Minimally Invasive Trigeminal Ablation: Long Buccal Nerve

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Aims: To describe a technique of minimally invasive trigeminal nerve ablation of the long buccal nerve that was performed at a tertiary care academic medical center. **Methods:** This case describes a 44-year-old woman with refractory left long buccal nerve neuropathy following a dental procedure. After failing medical management, she was taken for nerve exploration, which revealed no nerve discontinuity or neuroma formation. She was therefore counseled regarding the risks and possible benefits of a novel minimally invasive trigeminal nerve thermoablation of the long buccal nerve technique. **Results:** Postoperatively, the patient experienced mild anesthesia along the long buccal nerve division and no longer experiences any allodynia or hypersensitivity. Additionally, she no longer requires any additional medical therapy or interventions. **Conclusion:** Minimally invasive trigeminal nerve ablation of the long buccal nerve may be effective surgical intervention in treating refractory neuropathic pain in cases of no structural nerve defects. However, long-term well-designed studies are required to fully define its role. *J Oral Facial Pain Headache 2019;33:e19–e22. doi: 10.11607/ofph.2341*

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Iatrogenic injury to any of the terminal branches of the trigeminal nerve (lingual nerve, inferior alveolar nerve, or long buccal nerve) can result in debilitating loss of function and a full range of unpleasant and potentially painful neuropathic dysfunction in the oral and maxillofacial region. Complaints can include neurosensory deficits and sensory aberrations (such as hyperalgesia) and a sense of a burning pain distal from the site of injury.[1,2] Unfortunately, patients with these injuries can have reduced quality of life and mental health in up to 62% of cases.[3] Furthermore, up to 13% of patients will have no improvement or experience worsening of their symptoms as time passes.[4]

Although the morbidity of these injuries is well established, treatment options remain limited. Initial medical management includes antidepressants, antiepileptics, topical anesthetics (5% lidocaine patches), and topical neuropathic medications.[5,6] However, the role of most systemic medications is limited due to a multitude of side effects, patient compliance, and limited efficacy.[7] As a result, surgical solutions continue to evolve. Generally, operative intervention begins with exploratory surgery to confirm the integrity of the nerve in question, followed by external neurolysis for intact nerves or direct suturing, autogenous vein graft, and/or a GORE-TEX tube graft as a conduit for nerves with neuromas or discontinuities. The overall success rate of these interventions ranges from 25% to 66.7%.[5]

The heterogeneity and mixed results of surgical interventions testifies to the need for innovative surgical solutions for patients with incapacitating iatrogenic traumatic trigeminal neuropathy. Furthermore, to the present authors' knowledge, all prior descriptions for surgical intervention involve either the lingual or inferior alveolar nerves, with no publications regarding the long buccal nerve. In this case report, a new surgical technique for the treatment of long buccal nerve dysfunction,

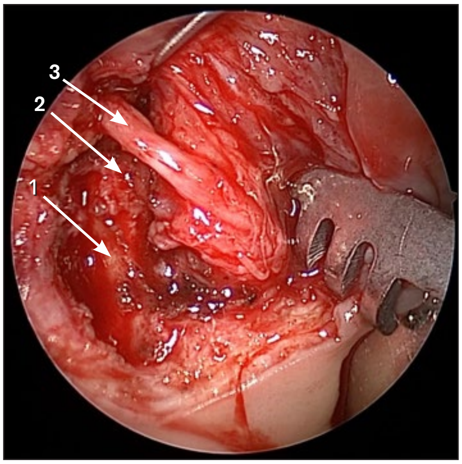

Fig 1 Intraoperative photograph of exposed long buccal nerve running over the left mandibular coronoid process to supply sensory innervation to the mandibular buccal gingiva, mandibular buccal sulcus, and cheek mucosa. *(1)* Left mandibular coronoid process. *(2)* Attachment of temporalis muscle to coronoid process. *(3)* Left long buccal nerve.

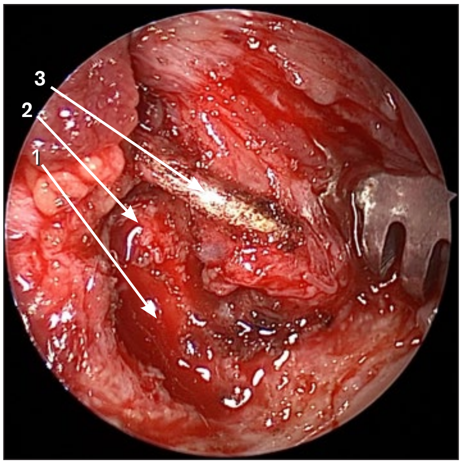

Fig 2 Intraoperative photograph of exposed long buccal nerve running over the left mandibular coronoid status post–epineural ablation. *(1)* Left mandibular coronoid process. *(2)* Attachment of temporalis muscle to coronoid process. *(3)* Left long buccal nerve.

with potential applicability to all terminal trigeminal branches, is described. The minimally invasive trigeminal ablation technique (MITA) for the long buccal nerve involves surgical decompression followed by targeted epineural thermolysis for debilitating and recalcitrant postoperative neuropathy.

Case Report

Presentation

The patient was a 44-year-old woman who had undergone left mandibular third molar surgery (#17, Universal Numbering System) and experienced postoperative persistent burning and hypersensitivity along the course of the long buccal nerve not responsive to alveoloplasty, steroid injections, or laser therapy. At presentation, the patient reported severe pain with light stroking of the buccal mucosa that was relieved with an infiltration of carbocaine at the site of the mandibular third molar extraction scar and was consistent with posttraumatic trigeminal neuropathy. Initial management included serial dexamethasone injections, oral tricyclics, and topical pregabalin, which failed to provide sufficient pain relief.

Preoperative Workup

Preoperatively, a long buccal nerve anesthesia block was performed using bupivacaine, resulting in both analgesia and nonbothersome anesthesia. This preoperative evaluation was repeated three times, and each proved to be efficacious for the patient, albeit temporarily. The patient was counseled on the option of an MITA long buccal nerve operation as a method to achieve long-lasting analgesia.

Procedure

Performed under general anesthesia in the usual manner of a clean contaminated case, an incision was made along the ramus of the mandible sharply dissecting until the periosteum was reached. Using endoscope guidance (a standard 4-mm Storz endoscope), the dissection was carried superiorly, exposing the long buccal nerve on the medial aspect of the coronoid process (Fig 1). The nerve was then widely exposed and decompressed from its fascial attachments and noted to be intact with no changes in caliber or quality along the nerve, indicating no neuroma present. Next, monopolar cautery with a Colorado Needle tip was used to ablate the epineurium of the nerve, with an emphasis on all work being performed medial to the coronoid (Fig 2).

Postoperative Course

Immediately postoperatively, the patient noted adequate analgesia with nonbothersome mild anesthesia along the long buccal nerve. This state has been maintained for 6 months postoperatively. Adjuvant treatment included a muscle relaxant and physical therapy for concurrent temporomandibular joint dysfunction. Complications include occasional cheek biting during mastication.

Discussion

The long buccal nerve is a branch of the mandibular division of the trigeminal nerve that carries sensory fibers supplying the mandibular buccal gingiva, mandibular buccal sulcus, and the cheek mucosa and may contribute to the extraoral cutaneous supply of the cheek.[8] As part of the course of the nerve, it passes across the external oblique ridge of the mandible and is at risk of being damaged by surgical incisions commonly used in oral surgical practice, such as the distal relieving incision for third molar surgery.[8]

However, clinically significant damage to this nerve appears to be exceedingly rare and is limited to case reports in the literature. One possible explanation is that the long buccal nerve is commonly injured in dental surgery, but the sensory changes in the buccal mucosa are asymptomatic.[9] Alternatively, others argue the nerve is not at risk as often as historically assumed. In a cadaver study involving the dissection of 20 long buccal nerves, Hendy et al found that in 70% of cases, the main trunk of the long buccal nerve was within 3 mm of the deepest concavity; a point at which incisions over the ramus do not normally extend during dental surgery. However, in the other 30% of specimens, the long buccal nerve crossed the ramus up to 12 mm below the deepest concavity, placing the nerve at much greater risk.[8]

Due to lack of evidence-based resources, the management of neurosensory deficits after peripheral trigeminal nerve injuries remains challenging. In the acute phase, reassurance with the possible addition of medical management (eg, steroids, nonsteroidal anti-inflammatories, neuropathic pain medications) may be all that is required in 51% to 92% of cases.[4,10–13] A reasonable window of conservative therapy is generally considered to be 6 months, after which surgical options should be considered.[4]

If medical management fails and the patient and physician choose to proceed with surgical intervention, there is limited preoperative testing that can characterize the nerve injury (eg, Sunderland I-V or neuroma formation); therefore, surgery should mainly be thought of as exploratory and the procedure dictated by intraoperative findings.[4] In cases where the nerve is found to be intact, the only currently described surgical option is external neurolysis.

External neurolysis is the dissection of a nerve from surrounding scar tissue. There are no studies specifically reporting outcomes for external neurolysis of the long buccal nerve. In a systematic review, Leung et al found that with external neurolysis of the lingual nerve, 25% (3/12) of subjects had complete recovery from the neurosensory deficit, 25% showed significant improvement, and another 25% showed some improvement.[9] However, this still left 25% with no improvement after the treatment. In contrast, when external neurolysis was performed for the inferior alveolar nerve for neurosensory deficits after mandibular surgery, Greenwood and Corbett noted that 28.6% (2/7) of subjects recovered completely, 28.6% (2/7) of subjects had some improvement in sensation, and 42.8% (3/7) of subjects had no improvement at all after the operation.[14]

Although external neurolysis can be effective, there can be no improvement in up to 42% of patients.[14] Although similar to external neurolysis, the present method of minimally invasive ablation of the long buccal nerve begins with nerve decompression, but has a distinct neurophysiologic advantage. Specifically, thermoablation of the nerve may selectively target the unmyelinated pain fibers over myelinated sensory fibers, resulting in minimizing postoperative anesthesia but maximizing analgesia. Using rat sciatic nerves as a model, Xu and Pollock studied the physiologic and morphologic patterns of thermal injury to nerves.[15] They noted that the unmyelinated nerve fibers (ie, pain-carrying fibers) showed a greater direct vulnerability to hyperthermia relative to the myelinated fibers (ie, general sensory fibers). This manifested as a reversible conduction block of C-fiber action potentials at higher temperatures via immediate and selective axonal degeneration. This concept has already been shown to be successful in the treatment of trigeminal neuralgia in a series of 36 patients.[16]

Although this technique appears promising for patients with refractory trigeminal neuropathy, it is important to note that it should be followed by robust and well-designed cohort studies with long-term follow-up describing the efficacy and complications of this novel procedure. Unfortunately, due to the retrospective nature of this study, only qualitative descriptions and not quantitative ratings (ie, pain intensity levels) were available for review, tempering the ability to fully define the patient's degree of improvement. Furthermore, patients with trigeminal neuropathy may have additional sources of pain, including temporal mandibular joint dysfunction and myofascial pain, that should be appropriately managed.

Conclusions

Treatment of iatrogenic trigeminal neuropathy remains challenging, and many patients fail to fully achieve self-recovery or find adequate relief with medical management. Surgical interventions have been shown to be effective in instances of a severed nerve or neuroma formation, while isolated nerve decompression of intact nerves can fail over half the time. This novel technique is minimally invasive, as endoscopes are utilized during the surgery through a small incision, allowing precise movement and the physiologic advantage of selectively targeting unmyelinated pain fibers. Additional studies are required to better define the efficacy of this promising procedure.

Acknowledgments

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