Isolated Upgaze Palsy in a Patient with Vertebrobasilar Artery Dolichoectasia; a Case Report

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Purpose: To report isolated upgaze palsy in a patient with a dolichoectatic vertebrobasilar artery.

Case Report: We report a 48-year-old man who showed upgaze palsy and convergence insufficiency. The left vertebral artery and basilar artery were shown to be greatly expanded, elongated and tortuous in cranial magnetic resonance imaging (MRI). The vertebrobasilar artery runs along the sulcus basilaris superior to the pontomesencephalic junction.

Conclusion: A dolichoectatic basilar artery may result in compression of midbrain structures related to vertical gaze.

Keywords: Vertical Gaze Palsy; Dolichoectasia; Midbrain; Magnetic Resonance Imaging

INTRODUCTION

Disorders of ocular motility are caused by damage to extraocular muscles, the cranial nerves supplying the muscles, or the neural pathways that control these nerves.¹ Vertical ocular motility disorders may be congenital or acquired in origin.² Bilateral vertical gaze palsy is a rare finding and may be caused by damage to midbrain structures such as the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), posterior commissure (PC), or the interstitial nucleus of Cajal (INC).³

Abnormal enlargement and elongation (dolichoectasia) of the vertebrobasilar artery rarely affects the midbrain. This condition may sometimes cause symptoms related to ischemia or compression. Compression by a dolichoectatic basilar artery has been associated with oculomotor nerve paresis.⁴,⁵

Herein, we report a patient with upgaze palsy and convergence insufficiency due to a dolichoectatic vertebrobasilar artery.

CASE REPORT

A 48-year-old man presented with difficulty in reading since three months ago. The patient was on antihypertensive medications for 5 years, taking them regularly. On ophthalmic examination, best corrected visual acuity was 20/20 and intraocular pressure was 14 mmHg in both eyes. The eyelids were symmetrical, the cornea was intact, the pupils were round and isocoric, and the lens was clear. Pupillary light reaction (direct and indirect) and fundus examination were normal bilaterally.
In primary position, the right and left eyes were normally located. His eye movements were as follows: upward gaze was markedly restricted; however, downward, left, right and forward gazes were normal (Figure 1). In addition he had convergence insufficiency. Nystagmus and ptosis were not present. The vestibulo-ocular reflex was preserved in the horizontal plane, but absent in the vertical plane during smooth pursuit. When vertical doll’s head maneuver was performed, an absence of smooth pursuit was detected.

No abnormalities were observed during general neurologic examination. Motor examination was normal. No signs of tremor, cogwheel, or rigidity were observed. Gait was normal with no hypokinesia. Blood tests such as complete blood counts, measurement of glucose levels, cholesterol levels and thyroid function tests were also within normal limits.

Magnetic resonance imaging (MRI) revealed a hypoplastic right vertebral artery while the left vertebral artery was greatly expanded, elongated and tortuous (Figures 2A and E). Mechanical compression of the pons by the left vertebral artery was noted.

**Figure 1.** Photographs of the patient in different gaze positions: A, Convergence; B, Upward gaze; C, Primary position; D, Downward gaze; E, Left gaze; F, Right gaze.

**Figure 2.** A, Magnetic resonance imaging (MRI) revealed a hypoplastic right vertebral artery and a dilated left vertebral artery (white arrows). B, Note the dilated vertebrobasilar artery, its elongation and association superiorly and inferiorly to the pons. Also note the hypoplastic right vertebral artery. C, Expanded basilar artery. D, MRI showing the basilar artery apex, left PCA, left SCA. E, Expanded basilar artery. F, MR angiography delineates the left vertebral and basilar arteries.

PCA, posterior cerebral artery; SCA, superior cerebellar artery
artery was shown but findings of the IX, X, XI, and XII cranial nerves were not determined (Figure 2B). The vertebrobasilar artery runs along the sulcus basilaris superiorly to the pontomesencephalic junction (Figure 2C). There was no aneurysm at the origin of the posterior cerebral artery (PCA) and superior cerebellar artery (SCA) and this was also confirmed by MR angiography (Figures 2D and F).

**DISCUSSION**

Herein, we describe a 48-year-old man who demonstrated no neurological abnormalities except for upward gaze palsy consisting of complete bilateral loss of upward saccades, smooth pursuit, vestibular eye movements and convergence insufficiency.

The vertical gaze center is thought to be located in the midbrain. The INC, riMLF and subnucleus of the third cranial nerve reside in the midbrain and are responsible for vertical gaze. INC and riMLF communicate via the PC with the nucleus on the other side. While fibers subserving upward gaze cross in the PC, downward gaze fibers directly go to the third cranial nerve. Vertical gaze palsies have been described in many studies and upgaze or downgaze palsies have been related to unilateral or bilateral ischemic midbrain lesions.

Even though an ischemic lesion was considered at initial presentation, repeat MRI disclosed no evidence of ischemia. In our patient, mechanical pressure due to the dolichoectatic basilar artery might have affected the PC and crossing fibers. This explains why upgaze was restricted while downgaze was preserved. The presence of convergence insufficiency, which is detected with lesions of the same region, further supports this notion.

Lesions that cause bilateral oculomotor palsy are most often located in the mesencephalon and mesopontine junction, areas of the oculomotor nuclei and third nerve roots. Lesions of the third nerve nucleus and its subnuclei are rare. The superior rectus muscle subnucleus is generally affected bilaterally, although it may be involved contralaterally and spared ipsilaterally.

The prevalence of dolichoectasia increases with age, and the condition is associated with cardiovascular risk factors. Basilar dolichoectasia is a condition in which pathological elongation and dilatation of the basilar artery is present. In our patient the ectatic artery was most likely the precipitating factor for compression of the midbrain. In our case there was no evidence of neurologic deficits other than upgaze palsy and convergence insufficiency. Accompanying neurological signs, ptosis, or abnormal pupil reactions were not present in this patient. Brain MRI, clearly demonstrated close contact between the ectatic basilar artery and the pons in the absence of ischemic findings. There was no aneurysm at the origin of the PCA and SCA which was also verified by MR angiography. We were able to dismiss diagnoses such as progressive supranuclear palsy, cortical basal degeneration, and Lewy body disease due to the fact that the rest of the neurologic and mental examination were normal.

**Conflicts of Interest**

None.

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