EDITORIAL COMMENT

Where There Is Smoke, There Is Fire*

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The ankle-brachial index (ABI), a ratio of systolic blood pressure measured at the ankle and the brachial artery, is a simple noninvasive screening test used to diagnose peripheral arterial disease (PAD). PAD affects over 12 million people in the United States and its prevalence increases with increasing age (1,2). Patients with an abnormal ABI, defined as ≤0.90 or ≥1.40, are at increased risk of having lower-extremity PAD independent of classic symptoms. The current method of measuring ABI, called the high ankle pressure (HAP) method, uses the systolic pressures in the dorsalis pedis and posterior tibial arteries of both legs. The higher values of these ankle pressures for each respective lower extremity are divided by the higher brachial systolic pressure. Interval ABI results can be used to monitor the efficacy of revascularization procedures of lower extremities (3). The ABI has also been used to predict the prognosis of limb salvage and wound healing (4). There is little data in the literature documenting the incidence and clinical implication of newly diagnosed abnormal ABI in patients found to have severe coronary artery disease (CAD) by coronary angiography.

In this issue of JACC: Cardiovascular Interventions, Lee et al. (5) report on an observational cohort and the prevalence of abnormal ABI in patients with no prior history of PAD undergoing cardiac catheterization and the increased risk in this patient population when followed for 3 years. Abnormal ABI was defined as ≤0.9 or ≥1.4. The primary endpoint was a composite of death, myocardial infarction, and stroke over 3 years. Of the 2,424 patients with at least 1 significant stenosis (≥50%) in a major epicardial coronary artery, 385 (15.9%) had abnormal ABI. During a median follow-up of 986 days, the 3-year major adverse event rate was significantly higher in patients with abnormal ABI (15.7% vs. 3.3%, p < 0.001) as compared with those with normal values. One of the seminal issues that this paper has confirmed is that when patients with CAD are ruled out by a dedicated claudication questionnaire (6), there is still a high prevalence of “silent” PAD: 15.3%. Atherosclerosis is a systemic disease, and it is imperative that ABI is not reserved for patients with symptoms only. Otherwise, a very simple, no-risk test that may help with risk assessment besides diagnosis will be underutilized.

The methodology for measuring the ABI using the higher ankle pressure was used and the cutoff was <0.9 or >1.4. However, this method may underestimate the diagnosis of PAD as patients with only below-the-knee disease affecting 1 of the tibial vessels will not be identified, thus missing the diagnosis of PAD. In addition, McDermott et al. (7) have shown very succinctly that patients develop PAD when the ABI starts dropping below 1.0 and the risk for adverse outcomes increases. It would be interesting to see how patients who had ABI between 0.9 and 0.99 fared. It is also well recognized that some patients with PAD may have normal resting ABI of 0.91 or greater, but exercising these patients may show the abnormal ABI (8). In this paper, exercise ABI was not done if the resting ABI was normal.

One of the major limitations that Lee et al. (5) have mentioned is the selection bias: They did not take all patients undergoing cardiac catheterization. In studies where all comers for cardiac catheterization have had an ABI, the prevalence of abnormal ABI may be 25% to 35.8% depending on how the ABI is reported (9). We and others have reported that the diagnosis of PAD should be based on using the lower ankle pressure divided by the higher brachial pressure as this increases the sensitivity and accuracy of the test. The sensitivity of the HAP and the LAP ABI was 83% and 70%, respectively, and the specificity of the HAP and the LAP method was 83% and 64%, respectively (10). Furthermore, the overall accuracy of the LAP ABI and HAP ABI was 80% and 72%, respectively. These findings suggest modification of the existing method of calculating the ABI. With its greater sensitivity and accuracy, the LAP ABI may result in earlier initiation of risk factor modification and primary intervention strategies in patients with PAD. In addition, as Lee et al. (5) have shown using the HAP ABI, abnormal ABI in patients with significant CAD may predict 3-year adverse outcomes and would be enhanced with the more accurate and sensitive LAP ABI. Secondary prevention measures may be initiated in these patients who would be otherwise missed by the HAP ABI as studies have shown the LAP ABI also predicts increased risk (9).

Lee et al. (5) also demonstrated that over a 3-year period patients with abnormal ABI had more events than those with normal ABI. This was significant when death,
myocardial infarction, and stroke were taken cumulatively, but individually only stroke was significant. One reason may be the number of patients studied and/or the period of 3 years, which may be too short to show any significant difference. Mortality has been shown to increase with an abnormal ABI when patients are followed for longer periods (11). Epidemiologic studies have reported up to a 4-fold increase in cardiovascular disease and mortality with abnormal ABI (12).

Lee et al. (5) did a meticulous data collection and diligent follow-up. Asymptomatic patients for PAD who are diagnosed with CAD, at least 15.9%, if not more will have PAD, which carries a poor prognosis when compared with that for patients with ABI 0.91 to 1.40. This should be an impetus for cardiologists to check ABI especially as it is a simple way of identifying high-risk patients who have CAD that may need more intensive risk factor modification and follow-up. Future studies may look into patients with normal fractional flow reserve who may be at a higher risk when the ABI is abnormal, association of vulnerable plaque to ABI, and development of methodologies for assessing ABI that are less operator-dependent so its utility may be increased.

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