

First bite syndrome: our experience of laser tympanic plexus ablation

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Abstract

Introduction: First bite syndrome is a condition characterised by severe facial pain brought on by the first bite of each meal. This can severely affect the patient's ability to eat.

Methods: We present a 70-year-old woman for whom we performed a laser ablation of the left ear tympanic plexus, as treatment of first bite syndrome. A permeal approach was used to raise a tympanomeatal flap. The tympanic plexus was identified on the promontory and a 4 mm² area of the plexus was ablated using CO₂ laser. The flap was repositioned and a dressing was placed with topical antibiotics.

Results: At two-month follow up, there was full resolution of the patient's symptoms.

Discussion: First bite syndrome carries a high morbidity; treatment options are variable, and often unsuccessful. We describe the first documented case of laser tympanic plexus ablation, with a very effective initial response. This procedure represents a useful therapeutic option for first bite syndrome.

Key words: Tympanic Cavity; Parotid Diseases; Lasers

Introduction

First bite syndrome is a condition characterised by severe spasm or cramping facial pain over the parotid region, brought on by the first bite of each meal and subsequently diminishing in severity.^{1,2}

First bite syndrome is a known complication of surgery involving the infratemporal fossa, parapharyngeal space and parotid gland. The aetiology of first bite syndrome is believed to be a loss of sympathetic innervation of the parotid gland.

Symptoms of first bite syndrome can be severe enough to affect a patient's quality of life and ability to eat, leading to a decline in systemic health.²

There are a variety of treatment options described for first bite syndrome; however, response is varied. Here, we describe our method of surgically treating first bite syndrome, which in our experience has minimal associated morbidity.

Materials and method

A 70-year-old woman underwent a left total parotidectomy in 2009 for a necrotic lump, which was subsequently noted to comprise squamous metaplasia within a previously obstructed salivary gland duct with abscess formation.

Post-operatively, she developed an unbearable pain over the left jaw which occurred with every meal. She described the pain as an 'electric shock' which was very severe with the first bite of each meal and then decreased in severity throughout the meal. The pain was reproducible, and the patient's symptoms persisted and progressed. The pain became significant enough to prevent her from eating

an adequate solid meal, leading to her replacing her solid diet with soft foods and liquids.

The patient was subsequently seen by the neurosurgical team and was advised to undergo review by the pain team. A trial of treatment with amitriptyline, carbamazepine and gabapentin was commenced, but none of these drugs had any significant effect on her symptoms.

In 2010, the patient had an injection of botulinum toxin, which also failed to provide symptomatic relief.

She then presented with a new neck lump, and we undertook a left level II lump excision biopsy, which revealed Warthin's tumour on histological analysis. During the same anaesthesia, we performed laser ablation of the left ear tympanic plexus in order to treat the patient's first bite syndrome symptoms.

The patient was positioned and draped in a supine position, as for any standard otological procedure (including mastoidectomy). A permeal approach was used. The external auditory canal was infiltrated with 2.2 ml of 2 per cent lignocaine with 1:80 000 adrenaline. A tympanomeatal flap was raised using an incision 5 mm lateral from the tympanic membrane annulus, from 3 o'clock to 8 o'clock (with the patient supine), taking care to keep the tympanic membrane intact. The promontory was fully exposed and the tympanic plexus identified (Figure 1). A 4 mm² area was ablated using a CO₂ laser set at 'super pulse continuous' with a 1.5 W power setting. Special care was taken to ensure that the anterior aspect of the plexus (which is often more difficult to demonstrate within the operative field) was also ablated (Figure 2). The tympanomeatal flap was repositioned and

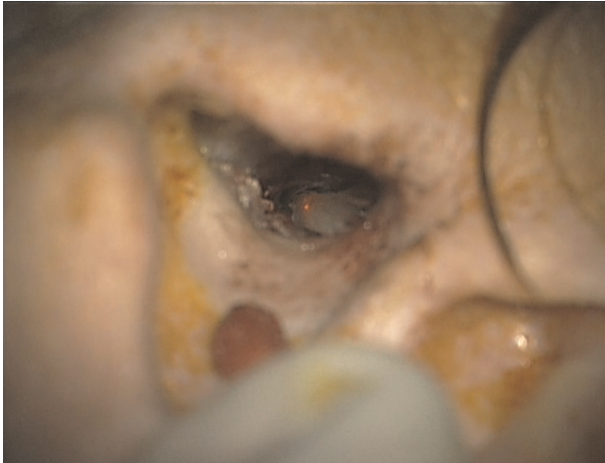


FIG. 1

Operating microscope view showing the tympanic plexus identified and fully exposed.

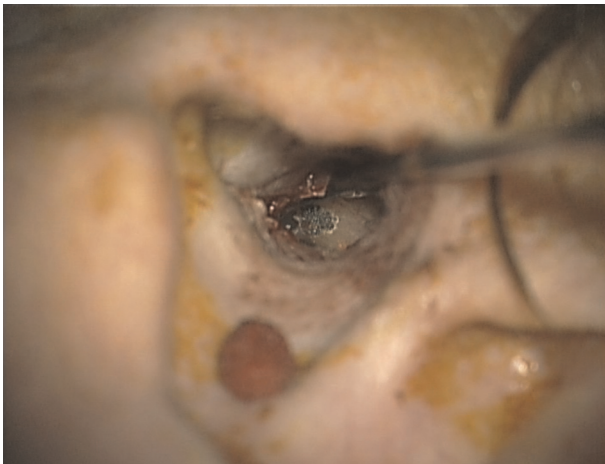


FIG. 2

Operating microscope view showing the laser tympanic plexus ablation; the surgeon ensured that the most anterior branches were also ablated.

Spongostan foam dressing (Ethicon, Livingston, UK) was placed together with topical antibiotics.

At three-week and two-month follow up appointments, the patient reported resolution of her first bite syndrome symptoms. At the time of writing, we intended to review the patient again, eight months post-operatively.

Discussion

The first description of first bite syndrome was published in 1986 by Haubrich, a gastrointestinal surgeon, who associated first bite syndrome with oesophageal dysfunction.³

The currently used description of first bite syndrome was first published by Netterville *et al.* in 1998. The symptoms comprise severe, ipsilateral spasm or cramping pain over the parotid region, brought on by eating the first bite of the meal, and thereafter decreasing with each subsequent bite.⁴

First bite syndrome was thought to be rare, although the exact prevalence was unknown. Linkov *et al.* assessed 499 patients who underwent surgery of the infratemporal fossa, parapharyngeal space or parotid gland. They found a first

bite syndrome prevalence of 9.6 per cent (45 of 499), indicating that first bite syndrome may be an often overlooked and under-reported complication of head and neck surgery.¹

The parotid gland is innervated by the sympathetic and parasympathetic nervous systems.

The sympathetic supply arises in the hypothalamus and travels via the brainstem before synapsing in the thoracic spinal cord. The neurons then enter the sympathetic chain and ascend through the inferior and middle cervical ganglia, synapsing at the superior cervical ganglia. The latter is a 2.5 cm long structure located at the level of the second and third cervical vertebrae, posterior to the carotid sheath. Postganglionically, nerve fibres travel via the external carotid artery plexus and then along the middle meningeal artery, to end in the parotid gland nerve branches.

The parasympathetic innervation to the parotid gland is from the inferior salivary nucleus to the otic ganglion via the glossopharyngeal nerve and tympanic plexus (Jacobson's nerve).

Netterville *et al.* described a case series of patients with vagal paragangliomas who developed the symptoms of first bite syndrome. They proposed that loss of sympathetic innervation to the parotid gland might cause a denervation hypersensitivity of salivary myoepithelial cells. During oral intake, parasympathetic neurotransmitter activation leads to cross-stimulation of the sympathetic receptor, resulting in over-activation of the myoepithelial cells in the residual parotid gland. This causes a spasm with the first oral intake of food after a period of salivary rest.^{4,5}

Chiu *et al.* tested this hypothesis in an assessment of 12 patients with first bite syndrome. Six of their patients exhibited post-operative Horner's syndrome, while the other six had undergone an external carotid artery ligation inferior to the parotid gland, supporting the theory that loss of sympathetic innervation to the parotid gland from the superior cervical ganglion may contribute to the formation of first bite syndrome symptoms.² Similarly, Kawashima *et al.* observed nine patients who developed first bite syndrome after surgery involving the parapharyngeal space, and noted that eight of these nine had undergone surgical ablation of the cervical sympathetic chain and/or external carotid artery.⁶

Due to the severity of the pain associated with first bite syndrome, many patients greatly reduce their oral intake, potentially risking malnutrition. Treatment of first bite syndrome is thus a priority, and there are a variety of potential management options.

Medical treatment options for first bite syndrome include non-steroidal anti-inflammatory drugs, gabapentin, amitriptyline and carbamazepine. Responses to all these medications varies between individual patients.

The use of botulinum toxin has been described, injected into the affected parotid gland. Ali *et al.* have reported a marked improvement in a female first bite syndrome sufferer who had previously experienced no symptom resolution with medical or surgical management.⁷

Surgical treatment options described in the literature include tympanic neurectomy. Chiu *et al.* described three patients undergoing this procedure. Two patients noticed a mild improvement in their symptoms but returned to their baseline level of symptoms after a week. The third patient noticed no improvement at all. The exact technique used for the tympanic neurectomy was not described.² Tympanic neurectomy has been used with good effect in cases of chronic parotitis.⁸

Our patient had a poor response to botulinum toxin injection, and was suffering from low mood and decreased oral intake secondary to first bite syndrome symptoms. We used laser tympanic plexus neurectomy to ablate the parasympathetic innervation to the parotid gland, therefore decreasing cross-stimulation of the sympathetic receptor and reducing over-activation of the myoepithelial cells. At three-week and two-month follow-up appointments, our patient demonstrated symptomatic resolution resulting in greater oral intake.

Conclusion

First bite syndrome is a complication of parotid gland surgery which carries a high morbidity rate. Treatment options have a variable effect and often only work in the short term. We describe the first documented case of treatment of first bite syndrome using laser tympanic plexus ablation, with a very effective initial response.

We believe that this procedure presents a useful therapeutic option for first bite syndrome.

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