



CASE REPORT

Cough-Induced Syncope as an Unusual Manifestation of Pericardial Effusion

Hazem El-Osta, M.D.¹

Salman Ashfaq, M.D.^{1,2}

¹University of Kansas

School of Medicine–Wichita

Department of Internal Medicine

²Heartland Cardiology, Wichita, KS

Introduction

Cough-induced syncope belongs to the heterogeneous group of situational syncopes. It consists of the loss of consciousness associated with a paroxysm of cough. It was first described by Charcot in 1876,¹ but the underlying mechanism remained a mystery until recently. While many clinical cases of cough syncope have been described in the literature, this report describes a unique case of cough-induced syncope in a patient with a newly-discovered pericardial effusion with near tamponade.

Case Report

A 70-year-old male presented with a five-day history of multiple episodes where he lost consciousness during or after cough. The syncopal events lasted for durations ranging from 30 seconds to about one minute. The patient also reported a two-week history of pleuritic chest pain, shortness of breath upon exercise, and cough. He initially presented with these symptoms to another facility where a computed tomography of the chest revealed a moderate to large pericardial effusion. He was transferred to our facility for further evaluation.

Upon admission, the patient was stable. His labs were unremarkable. A trans-thoracic echocardiogram showed a large pericardial effusion measuring 2.5 to 3.2 cm in maximal dimension (see Figure 1).

He manifested early right atrial collapse, with no clear evidence of right ventricular diastolic collapse. The patient did not meet criteria for pericardial tamponade. He simply had a large pericardial effusion. A sizeable amount of fibrinous material was noted within the pericardial space adjacent to the visceral pericardium.

During his hospital stay, many witnessed episodes of cough-induced syncope occurred. None were associated with bradycardia, arrhythmia, or block on telemetry. During one episode, his systolic blood pressure showed a reduction from 110 mmHg to 80 mmHg. The blood pressure returned to baseline as soon as the symptoms resolved.

A biopsy of the fibrinous material was completed, without any complications, by performing a pericardial window. Approximately one liter of blood-tinged pericardial fluid was drained. Cytology of the fluid and biopsy of the fibrinous material were negative for malignancy. Cultures performed on the fluid also were negative for infection. No other systemic inflammatory process was diagnosed, although no diagnostic viral test was obtained. A viral pericarditis complicated by the development of a large pericardial effusion was thought to be the most likely diagnosis. Interestingly, after drainage of the fluid, the patient experienced complete relief from the cough-syncope symptoms.

He was discharged a few days later for outpatient follow-up. He has had no more syncopal episodes since his pericardial effusion was drained.

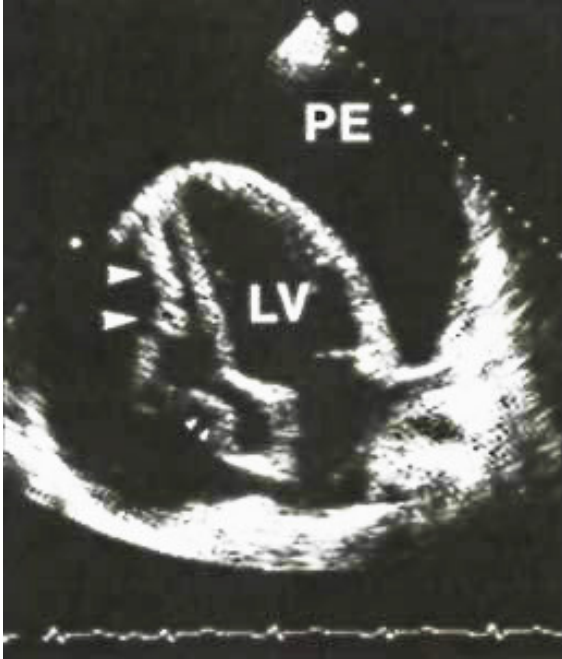


Figure 1. Pericardial effusion is evident on the two-dimension echocardiogram.

Discussion

Cough syncope is a well-recognized but uncommon phenomenon. The exact mechanism by which it occurs remains controversial and multiple theories have been put forward.¹ The most widely accepted theory is that the syncope occurs as a result of hemodynamic changes induced by the increase in the intrathoracic pressure associated with cough which can in turn limit venous return to the heart. Lowered cardiac output and blood pressure result in cerebral hypoperfusion. In some cases, impairment of the cerebral venous return precipitates cerebral hypoperfusion without necessarily a decrease in blood pressure.²⁻⁴

Arrhythmias also can cause cough-induced syncope. For example, atrioventricular conduction block due to

hypersensitive broncho-pulmonary reflex, a neurally-mediated reflex contributing to an inappropriate chronotropic response or a premature ventricular complex, may cause cough-induced syncope.^{1,5-8}

Cough-induced syncope in the patient described above was associated with hypotension. Bradycardia or arrhythmias were not documented during the episodes. Impediment of cardiac filling may have resulted from the superimposed cough causing an increase in the intrathoracic pressure. The increased intrathoracic pressure may have caused a rise in intra-pericardial pressure which likely changed the RV filling from a pre-tamponade state to a true tamponade physiology for a few seconds. The tamponade would cause a lowering in systemic blood pressure, which likely reduced cerebral perfusion causing the pre-syncope and syncope symptoms. After the coughing, the intra-pericardial pressure, the systemic blood pressure, and the cerebral perfusion return to the baseline levels and the symptoms resolve. In this patient, the syncopal episodes subsided completely after drainage of the pericardial fluid, thus supporting the correlation between the pericardial effusion and the syncopal episodes.

This case is the only known example of cough-induced syncope due to a pericardial effusion in the literature. Cough-induced syncope was described in a subject with constrictive pericarditis which has some common basic pathophysiology with pericardial effusion.⁸

Conclusion

This case presented a novel cause of cough-induced syncope. It is important to consider pericardial effusion in any case of new-onset cough-induced syncope, which consequently can provide earlier diagnosis and treatment.

References

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