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Case report

Rupture of free wall of left ventricle in a patient with takotsubo cardiomyopathy

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ABSTRACT

Takotsubo cardiomyopathy (TTC) belongs to rare heart disease with symptoms imitating acute coronary syndrome with ST segment elevation. Usually, it occurs predominantly in postmenopausal women. In most cases, the disease has an uncomplicated course, but some patients may develop severe complications (e.g. cardiogenic shock, severe heart failure, malignant arrhythmia, thromboembolism or myocardial wall rupture). This case report describes an 82-year-old man with TTC complicated rupture of the left ventricular free wall and interventricular septum. This rare complication often leads to sudden death. Due to the unknown etiopathogenesis, the treatment is symptomatic. Pharmacologic therapy is still a matter of debate and further research.

Introduction

Takotsubo cardiomyopathy, also called stress or stress-induced cardiomyopathy or broken-heart syndrome, was first described in 1983 by Japanese author K. Dote. Clinical, electrocardiographical and laboratory image imitates acute coronary syndrome. There is a characteristic morphology for TTC, where in most of the patients we can find akinesis of apical segments of left ventricle without significant coronary artery stenosis. TTC is often induced by stress and its course is usually uncomplicated. Some patients, however, may develop life-threatening complications such as cardiogenic shock, thromboembolism, ventricular arrhythmias and left ventricle rupture.

We present the case of a man with TTC complicated by rupture of the left ventricle free wall and apical segment of the interventricular septum.

Case report

An 82-year-old male was admitted to our clinic with interscapular pain, breathlessness and subsided attack of weakness. He was a non-smoker, treated for no disease so far. He never experienced these symptoms before. By the time he had

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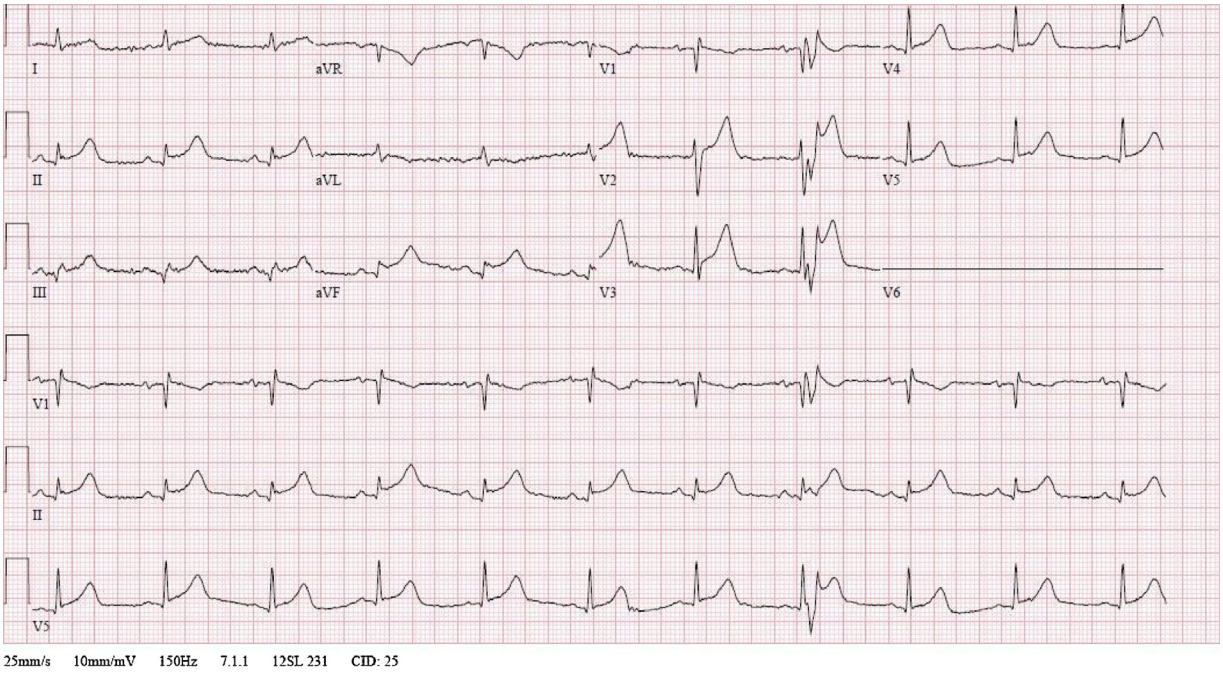

Fig. 1 – ECG in admission to the hospital.

been admitted at intensive care unit he was without pain, his heart rate was 61/min, his blood pressure was 86/56 mmHg with eupnea and no sign of congestive heart failure. Laboratory shows mild normocytic anemia and leukocytosis, normal CRP, normal blood minerals, cholesterol. Creatine phosphokinase was 2.92 μkat/l and high-sensitivity troponin T was 543 ng/l (cut-off value of 14 ng/l). ECG shows ST segment elevation in leads II, III, aVF and V1–V5 (Fig. 1), without progression in time. Due to ECG changes and patient's symptoms, coronarography and left ventricle ventriculography were indicated. These procedures reveal hyperkinesis in basal ventricular segments, akinetic apical left ventricular segments, ejection fraction of left ventricle 25% (Fig. 2) and no severe coronary atherosclerosis. Gradient between left ventricle and aorta was 35/20 mm Hg. Based on all findings, the diagnosis of an apical TTC was made. The patient was then stable with no sign of heart failure.

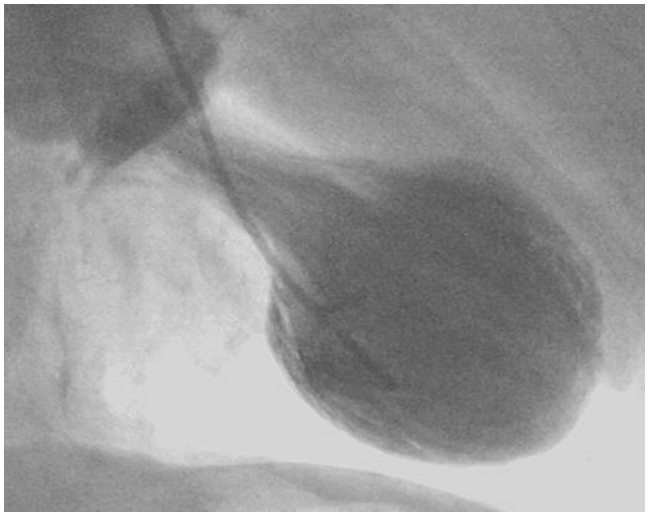

Fig. 2 – The ventriculography of left ventricle.

After 32 h in hospital, the patient suddenly died due to left ventricle free wall rupture with heart tamponade, which led to pulseless electrical activity. An autopsy revealed mainly hemopericardium (150 ml) due to 2 mm wide rupture in apical segment of the left ventricle free wall. Another tiny rupture was identified in apical part of interventricular septum and in trabecular part of the muscle.

There were atherosclerotic changes with stenosis up to 50% inside coronary arteries. Microscopic images show interstitial hemorrhagia with fibrin deposits in myocardial muscle and epicardial fat. There was also so-called ‘‘waving’’ identified in myocardium and single deposits of coagulation necrosis with some leukocytes infiltration without ‘‘contraction bands’’ (Fig. 3). Neither acute occlusion of coronary artery nor chronic occlusion by a plate could be demonstrated and necrosis was spotty in distribution and involved individual cardiomyocytes or small groups.

Discussion

TTC is a heart disease with low incidence and not exactly clarified etiopathogenesis so far. Various studies reported that TTC in Europe was diagnosed in 0.7–2.5% patients with suspicion of acute coronary syndrome with ST elevation [1,3–5]. By Mayo clinic criterion from 2004 [21], TTC is characterized by new originated changes on ECG (ST elevations and/or inversion of T waves) and/or elevation of troponin T, reversible akinesis or dyskinesis of middle segments of left ventricle (eventually with involvement of apex), absence of coronary artery obstruction and another etiology of state

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