Inflammatory diseases of bone

Acute alveolar osteitis (dry socket) د.صباح الهيتي

Alveolar osteitis, also known as dry socket, is a severely painful complication arising between one and three days post extraction. It is very common. The incidence of dry socket ranges from 0.5-5% for all routine extractions, but can reach up to 38% for extractions of impacted mandibular third molars.

localised fibrinolysis (resulting from conversion of plasminogen to plasmin, which acts to dissolve fibrin crosslinks) occurring within the socket and subsequently leading to loss of the blood clot is believed to underlie the pathogenesis of alveolar osteitis

predisposing factors: Smoking, premature mouth rinsing, hot liquids, surgical trauma, and oral contraceptives

clinical features: the patient usually presents within 2-4 days of the extraction complaining of

1- Persistent dull pain which is well localized to the socket.
2- The socket is either devoid of clot or contain friable disintegrated clot which is easily washed out.
3- Foul taste and odor
4- Gingival margin of socket is swollen and dusky red
5- Lymphadenopathy

The critical time for development of dry socket is during the first four days after extraction because at about the 3rd day granulation tissue starts to invade the clot. from this time therefore loss of the clot will no longer expose bare bone.

Preventive measures :

1- Preoperatively, the use of a 0.12% chlorhexidine rinse prior to the extraction
2- The use of both systemic and topical antibiotics has been shown to reduce the incidence of dry socket. Systemic penicillins, clindamycin and metronidazole, and topical tetracycline powder have all been shown to be effective

Treatment : Conservative management is indicated.

- ❖ The wound should be irrigated gently with slightly warmed saline. Local anesthesia may occasionally be required for this
- ❖ a sedative dressing should be placed like alvogyl. The dressing should be removed within 48 hr and replaced until the patient becomes asymptomatic.
- ❖ Systemic antibiotics are generally not indicated as they have no additional advantage over local treatments directed to the socket in a non-immune-compromised patient
- ❖ Non-steroidal anti-inflammatory analgesics should be prescribed if necessary

Why curette a dry socket is contraindicated?

— Curetting a dry socket can cause the condition to worsen because healing will be further delayed, any natural healing already taking place will be destroyed,

and there is a risk of causing the localized inflammatory process to be spread to the adjacent sound bone

Osteomyelitis

Osteomyelitis is defined as an inflammation of the bone marrow *with a tendency to progression.* This is what differentiates it in the jaw from the ubiquitous dentoalveolar abscess, "dry socket" and "osteitis," seen in infected fractures. It involves adjacent cortical plates and often periosteal tissues.

Pathogenesis

In the maxillofacial region, osteomyelitis primarily occurs as a result of contiguous spread of odontogenic infections or as a result of trauma. There are several potential sources of infection:

i. A Periapical infection
ii. A periodontal pocket involved in a fracture
iii. Acute necrotising gingivitis or pericoronitis (even more rarely)
iv. Penetrating, contaminated injuries (open fractures or gunshot wounds)

This initial insult results in a bacteria-induced inflammatory process.. Pus is formed when there is an overwhelming supply of bacteria and cellular debris that cannot be eliminated by the body's natural defense mechanisms. When the pus and subsequent inflammatory response occur in the bone marrow, an elevated intramedullary pressure is created which further decreases the blood supply to this region. The pus can travel via haversian and Volkmann's canals to spread throughout the medullary and cortical bones. Once the pus has perforated the cortical bone and collects under the periosteum, the periosteal blood supply is compromised and this further aggravates the local condition. The end point occurs when the pus exits the soft tissues either by intraoral or extraoral fistulas

Microbiology

As with most oral infections the prime pathogenic species are streptococci and anaerobic bacteria (bacteroides gram –ve or peptostreptococci gram +ve)

Classification

The classification system offered by Hudson is the most advantageous to the clinician. Osteomyelitis is divided into acute or chronic forms based on the presence of the disease for 1-month duration.

1. Acute osteomyelitis

a. Contiguous focus
b. Progressive
c. Hematogenous

2. Chronic osteomyelitis

a. Recurrent multifocal
b. Garré's
c. Suppurative or nonsuppurative
d. Sclerosing

Clinical Presentation

The patient with osteomyelitis of the maxillofacial region will present with classic symptoms:

- Pain (deep and boring pain, which is often out of proportion to the clinical picture)
- Swelling and erythema of overlying tissues
- Adenopathy
- Fever
- Paresthesia of the inferior alveolar nerve
- Trismus
- Malaise
- Fistulas

In the acute phase of osteomyelitis it is common to see a leukocytosis with left shift, common in any acute infection. Leukocytosis is relatively uncommon in the chronic phases of osteomyelitis. The patient may also exhibit an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).
Radiograph appear normal till 1-3 weeks. Gradual resorption around the periphery of the infarcted area of bone separates it off as sequestrum and the granulation tissue between the living and dead bone produce irregular lines and zones of R.L. one can often see the appearance of "moth-eaten which is the classic appearance of osteomyelitis (figure 1)

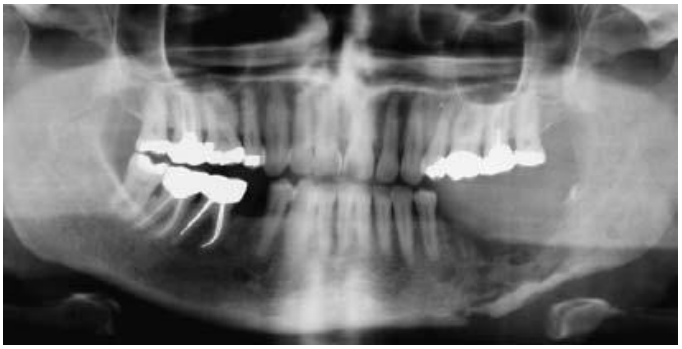


Figure 1: Acute osteomyelitis. ***The classic appearance of moth-eaten bone***

Computerized tomography (CT) scans have become the standard in evaluating maxillofacial pathology such as osteomyelitis. Its sensitive in late stage when 30%-50% of bone demineralized.
Magnetic resonance imaging (MRI) is generally considered more valuable in the evaluation of soft tissue lesions of the maxillofacial region. However, MRI can assist in the early diagnosis of osteomyelitis by loss of the marrow signal before cortical erosion or sequestrum of the bone appears. Thus, MRI may benefit in identifying the earlier stages of osteomyelitis.
Nuclear medicine has evolved to aid in the diagnosis of osteomyelitis. The technetium 99 bone scan is very sensitive in highlighting areas of increased bone turnover.

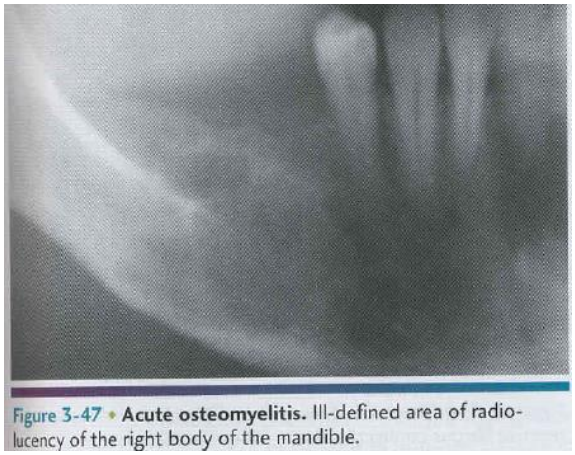


Figure 3-47 • **Acute osteomyelitis.** Ill-defined area of radiolucency of the right body of the mandible.

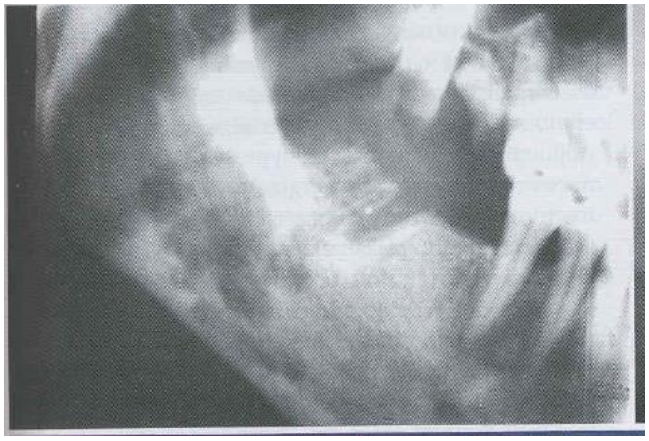


Figure 2: (A) Acute OM. **(B) Chronic OM**

Treatment

The management of osteomyelitis of the maxillofacial region requires both medical and surgical interventions. In rare cases of infantile osteomyelitis, intravenous antibiotic therapy alone may eradicate the disease. Antibiotic therapy is rarely curative in later-onset cases, and the overwhelming majority of osteomyelitis cases require surgical intervention.

Bacteriological diagnosis. A specimen of pus or a swab from the depths of the lesion must first be taken for culture and sensitivity testing.

Antimicrobial treatment. Immediately a specimen has been obtained, vigorous antibiotic treatment should be started. Initially, penicillin, 600-1200 mg daily can be given by injection (if the patient is not allergic), with metronidazole 200-400 mg 8-hourly. In acute OM, Period of AB is 1- 3 months (if immune compromised) while in Chronic OM should be continue for 3- 6 months

Debridement. Removal of foreign or necrotic material and immobilisation of any fracture are necessary if there has been a gunshot wound or other contaminating injury. Classic surgical treatment is sequestrectomy , saucerization and Decortication . The aim is to débride the necrotic or poorly vascularized bony sequestra in the infected area and improve blood flow.

Sequestrectomy involves removing infected and avascular pieces of bone—generally the cortical plates in the infected area.

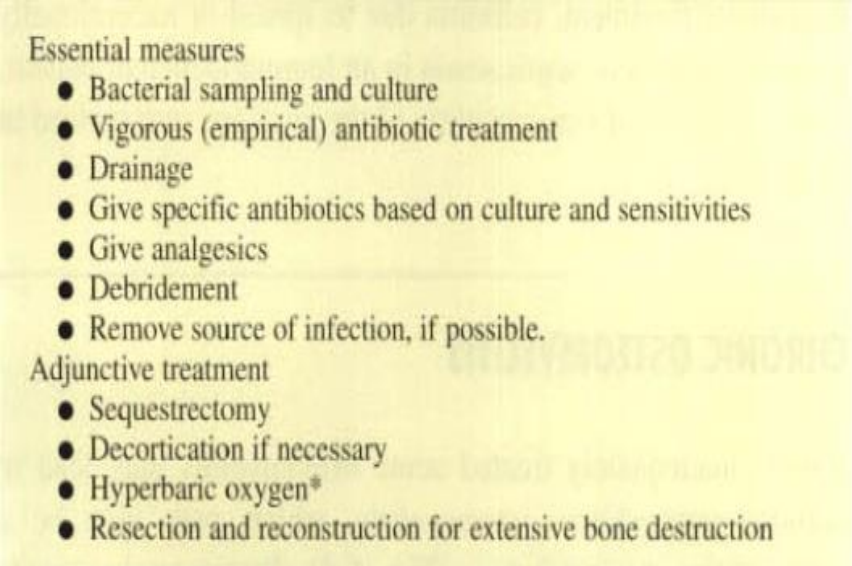
Saucerization involves the removal of the adjacent bony cortices and open packing to permit healing by secondary intention after the infected bone has been removed. The aim is to create a uniform bed which help in complete drainage and improve blood supply.

Decortication involves removal of the dense, often chronically infected and poorly vascularized bony cortex and placement of the vascular periosteum adjacent to the medullary bone to allow increased blood flow and healing in the affected area.

Drainage. Pressure should be relieved by tooth extraction, bur holes or decortication, as necessary, and exudate drained into the mouth or externally

Removal of sequestra. Dead bone should not be forcibly separated and vigorous curetting is inadvisable, but in the late stages a loosened sequestrum may have to be removed. Teeth should be extracted only if loosened by tissue destruction.

Adjunctive treatment. Decortication or hyperbaric oxygen therapy, or both, may be required, particularly in radiation-associated osteomyelitis.


Essential measures
- Bacterial sampling and culture
- Vigorous (empirical) antibiotic treatment
- Drainage
- Give specific antibiotics based on culture and sensitivities
- Give analgesics
- Debridement
- Remove source of infection, if possible.

Adjunctive treatment
- Sequestrectomy
- Decortication if necessary
- Hyperbaric oxygen*
- Resection and reconstruction for extensive bone destruction

Complications of acute OM

- Anaesthesia of the lower lip usually recovers with elimination of the infection
- pathological fracture
- chronic osteomyelitis after inadequate treatment

Differences between acute and chronic osteomyelitis

	Acute OM	**Chronic OM**
Pain	Common	Uncommon
History of present illness	Days (less than month)	more than 1- month
Systemic S.&S.	common	uncommon
Fistula or sinus	Not present	Present
Lip sensation	lost	preserved
Radiograph	moth eaten	ill defined R.L. contain central R.O. sequestrum
ESR, C-reactive protein	Elevated	Normal
Duration of AB.	1-3 months	3-6 months

Chronic osteomyelitis

May also be classified into suppurative (pus- production) and non-suppurative osteomyelitis. The latter include:

A- focal sclerosing osteomyelitis

B- Chronic diffuse sclerosing osteomyelitis

C- proliferative periostitis (Garre's OM)

Focal sclerosing OM: (figure 4)

- localized area of bony sclerosis associated with apices of teeth with pulpitis (from large carious lesion or deep coronal restoration) or pulp necrosis. It represent a bony reaction to low-grade periapical infection or unusually strong host defensive response
- Children and young adults affected
- Premolar or molar region of mandible affected
- Localised but uniform radiodensity related to tooth with widened periodontal ligament space or periapical area
- No expansion of the jaw (no swelling)
- Treated by elimination of the source of inflammation by extraction or endodontic treatment

Diffuse sclerosing OM: (figure 5)

Painful, non-suppurative, expansile swelling not associated with fistula or sinus.

Sclerosis round site of periapical or periodontal chronic inflammation arises almost exclusively in adulthood with mandible predilection.

Radiographically, diffuse area of sclerosis in tooth bearing area.

Treatment: Elimination of originating source of inflammation, but sclerotic areas remain radiographically

Proliferative periostitis (Garre OM) (figure 6)

- Children and young adult with mean age of 13 years mainly affected
- Usually associated with periapical but sometimes other inflammatory foci
- Periosteal reaction affecting lower border of mandible causing ‘'onion skin‘’ thickening and swelling of bone (hard non tender bony swelling of the inferior and/or lateral aspect of mandible).
- Radiographically: radiopaque laminations of bone parallel each other and the underlying cortex surface with radiolucent separations.
- *Treatment*:
    - Eliminate focus of infection by extraction oe endodontic treatment
    - Bone gradually remodels after 6 to 12 months

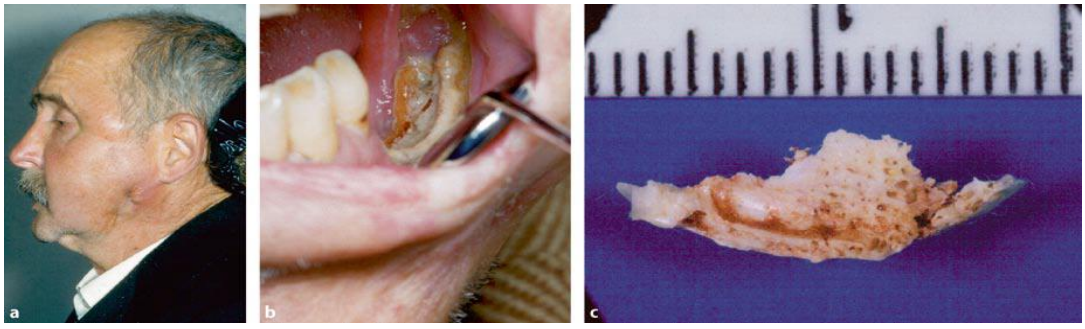


Figure 3: chronic osteomyelitis, extraoral and intraoral view.

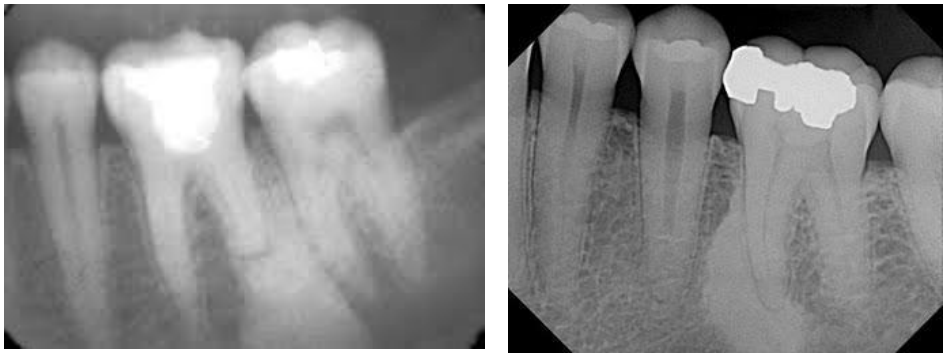


Figure 4: Focal sclerosing OM

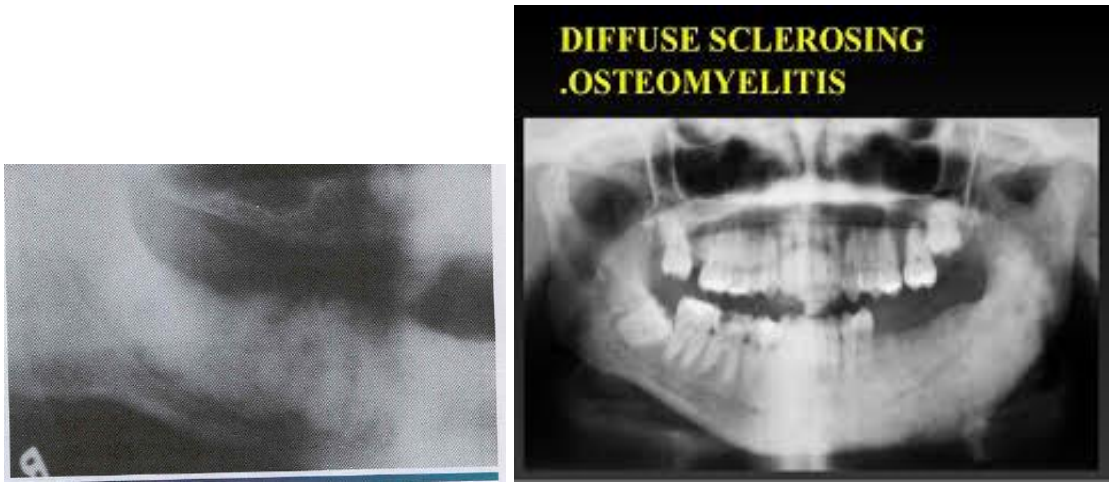


Figure 5: **Diffuse sclerosing OM**

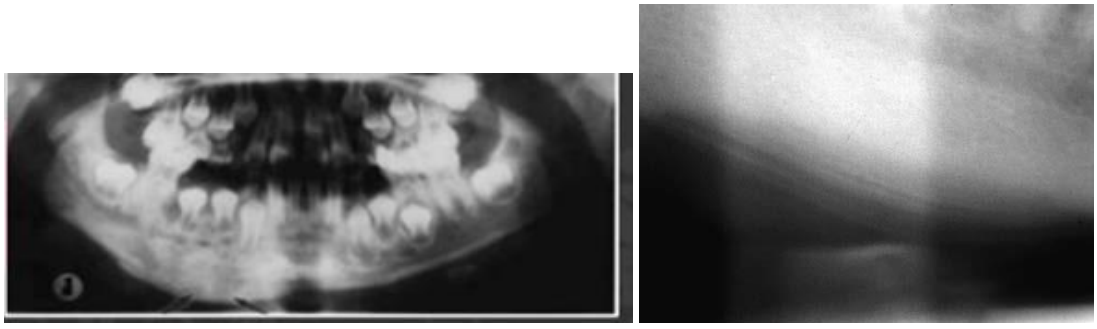


Figure 6: **Garre** OM.

Osteoradionecrosis

Radiation therapy is a valuable treatment modality in treating cancer of the maxillofacial region. Radiation therapy like any treatment modality has deleterious side effects, including mucositis and xerostomia. One of the most dreaded side effects is osteoradionecrosis (ORN). osteoradionecrosis represents a chronic nonhealing wound that is hypoxic, hypocellular, and hypovascular.

ORN is generally caused by trauma to the radiated area, usually by dental extraction, but it can also occur spontaneously. The clinical picture of ORN is most commonly seen with pain and exposed bone in the maxillofacial region (Figures 7). ORN is more common in the mandible than in the maxilla for reasons described earlier in this chapter. A dosage of radiation above 5,000 to 6,000 rads is generally felt to make the mandible susceptible to ORN. Radiographically, the appearance on the orthopantomogram or CT scan resembles conventional osteomyelitis with areas of osteolysis and bony sequestrum. Often there is an appearance of moth-eaten bone present on these films.

The treatment of ORN is aimed at removing the nonviable (necrotic) tissue and allowing the body to heal itself. Minor débridements of exposed bone may work in the most minor cases of ORN. Current therapy calls for augmentation of tissue healing response by the use of HBO. HBO therapy consists of 100% oxygen delivered in a pressurized manner. Tissues treated with HBO have increased levels of oxygen, which has a negative effect on bacteria and a positive effect on angiogenesis and increased blood flow to the area.

HBO treatment consists of dives or treatment sessions for 90 minutes based at 2.4 atm of pressure. Twenty to 30 dives are given preoperatively before any surgical intervention is performed. The area of ORN is then débrided and followed with 10 additional HBO treatments. Reconstruction of the maxillofacial region is based on the patient's response to the treatment protocol.

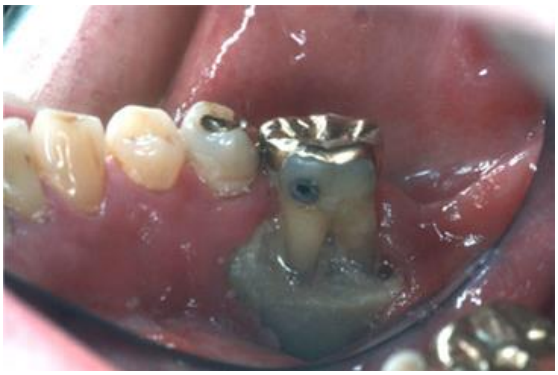


Figure 7: ORN of posterior mandible

Osteonecrosis of the jaws (ONJ):

also known as medication-related osteonecrosis of the jaws (MRONJ) is a breach in the oral mucosa leading to exposed bone that fails to heal in 6 to 8 weeks. The patients must have a history of receiving antiresorptive therapy and no previous head and neck radiation. While the occurrence rate of ONJ in oncology patients ranges from 1.5% to 15%, patients treated for benign conditions (most commonly osteoporosis) appear to have a much lower incidence of ONJ (1/10,000 to 1/100,000).

What causes ONJ?

it appears this disorder is multifactorial in nature. There seems to be a strong association between antiresorptive therapy (bisphosphonates, anti-RANK ligand) and ONJ. Development of ONJ is often preceded by a traumatic event, most commonly an extraction.

How is ONJ treated?

The management of patients with ONJ has traditionally been conservative, with the recommendation that surgical intervention only be performed in the most severe cases (Stage 3) where the patients have significant symptomatology.

Table 48-4. ONJ Staging System

STAGING	TREATMENT
At-risk category: No apparent necrotic bone in patients who have been treated with either oral or IV bisphosphonates	• No treatment indicated • Patient education
Stage 0: No clinical evidence of necrotic bone, but nonspecific clinical findings, radiographic changes, and symptoms	• Systemic management, including the use of pain medication and antibiotics
Stage 1: Exposed and necrotic bone, or fistulae that probe to bone, in patients who are asymptomatic and have no evidence of infection	• Antibacterial mouth rinse • Clinical follow-up on a quarterly basis • Patient education and review of indications for continued bisphosphonate therapy
Stage 2: Exposed and necrotic bone, or fistulae that probe to bone, associated with infection as evidenced by pain and erythema in the region of the exposed bone with or without purulent drainage	• Symptomatic treatment with oral antibiotics • Oral antibacterial mouth rinse • Pain control • Debridement to relieve soft tissue irritation and infection control
Stage 3: Exposed and necrotic bone or a fistula that probes to bone in patients with pain, infection, and one or more of the following: exposed and necrotic bone extending beyond the region of alveolar bone (i.e., inferior border and ramus in the mandible, maxillary sinus and zygoma in the maxilla) resulting in pathologic fracture, extraoral fistula, oroantral and oronasal communication, or osteolysis extending to the inferior border of the mandible or sinus floor	• Antibacterial mouth rinse • Antibiotic therapy and pain control • Surgical debridement/resection for longer term palliation of infection and pain