# Clinical Case Reports and Reviews



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# Atypical recovery from stress induced cardiomyopathy in a patient with anorexia nervosa

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#### **Abstract**

Anorexia nervosa is a psychiatric disease which is very often complicated by different cardiological abnormalities. There are many case reports, which described stress induced cardiomyopiathy, as a severe cardiological complication. Our unique case shows that during recovery from stress-induced cardiomyopathy, transient apical wall thickening (TAWT) can develop, mimicking apical hypertrophic cardiomyopathy.

## Case report

A 39-year-old female was admitted to our hospital because of anorexia nervosa with a body mass index of 10.5 and severe obsessivecompulsive disorder. Due to a spontaneous severe hypoglycaemia of 22 mg/dl and electrolyte disorders (sodium 128 mmol/l, potassium 2.8 mmol/l, chloride 89 mmol/l) she was transferred to the medical intensive care unit. She was disorientated and presented with a slurred speech which recovered after intravenous glucose administration. Her blood tests showed leukopenia (2.9 G/l), thrombocytopenia (91 G/l) with elevated transaminases (AST 1487 U/l, ALT 1425 U/l, GGT 177 U/l), lactate dehydrogenase (732 U/l) and creatine kinase (1813 U/l), while troponin levels were within normal range. Chest X-ray was normal. An electrocardiogram (ECG) showed sinus bradycardia and T-waves inversion in lead II, III, aVF, and V4-V6. Echocardiography revealed wall motion abnormalities of the apical and midventricular segments, typical for Tako-Tsubo cardiomyopathy and a pre-existing pericardial effusion above the right ventricle. These wall motion abnormalities regressed completely over two weeks. However, enddiastolic apical wall thickness increased to 14 mm. Therefore, she was scheduled for cardiac magnetic resonance imaging which showed thickening of the apex of the left ventricle (apical interventricular septum 19 mm) mimicking an apical hypertrophic cardiomyopathy. There was no evidence of myocarditis or myocardial oedema.

Due to enteral feeding via a nasogastric tube and psychological treatment, the patient recovered clinically within 3 months. Laboratory parameters normalised and the patient gained weight to a body mass index of 13. Echocardiography at follow up after 3 months showed a complete regression of the apical myocardial thickening and the pericardial effusion.

Anorexia nervosa is a psychiatric disease which is very often complicated by different cardiological abnormalities such as bradycardia, hypotension, mitral valve prolapse, or arrhythmias due to hypokalaemia or hypomagnesemia [1]. Enormous psychical stress and hypoglycaemia sometimes lead to catecholamine-mediated reactions

and development of stress-induced cardiomyopathy [2]. Our unique case shows that during recovery from stress-induced cardiomyopathy, transient apical wall thickening (TAWT) can develop, mimicking apical hypertrophic cardiomyopathy. TAWT which mimics apical hypertrophic cardiomyopathy during recovery from stress-induced cardiomyopathy has been reported, but not in patients with anorexia nervosa [3]. TAWT is one of the less-known phenomena of stressinduced cardiomyopathy. There are two mechanisms that could explain development of TAWT during the course of stress-induced cardiomyopathy. Firstly, TAWT can be a result of inflammation or interstitial oedema, for example in acute myocarditis. In our patient, we had no evidence of inflammation or myocardial oedema. A second theoretical mechanism could be effective via catecholamine stimulation. This may be related to the negative inotropic effect of epinephrine-ß2 adrenergic receptor- G, and the positive inotropic effect of norepinephrine-ß1 adrenergic receptor-G<sub>s</sub>, which lead to stress-induced cardiomyopathy [4]. Continuous B-adrenergic stimulation is known to cause cardiac hypertrophy, and such stimuli may also cause TAWT. One study showed that development of TWAT during the course of stress-induced cardiomyopathy is associated with more cardiac complications and higher hospital mortality [3]. Our case shows that the patients with anorexia nervosa are critically ill patients, and a cardiological examination has to be obligatory.

### References

- Thurston J, Marks P (1974) Electrocardiographic abnormalities in patients with anorexia nervosa. Br Heart J 36: 719-723.[Crossref]
- Ono T, Kasaoka S, Fujita M, Yamashita S, Kumagai K, et al. (2009) Complete recovery from severe myocardial dysfunction in a patient with anorexia nervosa. J Cardiol 54: 480-484. [Crossref]

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- 3. Shin DG, Cho IJ, Shim CY, Ryu SK, Chang HJ, et al. (2015) Transient apical wall thickening in patients with stress cardiomyopathy: Prevalence, profile and impact on clinical course. *Int J Cardiol* 194: 87–92. [Crossref]
- Osadchii OE (2007) Cardiac hypertrophy induced by sustained beta-adrenoreceptor activation: pathophysiological aspects. Heart Fail Rev 12: 66-86.[Crossref]

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