Reversible cortical auditory dysfunction caused by cerebral vasospasm after ruptured aneurysmal subarachnoid hemorrhage and evaluated by perfusion magnetic resonance imaging

Case report

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✓ A 52-year-old woman developed subarachnoid hemorrhage (SAH) caused by a ruptured right internal carotid artery (ICA) aneurysm. Because of the aneurysm configuration, the authors decided to delay surgery and instead undertook serial imaging studies of the aneurysm. The patient remained alert but developed acute bilateral deafness on Day 7. Audiological examination and auditory brainstem responses suggested that the hearing disturbance was cortical in origin. Three-dimensional computed tomography (CT) angiography showed severe vasospasm in the right middle cerebral artery (MCA) and moderate vasospasm in the left ICA and MCA. Three-tesla magnetic resonance (MR) imaging was performed 2 days after the onset of symptoms. Diffusion-weighted and T2-weighted MR images showed an acute infarction in the right insular cortex caused by vasospasm. Perfusion-weighted MR imaging, particularly mean transit time mapping, revealed hypoperfusion in both temporal lobes including the auditory cortex and right auditory radiation. The vasospasm was treated with induction of mild hypertension and hypervolemia. Follow-up MR images, 3D CT angiograms, and audiometry performed 2 weeks after the first examination showed recovery of vasospasm and resolution of perfusion abnormality and hearing disturbance. On Day 26, the aneurysm was successfully occluded with clips and the patient was discharged with no deficits. To the authors' knowledge, this is the first reported case of reversible cortical auditory dysfunction purely due to bilateral cerebral vasospasm detected using perfusion MR imaging after SAH. (*DOI: 10.3171/JNS-07/07/0161*)

KEY WORDS • auditory agnosia • cortical auditory dysfunction • cortical deafness • perfusion-weighted magnetic resonance imaging • subarachnoid hemorrhage • vasospasm

ORTICAL deafness is a rare symptom that is most often associated with cerebral infarction and is caused by bilateral lesions of the auditory cortex or its radiations.^{6,11} We encountered a case of acute deafness of cortical origin secondary to vasospasm after SAH. We identified the ischemic lesions in both temporal lobes with the aid of PW MR imaging. The patient's symptoms and perfusion abnormality resolved after cessation of vasospasm. To our knowledge this is the first report of reversible cortical auditory dysfunction caused purely by cerebral vasospasm after SAH.

Case Report

Presentation and Examination. This 52-year-old right-

handed woman was admitted to our institution with SAH caused by a ruptured right ICA aneurysm. Computed tomography scans showed diffuse thick SAH with no other lesions such as an old infarction or hemorrhage. Clinically, the SAH was evaluated as Grade I according to the World Federation of Neurological Societies system. Cerebral angiography demonstrated a blisterlike aneurysm located at the distal ICA. It was unrelated to arterial branching, and could therefore be classified as an anterior wall aneurysm.

Imaging Studies and Treatment. Because of the risk of premature rupture during early surgery, delayed surgery was scheduled and the patient underwent serial imaging of the aneurysm. During follow-up, she was alert; however, she developed acute bilateral deafness on Day 7 even though she had no history of hearing impairment. Because of the deafness, verbal communication was difficult. Although spontaneous speech and some recognition of verbal and nonverbal sounds existed, moderate bilateral hearing loss and an auditory agnostic component were present and communication was confined to writing. The patient's ability to read and write and execute written tasks was preserved. The

Abbreviations used in this paper: CT = computed tomography; DW = diffusion-weighted; ICA = internal carotid artery; MCA = middle cerebral artery; MR = magnetic resonance; MTT = mean transit time; PW = perfusion-weighted; rCBV = regional cerebral blood volume; SAH = subarachnoid hemorrhage.

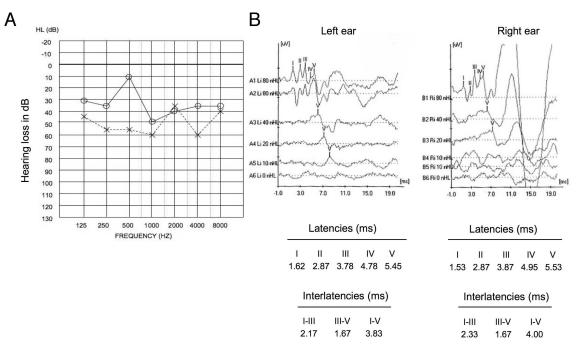


FIG. 1. A: Audiogram obtained at the onset of deafness, showing moderate hearing loss bilaterally. *Solid line* denotes the left ear; *dashed line*, the right ear. B: Tracings of auditory brainstem responses demonstrating a normal pattern of all components from wave I through V in both ears. HL = hearing level; Li = left side; ms = msec; nHL = normal hearing level; Ri = right side.

external auditory meatus, tympanic membrane, and vestibular function were normal. Conservative air conduction audiometry demonstrated moderate hearing loss bilaterally (Fig. 1A). Auditory brainstem responses showed normal waveforms and latencies from wave I to V, indicating intact auditory pathways (Fig. 1B). Therefore, we obtained 3D CT angiograms, 3-tesla PW MR images, and DW images. The 3D CT angiograms demonstrated severe vasospasm in the right MCA and moderate vasospasm in the left MCA, ICA, and basilar artery (Fig. 2A). The DW and T2-weighted images obtained 2 days after the onset of symptoms revealed high signal intensity at the right insular cortex, indicating acute infarction due to vasospasm (Fig. 2B). Measurement of MTT and rCBV showed mild hypoperfusion in both temporal lobes including the auditory cortex (Brodmann areas 22, 41, and 42), the right auditory radiation, and both occipital lobes (Fig. 2C). The right side was more extensively involved than the left. Left-sided areas included part of the Heschl gyrus and the parietal lobe, suggesting that the hearing difficulty originated in the cortex. Vasospasm was treated with induction of mild hypertension and hypervolemia. Follow-up MR images, 3D CT angiograms, and audiometry performed 2 weeks after the first examination showed recovery of vasospasm and resolution of perfusion abnormality and hearing disturbance (Fig. 3). The patient's hearing loss improved gradually and comprehension and identification of verbal sounds as well as environmental sounds and music normalized within 1 month. During 3 weeks of follow-up, the shape and size of the aneurysm changed from blisterlike to enlarged and saccular. On Day 26, the aneurysm was successfully occluded with clips.

Posttreatment Course. The patient's postoperative course was satisfactory and the patient was discharged with no neurological deficits. When the patient was discharged from the hospital, her Wechsler Adult Intelligence Scale-Revised and standard language test of aphasia scores were within the normal ranges.

Discussion

The clinical syndrome of cortical deafness in a woman with bitemporal infarction was described by Wernicke and Friedlander¹¹ in 1883. Cortical deafness and auditory agnosia are usually related to each other and are frequently associated with aphasia.^{2,3} Severe auditory deficit due to bilateral cerebral lesions is relatively rare. This condition is generally known as "cortical deafness" because in most cases damage to both temporal or temporoparietal lobes is observed, including the primary auditory cortex (Broadmann areas 41 and 42) on both transverse gyri (Heschl). The literature contains reports of a number of cases of cortical deafness in children and adults, some congenital and others occurring as a result of cerebral infarction or cerebral hemorrhage.^{1-3,5,6,9-11} In cortical deafness, auditory signals cannot be perceived in the cortex and audiometry reveals severe bilateral hearing loss. Because our case was atypical and demonstrated moderate bilateral hearing loss as well as an auditory agnostic component, we have used the term "cortical auditory dysfunction" instead of cortical deafness. Among the several causes of cortical deafness, there has been only one previously reported case in which SAH precipitated deafness.7 In that case, an old infarction was diagnosed in the left temporal lobe before the occurrence of

Cortical auditory dysfunction due to cerebral vasospasm

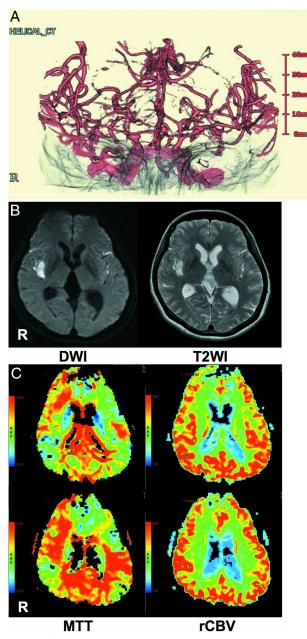


FIG. 2. Neuroimages. A: Three-dimensional CT angiogram obtained at the onset of deafness showing severe vasospasm in the right MCA and moderate vasospasm in the left MCA, ICA, and basilar artery. B: Three-tesla MR imaging performed 2 days after onset of deafness. The DW (DWI) and T2-weighted (T2WI) imaging sequences show high signal intensity at the right insular cortex, indicating acute infarction due to vasospasm. C: Perfusion-weighted MR images showing the MTT delay in both temporal lobes including the auditory cortex, the right auditory radiation, and both occipital lobes. Left-sided areas included part of the Heschl gyrus and the parietal lobe.

SAH; hence, cortical deafness was caused by vasospasm contralateral to this old temporal lobe infarction. Evaluation of cerebral blood flow was not performed in that case. In the present case, it is noteworthy that cortical auditory dysfunction was caused purely by bilateral cerebral vasospasm after SAH; no existing lesions were present.

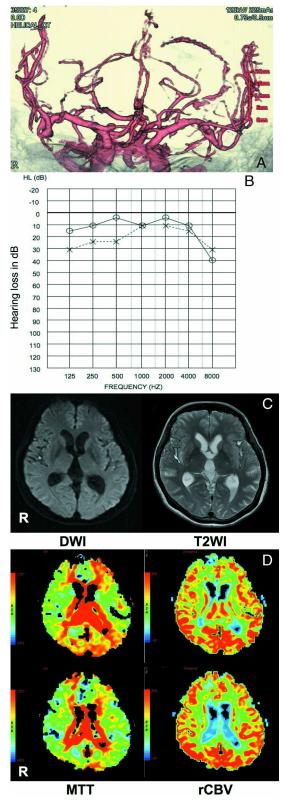


FIG. 3. A: Three-dimensional CT angiogram obtained 2 weeks after the first examination showing recovery of vasospasm. B and D: Resolution of the hearing disturbance is seen on the audiogram (B) and that of the perfusion abnormality is seen on the PW MR images (D). C: Magnetic resonance images showing only a small area of infarction in the right insular cortex.

Magnetic resonance imaging including DW and PW imaging sequences is a very promising technique to assess ischemia and brain damage in SAH. Perfusion-weighted imaging is a useful new tool in the treatment of patients with SAH, particularly those with cerebral vasospasm, and the temporary neurological deficits correlate well with the location of perfusion changes.⁴ In this study, hypoperfused regions corresponding to the responsible foci were demonstrated on PW images. Rordorf et al.⁸ reported that small ischemic lesions on DW images were seen to be encircled by a large area of increased MTT in all patients with symptomatic vasospasm. Our data were consistent with this finding.

To the best of our knowledge, this is the first case in which transient cortical auditory dysfunction caused by vasospasm after SAH has been detected in both temporal lobes on PW MR images. This case suggests that transient ischemia involving the bilateral auditory cortices and auditory radiations can cause reversible cortical auditory dysfunction.

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