

Is there a pathophysiological link between high arterial stiffness and left ventricular filling pressures in patients with acute myocardial infarction?

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It is known that early reperfusion of the obstructed coronary artery, obtained by current percutaneous intervention techniques, has a favorable effect on clinical outcomes¹ because it may reduce left ventricular (LV) remodeling and facilitate the recovery of LV function. However, despite improvements in the treatment of myocardial infarction (MI), almost 30% of patients show LV remodeling² and LV ejection fraction (EF) below 40% at 6 months after myocardial infarction (MI).³ In addition to abnormal myocardial contraction with consecutive LV systolic dysfunction, LV diastolic dysfunction characterized by elevated filling pressures is often documented, even in subjects with preserved EF. On the contrary, elevated filling pressure usually indicates a larger infarction with more pronounced systolic dysfunction and particular predisposition to remodeling.⁴ Although most frequently, it is diagnosed as a preclinical disease, LV diastolic dysfunction constitutes an independent predictor of all-cause mortality.⁵

Several echocardiographic indices have been used to measure LV filling pressure and, in recent years, tissue Doppler imaging has been studied in a wide variety of cardiac abnormalities, including acute MI.⁶ The ratio between early diastolic mitral flow velocity on conventional pulsed Doppler (E) and early diastolic mitral annular velocity on tissue Doppler (E'), known as the E/E' ratio, showed a strong correlation with invasively determined LV filling pressure.⁷ Many factors have been proved to determine LV diastolic function,⁷ and recent studies have investigated its relation with arterial stiffness,⁹ another strong predictor of cardiovascular morbidity and mortality.¹⁰ The carotid-femoral pulse wave velocity (PWV) is

currently used as the "gold standard" for measuring aortic stiffness, and there is substantial evidence linking it to cardiovascular outcomes. The majority of available methods for measuring arterial stiffness have proved to be both technically difficult to perform and time consuming,¹¹ specifically in terms of their use in risk assessment among large populations and in acute settings. The stiffness index derived from the analysis of digital volume pulse (DVP) is a noninvasive indirect technique of measuring arterial stiffness peripherally,¹² and it has been proved to be a validated and reproducible technique with minimal intraobserver variation.¹³

Milewska et al¹⁴ have recently documented an interesting relation between increased arterial stiffness, measured with the DVP waveform analysis, and LV diastolic dysfunction in patients with acute MI. Although previous studies have already reported this relationship in different chronic conditions, this assessment provides new insights into the potential mechanism that limits the adaptive responses to acute ischemia when systolic stress is increased by ejection into the stiffened vasculature.

In agreement with the results of Milewska et al,¹⁴ we also found, in our study,¹⁵ that patients with higher PWV had a higher E/E' ratio. We suggested that this association could compromise coronary perfusion and enhance subendocardial ischemia, leading to impairment of myocardial relaxation and promoting myocardial fibrosis. The further evidence of this association, as reported by Milewska et al,¹⁴ indicates the importance of vascular stiffness as a predictor of poor prognosis, even in patients with previous MI. Its assessment should be routinely extended during

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follow-up, considering that some drugs should be preferable in postinfarction individuals with increased arterial stiffness. However, considering that this is only an interesting hypothesis requiring further studies, it is necessary to analyze the critical aspects of the presented study, beyond those given by the authors. First of all, the authors evaluated the relationship between vascular stiffness and diastolic dysfunction in patients with acute coronary syndrome, regardless of the type of clinical presentation and severity of coronary artery disease. The second limitation is the absence of a direct linear estimation between the two key variables beyond their significant interaction according to the cut-off used by the authors.

In conclusion, the study by Milewska et al¹⁴ should encourage this novel approach to PWV, which would improve the understanding of its effect on cardiovascular risk estimation in patients with acute MI and after MI. In addition, the results of the study may be important in considering diagnostic and therapeutic strategies aimed at prognostic improvement.

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