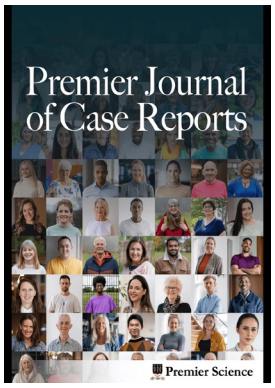

Silent Pulmonary Embolism in a Low-Risk Young Adult: A Case Report of Missed Diagnosis Behind Atypical Chest Pain

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ABSTRACT

BACKGROUND

Pulmonary embolism (PE) can present with highly variable symptoms, often leading to delayed diagnosis, particularly in young, low-risk individuals without classical features. Pleuritic chest pain, although common in peripheral PE, is frequently misattributed to benign causes, especially when reproducible on palpation. This case illustrates a diagnostic pitfall in evaluating chest pain and underscores the importance of a structured clinical assessment.

CASE PRESENTATION

A 38-year-old male software engineer presented with a 5-day history of sharp, right-sided pleuritic chest pain. He was hemodynamically stable, afebrile, and denied dyspnea or other cardiopulmonary symptoms. The pain was reproducible on palpation, leading to an initial diagnosis of musculoskeletal chest wall pain. However, due to symptom persistence and an elevated D-dimer (1.20 µg/mL), computed tomography pulmonary angiography was performed and revealed a segmental PE in the right lower lobe with associated wedge-shaped subpleural consolidation consistent with pulmonary infarction. The patient was managed as an outpatient with rivaroxaban and remained symptom-free at follow-up. Thrombophilia testing was deferred.

DISCUSSION

This case demonstrates how anchoring bias and low pre-test probability may delay critical diagnoses in younger patients. Reproducible chest pain does not exclude PE, and persistent pleuritic pain warrants structured evaluation. Segmental emboli and infarctions are more common in younger populations and may present without dyspnea or hypoxia.

CONCLUSION

Clinicians must maintain a high index of suspicion for PE in patients with pleuritic chest pain, regardless of age or apparent risk, and should use validated decision tools to guide imaging and treatment decisions.

Keywords: Silent pulmonary embolism, Pleuritic chest pain, Segmental pulmonary infarction, Diagnostic anchoring bias, Low-risk young adults

Introduction

Pulmonary embolism (PE) remains a major cause of preventable cardiovascular mortality[1] and is the third leading cause of cardiovascular death worldwide.[2] Despite diagnostic advancements, PE often presents with vague or atypical symptoms, making timely recognition difficult, especially in young or ostensibly low-risk individuals.[3,4]

While classical features such as sudden dyspnea, tachypnea, and hypoxemia are widely taught, population-based data show that fewer than half of patients with confirmed PE exhibit these findings.[3,4] Atypical symptoms, including pleuritic chest pain, syncope, or mild fever, are disproportionately observed in non-malignant, unprovoked PEs and in younger adults without cardiopulmonary comorbidities.[4,5] Among these, pleuritic chest pain is common but non-specific, reported in 66% of cases in the seminal Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study.[6] However, its presence is often falsely reassuring when deemed musculoskeletal, especially if reproducible on palpation.[7,8] Notably, prospective studies have shown that palpation-provoked chest pain does not significantly reduce the probability of PE (19.9% vs. 23.8%, $p = 0.25$), underscoring the limitations of bedside heuristics.[9]

Delayed or missed diagnosis is a well-documented problem. In one multicenter review, 47% of cases experienced at least a 3-day delay in diagnosis,[10] primarily due to low clinical suspicion in the absence of conventional risk factors or classic symptoms, and this was associated with worse short-term outcomes.

Peripheral or segmental emboli may produce wedge-shaped infarctions seen on imaging, typically prompting symptoms localized to the costal area rather than generalized dyspnea or shock. Yet, such presentations are commonly misattributed to musculoskeletal or viral pleuritic causes, particularly in outpatient or primary-care settings that lack immediate imaging access.[11,12]

This case describes a 38-year-old man with isolated right-sided pleuritic chest pain, initially misdiagnosed as musculoskeletal chest wall pain following a mild viral illness. Subsequent imaging revealed a segmental pulmonary embolus with pulmonary infarction. This case highlights the importance of maintaining diagnostic vigilance, even in low-risk patients, and demonstrates how subtle cues, such as persistent pleuritic pain, should prompt further investigation guided by clinical decision tools.

Case Presentation

A 38-year-old male software engineer presented to the emergency department (ED) with a 5-day history of sharp, right-sided pleuritic chest pain rated 6 out of 10 in intensity. The pain was sudden in onset, worsened by deep inspiration and coughing, and had shown minimal response to over-the-counter ibuprofen. He denied accompanying symptoms such as dyspnea, palpitations, syncope, fever, or hemoptysis. His past medical history was unremarkable. He was a non-smoker, used alcohol socially, denied recreational drug use, and was not taking any medications. There

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was no personal or family history of venous thromboembolism, malignancy, or premature cardiovascular disease. One week earlier, he had recovered from a mild, self-limiting upper respiratory tract infection, and there had been no recent travel, surgery, trauma, or prolonged immobility. A SARS-CoV-2 Polymerase Chain Reaction test was performed during the ED evaluation and returned negative, effectively ruling out COVID-19 as a contributing factor.

Three days prior to his ED visit, the patient was evaluated by his general practitioner, who attributed the chest pain to musculoskeletal strain based on focal tenderness over the right lower costal margin and the absence of systemic or respiratory "red-flag" symptoms. He was prescribed ibuprofen 400 mg three times daily and advised to rest. However, the pain persisted without significant improvement.

On presentation to the ED, he was alert, afebrile (36.8 °C), mildly tachycardic with a heart rate of 102 bpm, normotensive at 124/76 mmHg, and had a normal respiratory rate (18 breaths/min) and oxygen saturation (98% on room air). Physical examination revealed focal tenderness over the right eighth costochondral junction, with no crackles, wheeze, calf tenderness, or asymmetry. An electrocardiogram showed sinus tachycardia without signs of right heart strain. Laboratory investigations, including a complete blood count, electrolyte levels, and renal function tests, were within normal limits. High-sensitivity C-reactive protein was slightly elevated at 6.3 mg/L. Given the absence of clear findings and persistent pain, a structured risk assessment was performed. Clinical probability scores yielded mixed results: the Simplified Revised Geneva score placed him in the low-risk category (score 1), whereas the two-level Wells criteria produced a score of 4.5, classifying PE as likely. The Pulmonary Embolism Severity Index (PESI) score was approximately 68, placing him in Class II with an estimated 30-day mortality of <1.5%.

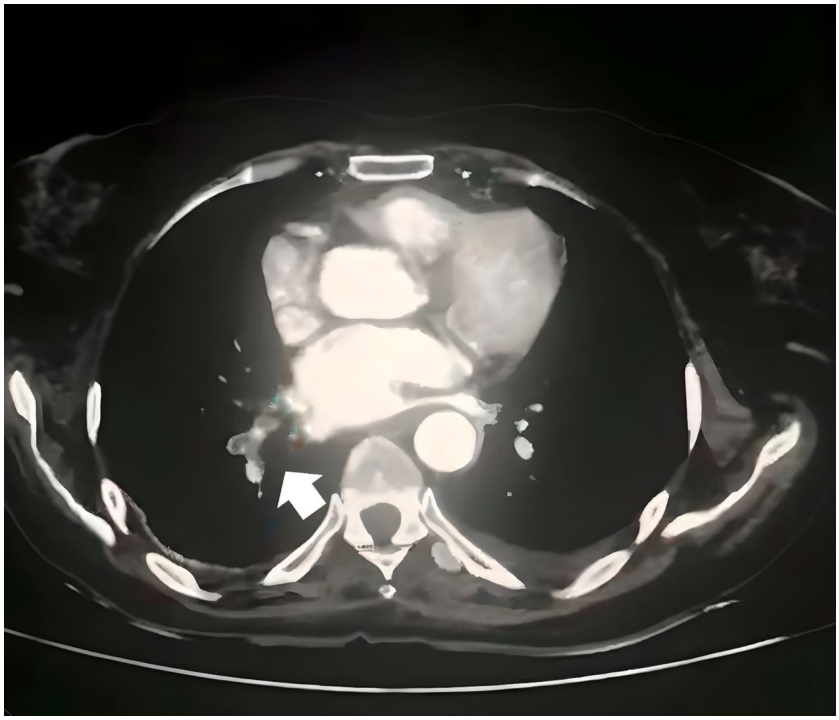

Fig 1 | Axial CT pulmonary angiography showing a segmental PE (pointed to with arrow) in the posterior-basal branch of the right lower lobe pulmonary artery. The filling defect is well-demarcated and located peripherally, consistent with segmental PE contributing to pulmonary infarction

A D-dimer level was elevated at 1.20 µg/mL Fibrinogen-Equivalent Units (reference <0.50), prompting computed tomography (CT) pulmonary angiography. Imaging revealed a segmental filling defect in the posterior-basal segment of the right lower lobe, accompanied by a 2.1 cm wedge-shaped subpleural consolidation consistent with pulmonary infarction (Figure 1). Lower-limb compression ultrasonography was negative for proximal deep vein thrombosis. Electrocardiographic and biomarker assessments revealed no evidence of right ventricular strain or myocardial injury.

A final diagnosis of isolated segmental PE with peripheral infarction was made in the absence of identifiable provoking factors. The patient was initiated on rivaroxaban 15 mg twice daily with meals for the first 21 days, followed by 20 mg once daily. Bleeding risk was assessed using the HAS-BLED score and found to be negligible (score of 0), with no contraindications to anticoagulation. Given the low PESI score and normal cardiorespiratory parameters, he was discharged the same day under an ambulatory PE pathway.

A comprehensive outpatient plan was initiated, including nurse-led anticoagulation counseling and a rivaroxaban starter pack, a follow-up with his general practitioner within 48 hours, and a hematology clinic review scheduled at 4 weeks. A repeat lower-limb Doppler ultrasound was planned for day 7, in line with American College of Chest Physicians (ACCP) guidance for isolated subsegmental or segmental PE. Thrombophilia testing was deferred until at least 4 weeks after cessation of anticoagulation, unless a second unprovoked event occurred.

At the 3-week follow-up, the patient reported complete resolution of symptoms, no bleeding complications, and good medication adherence. He was counseled on the risks and benefits of continuing anticoagulation beyond 3 months, including the estimated 20–25% 5-year recurrence risk if therapy were discontinued. A shared decision regarding long-term management was scheduled for the 3-month review. No changes were made to the therapeutic regimen during follow-up, and the patient experienced no adverse effects or bleeding complications throughout the treatment course. A summary of key clinical events is presented in Figure 1 to illustrate the temporal sequence from symptom onset to diagnosis, treatment, and resolution. This case report has been prepared in accordance with the CAse REport guidelines.[13]

Discussion

PE remains an ever-changing and frequently under-recognized entity, particularly when it presents with atypical features in younger, low-risk individuals. This

case of a 38-year-old man with isolated pleuritic chest pain, normal oxygen saturation, and no overt thrombotic risk factors illustrates how peripheral emboli can evade detection when clinicians anchor on the absence of dyspnea or hemodynamic instability. His diagnosis of segmental PE with radiologic infarction underscores the need for a structured, evidence-based approach to chest pain evaluation, even in patients deemed low-risk by demographic profiles alone.

Although traditional teaching emphasizes dyspnea, tachypnea, and hypoxia as cardinal signs of PE, population-based studies have consistently shown that these features are absent in a substantial minority. In the PIOPED I and II trials, pleuritic chest pain was present in 47–66% of confirmed PE cases, whereas dyspnea occurred in only 57–73%.[6,14] More recent registry data from low-risk PE cohorts similarly indicate that pleuritic pain can outnumber breathlessness as a presenting symptom.[3] Yet the diagnostic significance of such pain is often underappreciated, especially when it is reproducible on palpation, which many clinicians incorrectly interpret as pathognomonic for musculoskeletal origin. Prospective data have refuted this heuristic: in one large ED-based study, palpation-provoked chest pain had a negative likelihood ratio close to 1.0, rendering it diagnostically neutral.[9] Furthermore, the anatomical location of emboli significantly influences clinical presentation; segmental or subsegmental PEs are less likely to produce dyspnea or hypotension but more often lead to pleuritic pain due to distal vessel occlusion and subsequent pulmonary infarction.[15] In the PIOPED series, nearly one-quarter of patients with segmental PE reported no dyspnea at all.[14]

Pulmonary infarction, occurring in 16–36% of acute PE cases, often causes pleuritic pain via pleural irritation and alveolar hemorrhage.[15,16] Notably, infarction is not predominantly a disease of the elderly; in fact, younger age appears to confer a higher risk. A multicenter study found an inverse relationship between age and infarction risk, with younger patients more likely to present with pleuritic pain and hemoptysis.[17] This epidemiological profile aligns with our patient's presentation (Table 1).

Misdiagnosis of PE remains common, especially when symptoms are non-specific. Studies suggest that PE is missed or delayed in up to half of first-episode cases, with diagnostic lags averaging more than 6 days across healthcare systems.[18] Contributing factors include the absence of "classic" signs, clinician overreliance on gestalt,[19] and premature closure when an apparent benign cause, such as viral pleuritis or costochondritis, is identified.[20] In this case, the initial attribution of symptoms to musculoskeletal pain delayed definitive imaging. Importantly, even when structured tools such as the Revised Geneva or Wells scores are applied, interpretation can be inconsistent in younger patients. For example, our patient's Simplified Geneva score was low, primarily driven by his age and lack of comorbidities, whereas his Wells score exceeded the "PE likely" threshold, highlighting a discordance that often arises in borderline or atypical presentations. Despite the low Geneva score, the elevated D-dimer and persistent pleuritic pain prompted CT pulmonary angiography, which confirmed the diagnosis.

This case also illustrates the clinical rationale behind imaging decisions and outpatient management. Current European Society of Cardiology and National Institute for Health and Care Excellence guidelines recommend imaging without D-dimer testing in patients with a Wells score of 4 or higher, while the 2021 ACCP guidelines support structured outpatient management of low-risk PE with appropriate follow-up infrastructure;[21,22] however, many centers, including ours, still use D-dimer as an initial filter. This supported imaging, which confirmed the diagnosis. The decision to manage the patient as an outpatient was supported by his low PESI score, stable vital signs, absence of right heart strain, and access to follow-up. Outpatient treatment of low-risk PE has been validated in multiple trials and is endorsed by the British Thoracic Society when structured pathways are in place. In our case, the decision to manage the patient as an outpatient was based on validated predictors of low-risk PE (PESI Class II), absence of right heart strain, and access to timely review, all of which align with published outpatient pathway models.[23,24] The patient received direct oral anticoagulation with rivaroxaban, had nurse-led counseling at discharge, and attended timely primary and specialist follow-ups. Thrombophilia testing was deferred in the acute setting in accordance with current ACCP guidelines, which recommend postponing evaluation until at least 3 months after the event to avoid false positives due to inflammation or anticoagulation effects.[22]

Our study had some limitations. First, as with all single-patient case reports, the findings may not be generalizable, and no causal inference can be drawn regarding the observed infarction and clinical presentation. Second, the diagnosis of pulmonary infarction was based on radiological imaging without histopathological confirmation, and no follow-up imaging was conducted to confirm resolution. Third, we did not have access to a coronal view of the CT. Additionally, the role of a recent viral illness in thrombosis remains speculative without confirmatory virological testing.

The broader implications of this case are manifold. This case reinforces the value of structured clinical decision tools rather than relying on unstructured intuition. Chest pain in young adults, even when reproducible,

Table 1 | Clinical timeline of events

Day	Event
Day −7	Mild viral upper respiratory tract symptoms (self-resolving)
Day 0	Onset of sharp right-sided pleuritic chest pain
Day 2	Evaluated by General Practitioner → Diagnosed with musculoskeletal pain → Ibuprofen prescribed
Day 5	Presented to ED due to persistent symptoms
Day 5	Clinical examination + Risk scoring (Wells = 4.5, Geneva = 1) + Elevated D-dimer (1.20 µg/mL)
Day 5	CT Pulmonary Angiography → Segmental PE with infarction confirmed
Day 5	Initiated on rivaroxaban → Discharged on outpatient PE pathway
Day 7	Planned repeat Doppler ultrasound (per ACCP guidance)
Week 3	Outpatient follow-up → Complete resolution of symptoms, no complications

should not be dismissed without considering thromboembolic disease, especially if it is pleuritic and persistent. Importantly, post-viral states may temporarily increase the prothrombotic risk, and viral illness does not preclude the development of PE. Our experience also reinforces the value of follow-up planning and safety-netting, including a 48-hour review, 7-day repeat Doppler ultrasonography for occult deep venous thrombosis, and patient education on red-flag symptoms, all of which reduce adverse outcomes. Ultimately, this case highlights how PE can present deceptively, even in young, well-appearing individuals. Anchoring bias, such as assuming reproducible pain rules out serious pathology, and overconfidence in low pre-test probability can delay life-saving diagnoses. A structured, cautious, and evidence-based approach is crucial for identifying these silent yet dangerous presentations.

Patient Perspective

"At first, I didn't think my chest pain was anything serious. It felt sharp, especially when I breathed in, but I assumed it was a pulled muscle or something from the mild cold I had the week before. Even when I went to the doctor and they said it was probably musculoskeletal, I wasn't too worried. But when the pain didn't go away, I started to get concerned. I was shocked when the scans showed I had a blood clot in my lung. I'm grateful that the diagnosis was eventually made and that I could be treated at home. The medication was easy to take, and I didn't have any side effects. Within a couple of weeks, I felt completely normal again. I'm more aware now of how subtle the serious things can be."

Conclusion

This case highlights the diagnostic challenge of PE in young, otherwise healthy adults presenting with isolated pleuritic chest pain. Despite the absence of traditional risk factors and reassuring clinical findings, a segmental embolism with pulmonary infarction was ultimately diagnosed through structured assessment and appropriate imaging. The case underscores the limitations of relying on clinical heuristics, such as palpation-provoked pain, as well as the importance of maintaining a high index of suspicion when symptoms persist. Early recognition of atypical presentations and adherence to evidence-based decision-making tools are essential to avoid delays, prevent complications, and ensure safe and effective management, even in low-risk patients.

References

1 Vyas V, Abdulghani S, Goyal A. Acute pulmonary embolism. Nihgov; 2024.
2 Tarbox AK, Swaroop M. Pulmonary embolism. Int J Crit Illn Inj Sci. 2013;3(1):69–72. https://doi.org/10.4103/2229-5151.109427
3 Dušan Predrag R, Boris D, Jovan M, Marija B, Sonja S, Ljiljana K, et al. Signs and symptoms of acute pulmonary embolism and their predictive value for all-cause hospital death in respect of severity of the disease, age, sex and body mass index: retrospective analysis of the Regional PE Registry (REPER). BMJ Open Respir Res. 2023;10(1):e001559. https://doi.org/10.1136/bmjresp-2022-001559
4 Khasin M, Gur I, Evgrafov EV, Toledano K, Zalts R. Clinical presentations of acute pulmonary embolism: a retrospective cohort study. Medicine (Baltimore). 2023;102(28):e34224. https://doi.org/10.1097/md.0000000000034224
5 Jolobe OM. Atypical manifestations of pulmonary embolism. Arch Vas Med. 2020;4(1):008–18. https://doi.org/10.29328/journal.avm.1001012
6 Worsley DF, Alavi A. Comprehensive analysis of the results of the PIOPED study. J Nucl Med. 1995;36(12):2380–7.
7 Koenig KL. Does chest pain reproduced by palpation rule out PE? NEJM J Watch. 2005:EM200504270000008.
8 Menzies SM. Chest wall tenderness does not exclude pulmonary embolism. Thorax. 2005;60(6):461. https://doi.org/10.1136/thx.2005.la0153
9 Le Gal G, Testuz A, Righini M, Bounameaux H, Perrier A. Reproduction of chest pain by palpation: diagnostic accuracy in suspected pulmonary embolism. Bmj. 2005;330(7489):452–3. https://doi.org/10.1136/bmj.38331.602384.8f
10 Goyard C, Côté B, Looten V, Roche A, Pastré J, Marey J, et al. Determinants and prognostic implication of diagnostic delay in patients with a first episode of pulmonary embolism. Thromb Res. 2018;171:190–8. https://doi.org/10.1016/j.thromres.2018.08.015
11 Sankar J, Unniyal M, Ramprasad R, Khullar A. Peripheral pulmonary embolism: a missed entity. Med J Dr DY Patil Vidyapeeth. 2019;12(3):278–80. https://doi.org/10.4103/mjdrdypu.mjdrdypu_144_18
12 Yuan Y, Wang H-Y, Xing Z-C, Hou Z-L, Wang J-X, Zhang S. Characteristics of pulmonary infarction in patients with acute pulmonary embolism in China: a single-center retrospective observational study. J Thorac Dis. 2023;15(11):5961v70. https://doi.org/10.21037/jtd-23-944
13 Riley DS, Barber MS, Kienle GS, Aronson JK, von Schoen-Angerer T, Tugwell P, et al. CARE guidelines for case reports: explanation and elaboration document. J Clin Epidemiol. 2017;89:218–35. https://doi.org/10.1016/j.jclinepi.2017.04.026
14 Stein PD, Beemath A, Matta F, Weg JG, Yusen RD, Hales CA, et al. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med. 2007;120(10):871–9. https://doi.org/10.1016/j.amjmed.2007.03.024
15 Sonmez M, Aboussouan LS, Farver C, Murthy SC, Kaw R. Pulmonary infarction due to pulmonary embolism. Cleve Clin J Med. 2018;85(11):848–52. https://doi.org/10.3949/ccjm.85a.17132
16 Lio KU, O'Corragain O, Bashir R, Brosnahan S, Cohen G, Lakhter V, et al. Clinical outcomes and factors associated with pulmonary infarction following acute pulmonary embolism: a retrospective observational study at a US academic centre. BMJ Open. 2022;12(12):e067579. https://doi.org/10.1136/bmjopen-2022-067579
17 Miniati M, Bottai M, Ciccotosto C, Roberto L, Monti S. Predictors of pulmonary infarction. Medicine. 2015;94(41):e1488. https://doi.org/10.1097/md.0000000000001488
18 van Maanen R, Trinks-Roerdink EM, Rutten FH, Geersing GJ. A systematic review and meta-analysis of diagnostic delay in pulmonary embolism. Eur J Gen Pract. 2022;28(1):165–72. https://doi.org/10.1080/13814788.2022.2086232
19 Bellou E, Keramida E, Karampinis I, Dimakakos E, Misthos P, Demertzis P, et al. Outpatient treatment of pulmonary embolism. Breathe (Sheff). 2020;16(3):200069. https://doi.org/10.1183/20734735.0069-2020
20 Kwok CS, Wong CW, Lovatt S, Myint PK, Loke YK. Misdiagnosis of pulmonary embolism and missed pulmonary embolism: a systematic review of the literature. Health Sci Rev. 2022;3:100022. https://doi.org/10.1016/j.hsr.2022.100022
21 Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 2020;41(4):543–603. https://doi.org/10.1183/13993003.01647-2019
22 Stevens SM, Woller SC, Kreuziger LB, Bounameaux H, Doerschug K, Geersing G-J, et al. Antithrombotic therapy for VTE disease: second update of the CHEST guideline and expert panel report. CHEST. 2021;160(6):e545–608. https://doi.org/10.1016/j.chest.2021.07.055
23 Zondag W, Hiddinga BI, Crobach MJ, Labots G, Dolsma A, Durian M, et al. Hestia criteria can discriminate high- from low-risk patients with pulmonary embolism. Eur Respir J. 2013;41(3):588–92. https://doi.org/10.1183/09031936.00030412
24 Kabrhel C, Van Hylckama Vlieg A, Muzikanski A, Singer A, Fermann GJ, Francis S, et al. Multicenter evaluation of the YEARS criteria in emergency department patients evaluated for pulmonary embolism. Acad Emerg Med. 2018;25(9):987–94. https://doi.org/10.1111/acem.13417