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A Rare Presentation Of Pulmonary Embolism Diagnosed By Computed Tomography Pulmonary Angiography: A Case Report

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ABSTRACT:
Pulmonary embolism (PE) is a potentially fatal cardiovascular emergency characterized by obstruction of the pulmonary arterial circulation, most commonly due to thromboembolic disease. Because its clinical presentation is often nonspecific and may mimic several cardiopulmonary disorders, early diagnosis remains challenging. Computed tomography pulmonary angiography (CTPA) has become the gold standard imaging modality for confirming the diagnosis and guiding prompt management. We report the case of a 46-year-old male who presented with sudden-onset dyspnea, pleuritic chest pain, tachycardia, and hypoxemia without obvious clinical evidence of deep vein thrombosis. Initial laboratory investigations revealed markedly elevated D-dimer levels, while arterial blood gas analysis demonstrated hypoxemia with respiratory alkalosis. Electrocardiography showed sinus tachycardia with features of right ventricular strain, and echocardiography demonstrated mild right ventricular dilatation. CTPA revealed acute bilateral pulmonary embolism involving the right main pulmonary artery with extension into the lobar and segmental branches, along with additional emboli in the left lower lobe pulmonary artery and evidence of mild right ventricular strain. The patient was classified as having intermediate-risk (submassive) pulmonary embolism and was managed with oxygen supplementation, low-molecular-weight heparin, followed by oral anticoagulation. He demonstrated progressive clinical improvement, was discharged in stable condition, and remained asymptomatic during follow-up with near-complete thrombus resolution on repeat imaging. This case highlights the importance of maintaining a high index of suspicion for pulmonary embolism in patients presenting with unexplained acute respiratory symptoms, even in the absence of classical thromboembolic risk factors. CTPA plays a pivotal role in establishing an early and accurate diagnosis, determining thrombus burden, and assessing right ventricular involvement. Prompt recognition and timely anticoagulant therapy are essential for reducing morbidity and mortality. Increased awareness of atypical presentations can facilitate early diagnosis, optimize treatment, and improve overall clinical outcomes.

INTRODUCTION:

Pulmonary embolism (PE) is a potentially life-threatening cardiovascular emergency caused by obstruction of the pulmonary arterial circulation, most commonly by thrombi originating from the deep veins of the lower extremities. It represents the third most common acute cardiovascular syndrome after myocardial infarction and stroke, contributing substantially to global morbidity, mortality, and healthcare expenditure. Despite remarkable advances in diagnostic imaging and therapeutic strategies, PE continues to pose a significant clinical challenge because of its highly variable and often nonspecific clinical presentation. Patients may present with sudden-onset dyspnea, pleuritic chest pain, tachycardia, syncope, hemoptysis, or circulatory collapse, while others remain minimally symptomatic or exhibit atypical manifestations that closely resemble pneumonia, acute coronary syndrome, asthma exacerbation, or musculoskeletal chest pain. Consequently, delayed diagnosis remains common and is associated with increased mortality and adverse clinical outcomes.[1,2]

The pathophysiology of pulmonary embolism involves migration of thrombotic material into the pulmonary arterial tree, leading to mechanical obstruction of pulmonary blood flow, increased pulmonary vascular resistance, right ventricular pressure overload, impaired gas exchange, and, in severe cases, cardiogenic shock. The extent of hemodynamic compromise depends on clot burden, pre-existing cardiopulmonary reserve, and the patient's physiological response. Numerous predisposing factors have been identified, including prolonged immobilization, recent surgery, malignancy, pregnancy, inherited thrombophilia, obesity, advanced age, trauma, and previous venous thromboembolism. Nevertheless, a considerable proportion of patients develop PE without obvious provoking factors, making clinical suspicion essential for timely diagnosis and management.[3,4]

Early recognition of pulmonary embolism relies on a combination of clinical probability assessment, laboratory investigations, and appropriate imaging. Validated clinical prediction tools such as the Wells score and the revised Geneva score help stratify patients according to pre-test probability, while D-dimer testing is valuable for excluding PE in low-risk individuals. However, imaging remains the cornerstone for definitive diagnosis. Over the past two decades, computed tomography pulmonary angiography (CTPA) has become the imaging modality of choice because of its high sensitivity, specificity, rapid acquisition, and widespread availability. CTPA allows direct visualization of intraluminal filling defects within the pulmonary arteries while simultaneously providing valuable information regarding clot location, clot burden, right ventricular enlargement, pulmonary infarction, pleural abnormalities, and alternative thoracic diagnoses. These capabilities have significantly improved diagnostic accuracy and facilitated prompt therapeutic decision-making in patients with suspected PE.[5–7]

Radiologists play a pivotal role in identifying pulmonary embolism, particularly in patients with atypical clinical features where the diagnosis may not initially be suspected. Advances in multidetector CT technology have enabled improved spatial resolution, faster image acquisition, and enhanced visualization of segmental and subsegmental pulmonary arteries. Moreover, standardized reporting of CTPA findings, including assessment of right ventricular strain and associated thoracic abnormalities, has become increasingly important for risk stratification and prognostication. Contemporary international guidelines continue to recommend CTPA as the first-line imaging investigation for hemodynamically stable patients with suspected acute pulmonary embolism, emphasizing its central role in modern diagnostic algorithms.[2,8,9]

Although pulmonary embolism is frequently encountered in emergency medicine, unusual clinical presentations continue to challenge physicians and radiologists alike. Atypical manifestations may delay appropriate imaging, resulting in missed or late diagnosis with potentially fatal consequences. Reporting such uncommon cases is therefore essential to increase clinical awareness, highlight the diagnostic value of CTPA, and reinforce the importance of maintaining a high index of suspicion even in patients without classical symptoms or obvious risk factors. The present case describes a rare presentation of acute pulmonary embolism that was successfully diagnosed by computed tomography pulmonary angiography, underscoring the indispensable role of timely radiological evaluation in achieving an accurate diagnosis and facilitating appropriate management before the onset of life-threatening complications.

CASE PRESENTATION:

Patient Demographics

A 46-year-old previously healthy male presented to the emergency department with acute onset shortness of breath and pleuritic chest pain of one day's duration. He was a non-smoker with no known history of chronic

cardiopulmonary disease, diabetes mellitus, hypertension, or previous thromboembolic events. The patient was employed as an office executive, leading a predominantly sedentary lifestyle involving prolonged periods of sitting. There was no significant family history of venous thromboembolism, inherited coagulation disorders, or premature cardiovascular disease. On presentation, he appeared anxious and was in mild respiratory distress but remained conscious, alert, and oriented.

Chief Complaints

The patient complained of sudden-onset breathlessness that progressively worsened over the preceding 24 hours. The dyspnea was associated with sharp, right-sided pleuritic chest pain that increased during deep inspiration and coughing. He also reported palpitations and generalized fatigue but denied fever, productive cough, wheezing, hemoptysis, syncope, orthopnea, or lower limb swelling. The severity of breathlessness prompted him to seek emergency medical attention.

Timeline of Illness

Approximately one day before hospital admission, the patient experienced sudden onset of mild breathlessness while at work, which he initially attributed to fatigue. Over the following several hours, the dyspnea progressively intensified, accompanied by pleuritic chest pain and increasing difficulty performing routine activities. There was no history of preceding respiratory tract infection, chest trauma, or strenuous physical activity. Despite taking over-the-counter analgesics, his symptoms persisted and worsened overnight. On the day of admission, increasing respiratory discomfort and persistent chest pain led him to present to the emergency department. Initial clinical evaluation raised suspicion for an acute cardiopulmonary event, and an urgent diagnostic workup was initiated.

Relevant Medical History

The patient had no previous history of pulmonary embolism, deep vein thrombosis, ischemic heart disease, chronic obstructive pulmonary disease, bronchial asthma, tuberculosis, or malignancy. He had not undergone any recent surgical procedures and had no history of prolonged hospitalization or immobilization. There was no recent history of COVID-19 infection or other systemic inflammatory illnesses. He was not receiving anticoagulant therapy, hormonal medications, or any long-term prescription drugs. There was no documented history of inherited thrombophilia or autoimmune disorders. His immunization status was appropriate, and previous routine medical evaluations had been unremarkable.

Risk Factors

Further history revealed that the patient had undertaken a continuous long-distance road journey lasting approximately 12 hours three days before symptom onset, during which he had minimal physical activity and infrequent breaks. His sedentary occupation, prolonged immobilization during travel, and mildly elevated body mass index (BMI: 28.1 kg/m²) were identified as potential predisposing factors for venous thromboembolism. However, he had no history of smoking, alcohol abuse, malignancy, recent fractures, pregnancy-related risk factors, central venous catheterization, or previous thromboembolic disease. The absence of multiple conventional risk factors made the diagnosis less clinically apparent and contributed to the atypical nature of the presentation.

Initial Clinical Assessment

On arrival, the patient's vital signs revealed tachycardia with a heart rate of 118 beats/min, blood pressure of 118/74 mmHg, respiratory rate of 28 breaths/min, oxygen saturation of 88% on room air, and body temperature of 98.4°F (36.9°C). Supplemental oxygen improved oxygen saturation to 95%. Physical examination demonstrated mild respiratory distress with use of accessory muscles of respiration. Chest auscultation revealed mildly reduced air entry at the right lung base without significant wheeze or crackles. Cardiac examination showed tachycardia with regular rhythm and no audible murmurs. There was no evidence of raised jugular venous pressure or peripheral edema. Examination of both lower limbs did not reveal obvious calf tenderness, erythema, or swelling suggestive of deep vein thrombosis. Based on the combination of acute dyspnea, pleuritic chest pain, unexplained hypoxemia, and tachycardia, pulmonary embolism was considered among the leading differential diagnoses. The patient's Wells score indicated an intermediate clinical probability for pulmonary embolism, prompting immediate laboratory evaluation and radiological assessment, including computed tomography pulmonary angiography (CTPA), which subsequently established the definitive diagnosis.

CLINICAL HISTORY:

Present Illness

A 46-year-old male presented to the emergency department with complaints of sudden-onset breathlessness and

sharp right-sided pleuritic chest pain that had progressively worsened over the preceding 24 hours. The dyspnea initially developed while he was at work and gradually increased in severity, limiting his ability to perform routine daily activities. The chest pain was non-radiating, aggravated by deep inspiration and coughing, and partially relieved by rest. The patient also complained of palpitations, generalized weakness, and mild dizziness but denied fever, productive cough, wheezing, hemoptysis, syncope, orthopnea, or paroxysmal nocturnal dyspnea. There was no preceding history of upper respiratory tract infection or recent trauma. Because of persistent respiratory distress and worsening chest discomfort, he sought emergency medical attention.

Past Medical History

The patient had no known history of hypertension, diabetes mellitus, ischemic heart disease, chronic obstructive pulmonary disease, bronchial asthma, tuberculosis, chronic kidney disease, liver disease, or malignancy. He had never experienced previous episodes of deep vein thrombosis or pulmonary embolism and had no documented history of inherited thrombophilia or coagulation disorders. There was no history of autoimmune disease, chronic inflammatory disorders, or recent COVID-19 infection. Routine health examinations performed in previous years had been within normal limits, and he had no known allergies to medications or contrast agents.

Surgical History

The patient had no history of major surgical procedures, orthopedic interventions, abdominal or thoracic surgery, or vascular procedures. He denied any recent hospitalization, prolonged postoperative immobilization, trauma, fractures, or intensive care admission within the previous year. There was no history of central venous catheter placement or invasive procedures that could have increased the risk of venous thromboembolism.

Drug History

The patient was not receiving any regular prescription medications before admission. He denied the use of anticoagulants, antiplatelet agents, corticosteroids, hormonal therapy, or chemotherapeutic drugs. He had taken only over-the-counter analgesics after the onset of chest pain, without significant symptomatic relief. There was no history of recreational drug use, herbal medication consumption, or recent vaccination-associated complications. He also denied any previous adverse drug reactions or hypersensitivity to iodinated contrast media.

Family History

There was no significant family history of venous thromboembolism, pulmonary embolism, deep vein thrombosis, inherited thrombophilia, recurrent miscarriages, or coagulation disorders. The patient's parents were alive without any known history of premature cardiovascular disease or thrombotic events. None of his first-degree relatives had experienced stroke, myocardial infarction at a young age, or unexplained sudden cardiac death. The family history was therefore considered non-contributory to the current illness.

Social History

The patient was employed as an office executive, requiring prolonged sitting for approximately 8–10 hours daily with minimal physical activity. His lifestyle was predominantly sedentary, and he performed only occasional recreational exercise. He was a non-smoker and denied alcohol consumption or use of illicit substances. His dietary habits were mixed, and his body mass index was 28.1 kg/m², indicating overweight status. He lived with his family and was independent in all activities of daily living before the onset of the current illness.

Thromboembolic Risk Factors

Detailed evaluation identified several acquired risk factors for venous thromboembolism. The patient had completed a continuous 12-hour road journey three days before symptom onset, during which he remained seated for prolonged periods with minimal movement or scheduled breaks. His sedentary occupation and overweight status further increased the likelihood of venous stasis. However, he had no history of active malignancy, pregnancy-related risk factors, oral contraceptive or hormone replacement therapy, recent surgery, trauma, prolonged hospitalization, chronic inflammatory disease, nephrotic syndrome, inherited thrombophilia, or previous thromboembolic episodes. The relatively limited number of conventional risk factors and the absence of lower-limb symptoms made the diagnosis clinically challenging, emphasizing the importance of maintaining a high index of suspicion and utilizing computed tomography pulmonary angiography (CTPA) for definitive diagnosis.

PHYSICAL EXAMINATION:

General Examination
On admission, the patient was conscious, alert, and oriented to time, place, and person. He appeared anxious and was in mild to moderate respiratory distress because of acute breathlessness. The patient was able to speak only in short sentences due to dyspnea. He was afebrile, well-nourished, and moderately built, with a body mass index (BMI) of 28.1 kg/m². Mild central cyanosis was noted, reflecting hypoxemia, while peripheral cyanosis was absent. There was no pallor, icterus, clubbing, generalized lymphadenopathy, or pedal edema. Jugular venous pressure was not elevated, and there were no signs of dehydration or fluid overload.

Vital Signs
At the time of initial evaluation, the patient's vital parameters demonstrated evidence of acute cardiopulmonary compromise. His heart rate was 118 beats/min, regular in rhythm, indicating sinus tachycardia. Blood pressure was 118/74 mmHg, suggesting preserved hemodynamic stability. The respiratory rate was 28 breaths/min, consistent with tachypnea. Oxygen saturation measured by pulse oximetry was 88% on room air, which improved to 95% with supplemental oxygen administered via face mask at 5 L/min. His body temperature was 36.9°C (98.4°F), excluding significant febrile illness. These findings were suggestive of acute hypoxemic respiratory compromise without evidence of circulatory shock.

Respiratory Findings
Inspection of the chest revealed increased respiratory effort with mild use of accessory muscles of respiration. Chest expansion was symmetrical bilaterally, with no visible deformity or intercostal retractions. Trachea was centrally placed. On palpation, tactile vocal fremitus was preserved bilaterally. Percussion notes were predominantly resonant throughout both lung fields. Auscultation demonstrated mildly reduced breath sounds over the right lower lung zone with occasional fine inspiratory crackles at the right lung base. No wheezing, rhonchi, or pleural friction rub was appreciated. The overall respiratory examination showed relatively subtle findings despite significant hypoxemia, a feature commonly encountered in acute pulmonary embolism.

Cardiovascular Findings
Cardiovascular examination revealed sinus tachycardia with a regular pulse rhythm. Peripheral pulses were palpable, symmetrical, and of normal volume in all four extremities. Heart sounds (S1 and S2) were audible and normal, without additional heart sounds, murmurs, rubs, or gallops. There was no clinical evidence of right-sided heart failure, including elevated jugular venous pressure, hepatomegaly, or peripheral edema. Capillary refill time was less than two seconds, indicating adequate peripheral perfusion. Although the patient remained hemodynamically stable, the presence of persistent tachycardia and unexplained hypoxemia raised strong suspicion of an acute pulmonary vascular event.

Peripheral Examination
Examination of the extremities demonstrated warm skin with normal peripheral perfusion and intact peripheral pulses. There was no pedal edema, cyanosis of the extremities, varicose veins, skin discoloration, or evidence of chronic venous insufficiency. Neurological examination of both upper and lower limbs was unremarkable, with preserved muscle strength and normal sensory function. No signs of peripheral vascular disease or connective tissue disorders were identified. The absence of significant peripheral findings did not exclude the possibility of venous thromboembolism.

Signs of Deep Vein Thrombosis (DVT)
A meticulous examination of both lower limbs was performed to identify possible evidence of deep vein thrombosis. There was no unilateral calf swelling, localized tenderness, erythema, increased skin temperature, or pitting edema. Calf circumference measurements were symmetrical, and there was no palpable cord along the course of the deep veins. Homan's sign was negative bilaterally, although its diagnostic value is recognized to be limited. Despite the absence of overt clinical features suggestive of DVT, occult lower-extremity venous thrombosis could not be excluded, as a substantial proportion of patients with acute pulmonary embolism may have clinically silent deep vein thrombosis. Consequently, the combination of unexplained tachycardia, hypoxemia, pleuritic chest pain, and intermediate clinical probability warranted urgent diagnostic imaging with computed tomography pulmonary angiography, which subsequently confirmed the diagnosis of acute pulmonary embolism.

LABORATORY INVESTIGATIONS

Complete Blood Count (CBC)
The complete blood count was largely within normal limits, with a hemoglobin level of 13.8 g/dL, total leukocyte count of 8,900/mm³, and platelet count of 2.45 × 10^5/mm³. There was no evidence of significant anemia, leukocytosis, or thrombocytopenia.

D-dimer
Serum D-dimer was markedly elevated (>5,000 ng/mL FEU), strongly suggesting active thrombus formation and supporting the clinical suspicion of venous thromboembolism.

Arterial Blood Gas (ABG)
Arterial blood gas analysis demonstrated hypoxemia with mild respiratory alkalosis (pH 7.47, PaO_2 62 mmHg, $PaCO_2$ 32 mmHg, HCO_3^- 23 mmol/L), consistent with acute pulmonary embolism.

Coagulation Profile
Coagulation parameters, including prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR), were within normal limits prior to initiation of anticoagulant therapy.

Cardiac Biomarkers
Serum high-sensitivity Troponin-I was mildly elevated, indicating minor right ventricular myocardial strain. B-type natriuretic peptide (BNP) was also modestly elevated, suggesting right ventricular pressure overload.

Electrocardiography (ECG)
Electrocardiography revealed sinus tachycardia (118 beats/min) with T-wave inversions in leads V1–V3 and an S1Q3T3 pattern, findings suggestive of acute right heart strain secondary to pulmonary embolism.

Chest X-ray
Chest radiography showed no focal consolidation or pleural effusion. Mild elevation of the right hemidiaphragm was noted, with no significant abnormality to explain the patient's severe hypoxemia.

Echocardiography
Transthoracic echocardiography demonstrated mild right ventricular dilatation with preserved left ventricular systolic function. Mild tricuspid regurgitation and elevated pulmonary artery systolic pressure were noted, indicating right ventricular strain without hemodynamic instability. These findings supported the diagnosis of intermediate-risk acute pulmonary embolism.

RADIOLOGICAL FINDINGS:
Computed Tomography Pulmonary Angiography (CTPA) Findings
Computed tomography pulmonary angiography (CTPA) was performed using a multidetector CT scanner following intravenous administration of iodinated contrast material. The examination demonstrated multiple intraluminal contrast filling defects within the pulmonary arterial system, confirming the diagnosis of acute pulmonary embolism. The thrombi were visualized as low-attenuation filling defects surrounded by contrast, producing the characteristic "polo mint" and "railway track" signs in axial images. No evidence of chronic thromboembolic disease, vascular calcification, or pulmonary artery aneurysm was identified.

Site and Extent of Thrombus
The embolus involved the right main pulmonary artery, extending into the right upper, middle, and lower lobar arteries, with additional thrombi involving several segmental branches of the right lower lobe. Smaller emboli were also identified within segmental branches of the left lower lobe pulmonary artery. There was no evidence of complete occlusion of the main pulmonary trunk. The distribution was consistent with bilateral acute pulmonary embolism, with greater thrombus burden on the right side.

Filling Defects
CTPA demonstrated well-defined central and eccentric intraluminal filling defects within the affected pulmonary arteries, partially surrounded by contrast medium. The emboli resulted in partial luminal obstruction without complete vascular occlusion. Distal pulmonary arterial opacification was mildly reduced in the involved segments,

while the remaining pulmonary arterial branches were normally opacified.

Right Ventricular Strain
Evidence of mild right ventricular strain was present on CTPA. The right ventricular-to-left ventricular (RV/LV) diameter ratio was approximately 1.1, indicating mild right ventricular dilatation. Mild flattening of the interventricular septum was noted without bowing toward the left ventricle. The main pulmonary artery was mildly prominent, while no pericardial effusion or significant contrast reflux into the inferior vena cava or hepatic veins was observed.

Pulmonary Infarction
A small peripheral wedge-shaped subpleural area of ground-glass opacity with focal consolidation was identified in the posterior basal segment of the right lower lobe, compatible with early pulmonary infarction. No cavitation or lung abscess formation was seen. Minimal adjacent pleural reaction was present.

Associated Lung Findings
Both lungs were otherwise well expanded without evidence of focal pneumonia, pulmonary edema, emphysema, pneumothorax, or significant pleural effusion. Mild dependent basal atelectatic changes were present in the right lower lobe. Mediastinal structures appeared normal, and no mediastinal lymphadenopathy or pulmonary mass lesion was detected.

Image Reconstruction Views
Multiplanar reconstruction (MPR), maximum intensity projection (MIP), and three-dimensional volume-rendered (3D VR) images were generated to improve visualization of the pulmonary arterial tree. These reconstructed images clearly demonstrated the extent and distribution of thromboembolic occlusion within the bilateral pulmonary arteries and facilitated accurate assessment of thrombus burden and right ventricular strain. The reconstructed images significantly enhanced diagnostic confidence and aided in therapeutic planning.

DIFFERENTIAL DIAGNOSIS:
Acute Coronary Syndrome (ACS)
Acute coronary syndrome was initially considered because the patient presented with acute chest pain, dyspnea, and tachycardia. However, the chest pain was pleuritic rather than ischemic in nature, serial electrocardiograms did not demonstrate ischemic ST-segment changes, and only minimal elevation of cardiac troponin was observed, consistent with right ventricular strain rather than myocardial infarction. CTPA confirmed pulmonary embolism, effectively excluding ACS as the primary diagnosis.

Pneumonia
Community-acquired pneumonia was considered because of the patient's respiratory symptoms. However, the absence of fever, productive cough, leukocytosis, and focal crepitations made this diagnosis less likely. Chest radiography showed no focal pulmonary consolidation, while CTPA demonstrated pulmonary arterial thrombi with only a small peripheral pulmonary infarct, excluding infective pneumonia.

Pneumothorax
Acute pneumothorax was considered owing to the sudden onset of pleuritic chest pain and dyspnea. Physical examination did not reveal unilateral hyperresonance, absent breath sounds, or tracheal deviation. Chest X-ray and CTPA showed fully expanded lungs without pleural air collection, thereby ruling out pneumothorax.

Aortic Dissection
Acute aortic dissection was included in the differential diagnosis because of the sudden onset of chest pain. However, the pain was not described as tearing or radiating to the back, blood pressure was stable without inter-arm differences, peripheral pulses were symmetrical, and CTPA demonstrated a normal thoracic aorta without evidence of intimal flap or dissection.

COPD Exacerbation
Acute exacerbation of chronic obstructive pulmonary disease was considered because of dyspnea and hypoxemia. Nevertheless, the patient had no previous history of COPD, chronic smoking, chronic cough, or wheezing. Chest imaging did not reveal emphysematous changes or hyperinflation, making COPD exacerbation unlikely.

Asthma
Acute bronchial asthma was considered as a possible cause of respiratory distress. However, the patient had no prior history of asthma or allergic disease, and auscultation revealed no expiratory wheeze or prolonged expiration. The absence of bronchospasm and the presence of pulmonary arterial filling defects on CTPA excluded asthma as the underlying cause.

Heart Failure
Acute heart failure was considered because of breathlessness and mild elevation of BNP. However, there was no history of cardiac disease, peripheral edema, elevated jugular venous pressure, pulmonary edema, or cardiomegaly. Echocardiography demonstrated preserved left ventricular systolic function with only mild right ventricular strain secondary to pulmonary embolism, excluding primary heart failure.

COVID-19 Pneumonia
COVID-19 pneumonia was considered due to the presentation with acute dyspnea and hypoxemia. The patient had no recent history of fever, upper respiratory symptoms, or known exposure to COVID-19. Reverse transcriptase polymerase chain reaction (RT-PCR) testing for SARS-CoV-2 was negative, and CTPA did not demonstrate the bilateral diffuse ground-glass opacities characteristic of COVID-19 pneumonia. Instead, it revealed acute pulmonary arterial thromboembolism with a small pulmonary infarction, confirming pulmonary embolism as the definitive diagnosis.

Final Diagnosis
Based on the clinical presentation, elevated D-dimer levels, electrocardiographic evidence of right heart strain, echocardiographic findings, and computed tomography pulmonary angiography (CTPA), the patient was diagnosed with acute bilateral pulmonary embolism. The thrombus involved the right main pulmonary artery with extension into the right upper, middle, and lower lobar arteries, along with additional segmental emboli in the left lower lobe pulmonary artery. Although the patient exhibited mild right ventricular strain on CTPA and echocardiography, he remained hemodynamically stable without hypotension or shock. Therefore, the condition was classified as intermediate-risk (submassive) acute pulmonary embolism. The diagnosis prompted immediate initiation of anticoagulant therapy, resulting in favorable clinical recovery without the need for thrombolysis or surgical intervention.

TREATMENT AND MANAGEMENT:
Initial Stabilization
The patient was immediately admitted to the emergency department and placed under continuous cardiac and pulse oximetry monitoring. Intravenous access was secured, and baseline laboratory investigations were completed. As the patient was hemodynamically stable without hypotension or shock, conservative management with close observation was initiated while awaiting definitive imaging results.

Oxygen Therapy
Supplemental oxygen was administered via a face mask at 5 L/min, resulting in improvement of oxygen saturation from 88% to 95%. Oxygen therapy was continued to maintain arterial oxygen saturation above 94%, with regular monitoring of respiratory status and arterial blood gases.

Anticoagulation
Following confirmation of pulmonary embolism on CTPA, low-molecular-weight heparin (Enoxaparin 1 mg/kg subcutaneously every 12 hours) was initiated immediately. After clinical stabilization, the patient was transitioned to oral Apixaban (10 mg twice daily for 7 days, followed by 5 mg twice daily) for long-term anticoagulation. Renal function, complete blood count, and coagulation parameters were monitored throughout treatment, and no bleeding complications occurred.

Thrombolysis
Systemic thrombolytic therapy was not indicated, as the patient remained hemodynamically stable without persistent hypotension, cardiogenic shock, or severe right ventricular dysfunction. Therefore, anticoagulation alone was considered the appropriate treatment according to current management guidelines.

ICU Care
The patient was admitted to the Intensive Care Unit (ICU) for close monitoring during the initial 24–48 hours because of acute hypoxemia and evidence of mild right ventricular strain. Serial assessment of vital signs, oxygen saturation, electrocardiography, cardiac biomarkers, and echocardiography showed gradual clinical improvement without deterioration.

Supportive Management
Supportive treatment included adequate hydration, pain relief with analgesics for pleuritic chest pain, and early mobilization after clinical stabilization to reduce further thromboembolic risk. Compression stockings were advised during prolonged immobilization, and the patient received counseling regarding lifestyle modification, weight reduction, regular physical activity, avoidance of prolonged sitting, and adherence to anticoagulant therapy. He was discharged in stable condition with scheduled follow-up for clinical evaluation and repeat imaging as indicated.

OUTCOME AND FOLLOW-UP:
Clinical Improvement
The patient showed significant clinical improvement within 48–72 hours of initiating anticoagulant therapy. Breathlessness gradually resolved, pleuritic chest pain diminished, and oxygen saturation improved to 97% on room air. Heart rate returned to normal, and the patient remained hemodynamically stable without recurrent symptoms or bleeding complications.

Hospital Stay
The patient remained hospitalized for five days for close monitoring of respiratory status, cardiac function, and response to anticoagulation. Serial clinical assessments and laboratory investigations demonstrated steady recovery, and no further thromboembolic events or treatment-related adverse effects were observed during hospitalization.

Discharge Medications
At discharge, the patient was prescribed Apixaban 5 mg orally twice daily following completion of the initial loading dose regimen. He was also advised to take analgesics as needed for residual pleuritic chest pain. The patient received detailed counseling regarding strict adherence to anticoagulant therapy, recognition of bleeding symptoms, and avoidance of medications that could increase bleeding risk without medical advice.

Duration of Anticoagulation
Considering that the pulmonary embolism was likely provoked by prolonged immobilization during long-distance travel, anticoagulation therapy was planned for a minimum duration of three months, with reassessment at follow-up to determine the need for extended therapy based on clinical risk factors and recurrence risk.

Follow-up Imaging
The patient was reviewed six weeks after discharge, at which time he remained asymptomatic with normal oxygen saturation and good functional recovery. Repeat transthoracic echocardiography demonstrated resolution of right ventricular strain. Follow-up CT pulmonary angiography performed after three months showed near-complete resolution of the pulmonary arterial thrombi without evidence of chronic thromboembolic pulmonary hypertension or residual vascular obstruction.

Long-term Prognosis
The patient achieved complete clinical recovery and resumed normal daily activities without limitations. He was advised to maintain regular physical activity, avoid prolonged periods of immobilization, remain adequately hydrated during long-distance travel, and attend periodic follow-up visits. With timely diagnosis by computed tomography pulmonary angiography, prompt initiation of anticoagulation, and good treatment compliance, the long-term prognosis was excellent, with a low anticipated risk of recurrence following completion of the recommended anticoagulation course.

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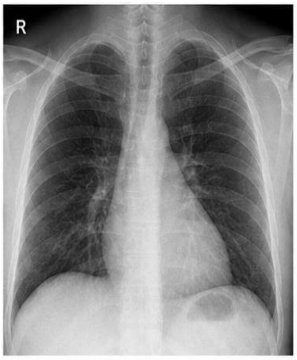
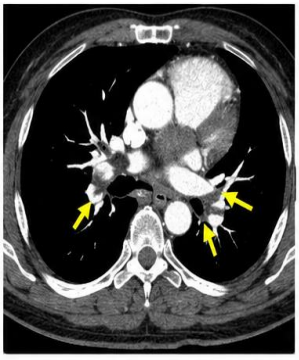
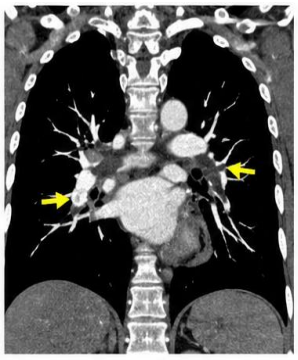


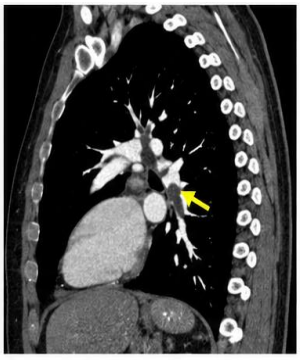
1. Chest X-ray: Normal cardiomediastinal silhouette. Lungs are clear bilaterally. No focal consolidation, pleural effusion or pneumothorax.



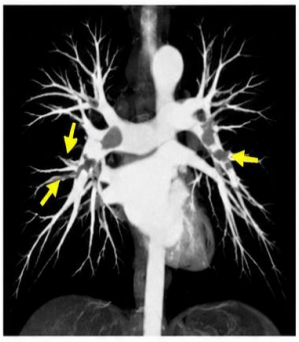
2. Axial CTPA: Axial image at the level of main pulmonary arteries showing filling defects (arrows) in the right and left pulmonary arteries consistent with acute pulmonary embolism.



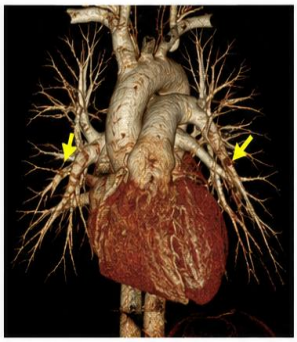
3. Coronal CTPA: Coronal reformatted image demonstrating filling defects (arrows) in bilateral pulmonary arteries extending into lobar branches.



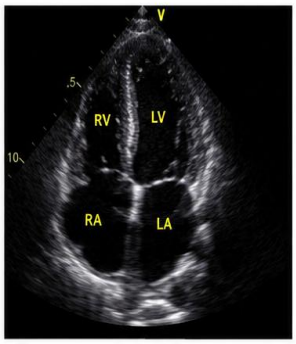
4. Sagittal CTPA: Sagittal reformatted image showing intraluminal filling defect (arrow) in a lobar branch of right pulmonary artery.



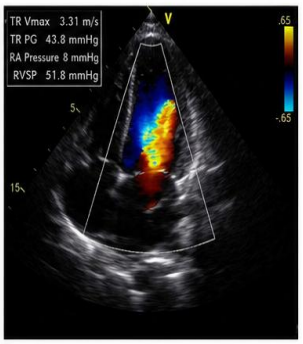
5. MIP Image: MIP (maximum intensity projection) image clearly demonstrates filling defects (arrows) in bilateral pulmonary arterial branches.



6. 3D Reconstruction: 3D volume-rendered CT image showing thrombi (arrows) in main and lobar pulmonary arteries bilaterally.



7. Echocardiography (Apical 4-chamber view): Mildly dilated right ventricle (RV) with interventricular septal flattening suggesting right ventricular strain.



8. Echocardiography (TR Doppler): Doppler image showing mild tricuspid regurgitation with estimated RV systolic pressure of ~52 mmHg, consistent with pulmonary hypertension due to PE.

DISCUSSION:

Pulmonary embolism (PE) is one of the most challenging cardiovascular emergencies because its clinical manifestations are often nonspecific and overlap with several cardiopulmonary disorders. Although dyspnea, pleuritic chest pain, tachycardia, and hypoxemia are considered classical manifestations, many patients present with atypical or subtle symptoms that delay diagnosis and increase the risk of morbidity and mortality. The present case illustrates an uncommon presentation of acute bilateral pulmonary embolism in a hemodynamically stable patient with minimal conventional thromboembolic risk factors and without obvious clinical evidence of deep vein thrombosis (DVT). The diagnosis was established only after maintaining a high index of suspicion and performing computed tomography pulmonary angiography (CTPA), highlighting the indispensable role of advanced imaging in patients with unexplained acute respiratory symptoms.

Several recent studies have emphasized that pulmonary embolism may occur even in the absence of clinically apparent DVT. Mishra et al. reported that more than half of patients with atypical pulmonary embolism had no clinical signs of lower-limb DVT despite subsequent Doppler evidence of thrombosis in many cases. Similar to the present case, unexplained hypoxemia and acute dyspnea were the principal presenting features, leading to definitive diagnosis only after CTPA was performed. Their findings reinforce the importance of considering pulmonary embolism in patients presenting with unexplained respiratory compromise, irrespective of the presence or absence of lower-extremity symptoms.[11]

The rarity of the present case lies in the combination of bilateral pulmonary embolism, absence of overt DVT manifestations, preserved hemodynamic stability, and relatively limited predisposing factors apart from prolonged immobilization during long-distance travel. While prolonged travel is a recognized risk factor for venous thromboembolism, many patients remain asymptomatic until embolization occurs. Mustafa et al. described a

similar case of massive bilateral pulmonary embolism in an otherwise healthy individual without classical risk factors, emphasizing that pulmonary embolism should remain an important differential diagnosis even in patients lacking significant medical comorbidities. Their observations closely parallel the diagnostic challenge encountered in our patient.[12]

The diagnostic evaluation of suspected pulmonary embolism requires careful integration of clinical probability scores, laboratory biomarkers, electrocardiography, echocardiography, and imaging studies. Although elevated D-dimer levels increase suspicion for thromboembolism, they lack specificity and should not be interpreted in isolation. Almarshad et al. demonstrated that combining modified Wells score assessment with D-dimer testing substantially improves appropriate utilization of CTPA while minimizing unnecessary imaging. In the present case, the patient's intermediate clinical probability, elevated D-dimer concentration, persistent hypoxemia, and sinus tachycardia appropriately justified urgent CTPA, ultimately confirming the diagnosis.[13]

Computed tomography pulmonary angiography remains the gold standard imaging modality for diagnosing acute pulmonary embolism because of its excellent sensitivity, specificity, rapid acquisition time, and ability to directly visualize intraluminal thrombi. Besides confirming pulmonary embolism, CTPA simultaneously evaluates clot burden, pulmonary infarction, right ventricular enlargement, and alternative thoracic pathologies. Yaghoobpoor et al., while evaluating trauma patients with pulmonary embolism, demonstrated that CTPA accurately identified embolic burden, associated pulmonary abnormalities, and predictors of mortality, thereby facilitating timely therapeutic intervention. The imaging findings in our patient similarly demonstrated bilateral pulmonary arterial filling defects, mild right ventricular strain, and peripheral pulmonary infarction, allowing accurate risk stratification and treatment planning.[14]

Another important aspect highlighted by this case is the value of recognizing subtle radiological indicators of right ventricular dysfunction. Although the patient remained normotensive, mild right ventricular dilatation observed on CTPA and echocardiography indicated intermediate-risk (submassive) pulmonary embolism. Early identification of right ventricular strain is clinically significant because it influences patient monitoring, need for intensive care, and decisions regarding thrombolytic therapy. Contemporary literature consistently demonstrates that imaging-based assessment of right ventricular function provides important prognostic information beyond simple confirmation of pulmonary embolism.[15]

The present case also underscores the importance of differentiating pulmonary embolism from other acute cardiopulmonary disorders. Initial symptoms closely resembled acute coronary syndrome, pneumonia, pneumothorax, and heart failure. However, absence of ischemic electrocardiographic changes, lack of pulmonary infiltrates on chest radiography, and normal left ventricular function on echocardiography shifted diagnostic consideration toward pulmonary embolism. Prompt performance of CTPA prevented further diagnostic delay and enabled immediate initiation of anticoagulation before hemodynamic deterioration occurred. Similar diagnostic pitfalls have been described in recent case reports where atypical clinical presentations initially resulted in alternative diagnoses before CTPA established pulmonary embolism.[16]

From a therapeutic perspective, this patient responded favorably to early anticoagulation with low-molecular-weight heparin followed by direct oral anticoagulant therapy, without requiring systemic thrombolysis. Current evidence indicates that patients with intermediate-risk pulmonary embolism who remain hemodynamically stable generally benefit from anticoagulation alone, reserving thrombolytic therapy for clinical deterioration or persistent hypotension. Early diagnosis through CTPA therefore directly influenced management decisions and contributed to an uncomplicated clinical recovery.[17]

The broader clinical implications of this case extend beyond individual patient management. Emergency physicians, pulmonologists, cardiologists, and radiologists should maintain a high index of suspicion for pulmonary embolism in patients presenting with unexplained dyspnea, pleuritic chest pain, tachycardia, or refractory hypoxemia, even when classical thromboembolic risk factors or clinical signs of DVT are absent. Reliance solely on clinical examination may result in delayed diagnosis, whereas timely application of validated clinical prediction tools and appropriate imaging significantly improves diagnostic accuracy and patient outcomes.[18]

Furthermore, this case emphasizes the indispensable contribution of multidetector CTPA with multiplanar

reconstruction in modern radiological practice. High-resolution axial, coronal, sagittal, and three-dimensional reconstructed images accurately delineated thrombus distribution, pulmonary infarction, and right ventricular morphology, thereby enhancing diagnostic confidence and facilitating comprehensive risk assessment. Recent advances in multidetector CT technology continue to improve visualization of segmental and subsegmental emboli while simultaneously reducing scan time and increasing diagnostic reliability.[19]

The principal lesson from this case is that pulmonary embolism should always remain an important differential diagnosis in patients with acute unexplained respiratory symptoms, irrespective of age or the presence of obvious thromboembolic risk factors. Careful clinical assessment, judicious use of D-dimer testing, prompt CTPA, and early initiation of anticoagulation remain the cornerstones of successful management. Increased awareness of atypical presentations among clinicians and radiologists may facilitate earlier diagnosis, reduce preventable mortality, and improve long-term patient outcomes.[20]

CONCLUSION:

Acute pulmonary embolism remains a potentially fatal medical emergency that frequently presents with nonspecific or atypical clinical manifestations, making early diagnosis particularly challenging. This case highlights the importance of maintaining a high index of clinical suspicion in patients presenting with unexplained acute dyspnea, pleuritic chest pain, tachycardia, and hypoxemia, even in the absence of classical risk factors or obvious signs of deep vein thrombosis. Computed tomography pulmonary angiography (CTPA) proved to be the definitive diagnostic modality by accurately demonstrating the location and extent of pulmonary arterial thrombi, assessing right ventricular strain, and excluding alternative thoracic pathologies. Prompt recognition of the condition and immediate initiation of anticoagulant therapy resulted in complete clinical recovery without significant complications. This case emphasizes that timely use of CTPA, combined with appropriate clinical assessment and evidence-based management, can significantly reduce morbidity and mortality. Increased awareness of atypical presentations among clinicians and radiologists is essential for ensuring early diagnosis, appropriate treatment, and improved long-term patient outcomes.

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