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Letter to the editor

Coronary artery systolic "milking" and "bridging" in Takotsubo syndrome: substrate or an epiphenomenon?

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To The Editor:

I enjoyed reading the case report by Spadotto et al.[1] published in issue 2 (2013) of the *Journal* about a 66-year-old woman with Takotsubo syndrome (TTS), involving the mid-apical segment of the left ventricle (LV), severe dynamic mid-ventricular gradient and mitral regurgitation, with systolic anterior motion of the anterior mitral leaflet caused by the hyperkinetic basal segments, with generation of a peak intra-ventricular gradient of 48 mmHg, and mild systolic "milking" of the mid to distal segment of the left anterior descending coronary artery (LAD). Cardiac magnetic resonance imaging (cMRI) showed intense myocardial edema of the mid-apical LV segments. The authors cite their previous report of "a recent clinically based study (which) showed that myocardial bridging of the LAD artery was detected in 76% of TTC patients either by angiography or MSCT".[2] It is conceivable that this out of proportion high rate of "bridging", and the occasionally reported systolic "milking" of the LAD in patients with TTS is due to an interplay of hypercontractility of the LV and the myocardial edema,[3] present in this condition. The issue can be easily resolved by coronary angiography (CA) (or MSCT), performed prior or after a bout of TTS in patients, who had/have a clinical indication for such a study, before or after their episode of TTS. In other words I am in doubt of the authors' previous explanation that "myocardial bridging of the LAD is a frequent finding in TTS patients as revealed both by CA and, mostly, by MSCT, suggesting a role of myocardial bridging as potential substrate in the pathogenesis of TTS; probably this is an epiphenomenon of TTS state rather than an existing substrate prion to the inception of the TTS.

Conflicts of Interest Disclosure

There is nothing to disclose.

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