Massive Pulmonary Embolism Initially Misdiagnosed as Anxiety Attack in a Young Adult: A Case Report

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ABSTRACT

Background: Massive pulmonary embolism (PE) is a life-threatening cardiovascular emergency associated with substantial morbidity and mortality, particularly when diagnosis is delayed because of atypical clinical presentation. Young adults may present with nonspecific symptoms resembling psychiatric disorders, resulting in diagnostic uncertainty and delayed treatment.

Case Presentation: A 29-year-old male presented with progressive dyspnea, palpitations, chest tightness, dizziness, hyperventilation, and severe anxiety symptoms. During repeated emergency department visits, he was initially diagnosed with panic attacks and treated with anxiolytic medication. Despite therapy, his symptoms progressively worsened, leading to respiratory distress and hemodynamic instability. Electrocardiography demonstrated sinus tachycardia, an S1Q3T3 pattern, incomplete right bundle branch block, and anterior T-wave inversion suggestive of acute right ventricular strain. Laboratory investigations revealed elevated D-dimer, troponin-I, and brain natriuretic peptide levels. Transthoracic echocardiography showed severe right ventricular dilatation, pulmonary hypertension, septal flattening, and McConnell's sign. Computed tomography pulmonary angiography confirmed extensive bilateral pulmonary embolism involving both main pulmonary arteries with evidence of right ventricular strain. **Result:** The patient underwent systemic thrombolytic

therapy followed by anticoagulation treatment. Following intervention, significant clinical improvement was observed, including stabilization of hemodynamic parameters, improvement in oxygenation, and recovery of right ventricular function. **Conclusion:**This case highlights the importance of maintaining a high index of suspicion for pulmonary embolism in young adults presenting with unexplained anxiety-like cardiopulmonary symptoms. Early recognition and prompt diagnostic evaluation are essential to prevent delayed treatment, hemodynamic collapse, and potentially fatal outcomes.

Keywords: anxiety attack; case report; pulmonary embolism; pulmonary hypertension; right ventricular dysfunction; venous thromboembolism.

Introduction

Pulmonary embolism (PE) is one of the most important causes of cardiovascular morbidity and mortality worldwide and remains a major contributor to preventable hospital death [1]. Acute pulmonary embolism occurs when thrombotic material obstructs pulmonary arterial circulation, most commonly originating from deep venous thrombosis of the lower extremities[2]. The resulting increase in pulmonary vascular resistance may produce severe right ventricular pressure overload, impaired cardiac output, systemic hypotension, and circulatory collapse.

The global incidence of venous thromboembolism (VTE), including pulmonary embolism, has shown a steady increase over recent decades. Current epidemiological studies estimate an annual incidence of approximately 1–2 cases per 1,000 individuals in the general population, with substantially higher rates among older adults. Population-based analyses from Europe and North America have demonstrated a progressive rise in VTE-related hospitalizations and mortality burden, largely attributed to aging populations, increasing prevalence of obesity, sedentary lifestyles, malignancy, prolonged immobilization, and improved diagnostic detection. Recent data from the European Respiratory Society further reported that pulmonary embolism remains a major contributor to cardiovascular mortality, accounting for a significant proportion of preventable in-hospital deaths worldwide. Despite advances in diagnostic imaging and anticoagulation therapy, delayed diagnosis continues to represent an important clinical challenge[3]. Despite advancements in cardiovascular imaging and antithrombotic therapy, pulmonary embolism remains underdiagnosed because of its highly variable and nonspecific clinical presentation. [4]

Massive pulmonary embolism represents the most severe form of PE and is associated with significant mortality rates reaching 25%–50% when diagnosis or treatment is delayed.[5] Massive PE is characterized by sustained hypotension, obstructive shock, or cardiac arrest

resulting from severe pulmonary arterial obstruction and acute right ventricular failure. [6]

Younger adults with pulmonary embolism frequently present diagnostic challenges because symptoms may mimic psychiatric disorders such as panic attacks or generalized anxiety disorder.[7] Hyperventilation, palpitations, dyspnea, chest discomfort, dizziness, diaphoresis, tremulousness, and intense fear sensation are common manifestations in both PE and anxiety disorders. Consequently, clinicians may underestimate the possibility of life-threatening cardiopulmonary pathology in younger individuals without significant cardiovascular history.[8]

Delayed recognition of pulmonary embolism significantly increases risk of recurrent thromboembolism, chronic thromboembolic pulmonary hypertension, right ventricular failure, and sudden death.[9] Several studies have demonstrated that missed pulmonary embolism diagnosis remains common during initial emergency department evaluation, particularly among younger patients presenting with atypical symptoms.[10]

The overlap between psychological and cardiovascular symptoms highlights the importance of maintaining broad differential diagnosis in emergency and cardiovascular medicine. Pulmonary embolism should always be considered in patients presenting with unexplained tachycardia, dyspnea, pleuritic chest pain, or hypoxemia regardless of age. [11] This case report describes a young adult with massive pulmonary embolism initially misdiagnosed as anxiety attacks, emphasizing the importance of repeated clinical reassessment and early cardiovascular investigation in patients presenting with persistent cardiopulmonary symptoms.

Material and Methods

This study was conducted as a single-patient case report involving a young adult diagnosed with massive pulmonary embolism initially misdiagnosed as anxiety attacks. Clinical data were collected retrospectively from hospital medical records, laboratory investigations, electrocardiography, echocardiography, radiological imaging, treatment documentation, and follow-up evaluations during hospitalization.

The patient underwent comprehensive clinical assessment including history taking, physical examination, vital sign monitoring, and cardiovascular evaluation upon admission. Laboratory investigations included complete blood count, coagulation profile, serum D-dimer, cardiac troponin-I, brain natriuretic peptide (BNP), renal function tests, serum electrolytes, and arterial blood gas analysis.

Electrocardiography (ECG) was performed using a standard 12-lead recording system

to evaluate cardiac rhythm abnormalities and signs of right ventricular strain. Transthoracic echocardiography was performed to assess right ventricular size, right ventricular systolic function, pulmonary artery pressure, interventricular septal motion, and associated structural abnormalities.

Computed tomography pulmonary angiography (CTPA) was conducted using contrast-enhanced multidetector computed tomography to confirm the presence, location, and extent of pulmonary arterial thromboembolism. Lower limb Doppler ultrasonography was additionally performed to evaluate deep venous thrombosis as the embolic source.

The patient received management according to current European Society of Cardiology (ESC) and American Heart Association (AHA) guidelines for high-risk pulmonary embolism, including anticoagulation therapy, systemic thrombolysis, oxygen supplementation, hemodynamic monitoring, and supportive intensive care treatment.

Relevant literature regarding pulmonary embolism, anxiety-like presentation, right ventricular dysfunction, thrombolytic therapy, and diagnostic strategies was identified through searches of PubMed, Google Scholar, ESC Guidelines, AHA Guidelines, and peer-reviewed cardiovascular journals published between 2000 and 2025. References were formatted using Vancouver citation style.

Written informed consent for publication of clinical information and radiological findings was obtained from the patient prior to manuscript preparation. Personal identifying information was omitted to maintain confidentiality and privacy.

Results

Case Presentation

A 29-year-old male presented to the emergency department complaining of intermittent shortness of breath, chest tightness, palpitations, dizziness, and severe anxiety symptoms that progressively worsened over five days before hospitalization.

The patient described sudden episodes of breathlessness associated with hyperventilation, diaphoresis, trembling, and intense fear sensation. Symptoms occurred both during physical activity and at rest. He also complained of reduced exercise tolerance and fatigue. Initially, symptoms were attributed to occupational stress because the patient had recently experienced increased workload and sleep deprivation related to prolonged office duties. He worked predominantly in a seated position for long periods and reported minimal physical activity over recent months.

One week before symptom onset, the patient had completed a prolonged 12-hour

intercity bus journey with minimal movement during travel. Past medical history was unremarkable. The patient denied hypertension, diabetes mellitus, ischemic heart disease, asthma, chronic lung disease, malignancy, or previous thromboembolic events. No recent surgery or trauma was reported. Social history revealed active cigarette smoking approximately one pack daily for eight years. Alcohol consumption was occasional. There was no known family history of inherited thrombophilia or premature cardiovascular disease.

During the initial emergency department visit, vital signs demonstrated mild tachycardia with heart rate 108 beats/minute and respiratory rate 22 breaths/minute. Blood pressure was 124/78 mmHg and oxygen saturation remained 96% on room air. Physical examination was largely unremarkable. Initial electrocardiography demonstrated sinus tachycardia without significant ischemic abnormalities. Chest radiography showed no pulmonary infiltrates or pleural effusion.

Because of severe anxiety symptoms and emotional distress, the patient was diagnosed with panic attacks associated with stress and discharged with oral anxiolytic medication. Over the subsequent 48 hours, symptoms progressed substantially. The patient developed worsening dyspnea, pleuritic chest pain, near-syncope episodes, diaphoresis, and severe generalized weakness.

He returned to the emergency department in significant respiratory distress. On second presentation, examination revealed; blood pressure: 92/58 mmHg, heart rate: 132 beats/minute, respiratory rate: 30 breaths/minute, oxygen saturation: 88% on room air, Temperature: 36.8°C

The patient appeared anxious, diaphoretic, and tachypneic. Jugular venous distention was noted. Cardiac examination revealed tachycardia without audible murmur. Pulmonary examination demonstrated decreased bilateral basal breath sounds without wheezing. Mild left lower limb swelling and calf tenderness were observed during repeated examination. Given progressive cardiopulmonary deterioration, additional cardiovascular investigations were initiated.

Laboratory Findings

Laboratory evaluation revealed evidence of acute right ventricular strain and systemic hypoperfusion.

Table 1. Initial Laboratory Findings

Parameter	Result	Reference Range
Hemoglobin	14.1 g/dL	13–17 g/dL
Leukocyte count	13.2 ×10⁹/L	4–10 ×10⁹/L
Platelet count	286 ×10⁹/L	150–450 ×10⁹/L
D-dimer	6.8 µg/mL	<0.5 µg/mL
Troponin-I	0.84 ng/mL	<0.04 ng/mL
BNP	932 pg/mL	<100 pg/mL
Lactate	3.2 mmol/L	0.5–2.0 mmol/L
Creatinine	1.0 mg/dL	0.7–1.3 mg/dL
pH	7.49	7.35–7.45
$PaCO_2$	30 mmHg	35–45 mmHg
PaO_2	61 mmHg	80–100 mmHg

Elevated D-dimer strongly suggested active thromboembolic disease. Elevated cardiac biomarkers indicated myocardial injury secondary to acute right ventricular overload. Arterial blood gas analysis demonstrated respiratory alkalosis and hypoxemia consistent with acute pulmonary vascular obstruction.

Electrocardiographic Findings

Repeat electrocardiography demonstrated several findings suggestive of acute pulmonary embolism; Sinus tachycardia, S1Q3T3 pattern, Right axis deviation, Incomplete right bundle branch block, T-wave inversion in leads V1–V4. These findings were consistent with acute right ventricular strain.

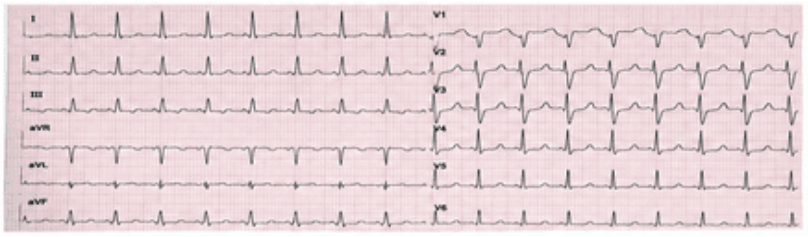

Figure 1. Twelve-lead electrocardiography on admission showing sinus tachycardia (rate 128 beats/min), S1Q3T3 pattern, right axis deviation, incomplete right bundle branch block, and T wave inversions in leads V1-V4 suggestive of acute right ventricular strain.

Echocardiographic Findings

Urgent transthoracic echocardiography demonstrated severe right ventricular dysfunction and pressure overload.

Table 2. Echocardiographic Findings

Parameter	Findings
Left ventricular ejection fraction	60%
Right ventricular size	Severely dilated

Parameter	Findings
RV systolic function	Moderately reduced
Septal flattening	Present
Pulmonary artery systolic pressure	58 mmHg
McConnell's sign	Present
Tricuspid regurgitation	Moderate

Echocardiography revealed significant right ventricular enlargement with interventricular septal flattening ("D-sign"), indicating acute pressure overload. McConnell's sign, characterized by akinesia of the right ventricular free wall with preserved apical contraction, strongly supported acute pulmonary embolism diagnosis.

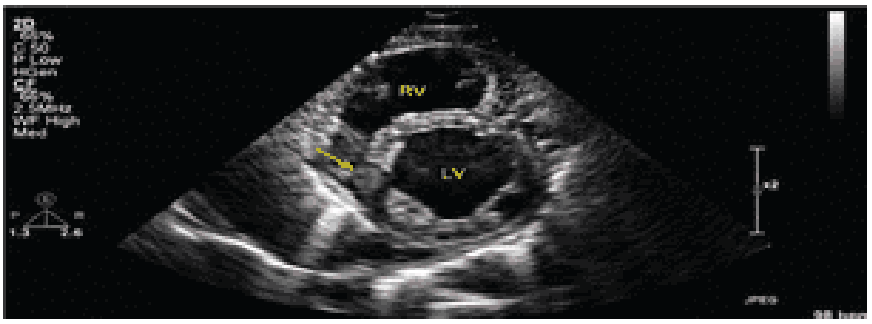

Figure 2. Transthoracic echocardiography (parasternal short-axis view) demonstrating a dilated right ventricle with interventricular septal flattening (D-shaped left ventricle), indicating right ventricular pressure overload.

Computed Tomography Pulmonary Angiography

Computed tomography pulmonary angiography demonstrated extensive bilateral pulmonary emboli involving both right and left main pulmonary arteries extending into segmental branches. Right ventricular enlargement with RV/LV ratio >1 indicated significant hemodynamic burden.

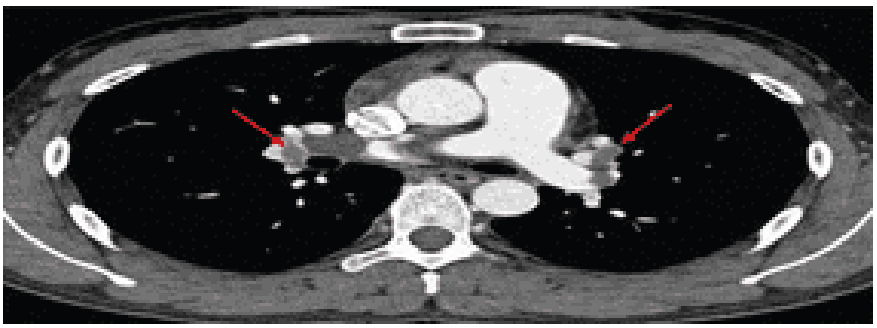

Figure 3. Computed tomography pulmonary angiography showing large filling defects in both right and left main pulmonary arteries (arrows) extending into the lobar and segmental branches, consistent with massive bilateral pulmonary embolism

Management

The patient was immediately admitted to the intensive care unit for advanced hemodynamic monitoring and treatment. Initial management included; High-flow oxygen therapy; Intravenous unfractionated heparin infusion; Intravenous crystalloid resuscitation ; Vasopressor support with norepinephrine; Given persistent hypotension and severe right ventricular dysfunction, the patient fulfilled criteria for high-risk massive pulmonary embolism.

After multidisciplinary discussion involving cardiology, intensive care, and cardiothoracic teams, systemic thrombolytic therapy was initiated using intravenous alteplase 100 mg over two hours according to ESC pulmonary embolism guidelines. Following thrombolysis, the patient demonstrated progressive clinical improvement including; Improved oxygenation; Reduction in tachycardia; Improved blood pressure stability; Reduced respiratory distress

Repeat echocardiography performed five days later demonstrated substantial improvement in right ventricular function and reduction in pulmonary artery systolic pressure from 58 mmHg to 36 mmHg. The patient was transitioned to oral rivaroxaban therapy prior to discharge. Prior to discharge, comprehensive education regarding smoking cessation, thrombosis prevention, physical activity to reduce future thrombotic risk.

Discussion

Pulmonary embolism remains one of the most challenging cardiovascular diagnoses because of highly variable clinical manifestations.[12] The present case highlights several important diagnostic and therapeutic considerations regarding acute massive pulmonary embolism in younger adults. The initial presentation strongly mimicked panic disorder or anxiety attack. Symptoms such as hyperventilation, chest discomfort, tachycardia, tremulousness, diaphoresis, dizziness, and fear sensation overlap substantially between psychiatric disorders and pulmonary embolism.[13]

This overlap contributes significantly to diagnostic delay. Several emergency medicine studies have demonstrated that pulmonary embolism may initially be overlooked in younger patients without traditional cardiovascular risk factors.[14] Pathophysiologically, acute pulmonary embolism results in abrupt pulmonary arterial obstruction causing increased pulmonary vascular resistance. Acute right ventricular afterload elevation leads to right ventricular dilatation and dysfunction.[15]

Unlike the left ventricle, the right ventricle possesses relatively thin myocardial walls

and limited ability to tolerate acute pressure overload. Progressive right ventricular dysfunction impairs left ventricular filling through interventricular septal displacement, resulting in reduced cardiac output and systemic hypotension.[16] Hypoxemia and reduced cerebral perfusion may contribute to anxiety-like symptoms and neuropsychological distress. Furthermore, sympathetic nervous system activation results in catecholamine surge causing palpitations, tremulousness, diaphoresis, and panic sensation.[17]

The present patient possessed several important thromboembolic risk factors including cigarette smoking, prolonged immobilization, sedentary lifestyle, and prolonged travel duration. Smoking contributes significantly to endothelial dysfunction, platelet activation, inflammation, and hypercoagulability.[18] Prolonged immobility further increases venous stasis and thrombus formation risk.

Electrocardiographic findings in pulmonary embolism remain nonspecific but may provide important clues. Sinus tachycardia represents the most common ECG finding.[19]

The classic S1Q3T3 pattern reflects acute right ventricular strain but demonstrates relatively low sensitivity. Nevertheless, anterior T-wave inversion, right axis deviation, and right bundle branch block strongly support right ventricular overload.[20] Echocardiography plays a critically important role in hemodynamically unstable patients. Right ventricular dysfunction represents one of the strongest prognostic markers in acute pulmonary embolism.[21] McConnell's sign observed in this patient demonstrates relatively high specificity for acute pulmonary embolism and reflects regional right ventricular dysfunction due to acute pressure overload.[22] CT pulmonary angiography remains the gold standard imaging modality for pulmonary embolism diagnosis because of excellent sensitivity and specificity.[23]

Risk stratification is essential for determining appropriate treatment strategy. According to ESC guidelines, high-risk pulmonary embolism is defined by presence of hemodynamic instability including sustained hypotension or shock.[24] Systemic thrombolytic therapy remains recommended first-line reperfusion strategy for high-risk PE because it rapidly restores pulmonary perfusion and reduces right ventricular afterload.[25]

However, thrombolysis carries substantial bleeding risk and therefore requires careful patient selection. Long-term complications following pulmonary embolism remain clinically important. Chronic thromboembolic pulmonary hypertension develops secondary to incomplete thrombus resolution and pulmonary vascular remodeling.[26] Persistent dyspnea, reduced exercise tolerance, fatigue, anxiety, and depression may significantly impair quality of life following acute PE.[27]

This case also emphasizes the importance of avoiding premature diagnostic closure. Initial attribution of symptoms to anxiety resulted in delayed recognition of life-threatening cardiovascular disease. Repeated clinical reassessment remains essential when symptoms persist or worsen despite initial treatment. Prevention of pulmonary embolism remains a critical component of reducing disease burden and mortality. Identification and modification of risk factors such as prolonged immobilization, sedentary lifestyle, cigarette smoking, obesity, dehydration, and prolonged travel are essential preventive strategies. Early mobilization during hospitalization, regular physical activity, adequate hydration, smoking cessation, and the use of pharmacologic or mechanical thromboprophylaxis in high-risk individuals have been shown to reduce the incidence of venous thromboembolism. Furthermore, increased awareness among both clinicians and patients regarding the early symptoms of pulmonary embolism may facilitate prompt diagnosis and intervention, thereby preventing progression to massive pulmonary embolism and hemodynamic collapse.

Several clinical prediction models including Wells score and Geneva score may improve early diagnostic stratification and guide imaging decisions.[28] Future advances in pulmonary embolism diagnosis may involve artificial intelligence-assisted triage systems integrating symptoms, biomarkers, ECG findings, and imaging data for rapid risk assessment.

Limitation

This report represents a single-patient case and may not fully reflect broader pulmonary embolism populations. Long-term thrombophilia investigations were limited during acute hospitalization. Extended follow-up regarding chronic thromboembolic pulmonary hypertension and recurrent venous thromboembolism was also unavailable.

Conclusion

Massive pulmonary embolism remains a potentially fatal cardiovascular emergency that may be difficult to recognize when the initial presentation resembles anxiety or panic disorder. This case demonstrates that symptoms such as dyspnea, palpitations, chest tightness, dizziness, hyperventilation, diaphoresis, and fear sensation should not automatically be attributed to psychiatric causes, particularly when they are persistent, progressive, or associated with tachycardia and hypoxemia. Young age does not exclude pulmonary embolism. In this patient, prolonged travel, sedentary activity, and smoking were important risk factors that should increase clinical suspicion for venous thromboembolism. Delayed recognition allowed progression to right ventricular strain and hemodynamic

instability, emphasizing the importance of repeated clinical reassessment when symptoms worsen despite initial treatment.

Early diagnostic evaluation using D-dimer, electrocardiography, echocardiography, lower-limb venous ultrasound, and computed tomography pulmonary angiography is essential for timely diagnosis. Echocardiographic evidence of right ventricular dilatation, septal flattening, pulmonary hypertension, and McConnell's sign may provide important clues in unstable patients before definitive imaging is performed. Prompt risk stratification and immediate treatment are crucial in massive pulmonary embolism. Systemic thrombolysis followed by anticoagulation can rapidly improve pulmonary perfusion, reduce right ventricular afterload, stabilize hemodynamics, and prevent fatal outcomes when used appropriately. Long-term follow-up remains necessary to monitor recurrent venous thromboembolism, bleeding complications from anticoagulation, persistent dyspnea, and chronic thromboembolic pulmonary hypertension.

Overall, this case highlights the need for clinicians to maintain a broad differential diagnosis in young adults presenting with unexplained cardiopulmonary symptoms. A diagnosis of anxiety should be made carefully only after potentially life-threatening causes have been considered. Greater awareness of atypical pulmonary embolism presentation may reduce diagnostic delay, improve emergency decision-making, and ultimately decrease preventable mortality.

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