

Takotsubo Cardiomyopathy Triggered by Acute Pancreatitis: A Case Report

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Abstract

Takotsubo cardiomyopathy is a heart condition that is widely known to be caused by stress. It presents with symptoms that are similar to a myocardial infarction even though the coronary arteries are clear. This case report details the clinical characteristics, diagnostic assessment, and management plan of a 55-year-old male patient with a past medical history of alcoholism who arrived at the emergency department with the typical symptoms of acute pancreatitis. The case demonstrates the progression of Takotsubo cardiomyopathy, which was triggered by acute pancreatitis in the context of alcoholism, and underlines the significance of early detection and management to enhance the patient's outcomes.

Keywords

Takotsubo Cardiomyopathy, Acute Pancreatitis, Alcoholism, Epigastric Pain

1. Introduction

Takotsubo cardiomyopathy (TCM), sometimes known as broken heart syndrome or stress-induced cardiomyopathy, is an uncommon condition accounting for about 2.2% of cardiomyopathies [1]. Emotional and physical stress is one of the known triggers of this cardiomyopathy. However, some reports showed that acute pancreatitis could be a trigger for TCM, although uncommon [2].

Acute pancreatitis is an inflammation of the pancreas. It ranges in severity from mild symptoms, which resolve with minimal intervention, to severe ones that can lead to life-threatening complications [3]. Pain, nausea, and vomiting are common symptoms of acute pancreatitis. The patient usually has one or more of the following symptoms: sudden, severe upper abdominal pain, nausea,

and vomiting; profound shock with cyanotic mottled skin; widespread tenderness; minimal to severe rigidity of the abdomen; and ecchymosis or bluish discoloration in either or both flanks or around the umbilicus.

TCM's specific mechanism remains yet to be discovered, however, studies suggest one of the following: catecholamine-induced neurogenic cardiotoxicity, coronary microvascular damage, and multi-vessel epicardial coronary artery vasospasm [4]. It has also been suggested that acute pancreatitis can cause inflammation that affects the central nervous system and releases stress hormones like catecholamines, which may contribute to the development of TCM [5].

Here, we present the case of a middle-aged man who presented with acute pancreatitis and shortly after it, he developed TCM.

2. Case Presentation

A 55-year-old male was brought to the emergency department due to constant, excruciating epigastric pain that radiates to his back and was slightly alleviated by leaning forward. The patient also complained of generalized weakness, fatigue, and a dry mouth. On physical examination, the patient appeared in visible acute distress, was profusely diaphoretic, and was notably malnourished. His vital signs showed tachycardia HR—110 - 120 bpm, sub febrile temperature T-37.5. Blood pressure, oxygen saturation was otherwise within normal range. The skin was pale, cool, and clammy, with decreased turgor. Chest auscultation revealed bilaterally diminished vesicular breath sounds; cardiovascular examination showed regular rate and rhythm, palpable peripheral pulses, and no jugular venous distention. Upon palpation, the abdomen was tender to touch, but no rebound tenderness or guarding was demonstrated; there was no palpable hepatosplenomegaly, and Murphy's sign was also negative.

Laboratory investigations were notable for elevated serum lipase levels 6171.1 U/L, suggesting pancreatic inflammation. There was a decreased platelet count, elevated C-reactive protein, an elevated total bilirubin level, and markedly elevated GGT. Notably, even though AST, ALT, and ALP were also elevated, their elevation was not as pronounced as it was the case for GGT. ABG analysis demonstrated normal pH with low pCO₂ and low bicarbonate concentration, which in the setting of an elevated lactate level pointed to respiratory compensation.

Abdominal ultrasound demonstrated a hyperechogenic pancreas with a normal gallbladder, ruling out biliary etiology of acute pancreatitis. There was mild hepatomegaly with hyperechogenic, macrovascular changes in liver structure, attributed to long-standing alcohol consumption. There was no ileus or fluid in the abdominal cavity.

The abdominal X-ray was unremarkable. Surgical consultation excluded acute abdomen. Based on the patient's laboratory and imaging studies, a diagnosis of acute pancreatitis was made. Considering classical clinical presentation and strongly positive laboratory results, we refrained from doing a contrast-enhanced CT scan.

The Ranson score was calculated which on admission was 2 (1 point for age > 55 years, 1 point for LDH > 350), confirming that severe pancreatitis was unlikely in this patient.

The patient was promptly admitted to the hospital's internal medicine department and placed on aggressive intravenous fluid resuscitation to correct dehydration and maintain hemodynamic stability. Pain management with analgesics and antiemetics was initiated. Given the patient's history of alcoholism, a multidisciplinary approach was undertaken to address alcohol cessation. Nutritional support was provided, and the patient was kept nil per os (NPO) to allow the pancreas to rest.

After several days of aggressive rehydration measures, the patients' symptoms including abdominal pain and discomfort improved, but he still stayed tachycardic with heart rate ranging from 110 to 120 bpm. This led to a follow-up ECG that showed inverted T waves in the V1 - V6 leads (**Figure 1**). Echocardiography was notable for slight left ventricular hypertrophy with moderate systolic dysfunction in LAD territory with an ejection fraction of 35%. Besides low grade (I/IV) mitral and tricuspid insufficiency, no significant changes were visualized in aortic valve, Valsalva sinus, ascending aorta, aortic arch or abdominal aorta. Systolic pressure in the pulmonary artery is within the normal range. Pericardial sac: without abnormalities. Cardiac biomarkers were ordered, which demonstrated elevated levels of troponin I (53.01 pg/mL).

Based on cardiac enzyme measurements and echocardiography (**Figure 2**), two possible diagnoses were outlined: on the one hand, atherosclerotic coronary disease was a possibility based on observed ECG and echocardiographic changes; conversely, Takotsubo cardiomyopathy was likely due to radiological changes.

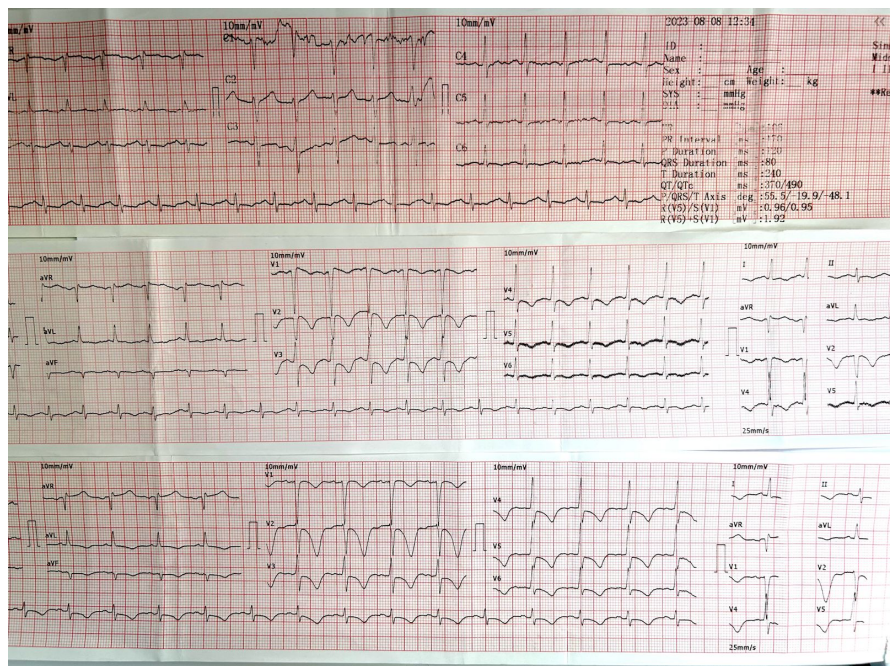


Figure 1. Inverted T waves in V leads.

As ECG changes were persistently observed in dynamic and the patient stayed tachycardic within the HR range of 110 - 120, coronary angiography was deemed necessary to exclude acute coronary syndrome and/or confirm the diagnosis of TCM.

Coronarography ruled out any coronary stenosis (**Figure 3**), so atherosclerotic



Figure 2. Echography showing the dilated apex in the right ventricle (white arrows).

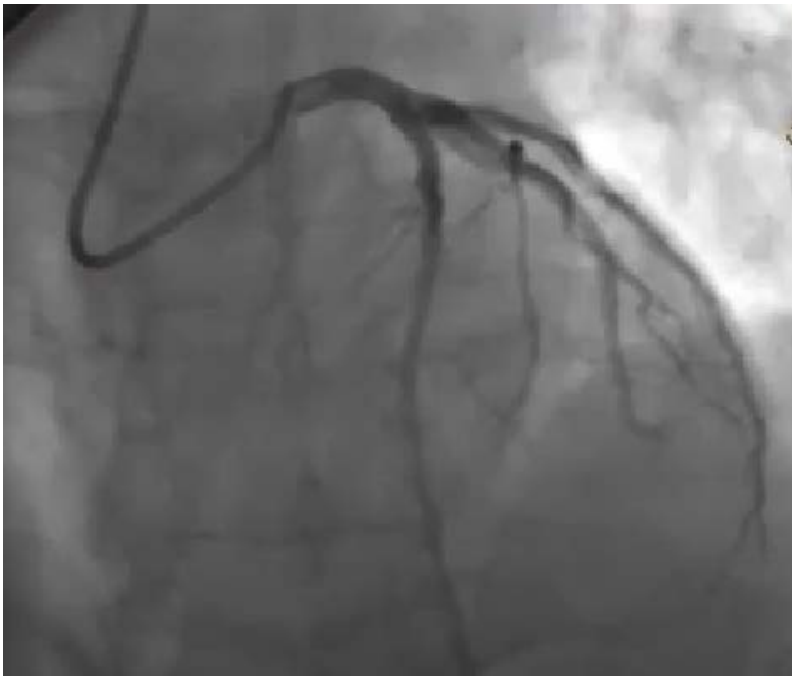


Figure 3. No stenosis was found during coronarography.

multivessel coronary artery disease was ruled out. Consequently, a diagnosis of TCM was made.

Following cardiorespiratory monitoring, aggressive crystalloid resuscitation, correction of electrolyte abnormalities, and supportive management, the patient's subjective condition improved; he no longer complained of pain in the epigastric area, profuse diaphoresis, or nausea and vomiting. Objectively, vital signs returned to normal values (HR 75 - 80 RRR, BP 105 - 60 mm Hg, T-36.5 C). Follow-up laboratory evaluation demonstrated a positive dynamic with platelet count ($184 \times 10^3/\mu\text{L}$) and lipase (96.7 U/L) returned to normal values. VBG was unremarkable, Troponin I normalized.

Based on subjective and objective findings, the patient was discharged home with thorough instructions and recommendations and a treatment plan aiming to control his heart rate and to maintain electrolyte balance. The patient was advised on the importance of regular follow-up with a primary care physician and cardiologist, adequate hydration daily, and alcohol cessation. The patient was also informed about the high likelihood of relapse and complications unless he followed the given instructions.

3. Discussion

Takotsubo cardiomyopathy, sometimes called stress-induced cardiomyopathy, was first described in Japan in the early nineties as an acute transient reversible left ventricular dysfunction with apical distention giving it the takotsubo-or octopus fishing trap-appearance. Emotional and physical stressors are known triggers for this condition, hence the name "Broken Heart Syndrome" [6]. The exact pathology is still unknown; however, it is believed to be related to catecholamine release. An intense stress (emotional or physical) activates the sympathetic nervous system and lead to a catecholamine surge. Interestingly, one identifiable source of this catecholamine surge is acute pancreatitis. Acute pancreatitis not only induces a systematic inflammation response, but also is a significant physical stressor which triggers the release of catecholamines. This, in turn, can either have a direct toxic effect on the cardiac myocytes, or lead to an indirect damage by causing multivessel spasm in predisposed individuals [7]. It has been proposed that regional differences in catecholamine-induced vasoconstriction, innervation, and adrenergic sensitivity might be the cause of the mechanism of the regional difference in contractility [8]. Multivessel spasms caused by this dysfunction, can be clinically similar to acute coronary syndrome [9]. Approximately 2% of all patients presenting to hospitals with suspected acute coronary syndromes have been identified as having TCM, with the vast majority being women aged ≥ 50 years. If only women are considered, up to 10% of patients with suspected acute coronary syndromes are ultimately diagnosed as having TCM [10]. To the best of our knowledge, acute pancreatitis as a trigger for TCM has been documented only 15 times [2], in addition to our case. Among these cases, only 5 of them (including our case) were male, making the majority of them female. This is consistent with what was found in the literature that pro-

poses that TCM is more common among post-menopausal women, most probably because of the protective effect that estrogen has on the myocardial cells in younger women.

An important factor that should be considered when discussing TCM, especially when it's triggered by acute pancreatitis is alcohol. It is well known that alcohol abuse is one of the most common etiologies for acute pancreatitis, because it increases the viscosity of the pancreatic secretions as well as precipitation, leading to formation of protein plugs in the small pancreatic ducts which eventually will lead to inflammation and fibrosis in the pancreas [11]. On the other hand, alcohol have a toxic effect on mitochondria and sarcoplasmic reticulum, and ethanol have a toxic effect on cardiac myocytes, which all can translate to a decrease in myocardial contractility, arrhythmias, and non-ischemic dilated cardiomyopathies [12]. In addition, chronic heavy alcohol consumption causes apoptosis and necrosis of the myocytes. With limited regeneration, reduced myofibrillar proteins and altered myosin isoforms result in weakened contractions. Over time, these changes lead to increased left ventricular dilatation, mass, and dysfunction [13]. Our case represents the 5th documented case in the literature of a TCM triggered by acute pancreatitis among the male population, with the majority being linked to alcohol-induced pancreatitis [2], thus underlining the distinctive susceptibility of the male population to this rare but major cardiac complication. The absence of the protective effect of estrogen among the male population, as well as the higher degree of sympathetic activation, likely contributes to a higher catecholamine surge in response to stress. Given this association, it is crucial to recognize the potential cardiac implications in patients with acute pancreatitis, especially in alcoholic patients. Therefore, we recommend high caution in this population and taking TCM into consideration among the differential diagnosis, with taking into consideration the importance of close cardiac monitoring to insure a timely intervention and better results.

It is difficult to clinically diagnose TCM when it appears with acute pancreatitis. This is mostly due to overlap of symptoms between these conditions, especially the epigastric pain that might mask the chest pain and shift the attention away from the heart. Since it is usually overlooked, the cardiomyopathy symptoms start to appear later, usually as shortness of breath and chest pain. In our case, the diagnosis of TCM was made before the development of severe cardiac complaints because of the constant cardiac monitoring that was provided to the patient, thus highlighting the importance of continuous monitoring for the patients presenting with pancreatitis to start an early management plan. Another diagnostic challenge lies in the similarities of the ECG and biochemical changes between TCM and acute coronary syndrome. This has led to several cases of TCM to be treated as acute coronary syndrome with emergent reperfusion and thrombolysis, thus predisposing the patient to more risk of bleeding and strokes, and delaying cardiac catheterization [14] [15]. In our case, the early coronary angiography performed has removed any doubt of having an acute coronary syndrome, thus once again emphasizing the importance of close cardiac moni-

toring and the timely intervention.

4. Conclusion

This case presents a rare, yet clinically significant association between acute pancreatitis and TCM, contributing to the limited literature about this uncommon trigger for TCM. It also sheds the light on the importance of a multidisciplinary approach when dealing with a complex medical problem, especially in alcoholics. The timely recognition amid the overlapping symptoms between acute pancreatitis and TCM, and acute coronary syndrome highlights the importance of vigilant monitoring to set an early management plan.

Informed Consent

An informed consent was obtained from the patient to publish this article.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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