Can quantitative regional myocardial dynamics contribute to the differential diagnosis of acute stress cardiomyopathy?

**ÖZET**


**Anahtar kelimeler:** Akut stress-induced cardiomyopathy, hypertension, left ventricular hypertrophy, tissue Doppler imaging

**ABSTRACT**

Acute stress-induced cardiomyopathy has excessive sympathetic stimulation, microvascular dysfunction similar to hypertension. Regional prominence of left ventricular (LV) septal base and stress-induced LV hypercontractility are the particular features of both acute and chronic stress-related conditions. Novel imaging methods have shown that stress-induced cavity dilation and myocardial wall abnormalities can be a reflection of underlying previous exaggerated hypertensive episodes due to sympathetic overdrive, which results in microvascular dysfunction. Hypertension-mediated chronic stress due to increased after load episodes is possibly the main reason for blunted LV myocardial wall motion capability in patients with stress-related exaggerated hypertension. In this short report, we discussed the interrelation of myocardial dynamics and stress-induced exaggerated hypertension episodes. In addition, quantitative echocardiographic methods which previously were used for description of particular features including LV regional dynamics in hypertensive heart disease can be an option in differential diagnosis of potential cases of acute stress-induced cardiomyopathy. (Anadolu Kardiyol Derg 2012; 12: 71-4)

**Key words:** Acute stress-induced cardiomyopathy, hypertension, left ventricular hypertrophy, tissue Doppler imaging

It has been demonstrated that hypertensive left ventricular hypertrophy (LVH) is associated with increased sympathetic activity largely confined to the heart suggesting the relation of norepinephrine release and degree of left ventricular (LV) mass (1). In secondary LVH to hypertension, we previously observed that basal intracavitary volume is decreased presumably by dominantly thickened basal LV myocardial segment using real-time three dimensional echocardiography (2). LV remodeling of hypertensive heart disease may have some regional features of LV myocardial mass distribution (Figure 1a, b). In hypertensives with stress-induced dynamic LV outflow tract obstruction, dynamic ejection by stress may contribute to hypertrophy induction of basal segment, which
is the closest part of septum increased afterload (3). In fact, it has been shown that LV septal base is thicker than midapical part even in mild and moderate hypertension (4).

Excessive sympathetic activity and stress-induced LV hypercontractility may be detected in patients with hypertension or hypertensive LVH (5). LV hypercontractile response to sympathetic stimulation is a common finding in LVH and it may result in a diagnostic dilemma for diagnosis of coronary artery disease (CAD) (6). Quantitative evaluation by tissue Doppler imaging of the regional dynamics of LV septal base supports that hypertensive patients may be associated with stress-induced hypercontractility of LV septal base which possibly is the reason of the dynamic LV outflow tract obstruction (7).

Similar to hypertensive heart disease, acute stress-induced cardiomyopathy (ASC) is also associated with excessive sympathetic stimulation and microvascular dysfunction and abnormal myocardial tissue metabolism (8). Wittstein et al. (9) documented that ASC is associated with increased catecholamine levels. LV basal hypercontractility is one of the components of clinical presentation with midapical systolic dysfunction in ASC (10). Predominant basal septal hypertrophy and stress-induced LV cavity obliteration are similar to that in hypertensive heart has been observed in this clinical entity (11).

Recently, we have suggested that this geometric and functional similarity of LV septal base may represent a morphologic conjunction that we have described as stressed heart morphology in clinical conditions with acute stress or chronic stress due to increased after load in hypertension (12). Dhoble et al. (13) have reported that LV geometry demonstrates a predominant regional LV septal base that is consistent with our description (12), however, akinetic LV base has been detected. In this report, despite the absence of hypertension diagnosis, exaggerated hypertensive response during exercise stress echocardiography supports the argument that previous increased after load episodes with exaggerated hypertensive response may result in regional predominance of LV septal base.

Majority of observations in patients with ASC show that hypercontractile LV base is associated with the LV basal predominance in ASC (10-12). In the report of Dhoble et al. (13), despite excessive hypertensive response possibly due to excessive sympathetic stimulation, LV base was not able to give a reasonable response to stress and stayed akinetic. Because of normal perfusion in the repetitive test, decision was the complete elimination of CAD (13). On the other hand, the test in which maximum blood pressure and/or double product are achieved should be considered as principal test that is a general paradigm for repetitive diagnostic tests. Nuclear studies have shown that presence of transient LV cavity dilation as well as abnormal myocardial kinetics could be a reflection of CAD and should be interrogated (14, 15).

Because there is no any gold standard diagnostic methodology in clinical practice and all methods have some limitations rather than angiography, complete exclusion of CAD still stays a big challenge. Currently, routine angiography is a debate for stress-induced exaggerated hypertensives since usually this group of patients are associated with hyperdynamic myocardial tissue response to stress (16). Since kinetic abnormality on LV base is not consistent finding with ASC and reflects an insufficient response to stress, those patients should be pre-diagnosed as hypertensive heart disease after precise elimination of coronary artery disease. Patients with stress-induced hypertensive response, which is the major risk for endothelial dysfunction possibly have previous exaggerated hypertensive episodes due to sympathetic overdrive (17-19).

Endothelial dysfunction-mediated sympathetic overdrive could be a potential reason for clinical conditions with increased after load episodes due to exaggerated hypertension and related stressed heart morphology (12) especially in the elderly patient as reported by Dhoble et al. (13). Nevertheless, myocardial fibrosis was clearly documented in the process of the hypertensive disease (20) and recently, an interesting report exploring myo-
cardial tissue fibrosis in hypertension-mediated heart failure patients using comprehensive cardiac diagnostic methods has been presented (21).

Comprehensive quantitative imaging including stress tissue Doppler imaging and 3 dimensional echocardiography combined with rigorous mitral annular reconstruction methodology provided additional information regarding global and regional myocardial contractility and showed that LV function at rest is preserved in hypertensive heart disease before heart failure development (22, 23). Although decreased myocardial velocities were obtained by tissue Doppler imaging in patients with essential hypertension, normal myocardial velocities is expected in patients with ASC and this difference may provide a clue for diagnosis.

At stress, LV contractile response using quantitative imaging methods was found to be unsatisfactory in well-treated older hypertensives (24) compared to younger patients (7) who have same disease and reasonable LV contractile response. Despite lack of CAD after precise elimination of CAD existence in elderly hypertensive cases like the report of Dhoble et al. (13), blunted stress-induced LV contractile function may be associated with hypertensive heart disease in addition to morphological details, which clearly support the existence of hypertensive heart disease (12).

Very recently, we have objectively confirmed the relation between regional dynamics of LV septal base and LV outflow tract blood flow velocities, which is directly related to afterload in patients with hypertension (25). This recent finding has shown the direct relation between stress-induced myocardial dynamics and stress-induced fluid dynamics supporting that increased afterload episodes during chronic course of hypertensive disease is possibly the main reason for abnormal blood flow and related LV myocardial wall motion abnormalities.

**Conclusion**

Firstly, we believe that the best description of elderly cases who have exaggerated hypertensive response to stress induction and abnormal wall motion could be hypertensive heart disease instead of ASC. Secondly, particular features of LV regional dynamics, which were described by quantitative echocardiographic methods in hypertensive heart disease can be quantified using similar imaging approach in differential diagnosis of potential ASC cases.

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**References**

12. Yalçın F, Müdderrisoğlu H. Takotsubo cardiomyopathy may be associated with cardiac geometric features as observed in hypertensive heart disease. Int J Cardiol 2009; 135: 251-2. [CrossRef]
15. Higgins JP, Higgins JA, Williams G. Stress-induced abnormalities in myocardial perfusion imaging that are not related to perfusion but are of diagnostic and prognostic importance. Eur J Nucl Med Mol Imaging 2007; 34: 584-95. [CrossRef]


