Reversible left ventricular dysfunction resembling Takotsubo syndrome after self-injection of adrenaline

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Transient left ventricular (LV) ballooning syndrome, or Takotsubo syndrome, is characterized by a reversible LV dysfunction. The pathophysiology has not been fully elucidated, but an excess of catecholamines seems to have an essential role. The case of a 27-year-old man who developed transient LV dysfunction resembling Takotsubo syndrome after self-injection of adrenaline is described. The present case may provide additional evidence to the hypothesis of excess sympathetic activation in LV ballooning syndrome.

Key Words: *Cardiogenic shock; Cardiomyopathy; Catecholamine*

Transient left ventricular (LV) ballooning syndrome, or Takotsubo syndrome, is characterized by a reversible LV dysfunction in the absence of obstructive coronary artery disease. The pathophysiology of this syndrome has not been fully elucidated, but an excess of catecholamines seems to have an essential role. A case of transient LV dysfunction that developed after self-injection of adrenaline is presented.

CASE PRESENTATION

A 27-year-old Caucasian man with a history of opioid and benzodiazepine drug abuse, and active hepatitis C was transferred from a regional hospital to the emergency room at Heidelberg University

Un dysfonctionnement ventriculaire gauche réversible évocateur du syndrome de Takotsubo après une auto-injection d'adrénaline

Un syndrome de ballonnement ventriculaire gauche (VG) transitoire, ou syndrome de Takotsubo, se caractérise par un dysfonctionnement VG réversible. Sa physiopathologie n'a pas été tout à fait dégagée, mais un excès de catécholamines semble y jouer un rôle essentiel. On décrit le cas d'un homme de 27 ans qui a développé un dysfonctionnement VG transitoire évocateur du syndrome de Takotsubo après une auto-injection d'adrénaline. Le présent cas peut fournir des données supplémentaires pour vérifier l'hypothèse d'une activation sympathique excessive en cas de syndrome de ballonnement VG.

Hospital (Heidelberg, Germany) after a self-administered intravenous injection of 2 mg adrenaline. The patient had no history of cardiovascular disease and showed signs of acute LV heart failure with pulmonary congestion and hypotension (70/40 mmHg) as well as chest pain. On admission, troponin T (0.96 µg/L) and N-terminal probrain natriuretic peptide (3151 ng/L) were elevated. The electrocardiogram showed a preterminal negative T wave in aVL. Echocardiography revealed markedly impaired LV function. Cardiac catheterization ruled out coronary artery disease, but confirmed a severely reduced LV function (ejection fraction less than 25%). Ventriculography (Figure 1 and Video) revealed midventricular akinesia but preserved basal and

Figure 1 Left ventricular angiography in diastole (A) and systole (B) revealing markedly impaired contractile function of the midventricular myocardium with preserved basal and apical contractility, reminiscent of 'reverse' Takotsubo syndrome

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apical contractility, highly reminiscent of a midventricular or 'reverse' Takotsubo syndrome (1). Insertion of an intra-aortic balloon pump resulted in blood pressure stabilization, while administration of vasopressors was avoided. The intra-aortic balloon pump was weaned 24 h later and, during the following days, the patient was started on heart failure medication with a beta-blocker and an angiotensin-converting enzyme inhibitor. Because the C-reactive protein and white blood cell counts were elevated, cardiac magnetic resonance imaging was also performed to rule out acute myocarditis. However, gadolinium-enhanced magnetic resonance imaging did not reveal myocardial edema or late enhancement. In echocardiographic follow-up studies, LV function improved significantly and returned to almost normal one week after the initial event. Likewise, Troponin T and N-terminal probrain natriuretic peptide levels significantly decreased (0.07 µg/L and 432 ng/L, respectively). The patient fully recovered from clinical signs of LV heart failure and was transferred back to the referring hospital.

DISCUSSION

The transient LV apical ballooning cardiomyopathy, or Takotsubo syndrome, is characterized by reversible LV dysfunction, either with apical ('typical') or midventricular ('atypical') akinesia (1,2). Clinically,

Takotsubo syndrome mimics the symptoms of an acute myocardial infarction. The precise pathophysiology is currently still unclear. However, growing evidence points toward a central role of sympathetic activation in this syndrome (3). The present report describes a case of reversible impaired LV function due to self-injection of adrenaline, clinically indistinguishable from Takotsubo syndrome. The present case may thus provide evidence for the principle that an excess of catecholamines is critical in the pathogenesis of Takotsubo syndrome.

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