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Case Report

Pulmonary embolism with fatigue as the main symptom: a case report

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Abstract: Pulmonary embolism (PE) presents with a wide range of symptoms, including shortness of breath, dyspnea, palpitations, chest tightness, chest pain, cough, restlessness, and syncope, which can often lead to misdiagnosis. We report a case of an 82-year-old patient who primarily complained of fatigue and was ultimately diagnosed with PE through computed tomography pulmonary angiography. After initiating anticoagulant therapy, the patient's fatigue significantly improved. This case highlights the importance of actively investigating the underlying cause of fatigue in elderly patients and raises awareness of pulmonary diseases as potential contributors. PE should be considered an important differential diagnosis in such cases.

Keywords: Pulmonary embolism, fatigue, elderly patient, case report

Introduction

Pulmonary embolism (PE) is a life-threatening cardiovascular condition with non-specific clinical manifestations, which poses significant challenges for early diagnosis and treatment [1]. Common symptoms include chest tightness, chest pain, dyspnea, hemoptysis, cough, and syncope; however, these symptoms may be absent or atypical in elderly patients, leading to high rates of misdiagnosis and missed diagnosis [2]. Although advancements in diagnostic techniques have improved detection rates, timely and accurate diagnosis remains critical for optimizing patient outcome.

The diagnostic approach for PE follows a hierarchical strategy based on clinical probability assessment. First-line screening typically involves D-dimer testing, which has high sensitivity but low specificity. A negative result effectively rules out PE in low-risk populations, while an elevated level warrants further investigation [3]. Arterial blood gas analysis may reveal hypoxemia or respiratory alkalosis, though normal results do not exclude PE, particularly in mild cases [4]. Electrocardiography (ECG) often shows non-specific changes such as sinus tachycardia, right bundle branch block, or the $S_1Q_3T_3$ pattern, but these findings lack both sensitivity and specificity [5]. Lower extremity arteriovenous ultrasound is valuable for detecting deep vein thrombosis (DVT), a major source of pulmonary emboli, though negative findings do not rule out PE risk [6].

Computed tomography pulmonary angiography (CTPA) is the first-line imaging modality for definitive diagnosis due to its high spatial resolution, wide availability, and ability to visualize emboli in pulmonary arteries [2, 3]. CTPA accurately identifies emboli in both central and peripheral vessels, making it indispensable for confirming PE. Other diagnostic tools include radionuclide pulmonary ventilation/perfusion scintigraphy, particularly useful in patients with contraindications to contrast agents (e.g., renal impairment), and invasive pulmonary angiography, once the "gold standard" but now reserved for complex cases due to its invasiveness [7, 8].

Elderly patients with PE often present with declined physical function, multiple comorbidities, and a blunted symptom response, result-

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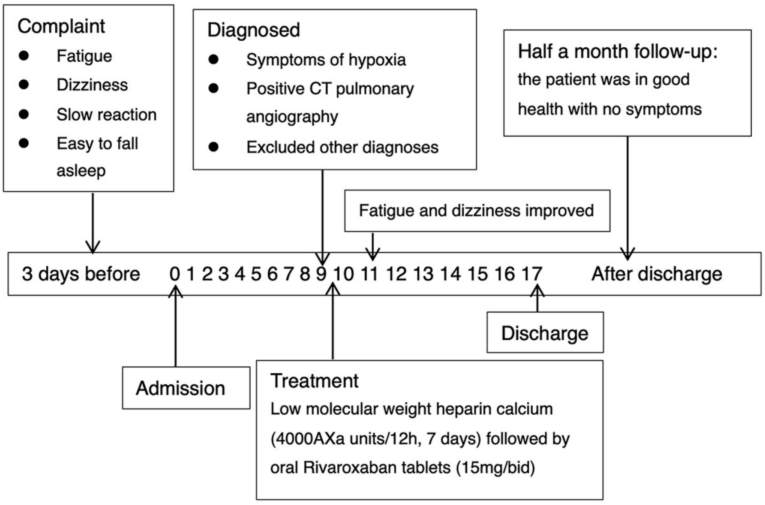

Figure 1. Timeline for important milestones.

ing in atypical presentations [9]. Fatigue, as the primary symptom of PE in elderly patients, is extremely rare and often overlooked, further complicating the diagnosis.

This case report presents an 82-year-old male patient with PE whose main complaint was fatigue. The aim is to highlight the clinical importance of recognizing non-classical symptoms in the elderly population. Specifically, it emphasizes the need for clinicians to consider PE in the differential diagnosis of unexplained fatigue in older adults, even in the absence of typical respiratory or cardiovascular symptoms. By detailing the diagnostic process and outcomes, this case contributes to raising awareness of atypical PE presentations, thereby facilitating earlier intervention.

Case presentation

The patient, an 82-year-old male, was admitted with a 3-day history of fatigue, accompanied by dizziness and excessive sleepiness. He was able to communicate and walk normally, without fever, palpitations, chest tightness, or chest pain, and had no gastrointestinal complaints.

Past medical history: The patient had a history of lacunar cerebral infarction and anxiety disorder for over 10 years. He had been on long-term medication, including buspirone hydrochloride tablets (5 mg twice daily) and duloxetine hydrochloride enteric-coated capsules (20 mg once daily). He denied any history of hypertension, diabetes, or genetic familial diseases. **Figure 1** showed timeline for important milestones.

Physical examination: The patient was alert, with a body temperature of 36.9°C, pulse rate of 66/min, respiration rate of 18/min, and blood pressure of 119/69 mmHg. His pupils were equal and reactive to light, with normal respiratory sounds in both lungs and no obvious rales. His heart rhythm was regular with no murmurs detected. Abdominal examination was negative, with no edema in the lower limbs and normal muscle strength and tone. Pathologic signs were negative. The Romberg test was positive (falling to the right), but the finger-to-nose test and rapid alternating movements test were normal.

Laboratory results: pH: 7.4; PCO_2: 39.7 mmHg; PO_2: 99.4 mmHg; Lactic acid: 1.41 mmol/L; C-reactive protein: 2.00 mg/L; White blood cell count: 6.04 × 10^9/L; Neutrophil percentage: 50.1%; Hemoglobin: 138 g/L; Platelets: 173 × 10^9/L; Creatinine: 65 μmol/L; Potassium: 3.81 mmol/L; Glucose: 5.7 mmol/L; Alanine aminotransferase: 19 U/L; Aspartate aminotransferase: 25 U/L; Troponin I: <0.002 ng/mL; Creatine kinase MB: 1.57 ng/mL; B-type natriuretic peptide: 9.7 pg/mL; D-dimer: 640 μg/L FEU (slightly elevated, normal range: 0-550 μg/L FEU); Tumor markers: within normal limits.

Imaging and examination: Electrocardiogram (ECG): Sinus rhythm with first-degree atrioventricular block and frequent premature atrial contractions with biphasic rhythm.

Chest CT scan: Revealed small airway lesions in both lungs, several small nodules in the right lung, and local calcification of the coronary arteries.

Echocardiography: Mild mitral and tricuspid regurgitation.

Intracranial magnetic resonance angiography: No significant abnormalities detected.

Lower limb arterial and venous ultrasound: Showed arterial sclerosis with plaque formation in both lower limbs.

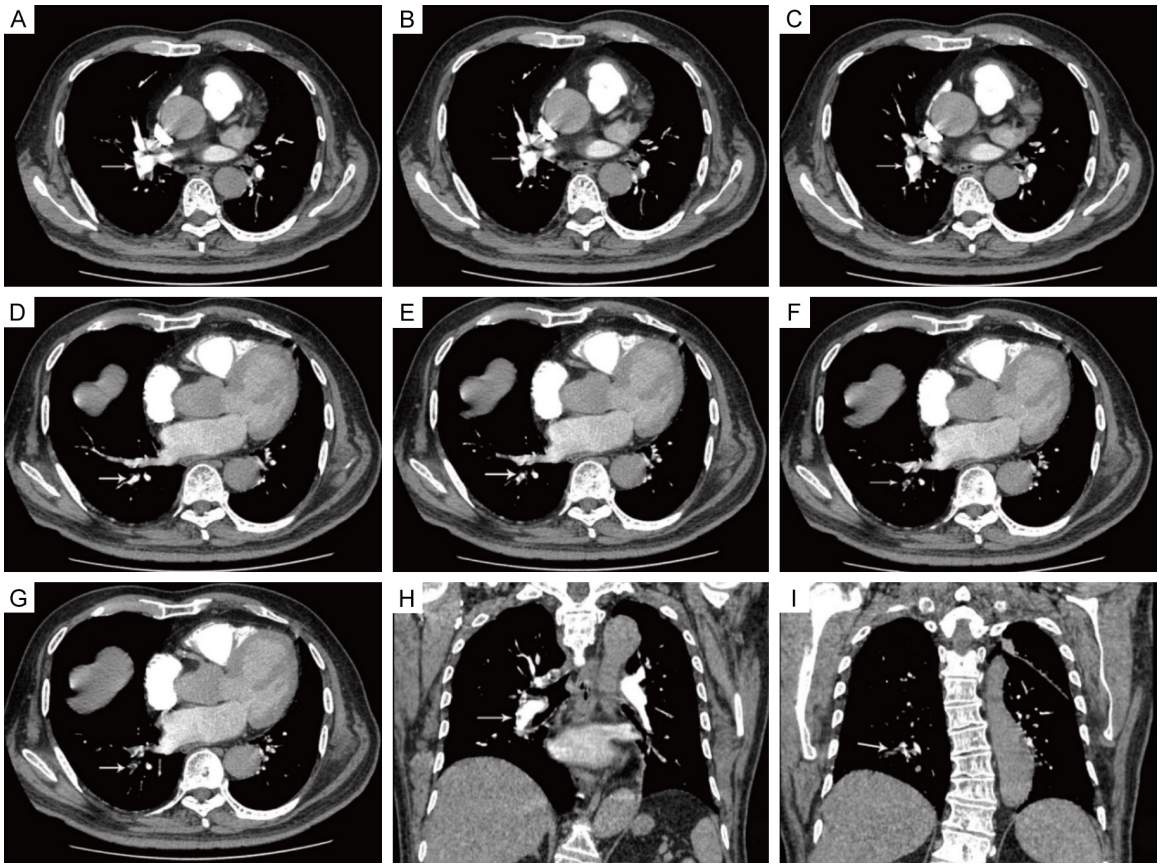

Figure 2. CT pulmonary angiography. Partial embolism of the main pulmonary artery in the lower lobe of the right lung (A-C and H); Pulmonary embolism in the secondary and tertiary branches of the lower lobe of the right lung (D-G and I).

CTPA: Confirmed pulmonary embolism in the main trunk and secondary/tertiary branches of the right lower lobe pulmonary artery (**Figure 2**).

Treatment

The patient was admitted with the primary complaint of "fatigue for 3 days, accompanied by dizziness and excessive somnolence". Upon admission, vital signs were stable (temperature 36.9°C, pulse 66/min, respiration 18/min, blood pressure 119/69 mmHg). A positive Romberg test (falling to the right) was observed, along with a slightly elevated D-dimer (640 μg/L FEU). Blood gas analysis, routine blood tests, cardiac markers, and imaging (chest CT, echocardiography) were normal, ruling out acute cerebrovascular events and lower extremity DVT. Given the unclear etiology of the fatigue, symptomatic treatment with gastrodin injection was initiated.

Despite this treatment, on day 8 of hospitalization, daily monitoring showed persistent fatigue (no improvement in activity tolerance), ongoing dizziness (2-3 episodes daily upon position changes), and easy somnolence (prolonged daytime naps). Vital signs remained stable, with no new symptoms. A repeat D-dimer test (620 μg/L FEU) and lower extremity ultrasound still excluded DVT. The patient continued pre-admission anti-anxiety medications (buspirone hydrochloride and duloxetine hydrochloride) without adverse interactions.

On hospital day 9, due to the persistence of symptoms, the patient's advanced age (82 years), and his history of lacunar infarction (a risk factor for PE), a CTPA confirmed PE in the main trunk and secondary/tertiary branches of the right lower lobe pulmonary artery. Anticoagulant therapy with subcutaneous low-molecular-weight heparin calcium (0.4 ml, 4000 AXa units every 12 hours) was initiated. By day 11, the patient's fatigue improved (he could walk 100 meters continuously), dizziness and somnolence were alleviated, and no bleeding oc-

curred. Coagulation function remained normal throughout the treatment.

On day 16, after patient education on medication administration (with meals) and bleeding prevention, the patient transitioned to oral rivaroxaban (15 mg twice daily), which he tolerated well. On day 17, the patient was discharged. A comprehensive assessment showed complete resolution of fatigue, dizziness, and somnolence, a negative Romberg test, and stable vital signs (temperature 36.8-37.0°C, pulse 65-68 beats/min, respiration 17-19 breaths/min, blood pressure 118-122/68-70 mmHg). Medication transition was smooth, and the patient was instructed to continue rivaroxaban for 3 months, follow up in two weeks, and seek medical attention for any new symptoms.

At the follow-up visit, the patient reported no recurrence of symptoms, a normal D-dimer level (420 μg/L FEU), and expressed satisfaction with the treatment outcome, indicating effective control of PE.

Discussion

With the growing understanding of PE among clinicians and the advancement of diagnostic technology, the detection rate of PE has gradually increased. However, misdiagnosis remains common, which can worsen early diagnosis, treatment, and patient prognosis [10]. The symptoms of PE are diverse and non-specific, ranging from asymptomatic in mild cases to sudden death in severe cases. Common manifestations include dyspnea, palpitations, chest tightness, chest pain, cough, and syncope, which involve the respiratory, cardiovascular, and cerebrovascular systems. The typical "triad" of pulmonary infarction - chest pain, hemoptysis, and dyspnea - is present in only about 20% of patients, making clinical identification difficult [11]. PE is often misdiagnosed as pneumonia, coronary artery disease, or stroke, and early diagnosis remains challenging [12].

Auxiliary examinations commonly used in clinical practice include D-dimer, blood gas analysis, ECG, lower limb arteriovenous ultrasound, cardiac ultrasound, and chest CT. Currently, CTPA is the most widely used diagnostic method, while radionuclide pulmonary ventilation/perfusion imaging and pulmonary angiography are also employed. A negative D-dimer test is valuable for excluding PE, while an elevated D-dimer is not diagnostic [13]. Blood gas analysis may show hypoxemia in PE patients, though some may present with normal results. Similarly, ECG and chest CT are not specific for PE diagnosis [14]. Lower limb arteriovenous ultrasound can detect DVT, and cardiac ultrasound can assess right ventricular function and pulmonary artery pressure, which are indicative of PE. However, mild cases may show no abnormalities on ultrasound.

Elderly patients are at high risk for PE due to numerous factors, including advanced age, smoking, fracture surgery, prolonged bed rest or immobilization, varicose veins, stroke, myocardial infarction, malignancies, hyperhomocysteinemia, and obesity. The rates of misdiagnosis and missed diagnosis are higher in this population due to physical decline, multiple comorbidities, reduced responsiveness, and atypical clinical presentations [15, 16].

This elderly patient presented primarily with fatigue, accompanied by dizziness, slow responses, and excessive somnolence, with a positive Romberg test. There was no lower limb edema. Blood gas analysis, routine blood tests, and biochemistry tests showed no significant abnormalities. D-dimer was slightly elevated, but lower extremity ultrasound, echocardiography, and ECG did not suggest PE. Intracranial magnetic resonance angiography also showed no significant abnormalities, ruling out many potential conditions. Ultimately, PE was confirmed through CTPA. The diagnostic process was prolonged, taking 9 days, which is relatively long. However, timely treatment was initiated, and no significant adverse effects were observed. At the follow-up visit after discharge, the patient was satisfied with his recovery. A limitation of this case is that CTPA was not performed after discharge.

Based on the patient's clinical characteristics, several potential causes of his PE could be identified: the patient's advanced age contributes to decreased vascular elasticity, increased blood viscosity, and an imbalance in the coagulation-anticoagulation system, which raises the risk of thrombosis; the lower extremity vascular ultrasound showed arteriosclerosis with plaques, leading to luminal stenosis and slowed blood flow, providing a pathologic basis for

thrombosis; and his history of lacunar cerebral infarction suggested underlying vascular lesions or abnormal coagulation mechanisms, further increasing the risk of thromboembolism. Although no DVT or clear predisposing factors, such as recent surgery or malignancy, were identified, the interplay of advanced age, vascular sclerosis, and potential vascular lesions created the pathophysiologic basis for PE in this case.

In conclusion, PE has become a common condition. Clinicians must be vigilant in excluding PE when diagnosing heart, brain, and lung diseases, particularly in elderly patients. If a patient presents with unexplained fatigue, lung disease, including PE, should be considered as a possible diagnosis to reduce the risk of misdiagnosis and missed diagnosis.

Disclosure of conflict of interest

None.

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References

[1] Westafer LM, Long B and Gottlieb M. Managing pulmonary embolism. Ann Emerg Med 2023; 82: 394-402.
[2] Trott T and Bowman J. Diagnosis and management of pulmonary embolism. Emerg Med Clin North Am 2022; 40: 565-581.
[3] Rošić D, Kočet N, Simić A, Prkačin I and Nesek Adam V. Diagnosis of pulmonary embolism in the emergency department. Acta Clin Croat 2022; 61: 33-37.
[4] Opitz CF and Meyer FJ. Pulmonary embolism: an update based on the revised AWMF-S2k guideline. Hamostaseologie 2024; 44: 111-118.
[5] Duffett L, Castellucci LA and Forgie MA. Pulmonary embolism: update on management and controversies. BMJ 2020; 370: m2177.
[6] Huang G, Tang Y, Wang H, Xiong R and Xu A. Lower limb vein thrombosis-induced pulmonary embolism and paradoxical multiple arterial embolisms: a case report with a 10-year follow-up. Medicine (Baltimore) 2019; 98: e16522.
[7] Bonnefoy PB, Prevot N, Mehdipoor G, Sanchez A, Lima J, Font L, Gil-Díaz A, Llamas P, Aibar J, Bikdeli B, Bertoletti L and Monreal M. Ventilation/perfusion (V/Q) scanning in contemporary patients with pulmonary embolism: utilization rates and predictors of use in a multinational study. J Thromb Thrombolysis 2022; 53: 829-840.
[8] Bae JY and Murugiah K. Invasive pulmonary angiogram performance and interpretation in the diagnosis of pulmonary thromboembolic disease. Interv Cardiol Clin 2023; 12: 299-307.
[9] Robert-Ebadi H and Righini M. Diagnosis and management of pulmonary embolism in the elderly. Eur J Intern Med 2014; 25: 343-349.
[10] Natour HH, Elgardt I, Rotman-Pikielny P, Natour A, Natour A and Levy Y. Masquerading pulmonary embolism: fever as an overlooked clinical manifestation illustrated by four mini case series and literature review. Am J Med 2025; 138: 1124-1127.
[11] National Project of the Diagnosis and Treatment Strategies for Pulmonary Thromboembolism (NATSPUTE). The clinical features of 516 patients with acute pulmonary thromboembolism. Zhonghua Yi Xue Za Zhi 2006; 86: 2161-2165.
[12] Yang X, Luo J and Chen J. Pulmonary artery sarcoma masquerading as refractory pulmonary embolism: a case report. Sci Prog 2025; 108: 368504251330987.
[13] Robert-Ebadi H, Combescure C, Bulla O, Righini M and Fontana P. Different D-dimer assays with age-adjusted cutoffs to exclude pulmonary embolism: secondary analysis of ADJUST-PE study. J Thromb Haemost 2025; 23: 2531-2539.
[14] Betjes MG, Corssmit EP and van der Sloot JA. Chest pain and electrocardiogram abnormalities: consider pulmonary embolism. Ned Tijdschr Geneeskd 1998; 142: 497-502.
[15] Weinstein T, Deshwal H and Brosnahan SB. Advanced management of intermediate-high risk pulmonary embolism. Crit Care 2021; 25: 311.
[16] Piazza G. Advanced management of intermediate- and high-risk pulmonary embolism: JACC Focus Seminar. J Am Coll Cardiol 2020; 76: 2117-2127.