

## CLINICAL VIGNETTE

# Pulmonary Embolism: An Atypical Presentation

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### **Case Report**

A 78-year-old male with hypertension, chronic kidney disease and hyperlipidemia presented to the Emergency Department for an elevated creatinine.

The patient had initially visited an outside ED for left-sided upper abdominal and flank pain. Work-up included basic labs, LFTS, lipase, troponin, BNP, chest x-ray and abdominal CT with IV contrast, which did not reveal any acute abnormality. The patient was sent home, and followed up at his primary care clinic.

At clinic the patient complained of one week of left-sided abdominal and flank pain, decreased appetite, and fatigue. He described the pain as “muscle spasms,” which worsened with deep breathing. The pain improved after taking ibuprofen and when sitting upright. He also complained of shortness of breath, but this was thought to be at baseline for him. He had a long history of dyspnea of uncertain etiology. He denied chest pain, fever, cough, hemoptysis, nausea, vomiting, diarrhea, dysuria, leg swelling, prolonged travel, or recent hospitalizations. At clinic, his basic labs were rechecked and his creatinine was noted to be elevated to 2.7 from baseline 1.9, and he was advised to return for additional evaluation.

At the Emergency Department, the patient reported that his pain started suddenly and was associated with dyspnea, nausea and diaphoresis. However, he again denied chest pain and was unclear if his shortness of breath was worse than his baseline. He complained of left upper quadrant and flank pain with deep inspiration. On physical exam, the vitals were T 97.8 F, Pulse 80, RR 20, BP 130/77 and pulse Ox 93% RA. The patient was comfortable without increased work of breathing, normal breath sounds and heart sounds. He had trace pedal edema bilaterally without unilateral swelling or pain. ECG was normal without evidence of right heart strain. Chest x-ray revealed a questionable RUL infiltrate. A d-dimer was elevated at >3000 ng/mL and he was empirically started on a heparin drip for presumed pulmonary embolism. CT of the chest without contrast was significant for an

irregular 1.5cm airspace opacity in the RUL suggestive of an infectious etiology and for a large diameter main pulmonary artery suggestive of pulmonary hypertension. V/Q scan confirmed a mismatched focal perfusion defect in the left lower lobe and lingula and at the right pulmonary apex; interpreted as high-intermediate probability for PE. Bilateral lower extremity ultrasound was remarkable for a RLE DVT and subsequent hypercoagulable workup revealed Factor V Leiden homozygosity.

### **Discussion**

Pulmonary embolism can be challenging to diagnose, and given its mortality and morbidity, the clinician should have a low threshold to consider its diagnosis in atypical presentations, particularly in the elderly<sup>1-2</sup>. This vignette illustrates a case where the diagnosis was delayed given its atypical presentation despite prior visits to two different clinicians.

The classic presentation of pulmonary embolism, abrupt onset pleuritic chest pain, hypoxia, hemoptysis and dyspnea is actually rather uncommon. Clinicians must rely on other clinical features to exclude or include it as a reasonable diagnosis. It has been reported that forty percent of patients who die suddenly from PE-diagnosed post-mortem had visited a physician for vague and nagging symptoms in the weeks prior to their death<sup>3</sup>. In the elderly who are more likely to have risk factors for thromboembolic disease, immobility and malignancy, pulmonary embolism should be considered in the differential for subtle symptoms such as weakness, malaise or dizziness. This is similar to including acute coronary syndrome or pneumonia in the differential of these same symptoms, despite lack of chest pain or fever. Medications such as beta blockers and narcotics, and cognitive decline can also contribute to the blunted responses to critical insults. In one review, the most common symptoms of pulmonary embolism in patients over 65 were dyspnea, tachypnea and tachycardia rather than chest pain<sup>1</sup>.

For this patient, the complaint of dyspnea was somewhat unclear, and he also reported malaise, anorexia and fatigue. The patient was very clear that

the pain worsened with deep breathing. The location of his pain in the left upper quadrant and flank area may have caused confusion. The first physician evaluated the patient for abdominal pathology and the second physician considered mild occult infection as responsible for the patient's overall vague symptoms. As with pneumonia presenting with upper abdominal pain, the diagnosis can often be missed. Because the pleura and diaphragm abut one another, lower chest pain and upper abdominal pain may not be easily distinguished. Likewise, back or flank pain of a pleuritic nature can also signify a pulmonary disease as the pleural cavity is not limited to the anterior chest wall.

Clinical decision tools have been created to assist in the evaluation of PE. They usually include an assessment of the clinical probability of disease, including the clinician's overall gestalt, hypercoagulability risk factors, and initial objective findings suggestive of pulmonary embolism.

Kline et al developed the Pulmonary Embolism Rule-out Criteria (PERC) rule<sup>4</sup>. It differs from other low-probability decision tools by excluding d-dimer testing when clinical suspicion is very low, as well as eight exclusion criteria. The eight exclusion criteria are: 1) age <50, 2) pulse ox > 94%, 3) HR<100, 4) no prior PE or DVT, 5) no recent surgery within 4 weeks, 6) no hemoptysis, 7) no hormone use, and 8) no unilateral swelling. The PERC rule could not be applied to this patient due to his age, 73, and his low pulse ox, 93%.

The more familiar Well's criteria for PE uses a more detailed scoring system<sup>5</sup>. The scoring criteria includes: 1) PE more likely than any other alternative: 3.0 points, 2) DVT suspected: 3.0 points, 3) HR>100: 1.5 points, 4) immobilization in 4 weeks: 1.5 points, 5) prior DVT or PE: 1.5 points, 6) hemoptysis: 1.0 points; 7) active malignancy: 1.0 points. An overall score of 0-2 is consider low probability (1.3% risk); 3-6 moderate probability (16.2% risk), and >6 is high probability (40.6% risk). For low risk patients, a negative d-dimer would effectively rule out disease; in other risk categories additional testing to diagnose PE, e.g. V/Q scan or CTA is needed. This patient's Well's score could be considered either 0 or 3; the latter, if PE was considered high in the differential, given his unexplained mild hypoxia and his pleuritic pain. If he were otherwise low-risk, a negative d-dimer would have effectively ruled out PE. His d-dimer was markedly elevated, and so additional testing would have revealed his PE. The key is to consider PE in the differential for pleuritic pain, whether it is in the

chest, back, flank or upper abdomen, and understanding the anatomical explanation.

## REFERENCES

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Submitted on November 16, 2011