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REVIEW ARTICLE

Differential Diagnosis and Treatment Proposal for Acute Endodontic Infection

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ABSTRACT

The objective of this study was to describe the main lesions that simulate clinically and propose a treatment protocol for acute endodontic infection. Signs and clinical symptoms of periodontal abscess, gingival abscess, odontoma, herpes simplex, pericoronitis, acute pulpitis and necrotizing ulcerative gingivitis/periodontitis (NUG/NUP) were described and compared with acute endodontic infections. A treatment protocol was described by optimizing the procedures in access cavity, microbial decontamination and detoxification of the root canal, apical debridement, intracanal and systemic medication and surgical drainage procedures. The convenience of the use of 5.25% sodium hypochlorite, root canal instrumentation using a crown-down technique, intracanal medication with 2% chlorhexidine or triple antibiotic paste and the convenience of the use of antibiotics, analgesics, and surgical drainage to solve cases of acute dentoalveolar abscess was discussed.

Keywords: Endodontics, Infection, Root canal treatment.

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INTRODUCTION

The acute endodontic infection, particularly the acute dentoalveolar abscess, is one of the most frequent situations in dental emergency. However, if there is persistence of signs and clinical symptoms after the initial treatment is possible that the cause is directly related to an incorrect diagnosis and/or inadequate clinical procedures.[1,2]

The acute dentoalveolar abscess is an inflammation characterized by presence of purulent exudate in the periradicular tissues.[3] Pulp necrosis and the presence of specific microbiota are essential factors for its development.[4,5] In accordance to signs and clinical symptoms presented, it is possible to classified into three phases: (A) periradicular or first phase; (B) intraosseous or second phase; (C) submucosal/subcutaneous or third phase. However, there are difficulties in establishing clinical parameters for each phase, allowing the use of a single local and systemic treatment protocol.[6]

Lack of response to the pulp sensitivity test, pain exacerbation to vertical percussion, presence of edema in the alveolar mucosa and/or subcutaneous (either localized or spread) and spontaneous pain are its most clinical manifestations.[3] The radiographic image shows a slight diffuse bone rarefaction, with a thickening of the space corresponding to the periodontal ligament. Indisposition and slight elevation of the body temperature can also be present.[6] Figures 1A to D illustrates the main acute dentoalveolar abscess clinical manifestations.

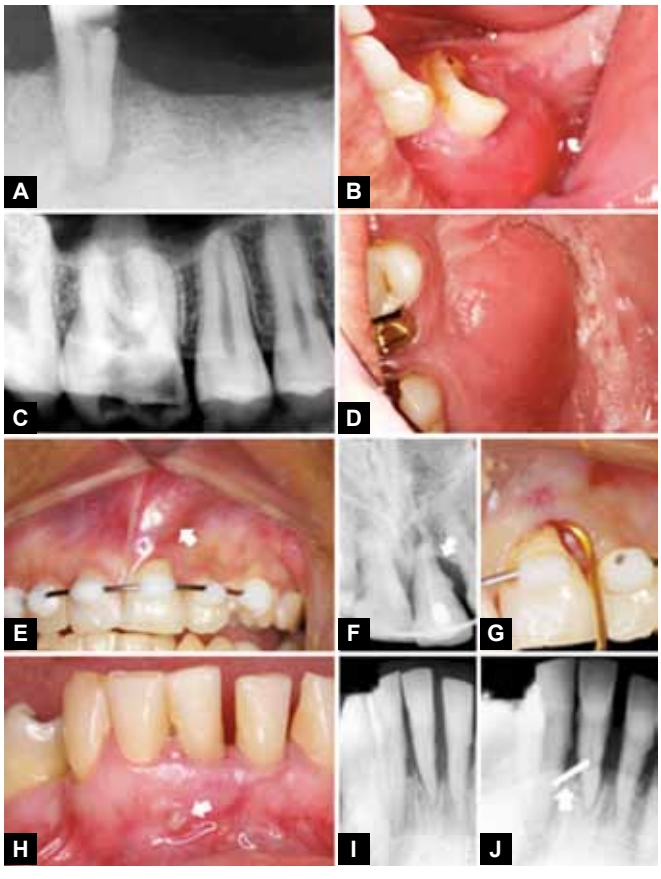
Nevertheless, several clinical situations can be similar of those presented in the acute dentoalveolar abscess, leading the professional to a mistaken diagnosis and, consequently, to set up an inadequate treatment plan. Thus, the objective of this study is to present the main

clinical cases that can be similar to those found in acute dentoalveolar abscesses and to optimize an effective local and systemic treatment protocol.

DIFFERENTIAL DIAGNOSIS

Periodontal abscess, gingival abscess, odontoma, herpes simplex, pericoronaritis, acute pulpitis and necrotizing ulcerative gingivitis/periodontitis (NUG/NUP) are diseases that frequently simulate the clinical situation of an acute dentoalveolar abscess.

Periodontal abscess: This comprises an acute periodontal destructive process, characterized by the presence of purulent exudate in periodontal tissues, not coming from the dental pulp.[7] The presence of microorganisms involving periodontal tissues is fundamental to its development, usually associated to a preexisting periodontal pocket.[7,8] This disease is the most confused with acute dentoalveolar abscess.[7] Figures 1E to J illustrates a case of periodontal abscess with an edema in the alveolar mucosa (Fig. 1E), presence of alveolar bone loss (Fig. 1F) and periodontal pocket (Fig. 1G), which are the clinical signs that differs it from the acute dentoalveolar abscess.



Figs 1A to J: Acute dentoalveolar abscess: Diffuse radiographic apical image (A and C) and its clinical repercussion, with local edema (B) and edema in palatine mucosa (D), Periodontal abscess: Edema in alveolar mucosa (E), alveolar bone loss (F) and presence of periodontal pocket (G), Fistula in alveolar mucosa (H), with bone loss and periodontal involvement (I and J)

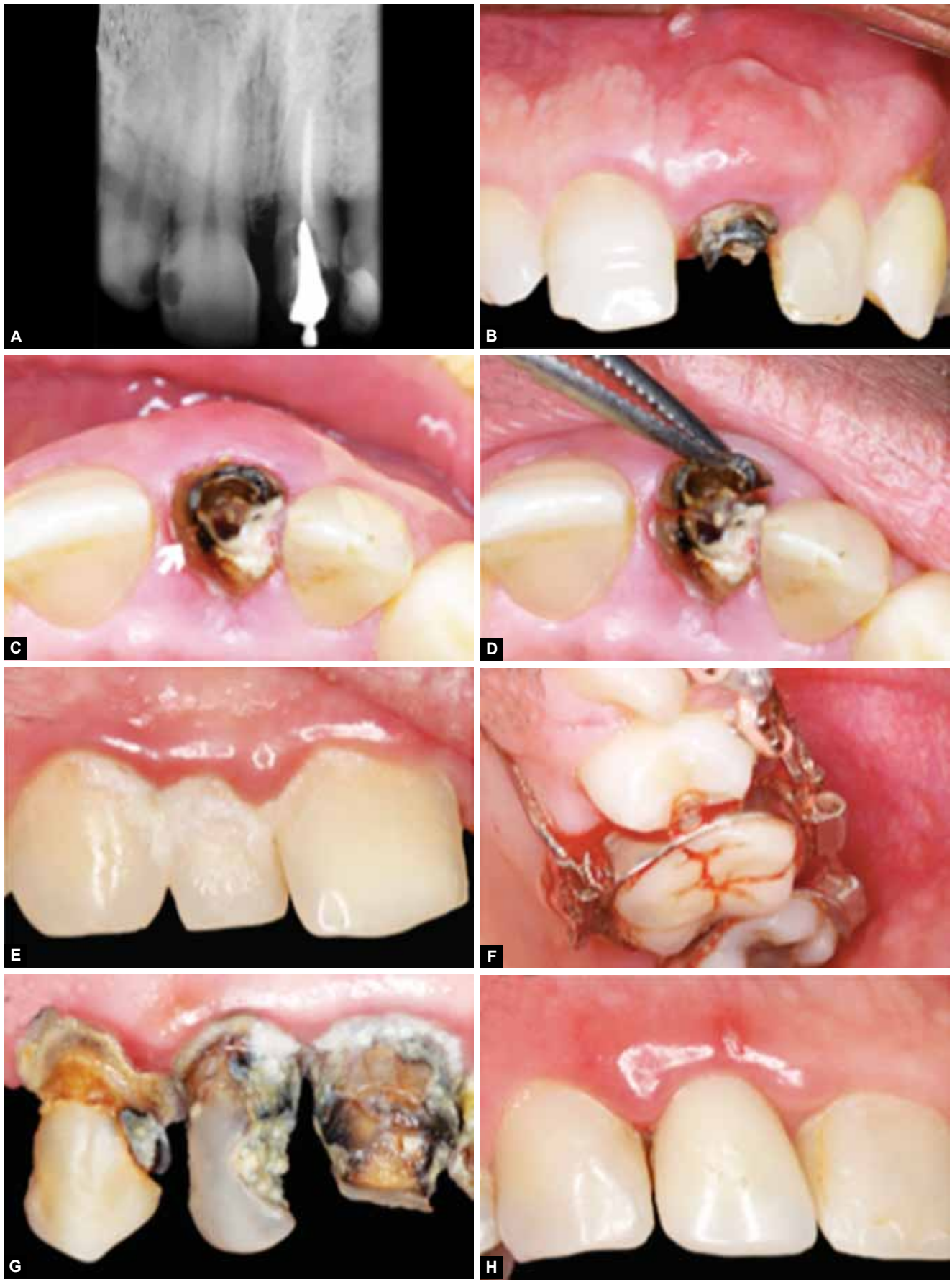
Figures 1H to J illustrate a similar clinical situation, but it also demonstrates the presence of a fistula, similar to those occurring in endodontic infections, but in teeth presenting pulp vitality with accentuated periodontal bone loss. Figures 2A to D demonstrate a clinical condition of a endodontically-treated tooth endodontic (Fig. 2A), but with periodontal abscess (Fig. 2B), due to a longitudinal radicular fracture (Figs 2C and D).

Gingival abscess: This comprises an abscess unrelated to periodontitis, limited to the marginal gingiva without previous infection.[9] Its main causes are local and microbial factors related to the presence of strange materials and/or supragingival and subgingival biofilm. The clinical sign is an edema in the mucosa of the gingival margin, sensitive to palpation and susceptible to bleeding on probing (Figs 2E to H). Figures 2E and G illustrate the situation of a gingival abscess caused by the presence of biofilm due. Figures 2F and H illustrate, respectively, the presence of an orthodontic band and a poorly adapted restoration, allowing the development of gingival abscess.

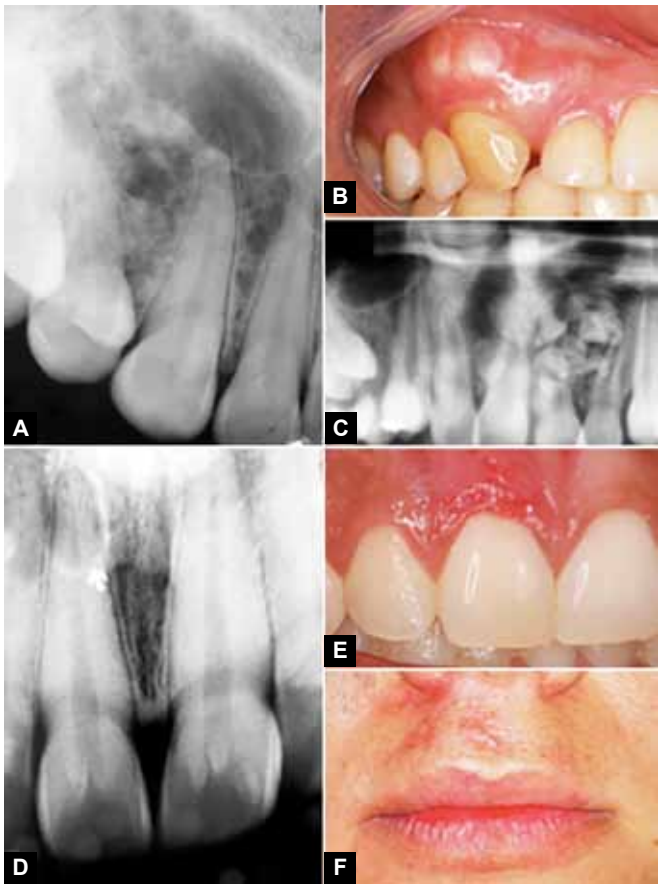
Odontoma: This comprises a benign intraosseous proliferation in the maxillar, in which all the odontogenic tissues are present, with the possibility of presenting abnormal morphology in the tooth structures.[10,11] Its etiology is discussed and still unknown.[11] They are classified into complex and compound, being frequently identified at the second decade of life, usually asymptomatic and associated with late dental eruption.[12] Figures 3A and B illustrate a case of alveolar tumefaction of the buccal mucosa wrongly diagnosed as an acute dentoalveolar abscess, due to the presence of the periapical image. Figure 3C illustrates a characteristic clinical case of odontoma.

Herpes simplex: This case present vesicular lesions that have affinity with epithelial and nerve cells. It is transmitted by herpes simplex virus type I (HSV-1), usually acquired through direct contact with previously infected lesions and/or organic fluids. Recurrent infections, appearing in variable periods, typically affect mucocutaneous margins, particularly in lips. The recurrence of HSV-1 infection inside of the oral cavity is uncommon in relatively healthy patients.[13] Figure 3D illustrates a case in which the incisive foramen was atypically projected over the central incisor apex, which clinically presented ulcerous lesions in the gingival mucosa (Fig. 3E). Figure 3F illustrates the lesion in its initial phase with vesicles.

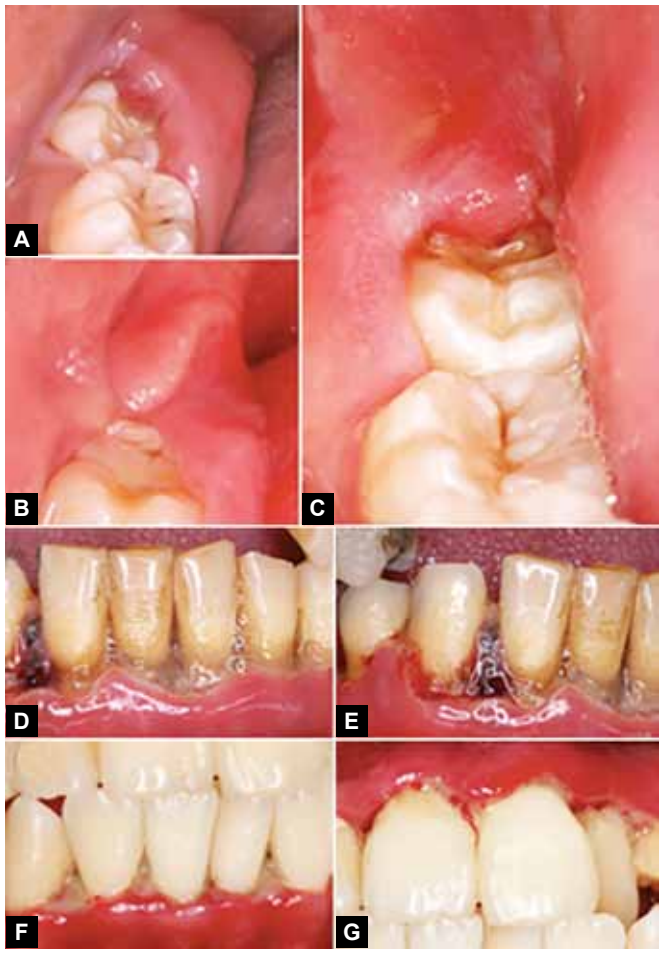
Pericoronaritis: This is an inflammation process in the adjacent tissues of a partially erupted tooth, exacerbated by the accumulation of food debris and the presence of bacteria.[14] There is extreme pain in the affected area and that can spread to ear and tongue frenulum (mouth floor).[15] Figures 4A to C illustrate cases of partially-erupted third molars with the presence of pericoronaritis.



Figs 2A to H: Periodontal abscess (A and B) with radicular longitudinal fracture (C and D), gingival abscess, resulting from coronal biofilm (E), poorly adapted orthodontic band (F), subgingival dental cavity (G) and cervical restorative material excess (H)



Figs 3A to F: Odontoma: Case with radiolucid image in the apical area of upper canine (A) and tumefaction of the alveolar mucosa (B) wrongly interpreted as acute dentoalveolar abscess. Typical radiographic image of an odontoma (C) Herpes simplex: Case with an image of the incisive foramen projected over the apex of the upper central incisor (D) and gingival mucosa vesicle (E) and lip (F)



Figs 4A to G: Pericoronitis. Presence of inflammatory purulent exudate associated with partially erupted teeth (A to C); Clinical manifestations of NUG (D to G)

Acute pulpitis: Acute pulp inflammation cause pain, which can be provoked or spontaneous, intermittent or continuous, and located or diffuse, depending on the degree of involvement of the dental pulp.[6] The presence of pulp sensitivity, diagnosed through thermal and cavity test, differs them from the dentoalveolar abscess.

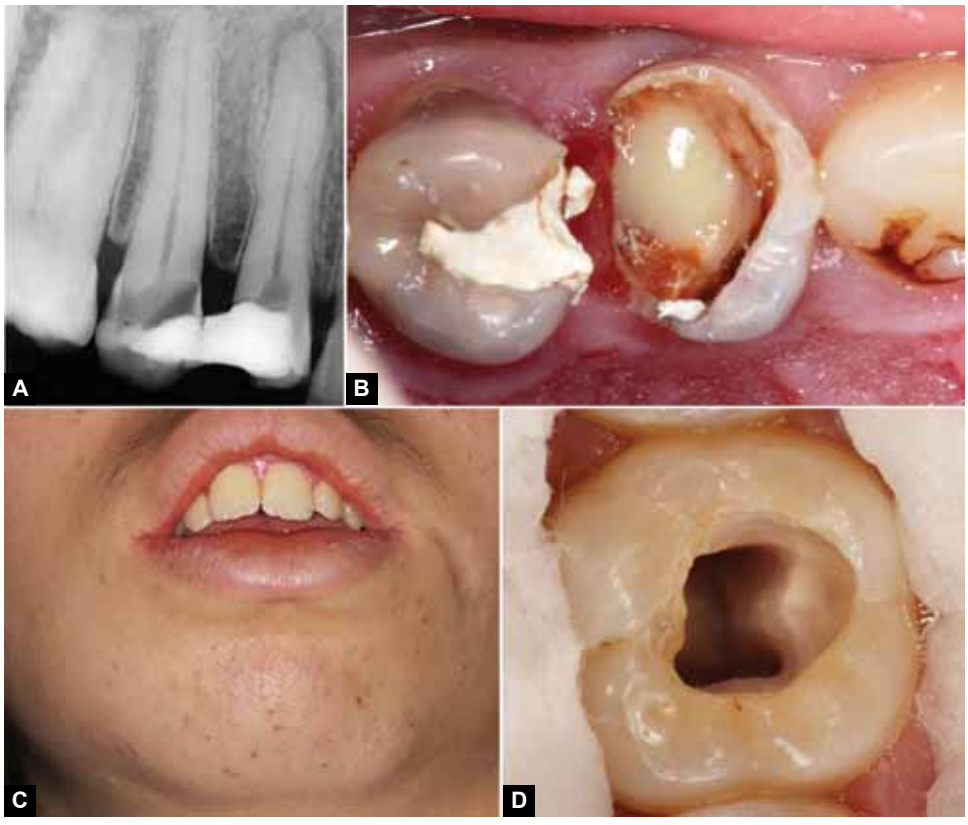
Necrotizing ulcerative gingivitis/periodontitis: Comprises an infection caused by bacterial associations and, in some cases there is also the presence the specific virus.[16] It is usually related to the release of stress hormones that alter the rates of T4/T8 lymphocytes, causing a decrease in the chemotaxis and the phagocytic response of neutrophils.[17] It clinically presents inflamed, edematous, and hemorrhagic interdental papillae (Fig. 4D). The gingival papillae often present crater-like necrosis, covered by a pseudomembrane (Figs 4E and 4F). There are fetid odor, intense pain and local hemorrhage (Fig. 4G).[16,17]

DISCUSSION

Considering the clinical situations previously described, several cases are wrongly diagnosed as acute dentoalveolar abscesses. Despite the technical and scientific advances in the endodontic practice, many procedures performed by dental surgeons are surrounded by empiricism and without scientific and clinical relevance.

A rational treatment sequence is fundamental to the re-establishment of the tooth with acute dentoalveolar abscess to normal functions. Once controlled the infection, clinical success of the endodontic treatment is similar to that of teeth without acute infections.[18] Therefore, based in clinical and scientific evidences, a rational treatment for acute endodontic infection treatment protocol is:

Cavity access: Despite the absence of pulp sensitivity, it is recommended anesthetize the area around the affected tooth, due to the intense sensitivity to percussion. Depending of the evolution and the condition of the adjacent alveolar bone, immediately after reaching the pulp chamber, there might happen the intracanal spontaneous drainage of inflammatory purulent exudate (Figs 5A and B). The intracanal drainage commonly ceases after the irrigation of the root canal. However, in any clinical situations , there is no intracanal drainage, presenting only exhalation of fetid odor, characteristic of the tissue necrosis (Figs 5C and D).



Figs 5A to D: Clinical situations after the access to the crown. Presence of spontaneous drainage (A and B) or absence of drainage, with presence of local edema (C and D)

An intense irrigation of the pulp chamber with 5.25% sodium hypochlorite must be performed after the conclusion of the cavity access. Lower concentrations are relatively inefficient to detoxification of the infected root.[19] As an alternative to potentize the antibacterial action of sodium hypochlorite, it is alternate the irrigation with acetic acid.[20]

Microbial decontamination and detoxification of the root canal: This comprises one of the fundamental step to solve acute endodontic infections. There is a direct relation between the microbial decontamination and detoxification of the root canal and the treatment sucess.[18] An inefficient performance of this process will cause the persistence of signs and/or clinical symptoms of the acute dentoalveolar abscess.

In this phase, it is recommended the endodontic irrigation with 5.25% sodium hypochlorite, reserving the use of the 2.5% concentration only during the root canal glyde path. The effects of the several sodium hypochlorite concentrations on the radicular dentin are similar.[19] Therefore, in endodontic infection is recommended the 5.25% concentration, due to its better antimicrobial activity.[19,20] Chlorhexidine no is recommended to irrigation endodontic in acute endodontic infection because present low tissue necrosis and microbial biofilm solving action.[21]

After detoxification initial, the root canal debridement must be performed preferably with rotary instruments, due to a lower probability of apical extrusion of the debris.[22] The action of the instruments must be progressive and in the crown-down direction; it is recommended the previous cervical flared with Gates Glidden or similar burs. Once close to the tooth real length, an copious irrigation with 5.25% NaOCl must be performed once again with sonic or ultrasonic agitation.[23]

Apical debridement: The apical debridement must be performed with #10K or #15K manual instruments (LK 10 or 15, for instance), with the objective of disorganizing apical biofilm and/or unblocking the apical foramen.[24] To refine the local antisepsis, it is recommended the execution of an apical patency at least until the #20K.[25]

Intracanal medication: Maintaining the access to the crown open in one of the greatest myths concerning the acute dentoalveolar abscess treatment. Endodontic spontaneous drainage tend to last just a few seconds and ceases spontaneously after septic content neutralization, with rare cases where there will be a need to keep it without intracanal medication and temporary restoration. The re-contamination and supply of substrate to root canal bacteria counter indicates the idea of keeping it open.[26]

The challenge is selecting a substance that acts in the presence of the microbial biofilm and decomposing

organic material in the root canal. Formocresol must not be used because it does not neutralize endotoxins and it has high toxicity.[27,28] Therefore, it is recommended the filling of the root canal with 2% chlorhexidine gel for 48 hours to 7 days, for no longer than 15 days.[26]

Recently, it has also been preconized the use of tri-antibiotic paste (metronidazole, minocycline and ciprofloxacin), but it still requires conclusive clinical studies.[29] Once occurred the remission of the acute endodontic infection, the conventional endodontic treatment must be conducted as soon as possible.

Systemic medication: The prescription of antibiotics must happen only in cases of fever, prostration, loss of function, or when the has a compromised immune system.[30] Once the local treatment was properly performed, the prescription of antibiotics provides no additional benefits.[3,31]

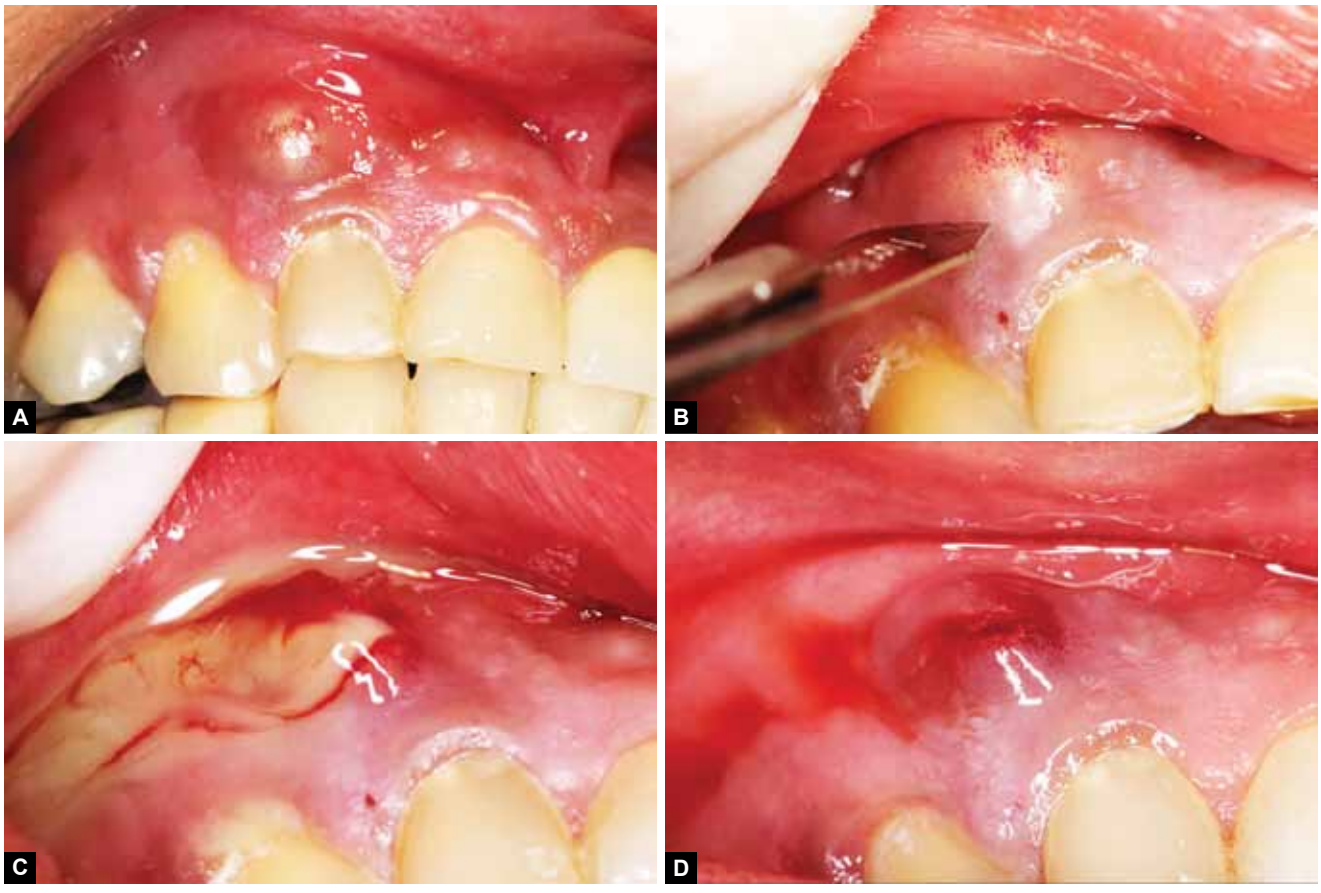
When recommended, the use of amoxicillin associated with potassium clavulanate is the best alternative, for 5 days. In case of allergic hypersensitivity to beta-lactam, can be prescribed azithromycin for 3 days or clindamycin for 5 days. For analgesics, codeine or tramadol are recommend only in case of pain.[6]

It is preferable avoiding prescription of antibiotics for the pregnant patient. In special cases, it is recommended amoxicillin or ampicillin, for 5 days. In cases of allergic hypersensitivity,[31,32] it is recommended the prescription of azithromycin, in similar doses to recommended in conventional patients. Paracetamol is analgesic recommended to these cases. On the other hand, for the lactating the cefaclor is the antibiotics recommended for 5 days. Clindamycin is to use in cases of allergic hypersensitivity.[31,32]

Surgical drainage: The option for surgical drainage is questionable. It must be performed only when it will bring benefits to the patient, such as comfort due tissue decompression and removal of inflammatory purulent exudate (Fig. 6).[3] Once drained and the microbial decontamination and detoxification of the root canal has been performed, there is no need to maintain the a surgical drain (Fig. 6D).

CONCLUSION

Through the showed clinical cases and based on the discussion of acute infections treatment protocols, it is possible to conclude that the persistence of signs and clinical symptoms is usually related to an incorrect diagnosis and inadequate clinical and systemic procedures.



Figs 6A to D: Surgical drainage of an acute dentoalveolar abscess. Edema containing inflammatory purulent exudate (A), local drainage selection (B), drainage of acute dentoalveolar abscess (C) and final surgical drainage (D)

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