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REVIEW ARTICLE

Syndromes in Maxillofacial Injuries

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A substantial proportion of traumatology involves the maxillofacial region. Injuries to this region are associated with several complications, among them being the different syndromes. These syndromes may arise depending on not only the localization of the initial injury, but also from the treatment itself. This review article aims to bring into the attention of medical community different syndromes that may be associated with maxillofacial injuries.

Key words: Maxillofacial injuries, superior orbital fissure syndrome, Horner's syndrome, Guillain–Barré syndrome, Frey's syndrome

INTRODUCTION

A substantial proportion of traumatology involves the maxillofacial region.[1] Maxillofacial injuries can occur due to a number of etiological factors,[2,3] which in most instances determine the pattern and severity of the injury sustained to this area.[1] The basic form of the maxillofacial region is determined by the hard tissues, while the skin, mucosa, and the underlying soft tissues in between create a soft-tissue envelope. These anatomical sites ultimately have been commonly used in epidemiological studies to classify the maxillofacial trauma.[1]

Injuries to this highly vascular zone are complicated by the presence of upper airway and proximity of the cranial and cervical structures that may be concomitantly involved.[4] Many authors support the notion that maxillofacial trauma alone is rarely life threatening unless associated with airway compromise.[4,5] Apart from the airway compromise, some other causes of the life-threatening complications following trauma to the maxillofacial region may be due to massive bleeding or undiagnosed cervical spine injury.[5]

However, there are situations in which some complications of the trauma may arise depending on not only the localization of the initial injury, but also from the treatment itself.[6] Among these complications are the injury-associated syndromes. These syndromes can either be due to direct effect of the trauma that leads to entrapment/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 and/or its management.

Literature search revealed few reported cases of syndromes associated with maxillofacial injuries; hence, this article aimed at conducting a narrative review of these rare conditions which can otherwise in some instances be overlooked in general practice. The few encountered syndromes in literature are thus discussed below, focusing on the pathogenesis and clinical presentation.

SYNDROMES ASSOCIATED WITH MAXILLOFACIAL INJURIES

Superior orbital fissure syndrome and orbital apex syndrome

The term superior orbital fissure syndrome (SOFS) or Rochon–Duvigneaud syndrome is applied to lesions located immediately anterior to the orbital apex, associated with impaired function of the cranial nerves (III, IV, V, and VI) that enter the orbit through the SOF.[7-9] An orbital apex syndrome (OAS) is a syndrome involving the same cranial nerves as in SOFS, but there is associated optic nerve dysfunction.[8,9]

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Anatomically [Figure 1], the SOF, which is a pear-shaped structure with the broadest part at the nasal side, is bound laterally by the greater wing of the sphenoid, medially by the lesser wing of sphenoid, and superiorly by the frontal bone, lying at the apex of the orbit and at the border between the roof and the lateral orbital wall.[10] The SOF serves as a pathway that allows communication between the orbit and the middle cranial fossa, thereby transmitting the oculomotor, trochlear, and abducens nerves (cranial nerves III, IV, and VI), as well as the first three branches of the trigeminal nerve: the frontal, lacrimal, and nasociliary nerves.[11]

Two pathogenetic mechanisms can be identified; the direct, which is when the nerves traversing the fissure are interrupted or compressed by displaced bone fragments.[7,12] 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.[10,12] The symptoms following the mechanisms described may either be complete or partial depending on the degree of compression of anatomical structures.

Clinical symptoms of the SOFS can be explained by the nerve involved, thus these symptoms may include some or all of the following: ophthalmoplegia, ptosis, proptosis, fixed and dilated pupils, anesthesia/paresthesia of forehead, hypolacrimation, diplopia, and decreased visual acuity.[10,13,14] External ophthalmoplegia occurs because of hypofunction of the oculomotor, trochlear, and abducens nerves, which may also explain the resulting diplopia.[10,11,14]

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.[11] The fixed dilated pupil which presents with dilatation, fixation, and loss of accommodation of the ipsilateral pupil arises due to disruption of the parasympathetic fibers coursing with the oculomotor nerve, resulting in paralysis of the pupillary ciliary muscle.[11,14]

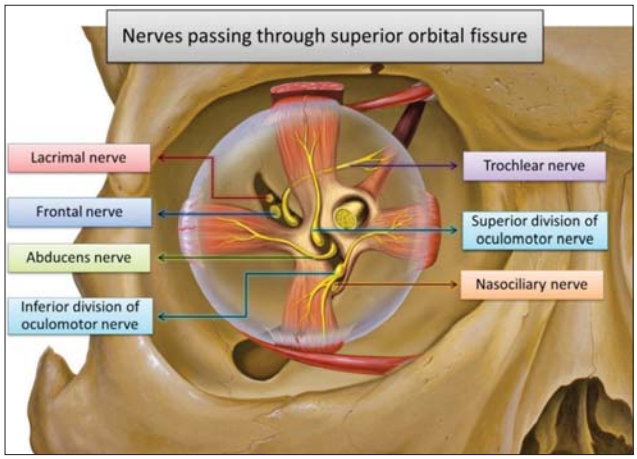

Figure 1: Orbital apex viewed anteriorly (adapted from https://pgblazer.com/wp-content/uploads/2010/08/Nerves-passing-through-superior-orbital-fissure2.jpg)

Compromise of the lacrimal and frontal nerves of the ophthalmic branches of the trigeminal nerve results in anesthesia of the forehead and upper eyelid, lacrimal hyposecretion, and possibly neuralgia along the path of the nerve, whereas, anesthesia of the cornea and the bridge of the nose with loss of the corneal reflex is attributed to the disruption of the sensory nasociliary nerve.,[10,14] The loss of tone of the extraocular muscles which normally exert a retracting force on the globe leads to outward protrusion of the eye (proptosis).[10] When the optic nerve is also involved including the above-mentioned symptoms, the condition is considered as the orbital apex syndrome, a term coined by Kjaer.[8-10]

SOFS is an uncommon complication after craniofacial trauma and is usually due to bony injury, foreign bodies, or hemorrhage at the orbital apex.[14] It is a rare condition that may accompany facial fractures of the Le Fort types II and III, frontobasal skull fractures, zygomatic fractures, and orbital fractures.[12] The reported incidence of traumatic SOFS is only 0.3%–0.8%, with incidence of traumatic OAS being much lower.[15]

SOFS and OAS have been documented in few articles, most of them being case reports. Deda and Demirci[12] reported on the case of SOFS in a 25-year-old male who had zygomaticomaxillary complex (ZMC) fracture following an automobile accident. In another case report, Evans *et al*.[11] described a case of a 44-year-old man who fell down concrete steps who was as well diagnosed with ZMC fracture with associated SOFS. In another case study, a 41-year-old man was diagnosed with SOFS after he was shot in the face with a handgun and sustained multiple comminuted fractures of the left orbit, with the bullet lodged near the SOF.[16] The SOFS is not always associated with trauma directly, but at times as a complication of management of facial bone fractures, as described in a case report by Fujiwara *et al*.[17]

OAS is as well a rare condition that is associated with the maxillofacial trauma. Sugamata[18] described two cases of OAS secondary to the fracture of orbit and, in both cases, the medial orbital wall was involved. Imaizumi *et al*.,[15] on the other hand, reported a case of traumatic OAS combined with the blow-in type of the orbital and ZMC fractures, in a 24-year-old male who sustained injury after a bicycle crash.

The rarity of traumatic OAS, as well as traumatic SOFS, has made it difficult to define treatment guidelines for this

condition.[15] The rationale behind the treatment of SOFS of traumatic origin lies primarily in minimizing further irreparable damage to the neuronal structures.[7] A general consensus lies toward the initial observation period of 10–14 days before any surgical manipulation of fracture segments is done since it prevents any hazard of further hemorrhage and involvement of the orbital apex or injury to the other nerves.[7] Varying doses of systemic corticosteroids have been advocated as treatment alone, or in conjunction with other modalities such as facial fracture reduction.[11] Surgical intervention may be indicated in the presence of significant narrowing of the SOF from the displaced fracture fragment.[10] In the cases of traumatic OAS, currently, the following three treatment options exist: observation alone, high-dose corticosteroids, and surgical optic canal decompression.[14]

Horner's syndrome

The Claude Bernard–Horner's syndrome (oculosympathetic paresis), commonly known as Horner's syndrome (HS), is due to injury to the sympathetic chain running over the carotid vessels.[19] It occurs when there is interruption of the oculosympathetic pathway which supplies sympathetic innervation to the sweat glands (ipsilateral body and face), dilator muscles of the eye, and retractor muscles of the upper and lower eyelids.[20]

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 [Figure 2].[20,21]

Clinical features of this syndrome include ptosis, miosis, and anhidrosis. Ptosis refers to a moderate drop of the upper eyelid. The ipsilateral upper eyelid appears slightly drooped due to paresis of the Müller muscle, a sympathetically innervated smooth muscle that also functions as an upper eyelid retractor. The smooth muscle fibers of the lower eyelid retractors may as well lose their sympathetic supply in

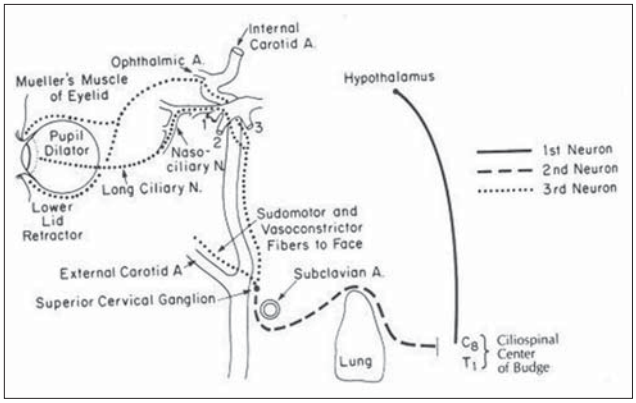

Figure 2: Neural pathway of sympathetic fibers (adapted from http://telemedicine.orbis. org/data/1/rec_imgs/5648_fig.%203.6.jpg)

patients with HS and thus, the lower eyelid appears slightly elevated. The combination of the upper-eyelid ptosis and the lower-eyelid elevation narrows the palpebral fissure, giving rise to an apparent enophthalmos.[20-22]

Miosis is a decrease in pupil size as a result of paralysis of the iris dilator muscles. The sphincter and dilator muscles of the pupil are innervated, respectively, by the sympathetic and parasympathetic systems. When the sympathetic system is interrupted, there are no forces to counteract the sphincter muscle; therefore, the pupil will decrease in size. Anisocoria is more pronounced in the dark, when the iris dilator should be acting, and may be most obvious within the first 5 s of the darkness. After 10–15 s in the dark, anisocoria will be less apparent (known as dilation lag).[20,22,23]

Anhidrosis occurs when there is interruption of sympathetic innervation to the sweat glands, resulting in a lack of sweat production. Unilateral absence of sweat to the forehead, face, or body is a good indication of HS. Different patterns in the distribution of anhidrosis are associated with first-order HS, second-order HS, and third-order HS. Anhidrosis is often not readily noticed by patients and it can be difficult to diagnose, thus it is not a routinely measurable sign.[20-22]

Among the several causes of HS, trauma is also included, especially of the neck, shoulder, and chest wall.[21-24] Trauma has been documented to account for <1% of all cases of HS[24,25] and secondary to maxillofacial trauma may be a very rare phenomenon. Worthington and Snape[26] reported a case of HS 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, HS 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. Another case of HS associated with maxillofacial injury was reported by Pruett.[25] The described case was of a 5-year-old child who sustained intraoral trauma

while running with an object in the mouth, following injury; he was eventually diagnosed with HS secondary to blunt intraoral trauma to the internal carotid sympathetic plexus.

Topical pharmacologic agents such as cocaine, phenylephrine, apraclonidine, or hydroxyamphetamine can be used to confirm a diagnosis of HS.[21] Cocaine acts by blocking the reuptake of norepinephrine in the neuromuscular junction of the iris dilator muscle, causing the pupil in a normal eye to dilate, while incomplete dilation is seen in HS.[21,27]

Due to rarity of the cases of traumatic HS, its management is not well defined. Some authors have managed the condition by observation.[25,27] However, when it is caused by the compression of the oculosympathetic pathway, the cause of the compression should be treated whenever possible.[27]

Orbital compartment syndrome

Orbital compartment syndrome (OCS) is a rare but potentially devastating ophthalmic emergency caused by a rise in intraorbital pressure (IOP) requiring emergent orbital decompression to preserve vision.[28] Typically, an IOP >20 mmHg is considered elevated, and an increased IOP may compress the optic nerve directly or cause compression of its vasculature.[29]

The orbit is a cone-shaped structure formed by rigid bony walls. It has a volume of approximately 30 ml, within which the globe, fat, extraocular muscles, vessels, nerves, lacrimal gland, lacrimal sac, and retrobulbar contents are encased within complex fascial compartments.[30,31] The globe is limited anteriorly by the orbital septum, eyelids, and by the attachment of the medial and lateral canthal tendons.[29,31]

The pathogenetic 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.[30,31] Both the tissue and venous pressure increases when fluid (e.g. blood) enters a fixed-volume compartment. When these pressures exceed the capillary perfusion pressure, capillaries will collapse, thereby causing ischemia to muscles and nerves.[32] 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.[29] 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 emergently.[33]

Clinically, patients typically present with acute visual acuity deterioration, diplopia, limited ocular movements, pain, proptosis, periocular edema, and ecchymosis, over minutes to hours. Despite previous animal studies demonstrating permanent retinal damage after 105 min of retinal ischemia, the duration before irreversible visual loss develops in human patients remains unclear,[28,30] though raised IOPs lasting for just 60–100 min can lead to permanent visual sequelae.[34] Decreasing visual acuity (for all practical purposes, the vital sign of the eye) over time should be considered one of the most alarming factors.[33]

The most common cause of OCS is trauma.[28] There are several published articles regarding the occurrence of OCS in patients who had sustained maxillofacial injuries. Sun *et al*.[28] did a retrospective study of patients that presented with OCS secondary to blunt trauma. In their study, majority of the patients had sustained fracture of the orbit, with some having concomitant ZMC, and Le Fort I, II, and III fractures. Carrim *et al*.,[31] on the other hand, reported a case of a 27-year-old man who had been assaulted and received a direct blow to the left orbit. The patient was diagnosed with OCS, despite having no any obvious fracture of the facial bones. Kloss and Patel[34] described a case of OCS in a 48-year-old male, who sustained facial and eye trauma after having been struck with a falling tree branch. In some cases, patients may develop OCS secondary to the management of facial bone fractures. Such an incidence was reported by Susarla *et al*.[35]

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. Computed tomography is commonly used to further evaluate the orbits as it is often immediately available unlike magnetic resonance imaging which often cannot be obtained in a timely manner.[30] Lateral canthotomy is regarded as a first-line intervention for decompression of the orbit in OCS,[31] and preferably, it should be performed within an hour or 2 h as it was suggested by some authors.[28,29] Immediate lateral canthotomy and inferior cantholysis are recommended to prevent visual loss owing to optic nerve compression in OCS. The major goal of these external procedures is to reduce the IOP and reestablish retinal arterial blood flow by allowing anterior prolapse of the orbital contents.[29]

Posttraumatic Guillain–Barré syndrome

Guillain–Barré syndrome (GBS) is a multifactorial and lethal inflammatory demyelinating neuronal disorder with concurrent polyradiculopathy and polyneuropathy, characterized by flaccid paralysis and acute demyelinating

changes in the peripheral nervous system.[36,37] Since GBS has also been reported to be triggered by noninfectious factors such as trauma, the concept of posttraumatic GBS was introduced. 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 (AMAN).[36,38]

The main pathological pathway for GBS is a triggered immune-mediated response resulting in multifocal mononuclear cell infiltration throughout the peripheral nervous system.[37] In the case of posttraumatic GBS, 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.[37,38]

Carr *et al*.[39] 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.

The most frequently encountered clinical picture consists of relatively symmetrical muscular weakness that begins distally in the lower limbs, followed a few days later by the involvement of more proximal muscles such as those of the trunk, neck, and face, hypotonia, hyporeflexia, or areflexia, paresthesia, and opthalmoplegia.[38,40] GBS is highly diverse with respect to the presence, distribution, and extent of cranial nerve deficits, sensory symptoms, weakness, ataxia, pain, autonomic dysfunction, and the course of the disease.[38]

Although a range of infectious factors are associated with GBS, it has also been reported to be triggered by noninfectious factors such as trauma, as described by Li *et al*.,[36] who reported several cases of GBS in trauma patients. Though it is very rare, maxillofacial trauma has also been documented to trigger GBS. Lin *et al*.[40] reported a case of GBS in a 22-year-old female patient who sustained panfacial injuries following motor traffic crash. In this case, the symptoms of GBS became apparent 10 days postinjury. In another case report, Carr *et al*.[39] reported a case of posttraumatic GBS in a 58-year-old man who sustained multiple facial bone fractures and traumatic brain injury after he was hit by a falling branch of a tree. The patient developed signs and symptoms of GBS on day 24 postinjury. Samieirad *et al*.[41] reported a case of a 39-year-old female who presented with symptoms of GBS 3 days after surgical fixation of her mandibular fracture.

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 nondiagnostic 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.[38,42] 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.[37,38,42]

Frey's syndrome

Frey's syndrome, also called gustatory sweating or auriculotemporal syndrome, is a rare disorder that comprises of facial sweating and flushing of the parotid region initiated by gustatory stimulus.[43,44] This condition is attributed to direct damage of the auriculotemporal nerve.[45]

Anatomically, 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.[46] The parasympathetic chain to parotid consists of the preganglionic fibers that 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 the

mandibular nerve, ultimately to supply blood vessels, glands, and other tissue.[47,48]

The specific mechanism involved in Frey's syndrome is still controversial. Several theories have been proposed over years, including the theory of invasion and irritation of damaged auriculotemporal nerve by healing tissue and the theory of parasympathetic hypersensitivity and stimulation secondary to destruction of sympathetic fibers as put forward by Lucie Frey and Freedberg, respectively.[45,49]

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.[44,45,47-49] Parasympathetic and sympathetic nerve fibers can undergo cross regeneration because both use acetylcholine as a neurotransmitter [Figure 3].[50]

Clinically, this syndrome is characterized by episodes of warmth, flushing, sweating, and occasionally pain around the preauricular and temporal areas, when eating, smelling, thinking, or even dreaming about food.[43,44,51] Stimulation of parotid gland consequently leads to concurrent stimulation of the sweat glands in the distribution area of the auriculotemporal nerve due to misdirectional regeneration of parasympathetic fibers.

The most common causes of gustatory sweating reported in literature are parotid surgery, traumatic lacerations to the parotid area, cervical sympathectomy, and cervical and radical neck dissection.[52,53] Though rare, Frey's syndrome secondary to the maxillofacial trauma has been reported by several authors worldwide, with the condition being apparent from as early as 7 months to several years later postinjury.[45,46,53-55] Regeneration of postganglionic parasympathetic nerve fibers in the skin takes a certain amount of time, suggesting a latent period between intraoperative auriculotemporal nerve injury and the onset of Frey's syndrome.[56] In all the case reports, the commonly associated injury is the fracture of the condyle or subcondylar region. It stands to reason that the condylar or subcondylar fractures can injure the auricotemporal nerve since the nerve leaves the main trunk medial to the neck of the condyle deep to the temporomandibular joint and turns upward through the parotid gland to run superficially anterior to the ear.[53,54]

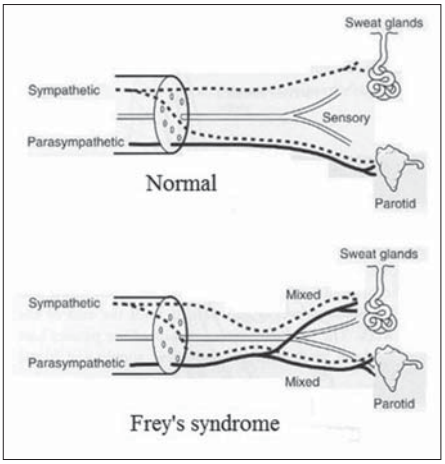

Figure 3: Proposed mechanism of Frey's syndrome (adapted from http://www.parotid. net/wp-content/uploads/2014/03/freys-syndrome.jpg)

The method used to diagnose the syndrome is the Minor iodine-starch test, which involves painting the affected site with a solution containing 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.[50]

Various forms of treatment of Frey syndrome, both medical and surgical, have been tried with varying degrees of success. However, some patients are managed conservatively by an explanation of the condition and reassurance.[56]

Surgical modalities tried include re-elevation of the cheek skin flap and interposition of various tissue barriers such as dermal graft, free grafts, superficial muscular aponeurotic system flaps, and temporoparietal fascia between the cheek skin and the parotid gland.[46,56,57]

Regarding medical treatment, different drugs and substances have been used, most of them applied topically, such as anticholinergic treatment, glycopyrrolate, and scopolamine.[46] Currently, the first-choice symptomatic treatment of Frey's syndrome is intracutaneous injection of botulinum toxin (a powerful neurotoxin, which acts by blocking acetylcholine release at the neuromuscular junction), and studies on the use of intradermic injections of botulinum toxin have proved its efficacy.[50,56]

CONCLUSION

Despite the fact that the syndromes associated with maxillofacial injuries are rare entities, they may lead to poor quality of life or even death. Regardless of the new technological developments in the field of medicine that have helped ease the situation, the diagnosis of these syndromes is eminently clinical. It is the presence of mind of emergency

personnel and surgeons' timely intervention and sheer skill that eventually count. The management of these syndromes at large is a multipronged approach requiring a partnership between several departments and specialties. By summarizing the syndromes associated with maxillofacial injuries, this article will be helpful to the clinicians and surgeons to have a high-suspicious eye when they encounter a patient with maxillofacial injuries and thus think about management that goes beyond the treatment of obvious injuries.

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Conflicts of interest

There are no conflicts of interest.

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