Jpn J Ophthalmol 2005;49:246–252
DOI 10.1007/s10384-004-0184-6

CLINICAL INVESTIGATION

Incarceration of the Inferior Oblique Muscle Branch of the Oculomotor Nerve in Two Cases of Orbital Floor Trapdoor Fracture

Hirohiko Kakizaki[1,2], Masahiro Zako[2], Masayoshi Iwaki[2], Hidenori Mito[3], and Nobutada Katori[1]

[1] Department of Oculoplastic and Orbital Surgery, Seirei Hamamatsu General Hospital, Hamamatsu, Japan; [2] Department of Ophthalmology, Aichi Medical University, Aichi, Japan; [3] Ide Eye Hospital, Yamagata, Japan

Abstract

Background: Incarceration of the inferior oblique muscle (IO) branch of the oculomotor nerve may occur in cases of orbital floor trapdoor fracture.

Cases: Two orbital floor trapdoor fracture cases, with lesions located just outside of the inferior rectus muscle but without its incarceration, were examined pre- and postoperatively for visual acuity, intraocular details, the nine diagnostic ocular positions of gaze, binocular single vision field with the Hess chart, and by computed tomography (CT). One case was also examined by magnetic resonance imaging (MRI; T1-weighted images). A forced duction test was conducted intraoperatively.

Observations: Each case presented good visual acuity and neither globe showed any injury. Motility disturbance of the IO was shown in each case by binocular single vision field testing and the Hess chart. The possibility of the incarceration of the IO branch of the oculomotor nerve, which runs from the incarcerated lesion to the superior belly of the IO, in an orbital floor trapdoor fracture was shown on CT and MRI. Intraoperative forced duction testing revealed a restriction due to the incarceration of the connective tissue septa.

Conclusions: As inferred from the CT and MRI analyses conducted in this study, IO palsy may be one of the causes of ocular motility disturbance of the IO in an orbital floor trapdoor fracture, in addition to the ocular motility disturbance due to the connective tissue septa. **Jpn J Ophthalmol** 2005;49:246–252

Key Words: computed tomography, inferior oblique muscle, magnetic resonance imaging, orbital floor trapdoor fracture, palsy

Introduction

Young people are particularly susceptible to orbital floor trapdoor fracture.[1–5] After such an injury, an ocular movement disorder can occur owing to the entrapment of muscles or connective tissue septa,[6,7] and to swelling and hemorrhage.[6,7] When the inferior rectus muscle (IR) is incarcerated among the fractured pieces, muscular necrosis may arise by ischemia without emergent release.[1,4,5] In the chronic phase, in addition to the muscular weakness of the IR, fibrosis may also occur from inflammation, causing permanent ocular movement disorders.[1,3,6,7] For this reason, necrosis caused by incarceration of the IR and ocular movement disorders caused by fibrosis have attracted attention in orbital floor trapdoor fractures. However, the inferior oblique muscle (IO) branch of the oculomotor nerve runs immediately outside the IR,[8] and in part accords with the most favored site for orbital floor trapdoor fracture.[9–12] IO palsy often occurs in such lesions;[12] however, to date, movement restriction of the IO in an orbital floor trapdoor fracture has not received much attention. This might be because this type of fracture often presents with severe

Received: May 24, 2004 / Accepted: September 3, 2004
Correspondence and reprint requests to: Hirohiko Kakizaki, Department of Ophthalmology, Aichi Medical University, Nagakute-cho, Aichi-gun, Aichi 480-1195, Japan
e-mail: cosme@d1.dion.ne.jp

ocular supraduction disturbances,[1–4] and so attention is focused, in most cases, on the movement disorders of the IR. Hence, incarceration of the IO branch has not been properly investigated. When incarceration occurs outside of the IR, and does not include the IR, ocular movement disorders can occur because of the incarceration of the IO branch in addition to that of the connective tissue septa.

In this study, using computed tomography (CT) and magnetic resonance imaging (MRI), we demonstrated the possibility of the incarceration of the IO branch in two cases of orbital floor trapdoor fractures.

Methods

We present two cases, with lesions located just outside the IR but without its incarceration, showing movement restriction of the IO in an orbital floor trapdoor fracture. Each patient was examined pre- and postoperatively for visual acuity, intraocular details, the nine diagnostic positions of gaze, by binocular single vision field testing and the Hess chart, and by CT. A forced duction test was conducted intraoperatively. The binocular single vision field was measured by Goldmann perimeter with an index of V-4. On CT (GE Medical Systems, Waukeshaw, WI, USA), to get coronal and sagittal views, images were reconstructed after scanning an axial view, sliced parallel to the optic nerve. Slice thickness was 2 mm, half of which overlapped. An MRI examination was also performed in Case 1, with images acquired on a 1.5-T GE SIGNA (GE Medical Systems), using a custom-designed 5-inch (about 13-cm) surface coil. Coronal and sagittal images of T1-weighted spin echo sequences (echo time = 9 ms, repetition time = 500 ms) were obtained. The sagittal images were sliced parallel to the optic nerve; slice thickness was 3 mm, interslice gap 0.5 mm, and the field of view 140 mm × 140 mm with a 256 × 192 matrix.

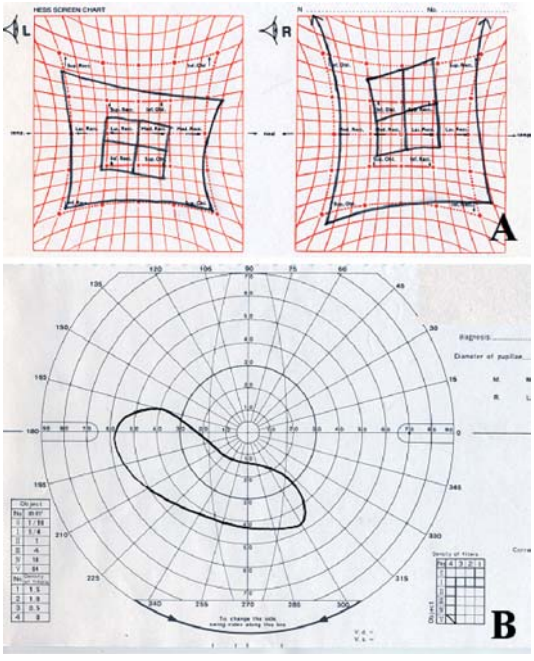

Figure 1A,B. Hess chart and binocular single vision field in Case 1, preoperatively. **A** Hess chart. Supraduction restriction and a motility disturbance of the inferior oblique muscle are observed in the left eye. **B** Binocular single vision field. Binocular single vision field is obtained only in the left lower area.

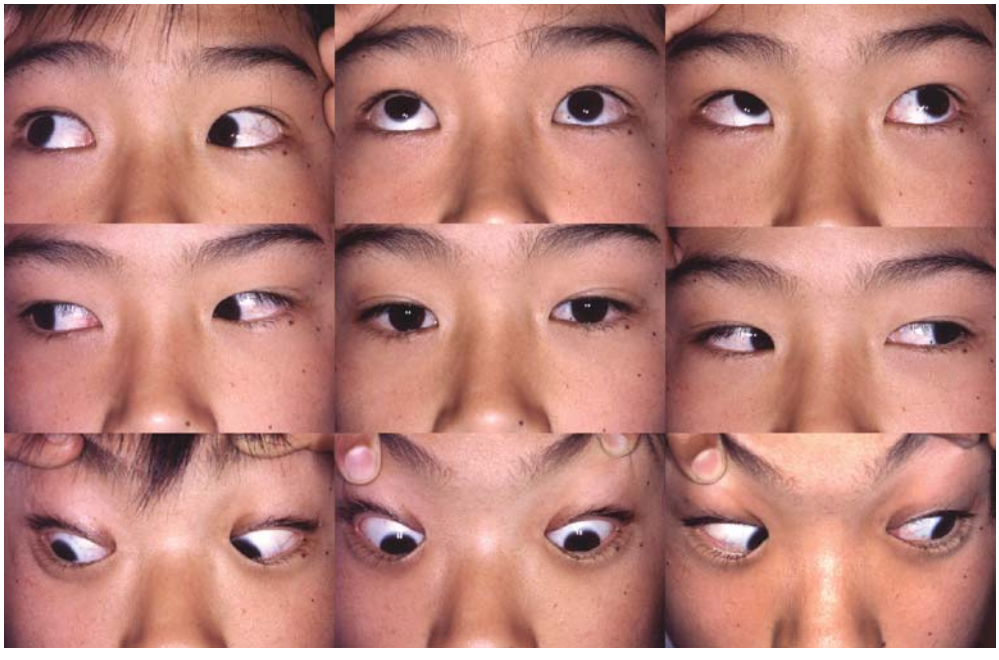

Figure 2. The nine diagnostic positions of gaze in Case 1 preoperatively. Supraduction toward the three different directions is restricted in the left eye.

Jpn J Ophthalmol
Vol 49: 246–252, 2005

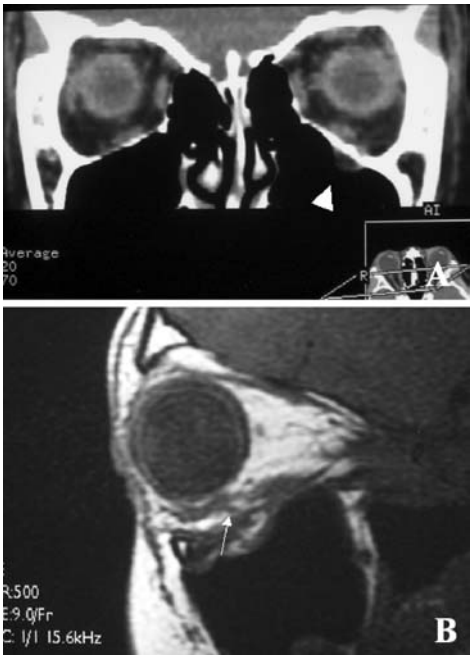

Figure 3A,B. Computed tomography (CT) and magnetic resonance (MR) images in Case 1, preoperatively. **A** Coronal view of CT image. A left orbital floor trapdoor fracture is observed outside of the inferior rectus muscle with a teardrop sign and findings of bony thickening. The inferior rectus muscle is not sandwiched between the fracture pieces. *Arrowhead*, teardrop sign **B** Sagittal view of MR image. The inferior oblique muscle branch of the oculomotor nerve that goes to the myoneural junction from the fracture lesion is identified. *Arrow*, inferior oblique muscle branch of the oculomotor nerve.

Case Reports

Case 1

A 12-year-old boy suffered a blow to his left eye while playing soccer. Immediately after the injury, he noticed diplopia and nausea. The same day, he had a medical examination by a neurosurgeon, who noted that there was no abnormality in the brain; but the symptoms persisted. The following day, the boy had another medical examination, this time by an ophthalmologist. Since an inferior orbital fracture was observed in his left eye on CT images, the Oculoplastic and Orbital Clinic at Seirei Hamamatsu General Hospital was consulted with a view to an operation. The boy's head position was found to be inclined to the left, with his chin up. Visual acuity was 20/20 (n.c.), OU, and neither globe showed evidence of rupture or any other traumatic breach of scleral integrity. Bilateral pupils were round and appeared the same, and the light reflex was both prompt and complete. A supraduction disturbance of the left eye and a motility disturbance of the left IO were observed on Hess chart examination (Fig. 1A). Diplopia, especially in the upper area including the primary position, was observed in the binocular single vision field (Fig. 1B), in particular in the right upper diplopia wide field. Supraduction toward the three different directions of the left eye was limited in the nine diagnostic positions of gaze (Fig. 2). By CT, an orbital floor trapdoor fracture was observed outside of the IR in the left eye, with a teardrop sign and findings of bony thickening, but the IR was not sandwiched between the fracture pieces (Fig. 3A). However, by CT we could not visualize the position of the IO branch, which was identified by MRI (sagittal view; Fig. 3B) as going to the superior side of the IO belly from the fracture lesion. Reduction of the left orbital floor was performed 8 days after the injury. An intraoperative forced duction test revealed a moderate restriction. Positive findings showing the incarceration of the IO branch could not be obtained intraoperatively because of the periosteum and the orbital fat. Three months postoperatively, although a slight restriction of supraduction of the left eye was observed from a Hess chart (Fig. 4A), the binocular single vision field was much improved (Fig. 4B). Head position and the nine diagnostic positions of gaze became normal. The left orbital floor was well reduced (Fig. 5A), and, in addition, the IO branch could be observed in sagittal magnetic resonance (MR) images, without incarceration in the floor (Fig. 5B).

Case 2

A 20-year-old man suffered a heavy elbow blow to his left eye. Immediately after the injury he noticed diplopia and nausea. That day, he had an examination by an ophthalmologist; an orbital floor fracture was observed by CT. Diplopia was monitored for some days, but did not improve; thus, the Oculoplastic and Orbital Clinic at Seirei Hamamatsu General Hospital was consulted 10 days after the injury. The patient's head position was found to be slightly inclined to the left, and visual acuity was 20/10 (n.c.), OU, but ocular examination showed no evidence of rupture of the globe or any other traumatic breach of scleral integrity. Bilateral pupils were round and appeared the same, and the light reflex was both prompt and complete. A supraduction disturbance of the left eye and a motility disturbance of the left IO were observed on a Hess chart (Fig. 6A). The diplopia was centered in and around the right upper area and was observed in the binocular single vision field (Fig. 6B). Supraduction in the adduction of the left eye was limited in the nine diagnostic positions of gaze, and abduction was observed on upgaze (Fig. 7). An orbital floor trapdoor fracture was observed outside of the IR with a teardrop sign and findings of bony thickening in the coronal view on CT images (Fig. 8A). However, the IR was not sandwiched between the fracture pieces. The IO branch going to the superior side of the IO belly from the fracture lesion was identified on sagittal view CT images (Fig. 8B). A medial orbital wall fracture was also observed in the left eye; however, since it was thought that this fracture had not contributed to the ocular movement disorders in the image findings and other examinations, we did not reduce the medial orbital wall. Reduction of the orbital floor was performed in the left eye 16 days after injury. An intraoperative forced duction test revealed a slight restriction. Positive

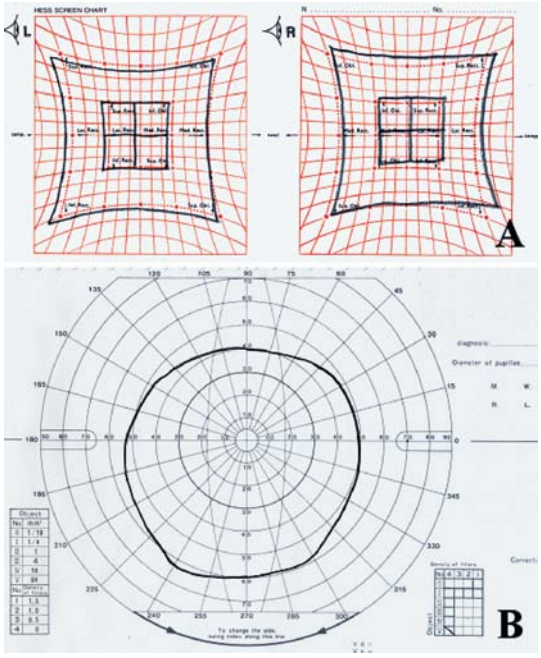

Figure 4A,B. Hess chart and the binocular single vision field in Case 1, postoperatively. **A** Hess chart. A slight restriction of supraduction of the left eye is observed. **B** Binocular single vision field. Binocular single vision field is much improved.

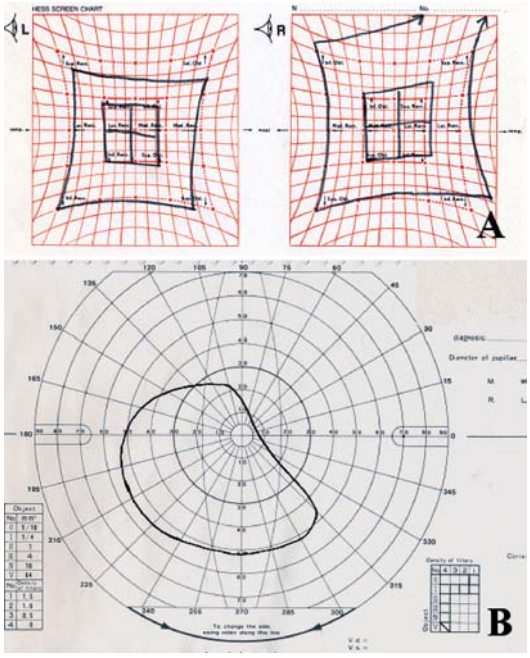

Figure 6A,B. Hess chart and the binocular single vision field in Case 2, preoperatively. **A** Hess chart. Supraduction restriction of the left eye and a motility disturbance of the inferior oblique muscle are observed in the left eye. **B** Binocular single vision field. The binocular single vision field is observed in the left lower area including the primary eye position.

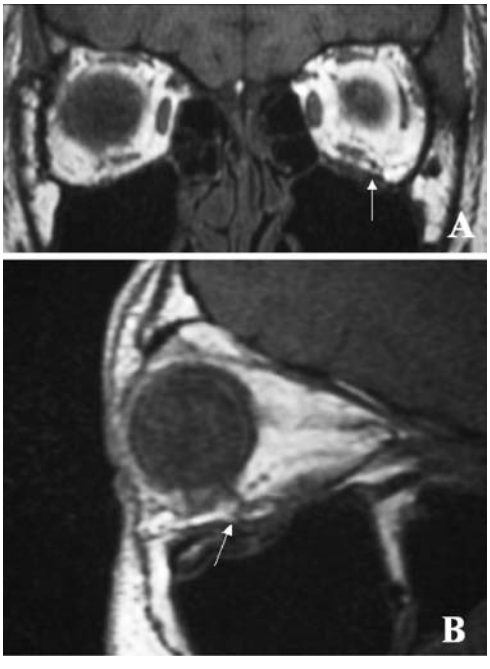

Figure 5A,B. The MR images in Case 1, postoperatively. **A** Coronal view of MR image. The orbital floor is well reduced. The inferior oblique muscle branch of the oculomotor nerve is detected in this image just outside of the inferior rectus muscle. *Arrow*, inferior oblique muscle branch of the oculomotor nerve. **B** Sagittal view of MR image. The inferior oblique muscle branch of the oculomotor nerve is observed in this image, without incarceration in the floor. *Arrow*, inferior oblique muscle branch of the oculomotor nerve

findings showing the incarceration of the IO branch could not be obtained intraoperatively because of the periosteum and the orbital fat. Three months postoperatively, although a slight restriction of supraduction was observed in his left eye on a Hess chart (Fig. 9A), the binocular single vision field was much improved (Fig. 9B). Head position and the nine diagnostic positions of gaze were normal. The orbital floor was well reduced in his left eye (Fig. 10A), but the IO branch could not be observed on sagittal MR images (Fig. 10B).

Discussion

Young people are particularly susceptible to orbital floor trapdoor fractures, a condition that is considered to necessitate an emergency operation.[1–5] In such cases, the IR is usually incarcerated between fracture pieces, and severe ocular movement disorders are often produced.[1–5] As a result of congestion, muscular necrosis can occur.[1,4,5] A typical case is the so-called condition of missing rectus,[13,14] in which the overall width of the muscle is caught among the fracture pieces, and the image of the IR disappears on coronal views in both CT and MRI. Connective tissue septa are then also tightly incarcerated in the fracture lesion.[1,6,7] The inflammation produces fibrosis around the fracture lesion, and, consequently, ocular movement disorders are aggravated.[1,6,7] For this reason, in orbital floor trapdoor

Jpn J Ophthalmol
Vol 49: 246–252, 2005

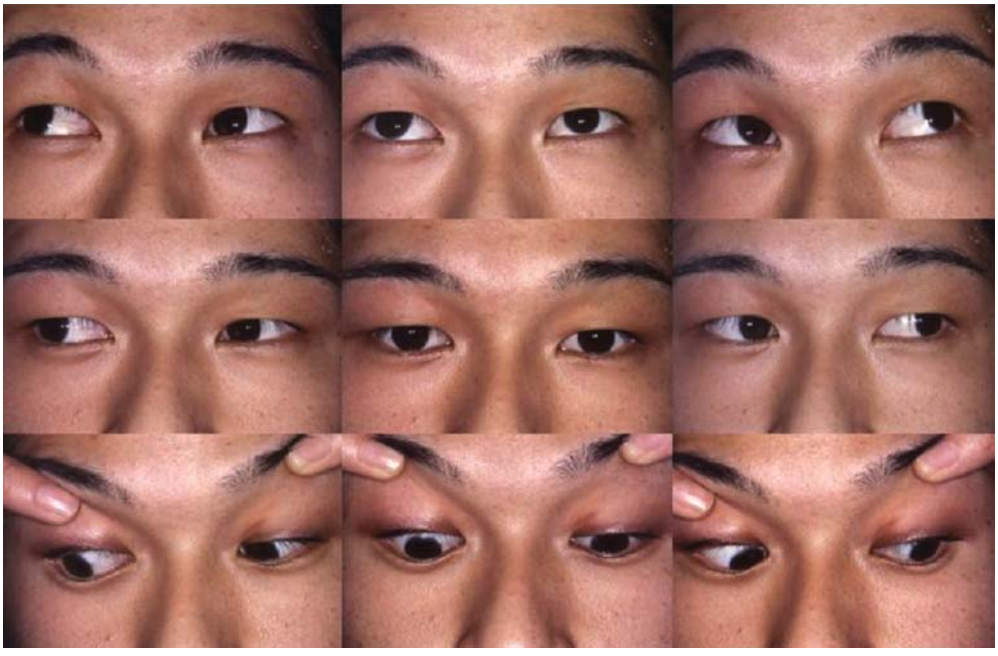

Figure 7. The nine diagnostic positions of gaze in Case 2, preoperatively. Supraduction in adduction of the left eye is restricted.

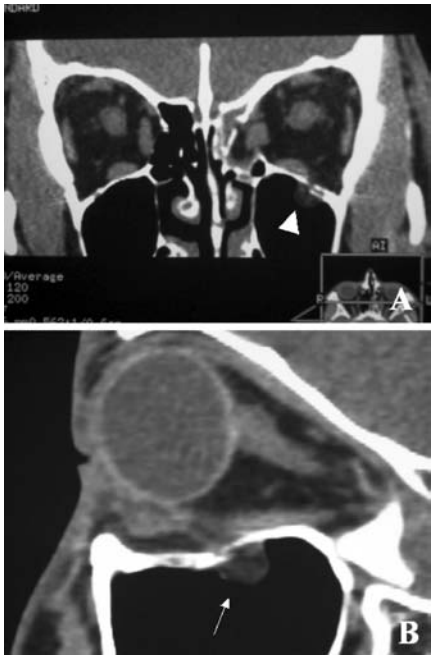

Figure 8A,B. CT and MR images in Case 2, preoperatively. **A** Coronal view of CT image. An orbital floor trapdoor fracture is observed outside of the inferior rectus muscle with a teardrop sign and findings of bony thickening. The inferior rectus muscle is not sandwiched between the fracture pieces. *Arrowhead*, teardrop sign. **B** Sagittal view of CT image. The inferior oblique muscle branch of the oculomotor nerve that goes to the myoneural junction from the fracture lesion is identified. *Arrow*, inferior oblique muscle branch of the oculomotor nerve.

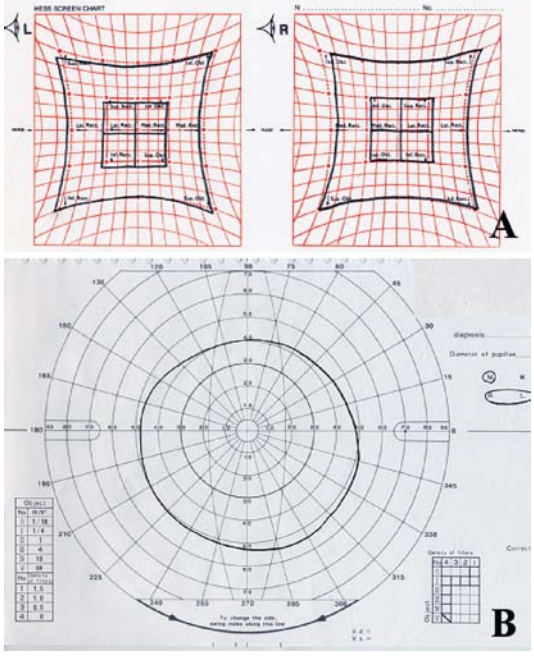

Figure 9A,B. Hess chart and the binocular single vision field in Case 2, postoperatively. **A** Hess chart. A slight restriction of supraduction of the left eye is observed. **B** Binocular single vision field. The binocular single vision field is much improved.

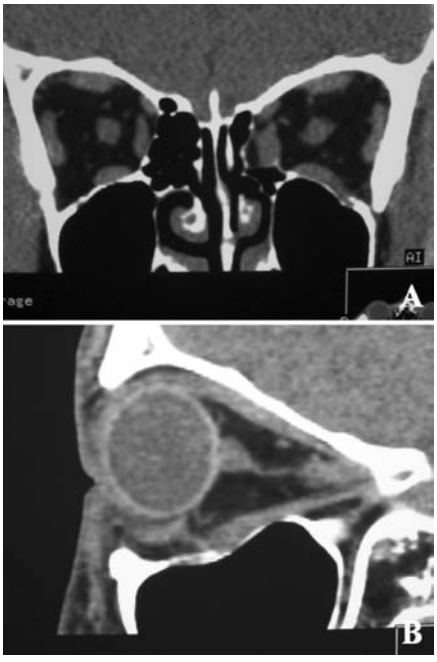

Figure 10A,B. CT and MR images in Case 2, postoperatively. **A** Coronal view of CT image. The orbital floor is well reduced. The inferior oblique muscle branch of the oculomotor nerve cannot be detected in this image because there is little postoperative contrast. **B** Sagittal view of CT image. The inferior oblique muscle branch of the oculomotor nerve cannot be observed on this image because there is little postoperative contrast.

fractures, it is the IR that has received most attention with the IO receiving much less.

The IO branch of the oculomotor nerve runs just outside of the IR,[9,12] and the most common site for orbital floor trapdoor fracture is just inside the infraorbital groove, namely, just outside the IR.[10–13] Thus, in very many cases of orbital floor trapdoor fracture, the IO branch is thought to become incarcerated between the fracture pieces. Iliff et al.[12] reported that owing to the anatomical characteristics of the IO branch, it was difficult for it not to be affected by a contusion injury in which IO palsy could become one of the causes of any ocular movement disorder. The two patients in this study apparently suffered from a contusion caused by the incarceration of the IO branch, which then became one of the causes of the movement restriction of the IO. However, we were not able to obtain positive findings showing the incarceration of the IO branch intraoperatively, because only the periosteum and the orbital fat are extracted from fracture sites in an operation for a blow-out fracture, and generally we cannot see any other orbital contents. Furthermore, a search for the orbit to obtain such positive findings of nerve incarceration would promote cicatrization around the IR and IO; thus, a search for the orbit is most certainly ruled out.

When, with incarceration of the IR, ocular motility disturbances become severe, any movement restriction of the IO may be obscured. However, movement restriction of the IO was seen on Hess charts in the two cases presented here, in images that showed incarceration of the IO branch without incarceration of the IR. In these image findings, as the connective tissue septa were also incarcerated in the fracture site, and taking the findings of the Hess chart into consideration, it is difficult to attribute the cause of the movement restriction of the IO entirely to IO palsy. However, from the findings presented here, IO palsy is considered to have been an cause of these ocular movement disorders. Overaction of the ipsilateral superior oblique muscle was not remarkable, but it is thought to have been caused by the mechanical movement restriction due to the incarceration of the connective tissue septa.

The basis on which we judged the strip of soft tissue structure as the IO branch, and not as the connective tissue septa, is as follows: (1) Sagittal images were sliced in accordance with the course of the IO branch just outside of the IR. The IO branch is around 1 mm in diameter, and it can be identified in the coronal views on CT[14] and MRI (Figs. 3B,8B).[15] It is hard to identify the nerve in the normal condition in sagittal view images. However, because the nerve was swollen owing to a blood flow disorder at this time, we believe we were able to identify it. (2) The myoneural junction of the IO is located on the superior side of the belly of the IO.[7,16,17] This strip of soft tissue structure goes toward that site. (3) If the strip of soft tissue structure were the connective tissue septa, it should go toward the side of the IO, a little lower than the myoneural junction of the IO.[6,7,18] In addition, considering the mechanism of the injury, it is difficult to accept that only one septum would be clearly observed and that the other septa would not also be observed in the area. Finally, (4), although Koornneef et al.[6,7,19] have reported in detail about the connective tissue septa, a large, uniform connective tissue septum in the floor region was not shown in their results.[6,7,19] In addition, Dutton's Atlas does not show a large, uniform connective tissue septum.[18] Septa reconstructed three-dimensionally in the floor region appear as various small pieces, not as uniform or large shapes.[6,7,18,19] Consequently, even if the septa had been sliced in a sagittal section, it would be impossible to describe them as long and thick, like they appeared in the cases presented in this study. For the above reasons, we judged the strip of soft tissue structure seen here as the IO branch, and not as connective tissue septa.

It may be possible to prove IO palsy by observing MR images of muscle contraction and relaxation[20] of the IO, although we did not do this in this study. The IO of the two cases here would probably show less change in muscle thickness during contraction and relaxation than the IO of normal cases.

The parasympathetic nerve controlling the pupil is included in the IO branch,[21] and branches off from it at a site posterior to the ciliary ganglion and then goes anteriorly toward the ganglion.[21] As the fracture lesions in our two cases were located in a part distal from the ciliary ganglion, these cases would not be expected to present with pupillary disorders.

We demonstrated the possibility of incarceration of the IO branch in orbital floor trapdoor fractures. To date, incarceration of the IO branch at a fracture site has not been visualized on images; we believe that this is the first study to show such incarceration of the IO branch using CT and MR images. In an orbital floor trapdoor fracture in which the IR is not incarcerated in the fracture lesion, the IO branch sometimes becomes so incarcerated. In such cases, movement restriction of the IO due to incarceration of the IO branch in the fracture site should be taken into consideration.

References

1. Jordan DR, Allen LH, White J, et al. Intervention within days for some orbital floor fractures: the white-eyed blowout. Ophthal Plast Reconstr Surg 1998;14:379–390.
2. Bansagi ZC, Meyer DR. Internal orbital fracture in the pediatric age group. Ophthalmology 2000;107:829–836.
3. Egbert JE, May K, Kerstern RC, et al. Pediatric orbital floor fracture. Direct extraocular muscle involvement. Ophthalmology 2000;107:1875–1879.
4. Baek SH, Lee EY. Clinical analysis of internal orbital fractures in children. Korean J Ophthalmol 2003;17:44–49.
5. Sires BS, Stanley RB, Levine LM. Oculocardiac reflex caused by orbital floor trapdoor fracture: an indication for urgent repair. Arch Ophthalmol 1998;116:955–956.
6. Koornneef L, Zonneveld FW. Orbital anatomy, the direct scanning of the orbit in three planes and their bearing on the treatment of motility disturbances of the eye after orbital "blow-out" fractures. Acta Morphol Neerl-Scand 1985;23:229–246.
7. Koornneef L. Current concepts on the management of orbital blow-out fractures. Ann Plast Surg 1982;9:185–200.
8. Bedrossian EH Jr. Surgical anatomy of the orbit. In: Della Rocca RC, Bedrossian EH Jr, Arthus BP, editors. Ophthalmic plastic surgery: decision making and techniques. New York: McGraw-Hill; 2002. p. 207–227.
9. Tajima S. The therapy of facial fracture. Tokyo: Kokuseido; 1999. p. 55–66.
10. Jones DEP, Evans JNG. "Blow-out" fractures of the orbit: an investigation into their anatomical basis. J Laryngol 1967;81:1109–1120.
11. Wilkins RB, Havins WE. Current treatment of blow-out fractures. Ophthalmology 1982;89:464–466.
12. Iliff N, Manson PN, Katz J, et al. Mechanism of extraocular muscle injury in orbital fracture. Plast Reconstr Surg 1999;103:787–799.
13. Anda S, Elsas T, Harstad HK. The missing rectus: a CT observation from blow-out fracture of the orbital floor. J Comput Assist Tomogr 1987;5:895–897.
14. Zonneveld FW, Koornneef L, Hillen B, et al. Normal direct multiplanar CT anatomy of the orbit with correlative anatomic cryosections. Radiol Clin North Am 1987;25:381–407.
15. Ettl A, Kramer J, Daxer A, et al. High resolution magnetic resonance imaging of neurovascular orbital anatomy. Ophthalmology 1997;104:869–877.
16. Bower Wachler BS, Holds JB. The missing muscle syndrome in blowout fractures: an indication for urgent surgery. Ophthal Plast Reconstr Surg 1998;14:17–18.
17. Demer JL, Oh SY, Clark RA, et al. Evidence for a pulley of the inferior oblique muscle. Invest Ophthalmol Vis Sci 2003;44:3856–3865.
18. Dutton JJ. Atlas of clinical and surgical orbital anatomy. Philadelphia: WB Saunders; 1994. p. 93–101.
19. Koornneef L. Orbital septa: anatomy and function. Ophthalmology 1979;86:876–880.
20. Kono R, Demer JL. Magnetic resonance imaging of the functional anatomy of the inferior oblique muscle in superior oblique palsy. Ophthalmology 2003;110:1219–1229.
21. Bron AJ, Tripathi RC, Tripathi BJ, editors. Wolff's anatomy of the eye and orbit, 8th ed. Chapter 5: Innervation and nerves of the orbit. London: Chapman & Hall Medical; 1997. p. 178–210.