Delayed traumatic intracerebral hematoma presenting as cortical deafness: case report

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ABSTRACT
Cortical deafness is a rare condition and is usually caused by bilateral cerebral lesions. Several cases of sudden cortical deafness caused by ischemic stroke or hypertensive intracerebral hematoma have been reported, however, no cases of traumatic cortical deafness were identified in our literature search.

We experienced a case of delayed traumatic intracerebral hematoma (DTICH) presenting as cortical deafness. In our patient, warfarin intake seemed to be a risk factor for DTICH. Head injury may adversely affect the coagulability and thrombolysis. A high risk of DTICH after traumatic head injury should be kept in mind when encountering patients taking anticoagulants.

1. Introduction
The bilateral cerebral cortex is involved in bilateral hearing perception, so unilateral cerebral lesions usually are not accompanied by cortical deafness. For this reason, cortical deafness is a rare condition and is usually caused by bilateral cerebral lesions. Several cases of sudden cortical deafness caused by ischemic stroke or hypertensive intracerebral hematoma have been reported in English [1, 2, 3, 4, 5]. However, no cases of traumatic cortical deafness were identified in our literature search.

We experienced a case of delayed traumatic intracerebral hematoma (DTICH) presenting as cortical deafness.

2. Case report
A 60-year-old man had hypertension and atrial fibrillation and was taking antihypertensive drugs and warfarin for his condition. One day, he fell on a steeply sloped road and hit his head in the occipital region. He immediately visited a local hospital for the therapy of scalp bruise, where brain computed tomography (CT) was performed. He had no symptoms, and no abnormalities were noted on CT (Figure 1). He therefore returned home without any further examinations. However, the next day, he found he could not understand others’ speech. He therefore visited our hospital complaining of “bilateral hearing loss”. He was alert and could speak fluently, but he could not understand any phonetic instructions. He was still able to understand written words and could also write words himself. He showed no paresis and was able to walk without any support. Mild headache and nausea were reported. Brain CT revealed bilateral temporal subcortical intracerebral hematoma and small subdural hematoma (Figure 2). Skull fracture or ear abnormality were not clinically and radiologically observed. Blood coagulation tests showed International normalization ratio 1.51 and prothrombin time 18.5 s. So we considered the effect of warfarin was mild, so although warfarin intake was stopped, reverse with vitamin K or Prothrombin complex concentrate was not administrated.

Brain magnetic resonance image (MRI) on the 6th day after head injury showed bilateral cerebral contusion and hematoma at middle and inferior temporal gyrus (Figure 3, left). Tc99m-ethylcysteinate dimer single-photon emission CT showed bilateral temporal low perfusion (Figure 4). We therefore diagnosed him with cortical deafness caused by DTICH.

We stopped his warfarin intake and treated this patient conservatively with physical and speech rehabilitation. His cortical deafness gradually improved and he became to understand phonetic instructions. Follow-up brain CT showed that his bilateral hematoma had gradually

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resolved (not shown). The auditory brain stem response (ABR) at one month after the injury showed a normal response. He returned to home after hospitalization for one month. The consent was gathered from this patient.

3. Discussion

Cortical blindness is not rare, as the bilateral occipital lobes are located near each other and supplied by the common basilar artery.
Cortical deafness, by contrast, is a rare condition. There have been several reported cases of cortical deafness following cerebral infarction or ICH [1, 2, 3, 4, 5]. In our literature search, however, we found no reports of cortical deafness after traumatic head injury, especially DTICH.

In head injury, cerebral contusion mainly occurs at the frontal and temporal base. In our patient, contusion and hematoma were found bilateral inferior and middle temporal gyrus. This hematoma location may explain good prognosis of cortical deafness in our patient.

Selective injury at the bilateral superior temporal gyrus is rare. DTICH is not limited to the skull base. In our patient, warfarin intake seemed to be a risk factor for DTICH. Head injury may adversely affect the coagulability and thrombolysis. A high risk of DTICH after traumatic head injury should be kept in mind when encountering patients taking anticoagulants. In such cases, anticoagulants must be stopped and reverse with vitamin K or Prothrombin complex concentrate must be considered.

**Declarations**

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**Figure 4.** Tc99m-ethylcysteinate dimer single-photon emission CT showed bitemporal low perfusion.