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Case Reports

Unexpected Heartbreak: Takotsubo Cardiomyopathy Induced by Esophagogastroduodenoscopy

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Abstract

Takotsubo cardiomyopathy (stress cardiomyopathy, or broken-heart syndrome), characterized by reversible left ventricular dysfunction without clinically significant coronary artery obstruction, is rare but has been more commonly reported recently. Esophagogastroduodenoscopy as a cause of takotsubo cardiomyopathy remains exceedingly rare. The case report of an 84-year-old female patient with a complex medical history who had acute stress cardiomyopathy following esophagogastroduodenoscopy, which required prolonged hospitalization and hemodynamic support, is presented. This case emphasizes the importance of recognizing and considering this uncommon pathology and its complications in differential diagnosis. Diagnostic modalities and treatment options for this condition are also discussed.

Keywords: Takotsubo cardiomyopathy; endoscopy, digestive system; heart failure

Case Report

Presentation and Physical Examination

An 84-year-old Hispanic female patient presented to the emergency department after having experienced coffee ground emesis, generalized abdominal pain, and dyspnea on exertion for 2 weeks. Her vital signs included a temperature of 98.4 °F, a heart rate of 90/min, blood pressure of 134/60 mm Hg, a respiratory rate of 20/min, and oxygen saturation of 98% on room air. Physical examination revealed pale conjunctiva and dry mucous membranes. The abdomen was mildly tender, with firmness over the epigastric and umbilical regions. Lung auscultation revealed clear bilateral air entry, with no adventitious sounds, and cardiac examination showed normal S_1 and S_2 sounds without murmurs, gallops, or friction rubs.

Medical History

The patient had a medical history of type 2 diabetes, hypertension, and hyperlipidemia.

Differential Diagnosis

During differential diagnosis, the following conditions were considered and tested for: (1) upper gastrointestinal bleeding (eg, peptic ulcer disease, malignancy), (2) acute coronary syndrome (ACS), (3) pulmonary embolism, (4) acute heart failure (HF) with reduced ejection fraction, (5) anemia of chronic disease, (6) gastrointestinal malignancy, and (7) stress-induced (or takotsubo) cardiomyopathy.

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Technique

Initial laboratory results indicated iron deficiency anemia, but other parameters were within normal limits (Table I). An electrocardiogram demonstrated normal sinus rhythm, with no ST-segment or T-wave changes (Fig. 1). A chest x-ray showed no evidence of acute cardiopulmonary pathology. Intravenous pantoprazole was initiated, and 3 units of packed red blood cells were transfused.

Transthoracic echocardiography performed to assess cardiac risk before esophagogastroduodenoscopy (EGD) showed a left ventricular ejection fraction (LVEF) of 50% to 55%, with normal LV size, wall thickness, and diastolic function (Fig. 2). The EGD revealed a nonbleeding gastric ulcer (Forrest class III). Examination of the duodenum was incomplete because of a mass that obstructed the gastric outlet, confirmed by biopsy to be a moderately differentiated gastric adenocarcinoma.

Key Points

- **Unexpected trigger.** Minor procedures like EGD can induce TCM in rare instances.
- **Diagnostic challenge.** Sudden cardiac dysfunction after EGD requires prompt differentiation from acute coronary events.
- **Successful recovery.** Early recognition and supportive care lead to substantial cardiac function recovery in most patients with TCM.

Abbreviations

ACS, acute coronary syndrome
EGD, esophagogastroduodenoscopy
HF, heart failure
LVEF, left ventricular ejection fraction
TCM, takotsubo cardiomyopathy

Following EGD, the patient developed severe respiratory distress with chest pain. She became hypertensive (blood pressure, 170/80 mm Hg), tachycardic

TABLE I. Patient Laboratory Data on Admission

Variable	Admission laboratory value	Reference range
Blood urea nitrogen, mg/dL	20	6-24
Creatinine, mg/dL	0.9	0.6-1.2
Aspartate aminotransferase, U/L	17	10-36
Alanine aminotransferase, U/L	17	9-46
Brain-type natriuretic peptide, pg/mL	101	0-100
High-sensitivity troponin, ng/L	4	<78
Lactic acid, mmol/L	1.0 L	0-2.0
White blood cell count, / µL	6.8 × 103	4,400-11,000
Hemoglobin, g/dL	5.4	13.5-17.5
Mean corpuscular volume, fL	83.4	81.6-98.3
Platelet count, /µL	437	150,000-450,000
Fecal occult blood test	Positive	Negative
Iron, µg/dL	10	34-145
Iron saturation, %	17.6	20-55
Total iron binding capacity, µg/dL	346	200-400
Ferritin, ng/mL	8.5	11-307

SI conversion factor: To convert mg/dL to mmol/L (blood urea nitrogen), multiply by 0.357. To convert mg/dL to µmol/L (creatinine), multiply by 76.25. To convert U/L to µkat/L, multiply by 0.0167. To convert pg/mL to ng/L, multiply by 1. To convert /µL to × 109/L, multiply by 0.001. To convert g/dL to g/L, multiply by 10. To convert µg/dL to µmol/L, multiply by 0.179. To convert ng/mL to µg/L, multiply by 1.

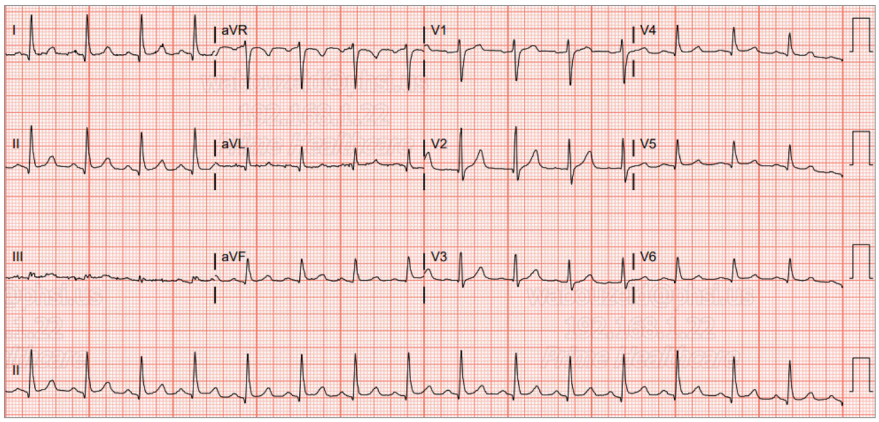

Fig. 1 Electrocardiogram on admission shows normal sinus rhythm, with a heart rate of 90/min and no ST-segment or T-wave changes.

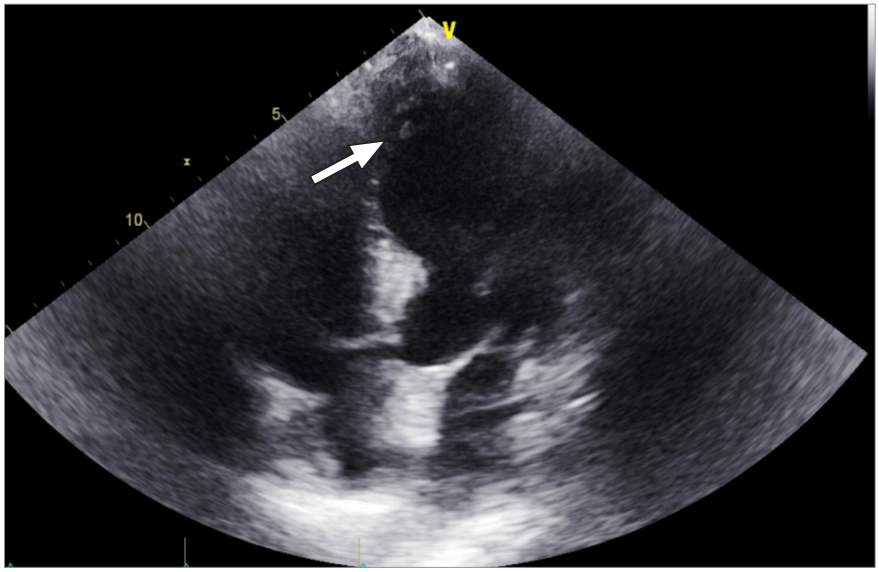

Fig. 2 Transthoracic echocardiogram on admission shows an apical 4-chamber view with normal left ventricular size, wall thickness, and diastolic function; no apical ballooning (arrow); and an estimated left ventricular ejection fraction of 50% to 55%.

(heart rate, 110/min), and tachypneic (respiratory rate, 24/min), and her oxygen saturation dropped to 80% on room air. Arterial blood gas analysis indicated severe hypoxia, with a partial oxygen pressure of 57 mm Hg on 60% fraction of inspired oxygen. The patient was intubated, and a chest x-ray showed bilateral lung infiltrates suggestive of pulmonary congestion.

A subsequent electrocardiogram showed sinus tachycardia at 110/min, new-onset left bundle branch block, and ST-segment elevations in leads V_1 through V_3 (Fig. 3). High-sensitivity troponin levels increased from normal to 0.653 μg/L (653 ng/L), peaking over 5 days before trending downward (Table II, Fig. 4). Repeat transthoracic echocardiography demonstrated a severely reduced LVEF of 10% to 15%, moderate LV dilatation, apical ballooning, mild left atrial dilatation, and severe global hypokinesia consistent with stress cardiomyopathy (Fig. 5).

Coronary angiography confirmed patent left main, left anterior descending, left circumflex, and right coronary arteries (Fig. 6A and 6B). Supportive management, including guideline-directed medical therapy for HF, was initiated. Transthoracic echocardiography on day 10 showed LVEF improvement to 30% to 35%, with global hypokinesia sparing the basal segments (Fig. 7). By day 15, LVEF had improved further to 40% to 45%, with regional wall motion abnormalities and areas of asynchronous contraction (Fig. 8). Laboratory results at discharge showed a troponin level of 0.326 μg/L (326 ng/L) and a hemoglobin level of 100 g/L (10 g/dL).

Outcome

The patient was discharged to a subacute rehabilitation center on optimal guideline-directed medical therapy for HF and enrolled in a physical therapy program.

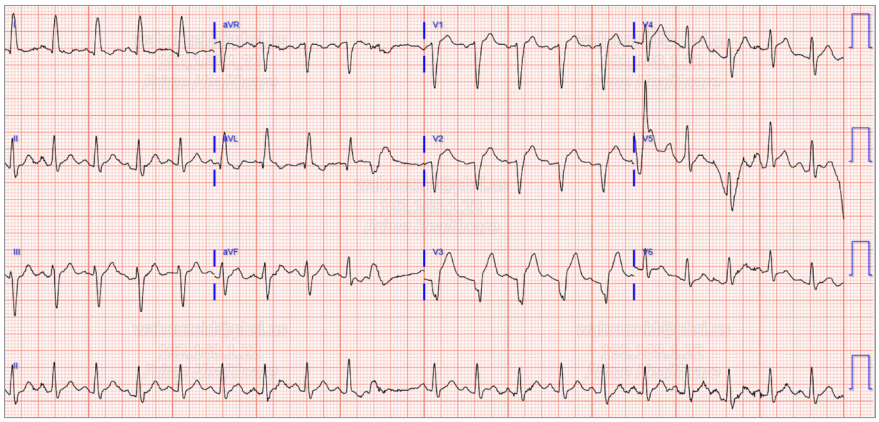

Fig. 3 A repeat electrocardiogram after esophagogastroduodenoscopy shows sinus tachycardia, with a heart rate of 110/min and clinically significant changes, including developing new-onset left bundle branch block and ST-segment elevation in leads V_1, V_2, and V_3.

TABLE II. High-Sensitivity Troponin Levels During Hospitalization

Timing	High-sensitivity troponin level, ng/L	Reference range, ng/L
On admission	4	<78
After EGD	653	<78
Day 3	7,127	<78
Day 5	8,923	<78
Day 10	3,251	<78
Day 15	326	<78

Abbreviation: EGD, esophagogastroduodenoscopy.

SI conversion factor: To convert ng/L to µg/L, multiply by 0.001.

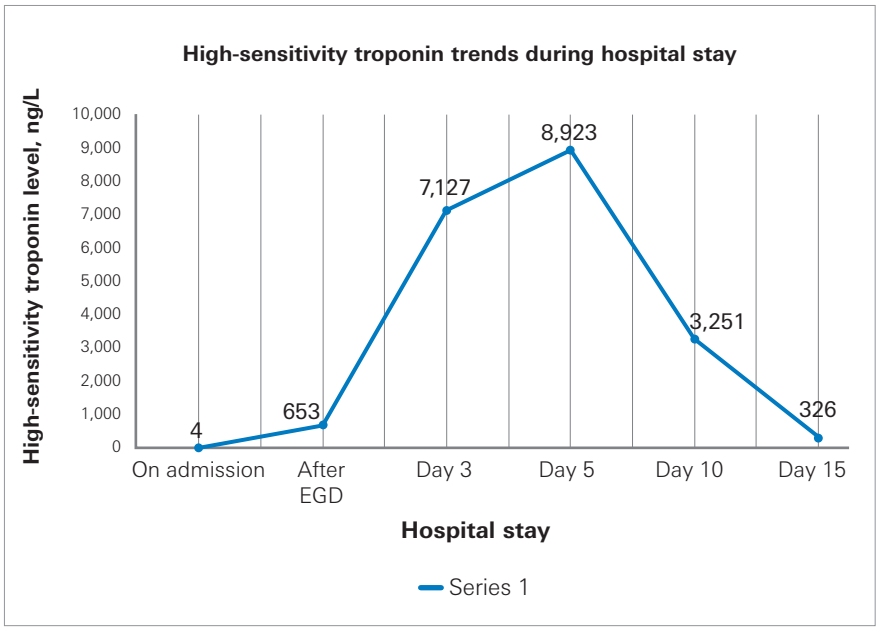

Fig. 4 High-sensitivity troponin trend during hospital stay.

EGD, esophagogastroduodenoscopy.

SI conversion factor: To convert ng/L to µg/L, multiply by 0.001.

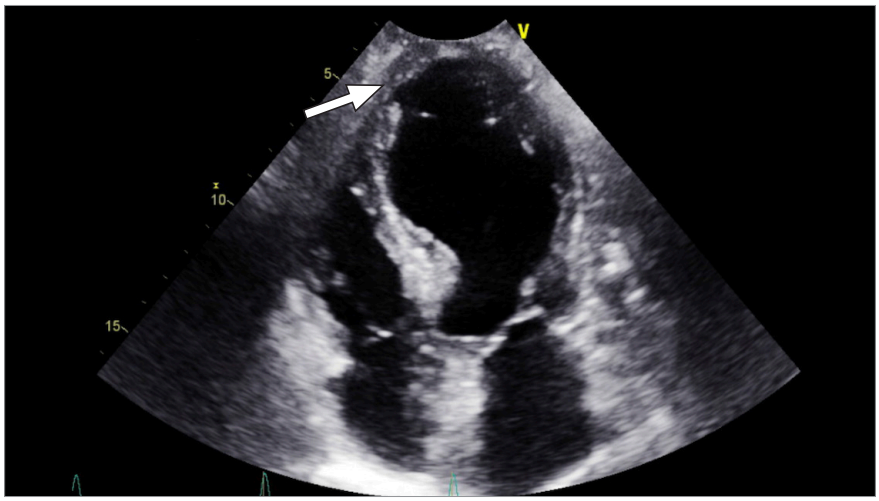

Fig. 5 Transthoracic echocardiogram after esophagogastroduodenoscopy shows an apical 4-chamber view with an estimated left ventricular ejection fraction of 10% to 15%, moderate left ventricular dilatation with apical ballooning (arrow), and mild left atrial dilatation.

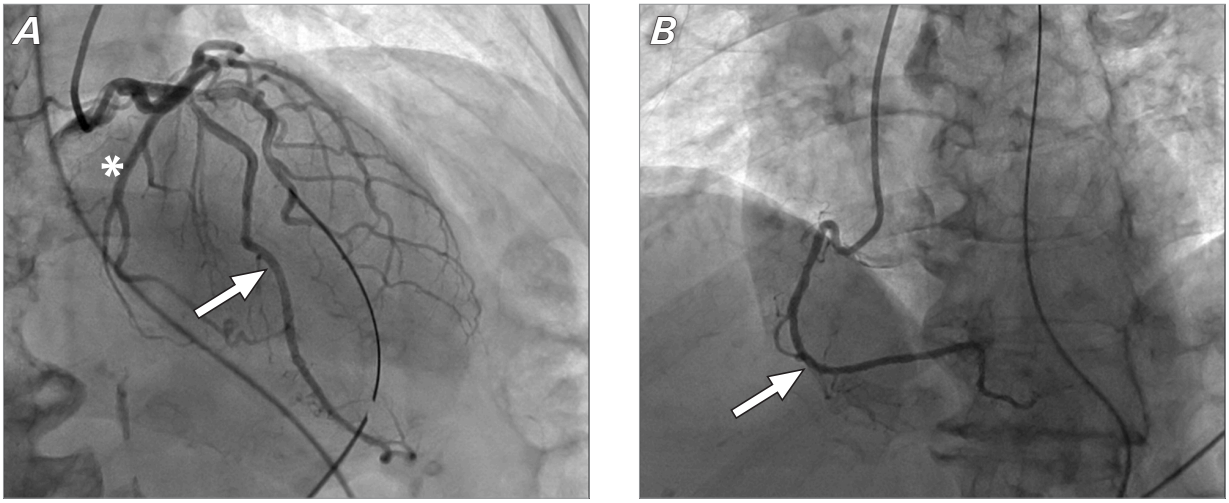

Fig. 6 (**A**) Coronary angiogram shows the patent left anterior descending coronary artery (arrow) and a patent left circumflex coronary artery (asterisk); (**B**) coronary angiogram shows a patent right coronary artery (arrow)

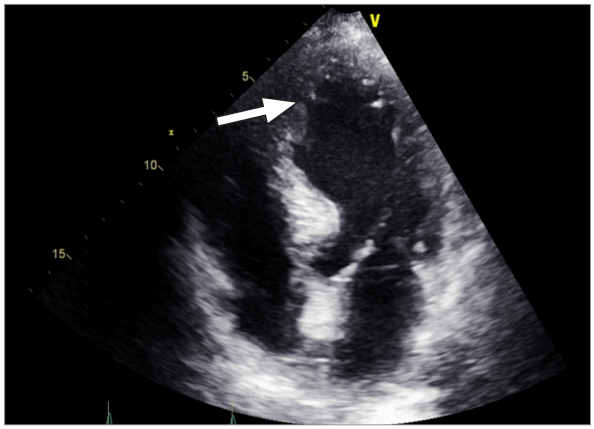

Fig. 7 A repeat transthoracic echocardiogram on day 10 shows an apical 4-chamber view with improved left ventricular ejection fraction and a decrease in the degree of apical ballooning (arrow).

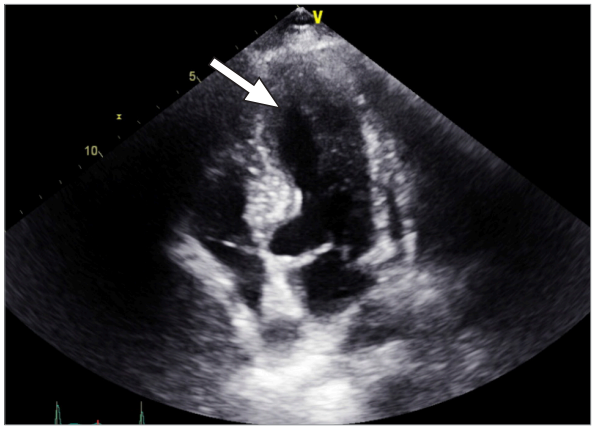

Fig. 8 Transthoracic echocardiogram on day 15 shows an apical 4-chamber view with resolution of apical ballooning (arrow) and an estimated left ventricular ejection fraction of 40% to 45%.

Last Follow-Up

At her 2-week follow-up appointment, the patient reported no HF symptoms. She remained clinically stable, with improved functional capacity and no signs of volume overload. Her guideline-directed medical therapy regimen was continued, and she was advised to follow a low-sodium diet. A follow-up appointment was scheduled in 3 months to reassess her cardiac function and adjust therapy as needed.

Discussion

Heart failure is a common clinical condition characterized by impaired ventricular filling or ejection of blood as a result of anatomic or functional cardiac problems. Even though takotsubo cardiomyopathy (TCM) is an uncommon cause of HF, it should be considered a crucial differential diagnosis. Takotsubo cardiomyopathy is distinguished by reversible LV failure in the absence of severe coronary artery blockage, and the term derives from the echocardiographic similarity to traditional takotsubo, or octopus fishing pots. This report presents the case of a patient who developed TCM after EGD.[1]

Takotsubo cardiomyopathy was once thought to be a rare pathology specific to Eastern Asian populations. The literature suggests, however, that it accounts for 1% to 2% of cases of hospitalized patients with suspected acute myocardial infarction.[2] Takotsubo cardiomyopathy was first identified in 1990 by Japanese scientists and affects mainly postmenopausal women between 60 and 75 years of age.[3] Although TCM most commonly occurs following physical or emotional stressors, it can occur after a minor procedure like EGD. Patients usually present with substantially variable symptoms, the most common of which are chest pain and dyspnea.[1]

Takotsubo cardiomyopathy is characterized by reversible contractile dysfunction at the apex and up to the midportion of the left ventricle, with sparing of the basal segment and without substantial coronary arterial blockage. Although TCM commonly occurs following physical or emotional stressors, the exact mechanism remains unclear. Theoretically, contractile dysfunction in TCM was thought to be secondary to exaggerated sympathetic stimulation, which can lead to epicardial coronary arterial spasm, microvascular spasm with abnormal coronary flow, or catecholamine surge that decreases the viability of myocytes.[4]

Diagnosing TCM can be challenging because patients with the disorder present with symptoms similar to ACS, including anginal chest pain; shortness of breath; and, in severe cases, hemodynamic instability. These patients should be evaluated urgently and promptly because the treatment, prognosis, and possible complications of TCM differ from those of ACS. Electrocardiographic abnormalities are common in patients with TCM, including ST-segment elevation, T-wave inversion, and QT-segment prolongation mimicking ACS. In addition, the cardiac enzyme (high-sensitivity troponin) is usually elevated in patients with TCM. Even so, the elevation is milder, does not correlate with the extent of heart dysfunction, and returns to baseline faster than it does in ACS.[5]

Given that a patient's TCM symptoms are similar to those of a patient with ACS, coronary angiography is an essential part of the investigation to rule out coronary artery disease. The detection of wall dyskinesia that extends beyond the distribution of a single heart artery is nearly pathognomonic for this dysfunction, so echocardiography is the most helpful diagnostic tool in identifying TCM.[1,5] There are no clear recommendations regarding TCM treatment, but as a general rule, management should focus on supportive care, monitoring, hemodynamic stabilization based on the severity of the cardiomyopathy, and prevention of common complications until normal cardiac function is recovered. Patients with severe cardiomyopathy should be observed in high-dependency units, with pharmacologic and mechanical support in cases of hemodynamic instability.[5,6]

Conclusion

Takotsubo cardiomyopathy, characterized by reversible ventricular contractile dysfunction, is a rare but potentially life-threatening complication of emotional or physical stressors. It most commonly occurs following substantial stress, sickness, or major procedures, and TCM secondary to EGD is exceptionally rare. This case report highlights the importance of considering TCM, even after minor procedures like EGD, when evaluating patients with acute HF and contractile dysfunction. Early recognition of and prompt intervention for TCM are crucial for successful management.

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