CASE REPORT

Stress induced (Takotsubo) cardiomyopathy triggered by the COVID-19 pandemic

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Abstract. Stress induced (Takotsubo) cardiomyopathy (TC) represents an acute heart failure syndrome triggered by physical or emotional stressors. COVID-19 pandemic has caused an unprecedented health crisis resulting in fear, distress and anxiety, with emerging cardiovascular implications. COVID-19 related stress can act as potential trigger for TC. We present a case of an elderly female who developed TC due to stress surrounding COVID-19.

Case presentation

A 79-year old female presented with acute chest pain after watching the daily TV report on the death toll of COVID-19 pandemic. Her medical history included arterial hypertension under treatment with ace-inhibitors. She was haemodynamically stable, with a blood pressure of 130/70 mmHg, a heart rate of 75 beats/min in sinus rhythm, and an oxygen saturation of 99% in room air. Physical examination and body temperature were normal. Electrocardiogram recorded diffuse ST segment elevation (Fig. 1A). Emergency coronary angiography excluded stenotic lesions (Fig. 1B and C). Left ventriculography illustrated severe hypokinesia in the mid-apical segments, hyperdynamic basal segments

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(Fig. 1D, systole; and E, diastole), and impaired left ventricular systolic function with an ejection fraction of 35%. High sensitive troponine-T was elevated reaching a peak after 12 h (2.950 pg/ml, normal <15.6 pg/ml), whereas other routine blood tests, including inflammatory markers, were normal.

The diagnosis

Stress induced (Takotsubo) cardiomyopathy.

Discussion

Takotsubo cardiomyopathy (TC), also called stress-induced cardiomyopathy, mimics an acute coronary syndrome (ACS), predominantly affecting elderly women and is triggered by an emotional or physical stressor (1), in our case the COVID-19 death update. The term 'Takotsubo' means 'octopus pot' in Japanese and describes the pathognomonic left ventricular end-systolic apical ballooning, illustrated in transthoracic echocardiogram (TTE) and left ventriculography during cononary angiography (1).

Clinical presentation, electrocardiographic findings, and biomarker profiles are similar to those of an ACS (2). TC represents an acute heart failure syndrome with substantial morbidity and mortality (2). The in-hospital and one-year mortality of TC has been reported 1-5 and 5.6%, respectively, with a recurrence rate of ≤5% (1,2). Left ventricular systolic function usually recovers within four weeks (1). TC is characterized by a low-grade chronic inflammatory state consisting of myocardial macrophage inflammatory infiltrates, substantial increase in the pro-inflammatory, classical monocyte subset CD14++CD16-, and an increase in the systemic pro-inflammatory cytokines interleukin-6 (IL-6), IL-8 and chemocine CXCL1 (3). This localized and systemic inflammatory response plays a pivotal role in the

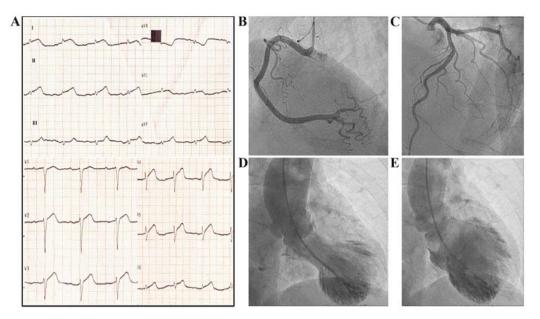


Figure 1. Electrocardiogram showing diffuse ST segment elevation (A). Emergency coronary angiography excluded stenotic lesions (B and C). Left ventriculography illustrating severe hypokinesia in the mid-apical segments and hyperdynamic basal segments (D, systole; E, diastole).

development of TC, while it might also serve as a promising therapeutic target for which no effective treatment currently exists (3).

The brain-heart interaction has been acknowledged as a significant factor in the pathogenesis of TC. Overstimulation of the sympathetic nervous system is hypothesized as the underlying mechanism (2,4). The hypothalamic-pituitary-adrenal axis (HPA-axis) is a major neuroendocrine system which regulates the release of cortisol from the adrenal gland, shifting the metabolism to higher stress levels. Higher serum cortisol levels and increased sympathetic activity may cause myocardial damage (5). Consequently, acute psychological or physical stress mediated via the sympathetic-adrenalmedulla axis with catecholamine release in the adrenal medulla and the HPA axis with consecutive cortisol release from the adrenal cortex may act as a trigger for TC (5). Additionally, regional differences in myocardial expression of \beta2-adrenergic receptor density have been shown, which mediate the cellular effects of the increased catecholamine blood concentrations and explain the regional left ventricular myocardial stunning (5).

Therefore, the enormous psychological strain posed on the community by the pandemic can activate the brain-heart axis and serve as a potential stressor for TC (4,6). COVID-19 pandemic has caused an unprecedented health crisis resulting in individual and societal fear and anxiety, with emerging cardiovascular implications, which we should be aware of (4).

The patient was transferred to the coronary unit where treatment with ace-inhibitor, aspirin, b-blocker and statin was initiated. Her course was uncomplicated with gradual normalization of troponin. She was discharged after one week in good condition and normal echocardiography, and was advised to avoid following news related to the pandemic.

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Authors' contributions

SG and AST wrote the original draft, edited and critically revised the manuscript. KT, DT, DV, ER, PT, LEP, MSK, DAS and AJM critically revised and edited the manuscript. All authors substantially contributed to the conception, writing and revision of the work and approved the final content of the manuscript.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

DAS is the Editor-in-Chief for the journal, but had no personal involvement in the reviewing process, or any influence in terms of adjudicating on the final decision, for this article. The other authors declare that they have no competing interests.

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