Quantitative oedema analysis using cardiac magnetic resonance T2-mapping techniques in diagnosis of takotsubo cardiomyopathy

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Takotsubo cardiomyopathy (TTC) is a reversible cardiomyopathy, often precipitated by a stressful event, with clinical features often indistinguishable from acute myocardial infarction (AMI). The syndrome is characterised as chest pain, electrocardiogram (ECG) alterations, a rise in cardiac necrosis markers, and left ventricular (LV) apical dyskinesia, all of which are reversible and are not consequences of coronary artery disease (CAD). The identification of reversible myocardial injury with the use of cardiac magnetic resonance (CMR) is possible by assessing pathologic conditions that alter the myocardial water content and consequently prolong the T2-relaxation time of the tissue. CMR T2-weighted imaging is used to detect inflammation with oedema in myocarditis and AMI. A 70-year-old female patient, with hypertension and chronic renal failure with estimated glomerular filtration rate (eGFR) 36.75 mL/min/1.73 m², presented to the Emergency Department with acute onset of severe chest pain occurring after a stressful event. The ECG revealed ST-segment elevation with associated Q waves in inferolateral leads. Cardiac necrosis markers were elevated, with a peak on the third day after the onset of symptoms. AMI was suspected, and emergency invasive coronary angiography was performed, showing no obstructive CAD. CMR contrast-free examination was performed to determine the cause of LV impairment. Apart from a standard protocol for cardiac function assessment, T2-weighted imaging techniques were used to detect myocardial oedema: standard T2-STIR images and additionally T2-maps. Cine CMR imaging confirmed LV wall motion abnormalities affecting the apex and the apical and mid segments (Fig. 1A, B), with hyperkinetic basal segments and ejection fraction (EF) of 44%. Both standard T2-STIR sequence (Fig. 1E) and myocardial T2-mapping (Fig. 1C, D) showed myocardial oedema confirmed by elevated T2-relaxation times in the affected regions: the mean T2 value measured within the apical and mid segments was 71 ± 7 ms compared with 54 ± 5.2 ms in basal segments. A follow-up CMR scan performed three months thereafter (eGFR improved to 61 mL/min/1.73 m²) showed complete restoration of global ventricular function (EF 66%) and no regional wall motion abnormalities (Fig. 2A, B). There was complete regression of myocardial oedema as shown on T2-mapping sequences (Fig. 2C, D), and no late gadolinium enhancement was observed (Fig. 2E). Standard T2-weighted imaging is limited by technical problems including surface coil intensity variation, bright signal from stagnant blood potentially interfering with elevated T2 values in the subendocardium, motion artefacts, and the subjective nature of image interpretation. Alternatively, using quantitative T2-mapping, the artefacts associated with standard T2-weighted imaging may be minimised, subjective interpretation can be reduced, and differences between tissues may be easily detected. The presented TTC illustrates the usefulness of myocardial T2-mapping CMR imaging in distinguishing the variety of potential aetiologies in patients with chest pain, troponin rise, and normal coronary arteries, who frequently represent a diagnostic challenge.

Figure 1. Symptomatic cardiac magnetic resonance scan of a patient with takotsubo cardiomyopathy. Notice the apical ballooning visualised during systole in the long axis (A, B), as well as the oedema in T2-mapping (C, D) and STIR images (E).

Figure 2. Follow-up cardiac magnetic resonance scan of a patient with takotsubo cardiomyopathy. Follow-up images show complete reversibility of the findings (A–D) with no late gadolinium enhancement (E).