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Case Report

The Master-Masquerader: Massive Pulmonary Embolism

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Abstract:

A 69-year-old woman was brought to the emergency department (ED) by ambulance with symptoms of severe crushing chest pressure and dyspnea. Her history was limited at the time of evaluation due to the acuity of her symptoms and absence of family.

Key words: anterior STEMI; acute pulmonary embolism; electrocardiogram

Abbreviations list:

CPR: cardiopulmonary resuscitation
ECG: electrocardiogram
ED: emergency department
LAD: left anterior descending artery
LCX: left circumflex artery
LPA: left pulmonary artery
MPA: main pulmonary artery
PE: pulmonary embolism
RA: right atrium
RCA: right coronary artery
RPA: right pulmonary artery
RV: right ventricle
STEMI: ST-segment elevation myocardial infarction
TVP: transvenous pacing lead

Case Presentation:

A 69-year-old woman was brought to the emergency department (ED) by ambulance with symptoms of severe crushing chest pressure and dyspnea. Her history was limited at the time of evaluation due to the acuity of her symptoms and absence of family. Her lone medication was lisinopril 40 mg daily. A 12-lead electrocardiogram (ECG) performed by emergency medical service prior to patient arrival showed anteroseptal ST-segment elevation (Figure 1) and she had received aspirin 325 mg en route to the hospital.

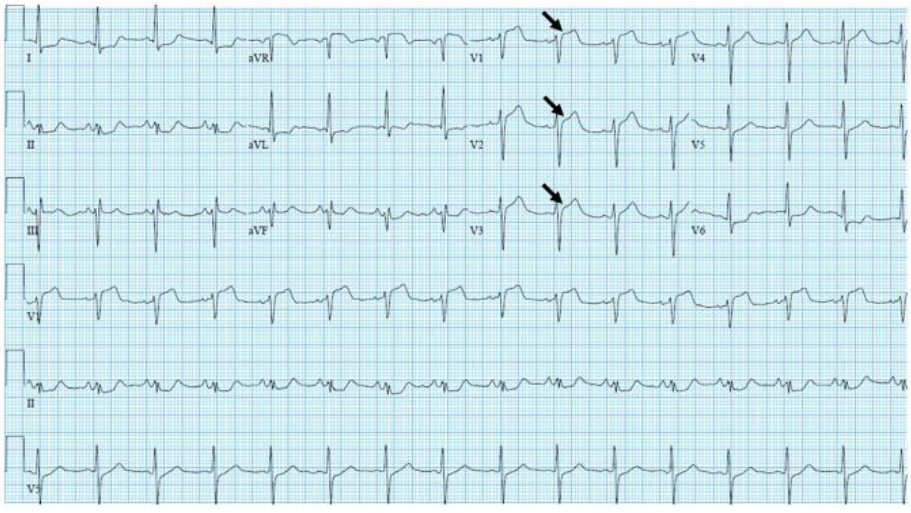


Figure 1: Initial electrocardiogram showing anterior ST-elevation (arrows) in leads V1, V2, V3 with corresponding reciprocal changes.

On presentation the patient was in severe respiratory distress. She was afebrile and examination revealed blood pressure 86/61 mmHg, pulse 94/min and oxygen saturation 82% on room air, and she was placed on supplemental oxygen. Cardiac assessment revealed a regular rhythm and normal heart sounds. There were no pulmonary crackles or peripheral edema**,** and jugular venous pulsation was mildly elevated. The remainder of the examination was unremarkable.

An ECG was repeated in the ED and confirmed anterior ST-segment elevation with reciprocal ST-segment depressions in the anterolateral leads. Shortly after examination, the patient became hypotensive, lightheaded, and increasingly dyspneic, with blood pressure falling to 62/40 mmHg and onset of sinus bradycardia to 44/min.

The patient was urgently intubated and placed on mechanical ventilation. Shortly thereafter, she became pulseless and required cardiopulmonary resuscitation (CPR) which resulted in return of spontaneous circulation within four minutes.

In the cardiac catheterization laboratory, the patient again became pulseless and responded to CPR with return of spontaneous circulation in six minutes. Therapy with intravenous fluids and vasopressors was initiated. Due to persistent bradycardia and hypotension, a transvenous cardiac pacemaker was placed. Coronary angiography was performed and demonstrated 70% stenosis in the proximal right coronary artery and minor irregularities of the left anterior descending and circumflex arteries (Figure 2).

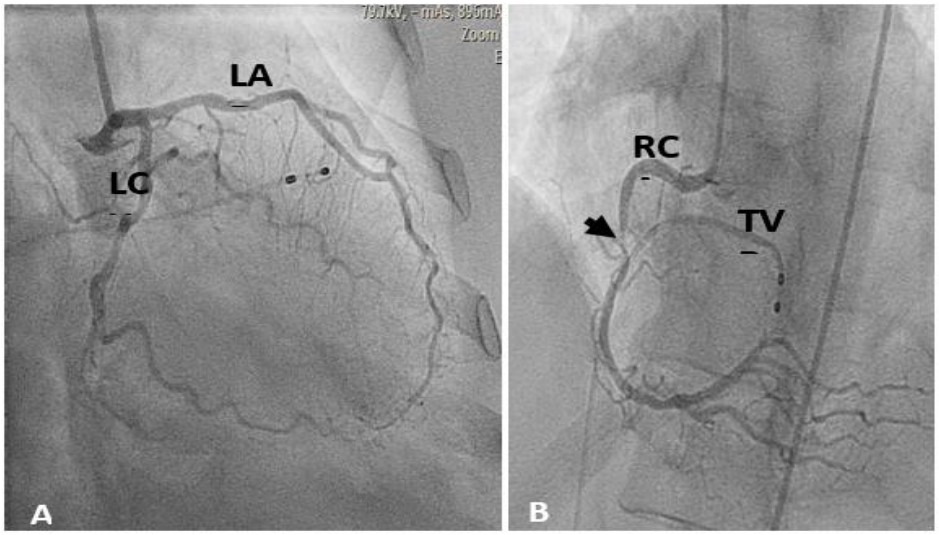
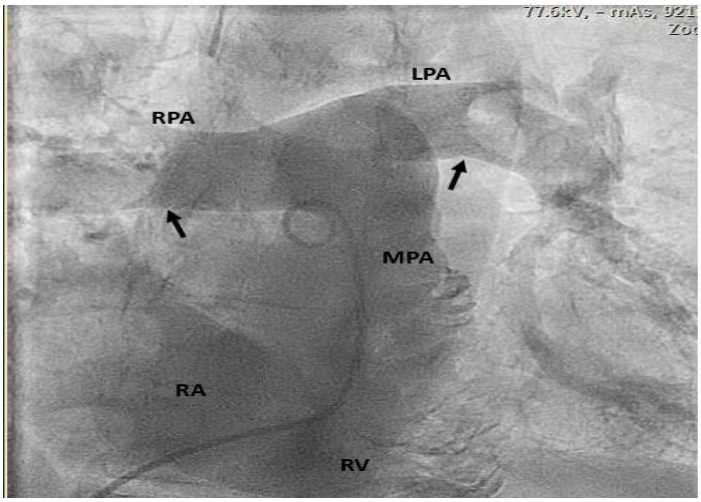


Figure 2: Coronary angiography: Panel A, right anterior oblique-caudal projection showing minor irregularities of the left anterior descending (LAD) and left circumflex (LCX); also seen is the transvenous pacing (TVP) lead in the right ventricle. Panel B, left anterior oblique projection showing 70% stenosis of the right coronary artery (RCA) (arrow); TVP lead noted in the right ventricle.

Because of lack of correlation between the ECG and results of coronary angiography, there was concern for alternative diagnoses. Right heart catheterization revealed pulmonary artery pressure of 37/12 mmHg (mean 23) and pulmonary angiography demonstrated extensive, bilateral pulmonary emboli (Figure 3).



RPA: right pulmonary artery; LPA: left pulmonary artery; MPA: main pulmonary artery; RA: right atrium; RV: right ventricle.

Figure 3: Pulmonary angiogram in anterior-posterior projection, with 5 French pigtail catheters in the main pulmonary artery: Arrows point to large, proximal intraluminal filling defects/abrupt cutoffs of both right and left pulmonary arteries, consistent with massive right and left pulmonary artery emboli.

Intravenous tissue plasminogen activator was emergently administered but the patient again became pulseless. A prolonged cardiopulmonary resuscitative effort was unsuccessful, and the patient expired.

Discussion:

Pulmonary embolism (PE) is a medical emergency whose presentation can masquerade as a variety of clinical syndromes. Moreover, multiple non-ischemic conditions can present with anterior ST-segment elevation1. These include 1-2 mm ST elevation that is common in the normal ECGs of healthy men, benign early repolarization, and ST elevation in the right precordial leads with left ventricular hypertrophy and with left bundle branch block. These elevated ST-segments can mask anterior ST elevation myocardial infarction (STEMI). Acute pericarditis can also produce diffuse ST-segment elevation. Similar changes may also be seen with acute myocarditis, stress cardiomyopathy, and hyperkalemia. Brugada syndrome is defined by a distinctive pattern of ST elevation in the right precordial leads. Acute coronary spasm can produce ST elevation due to transmural ischemia, which is indistinguishable from acute myocardial infarction. Transient ST-segment elevation of up to 5 mm may be seen following electrical cardioversion2. However, in PE the ECG is often nonspecific, showing sinus tachycardia and alterations suggestive, but not diagnostic, for PE (right bundle branch block, 'S1-Q3-T3' ECG pattern, and precordial T-wave inversions3. None of these patterns were present in our patient's ECGs. Although ST-elevation in the anteroseptal ECG leads in acute PE is unusual, it has been reported4,5. As in our patient, recognition of PE in these cases has usually followed exclusion of the leading diagnosis of acute coronary syndrome4.

The mechanism of anterior ST elevation as a presenting finding of acute PE is unclear. In a systematic literature review of all PE patients presenting with ST elevation, a total of 34 case reports of patients were identified5. ST elevation was most frequently located in the anterior ECG leads: V3 (74% of cases), V2 (71%), V1 (62%), V4 (47%). Mortality was very high (50%) in patients presenting with ST-segment elevations in these distributions. ST elevation in the inferior ECG leads was less frequent but not rare: a VF (21%), III (18%), II (12%) and mortality was considerably lower in this group (11%) than in those with ST elevation in the anterior ECG leads. In the combined group of those with PE and anterior or inferior ST elevation, overall mortality was 26%. There was also a higher frequency of bilateral compared to unilateral pulmonary emboli (72% vs. 10%), with 28% of bilateral PEs associated with an embolism in the main pulmonary artery. In our patient there were massive bilateral PEs (Figure 3).

Several mechanisms have been proposed for anteroseptal ST elevation in PE6,7: right ventricular 'strain' secondary to right ventricular pressure overload; microvascular coronary vasospasm induced by acute right ventricular strain; hypoxemia-related catecholamine surge; paradoxical coronary embolism through a pre-existing patent foramen ovale or small atrial septal defect6,7. Urgent coronary and pulmonary angiography revealed the correct diagnosis in our patient**,** but rapid administration of thrombolytic therapy did not alter the patient's course because of the massive extent of the pulmonary emboli. Both the left and right pulmonary arteries were obstructed by the large, proximal emboli which likely resulted in impaired delivery of oxygenated blood to the left ventricle and rapid degeneration of cardiac function.

Although anterior ST elevation with hemodynamic compromise typically reflects acute myocardial infarction related to occlusion of the left anterior descending coronary artery, our case highlights the necessity of considering PE in the differential diagnosis of this unusual presentation.

Conflict of Interest: None of the authors have any conflicts of interest to disclose.

Author Contributions: All authors had access to the data and a role in writing the manuscript.

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