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Central positioning upbeat nystagmus and vertigo due to pontine stroke

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Study concept and design: Habek and Gabelić. Acquisition of data: Habek, Gabelić, Pavliša and Brinar. Analysis and interpretation of data: Habek, Pavliša and Brinar. Drafting of the manuscript: Habek. Critical revision of the manuscript for important intellectual content: Habek, Gabelić, Pavliša and Brinar. Administrative, technical, and material support: Habek and Gabelić.

Abstract

We present a case of central positioning nystagmus and vertigo (c-PPV) due to pontine stroke. The presented case is the first case of central upbeat positioning nystagmus caused by pontine lacunar stroke. This case, together with previously published cases supports the existence of ventral tegmental tract in humans.

Key words: vertigo, nystagmus, upbeat, pontine stroke, ventral tegmental tract

We report a case of central positioning nystagmus and vertigo (c-PPV) due to pontine stroke.

Case report

A 60-year old female presented with 3 weeks history of vertigo. Her symptoms were most prominent when getting up from bed and turning in the bed, and vertigo was not accompanied with nausea and vomiting. At the time of symptoms onset she was on holiday and was examined in a local emergency department, neurological examination was normal and she was discharged home. As her symptoms persisted, when she returned home she presented in our emergency room. Neurological examination was again normal, there was no ophthalmoplegia, saccadic pursuit, spontaneous or gaze-evoked nystagmus, weakness, or cerebellar ataxia. Her medical history was unremarkable.

Because her symptoms were consistent with benign paroxysmal positional vertigo (BPPV) Dix-Halpike test was performed. In the right position pure up-beat nystagmus was noted without latency lasting for 15 s (Video 1). On repeated test there was no attenuation of the nystagmus. After repeated Epley maneuver there were no improvement.

Brain MRI was performed to reveal lacunar post-ischemic lesion visible in pons (Figure 1). MRI angiography was normal. Laboratory examination revealed hypercholesterolemia. Holter electrocardiography and heart ultrasound were normal. Blood pressure and glucose levels were normal. She was prescribed with Aspirin 100 mg QD and atorvastatin 20 mg QD.

Discussion

Central positioning nystagmus and vertigo is caused by a central lesion, usually in the posterior fossa either dorsolateral to the fourth ventricle or in the dorsal vermis.¹ This is usually due to a tumor or hemorrhages, and infarction appears to be seldom the cause, in contrast to its frequency of occurrence.¹ The direction of the nystagmus in c-PPV can be downbeat in the head-hanging position, counterclockwise in the right-hanging position, downbeat and left beating in the left-hanging position, upbeat in the supine position, or torsional with positioning.¹ Isolated upbeat nystagmus in c-PPV is extremely rare.

There are several reports in the literature of upbeat nystagmus due to pontine lesions. One of the most interesting one, pertinent to our case, is a patient with a lacunar infarction located in the paramedian and posterior part of the basis pontis, at the upper pons level which presented with spontaneous upbeat nystagmus.² It was suggested that lesions in this region could damage ventral tegmental tract (VTT). VTT connects the superior vestibular nucleus which receive the excitatory anterior canal inputs, thus generating the upward slow phases, to the superior rectus and inferior oblique motoneurons in the third nerve nucleus.^{2,3} The course of this tract could explain how a relatively small unilateral paramedian lesion involving the VTT decussation system may result in upbeat nystagmus.³

Differentiation between BPPV and c-PPV can be difficult, however careful examination of the nystagmus is usually sufficient in making the right diagnosis. The isolated upbeat

nystagmus (without rotatory component), no latency, no attenuation or resistance to Epley maneuver are “red flags” that should prompt the clinician to order MRI. The presented case is the first case of central upbeat positioning nystagmus caused by pontine lacunar stroke. This case, together with previously published cases supports the existence of ventral tegmental tract in humans.

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Video legend

Video1.

The patient in a Dix-Halpike test showing pure upbeat nystagmus without latency.

Figure legend

Figure 1.

Magnetic resonance imaging.

Lacunar post-ischemic lesion visible in pons on A: axial T2-weighted image, B: Fluid-attenuated inversion recovery (FLAIR) image and C: coronal T2-weighted image. D: a map of the apparent diffusion coefficient shows elevated diffusion in the lesion.

