A case of jugular bulb diverticulum causing pulsatile tinnitus

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Abstract
If you suspect pulsatile tinnitus from a medical interview, you should check for jugular bulb diverticulum and cortical bone defects on temporal bone CT, in addition to thorough physical examination and contrast-enhanced imaging.

Keywords
high jugular bulb, jugular bulb diverticulum, pulsatile tinnitus

1 | INTRODUCTION

Anatomical anomalies of the jugular bulb include a high jugular bulb and deficiency of the cortical bone. Most cases are asymptomatic; however, they may present with tinnitus, hearing loss, and dizziness. We encountered a rare case of jugular bulb diverticulum with the deficiency of the cortical bone that caused pulsatile tinnitus.

The jugular bulb is the vein structure of the posterior fossa that is formed when the sigmoid sinus shifts to the internal jugular vein of the jugular foramen. It is generally separated from the tympanic cavity and mastoid cavity by the cortical bone. Anatomical anomalies of the jugular bulb include a high jugular bulb (HJB), which exists in the middle ear cavity across the bottom end of the tympanic ring, and a dehiscent high jugular bulb (DHJB), which is deficient in the cortical bone. The prevalence of HJB is 6%, and there are more cases of HJB and DHJB on the right side than on the left. It is often asymptomatic; however, it can sometimes cause hearing loss, pulsatile tinnitus, dizziness, vertigo, and repeated facial paralysis. During surgery of the middle ear and lateral skull base, it is critical to confirm an HJB preoperatively. Moreover, the bleeding must be assessed when the jugular vein is damaged.

An HJB is common; however, a jugular bulb diverticulum is rare. The projection direction of the jugular diverticulum varies, including the middle ear cavity, mastoid cavity, and inside apex of the petrous bone.

Tinnitus is a phenomenon that produces sound perception even when there is no external sound. In most cases, the sound is subjective tinnitus, which does not exist in the body, but we occasionally encounter objective tinnitus, which is partly present in the body. Objective tinnitus is often due to muscular spasms or pulsation of blood vessels; however, there are few reports of pulsatile tinnitus. We encountered a case of jugular bulb diverticulum that caused pulsatile tinnitus.

2 | CASE HISTORY

A 30-year-old man presented to our hospital with a chief complaint of intermittent right tinnitus persisting for 2 months. His medical history included hypertension, dyslipidemia, and fatty liver disease. Tinnitus was vascular pulsatile with a rushing sound; it began suddenly and showed improvement in the cervical position. In addition, tinnitus was exacerbated on the day when the patient forgot to take his antihypertensive medications.
The eardrum was normal, and a carotid run abnormality or HJB was not suspected on eardrum examination. Although the patient had slight right vascular pulsatile tinnitus during the hospital visit, we could not detect objective tinnitus using an otoscope. The tinnitus disappeared when the internal jugular vein was pressed from the cervical skin. The audiometric test results were normal, and objective tinnitus was suspected based on the anatomic abnormalities in the temporal bone, including angiectopia, after interviewing the patient.

Jugular vein diverticulum was detected on the computed tomography (CT) scan of the cephalad side, and the deficiency of the cortical bone was detected in the mastoid sidewall of the diverticulum (Figure 1). Neoplastic lesions, such as glomus tumors, were absent on enhanced magnetic resonance imaging (MRI). In addition, there was no evidence of cerebral aneurysm, cerebral arteriovenous malformation, or brain arteriovenous fistulas on MR angiography (Figure 2). In MR venography (MRV), there was obstruction of the left transverse sinus, and hypoplasia was found from the left sigmoid sinus to the internal jugular vein (Figure 3). The occipital and marginal sinus developed, and it flowed into the right sigmoid sinus; consequently, the right jugular vein had significantly more return in quantity (Figure 4).

We suggested to the patients about surgical and endovascular treatment; however, he did not hope it and became the policy of the follow-up.

FIGURE 1 In the temporal bone computed tomography (CT), the jugular bulb diverticulum that goes above is present on the right side, and the cortical bone is deficient on the sigmoid side; however, extension to the tympanic cavity is not detected. In addition, a shadow suspected as a glomus tumor is absent.

FIGURE 2 Cerebral aneurysm, cerebral arteriovenous malformation, and brain arteriovenous fistulas are not found in the magnetic resonance angiography (MRA).

FIGURE 3 In magnetic resonance venography (MRV), (A) the right transverse sinus, sigmoid sinus, jugular bulb, and internal jugular vein are depicted; on the contrary, (B) the left side is not depicted. The venous return has clear laterality.

FIGURE 4 (A) In magnetic resonance venography (MRV), the right occipital and marginal sinuses are developed and flow into the right jugular bulb. (B) The right jugular bulb was considered to have many venous returns in quantity.
3 | DISCUSSION

The main causes of objective tinnitus are the muscle and blood vessels. The muscular mechanism includes activities of the tensor veli palatini, levator veli palatini, tensor tympani,\(^6\) stapedius, salpingopharyngeus, and superior constrictor muscle of the pharynx. The vascular mechanism is divided into arterial and phleboid tinnitus.\(^7\) The causes of arterial tinnitus include abnormalities of the internal carotid artery, carotid hardening-related lesions, aneurysms, arteriovenous malformations, and dural arteriovenous fistulas.\(^8\) On the contrary, the causes of phleboid tinnitus include HJBs, glomus tumors, and middle ear tumors.\(^9\)

It is comparatively simple to determine whether the causes of objective tinnitus are myogenic or vascular by conducting interviews and examining the properties of the sound. It is said that muscular tinnitus is often heard intermittently with clicks, and may also be heard at mastication or in a certain mandibular position. Moreover, there may be consistent movements with tinnitus in soft palates, peri-ear muscles, and tympanic membranes. Not surprisingly, vascular tinnitus often coincides with the pulse. If the causes are suspected to be vascular, it should be confirmed whether there are changes in the tinnitus on pressing the internal jugular vein from the neck skin to judge arterial or phleboid tinnitus. The anatomical anomaly should be examined using temporal bone CT in all patients with pulsatile tinnitus. Moreover, when tinnitus is suspected from the vasculature, MRA or MRV should also be performed.

The jugular bulb is located postlateral to the jugular foramen and receives drainage from the intracranial and extracranial parts. The sigmoid and inferior petrosal sinuses flow into the jugular bulb and form the internal jugular vein.\(^10\) Anatomic abnormalities of the jugular bulb include an HJB, deficiency of the cortical bone, and jugular bulb diverticulum; however, they are often asymptomatic. Nevertheless, it has been reported that an HJB and deficiency of the cortical bone cause vascular tinnitus,\(^11\) sensorineural hearing loss, facial paralysis, vertigo, and dizziness. Moreover, they can cause Meniere’s disease and endolymphatic hydrops.\(^12\)

In this case, it was difficult to prove that the cause of vascular tinnitus was the turbulence of flow in the jugular bulb diverticulum. However, it was more likely to be vascular tinnitus, in addition to phleboid tinnitus, because we confirmed that tinnitus is improved by pressing the internal jugular vein from the neck and changing the cervical position. Moreover, tinnitus may occur owing to jugular bulb diverticulum when the deficiency of the cortical bone is considered, along with the right venous return being completely dominant in MRV, and multiple blood vessels flowing to the jugular bulb; there are no neoplastic lesions reported previously.\(^13\)

Although the jugular diverticulum was present previously, the cause of its sudden occurrence at 30 years of age may be attributed to the change in the blood flow of the venation. There is no contradiction that regression of the blood flow, such as the left transverse sinuses, increased the right blood flow, causing turbulence in the jugular bulb. The D-dimer in consideration of thrombus was within the normal range. This patient had a past medical history of juvenile hypertension, and it was associated with phleboid tinnitus according to the previous report.\(^1\) As tinnitus was aggravated when the antihypertensive medications were not taken, hypertension can be considered one of the factors of sudden onset.

Although surgical therapy and endovascular treatment were recommended for this patient, he did not agree to the treatment as he was aware that the tinnitus was because of the jugular bulb and there were no aneurysms or tumors.

There is no established treatment for pulsatile tinnitus caused by jugular bulb anatomical anomaly. It has been reported that improvement can be achieved by placing bone wax in the cortical bone deficiency by surgery.\(^14\) Another study reported that improvement of the vascular tinnitus was detected after endovascular treatment.\(^15\) However, surgery and endovascular treatment cannot be considered as definitive treatments. The past report revealed that surgery should be considered in patients with severe tinnitus impairing everyday life, persistent vertigo, and progressive sensorineural hearing loss.\(^16\)

4 | CONCLUSION

We reported a rare case of jugular bulb diverticulum that caused pulsatile tinnitus. The jugular diverticulum and cortical bone deficiency must be confirmed using temporal bone CT when pulsatile tinnitus is suspected from an interview. In this case, it was assumed that the right venous return based on MRV was associated with the tinnitus.

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CONFLICT OF INTEREST

None declared.
AUTHOR CONTRIBUTION
IF drafted this article. KY, SY, and TI revised this article. YM and YS collected clinical information. TH gave final approval of this article.

ETHICAL APPROVAL
Written informed consent was obtained from the patient in the Japanese language to publish his clinical details.

DATA AVAILABILITY STATEMENT
The data that support the findings of this study are available from the corresponding author upon reasonable request.

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