTRODUCTION

Peripheral arterial disease (PAD) is a disease with significant morbidity and mortality. The recent focus on early screening and detection, aggressive medical management, and modification of risk factors has allowed many patients to avoid or delay more aggressive surgical intervention. The consequence of these practices is that a large majority of patients presenting for surgical lower extremity revascularization, through either endovascular or open techniques, have advanced systemic atherosclerotic disease involving not only peripheral limbs but also coronary, cerebral, and renal circulations. Vascular patients are among those with the highest perioperative risk, making them a challenge even for experienced anesthesiologists. Appropriate management of these patients requires an understanding of the pathophysiology of the disease, common comorbidities, perioperative complications, and possible surgical intervention strategies.

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PERIPHERAL ARTERIAL DISEASE

Epidemiology

PAD is common, affecting 3% to 10% of people younger than 70 years and 15% to 20% of people over age 70.[1] Globally, the number of people with PAD has increased by nearly one-fourth between 2000 and 2010,[2] despite the understanding that treatment of modifiable risk factors may decrease the burden of this disease. Lower limb PAD, specifically, is the third leading cause of atherosclerotic morbidity after coronary artery disease and stroke.[2]

Pathophysiology and Risk Factors

PAD is the term used to describe impairment of blood flow to the lower extremities. It is a progressive condition and results from any etiology causing an occlusion or stenosis of the lower limb arteries. Most commonly, the impaired blood flow is a result of atherosclerotic disease. Although much less common, there are nonatherosclerotic causes of PAD (**Box 1**).[3]

Atherosclerosis is a complicated physiologic phenomenon involving several highly interrelated processes, including lipid disturbance, platelet activation and thrombosis, endothelial dysfunction, inflammation, oxidative stress, vascular smooth muscle activation, altered matrix metabolism, and genetic factors.[4] It is a diffuse and progressive process, with a variable clinical presentation and distribution depending on the regional circulation involved.

A specific set of identifiable risk factors plays an important role in the initiation and acceleration of atherosclerotic lesions. Risk factor assessment remains the primary tool used for atherosclerotic disease screening. Race, age, and gender are the identified significant nonmodifiable risk factors. Smoking, diabetes, hypertension, and dyslipidemia make up the majority of the modifiable risk factors. Only smoking and dyslipidemia have been shown, through prospective controlled studies, to be risk factors where modification alters the development or course of PAD.[1] **Fig. 1** summarizes the influence of identified risk factors on PAD. Diabetes and smoking are the risk factors with the greatest risk of concomitant PAD. In patients with diabetes, for every 1% increase in hemoglobin A_{1c}, there is a 26% increased risk of PAD.[5] The severity of PAD found in smokers tends to increase with the number of cigarettes smoked. Cessation of smoking is associated with a decline in the development of intermittent claudication, a sign of symptomatic PAD.[1]

Box 1
Nonatherosclerotic causes of PAD

- Peripheral emboli
- Aneurysm thrombosis
- Arteritis (Takayasu, thromboangitis obliterans, giant cell arteritis, or polyarteritis nodosa)
- Fibromuscular dysplasia
- Prior trauma or irradiation injury
- Aortic coarctation
- Primary vascular tumors
- Pseudoxanthoma elasticum
- Young patients (advential cyst of popliteal artery, popliteal entrapment, or persistent sciatic artery)