Vertical upbeat nystagmus with isolated acute middle cerebellar peduncle stroke

Kinjal Desai, Thomas Bullock, Rahul Damani, Eric Bershad

CASE REPORT

A 68-year-old female with diabetes mellitus, hypertension, hyperlipidemia presents with hypertensive urgency, severe nausea, vomiting, and visual impairment. Initial blood pressure was 236/110 mmHg. She had a Glasgow Coma Scale of 15 and National Institute of Health Stroke Scale (NIHSS) of 3 (1 gaze impairment, 1 face weakness and 1 sensory loss). Neurological examination was significant for binocular vertical diplopia in all directions of gaze, worse on far gaze. She also had left lateral rectus weakness, left skew deviation, right hypertropia and a characteristic vertical upbeat nystagmus (left eye more than right eye), which was mostly evident in up and leftward gaze (Video 1). Routine laboratory testing for complete blood count, basic metabolic panel, and cardiac enzymes was unremarkable except for blood glucose of 260. Head computed tomography scan showed no acute abnormality. Brain magnetic resonance imaging scan (Figure 1) demonstrated an acute infarct in the medial aspect of left middle cerebellar peduncle. Her remaining workup, including magnetic resonance angiogram of head and neck and cardiac echo was unremarkable. The etiology of her ischemic stroke were thought to be related to small vessel disease.

Anatomy and Localization

Cerebellum is connected to brainstem by three bilaterally paired cerebellar peduncles [1]. The superior cerebellar peduncle which connects to Midbrain contains efferent fibers from the dentate, emboliform, globose and fastigial nucleus. The superior cerebellar artery supplies it. The inferior cerebellar peduncle connects to medulla oblongata and is supplied by posterior inferior cerebellar artery. The MCP connects to Pons and is the most lateral and largest of three peduncles [2]. It contains corticopontine fibers from contralateral side that crosses in pons as transverse pontine fibers and enters MCP to form the mossy fiber pathway. It also receives fibers from pontine tegmental nuclei [1]. The superior cerebellar and anterior inferior cerebellar artery supplies MCP.

In our case, given the location of acute ischemic stroke, i.e. medial aspect of MCP, the vertical nystagmus...
is likely related to fibers traversing through it. The MCP carries information to and from cerebellum (between flocculus and nucleus reticularis tegmenti pontes), which contains vertical pursuit signals encoded with a torsional component [14, 15].

Per Dr Cogan, vertical eye movement disorders can be seen with lesions of anterior vermis, middle vermis, roof nuclei and ocular motor centers located in the floor of the fourth ventricle. The neocerebellum i.e., pontocerebellum contains the decline, folium and tuber which are parts of the midline cerebellar vermis. These tracts then extend into lateral cerebellar hemisphere. A lesion to the roof of the fourth ventricle will often result in skew deviation, as can been seen on the MRI images [2–6].

**DISCUSSION**

Ocular findings are common in cerebellar disease in association with brainstem involvement; however, those that are isolated due to purely cerebellar involvement are not common [3]. The classic ocular manifestation of cerebellar disease is nystagmus, skew deviation, dysmetria and flutter-like oscillations of the eye. Nystagmus is often seen in acute cerebellar disease but is not uncommon in chronic conditions affecting cerebellum [3]. Horizontal nystagmus is more common than vertical or rotatory nystagmus [3]. Vertical nystagmus is greatest when gaze is directed upward. It is associated with jerky rhythmic eye movements [7] and gross weakness of conjugate gaze to the side of lesion [8, 9]. Nystagmus due to lesions of the vermis is usually vertical but less conspicuous [3]. Upbeat nystagmus from a lesion in brainstem usually involves the ventral tegmental tract in the pons and caudal medulla [10]. The ventral tegmental tract originates from superior vestibular nucleus and courses through ventral pons and transmits excitatory upward vestibular signals to the oculomotor nerve nucleus [10]. Gaze-evoked upbeat nystagmus localizes to interstitial nucleus of Cajal in upper midbrain and occasionally to the nucleus intercalatus of Staderini [11, 12].

The nucleus prepositus hypoglossi, medial vestibular nuclei, interstitial nucleus of Cajal and vestibulocerebellum are important for holding the eyes steady in eccentric gaze and form the neural integrator for eye movements [13]. Damage to any of these structures could potentially result in an upbeat or downbeat vertical nystagmus. Diseases involving bilateral middle cerebellar peduncle have been described, for example, degenerative diseases, metabolic diseases (adrenoleukodystrophy, Wilson disease, hypoglycemic coma), neoplasms (brainstem glioma, meningeal carcinomatosis), cerebrovascular disease due to AICA infarction, hypertensive encephalopathy and demyelinating disorders [16].

**CONCLUSION**

A similar case has not been reported before based on our literature search. We present an interesting case of vertical upbeat nystagmus in a patient with isolated middle cerebellar peduncle acute ischemic stroke.

**Keywords:** Brainstem, Cerebellar disease, Erebellar pe- duncle stroke, Middle cerebellar peduncle, Vertical up- beat nystagmus

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**How to cite this article**


**Article ID:** 100001VNP01KD2016

**doi:** 10.5348/VNP01-2016-1-CV-1

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**Author Contributions**

Kinjal Desai – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Thomas Bullock – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Rahul Damani – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Eric Bershad – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

**Guarantor**
The corresponding author is the guarantor of submission.

**Conflict of Interest**

Authors declare no conflict of interest.

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REFERENCES


ABOUT THE AUTHORS


Kinjal Desai is a Neurocritical Care Senior Fellow at Baylor College of Medicine in the Department of Vascular Neurology and Neurocritical Care in Houston, Texas, USA. He earned undergraduate Medical Degree MBBS from India at Smt. NHL Municipal Medical College and Residency Training from University of Mississippi Medical Center in Jackson, MS after a Graduate Degree in MPH from University of Southern Mississippi in Hattiesburg, MS. He has published multiple posters, given multiple oral presentations and has worked on book chapters, peer review journals including publications. His research interests include status epilepticus, neuroradiology, stroke and bedside ultrasound. He intends to pursue further training to complete his Doctorate in Public Health upon completion of his neurocritical care training.
E-mail: drkinjal.desai@gmail.com

Thomas Bullock is a faculty in Baylor College of Medicine, Department of Neurology.

Rahul Damani is a faculty in Baylor College of Medicine, Division of Neurocritical Care.
Eric Bershad is a faculty in Baylor College of Medicine, Division of Neurocritical Care.