Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Sepsis-Triggered Takotsubo Syndrome in a Critically ill Patient: A Fatal Diagnostic Challenge

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DOI: https://doi.org/10.36347/sjmcr.2026.v14i07.031 | **Received:** 05.06.2026 | **Accepted:** 28.07.2026 | **Published:** 30.07.2026

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Abstract | **Case Report**

Severe infection may trigger Takotsubo syndrome (TTS), but recognizing it in the intensive care unit can be challenging because its presentation may resemble both sepsis-induced cardiomyopathy and acute coronary syndrome. We describe a 61-year-old man with diabetes, hyperthyroidism, and severe chronic neurological disability following surgery for craniopharyngioma, who was admitted with bronchopneumonia, hypoxemic respiratory failure, and septic shock. Initial transthoracic echocardiography showed a hyperdynamic left ventricle with preserved systolic function. Less than 24 hours later, repeat echocardiography revealed apical ballooning, akinesia of the apical and mid-ventricular segments, basal hyperkinesia, and a left ventricular ejection fraction of approximately 25%. These changes were associated with a marked rise in troponin and the new onset of a complete bundle-branch block. Despite broad-spectrum antibiotics, invasive mechanical ventilation, vasopressor support, and supportive care, the patient developed refractory shock and died 48 hours after ICU admission. Coronary angiography was considered but could not be performed because his profound hemodynamic instability made transfer and invasive assessment unsafe. Cardiac magnetic resonance imaging was also not feasible, while temporary mechanical circulatory support was unavailable. The abrupt onset of a typical regional pattern of left ventricular dysfunction after a major physical stressor strongly suggested sepsis-triggered TTS. However, acute coronary syndrome and myocarditis could not be definitively excluded, and recovery of ventricular function could not be documented. This case highlights the importance of serial bedside echocardiography and of clearly acknowledging diagnostic uncertainty when confirmatory investigations cannot be safely performed.

Keywords: Takotsubo syndrome, sepsis, sepsis-induced cardiomyopathy, intensive care unit, refractory shock, echocardiography.

INTRODUCTION

Takotsubo syndrome (TTS) is an acute and usually reversible form of left ventricular dysfunction characterized by regional wall-motion abnormalities that commonly extend beyond the territory of a single epicardial coronary artery. Initially described in Japan, the syndrome is now known to occur after emotional stress as well as major physical stressors such as severe infection and critical illness (Sato *et al.,* 1990; Ghadri *et al.,* 2018a; Cammann *et al.,* 2021).

Diagnosis becomes particularly challenging in the intensive care unit (ICU), where TTS may resemble sepsis-induced cardiomyopathy, acute coronary syndrome, or myocarditis. Electrocardiographic changes, cardiac biomarker elevation, and ventricular dysfunction may be present in all of these conditions. In a prospective ICU study based on systematic cardiac screening, TTS was found in 4.6% of patients, and sepsis was one of the leading triggers (Doyen *et al.,* 2020). By contrast, a nationwide administrative analysis reported TTS in only 0.15% of severe-sepsis admissions, a difference that likely reflects under-recognition in routine practice (Vallabhajosyula *et al.,* 2018). We describe a fatal case with a typical echocardiographic pattern in whom confirmatory coronary and cardiac magnetic resonance investigations could not be safely performed.

CASE REPORT

A 61-year-old man was brought to the emergency department with fever and severe respiratory distress. His medical history included type 2 diabetes mellitus treated with metformin, hyperthyroidism, and

craniopharyngioma surgery in 2020, after which he developed aphasia and tetraparesis.

At presentation, his temperature was 38 °C, respiratory rate 66 breaths/min, and oxygen saturation 85% despite oxygen delivery at 15 L/min. He had pronounced respiratory effort and bilateral crackles. The Glasgow Coma Scale score was 11/15, interpreted in the context of his pre-existing aphasia. Laboratory investigations showed a neutrophil-predominant white blood cell count of 10.2×10^9/L, hemoglobin of 140 g/L, platelets of 120×10^9/L, and serum creatinine of 124 µmol/L, compared with a baseline value of 71 µmol/L. Troponin was negative on admission.

The first electrocardiogram showed sinus tachycardia at 113 beats/min. Bedside transthoracic echocardiography revealed a hyperdynamic left ventricle with preserved systolic function. Chest computed tomography demonstrated bilateral multifocal pulmonary opacities consistent with bronchopneumonia, while brain computed tomography showed no acute lesion and only chronic postoperative changes (Figure 1).

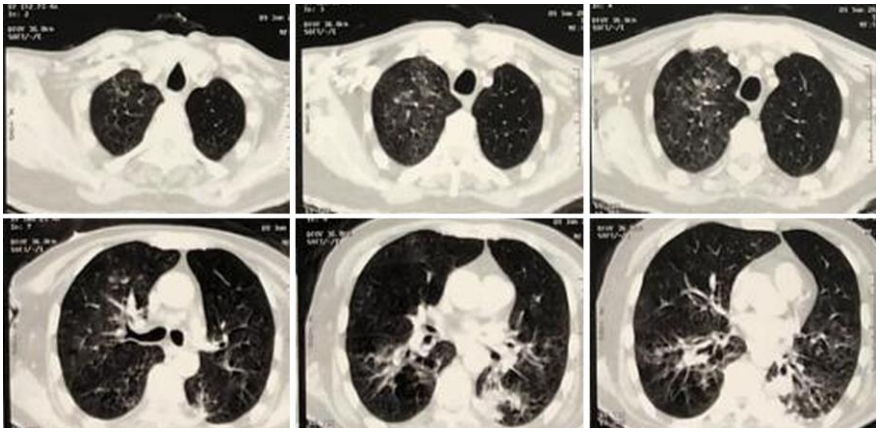
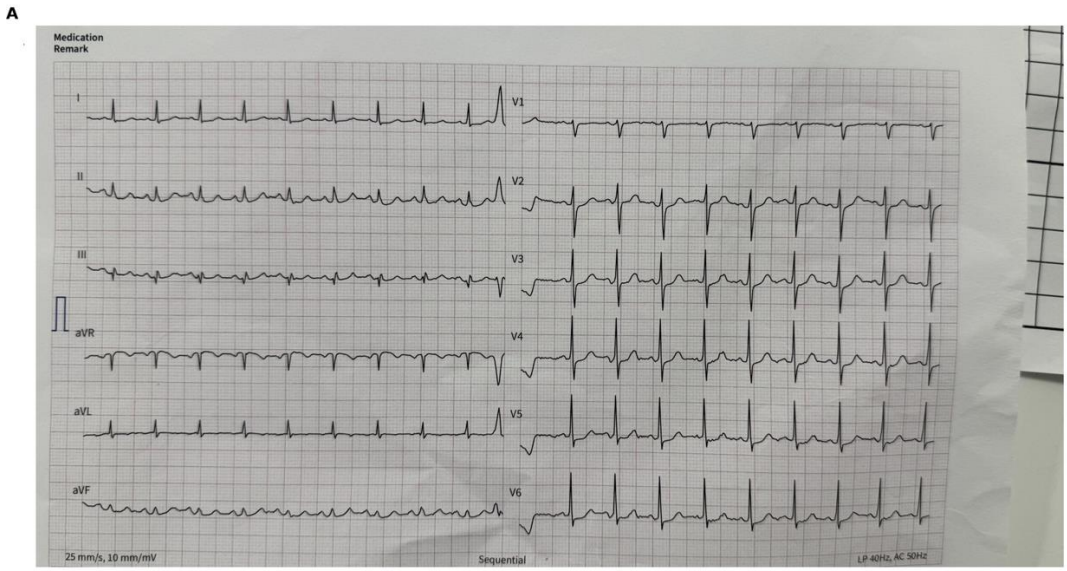


Figure 1: Representative axial chest computed tomography images demonstrating bilateral multifocal pulmonary opacities consistent with bronchopneumonia. The panels were cropped and arranged for presentation without alteration of the clinical image content

The patient was admitted to the ICU, intubated, and placed on invasive mechanical ventilation. Treatment included ceftriaxone and levofloxacin, corticosteroids, vasopressors, insulin infusion, and general supportive care. His hemodynamic condition nevertheless worsened, and septic shock developed.

Within 24 hours, repeat transthoracic echocardiography showed a striking change: apical ballooning with akinesia of the apical and mid-ventricular segments, preserved hypercontractility of the basal segments, and a left ventricular ejection fraction of approximately 25% (Figure 3). Troponin became markedly elevated. At the same time, a new bundle-branch block with secondary repolarization changes appeared on the electrocardiogram (Figure 2). The patient subsequently progressed to refractory shock associated with severe acute left ventricular systolic dysfunction.



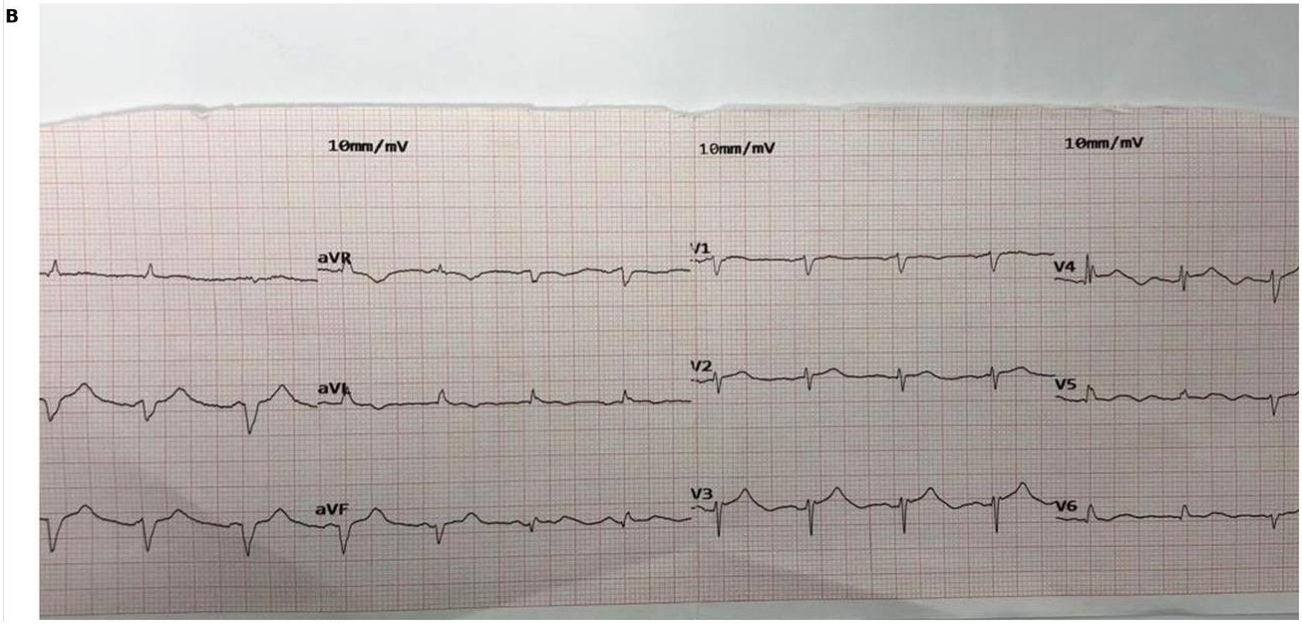


Figure 2: Electrocardiograms obtained during hospitalization. (A) Initial tracing showing sinus tachycardia at approximately 113 beats/min. (B) Follow-up tracing after clinical deterioration, demonstrating a new complete bundle-branch block with secondary repolarization abnormalities

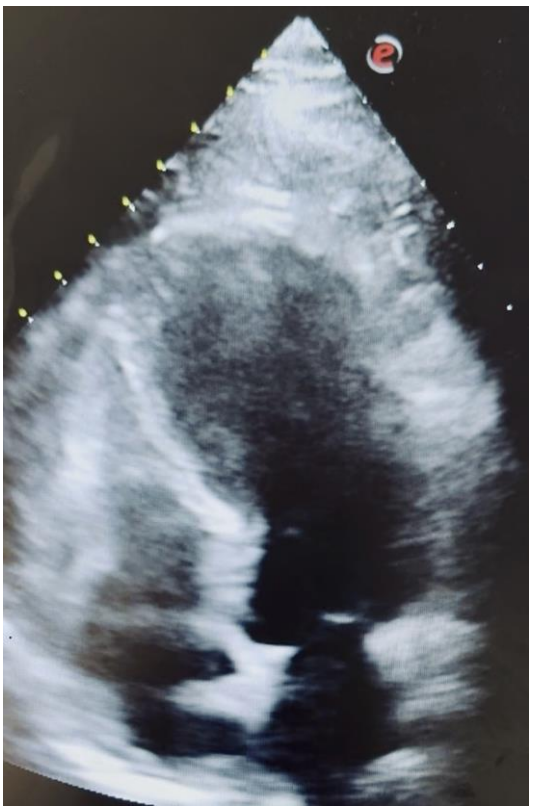


Figure 3: Representative apical four-chamber echocardiographic image recorded during hemodynamic deterioration. The dynamic bedside examination showed apical ballooning, mid-apical akinesia, basal hyperkinesia, and severe left ventricular systolic dysfunction, with an estimated LVEF of approximately 25%. Wall motion cannot be fully assessed from a single still image

Coronary angiography was considered but was not performed because the patient's profound hemodynamic instability made the procedure clinically unsafe. Cardiac magnetic resonance imaging was also not feasible, and temporary mechanical circulatory support was unavailable. He died 48 hours after ICU admission, before ventricular recovery could be assessed.

DISCUSSION

The most compelling feature in this case was the rapid change from a hyperdynamic ventricle to severe,

regionally distributed systolic dysfunction with apical ballooning. This pattern is more characteristic of TTS than of the usual diffuse or biventricular dysfunction described in sepsis-induced cardiomyopathy. The two conditions also differ in their proposed mechanisms. Sepsis-induced cardiomyopathy is mainly linked to systemic inflammation, endothelial and microcirculatory injury, mitochondrial dysfunction, and disturbed calcium handling. TTS, in contrast, is more strongly associated with sympathetic overactivity, catecholamine-mediated myocardial stunning, and coronary microvascular dysfunction (Hollenberg & Singer, 2021; Ghadri *et al.,* 2018a). In critically ill patients who cannot undergo angiography, the term clinical TTS has been proposed when the imaging pattern is strongly suggestive (Muratsu *et al.,* 2019).

Even with this characteristic pattern, the diagnosis cannot be considered definitive. The International Takotsubo expert consensus recommends urgent coronary evaluation in patients with a possible acute coronary syndrome and emphasizes echocardiographic assessment for left ventricular outflow tract obstruction in unstable patients (Ghadri *et al.,* 2018b). Similarly, the 2023 European Society of Cardiology guideline supports immediate invasive assessment when acute coronary syndrome is suspected in the presence of hemodynamic instability (Byrne *et al.,* 2023). In our patient, coronary angiography was considered but could not be performed because profound hemodynamic instability made transfer and invasive assessment clinically unsafe; therefore, an acute coronary occlusion remained a possible alternative diagnosis. Cardiac magnetic resonance imaging, which could have helped distinguish TTS from myocardial infarction or myocarditis, was also not feasible. For these reasons, the diagnosis is best described as probable sepsis-triggered TTS.

Management in this setting must address the infection and the cardiovascular phenotype at the same time. Expert recommendations stress the importance of looking for left ventricular outflow tract obstruction before increasing inotropic support, because treatment may differ substantially when obstruction is present. Catecholamine exposure should be limited whenever the clinical situation allows, although vasopressors are often unavoidable in septic shock. In refractory cases, non-catecholaminergic inotropes or temporary mechanical circulatory support may be considered in experienced centers. The evidence remains largely observational, and the choice of device depends on ventricular function and on whether outflow obstruction is present (Ghadri *et al.,* 2018b; Nabzdyk *et al.,* 2019).

The poor outcome was probably multifactorial. Severe pulmonary infection, septic shock, profound acute myocardial dysfunction, and the absence of advanced circulatory support all contributed to the patient's deterioration. In TTS, physical triggers, male sex, and markedly reduced left ventricular ejection fraction have been associated with a higher risk of acute complications (Ghadri *et al.,* 2018b). Current evidence also shows that no diagnostic pathway has yet been specifically validated for ICU patients. In practice, repeated assessment combining electrocardiography, cardiac biomarkers, and echocardiography remains essential when unexplained cardiac deterioration occurs (Visvanathan *et al.,* 2025).

Several limitations should be acknowledged. Coronary angiography and cardiac magnetic resonance imaging could not be performed because of the patient's hemodynamic instability. Invasive hemodynamic monitoring, strain imaging, and follow-up echocardiography were not obtained, and temporary mechanical circulatory support was unavailable. In addition, Figure 3 is a representative still image, whereas the wall-motion pattern was identified during the dynamic bedside examination. These limitations prevent definitive exclusion of acute coronary syndrome, myocarditis, or an atypical presentation of sepsis-induced cardiomyopathy.

CONCLUSION

Sepsis-triggered TTS should be suspected when a critically ill septic patient develops a sudden regional pattern of left ventricular dysfunction, particularly apical ballooning. Serial bedside echocardiography can provide an early diagnostic clue, but the level of certainty must be stated honestly when coronary imaging and cardiac magnetic resonance imaging cannot be safely performed and ventricular recovery cannot be documented. Early recognition remains clinically important because evaluation for outflow tract obstruction and timely access to advanced shock support may alter management.

Acknowledgements

The authors sincerely thank the intensive care and cardiology teams at Mohamed V Military Teaching Hospital for their dedicated patient care and multidisciplinary collaboration throughout the diagnostic evaluation and management of this case.

Funding: The authors received no specific funding for this work.

Competing Interests: The authors declare that they have no competing interests.

Use of Generative Artificial Intelligence

OpenAI ChatGPT (GPT-5.6 Thinking; OpenAI; accessed July 2026) was used to assist with English-language editing, reference verification, and journal-specific formatting. All clinical information, interpretations, citations, and references were reviewed and validated by the authors, who accept full responsibility for the final manuscript. Generative

artificial intelligence was not used to generate or modify the clinical content of the figures; the images were limited to cropping and panel arrangement for presentation.

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