Left ventricular global longitudinal strain by speckle tracking echocardiography in hypertension: a mini-review

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Abstract. The aim of this mini-review is to report current knowledge about left ventricular systolic function assessed by global longitudinal strain in hypertensive patients with and without left ventricular hypertrophy, that is, stage A and B heart failure. We conducted a literature search through PubMed, Web of science and Cochrane Library by using terms such as myocardial strain, speckle tracking echocardiography, systolic dysfunction, left ventricular hypertrophy, left ventricular geometry, essential hypertension. We identified 2 meta-analyses including 30 studies, 1 review including 8 studies and 5 other studies. The evaluation of published studies suggests that global longitudinal strain is significantly reduced in hypertensive patients than in normotensive subjects, despite normal ejection fraction. This alteration may be present in both patients with and without left ventricular hypertrophy, though it is more relevant in those with left ventricular hypertrophy. Global longitudinal strain is a more sensitive marker for early abnormalities of left ventricular function and can be of greater help than conventional methods in predicting hypertensive disease progression and outcomes. However, the prevalence of left ventricular longitudinal dysfunction in hypertension, its predicting factors and its clinical relevance are not yet completely clear. Thus, other studies are needed to shed further light on this topic.

Keywords: hypertension, left ventricular hypertrophy, global longitudinal strain.

INTRODUCTION

Hypertension is one of the most important factors for the development of heart failure (HF) (Rapsomanikis et al, Williams et al) which is becoming a major public health problem (Roger VL, Ponikowski et al). The population attributable risk of hypertension for HF has been reported to be as high as that of coronary artery disease (Dunlay et al).

According to the American College of Cardiology/American Heart Association guidelines, HF can be divided into different stages (Hunt et al).
Stage A includes patients at high risk for HF (for example hypertension, atherosclerotic disease, diabetes, obesity) but without structural heart disease or signs and symptoms of HF. Stage B includes patients with structural heart disease but without signs or symptoms of HF. Stage C includes symptomatic patients and Stage D includes those with refractory HF.

Historically, left ventricular (LV) systolic function has been assessed by ejection fraction (EF), but this index has some limitations. Strain and strain rate are promising technologies for the evaluation of cardiac function (Geyer et al, Voigt et al). Speckle tracking echocardiography (STE) is now the preferred method for strain measurements and global longitudinal strain (GLS) is the most frequent reported measure (Geyer et al, Voigt et al). Some studies have shown a reduction in myocardial contractile function, despite normal EF and absence of symptoms, in various cardiovascular disorders and measurement of GLS has been reported to provide incremental prognostic information (Stanton et al, Lee et al, Kuznetsova).

Thus, the use of GLS in the evaluation of cardiac function could be helpful in identifying Stage A and B patients at higher risk to develop clinical HF.

The aim of this mini-review is to report current knowledge about LV systolic function as assessed by GLS STE in hypertensive patients with and without LV structural alterations, that is, stage A and B HF, respectively.

METHODS

Search Strategy, inclusion criteria and study selection

We conducted a literature search through PubMed, Web of science and Cochrane Library up to June 28, 2022. The terms used to identify studies were myocardial strain, speckle tracking echocardiography, systolic dysfunction, left ventricular hypertrophy, left ventricular geometry, essential hypertension. Inclusion criteria were: full articles published in peer-reviewed journals; 2D STE echocardiographic studies assessing LV GLS in patients with hypertension with and without LV hypertrophy. Among published studies, we identified 2 recent meta-analyses (Tadic et al a, Tadic et al b), 1 recent review (Oh JK, Park JH) and 5 other studies (Imbalzano et al, Soufi Taleb Bendiab et al, Qingfeng et al, Salas-Pacheco et al, Takahashi et al). A study showed that, beyond LVH, the duration of hypertension, diabetes and increased filling pressures were associated with GLS reduction (Soufi Taleb Bendiab et al). Another study reported that patients with 3 or 4 risk factors, who also had higher LV mass, showed significantly reduced GLS (Takahashi et al).

RESULTS

In a meta-analysis (Tadic et al a), 2089 normotensive controls and 2187 hypertensive patients were included. EF was not significantly different between normotensive and hypertensive subjects. GLS was worse in subjects with hypertension (-18.14 ± 0.40) than in those with normotension (-20.58 ± 0.33). The standardized mean difference (SMD) between the groups was 1.07 ± 0.15, 95% confidence interval (CI) 0.77-1.36, P < 0.0001.

In the other meta-analysis (Tadic et al b), 267 normotensive subjects and 712 hypertensive patients (234 normal geometry, 97 LV concentric remodeling, 176 eccentric LVH, 205 concentric LVH) were included. EF was not different between normotensive and hypertensive individuals. GLS was worse in the whole hypertensive group (-18.1± 0.73) than in the normotensive one (-20.8 ± 0.7); the SMD between the groups was 0.87± 0.25, 95% CI 0.38–1.37, P < 0.0001. When the hypertensive group was analysed separately, compared to patients with normal LV geometry, those with eccentric (SMD 0.49 ± 0.10, 95% CI 0.29–0.70, P < 0.0001) and concentric LVH (SMD 0.90 ± 0.11, 95% CI 0.68–1.10, P < 0.0001) showed altered GLS, but those with LV concentric remodeling (0.18 ± 0.13, 95% CI - 0.06 to 0.44, P = 0.15) did not attain significance.

In the other studies, when compared with normotensive subjects, patients with hypertension had reduced GLS both in the absence and in the presence of LVH, though GLS was more impaired in patients with LVH (Imbalzano et al, Soufi Taleb Bendiab et al, Qingfeng et al, Salas-Pacheco et al, Takahashi et al). A study showed that, beyond LVH, the duration of hypertension, diabetes and increased filling pressures were associated with GLS reduction (Soufi Taleb Bendiab et al). Another study reported that patients with 3 or 4 risk factors, who also had higher LV mass, showed significantly reduced GLS (Takahashi et al).

DISCUSSION

Studies suggest that GLS is lower in hypertensive patients than in normotensive subjects. This alteration may be present in both patients with and without LV hypertrophy. Early impairment of myocardial contractility may be secondary to hemodynamic or biochemical changes. The increased end-systolic wall stress may play a crucial role in leading to longitudinal dysfunction in hypertensive heart disease. During time, chronic increase in end-systolic wall stress promotes progressive increase in LV wall thickness due to myocyte hypertro-
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phy and interstitial fibrosis which are associated with LV systolic longitudinal dysfunction. Indeed, GLS gradually reduces from hypertensive patients with normal LV geometry to those with concentric remodelling, eccentric LVH and concentric LVH. A possible explanation for the increased cardiovascular risk in patients with hypertension, and particularly in those with eccentric and concentric LVH, may be related to the subclinical reduction in longitudinal function, which is undetectable by traditional measures of LV systolic function, such as EF. In this context, GLS assessed by 2D STE is a more sensitive marker for early subtle abnormalities of LV myocardial function and can be of greater help than conventional methods in predicting hypertensive disease progression and outcomes. However, the prevalence of LV longitudinal dysfunction in both hypertensive patients with and without LVH, its predicting factors and its relevance for future LV deterioration and cardiovascular outcomes are not yet completely clear. Thus, other studies are needed to shed further light on the pathophysiology, predictors, epidemiology and short- and long-term clinical consequences of impaired longitudinal systolic function identified by GLS assessment.

REFERENCES


