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Case Report

Cochleovestibular Transient Ischemic Attack as a Manifestation of Patent Foramen Ovale

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Paradoxical embolization is the most commonly proposed mechanism for ischemia in patients with patent foramen ovale (PFO). We hypothesize that these patients can experience a microembolic genesis of cochleovestibular dysfunction, with a peripheral vestibular syndrome and simultaneous cochlear signs. Hence, we report the first case in literature associating PFO and acute transitory audiovestibular loss, which occurred in a male who had had an ischemic mesencephalic stroke several months ago. The patient's history and duration of the current event of less than 24 hours following complete functional recovery without any damage highlight the vascular cause in cochleovestibular dysfunction and support the existence of a clinical entity that could be named "cochleovestibular transient ischemic attack".

KEYWORDS: Patent foramen ovale, vertigo, sensorineural hearing loss, internal auditory artery, transient ischemic attack

INTRODUCTION

The foramen ovale is an important fetal structure that closes after birth in most individuals and remains open as a patent foramen ovale (PFO). The prevalence of PFO in the healthy adult population ranges from 15% to 25% on echocardiography and 15%-35% on autopsy studies [1, 2]. Under physiological conditions, a pressure gradient is maintained between the left and the right atrium, which results in the passive closure of the PFO. In the case of an increase in the right atrial pressure exceeding left atrial pressure (as observed at the end of Valsalva maneuvers, such as coughing, sneezing, squatting, defecation, or micturition), a transient right-to-left shunt may occur carrying particulate matter, such as thrombi, into the systemic circulation. A permanent increase in the right-sided cardiac pressures, as observed after pulmonary embolism or other causes of pulmonary arterial hypertension, results in a significant and possibly permanent right-to-left interatrial shunt, thereby increasing the risk for paradoxical embolism [2].

Acute audiovestibular loss is one of the most common neurotological dysfunctions in patients presenting with acute vertigo. It is characterized by acute onset of prolonged (lasting days) vertigo and hearing loss. Many neurological or neurotological conditions, including viral inflammations, vascular insults, trauma, hereditary or genetic causes, bacterial meningitis, connective tissue disorders, or metabolic derangements should be considered in the differential diagnosis of audiovestibular loss. Among the vascular causes, ischemic stroke in the territory of the anterior inferior cerebellar artery is known to be the leading cause of acute audiovestibular loss [3, 4]. We report the first case in literature associating PFO and acute transitory audiovestibular loss, which occurred in a male who had had an ischemic mesencephalic stroke several months ago. This highlights the vascular cause in cochleovestibular dysfunction and may validate audiovestibular disturbance as a sole manifestation of transient ischemic attack (TIA).

CASE PRESENTATION

A 43-year-old man was referred to our department with acute spontaneous vertigo with nausea/vomiting, left tinnitus and unsteadiness at his awakening. Several months before the patient had had an ischemic stroke in the posterior cerebral circulation, as proved by brain magnetic resonance imaging (MRI) that showed mesencephalic infarction (Figure 1). During the workup, PFO was diagnosed and high levels of homocysteine were observed; hence, he was treated with antiplatelet therapy and folic acid supplementation.

On admission, clinical ear, nose, and throat examination revealed horizontal spontaneous nystagmus toward the right side (unaffected ear) with a rotational component visible with and without Frenzel glasses. Nystagmus was persistent, one-directional and plu-

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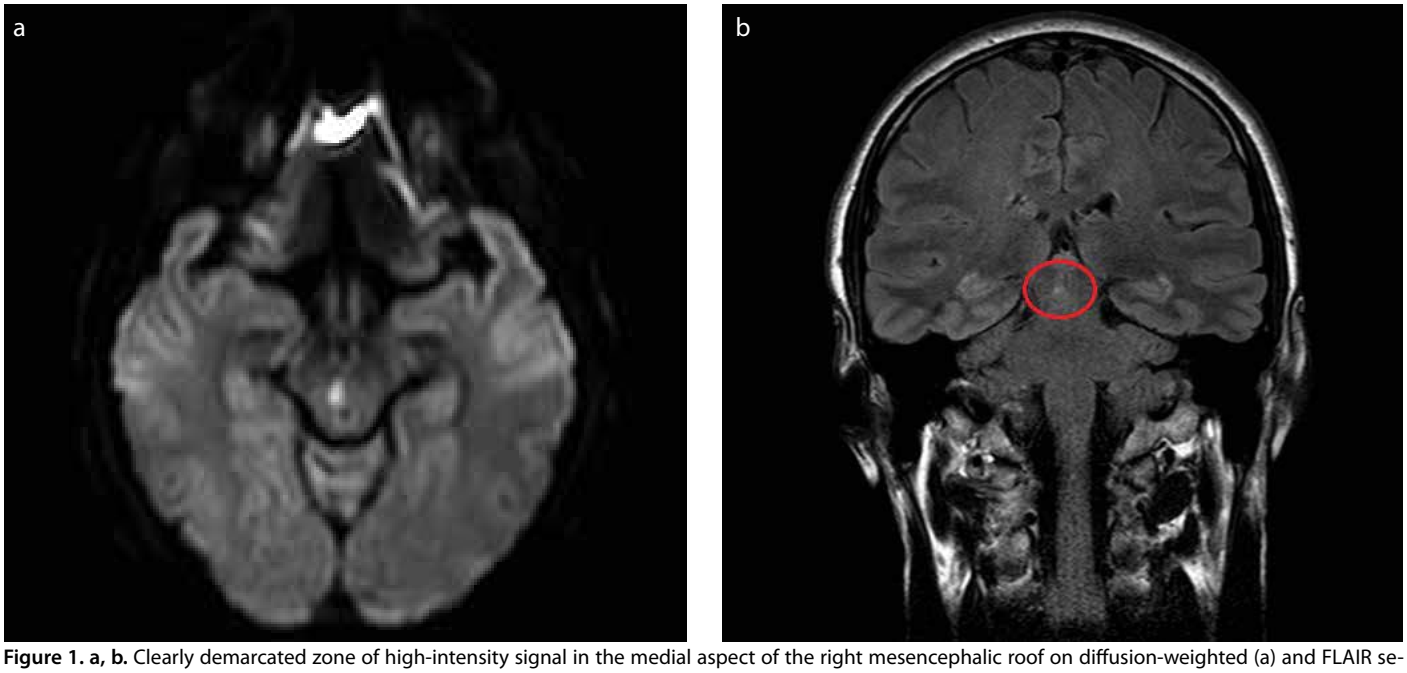

Figure 1. a, b. Clearly demarcated zone of high-intensity signal in the medial aspect of the right mesencephalic roof on diffusion-weighted (a) and FLAIR sequences (b) consistent with recent lacunar infarction

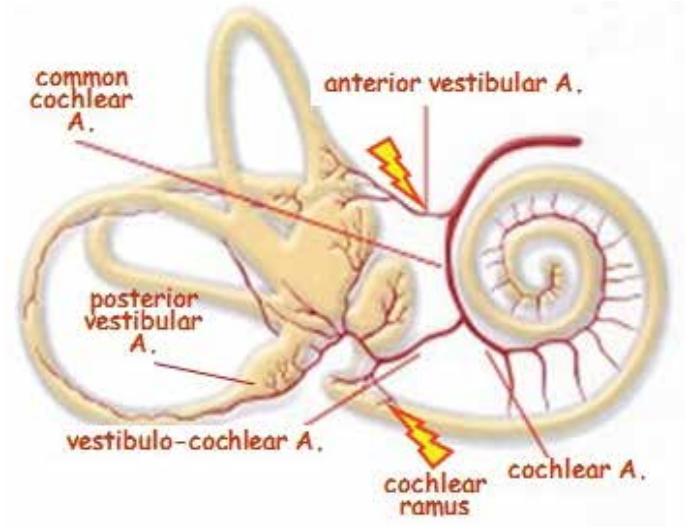

Figure 2. Schematic diagram of vascular distribution of the labyrinth. Arrows indicate vessels involved in this clinical case

ri-positional during bedside examination, while paroxysmal positional nystagmus was not evoked. Postural imbalance with falls toward the left (affected ear) was also present. The head impulse test (HIT) and caloric irrigation showed a deficit of the vestibulo-ocular reflex (VOR) on the left side, as confirmed by video HIT (vHIT). A deviation toward the left ear at the subjective visual vertical (SVV) test indicated an utricular damage. An early morphologically well-recognizable biphasic complex (p1n1) was present, and the values appeared normal for amplitude and latency (13-23 m/sec) in the recording of the cervical vestibular-evoked myogenic potentials ipsilateral to the lesion, indicating saccular function preservation. Pure tone audiometry diagnosed a moderate sensorineural hearing loss at high frequencies in the left ear.

On the following day, 24 hours after the onset of symptoms, patient reported the disappearance of vertigo and hearing loss and a clear improvement of tinnitus. Nystagmus was not present during bedside examination. Caloric test, SVV test, HIT, and vHIT were normal and did not show any deficit in VOR. A new MRI excluded recent ischemic or hemorrhagic lesions. Written informed consent was obtained from the patient for publication of the report.

DISCUSSION

In this case, a patient with PFO experienced acute spontaneous vertigo with nausea/vomiting, tinnitus, and sudden hearing loss. Patient's history, along with his clinical course directed us to a vascular cause. The absence of recent lesions in a cerebral MRI and the result of vHIT suggested a peripheral location of vascular injury within the area vascularized by the internal auditory artery (IAA) [5-7]. The clinical picture indicated an alteration of vestibular receptors moistened by the anterior vestibular artery, resulting in lateral semicircular canal (spontaneous nystagmus, caloric canal paresis, low VOR gain, and corrective saccades at vHIT) and utricular hypofunction (SVV tilt toward the lesion). Simultaneously, the audiological findings suggested a major involvement of the cochlear branch of the vestibule-cochlear artery vascularizing basal turn of the cochlea, appointed to high tone perception (Figure 2). Although this medical case did not appear dissimilar to a partial vestibule-cochlear stroke at the beginning, the duration of the event (<24 hour) and complete functional recovery without any damage pointed toward the diagnosis of TIA in the cochleovestibular region (Figure 3). For the same reasons and for the concomitant sudden hearing loss, a vestibular neuritis was to be excluded. Furthermore, pure tone audiometry that showed sensorineural hearing loss at high frequencies excluded a diagnosis of typical endolymphatic hydrops.

Patent foramen ovale increases the risk for paradoxical embolism and this may result in neurological disorders related to ischemia (stroke or TIA) [2]. Stroke is considered cryptogenic when a cause has not been identified. Depending on the classification system, cryptogenic strokes account for almost 30% of cases and are more common in young patients. Multiple studies have demonstrated an association between

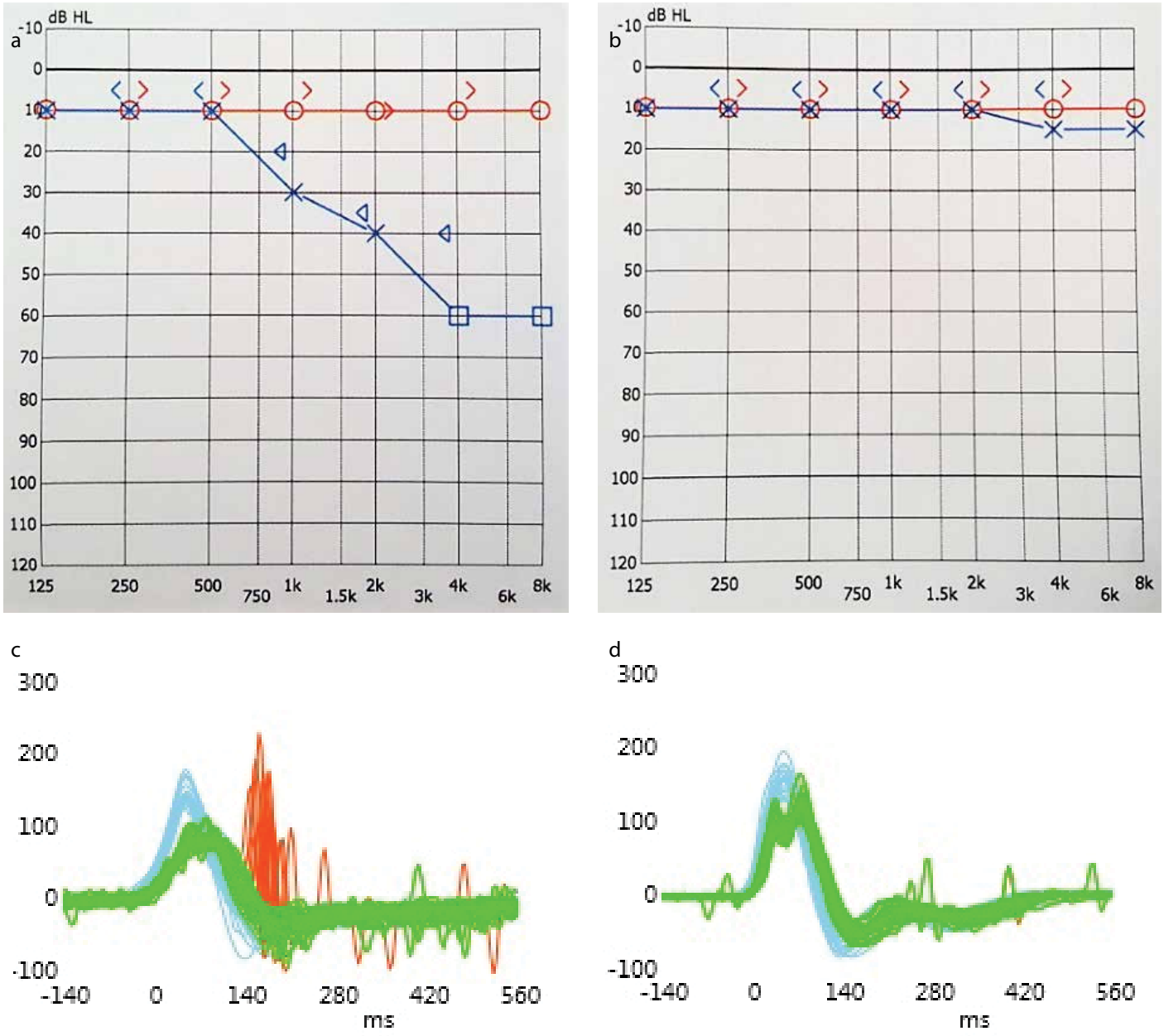

Figure 3. a-d. Pure tone audiometry (a,b) and vHIT (c,d) accomplished on admission (a,c) and after 24 hours (b,d) showed complete restoration of cochlear and vestibular functions

PFO and cryptogenic stroke [8, 9]. Since the inner ear requires a high-energy metabolism and the IAA is an end artery with little collaterals from the otic capsule, the inner ear is particularly vulnerable to ischemia [10]. Therefore, audiovestibular disturbance as a sole manifestation of TIA is possible.

CONCLUSION

This is the first case reported in literature about the association between PFO and cochleovestibular dysfunction. Acute audiovestibular loss in a patient with PFO could have an embolic source and differential diagnosis with neuritis is mandatory. At the same time, vHIT is always more important to differentiate peripheral lesions from central lesions. In particular, this case highlights the inner ear as a crucial site of embolization and supports the existence of a clinical entity that could be named "cochleovestibular transient ischemic attack".

Informed Consent: Written informed consent was obtained from the patients who participated in this study.

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