

Tuberculosis and Atrioventricular Block in a Patient with Takotsubo Syndrome: A Case Report

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Case Report

An 83-year-old female patient diagnosed with senile dementia and experiencing unintentional weight loss over the past few months was referred to a tertiary hospital due to complaints of typical chest pain. There is no previous history of cardiovascular diseases.

During transport, the patient developed bradycardia, with a heart rate (HR) of 32 beats per minute, and upon arrival at the emergency room, it was observed that she was suffering from complete atrioventricular block (Figure 1) with episodes of non-sustained ventricular tachycardia.¹

The patient received clinical support and was referred to the hemodynamics laboratory to undergo urgent coronary angiography, which showed no coronary artery obstruction (Figure 2 and 3). During ventriculography, hypokinesia was observed in the anterior, apical, and inferolateral regions with significant ventricular dysfunction. A temporary pacemaker was then implanted (Figure 4), and the patient was transferred to the intensive care unit (ICU).^{2,3}

The transthoracic echocardiogram confirmed the segmental alterations observed in ventriculography and documented a significant reduction in left ventricular systolic function, with an ejection fraction by Simpson's method of 26% (Figure 5).

Laboratory tests showed a significant increase (> 10 times the upper limit of normality) in troponin levels, as well as elevated Pro-BNP and metabolic acidosis.⁴

After discussion with the arrhythmology team, it was decided to proceed with the implantation of a permanent pacemaker, and the possibility of Takotsubo syndrome was considered. During ICU admission, the patient developed hemodynamic instability with signs of cardiogenic shock, requiring inotropic support with Dobutamine 10 mcg/kg/min, vasodilators, diuretic therapy, and supplemental oxygen with positive pressure (BiPAP), resulting in partial improvement of the condition.

Keywords

Takotsubo Cardiomyopathy; Pulmonary Tuberculosis; Atrioventricular Block; Acute Coronary Syndrome

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After 48 hours of admission, the patient began to present cough and dyspnea, progressing to acute respiratory failure and circulatory shock, requiring invasive mechanical ventilation via endotracheal tube and administration of noradrenaline in addition to dobutamine. The patient responded well to treatment and was extubated two days later but remained dependent on inotropic support and intermittent positive pressure (BIPAP) therapy.

A chest computed tomography, performed after clinical stabilization, revealed pulmonary consolidations on the right, likely of infectious origin, as well as fibrocatricular alterations resulting in a volumetric reduction of the lingula, minimal pneumothorax in the left hemithorax, and mild bilateral pleural effusion. (Figure 6) After bronchoalveolar lavage, the Genexpert test was positive for pulmonary tuberculosis, and treatment with rifampicin, isoniazid, pyrazinamide, and ethambutol (RHZE) was initiated immediately. The patient began to present fever, psychomotor agitation, leukocytosis, and increased C-reactive protein (CRP), along with worsening renal function. It was decided to change invasive devices, including the transvenous pacemaker, and to start antibiotic therapy with Meropenem, Polymyxin B, and Amikacin. After 48 hours of antibiotic therapy initiation, the patient developed septic shock with multiple organ failure and deceased.⁵

Discussion

Takotsubo syndrome is a condition of acute reversible heart failure, first described in 1990 in a Japanese journal. It is increasingly recognized in modern cardiology practice due to access to coronary angiography for patients with acute chest pain. Additionally, it is now known that some conditions, such as pheochromocytoma and intracranial hemorrhage, may be related to the development of the syndrome.^{1,2}

The pathophysiology of Takotsubo Syndrome is complex and reflects the cardiovascular response to acute stress situations, resulting in an increase in catecholamines due to sympathetic stimulation. Catecholamines play a central role in this process, with cognitive centers of the brain and the hypothalamic-pituitary-adrenal axis being considered initial elements to be considered.

Although Takotsubo syndrome is generally considered benign, recent evidence has shown a comparable incidence of cardiogenic shock and death compared to acute coronary syndrome. During the acute phase, 25% of patients may experience serious adverse events such as hemodynamic and electrical instability. Several factors have been identified as predictors of adverse outcomes, including physical stress, neurological or psychiatric diseases, initial troponin elevation of over 10 times the reference value, and reduced left ventricular ejection fraction on admission.

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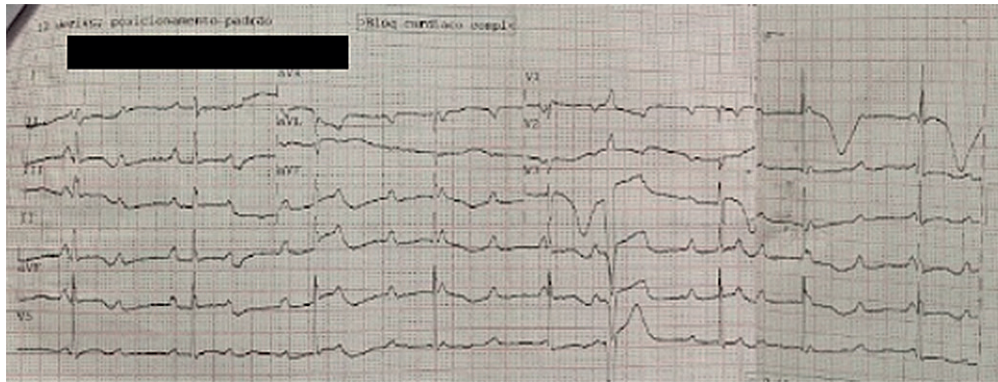


Figure 1 – Admission electrocardiogram showing complete atrioventricular block.



Figure 2 – Cardiac catheterization, demonstrating coronary arteries without lesions.

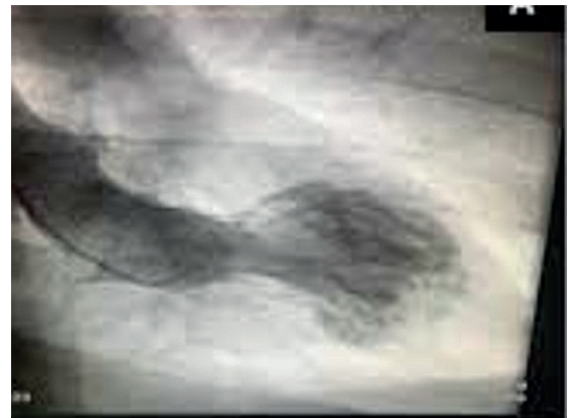


Figure 3 – Cardiac catheterization, revealing apical ballooning of the left ventricle during ventriculography.

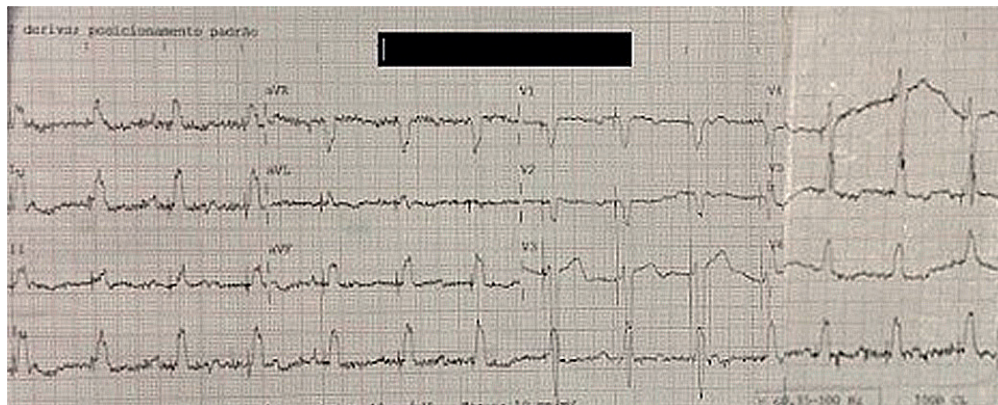


Figure 4 – Admission electrocardiogram in the intensive care unit showing pacemaker rhythm.

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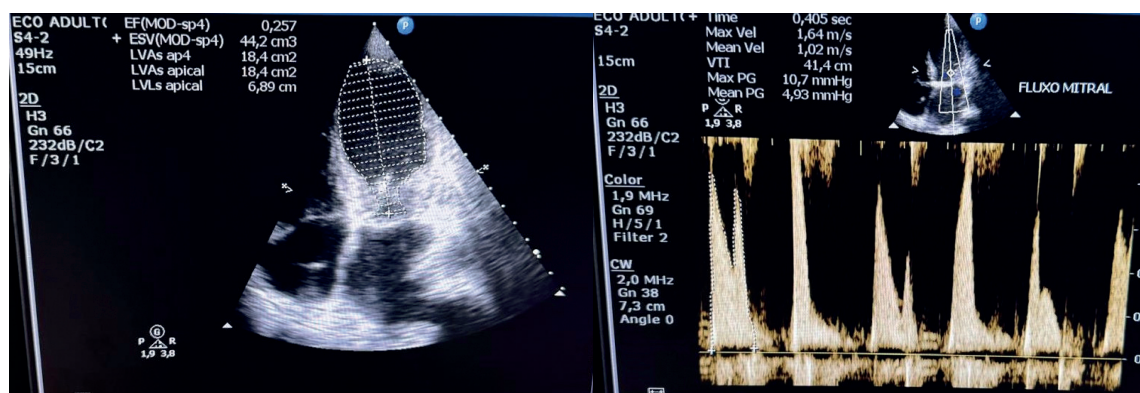


Figure 5 – Transthoracic echocardiogram shows akinesia of the mid-apical segments of all walls and preserved contractility only in the basal segments, with significant deterioration of the ejection fraction. Measurement by the Simpson method revealed an ejection fraction of 26%. The adjacent image also displays mitral flow, where the presence of cardiac arrhythmia compromised the assessment of diastolic function.

The reported case is complex and presents complications from the outset, including typical chest pain, electrocardiographic instability with complete atrioventricular block, and episodes of non-sustained ventricular tachycardia. Catheterization revealed segmental changes compatible with Takotsubo syndrome, along with increased troponin and deterioration of ejection fraction. The presence of pulmonary tuberculosis adds complexity to the case, with reports suggesting a possible association between the disease and elevated plasma noradrenaline concentrations, which could contribute to the development of Takotsubo syndrome in this patient.^{6,7} Other entities, such as cardiac sarcoidosis, although much less common, could also be considered due to some characteristics present in this case. However, a differential diagnosis through biopsy or autopsy was not performed.

In conclusion, the exact mechanisms for the development of Takotsubo syndrome are still not fully understood. Still, there seems to be a complex interaction between catecholamines, the central nervous system, and other factors. Although the incidence of the syndrome is still low, it is becoming more recognized as an important differential diagnosis of acute coronary syndrome.⁸⁻¹⁰

Author Contributions

Conception and design of the research; Acquisition of data; Analysis and interpretation of the data; Statistical analysis; Obtaining financing; Writing of the manuscript and Critical revision of the manuscript for content: Albarracin VKS

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

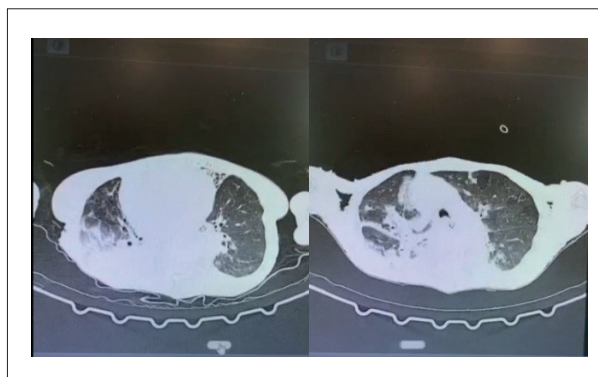


Figure 6 – Chest computed tomography shows extensive consolidations in the right lung, associated with scattered small consolidations throughout the left lung of nonspecific appearance. Fibrocatrictial alterations are causing a reduction of the lingula. There is also a small bilateral pleural effusion with adjacent compressive atelectasis.

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Study association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Liga Norteriograndense contra el Câncer under the protocol number 7.121.448. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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