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Tissue Damage after Inadvertent Citric Acid Extrusion during Root Canal Treatment: Report of a Case

SUMMARY

A female patient was seen for an emergency visit after accidental citric acid (40%) injection into soft tissues during root canal irrigation. The patient was experiencing pain, swelling in the right mandible, paraesthesia of the right lower lip and regional necrosis of the buccal mucosa. She was reassured and given analgesics and antibiotics. Follow-up visits were scheduled to monitor the case. When symptoms of swelling and pain resolved, completion of endodontic treatment was decided. A perforating defect was considered responsible for the citric acid extrusion. 6 months after the accident, complete rebound of sensation was noted without any symptoms from the affected area.

Keywords: Citric Acid; Mucosal Necrosis; Paraesthesia; Root Canal Treatment

G. Tomov[1], T. Lambrianidis[2], T. Zarra[2]

[1]Medical University, Faculty of Dental Medicine Oral Pathology Division, Plovdiv, Bulgaria
[2]Aristotle University of Thessaloniki, Department of Endodontology, Dental School Thessaloniki, Greece

CASE REPORT (CR)
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Introduction

Citric acid is a commonly used chelating agent in root canal treatment, offering removal of the smear layer[10,23,29], dentine wall cleaning and root canal disinfection[27,36,39]. Its efficacy is associated with the dissolution of Ca^{++} ions from dentine due to the formation of constants of complexes between citric acid and calcium[7]. It is used at different concentrations, ranging from 1% to 50%[10,11,13,15]. Higher concentrations (25-50%)[11,15,18,28], as well as accumulated application times[33], increase its efficacy. The maximum effectiveness is achieved at pH 1.2[17]. On the contrary, application of citric acid at neutral pH values is not effective for Ca^{++} ions removal[9].

Many studies have investigated the effectiveness of citric acid for smear layer removal[9,10,23,30,33]. However, its cytotoxic and tissue damaging effects have been poorly investigated. The available data on the topic is contradictory. Some studies[1,4] have shown that although citric acid is less cytotoxic compared to other chelating agents used in endodontics, such as EDTA and EGTA[1,24,32], it has short- and long-term damaging effects on cell cultures *in vitro*. The detrimental effect on vital cells is associated with its acidic pH[21]. Chan et al[4] investigated morphological alterations associated with the citric acid cytotoxic and cytostatic effects on cultured dental pulp cells[4]. The exposure of cells to pure 1% citric acid (pH = 2.26) for 60 sec caused immediate cellular death. Citric acid (15%) placed on murine macrophage cultures reduced macrophage viability by 50-70% within 24 hours[1]. Concentration and time of application are contributing factors to the cytotoxic behaviour of citric acid[4]. Other studies have demonstrated that the citric acid solution is non-cytotoxic to vital fibroblasts in cultures[24,32].

Citric acid can also potentially cause a decalcifying action on periapical bone and affect inflammatory and neuro-immune regulation when extruded into peri-radicular tissues[1].

A comprehensive literature review revealed that there are no available studies describing tissue damage after inadvertent citric acid extrusion into soft tissues during root canal treatment. The objective of this case report was to describe clinical signs, symptoms and management of a case of chemical injury caused by accidental citric acid injection into soft tissues during root canal treatment.

Case Report

A 46-year-old Caucasian female with non-contributory medical history was referred by her private

dentist to the Faculty of Dental Medicine (Plovdiv, Bulgaria) for assessment, consultation and treatment of progressively intense facial pain and swelling in the posterior region of her right mandible. The referring dentist informed us that root canal treatment of the tooth #46 had been initiated 3 hours earlier, following the diagnosis of chronic apical periodontitis. On questioning, the patient reported acute pain during the procedure and swelling in the right mandible some minutes later.

On extraoral examination, there was swelling in the right mandible (Fig. 1) and paraesthesia of her right lower lip. In particular, the patient reported tickling, tingling and numbness. There was no external appearance of ecchymosis or haematoma. The intraoral examination revealed a regional buccal mucosa necrosis adjacent to the tooth #46 (Fig. 2) and remnants of a class II (MOD) amalgam filling, as well as temporary cement used to seal the access cavity preparation on tooth #46. Radiographic examination revealed a periapical lesion around the mesial root of tooth #46 (Fig. 3a). On request for further details and after personal contact with the referring dentist, it was concluded that accidental injection of 40% citric acid (Cerkamed, Poland) into soft tissues had occurred during irrigation of the root canals.

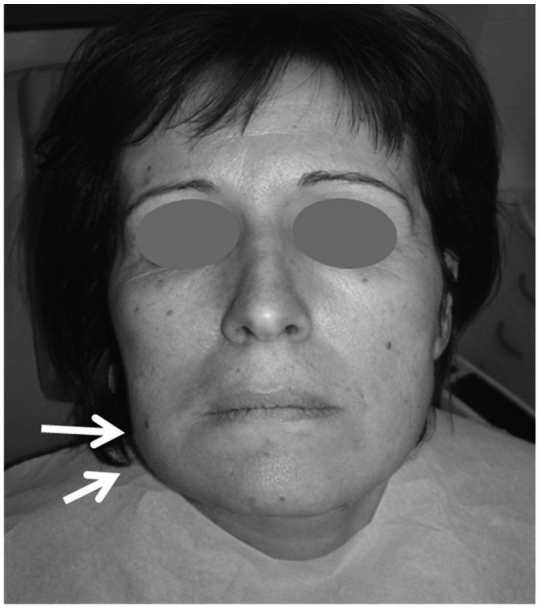

Figure 1. Mild swelling in the right mandible region

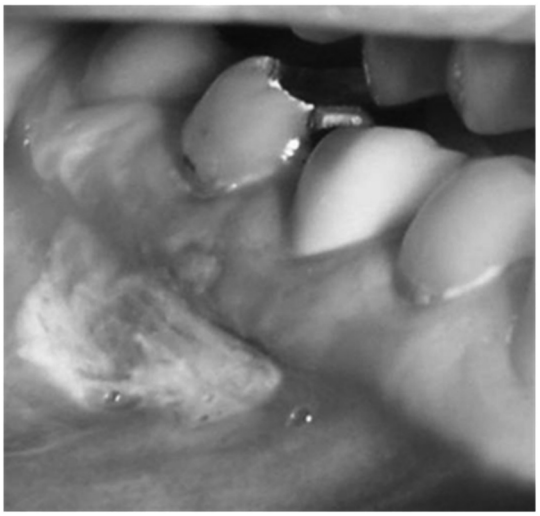

Figure 2. An area of soft tissue necrosis surrounded by well-vascularized tissue near the periapical area of tooth #46

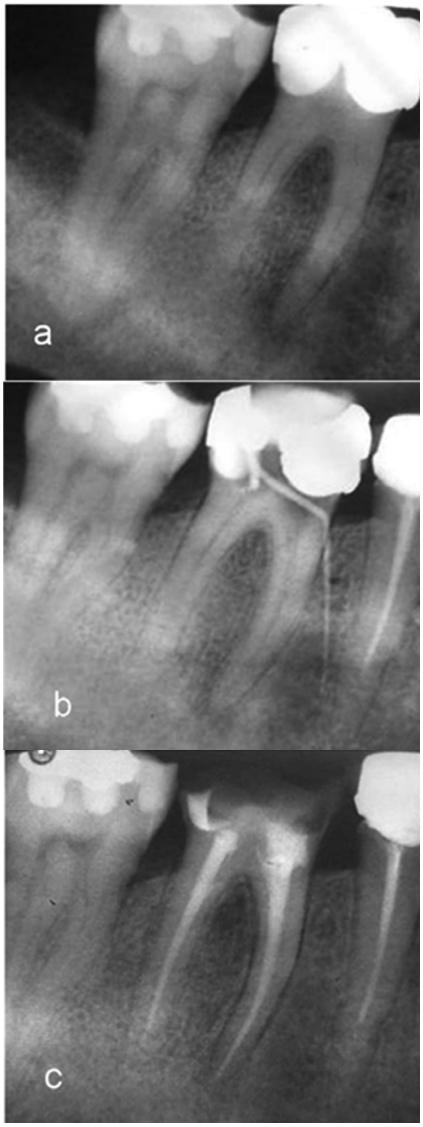

Figure 3. Periapical radiographs of tooth #46. a. Preoperative radiograph showing a periapical lesion around the mesial root of tooth #46; b. Radiograph with a gutta-percha point inside the perforating defect. The rubber dam was secured in place by ligatures of dental floss and wedges, so as to prevent the interference of the metallic dental clamp to the radiographic image; c. Postoperative radiograph taken immediately after root canal obturation

A complete explanation of the situation was provided to the patient. The initial treatment plan included administration of an oral analgesic (Diclofenac 50mg tid pc for 3 days), as well as an antibiotic (Amoxicillin 250 mg q8h for 7 days) for pain and infection control, and continuous monitoring of the case. Additionally, oral rinsing with Chlorhexidine gluconate 0.12% (Peridex, Periocard) was prescribed. Cold compresses applied for the first 4-6 hours, followed by warm saline soaks were recommended to reduce swelling. The patient was seen on day 2 and the swelling was reduced. A part of the necrotic tissue was spontaneously detached 5 days after the incident (Fig. 4). After a week, the swelling had disappeared and the motor function of the facial nerve remained intact. 3 weeks later, oral mucosa completely healed (Fig. 5), but there was residual paraesthesia of her right lower lip.

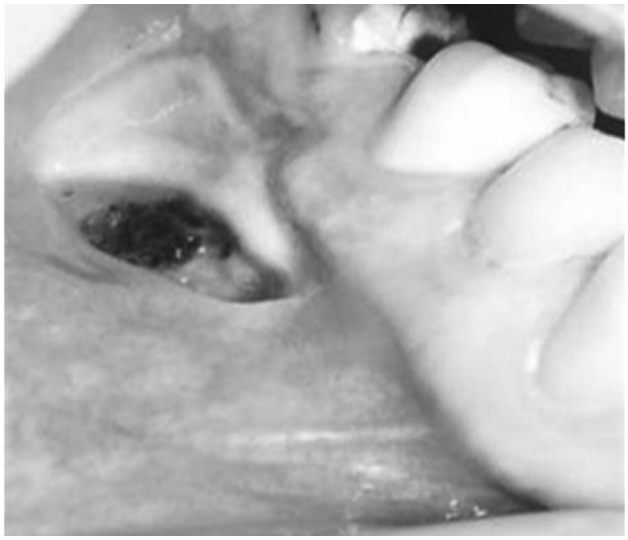

Figure 4. 5 days after the accident, necrotic tissue was evident after spontaneous detachment of the most severely affected mucosa

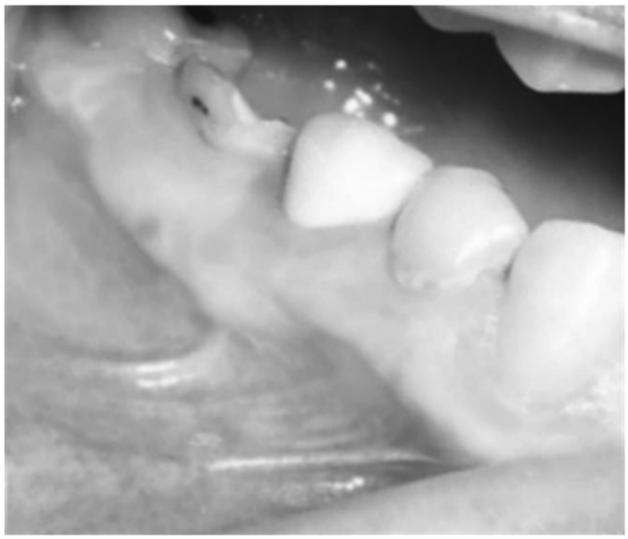

Figure 5. Complete healing of the oral mucosa 2 weeks after the accident

Completion of the root canal treatment on tooth #46 was recommended. Following rubber dam isolation and removal of the amalgam remnants and temporary cement, the pulp chamber floor was explored with the aid of magnification under operating microscope (Kaps International, USA). The examination revealed the presence of a perforation next to the mesial axial walls of the pulp chamber. Its presence was clinically confirmed with the aid of apex locator (Root ZX Morita, CA, USA) and radiographically following the insertion of a gutta-percha point into the perforating defect (Fig. 3b). Bleeding was arrested by clamping the area with a cotton pellet and the perforation was immediately sealed with resin-based glass-ionomer cement (Vitremer, 3M/ESPE, USA). Root canal instrumentation was completed with Pro Taper rotary files (Dentsply Maillefer, Switzerland) according to manufacturer's instructions under copious irrigation between successive instruments with 2.5% w/w NaOCl using 5-ml disposable plastic syringes and 30-gauge needle tips (Endo EZ; Ultradent Products Inc., UT, USA). The tip was placed passively into each canal, as far as 3 mm from the apical foramen, without binding. The mesial canals were enlarged to size F2 and the distal to size F3 to their full working lengths. A final flash with 17% EDTA at the end of the chemo-mechanical preparation was performed. The root canal system was dried with sterile paper points (Roeko Dental Products, Langenau, Germany) and chemically pure $Ca(OH)_2$ mixed with saline was placed in the canal with a #40 lentulo paste carrier (Antaeos; Vereinigte Dentalwerke & Co.). The access cavity was temporarily sealed (Cavit; Espe, Seefeld, Germany) and the patient was scheduled for the final appointment after 1 week. The 1-week period was uneventful. Only residual paraesthesia was observed at the final appointment. The intra-canal medicament was removed by means of instrumentation and copious irrigation and canals were obturated with gutta-percha and I-Root SP bio-ceramic sealer (IBC, Canada) using the lateral condensation technique. The tooth was sealed temporarily with resin-based glass ionomer cement (Fig. 3c). The patient was referred back to her private dentist for tooth restoration and scheduled for follow-up examinations on a 15-day basis to monitor the progress of paraesthesia.

The patient did not comply with the scheduled appointments. She re-appeared 6 months later with no symptoms from the affected area, but tooth remained un-restored. Clinical examination displayed rehabilitation of sensation in the areas of innervation of mental nerve branches. Upon questioning, the patient informed us that the paraesthesia symptoms were improving slowly and a total rebound of sensation was evident nearly 4 months after the completion of the root canal treatment. Radiographic examination revealed a reduction in size of the periapical lesion, despite the lack of permanent restoration (Fig. 6).

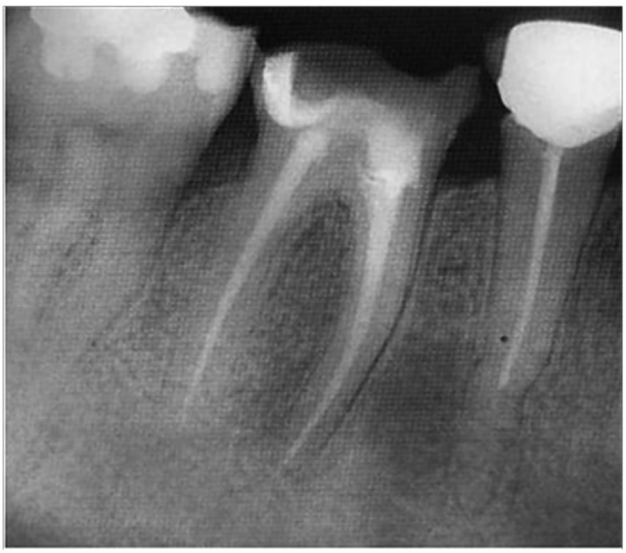

Figure 6. Follow-up radiograph 6 months after the completion of root canal treatment

Discussion

Although root canal irrigants and chelating agents are essential elements of root canal treatment, offering disinfection, debris and smear layer removal[10,23,26,29], they are cytotoxic in contact with vital tissues[1,4,16]. It is well-known that a small amount of irrigant may be extruded into periapical tissues during root canal instrumentation, irrespective of the preparation technique and type of the utilized instruments[19]. A questionnaire survey among diplomats of the American Board of Endodontics indicated that only 58% of the responders had never experienced NaOCl accident, while the remaining 42% had at least one NaOCl accident during their practice[20].

The clinical signs and symptoms following periapical extrusion of alkaline solutions, such as NaOCl, have been widely described in many previously published case reports[2,8,14,22,25,31]. However, this is the first case report in which the effects of inadvertent acid extrusion into soft tissues during root canal irrigation are presented.

The injection of acid solutions into soft tissues causes coagulation necrosis *via* intracellular dehydration and protein coagulation, limiting the spread of the chemical in the tissues[12,38]. On the contrary, alkaline agents, such as NaOCl, penetrate local tissues rapidly and deeply, causing liquefaction and necrosis, which facilitates the spread of the chemical[34,35].

NaOCl is the irrigant of choice in root canal treatment as it has the unique capacity to dissolve necrotic tissue and the organic components of smear layer[41]. Demineralising agents, such as citric acid, have been recommended as adjuvants in root canal therapy. In this case, only citric acid was utilized for root canal irrigation by the treating dentist. Although this is not recommended, the exclusive use of citric acid for irrigation prevented any further severe complications which might have been caused by NaOCl injection into soft tissues through the perforating defect[2,8,14,22,25,31].

In this case, a perforation of the mesial pulp chamber wall allowed for the injection of citric acid into soft tissues. The early recognition and seal of the perforation prior to root canal instrumentation would have prevented the accident. In a systematic review and critical analysis of published data upon irrigant extrusion and identification of causing factors, affecting or predisposing to irrigant extrusion during root canal irrigation in human mature permanent teeth, perforations were identified among the most frequently cited factors[13].

The cytotoxic behaviour of citric acid was enhanced[4] due to utilization of a high concentration (40%). The effectiveness of citric acid is a function of its concentration[11,15,18,28], but so is toxicity. This should be considered in clinical practice in order to select a citric acid solution with a concentration that can achieve maximum efficacy with minimum risk of tissue damage in cases of extrusion into soft tissues.

Citric acid has been also used in periodontology to promote periodontal healing and regeneration, as its demineralising effect can favourably affect migration, attachment and orientation of periodontal ligament cells to the diseased cementum and dentin[5,40]. The lack of toxic effects in these cases could be attributed to the low amount and concentration of citric acid coming in contact with vital tissues as compared to the corresponding amount and concentration of the solution in case of its accidental injection into soft tissues.

The clinical signs and symptoms following citric acid extrusion were alarming and their management was urgent. No specific treatment can reverse the deleterious effects caused by citric acid. The usage of a weak alkali to chemically neutralize the area is contraindicated, as this may stimulate an exothermic reaction, exacerbating tissue injury. The treatment of choice is supportive/palliative and targets to pain relief, control of swelling and prevention of secondary infection[12,38]. Reassurance should be provided to the patient for the symptoms from the involved area. For the immediate pain relief, a nerve block with a local anaesthetic should be considered. Adequate pain control can be achieved by administration of analgesics. Non-steroidal anti-inflammatory drugs and aspirin should be avoided in the acute stage to prevent the risk of interstitial haemorrhage in the soft tissues[12,38]. Because of the possible spread of infection, prophylactic antibiotic therapy is considered necessary[12,38]. Light corticosteroid and antihistamine therapy should be suggested in selected cases. Cold compresses should be used for the first 4-6 hours to minimize swelling in the affected area[6]. Thereafter, warm compression should be preferred to shorten the clearing up time of ecchymosis

by increasing circulation of the involved area[12,38]. Oral rinsing with normal saline can also improve the circulation to the affected intraoral tissues[12,38]. In cases of neurological damage, the patient should be referred to medical or dental specialists, who have experience in nerve assessment and repair, for follow-up and possible treatment[37].

The extrusion of citric acid into soft tissues is an extremely rare complication of root canal treatment and, to our knowledge, it has never been described before. In such a case, it is crucial to monitor closely the patient and provide appropriate medical care. The described case did not require hospitalization and surgical intervention of the affected area. No permanent damage to tissues occurred.

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Correspondence and request for offprints to:

Georgi Tomov
Faculty of Dental Medicine
Oral Pathology Department
3 Hristo Botev Blvd.
4000 Plovdiv, Bulgaria
E-mail: dr.g.tomov@gmail.com