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Extracellular Volume and Cardiac Mechanics: Have We Found a Missing Puzzle Piece?

We have read with great interest the recent report regarding increased cardiac extracellular volume (ECV) and impaired left ventricular (LV) mechanics in hypertensive patients (1). The study was innovative, and it may provide answers to many questions about left ventricular hypertrophy (LVH) and LV mechanics in hypertension. There are several important topics that deserve comment.

The investigators indicated that a small sample size was an obstacle for the evaluation of different effects: age, sex, diabetes, hypertension duration, and antihypertensive treatment. However, it would be useful to present at least the data regarding the use of medications, especially those that block the renin-angiotensin-aldosterone system or sympathetic nervous system. Cardiac extracellular matrix is significantly influenced by these biohumoral systems, so it is quite reasonable to hypothesize that these antihypertensive drugs influence ECV. In the present study, Kuruvilla et al. (1) reported that the non-LVH patients had significantly lower blood pressure levels than the LVH subjects. It is possible that this group had lower ECV because these patients were more often treated with renin-angiotensin-aldosterone system and sympathetic nervous system blockers.

Interestingly, the investigators did not provide data on the correlation between blood pressure level and ECV or native T1, a measurement of myocardial fibrosis. These results would be of interest because blood pressure could be a significant confounding variable in the relationship between ECV and LVH or between ECV and LV mechanics.

The investigators are not completely clear in terms of exclusion criteria. It seems that hypertensive patients with some common comorbidities, such as diabetes, obesity, and metabolic syndrome, were not excluded from the study. Investigations have previously shown that these risk factors are associated with cardiac extracellular matrix (2), and our study group demonstrated that these cardiovascular risk factors were associated with impairment of LV mechanics and increased LV mass (3). Therefore, inclusion of these risk factors certainly affects the relationship between ECV and LV mechanics. It would be useful to compare the levels of glucose and lipids, as well as body mass index, among the groups.

The investigators included patients with LV ejection fractions >45%, but they did not provide values of ejection fraction in different groups. Previous investigations have shown a strong correlation between LV mechanics and LV ejection fraction in hypertensive populations. Collins et al. (4) recently reported that ECV fraction was more closely associated with altered regional LV velocities than LV ejection fraction in patients with preserved ejection fraction, while Kuruvilla et al. (1) in the present study reveal the association between ECV and LV mechanics, without determination of the relation between ECV and LV ejection fraction. These findings may have opened a new era of LV function evaluation. Can we overcome LV ejection fraction in modern cardiac imaging?

Additionally, it would be interesting to investigate ECV in patients with different LVH patterns, concentric and eccentric. Previous studies have demonstrated significantly decreased 2-dimensional LV longitudinal, circumferential, and radial strain in hypertensive patients with concentric LVH compared with subjects with eccentric hypertrophy (5). The findings of the present study would potentially explain this difference.

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