Simultaneous Positron Emission Tomography/Magnetic Resonance Imaging Identifies Sustained Regional Abnormalities in Cardiac Metabolism and Function in Stress-Induced Transient Midventricular Ballooning Syndrome
A Variant of Takotsubo Cardiomyopathy

Tareq Ibrahim, MD; Stephan G. Nekolla, PhD; Nicolas Langwieser, MD; Christoph Rischpler, MD; Philipp Groha, MD; Karl-Ludwig Laugwitz, MD; Markus Schwaiger, MD

A 70-year-old white woman with a history of hyperlipidemia, hypertension, and former smoking and a family history of premature coronary artery disease presented with acute substernal chest pain. She complained of progressive exertional dyspnea during the preceding weeks and of being under personal stress during this time. Her initial ECG showed ST-segment elevation of a maximum 0.2 mV and a reduced R-wave progression in the anteroseptal leads (V1–3). Her troponin T level was moderately elevated (0.3 ng/mL), with normal creatine kinase (CK) and CK-myocardial band (CK-MB) fraction. Cardiac catheterization was performed and showed angiographically normal epicardial coronary arteries. However, the left ventriculogram demonstrated midventricular dilatation and akinesis with a hypercontractile apex and base compatible with midventricular ballooning syndrome, a variant of Takotsubo cardiomyopathy (TTC) (Figure 1A and 1B and Movie I in the online-only Data Supplement). Transthoracic echocardiographic monitoring during the next couple of days revealed no substantial changes in midventricular akinesis. Six days after the onset of symptoms, the patient underwent a simultaneous positron emission tomography (PET)/magnetic resonance (MR) examination of the heart performed on a whole-body integrated PET/MR scanner (Biograph mMR; Siemens Healthcare, Erlangen, Germany) using F-18 fluorodeoxyglucose (FDG) as a tracer. For PET scanning, the patient was prepared with a high-fat, low-carbohydrate diet the entire day before and a 12-hour fasting period before the examination. On cine-MR imaging, left ventricular midventricular dysfunction was unimproved (Figure 2 and Movie II in the online-only Data Supplement) and delayed-contrast MR imaging did not depict any myocardial hyperenhancement (Figure 3A and 3B). On PET scanning, suppressed FDG uptake within normal myocardium was noted, but a markedly increased glucose metabolism within the dysfunctional midventricular segments was indicated as well (Figure 3C and 3D). Thus, combined PET/MR findings are compatible with stunned but viable myocardium that typically emerge during the acute phase of TTC (Movie III in the online-only Data Supplement). The patient was initiated on an

Figure 1. The left ventriculogram in the right anterior oblique view in diastole (A) and systole (B) demonstrates akinesis of the midventricular segments with hypercontractility of the apical and basal segments during systole compatible with transient midventricular ballooning syndrome.

From the Medizinische Klinik, Klinikum rechts der Isar der Technischen Universität München, Germany (T.I., N.L., P.G., K.-L.L.); and the Nuklearmedizinische Klinik und Poliklinik, Klinikum rechts der Isar der Technischen Universität München, Germany (S.G.N., C.R., M.S.).

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Correspondence to Tareq Ibrahim, MD, Medizinische Klinik, Technische Universität München, Ismaningerstrasse 22, 81675 München, Germany. E-mail T.Ibrahim@lrz.tu-muenchen.de

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angiotensin-converting enzyme inhibitor and β-blocker medication, and transthoracic echocardiography 8 weeks later confirmed complete normalization of left ventricular function without any regional wall motion abnormalities while the patient was asymptomatic.

Transient apical ballooning syndrome, also known as Takotsubo or stress cardiomyopathy, is a reversible condition that occurs predominantly in postmenopausal women and is estimated to represent 1% to 2% of patients presenting with troponin-positive acute coronary syndromes.1 However, about 17% of patients with TTC present with a preserved apical contraction, referred to as atypical or midventricular ballooning syndrome.2 Although the underlying pathophysiology of this cardiomyopathy is not fully elucidated, neuro-

Figure 2. Cine-magnetic resonance imaging in a 4-chamber view during diastole (A) and systole (B) 6 days after admission shows the unimproved midventricular akinesis with a spadelike ballooning during systole and impairment of global left ventricular function (ejection fraction, 46%).

Figure 3. Contrast-enhanced cardiovascular magnetic resonance imaging in 2-chamber (A) and 4-chamber (B) orientations revealed no evidence of delayed myocardial hyperenhancement. Fusion overlay of fluorodeoxyglucose–positron emission tomography and magnetic resonance imaging in 2-chamber (C) and 4-chamber (D) orientations demonstrate the suppressed glucose uptake within normal myocardium but a preserved glucose metabolism within the midventricular myocardium that correlates closely with the extent of wall motion disturbance compatible with myocardial stunning typically associated with Takotsubo cardiomyopathy.
genic myocardial stunning that is mediated by stress-induced abnormal catecholamine dynamics seems to be the most relevant hypothesis. Cardiovascular MR imaging may provide important functional information helpful in establishing the diagnosis of TTC, and a lack of contrast enhancement in the presence of wall motion abnormality may be interpreted as myocardial stunning, and hence may be a predictor of wall motion recovery. Postischemic or stunned myocardium is characterized metabolically by a transient shift from fatty acid metabolism toward increased glucose utilization as a compensatory energy production, and PET scanning has been demonstrated successfully to display delayed regional recovery of both fatty acid and glucose metabolism, also termed ischemic memory. A recent study in acute TTC using FDG-PET scanning after glucose load has reported that dysfunctional regions exhibit an altered but reduced glucose metabolism relative to remote myocardium and perfusion that normalized on follow-up imaging after several weeks. However, by a targeted suppression of FDG uptake within normal myocardium using a high-fat, low-carbohydrate diet, FDG-PET scanning performed early after symptom onset depicts clearly an accelerated glucose metabolism within the dysfunctional midventricular myocardium as defined by MR imaging. The combined information of integrated PET/MR imaging in this variant of TTC allowed us to delineate neurogenic myocardial stunning. This type of multimodal imaging may provide further insight into the underlying pathophysiology of stress-induced cardiomyopathy.

Disclosures
None.

References
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