Neurovascular compression syndrome (NVCS) of the 8th cranial nerve typically presents with paroxysmal audiovestibular symptoms due to vascular compression of the nerve at the central segment lying in the cerebellopontine angle (CPA). This disorder can cause various combinations of auditory and vestibular symptoms, which have been called different disease categories, such as vestibular paroxysmia (VP) [1,2], disabling positional vertigo [3], typewriter tinnitus [4,5], and cochleovestibular compression syndrome (CVCS) [6]. Diagnostic criteria for CVCS and VP have been reported and revised recently, all of them defining CVCS and VP in terms of clinical symptoms and response to antiepileptic medications [1,2,4,5]. In this case report, we present a case of CPA meningioma presenting as NVCS of the 8th cranial nerve and discuss the value of laboratory tests.

This study has been approved by the Institutional Review Board of the Hallym University Sacred Heart Hospital (No. 2017-I059).

CASE REPORT

A 54-year-old woman presented to our clinic with a primary complaint of repeated tinnitus with spinning vertigo of 2 years’ duration. The paroxysmal audioves-
tibular symptoms lasted approximately 10 seconds and were triggered when she rapidly rotated her head laterally. She also had decreased hearing and aural fullness in her left ear. She showed spontaneous nystagmus and head-shaking nystagmus with a fast phase beating to the right during examination with Frenzel goggles. Audiovestibular laboratory tests and magnetic resonance imaging (MRI) of the internal auditory canal were scheduled for 1 week later and anticonvulsant medication (oral oxcarbazepine, 300 mg/day) was prescribed. She returned to the clinic after all the laboratory tests were completed. She reported that the paroxysmal dizziness and tinnitus had ceased, but once when she missed a dose of medication, the symptoms reappeared. She had a normal audiogram with 100% word recognition scores in both ears. Caloric testing showed 52% canal paresis in the left ear. Auditory brainstem response (ABR) testing showed an abnormal waveform on the left side (Fig. 1). MRI revealed a well-enhanced, 3.6 cm broad-based mass in the left CPA (Fig. 2). She underwent neurosurgical excision as well as postoperative gamma knife radiotherapy for the residual mass. Subsequent histopathologic examination confirmed a meningothelial meningioma. The patient discontinued anticonvulsant medication after surgery, and the paroxysmal attacks of dizziness and tinnitus resolved, though she had some continuous dizziness that improved with vestibular rehabilitation exercises.

**DISCUSSION**

NVCS of the 8th cranial nerve is characterized by repeated brief attacks of vestibulocochlear symptoms. It typically improves dramatically when treated with...
low-dose anticonvulsant medication, but microvascular decompression surgery is indicated for medically intractable cases [7,8]. Audiometry and vestibular function tests are usually recommended, as many patients exhibit signs of mild-to-moderate unilateral hypofunction during attack-free intervals, which may be helpful in determining the affected side [3,4]. ABR and MRI have been suggested as diagnostic tests for NVCS but both have questionable diagnostic efficacy. In a previous case series, pathologic ABR changes were found in less than one-third of cases [2]. Neurovascular contact demonstrated by MRI is only suggestive and does not definitively diagnose NVCS even with a high-tesla (7T) experimental machine [5].

The roles of both ABR testing and MRI in identifying NVCS need further evaluation. However, we suggest performing either ABR testing or MRI when evaluating patients with suspected NVCS of the 8th cranial nerve to exclude mass lesions in the CPA, which can be fatal. In this case, although the clinical symptoms and response to anti-epileptic treatment fulfilled the diagnostic criteria for VP, abnormalities found through ABR testing and MRI led to a different final diagnosis. The CPA meningioma stereotypically leads to symptoms relating to mass effect on cranial nerves III, V, VII, and VIII, while in NVCS syndrome, audiovestibular symptoms arise from ephaptic transmission between demyelinated axons by the pulsatile compression of the responsible vessel. Thus, there could be differences in symptom presentation between the two disease categories. However, the symptoms related to the compression on the 8th cranial nerve would be similar, whatever the compressing tissue is. We speculated that the mass effect on the 8th cranial nerve could also lead to audiovestibular symptoms mimicking those in NVCS. Still, the conclusion about pathomechanism should be substantiated with further study.

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REFERENCES
7. De Ridder D, Heijnen K, Haarman B, van der Loo E. Tinnitus in vascular conflict of the eighth cranial nerve: a surgi-