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Editorial

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Cardio-Ankle Vascular Index and Atrial Remodeling for Atrial Fibrillation

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Atrial fibrillation (AF) is the most common chronic arrhythmia in adults which is associated with an increased risk of mortality from cardiovascular events. Hypertension and elevated blood pressure are important risk factors for AF. For understanding the causal relationship between hypertension and AF, previous studies have evaluated the relationship between AF and markers of arterial stiffness, including pulse wave velocity and augmentation index[1, 2]. An analysis from the data of the Rotterdam Study showed that a higher carotid–femoral pulse wave velocity was associated with higher AF incidence[1]. Another study from the Framingham Heart Study demonstrated that a higher augmentation index, but not a higher carotid–femoral pulse wave velocity, was associated with an increased risk of AF incidence[2]. Thus, particular attention has been paid to the relationship between vascular functions and AF incidence. However, because blood pressure is a critical confounding factor between arterial stiffness and atrial remodeling, the contribution of arterial stiffness in relation to AF incidence remains unclear.

The cardio-ankle vascular index (CAVI) is a marker of arterial stiffness from the origin of the ascending aorta to the ankle[3]. The principle of CAVI is based on the stiffness parameter β; therefore, blood pressure does not theoretically affect this at the time of arterial stiffness examination. Several studies have demonstrated that CAVI is associated with target organ damage, such as the presence of left ventricular hypertrophy, coronary artery disease, and stroke[4]. Furthermore, studies have also reported an association between a greater CAVI and a high incidence of cardiovascular events in patients with diabetes mellitus, obesity, and several cardiovascular risk factors[5]. CAVI is an established marker of vascular function independent of blood pressure during testing, which is an ideal characteristic of arterial stiffness.

In this issue of the Journal of Atherosclerosis and Thrombosis, Nakamura *et al.* examined the relationship between CAVI and atrial remodeling in association with AF in 213 consecutive patients with AF undergoing ablation[6]. Computed tomography was used to determine the atrial volume, and a three-dimensional electromapping system was used to evaluate atrial electrical remodeling. They clearly demonstrated that the increase in CAVI was closely associated with a greater left atrial volume index and a greater right atrial volume index. CAVI was also involved in bi-atrial electrical remodeling. A marked increase in CAVI was noted in association with long-standing AF compared with persistent and paroxysmal AF. Finally, the authors showed high reproducibility for CAVI obtained from patients with sinus rhythm and AF rhythm.

The left atrial volume index is a robust and independent predictor of AF incidence. In this regard, this study provides valuable information on the underlying mechanism linking arterial stiffness shown by CAVI and AF incidence. This study has some similarities with other published studies. A cross-sectional study showed that CAVI was positively correlated with left atrial diameter in patients with paroxysmal AF[7]. Another case-control study showed an association between increased carotid–femoral pulse wave velocity and AF incidence, and carotid–femoral pulse wave velocity was higher in persistent more so than in patients with paroxysmal AF[8]. Of note, this study reported that increased CAVI was associated with both left and right atrial remodeling. The occurrence of functional mitral and tricuspid regurgitation in patients with AF that may likely be

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due to atrial enlargement and ventricular dysfunction has received recent literature attention. Further investigation of the mechanistic link between arterial stiffness and bi-atrial remodeling would be informative for understanding the pathophysiology of these functional regurgitation pathologies that cause heart failure with AF. Further, this study provided rigorous data on atrial electrical findings at catheter ablation for evaluating the relationship with arterial stiffness. Recently, CAVI was reported to be associated with left atrial phasic function determined by speckle-tracking echocardiography[9]. These findings suggest that CAVI has the potential to detect early left atrial functional remodeling before enlargement.

The authors also noted several limitations of the study[6]. The observational nature precludes any causal inference. They evaluated a middle-aged and older cohort and their results may therefore not be generalizable to younger individuals. Although good reproducibility was observed in this study, measuring CAVI during an AF rhythm is still challenging.

Taken together with previous literature, the data of this study contribute to an evolving mechanistic framework for how people with increased arterial stiffness develop an increased risk of developing AF. It would be of great interest in further studies to determine whether treatments for improving CAVI prevent the incidence and recurrence of AF.

Conflict of Interest

The author declares no conflict of interest.

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