



## Case report

## Cough syncope induced by post nasal drip successfully managed by Gabapentin

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## ABSTRACT

Syncope is a common complaint in both neurology clinic and emergency department. It is defined as transient loss of consciousness with loss of postural tone, which is usually self-limited and followed by a spontaneous recovery. Our report describes a case of cough syncope resulting from chronic intractable cough caused by post nasal drip. Although his experience was debilitating, we were able to control his symptoms significantly using a small dose of Gabapentin. This dose is much lower when compared with the already established licensed indicated higher doses used for the treatment of neuropathic pain and epilepsy. Cough syncope is a demanding condition that results in comprehensive costly investigations. In addition, cough syncope could be misinterpreted as epilepsy by the treating team. Pulmonologists should be aware of the use of Gabapentin as the management of cough refractory to standard antitussive therapy. Further studies are needed to assess the effectiveness of low doses of Gabapentin in the management of chronic cough.

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## 1. Introduction

Syncope is a common complaint in both neurology clinic and emergency department. It is defined as transient loss of consciousness with loss of postural tone, which is usually self-limited and followed by a spontaneous recovery [1]. Although most causes of syncope are benign, this symptom may presage a life-threatening event in a small subset of patients. In addition, it may provoke substantial anxiety among patients and their families and may lead to extensive investigations by the treating physicians [2]. In this article, we describe a patient who developed recurrent cough syncope which was successfully treated with low dose Gabapentin. The patient was referred with the diagnosis of generalized tonic-clonic seizures and his episodes were misinterpreted as epileptic seizures.

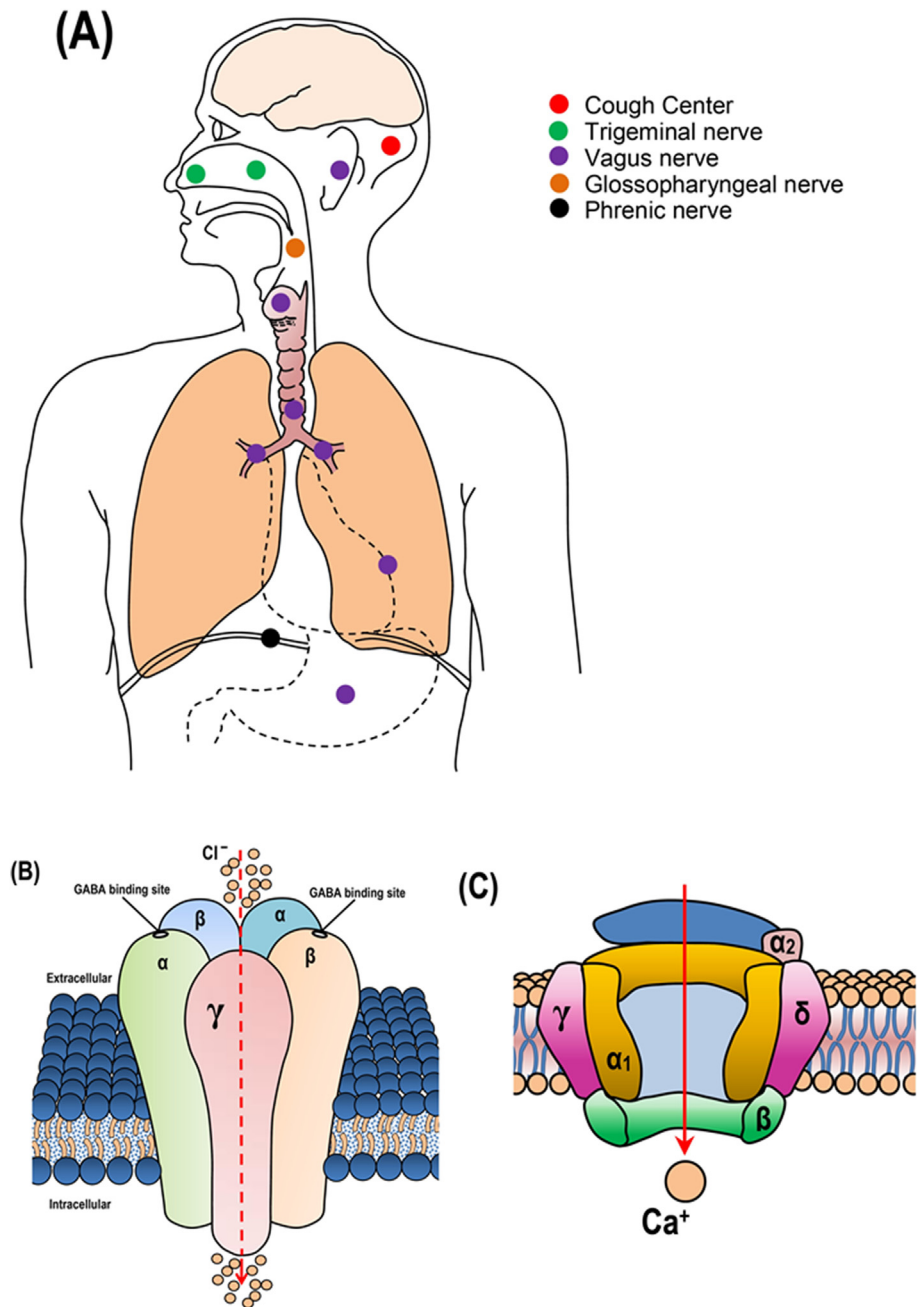
## 2. Case report

A 47-year-old male presented to the emergency department with an episodic loss of consciousness following bouts of cough for

two days. The fainting episodes occurred after seconds up to three minutes following the cough. Some of these episodes were followed by brief jerking of upper and lower extremities. Following each attack, he regained consciousness quickly with no postictal phenomena. During episodes, he was cyanosed and hypotonic. The cough started ten days prior to the presentation with a small amount of white sputum associated with streaks of blood and chest pain. He had no fever or shortness of breath but complained of nasal congestion and discharge. He had a history of Hodgkin's lymphoma treated with chemotherapy and autologous bone marrow transplantation (relapsed). He was disease free for four years. He also had a history of diabetes mellitus, hypertension, hypothyroidism, ischemic heart disease, and pulmonary embolism. His medications include antidiabetic, antidyslipidemic, and anti-ischemic heart disease medications including aspirin (81 mg). He was not on any medication known to cause cough including angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers. On examination, he was conscious, oriented, and alert. During examination, he had an episode of syncope for seconds following cough. He reported falling on his forehead during one of the episodes. His vital signs were all within normal limits with no postural drop of blood pressure or resting tachycardia. Cardiovascular and neurological examination were normal with no evidence of postural hypotension, autonomic, or length dependent

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**Fig. 1.** Schematic representation showing: (A) cough receptors involved in the normal cough mechanism, (B) structural model of GABA receptor, (C)  $\alpha_2\delta$  subunit of voltage-gated calcium channels.

peripheral neuropathy. Urgent computed tomography (CT) scan of the brain revealed no acute intracranial insult. The patient was admitted for evaluation of the cause of syncope. Investigations to evaluate syncope included brain CT scan and magnetic resonance imaging (MRI), electroencephalogram (EEG), echo-Doppler of the carotid arteries, electrocardiogram (ECG), 24 hour Holter monitoring, 24 hour blood pressure monitor, and Doppler echocardiogram. All investigations were normal which excludes cardiac and neurological causes. Investigations to evaluate the cough included chest X-ray, spirometry, bronchoprovocation test, pulmonary CT angiography, and upper gastrointestinal endoscopy. These investigations were normal, and the respiratory team suggested that the most likely cause is post nasal drip. A detailed ear, nose, and

throat examination with flexible nasolaryngoscopy was unremarkable apart from mild edema of the right vocal fold and moderate laryngopharynx acid reflux disease. The patient was given several trials of antitussive medications including codeine, lidocaine, dextromethorphan, chlorthalidol, levodropropizine, and morphine with no improvement. Following starting the patient on Gabapentin 300 mg daily, the patient responded well, and his cough episodes decreased in number until they disappeared completely. The patient was discharged home on Gabapentin 300 mg twice daily with complete resolution of his cough. In addition, post nasal drip was treated aggressively by the ear, nose, and throat team.

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