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Left Ventricular Diastolic Dyssynchrony in Post-Myocardial Infarction Patients: Does It Predict Future Left Ventricular Remodeling?

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Left ventricular (LV) remodeling is well-known complication after myocardial infarction (MI) and numerous studies have emphasized the clinical importance of ventricular remodeling.[1-4] In these literatures, progressive LV dilatation and decreased LV ejection fraction were major determinants in future development of heart failure and long term survival. That is why early recognition of patients at risk for LV remodeling after MI has vital importance and identification of the predictive markers for developing ventricular remodeling is clinically meaningful.

While the clinical importance of LV mechanical dyssynchrony is mainly described in heart failure patients,[5-7] its significance in patients with MI was less well established. Actually, ventricular dyssynchrony is not uncommon in post-MI patients even with narrow QRS complexes.[8][9] Zhang et al.[8] and Fahmy Elnoamany et al.[9] noted LV systolic dyssynchrony early after MI in 69.8% and 77.5% of the patients and it was mainly determined by the initial infarct size. After that, various parameters from tissue Doppler imaging and speckle-tracking technique have been used for detecting regional contraction and relaxation abnormalities and LV systolic and diastolic dyssynchrony early after MI. Several studies demonstrated that LV systolic dyssynchrony in post-MI patients is closely related with future LV remodeling[10-13] and poor prognosis.[14][15] Mollema et al.[10] showed that patients with more extensive LV dyssynchrony at baseline have larger LV end systolic volume after 6 months of follow up and increased risk of LV remodeling. Similar results were showed by Zhang et al.[11] that LV systolic dyssynchrony increased with worsening LV ejection fraction in the remodeling group. On the other hand, information on the role of LV diastolic dyssynchrony in post-MI patients is limited and only a few studies examined the clinical implication. In earlier studies, the prevalence of diastolic dyssynchrony was not significantly different in post-MI patients compared with control group.[8][9] In a study from Zhang et al.,[11] both standard deviation (SD) of time to peak myocardial contraction represents LV systolic dyssynchrony and SD of time to peak early relaxation (Te-SD) represents LV diastolic dyssynchrony at baseline were significantly higher in patients who experienced LV remodeling after 1 year with the marginal statistical significance of Te-SD (p = 0.048). However, in multivariate analysis to identify independent predictor of 1 year LV remodeling, Te-SD did not predict LV remodeling.

In the article published in this issue of the Journal of Cardiovascular Ultrasound titled "Diastolic dyssynchrony in acute ST segment elevation myocardial infarction and relationship with functional recovery of left ventricle,"[16] the authors investigated the incidence and clinical impact of LV diastolic dyssynchrony after ST-segment elevation MI (STEMI). Substantial number of patients (58% of STEMI patients) presented diastolic dyssynchrony. One of the interesting finding of this study is that diastolic dyssynchrony was positively correlated with change in ejection fraction and the patients who have baseline diastolic dyssynchrony less frequently experienced adverse LV remodeling. But diastolic dyssynchrony per se was not an independent predictor of LV remodeling or improving ejection fraction. The authors explained the inverse relationship between baseline diastolic dyssynchrony and 6-month LV remodeling with stunned myocardium in peri-infarct zone which can be improved after successful revascularization. Be-

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cause diastolic function can be affected by myocardial ischemia earlier than systolic function, diastolic dyssynchrony might come from stunned but viable myocardium. In other words, more prominent diastolic dyssynchrony may represent more viable myocardium. But the exact mechanism or clinical meaning is uncertain until now.

An almost certain thing relevant with previous studies is that there is no definite correlation between systolic dyssynchrony and diastolic dyssynchrony. While systolic dyssynchrony is seriously affected by infarct size and predicts adverse LV remodeling, diastolic dyssynchrony is considered distinct phenomena from systolic dyssynchrony which has different underlying mechanism and determinant. In a previous study,[17] there was no significant correlation between the systolic and diastolic dyssynchrony indexes and the determinants of LV diastolic dyssynchrony are different from those of systolic dyssynchrony in asymptomatic hypertensive patients. Similar findings were reported in patients with diastolic heart failure.[18]

The time is not yet ripe for defining the impact of LV diastolic dyssynchrony on LV remodeling after MI. A better understanding of pathophysiologic mechanism of LV diastolic dyssynchrony in post-MI patients might be helpful to clarifying the clinical significance.

References

1. White HD, Norris RM, Brown MA, Brandt PW, Whitlock RM, Wild CJ. *Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987;76:44-51.*
2. Pfeffer MA, Braunwald E. *Ventricular remodeling after myocardial infarction. Experimental observations and clinical implications. Circulation 1990;81:1161-72.*
3. Gaudron P, Eilles C, Kugler I, Ertl G. *Progressive left ventricular dysfunction and remodeling after myocardial infarction. Potential mechanisms and early predictors. Circulation 1993;87:755-63.*
4. Cohn JN, Ferrari R, Sharpe N. *Cardiac remodeling--concepts and clinical implications: a consensus paper from an international forum on cardiac remodeling. Behalf of an international forum on cardiac remodeling. J Am Coll Cardiol 2000;35:569-82.*
5. Penicka M, Bartunek J, Lang O, Medilek K, Tousek P, Vanderheyden M, De Bruyne B, Maruskova M, Widimsky P. *Severe left ventricular dyssynchrony is associated with poor prognosis in patients with moderate systolic heart failure undergoing coronary artery bypass grafting. J Am Coll Cardiol 2007;50:1315-23.*
6. Bader H, Garrigue S, Lafitte S, Reuter S, Jaïs P, Haïssaguerre M, Bonnet J, Clementy J, Roudaut R. *Intra-left ventricular electromechanical asynchrony. A new independent predictor of severe cardiac events in heart failure patients. J Am Coll Cardiol 2004;43:248-56.*
7. Cho GY, Song JK, Park WJ, Han SW, Choi SH, Doo YC, Oh DJ, Lee Y. *Mechanical dyssynchrony assessed by tissue Doppler imaging is a powerful predictor of mortality in congestive heart failure with normal QRS duration. J Am Coll Cardiol 2005;46:2237-43.*
8. Zhang Y, Chan AK, Yu CM, Lam WW, Yip GW, Fung WH, So NM, Wang M, Sanderson JE. *Left ventricular systolic asynchrony after acute myocardial infarction in patients with narrow QRS complexes. Am Heart J 2005;149:497-503.*
9. Fahmy Elnoamany M, Mahfouz Badran H, Helmy Abo Elazm T, Shawky Abdelaziz E. *Asynchrony of left ventricular systolic performance after the first acute myocardial infarction in patients with narrow QRS complexes: Doppler tissue imaging study. J Am Soc Echocardiogr 2006;19:1449-57.*
10. Mollema SA, Liem SS, Suffoletto MS, Bleeker GB, van der Hoeven BL, van de Veire NR, Boersma E, Holman ER, van der Wall EE, Schalij MJ, Gorcsan J 3rd, Bax JJ. *Left ventricular dyssynchrony acutely after myocardial infarction predicts left ventricular remodeling. J Am Coll Cardiol 2007;50:1532-40.*
11. Zhang Y, Yip GW, Chan AK, Wang M, Lam WW, Fung JW, Chan JY, Sanderson JE, Yu CM. *Left ventricular systolic dyssynchrony is a predictor of cardiac remodeling after myocardial infarction. Am Heart J 2008;156:1124-32.*
12. Ko JS, Jeong MH, Lee MG, Lee SE, Kang WY, Kim SH, Park KH, Sim DS, Yoon NS, Yoon HJ, Hong YJ, Park HW, Kim JH, Ahn Y, Cho JG, Park JC, Kang JC. *Left ventricular dyssynchrony after acute myocardial infarction is a powerful indicator of left ventricular remodeling. Korean Circ J 2009;39:236-42.*
13. Turan B, Yilmaz F, Karaahmet T, Tigen K, Mutlu B, Basaran Y. *Role of left ventricular dyssynchrony in predicting remodeling after ST elevation myocardial infarction. Echocardiography 2012;29:165-72.*
14. Shin SH, Hung CL, Uno H, Hassanein AH, Verma A, Bourgoun M, Køber L, Ghali JK, Velazquez EJ, Califf RM, Pfeffer MA, Solomon SD; Valsartan in Acute Myocardial Infarction Trial (VALIANT) Investigators. *Mechanical dyssynchrony after myocardial infarction in patients with left ventricular dysfunction, heart failure, or both. Circulation 2010;121:1096-103.*
15. Westholm C, Johnson J, Jernberg T, Winter R. *The prognostic value of mechanical left ventricular dyssynchrony in patients with acute coronary syndrome. Cardiovasc Ultrasound 2013;11:35.*
16. Turan B, Daşli T, Erkol A, Erden İ, Başaran Y. *Diastolic dyssynchrony in acute ST segment elevation myocardial infarction and relationship with functional recovery of left ventricle. J Cardiovasc Ultrasound 2016;24:208-14.*
17. Chang SA, Kim HK, Kim DH, Kim YJ, Sohn DW, Oh BH, Park YB. *Left ventricular systolic and diastolic dyssynchrony in asymptomatic hypertensive patients. J Am Soc Echocardiogr 2009;22:337-42.*
18. Yu CM, Zhang Q, Yip GW, Lee PW, Kum LC, Lam YY, Fung JW. *Diastolic and systolic asynchrony in patients with diastolic heart failure: a common but ignored condition. J Am Coll Cardiol 2007;49:97-105.*