Dentoalveolar infections can be defined as pus-producing (or pyogenic) infections associated with the teeth and surrounding supporting structures, such as the periodontium and the alveolar bone. Other terms for these conditions include periapical abscess, apical abscess, chronic periapical dental infection, dental pyogenic infection, periapical periodontitis and dentoalveolar abscess. The clinical presentation of dentoalveolar infections depends on the virulence of the causative microorganisms, the local and systemic defence mechanisms of the host, and the anatomical features of the region. Depending on the interactions of these factors, the resulting infection may present as:

• an abscess localized to the tooth that initiated the infection
• a diffuse cellulitis that spreads along fascial planes
• a mixture of both.

**Source of microorganisms**

Endogenous oral commensals, usually from the apex of a necrotic tooth or from periodontal pockets as a result of either caries or periodontal disease (Fig. 34.1).

**Dentoalveolar abscess**

A dentoalveolar abscess usually develops by the extension of the initial carious lesion into dentine, and spread of bacteria to the pulp via the dentinal tubules (Figs 34.1 and 34.2). The pulp responds to infection either by rapid acute inflammation involving the whole pulp, which quickly becomes necrosed, or by development of a chronic localized abscess with most of the pulp remaining viable. Other ways in which microbes reach the pulp are:

• by traumatic tooth fracture or pathological exposure due to tooth wear
• by traumatic exposure during dental treatment (iatrogenic)
• through the periodontal membrane (periodontitis and pericoronitis) and accessory root canals
• rarely by anachoresis, i.e. seeding of organisms directly into pulp via the pulpal blood supply during bacteraemia (e.g. tooth extraction at a different site).

**Sequelae**

Once pus formation occurs, it may remain localized at the root apex and develop into either an acute or a chronic abscess, develop into a focal osteomyelitis, or spread into the surrounding tissues (Figs 34.2 and 34.3).

**Direct spread**

1. Spread into the superficial soft tissues may:
   - localize as a soft-tissue abscess (Fig. 34.4)
   - extend through the overlying oral mucosa or skin, producing a sinus linking the main abscess cavity with the mouth or skin
   - extend through the soft tissue to produce a cellulitis.

2. Spread may occur into the adjacent fascial spaces, following the path of least resistance; such spread is dependent on the anatomical relation of the original abscess to the adjacent tissues (Table 34.1). Infection via fascial planes often spreads rapidly and for some distance from the original abscess site, and occasionally may cause severe respiratory distress as a result of occlusion of the airway by oedema (e.g. Ludwig’s angina).

3. Infection may extend into the deeper medullary spaces of alveolar bone, producing a spreading osteomyelitis; this may occur in compromised patients.

4. In maxillary teeth, odontogenic infection may directly spread into the maxillary sinus, especially if the sinus lining and the tooth apex are subjacent, leading to acute or chronic secondary maxillary sinusitis (as opposed to primary sinusitis due to direct sinus infection). Such infection, if not arrested, may rarely spread to the central nervous system, causing serious complications such as subdural empyema, brain abscesses or meningitis.

**Indirect spread**

Other sequelae entail indirect spread via:

• lymphatic routes, to regional nodes in the head and neck region (submental, submandibular, deep cervical, parotid and occipital). Usually, the involved nodes are
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- strict anaerobes are the predominant organisms, and the viridans group streptococci are less common than once thought.

The common species isolated from dentoalveolar abscesses are Prevotella, Porphyromonas and Fusobacterium spp., and anaerobic streptococci; facultative anaerobes are the second largest group, e.g. Streptococcus milleri (Table 34.2). There is evidence that some strictly anaerobic bacteria, especially Porphyromonas gingivalis and Fusobacterium spp., are more likely to cause severe infection than other species, and that synergistic microbial interactions play an important role in the severity of dentoalveolar abscesses.

Collection and transport of pus samples

1. Wherever possible, pus should be collected by needle aspiration or in a sterile container after external incision. Care must be exercised during recapping the syringe after needle aspiration, and a safety device must be used. Also, it is important to drain the residual pus, once the aspirate has been obtained via an appropriate incision (see Chapter 6).

2. If swabs must be used, then a strict aseptic collection technique is required (because of the indigenous flora on mucosal surfaces, it is difficult, if not impossible, to collect uncontaminated samples when intraoral swabs are used for pus collection). When the pus sample is contaminated with saliva or dental plaque during collection, this information must be recorded on the request form.

Management

The specific treatment for any given individual will vary. The major management guidelines entail:

1. draining the pus
2. removing the source of infection
3. prescribing antibiotics – probably not required for the majority of localized abscesses, although it may be necessary:
   - when drainage cannot be established immediately
   - if the abscess has spread to the superficial soft tissues
   - when the patient is febrile.

Standard antibiotics include:

- phenoxymethylpenicillin (penicillin V) or short-course, high-dose amoxicillin
- in penicillin-hypersensitive patients: erythromycin or metronidazole (as most infections are due to strict anaerobes).

Ludwig’s angina

Ludwig’s angina is a spreading, bilateral infection of the sublingual and submandibular spaces.

Aetiology

In the vast majority of cases (about 90%), Ludwig’s angina is precipitated by dental or post-extraction infection;
uncommon sources of infection include submandibular sialadenitis, infected mandibular fracture, oral soft-tissue laceration and puncture wounds of the floor of the mouth. The infection is essentially a cellulitis of the fascial spaces rather than true abscess formation.

**Clinical features**

The infection of sublingual and submandibular spaces raises the floor of the mouth and tongue and causes the tissues at the front of the neck to swell. The brawny swelling has a characteristic board-like consistency, which can barely be indented by the finger. There is severe systemic upset with fever. Complications include:

- airway obstruction due to either oedema of the glottis or a swollen tongue blocking the nasopharynx
- spread of infection to the masticator and pharyngeal spaces
- death due to asphyxiation is a certainty without immediate intervention.

Surgical drainage may yield little pus.

**Microbiology**

Oral commensal bacteria are common agents, especially *Porphyromonas* and *Prevotella* spp., fusobacteria and anaerobic streptococci; it is a mixed endogenous infection. Because of the severity of the condition, samples for microbiology assessment should always be obtained, if possible.

**Management**

1. Ensure that the patient’s airway remains open (surgically, if necessary).
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though the gingival sulcus and/or other periodontal sites (and not arising from the tooth pulp).

Aetiology

The abscess probably forms by occlusion or trauma to the orifice of a periodontal pocket, resulting in the extension of infection from the pocket into the supporting tissues. These events might result from impaction of food such as a fish bone, or of a detached toothbrush bristle, or compression

Table 34.1 Sites of contiguous spread of dentoalveolar infection
(see also Fig. 34.3)

<table>
<thead>
<tr>
<th>Site of spread</th>
<th>Maxillary teeth</th>
<th>Mandibular teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palate</td>
<td>Palatal roots of premolars and molars; also lateral incisors with a palatally curved root</td>
<td>—</td>
</tr>
<tr>
<td>Buccal space</td>
<td>Canines, premolars and molars</td>
<td>Canines, premolars and molars</td>
</tr>
<tr>
<td>Infraorbital/periorbital region</td>
<td>Canines mainly</td>
<td>—</td>
</tr>
<tr>
<td>Maxillary sinus</td>
<td>Canines, premolars and molars</td>
<td>—</td>
</tr>
<tr>
<td>Upper lip</td>
<td>Central and lateral incisors</td>
<td>—</td>
</tr>
<tr>
<td>Masseteric space, pterygomandibular space, lateral pharyngeal space</td>
<td>—</td>
<td>Lower third molars</td>
</tr>
<tr>
<td>Lower lip</td>
<td>Incisors and canines</td>
<td>—</td>
</tr>
<tr>
<td>Submandibular space</td>
<td>Root apices below insertion of mylohyoid – usually molars but can also be premolars</td>
<td>—</td>
</tr>
<tr>
<td>Submental space</td>
<td>Incisors and canines</td>
<td>—</td>
</tr>
<tr>
<td>Sublingual space</td>
<td>Root apices above mylohyoid/geniohyoid – usually incisors, canines and premolars; rarely molars</td>
<td>—</td>
</tr>
</tbody>
</table>

Table 34.2 Bacteria commonly isolated from dentoalveolar abscesses

<table>
<thead>
<tr>
<th>Facultative anaerobes</th>
<th>Streptococcus milleri</th>
<th>Streptococcus sanguinis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actinomyces spp.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obligate anaerobes</td>
<td>Peptostreptococcus spp.</td>
<td>Porphyromonas gingivalis</td>
</tr>
<tr>
<td>Prevotella intermedia</td>
<td></td>
<td>Prevotella melaninogenica</td>
</tr>
<tr>
<td>Prevotella melaninogenica</td>
<td></td>
<td>Fusobacterium nucleatum</td>
</tr>
</tbody>
</table>

Periodontal abscess

A periodontal abscess is caused by an acute or chronic destructive process in the periodontium, resulting in localized collection of pus communicating with the oral cavity.

3. Institute high-dose, empirical antibiotic therapy (usually intravenous penicillin, with or without metronidazole) immediately.
4. Collect a sample of pus before antibiotic therapy, if the patient’s condition permits, or immediately afterwards.
5. Change the prescribed antibiotic if necessary, once the bacteriological results are available.
6. Institute surgical drainage as soon as possible.
7. Eliminate the primary source of infection (e.g. a non-vital tooth).

Fig. 34.3 Pathways by which pus may spread from an acute dentoalveolar abscess (coronal section, at first molar tooth level).

Fig. 34.4 Extension of periapical infection from the left upper canine tooth to the infraorbital region in a teenager.
5. If pyrexia or cellulitis is present, antibiotics should be prescribed: penicillin, erythromycin and metronidazole are the drugs of choice.

**Suppurative osteomyelitis of the jaws**

Suppurative osteomyelitis is a relatively rare condition that may present as an acute or chronic infection, depending on a variety of factors.

**Definition**

An inflammation of the medullary cavity of the mandible or the maxilla, with possible extension of infection into the cortical bone and the periosteum as a sequela.

**Aetiology**

Osteomyelitis of the head and neck region is much rarer than dentoalveolar infections, probably because of the good vascular supply to the bone. Conditions that tend to reduce the vascularity of bone predispose to osteomyelitis, e.g. radiation, osteoporosis, Paget’s disease, fibrous dysplasia and bone tumours. A wide range of organisms have been associated with osteomyelitis of the jaws, including endogenous bacteria (described below) and, rarely, exogenous organisms such as *Treponema pallidum* and *Mycobacterium tuberculosis*.

1. The source of infection is usually a contiguous focus, or haematogenous seeding of bacteria may occur infrequently.
2. Bacteria multiply in bony medulla and elicit an acute inflammatory reaction.
3. This results in increased intramedullary pressure leading to venous stasis, ischaemia and pus formation.
4. Pus spreads through the haversian canal system, breaching the periosteum to drain through a sinus that opens on to the attached gingiva.
5. Because of intermittent drainage of pus, infection tends to remain localized, and extraoral swelling is uncommon.
6. Untreated abscesses may lead to severe destruction of periodontal tissues and tooth loss.

**Clinical features**

**Acute osteomyelitis**

Clinical features include pain, mild fever, paraesthesia or anaesthesia of the related skin; loosening of teeth; and exudation of pus from gingival margins or through sinuses or fistulae in the affected skin.

**Chronic osteomyelitis**

In chronic osteomyelitis, there is minimal systemic upset, chronic sinuses with little pus, and tender and indurated skin.

**Microbiology**

As the majority of osteomyelitis cases begin as a dentoalveolar infection, the causative organisms of both diseases are...
similar. **Anaerobes** are the most common isolates, e.g. *Tannella*, *Prevotella* and *Porphyromonas* spp., fusobacteria and anaerobic streptococci; rarely **enterobacteria** may be present. *Staphylococcus aureus*, the most common agent of osteomyelitis in long bones, is infrequently isolated from jaw lesions.

### Treatment
The management of osteomyelitis is complex. The main principles are:
1. rapid diagnosis of the disease
2. empirical prescription of antibiotics (to prevent further bone destruction and surgical intervention)
3. collection of a pus sample, if feasible, for investigations: collect pus with care when it is exuding from the gingival sulcus, to prevent contamination with commensal bacteria; aspirate pus from contiguous soft-tissue lesions
4. send the sample immediately to the laboratory in anaerobic transport medium for identification and sensitivity testing of causative bacteria
5. drugs of choice are penicillin, penicillinase-resistant penicillins (e.g. flucloxacillin) and, in penicillin-allergic patients, clindamycin and erythromycin
6. other treatment options include tooth extraction, sequestrectomy, and resection and reconstruction of the jaws.

### Cervicofacial actinomycosis
Actinomycosis (see Chapter 13) is an endogenous, granulomatous disease that may occur in the following sites:
- cervicofacial region – most common (60–65%)
- abdomen (10–20%)
- lung
- skin.

### Aetiology
In humans, the main infecting organism is *Actinomyces israelii*, which is a common oral commensal present in plaque, carious dentine and calculus. Trauma to the jaws, tooth extraction and teeth with gangrenous pulps may precipitate infection (e.g. calculus or plaque becoming impacted in the depths of a tooth socket at the time of extraction).

### Clinical features
Predominantly a disease of younger people, although all ages may be affected, the infection can present in an acute, subacute or chronic form. There is usually a history of trauma, such as a tooth extraction or a blow to the jaw. Most infections start as an acute swelling indistinguishable on clinical grounds from a dentoalveolar abscess. The chronic form of the disease follows, due to either inadequate or no therapy, or subacute infection related to trauma.

Swelling is common and is either localized or diffuse; if untreated, it may progress into discharging sinuses. Classically, this discharge of pus contains visible granules, which may be gritty to touch, yellow and known as ‘**sulphur granules**’ (a descriptive term, as sulphur is not found in the granules). These granules in pus are almost pathognomonic of the disease.

The submandibular region is most commonly affected; rarely the maxillary antrum, salivary glands and tongue may be involved. Pain is a variable feature. Other features, depending on the site of infection, are multiple discharging sinuses, trismus, pyrexia, fibrosis around the swelling, and the presence of infected teeth.

### Microbiology
The most common agent is *Actinomyces israelii*, although *Actinomyces bovis* and *Actinomyces naeslundii* may occasionally be isolated. In a minority, *Aggregatibacter actinomycetemcomitans* may be isolated in mixed culture with *Actinomyces israelii*.

### Laboratory diagnosis
If a fluctuant abscess is present, collect fluid pus by aspiration using a syringe, or in a sterile container if drainage by external incision is performed. Examine the pus for the presence of ‘sulphur granules’; Gram films are made from any part with a lumpy or granular appearance. The granules are washed and crushed in tissue grinders and cultured on blood agar under anaerobic conditions at 37°C for 7 days. Colonies often produce a typical ‘molar tooth’ morphology (see Fig. 13.1). Pure cultures are then identified using biochemical techniques. A Gram film of a colony will reveal moderate to large clumps of Gram-positive branching filaments.

### Management
#### Acute lesions
1. Removal of any associated dental focus.
2. Incision and drainage of facial abscess.
3. A 2- to 3-week course of antibiotics; penicillin is the drug of choice.

#### Subacute or chronic lesions
1. Surgical intervention, as in (1) and (2) above.
2. A longer antibiotic course, 5–6 weeks on average.

If penicillin cannot be given because of hypersensitivity, erythromycin, tetracycline and clindamycin are good alternatives. The latter drugs penetrate bony tissues well.
KEY FACTS

- Dental caries is the main cause of pulpal and periapical infection; other routes include periodontal pocket and, rarely, anachoresis (i.e. haematogenous seeding).
- **Dentoalveolar infections** are usually polymicrobial in nature and endogenous in origin, with a predominance of strict anaerobes.
- Ideally, an aspirated sample of pus should be collected for microbiological examination of a dentoalveolar abscess in the head and neck region.
- **Drainage of pus** is the mainstay of treatment of dentoalveolar and periodontal abscesses; elimination of the infective focus and antibiotic therapy should be considered on an individual basis.
- **Ludwig’s angina** is a spreading, bilateral infection of the sublingual and submandibular spaces; it is a life-threatening infection.
- Periodontal abscess: an acute or chronic destructive process in the periodontium, resulting in localized collection of pus communicating with the oral cavity through the gingival sulcus and/or other periodontal sites (and not arising from the tooth pulp).
- Periodontal abscess is an endogenous, polymicrobial infection with a predominantly anaerobic, periodontopathic flora.
- **Suppurative osteomyelitis of the jaws is uncommon**; it is mostly seen in immunocompromised patients. Usually a polymicrobial infection, it requires both medical and surgical intervention.
- **Cervicofacial actinomycosis:** an endogenous granulomatous disease, usually presenting at the angle of the mandible and related to trauma or a history of tooth extraction, mainly caused by *Actinomyces israelii*; ‘sulphur granules’ may be present in pus.
- Actinomycoses are managed by surgical drainage and long-term antibiotics, preferably penicillin.

Further reading


REVIEW QUESTIONS (answers on p. 355)

Please indicate which answers are true, and which are false.

34.1 Which of the following statements on dentoalveolar abscess are true?

A it is often precipitated by bacteria from the systemic route (anachoresis)
B it has a polymicrobial aetiology
C it is frequently implicated as a cause of brain abscess
D it often resolves without antibiotics after adequate drainage
E it is a localized collection of pus with an epithelial lining

34.2 Which of the following statements on Ludwig’s angina are true?

A the majority of cases are due to submandibular sialadenitis
B it may warrant an urgent tracheostomy
C often the patient is toxic
D it needs to be treated with high-dose, parenteral metronidazole and penicillin
E a copious amount of pus is yielded on surgical drainage

34.3 Microorganisms that are frequently implicated in the pathogenesis of periodontal abscess include:

A *Treponema pallidum*
B *Porphyromonas* spp.
C fusobacteria
D staphylococci
E *Actinomyces israelii*

34.4 Which of the following statements on actinomycosis are true?

A abdominal lesions are more prevalent than cervicofacial lesions
B *Aggregatibacter actinomycetemcomitans* is an associated co-pathogen
C lesions contain sulphur
D it is caused by a slow-growing filamentous Gram-positive organism
E a 1-week course of penicillin is adequate