



AURICULOTEMPORAL NERVE AND FREY'S SYNDROME

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ABSTRACT:

Background: The auriculotemporal nerve is one of the main branches of the mandibular nerve. It provides sensory innervation to the auricle, the scalp in the temporal region, the temporomandibular joint and part of the dura mater, the parasympathetic innervation of the parotid salivary gland, as well as the external surface of the tympanic membrane, and the sympathetic innervation of the sweat glands and of the blood vessels passing through the parotid gland. Its interruption during surgical interventions or trauma can result in its most common disease - Frey's syndrome. This article is an overview and is based on 23 foreign scientific works - articles published in refereed and renowned scientific publications and anatomy atlases by world-renowned and proven specialists in their field.

Review Results: The auriculotemporal nerve is a sensory nerve which includes secretory parasympathetic vasomotor sympathetic fibers coming from the otic ganglion. It is a branch of the mandibular nerve (V3). Its five terminal nerve branches are front auricular branches, branches for the external auditory canal, branches for the temporomandibular joint, branches for the parotid gland and superficial temporal branches.

Frey's syndrome occurs when the auriculotemporal nerve is cut or injured proximal to the site of separation of the branches for the parotid gland or are severed themselves. The clinical picture characteristic of Frey's syndrome includes redness, sweating, warmth and itching in the area of innervation of the nerve and less often pain.

Conclusion: Knowledge of the anatomy of the auriculotemporal nerve and Frey's syndrome are essential for understanding its nature and stand on the basis of its prevention.

Keywords: auriculotemporal nerve, auriculotemporal syndrome, facial sweating, Frey's syndrome, mandibular nerve, parotidectomy,

BACKGROUND

The auriculotemporal nerve is one of the main branches of the mandibular nerve and provides sensory innervation to the auricle, the scalp in the temporal region, the temporomandibular joint and part of the dura mater, the parasympathetic innervation of the parotid salivary gland, stimulating its salivation, as well as the external surface of the tympanic membrane, and the sympathetic innervation of the sweat glands in the skin area, whose sensory innervation it ensures, and of the blood vessels passing through the parotid gland, to whose vasodilation it leads [1, 2].

Its interruption during surgical interventions or trauma results in its symptomatology disappearing and possibly ending in Frey's syndrome, a benign condition in which parasympathetic innervation from the severed auriculotemporal nerve through aberrant connection of the nerve to the skin in the area it innervates is it also transfers to it the sweat glands and blood vessels containing it [3-5].

This article is an overview and is based on 23 foreign scientific works - articles published in refereed and renowned scientific publications and anatomy atlases by world-renowned and proven specialists in their field. The information from them, after a detailed review, was analyzed and systematized in order to obtain an easy-to-digest and reliable reading that will benefit the clinician in understanding the anatomical features of the auriculotemporal nerve and its most common disease, called Frey's syndrome.

REVIEW RESULTS

The auriculotemporal nerve is a sensory nerve which includes secretory parasympathetic vasomotor sympathetic fibers coming from the otic ganglion. It is a branch of the mandibular nerve (V3) [1, 2]. It separates from the posterior surface of the latter immediately after the separation of the meningeal branch of the mandibular nerve - a small sensory branch that carries out the sensory innervation of the dura mater located adjacent to the oval foramen on the skull base. After exiting the cranial cavity of the mandibular nerve through the oval foramen, the latter is located in the infratemporal fossa, where it gives its branches, in-

cluding the auriculotemporal nerve. After its separation from the mandibular nerve, the auriculotemporal nerve is divided into two roots – anterior, which some authors describe as upper, containing the sensory fibers of the nerve, and posterior, described by some as lower, made up of the sympathetic and parasympathetic autonomic nerve fibers [2]. They run up and back between the lateral pterygoid muscle above and the tensor veli palatine muscle below, where the middle meningeal artery passes between them. Continuing their course backwards and upwards, they pass laterally from the articular process of the lower jaw and then between the capsule of the temporomandibular joint from the outside and sphenomandibular ligament internally, where they give off branches for the sensory innervation of the ipsilateral joint. They are then united again into a common stem of the auriculotemporal nerve [3]. This common trunk, going up and laterally behind the temporomandibular joint, goes to the parotid gland, which crosses together with the superficial temporal artery. The course of the latter in the glandular parenchyma is upward and laterally [3, 4]. Emerging from the gland laterally from the zygomatic process of the temporal bone, it splits into its five terminal nerve branches. They are front auricular branches, branches for the external auditory canal, branches for the temporomandibular joint, branches for the parotid gland and superficial temporal branches.

The front auricular branches are most often two branches that carry out the sensory innervation of the auricle in the area of the tragus and helix.

The branches for the external auditory canal are two - upper (superior external meatus acustici nerve) and lower (inferior external meatus acustici nerve). They are located between the bony and cartilaginous part of the external auditory canal, which they innervate sensory. The upper branch separates the membrana tympani nerve from the outer side of the tympanic membrane, whose sensory and autonomic innervation takes place.

The branches for the temporomandibular joint are two branches that enter the temporo-mandibular joint through the back of the capsule and carry out its sensory innervation.

The branches of the parotid are the branches through which both sensory and autonomic innervation of the parotid gland takes place. At the very beginning, in the infratemporal fossa, to the auriculotemporal nerve are joined by sympathetic fibers from the sympathetic plexuses of the external carotid artery, maxillary artery and medial meningeal artery, and parasympathetic fibers from the otic ganglion via a short branch called *ramus communicans cum nervo auriculotemporalis*. The sympathetic fibers, after their separation from the sympathetic autonomic plexus around the external carotid artery, the maxillary artery and the media meningeal artery, reach and pass through the otic ganglion without breaking it in and thus pass into the composition of the auriculotemporal nerve. They carry out the vasomotor sympathetic innervation of the blood vessels passing through the parotid parenchyma and the sympathetic innervation of the sweat glands in the area innervated by the auriculotemporal nerve. Parasympathetic

fibers passing in the auriculotemporal nerve via *ramus communicans cum nervo auriculotemporalis* are postganglionic (postnodal) parasympathetic fibers, the second neuron of which forms the otic ganglion itself. Preganglionic (prenodal) parasympathetic fibers (the so-called first neuron) start from the inferior salivatory nucleus of the medulla oblongata and reach the otic ganglion through the tympanic nerve and the minor petrosal nerve – branches of the IX cranial nerve (glossopharyngeal nerve). They carry out the parasympathetic innervation of the glandular parenchyma of the parotid salivary gland. The otic ganglion itself is a small star-shaped formation located immediately below the oval foramen on the inner side of the mandibular nerve [2, 3].

The superficial temporal branches accompanying the terminal branches of the superficial temporal artery reach the skin in the temporal region and carry out its sensory innervation. There, they communicate with the final sensory branches of the zygomaticotemporal nerve of the maxillary nerve (V2) and the facial nerve (VII).

Some authors describe another branch of the auriculotemporal nerve - *ramus communicans cum nervo facialis*, through which the connection between the two is made.

It can be summarized that the auriculotemporal nerve provides the sensory innervation of the external ear, the scalp in the temple area, the temporomandibular joint and part of the dura mater, the parasympathetic innervation of the tympanic membrane and the parotid salivary gland, stimulating its salivation, and the sympathetic innervation of the blood vessels passing through the parotid gland, and of the sweat glands in the area of the skin innervated by it [1-5].

It is important to note that often inflammatory diseases of the parotid gland are accompanied by a shooting pain in the ear and temple, the basis of which is the complex path that the auriculotemporal nerve passes – through the parotid gland tissue to the external ear and the temple area.

There are three main diseases of the auriculotemporal nerve - auriculotemporal neuralgia, perineural tumor along its course and auriculotemporal syndrome. The subject of this article is the latter, also known as Frey's syndrome, Baillarger's syndrome, Dupuy's syndrome and gustatory hyperhidrosis - a disease based on the incorrect transmission of nerve impulses along the course of the auriculotemporal nerve [1].

Literary evidence of the existence of auriculotemporal syndrome as a disease in humans has been available since 1853 when Jules Baillarger first described the manifestations of the condition in two of his patients, in whom he had drained abscesses of the parotid gland. He described sweating through the auricle in patients on the side of their operation, which he associated, however, with the passage of saliva from the gland through the skin on the surface of the body.

It was only in 1923 that the Polish doctor Lucia Frey's, who was one of the first women involved in academics of her time, systematized knowledge about the condition that today bears her name – Frey's syndrome. She

describes a case of a 25-year-old female patient with a gunshot wound in the parotidomasseteric region, in which, about a month after the injury, redness and sweating appeared in the area innervated by the auriculotemporal nerve during feeding.

In 1927, André Thomas described aberrant regeneration of the auriculotemporal nerve as the cause of Frey's syndrome, and in 1932, Peter Basso described the first manifestation of the disease after parotidectomy.

Frey's syndrome occurs when the auriculotemporal nerve is cut or injured proximal to the site of separation of the branches for the parotid gland or are severed themselves [2, 6]. As the most common reason for this, operative interventions on the parotid glands and especially their removal, called parotidectomy, are indicated [2, 7]. There are data that the larger the tumor of the parotid gland, which led to its surgical removal, the greater the probability of the development of the disease, which is its postoperative complication [8]. Other possible causes of the syndrome are cervical dissection, fracture of the lower jaw in the area of its articular process and trauma in the same area [6, 9].

Cases of Frey's syndrome have been described in newborns with a history of forceps-assisted delivery, in which the auriculotemporal nerve is traumatized and compressed. However, it is not interrupted, and this is the reason for the subsidence of the complaints and their absence, not their persistence. They appear most often 5-6 months after birth and are expressed in redness and sweating in the area innervated by the nerve. They are often confused with a food allergy due to the coincidence of the feeding of children at this age, but in contrast, in the auriculotemporal syndrome, the symptoms subside very quickly after the cessation of feeding, while in allergic reactions, redness, and sweating have a longer-lasting effect [10].

There is no literature data on sex and age pre-exposure factors described as causes of the onset of the disease.

Different studies report a different frequency, moving in a large percentage range, of its occurrence in patients after parotidectomy – 4-90% [11].

Signal stimulation of the postganglionic parasympathetic fibers stimulating saliva production and salivation from the parotid gland is carried out by the neurotransmitter acetylcholine, which acts on muscarinic receptors. Postganglionic sympathetic fibers, the stimulation of which leads to vasodilatation and secretion of sweat from the sweat glands, are excited by the same mediator through the activation of the same type of receptors. Damage to the auriculotemporal nerve above the site of separation of the branches for the parotid gland or to the branches themselves may result in aberrant growth of parasympathetic nerve fibers over sympathetic ones, a condition that results in the transfer of nerve impulses from parasympathetic to sympathetic fibers, leading to activation of the sympathetic nerves, resulting in vasodilatation, which is the cause of the hyperemia in the affected area, and sweating as a result of the stimulation of the sweat glands to produce sweat. Some authors call this aberrant growth of nerve endings budding. The uniformity of the nerve receptors (muscarinic) and the mediator (acetylcho-

line) in its two parts helps to transfer the nerve impulses from one type of nerve to the other of the autonomic nervous system [7]. The budding process takes 6 to 18 months, which explains the time interval between surgery or trauma and its appearance [12].

The clinical picture characteristic of the auriculotemporal syndrome includes redness, sweating, warmth and itching in the area of innervation of the nerve and, less often, pain, which is usually mild and most often localized in the external auditory canal, in the depth of the ear and in the temporomandibular joint as it can radiate to the body of the lower jaw. The symptoms can vary in strength - from almost imperceptible to quite pronounced and bothering patients to the point of avoiding eating in public places. In general, spicy and acidic foods are a more potent stimulus for the manifestations of the condition [10-13]. During a physical examination, it is possible to establish the presence of a cicatrix from a previous surgical intervention or trauma in the ear region [13].

Differential diagnosis is made with primary and secondary hyperhidrosis, food allergy in children, emotional sweating and Bogorad syndrome (crocodile tears syndrome).

The diagnosis is made on the basis of the clinical picture and the anamnestic data of previous trauma or surgery. A method to confirm it is Minor's test, in which the skin surface is smeared with iodine and sprinkled with cornstarch. The two, in the presence of sweat, interact, and the mixture changes color from brown to blue [12].

In order to prevent the aberrant connection of parasympathetic fibers with sweat glands and to avoid creating a prerequisite for the occurrence of Frey's syndrome, it is necessary to create a barrier between them. It is more effective the deeper it is in the subcutaneous tissue because the deeper it is in relation to the hair follicles along which the sweat glands are located, the less chance there is of budding between the parasympathetic nerves and the sweat glands. The role of such barriers can be fulfilled by the following components: skin acellular matrix, autologous fat transplantation, the use of SMAS-flap, flap of temporoparietal fascia and flap of the sternocleidomastoid muscle [8, 12, 14-17].

Treatments for Frey's syndrome include topical antiperspirants and alcohol injections, scopolamine and glycopyrrolate, atropine administration, and topical injection of botulinum toxin A [18-21].

Antiperspirants in patients with auriculotemporal syndrome have an effectiveness of about 50%. In turn, the injection of alcohol solutions adjacent to the otic ganglion in order to prevent the transmission of nerve impulses between the first and second neurons of the parasympathetic innervation of the parotid gland can lead to damage to the adjacent trunk of the mandibular nerve with lost symptoms of the entire nerve. Oral atropine, in order to produce the desired anhidrosis effect, needs to be taken in such high doses that it leads to adverse side effects such as visual disturbance, tachycardia, and sometimes coma. Local application of scopolamine and/or glycopyrrolate significantly alleviates the symptoms of

Frey's syndrome without serious side effects being described. Its frequent application is noted as a disadvantage. The intradermal injection of botulinum toxin A every 1 square cm in the area affected by the process leads to a reduction and, in most cases, to the disappearance of the overall symptoms, and its regular application every 4 months has the effect of reducing the area of the affected area. The basis of the method is the property of botulinum toxin A to block the conduction of nerve impulses by blocking the release of acetylcholine in the neuromuscular synapse [18-21].

Operative treatment of Frey's syndrome involves neurotomy (cutting) of the auriculotemporal and/or glossopharyngeal nerve, placement of a subcutaneous barrier to separate the parasympathetic fibers from the sweat glands, and excision of the affected skin with grafting of new skin to the area of the resulting defect. The neurotomy of the auriculotemporal and glossopharyngeal nerves very often leads to failure due to their ability to regenerate, and the operative technique itself is complex and may be associated with a number of complications and, therefore, is

not recommended. Local skin grafting to cover the defect resulting from excision of the affected skin is often not aesthetically acceptable and is avoided. The use of a flap from the temporoparietal fascia and a flap from the sternocleidomastoid muscle as a barrier between parasympathetic fibers and sweat glands has a lasting effect but leads to a disfigurement of patients and an unacceptable appearance of the latter [22, 23].

Frey's syndrome is a benign disease that is very often neglected by head and neck surgeons, but its social importance for its sufferers cannot be underestimated because it significantly impairs the quality of life and the level of self-esteem in these patients.

CONCLUSION

Knowledge of the anatomy of the auriculotemporal nerve and its most common disease, Frey's syndrome, which is essentially the most common postoperative complication in patients after operations in the parotidomasseteric area, are essential for understanding its nature and stand on the basis of its prevention.

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