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# Pulmonary Embolism Mimicking Other Diseases: Diagnostic Pitfalls and Atypical Clinical Presentations

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Abstract

Background: Pulmonary embolism (PE) remains a clinically significant and potentially fatal condition whose diagnosis may be difficult because its manifestations often overlap with more common cardiopulmonary, abdominal, and neurological disorders. In addition to classic presentations, PE may resemble acute coronary syndrome, heart failure, pneumonia, syncope, abdominal pathology, or neurological disease, which may contribute to delayed recognition.

Aim: The aim of this review was to summarize atypical clinical presentations of PE, identify common diagnostic pitfalls, and examine factors that may help reduce misdiagnosis.

Material and methods: This manuscript was prepared as a narrative literature review based on selected publications addressing clinical variability, diagnostic pathways, disease mimicry, laboratory and imaging limitations, cognitive bias, and strategies to improve diagnostic accuracy in PE. The reviewed literature included observational studies, validation studies, systematic reviews, meta-analyses, guideline documents, and illustrative case reports.

Results: The reviewed literature suggests that PE may present across a broad and often misleading clinical spectrum. Diagnostic difficulty appears to arise from nonspecific symptoms, overlap with alternative diagnoses, imperfect performance of clinical prediction rules, limited specificity of biomarkers, interpretive limitations of electrocardiography, and technical or contextual limitations

of imaging. Cognitive biases may further contribute to diagnostic delay or initial misclassification. Evidence also suggests that probability-adjusted diagnostic approaches and careful integration of clinical assessment with imaging findings may improve diagnostic accuracy.

Conclusions: PE may mimic a wide range of other conditions and may remain unrecognized when early findings are nonspecific or misleading. A structured diagnostic approach combined with sustained clinical suspicion may help reduce missed or delayed diagnosis, particularly in patients with atypical presentations.

Keywords: pulmonary embolism; atypical presentation; diagnostic pitfalls; misdiagnosis; clinical mimicry; delayed diagnosis; narrative review

## 1. Introduction

Pulmonary embolism (PE) remains a clinically important cause of morbidity and mortality despite advances in diagnosis and treatment. Recent international mortality data suggest that PE-related mortality has declined overall during the last two decades, but this pattern has not been uniform across regions and income settings, indicating that the burden of disease likely remains substantial and unevenly distributed [1]. At the clinical level, PE continues to be difficult to recognize because its presentation is heterogeneous and often nonspecific. Large observational studies suggest that sudden dyspnea is the most frequent symptom, but chest pain, syncope, hemoptysis, cough, tachypnea, and hypoxemia may also occur, often in varying combinations [2-4]. In cardiology settings, this overlap may be especially problematic, as chest pain, chest tightness, shortness of breath, and even abdominal complaints may initially be attributed to coronary syndromes, heart failure, pulmonary infection, or other competing diagnoses [5].

This diagnostic uncertainty is central to the present review. Prior work suggests that PE may be underrecognized not only because symptoms lack specificity, but also because presentation may be atypical, asymptomatic, or masked by comorbidity [3,5,7]. Delayed diagnosis does not appear to be rare; prospective emergency department data suggest that patients whose diagnosis was made after admission were older, more likely to have altered mental status, and more likely to experience adverse in-hospital outcomes [6]. These observations support the view that PE should be approached as a disorder with broad clinical variability rather than a single stereotyped syndrome. Accordingly, this narrative review examines how PE may mimic other diseases, why diagnostic pitfalls persist, and which features from the literature may help clinicians maintain suspicion when the presentation is not classic [1-7].

## 2. Clinical Variability of Pulmonary Embolism

The clinical expression of acute pulmonary embolism (PE) is highly variable and may range from subtle exertional dyspnea to syncope, circulatory collapse, or rapidly progressive right ventricular failure. In PIOPED II, dyspnea was the most frequent symptom, but pleuritic pain, cough, tachypnea, tachycardia, orthopnea, and signs of deep venous thrombosis were also common, and no single feature was sufficiently sensitive to define the syndrome. Importantly, symptoms could be mild, oxygenation could remain relatively preserved, and even patients with proximal emboli could have a low-probability clinical assessment, underscoring that absence of classic findings does

not reliably exclude PE [8]. Clinical severity also appears to vary with thrombus burden and physiological response rather than with one uniform symptom profile alone.

This variability is further illustrated by syncope-related and high-risk presentations. In the PESIT study, PE was identified in 17.3% of patients hospitalized for a first episode of syncope, with higher prevalence among patients without an alternative explanation, suggesting that transient loss of consciousness may represent a clinically relevant presentation rather than a rare outlier [9]. At the opposite end of the spectrum, high-risk PE may present with acute dyspnea, tachycardia, hypoxemia, hypotension, right ventricular dilatation, and rapid hemodynamic deterioration, as illustrated by the intrapartum case reported by Poor et al., where PE evolved into obstructive shock requiring urgent multidisciplinary intervention [10]. Taken together, these data suggest that PE should be viewed as a syndrome of marked clinical heterogeneity, in which mild, atypical, and catastrophic presentations may all occur within the same disease process [8-10].

## 3. Typical Clinical Presentation and Diagnostic Pathway

In routine practice, the typical presentation of acute pulmonary embolism (PE) is often framed by sudden or progressive dyspnea, pleuritic chest pain, tachycardia, hypoxemia, or signs of deep venous thrombosis, but these findings usually require structured interpretation rather than symptom-based diagnosis alone. Contemporary guidelines broadly support a stepwise diagnostic approach that begins with estimation of pretest probability, most commonly with a validated rule such as the Wells or Geneva score, followed by D-dimer testing in patients with low or intermediate clinical probability and imaging in those with positive D-dimer results or higher-risk presentations [11-13]. This sequence is clinically important because prediction rules appear to perform reasonably well across care settings, although their efficiency and failure rates may vary somewhat with population and setting [12-14]. In primary care, for example, a Wells score of $\leq 4$ combined with a negative qualitative D-dimer test was reported to exclude PE with a low 3-month event rate while avoiding immediate imaging in a substantial proportion of patients [16]. Similar prospective data for the simplified Geneva score suggest that, when combined with D-dimer testing, it may offer comparable safety with the practical advantage of easier bedside use [12].

The role of D-dimer in this pathway is primarily to help exclude PE rather than confirm it. Systematic reviews suggest that D-dimer assays generally have high sensitivity but limited specificity, which makes a negative result useful in appropriately selected low-probability patients, while a positive result usually requires further imaging [15,18]. As a result, current diagnostic pathways generally reserve computed tomography pulmonary angiography (CTPA) or ventilation-perfusion imaging for patients in whom PE remains plausible after clinical probability assessment and biomarker testing [11,15,18]. CTPA is widely used because of its availability, diagnostic performance, and ability to identify alternative thoracic diagnoses, but its value also depends on technical quality and careful reporting [17,18]. Recent consensus recommendations have emphasized that CTPA should not be viewed merely as a binary test for clot detection; scan quality, level of analyzable pulmonary arteries, right ventricular findings, and alternative diagnoses may all influence interpretation and downstream management [17]. Thus, the typical diagnostic pathway for PE is best understood as an integrated process in which symptoms trigger suspicion, pretest probability guides testing, D-dimer helps avoid unnecessary imaging in selected patients, and CTPA or other imaging modalities are used selectively to confirm or refute the diagnosis [11-18].

## 4. PE Mimicking Other Diseases

One of the most clinically consequential features of pulmonary embolism (PE) is its ability to imitate other acute conditions rather than present as an immediately recognizable thromboembolic syndrome. This diagnostic ambiguity arises because PE may produce chest pain, dyspnea, biomarker elevation, fever, syncope, abdominal pain, neurological deficits, or incidental radiographic findings that are more readily attributed to cardiac, pulmonary, gastrointestinal, or neurological disease. As a result, the initial working diagnosis may follow the most familiar organ-based pattern instead of the underlying vascular process. The following subsections outline the principal ways in which PE may mimic other diseases and illustrate how these alternative clinical frames can contribute to delayed recognition or early diagnostic misclassification.

### 4.1 ACS-like presentation

Pulmonary embolism (PE) may resemble acute coronary syndrome (ACS) when chest pain, troponin elevation, and ischemic-appearing electrocardiographic changes dominate the initial presentation. Electrocardiographic abnormalities are common in PE, and patterns such as sinus tachycardia, T-wave inversion, S1Q3T3, and right bundle branch block may reflect acute right ventricular strain, although some patients may present with more misleading findings, including diffuse T-wave inversion or ST-segment changes that initially favor myocardial ischemia [19,20]. Case reports suggest that PE may occasionally present with features closely resembling unstable angina or ST-segment elevation myocardial infarction, sometimes prompting coronary evaluation before the correct diagnosis is established [21-24]. This overlap may be especially challenging in patients with pre-existing coronary disease, because symptoms and biomarkers may be attributed to ACS even when the underlying process is thromboembolic [22]. Accordingly, PE should remain in the differential diagnosis when chest pain syndromes are accompanied by unexplained hypoxemia, right heart strain, discordant coronary findings, or a clinical course that is not fully consistent with primary coronary occlusion [19-24].

### 4.2 Heart failure-like presentation

Pulmonary embolism (PE) may resemble acute heart failure when dyspnea, biomarker elevation, and evidence of right ventricular strain predominate, particularly because B-type natriuretic peptides rise in response to acute pressure overload rather than left-sided failure alone. In a meta-analysis, elevated natriuretic peptide levels in acute PE were associated with higher all-cause mortality, PE-related mortality, serious adverse events, and right ventricular dysfunction, while concurrent troponin elevation identified an even higher-risk subgroup [25]. These findings suggest that a heart failure-like biochemical profile in PE may reflect acute right ventricular overload and adverse prognosis rather than primary decompensated heart failure, and therefore natriuretic peptide elevation should be interpreted cautiously when the clinical picture is not fully explained by left ventricular dysfunction [25].

### 4.3 Pneumonia-like presentation

Pulmonary embolism (PE) may resemble pneumonia when pulmonary infarction produces pleuritic pain, fever, hemoptysis, pleural effusion, and peripheral parenchymal consolidation, particularly

in the lower lobes, where infarct-related changes are often identified on computed tomography [26-30]. This overlap appears clinically relevant because pulmonary infarction may be present in roughly one-third of acute PE cases in radiographic series, yet its recognition in the acute setting remains imperfect, as CT cannot always reliably distinguish true infarction from alveolar hemorrhage, atelectasis, or infectious consolidation [26-28,30]. In patients initially labelled as pneumonia, D-dimer may retain sensitivity for underlying PE but lacks sufficient specificity to clearly separate infarction pneumonia from community-acquired pneumonia, which may leave definitive imaging necessary in selected cases [29]. Fever may further misdirect early assessment, including occasional high-grade fever, especially when cultures are negative and antibiotics do not alter the clinical course, raising the possibility that the apparent "pneumonia" reflects thromboembolic lung injury rather than primary infection [28,29,31].

### 4.4 Syncope and collapse

Pulmonary embolism (PE) may present with syncope or collapse, but this manifestation appears to occupy an uncertain position between atypical presentation and marker of more severe cardiopulmonary compromise. Data from hospitalized PE cohorts suggest that syncope is not rare within confirmed PE and is more often associated with central embolic burden, right ventricular dysfunction, and troponin positivity, supporting a pathophysiologic link to transient hemodynamic deterioration rather than a purely incidental symptom [33,34]. At the same time, studies assessing PE prevalence among unselected patients presenting with syncope suggest that PE is an uncommon overall cause of syncope in the emergency setting, which argues against routine universal PE screening in all such patients [32]. Taken together, these findings suggest that syncope should not be viewed as either negligible or universally diagnostic; rather, it may be most informative when accompanied by dyspnea, tachycardia, right heart strain, or other features that increase pretest suspicion for PE [32-34].

### 4.5 Abdominal pain presentations

Pulmonary embolism (PE) may occasionally present with abdominal pain as the dominant or even isolated complaint, which may lead early assessment toward hepatobiliary, gastrointestinal, or other intra-abdominal diagnoses rather than thromboembolic disease. In the case report and literature review by Han and Gong, abdominal pain was described as the chief complaint in a patient initially misdiagnosed with cholecystitis and pneumonia before computed tomography pulmonary angiography established PE with pulmonary infarction; their review further suggested that abdominal pain may not be exceptionally rare, may often localize to the upper abdomen, and may be associated with lower lobe or basal pulmonary lesions, particularly when pleural or diaphragmatic irritation is present [35]. These observations suggest that PE should remain a diagnostic consideration when abdominal pain is accompanied by unexplained hypoxemia, pleural-based pulmonary findings, venous thromboembolic risk factors, or an otherwise discordant clinical course [35].

### 4.6 Neurological presentations

Pulmonary embolism (PE) may have neurological manifestations either through atypical presentation, such as syncope or seizure, or through paradoxical embolism causing cerebral ischemia. A literature review of neurological complications of PE suggests that ischemic

cerebrovascular events represent one of the more clinically significant central nervous system sequelae of venous thromboembolism, particularly in the presence of a right-to-left shunt such as a patent foramen ovale [36]. Reviews of paradoxical embolism further suggest that stroke is its most frequent systemic manifestation and that acute PE may facilitate this process by increasing right-sided pressures sufficiently to permit venous thrombi to enter the arterial circulation [37,38]. A recent case report illustrating thromboembolic stroke secondary to deep vein thrombosis and PE reinforces this mechanism and highlights how neurological deficits may be the presenting clue to an underlying venous thromboembolic process rather than a primary cerebrovascular disorder alone [39].

### 4.7 Incidental pulmonary embolism

Incidental pulmonary embolism may be identified on contrast-enhanced thoracic CT performed for reasons other than suspected thromboembolic disease, and available studies suggest that this is not a negligible phenomenon in either inpatient or outpatient populations. In a prospective inpatient study, unsuspected PE was found in 5.7% of contrast-enhanced multidetector CT examinations, with most emboli located at the segmental or subsegmental level and nearly one-third not recognized on the initial radiology report [40]. In hospital outpatients, unsuspected PE was identified in 2.23% of contrast-enhanced chest CT scans, with most cases occurring in older patients and many involving patients with known or suspected malignancy [41]. A larger retrospective analysis likewise suggested that PE may remain unsuspected in a substantial proportion of CT-detected cases and that incidental PE is particularly relevant during evaluation of acute pulmonary disease and oncologic follow-up imaging [42]. Together, these findings suggest that incidental PE represents a recurrent diagnostic scenario rather than an exceptional radiologic curiosity, and that careful review of the pulmonary arteries may be warranted even when PE is not the primary clinical indication for imaging [40-42].

## 5. Diagnostic Pitfalls

Diagnostic error in pulmonary embolism (PE) rarely arises from a single missed clue; more often it reflects the combined effect of imperfect prediction rules, nonspecific biomarkers, context-dependent imaging, and cognitive tendencies that favor more familiar alternative diagnoses. Because PE may present with symptoms and test results that are compatible with cardiac, pulmonary, or infectious disease, even a structured diagnostic pathway may remain vulnerable to misclassification when individual findings are interpreted in isolation. The following subsections summarize the major domains in which these pitfalls arise and explain how they may contribute to underrecognition, false reassurance, or delayed confirmation of PE.

### 5.1 Prediction rule limitations

Clinical prediction rules are central to the diagnostic workup of suspected pulmonary embolism, but they have important limitations that may affect individual patient classification. In a direct comparison of the revised Geneva score and the Wells rule, overall discriminatory performance was broadly similar, yet 44.7% of patients were classified differently by the two models, and in 97% of these discordant cases the difference was attributable to the subjective Wells item concerning whether an alternative diagnosis was less likely than pulmonary embolism [43]. This suggests that prediction rules may appear equivalent at the population level while still producing

meaningful variation at the bedside, particularly when subjective clinical judgment is embedded in the score. Accordingly, these tools may be most useful as structured aids to estimate pretest probability rather than as stand-alone determinants of diagnosis [43].

### 5.2 Misleading laboratory findings

Laboratory markers may support risk assessment in pulmonary embolism (PE), but they may also mislead diagnosis because they reflect right ventricular strain and myocardial injury rather than disease specificity. Troponin elevation has been associated with right ventricular dilatation, greater clot burden, adverse in-hospital events, and higher mortality in acute PE, while natriuretic peptide elevation has similarly been linked to right ventricular overload and worse short-term outcome [44-47]. However, these abnormalities may also occur in acute coronary syndromes and heart failure, so their presence does not distinguish PE from other cardiopulmonary causes of chest pain or dyspnea. Accordingly, elevated troponin or NT-proBNP should be interpreted as markers of physiological severity rather than diagnostic confirmation, particularly when the clinical picture remains ambiguous [44-47].

### 5.3 Imaging pitfalls

Imaging is indispensable in suspected pulmonary embolism (PE), but its performance depends heavily on clinical context and technical quality. Current appropriateness guidance supports computed tomography pulmonary angiography (CTPA) or ventilation-perfusion scanning as the principal imaging tests once PE remains plausible after pretest assessment, rather than as isolated screening tools divorced from probability-based evaluation [48]. Important pitfalls arise when small peripheral filling defects are overcalled, because subsegmental or very small arterial defects, particularly on lower-quality examinations, show reduced interobserver agreement and a meaningful false-positive rate, with respiratory motion artifact being a frequent cause of misinterpretation [49]. Conversely, a negative CTPA does not appear uniformly reassuring across all patient groups: in patients with high clinical probability, normal multidetector CTPA alone may fail to exclude PE with sufficient confidence, especially when no convincing alternative diagnosis is identified [50]. This concern is reinforced by meta-analytic data suggesting that the cumulative occurrence of venous thromboembolism after a negative CTPA rises with pretest PE prevalence and may reach clinically relevant levels in the highest-risk subgroups, indicating that CTPA may be insufficient as a stand-alone rule-out test in that setting [51].

### 5.4 Cognitive Bias

Cognitive bias may contribute to missed or delayed recognition of pulmonary embolism (PE), particularly because its presentation often overlaps with more common cardiopulmonary disorders. Studies of diagnostic delay in primary care suggest that delay is not uncommon and may be more likely in older patients, in those without typical chest symptoms, and in those initially thought to have another respiratory diagnosis, such as infection, all of which may favor premature attribution away from PE [52,53]. More broadly, emergency medicine literature suggests that diagnostic errors are frequently linked to cognitive processes such as anchoring, premature closure, confirmation bias, and availability bias, especially in busy and interrupted clinical environments [54,56]. The potential relevance of availability bias to PE is supported by evidence that physicians may transiently increase PE testing after recently diagnosing a PE case, suggesting that recent

experience can influence subsequent risk estimation [55]. Taken together, these findings suggest that cognitive bias may not only affect formal diagnostic reasoning, but may also shape when PE is considered at all [52-56].

## 6. Strategies to Reduce Misdiagnosis

Strategies to reduce misdiagnosis of pulmonary embolism may depend less on adding isolated tests and more on improving how pretest probability and D-dimer are integrated into individualized decision-making. A patient-level meta-analysis by van Es et al. suggested that absolute probability models using objective clinical variables together with quantitative D-dimer may discriminate better than some traditional rule-based approaches and may identify patients whose individualized risk remains clinically relevant despite classification as low risk by standard algorithms [57]. Likewise, the PEGeD study suggested that adjusting D-dimer thresholds to clinical probability could safely reduce unnecessary chest imaging in selected outpatients, particularly in those with low clinical pretest probability [58]. Taken together, these studies suggest that misdiagnosis may be reduced by using structured probability-based pathways, interpreting D-dimer in context rather than in isolation, and preserving diagnostic flexibility when standardized algorithms and bedside clinical judgment appear discordant [57,58].

## 7. Delayed Presentation

Diagnostic delay is an important contextual issue in pulmonary embolism because delayed presentation appears to be common and may further complicate an already nonspecific clinical syndrome. In a large analysis of 4,044 patients with suspected pulmonary embolism, delayed presentation beyond 7 days was observed in 18.6% of cases and was associated with older age, cardiopulmonary comorbidity, and fewer classic venous thromboembolic risk factors, suggesting that overlap with other chronic conditions may contribute to later recognition [59]. Importantly, delayed presentation was also associated with a higher proportion of centrally located emboli, although among hemodynamically stable patients who survived to diagnosis, short-term recurrent venous thromboembolism and mortality did not differ clearly from those diagnosed earlier [59]. These findings support the broader interpretation that pulmonary embolism may remain unrecognized not only because of atypical symptom patterns, but also because its clinical course may evolve against a background of competing explanations that delay suspicion and testing [59].

## 8. Conclusion

Pulmonary embolism remains a diagnostically challenging disorder because it may present with typical respiratory complaints, mimic other acute diseases, or be detected incidentally, and this heterogeneity may contribute to delayed or missed diagnosis. The literature reviewed here suggests that diagnostic error may arise not from a single weakness, but from the interaction of nonspecific symptoms, imperfect prediction tools, potentially misleading biomarkers, imaging limitations, and cognitive bias. For this reason, PE may be approached most safely through structured probability-based evaluation, careful interpretation of test results in context, and continued diagnostic flexibility when the initial working diagnosis does not fully explain the presentation. Contemporary guideline frameworks likewise emphasize integrated assessment, risk stratification, and context-sensitive use of imaging and biomarkers, which may help reduce underrecognition while avoiding unnecessary testing [60].

## Disclosure

### Author's contribution

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The authors deny any conflict of interest.

## References

[1] Hagiya H, Harada K, Nishimura Y, Yamamoto M, Nishimura S, Yamamoto M, Niimura T, Osaki Y, Vu QT, Fujii M, Sako N, Takeda T, Hamano H, Zamami Y, Koyama T. Global trends in mortality related to pulmonary embolism: an epidemiological analysis of data from the World Health Organization mortality database from 2001 to 2023. EClinicalMedicine. 2025 Jul 31;86:103389. doi: 10.1016/j.eclinm.2025.103389. PMID: 40791890; PMCID: PMC12336653.

[2] Miniati M, Cenci C, Monti S, Poli D. Clinical presentation of acute pulmonary embolism: survey of 800 cases. PLoS One. 2012;7(2):e30891. doi: 10.1371/journal.pone.0030891. Epub 2012 Feb 27. PMID: 22383978; PMCID: PMC3288010.

[3] Khasin M, Gur I, Evgrafov EV, Toledano K, Zalts R. Clinical presentations of acute pulmonary embolism: A retrospective cohort study. Medicine (Baltimore). 2023 Jul 14;102(28):e34224. doi: 10.1097/MD.0000000000034224. PMID: 37443506; PMCID: PMC10344497.

[4] Ji QY, Wang MF, Su CM, Yang QF, Feng LF, Zhao LY, Fang SY, Zhao FH, Li WM. Clinical symptoms and related risk factors in pulmonary embolism patients and cluster analysis based on these symptoms. Sci Rep. 2017 Nov 2;7(1):14887. doi: 10.1038/s41598-017-14888-7. PMID: 29097743; PMCID: PMC5668424.

[5] Yao D, Cao W, Liu X. Clinical manifestations and misdiagnosis factors of pulmonary embolism patients seeking treatment in cardiology. Medicine (Baltimore). 2024 Dec 6;103(49):e40821. doi: 10.1097/MD.0000000000040821. PMID: 39654238; PMCID: PMC11630920.

[6] Kline JA, Hernandez-Nino J, Jones AE, Rose GA, Norton HJ, Camargo CA Jr. Prospective study of the clinical features and outcomes of emergency department patients with delayed diagnosis of pulmonary embolism. Acad Emerg Med. 2007 Jul;14(7):592-8. doi: 10.1197/j.aem.2007.03.1356. Epub 2007 Jun 6. PMID: 17554011.

[7] Ishaaya E, Tapson VF. Advances in the diagnosis of acute pulmonary embolism. F1000Res. 2020 Jan 24;9:F1000 Faculty Rev-44. doi: 10.12688/f1000research.21347.1. PMID: 32047618; PMCID: PMC6993831.

[8] Stein PD, Beemath A, Matta F, Weg JG, Yusen RD, Hales CA, Hull RD, Leeper KV Jr, Sostman HD, Tapson VF, Buckley JD, Gottschalk A, Goodman LR, Wakefied TW, Woodard PK. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med. 2007 Oct;120(10):871-9. doi: 10.1016/j.amjmed.2007.03.024. PMID: 17904458; PMCID: PMC2071924.

[9] Prandoni P, Lensing AW, Prins MH, Ciammaichella M, Perlati M, Mumoli N, Bucherini E, Visonà A, Bova C, Imberti D, Campostrini S, Barbar S; PESIT Investigators. Prevalence of Pulmonary Embolism among Patients Hospitalized for Syncope. N Engl J Med. 2016 Oct 20;375(16):1524-1531. doi: 10.1056/NEJMoa1602172. PMID: 27797317.

[10] Poor H, Serrao G, Grapsa J, Chandrashekhar YS, Bianco A, Lookstein RA, Fuster V. High-Risk Pulmonary Embolism During Labor: JACC Patient Care Pathways. J Am Coll Cardiol. 2023 Jan 24;81(3):283-291. doi: 10.1016/j.jacc.2022.10.004. Epub 2022 Oct 17. PMID: 36265527.

[11] Zuin M, Bikdeli B, Ballard-Hernandez J, Barco S, Battinelli EM, Giannakoulas G, Jimenez D, Klok FA, Krishnathasan D, Lang IM, Moores L, Sylvester KW, Weitz JI, Piazza G. International Clinical Practice Guideline Recommendations for Acute Pulmonary Embolism: Harmony, Dissonance, and Silence. J Am Coll Cardiol. 2024 Oct 15;84(16):1561-1577. doi: 10.1016/j.jacc.2024.07.044. PMID: 39384264.

[12] Robert-Ebadi H, Mostaguir K, Hovens MM, Kare M, Verschuren F, Girard P, Huisman MV, Moustafa F, Kamphuisen PW, Buller HR, Righini M, Le Gal G. Assessing clinical probability of pulmonary embolism: prospective validation of the simplified Geneva score. J Thromb Haemost. 2017 Sep;15(9):1764-1769. doi: 10.1111/jth.13770. Epub 2017 Aug 17. PMID: 28688113.

[13] Gibson NS, Sohne M, Kruip MJ, Tick LW, Gerdes VE, Bossuyt PM, Wells PS, Buller HR; Christopher study investigators. Further validation and simplification of the Wells clinical decision rule in pulmonary embolism. Thromb Haemost. 2008 Jan;99(1):229-34. doi: 10.1160/TH07-05-0321. PMID: 18217159.

[14] Hendriksen JM, Geersing GJ, Lucassen WA, Erkens PM, Stoffers HE, van Weert HC, Büller HR, Hoes AW, Moons KG. Diagnostic prediction models for suspected pulmonary embolism:

systematic review and independent external validation in primary care. BMJ. 2015 Sep 8;351:h4438. doi: 10.1136/bmj.h4438. PMID: 26349907; PMCID: PMC4561760.

[15] Crawford F, Andras A, Welch K, Sheares K, Keeling D, Chappell FM. D-dimer test for excluding the diagnosis of pulmonary embolism. Cochrane Database Syst Rev. 2016 Aug 5;2016(8):CD010864. doi: 10.1002/14651858.CD010864.pub2. PMID: 27494075; PMCID: PMC6457638.

[16] Geersing GJ, Erkens PM, Lucassen WA, Büller HR, Cate HT, Hoes AW, Moons KG, Prins MH, Oudega R, van Weert HC, Stoffers HE. Safe exclusion of pulmonary embolism using the Wells rule and qualitative D-dimer testing in primary care: prospective cohort study. BMJ. 2012 Oct 4;345:e6564. doi: 10.1136/bmj.e6564. PMID: 23036917; PMCID: PMC3464185.

[17] Klok FA, Barco S, Bertoletti L, Bhalla S, Dubois S, Le Gal G, Haramati LB, van Hooidonk E, Humbert M, Konstantinides SV, Lang IM, Murphy WS, Ohno Y, Price S, Prokop M, Pruszczyk P, Rossi A, Thistlethwaite PA, West C, Remy-Jardin M. Optimal Approach to Performing and Reporting Computed Tomography Angiography for Suspected Acute Pulmonary Embolism: A Clinical Consensus Statement of the ESC Working Group on Pulmonary Circulation & Right Ventricular Function, the Fleischner Society, the Association for Acute Cardiovascular Care (ACVC) and the European Association of Cardiovascular Imaging (EACVI) of the ESC, Endorsed by European Respiratory Society (ERS), Asian Society of Thoracic Radiology (ASTR), European Society of Thoracic Imaging (ESTI), and Society of Thoracic Radiology (STR). Radiology. 2025 Jun;315(3):e243833. doi: 10.1148/radiol.243833. PMID: 40459417.

[18] Patel P, Patel P, Bhatt M, Braun C, Begum H, Wiercioch W, Varghese J, Wooldridge D, Alturkmani H, Thomas M, Baig M, Bahaj W, Khatib R, Kehar R, Ponnapureddy R, Sethi A, Mustafa A, Lim W, Le Gal G, Bates SM, Haramati LB, Kline J, Lang E, Righini M, Kalot MA, Husainat NM, Jabiri YNA, Schünemann HJ, Mustafa RA. Systematic review and meta-analysis of test accuracy for the diagnosis of suspected pulmonary embolism. Blood Adv. 2020 Sep 22;4(18):4296-4311. doi: 10.1182/bloodadvances.2019001052. PMID: 32915980; PMCID: PMC7509887.

[19] Krintratun S, Srichuachom W, Wongtanasarasin W. Prevalence of Electrocardiographic Abnormalities in Patients with Acute Pulmonary Embolism: A Systematic Review and Meta-

Analysis. J Clin Med. 2025 Jul 4;14(13):4750. doi: 10.3390/jcm14134750. PMID: 40649124; PMCID: PMC12251390.

[20] Hariharan P, Dudzinski DM, Okechukwu I, Takayesu JK, Chang Y, Kabrhel C. Association between electrocardiographic findings, right heart strain, and short-term adverse clinical events in patients with acute pulmonary embolism. Clin Cardiol. 2015 Apr;38(4):236-42. doi: 10.1002/clc.22383. Epub 2015 Apr 2. PMID: 25847482; PMCID: PMC6711008.

[21] Hamad M, Edwards G, Sharma A, Dasuqi S. Massive pulmonary embolism masquerading as acute coronary syndrome. Clin Med (Lond). 2026 Jan 21;26(2):100559. doi: 10.1016/j.clinme.2026.100559. Epub ahead of print. PMID: 41577138; PMCID: PMC12907221.

[22] Chen YH, Zhu XY, Fan LH, Xu HF. Pulmonary embolism in patients with chronic coronary syndrome masquerading as acute coronary syndrome: a case report and literature review. BMC Cardiovasc Disord. 2024 Jul 1;24(1):331. doi: 10.1186/s12872-024-03998-6. PMID: 38951773; PMCID: PMC11218115.

[23] Egini O, Dufresne A, Khalid M, Egini C, Jaffe E. Case Report: Diffuse T wave inversions as initial electrocardiographic evidence in acute pulmonary embolism. F1000Res. 2018 Jun 13;7:738. doi: 10.12688/f1000research.14927.2. PMID: 30356445; PMCID: PMC6178910.

[24] Yue XL, Shi XY, Jiang M, Li RJ. Acute pulmonary embolism presenting with electrocardiographic signs and serum biomarkers of ST-segment elevation myocardial infarction: a case report. J Int Med Res. 2023 Sep;51(9):3000605231197063. doi: 10.1177/03000605231197063. PMID: 37677144; PMCID: PMC10492495.

[25] Lega JC, Lacasse Y, Lakhal L, Provencher S. Natriuretic peptides and troponins in pulmonary embolism: a meta-analysis. Thorax. 2009 Oct;64(10):869-75. doi: 10.1136/thx.2008.110965. Epub 2009 Jun 11. PMID: 19525265.

[26] Kaptein FHJ, Kroft LJM, Hammerschlag G, Ninaber MK, Bauer MP, Huisman MV, Klok FA. Pulmonary infarction in acute pulmonary embolism. Thromb Res. 2021 Jun;202:162-169. doi: 10.1016/j.thromres.2021.03.022. Epub 2021 Apr 1. PMID: 33862471.

[27] Kaptein FHJ, Stöger JL, van Dam LF, Ninaber MK, Mertens BJA, Huisman MV, Klok FA, Kroft LJM. Computed tomography diagnosis of pulmonary infarction in acute pulmonary embolism. Thromb Res. 2024 Sep;241:109071. doi: 10.1016/j.thromres.2024.109071. Epub 2024 Jun 26. PMID: 38972272.

[28] Gagno G, Padoan L, D'Errico S, Baratella E, Radaelli D, Fluca AL, Pierri A, Janjusevic M, Aleksova Noveska E, Cova MA, Copetti R, Cominotto F, Sinagra G, Aleksova A. Pulmonary Embolism Presenting with Pulmonary Infarction: Update and Practical Review of Literature Data. J Clin Med. 2022 Aug 21;11(16):4916. doi: 10.3390/jcm11164916. PMID: 36013155; PMCID: PMC9409643.

[29] Paparoupa M, Spineli L, Framke T, Ho H, Schuppert F, Gillissen A. Pulmonary Embolism in Pneumonia: Still a Diagnostic Challenge? Results of a Case-Control Study in 100 Patients. Dis Markers. 2016;2016:8682506. doi: 10.1155/2016/8682506. Epub 2016 May 25. PMID: 27313336; PMCID: PMC4897677.

[30] Lio KU, O'Corragain O, Bashir R, Brosnahan S, Cohen G, Lakhter V, Panaro J, Rivera-Lebron B, Rali P. Clinical outcomes and factors associated with pulmonary infarction following acute pulmonary embolism: a retrospective observational study at a US academic centre. BMJ Open. 2022 Dec 29;12(12):e067579. doi: 10.1136/bmjopen-2022-067579. PMID: 36581412; PMCID: PMC9806066.

[31] Saad M, Shaikh DH, Adrish M. A rare case report of a saddle pulmonary embolism presenting with high grade fevers, responsive to anticoagulation. Medicine (Baltimore). 2018 Mar;97(9):e0002. doi: 10.1097/MD.0000000000010002. PMID: 29489640; PMCID: PMC5851743.

[32] Badertscher P, du Fay de Lavallaz J, Hammerer-Lercher A, Nestelberger T, Zimmermann T, Geiger M, Imahorn O, Miró Ò, Salgado E, Christ M, Cullen L, Than M, Martin-Sanchez FJ, Di Somma S, Peacock WF, Keller DI, Costabel JP, Walter J, Boeddinghaus J, Twerenbold R, Méndez A, Gospodinov B, Puelacher C, Wussler D, Koechlin L, Kawecki D, Geigy N, Strebel I, Lohrmann J, Kühne M, Reichlin T, Mueller C; BASEL IX Investigators. Prevalence of Pulmonary Embolism in Patients With Syncope. J Am Coll Cardiol. 2019 Aug 13;74(6):744-754. doi: 10.1016/j.jacc.2019.06.020. PMID: 31395124.

[33] Altınsoy B, Erboy F, Tanrıverdi H, Uygur F, Örnek T, Atalay F, Tor M. Syncope as a presentation of acute pulmonary embolism. Ther Clin Risk Manag. 2016 Jun 27;12:1023-8. doi: 10.2147/TCRM.S105722. PMID: 27390523; PMCID: PMC4930221.

[34] Keller K, Hobohm L, Münzel T, Ostad MA, Espinola-Klein C. Syncope in haemodynamically stable and unstable patients with acute pulmonary embolism - Results of the German nationwide inpatient sample. Sci Rep. 2018 Oct 25;8(1):15789. doi: 10.1038/s41598-018-33858-1. PMID: 30361542; PMCID: PMC6202331.

[35] Han Y, Gong Y. Pulmonary embolism with abdominal pain as the chief complaint: A case report and literature review. Medicine (Baltimore). 2019 Nov;98(44):e17791. doi: 10.1097/MD.0000000000017791. PMID: 31689854; PMCID: PMC6946195.

[36] Desai PV, Krepostman N, Collins M, De Sirkar S, Hinkleman A, Walsh K, Fareed J, Darki A. Neurological Complications of Pulmonary Embolism: a Literature Review. Curr Neurol Neurosci Rep. 2021 Oct 20;21(10):59. doi: 10.1007/s11910-021-01145-8. PMID: 34669060; PMCID: PMC8526526.

[37] Windecker S, Stortecky S, Meier B. Paradoxical embolism. J Am Coll Cardiol. 2014 Jul 29;64(4):403-15. doi: 10.1016/j.jacc.2014.04.063. PMID: 25060377.

[38] Jolobe O. Wide-ranging clinical spectrum of paradoxical embolism. Postgrad Med J. 2022 Dec;98(1166):958-966. doi: 10.1136/postgradmedj-2020-139691. Epub 2021 May 18. PMID: 34006630.

[39] Chen Y, Yin H, Kong BB, Wu S, Zhang P. Paradoxical embolism: A case of thromboembolic stroke secondary to pulmonary embolism and deep vein thrombosis. J Int Med Res. 2025 Nov;53(11):3000605251394958. doi: 10.1177/03000605251394958. Epub 2025 Nov 13. PMID: 41233288; PMCID: PMC12618822.

[40] Ritchie G, McGurk S, McCreath C, Graham C, Murchison JT. Prospective evaluation of unsuspected pulmonary embolism on contrast enhanced multidetector CT (MDCT) scanning. Thorax. 2007 Jun;62(6):536-40. doi: 10.1136/thx.2006.062299. Epub 2006 Dec 8. PMID: 17158631; PMCID: PMC2117210.

[41] Farrell C, Jones M, Girvin F, Ritchie G, Murchison JT. Unsuspected pulmonary embolism identified using multidetector computed tomography in hospital outpatients. Clin Radiol. 2010 Jan;65(1):1-5. doi: 10.1016/j.crad.2009.09.003. Epub 2009 Oct 28. PMID: 20103414.

[42] Bach AG, Meyer HJ, Taute BM, Surov A. The frequency of incidental pulmonary embolism in different CT examinations. Br J Radiol. 2016;89(1058):20150737. doi: 10.1259/bjr.20150737. Epub 2015 Nov 26. PMID: 26607645; PMCID: PMC4985219.

[43] Klok FA, Kruisman E, Spaan J, Nijkeuter M, Righini M, Aujesky D, Roy PM, Perrier A, Le Gal G, Huisman MV. Comparison of the revised Geneva score with the Wells rule for assessing clinical probability of pulmonary embolism. J Thromb Haemost. 2008 Jan;6(1):40-4. doi: 10.1111/j.1538-7836.2007.02820.x. Epub 2007 Oct 29. PMID: 17973649.

[44] Meyer T, Binder L, Hruska N, Luthe H, Buchwald AB. Cardiac troponin I elevation in acute pulmonary embolism is associated with right ventricular dysfunction. J Am Coll Cardiol. 2000 Nov 1;36(5):1632-6. doi: 10.1016/s0735-1097(00)00905-0. PMID: 11079669.

[45] Konstantinides S, Geibel A, Olschewski M, Kasper W, Hruska N, Jäckle S, Binder L. Importance of cardiac troponins I and T in risk stratification of patients with acute pulmonary embolism. Circulation. 2002 Sep 3;106(10):1263-8. doi: 10.1161/01.cir.0000028422.51668.a2. PMID: 12208803.

[46] El-Menyar A, Asim M, Nabir S, Ahmed MN, Al-Thani H. Implications of elevated cardiac troponin in patients presenting with acute pulmonary embolism: an observational study. J Thorac Dis. 2019 Aug;11(8):3302-3314. doi: 10.21037/jtd.2019.08.53. PMID: 31559033; PMCID: PMC6753431.

[47] Pruszczyk P, Kostrubiec M, Bochowicz A, Styczyński G, Szulc M, Kurzyna M, Fijałkowska A, Kuch-Wocial A, Chlewicka I, Torbicki A. N-terminal pro-brain natriuretic peptide in patients with acute pulmonary embolism. Eur Respir J. 2003 Oct;22(4):649-53. doi: 10.1183/09031936.03.00023303. PMID: 14582919.

[48] Expert Panel on Cardiac Imaging; Kirsch J, Wu CC, Bolen MA, Henry TS, Rajiah PS, Brown RKJ, Galizia MS, Lee E, Rajesh F, Raptis CA, Rybicki FJ, Sams CM, Verde F, Villines TC, Wolf SJ, Yu J, Donnelly EF, Abbara S. ACR Appropriateness Criteria® Suspected Pulmonary

Embolism: 2022 Update. J Am Coll Radiol. 2022 Nov;19(11S):S488-S501. doi: 10.1016/j.jacr.2022.09.014. PMID: 36436972.

[49] Miller WT Jr, Marinari LA, Barbosa E Jr, Litt HI, Schmitt JE, Mahne A, Lee V, Akers SR. Small pulmonary artery defects are not reliable indicators of pulmonary embolism. Ann Am Thorac Soc. 2015 Jul;12(7):1022-9. doi: 10.1513/AnnalsATS.201502-105OC. PMID: 25961445.

[50] Moores L, Kline J, Portillo AK, Resano S, Vicente A, Arrieta P, Corres J, Tapson V, Yusen RD, Jiménez D. Multidetector computed tomographic pulmonary angiography in patients with a high clinical probability of pulmonary embolism. J Thromb Haemost. 2016 Jan;14(1):114-20. doi: 10.1111/jth.13188. Epub 2015 Dec 14. PMID: 26559176.

[51] Belzile D, Jacquet S, Bertoletti L, Lacasse Y, Lambert C, Lega JC, Provencher S. Outcomes following a negative computed tomography pulmonary angiography according to pulmonary embolism prevalence: a meta-analysis of the management outcome studies. J Thromb Haemost. 2018 Jun;16(6):1107-1120. doi: 10.1111/jth.14021. Epub 2018 May 17. PMID: 29645405.

[52] Walen S, Damoiseaux RA, Uil SM, van den Berg JW. Diagnostic delay of pulmonary embolism in primary and secondary care: a retrospective cohort study. Br J Gen Pract. 2016 Jun;66(647):e444-50. doi: 10.3399/bjgp16X685201. Epub 2016 Apr 25. PMID: 27114207; PMCID: PMC4871310.

[53] Hendriksen JM, Koster-van Ree M, Morgenstern MJ, Oudega R, Schutgens RE, Moons KG, Geersing GJ. Clinical characteristics associated with diagnostic delay of pulmonary embolism in primary care: a retrospective observational study. BMJ Open. 2017 Mar 9;7(3):e012789. doi: 10.1136/bmjopen-2016-012789. PMID: 28279993; PMCID: PMC5353317.

[54] Kunitomo K, Harada T, Watari T. Cognitive biases encountered by physicians in the emergency room. BMC Emerg Med. 2022 Aug 26;22(1):148. doi: 10.1186/s12873-022-00708-3. PMID: 36028810; PMCID: PMC9414136.

[55] Ly DP. The Influence of the Availability Heuristic on Physicians in the Emergency Department. Ann Emerg Med. 2021 Nov;78(5):650-657. doi: 10.1016/j.annemergmed.2021.06.012. Epub 2021 Aug 6. PMID: 34373141.

[56] Watari T, Tokuda Y, Amano Y, Onigata K, Kanda H. Cognitive Bias and Diagnostic Errors among Physicians in Japan: A Self-Reflection Survey. Int J Environ Res Public Health. 2022 Apr 12;19(8):4645. doi: 10.3390/ijerph19084645. PMID: 35457511; PMCID: PMC9032995.

[57] van Es N, Takada T, Kraaijpoel N, Klok FA, Stals MAM, Büller HR, Courtney DM, Freund Y, Galipienzo J, Le Gal G, Ghanima W, Huisman MV, Kline JA, Moons KGM, Parpia S, Perrier A, Righini M, Robert-Ebadi H, Roy PM, Wells PS, de Wit K, van Smeden M, Geersing GJ. Diagnostic management of acute pulmonary embolism: a prediction model based on a patient data meta-analysis. Eur Heart J. 2023 Aug 22;44(32):3073-3081. doi: 10.1093/eurheartj/ehad417. PMID: 37452732; PMCID: PMC10917087.

[58] Kearon C, de Wit K, Parpia S, Schulman S, Afilalo M, Hirsch A, Spencer FA, Sharma S, D'Aragon F, Deshaies JF, Le Gal G, Lazo-Langner A, Wu C, Rudd-Scott L, Bates SM, Julian JA; PEGeD Study Investigators. Diagnosis of Pulmonary Embolism with d-Dimer Adjusted to Clinical Probability. N Engl J Med. 2019 Nov 28;381(22):2125-2134. doi: 10.1056/NEJMoa1909159. PMID: 31774957.

[59] den Exter PL, van Es J, Erkens PM, van Roosmalen MJ, van den Hoven P, Hovens MM, Kamphuisen PW, Klok FA, Huisman MV. Impact of delay in clinical presentation on the diagnostic management and prognosis of patients with suspected pulmonary embolism. Am J Respir Crit Care Med. 2013 Jun 15;187(12):1369-73. doi: 10.1164/rccm.201212-2219OC. PMID: 23590273.

[60] Writing Committee Members; Creager MA, Barnes GD, Giri J, Mukherjee D, Jones WS, Burnett AE, Carman T, Casanegra AI, Castellucci LA, Clark SM, Cushman M, de Wit K, Eaves JM, Fang MC, Goldberg JB, Henkin S, Johnston-Cox H, Kadavath S, Kadian-Dodov D, Keeling WB, Klein AJP, Li J, McDaniel MC, Moores LK, Piazza G, Prenger KS, Pugliese SC, Ranade M, Rosovsky RP, Russo F, Secemsky EA, Sista AK, Tefera L, Weinberg I, Westafer LM, Young MN. 2026 AHA/ACC/ACCP/ACEP/CHEST/SCAI/SHM/SIR/SVM/SVN Guideline for the Evaluation and Management of Acute Pulmonary Embolism in Adults: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2026 Mar 24;153(12):e977-e1051. doi: 10.1161/CIR.0000000000001415. Epub 2026 Feb 19. PMID: 41712677.