**Infections of the jaws**

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Severe infections of bone are uncommon despite the numerous pathogenic bacteria in the mouth and the easy access to the medullary cavity through tooth roots and extraction sockets. Indeed, the bone of the jaws appears remarkably resistant to osteomyelitis.

**ALVEOLAR OSTEITIS**

Alveolar osteitis (‘dry’ socket) is by far the most frequent painful complication of extractions. It is not really an infection but leads to superficial bacterial contamination of exposed bone and can progress to osteomyelitis, though extremely uncommonly. Osteitis simply means inflamed bone, not infection. Alveolar osteitis develops after 1%–2% of extractions, more frequently for lower-third molar extractions.

**Predisposing factors for alveolar osteitis**

Alveolar osteitis is frequently unpredictable and without any obvious predisposing cause, but numerous possible aetiological factors exist:

• Excessive extraction trauma

• Limited local blood supply

• Gingival infection such as acute ulcerative gingivitis, pericoronitis or abscess

• Local anaesthesia with vasoconstrictor

• Smoking

• Oral contraceptives

• Osteosclerotic disease: Paget’s disease, cementoosseous dysplasia

• Radiotherapy

• History of previous dry socket.

***Clinical features***

* Patients aged 20–40 years are most at risk, and women are more frequently affected.
* Pain usually starts a few days after the extraction, but sometimes may be delayed for a week or more. It is deep-seated, severe and aching or throbbing.
* The mucosa around the socket is red and tender. There is no clot in the socket, which contains, instead, saliva and often decomposing food debris.
* When debris is washed away, whitish, dead bone may be seen or may be felt as a rough area with a probe and probing is painful.
* The appearance of an empty socket and exposed bone is diagnostic.

***Pathology***

Infected food and other debris accumulate in direct contact with the bone. Bone damaged during the extraction. The necrotic bone and socket lodge bacteria which proliferate freely in the avascular spaces unhindered by host defences. Dead bone is gradually separated by osteoclasts, and sequestra are usually shed in tiny fragments. Healing is slow. Granulation tissue cannot grow in from the socket walls and base until the necrotic bone is removed.

Although there is no infection within the tissues, the colonization of the socket and sequestra by oral bacteria probably contributes to pain and slow healing. Anaerobes are thought to be significant and can produce fibrinolytic enzymes. However, antibiotics including metronidazole have not been shown to either prevent dry socket or speed healing reliably. Only chlorhexidine rinsing preoperatively has been shown to reduce incidence.

**Prevention of dry socket**

• Preoperative infection control

• Scaling teeth before extraction

• Chlorhexidine rinsing preoperatively and for 3 days postoperatively

• Atraumatic extraction

• Adherence to postoperative instructions

• No rinsing or forceful spitting

• No hot fluids

• No smoking

• Postoperative antibiotics only for those at particular risk

***Treatment***

* The socket should be irrigated with mild warm antiseptic or saline to remove all food debris.
* Place a dressing into the socket to deliver analgesia and close the opening.
* Minimum dressing to close the socket opening is used because dressing packed hard into the socket will delay healing.
* A dressing may only last 1–2 days, and the whole process needs repeating until pain subsides, normally after one or two dressings Frequent hot saline mouthwashes also help keep the socket free from debris.

**OSTEOMYELITIS OF THE JAWS**

Unlike the long bones, osteomyelitis in the jaws is almost always of local origin and not caused by blood-borne infection.

**ACUTE OSTEOMYELITIS**

In acute osteomyelitis bacteria and inflammation spread through the medullary bone from a focus of infection.

**Acute osteomyelitis of the jaws: potential sources of infection**

• Periapical infection

• Pericoronitis

• Fracture through periodontal pocket or open to the mouth

• Penetrating, contaminated injuries (open fractures or gunshot wounds).

**Important predisposing conditions for osteomyelitis**

Local damage to or disease of the jaws

• Radiation damage

• Causes of osteosclerosis

• Paget’s disease

• Fibro-osseous lesions, particularly cemento-osseous dysplasia

• Osteopetrosis

Impaired immune defences

* Poorly controlled diabetes mellitus
	+ Chronic alcoholism or malnutrition
	+ Drug abuse
	+ Tobacco smoking.
	+ Malignant neoplasms and their treatment

**Microbiology**

* Bacteroides spp.
* Staph.aureus
* Klebsseilla.
* Proteus.

***Clinical features***

* Most patients with osteomyelitis are adult almost all cases affect the mandible, which is less vascular than the maxilla.
* Early complaints are severe, throbbing, deep-seated painand swelling with external swelling due to inflammatory oedema.
* Distension of the periosteum with pus and, finally, subperiosteal bone formation cause the swelling to become firm.
* The overlying gingiva and mucosa is red, swollen and tender. Associated teeth are tender.
* Muscle oedema causes difficulty in opening the mouth and swallowing. Regional lymph nodes are enlarged and tender.
* Anesthesia or paraesthesia of the lower lip, caused by pressure on the inferior dental nerve, is characteristic.
* Radiographic changes do not appear until after at least 10 days, Later, there is loss of trabecular pattern and areas of radiolucency indicating bone destruction and . Affected areas have ill-defined margins and a moth-eaten appearance similar to a malignant neoplasm Later, in young persons particularly, subperiosteal new bone formation causes a buccal swelling and appears as a thin, curved strip of new bone below the lower border of the jaw in lateral or panoramic radiographs.

**Management of acute osteomyelitis**

Essential measures

• Bacterial sampling and culture specimen of pus or a swab from the depths of the lesion must first be taken for culture and sensitivity testing,

• Vigorous (empirical) antibiotic treatment

• Drainage

• Analgesics

• Give specific antibiotics once culture and sensitivities are available

• Debridement

• Remove source of infection, if possible

Adjunctive treatment

• Sequestrectomy

• Decortication if necessary

• Resection and reconstruction for extensive bone destruction

**CHRONIC OSTEOMYELITIS**

Chronic osteomyelitis is much more common than acute osteomyelitis and arises from infection by weakly virulent bacteria or in avascular bone. Most cases develop without a prior acute phase, and only rarely does acute osteomyelitis lead to chronic osteomyelitis. When it does, it usually follows inadequate treatment.

***Clinical features***

* Low-grade pain often relapsing, during a long period with a bad taste from pus draining to the mouth through sinuses.
* There may be exposed bone. Initially the original focus of infection can be identified, but chronic osteomyelitis may persist after its removal and the chronic infection becomes self-perpetuating in the bone.
* Radiographic appearances are variable but sometimes distinctive with patchy and poorly defined radiolucency and sclerosis
* Sequestra may be identified, and there may be a periosteal new bone layer seen.

***Pathology***

Persistent low-grade infection is associated with chronic inflammation, activation of osteoclastic bone destruction and granulation tissue formation.

. Healing is frustrated by inability of the inflammation and immune response to access bacteria in dead avascular bone and by the slow separation of dead bone as sequestra.

**Treatment**

* The source of infection must be removed.
* Prolonged antibiotic treatment is the mainstay of treatment and must continue for at least 6 weeks
* Role for surgery to remove sequestra and sclerotic bone.

**OSTEORADIONECROSIS**

When the predisposing cause for any type of osteomyelitis is radiotherapy, the condition is called *osteoradionecrosis*.

**Pathology**

Radiotherapy induces endarteritis of vessels causing a marked reduction in bone vascularity, inhibiting an effective host response to infection and reducing the sclerotic response to infection. The risk of osteoradionecrosis rises with the radiation dose. The causative bacteria are oral flora and periodontal pathogens, which gain entry to the bone after minor trauma, dental infection or tooth extraction. The mucosa

is atrophic and heals poorly after radiotherapy. Infection spreads rapidly and is difficult to treat. The clinical and radiographic features are those of chronic osteomyelitis except that healing is impaired, sequestra separate much more slowly and there is no periosteal reaction.

**Treatment**

Surgical intervention and aggressive antibiotic therapy are usually required and hyperbaric oxygen are claimed to aid healing, but results are variable and the latter is very expensive and not widely available. Unfortunately treatment is not always successful, and low grade grumbling osteomyelitis may persist for the rest of a patient’s life. Prevention is key, and the dentist plays an important role**.**

**Prevention of osteoradionecrosis**

• Before radiotherapy all patients should have a dental examination

• Institute aggressive preventive regime of diet change and fluoride

• All potential foci of infection must be aggressively treated, usually by extraction

• Sockets must be epithelialised before radiotherapy starts

• Other treatment should be completed in a low risk ‘window’ of 10 weeks after radiotherapy

• Dentures and postoperative obturators must not traumatise mucosa

• Close monitoring for dental infection and to prevent trauma continues for life

• Extractions in irradiated bone must be atraumatic

• Antibiotics are required after any oral surgical procedure until healing is complete