Serious complications of endodontic infections: Some cautionary tales

L. J. Walsh*

Abstract

While endodontic (dentoalveolar) abscesses can cause significant morbidity, in susceptible individuals they can pose life-threatening problems. This paper provides an overview of the more serious sequelae of endodontic abscesses, and provides examples of 'high risk' situations in practice in which these serious complications are more likely to occur.

Key words: Abscess, infection, periapical pathology.

(Received for publication November 1995. Revised May 1996. Accepted May 1996.)

Introduction

An endodontic (dentoalveolar or periapical) abscess can be defined as a localized, circumscribed, purulent area of inflammation arising in the periodontal ligament space. It is the most common of the three types of dental abscess (dentoalveolar, periodontal, and gingival). These three entities may resemble each other from a clinical standpoint, and all may exist in acute and chronic forms.

Chronic endodontic abscesses occur with long standing low grade infection and can frequently be asymptomatic, making detection difficult. However, exacerbation of a chronic abscess is accompanied by signs and symptoms which attract the patient's attention, and these initiate clinical intervention.

Development of the endodontic abscess

The endodontic abscess is the most important sequel to death of the dental pulp. Pulpal death may be caused by a range of physical and biological insults, including dental caries, thermal damage, mechanical trauma, tooth wear from grinding, microleakage of dental restorations, or tooth fracture. Injury to the pulp typically induces a local chronic abscess with advancing pulpal necrosis which extends through the root canal system. Bacterial infection of the necrotic pulp tissues and root canal

system of the tooth progresses rapidly to involve the periapical tissues, which become infected and chronically inflamed. The relationship between infection of the pulp and the development of infection in the periapical region in humans has been demonstrated convincingly.²

Many types of bacteria have been implicated in endodontic abscesses. However, prior to the work of Möller et al.,³ widely variable results were reported which have since been attributed to technical factors in sampling and culturing. Möller, who developed a technique for sampling and culturing anaerobic microbes from the root canal system of teeth, demonstrated a greater incidence of obligate anaerobic microorganisms than had been reported previously. Subsequent studies^{2,4} demonstrated similar high incidences of anaerobic bacteria.

In a more recent study, Brook et al. 5 sampled the exudate from endodontic abscesses in 39 patients, and demonstrated anaerobic bacteria in the majority of cases (94 per cent). Polymicrobial anaerobic and aerobic flora have been recovered from endodontic abscesses. The predominant anaerobes are Bacteroides spp, Porphyromonas gingivalis and P. endodontalis, Fusobacterium spp (especially F. nucleatum), Prevotella spp (P. intermedia and P. oralis), Peptostreptococcus spp and Actinomyces spp, 46 while the milleri group (Streptococcus anginosus, S. constellatus, and S. intermedius) are the predominant streptococci. Acute exacerbations of chronic infections are associated frequently with specific anaerobes (particularly P. gingivalis and P. endodontalis).

Clinical features

Endodontic and periodontal abscesses may resemble each other clinically, differing only in their point of origin and specific path of infection. Both periapical and periodontal abscesses may occur together on the one tooth and treatment of these lesions requires combined endodontic and periodontic therapy.

In most instances, periapical abscesses occur singly. The involved tooth is extremely tender to

^{*}Associate Professor, Department of Dentistry, The University of Queensland Dental School.

percussion and is extruded and often hypermobile. Marked lymphadenopathy is common, as is facial swelling and extension of the infection along tissue planes to points of drainage (either intra-oral or extra-oral). Fever and malaise occur commonly, and this is due to the action of the endogenous pyrogen interleukin-1, which is found in high levels in exudates from periapical abscesses.⁸

Treatment

The correct diagnosis is reached via the use of radiographs and pulp tests. Surgical drainage is established by incision of any soft tissue swelling, accompanied by either extraction of the tooth or extirpation of the pulp and endodontic therapy as indicated clinically. Use of antimicrobials (e.g., 250 mg amoxycillin plus 125 mg clavulanic acid tds, or metronidazole 200 mg tds) is indicated when pyrexia and other systemic signs are present. If amoxycillin is selected, the therapy should account for the fact that many anaerobes of importance in periapical abscesses produce beta-lactamase. Finally, it should be emphasized that antimicrobial therapy without surgical drainage is not an effective therapy over the long term.

Endodontic treatment aims to eliminate microorganisms from the root canal system of the affected tooth. The anaerobic bacteria responsible for the abscess are present within the apical area of the root canal system, but are usually not found in the area of the apical foramen or on the surface of the root apex. Thus, correct biological preparation of the root canal system itself is able to bring about resolution of the infection in approximately 90 per cent of cases. In the remaining cases, the presence of bacteria within the lesion or the persistence of pro-inflammatory bacterial products (such as lipopolysaccharides) within the apical root structure are implicated as causal factors. Bacteria belonging to the genera Actinomyces and Arachnia may survive for long periods of time in periapical sites and may prevent healing.11

Inability to obtain treatment

Where appropriate clinical management is unavailable, aggravation of dental abscesses can be expected to occur. This is important from three standpoints. First, the destruction of supporting tissues may result in the loss of the tooth, with the subsequent need for tooth replacement. Secondly, the infectious process may spread to involve the bone of the mandible or maxilla, and the associated tissue spaces of the head and neck. Such infections pose significant risks of morbidity and mortality. Ludwig's angina, an aggressive infection of the submandibular, sublingual, and submental fascial spaces can occur following the development of a dental abscess. Airway compromise, requirements for parenteral antibiotics, and the need to use either CT or MRI to image the tissue space

Table 1. Complications of endodontic abscesses

Osteomyelitis of the mandible 16 Maxillary sinusitis and orbital abscess¹⁷ Wound botulism18 Ludwig's angina19 Necrotizing fasciitis20 Cavernous sinus thrombosis 21 Persistent pyrexia of unknown origin²² Septicaemia – Streptococcus milleri and Pseudomonas spp²³ Septicaemia with disseminated intravascular coagulation²⁴ Pulmonary abscess²⁵ Pyogenic hepatic abscess²⁶ Brain abscess² Brain abscess and acute meningitis - Actinomyces viscosus²⁸ Paraspinal abscess and paraplegia²⁹ Bacterial endocarditis and splenic abscess 30 Mediastinal abscess and pneumonia³¹

infections prior to surgical intervention are major problems in the clinical management of such cases. 12,13

Cutaneous sinus tracts on the chin and cheek frequently are due to untreated endodontic abscesses, ¹⁴ and surgical management of these tracts can be disfiguring. Diagnostic errors can result in multiple excisions, biopsies, and ineffective long-term antibiotic therapy. Patients may require excision of the fistula once the abscess has been successfully treated by endodontic therapy or extraction. ¹⁵

Thirdly, the anatomical proximity of periapical regions to the bloodstream can facilitate bacteraemia and systemic spread of bacterial by-products and immune complexes from the abscess. Moreover, the manipulations involved in tooth extraction may cause translocation of microorganisms to the bloodstream, with circulation throughout the body. In these cases, microorganisms from the oral flora may be cultivated from distant lesions, such as brain abscesses.

Serious outcomes of untreated endodontic

A range of significant complications may arise from untreated dental abscesses. Complications of endodontic abscesses reported in the recent medical and dental literature (1991-1996) are listed in Table 1.¹⁶⁻³¹ Two cases which illustrate problems which can arise following inappropriate management of endodontic infections are presented below. These are representative of more than 20 cases encountered by the author in his hospital practice.

Case 1

A 40 year old male presented to the accident and emergency unit with protracted fevers and spontaneous severe haemorrhages from the oral cavity and urinary and gastrointestinal tracts. Six days previously he had had five mandibular teeth extracted by his general dental practitioner because of multiple endodontic abscesses. The wounds had

not been sutured. On examination, a massive enlargement of the oral wound sites was noted, and there was concern that the oral airway would be compromised. A provisional diagnosis of acute leukaemia was rendered. Subsequent haematological analysis revealed acute myeloid leukaemia in blast crisis. Emergency treatment comprised platelet support and three cycles of leukapheresis, the latter to decrease the tumour load. Empiric antimicrobials were commenced. These comprised vancomycin and ceftazidime, together with high dose metronidazole (500 mg tds iv). The patient's condition stabilized over the following three days, and remission induction chemotherapy was then commenced. The leukaemic infiltrates resolved completely over the ensuing three weeks, and spontaneous closure of the extraction wound sites occurred by four weeks.

Case 2

A 53 year old male with chronic myeloid leukaemia in accelerated phase had been scheduled for an allogeneic bone marrow transplant, using an overseas donor. One week prior to admission for marrow ablative conditioning he underwent an apicectomy procedure on the distobuccal root of a maxillary first molar. The tooth had been endodontically treated one year previously; however radiographically there was evidence of persistent infection at the site. The patient underwent this procedure against the advice of the dental consultant. On the day of scheduled admission a massive swelling of the right maxillary region was present. Intraoral examination indicated that the surgical site was infiltrated extensively with leukaemic cells, with significant infection extending into the maxilla itself. High dose metronidazole therapy was implemented immediately, and the patient's marrow conditioning (and subsequent transplant) were delayed for 10 days to allow the infection to resolve. The transplant was then able to proceed, and no additional complications were experienced.

Discussion

These two brief cases illustrate several important aspects regarding the management of acute and chronic endodontic infections. In Case 1, the presentation of multiple endodontic abscesses should have raised suspicions regarding the immune status of the patient. Multiple dental infections are relatively common in patients with acute leukaemia. Oncologists in the author's hospital unit consider that in approximately one third of neutropenic patients with a pyrexia of unknown origin, there is a dental focus, and this typically is an endodontic lesion. While examination of such patients frequently reveals heavily carious teeth or fistulae, conventional endodontic therapy or extractions cannot be undertaken on such patients for medical reasons. It is therefore necessary to manage the infections on an emergency basis with antimicrobials. According to the author's experience, in such febrile patients, a good response to metronidazole can be expected within 24 hours.

In Case 1, the infections present in the periapical regions prior to the extractions had not only persisted but had developed into a full-blown sepsis by the time the patient presented to hospital. The supervening infiltration of the wound sites by leukaemic cells posed a serious problem in terms of airway management. Once again, the selection of metronidazole as an empirical antimicrobial in this instance was based upon its effectiveness against the Gram-negative anaerobes implicated in periapical pathology. If an adequate clinical response is not seen in 24 hours, then the antimicrobial therapy is altered appropriately. Unfortunately, because of the fastidious nature of many of the Gram-negative anaerobes implicated in these infections, it is not always possible to successfully culture them from peripheral blood to allow conventional sensitivity tests to be undertaken.

In Case 2, the persistent infection present in the periapical region prior to surgery could have been stabilized effectively by high potency antimicrobials during the transplant procedure so as not to pose a major risk to the patient during the neutropenic period post-transplant. The surgery could have then been undertaken once the immune system of the patient had been reconstituted to normal levels (approximately six months post-transplant). In the present case, the surgical intervention was carried out without concurrent antimicrobial therapy, and was not only ineffective in dealing with the infection, but very likely assisted in its extension into the maxilla proper. To this was added the additional problem of infiltration of the wound site by myeloid leukaemic cells, an event which was predictable

Table 2. Disorders of the immune response in which dissemination of infection is more likely

- a. Low neutrophil count
 Cancer treatment with chemotherapy or radiotherapy
 Cyclic neutropenia
 Cytotoxic therapy
 Panism chemic routropenia
 - Benign chronic neutropenia
 Depressed neutrophil function
- Depressed neutropnii function
 Down's syndrome
 Poorly controlled diabetes mellitus
 Crohn's disease

Chediak-Higashi syndrome

c. Generalized depression of cell mediated immunity Leukaemias

Advanced HIV disease Advanced malignancy

Bone marrow transplant recipients

Organ transplant recipients

Treatment of auto-immune diseases with immunosuppressive agents such as cyclosporin A

d. Poor tissue healing

Localized radiotherapy (e.g., for head and neck cancer)
Malnutrition

Poorly controlled diabetes

under these circumstances. The final outcome in this case was favourable; however if the infection had not been controlled successfully, significant destruction of the maxilla and dissemination of the infection would have been likely.

Several risk situations can be identified in which inappropriate management of endodontic infections is more likely to result in major complications. Because of the protective role of the immune response in limiting the spread of infection, ³² patients affected by a variety of disorders of the immune system (Table 2) are more likely to develop complications from untreated or improperly treated endodontic abscesses.

Conclusions

Endodontic abscesses are a relatively common pathological entity, and there is a tendency for familiarity with these lesions to disguise their potential for serious complications. Clinicians should be diligent in their treatment and follow-up of endodontic abscesses, and should be aware of the potential for adverse events in patients whose immune system is compromised. In particular, consultation with medical or dental practitioners experienced in the care of immune compromised patients is suggested prior to undertaking complex or multiple surgical procedures.

References

- Cohen S, Burns RC. Pathways of the pulp. 6th edn. St Louis: Mosby, 1994:21-6, 337-40.
- Nair P, Sjögren U, Krey G, Kahnberg K, Sundqvist G. Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: A long term light and electron microscopic follow-up study. J Endod 1987;16:580-05
- 3. Möller A, Fabricius L, Dahlen G, Ohman A, Heyden G. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. Scand J Dent Res 1981;89:475-84
- Trowbridge HO, Stevens BH. Microbiologic and pathologic aspects of pulpal and periapical disease. Curr Opin Dent 1992:2:85-92.
- Brook I, Frazier E, Gher M. Aerobic and anaerobic microbiology of periapical abscesses. Oral Microbiol Immunol 1991;6:123-5.
- Wasfy MO, McMahon KT, Minah GE, Falkler WA. Microbiological evaluation of periapical infections in Egypt. Oral Microbiol Immunol 1992;7:100-5.
- Fisher LE, Russell RR. The isolation and characterization of milleri group streptococci from dental periapical abscesses. J Dent Res 1993;72:1191-3.
- 8. Matsuo T, Ebisu S, Nakanishi T, Yonemura K, Harada Y, Okada H. Interleukin-1 alpha and interleukin-1 beta periapical exudates of infected root canals: correlations with the clinical findings of the involved teeth. J Endod 1994;20:432-5.
- 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.
- 10. Finegold SM, Strong CA, McTeague M, Marina M. The importance of black-pigmented gram-negative anaerobes in human infections. FEMS Immunol Med Microbiol 1993;6:77-82.

- 11. Happonen R. Periapical actinomycosis: a follow-up study of 16 surgically treated cases. Endod Dent Traumatol 1986;2:205-6.
- Welsh LW, Welsh JJ, Kelly JJ. Massive orofacial abscesses of dental origin. Ann Otol Rhinol Laryngol 1991;100:768-73.
- Krishnan V, Johnson JV, Helfrick JF. Management of maxillofacial infections: a review of 50 cases. J Oral Maxillofac Surg 1993;51:868-73.
- 14. Foster KH, Primack PD, Kulild JC. Odontogenic cutaneous sinus tract. J Endod 1992;18:304-6.
- Marasco PV, Taylor RG, Marks MW, Argenta LC. Dentocutaneous fistula. Ann Plast Surg 1992;29:205-10.
- Taher AA. Osteomyelitis of the mandible in Tehran, Iran. Analysis of 88 cases. Oral Surg Oral Med Oral Pathol 1993;76:28-31.
- 17. Allan BP, Egbert MA, Myall RW. Orbital abscess of odontogenic origin. Case report and review of the literature. Int J Oral Maxillofac Surg 1991;20:268-70.
- 18. Weber JT, Goodpasture HC, Alexander H, Werner SB, Hatheway CL, Tauxe RV. Wound botulism in a patient with a tooth abscess: case report and review. Clin Infect Dis 1993;16:635-9.
- Irani BS, Martin-Hirsch D, Lannigan F. Infection of the neck spaces: a present day complication. J Laryngol Otol 1992;106:455-8.
- Rapoport Y, Himelfarb MZ, Zikk D, Bloom J. Cervical necrotizing fasciitis of odontogenic origin. Oral Surg Oral Med Oral Pathol 1991;72:15-8.
- 21. Yun MW, Hwang CF, Lui CC. Cavernous sinus thrombosis following odontogenic and cervicofacial infection. Eur Arch Otorhinolaryngol 1991;248:422-4.
- 22. Siminoski K. Persistent fever due to occult dental infection: case report and review. Clin Infect Dis 1993;16:550-4.
- 23. Cheatham BD, Henry RJ. A dental complication involving pseudomonas during chemotherapy for acute lymphoblastic leukemia. J Clin Pediatr Dent 1994;18:215-7.
- Currie WJ, Ho V. An unexpected death associated with an acute dentoalveolar abscess – report of a case. Br J Oral Maxillofac Surg 1993;31:296-8.
- Balloul H, de Vitry N, Cohen R, Reinert P. Septicemia due to Streptococcus miller i with pulmonary complications. Arch Pediatr 1994:1:264-7.
- Crippin JS, Wang KK. An unrecognized etiology for pyogenic hepatic abscesses in normal hosts: dental disease. Am J Gastroenterol 1992;87:1740-3.
- 27. Twomey CR. Brain abscess: an update. J Neurosci Nurs 1992;24:34-9.
- Vallee L, Pinton F, Martin BH, Debray P, Vamecq J, Hladky JP, Nuyts JP. Brain abscess complicating dental caries in children. Arch Pediatr 1994;1:166-9.
- 29. Larkin EB, Scott SD. Metastatic paraspinal abscess and paraplegia secondary to dental extraction. Br Dent J 1994;177:340-2.
- Robinson SL, Saxe JM, Lucas CE, Arbulu A, Ledgerwood AM, Lucas WF. Splenic abscess associated with endocarditis. Surgery 1992;112:781-6.
- 31. Petrone JA. Mediastinal abscess and pneumonia of dental origin. J NJ Dent Assoc 1992;63:19-23.
- Seymour GJ, Savage NW, Walsh LJ. Immunology: An introduction for the health sciences. Sydney: McGraw Hill, 1995:127-42.

Address for correspondence/reprints:

Department of Dentistry,
The University of Queensland Dental School,
Turbot Street,
Brisbane, Queensland 4000.