A 76-year-old lady without prior cardiac history, presented to the hospital with sudden onset, retrosternal chest pain. Her symptoms started while rushing to post office near closing time. She was annoyed as she forgot to bring the letter with her. Her symptoms started when she returned to her car and had an argument with her husband. She was a lifelong non-smoker. She had no history of chest pain and angina. She had no significant past medical history of note.

On admission, she was afebrile, normotensive (110/70 mmHg) and slightly tachycardic (110 bpm regular). Clinical examination revealed no signs of heart failure and no other abnormality. Initial laboratory investigation included Haemoglobin 13.1 g/dl (normal range (NR) 11.5–16.5 g/dl), leucocyte count 8.1 x 10^9/l (NR 4.0–10.0 x 10^9/l), Na⁺ 142 mmol/l (NR 135–145 mmol/l), K⁺ 4.3 mmol/l (NR 3.5–5.0 mmol/l), C-reactive protein 2.6 mg/l (NR 1.0–10.0 mg/l), Mg²⁺ 0.90 mmol/l (NR 0.75–1.15 mmol/l), Ca²⁺ 2.29 mmol/l (NR 2.10–2.60 mmol/l). Cardiac troponin-T was raised 12 h after onset of chest pain at 1.6 µg/l (normal <0.03 µg/l). She was clinically and biochemically euthyroid. As electrocardiogram (ECG) on presentation showed ST segment elevation in anterior leads (V1–V4), we proceeded to perform emergency coronary angiography. This was carried out via radial approach and revealed normal coronary anatomy. Transthoracic echocardiogram (Figure 1A) showed severely impaired systolic function with apical and mid segment akinesis and hyper-dynamic basal segments of left ventricle, raising the suspicion of apical ballooning syndrome. It also showed right ventricular apical akinesis. Subsequently, cardiac magnetic resonance (CMR) imaging performed at 60 h from presentation showed impaired left ventricular function (Figure 2A and B) with apical akinesis. This was associated with high-signal intensity areas in the T2-weighted images (Figure 2D, E, F) in the mid and apical region of left ventricle suggestive of myocardial oedema or inflammation. There was no evidence of left ventricular thrombus. The diagnosis was confirmed by the absence of subendocardial late gadolinium enhancement (Figure 2c). She was commenced on Bisoprolol 2.5 mg od and angiotensin-converting enzyme (ACE) inhibitor and discharged. Follow-up echocardiogram at 16 weeks showed complete recovery of left ventricular function with no regional wall motion abnormality (Figure 1B).

Takotsubo cardiomyopathy (TTC), also called apical ballooning syndrome, broken heart syndrome and stress-induced cardiomyopathy, is an emerging entity which is characterized by transient left ventricular (commonly apical and mid apical segments) dysfunction without significant coronary lesions to account for symptoms and ECG findings. Since it was first described in 1990 in Japan, it has been reported both in Asian and Caucasian population in the last decade. The presentation is usually preceded by emotional or physiological stress and particularly affects post-menopausal women. The mechanism of pathogenesis is not well understood, but suggested hypotheses include effect of catecholamine excess, microcirculatory spasm, left ventricular outflow tract obstruction and rupture of plaque with spontaneous resolution.

Often, patients with TTC present with acute chest pain and ECG changes mimicking acute anterior infarction and thus posing a diagnostic challenge. The diagnosis is often confirmed by absence of...
obstructive coronary artery stenosis on angiography and typical shape of left ventricle (which is similar to traditional Japanese vessel used for catching octopi) with complete reversibility of ventricular dysfunction. Cardiac magnetic resonance (CMR) is emerging as diagnostic modality of choice in these patients. A recent study revealed an incidence of 4.3% of suspected ST-elevation MI and completely normal coronary arteries. Early CMR was able to establish takotsubo cardiomyopathy as the diagnosis in 10% of these patients. The typical CMR findings in Takotsubo cardiomyopathy are presence of apical akinesis with or without dynamic left ventricular outflow obstruction, myocardial oedema in apical and mid-ventricular planes on T2-weighted sequences and absence of late gadolinium enhancement (LGE). T2-weighted CMR has important role in visualizing myocardial oedema that matches with areas of dysfunction. The absence of late gadolinium enhancement (LGE) is crucial to diagnosis of TTC.

Figure 1. Echocardiogram images (apical four chamber view during systole) at presentation (A) showing apical akinesis and at 16 weeks (B) showing complete recovery of left ventricular function.

Figure 2. CMR images (A and B) showing apical akinesis (producing apical ballooning resembling Japanese traditional pot (‘tako-tsubo’ used to catch octopi) in the absence of late gadolinium enhancement (C). T2-weighted images showing normal signal intensity of the basal myocardium (D), but global oedema (arrow) in the apical and mid-ventricular myocardium matching the distribution of the LV wall motion abnormalities (E, F).
and helps to differentiate from embolic myocardial infarction (transmural or subendocardial late enhancement in single epicardial artery region) and myocarditis (subepicardial or intramyocardial gadolinium uptake\textsuperscript{4}). Moreover, CMR is invaluable in detecting complications such as apical thrombus\textsuperscript{5} and assessing LV outflow tract obstruction.

CMR remains an under-utilized tool for confirmation of this entity in the acute setting. There is a need for systematic and multicentric register of Takotsubo cardiomyopathy studied by CMR.\textsuperscript{6}

\textit{Conflict of interest:} None declared.

\section*{References}


