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# Acute Left Ventricular Dysfunction During Septic Shock Suggestive of Takotsubo Syndrome: A Case Report

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**Abstract** **Case Report**

Takotsubo syndrome is an acute, transient stress-induced cardiomyopathy that can mimic acute coronary syndrome and has been increasingly described in settings of severe physical stress such as sepsis. Its diagnosis relies on a combination of clinical, biological, and echocardiographic findings, as well as the rapid reversibility of left ventricular dysfunction. We report the case of a patient admitted to the intensive care unit for acute respiratory failure secondary to severe pneumonia complicated by septic shock. The clinical course was marked by the subsequent development of severe left ventricular dysfunction, not confined to a single coronary territory, associated with suggestive echocardiographic findings and non-specific electrocardiographic abnormalities. In this context of major physical stress, these findings raised suspicion of Takotsubo syndrome. The subsequent clinical evolution was favorable, with rapid and near-complete recovery of left ventricular function within the first week of hospitalization, further supporting the diagnostic suspicion.

**Keywords:** Takotsubo syndrome; Left ventricular dysfunction; Septic shock.

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## INTRODUCTION

Takotsubo syndrome, also known as stress-induced cardiomyopathy, is an acute and transient cardiac condition characterized by reversible left ventricular systolic dysfunction, most often occurring in the setting of intense emotional or physical stress. Initially described in Japan in 1990, the syndrome derives its name from the characteristic systolic appearance of the left ventricle, resembling a Japanese octopus's trap called a "takotsubo" [1].

Clinically, Takotsubo syndrome frequently mimics acute coronary syndrome, presenting with chest pain, electrocardiographic abnormalities, and moderate elevation of cardiac biomarkers, making diagnosis challenging in the acute phase [2]. It predominantly affects elderly women, particularly after menopause, and accounts for approximately 1–2% of patients admitted with suspected acute coronary syndrome [3].

Forms triggered by physical stress, particularly in the context of sepsis, acute respiratory failure, or multiorgan dysfunction, are increasingly reported in intensive care units and appear to be associated with higher morbidity and mortality compared with classical emotionally triggered forms [4]. Transthoracic echocardiography plays a central role in the initial diagnostic evaluation by demonstrating regional wall motion abnormalities not confined to a single coronary territory, while rapid recovery of cardiac function represents a key diagnostic feature [5].

We report here the case of a patient admitted for acute respiratory failure complicated by septic shock, whose clinical course was marked by acute reversible left ventricular dysfunction highly suggestive of Takotsubo syndrome, highlighting diagnostic challenges and the value of echocardiography in the intensive care setting.

## CASE PRESENTATION

A 75-year-old woman was admitted to the intensive care unit for management of acute respiratory failure secondary to pneumonia (Figure 1). On admission, clinical examination revealed marked tachypnea and severe hypoxemia requiring high-flow oxygen therapy, followed by invasive mechanical ventilation due to worsening respiratory failure and altered consciousness. Broad-spectrum empirical antibiotic therapy was initiated immediately as part of the management of septic shock of pulmonary origin and was subsequently adjusted according to microbiological results and clinical evolution.

The initial course was unfavorable, with progression to septic shock originating from the pulmonary infection. The patient underwent cautious fluid resuscitation guided by hemodynamic monitoring, allowing optimization of preload while avoiding fluid overload, before initiation of norepinephrine. Given the evidence of significant myocardial contractile impairment, dobutamine was subsequently introduced for inotropic support. This clinical picture occurred in a context of major physical stress, combining severe infection, hypoxia, and early multiorgan dysfunction.

Electrocardiography revealed sinus tachycardia without ST-segment elevation, associated with QT interval prolongation (Figure 2). Cardiac biomarkers showed a moderate elevation of troponin. Serial measurements demonstrated an initial modest increase followed by rapid decline, without prolonged elevation or a secondary peak suggestive of extensive acute coronary syndrome.

Transthoracic echocardiography (TTE), performed during the acute phase of shock, demonstrated severe left ventricular systolic dysfunction characterized by akinesia of the apical and mid-ventricular segments with compensatory hyperkinesia of the basal segments. Left ventricular ejection fraction was estimated at approximately 25–30%. The right ventricle was of normal size with preserved systolic function, and no significant valvular abnormalities were identified (Figure 3).

In the absence of electrocardiographic evidence of acute coronary syndrome, given the non-territorial distribution of echocardiographic abnormalities, the context of intense physical stress, and the moderate elevation of cardiac biomarkers, a diagnosis of stress-induced cardiomyopathy consistent with Takotsubo syndrome was suspected. Coronary angiography was not performed in the acute phase due to the patient's hemodynamic instability.

Clinical evolution under etiological and supportive treatment, including appropriate antibiotic therapy for septic shock, ventilatory support, and hemodynamic management, was progressively favorable. Hemodynamic improvement allowed gradual reduction and discontinuation of vasoactive agents. In parallel, respiratory status improved under antibiotic therapy and ventilatory support, enabling progressive and complete weaning from invasive mechanical ventilation.

Serial follow-up echocardiograms performed during the first week of hospitalization demonstrated rapid and progressive recovery of left ventricular function, with marked improvement in regional wall motion. On day 7, left ventricular ejection fraction was estimated at 45–50%, with near-complete resolution of apical akinesia (Figure 4).

Overall, the association of acute and reversible left ventricular dysfunction occurring in a context of severe physical stress, absence of electrocardiographic features of acute coronary syndrome, characteristic echocardiographic findings, troponin kinetics, and rapid functional recovery was highly suggestive of suspected Takotsubo syndrome.

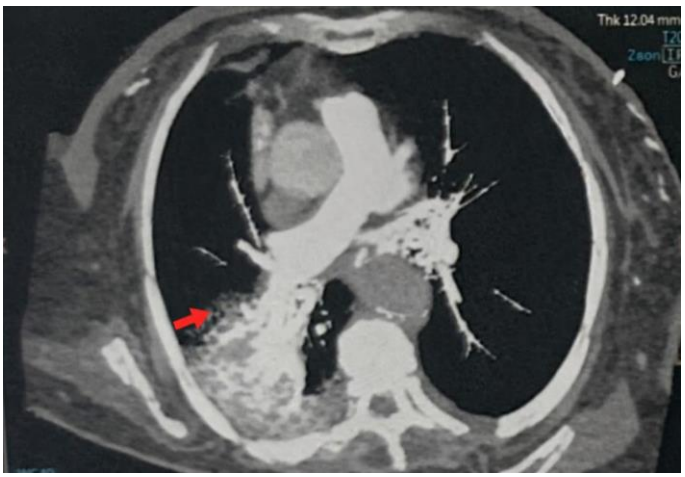


**Figure 1: Chest computed tomography (axial view) showing pulmonary parenchymal consolidation consistent with pneumonia (arrow)**

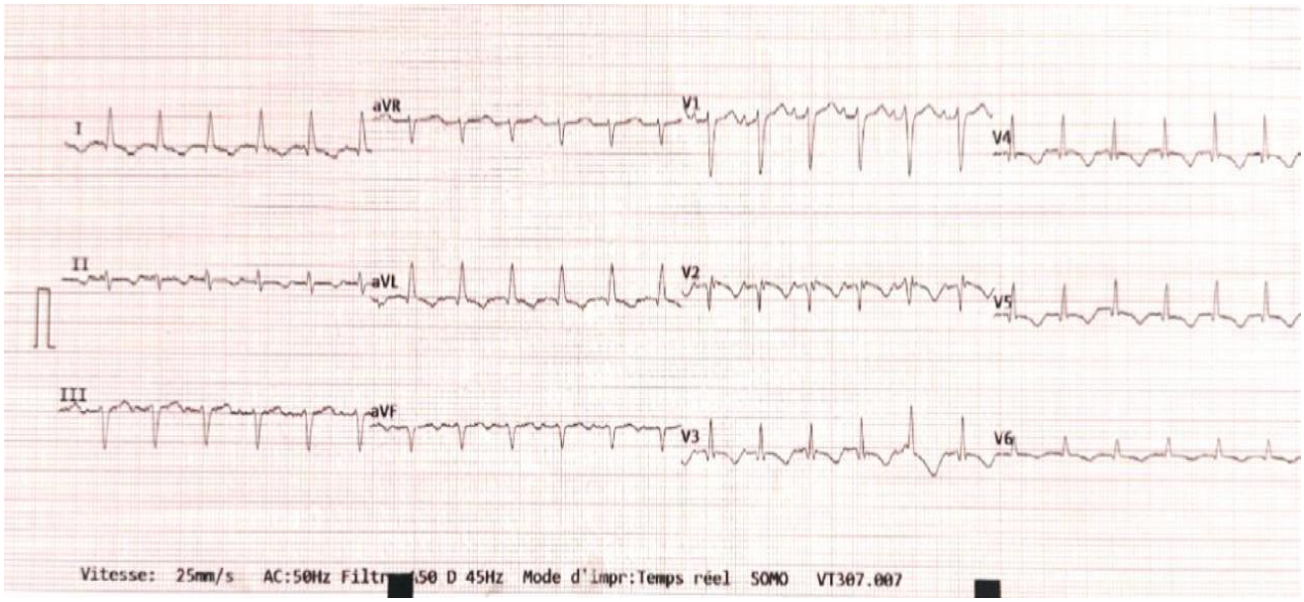


**Figure 2: Electrocardiogram showing sinus tachycardia with QT interval prolongation, without ST-segment elevation or depression**


**Figure 3: Initial transthoracic echocardiography demonstrating severe left ventricular systolic dysfunction, with akinesia of the apical and mid-ventricular segments and compensatory hyperkinesia of the basal segments, suggestive of stress-induced cardiomyopathy**

**Figure 4: Follow-up transthoracic echocardiography after favorable clinical evolution, showing recovery of left ventricular systolic function with marked improvement in regional wall motion**

## DISCUSSION

The occurrence of acute left ventricular dysfunction in patients admitted to the intensive care unit for septic shock represents a significant diagnostic challenge. Several mechanisms may be involved, including acute ischemic heart disease, septic cardiomyopathy, and stress-induced cardiomyopathy. In our case, the secondary onset of severe left ventricular systolic dysfunction in the setting of major physical stress related to sepsis rapidly led to suspicion of Takotsubo syndrome, although definitive diagnosis could not be confirmed in the absence of coronary angiography [1–3].

Sepsis is now recognized as a potential trigger for Takotsubo syndrome. Several series report a non-negligible incidence of this entity in critically ill patients, where physiological stress, systemic inflammation, and neurohormonal activation are particularly pronounced [4,5]. In our observation, septic shock secondary to severe pneumonia likely created a favorable milieu for acute myocardial dysfunction, possibly mediated by massive endogenous catecholamine release, potentially amplified by exogenous vasoactive agents, a mechanism well described in the literature [6].

Electrocardiographic abnormalities in Takotsubo syndrome are variable and nonspecific, typically including repolarization abnormalities, QT interval prolongation, and, less frequently, transient ST-segment elevation [7]. In our case, ECG showed sinus tachycardia with QT prolongation without ST-segment deviation. T-wave abnormalities could partly be explained by associated electrolyte disturbances, including hypokalemia, but remained consistent with stress cardiomyopathy patterns. The absence of clear ischemic ECG features made acute myocardial infarction less likely, although it could not be entirely excluded in the intensive care setting.

Transthoracic echocardiography represented the cornerstone of diagnostic orientation. It demonstrated severe left ventricular systolic dysfunction with extensive regional wall motion abnormalities not corresponding to a single coronary territory. This non-territorial pattern is a key feature supporting Takotsubo syndrome, as described in major series and international recommendations [2,3]. In our case, rapid and near-complete recovery of left ventricular function within one week constituted a major retrospective argument supporting the diagnostic suspicion.

The main differential diagnosis remains septic cardiomyopathy, a common entity in intensive care. It is typically characterized by global left ventricular dysfunction, sometimes associated with ventricular dilation, with recovery more gradual and closely dependent on infection control [8,9]. In our observation, the rapid improvement of left ventricular systolic function, relatively independent of respiratory and infectious evolution, contrasted with the typical profile of septic cardiomyopathy and supported a transient stress-induced cardiomyopathy. Acute coronary syndrome was another major differential diagnosis to exclude in the presence of acute left ventricular dysfunction. Coronary angiography remains the gold standard for ruling out obstructive coronary artery disease. However, in our case, the absence of ST-segment elevation, moderate and transient troponin elevation without prolonged kinetics, non-territorial echocardiographic abnormalities, and initial hemodynamic instability precluded coronary angiography in the acute phase, making extensive acute coronary syndrome unlikely. Acute myocarditis was also considered, but its clinical course is generally less rapidly favorable and is often associated with more pronounced and persistent elevation of cardiac biomarkers, as well as sustained ventricular dysfunction [10].

Management of Takotsubo syndrome is primarily supportive, focusing on hemodynamic stabilization and treatment of the triggering cause. In septic shock, this balance is particularly challenging, as vasoactive agents are often required to maintain tissue perfusion, despite their potential contribution to catecholamine-mediated myocardial toxicity [3,6]. In our case, management centered on treatment of the underlying infection, respiratory and circulatory support, and close cardiac monitoring, resulting in favorable outcome without major arrhythmic complications.

The observed evolution, characterized by near-complete recovery of left ventricular function within days, is consistent with published data reporting generally good short-term prognosis despite sometimes severe acute presentation [1,11]. This case highlights the importance of considering Takotsubo syndrome in any acute left ventricular dysfunction occurring in the context of major physical stress, particularly in critically ill patients. Early recognition allows appropriate management, avoidance of unnecessary invasive procedures, and anticipation of a generally favorable outcome.

## CONCLUSION

This case illustrates the diagnostic complexity of acute left ventricular dysfunction occurring in the setting of septic shock. In our observation, the combination of major physical stress, nonspecific electrocardiographic abnormalities, suggestive echocardiographic findings, and rapid and near-complete

recovery of left ventricular function led to strong suspicion of Takotsubo syndrome, although definitive diagnosis could not be formally established.

This report underscores the importance of considering Takotsubo syndrome in intensive care patients presenting with acute myocardial dysfunction, in order to tailor management, limit unnecessary invasive investigations, and optimize cardiac follow-up. Early recognition of this entity, even at the stage of suspicion, allows a rational therapeutic approach and better anticipation of the generally favorable clinical course.

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