

ST-elevation and complete heart block: A rare presentation of pulmonary embolism

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ABSTRACT

Pulmonary embolism may present with nonspecific symptoms that overlap with acute coronary syndrome, including chest pain, shortness of breath, and palpitations. Common electrocardiogram findings include sinus tachycardia and right bundle branch block, whereas ST-segment elevation and complete heart block are rare. We report a case of a 77-year-old man who initially presented with transient complete heart block and ST-segment elevation on electrocardiography, raising concern for acute coronary syndrome. Left heart catheterization showed no significant obstructive coronary artery disease.

Computed tomography pulmonary angiography revealed a saddle pulmonary embolism, suggesting that thromboembolic disease was the primary cause of the electrocardiographic changes. Clinicians should maintain a high index of suspicion for pulmonary embolism in patients with abnormal electrocardiographic findings and relevant risk factors to avoid missing this potentially life-threatening diagnosis.

Keywords: Pulmonary embolism, Electrocardiography, Embolism, Heart Block

INTRODUCTION

Pulmonary embolisms (PE) cause approximately 60,000–100,000 deaths in the United States per year.¹ Historically, high-risk PEs have been an estimated inpatient mortality rate of 20.6–42.1%.² Clinical presentations are often heterogeneous and nonspecific, including chest pain, shortness of breath, palpitations, and syncope, which may mimic acute coronary syndrome (ACS). Although multiple electrocardiogram (EKG) findings have been documented in PE, ST-segment elevation or complete heart block are extremely rare and can result in misdiagnosis.

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CASE PRESENTATION

A 77-year-old man with an uncertain past medical history presented after being found unresponsive at home. On evaluation by Emergency Medical Services, he was lethargic, bradycardic with a heart rate of 40 beats per minute, and hypoxic, with a pulse oximetry reading of <70% on room air. History obtained was limited because the patient sustained a cardiac arrest upon presentation to the emergency department. The initial rhythm was pulseless electrical activity. After resuscitation, he regained spontaneous circulation. He was subsequently cannulated for veno-arterial extracorporeal membrane oxygenation (ECMO) due to ongoing hemodynamic instability and started on vasopressors.

Initial physical examination performed during resuscitation noted dilated pupils unresponsive to light and decreased breath sounds bilaterally. The patient's abdomen was distended but soft to palpation, with a

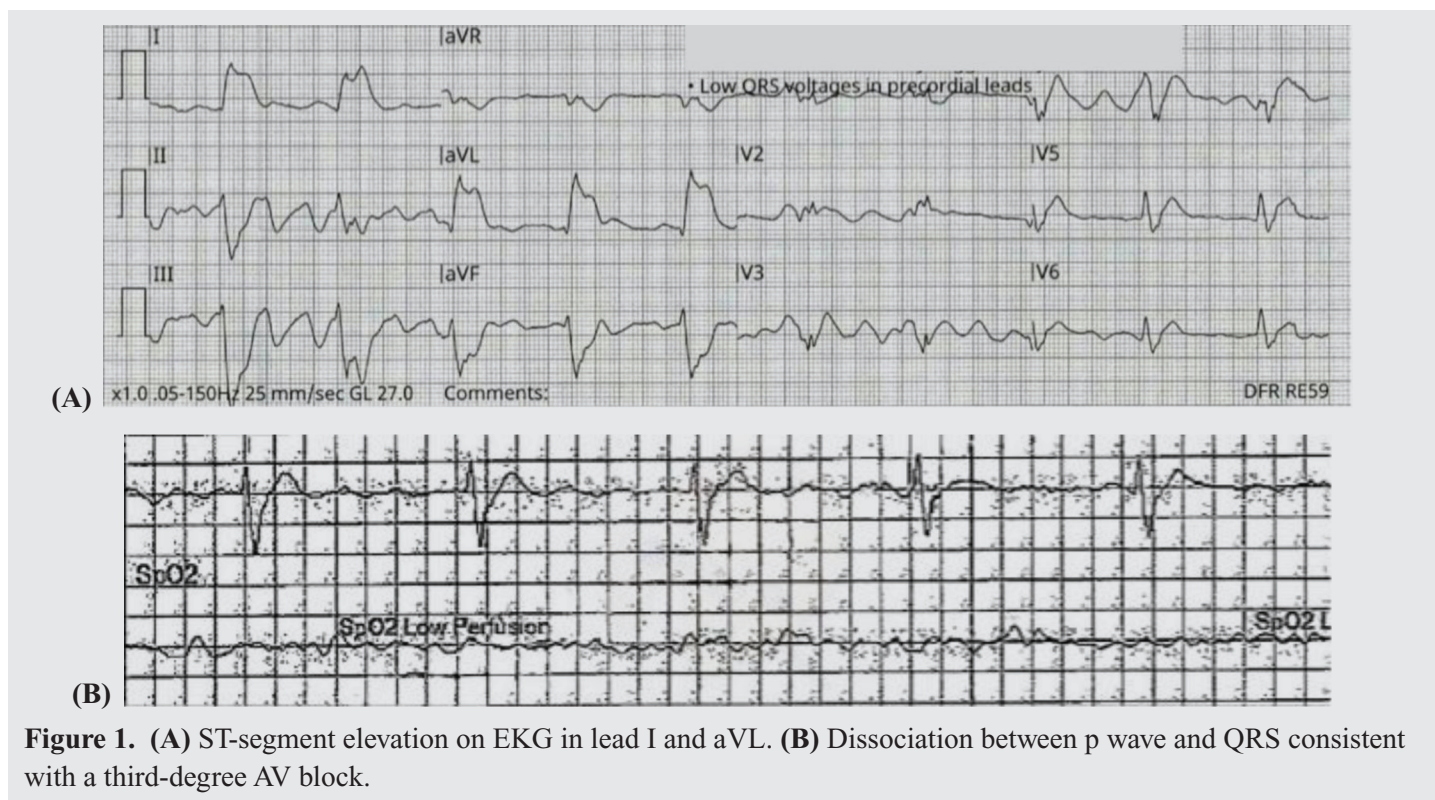


Figure 1. (A) ST-segment elevation on EKG in lead I and aVL. (B) Dissociation between p wave and QRS consistent with a third-degree AV block.

newly healed midline laparotomy incision. Bedside point-of-care ultrasound after return of spontaneous circulation revealed a severely reduced left ventricular ejection fraction and a dilated right ventricle with reduced systolic function.

Initial laboratory results showed a hemoglobin of 6.2 g/dL, white blood cell count of $11.3 \times 10^3/\mu\text{L}$, and platelet count of $193 \times 10^3/\mu\text{L}$. A comprehensive metabolic panel showed a creatinine of 1.43 mg/dL, potassium of 3.4 mEq/L, and phosphorus of 9.9 mEq/L. Lactic acid was 8.6 mmol/L. High-sensitivity troponin T was 185 ng/L and later peaked at 3,060 ng/L. An EKG performed en route raised concern for lateral ST-segment elevation myocardial infarction (STEMI) and possible complete heart block (Figure 1). Findings of complete heart block were transient and resolved on a 12-lead EKG obtained at presentation. The patient underwent emergent left heart catheterization, which showed 30% stenosis of the proximal left circumflex artery but no obstructive disease in the remaining

vessels. To evaluate for other causes of the EKG abnormalities and shock, a computed tomography pulmonary angiogram (CTA) was then obtained, which showed a saddle pulmonary embolism (Figure 2). Since this patient presented with severe hemodynamic instability due to pulmonary embolism, immediate endovascular thrombectomy was performed and yielded a large clot burden extending from the pulmonary artery bifurcation to the truncus anterior, left main pulmonary artery, and multiple segmental branches (Figure 3).

Further history obtained from family members indicated that the patient had recently been diagnosed with colonic adenocarcinoma and had undergone a partial colectomy. He had no prior history of heart failure. Several days into the admission, his left ventricular ejection fraction improved to 50%–55% on repeat transthoracic echocardiography, suggesting that the initial reduction in function may have been due to post-cardiac arrest myocardial stunning. Despite

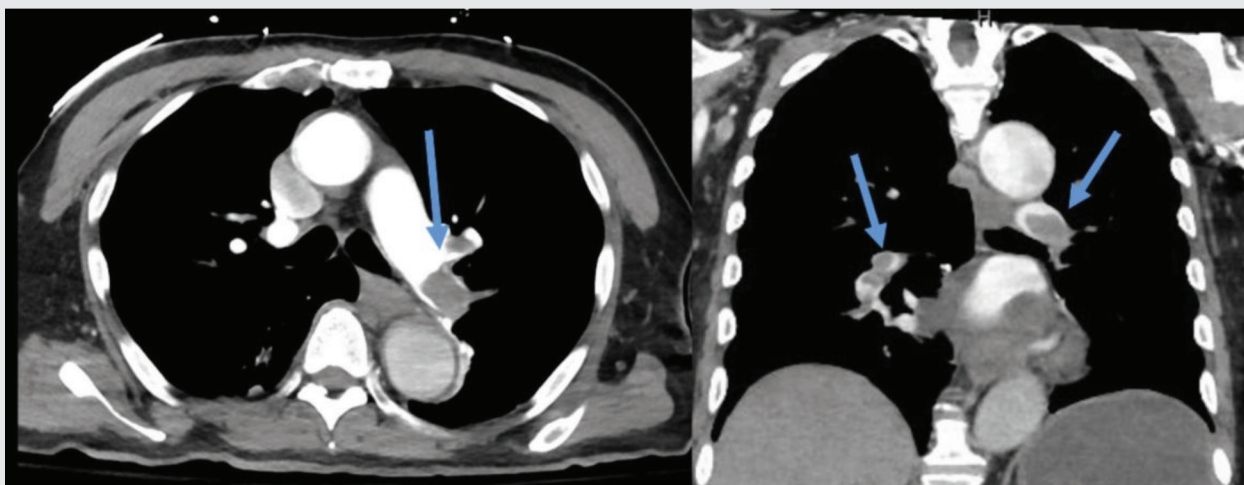


Figure 2. CTA pulmonary noted bilateral pulmonary embolism. Blue arrows indicate filling defects noted on imaging.

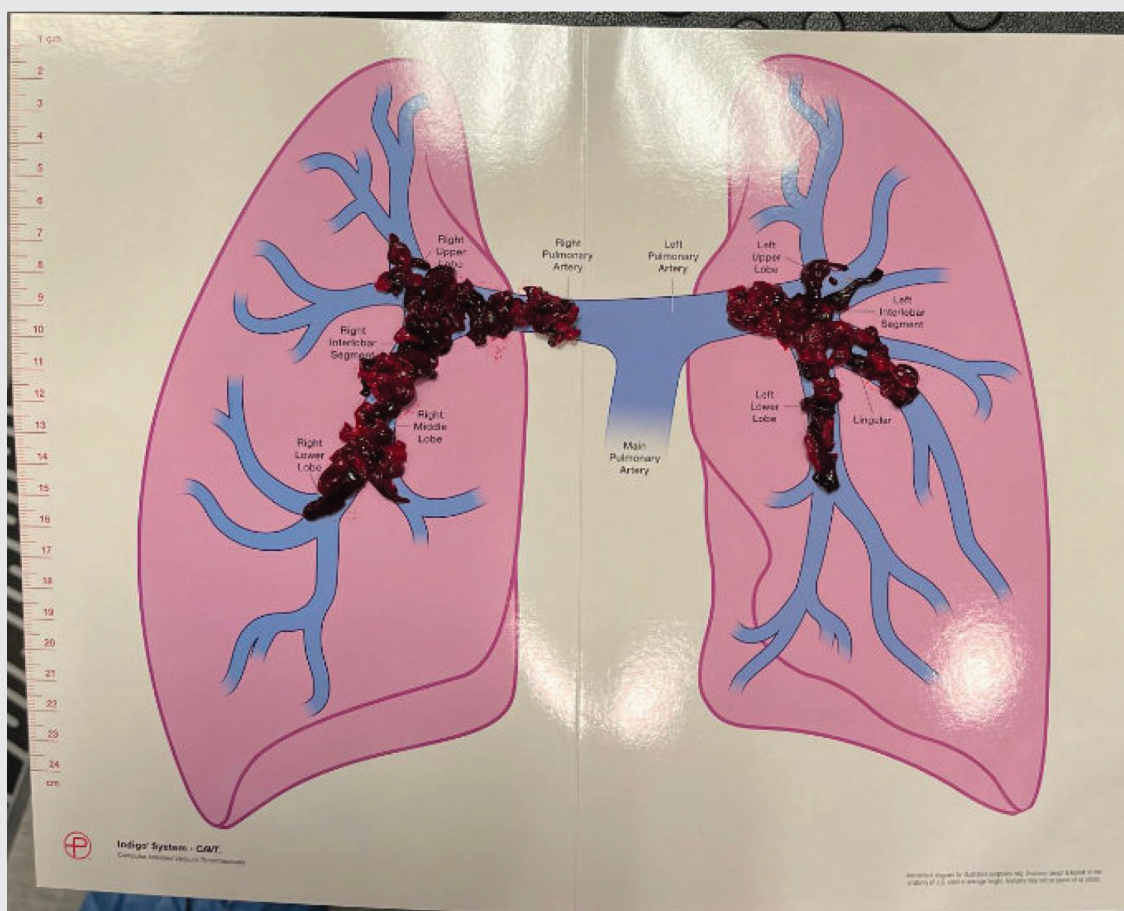


Figure 3. Image of clots removed during thrombectomy. Clot burden extends from the primary pulmonary artery bifurcation to several segmental branches.

maximal mechanical support with VA ECMO, a left ventricular assist device, and mechanical ventilation, the patient continued to deteriorate and showed signs of severe neurologic injury on imaging, absent cranial nerve reflexes, and lack of cortical response on somatosensory evoked potential testing. The family ultimately decided to withdraw life-sustaining therapies, and the patient expired.

DISCUSSION

This case underscores the clinical significance of maintaining a high suspicion for PE. The patient's presentation was highly concerning for ACS given the presence of ST elevations and complete heart block on EKG, which are uncharacteristic of PE. Further investigation of his medical history revealed a recent surgery that may have predisposed him to venous thrombosis, prompting an astute clinician to consider etiologies aside from ACS. The CTA findings of PE in combination with the EKG findings initially led the care team to suspect concurrent ACS and pulmonary embolism. However, his left heart catheterization showed no clinically significant obstructive disease, suggesting pulmonary embolism as the primary cause of his EKG changes.

Multiple EKG findings have been documented in patients with PE, the most common being sinus tachycardia, with reported incidence of 31% in a recent large meta-analysis.³ Other findings such as S1Q3T3 (i.e., a deep S wave in lead I, a Q wave in lead III, and T-wave inversion in lead III), also known as the McGinn-White sign, and right bundle branch block may also be seen.⁴ Less commonly, ST-segment elevation can occur, most often in the precordial and inferior leads.⁵ Complete heart block occurring with a PE is rarely reported. Those presenting with EKG changes such as ST segment elevation, complete right bundle branch block, or low voltage EKG have a higher risk of inpatient mortality.⁶ Furthermore, the overlap of clinical symptoms and EKG abnormalities between ACS and PE can lead to a delayed or missed diagnosis. To our knowledge, this is only the second reported case of a patient with pulmonary embolism presenting with both ST-segment elevation and complete heart block.

Two main mechanisms for ST-segment elevations in the setting of PE have been proposed. First, obstruction of the pulmonary artery increases right ventricular afterload, causing right ventricular failure, decreased left ventricular filling, and hypotension, which leads to myocardial ischemia.^{1,5} Secondly, neurohumoral activation due to hypotension, causing a release of catecholamines may cause coronary vasospasm and create a "pseudo-infarct" phenomenon.⁷

In cases of complete heart block with PE, prior investigators noted that an acute rise in right ventricular pressure impairs conduction of the right bundle branch in patients with a preexisting left bundle branch block, resulting in the development of third-degree AV block.^{8,9} The large clot burden, as noted from the thrombectomy (Figure 3), likely created strain on the right ventricle capable of impairing the right bundle branch conduction. In this case, preexisting left bundle branch block cannot be excluded without prior baseline EKG. Another proposed mechanism is that PE and resultant poor left ventricular filling may activate the Bezold-Jarisch reflex.¹⁰ This is a cardio-inhibitory reflex that occurs when the cardiac vagal afferent pathway is stimulated, resulting in bradycardia, hypotension, and peripheral vasodilation. This can progress to complete heart block in some patients. Proposed triggers for this reflex include chemo-mediators released during ischemia and subsequent reperfusion of the myocardium or mechanoreceptor activation; however, the exact physiology of this mechanism remains unclear. Moreover, the atrioventricular node receives its blood supply from the right coronary artery in most of the population. A low perfusion state or coronary spasm can transiently block perfusion of the AV node leading to complete heart block.⁴

CONCLUSION

Pulmonary embolism presents with non-specific clinical findings. Cardiac biomarkers and EKG may not adequately differentiate between ACS and PE. ST-segment elevation or third-degree AV block can indicate severe right ventricular strain in PE and is associated with a greater risk of mortality. If CTA is not readily available, a quick assessment of right

ventricular strain with bedside ultrasound should be performed in at-risk populations.

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