

Chest Pain in an 18-Year-Old Man

ECG CHALLENGE

An 18-year-old man presented to our hospital reporting sudden onset right-sided chest pain followed by dyspnea and syncope. On arrival of the emergency medical services, the patient was awake and in respiratory distress. He was afebrile with a blood pressure 95/66 mmHg, heart rate 121 beats/min, respiratory rate 38 breaths/min, and oxygen saturation of 88% improved to 98% on 4-L oxygen supply. Heart sounds were normal with no significant murmur. Pulses were intact bilaterally. Lung examination revealed rapid, shallow breathing with decreased air entry. ECG is shown in Figure 1. iSTAT Troponin-I was 0.39 ng/mL (normal <0.08 ng/mL). What is the most appropriate next step?

- A. Emergent cardiac catheterization
- B. Portable chest x-ray
- C. Thrombolytic therapy
- D. Contrast esophagogram

Please turn the page to read the diagnosis.

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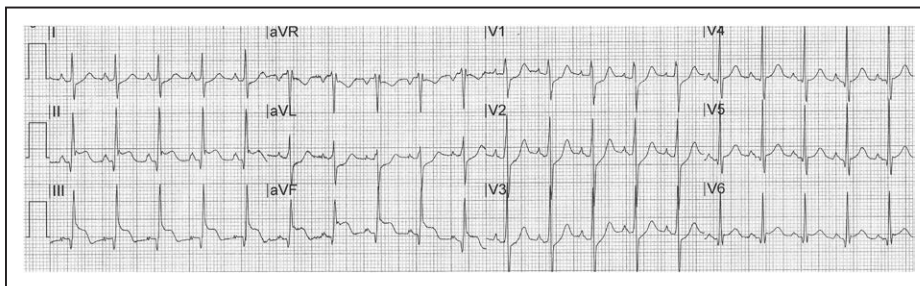


Figure 1. ECG showing sinus tachycardia with ST-segment elevation in leads II, III, aVF and reciprocal ST-segment depression in I, aVL.

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 ■ ECG ■ myocardial infarction
 ■ pneumothorax ■ ST elevation
 ■ young age

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RESPONSE TO ECG CHALLENGE

The correct answer is B, portable chest x-ray. A portable chest x-ray revealed large right-sided pneumothorax with right lung collapse and leftward deviation of mediastinal structures (Figure 2). The patient underwent emergent needle decompression followed by chest tube placement resulting in expansion of the right lung (Figure 3) and hemodynamic improvement. Repeat ECG showed resolution of the ST-segment elevation (Figure 4). Echocardiography showed normal ejection fraction with normal wall motion and no pericardial effusion. Given his age and resolution of symptoms and ECG changes with normal echocardiography, we did not proceed with cardiac catheterization.

The initial ECG shows ST-segment elevation in leads II, III, aVF with reciprocal ST-segment depression in I, aVL, worrisome of inferior ST-segment–elevation myocardial infarction. Coronary atherosclerosis is rare in young, otherwise healthy patients.¹ Exclusion of other more common causes of chest pain in this age should be considered before rushing for cardiac catheterization or administration of thrombolytic therapy (choices A and C). A portable chest x-ray is a quick, good modality that helps establish the diagnosis. Clues suggestive of pneumothorax include sudden-onset right-sided chest pain followed by dyspnea, respiratory distress, decreased air entry, and syncope.

Although the exact mechanism is unclear, numerous theories are postulated regarding the reason for ST-segment elevation in our patient. One hypothesis is true ischemia caused by external compression of the right coronary artery by the collapsed lung. Another possibility is right ventricular strain as supported by evidence of myocardial injury with elevated troponin and the ECG findings of S1Q3T3 following resolution of the ST-segment elevation. External cardiac compression, by pneu-

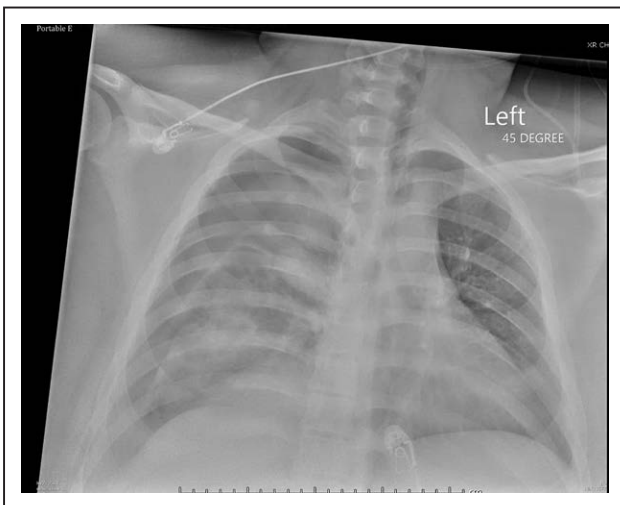


Figure 2. Portable chest x-ray showing large right-sided pneumothorax with right lung collapse and leftward deviation of mediastinal structures (note the tracheal deviation to the left side).

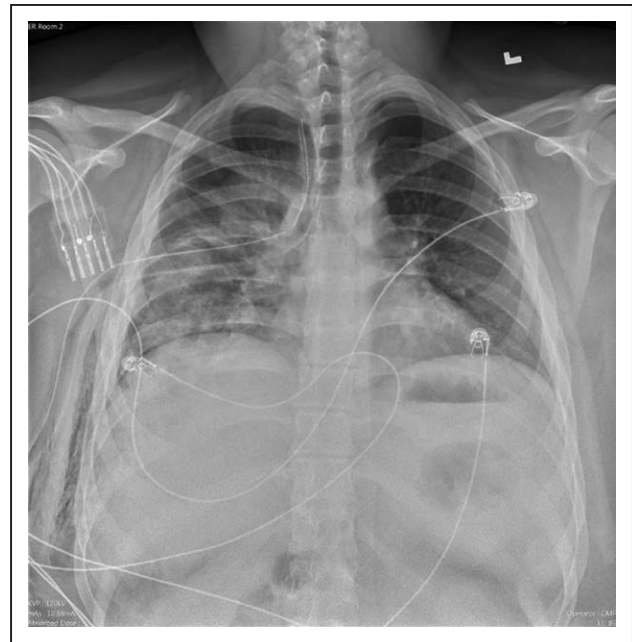


Figure 3. Chest x-ray showing right-sided chest tube placement with resolution of the right pneumothorax and mediastinal deviation.

mothorax¹ or hiatal hernia, causing ischemic ST-segment elevation has been previously documented in medical literature. Ischemia can also be triggered by cardiac displacement and rotation causing increasing pulmonary vascular resistance with acute right ventricular pressure overload,^{1,2} or perhaps even possible coronary arteries twisting with cardiac rotation causing ischemia, which is supported by the fact that the ECG generally shows deviation of the mean QRS vector. Hypotension and reduced venous return with decreased coronary blood flow and myocardial ischemia has also been described to cause ECG changes in pneumothorax.^{1,2}

The differential diagnosis of ST-segment elevation includes ST-segment–elevation myocardial infarction, coronary artery spasm, stress-induced cardiomyopathy, aortic dissection extending into the coronary arteries (mostly right coronary artery), acute pericarditis, J-point elevation, myocarditis, Brugada syndrome, pulmonary embolism, and bundle-branch blocks. In stress-induced cardiomyopathy, echocardiography typically shows apical ballooning with hyperdynamic base and reduced ejection fraction, which were absent in our patient. Patients with aortic dissection may have aortic regurgitation murmur with unequal pulse and blood pressure bilaterally. Chest x-ray may show wide mediastinum, and computed tomography angiography is diagnostic.³ Pulmonary embolism is a reasonable differential diagnosis; however, diagnosis confirmation by computed tomography angiography is essential before administration of thrombolytic therapy (choice C).³ Rupture esophagus is mostly iatrogenic. Although contrast esophagogram (choice D) confirms the diagnosis, it is usually done following chest and neck radiography.³

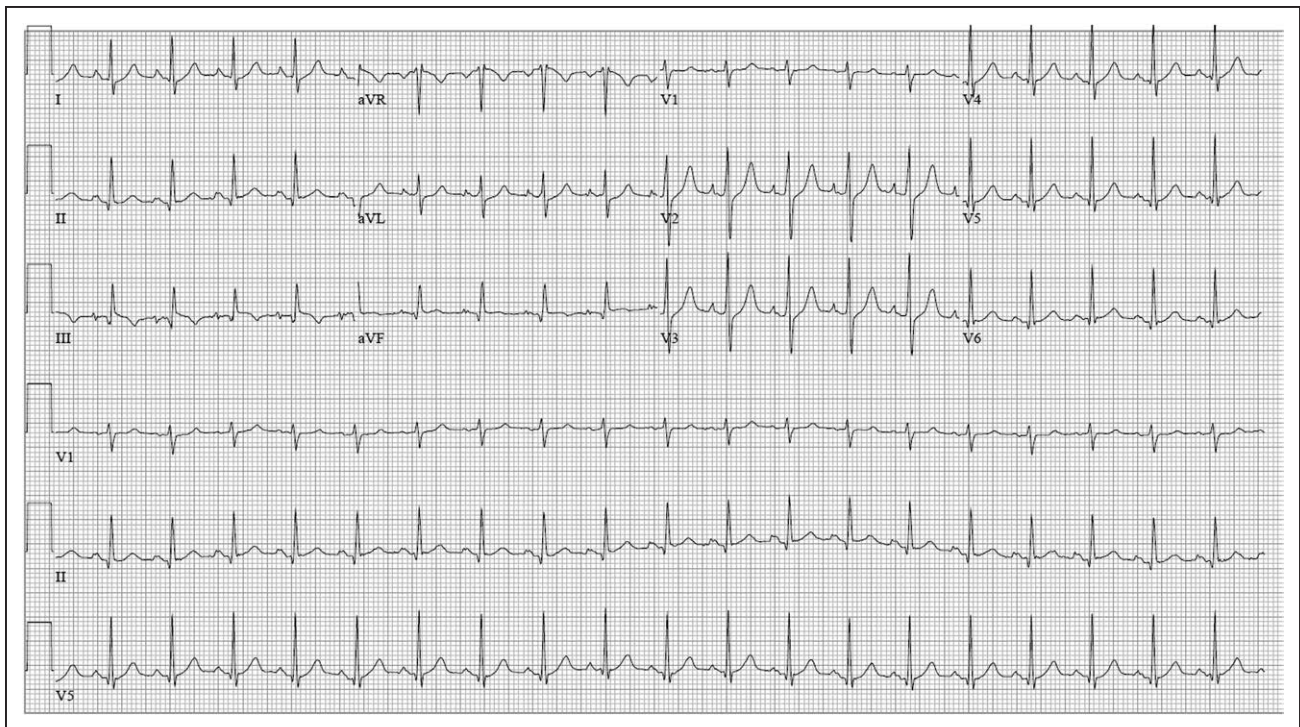


Figure 4. ECG showing sinus tachycardia with resolution of the inferior ST-segment elevation. Also note the presence of the S1Q3T3 pattern suggestive of right ventricular strain.

In conclusion, we emphasize that tension pneumothorax may present with ST-segment elevation and ECG changes should be interpreted in relation to the clinical presentation.

DISCLOSURES

None.

AFFILIATION

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FOOTNOTES

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