Dear Editor:

Recently, we observed the rapid recovery of a patient with persistent geotropic direction-changing positional nystagmus (DCPN) after transcutaneous vagus nerve stimulation (tVNS). Although the therapeutic mechanism of tVNS is not clearly understood, this is the first report to demonstrate that tVNS may be helpful for the management of persistent dizziness.

Methods

Patient

A 50-year-old female patient visited the emergency department of a university hospital complaining of sudden-onset vertigo. Symptoms initiated while the patient was seated at a table and worsened whenever she moved her head. One month prior, the patient had been admitted to a hospital for 2 weeks for geotropic benign paroxysmal positional vertigo of the right lateral semicircular canal (LSCC). At that time, she did not respond well to either repositioning maneuvers or vestibular suppressants. The patient had no other history of chronic illness, migraine, or alcohol or substance abuse, and did not report any auditory symptoms such as tinnitus, aural fullness, or hearing loss.

An initial bedside examination using infrared goggles (SLMED, Seoul, Korea) revealed the following findings: no spontaneous nystagmus in the supine position, similar intensities of bilateral apogeotropic DCPN in the supine head roll test, and negative findings in the horizontal head impulse test and on diffusion magnetic resonance imaging. The patient was admitted for further evaluation and treatment.

One day after admission, the patient complained of worsening dizziness when turning her head to the right side. The supine head roll test demonstrated persistent geotropic DCPN lasting >2 minutes (maximal slow-phase velocity for the right-ear-down position: 12.3°/s; for the left-ear-down position: 10.3°/s). Additionally, a null point was observed where the nystagmus stopped when the patient turned her head slightly to the right. Video head impulse test (vHIT) results indicated aberrantly increased gain in both LSCCs; however, the results of other vestibular tests were negative.

tVNS protocol

After obtaining written consent, we performed tVNS with a transcutaneous electrical nerve stimulation device (ES–420, Ito Co., Ltd., Tokyo, Japan). A ball-type electrode was placed on the cavum concha, the cymba concha, and the outer surface of the tragus in sequence (Fig. 1) [1]. Stimulation lasted for 4 minutes at each site. The pulse width was 200 μs and the frequency was 30 Hz. The amplitude was increased by 1 mA every few seconds until the threshold for patient discomfort was observed. The amplitude was then decreased to the highest threshold that could be tolerated by the patient without experiencing any discomfort.

Figure 1. Presentation of the three stimulation sites. tVNS was applied to the cymba, the cavum, and the outer surface of the tragus (encircled numbers) in a sequential order for 4 minutes at each site. The intensity was 7 mA, 5 mA, and 3 mA, respectively, on day 2, and 3 mA, 5 mA, and 4 mA, respectively, on day 3.
Results

Directly after the application of tVNS on the right side, the patient reported a tingling sensation. Surprisingly, a supine head roll test and vHIT revealed that all nystagmus had resolved and that all SCC gains had returned to a normal range.

On day 3, geotropic DCPN was once again observed, but the patient reported a lesser degree of dizziness (visual analog scale score decreased from 10 to 4). vHIT results showed a decreasing tendency of gain on the right side. tVNS on the left side had no effect on the patient’s symptoms or nystagmus.

On day 6, the patient did not report any dizziness and no nystagmus was observed, so the patient was discharged. At a follow-up examination on day 16, the patient reported that the dizziness had completely resolved. However, the gain of both LSCCs remained elevated.

Discussion and conclusions

Persistent DCPN observed in cases of peripheral vertigo can be categorized as geotropic or apogeotropic. The latter is thought to be caused by otolithic debris, but the etiology of geotropic DCPN is controversial [2]. Several mechanisms of geotropic DCPN have been previously hypothesized: 1) decreased density of the cupula due to alcohol intake; 2) increased specific gravity of the endolymph due to water-soluble macromolecules; 3) an alternative type of cupulolithiasis due to degenerated “light” otoconia; 4) free-floating inflammatory cells due to inner ear damage; 5) morphological changes in the cupula; and 6) utricular imbalance [3–6].

Repositioning maneuvers are ineffective for the treatment of geotropic DCPN. This inefficacy led Ban et al. to suggest that this condition is not associated with free-floating debris, but with the deflection of the cupula [7].

Noninvasive tVNS has been used to stimulate the auricular branch of the vagus nerve for intractable epilepsy, chronic pain, tinnitus, and coronary heart disease. It was hypothesized that tVNS corrects imbalances between the sympathetic and parasympathetic nervous systems, leading to changes in cortical organization [1,8]. In the vestibular system, activation of the semicircular canals and/or otolith organs may affect autonomic control; indeed, a vestibulosympathetic reflex has been reported [9].

In this case study, the patient showed increased gain in both LSCCs upon initial vHIT. We hypothesize that high gain may reflect the inherent hypersensitivity of hair cells in the LSCCs of some patients. This hypothesis is supported by increased gain in both LSCCs in the absence of dizziness and objective nystagmus in our patient.

Otoconia attached to the cupula may induce gravitational cupula deflection in patients, and could explain our initial observation of apogeotropic DCPN. Freeing the otoconia from the cupula or breaking down the otoconia can lead to rebound anti-gravitational cupula deflection and subsequent geotropic DCPN. All of these changes are likely to stimulate the superior vestibular nerve, which innervates the anterior and lateral semicircular canals and the utricle. Superior vestibular nerve stimulation may then in turn elicit an increased sympathetic response and autonomic imbalance. The application of tVNS may have corrected such an imbalance by normalizing LSCC hyperexcitability via a top-down mechanism. In addition, our patient only responded to tVNS on the right side. Response laterality may have been due to specific hyperactivity of the right side, and the location of a null point is strong evidence for this theory [4]. The assumption that tVNS directly suppresses vestibular afferents is an alternative explanation. In order to confirm our observations, a systematic prospective study is required to validate the utility of non-invasive tVNS for the treatment of persistent dizziness including persistent geotropic DCPN that is refractory to conventional treatment.

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