Correspondence

Letter by Liu Regarding Article, “Systolic and Diastolic Mechanics in Stress Cardiomyopathy”

To the Editor:

I have read the Medeiros et al1 article published on April 22, 2014, that analyzed systolic and diastolic mechanics of stress cardiomyopathy (SCM) and acute myocardial infarction (AMI). This interesting study leaves 2 unanswered questions: Why do SCM and AMI have similar systolic and diastolic properties, even though they have distinctive causes, pathophysiologies, and clinical manifestations? And do we have to rely on coronary angiogram to differentiate them?

Now that more real-time cardiac imaging is applied in patients with acute coronary syndrome, it is realized that SCM occurs much more often than we thought. Recent research suggests that SCM is mediated by a cardioprotective signal transduction pathway, which likely evolved as a self-protective strategy.2 Clinically, although the heart often responds to coronary artery stenosis with “myocardial stunning,” a wider myocardial involvement beyond the culprit coronary artery territory with delayed recovery of contractile function seems to be the same: an active (not passive) left ventricular (LV) dilation that prevents an abrupt increase in LV filling pressure and the onset of pulmonary edema. Thus, it is not surprising that the present study1 did not detect significant differences in LV filling pressure between SCM and AMI. Do SCM and myocardial stunning belong to a ubiquitous self-protection system of the heart to avoid adverse outcome when it is jeopardized?

If the difference in LV systolic/diastolic properties cannot be detected, coronary angiogram seems to be the only approach to differentiate SCM and AMI. Unfortunately, bedside clinicians often have a dilemma when cardiac catheterization or thrombolytic therapy is contraindicated or can cause potential adverse consequences in these patients. However, misdiagnosing SCM as AMI can lead to the initiation of harmful pressors, resulting in hemodynamic instability. Therefore, timely distinguishing SCM from AMI with noninvasive method remains crucial. Because current bedside echocardiographic examinations rarely cover right ventricular (RV) apex sufficiently, a characteristic RV feature in SCM is frequently missed: hyperkinetic RV basilar/middle walls with hypokinetic RV apex (reverse McConnell sign4,5). The RV hpercontractility likely represents an adaptive physiological response to the transiently suppressed LV function to maintain cardiac output. Tethering of the RV apex to an akinetic LV apex accounts for reduced RV apical contractility. This discordant RV contractile feature not only differentiates SCM and AMI but also helps explore the unique underlying pathophysiology of SCM. In the acute phase of SCM, less vulnerable myocardium, including basilar RV and LV walls, likely forms a transiently functional conduit within the base of the heart. This alternative pathway can detour blood flow toward the LV outflow tract to maintain hemodynamic stability until the excessive catecholamine surge subsides. Without the interruption (such as dynamic LV outflow tract obstruction4), this harmonious adventricular-dependent system maintains benign clinical courses in most SCM cases. Therefore, the therapeutic strategies for SCM should involve mainly the support, not the disruption, of this “rescue” mechanism to avoid adverse outcome.3

Disclosures

None.

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References


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