Frey's Syndrome Consequent to an Unusual Pattern of Temporomandibular Joint Dislocation: Case Report with Review of Its Incidence and Etiology

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Abstract

Frey's syndrome was first described in the 18th century. Recognizing it as a nonspecific condition, the symptom of gustatory sweating in patients with parotid gland inflammation was described by Duphenix and Baillarger. However, as a specific diagnostic entity, gustatory sweating, following trauma to parotid glands, was first described by Polish neurologist Lucie Frey, in 1923, and hence he proposed the term *auriculotemporal syndrome*. The condition is characterized by sweating, flushing, a sense of warmth, and occasional pain in the preauricular and temporal areas, following the production of a strong salivary stimulus. Several etiologies of Frey's syndrome have been mentioned in the literature; however, none attribute dislocation of the "intact" mandibular condyle as a cause of the syndrome. Reviewing its pathophysiology, etiology, and incidence in detail, we describe a case of Frey's syndrome subsequent to superolateral dislocation of the intact mandibular condyle following fracture of the anterior mandible. Its management and prevention are also discussed in brief.

Keywords

- Frey's syndrome
- superolateral dislocation
- mandibular condyle
- Minor's test

Frey's syndrome, also called *gustatory sweating* or *auriculotemporal syndrome*, comprises facial sweating, flushing, and sometimes pain in the preauricular and temporal areas following production of a strong salivary stimulus. The first description of unilateral focal gustatory hyperhidrosis consequential to a parotid abscess was provided as early as 1757 by Duphenix,¹ in 1853 by Baillarger,² and again in 1897 by Weber.³ However, it was Polish neurologist Lucie Frey who, in 1923, gave a detailed illustration of gustatory sweating in two cases following parotid trauma, and hence proposed the term *auriculotemporal syndrome*, recognizing it as a specific diagnostic entity.⁴

Medical literature is replete with explanations pertaining to different etiologies and mechanisms of Frey's syndrome. But, to our knowledge, no mention of dislocation of the

"intact" mandibular condyle as its cause is made. Therefore, a case of superolateral dislocation of the mandibular condyle (associated with a symphysis fracture), as the proposed etiology, is described.

Case Report

A 25-year-old man reported to the maxillofacial surgery outpatient department (OPD) 3 days after a traffic accident. After the accident, he experienced difficulty and pain on opening his mouth. He was apparently traveling by an auto rickshaw when it suddenly skidded and overturned, causing him to hit his lower jaw on a metallic sidebar. The patient reported a history of bleeding from both his ears and a brief episode of loss of consciousness with four episodes of vomiting

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immediately following the accident. Extraoral examination revealed a diffuse, bony, hard, left preauricular swelling and a sutured laceration over the chin (►Fig. 3). Mild left-sided facial nerve paresis was also apparent. Intraoral examination revealed a 1-cm mouth opening with a 12-mm symphyseal splay between the mandibular central incisors. Tenderness and a step deformity could be elicited in the midsymphysis region, with an obvious crossbite on the left side.

Routine investigations (hemogram and chest X-ray) were within normal limits. A 3-D computed tomography (CT) scan showed superolateral dislocation of the left condyle overriding the lateral surface of the left zygomatic arch, associated with a 12-mm splay in the midsymphyseal regions (►Fig. 4). A neurosurgical consultation was sought to rule out any head injury. Ear, nose, and throat examination revealed small healing lacerations in the anterior walls of both of the external auditory meati that required no active intervention.

In view of the difficulty in intubation, manual reduction of the dislocation was done under deep sedation and a mouth opening of 35 mm was achieved, following which definitive management of the fracture was done under general anesthesia. Postoperative recovery was uneventful (►Fig. 5). On discharge, the mouth opening was 32 mm; occlusion was satisfactory with a slight deviation of the jaw to the right on mouth opening. No decrease in mouth opening was observed on monthly follow-up; mouth opening remained the same at the last follow-up of 8 months. Complete resolution of the left-sided facial nerve paresis was also noted.

The patient failed to return for follow-up for 7 months, presumably due to the complete resolution of symptoms. However, at 9 months (17 months after initial treatment) the patient reported with restriction in right lateral excursive movements, due to intense pain and clicking in the left temporomandibular joint (TMJ). This was the predominant symptom. Mouth opening was normal (i.e., 35 mm) and occlusion was satisfactory. Hot-water fomentation to the left TMJ together with a soft diet and an analgesic-anxiolytic regimen were advised. The patient had temporary relief but reported again, 3 months later, complaining not only of persistent pain and clicking in the left TMJ but also of profuse sweating in the left preauricular region during meals, which was quite bothersome. The clinical findings appeared consistent with the diagnosis of Frey's syndrome. To confirm the diagnosis, Minor's test was done and a moderately positive result was obtained (►Fig. 6A, B).

Discussion

Modernization is the cornerstone of human behavior. With the advent of scientific technology and research, our understanding of the world around us has been constantly improving. Frey's syndrome is characterized by sweating, flushing, a sense of warmth, and occasionally pain in the preauricular and temporal areas following production of a strong salivary stimulus. Through the years, several theories have been proposed to explain its existence. Frey suggested that the damaged auriculotemporal nerve (ATN) is invaded and irritated by healing tissue.⁵ Freedberg⁶ believed that damage to

the nerve may cause destruction of sympathetic fibers, leading to parasympathetic hypersensitivity and stimulation. But the theory of aberrant regeneration by Ford and Woodhall,⁷ 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 (1) damage to the ATN and subsequent reinnervation of sweat glands by parasympathetic (salivary) fibers and (2) some form of transaxonal excitation from adjacent fibers or ganglion (►Figs. 1 and 2). 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.⁸ Haxtun has summarized his study results, bringing forth four possible theories for gustatory sweating.^{5,9}

1. Secretomotor fibers to the parotid that run in the ATN regenerate along the wrong neurilemma sheath following its interruption and come to innervate the sympathetic endings in the skin.
2. Scarred gland tissue causes excessive tension during parotid activity, consequently leading to irritation of sympathetic fibers traversing the gland in the auriculotemporal nerve.
3. 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.
4. The auriculotemporal syndrome normally exists as a spontaneous effect, deprived of inhibitory fibers by damage to the nerve supply of the area.

Anatomically, the ATN runs partly in the groove formed by the posterior edge of the ascending mandibular ramus and the cartilage of the external auditory canal. From here, it is distributed in fine branches over the preauricular and temporal areas, in part via the terminal branches of the seventh nerve. At this level in its course, surgical disruption most commonly occurs and surgeons may identify it as a small nerve, seldom in excess of 1 mm in diameter (►Fig. 7). At this level the nerve contains:

1. Sensory fibers conveying impulses from the skin,
2. Vasodilator fibers to the subcutaneous arterioles,
3. Secretory fibers to the sweat glands from the cervical sympathetics,
4. Salivary secretory fibers to the parotid, and
5. Vasodilator fibers to the parotid.

Using the PubMed search criteria "Frey's syndrome," the etiologic aspect of this condition was thoroughly reviewed. The 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.

The incidence of Frey's syndrome following parotid surgery, most notably parotidectomy, varies greatly in the

Figure 1 Schematic diagram depicting the normal innervation to the parotid gland and sweat glands of the parotid region. (1) Sweat gland; (2) parotid gland; (3) sympathetic supply to sweat gland; (4) parasympathetic supply to the parotid; (5) ATN. Abbreviation: ATN, auriculotemporal nerve.

literature and thus remains controversial (► **Fig. 2**). Laage-Hellman reported the syndrome's appearance regularly after conservative parotidectomy.^{10,11} He observed that although 60% of his patients affected were aware of symptoms, only 13% complained about it, and thus objective tests would indicate the actual incidence of the condition in them. Morfit and Kramish reported a 54% incidence in 100 cases that underwent superficial parotidectomy with seventh nerve preservation.^{5,12} Glaister et al observed 16 cases of gustatory sweating following 42 parotidectomies.⁸ However, in 2008, Rustmeyer, in an extensive review, stated a wide incidence ranging of 2.6% and 97.6%.^{10,13,14}

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.^{15,16} In 1982, Tuinzing et al described the syndrome as a complication following a sagittal ramus

Figure 2 Schematic diagram indicating the disrupted parotid gland innervation (following parotidectomy) leading to Frey's syndrome. (1) Sweat gland; (2) parotid gland; (3) aberrant re-innervation of parasympathetic nerve fibers to a regional sweat gland; (4) injured ATN. Abbreviation: ATN, auriculotemporal nerve.

Figure 3 Preoperative photograph showing a diffuse swelling over the left temporomandibular joint.

osteotomy.¹⁷ Eleven years later, Zöller et al reported Frey's syndrome secondary to a subcondylar fracture.¹⁸ Again, in 1996, Mellor and Shaw encountered the syndrome following a condylar fracture.¹⁹ The following year, a similar case was reported by Sengezer et al after closed treatment of bilateral condylar fractures.²⁰ Another such case was reported by Ellis in 2004.²¹ Interestingly, in 2010 the Armed Forces Medical College in the city of Pune, Maharashtra, India, described a Frey syndrome-like condition with an incidence of only 0.5%.²² More recently, however, Tue et al made an extensive

Figure 4 Preoperative computed tomography scan showing superolateral dislocation of the left condyle with an associated midsymphysal fracture.

Figure 5 Postoperative orthopantomogram (OPT) showing the reduced and fixed midsymphysis fracture with both the condyles seated in the glenoid fossa.

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.²³ 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).

Figure 6 (A) Gustatory sweating observed over the left preauricular region. (B) A positive Minor's test over the left preauricular region.

Figure 7 (A) Relations of the medial aspect of the mandible: (1) medial pterygoid muscle; (2) maxillary artery; (3) lingual nerve; (4) inferior alveolar nerve; (5) middle meningeal artery; (6) motor root of mandibular nerve; (7) sensory root of mandibular nerve; (8) auriculotemporal nerve; (9) lateral pterygoid muscle; (10) temporomandibular joint capsule; (11) external carotid artery; (12) inferior alveolar artery. (B) Frontal view depicting the vertical distance from superior border of the condyle to the auriculotemporal nerve (0 to 13 mm) and horizontal distance averaging 0 to 1 mm.

Table 1 Classification according to Laskawi et al²⁸

Type	Pathological state	Pathological basis
Type 1 Sympathetic/ parasympathetic	Salivary gland surgery, salivary gland disease, neck dissection	Lesion of thermoregulated fibers + a lesion of gustatory-controlled parasympathetic fibers
Type 2 Sympathetic	Sympathetic neck surgery with sympathetic (trunk) lesions, cervical and cephalic trauma	Lesion of thermoregulated sympathetic fibers + a lesion of preganglionic (cholinergic) gustatory-controlled sympathetic fibers
Type 3 Central	Healthy individuals, feeble, central nervous diseases (e.g., encephalitis, spinal process such as syringomyelia, emotional stress)	Central nervous lesion of thermoregulatory or gustatory regulation centers, low reflex threshold of gustatory sweating

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.²⁴ ►**Table 1** broadly classifies Frey's syndrome into three types on a pathologic state-pathologic basis correlation.

Nature virtually never seems stagnant and thus our case description here seems pertinent. Our explanation is based on the following assumptions. On a 3-D CT scan, concomitant with a mandibular symphyseal fracture, the medial pole of the left condyle is seen to hook the superior border of the ipsilateral zygomatic arch (type IIIB).²⁵ From the etiologic perspective, the following possibilities may be deduced.

1. A high-velocity injury impacts the mandible twice: one impact at the mandibular symphysis causes it to fracture and the other dislocates the "intact" condyle in a backward, upward, and outward direction in an already fractured lower jaw. Recollecting the anatomy of the medial aspect of the TMJ, the ATN could have sustained injury in its course between the TMJ and the external auditory meatus (►**Fig. 7A**); here, at the posterior border of the lateral pterygoid muscle, the nerve trunk is in direct contact with the condylar neck. The horizontal distance is 0 mm at this level (►**Fig. 7B**),²⁶ prior to its termination in the temporal region.
2. The phenomenon of the condyle hooking the superior border of the ipsilateral zygomatic arch following traumatic (superolateral) dislocation may have caused possible impingement injury to the terminal branches of the ATN (►**Fig. 8**).
3. As the ATN gives off branches to the retrodiscal tissue of the TMJ following dislocation, the undue stretching of the joint capsule and the ATN may have resulted in nerve damage.
4. During deep sedation-assisted reduction, the sudden snapback of the condyle into the glenoid fossa could have possibly resulted in ATN injury (assuming the nerve's displacement in the glenoid fossa at the time of dislocation) (►**Fig. 9**).

With this in mind, such dislocations need to be addressed with extreme caution (i.e., the condyle should not be allowed to snap back into the glenoid fossa, but rather should be "guided" into the fossa with concerted effort—in a direction opposite to that of muscle pull) to avoid possible ATN injury.

The clinical manifestations of the syndrome may develop from as early as 5 weeks to as late as 2 years following surgical intervention. However, the average time is ~9 months, thus making it very difficult to determine its real etiologic factor. In our case, the symptoms appeared almost 9 months following surgical intervention and were seen to occur only during meals. For diagnostic confirmation, Minor's test is employed. The test results are categorized as mildly, moderately, and strongly positive.²⁷ Other tests include the blotting paper

Figure 8 Schematic diagram depicting the possible etiopathogenetic mechanism (of superolateral temporomandibular joint dislocation) leading to Frey's syndrome. (1) Parotid gland; (2) injury to ATN; (3) ATN; (4) superolaterally dislocated mandibular condyle. Abbreviations: ATN, auriculotemporal nerve.

Figure 9 Schematic diagram depicting the possible etiopathogenetic mechanism of superolateral dislocation of the mandibular condyle entailing Frey's syndrome. Also seen is the facial nerve coursing through the substance of the ipsilateral parotid gland: (1) facial nerve; (2) parotid gland; (3) injured auriculotemporal nerve; (4) sweat gland; (5) misdirected parasympathetic fibers to a regional sweat gland; (6) superolaterally dislocated mandibular condyle entailing Frey's syndrome.

technique, the iodine-sublimated paper histogram, and the monitoring of L-lactate rate production on skin using a biosensing method.²⁴

Several treatment strategies aimed at effectively treating Frey's syndrome have been proposed, including a local application containing 3% scopolamine hydrobromide,⁵ surgical transection of the parasympathetic nerve, anticholinergic drugs such as glycopyrrolate and diphemuniol,²⁷ division of the Jacobson's nerve,²⁹ radiotherapy,⁵ and more recently chemodenervation with botulinum toxin type A (Botox; Allergan, Inc., Irvine, CA). In the United States, Botox is manufactured by Allergan, Inc. for both therapeutic and cosmetic use (100-unit). In 2011, Allergan required less than one gram of raw botulinum toxin neurotoxin to supply the world's requirements for 25 indications approved by government agencies around the world.

Botulinum neurotoxin (BoNT), isolated from anaerobic spore-forming *Clostridium botulinum*, is chemically a two-chain metalloproteinase composed of a heavy chain and a light chain with eight immunologically distinct serotypes (A to G). Currently, serotype A is used most widely for a variety of movement and spasticity disorders as well as in cosmetic procedures. BoNT irreversibly blocks the presynaptic release of acetylcholine at the neuromuscular junction, leading to chemodenervation. However, the chemodenervation is temporary, as the neuron regenerates functional synapses at the nerve terminals. In addition to anticholinergic activity at the motor end plate, BoNT also acts on the synapses of the

autonomic nervous system, providing anticholinergic effects at the neurosecretory end plates of both the parasympathetic and sympathetic systems. With regard to dose and frequency of administration, one ampoule of Botox contains 100 U of botulinum toxin type A as a freeze-dried powder. Immediately before injection, the toxin is reconstituted with 4 mL of sterile saline solution to a final concentration of 2.5 U/0.1 mL. The borders of the involved skin are marked out according to the starch-iodine test, and then the whole area is divided into squares of $\sim 1 \text{ cm}^2$ each. Recently, several studies have described the effectiveness of botulinum toxin type A intracutaneous injection for gustatory sweating. Consensus exists on the 1-cm interinjection distance and an injection dose of 0.1 mL at a concentration of 25 U/mL. The doses that are reached for each individual patient depend on the size of the sweating area to be treated. The immediate effects may well be rewarding; however, with regard to the period of effectiveness (following the injection of Botox in Frey's syndrome), studies have reported efficacy ranging between 6 months to 2 years on average.^{30,31}

Insofar as reducing the incidence of Frey's syndrome thereby obtaining better aesthetic results, various techniques have been developed using different materials. The following have been suggested and tried with varying degrees of success.

1. Partial superficial parotidectomy.³² The use of partial superficial parotidectomy instead of superficial or total parotidectomy produces improved cosmetic results and salivary function.
2. Free abdominal dermal fat grafts.³³ These grafts provide good facial contouring that reduces the incidence of postoperative gustatory sweating without significantly increasing operating time or lengthening in-hospital stay.
3. Superficial musculoaponeurotic system (SMAS) interpositional flaps.^{34,35} After partial superficial parotidectomy, the remainder of the parotid gland and the SMAS may be sutured back to the sternocleidomastoid muscle. This SMAS interposition seems to reduce the incidence of Frey's syndrome. At the same time, the connection between the ATN and the facial nerve is preserved.
4. Fascia lata interpositional flap.³⁶
5. Sternocleidomastoid flaps.³⁷
6. Permacol (Permacol™; Dublin, Ireland) (porcine dermal collagen).³⁸ It is a strong biological, nonabsorbable implant with natural properties.
7. Allogenic acellular dermal matrix flap.³⁹

Conclusion

The incidence of Frey's syndrome is influenced by the period of postoperative follow-up and the methods employed in its diagnostic confirmation. Insofar as the etiology is concerned, most studies have reported high variations in the incidence of gustatory sweating with regard to surgeries on the parotid and fractured condyle. To our knowledge, superolateral dislocation of the "intact" mandibular condyle has never been implicated as an etiologic factor in Frey's syndrome, thus

making this case unique. The diagnosis of the condition is not difficult as there are many objective clinical tests to indicate its presence. Treatment is controversial because many strategies have been proposed to manage the syndrome, but in recent years botulinum toxin type A has proved far more successful than any other technique; however, owing to the immense subjective discomfort and social embarrassment experienced with this condition, its prevention is more important.

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