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From assault to stroke: Takotsubo cardiomyopathy complicated by embolic infarctions: a case report

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Abstract

Takotsubo cardiomyopathy is a transient cardiac condition often triggered by sudden, severe emotional or physical stress. Although initially regarded as benign, thromboembolic complications occur in about 12% of patients, with cerebral infarction reported in up to 9.5% of cases. We describe a 57-year-old woman with hypertension who developed takotsubo cardiomyopathy following physical assault with chest trauma. She presented with chest pain and altered consciousness and was initially treated for acute coronary syndrome. Her coronary angiography was normal, and takotsubo cardiomyopathy was diagnosed based on characteristic echocardiographic features. The patient then developed an acute embolic stroke affecting both sides of the middle cerebral artery territory, requiring anticoagulation therapy. This case underscores the potentially serious thromboembolic complications of TC and highlights the importance of early recognition, appropriate consideration of anticoagulation, and multidisciplinary management. The clinical challenge of balancing bleeding and thrombotic risks in acute TC requires personalised decision-making and close monitoring.

Key words: Takotsubo cardiomyopathy, stress cardiomyopathy, embolic stroke, anticoagulation, thromboembolic complications

Introduction

Takotsubo cardiomyopathy (TC), also known as stress-induced cardiomyopathy or "broken heart syndrome," is characterised by acute transient left ventricular dysfunction with apical ballooning, which mimics acute myocardial infarction in the absence of significant coronary artery disease.[1,2] The incidence of TC has been increasing (15-30 cases per 100,000 per year), although the actual prevalence remains unknown due to possible underdiagnosis.[2]

The condition is usually triggered by emotional stressors such as bereavement or physical stressors like acute illness, surgery, or trauma.[2] Although previously considered a benign, self-limiting condition, recent studies have shown that patients with TC experience persistent, subtle, ongoing cardiac dysfunction, and many continue to have limited symptoms despite the restoration of left ventricular ejection fraction.[2,3]

Of particular concern are the thromboembolic complications that can occur in TC. The incidence of acute thromboembolic events related to TC is approximately 12.2%, which includes ventricular thrombi, cerebrovascular events, retinal and brachial artery pathologies, and renal, splenic, and aortic involvement.[4] The incidence of cerebral infarction due to TC ranges from 0 to 9.5%.[5]

The pathophysiology of thromboembolism in TC involves multiple mechanisms, including severe wall motion abnormalities that lead of blood stasis, especially in the apical region; activation of the coagulation cascade due to stress-induced inflammatory responses; and possible endothelial dysfunction.[4,5] Some advocate anticoagulation therapy for all patients with TC who have severe LV dysfunction, the apical subtype, or evidence of LV thrombus.[4,5] The recommended duration is three months or until wall motion abnormalities resolve.[4,6]

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However, no guidelines currently exist for the treatment of TC, leaving clinicians with a clinical dilemma regarding optimal anticoagulation strategies.[6] This case report illustrates the complexity of managing TC complicated by embolic stroke and adds to the growing literature on thromboembolic complications in this condition.[4,5]

Case presentation

A 57-year-old woman presented to a tertiary care hospital six hours after being physically assaulted with a direct blow to the chest. Her medical history was significant only for hypertension, managed with losartan 50 mg twice daily, with excellent adherence and regular follow-up care. She had no family history of cardiovascular disease, was a non-smoker, and was unemployed.

The patient complained of generalised chest pain without radiation, not associated with autonomic symptoms such as diaphoresis or nausea. She also reported a sudden onset of altered consciousness lasting about one hour, which was not associated with seizure activity or focal neurological deficits at the time of presentation.

On admission, she appeared distressed but was hemodynamically stable with a blood pressure of 100/80 mmHg and a regular pulse rate of 88 bpm. The Glasgow Coma Scale score was 12 (Eye: 4, Verbal: 2, Motor: 6). She had slurred, incomprehensible speech consistent with dysarthria. Cranial nerve examination was normal, and there were no other focal neurological deficits with bilateral pupils measuring 2 mm and equally reactive to light. Cardiovascular, respiratory and abdominal system examinations were unremarkable.

Laboratory investigations showed normal capillary blood glucose of 152 mg/dL, and cardiac troponin I was significantly elevated at 2374 ng/L (normal <14 ng/L). Complete blood count and basic metabolic panel were within normal limits. The lipid profile was also within the normal range.

The initial ECG showed deep symmetrical T wave inversions in anterolateral leads (V3-V6, I, aVL). Non-contrast CT brain on admission was normal, with no evidence of haemorrhage or acute infarction. Echocardiogram revealed a normal-sized left ventricle with severely reduced ejection fraction (25-30%), anterior and anteroseptal hypokinesia, and apical akinesia. The left atrium was dilated, but there was no visible thrombus. The right heart chambers were normal, and there was no pericardial effusion (Figure 1). However, coronary angiography revealed normal coronary arteries without significant stenosis.

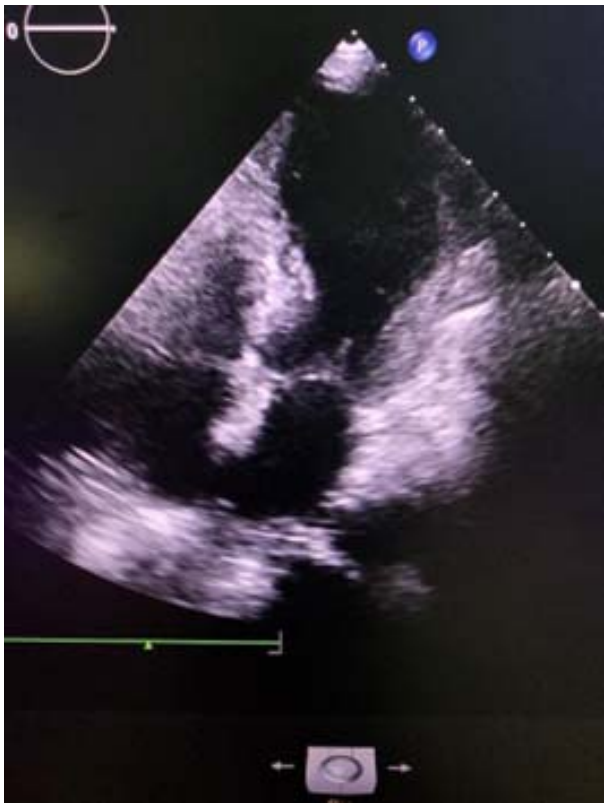

Figure 1. Echocardiogram showing a normal-sized left ventricle with anterior and anteroseptal hypokinesia and apical akinesia.

Based on the clinical presentation suggesting acute coronary syndrome, the patient received a 300 mg loading dose of aspirin, followed by 75 mg daily, a 300 mg loading dose of clopidogrel, then 75 mg daily, 40 mg of atorvastatin daily, and subcutaneous enoxaparin 60 mg twice daily.

In view of the patient's persistent slurring of speech, a repeat CT scan of the brain was performed on Day 3, which demonstrated evidence of multiple lacunar infarcts in the bilateral middle cerebral artery territories. Following the diagnosis of embolic stroke, dual antiplatelet therapy and enoxaparin were discontinued, and rivaroxaban 20 mg daily was commenced. Heart failure medications (bisoprolol, ramipril, and spironolactone) were also initiated.

Four days post-admission, a repeat echocardiogram demonstrated modest improvement in ejection fraction to 30-35% with persistent apical hypokinesia. No left atrial or ventricular thrombi were visualised.

At two weeks, significant improvement in left ventricular systolic function was noted on echocardiography. Bilateral carotid duplex ultrasound was

normal, and a 24-hour Holter monitoring revealed no significant arrhythmia.

In the long-term management, rivaroxaban was continued for secondary stroke prevention, and a cardiac rehabilitation programme was initiated. A six-month follow-up echocardiogram was planned, and a neurological follow-up for stroke recovery assessment was arranged.

Discussion

This case illustrates the complex pathophysiology behind thromboembolic complications in TC. The severe apical akinesia characteristic of TC creates conditions conducive to thrombus formation through several mechanisms, including blood stasis in the akinetic segments, activation of the coagulation cascade during the acute stress response, and potential endothelial dysfunction related to catecholamine excess.[4,5] The catecholamine surge that triggers TC may also promote hypercoagulability through direct effects on platelet function and coagulation factors.[1,4] Additionally, the inflammatory response linked to myocardial stunning may further increase the risk of thrombosis.[7]

Several case reports have documented the association between TC and cerebrovascular events, supporting the clinical significance of this complication. Jabiri et al (2010) first described TC as a cardioembolic cause of brain infarction,[5] establishing the connection between stress cardiomyopathy and stroke. Hassan et al. (2019) provided a detailed review of thrombo-embolic complications in TC with illustrative cases, emphasising the need for heightened awareness of these complications.[4] Multiple recent publications have documented similar cases, including a case report by Patel et al (2025), describing "a cycle of complications" involving trastuzumab-induced TC causing left ventricular thrombosis complicated by thromboembolic stroke.[8] Studies have shown that stroke associated with TC is often severe, with more than 30% of reported cases related to major neurological deficits, emphasising the clinical importance of early recognition and prevention.[4,5]

Managing anticoagulation in TC presents a major clinical challenge. Although no formal guidelines currently exist for treating TC, several factors must be considered. Arguments in favour of anticoagulation include the high risk of thromboembolism (12.2% overall incidence),[6] severe left ventricular dysfunction with apical akinesia, and the potential for rapid clinical deterioration if embolic events occur.[2,4] Arguments against anticoagulation include the risk of bleeding, especially in patients with recent trauma, the transient nature of the condition, and the lack of definitive, evidence-based guidelines.[6] Risk stratification may be the most appropriate approach. Current literature suggests considering anticoagulation for patients with severe LV dysfunction (EF<35%), apical ballooning pattern, visible LV thrombus on echocardiography and the presence of additional risk factors for thromboembolism.[4,6]

This case emphasises the importance of multidisciplinary care in TC management, with cardiology input essential for making the primary diagnosis, echocardiographic monitoring, and heart failure management; neurology input for stroke evaluation, anticoagulation guidance, and neurological follow-up; and internal medicine for initial stabilisation and differential diagnosis, along with clinical pharmacists' involvement in medication reconciliation and drug interaction monitoring.[4,6,9.]

This case adds to the growing evidence that TC is not the benign condition initially thought. The risk of serious complications highlights the importance of clinicians being more aware of thromboembolic risks, developing clinical scoring systems to identify high-risk patients, implementing systematic echocardiographic monitoring for thrombus formation, and adhering to evidence-based guidelines for anticoagulation therapy.[4,6,9] Limitations of this case include limited generalisability, lack of long-term follow-up data, and the absence of advanced imaging techniques such as cardiac MRI.

Conclusion

Takotsubo cardiomyopathy, although often considered a benign stress-related condition, can result in significant thromboembolic complications, including embolic stroke. This case demonstrates the importance of maintaining a high index of suspicion for such complications, especially in patients with severe left ventricular dysfunction and apical involvement.

The clinical management of TC complicated by thromboembolic events requires individualised decision-making, balancing the risks of bleeding and thrombosis while considering the patient's overall clinical context. A multidisciplinary approach involving cardiology, neurology, and other specialities is essential for achieving optimal outcomes.

Author declaration

Conflict of interests

The authors declare no conflict of interests.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Criteria for authorship

GAMC and SDS conceptualised the case report. GAMC and KRF conducted the literature review and drafted the initial manuscript, which SDS subsequently edited. GAMC revised the manuscript. SDS supervised and reviewed the revised manuscript. All authors read and approved the final version.

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