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CASE REPORT

Tako Tsubo cardiomyopathy, presenting with cardiogenic shock in a 24-year-old patient with anorexia nervosa

M.N.M. Volman[1,2*], R.W. ten Kate[2], R. Tukkie[1]

Departments of [1]Cardiology, [2]Internal Medicine, Kennemer Gasthuis, Haarlem, the Netherlands,
*corresponding author: tel.: +31 (0) tel: 023-545 35 45, fax: +31 (0)23-545 36 29, e-mail: volman@kg.nl

ABSTRACT

Tako Tsubo cardiomyopathy is a serious condition that is caused by heart failure due to inordinate stress. We here present a case of a young woman with this disorder in association with anorexia nervosa. We postulate a pathophysiological relationship and discuss the management of Tako Tsubo cardiomyopathy.

Keywords: Tako Tsubo cardiomyopathy, apical ballooning syndrome, stress-induced cardiomyopathy, anorexia nervosa, hypoglycaemia, takotsubo

INTRODUCTION

We describe a rare case of Tako Tsubo cardiomyopathy (TTC) in a young female with anorexia nervosa presenting with cardiogenic shock. This case illustrates that TTC can be a serious complication in young females with anorexia nervosa. TTC is characterised by acute left ventricular contractile dysfunction, following intense emotional or physical stress. Tako Tsubo usually affects postmenopausal women. Presentation often resembles acute myocardial infarction with chest pain, ST elevation and a rise in cardiac enzymes.[1-3] However, no coronary event is found. The syndrome accounts for about 1 to 2% of the cases presenting with suspected acute coronary syndrome.[1,4] The left ventricle (LV) typically shows apical and mid-ventricular akinesis or dyskinesis with hypercontractile basal segments, resulting in an apical ballooning pattern.[1,5-7] The syndrome, first described in Japan in 1991, was named Tako Tsubo after a round-bottomed narrow-necked Japanese pot used for octopus fishing.[8] TTC is also known as stress-induced cardiomyopathy, broken heart syndrome or transient left ventricular apical ballooning syndrome. Recently, the Mayo Clinic proposed criteria for TTC *(table 1)*.[1,5] Therapy for TTC is supportive, since TTC is a self-limiting disease. Prognosis is favourable with full recovery of LV function in almost all cases. However, acute symptoms can be severe, including cardiogenic shock, ventricular arrhythmias and death.[5,6,9]

CASE REPORT

A 24-year-old woman was admitted to the department of psychiatry for treatment of severe emaciation. Her history revealed anorexia nervosa since the age of 18, resulting in multiple admissions. On admission her weight was 25 kg. During her admission to the psychiatric ward she lost consciousness due to severe hypoglycaemia, and she was transferred to the medium care unit for treatment with glucose and thiamine. Electrolyte disturbances,

Table 1. *Proposed Mayo Clinic criteria for apical ballooning syndrome or Tako Tsubo cardiomyopathy*

1	Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement The regional wall motion abnormalities extend beyond a single epicardial vascular distribution A stressful trigger is often, but not always present
2	Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture
3	New electrocardiographic abnormalities (either ST-segmental elevation and/or T-wave inversion) or modest elevation in cardiac troponin
4	Absence of: pheochromocytoma myocarditis

namely hypokaliaemia and hypocalcaemia, were corrected, without signs of refeeding. The electrocardiogram (ECG) showed new slightly negative T waves in V3-4, thought to be caused by electrolyte disturbances. The patient recovered and was returned to psychiatry. Next morning she suffered severe hypotension: 75/45 mmHg (115/75 on admission), heart rate was 96 beats/min. Her glucose was 2.1 mmol/l, so glucose was administered. There were no complaints of chest pain or dyspnoea. The ECG showed new ST-segment elevation in the inferior and anterior leads *(figure 1)*. She was immediately transferred to the coronary care unit. On arrival, the glucose was 6.0. Echocardiography revealed a contractile pattern typical for Tako Tsubo: apical ballooning with apical akinesia and basal hyperkinesia *(figure 2)*. Blood tests showed elevated cardiac enzymes: creatinine kinase (CK) 1428 U/l, CK-MB 98 µg/l, troponin T 0.10 µg/l. The patient was urgently transferred to a nearby tertiary centre for immediate cardiac catheterisation. The coronary angiogram showed normal coronary arteries. To treat the persisting cardiogenic shock an intra-aortic balloon pump (IABP) was inserted and treatment with inotropes, fluid resuscitation and low-molecular-weight heparin was initiated. Electrolyte disturbances were corrected. The echocardiogram showed a pattern, consistent with features of TTC.

Figure 1.

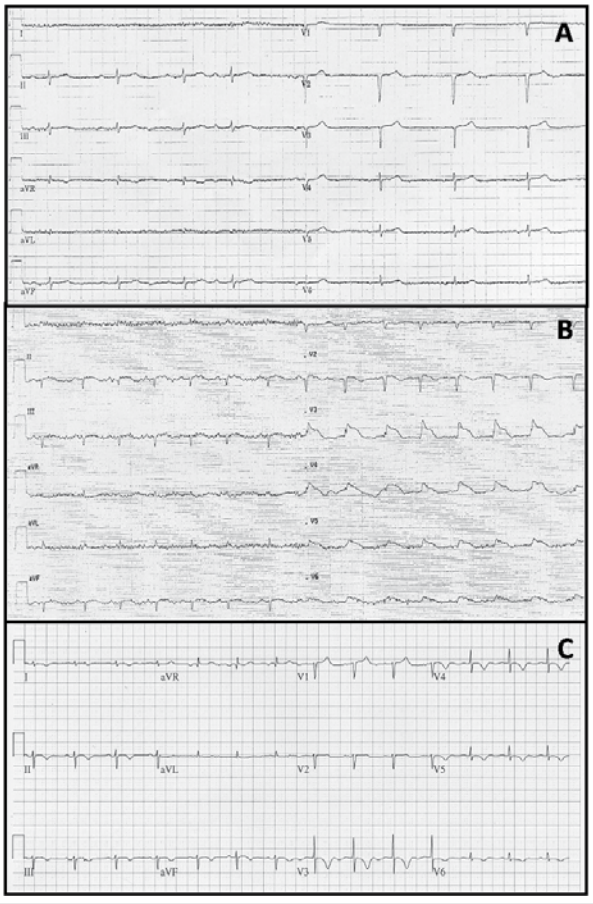
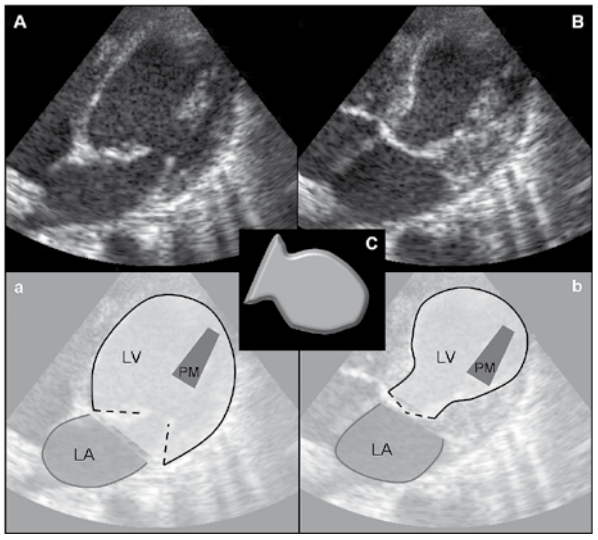


Figure 2.



Cardiac enzymes increased, topping at CK 5382 U/l and CK-MB 255 µg/l. Over the following days the ST elevation decreased. QT-interval prolongation and deep negative T waves emerged *(figure 1)*. After two days she was haemodynamically stable. IABP and inotropic support were withdrawn. Left ventricular function recovered substantially to an ejection fraction of 45 to 50% with mild wall motion abnormalities apicoanteroseptal over three months on ACE inhibitors.

DISCUSSION

In this report we present a case of a young female who suffered severe cardiogenic shock caused by Tako Tsubo cardiomyopathy, requiring intra-aortic balloon pump treatment. The rationale for reporting this case is the fact that it gives an opening to novel insights: it provides data that might increase our current understanding of TTC. The precise pathophysiology of TTC is unknown. The most favoured explanation is catecholamine-mediated myocardial stunning, as TTC is typically preceded by an emotional or physical stressor.[2,4,10,11] Commonly reported emotional stressors include death of a relative, severe argument or financial loss.[2,6,7,9] Among reported physical stressors are noncardiac surgery, asthma exacerbation, thyrotoxicosis and severe illness.[9,12] In our patient, the acute stressor appears to be hypoglycaemia, although refeeding and the mental stress of admission could also be stressors. It is known that hypoglycaemic stress increases plasma catecholamine

levels.[13] Therefore, hypoglycaemic stress may induce TTC. This case is unusual as TTC predominantly affects postmenopausal women (82 to 100% of cases).[5,6] Reduced oestrogen levels in postmenopausal women may alter endothelial function and microcirculatory vasomotor reactivity in response to catecholamine-mediated stimuli, possibly causing greater vulnerability to sympathetic-mediated myocardial stunning.[1,4,5,10] It may therefore be possible that the hormonal changes seen in young, severe anorectic patients sensitise them to TTC. To our knowledge only one report has been published on TTC in young females suffering from anorexia nervosa, describing three cases of possible TTC following hypoglycaemia.[14] All three cases showed ECG changes (either negative T waves or ST depression) and a rise in CK or CK-MB. Urgent echocardiography showed a Tako Tsubo pattern in only one case. In the other cases imaging was conducted only after five to seven days, showing no abnormalities. Cardiac catheterisation to exclude coronary occlusion was not performed in any of the cases. Therefore TTC was not proven, as in our case. As in the three cases mentioned above,[14] our case also suggests a relationship between TTC and anorexia-induced hypoglycaemia in young females. Further research is needed to reveal the pathophysiological mechanism of TTC and the relation with anorexia nervosa, oestrogen deficiency and hypoglycaemia. Treatment of TTC is mainly supportive. Besides ruling out acute myocardial infarction, management includes resolution of stress, monitoring and hydration.[1,4,5] Anticoagulation can be used to prevent left ventricular thrombosis. Beta-blockers are used empirically. Heart failure can be treated with diuretics and ACE inhibitors.[1,4,5] Cardiogenic shock due to pump failure is treated with inotropes and IABP. In case of cardiogenic shock left ventricular outflow tract obstruction must be excluded, because with this serious complication inotropes are contraindicated.[1,4,5] We believe that physicians treating anorectic patients need to be aware of TTC. We recommend routinely performing an ECG and echocardiogram to detect TTC in anorectic patients with chest pain, dyspnoea or hypotension. Concluding, Tako Tsubo cardiomyopathy can occur in young females with anorexia nervosa and hypoglycaemia might be a trigger. TTC has a favourable prognosis and LV function usually recovers fully. Despite its relatively benign nature in most cases, TCC can be characterised by severe clinical symptoms in the acute phase.

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