Glynn Dale Buchanan, BChD, MSc, PhD,^{*} Chane Smit, BChD, MSc,[†] Mohamed Yasin Gamieldien, BChD, MSc,[‡] and Ahmed S. ElSheshtawy, BDS, MSc, PhD[§]

CASE REPORT/CLINICAL TECHNIQUES

Resolution of Apical Periodontitis-induced Mental Nerve Paresthesia Through Nonsurgical Endodontic Retreatment: A Case Report



SIGNIFICANCE

Untreated periapical lesions may enlarge sufficiently to cause complications such as paresthesia. Such complications may be resolved through nonsurgical endodontic retreatment without the need for surgery. ABSTRACT

Paresthesia is a potential consequence of unsuccessful root canal treatment. Persistent infection resulting in apical periodontitis may enlarge sufficiently to involve the mental neurovascular bundle. The case presented in this report was referred for endodontic evaluation with a chief complaint of discomfort on mastication and persistent numbness of the lower left lip. Clinical and radiographic evaluation revealed incomplete primary endodontic treatment of the left mandibular second premolar with complex internal anatomy. The untreated root canal system resulted in the progression of apical periodontitis involving the left mental neurovascular bundle as confirmed by cone-beam computed tomography imaging. Nonsurgical root canal retreatment was performed over 2 visits. At the 3-year follow-up visit, the paresthesia had resolved with return of normal sensation. Nonsurgical endodontic retreatment may be sufficient to allow healing of large periapical lesions and resolve complications including paresthesia without the need for surgical intervention. (*J Endod 2023;49:920–924.*)

KEY WORDS

Endodontics; mental nerve; nonsurgical retreatment; paresthesia; root canal treatment

From the *Faculty of Health Sciences, Department of Odontology, [†]Faculty of Health Sciences, Department of Oral and Maxillofacial Pathology, and [‡]Faculty of Health Sciences, Department of Maxillofacial and Oral Surgery, School of Dentistry, University of Pretoria, Pretoria, South Africa; and [§]Faculty of Dentistry, Department of Endodontics, Cairo University, Cairo, Egypt

Address requests for reprints to Glynn Dale Buchanan, Department of Odontology, University of Pretoria Oral Health Centre, 31 Bophelo Road, Prinshof Campus, Riviera, Pretoria 0002, South Africa.

E-mail address: glynn.buchanan@up.ac. za

0099-2399

Copyright © 2023 The Authors. Published by Elsevier Inc. on behalf of American Association of Endodontists. This is an open access article under the CC BY-NC-ND licenses (http://creativecommons.org/ licenses/by-nc-nd/4.0/). https://doi.org/10.1016/ j.joen.2023.05.004 The presence of bacteria within the root canal system, organized into intraradicular biofilms, is known to cause apical periodontitis¹. In approximately 80% of failed primary endodontic treatments, bacterial biofilms can be found in the apical third of the root canal system. Successful endodontic treatment aims to resolve apical periodontitis by removing intraradicular biofilms by means of thorough chemomechanical preparation and obturation of the entire root canal system². Bacteria surviving canal disinfection procedures may result in the persistence of apical periodontitis and possible progression of these lesions¹. Whilst the majority of apical periodontitis lesions are asymptomatic², some may progress to involve nearby anatomical structures, including the maxillary sinus or mental neurovascular bundle. This may cause complications in the form of either sinusitis or neurosensory alterations or, in other instances, the symptoms of apical periodontitis and its associated complications, including neurosensory impairment, can be successfully treated through surgical or nonsurgical endodontic treatment¹.

The following report documents a case of paresthesia in the area innervated by the mental nerve, which occurred secondary to persistent apical periodontitis in a previously treated left mandibular second premolar. The case was successfully managed by means of nonsurgical endodontic retreatment without the need for surgical intervention.

CASE REPORT

This report was granted ethical approval from the Research Ethics Committee of the Faculty of Health Sciences, University of Pretoria (Protocol number: 20/2023).

A 59-year-old man was referred to the Division of Endodontics, University of Pretoria, in May 2018 with a complaint of tenderness to mastication on the left mandibular premolar region and paresthesia of the lower left lip of approximately two-month's duration. The medical history revealed well-controlled hypertension.

Clinical Examination

Upon clinical examination, the left mandibular second premolar displayed a large amalgam restoration with evidence of a previous endodontic access cavity sealed with composite resin. The examined area demonstrated a normal soft tissue appearance as well as normal periodontal probing around the tooth. The tooth showed mild tenderness to vertical percussion and no tenderness to lateral percussion or palpation.

Clinical Evaluation of the Neural Injury

The patient's self-evaluation was recorded. The patient reported reduced/altered sensation in the area of the left lip and a history of root canal treatment performed many years previously. No interferences with daily activities, such as eating or speaking were noted. Further neurosensory evaluation using standardized tests as recommended by Robinson et al³ were performed and included the following:

- 1. **Mapping the injury area:** the skin of the lower left lip and chin in the area supplied by the left mental nerve demonstrated no response to sensory testing. The neuropathic area was mapped and recorded using a ballpoint pen on the patient's face (Fig. 1).
- 2. **Function scoring:** the patient was asked to assess his overall level of sensory function on the affected side, as compared to the contralateral (normal) side, using a

scale ranging from 0 to 10 (0 = noperception of touch and 10 = normal perception). The patient reported 0 on the left side in relation to the lip and chin area.

- Light touch test: the corner of a paper was moved over the injured area and repeated 5 times. The patient reported lack of light touch perception.
- 4. **Sharp/blunt discrimination test:** the patient reported inability to differentiate between the sharp pricks and the blunt pressure made by both ends of a dental explorer.
- Two-point discrimination test: the patient was unable to discriminate the between 2 pressure points (open-beak tweezer) and a single pressure point (closed-beak tweezer).

Radiographic Examination

Digital intraoral radiography revealed incomplete root canal treatment of the left mandibular second premolar, with evidence of missed canal anatomy. A well-defined radiolucency surrounding the apex of the tooth was also observed (Fig. 2A). The patient was referred for a small field-of-view cone-beam computed tomography (CBCT) scan, with a resolution of 200 μ m (Planmeca Promax 3D Max, Planmeca OY, Helsingfors, Finland). The preoperative CBCT scan assisted in the mapping of the complex canal anatomy, confirmed the extent of the periapical lesion and possible communication of the pathosis with the mental foramen (Fig. 3A–C).

Clinical Procedures

Treatment options offered to the patient included either nonsurgical endodontic retreatment (with or without periapical surgery following a period of assessment) or extraction with replacement of the missing tooth. Being motivated to retain the tooth, the patient provided informed consent and



FIGURE 1 – The skin in the region of the left mental nerve distribution (indicated by *white arrows*) presented with paresthesia characterized by numbness to sensory testing. The affected area was marked using a ballpoint pen.

nonsurgical endodontic retreatment was initiated.

Following local anesthesia (Xylotox E80, Adcock Ingram, Johannesburg, South Africa), the tooth was reaccessed under dental dam isolation. Magnification by means of a dental operating microscope (Global Surgical Corporation, St. Louis, MO) was used for all clinical procedures. The majority of existing gutta percha (GP) and sealer were removed using a combination of Gates-glidden #2 and #3, rotary retreatment files (ProTaper D files, DentsplyMaillefer, Ballaigues, Switzerland) and heat pluggers (VDW Beefill, VDW GmbH, Munich, Germany). Patency across the apical foramen was established and working length confirmed using an electronic apex locator (ProPex Pixi, DentsplyMaillefer, Ballaigues, Switzerland) and confirmed radiographically (Fig. 2B).

Canal shaping was performed using a reciprocating instrument (WaveOne Gold primary, DentsplyMaillefer, Ballaigues, Switzerland) and irrigation performed using 3.5% sodium hypochlorite (Jik, Reckitt Benckiser, South Africa). After drying with sterile paper points, a medicated paste (Ledermix, Lederle Pharmaceuticals, Wolfratshausen, Germany) was placed into the canals and the access cavity temporized using glass-ionomer cement (Riva Self Cure, SDI Ltd, Australia).

Two months after the initial visit, the patient reported that the paresthesia in the area remained, but no further discomfort to mastication was experienced. In light of this improvement, the decision was made to complete the root canal treatment. The tooth was reaccessed under dental dam isolation and the intracanal medication removed. The final rinse protocol included 3.5% sodium hypochlorite and 17%

ethylenediaminetetraacetic acid (Topclear EDTA, Dental Discounts, Johannesburg, South Africa), followed by a final rinse of sterile saline and 2% chlorhexidine.

The canals were dried using sterile paper points and obturated using a bioceramic sealer (TotalFill BC sealer, FKG Dentaire, Switzerland) and single matching cones in the apical half (Fig. 2C). The remainder of the canal system in the coronal segment was backfilled using warm GP (VDW BeeFill, VDW GmbH, Munich, Germany). The access cavity was restored using a thin layer of resin-modified glass-ionomer (Vitrebond, 3M ESPE, St. Paul, MN) applied to cover the GP as an intraorifice barrier (for the purpose of providing additional coronal seal). This was followed by etching, application of dentin adhesive (Single Bond Universal, 3M ESPE, St. Paul, MN) and composite resin (Filtek XTE, 3M ESPE, St. Paul, MN) as a final restoration.



FIGURE 2 – (*A*) Periapical radiography revealed a well-defined radiolucency surrounding the apex of the left mandibular second premolar with previous endodontic treatment, involving the mental foramen. (*B*) The existing GP and sealer were removed and working length re-established. (*C*) Bioceramic sealer and single cones were used to obturate the apical half of the canal system. (*D*) The 3-year follow-up periapical radiograph demonstrated good bone healing around the apex of the left mandibular second premolar and complete resolution of the paresthesia. GP, Gutta Percha.

Three-year Recall

Despite scheduling follow-up recalls at 3month intervals after treatment for 1 year (then yearly), the patient could not attend as scheduled due to the Covid-19 pandemic. Three years following nonsurgical retreatment in June 2021, the patient presented for followup reporting no symptoms. At this stage, the paresthesia had subsided completely and normal sensation had returned to the skin innervated by the left mental nerve. Restoration of neurosensory function in the area was confirmed by the same tests performed at the time of case evaluation³. Periapical radiography demonstrated good healing of the periapical periodontitis with new bone formation (Fig. 2D). A follow-up CBCT scan (with the same acquisition parameters, using the same machine) confirmed radiographic healing and the re-establishment of bone in the apical area surrounding the tooth, with adequate hard tissue formation between the apex and the mental foramen (Fig. 3D–F).

DISCUSSION

Paresthesia is defined as a neurosensory alteration resulting from a type of nerve injury called "neuropraxia."⁴ Neuropraxia can be described by patients to include the following symptoms: episodic or continuous tingling, numbness or a prickling sensation, a sensation of unusual cold/warmth, or formication in the affected area which may occur secondary to local or systemic factors^{4,5}.

Paresthesia may occur due to several conditions affecting the peripheral nervous system, including: traumatic injuries, osteomyelitis, viral infections (eg, herpes zoster/shingles), or as a complication of a chronic systemic disease (eg, diabetic neuropathy). Some diseases originating in or affecting the central nervous system, such as stroke or malignancies may also cause paresthesia^{5,6}.

Trigeminal nerve injury in endodontics (presenting as paresthesia) most commonly affects the mandibular division, and has been reported to be caused by a number of factors. These include; primary apical periodontitis/ periapical lesions^{7,8}, sealer extrusion^{9,10}, extrusion of intracanal medicaments^{11,12}, GP overfilling¹³ and inadequate primary endodontic treatment causing persistent apical periodontitis^{1,8,14}, as demonstrated in the present case. The maxillary division of the trigeminal nerve is reportedly less affected than the mandibular division and injury to these structures is usually the result of sodium hypochlorite extrusion¹⁵.

Several mechanisms of injury to the inferior alveolar nerve (IAN) or the mental neurovascular bundle during endodontic procedures have been reported. Albeit uncommon, IAN injury during local anesthetic block injection may occur due to direct mechanical stimulation of the nerve fibers or trauma to the sheath which may lead to pressure on the nerve. Prolonged pressure to the nerve fibers can lead to nerve atrophy¹⁶. A higher incidence of transient or permanent paresthesia has been reported with the use of prilocaine or articaine for mandibular block anesthesia¹⁵. Mechanical injury can be also caused by over-instrumentation; however, it is difficult to determine whether such nerve injury is due to over-instrumentation itself or overfilling, especially if treatment was completed in a single visit.

Chemical injury can result from extrusion of endodontic materials into the mandibular canal. A number of case reports have demonstrated paresthesia due to paraformaldehyde neurotoxicity from materials such as AH26 or AH-plus endodontic sealers^{17,18}. However, many endodontic materials have a high pH, including calcium hydroxide and bioceramic sealers, which could be neurotoxic if extruded in close proximity to a neural structure^{11,12}. Even inert materials, such as GP, could induce nerve injury if extruded into the IAN. In such cases, nerve injury may be due to a combination of direct thermal injury caused by warm GP and/or mechanical compression of the neurovascular bundle^{6,13}.

Periapical inflammation due to infected root canal systems may be a causative factor for paresthesia, both in primary cases as well as failed endodontic treatments. Bacterial toxins (eq, endotoxins of gram-negative bacteria) and metabolic by-products can cause direct nerve injury especially in teeth in close proximity to the inferior alveolar canal or the mental neurovascular bundle¹. Moreover, the inflammatory reaction toward the bacterial challenge may result in mechanical compression and ischemia of the nerve fibers. In the present case, the previously treated mandibular second premolar demonstrated apical periodontitis due to a failure to manage the complex internal anatomy during primary treatment. The missed anatomy was the source of the bacteria responsible for the persistent disease. Left untreated, the periapical lesion extended to involve the mental nerve bundle causing paresthesia.

Surgical treatment was not considered in the present case due to missed anatomy in the root canal system and the proximity of the lesion to the mental nerve. Three-dimensional imaging (CBCT) provided a reliable baseline reference for recalls and the assessment of bone healing. At the time of the 3-year recall, CBCT imaging was requested for the scheduled extraction and implant planning of



FIGURE 3 – Upper panel: CBCT images of the initial visit. (*A*) Sagittal slice showed a hypodense periapical lesion measuring 8.9 by 7.9 mm with close proximity to the mental nerve (*white arrow*). The missed canal is evident on this view. On coronal view (*B*) the lesion was indistinguishable from the mental foramen. (*C*) Three-dimensional reconstruction and nerve mapping showed the close approximation with the periapical lesion and the inferior alveolar and mental nerve. Lower panel: CBCT images of the follow-up visit after 3 years. (*D*) The sagittal slice showed bone healing with the absence of a periapical lesion. On coronal view (*E*), the healing is apparent by cortication and narrowing of the mental foramen (*white arrow*). (*F*) Three-dimensional reconstruction showed the extent of bone fill and healing in the periapical region. CBCT, cone-beam computed tomography.

the left mandibular first molar. This second CBCT scan was beneficial to help demonstrate endodontic healing of the neighboring left second premolar but was not taken primarily for this purpose.

Elimination of bacteria from the previously treated root canal system was performed using strict infection control measures, including: dental dam isolation, thorough debridement of the root canal system, a well-defined irrigation protocol, interappointment medication and proper sealing of the root canal system both apically and coronally. Obturation of the present case was challenging, due to a deep split and complex internal anatomy of the canal system. Bioceramic sealer was chosen to fill these complex intercanal communications, and single matching GP cones were placed in areas where canal shaping with reciprocating instrumentation could reliably be achieved. A dense fill of the main canals was seen, but some voids were present in areas of intercanal communication (Fig. 2*C* and *D*). A small amount of sealer extrusion was furthermore seen apically. Following endodontic retreatment, healing of the apical periodontitis and resolution of the associated nerve injury occurred without the need for surgical intervention.

Whilst it is not known exactly how long after nonsurgical retreatment the paresthesia had resolved, it is known that resolution of this condition may require several weeks, months or even years^{1,19}. Furthermore, mandibular second premolars are known to display complex internal anatomy at times²⁰, contributing to the development of the apical periodontitis in the present case.

In conclusion, long-standing untreated apical periodontitis may result in complications such as paresthesia involving a nearby neural structure. Patients should therefore be informed of this, as well as other possible sequelae of untreated periapical disease. The present case demonstrates that conservative nonsurgical endodontic retreatment may be successful in managing such cases and should be considered by clinicians prior to surgical intervention, especially where clear deficits in the existing endodontic treatment are observed.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Glynn Dale Buchanan: Conceptualization, Methodology, Writing – original draft. Chane Smit: Resources, Writing – review & editing. Mohamed Yasin Gamieldien: Resources, Writing – review & editing. Ahmed S. ElSheshtawy: Writing – review & editing.

ACKNOWLEDGMENTS

The authors deny any conflicts of interest related to this study.

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