Journal of Cranio-Maxillo-Facial Surgery 43 (2015) 1541–1545

Contents lists available at ScienceDirect

Journal of Cranio-Maxillo-Facial Surgery

journal homepage: www.jcmfs.com

Surgical treatment of painful lesions of the inferior alveolar nerve

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ARTICLE INFO

Article history:
Paper received 1 April 2015
Accepted 30 July 2015
Available online 5 August 2015

Keywords:
Inferior alveolar nerve
Labial anesthesia
Labial pain

ABSTRACT

Nerve-related complications are being reported with increasing frequency following oral and dental surgery, and typically involve the inferior alveolar nerve (IAN). We assess herein the etiology of neuropathic pain related to IAN injuries, and describe the various surgical treatment techniques available.

Between 2007 and 2013, 19 patients were referred to the Maxillofacial Surgery Department of San Paolo Hospital (Milan, Italy) with pain in the area supplied by the IAN, which developed following endodontic treatment, oral surgery and maxillofacial surgery. All patients underwent IAN surgery by several different microsurgical procedures. Most of the patients affected by pain before surgery experienced complete or partial amelioration of symptoms. All patients receiving sural nerve grafts were pain-free 12 months after surgery. In five patients the operation was unsuccessful. In 78.94% of cases, a significant increase in nerve function was observed.

Pain following IAN surgical damage may be addressed by microsurgery; nerve substitution with a sural nerve interpositional graft appears to represent the most efficacious procedure. Scar releasing, nerve decompression and nerve substitution using vein grafts are less effective. Removal of endodontic material extravasated into the mandibular canal is mandatory and effective in patients experiencing severe pain. Surgery should be performed within 12 months postoperatively, ideally during the first few weeks after symptoms onset.

1. Introduction

Nerve-related complications following oral and dental surgery are being reported with increasing frequency, and typically affect the inferior alveolar nerve (IAN; 64.4% of all cases) (Tay and Zuniga, 2007; Juodzbalys et al., 2011; Biglioli et al., 2014a).

Nerve damage can occur during third molar, implant, orthognathic, pre-prosthetic and salivary gland surgery, or during the resection of benign or malignant neoplasms. Endodontic treatment and local anesthetic injections can also result in nerve damage (Tay and Zuniga, 2007; Biglioli, 2010; Cespedes-Sanchez et al., 2014), which can reduce quality of life by affecting speech, chewing and social interaction (Biglioli, 2010; Juodzbalys et al., 2011; Cespedes-Sanchez et al., 2014). Patients may present with either total anesthesia or hypesthesia. If associated neuropathic pain is present, immediate treatment is particularly important to prevent chronic and irreversible nerve damage (Tay and Zuniga, 2007; Biglioli, 2010).

We assess herein, the etiology of neuropathic pain related to IAN injury, and discuss the various surgical treatment options currently available.

2. Materials and methods

Between 2007 and 2013, 19 patients were referred to the Maxillofacial Surgery Department of San Paolo Hospital (Milan, Italy) with pain in the area supplied by the IAN (chin and lower lip), which developed after endodontic treatment or oral or maxillofacial surgery.

In four patients (21.05%), pain began following removal of a lower third molar; in seven others (36.84%) mandibular implant placement was the catalyzing event. In five patients, extravasation of material routinely used during endodontic treatment in the mandibular canal induced labial pain (26.31%); one patient (5.26%) experienced referred pain following the removal of a benign mandibular tumor; one patient (5.26%) experienced pain after

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http://dx.doi.org/10.1016/j.jcms.2015.07.036
1010-5182/

removal of a chondroblastoma extending from the temporomandibular joint to the ramus; and in one patient (5.26%) pain began after a sagittal mandibular osteotomy for obstructive sleep apnea syndrome.

Symptoms varied among patients, in several the pain was localized to a particular area. Other patients experienced pain in tissues innervated by the third trigeminal branch, which occasionally also extended into the second branch. In several cases, pain was constant, whereas in others it was more evident later in the day, commensurate with increased fatigue. For some patients nonsteroidal anti-inflammatory drugs provided temporary relief, but in the majority of cases they were ineffective except at excessively high doses. Eight patients (42.1%) had previously been prescribed antiepileptic drugs such as pregabalin and gabapentin. These patients were unwilling to take these drugs long term—potentially for the rest of their life—and were, therefore, motivated to explore surgical options. Others reported insufficient symptom relief with drug therapy.

Five patients (26.31%), experienced severe and constant dysesthesia, manifested in a burning sensation and electric shocks in the lips and chin. Although these patients did not initially refer to these symptoms as 'real pain', further questioning led to them being classified as such. We excluded patients exhibiting mild or moderate dysesthesia (that is cases in which symptoms were characterized as an intermittent 'nuisance' rather than as constant 'real pain').

The mean interval between patients' nerve lesions and their initial visit to our clinic was 90 days (range: 14–360 days). All patients underwent a neurophysiological examination that assessed tactile and pain sensory thresholds in the cutaneous area innervated by the IAN, and evaluated the masseter inhibitory reflex (MIR) in accordance with electrical stimulation on both the healthy and affected side. The examination did not reveal qualitative alterations in sensation (pain, dysesthesia) but reduced nerve conduction (hypesthesia, anesthesia) was consistently observed. The extent of the quantitative reduction in pain and sensitivity in the affected vs. the contralateral side, varied markedly among patients.

Panoramic X-ray (Panorex) and cone-beam computed tomography images of affected nerves and the surrounding area were acquired prior to surgery. In one of the four patients who developed pain following lower third molar extraction, part of the tooth's root was visible. In the other three cases the roof of the canal was damaged ($n = 2$) or partially displaced ($n = 1$).

Of the seven patients exhibiting IAN damage due to implantology, a continuation of drilling into the mandibular canal was evident in three cases (42.85%); in the remaining four patients (57.15%), an implant partially occupying the canal was observed. In two of these patients implants were cylindrical, and in the remaining two they were in blade-form.

Among the patients experiencing painful lesions after endodontic treatment, radiological images revealed a large quantity of endodontic material along the axis of the mandibular canal. CT scan findings for patients with benign lesions or sleep apnea indicated canal anatomy subversion after surgery.

Within 3 weeks after their first visit, all patients underwent IAN surgery.

All surgical procedures were performed by the first author (FB), under general anesthesia and using a surgical microscope. After achieving mucosal access into the lower vestibular fornix, the underlying mandibular bone was skeletonized and a large bony window (2.5 cm distal and 2.5 cm proximal to the lesion) was carved into the vestibular cortex using piezoelectric instrumentation (Figs. 1 and 2). The amplitude of the access ostectomy guaranteed good surgical vision and thus allowed for microsurgical

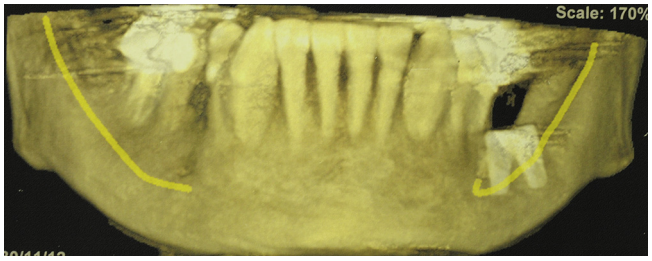

Fig. 1. Patient experiencing pain in the left IAN, consequent to a double lesion of the nerve after oral implant surgery.

nerve reconstruction when necessary (Figs. 3–5). After surgery the bony window was placed back in its original position and stabilized using titanium micro-plates and screws (Fig. 6). A simple suture closed the wound.

In the five patients experiencing pain following endodontic treatment, intraoperative findings revealed that the IAN was either completely destroyed ($n = 1$; 20%) or only a small portion remained ($n = 3$; 60%) due to chemical burns caused by the endodontic material; however, in one case (20%) the IAN appeared intact upon inspection. In all cases, surgery consisted of meticulous curettage of the canal; substitution of the nerve with an interpositional sural nerve ($n = 2$) or venous graft ($n = 1$) was additionally performed in cases of apparent nerve damage. Postoperative panoramic X-ray revealed good clearance of extravasated endodontic material, but small remnants were present in all cases.

Among the seven patients experiencing pain after implant surgery, three (42.85%) underwent immediate IAN reconstruction using either an interpositional nerve ($n = 2$) or vein graft ($n = 1$). In the two patients (28.57%) in whom a blade implant was partially displaced into the mandibular canal, the IAN was decompressed by removing the foreign body, with piezoelectric instrumentation used to break down the mandibular canal walls and bone spongiosa surrounding it. In the remaining two patients (28.57%), a cylindrical implant partially occupying the canal was trimmed intraoperatively, because surgical removal risked damage to the IAN and the related prosthesis was also already in place. Implants were skinned using a diamond bur to eliminate interference with the nerve.

Among the four patients with painful lesions caused by lower third molar extraction, two (50%) underwent IAN decompression, one (25%) of whom had an interpositional nerve graft, and the other (25%) had an interpositional vein graft substitution. The patient

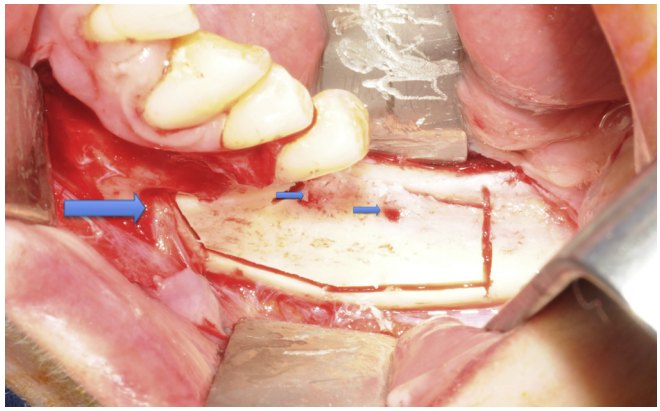

Fig. 2. A bony window is drawn on the cranial and lateral mandibular cortex by a piezoelectric device, to allow access to the IAN. The mental foramen (large arrow) and previous implant sites (small arrows) are visible.