Complications of Delayed or Inadequately Treated Nasoorbitoethmoid Fractures

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ABSTRACT:

Nasoorbitoethmoid (NOE) fractures are associated with high force trauma wherein the nasal bones are unable to contain the incoming pressure and force is dispersed into the ethmoid sinuses and orbit, as the bone is thin.

When these fractures occur, they cause devastating complications because of their proximity to the brain, eyes and nose. NOE injuries causing facial deformity are difficult to treat if left undetected for longer than 2 weeks. Delayed surgical intervention allows the resultant scarring, contracture, impeding reduction of the fractures and instability of the skeletal buttresses and surrounding soft tissue to progress, leading to deficits in facial function and aesthetics.

Saddle nose deformity, telecanthus, enophthalmos, nasolacrimal duct obstruction and soft tissue scarring are often encountered in the secondary management of NOE fractures. Problem based approach with special attention to the intercanthal distance and nasal projection should be used in the management of such conditions.

The NOE injury continues to be one of the most challenging from both a diagnostic and a treatment standpoint in reconstruction of the maxillofacial skeleton after trauma.

Thus, this article showcases the possible complications that are encountered which can be avoided with an accurate assessment of injury with timely intervention and a comprehensive treatment necessary for optimal results.

INTRODUCTION

Nasoorbitoethmoid (NOE) fractures present some of the most challenging scenarios in facial fracture reconstruction.

Fractures of the frontal bone and the NOE complex are infrequent, occurring among 2 to 15% of patients with facial fractures. When these fractures occur, they cause devasting complications because of their proximity to the brain, eyes, nose.

Despite their overall reduced incidence, understanding the management of these injuries remains important. Suboptimal initial management routinely results in very poor results and late complications, including shortened palpebral fissures, telecanthus, enophthalmos, dystopia, and saddle nasal deformity, which are almost impossible to fully treat secondarily. Improper attention to concomitant injuries can also lead to significant sequelae, as close to 60% of NOE fractures are associated with orbitozygomatic fractures and 20% are seen in the setting of panfacial fractures.

Prompt surgical reduction should, therefore, be the reconstructive surgeon's primary aim. Delayed surgical intervention allows the resultant scarring, contracture, and instability of the skeletal buttresses and surrounding soft tissue to progress, leading to deformities that are difficult to address.

As part of the initial work-up, apart from a detailed clinical examination, computed tomography (CT) imaging of the maxillofacial area with 1 to 2 mm cuts should be acquired to adequately evaluate the distorted skeletal anatomy and possible surgical approaches. Special attention should be placed to the medial canthal tendon and the degree of bony comminution in the medial orbital rim bone and medial orbital wall as this will suggest the necessity of transnasal wiring, extent of exposure needed, and bone grafting.In patients who have had an unsatisfactory primary reduction, plating and wiring systems should be noted with their position and effect on orbit volume.

The current paradigm of primary treatment includes meticulous open reduction and internal fixation with titanium plates and screws and the use of transnasal wiring when appropriate given the degree of comminution that the medial orbital rim displays in relation to the attachment of the medial canthal tendon. Principles of secondary reconstruction follow closely with those of primary treatment; however, posttraumatic scarring and contracture with poorly defined tissue planes reemphasize the need for careful dissection.

ETIOLOGY

The NOE fracture is caused by forceful trauma to the upper central midface. Due to the high energy involved, these fractures often occur in combination with injuries to other parts of the face and body. Road traffic accidents are the most common cause of NOE fractures, especially involving motorcycles.

The introduction of seatbelts and airbags has helped to decrease the overall frequency of facial fractures. Physical assault, sport, and horse kicks have also been associated with this fracture pattern.

CLINICAL PRESENTATION OF NOE COMPLEX FRACTURES :

- -The characteristics of NOE injuries are a
- short and retruded nasal bridge, telecanthus, enophthalmos and shortened palpebral fissure.
- -In addition, ocular dystopia may accompany such injuries.

Periorbital ecchymosis and pain are the most common signs and symptoms associated with fractures of frontal bone. Subconjunctival hemorrhage ,Nasal deformity, edema and ecchymosis of eyelids ,CSF leakage , hyposmia ,traumatic telencanthus , increased canthal angles ,blindness ,soft tissue laceration in the regions of glabella and supraorbital rim region ,mobility of the nasal bones ,wide and flattened nasal dorsum, upturned nasal tip.

Palpation will reveal crepitation and tenderness over the fractured site, depression of bone fragments into the orbit may cause exophthalmos, proptosis or ptosis. Depressed injury causes restriction in the ocular movement. Medial orbital wall fractures causes enophthalmos.

COMPLICATIONS

Saddle Nose Deformity

The saddle nose deformity following NOE fractures presents a unique challenge to the reconstructive surgeon. Characteristically, the saddle nose deformity is caused by a loss of nasal support resulting in a loss of nasal projection, nasal shortening, tip overprojection, and middle vault flattening. Not only do these injuries have potentially devastating aesthetic repercussions, but they also have functional implications as well. Therefore, any repair must attempt to restore nasal projection and alleviate symptoms of nasal obstruction. Ultimately, the reconstructive technique depends on the severity of the deformity. Milder defects with good septal support only require dorsal augmentation, best achieved with a septal cartilage onlay graft or via the Turkish Delight technique, in which finely diced cartilage is wrapped in Surgicel or fascia, our preferred modification. Conversely, costochondral or bone strut grafts can be necessary to restore nasal projection in those more severe defects with loss of septal support

Telecanthus

Disruption in the medial canthal tendon and its bony attachment to the medial orbital rim will lead to telecanthus. increased intercanthal distance. Normal intercanthal distance varies greatly within the population, but averages between 29 to 34 mm in adult females and 29 to 36 mm in adult males. During fracture reduction, the canthal-bearing bone fragments must be precisely reduced to restore the preinjury intercanthal distance. In less comminuted fractures with large bony fragments, the use of a two- or three-point plate fixation usually suffices; any nasal bone fractures should also be reduced to maintain the patient's preinjury nasal projection. However, for NOE type III and occasionally for NOE type II fractures, the additional use of transnasal canthopexy is required to secure the avulsed medial canthal tendon to its insertion point. Unrepaired or inadequately repaired NOE fractures can result in persistent telecanthus, requiring secondary repair. This often necessitates the use of osteotomies and bone grafting with resulting less predictable outcomes.

Enophthalmos and Diplopia

Posttraumatic enophthalmos is the posterior displacement of the orbital contents caused by disruption of the orbital architecture and resultant increase in orbital volume which becomes more pronounced after resolution of posttraumatic edema and with tissue remodeling. It is characterized by a sunken eye, pseudoptosis, and an exaggerated supratarsal fold. Enophthalmos is easiest to appreciate from an inferior view and only 2 mm of displacement is necessary for the difference to be noticeable. Bone and cartilage grafts or alloplastic implants may be needed to further repair orbital walls when bone loss is evident.

Nasolacrimal Duct Obstruction

Nasoorbitoethmoid fractures are the leading cause of traumatic nasolacrimal duct obstruction. The nasolacrimal sac is hidden within the lateral nasal wall, enclosed between the frontal process of the maxillary bone and the lacrimal bone. Between 5 and 21% of NOE fractures may involve the bony lacrimal canals leading to posttraumatic dacryostenosis. Radiologic features significantly associated with development of epiphora or Dacryocystitis were lacrimal crest avulsion, displaced bone fragments within the lacrimal fossa or duct, more than 50% compression of the duct, and shift of the nasomaxillary buttress. Posttraumatic patients with epiphora that are found to have identifiable canalicular puncta should undergo Dacryocystography to identify the specific area of obstruction as well as the overarching size of the lacrimal sac and integrity of the canalicular system. Early and meticulous open reduction of NOE fractures decreases the risk of posttraumatic nasolacrimal obstruction and epiphora; waiting 2 weeks or more after initial trauma may lead to further bone loss and scarring of the lacrimal area resulting in worsening obstruction.

Epiphora

Epiphora in the early stage of trauma may be caused by facial and conjunctival edema, NLD obstruction, or reactive hypersecretion due to pain. As the facial edema and hypersecretion usually resolve quickly, the accompanying epiphora may improve in a relatively short time, but that due to NLD obstruction can last for months. The NLD obstruction after injury may be temporary and spontaneously recover over time; however, development of prolonged obstruction is possible due to soft tissue stricture, adhesions, or bony remodeling.

Soft-Tissue Scarring

Overlying soft-tissue injury is often present in conjunction with NOE fractures and subsequent scarring is difficult to treat. Delayed treatment of fractures allows time for fibrosis and scarring of traumatically repositioned tissues, leading to aesthetically poor outcomes. Moreover, the contractured overlying soft tissue increases the risk of underlying bone resorption, accentuating contour deformities. Similarly, failure of adequate external compression in the medial canthal area after bicoronal approaches during the primary repair of NOE fractures can allow blood and fluid to build up underneath the degloved tissue with resultant poor contouring of lateral nasal and infraorbital areas. Contour deformities in the nose can be addressed with fillers as well; Restylane (Galderma)

is a preferred filler for use in the nose and has a low hydrophilicity and a high concentration of hyaluronic acid, an optimal combination in the thin skinned nose which leads to longevity of augmentation and decreased risk of fluid retention and edema in the injected area. To prevent further scarring after debulking and all fracture stabilization, a Xeroform or felt bolster should be fashioned and secured with a transnasal suture and left for at least 1 week. External Aquaplast nasal splint formation with stabilization using septal or transnasal K wires (0.7–0.9 mm) and protective K-wire balls can be very helpful to reduce swelling and postoperative fluid collection.

CONCLUSION

Delayed surgery allows scaring to develop, impeding reduction of the fractures and any bone grafting that may be necessary. Secondary reduction is challenging due to the scarring and contracture of soft tissue and bony fragments. A problem based approach with special attention to the intercanthal distance and nasal projection should be used in the management of secondary NOE fracture reduction.

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