

Assembling the Puzzle: Taking into Account Clinical Presentation and Predictive Scores

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Syncope is a common cause of emergency department visits. Physicians must scrutinize for life-threatening causes to avoid patient morbidity and mortality. Clinical decision rules are used to stratify risks and guide the course of action, including the need for further testing.³ This is the case of a 83-year-old man was brought to the emergency department after a 5-minute episode of sudden loss of consciousness. Vital signs showed hypotension and physical examination was unremarkable. Despite Wells score of 0, clinical suspicion for pulmonary embolism persisted, for which further testing was pursued. D-dimer was elevated at 13.77 mcg/mL and a chest computed tomography with angiography showed an extensive bilateral pulmonary embolism involving the distal right and left main pulmonary arteries. He was started on full-dose anticoagulation. This case exemplifies the need of high clinical suspicion along with the importance of applying predictive scores for diagnosing unusual causes of syncope. [P R Health Sci J 2022;41(2):104-106]

Key words: Pulmonary Embolism, Syncope, Well's Score, Predictive Score, Pulmonary embolism rule-out criteria

Syncope is a symptom of cerebral hypoperfusion provoking a transient loss of consciousness with an inability to sustain muscle tone. The prevalence in adults over the age of 45 is approximately 19% (1); reflex syncope accounts for up to two-thirds of cases. Cardiopulmonary etiology has a lower prevalence of 10-20% (2), yet remains one of the most feared sources given its lethality (3). An infrequent etiology of syncope is pulmonary embolism which is associated with an increased 30-day all-cause mortality risk. It is of the highest importance to ascertain a definitive source as it has a direct impact on the patient's prognosis (4). A thorough evaluation should be performed to reach the correct diagnosis.

Case Report

An 83-year-old man with a medical history of advanced Alzheimer's dementia, dysphagia, and hypothyroidism presented to the emergency department after a family witnessed five-minute episode of sudden loss of consciousness and muscle tone. He had no signs of focal neurological deficit, involuntary movements, or loss of sphincter control. Immediately after regaining consciousness, he developed acute chest pain lasting fifteen minutes but was unable to describe the nature of pain. On arrival, he was hypotensive with 87/58 mmHg, heart rate of 67 beats/minute, temperature of 97.4°F, and respiratory rate of 19 breaths/minute with a peripheral oxygen saturation of 98%. The physical exam was remarkable for a normal cardiopulmonary exam and no carotid bruit, focal neurologic deficits, or lower extremity swelling.

Laboratory tests revealed elevated high-sensitive Troponin T (280 ng/L, 185 ng/L), otherwise unremarkable. The ECG (Figure 1) on arrival showed normal sinus rhythm without arrhythmia, QT prolongation, or acute ST-segment changes. An emergent head CT scan was negative for any acute intracranial pathology. The Well's score was 0 consistent with a <1.3% risk for PE. Nevertheless, he failed to meet all PERC rule out criteria and clinical judgement at this time could not rule out a pulmonary embolism. Further testing showed an elevated D-dimer of 13.77 ug/mL (expected of 0.083 mcg/mL). Given his clinical presentation, abnormal cardiac enzymes, and elevated D-dimer, a chest CTA was performed, revealing an extensive bilateral pulmonary embolism (PE) involving the distal right and left main pulmonary arteries (Figure 2). The patient was initiated on low-molecular weight heparin and later discharged following transition to a direct oral anticoagulant. Patient subsequently followed in primary care clinics with adequate tolerance of anticoagulation and no recurrence.

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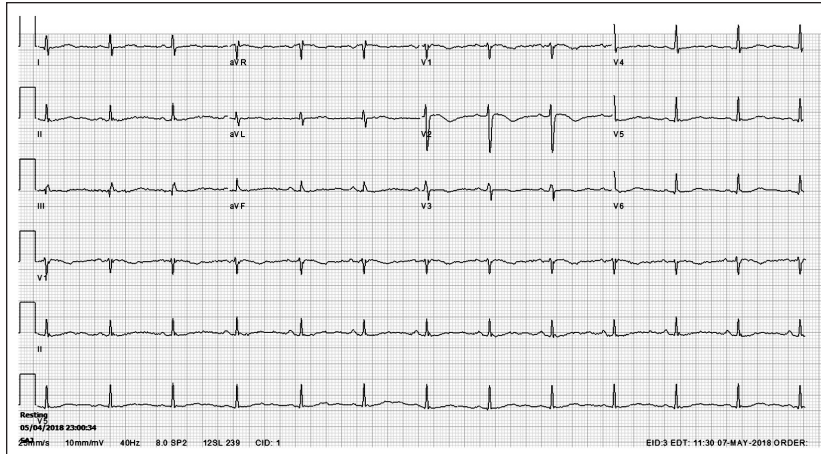


Figure 1. Electrocardiogram (ECG) performed at arrival to the emergency room. Here we see a normal sinus rhythm ECG with no arrhythmia, QT-prolongation or acute ST-segment changes.

Discussion

With this case, we highlight the importance of combining a thorough history and physical exam to attain a correct diagnosis. As previously stated, syncopal episodes have a wide variety of etiologies, and a correct diagnosis is critical as certain etiologies lead to increased mortality (3, 5). There has been a recent development of predictive scores designed to evaluate syncope, but they have not been found to be superior to clinical judgement (6,7). Clinical decision rules are currently used to risk-stratify patients, taking into consideration the probability of an adverse outcome to guide decision making (3).

Numerous studies have assessed both the incidence and prevalence of PE manifesting as a syncopal episode, with varied results. In 2016, Paolo et al. described a prevalence of PE in hospitalized patients with syncope of 17.3% (8). However, other studies have not shown such a high prevalence. A recent meta-analysis published by Oqab et al. in 2017 suggested a lower presence of syncope attributed to PE with a prevalence of PE 0.9% and 1.0% in all syncopal patients and hospitalized patients, respectively (9).

Pulmonary embolism as a manifestation of syncope can be explained by three plausible pathophysiologic mechanisms. First, a large embolus can cause right ventricular overload provoking arrhythmia, resulting in insufficient tissue perfusion and subsequent syncope. Second, which accounts for two thirds of cases, the embolus can trigger a vasovagal reflex provoking a neurocardiogenic syncope. Finally, the extensive occlusion of the pulmonary vasculature can precipitate right ventricular failure, which decreases left ventricular filling reducing cardiac output resulting in

arterial hypotension and the eventual loss of consciousness (10,11). The last mechanism is the likely cause of our patient's syncope, as he presented with hypotension, elevated troponins, and CTA disclosed an extensive bilateral pulmonary embolism.

Despite chest pain, shortness of breath, and elevated troponins, there was a low suspicion for acute coronary syndrome or arrhythmia given normal ECG. As recommended in the 2017 American College of Cardiology Guideline for Evaluation and Management of Patients with Syncope and the 2018 European Society of Cardiology Diagnosis and Management of Syncope, the initial evaluation is centered on a detailed history

and physical examination as well as an ECG. However, when the diagnosis is uncertain, further stratification should be done based on symptoms (12, 13). The Well's Score is an established noninvasive validated tool for assessment of risk for PE (14, 15, 16). To reduce unnecessary testing in low probability patients, Kline et al. proposed eight discerning criteria known as the PERC rule (17). Meeting one of these conditions, warrants evaluation with D-dimer, and if elevated diagnostic testing is recommended (18). Although PE may present with syncope, the prevalence is very low (10). In the pursuit of etiologies provoking cardiac strain and syncope, pulmonary embolism was suspected. Above all, clinical suspicion at the initial presentation encouraged additional evaluation with D-dimer followed by chest CTA despite Well's score of 0 attaining a diagnosis in our patient.

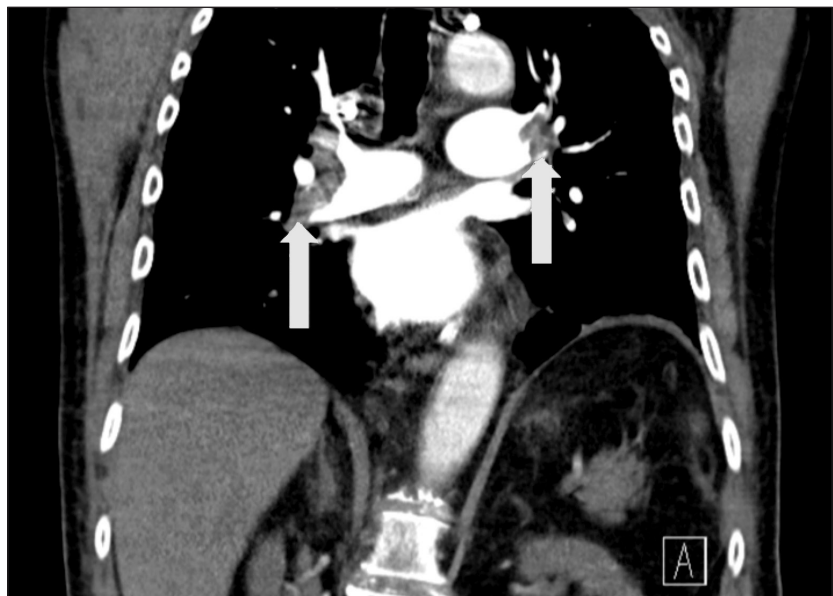


Figure 2. Chest computer tomography angiography (CTA) performed due to suspected pulmonary embolism. The CTA reveals bilateral extensive pulmonary emboli along the distal right and left main pulmonary arteries (red arrows).

Conclusion

We report an atypical presentation of syncope secondary to a pulmonary embolism. A diagnosis of PE was made even though there was a low pre-test probability consistent with a prevalence of <1.3%. Multiple risk scores have been developed in the emergency department for assessing the risk for patients presenting with syncope, but none have been found to be superior to clinical judgement. This case highlights the importance of pursuing life-threatening etiologies of syncope as misdiagnosis may lead to increased morbidity and mortality.

Resumen

Síncope es una causa muy común para visitar a la sala de emergencia. Los médicos deben ser capaces de poder detectar causas letales para evadir morbilidad y mortalidad. Guías y reglas se han creado para ayudar a estratificar los riesgos de cada paciente, guiar el manejo y determinar si amerita más exámenes. Presentamos un caso de un paciente de 83 años de edad que llegó a la sala de emergencia después de un episodio de pérdida de conciencia que duró cinco minutos. Signos vitales enseñaron hipotensión y la examinación física estaba normal. A pesar de tener una puntuación de Wells 0, la sospecha por una embolia pulmonar persistía, por lo cual se le ordenó un “D-dímer” que salió elevado en 13.77 mcg/mL. Se le ordenó una angiografía por CT que enseñó embolias pulmonares extensas que envolvían las arterias pulmonares derecha e izquierda. Debido a estos hallazgos, se decidió empezar el paciente en anticoagulación. Este caso enseña la necesidad de tener una sospecha alta en combinación de escalas de puntuación ya establecidas para poder determinar causas no usuales de síncope.

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