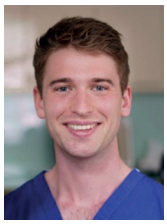
Enhanced CPD DO C



Thomas Hennebry

Harpal Chana

Management of Root Perforation and Sodium Hypochlorite Extrusion Injury

Abstract: A patient was brought to A&E by their GDP who had injected sodium hypochlorite (NaOCl) through a perforation in the patient's LR4, resulting in pain, swelling and immediate formation of a large, necrotic ulcer. Within 2 days, the patient developed paraesthesia in the distribution of the right mental nerve. Antibiotics and steroids were prescribed to alleviate the acute symptoms. The perforation was repaired with mineral trioxide aggregate (MTA) and root canal treatment was completed with the aid of a microscope. Review appointments were arranged to monitor healing of the injury.

CPD/Clinical Relevance: Management of iatrogenic root perforation and associated NaOCl injury is useful knowledge.

Dent Update 2023; 50: 107–112

Sodium hypochlorite (NaOCl) is usually encountered as a dilute solution and has been used as household bleach since the 18th century. It has a pH between 11 and 12. Owing to its bactericidal properties and its ability to dissolve organic matter, it has become the most widely used irrigant in endodontics and is typically found in preparations of between 0.5% and 6% concentration.[1,2] However, care must be taken over its safe use because it is highly caustic to the tissues of the human body and related injuries are a well-documented risk. When NaOCl solution is extruded beyond the root canal system, either through the apical foramen or through a root perforation, it causes damage to organic structures and results in an intense inflammatory response. Scarring, deformation of soft tissues and nerve damage can result and these may be permanent.[3–6]

It is vital that dentists carrying out root canal treatment are well informed of the risks associated with using NaOCl and are confident in managing NaOCl-related injuries. The authors present their management of one such case, along with clinical photographs and radiographs.

History and examination

A 33-year-old female presented to the A&E department, accompanied by her general dental practitioner (GDP) with a large, painful swelling on the right side of her mandible. Medically, the patient was fit and well, took no regular medication and had no known allergies. She gave consent to the taking of clinical photographs and the use of her radiographs in writing up this case report.

The patient had been undergoing the first stage of root canal treatment on her LR4. Root canal treatment was indicated for LR4 as the patient had persistent discomfort from the tooth and tenderness to percussion. A diagnosis of symptomatic apical periodontitis associated with a non-vital LR4 was made. During irrigation of the root canal system with NaOCl solution, the patient complained of severe pain. The GDP suspected an iatrogenic perforation had occurred and removed the rubber dam to examine the tissues surrounding LR4. He attempted to manage the situation by irrigating the access cavity of LR4 with saline and applying cold compression to the patient's face. The tooth was not temporized before bringing the patient to hospital.

Thomas Hennebry, BDS, PgCert Dip, MSc, MFDS RCS Eng, Dentist with special interest in Endodontics, Kingston Hospital and Elmfield House, Teddington. **Harpal Chana,** BDS, MSc, FDS (Rest Dent) RCS, Consultant in Restorative Dentistry and Dental Implantology, Specialist in Restorative Dentistry, Endodontics, Periodontics and Prosthodontics, Kingston Hospital and Elmfield House, Teddington.
email: thomas.hennebry2@nhs.net

Figure 1. Extra-oral view of the patient's face, showing the swelling on the right side.

a

b

Figure 2. (a,b) Intra-oral views of LR4 and its adjacent lesion of necrosed tissue.

On examination, extra-orally there was a facial swelling localized around LR4, extending to the angle of the mandible on the right side. In addition, the right submandibular lymph node was tender to palpation. There was normal mouth opening. Intra-orally, there was a large ulcer forming adjacent to LR4 and a dark-coloured area of necrotic tissue in the buccal sulcus. There was no sinus tract visible. The endodontic access cavity in LR4 was open and the tooth was otherwise restored with the remains of an occlusal amalgam. Clinical photographs were taken (Figures 1 and 2).

The patient was reviewed the following day, where it was found that the extra-oral swelling had increased in size. Pain was still present but the patient reported it as 'manageable'. The patient was reviewed again 48 hours after the original injury. This time she reported the pain had gone but instead, she now had paraesthesia in of the right side of her lower lip and chin (distribution of the right mental nerve) (Figure 3).

	LR1	LR2	LR3	LR4	LR5	LR6
Tender to percussion?	-	-	-	++	-	-
Mobility	-	-	-	-	-	-
Ethyl chloride sensitive?	+	+	+	-	+	+

Table 1. Summary of special tests carried out as part of endodontic examination.

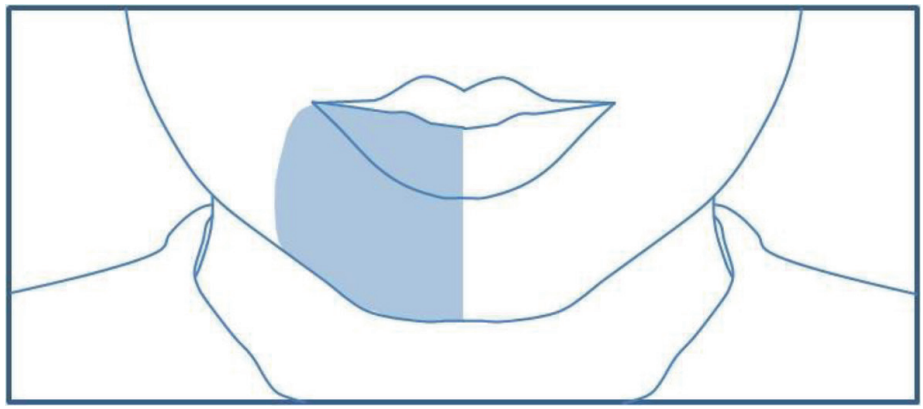


Figure 3. Neurosensory map of the affected area of hypoaesthesia.

Special investigations

The special investigations are summarized in Table 1. Neurosensory testing was conducted to assess the nature, severity and extent of the paraesthesia. A Boley gauge was used to test for two-point discrimination. On the unaffected left side of the lower lip and chin, the patient could discriminate between points 3 mm apart, which is considered normal. On the right, the value was 14 mm. Light touch sensation was tested using a cotton tip applicator and a sterile needle was used to test nociception. Both were found to be reduced (hypo-aesthesia) in the distribution of the right mental nerve (Figure 3).

A cone beam computed tomography (CBCT) scan with a limited field of view was taken to show LR4 (Figures 4–6). It confirmed the presence of a perforation exiting LR4 at the level of the crestal bone distobuccally. It demonstrated that the access cavity did not communicate with the pulp chamber, which was located centrally. The pulp chamber was entirely below the level of the alveolar bone, indicating some level of sclerosis, which may explain why this tooth was at higher risk of perforation. Only one, central root canal was present.

The CBCT showed bone loss at the point where the perforation exited the tooth and another radiolucent area in the soft tissue buccal to LR4 where NaOCl had entered the buccal sulcus. The radiolucency resulted from a fluid-filled space within the soft tissue.

Immediate management

The focus in immediate management of the injury was to limit the caustic effects of the NaOCl and to prevent an excessive immune response in the damaged tissues. It began when the patient's GDP irrigated the suspected perforation with saline and applied cold compression to the patient's face. The objective of saline irrigation was to dilute the strength of the NaOCl as it has been demonstrated *in vitro* that cytotoxicity of NaOCl is greater at higher concentrations.[7] Dilution also neutralizes the highly alkaline pH, which contributes to its penetrating capabilities and thus its damaging effects on vital tissues.[8–10] Cold compression is a commonly employed method to reduce tissue swelling and pain because it promotes vasoconstriction and therefore limits the accumulation of tissue fluid and the influx of pro-inflammatory cells and cytokines.

After assessment at hospital, the patient was prescribed a 5-day course of amoxicillin and metronidazole to prevent infection in the necrotic tissue. She was also prescribed dexamethasone (10 mg once/day) for 1 week, followed by a gradual decrease in strength. The purpose of the steroid

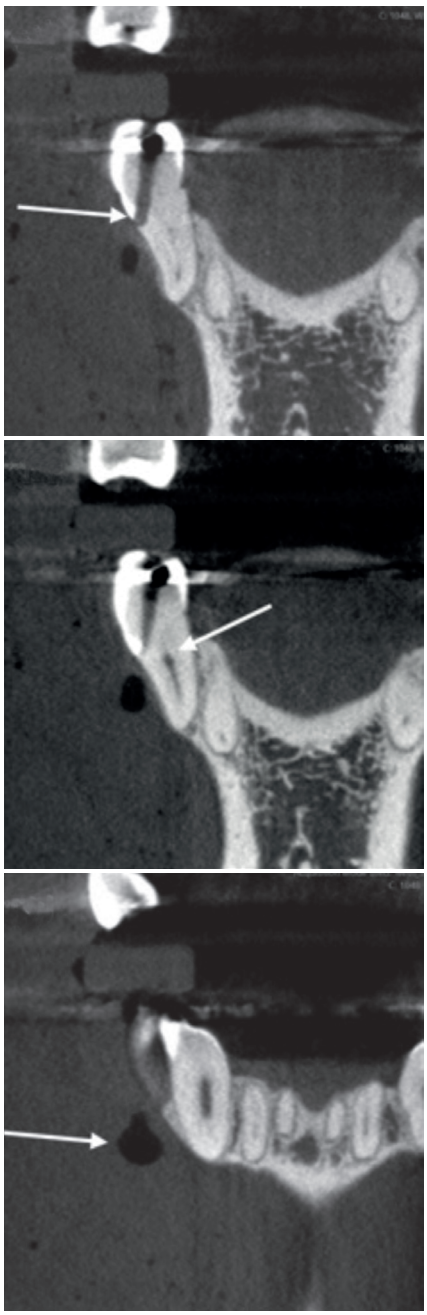


Figure 4. Coronal views of the perforation in LR4 and the adjacent area of radiolucency in the soft tissue. **(a)** The line of access is angled buccally within the tooth's crown and exits at the level of the alveolar bone. **(b)** The pulp chamber is below the level of the alveolar bone. The most coronal point of the pulp chamber is indicated with the arrow. It has sclerosed, making it deeper than might usually be anticipated. **(c)** The arrow points out the radiolucent, fluid-filled space in the soft tissue at its widest point in the coronal plane.

prescription was to reduce the patient's inflammatory response to the NaOCl because excessive inflammation leads to greater pain and swelling.[6]

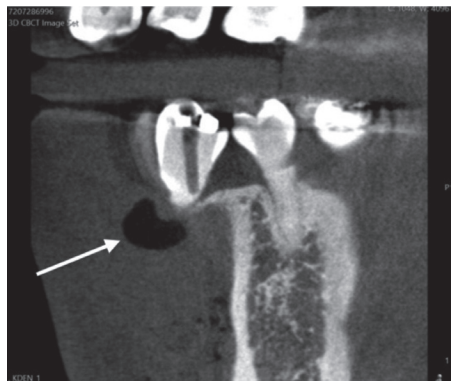


Figure 5. Sagittal view of LR4, which shows the path of the perforation is angled distally within the crown of the tooth. The radiolucent, fluid-filled space in the soft tissue (indicated with a white arrow) is below the alveolar crest.

Monitoring of healing

Review appointments were arranged at 24 hours, 48 hours, 1 week, 2 weeks, 6 weeks and 4 months. Follow-up is vital in cases of NaOCl injury to monitor symptoms and sequelae. In some cases, it is necessary to arrange surgical debridement of the necrotic tissue[11] but, fortunately, in this instance, this was not required.

At the 24 hour review, the swelling was found to have increased in size, but the patient reported their pain as manageable. At 48 hours, the swelling was still present and patient complained of altered sensation in the distribution of the right mental nerve. At the 1 week review, the patient complained that the right cheek was getting bigger and the paraesthesia persisted. By 2 weeks, the swelling was starting to reduce, the ulcer in the buccal sulcus was showing signs of healing and there had been some sensory recovery in the right side of the lower lip. By 6 weeks, the swelling had resolved, and the ulcer had healed. However, the mucosa adjacent to LR4 retained a dark, pigmented colour. There was no pain on palpation of the sulcus, but there was reduced sulcal depth owing to thick, underlying submucosal fibrous tissue. The paraesthesia took approximately 2 months to resolve.

Restorative management

Restorative treatment of LR4 was planned to take place over two appointments, under local anaesthesia, isolated with a rubber dam, and with an endodontic microscope to aid perforation repair and intra-canal visualization.

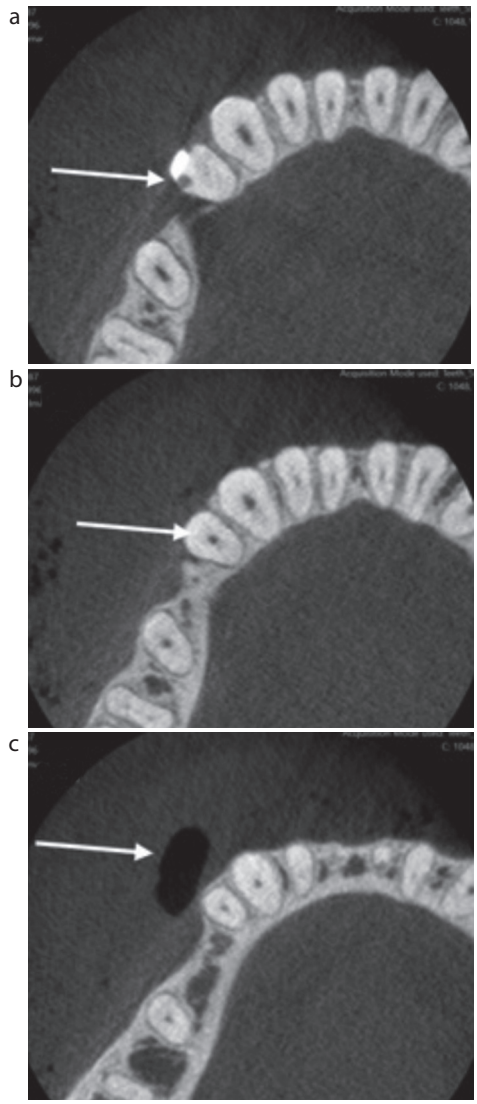


Figure 6. Axial views of LR4, showing the perforation in the distobuccal aspect of the tooth and the location of the pulp chamber deeper down. Only one root canal is present. Image **(a)** shows there has been bone loss at the point where the perforation exits the tooth. The white arrow indicates the exit point. Image **(b)** shows the correct location of the root canal in the centre of the root, while image **(c)** shows the radiolucent, fluid-filled space in the soft tissue.

Perforation repair in LR4 commenced approximately 6 weeks after the injury. According to the literature, perforations should ideally be repaired immediately, and a delay results in a poorer prognosis.[12–15] Immediate repair prevents infection from establishing, which results in less periodontal destruction, a better peri-radicular environment and more favourable healing.[16] However, in this case, it was impractical to do so owing to the patient's level of distress.

Figure 7. Post-operative peri-apical radiograph of LR4 showing root filling and perforation repair.

a

b

Figure 8. Post-operative images taken immediately following obturation and restoration of LR4 show that the necrotic ulcer has healed, but the buccal sulcus retains a dark, pigmented colour, 2 months after the initial injury.

Figure 9. Post-operative image taken immediately following obturation and restoration of LR4 shows the swelling has subsided and facial symmetry has been restored.

a

b

Figure 10. Post-operative images taken 4 months after the initial injury show the necrotic ulcer has completely healed, normal colour of the mucosa is restored and LR4 has been prepared for an onlay.

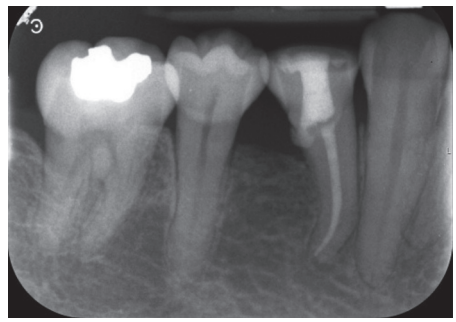


Figure 11. Peri-apical radiograph taken at the 4-month review shows healing of the area of peri-apical pathology and no further bone loss.

A rubber dam was placed on LR4 and the access cavity was re-opened. The tooth was examined under a dental operating microscope, and it was found that the original access had deviated and access had not been gained to the root canal system. The level at which the perforation exits the tooth is the most significant prognostic factor in treatment success and crestal perforations carry the worst prognosis.[16–18] The histological reaction to perforation in this critical zone is periodontal inflammation and downward migration of epithelial tissue, resulting in periodontal pocketing and a higher chance of contamination from the oral environment, which in turn maintains irritation of the periodontium.[19]

The perforation was repaired non-surgically before accessing the pulp chamber to avoid cross contamination. No NaOCl was used to clean the perforation. Under the microscope, it could be seen that gingival tissue had grown into the opening of the perforation but the cavity was dry and there was no bleeding. Mineral trioxide aggregate (MTA) cement was carefully packed into the perforation using a micro-apical placement needle so that it pushed out the in-growing epithelium. Then, a layer of glass ionomer cement (GIC) was placed to shield the setting MTA cement during pulp chamber access. MTA is antibacterial to facultative bacteria[20] without being cytotoxic to the periodontal tissues. It is biocompatible and promotes tissue repair and regeneration.[18,21] Furthermore, it has been shown to exhibit better healing results than calcium hydroxide based materials.[22] This non-surgical approach to repair was adopted rather than surgically exposing the perforation site because calcium silicate-based cements exhibit reduced bond strength and antibmicrobial activity when set in contact with blood.[23,24]

The pulp chamber was accessed and a single, large, central canal was found. Shaping was achieved using nickel–titanium rotary files in conjunction with an ethylenediaminetetraacetic acid (EDTA)-based lubricant. NaOCl (5.25%) and EDTA (17%) solutions were used to irrigate and disinfect the root systems and were agitated using an ultrasonic 'activator'. EDTA functions to remove the smear layer and allow better NaOCl penetration into the dentinal tubules,[25] while agitation of irrigants improves the tissue-dissolving properties of NaOCl solutions.[26]

Between appointments, the canals were dressed using a non-setting calcium hydroxide (CaOH) medicament, which has a pH of 12.5 and has been shown experimentally to penetrate dentinal tubules and eliminate bacteria within 24 hours.[27] Strong antimicrobial activity has also been demonstrated by CaOH dressings left for 7 days.[28]

A gutta-percha (GP) master cone was tried in the canal and a radiograph was taken to check its position. Obturation was achieved with warm vertical condensation and a zinc oxide–eugenol sealer. The thermoplasticized

GP was deposited in small increments and packed down using pluggers to minimize shrinkage of the material and risk of air bubbles. The GP filling extended to just above the level of the alveolar bone and a thin layer of GIC was placed on top to seal it over. The tooth was definitively restored with a composite core build-up.

The post-operative radiographic result (Figure 7) is significant in determining the tooth's prognosis. In this case, it showed the length of the root filling was within 2 mm of the radiographic apex without extruding beyond it, there were no voids visible within the root filling and there was a good coronal seal. A review of the literature on the influence of clinical factors on endodontic outcomes concluded these features are the best predictors for treatment success.[29]

Treatment outcomes

The post-operative radiograph reveals that there was approximately 3 mm of bone loss in the distal aspect of the tooth. Bone loss is a likely outcome when perforation has occurred at the level of the crestal bone. The likelihood was increased because 6 weeks had passed before the perforation was repaired, meaning that apical migration of gingival epithelium was inevitable. Fortunately, the bone loss did not result in any pathological mobility and there has been no persistent pocketing. The patient was happy to report the painful symptoms settled within 4 days following the first stage of root canal treatment, and at all follow-up appointments the tooth remained asymptomatic.

At the 4 month review, the ulcer had completely healed and a normal colour was restored to the mucosa. All swelling had resolved and normal sensation had returned to the lower lip. LR4 had been prepared for an onlay, and the patient was delighted with the outcome of her treatment.

Conclusions

NaOCl is an essential irrigant in root canal treatment. However, it is vital that practitioners are aware of the caustic effects it can have. It is recommended that an electronic apex locator, or a working length radiograph, should always be used to check for perforations before irrigating with NaOCl.

In the event of an NaOCl injury, the patient should be informed, a full clinical assessment should be conducted and appropriate treatment and monitoring planned. In severe cases, an urgent referral to a maxillofacial unit will be required.

Perforations may be restorable with specialist equipment, and a referral should be discussed as one of the possible treatment options with the patient.

CBCT scans are a useful assessment tool in treatment planning perforation repairs.

Compliance with Ethical Standards

Conflict of Interest: The authors declare that they have no conflict of interest.

Informed Consent: Informed consent was obtained from all individual participants included in the article.

References

1. Baumgartner JC, Cuenin PR. Efficacy of several concentrations of sodium hypochlorite for root canal irrigation. *J Endod* 1992; **18:** 605–612. https://doi.org/10.1016/S0099-2399(06)81331-2
2. Clarkson RM, Moule AJ. Sodium hypochlorite and its use as an endodontic irrigant. *Aust Dent J* 1998; **43:** 250–256. https://doi.org/10.1111/j.1834-7819.1998.tb00173.x
3. Becker GL, Cohen S, Borer R. The sequelae of accidentally injecting sodium hypochlorite beyond the root apex. Report of a case. *Oral Surg Oral Med and Oral Pathol* 1974; **38:** 633–638. https://doi.org/10.1016/0030-4220(74)90097-8
4. Balto H, Al-Nazhan S. Accidental injection of sodium hypochlorite beyond the root apex. *Saudi Dent J* 2002; **14:** 36–38.
5. Hatton J, Walsh S, Wilson A. Management of the sodium hypochlorite accident: a rare but significant complication of root canal treatment. *BMJ Case Rep* 2015; 2015:bcr2014207480. https://doi.org/10.1136/bcr-2014-207480
6. Patel E, Gangadin M. Managing sodium hypochlorite accidents: the reality of toxicity. *S Afr Dent J* 2017; **72:** 271–274. https://doi.org/10.17159/2519-0105/2017/v72no6a5
7. Alkahtani A, Alkahtany SM, Anil S. An *in vitro* evaluation of the cytotoxicity of varying concentrations of sodium hypochlorite on human mesenchymal stem cells. *J Contemp Dent Pract* 2014; **15:** 473–481. https://doi.org/10.5005/jp-journals-10024-1565.
8. Hülsmann M, Hahn W. Complications during root canal irrigation – literature review and case reports. *Int Endod J* 2000; **33:** 186–193. https://doi.org/10.1046/j.1365-2591.2000.00303.x
9. Hales JJ, Jackson CR, Everett AP, Moore SH. Treatment protocol for the management of a sodium hypochlorite accident during endodontic therapy. *Gen Dent* 2001; **49:** 278–281.
10. Lam TSK, Wong OF, Tang SYH. A case report of sodium hypochlorite accident. *Hong Kong J Emerg Med* 2010; **17:** 173–176. https://doi.org/10.1177/102490791001700212
11. Farook SA, Shah V, Lenouvel D *et al*. Guidelines for management of sodium hypochlorite extrusion injuries. *Br Dent J* 2014; **217:** 679–684. https://doi.org/10.1038/sj.bdj.2014.1099
12. Seltzer S, Sinai I, August D. Periodontal effects of root perforations before and during endodontic procedures. *J Dent Res* 1970; **49:** 332–339. https://doi.org/10.1177/00220345700490022301
13. Beavers RA, Bergenholtz G, Cox CF. Periodontal wound healing following intentional root perforations in permanent teeth of *Macaca mulatta*. *Int Endod J* 1986; **19:** 36–44. https://doi.org/10.1111/j.1365-2591.1986.tb00888.x
14. Lantz B, Persson PA. Periodontal tissue reactions after root perforations in dogs' teeth - a histological study. *Odontol Tidskr* 1976; **75:** 209–220.
15. Holland R, Ferreira LB, de Souza V *et al*. Reaction of the lateral periodontium of dogs' teeth to contaminated and noncontaminated perforations filled with mineral trioxide aggregate. *J Endod* 2007; **33:** 1192–1197. https://doi.org/10.1016/j.joen.2007.07.013
16. Fuss Z, Trope M. Root perforations: classification and treatment choices based on prognostic factors. *Endod Dent Traumatol* 1996; **12:** 255–264. https://doi.org/10.1111/j.1600-9657.1996.tb00524.x
17. Sinai IH. Endodontic perforations: their prognosis and treatment. *J Am Dent Assoc* 1977; **95:** 90–95. https://doi.org/10.14219/jada.archive.1977.0531
18. Saed SM, Ashley MP, Darcey J. Root perforations: aetiology, management strategies and outcomes. The hole truth. *Br Dent J* 2016; **220:** 171–180. https://doi.org/10.1038/sj.bdj.2016.132
19. Petersson K, Hasselgren G, Tronstad L. Endodontic treatment of experimental root perforations in dog teeth. *Endod Dent Traumatol* 1985; **1:** 22–28. https://doi.org/10.1111/j.1600-9657.1985.tb00554.x
20. Torabinejad M, Hong CU, Pitt Ford TR, Kettering JD. Antibacterial effects of some root end filling materials. *J Endod* 1995; **21:** 403–406. https://doi.org/10.1016/s0099-2399(06)80824-1
21. Torabinejad M, Chivian N. Clinical applications of mineral trioxide aggregate. *J Endod* 1999; **25:** 197–205. https://doi.org/10.1016/S0099-2399(99)80142-3
22. Holland R, Filho JAO, de Souza V *et al*. Mineral trioxide aggregate repair of lateral root perforations. *J Endod* 2001; **27:** 281–284. https://doi.org/10.1097/00004770-200104000-00011
23. Farrugia C, Baca P, Camilleri J, Arias Moliz MT. Antimicrobial activity of ProRoot MTA in contact with blood. *Sci Rep* 2017; **7**: 41359. https://doi.org/10.1038/srep41359
24. Shalabi M, Saber S, Elsewify T. Influence of blood contamination on the bond strength and biointeractivity of Biodentine used as a root-end filling. *Saudi Dent J* 2020; **32**: 373-381. https://doi.org/10.1016/j.sdentj.2019.11.005
25. Bystrom A, Sundqvist G. The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic therapy. *Int Endod J* 1985; **18:** 35–40. https://doi.org/10.1111/j.1365-2591.1985.tb00416.x
26. Stojicic S, Zivkovic S, Qian W *et al*. Tissue dissolution by sodium hypochlorite: effect of concentration, temperature, agitation, and surfactant. *J Endod* 2010; **36:** 1558–1562. https://doi.org/10.1016/j.joen.2010.06.021
27. Behnen MJ, West LA, Liewehr FR *et al*. Antimicrobial activity of several calcium hydroxide preparations in root canal dentin. *J Endod* 2001; **27:** 765–767. https://doi.org/10.1097/00004770-200112000-00013
28. Sjögren U, Figdor D, Spångberg L, Sundqvist G. The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing. *Int Endod J* 1991; **24:** 119–125. https://doi.org/10.1111/j.1365-2591.1991.tb00117.x
29. Ng YL, Mann V, Rahbaran S *et al*. Outcome of primary root canal treatment: systematic review of the literature. Part 2. Influence of clinical factors. *Int Endod J* 2008; **41:** 6–31. https://doi.org/10.1111/j.1365-2591.2007.01323.x