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Letter to the Editor

# Assessment of Strain Changes in the Triple Layers of Left Ventricle in Normotensive Versus Hypertensive Patients

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The phenomenon of left ventricular remodeling caused by hypertension is a well-known affair that can be evaluated through echocardiography techniques. In recent decade, modern echocardiographic methods such as speckle tracking echocardiography have been used to evaluate left ventricular mechanical changes in hypertensive conditions. The evaluation of the strain index as an important part of the evaluation of the ventricular wall in various ventricular layers has been highly regarded in hypertensive patients. In this regard, the left global ventricular longitudinal strain (GLS) especially in the three ventricular wall layers can strongly predict mortality and morbidity of cardiovascular disease. Given that hypertensive disease is one of the most important risk factors for the development of heart failure and systolic and diastolic cardiovascular dysfunction, even in cases with preserved left ventricular ejection fraction, the exact determination of ventricular strain changes in various layers in hypertensive states would be beneficial for preventing hypertensive cardiomyopathy. In a cross-sectional study, we assessed strain changes in the triple layers of the ventricular wall in hypertensive and normotensive individuals with presence or absence of left ventricular diastolic dysfunction. In this study, both global longitudinal strain (GLS) and global circumferential strain (GCS) indices in endocardial, myocardial and epicardial layers were evaluated. We also considered the effect of diastolic ventricular dysfunction as an important confounder with the effect of hypertension on the ventricular strain. An important finding of this study was the significant effect of hypertension on reducing GLS and GCS in mid myocardial and epicardial layers in the presence of left ventricular diastolic dysfunction. At first, in case of hypertension, the presence of diastolic dysfunction in reducing the strain of the left ventricular wall seems to be necessary as a trigger factor; thus in hypertensive cases with preserved diastolic function, the strain change in left ventricular wall may not be evident. Secondly, reduction in the ventricular wall strain in the endocardial layer was not revealed but it seems that the exacerbation of uncontrolled hypertension may also lead to endocardial involvement as shown by prior studies. In total, proper control of blood pressure in patients with a history of hypertension, especially in the field of left ventricular diastolic dysfunction will improve the function of the left ventricular wall and thus improve the prognosis of these patients.

Disturbance in left ventricle wall layers in the field of hypertension has been studied and confirmed in several studies, although the effective role of diastolic dysfunction has been studied less. For instance, in a study by Tadic et al., GLS was significantly lower in hypertensive than in normotensive patients (1). In Navarini et al study, although no difference was found in left ventricular volume or ejection fraction, both GLS and GCS reduced significantly in hypertensive status (2). In a study by Craciunescu et al, LV mass was higher and both the GLS and GCS parameters were significantly lower in the uncontrolled hypertension group than in patients with controlled hypertension (3). In a study by Nagata et al., the values of GLS and GCS in the endocardial layer were higher than the other layers, and this could justify the preservation of the strain in the field of hypertension in the endocardial layer (4). Finally, in a study by Sharif et al, patients with diastolic dysfunction experienced a relative decrease in GLS in all three layers of myocardial infarction, compared with those without diastolic dysfunction, which was consistent with our findings (5). What can be emphasized as the final result is that prolonged and uncontrolled hypertension with an effect on cell growth as well as cardiomyocyte fibrosis through the secretion of inflammatory and growth factors leading left ventricular hypertrophy can reduce strain in various ventricular wall layers particularly in myocardial and epicardial layers. Of course, the role of the presence of diastolic ventricular dysfunction is highly indicative in these changes.

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