

> THE PATIENT

58-year-old woman

> SIGNS & SYMPTOMS

- Cluster of syncopal and near-syncopal events in a patient with a history of occasional vasovagal events
- Recent surgical repair of a left hip fracture

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> THE CASE

A 58-year-old woman sought care at our clinic for recurrent syncopal and near-syncopal events following surgical repair of a left hip fracture. The first syncopal event occurred one day post-surgery shortly after standing and was attributed to orthostatic hypotension. Subsequently, the patient experienced 2 events during her hospital stay. Both events occurred in the upright position and were preceded by lightheadedness, warmth, and diaphoresis. They were short in duration (<30 seconds) with spontaneous and complete recovery. The patient had no associated chest pain or palpitations.

The patient's past medical history included osteopenia, dyslipidemia, and vasovagal syncope, averaging one to 2 events per year. Given her past history, the physicians caring for her assumed that she was having recurrences of her vasovagal syncope. She was discharged home on fludrocortisone 0.1 mg/d, sodium chloride 1 g tid, enoxaparin 40 mg/d, and acetaminophen and oxycodone as needed for pain.

One week later, the patient experienced another syncopal event at home, prompting her to visit our clinic for further evaluation. On arrival, her vital signs were stable. Her oxygen saturation level was 98%, she was not orthostatic, and her physical exam and blood studies were unremarkable. An echocardiogram showed preserved left ventricular function with no evidence of right ventricular dilatation or strain.

THE DIAGNOSIS

The patient's revised Geneva Score for pulmonary embolism (PE) was 2 to 5 depending on the heart rate used (66-80 beats per minute), putting her in a low-to-intermediate risk group with an estimated PE prevalence between 8% and 28%.¹ Given her recent surgery and the increase in the frequency of her vasovagal events, a computed tomography pulmonary angiogram (CT-PA) was performed. The CT-PA showed a PE in the lateral and posterior basal subsegmental branches of the right lower lobe. Doppler ultrasound revealed no evidence of acute deep vein thrombosis.

DISCUSSION

Syncope may develop in 9% to 19% of patients with PE.²⁻⁶ While syncope in patients with PE is often attributed to reduced cardiac filling secondary to massive emboli, it is important to recognize that patients can also present with vasovagal syncope in the absence of massive emboli.

One mechanism for the development of syncope is right ventricular failure with subsequent impairment of left ventricular filling, leading to arterial hypotension. Indeed, the majority of patients with PE and syncope have a massive embolism defined as greater than

a 50% reduction in the pulmonary circulation.⁷ In one study, 60% of patients with PE who presented with syncope had a massive PE compared to 39% of patients presenting without syncope ($P=.036$).⁸

Another reported mechanism for syncope in a patient with PE is transient high-degree atrioventricular (AV) block.⁹ Sudden increases in right-sided pressure can lead to transient right bundle branch block, which may result in complete heart block in the setting of baseline left bundle branch block.

Lastly, patients with PE may develop a vasovagal-like reaction, such as the Bezold-Jarisch reflex, which results in transient arterial hypotension and cerebral hypoperfusion.¹⁰ In such instances, the postulated mechanism is activation of cardiac vagal afferents, which results in an increase in vagal tone and peripheral sympathetic withdrawal leading to hypotension and syncope. It is important to note that this mechanism can occur in the absence of massive PE. In one study, up to 40% of patients with PE and syncope did not have a massive PE, and almost 6% had thrombi only in small branches of the pulmonary artery.⁸

■ **This patient had isolated subsegmental defects**, identified on the CT-PA. The sensitivity of CT-PA to detect subsegmental PE ranges from 53% to 100%.¹¹ While this test has its limitations, the introduction of the multi-detector CT technique has significantly increased the rate of detection with a specificity of 96%.^{12,13}

Was PE the cause of the syncope, or just an incidental finding?

In this case, we believe the CT-PA findings were diagnostic for PE. What is less clear is whether the PE was the cause of the syncope.

Asymptomatic post-operative PE with isolated subsegmental defects has been reported.¹⁴⁻¹⁶ When compared to patients with a defect at a segmental or more proximal level, these patients often have less dyspnea, are less likely to be classified as having a high clinical probability of PE, and have a lower prevalence of proximal deep vein thrombosis (3.3% vs 43.8%; $P<.0001$).¹⁷ Therefore, one could argue that the PE finding in our case

was incidental. While this is a possibility, we believe the patient's syncope was due to PE for the following reasons.

First, several investigators have reported transient increases in vagal tone and syncope following PE consistent with a vasovagal-like response.^{7,18} Therefore, it is possible that the reduction in preload associated with PE triggered a Bezold-Jarisch-like reflex leading to syncope. The patient's history of vasovagal syncope was certainly indicative of increased susceptibility to reflex-mediated events, thus supporting our hypothesis.

Second, our patient had a cluster of events following surgery compared to the one to 2 events she experienced per year prior to surgery. The increased incidence of events would be an unusual progression of her syncope in the absence of clear triggers, again rendering our hypothesis more plausible.

■ **The patient was admitted to our hospital** and started on a higher dose of enoxaparin (60 mg twice daily). She was subsequently discharged home on rivaroxaban 15 mg twice daily and midodrine 2.5 mg twice daily in addition to the medications she was already taking. At her 6-week follow-up visit, she reported no recurrences.

THE TAKEAWAY

This case demonstrates that non-massive PE can present as vasovagal syncope. Recognizing that PE could lead to reflex-mediated syncope in the absence of massive emboli, it is important to rule it out in the evaluation of patients with vasovagal syncope when risk factors for PE are present. **JFP**

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➤ One could argue that the PE finding in our case was incidental, but we had several reasons for believing it was the cause of our patient's syncope.

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