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

Bölgesel miyokart dinamiklerinin niceliği akut stres kardiyomiyopati ayrıcı tanısına katkı sağlayabilir mi?

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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

ÖZET

Akut stresle tetiklenen kardiyomiyopatide aşırı sempatik stimülasyon, hipertansiyona benzer mikrovasküler disfonksiyon vardır. Sol ventrikül (SV) bazal septal bölgesel belirginleşme ve stresin indüklediği SV hiperkontraktilitesi hem akut hem de kronik stres-ilişkili durumların belirli özellikleridir. Yeni görüntüleme metotları göstermiştir ki, stresin tetiklediği kavite dilatasyonu ve miyokardiyal anormallikler mikrovasküler dolaşım bozukluğu ile sonuçlanan altta yatan aşırı sempatik yüklenmeye bağlı önceki aşırı hipertansif epizotların yansıması olabilir. Artmış ardyük epizotlarına bağlı hipertansiyon aracılı kronik stres, strese bağlı aşırı hipertansiyonu olan hastalarda körelmiş SV miyokardiyal duvar hareket kabiliyeti için ana neden olabilir. Burada miyokardiyal dinamiklerin ve stresle tetiklenen aşırı hipertansiyon epizotlarının karşılıklı ilişkisini tartıştık. Ek olarak, hipertansif kalp hastalığında SV bölgesel dinamiklerinin tanımı için daha önce kullanılmış olan kantitatif ekokardiyografik yöntemler olası akut stresle tetiklenen kardiyomiyopati vakalarının ayırıcı tanısında bir seçenek olabilir. *(Anadolu Kardiyol Derg 2012; 12: 71-4)*

Anahtar kelimeler: Akut stresle tetiklenen kardiyomiyopati, hipertansiyon, sol ventrikül hipertrofisi, doku Doppler görüntüleme

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

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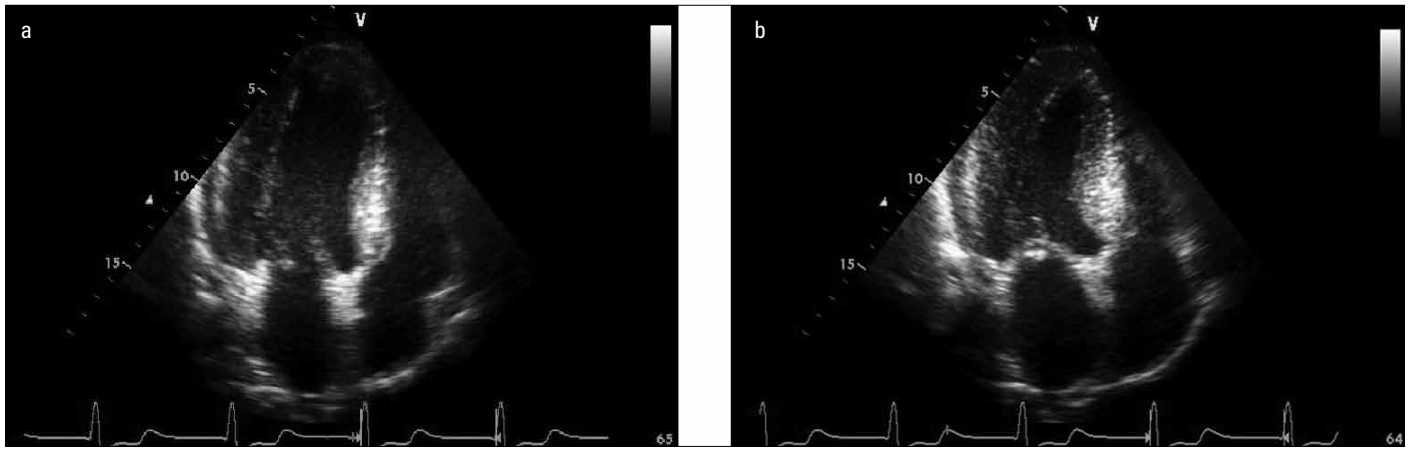

Figure 1a, b. End-diastolic apical LV cavity geometry during diastole and end-systolic LV intracavitary obliteration by remarkable hypertrophied septal base protruding into the LV outflow tract in an advance hypertensive patient with left ventricular hypertrophy and pericardial effusion on the lateral wall, respectively
LV - left ventricle

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

1. Schlaich MP, Kaye DM, Lambert E, Sommerville M, Socratous F, Esler MD. Relation between cardiac sympathetic activity and hypertensive left ventricular hypertrophy. Circulation 2003; 108: 560-5. [CrossRef]
2. Yalçın F, Shiota T, Odabashian J, Agler D, Greenberg NL, Garcia MJ, et al. Comparison by real-time three dimensional echocardiography of left ventricular geometry in hypertrophic cardiomyopathy versus secondary left ventricular hypertrophy. Am J Cardiol 2000; 85:1035-8. [CrossRef]
3. Yalçın F, Müderrisoğlu H, Korkmaz ME, Özin B, Baltalı M, Yiğit F. The effect of dobutamine stress on left ventricular outflow tract gradients in hypertensive patients with basal septal hypertrophy. Angiology 2004; 55: 295-301. [CrossRef]
4. Baltabavea AT, Marciniak M, Bijnens B, Moggridge J, He FJ, Antonios TF, et al. Regional left ventricular deformation and geometry analysis provides insight in myocardial remodelling in mild to moderate hypertension. Eur J Echocardiogr 2008; 9: 501-8.
5. Yalçın F, Yalçın H, Seyfeli E, Akgül F. Stress-induced hypercontractility in patients with hypertension: an interesting imaging finding. Int J Cardiol 2010; 143: e1-3. [CrossRef]
6. Smart SC, Knickelbine T, Malik F, Sagar KB. Dobutamine-atropine stress echocardiography for the detection of coronary artery disease in patients with left ventricular hypertrophy. Importance of chamber size and systolic wall stress. Circulation 2000; 101: 258-63.
7. Yalçın F, Yiğit F, Erol T, Baltalı M, Korkmaz ME, Müderrisoğlu H. Effect of dobutamine stress on basal septal tissue dynamics in hypertensive patients with basal septal hypertrophy. J Hum Hypertens 2006; 20: 628-30. [CrossRef]
8. Dorfman TA, Iskandrian AE. Takotsubo cardiomyopathy: state-of-the-art review. J Nucl Cardiol 2009; 16: 122-34. [CrossRef]
9. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med 2005; 352: 539-48. [CrossRef]
10. Sharkey SW, Lesser JR, Zenovich AG, Maron MS, Lindberg J, Longe TF, et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation 2005; 111: 472-9. [CrossRef]
11. Azzarelli S, Galassi AR, Amico F, Giacoppo M, Argentino V, Fiscella A. Intraventricular obstruction in a patient with tako-tsubo cardiomyopathy. Int J Cardiol 2007; 121: 22-4. [CrossRef]
12. Yalçın F, Müderrisoğlu H. Takotsubo cardiomyopathy may be associated with cardiac geometric features as observed in hypertensive heart disease. Int J Cardiol 2009; 135: 251-2. [CrossRef]
13. Dhoble A, Abdelmoneim SS, Bernier M, Oh JK, Mulvagh SL. Transient left ventricular apical ballooning and exercise induced hypertension during treadmill exercise testing: is there a common hypersympathetic mechanism? Cardiovasc Ultrasound 2008; 6: 37. [CrossRef]
14. Bestetti A, Bigi R, Terranova P, Lombardi F, Fiorentini C. Prognostic implications of stress-induced transient ischemic dilation of the left ventricle in patients with systolic dysfunction and fixed perfusion defects. Int J Cardiol. 2010; 140: 323-7. [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]

16. Küçükler N, Yalçın F, Abraham TP, Garcia MJ. Stress induced hypertensive response: should it be evaluated more carefully? Cardiovasc Ultrasound 2011; 9: 22. [CrossRef]
17. Stewart KJ, Sung J, Silber HA, Fleg JL, Kelemen MD, Turner KL, et al. Exaggerated exercise blood pressure is related to impaired endothelial vasodilator function. Am J Hypertens 2004; 17: 314-20. [CrossRef]
18. Jae SY, Fernhall B, Lee M, Heffernan KS, Lee MK, Choi YH, et al. Exaggerated blood pressure response to exercise is associated with inflammatory markers. J Cardiopulm Rehabil 2006; 26: 145-9. [CrossRef]
19. Tzemos N, Lim PO, MacDonald TM. Exercise blood pressure and endothelial dysfunction in hypertension. Int J Clin Pract 2009; 63: 202-6. [CrossRef]
20. Diez J, Frohlich ED. A translational approach to hypertensive heart disease. Hypertension 2010; 55: 1-8. [CrossRef]
21. Nakagawa Y, Kijima Y, Nishibe A, et al. Myocardial fibrosis is associated with diastolic heart failure in hypertensive patients - noninvasive assessment by cardiac magnetic resonance. Eur Heart J 2010; 31: S-457.
22. Yalçın F, Yalçın H, Küçükler N, Abraham TP. Quantitative left ventricular contractility analysis under stress: a new practical approach in follow-up of hypertensive patients. J Hum Hypertens 2011; 25: 578-84. [CrossRef]
23. Yalçın F, Shiota M, Greenberg N, Thomas JD, Shiota T. Real time three-dimensional echocardiography evaluation of mitral annular characteristics in patients with myocardial hypertrophy. Echocardiography 2008; 25: 424-8. [CrossRef]
24. Tan YT, Wenzelburger F, Lee E, Heatlie G, Frenneaux M, Sanderson JE. Abnormal left ventricular function occurs on exercise in well-treated hypertensive subjects with normal resting echocardiography. Heart 2010; 96: 948-55. [CrossRef]
25. Yalçın F, Küçükler N, Haq N, Abraham TP. Evaluation of regional myocardial dynamics in left ventricular hypertrophy secondary to essential hypertension. J Am Coll Cardiol 2011; 57:E2000. [CrossRef]