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Influence of fractures in different inferior orbital wall locations on ocular motility disorders

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Orbital floor fractures affect eye movement, but the underlying mechanism remains unclear. Therefore, the present retrospective study aimed to clinically characterize fractures in different inferior orbital wall locations, and to determine the relationship between fracture type and ocular motility disorder.

All patients with ocular motility disorders caused by orbital floor fractures within 7 days of trauma treated in the Ophthalmology Department of the First Affiliated Hospital of Zhengzhou University from August 2018 to August 2020 were retrospectively assessed. The inclusion criteria were: (1) age ≥12 years; (2) ability to clearly describe diplopia symptoms and cooperation in the forced duction test (FDT); (3) confirmed diagnosis of inferior orbital wall fracture by computed tomography (CT) scanning. Exclusion criteria were: (1) concurrent ocular rupture or severe vision loss; (2) another concurrent orbital wall fracture; (3) a history of ocular trauma. This study was approved by the ethics committee of the First Affiliated Hospital of Zhengzhou University (No. 2020-KY-478); informed consent was waived due to the retrospective nature of the study.

The routine eye examinations included the uncorrected visual acuity and best-corrected visual acuity tests, slit-lamp examination, and ocular fundus and intraocular pressure (IOP) measurements. The ocular motor examination included ductions and versions that were assessed by the same examiner. Ductions were examined with one eye occluded, forcing fixation to the eye being examined. Versions were assessed via eye movements through the nine cardinal positions of the gaze. Restrictive and paralytic eye movement disorders were assessed by the FDT. A positive FDT indicates that the inferior rectus or the inferior rectus and inferior oblique muscles are entrapped in the fracture area. A negative test is indicative of superior rectus paralysis or injured muscle.

Hess screen test was performed for all patients wearing red-green goggles who were required to sit 50 cm from the screen, preferably with the head fixed on a headrest. The examiner marked the positions indicated by the patient on a small card with a reduced copy of the screen.

For diplopia testing, the results were assessed using the following rules: (1) the damaged muscle corresponds to the eye generating an image (or ghost image) situated further from the median horizontal or vertical line; (2) the ghost image is consistently projected toward the paralyzed muscle; homonymous diplopia is reflected by impacted abductor muscles, while heteronymous diplopia results from damaged adductor muscles; and (3) the distance between double images increases as the gaze moves toward the injured muscle. Axial dystopia (enophthalmos or exophthalmos) was determined with respect to the relatively healthy eye on a Hertel/Krahn exophthalmometer. All patients underwent 64-slice spiral CT (Siemens, Munich, Germany) scanning in the horizontal, coronal, and sagittal planes, with a slice thickness of <3 mm. Images were reconstructed using a bone algorithm and a soft-tissue algorithm.

The 32 patients included 26 males and six females, with an average age of 29 years (range, 13–55 years). All patients had corrected visual acuity > 0.3 logarithm of the minimum angle of resolution (logMAR), clear refractive system, normal ocular fundus, and normal IOP. The fractures were caused by car accidents ($n = 14$), violent striking ($n = 13$), and others ($n = 5$).

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Table 1: Clinical manifestations of patients with fractures in different inferior orbital wall locations.

Clinical characteristics	All patients ($n = 32$)	Anterior ($n = 5$)	Posterior ($n = 6$)	Anterior + posterior ($n = 21$)
Factors involved in eye movement disorders				
Restrictive	8 (25.0)	5	0	3 (14.3)
Paralytic	10 (31.2)	0	6	4 (19.0)
Restrictive and paralytic	14 (43.8)	0	0	14 (66.7)
Types of eye movement disorders				
Limited upward movement	7 (21.9)	4	0	3 (14.3)
Limited downward movement	10 (31.2)	0	6	4 (19.0)
Limited vertical movement	15 (46.9)	1	0	14 (66.7)
Diplopia				
Upward gazing	8 (25.0)	5	0	3 (14.3)
Primary position on gaze	5 (15.6)	0	1	4 (19.0)
Downward gazing	19 (59.4)	0	5	14 (66.7)
Enophthalmos	22 (68.8)	0	4	18 (85.7)
Exophthalmos	3 (9.4)	0	1	2 (9.5)
Hypesthesia of trigeminal nerve region	23 (71.9)	3	4	16 (76.2)
Hyphema	2 (6.3)	0	0	2 (9.5)
Blunt inferior rectus muscle belly on CT	8 (25.0)	5	0	3 (14.3)
Subconjunctival hemorrhage	21 (65.6)	4	1	16 (76.2)
Chemosis	15 (46.9)	4	0	11 (52.4)
Swelling of the eyelids	11 (34.4)	0	0	11 (52.4)
Peri-orbital ecchymosis	25 (78.1)	3	5	17 (81.0)
Abnormal pupil reflex (RAPD positive)	4 (12.5)	0	0	4 (19.0)

Data are presented as *n* (%). CT: Computed tomography; RAPD: Relative afferent pupillary defect.

There were five patients with anterior segment fractures. In these patients, CT showed anterior orbital floor fractures with intact posterior segment and incarceration of the inferior rectus muscle or the surrounding soft tissue. There were six patients with posterior segment fractures. In these cases, CT showed posterior orbital floor fractures with intact anterior segment and orbital contents partially trapped in the fracture orifice. There were 21 cases of combined anterior and posterior segment fractures. In these patients, CT showed a large collapsed area of the orbital floor and substantial amounts of orbital contents trapped.

In the five cases with anterior segment fractures, the ocular motor examination showed obvious restriction on supraduction and slight restriction on infraduction. A positive FDT reflected impeded passive upward displacement of the eyeball, whose downward motility remained normal. The Hess screen test showed an obvious contraction at the top graph and a slight contraction at the bottom graph. The ocular motility disorder was of restrictive nature in all patients. In this group, four (80.0%) and one (20.0%) had limited upward and limited vertical movements, respectively. All had upward gazing deficiency.

In the six posterior segment fracture cases, the ocular motility disorder was paralytic. Positive FDT results were obtained in the vertical movement. All six (100%) patients showed limited downward movements. There was no obvious restriction on supraduction and infraduction. A total of 5 (83.3%) cases had downward gazing deficiency. The Hess screen test showed obvious contraction at the bottom graphs.

In the 21 cases with combined anterior and posterior segment fractures, there were 3, 4, and 14 patients with restrictive, paralytic, and combined restrictive and paralytic ocular motility disorder, respectively. The ocular motor examination showed obvious restriction on both supraduction and infraduction. Most cases had enophthalmos ($n = 18$), hypesthesia of the trigeminal nerve region ($n = 16$), subconjunctival hemorrhage ($n = 16$) and periorbital ecchymosis ($n = 17$). The detailed data for all groups are provided in Table 1.

This study demonstrated that fractures in various inferior orbital wall locations cause ocular motility disorders of distinct features. As shown above, there were five cases with anterior segment fractures involving restrictive eye movement disorder. The main characteristic of this ailment is that the fracture is between the orbital rim and the junction between the inferior orbital sulcus and the inferior orbital margin in the anterior orbital floor. The tilted end of the fracture plate is located in the maxillary sinus while the other is connected to the orbital wall, resulting in the lower rectus sheath or surrounding soft tissues being clamped on the fracture site. Because the inferior rectus muscle has abundant adipose tissue between this site and the orbital floor, it plays a certain protective role.[1] Therefore, there was less direct muscle injury during trauma, with mainly muscle sheath or surrounding soft tissue incarceration. It was also suggested that the limited movement of the extraocular muscles is more obvious in cases of small fracture areas, and eye movement could still be restricted even after the surgical reconstruction of the orbital wall. This limitation may be due to ischemia, fibrosis, and contraction following the incarceration of the

extraocular muscles, requiring further surgical treatment if necessary.[2]

In this study, there were six cases of posterior orbital floor fractures, which all involved paralytic factors. The main feature here is that the fracture is located in the suborbital groove where the suborbital fissure meets the orbital apex; most cases were large-area collapsed fractures of the orbital floor between the inferior orbital fissure and the ethmoid maxillary suture, and the orbital contents were partly emptied into the maxillary sinus cavity. Because the oculomotor nerve branches into the lower rectus muscle site while the muscle itself may also show contusion, and ophthalmoplegia may occur, affecting eye performance. Lee *et al*[3] suggested that during the movement of the inferior rectus muscle from the orbital tip to the end of the sclera in the eyeball, there is less adipose tissue between the lower part of the inferior orbital wall and the orbital floor. Therefore, when a traumatic force causes the posterior orbital floor fracture, the inferior rectus muscle often comes into contact with the orbital floor, resulting in direct muscle contusion and subsequent paralysis. Moreover, because of the large extent of fractures and the possible incarceration of the orbital contents including the inferior rectus muscle and its surrounding soft tissues, ocular motor paralytic disorder is characteristic of this type of fracture.

Beyond that, there were 21 cases of combined anterior and posterior segment fractures, among whom 14 cases showed a restrictive complicated with paralytic movement disorder. The common feature of these cases is that the fracture is extensive, involving the anterior and posterior segments of the inferior orbital wall.[4] This can cause most of the orbital contents to exit into the maxillary sinus, damage the inferior rectus muscle and surrounding soft tissues, and promote nerve damage in the innervating muscles. Therefore, both ocular muscle restriction and paralytic dyskinesia are present. It has been proposed that hemorrhage, tear, direct contusion, or related innervation injury of the incarcerated rectus muscle could also affect the motor function of the muscle from both restrictive and paralytic aspects.[5]

In conclusion, we demonstrated that fractures in different inferior orbital wall locations cause ocular motility disorders of distinct types, which show the relationship between fracture type and ocular motility disorder. The findings may help determine prognosis and select the most appropriate operation time in patients with ocular motility disorders.

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Conflicts of interest

None.

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