Journal of Neurology (2019) 266:2575–2577
https://doi.org/10.1007/s00415-019-09456-0

LETTER TO THE EDITORS

Labyrinthine infarction caused by vertebral artery dissection: consideration based on MRI

Michael Eliezer[1] · Benjamin Verillaud[2] · Jean-Pierre Guichard[1] · Romain Kania[2] · Michel Toupet[3] · Philippe Herman[2] · Emmanuel Houdart[1] · Charlotte Hautefort[2]

Received: 10 June 2019 / Revised: 23 June 2019 / Accepted: 28 June 2019 / Published online: 2 July 2019

Dear Sirs,

Vertebral artery dissection (VAD) is the most common cause of ischemic stroke in young patients with an estimated incidence of 1 of 100,000 per year [1]. VAD is characterized by a mural hematoma, which leads to stenosis with a high embolic and thrombogenic potentials [2]. Since the anterior inferior cerebellar artery provides the blood supply to the central (brainstem and cerebellum) and peripheral (labyrinth) cochleovestibular pathways, patients with VAD associated with a cochleovestibular symptoms might present brainstem infarction and/or labyrinthine infarction caused by microembolism in the vertebrobasilar system [3, 4]. Recently, a radiological report demonstrated that 3D-FLAIR sequences performed 4 h after gadolinium administration might detect labyrinthine infarction due to the presence of a slight impairment of the blood–labyrinth barrier while 10 min post-contrast 3D-FLAIR showed no anomalies [5].

A 28-year-old female was referred to our tertiary neuro-otological center. She presented an acute left cervicalgia followed 3 days later by an acute rotatory vertigo with left cochlear symptoms, such as tinnitus and sudden hearing loss. She had a history of Willebrand disease. At the examination, a spontaneous right nystagmus with horizontal and torsional component was observed. No gaze-evoked nystagmus, no skew deviation or evidence of positional nystagmus were found. The neurological examination was normal. The audiogram revealed a left high-tone sensorineural hearing loss (Fig. 1) and a decreased left VOR gain of all semicircular canals was observed on VHIT (ICS Impulse, GN Otometrics, Taastrup, Denmark) (Fig. 1). Left saccular and utricular responses were not found on cVEMP and oVEMP, respectively (Fig. 1). The medical history supported the diagnosis of inner ear stroke due to a vertebral artery dissection. To confirm the hypothesis, a brain and inner-ear MRI (3T Siemens®, Skyra with a 64-channel head coil) with a 3D-FLAIR sequence was performed 4 h after a single intravenous dose of gadobutrol (Gadovist®, 0.1 mL/kg) in emergency the same day. The mural hematoma of the left vertebral artery was observed with non-contrast T1-weighted sequence with fat saturation and appeared as hyperintense with an eccentric "crescent" shape. Since, there was no evidence of vertebrobasilar infarction on diffusion-weighted imaging, 4 h post-contrast 3D-FLAIR was performed to assess the blood–labyrinthine barrier and found an increased enhancement of the left cochlea, vestibule and all three semi-circular canals (Fig. 1). Aspirin (100 mg) treatment was started.

We report a patient that presented a left VAD followed a few days later by an acute peripheral cochleovestibular deficit on the affected side, confirmed both clinically and on the MRI. The absence of a vertebrobasilar infarction and the presence of blood–labyrinth barrier impairment on MRI suggest the hypothesis of an inner ear stroke of the labyrinthine artery caused by microembolism. Few studies have reported various combinations of symptoms (isolated vertigo and/or hearing loss) in VAD patients and suggested an artery-to-artery embolism as a possible mechanism of labyrinthine ischemia depending on the branches involved [6]. The labyrinthine artery divides into the common cochlear and anterior vestibular arteries. The latter provides the blood supply to the anterior and lateral ampullas, the utricle and the superior part of the saccule. The common cochlear artery divides into the main cochlear and the cochleovestibular arteries. The cochleovestibular artery

✉ Michael Eliezer
michael.eliezer@aphp.fr

[1] Department of Neuroradiology, Neuroradiology Unit, Lariboisière University Hospital, 75010 Paris, France

[2] Department of Head and Neck Surgery, Lariboisière University Hospital, Paris, France

[3] Centre d'Exporations Fonctionnelles Otoneurologiques, Paris, France

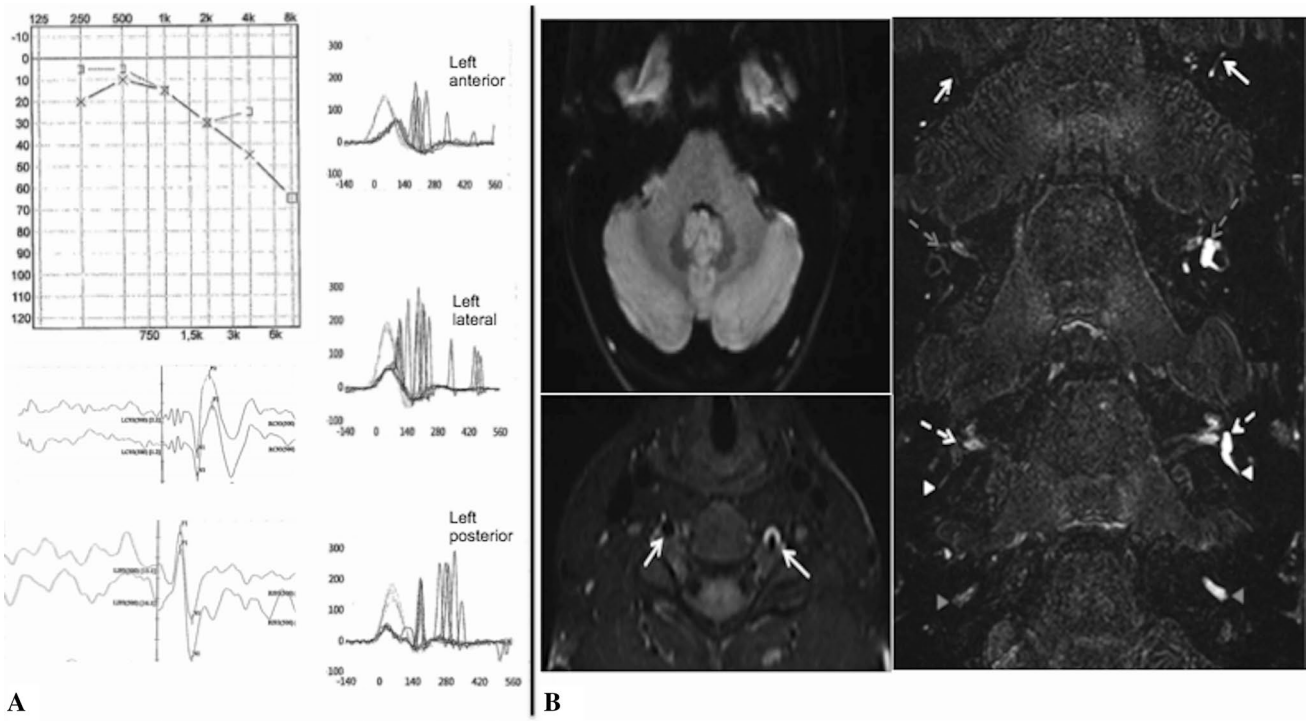

Fig. 1 **a** Upper left: pure-tone audiometry. High-tone sensorineural hearing loss; lower left: oVEMP and cVEMP responses. Utricular and saccular dysfunction on the left side; Right: VHIT. Decreased VOR gain of the left superior (upper), lateral (middle) and posterior (lower) semi-circular canals. **b** Upper left: axial diffusion-weighted images showing no anomalies; lower left: axial non-contrast T1-weighted images demonstrating left vertebral artery dissection on the left side (white arrow), appearing as an hyperintensity with an eccentric "crescent" shape. Right: 4 h post-contrast axial 3D-FLAIR at the level of the superior semicircular canal (white arrow), utricle (gray dotted arrow), saccule (white dotted arrow) and basal turn of the cochlea (gray arrow head). A marked enhancement is observed on the left side involving the whole labyrinthine structures

provides the blood supply to the basal turn of the cochlea, the inferior part of the saccule and the posterior ampulla [7].

As suggested by many studies, 4 h post-contrast 3D-FLAIR sequences might evaluate slight impairment of the permeability of the blood–labyrinth barrier [8, 9]. We cannot exclude the possibility of non-vascular causes such as infection since immediate post-contrast 3D-FLAIR sequence has not been performed. However, in our case, since the patient developed cochleovestibular deficit following VAD, we have suggested that the impairment of the blood–labyrinth barrier of the entire labyrinth was probably related to a microembolism in the labyrinthine artery from the VAD. Our presumption is also supported by a recent report on a patient with cochleovestibular artery syndrome that presented a blood–labyrinth barrier impairment on 4 h post-contrast 3D-FLAIR sequence restricted to the area of the cochleovestibular artery [5].

Compliance with ethical standards

Conflicts of interest There is no conflict of interest in this study.

Ethical approval This study was performed in accordance with Declaration of Helsinki (1964) and its later amendments.

References

1. Debette S, Grond-Ginsbach C, Bodenant M, Kloss M, Engelter S, Metso T, Pezzini A, Brandt T, Caso V, Touze E, Metso A, Canaple S, Abboud S, Giacalone G, Lyrer P, Del Zotto E, Giroud M, Samson Y, Dallongeville J, Tatlisumak T, Leys D, Martin JJ (2011) Cervical artery dissection ischemic stroke patients (CADISP) group: differential features of carotid and vertebral artery dissections: the CADISP study. Neurology 77:1174–1181
2. Debette S, Leys D (2009) Cervical-artery dissections: predisposing factors, diagnosis, and outcome. Lancet Neurol 8:668–678
3. Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, Yi SD et al (2002) Sudden deafness and anterior inferior cerebellar artery infarction. Stroke 33:2807–2812

4. Kim JS, Lopez I, DiPatre PL, Liu F, Ishiyama A, Baloh RW (1999) Internal auditory artery infarction: clinicopathologic correlation. Neurology 52:40–44
5. Eliezer M, Toupet M, Guichard JP, Kania R, Houdart E, Hautefort C (2019) Cochleovestibular artery syndrome: consideration based on VHIT, cVEMP and inner ear MRI. J Neurol. https://doi.org/10.1007/s00415-019-09407-9
6. Choi KD, Chun JU, Han MG, Park SH, Kim JS (2006) Embolic internal auditory artery infarction from vertebral artery dissection. J Neuro Sci 246:169–172
7. Merchant S, Nadol J (eds) (2019) Schuknecht's pathology of the ear, 3rd edn. McGraw-Hill Education, London
8. Sugiura M, Naganawa S, Teranishi M, Nakashima T (2006) Three-dimensional fluid-attenuated inversion recovery magnetic resonance imaging findings in patients with sudden sensorineural hearing loss. Laryngoscope 116:1451–1454
9. Tagaya M, Yamazaki M, Teranishi M, Naganawa S, Yshida T, Otake H, Nakata S, Sone M, Nakashima T (2011) Endolymphatic hydrops and blood-labyrinth barrier in ménière's disease. Acta Otolaryngol 131:474–479