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SUPERIOR ORBITAL FISSURE SYNDROME ASSOCIATED WITH FACIAL TRAUMA: CASE REPORT

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ABSTRACT

Superior orbital cleft syndrome is a complex of signs and symptoms developed from damage to noble vascular and neurological structures that are found in the orbital cavity, more precisely in the region of the superior orbital fissure. The objective of this study is to report a case of superior orbital cleft syndrome after facial trauma, detailing and discussing the therapy adopted, as well as the challenges of surgical treatment. A 31-year-old male patient, melanoderma, victim of physical aggression, presented to the emergency room of the General Hospital of the State of Bahia, with facial trauma caused by physical aggression. Oral and maxillofacial physical examination revealed preserved facial contours in the upper and lower thirds of the face, bone steps in frontozygomatic and infraorbital rim regions, facial flattening in the malar region on the left, visual acuity preserved bilaterally, extrinsic orbicularis motricity preserved in the right eye and presence of total ophthalmoplegia in the left eye, exophthalmosis, periorbital ecchymosis and upper eyelid ptosis in the left eye, pupil in the left eye was not photoreactive, nasal bones and maxillae were stable, there was no mobility atypical to the manipulation of the mandible, partial edentulism in both arches, and stable dental occlusion. hypoesthesia in the frontal and infraorbital regions, both on the left. Facial computed tomography showed fractures of the orbitozygomatic-maxillary complex (MCOC), roof and floor of the orbit, all on the left, as well as the presence of bone synthesis material in an appropriate position in the region of the left mandibular angle resulting from previous facial trauma treatment. The proposed surgical treatment was osteosynthesis of the orbitozygomatic-maxillary complex and conservative treatment of roof and floor fractures of the orbit, which was successfully performed by the oral and maxillofacial surgery and traumatology team. In the postoperative period, the patient had a good evolution without significant clinical complications, and remains under clinical follow-up to the present day. Thus, the present study highlights the importance of accurate diagnosis and appropriate treatment of this syndrome, emphasizing the need for an individualized therapeutic approach, considering the signs and symptoms and the risks involved. Therefore, it is essential for the professional to know about the clinical presentation and etiological factors of this condition to avoid possible diagnostic errors and the use of inappropriate forms of treatment so that health, function and aesthetics can be returned to these patients.

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INTRODUCTION

Superior orbital fissure syndrome (SFOS) involves the impairment of various tissues and structures that make up the orbit or that are related to it in some way. In 1858, Hirschfeld described for the first time the clinical manifestations of this syndrome associated with a trauma patient, and most of the signs and symptoms described were explained by the involvement of structures of the nervous system present in the orbital anatomical region called the superior orbital fissure (FOS). [1.2]

The superior orbital fissure is a structure formed by bones of the neurocranium and through it occurs the connection between the orbital cavity and the middle cranial fossa. It is located adjacent to the orbital apex and its delimitation occurs through the sphenoid and frontal bones, which gives it an elongated pear shape with an average length of 22 mm and different thickness measurements along its path. In addition, it is divided into two parts by the tendon of the lateral rectus muscle, a component of the extrinsic orbicularis musculature, where structures responsible for ocular vascularization and innervation will pass through each subdivision. [3.4]

The superior region of the superior orbital fissure division allows passage of the trochlear nerve, branches of the ophthalmic division of the trigeminal nerve, such as the frontal and lacrimal nerves, and the superior orbital vein. In the inferior region, the trigeminal nerve branch, nasociliar nerve, oculomotor and abducens nerve branches, in addition to the inferior orbital vein, and the members of this group are considered to be orbital components more susceptible to joint injuries when there is craniofacial trauma, as they all cross the fissure confined within a tendon ring. [3.5]

Craniofacial traumas, especially those involving the middle and upper thirds of the face, usually affect the orbit, as they can involve the contents of the orbit and the bones that surround it. [5,6] Similarly, when traumas result in orbital fractures, they occasionally affect these anatomical components and are capable of causing impairments to visual acuity, extrinsic orbicularis motility, aesthetic impairments involving the loss of facial projections and contours, greater predisposition to the development of infectious processes, in addition to causing other neurological repercussions. [6.7]

Regarding the ways of classifying orbital fractures, this is done through their anatomical location, such as fractures of the orbital floor, orbital roof, lateral wall, and medial wall. [6,8] Therefore, when the bone fragments present in some of these fractures promote compression or injury to the content present in the superior orbital fissure, some signs and symptoms may appear depending on which structures were affected. In these cases, the patient may present ophthalmoplegia, eyelid ptosis, hypoesthesia in the

supraorbital region, ocular proptosis, mydriasis, retro-orbital pain, lacrimal hyposecretion, and corneal hypoesthesia. [1,6,8]

In addition, other signs and symptoms may be present due to damage to other components of the orbital content or adjacent structures, such as in cases of optic nerve involvement where the individual may present amaurosis, characteristic of orbital apex syndrome, or in cases of cavernous sinus involvement and maxillary division of the trigeminal nerve present in cavernous sinus syndrome.[7,8,9]

Superior orbital fissure syndrome is found when there is damage to the structures of the superior orbital fissure and the individual with it presents signs and symptoms such as ophthalmoplegia, eyelid ptosis, hypoesthesia in the supraorbital region, lacrimal hyposecretion, ocular proptosis and mydriasis. These clinical manifestations are explained by the nature of the components located in this anatomical region, which are mainly responsible for extrinsic motor innervation and orbicular venous drainage. Accordingly, imaging tests can also be used in the diagnostic process, such as CT scans and CT angiography of the face, angiographies and extraoral radiographs. [10.11]

However, in addition to trauma, there are other possible etiological factors for the development of this syndrome. Ocular neoplasms, bacterine infections such as syphilis, retrobulbar hematomas, internal carotid artery aneurysms, and arteriovenous fistulas are also in the group of causes of OSFS.[1.3]

Furthermore, the treatment for these cases is discussed and not fully established in the literature, but the use of corticosteroids and the surgical approach in cases of physical damage, neoplasms, hematomas and infections usually have satisfactory results. The addition of antibiotics to the therapy may be necessary, especially when the cause is of infectious origin and the patient's recovery occurs around 6 months after these interventions. [1,3,10,13]

In cases of fractures involving the FOS, open reduction and internal fixation have been shown to have an effect on the elimination of the signs and symptoms present in the syndrome, since they reestablish the local dimensions and remove non-viable bone fragments that are harmful to the tissues. However, in cases where there is severe damage to the structures present, such as cranial nerve neurotmesis, the prognosis tends to be worse. [1,3,12,13]

Another point is that the administration of corticosteroids, such as dexamethasone and methylprednisolone, is used for the management of these cases, either alone or in association with surgical approaches, as they have demonstrated beneficial effects on the

treatment, reducing edema, which causes compression and ischemia of orbital structures, in addition to having antioxidant properties. [1,2,14,15]

Thus, in order to institute an appropriate treatment, it is necessary for the professional to pay attention to the patient's medical history, to the signs and symptoms presented clinically, and to use complementary imaging tests in order to obtain more details about the disorders present. [2.3]

Thus, this study describes a case of superior orbital cleft syndrome in the left orbit, after facial trauma by physical aggression that resulted in fractures of the orbital-zygomatic-maxillary complex, roof and floor of the orbit, all on the left, addressing its nature, diagnosis and treatment. Therefore, it aims to present the clinical and imaging aspects of this syndrome, a brief anatomical review of the affected structures, possible complications involving these types of cases, as well as the appropriate management of the patient with this condition.

CASE REPORT

A 31-year-old male patient, melanoderma, victim of physical aggression, presented to the emergency room of the General Hospital of the State of Bahia, with facial trauma, reporting syncope and denying emesis after the trauma, denying other pathologies and drug allergies, and was initially evaluated by the hospital's general surgery team.

An evaluation was requested with the teams of neurology, neurosurgery, orthopedics, ophthalmology and oral and maxillofacial surgery and traumatology. Oral and maxillofacial examination showed preserved facial contours only in the upper and lower thirds of the face, bone steps in frontozygomatic regions and infraorbital ridges, facial flattening in the malar region on the left, visual acuity preserved bilaterally on visual acuity examination, extrinsic orbicular motricity preserved in the right eye and presence of total ophthalmoplegia in the left eye, adduction movements, abduction, impaired supraversion and infraversion, exophthalmos, periorbital ecchymosis and upper eyelid ptosis in the left eye, pupil in the left eye that was not photoreactive, nasal bones and stable maxillae, absence of mobility atypical to manipulation of the mandible, partial edentulism in both arches and stable dental occlusion. The patient also reported facial hypoesthesia in the frontal and infraorbital regions, both on the left.

An imaging examination was requested, the computed tomography of the face and skull revealed fractures of the orbital complex, all on the left, and also showed the presence of bone synthesis material in an appropriate position in the left mandibular angle region, resulting from previous facial trauma treatment, informed by the patient. In addition,

neurology found epidural hematoma, and neurological surveillance was maintained for two weeks with subsequent discharge from the specialty without the need for other interventions by the team.

The specialties of neurosurgery and orthopedics discharged the patient after his initial evaluation, without the need for interventional conducts. However, the ophthalmologist maintained expectant treatment, requesting reassessment after the conduct established by oral and maxillofacial surgery and traumatology.

Figure 1: Extraoral preoperative clinical images (A, B, C, and D). Clinical images showing impaired extrinsic orbicularis motility (D, E, F, and G).

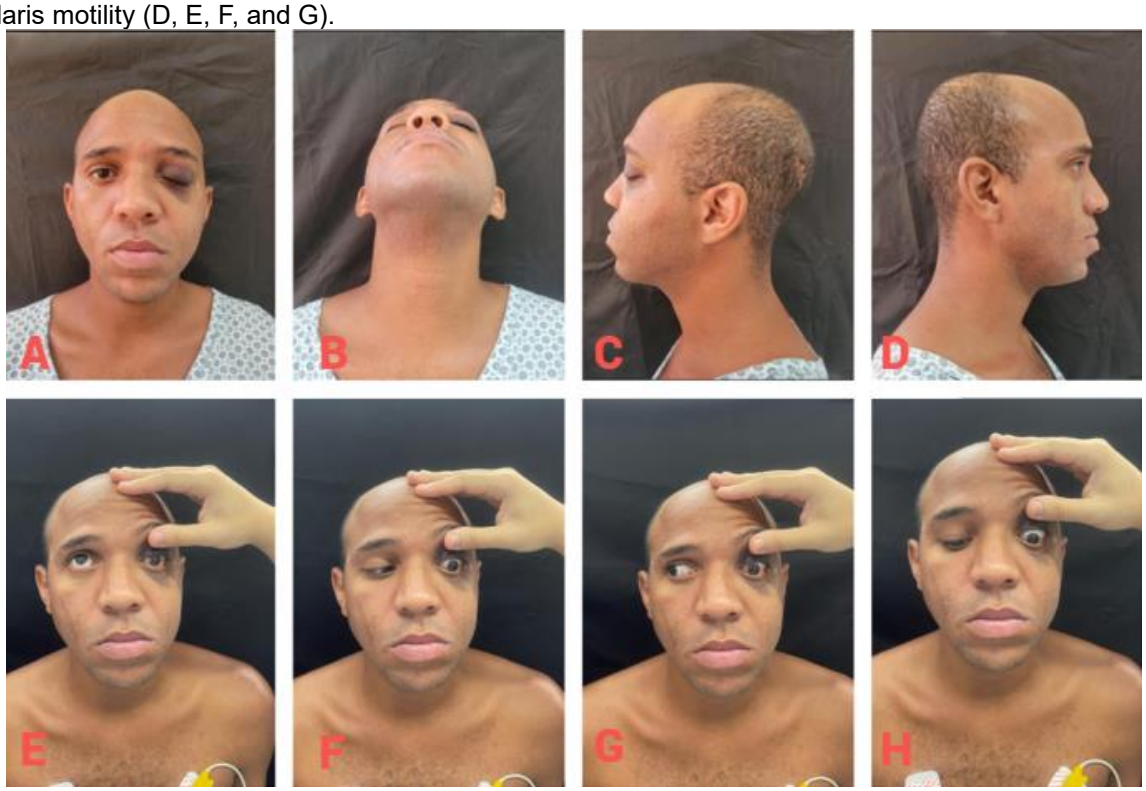
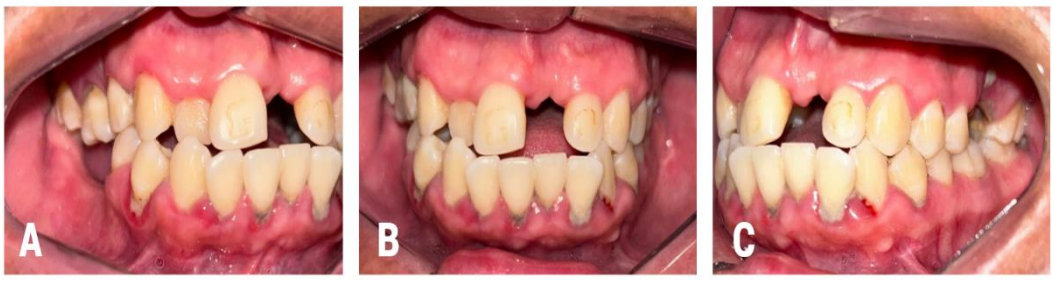


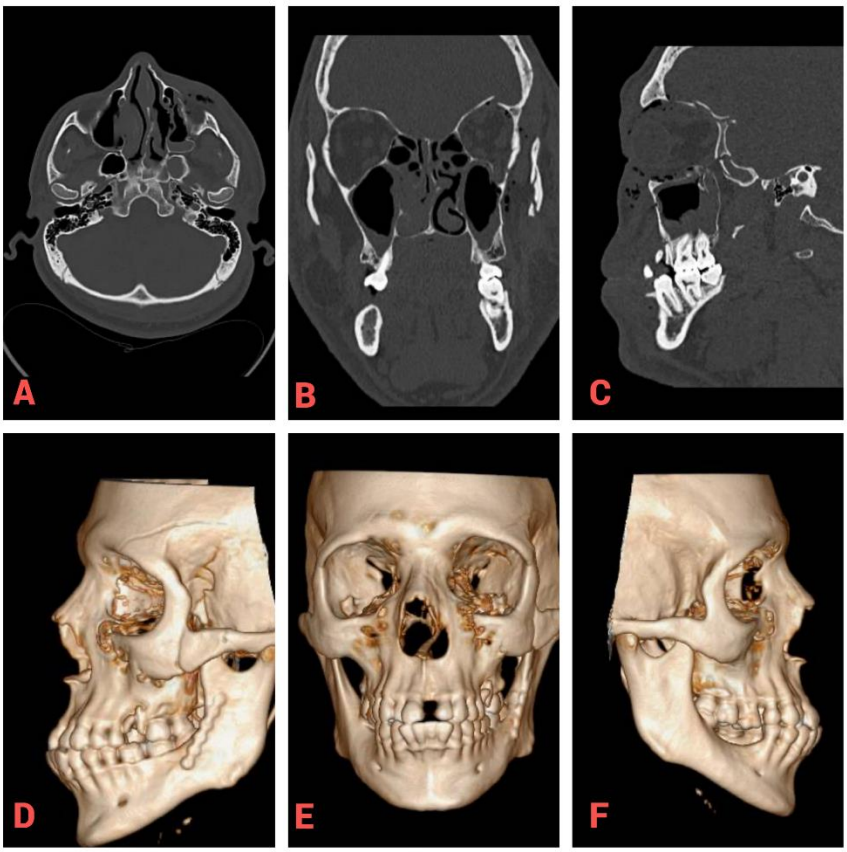
Figure 2: Preoperative intraoral clinical images (A, B, and C).



Thus, considering the fractures present and the signs and symptoms observed, it was decided to admit the patient to the hospital through the oral and maxillofacial surgery and traumatology team for the subsequent surgical treatment of traumatic injuries on the face.

In line with hospitalization, intravenous drug therapy was initiated through prophylactic antibiotic therapy, using 01 gram of a first-generation cephalosporin (cafalothin), every 06 hours for 07 days, associated with corticosteroid therapy through Dexamethasone with a dose of 10mg, every 12 hours, which was maintained for 03 days after hospitalization.

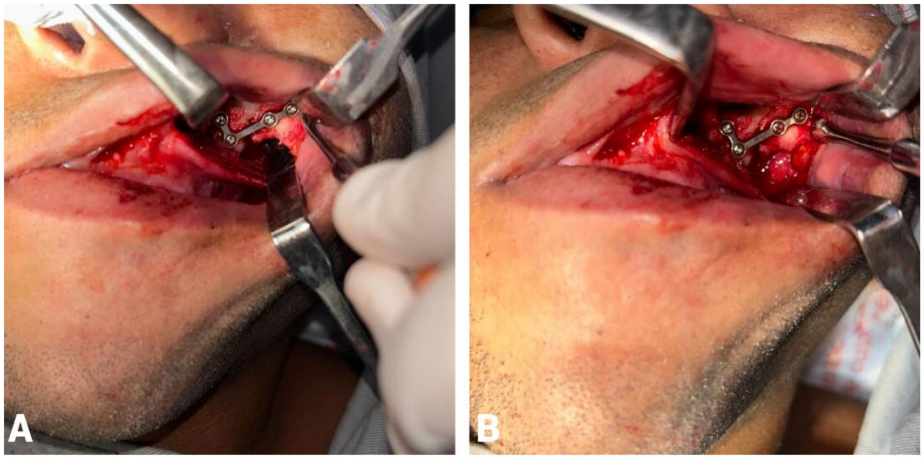
Figure 3: Computed tomography scan of the face showing fractures of the left orbital orbital complex, roof and floor of the orbit through axial (A), coronal (B), sagittal (C) and three-dimensional (D, E and F) sections.



Then, after surgical planning, preoperative exams and reduction of facial edema resulting from the trauma, the patient entered surgical programming, and was subsequently referred to the operating room and submitted to osteosynthesis of the fracture of the left orbitozygomatic-maxillary complex and conservative treatment of fractures of the left roof and floor of the orbit, under general anesthesia and nasotracheal intubation.

Access was performed in the fundus region of the left maxillary vestibule and mucoperiosteal detachment to expose the fracture, followed by maxillomandibular block and reduction and fixation of the fracture in the zygomatic-maxillary pillar region, through the system of plates and 2.0 mm screws, where only one L-type titanium plate containing 04 holes and 04 screws was installed.

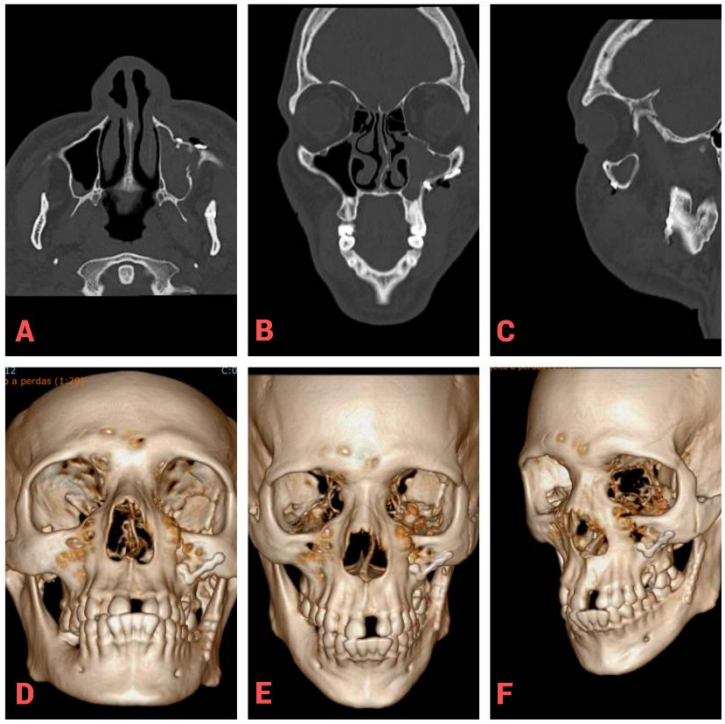
Figure 4: Intraoperative images. Open reduction and internal fixation of fractured bone stumps in the region of the left maxillary zygomatid pillar (A and B).



In the immediate postoperative follow-up, the patient progressed well, with no active bleeding and no pain complaints. A new CT scan of the face was requested and he remained under observation under the care of the anesthesiology team initially and later by the hospital's nursing team. After the first 24 hours after surgery, there were no signs suggestive of infection and other postoperative complications.

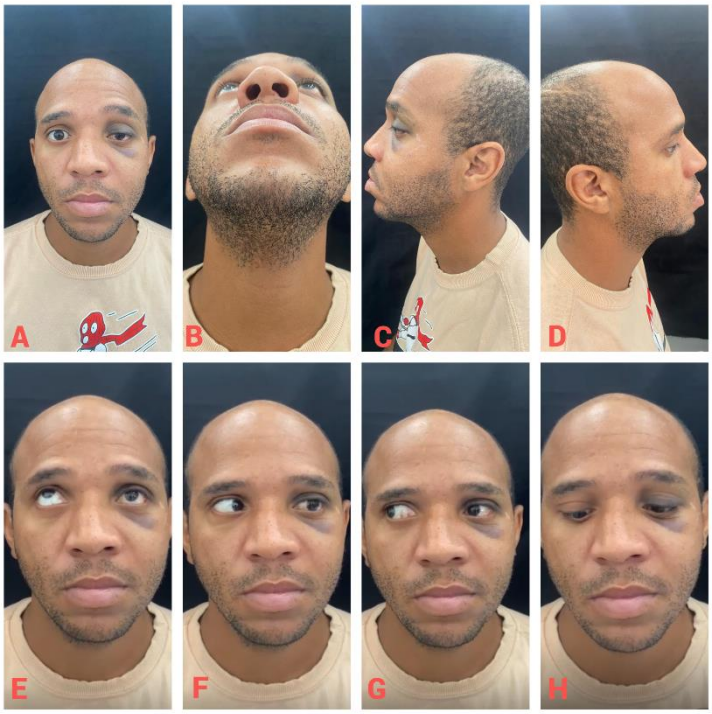
Thus, the ophthalmology team carried out its evaluation after surgery, defining that there was no need for ophthalmological conducts and, thus, the patient was discharged from the hospital after the first day of post-surgical hospitalization. However, drug therapy was maintained, and home prescription was made containing antibiotics, amoxicillin 500mg orally, every 8 hours, for 07 days, also prescribing analgesics, such as 1g sodium dipyrone, in order to provide analgesia to the patient and, in addition, dexamethasone was prescribed, renewing the corticosteroid therapy, but with a dose of 04 mg, every 08 hours, for another 03 days.

Figure 5: Postoperative computed tomography of the face. The bone synthesis material is observed in position in the region of the left zygomatic-maxillary pillar through axial (A), coronal (B), sagittal (C) and three-dimensional (D, E and F) sections.



On the seventh day after surgery, on physical examination, preserved facial contours were observed, in the left eye there was regression of periorbital ecchymosis, pupillary photoreactivity, partial reduction of eyelid ptosis and exophthalmos, presence of partial ophthalmoplegia with satisfactory performance of supraversion, infraversion and adduction movements, but there was still limitation in the abduction movement. The patient also presented preserved visual acuity, good mouth opening, and also reported hypoesthesia in the frontal region on the left, due to the condition prior to surgery, and in the infraorbital region on the left, due to tissue manipulation near the infraorbital nerve during surgery.

Figure 6: Extraoral postoperative clinical images after 07 days (A, B and C). Partial recovery of extrinsic orbicularis motility (D, E and F).



After 03 weeks, there was a considerable improvement in the patient's condition, since the physical examination showed periorbital ecchymosis in the regression stage, reduction of upper eyelid ptosis, absence of exophthalmos and recovery of extrinsic orbicularis motility in the left eye, being able to perform the movements during the exam performed, where the patient was asked to fix his gaze on one of the examiner's fingers and follow the movement in multiple directions. In line with this, the patient reported regression in the condition of hypoesthesia in the frontal and infraorbital regions, alleging recovery of local sensitivity. In this way, the characteristic signs and symptoms of superior orbital fissure syndrome are eliminated.

Figure 7: Extraoral (A, B and C) and intraoral (D, E and F) postoperative clinical images of 03 weeks.

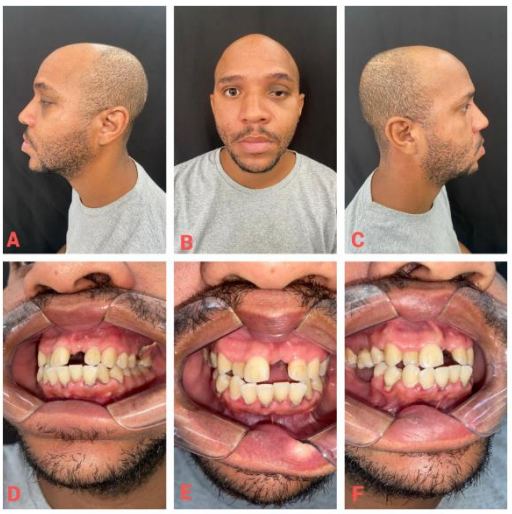
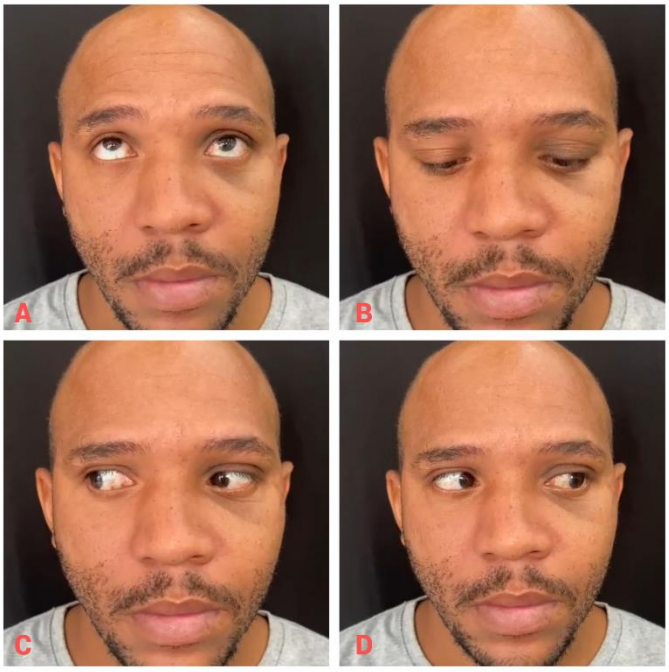


Figure 8: Postoperative clinical images demonstrating recovery of extrinsic orbicularis motility and regression of upper eyelid ptosis in the left eye after 3 weeks (A, B and C, D, E and F).



DISCUSSION

Superior orbital cleft syndrome is an uncommon condition among the complications that can arise associated with craniofacial damage and defects. [1,8,9] Therefore, the clinical manifestations presented by the patient should be correlated with the anatomical structures that are part of the FOS content and their respective functions at the time of establishing a diagnosis. Thus, the presence of hypoesthesia in the supraorbital region, for example, evidences the involvement of the frontal and lacrimal nerves, while lacrimal hyposecretion only the latter. Ophthalmoplegia, on the other hand, can represent damage to the III, IV and VI cranial nerves, and the injury to the oculomotor nerve is also responsible for the signs of eyelid ptosis, since it is responsible for the tone of the levator muscle of the upper eyelid, and ipsilateral mydriasis because there are parasympathetic fibers present in the path of this nerve and that are responsible for innervating the pupillary ciliary muscle. [1,8,9]

On the other hand, the characteristic signs and symptoms of this syndrome are common to other conditions and pathologies that affect the orbit and other structures of the skull and face. [5,7,11] This aspect makes the identification of SFOS a challenge for the professional because it makes a differential diagnosis with other diseases that have similar clinical presentation and etiologies in common, such as the orbital apex syndrome (OAS) where the impairment of the patient's visual acuity, resulting from damage to the optic nerve, is a primordial symptom in the differentiation of these two conditions. [3,4,9]

In addition to OAS, other conditions are similar to the case in question, such as arteriovenous fistulas of the cavernous sinuses, Horner's syndrome, Tolosa-Hunt syndrome,

cavernous sinus syndrome, among others. Although there are similarities between them, the identification of the etiology and symptomatology are crucial points, since the non-observance of one of the signs and symptoms can lead to an erroneous diagnosis and, consequently, to the institution of inappropriate treatment. [1,2,9,15]

Pathologies such as arteriovenous fistulas of the cavernous sinuses sometimes require other associated treatment methods, in addition to those mentioned for SFOS. Procedures such as embolization are commonly associated with interventions in these cases. Based on this thought, the patient's prognosis can be affected when the diagnosis is not accurate, as the implementation of some necessary procedure may not happen. [9,10,11]

However, something that can be analyzed by the examiner is that SFOS will have a clinical presentation according to the degree of the lesion and its scarring stage, with different levels of supraorbital hypoesthesia, lacrimal hyposecretion, ophthalmoplegia, eyelid ptosis, etc. This characteristic, although it may be common to some diseases, is not present in some cases, such as Tolosa-Hunt syndrome. [1,3,4,9]

In the case in question, the patient presented varying degrees of clinical manifestations after the conduct established by the oral and maxillofacial surgery and traumatology team, demonstrating not only the efficacy of the treatment employed, but also this characteristic of SFOS regression as the injured structures heal.

Another point is that, although there is no consensus in the literature on a standard of treatment for superior orbital cleft syndrome, the use of corticosteroids and surgical exploration are present in most of the reported cases. [1,3,4,6]

Some authors recommend the use of steroidal anti-inflammatory drugs in lower doses, 04 mg of dexamethasone every 6 hours for a short period, evaluating the patient's response to treatment, while there are professionals who prefer the administration of higher doses such as 30 mg/kg of methylprednisolone, initially, followed by 15 mg/kg every 06 hours for 03 days. [1,3,7,14]

When thinking about surgical intervention, it is consolidated that the reduction and fixation of fractures, in cases of trauma, are beneficial since they correct the narrowing of the FOS and remove the physical impaction of the structures that pass through it, but in cases where facial trauma causes retrobulbar hematomas, culminating in an SFOS, the literary reports show the possibility of surgical exploration and aspiration of the hematoma and, also, conservative treatments where the patient's body spontaneously reabsorbs the trapped content. [1,3,7,14]

The case described here obtained a satisfactory evolution through the association of both drug therapy and surgical approach and, despite the reduction and internal fixation, it

was not necessary to approach all fractured orbital segments, because in the intraoperative period it was not considered that there was a need for reconstruction of the orbital floor or management for the fracture present in the orbital roof. Thus, the importance of both forms of treatment is evidenced, since the correction of bone structures was important only for the segments that compromised the content and dimensions of the cleft, and corticosteroid therapy acted to reduce the edema present and, consequently, cause decompression of the vessels and nerves present.

CONCLUSION

The superior orbital fissure syndrome, although it does not develop from all cases of trauma and pathologies that affect the orbital cavity, is of considerable relevance to the health area because, in addition to causing important repercussions in the life of the individual who wears it, bringing aesthetic and functional damage, it also performs a differential diagnosis with several other orbital problems.

Clinically and by imaging tests, the diagnosis can be challenging, considering that the similarity with other pathologies is present. CT angiography and computed tomography of the face play an essential role in the diagnostic direction and therapeutic planning of these cases, providing detailed information on the extent and involvement of noble structures. Therefore, it is essential to know its pathophysiology and clinical manifestations linked to the appropriate interpretation of complementary tests in order to establish a correct diagnosis and management.

Corticosteroid therapy proved to be efficient both at the time of hospitalization and in the postoperative period, accelerating the resolution of signs and symptoms. However, the reduction and internal fixation of fractures, by promoting the return of the orbital volume and decompression of the structures present in the cleft, proved to be a decisive factor for the success of the treatment.

Therefore, the treatment instituted must be individualized, and there is no established protocol, and, therefore, it is essential to consider aspects such as the etiological factor, previous medical history, current general health status, degree and extent of the lesions present, and dimensions of the signs and symptoms presented by the patient at the time of choosing the appropriate therapy.

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