Analysis of neurosensory dysfunction after dental implant surgery

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Analysis of neurosensory dysfunction after dental implant surgery

A Masters Thesis

Submitted to the Department of Dental Science

And the Graduate School of Yonsei University

in partial fulfillment of the

requirements for the degree of

Master of Dental Science

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December 2010

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December 2010

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힘든 타향살이를 함께하면서도 형을 먼저 생각해주는 동생 영빈이에게 고맙고, 마지막으로 항상 걱정만 끼쳐드리는 부족하고 살갑지 못한 아들을 멀리서 믿고, 격려해주시고, 기대해주시는 아버지 어머니께 진심으로 감사한 마음을 전합니다.

2010년 12월

저자 씀.

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ABSTRACT

Analysis of neurosensory dysfunction after dental implant surgery

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Background: There have been some reports on the various factors that are associated with the level of discomfort and recovery of neurosensory discomfort in patients with trigeminal nerve injury, however their relation remains uncertain and poorly understood.

Objective: The purpose of this analysis was to investigate the possible association between some of the factors thought to affect neurosensory dysfunction and recovery from it in patients with mandibular nerve injury after dental implant surgery.

Methods: 89 patients with mandibular nerve injury after dental implant surgery from January 2007 to October 2010 enrolled in this analysis. Level of invasion into the mandibular canal as assessed by CT, retrieval of the affecting implant to decompress the injured nerve, and time elapsed since nerve injury were evaluated through medical record review.

Results:

1. There was no significant linear relationship between pain intensity and symptomatic improvement (p=0.319).
2. There was no significant linear relationship between the level of mandibular canal invasion and either pain intensity (p=0.588) or symptomatic improvement (p=0.760).

3. There was no significant linear relationship between retrieval of the affecting implant fixture to decompress the injured nerve and either pain intensity (p=0.934) or symptomatic improvement (p=0.121).
4. There was a statistically significant linear relationship between time elapsed since nerve damage and both pain intensity (p=0.004) and symptomatic improvement after conservative treatment (p=0.024).

Conclusion: Obtained findings indicate that the lapsed time from nerve injury to initiation of conservative treatment is most closely related to the pain intensity and symptomatic improvement than other factors including level of mandibular canal invasion. And increased pain intensity and decreased symptomatic improvement can be expected as time goes on, because of their linear trend. Therefore, although direct nerve injury is the most important factor in the initiation of neurosensory disturbance, if a nerve injury is suspected during implant surgery, early initiation of conservative treatment followed by neurosensory evaluation should be performed to reduce patient discomfort and improved the probability of nerve recovery.

Key words: nerve injury, implant, mandibular nerve

Analysis of neurosensory dysfunction after dental implant surgery

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I. INTRODUCTION

Injury to the branches of the trigeminal nerve is a well-known risk of major maxillofacial surgery and dental procedures. Reported causes of nerve injury in dentistry include third-molar extractions, dental implant surgery, needle trauma following block anesthesia, periapical inflammation, nerve compression by dentures, and nerve injury following root-canal treatment. Altered sensation of injured nerve is also a recognized complication that may follow dental and surgical procedures of the mandible (Bartling R, 1999). The most commonly injured branches are the inferior alveolar nerve (IAN), lingual nerve (LN), and mental nerve (MN) of the mandibular branch (Burstein J et al., 2008).

Extensive mechanical nerve damage is associated with myelinated and unmyelinated nerve fiber hyposensitivity, and is characterized clinically by elevated threshold of detection to heat, electrical, and mechanical stimulation. Partial damage may be followed by either hyposensitivity or hypersensitivity (Eliav E, 2004). Some injuries may induce significant discomfort to patients, including liquid competence and untoward effects on speech, chewing, gestation, and swallowing (Susarla SM et al., 2007). In more than 60% of symptomatic patients, the onset occurs within 1 week of surgery, and most frequently involves the ipsilateral lip and chin (Lesley G, 1992).

Neurosensory dysfunction and pain can be present simultaneously. Positive symptomatology such as dysesthesia and negative symptomatology such as numbness may be present, particularly in cases with injury to a major nerve branch, such as the infraorbital or inferior alveolar branches (Susarla SM, 2007; Benoliel R & Eliav E, 2008). The neuropathic pain following tissue or nerve injury in the trigeminal nerve distribution is called traumatic trigeminal neuralgia, and is defined as a continuous pain following complete or partial damage to a peripheral nerve or central nervous system structure. The pain associated with traumatic neuralgia is of moderate to severe intensity and is usually continuous, burning numbness, and often pulling pain. Painful neuropathies often present with a clinical phenotype involving combinations of spontaneous and evoked pain. It can sometimes also possess paroxysmal qualities. Patients may complain of a feeling of swelling, presence of foreign body, hot or cold, local redness, or flushing. Local anesthetic blockade may not totally abolish the pain of traumatic neuralgia due to the possible presence of a sympathetic component (Benoliel R & Eliav E, 2008; Graff-Radford SB, 2009). Significant neurosensory dysfunction or painful nerve injuries such as traumatic trigeminal neuralgia may affect the patient's quality of life. The patient may sometimes require the skill of a clinical psychologist or psychiatrist with clinical experience in pain (Vickers ER & Cousins MJ, 2000).

Nerve injury during implant surgery is considered a rare, unintentional, and generally unsuspected, but serious complication. It is one of the most notable complications along with hemorrhage and infection (Burstein J et al., 2008). The recent, increase in the use of dental implants in the mandible may be associated with a greater risk of trauma to the IAN. Results from a prospective multicenter study of partially edentulous patients in which implants were placed to the mandible suggested that altered sensation is more prevalent than was previously reported (van Steenberghe et al., 1990), and there is considerable evidence that IAN injury is the most common injury experienced by patients after dental implant surgery (Delcanho RE, 1995).

The incidence of nerve injury after dental implant surgery has been found to be 3.7-39.1% at 1 week postoperatively in prospective studies, with a higher incidence in the posterior mandible (Anderson LC et al., 1991; Pogrel MA & Thamby S, 1999; Wismeijer et al., 1997; Zarf GA & Schmitt A, 1990). Ellis and Hawker reported on a retrospective analysis of a multicenter study. They found altered sensation of the IAN in 37% and 36% of patients respectively, 2 weeks after implant surgery. In both centers, these complaints persisted in 13% of patients (Ellies LG & Hawker PB, 1993). The incidence of permanent sensory dysfunction has been thought to be 1-8%, but the incidence of chronic pain is unclear (Benoliel R & Eliav E, 1990). In a recent domestic study, Ryu and Kwon found the incidence of dysesthesia after dental implant surgery to be 24.6%, and the incidence of permanent dysesthesia among the dysesthetic cases was 25.4%. Pain was the most closely related symptom, representing 44.8% of cases in implant surgery (Ryu JW & Kwon JS, 2007).

In the treatment of traumatic trigeminal neuropathy, the aim is reducing peripheral nociceptive inputs and simultaneously enhancing CNS pain inhibitory system (Scrivani SJ et al., 1999). Treatments are composed of surgical interventions and non-surgical treatments for the nerve injury. Non-surgical treatments are such as behavioral (counseling, yoga, stress management, psychotherapy), physiologic (exercise, sensory

reeducation, transcutaneous electric nerve stimulation), and pharmacologic treatment (Meyer RA & Ruggiero SL. 2001).

There have been some reports on the various factors that are related to the level of discomfort and recovery of neurosensory dysfunction. Merril suggested that nerve damage leads to a transient or persistent alteration in facial and oral sensation depending on the severity of the injury sustained (Merril RG, 1979). Hillerup suggested that the recovery of neurosensory function was related to the etiology of the injury (Hillerup S, 2008), while Kraut suggested that spontaneous recovery of normal sensation after nerve injury depends on both the severity of the injury and the nerve involved. Partial transection of the LN is less likely to result in spontaneous resolution of symptoms than is a similar injury involving the IAN (Kraut RA & Chahal O, 2002), however Tay and Zuniga reported there was no significant correlation between the severity of nerve injury and the time elapsed since injury (Tay ABG & Zuniga JR, 2007).

Based on these reports, in the present study I investigated the possible association between some of the factors thought to affect neurosensory dysfunction and recovery from it in patients with mandibular nerve injury after dental implant surgery.

II. PATIENTS AND METHODS

1. Patients

Data collection occurred from January 2007 to October 2010, and involved patients complaining of abnormal sensation and/or neuropathic pain after dental implant surgery, who were referred to Department of Oral Medicine and the Temporomandibular Joint and Orofacial Pain Clinic at the Dental Hospital of Yonsei University, Seoul, Korea.

Patients who had neuropathic pain or neurosensory disturbance caused by nerve injury to the mandibular branch of the trigeminal nerve after dental implant surgery and who had submitted to radiological evaluation using computed tomography (CT) were included (*N*=94).

Exclusion criteria were (1) trigeminal nerve injury caused by dental procedures other than dental implant surgery, (2) injury of the maxillary branch, and (3) patients who were seen only once.

This resulted in 5 individuals being excluded, and so the final sample comprised 89 patients with nerve injury: 61 females (68.5%) and 28 males (31.5%). The age of the cohort was 53.9±9.9 years (mean±SD, range 27-75 years).

2. Methods

Some factors, that had been expected to affect neurosensory dysfunction and symptomatic improvement after conservative treatment of patients with mandibular nerve injury after dental implant surgery were evaluated through medical record review. Those factors were pain intensity, level of invasion into the mandibular canal (as assessed by CT), retrieval of the affecting implant fixture to decompress the injured nerve, and time elapsed since nerve injury to the initiation of conservative treatment.

2.1 Neurosensory disturbance

At the initial visit, neurosensory changes were evaluated in patients using clinical neurosensory tests (pin-prick, light touch, 2-point discrimination, pressure-pain threshold, brush stroke direction test), and the current perception threshold (CPT) was measured using a Neurometer CPT/C (Neurotron, Baltimore, MD, U.S.A). The test results were compared between the injured and healthy sides.

Patients who had electric or burning pain were asked to grade the severity of their pain on a 10-point visual analogue scale (VAS), and the entire cohort was divided into four groups according to their VAS scores: (1) no pain (VAS score=0), (2) mild pain (0<VAS score <4), (3) moderate pain (4≤VAS score <7), and (4) severe pain (7≤VAS score ≤10)

2.2 Symptomatic improvement after conservative treatment

Patients, who received conservative treatment comprising behavioral, physical, and pharmacologic management, were divided into three groups depending on subjective self-reporting: (1) “good improvement” - neurosensory disturbance was satisfactorily improved and there were almost no affects on their activities of daily living, (2) “some improvement” – the improvement of neurosensory disturbance was not sufficient and there remained some affect on their activities of daily living, and (3) “no improvement”

2.3 Level of invasion into the mandibular canal, as assessed by CT

Patients submitted to CT to indirectly confirm invasion of the injured mandibular nerve, and were divided into three groups according to the relationship between the mandibular canal and the implant fixture: (1) “non-contact” – the implant fixture and mandibular canal were not in contact with each other, (2) “contact” – the implant fixture and mandibular canal were in close contact, but the cortical layer of the mandibular canal was preserved, and (3) “penetrated” – the mandibular canal wall was penetrated by the implant fixture, and discontinuity of cortical layer was observed

2.4 Retrieval of the affecting implant fixture to decompress the injured nerve

Patients were divided into two groups according to the manipulation of the affecting implant fixture to decompress the injured nerve: (1) the affecting implant fixture was not adjusted, and (2) the affecting implant fixture was partially unscrewed, changed to a shorter fixture, or removed to decompress the injured nerve

2.5 Time elapsed since nerve injury

Patients were divided into two groups according to the time from nerve injury to the initiation of conservative treatment involving behavioral, physical, and pharmacologic management: (1) within 6 months from nerve injury, and (2) more than 6 months after nerve injury

2.6 Relationship between pain intensity and other factors

The relationship between pain intensity and the following factors was analyzed: symptomatic improvement after conservative treatment, level of invasion into the mandibular canal, retrieval of the affecting implant fixture, and time elapsed since nerve injury.

2.7 Relationship between symptomatic improvement and other factors

Additional validation was performed on the relationship between symptomatic improvement after conservative treatment and the following factors: level of invasion into the mandibular canal, retrieval of the affecting implant fixture, and time elapsed since nerve injury.

3. Statistics

Statistical analyses were performed using the Wilcoxon signed-rank test, Kruskal-Wallis test, Mantel-Haenszel χ^2 test, and Mann-Whitney test. Statistical significance was established at 95%. SPSS version 17.0 (SPSS, Chicago, IL, USA) was used for all statistical analyses.

III. RESULTS

1. Subject demographics

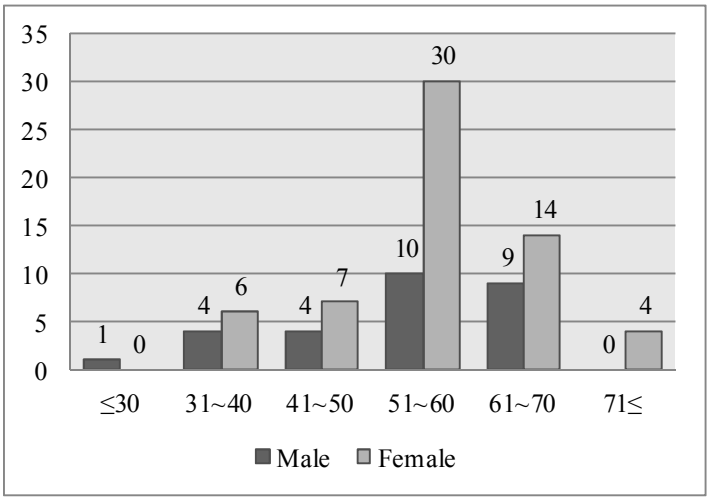

Fig. 1. Age and gender distribution of the patients (*N*=89)

Figure 1 illustrates the distribution of patients with mandibular nerve injury following dental implant surgery. The females in the sample were aged 54.7±9.4 years, and the males were aged 52.1±10.9 years.

2. Test results for neurosensory disturbance

Table 1. Results of the clinical neurosensory testing [a]

Test	Injured side (*N*=89)	Healthy side (*N*=89)	*p* *
Pin-prick (%)	65.0 (0.0-3000.0)	100.0	0.008*
Light touch (%)	60.0 (0.0-1000.0)	100.0	0.004*
Two-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*

a. Values are given as median (range)
*. Statistically significant at the 95% level

As presented in Table 1, the median values of both the pinprick (65.0, range=0.0–3000.0) and light-touch (60.0, range=0.0–1000.0) perception tests were significantly lower on the injured side than on the healthy side (pinprick: 100.0, p=0.008; light touch: 100.0, p=0.004). Furthermore, the median perceived count of brush-stroke direction was significantly lower on the injured side (29.0, range=0–30.0) than on the healthy side (30.0, range=4–30.0; p<0.001). However, the median distance for two-point discrimination was significantly higher on the injured side (13.4, range=0–55.0) than on the healthy side (10.2, range=0–72.4; p<0.001). The median force of the pressure–pain threshold did not differ significantly between the injured (160.0, range=0–300) and healthy (160.0, range=50–270) sides (p=0.785).

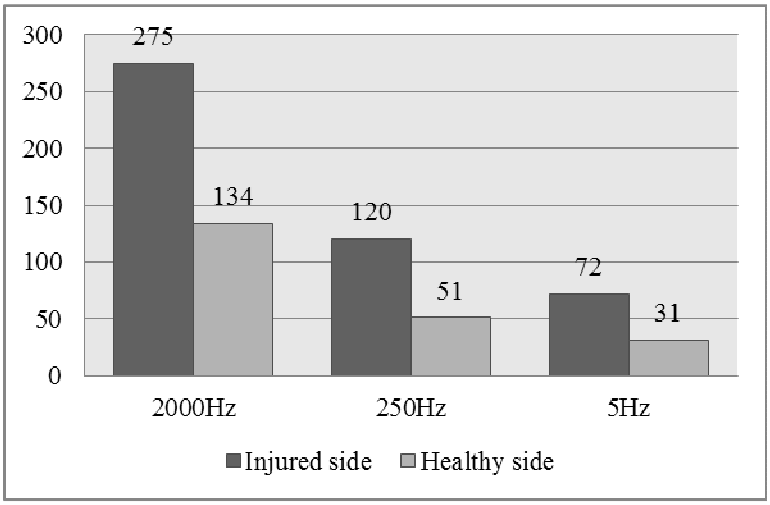

Figure 2. Result of the CPT test

Table 2. Results of the CPT test [a]

Frequency	Injured side (*N*=23)	Healthy side (*N*=23)	*p* *
2000Hz	275.0 (138.0-524.0)	134.0 (89.0-255.0)	<0.001*
250Hz	120.0 (41.0-292.0)	51.0 (10.0-188.0)	0.001*
5Hz	72.0 (7.0-240.0)	31.0 (8.0-115.0)	0.003*

a. Values are given as median (range)
*. Statistically significant at the 95% level

The median CPT values were statistically significantly higher on the injured side than on the healthy side at frequencies of 2000, 250, and 5 Hz (p<0.001, p=0.001, and p=0.003, respectively; Figure2 and Table 2).

3. Pain intensity and improvement by conservative treatment

Table 3. Analysis of pain intensity relative to symptomatic improvement[a]

		Improvement			Total (*N*)	*p**
		Good	Some	None		
Pain intensity	None	7 (41.2%)	5 (29.4%)	5 (29.4%)	17 (100.0%)	0.319
	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

a. Values are given as median (range)
*. Statistically significant at the 95% level

Table 3 presents the relationship between pain intensity and symptomatic improvement after conservative treatment. Of the entire cohort, 17 patients (19.1%) complained about neurosensory change alone, while 72 patients (80.9%) complained about both neurosensory change and pain. Good improvement after conservative treatment was experienced by 32 patients (36.0%), some improvement was experienced by 29 (32.6%), and no improvement was experienced by 28 (31.5%).

The median VAS score was 5.0 (range=0–9) in those who experienced a good improvement, 4.0 (range=0–9) in those with some improvement, and 6.0 (range=0–10) in those with no improvement; there were no significant differences between these groups (p=0.129). Furthermore, there was no significant linear relationship between pain intensity and symptomatic improvement (p=0.319).

4. Relationship between implant fixture and invasion of the mandibular canal, as assessed by CT

Table 4. Analysis of level of invasion into the mandibular canal, as assessed by CT, relative to pain intensity[a]

		Pain intensity				Total (*N*)	VAS
		None	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[*]						0.588	0.484

a. Values are given as median (range)
*. Statistically significant at the 95% level

Table 5. Analysis of level of invasion into the mandibular canal, as assessed by CT, relative to symptomatic improvement

		Improvement			Total (*N*)
		Good	Some	None	
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[*]					0.760

*. Statistically significant at the 95% level

As presented in Table 4, the noncontact, contact, and penetrated groups comprised 9 (10.1%), 14 (15.7%), and 66 (74.2%) patients, respectively. Their respective median VAS scores were 4.4 (range=0–9), 6.0 (range=2–9), and 5.0 (range=0–10); there were no significant differences between the groups (p=0.484). In addition, there was no significant linear relationship between the level of mandibular canal invasion and either pain intensity (p=0.588) or symptomatic improvement (p=0.760; Table 5).

5. Retrieval of the affecting implant fixture to decompress the injured nerve

Table 6. Analysis of retrieval of affecting implant relative to pain intensity[a]

		Pain intensity				Total (*N*)	VAS[a]
		None	Mild	Moderate	Severe		
Retrieval	No	7 (15.9%)	6 (13.6%)	17 (38.6%)	14 (31.8%)	44 (100.0%)	5.0 (0-10)
	Yes	10 (22.2%)	3 (6.7%)	16 (35.6%)	16 (35.6%)	45 (100.0%)	5.0 (0-9)
p[*]						0.934	0.156

a. Values are given as median (range)
*. Statistically significant at the 95% level

Table 7. Analysis of retrieval of affecting implant relative to symptomatic improvement

		Improvement			Total (*N*)
		Good	Some	None	
Retrieval	No	20 (45.5%)	12 (27.3%)	12 (27.3%)	44 (100.0%)
	Yes	12 (26.7%)	17 (37.8%)	16 (35.6%)	45 (100.0%)
p[*]					0.121

*. Statistically significant at the 95% level

The affecting implant was either partially unscrewed or removed to decompress the injured nerve in 44 patients (49.4%); the affecting implant was not adjusted in 45 (50.6%; Table 6). The median VAS score was 5.0 (range=0–10) in the unscrewed group and 5.0 (range=0–9) in the nonadjusted group. There was no significant difference between any of the groups (p=0.156). There was also no significant linear relationship between retrieval of the affecting implant fixture to decompress the injured nerve and either pain intensity (p=0.934) or symptomatic improvement (p=0.121; Table 7).

6. Effects of time elapsed since nerve injury to the initiation of conservative treatment

Table 8. Analysis of time elapsed since nerve injury relative to pain intensity [a]

		Pain intensity				Total (*N*)	VAS
		None	Mild	Moderate	Severe		
Time elapsed	≤ 6mo	14 (27.5%)	6 (11.8%)	19 (37.3%)	12 (23.5%)	51 (100.0%)	4.0 (0-9)
	> 6mo	3 (7.9%)	3 (7.9%)	14 (36.8%)	18 (47.4%)	38 (100.0%)	6.0 (0-10)
p [*]						0.004[*]	0.047[*]

a. Values are given as median (range)
*. Statistically significant at the 95% level

Table 9. Analysis of lapsed time from nerve injury to symptomatic improvement

		Improvement			Total (*N*)
		Good	Some	None	
Time elapsed	≤ 6mo	21 (41.2%)	20 (39.2%)	10 (19.6%)	51 (100.0%)
	> 6mo	11 (28.9%)	9 (23.7%)	18 (47.4%)	38 (100.0%)
p [*]		0.024[*]			

*. Statistically significant at the 95% level

As presented in Table 8, the median VAS scores were significantly lower for the 51 patients (57.3%) who obtained conservative treatment within 6 months from nerve injury (4.0, range=0–9) than for the 38 (42.7%) who did not receive conservative treatment until more than 6 months postinjury (6.0, range=0–10; p=0.047). There was a statistically significant linear relationship between time elapsed since nerve damage and both pain intensity (p=0.004) and symptomatic improvement after conservative treatment (p=0.024; Table 9).

Ⅳ. DISCUSSION

There are a number of mechanisms described as causing trauma induced neuropathy. They can be described as: (a) peripheral sensitization, (b) ectopic activity due to sodium channel expression, (c) central sensitization, (d) Aβ fiber reorganization, (e) alteration in central inhibition systems, and (f) sympathetically maintained pain due to α-receptor sprouting. More than 2 mechanisms may be active to create individual clinical presentations (Woolf CJ & Mannion RJ. 1999). As a result, following 4 clinical symptom subtypes were identified: (1) anesthesia dolorosa: pain or dysesthesia felt in an area of numbness, (2) Sympathetically maintained pain (SMP): pain aggravated by increased sympathetic tone, cold, and emotional stimuli and reduced by α-adrenergic or sympathetic nerve blockade. Edema, erythema, hyperkeratosis, atrophy, ulcerations, and hypohidrosis could be associateds signs, (3) allodynia: quick response pain to low-intensity, normally nonnoxious stimuli, and (4) Hyperpathia: delayed pain response to moderate mechanical stimuli. The elicited pain is of increased intensity and displays “overshooting” and “after image qualities (Gregg JM. 1990).

In the present study, females outnumbered males at more than two to one. There are similar reports suggestive of a higher tendency toward neurosensory dysfunction after dental implant surgery among females. It may be that females are more at risk because of their prevalent hormones, such estrogen, their lower pain threshold relative to males, or their greater tendency to see doctors in general. However, we should remember that patients referred to a tertiary care center do not represent the true incidence of nerve injury from various causes (Pogrel MA & Thamby S, 1999; Tay ABG & Zuniga JR, 2007; Hillerup S, 2007).

Kraut and Chahal suggested that when a nerve injury is suspected, a thorough neurosensory examination (light touch, pain perception, direction of brush movement, two-point

discrimination, temperature sensitivity) should be performed, and that any unusual patient reactions (such as sharp pain or an electrical shock-like sensation) that occur during surgery should be documented (Kraut RA & Chahal O, 2002). In the present analysis, all clinical neurosensory tests except the pressure-pain threshold test revealed significant differences between the injured and healthy sides except the pressure-pain threshold test revealed significant differences between the injured and healthy sides. However, these types of test have some disadvantages, such as examiner subjectivity and technical discord between examiners. It is thus necessary to develop a more standardized and objective examination for neurosensory disturbances.

A CPT test may be performed to assess damages to peripheral nerves in the trigeminal nerve area. The thicknesses of peripheral nerve fibers vary and they are selectively responsive to electrical stimulation at various frequencies. Thus, CPT testing allows the measurement of changes (increases and decreases) in the CPT of Aβ, Aδ and C fibers, since they respond specifically to stimulation at frequencies of 2000, 250, and 5Hz, respectively. Therefore, sensory disturbances such as hyperesthesia or hypoesthesia can be diagnosed (Masson EA & Boulton AJ, 1989; Masson EA et al., 1991).

When starting this analysis, it was expected that pain intensity would increase and symptomatic improvement would decrease with increasing levels of invasion into the mandibular canal. However, no significant relationship between the level of invasion and either pain intensity or symptomatic improvement was found.

A review of the CT images showed that most of the mandibular nerve injuries after dental implant were caused by direct contact between the implant fixture and the mandibular canal. These nerve injuries occur directly as a result of drilling procedures, lack of attention to diagnostic information, and/or direct compression of the nerve during implant insertion (Lamas-Pelayo J et al., 2008; Kraut RA & Chahal O, 2002; Anderson LC et al., 1991). Variables such as magnification errors, variable ridge anatomy (such as presence of an anterior

loop of the MN) and other anatomical variations, and operator technique can increase the probability of complications. More than one IAN and canal may exist (Carter RB & Keen EN, 1971), and a plexus of nerve branches may lie superior to and outside the mandibular canal. Infrequently, the preparation drill is 1 mm longer than the implant. Allowance for these variations must be incorporated into the calculation of the bone height available for the implant (Anderson LC et al. 1991).

Interestingly, in 10.1% (nine cases) of patients there was no direct contact between the affecting implant fixture and the mandibular canal. The distances between the fixture and canal wall ranged from 1 to 3mm. Indirect loading through trabecular bone compression or bone condensation from the implant placement might be a possible cause (Rodríguez-Lozano FJ et al., 2010) and heat by drilling could be an another possible cause of indirect mandibular nerve injury. Hirsch and Branemark suggested following causes of nerve injury linked to implant surgery: (1) direct mechanical damage, (2) pressure on nerve and blood vessel, and (3) formation of hemangioma or osteoma followed by vascular damage (Hirsch JM & Branemark PI, 1995). On the other hand, in some cases of LN and MN injury, the nerve may be traumatized by incision, flap elevation, and retraction or during suturing (Kraut RA & Chahal O, 2002; Miloro M et al. 1997). Clinicians should therefore not ignore the possibility of indirect nerve damage when placing a dental implant.

There are several ways of dealing with nerve injury once it has occurred. For example, early decompression of the injured nerve through partial unscrewing, changing to a shorter fixture, or removal of the affecting implant immediately or within days, is one of the most widely known procedures. Gregg suggested that when nerve injury occurs, the clinician should clear the site of debris and ensure that the nerve is decompressed away from the surrounding bone, and should perform a meticulous layered tissue closure. (Gregg JM. 1990). However, it is unclear whether patients with nerve injuries would benefit from early surgical decompression or repair compared to nonsurgical management (Gregg GM &

Zuniga JR, 2001). Kwon et al. reported that almost half of all practitioners had attempted surgical intervention to the implants, such as removing the fixture, partial unscrewing, or reimplantation of a shorter fixture, and that this was regarded as being effective in 53% of cases (Kwon TG et al. 2004).

The present study found no significant difference between the retrieval and non-adjustment groups. However, symptomatic improvement was reported by some patients after early decompression of the injured nerve by retrieval of the affecting implant. This suggests that immediate retrieval of the affecting implant is better than doing nothing, although it should be avoided if osseointegration is already in progress since secondary injury from the surgical procedure may exacerbate and further sensitize an already hyperesthetic nervous system (Ziccardi VB & Steinberg MJ, 2007; Evans GR, 2001).

Tay and Zuniga reported no significant correlation between the severity of the nerve injury and the time elapsed since injury (Tay ABG & Zuniga JR, 2007). However, Ryu and Kwon reported that the recovery ratio of injured nerves after implant surgery was 71.8%, and that recovery had been achieved within 6 months since injury in 88.2% of the recovered group (Ryu JW & Kwon JS, 2007).

In the present analysis, there was a significant linear relationship between time elapsed since injury and both pain intensity and symptomatic improvement. Another encouraging finding was that 26.3% of patients with a 1-year-old nerve injury showed satisfactory symptomatic improvement after conservative treatment. Thus, the commencement of conservative treatment is not necessarily hopeless for patients who have had a nerve injury for more than 1 year. In the present analysis, 36% of patients experienced satisfactory symptomatic improvement after conservative treatment.

The present analysis was subject to some limitations. Measurements of pain intensity and improvement of neurosensory symptoms were based on self-reported and subjective information. Furthermore, the cohort in this analysis was small, which resulted in difficulties

controlling external factors such as age and gender distributions. Future studies should involve regular clinical neurosensory and CPT testing for a more objective evaluation of neurosensory recovery.

V. CONCLUSION

To investigate the possible association between neurosensory discomfort, Symptomatic improvement after conservative treatment, and following factors (level of invasion into the mandibular canal assessed by CT, retrieval of the affecting implant to decompress the injured nerve, and time elapsed since nerve injury to initiation), this analysis was performed in 89 patients with mandibular nerve injury after dental implant surgery from January 2007 to October 2010.

The results are summarized as follows.

1. There was no significant linear relationship between pain intensity and symptomatic improvement (p=0.319).
2. There was no significant linear relationship between the level of mandibular canal invasion and either pain intensity (p=0.588) or symptomatic improvement (p=0.760).
3. There was also no significant linear relationship between retrieval of the affecting implant fixture to decompress the injured nerve and either pain intensity (p=0.934) or symptomatic improvement (p=0.121).
4. There was a statistically significant linear relationship between time elapsed since nerve damage and both pain intensity (p=0.004) and symptomatic improvement after conservative treatment (p=0.024).

Direct nerve injury is the most important factor in the initiation of neurosensory disturbances, but the present findings demonstrate that time elapsed since nerve injury is most closely related to pain intensity and symptomatic improvement than to other factors,

including the level of mandibular canal invasion. Therefore, if a nerve injury is suspected during implant surgery, early initiation of conservative treatment followed by neurosensory evaluation should be performed to reduce patient discomfort and improve the probability of nerve recovery.

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ABSTRACT (IN KOREAN)

임플란트 수술 후 발생한

감각신경 이상에 대한 분석

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최 영 찬

삼차신경 손상 환자들의 불편감 정도나 감각 이상의 회복에 영향을 미치는 다양한 요인들에 대한 관계가 여러 연구들에서 보고되어 왔다. 그러나 이들 간의 연관성은 아직은 명확히 설명되고 있지 않다. 이에, 본 분석에서는 임플란트 수술 후 발생한 하악신경손상 환자들에서 감각이상과 그 회복에 영향을 미칠 것으로 예상되는 몇가지 요인들의 관계에 대한 확인을 시행하였다.

2007 년 1 월부터 2010 년 10 월까지 총 89 명의 하악신경손상 환자들이 이 연구에 포함되었다. 전산화단층촬영 영상에서 확인한 임플란트와 하악관의 관계, 손상된 신경을 감압하기 위한 임플란트의 조절 여부, 손상시점으로부터 보존적인 치료가 시작되기까지 경과한 기간이 불편감의 정도나 감각이상의 회복과 연관성을 갖는지 의무기록 분석을 통하여 평가하였다. 분석 결과를 요약하면 다음과 같다.

1. 통증 강도와 증상 개선 정도 사이에 통계학적으로 유의한 연관성은 없었다 (p=0.319).
2. 전산화단층촬영 영상에서 하악신경관 침범 정도와 통증 강도 사이에는 통계학적으로 유의한 연관성이 보이지 않았으며 (p=0.588), 증상의 개선 정도에서도 유의한 연관성을 보이지 않았다 (p=0.760).
3. 임플란트의 조절을 통한 손상 신경의 감압 여부와 통증 강도 사이에는 통계학적으로 유의한 연관성이 보이지 않았으며 (p=0.934), 증상의 개선 정도에서도 유의한 연관성을 보이지 않았다 (p=0.121).
4. 신경손상 발생부터 약물치료 등의 보존적 치료가 시작되기까지 경과한 시간과 통증 강도 사이에는 통계학적으로 유의한 선형적 연관성이 있었으며 (p=0.004), 증상의 개선 정도에서도 유의한 선형적 연관성을 볼 수 있었다 (p=0.024).

상기 분석 결과에 의하면, 신경손상 발생 후 보존적인 치료를 시작하기까지 경과한 시간이, 하악신경관 침범 수준 같은 다른 요인에 비하여 통증 강도와 증상의 개선에 더 많이 연관되어 있으며 시간이 많이 지체될수록 통증 강도는 증가하고 증상의 개선은 작아짐을 예상 할 수 있다. 감각 이상의 발생에서 신경의 직접적인 손상은 가장 중요한 요인이지만, 임플란트 수술 과정에서 신경 손상이 의심된다면 불편감의 감소와 더 나은 회복을 위하여 조기에 보존적인 치료가 시작되어야 할 것이다.

핵심되는 말: 신경손상, 임플란트, 하악신경, 감각이상