

Pulmonary Embolism Mimicking Anteroseptal Acute Myocardial Infarction

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Pulmonary embolism (PE) is a potentially lethal condition that presents in patients with chest pain or shortness of breath. Although electrocardiograms (ECGs) typically demonstrate abnormalities associated with PE, ST-segment elevation, which can indicate anteroseptal acute myocardial infarction (AMI), has—on rare occasions—been noted on ECGs of patients with acute PE. The current report documents the case of a 57-year-old man who presented to the emergency department with chest pain. Findings from an ECG suggested anteroseptal AMI; however, cardiac catheterization indicated that the patient did not have critical ischemic heart disease. On further examination, the patient was found to have a massive bilateral PE. The present report emphasizes that physicians must investigate PE in all patients presenting with chest pain, dyspnea, or both, even in the face of ECG changes that are suggestive of a cardiac etiology. A brief discussion of the current theories of ST-segment elevation in the setting of PE is also included.

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Acute pulmonary embolism (PE), common in patients presenting with chest pain and dyspnea, can be lethal, particularly if the condition is not diagnosed. Electrocardiograms (ECGs) are typically used to diagnose PE in patients. Sinus tachycardia, complete or incomplete right bundle-branch block, the S1Q3T3 pattern (prominence of the S wave in lead I, Q wave in lead III, and T wave in lead III), and ST-segment depression in the precordial leads are among the most common ECG findings characteristic of this condition (Figure 1).¹ On rare occasions, ST-segment elevation, which can indicate anteroseptal acute myocardial infarction (AMI), is associated with acute PE.²⁻⁴

The present report describes a man who demonstrated dramatic and dynamic ST-segment elevation suggestive of

- S1Q3 or S1Q3T3 pattern
- Right QRS-axis deviation
- Complete or incomplete right bundle-branch block
- T-wave inversions in the right precordial leads
- Sinus tachycardia
- ST-segment depression in the right precordial leads
- Atrial dysrhythmias
- First-degree atrioventricular block
- "P pulmonale" pattern
- QR pattern in V₁
- Displacement of the transition zone to the left

Figure 1. Typical electrocardiogram changes associated with pulmonary embolism. This figure was adapted from *Electrocardiography in Clinical Practice: Adult and Pediatric*.¹ Copyright Elsevier, 2008. **Abbreviations:** S1Q3, prominence of the S wave in lead I and the Q wave in lead III; S1Q3T3, prominence of the S wave in lead I, Q wave in lead III, and T wave in lead III.

anteroseptal AMI. However, diagnostic testing revealed a large bilateral PE. In addition, we review the changes in ECG results that are typically associated with PE and summarize the theory of ST-segment elevation in such cases.

Report of Case

A 57-year-old man presented to the emergency department after he had a syncopal episode while at home alone. The patient stated that he had no prodrome and was unaware as to how long he was unconscious. He described substantial midsternal chest pain and shortness of breath on awakening. Although he had a history of coronary artery disease, the chest discomfort differed from his typical angina. The patient stated that, in the weeks before presenting to the emergency department, he had increasing fatigue as well as episodic chest pain and shortness of breath unrelated to physical activity.

The patient's medical history was significant for coronary artery disease, hyperlipidemia, hypertension, depression, and renal insufficiency. The patient had three intracoronary stents placed more than 3 years earlier: two in the left anterior descending artery and one in the right coronary artery. The patient had no known history of myocardial infarction, cardiomyopathy, arrhythmia, or central nervous system disease. He had a 6-year history of minimal tobacco use (approximately 1 pack per year), admitted to occasional alcohol con-

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■ Serum Electrolytes

- Bicarbonate, 21 mEq/L
- Calcium, 9.8 mg/dL
- Chloride, 97 mEq/L
- Creatinine, 1.6 mg/dL
- Glucose, 147 mg/dL
- Magnesium, 1.9 mEq/L
- Phosphorus, 4.3 mEq/L
- Potassium, 5.6 mEq/L
- Sodium, 138 mEq/L

■ Complete Blood Cell Count

- Hematocrit, 39%
- Hemoglobin, 14.2 g/dL
- Platelets, $158 \times 10^3/\mu\text{L}$
- Red blood cell, $4.16 \times 10^6/\mu\text{L}$
- White blood cell, $6400/\mu\text{L}$

Figure 2. Laboratory test results of a 57-year-old man presenting with chest pain suggestive of antero-septal acute myocardial infarction.

sumption, and denied illegal drug use. His prescribed medications included clopidogrel bisulfate, 75 mg/d, for coronary artery disease; metoprolol tartrate, 50 mg twice a day, for hypertension; isosorbide mononitrate, 60 mg/d, for angina; lansoprazole, 30 mg/d, for heart burn; amitriptyline hydrochloride, 25 mg at bedtime, and citalopram hydrobromide, 20 mg/d, for depression; inhaled beclomethasone dipropionate, 42 μg twice a day, as a prophylaxis for asthma; and aspirin, 325 mg/d, for cardiac protection.

On examination, the patient's blood pressure was 123/80 mm Hg in the supine and orthostatic positions; oxygen saturation, 96%; and heart rate, 111 beats per minute and regular. He appeared to be well nourished. Although he was in mild respiratory distress, the patient was able to complete full sentences. His trachea was midline and his lung fields were clear. No jugular venous distension was noted. Cardiac examination revealed tachycardia and a grade 1 or 2 early-peaking systolic

murmur at the lower left sternal border without an audible third heart sound and no parasternal heave. Findings from the abdominal examination were unremarkable. The patient's extremities were warm with normal pulses and without edema.

Laboratory data revealed near-baseline serum electrolyte levels and unremarkable complete blood cell count (Figure 2). The patient had a mildly elevated creatinine level of 1.6 mg/dL and a brain-type natriuretic peptide level of 21 pg/mL. Troponin I was normal (0.03 ng/mL). Results from the initial ECG demonstrated sinus tachycardia with first-degree block in the interval from atrial stimulus to succeeding ventricular stimulus, incomplete right bundle-branch block, and subtle ST-segment elevation in the antero-septal leads. Inverted T waves were noted in the right precordial and inferior leads (Figure 3). Chest X-ray findings were unremarkable.

The patient was admitted to the telemetry unit for further laboratory testing, including serial cardiac enzyme measurements. Intravenous heparin was administered as a 5000 U bolus dose followed by an infusion of 1000 U/hr. Partial thromboplastin time was measured after 1 hour, and the infusion was adjusted according to a previously established nomogram. Several hours later, the patient again experienced substantial midsternal chest discomfort (rated 8 on a 10-point scale, with 10 being "the worst pain imaginable") and shortness of breath similar to that prompting his admission. After initiating transdermal nitroglycerin therapy, the patient's blood pressure decreased from 110/70 mm Hg to 78/40 mm Hg. Nitroglycerin was removed, and intravenous fluids were administered. A second ECG revealed substantial ST-segment elevation and pathologic Q waves consistent with AMI (Figure 4). As a result, therapy for acute coronary syndrome was initiated and arrangements were made for emergency cardiac catheterization for suspected acute coronary occlusion.

Results from an angiogram, however, failed to reveal crit-

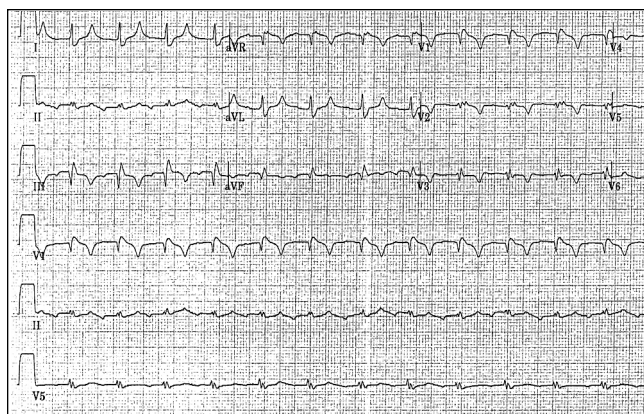


Figure 3. Initial electrocardiogram of a 57-year-old man shows sinus rhythm with incomplete right bundle-branch block and anterior and inferior T-wave inversions consistent with anterior and inferior myocardial ischemia. In the inferior leads, Q waves were also noted, suggesting age-indeterminate inferior myocardial infarction.

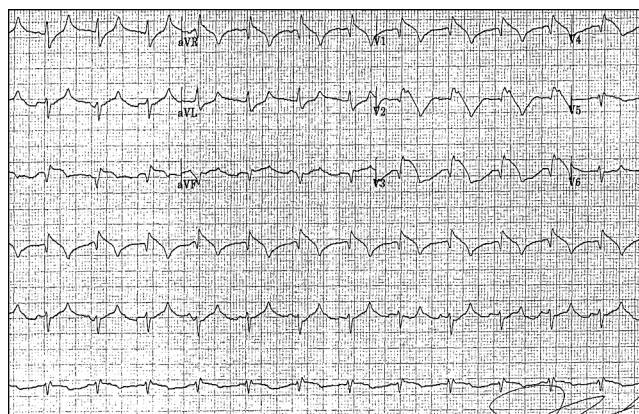


Figure 4. Second electrocardiogram (ECG) taken several hours after the initial ECG of a 57-year-old man demonstrates substantial ST-segment elevation and pathologic Q waves in the anterior and inferior leads consistent with antero-septal acute myocardial infarction.

CASE REPORT

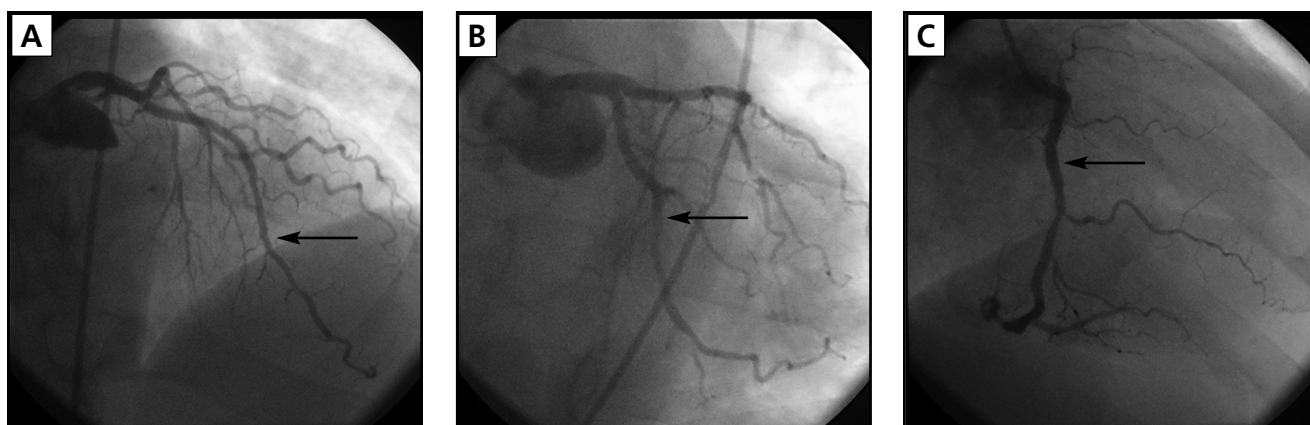


Figure 5. Cardiac catheterization images of a 57-year-old man suspected of having acute coronary occlusion. However, results revealed only (A) minor distal irregularities of the left anterior descending coronary artery (arrow); (B) moderate plaque in the circumflex artery (arrow) without substantial obstruction and no substantial left main coronary disease; and (C) moderate plaque in the right coronary artery (arrow).

ical obstructive coronary disease. In fact, all of the patient's stents were widely patent with only mild to moderate atherosclerotic disease in the left anterior descending coronary artery, circumflex artery, and right coronary artery (Figure 5). Although the ECG changes suggested compromised anterior circulation, the patient's response to nitroglycerin suggested right ventricular injury. However, the angiogram revealed a preserved vascular supply to the right ventricle.

After cardiac catheterization, transthoracic echocardiogram revealed dramatic right ventricular enlargement and displacement of the interventricular septum into the left ven-

tricle (Figure 6); however, left ventricular systolic function was normal. In addition, cardiac pressures on the right side were elevated, and right ventricular systolic function was mildly reduced. The patient did not demonstrate "McConnell's sign," which is described as ventricular free-wall hypokinesis with preservation of right ventricular apical function.⁵ McConnell's sign has been reported in instances of massive PE.⁵

As a result of the patient's renal insufficiency and contrast load from angiography, a ventilation-perfusion (V/Q) scan was promptly ordered instead of a computed tomography scan to evaluate for PE. The V/Q scan demonstrated large

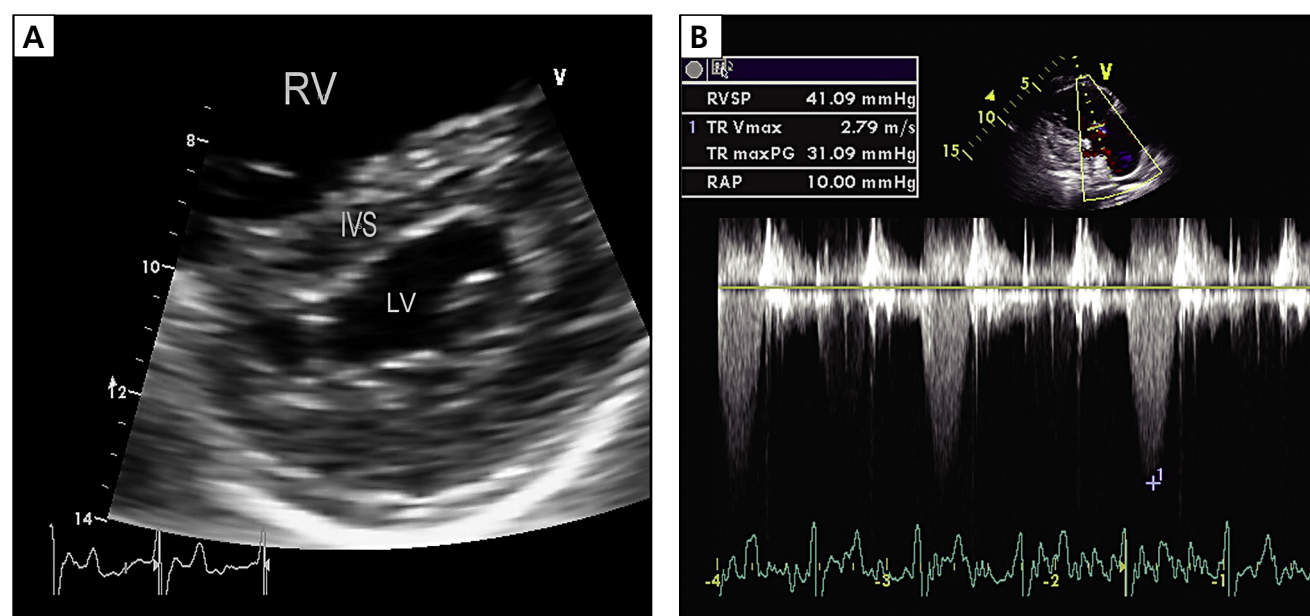


Figure 6. Echocardiographic images of a 57-year-old man after cardiac catheterization did not reveal acute coronary occlusion. Short-axis two-dimensional transthoracic echocardiogram (A) reveals an enlarged right ventricle (RV) and leftward displacement of the interventricular septum (IVS) during diastole. Compression of the left ventricle (LV) suggests elevated pulmonary pressures on the right side. Spectral Doppler echocardiogram of the tricuspid regurgitant jet (B) reveals elevated right ventricular systolic pressure (41 mm Hg).

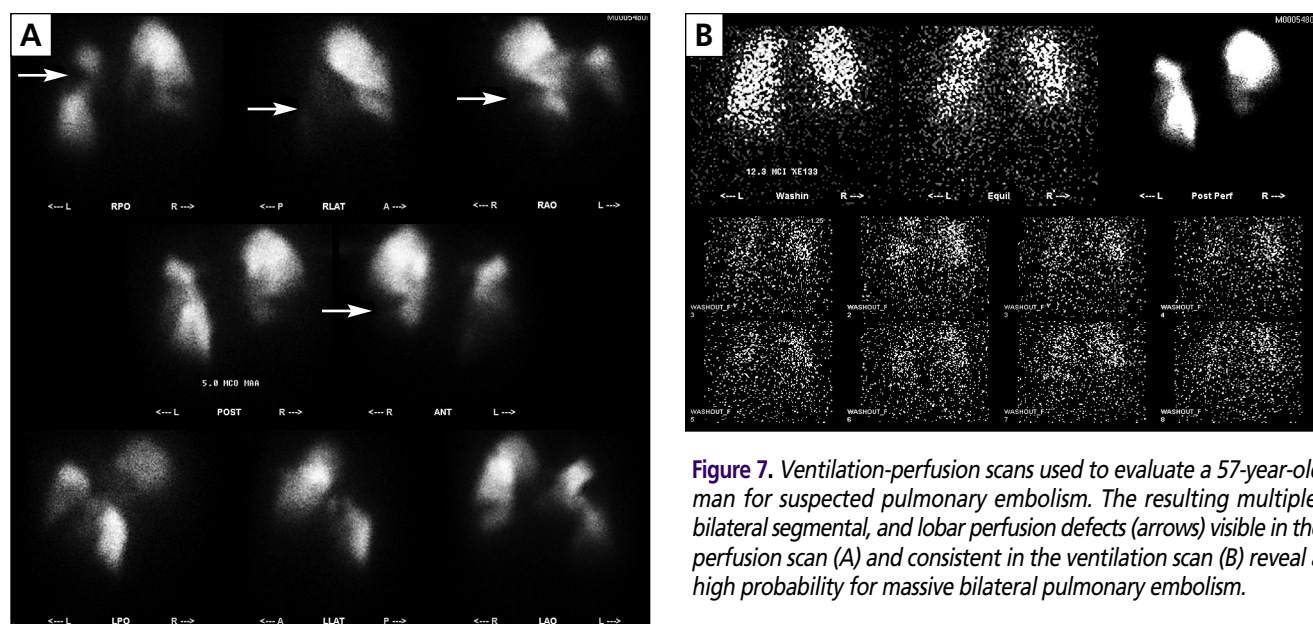


Figure 7. Ventilation-perfusion scans used to evaluate a 57-year-old man for suspected pulmonary embolism. The resulting multiple, bilateral segmental, and lobar perfusion defects (arrows) visible in the perfusion scan (A) and consistent in the ventilation scan (B) reveal a high probability for massive bilateral pulmonary embolism.

bilateral ventilation-perfusion mismatches highly suggestive of PE (Figure 7). The patient's troponin I level increased to a peak of 2.6 ng/mL, and creatine kinase-MB increased to 18.3 ng/mL.

After continuation of intravenous heparin therapy and hydration, the patient's condition stabilized. The remainder of the patient's hospital stay was unremarkable. Three days after presentation, a second echocardiogram revealed normalization of the right ventricular cavity size and a decrease in estimated

pulmonary artery pressures (Figure 8). Likewise, the anterior ST-segment changes and conduction defects resolved without the occurrence of pathologic Q waves. The right precordial T-wave inversions also improved (Figure 9). All cardiac enzyme markers normalized. Ten grams of coumadin was administered on the day before discharge, and 7.5 mg on the day of patient's discharge. Arrangements were made with the patient's internist for follow-up care.

(continued)

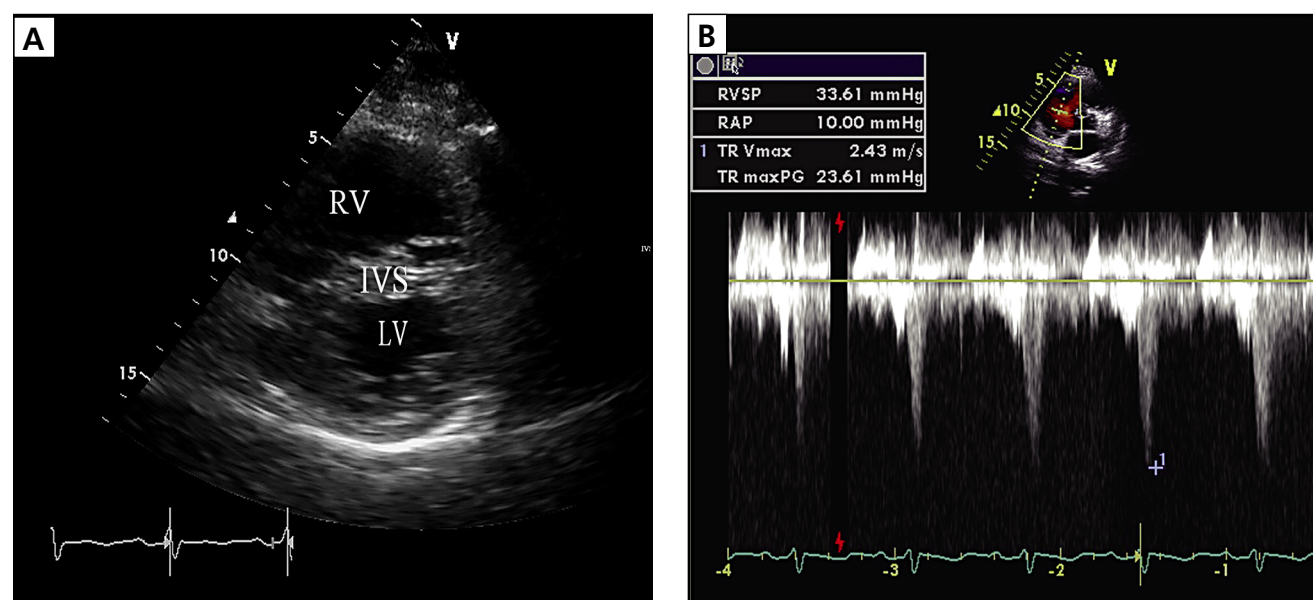


Figure 8. A second set of echocardiographic images from a 57-year-old man after treatment for pulmonary embolism. Short-axis two-dimensional transthoracic echocardiogram (A) shows improvement in the size of the right ventricle (RV) and a return of the left ventricle (LV) to a normal circular shape. Spectral Doppler echocardiography of the tricuspid regurgitant jet (B) reveals interval improvement in right ventricular systolic pressure (34 mm Hg). **Abbreviation:** IVS, interventricular septum.

CASE REPORT

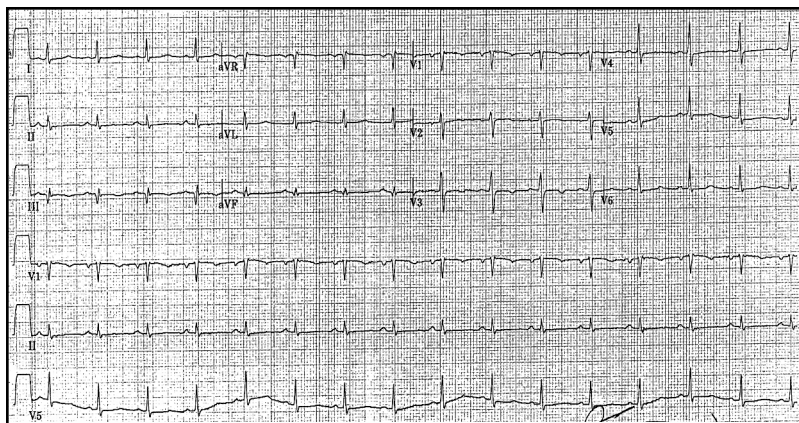


Figure 9. Follow-up electrocardiogram (ECG) taken 3 days after patient presentation to the emergency department demonstrates interval resolution of the anterior and inferior ST changes without Q waves. The incomplete right bundle-branch block noted in the initial ECG has also resolved.

Discussion

As stated previously, ST-segment elevation associated with PE is rare, and the direct relationship remains unclear. Anteroseptal ST-segment elevation was noted in one case² involving a 42-year-old woman with echocardiographic evidence of right ventricular pressure overload, which resolved after administration of thrombolytics (80 mg of tenecteplase). Another report³ described a 62-year-old man with substantial anteroseptal ST-segment elevation. Autopsy revealed that the patient had a massive PE without significant coronary artery atherosclerotic lesions. The distribution and contour of ST-segment elevation in each of these cases^{2,3} are similar to those in the present case. However, those two reports^{2,3} do not describe the patients' metabolic status—particularly potassium and pH levels—at the time of ECG.

The anteroseptal leads visible on ECGs are most likely to show ST-segment elevation. Paradoxical embolization of a venous clot across an atrial septal defect or across a patent foramen ovale into a coronary vessel represents one possible mechanism of ST-segment elevation in patients with deep venous thrombosis and PE.⁴ In such patients, coronary angiography will likely demonstrate a filling abnormality consistent with embolic debris. Furthermore, most ECG abnormalities associated with PE are thought to be a consequence of a sudden pressure load on a noncompensatory right ventricle.³ This additional strain may induce global or focal myocardial ischemia. Therefore, another potential theory suggests that ST-segment elevation in PE results from epicardial or microvascular coronary vasospasm induced by such strain.³ A third theory suggests that severe hypoxemia induces a catecholamine surge, which increases myocardial workload and results in ischemia.³

Several metabolic abnormalities, including hyperkalemia, have also been associated with ST-segment elevation evidenced on ECGs.⁶ Severe acidosis—specifically diabetic acidosis—has also been associated with ST-segment elevation, which is believed to be caused by potassium shifts into extracellular spaces.⁶ As described in one case report⁶ where substantial anteroseptal ST-segment elevation was present, the patient had a serum bicarbonate level of 4 mEq/L; pH, 7.06; and potassium, 8.9 mEq/L.

Elevation of the ST segment in patients with hyperkalemia typically slopes down.⁷ This characteristic can be helpful to physicians attempting to differentiate it from myocardial injury, in which the ST segment typically has a

plateau, shoulder, or upsloping elevation.⁷ Potassium levels, when associated with ST-segment elevation, are typically high and are joined by other ECG abnormalities (eg, peaked T waves, widening of the QRS complex, loss of atrial P waves).⁷ Although the mild increase in potassium in our patient was unlikely to result in such dramatic ST-segment elevation, the second ECG demonstrated downsloping elevation and narrow-based T waves, which are similar to changes documented in hyperkalemia.⁷ The serum potassium level in our patient at the time of the ECG was 5.5 mEq/L, lower than that seen in previous cases of ST-segment elevation.⁶ In addition the severity of metabolic acidosis in our patient (bicarbonate, 21 mEq/L) is less than that seen in other cases associated with ST elevation attributed to metabolic derangements.⁶

Troponin elevation in the setting of PE has a clinically significant effect on patient prognosis.⁸ The rapid and significant rise in right ventricular pressure with PE can lead to right ventricular myocardial ischemia and troponin release, which correlates with increased risk of mortality.⁸ In one prospective study⁹ of 56 patients with confirmed PE, elevated troponin T (≥ 0.1 $\mu\text{g/L}$) was associated with an increased risk of in-hospital death (odds ratio [OR], 29.6; 95% confidence interval [CI], 3.3-265.3) and was an independent predictor of 30-day mortality (OR, 15.2; 95% CI, 1.22-190.4). Although troponin I was measured in our patient rather than troponin T, it is plausible that troponin I elevation portends a poorer outcome than in patients without elevation of either cardiac biomarker.

The present report is particularly interesting for several reasons. When viewed retrospectively, the patient's initial ECG demonstrates several potential signs of PE. Specifically, the subtle S1Q3T3 pattern, incomplete right bundle-branch block, and nonspecific anterior T-wave inversions were potential signs of PE. The patient also had several risk factors for PE, including chronic tobacco use, chronic coronary artery disease, and hypertension.¹⁰ Although PE was an early consideration, initial evaluation and clinical management focused on a cardiac etiology for the patient's symptoms because of his history of coronary problems. Furthermore, though the dramatic anteroseptal ST-segment elevation in our patient likely

represented true myocardial injury given the increase in cardiac enzymes, angiography did not reveal critical coronary lesions, making paradoxical embolization unlikely.

One potential explanation for the ECG findings in the present report is intense coronary vasospasm superimposed on nonobstructive atherosclerosis in the anterior circulation—which together could result in myocardial injury and cause the ECG changes noted. The fact that the ECG abnormalities resolved concomitantly with a reduction in right ventricular pressures and chamber dimensions implies a potential link between right ventricular dynamics and the anterior ECG findings as demonstrated by the patient's response to nitroglycerin. The right ventricle is dependent on adequate venous return and preload to maintain forward flow against the elevated pulmonary pressures. In the present report, as a result of nitroglycerin, venous dilation reduced preload and caused hemodynamic deterioration.

Because most patients with acute coronary syndromes are treated with early heparin therapy—either low molecular weight or unfractionated—patients would theoretically be treated for nonlethal PE as well as for emergent coronary conditions. However, patients with PE would require long-term anticoagulation therapy, which is not a standard treatment after myocardial infarction. Therefore, though initial management of these two conditions may be similar, the diagnosis of PE substantially changes postacute care from that of coronary disease and may prompt a work-up for coagulopathy. Clearly, the diagnosis of PE in those with chest pain has clinically significant treatment implications.

Conclusion

Specific abnormal findings on ECGs may provide clues to the diagnosis of PE in patients presenting with chest pain, dyspnea, or both. Typically, these changes involve nonspecific precordial ST-segment depression, T-wave inversion, or changes consistent with right ventricular strain. However, the present case illustrates the rare association of PE with ST-segment elevation, particularly in the anteroseptal leads. Therefore, we recommend that physicians consider the presence of PE in patients with chest pain or dyspnea, even when ST-segment elevation is present.

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