



## Cochleovestibular artery syndrome: consideration based on VHIT, VEMP, and inner ear MRI

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Several hypotheses of acute cochleovestibular deficit (also known as “Labyrinthitis”) are discussed in the literature [1, 2]. Recently, Murofushi et al. described three patients with acute high-tone sensorineural hearing loss with vertigo and damage of the posterior semicircular canal function suggesting an impairment of the cochleovestibular artery [3]. The anterior vestibular artery provides the blood supply to the utricle, the superior, and the lateral ampullas, while the cochleovestibular artery provides the blood supply to the basal turn of the cochlea, the saccule, and the posterior ampulla [4].

A 60-year-old female was referred to our tertiary neurological center with an acute and harmonious unilateral cochleovestibular deficit. She presented an acute rotatory vertigo with right cochlear symptoms. She had a history of dyslipidemia and rheumatoid polyarthritis. At the examination, an inferior and counterclockwise spontaneous nystagmus was observed. No gaze-evoked nystagmus, no skew deviation, or evidence of positional nystagmus was found. The neurological examination was normal. The audiogram revealed a right high-tone sensorineural hearing loss (Fig. 1). A decreased right VOR gain (0.4) of the posterior semicircular canal was observed on VHIT (ICS Impulse, GN Otometrics, Taastrup, Denmark) on the affected side,

while the superior and lateral semicircular canals presented a normal VOR gain (Fig. 1). The oVEMP showed a normal utricular function with symmetrical responses. By contrast, cVEMP demonstrated an impairment of the right saccular function with asymmetrical responses (Fig. 1, asymmetry ratio = 0.49).

A brain and inner ear MRI (3T Siemens®, Skyra with a 64-channel head coil) with a 3D-FLAIR sequence performed 10 min and 4 h after a single intravenous dose of gadobutrol (Gadovist®, 0.1 mL/kg) was done the same day. Brain MRI showed no evidence of anterior inferior cerebellar artery infarction. While there were no labyrinthine anomalies on 10-min post-contrast 3D-FLAIR sequence, we found an increased enhancement of the right posterior semicircular canal, the cochlea, and the perilymph surrounding the saccule on 4-h post-contrast 3D-FLAIR sequence (Fig. 2). There was no radiological sign of endolymphatic hydrops.

Here, we discuss a patient that presented a specific involvement of the cochlea, the saccule, and the posterior ampulla on the affected side, which confirmed both clinically and on the MRI. The lesions and their location limited to these three organs suggest the hypothesis of an inner ear stroke of the right cochleovestibular artery [5–9].

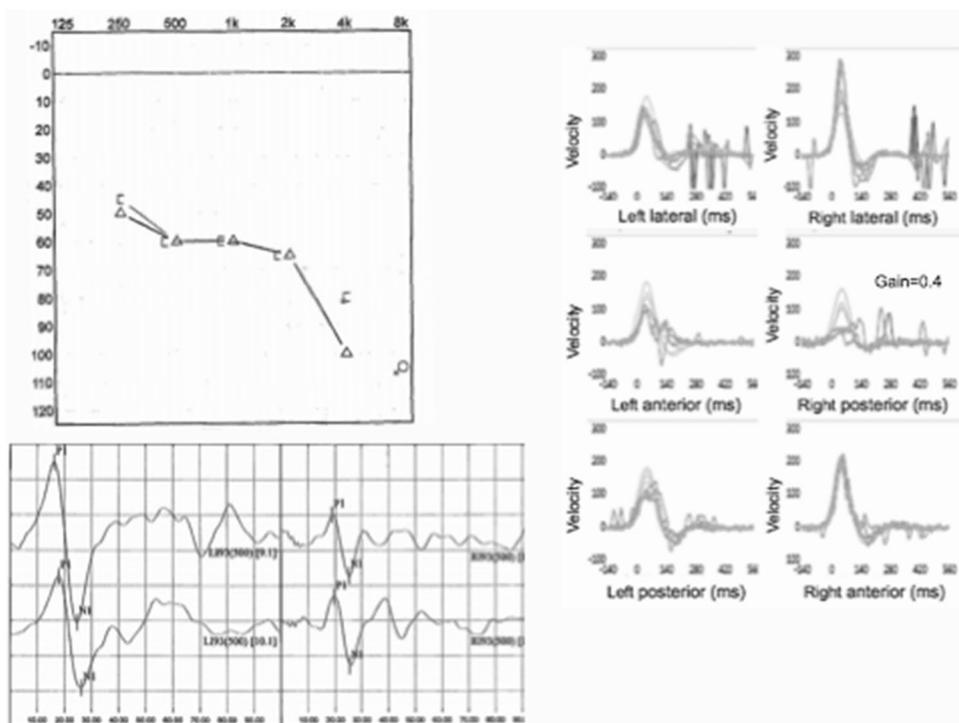
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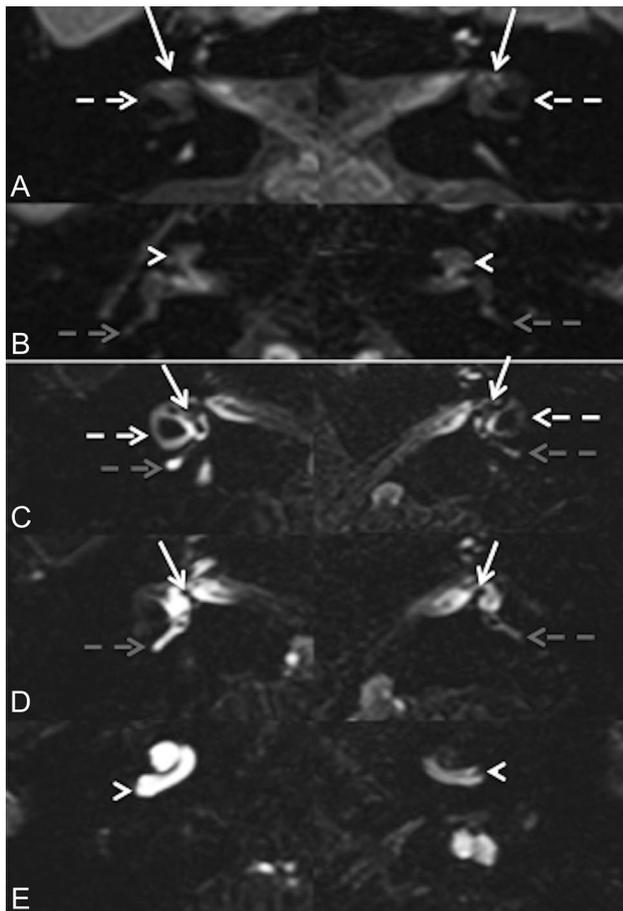
**Fig. 1** Upper left: pure-tone audiometry. High-tone sensorineural hearing loss; lower left: cVEMP responses. Saccular dysfunction on the right side (asymmetry ratio: 0.49); right: VHIT. Decreased VOR gain of the right posterior semicircular canal



3D-FLAIR sequences have the advantage of being more sensitive than the conventional sequences to detect T1-shortening [10, 11]. 10-min post-contrast 3D-FLAIR sequences allow assessing the breakdown of the blood–labyrinth barrier that could reflect marked inflammation or micro-vascular mechanisms [12]. 4-h post-contrast 3D-FLAIR sequences might evaluate slight impairment of the permeability of the blood-labyrinth barrier [13]. In our case, since the anomalies were only observed on 4-h post-contrast 3D-FLAIR, we have suggested that there was a slight impairment of the blood–labyrinth barrier of the cochleovestibular artery probably related to a vascular occlusion rather than an

inflammation or a hemorrhage. Moreover, the topography of the enhancement, observed on MRI, located at the cochlea, saccule and posterior ampulla, fits exactly with the cochleovestibular artery territory. If this acute cochleovestibular deficit was caused by viral labyrinthitis, diffuse enhancement of the labyrinthine structures should be observed on MRI [14]. Yet, partial labyrinthine enhancement has been described in viral labyrinthitis [15].

In this report, we suggest that cochleovestibular deficit associated with high-tone sensorineural hearing loss, isolated posterior semicircular hypofunction, and blood–labyrinth barrier impairment restricted to the area of



**Fig. 2** **a** 10-min post-contrast axial 3D-FLAIR at the level of the vestibule (white arrow) and the lateral semicircular canal (white dotted arrow) showing a symmetrical signal on both labyrinths. **b** 10-min post-contrast axial 3D-FLAIR at the level of the cochlea (white arrowhead) and the posterior semicircular canal (gray dotted arrow) demonstrating no signal anomaly. **c** 4-h post-contrast axial 3D-FLAIR at the level of the utricle (white arrow), the lateral (white dotted arrow), and posterior (gray dotted arrow) semicircular canals showing a marked enhancement of the right posterior semicircular canal. **d** 4-h post-contrast axial 3D-FLAIR at the level of the saccule (white arrow) and the posterior semicircular canal (gray dotted arrow) demonstrating an increased enhancement of the perilymph surrounding the saccule and of the posterior semicircular canal on the right side. **e** 4-h post-contrast axial 3D-FLAIR at the level of the cochlea (white arrowhead) showing an increased enhancement of the basal turn and the second turn of the right cochlea

the cochleovestibular artery could be diagnosed as “cochleovestibular artery syndrome”.

## Compliance with ethical standards

**Conflicts of interest** No conflict of interest.

**Ethical standards** All studies in this review have been approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 declaration of Helsinki and its later amendments.

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