

Case Report

Intraarterial vasodilator therapy immediately rescued pure cortical deafness due to bilateral cerebral vasospasm

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Abstract

Background: Cortical deafness is a rare symptom that is associated with bilateral lesions of the auditory cortex. To date, cortical deafness has been reported in only three cases of subarachnoid hemorrhage (SAH).

Case Description: This 55-year-old female was admitted to our hospital with SAH caused by a ruptured left internal carotid artery (ICA) paraclinoid aneurysm. Computed tomography (CT) scans showed diffuse thick SAH with no other lesions such as an old infarction or hemorrhage. Emergent stent-assisted coil embolization was performed successfully and subsequent cisternal irrigation with urokinase almost completely washed out the thick SAH. During follow-up, she was alert and without any neurological deficits, 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. She became almost completely unable to hear and communication was confined to writing. Immediate diffusion-weighted (DW) image showed high intensities in bilateral superior temporal gyri due to severe vasospasm of bilateral middle cerebral arteries (MCAs). Immediate angiography showed severe vasospasm especially right MCA. A microcatheter was advanced to the right M1 and papaverine was administered. Soon after that, her hearing impairment dramatically improved. Our simple audiometry showed a hearing threshold average for both 1000 and 4000 Hz at 25 dB in both ears. She was discharged without any deficits in 2 weeks.

Conclusions: To our knowledge, this is the first reported case of pure cortical deafness due to bilateral vasospasm, which was immediately resolved by intraarterial administration of papaverine.

Key Words: Cerebral vasospasm, Cortical deafness, Intra-arterial papaverine

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INTRODUCTION

Posthemorrhagic cerebral vasospasm (PHCV) is a common problem and a significant cause of mortality and permanent disability following aneurysmal subarachnoid hemorrhage (SAH). While medical therapy remains the

mainstay of prevention against PHCV and the first-line treatment for symptomatic patients, endovascular options should not be delayed in medically refractory cases.^[7] We encountered a rare case of acute deafness of cortical origin secondary to vasospasm after SAH without any other neurological deficits. We identified

the ischemic lesions in both temporal lobes with the aid of perfusion-weighted (PW) magnetic resonance imaging (MRI). Immediate endovascular treatment rescued the hearing impairment. To our knowledge this is the first report of pure cortical auditory dysfunction caused by cerebral vasospasm after SAH, which was resolved immediately with endovascular treatment.

CASE REPORT

Clinical presentation

A 55-year-old right-handed female was admitted to the intensive care unit (ICU) for an SAH caused by the rupture of a left internal carotid artery (ICA) paraclinoid aneurysm [Figure 1]. The patient had no previous medical history, and her hearing was normal before this episode. On admission, her Glasgow Coma Scale (GCS) score was 15/15, corresponding to Grade I according to the World Federation of Neurological Societies grading scale. Under general anesthesia, the left ICA paraclinoid saccular aneurysm was successfully treated by endovascular coil embolization with a stent assist (Neuroform EZ™; Stryker Neurovascular, Fremont, CA) [Figure 1]. Urokinase (12000 units) irrigation from a lumbar drainage on days 1 and 2 cleared the thick SAH [Figure 1] in order to prevent forthcoming vasospasm.

Postoperative course

In order to prevent vasospasm, she was treated with induction of mild hypertension and hypervolemia. Oral administration of cilostazol was also done for the purpose.^[8] She did not show any neurological deficits and MR angiogram did not show any angiographical vasospasm postoperatively. However, she suddenly 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, 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 external auditory meatus, tympanic membrane, and vestibular function were normal. Conservative air conduction audiometry demonstrated severe hearing loss bilaterally. The MR angiograms demonstrated severe vasospasm in the right middle cerebral artery (MCA) and moderate vasospasm in the left MCA [Figure 2]. The diffusion-weighted (DW) images revealed high signal intensity at the right insular cortex and left superior temporal gyrus, indicating acute infarction due to vasospasm [Figure 2]. Compared with conventional perfusion techniques, advantage of arterial spin-labeling (ASL) is the avoidance of intravenous contrast administration.^[4] ASL showed mild hypoperfusion in both temporal lobes including the auditory cortex [Figure 2]. The right side was more extensively involved than the left. Left-sided areas included part of the Heschl gyrus suggesting that the hearing difficulty originated in the cortex.

Endovascular treatment and afterward

Immediate conventional catheter digital subtraction angiography was done and it showed severe vasospasm in the right MCA [Figure 3] and moderate vasospasm in the left MCA. We decided to expand the narrowed MCA chemically. A microcatheter (Tangent™; Stryker Neurovascular, Fremont, CA) through a 6F guiding catheter (Envoy™; Cordis, Miami Lakes, FL) over a 0.014-inch guidewire (Transend EX™; Stryker Neurovascular, Fremont, CA) was advanced to the right M1 [Figure 3]. Papaverine was administered intraarterially directly within the right MCA (2 mg each,

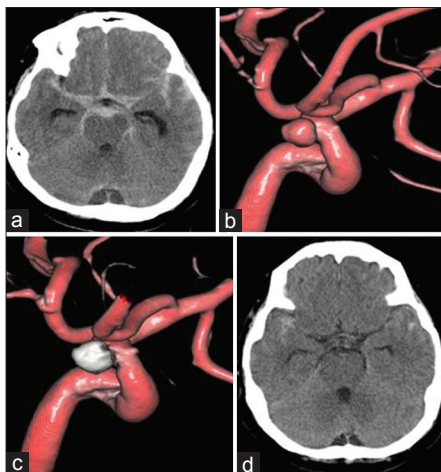


Figure 1: (a) Head CT scan showing diffuse and thick SAH on admission. (b) 3D image of left IC angiography showing a paraclinoid aneurysm. (c) The aneurysm was successfully obliterated with coiling. (d) Head CT scan on day 3 showing most of the SAH was washed out

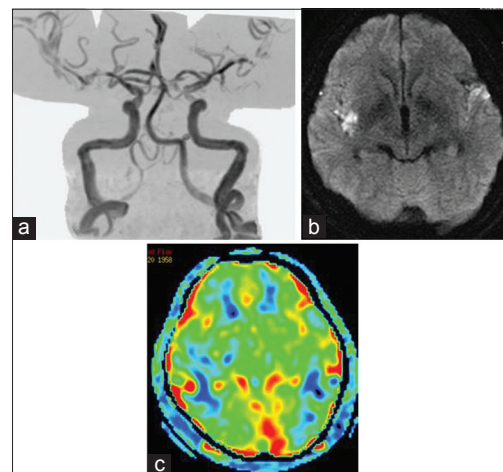


Figure 2: Neuroimages obtained at the onset of deafness. (a) MR angiogram showing severe vasospasm in the right MCA and moderate vasospasm in the left MCA. (b) DW image showing high signal intensity at the right insular cortex and the left superior temporal gyrus, indicating acute infarction due to vasospasm. (c) ASL images show bilateral superior temporal gyri

total 20 mg). This resulted in transient angiographic improvement [Figure 3]. At this time, she was able to hear almost normally. Therefore, we concluded the endovascular treatment and she underwent the conventional audiometry again. She could hear the lowest level of the device bilaterally [Figure 3]. After that, she did not have any deficits and was discharged in a week without hydrocephalus.

DISCUSSION

Cortical deafness and auditory agnosia are usually related to each other and are frequently associated with aphasia.^[1] 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 (Brodmann areas 41 and 42) on both transverse gyri (Heschl).^[2,3]

A number of cases of cortical deafness have been reported in the literature, with the most frequent causes being congenital and cerebral infarction or hemorrhage.^[2] To date, cortical deafness has been reported in only three cases related to SAH.^[5,6,9] In the first reported case, an old infarction was found in the left temporal lobe prior to the occurrence of SAH.^[5] Cortical deafness was caused by vasospasm contralateral to a preexisting temporal lobe infarction. In the second case, transient cortical auditory dysfunction was caused by SAH-related vasospasm of which subsequent parenchymal ischemic damage was depicted in both temporal lobes on DW MRI.^[9] The authors hypothesized that transient ischemia involving bilateral auditory cortices and radiations was the cause of reversible cortical auditory dysfunction. In the case, the hearing impairment was realized on day 7 and gradually improved until one month without any interventional treatment. In the third case, severe auditory dysfunction was apparent from day 10 and intraarterial infusion of nimodipine in the right MCA and angioplasty were not reactive to speech or environmental sounds.^[6] Brain MRI on day 17 demonstrated infarction areas mainly in the right hippocampus, medial occipital lobe, and thalamus. The central auditory dysfunction improved over 6 months, but not fully recovered. In the present case, it is noteworthy that cortical auditory dysfunction was caused purely by bilateral cerebral vasospasm after SAH, which fully recovered immediately after intraarterial papaverine administration.

Although both transluminal balloon angioplasty (TBA) and intraarterial vasodilator therapy (IAVT) can be effective in relieving proximal symptomatic PHCV, only IAVT is a viable treatment option for distal vasospasm. The main advantage of TBA is its long-lasting therapeutic effect and the very low rate of retreatment. However, its use has been associated with a significant risk of serious complications, particularly vessel rupture and reperfusion hemorrhage. Conversely, IAVT is generally considered an effective and low-risk procedure, despite the transient

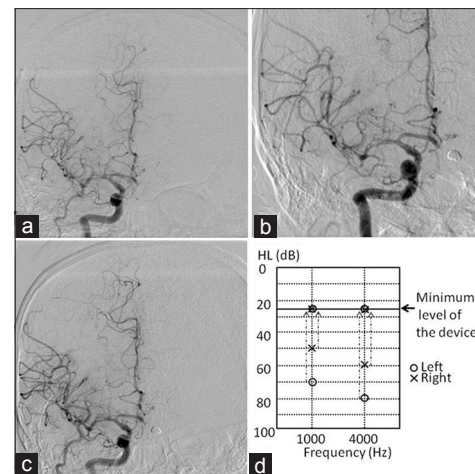


Figure 3: (a) Right ICA angiography showing spasm in the right MCA. (b) A microcatheter was advanced to the right M1 and papaverine was administered from there. (c) The right MCA spasm improved after the papaverine administration. (d) Conservative air conduction audiogram obtained at the onset of deafness, showing severe hearing loss bilaterally. After the endovascular treatment, her hearing ability became normal level bilaterally

nature of its therapeutic effects.^[7] In this case, IAVT for right MCA improved the hearing impairment caused by bilateral temporal ischemia immediately and dramatically, which in turn, proved that the hearing dysfunction was caused by bilateral temporal ischemia. This is an extremely rare case that only cortical deafness was the symptom caused by bilateral MCA vasospasm. Fortunately, the DWI high intensities were reversible but surely she was in a risk of further infarction and symptoms. Therefore, we believe that endovascular options should not be hesitated and delayed.

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