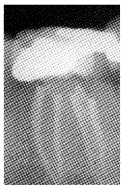
doi:10.1111/j.1365-2591.2007.01340.x



CASE REPORT

Gingival and bone necrosis caused by accidental sodium hypochlorite injection instead of anaesthetic solution

F. Pontes[1], H. Pontes[2], P. Adachi[1], C. Rodini[1], D. Almeida[2] & D. Pinto Jr[1]

[1]Faculdade de Odontologia da Universidade de São Paulo-USP, São Paulo, SP; and [2]Faculdade de Odontologia da Universidade Federal do Pará, Belém, Pará, Brazil

Abstract

Pontes F, Pontes H, Adachi P, Rodini C, Almeida D, Pinto D Jr. Gingival and bone necrosis caused by accidental sodium hypochlorite injection instead of anaesthetic solution. *International Endodontic Journal*, **41**, 267–270, 2008.

Aim To report clinical complications (pain, necrotic gingival tissue and bone sequestration) resulting from accidental injection of sodium hypochlorite.

Summary Root canal treatment is a routine clinical procedure with few reported complications. Sodium hypochlorite (NaOCl) is commonly used as an irrigant during the procedure because of its tissue-dissolving, antibacterial and lubricating properties. This paper presents a case in which accidental injection of sodium hypochlorite into the lingual gingiva of a female patient caused gingival and bone necrosis. Surgical intervention was required.

Key learning points

- Sodium hypochlorite is dangerous if injected into the tissues.
- The presentation of sodium hypochlorite in glass, anaesthetic type cartridges is potentially dangerous, and should be condemned.
- All healthcare workers should check carefully the contents of any syringe before injecting into patients.

Keywords: bone necrosis, gingival necrosis, sodium hypochlorite.

Received 13 April 2007; accepted 9 August 2007

Introduction

Sodium hypochlorite (NaOCl) is the irrigant most widely used in endodontic treatment. It is effective against a broad spectrum of microorganisms, dissolves necrotic tissues, and has lubricating properties (Kaufman & Keila 1989, Ehrich *et al*. 1993, Gursoy *et al*. 2006).

Correspondence: Patricia Leite de Godoi Adachi, Disciplina de Patologia Bucal, Faculdade de Odontologia da Universidade de São Paulo, Av: Prof. Lineu Prestes, 2227 Cidade Universitária, São Paulo, Brazil (Tel.: +55 11 30917902; fax: +55 11 30917894; e-mail: paricardo@usp.br).

However, it is also toxic to vital tissues, causing haemolysis, ulceration, inhibition of neutrophil migration, damage to endothelial and fibroblast cells, facial nerve weakness and necrosis following extrusion during root canal treatment (Gatot *et al.* 1991, Hülsmann & Hahn 2000, Gernhardt *et al.* 2004, Witton & Brennan 2005, Witton *et al.* 2005). This report presents a case in which NaOCl was inadvertently injected instead of local anaesthetic solution, resulting in severe gingival necrosis and bone sequestration.

Case report

A 35-year-old woman with noncontributory medical history was referred to the Oral Pathology Service of João de Barros Barreto University Hospital, Belém, State of Pará, Brazil, suffering from mucosal necrosis of the lingual gingiva adjacent to tooth 37 (FDI). Two months before referral, she had attended a private dentist with the chief complaint of toothache in the mandibular left first molar. A clinical diagnosis of irreversible pulpitis was made and pulpectomy was performed under local anaesthesia. Treatment was continued on two further sessions. During the final session for root canal filling, the dentist decided to apply local anaesthesia only in the lingual region around teeth 35–37 before placing rubber dam. However, instead of anaesthetic solution, 1% NaOCl (Biodinâmica LTDA, Paraná, Brazil) was injected into the tissues.

This mistake occurred because both solutions were dispensed in identical containers, and because the dentist had not carefully checked before injecting. During application of the NaOCl solution, the patient reported severe sudden pain. This procedure was immediately ceased and the dentist recognized the mistake at that time. Anti-inflammatory (diclofenac, 50 mg thrice per day for 3 days) and antibiotic drugs (amoxicillin 1500 mg thrice per day for 10 days) were prescribed. The pain worsened significantly over the following 24 h and continued for 3 days, after which it subsided.

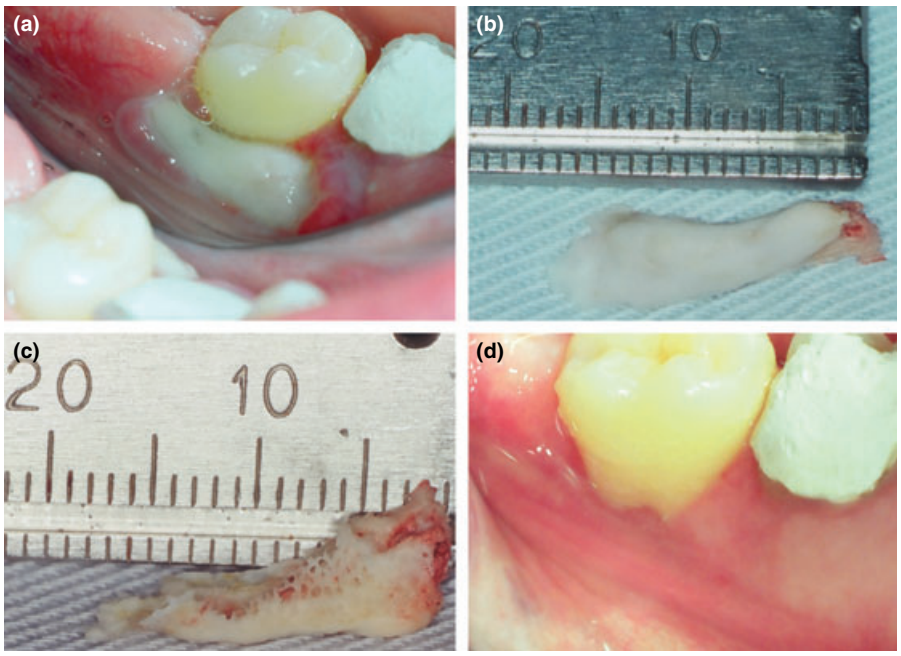


Figure 1 Clinical view of the lesion (a), gingival tissue (b), bone sequestration (c), removed during surgery, and clinical view 20 days after surgery (d).

On clinical examination approximately 60 days after the incident, there was marked necrosis of the lingual mucosa around tooth 37 (Fig. 1a), but the patient was pain free. Surgical coverage with a laterally positioned flap was performed using the mucosa on the retromolar region as the donor area. During the surgical procedure, the mucosa and bone in the affected area were easily detached (Fig. 1b,c), as a result of the formation of bony sequestration. Three weeks later, the area affected by chemical necrosis had healed and there was no evidence of tissue necrosis (Fig. 1d). The root canal treatment of the tooth 36 was completed 30 days after the surgical procedure.

Discussion

Sodium hypochlorite is an effective endodontic irrigant solution in a variety of concentrations, ranging from 0.5% to 6% (Hülsmann & Hahn 2000, Zehnder *et al*. 2002). However, NaOCl may cause hypersensitivity (Kaufman & Keila 1989), as well as serious complications after inadvertent use. The most common complication is accidental injection into the periapical tissues in teeth with immature apical foramina or when the apical constriction has been destroyed during root canal preparation or by resorption (Sabala & Powell 1989, Hülsmann & Hahn 2000, Witton & Brennan 2005). The immediate sequelae of these accidents include severe sudden excruciating pain of 2–5 min duration, probably related to tissue destruction and distention, as well as immediate swelling of the tissue in the area. Several days of increasing oedema and ecchymosis occur, accompanied by tissue necrosis and paraesthesia, in addition to secondary infections and possible obstruction of the airway from oedema in some cases (Sabala & Powell 1989, Gatot *et al*. 1991, Hauman *et al*. 2002). The caustic effects of NaOCl occur because of its alkalinity (pH 10.8–12.9) and hypertonicity that causes injury primarily by oxidation of proteins (Kaufman & Keila 1989, Gatot *et al*. 1991, Serper *et al*. 2004). Heling *et al*. (2001) reported that NaOCl concentrations above 0.01% are lethal to fibroblasts *in vitro.*

Management of complications related to NaOCl have been described (Hülsmann & Hahn 2000, Halles *et al*. 2001). Initially, the swelling should be treated by cold compresses. After 1 day, these should be replaced by warm compresses and warm mouthrinses to stimulate local microcirculation. Hospital admission and aggressive supportive measures must be considered in cases of unfavourable clinical outcome. The patient should be informed that healing will take some days or even weeks, and that symptoms resolve completely in most cases (Kaufman & Keila 1989, Hauman *et al*. 2002, Witton & Brennan 2005).

Surgical intervention depends on the nature and severity of the incident. To reduce the acute pain, local anaesthesia may be helpful along with the prescription of analgesics. The use of antibiotics is routinely recommended in these incidents, because of the presence of necrotic tissue and the risk of infection. Intravenous steroids, although not used in this case, have also been recommended (Hülsmann & Hahn 2000, Witton & Brennan 2005).

The appearance in this case was consistent with severe tissue damage associated with chemical necrosis following injection of 1% NaOCl into the gingiva and alveolar bone. Approximately 0.6 mL of NaOCl was injected. Some clinicians unwisely use empty anaesthetic carpules to store their irrigating solutions, in the belief that the long fine needles are helpful. The present case, however, shows that this practice is potentially dangerous. Therefore, it is strongly recommend that clinicians do not use NaOCl supplied in local anaesthetic delivery devices or unclearly labelled cartridges and carefully check all solutions before injecting patients. Fortunately, no clinical symptoms remained in this patient.

Disclaimer

Whilst this article has been subjected to Editorial review, the opinions expressed, unless specifically indicated, are those of the author. The views expressed do not necessarily represent best practice, or the views of the IEJ Editorial Board, or of its affiliated Specialist Societies.

References

Ehrich DG, Brian JD Jr, Walker WA (1993) Sodium hypochlorite accident: inadvertent injection into the maxillary sinus. *Journal of Endodontics* **19**, 180–2.

Gatot A, Arbelle J, Leiberman A, Yanai-Inbar I (1991) Effects of sodium hypochlorite on soft tissue after its inadvertent injection beyond the root apex. *Journal of Endodontics* **17**, 573–4.

Gernhardt CR, Eppendorf K, Kozlowski A, Brandt M (2004) Toxicity of concentrated sodium hypochlorite used as an endodontic irrigant. *International Endodontic Journal* **37**, 272–80.

Gursoy UK, Bostanci V, Kosger HH (2006) Palatal mucosa necrosis because of accidental sodium hypochlorite injection instead of anaesthetic solution. *International Endodontic Journal* **39**, 157–61.

Halles JJ, Jackson CR, Everett AP, Moore SH (2001) Treatment protocol for the management of a sodium hypochlorite accident during endodontic therapy. *General Dentistry* **49**, 278–81.

Hauman CH, Chandler NP, Tong DC (2002) Endodontic implications of the maxillary sinus: a review. *International Endodontic Journal* **35**, 127–41.

Heling I, Rotstein I, Dinur T, Szwec-Levine Y, Steinberg D (2001) Bactericidal and cytotoxic effects of sodium hypochlorite and sodium dichloroisocyanurate solutions *in vitro*. *Journal of Endodontics* **27**, 278–80.

Hülsmann M, Hahn W (2000) Complications during root canal irrigation – literature review and case reports. *International Endodontic Journal* **33**, 186–93.

Kaufman AY, Keila S (1989) Hypersensitivity to sodium hypochlorite. *Journal of Endodontics* **15**, 224–6.

Sabala CL, Powell SE (1989) Sodium hypochlorite injection into periapical tissues. *Journal of Endodontics* **15**, 490–2.

Serper A, Ozbek M, Calt S (2004) Accidental sodium hypochlorite-induced skin injury during endodontic treatment. *Journal of Endodontics* **30**, 180–1.

Witton R, Brennan PA (2005) Severe tissue damage and neurological deficit following extravasation of sodium hypochlorite solution during routine endodontic treatment. *Bristish Dental Journal* **198**, 749–50.

Witton R, Henthorn K, Ethunandan M, Harmer S, Brennan PA (2005) Neurological complications following extrusion of sodium hypochlorite solution during root canal treatment. *International Endodontic Journal* **38**, 843–8.

Zehnder M, Kosicki D, Luder H, Sener B, Waltimo T (2002) Tissue-dissolving capacity and antibacterial effect of buffered and unbuffered hypochlorite solutions. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* **94**, 756–62.

