CASE REPORT

Cough Syncope: Constrictive Pericarditis

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Abstract

An 87-year-old man with a diagnosis of constrictive pericarditis suffered from cough syncope up to 10 times per day on most days during his three-day stay at our hospital. After undergoing a series of treatments (diuretics, codeine and intravenous ceftizoxime), the patient still had a mild cough, although he did not experience any further syncopal episodes associated with coughing. Two months later, the syncopal episodes associated with coughing returned, but at a lower rate.

Key words: constrictive pericarditis, cough syncope

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Introduction

Tussive syncope, the transient loss of consciousness and postural tone associated with paroxysms of coughing, was first described by Charcot in 1876 using the colorful descriptor ‘laryngeal vertigo’ (1). Cough syncope classically occurs in patients with obstructive pulmonary disorders or right ventricular myocardial infarction and sometimes in cases of constrictive pericarditis. To the best of our knowledge, only two cases of cough syncope induced by constrictive pericarditis have previously been described in the literature (2, 3). In typical constrictive pericarditis, diastolic filling of the heart is inhibited due to chronic fibrous thickening of the wall of the pericardial sac, which may in turn cause a series of physical problems such as an increase in jugular venous pressure, ascites, hepatosplenomegaly or edema, with the former especially occurring in the form of Kussmaul’s sign (4).

Case Report

An 87-year-old man presented with a persistent cough lasting for three days. He reported having experienced shortness of breath during less-than-ordinary activity. One day later, he began to experience paroxysms of fainting that were always associated with coughing spells. During his three-day stay at our hospital, the patient experienced paroxysms up to 10 times a day. At onset, he experienced facial cyanosis, staring, loss of consciousness for 10-20 seconds and sinus bradycardia with a heart rate of 40-50 beats/min (based on electrocardiography). However, immediately after each episode, all of the symptoms subsided. Physical examinations conducted during the episodes of syncope from coughing showed an elevated jugular venous pressure and reduced heart sounds, although no other neurological symptoms were observed.

The patient had moderate hypertension with a peak blood pressure of 160/100 mmHg. Nifedipine (controlled-release tablets) was administered for blood pressure control, and the patient’s blood pressure was reduced to between 150/92 and 140/80 mmHg. The patient also had Parkinson’s disease, for which he was taking levodopa. He denied having any history of diabetes mellitus or epilepsy. He had a history of smoking for 10 years earlier in life, but had not smoked for the past 30 years. He did not have a history of drinking.

Upon physical examination, the patient was found to be well-proportioned in appearance. His heart rate was 94 beats/min with a blood pressure of 163/94 mmHg. He did not have any carotid bruits, pericardial friction rub, wet rales or wheezes; however, he exhibited jugular vein distention. Chest X-ray revealed an enlarged heart shadow. Computerized tomography (CT) of the thorax (Figs. 1, 2) revealed a moderate pleural effusion with a thickened pericardium in which the maximum thickness was up to 8 mm and the CT values were 31-42 Hounsfield units (HU). Bedside echocardiography showed a moderate pericardial effusion with left ventricular thickening. Echoencephalography was normal.
routine blood test showed a white blood cell (WBC) count of 16.86*10^9/L with 88.6% neutrophils, and a liver function examination showed an aspartate transaminase (AST) level of 103 U/L, an alanine transaminase (ALT) level of 76 U/L and an albumin level of 29 g/L. The patient was asked to eat more nutritious food, given oxygen supplementation and ultimately prescribed diuretics, codeine for one month and intravenous ceftizoxime for 10 days. A few days later, he still had a mild cough but did not experience any further syncopal episodes associated with coughing. A routine blood test showed a WBC count of 6.29*10^9/L with 68.5% neutrophils. However, two months later, the syncopal episodes associated with coughing returned, albeit at a lower rate. Unfortunately, pericardectomy was never performed due to the patient’s advanced age (being a member of the oldest age group of 85 years of age and older) (5) and poor cardiac function of class III status according to the New York Heart Association (NYHA) functional classification system and, most importantly, because he and his family refused the surgery.

Discussion

A great number of patients with constrictive pericarditis require pericardectomy. However, a proportion of these patients have a predominant inflammatory and reversible pericardial reaction, which may be improved by treating the underlying cause and using anti-inflammatory agents (6). In the present case, the patient was treated with antiinfective agents and antibiotics, which stopped the syncopal episodes. We continued to monitor the patient for improvement over a period of 30 days via imaging modalities such as contrast-enhanced magnetic resonance imaging and nuclear imaging to identify whether the pericardium showed inflammation.

Syncope is a hemodynamic disorder caused by a reversible reduction in perfusion to the cerebral reticular activating system (7). During coughing, forceful contraction of the respiratory and abdominal muscles, propelling air out against a closed glottis, greatly increases the intrathoracic pressure, which decreases venous return to the heart by diminishing the pressure gradient between the central and peripheral venous systems. It also transiently increases the intracranial pressure via both direct transmission of thoracic pressure to the cerebrospinal fluid and retrograde transmission of pressure through the venous system (8). In addition, cerebral perfusion depends on the gradient between the arterial and intracranial pressures (if the latter exceeds the venous pressure); therefore, coughing may cause a significant reduction in perfusion and induce focal cerebral ischemia and syncope. In patients with constrictive pericarditis, the fundamental dysfunction involves the restriction of diastolic filling, which can reduce cardiac output, and coughing may increase cerebral insufficiency. On the other hand, analogous to the prevalent theory of neurocardiogenic syncope, if the left ventricle is forced to contract vigorously against a relatively empty chamber, this can stimulate excessive afferent mechanoreceptor discharge (2, 9), which can precipitate a rise in parasympathetic efferent activity, leading to vasodilatation and bradycardia.

In the present case, the patient did not suffer from syncope during coughing after undergoing diuresis treatment. This may be attributable to the paradoxical increase in jugular venous pressure with inspiration, which reflects the inability of the cardiac chambers to accommodate the excess venous return induced by inspiration (2, 9, 10). Therefore, when our patient was coughing, deep inspiration was induced, which caused a consequent increase in the venous return as the intrathoracic pressure decreased. Hence, coughing did not cause a drop in cardiac output.

In conclusion, constrictive pericarditis may be another factor that causes cough syncope. Future studies should investigate this phenomenon in further detail and identify the causative mechanisms.
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References