Discussion. Sinusitis should be recognized as an early sign of aspergillosis and often presents with new onset headache. Untreated, it can be followed by symptoms of chronic meningitis and ischemic or hemorrhagic stroke. *Aspergillus* species have an affinity for invading blood vessels and spreading along the internal elastic lamina of these vessels, inducing vasculitis and thrombosis.

Fungal cultures of CSF often remain negative. Galactomannan applied to serum can be used as screening tools for the identification of patients with invasive aspergillosis.² The sensitivity of this test in immunocompromised patients has been reported up to 90% (specificity 93-99%), but is decreased by antifungal therapy.2 A few studies have reported detection of galactomannan in CSF in patients with cerebral aspergillosis.3-7 CSF galactomannan sensitively detected cerebral aspergillosis with 12 of 13 reported patients having elevated CSF indexes.3-7 Serum antigen titers were reported in seven of these patients and all were substantially elevated. Serum galactomannan antigen titers were normal in our patients, whereas CSF indexes were elevated. Detection of circulating galactomannan in serum requires substantial leakage of antigens from the site of infection into the circulation, which might not be present in isolated cerebral aspergillosis. A prospective multicenter study on the diagnostic value of galactomannan in patients with cerebral aspergillosis is needed. Our findings indicate that, in cases of cerebral aspergillosis, galactomannan antigen may be detected in CSF when serum indexes are normal.

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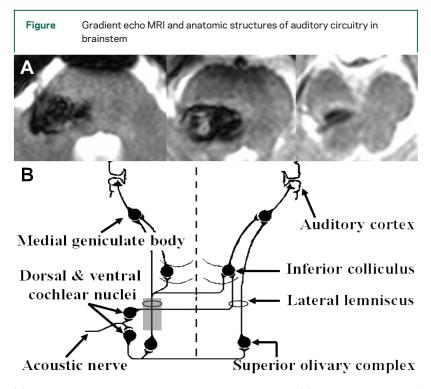
CONTRALATERAL HYPERACUSIS IN UNILAT-ERAL PONTINE HEMORRHAGE

Hyperacusis, an increased sensitivity to auditory stimulation,¹ may occur in patients with peripheral auditory system dysfunction, migraine, depression, and certain infectious diseases. However, hyperacusis is a rare manifestation of CNS lesions.² Previous studies have reported paracusia (altered perception of loudness, timbre, or pitch) and palinacousis (perseveration of aural sensation) after medial geniculate body hemorrhage,³ auditory hallucination due to pontine hemorrhage,⁴ and bilateral hyperacusis due to caudal tectal hemorrhage.⁵ We report a unique patient presenting with unilateral hyperacusis due to a pontine hemorrhage. Case report. A 51-year-old hypertensive woman suddenly felt that environmental sounds were heard louder in her left ear than before. She said that "I feel as if an amplifier is inserted into my left ear hole." At the same time, a machinery, "MRI noise-like" sound was heard in her left ear. She also had unpleasant tingling sensation in her left hemibody. On admission to a six-bed room of our hospital, she could not tolerate the noise, especially a loud, high pitched female voice, and requested us to move her to a more quiet two-bed room. She could not sleep in the left decubitus position, partly because tactile auricle stimulation aggravated the hyperacusis and tinnitus (allodynia) and partly because of the uncomfortable

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(A) Gradient echo MRI shows right, dorsal pontine hemorrhage. (B) Anatomic structures of auditory circuitry in brainstem. Shaded area indicates the lesion involving the auditory pathway between superior olivary nuclei and lateral lemniscus.

noise developed when her left auricle was rubbed against a pillow. The hyperacusis was exaggerated when she got tired or hungry. There was no complex auditory or visual hallucination.

She denied any history of ear diseases, seizures, psychiatric illness, or regular ingestion of drugs or alcohol. On neurologic examination, she was alert and well-oriented. In the Weber test, the sound was lateralized to the left while Rinne test was bilaterally positive. Other cranial nerves were normal except for subtle bilateral gaze evoked nystagmus. She had mildly decreased perception on pinprick, warm and positional sensation, but had hyperpathia on cold stimulation in her left face, trunk, arm, and leg. She had continuous paresthesia in the left hemibody which was described as if her left body was put on cold iron armor. There was mild clumsiness and ataxia in the left arm and leg. Brain MRI showed a hemorrhage in the right dorsal pons (figure, A). There were no lesions in other parts of the brain including the midbrain, thalamus, or temporal lobes. CT angiography revealed no abnormal vessels.

On otologic examination, tympanic membranes and eustachian tubes were intact. The results of pure tone audiometry, speech audiometry, tympanometry, stapedial reflex (acoustic reflex) test, and blink reflex test were all normal. Brainstem auditory evoked responses (BAERs) showed normal latencies of wave I to V with normal wave forms on both sides. In addition, receptive and expressive language abilities were normal. She showed normal results in speech detection threshold, speech discrimination, and speech recognition test. The result of click fusion test revealed that her temporal auditory processing ability was normal.

She was treated with antihypertensives and gabapentin 600 mg/day. One month later, hyperacusis and tinnitus persisted, but much diminished in intensity. At this time, she could sleep in the left decubitus position.

Discussion. In our patient, the sound was lateralized to the left on the Weber test, which could suggest ipsilateral conductive hearing loss or a contralateral sensorineural hearing loss. However, with positive Rinne tests, and the normal results of otologic examination, pure tone audiometry, speech audiometry, tympanometry, acoustic reflex test, and BAERs, we could exclude the possibility of peripheral auditory pathway dysfunction. Thus, the sound lateralized to the left seems to reflect hypersensitized aural pathway in the left. Our imaging results suggest that the lesion may have involved the pathway between the superior olivary nuclei and lateral lemniscus (figure, B). Although stapedial reflexes and BAERs revealed normal results, this may be due to the redundancy of the central auditory system above the level of the cochlear nuclei2 or incomplete injury of the structure.

The uncomfortable paresthesia in her left body part was probably related to the involvement of the ascending sensory tract. This socalled central pain or paresthesia has been shown to be related to development of hyperexcitation secondary to partially damaged sensory tracts, or damage to central inhibitor pathways.6 Similarly, the hyperacusis shown in this patient may be a hypersensitization phenomenon after the partial injury of the central auditory pathway. Indeed, a recent study on dystonia related to chronic regional pain syndrome showed that 38% of patients reported hyperacusis which was associated with hyperalgesia and allodynia.7 Thus, our patient shows that unilateral hyperacusis may develop after a pontine lesion affecting the central auditory pathway possibly through hypersensitization phenomenon.

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MULTIPLE INTRACEREBRAL HEMORRHAGES AFTER CERVICAL EPIDURAL INJECTIONS

Epidural steroid injections are commonly used to treat chronic pain secondary to radiculopathy. Complications of epidural injections include unintentional dural puncture, vasovagal syncope, apnea, and hypotension. Cervical epidural injections are associated with higher rates of complications than lumbar or caudal epidural injections.¹ Recent American Academy of Neurology guidelines do not recommend epidural injections for radicular pain.² We report a rare complication of low CSF pressure headache and multiple simultaneous intraparenchymal hemorrhages, occurring 7 days after cervical epidural injection.

Case report. A 36-year-old left-handed woman with a history of a motor vehicle accident 1 year prior to admission presented with leftsided weakness and paresthesias of her right hand. She had chronic cervical neck pain since her motor vehicle accident, and was treated with a series of four cervical epidural anesthetic injections. She had no complications following the first three injections. The patient then had a fourth cervical epidural injection with no indication of dural puncture. However, approximately 4 days after this epidural injection she developed a positional, holoacranial, throbbing headache. The headache became severe when she was in the upright or sitting position and dramatically decreased with recumbency. She was encouraged to increase her fluid intake, but her symptoms did not resolve.

Seven days after her fourth epidural injection, she developed new-onset numbness and tingling in her right hand and weakness in her left upper and lower extremities. On admission, she continued to complain of a diffuse, throbbing headache, which increased in severity upon sitting up. Vital signs and general examination were normal. Neurologic examination demonstrated slightly dysarthric speech, a left central facial palsy, and a left-sided hemiparesis with inability to perform fine motor movements with her left hand. She had decreased pinprick, vibration, light touch, and proprioception in the left hemibody and distal right upper extremity. Her reflexes were bilaterally brisk and Babinski sign was present bilaterally.

Head CT (figure, A) showed hemorrhages in the right frontal and right and left parietal lobes, centered in the region of the gray–white junction. MRI (figure, C) showed low-lying cerebellar tonsils and diffuse pachymeningeal enhancement (figure, B) consistent with intracranial hypotension. There was no evidence of sinus thrombosis or arterial malformation.

MRI of cervical spine was not made available; however, it was reportedly normal. Coagulation studies and hepatic and autoimmune laboratory panels were normal. She refused confirmatory lumbar puncture. She was treated with IV hydration and caffeine. The headache was greatly reduced after 3 days and she regained strength in her left hemibody, but her sensory abnormalities persisted at the time of discharge.

Discussion. Intracranial hypotension (or hypovolemia) is characterized by a postural headache that is worse on sitting or standing and relieved with recumbency. Other features include neck stiffness, nausea and emesis, diplopia, dizziness, change in hearing, and photophobia. Intracranial hypotension can be spontaneous or secondary to recent spinal procedures. The characteristic MRI brain findings are diffuse pachymeningeal enhancement (following gadolinium), downward displacement of the brain, subdural fluid collection or hematoma,

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