KARDIOLOGIA POLSKA

Polish Heart Journal
The Official Peer-reviewed Journal of the Polish Cardiac Society since 1957

Online first
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ISSN 0022–9032 e-ISSN 1897–4279

# Myocardial infarction with nonobstructive coronary arteries in a woman: Takotsubo cardiomyopathy or true myocardial infarction?

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**Article type:** Clinical vignette

**Received:** June 1, 2022

**Accepted:** September 8, 2022

**Early publication date:** September 27, 2022

**Myocardial infarction with nonobstructive coronary arteries in a woman: Takotsubo cardiomyopathy or true myocardial infarction?**

**Short title:** Myocardial infarction with non-obstructive coronary arteries in a woman

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Myocardial infarction with nonobstructive coronary arteries (MINOCA) is a working diagnosis for patients with manifestation of myocardial infarction, no significant coronary stenosis on angiography and no other specific pathology responsible for acute presentation [1]. Patients with MINOCA constitute a diagnostic and therapeutic challenge in everyday practice. Diagnostic algorithms recommend several tests, including cardiac magnetic resonance (CMR) [1]. The recent studies underscore the important role of early CMR (median: 3 days from index event), which

allows for establishing diagnosis in 77% of patients, with Takotsubo cardiomyopathy (TC; 33%), myocardial infarction (22%) and myocarditis (17%) as most frequent pathologies [2].

We present this case to underscore the role of: (1) early CMR in the diagnostic algorithm of MINOCA; (2) ischemic cause of MINOCA in women.

A 52-year-old hypertensive woman presented to the emergency department with a 24-hour recurrent chest pain. Electrocardiogram showed abnormalities typical of inferior ST- segment elevation myocardial infarction. Coronary angiography revealed minimal luminal irregularities (Supplementary material, *Videos S1–S6*). In the laboratory tests the troponin T and CK-MB (creatine kinase-muscle/brain) levels were elevated (695 pg/ml, 44.6 U/l), while other parameters were within the normal range. Echocardiography showed akinetic apex and apical segments of the left ventricle with mildly reduced left ventricular ejection fraction (LVEF) — 45%. The patient reported emotional stress in the previous weeks. Given the female sex, the history of emotional stress, no obstructive coronary stenosis on angiography and the echocardiographic presentation, the initial diagnosis of TC was established. The control echocardiography performed on day 4 showed improvement in LVEF (52%) with partial resolution of wall motion abnormalities, which was another argument for the diagnosis of TC. To confirm the initial diagnosis, CMR was performed on day 4.

CMR revealed preserved LVEF (50%) and akinesis of apical inferior segment and adjacent fragment of apical septal segment of the left ventricle. Within the akinetic segments oedema in T2-weighted imaging with fat suppression and subendocardial lesion in late gadolinium enhancement sequences were detected (Figure 1A–D). Stress CMR with regadenoson showed subendocardial ischemia (Figure 1E and Supplementary material, *Video S7*). Ultimately, the diagnosis was changed into myocardial infarction, 12-month dual antiplatelet therapy (aspirin and clopidogrel) along with a statin, beta- adrenergic receptor blocker and angiotensin-converting enzyme inhibitor were recommended and the patient was referred for cardiac rehabilitation. The control MR performed 12 weeks after index event confirmed the presence of post-myocardial scar within the apical inferior and apical septal segments (Figure 1F).

In our case, early CMR allowed for establishment of a proper diagnosis and implementation of targeted therapy and rehabilitation. It is estimated that more than one third MINOCA patients does not receive a proper pharmacotherapy [3]. Our patient at first presented as typical TC and her sex and the history of emotional stress were major arguments for that. Female sex is a strong predictor

of TC, accounting for the majority of points in InterTAK score. However, there are studies that show that it is myocardial infarction, not TC, that constitutes the most frequent pathology in middle-aged women with MINOCA [4]. Another challenging problem is possible co-occurrence of myocardial infarction and TC, which has previously been described in literature [5]. Therefore, each patient with MINOCA requires multimodality assessment, including early CMR and advanced coronary techniques (not performed in our case because of the presumption of TC).

**Article information**

**Conflict of interest:** None declared.

**Funding:** None.



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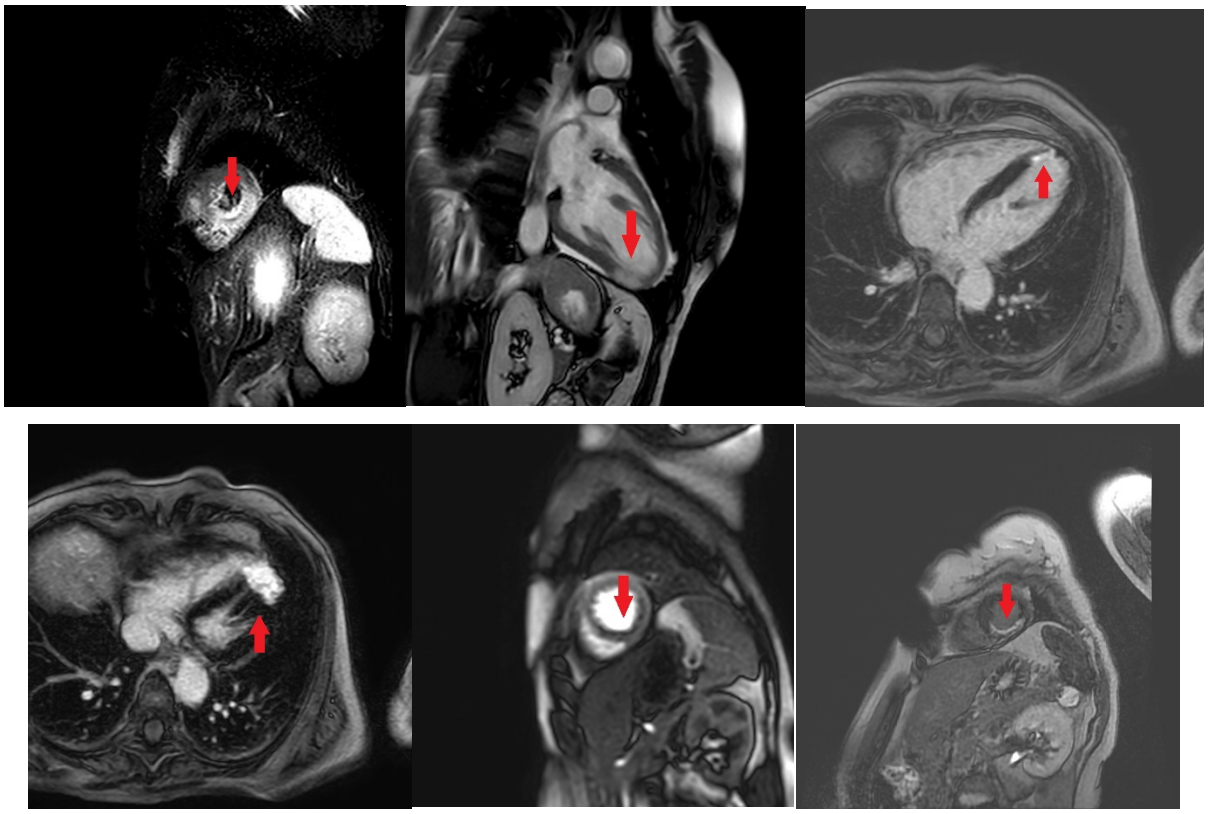

**Figure 1. A–E.** Initial examination. **A.** T2-STIR image, short axis view: transmural signal hyperintensity (myocardial oedema) in apical inferior and adjacent fragment of the apical septal segment of the left ventricle. **B.** sBTF cine imaging, 2-chamber view: transmural signal hyperintensity (myocardial oedema) in the apical inferior segment of the left ventricle. **C.** 4-chamber view: subendocardial distribution of late gadolinium enhancement (LGE) within the apical septal segment. **D.** 4-chamber view: LGE within the apical inferior and apical septal segment. **E.** Stress CMR with regadenoson, short-axis view: impaired subendocardial perfusion within the apical inferior and apical septal segment. Follow-up

examination. **F.** Follow-up examination, short axis view: subendocardial LGE within the apical inferior and apical septal segments of the left ventricle (post-myocardial scar)