LOWER LIP PARESTHESIA IN A PATIENT WITH CARIOUS MANDIBULAR PREMOLAR: A CASE REPORT

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ABSTRACT

Several dental operations, including local anaesthetic administrations, wisdom teeth procedures, orthognathic surgical treatments, ablative operations, implantation, and root canal therapy, can cause numbness and tingling of the inferior alveolar nerve. The present case report explains a case report of lower lip paresthesia arising due to carious mandibular premolar.

KEYWORDS: Mandibular Premolar, Caries, Paresthesia, Lower Lip

INTRODUCTION

A sensory impairment known as paresthesia is characterized by clinical signs like burning, tickling perception, stinging, numbing, itching, and any other change from such a healthy experience.⁰ Though atypical, paresthesia is not agonizing, per the International Association of Study of Pain (IASP) classification.¹ Several dental operations, including local anaesthetic administrations, wisdom teeth procedures, orthognathic surgical treatments, ablative operations, implantation, and root canal therapy, can cause numbness and tingling of the inferior alveolar nerve.² An important morphological component of the dorsal aspect of the mandible is the mental foramen, which is important in dentistry. The mandibular canals, which come in such an inclined manner to the surfaces, end here. The mental nerve bundles travel through into the mental foramen and provide nourishment and sensory stimulation to the gums, jawline, and lower lip tissues upon the mandible’s ipsilateral region of the mandible.³ The mental foramen might lead to a false-positive radiological diagnostic of a radiolucent pathology within the lower premolar teeth’s apex region. It’s crucial to locate the mental foramen precisely for diagnosing and therapeutic treatments.³ Moreover carefully considered should be the close anatomic vicinity of the mental nerve bundle and the root apex of the lower premolars. It is also well-known that pre- and post-endodontic periapical disease can cause inferior alveolar nerve paresthesia.⁴ Paresthesia brought on by periapical pathogens may result from pressure from inflammatory oedema pressing against the mental nerve. A suppurative discharge builds up in the mandibular bone due to the periapical inflammatory activity, which releases proinflammatory agents as a byproduct of tissue injury and hazardous metabolized byproducts of microorganisms. The ensuing hematoma or related oedema may strain the nerve endings, resulting in paresthesia sensations.⁵ Paresthesia could last anywhere between a few days to many weeks or months; in certain circumstances, it may even last forever. Cases of irreparable nerve injury that may be caused by laceration, sustained strain over the nerve, or exposure to hazardous overfilled endodontic substances, can lead to chronic paresthesia.⁶ The present case report explains a case report of lower lip paresthesia arising due to carious mandibular premolar.

CASE

A 50-year-old female presented to the clinic with pain in her mandibular second premolar. Intro-oral examination revealed that the patient had a carious mandibular second premolar, as shown in Figure 1.
The cavity preparation was done, and there was a pin point pulpal exposure during excavation. Calcium hydroxide was placed, and the tooth was restored with temporary GIC filling, as shown in Figure 2.

On the third day, a dressing of calcium hydroxide was placed. In the following three days, the lip paresthesia was unresolved and spread up to the midline of the chin. On the fourth day, the paresthesia started resolving, but the patient experienced a lacerating pain in her lip which continued for two days. During this time, the patient also reported that she experienced sensitivity to air to the lip’s skin. After two days, the paresthesia and the lacerating pain resolved greatly and were limited to a small part of the lip near the corner of the mouth. The patient was called for a follow-up exam, and a radiographic examination revealed the radiolucency to have undergone a slight shrinkage. Another dressing of cryosphere was placed, and the patient was recalled after three days. On the third day, the lip paresthesia had resolved greatly. The root canal was obturated, and a tooth restoration was placed, as shown in Figure 4.
The patient was recalled for follow-up after one month, and she reported that the lip numbness had resolved to a moderate degree, but lip paresthesia persisted. The patient was identified after three months and wrote a complete resolution of numbnness but paresthesia persisted moderately. Regular follow-up sessions were conducted for the patient, and she reported that after one year, the paresthesia had resolved to a great degree, but a mild electric shock-like sensation persisted on consuming a cold beverage in the lip.

**DISCUSSION**

A typical indication of insult to, discomfort of, or disruption to the inferior alveolar nerve or even its mental branches is numbness of the lower jaw. Patients typically report a unilateral impairment of responsiveness in the lower lip and gums and numbing, stinging, and dehydration of the afflicted tissues. It frequently begins with excruciating discomfort and a burning feeling in the damaged region. In the domain of dentistry, paresthesia may be caused by a vast range of systemic and local potential causes, including severe trauma like maxillofacial cracks, widening and compressive growths (cysts and malignant or benign cancers), impacted teeth, local diseases (osteomyelitis and periapical lesions), iatrogenic injury after dental surgery, infusion of anaesthetic, and endodontic therapy. Approximately 48% of incidences of facial paresthesia have indeed been linked to an oral operation or disease, according to reports, with 83% of instances having an established aetiology. The nerves that are affected the most frequently are the inferior alveolar and mental nerves. According to a study the perception abnormalities of the mandibular premolars have a 0.96% occurrence estimate. There are 3.7 mm and 6.9 mm between the upper boundary of the mandibular canals and the apex of both the second molar and the tip of the mesial root with the first molar, accordingly. Due to this proximity, the inferior alveolar nerve endings may be affected by periapical illnesses. Localized stress upon that mental nerve brought on by deposition of abscess oedema inside the mandible bone, mechanical stress and hypoxia in inflammatory mechanisms (oedema), microbial toxicants or inflammatory items produced from damaged cells may lead to periapical abnormalities, and eventually paresthesia. The primary pathogenic factor for neurological illnesses has only been identified in a few percentages of articles. The pulp has been diagnosed in the majority of the occurrences, and radiological analysis indicated a periapical disease. The teeth implicated in the scenario, as is now, had root canal therapy in the past and also had periapical diseases. Neuropraxia may result from a periapical infection brought on by infected root canals because of hyperemia and reactive oedema that pressures the nerve and damages the sensory components. Additionally, hypoxia may result in nerve malfunction, which is connected to the paresthesia complaints and manifestations. Additionally, the endotoxins and biochemical byproducts of Gram-negative microorganisms may have toxic consequences. However, another mechanism that could harm the sensory systems is the straight bacterial infiltration of those components. Infection-induced paresthesia is typically resolved after inflammation and infections are treated through prophylactic antibiotics, endodontic treatments, periapical resection, or dental surgery, infection-induced paresthesia is typically resolved. The teeth are associated with having undergone root canal re-treatment since it is a conservative therapeutic option and also can remove the pathophysiological agent by discarding the intra-canal illness, enabling the rejuvenation of the mental nerve and periodontal cells.

**CONCLUSIONS**

Neuropraxia may result from a periapical infection brought on by infected root canals because of hyperemia and reactive oedema that pressures the nerve and damages the sensory components. The pathogenic element for the given scenario has been the periapical disease of a mandibular second premolar. Additionally, if this course of therapy doesn’t work, there are still additional possibilities, such as periapical resection or tooth extraction.

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**REFERENCES**


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