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Low Ankle Brachial Indices Reflect Left Ventricular Systolic Dysfunction: A Potential Explanation for Co-Existing Heart Failure and Peripheral Artery Disease?

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Abstract

Peripheral artery disease (PAD) is a growing health concern in the United States due to its rising prevalence, negative impact on functional disability and increased risk for cardiovascular and cerebrovascular events. Less widely appreciated is that PAD is associated with nearly double the prevalence of congestive heart failure (CHF) and that 17% of heart failure patients have co-existing PAD. Multiple reasons may account for co-existing PAD and CHF. The prevalence of each disorder rises with age and they share common risk factors as well as metabolic disturbances. In addition, while ankle brachial index (ABI) testing has long been the primary modality used to detect PAD, there is accumulating evidence that low ABI values may reflect left ventricular (LV) systolic dysfunction and reduced ejection fraction (EF). The low ABI-low EF relation appears independent of CAD and may in part explain the prognostic value of low ABI for predicting increased mortality. Accordingly ABI values should be interpreted in the context of LV systolic function.

Keywords: Ankle brachial index; Peripheral artery disease; Congestive heart failure; Left ventricular function

Introduction

Peripheral artery disease (PAD) is a growing health concern in the United States due its rising prevalence, its impact on functional disability and increased risk for cardiovascular and cerebrovascular events. A recent review by Basgoz et al. [1] summarized the importance of screening for PAD and its association with coronary artery disease (CAD). Importantly, the authors delved into the less widely appreciated association between PAD and congestive heart failure (CHF). The presence of PAD has been found associated with nearly a doubling the prevalence of CHF [2]. Conversely, an earlier study showed a high prevalence of PAD in heart failure patients as 17.1% of CHF patients with low left ventricular (LV) ejection fraction (EF) ≤40% enrolled in disease management programs in Louisiana and Florida had PAD [3]. In another study of more than 18,000 subjects, aimed at deriving a clinical risk score derived to predict PAD, heart failure conferred a higher likelihood of a low ABI among the general population [4].

These prior studies measured the ankle brachial systolic blood pressure index (ABI), which is the most common method used to assess for PAD [5-7]. While multiple reasons may account for co-existing PAD and CHF including age dependence, common risk factors and similar metabolic disturbances, ABI determination may play a role as well. The ABI is essentially ratio of lower extremity systolic blood pressure (BP) to brachial artery BP measured by Doppler or sphygmomanometry. Normal values range from 1.2 to 1.4, indicating that systolic BP is normally higher in the lower than the upper extremities. Systolic BP increases with increasing distance from the heart as the arterial pressure waveform changes shape from the central aorta to the periphery due to arterial pressure amplification resulting from vascular branching and tapering as well as peripheral arterial wave reflections [8-11].

Values of ABI <0.9 in either lower extremity is considered evidence of PAD and numerous studies have found low ABI values

to be an independent predictor of cardiovascular events including myocardial infarction, stroke and death [12,13]. More than 2 decades ago, Fine et al first suggested that lowered ABI values may reflect LV function upon finding LV systolic dysfunction present in 20% of diabetic patients found to have low ABI [14]. In 2010, our group hypothesized that LV systolic dysfunction would attenuate BP amplification and we demonstrated that ABI values were directly correlated with LVEF in patients suspected of PAD [15]. The ABI-LVEF relation appeared independent of the presence of CAD, suggesting that coexisting CAD and PAD did not account for the association. Since then, other studies have similarly found ABI values related to LVEF in a variety of patients including: elderly with ischemic heart disease, those with and without diabetes, atrial fibrillation and hemodialysis [16-21].

While the exact mechanism(s) underlying this relation remains unknown, similar correlations between LVEF and ABI were found in patients with and without significant CAD and higher LVEF was associated with higher ABI values [15]. Moreover, we observed a strong ABI-LVEF correlation observed in a species of non-human primates that generally do not develop atherosclerosis [22]. These findings would implicate that LV systolic function directly affects the step up in systolic BP from upper to lower extremities, known as pressure amplification. Given that LV systolic dysfunction is often accompanied by increased arterial stiffness and lower arterial pressure wave reflections and that these arterial properties may influence systolic BP amplification along the arterial tree, dampened arterial pressure wave reflections could be expected to lower ABI values [23,24]. Whether LVEF simply reflects lowered LV stroke volume or a diminished contractile force that in turn dampens SBP amplification that normally occurs along the arterial tree because of arterial branching and tapering merits further study.

Moreover, lower ABI values have also been found to be associated with cardiac structural abnormalities including: LV wall thickening and cavity dilation that predispose to LV dysfunction and CHF as well as to left atrial dilation [24,25]. Similar findings have been reported for patients with borderline low ABI values [27]. Of note, ABI values were found to increase in response to low dose dobutamine, which predominantly increases ventricular contractility, but to decrease at higher doses sufficient to cause systemic vasodilation in patients without PAD [28].

Conclusion

Therefore, despite growing evidence relating ABI to LV structure and function, low ABI values are generally deemed to be indicative of PAD. Depressed LV function has been seldom considered. These studies suggest ABI values to reflect functional and structural properties related to ventriculo-arterial coupling [29]. We believe that there is ample evidence to support that low ABI values may not reflect PAD but rather LV systolic dysfunction. The predictive value of low ABI values for cardiovascular events and mortality may in part relate to depressed LV systolic function rather than atherosclerosis alone. Accordingly, ABI values should be interpreted in the context of LV systolic function.

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