A Silent PE and a Big Bleed
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Story from the Front Lines

A woman in her 60s with a history of coronary artery disease, anxiety, and non-epileptiform seizures presented to the medical intensive care unit for a bleeding rectus abdominis hematoma causing severe blood loss anemia requiring aggressive volume resuscitation and blood product transfusion. At presentation she was noted to be anti-coagulated with low molecular weight heparin as a bridge while awaiting a therapeutic INR for a pulmonary embolism (PE) that was incidentally discovered 6 weeks prior. Her hematoma was related to the injections she had been giving herself off and on over the prior 6 weeks in the context of a sub-therapeutic INR.

INR on admission was 4. Six weeks prior she had presented to the emergency department for after two syncopal episodes. She had denied chest pain, worsening shortness of breath, recent travel, or a prior history of clotting or bleeding disorder. Pertinent positives included chronic left lower extremity pain. Initial evaluation was significant for a HR 60, BP 110/80 (her baseline), oxygen saturation 94% on room air, and a Wells score of 0. Her work-up for syncope included an ECG without evidence of ischemia or RV strain, negative troponin, normal chest x-ray, and negative lower extremity ultrasound. She did have an elevated d-dimer leading to CT angiography of the chest which revealed a small, sub-segmental pulmonary embolism (PE). Her syncopal episodes were ultimately thought more likely secondary to a vasovagal response than PE though anticoagulation was started given the acute nature of the thrombus.

Teachable Moment

This case highlights important teaching points in the evaluation of syncope and the risks of finding incidental pulmonary emboli.

Syncope is an extremely common syndrome, accounting for 1-6% of all hospital admissions and 3% of emergency room visits (1). The differential diagnosis for syncope does include pulmonary embolism, yet syncope as the sole manifestation of PE alone is uncommon, typically occurring in patients with noted hemodynamic instability (2). Risk prediction models have been developed to help assess patients at risk of pulmonary embolism such as the Wells score to assist clinicians in determining pre-test probability. Ordering tests such as d-dimer or CT angiography in patient with a very low pre-test probability of venous thrombosis increases the risk of overdiagnosis and subsequent overtreatment which places patients at risk of harm.
The current standard of care is treatment of PE with anti-coagulant therapy. Concern has recently been raised that we may overdiagnose clinically insignificant PE with the increased sensitivity of CT pulmonary angiography (3). With the widespread adoption of CT angiography over traditional V/Q scanning, the incidence of PE in the US has increased by 80% from 1996-2006 (4). Over this time there has not been a decrease in mortality from PE, while the individual case fatality rate for PE has decreased (5). These observations indicate that some PEs are incidental and may not require treatment. It is hypothesized that one role of the pulmonary microvasculature is to serve as a filter keeping thrombi from entering the systemic arterial circulation and thus reducing risk of stroke.

Risks stemming from overdiagnosis of PE are not negligible. Anti-coagulation, though potentially life-saving when indicated, comes with important risks and costs to patients and our health care system. Though impossible to know with certainty that our patient was overdiagnosed, she had serious complications of anticoagulation resulting in ICU admission and transfusion of multiple blood products. It is conceivable that diagnosing PE and the anticoagulation that followed was unnecessary. Conscientious decision-making prior to ordering diagnostic studies, such as d-dimer and CT-angiography in hemodynamically stable patients with syncope is imperative to ensure high value care for our patients and avoidance of avoidable harm.

References