

Pulmonary Embolism With Pulmonary Infarction Despite Low-Risk Prediction Scores: Atypical Pleuritic, Abdominal, and Shoulder Pain

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Abstract

Clinical prediction tools for pulmonary embolism (PE), such as the Pulmonary Embolism Rule-out Criteria (PERC) rule, Wells score, and Geneva score, are designed to detect the classically described clinical signs and risk factors of PE and do not account for pleuritic pain syndrome, which can be the leading clinical presentation when PE causes pulmonary infarction (PI). We describe a 27-year-old male driver and hookah user who presented with dyspnea, pleuritic chest pain, back pain, and abdominal pain - atypical features that initially raised concern for aortic dissection. Clinical prediction scores yielded a low pretest probability despite subsequently confirmed PE, and D-dimer was near-threshold. CT angiography revealed subsegmental PE with PI.

Categories: Hematology, Pulmonology, Internal Medicine

Keywords: atypical presentation, d-dimer, geneva score, perc rule, pulmonary embolism, pulmonary infarction, right shoulder pain, ruq pain, wells score

Introduction

Clinical prediction tools such as the Pulmonary Embolism Rule-out Criteria (PERC) rule [1], Wells score [2], and revised Geneva score [3] are widely used to support pulmonary embolism (PE) risk stratification in the emergency department [4]. These instruments incorporate selected clinical findings and risk factors associated with PE but may not capture all clinical manifestations of PE, particularly presentations dominated by pleuritic pain. Although the specific variables and their weighting differ among these tools, common elements include heart rate, hemoptysis, and signs or history of deep vein thrombosis (DVT), along with risk factors such as recent immobilization or surgery. The PERC rule additionally incorporates oxygen saturation, Wells score incorporates a clinical probability assessment of whether PE is the leading diagnosis, and the Wells and Geneva scores additionally account for malignancy. Pulmonary infarction (PI) occurs in up to 30% of patients with PE and is more commonly associated with distal or subsegmental emboli [5]. When PI occurs, pleural inflammation and peripheral parenchymal injury may produce a predominantly pleuritic pain syndrome that falls outside the variables these prediction tools were designed to assess. We present a case of PE with PI in which pleuritic chest, back, abdominal, and referred shoulder pain broadened the initial differential diagnosis and contributed to diagnostic uncertainty despite low-risk clinical prediction scores.

Case Presentation

A 27-year-old man with childhood asthma (not on any treatment) presented to the emergency department with two days of progressive dyspnea and pleuritic left-sided chest and back pain. He also reported right shoulder pain and mild nausea. He denied fever, cough, leg swelling, or personal or family history of thromboembolism.

His occupational history was significant for working as a driver, requiring him to remain seated behind the wheel without standing or ambulating for stretches of up to six hours at a time, across shifts occurring up to six days per week. He reported regular hookah use on weekends and denied cigarette smoking or illicit drug use. There was no family history of inherited thrombophilia, and no other provoking factors for venous thromboembolism were identified.

The vital signs on arrival were as follows: temperature 98.3°F, heart rate 81 bpm, respiratory rate 16/min, blood pressure 144/80 mmHg, oxygen saturation 100% on room air. Examination showed mild respiratory distress with pain-limited inspiratory effort. Lungs were clear to auscultation. Cardiovascular, abdominal, musculoskeletal, and neurologic exams were otherwise unremarkable.

Venous blood gas demonstrated acute respiratory acidosis, with a calculated bicarbonate of 27.4 mmol/L (pH 7.26, pCO₂ 63.1 mmHg). Serum bicarbonate on the concurrent basic metabolic panel was 22 mmol/L (this difference likely reflects the distinct methodology rather than a physiologic discrepancy). Lactate was 2.8 mmol/L. D-dimer, reported in D-dimer units (DDU), was 239 ng/mL, marginally above this assay's laboratory

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reference range (upper limit 230 ng/mL DDU) but below the conventional PE rule-out threshold for this assay (~250 ng/mL DDU, equivalent to 500 ng/mL fibrinogen equivalent units (FEU)). Troponin T was <6 ng/L; pro-BNP <36 pg/mL. The laboratory values on admission are presented in Table 1.

Parameter	Patient Value	Reference Range
<i>Complete Blood Count</i>		
WBC count	10.2×10 ³ /μL	4.8-10.8×10 ³ /μL
RBC count	6.06×10 ⁶ /μL	4.50-5.90×10 ⁶ /μL
Hemoglobin	17.3	12.0-16.0 g/dL
Hematocrit	50.6	42.0-51.0%
MCV	83.5	80.0-96.0 fL
MCH	28.5	27.0-33.0 pg
MCHC	34.2	33.0-36.0 g/dL
MPV	7.2	8.0-12.0 fL
RDW	13.8	10.5-14.5%
Platelet count	173×10 ³ /μL	150-400×10 ³ /μL
Neutrophils	56.9% (5.8×10 ³ /μL)	40.0-70.0% (1.5-8.0×10 ³ /μL)
Lymphocytes	35.1% (3.6×10 ³ /μL)	20.0–50.0% (1.0-4.8×10 ³ /μL)
Monocytes	7.3% (0.7×10 ³ /μL)	1.0–8.0% (0.3-0.5×10 ³ /μL)
Eosinophils	0.5% (0.005×10 ³ /μL)	≤5.0% (0.05-0.25×10 ³ /μL)
Basophils	0.2% (0.002×10 ³ /μL)	≤2.0% (≤2.0×10 ³ /μL)
<i>Basic Metabolic Panel</i>		
Sodium	138	135-145 mEq/L
Potassium	4.1	3.5-5.0 mEq/L
Bicarbonate	22	24-30 mEq/L
Chloride	102	98-108 mEq/L
Glucose	98	70-120 mg/dL
Blood urea nitrogen	12.0	8.0-26.0 mg/dL
Creatinine	1.0	0.5-1.5 mg/dL
Calcium, total	9.2	8.5-10.5 mg/dL
Anion gap	14	9-15 mmol/L
<i>Hepatic Function Panel</i>		
Albumin	4.6	3.4-4.8 g/dL
Total bilirubin	1	0.2-1.2 mg/dL
Direct (conjugated) bilirubin	Specimen hemolyzed	0.0-0.3 mg/dL
Alkaline phosphatase	151	53-128 U/L
AST	24	9-48 U/L
ALT	35	5-40 U/L
Total protein	7.8	6.0-8.5 g/dL
<i>Lipid Panel</i>		
Total cholesterol	186	133-240 mg/dL

Triglycerides	158	46-150 mg/dL
HDL cholesterol	34	29-63 mg/dL
LDL cholesterol	120	≤160 mg/dL
<i>Cardiac and Coagulation Markers</i>		
NT-proBNP	<36	0-125 pg/mL
D-dimer	239	0-230 ng/mL
Troponin T	<6	≤12 ng/L
<i>Venous Blood Gas (room air, FiO₂ 21%)</i>		
pH	7.261	7.350-7.450
PCO ₂	63.1	35.0–45.0 mmHg
Base excess	1.10	-3.00 to 3.00 mmol/L
Bicarbonate	27.4	22.0-28.0 mmol/L
Ionized calcium	1.27	1.15-1.29 mmol/L
Lactic acid	2.8	0.5-1.6 mmol/L

TABLE 1: Laboratory values on admission

AST: Aspartate transaminase; ALT: alanine aminotransferase; NT-proBNP: N-terminal pro B-type natriuretic peptide; pH: power of hydrogen; HDL: high-density lipoprotein; LDL: low-density lipoprotein; PCO₂: partial pressure of carbon dioxide.

Chest radiograph showed bilateral basilar airspace opacities (Figure 1). ECG demonstrated normal sinus rhythm with 79 bpm (Figure 2).

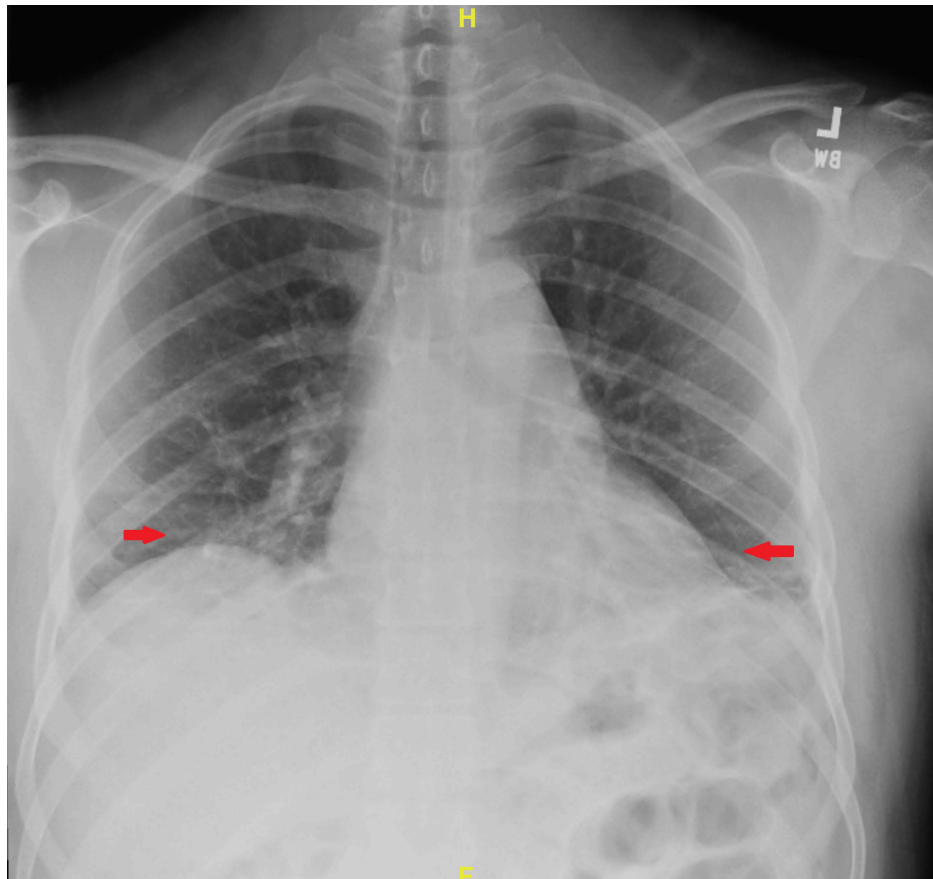


FIGURE 1: Posteroanterior chest X-ray with bilateral basilar airspace opacities (red arrows)

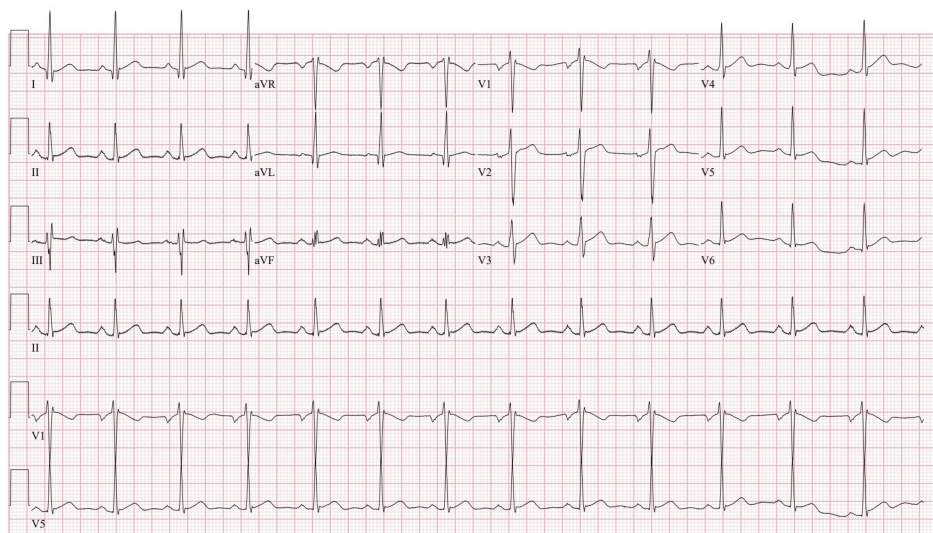


FIGURE 2: Electrocardiogram. Normal sinus rhythm, 79 bpm.

The Wells and Geneva scores were both calculated, yielding a Wells score of 0 and a Geneva score of 3 (heart rate 75-94 bpm), both indicating a low probability of PE. Although the PERC rule is not required once Wells and Geneva scores have been calculated, we applied it here for completeness, and it yielded a score of 0, satisfying rule-out criteria.

Notably, while in the emergency department (ED), he developed acute severe right upper quadrant (RUQ) abdominal pain with guarding. This, along with back pain and dyspnea, raised suspicion for aortic dissection

and prompted a dedicated CT angiography protocol of the chest, abdomen, and pelvis.

Imaging revealed a right lower lobe subsegmental PE (Figure 3) with associated PI and bilateral basal atelectatic changes (Figure 4). The main pulmonary artery was dilated at 3.7 cm (normal <2.9 cm). No aortic pathology was identified, and CT angiography of the abdomen and pelvis with and without contrast showed a nonaneurysmal abdominal aorta and no other intra-abdominal abnormalities. Echocardiography showed preserved ejection fraction (62.54%), right ventricular systolic pressure (RVSP) 35.6 mmHg, and no pericardial effusion. Duplex ultrasound of the bilateral lower extremities was negative for deep vein thrombosis (DVT). The patient was started on a heparin drip, transitioned to therapeutic enoxaparin, and discharged on warfarin (direct oral anticoagulants (DOACs) were not covered by insurance) with symptomatic improvement. He remained hospitalized for four days and was discharged with hematology follow-up; he was receiving warfarin for six months before it was discontinued.

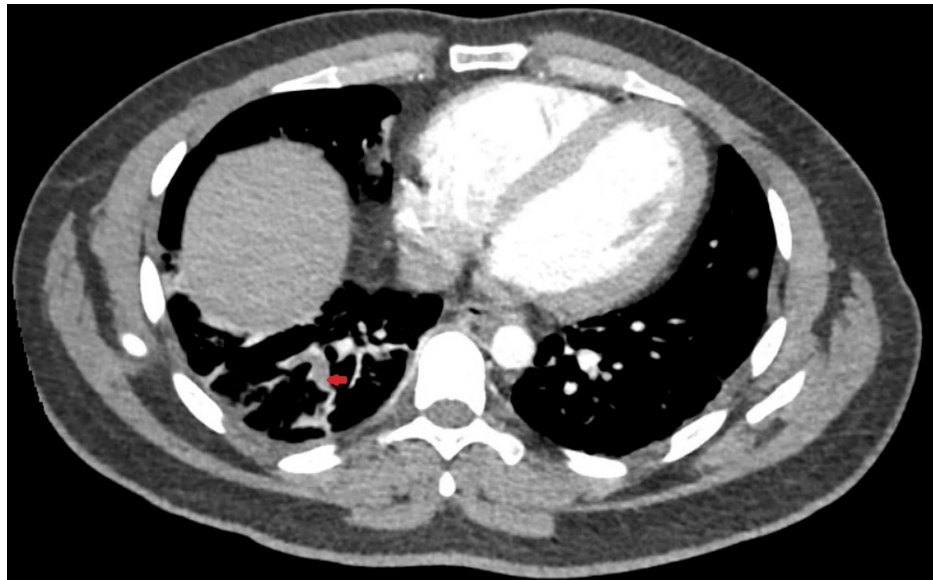


FIGURE 3: CT Angiography of the chest with contrast. Axial section. Right lower lobe subsegmental pulmonary embolus (red arrow).

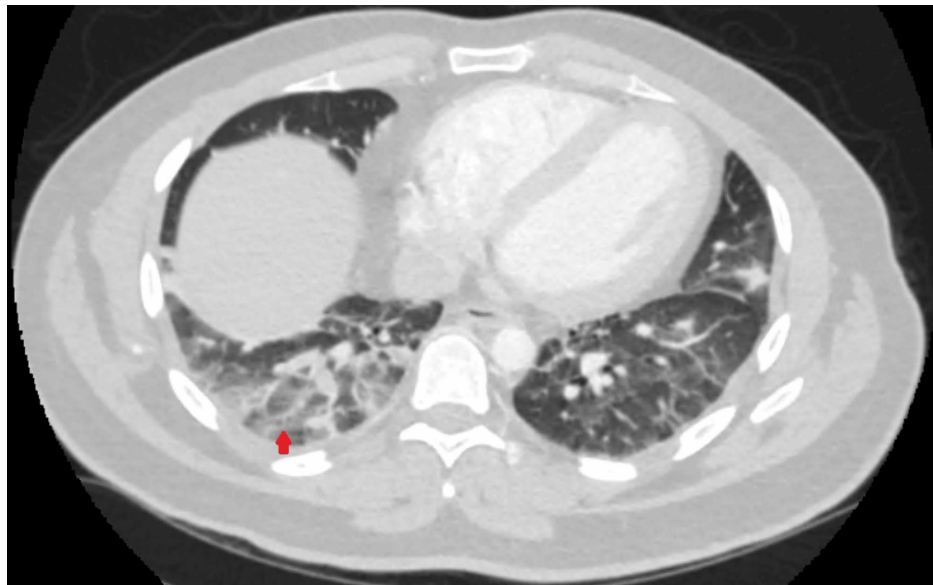


FIGURE 4: CT Angiography of the chest with contrast. Axial section. Lung window. Groundglass attenuation subtends the posterior portion of the right lower lobe consistent with pulmonary infarct (red arrow). Linear atelectatic changes are present in both lung bases.

Discussion

The incidence of diagnosed PE in the United States has increased over time, although estimates vary according to the population studied and diagnostic practices [6].

The PERC rule is intended for patients with a sufficiently low pretest probability of PE, whereas the Wells and revised Geneva scores are used to estimate pretest probability and guide subsequent diagnostic testing [4]. In this case, the prediction tools suggested a low probability of PE.

The prediction tools yielded low pretest probability estimates in this patient despite subsequently confirmed PE. The case illustrates that clinical presentations dominated by pleuritic pain syndrome may not be fully represented by the variables incorporated into these prediction rules.

PE is associated with PI in approximately 30% of cases [5]. The triad described in the literature for PI (hemoptysis, pleuritic chest pain, and pleural friction rub) [7] was only partially present in our case, manifesting solely as pleuritic pain. Young patients without cardiopulmonary comorbidities are paradoxically more vulnerable to PI, likely because of a less developed collateral circulation [5]. Thus, PI secondary to PE should be an important consideration in the differential diagnosis of pleuritic pain in young adults.

More diagnostically challenging were other atypical clinical features: acute right upper quadrant (RUQ) pain with guarding, back pain, and referred right shoulder pain. Right upper quadrant and referred shoulder pain may occur with diaphragmatic or basal pleural inflammation, potentially mediated by phrenic nerve irritation (C3-C5) overlying the right lower lobe infarction. Although anatomically plausible, these findings are underappreciated manifestations of basal pleuritis. According to the EMPEROR (Emergency Medicine Pulmonary Embolism in the Real World Registry) registry, which included data from 1,880 patients with confirmed PE, pleuritic chest pain was present in 39.4% of cases, upper abdominal pain was present in 10.7% of cases, and right shoulder pain was not reported [8].

Abdominal pain remains a misleading sign that shifts the initial differential diagnosis toward pathologies other than PE. For example, a case from the UK describes a young patient whose main clinical presentation was left-sided abdominal pain prompting surgical and urologic consults, although the Wells score indicated a moderate risk and a significantly elevated D-dimer allowed the diagnosis to be made [9]. Another case describes a patient with PE who presented with tachycardia and RUQ pain, in whom the initial diagnostic search was aimed at intra-abdominal pathologies [10]. In our case, the primary suspicion for an entity other than PE (aortic pathology) prompted further CT angiography.

Presentation with shoulder and back pain as the main complaint has been infrequently reported. In one such

case, left shoulder and back pain were the main clinical presentation, and only tachycardia and a small pleural effusion on chest X-ray led to the correct diagnosis [11].

Although PE commonly produces hypoxemia with hypocapnia or respiratory alkalosis, this patient had hypercapnia (partial pressure of carbon dioxide (PCO₂) 63.1 mmHg in the venous blood gas) and acidemia. Severe pain and pain-limited ventilation may have contributed to hypoventilation and CO₂ retention.

D-dimer testing is generally used in patients with low or intermediate pretest probability, depending on the diagnostic algorithm and clinical setting. In our case, despite the calculated low probability, PE remained part of the differential diagnosis, and the patient's development of new, acute RUQ pain prompted a broader diagnostic workup, including a D-dimer level. The result came back as borderline-elevated (239 ng/mL DDU). The relatively low D-dimer concentration may have contributed to the diagnostic uncertainty; however, the relationship between D-dimer concentration and embolus size or chronicity is not sufficiently established to attribute the finding specifically to the subsegmental or potentially subacute nature of the PE.

Conclusions

In cases where PE causes PI, the clinical presentation can be dominated by a pleuritic pain syndrome rather than the clinical signs of PE that the prediction scores are designed to detect, as is evident from our case.

Clinical prediction rules should be applied within the appropriate pretest-probability framework, and clinicians should weigh the patient's risk-factor profile, such as occupational immobilization, together with the overall symptom pattern. Evolving or atypical symptoms, as in this case, may warrant reassessment of the differential diagnosis and a lower threshold for imaging, even when calculated scores are reassuring.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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