A Broken Heart: A Woman with Chest Pain and an Abnormal ECG
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From Case Records of the Alpert Medical School of Brown University Residency in Emergency Medicine

DR. COURTENEY MACKUEN: Today’s patient is a 56-year-old woman who presents with complaint of mid-sternal and left-sided chest discomfort beginning after several episodes of nausea and non-bloody emesis. She had been drinking alcohol the evening prior to presentation. She became concerned when she developed persistent, penetrating chest pain after vomiting. She did not describe a tearing sensation. She presented to the emergency department via ambulance 4 hours after the onset of pain. She received an aspirin and a sub-lingual nitroglycerin tablet without relief from the Emergency Service personnel.

Her history is significant for hypertension and an episode of chest pain two years ago with resultant hospitalization. She stated that she had an elevated troponin but minimal coronary artery disease (CAD) on catheterization. She was told she had a “broken heart.” She denied history of deep venous thrombosis (DVT) or pulmonary embolism (PE). She is a non-smoker. She reported a great deal of stress in her employment. Her only medication is venlafaxine hydrochloride.

DR. ANDREW NATHANSON: Can you describe her physical exam?

DR. MACKUEN: The patient appeared uncomfortable. Her brachial artery blood pressures were equal – 144/88 mm Hg. Her pulse was 96 and regular, respiratory rate was 16, and her room air oxygen saturation was 98%. Her neck was supple, with no bruits; her lungs were clear bilaterally, and her chest wall had no crepitus. She had a normal s1s2, no murmurs, a regular rhythm, and distal pulses were equal bilaterally. Her abdomen was soft, non-tender and without rebound or pulsatile masses. Her neurologic exam was normal.

DR. DAVID CURLEY: What were your initial concerns? How did you limit your differential?

DR. WILLIAM BINDER: Chest pain is the 2nd most common presentation to the emergency department, causing 6–8 million visits in the US annually. In our patient, the most concerning and life-threatening causes of chest pain were acute coronary syndrome, thoracic aortic dissection, pulmonary embolism, pneumothorax, and an esophageal rupture.

We obtained labs, including a cbc, chem 7, and troponin, all of which were normal. A chest radiograph was unremarkable and did not show mediastinal widening or air. A bedside echocardiogram performed by emergency physicians was negative for a pericardial effusion. An electrocardiogram demonstrated inverted and deep t waves in leads V2–V4 and a biphasic t wave in V5 (see figure 1).

In creating a differential diagnosis, esophageal rupture was certainly a possibility in this case. While about 75% of cases in the United States are iatrogenic or due to trauma, spontaneous rupture is seen in approximately 15% of cases. Pain is the most common finding in an esophageal perforation (90%). Subcutaneous emphysema can be palpated in 60% of

Figure 1. Note the deeply inverted T waves in V2–V4, as well as inversion in V5.
patients with a cervical esophageal rupture, whereas 30% of patients with a thoracic esophageal injury will have palpable crepitus. Mackler’s triad – emesis, chest pain, and subcutaneous emphysema – is seen in only 20–25% of cases. While initial radiography is normal in up to 33% of cases, within several hours an abnormal chest radiograph is noted in over 90%. Pneumothorax and pulmonary embolism seem less likely in this patient.

An acute aortic dissection was of significant concern. A type A dissection is highly lethal with mortality approaching 1–2% per hour within the first 48 hours of the event. Data from the International Registry for Acute Aortic Dissection (IRAD) revealed that approximately 66% of cases are seen in males [mean age is 63]. Women have a higher mean age at presentation. Data from IRAD suggested that pain was not tearing or ripping but sudden and sharp in 90% of patients, while in 4.5% of patients there was no report of pain. Pulse deficits are intermittently seen (20%), and aortic insufficiency murmurs are noted in 40–50% of patients. Chest radiography is abnormal in up to 90% of cases of a proximal dissection, with mediastinal widening [63%] and pleural effusion [19%] the most common abnormalities. Our patient had a normal chest radiograph, no pulse deficits or abnormal blood pressures, and she did not have a murmur of aortic insufficiency. However, none of these findings completely excluded an aortic dissection or spontaneous esophageal rupture [Boerhaave’s syndrome].

Acute Coronary Syndrome (ACS) remains in the differential. The patient had been hospitalized previously and had a catheterization revealing minimal coronary artery disease but with a positive troponin, suggesting a non-thrombotic origin to her elevated cardiac biomarker. Additionally, her current ECG is abnormal.

**DR. CATHERINE PETTIT:** What are the causes of troponin elevation in patients without CAD?

**DR. MACKUEN:** There are numerous processes causing myocardial necrosis and troponinemia. ECG abnormalities are frequently seen in these cases but are not a direct result of thrombus within the coronary vessels. Acute aortic dissection, ischemic stroke, intracerebral hemorrhage all can lead to elevated troponin. Cardiac inflammatory states – endocarditis, myocarditis, and myopericarditis – as well as infiltrative states such as amyloid and sarcoid also result in troponin leaks. High cardiac demand states including SVT, atrial fibrillation with rapid ventricular response, pulmonary embolism, and sepsis can lead to a Type II non-ST elevation myocardial infarction [NSTEMI], or demand ischemia. Other causes of non-thrombotic acute coronary syndrome include coronary artery vasospasm and Takotsubo cardiomyopathy. Coronary artery vasospasm can cause transient symptoms of ischemia and can be provoked by stimulant drugs such as cocaine or other amphetamines. Takotsubo cardiomyopathy, the “broken heart” syndrome, is an important cause of non-thrombotic acute coronary syndrome. Given that our patient mentioned a “broken heart,” this is high on our differential today.

**DR. THOMAS HARONIAN:** What is the cause of Takotsubo cardiomyopathy and what are the complications?

**DR. MACKUEN:** Takotsubo cardiomyopathy is a reversible condition that can mimic acute coronary syndrome in the absence of coronary artery disease. This cardiomyopathy was named Takotsubo because on echocardiogram it resembled a Japanese octopus trap with a large bottom and narrow top. It classically presents as chest pain in a post-menopausal woman after an emotional or physical stressor. Between 80–90% of cases of Takotsubo cardiomyopathy occur in woman [mean age 58–75]. In 66% of patients the disorder is preceded by an emotional or physical stressor. Takotsubo appears to have a circadian predilection for the early hours of the day and summer months. Electrocardiographic changes such as ST elevation [68%], and T wave inversions [97%] are the most common findings. Troponin elevation is noted in 85% of patients, and are usually mild and rapidly normalize. Its prevalence in ACS is noted to be between 0.7%–2.5%, and in woman may be as high as 6%. Etiology has not been elucidated. One hypothesis suggests that increasing circulating catecholamines cause transient epicardial spasm. Other theories speculate about microvascular dysfunction and cardiac fatty acid metabolism. Takotsubo is a diagnosis of exclusion and standard treatment for ACS is followed. Complications include heart failure, and ACE inhibitors have been used in the management of this disorder. Mortality is low (3%) and complete resolution occurs in 1–8 weeks. The 4-year recurrence rate is 5–10%.

**DR. JESSICA SMITH:** What was the clinical course for the patient?

**DR. MACKUEN:** In the ED the patient received sublingual nitroglycerin and morphine without relief. She received hydromorphone and lorazepam and her pain abated. Interestingly, sedative use has been previously reported to be effective in Takotsubo cardiomyopathy. CT of the chest with intravenous contrast was negative for both dissection and esophageal rupture. The patient was admitted to the CCU. Her second troponin 6 hours later was elevated at 5.67 ng/ml. A subsequent troponin was 1.91 ng/ml. Cardiac catheterization revealed minimal luminal irregularities in her coronary arteries, mild hypokinesis in the apical portion of the heart, and an EF approximately 48%. She was discharged to home and has done well.

**FINAL DIAGNOSIS:** Takotsubo’s cardiomyopathy

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**DR. MACKUEN:** What was the clinical course for the patient?
References


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