Periodontal Abscess: A Review

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ABSTRACT

Periodontal abscess is the third most frequent dental emergency, representing 7–14% of all the dental emergencies. Numerous etiologies have been implicated: exacerbations of the existing disease, post-therapy abscesses, the impaction of foreign objects, the factors altering root morphology etc. The diagnosis is done after analysis of the signs and symptoms and by the using supplemental diagnostic aids. Evidences suggest that the micro flora which are related to periodontal abscesses are not specific and that they are usually dominated by gram negative anaerobe, rods etc. The treatment of the periodontal abscess has been a challenge for many years. Today three therapeutic approaches are being discussed in dentistry that include, drainage and debridement, systemic antibiotics and periodontal surgical procedures which are applied in the chronic phase of the disease. Antibiotics like Penicillin, Metronidazole, Tetracyclines and Clindamycin are the drugs of choice. This review focuses on the classification, etiology and clinical characteristics of periodontal abscesses and its management in the clinical practice.

Keywords: Periodontal abscess, incision and drainage, antibiotics, periodontal surgery.

1. INTRODUCTION

Periodontal Abscess is a localized purulent infection in the periodontal pocket that may lead to the destruction of periodontal ligament and alveolar bone. The international conference on research in the biology of periodontal disease in 1977 defined a periodontal abscess as, “an acute, destructive process in the periodontium resulting in localized collection of pus communicating with oral cavity through the gingival sulcus or other periodontal sites and not arising from the tooth pulp.”[1]

The important characteristics of the periodontal abscess include: a localized accumulation of pus in the gingival wall of the periodontal pockets; usually occurring on the lateral aspect of the tooth; the appearance of edematous red and shiny gingiva; may have a dome like appearance or may come to a distinct point. These conditions have to be promptly managed, failure of which could lead to loss of teeth and danger of cellulitis in susceptible patients. Depending on the nature and course of the periodontal abscess an immediate attention is required to relieve pain and systemic complications. Moreover, the presence of an abscess may also modify the prognosis of the involved tooth and in many cases, may be responsible for extraction of tooth. Therefore accurate diagnosis and the immediate treatment of the abscess is an important step in the management of periodontal abscess.

2. ETIOLOGY OF PERIODONTAL ABSCESS

The etiology of periodontal abscess is at times difficult to determine basically due to the size of the abscess, the possible condition that can result in a purulent infection and the vast number of potentially infective microbiota present.

Following are the sum of factors causing periodontal abscess:

a. Tortuous periodontal pocket especially associated with furcation defects. These can eventually become isolated and can favor formation of an abscess.

b. Closure of margins of periodontal pocket may lead to extension of the infection into the surrounding supporting periodontal tissue due to the pressure of the suppuration inside the obstructed periodontal pocket. Fibrin secretions, leading to the local accumulation of pus may favor the closure of gingival margin to the tooth surface. [2]

c. Changes in composition of the micro flora, bacterial virulence or defect in host defense could also make the pocket lumen inefficient to drain the increased suppuration. [3]

d. After procedures like scaling and root planning where calculus is dislodged and pushed into the soft periodontal tissue. It may also be due to inadequate scaling which will allow calculus to remain in the deepest periodontal pocket area while the resolution of the inflammation at the coronal pocket area will occlude the normal drainage and entrapment of the sub gingival flora in the deepest part of the pocket which cause abscess formation. [4]

e. Treatment with systemic antibiotics without sub gingival debridement in patients with advanced periodontitis leads to a change in the composition of the sub gingival microbiota leading to super infection and abscess formation. [5]

f. As a consequence of perforation of the lateral wall of a tooth by an endodontic instrument during root canal therapy. [6]
3. CLASSIFICATION OF PERIODONTAL ABSCESS [7]

3.1 Based on Etiological Criteria
a. Periodontitis related abscess: When acute infections originate from a biofilm in the deepened periodontal pocket.

b. Non-Periodontitis related abscess: When the acute infections originate from other local source. eg. Foreign body impaction, alteration in root integrity.

3.2 Based on the Course of the Disease
a. Acute periodontal abscess: The abscess develops in a short period of time and lasts for a few days or weeks. An acute abscess often presents as a sudden onset of pain on biting and a deep throbbing pain in a tooth in which the patient has been tending to clench. Associated lymph node enlargement may be present.

b. Chronic periodontal abscess: This is the condition that lasts for a long time and often develop slowly. In The chronic stages a nasty taste and spontaneous bleeding may accompany discomfort. The adjacent tooth is tender to bite on and is sometimes loose. Pus may be present as also may be discharges from the gingival crevice or from a sinus in the mucosa overlying the affected root. Pain is usually of low intensity. Occasionally the abscess may already be draining through one or more sinus tracts into the oral cavity. Often regional lymphadenopathy and occasionally slight elevation of body temperature may also associated due to abscess.

4. MICROBIOLOGY OF PERIODONTAL ABSCESS
a. Bacteroids melaninogenicus subspecies, Fusobacterium subspecies, Vibrio corroders, Capnocytophaga species, Peptococcus species and Peptostreptococcus species.[1]

b. Purulent oral infections are polymicrobial and usually caused by endogenous bacteria, however very few studies have investigated the specific microbiota of periodontal abscesses [8].

c. About 60% of cultured bacteria were strict anaerobes [9].

d. Most frequent type of bacteria was gram negative anaerobic rods and gram positive facultative cocci.

e. In general, gram negatives predominated over gram positive and rods over cocci [10].

f. Reports showed that high percentage of abscesses harbor lactamase producing bacteria [11].

g. Culture studies of periodontal abscesses have revealed high prevalence of the following bacteria:

- Prevotella intermedia- 25-100% [9]
- Porphyromonas gingivalis-55-100% [10]
- Fusobacterium nucleatum -44-65% [12]
- Actinobacillus actinomyctemcomitans-25% [11],[12]
- Camphylobacter rectus- 80% [12]
- Prevotella melaninogenica-22% [12]

5. PATHOGENESIS
After the infiltration of pathogenic bacteria to the periodontium, the bacteria and or bacterial products initiate the inflammatory process consequently activating the inflammatory response. Tissue destruction is caused by the inflammatory cells and their extracellular enzymes. An inflammatory infiltrate is formed, followed by the destruction of the connective tissue. The encapsulation of the bacterial mass occur and pus formation takes place. The lowered tissue resistance and the virulence as well as the number of bacteria present determine the course of infection. The entry of bacteria into the soft tissue wall initiates the formation of the periodontal abscess.

6. INVESTIGATIONS FOR PERIODONTAL ABSCESS

6.1 Radiographs
Dental radiographs (periapical, bitewings and OPG), Gutta percha point placed through sinus might locate the source of the abscess.

6.2 Pulp Vitality Test
Thermal or electrical tests to assess the vitality of the tooth.

6.3 Microbial Test
Sample of pus from the sinus, abscess or purulent material expressed from the gingival crevice could be sent for culture and sensitivity test.

6.4 Others
Assessment of diabetic status through blood glucose and glycosylated hemoglobin.

7. GUIDELINES FOR ASSESSING PROGNOSIS [13]

a. Horizontal mobility more than 1mm.
b. Class II-III furcation involvement of multirooted tooth.
c. Probing pocket depth > 8 mm.
d. Poor response to periodontal therapy.
e. More than 40% alveolar bone loss.

8. DIAGNOSIS OF PERIODONTAL ABSCESS
A periodontal abscess is diagnosed on the basis of chief complaints and history of present illness. Usually, the severity of the pain and distress will differentiate an acute from a chronic abscess. The relevant medical and dental history is mandatory for the proper diagnosis of such cases.
The important point to be considered while taking the history includes:

a. Whether the patient is under the care of a physician or a dentist
b. Whether the patient is presently on any medication or whether he/she has any medical condition that may affect the periodontal diagnosis and treatment.
c. Any previous dental treatment that may affect the diagnosis or the treatment plan
d. The history of smoking is important because heavy smokers can develop a more severe periodontal disease and they do not respond very well to periodontal therapy.

9. DIFFERENTIAL DIAGNOSIS OF PERIODONTAL ABSCESS

9.1 Gingival Abscess
A localized purulent infection that involves the marginal gingiva or interdental papilla. Features that differentiate the gingival abscess from the periodontal abscess are:

i. History of recent trauma;
ii. Localisation to the gingiva;
iii. No periodontal pocketing

9.2 Periapical Abscess
Inflammatory condition characterized by formation of purulent exudate involving the dental pulp or pulpal remnants and the tissues surrounding the apex of a tooth.

Periapical abscess can be differentiated by the following features:

i. Located over the root apex
ii. Non-vital tooth, heavily restored or large filling
iii. Large caries with pulpal involvement
iv. History of sensitivity to hot and cold food
v. No signs / symptoms of periodontal diseases.
vi. Periapical radiolucency on intraoral radiographs.

9.3 Perio-Endo lesion
The Perio-endo lesion usually shows:

i. Severe periodontal disease which may involve the furcation
   i. Severe bone loss close to the apex, causing pulpal infection
   ii. Non-vital tooth which is sound or minimally restored

9.4 Endo-Perio lesion
Endo-perio lesion can be differentiated by:

i. Pulp infection spreading via the lateral canals into the perodontal pockets
ii. Tooth usually non-vital, with periapical radiolucency
iii. Localised deep pocketing

9.5 Cracked tooth Syndrome
Cracked tooth Syndrome can be differentiated by:

i. History of pain on mastication
ii. Crack line noted on the crown.
iii. Vital tooth
iv. Pain upon release after biting on cotton roll, rubber disc or tooth sleuth
v. No relief of pain after endodontic treatment

9.6 Root fracture
Root fracture can be differentiated by the presence of following characteristics.

i. Heavily restored crown
ii. Non-vital tooth with mobility
iii. Post crown with threaded post
iv. Possible fracture line and radiolucency around the root which are visible in periapical radiographs
v. Localised deep pocketing, normally one site only
vi. Might need an open flap exploration to confirm diagnosis

10. TREATMENT OF PERIODONTAL ABSCESS

The principles of management of periodontal abscess are as follows:

1. Local measures
   a. Drainage
   b. Eliminate cause

2. Systemic measures: Antibiotics in conjunction with local measures

The management of a patient with periodontal abscess can divided into three stages:

• Immediate management
• Initial management
• Definitive therapy

10.1 Immediate Management [14]

a. In life threatening infections, hospitalization, supportive therapy together with antimicrobial therapy will be necessary.
b. Depending on the severity of the infection and local signs and symptoms the clinical examination, investigations and initial therapy can be delayed.
c. In non life threatening conditions systemic measures such as oral analgesics and antimicrobial chemotherapy will be sufficient to eliminate systemic symptoms, severe trismus and diffuse spreading infection (facial cellulites).
d. Antibiotics are prescribed empirically after microbiological analysis and antibiotic sensitivity of pus and tissue specimen.
e. The empirical regimens are dependent on the severity of the infection. The common antibiotics used are
Phenoxymethylene penicillin 250 -500 mg qid 5-7 days
Amoxicillin 250 - 500 mg tds 5-7 days
Metronidazole 200 - 400 mg tds 5-7 days
If allergic to penicillin
Erythromycin 250 –500 mg qid 5-7 days
Doxycycline 100mg bd 7-14 days
Clindamycin 150-300 mg qid 5-7 days

10.2 Initial Management:
  a. Irrigation of abscessed pocket with saline or antiseptics [15]
  b. Removal of foreign bodies [16]
  c. Drainage through sulcus with a probe or light scaling of tooth surface [17]
  d. Compression and debridement of soft tissue wall [17]
  e. Oral hygiene instructions
  f. Review after 24-48 hours.

Alternative Treatments [17]
  3. Extraction of teeth with hopeless prognosis

10.3 Definitive Therapy
Definitive treatment following reassessment after initial therapy has to perform to restore, function, esthetics of periodontium & to enable patient maintain periodontal initial therapy has to perform to restore, function, esthetics.

10.3.1 Complications of Periodontal Abscess:[19]
  a. Tooth loss: Periodontal abscesses have been suggested as the main cause for tooth extraction during the phase of supportive periodontal therapy (SPT). A tooth with a history of repeated abscess formation is considered to be a tooth with a questionable prognosis. In a retrospective study, 45% of teeth with periodontal abscesses in a SPT population were extracted.

  b. Dissemination of the infection: A number of publications, mainly case reports, have described different systemic infections in different parts of the body, in which the suspected source of infection was a periodontal abscess.

Two possible sources of dissemination have been described:
  • Dissemination of the bacteria inside the tissues during therapy. A case of pulmonary actinomycosis was related to the treatment of a periodontal abscess, which was ultrasonically scaled 1 month earlier. A case of brain abscess was observed in a healthy patient with a periodontal abscess who was treated with drainage and curettage without systemic antibiotic 2 weeks earlier.
  • Bacterial dissemination through the blood stream due to bacteremia from an untreated abscess. Cellulitis in breast cancer patients have been reported following gingivitis or an abscess, due to transient bacteremia and reduced host defenses (radiation therapy and axillary dissection). A periodontal abscess was associated with the development of a cervical necrotizing fasciitis. A necrotizing cavernositis was thought to be related to a severe periodontal infection, including three periodontal abscesses.

11. SUMMARY
In spite of their long historical recognition, periodontal abscess after present challenge in terms of diagnosis, etiology, treatment and prognosis. The periodontal abscess is the 3rd most frequent dental emergency and it is especially prevalent among untreated periodontal patients and periodontal patients during maintenance. Different etiologies have been proposed, and two main groups can be distinguished, depending on its relation with periodontal pockets. In the case of a periodontitis related abscess, the condition may appear as an exacerbation of a non-treated periodontitis or during the course of periodontal therapy. In non-periodontitis related abscesses, impact of foreign objects and ridicular abnormalities are the two major causes. The abscess micro flora seems to be similar to that of adult periodontitis and it is dominated by gram negative anaerobic rods, including well known periodontal pathogens. Complications and consequences include tooth loss and the spread of the infection to other body sites. Diagnosis and treatment is mainly based on empiricism, review; microbiology; diagnosis; classification; etiology; prevalence.

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**Fig 1:** Periodontal abscess mesial to maxillary right central incisor, copious pus exudate after pocket is probed.

**Fig 2:** Radiograph dipicts the periodontal probe inserted the base of osseous defect

**Fig 3:** The periodontal abscess mesial to maxillary right first central incisor has opened through gingival sulcus. Once the acute symptoms have subsided, definitive treatment can be undertaken.

**Fig 4:** Radiograph 6 months after definitive therapy. Some new bone formation is apparent.