Diagnosis, Prevention, and Treatment of Claudication

Jordan P. Knepper, MD, Peter K. Henke, MD*

KEYWORDS

• Claudication • Atherosclerosis • Peripheral arterial disease

KEY POINTS

- Claudication is a marker for systemic atherosclerosis, which mandates proper medical therapy and risk factor reduction.
- Increasing use of invasive therapies for patients with claudication has possible risks including thrombosis and progression to critical limb ischemia, and as such, providers must advise patients appropriately.
- For patients with true lifestyle limitations, consideration of the lesion length and location needs to be considered when offering endovascular or surgical revascularization.
- In those patients undergoing revascularization for claudication, long-term follow-up of the repair is essential.

BACKGROUND

Lower extremity chronic ischemia due to atherosclerosis represents the continuum of peripheral arterial disease (PAD), encompassing intermittent claudication (ranging from mild to lifestyle-limiting), rest pain, and tissue loss. Traditionally, the indication for invasive intervention has been critical limb ischemia (CLI), including those with rest pain and/or tissue loss, as a means to prevent amputation.

The Trans-Atlantic Inter-Society Consensus (TASC II) guideline regarding PAD was published in 2007, and defines claudication as muscle discomfort in the lower extremities, which is relieved by rest within 10 minutes.[1]

The frequency of claudication ranges with age, from 0.6% in a group of 45- to 54-year-olds, to 2.5% in 55- to 64-year-olds and 8.8% in 65- to 74-year-olds.[2] Importantly, there are other reasons for claudication, as studies have noted that only 16% to 20% people with symptoms have noninvasive evidence of PAD-related claudication. PAD is defined as an at rest ankle brachial index (ABI) of less than 0.9.[3]

Section of Vascular Surgery, University of Michigan, CVC - 5463, 1500 East Medical Center Drive, SPC-5867, Ann Arbor, MI 48109, USA
* Corresponding author.
E-mail address: henke@umich.edu

Surg Clin N Am 93 (2013) 779–788
http://dx.doi.org/10.1016/j.suc.2013.04.005 **surgical.theclinics.com**
0039-6109/13/$ – see front matter

Given the systemic nature of atherosclerosis understanding the natural history of PAD related to claudication is important for determining clinical decisions. Although angiographic progression may be seen in more than 60% of patients,[2] only 2% of patients presenting with claudication will eventually progress to amputation in the 5 years following diagnosis.[3] This indicates a relatively benign nature of claudication, and highlights the importance of diagnosis for medical but rarely surgical intervention. In contrast, those with CLI have an amputation rate approaching 12% at just 3 months,[4] and endoluminal or open surgical procedures are commonly indicated.

Symptoms may be related to degree and length of stenosis, or in the case of plaque rupture and thrombosis, they may relate to segmental arterial occlusion. The specific pathophysiology of PAD progression is associated with genetics, modifiable (eg, smoking) and nonmodifiable (eg, gender) risk factors, and age. As with so many diseases, risk factor reduction to stabilize mild disease (ie, claudication) is paramount.

DIAGNOSIS OF CLAUDICATION

An accurate history is essential as the initial phase of evaluation, as multiple etiologies exist for limb pain and fatigue (**Table 1**). Claudication presents as pain with ambulation/exertion in the calf, thigh, and/or buttock, depending on the location and extent of the arterial lesions.[2] The symptoms are reproducible with a given level of activity or distance, and they do not depend on position. The clinical presentation is not often classic, and claudication may coexist with other etiologies of limb pain. However, pain radiating down the back of the whole leg, or pain localized to major joints, is usually not vascular in origin. Multiple causes for limb pain often coexist in those with PAD given age and sometimes debility.[5]

Once a history suggestive of claudication has been elicited, noninvasive vascular studies should be obtained. The primary means of establishing the diagnosis of claudication uses ABIs.[6] This test can be done in any office with a hand-held Doppler, and is the occlusive systolic pressure of the posterior tibial and dorsalis pedis artery divided by the highest systolic arm pressure. An ABI of less than 0.9 at rest indicates a vascular origin to the patient's limb symptoms.[7] Although ABIs are sensitive for most patients with a diagnosis of PAD, patients with end-stage renal disease or longstanding diabetes may have medial calcification and noncompressible vessels, typically causing the ABI to be greater than 1.5. In these patients with invalid ABIs, toe brachial indices (TBIs) may be useful, as digital vessels are often preserved. If these values are abnormal, one can consider PAD as a source of claudication in these patients.

Table 1
Differential diagnosis of lower limb pain

Class	Pathophysiology	Distinguishing Features
Musculoskeletal	Osteoarthritis	Focal to joints, may be positional
	Myopathies	May have autoimmune markers and occur at rest
Neuropathic	Sensory neuropathy	Often position, burning in quality and not related to activity
	Lumbar radiculopathy	May be positional and related to a history of back pain
Venous	Venous claudication	Swelling, often described as bursting
Other	Includes chronic compartment syndrome	More often in athletes often young