

Analysis of Neurosensory Dysfunction after Dental Implant Surgery

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Purpose: There have been reports regarding the various factors associated with the level of discomfort and recovery from neurosensory symptoms in patients with trigeminal nerve injury. However, the contributing factors remain uncertain and poorly understood. The purpose of this paper was to investigate the possible association between various factors expected to affect neurosensory discomfort and recovery in patients with mandibular nerve injury after dental implant surgery.

Methods: Eighty-nine post-dental implant surgery patients with mandibular nerve injury were enrolled in this retrospective analysis. A medical records review of the patients was done to determine if the patients' improvement was related to pain intensity, the length of time between the injury and removal of the implant or the depth of penetration of the implant into the mandibular canal as determined by cone-beam computed tomography.

Results: There was no significant linear relationship between pain intensity and symptomatic improvement ($p=0.319$). There was no significant linear relationship between the level of mandibular canal penetration and either pain intensity ($p=0.588$) or symptomatic improvement ($p=0.760$). There was a statistically significant linear relationship between length of time before the injury was treated, both with pain intensity ($p=0.004$), and symptomatic improvement ($p=0.024$).

Conclusions: Our findings indicate that the length of time between nerve injury and initiation of conservative treatment is more closely related to the pain intensity and symptomatic improvement than other factors, including the level of mandibular canal invasion. Additionally, increased pain intensity and decreased symptomatic improvement can be expected over time, because of this linear trend. Therefore, although direct injury to the nerve is the most important factor contributing to a neurosensory disturbances, early neurosensory assessment and initiation of conservative treatment should be done to optimize recovery.

Key Words: Dental implants; Mandibular nerve; Trigeminal nerve injuries

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INTRODUCTION

Injury to the branches of trigeminal nerve is a well-known risk factor in dental procedures and maxillofacial surgery. Altered sensations or dysesthesias related to nerve injury are recognized complications that may follow dental and surgical procedures of the mandible.¹⁾ The most commonly injured branches are the inferior alveolar nerve (IAN), the lingual nerve (LN), and the mental nerve (MN).²⁾

Although nerve injury during implant surgery is considered rare, unintentional and generally unsuspected serious complications do occur, two of the most notable being hemorrhage and infection.²⁾ Recent literature suggests that, increased use of mandible implants is associated with greater risk of trauma to the IAN. Results from a prospective multicenter study of partially edentulous patients with mandibular implants suggest that altered sensations may be more prevalent than previously reported,³⁾ and there is

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considerable evidence suggesting that IAN injury is the most common injury experienced by patient undergoing dental implant surgery.⁴⁾

Prospective studies have reported higher incidences of injury to the IAN with implant placement in the posterior mandible. The incidence is variably reported from 3.7% to 39.1% at 1 week postoperative examination.⁵⁻⁸⁾ Ellies and Hawker,⁹⁾ in a two-center retrospective study done 2 weeks after implant surgery, reported altered sensation of the IAN in 37% and 36% of the patients respectively. In both centers, these complaints were persistent in 13% of patients. The incidence of permanent sensory dysfunction (including dysesthesia, anesthesia or paresthesia) was 1% to 8% respective to the center, but the incidence of chronic pain is unclear.¹⁰⁾ In a Korean study, Ryu and Kwon¹¹⁾ reported the incidence of dysesthesia after dental implant surgery was 24.6%, and the incidence of permanent dysesthesia was 6.2%.

There have been a number of reports on various factors related to the level of discomfort and recovery of normal sensation in post dental implant patients. Merrill¹²⁾ has suggested that nerve damage can lead to transient or persistent alteration in orofacial sensations depending on the severity of the injury. Hillerup¹³⁾ stated that recovery of normal sensation was related to the etiology of the injury. Kraut and Chahal¹⁴⁾ suggested that spontaneous recovery of normal sensation after nerve injury is dependent both on the severity of the injury and the nerve involved. For example, partial transection of the LN is less likely to result in spontaneous resolution of symptoms, compared with similar injury involving the IAN. In another study, Tay and Zuniga¹⁵⁾ reported that there was no significant correlation between the severity of nerve injury and the time elapsed from injury.

In this study of post dental implant surgery patient who sustained mandibular nerve injury, we investigated the possible relationship between factors expected to affect neurosensory dysfunction and recovery.

MATERIALS AND METHODS

1. Patients

Ninety-four subjects who had post-dental implant abnormal sensation and/or neuropathic pain in the jaw were referred to Department of Orofacial Pain and Oral Medicine,

in the Dental Hospital of Yonsei University, Seoul, Korea. Each patient was examined and received a radiological evaluation with computed tomography. We excluded subject from the study if their injury was due to procedures other than dental implant surgery, if the injury was to the maxillary division or if the patient was seen only once. On this basis, five individuals were excluded, and the final sample was comprised of 89 patients who gave a written informed consent, 61 (68.5%) being female and 28 (31.5%) being male. Their mean age was 53.9 ± 9.9 years (range 27-75 years). The study protocol was approved by the institutional review board of Yonsei University Hospital (2-2013-3301).

2. Methods

We reviewed each subject's medical records to identify confounding factors that could influence neurosensory dysfunction or symptomatic improvement after conservative treatment. Those factors were pain intensity, level of invasion into mandibular canal in computed tomography (CT), and duration of time from nerve injury to initiation of conservative treatment.

1) Neurosensory disturbance

At the initial visit, the patients' neurosensory changes were evaluated using pin-prick, light touch, 2-point discrimination, pressure-pain threshold, brush stroke direction test, and current perception threshold (CPT) using Neurometer CPT/C (Neurotron Inc., Baltimore, MD, USA).

The patients with electric or burning pain were asked to grade the severity of their pain on a 10-point visual analogue scale (VAS) and the patients were divided into four groups according to VAS: (1) no pain (VAS=0); (2) mild pain ($0 < \text{VAS} < 4$); (3) moderate pain ($4 \leq \text{VAS} < 7$); (4) severe pain ($7 \leq \text{VAS} \leq 10$).

2) Symptomatic improvement after conservative treatment

The conservative treatment group received treatment consisting of behavioral, physical, and pharmacologic management.¹⁶⁾ This group was divided into three groups: (1) 'good improvement'—satisfactory improvement and few disabilities on routine daily work; (2) 'some improvement'—unsatisfactory improvement with some remaining disabilities during

routine daily work; or (3) 'no improvement'.

3) Level of invasion into mandibular canal in CT

CTs were taken to indirectly confirm implant invasion into the IAN. The confirmed cases were divided into three groups: (1) 'Non-contact'—the implant was not in contact with the mandibular canal; (2) 'Contact'—the implant was in close contact with the mandibular canal, but the cortical layer of canal was preserved; or (3) 'Penetrated'—the implant penetrated the mandibular canal wall and discontinuity of cortical layer was observed.

4) Duration of time from nerve injury

Patients were divided into two groups according to the time from nerve injury to initiation of conservative treatment involving behavioral, physical, and pharmacologic management. The criteria for were: (1) within 6 months of the nerve injury; (2) over 6 months from nerve injury.

5) Relation between pain intensity and other factors

A validation was performed on the relationship between pain intensity and other factors, i.e., symptomatic improvement after conservative treatment, level of invasion into mandibular canal, duration of time from nerve injury.

6) Relation between symptomatic improvement and other factors

An additional validation was performed on the relationship between symptomatic improvement after conservative treatment and other factors, i.e., the level of invasion into mandibular canal, duration of time from nerve injury.

3. Statistics

Statistical analyses were performed using the Wilcoxon

signed-rank test, Kruskal-Wallis test, Mantel-Haenszel χ^2 test, Mann-Whitney test. Statistical significance was established at 95%. SPSS Statistics version 17.0 statistical package (SPSS Inc., Chicago, IL, USA) was used for all statistical analyses.

RESULTS

1. Subject Demographics

The sample comprised 89 patients with nerve injury. Female patients were 61 (68.5%) and male patients were 28 (31.5%). Their mean age was 53.9±9.9 years (range 27-75 years). The mean age by sex distribution was 54.7±9.4 years in female, and 52.1±10.9 years in male.

2. Test Result for Neurosensory Disturbance

As presented in Table 1, both median value of pin-prick and light touch perception were significantly decreased on the injured side (65.0, 0-3,000.0; 60.0, 0-1,000.0) compared with the uninjured side (100.0, p=0.008; 100.0, p=0.004), and median perceived count of brush stroke was significantly decreased in injured side (29.0, 0-30.0) than in uninjured side (30.0, 4-30.0; p<0.001). The median distance for 2-point discrimination was significantly increased in injured side (13.4, 0-55.0) compared to the uninjured side (10.2, 0-72.4; p<0.001). Median force of pressure-pain threshold showed no significant difference between the injured side (160.0, 0-300) and the uninjured side (160.0, 50-270; p=0.785).

As presented in Table 2, in the frequencies of 2,000 Hz, 250 Hz, and 5 Hz, the median CPT values were statistically significantly increased in injured side compared with uninjured side (2,000 Hz, p<0.001; 250 Hz, p=0.001; 5 Hz, p=0.003).

Table 1. Result for clinical neurosensory test

Test	Injured side (n=89)	Uninjured side (n=89)	p-value
Pin-Prick (%)	65.0 (0-3,000.0)	100.0	0.008*
Light touch (%)	60.0 (0-1,000.0)	100.0	0.004*
2-Point discrimination (mm)	13.4 (0-55.0)	10.2 (0-72.4)	<0.001*
Pressure-pain threshold (cN)	160 (0-300.0)	160 (50-270)	0.785
Brush stroke direction (count)	29.0 (0-30.0)	30 (4-30)	<0.001*

Values are presented as median (range).

*Statistically significant at significance level of p<0.05.

3. Pain Intensity and Improvement with Conservative Treatment

Table 3 presents the relationship between pain intensity and symptomatic improvement after conservative treatment. There were 17 (19.1%) patients who complained of sensory change only, and 72 (80.9%) who complained of both pain and sensory changes.

Thirty two (36.0%) patients reported good improvement after conservative treatment, 29 (32.6%) reported some improvement, and 28 (31.5%) reported no improvement.

Median VAS scores were 5.0 (0-9) in the group with good improvement, 4.0 (0-9) in the group reporting some

improvement, and 6.0 (0-10) in the group reporting no improvement. There was no significant statistically significant difference between the group ($p=0.129$), and there was no significant linear-relationship between pain intensity and symptomatic improvement ($p=0.319$).

4. Relation between the Implant and the Mandibular Canal by CT

As presented in Table 4, there were 9 (10.1%) subject in the non-contact group, 14 (15.7%) subjects in the contact group, and 66 (74.2%) subjects in the penetrated group.

Median VAS score were 4.4 (0-9) in the non-contact group, 6.0 (2-9) in the contact' group, and 5.0 (0-10) in the penetrated group. There were no significant differences between each groups ($p=0.484$), and no significant linear-relationship between the level of mandibular canal invasion and pain intensity ($p=0.588$).

As shown in Table 5, there was no significant linear-relationship between the level of mandibular canal invasion and symptomatic improvement ($p=0.760$).

Table 2. Result for current perception threshold test

Frequency (Hz)	Injured side (n=23)	Uninjured side (n=23)	p-value
2,000	275.0 (138.0-524.0)	134.0 (89.0-255.0)	0.000*
250	120.0 (41.0-292.0)	51.0 (10.0-188.0)	0.001*
5	72.0 (7.0-240.0)	31.0 (8.0-115.0)	0.003*

Values are presented as median (range).

*Statistically significant at significance level of $p<0.05$.

Table 3. Analysis of pain intensity after symptomatic treatment

Variable	Improvement			Total	p-value
	Good	Some	No		
Pain intensity					0.319
No	7 (41.2)	5 (29.4)	5 (29.4)	17 (100.0)	
Mild	4 (44.4)	5 (55.6)	0 (0)	9 (100.0)	
Moderate	9 (27.3)	14 (42.4)	10 (30.3)	33 (100.0)	
Severe	12 (40.0)	5 (16.7)	13 (43.3)	30 (100.0)	
Total	32 (36.0)	29 (32.6)	28 (31.5)	89 (100.0)	
VAS	5.0 (0-9)	4.0 (0-9)	6.0 (0-10)		0.129

Values are presented as number (%) or median (range).

VAS, visual analogue scale.

Statistically significant at significance level of $p<0.05$.

Table 4. Analysis of level of invasion into mandibular canal in computed tomography compared with pain intensity

Variable	Pain intensity				Total	VAS
	No	Mild	Moderate	Severe		
Invasion level						
Non-contact	2 (22.2)	0 (0)	5 (55.6)	2 (22.2)	9 (100.0)	4.4 (0-9)
Contact	0 (0)	1 (7.1)	9 (64.3)	4 (28.6)	14 (100.0)	6.0 (2-9)
Penetrated	15 (22.7)	8 (12.1)	19 (28.8)	24 (36.4)	66 (100.0)	5.0 (0-10)
p-value			0.588			0.484

Values are presented as number (%) or median (range).

VAS, visual analogue scale.

Statistically significant at significance level of $p<0.05$.

5. Effects of Duration of Time from Nerve Injury to

Initiation of Conservative Treatment

As presented in Table 6, 51 (57.3%) patients were seen within 6 months of the nerve injury and 38 (42.7%) were seen more than 6 months after the nerve injury. Median VAS score were significantly decreased in within 6 months group (4.0, 0-9), than in over 6 months group (6.0, 0-10; p=0.047).

There was a statistically significant linear-relationship between the duration of time from nerve damage and pain intensity (p=0.004) and, as shown in Table 7, there was statistically significant linear-relationship between duration of time from nerve damage and symptomatic improvement with conservative treatment (p=0.024).

Table 5. Analysis of level of invasion into mandibular canal in computed tomography compared with symptomatic improvement

Variable	Improvement			Total
	Good	Some	No	
Invasion level				
Non-contact	4 (44.4)	1 (11.1)	4 (44.4)	9 (100.0)
Close contact	5 (35.7)	7 (50.0)	2 (14.3)	14 (100.0)
Penetrated	23 (34.8)	21 (31.8)	22 (33.3)	66 (100.0)
p-value	0.760			

Values are presented as number (%).

Statistically significant at significance level of p<0.05.

Table 6. Analysis of duration of time from nerve injury to pain intensity

Variable	Pain intensity				Total	VAS
	No	Mild	Moderate	Severe		
Duration of time (mo)						
≤6	14 (27.5)	6 (11.8)	19 (37.3)	12 (23.5)	51 (100.0)	4.0 (0-9)
>6	3 (7.9)	3 (7.9)	14 (36.8)	18 (47.4)	38 (100.0)	6.0 (0-10)
p-value	0.004*					0.047*

Values are presented as number (%) or median (range).

VAS, visual analogue scale.

*Statistically significant at significance level of p<0.05.

Table 7. Analysis of duration of time from nerve injury to symptomatic improvement

Variable	Improvement			Total
	Good	Some	No	
Duration of time (mo)				
≤6	21 (41.2)	20 (39.2)	10 (19.6)	51 (100.0)
>6	11 (28.9)	9 (23.7)	18 (47.4)	38 (100.0)
p-value	0.024*			

Values are presented as number (%).

*Statistically significant at significance level of p<0.05.

DISCUSSION

Trigeminal neuropathic pain following tissue or nerve injury in the nerve distribution is classified as traumatic trigeminal neuralgia. Traumatic neuralgia is defined as continuous pain following complete or partial damage to a part of peripheral or central nervous system. Local anesthetic blockade may not totally abolish the pain due to centralization of the pain mechanism and/or a sympathetic component.^{10,17)} Significant neurosensory dysfunction or pain due to the trauma may affect the patient's quality of life. Sometimes the patient requires the skill of a clinical psychologist or psychiatrist with clinical experience in pain.¹⁸⁾

In the present study, females outnumbered males at least two to one. This ratio has been consistently noted in studies from various pain centers and is suggestive of a potential hormonal link with the tendency to develop neuropathic pain after nerve injury. Smith et al.¹⁹⁾ have reported on the genetic variability associated with increased risk of developing chronic pain after nerve injury. But, Pogrel and Thamby,⁶⁾ Hillerup,¹³⁾ Tay and Zuniga¹⁵⁾ have suggested that patients referred to tertiary care centers do not represent the true incidence of nerve injury from various causes.

Kraut and Chahal¹⁴⁾ suggested that when nerve injury is suspected, a thorough neurosensory examination (light

touch, pain perception, direction of brush movement, two-point discrimination, temperature sensitivity) should be performed. Further, any unusual patient reactions during surgery, such as sharp pain or an electrical shock-like sensation should be documented. In the present analysis, clinical neurosensory testing showed significant differences between the injured side and the uninjured side with the exception of the pressure-pain threshold test. It should be noted, however, that these types of tests have some attached difficulties such as the danger of examiner bias or lack of technical equilibration between examiners. Consequently, there is a necessity for more standardized and objective examination procedures to test for neurosensory disturbance.

CPT testing may be performed to assess the damages of peripheral nerve in trigeminal nerve area. The nerve fibers with varied thickness are responsive selectively to the electric stimulation with different frequencies. Therefore sensory disturbances such as hyperesthesia or hypoesthesia can be diagnosed when responses outside of these norms are observed.^{20,21)}

At the onset of this study, we anticipated that pain intensity would increase and symptomatic improvement would decrease relative to the level of implant invasion into mandibular canal. However, there was no significant relationship between the level of invasion and pain intensity in this analysis. Additionally, there was no significant relationship between the anatomic invasion depth and symptomatic improvement.

When we studied the CT images, we noted that most of implant injuries to the mandibular nerve were caused by direct contact of implant itself with the mandibular canal. These nerve injuries may occur directly from the drilling procedures, lack of attention to diagnostic information, and/or direct compression of the nerve during implant insertion.^{5,14,22)} Variables such as magnification errors, unexpected anterior loop of the MN, operator technique, or anatomical variability can increase the chance of complications. Additionally, there may be more than one IAN or canal,²³⁾ and a plexus of nerve branches may lie superior to and outside the mandibular canal or infrequently, the preparation drill may be 1 mm longer than implant. Allowances for these variabilities must be incorporated into the calculation of available bone height.⁵⁾

Interestingly, 10.1% (9 cases) of patients didn't show direct contact between affecting implant fixture and mandibular canal and the distances between fixture and canal wall ranged from 1 mm to 3 mm. In cases such as these, it has been postulated that the damage may be due to implant induced indirect trabecular bone compression or condensation of the nerve,²⁴⁾ or thermal injury due to excessive heat build-up during the drilling procedure. Hirsch and Brånemark²⁵⁾ have suggested the following causes of nerve injury linked to implant surgery: 1) direct mechanical damage, 2) pressure on nerve and blood vessel, or 3) formation of hemangioma or osteoma followed by vascular damage. On the other hand, in some cases of LN and MN injury, the nerve may be traumatized by incision, flap elevation, retraction, or suturing procedures.^{14,26)} Based on these possibilities, clinicians shouldn't ignore the potential of indirect nerve damage when placing a dental implant.

Additionally, current recommendations in are to start the patient on high doses of steroids to decrease the neurogenic inflammation that will further injure the traumatized nerve.²⁷⁾

Tay and Zuniga¹⁵⁾ reported that there was no significant correlation between the severity of nerve injury and the length of time from injury. However, Ryu and Kwon¹¹⁾ reported that recovery ratio of injured nerve after implant surgery was 71.8%, and 88.2% of recovered group was recovered within 6 months.

In the present study, there was a significant linear relationship between duration of time and pain intensity as well as between duration of time and symptomatic improvement. Additionally, 26.3% of patients who suffered implant placement nerve injury over 1 year prior to this study showed satisfactory symptomatic improvement after conservative treatment. Based on this observation, conservative treatment should be initiated for patients who have had an implant-induced nerve injury for over 1 year. In the present study, 36% of the patients showed satisfactory symptomatic improvement after conservative treatment.

The present analysis was subject to some limitation. Intensity of pain and improvement of neurosensory symptom were based on self-reported and subjective information. And this analysis can't include a large number of patients, which resulted in difficulties controlling external factors such as the age and gender distributions. And in future study,

clinical neurosensory test and CPT test should take place regularly for more objective, regular evaluation of neurosensory recovery.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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