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Paresthesia of the Mental Nerve Due to Chronic Apical Periodontitis of a Non-Vital Tooth: A Case Report

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Abstract: Chronic apical periodontitis of mandibular premolars and molars teeth is quite common phenomenon. In this case report, it is described an unusual case of chronic periodontitis of a right mandibular second premolar tooth in proximity to mental foramen which was the cause of a dysaesthesia in the distribution of the mental nerve. The reported symptoms of sensory disturbance disappeared 3 months after conventional endodontic treatment associated with antibiotic therapy. One year later the tooth was still asymptomatic and the resolution of mental nerve paresthesia was completed.

Key words: Mental nerve paresthesia, periapical infection, mental nerve, chronic apical periodontitis

INTRODUCTION

Paresthesia is defined as a sensory disturbance with clinical management manifestations such as burning, prickling, tingling, numbness, or itching (Zmener, 2004). It can include any deviation from normal sensation (Gilbert and Dickerson, 1981). If the mandibular mental nerve is affected, the most common complaints include a transient or permanent loss of sensitivity of the lip, chin and oral mucosa that is often associated with a limited intraoral xerostomia (Zmener, 2004).

A common area for nerve damage to occur is the mental foramen region due to its more superior position in the jaw compared with the rest of the inferior dental canal (Abbott, 1997). Therefore, it is important for dentists to be aware of the position of the mental foramen which some studies report to be situated apical to the second mandibular premolar or between apices of the premolars (Abbott, 1997). However its location can vary from the mandibular canine to the first molar. The mental foramen is a strategically important landmark during dental procedures. Its location and the possibility that an anterior loop of the mental nerve may be present mesial to the mental foramen needs to be considered before any surgery in the foramina area in order to avoid any nerve damage (Abbott, 1997).

According to Seddon classification for peripheral nerve injury, there are three basic injuries: neurapraxia, axonotmesis and neurotmesis (Seddon, 1943). Neurapraxia is a temporary conduction block after mild compression of the nerve trunk (i.e., paresthesia or dysaesthesia of the lip and chin region in case of IAN/mental nerve) (Becelli *et al*., 2002). Axonotmesis, a more serious condition, results from degeneration of the afferent fibers as a result of internal/external irritation (i.e., anaesthesia) (Donoff, 1995). While in neurotmesis the nerve is completely severed which results in permanent paresthesia which can be corrected with microneurosurgical interventions with variable prognosis (Seddon, 1943).

Nerve injury can be related to mechanical, chemical and thermal factors (Yeler *et al*., 2004). Furthermore local and systemic factors have been reported associated with mandibular paresthesia or dysaesthesia in the distribution of the IAN/mental nerve.

The local factors are divided to endodontic and surgical etiology.

The endodontic etiology factors are the severe endodontic infection involving the nerve and the iatrogenic sequelae of endodontic therapy. The iatrogenic sequelae of endodontic therapy are the following: overfilling of root canals close or in contact with the nerve (mechanical or chemical damage), neurotoxicity of the materials used (chemical damage), over-extension of endodontic files during the endodontic therapy (mechanical or chemical damage) and broken endodontic files left at the periapical region (mechanical pressure) (Abbott, 1997; Poveda *et al*., 2006; Lenarda *et al*., 2000).

The surgical etiology factors of mental paresthesia are the improper implant placement, mandibular fracture distal to the lingula, compression of the nerve during dental/surgical instrumentation and orthognathic surgery

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(Lenarda *et al*., 2000; Kafas *et al*., 2009). However in some cases reported, paresthesia may be due to infected impacted tooth (Yeler *et al*., 2004; Lenarda *et al*., 2000) or foreign body reaction (Kafas *et al*., 2009).

Systemic pathoses include metastatic malignancy, schwannoma, compound odontoma and systematic cancer (Zmener, 2004; Yeler *et al*., 2004; Lenarda *et al*., 2000).

Infection-related paresthesia is usually related to mechanical pressure and ischemia associated with the inflammatory process (edema) or it is caused by the local pressure to the mental nerve consequent to the accumulation of purulent exudate in the mandibular bone (Gilbert and Dickerson, 1981; Yeler *et al*., 2004; Lenarda *et al*., 2000). Another cause of paresthesia could be the toxic metabolic products of bacteria or inflammatory products released following periapical tissue damage (Yeler *et al*., 2004; Lenarda *et al*., 2000).

The following case report is refers to an extended chronic apical periodontitis of a mandibular right second premolar tooth which was the cause of mental nerve dysaesthesia to a patient.

CASE REPORT

A 45-years-old Mediterranean male was referred to the School of Dentistry of Aristotle University of Thessaloniki in September of 2009. The patient had a complaint of numbness of the right skin mucosa of the lower lip and right chin region for the last 3 months. The patient, also reported that he has not been to a dentist for several years but he was aware that he had dental problems because of sensitive teeth and loose tooth. Earlier episodes of sensitivity were recalled in the area of the right mandible.

Furthermore the patient reported that one day before he was referred to us he had awakened with severe pain in the area and a slight submandibular swelling on the right side. He also stated that certain swelling disappeared after taking antibiotic and analgesic by his own initiative. Even though the certain swelling and pain disappeared, the numbness of the lower lip and chin area was still occurred. When questioned for his late seek of dental assistance, the patient neglect to report paresthesia thinking it an un-important symptom which would pass soon.

The patient's medical history was remarkable and local factors such as jaw fractures, facial trauma, third molar surgery, odontogenic cyst, local tumor infiltration and metastasis of cancer, all of which have been proven to be responsible for such symptoms, were eliminated. No allergies were reported.

In the clinical examination, there was no lympadenopathy and any swelling in the area prescribed wasn't detected. The intraoral examination showed carious right mandibular 1st molar and 2nd premolar teeth. Vital pulp examination on the premolars revealed a vital 1st premolar and necrotic pulp of 2nd premolar teeth. The electronic pulp tester did not elicit a response from these teeth whereas all the other teeth in the area responded within normal limits.

The patient was evaluated radiographically by periapical radiograph on the first visit (Fig. 1). Radiographic examination revealed a carious and fractured 1st mandibular right molar and a disclosed deep carious involvement of the mandibular right second premolar near the pulp chamber and a radiolucent area associated with the apex. The mental foramen appeared in close proximity to the periapical radiolucent area.

The radiographic and clinical examination as well as the recorded data enable us to diagnose the patient's condition as a chronic periapical pathosis associated with the apex of the mandibular right second premolar. At the first visit, the 1st molar was extracted and the patient was recalled on the second visit to be submitted to conventional endodontic therapy of the 2nd premolar. The mandibular right 2nd premolar was isolated with a rubber dam and the caries was removed. Only one canal was found. Vital tissue was absent and a little exudates was present in the pulp chamber that drained. The root canal was irrigated with 5% sodium hypochlorite. Working length was established with an electronic apex locator (Root Zx; J. Morita Corp., Tustin, CA) and confirmed with a radiograph. The canal was instrumented with a step-back technique after which the root canal was dried with sterile paper points. The tooth was provisionally sealed with a dry cotton pellet and Cavit G on the first visit and second visit (3M Espe, Seefeld, Germany) to allow purulent exudates drainage. The patient was given a prescription for 20 tablets, 500 mg of each of V-cillin K to be taken two times a day. Root canal treatment was completed in 2 visits, within 2 weeks. On the third visit, 14 days after the first visit, the canal was obturated with gutta-percha and a zinc oxide-eugenol cement using a vertical condensation technique after the root canal was dried with sterile paper points. A final radiograph was taken. The root canal lilling was in good condition. The patient was advised to return to his general dentist for restoration of the crown.

The patient was seen for a recall appointment after 3 months. The tooth had been restored with a resin restoration. Recall radiograph showed a reduction in size of the periapical radiolucent area (Fig. 2). Also the patient reported that he was free of any pain and the related tooth

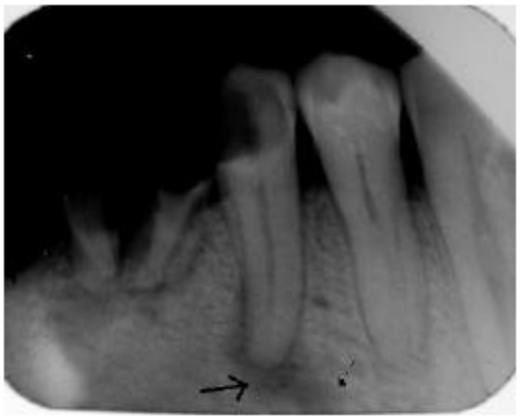

Fig. 1: Periapical radiograph of the right mandible premolar region on the first visit. Radiographic examination revealed a carious and fractured 1st molar and a disclosed deep carious involvement of the 2nd premolar near the pulp chamber. The presence of a periapical radiolucency (dashed arrow) at the 2nd premolar was found to be large enough and extended toward the mental foramen (non-dashed arrow)

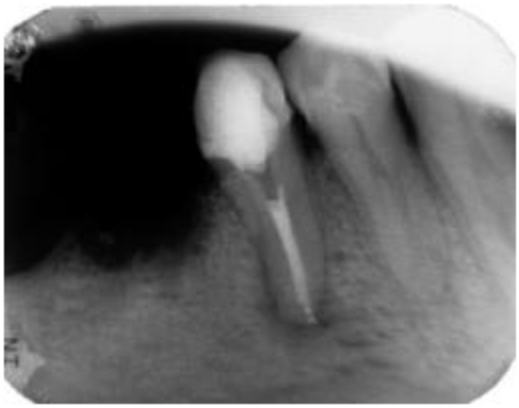

Fig. 2: Periapical radiograph of the right mandible premolar area on the recall visit after 3 months from the endodontic therapy. The periapical radiolucent area was found to be decreased in size which means healing of the periapical tissues and reconstruction of bone tissue

was still asymptomatic. The patient reported that sensation had progressively returned to his lip skin and mucosa, starting proximally and extending to the midline. At the recall after one year from the initial treatment, the patient was still in comfort without any previous symptoms and the resolution of mental nerve paresthesia was completed.

DISCUSSION

In bibliography, several cases of mental nerve dysaesthesia due to non-vital teeth and periapical infection of mandibular canines and premolars are quoted (Zmener, 2004; Gilbert and Dickerson, 1981; Yeler *et al*., 2004; Lenarda *et al*., 2000). In these cases there is a little doubt that the periapical infection and resulting inflammation caused the paresthesia. Also, adequate endodontic therapy was efficiently resolved the paresthesia of these patients, without further clinical complications.

The unusual case report described above, shows an incidence of mental nerve paresthesia which was due to a chronic periapical pathosis associated with the apex of the mandibular right 2nd premolar tooth. The periapical lesion was clearly seen in close proximity to the mental foramen where the mental nerve emerges. It is believed that the toxic metabolic products of bacteria or inflammatory products released following periapical tissue damage was the certain etiology of dysaethesia (Yeler *et al*., 2004; Lenarda *et al*., 2000). The paresthesia of the mental nerve was resolved within 3 months after conventional endodontic treatment and antibiotic therapy. After 1 year recall revealed completely resolution of mental nerve dysaesthesia.

CONCLUSION

Clinicians should be aware of the fact that severe chronic apical periodontitis of mandibular premolars can be extended in such a degree that can lead to mental nerve neurological complications, i.e., dysaesthesia of the mental nerve, due to their proximity to the mental foramen. Conventional endodontic therapy associated with the appropriate antibiotics can completely resolve the dysaesthesia of the mental nerve.

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