Pedal Pulse as an Indicator of Coronary Disease

This underutilized test may provide critical insights into patients' health.

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Coronary artery disease is the leading cause of death in the US, with cerebrovascular accident being the third leading cause. Frequently, carotid bruits indicate carotid stenosis, but there are no clear physical signs of coronary disease. Atherosclerosis is a systemic disease, so physical findings in other vascular beds can predict that a patient is at a much higher risk of cardiovascular death. This is particularly important in patients with peripheral vascular disease, in whom diminished exercise capacity may limit the development of exertional angina, and in diabetic patients who often have faulty angina warning signs. A diminished foot pulse may be the only clue that a patient is at increased risk of cardiovascular death. There is a strong association among peripheral artery disease (PAD), coronary artery disease, carotid artery disease, and abdominal aortic aneurysm.[7]

There are more than 2 million Americans who have symptomatic PAD and many more remain asymptomatic.[1] A thorough cardiovascular examination is incomplete without assessing the pedal pulse, which provides a window view of overall cardiovascular integrity and health. The typical physical examination findings in patients with peripheral vascular disease include absent or diminished pulses, abnormal skin color, poor hair growth, and cool skin. The most reliable physical findings are diminished or absent pedal pulses.

Most of the fatal or disabling clinical events associated with PAD are attributed to the coronary vasculature regardless of whether a diagnosis of coronary artery disease has already been established.[2] Absent dorsalis pedis and posterior tibial pulses have a sensitivity and specificity of 63% and 99%, repectively.[3] Once PAD is suspected, patients are normally screened with ankle-brachial index (ABI) measurements of one or both extremities.

BACKGROUND

In 1950, Winsor was first to use ABI measurements in patients with PAD.[4] The ABI is a simple and inexpensive test that can identify patients with PAD by determining the ratio of systolic blood pressure at the ankle arteries relative to that at the brachial arteries if abnormal. ABI is a surrogate marker of atherosclerosis and is a predictor of future cardiovascular disease and all-cause mortality. This clinical test is underutilized even though it requires a minimal use of resources and investment—only a simple blood pressure cuff and a handheld continuous-wave 5- to 10-MHz Doppler probe is needed. Measurements for the ABI should be obtained after the patient has been supine for 5 to 10 minutes. The test requires that the systolic blood pressure be recorded in both brachial arteries and in both dorsalis pedis and posterior tibial arteries. The ABI is calculated for each leg by dividing the highest ankle systolic pressure by the highest brachial systolic pressure. The ankle pressure will exceed the brachial pressure by 10 to 15 mm Hg in healthy individuals as a result of higher peripheral resistance at the ankles.

ABI INTERPRETATION

According to the published practice guidelines for PAD management from the American College of Cardiology and the American Heart Association, ABI ratios are interpreted as follows:

- The patient is diagnosed with PAD when the ABI is ≤ 0.9.[5,6] An ABI value of 0.91 to 0.99 is considered "borderline." PAD is graded as mild to moderate if the ABI is between 0.4 and 0.9, and an ABI < 0.4 is suggestive of severe PAD.[7] An ABI value > 1.4 is also considered abnormal and is suggestive of noncompressible vessels.
- An ABI of 0.9 or less has a sensitivity of 95% and a specificity of 100%, relative to contrast angiography, for detecting a stenotic lesion of at least 50% in the limb.[8]

DATA AND REPORTED OUTCOMES

Robust data support that PAD directly relates to cardiac and cerebrovascular morbidity and mortality independent of other adjusted risk factors. Having a low ABI ratio is an independent risk factor for cardiovascular disease, including fatal and nonfatal complications. The predictive value

of ABI in cardiovascular morbidity and mortality is similar to that of traditional Framingham risk factors.[2] Several epidemiologic studies have reported up to four-fold increased rates of cardiovascular disease and mortality with abnormal ABI.[3,5,6,9-12]

McDermott et al[13] found that an ABI of 0.9 to 0.99 was associated with a significantly higher prevalence of subclinical atherosclerosis (increased carotid intima-media thickness and coronary artery calcium assessed by computed tomography) when compared with a normal ABI (defined as 1.1–1.29) in both men and women.

Vogt et al[14] reported that the mortality rates from atherosclerotic heart disease doubled (risk ratio, 2; 95% confidence interval, 1.4–2.9) with each 0.5 unit drop in the ABI. An ABI ratio of < 0.9 has been associated with up to a three-fold relative increase in all-cause and cardiac mortality in both men and women.[3,15]

Vessels are noncompressible when there is significant medial artery calcification. This is most commonly seen in diabetic patients, elderly individuals, patients with chronic renal failure who are on dialysis, and patients receiving chronic steroid therapy. In these patients, an elevated ABI of 1.4 is also a predictor for an increase in cardiovascular and all-cause mortality.[16]

A recent meta-analysis found that ABI is an independent risk factor for cardiovascular mortality alongside the traditional Framingham risk factors. Having an ABI of 0.9 or less resulted in a doubling of cardiovascular mortality and morbidity across all of the Framingham risk categories.[17]

The rates of cardiovascular disease and mortality associated with different levels of ABI were investigated by O'Hare et al[5] in more than 5,000 patients. The study found that patients with an ABI < 0.6 were consistently associated with increased cardiovascular events, cardiovascular mortality, and all-cause mortality.

In the HOPE trial, Ostergren et al found that the rates of cardiovascular disease, cardiovascular death, and all-cause mortality in subjects with an ABI > 0.9 were 10.1%, 5.3%, and 8.8%, respectively, compared to 13.7%, 8.6%, and 12.8% in the 0.6 to 0.9 ABI group and 13.4%, 9.4%, and 14.7% in the < 0.6 ABI group.[12]

Atherosclerosis is a systemic disease, and if present in the peripheral arteries, there is a great likelihood that other parts of the arterial tree are also affected. Patients with PAD have more than a sixfold increase in the risk of death from coronary artery disease when compared to patients without the disease. Even asymptomatic patients with PAD have an increased risk of future cardiac and cerebrovascular events, as well as being six times more likely to die within 10 years when compared to healthy individuals.[1]

CONCLUSION

Signs of PAD may suggest an impending or coexistent atherosclerotic process, so careful examination of arterial circulation by evaluating peripheral pulses is important. Palpation of the pedal pulses and measurement of ABIs is highly underutilized, and it should be part of a comprehensive cardiovascular evaluation. Although a normal ABI may be found with advanced disease, an abnormal ABI is almost always associated with significant peripheral atherosclerotic disease.

Asymptomatic patients with decreased peripheral pulses do not require an invasive interventional procedure but should have a medical intervention. These patients should, at a minimum, be counseled to stop smoking, and hypertension and dyslipidemia should be controlled. Careful examination for carotid bruits and abdominal bruits should be performed. Consideration of elective evaluation of myocardial ischemia is warranted in patients with claudication, which may limit them from exercise and provoke angina or diabetes. All patients who have had peripheral interventional procedures or open vascular surgery should have aggressive evaluation and therapy aimed at limiting overall cardiovascular morbidity and mortality. ■

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