

# Sudden bilateral deafness revealing fatal basilar artery occlusion

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## Introduction

Sudden bilateral deafness (SBD) can be a prodromal symptom of basilar artery occlusion [1]. We aim to highlight this unusual clinical presentation.

## Case-report

A 65-year-old man with a previous history of hypertension and hypercholesterolemia developed SBD, tinnitus, vertigo and vomiting, followed a few hours later by unsteadiness with a leftward bias. On admission, vital parameters were normal. Neurological examination revealed a severe bilateral hearing loss confirmed by tonal audiometry. He was otherwise alert and could understand written orders. There was no facial palsy, ophthalmoplegia, muscle weakness, dysmetria, or limb ataxia.

Brain MRI performed 11 h after onset revealed several infracentimetric acute ischemic lesions in the left cerebellum and the left occipital lobe (Fig. 1a, b). MR angiography

(TOF) showed basilar and left posterior cerebral arteries occlusion (Fig. 1c, d). An arteriography performed 14 h after onset confirmed the occlusion of the left vertebral and basilar arteries (Fig. 2). At that time, the patient became drowsy, dysarthric, with a downward and right deviation of gaze. A local intra-arterial thrombolysis (Cantata<sup>®</sup> superselective microcatheter) was initiated with a bolus of 1,000 U of Urokinase followed by an infusion of 500,000 U during the first hour and 250,000 U during the second hour without recanalization.

The clinical condition of the patient progressively worsened during the following hours. He became comatose and developed bilateral areactive mydriasis and loss of brainstem reflexes. Cerebral CT-scanner revealed massive infra-tentorial oedema and acute hydrocephalus. Clinical and neurophysiological testings confirmed brain death 2 days after admission.

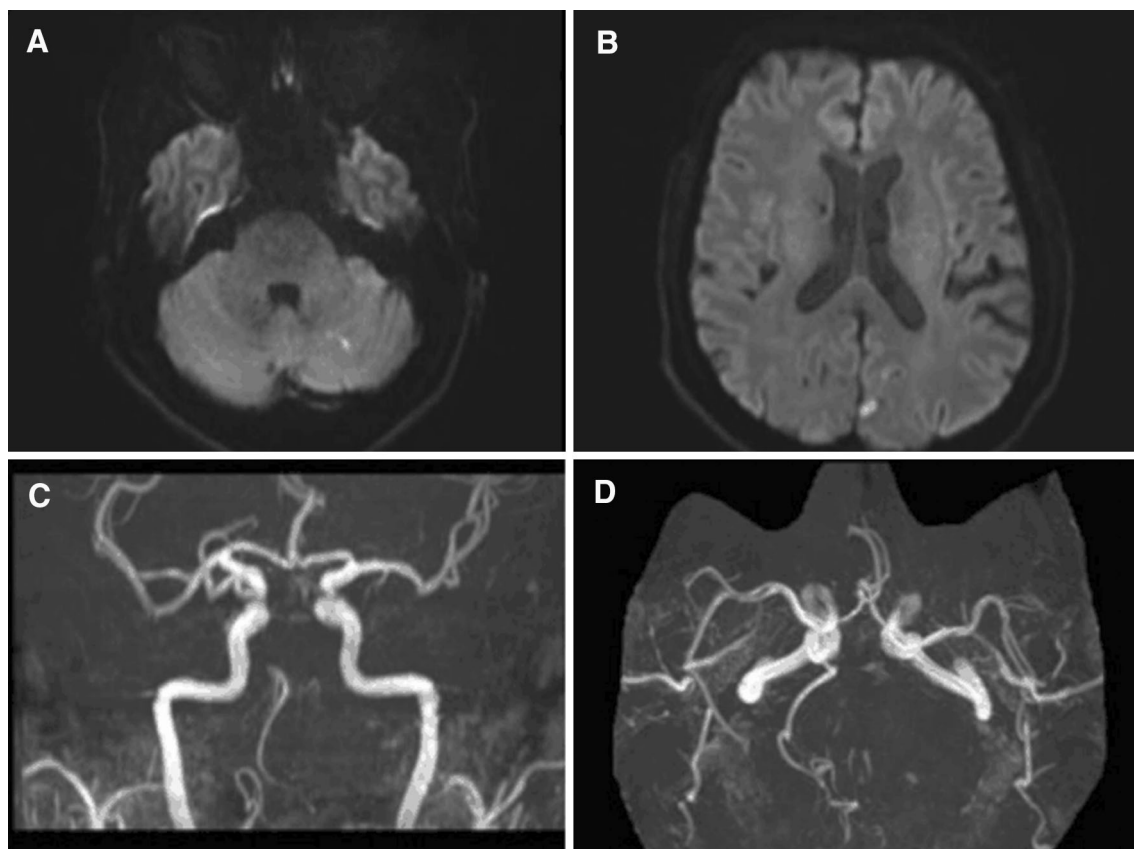
## Discussion

Sudden bilateral deafness (SBD) due to vertebrobasilar occlusive disease (VOD) is rare. In a retrospective analysis of 333 patients admitted for sudden sensorineural hearing loss, VOD was diagnosed as the unique aetiology in four cases (1.2 %) [2]. Basilar artery occlusion related strokes were preceded by prodromal symptoms in more than 50 % of cases, mostly vertigo, nausea and headaches but rarely by hearing loss and tinnitus (5 patients among 85 cases, 6 %) [3]. Among 503 patients with VOD, 7 (1.4 %) had sudden bilateral hearing impairment, tinnitus and vertigo. However, three of them had other symptoms of brainstem involvement at onset [4]. VOD was related to atherothrombosis of the vertebral or the basilar arteries in most cases but vertebral artery dissection has also been reported [1, 2].

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**Fig. 1** Brain MRI. **a, b** DWI-weighted images showing acute ischemic lesions in left cerebellum and the left occipital lobe. **c, d** MR angiography showing absent blood flow in the basilar artery and left posterior cerebral artery



**Fig. 2** Conventional angiography showing basilar artery occlusion

As stroke is the cause of sudden deafness only in a minority of patients, it is important to select those who require urgent work-up. According to recent guidelines, a sudden onset of bilateral hearing loss is rare [5]. A bilateral onset should thus raise concern for specific causes including VOD, as should the following red flags: concomitant or preceding focal neurological symptoms or signs, vertigo and severe vestibular impairment.

Deafness is consecutive to ischemia of the cochlea in most cases but may also result from infarction of the cochlear nucleus or auditory pathway in the pons [4]. The inner ear is supplied by the internal auditory artery, which usually originates from the anterior inferior cerebellar artery. The outcome of patients with SBD due to VOD is variable. In some of them, coma and death occur rapidly after deafness as in our case [1]. Some patients remain in locked-in state while others recover without neurological deficits [4]. In case of basilar artery occlusion, an early recanalization significantly improves the prognosis but the best treatment strategy needs to be ascertained. Intravenous thrombolysis (IVT) should be initiated without delay to all

patients who are in the appropriate time window. Intra-arterial thrombolysis should be considered if the patient does not respond to IVT or if the delay from stroke onset exceeds the time window.

In conclusion, SBD is rare but should raise the hypothesis of vertebrobasilar stroke and warrants urgent neurological examination. The recognition of a cerebrovascular aetiology is important to provide the best acute treatment and an optimal secondary prevention.

**Conflict of interest** The authors declare no conflict of interest.

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