



A Case of Asymptomatic Bilateral Large Pulmonary Embolism Masquerading as ST Elevation Myocardial Infarction

Belirti Vermeyen İki Taraflı Büyük Pulmoner Emboli Olgusu: ST Elevasyonlu Miyokard İnfarktüsü Taklısı

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ABSTRACT

Pulmonary embolism (PE) is a common and life-threatening medical emergency, but it is still often misdiagnosed due to its wide variety of clinical manifestations. We report a case of a 78-year-old man who presented with urinary retention without any classical symptoms, and was found to have bilateral large PE as confirmed by computed tomography pulmonary angiogram. Initial electrocardiogram (ECG) showed ST segment elevation and T-wave inversion in anteroseptal and inferior leads, with reciprocal changes in lateral leads. Clinicians need to be aware that a diagnostic dilemma between PE and acute coronary syndrome is not uncommon, as both conditions can present with "ischemic-looking" ECG and elevated troponin levels. To our knowledge, this is the first case of an atypical, incidental finding of PE in a patient who presented with urinary retention reported in the literature. Careful analysis and interpretation of ECG are necessary to improve patient evaluation and support clinical decision-making in order to provide the best possible care.

Keywords: Pulmonary embolism, STEMI, urinary retention, prostate malignancy

Öz

Pulmoner emboli (PE), yaygın ve yaşamı tehdit eden bir acil durum olmasına rağmen, klinik belirtilerinin çeşitliliği nedeniyle sıkılıkla yanlış tanı almaktadır. Biz, klasik semptomları olmayan ve üriner retansiyon ile başvuran 78 yaşında bir erkek olguya sunuyoruz; yapılan bilgisayarlı tomografi pulmoner anjiyografi ile bilateral büyük PE saptanmıştır. İlk elektrokardiyogramda (EKG), anteroseptal ve inferior derivasyonlarda ST segment elevasyonu ve T dalga inversionsı, lateral derivasyonlarda ise karşıt değişiklikler gözlenmiştir. Kliniklerin, PE ile akut koroner sendrom arasında tanışılabilirlerin nadir olduğunu ve her iki durumunda "iskemiye benzer" EKG değişiklikleri ve artmış troponin seviyeleri ile kendini gösterebileceğini bilmesi önemlidir. Bildiğimiz kadariyla, üriner retansiyon ile başvuran bir hastada PE'nin bu şekilde atipik ve tesadüfi olarak saptandığı ilk olgudur. Hastaların değerlendirilmesinde ve en iyi bakımin sağlanmasında EKG'nin dikkatli analizi ve yorumlanması, klinik karar verme sürecini desteklemek açısından gereklidir.

Anahtar Sözcükler: Pulmoner emboli, STEMI, üriner retansiyon, prostat malignitesi

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INTRODUCTION

Pulmonary embolism (PE) is a common cardiovascular emergency that is notoriously difficult to diagnose due to a wide spectrum of clinical manifestations. Classic presentations of PE include sudden-onset pleuritic chest pain associated with breathlessness. However, it can also manifest as silent PE, deep vein thrombosis, postural dizziness, or syncope, hemoptysis, sudden cardiac arrest, acute respiratory distress syndrome, arrhythmia, acute heart failure, abdominal pain, delirium, hypoxia, and shock. In some cases, there may be a less obvious connection to paradoxical embolism, such as myocardial infarction, stroke, or lower limb embolism (1). Electrocardiogram (ECG) changes in PE can be inconsistent and non-specific, with common findings including sinus tachycardia, a normal ECG, McGinn-White Sign (S1Q3T3 pattern), rightward axis shift, P pulmonale, complete or incomplete right bundle branch block (RsR' pattern in right precordial lead), atrial dysrhythmia, atrioventricular block, low voltage complexes, and T-wave inversion in the right precordial leads, as well as the less common ST segment elevation (2-6) and ventricular tachycardia (6). Some case reports propose that elevated troponin and ST segment elevation in PE may be due to two possible mechanisms: 1) right ventricular dilatation and failure resulting from increased right heart pressure and afterload due to outflow obstruction, leading to right ventricular ischemia; and 2) paradoxical coronary artery thromboembolism via intra-atrial communication (7). Here, we present a case of an elderly man with atypical presentation who was diagnosed with bilateral PE that mimicked ST-elevation myocardial infarction (STEMI) on the ECG.

CASE REPORT

A 78-year-old gentleman presented with a 20-hour history of urinary retention, abdominal distension for a week, and bilateral leg swelling for a month. Upon arrival at the emergency department, a urinary catheter was immediately inserted, as a bladder scan showed over 1000 mL of urinary retention, with over 2 litres of clear urine being drained. The patient denied experiencing any other symptoms such as breathlessness, chest pain, or urinary symptoms. His medical history included hypothyroidism, hypertension, mild aortic stenosis

(AS), and prostate cancer. He was on regular medication, including Levothyroxine 125 mcg, Tamsulosin 400 mcg modified release, and Verapamil 240 mg slow release for essential hypertension. Additionally, he received Leuprorelin injections every 3 months for prostate cancer. The patient is an ex-smoker and lives independently with his wife. Vital signs were relatively stable, with a temperature of 36 °C, blood pressure of 120/78 mmHg, pulse rate of 88 beats per minute, respiratory rate of 17 per minute, and oxygen saturation of 93% under room air. He was fully conscious and well oriented to time, place, and person. On cardiovascular examination, normal heart sounds were noted, along with an ejection systolic murmur. Reduced air entry was observed on the left side of the chest. The abdomen was slightly distended without shifting dullness, and bilateral pitting pedal edema was present.

The chest X-ray revealed a left-sided pleural effusion with consolidation underneath. Routine blood tests showed a slightly elevated white cell count of $12.9 \times 10^9/L$ (normal range: $4-11 \times 10^9/L$), neutrophil count of $9.6 \times 10^9/L$ (normal range: $2.0-7.5 \times 10^9/L$), a raised platelet count of $473 \times 10^9/L$ (normal range: $150-400 \times 10^9/L$), and normal electrolyte levels with creatinine of $109 \mu\text{mol}/L$ (normal range: $60-110 \mu\text{mol}/L$) and eGFR of $56 \text{ mL/min}/1.73 \text{ m}^2$ (normal range: $>60 \text{ mL/min}/1.73 \text{ m}^2$). The total protein level was 65 g/L (normal range: $60-80 \text{ g/L}$), albumin was 28 g/L (normal range: $35-50 \text{ g/L}$), CRP was 50 mg/L (normal range $<1 \text{ mg/L}$), and troponin was modestly raised at 89.1 ng/L (normal range $<14 \text{ ng/L}$). The Prostate-specific antigen level was 40 ng/mL (normal range: $<4 \text{ ng/mL}$), and D-dimer was 7754 ng/mL (normal range: $<500 \text{ ng/mL}$). International normalized ratio was 1.5 (normal range: $0.9-1.2$); and prothrombin time and APTT were normal. A resting 12-leads ECG showed sinus rhythm, normal axis, and the S1Q3T3 pattern (presence of Q wave and T-wave inversion in Lead III, prominent S wave in Lead I). It also showed ST-segment elevation and T-wave inversion in Leads V1-4, Lead III, and aVF, along with reciprocal ST depression in Lead I and aVL (Figures 1, 2). These ECG findings led to a differential diagnosis of PE versus acute coronary syndrome (ACS). His echocardiogram showed normal biventricular size and wall thickness, normal systolic function with an estimated EF of $\sim 60-65\%$ (normal range: $55-70\%$), moderate AS, and 4 cm of left pleural effusion.

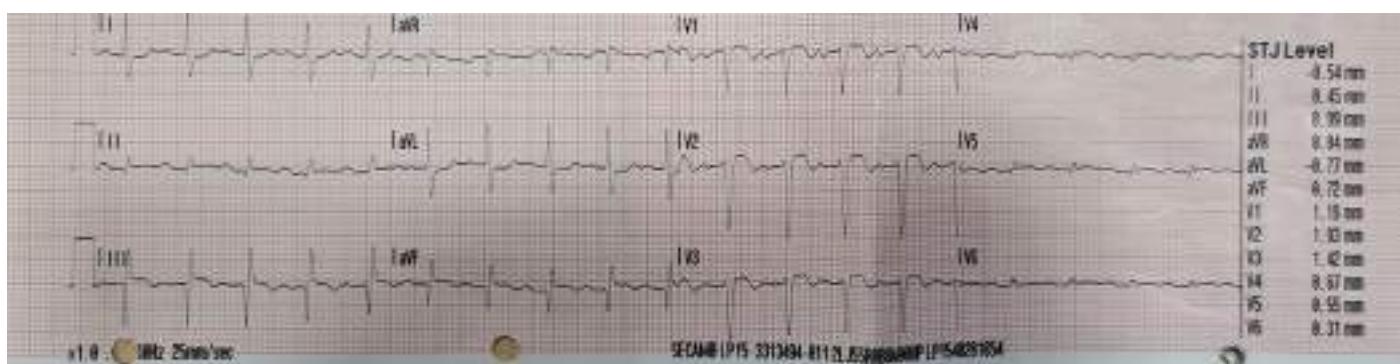


Figure 1. Electrocardiogram showed ST elevation in lead III, aVF, V1-4, along with reciprocal subtle ST depression in lead I, aVL, and an S1Q3T3 pattern.

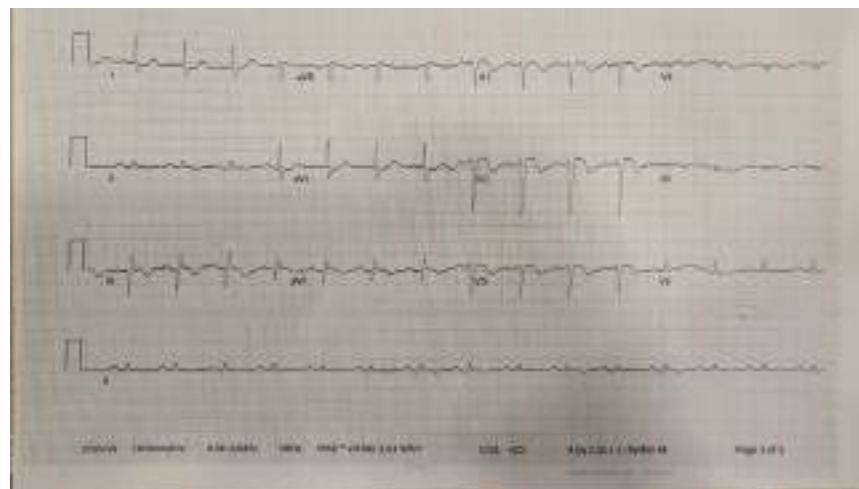


Figure 2. Electrocardiogram showed ST elevation in lead III, aVF, V1-4 along with subtle ST depression in lead I, aVL and S1Q3T3 pattern.

Nasal prong oxygen support of 2 liters was given, and Aspirin 300 mg was initially administered, considering the possibility of myocardial ischemia. Subsequent blood tests on serial troponin levels and an urgent computed tomography pulmonary angiogram (CTPA) were requested. As per trust protocol, Antibiotics were also provided to cover chest infection. The second troponin was measured at 187. CTPA revealed large volume central filling defects involving the left main pulmonary artery, extending just across the main bifurcation, and bilateral upper lobe and lower lobe pulmonary arteries, with an enlarged pulmonary artery measuring 32 mm and evidence of right heart strain (Figure 3). In the context of a hemodynamically stable condition, he was treated low molecular weight heparin (Dalteparin SC injection 15,000 units once daily) for bilateral PE. After treatment, normalized troponin levels and resolved ECG changes were reassuring to exclude an acute coronary event. Coronary angiogram was therefore not pursued (Figure 4). The case was discussed with the urology team and in. The bone scan revealed widespread osseous metastases within the spinal column, pelvic bones, ribs, scapulae and the proximal right femur (Figure 5). Bicalutamide 50 mg, for maximum androgen blockade treatment, was commenced, and then the patient was discharged home with oral anticoagulant (Apixaban 5mg twice daily), long-term catheter, and a follow-up plan with the urology team.

DISCUSSION

PE is a prevalent and potentially life-threatening cardiovascular condition with multiple diagnostic challenges. To our knowledge, this case represents a silent presentation of PE, mimicking STEMI, which is unique in the literature. It is also noteworthy that, despite having a large bilateral PE with right heart strain, the patient exhibited no chest pain, dyspnea, hypotension, or tachycardia, unlike other cases reported in the literature (4,5,8). Moreover, despite showing mildly low oxygen saturation, which is a common feature of massive or submassive PE, a relatively low suspicion of PE

was due to the presence of pleural effusion and consolidation on the chest radiograph.

Secondly, in our case, the patient's history and presentation did not suggest silent PE, which could have led to miss ECG examination due to the lack of indication and absence of classical cardiovascular or pulmonary symptoms. The literature on classical features of reciprocal ischemic ECG changes and modestly raised troponin levels with an upward trend, favors the alternative diagnosis of ACS rather than PE, which prompted ACS treatment initially. According to the literature, reciprocal ECG changes are specific for coronary ischemic disease with a high predictive value (9). However, it is worth noting



Figure 3. Computed tomography pulmonary angiogram showed bilateral pulmonary emboli.

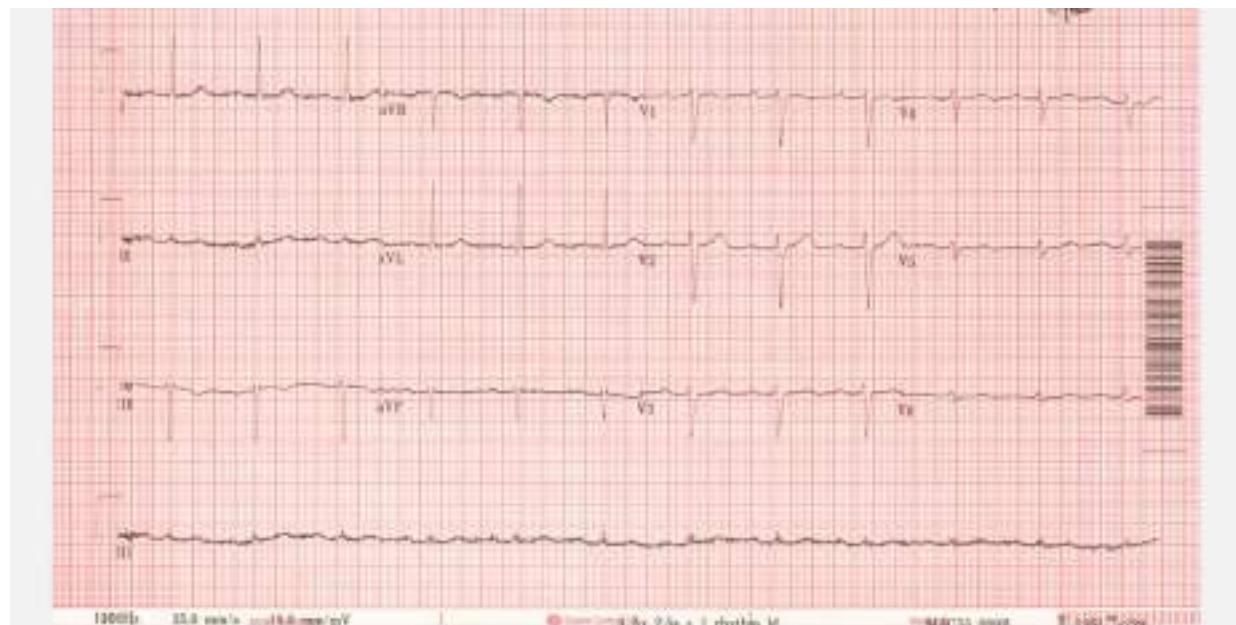


Figure 4. The electrocardiogram showed resolved changes of ischemia.

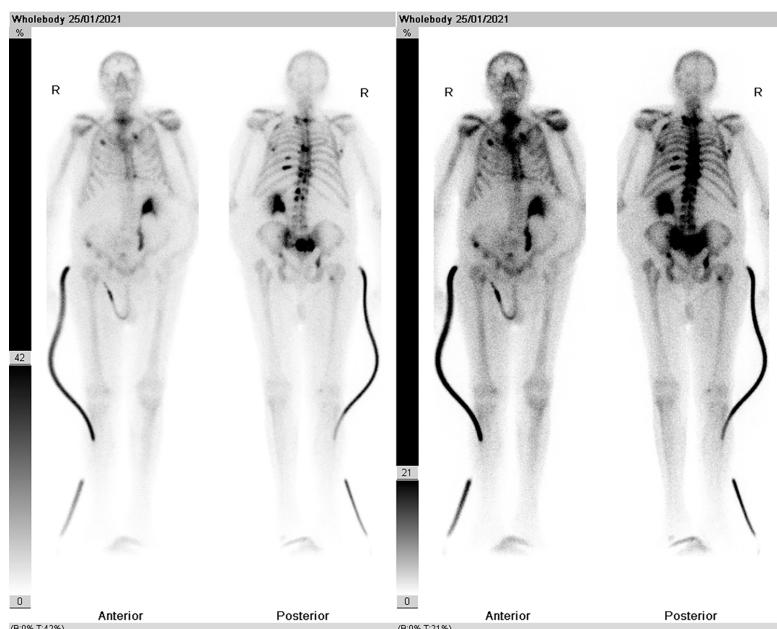


Figure 5. Bone scan showed extensive bone metastases.

that Ladage et al. (8) recently reported a non-coronary artery occlusion case with anterior STEMI and reciprocal ST changes in inferior leads, but without evidence of right heart strain in a patient with PE. Additionally, T-waves inversion in precordial or inferior leads, could indicate right heart strain in massive PE (4), but these findings can also be found in ACS.

In this case, the presence of active malignancy as a significant risk factor for PE, in the context of abnormal ECG findings, including right heart strain pattern, prompted investigation with CTPA, confirming bilateral large PE. Many cases of PE with ST elevation V1-V3/V4 have been described in the literature (3,4,8,9). If ST elevation is present

in PE, the likelihood of RV strain (100%), RV dysfunction (100%), and bilateral PE (75%) is high. Therefore, clinicians should be vigilant in carefully examining the ECG, even in elderly patients who do not complain of chest pain or dyspnea. Elevated troponin levels due to right ventricular ischemia and failure are associated with increased morbidity and mortality in patients with PE (10-13).

Timely diagnosis of acute PE is critical to prevent hemodynamic decompensation and potential complications, including cardiac arrest. Differentiating between acute and chronic PE in asymptomatic patients remains challenging (14). Certain features, such as ECG reversibility with anticoagulation, an enlarged pulmonary artery

diameter, and the difficulty in distinguishing acute from chronic PE in asymptomatic patients, suggest an acute PE diagnosis. Promptly distinguishing between PE and ACS is essential for appropriate management and to prevent mortality. Clinicians should be aware that bilateral PE can manifest atypically in elderly patients, resembling ST elevation myocardial infarction, due to right ventricular strain and ischemia.

CONCLUSION

Our case presents a unique and atypical presentation of bilateral large PE in an elderly patient without cognitive impairment. The absence of classical symptoms like chest pain or dyspnea posed diagnostic challenges, and the initial ECG findings mimicking ACS prompted timely treatment for ACS. However, thorough ECG analysis and subsequent investigations confirmed the diagnosis of PE. This case underscores the need for careful evaluation and a high index of suspicion to differentiate between PE and ACS promptly. It also highlights the importance of considering atypical presentations in elderly patients. Overall, our report adds clinical significance to the existing literature on PE and emphasizes the need for timely and accurate management decisions to prevent adverse outcomes and improve patient care.

Ethics

Informed Consent: The patient has provided informed consent for the publication of this case and has signed the consent form.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.H.O., T.T.M., Concept: T.T.M., Design: T.T.M., Data Collection or Processing: T.T.M., Analysis or Interpretation: M.H.O., Literature Search: M.H.O., Writing: M.H.O., T.T.M.

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