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Management of atypical mucosal lesions from primary endodontic infection: Two cases report

Guilherme de Jesus Souza Sirino[1], Susilena Arouche Costa[2], Erika Martins Pereira[3], Soraia de Fátima Carvalho Souza[4,*]

[1] School of Dentistry, Federal University of Maranhão, São Luis, MA, Brazil

[2] Postgraduate Dentistry Program, Federal University of Maranhão, São Luis, MA, Brazil

[3] Department of Oral Pathology, School of Dentistry, Federal University of Maranhão, São Luis, MA, Brazil

[4] Department of Endodontics, School of Dentistry, Federal University of Maranhão, São Luis, MA, Brazil

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Corresponding author:
Soraia de Fátima Carvalho Souza
Universidade Federal do Maranhão
Faculdade de Odontologia
Av dos Portugueses,1966
Cidade Universitária Dom Delgado
São Luis, MA, Brasil
Cep- 65.085-580
Tel.: +55 98 3272 8575
E-mail: endosoraia@gmail.com

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Aim/Case report: We have described here the diagnosis and treatment of two cases involving atypical mucosal lesions originating from primary endodontic infections in young patients. Differential diagnoses were established by clinical and radiographic examinations. In both the cases, a red-colored lesion was observed in the alveolar mucosa, with a soft consistency and a sessile-base without any drainage path in tooth 31 (case 1) and with a sinus tract in tooth 36 (case 2), as confirmed by X-ray. The cleaning and shaping of the root canals were performed, and camphorated paramonochlorofenol (CPMC)-enriched calcium hydroxide paste was used as an intracanal dressing for 6 months, with replacement every 60 days. **Conclusion:** Based on our experience, we thus conclude that long-term root canal maintenance with CPMC-enriched calcium hydroxide was an effective therapeutic protocol for promoting the healing of atypical mucosal lesions.

Keywords: Diagnosis, differential. Root canal therapy. Calcium hydroxide.

Introduction

A local volumetric increase in the oral cavity soft tissues is an unreliable diagnostic feature considering the similar changes produced by several different pathologies. The presentation of excess tissues could be a normal variation or evidence of an inflammatory proliferative process, a cyst, or a neoplasm[1]. Parulides (or gumboils) are the frequent manifestation of an inflammatory proliferative process in the oral cavity, and they commonly affect the gingival margin or alveolar mucosa—the most external portion of communication between the dental apex and the intraoral environment. Parulides originate from the sites of primary endodontic infection[2], wherein invasive bacteria and their toxins pass from caries lesions to the pulp space and dentinal tubules, including apical ramifications, isthmuses, and deltas[3]. In terms of clinical appearance, a parulis may be reddish or yellowish in color or may be of normal mucosal color and may have a nodular or papular appearance[4]. In some cases, due to low-intensity exposure to a chronic irritant, these lesions turn atypical and exhibit tissue hyperproliferation. This tissue hyperproliferation may depend on several factors, including the virulence of the irritant stimulus present in the root canal system (microorganisms), duration of the disease, and host defense mechanisms[5]. Over a period of time, parulides can come to resemble other lesions, which makes their definitive diagnosis difficult. Differential diagnoses include inflammatory fibrous hyperplasia, peripheral giant cell granuloma, and pyogenic granuloma[2,4]. A precise diagnosis defines the best therapeutic approach and can avoid unnecessary or non-operative surgical procedures. The clinical cases presented below describe the diagnosis and treatment of atypical inflammatory proliferative mucosal lesions with a nodular appearance originating from primary endodontic infection.

Cases Report

Case 1

A 16-year-old girl presented with a complaint of a blow to her face 2 years ago. Her intraoral visual inspection revealed coronal fractures without pulp exposure of teeth 42, 41, and 32 as well as a coronal fracture with pulp exposure of tooth 31 and a darkened crown. A red-colored lesion was observed in the alveolar mucosa, extending from the distal surface of tooth 41 to the distal surface of tooth 32 (Fig. 1A). Closer examination revealed that the lesion had a soft consistency and a sessile-base without any drainage path; this feature is consistent with that of a nodule (Fig. 1B).

The patient anamnesis revealed pain when chewing. Tooth 31 showed sensitivity in vertical percussion and apical palpation tests, with absence of periodontal pocket and grade I dental mobility. Teeth 42, 41, and 32 responded to a pulp sensitivity test to cold when performed with dichlorofluoromethane gas (Endo-Ice®, Maquira, Maringá, PR, Brazil), whereas tooth 31 did not respond. X-rays showed diffuse periapical bone rarefaction associated with tooth 31 (Fig. 1E). On the basis of the clinical and radiographic findings, a diagnosis of chronic apical periodontitis with a parulis or associated pyogenic granuloma was established. We accordingly chose to perform an endodontic treatment to tackle the evolution of the mucosal lesion.

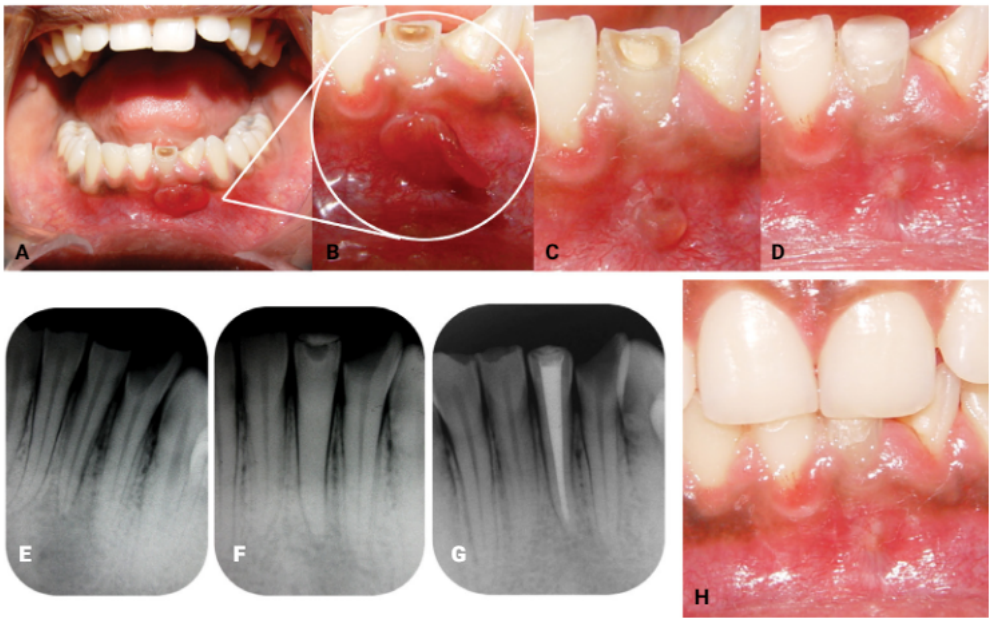

Figure 1. Case 1. (A) General visual inspection; (B) initial appearance of the lesion, close-up view; the appearance of the lesion after 15(C) and (D) 45 days with intracanal dressing maintenance of CPMC-enriched calcium hydroxide; (E) initial X-ray; (F) evidence of bone repair in X-ray 45 days later; (G) complete bone repair; and (H) healed lesion site 6 months later.

After administration of anesthesia and a rubber dam to tooth 31, non-conventional endodontic access was created. The root canal was enlarged up to K-file #50 (Dentsply Maillefer, Ballaigues, Switzerland) to 1 mm short of the apical foramen. A step-back flaring technique was performed at 2 mm increments with Gates-Glidden drills (Dentsply Maillefer) #1 to #3. Intracanal irrigation was performed with 2 mL of 2.5% sodium hypochlorite (NaOCl) solution between each instrument. Foramen patency was made with a #15 K-file. To remove the smear layer, 17% ethylenediaminetetraacetic acid (EDTA) was left in the canal (3 times for 1 min), followed by the application of 5 mL of 2.5% NaOCl. The canal was dried using paper points (Dentsply, Petrópolis, RJ, Brazil) and dressed with a camphorated paramonochlorofenol (CPMC)-enriched calcium hydroxide paste (Calen PMCC®, SS White Artigos Dentários Ltda, Rio de Janeiro, RJ, Brazil). Temporary cervical and coronal sealing was fulfilled with Coltosol® (Dentalville, Joinville, SC, Brazil) and light-cured resin (Z100 3M ESPE, Irvine, USA).

After 15 days, the atypical mucosal lesions were markedly reduced, and the patient reported that she was no longer suffering pain when chewing (Fig. 1C). The patient's follow-up vertical percussion and apical palpation tests were also negative. After 45 days, the lesion had disappeared almost completely (Fig. 1D) and bone tissue reorganization was evident (Fig. 1E, 1F). The intracanal dressing was maintained for a 6 month period, during which it was replaced every 60 days.

Next, the intracanal dressing was removed by abundant irrigation with 2.5% NaOCl, followed by the application of 17% EDTA and 2.5% NaOCl. The canal was dried with

paper points and filled by gutta-percha cones and resin-based sealer (Sealer-26®, Dentsply) using the lateral condensation technique. Cervical and coronal sealing were made with Coltosol® (Dentalville) and a light-cured resin (Z100 3M ESPE). Next, X-rays confirmed that the periapical bone tissue was completely repaired (Fig. 1G). Clinical observation revealed complete resolution of the lesion (Fig. 1H).

Case 2

A 10-year-old girl presented with the complaints of a bubble beside her tooth. Her clinical intraoral examination revealed a nodular proliferation of tissues located on the interface mucogingival junction that extended from the mesial surface to the top of the medial cusp of tooth 36. The lesion was red in color, had a soft consistency and a sessile-base (Fig. 2A), and was draining by a sinus tract. We observed an unsatisfactory amalgam restoration on tooth 36 without coronal color change.

The patient reported that she was not experiencing any symptoms. The pulp sensitivity, vertical percussion, and apical palpation tests were negative. The tooth did not show dentine sensitivity, mobility, or a periodontal pocket. X-ray examination showed radiolucent image on the mesial and distal roots and furcation area. Mapping of the sinus tract confirmed that the lesion was made of endodontic origin (Fig. 2B). On the basis of the clinical and X-ray findings, we made a diagnosis of chronic periapical abscess and nodular parulie.

The endodontic access was performed following the conventional guidelines. Canals were prepared by the crown-down technique to 1 mm short of the apical foramen. The mesial and distal root canals were enlarged up to #40 and #55 K-files. The intracanal dressing and temporary coronal sealing used were the same as those described for Case 1.

After 15 days, we observed a significant reduction in the atypical mucosal lesion (Fig. 2C), and X-rays showed evidence of bone tissue reorganization (Fig. 2D). At 45 days, the lesion had disappeared completely (Fig. 2E) and X-rays revealed signs of regression of periapical bone rarefaction around the distal root (Fig. 2F). We followed the same maintenance protocol and change in the intracanal dressing as in Case 1.

After 6 months, the area where the lesion had been located showed a normal clinical appearance (Fig. 2G). We performed root filling using the techniques described above. Cervical and coronal sealing were made with materials described previously.

Complete remission of the radiolucency was noted in the furcation area and in the periapical area of the distal root, including the formation of a hard coat, suggesting cell reorganization and reinsertion of periodontal ligament fibers. However, we still observed radiolucency in the mesial root, although it was filled completely with radiopaque points, suggesting that lesion repair processes were ongoing (Fig. 2H).

Discussion

It is well established that microbial infection of the root canal system is the cause of apical periodontites[3]. In Case 1, the pulp space was exposed to the oral environment

via a coronal fracture, whereas it was exposed in Case 2 by means of marginal infiltration at the enamel/dentine/restoration interfaces.

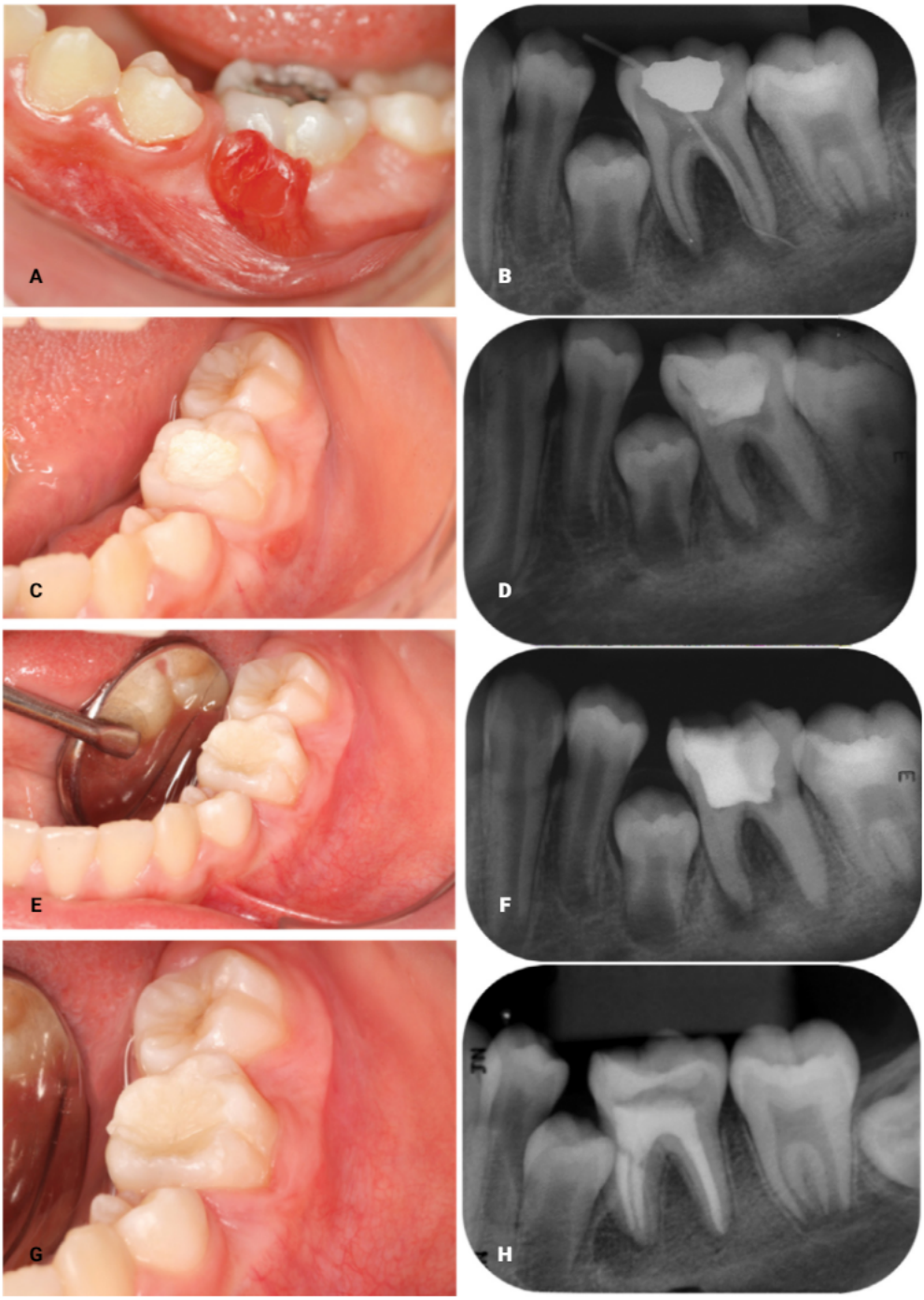

Figure 2. Case 2. (A) Initial appearance of the lesion; (B) mapping of the fistula; (C) clinical and (D) radiographic appearance of the lesion after 15 days with intracanal dressing maintenance of CPMC-enriched calcium hydroxide; (E) clinical appearance; and (F) radiographic evidence of bone reorganization after 45 days; (G) total regeneration of tissue at the lesion site; and H) evidence of bone repair after 6 months.

We observed a commonality between the two cases in terms of the presence of an atypical mucosal lesion with a nodular appearance that was associated with devitalized teeth and "open" communication (e.g., a sinus tract or deep caries) with the oral cavity. In Case 1, clinical inspection of the lesion suggested tissue proliferation with an increase in the number of blood vessels (Fig. 1B), probably because there was no way for the exudate to drain from the infected root canal via the periapical tissues into the oral environment. This lack of sinus tract may have occurred as a result of caries development at the site of the fracture, which allowed for an exchange of irritant substances between the pulp cavity and the oral environment. In Case 2, the presence of a sinus tract in the nodule was accompanied by radiolucency on the distal root of tooth 36 (Fig. 2B), as confirmed by X-ray. The clinical management of these cases require specific strategies to eliminate the microorganisms and their toxins[6].

In both the cases, differential clinical diagnoses were made during the first appointment and then later confirmed as conclusive diagnoses during the second endodontic handling, after the canals had been maintained with CPMC-enriched calcium hydroxide paste for 15 days, taking into consideration the significant remission of the atypical mucosal lesion (Fig. 1C and 2C), demonstrating its broad antimicrobial spectrum. Although the toxicity of CPMC has been proven in vitro and in vivo, phenols exhibit low toxicity when in contact with living tissues at low concentrations, as observed in Calen PMCC®, thus maintaining its antimicrobial effect[7]. This can be explained by the production of a weak salt, para-chlorophenolate, which helps maintain a high pH[76].

The atypical mucosal lesion could have been confused with the lesions of non-endodontic origin, such as the peripheral giant cell granuloma or pyogenic granuloma. Peripheral giant cell granuloma usually occurs due to local irritation or chronic trauma[8]. Pyogenic granuloma can arise as a result of trauma, as well as due to hormonal changes and the use of some types of medications, such as carbamazepine[9]. This lesions can manifest clinically as red-colored nodules with a sessile-base[2,10], similar to the lesions observed in our patients. Thus, the differential diagnosis between these lesions can be made based on anamnesis and clinical and radiographic examinations aimed at these possible etiological factors. Oral biopsy contributes to the provision of accurate diagnoses that guide the treatment of oral lesions[11]. However, only through anamneses, clinical, and radiographic examinations allowed us to develop diagnostic hypotheses of atypical mucosal lesion caused by long-term primary endodontic infection, which can occur in case of an equilibrium between microorganism's virulence and the host's immune response[12].

Teeth with primary endodontic infection had higher contents of endotoxins, and it was associated with the severity of bone destruction in the periapical tissues[6]. Vianna et al.[13] reported the ability to remove or inactivate the bacterial endotoxins unrelated to the action of the endodontic irrigants. Therefore, they suggest that a chelant, such as EDTA, be used prior to long-term intracanal treatment with calcium hydroxide in order to support the elimination of endotoxins. This therapeutic protocol was adopted in the two clinical cases, although we did not identify the bacterial species present or quantify the endotoxin load of the canals, which may be considered as limitations of this study.

Despite the variety of bacterial species known to be involved in primary endodontic infection, calcium hydroxide has become accepted as a broad-spectrum antimicrobial agent. Its efficacy can be attributed to its immediate antimicrobial action, raised pH, inactivation of bacterial endotoxins, and osteoinductive ability[14]. It acts by releasing OH^- and Ca^{++} ions in an aqueous environment[15]. The OH^- ions raise the pH of the environment, stimulating the denaturing of the proteins present in the bacterial cytoplasmic membrane and causing damage to this membrane and to the bacterial DNA; simultaneously, the high concentration of Ca^{++} can activate tissue enzymes, such as alkaline phosphatase, inducing the process of bone mineralization[15].

We thus conclude that, through anamneses, clinical examinations, and X-ray investigations, we could develop accurate diagnostic hypotheses of atypical mucosal lesion originating from primary endodontic infection, avoiding the unnecessary removal of tissues for histopathology. Moreover, irrespective of the inherent bacterial populations in the endodontic infections, the therapeutic protocol of long-term intracanal dressing maintenance of CPMC-enriched calcium hydroxide was effective in promoting the regeneration of healthy tissues at the mucosal lesion sites in both the cases.

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References

1. Amirchaghmaghi M, Falaki F, Mohtasham N, Mozafari PM. Extragingival pyogenic granuloma: a case report. Cases J. 2008 Dec 3;1(1):371. doi: 10.1186/1757-1626-1-371..
2. Krishnapillai R, Punnoose K, Angadi P V., Koneru A. Oral pyogenic granuloma — a review of 215 cases in a South Indian Teaching Hospital , Karnataka , over a period of 20 years. Oral Maxillofac Surg. 2012 Sep;16(3):305-9. doi: 10.1007/s10006-012-0315-z.
3. Kakehashi S, Stanley H, Fitzgerald R, Bethesda. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. Oral Surg. 1965 Sep;20(3):340-9.
4. Harrison JW, Larson WJ, Wash T. The epithelized oral sinus tract. Oral Surg Oral Med Oral Pathol. 1976;42(4):511-7. doi: 10.1016/0030-4220(76)90299-1.
5. Drăghici EC, Crăiţoiu Ş, Mercuţ V, Scrieciu M, Popescu SM, Diaconu OA, et al. Local cause of gingival overgrowth. Clinical and histological study. Rom J Morphol Embryol. 2016;57(2):427-35.
6. Gomes BPFA, Endo MS, Martinho FC. Comparison of endotoxin levels found in primary and secondary endodontic infections. J Endod. 2012 Aug;38(8):1082-6. doi: 10.1016/j.joen.2012.04.021.
7. Pereira TC, da Silva Munhoz Vasconcelos LR, Graeff MSZ, Ribeiro MCM, Duarte MAH, de Andrade FB. Intratubular decontamination ability and physicochemical properties of calcium hydroxide pastes. Clin Oral Investig. 2018 Jul 7. doi: 10.1007/s00784-018-2549-0.
8. Mighetl A, Robinson P, Hume W. Peripheral giant cell granuloma: a clinical study of 77 cases from 62 patients, and literature review. Oral Dis. 1995 Mar;1(1):12-9.
9. Marla V, Shrestha A, Goel K, Shrestha S. The histopathological spectrum of pyogenic granuloma: A case series. Case Rep Dent. 2016;2016:1323798. doi: 10.1155/2016/1323798.
10. Shadman N, Ebrahimi SF, Jafari S, Eslami M. Peripheral giant cell granuloma : a review of 123 Cases. Dent Res J (Isfahan). 2009 Spring;6(1):47-50.

11. Bradley G, Magalhaes M. Oral manifestation of systemic diseases — a perspective from an oral pathology diagnostic service. Oral Dis. 2018 Mar;24(1-2):219-223. doi: 10.1111/odi.12751.

12. Zehnder M, Belibasakis GN. On the dynamics of root canal infections — what we understand and what we don ' t. Virulence. 2015;6(3):216-22. doi: 10.4161/21505594.2014.984567.

13. Vianna M., Horz H., Conrads G, Zaia AA, Souza-filho F., Gomes BPF. Effect of root canal procedures on endotoxins and endodontic pathogens. Oral Microbiol Immunol. 2007 Dec;22(6):411-8.

14. Mohammadi Z, Dummer PMH. Properties and applications of calcium hydroxide in endodontics and dental traumatology. Int Endod J. 2011 Aug;44(8):697-730. doi: 10.1111/j.1365-2591.2011.01886.x.

15. Mohammadi Z, Shalavi S, Yazdizadeh M. Antimicrobial activity of calcium hydroxide in endodontics : a review. Chonnam Med J. 2012 Dec;48(3):133-40. doi: 10.4068/cmj.2012.48.3.133.