DR ELYSE MERCIECA, DR CHANTELLE ZAMMIT, DR JAMES BORG

Pulmonary Embolism Mimicking Congestive Heart Failure

ABSTRACT

Pulmonary embolism is a life-threatening condition that can present with a wide range of non-specific symptoms, often overlapping with cardiovascular diseases. Here, we report a case of an 83-year-old female with a history of hypertension, rheumatoid arthritis and mild heart failure presenting with signs and symptoms initially suggestive of acute decompensated heart failure. Further diagnostic workup revealed that the true underlying pathology was a bilateral submassive pulmonary embolism. This case highlights the diagnostic challenges and importance of considering pulmonary embolism in patients with seemingly classic signs of congestive heart failure.

KEYWORDS

Pulmonary embolism, Congestive heart failure, Diagnostic challenges, POC echo.

INTRODUCTION

Pulmonary embolism (PE) is caused by the obstruction of the pulmonary arteries, usually by a thrombus originating from the deep veins of the legs.[1] It can present with non-specific symptoms, often mimicking other cardiopulmonary conditions such as congestive heart failure (CHF) and chronic obstructive pulmonary disease (COPD).[2,3] Distinguishing between PE and CHF is crucial, as both require distinct management approaches but may present similarly, especially in elderly patients with co-morbidities.[4]

CASE PRESENTATION

HISTORY AND PRESENTATION

An 83-year-old female presented to the emergency department (ED) in August 2024 with complaints of progressive shortness of breath and fatigue for five days. Relatives reported a gradual worsening of dyspnea, particularly on exertion, along with orthopnea and paroxysmal nocturnal dyspnea. The patient weighed 80 kg and had a history of hypertension; previous echocardiograms showed left diastolic dysfunction. Her last NT-proBNP taken in April 2024 was 222 pg/mL. She was on multiple medications including doxazosin 1 mg BD, candesartan 16 mg daily, bumetanide 1 mg daily, aspirin 75 mg daily, tramadol 50 mg x 6 daily and prednisolone 15 mg daily (the latter two for control of her rheumatoid arthritis).

Upon physical examination, the patient appeared dyspneic but was alert and oriented. Vital signs revealed:

- Heart rate: 73 beats per minute
- Blood pressure: 100/53 mmHg
- Respiratory rate: 32 breaths per minute
- Oxygen saturation: 80% on room air
- Jugular venous distention (JVD) was present, however pitting oedema of lower limbs was minimal. Facial oedema was pronounced; deemed secondary to the patient's intermittent use of 15 mg of prednisolone daily for 5 years.

Lung auscultation revealed crepitations at the lung bases bilaterally, and heart sounds were normal. Given her history and these findings, a provisional initial diagnosis of acute decompensated heart failure was made.

INITIAL INVESTIGATIONS

- Electrocardiogram (ECG): Tall R waves consistent with left ventricular hypertrophy and a right bundle branch block was present. These findings were also presented in previous ECGs.
- Chest X-ray: Cardiomegaly was visible, as well as bilateral increased interstitial lung markings in keeping with pulmonary congestion. There was no pneumothorax but there was a small left-sided pleural effusion.
- Lab tests
  - Elevated NT-proBNP: 3852 pg/mL (N: ≤ 150 pg/ml).
  - Troponin I: 88.20 pg/ml (N: ≤ 47.8 pg/ml).
  - White cell count: 14.14 x10^9/L (N: 4.3 - 11.4 x10^9/L).
  - C-reactive protein: 62 (N: 0-5 mg/L).

INITIAL MANAGEMENT

The patient was treated with oxygen therapy, with SpO2 improving to 93%. In view of the borderline hypotension, diuretics were initially withheld, and a point of care (POC) echocardiogram was requested for further assessment.

DIAGNOSTIC DILEMMA

For assurance of diagnosis of CHF exacerbation, a POC echocardiogram was performed. This revealed a grossly normal left ventricular function with right ventricular dilatation - RV/LV diameter ratio ≥1.0. There was impaired right ventricular systolic function of the RV free wall with right apical hypercontractility (McConell's sign).[5,6] The inferior vena cava (IVC) was plethoric (>2cm) with poor collapse on inspiration (<50% collapsibility).

These findings, along with the significantly reduced oxygen saturations on room air, raised the suspicion of a PE.[5-7]

A CT pulmonary angiogram (CTPA) was therefore performed, which revealed dilation of the pulmonary main trunk, measuring 3.2 cm, as well as bilateral pulmonary emboli in the distal main, segmental, and sub-segmental arteries. These findings were consistent with the diagnosis of a submassive PE.[7,8]

DISCUSSION

This case underscores the diagnostic challenges posed by PE, particularly when it mimics CHF. Both conditions can present with dyspnea, peripheral oedema, elevated BNP, and jugular venous distention, making initial differentiation difficult.[3,9] Key distinguishing features that raised suspicion for PE in this case were:

- Significantly low oxygen saturations on room air: The patient's clinical presentation was not consistent with significant fluid overload as the underlying cause for this degree of hypoxia.[9]
- Response to oxygen therapy: The patient demonstrated a positive response to oxygen therapy without the administration of diuretics.[10,11]
- Echocardiographic findings: The presence of right ventricular dilatation and McConnell's sign indicated acute right ventricular strain, indicating the possibility of a PE.[5,6]

CHF and PE share several overlapping features, especially in patients with a history of heart failure or other co-morbidities. Both conditions may result in increased pulmonary pressures, leading to right heart strain. The elevated NT-proBNP in this patient initially pointed towards CHF, but an elevated NT-proBNP can also occur in PE due to right ventricular strain.[3,9]

In this case, the misdiagnosis of CHF could have led to inappropriate management with diuretics alone, delaying the administration of life-saving anticoagulation or thrombolysis.[12] Given the large clot burden, prompt initiation of anticoagulation therapy with enoxaparin 80mg BD was critical to prevent further complications such as hemodynamic collapse or right heart failure.[13] Aspirin was discontinued, however the rest of her medications remained unchanged when she was admitted from A&E.

Thrombolysis in PE is primarily indicated for patients at high risk of mortality or hemodynamic collapse. This includes those with massive PE, characterized by hemodynamic instability such as sustained hypotension (systolic blood pressure < 90 mmHg for more than 15 minutes) or cardiogenic shock. In these cases, thrombolysis is crucial to rapidly dissolve the clot, restore pulmonary circulation, and prevent right ventricular failure and death.[1,10] Thrombolytic therapy is most commonly administered using agents like alteplase (tPA).[3] For patients with submassive PE - those with right ventricular dysfunction or myocardial injury but without hypotension - thrombolysis can be considered if there is evidence of clinical deterioration, such as worsening hypoxia, respiratory distress, or progressive right ventricular dysfunction.[14] However, its use in these cases is more controversial due to the increased risk of bleeding, and the decision must be based on a careful risk-benefit assessment.[15] Contraindications to thrombolysis include factors such as a history of hemorrhagic stroke, active bleeding, or recent major surgery, all of which increase the risk of serious bleeding complications.[16,17] In situations where PE causes cardiac arrest, thrombolysis may also be considered as part of resuscitation efforts.[18] Ultimately, thrombolysis should be used when the potential benefits outweigh the risks, particularly in life-threatening cases of PE.[19]

In this case, the patient remained hemodynamically stable, demonstrated a positive response to oxygen therapy, and exhibited no signs of clinical deterioration. As a result, treatment with low molecular weight heparin was considered the more appropriate therapeutic option.[11]

MANAGEMENT AND OUTCOME

After the diagnosis of a submassive PE, the patient was immediately started on low molecular weight heparin and was admitted on a monitored bed. Her clinical condition stabilised over the next 48 hours, with respiratory rate improving from 32 breaths/minute at initial presentation to 20 breaths/min. Oxygen saturations also stabilised to 94-96% on room air, even after mobilisation.

During admission, the patient underwent physiotherapy and occupational therapy to improve mobility and quality of life. Her treatment was also adjusted with bumetanide increased to 1 mg 1-1-0, candesartan decreased to 8 mg daily and tramadol decreased to 50 mg 1-1-2 (the latter was adjusted to decrease events of low respiratory drive). The patient was investigated for a potential trigger for a pulmonary embolus with an abdominal ultrasound and tumour markers all of which were clear.

The patient was discharged after 18 days and prescribed rivaroxaban 15 mg for a total of 21 days, which was then to be increased to 20 mg daily for 6 months, with close follow-up. A CT abdomen pelvis was done later which also excluded malignancy as a trigger for the pulmonary emboli. The patient continued to be followed up and investigated as an outpatient.

CONCLUSION

PE can closely mimic CHF, particularly in patients with pre-existing cardiovascular disease. This case highlights the importance of considering PE in the differential diagnosis of dyspnea and signs of heart failure, especially when the clinical response to standard heart failure treatment is suboptimal.[20] It also underscores the utility of POC echocardiography in distinguishing between PE and CHF.[5] Early diagnosis and management of PE are critical in preventing potentially fatal outcomes.[11]

KEY LEARNING POINTS

1. PE should be considered in patients presenting with signs and symptoms of CHF, particularly if there are significantly low oxygen saturations and/or an inadequate response to diuretic therapy which are not consistent with significant fluid overload.
2. Elevated NT-proBNP and troponin may be present in both PE and CHF due to right heart strain.
3. Right ventricular dysfunction on echocardiography should raise suspicion for PE, especially if left ventricular function is preserved or only mildly reduced.
4. POC echo has a role in differentiating between PE and CHF.
5. Prompt imaging, such as CTPA, is crucial for the definitive diagnosis of PE.

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COMPETING INTERESTS

None declared.

PATIENT CONSENT FOR PUBLICATION

Consent obtained directly from patient.

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