Aortic strain in hypertensive patients, are we ready for it?

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The elastic properties of the aorta incorporate both the property of dilating by increasing pressure in systole, and the property of recoiling slowly to its initial shape when blood pressure falls in diastole. Aortic stiffness describes the elastic resistance that the aorta sets against its distension and its increase is associated with increased risk of cardiovascular morbidity and mortality [1].

Aortic remodeling in hypertensive patients has been demonstrated to be one of the main expression of hypertensive mediated target organ damage, which can lead to the development of aortic dilatation over time [2,3].

Recently, the introduction of aortic strain analysis using speckle-tracking echocardiography (STE) has provided a new tool to identify possible functional abnormalities of ascending aorta [4].

In particular, using STE deformation stiffness of ascending aorta can be calculated using B2 stiffness index, which represents an index of local aortic stiffness. Previous studies have demonstrated that dilatation of ascending aorta is associated with reduced strain, distensibility and increased stiffness [5].

In this issue of Journal of Hypertension, Cesareo and colleagues analyzed local transverse aortic strain in a population of patients with arterial hypertension [6]. These authors clearly demonstrated that increased local aortic stiffness, evaluated as beta-stiffness index, is mainly associated with ascending aorta dilatation and more prevalent cardiac target organ damage, strongly suggesting that the functional abnormalities in ascending aorta may reflect a higher hemodynamic burden in hypertensive patients [6]. Data from animal studies suggest that the degree of mechano-mediated remodeling of the aorta might be partially due to dysfunction of smooth muscular cells with a specific biomechanical phenotype, characterized by a grossly maladaptive thickening and thus structural stiffening of the wall [7]. In this regard, early identification of abnormal aortic deformation with STE might be a key option for personalized pharmacological approach.

Recent studies involving mainly abdominal aorta have identified STE as an important diagnostic tool for examining biomechanical properties of the aorta and correlation to disease states [8,9]. In particular, evidence indicates that aortic compliance is reduced in dilated/aneurysmal aortas compared to normal controls, in line with results from Cesareo et al. in ascending aorta [6]. Strain analysis of ascending aorta represents a very sensible marker of atherosclerosis. Population-based study has demonstrated a strong association of global circumferential ascending aortic strain with prevalent coronary artery disease and atherosclerosis [10]. This confirms its potential clinical application in the management of patients with cardiovascular diseases.

The main aspect to be clarified is the relationship between increased aortic stiffness and its dilatation. Whether the increased stiffness that predisposes to the dilatation of the aorta or the dilatations per se that reduced the possibility of distension during systole needs to be better clarified by future prospective studies with a specific focus on the main determinant of aortic remodeling.

For routine use of aortic STE, some major limitations need to be overcome: normal threshold values and outcome-related study are lacking. Additionally, STE relies on good acoustic windows in order to adequately analyze all segments of the aorta, which is often not possible by the standard echo protocol as demonstrated by the high drop-out rate of the current study for not suboptimal images (32%). The presence of atherosclerotic plaque in the aorta can cause artifact, which can interfere with the speckle-tracking algorithm. Despite good inter- and intraobserver variability, demonstrated by Cesareo et al., aortic STE is operator dependent, which might limit its wide use in clinical practice. Finally, assessment of the ascending aorta in the region visualized by echo may not be representative of the entire thoracic aorta.
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Conflicts of interest
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REFERENCES