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Persistent paresthesia of inferior alveolar nerve after accidental extrusion of calcium hydroxide paste containing iodoform into the mandibular canal

KEYWORDS

Paresthesia;
Inferior alveolar nerve;
Mandibular canal;
Calcium hydroxide paste

The inadvertent displacement of endodontic materials into the mandibular canal (MC) can lead to inferior alveolar nerve paresthesia (IANP).¹ While the majority of cases (91%) show either full or partial recovery over time, occurrences of permanent paresthesia are rare.¹ Prognostic factors encompass the tooth location, the types of extruded materials, the obturation techniques, and post-injury treatments.¹ This article presented a case involving persistent IANP resulting from the extrusion of calcium hydroxide paste containing iodoform (CHI) into the MC.

A 46-year-old female patient presented with a chief complaint of spontaneous pain in tooth #37 for one week. The tooth exhibited heightened sensitivity when subjected to a cold pulp test and was accompanied by persistent, severe pain. Periapical radiograph revealed a substantial caries lesion affecting the pulp chamber of tooth #37. The diagnosis for tooth #37 was symptomatic irreversible pulpitis. Root canal treatment was proposed, and the patient provided written informed consent. Following the delivery of CHI into the canals for intracanal medication, the patient immediately complained of paresthesia in the left lower lip and chin area. The CHI inside the canals was removed by irrigation with physiological saline solution, and the tooth was restored with temporary filling material. Cone beam computed tomography (CBCT) images showed the presence of radiopaque material in the periapical area of tooth #37,

spreading around and within the MC (Fig. 1A and A1-A5). The patient was informed about the occurrence and potential complications of the extrusion of CHI into the MC. Two treatment options were presented: (1) Conservative treatment with neurotrophic drug; (2) Extraction of tooth #37 and surgical removal of CHI. Opting for the former, the patient was prescribed with Mecobalamin. At the 2-week follow-up, tooth #37 was asymptomatic. The patient reported the reduced paresthesia in the left lower lip and chin area. Clinical examinations showed no signs of infection, such as percussion or palpation tenderness, mucosa swelling or redness in tooth #37. The patient declined to complete root canal treatment and the final restoration of the tooth. During the 10-month follow-up, tooth #37 remained asymptomatic and functional. The patient reported significant recovery from paresthesia in the left lower lip and chin area. Despite this improvement, the patient still declined to complete root canal treatment and required temporary filling for tooth #37. CBCT images revealed a reduced amount of radiopaque material in the periapical area of tooth #37 and the MC (Fig. 1B and B1-B5). At the 16-month follow-up, the patient reported further resolution of paresthesia in the left lower lip and chin area.

The prevention of IANP is prioritized through preoperative assessment of the relationship between root apices and the MC.^{1–5} Early diagnosis and timely, appropriate

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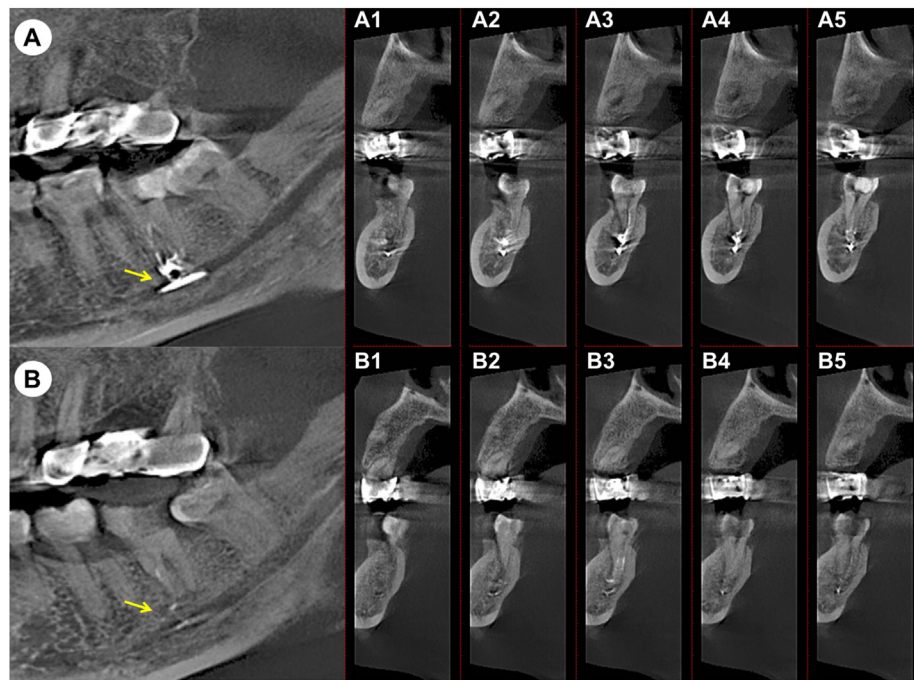


Figure 1 Coronal (A) and sagittal (A1–A5) sections of cone beam computed tomography (CBCT) images showed the presence of radiopaque material in the periapical area of tooth #37, spreading around and within the mandibular canal (yellow arrow). At the 10-month follow-up, coronal (B) and sagittal (B1–B5) sections of CBCT images revealed a reduced amount of radiopaque material in the periapical area of tooth #37 and the mandibular canal (yellow arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

interventions remain crucial.^{1–5} There is no consensus regarding the indications for conservative versus surgical IANP treatment. For this kind of case, high-dose steroids for 1–2 weeks are the first-line therapy. Among reported cases, the recovery rate for nonsurgically treated cases (63%) exceeded that of surgically treated cases (46%).¹ Complete sensory recovery cannot be guaranteed with surgical intervention. There is a potential risk of secondary nerve damage associated with such treatments.¹

Declaration of competing interest

The authors have no conflicts of interest relevant to this article to declare.

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