Neuropathic pain after bilateral sagittal split osteotomy: management and prevention

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ABSTRACT

Neuropathic pain is characterized by spontaneous and provoked pain and other signs reflecting neural damage. Aberrant regeneration following peripheral nerve lesions leaves neurons unusually sensitive and prone to spontaneous pathological activity, abnormal excitability and heightened sensitivity to stimuli. This review covers the current understanding of neuropathic pain after bilateral sagittal split osteotomy (BSSO) of the lower jaw. The reported incidence of neuropathic pain after mandibular osteotomies is less than 1%, while the incidence in patients with iatrogenic inferior alveolar nerve (IAN) injuries during BSSO can be as high as 45%. The factors which modulate the healing process toward neuropathic pain during or after nerve damage have not yet been elucidated. Patients at highest risk for developing post-BSSO neuropathic pain are older than 45 years and have undergone procedures involving IAN compression, partial severance, or complete discontinuity of the lingual nerve with a proximal stump neuroma, patients with nerve injury repair delayed longer than 12 months and patients with chronic illnesses that compromise healing or increase risk for peripheral neuropathy. Although neuropathic pain tends to be long-lasting, some patients can recover completely. Preventive measures include risk assessment prior to surgery, prevention of nerve damage during surgery, and early repair of nerve injury.

Key words:

Bilateral sagittal split osteotomy, incidence, management, neuropathic pain, risk factor

INTRODUCTION

Neuropathic pain is a complex, chronic pain state caused by a lesion of the somatosensory nervous system.[1] It usually results from tissue injury and excludes pain from a condition preceding surgery.[2] Neuropathic pain can arise from damage to the nerve pathways at any point from the terminals of the peripheral nociceptors to the cortical neurons in the brain. In this type of pain, nerve fibers may be damaged, dysfunctional, or injured, resulting in a change in nerve function at both the site of injury and adjacent tissue. These damaged nerve fibers in turn send incorrect signals to other pain centers.[3]

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Neuropathic pain is characterized by spontaneous and provoked pain mostly of a burning character, by positive symptoms such as paresthesias and dysesthesias, and by negative signs (sensory deficits) reflecting neural damage. Sensory disturbances in the area of surgery show a strikingly strong association with persistent postsurgical pain, suggesting nerve damage as a contributing factor in a significant portion of cases.[2,3] Many investigations have confirmed the relevance of surgery as the initiating event for the development of persistent pain, even after a minor operation, such as tooth extraction.[1]

Bilateral sagittal split osteotomy (BSSO) is a common procedure used to treat mandibular deformity. Because mandibular osteotomies are performed in close proximity to the neurovascular bundle in the mandibular canal, there is a high risk of injury to the inferior alveolar nerve (IAN).[4-6] IAN injury during surgery largely results from manipulation

of the nerve, its vascular supply, or structures surrounding the nerve during surgery.[7-9] The placement of semi-rigid fixation plates and screws may also cause nerve damage either directly or via compression of the nerve between bony segments after screw fixation.

Inferior alveolar nerve-related neuropathic pain following iatrogenic damage to the nerve is a disabling condition that severely affects the quality of daily life.[10-12] This review covers the current knowledge regarding neuropathic pain after BSSO and its incidence, pathophysiology, risk factors, management, and steps for prevention.

NEUROPATHIC PAIN AFTER BILATERAL SAGITTAL SPLIT OSTEOTOMY

Incidence

No single accurate value appears to be available for the overall prevalence of neuropathic pain. The development of chronic pain after surgery is fairly common, with estimates ranging from 10% to 50% after many common operations.[13] The pain may be severe in 2-10% of these patients and is usually considered to be neuropathic.[14,15]

Information about neuropathic pain following orthognathic surgery is sparse.[16,17] Borstlap *et al.*[18] prospectively followed 222 patients after BSSO surgery and reported no incidence of neuropathic pain. The reported incidence of neuropathic pain in the literature after mandibular osteotomies is less than 1% while the reported incidence in patients with iatrogenic IAN injuries during BSSO can be as high as 45%. Marchiori *et al.*[19] reported seven cases of neuropathic pain among 1671 patients after BSSO, for an incidence of 0.42%, while Politis *et al.*[20] reported 6 cases of neuropathic pain from 900 BSSOs with an incidence of 0.67%.

Other reports[16,21] describe an incidence as high as 5% of neuropathic pain among patients who sustained peripheral trigeminal nerve injuries after sagittal split ramus osteotomy. Teerijoki-Oksa *et al.*[22] prospectively followed 19 patients after BSSO surgery and found a 5% overall occurrence of neuropathic pain at 1-year follow-up, which is similar to the overall estimated incidence of neuropathic pain after traumatic and iatrogenic nerve injuries.[23] Jääskeläinen *et al.*,[21] on the other hand, found a 45% incidence of neuropathic pain in 58 patients with iatrogenic sensory deficits of the IAN and lingual nerve (LN).

Microsurgical repair of a damaged IAN after orthognathic surgery does not alleviate neuropathic pain if the latter was present before the repair. Furthermore, it does not cause neuropathic pain if the pain was not present beforehand.[17]

Mechanism of nerve damage

The IAN is at significant risk in all stages of surgery [Table 1], and nerve manipulation during BSSO is a known risk factor for nerve injury.[24] This nerve can be damaged at the following points: the spyx during the placement of a retractor posterior to or above the lingual, the ascending ramus during a horizontal osteotomy cut, the bone cut at the lower border of the mandible, the connecting bone cut between the lower border and the buccal osteotomy of the mandibular body, with chiseling during the sagittal split, between bone fragments after the bony movement, during placement of the osteosynthesis material and during insertion of an osteosynthesis screw.

Table 1: Location, cause, and type of nerve damage during BSSO

Location	Cause	Type of lesion
Spyx	Retractors	Compression
Osteotomy area	Chisels, compression bony surfaces, freeing nerve, screws, piezo, drill, saw	Compression, crushing, transection
Lower border	Partial or total transection	Drill, saw, piezo

BSSO: Bilateral sagittal split osteotomy

Grades of nerve injury are categorized into neuropraxia, axonotmesis, or neurotmesis, depending on the extent of the damage.[25] In clinical settings, various combinations of nerve damage can coexist, giving rise to a variety of sensory dysfunctions. After a peripheral nerve lesion, aberrant regeneration may occur.[26] In some patients, neurons become unusually sensitive and develop a spontaneous pathological activity, abnormal excitability, and heightened sensitivity to chemical, thermal, and mechanical stimuli. Persistent pain or neuropathic pain such as allodynia, and pain and discomfort with occlusion[27,28] can occur.

CLINICAL CHARACTERISTICS OF NEUROPATHIC PAIN

The main features of neuropathic pain include constant pain, which can be superficial or deep, sharp or aching, lancinating pain (i.e. sudden and sharp, severe bursts of pain), and allodynia (i.e. pain experienced after normally nonpainful stimuli, like light touch). The discomfort is usually of a chronic nature and may be described by the patient as a burning sensation, a sharp, stabbing, or shooting pain, or "like an electric shock".[20]

The complaints often seem to be out of proportion to the pain that would be expected to accompany the original injury.[3,19] Neuropathic pain resulting from axonal nerve injury is often associated with crushing or stretching nerve injuries rather than total nerve transaction.[20] Other characteristics of neuropathic pain include a lack of response to anti-inflammatory pain killers (nonsteroidal anti-inflammatories, paracetamol), improved symptoms in the mornings, minimal sleep disturbance, and worsening during the day or with stress, fatigue, and illness.

RISK FACTORS FOR NERVE DAMAGE AND NEUROPATHIC PAIN

The proximity of the mandibular canal to the lower border of the mandible is an important factor in self-reported hypoesthesia of the lower lip.[27] The exposure and

dissection of the IAN from the mandibular canal during surgery has been shown to significantly increase the risk of neurosensory disturbance, while patients with a laceration of the IAN have higher chance of developing neuropathic pain.[29]

Genioplasty and age at the time of surgery are significant predictors of hypoesthesia after BSSO, a 1-year increase in age may increase the odds of hypoesthesia of the lower lip by 5%, and the odds of hypoesthesia for patients with concurrent genioplasty are 4.5-fold greater than the odds for patients without concurrent genioplasty. Other factors include smoking and gender (women are at higher risk for hypoesthesia).[27,29]

Patients most likely to develop neuropathic pain after BSSO are older than fourty-five years and have undergone a procedure involving compression or partial severance of the IAN or complete discontinuity of the LN with a proximal stump neuroma. Others at risk include those with nerve injury repair delayed past twelve months, patients with chronic illnesses that compromise healing or enhance the risk for developing peripheral neuropathy (e.g. diabetes mellitus) and patients with preexisting chronic pain from any cause (e.g. lower back pain, postthoracotomy syndrome). Furthermore, potentially at risk are patients with certain psychological features such as depression, anger issues, posttraumatic stress disorder, and victims of abuse who have lost the ability to trust.[19,26,30,31] Patients undergoing orthognathic surgery are usually young and healthy, which may explain the low incidence of neuropathic pain after BSSO surgery.

THE PATHOPHYSIOLOGY OF NEUROPATHIC PAIN

Chronic neuropathic pain represents a heterogeneous group of diseases in which pain is caused by nerve damage owing to various etiologies. Before pain is perceived in the central nervous system, different descending mechanisms must modulate the initial nociceptive stimulus. The imbalance between the amount of stimuli and the efficacy of modulation mechanisms is processed as the sensation of pain. High-magnitude or repetitive nociceptive impulses cause peripheral and central neuronal changes, leading to the maintenance and exacerbation of the pain sensation.[26] These alterations are often irreversible and responsible for patient reports of long-term pain, even after many unsuccessful treatments. Most of the current ideas regarding the pathophysiology of neuropathic pain originated from experimental work in animal models. The underlying mechanisms are described below.[26]

Peripheral sensitization

Pain sensations are normally elicited by activity in unmyelinated (C-) and thinly myelinated (Aδ-) primary afferent neurons. These nociceptors are usually silent in the absence of stimulation and respond best to potentially noxious stimuli. Neurons become abnormally sensitive after damage to peripheral nerves and develop pathological spontaneous activity.[32] These pathological changes result from molecular and cellular changes at the level of the primary afferent nociceptor that are triggered by the nerve lesion. They are expressed as increased spontaneous firing, lowered activation threshold, and expanded receptive fields.[33]

Central sensitization

Hyperactivity of the peripheral nociceptor results in secondary changes in the dorsal horn of the spinal cord with an associated increase in general excitability of multi-receptive spinal cord neurons. This hyper-excitability is manifested by increased neuronal activity in response to noxious stimuli, expansion of neuronal receptive fields, and spread of spinal hyper-excitability to other segments. Central sensitization is initiated and maintained by activity in pathologically sensitized C-fibers. Importantly, the activation of both descending facilitatory and inhibitory supraspinal pain control systems requires intense noxious stimulation, resulting in activation of these brainstem centers to finally activate the descending arm of thespino-bulbo-spinal circuit.[26] An imbalance between facilitatory and inhibitory systems, with higher activity in the former and lower in the latter, contributes to central neuronal sensitization and to the development and maintenance of pain.[26]

Deafferentation: hyperactivity of central pain transmission neurons

Some patients experience a profound cutaneous deafferentation of the painful area without significant allodynia. In BSSO and orofacial neuropathic pain, the simultaneous occurrence of an exposed nerve or partial axonal IAN injury together with disruption of the bony environment of the IAN is a risk factor.[34] The formation of a neuroma from a severed nerve ending has been associated with neuropathic pain, which is attributed to altered sensory processing in either the trigeminal ganglion or the central trigeminal nerve center.[35] Politis *et al*.[20] found no visible nerve damage on panoramic radiographs or magnetic resonance imaging (MRI) or computerized tomography (CT) scans in their case series of neuropathic pain after BSSO surgery, except in one patient in whom neuropathic pain started after loss of fixation and pathological movement of the bone segments due to pseudarthrosis. In this patient, the neuropathic pain disappeared after bone grafting and stabilization of the segments with adequate fixation.

Compression or crush lesions cannot be routinely visualized after orthognathic surgery by either CT or MRI secondary to artifacts from orthodontic appliances. Pathologic elongation of the nerve in BSSO surgery is certainly possible when the mandible has been surgically widened after a BSSO advancement with a midline split. Here too, cone beam CT, CT, and MRI cannot be used to directly visualize the nerve damage.

DIAGNOSIS

The diagnosis of neuropathic pain should be made only when the history and signs are indicative of neuropathy

in conjunction with a neuro-anatomically correlated pain distribution and sensory abnormalities within the area of pain. There should be partial or complete sensory loss in all or part of the painful area, and confirmation of a lesion or disease by quantitative sensory testing, surgical evidence, imaging, clinical neurophysiology, and/or biopsy.[23]

Neuropathic pain should also be differentiated from other similar orofacial pain. The differential diagnosis of neuropathic pain includes inflammatory pain, traumatic trigeminal neuropathy, persistent idiopathic facial pain (atypical facial pain), atypical odontalgia, complex regional pain syndrome, and trigeminal neuralgia.[23]

MANAGEMENT

Neuropathic pain tends to be long-lasting, although some patients recover completely, and others may find relief with pharmacotherapy and learn to cope with their symptoms. Neuropathic pain is treated mainly with anti-depressants and anti-epileptics, whereas simple analgesics are not efficacious. Management of pain should be tailored to the individual patient on the basis of pain type(s), the causative disease(s), and psychosocial aspects.

Psychological management

The assessment of neuropathic pain needs to include the measurement of multiple aspects of the quality of life. Mood, physical and social functioning, and pain-coping strategies such as catastrophizing and social support are all important domains. As with other chronically painful conditions, cognitive-behavioral interventions may improve the quality of life in neuropathic pain conditions.[31] Reassurance and counseling of patients with neuropathic pain will go a long way toward alleviating their condition.

Medication

Neuropathic pain treatment remains unsatisfactory despite a substantial increase in the number of trials.[36] The use of low-dose anti-depressants (amitriptyline, nortriptyline) is effective for symptomatic relief.[37] carbamazepine, phenytoin, and valproic acid are effective in ameliorating diabetic neuropathy-related pain. Other anti-epileptic agents, including lamotrigine, oxcarbazepine, and topiramate, show some benefit for the treatment of neuropathic pain, although some studies have found them to be ineffective.[37,38]

Topical 5% lidocaine patches offer a new therapeutic alternative for patients suffering from neuropathic pain. These patches have been shown to be useful in a subgroup of patients.[39]

In BSSO patients, an accurate preoperative patient history, as well as early identification of the patient with severe or prolonged pain with the aim of initiating pain treatment as early as possible, is the key to success.[29,34] Kuhlefelt *et al*.[29] suggest that patients with IAN damage after BSSO be put on neuropathic pain medication immediately postoperatively before pain is well established. Psychological support and the volunteer of information by the surgeon are also important at this time.

Surgical management

Early repair of nerve injury has been deemed to be the most critical factor in the surgical management and prevention of neuropathic pain. For example, once the neuropathic pain has set in, late surgical trigeminal nerve repair will not improve the patient's symptoms.[20] When an iatrogenic nerve injury is suspected, regular follow-up is advised. If there is no improvement during 10-12 weeks of follow-up or there are complaints of dysesthesia, surgical exploration, localization, and immediate repair or repair within days is advised. Repair should be carried out with a tension-free approximation.

PREVENTION

Patient profiling should be done and identification of risk factors for developing neuropathic pain made in all patients scheduled for orthognathic surgery. Proper localization of the IAN before BSSO is also an essential preventive step. The advent of cone beam CT has made IAN canal assessment in three-dimensions possible.

Furthermore, the development and modification of surgical techniques to reduce nerve injury during BSSO, such as safe surgical access to the mandibular nerve at the infratemporal fossa,[40] and a modified technique to control the lower mandibular border cut,[41] have been critical in reducing the incidence of damage to the IAN. Also useful is assessment of the IAN during BSSO, as by continuous monitoring of the status of the mandibular nerve through observation of changes in the sensory action potentials of the nerve during surgery.

Severe nerve injuries often result from drilling too deep past the bone into the nerve, or from placing the osteosynthesis screw on the nerve during fixation. The use of intraoperative CT during BSSO allows for the intraoperative evaluation of osteosynthesis screw penetration and depth. Intraoperative CT also allows for immediate assessment of treatment and provides the option to modify treatment if necessary. These preventive measures will help reduce the incidence of neuropathic pain and improve the quality of life of BSSO patients.

CURRENT TRENDS AND FUTURE PROSPECTS

Because neuropathic pain after BSSO involves an injured peripheral nerve which sends incorrect signals to neurons located in Meckel's cave, a temporary inhibition of such signals might be beneficial. Affordable long-acting liposomal local anesthetics, navigation guided procedures targeted at the exit of the mandibular nerve in the oval foramen, and miniaturized intra-oral neurostimulators applied proximal to the site of the nerve damage are possible treatment options that are currently under investigation.

CONCLUSION

Neuropathic pain after BSSO surgery is rare in spite of the frequent hypoesthesia that accompanies this surgical procedure. Contributing factors include patient factors (age, gender, patient profile), nerve-related factors (elongation, crushing, compression, transection), and local factors around the nerve (ischemia, bone infection). Once neuropathic pain has been established for more than three months, microsurgical nerve repair is unlikely to be successful in relieving the pain.

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