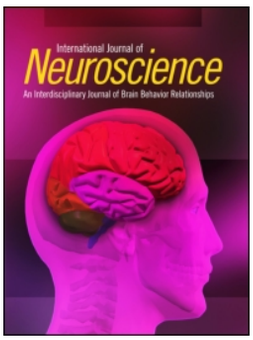

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CASE REPORT

Multiple sclerosis attack case presenting with pseudo-vestibular neuritis

Reyhan Surmeli[a], Mehmet Surmeli[b], Ayse Destina Yalcin[a], Ayse Asli Sahin Yilmaz[b] and Ferhat Kucuk[b]

[a]Department of Neurology, University of Health Sciences, Umraniye Training and Research Hospital, Istanbul, Turkey; [b]Department of Otolaryngology, University of Health Sciences, Umraniye Training and Research Hospital, Istanbul, Turkey

ABSTRACT

Purpose: Pseudo-vestibular neuritis is a clinical diagnosis for patients presenting with acute vestibular syndrome due to a central pathology.

Case report: We reported a case of multiple sclerosis characterized by pseudo-vestibular neuritis. Our case was a 32-year-old male patient. The patient, who was diagnosed with multiple sclerosis in September 2019, came to the emergency clinic in January 2020 with the complaint of severe vertigo, vomiting-nausea. A newly developed demyelinating plaque was detected in the left vestibular nucleus in cranial MRI. The patient had no hearing loss. On examination of the patient, nystagmus findings supporting peripheral vestibular involvement were present on the left side. Neurologic examination showed left-sided hyperactive deep tendon reflexes, Achilles clonus, dysmetria, ataxia to the left and plantar reflex with extensor response on the left. Video head impulse test and cervical evoked myogenic potential tests were performed. Vestibular hypofunction was present on the left side. Steroid pulse therapy was administered as 1000 mg/day, i.v for 7 days. After treatment, his complaints decreased. In addition, there was an improvement in examination findings.

Conclusion: Multiple sclerosis is shown to be an etiological factor in patients with pseudo-vestibular neuritis.

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KEYWORDS
Multiple sclerosis; pseudo-vestibular neuritis; vertigo; video head impulse test; cervical evoked myogenic potential

1. Introduction

Acute dizziness or vertigo symptoms appear clinically as acute vestibular syndrome (AVS) and positional vertigo. AVS is defined as the sudden onset of acute, 'continuous' vertigo or dizziness (lasting longer than 24 h), coupled with nausea, vomiting and head motion intolerance [1]. The most common cause of AVS is acute peripheral vestibulopathy, but some patients with AVS have an acute central vestibular syndrome which results from lesions with pons, inferior cerebellum or vestibular cortex involvement [2]. Therefore, in patients with AVS, abnormal neurological examination, lack of improvement of symptoms in two days, postural imbalance, central patterns on vestibular function tests suggest a clinical pathologic condition called pseudo-vestibular neuritis [3,4].

Multiple sclerosis (MS) can lead to a wide variety of clinical presentations. Vestibular symptoms may develop as the initial sign of disease or during the course of disease. Dizziness is a symptom reported at a rate of 49–59% and vertigo is found in 5% of MS patients [5].

Clinical presentations of patients with acute demyelination causing AVS are not recognized as frequently as other more common causes, such as vestibular neuritis. Here, we aimed to discuss a pseudo-vestibular neuritis (pseudo-VN) case with its clinical and radiological features which developed due to acute demyelination.

2. Case report

2.1. Medical history

A 32-year-old male patient presented to the emergency clinic in January 2020 with severe dizziness and accompanying nausea-vomiting for 3 days. There was no headache, neck pain or hearing loss. The patient had a previous history of MS. In 2017, the patient had a complaint of numbness in the left arm and leg lasting more than 24 h, and MRI showed a finding in favor

CONTACT Reyhan Surmeli reyhansurmeli@gmail.com Department of Neurology, University of Health Sciences, Umraniye Training and Research Hospital, Istanbul, Turkey

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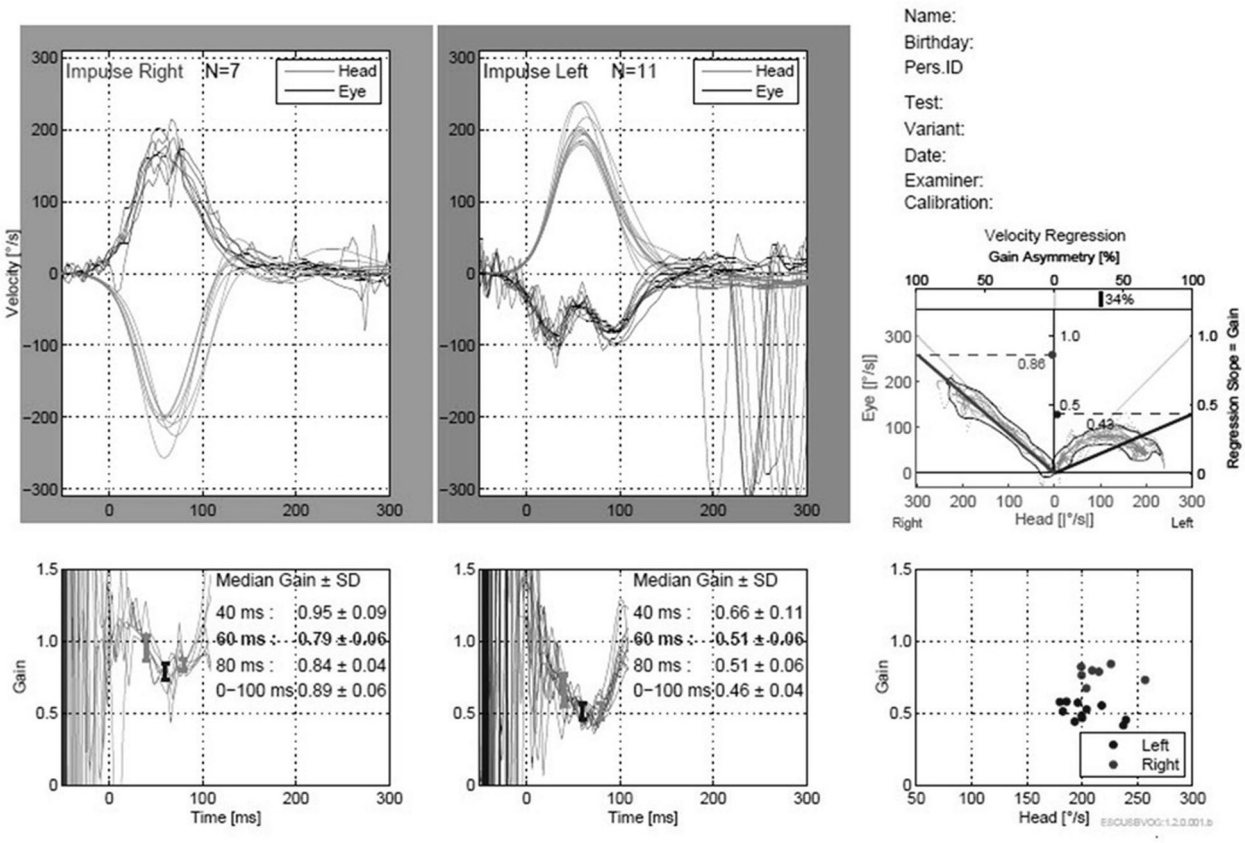

Figure 1. In the Video Head Impulse Test (vHIT) performed during the pseudo-VN attack, Vestibuloocular Reflex (VOR) Gain was low on the left side (Gain 60 ms: 0.51 ± 0.06). The velocity regression has shown 34% asymmetry on the left side. In addition, corrective overt saccades were detected.

of an active demyelinating lesion in the right parietal region. The patient, who had left peripheral facial paralysis lasting more than 24 h in September 2019, had cranial, cervical and thoracic lesions showing contrast enhancement in the MRI taken at that time. Oligoclonal band type 2 is evaluated as positive. After that patient met the McDonald Multiple Sclerosis Criteria, which were revised in 2017 [6]. He was on dimethyl fumarate treatment with a dosage of 120 mg two times a day.

2.2. Clinical findings

Otoscopic examination was bilaterally normal. Neuro-otologic examination revealed a right-sided horizontal-rotatory spontaneous nystagmus. Nystagmus accelerated in the fast phase direction. In head impulse test, there was a catching saccade on the left side (Supplemental Video 1). In addition, there was no skew deviation. The HINTS examination was performed. Head impulse test was abnormal on left side. There is a right-sided horizontal-rotatory spontaneous nystagmus and absent skew. Neurologic examination showed left sided ataxia, dysmetria on the left side, hyperkinetic deep tendon reflex in the left upper and lower extremities and left Achilles clonus. The left side plantar reflex showed an extensor response. In pure tone audiometry, there was no hearing loss in both ears.

Electrophysiological tests were performed. Video head impulse test (vHIT) was evaluated on the left side as abnormal (VOR Gain: 0.51) (Figure 1). In the cervical vestibular evoked myogenic potential (cVEMP) test, there was no response on the left side (Figure 2). Responses obtained in vHIT and cVEMP tests were considered normal for the right side.

2.3. Radiological findings

Contrast-enhanced cranial magnetic resonance imaging (MRI) was performed which showed a newly developed demyelinating plaque in the left vestibular nucleus. The current MRI and the previous MRI (3 months earlier) are shown in Figures 3 and 4. In addition, MRIs had demyelinating lesions located in bilateral periventricular white matter, centrum

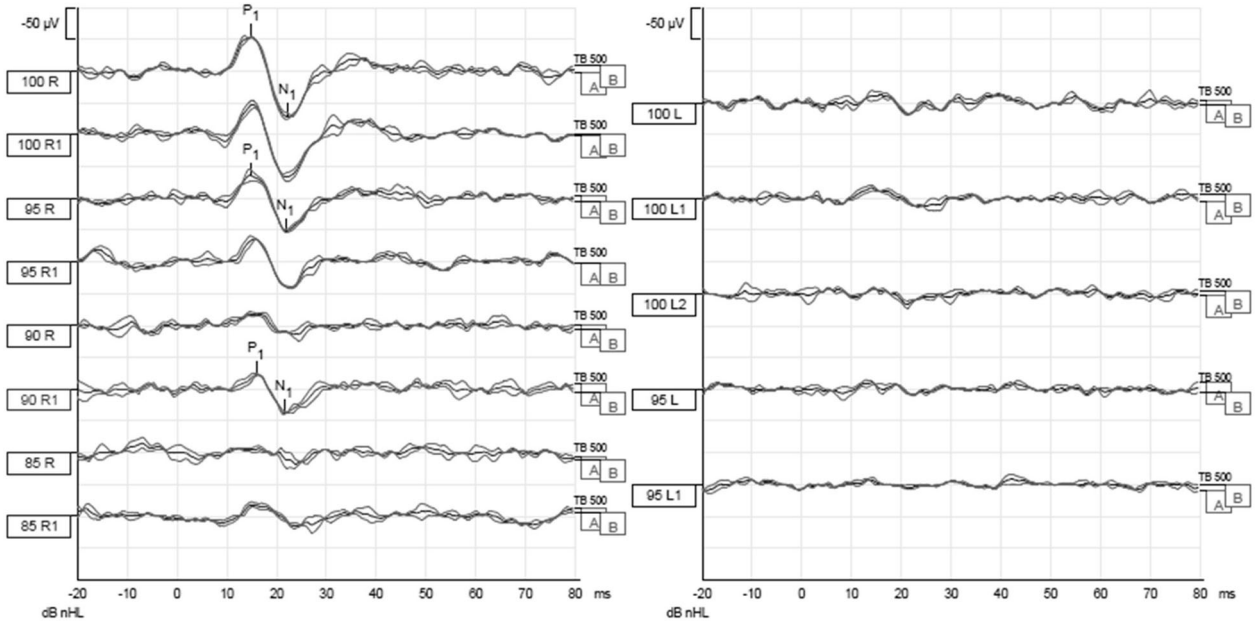

Figure 2. In the cervical evoked myogenic potential (cVEMP) test performed during the pseudo-VN attack, there was no response on the left.

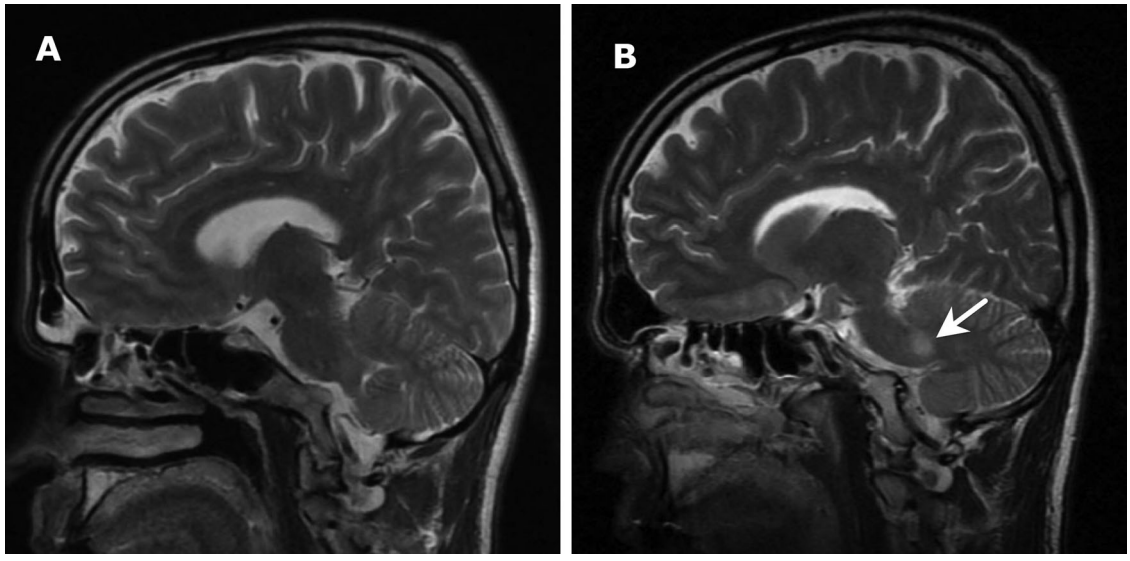

Figure 3. Sagittal T2 weighted brain MRI in September 2019 (A). Sagittal T2 weighted brain MRI in January 2020. The responsible demyelinated lesion of the vestibular nucleus is observed on the left side (B).

semiovale, corona radiata, frontoparietal convexity, cervical C3 and thoracic T8.

2.3.1. Clinical course

The patient's acute developing vertigo symptoms was considered as a MS attack. Steroid pulse therapy was administered as 1000 mg/day, i.v for 7 days. A marked improvement in the patient's vertigo symptoms was observed within days. Spontaneous nystagmus disappeared in his neuro-otologic examination. Also, bedside head impulse test was found to be negative on both sides. Prophylactic therapy with dimethyl fumarate 120 mg two times a day was continued.

3. Discussion

Around 10% of patients with demyelinating diseases suffer from vertigo and nausea – clinical features mimicking vestibular neuritis (VN) [1]. Only one study to date has reported seven cases of acute vestibular syndrome, which have been shown to be due to demyelinating disease [7]. Therefore, we aimed to share the

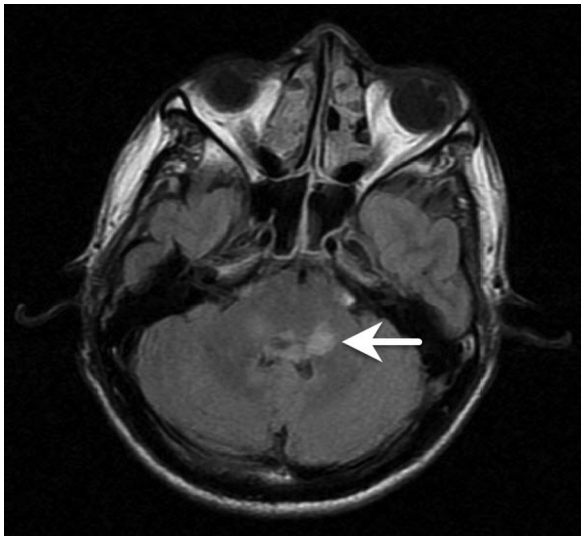

Figure 4. Axial Flair weighted brain MRI in January 2020. The demyelinated area in the vestibular nucleus is shown.

clinical and radiological features of our MS-related pseudo-VN case.

Vertigo and dizziness are common in the evolution of multiple sclerosis and vertigo is reported as the presenting symptom of up to 15% of MS patients [8]. Besides, demyelinating diseases are not as frequent causes of acute vertigo as stroke or peripheral vestibular diseases [1].

In the otological examination of our patient, bilateral normal hearing, nystagmus findings supporting peripheral vestibular involvement were present. Consistent with these findings, the head impulse test was positive on the left side. However, the presence of pathological findings that support central involvement in the neurological examination of the patient suggested that pseudo-VN would be a possible diagnosis. Then, in the patient's brain imaging, the localized lesion in the vestibular nucleus caused by central demyelinating plaque was thought to be the cause of central vestibulopathy. Clinically, it is important to differentiate central vestibular syndrome from acute peripheral vestibulopathy.

In the presence of pathological neuro-otological examination findings that clinically support central vestibulopathy, brain MRI is mandatory. As a result of radiological examinations, it is accepted that the most common cause of AVS in MS develops by affecting either the vestibular nucleus or the fascicular part of the eighth nerve of a lesion in the lower pons or upper medulla [9]. In our case, the location of the lesion was found at the level of the left vestibular nucleus.

Further clinical tests that may help to differentiate between VN and pseudo-VN are the cVEMP and vHIT. These tests are useful bedside tests in AVS patients as they are quick and easy to perform. cVEMP evaluates the vestibulo-collic reflex and is mostly used in peripheral diseases such as acoustic neurinoma, Meniere's disease, but also provides information about the brainstem. vHIT evaluates the gain of vestibulo-ocular reflex (VOR), and the abnormal vHIT is suggested to be the most reliable single tool for prospectively differentiating between central and peripheral causes of AVS [10]. In our patient, horizontal-torsional nystagmus, increasing in intensity in gaze towards the fast phase was consistent with the peripheral lesion. vHIT was positive on the same side and in the cVEMP test, there was no response on the left side, which suggested an acute VN.

In this case, there were clinical signs of central involvement accompanying peripheral oculomotor involvement. This suggests that pseudo-VN clinic of our patient was due to a demyelinating plaque in the vestibular nucleus.

4. Conclusion

MS is an uncommon cause of pseudo-VN. The clinico-anatomic correlation of the cause of pseudo-VN in MS has never been reported. In patients with AVS, there may be abnormal central neurological examination findings accompanying vestibular neuritis findings. These clinical findings should suggest pseudo-vestibular neuritis. As in our case, central involvement and peripheral vestibular system findings can be detected together. Consequently, the presence of any atypical signs for acute VN (here left-sided ataxia/dysmetria) should exclude a peripheral source and MRI imaging should be performed.

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Disclosure statement

None of the authors have a conflict of interest.

Ethical approval

All procedures performed in study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

ORCID

Reyhan Surmeli http://orcid.org/0000-0002-0215-0614
Mehmet Surmeli http://orcid.org/0000-0002-7936-2957
Ayse Destina Yalcin http://orcid.org/0000-0003-4719-5741
Ayse Asli Sahin Yilmaz http://orcid.org/0000-0002-7846-9453
Ferhat Kucuk http://orcid.org/0000-0001-8146-5675

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