THE PATIENT

75-year-old woman

SIGNS & SYMPTOMS

- Right-side rib pain
- Radiating shoulder pain
- History of hypertension & hypercholesterolemia

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THE CASE

A 75-year-old woman presented to the primary care clinic with right-side rib pain. The patient said the pain started 1 week earlier, after she ate fried chicken for dinner, and had since been exacerbated by rich meals, lying supine, and taking a deep inspiratory breath. She also said that prior to coming to the clinic that day, the pain had been radiating to her right shoulder.

The patient denied experiencing associated fevers, chills, shortness of breath, chest pain, nausea, vomiting, constipation, diarrhea, or changes in stool color. She had a history of hypertension, for which she was taking lisinopril 20 mg/d, and hypercholesterolemia, for which she was on simvastatin 10 mg/d. She was additionally using timolol ophthalmic solution for her glaucoma.

During the examination, the patient's vital signs were stable, with a pulse of 80 beats/min, a respiratory rate of 16 breaths/min, and an oxygen saturation of 98% on room air. The patient had no abdominal tenderness upon palpation, and the physical exam revealed no abnormalities. An in-office electrocardiogram was performed, with normal results. Additionally, a comprehensive metabolic panel, lipase test, and D-dimer test were ordered. Lab results showed an isolated elevated D-dimer of 2.66 mcg/mL (normal range, < 0.54 mcg/mL), while all other labs were normal.

THE DIAGNOSIS

Based on the lab results, a stat computed tomography pulmonary angiogram (CTPA) was ordered and showed a right segmental and subsegmental pulmonary embolism (PE; **FIGURE 1**).

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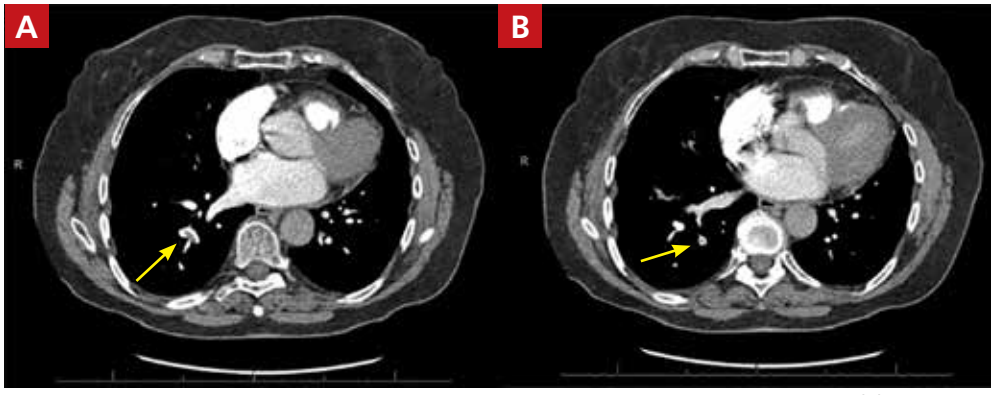
FIGURE

Imaging confirmed the diagnosis



IMAGING COURTESY OF PENN STATE MILTON S. HERSHEY MEDICAL CENTER

A computed tomography pulmonary angiogram revealed a right segmental pulmonary embolism (**A**) and subsegmental pulmonary embolism (**B**).

DISCUSSION

PE shares pathophysiologic mechanisms with deep vein thrombosis (DVT), and together these comprise venous thromboembolism (VTE). Risk factors for VTE include hypercoagulable disorders, use of estrogens, active malignancy, and immobilization.[1] Unprovoked VTE occurs in the absence of identifiable risk factors and carries a higher risk of recurrence.[2,3] While PE is classically thought to occur in the setting of a DVT, there is increasing literature describing de novo PE that can occur independent of a DVT.[4]

Common symptoms of PE include tachycardia, tachypnea, and pleuritic chest pain.[5] Abdominal pain is a rare symptom described in some case reports.[6,7] Thus, a high clinical suspicion is needed for diagnosis of PE.

The Wells criteria is an established model for risk stratifying patients presenting with possible VTE (**TABLE**).[8] For patients with low pretest probability, as in this case, a D-dimer is an effective diagnostic work-up, as a negative result will rule out PE. (If the D-dimer had been negative in this case, we would have considered other diagnoses, such as acute coronary syndrome, biliary colic, gastritis, pancreatitis, or musculoskeletal pain.) For high-risk patients, immediate anticoagulation and imaging should be performed, frequently with heparin and CTPA.[9]

Length of treatment depends on gender and etiology

The cornerstone treatment for stable patients with VTE is therapeutic anticoagulation. The new oral anticoagulants, which directly inhibit factor Xa or thrombin, have become increasingly popular for management of VTE, in part because they don't require INR testing and monitoring.[2]

The duration of anticoagulation, particularly in unprovoked PE, is debatable. As noted earlier, patients with an unprovoked PE are at higher risk of recurrence than those with a reversible cause, so the question becomes whether these patients should have indefinite anticoagulation.[2,3] Studies examining risk stratification of patients with a first, unprovoked VTE have found that men have the highest risk of recurrence, followed by women who were not taking estrogen during the index VTE, and lastly women who were taking estrogen therapy during the index VTE and subsequently discontinued it.[2,3,10]

Thus, it is reasonable to give women the option to discontinue anticoagulation in the setting of a negative D-dimer follow-up.[3] The 2016 CHEST guidelines recommend extended anticoagulation for a first-time, unprovoked VTE, but acknowledge this recommendation is strongest for men and that women with negative D-dimer assays may consider discontinuation.[10]

Our patient was directed to the emergency department for further monitoring following CT confirmation. She was discharged home after being deemed stable and prescribed apixaban 10 mg/d. A venous duplex ultrasound performed 12 days later for knee pain revealed no venous thrombosis. A CT of the abdomen performed 3 months later for other reasons revealed a normal gallbladder with no visible stones.

Apixaban was continued for 3 months

TABLE

Wells criteria for venous thromboembolism[8]

Low risk: 0-2 points; moderate risk: 2-4 points; high risk: ≥ 4 points

Parameter	Points if Yes	Points if No
Clinical signs and symptoms of DVT	3	0
PE is #1 diagnosis OR equally likely	3	0
Heart rate > 100 bpm	1.5	0
Immobilization ≥ 3 days OR surgery in the previous 4 weeks	1.5	0
Previous, objectively diagnosed PE or DVT	1.5	0
Hemoptysis	1	0
Malignancy	1	0

BPM, beats per minute; DVT, deep vein thrombosis; PE, pulmonary embolism.

and discontinued after discussion of risks and benefits of therapy cessation in the setting of a normal D-dimer and the 2016 CHEST guidelines for anticoagulation in VTE.[10]

THE TAKEAWAY

PE carries a significantly high mortality rate and can manifest with nonspecific and masquerading signs. A high index of suspicion is required to place PE on the differential diagnosis and carry out appropriate testing. Our patient presented with a history consistent with biliary colic but with pleuritic chest pain that warranted consideration of a PE. JFP

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CHALLENGES TO IMPLEMENTATION

Primary care approach may not sync with specialist practice

We see no challenges to implementation except for potential differences between primary care physicians and specialists regarding the use of antiplatelet agents in this patient population. JFP

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