Case Report

Inferior Peduncle Lesion Presenting With Bilaterally Impaired Vestibular Responses to Horizontal and Posterior Head Impulses

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Differentiating central from peripheral origins of vestibulo-ocular reflex (VOR) lesions can be challenging. A 36-year old man presented with a 1-year history of progressive unsteadiness. The video-Head Impulse Test revealed a significantly reduced VOR gain in both horizontal and posterior canals (0.49 ± 0.05 and 0.38 ± 0.06) but normal VOR responses in both anterior canals (0.89 ± 0.08 and 1.04 ± 0.15). No plausible combination of end-organ lesion should be responsible for these observations. A brain magnetic resonance imaging disclosed a left inferior cerebellar peduncle lesion suggestive of a glioma.

Key Words: vestibulo-ocular reflex (VOR), vestibular, cerebellar lesion.

Level of Evidence: NA

CASE REPORT

A 36-year old man presented with a 1-year history of progressive unsteadiness, particularly when in the darkness. He had no cochlear symptoms; history of trauma; prior complaints of vertigo or dizziness; or vestibular, visual, or postural symptoms.

Examination revealed no spontaneous, gaze-evoked, or positional nystagmus. The clinical head-impulse test (HIT) was pathologic bilaterally when testing the horizontal canals (see Supp. Video S1) and normal when testing the vertical canals (see Supp. Video S2). The neurological examination was otherwise unremarkable.

The video-Head Impulse Test (vHIT) revealed a significantly reduced vestibulo-ocular reflex (VOR) gain in both the horizontal (0.38 ± 0.07 and 0.29 ± 0.05) and posterior canals (0.49 ± 0.05 and 0.38 ± 0.06), with covert and overt corrective saccades but normal VOR responses in both anterior canals (0.89 ± 0.08 and 1.04 ± 0.15) for right and left impulses, respectively. With caloric testing, there was a right unilateral weakness (56%). The remaining oculomotor tests were normal. The ocular vestibular-evoked myogenic potential (VEMP) and cervical VEMP responses were normal and symmetrical. A brain magnetic resonance imaging (Fig. 1) disclosed a left inferior cerebellar peduncle lesion that was suggestive of a glioma.

The superior vestibular nerve carries primary afferents from the anterior and lateral ampullae and the inferior vestibular nerve from the posterior ampullae. These primary vestibular afferents connect to the vestibular nuclei and the cerebellum. End-organ lesions such as vestibular neuritis usually affect only part of the vestibular nerve, mostly the superior division, with the inferior division commonly being spared. When evaluating our patient in the physiological frequency domain with the vHIT, VOR was deficient in both horizontal and posterior semicircular canal (SCC) directions and normal in both anterior SCC directions. In the plane of each SCC, head impulses in the direction of the ampulla typically result in excitatory VOR responses. No plausible combination of end-organ lesion, therefore, should be responsible for these observations.

The primary afferents that are destined for the cerebellum bypass the vestibular nuclei and proceed through the inferior cerebellar peduncle to the ipsilateral flocculus, nodulus, and anterior uvula. The flocculus governs by inhibition the central connections of the anterior SCC but not the posterior canals. We speculate that this disinhibitory effect could contribute to the rather compensatory eye responses in the anterior SCC direction. There was a diminished caloric response in the right side, but cerebellar lesions may increase, decrease, or have no influence in VOR gain at low frequencies. VEMP results also match previous results on circumscribed cerebellar lesions.

Differentiating central from peripheral origins of VOR lesions can be challenging. To the best of our knowledge, this is the first report where three-dimensional vHIT, by means of peripheral-unlikely combinations of VOR lesion, has shown to be of topodiagnostic value.
Fig. 1. Brain magnetic resonance imaging with contrast, three-dimensional video-Head Impulse Test, and vestibular evoked myogenic potential. (A) Left inferior peduncle lesion on axial T2-image. (B) Video Head Impulse. The plots show the velocity trajectories (o/s) of the eye and head for LARP (left anterior–right posterior), RALP (right anterior–left posterior), and lateral semicircular canal (SCC) stimulation with about 20 brief, unpredictable head turns in the direction of each SCC. Each trace corresponds to one head impulse. With head impulses in the direction of both horizontal and posterior SCCs, the vestibulo-ocular reflex (VOR) (green) does not compensate for the head (blue for left; red for right). The VOR is deficient, and the patient makes compensatory refixation saccades during and after the head impulses. With head impulses in the direction of both anterior SCCs, the VORs compensate for the head thrust. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

BIBLIOGRAPHY


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