Irreversible apical ballooning may also occur

Sir,

Contrary to the assertion that 'the central tenet of takotsubo cardiomyopathy is that ultimately the myocardial function will return to normal' (vol. 74(2), 2013, p. 96), a 72-year-old woman has been reported in whom takotsubo cardiomyopathy was characterized by chest pain and dyspnoea, and complicated, on 3-month follow up, by persistent left ventricular apical akinesis, the latter in the absence of significant stenosis on coronary angiography (Miyata et al, 2013). Owing to persisting heart failure, aneurysm resection and left ventricular volume reduction was undertaken. Histological examination of excised myocardial tissue revealed damaged cardiomyocytes replaced by interstitial fibrosis and adipose tissue. Subsequently, both her symptoms and left ventricular systolic function improved (Miyata et al, 2013).

Persistent apical ballooning consistent with left ventricular aneurysm was also a feature in a 62-year-old woman in whom takotsubo cardiomyopathy was associated with angiographically normal coronary arteries (Lee et al, 2011). On 5-week follow up, a newly developed left ventricular apical thrombus was noted, necessitating oral anticoagulation. Repeat echocardiography 3 months thereafter showed complete resolution of the apical thrombus, but there was persistent akinesia of the left ventricular apex. Irreversible myocardial damage may, therefore, occasionally occur in takotsubo cardiomyopathy.

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Lee PH, Song J-K, Park IK et al (2011) Takotsubo cardiomyopathy: a case of persistent apical ballooning complicated by an apical mural thrombus. *Korean J Intern Med* **26**: 455–9
Miyata M, Nakazato K, Sakamoto N et al (2013) Left ventricular plasty improved cardiac function in a case of takotsubo cardiomyopathy with persistent aneurysm. *J Cardiol Cases* **7**(5): e133–6

Sir,

We are grateful to Dr Jolobe for highlighting these interesting patients whose problems have been attributed to takotsubo cardiomyopathy with persistent apical dysfunction. We agree that there is increasing recognition of atypical forms of takotsubo cardiomyopathy, and cases where the ventricle fails to recover would fall into this category.

Published data suggests that clinical recurrence occurs in 3–5% of cases (e.g. Gianni et al, 2006). In our experience this manifests as recurrent admissions related to emotional or physical stress, with systolic function returning to normal after each episode, although the repetitive nature of the insult can lead to some patients displaying evidence of permanent injury (e.g. apical late gadolinium enhancement on cardiac magnetic resonance imaging).

The two cases referenced concern a phenomenon distinct from clinical recurrence, namely cases where a larger injury occurs resulting in failure of ventricular function to recover. At one level this makes the diagnosis more challenging, given that recovery at follow up is the hallmark of typical takotsubo cardiomyopathy (>99% cases reported). But permanent dysfunction may occur, and it is important to acknowledge this rare and atypical form. We would recommend a careful review of the index presentation, to ensure it fulfilled the initial criteria for a case of takotsubo cardiomyopathy (as these two cases indeed do). Otherwise differential diagnoses must be considered, especially in the first case where the histology appears more consistent with an infarct than catecholamine cardiotoxicity (Miyata et al, 2013).

Understanding of the patterns, aetiology and variants of takotsubo cardiomyopathy continues to expand, and with time we may indeed define a larger cohort of patients in whom persistent dysfunction is found. However, currently this remains an extremely rare occurrence, and at this time we do not think the diagnostic criteria, including reversibility, should be amended. This question also underlines the need for complete national and international registries of takotsubo cardiomyopathy that will help to provide more information to help answer these exact questions.

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