

Sensorineural hearing loss due to delayed cerebral ischemia in bilateral auditory cortices following aneurysmal subarachnoid hemorrhage: illustrative case

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BACKGROUND Recognizing rare signs of delayed cerebral ischemia (DCI) is crucial to caring for patients with subarachnoid hemorrhage. The authors presented a case of central hearing loss that occurred during the clinical course of a patient treated for aneurysmal subarachnoid hemorrhage.

OBSERVATIONS The patient had a ruptured right posterior communicating artery aneurysm successfully treated with coil embolization but later developed severe vasospasm and DCI. She developed bilateral hearing loss, and imaging revealed DCI to the left temporal lobe and the right auditory cortex. Computed tomography angiography and digital subtraction angiography demonstrated severe vasospasm of bilateral internal carotid arteries, bilateral middle cerebral arteries, and bilateral anterior cerebral arteries. One month after hospitalization, the patient had recovered fully neurologically intact except for persistent hearing loss.

LESSONS This case serves to teach important neuroanatomical features and discuss the unique pathophysiology of DCI affecting the auditory cortex.

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KEYWORDS subarachnoid hemorrhage; sensorineural hearing loss; delayed cerebral ischemia; aneurysm

Aneurysmal subarachnoid hemorrhage (aSAH) causes high morbidity and mortality; the case fatality rate is approximately 40% to 50%, and many survivors have chronic neurological deficits.¹ Delayed cerebral ischemia (DCI) is a leading cause of death and disability among patients with aSAH.^{2,3} Hemiparesis, aphasia, and altered consciousness are the most common symptoms of DCI⁴ although any cerebral tissue distribution could be affected. Reports describing rare sequelae of DCI are valuable contributions to clinical research as well as important educational resources. Some degree of hearing impairment occurs in approximately 15% of patients with SAH;⁵ however, central hearing loss is a unique complication of DCI due to bilateral afferent pathways to each auditory cortex. Here we present a case of central hearing loss that developed secondary to DCI and severe bilateral vasospasm after aSAH.

Illustrative Case

A 38-year-old woman developed the worst headache of her life and was brought to the hospital by her family. At the time of arrival

to the emergency department, she was somnolent and required emergency intubation. Her initial Hunt Hess grade was 4; she briskly localized and exhibited purposeful movements to stimulation but did not follow commands. Computed tomography arteriography (CTA) of the head showed a ruptured right posterior communicating aneurysm, Fisher grade 3 SAH (Fig. 1). A left frontal external ventricular drain was placed for management of hydrocephalus, and coil embolization of the aneurysm was performed successfully.

After the procedure, the patient was admitted to the intensive care unit and treated with oral nimodipine and permissive hypertension (approximately 140 to 180 mm Hg) as DCI prophylactics in addition to daily transcranial Doppler studies to monitor cerebral blood flow. The patient was extubated on post-bleed day (PBD) 3, at which time she was able to open her eyes in response to verbal commands, had full mentation elicited with gesturing, and followed commands in all extremities without noted asymmetry in her ability to orient to verbal commands. However, she declined neurologically

ABBREVIATIONS ABR = auditory brainstem response; ACA = anterior cerebral artery; aSAH = aneurysmal subarachnoid hemorrhage; CTA = computed tomography arteriography; DCI = delayed cerebral ischemia; ICA = internal carotid artery; MCA = middle cerebral artery; MRI = magnetic resonance imaging; PBD = post-bleed day.

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FIG. 1. Admission and intraoperative imaging showing severity of SAH and location of culprit aneurysm. **A:** CT at time of admission revealed Fisher grade 3 distribution of blood. Of note, in addition to SAH along the circle of Willis and bilateral Sylvian fissures, intraparenchymal hemorrhage was apparent in the left temporal lobe (*blue arrowhead*). **B and C:** Cerebral angiograms showing multilobulated right posterior communicating artery aneurysm (*white arrows*).

on PBD 4 and became somnolent, only awakening to painful stimuli and requiring significant prompting to follow commands. CTA demonstrated bilateral internal carotid artery (ICA), bilateral middle cerebral artery (MCA), and bilateral anterior cerebral artery (ACA) vasospasm.⁶ Blood pressure augmentation was implemented, with clinical improvement in the degree of mental status and ability to orient to verbal commands. On PBD 5, staff noted that patient had difficulty hearing. An MRI study performed with vestibulocochlear protocol showed confluent restriction in the right parietal region and bilateral Sylvian fissures (right > left) that involved the inferior frontal gyrus, superior temporal gyrus, and insula; right temporal gyrus and right auditory cortex; and the left temporal lobe involving left superior temporal gyrus and left auditory cortex (Fig. 2).

The patient received multiple endovascular procedures to treat the refractory vasospasm, including intraarterial verapamil and angioplasty. Severe vasospasm of bilateral ICAs, ACAs, and MCAs was noted during these procedures (Fig. 3). There was minimal to no vasospasm of the posterior circulation. The patient was eventually discharged to an inpatient rehabilitation facility after having ventriculoperitoneal shunt placement, tracheostomy, and percutaneous endoscopic gastrostomy tube placement. At time of discharge, she could open her eyes, regard, and have purposeful movement in all

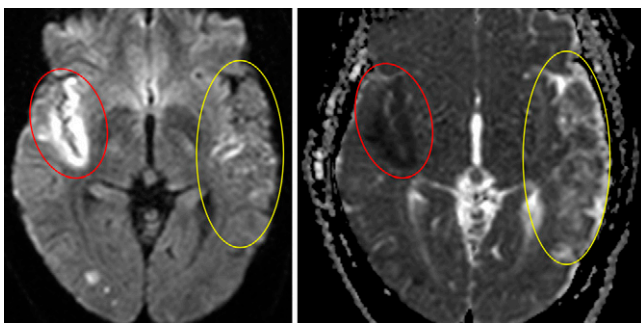


FIG. 2. MRI showing cortical ischemic injuries. MRI with vestibulocochlear protocol was performed on the fifth day after SAH when the patient was first noted to have difficulty hearing. Diffusion-weighted imaging (*left*) and apparent diffusion coefficient (*right*) images obtained from the MRI study showed acute ischemia in the right superior temporal gyrus (*red oval*) and encephalomalacia in the left temporal lobe with significant volume loss (*yellow oval*), consistent with auditory cortex injury and central hearing loss.

extremities. At 6-month follow-up, she was neurologically intact except for persistent hearing loss. The patient reported steady improvements and stated she felt back to baseline aside from the hearing loss and

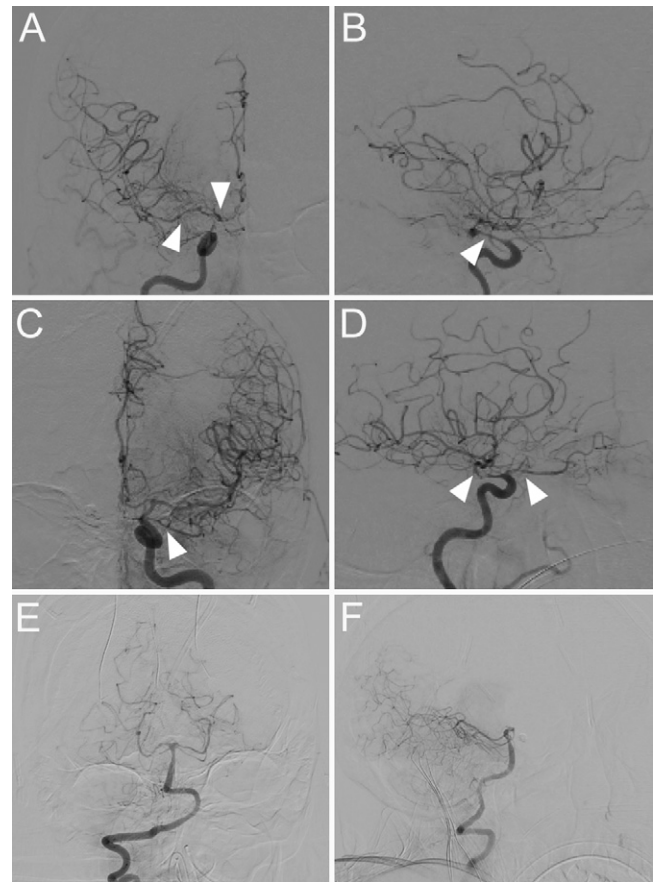


FIG. 3. Angiographic images showing evidence of vasospasm. After neurological decline 4 days after SAH, the patient received endovascular treatment for refractory vasospasm. Cerebral angiography during these procedures confirmed vasospasm involving bilateral ICAs, bilateral MCAs, and bilateral ACAs. Vasospasm in the posterior circulation was minimal. **A and B:** Right ICA angiograms. **C and D:** Left ICA angiograms. **E and F:** Right vertebral artery angiograms. *White arrowheads* indicate areas of most severe vasospasm.

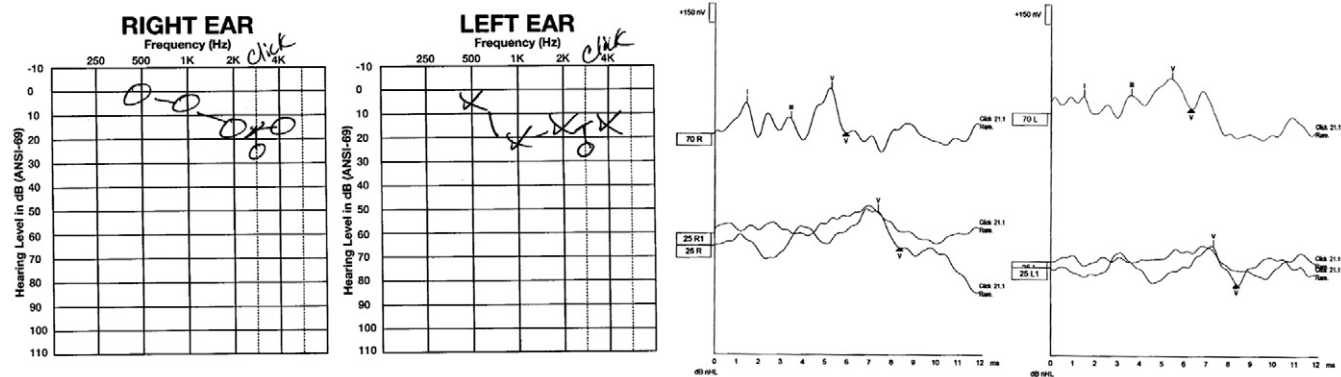


FIG. 4. Audiometric testing of the patient 11 months after SAH. **Left:** Audiogram showing estimated sensitivity thresholds for each ear. **Right:** ABR waveforms of the right (*left*) and left (*right*) ears. Wave peaks are labeled I–V.

trouble finding words. She was able to complete activities of daily living, including cooking, cleaning, bathing, and exercise.

Auditory examination revealed central hearing loss. Weber testing lateralized to the right side and Rinne test showed decreased sound perception, with air conduction on the left side greater than the right side. Otoscope examination and tympanometry were performed at 6 months after SAH and showed clear ear canals, intact tympanic membranes, normal ear canal volume, normal peak pressure, and normal static compliance bilaterally. Otoacoustic emissions testing demonstrated presence of emissions at 1.5 to 11 kHz in the right ear with reduced emissions at 12 kHz only and presence of emissions at 1.5 to 10 kHz in the left ear with reduced emissions at 11 to 12 kHz. Auditory brainstem response (ABR) testing was performed at 6 and 11 months after SAH. ABR demonstrated reliable waveforms with normal absolute latencies, interwave latencies, and interaural latencies. Estimated sensitivity thresholds were within normal limits for all frequencies tested in each ear (Fig. 4; Supplemental Figure 1). Word recognition scores at 60 dB were 0% bilaterally at 6-month follow-up and 32% and 64% in right and left ears, respectively, at 11-month follow-up.

Discussion

Observations

We present a case of central hearing loss after aSAH. Cerebral vasospasm and DCI are common complications of aSAH, occurring in approximately 70% and 30% of cases, respectively;⁷ however, central hearing loss is exceedingly rare. Three case reports have described permanent hearing loss after aSAH; however, these cases involved anterior inferior cerebellar artery aneurysms that disrupted flow through the labyrinthine artery^{8,9} and/or patients with preexisting unilateral temporal lobe infarcts.¹⁰ This case is remarkable because the ischemic injury to the bilateral auditory cortices was due to DCI and vasospasm.

Lessons

The pathology of DCI after aSAH is complex and dynamic. Cerebral vasospasm is one of many factors, including microcirculatory dysfunction, spreading depolarizations, and neuroinflammation, that culminate in cerebral ischemia.¹¹ The patient in this case developed severe, bilateral vasospasm of the ICA, MCA, and ACA. The ischemia in the right auditory cortex could be at least partially explained

by the more extensive hematoma on this side. This can provoke cerebral edema, microthrombosis, and activation of inflammatory cascades, leading to increased risk of vasospasm and subsequent DCI. The ischemia in the left auditory cortex highlights the widespread vasospasm that can occur after SAH.

The rarity of central hearing loss as a manifestation of DCI is partly explained by the intricate neuroanatomy involved. The auditory cortex receives inputs from bilateral cochlear nuclei via the inferior colliculi and medial geniculate nucleus;¹² thus, unilateral auditory cortex injury typically results in slight hearing loss while bilateral lesions result in complete central hearing loss. The patient in this case suffered bilateral ischemic injury to the superior temporal gyri secondary to aSAH and severe vasospasm. Audiometry performed after hospitalization was also suggestive of central hearing loss rather than pathology of the inner ear or vestibulocochlear nerve. In summary, this case offers an important clinical pearl because of its illustration of the complex pathophysiology of cerebral ischemia after aSAH manifesting as the rare complication of central hearing loss.

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Author Contributions

Conception and design: Hoh, Dodd, Laurent, Lucke-Wold. Acquisition of data: Dodd, Laurent, Lucke-Wold. Analysis and interpretation of data: Dodd, Lucke-Wold, Busl, Williams. Drafting the article: Dodd, Lucke-Wold, Williams. Critically revising the article: Hoh, Dodd, Laurent, Lucke-Wold, Busl. Reviewed submitted version of manuscript: Hoh, Dodd, Laurent, Lucke-Wold, Busl. Administrative/technical/material support: Hoh. Study supervision: Hoh.

Supplemental Information

Online-Only Content

Supplemental material is available with the online version of the article.

Supplemental Figure 1. <https://thejns.org/doi/suppl/10.3171/CASE21700>.

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