MEDICO-LEGAL AND TRAUMATOLOGICAL ASPECTS OF ORBITAL FRACTURES: A CASE REPORT

Alessandro Feola¹, Valeria Marino², Simona Sorrentino³, Luigi T. Marsella⁴

SUMMARY
Orbital fractures account for 10-25% of all facial fractures. The etiology of such injuries can be traced to: acts of violence (36%), motor vehicle accidents (32%), falls (18%), sports injuries (11%), occupational accidents (3%) and gunshot wounds (2%). In 30% of cases, orbital fractures are associated with intraocular complications, 11-15% of which are serious ophthalmic emergencies that may cause reduced visual acuity. In this paper, we report the case of a motorcyclist who was treated for complex facial fractures (orbital, sphenoid, ethmoid and zygomaticomaxillary complex fractures involving the mastoid air cells and nasal conchae as well as the septum, nasal fractures and fractures in the pterygoid plates). After an initial diagnostic assessment, the doctors determined that the most critical damage was caused by the trauma inflicted to the left orbital walls. An open reduction was therefore carried out on this injury about a month later. The trauma had caused, on the one hand, a marked alteration of the patient’s facial appearance, and on the other hand, at a functional level, the onset of diplopia. This article analyses the long-term effects of orbital fractures on the patients’ physical fitness, considering the specific experience from the clinical case reported.

Introduction
In 2011, 205,638 road traffic accidents involving personal injury were recorded in Italy; the number of deaths – within thirty days from the accident – was 3,860, while 292,019 injuries were documented. Through these statistics it can be determined that of the latter, about 67,227 involved motorcycle or moped riders [1]. This article reviews a case of zygomaticomaxillary complex fracture.

Case Report
While riding his motorcycle, Mr. X, aged 60, was involved in a rear-end collision with a car that was traveling in the same direction at high speed. After the car struck the rear of his motorcycle, Mr. X, who was not wearing a helmet at the time, was thrown into the air and

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hit his head, specifically the left side of his face, on the front of the car. The patient was transported to the emergency department of the nearest hospital by emergency medical services (118), and successively hospitalized. The initial diagnostic procedures, and in particular, a craniofacial CT scan, showed an extensive fronto-orbital hematoma in addition to multiple cranial fractures (orbital, sphenoid, ethmoid and zygomaticomaxillary complex fractures involving the mastoid air cells and nasal conchae as well as the septum, nasal fractures and fractures of the pterygoid plates). In detail, the initial medical report documented the presence of a prominent hematoma of the superficial tissues of the fronto-orbital region, complex cranial fractures within the same region, with angulated fragments that were displaced both towards the brain tissue (penetrating the frontal lobes, in particular the left one) and the orbits. It was determined that the most clinically critical injury consisted in the outward displaced radial fractures of the left orbital walls (especially the orbital roof and floor), causing the inferior oblique and rectus muscles to protrude inferiorly. After stabilizing the patient, he was admitted to the hospital; approximately one month later, the zygomaticomaxillary complex (ZMC) fracture on the left side of his cranium was surgically reduced. The patient attended subsequent ophthalmic and clinical follow-up assessments (Figure 1) to monitor his post-operative progress and functional recovery. About a year after the accident, Mr. X’s case was brought to our attention in order to carry out a medicolegal evaluation for compensation purposes. An objective visual assessment determined a marked asymmetry caused by a zygomatic depression on the left side of his face with a flattening of the left lateral and inferior orbital rim, noticeable at a normal conversational distance with a casual glance. Moreover, an eye globe asymmetry was also observed, with the left eye appearing lower than the right one, although eyelid closure was found to be adequate. Mr. X also presented a left concave deviation of the nasal pyramid, visually accentuated by the skin being stretched by the depression of the left zygomatic area. Finally, peripheral diplopia caused by dysfunction of the lateral muscles was observed.

Discussion
Orbital fractures account for 10-25% of all facial fractures. The etiology of such injuries can be traced to: acts of violence (36%), motor vehicle accidents (32%), falls (18%), sports injuries (11%), occupational accidents (3%) and gunshot wounds (2%) [2]. In 30% of cases, orbital fractures are associated with intraocular complications, 11-15% of which are serious ophthalmic emergencies that may cause a reduced visual acuity [3].

**Figure 1:** Follow-up CT scan performed two months after the initial trauma.
Orbital compartment syndrome is considered to be the most serious of such ocular emergencies, since it can cause optic nerve infarct. This syndrome may occur following acute hemorrhage (caused by the laceration of the infraorbital artery or the anterior or posterior ethmoidal artery), soft tissue edema, or orbital emphysema [4].

Orbital fractures can be classified according to the injury pattern into blow-in fractures, in which the trauma leads to displacement of bone fragments towards the orbital space, and blow-out fractures, with displacement of bone fragments outwards from the orbit, as in the present case. For the more frequent blow-out fractures, that account for approximately 11% of all orbital fractures [5], two etiopathogenetic theories have been proposed (Table 1) [6].

The diagnostic approach to a patient with a blow-out fracture requires a complete clinical eye examination involving [7]:

- Vision test and examination of the pupil to detect relative afferent papillary defect or post-traumatic tonic pupil;
- Extraocular motility assessment with forcedduction test to detect potential muscle entrapments, or Hess-Lancaster screen. Muscle entrapment can be caused by direct trauma to the inferior orbital rim. Depressed fractures in this area can, in fact, displace the orbital soft tissues towards the maxillary sinuses, causing the inferior rectus and oblique muscles to become entrapped within the fracture, and ultimately resulting in dysmotility. The forcedduction test is a valuable tool used to differentiate between diplopia caused by muscle entrapment and that caused by lesions of the third cranial nerve;
- Hertel exophthalmometry to measure the globe position and exclude/diagnose enophthalmos. When present, peri- and retrobulbar hemorrhage should be ruled out;
- Slit lamp examination to detect corneal abrasions, traumatic iridocyclitis, hyphema and/or lens dislocation within the anterior segment;
- Dilated fundus examination, fundamental in the diagnosis of fundus retinæ, vitreous hemorrhage, and retinal tear or detachment. It is always important to rule out possible globe rupture [3].

When it comes to diagnostic imaging, computed tomography (CT) is considered the gold standard in the evaluation of orbital fractures. This method offers a high level of sensitivity, reported at 79-96%, and usually axial and coronal scans with 3 mm cuts are sufficient to establish the extent of the fracture and the associated dislocation of the bone fragments, as well as to determine whether muscle entrapment has occurred [8]. Plain radiographs and magnetic resonance imaging have not proved as useful for orbital fracture diagnostics, due to the high frequency of false negative results of the former and the contraindications related to the use of the latter for initial diagnostics; however, MR imaging can be a valuable tool for a successive assessment of the orbital contents and muscles [9].

The clinical symptoms and treatment options vary according to the location of the fracture. Fractures with orbital floor involvement present characteristic clinical features including periorbital hematoma, ptosis, ecchymosis, canalicular lacerations, tenderness and/or discontinuity on palpation and hypoesthesia, dyesthesia or hyperaglesia caused by ipsilateral injury to the infraorbital nerve [10]. Impaired ocular motility, caused by entrapment of the inferior rectus muscle or the surrounding tissue, may also be observed; typical presentation of muscle entrapment involves limited eye elevation and increased diplopia on upgaze (restrictive disorder); it may also be associated with

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<th>HYDRAULIC PRESSURE THEORY</th>
<th>BUCKLING FORCE THEORY</th>
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<td>Direct Impact to the globe causes an increase in hydraulic pressure, and the energy created is transmitted into the orbital walls, causing fracturing where the bone is thinner, i.e. the orbital floor and medial wall.</td>
<td>Blunt impact mainly to the orbital rim transmits a pressure wave posteriorly, thus creating compression fracture(s) in the orbital floor (Le Fort and Lagrange 1917).</td>
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Table 1: etiopathogenetic mechanism theories for blow-out fractures.
impaired infrafixed with diplopia on downgaze (paretic disorder) [10]. When entrapment is suspected, it is essential to determine the presence or absence of the oculocardiac reflex [11].

The second most common orbital fractures are those of the medial wall. These usually involve also the orbital wall and/or maxilloethmoidal strut, and can cause periorbital edema, ecchymosis, secondary subcutaneous emphysema and damage to the ethmoidal air cells, epistaxis and cerebrospinal fluid rhinorrhea. The presence of the latter indicates meningeal damage, and thus antibiotic treatment and neurosurgical assessment are indispensable [10]. Medial rectus muscle entrapment presenting with horizontal diplopia and abduction deficits may also be observed.

Finally, the least common orbital fracture, accounting for approximately 5% of all orbital fractures, is the orbital roof fracture. In adults, such fractures are usually caused by high-impact craniofacial trauma sustained in motor vehicle accidents. In 57-90% of such cases, neurologic damage is also present, while 14-38% of orbital roof fractures are associated with ocular injury, and additional orbital fractures are sustained in 76% of cases [4]. Surgical management of orbital fractures varies depending on the topographic location of the lesion, as outlined in Table 2, with the primary objective being the reinstatement of the integrity of the orbital anatomy.

Immediate surgical intervention is required in the rare cases where muscle entrapment

<table>
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<th>LOCATION</th>
<th>SURGICAL INDICATIONS</th>
<th>SURGICAL TECHNIQUE</th>
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<tr>
<td>Orbital floor</td>
<td>• entrapment of inferior rectus muscle or surrounding tissue</td>
<td>• transcutaneous subocular approach offers a wide exposure of the orbital floor, but has a high incidence of entropion and visible scarring.</td>
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<tr>
<td>[4,12,13]</td>
<td>• fractures involving &gt;50% of the orbital floor</td>
<td>• transantral, or endoscopic approach offers a limited exposure of the orbit.</td>
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<td>• fractures larger than 1 cm²</td>
<td>• transconjunctival approach (lower eyelid) with or without lateral canthotomy and inferior cantholysis: ample exposure of the entire orbital floor that can be further enlarged with a transcaruncular approach through the medial wall. Reduced incidence of postoperative entropion and/or cicatricial eyelid retraction.</td>
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<td></td>
<td>• hypoglobus</td>
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<td></td>
<td>• enophthalmos &gt;2mm</td>
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<tr>
<td>Medial wall</td>
<td>• entrapment of medial rectus muscle or enophthalmos (especially associated with large orbital floor fractures or loss of bone support at the junction of the orbital floor and medial wall).</td>
<td>transconjunctival approach combined with either transcaruncular (preferred) or retrocaruncular approach: negligible risk of visible scarring.</td>
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<tr>
<td>[4,14]</td>
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<tr>
<td>Lateral wall</td>
<td>Orbitysozyomatic fractures. If reduction is not carried out, the lateral canthal tendon and globe displacement can occur.</td>
<td>• transcutaneous approach</td>
</tr>
<tr>
<td>[14,15]</td>
<td></td>
<td>• transconjuctival approach</td>
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<tr>
<td>Orbital roof</td>
<td>• diplopia</td>
<td>transcranial approach</td>
</tr>
<tr>
<td>[4,15]</td>
<td>• motility impairment</td>
<td></td>
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<td></td>
<td>• lagophthalmos (caused by damage to the seventh cranial nerve)</td>
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<td></td>
<td>• entrapment of the superior oblique, rectus and levator muscles.</td>
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Table 2: Summary of the surgical treatment options of orbital fractures based on their location.
is associated with oculocardiac reflex. In all other cases, surgery in children should be carried out within 48 hours to minimize the risk of muscle fibrosis and permanent vertical diplopia; whereas in adults, the surgery can be delayed for up to 15 days from the initial trauma to allow for resorption of edemas, until the ultimate enophthalmos can be assessed in a more accurate manner. In this regard, it has also been shown that in 80% of patients the initially reduced ocular motility is spontaneously resolved within a relatively short period of time. In fact, this symptom can be caused by soft tissue edemas, nerve paresis and inferior rectus muscle contusion [16]. No orbital surgery is free of potential complications [10]. Misdiagnosis of an orbital fracture that requires urgent treatment may lead to intra- or post-operative complications brought on by fibrosis or contraction. Post-operative complications include loss of vision, traumatic optic neuropathy, diplopia, under- or overcorrection of enophthalmos, eyelid retraction, bleeding, infection, orbital implant extrusion, as well as hypoesthesia, orbital congestion and epiphora secondary to infraorbital nerve damage [17]. The majority of complications result from both implant malposition and the use of an inappropriately sized implant. Diplopia is the most common complication. This condition may be temporary or permanent, but since in the latter case it manifests only within the far peripheral visual field, further treatment is not recommended [18].

Transient weakness of the rectus muscle, presenting with persistent diplopia that usually improves over time, may be observed following entrapment release surgery. In the absence of marked improvements, prisms or, more rarely, surgical muscle repositioning may be implemented. Another significant complication, occurring in 0.5 – 5% of patients, is traumatic optic neuropathy [17].

Conclusions

As stated above, Mr. X’s case was brought to our attention for a medicolegal evaluation for compensation purposes. In such situations, in order to produce an accurate assessment of the patient’s condition, it is advisable to request an in-depth ophthalmic evaluation considering that the most common complication of fractures of the zygomaticomaxillary complex is diplopia, caused by muscle entrapment within the fracture. The medicolegal evaluation of diplopia and its effects on visual acuity can be very complicated, given the significant variations in the quantification of the degree of impairment depending on the area of the visual field affected. In the present case, Mr. X presented with diplopia on lateral gaze, associated, however, with a significant facial disymmetry, or evident deformities, noticeable even with a casual glance, and a depression of the zygomatic-orbital region with an asymmetry of the eye globes. Finally, it can be concluded that in personal injury assessments involving highly specialized medical fields, it is always wise to combine the expertise of the medical examiner with that of a specialist in the relevant field, in order to ensure rigorous objectivity, considered one of the fundamental principles of the medicolegal institution [19].

References

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