Clinical Case Report

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Convergent strabismus fixus after bilateral abducens nerve palsies due to aneurysms

A case report

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Abstract

Rationale: Convergent strabismus fixus is an ocular motor abnormality in which the eye is fixed in adduction. This condition is mostly associated with high myopia and is caused by a displacement of the extraocular muscles. We report a nonmyopic woman with convergent strabismus fixus due to aneurysms.

Patient concerns: A 79-year-old woman complained of progressive worsening of esotropia about 50 years prior to her visit. Neuroimaging showed that the eye was not dislocated, and the extraocular muscles were not displaced. However, aneurysms were found bilaterally from the intracavernous carotid arteries and the location was on both abducens nerves.

Diagnoses: Chronic bilateral abducens nerve palsies due to aneurysms.

Interventions: Endovascular treatment was successfully performed for the aneurysms.

Outcomes: Convergent strabismus fixus still remained.

Lessons: Chronic abducens nerve palsies may develop to nonmyopic convergent strabismus fixus without displacement of extraocular muscles, and mass lesions in the brain including aneurysms should be ruled out when orbital MRI cannot explain the condition.

Abbreviations: CT = computed tomography, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging, OD = oculus dexter, OS = oculus sinister, OU = oculi uterque, STIR = short inversion time inversion recovery.

Keywords: abducens nerve palsy, convergent strabismus fixus, dislocation of eyeball, displacement of lateral rectus muscle, internal carotid artery aneurysm

1. Introduction

Convergent strabismus fixus is an ocular motor abnormality in which the eye is fixed in adduction.[1] This condition is mostly associated with high myopia and is caused by a displacement of the extraocular muscles.[2,3] Typically, the lateral rectus muscle is displaced inferiorly and the superior rectus muscle is displaced medially.[4] The eye is then dislocated between these muscles[5] leading to the globe being fixed at an esotropic and hypotropic position.

We report an unusual case of acquired convergent strabismus fixus that developed after bilateral chronic abducens nerve palsy. The axial length was not elongated but the abduction movements were almost completely absent bilaterally. Orbital MRI did not show displacements of the superior or lateral rectus muscles. However, unruptured aneurysms of the internal carotid arteries were found along the course of both abducens nerves.

2. Case report

This is a retrospective case report and approval of the Ethics Committee of Osaka Medical College does not deem necessary. However, the study was performed in accordance with the tenets set forth in the Declaration of Helsinki, and informed written consent was obtained from the patient for the publication.

A 79-year-old woman was referred to our hospital to determine whether strabismus surgery of her large angle esotropia would be beneficial. She complained of a progressive worsening of her esotropia that began about 50 years prior to our examination. An esotropia of about 30° was present in the primary position, and her right eye was fixed in an extremely adducted position in horizontal fields of gaze (Fig. 1A). The left eye did not move beyond the midline. The eye positions in the 9 fields of gaze are shown in Figure 1B. Unlike typical cases of convergent strabismus fixus, supraduction movement was more impaired in the left eye.

The axial lengths were 24.13 OD and 23.51 mm OS which are comparable to the average length of 23.4 ± 1.3 mm in elderly Japanese women.[6] Cataract surgery had been performed on the left eye 4 years prior to her visit, and her best-corrected visual acuity and refractive error were 20/100 with X S-1.25 D OD and 20/25 with X S-0.75 D OS. Pathological alterations associated with high myopia were not present in her fundus.

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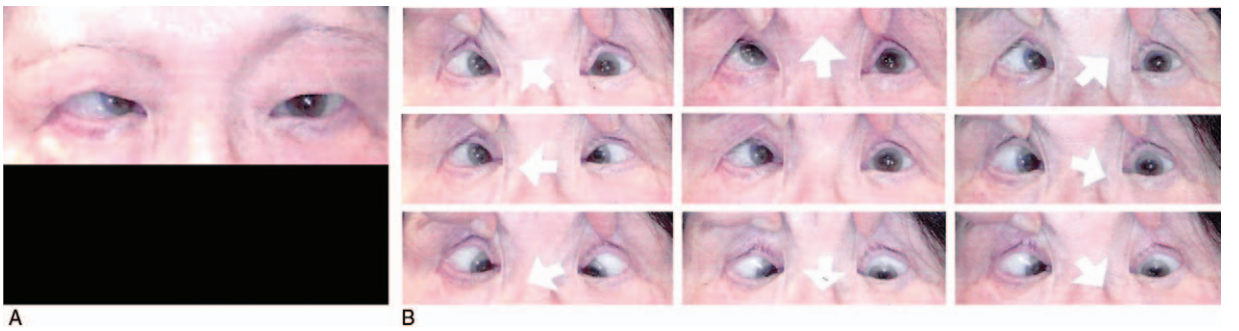

Figure 1. Eye positions of a patient with convergent esotropia fixus. (A) Eye position in the primary position. Right eye is fixed at an extremely adducted position. Ptosis is not present on either side. (B) Eye positions in nine fields of gaze. The right eye is fixed at an adducted position and the left eye does not move beyond the midline in horizontal gazes. Supraduction movement is also impaired bilaterally but more in the left eye. Right hypertropia is present.

Forced duction test was positive in the abduction directions in both eyes with stronger resistance in the right, and lateral movement of the right eye was restricted to the midline. Ptosis was absent bilaterally (Fig. 1A), and it was not induced by fatigue on sustained upward gaze. The pupil diameter in dim lighting was 4.5 mm OU. Her pupils reacted well to light stimuli, and a relative afferent pupillary defect was not present. Other neurological deficits were absent.

Her ocular signs corresponded well to the convergent strabismus fixus, and old photographs indicated that the condition was not congenital. She reported that her strabismus progressed after she had a traffic accident 49 years earlier. She did not report other past histories of ocular diseases or family histories of strabismus.

Neuroimaging showed that the eye was not dislocated, and an orbital fracture was not present. The superior rectus and lateral rectus muscles were not displaced (Fig. 2A). The angle formed by

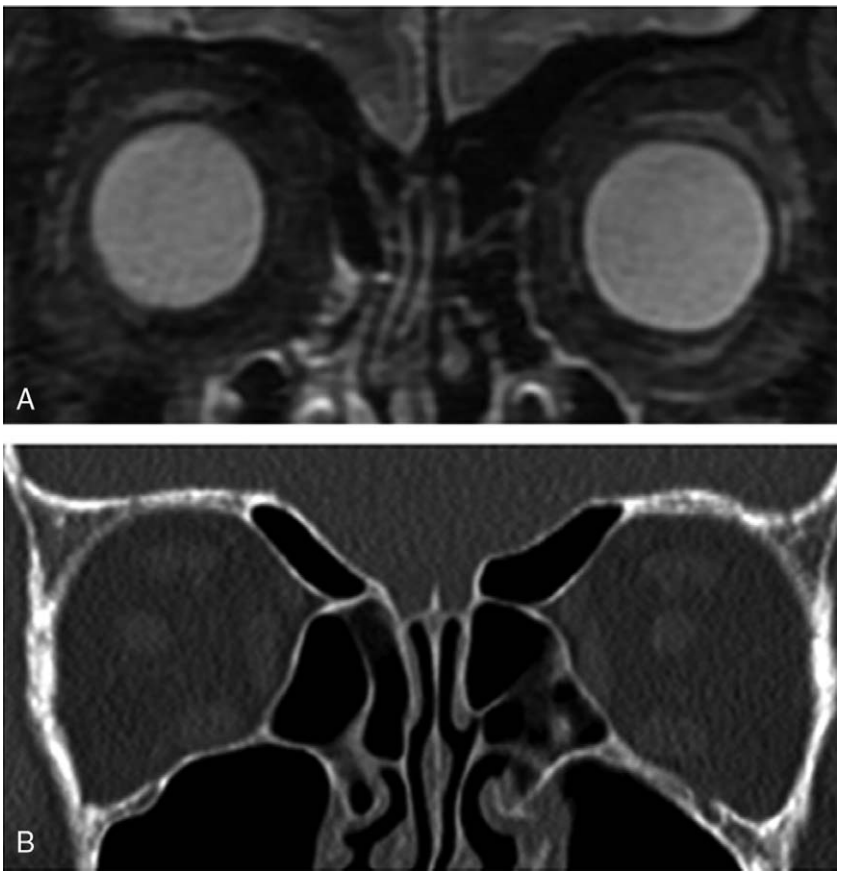

Figure 2. Orbital neuroimaging of a patient with convergent esotropia fixus. (A) STIR image of orbital MRI coronal section. Neither an inferior displacement of lateral rectus muscle nor an extrusion of the eye from the space between superior and lateral rectus muscles is present. Images are taken at about 6 mm anterior to the globe-optic nerve junction. (B) Orbital CT scan indicates there is no orbital fracture. CT = computed tomography, MRI = magnetic resonance imaging, STIR = short inversion time inversion recovery.

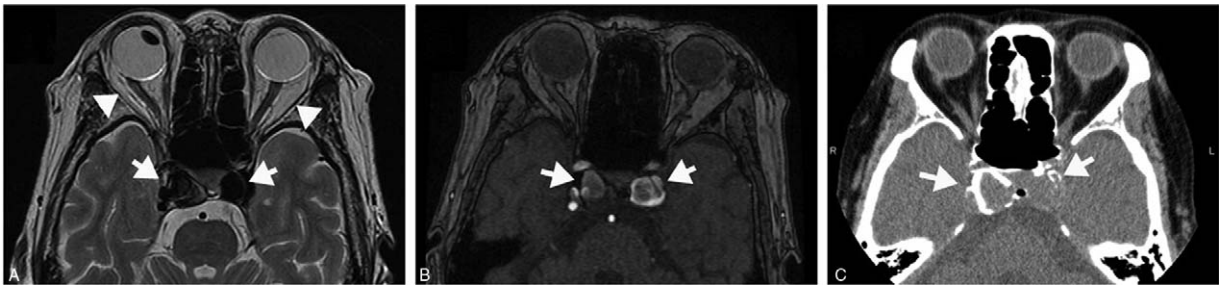

Figure 3. Axial images of MRI, MRA and CT. (A) Lateral rectus muscles (arrowheads) are thin compared to medial rectus muscles indicating atrophic changes. There are round shaped lesions with signal loss just ventral to the pons bilaterally (arrows). (B) MRA image suggests that these lesions are aneurysms, and the signal loss is due to a flow void. Each aneurysm is around 13 mm in diameter. (C) Calcification is present in the aneurysmal walls that is more intense on the right side (arrow). CT = computed tomography, MRA = magnetic resonance angiography, MRI = magnetic resonance imaging.

the centroids of the lateral rectus muscle, superior rectus muscle and the centroid of the globe, indicators of displacement of these muscles and dislocation of the globe,[5,7] was not enlarged. An orbital fracture was not recognized in the CT examination (Fig. 2B).

Axial T2-weighted MRI image showed a signal loss, a flow void sign, just ventral to the pons on both sides (Fig. 3A). Time-of-flight MRA revealed the presence of aneurysms (Fig. 3B). The aneurysms protruded from the intracavernous carotid arteries and the location of the aneurysms was on both abducens nerves (Fig. 3B). The lateral muscles were very thin and atrophic on both sides (Fig. 3A), and the walls of the aneurysms were calcified (Fig. 3C). She was transferred to the Department of Neuro-Surgery, and endovascular treatment was successfully performed for the aneurysms without significant complications. However, her ocular movements and large angle esotropia did not change 6 months after the surgery.

3. Discussion

We report our findings in an elderly nonmyopic woman with convergent strabismus fixus and bilateral unruptured aneurysms in both abducens nerves. In addition, both lateral rectus muscles were very thin. The exact onset of the esotropia was not clear but she reported of progressive changes of her strabismus. The atrophic changes in the lateral muscles supported the idea that the palsies had persisted for a long time.

Congenital fibrosis may occur in the extraocular muscles, often the medial rectus muscle, causing convergent strabismus fixus.[8] However, this syndrome is hereditary, nonprogressive, and is often accompanied by ptosis. Our case reported that the esotropia was progressive, ptosis was not present, and there was no family history of strabismus. We concluded that this case was not likely to be this syndrome.

It has been suggested that a degeneration of the lateral and superior rectus band occurs in convergent strabismus fixus in highly myopic eyes that causes an inferior displacement of the lateral rectus muscle, medial displacement of the superior rectus muscle, and dislocation of the eye.[4,9] Similar changes can also occur during the aging process that produce the sagging eye syndrome that is usually manifested as mild esotropia with a limitation of supraduction movement in elderly individuals.[10] Degeneration of the lateral and superior rectus band may even cause convergent strabismus fixus without high myopia.[9,11] However, displacement of lateral rectus muscle and dislocation of the eyeball were not present in our case.

Villaseca postulated that fibrosis of the medial rectus muscle can develop following a chronic lateral rectus muscle palsy that leads to a contracture of medial rectus and convergent strabismus fixus.[12] Thus, we had suspected a contracture of the medial rectus muscle had progressed during the course of chronic abducens nerve palsy.[13] Calcification of the aneurysmal walls suggested that the aneurysms were present for a long time. Greater calcification of the right aneurysms also suggested that the right abducens nerve palsy occurred earlier leading to the right convergent esotropia fixus. However, we did not perform strabismus surgery and could not determine the condition of the extraocular muscles, and this is a limitation of this report.

In conclusion, convergent strabismus fixus can occur after chronic abducens nerve palsy,[12] and physicians should be aware that chronic abducens nerve palsy can cause this rare condition. However, mass lesions in the brain including aneurysms should be ruled out when orbital MRI cannot explain the condition.

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Writing – review & editing: Teruyo Kida, Jun Sugasawa, Tsunehiko Ikeda.

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