

Acute Pancreatitis Associated with Elevated Troponin Levels: Whether to Thrombolysate or Not?

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Abstract

The presentation of retrosternal chest pain with normal electrocardiogram (ECG) during chest pain followed by initial presentation of acute pancreatitis can lead to a dilemma in managing such a patient, and whether to thrombolysate such a patient is a real controversy. We hereby present a similar case who was diagnosed to be having acute pancreatitis on admission, on clinical grounds, confirmed by laboratory investigations and ultrasonography, who subsequently developed retrosternal chest pain with normal ECG during the chest pain. All further serial ECGs after the onset of chest pain were within normal limits. The troponin-I level of this patient was positive twice (done 16 h apart). The patient was treated as acute coronary syndrome along with the standard management protocol of acute pancreatitis.

Keywords: Chest pain, Pancreatitis, Troponin-I

Introduction

Pancreatitis is commonly observed in alcoholics, but the evidence of the cardiac variety of chest pain along with positive troponin-I and no significant electrocardiogram (ECG) changes in the setting of pancreatitis can complicate the management of both the diseases, and the decision to thrombolysate the patient becomes debatable. To the best of our knowledge, there are no such reported cases in the medical literature of acute pancreatitis with non ST-segment elevation myocardial infarction. The most common cause of elevated troponin levels in the absence of acute coronary syndrome (ACS) is the myocyte-necrosis, mainly due to mismatch between oxygen supply and demand due to abnormal loading of the right or left heart (chronic or acute).^[1,2] Troponin also increases with tachycardia, after strenuous exercise, catecholamine release or infusion, autonomic nervous system imbalance and in conditions affecting, directly, the membrane permeability.^[1] Other conditions causing troponin release in the absence of ACS include direct damage to the cardiomyocytes by inflammation, infection, toxins, chest contusion, infiltration or electricity.^[1] Here, we will discuss the management of such a patient presenting with pancreatitis

with elevated troponin levels and its outcome. Our case firstly illustrates the unusual forms of coronary disease represented by the non-atherosclerotic myocardial injury. Secondly, it shows that the visceral damage associated with acute pancreatitis can mimic non ST-segment myocardial infarction along with elevated troponin levels, which complicates the treatment and prognosis.

Case History

A 30-year-old male patient came to Krishna Hospital with chief complaints of pain in the abdomen since 3 days prior to admission, which was of the generalized, dull-aching type, intermittent in nature and non-radiating, and was getting relieved by squatting. He also had four episodes of non-projectile vomiting at night before coming to the hospital, and the vomitus contained food particles. He started complaining of retrosternal chest pain 15 min after admission, which was of the constricting type, continuous in nature and non-radiating, and subsided within 30 min. The chest pain was associated with sweating. There was no history of breathlessness, giddiness, palpitation or syncope. There was no other history of any major medical illness in the past. He had no addictions. He was newly diagnosed to be having diabetes mellitus on admission. On examination, his pulse rate was 80/min, blood pressure was 110/80 mmHg and respiratory rate was 14 cycles/min. His laboratory reports were as follows:

Hb – 15G%; TLC – 18,500; platelet count – 3 lac;
PT (INR) – 1.3; RBSL – 250 mg/dL; blood urea – 40;

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serum creatinine – 1.1 mg/dL; urine amylase > 1000 IU/L; serum amylase – 317 IU/L (normal value 25-125); CPK MB – 38 IU/L (normal value 0-25); serum lipase – 420 (normal value 73-393); Troponin-I – positive (0.54 mcg/L); Electrocardiogram – within normal limits; X-ray chest PA view – within normal limits; liver function tests – within normal limits; serum calcium – 7.4 mg/dL; and serum phosphorus – 5.3 mg/dL.

The lipid profile was as follows → total cholesterol: 251 mg/dL (normal value 140-250 mg/dL); serum triglycerides – 421 mg/dL (normal value 60-165 mg/dL); serum VLDL – 84 mg/dL (normal value 17-30 mg/dL); serum LDL – 117 mg/dL (normal value 80-160 mg/dL); serum HDL – 50 mg/dL (normal value 30-80 mg/dL); and glycosylated hemoglobin – 7.5%.

The 2D echocardiography results were within normal limits, with left ventricular ejection fraction 68%.

Ultrasonography of the abdomen and pelvis [Figure 1] - was suggestive of bulky pancreas.

Computed tomography (CT) of the abdomen was suggestive of mild infiltrative pancreatitis Balthazar Grade CTSI (CT Severity Index) = B.

The patient was started on standard protocol for ACS, and was managed conservatively for acute pancreatitis. The patient gradually became totally asymptomatic. He was discharged after 7 days of admission in a hemodynamically stable condition after performing the stress test, which was negative. The patient was advised to get the coronary angiogram done from a higher center (due to lack of facilities in our hospital) on follow-up, which turned out to be normal.

Discussion

Cardiac troponins are specific to the heart muscle only, and can reliably detect heart muscle injury;^[3] however, a peak in



Figure 1: Ultrasonography of the abdomen showing bulky pancreas

their levels is noticed as early as 8 h post-myocardial injury. Hence, these were assayed 8 h after the onset of chest pain. The presentation of acute pancreatitis with ECG changes has previously been reported.^[4,5] Troponin elevation is indicative of myocardial cell injury, which may be due to ischemia, inflammation, trauma, tachycardia, after strenuous exercise, catecholamine release or infusion, autonomic nervous system imbalance and in conditions directly affecting membrane permeability, toxins, chest contusion, electricity infiltrative diseases, systemic infection or renal failure. The mechanism of such a troponin release in pancreatitis is unknown. Because pancreatic enzymes are known to travel along tissue planes and cause tissue destruction and pseudocyst formation at sites distant to the pancreas, one theory is that a similar movement across the diaphragm may be responsible for direct myocardial injury. Another possibility, as seen in experimental animal models, is that pancreatic enzymes may have entered the bloodstream and produced myocardial injury. Other possibilities include coronary vasospasm or changes in myocardial cell membrane permeability by inflammatory mediators secondary to acute pancreatitis. Other possibilities also include catecholamine-induced microvascular dysfunction due to pancreatitis resulting in myocardial stunning.^[6]

Complications of acute pancreatitis involving pseudo or true myocardial infarction are very rare.^[7] ECG changes mimicking acute myocardial infarction in patients with acute pancreatitis have been documented before now.^[7] But, to the best of our knowledge, the occurrence of non ST-segment elevation myocardial infarction in the presence of acute pancreatitis, as described by us, has never been reported in the medical literature till date. Irrational thrombolysis of such a patient of pancreatitis with elevated troponin levels may result in disastrous outcome. Main *et al.* reported a 47-year-old male with alcohol-related acute pancreatitis who died of severe retroperitoneal hematoma apparently related to thrombolytic therapy.^[8] Thus, a standard management protocol needs to be defined for managing such patients and intensive interventions be considered in such complicated cases and, when in doubt, confirm the findings with coronary angiogram for further treatment, which, unfortunately, we were unable to do during the patient's stay in our hospital due to the lack of facilities.

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