Osteomyelitis of jaws. Classification, features of motion, diagnostics, prophylaxis, medical treatment, prognosis.

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LECTURE PLAN

1. Definition of osteomyelitis as a disease.
2. Etiology of osteomyelitis.
3. Theories of the pathogenesis of osteomyelitis.
4. Osteomyelitis classification.
5. The clinical course of osteomyelitis:
   - general manifestations;
   - local symptoms;
   - differences in the flow on the upper and lower jaws.
6. Characteristics of the acute and chronic phases of osteomyelitis.
7. Principles of complex treatment.
8. Prevention of osteomyelitis.

OSTEOMYELITIS OF JAWS – INFECTIOUS-ALLERGIC PURULENT - NECROTIC INFLAMMATORY PROCESS, WHICH ARISES IN A BONE UNDER INFLUENCE AS EXOGENOUS (PHYSICAL, CHEMICAL, BIOLOGICAL), AND ENDOGENIC (NEUROHUMORAL, THE ENDOINTOXICATION) FACTORS ON A BACKGROUND OF THE PREVIOUS SENSIBILIZATION AND SECONDARY IMMUNOSUPPRESSION OF AN ORGANISM AND IS ACCOMPANIED BY A NECROSIS OF A BONE TISSUE.

Classification

Originally:	An extension:
• odontogenous	• limited (within the scope parodont 2-3 tooth)
• traumatic: - mechanical trauma - radiation trauma	• focal (alveolar process and parts ramus or body of the jaws)
• hematogenic	• diffuse (total injury of jaw)
• specific	

1. Acute osteomyelitis

a. Contiguous focus (osteomyelitis due to spread from a **contiguous focus of infection** without vascular insufficiency, is seen most often after trauma or surgery, and is caused by bacteria which gain access to bone by direct inoculation (for example, a contaminated compound fracture)

b. Progressive

c. Hematogenous

2. Chronic osteomyelitis

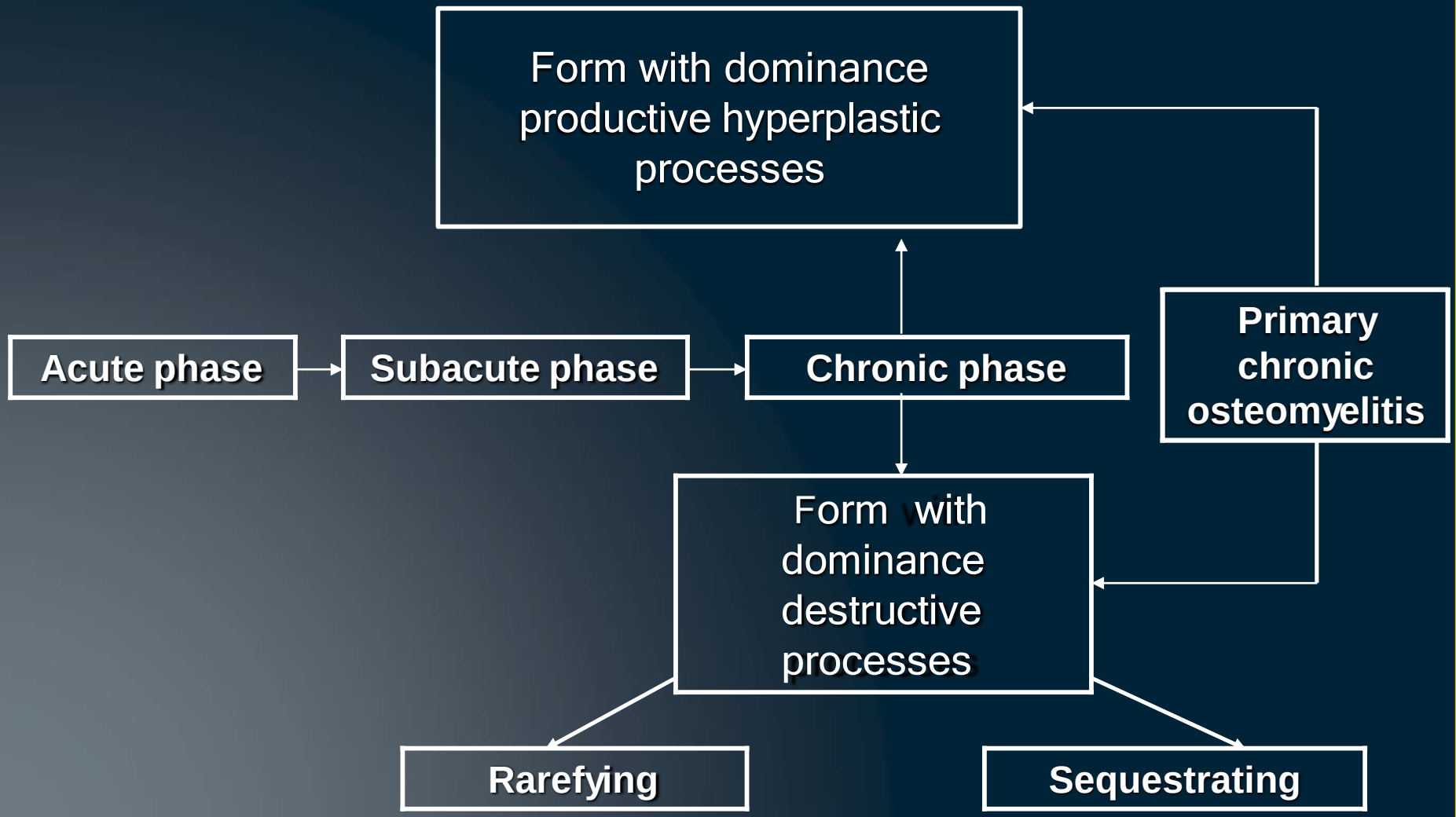
a. Recurrent multifocal

b. Garré's (**periostitis ossificans**)

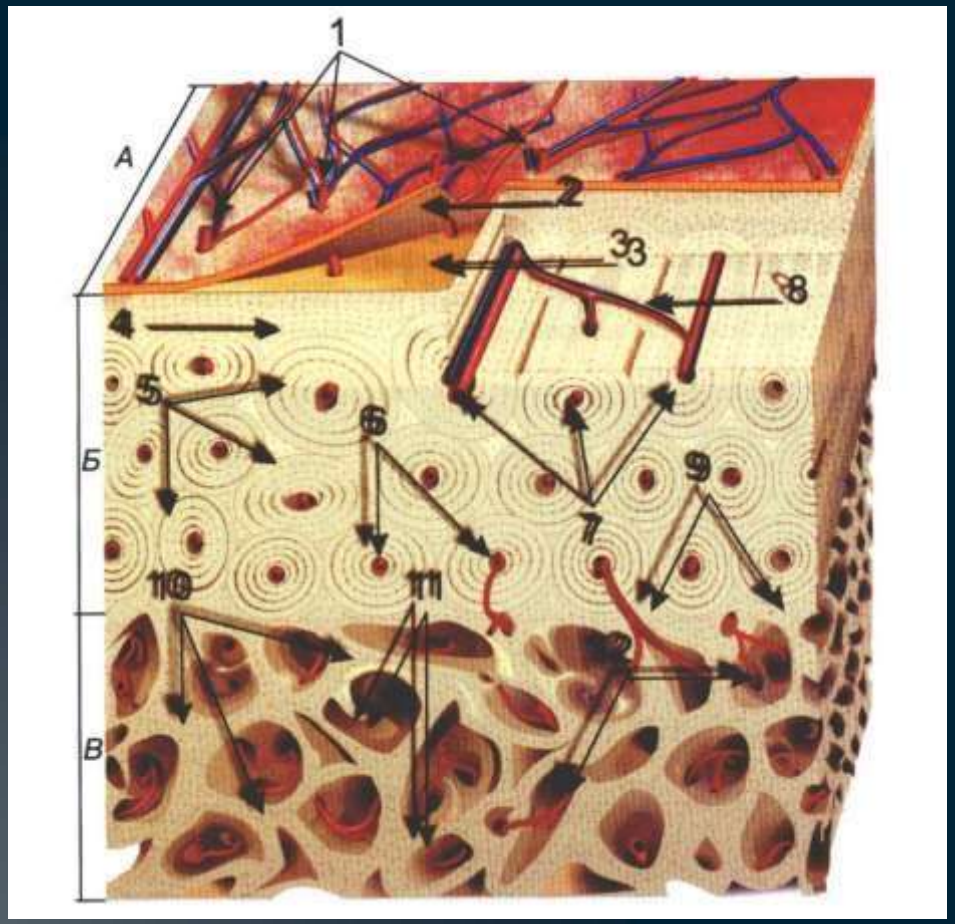
c. Suppurative or nonsuppurative

d. Sclerosing

Clinical-radiologic classification



Bone structure



А – periosteum; Б - compact layer; *В* – spongiosus layer; 1 - vessels; *2* – osteogenous layer of *periosteum*; *3 – periostal surface of the bone*; *4* – external layer overall общих plates; *5* – osteons; *6 – Haversian canals of osteons*; 7 - vessels of the osteons; *8 – Volkmann's canal (vesells)*; 9 - internal layer overall plates; *10* – trabeculs; *11 – inter*trabeculs spaces; *12* – vessels of spongiosus layer.

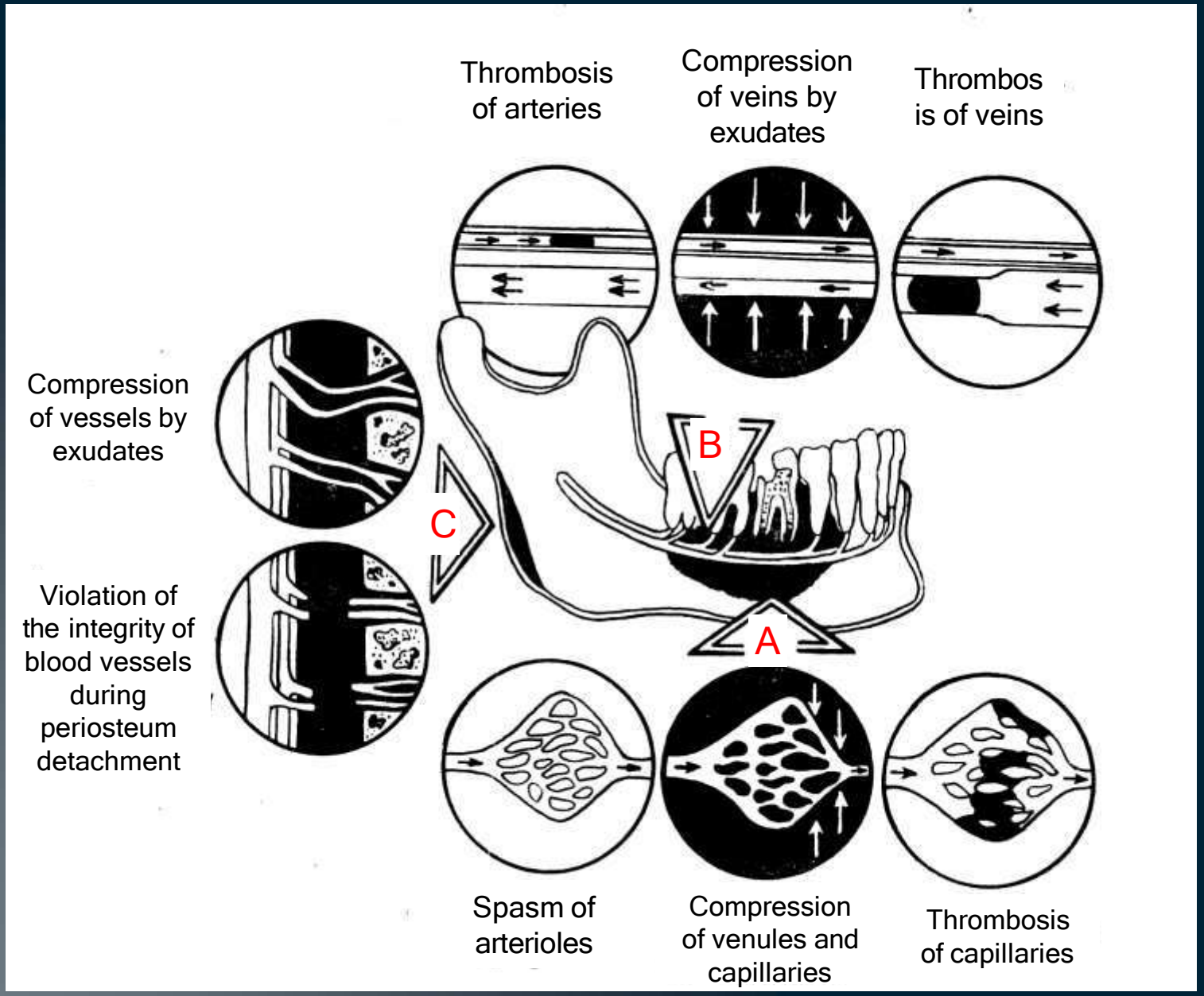
Pathogenesis

A. When the micro-organisms gain access to the bone it provokes an acute suppurative inflammation. The bacteria the pH change, the local oedema which accumulate under pressure, the leukocytes, all contribute to the necrosis of tissue, the break-down, of bone trabeculae, and the removal of matrix and calcium deposits.

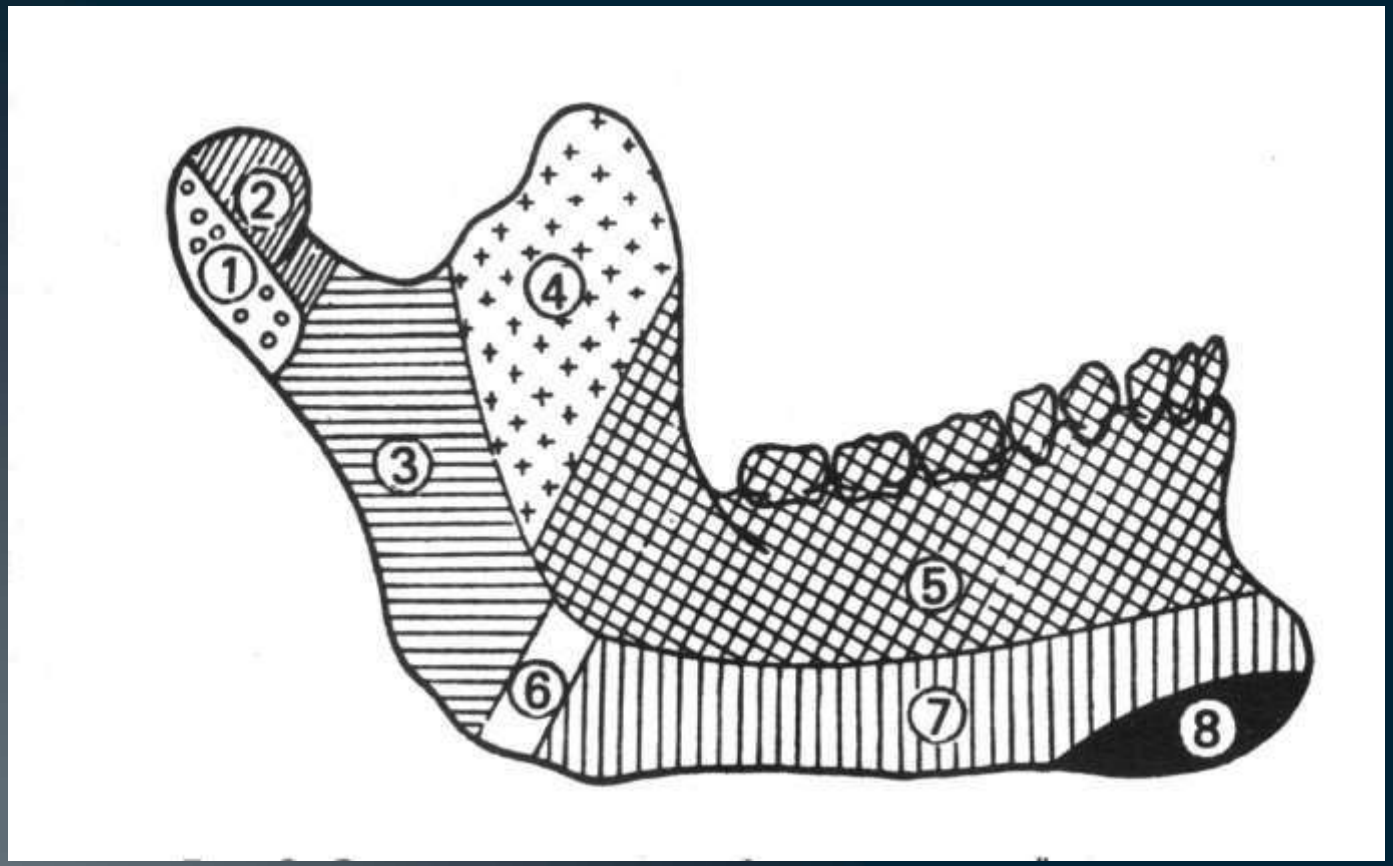
B. The part played by vascular obstruction is very important. Within few days thrombosis of vessels occur because of increased intramedullary pressure. This is followed by ischemia and bone necrosis. The infection extends to the neighboring osseous structures through the haversian and Volkmann's canals. This shuts off more vascular channels and causes the death of more osteocytes in their lacunae. Large segments of bone devoid of any blood supply can become separated to form the well known sequestra. Meanwhile, osteoblastic opposition can take place on smaller pieces of already dead bone.

C. When the infection reaches the outer part of the cortex, it causes stripping of the periosteum from the underlying cortical bone and results in a subperiosteal abscess. The stripping up of the periosteum interferes with the periosteal blood supply to the immediate underlying portion of cortical plate, which may then undergo progressive avascular necrosis leading to superficial minute sequestration. The accumulation of bacteria and its toxic products underneath the periosteum induces the formation of subperiosteal bone (involucrum). Destruction of the cortex can be followed by a spontaneous, pathological fracture.

Механізм порушення мікроциркуляції



The scheme of the blood supply to the lower jaw according to V.M.Uvarov



1 - maxillary artery; 2 - artery of the pterygoid canal; 3 - the transverse artery of the face; 4 - chewing artery; 5 - inferior alveolar artery; 6 - mylo-hyoid branch; 7 - facial artery; 8 - lingual artery.

The basic clinical signs.

A) Cuff inflammatory infiltrate, which covers a jaw;

B) Paresthesia on a course of the conforming nerve;

C) Morbid percussion next, with causal, teeth;

D) Change percussion of a note of teeth;

E) Absence of enriching of a state of the patient after a incision pyogenic abscess and extraction causal of tooth, intensifying of a flow of pus, change of character of pus from yellow thick to dirty - grey, infrequent;

I) Absence of down stroke of temperature of a body after a surgical intervention.

The pain in osteomyelitis is often described as a deep and boring pain, which is often out of proportion to the clinical picture. In acute osteomyelitis it is very common to see swelling and erythema of the overlying tissues, which are indicative of the cellulitic phase of the inflammatory process of the underlying bone.

Fever often accompanies acute osteomyelitis, whereas it is relatively rare in chronic osteomyelitis.

Paresthesia of the inferior alveolar nerve is a classic sign of a pressure on the inferior alveolar nerve from the inflammatory process within the medullary bone of the mandible.

Trismus may be present if there is inflammatory response in the muscles of mastication of the maxillofacial region.

The patient commonly has malaise or a feeling of overall illness and fatigue, which would accompany any systemic infection.

Lastly both intraoral and extraoral fistulas are generally present with the chronic phase of osteomyelitis of the maxillofacial region.

The management of osteomyelitis

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.

!Clearly the first step in the treatment of osteomyelitis is diagnosing the condition correctly. The tentative diagnosis is made from clinical evaluation, radiographic evaluation, and tissue diagnosis. The clinician must be aware that malignancies can mimic the presentation of osteomyelitis and must be kept in the differential diagnosis until ruled out by tissue histopathology.

Tissues from the affected site should be sent for Gram stain, culture, sensitivity, and histopathologic evaluations. The clinical response to the treatment of any patient will be compromised unless altered host factors can be optimized. Medical evaluation and management in defining and treating any immunocompromised state is indicated and often helpful. For example, glucose control in a diabetic patient should be stabilized for best response to therapy.

Empiric antibiotic treatment should be started based on Gram stain results of the exudate or the suspected pathogens likely to be involved in the maxillofacial region. Definitive culture and sensitivity reports generally take several days or longer to be obtained but are valuable in guiding the surgeon to the best choice of antibiotics based on the patient's specific causative organisms. Infectious disease consultation may illustrate the most current antimicrobials and/or regimens.

Supporting the weakened area with a fixation device (external fixator or reconstruction type plate) and/or placing the patient in maxillomandibular fixation is frequently used to prevent pathologic fracture. Indeed, we have primarily grafted such areas when the sequestrectomy and saucerization have been deemed adequate.

Some authors have proposed adjunctive treatment methods that deliver high doses of antibiotic to the area using antibiotic impregnated beads or wound irrigation systems. This therapy works on the premise that high local levels of antibiotics are made available and the overall systemic load is very low, thus reducing the possible side effect and complication rate.

Hyperbaric oxygen (HBO) treatment has also been advocated for the treatment of refractory osteomyelitis. This treatment method works by increasing tissue oxygenation levels that would help fight off any anaerobic bacteria present in these wounds. The widespread use of HBO treatment of osteomyelitis still remains ontroversial.

Chronic osteomyelitis.

Proceeds from 4-6 weeks about several months, or even of years.

The clinical picture of a chronic osteomyelitis is caused by formation demarcation of the shaft around of necrotic fragment of a bone, appearance of fistulas on a mucosa of an oral cavity or on a skin and casting-off of sequesters (necrotic of fragments of a bone, which can be various under the form and dimensions - from very small up to large).

Together with a sequestration during a chronic phase of an osteomyelitis the proliferative phenomena periosteal and endoosseous of a parentage educe. On these causes the chronic phase of an osteomyelitis is the longest term of disease and the major value in clinic is got by local changes in a bone and accumbent mild tissues, at the same time reaction of an integrated organism insignificant.

The transition of an acute phase of an osteomyelitis in chronic descends gradually, slowly. The pain in a field of a jaw remits, the infiltration of mild tissues decreases, temperature of a body is reduced, the picture of a blood is normalized.

As the first clinical sign of a chronic osteomyelitis it is necessary to be considered formation of fistulas. The appearance of granulations from fistulas specifies a casting-off of sequesters.

The formation of sequesters on the upper jaw descends within 3-4 week from a beginning of disease, on inferior - 6-8, sometimes 12-14 weeks. The teeth in the locus of an inflammation are loosened.

On the upper jaw sequestration huge fragments in limens of an alveolar process. On a mandible in process a body sometimes can be involved.

At development of a destructive osteomyelitis of a mandible there can be a pathological fracture.

Visually and palpation in a region osteomyelitis of the locus it is possible to note a thickening or inflating of a bone at the expense of a thickening of a periosteum. The probing of a fistula sometimes allows to define presence of a sequester.

At a chronic osteomyelitis the parallel development of processes of a destruction and neogenesis is observed, and dominate regenerative or osteoplastic processes.

Surgical Options

Classic treatment is sequestrectomy and saucerization. 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. 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. The key element in the above procedures is determined clinically by cutting back to good bleeding bone. Clinical judgment is crucial in these steps but can be aided by preoperative imaging that shows the bony extent of the pathology. It is often necessary to remove teeth adjacent to an area of osteomyelitis. In removing adjacent teeth and bone the clinician must be aware that these surgical procedures may weaken the jaw bone and make it susceptible to pathologic fracture

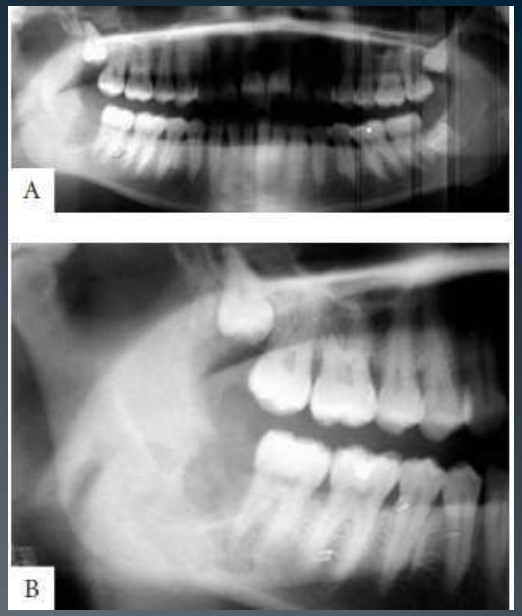
Pathological anatomy of a chronic osteomyelitis.

On border necrotizing and escaped fragment of a bone educes granulation a tissue, which contains osteoclasts. Simultaneously there is a neogenesis of a bone around of a sequester, this young tissue forms sequestral capsule. The sequesters can be: petty loop (inside), is large - loop (outside). The pus from capsule leaves through a fistula.

On a roentgenogram of change occur for 21 day.

In a chronic phase at first there are locuses of a destructions surrounded dense sclerosed with walls, that is caused by the locus of neogenesis of a bone around of a sequester. Sequesters are defined later. The shade of a sequester is seldom excreted on a background of more transparent bone elements.

Hyper osteitis of the form the appreciable difficulties for diagnostics represent sometimes, them sometimes diagnosticate by means of a biopsy.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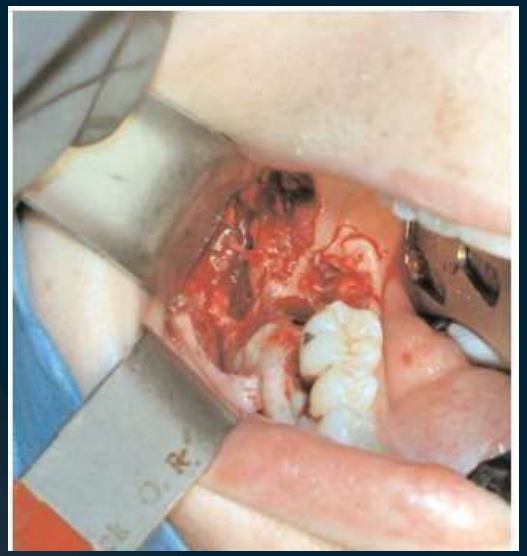

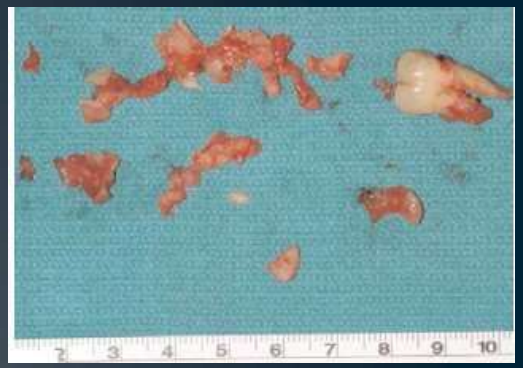
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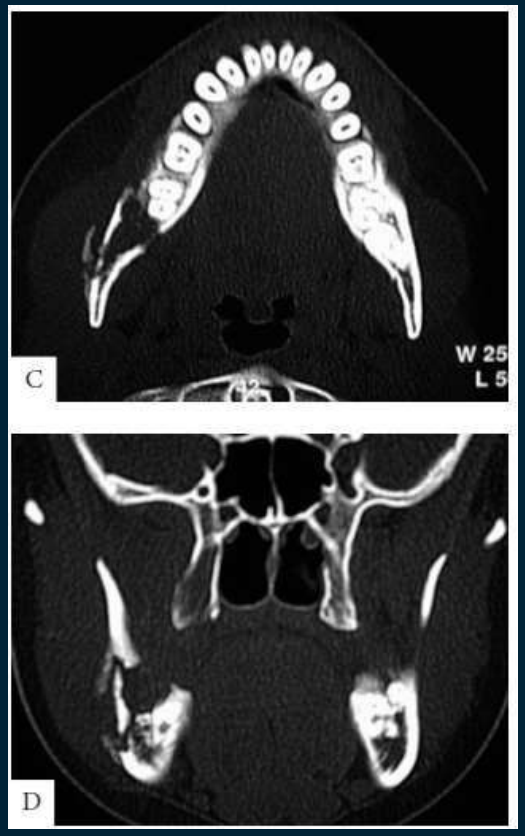
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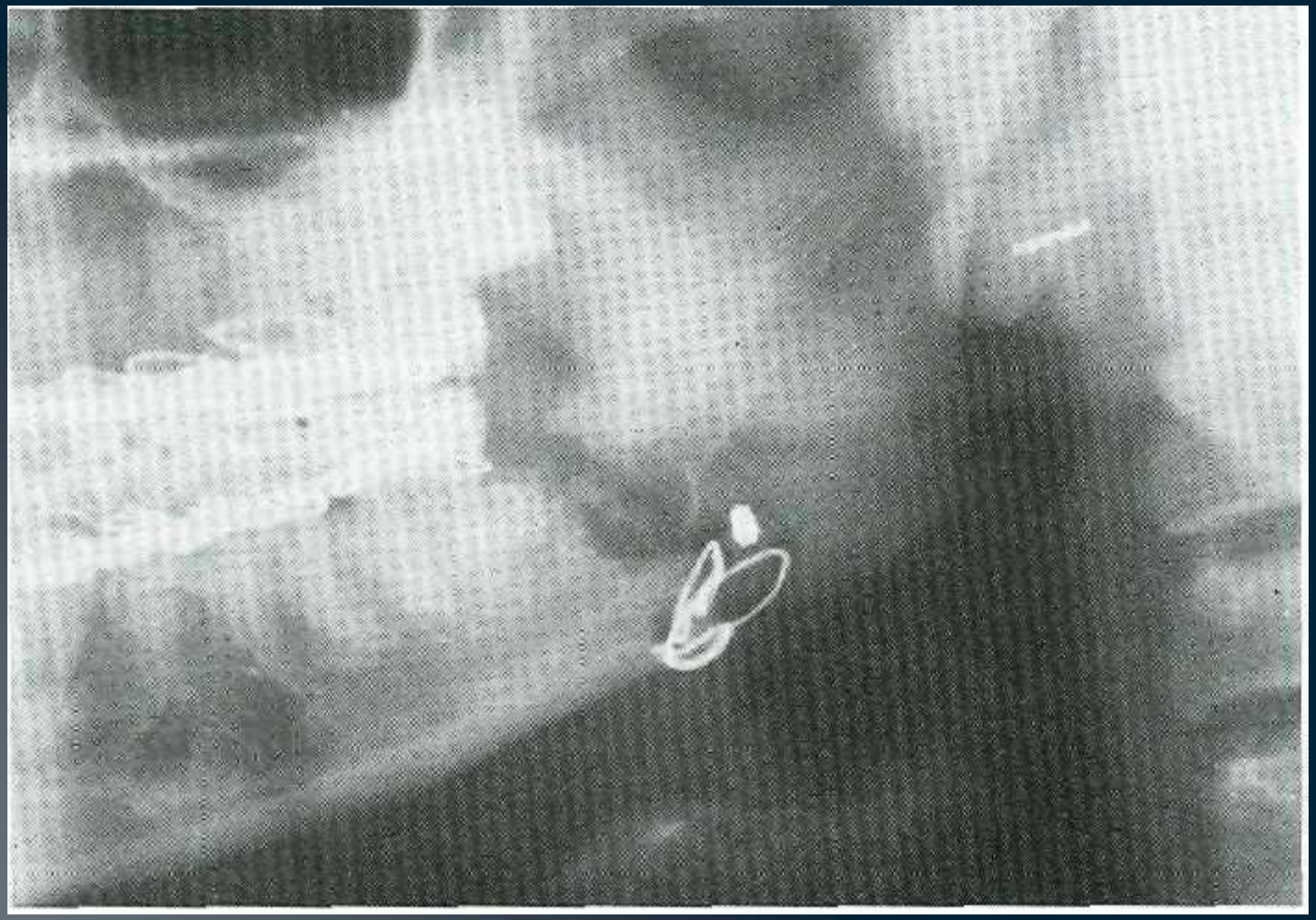

Osteomyelitis occurred in the area of a fracture of the mandible of a patient who was poorly nourished and abused ethanol. The sequestra is surrounded by the radiolucency.

Chronic osteomyelitis requires not only aggressive antibiotic therapy but also aggressive surgical therapy.

Because of the severe compromise in the blood supply to the area of osteomyelitis, the patient is usually admitted to the hospital and given high-dose IV antibiotics to control the initial symptoms. Clindamycin is the drug of choice. An effort should be made to obtain culture material at the time of surgery so that the selection of an antibiotic can be based on the specific microbiology of the infection.

Therapy for both acute and chronic osteomyelitis, most authorities agree, should ensure that antibiotics are continued for a much longer time than is usual for odon-togenic infections. For mild acute osteomyelitis that has responded well, antibiotics should be continued for at least 4 weeks. For severe chronic osteomyelitis that has been difficult to control, antibiotic administration may continue for up to 6 months.

Osteomyelitis of the mandible is a severe infection that may result in loss of a large portion of the mandible. Therefore a clinician who has the training and experience to handle the problem expeditiously should manage this infection. In addition, it is likely that medical consulta-tion will be required to help correct any underlying com-promise of host defenses.

Схемы секвестрации различных областей нижней челюсти в зависимости от выключения различных питательных сосудов