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Vestibular Schwannoma Presenting as Acute Vertigo Mimicking Vestibular Neuritis

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Keywords
Vestibular schwannoma · Vestibular neuritis · Vertigo · Dizziness · Cerebellopontine angle

Abstract
Vestibular schwannoma (VS) is commonly accompanied by hearing loss, tinnitus, and dizziness and tends to be chronically progressive in nature. We report a case of VS presenting as left vestibular neuritis (VN) in a previously healthy 57-year-old patient. Right-beating horizontal-torsional spontaneous nystagmus was observed, and the bedside head impulse test revealed a left catch-up saccade. The bithermal caloric test showed left canal paresis, and pure-tone audiometry revealed an average threshold of 22.5 dB bilaterally. Brain magnetic resonance imaging (MRI) demonstrated a 0.7-cm enhancing mass in the left internal auditory canal, consistent with VS. The patient was administered with high-dose systemic corticosteroids and vestibular suppressants with antiemetic, which relieved acute vertigo. Although dizziness in VS is chronically progressive in nature, VS may present as an acute vestibular syndrome that mimics VN. VS should be considered a potential cause of acute vestibular syndrome, and thorough neurological examination with MRI may be helpful for accurate diagnosis.

Introduction

Vestibular schwannoma (VS) is a common benign intracranial tumor that accounts for approximately 6% of all intracranial neoplasms [1, 2]. The most common manifestation of VS is slowly progressing sensorineural hearing loss with tinnitus. Hearing loss, tinnitus, and

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dizziness are present in 39–95%, 45–75%, and 49–66% of patients with VS, respectively [3, 4]. Although mild vertigo is assumed to be common in VS, features of vertigo in VS have been poorly characterized [5]. Approximately 9% of patients with VS report chronic dizziness as the first symptom [5], and approximately 31% of patients with VS have chronic dysequilibrium [6]. In addition, the symptoms may mimic those observed in Meniere's disease in 4–5% of the cases [7]. Thus, dizziness in VS generally has chronic or episodic features. Here, we report a case of VS in a 57-year-old man who presented with characteristic clinical manifestations of vestibular neuritis (VN).

Case Presentation

A 57-year-old man visited the emergency room because of acute continuous vertigo for 3 h. He did not complain of hearing loss, tinnitus, or headache. On video-Frenzel goggle examination, right-beating horizontal-torsional spontaneous nystagmus obeying Alexander's law was observed (online suppl. Video 1; for all online suppl. material, see www.karger.com/doi/10.1159/000527989). The nystagmus direction was unaffected by positioning maneuvers, such as in the Dix-Hallpike and head-roll tests. The bedside head impulse test revealed a left catch-up saccade. Neurological examination, including the cerebellar function test and lower cranial nerve examination, revealed no abnormalities. Skew deviation was not detected, and hearing evaluation using tuning forks was unremarkable. The patient was clinically diagnosed with VN and admitted to our department.

Pure-tone audiometry showed an average threshold of 22.5 dB bilaterally (Fig. 1a), with a speech discrimination score of 92% bilaterally. The click-evoked auditory brainstem response revealed a threshold of 45 dB bilaterally and no significant delay in latencies on the left side (Fig. 1b). The bithermal caloric test demonstrated left canal paresis of 72% (Fig. 1c), and the video head impulse test showed reduced gains in the left horizontal and anterior semicircular canals (Fig. 1d). The cervical vestibular-evoked myogenic potential test revealed no response on the left side (Fig. 1e). Contrast-enhanced brain magnetic resonance imaging showed a 0.7-cm enhancing mass in the left internal auditory canal, consistent with VS (Fig. 2).

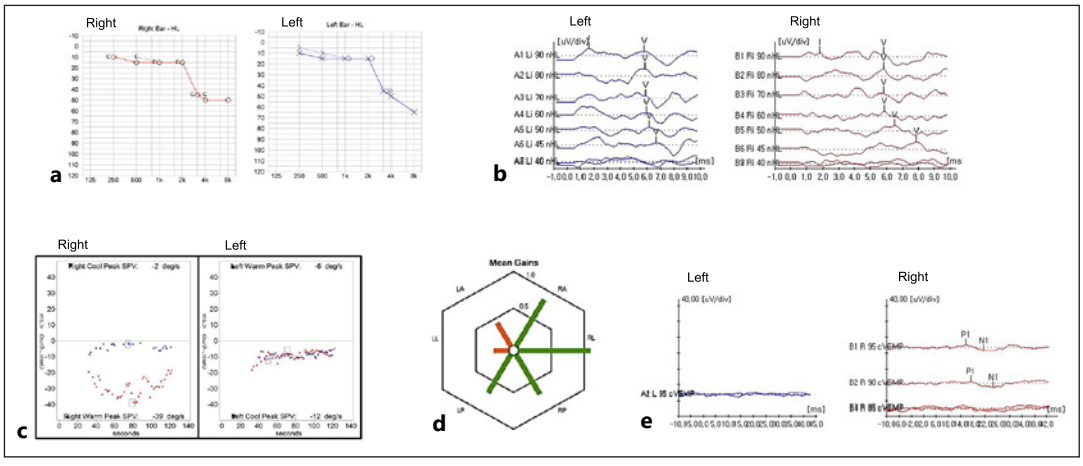

Fig. 1. a Bithermal caloric tests reveal left unilateral canal paresis. **b** Pure-tone audiometry shows an average threshold of 22.5 dB bilaterally. **c** Bithermal caloric tests reveal left unilateral canal paresis. **d** Video head impulse tests reveal significantly decreased vestibulo-ocular reflex gain in the left horizontal and anterior semicircular canals. **e** The cervical vestibular-evoked myogenic potential test shows no response on the left side.

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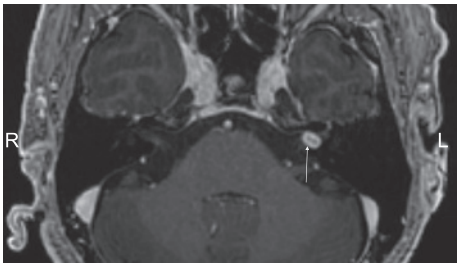

Fig. 2. Brain magnetic resonance imaging (MRI). T1-weighted contrast-enhanced MRI reveals a 0.7-cm enhancing mass in the left internal auditory canal (arrow).

The patient was administered with high-dose systemic corticosteroids (prednisolone 1 mg/kg/day for 4 days, tapered off for the subsequent 10 days) and vestibular suppressants with antiemetic to relieve acute symptoms. Vestibular rehabilitation exercises were recommended when the patient's condition was tolerable. After discharge, the patient was referred to a neurosurgeon for follow-up.

Discussion

Common symptoms of VS, such as hearing loss, tinnitus, and dizziness, generally show a chronic progression. Although VS has often been reported to present with sudden hearing loss [8], it has rarely been reported to present as VN. Sunara et al. [9] reported a patient previously diagnosed with VS who presented with acute vertigo caused by VN; however, in that patient, vertigo was complicated with worsening of hearing loss and tinnitus. The authors suggested that acute vestibular syndrome in that patient could be explained by overlapping VN in the vestibular nerve previously affected by VS [9]. We reported a case of VS that initially presented with VN without hearing loss or tinnitus.

Patients with VS complain of dizziness, including dysequilibrium or vertigo. Dizziness is the first symptom of VS in 7–9% of patients [5, 10]. Because dizziness in VS tends to be chronic and progressive, approximately 50% of patients present with imbalance at the time of diagnosis [10]. Regarding the etiology of dizziness and vertigo in VS, the tumor or underlying comorbidity has been suggested. Vertigo is attributable to compression of the cerebellum or pons by tumors and impingement of the vestibular nerve by the tumor growing into the internal auditory canal [11]. VS with comorbidities, such as vestibular migraine and benign paroxysmal positional vertigo, may cause vertigo [12]. In addition, Kentala and Pyykko reported that clinical presentations of VS and Meniere's disease were similar in 14% of patients with VS, and the differentiation between VS and true Meniere's disease may be difficult in these patients [13]. In our patient, the clinical manifestation mimicking VN can be explained as follows: (1) the size of intracanalicular VS might have increased because of intralesional edema, impinging the eighth cranial nerve; (2) VN occurred in the eighth cranial nerve already compromised by VS; (3) acute vestibular syndrome is an initial vertiginous episode of VS mimicking Meniere's disease [13]; (4) acute ischemia is caused by tumor. The finding, that the gain of the posterior semicircular canal in the video head impulse test was normal (Fig. 1d), may favor "acute vestibular neuritis" theory rather than compression of nerve as a cause of acute vestibular syndrome. Further studies are warranted to assess the clinical features of these patients. In conclusion, although dizziness in VS is chronically progressive in nature, VS may present as an acute vestibular syndrome mimicking VN. VS should be considered a potential cause of acute vestibular syndrome, and thorough neurological examination with magnetic resonance imaging may be helpful for accurate diagnosis. The CARE Checklist has been completed by the authors for this case report, attached as supplementary material.

Statement of Ethics

This study was reviewed and approved by the Institutional Review Board of Konkuk University Medical Center (No. 2021-03-087). Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflicts of Interest Statement

The authors declare that there is no conflict of interest.

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Author Contributions

Joon Yong Park wrote the first draft of the paper and interpreted the data. Chang-Hee Kim supervised the study and critically reviewed the manuscript. All authors contributed to the clinical care of the patient and have read and approved the final manuscript.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to their containing information that could compromise the privacy of research participants.

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Case Rep Neurol 2022;14:464–468
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