Peripheral Arterial Disease: How to Diagnose, When to Refer

Peripheral arterial disease (PAD) affects 12% of the general population. How is it diagnosed and when do you refer your patients to a specialist?

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Atherosclerosis is a systemic condition that affects the peripheral arteries, the cerebral vasculature and the coronary arteries, to a greater or lesser extent. In a population of men and women over 62, 41% had more than one manifestation of atherosclerotic disease, and 9% had involvement of all three vascular beds. Peripheral arterial disease (PAD) has been found to affect 12% of the general population and its incidence increases with age. Of individuals over 70, 20% will be affected by this condition.

In spite of the large numbers of patients with this disease, PAD has been underdiagnosed and undertreated as a risk factor for mortality. Patients with PAD had an all-cause mortality rate of 4.8% per year, substantially greater than the 1.6% all-cause mortality faced by those without PAD. Cardiovascular mortality rates are five times higher in patients with PAD than in those without. The five-year and 10-year survival was significantly lower in patients with PAD, with approximately 40% and 55% mortality rates, respectively. However, the overall 10-year limb loss rate experienced by these patients is 11%. It is rather striking to realize that a patient who presents with non-limb threatening arterial insufficiency is at greater risk of dying than losing a limb in the next 10 years.

PAD is, by its very nature, a multi-faceted problem. It is imperative to seek out and accurately diagnose the presence of PAD. The patient is often more immediately concerned about the affected limb itself. Initial non-invasive testing can be undertaken to provide some early feedback with regard to the severity of the PAD. It is of paramount importance to educate both the medical institution and the patient about the systemic atherosclerotic problem.

Mrs. Munro’s Case

Mrs. Munro, 75, presents to your office complaining of progressive pain in her left foot, which is worse at night and better with her foot hanging off the side of the bed. She has had a long history of cramping in her left calf when walking, but she is now barely able to walk around her home. She has a 60 pack/year history of smoking. When she removes her stockings, you see ulcers on one of her toes (Figure 1).

What is the cause of these wounds, and what should the doctor’s next steps be?
How do I know it’s PAD?

PAD has a spectrum of presentation. It may be entirely asymptomatic, being identified as a reduction of peripheral pulses confirmed by ankle pressure testing during the course of physical examination. Patients may complain of mild leg symptoms, which they attributed to normal aging. They may have also purposefully or unconsciously reduced their activity levels to avoid these symptoms. Other patients may admit to some exercise-related leg discomfort, but find that it does not limit them in any way. Asymptomatic patients must not be dismissed as not requiring intervention. It is these patients, who are otherwise unaware of the development of atherosclerosis, who need careful assessment and counselling regarding their disease. They are also often the most difficult patients to convince of the need to alter their lifestyle and begin risk reduction therapy.

The second presentation is that of intermittent claudication, an exercise-related, reproducible, muscular leg pain, that is relieved with rest. It occurs as a result of an inability of the arterial system to provide adequate oxygen for muscular aerobic metabolism. Consequently, the muscles switch to anaerobic metabolism, producing lactic acid, which initiates the muscular burning or cramping pain typically described by patients.

A patient with non-limb threatening arterial insufficiency is at greater risk of dying than losing a limb.

About the author

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What can I do for the pain?

Although symptoms are brought on more quickly with increased levels of activity (such as increased walking pace, carrying heavy objects, or climbing inclines), patients with claudication pain should be reassured that the muscle cramping is not dangerous and they should be advised to walk and exercise as much as possible. This will improve the athletic training of their muscles, thereby improving walking distance and lessening the discomfort.

Most often, a single level of occlusion in the arterial tree brings on symptoms. Collateral circulation is adequate to maintain the metabolic requirements of resting tissue, but the collateral bed has a very limited ability to allow for the augmentation in flow that is necessary to properly oxygenate active muscle tissue (Figure 2).

It is important to differentiate this exercise-related pain to similar lower leg discomfort brought about by neurogenic causes, such as spinal stenosis or lumbosacral nerve root compression. In these cases, exercise may bring about the pain, but prolonged standing (or even sitting) may also cause the discomfort.

Frequently, there is day-to-day variability in the intensity and onset of the symptoms. Pain relief is often accomplished with positional changes, and...
complete relief is often not achieved. The very similar nature of the lower extremity symptoms makes it difficult to accurately diagnose the true cause (especially as there may be some degree of arterial insufficiency and many of these patients are at risk for developing both problems due to their age). In these cases, non-invasive exercise testing can be useful.

PAD may be so severe as to render even resting tissues inadequately perfused. Usually a second level of occlusion is present and it is the need to perfuse the limb through two collateral beds which results in a critical drop in the distal perfusion pressure. Consequently, ischemic neuritis ensues causing rest pain, described as a burning sensation that usually involves the distal forefoot and toes. The soft tissues may ulcerate spontaneously and are at risk of developing non-healing wounds due to the most minimal trauma. In cases of critical ischemia, the risk of limb loss is high and urgent evaluation is required.

**What are the symptoms and signs of critical arterial insufficiency?**

Patients with critical ischemia often have an antecedent history of claudication that has deteriorated to the point where their walking distance is very short. Their main complaint is usually the development of rest pain, with or without ulceration. This pain is worse with elevation and better with dependency, thought to be as a result of gravitational assistance with perfusion. Therefore, many of these patients will sleep sitting in a chair to limit their pain. When examining the limb, pay special attention to the characteristics of the ulcer and the surrounding tissues.

Arterial ulcers (Figure 3) occur at the most distal aspects of the extremity. They may also occur at sites of repetitive trauma, such as pressure or weight-bearing points. They have distinct margins and are often deep, described as “punched-out.” A central eschar of necrotic tissue may be present.

**If non-invasive testing is insufficient or if operative intervention is being considered, imaging of the vascular system is required.**

Figure 5. Exercise stress test graph demonstrating depression of ankle pressure with exertion.
The remaining limb also shows changes consistent with chronic ischemia. The skin is thin and shiny due to atrophy of the dermis. There is no hair growth as a result of follicle loss. The nails are often thickened because of the very slow metabolic turnover of the nail bed. With dependency of the limb there is a ruborous appearance to the foot and, with elevation, pallor. The foot is hyperesthetic and even the lightest touch can cause severe pain.

What tests are available?

Assessment of the peripheral arterial tree can be undertaken with both non-invasive and invasive tests. The first and easiest test is the calculation of the ankle-brachial index (ABI). Continuous wave Doppler assessment of the pedal and peroneal vessels is useful in establishing the presence of arterial flow in the named vessels at the level of the ankle. A standard blood pressure cuff is then inflated above the ankle until the arterial flow signal detected by the Doppler probe disappears (Figure 4). The cuff is slowly deflated until the signal reappears. This “opening pressure” is the systolic pressure. Doppler cannot record a diastolic pressure. The ankle pressure is then divided by the best brachial systolic pressure to get an ABI ratio. A normal ABI range is from 0.9 to 1.1. If a patient is identified as having an ABI of < 0.9, there should be a high index of suspicion for the presence of PAD. Although not absolute, and not indicative of total limb arterial blood flow, most critical ischemia occurs when the ABI drops below 0.3.7

The ABI does have some pitfalls, however. The most common is the presence of calcified tibial vessels, frequently occurring in patients with diabetes due to Mönckeberg’s sclerosis. The vessels may be rendered completely non-compressible or the opening pressure may be spur-
ously elevated due to the extra resistance to cuff occlusion imparted by the stiff arterial walls. Another difficulty with obtaining ankle pressure may be the presence of local, painful wounds that make patient co-operation problematic. Finally, the degree of perfusion may be so poor that no arterial signals are identifiable by Doppler.

Referral to a vascular laboratory for further, more sophisticated, non-invasive testing can be very useful. Exercise testing and performance of ABI assessments at rest can be useful in differentiating neurogenic from vasculogenic claudication. The ABI drops with exercise as a result of PAD. There is slow recovery, with the limb having better collateral circulation around the obstruction recovering faster. In neurogenic claudication, the ankle pressures should parallel the brachial pressure with no drop in the ABI (Figure 5).

Toe pressures can also be performed in the vascular laboratory. Digital pressures are especially useful in patients with diabetes with non-compressible arteries as the digital arteries are often spared calcification and give a truer assessment of the actual perfusion pressure of the foot. A normal toe has a toe-brachial index of > 0.75.

Ultimately, if non-invasive testing is insufficient, or if operative intervention is being considered, imaging of the vascular system is required. Space precludes a detailed discussion of the various modalities available, however, catheter-based angiography, though invasive, remains the gold standard at this time. Disadvantages of this test include puncture site problems, such as hematoma and pseudoaneurysm, as well as systemic complications, including renal dysfunction and contrast reactions.

Magnetic resonance angiography (Figure 6) is becoming more widely used in Canada for non-invasive imaging. Although prone to interpretation errors, it does not impart any nephrotoxic risk and vascular calcification does not affect the
imaging, as it does with computed tomography angiography. It is often an ideal method for imaging the diabetic arterial tree.

**When should I refer to a vascular surgeon?**

Many patients present to their primary care physician with complaints of leg pain. These complaints can be difficult to sort out. If there is evidence to suggest the presence of PAD as a cause, a vascular surgical opinion may be useful in trying to obtain an accurate diagnosis. If a patient with known claudicant begins to complain of a significant deterioration in their walking distance, it may portend advancement in their occlusive disease, with possible need for earlier intervention. Therefore, it is reasonable to obtain an assessment by a vascular surgeon.

Any patient presenting with symptoms and signs of critical arterial ischemia, with or without ulceration, requires urgent assessment by a vascular surgeon to determine if arterial reconstruction is feasible, and to limit the extent of tissue loss.  

**References**