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Case report

Massive Pulmonary Embolism Presenting as Septic Shock Despite Repeatedly Negative D-dimer and Negative Lower Limb Doppler: A Diagnostic Challenge

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A B S T R A C T

Pulmonary embolism (PE) may present atypically and mimic septic shock, posing a significant diagnostic challenge in critically ill patients. A 45-year-old woman initially presented with symptoms suggestive of acute gastroenteritis and was discharged. She re-presented within 24 hours with hypotension, tachycardia, and hypoxemia. Laboratory findings revealed severe anemia (hemoglobin 6.5 g/dL), leukocytosis (14 ×10⁹/L), platelet count 153 ×10⁹/L, elevated lactate (7.6 mmol/L), and metabolic acidosis (pH 7.23, HCO_3^- 12 mmol/L, $PaCO_2$ 24 mmHg, PaO_2 56 mmHg). No clear infectious source was identified apart from the initial gastrointestinal presentation. Early transthoracic echocardiography demonstrated right ventricular dilatation and dysfunction suggestive of acute pressure overload. Lower limb Doppler ultrasound was negative. Computed tomography pulmonary angiography confirmed a massive saddle pulmonary embolism. Thrombolysis was withheld due to high bleeding risk. The patient improved with anticoagulation and was discharged on apixaban. Further evaluation for underlying prothrombotic conditions, including thrombophilia and rheumatologic screening, was unremarkable. This case highlights the limitations of D-dimer and Doppler ultrasound in high-risk patients and underscores the critical role of clinical judgment and early echocardiography in diagnosing PE.

Introduction

Pulmonary embolism (PE) remains a major cause of cardiovascular morbidity and mortality, particularly in cases of delayed diagnosis, where failure to promptly recognize the condition may lead to significant clinical deterioration and increased mortality [1]. Despite advances in diagnostic strategies, PE continues to pose a substantial clinical challenge due to its highly variable and often non-specific presentation. While classical manifestations include dyspnea, chest pain, and tachycardia, PE may present atypically, mimicking conditions such as septic shock or intra-abdominal pathology. This diagnostic overlap may lead to misdiagnosis and delay in appropriate management.

Current diagnostic approaches rely on structured clinical probability assessment using validated tools such as the Wells score and the revised Geneva score, followed by D-dimer testing and confirmatory imaging. However, guidelines emphasize that D-dimer testing should not be used to exclude PE in patients with high clinical probability or hemodynamic instability [1,2]. Furthermore, the diagnostic performance of D-dimer is reduced in critically ill patients, where false-negative results, although uncommon, may occur [5]. We report a case of massive PE presenting as septic shock with repeatedly negative D-dimer and negative lower limb Doppler ultrasound, highlighting the limitations of standard diagnostic pathways and emphasizing the importance of clinical judgment.

Case presentation

A 45-year-old previously healthy woman initially presented with vomiting and diarrhea and was diagnosed with presumed acute gastroenteritis and discharged. Within 24 hours, she re-presented with hypotension (BP 85/55 mmHg), tachycardia (HR 120 bpm), and hypoxemia (SpO_2 88% on room air). Laboratory evaluation revealed severe anemia (hemoglobin 6.5 g/dL), leukocytosis (14 ×10^9/L), and platelet count 153 ×10^9/L. D-dimer values were repeatedly within the normal range (0.44 and 0.42 mg/L; reference cutoff <0.5 mg/L). Lactate was markedly elevated (7.6 mmol/L). Arterial blood gas analysis demonstrated metabolic acidosis (pH 7.23, HCO_3^- 12 mmol/L, $PaCO_2$ 24 mmHg) with hypoxemia (PaO_2 56 mmHg).

Initial management targeted septic shock with intravenous fluids, broad-spectrum antibiotics, and vasopressors. However, no clear infectious source was identified apart from the initial presentation suggestive of gastroenteritis.

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Early transthoracic echocardiography was performed on hospital day 1 due to persistent hypotension disproportionate to the degree of dehydration, in the presence of lower limb edema and relatively preserved mucous membrane hydration. The study revealed preserved left ventricular function with right ventricular dilatation, reduced RV systolic function, and severe tricuspid regurgitation, consistent with acute right ventricular pressure overload. Lower limb Doppler ultrasound showed no evidence of deep vein thrombosis. Given the clinical and echocardiographic findings, pulmonary embolism was strongly suspected. Empiric anticoagulation with unfractionated heparin was initiated. Systemic thrombolysis was withheld due to severe anemia and increased bleeding risk in the context of recent gastrointestinal symptoms. Following partial stabilization, computed tomography pulmonary angiography confirmed a massive saddle pulmonary embolism. The patient improved clinically and was discharged on apixaban. Further evaluation for underlying prothrombotic conditions, including thrombophilia and rheumatologic screening, was unremarkable, suggesting an unprovoked event. Outpatient follow-up was arranged. The diagnostic reasoning pathway and key decision points are summarized in Figure 1.

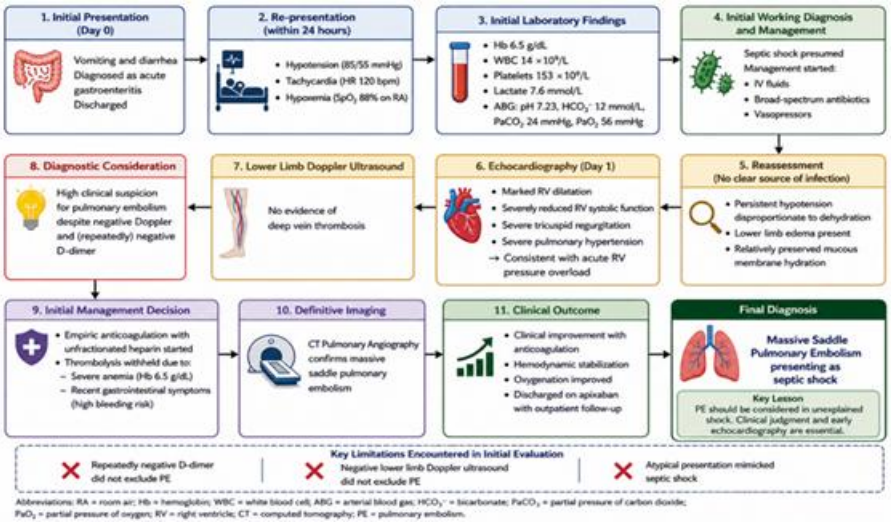

Figure 1. Diagnostic reasoning pathway illustrating the progression from initial presentation with presumed gastroenteritis to the diagnosis of massive pulmonary embolism, highlighting key clinical decision points.

(A) Apical four-chamber view showing marked right ventricular dilatation.

(B) Short-axis view showing right ventricular enlargement

Figure 2. Transthoracic echocardiography demonstrating features of acute right ventricular pressure overload. (A) Apical four-chamber view showing marked right ventricular dilatation, (B) Short-axis view showing right ventricular enlargement.

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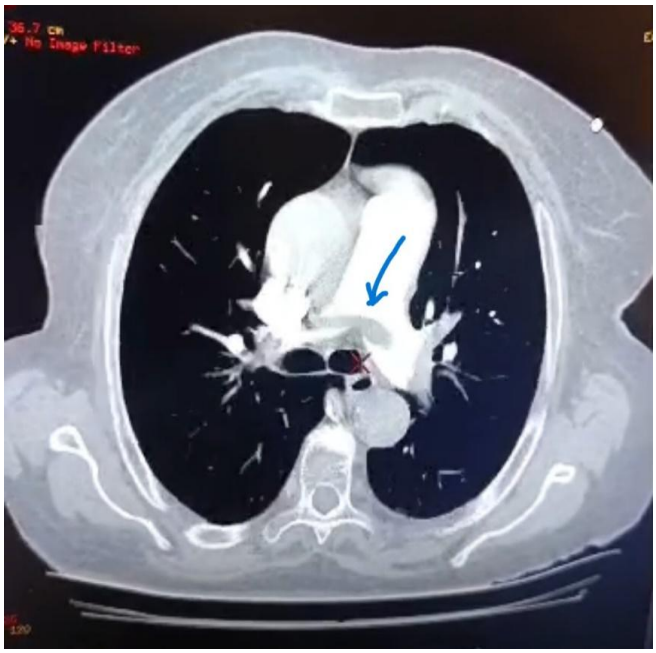

Figure 3. CT pulmonary angiography demonstrating a large saddle pulmonary embolism involving the main pulmonary arteries (arrow)

Table 1. Wells Score for Pulmonary Embolism

Clinical Feature	Points
Clinical signs of DVT	3
PE most likely diagnosis	3
Heart rate >100	1.5
Immobilization/surgery	1.5
Previous DVT/PE	1.5
Hemoptysis	1
Malignancy	1

Interpretation: >6 high, 2–6 moderate, <2 low

Table 2. Revised Geneva Score

Clinical Feature	Points
Age >65	1
Previous DVT/PE	3
Surgery/fracture	2
Active malignancy	2
Unilateral leg pain	3
Hemoptysis	2
Heart rate 75–94	3
Heart rate ≥95	5
Pain on palpation + edema	4

Interpretation: 0–3 low, 4–10

Discussion

This case underscores a critical limitation of conventional diagnostic algorithms for pulmonary embolism, particularly when applied in critically ill patients with high pretest probability. The initial presentation with gastrointestinal symptoms followed by hemodynamic instability led to a working diagnosis of septic shock. However, the absence of a clear infectious source and lack of sustained response to standard therapy necessitated reconsideration of alternative diagnoses. Clinical probability was assessed retrospectively using established scoring systems (Tables 1 and 2). In such cases, reliance on D-dimer testing is inappropriate when clinical suspicion is high, as emphasized in current guidelines [1,2].

In the present case, retrospective application of these scoring systems would classify the patient as having a high probability of pulmonary embolism, further supporting the need for immediate imaging rather than

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reliance on D-dimer testing. Although D-dimer is highly sensitive, false-negative results may occur, particularly in patients with high clinical probability or altered fibrinolytic activity. Therefore, D-dimer should not be used to exclude PE in high-risk or critically ill patients [1,5]. In the present case, D-dimer values (0.44 and 0.42 mg/L; cutoff <0.5 mg/L) were repeatedly within the normal range, highlighting the potential for false reassurance and the limitations of relying solely on D-dimer in high-risk clinical settings. The absence of deep vein thrombosis on lower limb Doppler ultrasound does not exclude PE. Emboli may originate from pelvic veins or may have already embolized, leaving no detectable thrombus.

Transthoracic echocardiography played a pivotal role by demonstrating right ventricular dysfunction consistent with acute pressure overload. In hemodynamically unstable patients, echocardiography provides rapid bedside assessment and may guide early management decisions when definitive imaging is delayed [3,4]. The decision to withhold thrombolysis reflected a careful balance between potential benefits and bleeding risk, particularly in the presence of severe anemia. This highlights the importance of individualized clinical decision-making. In similar scenarios, catheter-directed therapies may be considered where available, offering a potential balance between efficacy and bleeding risk.

Conclusion

Massive pulmonary embolism may present atypically and mimic septic shock. Negative D-dimer and Doppler ultrasound findings do not exclude PE in high-risk patients. Clinicians should maintain a high index of suspicion for PE in cases of unexplained shock. This case emphasizes the importance of prioritizing clinical judgment and bedside echocardiography over isolated diagnostic tests in critically ill patients.

Conflict of interest. Nil

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