A Case of Tinnitus and Hearing Loss After Cerebellar Hemorrhage

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Background: Approximately 10% of cases of hypertensive intracerebral hemorrhage are cerebellar hemorrhage. We report a case of intracerebellar hemorrhage in which the initial symptoms were hearing loss and tinnitus.

Summary of Report: A 45-year-old man suddenly complained of hearing loss and tinnitus on the right side. Computed tomography demonstrated a high-density area 1.2 cm in diameter in the right cerebellar peduncle. Because of enlargement of the high-density area, the patient underwent surgery based on the diagnosis of hypertensive intracerebellar hemorrhage, and the hematoma was evacuated. The right-sided acoustic nerve was remarkably swollen by a subpial hematoma.

Conclusions: In this case, hemorrhage of the right cerebellar peduncle extended to the eighth nerve through the subpial space, causing hearing loss and tinnitus. (Stroke 1993;24:906-908)

Key Words • cerebellar hemorrhage • hearing disorders

Cerebellar hemorrhage constitutes approximately 10% of the cases of hypertensive intracerebral hemorrhage.1 The characteristic clinical symptoms of cerebellar hemorrhage include headache, vomiting, vertigo, ataxia of the body and the four extremities, and occasionally gaze disturbance and dysarthria.2-4 In the present report, a rare case of intracerebellar hemorrhage, whose initial symptoms were hearing loss and tinnitus, is presented.

Case Report

A 45-year-old man who had been treated for hypertension, diabetes, and hepatopathy for several years was admitted on June 16, 1989, to the Department of Otology of Saiseikai Goutsu General Hospital with sudden right-sided hearing loss, tinnitus, and pain of the lateral angle of the right eye. At that time, right peripheral facial palsy and Bruns' nystagmus were observed. Audiology demonstrated right sensorineural hearing loss in the low-frequency area (Figure 1), and low response was observed in a caloric test of the right side. An auditory brain stem response (ABR) test showed prolongation of wave I latency on the right side (1.84 msec) compared with the left (1.76 msec). Computed tomographic (CT) scan showed a high-density area 1.2 cm in diameter in the right cerebellar peduncle (Figure 2). The patient's symptoms thereafter gradually deteriorated, and a repeat enhanced CT scan on day 7 revealed enlargement of the high-density area. The patient was referred to the Department of Neurosurgery at Shimane Prefectural Central Hospital.

On admission, the patient's blood pressure was 158/90 mm Hg. He was alert but complained of right-sided hearing loss, tinnitus, and pain of the lateral angle of the right eye, with diplopia. Headache and vomiting were absent. Neurological examination revealed peripheral facial palsy, Bruns' nystagmus, right intentional tremor on the nose-finger-nose test, and ataxic gait. After admission, however, the patient began to complain of headache and vomiting, accompanied by scanning speech. Repeat CT scan showed a high-density area 4.5 cm in diameter in the right cerebellar hemisphere that was compressing the brain stem (Figure 3). Retrograde vertebral angiography showed a right-to-left shift of the bilateral posterior inferior cerebellar arteries, with no aneurysms or vascular malformations. On June 23, 1989, based on the diagnosis of hypertensive cerebellar hemorrhage, right suboccipital craniotomy was performed, and the hematoma was evacuated. The cerebrospinal fluid was watery clear. The hematoma was found in the middle cerebellar peduncle, extending into the right cerebellar hemisphere. The site of primary hemorrhage was considered to be the middle cerebellar peduncle. No vascular malformation was found. Close examination of the right cerebellopontine angle showed a remarkably swollen acoustic nerve, with a hematoma extending from its interior to the surface (Figure 4). The trigeminal, glossopharyngeal, and vagus nerves were normal in appearance.

A postoperative CT scan confirmed total removal of the hematoma. Rehabilitation enabled the patient to walk 2 weeks after surgery. Improvements were also observed in tinnitus, peripheral facial palsy, and scanning speech. The patient was then discharged with a slight ataxic gait and diplopia. Audiography performed 6 months after surgery revealed improvement in the patient's right sensorineural hearing loss in the low-frequency area (Figure 1).
Preoperative audiogram

Postoperative audiogram

**FIGURE 1.** Preoperative and postoperative audiograms. Preoperative audiogram shows right sensorineural hearing loss in low-frequency area. Postoperative audiogram indicates improvement in right sensorineural hearing loss in low-frequency area.

**Discussion**

The pathways to and from the cerebellum pass through the three cerebellar peduncles. Afferent pathways of the anterior spinocerebellar tract and efferent fibers that originate from the dentate, emboliform, globose, and fastigial nuclei pass through the superior cerebellar peduncle. The middle cerebellar peduncle is a large nerve bundle formed by afferent fibers from the contralateral pontine nuclei. The afferent fibers reach the cortex of the opposite cerebellum, except for the flocculonodular lobe forming the secondary neurons communicating with the corticopontine tracts. The inferior cerebellar peduncle consists of afferent fibers from the vestibular nuclei that end in the flocculonodular lobe, afferent fibers of the olivocerebellar tract, and efferent pathways of the fastigiobulbar tract.

Hemorrhage into the cerebellar vermis causes severe ataxia, but when it extends from cerebellar lingula to the superior medullary velum, fourth nerve palsy develops; if it extends to the anterior spinocerebellar tract that passes through the superior cerebellar peduncle, severe tremor of the ipsilateral upper extremity occurs. Hemorrhage to the cerebellar hemisphere generally causes ataxia of the four extremities, dysmetria, asynergia, adiadochokinesis, and scanning speech. When the dentate nucleus and its efferent pathway are involved, intentional tremor develops. If hemorrhage occurs in the flocculonodular lobe, astasia, abasia, trunc-
cal ataxia, vertigo, and nystagmus develop because of damage to the vestibular feedback circuit. In these cases, nystagmus is maximum toward the ipsilateral side, and sometimes reaction in the caloric and rotational tests of vestibular function is lost. When the hemorrhage extends to the fourth ventricle, elevation of intracranial pressure because of obstruction of the fourth ventricle or the cerebral aqueduct causes headache, vomiting, and papilledema. In addition, when the hemorrhage reaches the brain stem through the cerebellar peduncles, symptoms such as facial palsy, gaze palsy, abducens palsy, and dysphagia occur.

To our knowledge, this is the first reported case of a patient with intracerebellar hemorrhage who exhibited initial symptoms of hearing loss and tinnitus. The symptoms in the present case can be explained by analyzing the CT scan and intraoperative findings. Hemorrhage of the middle cerebellar peduncle damaged some of the crossed pontocerebellar fibers, but the damage was not of a sufficient degree to cause such symptoms as ataxia of the four extremities. In this case, we assume that the crossed pontocerebellar fibers, trigeminal nerve, cochlear nerve, vestibular nerve, and abducens nerve were damaged by hemorrhage of the middle cerebellar peduncle; therefore, hearing loss, tinnitus, and diplopia developed. Thereafter, as the hemorrhage increased and spread to the right cerebellar hemisphere, ataxia of the four extremities, intentional tremor, and scanning speech developed.

References
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