Rare case of obstructive hypertrophic cardiomyopathy imitating takotsubo syndrome

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A 45-year-old man was urgently admitted to the cardiology department due to acute chest pain. Electrocardiography (ECG) showed dynamic (absent on outpatient ECG 7 days earlier) changes: negative T waves in the precordial leads, and prolonged QT interval but no signs of left ventricular hypertrophy (Figure 1A). Laboratory tests showed elevated troponin T at 361 pg/ml (reference range [RR], <13 pg/ml), N-terminal pro-B-type natriuretic peptide 4275.0 pg/ml (RR, <121.0 pg/ml) and C-reactive protein 58.70 mg/l (RR, <6.0 mg/l). Urgent transthoracic echocardiography (TTE) showed akinesis of the apical segments and hyperkinesis of the thickened basal segments of the left ventricle (LV) with reduced ejection fraction (35%) (Figure 1B), systolic anterior motion of the mitral valve, and a turbulent flow in the left ventricular outflow tract with a peak velocity of 5.5 m/s (Figure 1C). Urgent coronary angiography performed due to a suspicion of acute coronary syndrome showed no coronary artery stenosis. Potentially life-threatening [1] takotsubo syndrome (TTS) was suspected based on apical ballooning of the LV, although with no predisposing emotional or physical stress and low probability according to the InterTAK Diagnostic Score [2]. Beta-blocker and fluid therapy due to left ventricular outflow track obstruction (LVOTO) and angiotensin-converting enzyme inhibitor due to LV dysfunction were implemented, but beta-blocker was discontinued shortly thereafter due to bradycardia. Cardiac magnetic resonance imaging (MRI) performed to exclude myocarditis showed a myocardial edema (typical of TTS) in the apical segments of the LV (Figure 1D), but also areas of late gadolinium enhancement (typical for hypertrophic cardiomyopathy [HCM] but rare in TTS) involving the interventricular septum (Figure 1E). Over the following days, the patient's condition remained stable. In a follow-up TTE 5 days after admission, an improvement of LV systolic function and the disappearance of regional wall motion abnormalities, along with a regression of LVOTO, were noted, but on control ECG T wave inversion persisted. After a regression of apical ballooning, attention was drawn to asymmetric LV hypertrophy and to the mitral valve apparatus with its significantly elongated anterior leaflet (Figure 1F) suggestive of an HCM diagnosis, reinforced also by a typical location of late gadolinium enhancement on cardiac MRI. Although apical ballooning with dynamic LVOTO can be a typical presentation of TTS (severe LVOTO in TTS may even provoke rapid transition of wall motion abnormalities) [3], it has been postulated recently that this phenotype may result also from HCM with sudden LVOTO and subsequent severe afterload mismatch [4]. This constitutes further evidence of HCM playing the role of the great 'masquerader' [5], and shows that in a case of apical ballooning with LVOTO, the emphasis should be put on a detailed TTE with attention paid to subtle features of mitral valve apparatus and on tissue characterization on cardiac MRI in pursuit

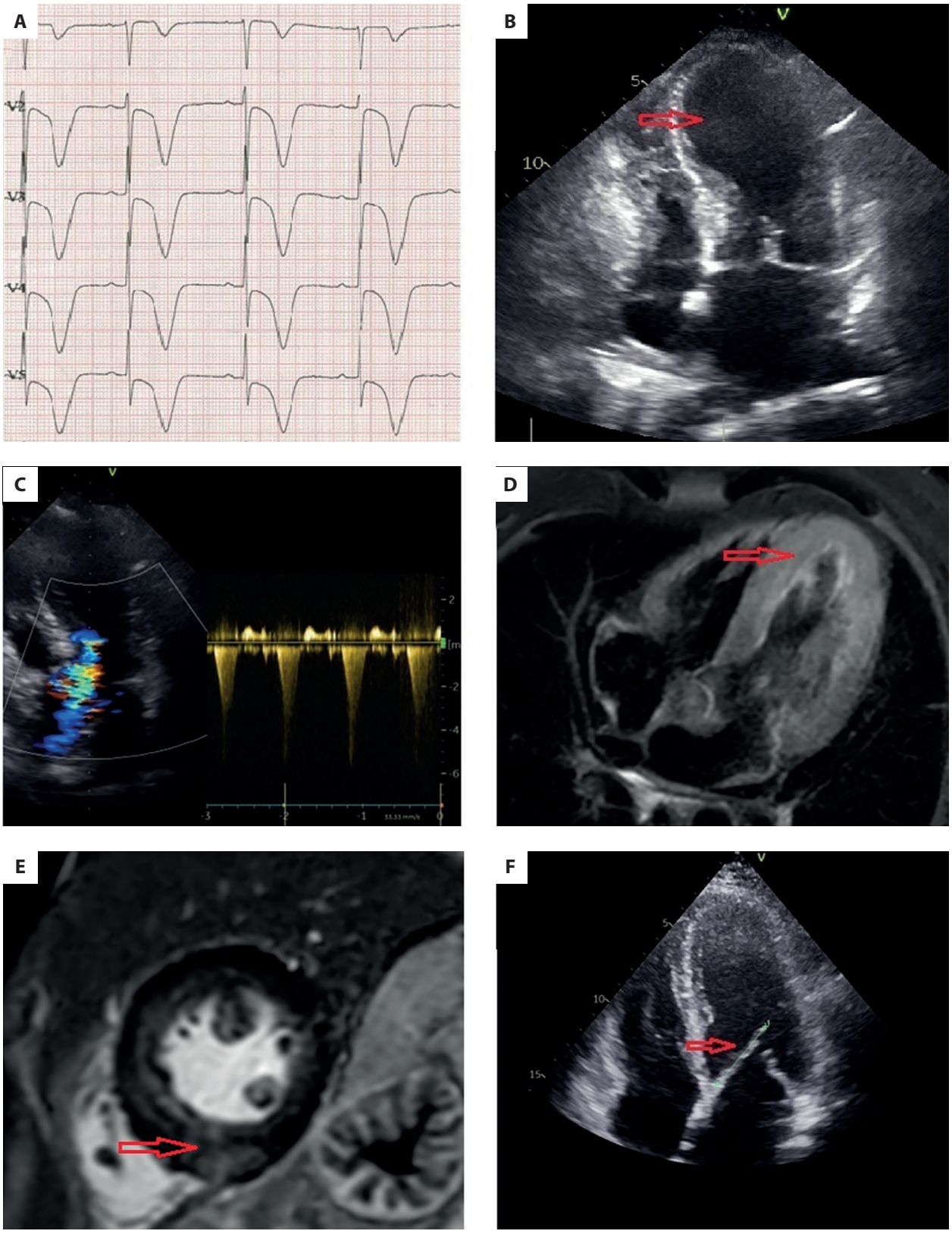

Figure 1. A. Electrocardiogram showing negative T waves in precordial leads and prolonged QT interval. **B.** Echocardiogram (4-chamber view) showing akinesis of apical segments and hyperkinesis of thickened basal segments of the left ventricle and its significantly reduced ejection fraction (apical ballooning) (red arrow). **C.** Echocardiogram (5-chamber view) showing left ventricular outflow track obstruction with a peak velocity of flow at 5.5 m/s. **D.** Cardiac magnetic resonance imaging demonstrating a large area of transmural hyperintense signal on STIR images (4CH) in apical and medial segments representing myocardial edema (red arrow). **E.** Cardiac magnetic resonance imaging presenting a patchy/streaky intramyocardial pattern of late gadolinium enhancement in the septum and at the right ventricular insertion — myocardial fibrosis. **F.** Echocardiogram (4-chamber view) showing elongation of the anterior leaflet of the mitral valve up to 38 mm (red arrow)

of the correct diagnosis, leading to implementation of the necessary steps including pharmacological prevention of LVOTO recurrence and death risk stratification with the use of dedicated tools. A timely diagnosis is important considering the cases of HCM with sudden LVOTO complicated by apical ballooning with refractory shock treated with urgent myectomy that have been reported in the literature [4].

Article information

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