ISSN 1392-6373
SVEIKATOS MOKSLAI
Volume 21, Number 2, 2011, p. 89-95

CASE REPORT: INFERIOR ALVEOLAR NERVE INJURY DUE TO ROOT CANAL TREATMENT, DIAGNOSIS AND TREATMENT POSSIBILITIES

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Key words: *inferior alveolar nerve injury, inferior alveolar nerve paresthesia, overextension of endodontic filling materials, endodontic medicaments toxicity.*

Summary
This article reports a case of inferior alveolar nerve anaesthesia caused by overextension of root canal filling material, reviews the classification and diagnostics of inferior alveolar nerve injury and makes suggestions for its management.

INTRODUCTION

Inferior alveolar nerve (IAN) injury may be caused by dental implant therapy, during local anaesthesia, endodontic treatment such as periapical surgery, overinstrumentation, irritant root canal medicaments and overfilling by filling materials. It is estimated that incidence of implant-related IAN injuries vary from 0–40%, injuries caused by local analgesia block injections have an estimated injury incidence of between 1:26,762 to 1:800,000. Third molar surgery-related inferior alveolar nerve injury is reported to occur in up to 3.6 % of cases permanently and 8% of cases temporarily [1].

There are no certain numbers of IAN injuries due to root canal treatment, whereas these complications are relatively rare, but the use of thermoplastic endodontic filling materials is becoming more popular with practitioners who perform endodontic therapy, thus nerve injury may be encountered more frequently [2]. Moreover after review of many articles on IAN injury it turned out that extrusion of any other root canal filling material in mandibular teeth has damaging effect on the nerve. Clinically IAN injury occurs as sensory disturbances such as paresthesia, anaesthesia, hypoesthesia and hyperaesthesia in the oral cavity and IAN innervated skin area.

CASE REPORT

A middle-aged woman was seen in a clinic regarding pain and swelling in the left side of upper jaw. She had seen an oral-surgeon approximately 2 years earlier regarding same symptoms in the left premolar area of upper jaw. Patient undergone retreatment of root canals and apisectomy of first and second premolars. Following another 2 years of continuing symptoms, she sought the advice of a second oral-surgeon and came to our clinic.

During accurate examination it was discovered that a patient has a numbness of right side in the mental area that occured after root canal treatment in 47 tooth 10 years ago.

She reported that the pain after treatment 10 years ago was diminishing slowly, but the numbness was unchanged. Her teeth in the third quadrant felt "wooden." She complained of drooling and of difficulty applying lipstick. The patient had no history of any general disease.

A dental 3D cone beam CT imaging was taken. It showed radiopaque material in the area of the inferior alveolar canal extending in a posterior direction from the apex of the mesial root of tooth 47 (Fig.). No other specific details of the endodontic procedure or used filling material were available.

The aim of this review is to discuss clinical diagnostics of IAN injury after oral treatment, treatment possibilities and chances of recovery in a case that we present.

RESULTS

Using keywords, such as "inferior alveolar nerve injury", "inferior alveolar nerve paresthesia", "overextension of endodontic filling materials", "endodontic medicaments toxicity", we selected literature through a search of PubMed, Embase and Cochrane electronic database. The research was restricted to articles published from 1991 to 2010. More than 30 articles were reviewed, but only 16 of them were used as relevant.

Iatrogenic injury is the most frequent cause of sensory disturbances in the distributions of the inferior alveolar

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and mental nerves [3] . However, this neurosensory disturbance of the lower lip and chin is often something that does not bother the patient or only rarely does so. Nevertheless, IAN injury can not only give rise to unpleasant sensations, but may also affect the ability to talk and masticate effectively without traumatizing the affected area [4]. The nerve deficit may give rise to continuous aching in the lower face (hyperalgesia, neuralgia) and social suffering. Some patients complain of pain or other strange sensations (allodynia, dysesthesia, paresthesia) when touching the area of altered sensation in the lower lip.

According to Littner and colleagues [5] the upper border of the mandibular canal is located 3.5 to 5.4 mm below the root apices of the first and second molars. Even though the relation between the IAN and the molar root apices varies, these structures are sometimes very close, allowing pathologic periapical conditions or careless endodontic procedures to affect the nerve structures in the mandibular canal.

It is known that root canal filling materials, including gutta- percha and sealers, can induce paresthesia via mechanical or chemical mechanisms [6]. Their spread beyond the apical foramen can result in clinical manifestations related to the toxicity of the product, although minor material extrusions are generally well tolerated by the periradicular tissues [7].

Morphologically the nerve fiber is the functional component of the peripheral nerve responsible for transmitting stimuli.The A-beta fibers are the next largest myelinated axons so sensation of touch is attributed to these axons. The smallest of the myelinated fibers are the A-delta fibers, which transmit stimuli encoded for temperature and pain. The smallest axons are the unmyelinated C-fibers. They transmit stimuli encoded for slow or second pain, temperature [8].

In 1943, Seddon described a triple classification of mechanical nerve injuries to characterize the morphophysiologic types of mechanical nerve injuries [9]:

1. Neuropraxia- is the least severe form of nerve injury, with complete recovery.In this case, the actual structure of the nerve remains intact, but there is an interruption in conduction of the impulse down the nerve fiber. Most commonly, this involves compression of the nerve or disruption to the blood supply (ischemia. The response to this type of injury is paresthesia. Normal sensation or function returns within 1 to 2 days following the resolution of intrafascicular edema, generally within 1 week following nerve injury [10].

2. Axonotmesis-This is a more severe nerve injury

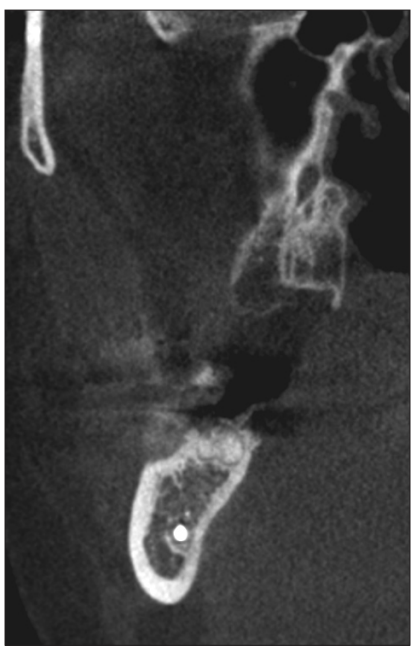
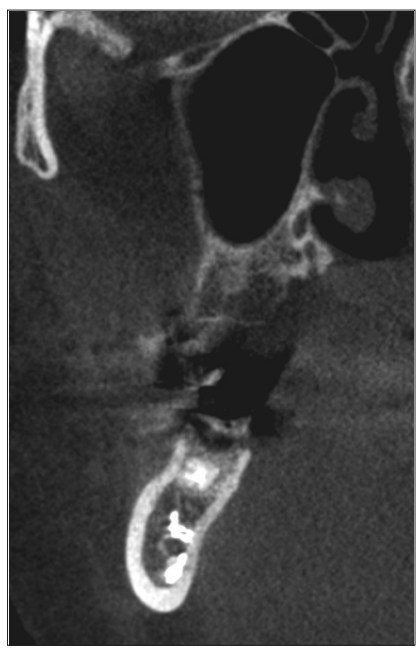


Fig. 1.



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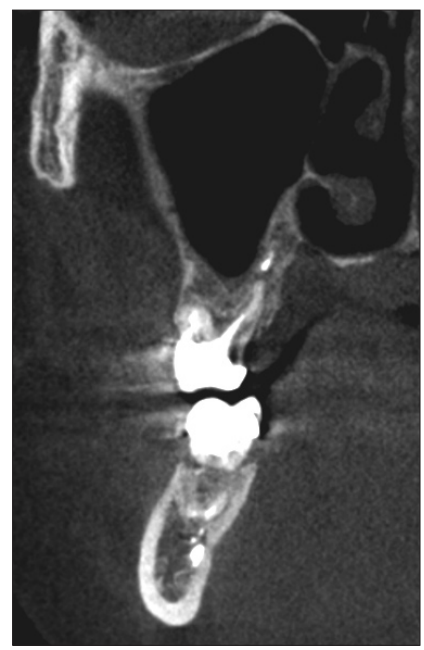


Fig. 3.

Fig. 1,2,3,4,11 - transversal section of the jaw, showing radio opaque root filling material in the mandibular canal and around it in the area of 47 tooth.

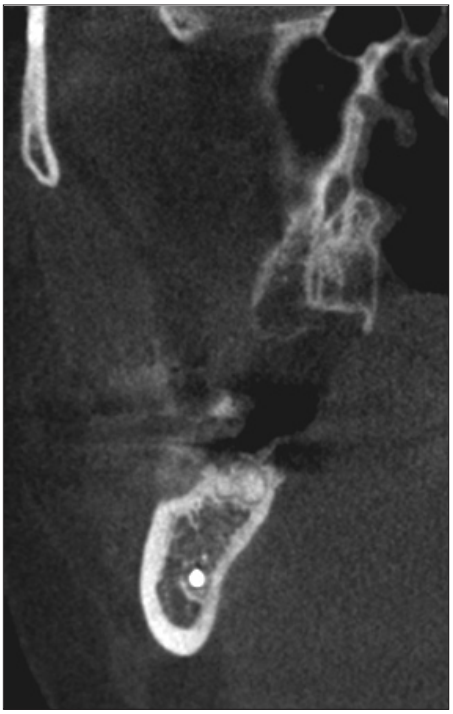
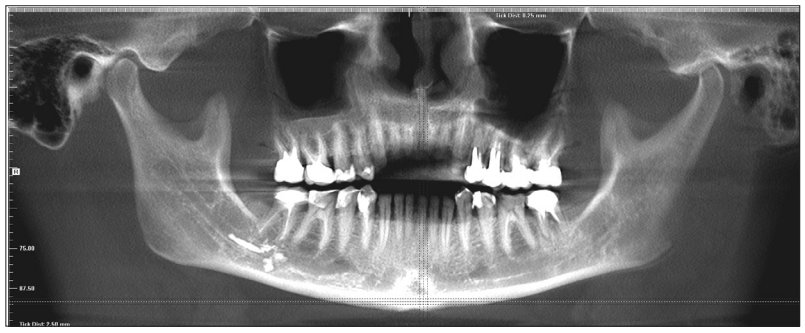


Fig. 4.

with disruption of the neuronal axon, but with maintenance of the myelin sheath. Traction and compression are the usual mechanisms of this type of injury and may cause severe ischemia, intrafascicular edema, or demyelination.Complete recovery occurs in 2 to 4 months, but improvement leading to complete recovery may take as long as 12 months. The psychophysical response to an axonotmesis is an initial anaesthesia followed by a paresthesia as recovery begins [10].

3. Neurotmesis is characterized by severe disruption of the connective tissue components of the nerve trunk with compromised sensory and functional recovery. It occurs on severe contusion, stretch, laceration, or local medicaments toxicity. The psychophysical response to these injuries is an immediate anestesia. This may be followed by paresthesia or possibly neuropathic responses such as allodynia, hyperpathia, hyperalgesia, or chronic pain. This type of nerve injury has a high



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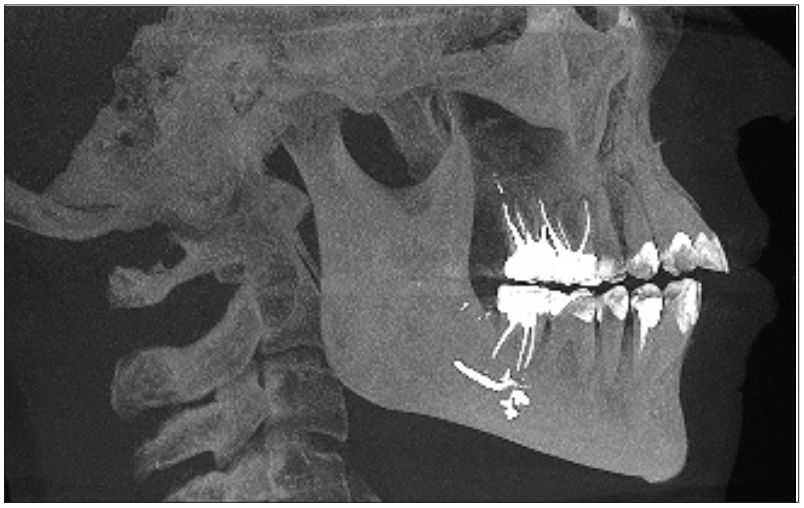

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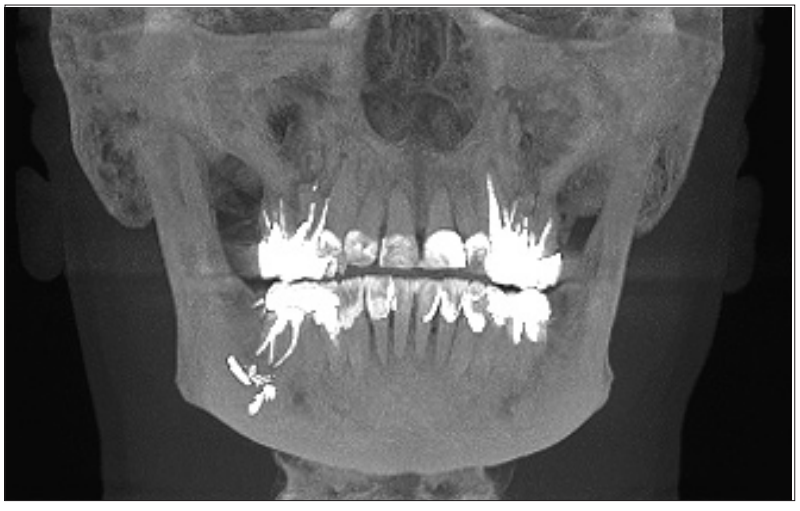


Fig. 7.

Fig. 5,6,7 - root filling material extended from apexes of 47 tooth's roots to retromolar region seen in orthopantomogram, sagittal and transversal cephalograms.

probability of development of a central neuroma [10].

It is was observed chemical nerve damage caused by root canal filling materials is usually followed by neurapraxia and axonotmesis of the nerve fibers, while mechanical trauma or mechanical nerve compression causes neurotmesis [11].

The methods of evaluation of the neurosensory function of the lower lip and chin vary widely, from pure patient questioning to sophisticated, high-technological examination modalities. The sensory diagnostic evaluation is important to document whether or not a neurosensory disturbance exists, to quantitate the disturbance, to monitor sensory recovery, to determine whether or not special treatment may be effective.

At present, diagnosis of sensory disturbances of the IAN is still mostly based on clinical sensory testing that is subjective. It can be divided into two basic categories, mechanoceptive and nociceptive, based upon the specific receptors stimulated through cutaneous contact. Mechanoceptive tests include static light touch, two-point discrimination and brush stroke direction. Pin tactile discrimination and thermal discrimination are nociceptive tests. Each test assesses specific categories of receptors and axons. Trigeminal small-fiber function (A-delta and C) can be studied with thermal QST (quantitative sensory testing) of the cool, warm, heat pain and cold pain detection thresholds or with laser-evoked potential recording. Thermal QST may remain abnormal years after axonal damage and aids in the diagnosis of late sequelae of trigeminal nerve injury. Another subjective clinical sensory test is diagnostic nerve block. It is one part of the diagnostic evaluation when pain is a symptom.

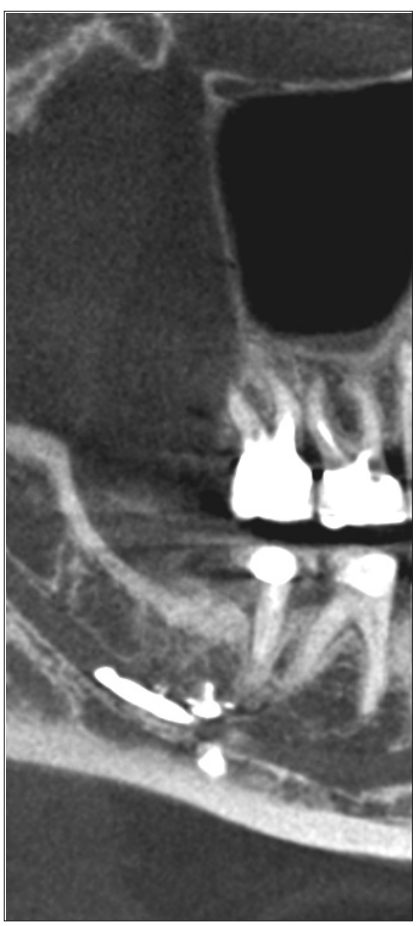
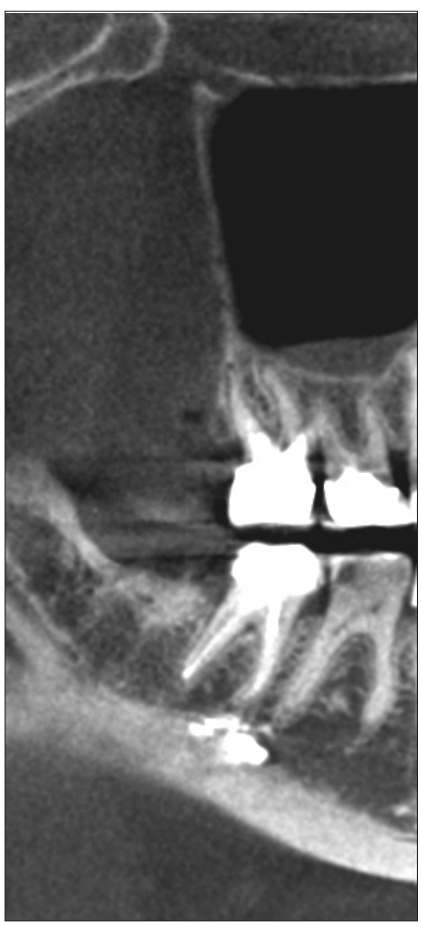


Fig. 8.



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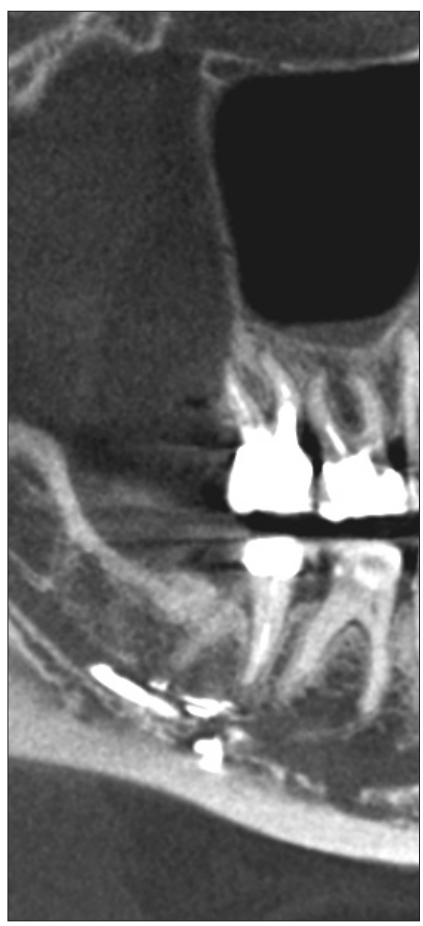

Fig. 10.

Fig. 8,9,10 - root filling material seen packed in mandibular canal and bone lesion around mesial roots of the tooth in sagittal sections of radiogram.

The purpose of it is to aid in determining the mechanism of pain, locating the source of the pain, identifying the pain pathway, and determining the prognosis for decreasing or eliminating the pain.

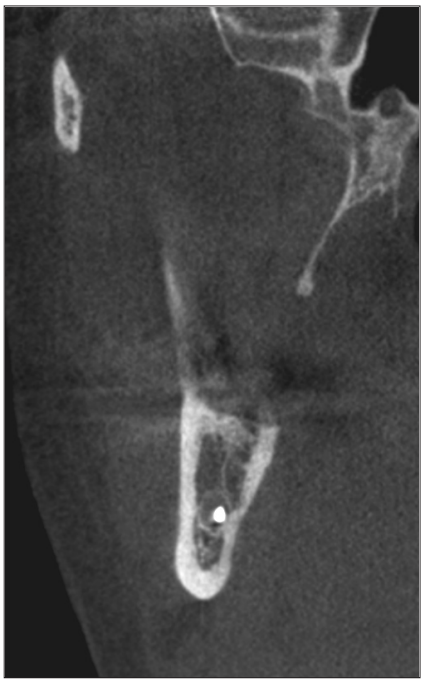

Fig. 11.

Objective sensory tests are:

1. Trigeminal somatosensory evoked potentials (TSEP) is an electrophysiologic method of evaluating the trigeminal pathway. The potential changes of cerebral origin will be detected on the scalp in human subjects after electrical stimulation of peripheral nerves [12].

2. Orthodromic sensory nerve action potential (SNAP) recording [13]. It is used routinely in combination with electromyography (EMG) to assess peripheral nerve function

3. Blink reflex with stimulation of the mental nerve. In this technique, the active recording electrodes are placed on the outer border eyelids on the orbicularis oculi muscles on both sides. The stimulating cathode is placed on the vermilion border of the lower lip midway between the midline and the corner of the mouth. Stimulation of the mental nerve is made with a larger bipolar surface electrode between the stimulating cathode and the anode. The blink reflex responses are recorded simultaneously on both sides. Blink reflex proved to be a sensitive test in detecting IAN lesions within two to three months from injury [14].

Nerve damage following endodontic treatment may result from physical and/or chemical injuries from filling sealers and application of root canal medicaments [15] containing formaldehyde, calcium hydroxide, eugenol into the canal of the root, which is close to the nerve trunk.

Treatments for paresthesia include a removal of the cause and conservative (promotion of nerve regeneration) or surgical (nerve repair) procedures. The former can be applied to neurapraxia and axonotmesis, while the latter can be applied to neurotmesis.

According to Zuniga [16], better treatment outcomes are achieved if mental nerve paresthesia is treated as early as possible. The longer the mechanical or chemical irritation persists, the more the nerve fibres degenerate and the greater the risk that the paresthesia will become permanent [11].

In cases of nonpersistent episodes of nerve irritation, paresthesia should resolve within days or weeks as the cause is removed. The immediate therapy should be based on removal of the cause (when possible) and control of inflammation, edema, hematoma or infection. Repetitive microscopic endodontic irrigation using physiological saline solution can reliably remove root canal medicaments that cause nerve damage. Drugs for such therapy include antibiotics, nonsteroidal anti-inflammatory drugs and corticosteroids, proteolytic enzymes to disintegrate the coagulum and vitamin C, which has antioxidative action and reduces the effects of ischemia.

During the reparative phase (within 30 days of the damage), both pharmacologic and instrumental methods can be used. Drugs include topical steroids, cocarnitine, somatotropic hormone, nerve growth factor, vitamin C and E (antioxidative), vasodilators (to reduce ischemia) and ozone, which improves the activity of red corpuscles and increases tissue oxygenation [17,18]. Instrumental therapy includes magnetotherapy, laser therapy and application of electrical fields. In a late phase, when repair is no longer possible, the pharmacologic approach is limited to the treatment of persistent neuralgia [18].

In cases of non-resorbable filling materials and the nerve injury solely due to mechanical causes surgical debridement of the inferior alveolar canal and decompression of the inferior alveolar nerve is considered. Strauss and colleagues[19] found that 92.2% of patients who underwent IAN microsurgery had statistically significant neurosensory improvement.

DISCUSSION

Examination of our patient showed evidence of altered sensation in the right lower lip from the midline to the commissure, extending upward to and including the vermilion of the lower lip and down to the inferior border of the mandible in comparison with the contralateral side. She had greatly reduced cold, pinprick and light-

touch detection and 2-point discrimination in the entire field in question. Intraoral examination revealed normal sensation in the tongue and lingual gingiva and complete anaesthesia of the labial gingiva from the mandibular right second bicuspid to the midline. The cranial nerves were otherwise unremarkable. Physical examination was unremarkable aswell. According to patient, there was no changes in lip sensation since root canal treatment.

However, there was no details about filling material in previously described case. Considering most commonly used root canal filling materials in Lithuania at the time when patient undergone treatment, it's radiologic view and nonresorbability after 10 years, we may assume that roots were filled with calcium phosphate cement, commonly known as hydroxyapatite cement. "It is composed of tetracalcium phosphate and dicalcium phosphate reactants. These compounds, when mixed with water, react isothermally to form a solid implant composed of carbonated hydroxyapatite. It is as radio opaque as bone. It demonstrates excellent biocompatibility, does not cause a sustained inflammatory response or toxic reaction" [20]. Considering examination results and the fact that there was no recovery signs of the nerve since the injury, the patient most likely suffered mechanical nerve compression that caused neurotmesis of the right IAN. According to Yatsuhashi [15], in several cases of IAN injury due to root canal treatment all successful treatment measures were taken within a month after injury. Therefor, we concluded that in a given situation no treatment would improve present condition.

CONCLUSIONS

Long-term or even permanent paresthesia can result in cases of nerve fibre laceration, prolonged pressure on the nerve or contact with toxic overfilled endodontic materials. In most cases damage can be reversible if a necessary conservative or surgical treatment is applied in time. However, in earlier discussed case no treatment could be beneficial. Thus the most important lesson from this case is prevention of this type of nerve injury. Dentists and endodontists must be aware of the consequences of overextension or periapical extrusion of endodontic filling materials.

References

1. Mohammadi Z. Endodontics-Related Paresthesia of the Mental and Inferior Alveolar Nerves: An Updated Review. J Can Dent Assoc 2010;76:117.

2. Blanas N. Inferior Alveolar Nerve Injury Caused by Thermoplastic Gutta-Percha Overextension. J Can Dent Assoc 2004;70(6):384–7.

3. Ventä I, Lindqvist C, Ylipaavalniemi P. Malpractice claims for permanent nerve injuries related to third molar removals. Acta Odontologica Scandinavica 1998;56(4):193-196.

4. Jones DL, Wolford LM, Hartog JM. Comparison of methods to assess neurosensory alterations following orthognathic surgery. Int Adult Orthod Orthognath Surg 1990;5:35-42.

5. Littner MM, Kaffe I, Tamse A, Dicapua P. Relationship between the apices of the lower molars and mandibular canal — a radiographic study. Oral Surg Oral Med Oral Pathol 1986;62(5):595-602.

6. Tilotta-Yasukawa F, Millot S, El Haddioui A, Bravetti P, Gaudy JF. Labiomandibular paresthesia caused by endodontic treatment: an anatomic and clinical study. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102(4):47-59.

7. Pertot W, Camps J, Remusat M, Proust J. In vivo comparison of the bio- compatibility of two root canal sealers implanted into the mandibular bone of rabbits. Oral Surg Oral Med Oral Pathol 1992;73(5):613-20.

8. Ylikontiola L. Neurosensory disturbance after bilateral sagittal split osteotomy. Oulu university press. Oulu, 2002.

9. May M, Schaitkin BM. The Facial Nerve, 2 edition; 115.

10. La Banc JP. Reconstructive microneurosurgery of the trigeminal nerve. In: Bell WH (ed) Modern practice in orthognathic and reconstructive surgery, WB Saunders Co, Philadelphia, 1992; 1083-1089.

11. Malamed SF. Local complications. Handbook of local anaesthesia. 4th ed. St Louis: Mosby 1997; 248-9.

12. Nakagawa K, Koichiro U, Takatsuka S, Takazakura D, Yamamoto E. Somatosensory-evoked potential to evaluate the trigeminal nerve after sagittal split osteotomy. Oral Surg Oral Med Oral Pathol 2001; 91:146-152.

13. Jääskeläinen SK, Peltola JK, Forssell K & Vähätalo K. Evaluating function of the inferior alveolar nerve with repeated nerve conduction tests during mandibular sagittal split osteotomy. J Oral Maxillofac Surg 1995; 53:269-279.

14. Jääskeläinen SK, Peltola JK. Clinical application of the blink reflex with stimulation of the mental nerve in lesions of the inferior alveolar nerve. Neurology 1994;44:2356-2361.

15. Yatsuhashi T. Inferior alveolar nerve paresthesia relieved by microscopic endodontic treatment: case report. Bull. Tokyo Dent. Coll.2003; 44(4):209-212.

16. Zuniga JR, Meyer RA, Gregg JM, Miloro M, Davis LF. The accuracy of clinical neurosensory testing for nerve injury diagnosis. J Oral Maxillofac Surg 1998;56:2-8.

17. Ozkan BT, Celik S, Durmus E. Paresthesia of the mental nerve stem from periapical infection of mandibular canine tooth: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008; 105(5):28-31.

18. Franco A, Ferronato G. Nervo mandibolare in odontostomatologia. Padova, Italy, Frafin Sas, 1996.

19. Strauss ER, Ziccardi VB, Janal MN. Outcome assessment of inferior alveolar nerve microsurgery: a retrospective review. J Oral Maxillofac Surg. 2006;64(12):1767-70.

20. Vasudev SK, Goel BR, Tyagi S. Root end filling materials — A review. Endodontology, 2003 Vol. 15.

21. Zuniga JR. Surgical management of trigeminal neuropathic pain. Atlas Oral Maxillofac Clin North Am. 2001;9(2):59-75.

22. Gregg JM. Surgical management of inferior alveolar nerve injuries (Part II): The case for delayed management. J Oral Maxillofac Surg. 1995;53(11):1330 -3.

23. Pertot W, Camps J, Remusat M, Proust J. In vivo comparison of the bio- compatibility of two root canal sealers implanted into the mandibular bone of rabbits. Oral Surg Oral Med Oral Pathol. 1992;73(5):613-20.

24. Hong YC, Wang JT, Hong CY, BROWN WE, Chow LC. The periapical tissue reactions to a calcium phosphate cement in the teeth of monkeys. J Biomed Mater Res. 1991;25(4):485-98.

KLINIKINIS ATVEJIS: APATINIO ALVEOLINIO NERVO PAŽEIDIMAS PO ŠAKNŲ KANALŲ GYDYMO, PAŽEIDIMO DIAGNOSTIKA IR GYDYMO GALIMYBĖS

Vaidas Varinauskas, Tatjana Nimčenko, Ričardas Kubilius

Raktažodžiai: apatinio alveolinio nervo pažeidimas, apatinio alveolinio nervo parestezija, šaknų kanalų plombinės medžiagos prastūmimas, endodontinių preparatų toksiškumas.

Santrauka

Straipsnyje aprašoma klinikinė apatinio alveolinio nervo anestezijos situacija, kai dėl nervo pažeidimo prastumta už šaknies kanalo plombine medžiaga. Nurodoma nervo pažeidimo klasifikacija, diagnostikos bei galimo gydymo būdai.

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Gauta 2011-03-07

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