Effect of Calcium Hydroxide/Iodoform Paste on Necrosis of Gingiva : A Case Report

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Abstract

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Case Report

The present case demonstrates the possible detrimental effect of an overextension of a calcium hydroxide/ Iodoform paste in intracanal dressing into the periradicular and soft tissue after iatrogenic buccal root perforation of a maxillary central incisor. Initially this perforation was not recognized by the dentist, which resulted in the introduction of a large amount of nonsetting calcium hydroxide/Iodoform paste under the gingival tissues through a dehiscence on the buccal side of the root. This report describes the consequences and management of the necrosis of the buccal gingiva and mucosa, and the subsequent treatment and follow-up of the root perforation.

Key Words: Glass ionomer cement, calcium hydroxide/Iodoform paste, necrosis, perforation, surgical repair.

Introduction

Since its introduction in dentistry, calcium hydroxide Ca (OH) 2 /Iodoform paste has been used for a wide variety of purposes including lining of cavities, indirect and direct pulp capping, dressing after pulpotomy, dressing of the root canal between appointments, prevention of root resorption, repair of iatrogenic perforations, treatment of horizontal root fractures and as a constituent of root canal sealers (Foreman & Barnes 1990). Few reports deal with the negative side-effects of Ca(OH)2, that include bone necrosis and continuing inflammatory response in repaired mechanical perforations (Himel et al. 1985), the neurotoxic effect of root canal sealers (Boiesen & Brodin 1991), cytotoxicity on cell cultures, damaged epithelium with or without cellular atypia when applied on hamster cheek pouches (Dunham et al. 1966) and cellular damage following early calcium hydroxide _dressing of avulsed teeth (Andreasen & Kristerson 1981). The following report describes the deleterious effects of Ca (OH) 2 in a case of iatrogenic root perforation.

Case Report

A 25-year-old male was referred by his dental practitioner to the emergency clinic for further endodontic treatment of his maxillary left central incisor. The patient presented with a swollen upper lip and, on clinical inspection, an extensive necrotic zone on the buccal side of 21 and 11 was noted (Fig. 1).

Tooth 21 was not painful on percussion. From the dental history it was clear that the practitioner had commenced the root canal treatment 1 week earlier. Since the pulp chamber and the coronal part of the root canal were obliterated with dentine, the dentist had difficulty locating the canal. Various radiographs were taken but these did not show any evidence of incorrect angulations of the file compared with the long axis of the tooth and the Mesiodistal location of the root canal (Fig. 2). A provisional root canal preparation was performed, using 2.5% NaOCl as a canal irrigants. No intracanal dressing was placed. In a second session the final root canal shaping was done. As it was difficult to obtain a dry canal, an inter appointment calcium hydroxide dressing was placed, Control; pH = 12, unfortunately, the radiograph of this dressing (Fig. 3) revealed a large amount of material extruded through the root. One day later, the upper lip was swollen and 2 days later the gingiva showed sudden perforation in this area. The patient experienced no pain. For 2 weeks the necrotic gingival zone was treated with rinses of hydrogen peroxide 3% and Chlorhexidine 2% in water once every 2 or 3 days. During this time a regimen of daily application (BID) of Chlorhexidine digluconate 10 mg g1 gel was also prescribed. The gingival perforation healed and closed completely within 2 months (Fig. 4). Two millimeters of gingival recession measured from the cemento-enamel junction resulted. A surgical endodontic intervention was planned, but postponed by the patient for 6 months. At this surgical treatment session all temporary filling material in the access cavity was removed. As the introduction of a file in the apical part of the root canal was complicated by the palatal inclination of the tooth crown and the presence of an extensive mid root ledge, it was decided to combine surgery and root canal preparation in one session. (Fig. 1, 2 & 3)

Under local anesthesia a full thickness flap was raised. Following flap reflection, a pronounced bony Dehiscence was observed. The buccal side of the midroot had been perforated. The buccal opening in the root facilitated the visualization of the location of the opening of the apical part of the root canal. After cleaning, shaping and root canal obturation with cold lateral gutta percha condensation and AH26,the buccal perforation was closed with glass ionomer cement (Figs 4 and 5). At this time scar tissue from the healing soft tissues and the periosteum were cut through to mobilize the flap. The root surface was conditioned with saturated citric acid (pH = 1) for 1 min.

The flap was repositioned coronally and closed with sutures. Compression of the flap was carried out and the access cavity filled with Ketac-Fil glass ionomer cement . Ten days later sutures were removed and the surgical area cleaned; healing was uneventful. At the three-month recall the tooth was free of clinical symptoms and the patient had no complaints. The buccal gingival recession measured from the cementoenamel junction was limited to 1 mm. At 6 months the maximum probing depth was 3 mm, indicating that the periodontal intervention had resulted in a new attachment over the treated root. Radiographic examination revealed a normal status (i.e. absence of any periapical or periodontal pathology) and the gingival recession remained stable. (Fig. 4, Fig. 5)

Discussion

The initiation and stimulation of mineralization, the antibacterial characteristics and the dissolution of necrotic material are the main indications for the extensive use of Ca (OH)2 and Iodoform paste in dentistry. These properties are mainly correlated to the high pH; however, not only these positive effects can occur from its necrotizing capacity. Following use of Ca(OH)2, Sahli (1988), Sahli (1990a), Sahli (1990b) described the destruction of epithelium present in periradicular lesions, allowing connective tissue invagination and healing. In this situation the necrotizing ability resulted in a positive outcome. Dunham et al. (1966), however, investigated the effect of repeated applications of Ca(OH)2 on the epithelium of hamster cheek pouches. After the initial alkaline burn the lesions progressed, some of them showing distinct cellular atypia. AlacËam et al. (1993) found that Ca (OH)2 had a cytopathological effect on vital cells comparable to that of NaOCl. On the other hand, Fava (1993) described a case of overfilling of the palatal root canal of a maxillary right first premolar leading to Ca(OH)2 in the maxillary sinus. The patient experienced some pain, but 4 days later the symptoms had decreased to a minimum. At the three-month recall the tooth was symptom-free and showed normal periapical tissues, even though a radiopaque

mass was visible in the sinus. Of course, it has been reported that Ca(OH)2 inadvertently forced through the apical foramen is absorbed (Foreman & Barnes 1990) with the result that Ca(OH)2 and Iodoform paste placement beyond the confines of the root canal and into the periradicular lesion has been advocated (Maalouf & Gutmann 1994). Gordon et al. (1981) described the tissue dissolving and necrotizing capacities of NaOCl, which might have contributed to the lesion described in this report. However, the lesion presented only after the second appointment, the time the Ca (OH)2 had been placed into the root canal.

From these observations it can be concluded that as long as Ca (OH)2 and Iodoform paste does not come into direct contact with surrounding soft tissues, problems either do not occur or they are of a mild, transient nature. In the present case however, no buccal bony plate was present and the Ca (OH)2 came into direct contact with the periodontal and gingival tissues. Therefore, in such cases, instead of awaiting the consequences, it would be appropriate to remove the extruded material immediately in order to avoid an alkaline burn and so eliminate extensive tissue damage.

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Legends

Fig. 1 Necrotic gingiva extending from tooth 21 to 11.
Fig. 2 Radiographs showing no evidence of incorrect angulations of the file.
Fig. 3 Radiograph of the interappointment calcium hydroxide/Iodoform paste.
Fig. 4 Closure of the perforation with Glass ionomer cement
Fig. 5 Radiograph of the completed root filling.


Fig. 1


Fig. 2


Fig. 3


Fig. 4


Fig. 5

FORM-IV

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