

CASE REPORT

Medicine Science 2018;7(2):441-2

Takotsubo cardiomyopathy in a patient with subacute sclerosing panencephalitis and type 1 diabetes mellitus

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Received 15 December 2017; Accepted 27 December 2017

Available online .27.03.2018 with doi: 10.5455/medscience.2017.06.8762

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Abstract

Takotsubo cardiomyopathy (TTC) is a rare clinical syndrome and is associated with intense emotional and physical stress. It has been demonstrated that TTC may be triggered by sepsis, however there is no data relation between type 1 diabetes mellitus (DM), subacute sclerosing panencephalitis (SSPE) and TTC. We present a patient with TTC who was previously diagnosed SSPE and type 1 DM and was admitted to emergency service with sepsis which is triggered by aspiration pneumonia.

Keywords: Takotsubo cardiomyopathy, subacute sclerosing panencephalitis, type 1 diabetes mellitus

Introduction

Takotsubo cardiomyopathy (TTC) is characterized by transient left ventricular (LV) dysfunction, myocardial infarction-like ST elevation but absence of any obstructive coronary artery disease [1]. Although several pathological mechanisms have been proposed, the pathophysiology of TTC is not well understood [2,3]. Previous studies have demonstrated relationship between sepsis and TTC but there were no cases which have been reported relationship between TTC, Type 1 diabetes mellitus (DM) and subacute sclerosing panencephalitis (SSPE) [4]. Herein, we present a patient with history of SSPE and type 1 DM who presented with sepsis and TTC.

Case Report

A 19-year-old male patient with a past medical history of SSPE and type 1 DM was admitted to the emergency service with severe respiratory distress, fever and altered mentation. SSPE was diagnosed 9 years ago and DM was diagnosed 4 years ago. On admission his blood pressure was 100/60 mmHg, heart rate was 112 bpm, respiratory rate was 26 per minute, temperature was 40.2°C and oxygen saturation was 85% on room air. On examination, auscultation revealed bilateral inspiratory crackles till middle zones of the lungs. Admission ECG revealed ST elevation in D1, AVL V2-6 derivations (Figure 1). Transthoracic echocardiography (TTE) showed LV apical ballooning with hyperkinesia of basal segment of LV wall and mildly systolic dysfunction (EF: 45%). He was immediately transferred to cardiac

catheterization laboratory and coronary angiography revealed normal coronary arteries (Figure 2). Thorax computed tomography was in accordance with aspiration pneumonia. Clinical scenario was compatible with sepsis triggered by aspiration pneumonia. He was intubated and transferred to intensive care unit. Invasive mechanical ventilatory support was given to decrease the work of breathing but despite aggressive medical treatment including intravenous antibiotics and inotropes, multiorgan failure occurred. Serum Alanin aminotransferase increased to 7000 U/L, Aspartat aminotransferase 2000 U/L, International normalized ratio 4, Creatinine 3,7 mg/dl CRP 42 mg/dl and WBC 19000/mm³, peak level troponin T (high sensitive) 1500 IU/L and CKMB 40ng/dl. Although deterioration of his general state, ST elevation returned towards baseline at third day. During follow-up in the intensive care unit TTE was repeated several times and it revealed complete resolution of apical ballooning and systolic dysfunction after 35 days of admission (Figure 2). Unfortunately, he developed septic shock and expired on 41th day of admission.

Discussion

Takotsubo cardiomyopathy may mimic acute coronary syndrome and usually presents with chest pain, ST-T-segment changes at the ECG and mildly elevated cardiac enzymes. TTC is a transient syndrome and resolution of symptoms with complete echocardiographic and clinical recovery is expected within 1 to 4 weeks of presentation.

Although several pathological mechanisms have been proposed like catecholamine overload, alterations in the coronary microcirculation, vasospasm, and pro-inflammatory processes, the exact mechanism of TTC is not well understood. Activation

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of the sympathetic nervous system and elevated levels of catecholamines in sepsis can cause myocardial injury and segmental wall motion abnormality. Additive or synergistic effects of cytokines, prostanoids, and nitric oxide all together likely explain the myocardial depression seen in patients with sepsis [5].

TTC is usually triggered by an acute medical illness or by intense emotional or physical stress [6]. We believe that in our case TTC was triggered by sepsis which started as aspiration pneumonia. Aspiration pneumonia is relatively common toward end stage SSPE. Additionally, diffuse microvascular dysfunction due to diabetes mellitus and emotional stress due to the SSPE might have contributed to the clinical scenario. In the present case, reversible apical ballooning, ECG and clinical findings were compatible with TTC despite the markedly elevated cardiac enzymes. Multi-organ failure and deterioration of his general state may be due to markedly increased cardiac enzymes and late improvement of apical ballooning.

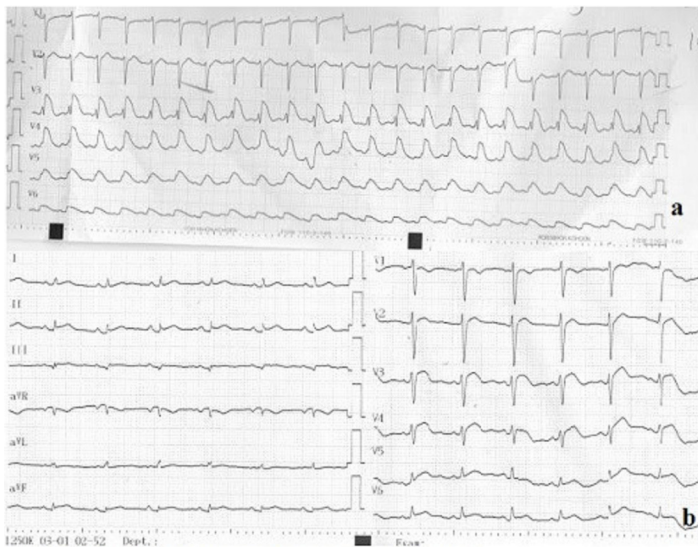


Figure 1. ECG samples of patient obtained at admission (a) and 3 days later (b)

Conclusion

We report a case with TTC which is triggered by sepsis with coexistence of SSPE and type 1 DM was not reported in the literature. Diagnosis of TTC may easily be confused with acute myocardial infarction. Physicians, particularly those dealing patients in the emergency conditions should keep in mind that presence of ST elevation on ECG can not preclude TTC especially in the setting of accompanying comorbidities as in the present case. Nevertheless, coronary angiography should not be delayed. We believe, the present case report contributes to growing body of literature in this field.

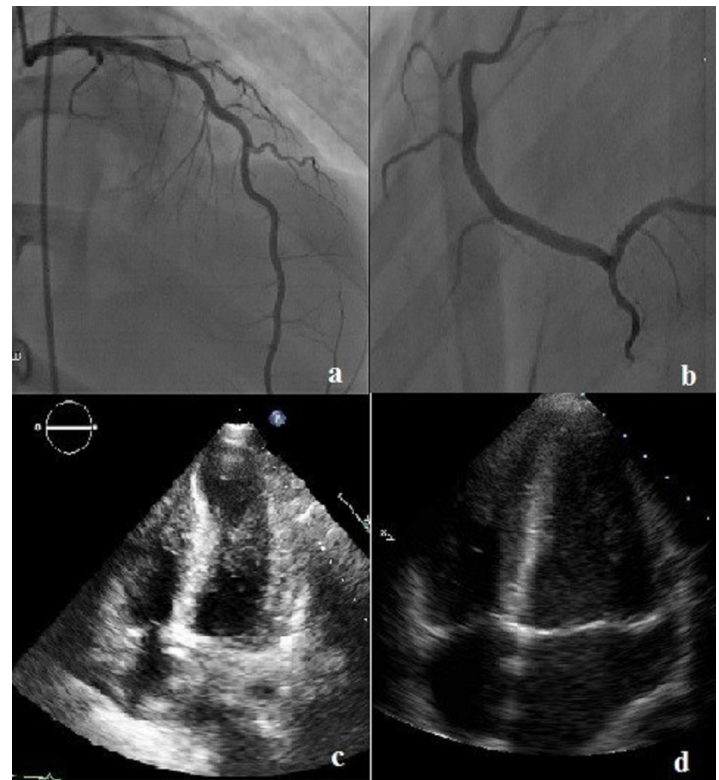


Figure 2 Coronary angiography of patient (a,b); apical ballooning at admission (c) and complete resolution of apical ballooning (d)

Conflict of interest

There is no conflict of interest related to the study.

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