**CLINICAL VIGNETTE**

# Recurrent Syncope in a Post-Operative Patient

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***Case***

A 69-year-old male was admitted to the hospital from a skilled nursing facility (SNF) after an episode of syncope. He was ambulating with his physical therapist when he had sudden onset of fatigue and lightheadedness, followed by approximately 15 seconds of loss of consciousness. His physical therapist was able to catch him using his gait belt, and the patient did not sustain any head trauma. No tonic-clonic movements were noted at that time. The patient was taken to the emergency room and admitted to the hospital for monitoring and further evaluation.

This loss of consciousness was the patient's third episode in the past month. The patient's other notable medical conditions included a fall 9 months prior that was complicated by bilateral subdural hematomas and possible seizures, and renal cell carcinoma status post partial nephrectomy 8 years prior with recurrence to the pancreas requiring resection via Whipple procedure 3 months prior. His post-operative course after the Whipple procedure was complicated by an anastamotic leak requiring drainage by interventional radiology, worsening anemia requiring multiple packed red blood cell transfusions, sepsis requiring intravenous antibiotics, and two episodes of syncope, which were attributed to sepsis and anemia. Of note, he also developed urinary retention requiring temporary foley catheter placement, and his tamsulosin dose was increased to 0.4mg twice a day. His other medications were lacosamide, pancrealipase, pantoprazole, levothyroxine, acetaminophen, and budesonide/formoterol inhaler.

Upon admission to the hospital, his vital signs were notable for a temperature of 36.8 degrees Celsius, heart rate of 104 beats per minute, blood pressure of 100/75 mmHg, respiratory rate of 18, and oxygen saturation of 94% on room air. The tachycardia improved to a heart rate in the 80s without intervention. Cardiopulmonary exam was unremarkable. Abdomen was soft and a left upper quadrant surgical drain was noted to be draining small amounts of thin yellow discharge. Lower extremity edema was also noted bilaterally, which the patient reported as chronic. His basic metabolic panel was within normal limits. The complete blood count showed a stable anemia with hemoglobin of 9.3 g/dL. Initial troponin was elevated at 0.11 ng/mL and subsequently peaked at 0.14 ng/mL several hours later. Chest x-ray was unremarkable. Electrocardiogram did not show any acute changes. Orthostatic vital signs were negative. A treadmill stress echocardiogram from a year prior was normal.

By hospital day 2, the patient was able to ambulate around the unit without recurrent symptoms and no events were noted on telemetry. Given the patient's stable vital signs, low likelihood of acute cardiac ischemic event, and known previous episodes of loss on consciousness attributed to anemia and sepsis, the team considered discharging him back to SNF. However, given the unclear etiology of his troponin elevation and lack of detail regarding any valvular disease, hypertrophy or dilation of cardiac chambers on the prior stress echocardiogram, the decision was made to continue inpatient evaluation with a transthoracic echocardiogram and continued telemetry monitoring. At this time, pulmonary embolism was considered to be unlikely in light of his Wells Score of 1.

The patient's transthoracic echocardiogram revealed a dilated right ventricle, dilated right atrium, severe tricuspid regurgitation, severely elevated pulmonary artery systolic pressure of 79mmHg, and severely elevated right atrial pressure. The pulmonology team was consulted and obtained computer tomography angiogram of the chest showed "multiple occlusive and nonocclusive filling defects in the distal main pulmonary arteries extending to lobar and segmental pulmonary arteries in bilateral lungs." Bilateral lower extremity venous duplex showed multiple bilateral acute deep vein thromboses. The patient was evaluated by interventional radiology for consideration of an inferior vena cava filter to prevent further pulmonary embolisms (PEs) and for thrombolysis for existing pulmonary embolisms. However, given his clinical stability, the procedures were deemed unnecessary. He was started on a heparin drip for anticoagulation and eventually transitioned to oral anticoagulation.

***Discussion***

The initial thought process to discharge the patient back to SNF on hospital day 2 when he was medically stable demonstrated three types of common errors in medicine: anchoring; diagnostic momentum and premature closure. An anchoring heuristic is the tendency to fixate on first impressions and accept a diagnosis previously made before it has been fully verified by considering alternative diagnoses and searching for data that challenge the provisional diagnosis.[1] Diagnostic momentum is similar to anchoring.[2] Once a diagnostic label has been assigned it can sometimes be difficult to remove that label and freshly interpret the symptoms. The team anchored the current episode of syncope to the two prior presentations, which

were attributed to anemia and sepsis. The team came close to a premature closure.[3] Premature closure is a type of cognitive error in which the physician fails to consider reasonable alternatives after an initial diagnosis is made.

The team considered pulmonary embolism (PE) by calculating Wells criteria in this patient. His score on presentation was technically low at 1, with the only positive point for malignancy with treatment within the past 6 months. The team believed that his syncope was likely due to his anemia rather than a PE. The bilateral leg swelling was reported by the patient to be chronic and not acute. His tachycardia on presentation resolved on its own. His surgery was over 4 weeks ago and no other recent immobilization. The patient had no history of prior PE or deep venous thrombosis and did not have hemoptysis as a presenting symptom.

Of note, had the Pulmonary Embolism Rule out Criteria (PERC rule)[4-5] been applied to the patient after the low Wells score, this patient would have scored positive on the PERC based on his age (>50 years) and having tachycardia with a heart rate > 100 beats per minute during part of his course in the emergency room. This positive PERC would have prompted some type of pulmonary vascular imaging.

Pulmonary embolism is a common cause of hypoxia but it is clinically difficult to diagnose and often missed as it may not cause typical symptoms.[6-10] Atypical presentations of PE include back pain, acute confusion and syncope. To further complicate matters, reported rates of PE in the setting of syncope are variable. Costantino et al found that PE was identified in fewer than 1% of patients presenting to the ER with syncope and Prandoni et al found that 17.3% of patients hospitalized for a first episode of syncope had a PE.[11-12]

In summary, it is important not to let cognitive biases like anchoring and diagnostic momentum lead to premature closure as some disease states present atypically.

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