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Syndromes Associated With Maxillofacial Injury: A Generaized Overview

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Abstract

Maxillofacial injuries are one of the common features of road traffic accidents. Every maxillofacial fracture patient must be carefully evaluated clinically and radiologically to rule out any underlying complications. Among these complications are the injury-associated syndromes. This review article aimed to bring a generalized overview of different syndromes that may be associated with maxillofacial injuries.

Keywords: Syndromes in maxillofacial injury, Posttraumatic Syndromes.

Introduction

One of the common features of road traffic accidents (RTA) and other means of trauma are maxillofacial injuries. Hence every maxillofacial fracture patient must be carefully evaluated clinically and radiologically to rule out any underlying head injury and to decrease the incidence of mortality rate or any complications.[1]

Sometimes these complications may also lead to a life threatening conditions and in these situations the first priority is to stabilize the patient. Many times the post-trauma complications are inevitable, but it is important to be aware of these complications so as to provide the best possible treatment. These complications may depend on the localization of the initial injury, but it can also arise from the treatment itself.[2]

Among these complications are the injury-associated syndromes. These syndromes can either be due to direct effect of the trauma that leads to entrapment or compression of vital structures or may be secondary to the consequence of other local and systemic body reactions to the stress induced by the trauma/or its management.[3]

There are several literatures, which have reported cases of syndromes associated with maxillofacial injuries. This article aimed to do a brief review of few literatures and to

bring a generalized overview of different syndromes that may be associated with maxillofacial injuries.

Syndromes associated with maxillofacial injuries

Superior orbital fissure syndrome: The superior orbital fissure syndrome (SOFS) is a complex of impaired function of the cranial nerves (III, IV, V, and VI) that enter the orbit through the superior orbital fissure (SOF). The three major precipitating factors for SOFS are trauma, tumor, and inflammation.[4]

According to Banks, Hirschfeld first described a trauma patient with the physical sign of SOFS in 1858. In 1896, Rochon-Duvignaud reported the syndrome as a pathological entity in four syphilis patients. Lakke later in the year 1962 further defined the complete SOFS, which consisted of paresis of the ocular muscle, sensory disturbance in the distribution of the first branch of trigeminal nerve, and retroorbital pain.[5, 6]

Anatomic Considerations

The SOF serves as a pathway that allows communication between the orbit and the middle cranial fossa.[7] The SOF is bounded laterally by the greater wing of the sphenoid, medially by the lesser wing of sphenoid, and superiorly by the frontal bone. It lies at the apex of the orbit and at the border between the roof and the lateral orbital wall. The shape of SOF is like an elongated pear with the broadest part at the nasal side. The long axis extends upward from nasal to lateral at an angle of 45 degrees.[6]

Reymond, et al. in their cadaveric study on 100 skulls concluded that SOF had mainly two morphological variants: Type “a” with characteristic narrowing within the fissure and type “b” which lacked such narrowing and was significantly shorter. The morphological types of the SOF did not present any statistical variation correlating with gender and body type.[8, 4]

The size of the SOF is around 22 mm in length, 2–3 mm in width in the narrow part, and 7–8 mm in the broader part. Fujiwara et al further investigated the anatomic width of the SOF using the cadavers and computed tomographic (CT) scan. The width of SOF in the cadavers (3.2 +/-1.09 mm) was similar to that found in the CT scans (3.73+/-1.64 mm). Their study concluded that narrow SOF (less than 1.6 mm) was a risk factor for SOFS in some anatomic variation.[6, 9]

The tendon of the lateral rectus muscle divides the fissure into two parts: The **superior part** contains trochlear nerve (IV),frontal and lacrimal branches of the ophthalmic division of the trigeminal nerve (V), and the superior branch of ophthalmic vein; and the **inferior part** which is confined within the tendinous ring contains superior and inferior branches of the oculomotor nerve (III), abducent nerve (VI), nasociliary nerves (V), and the inferior branch of ophthalmic vein, making them more susceptible to shearing injury during craniofacial trauma.[4] These structures in the inferior part of SOF are confined within the tendinous ring, which makes the oculomotor, the abducent, and the nasociliary nerves more susceptible to shearing injury during craniofacial trauma. On the contrary, the trochlear nerve passing superiorly above the tendinous ring has anatomically enhanced protection from the traction injury.

The motor nerves to the eye with the ophthalmic branch of the fifth nerve are grouped together in the wall of the cavernous sinus. This may explain that any pathologic process in the cavernous sinus such as traumatic carotid-cavernous sinus fistula (CCSF) can produce SOFS. The above-mentioned nerves transverse the fissure into the periorbital tissue. The fissure also contains superior and inferior orbital veins, as well as some sympathetic filaments from the cavernous plexus. [6]

Etiology

Numerous etiologies of the syndrome have been reported in the literature including craniofacial trauma, hematoma

of the cavernous sinus or retrobulbar space, infection, neoplasm, aneurysm of the internal carotid artery or arteriovenous fistulae, or idiopathic etiologies.[7]

Craniofacial trauma including skull fracture, zygomaticomaxillary complex, and orbit, and LeFort II and LeFort III fractures account for a major cause of SOFS. . A blow on the skull bone might radiate forward to the roof of the orbit involving the greater wing of the sphenoid and result in **hematoma** formation around the SOF resulting in SOFS. The **infection and inflammation** of the meninges, cavernous sinus or retrobulbar space that causes SOFS are mainly caused by syphilis and tuberculosis **Neoplasms** arising from the meninges, bone, and the cerebral tissue near the site of SOF or tumors from remote primary site are capable of producing SOFS. **Aneurysm** of the internal carotid artery or arteriovenous fistulae induced by trauma has been reported as another cause for SOFS in many literature.[6]

Two **mechanisms** can be identified; the direct, which is when the nerves traversing the fissure are interrupted or compressed by displaced bone fragments. The second is indirect mechanism, in which the orbital walls behave like a nonexpandable box, so every increase in internal orbital pressure caused at the moment of the injury by posterior displacement of the eyeball or later by edema and bleeding may compress the nerves against the rims of the fissure.[3]

Chien and Yu ray in his study retrospectively reviewed 11,284 patients with Craniomaxillofacial fractures and identified 33 cases of SOFS, which is about 0.3%. The incidence of traumatic SOFS is1 (0.8%) in every 130 cases of facial fractures according to **Zachariades et al. Antonyshyn et al** reported four cases of traumatic SOFS in a series of 841 complex facial injuries, including 41 blow-in fractures of the orbit. According to **Chien and Yu ray**, the impact force close to the orbit during trauma gives the higher incidence of SOFS.[6]

CLINICAL SIGN & SYMPTOM

The clinical symptoms are primarily the result of inflammation and compression of adjacent nervous tissue. Lid ptosis is caused by either the involvement of the sympathetic fibers arising from the cavernous sinus, resulting in loss of tone of Mueller muscles, or the involvement of the somatic efferent fibers that course along the superior branch of the oculomotor nerve, resulting in loss of tone of the levator palpebrae superioris muscle. The ophthalmoplegia is secondary to impairment of cranial nerves III, IV, and VI.

Disturbance of the lacrimal and frontal nerves leads to anesthesia of the forehead and upper eyelid. Compromise of the parasympathetic innervation, which travels with the oculomotor nerve, results in paralysis of the pupillary ciliary muscle. This paralysis causes dilatation, fixation, and loss of accommodation of the ipsilateral pupil. The proptosis can be attributed to the loss of tone of the extraocular muscles which normally exert a retracting force on the globe.[7]

These symptoms are present in various degrees, depending on the severity of the damaged structures and the stage of healing at the time of examining the patient. The term ‘‘partial superior orbital fissure syndrome’’ indicated partial or complete involvement of the third and sixth cranial nerves along with the nasociliary nerve. These three structures passing inside the tendinous ring are more susceptible to compression injury than other contents of SOFS.[6]

Diagnosis

Any patients with maxillofacial injury and with above signs and symptoms can be easily diagnosed as SOFS. Most of the literatures have shown that CT has been the

main criteria for the radiographic diagnosis of SOFS.[7] However before the CT emerged, optimal plain film viewing of the SOF with posterior/anterior orbital projection in a 20-25 degree tilt of the head (Caldwell projection) were used for radiographic examination. One of the limitation of the plain film is that in severely traumatized patient it is often difficult to obtain and can be hazardous especially when there is a concomitant cervical trauma. On the other hand, in CT scan a small bony fragments or retroorbital hematoma with compression around SOF can be easily visualized by fine-cut of 2 mm slices. After the advent of spiral CT in nineties, the new spiral CT equipment is a promising tool to improve the diagnostic accuracy, which can provide a detailed information of SOF in the axial plane, coronal projection, and three-dimensional CT reformatted images. It can help avoid the discomfort of neck hypertension while obtaining the coronal CT slices by the traditional CT machine, and provide information related to neighbouring brain injury and concomitant craniofacial fractures.

Angiography is also a useful tool in revealing the traumatic carotid cavernous sinus fistula or carotid aneurysm causing SOFS.[6]

Treatment

The rarity of traumatic SOFS has made it difficult to define treatment guidelines for this condition.[3] Most authors agree that exploration is warranted in cases of neoplasm, physical impingement, infection, or retrobulbar haemorrhage.[7] The rationale behind the treatment of SOFS of traumatic origin lies primarily in minimizing further irreparable damage to the neuronal structures. Fracture repair and restoration of the bony anatomy come secondary.[4]

Except the etiology of traumatic SOFS, proper management of traumatic SOFS in not clearly defined in the literature because of the relatively small number of cases. Treatment varied from conservative treatment to steroid administration, and surgical intervention is reported. Conservative treatment with observation alone has been proposed because complete or partial spontaneous recovery of the motor and sensory functions usually occurs when the syndrome results from trauma. Moreover, the hazard of further haemorrhage or injury to nerve and operative difficulties are the major drawback of surgical exploration.[6] Sometime in traumatic SOFS, a complete or partial recovery can be expected without any intervention aimed at the fissure itself, as long as the nerves are intact. The prognosis is obviously poor if the nerves have been severed or severely damaged by fractured bones.[7]

On the other hand, steroid therapy may be beneficial in patients with SOFS caused by edema from craniofacial fracture[6] and varying doses of systemic corticosteroids have been advocated as treatment alone, or in conjunction with other modalities such as facial fracture reduction.[7]

The short-term use of dexamethasone (4 mg every 6 hours) in patients with traumatic SOFS caused by zygomaticomaxillary complex fracture and sphenoid fracture was first reported by **Postma et al**. Within the 3-month follow up the patient was free from neurological and ocular symptoms.[10,6] Similar medical treatment with loading dose of 1 mg/kg dexamethasone followed by 0.5 mg/kg every 6 hours was reported by **Rohrich et al** to treat SOFS in addition to the reduction of associated facial fracture.[11,6] In the year, 2004 **Acarturk et al** reported excellent outcome in five patients with traumatic SOFS using megadose steroid of 30 mg/kg methylprednisolone followed by 5.4 mg/kg per hour for 48 hours. All of them recovered completely by 6 months without any complications attributed to high-dose steroid.[12,6] **Chien et al** reviewed the literature on steroid treatment of traumatic SOFS in which 7 (70%) of 10 cases of traumatic SOFS

resolved completely after steroid treatment. It seems that the patients treated with steroids have a better chance of neurologic recovery than those with observation alone.[6]

Retroorbital hematoma has been one of the causes of traumatic SOFS. Although some authors suggest aspiration of retrobulbar hematomas associated with fractures, generally resorbs spontaneously within 3 weeks to 4 months.[13] According to **Rohrich et al** traumatic SOFS can be easily treated with open reduction of facial fractures and intravenous steroids without specific treatment of retrobulbar hematoma.[11] **Mortada** recommended that an exploration of the orbital apex is indicated through a lateral transconjunctival orbitotomy to evacuate the blood if medical treatment is unsuccessful in approximately 4 months.[14]

Surgical intervention is considered when there is an obvious retrobulbar hematoma showing no signs to resolve and the presence of significant narrowing of the SOF from the displaced fracture fragment.[4, 3, 6] A CT scan depicting an evident bony compression from a displaced sphenoid fracture or an orbital blow-in fracture with narrowing of SOF, one should prompt for a surgical decompression to reduce intraorbital pressure.[4]

Murakami described four different routes for decompression of the SOFS:

(1) The extra nasal intraorbital route, for decompression of the lateral wall of the SOF,

(2) The modified extranasal intraorbital route with protection of the trochlear nerve,

(3) The extranasal transethmoidal route to decompress the medial wall of the SOF, and

(4) The transtemporal route, which is indicated in case of fulminating suppuration around the SOFS.[15,6]

Chein and Yu ray propose the direct decompression of SOF through the coronal approach and zygomatic osteotomy followed by reduction of sphenoid bone under direct vision. It was observed that following the operation the movement of the eyeball and sensory function usually recover within 3 to 6 months.[6]

In summary, SOFS is a rare complication of craniofacial injury. Diagnosis is based on clinical presentation which can be confirmed by radiological examination. In the absence of compression by the fracture fragments, conservative approach with megadose steroids should be the first line of treatment. In presence of bony compression, a surgical decompression should be planned. The associated facial fractures, especially orbital blow-in fracture, should be reduced early to relieve intraorbital pressure, if the general condition is stable. Partial to complete recovery of cranial nerve function can be expected after proper treatment and improvement reaches its plateau by the end of six months.[4]

Orbital Apex Syndrome

An orbital apex syndrome (OAS) is a syndrome involving the same cranial nerves as in SOFS, but there is associated optic nerve dysfunction.[3] Kjoer was the first who described OAS as a loss of visual acuity with symptoms of SOFS.[7] The primary focus of a surgeon should be to locate the lesion first and then identify the etiology.[16] OAS is a complex disease caused by a various etiological factors, such as trauma, foreign bodies, fungi, cephalic and facial infections, and tumor invasion of the orbital apex, (basically it's a combine feature of SOFS along with traumatic optic neuropathy).[17]

The etiological factor, clinical sign/symptom, diagnosis and treatment of OAS is more or less same as that of SOFS, which have been discussed above in detail.

However according to **Chien and Yu ray** surgical decompression in case of OAS should be done using transconjunctival approach only if the patients have following conditions:

(1) CT evidence of impingement of bony fragments to the optic nerve

(2) Failure of improvement of vision after 3 days of megadose steroid therapy with visual impairment of finger counting or worse

(3) Progressive visual deterioration during steroid treatment.[6]

The treatment strategies of traumatic OAS is different between direct and indirect injuries, in terms of the indications for, and urgency of surgery. Surgical intervention is recommended for the direct type of traumatic OAS. **Yuhua Tong et al** in his case study reported that a traumatic OAS patient was treated successfully by nasal endoscopy by decompression of optic nerve and orbital apex. The patient visual acuity and extraocular function was completely restored after 3 week and 1 year respectively.[17]

Overall, the three treatment options exist: observation alone, high-dose corticosteroids, and surgical optic canal decompression.[3]

Orbital Compartment Syndrome

Gordan and McCare first described orbital compartment syndrome (OCS) in 1950, following the traumatic malar fracture repair.[18] OCS is characterized by an acute or subacute rise in intra-orbital pressure (IOP) and if it is not treated immediately, permanent loss of vision may develop due to damage to ocular and other intra-orbital structures.[19] Typically, an IOP >20 mmHg is considered elevated, and an increased IOP may compress the optic nerve directly or cause compression of its vasculature.[3]

Anatomic Considerations

The orbit is composed of seven bones. The seven bone includes frontal bone, sphenoid bone, zygomatic bone, maxillary bone, palatine bone, lacrimal bone and ethmoid bone. The lateral wall is formed by the greater wing of the sphenoid apically and the frontal and zygomatic bones facially. The floor is formed from the sphenoid, the orbital process of the palatine bone, and the orbital process of the maxillary bone. The medial wall is formed from the lesser wing of the sphenoid, the ethmoid bone, the lacrimal bone, and the frontal process of the maxilla. The roof of the orbit is derived from the sphenoid and the frontal bones. The height of the orbit is usually 35 mm, whereas the width is approximately 40 mm as measured at the rims.[20] The orbit is a confined, cone-shaped space, which apart from its anterior aspect is bound on all sides by bony walls. The orbit contains the globe, orbital fat, extraocular muscles, lacrimal gland, lacrimal sac, retrobulbar contents and neurovascular anatomy. Anteriorly, the orbit is limited by the orbital septum and tarsal plates of the upper and lower eyelid and by the attachment of medial and lateral canthus tendons. The average adult orbit has a volume of approximately 30mL, with an intraorbital pressure of 3–6mmHg. It is recognized that the orbit has limited compliance related to the limited elasticity of the septum and tarsal plates, beyond which results in an increase in intra-orbital pressure.[21, 3]

Pathological Mechanism

The orbit has limited capacity to expand as it is restricted by an enclosed space.[19] The pathogenic mechanism of OCS can be described in terms of a rapid rise in IOP that is beyond systolic pressure due to acute rise in volume within confined orbital spaces, causing a fall in perfusion below the critical level. Both the tissue and venous pressure increases when fluid (e.g. blood) enters a fixed-volume compartment.[3] When these pressures exceed in the central retinal artery and ophthalmic artery, blood flow in the vessels stops, causing ischemia of the retina, optic disc, and other ocular tissues, and eventually irreversible vision loss. It has been demonstrated that increased IOP lasting 60 to 100 minutes can cause permanent vision loss. The exact mechanism of damage to various nerves and

their branches is unknown. Direct compression and impairment of blood supply to the nerves have been proposed as the possible mechanisms.[19] In case of retrobulbar hemorrhage, the globe is displaced anteriorly to the extent allowable by the canthal tendons (and to a lesser degree, the prolapse of bulbar fat). Anterior displacement squeezes the globe between the immobile eyelids and the expanding hematoma. When anterior displacement of the globe reaches the limits of its anatomical restraints, intraorbital and intraocular pressures can go up precipitously, leading to permanent damage to the optic nerve. The result of OCS may be central retinal artery occlusion, anterior ischemic neuropathy, and blindness, if not reversed immediately.[3] Hargarden et al simulated OCS in primates, by placing a catheter in the retro-bulbar space and inflating it with saline for a minimum of 180 minutes. Following histopathological analysis, the authors concluded that visual loss occurred as a result of damage to the optic nerve from prolonged ischemia.[22,21]

Etiology

Any process that results in an increase in mass effect within the confines of the orbit can result in OCS.[21] OCS primarily occurs due to hemorrhage, abscess, tumor, orbital edema or emphysema, orbital cellulitis, retrobulbar injection, or pre-existing medical disorders. However, it may also result from acute orbital inflammation or an allergic reaction following any peribulbar drug injection, prolonged hypoxemia with a capillary leak, an intra-orbital foreign body, retained foreign material, such as bacitracin ointment, or oxidized regenerated cellulose, especially in cases of sinus surgery.[19]

Among all these, the most common precipitating event of OCS is hemorrhage secondary to trauma, which may be within the orbit contents or sub periosteal.[21] This is frequently seen in trauma like Zygomatic complex fracture, Le Fort I, II, and III fractures.[3] Hemorrhage in OCS can also be caused in blepharoplasty and orthognathic surgery which can cause acute retrobulbar haemorrhage leading to blindness. Although the vison loss associated with orthognathic surgery is very rare.[23]

Sun et al did a retrospective case series of eight patients with OCS secondary to blunt trauma presenting to the Royal Adelaide Hospital between 2004 and 2013. In his study, he found that six patients suffered orbital fractures secondary to their injury, and five patients had associated facial or nasal fractures including Zygomatic complex fracture, Le Fort I, II, and III fractures.[24,3] Blindness following repair of orbital fractures is an infrequent but well-documented phenomenon. **Susarla et al** reported a case of OCS leading to visual loss following orbital floor reconstruction. It is possible that the patient may sustain an optic nerve injury during the operation, therefore craniomaxillofacial surgeon treating orbital fractures must be aware of these potential complications and perform meticulous assessments of vision and globe function pre-, intra-, and postoperative.[25,3] **Kasey et al** reported an unusual case of blindness resulting from orbital compartment syndrome caused by retrobulbar hemorrhage following bilateral posterior maxillary segmental osteotomy.[23] **Lanigan et al** reported eight cases of ophthalmic complications associated with orthognathic surgery. In one case in which retrobulbar hemorrhage occurred following Le Fort I osteotomy the patient had no long term visual problem due to prompt intervention by the oral and maxillofacial surgeon and the ophthalmologist. Lanigan also noted that etiology of orbital injury might be due to force transmitted during the pterygo maxillary dysjunction using an osteotome or from fracture extending to the base of skull or orbit associated with the pterygo maxillary dysjunction or the maxillary down fracture.[26, 23] So basically, the trauma result in

oedema, bleeding, or both within the orbital space, which if severe can lead to increased pressure within the orbit. The tightly contained nature of the orbital anatomy restricts the amount of volume expansion and therefore gives rise to a compartment syndrome in a similar fashion to that seen in other body regions. In all causes, the 'final common pathway' is ischaemia and cellular hypoxia resulting in vison loss.[27]

There are various other causes of OCS other than trauma which are beyond the scope of this paper to discuss, as this article mainly focus on maxillofacial injuries related syndromes.

Clinical Signs & Symptoms

Examination of the patient should be performed quickly if OCS is suspected, so as not to delay treatment.[21] The clinical features of an OCS should be familiar to all surgeons operating in the periorbital area. The clinical findings include proptosis, ecchymosis of the eyelid, subconjunctival hemorrhage or chemosis, acute visual acuity deterioration, diplopia, decreased vison, limited ocular movements, pain, periocular edema, fixed dilated pupil or an afferent pupillary defect, increasing IOP, palpable tenderness of the orbital soft tissues with increase resistance to retropulsion of the globe, sensation may also be diminished in the distribution of the supra- and infra-orbital nerves.. If ophthalmoscopy can be performed, it may reveal optic disc and/ or retinal edema, retinal venous congestion, pulsation in the central retinal artery or central retinal artery occlusion (CRAO), retinal edema and optic nerve head swelling. Additionally, the presence of periorbital crepitus should provoke the suspicion of orbital emphysema, which is a traumatic cause of OCS.[3, 19, 23,]

Hueston suggested that during retrobulbar hemorrhage, the retina or optic nerve can tolerate ischemia for 60-90 minutes. If there is no immediate intervention permanent visual sequelae may occur.[28, 23] Experiments performed on rhesus monkeys indicate that retinal ischaemia of more than 100 min results in irreversible retinal damage, but that the retina can tolerate ischaemia up to 97 min though the duration before irreversible visual loss develops in human patients remains unclear.[27, 3]

Diagnosis

The diagnosis of OCS is often based on the history and clinical signs without the need for radiological imaging. Computed tomography imaging may be helpful in establishing the diagnosis in milder cases where there is uncertainty and vision remains intact. The imaging may help locate a hematoma, emphysema, foreign body or soft tissue expansion (to some degree). This can be useful to guide when considering further decompression of the orbit. When a vascular abnormality is thought to be the cause of OCS, magnetic resonance imaging (MRI) has an application. MRI with an angiography/venography protocol may assist in finding venous or arterial malformations, lymphangiomatosis of the orbit or carotid artery pathologies. Slit lamp examination can also be done which demonstrate features of ischaemia which includes retinal hemorrhage, microaneurysm, cotton wool spots and optic disc pallor. However, these findings are variable and maybe the result of numerous causes other than ischemia due to OCS. However when the diagnosis of OCS is clear based on clinical findings it should not be delayed for initial treatment.[21, 27]

Treatment

OCS is one of the few ophthalmic surgical emergencies whose diagnosis should be made clinically and treatment must be initiated immediately because of the risk of rapid, irreversible, vision loss.[3]

For cases in which the orbital intraocular pressure does not reach dangerously high levels, it is possible to manage patients medically with topical treatments, systemic carbonic anhydrase inhibitors and oral or intravenous

osmotic agents. However, in the setting of extremely high orbital pressures and obvious profound visual compromise, these medical measures are not sufficient in rapidly reducing the orbital pressure.[23] The effectiveness of systemic corticosteroids in OCS is controversial. Intravenous or oral corticosteroids may be used to provide neuroprotection if an inflammatory cause is responsible. OCS patients should be warned to avoid coughing/straining or taking antitussives, antiemetics, and laxatives. The head of the bed should be elevated to 45°. Cold/ice compression may reduce periorbital/ orbital edema. Blood pressure and coagulopathies should be normalized. All cases of OCS should be closely monitored for progression or recurrence.[19]

A common first-line approach for reducing intra-orbital pressure is a Lateral Canthotomy and

Cantholysis (LC/C). This can be done expediently at the bedside under local anesthesia.[21] It is also important to emphasise that lateral canthotomy alone does not adequately reduce the intraorbital pressure. Canthotomy must be combined with cantholysis to sufficiently reduce the orbital pressure. The benefit of LC/C is to effectively reduce the orbital septum diaphragm and permit anterior movement of the globe and reduction of the intraorbital pressure. It is important to realize that the resulting anterior movement of the globe is beneficial and does not usually cause a traction optic nerve injury in the setting of an orbital hemorrhage. The optic nerve and its dura are tightly adherent to the bony optic canal and the passive movement of the globe following the canthotomy and cantholysis does not cause traction at the level of optic chiasm.[23]

To perform a lateral canthotomy, 1 to 2 cc of a local anesthetic (1% lidocaine with 1/100.000 adrenaline) is injected into the Lateral Canthal Tendon (LCT). A tissue clamp is applied to the region for 30 seconds to 1 minute to achieve devascularization and hemostasis. The area around the lateral canthus is irrigated with saline or chlorhexidine. Scissors are carefully inserted in the lateral palpebral artery along the internal face of the lateral canthus. The incision of the skin and underlying eyelid tissue should be approximately 1 cm in length and extend to the lateral bony orbital rim. Maximum care and attention must be given while directing the scissors laterally and superficially to avoid iatrogenic injury to the globe. A lateral canthotomy achieves separation of the skin, fascial septum, orbicularis oculi muscle, and conjunctiva, and exposes orbital fat tissue. Although the LCT can be easily identified, a lateral canthotomy cannot accomplish a significant increase in the laxity of the eyelid.

A mnemonic "one is the number" has been proposed by some **authors** to highlight the important steps in a lateral canthotomy since it is an uncommon emergent procedure:

• 1 cc of 1% lidocaine with epinephrine for local anesthesia

• 1 minute hemostasis time

• 1 cm incision length. [19, 29]

For inferior cantholysis, following the exposure of orbital rim with lateral canthotomy the LCT is identified with palpation around the point of inferior insertion. The inferior crus of the LCT is isolated with inferior retraction of the lower lid. The tips of the scissors is directed away from the globe. Anterior traction is placed on the free lateral edge of the lower lid, and the inferior crus of the LCT can be identified as a taut band. Next, the inferior crus of the LCT is cut. The fibrous tarsal plate of the lower lid relaxes. This step allows for a significant decrease in intra-orbital volume and eventually decreases the INOP. The result is a completely mobile lower lid. Usually, the incision site will heal spontaneously and there is no need for suturing. However, if an oculoplastic or

cosmetic deformity occurs in the lower lid, further surgical repair may be needed. Significant relief of symptoms and a reduction of IOP typically occurs within several minutes of a successful LCIC. A lateral canthotomy alone can achieve an INOP reduction of approximately 14 mmHg, while an inferior cantholysis alone can reduce pressure by approximately 19 mmHg. The LCIC procedure can attain a pressure reduction of about 30 mmHg.[19]

Along with LC/C bony orbital decompression can be considered as an adjuvant procedure or as a secondary procedure if adequate response is not achieved after LC/C.[21]

Lee et al[30] and **Mootha et al**[31] highlighted the importance, not only of early treatment, but also of ensuring that adequate decompression has occurred. Both authors report cases where challenge remain when LC/C does not adequately decompress the orbit as additional decompression The decision to perform a secondary bony orbital decompression is not always straightforward; improvement in visual acuity may not be immediate after the LC/C despite an improvement in intra-orbital pressure. So secondary bony orbital decompression should also be considered in mind.[21,30,31]

Horner Syndrome

The Claude Bernard–Horner's syndrome (oculosympathetic paresis) is commonly known as Horner's syndrome (HS).[3]This syndrome was initially described in animals by the French physiologist Claude Bernard in 1854 and subsequently in a soldier who sustained a gunshot injury to his neck. However, Swiss ophthalmologist Johann Friedrich Horner is largely credited to be the first to completely describe this syndrome in 1869 and to correctly attribute it to oculosympathetic paresis .[32]

The basic triad of Horner's syndrome consist of relative miosis (pupillary constriction), slight ptosis (narrowing of the palpebral fissure) and slight enophthalmus. Other features, which are occasionally seen, include increase accommodative amplitude, transitory vasodilation of retinal, uveal, conjunctival and facial vessels, transitory lowering of intra-ocular pressure, an increase in temperature and sweating on the affected side of the face with some ultimate facial hemiatrophy and eventually slight discolouration of iris and cataract formation.

Anatomic Considerations

Anatomically, the sympathetic fibers in the posterolateral hypothalamus pass through the lateral brain stem and to the ciliospinal center of Budge and Waller in the intermediolateral gray column of the spinal cord at C8–T1. From this point, the preganglionic sympathetic neurons exit from the ciliospinal center of Budge and Waller and pass across the pulmonary apex and ascend up the carotid sheath to the superior cervical ganglion. The postganglionic sympathetic neurons originate in the superior cervical ganglion and travel up the wall of the internal carotid artery. Once the fibers reach the cavernous sinus, they travel with the abducens nerve before joining the ophthalmic division of the trigeminal nerve and entering the orbit with its nasociliary branch. From here, they divide into two long ciliary nerves to reach the iris dilator muscle. Disruption of this pathway thus can occur in any of the three levels, hence giving rise to central (first-order neuron) HS, preganglionic (second-order neuron) HS, and postganglionic (third-order neuron) HS.[3]

Etiology

Central Horner's syndrome is uncommon. Most commonly, it results from infarction of the vascular territories of the posterior inferior cerebellar artery or distal vertebral artery territory.

Postganglionic Horner's syndrome may result from lesions involving the internal carotid artery, skull base or cavernous sinus/orbital apex. Third, fifth and sixth cranial nerve palsies suggest lesions involving the cavernous sinus. Preganglionic Horner's syndrome may result from Injury to the brachial plexus or spinal roots, pneumothorax, fracture of the first rib, or neck hematoma.[34]

In 1958, Giles and Henderson reported a review of 216 cases of the syndrome seen during a 21-year period. Lesions of the cervical sympathetic trunk accounted for 46.8 per cent of cases, in 19.9 per cent, the thoracic spinal roots were affected and in 9.2 per cent, the lesions were in the central nervous system. Neoplasia accounted for 35.6 per cent of all cases; bronchial carcinoma and metastatic carcinoma from breast tumours were the most common. 18.5 per cent of lesions were the result of operative trauma and 13 per cent were attributed to other trauma. In the latter group birth trauma was responsible in 12 cases whilst various other traumatic episodes accounted for a further 16 cases. Vascular disease of the brain stem was the cause in 4.6 per cent of cases. Other minor causes included multiple sclerosis, caudal analgesia, congenital malformation, encephalitis, tuberculosis, chronic infective granuloma, syringomyelia, tabes dorsalis and herpes zoster.

Simpson (1948) summarised the aetiology of Horner's Syndrome and reported a case in which an osteochondroma of the first rib produced the syndrome. Other authors in relation to facial trauma have noted Horner's Syndrome. In 1970, Kannen and Belifente reported a case in which the patient received a gunshot wound. The mandibular symphysis was fractured and the bullet lodged adjacent to the carotid sheath interrupting cervical sympathetic activity. Pruett recorded another case (1967) in which the syndrome was seen in a five-year-old child who had sustained an injury to the tonsillar fossa.[33]

Among the several causes of Horner syndrome, trauma is also included, especially of the neck, shoulder, and chest wall. Trauma has also been documented to account for <1% of all cases of Horner syndrome and secondary to maxillofacial trauma may be a very rare phenomenon.[35,36] Worthington and Snape reported a case of Horner syndrome in a 17-year-old girl with cranial nerve involvement, as an unusual manifestation of skull base fracture in a patient with maxillofacial injuries. In this case, Horner syndrome associated with basilar skull fractures is most likely caused by trauma to the postganglionic pericarotid plexus as it traverses the carotid canal within the petrous bone.[37] Pruett reported another case of Horner syndrome associated with maxillofacial injury.[3,36]

Clinical Signs & Symptoms

Ptosis

The ipsilateral upper eyelid appears slightly drooped due to paresis of the Muller muscle, a sympathetically innervated smooth muscle that also functions as an upper eyelid retractor and is responsible for about 2 mm of upper eyelid elevation. Nonetheless, this ptosis may be subtle, variable, and go unnoticed. In addition, one study noted that in 12% of patients with Horner syndrome, the ptosis was in fact absent.[32]

Enophthalmos

Atrophy of the orbital fat and weakness of the muscle of Muller have been cited as causes of enophthalmos in Horner's Syndrome.[33] The smooth muscle fibers of the lower eyelid retractors also lose their sympathetic supply in patients with Horner syndrome and, thus, the lower eyelid appears slightly elevated. This appearance has been termed "upside-down ptosis" or "reverse ptosis". The combination of the upper eyelid ptosis and the lower eyelid elevation narrows the palpebral fissure, giving rise to an apparent enophthalmos. Several authors have since

proven that this apparent enophthalmos is not of measurable significance, and hence is not true enophthalmos.[32]

Miosis

A balance of the dilator pupillae and sphincter pupillae in the iris determines the pupil size. Sympathetic and parasympathetic nerves respectively innervate them.[32]The unopposed parasympathetic action of the iris constrictor muscle produces a smaller ipsilateral pupil. Anisocoria is most apparent in darkness and may in fact overlook the resulting if the patient is evaluated in bright light. Several factors influence the degree of anisocoria in patients with Horner syndrome. Paresis of the iris dilator muscle also impairs pupillary movement during dilation, termed dilation lag. Dilation lag can be seen clinically by illuminating the patient's eyes tangentially from below with a hand-held flashlight and then abruptly turning the room lights out. The normal pupil will immediately dilate, but the Horner pupil begins to dilate several seconds later. The difference in anisocoria is greatest after 4–5 seconds in the darkness. Thereafter, the Horner pupil slowly dilates from decreasing parasympathetic tone and eventually catches up in size to the normal pupil. Thus, if both pupils are observed simultaneously for 15–20 seconds after turning off the room light, one sees an initial increase in the degree of anisocoria, followed by decreasing anisocoria. Thus, a diagnosis of Horner syndrome should not eliminated in the absence of a demonstrable dilation lag of the smaller pupil.

Iris hypochromia

Sympathetic innervation is thought to be required for the formation of melanin by stromal melanocytes. Interruption of the sympathetic supply can lead to iris hypochromia on the affected side. This is a typical feature of congenital Horner syndrome. It is also occasionally seen in patients with a long-standing, acquired Horner syndrome, but never in patients with an acute or recently acquired Horner syndrome.

Accommodation

A few authors have noted that patients with Horner syndrome can experience an increase in accommodative amplitude on the ipsilateral side.

Anhidrosis

Characteristic vasomotor and sudomotor changes of the facial skin can occur on the affected side in some patients with Horner syndrome. Immediately following sympathetic denervation, the temperature of the skin rises on the side of the lesion because of loss of vasomotor control and consequent dilation of blood vessels. Additionally, there may be facial flushing, conjunctival hyperemia, epiphora, and nasal stuffiness in the acute stage. Some time after the injury, the skin of the ipsilateral face and neck may have a lower temperature and may be paler than that of the normal side. This occurs from supersensitivity of the denervated blood vessels to circulating adrenergic substances, with resultant vasoconstriction. However, in modern temperature-controlled spaces, patients with Horner syndrome rarely complain of disturbances of sweating or asymmetric facial flushing.

Paradoxical unilateral sweating with flushing of the face, neck, shoulder, and arm can be a late development in patients with a surgically induced Horner syndrome following cervical sympathectomy. Some axons in the vagus nerve normally pass into the superior cervical ganglion. These parasympathetic axons can establish, by collateral sprouting, anomalous vagal connections with postganglionic sympathetic neurons to the head and neck.[32]

Diagnosis

The diagnosis of Horner syndrome should be considered in any patient with anisocoria, presence of dilation lag,

anhydrosis, ptosis and other associated symptoms. Patients should be evaluated for evidence of cranial nerve dysfunction, particularly an ipsilateral abducens nerve paresis that may indicate a lesion of the cavernous sinus or, in very rare cases, of the brain stem.[32] Proper neurological and ophthalmic assessment should be done along with any history of trauma and anesthetic problems.[33]

Topical pharmacologic agents such as cocaine, phenylephrine, apraclonidine, or hydroxyamphetamine can be used to confirm a diagnosis of Horner syndrome.[3]

Once the diagnosis of a Horner syndrome has been confirmed, an appropriate evaluation should be performed. As noted above, in infants and children, this should involve a complete physical examination, magnetic resonance imaging of the brain, neck, and chest, and an assay for urinary catecholamines. In adults with an acquired Horner syndrome, a simple x-ray of the neck in flexion and extension can identify cervical spondylosis, a common cause of both central and preganglionic Horner syndromes. In addition, magnetic resonance imaging of the brain with contrast should reveal many other causes of a central or postganglionic Horner syndrome.[32]

Treatment

Due to rarity of the cases of traumatic HS, its management is not well defined. Some authors have managed the condition by observation. However, when it is caused by the compression of the oculosympathetic pathway, the cause of the compression should be treated whenever possible.[3,38]

Posttraumatic Guillain–Barre syndrome

Guillain-Barre syndrome (GBS) is a multifactorial and lethal inflammatory demyelinating polyradiculopathy and polyneuropathy, characterized by flaccid paralysis and acute demyelinating changes in the peripheral nervous system.[39,40,41] Since GBS has also been reported to be triggered by non-infectious factors such as trauma, the concept of posttraumatic GBS was introduced.[3]

This rare syndrome affects the peripheral nerve myelin sheets. There have been rare reports of GBS after head or brachial plexus trauma, general anesthesia, neurosurgical, orthopaedic, caesarean section, laparoscopy and general surgery; but the occurrence of GBS following oral and maxillofacial surgery is not common.[42]

GBS is classified into various subtypes that differ in their clinical, electrophysiological, and histological features, the commonest being subgroups of acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy.[3]

The development of GBS following head injury or cranial surgery has been infrequently reported. Tan et al. reported a case with closed head trauma resulting in a comminuted fracture of the frontal and temporal bones, frontal lobe contusion and subarachnoid haemorrhage. Wu et al. reported two cases with cerebral haemorrhagic stroke (spontaneous subarachnoid hemorrhage and middle cerebral artery infarction) developed progressive flaccid quadriplegia diagnosed as GBS.[43]

The requirement for establishing a temporal relationship between a traumatic event and subsequent neuropathy is that the neuropathic symptoms must start within 30 days of the trauma.[39]

Pathogenesis

The main pathological pathway for GBS is a triggered immune-mediated response resulting in multifocal mononuclear cell infiltration throughout the peripheral nervous system. It is postulated that the underlying mechanisms are based on a trauma-related disruption of the cellular and humoral immune system. The antigens targeted in AMAN are located at or near the node of Ranvier. The anti-GM1 and anti-GD1a antibodies bind to the nodal axolemma, leading to complement activation

followed by membrane attack complex (MAC) formation and disappearance of voltage-gated sodium channels. This damage can lead to the detachment of paranodal myelin and nerve conduction failure. Macrophages then invade from the nodes into the periaxonal space, scavenging the injured axons. In case of AIDP, the targeted antigens are, presumably, located on the myelin sheath. The antibodies can activate complement, which leads to the formation of the MAC on the outer surface of Schwann cells, initiation of vesicular degeneration, and invasion of myelin by macrophages.[3]

Carr et al postulated that disintegration of the blood–brain barrier during neurotrauma allows the accumulation of leukocytes from systemic circulation including T-lymphocytes and macrophages, which in turn induce native glia to function as antigen-presenting cells. Activated microglia act as scavenger cells and remove posttraumatic neuronal debris, and thus present certain neuronal cell components as antigens to the immune system.[45,3]

The pathogenesis of GBS following head trauma or surgery is not clear. Tan suggested that

head trauma and surgery had elevated serum and CSF myelin basic protein levels. These

proteins led to immune system activation to produce anti-myelin antibodies that cause

demyelination. Also blood-brain barrier damage has an important role in this pathogenesis.[44,46,]

Signs And Symptoms

GBS is characterized by an acute onset of muscular weakness, decrease or absent deep tendon reflexes. In severe cases, however, the swallowing and respiratory muscles may be involved, familial periodic paralysis accompanied by thyroid disease. It also include the muscle weakness of trunk, neck, and face, hypotonia, hyporeflexia, or areflexia, paresthesia, and ophthalmoplegia. GBS is highly diverse with respect to the presence, distribution, and extent of cranial nerve deficits, sensory symptoms, weakness, ataxia, pain, autonomic dysfunction[42,3]

Diagnosis

The diagnostic workup for patients with GBS include brain and spinal cord imaging to exclude a structural cause, followed by lumbar puncture, which characteristically demonstrates raised cerebrospinal fluid protein in the absence of inflammatory cells. Nerve conduction studies (NCS) help to confirm the diagnosis, but, like cerebrospinal fluid, they are non diagnostic in up to 50% of patients in the 1st week of disease. The presence of antiganglioside (IgG) antibodies supports diagnosis. NCS can help to support the clinical diagnosis of GBS and discriminate between axonal and demyelinating subtypes.[3]

Facial nerve deficits were described in the majority of reported cases. Electrophysiologic study can confirm the presence and classification of GBS by detecting abnormally prolonged distal latencies and reduced amplitudes on conduction studies (NCS).[43]

Treatment

Treatment of GBS usually combines multidisciplinary supportive medical care and immunotherapy. Proven effective treatments for GBS are intravenous immunoglobulin and plasma exchange. Unlike many inflammatory conditions, corticosteroids are of no benefit in GBS.[47,3]

Frey's Syndrome

Frey syndrome was first reported by Frey in the year 1923. The syndrome which usually occurs after parotidectomy is characterized by unilateral localized facial flushing and sweating over the sensory distribution of the auriculo temporal nerve in response to the ingestion of certain foods.[48]

Frey syndrome is a postoperative phenomenon following salivary gland surgery and less commonly neck dissection, facelift procedures, and trauma that is characterized by gustatory sweating and flushing. Frey syndrome was first described by Lucie Frey in 1923 and was termed auriculotemporal syndrome.[49,3] It described sweating and flushing in the preauricular area in response to mastication or a salivary stimulus. Initially thought to be rare, it was later recognized as a common occurrence after salivary gland surgery. The synkinetic mechanism for Frey syndrome is aberrant reinnervation of postganglionic parasympathetic neurons to nearby denervated sweat glands and cutaneous blood vessels.Consequently, this results in flushing and sweating in the sympathetically void skin in response to mastication and salivation.[3]

The symptoms of Frey syndrome can include flushing, sweating, burning, neuralgia, and itching. Generally, the symptoms are mild but can result in discomfort as well as social anxiety and avoidance.[50,3]

Anatomy

The auriculotemporal nerve runs partly in the groove formed by the posterior edge of the ascending mandibular ramus and the cartilage of the external auditory canal. It then gives branches over the preauricular and temporal areas. The parasympathetic chain to parotid consists ofthe preganglionic fibersthat originate in the inferior salivatory nucleus and travels with the glossopharyngeal nerve, tympanic nerve, and lesser petrosal nerve to the otic ganglion. The postganglionic fibers join branches of the mandibular nerve (primarily the auriculotemporal and buccal nerves) to supply the parotid gland and nearby mucous glands with secretomotor fibers and also supply the vasculature with vasodilator fibers. On the other hand, the postganglionic sympathetic neurons originate in the superior cervical ganglion. These fibers project in a plexus around branches of the carotid artery (middle meningeal artery). Bundles of fibers periodically leave this plexus and traverse the otic ganglion without synapsing to join branches of themandibular nerve, ultimately to supply blood vessels, glands, and other tissue.[49,3]

Pathophysiology

Through the years, several theories have been proposed to explain its existence.

- Frey suggested the invasion theory that is the damaged auriculotemporal nerve (ATN) is invaded and irritated by healing tissue.
- Freedberg believed that damage to the nerve may cause destruction of sympathetic fibers, leading to theory of parasympathetic hypersensitivity and stimulation.
- But the theory of aberrant regeneration by Ford and Woodhall, which is based on defective nervous regeneration, has gained wide acceptance. However, its exact mechanism is yet to be elucidated.

The syndrome's pathophysiology is explained on the basis of damage to the ATN and subsequent reinnervation of sweat glands by parasympathetic (salivary) fibers and some form of transaxonal excitation from adjacent fibers or ganglion. However, Glaister et al, following parotidectomy, proposed the "reverse phenomenon" of pathological saliva production, caused by misdirected sympathetic fibers in the region of parotid remnant's"

The more acceptable theory is called the Theory of Aberrant Regeneration by André Thomas, which is based on defective nervous regeneration. Due to trauma, the postganglionic parasympathetic fibers of the auriculotemporal nerve supplying the parotid gland are sectioned. In addition, the sympathetic fibers that supply local sweat glands are also interrupted. It is believed that the severed parasympathetic fibers regenerate and connect with severed distal sympathetic nerves that innervate subcutaneous sweat glands. The misdirection of

regenerating parasympathetic fibers to denervated sweat glands and cutaneous blood vessels, consequently, causes salivation to be accompanied by flushing and sweating in the sympathetically denervated region of skin.Parasympathetic and sympathetic nervefibers can undergo crossregeneration because both use acetylcholine as a neurotransmitter.

Scarred gland tissue causes excessive tension during parotid activity, consequently leading to irritation of sympathetic fibers traversing the gland in the auriculotemporal nerve

Abnormal irritability of the cholinergic nerve endings results when the regeneration attempt of fibers of the auriculotemporal nerve is strangulated by scar tissue in the parotid gland.

The auriculotemporal syndrome normally exists as a spontaneous effect, deprived of inhibitory fibers by damage to the nerve supply of the area. [48,49,50,3]

Etiology

This condition is more commonly seen following penetrating wounds and chronic infections of the parotid region, parotidectomy, jugular-carotid lymph node dissection, submandibular gland surgery, TMJ surgery, mandibular and zygomatic fractures, and cervicothoracic sympathectomy.[49,50]

However, few cases describing the onset of Frey's syndrome following fractures of the lower jaw, most notably the condyle, have been reported in the literature. One such case was reported as early as 1969 by Martis and Athanassiades, and by Pansino in 1971. In 1982, Tuinzing et al described the syndrome as a complication following a sagittal ramus osteotomy.[50]

More recently, however, Tue et al made an extensive literature review (10 case reports and one prospective study; 1969 to 2010) and concluded that trauma of considerable impact, the existence of more than one mandibular fracture site, dislocation of the condyle, and altered sensibility in the preauricular region appear as major risk factors for Frey's syndrome. All these events lend great support to the correlation between fracture/fracture-dislocation of the condyle and onset of Frey's syndrome (due to the intimate anatomical relationship between the ATN and the TMJ). [48,49]

On the other hand, this syndrome has developed in few congenital cases; these have been explained based on certain theories that include: (1) congenital aberration, with a crossed cranial nerve pathway; (2) loss of insulation around the neural sheaths leading to crossover of nerve fibers; (3) neural irritation caused by scar tissue formation after local trauma; (4) subclinical viral infection; and (5) mild unreported injury to the parotid gland.[50]

Diagnosis

It is based on clinical history, but confirmatory testing can be done with a Minor starch-iodine test. The starch-iodine test consists of painting the patient's postsurgical affected region with iodine. When the painted area becomes dry, it is covered with starch, and the patient is asked to eat something sour to induce gustatory sweating, thus the skin area involved by Frey's syndrome turns purple when the sweat gland's secretions react with the starch and iodine. Patients who underwent parotidectomy had a positive Minor starchiodine test in 62% of cases, whereas the self-reported incidence of symptoms was only 23% in the same group. These numbers attest both to the high incidence of the synkinesis and to the subclinical nature of Frey syndrome.[49,3]

Surgical methods for the prevention of frey syndrome

Prevention of Frey syndrome has been guided by the alteration of surgical techniques or the addition of procedures focused on preventing synkinesis. The overarching theme for the surgical prevention of Frey

syndrome has been the incorporation and maintenance of a barrier between the underlying postganglionic parasympathetic nerve endings within the transected parotid and the overlying cutaneous tissue. Many techniques aimed at accomplishing this have been described and include increased skin flap thickness, local fascia or muscle flaps, and the use of acellular dermal matrix (ADM) or free fat grafts like-

Increased Skin Flap Thickness, Transposition Muscle or Fascia Flaps, Temporoparietal fascia flap, Sternocleidomastoid muscle flap, Superficial musculoaponeurotic system flap, Biomaterial and Autologous Implantation, Acellular dermal matrix, Abdominal fat grafting :

Postsurgical treatment of frey syndrome

Medical Management: Although intraoperative techniques try to reduce severity and incidence of Frey syndrome, postoperative interventions have been focused on ameliorating symptoms once they develop. Most of the therapies used are given via injection therapy or by topical application. Previous agents have included topical antiperspirants as well as injection with alcohol, scopolamine, glycopyrrolate, or botulinum toxin A (BTA). Currently, BTA is the most widely used agent for intradermal injection. Previous studies have demonstrated that patients undergoing BTA injection demonstrate improvement in symptoms of gustatory sweating and flushing. Inaddition, it has been shown to improve patient quality of life.However, despite a high rate of return symptoms after BTA injection, repeat BTA injection has been shown to be effective. For the studies investigating BTA, the injection dose was between 1.9 and 2.5 U/cm2 in the involved area.

Surgical Management : Reports of surgical transection of the auriculotemporal nerve, tympanic nerve, and greater auricular nerve have been described for the management of Frey syndrome, but they are not commonly practiced. Recently, a cohort of 17 patients with postparotidectomy Frey syndrome who underwent both SCM and temporalis fascia transposition was reported by Dia and colleagues. This report demonstrated that greater than 50% (9/17) of patients who underwent the transposition procedure had complete resolution by starch-iodine testing.60 In addition, there was a significant reduction in the average surface area of gustatory-sweating–positive skin from 12.80 to 1.32 cm^2 in all patients postoperatively. Although this method is compelling and does appear to be a feasible option for surgical management of Frey syndrome, it does have an increased risk for facial nerve injury. However, if surgery for Frey syndrome is to be attempted, it should be only be used in cases that are refractory to conservative nonsurgical measures.[48, 49,50,3]

Conclusion

Although the syndromes associated with maxillofacial injury is very rare, but the maxiilofacial surgeon should be always careful while clinically examining the maxillofacial trauma patient as it might be associated with any of the syndrome. This article will give a brief knowledge of syndromes associated with maxillofacial injury and be helpful to the clinicians and surgeons to have a high-suspicious eye when they encounter a patient with maxillofacial injuries.

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