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What is This?
A Case of Isolated Cerebellar Hemorrhage Presenting as Vestibular Neuritis Combined with Contralateral Benign Paroxysmal Positional Vertigo

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Keywords
- cerebellar hemorrhage, vertigo, vestibular neuritis, benign paroxysmal positional vertigo

Case Report

A 61-year-old female patient without any cardiovascular risk factors was hospitalized after acute onset of severe vertigo, nausea, and vomiting that had continued for 4 days. The patient showed spontaneous, left-beating horizontal nystagmus in the primary and left gazing positions (Figure 1A). After head shaking, the degree of left-beating nystagmus was augmented, consistent with the findings typically seen in acute VN of the right side (Figure 1B). Left-beating horizontal nystagmus remained unchanged during the Dix-Hallpike test. However, direction-changing horizontal nystagmus was induced by rotating the patient’s head from the right to the left while in the supine position (Figure 1C, D). Head thrust test (HTT) did not reveal a corrective saccade typically seen in contralateral BPPV involving the horizontal semicircular canal. Magnetic resonance imaging (MRI) of the brain was performed to confirm VN and evaluate any central lesions. MRI demonstrated left cerebellar hemorrhage in the subacute stage (Figure 2).

The patient was transferred to a neurovascular center and carefully observed without any special treatment. Vertigo disappeared completely by the third day of hospitalization, and no abnormality was found in the vestibular function tests performed 4 days after disappearance of vertigo. The patient was discharged after improvement of hematoma on hospital day 14, which was confirmed by follow-up brain computed tomography. The present study was approved by the Institutional Review Board of Kwandong University College of Medicine.

Discussion

There have been numerous reports on pseudo-VN, but most cases involved cerebellar infarction in the territory of the medial branch of the posterior inferior cerebellar artery (PICA).²,³ Disruption of blood supply to the vestibulocerebellum by PICA infarction is known to cause disconnection of the inhibitory nodulovestibular Purkinje fibers, leading to hyperactivity of the ipsilesional medial and superior vestibular nuclei with resultant spontaneous unidirectional ipsilesional nystagmus.³ Central dysmetria was not seen, and there were no other signs suggesting cerebellar dysfunction. The results of auditory brainstem-evoked potentials and pure-tone audiogram were unremarkable.

The patient was initially considered to be in the subacute stage of VN on the right side combined with contralateral BPPV involving the horizontal semicircular canal. Magnetic resonance imaging (MRI) of the brain was performed to confirm VN and evaluate any central lesions. MRI demonstrated left cerebellar hemorrhage in the subacute stage (Figure 2).
Paroxysmal positional vertigo is another central vestibulopathy that results mostly from central lesions involving the cerebellar vermis or the fourth ventricle. Although the pathophysiology of CPPV is not completely understood, an abnormal pathway between the vestibular nucleus and the archicerebellum located at the central portion of the cerebellar vermis has been suggested as a possible cause.

The present case demonstrated cerebellar hemorrhage in the territory of the medial branch of PICA, including portions of the cerebellar nodulus and left cerebellar hemisphere, leading to presentation of the clinical signs suggesting VN. Because there was no direct involvement of cerebellar vermis by the hemorrhage, signs of CPPV and positional nystagmus were suspected to be the result of peripheral edema and mass effect of the hemorrhagic lesion compressing the fourth ventricle. To our knowledge, no such case of cerebellar hemorrhage presenting with clinical features of both VN and BPPV has been reported in the literature. Despite the lack of any risk factors or clear neurological signs to suspect a central pathology, there were subtle findings that could have been easily overlooked without careful attention. Normal HTT and direction-changing positional nystagmus (DCPN) provided clues to perform brain MRI in this patient. Although DCPN is recently considered to be associated more with horizontal canal BPPV than central vestibulopathy, DCPN suggesting BPPV on the contralateral side of VN raised suspicion of a central cause.

Physicians should pay careful attention to the subtle findings that could aid in differentiating peripheral and central vertigo so that brain imaging can be administered in selected patients at an appropriate timing.

**Author Contributions**

Dae Bo Shim, data collection/analysis, writing; Chang Eun Song, data collection/analysis; Seung Jae Baek, data collection/analysis; Mee Hyun Song, data collection/analysis, writing.

**Disclosures**

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