GEORGIAN BIOMEDICAL NEWS

VOLUME 3 ISSUE 3. JUL-SEP 2025

Gustatory Sweating Following Parotid Trauma: A Case of Frey's Syndrome in a 34-Year-Old Male

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DOI: 10.52340/GBMN.2025.01.01.127

ABSTRACT

Frey's syndrome (auriculotemporal nerve syndrome), a rare yet well-documented complication following parotid gland surgery or trauma, results in unilateral sweating and flushing on the same side of the face during eating. Although harmless, it can substantially affect quality of life, and diagnosis may be challenging for clinicians unfamiliar with Frey's syndrome. The primary mechanism of Frey's syndrome involves abnormal regeneration: parasympathetic secretomotor fibers intended for the parotid gland reinnervate facial sweat glands and skin vessels, resulting in sweating and flushing rather than salivation. This case report outlines a typical presentation of auriculotemporal nerve syndrome in a young adult male after parotid trauma and surgery, which was confirmed by the starch-iodine test and successfully managed with intradermal botulinum toxin A injections. It covers key aspects of diagnosis, treatment, and follow-up care. Current management approaches are discussed, drawing on recent research and consensus recommendations.

Keywords: Auriculotemporal nerve syndrome; gustatory sweating; Minor's starch-iodine test; onabotulinumtoxin A; parotid gland injury/surgery.

INTRODUCTION

Frey's syndrome (auriculotemporal nerve syndrome) is a type of gustatory hyperhidrosis characterized by sweating, warmth, flushing, and redness in the skin area supplied by the auriculotemporal nerve. These symptoms are triggered by eating or even thinking about food. It most often occurs after parotidectomy but can also happen following other injuries to the parotid region.[1-4]

The earliest descriptions of gustatory sweating linked it to parotid disease and treatment in the 19th and early 20th centuries. In 1853, Charles-Philippe Robin and Jules Félix Baillarger first noted facial sweating associated with parotid disease and drainage procedures, but without precise anatomic correlation. In 1923, Lucja Frey published the classic clinico-anatomic description, correlating gustatory symptoms with injury to the auriculotemporal nerve and coining "auriculotemporal syndrome." V. Minor later developed the starch-iodine test (1928), a standard objective method used to confirm and map gustatory sweating.[1-4]

The incidence of symptoms after parotidectomy or parotid gland injury varies depending on the method of assessment. Objective tests, such as the Minor starch-iodine test, often reveal high rates of occurrence, typically ranging from 30% to 80%. However, the proportion of patients who report symptoms themselves tends to be lower, typically ranging from 10% to 30%. This discrepancy suggests that many cases are mild or unrecognized by patients, and that detection rates depend significantly on the testing methods used. Across different studies and diagnostic techniques, the reported incidence ranges widely from approximately 4% to as high as 96%.[2,3,5-7]

The time to onset of symptoms following surgery usually occurs within six to twelve months. Nevertheless, there are cases where symptoms may appear years after the procedure. Although pediatric and congenital forms of this condition are pretty rare, they have been documented in the literature.[2,3]

The anatomic distribution of the symptoms predominantly involves the skin regions adjacent to the parotid gland. This includes the preauricular area (in front of the ear), the temporal region (side of the head), and the malar skin (over the cheekbone), corresponding to the territory innervated by the auriculotemporal nerve. Less commonly, similar symptoms may be seen in the submandibular area, particularly following surgeries involving the submandibular gland.[2,3,6]

The underlying mechanism relates to auriculotemporal nerve injury. Specifically, damage occurs to the postganglionic parasympathetic fibers that originate from the otic ganglion and travel via the auriculotemporal nerve, as well as nearby sympathetic fibers or their target tissues. During the nerve regeneration process, the cholinergic parasympathetic fibers aberrantly reinnervate structures such as the eccrine sweat glands, which possess M3 muscarinic receptors, and vasodilator targets in the skin over the affected area. As a result, stimuli that typically provoke salivary secretion, such as gustatory (taste) stimuli, can also trigger sweating and flushing in the skin. Alternative hypotheses, like ephaptic transmission (abnormal electrical cross-talk between nerve fibers), have been proposed; however, the concept of abnormal

reinnervation best accounts for the clinical presentation and the observed response to therapies. Treatments such as anticholinergic medications and botulinum toxin injections have proven effective, supporting this mechanism.[2,3,8]

The clinical presentation of the condition includes core symptoms, such as gustatory sweating, characterized by localized, unilateral perspiration precipitated by eating, thinking about food, or gustatory stimulation. Additionally, flushing and warmth occur as transient erythema and heat in the same distribution. Common triggers for these symptoms include sour and spicy foods, with citrus, vinegar, and peppers often eliciting robust responses. The affected area typically has sharp borders, presenting as a patch or field over the preauricular–malar area corresponding to the parotid bed. The quality-of-life impact can be significant, as the visible sweating and flushing may cause psychosocial burden; the severity ranges from incidental to socially disabling.[2,3,6] Differential diagnosis to consider includes primary focal hyperhidrosis, which is non-gustatory and has a different distribution; Harlequin syndrome, characterized by sympathetic denervation with contralateral compensatory flushing and sweating; Horner syndrome, identified by the ptosis, miosis, and anhidrosis triad; medication- or systemic-induced sweating, which presents with a diffuse pattern; and gustatory tearing, or crocodile tears, seen when facial nerve injuries involve the lacrimal pathway.[2,3]

Diagnostic modalities for the condition include bedside confirmation through a provocation test, where a citrus or gustatory stimulus is used to reproduce symptoms and confirm gustatory specificity in a focal area. Objective mapping can be performed using the Minor starch-iodine test, which involves applying iodine to the skin, allowing it to dry, dusting starch over the area, and then applying a gustatory stimulus. The involved skin turns blue-black as sweat appears, enabling precise mapping for documentation and treatment planning. This method has high sensitivity, allowing for the quantification of the response area and intensity over time.[4] Adjunctive tools include infrared thermography, which detects localized heat changes corresponding to flushing and is particularly helpful in cases where flushing predominates. Gravimetric or evaporimetry methods may be used to quantify sweat production for research purposes or in refractory cases. Imaging studies are not routinely required unless alternative diagnoses, such as a recurrent tumor, are suspected based on the patient's history or examination findings.[2,3,6,7]

Treatment options for the condition include both non-surgical management and surgical interventions. Non-surgical management begins with counseling and dietary modification, emphasizing expectation setting since many cases are mild, and avoidance of potent triggers can reduce episodes. Topical antiperspirants, such as aluminum chloride hexahydrate (15-20%), work by obstructing eccrine ducts; they are inexpensive and accessible, but can cause irritation, require frequent application, and may have limited efficacy for moderate to severe cases.[2,3] Topical anticholinergics, such as glycopyrronium or scopolamine (compounded into wipes or gels), act by muscarinic blockade at eccrine glands and help treat focal fields. However, overuse or application over large areas may lead to anticholinergic side effects.[2,3] Systemic anticholinergics, such as low-dose oxybutynin or glycopyrrolate, can be considered for multifocal or refractory cases when topical therapy fails and botulinum toxin is unavailable. However, systemic side effects, including dry mouth, constipation, and urinary retention, may limit long-term use.[2,3]

Botulinum toxin injections are considered first-line treatment for moderate to severe symptoms. OnabotulinumtoxinA is most commonly used, with rimabotulinumtoxinB as an alternative. The technique involves mapping the affected area using the Minor starch-iodine test and administering intradermal injections in a 1-1.5 cm grid with small aliquots per site (e.g., 1–2.5 units of onabotulinumtoxin A per injection), avoiding intramuscular placement. The total dose is individualized based on the size of the affected field. Botulinum toxin yields high response rates, providing near-complete control in most patients, with onset occurring within days and peak effect typically reached within two weeks. The duration of benefit usually lasts 6 to 12 months, and repeat injections maintain efficacy with a favorable safety profile. Adverse effects are generally mild and transient, including injection-site discomfort and, in rare cases, transient muscle weakness if the toxin spreads to the surrounding muscles.[2,3,6,9-11]

Surgical treatment is reserved for cases that are refractory to other forms of treatment. Procedures such as auriculotemporal or tympanic neurectomy aim to interrupt aberrant parasympathetic pathways but have variable durability due to nerve regeneration and carry risks such as sensory changes and dry mouth.[2,3] Interpositional barrier procedures used in revisional surgery include options such as superficial musculoaponeurotic system (SMAS) advancement, temporoparietal fascia flap, sternocleidomastoid fascia, dermofat or free fat grafts, and acellular dermal matrix. These create a barrier between the parotid bed and the overlying skin, disrupting aberrant cholinergic signaling. While evidence exists to support their therapeutic use, it is less robust than that for preventive strategies.[2,6,7]

Preventive strategies employed at the time of index parotid surgery include barrier or interposition techniques, such as SMAS advancement, fascia flaps, and dermal or alloplastic barriers, which can reduce the incidence and extent of Frey's syndrome by separating regenerating nerve fibers from sweat glands. Although multiple observational studies and some controlled trials suggest benefit, heterogeneity in study design and outcome definitions limits the certainty of conclusions. Reviews emphasize the need for higher-quality trials.[6,7,12]

In practical terms, for established symptomatic Frey's syndrome, botulinum toxin injections offer the best balance of efficacy, safety, and repeatability, with surgery reserved for

ISSN (Online): 2720-8796 ISSN (Print): 2720-7994

highly selected refractory cases or when concurrent revisional surgery is indicated.

The prognosis of Frey's syndrome indicates that once established, the condition rarely resolves completely without intervention. Its severity may remain stable or slowly diminish over the years. Although medically benign, Frey's syndrome can have significant social and professional impacts, making it important to manage patient expectations and provide effective, minimally invasive therapies. Long-term control is achievable with botulinum toxin, which offers reproducible and durable relief through periodic reinjections. Surgical options may be beneficial in select cases of refractory disease but involve trade-offs and variable durability.[2,3,6,7,12]

CASE

The patient reported undergoing surgery in 2011 after a knife wound to the left side of the neck. During the incident, the parotid gland was injured, and the carotid artery was lacerated, leading to significant blood loss. After the procedure, it was discovered that the trigeminal nerve had also been damaged. According to medical records, this injury may have occurred either at the time of the trauma or during the surgical procedure.

Due to the trigeminal nerve injury, the patient developed facial palsy, causing significant difficulty in moving the facial muscles on the left side. This included an inability to close the eye, raise the eyebrow, or move the jaw. The patient received facial massage therapy and electrical stimulation as part of their rehab. Over time, some motor functions were restored; however, complete recovery of facial symmetry and movement was not achieved.

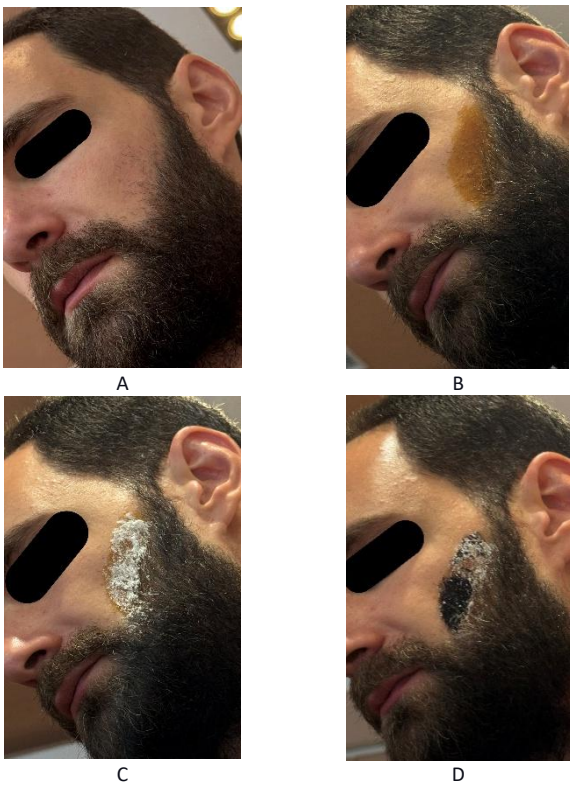
About three to four years after the surgery, the patient began to notice wetness on the left side of their cheek while eating. This symptom has persisted since its initial appearance, with its severity gradually increasing and becoming more noticeable over the past few years. The discharge usually begins after five to seven chews of any food, but it occurs immediately when eating sweets or nuts and persists throughout the chewing process.

To address the condition, the patient visited an ENT specialist a few years ago, during which topical steroid therapy was prescribed. However, this treatment did not provide any benefit.

At the first outpatient visit to the Department of Internal Medicine at TSMU & Ingorokva High Medical Technologies University Clinic in August 2025, physical examination showed normal facial nerve function. There were no signs of local recurrence, masses, or lymphadenopathy. However, shortly after chewing a lemon slice during the visit, sweating and erythema appeared in the preauricular and malar areas, clearly aligned with the previous surgical site.

To confirm the diagnosis, a Minor's starch-iodine test was performed.[3] The procedure showed a characteristic purple-black discoloration in the affected area when exposed to a gustatory stimulus, confirming gustatory sweating and outlining the symptomatic region (Fig.1).

FIGURE 1. Minor's starch-iodine test



Explanations: A. Before the Minor's starch-iodine test, B. After applying iodine, C. After applying starh, D. Characteristic blue-black discoloration in the affected area when exposed to a gustatory stimulus, confirming Frey's syndrome. Images published with informed consent and permission of the patient.

After trying 20% aluminum chloride hexahydrate with limited success, he received intradermal botulinum toxin A injections distributed over the marked area (approximately 2–2.5 units per injection in a 1 cm grid). Symptoms were nearly fully resolved within a week. A repeat injection is scheduled for six months after the initial injection.

DISCUSSION

Frey's syndrome or auriculotemporal nerve syndrome is a distressing yet benign condition that arises primarily due to injury and aberrant regeneration of the auriculotemporal nerve following trauma or surgical intervention in the parotid region.[4] The syndrome manifests as unilateral gustatory sweating and flushing aligned with the anatomical distribution of the auriculotemporal nerve, typically overlying the preauricular and malar areas.[4]

The case presented highlights a classic clinical course of Frey's syndrome secondary to penetrating trauma and subsequent parotid surgery. The patient suffered a knife wound involving the left parotid gland and carotid artery, complicated by trigeminal nerve injury, causing initial facial palsy with incomplete recovery. Several years postoperatively,

ISSN (Online): 2720-8796 ISSN (Print): 2720-7994

the patient developed unilateral gustatory hyperhidrosis localized to the surgical site, corroborating the typical delayed onset reported in literature, usually within months to a few years after parotid region insult.[4,5]

This temporal delay highlights the aberrant nerve regeneration process that drives symptom development and aligns with the pathophysiological understanding that regenerating parasympathetic fibers destined for salivary secretion aberrantly innervate eccrine sweat glands via the sympathetic pathway.[4] The patient's symptom onset and pattern, sweating starting after several chews with rapid onset on sweets or nuts, are consistent with gustatory triggers described in Frey's syndrome.

The diagnosis in this case was clinically suspected and objectively confirmed by Minor's starch-iodine test, which demonstrated characteristic blue-black staining following gustatory stimulation, precisely delineating the affected area. This diagnostic approach remains the gold standard for confirming gustatory hyperhidrosis and assists in mapping treatment zones.[1,4]

Initial attempts to manage the symptoms with topical 20% aluminum chloride hexahydrate yielded limited improvement, which is consistent with the known modest efficacy of topical antiperspirants in moderate to severe cases, due to limited penetration and risks of local irritation.[4] Consequently, intradermal botulinum toxin A injections were administered across the Minor test-mapped area using roughly 2-2.5 units per site on a 1 cm grid. Botulinum toxin acts by inhibiting the release of acetylcholine at eccrine sweat glands, effectively disrupting aberrant parasympathetic signaling.[4] The patient experienced near-complete symptom resolution within one week, corroborating its established role as first-line therapy in Frey's syndrome with durable effects lasting 6-12 months.[4,5]

This case exemplifies the typical management algorithm: initial conservative measures with counseling and topical agents, followed by botulinum toxin injections for symptomatic relief. Surgical treatments, such as auriculotemporal or tympanic neurectomy and interpositional barrier procedures, remain secondary options, reserved for refractory cases or those requiring revisional surgery, due to variable efficacy and risks, including sensory deficits.[5]

In sum, the patient's presentation and therapeutic response illustrate the classical natural history and pathophysiology of Frey's syndrome. The successful use of botulinum toxin corroborates existing evidence on its effectiveness and safety, reaffirming its position as the treatment of choice for moderate to severe cases. The case also highlights the importance of clinical recognition, accurate diagnostic confirmation, and tailored therapy in substantially improving the patient's quality of life.[4]

CONCLUSIONS

Recognizing Frey's syndrome (auriculotemporal nerve syndrome) and applying evidence-based, minimally invasive therapies are essential to improving patient outcomes and quality of life. Future research should focus on refining preventive surgical techniques and exploring the long-term effectiveness of emerging therapies.

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ACKNOWLEDGMENT

Artificial intelligence-assisted technology (Grammarly, Grammarly Inc., San Francisco, CA) was employed to support language editing and grammar correction during manuscript preparation. The use of this tool was limited to linguistic refinement; all intellectual and scientific content was conceived, verified, and approved by the authors.

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ISSN (Online): 2720-8796 ISSN (Print): 2720-7994