Nocturnal Choking, as an Isolated **Manifestation of Epilepsy**

Ahsan Bashir, MD¹, and Louella B. Amos, MD¹

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Introduction

Nocturnal paroxysmal events can be easily misdiagnosed. Nighttime gasping is most often associated with obstructive sleep apnea (OSA); however, sudden awakenings with a choking sensation are also features of nocturnal epilepsy. Moreover, there may be an absence of epileptiform activity on a scalp electroencephalography (EEG) interictally and ictally, which creates a diagnostic dilemma.

Case Report

An otherwise healthy 10-year-old male had a 3-week history of nighttime choking episodes, followed by stridor, difficulty swallowing, involuntary clenching of teeth and jaw, mild throat discomfort, and occasional emesis. These episodes occurred within 10 minutes of falling asleep, lasted 10 to 30 seconds, and occurred 8 to 10 times a night. He recalled half of the episodes. He reported fewer episodes when he slept upright in a recliner. He had no associated incontinence, rhythmic shaking movements, or tongue biting, and these episodes occurred only at night.

His physical exam revealed postnasal drainage, small pale ulcers on buccal mucosa, 2+ tonsils bilaterally, and Modified Mallampati Class II airway. His neurological exam was normal including his memory, speech, cranial nerves, coordination, and motor and sensory examination.

Overnight polysomnography with pH probe showed no OSA. He had 4 episodes of choking during non-rapid eye movement sleep. He was awake and interactive after the first 2 episodes (Table 1). He had 23 reflux episodes to distal esophagus, none of which were associated with his choking episodes. An awake "routine" EEG showed bihemispheric theta frequency slowing of the background activity without focal or epileptiform features. Long-term video EEG monitoring revealed 9 episodes of gasping/ choking lasting from 32 to 90 seconds with an average of 45 seconds. Filtering electromyographic artifact and remontaging uncovered rhythmic theta activity beginning approximately 10 seconds after the onset of the event and compartmentalized in F3/C3 region followed by a brief period of generalized slowing. The interictal EEG had no

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Table I.	Results of pH Probe in Relation With Events
During No	on-Rapid Eye Movement (NREM) Sleep.

Episode	Time of Episode	Sleep Stage	pН	Awake/Alert After?
I	7 Minutes after sleep onset	NREM 2	7	Yes
2	0023 (3 hours after sleep onset)	NREM 3	4	Yes
3	0100 (3.5 hours after sleep onset)	NREM 2	6	Unsure
4	0338 (6 hours after sleep onset)	NREM 2	5	No

epileptiform discharges. On retrospective review, his sleep EEG montage showed similar abnormalities associated with his gasping episodes (Figure 1). He required levetiracetam, phenytoin, and valproic acid to achieve adequate control of his seizures, and he was eventually discharged on carbamazepine and levetiracetam.

Discussion

Nocturnal choking as an isolated manifestation of epilepsy is a rare phenomenon and can be difficult to clinically distinguish from OSA. Nocturnal frontal lobe epilepsy is difficult to diagnose because the paroxysmal episodes associated with this seizure disorder, such as nocturnal wandering and nighttime gasping, can be misconstrued as sleep walking and sleep-disordered breathing, respectively.¹ The age of onset in nocturnal frontal lobe epilepsy varies widely, with 14 years being the mean age of onset. On average, 3 to 8 seizures occur per night,¹ but more than 20 episodes per night have been reported.² The episodes

¹Medical College of Wisconsin, Milwaukee, WI, USA

Corresponding Author:

Louella B. Amos, Pediatric Pulmonary and Sleep Medicine, Children's Hospital of Wisconsin, Medical College of Wisconsin, 9000 W. Wisconsin Ave MS # B620, Milwaukee, WI 53226, USA. Email: lamos@mcw.edu

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Figure I. Rhythmic theta activity compartmentalized in F3/C3 region followed by a brief period of generalized slowing.

are usually brief and the patient may be aware of surroundings with recollection of some of the events.³ Provini et al found that 97% of these episodes occur during non–rapid eye movement sleep.¹ The interictal EEG is often normal and even the ictal EEG may be equivocal, making the diagnosis clinically challenging.³

The pathophysiology linking nocturnal choking or sleep-related laryngospasm with epilepsy is unknown. The vagus nerve, via the superior and recurrent laryngeal nerve, provides the nerve supply of the larynx. The superior laryngeal nerve carries sensory information from and supplies motor output to the cricothyroid muscle, which tenses the vocal folds. Abnormal stimulation of the superior laryngeal nerve results in laryngospasm.⁴ In our case and other case reports, the frontal and/or temporal discharges may be the origin of such stimulation.⁴

Conclusion

The diagnostic approach for nocturnal gasping/choking can be challenging. Clinical history and video polysomnography supported by EEG findings, as in this case, may lead to the diagnosis, but the absence of EEG abnormalities does not exclude nocturnal epilepsy.

Author Contributions

AB: Contributed to conception and design; contributed to acquisition; drafted manuscript; critically revised manuscript;

gave final approval; agrees to be accountable for all aspects of work ensuring integrity and accuracy.

LBA: Contributed to conception and design; contributed to acquisition, analysis, and interpretation; drafted manuscript; critically revised manuscript; gave final approval; agrees to be accountable for all aspects of work ensuring integrity and accuracy.

Declaration of Conflicting Interests

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