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Inferior alveolar nerve injury associated with implant surgery

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Abstract

Objectives: Inferior alveolar nerve (IAN) is the most commonly injured nerve (64.4%) during implant treatment. At present, no standardized protocol exists for clinicians to manage IAN injury related with implant surgery. Therefore, the purposes of the present article were to analyze the reasons for nerve injury and to propose guidelines in managing IAN injury.

Material and methods: Patients with IAN sensory disturbances after implant treatment were recruited for the study. Sixteen patients, eight men and eight women, with a mean age of 52.2 ± 8.1 years participated in this study. Patient examination, treatment, and IAN sensory function recovery monitoring were performed following six-step IAN injury during dental implant surgery (IANIDIS) protocol. The control group was composed of 25 healthy volunteers who never had IAN sensory disturbances or any trauma in the maxillofacial region.

Results: The IAN sensory disturbances were scored as following: 5 (31.25%) had hyperalgesia and 11 (68.75%) expressed hypoalgesia. The mean asymmetry index (AI) was calculated for each patient and varied from 0.6 to 3.2. Overall, 31.3% of nerve injury patients were classified as mild, 31.3% as moderate, and remaining 37.5% as severe injury. All patients were successfully treated with proposed IANIDIS protocol.

Conclusion: The most frequent (50%) risk factor for IAN injury was intraoperative bleeding during bone preparation. The most common (56.3%) etiological risk factor of nerve injury was dental implant. A six-step protocol aimed at managing patients with IAN injury, during dental implant surgery, was a useful tool that could provide successful treatment outcome.

In 1995, Worthington wrote: "The number of practitioners performing implant surgery has increased dramatically over the last 15 years. As confidence is gained they tend to accept increasingly challenging cases and it is to be expected that the incidence of problems and complications will increase" (Worthington 1995). It was a discerning remark; inferior alveolar nerve (IAN) injuries remain a serious complication with incidence ranged from 0% to 40% (Delcanho 1995; Rubenstein & Taylor 1997; Wismeijer et al. 1997; Dao & Mellor 1998; Bartling et al. 1999; Walton 2000; Ziccardi & Assael 2001; von Arx et al. 2005; Abarca et al. 2006; Greenstein & Tarnow 2006; Hegedus & Diecidue 2006; Tay & Zuniga 2007; Misch 2008; Alhassani & AlGhamdi 2010; Misch & Resnik 2010).

The IAN supplies the mandibular molar and premolar teeth and adjacent parts of the gingival. Its larger terminal branch emerges from the mental foramen as the mental nerve. Three nerve branches come out of the mental foramen. One innervates the skin of the mental area, and the other two proceed to the skin of the lower lip, mucous membranes, and the gingiva as far posteriorly as the second premolar. The incisive branch, a continuation of the IAN, supplies the canine and incisor teeth (Ziccardi & Assael 2001; Abarca et al. 2006).

It is interesting to know that the IAN is the most commonly injured nerve (64.4%), followed by the lingual nerve (28.8%) (Tay & Zuniga 2007). The differences between IAN injuries and other peripheral sensory nerve injuries are predominantly iatrogenic and not resolved within the first 8 weeks after injury.

Inferior alveolar nerve injury can result from traumatic local anesthetic injections, during dental implant site preparation or placement (Hegedus & Diecidue 2006), or

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poor surgical technique (Ellies & Hawker 1993; Bartling et al. 1999; Gregg 2000; Cranin 2002; von Arx et al. 2005; Smith & Lung 2006). This injury is one of the most unpleasant experiences, from mild paresthesia to complete anesthesia and/or pain (Alhassani & AlGhamdi 2010). As a result, many important functions such as speech, eating, kissing, make-up application, shaving and drinking were affected (Ziccardi & Assael 2001). This influences patient's quality of life and often resulted in negative psychologic adverse effects (Abarca et al. 2006). This injury has also created a lot of disharmony between dentists and patients that dearly cared. Clinicians should recognize related risk factors and identify etiological factors that may lead to nerve injury and do their best in avoiding these injuries. Proper pre-surgery treatment planning, timely diagnosis and treatment, when suspect nerve injuries, are the key to avoid nerve sensory disturbances (Juodzbalys et al. 2011). At present, no standardized protocol exists for clinicians to manage IAN injury related with implant surgery. Therefore, the purposes of the present article were to analyze the reasons for nerve injury and to propose guidelines to manage IAN injury.

Material and methods

Subject sample

Patients admitted to the Department of Maxillofacial Surgery, Lithuanian University of Health Sciences, Kaunas, Lithuania, between May 2007 and December 2010, with IAN injury during the dental implant placement were recruited to the study. All participants have read and signed informed consent form. The use of human subjects in this study has been reviewed and approved by the Health Science Institutional Review Board of the Lithuanian University of Health Sciences, Kaunas, Lithuania. The subjects enrolled in the study had to fulfill the following inclusion criteria:

- They have had unilateral iatrogenic IAN injury with neurosensory disturbance after dental implant surgery.
- Patients had no IAN sensory disturbances in the surgery sites prior to the treatment.
- Patients could be available for 6 clinical examinations and treatment visits within 3 months after IAN injury.

All patients were examined within 10–52 h following the injury except one patient who was seen at 336 h after surgery (however, patient was seen within 52 h of noticing sensory alteration). Patient examination, treatment, and IAN sensory function recovery monitoring were performed following six-step IAN injury during dental implant surgery (IANIDIS) protocol. The surgeon who performed implant surgery provided all necessary background information such as general and intraoperative risk factors. The control group composed of 25 healthy volunteers who never had IAN sensory disturbances or any trauma in the maxillofacial region.

IANIDIS protocol

Step 1. Confirmation of injury

The IAN sensory disturbances in the affected IAN distribution were diagnosed based upon patients' complains of altered sensation and clinical symptoms. If patient reports altered sensation, typical patient complains can be as follows: "numbness, tingling, itching or, pain evoked by touching the skin in the region of the mental and lower lip area of the affected side."

Step 2. Related risk factors identification

Possible risk factors were identified in all cases. They were classified as general, intraoperative and post-operative risk factors. General risk factors are related to patient's realistic expectations and obtaining the informed consent form including signature. Neurosensory examination of IAN function prior to implant therapy is essential to rule out any predisposing problems. Intraoperative risk factors include pain ("sudden give" or an "electric shock") induced at the time of local anesthesia injection or bone preparation, drill slippage as well as change in pre-planned implant dimension (diameter and length). Post-operative risk factors are often related to post-surgery infection, induced hematoma or pressure that compresses the nerve.

Step 3. Etiological factors identification

Table 1 shows possible etiological factors of IAN injury during implant placement. Etiological factors of IAN injury can be classified, based upon time of incident, as intraoperative and post-operative (Juodzbalys et al. 2011). Intraoperative etiological factors include mechanical, thermal, and chemical. Post-operative etiological factors consist of peri-implant infection and hematoma with subsequent scaring and ischemia.

Mechanical traumatic factors such as injection needle, implant drill, implants itself or bone debris (foreign body), scalpel, soft tissue retraction instruments may evoke direct mechanical injury, i.e. pressure, encroach, transection, or laceration of the nerve. Indirect nerve injuries are often due to hematoma, compression, and secondary ischemia.

Step 4. Diagnosis of nerve disturbances

Nerve injury clinical symptoms include hyperalgesia or hypoalgesia of the skin in areas of nerve innervation. Hyperalgesia or hypoalgesia was diagnosed by comparing pain detection threshold (PDT) at the skin of innervation zone of the healthy and affected sides. The PDT assessment was performed applying non-invasive electrocutaneous stimulation of the dry skin in the region of the mental foramina by active 4 mm diameter steel electrode and passive hook from electrode fixed on the same side of the patients' ear.

The electrical stimulation unit Pulptester Pt1 (Lumen, Kaunas, Lithuania) was used for the PDT testing. This unit generates monopolar constant-current rectangular impulses of negative polarity. Stimulus frequency was 6 Hz. The PDT was assessed by an ascending method of limits. Stimulating current was increased at a fixed rate until the subject indicated first pain sensation. For the subjects of control group, results of three PDTs measurements were obtained and mean value calculated. Three PDTs for the injury patients were also evaluated at both healthy and affected sides. The assessments were performed before treatment and during follow-up by one calibrated examiner (G. J.) (weighted Cohen's k was 0.91 for PDT measurements; Polson 1997).

Asymmetry index (AI) was used to assess extent of sensory alteration (nerve injury). AI was calculated using a ratio of electric PDT measurements at the left and right sides for control group and healthy and affected patients with nerve injury. For the healthy persons, the ratio does not differ significantly ($P > 0.05$) from a value of 1; lower ratios of AI score (<1) indicate hyperalgesia whereas higher ratios (AI $\geq$ 1) suggest hypoalgesia.

The IAN injury severity degree was assessed using AI score (Sakavicius et al. 2008). In brief, IAN injury with diagnosed hyperalgesia and AI <1 was classified as mild. IAN injury with hypoalgesia and AI ranging between 1 and 2 was classified as moderate. IAN injury with hypoalgesia and AI >2 was classified as severe.

Following the neurosensory assessment, X-ray examination (CT or cone beam CT) was performed. The spiral CT scans were all derived from a Somatom plus SA CT scanner (Siemens, Erlangen, Germany) following a

Table 1. **Etiological factors and mechanism of traumatic inferior alveolar nerve (IAN) injury**

Intraoperative etiological factor	Indirect or direct and injury mechanism	Post-operative etiological factor	Indirect and injury mechanism
Traumatic local anesthesia			
Chemical (cytotoxic) injury by local anesthetic	Indirect; endoneurial edema, compression and secondary ischemia Direct; IAN degeneration	Injection needle trauma to epineurial blood vessels or inferior alveolar artery	Indirect; hematoma with reactive fibrosis and scar formation, compression and secondary ischemia
Injection needle	Direct; transection of multiple IAN fibers and entire fascicles		
Implant drill			
Partial intrusion into MC	Indirect; hematoma and secondary ischemia	Thermal injury	Indirect; inflammation of bone and IAN with secondary ischemia
Full intrusion into MC	Direct; mechanical trauma – encroach, transection, or laceration and/or compression and primary ischemia of IAN		
Chemical (cytotoxic) injury	Direct; IAN degeneration		
Thermal injury	Direct; IAN degeneration		
Dental implant			
Partial intrusion into MC	Indirect; hematoma or/and deposition of debris, compression and secondary ischemia	Infection	Indirect; inflammation of bone and IAN with secondary ischemia
		Implant is too close to MC	Indirect; bone and IAN stress, compression with secondary ischemia
Full intrusion into MC	Direct; mechanical trauma – encroach, transection, or laceration and/or compression and primary ischemia of IAN	Chronic stimulation	Indirect; implant is situated aside of or on top of the nerve with chronic neuropathy formation
Wrong operation technique			
Scalpel	Direct; mental nerve injury or transection	Soft tissue swelling	Indirect; mental nerve compression caused by soft tissue edema
Soft tissue reflection and retraction	Direct; mental nerve injury caused by reflection, retraction and pressure		
Soft tissue suturing	Direct; mental nerve compression caused by suture material		

MC, mandibular canal.

standard exposure protocol that was developed by Department of Radiology of the University Hospital of the Lithuanian University of Health Sciences. Axial CT scans with 1 mm sections (without overlap) were obtained. Reformatted cross-sectional images, 2 mm apart, were obtained using the Dental CTA software (Somatom plus SA; Siemens, Erlangen, Germany). A cone beam CT scan was performed using the cone beam CT unit Kodak 9000 3D (Carestream Health, Rochester, NY, USA) and Kodak Dental Imaging software (Carestream Health, Rochester, NY, USA). Radiographic examination was essential to pin-point the lesion location as well as confirmation of INA injury. Dental implant position to mandibular canal was graded as too close (<1 mm) but no contact, contact without intrusion into canal, and partial or full intrusion into mandibular canal. In the case when implant is not in contact with canal, but osteotomy is reaching canal it was graded as IAN injury by implant drill.

Step 5. Treatment inferior alveolar nerve injury

All patients with IAN injury went through psychologic consultation which includes background information, detail explanation, support, and realistic expectations from the injury treatment. After consultation, a physiologic treatment was provided that includes: removal of the implant, within 36 h post-surgery that may be in any contact or causing pressure to the mandibular canal. Subsequently, any irritants (bone debris, hematoma) in close approximation was removed to allow faster dispersion of the hemorrhage/debris. If during surgery, known or observed trauma (including traction or compression of the nerve trunk) has occurred, the topical application of intravenous form steroids, one to two milliliters of dexamethasone (4 mg/ml), was applied for 1 –2 min.

Medicament treatment depends on degree of severity of the nerve injury. In case of mild degree of nerve injury, a large dose of non-steroidal anti-inflammatory drug (such as 400 –600 mg ibuprofen) three times daily for 1 week was prescribed. In case of moderate or severe nerve injury, a course of oral steroids was prescribed. Oral dexamethasone 4 mg, two tablets AM for 3 days and one tablet AM for next 3 days or oral prednisolone 1 mg per kg per day (maximum 80 mg) might be prescribed. As an alternative or adjunct would be a large dose of non-steroidal anti-inflammatory drug (such as 800 mg ibuprofen) three times daily for 3 weeks was also given. Prescription of these drugs was undertaken with consideration to the patient's medical history and caution. In all cases, additionally diuretics (torasemidum, 10 mg per day, for 5 days), vasodilators (pentoxifylline, 1200 mg per day for 10 days), and B-group vitamins (neurorubine forte lactab once per day for 2 weeks) and antihistaminic drugs (loratadinum 10 mg per day) were prescribed. If the situation improves, course of nerve recovery drugs were repeated during 3 months period (B-group vitamins, vasodilators). In some complicated cases additional pharmacologic agents were used. They include antidepressants, anticonvulsants, antisympathetic agents, and topical medications. Additional physiologic therapies, such as transcutaneous electric nerve stimulation, acupuncture, and low level laser therapy, can be indicated and prescribed by a nerve specialist.

Step 6. Monitoring inferior alveolar nerve recovery

Follow-up was undertaken and IAN function recovery monitoring was performed after 7, 14 and 21 days, 1, 2 and 3 months. Patient should always feel psychologic support.

Statistical analysis

Statistical analysis was performed using a statistical program. Mean values and standard deviations were calculated (SPSS/PC + statistical program version 13.0 for Windows; SPSS Inc., Chicago, IL, USA). Descriptive

statistics were used for the ratings of single characteristics. Weighted Cohen's k was used to calculate intra-examiner reliability. Mann–Whitney test was used for comparing two independent groups of observations. Parametric paired t-test and non-parametric Wilcoxon test was used for related samples quantitative comparison, when measurements were taken from the same subject before and after manipulation to determine significance levels. A non-parametric Kruskal–Wallis test (one-way ANOVA by ranks) was used for testing equality of population medians among groups. Non-parametric Chi-squared test (P-value exactly) was used to compare qualitative data. A significant level of $P < 0.05$ was used. Data were expressed as mean values and standard deviation.

Results

Demographic information

The control group consisted of 25 persons (12 men and 13 women), with a mean age of 36.9 ± 6.8 (min 25, max 52) years. Data for subjects and IAN injury are shown in Table 2.

A total of 16 patients with IAN sensory disturbances, eight men and eight women, with a mean age of 52.2 ± 8.1 (min 36, max 65) years were enrolled in this study. The IAN injury occurred in left and right first and second mandibular premolars and molars dental segments. Although the size of the sample is small and this could be a limitation of the study, to minimize differences between control and test samples, we tried to match some variables in both group as much as possible, as gender. Anyway there were no significant differences between patients' age ($P = 0.4$; Mann–Whitney test) and gender ($P = 0.4$; Chi-squared test). The duration of post-injury was from 13 (subject 9) to 336 (subject 10) h (77.5 ± 109.9 for men and 29.5 ± 18.1 h for women, $P = 0.4$). In eight (50%) cases, it was associated with bleeding during operation, pain during drilling, drill slippage, and changed pre-planned implant size. In six cases (37.5%) possible risk factor was not identified. The implant was the most common etiological factor of nerve injury. It was registered in nine cases (56.3%) including six cases of partial intrusion into mandibular canal. Implant drill was the second most common etiological factor that counts in four (25%) cases.

Diagnosis of IAN sensory disturbances

Measurements of the PDT for the control group revealed the following results: left mental foramina projection 36.8 ± 6.82 μA, right 37.01 ± 6.8 μA. The PDT did not differ significantly between both sides' examination results. Mean and 95% confidence interval of the paired differences were 0.21 [−.48, 0.05]. The mean AI was 1 ± 0.15 (min 0.95, max 1.02, range 0.07).

Results of IAN PDT assessments, mean AI calculated following iatrogenic nerve injury, and injury severity classification are shown in Table 3. IAN sensory disturbances in the affected nerve distribution were registered for all 16 patients. In five (31.25%) of the cases (12.5% men and 50% women, $P = 0.3$ [Chi-squared test]) there was hyperalgesia and in 11 (68.75%) of cases hypoalgesia. The mean AI was calculated for each patient and varied from 0.6 to 3.2. Analysis of IAN injury severity revealed that 31.3% of patients were classified as mild, 31.3% as moderate, and 37.5% as severe. The gender has no influence on injury severity degree ($\chi^2 = 2.7$; df = 2; $P = 0.3$). The duration after trauma has more influence, but not significant and ranged for mild nerve injury 23.8 ± 15.3, moderate 49.8 ± 41.5 and severe 81.3 ± 125.8 h ($\chi^2 = 2.3$; df = 2; $P = 0.3$). The age has significant influence on IAN injury severity degree and was registered for patients with mild nerve injury of 43.8 ± 4.7, moderate 53.2 ± 7.3 and severe 58.3 ± 4.5 years ($\chi^2 = 8.7$; df = 2; $P = 0.02$) by Kruskal–Wallis test.

IAN injury treatment results

Clinical and radiographic patients' examination revealed that in 13 (81.3%) cases etiological factor was implant drill (Fig. 1) or implant itself (Fig. 2). When implant was in any contact with or causing pressure to the mandibular canal or it was diagnosed moderate or severe degree of IAN injury (hypoalgesia), implant was removed. All 13 sites were cleaned gently with curette by removing all irritants such as bone debris, hematoma and sharp bone edges. One ml of the intravenous form of dexamethasone (4 mg/ml) was then topically applied for 1–2 min. After these, sites were left open to heal under blood clot. Medicament treatment, based upon severity of nerve injury, was prescribed following the protocol mentioned before.

In subjects 4, 7 and 14, implants were left to osseointegrate because there was no identified possible implant drill or implant contact with mandibular canal. In addition, only a mild degree of nerve injury was reported in these three cases. In subject 4, sutures were placed too deep and mental nerve compression was suspected. Two sutures were removed and placed appropriately. In subject 7, the etiological factor was not identified, and in subject 14 injury of IAN was probably

Table 2. **Data for subjects and inferior alveolar nerve injury**

Subject no.	Gender	Age (years)	Affected JDS no.	Duration post-injury	Intraoperative risk factor	Etiological factor
1	Female	44	30	18 h	Change pre-planned implant size (wider)	Implant drill
2	Female	56	18	52 h	Not identified	Implant (partial intrusion)
3	Male	60	19	26 h	Pain during bone preparation, bleeding	Implant (partial intrusion)
4	Female	48	19	50 h	Not identified	Wrong operation technique (suturing)
5	Male	52	30	28 h	Bleeding	Implant drill
6	Male	47	31	36 h	Drill slippage, bleeding	Implant (partial intrusion)
7	Male	36	29	14 h	Not identified	Not identified
8	Male	61	20	46 h	Bleeding	Implant (full intrusion)
9	Female	47	21	13 h	Change pre-planned implant size (longer), bleeding	Implant drill
10	Male	55	19	2 weeks	Not identified	Implant, infection
11	Female	58	31	51 h	Drill slippage, bleeding	Implant drill
12	Male	63	18	5 days	Not identified	Implant (too close)
13	Female	65	19	14 h	Pain during bone preparation, bleeding	Implant (partial intrusion)
14	Female	44	20	24 h	Pain during local anesthesia	Injection needle
15	Female	46	30	14 h	Not identified	Implant (partial intrusion)
16	Male	53	28	14 h	Change pre-planned implant size (longer), bleeding	Implant (partial intrusion)

JDS, jaw dental segment.

Table 3. **Results of inferior alveolar nerve (IAN) pain detection threshold (PDT) assessments, asymmetry index (AI) calculated following iatrogenic IAN injury, and injury severity classification**

Subject no.	JDS no.	PDT (mean ± SD)		AI (Mean ± SD)	Type of IAN sensory disturbance	IAN injury severity degree
		Intact side	Affected side			
1	30	46.7 ± 1.53	33.7 ± 1.53	0.7 ± 0.01	Hyperalgesia	Mild
2	18	48.3 ± 1.53	116.7 ± 4.16	2.4 ± 0.15	Hypoalgesia	Severe
3	19	38.7 ± 1.15	95.3 ± 5.03	2.5 ± 0.2	Hypoalgesia	Severe
4	19	33.3 ± 2.08	26 ± 2	0.8 ± 0.1	Hyperalgesia	Mild
5	30	48.7 ± 2.08	67.3 ± 2.52	1.4 ± 0.01	Hypoalgesia	Moderate
6	31	35 ± 3	61 ± 2.65	1.7 ± 0.08	Hypoalgesia	Moderate
7	29	53 ± 2	33.3 ± 3.06	0.6 ± 0.04	Hyperalgesia	Mild
8	20	38.3 ± 2.52	122.3 ± 2.52	3.2 ± 0.27	Hypoalgesia	Severe
9	21	36.3 ± 1.53	23 ± 2	0.6 ± 0.05	Hyperalgesia	Mild
10	19	50.3 ± 3.51	144.3 ± 4.04	2.9 ± 0.28	Hypoalgesia	Severe
11	31	37.7 ± 2.08	66 ± 1.73	1.8 ± 0.05	Hypoalgesia	Moderate
12	18	32 ± 2	64.3 ± 2.52	2 ± 0.12	Hypoalgesia	Moderate
13	19	46.3 ± 1.15	148 ± 3	3.2 ± 0.1	Hypoalgesia	Severe
14	20	62.3 ± 3.06	45 ± 1	0.7 ± 0.43	Hyperalgesia	Mild
15	30	64.3 ± 2.52	97.3 ± 2.08	1.5 ± 0.09	Hypoalgesia	Moderate
16	28	36.7 ± 3.21	106.7 ± 3.06	2.9 ± 0.35	Hypoalgesia	Severe

JDS, jaw dental segment.

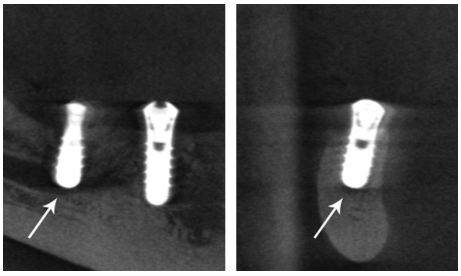

Fig. 1. Cone-beam computed tomography scans shows full dental implant intrusion into mandibular canal in 35 jaw dental segment region (subject 8). There is direct mechanical trauma – inferior alveolar nerve transection.

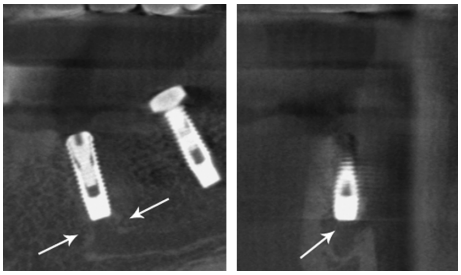

Fig. 2. Cone-beam computed tomography scans shows that implant is not in contact with mandibular canal, but clear borders of osteotomy are reaching canal in 34 jaw dental segment region (subject 9).

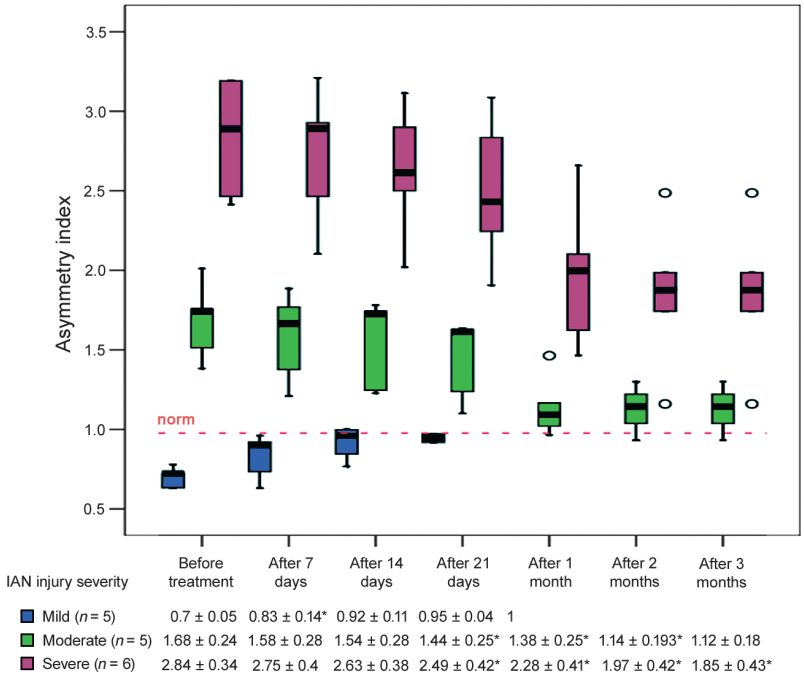

Fig. 3. Boxplot diagram illustrating the dynamics of the inferior alveolar nerve (IAN) recovery depending on nerve injury severity. All the data were plotted as the mean value ± SD. *Significant difference in distribution of one group asymmetry index values in time, depending on asymmetry index value before treatment ($P < 0.05$).

due to injection as patient reported a major painful sensation during local anesthesia. In these three cases only medicament treatment was used.

Recovery of IAN function

The dynamics of functional IAN recovery depended on the injury of the nerve (Fig. 3). In cases with mild injury, after 7 days follow-up the mean AI 0.83 ± 0.14 increased significantly ($P < 0.05$), but still remained lower when compared with the norm. One month after treatment, AI was equal with the norm.

The AI for the patients with moderate nerve injury dropped down statistically significantly after 21 days of treatment and continued to decrease during 2 months period ($P < 0.05$). Nonetheless, after 3 months the mean AI (1.12 ± 0.18 [subjects 6 and 11]) remained slightly higher than the norm.

AI for patients with severe nerve injury showed a statistically significant ($P < 0.05$) decrease after 21 days and continued to decrease till 3 months ($P < 0.05$). In contrast with patients with moderate IAN injury AI remained dramatically increased (1.85 ± 0.43) even after 3-month treatment. All patients in this group demonstrated moderate nerve injury except subject 8 which showed severe injury (AI = 2.5) due to full implant intrusion into mandibular canal (Fig. 1).

Discussion

Nerve sensory impairments are related to nerve injury severity (Hubbard 1972). All patients presented here with mild IAN injury recovered within 1-month after treatment.

Expressed considerable improvement in patients with moderate nerve injury: subjects 5 and 15 recovered after 2 months, subject 12 after 3 months and the remaining two cases (subjects 6 and 11) had AI close to norm 1.1 and 1.3, respectively. In contrast, patients with severe IAN injury demonstrated insufficient nerve recovery result, where mean AI was 1.85 ± 0.43. The duration of post-injury was from 13 (subject 9) to 336 (subject 10) h. If subject 10 was excluded then duration of post-injury was between 13 and 52 h. Hence, peri-implant infection was suspected as the possible cause for subject 10.

To be able to successfully manage IAN injury, it is important to evaluate possible related risk factors that included general, intraoperative, and post-operative risk factors (Juodzbalys et al. 2011). General risk factors include patient's expectations as well as the possibility of IAN injury. Patient must sign informed consent form prior to implant surgery (Nazarian et al. 2003). It is important for clinicians to perform a neurosensory examination, especially mandibular nerve function, before placing the implant to rule out preexisting altered sensation. Great care must be taken when selecting possible sites for implant placement (Kraut & Chahal 2002).

Intraoperative risk factors can be an indicator of possible IAN damage. For example, pain during local anesthesia was noticed in subject 14 hence IAN injury due to injection needle was suspected. Injury of an IAN can occur during a traumatic local anesthesia injection (Malamed 2010). Although very rare, nerve injury after administration of an IAN block was well documented (Haas & Lennon 1995; Pogrel et al. 1995; Ruggiero 1996; Lustig & Zusman 1999; Pogrel & Thamby 1999; Chang & Mulford 2000; Pogrel & Thamby 2000; Smith & Lung 2006; Pogrel 2010; Renton et al. 2010; Wyman 2010). The exact mechanism of the injury by injection needle is yet to be determined (Smith & Lung 2006). Nevertheless, following theories: direct trauma from the injection needle (Haas & Lennon 1995; Crean & Powis 1999; Pogrel & Thamby 2000), hematoma formation(Haas & Lennon 1995; Pogrel et al. 1995; Ruggiero 1996; Crean & Powis 1999; Pogrel & Thamby 2000) and neurotoxicity of local anesthetic (Haas & Lennon 1995; Pogrel et al. 1995; Pogrel & Thamby 1999; Chang & Mulford 2000; Kirihara et al. 2003; Saray et al. 2003) were proposed.

Pain during bone preparation was registered in subjects 3 and 13 and radiographic examination confirmed partial implant intrusion in both cases. In addition, slippage of the drill (subjects 6 and 11), changed pre-planned implant size (deeper – subject 9 and 16 or wider – subject 1) were other intraoperative contributing factors noted in here. Many implant drills are slightly longer, for drilling efficiency, than their corresponding implants. Implant drill length varies and must be understood by the surgeon because the specified length may not reflect an additional millimeter so-called "y" dimension (Alhassani & AlGhamdi 2010). Lack of knowledge about this may cause avoidable complications (Kraut & Chahal 2002). Damage to the IAN can occur when the twist drill or implant encroaches, transects, or lacerates the nerve.

Over penetration of the drill (drill slippage) can be triggered by the low resistance of the spongy bone (Worthington 2004). It is interesting to know that Başa & Dilek (2011) assessed the risk of perforation of the mandibular canal by implant drill using density and thickness parameters. They investigated whether the resistance of the bone surrounding the mandibular canal had sufficient density and thickness to avoid perforation by implant drills. The results showed the risk of IAN injury can be avoided by accurately determine the bone mass around the canal and avoid use excessive force when approaching the canal (Başa & Dilek 2011).

Analysis of radiologic examination showed that in four (25%) cases, implant drill was identified as the etiological factor, with two cases caused by drill slippage during osteotomy preparation. The IAN may be affected by perforation of the mandibular canal during drilling, or positioning the implant close to the canal and the subsequent formation of an adjacent hematoma that presses against the nerve (Lamas Pelayo et al. 2008). Khawaja & Renton (2009) indicated that "cracking" of the IAN canal roof by its close proximity to preparation of the implant bed (millimeters) may cause hemorrhage into the canal or deposition of debris which may compress and cause ischemia of the nerve.

The implant was the most common etiological factor of nerve injury in our study. It was registered in nine cases (56.3%) including six cases of partial and one with full intrusion into mandibular canal. Limited evidence exists with regard to the proper distance between the implant and the mandibular canal to ensure the nerve's integrity and physiologic activity. The proper distance should come from evaluation of clinical data as well as from biomechanical analyses (Sammartino et al. 2008; Guan et al. 2009). Sammartino et al. (2008) created a numeric mandibular model based on the boundary element method to simulate a mandibular segment containing a threaded fixture so that the pressure on the trigeminal nerve, as induced by the occlusal loads, could be assessed. They found that the nerve pressure increased rapidly with a bone density decrease. A low mandibular cortical bone density caused a major nerve pressure increase. In conclusion, they suggested a distance of 1.5 mm to prevent implant damage to the underlying IAN, when biomechanical loading was taken into consideration. After radiologic examination we concluded that in subject 12 implant was too close (<1 mm) to mandibular canal. Moderate IAN injury was registered and this was the reason for dental implant removal. After 3 months AI was back to norm.

Post-operative risk factors are often associated with post-surgery nerve compression due to infection or swelling compression. Sensory IAN injury can be evoked by post-operative peri-implant infection. Implant periapical lesions are infectious-inflammatory alterations surrounding an implant apex, and can be caused by a number of situations – including contamination at instrumentation, overheating of bone, and the prior existence of bone pathology (Peñarrocha Diago et al. 2006). Elian et al. (2005) reported a patient with typical signs of peri-implantitis and IAN injury. The implant was placed in proximity to the mental foramen and possibly had traumatized the mental nerve. After removal of the implant, a considerable diminishing of the paresthesia had occurred, patient reported at least 40% improvement (Elian et al. 2005). This is in coincidence with subject 10 in our study, where patient developed numbness 2 weeks after surgery and the most likely cause is post-surgical infection. Three months after treatment, nerve recovery showed positive improvement (AI = 1.2).

It is very important to note that in the case of clinician sending a patient to specialist for consultation, it is essential to transfer all background information about intraoperative risk factors and possible etiological risk factors that can lead to nerve injury. In six cases (37.5%), no information was given and in one case (6.3%) it was impossible to identify true etiological factor.

In this study, IAN sensory disturbances were diagnosed based on patients' complaints and clinical symptoms, which included hyperalgesia or hypoalgesia of the skin. An electrical stimulus of the skin was employed for PDT assessment. It was shown that electrical stimuli selectively activate thick mye-

linated Ab fibers (Misch & Resnik 2010). Hyperalgesia or hypoalgesia was diagnosed by comparing electric PDT at the skin of both non-affected and affected sides. The results were expressed as AI, which was calculated as a ratio between the injured side and the intact side PDT. In previous clinical and animal studies, it has been shown that hyperalgesic responses to electrical stimuli are caused by inflammation whereas hypoalgesia was triggered by nerve damage (Pogrel 2010; Renton et al. 2010). In this study, 31.25% had hyperalgesia and remaining 68.75% expressed hypoalgesia. Using proposed IANI-DIS protocol to point our risk factors, identify etiological factors, to treat and monitor IAN injury proof to be a valid approach as all patients showed substantial improvement. Eight of them completely healed and the remaining seven (including five with severe nerve injury) had moderate sensory alteration and remaining one (severe injury case) continued experience of severe sensory alteration.

Conclusions

Injury of IAN during dental implant placement can be a serious complication. Clinician should recognize and exclude possible risk and etiological factors that might lead to nerve injury. The most frequent (50%) risk factor was intraoperative bleeding during bone preparation. The most common (56.3%) etiological risk factor of nerve injury was dental implant. The worst treatment results were registered for patients with severe nerve injury. A six-step protocol aimed at managing patients with IAN injury during dental implant surgery was a useful tool that could provide successful treatment outcome. Proper pre-surgery planning, timely diagnosis, and treatment are the key to avoid nerve sensory disturbances management.

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