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Vestibular Syncope and Acute Peripheral Vestibular Deficit: A Case Report

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ABSTRACT

Vestibular syncope is a rare condition in which vertigo may cause syncopal attacks; however, the term has been associated with confusion because it has been ascribed to completely different vestibular and neurological conditions, from dizziness to Menière disease (MD), to the neurovegetative symptoms in benign paroxysmal positional vertigo (BPPV) and central vertebrobasilar hyperfusion. A 75-year-old woman with vasodepressive vasovagal syncope, confirmed by a tilt test with trinitrine administration, was referred for an audiological and vestibular assessment showing an acute unilateral peripheral vestibular deficit on the right side. The diagnosis is peripheral acute vestibular deficits. Interventions and outcomes are vestibular treatment and rehabilitation. The patient's vasovagal symptoms immediately improved and were completely resolved. Peripheral vestibular deficits might also trigger syncopal episodes and must be considered and studied by a complete audiological and vestibular evaluation. By restoring the peripheral vestibular function of the right labyrinth after vestibular treatment, a complete long-term resolution of multiple vasovagal syncopal episodes was observed together with normalization of the tilt test.

Taxonomy Classification: Otolaryngology

1 | Introduction

Syncope is a transient, self-limiting loss of consciousness and an inability to maintain postural tone because of reduced cerebral blood flow, which is followed by spontaneous recovery. Affecting 1%–3% of the elderly population, it represents a challenging problem in medical practice because the causal mechanism often remains uncertain even after extensive and expensive evaluations [1]. Syncope may be caused by a variety of factors, including cardiovascular, neurological, and metabolic disorders, and its presentations are quite variable. However, the most common causes of syncope are neurocardiogenic or vasovagal syncope, which account for up to 50% of cases [1]. This type of syncope is typically triggered by emotional distress, pain, or orthostatic stress and is characterized by a sudden drop in blood pressure and heart rate.

This report describes a case of recurrent syncope associated with documented peripheral vestibular dysfunction, where vestibular rehabilitation led to resolution of both vestibular deficits and syncopal episodes. The temporal correlation between vestibular treatment and the complete resolution of the syncopal episodes, supported by objective vestibular testing and a 30-month follow-up period, suggests a potential mechanistic link between vestibular dysfunction and autonomic dysregulation in selected cases of recurrent syncope.

Summary

- Acute unilateral peripheral vestibular deficit can trigger vasovagal syncope.
- Comprehensive vestibular assessment and targeted rehabilitation can lead to complete resolution of syncopal episodes.

In the literature, some authors have described cases of "vestibular syncope" in which vertigo may cause syncopal attacks [2, 3]. However, the term "vestibular syncope" determined a lot of confusion as it has been ascribed to completely different vestibular conditions, varying from dizziness [4] to Tumarkin in Menière disease (MD) [5], in which the patients experienced a severe immediate drop episodes without loss of consciousness [6]. The same term has also been ascribed to the neurovegetative symptoms in benign paroxysmal positional vertigo (BPPV) or fainting after otolithic maneuvers [7]. Vestibular syncope has also been addressed in the nonautonomic neurological causes of syncope [8] and in central vertebrobasilar hypoperfusion [9].

2 | Case Report

We report the case of a 75-year-old woman who visited the Audiology Unit of a tertiary referral University Hospital for recurrent syncopal episodes, having an episode once a year with an increase in frequency over the past 4 months, from September 2021. At the time of the first visit (November 2021), the patient had one episode per month with minor subcontinuous lipothymic episodes (once every week). The episodes always occurred with loss of consciousness in various situations but not always in the orthostatic position, mostly after meals. They were usually preceded by prodromes of pallor and sweating, a sense of prelipothymic symptoms, and nausea, and did not result in morsus, sphincter release, or clonias; the patient quickly regained consciousness without any postcrisis confusion.

Several diagnostic methods have already been employed to decipher the nature of the patient's syncopal episodes, including physical examination, laboratory testing, cardiologic, neurologic, and endocrinological examinations, and cerebral computed tomography with negative results for retrocochlear lesions. Echocardiocolor Doppler revealed a left atrial septal aneurysm, and ECG-Holter for 7 days showed a bradycardic rate with 40 sinusoidal pauses lasting < 3 s at night, but both were not deemed to be a cause of the symptoms.

The patient underwent a tilt test with trinitrin administration that confirmed vasodepressive vasovagal syncope, with presyncopal symptoms mirroring these events. A loop recorder was positioned to monitor the cardiac rhythm. After many cardiologic evaluations with negative results, she was referred to the Syncope Unit of Internal Medicine, which recommended an audiological examination to further investigate audiovestibular function. The indication was based only on the fact that nausea is a reported prodromic symptom.

3 | Clinical Findings

Bedside examination showed a right acute peripheral vestibular deficit with a slight reduction in the vestibulospinal reflexes on the right side in Romberg and Unterberger tests (60°). Ocular movement evaluation showed only hypometric saccades on the left side, whereas with the Frenzel eyeglasses, horizontal spontaneous nystagmus of the first and second degree to the left side in both supine and upright positions was observed. Spontaneous nystagmus is inhibited by fixation. No paroxysms were observed in any placement.

3.1 | Diagnostic Assessment

Pure tone threshold, speech audiometry, and immittance audiometry were normal.

The video-head impulse test (V-HIT), videoculography (VOG), and caloric testing (Veitz 20°) showed significative peripheral vestibular asymmetry of the vestibulo-ocular reflexes (right < left). In particular, the VOG also highlighted irregular saccades with increased latency and reduced accuracy on the right side, within age-adjusted normal range for age, along with a mainly leftward saccadic pursuit at low velocities, which improved at higher velocities; spontaneous nystagmus recording revealed left-beating horizontal nystagmus, grades I–II; visuo-vestibulo-ocular reflex gain was at the lower limit within the normal range on both sides, and a bilateral cervical nystagmus was detected. V-HIT and caloric testing confirmed an acute right peripheral vestibular hyporeflexia.

3.2 | Therapeutic Intervention

In December 2021, the patient immediately started a vestibular rehabilitation cycle including eight sessions of cervical electrical stimulation [10]. The patient also started daily administration of supplements containing choline, phospholipids, essential fatty acids omega 3–6, thioctic acid, tryptophan, taurine, vitamin E, vitamins B, magnesium, zinc, and selenium for 2 months, repeated every 3 months.

At the end of the rehabilitation cycle (December 2021), the patient reported immediate subjective relief from symptoms (nausea and prelipothymic episodes).

After the rehabilitation cycle (January 2022), immediate improvement in the vestibular-ocular reflex (VOR) was observed, restoring the balance between the two sides (Figure 1). In particular, the gain increased from 20% to 56% and the symmetry index increased from −21% to −4%. Visuo-vestibular-ocular reflex (VVOR) gain, phase, and symmetry index also reached normal symmetric values (gain 76 vs. 86, phase −1° vs. +3° and symmetry index −6% vs. +2% for the right side; 74% vs. 94%, −1° vs. +4°, and 11% vs. −2% for the left side, pre- vs. postrehabilitation values, respectively). This improvement was confirmed by static posturography. The audiovestibular examination 1 month after therapy showed that the resolution of the clinical picture was constant.

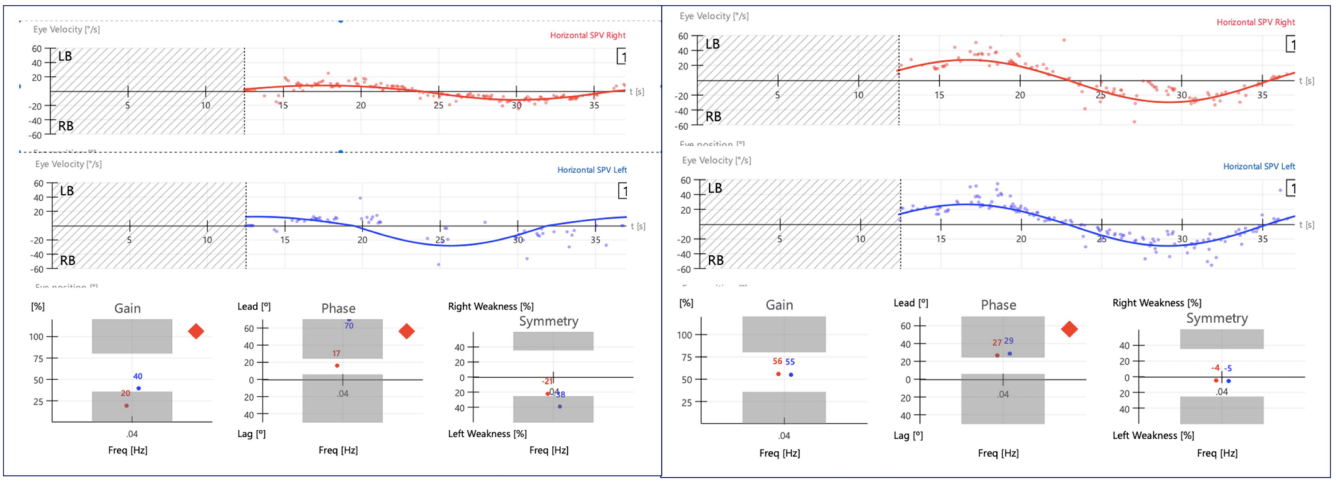

FIGURE 1 | VOR before and after rehabilitation of the right side peripheral vestibular deficit.

During the follow-up period, with an audiological checkup every 3 months, the resolution of the acute right vestibular deficit and the absence of syncopal attacks were confirmed. The loop recorder positioned in November 2021, which did not detect any anomaly during the entire period, was removed in June 2023. The tilt test was repeated during the same period and showed normal responses.

Currently, the patient has no syncopal or lipothymic episodes (at a follow-up: of 2 years and 6 months).

4 | Discussion

This case highlights the importance of considering a vestibular involvement in patients presenting with syncopal episodes.

The vestibular nuclei are related to nucleus solitarius and nucleus ambiguous, dorsal motor nucleus of the vagus, and caudal and rostral ventrolateral medulla [11]. The cardiovascular center decreases the sympathetic outflow by inhibiting the rostral ventrolateral medulla and spinal sympathetic preganglionic neurons; in contrast, the parasympathetic outflow stimulates the nucleus ambiguus and dorsal motor nucleus of the vagus [12]. This baroreflex controls the cardiovascular tone sustainably, but it takes up to 2 s to change the blood pressure, not enough to maintain the homeostatic condition in response to cardiovascular perturbation, especially for the human who continues to move up and down, resulting in a large vertical fluid motion within the body [13]. Therefore, vestibulo-sympathetic reflexes, principally from semicircular canal stimulations, are considered to participate in maintaining the cardiovascular tone, especially in response to sudden postural change [14]. In fact, erroneous vestibular information from the otolithic organs, during BPPV, might alter the vestibular-sympathetic reflex that normally compensates for a relative body fluid shift associated with every motion [5], and a sympathetic hyperactivity and parasympathetic suppression has been proved in the irritative vestibular attacks of MD [15]. In this case report, it is deemed that an acute unilateral vestibular deficit might cause a dysautonomia caused by the overexpression of the parasympathetic tone. It is very interesting and peculiar to this case that a successful vestibular treatment provided a relief from the vasovagal symptoms up to almost 3 years of follow-up.

Author Contributions

Noemi Motta: data curation, writing – original draft. **Marco Gitto:** writing – original draft. **Valeria Castelli:** investigation. **Eleonora Tobaldini:** investigation. **Nicola Montano:** conceptualization, investigation. **Federica Di Berardino:** data curation, visualization, writing – original draft, writing – review and editing.

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Ethics Statement

All procedures performed in the study were in accordance with the ethical standards of the institutional and/or national research committee and the 1975 Helsinki Declaration, as revised in 2008. This case report, written according to the CARE guidelines, is in line with the principles of our internal IRB (Ethics Committee of Fondazione IRCCS Ca' Granda, Ospedale Maggiore Policlinico of Milan, Italy [identification number: 616, June 19, 2019:473_2018]).

Consent

The patient provided written informed consent for the publication of the study. Patient anonymity was guaranteed.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

All relevant data are within the paper. Any additional information and data are available from the corresponding author on reasonable request.

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