

Peculiar association of pulmonary embolism with evolved inferior wall myocardial infarction

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ABSTRACT

Inferior wall myocardial infarction (MI) is one of the common straightforward cardiac conditions in the emergency department (ED) but inferior wall MI masquerading pulmonary embolism (PE) is extremely rare and can be missed if not evaluated promptly in ED. Misdiagnosis of PE is associated with high mortality. Here, we report a case of a 67-year-old male who was admitted to the ED and has been diagnosed with evolved inferior wall MI based on his clinical presentation and electrocardiogram. Later, he developed syncope following which he was reassessed and evaluated for the other possible conditions. Bedside echocardiography findings raised suspicion for PE, which was further confirmed by computed tomography pulmonary angiogram (CTPA). The patient underwent successful thrombolysis and was scheduled for an elective coronary angiogram. This case report highlights the importance of clinical presentation and the benefits of bedside echocardiography that helped in suspecting the association of PE with evolved inferior wall MI.

Key words: Inferior wall myocardial infarction, Pulmonary embolism, Electrocardiogram, Echocardiography, Computed tomography pulmonary angiogram, Thrombolysis

Chest discomfort and dyspnea are the most common symptoms in the emergency department (ED) with a number of differential diagnoses requiring rapid refinement [1]. Symptoms of myocardial infarction (MI) such as chest pain, dyspnea, syncope, and palpitations can be similar to other cardiovascular emergencies such as aortic dissection, pulmonary embolism (PE), and heart failure [1]. The challenging part in such an emergency scenario is to diagnose the one serious condition that is hidden behind or masqueraded by another straightforward diagnosis. Even with a complete history and physical examination, there will be diagnostic uncertainty leading to a delay in definitive treatment [1]. Once the examination gets completed, further evaluation with an electrocardiogram (ECG) begins. In MI, the acute presentation shows ST-segment elevation, while a delayed presentation shows pathological Q waves and T inversion. In PE, there are numerous non-specific ECG findings [2]. Patients with PE predominantly showed ST-segment elevation in the anterior leads rather than inferior leads [3]. Echocardiography could be a better imaging tool for early diagnosis and risk stratification of PE in ED [4].

Here, we are reporting a rare case of PE masqueraded by the evolved inferior wall MI. Because of high mortality, early diagnosis and treatment are important to prevent life-threatening sequelae associated with both conditions.

CASE REPORT

A 67-year-old male presented to the ED with a history of chest discomfort and exertional dyspnea, while working at his farm on the previous day morning which lasted for 10 minutes. He had a similar episode the previous evening, which got resolved after a few minutes. He has been brought to the ED with chest discomfort and NYHA class IV dyspnea. No history of sweating, palpitations, lightheadedness, syncope, nausea, or vomiting present. He is a known case of type II diabetes mellitus for 6 years but there was no history of coronary artery disease (CAD), chronic obstructive pulmonary disease, dyslipidemia, chest wall diseases, malignancy, or COVID pneumonia. There's no family history of CAD. He is a non-alcoholic and non-smoker.

On examination, the patient's build and nourishment were normal. He was afebrile with normal jugular venous pressure and no pitting pedal edema. His initial vitals were heart rate of 128 beats/min, blood pressure of 130/90 mmHg, respiratory rate of 28 cycles/min, a saturation of 96% on room air, and capillary

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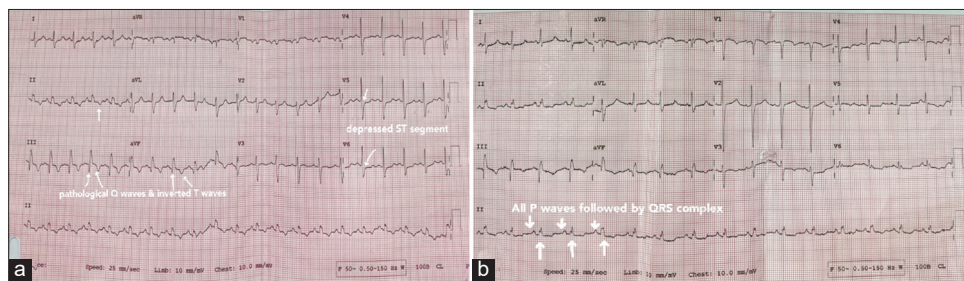


Figure 1: (a) Electrocardiogram (ECG) changes are sinus tachycardia, right axis deviation with lead II, avF showing pathological Q waves, leads II, III, avF showing T wave inversion, and leads V5, V6 showing reciprocal ST segment depression; (b) repeat ECG showing similar findings to the initial ECG with no AV nodal block

blood glucose of 314 mg/dL. Systemic examinations revealed normal findings.

Laboratory investigations were sent and ECG was taken. ECG findings were suggestive of evolved inferior wall MI (Fig. 1a).

The patient was initially treated with antiplatelet (aspirin 150 mg po), anticoagulant (enoxaparin 60 mg SC), and statin (atorvastatin 40 mg po) in ED. His glycemic control was maintained using an insulin sliding scale. After 20 min of the ED arrival, he developed lightheadedness followed by syncope. Following the syncope, we reassessed him and repeated ECG. His vital parameters and neurological examinations were found to be normal. Repeat ECG (Fig. 1b) was showing similar findings to the previous ECG and ruled out a high degree atrioventricular (AV) block. Due to syncope, we looked for other possibilities such as PE, aortic dissection, and aortic stenosis.

Bedside echocardiography (Fig. 2) was done which showed inferior wall hypokinesia, mild left ventricular dysfunction (ejection fraction 45%), right ventricular dilatation with moderate right ventricular dysfunction-tricuspid annular plane systolic excursion (TAPSE) of 12 mm, and moderate pulmonary artery hypertension with moderate tricuspid regurgitation-right ventricular systolic pressure (RVSP) of 80 mmHg.

The new-onset syncope, absent AV nodal block, normal blood pressure, and elevated RVSP (rare in the setting of MI) led to the suspicion of PE. By that time, blood investigations (Table 1) for complete blood counts, electrolytes, urea, creatinine, and coagulation profile were unremarkable except for elevation of troponin T of 220.4 NG/L (normal <5 NG/L).

CTPA (Fig. 3) revealed bilateral pulmonary artery thrombosis with more thrombus load in the right main pulmonary artery. Patients underwent thrombolysis using tenecteplase 30 mg IV (recombinant tissue plasminogen activator). His vital parameters before, during, and after thrombolysis were remaining stable, after which he was shifted to the coronary care unit with a plan of an elective coronary angiogram.

DISCUSSION

Emergency physicians should always be aware of the three prevalent diagnoses (MI, aortic dissection, and PE) when patients come with chest pain to the ED. Chest pain is always worrisome symptoms with causes ranging from benign to

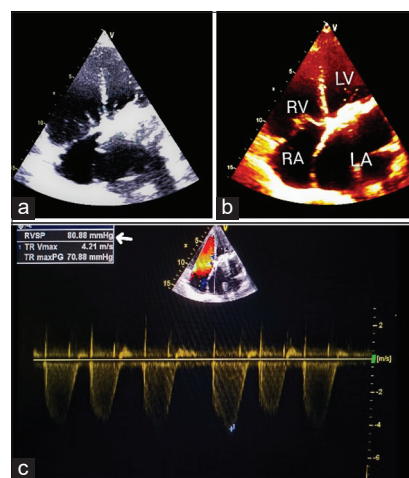


Figure 2: (a and b) Echocardiography images taken from apical four chamber view showing RV dilatation; (c) right ventricular systolic pressure measurements of 80 mmHg

Table 1: Blood investigations of the patient

Blood parameters	Patient values	Normal range
Hemoglobin	12.1	13.0–17.5 g/dL
Total WBC counts	11,100	4000–11,300 cells/cumm
Platelet counts	140,000	1,505,000–450,000 cells/uL
Serum urea	33	10–50 mg/dL
Serum creatinine	1.0	0.7–1.2 mg/dL
Serum sodium	134	132–146 meq/L
Serum potassium	4.3	3.7–5.4 meq/L
Prothrombin time	12	11–16 s
Activated partial thromboplastin time	32	29–34 s
INR (international normalized ratio)	1.00	

immediately life threatening. Delayed diagnosis often leads to significant morbidity and mortality. Identifying those high-risk diagnoses always remain challenging [5]. There occurs a delay in definitive diagnosis when patients present without chest pain but with atypical symptoms. Those patients were likely to be misdiagnosed when clinical findings and ECG changes were non-specific, resulting in worse outcomes [6].

The current American College of Cardiology/American Heart Association guidelines for STEMI recommends that the patients with symptoms suggestive of myocardial ischemia, who have

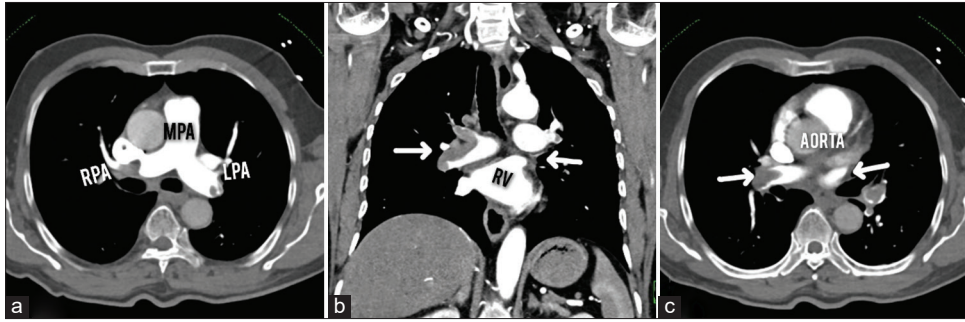


Figure 3: (a-c) CTPA images showing multifocal near complete and partial filling defects are noted in the central branch of pulmonary, lobar, segmental, and subsegmental arteries of both lungs. Features of the right heart strain were also noted (QANLNDI clot load score – 55%)

ST-segment elevation at the J point (in two contiguous leads or more of 0.2 mV or more in males or 0.15 mV or more in women in leads V2, V3, and/or of 0.1 mV or more in all other leads in threshold) should undergo immediate reperfusion therapy [7].

In some patients, ECG may show pathological Q waves. Once infarction begins to resolve, repolarization abnormalities (ST-segment and T-wave changes) get stabilized and recognition of a remote MI is more difficult. Pathological Q waves are considered as the classic ECG sign of necrosis and remain the only universally recognized sign of MI. The fragmented QRS (fQRS) is another recently described sign of a remote MI. The presence of both Q wave and fQRS increases sensitivity up to 91.4% in identifying evolved MI. The emergence of Q waves in the ECG constitutes important prognostic value for clinicians [8].

Otto found that 63 patients among 123 (51%) with chest pain and ST elevation in pre-hospital care had a diagnosis rather than acute MI but the presence of reciprocal changes would improve the positive predictive value to 90% [9]. This condition is challenging for emergency physicians and even cardiologists.

ECG still has a major role in diagnosing and triage of patients presenting with chest pain but is limited by its sensitivity and specificity. ECG alone is not sufficiently sensitive or specific to diagnose PE as the most common changes occur infrequently and pose a challenge in clinical practice due to rare pathognomonic findings [10]. ST deviations are not among the usual findings associated with PE. It probably occurs due to acute right ventricular strain and elevated pressures resulting from a submassive or massive PE [11].

Patients presenting to the ED with chest pain require rapid evaluation for earlier definitive treatment to prevent mortality. Echocardiography is the one diagnostic modality by which we can rule in or rule out major cardiovascular diseases. It helps in the rapid identification of the abnormal findings pertaining to MI, PE, acute aortic diseases, or pericardial emergencies, because of its rapidity and accuracy. In case of MI, the most common echocardiography finding is regional wall motion abnormality [12].

In case of PE, echocardiography helps in detecting right ventricular dilatation, right ventricular dysfunction, tricuspid regurgitation, McConnell's sign, and pulmonary hypertension. It may play a major role in the diagnosis of PE even in the setting of hemodynamic instability. In addition to the above findings,

one independent predictor of poor prognosis is decreased TAPSE which measures RV systolic dysfunction in acute PE cases [4]. Echocardiographic evidence of RV dysfunction might increase mortality up to 2-fold in 2 weeks and 3 months. In addition, it helps in risk stratification and guides management plans [13]. CTPA has to be done as soon as PE is suspected and screened for thrombus in the major pulmonary arteries. The presence of a thrombus in the main pulmonary trunk always poses a high risk for adverse cardiac events [4].

High-risk PE cases always require rapid intervention like thrombolysis to potentially save a life. Thrombolytic therapy is effective if applied within the first 48 h of symptom onset. Its efficacy decreases significantly after 7 days, but it may be beneficial up to 14 days from symptom onset. It is indicated for all high risk cases to achieve rapid dissolution of thrombus in the main pulmonary artery. Thrombolysis promotes pulmonary gaseous exchange and perfusion, thereby improving right ventricular dysfunction [14].

CONCLUSION

Emergency physicians must be aware of all the possible life-threatening cardiovascular conditions when patients present with chest pain, dyspnea, or syncope to the ED. Syncope occurring before hospital arrival or during hospital stay should raise high suspicion for PE rather than MI. Bedside echocardiography findings like raised RVSP favored the suspicion of PE. Once the involvement of the main pulmonary artery has been confirmed by the CTPA, patients should undergo thrombolysis. Earlier the diagnosis better the clinical outcomes and survival.

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