Left Ventricular End Diastolic Pressure, Suspected Underlying Hypertrophic Cardiomyopathy, and Takotsubo Syndrome – Reply –

We appreciate the opportunity to respond to the interesting comments from Dr Madias regarding left ventricular end-diastolic pressure (LVEDP) and suspected hypertrophic cardiomyopathy in the context of our recent tako-tsubo cardiomyopathy (TTC) review article.¹

First, we respectfully disagree with Dr Madias' first comment regarding LVEDP in TTC: "most authors report LVEDP is normal or even low". A number of reports have documented significantly increased LVEDP in acute TTC, including Medeiros et al² (mean 25±6 mmHg); Madhavan et al³ (mean 26 mmHg); and Wittstein et al⁴ (median 30 mmHg). In our own single institution experience, LVEDP was measured in 271 of 350 patients (77%); the mean LVEDP was 22±8 mmHg (range, 2–41 mmHg) and was ≥18 mmHg in 195 patients (72%). Further, Dr Madias refers to the publication by Chong et al,⁵ in which right heart catheterization findings were reported in 80 TTC patients with mean pulmonary capillary wedge pressure 15±7 mmHg. However, LVEDP was not reported in those patients. It is well established that pulmonary capillary wedge pressure may significantly underestimate LVEDP in acute myocardial infarction, and a similar dissociation might be expected in TTC.⁶ Therefore, pulmonary capillary wedge pressure is not necessarily a surrogate for LVEDP in TTC. These observations strongly suggest LVEDP is in fact frequently elevated during acute TTC.

Second, Dr Madias is correct to urge caution in establishing the diagnosis of coexisting hypertrophic cardiomyopathy during the acute phase of TTC. Left ventricular (LV) outflow tract obstruction because of mitral valve systolic anterior motion (SAM) is not uncommon in TTC and often observed in patients administered catecholamine drugs as treatment for hypotension.⁷ We have emphasized that LV outflow tract obstruction because of SAM during acute TTC is a nonspecific finding with many potential determinants, including the magnitude and anatomic location of non-contracting myocardium, wall thickness and contractile force of the proximal septum, LV chamber size, mitral valve anatomy, and use of catechol-

amine drugs. Nonetheless, a subset of TTC patients with SAM and LV outflow tract obstruction, may prove to have coexisting hypertrophic cardiomyopathy. This diagnosis should be considered after recovery from TTC by carefully evaluating follow-up imaging modalities (including echocardiography and cardiac MRI) for otherwise unexplained LV hypertrophy.

Third, Dr Madias is also correct to consider that myocardial edema during acute TTC may cause apical pseudo-hypertrophy and mimic apical hypertrophic cardiomyopathy. However, this process should not influence the thickness of the proximal septum, which is typically hyper-contractile during acute TTC, nor would it be expected to cause SAM and LV outflow tract obstruction.

References

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Scott W Sharkey, MD
Minneapolis Heart Institute,
Minneapolis, MN, USA

Barry J Maron, MD
Director, Hypertrophic Cardiomyopathy Center,
Minneapolis Heart Institute,
Minneapolis, MN, USA

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