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The Reply

We thank Ratanapo et al[1] for their insightful comments regarding migratory takotsubo. They correctly highlight previous isolated reports of stress cardiomyopathy involving variable myocardial segments at different time points. We should have been clearer in distinguishing our report as the first description of this phenomenon in the setting of an "indolent" stressor, in this case cholecystitis. In addition, our patient developed flash pulmonary edema on her third presentation, presumably related to diastolic dysfunction given no evidence of segmental wall motion abnormalities or a reduction in ejection fraction. To our knowledge, the clinical syndrome of acute decompensated heart failure solely attributable to isolated diastolic dysfunction has not been described in the setting of stress cardiomyopathy.

Migratory wall motion abnormalities associated with stress cardiomyopathy remain an unusual clinical entity. Although takotsubo syndrome typically presents with apical ballooning and less often with midventricular ballooning, any segment of the myocardium can be involved and is likely attributable to catecholamine excess and microvascular dysfunction.[2,3] The mechanism for reversible diastolic dysfunction in this syndrome remains poorly elucidated.

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As Ratanapo et al[1] note, although the mechanism for apical ballooning has been proposed,[3] the basis for within-patient variation of myocardial dysfunction remains unclear. Our intent in highlighting this case is to provide a moniker for this phenomenon in hopes of eliciting further investigation into the pathobiology of this unusual syndrome.

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