

Case Report

Pericardial Effusion presenting as Cough Syncope

J Evans & S Ullah

Abstract

Syncope is a common clinical presentation and accounts for 1-3% of Emergency Department (ED) visits.¹ Cough syncope is a rare type of situational syncope, often caused by respiratory conditions like bronchial asthma and chronic obstructive pulmonary disease. Cough syncope due to pericardial effusion is a rare but treatable condition. Delay in diagnosis can lead to fatal complications due to cardiac tamponade. We present a case of recurrent cough syncope caused by large pericardial effusion.

Keywords

Syncope, Cough syncope, Pericardial effusion.

Teaching point

- Pericardial effusion is a serious but reversible cause of cough syncope

Case history

A 58 year old male was admitted to the Emergency Department (ED) with a syncopal episode. The syncope was triggered by a bout of coughing whilst walking in his garden. He recovered quickly over a few minutes. There were no features of seizure like activity. The event was preceded by two days history of dry cough associated with mild shortness of breath but no chest pain. At this point he was taking antibiotics prescribed by the general practitioner (GP) for a lower respiratory tract infection.

His past history included type 2 diabetes mellitus, obesity, hypertension, osteoporosis and cervical spine fixation surgery. His medications at the time of presentation included rosuvastatin, gabapentin, omeprazole, gemfibrozil, imipramine, tramadol, timolol eye drops and metformin. Two weeks previously, he had been an inpatient with acute cholecystitis, managed conservatively with antibiotics and analgesia.

When he was assessed in the ED, he looked well and his observations were stable. On examination, he was morbidly obese, alert and oriented. There was mild inspiratory and expiratory wheeze bilaterally on chest auscultation. He had unremarkable neurological and cardiovascular examination findings. His electrocardiogram (ECG) showed sinus rhythm with a rate of 65 beats per minute and a blood pressure (BP) of 129/79. His blood tests showed normocytic normochromic anemia and mildly raised C-reactive protein. All other biochemical investigations including blood sugar were within normal range (Table 1). He was discharged from the ED with diagnosis of cough syncope associated with a chest infection. He was advised to complete the course of antibiotics he was already taking.

He had a second syncopal episode at home on the following day, again precipitated by a coughing fit. He was referred to the acute medical unit for further investigation by his General Practitioner. On admission, he had a regular pulse rate of 86/minute and pulse oximetry reading of 94% on air. His lying BP was 125/75 and standing BP 120/90. His jugular venous pressure was not visible because of obesity and spinal fixation. His heart sounds were normal. On auscultation, his chest was clear with good air entry bilaterally and no added sounds. There were no signs of deep vein thrombosis. Neurological examination was unremarkable.

Arterial blood gas analysis on air showed pH 7.450, pCO₂ 4.89 kPa, pO₂ 9.67 kPa, Standard Bicarbonate 25.7 mmol/L, Base Excess 1.4 mmol/L, Lactate 1.3 mmol/L.

His electrocardiogram showed sinus rhythm with no evidence of acute ischemia or right heart strain. The QRS complexes were small (Figure 1). A chest radiograph (CXR) showed a large cardiac silhouette and small right sided pleural effusion (Figure 2).

Blood test	Admission 1 (ED)	Admission 2 (Acute medical unit)
Haemoglobin (g/L)	98	100
Mean Corpuscular Volume (fl)	83	83
White blood cells (x 10 ⁹ cells per liter)	9.4	10
Platelets (x 10 ⁹ per liter)	373	427
C-reactive protein (mg/L)	19.4	24
Sodium (mmol/L)	136	137
Potassium (mmol/L)	4.4	4.5
Urea (mmol/L)	4.6	4.6
Creatinine (μmol/L)	96	97
Glucose (mmol/L)	6.5	7.3
Albumin (g/L)	38	35
Alkaline phosphatase (U/L)	79	79
Aspartate aminotransferase (U/L)	15	12
Bilirubin (μmol/L)	12	18
Corrected calcium (mmol/L)	2.30	2.31
D-dimer ng/ml	Not performed	1120

Table 1. Full blood test results from both admissions

Jack Evans

MBBCH. PgDip Med Ed.
ACCS CT3. Great Western
Hospitals NHS Foundation
Trust

Sharif Ullah

FRCP.
Consultant Physician. Acute
medical unit. Great Western
Hospitals NHS Foundation
Trust

Correspondence

Jack Evans
MBBCH. PgDip Med Ed.
ACCS CT3. Great Western
Hospitals NHS Foundation
Trust
Email: jackevans28@doctors.
org.uk

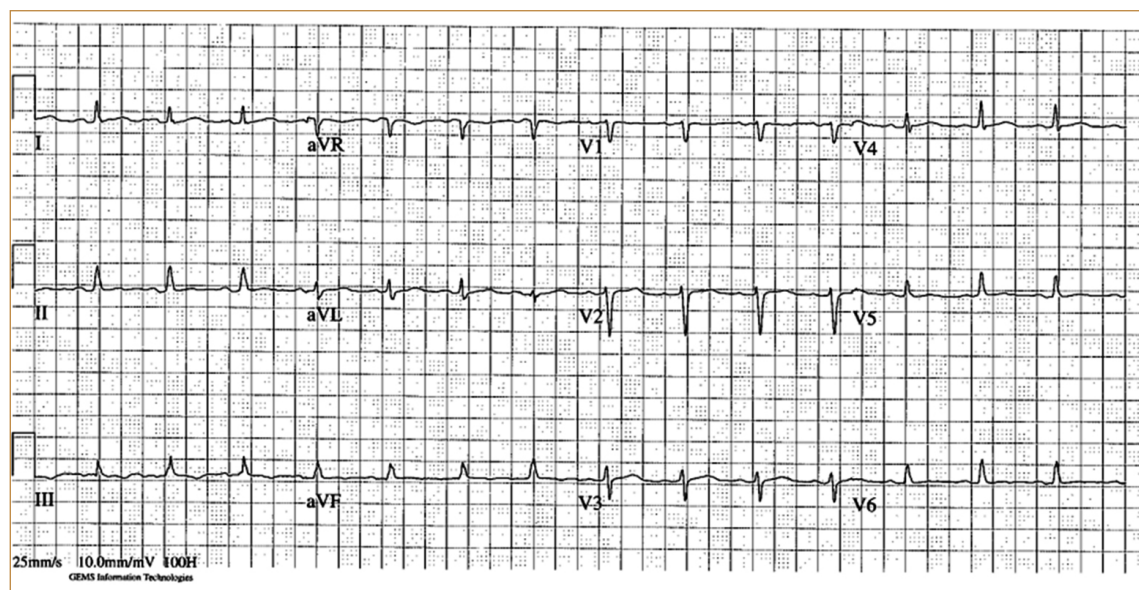
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Figure 1. Patients admission 12 lead ECG demonstrating low voltage QRS complexes.

There was a clinical concern for pulmonary embolism given recent immobility, syncopal episodes, hypoxia and raised D-Dimer. Therefore a computed tomography pulmonary angiogram (CTPA) was done. This scan showed a large pericardial effusion, small bilateral pleural effusion and consolidation (Figure 3 and 4). There were no pulmonary emboli. An urgent echocardiogram was performed, demonstrating a large pericardial effusion, measuring more than 4cm on the lateral wall and 3.9cm at the posterior wall. There was right ventricular (RV) collapse in systole (Figure 5) and significant respiratory variation seen in transmitral inflow (Figure 6). These findings suggested a haemodynamically significant pericardial effusion. The patient required emergency pericardiocentesis,

with 700mls of serosanguineous pericardial fluid drained initially, resulting in a rapid improvement of symptoms. The drain was removed after 24 hours. The cough syncope did not reoccur as an inpatient. Serial echocardiography showed no recurrence of pericardial effusion and good biventricular systolic function. Figure 7 shows a repeat CXR post pericardiocentesis.

Blood cultures and pericardial fluid were sterile on culture, and acid fast bacilli were not grown. Pericardial fluid cytology was consistent with a reactive effusion. Atypical myopericarditis serology was normal. A respiratory tract infection with an unconfirmed organism was thought to have caused the reactive pericardial effusion. Subsequent outpatient echocardiography demonstrated full

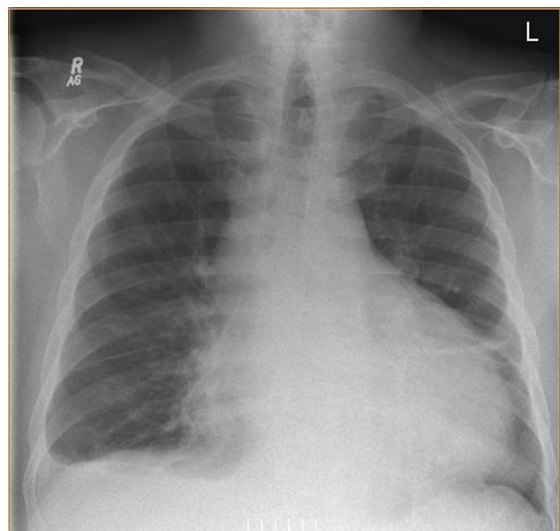


Figure 2. Chest radiograph on admission – enlarged cardiac silhouette and small right pleural effusion

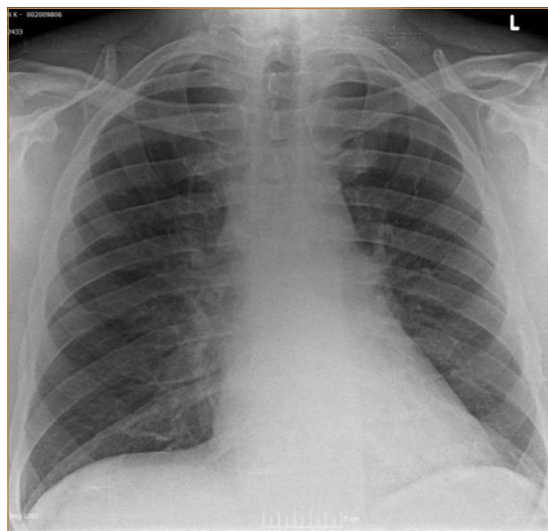


Figure 7. Chest radiograph - 1 month post pericardiocentesis.

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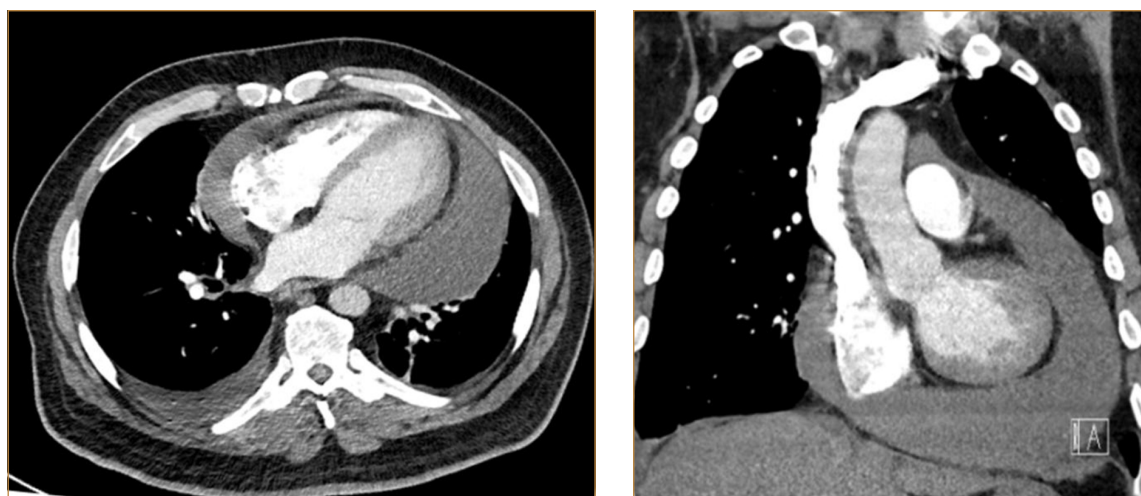


Figure 3 & 4. CTPA demonstrating a large pericardial effusion and small right pleural effusion

resolution of the effusion and the patient remained asymptomatic with no further cough or syncopal episodes.

Discussion

Cough syncope (CS) is a rare medical presentation, usually associated with chronic obstructive pulmonary disease.² The classic case being in an overweight middle aged male² such as in this case. There have been associations with central nervous system disorders as well as cardiovascular pathologies, namely constrictive pericarditis³ and atrioventricular block.^{4,5} Cough Syncope induced by pericardial effusion is very rare, with only three published cases in the literature.^{6,7,8} There was resolution of symptoms in all known cases after pericardiocentesis. This supports the correlation between the pericardial effusion and cough induced syncope episodes. There is some suggestion that even small pericardial effusions can produce this phenomenon.⁹

The pathophysiological basis for cough syncope is likely to be due to a large inspiration causing reduced cardiac filling. Some authors present it

as multifactorial. One hypothesis considered, is transmission of increased intrathoracic pressure to the cerebrospinal fluid. The theory being the acute increase in intracranial pressure compromises cerebral perfusion.¹⁰ Mattle *et al.*¹¹ demonstrated a critical reduction in middle cerebral artery flow velocities during the coughing, resulting in transient cerebral circulatory arrest.

Another hypothesis is that high intra thoracic pressure generated by the cough causes syncope through diminished venous return to the heart.¹⁰ Dessier *et al.*¹² demonstrated a significant decline in peak carotid blood velocity within one second of a cough, using Doppler ultrasonic flowmetry in healthy subjects. They found a low degree of correlation between the recorded mean right atrial pressure and peak carotid blood velocity. This suggests impaired venous return was not the only factor responsible for reduced carotid artery blood velocity.¹²

The baroreflex in humans is incompletely understood but thought to play a role in cough syncope.¹⁰ Coughing generates transient hypertensive arterial states, triggering a compensatory baroreflex

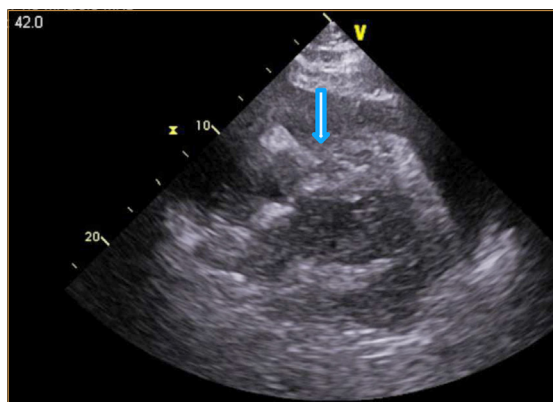


Figure 5. Echocardiogram sub-costal view demonstrating pericardial effusion with right ventricular collapse in systole (see blue arrow).

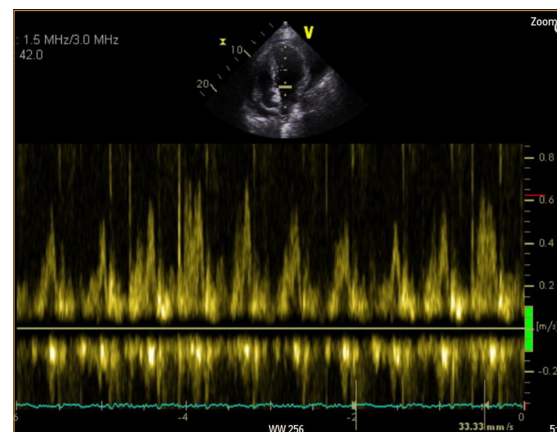


Figure 6. Pulsed Doppler of trans-mitral inflow showing significant respiratory variation.

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to buffer the pressure fluctuation.¹⁰ In CS patients, the magnitude of the reflex response is thought to be greater.¹⁰ Additionally, Benditt *et al.*¹³ and Dickenson *et al.*¹⁰ suggest a blunted chronotropic response in patients with cough syncope. This neural reflex in response to cough may explain the association between CS and atrioventricular node blocks.^{4,5}

Pericardial effusion with cardiac tamponade is the probable cause of cough syncope in our case. The resolution of CS after pericardiocentesis supports this. Saseedharan *et al.*⁸ describe a similar case where during a witnessed coughing episode the patient went into cardiac tamponade. After recovery from the CS episode they found the echocardiographic findings of tamponade disappeared. They concluded that cardiac output may drop significantly during coughing fits in patients with large pericardial effusions.⁸ They describe a ‘pre tamponade’ state, where the amount of pericardial fluid is just below the amount that would compromise cardiac output.⁸

The pericardium is not compliant so an increased volume of pericardial fluid will reduce filling.¹⁴ Our patient had pericardial effusion which exerted pressure on the cardiac chambers. This reduces

diastolic compliance.¹⁴ The external pericardial pressure and intrathoracic pressure contributes to transmural pressure on the heart.⁸

We favour the theory that an acute increase in intrathoracic pressure during the cough causes an increased transmural pressure on the heart. This increase in transmural pressure causes a compressional effect consistent with the pathophysiology of cardiac tamponade. This transient hemodynamic change clinically only occurs during the coughing episode. When coughing stops, cardiac tamponade ceases resulting in recovery from the syncopal episode.

Cough syncope is a type of situational syncope. In benign cases, it has implications of driving restrictions. Group 1 drivers must not drive for 6 months following a single episode.¹⁵ Group 2 drivers must not drive for 12 months.¹⁵ Pericardial effusion is a serious but reversible cause of cough syncope and should be considered in the differential diagnosis. Urgent echocardiography will help facilitate early diagnosis and treatment.

Competing interest

Nothing to declare.

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