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Clinico-radiologic discordance: A case of superior semicircular canal dehiscence by superior petrosal sinus

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Abstract

The audio-vestibular symptoms caused by the partial absence of the bony structure surrounding the superior semicircular canal (SCC) are known as superior canal dehiscence syndrome (SCDS). The dehiscence region can be seen in high-resolution computed tomography (HRCT). Dehiscence is often seen at the arcuate eminence level in the apical region of the SCC. The superior petrosal sinus may rarely course in the vicinity of the medial wall of the SCC and can even cause SCDS. The vascular origin of the dehiscence cannot be exactly determined in routine HRCT without contrast agent administration. In the literature, the use of contrast-enhanced magnetic resonance imaging (MRI) has been reported in a small number of cases to demonstrate this pathology. There may be a relationship between the degree of dehiscence demonstrated by MRI and the patient's symptoms. Here, we present a case that is thought to be superior petrosal sinus dehiscence to SCC using HRCT. Contrast-enhanced arterial and venous phase 3D T1-weighted MRI was performed for the confirmation of the diagnosis, but there was no good correlation between the degree of radiological dehiscence and symptoms in contrast to the previous literature.

Keywords: superior semicircular canal, dehiscence, superior petrosal sinus, computed tomography, magnetic resonance imaging

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The authors stated that the written consent was obtained from the patient presented with images in the study.

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Introduction

Hearing loss may occur due to defects in different parts of the bony roof surrounding the inner ear structures in the spectrum of otic capsule dehiscence or in the third window anomaly [1]. Of these, SCC dehiscence has been reported as 2.1%-10.7% in temporal bone CT examinations [2]. A subtype of SCC dehiscence is superior petrosal sinus (SPS) dehiscence reported in 4%-9% of patients with symptomatic SCC dehiscence [3]. The cause of SCC dehiscence is unclear. Dehiscence might occur during fetal development, but trauma, infection, and/or malignancies could also trigger the emergence of clinical findings in some patients [4]. These patients typically have conductive hearing loss, tinnitus, autophony, and pressure-induced vertigo [5]. In audiometric examinations, low-frequency air-bone gap and increased bone conduction can be detected due to decreased air conduction [2]. A few surgical treatments have been reported in cases of dehiscence of SPS to SCC [4,6-7]. Less invasive interventional methods such as endovascular stenting treatment have recently been applied to SPS in these cases [3]. We only notice the groove-shaped impression of the SPS in the adjacent petrosal bone with HRCT, but it is not possible to visualize the SPS itself. A few cases have been reported in the literature in which contrast-enhanced MRI was performed to reveal SPS [3,5]. Here, we present the incidental findings of a patient who had SCC dehiscence of SPS in the HRCT examination but did not have obvious symptoms. We further present the 3D contrast-enhanced T1-weighted (W) MRI findings that were performed to confirm these findings.

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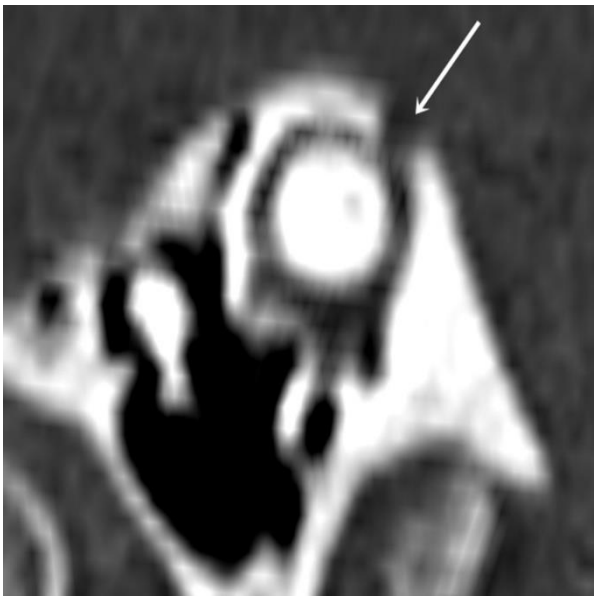
Case presentation

A 38-year-old female patient was admitted to our hospital with complaints of headache, nasal congestion, and decreased sense of smell. No findings were found except postnasal discharge during the physical examination; the patient had no complaints of hearing or balance. In the paranasal sinus HRCT examination, we incidentally evaluated that the left SPS-caused dehiscence in the SCC (Figure 1) in addition to the chronic sinusitis findings. When the patient was questioned again in terms of SHC dehiscence, she reported some symptoms such as mildly feeling her own voice in her ear as well as increased awareness of eye movements and her own steps. However, these symptoms did not affect the patient's quality of life.

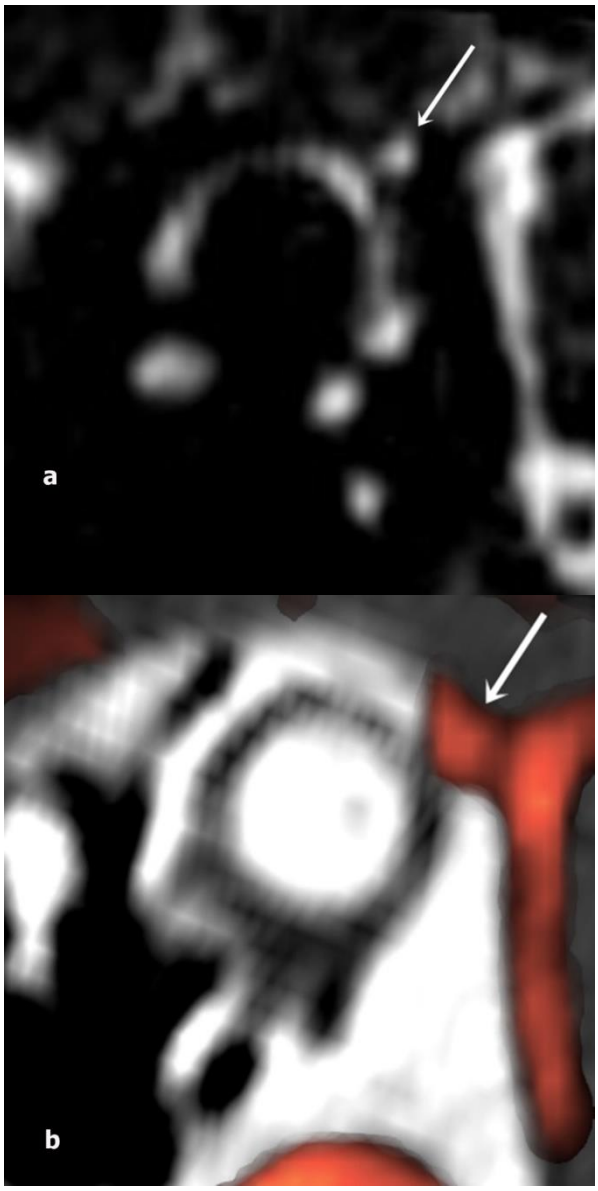
Figure 1: Bony coverage is absent over the superior semicircular canal and “cookie bite” appearance (white arrow) is seen in Pöschl plane high resolution computed tomography (HRCT).



Pure tone audiometry (PTA) showed average thresholds of air conduction at 0.5, 1, 2, and 4 kHz of 7 dB for the right and left ears. The average threshold of bone conduction was 3 dB for the right and left ears. There was no air/bone gap noted for either ear. The function of the bilateral SCC in VHIT was within the normal limits, but there were impaired responses in both lateral semicircular canals (LSC). There was no response in the left ear on cVEMP. These tests could not verify the preliminary diagnosis of SCC dehiscence.

Contrast-enhanced MRI was performed to reveal the dehiscence of SPS to SCC observed on HRCT. The examination was carried out with a 1.5-Tesla MRI scanner (Ingenia, Philips). The scans included axial plane T1, T2 W turbo spin echo, 3 dimensional (D) T2 W gradient echo (GRE), and 3D fat-suppressed T1 W sequences in the arterial and venous phases after contrast agent injection. Contrast enhancement of the SPS was observed in venous phase images. A three-class classification of SCC dehiscence by SPS was described in a recent article [4]. According this report, there was a cookie-bite appearance in the HRCT images and obvious compression of the membranous SCC by SPS on the MRI images, i.e., a ‘Class C’ category. In light of the imaging findings, we suggested Class C dehiscence in our patient (Figure 2). No operation was planned for the dehiscence of SCC because there was no impact on quality of life. Informed consent was obtained from the patient for this case report.

Figure 2: a: In a Pöschl projection, a T2 GRE sequence can image the vascular structure (white arrow). It has a contact with the membranous SSC, b: Class C dehiscence is confirmed with a merged image by HRCT and postcontrast T1W sequence.



Discussion

The rate of diagnosis of SCC dehiscence has increased gradually in the last two decades due to developments in imaging [8]. The vestibular system receives a large number of data inputs from many different systems and the compensatory mechanisms are high. The clinical findings of SCC dehiscence patients may be neglected by the patients as in our case. The patients can continue their lives asymptomatically. In our case, symptoms may differ depending on the variable resistance caused by vascular compression in SCC dehiscence associated with vascular structures such as SPS. Prominent pulsatile tinnitus finding is prominent in patients with SPS-related dehiscence [9]. Dehiscence is usually classified via radiological images, and there are a few articles reporting good correlation between the severity of clinical findings and the degree of radiological dehiscence [5]. In our case, however, a marked dehiscence appearance was observed on HRCT and contrast-enhanced MRI images, but faint patient complaints were described in a way that did not correlate well with these radiological findings.

Diagnosis can be made based on the impression of the SPS on the adjacent bone on CT. Contrast material was needed to

visualize the SPS (a vascular structure) radiologically and to study the relationship with SCC more clearly. A prior report used contrast-enhanced CT examination to reveal SPS involvement [9]. However, this step will increase the patient's radiation exposure because this contrast-enhanced CT examination will be performed in addition to the routine HRCT examination. Therefore, 3D T1 W venous phase images with contrast may be an alternative to reveal SPS involvement.

A few cases have previously been similarly studied using this 3 T MRI scanner [5]: Similar to our work, T2W images were fused with 3D contrast T1W images after the SCC were clearly revealed with 3-dimensional T2 W GRE images in addition to HRCT. Thus, the relationship between the two imaging methods could be seen more clearly. This prior work showed relatively good correlation between the degree of compression by the venous structures on the membranous SCC and the intensity of symptoms (based on their experience with only a few patients). Accordingly, three categories were defined. Asymptomatic patients with "cookie bite" appearance on CT examination but no connection at the labyrinth between SPS and membranous SSC in MR imaging were classified as Class A. Unlike Class A, cases with mild symptoms and limited contact between SPS and membranous SSC on MR imaging were classified into Class B. Cases with severe symptoms with obvious contact between SPS and membranous SSC on MR imaging were categorized as Class C [5].

One of the main problems in MRI is a lack of specific signal intensity for each tissue despite using the same protocol, scanning the same body area, and even imaging the same patient with the same device. Although many efforts have been made to quantitatively measure the tissues' intensity values, no practical method has yet been deployed broadly [10]. Thus, even small deviations in MRI signals could adversely affect clinical and radiological matches in this classification. Moreover, this error source may lead to different MRI signal intensities for each patient even if the same dose of the contrast agent and technical parameters are used [10]. Therefore, inconsistencies may occur in the classification of the degree of dehiscence because this very thin vascular structure cannot be fully reflected on the image in its real size. Our patient was clinically consistent with class A with a "cookie bite" appearance without any bony coverage in the HRCT images. The radiological appearance was different from that seen during Class A. Furthermore, the dehiscence size on MRI resembles Class C more than Class B. Our case had no good correlation between the imaging findings and the symptoms in our case. We could not distinguish between Class B and Class C with this inconsistency. This may be because we performed our examination with a 1.5-T MRI device, which did not have sufficient gradient power. We may not be able to fully distinguish between SPS contacting the endolymphatic canal and that causing compression.

Conclusion

SCC dehiscence of SPS is a rare entity that can be symptomatic with findings such as pulsatile tinnitus. In addition to HRCT, radiological examinations such as contrast- enhanced MRI can reveal the degree of dehiscence of SPS to SCC. Contrast-enhanced MRI can guide endovascular radiological interventional methods for therapy—these have become increasingly important. The classification of dehiscence from fused images created by combining different radiological techniques may vary depending on the differences in technical parameters. As a result, to reach more precise data on this subject, comparative studies with larger series and using different technical parameters and MRI devices with higher gradient power are needed.

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