

## Case report: inverted Takotsubo syndrome secondary to stress during anesthesia

### Reporte de caso: síndrome de takotsubo invertido secundario al estrés durante la anestesia

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### Case presentation

#### Patient information

A 68-year-old male with type 2 diabetes mellitus and renal lithiasis presented to the emergency department intubated and receiving vasopressor support. His condition deteriorated intraoperatively during preparation for lithotripsy under anesthesia. Following a subarachnoid block, he developed a hypertensive crisis, profuse diaphoresis, precordial pain, vertigo, and subsequent syncope. The anesthesiology team-initiated intubation, central venous catheter placement, and vasopressor administration.

#### Clinical findings

Electrocardiography (ECG) showed significant ST-segment depression in the inferoanterior leads (DII, DIII, aVF, V3-V6) (Fig. 1A), raising concerns about an acute coronary event. Initial blood tests revealed a

mildly elevated troponin I level of 0.1 ng/mL, suggesting possible myocardial injury. Upon admission to the coronary care unit, troponin I increased to 378 ng/mL. A follow-up ECG demonstrated ST-segment normalization (Fig. 1B), while a chest radiograph revealed pulmonary edema.

#### Diagnostic assessment

Transthoracic echocardiography identified significant hypokinesia of the inferior wall with a reduced left ventricular ejection fraction (LVEF) of 37% (Fig. 1C), prompting urgent coronary angiography and ventriculography. Coronary angiography demonstrated normal epicardial vessels without stenosis or obstruction (Fig. 2A-E). Ventriculography revealed akinesia of the inferior wall and pronounced hyperkinesia of the apical region (Fig. 2F-I), consistent with inverted Takotsubo cardiomyopathy (TTC), a rare reverse variant of stress-induced cardiomyopathy.

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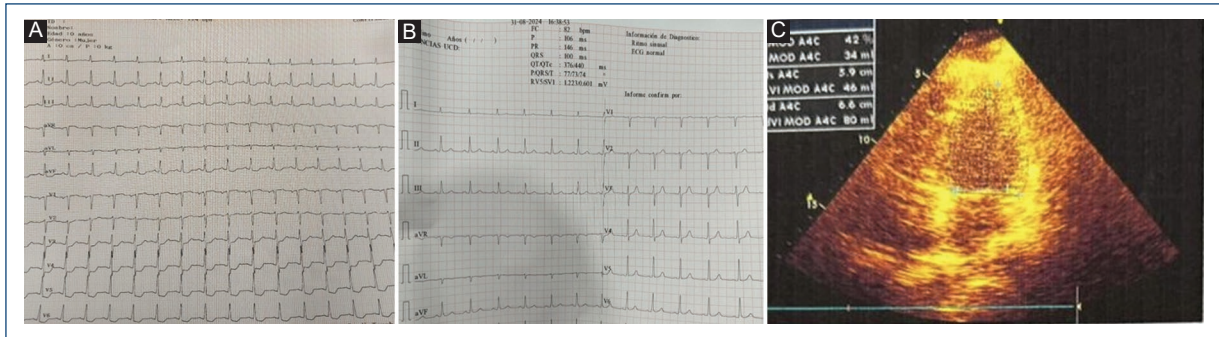
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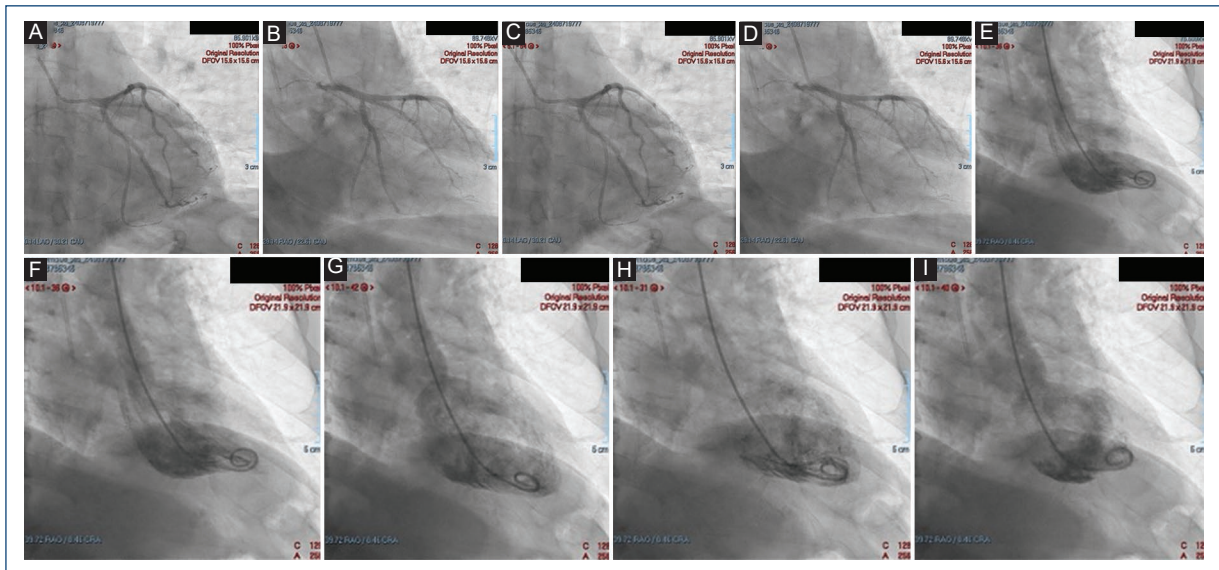
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**Figure 1.** **A:** follow-up 12-lead electrocardiogram (ECG). Showing ST-segment depression in the inferoanterior leads (DII, DIII, aVF, and V3-V6), suggestive of myocardial ischemia, a finding common in Takotsubo Cardiomyopathy, despite the absence of obstructive coronary artery disease. **B:** initial ECG. Showing normalization of the previously depressed ST segments, indicative of the transient nature of ST-segment changes typically seen in Takotsubo Cardiomyopathy. **C:** transthoracic echocardiogram. Highlighting hypokinesia of the inferior wall and hyperkinesia of the apical region, consistent with Inverted Takotsubo Cardiomyopathy of ST-segment changes typically seen in Takotsubo Cardiomyopathy.



**Figure 2.** **A and B:** coronary angiographic. Anteroposterior projection showing the left main coronary artery, left anterior descending (LAD) artery, and circumflex artery (Cx) in their proximal segments, without significant lesions. **C and D:** coronary angiographic. Left anterior oblique projection at 45°, showing the mid and proximal segments of the LAD artery, along with diagonal and septal branches. At 25°, diagonals and the posterior interventricular (PIV) branch of the Cx are observed, with no significant lesions. **E:** coronary angiographic. Right anterior oblique projection at 30°, showing the right coronary artery, posterolateral branch, and PIV branch, without significant lesions. **F-I:** cardiac ventriculography. Left ventriculography: apex contraction and base akinesis.

### **Therapeutic intervention**

The patient was managed with diuretic therapy (40 mg of furosemide), sedation (midazolam and propofol), and norepinephrine titration to maintain hemodynamic stability (blood pressure 120/78 mmHg, heart rate 86 bpm). Given the absence of obstructive coronary artery disease,

standard heart failure therapies were considered. Alternative management strategies, including  $\beta$ -blockers (esmolol, bisoprolol) and inotropic agents (dopamine, dobutamine), were evaluated. Additionally, levosimendan was considered due to its calcium-sensitizing properties, which may enhance left ventricular function without exacerbating Left ventricular outflow tract obstruction (LVOTO).

## ***Follow-up and outcomes***

The patient remained hemodynamically stable, and LVEF improved progressively over the following weeks. Follow-up echocardiography at 1 month demonstrated near-complete resolution of wall motion abnormalities and improved cardiac function. He was discharged on guideline-directed medical therapy with  $\beta$ -blockers and an Angiotensin-converting-enzyme (ACE) inhibitor. Long-term management strategies, including stress reduction, lifestyle modifications, and close monitoring for recurrence, were discussed.

## **Discussion**

### ***Pathophysiology and clinical considerations***

TTC, or “stress cardiomyopathy,” was first described in 1983 in Japanese females. It predominantly affects postmenopausal women and is triggered by emotional or physical stress<sup>1</sup>. It mimics acute coronary syndrome without obstructive coronary artery disease on angiogram. Besides the classic apical variant, three additional patterns exist: inverted (reverse), mid-ventricular, and localized types. Catecholamine-induced cardiotoxicity is a leading theory in TTC pathophysiology, with animal models demonstrating acute reversible hypokinesia after catecholamine administration, associated with myocardial contraction band necrosis and inflammation<sup>2</sup>.

### ***Therapeutic approaches and alternative management***

While standard heart failure therapies are commonly used, there are no established guidelines for TTC management. Vasopressors, such as norepinephrine and vasopressin, are controversial due to their potential to exacerbate LVOTO<sup>3</sup>. Some studies advocate for inotropic agents, such as dopamine or dobutamine, which may restore cardiac function without increasing LVOTO risk<sup>4</sup>.  $\beta$ -blockers like esmolol and bisoprolol have shown efficacy in reducing the LVOTO gradient and improving outcomes<sup>5</sup>. Levosimendan, a calcium-sensitizing agent, has been found to enhance left ventricular function in TTC-related shock<sup>6</sup>. Given these options, a tailored approach based on hemodynamic parameters is essential. The role of antithrombotic therapy remains debated, particularly in the absence of obstructive coronary artery disease. Additionally, novel therapeutic

strategies, including ivabradine and ranolazine, have been proposed to mitigate recurrent episodes in select patients<sup>7</sup>.

### ***Long-term management and prevention***

Despite a favorable prognosis, with over 90% of patients recovering within 1-2 months, recurrence rates range from 1.8 to 10%<sup>8</sup>. This case highlights the need for structured long-term strategies, particularly in male patients with diabetes, who have a higher risk of mortality. Studies indicate that diabetes and high Killip classification at admission are independent mortality predictors in male TTC patients<sup>9</sup>. Preventative strategies include  $\beta$ -blocker therapy, which has been associated with lower recurrence and mortality rates than ACE inhibitors or angiotensin receptor blockers (ARBs)<sup>10</sup>. Additionally, lifestyle modifications such as stress management, reduced caffeine intake, and smoking cessation may help prevent recurrence. Recent studies have also suggested the potential role of sodium-glucose transport protein 2 inhibitors in mitigating cardiovascular risk in diabetic patients with TTC<sup>9</sup>.

### ***Patient perspective***

Upon follow-up, the patient reported improved functional capacity and adherence to prescribed medical therapy. He was counseled on lifestyle modifications to mitigate recurrence risk, including dietary adjustments, glycemic control, and stress management techniques.

## **Conclusion**

This case underscores the importance of recognizing atypical TTC presentations and implementing individualized treatment strategies. Expanding research on therapeutic alternatives, particularly in male patients, remains crucial for optimizing outcomes and preventing recurrence. Future studies should focus on refining guidelines for TTC management, integrating emerging pharmacologic therapies, and tailoring preventive measures based on patient-specific risk factors.

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## Conflicts of interest

The authors declare that they have no conflicts of interest.

## Ethical considerations

**Protection of humans and animals.** The authors declare that no experiments were performed on humans or animals for this research.

**Confidentiality, informed consent, and ethics approval.** The authors followed their institution's confidentiality protocols, obtained informed consent from the patients, and received approval from the Ethics Committee. The recommendations of the SAGER guidelines were followed, based on the nature of the study.

**Declaration on the use of artificial intelligence.** The authors declare that they did not use any type of generative artificial intelligence in the writing of this manuscript.

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