Short Communication

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Frey's syndrome: A study of the anatomy, physiology and possible role of neurotrophic factors

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Abstract

Frey's syndrome describes the phenomenon of gustatory sweating and is a cause of significant social embarrassment for sufferers. It has been attributed to aberrant growth of parasympathetic salivatory fibres in the auriculotemporal nerve towards overlying sweat glands. However, the exact mechanism behind this growth is unknown. Frey's syndrome is a common sequela of parotidectomy and is usually preceded by trauma to the auriculotemporal nerve. In some neonates, however, Frey's syndrome presents with no history of nerve injury. In these patients, the underlying trigger for Frey's syndrome is unclear. A review of the recent literature on nerve regeneration after injury and on nervous development in utero was conducted with the aim of developing further insights into the aetiology of both adult onset and paediatric Frey's syndrome. Neurturin, a neurotrophic factor released by both salivary and sweat glands, was identified as a possible key player in the aetiology of Frey's syndrome. This factor is released both in utero and after nerve damage, so could connect the two presentations of gustatory sweating. Further research into the role of neurturin could help to elucidate the pathogenic mechanisms underlying the condition and might reveal neurturin to be a potential target for pharmacological intervention.

Introduction:

Signs & Symptoms:

The symptoms of Frey syndrome typically develop within the first year after surgery in the area near the parotid glands. In some cases, Frey syndrome may not develop until several years after surgery. The characteristic symptom of Frey syndrome is gustatory sweating, which is excessive sweating on the cheek, forehead, and around the ears shortly after eating certain foods, specifically foods that produce a strong salivary response such as sour, spicy or salty foods.

Additional symptoms that may be associated with Frey syndrome include flushing and warmth in the affected areas. This is rarely an important complaint.

While other symptoms have been associated with the syndrome, they are probably unrelated. Pain is sometimes described, but it is probably more related to the surgery than actually to Frey syndrome. The specific area affected, the size of the area, and the degree of sweating and flushing vary greatly among affected individuals. In some patients, symptoms may be mild and affected individuals may not be bothered by the symptoms. In other cases, such as those that experience profuse sweating, affected individuals may require therapy.

Causes:

The exact underlying cause of Frey syndrome is not completely understood. The most widely held theory is that Frey syndrome results from simultaneous damage to sympathetic and parasympathetic nerves in the region of the face or neck near the parotid glands. Parasympathetic nerves are part of the autonomic nervous system, which is the portion of the nerve system that controls or regulates involuntary body functions. One function of parasympathetic nerves is to regulate the activity of glands including the parotid glands, but not the sweat glands. Sweat glands and blood vessels throughout the body are controlled by sympathetic fibers.

In Frey syndrome, researchers believe that the parasympathetic and sympathetic nerves near the parotid glands are cut, especially tiny branches originating from the auriculotemporal nerve. The auriculotemporal nerve supplies nerves (innervates) to certain structures in the face including the parotid glands.

Normally, damaged nerve fiber(s) eventually heal themselves (regenerate). In Frey syndrome, it is believed that damaged nerve fibers regenerate abnormally by growing along the sympathetic fiber pathways, ultimately connecting to the miniscule sweat glands found along the skin. Therefore, the parasympathetic nerves that normally tell the parotid glands to produce saliva in response to tasting food now respond by instructing the sweat glands to produce sweat and the blood vessels to widen (dilate). The cumulative result is excessive sweating and flushing when eating certain foods.

Damage to the nerves in the parotid gland region of the face may occur for several different reasons including as a complication of surgery or blunt trauma to the side of the face. In older reports, infections of the parotid glands were suspected, but a detailed examination always points to a surgical drainage of a parotid abscess. The most common reported cause of Frey syndrome is a surgical procedure called a parotidectomy (the surgical removal of a parotid gland). Although the exact percentage is not agreed upon in the medical literature, some sources suggest that more than half of all individuals who undergo a parotidectomy eventually develop Frey syndrome. A recent meta-analysis concluded that the interposition of tissue after

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parotidectomy might decrease the incidence of Frey syndrome after parotidectomy.

Another rarely described cause (etiology) of Frey syndrome is damage to the main sympathetic nerve chain in the neck.

In extremely rare cases, Frey syndrome has been described in newborns, possibly following trauma due to delivery with forceps. Actual careful examination reveals that the principal symptom is flushing which might be physiologic at a younger age. The key symptom of facial sweating is not emphasized in newborns rising doubts about the correctness of these observations.

Diagnosis:

Diagnosis is made based on clinical signs and symptoms and a starch-iodine test, also known as the Minor test. The affected area of the face is painted with iodine which is allowed to dry, then dry corn starch is applied to the face. The starch turns blue on exposure to iodine in the presence of sweat

- Injection of botulinum toxin
- Surgical transection of the nerve fibers
- Application of an ointment containing an anticholinergic drug such as scopolamine

Cochrane reviews of interventions to either prevent or treat. Frey's syndrome have found little or no evidence to support their effectiveness or safety, and conclude that further clinical trials are needed.

Conclusion:

Soon after its discovery, in the middle of the twentieth century, it became clear that NGF had great pharmacological potentialities, for the treatment of major central neurodegenerative diseases and of peripheral neuropathies. After preclinical characterization and clinical trials have been performed by treating AD, Parkinson's, and diabetic patients severe limitations in the clinical use of NGF emerged, coming from its physiological action on the sensory and autonomic systems and from the high pharmacological doses needed to obtain disease improvements. Despite the discouraging results coming from trials mainly performed across the 1990's, the translational research on NGF was not stopped, widening the spectrum of diseases that could benefit from NGF-based therapy and investigating new delivery strategies, aimed at maximizing positive outcomes and limiting or fully circumventing the deleterious side effects described in earlier clinical trials. Today we know that epithelial derangements based on poor neurotrophism could be safely treated with topical NGF, while a wide spectrum of CNS and PNS diseases will probably benefit from NGF therapy, once intranasal or gene delivery systems will be finally set-up and fully translated into clinical practice. A further challenge, in conclusion, is represented by the increasing knowledge on the role of NGF in immune system regulation, opening a promising field for development of innovative NGF-based therapies in the care of, in example, chronic inflammatory or autoimmune diseases, and a novel and challenging aspect in the NGF saga.