A 58-year-old female presented with severe chest pain following news of bereavement. ECG suggested myocardial ischaemia, and troponin-I (2.19 μg/mL) was raised. Left ventricular (LV) echocardiogram (Panel 1) showed apico-midventricular akinesia and basal hyperkinesia. Forty-eight hours later, coronary arteries were normal (Panel 2a and 2b) and LV angiogram (Panel 3a and 3b) showed mid-ventricular ballooning and apico-basal hyperkinesia. Twenty-four-hour urinary catecholamines were normal. The LV recovered in 4 weeks (Panel 4a and 4b). Stress-induced cardiomyopathy (SCM) was diagnosed.

The two patterns of LV dysfunction (tako-tsubo and reverse tako-tsubo) in SCM may occur in the same patient during two different episodes of stress separated by long durations. This case, for the first time, demonstrates that SCM that may start as apico-midventricular dysfunction may evolve into mid-ventricular dysfunction over hours before recovering completely over weeks.

This may explain the different patterns of myocardial dysfunction seen in SCM. The initial pattern of injury is reportedly apical, as in our patient. The temporal variation in the hitherto unknown evolution of the LV dysfunction, which has never been examined, may range from few minutes to weeks. The pattern of LV dysfunction may thus depend on the timing of the angiography in relation to speed of this evolution.

I123-meta-iodobenzylguanidine myocardial scintigraphy suggests that a base-to-apex incremental abnormality in myocardial sympathetic innervation may be a primary defect in tako-tsubo cardiomyopathy. This provokes a graded myocardial stunning, worst affecting the apex, during a catecholamine surge. The ebb in catecholamine level may interact with this gradient in a manner leading to a quicker apical recovery compared with the mid-ventricular myocardium giving rise to the reverse tako-tsubo pattern.

Supplementary material is available at European Heart Journal online.