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Audiogram Bone-Conduction Testing Induced Seizure in Patient with Traumatic Brain Injury

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Abstract

In this case report, we discuss a patient who presented with Tullio's phenomenon, who also experienced bone-conduction induced seizures on two occasions. Tullio's phenomenon refers to sound induced vestibular symptoms, including disequilibrium oscillopsia, and vertical nystagmus. We were ultimately able to rule out some of the more common pathologies associated with Tullio's phenomenon for this patient based on imaging findings. However, given the specific nature of her chronic symptoms, as well as her seizure like activity in clinic, we performed a literature search to investigate other less common pathologies associated with Tullio's phenomenon. Given her past medical history of mixed psychogenic non-epileptic seizures (PNES), there is likely a somatic component to her presentation. However, given the specific and unexpected nature of these events, we propose that her symptoms may also be related to a unique inner ear pathology. Specifically, we feel that she may have exhibited symptoms of vestibular atelectasis, a relatively new otologic diagnosis characterizing the pathologic collapse of the ampulla and utricle, such that the membranous labyrinth contacts the stapes. In this way, loud sounds or changes in pressure may induce vestibular symptoms. Dizzy patients can be a difficult demographic to diagnose and manage, especially when their presentation is complicated by other functional neurologic disorders. Ultimately, we believe that this case report offers helpful insights into a new disease process associated with Tullio's phenomenon.

Keywords

Seizure, Audiogram, Traumatic Brain Injury

1. Introduction

Tullio's phenomenon (TP) describes the manifestation of sound induced vestibular symptoms, including disequilibrium, oscillopsia, or vertical nystagmus.

Most often, TP is associated with superior semicircular canal dehiscence syndrome (SSCDS). SSCDS describes the pathophysiology of a "third window" at the level of the superior semicircular canal into the inner ear. Besides TP, other common presenting features of SSCDS include pressure-induced vertigo (Hennebert's phenomenon), autophony, and pulsatile tinnitus. Patients demonstrate a conductive hearing loss (CHL) with either normal or classically negative bone conduction thresholds, signifying a conductive hyperacusis. SSCDS is definitively diagnosed with vestibular-evoked myogenic potential (VEMP) testing in conjunction with high-resolution computed tomography (CT) [1].

Although TP is classically associated with SSCDS, other etiologies include peri-lymphatic fistula, an enlarged vestibular aqueduct, and other inner ear diseases [2]. One less commonly referenced disease is vestibular atelectasis (VA). In this article, we describe a patient who presented with Tullio phenomenon with initial concern for SSCDS, but without radiographic evidence of disease. Her presentation differs from previously published reports, in that she also experienced bone-conduction induced seizures on two occasions. Given her past medical history of mixed psychogenic non-epileptic seizures (PNES), we have multiple explanations for her seizure events. However, given the specific and unexpected nature of these events, we propose that her symptoms may be related to a unique inner ear pathology.

2. Case Report

A 45-year-old female presented to an otology clinic with a seven-year history of bilateral noise-induced vertigo and pulsatile and non-pulsatile tinnitus that began after traumatic brain injury. The patient also developed seizures and migraines with aura following the accident. Her migraines are often noise-induced, and she wears a pink-noise generating device to assist with her sound sensitivities. She reports occasional seizures following migraines. Prior video-EEG in 2017 did not demonstrate epileptic seizures (ES). Outside documentation from her neurologist still reflects a diagnosis of mixed PNES and ES, although to our knowledge, she has not had a documented ES. A 2019 audiogram demonstrated left-sided supra-bone conduction at 2000 and 4000 Hz and mild conductive hearing loss. In February 2020, hearing testing had to be aborted due to seizure activity with left-sided bone conduction vibration (BCV). Given her sound-induced symptoms, the patient was advised to obtain CT-temporal bone, which demonstrated no structural abnormalities. The patient felt that her prior seizure was coincidental and agreed to repeat the audiogram. However, upon repeat testing, she experienced another seizure event with left-sided BCV. Patient was able to tolerate testing at 250 and 500 Hz, but once the level reached 1000 Hz, she became unresponsive and started what appeared to look like shivering. She then developed full body convulsions with eye fluttering, which initially lasted two minutes. She stopped convulsing for around one minute, before the episode repeated once more. After fifteen minutes, the patient recovered awareness and

responded to questions. She reported nausea and vomited several times. Patient eventually recovered and left clinic in a stable condition.

3. Discussion

Initially, there was concern for SSCDS given the patient's history of TP and severe sound sensitivities, as well as her left-sided CHL. However, the CT temporal bone effectively ruled out SSCDS, as well as other structural etiologies, such as perilymphatic fistula or cholesteatoma. Therefore, we believe that she may be demonstrating symptoms of VA, which would not be observed on CT. VA is still a new otologic diagnosis, and our patient's case offers insights into the pathophysiology of this disease, as well as how it might relate to patients with functional neurologic disorders.

To understand this patient's case, it is vital to differentiate epileptic and non-epileptic seizures. PNES are classified as a Functional Neurologic Disorder, and patients with this disorder experience convulsive events without evidence of epileptiform activity on video-EEG. In this way, our patient meets criteria for PNES, and it is likely that her two convulsive episodes in clinic represent non-epileptic seizures based on the description of the events. That being said, it is often difficult to delineate between the two based on purely observed symptoms. Based on the audiologist report of the events, she had two transient episodes of asynchronous body movements, lasting around two minutes each. She then had what seemed to be a postictal period of around fifteen minutes before she regained awareness and began responding to questions again. Her convulsions seem to align more with PNES based on a review article by Perez *et al.* from 2016, as patients with PNES are more likely to have asynchronous movements. However, the article also notes that movement asynchrony is not always a reliable differentiator between PNES and ES. PNES episodes are also characterized by longer duration, ictal crying, memory recall, and side to side head or body movements. On the other hand, ES are more often to occur from physiologic sleep and present with stertorous breathing, as well as postictal confusion [3]. After discussion with an epilepsy specialist at our institution, it seemed most likely that these episodes represented non-epileptic seizures. Interestingly, around half of patients diagnosed with PNES also have at least one additional medically unexplained symptom [3]. In our opinion, PNES does not explain the entirety of her symptoms, nor does it elucidate the mechanism of bone conduction vibration (BCV) as a trigger for PNES.

As previously described, we feel that our patient demonstrates clinical evidence of VA which was first introduced in 1988. More recently, Wenzel *et al.* further delineated the pathophysiology of TP in patients without radiographic evidence of disease. They hypothesized that the collapse of ampulla and utricle walls would bring the membranous labyrinth into contact with the stapes [4]. Ultimately, stapedial movement secondary to loud noise could stimulate either or both end organs and induce vestibular symptoms [4] [5]. Their work was sup-

ported by Eliezer *et al.*, who observed *in vivo* collapse of the utricle and ampullas (pars superior) on magnetic resonance imaging (MRI) in four patients with unilateral vestibular symptoms [4]. Unfortunately, our patient was ultimately lost to follow up, but we would expect that vestibular testing would reveal an absent caloric response or reduced vestibule-ocular reflex (VOR) gain on vestibular-evoked myogenic potential (VEMP) testing. Although we were not able to obtain additional testing to confirm this diagnosis for our patient, we feel that her unusual presentation, including seizure episode, nausea, and emesis can be explained by VA.

If she does have VA, then it is possible that BCV could have initiated her seizure episodes. Using guinea pig models, it was shown that BCV stimulates otolith irregular afferents from the utricle and saccule. The highest proportion of these afferents is stimulated at low-intensity BCV (500 Hz) [6]. In a patient with VA, activating the utricle and saccule could lead to energy transfer to the stapes, resulting in pathologic stapedial movement and vertiginous symptoms. While vertigo is not a common trigger for episodes of PNES, we feel that the emotional response to rapid and unexpected vertigo could have contributed to our patient's seizure-like activity. Other explanations for our patient's symptoms include BCV induced migraine or somatization. However, given the consistency of events and our patient's unusual history, we feel that VA better explains her symptoms. While it would have been useful to have additional work up to confirm our suspicion, this patient offers important insights into the importance of maintaining a broad differential for patients presenting with Tullio's phenomenon. While it might be tempting to attribute this patient's presentation to her functional neurologic disorder, the specific nature of her symptoms point to an otologic pathology.

4. Conclusion

Although this patient's presentation is still not completely understood, it offers valuable insights for otologic providers. "Dizzy" patients can be challenging, especially when there is not a clear structural cause for their symptoms. While Tullio's phenomenon has been classically associated with SSCDS, there is emerging evidence to suggest that the phenomenon also occurs in VA. Further work up would ideally be obtained to support the diagnosis of VA for this patient, but her clinical presentation is concerning for an otologic pathology in conjunction with her diagnosis of PNES. This case reports highlights the impact that an otologic diagnosis such as VA may have on patients with functional neurologic disorders. Ultimately, there is a paucity of literature on vestibular atelectasis, particularly in regards to patients with complex neurologic disorders, and it is our hope that this case presentation offers a platform for future study.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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