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Dental abscess: A microbiological review

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ABSTRACT

Dental abscess is a frequently occurring infectious process known to the health practice. The fate of the infection depends on the virulence of the bacteria, host resistance factors, and regional anatomy. Serious consequences arising from the spread of a dental abscess lead to significant morbidity and mortality. Acute dental abscess is polymicrobial, comprising of strict anaerobes, such as anaerobic cocci, *Prevotella*, *Fusobacterium* species, and facultative anaerobes, such as viridans group *streptococci* and the *Streptococcus anginosus* group. Numerous novel, uncultivable and fastidious organisms have been identified as potential pathogens with the use of non-culture techniques. The majority of localized dental abscesses respond to surgical treatment while the use of antimicrobials is limited to severe spreading infections. There is a need for good-quality clinical trials of sufficient size to identify the ideal treatment. The microbiology of the acute dentoalveolar abscess and its treatment in the light of improved culture and diagnostic methods are reviewed.

Key Words: Antibiotic sensitivity, dental abscess, diagnosis, microbiology, therapy

INTRODUCTION

Dental abscess was a poorly discussed topic of medical science until the late 1900s. This clinical entity was frequently underestimated in terms of its morbidity and mortality. Dental or dentoalveolar abscess is a denomination used to describe localized collection of pus in the alveolar bone at the root apex of the tooth. It usually occurs secondary to dental caries, trauma, deep fillings or failed root canal treatment. Once the intact pulp chamber is breached, colonization of the root canals occurs with a diverse mix of bacteriological agents. These microorganisms are capable of forming biofilms in root canals, hence making application of the "biofilm concept" plausible in such infections.[1] After entering the periapical tissues via the apical foramen, these bacteriae are capable of inducing acute inflammation leading to pus formation. The pathogenesis of dentoalveolar abscess is polymicrobial in nature, comprising of various facultative anaerobes, such as the viridans group *streptococci* and the *Streptococcus anginosus* group, and strict anaerobes, especially anaerobic cocci, *Prevotella* and *Fusobacterium* species.[2] If not treated at an early stage it may rapidly evolve and spread to adjacent anatomic structures, leading to serious complications such as septicemia, cavernous sinus thrombosis, brain abscess, shock, and occasionally to death. Possibility of development of complications and the associated morbidity and mortality makes it an important public health problem. In this paper, current knowledge of the pathogenesis, diagnosis, and management of dental abscess is reviewed.

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EPIDEMIOLOGY AND RISK FACTORS

In the early 1600s, the London Bills of Mortality began listing the causes of death with teeth being continually listed as the fifth or sixth leading cause of death.[3] By 20th century, the potential of dental abscesses to spread and cause severe sepsis leading to death was recognized. An audit carried out at the

Hull Royal Infirmary between 1999 and 2004 showed an increase in the number of patients presenting to oral and maxillofacial surgery services with dental sepsis.[4] In the United States, a large prospective study reported that 13% of adult patients sought treatment for dental pain and infection over a 24 month follow-up.[5] The incidence of dentoalveolar abscess was 6.4% among children attending an outpatient dental clinic in Nigeria.[6] In India, dental caries affect 60-65% of the general population.[7] In addition, periodontal disease is estimated to occur in 50-90% of the population in India, depending on age.[8] Improved methods of diagnosis and reporting of this common problem are required to allow exhaustive epidemiological analysis and its implications on health-care system. Nevertheless, oral diseases have been identified as one of the priority health conditions because, in late stages, they cause severe pain and are expensive to treat. This translates into a loss of man-hours, which has a significant negative impact on economic productivity.[9]

The various host factors play a significant role in pathogenesis of dental infections and their complications. It has been observed that there are specific "at-risk" population groups. In a retrospective series of 185 cases, Huang *et al*. found a statistically significant correlation of acute dental infections, complications and death with medically compromising diseases, such as diabetes, renal insufficiency, hepatic cirrhosis, myeloproliferative disorders, and chemotherapy.[10] Most studies report a male preponderance of the severe odontogenic infections in both adult[11] and pediatric[6] populations.

CAUSATIVE AGENTS

Bacteriological agents implicated in causation of dental abscesses comprise of the complex mix of strict anaerobes and facultative anaerobes. Datasets from culture and molecular studies show that over 460 unique bacterial taxa belonging to 100 genera and 9 phyla have been identified in different types of endodontic infections.[12] Depending upon the recovery and cultural conditions, strict anaerobes outnumber facultative by a ratio which varies between 1.5 and 3:1 in mixed infections.[13,14] The mean number of species recovered by culture from dentoalveolar aspirates is four with a range of between 1 and 7.5.[13,15,16] Dental abscesses caused solely by strict anaerobes occur in approximately 20% of cases. Although there is a wide range depending upon recovery conditions (6-63%) it has been observed that pure cultures from an acute dental abscess are unusual[15-19] and mixed aerobic infections are also uncommon, accounting for 6% of abscesses.[19] Polymicrobial nature of such infections and presence of cultivable and uncultivable microbes may pose a challenge toward diagnostic analysis in routine microbiological laboratories.

STRICT ANAEROBES

The most commonly isolated genera include anaerobic *streptococci*, *Fusobacterium* species and the black-pigmented anaerobes such as *Prevotella* and *Porphyromonas* species.[20] *Prevotella* species have been reported as the most frequent isolates in numerous studies, found in 10-87% of dentoalveolar abscesses.[12,14,15,21] *Prevotella* intermedia, *Prevotella* nigrescens and *Prevotella* pallens, *Porphyromonas endodontalis,* and *Porphyromonas gingivalis* are the commonly detected pathogens.[22]

Bacteroides fragilis, a more common isolate from intra-abdominal infections, has only infrequently been reported from acute dentoalveolar infections and is not regarded as an oral commensal. The member of the *Bacteroides* genus most likely to be recovered from an acute dental abscess is *Bacteroides* forsythus (now transferred to a new genus as *Tannerella forsythia*).[23]

Fusobacterium periodonticum and *Fusobacterium nucleatum* (which includes subsp. nucleatum, subsp. polymorphum, subsp. animalis, subsp. vincentii, and subsp. fusiforme) are frequently detected with *F. nucleatum* recovered most frequently from the acute dental abscess.[24,25] Baumgartner *et al*. performed polymerase chain reaction for *F. nucleatum* on samples from endodontic origin and found prevalence of 73%.[26]

Studies have shown the presence of *Clostridium* species causing dentoalveolar abscess in a range varying from 2% to 20%. Important species isolated include *Clostridium hastiforme, Clostridium histolyticum*, *Clostridium perfringens*, *Clostridium subterminale*, and *Clostridium clostridioforme*.[13,27] Some infrequent pathogens in the oral cavity belonging to *Clostridium* genus are *Clostridium sporogenes*, *Clostridium bifermentans, Clostridium botulinum*, "*Clostridium oedematiens*," *and* "*Clostridium welchii*."[28]

With the help of Polymerase Chain Reaction, high prevalence of *Treponema* species has been reported within the acute dental abscess. It was found that

Treponema denticola was present in up to 79% of dental abscesses.[29] Other *Treponema* species were found in lower numbers, including Treponema *socranskii*, Treponema *pectinovorum*, Treponema *amylovorum*, and Treponema *medium*.

Certain unusual or new bacteria isolated from cases of dental abscess include members of the genus *Atopobium* (Gram-positive strictly anaerobic coccobacilli), for example, *Atopobium parvulum* and *Atopobium rimae*. Other anaerobic Gram-positive rods include *Bulleidia extructa*, *Cryptobacterium curtum*, *Eubacterium sulci*, *Mogibacterium timidum* and *Mogibacterium vescum*,[30] *Pseudoramibacter alactolyticus*, and *Slakia exigua*.[31] While unfamiliar anaerobic Gram-negative rods include *Filifactor alocis*, *Dialister pneumosintes*,[23,32,33] *Centipeda periodontii* and *Selenomonas sputigena*.[34] Catonella morbi, a Gram-negative anaerobe formerly known as *Bacteroides* D42, was found in 16% of 19 aspirates, and Granulicatella adiacens, a facultative anaerobic Gram-positive coccus formerly known as nutritionally variant *streptococci*, was present in 11% of 19 aspirates.[35,36] The detection of these unfamiliar species has expanded our insight into the potential of virulence and pathogenicity of these organisms in acute dental abscess and interactions with more commonly isolated and better understood pathogens.

FACULTATIVE ANAEROBES

Facultative anaerobes belong to the viridans group *streptococci* and the anginosus group *streptococci* are commonly implicated in dental abscess. The viridans group *streptococci* includes mitis group, oralis group, salivarius group, sanguinis group, and the mutans group.[37] The anginosus group (formerly referred to as "*Streptococcus milleri*" or *S. anginosus*) has also been reported with varying degrees of accuracy.

Staphylococcus aureus has been frequently reported from acute dental abscess, ranging from 0.7% to 15%.[17,19,27,38] Recovery rates of coagulase-negative strains of *staphylococci* (usually reported as *Staphylococcus epidermidis*) are generally higher with figures ranging from 4% to 65%.[14,17,19,30,38,39]

CLINICAL FEATURES

The signs and symptoms of the acute dental abscess are pain, swelling, and erythema usually localized to the affected tooth, although the suppuration can frequently spread to the nearby tissues causing fatal complications. Fever, extraoral and intraoral swelling, erythema, tenderness to palpation are notable. Trismus in addition to any changes in the voice such as hoarseness and drooling should prompt the dentist to an emergency situation. The clinical examination should focus on the general status of the patient such as lethargy or extreme sickness. Deep neck and descending necrotizing mediastinal abscesses are a rare complication of the dental abscess and spread of odontogenic infections accounts for a large number of deep neck abscesses.[40] Delay of diagnosis owing to vagueness of early symptoms is one of the primary reasons for the high mortality. The literature describes mortality rate of mediastinitis up to 40% despite aggressive use of antibiotics and advances in intensive care facilities.[41] Death usually occurs due to sepsis and multiorgan failure although airway occlusion is also a significant complication and requires early management by tracheostomy.

MICROBIOLOGICAL ANALYSIS

Sample collection

In the past, inappropriate methods of sampling hampered correct identification of the causative pathogens involved in the development of the dental abscess. The studies using swabs of purulent material have demonstrated poor recovery of strict anaerobes and low mean numbers of isolates per sample (range 1.0-1.6)[42] choice of sample type and method of sampling are crucial to optimal diagnostic efficacy. Ideally, an aspirate through intact mucosa after disinfection by an appropriate antiseptic mouthwash, e.g., chlorhexidine should be collected. This will reduce contamination from the normal oral flora. Some researchers have also sampled purulent exudates from within infected canals.[24,42]

Cultural and non-cultural techniques

Significant improvement in the routine diagnostic yield from acute dental abscesses has occurred with employment of meticulous specimen collection and processing on selective and nonselective agars under appropriate atmospheric conditions. However, despite the close attention to detail, it is apparent that many genera of bacteria have yet to be cultured. A major limitation of past cultural studies is that a large percentage of the oral microflora does not grow on conventional artificial culture media in the laboratory.[43]

Introduction of molecular techniques has helped us to understand the microbial bionomics of dental abscesses. Use of PCR or deoxyribonucleic acid (DNA) — DNA hybridization chequerboard techniques and more recently 16S rRNA gene sequencing and species-specific primers have helped in searching for the presence of specific microbes.[30,32,35,44,45] The use of 16S rRNA gene sequence analysis for the identification of isolates and clones has greater precision to discriminate between taxa and recognize novel taxa than conventional identification methods.[30] Genetic methods of identification have also helped in detection and speciation of fastidious organisms like Treponemes in samples from dental abscesses.[29] Culture-independent, molecular analysis has revealed a more diverse microflora associated with endodontic infections than that revealed by cultural methods alone.

ANTIBIOTIC RESISTANCE

Antimicrobials must never be used as a replacement for appropriate surgical drainage and/or debridement. The maintenance of an airway and abscess drainage is a condition sine qua non. However, antimicrobial therapy initiated soon after diagnosis and before surgery can shorten the period of infection and minimize associated risks like bacteremia.

Penicillins and cephalosporins

Historically, the penicillins have been used as first-line agents in the treatment of odontogenic infections. Increasing rates of penicillin resistance and treatment failures have been reported. The highest rates of penicillin resistance have been observed with the members of the genus *Bacteroides* and *Prevotella*.[46-48]

Penicillin resistance in these pathogens has been correlated with β-lactamase production. Heimdahl *et al*. reported on a series of patients with orofacial infections who failed to respond to penicillin therapy due to β-lactamase producing *Bacteroides*.[49] Using an animal model, β-lactamase production by strains of *Prevotella. melaninogenicus* in a mixed infection has been shown to protect both *Prevotella. melaninogenicus* and other bacteria from penicillin.[50]

Reduced susceptibility to penicillin is more prevalent in the mitis group *streptococci* than in the anginosus group. In susceptibility test to antibiotics, imipenem was the most active molecule tested, confirming its general good activity against oral *streptococci*. Also, third generation cephalosporins such as ceftriaxone and fourth generation cephalosporins like cefepime, showed good activity. Chinolones, glycopeptides, and rifampicin confirmed a good activity against oral *streptococci*.[51]

Macrolides

Macrolide resistance is most commonly due to acquisition of one of a number of erm genes (erythromycin methylases resulting in reduced binding of macrolides to the 50S ribosomal subunit). Resistance to macrolides appears to have a higher prevalence in the "viridans group *streptococci*," anaerobic *streptococci,* and *Prevotella* species. The newer macrolides, clarithromycin and azithromycin, offer improved pharmacokinetics compared to erythromycin.[38,52,53] Erythromycin has adequate activity against the majority of odontogenic pathogens, but up to 50% of *Fusobacterium* are resistant to erythromycin.[54,55] The macrolides should not be considered as first-line therapy in treating odontogenic infections and should be reserved for patients with penicillin allergy.

Metronidazole

Metronidazole is a bactericidal agent that is highly active against most anaerobes, but it lacks activity against aerobic bacteria. Similarly, although it retains activity against penicillin-resistant anaerobic Gram-negative bacilli, it only has moderate activity against microaerophilic Gram-positive cocci. In serious infections, metronidazole is best used in conjunction with penicillin to ensure coverage against aerobic Gram-positive bacteria. However, a combination of two drugs with different dosing schedules may lower patient compliance. The development of resistance to this agent by common odontogenic pathogens is rare.

Clindamycin

Clindamycin has excellent activity against Gram-positive organisms, including anaerobes and β-lactamase producing strains. Low concentrations of the drug are bacteriostatic, but bactericidal activity is achieved clinically with the usual recommended doses. A number of clinical trials have demonstrated clindamycin's efficacy in treating odontogenic infections. Gilmore *et al*. demonstrated comparable activity between clindamycin and penicillin V in the treatment of moderate to severe odontogenic infections.[56] von Konow *et al*. reported similar findings, but the clindamycin group had a shorter duration of fever, pain, and swelling.[57] In one study, moxifloxacin was significantly more effective in reducing pain at days 2-3 of therapy than clindamycin.[58] Clindamycin

has recently been considered for the management of odontogenic infections because of the bacterial susceptibility to this drug, great oral absorption, low emergence of bacterial resistance and good antibiotic levels in bone.

NEED OF GOOD QUALITY CLINICAL TRIALS

There is a lack of sufficient evidence to support the use of one antibiotic regimen over another or to indicate one treatment modality over another. Clinical trials in the treatment of the dental abscess are often flawed in design, limiting validity and applicability of results. Many studies are inadequately blinded and do not have appropriate inclusion criteria. Measurement of the primary outcome is also faulty due to lack of standardization. Some studies rely on clinical assessment of relatively crude parameters that is failure, slight improvement, cure[56,59,60] while others a combination of both patient responses and clinical examination with well-defined but subjective criteria.[15,57,61,62] This has resulted in patients receiving multiple surgical and medical interventions making it impossible to analyze the relative contributions of each intervention to the success of treatment.

Nonetheless, there are a number of recommendations which can be suggested based on the current evidence. If empirical antibiotics are required, the following may be considered. Amoxicillin remains the antimicrobial of first choice. If local patterns of antimicrobial resistance indicate a high prevalence of resistance to amoxicillin then the use of either metronidazole[27] or amoxicillin in combination with clavulanic acid[63] should be considered as alternatives. Clindamycin remains an alternative in individuals who are allergic to the penicillin group of antibiotics.[56,64]

CONCLUSION

Dental abscess and its complications position a substantial burden on individuals, communities, and the health-care system; hence, early diagnosis and appropriate intervention are extremely important. Determination of various host and environmental factors that put an individual at risk for development of dental abscess, influence the spread of infection from a localized collection at the apex of a tooth to a cellulitis and further life-threatening sepsis would aid treatment decisions. Increased reliance on novel molecular techniques has enriched our knowledge of the diverse polymicrobial collection that constitutes a dental abscess. At present, there is no consensus over the gold standard treatment as evidenced by the wide variety of surgical protocols and prescription of antibiotic. Nevertheless, available data suggest that at present most isolates are still susceptible to first-line β-lactam agents. Antimicrobials should be reserved for patients with evidence of cellulitis and signs of sepsis. Most of the evidence pointing toward a key role for prompts surgical intervention and timely review.

REFERENCES

1. Shu M, Wong L, Miller JH, Sissons CH. Development of multi-species consortia biofilms of oral bacteria as an enamel and root caries model system. Arch Oral Biol 2000;45:27-40.
2. Nair PN. Pathogenesis of apical periodontitis and the causes of endodontic failures. Crit Rev Oral Biol Med 2004;15:348-81.
3. Clarke JH. Toothaches and death. J Hist Dent 1999;47:11-3.
4. Carter L, Starr D. Alarming increase in dental sepsis. Br Dent J 2006;200:243.
5. Boykin MJ, Gilbert GH, Tilashalski KR, Shelton BJ. Incidence of endodontic treatment: A 48-month prospective study. J Endod 2003;29:806-9.
6. Azodo CC, Chukwumah NM, Ezeja EB. Dentoalveolar abscess among children attending a dental clinic in Nigeria. Odontostomatol Trop 2012;35:41-6.
7. Kaur J. Dental education and oral health problems in India. Indian J Dent Educ 2009;2:167-71.
8. Agarwal V, Khatri M, Singh G, Gupta G, Marya C, Kumar V. Prevalence of periodontal diseases in India. J Oral Health Community Dent 2010;4:7-16.
9. Goldman AS, Yee R, Holmgren CJ, Benzian H. Global affordability of fluoride toothpaste. Global Health 2008;4:7.
10. Huang TT, Liu TC, Chen PR, Tseng FY, Yeh TH, Chen YS. Deep neck infection: Analysis of 185 cases. Head Neck 2004;26:854-60.
11. Flynn TR, Shanti RM, Hayes C. Severe odontogenic infections, part 2: Prospective outcomes study. J Oral Maxillofac Surg 2006; 64:1104-13.
12. Siqueira JF Jr, Rôças IN. Diversity of endodontic microbiota revisited. J Dent Res 2009;88:969-81.
13. Khemaleelakul S, Baumgartner JC, Pruksakorn S. Identification of bacteria in acute endodontic infections and their antimicrobial susceptibility. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2002;94:746-55.
14. Külekçi G, Inanç D, Koçak H, Kasapoglu C, Gümrü OZ. Bacteriology of dentoalveolar abscesses in patients who have received empirical antibiotic therapy. Clin Infect Dis 1996;23:S51-3.
15. Fazakerley MW, McGowan P, Hardy P, Martin MV. A comparative study of cephradine, amoxycillin and phenoxymethylpenicillin in the treatment of acute dentoalveolar infection. Br Dent J 1993;174:359-63.

16. Reader CM, Boniface M, Bujanda-Wagner S. Refractory endodontic lesion associated with *Staphylococci* aureus. J Endod 1994;20:607-9.
17. Brook I, Frazier EH, Gher ME. Aerobic and anaerobic microbiology of periapical abscess. Oral Microbiol Immunol 1991;6:123-5.
18. Spijkervet FK, Vissink A, Raghoebar GM. The odontogenic abscess. Aetiology, treatment and involvement in the orofacial region. Ned Tijdschr Tandheelkd 2004;111:120-7.
19. Goumas PD, Naxakis SS, Papavasiliou DA, Moschovakis ED, Tsintsos SJ, Skoutelis A. Periapical abscesses: Causal bacteria and antibiotic sensitivity. J Chemother 1997;9:415-9.
20. Jacinto RC, Gomes BP, Shah HN, Ferraz CC, Zaia AA, Souza-Filho FJ. Incidence and antimicrobial susceptibility of *Porphyromonas gingivalis* isolated from mixed endodontic infections. Int Endod J 2006;39:62-70.
21. Kuriyama T, Absi EG, Williams DW, Lewis MA. An outcome audit of the treatment of acute dentoalveolar infection: Impact of penicillin resistance. Br Dent J 2005;198:759-63.
22. Tomazinho LF, Avila-Campos MJ. Detection of *Porphyromonas gingivalis*, *Porphyromonas endodontalis*, *Prevotella* intermedia, and *Prevotella* nigrescens in chronic endodontic infection. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007;103:285-8.
23. Gomes BP, Jacinto RC, Pinheiro ET, Sousa EL, Zaia AA, Ferraz CC, *et al*. Molecular analysis of *Filifactor alocis*, *Tannerella forsythia*, and *Treponema denticola* associated with primary endodontic infections and failed endodontic treatment. J Endod 2006;32:937-40.
24. Gomes BP, Pinheiro ET, Gadê-Neto CR, Sousa EL, Ferraz CC, Zaia AA, *et al*. Microbiological examination of infected dental root canals. Oral Microbiol Immunol 2004;19:71-6.
25. Sassone LM, Fidel RA, Faveri M, Guerra R, Figueiredo L, Fidel SR, *et al*. A microbiological profile of symptomatic teeth with primary endodontic infections. J Endod 2008;34:541-5.
26. Baumgartner JC, Siqueira JF Jr, Xia T, Róças IN. Geographical differences in bacteria detected in endodontic infections using polymerase chain reaction. J Endod 2004;30:141-4.
27. Roche Y, Yoshimori RN. *In-vitro* activity of spiramycin and metronidazole alone or in combination against clinical isolates from odontogenic abscesses. J Antimicrob Chemother 1997;40:353-7.
28. Ledezma-Rasillo G, Flores-Reyes H, Gonzalez-Amaro AM, Garrocho-Rangel A, Ruiz-Rodriguez Mdel S, Pozos-Guillen AJ. Identification of cultivable microorganisms from primary teeth with necrotic pulps. J Clin Pediatr Dent 2010;34:329-33.
29. Siqueira JF Jr, Rôças IN. *Treponema* species associated with abscesses of endodontic origin. Oral Microbiol Immunol 2004;19:336-9.
30. Sakamoto M, Rôças IN, Siqueira JF Jr, Benno Y. Molecular analysis of bacteria in asymptomatic and symptomatic endodontic infections. Oral Microbiol Immunol 2006;21:112-22.
31. Siqueira JF Jr, Rôças IN. *Pseudoramibacter alactolyticus* in primary endodontic infections. J Endod 2003;29:735-8.
32. Siqueira JF Jr, Rôças IN. Detection of *Filifactor alocis* in endodontic infections associated with different forms of periradicular diseases. Oral Microbiol Immunol 2003;18:263-5.
33. Siqueira JF Jr, Rôças IN. Simultaneous detection of *Dialister pneumosintes* and *Filifactor alocis* in endodontic infections by 16S rDNA-directed multiplex PCR. J Endod 2004;30:851-4.
34. Siqueira JF Jr, Rôças IN. Nested PCR detection of *Centipeda periodontii* in primary endodontic infections. J Endod 2004;30:135-7.
35. Rôças IN, Siqueira JF Jr. Detection of novel oral species and phylotypes in symptomatic endodontic infections including abscesses. FEMS Microbiol Lett 2005;250:279-85.
36. Siqueira JF Jr, Rôças IN. Catonella morbi and Granulicatella adiacens: New species in endodontic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102:259-64.
37. Fowell C, Igbokwe B, MacBean A. The clinical relevance of microbiology specimens in orofacial abscesses of dental origin. Ann R Coll Surg Engl 2012;94:490-2.
38. Kuriyama T, Nakagawa K, Karasawa T, Saiki Y, Yamamoto E, Nakamura S. Past administration of beta-lactam antibiotics and increase in the emergence of beta-lactamase-producing bacteria in patients with orofacial odontogenic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000;89:186-92.
39. Niazi SA, Clarke D, Do T, Gilbert SC, Mannocci F, Beighton D. *Propionibacterium acnes* and *Staphylococcus epidermidis* isolated from refractory endodontic lesions are opportunistic pathogens. J Clin Microbiol 2010;48:3859-69.
40. González-García R, Risco-Rojas R, Román-Romero L, Moreno-García C, López García C. Descending necrotizing mediastinitis following dental extraction. Radiological features and surgical treatment considerations. J Craniomaxillofac Surg 2011;39:335-9.
41. Jarboui S, Jerraya H, Moussi A, Ben Moussa M, Marrakchi M, Kaffel N, *et al*. Descending necrotizing mediastinitis of odontogenic origin. Tunis Med 2009;87:770-5.
42. Lewis MA, MacFarlane TW, McGowan DA. A microbiological and clinical review of the acute dentoalveolar abscess. Br J Oral Maxillofac Surg 1990;28:359-66.
43. Blome B, Braun A, Sobarzo V, Jepsen S. Molecular identification and quantification of bacteria from endodontic infections using real-time polymerase chain reaction. Oral Microbiol Immunol 2008;23:384-90.
44. Riggio MP, Lennon A. Development of a novel PCR assay for detection of *Prevotella* oris in clinical specimens. FEMS Microbiol Lett 2007;276:123-8.
45. Siqueira JF Jr, Rôças IN, Souto R, de Uzeda M, Colombo AP. Actinomyces species, *streptococci*, and *Enterococcus faecalis* in primary root canal infections. J Endod 2002;28:168-72.
46. Snydman DR, Jacobus NV, McDermott LA, Golan Y, Goldstein EJ, Harrell L, *et al*. Update on resistance of *Bacteroides fragilis* group and related species with special attention to carbapenems 2006-2009. Anaerobe 2011;17:147-51.
47. Snydman DR, Jacobus NV, McDermott LA, Ruthazer R, Golan Y, Goldstein EJ, *et al*. National survey on the susceptibility of *Bacteroides fragilis* group: Report and analysis of trends in the United States from 1997 to 2004. Antimicrob Agents Chemother 2007;51:1649-55.
48. Boyanova L, Kolarov R, Gergova G, Dimitrova L, Mitov I. Trends in antibiotic resistance in *Prevotella* species from patients of the University Hospital of Maxillofacial Surgery, Sofia, Bulgaria, in 2003-2009. Anaerobe 2010;16:489-92.

49. Heimdahl A, von Konow L, Nord CE. Isolation of beta-lactamase-producing *Bacteroides* strains associated with clinical failures with penicillin treatment of human orofacial infections. Arch Oral Biol 1980;25:689-92.
50. Hackman AS, Wilkins TD. Influence of pencillinase production by strains of *Bacteroides melaninogenicus* and *Bacteriodes oralis* on pencillin therapy of an experimental mixed anaerobic infection in mice. Arch Oral Biol 1976;21:385-9.
51. Pasquantonio G, Condò S, Cerroni L, Bikiqu L, Nicoletti M, Prenna M, *et al*. Antibacterial activity of various antibiotics against oral *streptococci* isolated in the oral cavity. Int J Immunopathol Pharmacol 2012;25:805-9.
52. Kuriyama T, Karasawa T, Nakagawa K, Saiki Y, Yamamoto E, Nakamura S. Bacteriologic features and antimicrobial susceptibility in isolates from orofacial odontogenic infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2000; 90:600-8.
53. Soares GM, Figueiredo LC, Faveri M, Cortelli SC, Duarte PM, Feres M. Mechanisms of action of systemic antibiotics used in periodontal treatment and mechanisms of bacterial resistance to these drugs. J Appl Oral Sci 2012;20:295-309.
54. Karlowsky J, Ferguson J, Zhanel G. A review of commonly prescribed oral antibiotics in general dentistry. J Can Dent Assoc 1993;59:292-4, 297-300.
55. Senhorinho GN, Nakano V, Liu C, Song Y, Finegold SM, Avila-Campos MJ. Occurrence and antimicrobial susceptibility of *Porphyromonas* spp. and *Fusobacterium* spp. in dogs with and without periodontitis. Anaerobe 2012;18:381-5.
56. Gilmore WC, Jacobus NV, Gorbach SL, Doku HC, Tally FP. A prospective double-blind evaluation of penicillin versus clindamycin in the treatment of odontogenic infections. J Oral Maxillofac Surg 1988;46:1065-70.
57. von Konow L, Köndell PA, Nord CE, Heimdahl A. Clindamycin versus phenoxymethylpenicillin in the treatment of acute orofacial infections. Eur J Clin Microbiol Infect Dis 1992;11:1129-35.
58. Cachovan G, Böger RH, Giersdorf I, Hallier O, Streichert T, Haddad M, *et al*. Comparative efficacy and safety of moxifloxacin and clindamycin in the treatment of odontogenic abscesses and inflammatory infiltrates: A phase II, double-blind, randomized trial. Antimicrob Agents Chemother 2011;55:1142-7.
59. Adriaenssen CF. Comparison of the efficacy, safety and tolerability of azithromycin and co-amoxiclav in the treatment of acute periapical abscesses. J Int Med Res 1998;26:257-65.
60. Hanna CB Jr. Cefadroxil in the management of facial cellulitis of odontogenic origin. Oral Surg Oral Med Oral Pathol 1991; 71:496-8.
61. Fouad AF, Rivera EM, Walton RE. Penicillin as a supplement in resolving the localized acute apical abscess. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:590-5.
62. Paterson SA, Curzon ME. The effect of amoxycillin versus penicillin V in the treatment of acutely abscessed primary teeth. Br Dent J 1993;174:443-9.
63. Lewis MA, Carmichael F, MacFarlane TW, Milligan SG. A randomised trial of co-amoxiclav (Augmentin) versus penicillin V in the treatment of acute dentoalveolar abscess. Br Dent J 1993;175:169-74.
64. Mangundjaja S, Hardjawinata K. Clindamycin versus ampicillin in the treatment of odontogenic infections. Clin Ther 1990;12:242-9.

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