

Short communication

## Bilateral deafness as a prodromal symptom of basilar artery occlusion

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### Abstract

Bilateral deafness is a rare but possible symptom for ischemia of the vertebrobasilar system, primarily derived from occlusion of anterior inferior cerebellar arteries or their branches. Patients 1 and 2 developed sudden bilateral deafness, soon followed by coma. The proximal segment of the basilar artery was occluded due to atherothrombosis in Patient 1 and arterial dissection in Patient 2. Thrombolytic therapy failed to recanalize the basilar arterial flow. Both patients died of extensive infarction in the vertebrobasilar arterial territory. Sudden bilateral deafness can be a warning sign of imminent brainstem ischemia by occlusion of the basilar artery regardless of age. Prompt and intensive management for stroke is needed for patients with sudden bilateral deafness. © 2002 Elsevier Science B.V. All rights reserved.

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### 1. Introduction

Basilar artery occlusion often ends in death or major disability, including tetraplegia and coma [1–3], although a very recent review stresses low possibility of such miserable outcomes (7.7%) [4]. Patients with basilar artery occlusion often have minor stroke or transient ischemic attacks preceding fatal stroke [2,5,6]. The most common prodromal symptoms are motor and oculomotor deficits, with vertigo, nausea and headache occurring less frequently [2,5,6]. Hearing loss or tinnitus is not a major prodromal symptom (6% or less), and usually appears to develop with other symptoms [2,5,6]. Thus, it may be difficult to consider sudden deafness alone as a prodromal symptom of basilar artery occlusion.

Because basilar artery occlusion is potentially treatable, for example by thrombolysis [7], it is essential not to overlook any prodromal symptoms of this disease. Bilateral deafness seems to be a rare but one of possible prodromal symptoms. Here, we describe two patients with basilar artery occlusion who developed sudden bilateral deafness, soon followed by coma.

### 2. Case report

*Patient 1:* A 51-year-old barber had histories of hypertension, diabetes, and hypercholesterolemia, and a family history of ischemic stroke that his father and brother developed at the age of 42. He did not notice the alarm clock one morning. Right after being woken up by his wife, he barely heard her speaking, and soon afterward, he could not hear at all. He felt dizziness while walking.

On arrival at our hospital an hour after waking up, his blood pressure was 145/80 mm Hg, his pulse rate was 59 bpm and regular, and his respiration was normal. He did not speak or respond to verbal orders or nonverbal sounds. He wrote that he had tinnitus of both ears. He responded to written orders. Neurological examination was normal except for complete deafness. Cranial CT was normal. We started infusion of a volume expander in case of vertebrobasilar insufficiency. Forty minutes later, he suddenly fell into coma and tetraplegia. His eyes were deviated rightward, and then fixed in the midline. Oculocephalic maneuvers elicited vertical, but not horizontal, eye movements. Blood tests showed severe hyperglycemia and elevated levels of thrombin anti-thrombin III complex and D-dimer, indicating hypercoagulative state. Echocardiography or long-time electrocardiography did not reveal any cardiac source of emboli.

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On digital subtraction angiography (DSA, Fig. 1A–C), the left vertebral artery was open proximal to, and occluded distal to the origin of the left posterior inferior cerebellar artery (PICA). The basilar artery barely appeared via the collateral of left PICA and left anterior inferior cerebellar artery (AICA). We advanced the tip of a microcatheter through the thrombus of the basilar artery, and infused urokinase 3 h after the onset of coma. DSA revealed the distal basilar artery with superior cerebellar arteries (SCAs) and posterior cerebral arteries (PCAs). Then, we pulled the microcatheter back and infused urokinase into the thrombus. The mid-basilar artery with AICAs appeared, although the distal basilar artery was not detected. Pupils began to dilate when a total of 300,000 U of urokinase was infused, and we quit thrombolysis. Pupil size returned to normal soon afterwards, but any other signs did not improve.

On the second hospital day, his respiration became shallow, and mechanical ventilation was started. Oculocephalic maneuvers no longer elicited vertical eye movements. CT revealed extensive fresh infarcts in both cerebellar hemispheres fed by SCAs and AICAs, whole pons, tegmentum of left midbrain, left thalamus, and left posterior lobe. On the 9th day, the patient died of heart and respiratory failure.

**Patient 2:** A 30-year-old raftsmen without risk factors for atherosclerosis felt headache and right tinnitus, and soon suffered bilateral deafness when he just started to work one morning. An ENT doctor diagnosed his state as idiopathic deafness. Five hours later, his right eye was suddenly fixed in the midline, and his left eye could only abduct. Soon afterwards, he went into coma.

On arrival at our hospital 8 h after the onset of deafness, his blood pressure was 129/66 mm Hg, and his pulse rate was 90 bpm and regular. His respiration was shallow, and he needed an intubation. He did not speak, and did not respond to any sounds. He did not open eyes to any stimuli, and his eyes were bobbing conjugately. He had tetraplegia and hyperreflexia.

The left vertebral artery had double lumen with a floating intimal flap between the level of C4 and C5 on B-mode echography (Fig. 2A), and no flow signals in the false lumen on pulsed-wave Doppler techniques. Cranial MRA did not demonstrate the basilar artery. MRI revealed double lumen in the basilar artery without any infarcts (Fig. 2B, T2-weighted image). We diagnosed that left extracranial vertebral dissection extended intracranially resulting in the basilar occlusion, and immediately infused heparin and 24,000,000 U of tissue plasminogen activator intravenously. On DSA 1

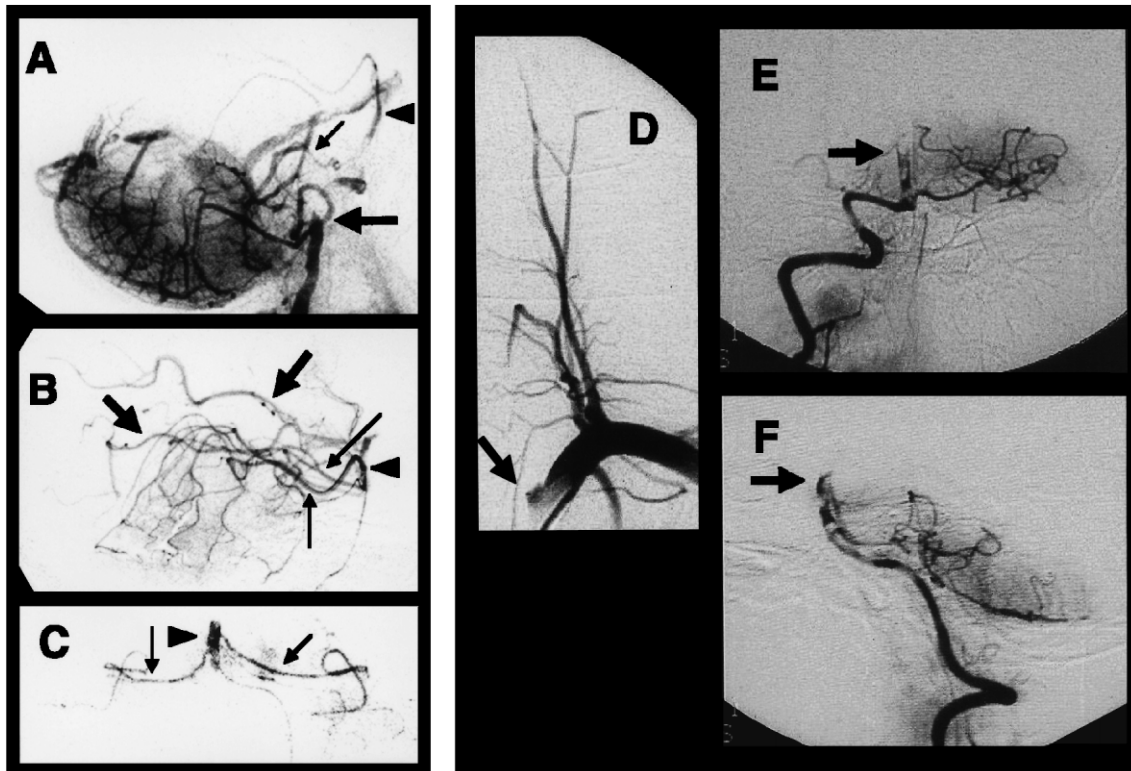


Fig. 1. DSA of Patient 1 (A and B, lateral view; C, frontal view) and Patient 2 (D and E, frontal view; F, lateral view) on the day of admission. (A) The left vertebral artery was occluded (wide arrow) distal to the origin of PICA. The basilar artery (arrowhead) barely appeared via left PICA and left AICA (narrow arrow). (B) After injection of urokinase at the far side of the thrombus, the distal portion of basilar artery (arrowhead), PCAs (wide arrows), SCAs (narrow arrows), and perforator arteries from the basilar top were detected. (C) After injection of urokinase into the thrombus, the middle portion of the basilar artery (arrowhead) and AICAs (narrow arrows) turned to appear. (D) The left vertebral artery was occluded in the origin (arrow). (E, F) The basilar artery had irregular narrowing and occlusion at the proximal portion (arrow).

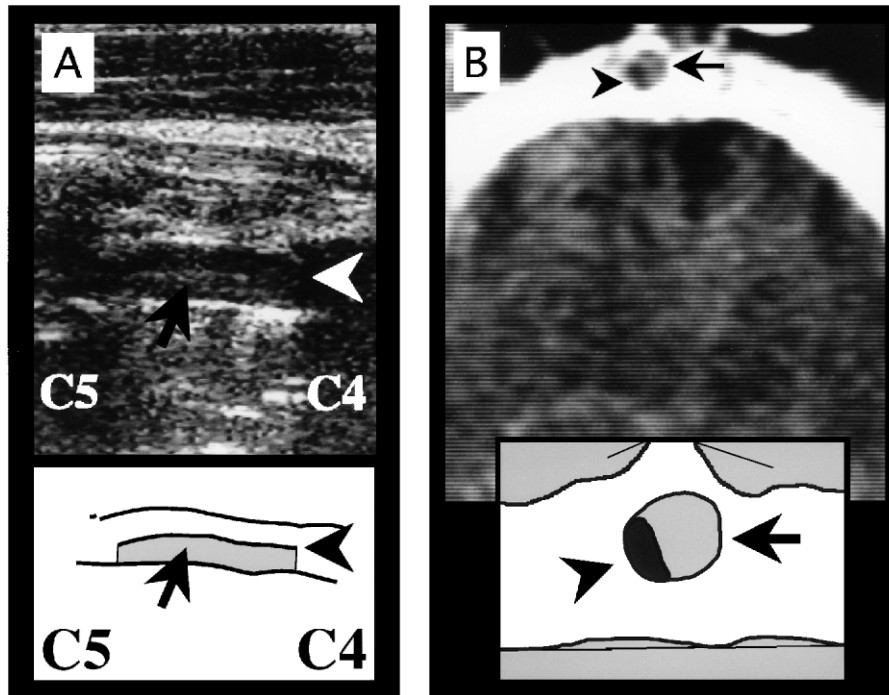


Fig. 2. B-mode echography (A) and MRI (T2-weighted image, B) of Patient 2 on the day of admission. (A) The left vertebral artery had double lumen (arrow shows false lumen) with a floating intimal flap (arrowhead). (B) The left vertebral artery had true (arrowhead) and false (arrow) lumens.

h after thrombolysis (Fig. 1D–F), the left vertebral artery was occluded in the origin. The basilar artery had irregular narrowing and was occluded at the proximal portion. Carotid arteriograms revealed PCAs via posterior communicating arteries. Blood chemistry and cardiac investigations were normal.

Soon after the thrombolysis, he opened his eyes to pain stimuli and ceased ocular bobbing. On the second hospital day, however, he fell into coma again. CT revealed fresh infarcts in whole pons and right cerebellar hemisphere fed by SCAs and AICAs. On the 5th day, the patient died of respiratory failure.

### 3. Discussion

An important point of this report is that sudden bilateral deafness can precede fatal neurological deficits by basilar artery occlusion. Occlusion of the basilar arterial origin suggests that thrombosis, but not embolism, is the primary mechanism of the stroke [4,6]. In addition, Patient 1 had risk factors for atherosclerosis and a family history of young-onset stroke. Dissection is another possible mechanism for occlusion of the basilar origin, although much rarer compared to thrombosis [8]. Double lumen and intimal flap of the left vertebral artery in Patient 2 indicate that vertebral dissection was the cause of basilar occlusion, probably because of an intramural hematoma extending around the orifice of AICAs. Vertebrobasilar dissection has already been reported as a cause of unilateral, but not bilateral, hearing

loss in aged patients [9]. The present report indicates that we should not neglect the possibility of stroke as a cause of bilateral deafness even for young patients free from atherosclerosis. Headache may be a good sign for arterial dissection.

The mechanism of sudden bilateral deafness is usually psychogenic or idiopathic. Deafness of vascular etiology usually occurs unilaterally, associated with other neurological deficits [10]. In a previous study [11], only 7 out of 503 patients (1.4%) with vertebrobasilar territorial infarction had bilateral hearing impairment. The internal auditory artery usually originates from AICA [12], and supplies the membranous labyrinth and the auditory nerves in the internal auditory canal. Among 49 patients of unilateral or bilateral AICA territory infarcts documented by MRI or autopsy in recent studies [13–16], 15 patients (31%) suffered from hearing loss, either unilaterally or unstated on laterality. In the present patients, bilateral AICA territory is thought to be a terminal zone for retrograde collateral flow from the circle of Willis after the occlusion of the basilar origin. In addition, the inner ear artery is an end artery with minimal collaterals, and the inner ear needs high energy [12]. This is a probable explanation for bilateral deafness as a prodromal symptom of the basilar artery occlusion. Deafness as AICA syndrome could be linked to the direct involvement of the cochlear nuclei at the pontomedullary junction [13]. Deafness in isolation, however, seems to be unlikely in such a lesion.

SCA territory infarction also causes hearing loss, which is usually contralateral due to involvement of the crossed

auditory fibers of the lateral lemniscus [17]. Because the lateral lemniscus also has uncrossed fibers, bilateral hearing loss may develop by unilateral SCA territory infarction. Such a symptom is not complete but partial deafness [18]. Occlusion of both SCAs occurs together with the basilar apex, most often by emboli [4], and hearing loss develops together with the so-called top of the basilar syndrome. Thus, bilateral deafness in isolation does not seem to result from ischemia in SCA territory. Severe cortical deafness can be caused by bitemporal cerebrovascular accidents, which are usually combinations of fresh and old lesions, and sometimes bitemporal fresh infarcts probably due to cardioembolism [19,20]. The present patients did not have lesions around auditory cortices.

Thrombolysis is a possible therapeutic strategy for recanalization of the basilar artery and improvement of neurological deficits, although often unsuccessful [7]. The therapy may be available only in the hyperacute stage of stroke. Thus, it is essential to detect basilar artery occlusion while patients have only minor symptoms before they fell into a fatal state, and to start treatment immediately to prevent vertebrobasilar circulation from ischemia. For those patients with sudden bilateral deafness, we should at least keep them lying, start drip intravenous infusion, and restrain usage of antihypertensive drugs before excluding stroke.

In conclusion, this is the first report to demonstrate that sudden bilateral deafness can be a warning sign of imminent brainstem ischemia by occlusion of the basilar artery regardless of age and history of risk factors. Prompt and intensive management for stroke is needed for such patients with sudden deafness.

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