

## Acute Pancreatitis-Induced Takotsubo Cardiomyopathy in an African American Male

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### Abstract

Takotsubo cardiomyopathy (TCM) is triggered by multiple physical and psychological stressors and frequently mimics acute coronary syndrome. Acute pancreatitis as a trigger for TCM has rarely been reported. We report a 55-year-old African American man with hypertension and alcohol abuse history, who presented with epigastric and sub-sternal pain and electrocardiogram demonstrating ischemic changes. Laboratory parameters revealed elevated troponin-I, amylase, lipase, and metabolic acidosis. He was diagnosed with acute pancreatitis and ACS. Coronary angiogram was unrevealing for coronary atherosclerosis and he was managed conservatively for acute pancreatitis and heart failure from TCM.

### Introduction

Takotsubo cardiomyopathy (TCM), also known as left ventricular (LV) apical ballooning, broken heart syndrome, and stress cardiomyopathy, is so named due to its resemblance of a Japanese octopus trap, a tako tsubo.<sup>1</sup> Typically reported triggers include emotional stress, pheochromocytoma, and sub-arachnoid hemorrhage.<sup>2-4</sup> Multiple physical stressors have been documented as triggers; however, there is only sporadic mention in literature about the role of acute pancreatitis.

### Case Report

A 55-year-old African American man with a history of alcohol abuse and hypertension was admitted to the hospital with severe, 10/10, non-radiating pain in the epigastrium and left lower sternal border. This was associated with diaphoresis, nausea, and non-bloody vomiting. He reported a 2-week alcohol binge until the morning of admission. The patient was afebrile with stable hemodynamics, and cardiac examination revealed a regular rhythm with a soft S3. Respiratory examination was unremarkable and abdomen was diffusely tender with involuntary guarding.

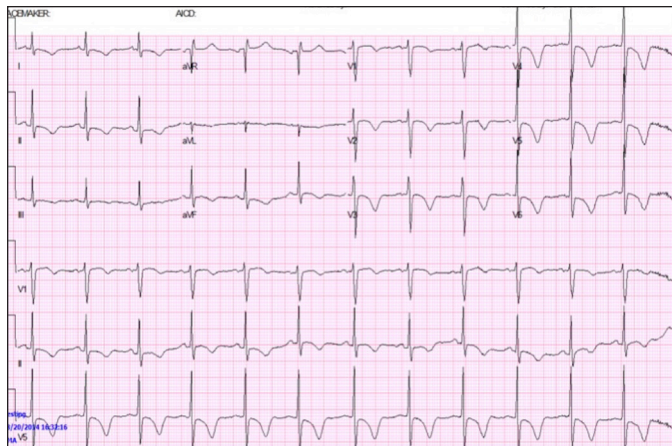
The initial cardiac biomarkers troponin-I and N-terminal pro-beta natriuretic polypeptide were elevated at 0.29 ng/mL and 4420 pg/mL, respectively. Admission laboratory parameters revealed elevated serum lipase and amylase at 773 IU/L and 97 IU/L, respectively, and metabolic profile demonstrated elevated anion-gap metabolic acidosis with elevated serum lactate and ketones. Serial troponin-I was trended with peak level of 0.658 ng/mL. Electrocardiogram (EKG) showed normal sinus rhythm with generalized ST-T segment depression and T-wave inversions (Figure 1). Chest radiography and hepatobiliary ultrasound were negative for any acute pathology. Transthoracic echocardiogram showed reduced LV ejection fraction (30–35%) with dilated and dyskinetic apex. Multiple hypokinetic LV segments in mid- and apical-anterior wall, apical lateral wall, mid- and apical-septum,

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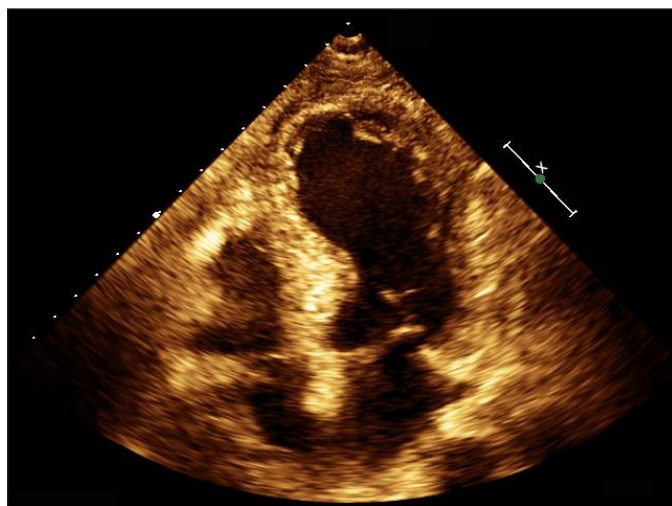
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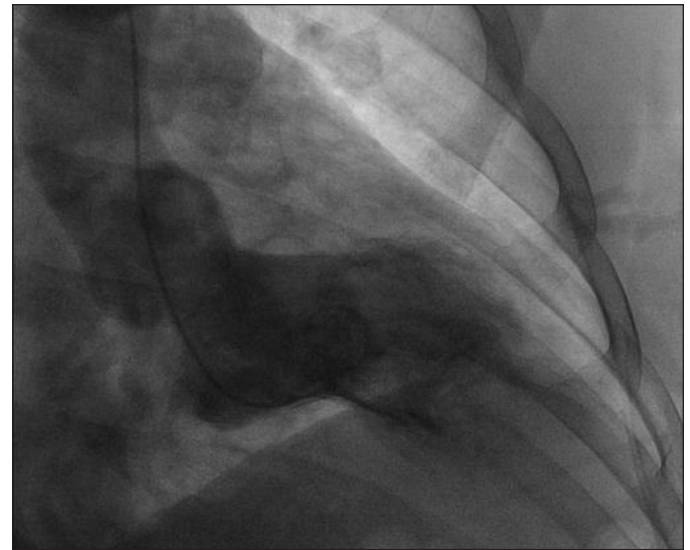
**Figure 1.** EKG on admission demonstrating normal sinus rhythm with ST-T segment depression and T-wave inversions in anterior, inferior, and lateral leads.

and apical inferolateral walls with compensatory basal hyperkinesis were noted (Figure 2). Abdominal computed tomography (CT) revealed peripancreatic infiltration and edema without identifiable pancreatic necrosis or drainable fluid collections.

The patient was admitted to the coronary care unit and started on medical management with aspirin, atorvastatin, metoprolol, clopidogrel, and heparin. He was started on intravenous hydration, analgesics, and anti-emetics for conservative management of acute pancreatitis. Emergent coronary angiogram demonstrated absence of obstructive coronary artery disease and left ventriculography demonstrated reduced LV ejection fraction (25%) with apical ballooning and hyper-contractile basal segments, consistent with TCM (Figure 3). The patient was medically stabilized on aspirin, metoprolol, lisinopril, and warfarin. He was counselled extensively on tobacco and alcohol cessation.



**Figure 2.** Transthoracic echocardiogram demonstrating apical ballooning and hypercontractile base.



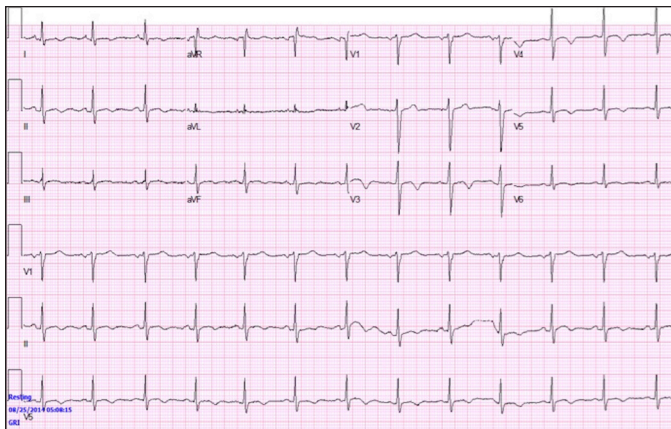
**Figure 3.** Left ventriculography demonstrating reduced LV ejection fraction and apical ballooning consistent with TCM.

Over the next 2-3 days, his pancreatitis improved and he was able to tolerate an oral diet with minimal pain and regular bowel movements. EKG at discharge showed normalization of ST-T segment depression and improving T-wave inversions (Figure 4). A repeat echocardiogram 3 weeks later demonstrated complete normalization of LV function highlighting the transient nature of this disease, essential for diagnosis (Figure 5).<sup>5</sup> His anticoagulation was discontinued and he continues to be asymptomatic on follow-up.

## Discussion

First described in Japan in 1990, TCM constitutes 2% of acute coronary syndrome cases and is usually preceded by emotional or physical stress.<sup>6,7</sup> Postulated pathogenesis includes coronary microvascular dysfunction, coronary spasm, catecholamine-induced myocardial stunning, reperfusion injury, microinfarction, and abnormal fatty acid metabolism.<sup>1</sup> Currently, the most widely accepted hypotheses are catecholamine-toxicity and microvascular dysfunction. The Mayo Criteria is the gold standard for diagnosis, defined as: 1) transient hypokinesis, akinesis, or dyskinesis of the LV mid-segments with/without apical involvement; 2) absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture; 3) new EKG abnormalities (ST-T segment elevation or T-wave inversions) or modest elevation in troponin-I; and 4) absence of pheochromocytoma or myocarditis.<sup>5</sup>

Traditionally, TCM is more common in post-menopausal women, and about 90% of diagnosed patients are women aged 61-76 years.<sup>8,9</sup> This phenomenon is hypothesized to be due to estrogen deficiency in the later years of life. Estrogen is cardioprotective through down-regulation of hypothalamic-



**Figure 4.** EKG at discharge demonstrating normal sinus rhythm with normalization of the ST-T segment depression and resolving T-wave inversions.

adrenal axis and stimulation of atrial natriuretic peptide and shock-protein-70. Additionally, the role of cardiac glucose uptake in these patients has been suggested. These patients typically exhibit a decreased myocardial glucose uptake, an effect that persists after the resolution of the acute illness.<sup>10</sup> In addition to these postulated mechanisms, the role of genetic mutations is similar to those responsible for fragile-X syndrome and cardiovascular disease, increasing the prevalence in females. All these factors strongly suggest a female preponderance of TCM and, consistent with the same, the 5 reported cases of pancreatitis-induced TCM are women older than 40 years.<sup>2,4,9,11,12</sup>

Alcoholic pancreatitis was first implicated as a physical stressor for ‘inverted’ TCM in 2006.<sup>3</sup> Acute pancreatitis is a significant physical stressor and has been postulated to precipitate TCM by various pathophysiological mechanisms. Pancreatitis causes exaggerated sympathetic stimulation,



**Figure 5.** Transthoracic echocardiogram 3 weeks after discharge demonstrating normal LV ejection fraction with no wall motion abnormalities.

which is closely associated with TCM.<sup>3,4,12</sup> Patients with pre-disposition to TCM have high endogenous levels of catecholamines and differential distribution of adrenergic receptors on the myocardial surface leading to involvement of discrete myocardial segments.<sup>3</sup> This hypothesis is further strengthened by highlighting the role of other sympathetic overdrive states such as pheochromocytoma and subarachnoid haemorrhage in the causation of TCM.<sup>3,4,9,12</sup> Additionally, the distributive shock that develops with severe acute pancreatitis can cause transient myocardial dysfunction and microvascular hypoperfusion. This phenomenon has been well-described as the causative mechanism of acute decompensated heart failure in septic shock, but its role in acute pancreatitis is not well elucidated.<sup>13</sup> However, both these mechanisms are commonly postulated pathophysiological principles of TCM.<sup>1</sup> Additionally, severe acute pancreatitis has been known to cause an endocrine insufficiency over months, and whether this insulin-deficiency in combination with the glucose-dependent mechanisms of the human heart predispose TCM merits further investigation.<sup>14</sup>

Our case highlights the rare association of acute pancreatitis with TCM. Ours is the second reported case in an African American, and the first reported case in a man.<sup>15,16</sup> Further clinical studies are required to broaden the scope of our knowledge on this issue and to frame guidelines to standardize the care of these patients.

## Disclosures

Author contributions: JD Bruenjes and CJ Vacek reviewed the literature and drafted the manuscript. S. Vallabhajosyula reviewed the literature, wrote and edited the manuscript, and is the article guarantor. JE Fixley critically revised the manuscript and supervised the manuscript creation.

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