Exploding Head Syndrome: A Case Report

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Key Words

Exploding head syndrome · Parasomnia · Headache disorder

Abstract

Introduction: Exploding head syndrome (EHS) is a rare parasomnia in which affected individuals awaken from sleep with the sensation of a loud bang. The etiology is unknown, but other conditions including primary and secondary headache disorders and nocturnal seizures need to be excluded. **Case Presentation:** A 57-year-old Indian male presented with four separate episodes of awakening from sleep at night after hearing a flashing sound on the right side of his head over the last 2 years. These events were described ‘as if there are explosions in my head’. A neurologic examination, imaging studies, and a polysomnogram ensued, and the results led to the diagnosis of EHS. **Conclusion:** EHS is a benign, uncommon, predominately nocturnal disorder that is self-limited. No treatment is generally required. Reassurance to the patient is often all that is needed.

Introduction

Nocturnal headaches comprise a variety of primary and secondary headaches including migraines, cluster headaches, and hypnic headaches. Exploding head syndrome (EHS) is an odd and rare parasomnia that can mimic nocturnal headache syndromes and seizures. Patients experience a tremendously loud noise as originating from within their head, usually described as the sound of an explosion, gunshot, door slamming, roar, waves crashing against rocks, loud voices, a ringing noise, a terrific bang on a tin tray, or the sound of an electrical buzzing. In some cases, an instant flash of what is perceived as video ‘static’ is reported both audibly and visually for a fraction of a second [1].

We present a case of a male patient diagnosed with EHS following history taking, neurologic examination, and investigations.

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Case Presentation

A 57-year-old, right-handed Indian male presented for evaluation of four separate episodes of awakening after hearing a flashing sound on the right side of his head over the last 2 years. He described these events ‘as if there are explosions in my head’.

The patient stated that all episodes occurred in the early part of his sleep and that these flashing sounds awaken him with a sudden electric jolt-like sound in the right side of his head. He was able to pinpoint the moment when the symptoms started during the night. Once awakened, he did not feel any pain or headaches. All four episodes had identical symptoms and he was in the supine position sleeping in his bed at night when the events occurred. These episodes were not associated with any autonomic symptoms, focal weakness, visual or speech disturbances, seizures, postictal confusion, or incontinence.

His other neurological complaints included mild forgetfulness and increased tiredness and fatigue, mostly towards the end of the day. He denied any difficulty in falling asleep or any disturbance in the legs to suggest restless leg syndrome. He was known to snore, but there was no evidence of any witnessed apneas or snorting. He generally woke up feeling refreshed, but as the day progressed he became more tired but not sleepy. He attributed his fatigue to stress at work and the fact that he had to make greater effort to finish his daily tasks. He denied any other history of regular headaches, migraines, or cluster headaches in the past. His family history was non-contributory except for hypertension and diabetes.

The patient’s past medical history was significant for hypertension, hypercholesterolemia, long-standing type 2 diabetes mellitus, and a hemorrhagic stroke 3 years ago with secondary mild cognitive impairment, but fortunately without physical impairment.

On physical exam, he was afebrile and normotensive without tachypnea. His body mass index was 30 with a Mallampati score of 1, and his general physical examination was unremarkable. His neurological exam suggested a right temporal homonymous hemianopsia from his previous stroke, but the rest of the results was within normal limits.

The investigations included a magnetic resonance image and a magnetic resonance angiogram of the brain, which did not show any new vascular events. The magnetic resonance image of the brain demonstrated left temporal-occipital gliosis corresponding with the patient’s prior stroke. The magnetic resonance angiogram of the brain was within normal limits. An electroencephalogram (EEG) revealed slowing in the left parietal region on an otherwise normal background. There was no evidence of any epileptiform discharges. A polysomnogram did not reveal any clinically significant obstructive sleep apnea/hypopnea syndrome.

No specific treatment other than reassurance was given. The patient was followed up 6 months later for a routine visit and did not have an interval recurrence of symptoms.

Discussion

EHS is an uncommon, usually nocturnal parasomnia that arises from the transition between different sleep stages. Patients complain of a sensation of a sudden explosive noise in the head, usually while falling asleep, which then awakens them. The sensation is very brief (typically for a few seconds only) and disappears completely when awake, although it may recur on further sleep attempts. The noise has been described as a loud bang, shotgun, or bomb explosion, and some patients have reported a simultaneous flash of light as deep sleep is entered from the lighter stages of sleep. There is no actual headache or persistent pain, but some patients may experience a brief, mild jab-like sensation. The syndrome is

often so disturbing that affected individuals are understandably quite concerned. Although EHS has been reported in patients as young as 10 years, the age of onset is usually after age 50 as in hypnic headache, and the condition has a slight female preponderance. The attacks occur with variable frequency for a few weeks or months. Attacks can be one-time events or can recur with attacks increasing or decreasing over time, sometimes with no incidents over long periods of time [1, 2].

In the present case, the differential diagnoses included nocturnal headache syndromes and seizures. Nocturnal headache syndromes include hypnic headache, cluster headaches, and migraine. All of these headache disorders usually cause the patient to awaken with an actual headache, which our patient did not have. Similarly, cephalgias occurring from subarachnoid etiologies, space occupying lesions, or obstructive sleep apnea result in persistent moderate to severe headaches, which our patient did not have either. Nocturnal seizures are prone to occur in the non-rapid eye movement sleep, but patients are mostly amnesic about the seizures. In contrast, our patient had a clear recollection of the events, without any postictal confusion and a with normal EEG. Additionally, imaging studies along with a polysomnogram also excluded the above causes. In particular, there was no evidence of periodic limb movements of sleep or another parasomnia such as non-rapid eye movement sleep without atonia. Given the history and investigations, it was therefore felt that our patient's diagnosis was EHS.

The cause of EHS is unknown. Some possibilities are a sudden movement of a middle ear component or the Eustachian tube, or perhaps a brief temporal lobe complex partial seizure (though EEG studies have generally been reported as normal) [3]. There is a correlation with stress or extreme fatigue. EHS has also been linked to rapid withdrawal from benzodiazepines and selective serotonin reuptake inhibitors [4] (which our patient was not taking).

EHS is benign and self-limited, and no specific drug therapy is warranted. Reassurance reduces anxiety; however, in some patients, EHS is very troublesome and can cause sleep-onset insomnia. For this small subgroup of patients, medications including calcium channel blockers like nifedipine [5] or flunarizine (not available in the United States) [6], topiramate [7], or clomipramine have been found to be effective in reducing the frequency or resolution of the EHS symptoms.

Conclusion

EHS is a benign, uncommon, predominately nocturnal parasomnia that can mimic primary and secondary headache disorders along with seizures. No treatment is generally required as the condition is self-limiting. Reassurance to the patient is often all that is needed.

Disclosure Statement

G.G., B.M., and A.F. declare that they have no competing interests. R.A.R. is a Deputy Editor for the *Journal of Medical Case Reports* and an Associate Neurology Editor for *Case Reports in Neurology* and *Grand Rounds*.

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