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Atypical presentation of acute massive pulmonary embolism

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ABSTRACT
The diagnosis of pulmonary embolism (PE)can be challengingand continues to be difficult due to wide range of atypical presentations.
We report a 58-year-old female who presented to the emergency department with bilateral shoulder pains radiating to the neck and lower jaw.ECG together with high level of cardiac troponins was suggestive of acute coronary syndrome, on the other hand echocardiography together with D-Dimer raised the probability of pulmonary embolism. CT pulmonary angiography confirmed the final diagnosis of acute massive pulmonary embolism.
She was treated with unfractionated heparin infusion followed by Non-Vitamin-K-antagonist Oral Anticoagulants(NOAC) and was stable throughout her hospital stay.
This case highlights the necessity of keeping a broad differential diagnosis maintaining a systematic approach when dealing with nonspecific complaints.
Key wards: Acute coronary syndrome, Acute pulmonary embolism, Non-Vitamin-K-antagonist Oral Anticoagulants

I. INTRODUCTION

Acute pulmonary embolism (PE) is a common life-threatening cardiovascular emergency that is globally the third most frequent behind myocardial infarction and stroke. The diagnosis of PE may be challenging, as there can be a wide range of atypical presentations.[1]

The clinical signs and symptoms of acute PE are non-specific. In most cases, PE is suspected in a patient with dyspnea, chest pain, pre-syncope or syncope, or hemoptysis. Hemodynamic instability is a rare but important form of clinical presentation, as it indicates central or extensive PE with severely reduced hemodynamic reserve and a fatal prognosis. [**2]**

Acute PE interferes with both circulation and gas exchange. Right ventricular (RV) failure due to acute pressure overload is considered the primary cause of death in severe PE. Pulmonary artery pressure (PAP) increases if >30–50% of the total cross-sectional area of the pulmonary arterial bed is occluded by thromboemboli. [**3]**

II. CASE REPORT

A 58-year-old female with a known history of diabetes mellitus type 2, hypertension, chronic kidney disease and dyslipidemia, chronic low back pain and with difficulty in walking, no past history of surgical interventions presented to emergency department with recurrent attacks of bilateral shoulder pain radiated to the neck and lower jaw and associated with sweating, dizziness and light headedness. This was for 3 days duration occurring at rest and lasting for few minutes. No chest pain was reported and there were no previous similar attacks.

At the time of presentation,the patient was afebrile but was shocked, with a blood pressure of 80/41mmHg which failed to IV fluids infusion. Her pulse was 92/min bilateral equal, respiratory rate of 24/ min, and SPO2 saturation of 78% on room air.

Physical exam revealed conscious, oriented, GCS 15/15, who appeared uncomfortable and anxious. There was no orthopnea, dyspnea, increased JVD or calf tenderness/erythema. She had diminished peripheral pulsation, cold, dry extremities, with no peripheral dependent edema.

Heart sounds were muffled, with no rubs, murmurs, or gallops. Lungs were clear to auscultation bilaterally. Abdomen was lax, soft and non-tender.

Significant labs including Troponin T of [0.114 ng/ml (0.05ng/ml)], normal CK and CKMB, and creatinine of 1.8mg/dl.

ECG showed inferior Q waves and poor R in v1-v6 **(figure 1)**

Bedside Transthoracic echocardiography revealed an ejection fraction of 55-60%, mild left ventricular hypertrophy, trace amount of mitral regurgitation, flattened IVS, D shaped LV, and severe tricuspid regurgitation with EPAP=70mmHg, dilated Rt ventricle **(figure2)**.

This made acute pulmonary embolism a highly probable diagnosis, but at that time patient decided to go against medical advice and refused to do D-dimer or Computed tomography pulmonary angiogram (CTPA) as her GFR was 28 mL/min/1.73m2.

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After 24 hours she went to another hospital and when they review our suspicion of PE, D-dimer was found to be [16 ug/mL (0.5ug/ml)] and CTPA revealed extensive pulmonary emboli which straddles the bifurcation of pulmonary trunk and nearly filling and extending to right and left pulmonary arteries and their segmental branches **(figure3)**, associated with right ventricular strain. Lower extremity venous doppler study showed an echogenic thrombus in right popliteal vein.

Patient was admitted and diagnosed as massive pulmonary embolism and was started on unfractionated heparin infusion for 4 days and then NOAC(Rivaroxaban 15 mg) daily.

Coronary angiography was done before hospital discharge to rule out concomitant CAD showed atherosclerotic coronary artery disease with<30% nonsignificant calcific stenosis of proximal LAD.

Her vital signs remained stable throughout her hospital stay and she was discharged on Rivaroxaban with close outpatient follow-up.

III. DISCUSSION

Acute PE is a common disease and has a high mortality rate of nearly 30% without therapy. Mortality can be reduced by accurate diagnosis followed by effective. anticoagulant therapy. Because of nonspecific manifestations of acute PE, it can lead to an erroneous diagnosis and compromise the patient's outcome. The most common symptoms are dyspnea with or without exertion, pleuritic pain, and cough. The most common signs are tachypnea, tachycardia, and rales. In addition, the ECG is usually nonspecific, and around one-third of patients have normal findings.[4]

Here we reported a case of acute PE masquerading as ACS. ACS was suspected by elevated troponin, abnormal ECG, and typical anginal pain. However, hypoxia, shock together with echocardiography findings of normal LV systolic function, D shaped LV and flattened IVS in addition to dilated RV and elevated RV systolic pressure, kept pulmonary embolism as a highly probable differential diagnosis. In a small study of 24 patients with sub massive pulmonary embolism, 20.8% had elevated cTnI levels, and this finding is clinically relevant because pulmonary embolism and acute coronary syndromes are common diseases that can present with nonspecific and overlapping clinical features. The potential for misdiagnosis, which may be influenced by elevated troponin levels, was illustrated in a recent report in which a patient with pulmonary embolism was initially considered to have a myocardial infarction because of elevated cTnT levels and suggestive electrocardiographic findings.[5]

In patients with moderate to large pulmonary embolism 50% have elevated serum troponin levels [6] and this is of prognostic information. In a Metanalysis of twenty studies, those with an elevated troponin level (either troponin-I or troponin-T), 19.7% died versus 3.7% of those without a troponin elevation.[7]

Variable ECG findings reported in association with PE included alterations in rate, rhythm, conduction, axis and morphology with sinus tachycardia being the most common abnormality. The online literature described many examples of similar pres- entation.

The mechanism for the accompanying ECG changes is usually right ventricular strain secondary to increased pressure within the right side leading to unmatched high oxygen demand, but other accounts exist in the literature including paradoxical embolisation leading to coronary occlusion.

Microvascular coronary vasospasm induced by sudden right ven-tricular strain or hypoxaemia-induced catecholamine surge ar Variable ECG findings reported in association with PE included alterations in rate, rhythm, conduction, axis and morphology with sinus tachycardia being the most common abnormality.

The variable ECG findings reported in association with PE is usually secondary to increased pressure within the right side leading to unmatched high oxygen demand, but other accounts exist in literatures including paradoxical embolization leading to coronary occlusion. Microvascular coronary vasospasm induced by sudden right ventricular strain or hypoxemia-induced catecholamine surge. [8]

PE can mimic acute coronary syndrome or may be concomitant with it. Otherwise, the diagnosis of PE can be easily missed, and patients may not receive appropriate treatment resulting in increased mortality.[9]

IV. CONCLUSION

Acute PE can present with symptoms mimicking acute coronary syndrome and can be easily missed. This case highlights the beneficial role of bedside echocardiography together with D dimer as rapid available diagnostic tools that can support the diagnosis of PE in hemodynamically unstable patients especially when CTPA is not feasible.

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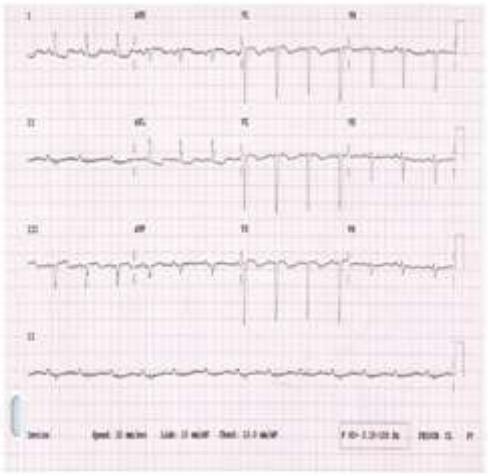

Figure (1) 12 leads ECG shows inferior Q waves and poor R in v1-v6.

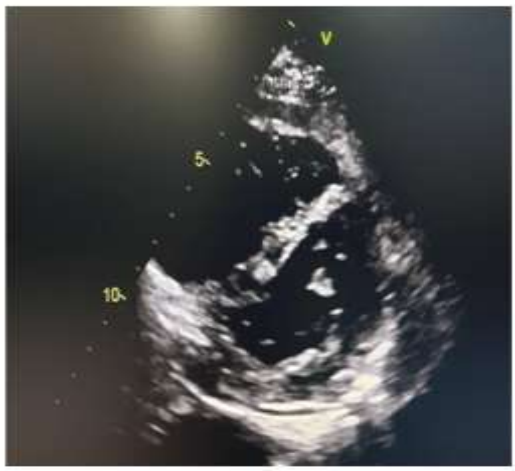

Figure (2) TTE Shows flattened IVS, D shaped LV, dilated Rt ventricle.

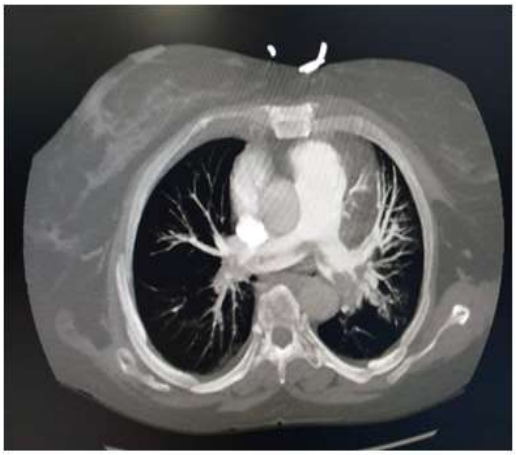

Figure (3) CT pulmonary angiography revealing saddle thrombus within the main pulmonary trunk, right and left pulmonary arteries.

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