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Acute Vertigo with Double Vision – Brainstem Stroke or Stroke Mimic?

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Introduction

We present a patient with acute-onset vertigo and vertical diplopia in whom posterior circulation ischemia was initially suspected. Further clinical and radiological tests resulted in a diagnosis of acute vestibular neuropathy mimicking ischemia of the vestibular nerve entry zone. This case illustrates the importance of subtle clinical bedside assessment to distinguish between central and peripheral vestibular disorders.

Case Description

A 56-year-old male with hypercholesterolemia presented with a 2-day history of acute-onset vertical diplopia, vertigo and nausea. The vertigo was aggravated by head movements and best tolerated in the recumbent position. On admission the blood pressure was 180/110 mm Hg. Evaluation of diplopia revealed left hypotropia without any change in vertical ocular deviation with gaze position, suggesting skew deviation (SD). The perceived visual vertical deviated towards the left by 6.25° bilaterally [1]. Spontaneous right-beating horizontal-torsional nystagmus with increasing drift velocity at right gaze was observed. Head impulse testing showed rightward catch-up saccades after head thrusts to the left. The combination of an acute vestibular syndrome and SD suggested a diagnosis of posterior inferior cerebellar artery infarction involving the vestibular nerve root entry zone. Cerebral MRI including DWI on day 2 after symptom onset showed no ischemic lesion or other parenchymal abnormalities. Also a follow-up MRI on day 5 (including high-resolution T₂-weighted 3-dimensional sequences, 3-mm slices) was normal. Caloric irrigation indicated complete canal paresis on the left according to Jonkees' formula. Serological tests did not reveal recent infections with *Borrelia burgdorferi*, varicella zoster or herpes simplex virus. Together, these findings were more consistent with idiopathic acute left peripheral vestibular loss. Therefore we recommended treatment with corticosteroids [2] and vestibular physiotherapy. The patient fully recovered within a month.

Discussion

The combination of an acute vestibular syndrome with SD usually indicates posterior circulation ischemia [3]. In a series of 83 patients presenting with acute vestibular syndrome, SD was by far the most specific indicator of a central vestibular lesion [3], which in the context of apoplectiform onset and vascular risk factors is often ischemic in nature. SD is caused by a unilateral or asymmetric deficit of otolith organs or graviceptive pathways projecting to oculomotor and thalamic nuclei (ventral posterolateral nucleus) and is most often observed in the context of brainstem or cerebellar lesions [4]. SD has also rarely been described in peripheral vestibular lesions, e.g. after vestibular neurectomy [5]. In a small case series, Safran et al. [6] reported a case with vestibular neuropathy and prominent vertical diplopia due to SD, but no MRI was performed. Thus, abnormalities of the central vestibular system may have gone undetected. In the present case, repeated MRI studies with thin sections and combined axial and coronal DWI sequences were without pathological findings after 2 and 5 days.

The combination of spontaneous horizontal-torsional nystagmus, pathological head impulse test and pathological response to caloric stimulation suggested a unilateral peripheral vestibular loss, most probably due to vestibular neuritis. The absence of auditory symptoms argues against an ischemic labyrinthine dysfunction, which only rarely results in isolated loss of vestibular function [7], and would be expected to be associated with DWI changes in the territory of the anterior inferior cerebellar artery.

Vestibular disorders account for 1–6.4% of the patients presenting with stroke mimics in 2 large series [8, 9]. However, the occurrence of SD in this context has not been evaluated systematically.

In order to increase the sensitivity of lesion identification in the posterior fossa, high-resolution imaging with thin sections and combined axial and coronal DWI sequences as well as dynamic susceptibility contrast perfusion MRI are usually recommended in patients with posterior circulation syndromes [10, 11].

References

- 1 Zwergal A, Rettinger N, Frenzel C, Dieterich M, Brandt T, Strupp M: A bucket of static vestibular function. *Neurology* 2009;72:1689–1692.
- 2 Strupp M, Zingler VC, Arbusow V, Niklas D, Maag KP, Dieterich M, Bense S, Theil D, Jahn K, Brandt T: Methylprednisolone, valacyclovir, or the combination for vestibular neuritis. *N Engl J Med* 2004;351:354–361.
- 3 Cnyrim CD, Newman-Toker D, Karch C, Brandt T, Strupp M: Bedside differentiation of vestibular neuritis from central 'vestibular pseudo-neuritis'. *J Neurol Neurosurg Psychiatry* 2008;79:458–460.

- 4 Brodsky MC, Donahue SP, Vaphiades M, Brandt T: Skew deviation revisited. *Surv Ophthalmol* 2006;51:105–128.
- 5 Riordan-Eva P, Harcourt JP, Faldon M, Brookes GB, Gresty MA: Skew deviation following vestibular nerve surgery. *Ann Neurol* 1997;41:94–99.
- 6 Safran AB, Vibert D, Issoua D, Haeusler R: Skew deviation after vestibular neuritis. *Am J Ophthalmol* 1994;118:238–245.
- 7 Lee H, Kim JS, Chung EJ, Yi HA, Chung IS, Lee SR, Shin JY: Infarction in the territory of anterior inferior cerebellar artery: spectrum of audiovestibular loss. *Stroke* 2009;40:3745–3751.
- 8 Hand PJ, Kwan J, Lindley RI, Dennis MS, Wardlaw JM: Distinguishing between stroke and mimic at the bedside: the brain attack study. *Stroke* 2006;37:769–775.
- 9 Hemmen TM, Meyer BC, McClean TL, Lyden PD: Identification of nonischemic stroke mimics among 411 code strokes at the University of California, San Diego, Stroke Center. *J Stroke Cerebrovasc Dis* 2008;17:23–25.
- 10 Warach S, Kidwell CS: The redefinition of TIA: the uses and limitations of DWI in acute ischemic cerebrovascular syndromes. *Neurology* 2004;62:359–360.
- 11 Förster A, Ottomeyer C, Wolf ME, Kern R, Griebe M, Gass A, Hennerici MG, Szabo K: Dynamic susceptibility contrast perfusion MRI identifies persistent vessel pathology in acute pontine stroke. *Cerebrovasc Dis* 2010;29:389–394.

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