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Pictorial Review

Cardiac magnetic resonance assessment of takotsubo cardiomyopathy

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Takotsubo cardiomyopathy is an important condition that can be difficult to differentiate from acute coronary syndrome on the basis of clinical, electrocardiogram, and cardiac enzyme assessment alone. Although coronary angiography remains important in the acute assessment of patients with suspected takotsubo cardiomyopathy, cardiac magnetic resonance (CMR) has emerged over the last decade as an important non-invasive imaging tool in the diagnosis and follow-up of this condition. We present a review highlighting the CMR features of takotsubo cardiomyopathy and its complications with particular focus on differentiating this condition from acute myocardial infarction and myocarditis.

Introduction

Since its initial description in 1991, takotsubo cardiomyopathy has emerged as an important consideration in the differential diagnosis of acute chest pain.[1–4] It accounts for at least 2% of patients presenting with acute coronary syndrome (ACS) and is identified in 0.02% of all patients hospitalised in the United States each year.[4,5] Over 90% of reported cases are among women, with an incidence in post-menopausal women almost five-times that of women under the age of 55 years.[3,5] Due to under recognition of this condition, it is likely that the true prevalence is greater than that reported.[2,3]

The term “takotsubo cardiomyopathy” was first coined because the apical “ballooning” morphology of the left ventricle (LV) during systole resembles that of a traditional Japanese octopus fishing trap (“tako-tsubo”), which has a narrow neck and wide bottom.[2,3] It has since been referred to in the literature by a variety of names including “broken heart syndrome”, “stress-induced cardiomyopathy”, and “transient LV apical ballooning syndrome”.[2,3] Although controversy and debate persists regarding the nomenclature and pathophysiology of this condition, the American Heart Association has classified it as an acquired cardiomyopathy.[3]

Takotsubo cardiomyopathy is characterised by transient regional wall motion abnormalities that extend beyond recognised epicardial coronary distributions and can be difficult to distinguish from ACS on the basis of history, physical examination, electrocardiogram (ECG), and cardiac enzymes.[2,3] Formal coronary catheter angiography remains integral to the assessment of ACS and raises the possibility of takotsubo cardiomyopathy or acute myocarditis when there is a typical appearance on LV angiography without obstructive epicardial coronary artery disease[2,3] (Fig 1). Cardiac magnetic resonance (CMR) has emerged over the last decade as an important imaging technique in the acute assessment and

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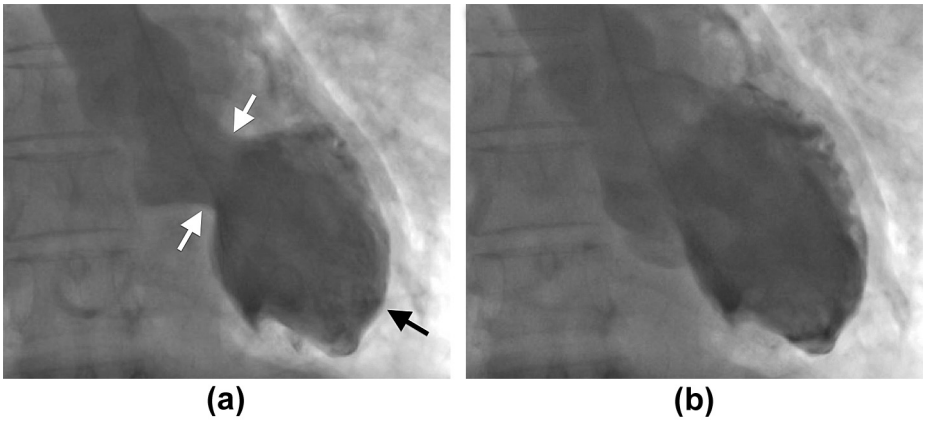

Figure 1 A 56-year-old female patient with a history of recent bereavement who presented to the emergency department with chest pain. Images during systole (a) and diastole (b) from LV angiogram performed as part of catheter angiography. This shows classical LV apical ballooning morphology (black arrow) and basal hyper-contraction (white arrows).

follow-up of these patients.[6–8] In particular, CMR provides accurate assessment of global ventricular function, defines regional wall motion abnormalities, shows the presence or absence of myocardial oedema, and detects common complications associated with takotsubo cardiomyopathy.[6–10] In addition, CMR allows differentiation of these patients from those with occult myocardial infarction without obstructive coronary disease and acute myocarditis.[6–10]

Pathophysiology

Detailed understanding of the pathophysiology of takotsubo cardiomyopathy remains lacking.[2,3] Catecholamine excess, microvascular dysfunction, and epicardial coronary spasm have all been postulated as potential mechanisms underlying this condition.[3] It is likely that catecholamine excess plays an important role as circulating plasma catecholamine levels have been shown to be significantly elevated in many patients with takotsubo cardiomyopathy.[2,3,11] Exogenous administration of catecholamine or excess catecholamine secretion by phaeochromocytomas, can provoke takotsubo cardiomyopathy.[2,3,12] It has been postulated that high levels of plasma epinephrine switches β-2 adrenoreceptor coupling from the positively inotropic G_s-cyclic adenosine monophosphate (cAMP) mediated pathway to the negatively inotropic G_i signaling pathway.[13] Interestingly, the density of sympathetic nerve endings is much higher at the apex, corresponding to the site of greatest LV dysfunction in the classical form of takotsubo cardiomyopathy.[14] Although promising, this proposed mechanism requires further validation and does not explain why postmenopausal women are affected disproportionately or why some patients present with non-classical patterns of takotsubo cardiomyopathy.[2,3]

Epicardial coronary spasm is no longer believed to be important in the pathogenesis of takotsubo cardiomyopathy, although microvascular dysfunction, as indicated by reduced coronary flow reserve on cardiac positron-emission tomography (PET) perfusion imaging, may be seen acutely in this group of patients.[2,3] Microvascular dysfunction also potentially accounts for the slow distal coronary perfusion, sometimes seen in these patients during coronary angiography.[15] Whether microvascular dysfunction contributes to the pathogenesis of takotsubo cardiomyopathy, or merely represents a secondary effect, has yet to be established.

Clinical presentation

Patients with takotsubo cardiomyopathy most commonly present with acute, sustained chest pain, which may be indistinguishable from ACS.[3,16] Less common presentations include dyspnoea, syncope, arrhythmias, and even cardiac arrest.[16] Patients without typical clinical features are sometimes identified during inpatient hospital stays during the work-up of incidental ECG or cardiac enzyme abnormalities.[3]

Characteristically takotsubo cardiomyopathy is provoked by pronounced negative emotional, psychological, or physical stress.[2,3,16] Common emotional triggers include anger and sadness, which may relate to recent arguments, bereavements, or financial difficulties.[3,16] Physical stressors include surgery, neurological disorders, severe pain, recreational cocaine use, and opiate withdrawal.[16] A number of iatrogenic triggers have been reported in association with takotsubo cardiomyopathy, including dobutamine stress imaging, exogenous catecholamine administration, and pain or anxiety related to a variety of medical procedures.[2,3] Although a preceding trigger is not always identified, a careful history must be elicited from these patients, as critical information is not always disclosed during the initial clinical assessment.[3]

ECG findings in patients with takotsubo cardiomyopathy are varied but may resemble those of ST-elevation or non-ST-elevation myocardial infarction.[2,3,17] Marked ST-elevation is seen in up to 34% of patients, most typically in the anterior leads.[17] Cardiac biomarkers are frequently elevated, usually peaking within 24-hours of presentation.[2,3,16] Levels, however, are lower than would be expected for the severity of associated wall motion