CLINICS

READERS OPINION

Ankle brachial index in coronary artery disease – Author's reply

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We would like to thank Balta and colleagues for their consideration of our article and the editorial board for providing an opportunity to clarify some issues.

Ankle–brachial index (ABI) measurement may be influenced by age, body weight, race and anatomic variations of the lower limb arteries (1). After the measurement has been completed, the pulse locations of the lower limbs are found by palpation. However, in 3.1% to 32.5% of the normal population, the dorsalis pedis artery may not be palpable. In such cases, Doppler ultrasound allows the dorsalis pedis pulse to be located with great accuracy. The congenital absence of the dorsalis pedis pulse occurs in only 2% of the normal population when evaluated by Doppler. Considering this rarity (0.18%), when the dorsalis pedis pulse is absent, we use the pulse of the posterior tibial artery to calculate the ABI. Therefore, when the pulse of the posterior tibial artery is not detected by Doppler, a diagnosis of peripheral artery disease is likely (2). According to the American Heart Association guidelines, the ABI in our study was calculated for each leg by dividing the greater value of the posterior tibial or dorsalis pedis pressure by the greater value of the right or left arm systolic blood pressure (according to Doppler); as a marker of cardiovascular risk, the lowest value obtained for the lower limbs should be used, thereby increasing the accuracy of this method (1).

In our series, each patients with an ABI >1.3 was considered to have no compressible arteries (more common in diabetic patients) and was excluded. Although ABI analysis may be imprecise in these cases, ''The Strong Heart Study'' demonstrated that patients with ABI measurements >1.4 had increased cardiovascular mortality, as did the patients with ABI measurements <0.9 in the same study. An ABI >1.4 is also considered to be a strong predictor of cardiovascular mortality (3).

The effect of antihypertensive drugs and statins on ABI has been reported by some authors. Ichihara et al. studied the effect of statins in patients with dyslipidemia and uncontrolled hypertension. The authors observed long-term improvement in aortic compliance with the use of fluvastatin, with no changes in the blood pressure and ABI values of these patients (4). Similarly, a recent systematic review did not demonstrate the influence of various antihypertensive drugs on ABI and the progression of peripheral arterial disease in hypertensive patients (5). In our study, 47% of the patients had ABI measurements <0.9 and all patients were prescribed statins, angiotensin conversive enzyme inhibitors, angiotensin receptor blockers, beta blockers and antiplatelet agents in similar proportions. Therefore, if the effects of these medications on ABI were important, we would likely find that ABI decreased in a greater proportion of patients, which did not occur.

Finally, the correlation between ABI and the severity of coronary artery disease has been demonstrated in several studies that used the number of coronary arteries involved as the criteria for the severity of coronary artery disease (CAD) (6,7). At our institution, an inverse correlation between an ABI measurement of <0.9 and the Syntax score has been observed. In an evaluation of elderly patients with CAD diagnosed by coronary angiography, Falcão et al. have demonstrated that, although an ABI <0.9 was associated with the complexity and extent of coronary disease (a higher proportion of patients with lesions B2 and C), the Syntax average score and the proportion of patients with Syntax scores >16 were similar, regardless of ABI. These findings indicate the need for more detailed studies (8).

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