Pediatric surgical stomatology

Features of clinical course of inflammatory diseases in face and neck in children: Periostitis Osteomyelitis

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Content of lecture

- Terminology by inflammation
- Etiology of odontogenic infection
- Pathways of infection spreed
- Dentoalveolar abscess: clinical picture and features of treatment
- Classification of osteomyelitis. Pathogenesis. Clinical stages. Features of odontogenic osteomyelitis. Diagnostic images. Treatment.

Background

- A dentoalveolar abscess is an acute lesion characterized by localization of pus in the structures that surround the teeth. Most patients are treated easily with analgesia, antibiotics, drainage, and/or referral to a dentist or oral-maxillofacial surgeon. However, the physician should be aware of potential complications of simple dentoalveolar abscess.

Terminology

- **Dentoalveolar abscess -** acute lesion characterized by localization of pus in the structures that surround the teeth
- **Dentoalveolar abscess** is a pus-filled sac in the tissue around the root of a tooth.

- **Periostitis** is a medical condition caused by inflammation of the periosteum, a layer of connective tissue that surrounds bone.

Terminology

- The strict definition *of osteomyelitis* is an inflammation of the medullary portion of the bone. Inflammation of cortical bone is termed *osteitis*. In practice, this distinction is probably not important, as infection of the medullary cavity of the bone easily enters the Haversian systems and Volksman canals to involve the cortical bone with its periosteum.
- Bones without a medullary cavity, such as the posterior wall of the maxillary sinus and posterior table of the frontal sinus, cannot be involved with osteomyelitis, but osteitis can and does occur in these areas. The treatment of bone infection varies little regardless of whether osteitis or osteomyelitis exists, so this distinction will not be emphasized.

Terminology

- The word “osteomyelitis” originates from the ancient Greek words osteon (bone) and muelinos (marrow) and means infection of medullary portion of the bone. Common medical literature extends the definition to an inflammation process of the entire bone including the cortex and the periosteum, recognizing that the pathological process is rarely confined to the endosteum. It usually encompasses the cortical bone and periosteum as well. It can therefore be considered as an inflammatory condition of the bone, beginning in the medullar cavity and havarian systems and extending to involve the periosteum of the affected area. The infection becomes established in calcified portion of the bone when pus and edema in the medullary cavity and beneath the periosteum compromises or obstructs the local blood supply. Following ischemia, the infected bone becomes necrotic and leads to sequester formation, which is considered a classical sign of osteomyelitis.

Terminology

- Although other etiological factors, such as traumatic injuries, radiation, and certain chemical substances, among others, may also produce inflammation of the medullar space, the term "osteomyelitis" is mostly used in the medical literature to describe a **true infection of the bone induced by pyogenic microorganisms**.

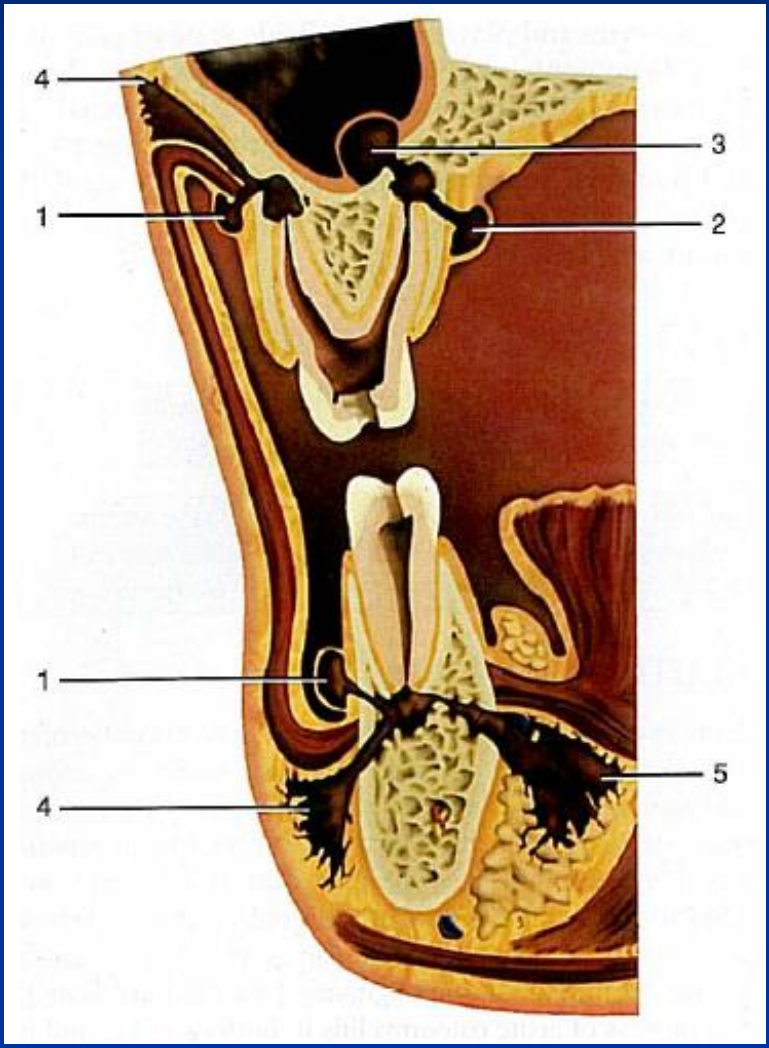
Cause

Age group	Most common organisms
Newborns (younger than 4 mo)	*S. aureus*, *Enterobacter* species, and group A and B *Streptococcus* species
Children (aged 4 mo to 4 y)	*S. aureus*, group A *Streptococcus* species, *Haemophilus influenzae*, and *Enterobacter* species
Children, adolescents (aged 4 y to adult)	*S. aureus* (80%), group A *Streptococcus* species, *H. influenzae*, and *Enterobacter* species
Adult	*S. aureus* and occasionally *Enterobacter* or *Streptococcus* species

Etiology

- Osteomyelitis is uncommon in the immunocompetent host. Considering the high incidence of local infections in the Craniomaxillofacial region (e.g., sinusitis, pharyngitis, periodontitis) and the potentially pathogenic bacteria present in the upper aerodigestive tract, it is surprising that bone infection is not seen more frequently. One explanation for this may be the excellent blood supply normally present in this region. Another may be the inherent resistance of the host.
- Osteomyelitis is seen more frequently in patients with vascular and immune compromise

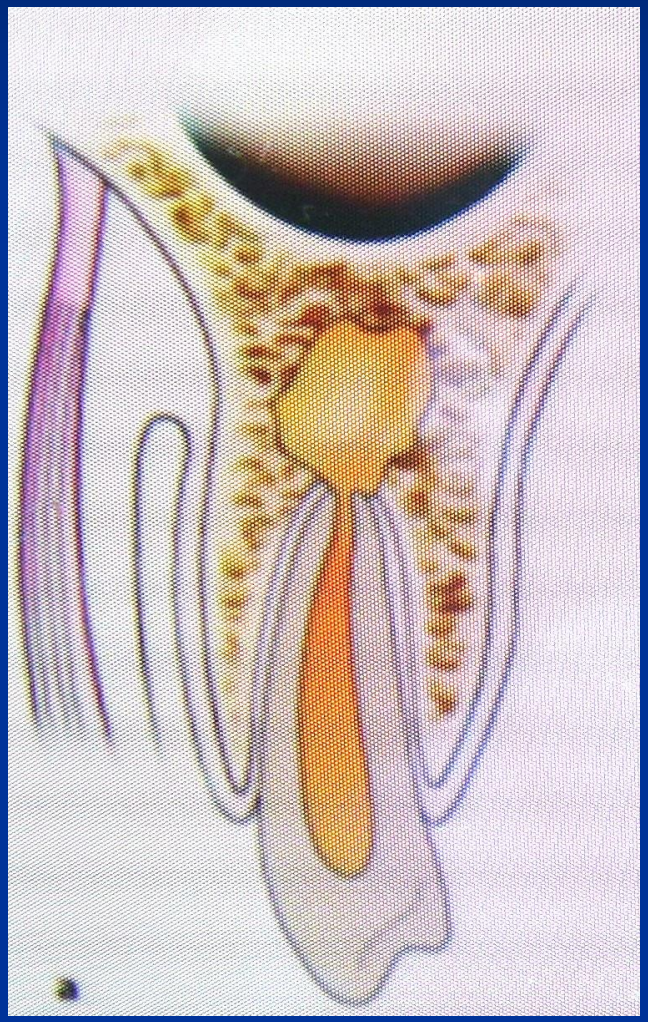
Drainage pathways of acute periapical infection

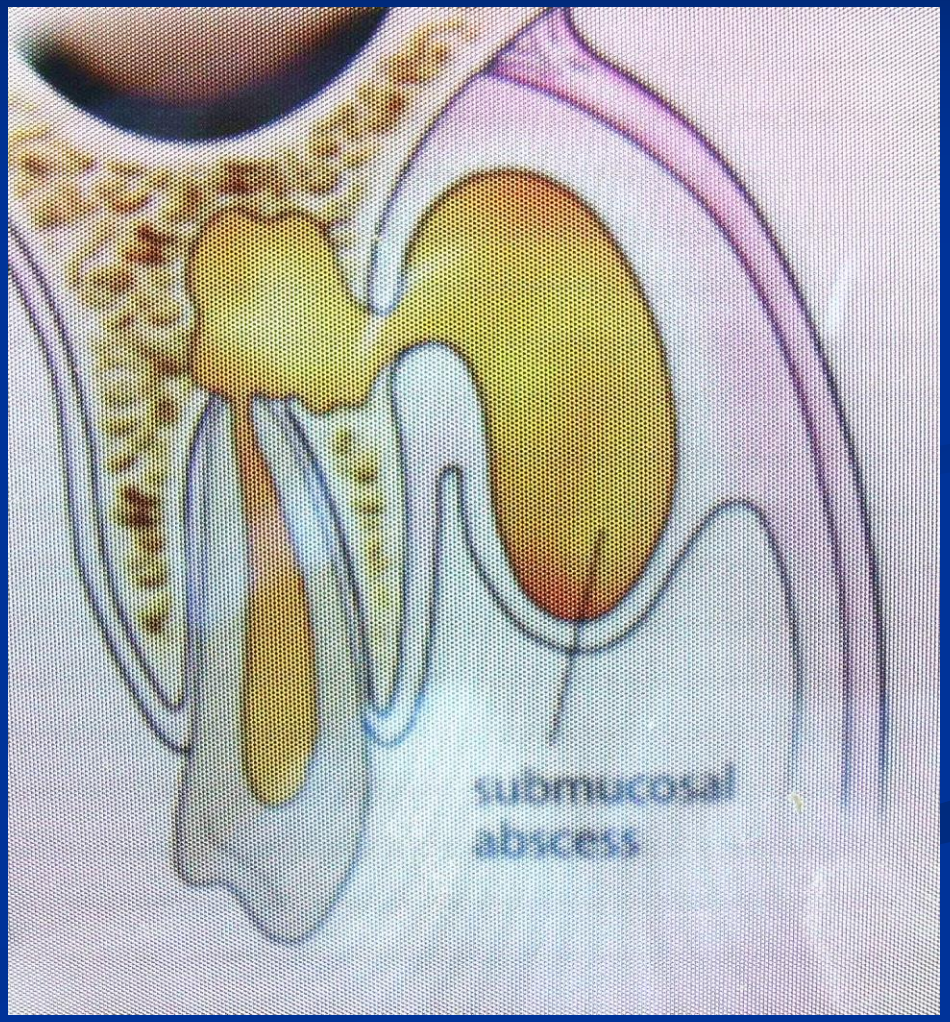


- The location of the various points of drainage is determined by the anatomic location of the root apex. Common locations are (1) surface of gingiva (parulis), (2) palate, (3) maxillary sinus, (4) soft tissue spaces superior (maxilla) and inferior (mandible) to buccinator muscle (cellulitis), and (5) floor of the mouth (Ludwig angina)

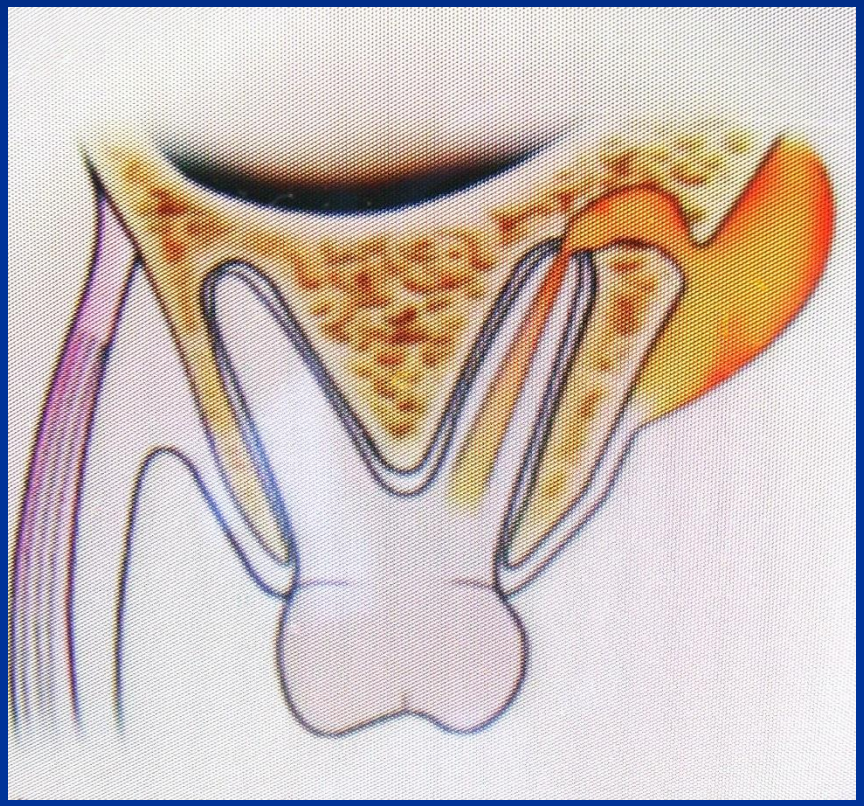
Dento-alveolar abscess

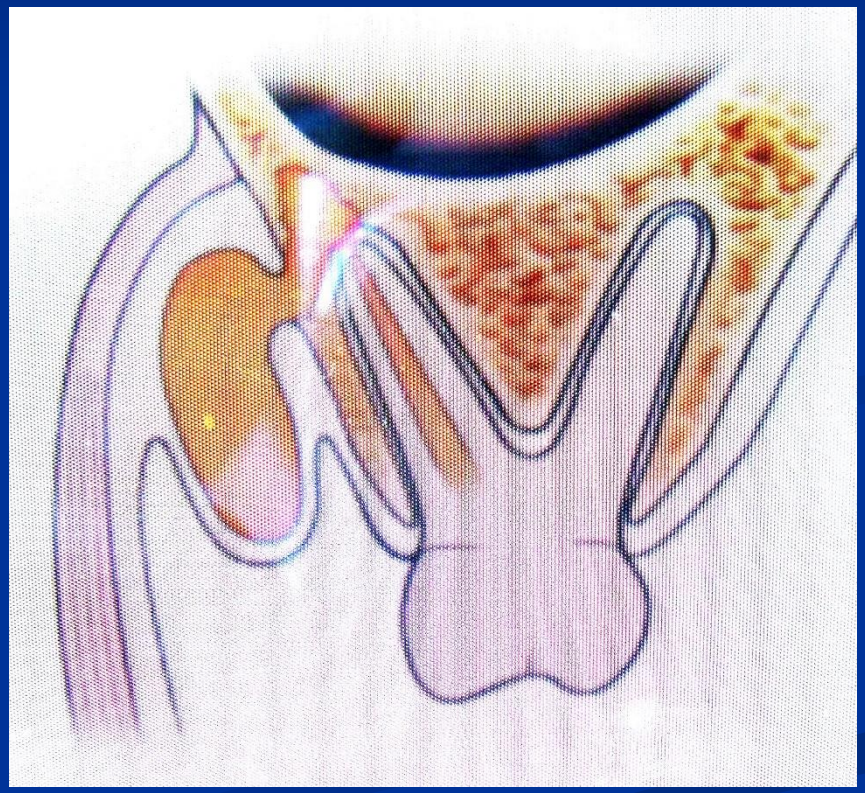
(upper jaw)



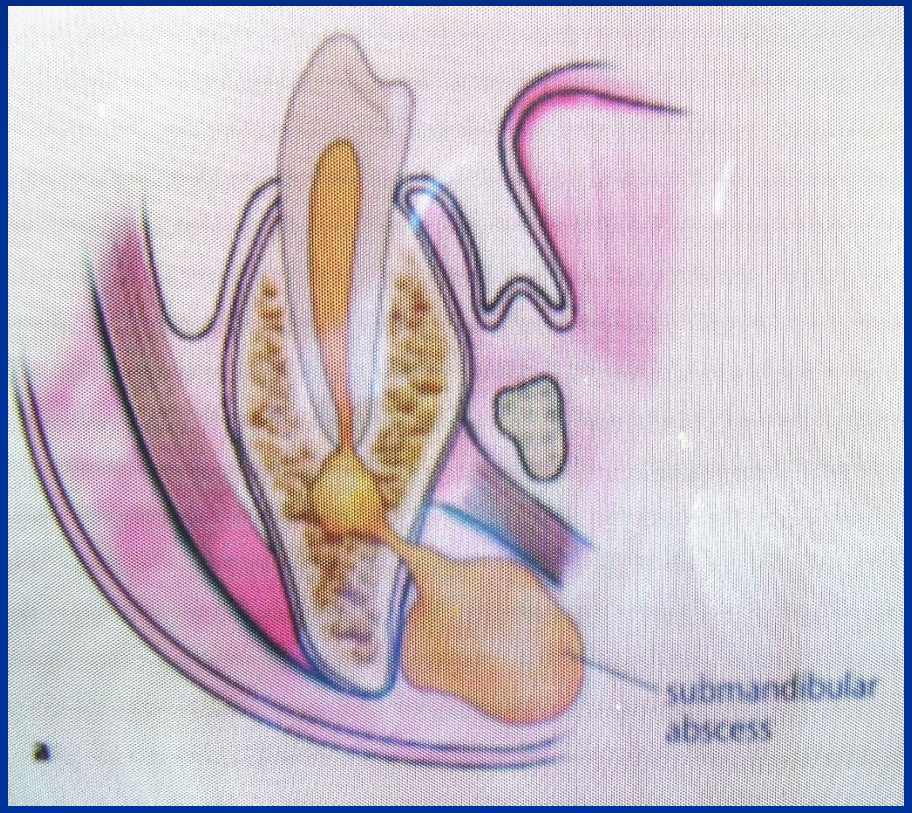


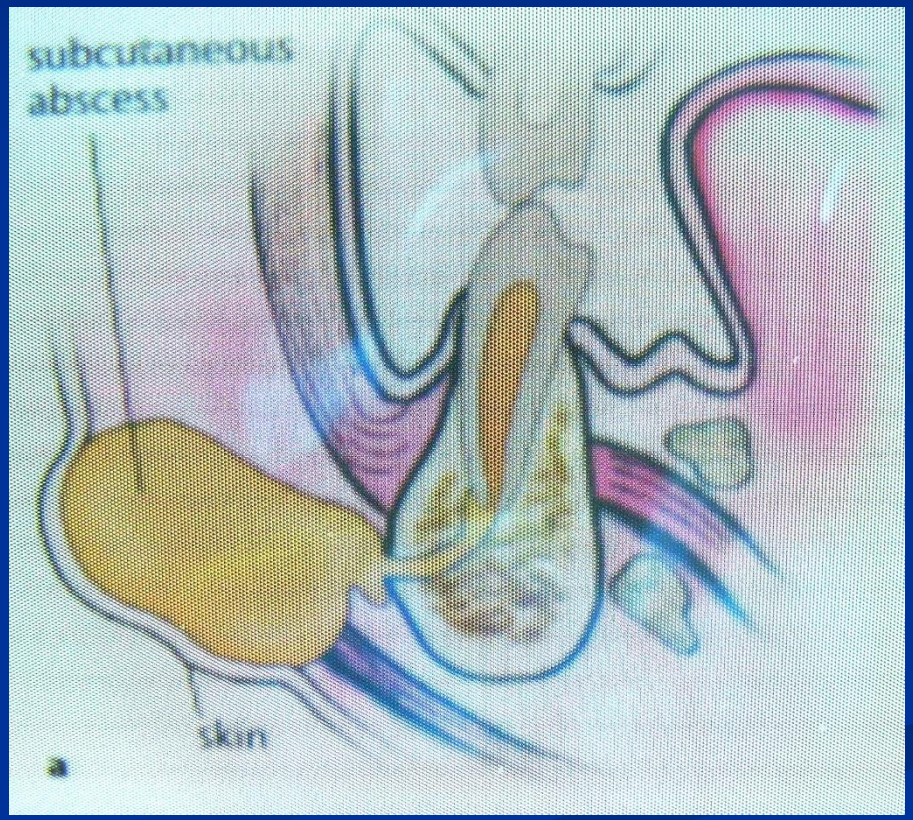
Dento-alveolar abscess
(upper jaw)



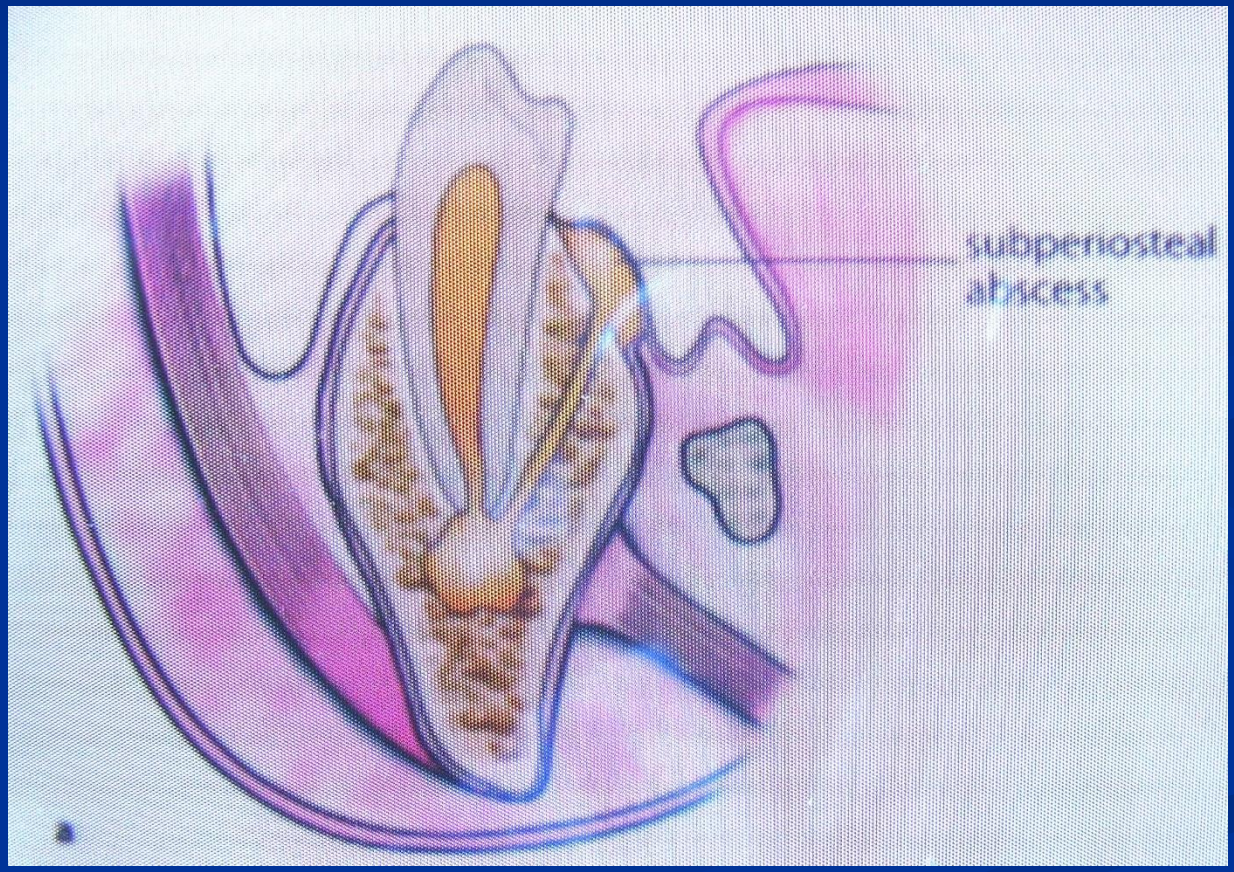


Dento-alveolar abscess
(upper jaw)





Dento-alveolar abscess
(lower jaw)



Dentoalveolar abscess (subperiosteal)

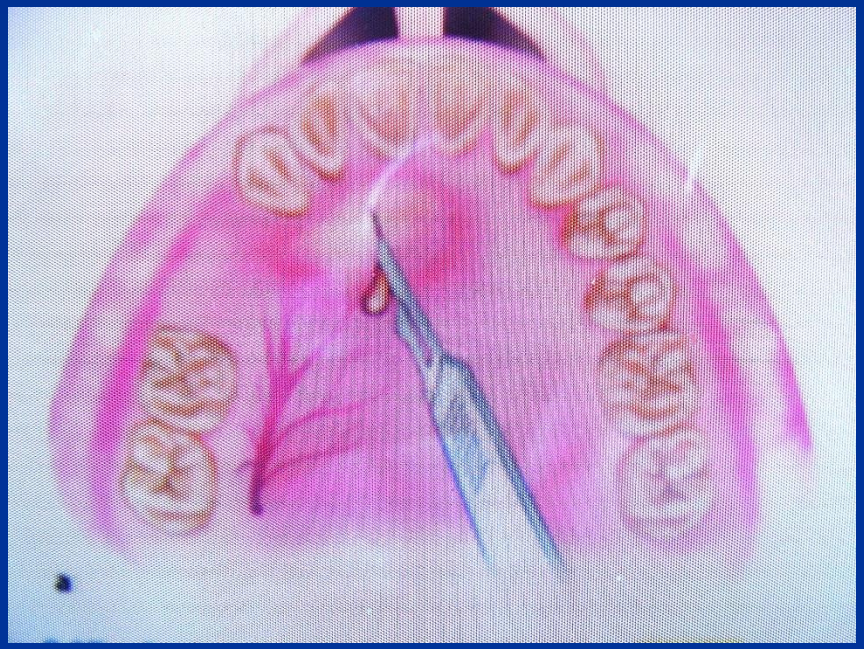
- Localized pain and swelling (may progress over a few hours to days): Examples of swelling are shown in the following image.

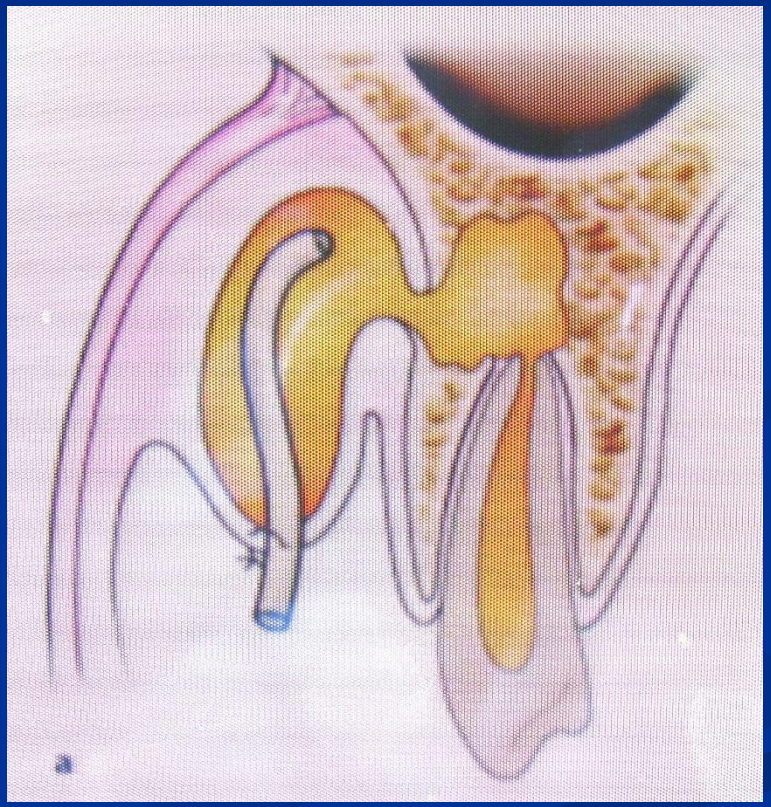
Dentoalveolar abscess (subperiosteal)

- Swelling
- Warmth
- Erythema
- Fluctuant mass that usually extends toward the buccal side of the gum and to the gingival-buccal reflection

Dentoalveolar abscess (subperiosteal)

Dentoalveolar abscess (subperiosteal)





Pathways of acute and chronic periapical infection

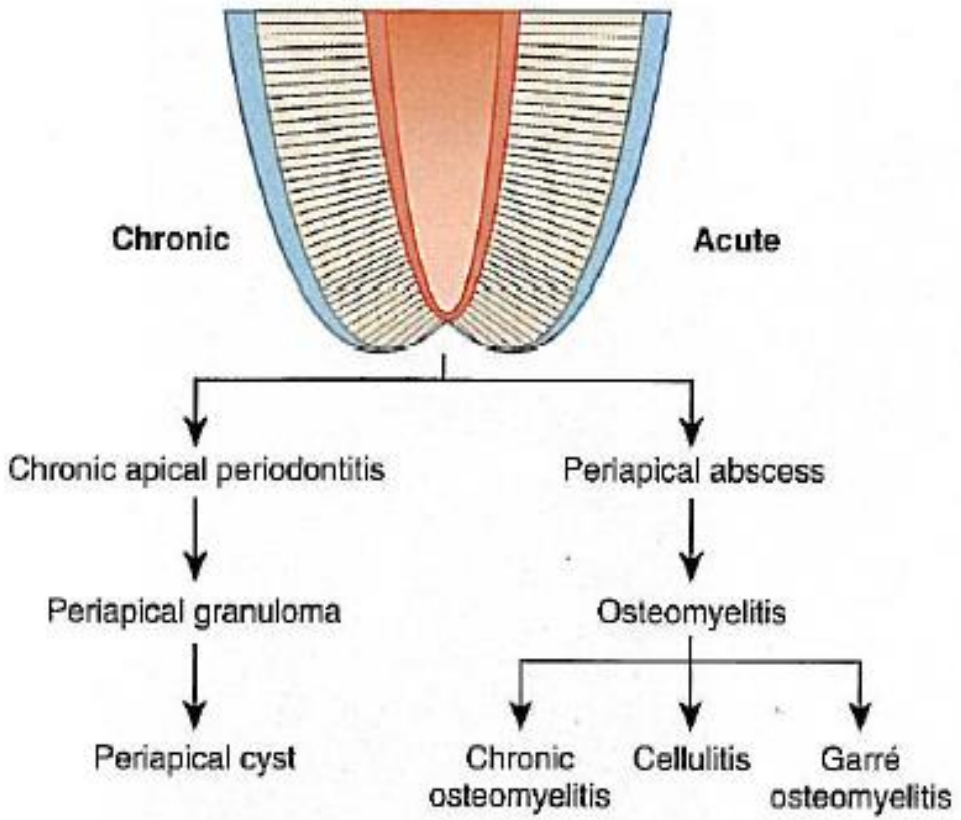


FIGURE 3-23

Periapical infections. Chronic and acute pathways that untreated infections and their accompanying clinical lesions may take, depending on the type of the preceding pulpitis, virulence of the bacteria, and the presence or absence of drainage.

Classification of osteomyelitis in maxillofacial region in children:

On the way of penetration of infection:

- a) odontogenic;
- b) nonodontogenic:
- – vascular (hematogenic, lymphogenic);
- – stomatogenic;
- – posttraumatic;
- – contact.

Depending on the type of infection:

- a) specific (syphilitic, tubercular, actinomycotic) – in children these forms meet rarely;
- b) heterospecific (banal):
- – caused by the incorporated action of strepto-and staphylococcus flora;
- – by fusospirochetal symbiosis;
- – by the incorporated action of anaerobic and aerobic microorganisms;
- – caused by an anaerobic microflora.

Classification of osteomyelitis in maxillofacial region in children

On clinical couse of inflammatory process:

- a) acute;
- b) initially-chronic:
- c) chronic as a result acute:
- – destructive (rarefying);
- – productive (hyperplastic);
- – destructive-productive;
- d) chronic in the stage of exacerbation.

On anatomical location:

- a) osteomyelitis upper or lower jaw (with concrete localization of process);
- b) osteomyelitis of other bones of maxillofacial region.
- On prevalence of process:
- a) focal (localized);
- b) generalized.

Classification

- Classifications proposed are based on different aspects such as clinical course, pathological–anatomical and/or radiological features, etiology, and pathogenesis.
- A mixture of these classification systems has been used in many instances, leading to confusion and thereby hindering comparative studies and obscuring classification criteria.

Reference	Classification	Classification criteria
Schelhorn P, Zenk W [*Clinics and therapy of the osteomyelitis of the lower jaw*]. *Stomatol DDR 1989 Oct;39(10):672-6*	I. Acute osteomyelitis II. Secondary chronic osteomyelitis III. Primary chronic osteomyelitis IV. Special forms – Osteomyelitis sicca (pseudo-paget Axhausen) – Chronic sclerosing osteomyelitis Garrè	Classification based on clinical picture
Topazian RG *Osteomyelitis of the Jaws. In Topizan RG, Goldberg MH (eds): Oral and Maxillofacial Infections.* *Philadelphia, WB Saunders 1994, Chapter 7, pp 251-88*	I. Suppurative osteomyelitis 1. Acute suppurative osteomyelitis 2. Chronic suppurative osteomyelitis – Primary chronic suppurative osteomyelitis – Secondary chronic suppurative osteomyelitis 3. Infantile osteomyelitis II. Nonsuppurative osteomyelitis 1. Chronic sclerosing osteomyelitis – Focal sclerosing osteomyelitis – Diffuse sclerosing osteomyelitis 2. Garrè's sclerosing osteomyelitis 3. Actinomycotic osteomyelitis 4. Radiation osteomyelitis and necrosis	Classification based on clinical picture, radiology, and etiology (specific forms such as syphilitic, tuberculous, brucellar, viral, chemical, *Escherichia coli* and *Salmonella* osteomyelitis not integrated in classification)

Reference	Classification	Classification criteria
Hudson JW Osteomyelitis of the jaws: a 50-year perspective. *J Oral Maxillofac Surg 1993 Dec;51(12):1294-301*	I. Hematogenous osteomyelitis II. Osteomyelitis secondary to a contiguous focus of infection III. Osteomyelitis associated with or without peripheral vascular disease	Classification based on pathogenesis. From Vibhagool 1993
Hudson JW Osteomyelitis of the jaws: a 50-year perspective. *J Oral Maxillofac Surg 1993 Dec;51(12):1294-301*	I. Anatomic Types Stage I: medullar osteomyelitis – involved medullar bone without cortical involvement; usually hematogenous Stage II: superficial osteomyelitis – less than 2 cm bony defect without cancellous bone Stage III: localized osteomyelitis – less than 2 cm bony defect on radiograph, defect does not appear to involve both cortices Stage IV: diffuse osteomyelitis – defect greater than 2 cm. Pathologic fracture, infection, nonunion II. Physiological class A host: normal host B host: systemic compromised host, local compromised host C host: treatment worse than disease	Dual classification based on pathological anatomy and pathophysiology From Vibhagool 1993 and Cierny 1985

Acute/Subacute Osteomyelitis

- Although acute forms of osteomyelitis are seen only rarely these days, most authors in common medical literature still describe this form as an entity of its own. Mercuri (1991) and Marx (1991) arbitrarily defined the time element as **being 1 month after onset of symptoms.** Endurance past this arbitrary set time limit is then considered as chronic osteomyelitis reflecting the inability of host defense mechanisms to eradicate the responsible pathogen.

Acute/Subacute Osteomyelitis

- The term "subacute osteomyelitis" is not clearly defined in the literature. Many authors use the term interchangeably with acute osteomyelitis, and some use it to describe cases of chronic osteomyelitis with more prominent (subacute) symptoms. In some instances, subacute osteomyelitis is referred to as a transitional stage within the time frame of acute osteomyelitis and corresponds to the third and fourth week after onset of symptoms.

Chronic osteomyelitis

- Many authors agree that chronic osteomyelitis involving the jawbone may be divided in two major categories: suppurative an nonsuppurative forms
- **Chronic suppurative osteomyelitis** is by far the most common osteomyelitis type, which is usually caused by bacterial invasion from a contagious focus. Most frequent sources are odontogenic foci, periodontal diseases and pulpal infections, extraction wounds, and infected fractures. **Pus, fistula, and sequestration are typical clinical findings of this disease.** Clinically and radiographically, a broad spectrum ranging from an aggressive osteolytic putrefactive phase to a dry osteosclerotic phase may be observed

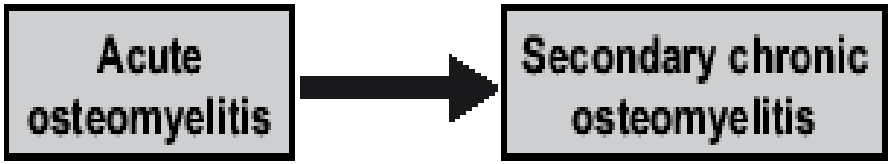
Chronic osteomyelitis

- The term “nonsuppurative osteomyelitis” describes a more heterogenic group of chronic osteomyelitis forms, which lacks the formation of pus and fistula. Topazian 1994, 2002) includes chronic sclerosing types of osteomyelitis, proliferative periostitis, as well as actinomycotic and radiation-induced forms to this group, whereas Bernier et al. (1995) advocate a more restrictive use of this term. Hudson (1993) uses the term to describe a condition of prolonged refractory osteomyelitis due to inadequate treatment, a compromised host, or increased virulence and antibiotic resistance of the involved microorganisms. This classification therefore also incorporates those cases in which a suppurative form of osteomyelitis can present as a nonsuppurative form in an advanced stage.

Periostitis Ossificans, Garres Osteomyelitis

- Strictly periostitis ossificans or ossifying periostitis is, like diffuse sclerosing osteomyelitis, a descriptive term for a condition that may be caused by several similar entities. It is merely a periosteal inflammatory reaction to many nonspecific stimuli, leading to the formation of an immature type of new bone outside the normal cortical layer.

The Zurich classification of osteomyelitis of the jaws

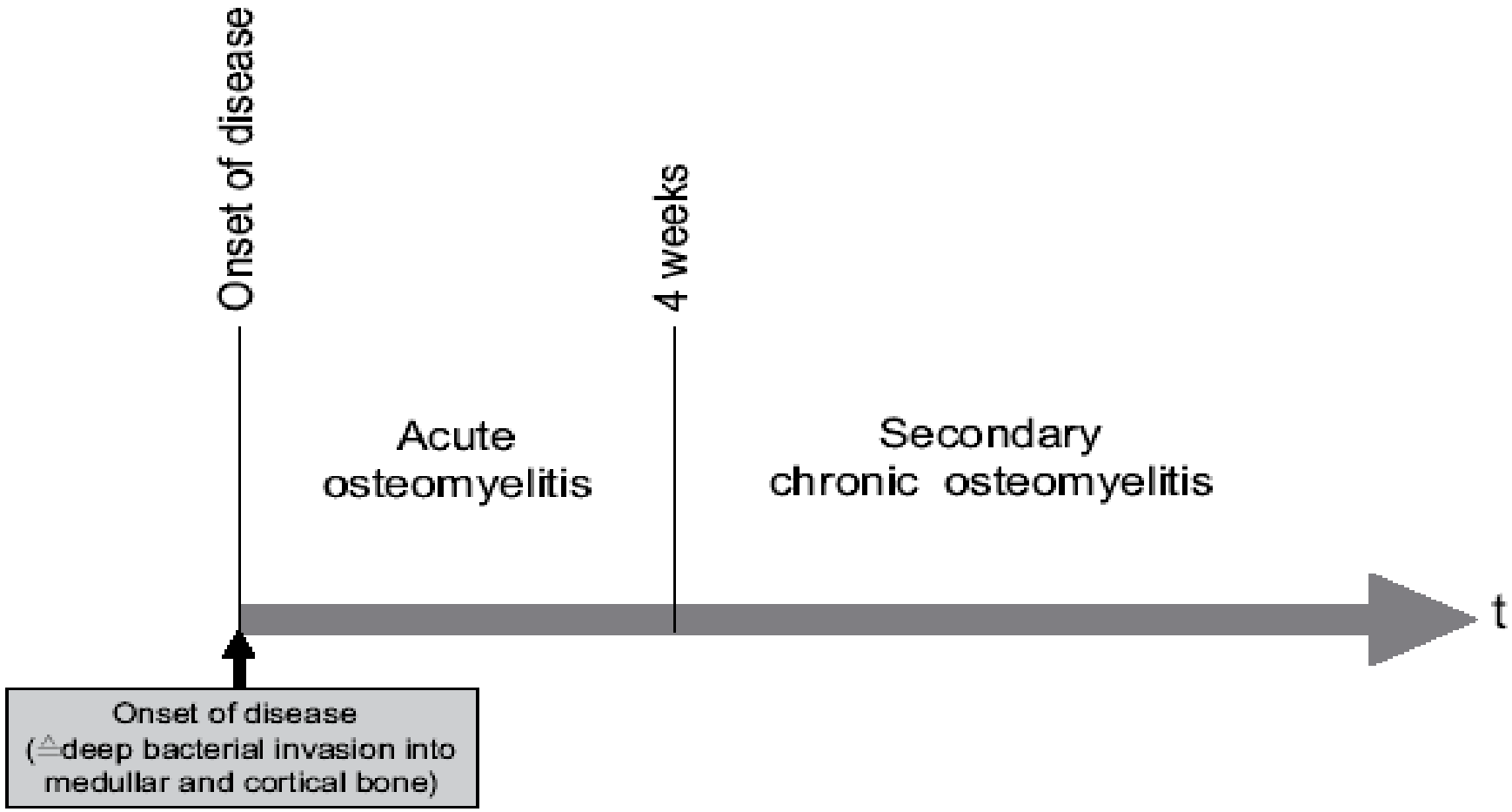


- Neonatal, tooth germ associated
- Trauma/fracture related
- Odontogenic
- Foreign body, transplant/implant induced
- Associated with bone pathology and/ or systemic disease
- Other (not further classifiable) cases

Primary chronic osteomyelitis

- Early onset (juvenile chronic osteomyelitis)
- Adult onset
- Syndrome associated

- The Zurich classification of osteomyelitis of the aws: since secondary chronic osteomyelitis is a sequel of the prolonged and chronified acute form, both basically have the same subclassification groups



- Definition of acute and secondary chronic osteomyelitis of the jawbone

Pathogenesis

- In general, microorganisms may infect bone through one or more of three basic methods: via the bloodstream, contiguously from local areas of infection (as in cellulitis), or penetrating trauma, including iatrogenic causes such as joint replacements or internal fixation of fractures or root-canaled teeth. Once the bone is infected, leukocytes enter the infected area, and, in their attempt to engulf the infectious organisms, release enzymes that lyse the bone. Pus spreads into the bone's blood vessels, impairing their flow, and areas of devitalized infected bone, known as *sequestra*, form the basis of a chronic infection. Often, the body will try to create new bone around the area of necrosis. The resulting new bone is often called an involucrum. On histologic examination, these areas of necrotic bone are the basis for distinguishing between *acute osteomyelitis* and *chronic osteomyelitis*. Osteomyelitis is an infective process which encompasses all of the bone (osseous) components, including the bone marrow. When it is chronic it can lead to bone sclerosis and deformity.

Pathogenesis

- Chronic osteomyelitis may be due to the presence of intracellular bacteria (inside bone cells). Also, once intracellular, the bacteria are able to escape and invade other bone cells. In addition, once intracellular, the bacteria becomes resistant to antibiotics. These combined facts may explain the chronicity and difficult eradication of this disease. This results in significant costs and disability and may even lead to amputation. Intracellular existence of bacteria in osteomyelitis is likely an unrecognized contributing factor to its chronic form.
- In infants, the infection can spread to the joint and cause arthritis. In children, large subperiosteal abscesses can form because the periosteum is loosely attached to the surface of the bone.

Pathogenesis

Explanation for the low incidence of osteomyelitis of the jawbones can be explained by four primary factorswhich are responsible for deep bacterial invasion into the medullar cavity and cortical bone and hence establishment of the infection:

- 1. Number of pathogens
- 2. Virulence of pathogens
- 3. Local and systemic host immunity
- 4. Local tissue perfusion

Pathogenesis

- **Osteomyelitis more commonly affect the mandible than the maxille due to the following factors:**
- **Blood Supply:** The mandible has less blood supply, hence less resistance to infection than the maxilla which **is** much more vascularized with collateral circulation.
- **Cortical Plates:** Of the mandible are much more dense and are not easily penetrated by the infection thus pus is trapped within cortics and is forced to extend through the spongiosa of the mandible. The presence of the inferior dental canal also helps in that extention. The presence of a wide spongy portion in the mandible helps the infection to travel and destroy more areas.
- **Trauma:** The mandible is much more exposed to trauma than the maxilla (more difficult extractions, and high incidence of fracture).

Local and Systemic Host Immunity

- The oral cavity, like no other part of the human body, is constantly exposed to various potential aggressors. Many of these bacteria, given the chance, may cause severe infection and damage to the tissue if they are not kept at distance. Due to its unique environment, many potent strategies have been developed to prevent deep tissue invasion of bacteria. **Specific local immunological mechanisms**, potent barrier systems, such as the periodontal membrane and a rich local vascular supply, are the most important.

Local and Systemic Host Immunity

- Chronic infection of the periapical bone as a sequel of endodontic disease. This frequently observed condition represents a classical equilibrium between microbiological aggressors and host factors hindering further spread of the bacteria. If this balance is disturbed and shifts toward the side of the microorganisms, deep invasion into the medullar and cortical bone may occur and osteomyelitis is established.

Local and Systemic Alterations in Bone Vascularity

Local and systemic factors altering bone vascularity

- Smoking
- Diabetes mellitus
- Florid osseous dysplasia
- Fibrous dysplasia
- Paget's disease
- Osteopetrosis (Albers–Schonberg Disease)
- Osteoporosis
- Bisphosphonate induced osteochemonecrosis
- Other forms of osteonecrosis (mercury, bismuth, arsenic)
- Tobacco
- Radiation therapy and osteoradionecrosis
- Bone malignancy (primary or metastatic)

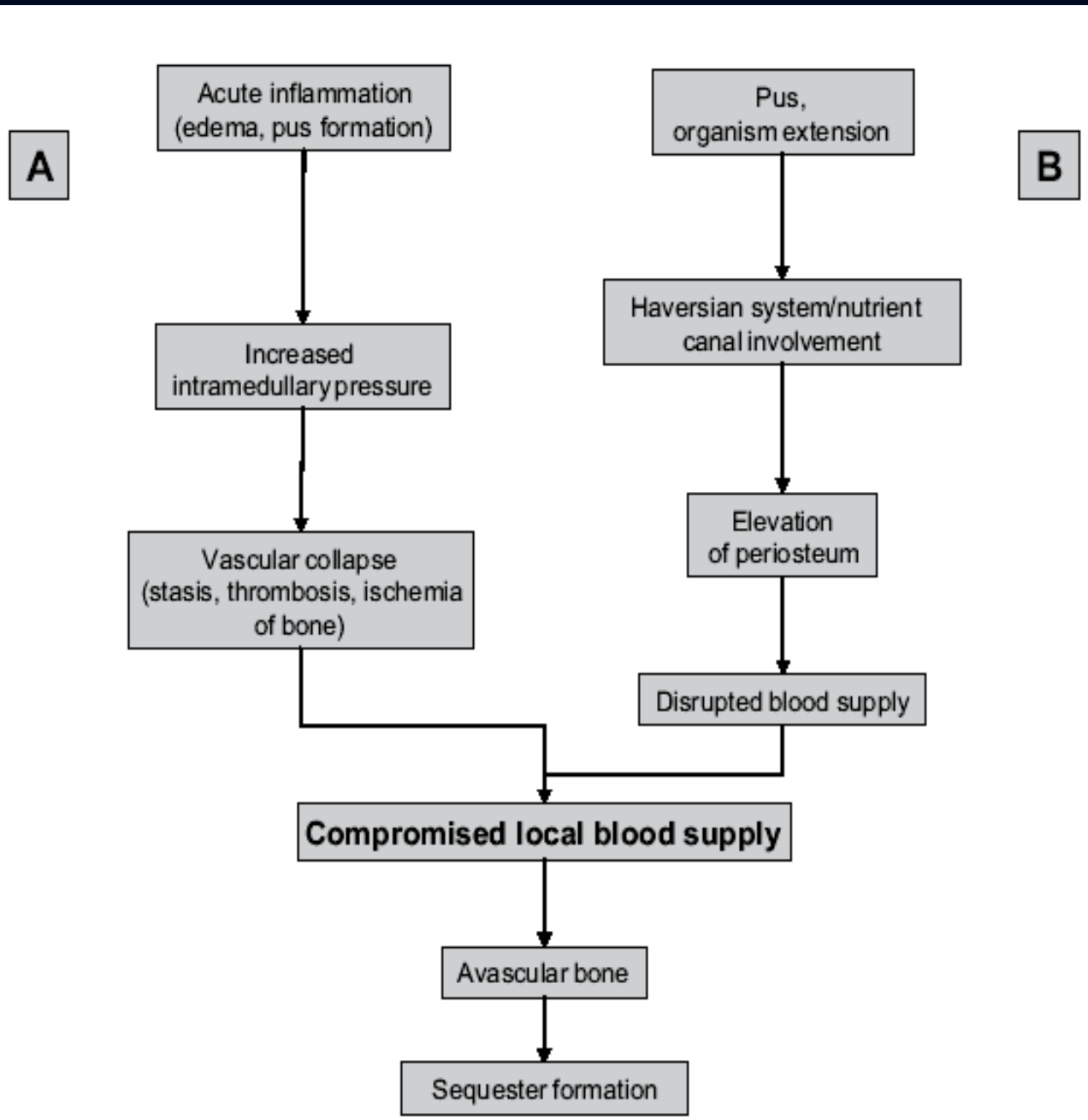
- Compromise of local blood supply must be considered a critical factor in the establishment of osteomyelitis. Systemic and local conditions that alter the vascularity of bone predispose the development of osteomyelitis. In these conditions immune cells and oxygen cannot reach the target area in an adequate manner. This facilitates the growth and spread of microorganisms, especially anaerobes, leading to establishment and progression of osteomyelitis. An overview of conditions compromising

Systemic factors altering host immunity

- Diabetes mellitus
- Autoimmune disorders
- AIDS
- Agranulocytosis
- Anemia (especially sickle cell)
- Leukemia
- Syphilis
- Malnutrition
- Chemotherapy
- Corticosteroid and other immunosuppressive therapy
- Alcohol and tobacco
- Drug abuse
- Prior major surgery
- Herpes simplex virus (Zoster) and cytomegalovirus infection

- Every systemic disease with concomitant alterations host defenses may influence profoundly the onset and course of acute and secondary chronic osteomyelitis

Disease	Mechanism facilitating bone infection
Diabetes	Diminished leukocyte chemotaxis, phagocytosis, and lifespan; diminished vascularity of tissue due to vasculopathy, thus reducing perfusion and the ability for an effective inflammatory response; slower healing rate due to reduced tissue perfusion and defective glucose utilization
Leukemia	Deficient leukocyte function and associated anemia
Malnutrition	Reduced wound healing and reduction of immunological response
Cancer	Reduced wound healing and reduction of immunological response
Osteopetrosis (Albers–Schonberg disease)	Reduction of bone vascularization due to enhanced mineralization, replacement of hematopoietic marrow causing anemia and leukopenia
Severe anemia (particularly sickle-cell anemia)	Systemic debilitation, reduced tissue oxygenation, bone infarction (sickle cell anemia), especially in patients with a homozygous anemia trait
IV drug abuse	Repeated septic injections, spreading of septic emboli (especially with harboring septic vegetation on heart valves, in skin or within veins)
AIDS	Impaired immune response
Immunosuppression (steroids, cytostatic drugs)	Impaired immune response



- **Pathogenesis of acute and secondary chronic osteomyelitis of the jaws.** Pathway *A* shows the role of inflammation and pathway *B* the role of pus formation in compromising blood supply of the infected bone, which can be considered as the final common pathway in the formation of sequestra (

Clinical findings

- Cases of acute osteomyelitis of the jawbone with an acute suppurative clinical course usually show impressive signs of inflammation. Pain can be intense and is mostly described by a deep sensation within the bone by the patient, which may be a valuable clue in the patient's history. Local swelling and edema due to abscess formation can also be substantial causing trismus and limitation of jaw function. The patients experiences a general malaise caused by high intermittent fever with temperatures reaching up to 39–40°C, often accompanied by regional lymphadenopathy. In some instances paresthesia or anesthesia of the lower lip is described (Vincent's symptom), indicating involvement of the inferior alveolar nerve. In most cases the cause of infection is odontogenic and can easily be Pus may exude around the gingival sulcusthrough mucosal and, possibly cutaneous, fistulas.

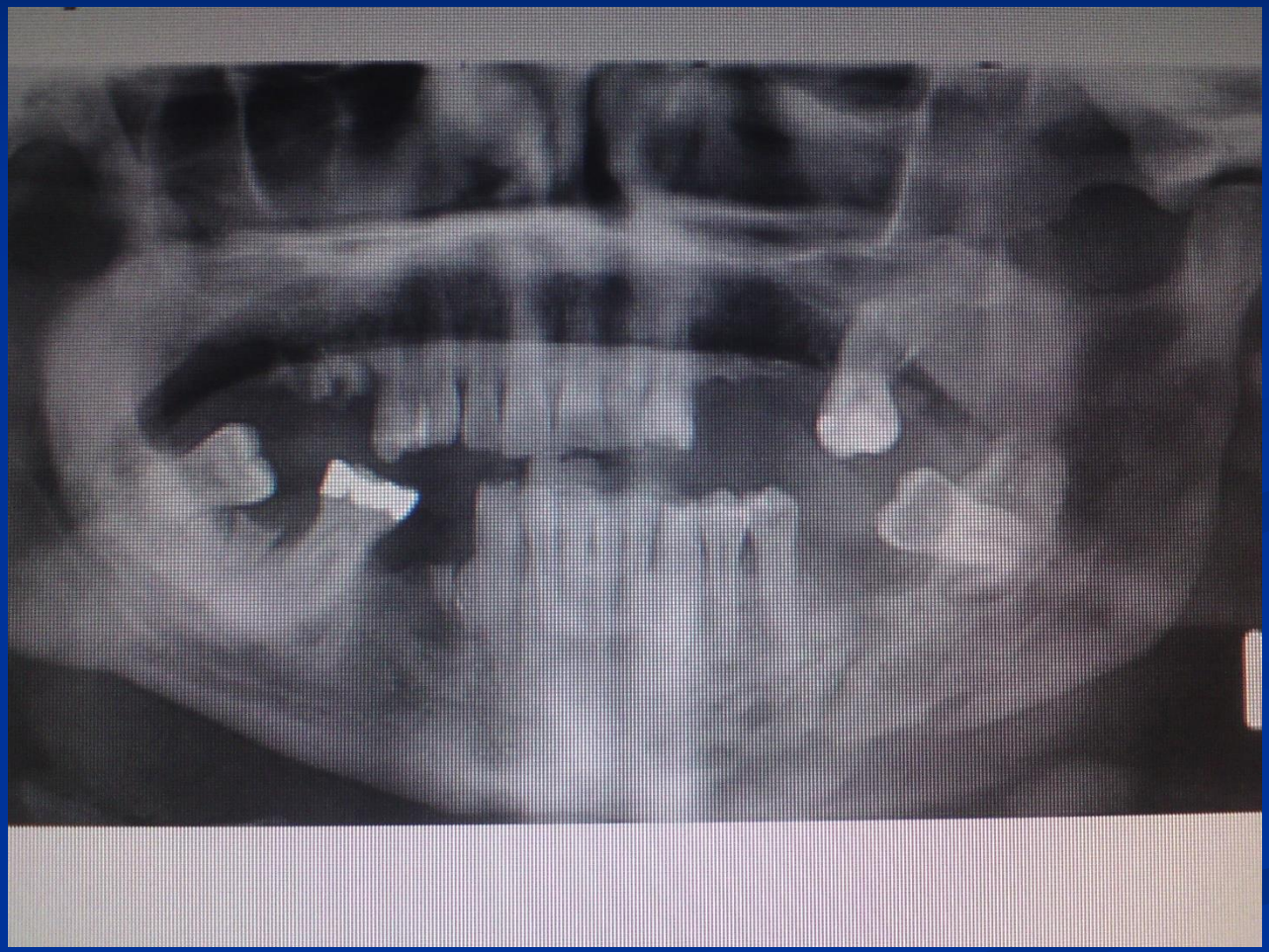
Clinical picture

Clinical picture

Clinical picture

Clinical picture

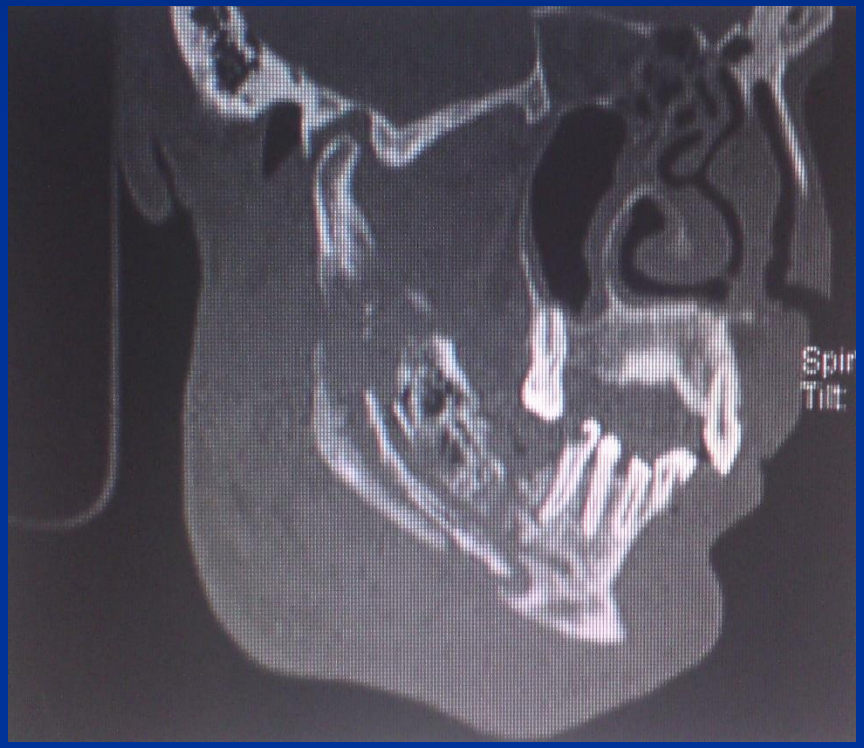
Dignostic images

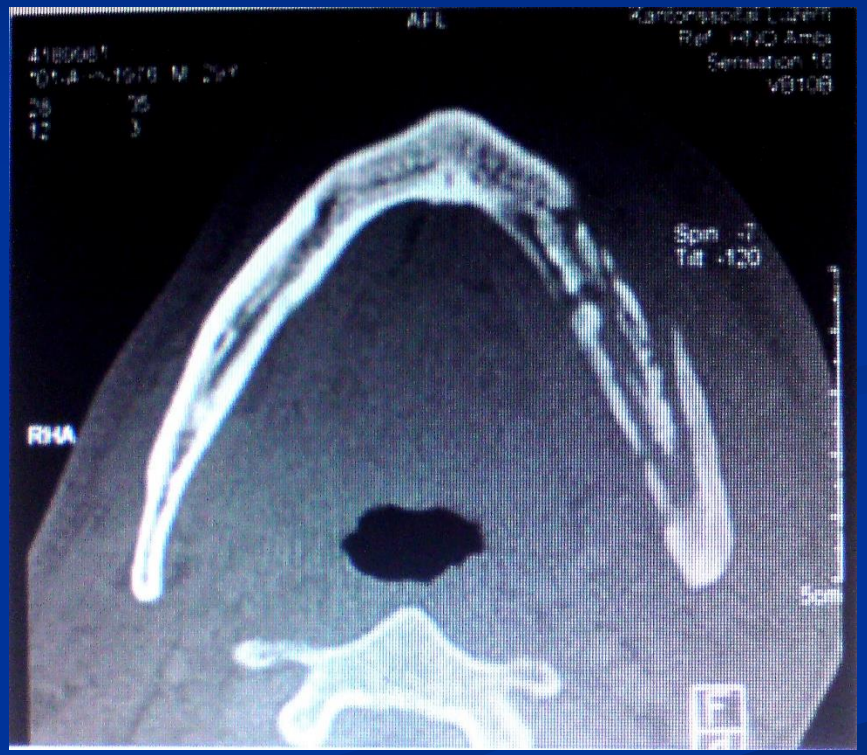


Dignostic images

- **Acute osteomyelitis**. Radiograph of pasterior mandible exhibiting the mottled and blotchy pattern of radiolucent and radiopaque areas, indistinct borders and islands of residual bone (sequestra)

Dignostic images





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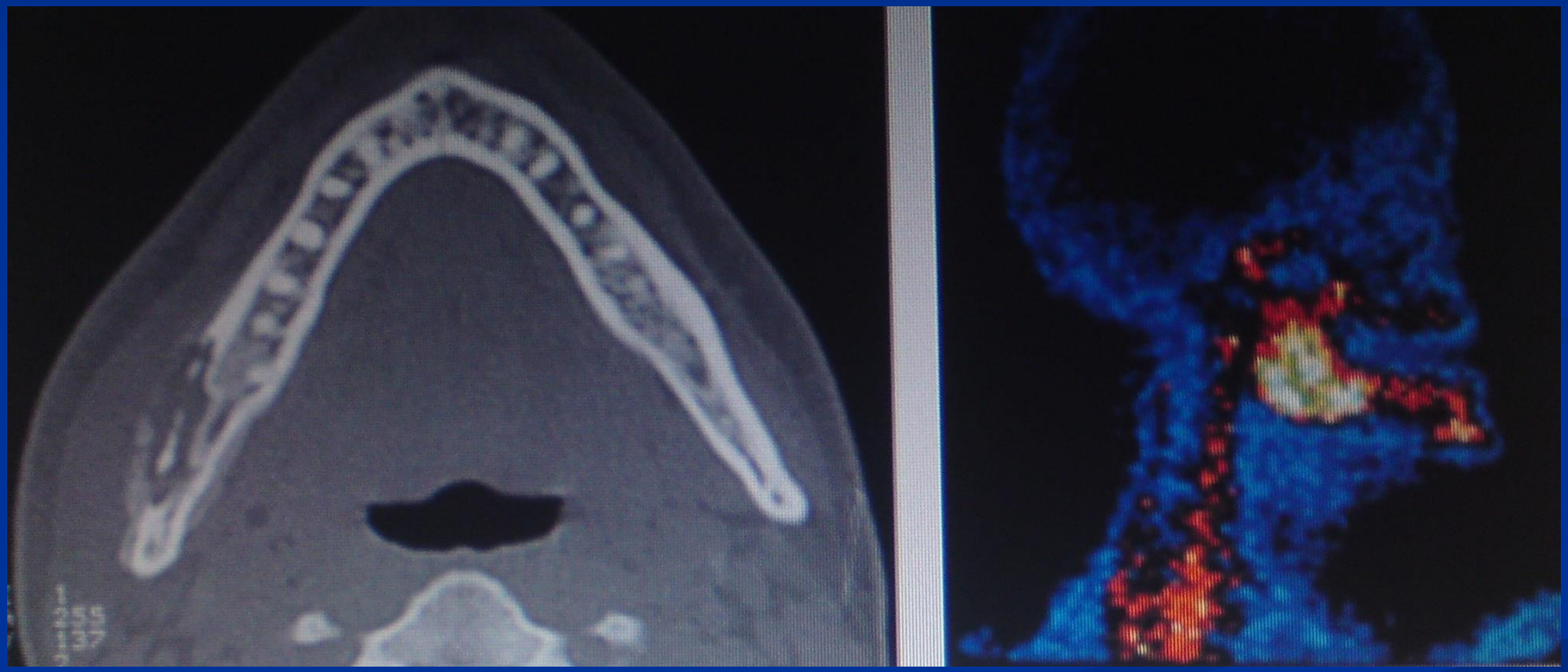

Treatment

I. **General supportive measures (acute** and **chronic):**

- Bed rest or hospitalization.
- Good nutrition (polyvitamins, high protein and high caloric soft diet).
- Restoration of fluid balance.
- Sedatives and analgesics.
- Diagnosis and treatment of the underlying systemic disease if present e.g. syphilis or diabetis etc.

II. **Heat therapy (acute and chronic).**

- **Antibiotics (acute and chronic).**

Treatment

Surgical procedures in the treatment:

- I & D in soft tissue or intrabony (acute and chronic).
- Removal of the etiologic teeth or cause (subacute, chronic).
- Decortication.
- Sequestrectomy.
- Saucerization.
- Dissection and excision of fistulas.
- Jaw immobilization by intermaxillary fixation.
- Resection and bone graft.

V. Hyperbaric Oxygen, (chronic).