Pearls & Oy-sters: Labyrinthine Infarction Mimicking Vestibular Neuritis

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PEARLS

- Acute unilateral audiovestibulopathy is mostly caused by inflammatory disorders
involving the labyrinth.

- Vascular compromise of the inner ear rarely causes acute unilateral audiovestibulopathy.
- The flocculus and labyrinth share a vascular supply from the anterior inferior cerebellar artery.

OY-STERS

- Labyrinthine infarction may cause acute unilateral peripheral vestibulopathy and thus mimic vestibular neuritis.
- Concomitant labyrinthine infarction should be suspected in acute vestibular syndrome in patients with acute infarction in the territory of the anterior inferior cerebellar artery.
CASE REPORT

A 63-year-old man with a history of diabetes mellitus and hypertension presented with acute vertigo for 3 hours. The vertigo began abruptly while driving and was accompanied by a popping sound and fullness in the left ear that lasted only tens of seconds. The patient also had nausea and vomiting, yet denied positional aggravation of the vertigo, or associated diplopia or hearing loss. He had no antecedent history of infection.

Examination showed spontaneous nystagmus beating rightward, upward and clockwise (the upper poles of the eyes beating to the patient’s right shoulder.) without visual fixation (Figure A). Saccades were normal, but smooth pursuit seemed impaired to the right. The patient did not show direction-changing gaze-evoked nystagmus (GEN) or skew deviation. Hearing was decreased on the left side on finger rub test. Video head-impulse tests (HITs) were positive for the left horizontal canal (HC) with a decreased gain and overt saccades, but normal for the vertical canals (Figure B, Video http://links.lww.com/WNL/B446 ). Horizontal saccades were normal. After correction of the spontaneous nystagmus, smooth pursuit at a peak target velocity of 20°/s was normal with a gain of 0.79 to the right and 0.71 to the left. Bithermal caloric tests showed a left canal paresis of 29%. Fundus photography showed abnormal extorsion of the left eye (Figure C). The subjective visual vertical was tilted to the left only during left-eye viewing (-3.3°, normal range: -3.8 – 3.1°), but normal during right-eye (-2.7°, normal range: -3.1 – 3.0°) or binocular viewing (-2.4°, normal range: -2.4 to 2.6°). Rotatory chair tests documented decreased gains of the horizontal vestibulo-ocular reflex (VOR) without an asymmetry, as well as phase leads during sinusoidal harmonic acceleration at frequencies from 0.01 to 0.04 Hz. The patient showed a high-tone sensorineural hearing loss in both ears, slightly more prominent in the left ear (Figure D). Speech discrimination was normal. Results of cervical and ocular vestibular-evoked myogenic potentials and auditory evoked potentials were also unrevealing. MRIs disclosed an acute infarction involving the left flocculus (Figure E). MR angiography
and perfusion CT were normal. The patient was placed on 100 mg of aspirin, 75 mg of clopidogrel, and 40 mg of atorvastatin. The vertigo and vomiting improved over the following days along with resolution of the spontaneous nystagmus.

**DISCUSSION**

Our patient with acute unilateral floccular infarction documented with MRIs masqueraded as vestibular neuritis to exhibit horizontal-torsional nystagmus beating away from the lesion side, ipsilesional canal paresis and abnormal HITs, and ipsiversive ocular torsion and SVV tilt along with subtle auditory symptoms.

*Concomitant labyrinthine and cerebellar infarction*

Given the common vascular supply to the cerebellar flocculus and inner ear, mostly from the anterior inferior cerebellar artery (AICA, Figure F), findings of acute unilateral peripheral vestibulopathy may be ascribed to concomitant labyrinthine infarction that is hardly visualized with current imaging technique in our patient. Indeed, the diagnosis of labyrinthine infarction is usually made when patients show combined loss of hearing and peripheral vestibular function in association with brainstem or cerebellar infarction in the territory of AICA. Occasionally, diffusion-weighted images (DWI) can also visualize an infarction directly involving the vestibulocochlear nerve, or the labyrinth. Given the common vascular supply of the vestibulocochlear nerve and the labyrinth, infarction are hardly expected to involve the vestibular nerve only and spare the cochlear nerve. Indeed, only 4% of patients with infarction in the territory of AICA can present with acute vestibulopathy in isolation. However, as in our patient, the auditory symptoms may not be striking in embolic labyrinthine infarction when the anterior vestibular artery is preferentially involved. Indeed, laboratory evaluation showed predominant involvement of the HC. Given the normal angiogram, steno-occlusion of the proximal portion of the AICA and subsequent
recanalization or embolism may be presumed as the mechanism.

**Differentiation of labyrinthine infarction from vestibular neuritis**

The diagnostic hallmarks of vestibular neuritis are spontaneous contralesional horizontal-torsional nystagmus, positive HITs for the ipsilesional canals, and ipsilesional canal paresis. Unlike Ramsay-Hunt syndrome or labyrinthitis that can show swelling or gadolinium enhancement in the vestibular nerve or labyrinth, conventional MRIs of the inner ear are almost always normal in vestibular neuritis. As such, the diagnostic utility of MRIs is limited unless using a high-dose gadolinium or a 4-hour delayed 3D FLAIR images. Thus, the diagnosis of labyrinthine infarction should be based on findings of history taking and clinical evaluation. Absence of antecedent infection and the hyperacute onset of vertigo are also atypical for vestibular neuritis.

**Catching the subtle auditory and central ocular motor signs**

Although the three-item bedside eye movement screening (HINTS) shows excellent sensitivity, it can be often misleading in AICA infarction. Moreover, DWI often fail to detect the labyrinthine infarction which can be a harbinger of AICA infarction. Thus, scrutinized neurotologic evaluation is important for diagnosing infarction involving the labyrinth and cerebellum. In our patient, the diagnosis of labyrinthine infarction was challenging since the auditory symptoms were only subtle and transient. Patients may not notice hearing impairments during an attack of vertigo when the hearing loss is subtle. In this case, the finger rub test may help detecting asymmetric hearing on bedside. Since HINTS may fail to detect labyrinthine infarction, detection of hearing loss using the finger rub test (HINTS-plus) is important for diagnosing vascular inner ear damage.

Even though floccular infarction was found on MRI, our patient did not show ipsilesional spontaneous nystagmus, GEN, impaired smooth pursuit or increased gain of the VOR during rotatory chair test, which are ocular motor signs indicating floccular dysfunction.
However, the small corrective saccades for the contralesional right HC may be ascribed to a partial involvement of the left flocculus or adaptation to the labyrinthine infarction.\textsuperscript{14}

In conclusion, a scrutinized history taking and thorough neurotologic evaluation are important for differentiating labyrinthine infarction from inflammatory disorders involving the labyrinth.
References


Figure legend

**Figure. A.** Video-oculography shows spontaneous nystagmus beating rightward, upward and clockwise (the upper poles of the eyes beating to the patient’s right ear.) without visual fixation in darkness. *Upward deflection represents rightward, upward, and clockwise eye motion in each video-oculography recording.* **B.** Video head-impulse tests show a decreased gain and covert (black arrow heads) and overt saccades (black arrows) for the left horizontal canal along with small corrective saccades for the right horizontal canal. **C.** Pure-tone audiometry revealed bilateral sensorineural hearing loss with a pure tone average at 22.5 dB in the right, 36.3 dB in the left. **D.** Fundus photography shows extorsion of the left eye (normal range: 0° ~ 12.6°, A negative value indicates intorsion.). **E.** Diffusion-weighted images (Intera, Philips Medical Systems, Best, The Netherlands) 4 hours from the onset of vertigo disclosed a discrete lesion in the left flocculus (yellow arrow). **F.** Illustration of the vascular supply to the flocculus and labyrinth that share a common arterial supply from the anterior inferior cerebellar artery. AC=anterior canal; H=horizontal position of the left eye; HC=horizontal canal; PC=posterior canal; T=torsional position of the left eye; V=vertical position of the left eye.
Video Legend

Video. Video head-impulse tests are positive for the left horizontal canal. The horizontal position of the right eye is marked in red. Upward deflection represents rightward eye motion in the recording.
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