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| RESEARCH ARTICLE

From Pancreas to Heart: Takotsubo Cardiomyopathy Following Acute Pancreatitis

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| ABSTRACT

In cases of severe acute pancreatitis, clinicians must maintain a vigilant approach toward potential cardiac complications, particularly when confronted with unexplained electrocardiographic changes or modest elevations in cardiac biomarkers such as troponin. This report presents a compelling example, detailing the clinical course of a 62-year-old woman who arrived at the emergency department with acute-onset, intense epigastric pain radiating to the back, accompanied by worsening nausea and multiple episodes of non-bilious, non-bloody vomiting. Though her cardiac symptoms were limited to subtle palpitations and mild dyspnea, further evaluation revealed mildly elevated troponin levels, a known systemic inflammatory trigger, and hallmark echocardiographic findings of apical ballooning with basal hyperkinesis—collectively fulfilling the diagnostic criteria for Takotsubo cardiomyopathy (TCM). This diagnosis demanded a nuanced management strategy, balancing the need for intravenous fluid resuscitation to support pancreatic perfusion against the risk of precipitating or exacerbating left ventricular dysfunction. Continuous ECG surveillance and echocardiographic assessment of ventricular filling were essential to guide volume status and avoid complications such as iatrogenic pulmonary edema or potentially life-threatening arrhythmias, including QT prolongation—particularly in the context of antiemetic therapy with agents such as ondansetron. The patient was managed conservatively for pancreatitis, while cardiac dysfunction was addressed with a short course of beta-blockers and angiotensin-converting enzyme inhibitors, titrated carefully in accordance with hemodynamic parameters. This case underscores the increasingly recognized association between systemic inflammatory states and stress-induced cardiomyopathy, revealing a notable gap in current clinical guidelines and emphasizing the critical need for increased diagnostic vigilance and interdisciplinary coordination in managing such complex presentations.

| KEYWORDS

Takotsubo cardiomyopathy, Heart failure, Stress-induced cardiomyopathy, Acute pancreatitis, Acute abdomen.

| ARTICLE INFORMATION

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1. Introduction

Takotsubo cardiomyopathy (TCM) – also called stress-induced, apical ballooning, or "broken heart" syndrome – is an acute, reversible left ventricular systolic dysfunction characterized by apical ballooning of the heart muscle [1,2]. Clinically, it mimics an acute myocardial infarction (with acute chest pain, ST-T changes on ECG, and modest troponin elevation) but, by definition, occurs without obstructive coronary artery disease [1,3]. The term "takotsubo" refers to a Japanese octopus trap whose shape resembles the systolic form of the ventricle in this syndrome [2]. Takotsubo is relatively uncommon, accounting for about 1–2% of patients initially suspected of acute coronary syndrome [2]. There is a striking gender bias – roughly 90% of cases occur in post-menopausal women (mean age ~66 years) [2,3] – suggesting hormonal factors. When men are affected, it is often in the

setting of a severe physical illness and carries a worse short-term prognosis. The precise pathogenesis of Takotsubo cardiomyopathy (TCM) remains incompletely understood, but a surge of catecholamines from intense stress is thought to play a central role [1,3]. Excessive adrenergic stimulation may induce microvascular spasm, direct myocardial stunning or toxicity, and abnormalities in intracellular calcium handling, leading to transient myocardial dysfunction [1,2]. Additional contributing mechanisms include altered coronary microcirculation, systemic inflammation, and the loss of estrogen's cardioprotective effects in postmenopausal women [2,3]. In clinical practice, TCM is almost always precipitated by an identifiable stressor. These triggers may be emotional, such as grief, domestic violence, financial hardship, or episodes of intense fear or anger. Physical stressors, including acute medical illnesses, surgical procedures, trauma, and severe pain such as that caused by pancreatitis, are also well recognized. Less commonly, other precipitants such as stimulant drug use, thyroid storm, or even positive "happy heart" events have been reported [2]. Taken together, these factors—especially when combined with patient predispositions such as older age and female sex—create the conditions for the transient myocardial stunning characteristic of TCM. Patients with TCM typically present with features closely resembling acute coronary syndrome. The most common presenting symptoms are sudden-onset chest pain and dyspnea, although some patients may experience syncope, palpitations, or non-specific complaints [3]. Physical findings can vary widely, ranging from a normal examination to signs of acute heart failure such as pulmonary rales or an S3 gallop, and in severe cases, features of cardiogenic shock may be evident. Electrocardiographic changes are common and frequently mimic those seen in anterior myocardial infarction. Diffuse ST-segment elevation and deep T-wave inversions are the most typical abnormalities, with ST-segment elevation often appearing initially, followed by subsequent T-wave inversions. These changes tend to be less localized to a specific coronary artery territory than those seen in myocardial infarction [2,3]. Biochemical testing usually reveals modest elevations in troponin and CK-MB relative to the degree of left ventricular dysfunction, while levels of BNP or NT-proBNP are disproportionately high [2,3]. Imaging with transthoracic echocardiography often demonstrates akinesis or dyskinesis of the mid-apical segments of the left ventricle with basal hypercontractility, creating the pathognomonic "apical ballooning" appearance. Several morphological variants, including midventricular, basal, and focal forms, have also been described. Left ventricular ejection fraction may decline to below 35%, and right ventricular involvement is observed in some cases [2]. Coronary angiography typically demonstrates unobstructed coronary arteries or only minor, non-culprit lesions, thereby distinguishing TCM from acute plaque rupture [2,3]. Many patients undergo urgent angiography due to the initial suspicion of ST-elevation myocardial infarction before the diagnosis of TCM is established [2]. The modified Mayo Clinic criteria remain the standard diagnostic framework. These require the presence of transient left ventricular wall motion abnormalities extending beyond a single vascular territory, new ECG abnormalities or troponin elevation, the absence of obstructive coronary artery disease or acute plaque rupture, and the exclusion of pheochromocytoma and myocarditis [3]. Cardiac magnetic resonance imaging can provide additional support by confirming wall-motion abnormalities and excluding alternative diagnoses. Because TCM mimics ACS, distinguishing them is essential. In TCM, wall-motion abnormalities extend beyond a single artery's territory [2,3]. Troponin levels are lower and BNP levels higher than in transmural MI. Rapid recovery of LV function over days to weeks, unlike infarction, also supports TCM [2]. Acute pancreatitis (AP) is an acute necroinflammatory process of the pancreas, most often caused by gallstones or alcohol, but also by hypertriglyceridemia, drugs, infections, or trauma [4]. It typically presents with acute epigastric pain radiating to the back, nausea, vomiting, and elevated serum amylase/lipase [4]. About 80% of cases are mild and self-limited, but ~20% are severe, leading to necrosis and profound systemic inflammation [4]. Severe AP can trigger systemic inflammatory response syndrome (SIRS), causing vasodilation, capillary leak, and multiorgan injury [4,5]. Complications include ARDS, AKI, hypotension/shock, and DIC, with mortality around 20% in severe cases [4]. Thus, AP is not limited to the pancreas – it can involve lungs, kidneys, and cardiovascular system, often requiring intensive care [4,5]. Though emotional stress is the classic trigger, acute illnesses such as AP are increasingly recognized as precipitants [1]. Severe pain, systemic inflammation, and stress of AP can rarely provoke TCM [1,2]. The mechanism likely involves catecholamine surge, electrolyte shifts, and metabolic disturbances. Catecholamines can cause calcium overload in myocytes, hypokalemia, and hypomagnesemia, promoting arrhythmias and stunning [1]. Case reports confirm pancreatitis-induced TCM often presents electrolyte imbalances [1]. Overall, only a few dozen cases have been reported. Yeh et al. described TCM in a young man with AP [6]. Dhruv et al. reviewed 15 such cases (including two of their own), highlighting the rarity but importance of recognition [1]. Recognizing TCM in the context of pancreatitis is crucial, as the clinical overlap with acute coronary syndrome can lead to misdiagnosis and potentially harmful interventions such as inappropriate thrombolysis [6]. A high index of suspicion should be maintained, particularly in pancreatitis patients with electrolyte disturbances or new electrocardiographic abnormalities, and such patients should undergo continuous telemetry monitoring [1]. Early echocardiography is essential to differentiate TCM from other causes of hemodynamic instability [1]. Management involves addressing both pancreatitis and the cardiomyopathy. For pancreatitis, standard treatment includes aggressive fluid resuscitation, adequate analgesia, fasting (nil per os), and correction of the underlying cause, such as endoscopic retrograde cholangiopancreatography (ERCP) in gallstone-associated cases [4]. For TCM, therapy is largely supportive and parallels the management of acute heart failure, typically involving beta-blockers, angiotensin-converting enzyme inhibitors, and diuretics. Inotropes should be avoided in patients with left ventricular outflow tract obstruction, and anticoagulation may be warranted if left ventricular thrombus formation is detected [2]. Although no specific guidelines exist for TCM precipitated by acute pancreatitis, optimal care centers on supportive management of both conditions while avoiding unnecessary acute coronary syndrome-directed interventions. This case, "When the Heart Sinks,"

underscores a rare but critical link between pancreas and heart. TCM usually affects older women after emotional stress, but here it followed pancreatitis – an abdominal catastrophe. Misdiagnosis is likely when patients do not fit the typical profile [6]. Clinicians should consider TCM in any patient with acute stress (including visceral illness) who develops new cardiac findings. Early recognition prevents inappropriate treatment and improves outcomes [1].

2. Case Presentation

2.1 Patient's history and Physical Examination

This case report delineates the presentation of a 62-year-old female who presented to our emergency department with an abrupt onset of severe epigastric pain radiating posteriorly toward the back, accompanied by progressive nausea and multiple episodes of non-bilious, non-projectile, non-bloody vomiting, occurring four times prior to evaluation. The pain was described as intense (9/10 in severity), constant in character, aggravated by oral intake, and unresponsive to antacids or positional changes. She also reported transient episodes of rapid, regular palpitations and mild exertional dyspnea but denied associated symptoms such as chest pain, diarrhea, constipation, jaundice, or urinary complaints. Her medical history was notable for well-controlled hypertension managed with amlodipine 5 mg daily and dyslipidemia treated with atorvastatin 20 mg; there was no prior history of pancreatitis, diabetes mellitus, or cardiovascular disease. Family history was non-contributory, and the patient denied tobacco use, alcohol consumption, or illicit drug use. On physical examination, vital signs revealed a blood pressure of 140/85 mmHg, heart rate of 110 beats per minute, respiratory rate of 22 breaths per minute, temperature of 37.2°C, and oxygen saturation of 96% on ambient air. Cardiovascular examination demonstrated a normal jugular venous pulse with no murmurs, gallops, or rubs, though mild tachycardia was evident. Pulmonary auscultation revealed equal bilateral air entry without adventitious sounds. Abdominal examination was significant for epigastric tenderness with mild guarding but without rebound tenderness, organomegaly, or palpable masses. Examination of the lower extremities showed no peripheral edema.

2.2 Investigations

The pertinent laboratory investigations, summarized in Table 1, were consistent with a diagnosis of acute pancreatitis, demonstrating elevated pancreatic enzymes alongside supportive inflammatory markers. In light of the clinical suspicion, an abdominal ultrasonography was performed, which revealed the presence of gallbladder sludge and multiple cholelithiasis, along with mild pancreatic enlargement; however, there were no sonographic features indicative of acute cholecystitis, such as gallbladder wall thickening or pericholecystic fluid. To further delineate the extent of pancreatic involvement and rule out complications, a contrast-enhanced computed tomography (CT) scan of the abdomen was subsequently obtained. The CT findings confirmed interstitial edematous pancreatitis without evidence of pancreatic necrosis, but with mild peripancreatic fluid collections suggestive of early local inflammation. Concurrently, a 12-lead electrocardiogram (ECG) was performed at the bedside due to the patient's reported palpitations and elevated heart rate, which revealed sinus tachycardia without any ischemic changes or ST-T segment abnormalities. Given the unexpected rise in serum troponin levels in a patient with low pretest probability for obstructive coronary artery disease (CAD), a transthoracic echocardiogram was undertaken to assess cardiac function and exclude structural abnormalities. Echocardiographic evaluation demonstrated classical findings supportive of Takotsubo cardiomyopathy, including apical ballooning, hypercontractility of basal segments, and a left ventricular ejection fraction (LVEF) reduced to approximately 35%, in the absence of valvular pathology or pericardial effusion. The constellation of an identifiable acute physical stressor (acute pancreatitis), modest troponin elevation, characteristic echocardiographic morphology, and lack of obstructive CAD collectively fulfilled the modified diagnostic criteria for Takotsubo cardiomyopathy.

Test	Result	Normal Range
Hemoglobin	14.4	12-16 g\dL
WBC	$13.2x10^9$	$4.0\text{-}11x10^9$\L
Platelets	$230x10^9$	$150\text{-}450x10^9$\L
Sodium	136	135-145 mmol\L
Potassium	4.2	3.5-5.0 mmol\L
Creatinine	0.9	0.6-1.2 mg\dL
ALT	58	<40 U\L
AST	60	<40 U\L
Troponins	1.8	<0.04 ng\mL
BNP	220	<100 pg\mL
CRP	65	<5 mg\L
Serum Lipase	1250	0-160 U\L
Serum Amylase	820 U\L	23-85 U\L

Table 1: results of relevant laboratory investigations.

2.3 Management course

Initial stabilization focused on hemodynamic and respiratory support, beginning with the administration of supplemental oxygen via nasal cannula to maintain oxygen saturation above 94%, alongside close observation for any signs of acute pulmonary

edema secondary to left ventricular systolic dysfunction. Continuous electrocardiographic monitoring was instituted to detect arrhythmias or evolving ischemic changes. Despite the presence of sinus tachycardia (heart rate of 110 bpm), vasopressor support was not required, as the patient's blood pressure remained stable at 140/85 mmHg. Analgesia for severe epigastric pain was provided with intravenous morphine at a dose of 4 mg, carefully titrated to effect. Concurrently, intravenous isotonic crystalloids were administered cautiously—initially at a bolus rate of 5 mL/kg over the first hour—guided by frequent reassessment of vital signs and echocardiographic evaluation of left ventricular filling status, to avoid volume overload and exacerbation of cardiac dysfunction. In light of the reduced left ventricular ejection fraction (LVEF) of 35% attributed to Takotsubo cardiomyopathy, low-dose oral beta-blocker therapy (metoprolol 25 mg twice daily) was initiated and gradually uptitrated as tolerated, with the aim of attenuating sympathetic overactivity. Once hemodynamically appropriate, lisinopril 5 mg once daily was added to promote myocardial recovery and prevent adverse ventricular remodeling. Management of acute pancreatitis involved complete bowel rest (NPO status) to minimize pancreatic stimulation, ongoing fluid resuscitation, and close monitoring for potential electrolyte imbalances or systemic inflammatory complications. Antiemetic therapy with ondansetron 4 mg IV every 8 hours as needed was used to control nausea, with careful ECG monitoring due to the QT-prolonging potential of the drug. Daily laboratory surveillance included complete blood count (CBC), basic metabolic panel (BMP), liver function tests (LFTs), serum amylase and lipase, and C-reactive protein (CRP) to track disease progression and inflammatory status. Given the presence of gallstones and sludge on imaging, interval cholecystectomy was planned electively after clinical resolution, especially in the event of recurrent pancreatitis. As the patient remained hemodynamically stable without evidence of life-threatening arrhythmias, cardiogenic shock, or pulmonary edema, escalation to intensive care was deemed unnecessary. A repeat transthoracic echocardiogram performed four weeks later demonstrated marked improvement in systolic function, with normalization of LVEF to 60%. In light of the recovery in ventricular function, the ACE inhibitor was discontinued, while beta-blocker therapy was continued for an additional 3 to 6 months, with plans for tapering under close outpatient cardiology follow-up, contingent on clinical stability and absence of recurrent symptoms.

3. Discussion

Elevation of cardiac troponins in the context of acute pancreatitis presents a significant diagnostic challenge, as systemic inflammatory stress alone may lead to mild, transient increases in these biomarkers without true myocardial injury. This nonspecific elevation—typically less than 0.1–0.2 ng/mL and demonstrating a stable pattern—must be carefully distinguished from elevations suggestive of direct myocardial involvement. In Takotsubo cardiomyopathy (TCM), troponin levels, though generally modest (ranging from approximately 0.5 to 2.0 ng/mL), often exhibit a characteristic rise-and-fall pattern analogous to that seen in acute coronary syndromes (ACS), thus complicating differentiation. Importantly, B-type natriuretic peptide (BNP) levels tend to be disproportionately elevated in TCM compared to troponins, and a BNP-to-troponin ratio exceeding 2,500 has been shown to favor TCM over ACS with a reported sensitivity of 90% and specificity of 85% [2]. Imaging plays a pivotal role in the diagnostic workup; specifically, regional wall motion abnormalities on echocardiography that do not conform to a single coronary artery distribution are strongly suggestive of TCM. In cases where diagnostic ambiguity persists, coronary angiography remains the definitive modality for exclusion of obstructive coronary artery disease, although in the present case, angiography was not deemed necessary given the patient's low cardiovascular risk profile—marked by absence of classical anginal symptoms, minimal atherosclerotic risk factors, and an unremarkable cardiac history [2]. Fluid management in patients with concurrent acute pancreatitis and stress cardiomyopathy necessitates a carefully individualized approach. While standard pancreatitis guidelines advocate for aggressive intravenous hydration, such protocols may be detrimental in patients with impaired left ventricular function, heightening the risk of volume overload, pulmonary edema, and adverse outcomes. A growing body of evidence warns against unmonitored fluid resuscitation in this subset of patients. A 2021 study involving 210 patients revealed that fluid overload was associated with a twofold increase in the incidence of respiratory failure (OR 2.1; 95% CI, 1.4–3.2) [3]. A more nuanced and practical resuscitation strategy involves serial bedside echocardiography to assess left ventricular filling pressures and dynamic inferior vena cava (IVC) diameter variation as surrogates for volume status, thereby guiding judicious administration of 5–10 mL/kg fluid boluses followed by reassessment within 30–60 minutes [3]. Fixed-rate fluid infusions without real-time hemodynamic assessment should be strictly avoided in such clinical scenarios [2,3]. QT interval prolongation is another significant consideration in TCM, occurring in approximately 40% of patients, often peaking within 48 hours of symptom onset. This is particularly relevant when antiemetics such as ondansetron are administered, given their known propensity to exacerbate QT prolongation. Therefore, continuous cardiac telemetry is essential, ideally for 5–7 days, and should be accompanied by vigilant monitoring and correction of serum potassium and magnesium levels to mitigate the risk of torsades de pointes and other malignant arrhythmias [3]. Furthermore, the pathognomonic apical akinesia in TCM predisposes to mural thrombus formation due to regional stasis—an effect potentiated by the systemic pro-inflammatory state observed in acute pancreatitis. Anticoagulation should be considered after individual risk stratification, particularly when the ejection fraction (EF) falls below 30%, even in the absence of echocardiographic evidence of thrombus. Prophylactic anticoagulation is increasingly recognized as essential in such settings [7]. Monitoring C-reactive protein (CRP) levels may also provide prognostic value, as CRP concentrations exceeding 50 mg/L have been linked with a 15% increase in complications. The inflammatory cascade involving cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) not only elevates myocardial oxygen demand but

also contributes to coronary microvascular dysfunction, potentially triggering coronary vasospasm and exacerbating myocardial injury [7]. A left ventricular ejection fraction below 35% is a well-established marker of poor prognosis in TCM, associated with a four- to fivefold increase in the likelihood of cardiogenic shock and a rise in mortality from approximately 2% to 18%, thus serving as one of the criteria for ICU admission [2]. Additional ICU indicators include sustained ventricular arrhythmias, hemodynamic instability requiring vasopressors or inotropes, and evidence of multiorgan dysfunction. Beyond baseline echocardiographic evaluation, repeat echocardiography within 48–72 hours is essential to identify early deterioration, complications such as thrombus formation, or progressive ventricular impairment. A follow-up study at 2–4 weeks is recommended to confirm recovery, which is achieved in over 50% of patients. Persistent LV dysfunction beyond three months should prompt reassessment for underlying ischemic heart disease or other cardiomyopathies. Although beta-blockers are routinely employed in hemodynamically stable patients to mitigate sympathetic overactivity and reduce the risk of arrhythmias, their long-term benefit remains uncertain. Current evidence does not demonstrate a definitive mortality advantage with chronic beta-blocker therapy in TCM, and no randomized controlled trials to date have validated their extended use. Therefore, a pragmatic approach would be to continue beta-blocker therapy for approximately 3 to 6 months, followed by reevaluation contingent on symptomatic recovery and echocardiographic normalization [3]. Regarding the surgical management of gallstone-related pancreatitis, elective cholecystectomy remains the standard of care to prevent recurrence. However, in the setting of Takotsubo cardiomyopathy with a persistently reduced EF (<35%), the procedure should be deferred until adequate myocardial recovery is achieved, unless urgent intervention is warranted—for instance, in cases complicated by acute cholangitis. In such high-risk scenarios, surgery must proceed under intensive preoperative assessment and intraoperative hemodynamic monitoring to minimize perioperative risk [6].

4. Conclusion

Severe acute pancreatitis necessitates a high index of suspicion for concurrent cardiac involvement, particularly in the presence of unexplained electrocardiographic abnormalities or elevations in cardiac biomarkers such as troponin. This case underscores a critical clinical insight: even subtle cardiopulmonary symptoms—such as mild dyspnea or transient palpitations—may herald the onset of significant myocardial dysfunction, including stress-induced cardiomyopathy. The utility of bedside transthoracic echocardiography, combined with vigilant hemodynamic monitoring, is indispensable in navigating the therapeutic tension between the need for aggressive fluid resuscitation in pancreatitis and the risk of exacerbating left ventricular impairment in the context of Takotsubo cardiomyopathy. While the majority of patients ultimately experience full functional recovery, acute stabilization does not represent the totality of care. Continued cardiac surveillance remains essential, as delayed deterioration or recurrence may occur in a subset of patients. This case exemplifies the emerging recognition of systemic inflammatory states—such as pancreatitis—as potent triggers for stress cardiomyopathy, highlighting both a gap in current management guidelines and the pressing need for heightened clinical awareness in such intersecting pathologies.

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