Takotsubo Cardiomyopathy after Acute Diarrhea

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Abstract

Takotsubo cardiomyopathy is a recently described syndrome characterized by transient left ventricular dysfunction, mimicking an acute coronary syndrome and usually precipitated by a physical or emotional stress. We report the first case of Takotsubo cardiomyopathy after acute diarrhea in a man. It may be argued that severe diarrhea in predisposed individuals may cause an acute stress resulting in increased sympathetic activity leading to this syndrome. Probably the relationship between the adrenergic system and the heart is more complex than general thought and the stimuli which favor an autonomic imbalance and precipitate the syndrome are very disparate in clinical practice.

Key words: Takotsubo, diarrhea, stress, adrenergic system

A 71-year-old man, a smoker, who suffered from COPD and arterial hypertension, presented to the emergency department due to central chest pain. In the previous four days he had suffered from acute diarrhea.

Upon admission the patient was symptomatic for chest pain. His blood pressure was 120/80 mmHg and clinical examination was unremarkable except for a 3/6 L systolic murmur louder at the mitral outbreak. EKG on admission showed sinus tachycardia and left bundle branch block that was not present in a previously recorded EKG (Fig. 1). Markers of myocardial necrosis were mildly elevated (cTnI max 2.61 ng/mL). The patient was taken to the cardiology department and an echocardiogram showed a normal left ventricle (LV) size, increased wall thickness, akinesis of all mid and apical segments with basal segments hyperkinesis, systolic dysfunction (EF 35%), increased end-diastolic LV pressure and a LV outflow tract (OT) obstruction (peak velocity 2.5 m/s) (Fig. 2). These findings were suggestive of Takotsubo cardiomyopathy. He underwent coronary angiography that excluded significant coronary artery stenosis and ventricular angiography, confirming the echocardiographical data (Fig. 3). He was treated with beta-blockers, ACE-inhibitor, statins, heparin and ASA. After six days the patient was discharged in good clinical condition and no adverse events occurred during hospitalization. The echocardiogram before discharge, showed complete normalization of segmental kinesis and systolic function (EF 60%), and a reduction of LVOT velocity (1.5 m/s) (Fig. 4). A subsequently performed cardiac MRI excluded the diagnosis of myocarditis. In the six-month follow-up the patient was doing well and had not experienced any other episode of chest pain or shortness of breath.

Discussion

Takotsubo cardiomyopathy, also known as apical ballooning syndrome or stress-induced cardiomyopathy, is a recently described syndrome characterized by transient left ventricular dysfunction, which mimicks acute coronary syndrome and is usually precipitated by physical or emotional stress. The syndrome is usually self limited and complete recovery can be achieved in two to three weeks (1, 2). The clinical presentation of the present case shows the typical symptoms of Takotsubo cardiomyopathy, which are chest pain and/or dyspnea associated with new onset EKG changes (ST segment elevation or T wave inversion) (3).

However, in our patient the triggering condition was quite unusual; in fact, here we report the first case of neurogenic...
stunned myocardium after acute diarrhea in a man. There are a few case reports in the literature which discuss Tako-tsubo cardiomyopathy following vomiting in women (4). It may be argued that severe diarrhea in predisposed individuals may cause an acute stress resulting in increased sympathetic activity leading to this syndrome. Moreover the interaction between the autonomic system and the gastrointestinal tract is well known to frequently result in psychosomatic gastrointestinal disorders. It is likely that the relationship between the adrenergic system and the heart is more complex than expected and the stimuli which favour an autonomic imbalance and precipitate this syndrome are very disparate in
clinical practice. Therefore, rather than to define all of the possible stimuli that can induce this syndrome, the true challenge for cardiologists is to determine why a certain individual develops a stress cardiomyopathy while others do not.

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References