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Breathless Laughter: Tackling Tussive Syncope in the Shadows of Obstructive Sleep Apnea

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Abstract

Cough is a common complaint in the inpatient and outpatient setting. Most cases are caused by upper airway syndrome, gastroesophageal reflux disease, asthma, bronchitis, or infections. Complications that can be life-threatening include syncope and arrhythmias. Our case is of a 67-year-old obese male who had a chronic cough that led to syncope. The chronic cough was present for months, non-responsive to symptomatic treatment, and negative on outpatient work up with no clear common causes leading to hospitalization. After extensive imaging and cardiopulmonary work up yielding normal results, his underlying obstructive sleep apnea and therapy noncompliance was a likely cause. After symptomatic treatment and positive pressure therapy compliance, his cough improved, and syncope resolved. Obstructive sleep apnea, commonly seen in obese individuals, may be a contributor to tussive syncope that has not been described in the literature, which healthcare providers should be aware of.

Keywords

obesity, obstructive sleep apnea, cough, syncope, tussive syncope

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Cover Page Footnote

Milan Terzic and Yash Patel both contributed equally to the case.

Breathless Laughter: Tackling Tussive Syncope in the Shadows of Obstructive Sleep Apnea

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Abstract

Cough is a common complaint in the inpatient and outpatient setting. Most cases are caused by upper airway syndrome, gastroesophageal reflux disease, asthma, bronchitis, or infections. Complications that can be life-threatening include syncope and arrhythmias. Our case is of a 67-year-old obese male who had a chronic cough that led to syncope. The chronic cough was present for months, non-responsive to symptomatic treatment, and negative on outpatient work up with no clear common causes leading to hospitalization. After extensive imaging and cardiopulmonary work up yielding normal results, his underlying obstructive sleep apnea and therapy noncompliance was a likely cause. After symptomatic treatment and positive pressure therapy compliance, his cough improved, and syncope resolved. Obstructive sleep apnea, commonly seen in obese individuals, may be a contributor to tussive syncope that has not been described in the literature, which health-care providers should be aware of.

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Introduction

Cough syncope, or "laryngeal vertigo" as it was formerly known, was originally noted in 1876.¹ It was initially proposed to be a form of epilepsy, however, there have since been several alternative mechanisms proposed.¹ It is a well-known condition most known to occur in patients with a history of smoking and chronic obstructive pulmonary disease (COPD).² The current literature also suggests that the vast majority of these patients were specifically overweight, middle-aged males who had a comorbidity of COPD.³

Below, we discuss a patient with morbid obesity and obstructive sleep apnea (OSA) who presented with tussive syncope. We attempt to highlight the importance of identifying syncope in such patients with a possible alternative mechanism for elevated intrathoracic pressure predisposing them to syncope.

Case

A 67-year-old male with a past medical history of hypertension, depression, OSA,

hyperlipidemia, morbid obesity, and major depressive disorder presented with syncope. On presentation, the patient was describing multiple episodes of syncope over the last couple of months related to a cough. He stated that they seemed to come on after having multiple coughing bouts. Prior to hospital presentation, outpatient work-up by his primary care provider included normal upper respiratory physical exams, negative viral panels, negative chest X-rays, and no response to over-the-counter antihistamine therapy, along with a prescribed trial of intranasal corticosteroids. On review of other possible contributors, provider documentation was notable for intermittent compliance with continuous positive airway pressure (CPAP) machine and no marijuana, tobacco, or inhalation drug use. Before presenting to the hospital, the patient described sitting in a recliner and beginning to have coughing episodes. They lasted approximately 10 - 30 seconds and were followed by syncope. Surrounding family members denied any convulsive activity and noticed the patient regain consciousness within 30 seconds. The patient denied any viral illnesses, allergies, sick contacts, new medications, smoking, recent travel, or animal exposure. The patient was not on medications that are commonly associated with cough, including angiotensin-converting enzyme (ACE) inhibitors. During the review of systems, the patient denied sinus congestion, rhinorrhea, fevers, chills, chest pain, palpitations, difficulty swallowing, nausea, and orthopnea. Physical examination revealed an obese habitus,

normal breath sounds and air entry, no stridor, no wheezing, rhonchi, rales, no pharyngeal erythema, moist oral mucosa, normal tongue without bite marks, normal nasal mucosa without crusting or bleeding, normal heart rate and rhythm and without any abdominal distension or swelling. While admitted, the patient had an episode of a coughing spell followed by syncope, witnessed by medical providers. The cough was severe and followed by a staccato-like sequence at which the patient's face turned red, followed by syncope. Physical examination at this time was unchanged from prior to the initial evaluation. Vital signs and telemetry during these episodes were normal, and a spot electrocardiogram (EKG) showed normal sinus rhythm (**Figure 1**).

Laboratory workup showed a normal comprehensive metabolic panel, normal ionized calcium, hemoglobin 12.5g/dL [baseline 13g/dL], and an otherwise unremarkable complete blood count. A comprehensive viral panel was obtained, showing negative results. Urinalysis showed squamous epithelial cells but otherwise was unremarkable. Computed tomography (CT) scans of the head and neck, chest, abdomen, and pelvis were unremarkable, without any signs of emphysema, bronchiectasis, sinusitis, or obstructive masses. Due to the syncopal episodes, the patient was evaluated by cardiology, who agreed with an ischemic evaluation, given his underlying risk factors for the disease. A transthoracic echocardiogram was unremarkable, and cardiac catheterization showed normal coronary arteries and a structurally

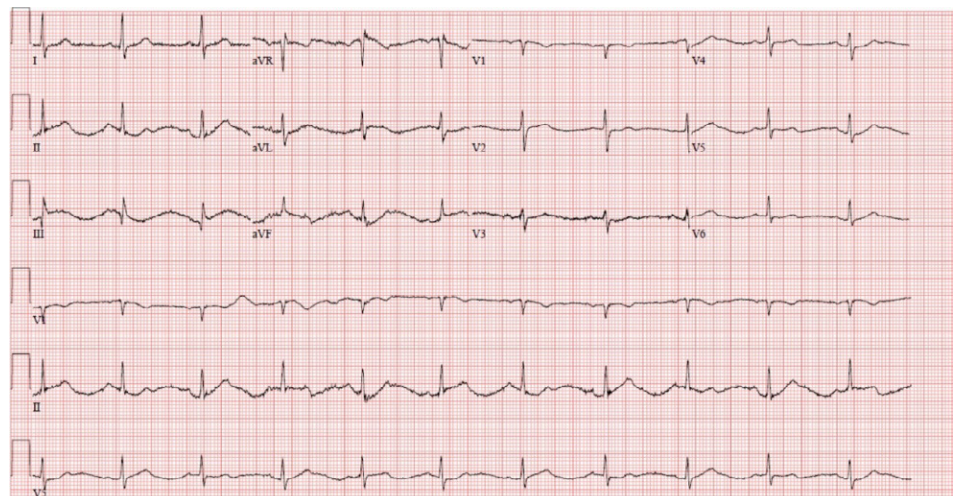


Figure 1. Electrocardiogram displaying Normal Sinus Rhythm.

normal heart. While hospitalized, the patient was placed on CPAP and symptomatically treated with dextromethorphan and guaifenesin, along with fluticasone nasal spray. This led to the successful suppression of his coughing for 48 hours. Education on compliance with CPAP was provided due to the intermittent compliance obtained during history and outpatient visits. This combination led to the resolution of his syncopal episodes while hospitalized.

The patient was seen 1-week post-discharge and stated that his coughing episodes had reduced in severity greater than in previous courses of symptomatic treatment. Further workup included a 3-month LINQ insertable cardiac monitor, a cardiac magnetic resonance imaging (MRI) to rule out the underlying scar, and outpatient pulmonary function tests, all of which did not show any abnormal results. During his subsequent visits with his primary care provider, the patient showed compliance with his CPAP machine and a syncope-free course since discharge without the use of any over-the-counter cough suppressants or antihistamines.

Discussion

Post-tussive syncope is a well-described condition with various suggested pathophysiological bases. The initial proposed mechanisms suggested it to be a possible laryngeal reflex or a result of significant peripheral vasodilation or possible pulmonary vasoconstriction. However, these proposals did not accurately account for the rapid recovery in consciousness without remaining vasomotor symptoms.⁴ An alternative pathophysiological basis for these findings was suggested due to cerebral hypoperfusion. It was previously observed by documentation of continuous blood pressure and middle cerebral artery (MCA) flow velocity data that syncope resulted when the central venous and arterial blood pressures equalized.⁵ During coughing episodes, what leads to diminished cardiac output or arterial pressure is the sudden increase in intrathoracic pressure from the cough, leading to an exaggerated Valsalva maneuver-type hemodynamic response.⁶ In one paper it was demonstrated with the use of transcranial doppler sonographies of the MCA that there was a transient cerebral circulatory arrest during coughing episodes.⁷ It was explained that the elevated intrathoracic pressures would be transmitted to the intracranial compartment, leading to transient elevated intracranial pressure impairing cerebral blood flow.⁷

Patients with COPD are at elevated risk for post-tussive syncope due to hyperinflated lungs that lead to elevated end-expiratory lung volumes, which result in elevated baseline intrathoracic pressure.⁸ In an additional case report, it was previously demonstrated that cough syncope was initially undiagnosed due to what was initially thought to be sleep apnea-induced asystole.⁹ In our patient, there was no history of tobacco use or COPD, suggesting that underlying obstructive pulmonary disease may not always be one of the main risk factors, although his body mass index (BMI) was well within the profile of a typical patient.

Cases of tussive syncope have been reported in patients with the Arnold-Chiari malformation and literature.¹⁰ It is thought to be due to compression-mediated displacement of the brainstem causing transient dysfunctions of the reticular formation. Imaging in our patient did not show any signs of Arnold-Chiari malformations. Additional causes include premature ventricular contraction (PVC) beats induced by chronic cough leading to syncope. Treatment with radiofrequency ablation of the focus leads to resolution of the syncope.¹¹ In our patient, 48-hour telemetry monitoring and 3-month outpatient LINQ monitoring did not show any arrhythmias. Untreated gastroesophageal reflux disease (GERD) leading to cough syncope has been described in the literature.¹² On questioning our patient, we found no concerns or symptoms attributed to GERD.

Our patient had an atypical presentation of tussive syncope, as the typical risk factors of asthma, heart failure, COPD, and viral illnesses causing a chronic cough were absent, leading to further investigation of the cause. The main symptoms included coughing bouts leading to short and brief losses of consciousness. Multiple outpatient evaluations and treatment failures led to hospitalization for further workup. An extensive cardiopulmonary assessment was obtained with largely unremarkable findings. However, through detailed history taking, the etiology was correlated with non-compliance with a CPAP machine for his underlying diagnosis of OSA. Long-term compliance with CPAP and brief symptomatic treatment with anti-tussive medications led to the resolution of the syncope and improvement of the chronic cough.

Conclusion

There are many etiologies and underlying conditions that can be attributed to causes of chronic cough. Ensuring a comprehensive workup is performed is essential, as tussive syncope can be treated when there is an

underlying problem. Management is typically symptomatic with antitussive medications or treatment of the underlying disorder (GERD, PVCs, Viral Illness, COPD). Obstructive sleep apnea, commonly seen in obese individuals, may be a contributor to tussive syncope that has not been described in the literature, which healthcare providers should be aware of as treatment with noninvasive ventilation provides excellent outcomes. ■

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Conflicts of Interest

The authors declare no relevant or material financial interests that relate to the research described in this paper.

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