Frey’s syndrome occurs as a result of damage to the auriculotemporal nerve, which causes inappropriate regeneration of damaged parasympathetic fibres to salivary glands to innervate the sympathetic receptors of sweat glands in the face. The symptoms are pathological flushing and sweating with gustatory stimuli. It most commonly occurs following parotid surgery and has not previously been reported following burn injury. We present a 50-year-old man who sustained 1% TBSA full thickness burn to the right side of his face as a child. This was excised and reconstructed with skin grafts as well as further revision procedures in his adult life. He incidentally reported copious amounts of gustatory sweating over his right temple region that had been present since his initial injury, occurring prior to any reconstruction, consistent with Frey’s syndrome. This was confirmed with a starch iodine test, and successfully treated with Botulinum toxin injections post reconstruction. This case is the first report of Frey’s syndrome following burn injury. We highlight the potential development of Frey’s syndrome following facial burns, even in the reconstructed area. Botulinum toxin treatment remains effective.

SUMMARY. Frey’s syndrome occurs as a result of damage to the auriculotemporal nerve, which causes inappropriate regeneration of damaged parasympathetic fibres to salivary glands to innervate the sympathetic receptors of sweat glands in the face. The symptoms are pathological flushing and sweating with gustatory stimuli. It most commonly occurs following parotid surgery and has not previously been reported following burn injury. We present a 50-year-old man who sustained 1% TBSA full thickness burn to the right side of his face as a child. This was excised and reconstructed with skin grafts as well as further revision procedures in his adult life. He incidentally reported copious amounts of gustatory sweating over his right temple region that had been present since his initial injury, occurring prior to any reconstruction, consistent with Frey’s syndrome. This was confirmed with a starch iodine test, and successfully treated with Botulinum toxin injections post reconstruction. This case is the first report of Frey’s syndrome following burn injury. We highlight the potential development of Frey’s syndrome following facial burns, even in the reconstructed area. Botulinum toxin treatment remains effective.

Keywords: facial burn, Frey’s syndrome, botulinum toxin

Case presentation

In this case we present a 50-year-old male who sustained a 1% TBSA full thickness burn to the right forehead, cheek and temporal region as a child. This was excised and reconstructed with split thickness skin grafts at the time of injury. Since presenting to our institution, he has undergone multiple scar revisions to his forehead, lipofilling to his cheek, and a lateral canthoplasty. Incidentally, at a follow-up clinic, he reported copious amounts of gustatory sweating over his right temple region that had been present since his original injury, prior to any

such as lemon juice, is given to the patient to induce sweating. The starch reacts with the iodine in the presence of moisture to produce a blue-black iodide compound.
reconstruction. This was consistent with Frey’s syndrome. This was then confirmed using a starch iodine test. The patient was treated with 100 units of botulinum toxin type A (Botox™, Allergan) in 2.5ml of 0.9% saline, to half of the affected area. On follow-up at 3 months his symptoms had completely resolved in the treated area, and so the remainder of the symptomatic area was also treated.

Discussion

This is the first reported case of Frey’s syndrome following burn injury. Furthermore, the ability to develop hyperhidrosis in a skin grafted area is noteworthy.

Full thickness burns involve the entirety of the skin thickness, including the adnexal structures of the dermis. This results in disruption of the neuro-vascular supply as well as sweat gland ducts that are removed or disrupted. For donor skin, the vascular bed, neural connections and the duct portion of the sweat gland are disrupted by the harvesting procedure. It has been shown that grafted skin is often not subject to heightened vascularity and sweat production during heat stress. Furthermore, there is no evidence that this improves as the graft matures, and thus, sweat gland responsiveness remains the same. Multiple studies have shown a reduction in sweating and vasodilation in skin-grafted areas, thought to be due to a combination of the initial injury and lack of sweat glands in the harvested skin. It is therefore notable that hyperhidrosis has occurred within the skin grafted area in this case.

Spontaneous regeneration of sweat glands has not been known to occur, and the regeneration of sweat glands has been a key topic of stem cell research. Patients with deep burn injury often heal by hypertrophic scarring without regeneration of sweat glands and therefore lose the function of perspiration. Intradermal injections of Botulinum toxin around the eccrine sweat glands inhibits the release of presynaptic acetylcholine and thus leads to temporary chemodenervation with the loss or reduction of sweating. It can therefore be concluded that either the initial injury must not have been full thickness, as initially reported during the patient’s childhood, or there were islands of surviving dermis within the reconstructed area.

Whilst there are no clearly defined recommendations on dosing, Botulinum toxin A has become a proven method for the treatment of gustatory sweating. Intradermal injections of Botulinum toxin around the eccrine sweat glands inhibits the release of presynaptic acetylcholine and thus leads to temporary chemodenervation with the loss or reduction of sweating. Symptomatic relief has been achieved in this case.

Conclusion

In conclusion, we highlight the potential development of Frey’s syndrome following facial burns, even in a reconstructed area. This can be treated successfully using botulinum toxin.