Case Report

Endodontic periapical lesion-induced mental nerve paresthesia

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ABSTRACT

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Address for correspondence: Dr. Neda Shekarchizade, Department of Endodontics, School of Dentistry, Isfahan University of Medical Sciences, Isfahan, Iran. E-mail: neda_ shekarchizade@dnt.mui.ac.ir Paresthesia is a burning or prickling sensation or partial numbness, resulting from neural injury. The symptoms can vary from mild neurosensory dysfunction to total loss of sensation in the innervated area. Only a few cases have described apical periodontitis to be the etiological factor of impaired sensation in the area innervated by the inferior alveolar and mental nerves. The aim of the present paper is to report a case of periapical lesion-induced paresthesia in the innervation area of the mental nerve, which was successfully treated with endodontic retreatment.

Key Words: Apical periodontitis, endodontic therapy, inferior alveolar nerve, paresthesia

INTRODUCTION

Paresthesia, anesthesia, hypoesthesia and hyperesthesia are some of the sensory disorders occurring in the oral cavity [Table 1].^[1] Paresthesia has been defined as a feeling of burning or prickling or partial numbness, resulting from a traumatic injury of a nerve. Patients might have complaints of a sensation of warmth and coldness, along with burning, prickling, aching, tingling, pins and needles and formication experiences so far.^[2-4]

Paresthesia might be attributed in the dental field, to a wide variety of local and systemic etiologic factors; traumatic injuries such as mandibular fractures, expanding and compressive lesions (cysts and benign and malignant neoplasms), impacted teeth, local infections (osteomyelitis and periapical and periimplant infections), iatrogenic lesions after tooth extraction, injection of anesthetic agents, root canal therapy (overfilling and periapical surgery), orthodontic surgery and preprosthetic surgery are some of the local factors reported in the literature.^[5]

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Some systemic conditions resulting in orofacial region paresthesia are multiple sclerosis, sarcoidosis, viral and bacterial infections, metastasis, drug-induced conditions and infectious diseases.^[6-14]

Facial paresthesia has been reported to have a known etiology in 83% of cases and only in 48% of cases it has been attributed to a dental procedure or condition.^[3] Inferior alveolar and mental nerves are the most commonly afflicted nerves.^[3,15,16] A prevalence rate of 0.96% for sensory disorders of mandibular premolars has been reported by Knowles *et al.*^[17]

Littner *et al.* reported a distance of 3.5-5.4 mm between the superior border of the mandibular canal and the apices of the first and second molars.^[18] Denio *et al.* reported distances of 3.7 mm and 6.9 mm between the apex of the second molar and the apex of the mesial root of the first mandibular molar and the superior border of the mandibular canal, respectively.^[19] This short distance might pave the way for periapical infections to affect the inferior alveolar nerve. Mechanical pressure and ischemia in inflammatory processes (edema), local pressure on the mental nerve due to accumulation of suppurative exudate within the mandibular bone, bacterial toxins or inflammatory products released from injured tissues might lead to periapical lesions, and finally resulting in paresthesia.^[20]

Only a limited number of papers have reported apical periodontitis as the main etiologic agent for neurologic disorders.^[1,13,15,16] In the majority of these cases, the pulp has been necrotic and radiographic examination has revealed a periapical lesion. In the case presented here, the teeth involved had undergone endodontic treatment previously and had periapical lesions.

CASE REPORT

A 24-year-old female patient, suffering from paresthesia of the left lower lip, was referred to the Department of Endodontics, School of Dentistry, Isfahan University of Medical Sciences. The medical history of the patient was noncontributory. The patient's chief complaints were unilateral anesthesia and a tingling sensation on the left lower lip. This sensory disorder had initiated 3-month previously, with the patient referring to a neurologist, who had prescribed Vitamin B. Despite taking the medication, the condition had persisted. The physician had suspected a problem with a dental origin, therefore, referred the patient to Endodontics Department of Isfahan University Dental School. Panoramic radiographic examination [Figure 1] revealed that the mandibular second premolar and first molar teeth had undergone root canal therapy and had periapical lesions.

Extra-oral examination using sharp/dull and two-point discrimination tests revealed sensory disorders in the mental nerve dermatome on the lower lip [Figure 2]. The patient had facial symmetry with no pain or sinus tracts. Intra-oral examination showed no pathologic changes. The second premolar and the first and second molars on the left side of the mandible had amalgam restorations. There were no recurrent carious lesions at restoration margins. The first premolar and the second molar exhibited positive responses to Endo ice and electrical pulp tests were vital. The first premolar



Figure 1: Preoperative panoramic view of the patient

and the second molar exhibited no tenderness to percussion and palpation of the apical areas [Table 2]. Tooth mobility was in the normal range, and the probing depths were 2.5-3 mm. Intra-oral radiographic examination revealed that the mandibular second premolar and the first molar had unfavorable root canal therapies, with underfillings of the roots in both teeth, and radiolucent lesion in periapical areas of the affected teeth. There was no evidence of perforations or overfillings in the two teeth mentioned above. The mental foramen was located near the radiographic apex of the mandibular second premolar and could be

Table 1: Sensory nerve dysfunctions

Definition
Absence of all sensory modalities
An abnormal sensation (tingling), whether spontaneous or evoked
An unpleasant abnormal sensation, whether spontaneous or evoked
Increased sensitivity to stimulation, excluding special senses
Diminished sensitivity to stimulation, excluding special senses

Table 2: Pulpal and periapical diagnosis of the case and control teeth

Test	The teeth involved		Control teeth	
	Number 19	Number 20	Number 29	Number 30
Pulp vitality tests				
EPT	—	—	+	+
Cold	—	—	+	+
Heat	—	—	+	+
Periapical tests				
Percussion	—	—	—	—
Palpation	—	—	—	—
Mobility	0	0	0	0

EPT: Electrical pulp test

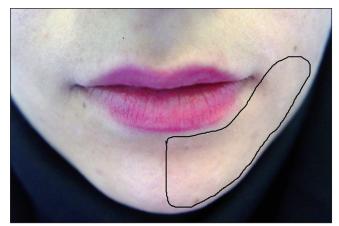


Figure 2: Region of the left lower lip affected by paresthesia

distinguished from the radiolucent periapical lesion of the second premolar by changing the X-ray tube's vertical angulation. Based on clinical and radiographic examinations, a diagnosis of apical periodontitis with endodontic origin was reached in the left mandibular second premolar and the first molar associated with paresthesia of the mental nerve dermatome. Therefore, the treatment plan consisted of endodontic retreatment of the mandibular second premolar and the first molar on the left side. A written informed consent was obtained from the patient. During the first visit, local anesthesia was achieved with 2% lidocaine containing epinephrine 1:100,000 (DAROPAKHSH, Iran). Both teeth were isolated with a rubber dam, and access cavities were prepared. The root canal fillings of the coronal segments were removed with a heat carrier (Dentsply Maillefer, Ballaiques, Switzerland). The remaining Gutta-percha from the apical segments of the root canals was removed by xylene solvent (Ajax Finechem, PTY, India) and k-file no. 15 (Dentsply, Maillefer, Ballaigues, Switzerland). The root canals were irrigated with 2.5% sodium hypochlorite solution, and working length was determined with electronic apex locator (Root ZX® II, Densply, J-Morita Inc., USA). The root canals were instrumented with Protaper system (Dentsply Maillefer; Ballaigues, Switzerland) with a low-torque motor that has torque control and constant speed of 300 rpm (ATR Tecnika, Advanced Technology Research, Pistoia, Italy). SX Protaper file was used to prepare the coronal half of the root canals. Then S1, S2, F1, F2 and F3 files were used to the working length. Between instruments, the root canals were irrigated with 2 mL of 2.5% sodium hypochlorite solution. Calcium Hydroxide (Ca[OH]2) paste (powder mixed with saline) was used as an inter-appointment intra-canal medication, and the access cavity was sealed with Cavit (3M ESPE, Neuss, Germany).

After 2 weeks, the teeth exhibited no tenderness to percussion or palpation. However, paresthesia had not resolved completely. The temporary restoration was removed, and the intra-canal medication was removed with 5% Naocl irrigation. Fresh Ca(OH)2 paste was placed in the root canals. Then, the access cavities were sealed with a temporary restoration.

After 4 weeks, the patient reported complete relieve of the paresthesia. Therefore, the root canals were obturated with Gutta-percha (Gapadent Co. Ltd., Korea) and AH-plus sealer (Dentsply, Maillefer, Germany) using the lateral condensation technique and a final restoration were placed with amalgam build up [Figure 3]. Follow-ups were scheduled at 3-, 6-, 9-, 12- and 18-month intervals. At final followup, radiograph showed a normal structure of the periodontal ligament (PDL) and surrounding bone of the mandibular first molar and second premolar. Patient had no complaint of neurosensory dysfunction of the area innervated by the mental nerve.

DISCUSSION

Traumatic injuries to nerve are classified into three categories based on the severity of trauma and the feasibility of complete postoperative regeneration [Table 3].^[21] In cases of neuropraxia and axonotmesis, elimination of the etiologic agent gives rise to nerve regeneration in a short time.^[22] Most paresthesia cases are due to the neuropraxia, in which a transient trauma is inflicted on the myelin sheath of the nerve, with no injury to the nerve axon.^[23] It appears that the nerve injury in the present case was of this type because, after timely and proper treatment, conduction

Table 3: Neural injuries (Seddon's classification)

Types of neural injuries	Definition
Neuropraxia	Occurs when a nerve is injured and conduction is blocked, but this does not lead to Wallerian degeneration
Axonotmesis	Results in damage to the axons, but the endoneurial and epineurial sheaths are preserved
Neurotmesis	Conduction is completely disrupted resulting in the loss of anatomic integrity of the endoneurium, perineurium and epineurium

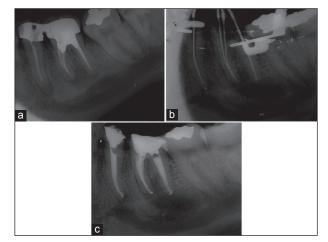


Figure 3: Radiograph: (a) Diagnostic radiograph before retreatment and approximation of mental foramen to apex of left mandibular second premolar, (b) working length radiograph, (c) control radiograph after root canal filling

of nerve impulses returned to normal. Periapical inflammation of mandibular premolars and the distal roots of second molars is the most common etiologic factor for paresthesia of the inferior alveolar and mental nerves.^[17,19] Neurologic symptoms and signs are usually manifested subsequent to re-infection of the root canal system after root canal obturation or during postoperative flare-ups.^[24]

The mental foramen is usually located below the mandibular second premolar apex.^[25] In the present case, mental nerve paresthesia had resulted from the periapical infection of the mandibular second premolar and the first molar; the close proximity between the periapical lesion of the second premolar and the mental foramen had resulted in the paresthesia of the left lower lip.

Periapical inflammation due to an infectious root canal system might give rise to neuropraxia due to hyperemia and inflammatory edema, which compresses the nerve and injuring the nerve structures. Furthermore, ischemia might lead to nerve dysfunction, associated with the symptoms and signs of paresthesia.^[3] Furthermore, Gram-negative bacteria endotoxins and metabolic products of bacteria might have neurotoxic effects.[4,15,16,20] On the other hand, direct invasion of nerve structures by bacteria themselves might be another factor injuring the nerve structures.^[26] Infection-induced paresthesia is usually relieved after inflammation and infection are removed, which can be brought about by antibiotic therapy, endodontic therapy, periapical surgery or tooth extraction.^[14] Medications available to this end consist of antibiotics, nonsteroidal antiinflammatory drugs, corticosteroids, proteolytic enzymes for disintegration of the coagulum, Vitamin C (for its antioxidative activity and its effect on ischemia), Vitamin B_{12} and adenosine triphosphate to promote tissue regeneration. During the reparative phase (a 30-day period after injury), both pharmacologic and instrumental techniques can be used. The medications used include topical steroids, cocarnitine, somatotropic hormones, vasodilators to improve the ischemia, and ozone to promote the activity of red blood cells and increase oxygenation of tissues. Instrumental techniques include magnetotherapy, laser therapy and use of electrical fields. Surgical intervention is effective when the nerve is under pressure by a foreign body or neoplasm or in cases of paresthesia or anesthesia not relieved by previous treatment interventions.^[27,28]

Dental Research Journal / March 2015 / Vol 12 / Issue 2

Since the etiologic factor in the present case was periapical infection of mandibular first molar and second premolar after root canal therapy, the teeth involved underwent an endodontic retreatment because it is a conservative treatment modality, and can eliminate the etiologic agent by removing the intra-canal infection, allowing the regeneration of the mental nerve and periapical tissues. In addition, if this treatment fails, other treatment options such as periapical surgery or tooth extraction are still available.

During retreatment of these teeth care should be exercised in working length determination (with the use of radiography and apex locators), preparation and root canal obturation steps due to the close proximity of the apices of these teeth to nerves. In the case presented here, endodontic retreatment was rendered in three sessions (initiation of treatment, and after 2 and 4 weeks). In this context, after preparation of the canals during the first session, Ca(OH)2 paste was placed in the root canals, and the teeth were sealed with a temporary restoration.

In the second session after 2 weeks, paresthesia had not relieved completely, so fresh Ca(OH)2 paste was placed in the root canals again. During the third session, since paresthesia signs had disappeared, the root canals were obturated with great care and a permanent restoration was placed.

Different times have been reported for the relief of paresthesia symptoms and signs (2-day to 1-year). However, in most cases it had been reported to have resolved in a period of 1-3 weeks.^[1,17,29,30]

In the case presented here, the paresthesia of the left lower lip was relieved in 4 weeks after endodontic retreatment, and follow-ups were scheduled at 3-, 6-, 9-, 12- and 18-month intervals. At 6-month recall visit, radiographic examination showed complete regeneration of periapical structures and tissues. An 18-month follow-up radiograph showed a continuous PDL space with normal width around the mandibular first molar and second premolar. The mental foramen was located near the apex of the second premolar. Clinically, the response of the teeth to periapical tests was within the normal range, and sign of paresthesia had disappeared completely [Figure 4].

This case showed that endodontic retreatment supported with Ca(OH)2 medicament is a treatment option to resolve periapical lesion-induced parasthesia. The major factor contributed to the favorable outcome was a proper endodontic retreatment.

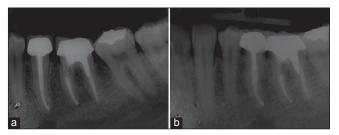


Figure 4: Radiograph check-up examinations: (a) Radiologic follow-up after 6-month, (b) radiologic follow-up after 18-month; bone regeneration and complete recovery of sensation

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