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Letter to the Editor

Transient ST-segment elevation in lead aVR associated with tako-tsubo cardiomyopathy

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Abstract

ST-segment elevation in lead aVR in patients with angina at rest can be related to transmural ischemia of the basal part of the interventricular septum, frequently due to left main or multivessel coronary disease. However, this electrocardiographic (ECG) sign may also occur in other clinical conditions manifesting by acute chest pain. We present a case of a 76-year-old Caucasian woman with transient ST-segment elevation in lead aVR associated with tako-tsubo cardiomyopathy. Our report seems to confirm the hypothesis about the role of reversible myocardial ischemia involving the basal part of the interventricular septum in the pathogenesis of tako-tsubo cardiomyopathy. In conclusion, ST-segment elevation in lead aVR in patients with a clinical presentation of acute coronary syndrome may be not related to coronary artery disease. Tako-tsubo cardiomyopathy should be considered among the causes of ST-segment elevation in lead aVR in patients with angina at rest. Further studies are needed to evaluate the occurrence and importance of this ECG sign in patients with tako-tsubo cardiomyopathy.

Keywords: Electrocardiography; Lead aVR; Apical ballooning; Tako-tsubo cardiomyopathy

1. Introduction

ST-segment elevation in lead aVR in patients with angina at rest can be related to transmural ischemia of the basal part of the interventricular septum, frequently due to left main (LMCA) and/or multivessel coronary disease [1,2]. However, this electrocardiographic (ECG) sign may also occur in other clinical conditions manifesting by acute chest pain [1,2].

2. Case presentation

A 76-year-old Caucasian woman with arterial hypertension and hypercholesterolemia, treated with adjuvant tamoxifen monotherapy, because of grade 2, infiltrating ductal carcinoma of the left breast, was admitted to the emergency department with severe angina at rest and exertional dyspnea (NYHA class III), triggered by acute emotional stress.

On admission, her blood pressure was 160/90 mmHg, and pulse rate was 76 beats/min. Electrocardiography showed ST-segment elevation in leads aVR and V_1 (Fig. 1a). The corrected QT interval (QTc) was 400 ms. Serum levels of troponin I, creatine kinase and MB isoenzyme were: 3.23 ng/mL (normal <0.1 ng/mL), 153 U/L (normal <145 U/L), and 19 U/L (normal <24 U/L), respectively. NT-proBNP was 2241 pg/mL (normal <450 pg/mL).

The ECG recorded 4 h later showed QS-waves in leads V_5–V_6, ST-segment elevation in V_3–V_6, and biphasic T-waves in V_5–V_6 (Fig. 1b). The QTc was 455 ms. Interestingly, previously observed ST-segment changes in leads aVR and V_1 were absent (Fig. 1b).

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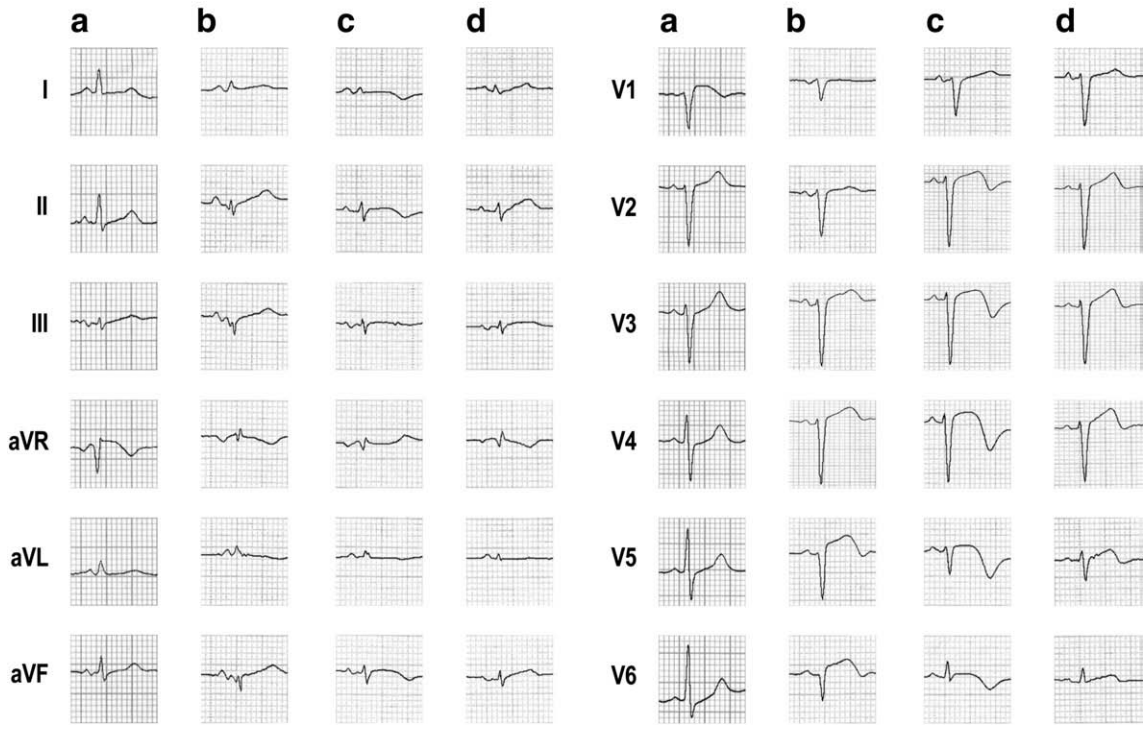

Fig. 1. Time course of electrocardiographic changes: 12-lead ECG on admission (a), after 4 h (b), on day 7 of hospitalization (c), and 4 weeks after hospital discharge (d).

Coronary angiography revealed no significant coronary stenoses (Fig. 2a,b). Left ventriculography, however, disclosed apical ballooning with concomitant compensatory basal hyperkinesis (Fig. 2c,d). Transthoracic echocardiography (TTE) confirmed the presence of left ventricular dyssynergy with the ejection fraction (LVEF) 35%. In addition, the

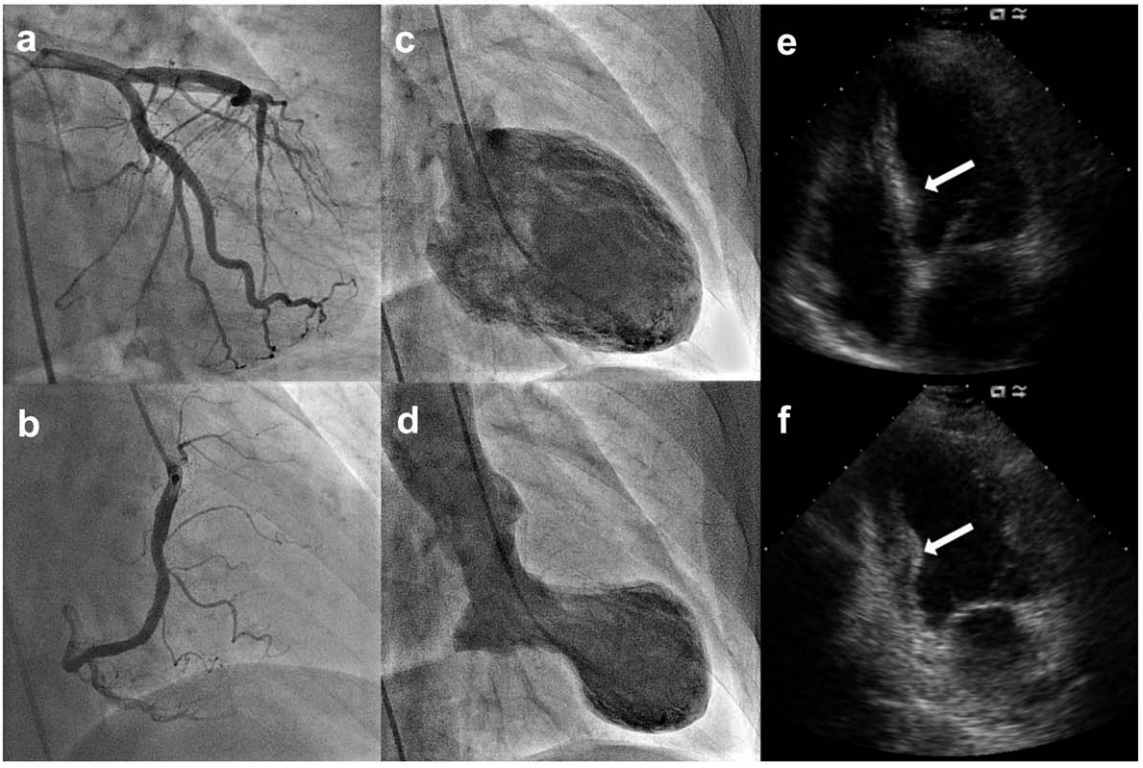

Fig. 2. Coronary angiography revealed no significant stenosis in the left (a) and right (b) coronary arteries. Left ventriculography disclosed apical ballooning with basal hyperkinesis (c,d). Transthoracic echocardiography showed a sigmoid deformity of the interventricular septum (e,f).