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УО «Витебский государственный медицинский университет»

ВОСПАЛИТЕЛЬНЫЕ ЗАБОЛЕВАНИЯ ЧЕЛЮСТНО-ЛИЦЕВОЙ ОБЛАСТИ

MAXILLOFACIAL INFECTIONS

учебно-методическое пособие

Рекомендовано учебно-методическим объединением
по высшему медицинскому, фармацевтическому образованию в
качестве учебно-методического пособия для студентов учреждений
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Предназначено для студентов 3, 4, 5 курсов, изучающих дисциплину «Челюстно-лицевая хирургия и хирургическая стоматология» на английском языке, для студентов лечебного факультета, факультета подготовки иностранных граждан, субординаторов, магистрантов, интернов, аспирантов, клинических ординаторов, слушателей факультета повышения квалификации.

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Content

MAXILLOFACIAL INFECTIONS. INTRODUCTION

The majority of infections that manifest in the orofacial region are odontogenic. Of these infections, approximately 70% present as periapical inflammation periodontal abscess following.

The cardinal causes of orofacial infections are non-vital teeth, pericoronitis (due to a semi-impacted mandibular tooth), tooth extractions, periapical granulomas that cannot be treated, and infected cysts. Rarer causes include postoperative trauma, defects due to fracture, salivary gland or lymph node lesions, and infection as a result of local anesthesia.

In order to understand how odontogenic infections are treated, the dentist must be familiar with the terminology concerning infection and the pathophysiology of inflammation, which are described below:

Inoculation is characterized by the entry of pathogenic microbes into the body without disease occurring.

An *infection* involves the proliferation of microbes resulting in triggering of the defense mechanism, a process manifesting as inflammation.

Inflammation is the localized reaction of vascular and connective tissue of the body to an irritant, resulting in the development of an exudate rich in proteins and cells. This reaction is protective and aims at limiting or eliminating the irritant with various procedures while the mechanism of tissue repair is triggered. Depending on the duration and severity, inflammation is distinguished as acute, subacute or chronic.

Acute Inflammation is characterized by rapid progression and it's associated with typical signs and symptoms. If it does not regress completely, it may become subacute or chronic.

Subacute Inflammation. This is considered a transition phase between acute and chronic inflammation.

Chronic Inflammation.This procedure presents a prolonged time frame with slight clinical symptoms and is characterized mainly by the development of connective tissue.

Inflammation may be caused by, among other things, microbes, physical and chemical factors, heat, and irradiation.

Regardless of the type of irritant and the location of the defect, the manifestation of inflammation is typical and is characterized by the following clinical signs and symptoms: rubor (redness), calor (heat), tumor (swelling or edema), dolor (pain), and functio laesa (loss of function).

The natural progression of inflammation is distinguished into various phases. Initially vascular reactions with exudate are observed (serous phase), and then the cellular factors are triggered (exudative or cellular

phase). The inflammation finally resolves and the destroyed tissues are repaired. On the other hand, chronic inflammation is characterized by factors of reparation and healing. Therefore, while acute inflammation is exudative, chronic inflammation is productive (exudative and reparative).

Etiology and pathogenesis of purulent-inflammatory diseases of the maxillofacial region

The cardinal causes of orofacial infections are non-vital teeth, pericoronitis (due to a semi-impacted mandibular tooth), tooth extractions, periapical granulomas that cannot be treated, and infected cysts. Rarer causes include postoperative trauma, defects due to fracture, salivary gland or lymph node lesions, and infection as a result of local anesthesia.

The qualitative and quantitative composition of the microflora plays a significant role in the development of odontogenic inflammation. Odontogenic inflammatory processes occur as a result of autoinfection with mixed flora, where staphylococci, other cocci and, often, putrefactive bacteria are most common. Great importance is attached to the role of pathogenic staphylococci with special resistance to antibiotics and increased resistance to them. Microbial associations are increasingly isolated, and such cases represent a particularly difficult task for treatment. Of great importance in the pathogenesis of odontogenic inflammatory processes is the general state of the organism, the state of its adaptive and compensatory mechanisms. Odontogenic disease can occur in different ways in different people. Three types of the patient's reaction to the inflammatory process should be distinguished: normergic, hyperergic and hypoergic. This suggests different manifestations of local manifestations of inflammation and the general reaction of the body. The development and flow of odontogenic inflammatory processes is determined by the complex process of interaction of the protective forces of the organism and pyogenic microorganisms. Sensitivity is closely related to the reactivity of the organism and the microbial factor. It is important here as an increased sensitivity of the organism to microbial pathogens in odontogenic foci, as well as other manifestations of sensitization, allergizing the body. These conditions are most favorable for the development of odontogenic inflammation, and its nature directly depends on these factors. In the development of odontogenic inflammatory processes, the anatomical and topographic relationships between the entrance gates-the odontogenic focus and surrounding tissues, bone, periosteum, soft tissues, are important.

Phases of inflammation:

1. Serous Phase. This is a phase that lasts approximately 36 h, and is characterized by local inflammatory edema, hyperemia or redness with elevated temperature, and pain. Serous exudate is observed at this stage, which contains proteins and rarely polymorphonuclear leukocytes.

2. Cellular Phase. This is the progression of the serous phase. It is characterized by massive accumulation of polymorphonuclear leukocytes, especially neutrophil granulocytes, leading to pus formation. If pus forms in a newly developed cavity, it is called an abscess. If it develops in a cavity that already exists, e.g., the maxillary sinus, it is called an empyema.

3. Reparative Phase.During inflammation, the reparative phenomena begin almost immediately after inoculation. With the reparative mechanism of inflammation, the products of the acute inflammatory reaction are removed and reparation of the destroyed tissues follows. Repair is achieved with development of granulation tissue, which is converted to fibrous connective tissue, whose development ensures the return of the region to normal.

Anatomico-topographic features of the maxillofacial region, causing the development and spread of odontogenic infection

1. Blood supply:

– abundant blood supply contributes to good regeneration, but at the same time, an abundant vascularization leads to active absorption of toxins into the blood and intoxication of the body;

– the presence of venous plexuses, as well as veins without valves, promotes the rapid spread of the inflammatory process along the vascular system. Penetration of pus in the venous system of the face can lead to the development of phlebitis, and then – thrombophlebitis. This process through v.ophtatmica (the ocular vein) ascending way can spread to the veins of the middle cerebral cortex, and then to the cavernous sinus. Directly the facial vein is connected with v. angularis nasi. The intimate connection of the venous plexuses of this region with the pterygoid splenght promotes the penetration of the process to the base of the skull through the round hole.

2. Innervation

Well expressed innervation causes a significant soreness of purulent-inflammatory diseases. Especially painful are the processes located in the zone of localization of the sensory nerves (the region of the canine fossa).

The incisions during drainage must be carried out parallel to the course of the branches of the facial nerve, which is motor, goes out on the face anterior to the tragus of the ear, is located radially and forms a large «crow's foot».

3. Lymphatic supply

There are submaxillary, sub-chin, parotid, maxillary, buccal, cervical, supraclavicular lymph nodes. Significant swelling on the face is associated with a good development of the lymphatic network, lymphostasis is possible (especially with traumas of the infraorbital area).

4. The structure of bone tissue

Osteomyelitis of the upper jaw is more often limited, since a thin cortical plate with a large number of usurpations (pores) is not a serious obstacle to the penetration of exudate from the intraosseous focus of inflammation. This is also facilitated by a small amount of bone marrow in the chelas.

Dense and comparatively thick cortical plates in the region of the lower jaw complicate the breakdown of pus on the outer surface of the bone. In the zone of the focus of osteomyelitis intraosseous pressure is 40-50 times higher than its normal indices. A large number of bone marrow also contributes to a diffuse lesion of the lower jaw in the inflammatory process.

5. Distribution of pus through intermuscular and interfascial cellular spaces.

6.The presence of the maxillary sinus and the proximity of vital organs – sight, smell, hearing, respiratory and digestive system, as well as the brain – promote the spread of the purulent-inflammatory process and their defeat.

In patients with phlegmons of the floor of the mouth, deep cell spaces and neck, respiratory disorders are identified, which are intensified during anesthesia. Carrying out anesthesia in such patients seems most difficult. Intubation is difficult due to the restriction of opening the mouth, edema in the upper respiratory tract, congestion of pus and mucus. Conduction of intravenous anesthesia is dangerous due to muscle relaxation and compression of the inflammatory infiltration of the lumen of the respiratory tube, which can lead to asphyxia. In such cases, intubation of the patient through the tracheostomy, with the help of a bronchoscope or nasopharyngeal intubation is possible.

Spread of infection Inside Tissues

From the site of the initial lesion, inflammation may spread in three ways:

1. By continuity through tissue spaces and planes.
2. Byway of the lymphatic system.
3. By way of blood circulation.

The most common route of spread of inflammation is by continuity through tissue spaces and planes and usually occurs as described below. First of all, pus is formed in the cancellous bone, and spreads in various directions by way of the tissues presenting the least resistance.

Classification of purulent-inflammatory diseases of the maxillofacial region

By localization:

1. Periodontitis – the zone of distribution of the process is limited by periodontal tissues of one tooth.
2. Osteomyelitis is a purulent-necrotic process that develops in the bones and surrounding soft tissues.
3. Dentoalveolar abscess – inflammation of the periosteum.
4. Soft tissues abscess is a limited inflammation of the cellular spase.
5. Phlegmon – diffuse purulent inflammation of the cellular spase.

On the etiology:

1. Odontogenic (the occurrence is associated with the pathology of the teeth: complications of caries, periodontitis, trauma).
2. Nonodontogenic (the occurrence is associated with the penetration of the pathogen through the damaged mucous membrane of the oral cavity, the skin of the head and neck.It is usually preceded by stomatitis, furuncles, erysipelas, trauma.

PERIAPICAL PERIODONTITIS

Periapical periodontitis(also termedapical periodontitis, AP, orperiradicular peridontitis) is an acute or chronic inflammatorylesion around theapex of a tooth root which is caused by bacterial invasion of the pulp of the tooth. The term is derived fromperi- meaning «around», apical referring to the apex of the root (the tip of the root), and -itis meaning a disease characterized by inflammation. Periapical periodontitis can be considered a sequela in the natural history of dental caries (tooth decay),irreversible pulpitisandpulpal necrosis, since it is the likely outcome of untreated dental caries, although not always. Periapical periodontitis may develop into a periapical abscess, where a collection of pus forms at the end of the root, the consequence of spread of infection from the tooth pulp (odontogenic infection), or into aperiapical cyst, where an epithelial lined, fluid filled structure forms.

The type of periapical periodontitis is usually classified according to whether it is an acute process or a chronic process.

Acute periapical periodontitis. Acute periapical periodontitis, also termed acute apical periodontitis, acute periradicular periodontitis, or symptomatic periapical periodontitis.

Chronic periapical periodontitis. Chronic periapical periodontitis, also termed chronic apical periodontitis, chronic periradicular periodontitis, or assymptomatic periapical periodontitis. A periapical granuloma (also termed an apical granuloma or a radicular granuloma) is mass of chronically inflamed granulation tissue that forms at the apex of the root of anonvital (dead) tooth. However, a periapical granuloma does not contain granulomatous inflammation, and therefore is not a true granuloma, but the term *periapical granuloma*is in common use.

ICD-10 classification

K04.4 Acute apical periodontitis pulp origin
K04.5 Chronic apical periodontitis apical granuloma
K04.6 periapical abscess with fistula
K04.60 What is communication (fistula) with maxillary bosom
K04.61 What is communication (fistula) with nasal cavity
K04.62 What is communication (fistula) with oral
K04.63 What is communication (fistula) with skin
K04.69 periapical abscess with fistula unspecified
K04.7 periapical abscess without fistula
K04.8 root cyst
K04.9 Other and unspecified diseases of pulp and tissue periapical

Epidemiology. Periapical periodontitis of some form is a very common condition. The prevalence of periapical periodonitis is generally reported to vary according to age group, e.g. 33% in those aged 20-30, 40% in 30- to 40-year-olds, 48% in 40- to 50-year-olds, 57% in 50- to 60-year-olds and 62% in those over the age of 60. Most eidemiologic data has been generated in European countries, especially Scandinavia. Millions of root canal treatments are carried out in the United States each year, although the total number of root canal treatments is an imperfect indicator of the prevalence of periapical periodontitis, since not always is it performed due to the presence of periapacial periodontitis, and not all cases of asymptomatic periodontitis will be treated in this manner, either due to lack of patient attendance or watchful waiting.

Pathogenesis of periodontitis

The major changes in the case of periodontal disease include various forms of inflammatory manifestations of alteration, exudation and proliferation. No matter how etiological factor caused by a inflammatory response – the action of infectious and toxic agents, as a result of traumatic or chemical injury, inflammation begins of alteration cells or tissues periodontium.

In the area of damage accumulate inflamed mediators (histamine, serotonin, acetylcholine, etc.) as well as tissue proteolytic enzymes along with other alternative change begins we automatically trigger inflammatory reactions. As a result, there are changes in the vascular wall, circulation disorders, is the output of plasma proteins and fluid forms. Along with the changes that come with alteration and exudation, early inflammatory response showing proliferative processes inherent in the acute, subacute and chronic inflammation begins Lenny. The process ends with the formation of proliferation granulator tissue, which further transformed into fibrous, scaring tissue.

The nature of inflammation in periodontal depends on the intensity and duration of the etiological factor, reflect the characteristics destruction area and the state of the defenses.

Inflammatory processes in the degree and nature of the pathological manifestations of the clinical course can be divided into *two main groups*:

– acute inflammation – characterized advantage of alternative-exudative changes, less prolonged and intensive course, a more severe clinical picture,

– chronic inflammation – characterized advantage liferatively-regenerative processes, longer course and less severe clinical symptoms.

Pathogenesis of acute periodontitis

If high levels of defenses and low intensity pathogenic etiological factor arises acure serous periodontitis. This form can be developed by root canal treatment of necrotic pulp tissue when the canal through apical hole gets infectious with severe virulence or when the effect of trauma or chemical agent is negligible. Acute serous inflammation Periodontal early characterized hyperemia. There have been filling vessels and slow down of blood flow. Leukocytes are shifted to the vascular wall and reached stasis. Because vascular wall begin to penetrate the liquid part of blood proteins that are able colloid, and small of neutrophilic leukocytes, lymphocytes and monocytes. Fluid loosens the connective tissue elements and collagen fibrils periodontium. At this stage of the inflammatory process extends to the bone, which is very responsive thanks to its close association with periodontal. In the adjacent periodontal bone marrow-filled intervals spongy substance possible extension blood vessels and stagnation.

Depending on the reactive capacity of the organism and timely therapeutic intervention process may cease or intensify and move in purulent inflammation.

Acute suppurative periodontitis develops if the infection that got into Periodontal has high virulence, damaged factor very aggressive and protective reactions of the organism at a low level. Due to the high penetration of blood vessels due to activation such enzymes as leykotoksyn, trypsin, penetrate through the vascular wall in a large number of white blood cells, lymphocytes and monocytes. Neutrophilic leukocytes phage bacteria and then die. Their destruction accompanied by the release of enzymes – protease, cathepsin, chymotrypsin, alkaline phosphatase and others, and also spe There is a breakdown of tissue with the formation of pus. In hyperacidosis tissue in the area of inflammation are active lymphocytes monocytes blood, and settled macrophagocytes. Macrophages clean the area of inflammation from dead cells and large unorganizedcific antigens that are needed for the next formation Rennie antibodies.

Acute periodontitis

Acute serous periodontitis (periodontitis acute serosa).

In clinical practice is most common periodontitis, which occurs under the influence of infection and usually develops as a complication of inflammation of the pulp or because of mistakes that were made during endodontic therapy.

Symptoms. Complaints patient so characteristic that often their is sufficient to establish a virtually error-free diagnosis. Initially, the patient

feels heaviness and tension in the tooth, which was like the big, longer than others. Gradually there is quite a lot of pain spontaneous nature. The pain is constant, localized, not radiating, worse at night and barely suppressed conventional analgesics. Since the process is constantly evolving, pain intensity grows.

Also, can occur characteristic provoked pain. All that can increase blood flow in the area of the tooth and change its mobility, provokes attacks of pain. Yes, there is pain during eating. In the initial stage, however, passive, slow, long-term pressing reduces the pain that is associated with the outflow of fluid from periodontal reducing congestion and compression of nerve endings. Therefore, clutching a tooth in the alveoli, patients temporarily improve their condition. Pain when touching the tooth can occur under the influence of heat, if periodontitis is a complication of gangrene of the pulp with a closed cavity of a tooth. The temperature difference can cause pain if the change is sudden. In the case of a gradual increase in temperature and prolonged exposure to heat achieved calming effect due to sustained vasodilation, which promotes blood flow areas of inflammation.

Objective**.** Sick tooth may be intact, that does not exclude injury (such as when you use orthodontic appliances). Often, however, it is caries, devital, with an open cavity of a tooth filled or a great seal. Enamel loses its characteristic luster, is gray. It is clear in the area of apex often hiperemic and swollen, sometimes existing congestion and adjacent areas gums. Vertical percussion painful. The reason for such a reaction is to increase the sensitivity of nerve receptors in the area of periapical inflammation.

Palpation of the gums in the area of the top teeth (especially front) painful, because of the proximity of the root to the periosteum. Regional lymph nodes are enlarged, become painful during palpation. Depending on the top «that limphatic nodes swollen in diagnostically difficult cases can be differentiated tooth. Yes, periodontitis lower front teeth accompanied by inflammation limphatic Submental nodes periodontitis upper incisors and upper and lower canines and premolars - Front submandybulyar lymph node corresponding side and periodontitis molars of both jaws - middle and rear submandybulyar lymph nodes.

Electrical conductivity – higher than 100 mi-A, except traumatic injury period when kept alive pulp and response to constant current associated with its response. X-ray changes usually are not found only in the later stages of a possible slight expansion pieriodontal slit. Depending on the etiology of the clinical picture of acute serous periodontitis can have its own specifics that should be considered during the differential diagnosis.

In patients with traumatic periodontitis clinical picture depends largely on the state of the pulp is exposed to 'severe injury. If the pulp is alive, the course of the process becomes lighter forms, weather favorable treatment. In the case of septic necrosis of the pulp always joins periodontal infection and there is clinical picture of infectious periodontitis. Often inflammation may be caused by medications, or used in the treatment of pulpitis (eg arsenious paste trykrezol (formalin) or filling materials that have a necrotizing effect on periodontal tissue. Periodontitis For this group, the typical steady nature of the flow and resistance to therapy.

In practice often become allergic meet periodontitis, which is associated with sensitization of patients to drugs used. Serous overall process in this form of periodontitis allergy accompany such as skin rash, swelling of the face and mucous membranes of the mouth, throat irritation with characteristic cough etc. that contribute clarifying the nature of the disease. Revealing a history of exposure to allergic reactions, as well as positive results allergy tests help clarify the diagnosis and identify therapies.

The differential diagnosis of acute serous periodontitis should be conducted with acute diffuse pulpitis. Characteristic for irradiation pulpitis pain, acute onset, remission and intermission in progress sharply distinguish it from periodontitis. Pain in patients with periodontitis is dumber, is not as sharp as with pulpitis. Lymph nodes in patients with pulpitis not affected.

Differential diagnosis between serous and purulent periodontitis based on the severity of the patient and the nature of pain and overall clinical picture. In patients with serous periodontitis pain less pronounced, not as intense, strictly localized. Changes in the mucosa in the area of the root apex small, often in the form of mild hyperemia. Tooth barely moving only in the transverse direction. The general condition of the patient does not suffer.

Acute suppurative periodontitis (periodontitis acuta purulenta) usually develops after serous. But often it can begin spontaneously in the case of massive penetration of virulent infections in periodontal and reduced reactivity of the patient. The clinical picture of this fairly typical periodontitis. In comparison with serous form of his more rapid progress, expressed common manifestations. Founded in periodontal space purulent exudate, which looks out, often breaks out, destroying the periodontal tissue.

Patients complain of spontaneous acute continuous pain pulsating character. At the beginning of the pain is localized. However, he soon

becomes diffuse, radiating from the teeth of the mandible in the ear, and the top – in the temporal area. Patient always indicates tooth that he feels like «higher» very painful when pressed, contact with antagonists or even if you touch your tongue while talking. Pain aggravated by heat, whereas cold, on the contrary, has a sedative effect. Any physical effort leading to increased pain.

Objective. Sick tooth may be intact, although its color is changed, sometimes significantly carious defect or seal. Pulp cavity in most cases closed, but may be open. elektrosensitivity – 120-150 mA, which determines necrosis of the pulp. In canals during sensing there gangrenous decay, often under pressure turns manure. Horizontal and vertical percussion tooth is very painful. Mobility is particularly significant, if manure reaches the circular connections and looking out in the area of gingival pockets. In this case, the tooth as if floating in the accumulation of manure

Tooth allegedly grew not only a subjective feeling sick, but determined during the review, because it really is somewhat supplanted with alveoli accumulated indepth inflammatory exudate. Mucosa in the area of the top bloodshots and edematous. Transitional fold smoothed due to the accumulation of inflammatory infiltrate, very painful during palpation. Depending upon the stage of suppurative periodontitis can be detected by palpation extremely painful hardening of the periosteum in the case of formation of subperiosteal abscess.

In the case of case of protruding abscess during palpation reveal not only the pain but also the phenomenon of fluctuations arise collateral changes, such as edema of the soft tissues of the face, the size of which does not always correspond to the severity of injury. Swelling can lead to significant asymmetry and deformation face, especially in pasty tissues. If collateral edema should always perform a differential diagnosis of cellulitis, phlegmon but for the expressed pain and tension and elegance skin. Promotion purulent exudate and abscess localization depend on the location of the root, which is the source of infection, and anatomic and histologic features section of the jaw.

In some cases, pus, which met in periodontal could spill across the canal tooth. This is the most favorable option evacuation of pus, but it is possible only when the canal is open and passable. Often in the case of lesions of the lower molars manure flows through the marginal gingival pocket that after melting circular links periodontium. This path is unfavorable because the cortical plate subsequently melted and formed bone pocket.

Chronic periodontitis

Chronic periodontitis is a disease that has a variety of clinical and radiological picture and the most common among people with periodontal pathology.

If a patient with acute periodontitis causes pain without hesitation to seek dental care, then chronic periodontitis usually does not cause subjective sensations. Often he found by chance on radiographs when the patient is not even aware that he has the disease. Accurate diagnosis can be established only after a thorough clinical and radiological examination.

Chronic fibrotic periodontitis (periodontitis chronica fibrosa). Symptoms. Chronic fibrotic periodontitis is asymptomatic, only occasionally patients experience minor pain during chewing coarse meal. The same may be determined by gangrene of the pulp if cavities filled remnants of food. Diseases found by x-ray. From history establish that before (1-2 years ago), the patient was unwarranted causal or pain and were treated tooth root.

Objective. They exhibit a carious tooth or sealed devitalization. The pain from the effects of thermal stimuli and percussion available. Palpation in the area of apex painless. If fibrotic periodontitis developed after treatment of acute suppurative or chronic granulating periodontitis, it may be outdated scar. Sometimes fibrotic periodontitis may be a patient with intact teeth. In such cases, fibrotic periodontitis arose as a result of chronic injury or traumatic occlusion. Radiologically often exhibit periodontitis expansion slot in the top section in the form of genital cap.

ENDODONTIC SURGERY

During the past 20 years, endodontics has seen a dramatic shift in periradicular surgery and the part it plays in the delivery of endodontic services. Previously it was considered the treatment of choice when nonsurgical treatment had failed or there was the presence of large or intruding periapical lesions, overfilled canals, incomplete apical formation, or destruction of the apical constricture by over instrumentation. Today, a more conservative approach solves many of these problems sans surgery.

Indications for endodontic surgery:

1. Surgical drainage
2. Failed endodontic treatment
    - Irretrievable filling material
    - Irretrievable post
3. Calcification of pulp space
4. Procedural errors

- Instrument breakage
- Non-negotiable ledging
- Root perforation
- Gross overfilling

5. Anatomic variations
   - Root dilacerations
   - Apical root fenestration
6. Biopsy
7. Corrective surgery
   - Root resorptive defects
   - Root caries
   - Hemisection
   - Bicuspidization
8. Replacement surgery
   - Intentional replantation
   - Post-traumatic replantation
   - Osseointegrated implant

Contraindications for endodontic surgery:

1. Patient's medical status.

If the patient reports any major system disorder – cardiovascular, respiratory, digestive, hepatic, renal, neural, immune, or musculoskeletal – a thorough medical history is mandatory and the patient's physician must be consulted. Surgery in the first trimester of pregnancy is also ill advised, and in the third trimester is uncomfortable for the patient. Most miscarriages take place in the first trimester, and the endodontic surgeon may be blamed for the miscarriage. Before any surgery is undertaken, be sure the patient is not on warfarin (Coumadin) or ximelagatran (Exanta), the newest of the anticlotting drugs.

2. Anatomic considerations: nasal floor, maxillary sinus, mandibular canal and its neurovascular bundle, mental foramen and its neurovascular bundle, adequate visual and mechanical access.

3. Limited skill and knowledge of the surgeon. One must know one's limitations. Endodontic surgical procedures are best done by a trained endodontic specialist. The standard of care in dentistry is that practiced by the region's specialists in any given dental discipline

Endodontic surgical procedures

Endodontic surgery is performed to remove causative agents of periradicular pathosis and restore the periodontium to a state of functional health. Classification of endodontic surgical procedures:

1. Surgical drainage
    - Incision and drainage
    - Trephination (fistulization)
2. Periradicular surgery
    - Curettage
    - Biopsy
    - Root-end resection, preparation, and filling (retrofill)
    - Corrective surgery: perforation repair (iatrogenic; resorptive); root resection; hemisection
3. Replacement surgery (extraction/replantation)
    - Endodontic implants
    - Osseointegrated implants

Surgical drainage

Surgical drainage is indicated when purulent and/or hemorrhagic exudate forms within the soft tissue or alveolar bone – an abscess. Pain is reduced and morbidity shortened following the surgical release of pressure and infection. This can be accomplished by either incision and drainage or trephination of the alveolar cortical plate. Incision and Drainage Initially when a periradicular abscess forms, the area appears and feels hard (indurated). This is when the lesion is the most painful. It is seldom productive to drain an abscess at this stage. The patient should be started on antibiotics and the abscess encouraged to «point» with the use of «hot holds» – a half-teaspoon of salt in 250 mL (8 ounces) of hot water, repeatedly held in the area, to speed the inflammatory process. The area becomes fluctuant (soft) once the purulent/hemorrhagic (pus) abscess breaks through the cortical bone. Timing is of the essence! If allowed to continue, the lesion could spread laterally and become full-blown cellulitis. It is at the fluctuant stage that the abscess should be incised and drained.

After nerve block anesthesia has been established, the proper instruments for incision and drainage should be assembled. If complete anesthesia is not achieved, local infiltration with mepivacaine is in order, carefully avoiding the infected area. Failing all else, supplemental freezing of the area with ethyl chloride spray has been recommended. The surgical area should be isolated with 5 × 5 cm (2 × 2 inch) gauze squares, and a horizontal incision with a no. 11 or 12 blade should be made at the lowest dependent base of the fluctuant area. This is immediately followed by aspiration of the purulent material. To ensure total evacuation, a curved hemostat may be inserted through the incision to the base of the abscess and the beaks spread to drain the last of the pus. It has not been found

necessary to insert a drain in the incision. Healing should occur within a week, and, when comfortable for the patient, endodontic treatment may be instituted. Regular orthograde endodontics is the preferred treatment.

Trephination Drainage

Trephination is a limited-use procedure and is fraught with peril. Likely candidates present themselves with apical pain and no swelling, but drainage of the immediate apical region seems necessary. However, apical trephination through the canal should be attempted first. The exact tooth length is calculated and with a fine file, the apical orifice is just perforated. This opening is then enlarged with up to a no. 20 or 25 file in the hope that drainage will occur through the canal. If this does not happen, then a surgical approach may be indicated. Another surgical indication may be the canal with a post present.

Cortical trephination involves making an incision through the mucoperiosteal tissues and then perforating with a no. 6 or 8 round bur through the cortical plate to the inflamed area. Once the cortical plate is perforated, a large reamer can often be used to open through the cancellous bone to the fluid collection. Hopefully, drainage ensues. Perfect radiographs, careful measurements, and tooth alignment in the arch are imperative. The floor of the nose, the maxillary sinus, and the mandibular canal must be avoided.

Periradicular surgery

In the case of a failed nonsurgical endodontic treatment, the first choice of correction is re-treatment by the usual orthograde approach. Periradicular surgery may be the answer to retaining the tooth in the event other factors preclude an approach through the canal, a perfect jacket crown should be preserved, or a well-positioned post is in place.

So surgery should not be considered the first approach in the event a periapical lesion is present. One exception might be the presence of an apical cyst, with the caveat that many so-called granulomas very much resemble a cyst on radiographs. In any event, some cysts continue to grow in spite of the fact a perfect root canal filling has been completed. To be on the safe side, if a cyst is suspected, it should be removed surgically.

Root-end resection

An often-treated but persistent periapical lesion is a perfect example of an indication for root-end resection. Investigation of root ends from cases refractory to healing have shown colonies of bacteria clinging to root surfaces. Their removal, along with Burs for hard tissue removal the root tip, combined with antibiotic therapy is often necessary to bring about

healing. Persistent discomfort following «successful» endodontic treatment is another example of a situation that requires root-end resection or beveling. Instances of root-end perforations, fractures, resorptions, unfilled curvatures or multiple unfilled canals, broken instruments through the apex, and gross overextension of filling material might also call for resection. How does one choose which bur to use in resection and how much root to remove? It appears that a plain fissure bur in a low-speed handpiece not only provides the smoothest resected surface but the least distortion of the previous orthograde gutta-percha filling. Remove as little root as necessary! Most lateral, accessory, and secondary canals lie within the last 2 to 3 mm of the root end, and these are best removed. And, of course, any area damaged by a procedural error should be resected. But remember, the longer the root retained, the more retentive and functional the tooth will be. Most often, however, following root-end resection, a retrofilling is necessary to ensure success.

Whole root amputation

The indications for root amputation are as follows:

• Periodontal bone loss to such an extent periodontal therapy is inadvisable

• Destruction of the root through resorption, caries, or perforation

• Surgically inoperable roots that are calcified or contain separated instruments

• Fracture of one root without the involvement of another

Contraindications are the following:

• Lack of osseous support for remaining roots

• Fused roots

• Remaining roots that are endodontically inoperable

• Lack of patient motivation to perform home care

Most cases requiring root amputation involve huge periodontal lesions that completely denude one root of a multirooted tooth, with the remaining roots well supported. In such a case the root canal treatment is carried out on the roots that are to remain; the unsupported root, which is to be amputated at the crown, should have an enlarged, permanent, internal filling such as an amalgam filling placed in the canal from within the crown. The sectioning through the root and filling should be done after the filling material has set. In some cases a simple tissue flap exposes the root at the furcation and amputation with a fissure bur is quite simple. Sometimes the furcations are exposed and no flap is necessary.

Hemisection of the effected tooth allows the preservation of tooth structure, alveolar bone and cost savings (time and money) over other treatment options. The term hemi-section refers to the sectioning of a molar tooth with the removal of an unrestorable root which maybe affected by periodontal, endodontic, structural (cracked roots), or caries. Careful case selection determines the long term success of the procedure.

Indications for hemisection include:

1. The tooth is affected by caries, vertical root fracture, periodontal disease or iatrogenic root perforation where only one root of a multirooted tooth is affected.
2. The surviving root is accessible and treatable endodontically.
3. The surviving root is structurally capable of supporting a dowel and core restoration.
4. The surviving root is aligned so as to provide proper draw for the resulting fixed prosthetic restoration.
5. The root morphology allows for surgical access and proper periodontal maintenance of the final restoration.

Contraindications to using a tooth root as an abutment can include:

1. Poorly shaped roots or fused roots.
2. Poor endodontic candidates or inoperable endodontic roots.
3. Patient unwilling to undergo surgical and endodontic treatments and undertake the care or the resulting restoration.

WISDOM TEETH INFECTION

The management of impacted teeth is probably the most common problem in oral and maxillofacial surgery worldwide. Teeth that fail to erupt are usually the teeth which erupt last in a certain region where there is not enough space or because of crowding of teeth. The most common teeth which are impacted are mandibular and maxillary third molars followed by maxillary canines and mandibular premolars. An impacted tooth is one which is prevented from completely erupting into a normal functional position. This may be due to lack of space, obstruction by another tooth, or an abnormal eruption path. The tooth may be soft tissue impacted or hard tissue impacted and may be unerupted or partially erupted. Impaction per itself is not an indication for removal, it is only a description of the position of the tooth.

Since surgery is sometimes associated with complications, one must always balance the risk of complications caused by leaving the impacted tooth in place against the risk for complications and discomfort associated with surgery. The main complications related to third molar surgery are alveolar osteitis (fibrinolytic alveolitis, dry socket) infection and temporary or permanent nerve injury. Nerve injuries often cause temporary and permanent neuropathic pain or altered sensation in and around the mouth, resulting in significant difficulties for the patient with regard to eating, drinking, speaking, kissing, shaving, applying makeup etc. A nerve injury is one of the main causes of litigation in dentistry. Prevention of these injuries must therefore be taken into consideration before surgical removal of impacted teeth. The patient should always be advised of the risk of inferior alveolar nerve injury. It is probably easier for a patient to accept a permanent complication or disability of an intervention if it was properly based on a therapeutic indication than if surgery was performed on prevention and prophylaxis. Patient information about risks and complications is very important and the patient should always be part of the decision.

Medical History. A detailed medical history is necessary because useful information may be found concerning the general health of the patient to be operated on. This information determines the preoperative preparation of the patient, as well as the postoperative care instructions.

Clinical Examination. During the intraoral clinical examination, the degree of difficulty of access to the tooth is determined, especially concerning impacted third molars.

When the patient cannot open his or her mouth, because of trismus that is mainly due to inflammation, the trismus is treated first, and extraction of the third molar is performed at a later date.

In certain cases of impacted teeth, especially canines, buccal or palatal protuberance may be observed during palpation or even inspection, which suggests that the impacted tooth is located underneath. Also, the adjacent teeth are examined and inspected (extensive caries, large amalgam restorations, prosthetic appliance, etc.) to ensure their integrity during manipulations with various instruments during the extraction procedure.

*Radiographic Examination***.** The radiographic examination provides us with all the necessary information to program and correctly plan the surgical removal of impacted teeth. This information includes: position and type of impaction, relationship of impacted tooth to adjacent teeth, size and shape of impacted tooth, depth of impaction in bone, density of bone surrounding impacted tooth, and the relationship of the impacted tooth to various anatomic structures, such as the mandibular canal, mental foramen, and the maxillary sinus. These aforementioned data may also be provided by periapical radiographs and panoramic radiographs, as well as occlusal radiographs.

*Indications for Extraction***.** Specialists have divergent points of view concerning the necessity to extract impacted teeth. Certain people suggest that the removal of impacted teeth is necessary as soon as their presence is confirmed, which is usually by chance. They even believe that it must be done as soon as possible, as long as there is no possibility that the impacted tooth may be brought into alignment in the dental arch using a combination of orthodontic and surgical techniques.

On the other hand, others suggest that the preventive removal of asymptomatic impacted teeth, besides subjecting the patient to undue discomfort, entails the risk of causing serious local complications (e.g., nerve damage, displacement of the tooth into the maxillary sinus, fracture of the maxillary tuberosity, loss of support of adjacent teeth, etc.). As far as impacted teeth that have already caused problems are concerned, everyone agrees that they should be removed, regardless of the degree of difficulty of the surgical procedure. The most common of these problems are now given.

There should always be an appropriate indication for third molar removal. Indications can be local or for medical reasons.

Indications for removal of third molar teeth:

– Pain, specifc to the third molars (myofacial pain is not an indication).
– Pericoronitis or infection.
– Advanced caries.
– Periodontal disease.
– Periapical pathology.
– Disease of follicle, including cyst or tumour.
– Fractured tooth.
– Resorption of adjacent teeth.
– As part of an orthodontic or orthognathic treatment plan.
– Mandibular angle fracture.
– Prophylactic removal in the 'at risk' patient (e.g. bacterial endocarditis).
– As an aid to denture provision.

*Radiological assessment***.**Radiological examination is a complement to the clinical examination and is necessary in order to make decisions related to the procedure. A radiological evaluation will give information about anatomy of the impacted tooth, the region and the relation of structures. Usually a pair of periapical radiographs taken in different projections is enough and can be supplemented by a panoramic radiograph when more than one third molar requires assessment. If the initial radiographs suggest a close relationship between the roots of the lower third molar and the inferior alveolar nerve (IAN) canal, cone-beam computer tomography (CBCT) scanning can be used to give more detailed information of the anatomy in the region.

The following local factors are important to take into consideration when planning third molar surgery. The depth of application and the point of elevation will dictate the Local therapeutic indications

• Recurrent or severe pericoronitis;
• Periodontal disease with a pocket depth of 5mm or more distal to the second molar;
• non-restorable caries in the third molar;
• Resorption of the third molar or adjacent tooth;
• Caries in the second molar where the third molar removal would render restoration possible or more simple;
• Apical periodontitis;
• Cysts or tumors associated with the third molar (or adjacent tooth);

• When required prior to orthognatic surgery;
• Removal of third molar in a fracture line;
• When a third molar may be considered for autogenous transplantation.

Medical conditions that require a serious consideration of prophylactic third molar removal Prior to:

• radiation therapy for head and neck malignancies; • organ transplantation;
• chemotherapy;
• bisphosphonate therapy.

Contraindications for third molar removal:

– third molar buds in young people should not be enucleated;
– asymptomatic pathology free third molars totally covered by bone should not be removed;
– routine removal of pathology free third molars totally or partially covered by soft tissue is not recommended but specific medical and local conditions may prove a prophylactic approach appropriate;
– third molar surgery is contraindicated in patients whose medical history or conditions expose the patient to an unacceptable risk to their health.

The patient must be competent and understand the risks and benefits). The benefits are, fairly obviously, the removal of symptoms any (more difficult to convince people of) prevention of further disease.

All patients having third molars (particularly lower) removed will have:

- Some pain or discomfort.
- Swelling (widely variable), possibly with bruising.
- Stillness of the jaws that makes eating difficult.

These symptoms last for about 7 days and gradually get better; if not, then the usual problem is wound infection, which can occur in 5% to 10% of patients even if prophylactic antibiotics have been used. Very rarely iatrogenic fracture of the mandible is the cause of worsening pain. From 5% to 10% of patients experience temporary altered sensation in the lingual or inferior alveolar nerve distribution. In 0.5% to 1% of cases this is permanent.

Patients should take 2 to 5 days off work in most instances.

Infection

An uncommon postsurgical complication related to the removal of impacted third molars is infection. The incidence of infection following the removal of third molars is very low, ranging from 1.7 to 2.7%. Infection after removal of mandibular third molars is almost always a minor complication. About 50% of infections are localized subperiosteal abscess-type infections, which occur 2 to 4 weeks after a previously uneventful postoperative course. These are usually attributed to debris that is left under the mucoperiosteal flap and are easily treated by surgical debridement and drainage. Of the remaining 50%, few postoperative infections are significant enough to warrant surgery, antibiotics, and hospitalization. Infections occur in the first postoperative week after third molar surgery approximately 0.5 to 1% of the time. This is an acceptable infection rate and would not be decreased with the administration of prophylactic antibiotics.

Pericoronitis

Pericoronitis is defined as inflammation in the soft tissues surrounding the crown of a partially erupted tooth. It generally does not arise in teeth that erupt normally; usually, it is seen in teeth that erupt very slowly or become impacted, and it most commonly affects the lower third molar. Once the follicle of the tooth communicates with the oral cavity, it is thought that bacterial ingress into the follicular space initiates the infection. Several studies have shown that the microflora of pericoronitis are predominantly anaerobic.

It is generally agreed that this process is potentiated by food debris accumulating in the vicinity of the operculum and occlusal trauma of the pericoronal tissues by the opposing tooth. Pregnancy and fatigue are associated with an increased occurrence of pericoronitis. The highest incidence of pericoronitis was found in the 20-29 year age group (81%).

Clinically, pericoronitis can be acute or chronic.

Acute symptoms usually last 3 to 4 days and can include:

- severe pain that can cause loss of sleep
- swelling on the affected side of the face
- discharge of pus
- pain when swallowing
- swollen lymph nodes under the chin
- fever

The acute form is characterised by severe pain, often referred to adjacent areas, causing loss of sleep, swelling of the pericoronal tissues, discharge of pus, trismus, regional lymphadenopathy, pain on swallowing, pyrexia, and in some cases spread of the infection to adjacent tissue spaces.

The acute form tended to appear in cases of moderate or poor oral hygiene, while the chronic type was associated with good or moderate hygiene.

Chronic symptoms include:

- dull pain
- mild discomfort
- bad taste in the mouth
- swollen gum in the affected area

Chronic symptoms often only last for 1 to 2 days but keep recurring over a period of months. Patients with chronic pericoronitis complain of a dull pain or mild discomfort lasting a day or two, with remission lasting many months. They may also complain of a bad taste.

Treatment:

- managing or alleviating the pain near the molar
- removing the flap covering the tooth
- removing the tooth

For patients presenting with localised pain and swelling involving the pericoronal tissues, and in the absence of regional and systemic symptoms, it is recommended that local measures only are used. These include debridement of plaque and food debris, drainage of pus, irrigation with sterile saline, chlorhexidine or hydrogen peroxide, and elimination of occlusal trauma. In the past the use of caustic agents such as chromic acid, phenol liquefactum, trichloroacetic acid or Howe's ammoniacal solution was advocated to control pain by placing a small amount on a cotton pledget under the operculum. The resultant chemical cauterisation of the pain nerve endings in the superficial tissues gave rapid pain relief; however, the use of these toxic chemicals in the oral cavity is no longer encouraged. Ozone has been put forward as a local antimicrobial that might be a useful adjunct in the treatment of pericoronitis; however, there is no research available to show its efficacy as yet. In addition to local pain and swelling, if the patient is exhibiting regional or systemic signs and symptoms, antimicrobial therapy is recommended; however, it should be emphasised that it is as an adjunct rather than a first-line treatment. Systemic symptoms include pyrexia, tachycardia and hypotension. The

antibiotic of choice is either metronidazole 400mg three times a day for five days or phenoxymethylpenicillin 500mg four times a day for five days. The two can be used in combination for severe infections. For patients who are allergic to penicillin, erythromycin 500mg four times a day for five days is suitable. These are all active against anaerobic bacteria, which are the predominant cultivable microflora found in pericoronitis and are the first-line antibiotics of choice. Once the acute phase of this condition has passed, operculectomy has been used as a preventive measure; however, there is no research to support or condemn this mode of treatment.

Alveolar Osteitis

The incidence of alveolar osteitis or dry socket following the removal of impacted mandibular third molars varies between 3 and 25%. Most of the variation is most likely a result of the definition of the syndrome.When dry socketis defined in terms of pain that requires the patient to return to the surgeon's office, the incidence is probably in the range of 20 to 25%.

The pathogenesis of alveolar osteitis has not been clearly defined,but the condition is most likely the result of lysis of a fully formed blood clot before the clot is replaced with granulation tissue.This fibrinolysis occurs during the third and fourth days and results in symptoms of pain and malodor after the third day or so following extraction. The source of the fibrinolytic agents may be tissue, saliva, or bacteria. The role of bacteria in this process can be confirmed empirically based on the fact that systemic and topical antibiotic prophylaxis reduces the incidence of dry socket by approximately 50 to 75%. The periodontal ligament may also play a role in the development of alveolar osteitis. The incidence of dry socket seems to be higher in patients who smoke and infemale patients who take oral contraceptives. Its occurrence can be reduced by several techniques, most of which are aimed at reducing the bacterial contamination of the surgical site. Presurgical irrigation with antimicrobial agents such as chlorhexidine reduces the incidence of dry socket by up to 50%.

Copious irrigation of the surgical site with large volumes of saline is also effective in reducing dry socket. Topical placement of small amounts of antibiotics such as tetracycline or lincomycin may also decrease the incidence of alveolar osteitis. The goal of treatment of dry socket is to relieve the patient's pain during the delayed healing process. This is usually accomplished by irrigation of the involved socket, gentle mechanical débridement, and placement of an obtundent dressing, which usually contains eugenol.The dressing may need to be changed on a daily basis for several days and then less frequently after that. The pain syndrome usually

resolves within 3 to 5 days, although it may take as long as 10 to 14 days in some patients. There is some evidence that topical antibiotics such as metronidazole may hasten resolution of the dry socket.

In summary, alveolar osteitis is a disturbance in healing that occurs after the formation of a mature blood clot but before the blood clot is replaced with granulation tissue. The primary etiology appears to be one of excess fibrinolysis, with bacteria playing an important but yet ill-defined role.Antimicrobial agents delivered by perioperative mouthrinses,topically placed in the socket,or administered systemically all help to reduce the incidence of dry socket. Mechanical débridement and copious saline irrigation of the surgical wound also are effective in reducing the incidence of dry socket. A rational approach may be to provide preoperative chlorhexidine rinses for approximately 1 week before surgery, irrigate the wound thoroughly with normal saline at the conclusion of surgery, place a small square of gelatin sponge saturated with tetracycline in the socket, and continue chlorhexidine rinses for 1 additional week.This combination approach should substantially reduce the incidence of dry socket.

Infected haematoma

An infected haematoma is a more significant problem than dry socket, mainly because the mass of haematoma can rapidly liquefy and become pus, which will create a large abscess. Try to avoid this by careful haemostasis before closing the wound (i.e. if it is continuing to bleed, do not just ignore it; identify the bleeding point and control before you close). If a patient has a large, tense swelling, start antibiotics immediately. Common regimens are amoxycillin 250 mg three times daily or ampicillin 250 mg four times daily and metronidazole 400 mg three times daily. Either clarithromycin or clindamycin is given to penicillin-allergic patients.

Osteomyelitis

The incidence of osteomyelitis as a result of third molar extraction is not reported in the literature however it is a known complication of infection, fracture, and/or extraction in medically compromised patients. Osteomyelitis is an inflammation of the bone marrow and is most common in the mandible due to its dependence on blood supply from the inferior alveolar artery and poorly vascularized thick cortical bone. Because the maxilla has a rich vascular supply from multiple vessels it is less likely to develop osteomyelitis. The presence of bacteria within the marrow space leads to inflammation and edema with subsequent compression of blood vessels and a decrease in blood supply. This decrease in blood flow results in ischemia, bone necrosis, and proliferation of bacteria. Purulence and

bacteria can spread within the marrow via Haversian and Volkmann's canals and extend into cortical bone. Once the cortical bone and periosteum are involved, the blood supply is further compromised and perforation of soft tissues can occur resulting in fistula formation. Predisposing factors in the development of osteomyelitis involve suppression of host defenses in some form. Diabetes, alcoholism, autoimmune disease, radiation therapy, chemotherapy, steroid use, osteopetrosis, myeloproliferative diseases, and malnutrition can contribute to the development of osteomyelitis.

Patients with osteomyelitis will often present with complaints of a dull and deep pain, swelling and erythema of overlying tissues, paresthesia of the inferior alveolar nerve, trismus, adenopathy, fistula, fever, and malaise.

In patients with chronic osteomyelitis, signs of acute infection such as fever are often not present; however, fistulas, both intra- and extraorally, are more common. Radiographs typically demonstrate a «moth-eaten» appearance of bony sequestrum. CT scanning can assist in the demarcation of lesion extent although it should be noted that 30-50% demineralization of bone is necessary before radiographic changes.

In chronic osteomyelitis there may be radiopacity due to an osteitis-type reaction and proliferation of bone. A laboratory workup will demonstrate leukocytosis in acute forms, elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CPR). Further laboratory evaluation of ESR and CRP levels during treatment can assist in assessment of healing. Culture specimens will often reveal bacteria traditionally responsible for odontogenic infections such as Bacteroides, Peptostreptococci, Fusobacterium, and Streptococci. Occasionally, less common odontogenic bacteria are present. These include Lactobacillus, Eubacterium, Klebsiella, Acinetobacter,and Pseudomonas aeruginosa. Osteomyelitis of the jaws is different from osteomyelitis of other bones in that Staphylococci are not the predominant bacteria.

The treatment of osteomyelitis is combination of surgical and medical management. Treatment of systemic diseases must be considered along with medical consultation when appropriate. Empiric antibiotics should be administered while awaiting final culture results. Penicillin/metronidazole or clindamycin are excellent first-line antibiotics. In chronic cases, sequestrectomy, decortication, and saucerization are necessary and extend to vital, bleeding bone. Removal of the cortex with placement of periosteum directly on the marrow space assists with blood flow. After aggressive debridement, that may lead to further weakening of the mandible, fixation may need to be employed to prevent fracture or for stabilization of a known fracture. External fixation, rigid internal fixation,

or intermaxillary fixation may be used with the fixation type dependent on the surgeon's preference and degree of success of surgical debridement. Other methods of treatment have been proposed such as local antibiotic administration with both resorbable and nonresorbable carriers and hyperbaric oxygen. Polymethylmethacrylate beads impregnated with gentamycin have been discussed in the orthopedic literature; however, results can be disappointing due to inadequate local release and subinhibitory antibiotic levels.

Also, a second surgery is necessary to remove the beads. Hyperbaric oxygen has not been demonstrated to have a significant effect on outcome based on the limited available literature.

ACUTE DENTOALVEOLAR ABSCESS

A common dental emergency facing the dentist is a patient with an acute alveolar abscess.

There are a number of possible conditions that may lead to an abscess, including:

- periapical periodontitis,
- periodontal disease,
- pericoronitis,
- infection of a cyst of the jaws.

Pathology

An abscess may be defined as a pathological cavity filled with pus and lined by a pyogenic membrane. The latter classically consists of granulation tissue but in a rapidly expanding lesion it may simply be a rim of inflammatory cells. The soft tissue surrounding an alveolar abscess may become swollen as a result of the inflammatory exudation and reactive to bacterial products, which have diffused from the abscess.

Local Symptoms

*Pain.*The severity of the pain depends on the stage ofdevelopment of the inflammation. In the initial phasethe pain is dull and continuous and worsens duringpercussion of the responsible tooth or when it comesinto contact with antagonist teeth. If the pain is verysevere and pulsates, it means that the accumulation ofpus is still within the bone or underneath the periosteum. Relief of pain begins as soon as the pus perforatesthe periosteumand exits into the soft tissues.

*Edema.*Edema appears intraorally or extraorally andit usually has a buccal localization and more rarelypalatal or lingual. In the initial phase soft swelling ofthe soft tissues of the affected side is observed, dueto the reflex neuroregulating reaction of the tissues,especially of the periosteum. This swelling presentsbefore suppuration, particularly in areas with loosetissue, such as the sublingual region, lips, or eyelids.Usually the edema is soft with redness of the skin.During the final stages, the swelling fluctuates, especiallyat the mucosa of the oral cavity. This stage isconsidered the most suitable for incision and drainageof the abscess.

Other Symptoms. There is a sense of elongation ofthe responsible tooth and slight mobility; the toothfeels extremely sensitive to touch, while difficulty inswallowing is also observed.

Systemic Symptoms. The systemic symptoms usually observed are: fever,which may rise to 39-40 °C, chills, malaise with paininmuscles and joints, anorexia, insomnia, nausea, andvomiting. The laboratory tests show leukocytosis orrarely leukopenia, an increased erythrocyte sedimentationrate, and a raised C-reactive protein level.

Diagnosis

Diagnosis is usually based upon clinical examinationand the patient's history. What mainly matters, especiallyin the initial stages, is the localization of the responsibletooth. In the initial phase of inflammation,there is soft swelling of the soft tissues. The tooth isalso sensitive during palpation of the apical area andduring percussion with an instrument, while the toothis hypermobile and there is a sense of elongation. Inmore advanced stages, the pain is exceptionally severe,even after the slightest contact with the tooth surface.

Tooth reaction during a test with an electric vitalometeris negative; however, sometimes it appears positive,which is due to conductivity of the fluid inside theroot canal.

Radiographically, in the acute phase, no signs areobservedat the bone (whichmaybeobserved8-10 dayslater), unless there is recurrence of a chronic abscess,whereupon osteolysis is observed. Radiographic verificationof a deeply carious tooth or restoration veryclose to the pulp, as well as thickening of the periodontalligament, are data that indicate the causative tooth.

Differential diagnosis of the acute dentoalveolarabscess includes the periodontal abscess, and the dentistmust be certain of his or her diagnosis, becausetreatment between the two differs.

Complications

If the inflammation is not treated promptly, the followingcomplications may occur: trismus, lymphadenitisat the respective lymph nodes, osteomyelitis, bacteremia,and septicemia.

Depending on the pathway andinoculation site of the pus, the acute dentoalveolar abscess may have various **clinical presentations**, such as:

- intraalveolar,
- subperiosteal,
- submucosal,
- subcutaneous,
- fascial ormigratory – cervicofacial.

The initial stage of the cellular phase is characterizedby accumulation of pus in the alveolar bone and istermed *an intraalveolar abscess*.

The pus spreads outwards from this site and, after perforating the bone, spreads to the subperiosteal space, from which the *subperiosteal abscess*originates, where a limited amount of pus accumulates between the bone and periosteum.

After perforation of the periosteum, the pus continues to spread through the intraorally, spreading underneath the mucosa forming the *submucosal abscess*.

Sometimes, though, it spreads through the loose connective tissue and, after its pathway underneath the skin, forms a *subcutaneous abscess*, while other times it spreads towards the fascial spaces, forming serious abscesses called *fascial space abscesses*.

The fascial spaces are bounded by the fascia, whichmay stretch or be perforated by the purulent exudate,facilitating the spread of infection. These spaces arepotential areas and do not exist in healthy individuals, developing only in cases of spread of infection thathave not been treated promptly.

Some of these spaces contain loose connective tissue,fatty tissue, and salivary glands,while others containneurovascular structures. Acute diffuse infection,which spreads into the loose connective tissue to agreat extent underneath the skin with or without suppuration,is termed cellulitis (phlegmon).

Intraalveolar Abscess

*Anatomic Location.*This is an acute purulent infection,which develops at the apical region of the tooth incancellous bone.

*Etiology***.** It is usually caused by bacteria originatingfromany infected tooth of the maxilla ormandible.

*Clinical Presentation.*The symptoms that are characteristicof this condition are severe pulsating pain, toothmobility, and sense of elongation of causative tooth.

*Treatment.*Treatment aims at relieving the patient ofpain initially, and then saving the tooth. First, drainageis attempted through the root canal.

The tooth is drilled with a high-speed handpiece withmanipulations as gentle as possible, because the toothis exceptionally sensitive even after mere contact. Tofacilitate the evacuation of pus, the necrotic materialmust be removed with a barbed broach from the rootcanal and then slight pressure is applied at the apicalregion of the tooth.

If drainage through the root canal is not possible,then treatment consists of trephination after the positionof the apex is establishedwith a radiograph.Duringthe surgical procedure, a small horizontal incisionis made buccally on the mucosa, as close to the apex ofthe tooth as possible. Afterwards, the periosteum isreflected as far as the tip of the root and the buccalbone is exposed. Using a round blunt bur, with slowrotation and under a steady stream of saline solution,bone is removed, establishing communication withthe periapical infection. This procedure resultsin drainage of exudate and relief of pain. Aftercompletion, the wound is sutured, without placementof a rubber drain being necessary.

Subperiosteal Abscess

Anatomic Location.The subperiosteal abscess involveslimited accumulation of pus that is semi-fluctuant.

It is located between bone and the periosteum, atthe buccal, palatal, or lingual region, relative to thetooth responsible for the infection.

Etiology.This type of abscess is the result of spread ofan intraalveolar abscess, when the pus perforates thebone and becomes established underneath the periosteum.

*Clinical Presentation.*It is characterized by mildedema, severe pain due to tension of the periosteum,and sensitivity during palpation.

*Treatment.*This abscess is treated with an intraoralincision and drainage. The incision is performed onthe mucosa, taking into consideration the course ofthe vessels and nerves in the region (mental nerve andpalatal vessels and nerves) in order to avoid injury.Thescalpel blade reaches bone, to ensure greater drainageof pus.

Submucosal Abscess

*Anatomic Location.*This abscess is located exactlyunderneath the buccal or labial vestibular mucosa ofthe maxilla or mandible, as well as the palatal or lingualregion, respective to the tooth responsible for theinfection abscesses also cause this type of abscess. The teethnormally considered responsible for the developmentof a palatal abscess are themolars and lateral incisor ofthe maxilla.

*Clinical Presentation.*Swelling of the mucosa withobvious fluctuation is observed, as are sensitivity duringpalpation, and obliteration of the mucobuccal foldin the area of infection. As far as the palatal abscess isconcerned, it manifests as a circumscribed swelling,respective to the responsible tooth. Themucosaappears reddish, while sensitivity is observedduring palpation and fluctuation.

*Treatment.*The incision is made superficially with ascalpel blade. A small hemostat is then inserted insidethe cavity in order to create a broader drainage routeand a rubber drain is inserted so thatthe drainage route is kept open for at least 48 h. Incisionand drainage of palatal abscesses require specialattention to ensure avoiding injury to the greater palatineartery, vein, and nerve. Therefore, the incisionmust not be made perpendicular to the course of theaforementioned vessels and nerve, but near the borderof the gingivae or towards the midline and parallel tothe dental arch. Drainage of the abscess isachieved with a curved hemostat. Afterdrainage, the patient is relieved of pain, and resolutionof the abscess, in other words the healing stage,begins.

Management of an acute alveolar abscess

1. Drainage
   - tooth extraction
   - root canal drainage
   - incision
2. Removal of source of infection
   - endodontic treatment
   - extraction
   - periradicular surgery
3. Supportive antibiotic therapy
   - severe spreading infection
   - systemic toxicity
   - medically compromised

Drainage. Provision of drainage remains the most importantmeasure where pus has formed.

Extraction of the tooth is usually carried out if it isunconservable or if the acuteness of the conditionwarrants it. This provides excellent drainage, especiallyin upper teeth. If the abscess is still confined to thealveolar bone, or even where spread to soft tissues is inits early oedematous phase, extraction is often sufficientto allow resolution. There is no justification whatsoeverin delaying the extraction on account of swelling becausethis is one of the best ways of achieving good drainage,and thereby speeding resolution.

Opening of the root canal can, in the early intrabonyabscess, be sufficient in itself. When opened and pus isobtained, it is prudent not to close the canal immediatelybut allow sufficient time for adequate drainage to occur.This may take only 12-24 h but gross contamination andeven caries within the root canal can occur if the tooth isleft open for too long.

Incision of intraoral abscesses

The presence of pus is usually detected by a palpable«bounce» in the swelling within the buccal sulcus. Local anaesthesia within the outer wall only of the abscessis normally sufficient to allow incision with a pointedscalpel blade, which should be used with astab action and a cut of suitable size made on withdrawal.

If the pus is deeper, fine artery forceps may be introducedto explore the abscess and gently open it to encouragefree flow of pus. This may require more extensive localanaesthesia. Palatal abscesses may require excision of asmall ellipse of soft tissue or insertion of a small drainafter incision as this mucoperiosteum tends to reboundand seal following the release of pus, thus preventingfurther free drainage.

Removal of source of infection

This will be accomplished immediately if the tooth isextracted in the process of gaining drainage but may be alater procedure in the form of either endodontic treatmentor periradicular surgery.

Supportive antibiotic therapy

The decision to prescribe antibiotics for acute abscesseswill depend upon a number of factors and is notinvariable. Extraction of a maxillary tooth or incision ofa well-localised sulcus abscess may well be sufficient toallow resolution of the acute infection without antibioticsupplemental treatment. Clinical experience is invaluablebut some features may well influence the decision toprescribe an antibiotic.

The swelling may encroach on the airway as, forexample, in a submandibular or sublingual infection.

Difficulty in swallowing normally implies that the tongue,floor of mouth or parapharyngeal spaces are affected. Ifincision and drainage have yielded poor quantities ofpus, this suggests that management may be inadequatewithout antibiotics. Regional nodes are often late to reactto acute dental infection or are masked by the swelling.

If present, this is good evidence of spreading infection,which would require supplemental antibiotic treatment.

If the patient is medically compromised, and particularlyif the immune system is compromised, patients may nothave the capability to respond adequately to this typeof infection. Patients with conditions such as diabetesmellitus, HIV infection, or who are receiving treatmentwith corticosteroids or cytotoxic drugs, can be includedin this group. Where infection is evident in the floor ofthe mouth, there appears to be an

increased likelihoodof rapid spread. The patient may feel unwell, with anelevated temperature and a tachycardia, signifyingsystemic toxicity. Most severe infections will cause a degree of trismus by simply stretching the soft tissues,but profound trismus often implies infection affectingmuscles either directly or indirectly involved in jawmovement.

The choice of antibiotic is initially empirical, as theresults of antibiotic sensitivity testing will not be available.

The penicillin group is often chosen in non-allergicpatients, with the wider-spectrum amoxicillin being acommon choice. Metronidazole is becoming more popularas a first choice antimicrobial because anaerobes are thepredominant pathogens. Cephalosporins, erythromycinand, where antibiotic sensitivity indicates, clindamycinare all possible useful agents. In severe acute infections,it is sometimes considered necessary to prescribe two ormore antibiotics. Metronidazole and amoxicllin haveproved to be a popular choice where two antibiotics are deemed necessary.

OSTEOMYELITIS OFTHE JAWS

Osteomyelitis of the jaws is still a fairly common disease in maxillofacial clinics and offices, despite the introduction of antibiotics and the improvement of dental and medical care.

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. 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.

History. The prevalence, clinical course, and management of osteomyelitis of the jawbones have changed profoundly over the past 50 years. This is due to mainly one factor: the introduction of antibiotic therapy, specifically penicillin. The integration of antibiotics into the therapeutic armamentarium has led to a complete renaissance in the treatment of most infectious diseases, including osteomyelitis. Further factors, such as sophistication in medical and dental science as well as the widespread availability for adequate treatment, have additionally led to improvement in the management of this disease. Modern diagnostic imaging allows much earlier treatment of bone infections at a more localized stage. In the preantibiotic era, the classical presentation of jawbone osteomyelitis was an acute onset, usually followed by a later transition to a secondary chronic process. Massive clinical symptoms with widespread bone necroses, neoosteogenesis, large sequester formation, and intra- and extraoral fistula formation were common presentations, sometimes leading to significant facial disfigurement. After the introduction of antibiotics, acute phases were often concealed by these antimicrobial drugs without fully eliminating the infection.

Classification for osteomyelitis of the jaws:

I. Acute forms of osteomyelitis (suppurative or nonsuppurative)

1. Contagious focus
   - Trauma
   - Surgery
   - Odontogenic Infection
2. Progressive
   - Burns
   - Sinusitis
   - Vascular insufficiency
3. Hematogenous(metastatic)

II. Chronic forms of osteomyelitis

1. Recurrent multifocal
   - Developing skeleton (children)
   - Escalated osteogenic (activity < age 25 years)
2. Garrè's
   - Unique proliferativesubperiosteal reaction
   - Developing skeleton (children and young adults)
3. Suppurative or nonsuppurative
   - Inadequately treated forms
   - Systemically compromisedforms
   - Refractory forms (chronic recurrent multifocal osteomyelitis)
4. Diffuse sclerosing
   - Fastidious microorganisms
   - Compromised host/pathogeninterface

Diagnostic of osteomyelitis of the jaws

Imaging is a crucial diagnostic tool in the assessment of acute and chronic osteomyelitis of the jaws. Before any cross-sectional imaging modality is applied, the orthopanoramic view is the first image to assess the status of dentition, recognize direct radiographic signs of osteomyelitis, narrow the differential diagnosis, and depict potential predisposing conditions such as a fracture or systemic bone disease. The orthopanoramic view is furthermore the first-line image when follow-up examinations are performed.

In acute osteomyelitis the higher sensitivity of magnetic resonance imaging (MRI), with respect to detection of intramedullary inflammation, advocates its use as the imaging modality of choice to confirm the

diagnosis and provide an estimate of the intraosseous extent and soft tissue involvement.

In case surgical treatment is planned, high-resolution computed tomography (CT) is required to specify the degree of cortical destruction, delineate the presence of sequestra, and to define the extent of osseous removal required.

In chronic osteomyelitis the higher sensitivity of CT with respect to detection of sequester and sclerotic bone changes renders CT the examination of choice to distinguish the usually more uniform and extensive primary chronic osteomyelitis from the more localized type of secondary chronic osteomyelitis. Magnetic resonance imaging is superior to detect periosteal inflammation and soft tissue involvement and thus aids in determining the persistence or recurrence of infection. Following surgery, CT is preferred as follow-up examination for a period of 6 months to distinguish postoperative and reparative changes from recurrent or persistent infection.

Complimentary information is gained in particular situations by a combination of imaging modalities adapted to the individual patient's course of disease and the panoramic view findings.

In order to confirm and assess osteomyelitis of the jaws, a spectrum of radiological techniques from which to choose is available. Conventional radiographs serve as first-line examination. Computed tomography (CT) and MRI are well-established high-resolution cross-sectional imaging techniques which provide precise morphological information regarding bone and soft tissue involvement.

With scintigraphy a sensitive technique is available − limited only by low spatial resolution and specificity. Fusion positron emission tomography – CT (PET-CT) offers a combined technique using both the advantages of CT imaging and labelled radionucleotides to gather information on anatomical structure and metabolic activity of the examined region.

The panoramic view is the first examination in a patient clinically suspected of having developed osteomyelitis of the jaw. Depiction of the status of the dentition and of the bone structure is readily provided by the OPT.

Following a dental procedure, a tooth extraction in the molar region in particular, osteomyelitis may develop either due to persistence of a preexisting focus or due to de novo infection of the tooth socket. Comparison of the recent panoramic view with previously performed radiographs facilitates recognition and distinction of an incipient new infection or persistence and reactivation of a previous process.

The area of osteolysis is commonly related to an empty tooth socket or a diseased tooth. Initial radiographic indicators may be a widened periodontal ligament space or a defect of the lamina dura. Destruction of bone initially proceeds within cancellous bone.

The cortical plate is secondarily involved by progressive bone resorption and increasing pressure exerted by the inflammation. Additional early signs are erosion of the endosteal contour of the basal mandibular cortical bone or in the upper jaw effacement of the contour of the alveolar maxillary recess. Within the third and fourth weeks radiographs tend to become mostly pathological. Findings consist of usually ill-defined areas of radiolucency, sequestra, calcified periosteal reactions and occasionally fistulae. In this advanced stage of acute osteomyelitis sequester may add to the radiological findings. Based on conventional images, the occurrence of sequester is considered rare within the first 4 weeks and more typically is referred to the chronic stage

Conventional radiological signs in acute osteomyelitis of the jaws:

• Increased radiolucency
• Loss of trabecular structure
• Loss of contour of mandibular canal
• Pseudo-widening of mental foramen and mandibular canal
• Erosion of cortical bone
• Calcified periosteal reaction
• Minor areas of sclerosis interspersed with a zone of increased radiolucency
• Fracture as potential complication

Conventional radiological signs in chronic osteomyelitis of the jaws:

Secondary chronic osteomyelitis:

• Areas of increased radiopacity with loss of bone trabeculae
• Minor areas of radiolucency, interruption of cortical bone
• Sequester formation
• Calcified periosteal reaction
• Pathological fracture

Primary chronic osteomyelitis

• Areas of increased radiopacity with loss of bone trabeculae, effacement of cortical–cancellous bone junction affecting a hemimandible
• Minor spots of radiolucency
• Rarely periosteal reaction
• Temporo mandibular joint involvement

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. 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.

Etiology. As mentioned previously, there are several etiological factors, such as traumatic injuries, radiation, and certain chemical substances, among others, which may cause inflammation in the medullar space of the bone; however, acute and secondary chronic osteomyelitis, as these terms are generally used in the medical and dental literature and in this textbook, represent a true infection of the bone induced by pyogenic microorganisms. The oral cavity harbors a large number of bacteria, among which many may be identified as possible pathogens to cause infection of the jawbone. Regarding the high frequency and sometimes severity of odontogenic infections in the daily dental and oral surgery practice, and the intimate relationship of dental roots apices with the medullar cavity of the jawbone, it is remarkable that osteomyelitis cases are not more frequently observed.

The clinical appearance of acute osteomyelitis of the jaws may show a great variety, depending on the intensity of the disease and the magnitude of imbalance between the host and the microbiological aggressors.

Three principal types of clinical courses of acute osteomyelitis can be distinguished:

- Acute suppurative
- Subacute suppurative
- Clinically silent with or without suppuration

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 identified. Pus may exude around the gingival sulcus and through mucosal and, possibly cutaneous, fistulas. A fetid oral odor caused by anaerobic pyogenic bacteria often is present. Teeth in the affected region may demonstrate increased mobility even leading to malocclusion and show decreased or loss of sensitivity. Sequester formation and appositional neoosteogenesis are limited, if not absent, due to the short period since establishment of deep bone infection, which is the definition of acute osteomyelitis. Neonatal or tooth-germ-induced acute osteomyelitis of the jaws, as described previously, is a classical representative of this group, although this form of osteomyelitis has become a rarity in modern maxillofacial practice. But also in elderly patients this form of acute osteomyelitis has been seen much less frequently since the introduction of antibiotics and sophistication of medical and dental practice. In cases of a subacute or silent course, with or without suppuration, the clinical presentation is by definition less impressive. This can make an early diagnosis increasingly difficult, and in many instances these cases are not detected until they have become secondary chronic.

Laboratory findings. Depending on the intensity of the infection, laboratory results in acute osteomyelitis may demonstrate a wide range. While in cases with little inflammation the laboratory will only reveal moderate evidence of acute infection, cases which are accompanied by abscess formation will show more pronounced findings. When osteomyelitis highly demonstrative changes in the blood: MPD 35 mm/h and higher, while in acute purulent periodontitis ESR does not exceed 15 to 20 mm/h and only in rare cases up to 30 mm/H.

Characterized by the change of resistance of erythrocytes in the direction of maximum lifting and falling minimum value of resistance in contrast to the severe periodontitis, which is on the rise only maximum values. The number of leukocytes $1 \cdot 10^1$ in 1 mm. Nuclear shift white blood characterized by the increase in the number of stab and young forms, reducing the number of eosinophils. In acute periodontitis, on the contrary, the number of eosinophils usually does not change. In severe cases of osteomyelitis of jaw disappearing eosinophils is a feature, rather unfavorable prognostic. Adverse prognostic symptom is and the fall in the number of monocytes. In cases there is a septic limfopenia indicating poor resistance.

Urine in acute osteomyelitis concentrated often with traces of protein; sometimes you can see the cylinders.

*Treatment*of patients with acute osteomyelitis of the jaws should be done in a hospital and is in the surgical intervention consisting in the exposure of the bone by intra- or extraoral cuts and the General treatment measures. When deciding about the destruction caused osteomyelitis tooth need to carefully analyze the phase in which there is inflammation. Above it was said that when periodontitis, i.e. in the inflammatory process in the developing periodontal disease, tooth extraction is the main method that provides sufficient outflow of pus. Certainly shows the removal of the tooth and when inflammation acute purulent periodontitis are increasing. Isolated tooth extraction acute diffuse osteomyelitis cannot be considered appropriate, as such interference cannot provide emptying scattered in the jaw bone of a purulent foci. Some Russian dentists adhere to the view that the removal of the tooth is advisable in any stage of odontogenic inflammatory process jaw. We believe that the removal of the cause of tooth shown in an early stage during acute osteomyelitis with increasing inflammatory effects. Such intervention, providing outflow of exudate, may suspend the further spread of the process, but in later stages, when the process has already spread from the wells of cause tooth removal it without the simultaneous opening of the suppurative focus in the soft tissues inefficient. From deletion in acute osteomyelitis of jaw involved in the process neighboring cause teeth should be avoided, as they are after the liquidation process usually get stronger again in the jaw, unless rejected together with the decline in the transition process in the chronic form. Outpatient advisable in order of first aid to dissect the mucous membrane and the periosteum to the bone on all extent of the inflammatory process on the level of tops involved in the process of teeth.

Chronic osteomyelitis

Chronic osteomyelitis is characterised by a clinical course lasting over a month. It may occur after the acute phase or it may be a complication of tooth-related infection without a preceding acute phase. The clinical presentation is milder, with painful exacerbations and discharge of pus or sinus tracts.

Chronic osteomyelitisis characterised by a clinical course lasting over a month. It may occur after the acute phase or it may be a complication of tooth-related infection without a preceding acute phase. The clinical presentation is milder, with painful exacerbations and discharge of pus or sinus tracts.

After the acute inflammatory process occurs andlocal blood supply is compromised, necrosis of the endostealbone takes place. The bone fragments die andbecome sequestra. Osteoclastic activity is thenresponsible for separating the dead bone from vitalbone. Devital bone tissue clinically appears dirty, whitish-gray with an opaque appearance. Its fatty tissue hasbeen destroyed and it does not bleed if scraped. In some instances the bone sequester can demonstrateconsiderable dimensions.

The elevated periosteum involved in the inflammatoryprocess still contains vital cells. These cells, oncethe acute phase has passed, form a new bony shell (involucrum)covering the sequester. The involucrum maybe penetrated by sinuses called cloacae, through whichpus discharges, elevating the periosteum or formingfistula. As chronification progresses this scenario maybe repeated. The involucrum tends tohinder sequester from extruding, which perpetuates theprocess because the whole area isbathed in increasingamounts of pus unless treated promptly and adequately.In secondary chronic osteomyelitis of the jaws, eventuallya new equilibrium is established between the hostand the aggressor causing the infection. The nature ofthis newly formed equilibrium is dependent on host immunitysupported by medical therapy and the causativebacteria.

Secondary Chronic Osteomyelitis

As a sequel of acute osteomyelitis, the clinical presentationof secondary chronic osteomyelitis of the jaws mayalso show a great variety, depending on the intensity ofthe disease and the magnitude of imbalance betweenthe host and the microbiological aggressors and thetime. Following an acute or subacute clinicalphase with suppuration, the chronification of thedisease is reflected by the clinical course and findings.

Most symptoms, such as pain and swelling, are usuallyless extensive in the chronic than in the acute stage. Thedeep and intense pain frequently observed in the acutestage is replaced by a more dull pain. Painful swellingcaused by local edema and abscess formation in theacute stage is subsided by a harder palpable tendernesscaused by periosteal reaction.Other symptoms are somewhat more predominant inadvanced stages, such as sequester and fistula formation,and are regarded as classical signs of secondarychronic osteomyelitis. Thenoted fetid odor often noted in cases of acute abscessformation is less frequent in patients with secondarychronic osteomyelitis. A disturbed occlusion can sometimesbe noted when teeth of an affected region becomemore mobile and elongate due to rise of intraosseouspressure or a fracture present as a result or initiator ofthe osteomyelitic process.

In cases where the acute phase was clinically silent,secondary chronic osteomyelitis may begin as a hideousdisease with little and somewhat unspecific clinicalsymptoms. In such instances the cause of the infectionis considered to be a low-grade infection, which, however,cannot be fully eradicated by host defenses. Thesecases of secondary chronic osteomyelitis demonstrateless pus, fistula, and sequester formation, or may evenlack these symptoms at a certain (progressive) stage ofthe disease. Furthermore, their radiological appearancemay predominantly show a diffuse sclerosis with littleto no osteolysis. Probably a large portion of the casesdescribed in the literature as diffuse sclerosing osteomyelitis(DSO) falls into this category. A differentiationfrom primary chronic osteomyelitis may be difficult, ifnot impossible, in such cases; hence,it is most important to review the whole course of thedisease and possibly obtain repeated imaging over timein such cases to establish the correct diagnosis.

Laboratory FindingsIn analogy to the clinical symptoms, the laboratoryfindings in secondary chronic osteomyelitis of the jawsare usually less prominent than in acute osteomyelitis.

The overall moderate systemic reactions are reflectedby these results and indicate a more localized infectiousprocess, especially in secondary chronic osteomyelitiscases. This is especially true in cases with little ormild clinical symptoms where laboratory findings canbe almost normal and hence are of little diagnostic ormonitoring value.

Primary Chronic Osteomyelitis

Acute and secondary chronic osteomyelitis of the jaw,as being the same disease at a different stage, share thesame etiology, a bacterial or, in rare cases, a fungal infection.

In the literature acute and secondary chronicpurativeosteomyelitis, indicating a true bacterial infectionwith formation of pus.

The term «primary chronic osteomyelitis», as used inthe Zurich classification of osteomyelitis of the jaws, refersto a rare inflammatory disease of unknown etiology.

It is characterized as a strictly nonsuppurative chronicinflammation of the jawbone with the absence of pusformation, extra- or intraoral fistula, or sequestration.

The absence of these symptoms represents a *conditio sinequa non* and clearly differentiates primary from acuteand secondary chronic osteomyelitis in most cases. Theterm«primary chronic osteomyelitis» also implies thatthe patient has never undergone an appreciable acutephase and lacks a definitive initiating event.

The disease tends to a rise de novo without an actualacute phase and follows an insidious course. Inmost cases of primary chronic osteomyelitis, periodicepisodes of onset with varying intensity last from a fewdays to several weeks and are intersected by periods ofsilence where the patient may experience little to noclinical symptoms. In active periods dull to severe pain,limitation of jaw opening and/or myofacial pain, as wellas variable swelling, may be observed. In certain casesregional lymphadenopathy and reduced sensation ofthe inferior alveolar nerve (Vincent's symptom) are alsoaccompanying symptoms.

Primary chronic osteomyelitis of the jaws almost alwaystargets the mandible. In our patient data all butone case of primary chronic osteomyelitis involvedexclusively the lower jaw. In the remaining case, thezygoma demonstrated the clinical, radiological, andhistopathology findings as the mandible, indicating apossible spread of the pathological condition.

Osteoradionecrosis

Radiotherapy is considered a major column in the treatmentof head and neck malignancies. Despite recentadvances in radiotherapy, such as using modern threedimensionaltechniques, as well as hyperfractionationor moderately accelerated fractionation and consequentprophylactic dental treatment, osteoradionecrosis is stillan observed condition in maxillofacial units.

Aside from its effect on the tumor cells, radiationalso has serious side effects on the soft and hard tissuesadjacent to the neoplasm. Mucositis, atrophic mucosa,xerostomia, and radiation caries are well-known sideeffects of head and neck radiotherapy. Because of itsmineral composition, bone tissue absorbs more energythan soft tissues and is therefore more susceptible tosecondary radiation. In cases where the bone is irradiatedexceeding a certain local dose, osteoradionecrosismay develop, leading to marked pain in the patient andpossible loss of bone leading to functional and aestheticimpairment.

Osteoradionecrosis was once considered an infectioninitiated by bacteria, which invaded the radiationdamagedbone; hence, the term «radiation-induced osteomyelitis»or radioosteomyelitis was commonly used. Conclusively identified this condition as aradiation-induced avascular necrosis of bone. He wasable to demonstrate that radiation caused a hypoxic, hypocellular,and hypovascular tissue, leading to a spontaneousor trauma-initiated tissue breakdown. The resultis a chronic nonhealing wound, susceptible to superinfection.

Osteochemonecrosis

The medical literature describes several drugs and substancesthat facilitate or induce conditions known asosteonecrosis of the jaws, such as corticosteroids andother cancer and antineoplastic drugs. Exposure towhite phosphorous among workers in the matchmakingindustry in the nineteenth century has led to unusualnecroses of the jaws, which became known in theliterature as phossy jaw or phosphorous necrosis of thejaw.In the recent years bisphosphonate therapy has becomea widely accepted mainstay of therapy in variousclinical settings such as multiple myeloma, metastaticcancer therapy, and treatment of advanced osteoporosis.

With the increased prescription of these drugs, theincidence and prevalence of bisphosphonate-associatedcomplications of the jaw continues to be elucidated. Thistrend seems to be even more the case in patients receivinginjectable bisphosphonates, such as pamidronate andzoledronic acid, but cases involving osteochemonecrosisof the jaw associated with chronic peroral administeredbisphosphonates have also been reported.

The pathophysiological mechanisms leading to bisphosphonate-induced osteochemonecrosis of the jawsare yet far from being fully understood; however, it seemsapparent that important differences to the pathogenesisof osteoradionecrosis do occur.

In bisphosphonate-induced osteochemonecrosisof the jaws osteoclastic action is reduced, but osteoblasticproduction continues, leading to an osteopetrosislikecondition. These alterations inbone physiology with eventual increase of the medullarybone as the disease progresses and the inability ofosteoclasts to remove superinfected diseased bone areregarded as causative factors.

In contrast to osteoradionecrosis,where a radiation-induced avascular necrosisis the major cause, avascularity does not appear to be amajor cofactor to date; however, inhibition of angiogenesisis currently being actively investigated, and further research willhopefully help fully understanding its role in pathogenesisof this disease.

Regarding the current data and knowledge, we favorthe term «bisphosphonate-induced osteochemonecrosisof the jaw» because it is not restricted to a certainpathogenesis. The term «bisphosphonate osteomyelitis» should not be used for the same reasons as the termradioosteomyelitis should be abandoned.

Differential diagnosis

Acute and early-stage secondary chronic osteomyelitis (predominant osteolysis)

– Primary bone tumors
– Bone metastasis
– Primary intraosseous or invasive growing squamous cell carcinoma
– Early osteoradionecrosis
– Eosinophilic granuloma
– Plasmocytoma
– Demineralized bone in dialysis patients

Acute and Advanced-stage secondary chronic osteomyelitis (osteolysis and sclerosis):

– Primary bone tumors
– Bone metastasis
– Primary intraosseous or invasive growing squamous cell carcinoma
– Osteoradionecrosis
– Osteochemonecrosis (bisphosphonate induced)
– Plasmocytoma
– Demineralized bone in dialysis patients
– Tendoperiostitis
– Primary chronic osteomyelitis

Differential diagnosis of primary chronic osteomyelitis of the jaws

• Fibrous osseous dyplasia (FOD/Jaffé-Lichtenstein)
• Periapical cemental dysplasia
• Enostosis (compact island), bone scar
• Ossifying fibroma, osteoma, osteochondroa
• Osteosarcoma
• Tendoperiostitis
• Paget's disease (deforming ostitis)
• Osteopetrosis (Albers-Schonberg disease)

General Aspectsof Osteomyelitis Therapy

Until the mid-twentieth century, the treatment of osteomyelitisof the jaws, like osteomyelitis of long bones in other parts of the skeleton had been primarily surgical.

Back then, osteomyelitis of the jaws was an infectiousdisease with an often complicated course, involvingmultiple surgical interventions and not seldom leadingto facial disfigurement as a result of loss of affected boneand teeth and the accompanying scarring. The outcomewas usually all but certain and hence prolonged treatmentand frequent relapses have been associated withthis disease in the past; however, since the second halfof the past century there has been a dramatic reductionof the incidence of osteomyelitis cases involving the jawsand other bones of the skeleton. Themajor responsible factor leading to this developmentmust probably be seen in the introduction of antibioticsto the therapeutic armamentarium; however, other factorshave also contributed to this fact such as improvednutrition, and better availability to medical and dentalcare, especially including advances in preventive dentistryand oral hygiene. Earlier diagnosis due to moresophisticated diagnostic imaging modalities has additionallyimproved the morbidity associated with thisdisease.

Current treatment of osteomyelitis of the jaws usuallyconsists of a combination of surgical and antibiotictherapy. Hyperbaric oxygen (HBO) has been establishedfor treatment and prevention of osteoradionecrosis withgood scientific documentation of its therapeutic valuefor this indication; however, the role of adjunctiveHBO in the treatment of osteomyelitis of the jaws has todate not been well defined, and hard scientific evidenceof its therapeutic value is still lacking. Indeed, in acuteand secondary chronic osteomyelitis of the jaws, HBOis usually less frequently required than in cases affectingthe long bones and other parts of the skeleton, becauseof the greater vascularity of the head and neck. In general, in most cases of acute and secondarychronic osteomyelitis of the jaws, resolution is attainablewithout HBO. Despite the aforementioned information,clinical experience shows the adjunctive use ofHBO beneficial in cases proved refractory to surgicaland antibiotic therapy and in patients who are seriouslymedically compromised with no HBO contraindications

Therapeutic goals in treatment of acute and secondary chronic osteomyelitis of the jaws:

- Eradication of infection and removal of infectious focus
- Pain management
- Limitation of further spreading of the disease
- Fracture prophylaxis, and stabilization of infected fractures

- Preservation of anatomic structures when possible
- Prevention of relapse of disease
- Prevention of (further) chronification of the infection
- Reestablishment of anatomy and function

Principles of treatment of acute osteomyelitis of the jaws:

- Establish correct diagnosis, based on history, clinical evaluation, and imaging studies
- Biopsy in unclear cases to rule out other pathology (e.g., malignancy)
- Determine extent of infected bone and soft tissue
- Evaluation and correction of host defense deficiencies when possible
- Removal of source of infection, usually a dental focus, foreign bodies/implants
- Local incision and drainage of pus
- Local curettage with removal of superficial sequestra and saucerization if necessary
- Collection of specimens for Gram stain, culture and sensitivity, histopathology
- Begin with empiric broad-spectrum antibiotic therapy and change to culture-guided antibiotics as soon as possible
- More extensive surgical debridement if necessary (e.g., decortication, resection)
- Possible adjunctive hyperbaric oxygen therapy

Principles of treatment of secondary chronic osteomyelitis of the jaws:

- Establish correct diagnosis, based on history, clinical evaluation, and imaging studies
- Biopsy in unclear cases to rule out other pathology (e.g., malignancy)
- Determine extent of infected bone and soft tissue
- Evaluation and correction of host defense deficiencies when possible Surgical debridement of infected tissue dictated by extent of the lesion (removal of affected teeth and foreign bodies/implants,
- sequestrectomy, local curettage, saucerization, decortication, resection)
- Collection of specimens for Gram stain, culture and sensitivity, histopathology

- Begin with empiric broad-spectrum antibiotic therapy and change to culture-guided antibiotics as soon as possible
- Possible adjunctive hyperbaric oxygen therapy
- More extensive surgical debridement if necessary (e.g., repeated decortication, resection)

Surgery must be considered as the major pillar in the treatment of acute and secondary chronic osteomyelitisof the jaws.

Surgical procedures in acute and secondary chronic osteomyelitis of the jaws pursue three major goals:

- decompression of intramedullary pressure caused by the osteomyelitic process and drainage of subperiosteal abscess formation;
- surgical debridement of infected tissue and removal of the infectious focus;
- bringing well-perfused tissue adjacent to the infected area.

Types of Surgical procedures:

- Incision and drainage of abscess formation
- Removal of loosened teeth, foreign bodies/
- implants and sequestra
- Local curettage and saucerization of the
- infected bone
- Decortication
- Resection and reconstruction

While local incision and drainage ofabscess formation mainly facilitates decompression ofintramedullary pressure, most other procedures mainlytarget surgical debridement, while decortication and(microvascular) reconstruction additionally bring wellperfusedvital tissue to the affected area

Sequestrectomy

Sequester formation is a classical sign of secondarychronic and advanced acute osteomyelitis cases. Usuallya time frame of at least 2 weeks after onset of infectionis necessary until presentation. In general, sequestra areconfined to the cortical bone but may also be cancellousor cortical-cancellous. Once a sequester is fully formed,it may persist for several months in untreated cases beforebeing resorbed or spontaneously expelled throughthe oral mucosa or the facial skin. Resorption of

sequesteris achieved by lytic activity of the osteoclast cells inthe surrounding granulation tissue. Gradually the granulationtissue may ingrow the sequester and promote itsdegradation. If sequestra are not fully removed in treatment,partial, superficial healing may occur. Because sequesterare avascular, they are poorly penetrated by antibioticsor HBO and hence are ideal breading groundsfor bacteria.

Saucerization

The next more extensive step in the surgical debridementof infected jawbone is saucerization. This surgicalprocedure describes the «unroofing» of the oral-facedjawbone to expose the medullary cavity for subsequentthorough debridement. The margins of necrotic boneoverlying the focus of osteomyelitis are excised creatingdirect visualization of the infected medullary cavity.

This allows direct access to formed and formingsequestra, granulation tissue, and affected bone. In alimited fashion the affected alveolar nerve may also beaddressed; however, in cases of advanced acute and secondarychronic osteomyelitis with significant granulationtissue surrounding the inferior alveolar nerve, thecreated access by saucerization is insufficient.The saucerization procedure is usually performed byan oral approach with the advantage of direct access tothe jawbone and avoidance of facial scarring. The oralapproach is, however, more challenging for collecting anoncontaminated specimen for microbiological investigation.

Saucerization can be useful in early acute osteomyelitiscases and cases of limited extent. In early stagesof the infection is benefits decompression of the medullarycavity and allow ready extrusion of pus, debris,granulation tissue, and avascular fragments.

Saucerization of the mandible, step-by-step procedure:

1. Access to the bone by creating a mucoperosteal flap, usually using a gingival crest incision.
2. Reflection of flap should be as limited as possible to preserve local bood supply.
3. Affected teeth (loosened and other dental foci within the affected area) are extracted.
4. The lateral cortex of the mandible is reduced using burs or rongeurs until the sufficient bleeding bone is encountered at all margins, approximately to the level of the unattached mucosa, thus producing a saucerlike defect.
5. Local debridement is performed by removing granulation tissue and loose bone fragments from the bone bed using curettes.

6. The debrided area is thoroughly irrigated with sterile saline solution with or without additional antibiotic.
7. If there is substantial local bleeding due to hyperemia caused by the inflammatory process, a medicated pack may be placed and serve as local compression device.
8. The buccal flap is trimmed and a medicated pack (such as iodoform gauze lightly covered with antibiotic and local steroid ointment is placed for hemostasis and to maintain the flap in a retracted position. The pack is placed firmly without pressure and retained by several nonresorbable sutures, extending over the pack from the lingual to the buccal flap.
9. The pack is remained in situ for several days up to 2 weeks or even more in some instances and may be replaced serveral times untilthe surface of the bed of granulation tissue is epithelialized and the margins have healed.

Decortication

The decortication procedure quicklybecame an established and widespread procedure andmust be seen as the workhorse in surgical osteomyelitistherapy.

In advanced acute and secondary chronic osteomyelitisof the jaws, especially the mandible, use of decorticationpromotes resolution based on the premise thatthe affected cortical bone is avascular and harbors microorganisms. The medullary cavityshows destruction and is largely replaced by granulationtissue and pus. Parenteral or per oral administeredantibiotics cannot reach the affected region. Waitingfor sequester formation and reducing surgery to sequestrectomyas described previously is not an optionbecause of the advanced stage of the infection with riskfor further spread, abscess formation, and cellulitis.

Furthermore, the disadvantages associated with prolongedantibiotic therapy may become more prominentwith time.

The major purpose of the decortication procedure isto remove the chronically infected cortex of the jawboneand gain access to affected medullary cavity to allow asufficient decompression of intramedullary pressureand meticulous surgical debridement under direct visualization.Furthermore, this procedure allows bringingwell-perfused tissue (e.g., masseter muscle) into contactwith bone, promoting further healing.

Irrigation and Drainage

After completing surgical debridement of the osteomyeliticbone, the question of whether or not to place anirrigation drain must be decided. In the past decadessome authors have advocated the use of drainage and/or irrigation devices (with/without) suction in analogyto the treatment of long bone osteomyelitis. In advanced cases of acute and secondarychronic osteomyelitis, especially involving the mandible,the infection represents a deep-seated, well-establishedcondition that may still retain necrotic tissueand microorganisms even after debridement. Irrigationdrains and frequent irrigations reduce the number ofmicroorganisms, the accumulation of toxins, and residualnecrotic tissue promotesas a general rule the use of debriding-type irrigants untilthe outflow has been clear for 24 h and then to switch tophysiological irrigants such as normal saline or Ringer'slactate.

Resection and Reconstruction

The question as to whether reconstruction of a defectafter surgical debridement should be addressed simultaneouslyor in a second-stage procedure is addresseddifferently in the literature. Advocates atwo-stage procedure to reconstruct continuity defectsresulting from surgical debridement of osteomyelitiswith reconstruction of the bone commencing as earlyas 3 months after debridement, provided that skin andmucosa are intact and the tissue is free of contaminationand infection.

The two-stage procedure obviates simultaneous reconstructionand thus placement of a reconstructionplate and/or an immediate bone graft, which possibleharbors the risk of contamination and infection.

Antibiotic Therapy

The therapy of *primary chronic osteomyelitis* remainscontroversial since the etiology and pathogenesisof this rare disease are not yet understood. A bacterialcause is discussed by some authors but has not beenproven; hence, the role of antibiotic therapy in thesecases is unclear.In cases of acute osteomyelitis, antibiotic treatmentdoes not differ from other deep-seated infections orsepsis. High-dose therapy with a bactericidal antibioticfor about 6 weeks is required. With all betalactams, high-dose signifies intravenoustherapy, since the tolerance of oral betalactamprecludes very high doses and the oral bioavailabilityis limited. In contrast, with quinolones, clindamycin,metronidazole, fusidic acid, trimethoprim/sulphametoxazoleand rifampin, serum and tissue levels are similarwhether these agents are given orally or by the i.v.route.

As a general rule in management of infectious diseases,the narrowest possible spectrum of antibioticsshould be used, in order to avoid significant alterationsof the normal mucosal flora with a shift to

multiresistantmicroorganisms and fungal agents. Since narrowspectrumantibiotics can only be used in microbiologicallywell-defined osteomyelitis, sampling of biopsiesfor culture ideally should precede antimicrobial therapy.

Unfortunately, harvesting deep tissue samples maybe surgically demanding in some cases, and thereforeit is often combined with debridement procedure, dictatinga different order of proceeding. Fusidic acid orrifampin should not be used as single agent, in order toavoid emergence of resistance.

Secondary chronic osteomyelitis of the jaws is definedas infection of more than 1-month duration. Inthis situation, antibiotic therapy must usually be combinedwith sufficient debridement surgery (e.g., decortication,partial resection if necessary). Surgery is mainlyrequired for removal of necrotic bone, and bringingwell-perfused vital tissue adjacent to the site of infection.

The former is important since bacteria tend to persiston the surface of dead bone. Persistence is caused by bacterial adherence tobone. Bacteria have similar properties, whether theyadhere to foreign devices (implants) or to dead bone.

Adherent bacteria are in the stationary phase of growth. Thisexplains their phenotypic resistance to many antibiotics.

The antibacterial efficacy of Betalactams on non-growingbacteria is limited, since they interfere with cell wallsynthesis, which does not take place during stationaryphase. In secondary chronic osteomyelitis, especially incases associated with an infected implant, transplant,or foreign body, antibiotics need to act on stationaryphasebacteria. This is the case for quinolones againstGram-negative aerobes and for rifampin or clindamycinagainst staphylococci. The excellent efficacy of clindamycinmonotherapy and rifampin combination therapy in secondarychronic osteomyelitis due to *S. aureus* has beenshown in a rabbit model

LYMPHADENITIS OF THE MAXILLOFACIAL AREA

Lymphadenitis is the inflammation or enlargement of a lymph node. Lymph nodes are small, ovoid nodules normally ranging in size from a few millimeters to 2 cm. They are distributed in clusters along the course of lymphatic vessels located throughout the body. The primary function of lymph nodes is to filter out microorganisms and abnormal cells that have collected in lymph fluid.

Lymph node enlargement is a common feature in a variety of diseases and may serve as a focal point for subsequent clinical investigation of diseases of the reticuloendothelial system or regional infection. The majority of cases represent a benign response to localized or systemic infection. Most children with lymphadenitis exhibit small, palpable cervical, axillary, and inguinal lymph nodes. Less common is enlargement of the suboccipital or postauricular nodes. Palpable supraclavicular, epitrochlear, and popliteal lymph nodes are uncommon, as are enlarged mediastinal and abdominal nodes.

Lymphadenitis may affect a single node or a group of nodes (regional adenopathy) and may be unilateral or bilateral. The onset and course of lymphadenitis may be acute, subacute, or chronic.

Lymphadenitis: ICD-10 code

The international classifier of diseases of the tenth revision includes the XII class – «Infections of the skin and subcutaneous tissue» with a rubricator in which the lymphadenitis of the acute form corresponds to the L04 encoding. If there is a need to specify an infectious agent, use an additional identification with code B95-B97.

In turn, acute lymphadenitis is divided:

- L04.0 – pathological foci located in the face, neck, head;
- L04.1 – lymph nodes of the trunk are inflamed;
- L04.2 – the disease is found on the upper limbs (shoulders, axillary hollows);
- L04.3 – detection of affected nodes (pathology is acute) on the lower extremities (pelvic region);
- L04.8 – localization in other areas;
- L04.9 – acute lymphadenitis of unspecified type.

Causes of lymphadenitis

Lymphadenitis is a consequence of infection of the lymph node with pathogenic microorganisms, as a primary and independent disease develops

extremely rarely. Bacteria-provocateurs of pathology are: *Streptococcus spp*, *Staphylococcus spp.*, *Pseudomonas aeruginosa*, *Escherichia coli*, *Pneumococcus spp.* Lymph node increases as a result of accumulation of cells in the inflammation zone. The ingestion of microorganisms into the lymph node is also possible through the lymphatic flow from the original lesion focus. For example, as a consequence of caries, purulent rashes on the skin, furuncle, etc.

Nonspecific infection is the most common cause of compaction, growth and inflammatory response from the lymph nodes. Called conditionally by pathogenic microorganisms, lymphadenitis is characteristic for: submaxillary, cervical, ulnar, inguinal, axillary, femoral, popliteal zones. Favorable conditions for the propagation of pathogenic microorganisms will be trauma, hypothermia, stress or pain, and so on.

Lymph nodes are protective filters that prevent the penetration and reproduction of pathogenic microflora in the human body. When the level of infectious particles (elements of dead cells, microorganisms, tumor components and others) is excessively large, the lymphatic system can not cope and the inflammatory process develops. Lymphadenitis indicates the weakening of immunity in view of various factors – elderly or vice versa, a young, not strengthened organism, mental or physical overwork, previous illnesses, etc.

Do not confuse the increase in lymph nodes and the inflammatory process in their tissues. The growth of the lymph node is caused by the production of a larger number of lymphocytes, in which antibodies are produced to fight a potential threat, which in itself indicates the performance of the lymphatic system of the protective function and does not belong to pathology.

Lymphadenitis occurs when the lymph nodes become swollen and enlarged. This is often in response to bacteria, viruses or fungi in the surrounding areas of the body, like the skin, ear, nose or eye. It is often a complication of certain bacterial infections caused by bacteria like streptococcus (which causes strep throat) or staphylococcus.

In some cases, lymphadenitis is caused by other infections like tuberculosis, cat scratch disease or mono. Tuberculosis is an infectious disease that usually affects the lungs. You may experience gradually increasing painless swelling that occurs in one or more lymph nodes. In more severe cases, tuberculosis lymphadenitis can also cause systematic symptoms like fever, weight loss, fatigue and night sweats.

Cat scratch disease is a bacterial infection spread by cats infected with Bartonella henselae, bacteria that comes from flea bites or when flea droppings get into their wounds. People with cat scratch disease may

develop an enlarged lymph node in the armpit region, along with pain, redness and raised lesions in the infected area.

The Epstein-Barr virus most commonly causes mono. It can lead to swollen lymph nodes in the neck and armpits, and other **mono symptoms** like extreme fatigue, sore throat and body aches.

Pathophysiology

Increased lymph node size may be caused by the following:

- Multiplication of cells within the node, including lymphocytes, plasma cells, monocytes, or histiocytes
- Infiltration of cells from outside the node, such as malignant cells or neutrophils
- Draining of an infection (eg, abscess) into local lymph nodes.

Symptoms of lymphadenitis

The main symptom of lymphadenitis is enlarged lymph nodes. A lymph node is considered enlarged if it is about one-half inch wide.

The following symptoms of lymphadenitis are distinguished:

- nonspecific chronic inflammation is a sluggish, latent process that does not manifest itself for a long time. It is characterized by a slight swelling of the skin adjacent to the affected lymph node and a low-grade fever (37 C);
- acute lymphadenitis – has a pronounced symptomatology, namely: sharp soreness and an increase in the nodes, limiting the motor ability. Often, the condition is aggravated by a aching or dull character with a headache, general weakness, and temperature;
- the state of the purulent process is determined by a tugging, sharp pain syndrome. When palpation, the patient feels pain. The skin is red. As the disease progresses, the affected lymph nodes grow together with adjacent tissues, forming immobile seals;
- pathology of serous type – dull pain syndrome is localized in the region of regional lymph nodes, which are enlarged and are dense. For the initial stage, there is no evidence of inflammation on the skin, only after destructive processes in the tissue of the lymph node and accumulation of purulent contents, necrotic areas appear;
- adenoflegona – the stage into which purulent inflammation passes without proper therapy. Skin with signs of hyperemia, edema has blurred borders with softening foci. Among the obvious signs of pathology – high temperature, frequent heartbeats, chills, severe weakness, headache.

In severe cases, complications of untreated lymphadenitis may lead to the formation of an abscess that must be drained surgically, cellulitis (skin infection),(bloodstream infection) or fistulas that can develop with tuberculosis lymphadenitis.

The symptoms of lymphadenitis may look like other medical conditions or problems. Always see your healthcare provider for a diagnosis.

Diagnosis of lymphadenitis

Lymph nodes of a healthy person can be palpable with difficulty. It is possible to do this with a lean physique in childhood and adolescence. The growth of nodes in size is an important criterion for differentiating diseases on an early form, but signals the presence of viruses and bacteria in slow, latent processes.

The initial diagnosis of lymphadenitis involves the palpation of inflamed nodes, after which instrumental and laboratory techniques are prescribed:

- study of blood composition;
- histological analysis (taking a sample of tissue from the affected node);
- when lymphadenitis of a specific species, focus on the possibility of contact with the carrier of tuberculosis and make skin tests with laboratory testing (blood, sputum), as well as x-rays;
- purulent inflammation requires surgical opening of the capsule of the lymph node, if necessary - draining the wound;
- an increase in inguinal nodules is a signal for the exclusion of a hernia in the groin;
- the examination of children begins with suspicion of Quincke's edema, tumor formation of the cervical zone and exclusion of congenital cysts;
- often used – ultrasound, counseling ENT doctor, computed tomography, HIV test.

Blood test for lymphadenitis

Quantitative and qualitative characteristics of the composition can reveal a blood test for lymphadenitis. In addition to the general analysis, the leukoformula and the LDH (lactate dehydrogenase) level, characteristic of leukemia and lymphoma, are counted. Excess of ESR content indicates inflammatory and tumor processes. Required examination of the peripheral blood smear for the purpose of determining infectious mononucleosis.

Uric acid and transaminase (indicating hepatitis) in the analysis of blood for biochemistry are the basic criteria for systemic pathologies (autoimmune diseases, malignant neoplasms).

Taking a sample of tissue from the lymph node or fluid from inside the lymph node to study under a microscope

Submandibular lymphadenitis

In clinical practice, the most common cases of inflammation of the submaxillary lymph nodes. This pathology develops due to chronic tonsillitis, inflammation of the gums or neglected caries.Submandibular lymphadenitis is characterized by a gradual increase in symptoms. If at the first signs of pathology it is possible to determine the source of infection, then recovery comes quickly.

Inguinal lymphadenitis

Inguinal lymphadenitis is an example of a secondary inflammatory process when a pathogenic microflora enters the lymph node with a blood or lymph flow. Inguinal nodes are divided into three groups. The first - the largest – receives lymph from the buttock zone and the lower part of the peritoneum. Lymph in the second or medial group comes from the external genitalia, excretory and perineum. The latter group contains lymph of the lower extremities. The reaction of the nodes of the second group will indicate the presence of genital tract infection.

Acute lymphadenitis

The presence of an infection in the body, such as a boil, a purulent wound or a scratch, promotes the entry of bacteria into the lymphatic channel. The lymph brings the pathogenic flora to the lymph nodes, which become inflamed. So there is acute lymphadenitis, which manifests itself as a sharp, growing soreness, an increase in temperature and a worsening of the general condition.

Subacute lymphadenitis

A very rare disease – subacute lymphadenitis in clinical manifestations in many ways resembles an acute inflammatory process in the lymph nodes. Differentiate this pathology by the primary immune response. The subtype of the species is characterized by a more intense red coloration of the skin in the region of the infected lymph node, which has a dense consistency than in the acute lymphadenitis. To confirm the diagnosis, a visual examination is not enough, therefore, cytological and histological examination is used.

Cytology reveals macrophages with a large number of cellular particles and leukocytes, as well as follicular hyperplasia at the cellular level. The analysis reveals single mast cells, basophilic cells and a huge number of lymphoblasts. The histological method allows to define a sharp outline of lymphatic follicles, an increase in blood vessels filled with blood.

In subacute form, a significant increase in body temperature is possible if pus formation occurs. In other cases, the temperature is close to subfebrile.

Chronic lymphadenitis

The chronic course of lymphadenitis is a consequence of an acute process or occurs as an independent disease, bypassing the acute stage. This difference is associated with microorganisms, pathogens.

Chronic lymphadenitis is accompanied by an increase in the nodes (often painless) and the preservation of their shape. The lymph node is not connected to nearby tissues, it has a round or oval shape. There are cases when the affected tissues of the lymph node are replaced by granulation cells, often sprouting beyond the node and thinning surrounding tissues. After a while, the skin breaks, a fistula is formed. Chronic inflammation can occur with the release of a small amount of pus, oozing outward and drying up in the form of a crust.

Generalized lymphadenitis

Simultaneous inflammation of several lymph nodes or their successive lesions is a generalized lymphadenitis. A fairly rare disease is a consequence of a primary infectious process, for example, generalized tuberculosis. Often the disease manifests itself and proceeds brightly with pronounced intoxication, and also progresses rapidly. In this case, all groups of lymph nodes are significantly enlarged, the inflammation rapidly envelops nearby tissues, spreading to internal organs. The generalized form can acquire a chronic course, gradually depleting the defenses of the body.

Lymphadenitis of the face and neck

The face is the site of localization of the buccal, mandibular, chin, parotid, and also the smallest lymph nodes located near the nasolabial fold and in the inner corners of the eyes. On the neck are the chains of superficial and deep (pharyngeal) lymph nodes. The pharyngeal nodes receive lymph from the posterior parts of the nasal cavity, partly from the sky zone. In the lymph nodes of the cheeks, lower and submandibular, the chin lymph flows from: the oral cavity, the paranasal sinuses, teeth, mucous membranes, jaws, salivary glands. Diseases of these organs

contribute to the spread of infection through the lymphatic system and cause lymphadenitis of the face and neck.

Inflammation of the nodes of the submaxillary, chin and cervical zones can be odontogenic or non-dental. Odontogenic processes are characterized by a pathological relationship with the dentoalveolar system, often develop against a background of periostitis of the period of exacerbation, chronic periodontitis, acute pericoronitis. Inflammations of the lymph nodes of the non-pediatric type include otogenic, rhinogenic and dental (formed as a result of stomatitis, otitis, glossitis, gingivitis, etc.).

Lymphangitis and lymphadenitis

Secondary inflammatory process in the capillaries and trunks of the lymphatic channel is called lymphangitis. Pathology is observed in purulent-inflammatory diseases as a result of superficial (scratch, wound) or deep lesions (carbuncle, furuncle). Infectious agents in most cases are strepto-, staphylococci, but in clinical practice there are such pathogens as: protey, intestinal and tubercle bacillus and other microorganisms.

Quite often simultaneously, lymphangitis and lymphadenitis of regional type are detected accompanied by puffiness, hyperemia along the course of lymphocytes, pain syndrome, fever, chills and general weakness. Manifestations of superficial lymphangitis outwardly resemble erysipelas, along the vascular bed, seals in the form of a cord or rosary are felt. The defeat of the deep vessels of the lymphatic system is not accompanied by severe hyperemia, but the swelling is clearly visible and painful sensations remain.

For diagnostic purposes, computer thermal scanning, ultrasonic angioscanning, detection of the source of infection and excretion of the pathogen are used. In the treatment of lymphangitis an important place is occupied by the elimination of the primary foci of suppuration and the use of antibiotics. Locally apply compresses and ointment dressings, use mud treatment and X-ray treatment.

Odontogenic lymphadenitis

Acute serous inflammatory process in the submaxillary lymph nodes without the necessary treatment is transformed into a new stage, called odontogenic lymphadenitis. Lymph node – increased, pain syndrome – shooting character. Often the body temperature rises, the appetite and general condition of the patient worsen. In the case of pus formation, a person is able to acquire asymmetry due to accumulated infiltration. The skin of the skin becomes swollen with a red tinge. Palpation causes

discomfort. Acute purulent process covers nearby tissues, symptoms of intoxication appear.

The cause of pathology is dental disease. Infection of the oral cavity penetrates not only in the proximal jaws, but also in the parotid, buccal, chin, superficial and deep cervical lymph nodes. The pain of the area of the affected nodes increases with head movement. Difficulty with opening the mouth is only observed if the purulent process is spreading to the chewing muscles.

Complication of odontogenic lymphadenitis is an abscess or adenophlegmon.

Treatment of lymphadenitis

Lymphadenitis therapy primarily depends on the form of the inflammatory process (acute / chronic course, specific/non-specific nature of the lesion). At the initial stage, lymphadenitis treatment combines conservative methods with physiotherapy and gentle regimen. An important place is to eliminate the primary focus of infection: purging a purulent wound, installing a drainage system, etc. When a pathogen is identified, antibacterial therapy is prescribed. In case of purulent inflammation of the lymph node, the capsule is opened and cleaned. For chronic inflammation of the lymph nodes, the main task will be to get rid of the source of infection.

Treatment for lymphadenitis may include:

- Antibiotics given by mouth or injection to fight an infection caused by bacteria
- Medicine to control pain and fever
- Medicine to reduce swelling
- Surgery to drain a lymph node that has filled with pus

Cellulitis associated with lymphadenitis should not be treated surgically because of the risk of spreading the infection. Pus is drained only if there is an abscess and usually after the child has begun antibiotic treatment. In some cases, biopsy of an inflamed lymph node is necessary if no diagnosis has been made and no response to treatment has occurred.

Inflammation of lymph nodes due to other diseases requires treatment of the underlying causes.

Antimicrobial therapy is used when nodes are greater than 2-3 cm, are unilateral, have overlying erythema, and are tender. Antibiotics should target common infectious causes of lymphadenopathy, including *S. aureus*and GAS. Owing to the increasing prevalence of community-

acquired methicillin-resistant *S. aureus* (MRSA), empiric therapy with clindamycin should be considered. Trimethoprim-sulfamethoxazole is often effective for MRSA infection, but it is not appropriate for GAS infections.

Prevention of lymphadenitis

Prevention of acute and chronic type of inflammation of lymph nodes is the timely treatment of primary diseases: tonsillitis, tonsillitis, purulent skin lesions, osteomyelitis, rhinitis, gingivitis, boils, carbuncles and various infectious diseases (influenza, ARVI, etc.). Prevention of lymphadenitis also includes anti-caries measures: systematic visits to the dentist for the treatment of caries, stomatitis and other pathological foci of the oral cavity.

Nursing mothers are recommended to carefully monitor the hygiene of the mammary glands, not to allow the development of lactostasis. In cases of infectious diseases it is important to comply with all the prescribing doctor's instructions regarding antibacterial therapy. Do not independently reduce the period of taking antibiotics or replace the prescribed drug with another drug.

Preventive measures to prevent inflammation of the lymph nodes are the timely removal of splinters, the treatment of abrasions, microcracks and cuts with the imposition of antiseptic dressings.

Immunocorrecting programs help to increase the defenses of the body and more effectively combat the pathogenic microflora.

Prognosis of lymphadenitis

Nonspecific acute lymphadenitis at the onset of development in the provision of quality treatment often has a favorable prognosis. The destructive course of the inflammatory process terminates with the destruction of the lymph node and subsequent scarring. Progression of acute forms of lymphadenitis of the extremities provokes impaired lymphatic drainage, the formation of lymphostasis, and subsequently – to elephantiasis.

Purulent type of inflammation threatens with periadenitis (pathology extends to surrounding tissues) an abscess or phlegmon/adenophlegmon that requires prolonged treatment is formed around the melted node. The outcome of the disease is often thrombophlebitis, lymphatic fistula.

Prognosis of chronic lymphadenitis is favorable in establishing the root cause of inflammation and timely therapy. Negative consequences may be scarring, swelling of the tissue over the affected node. As a result of contraction and consolidation of the lymph node, connective cells are spreading, and lymph circulation is disturbed.

Lymphadenitis is able to pass independently in the case of timely and effective treatment of the main purulent-inflammatory disease. Therefore, at the first symptoms of inflammation of the lymph nodes it is necessary to visit the doctor and follow his prescriptions exactly.

FURUNCLES AND CARBUNCLESOF THE MAXILLOFACIAL AREA

Furuncle (boil) is purulent infection involving the hair follicle and extending to surrounding subcutaneous tissue. Furuncles can occur anywhere on hairy skin. In immunocompetent individuals, furuncles and carbuncles are usually caused by *S. aureus.*

The pathology of furuncle

A boil is an inflammatory formation having its starting point in a sebaceous-gland, sweat-gland, or hair-follicle. The core, or central slough, is composed of pus and of the tissue of the gland in which it had its origin.

A carbuncle is the coalescence of several furuncles with pus draining from multiple follicular orifices. Carbuncles are commonly associated with diabetic patients. The treatment typically involves early administration of antibiotics and surgery. Opinions on the surgical treatment are divided between saucerization, and simple incision and drainage (I&D). Despite this, there are no studies published in the English language over the last four decades that address the surgical outcome of these contrasting techniques. This case series illustrate the difference of these two surgical approaches and their result.

Clinical presentation

In the face, furuncles are frequently seen on the chin, upper lip and paranasal area. Each lesion consists of an inflammatory nodule and an overlying pustule through which hair emerges. Furuncles of the nasal vestibule can be insidious and not obvious upon cursory examination and their symptoms, namely swelling of upper lip and infiltrate of upper oral vestibule, can lead to false impression of odontogenic infection. In patients affected by a facial furuncle, fever and malaise are common. Lesions are extremely painful and they are surrounded by area of cellulitis and collateral edema.

Treatment

Small furuncles may burst and heal spontaneously. Application of moist hot dressing can promote drainage. Also gentle removal of overlying crust and necrotic central plug can be helpful; however attempts to express purulent content should be discouraged. Conservative management is preferable and only rarely cases of furuncles or carbuncles progressing into subcutaneous abscess require incision and drainage. In the face, whenever possible, this should be done through intraoral route to avoid facial scarring. Systemic antibiotics are necessary in instances of substantial collateral cellulitis, alteration of general condition and signs of developing

facial thrombophlebitis. This initial empirical therapy should be aimed at supposed staphylococcal etiology. Until recently, staphylococcal infections acquired outside of the healthcare setting have been frequently methicillin-sensitive and responsive to a wide range of antibiotics. Since 1980, methicillin-resistent staphylococcus aureus (MRSA) infections have been reported in community outbreaks. These organisms have been called community-acquired or community-associated MRSA, as opposed to hospital acquired MRSA. Hospital acquired MRSA is usually resistant to at least three β-lactam antibiotics and is usually susceptible only to vancomycin, sulfamethoxazole, and nitrofurantoin. Community acquired MRSA is more likely to be susceptible to clindamycin and has varying susceptibility to tetracycline, fluoroquinolone, erythromycin and vancomycin. Outbreaks of furunculosis may occur in families and other groups involved in close personal contact, like prisoners, members of sports teams or outdoor recreation groups. Inadequate personal hygiene and exposure to others with furuncles play important role. Control of outbreaks may require bathing with antibacterial soaps, thorough laundering of clothing, towels, bed spreads, separate use of towels and washcloths. Eradication of staphylococcal carriage among colonized persons should be attempted. The prevalence of nasal staphylococcal colonization in the general population is 20-40%, but not all carriers develop recurrent skin infections. Eradication of nasal colonization can be achieved by application of mupirocin ointment twice daily in the anterior nares for the first 5 days each month.

Carbuncle

A carbuncle is an aggregation of multiple furuncles that form an inflammatory mass. The infected necrotic centre is walled off by a pseudocapsule. This mass typically drains onto the skin surface via several openings. There is usually a rim of cellulitis and inflammation around the central necrosis. This condition is commonly associated with diabetes mellitus.

The Latin carbunculus means «small, live coal» and is related to other forms describing coal and charcoal (carbon-, carbo, carb- and carbonem). The 13th-century Old French spellings included carbuncle, charbocle, and charboncle, and later became the Middle English carbuncle.

Opinions on the surgical treatment of carbuncle are divided. One group of surgeons believes that carbuncles should be widely excised in a technique called saucerisation. This includes excision of the necrotic center and its surrounding cellulitis. The excision is deemed adequate when the limits of the surgery are healthy and completely un-inflamed. Antibiotics may not be required after saucerization. This technique results in a large wound, which is dressed and allowed to heal by secondary intention.

Occasionally, a very large wound would be closed with skin graft. Some might even require musculocutaneous flap or graft to cover the defect.

Another group of surgeons treat carbuncles by I&D, and debridement of only the necrotic centre. The surrounding inflamed tissue is not excised but is instead treated with a course of antibiotics. The resulting wound is smaller in this case. Similarly, it is dressed until it heals by secondary intention. This technique rarely requires grafting for wound cover because it heals fairly quickly. In comparison, the saucerized wound needed dressings for more than 8-weeks. Perhaps that was necessary in the era before effective antibiotics are widely available. However, we are now able to treat skin infections very effectively with various types of antibiotics.

Management

If lesions are not fluctuant (fluctuance is a wave-like feeling on palpating skin overlying a fluid-filled cavity with non-rigid walls – eg, a cavity containing pus), the application of moist heat 3-4 times daily relieves discomfort, helps to localise the infection and promotes drainage.

Treatment with oral antibiotics (until the inflammation resolves) is recommended:

1. If there is fever or surrounding cellulitis, oral antibiotics for seven days are indicated.
2. If infection occurs where complications can be dangerous (eg, the face), antibiotics should be started promptly.
3. If there is a large area of cellulitis.
4. If there is significant comorbidity – eg, diabetes or immunocompromise.
5. Oral flucloxacillin is usually the drug of choice against S. aureus, with erythromycin or clarithromycin if penicillin is contra-indicated.
6. Meticillin-resistant S. aureus (MRSA) is a growing threat in hospitals but is also being reported in the community.
7. Drainage may be spontaneous or surgical but cover the lesion with a sterile dressing to prevent autoinoculation.
8. Incision and drainage are indicated for lesions that are large, localised, painful and fluctuant.
9. Observe the patient for signs of systemic upset.

Most cases can be treated in primary care; however, the decision of whether to admit the person will depend on clinical judgement, taking into account the rapidity and degree of spread and comorbidities – eg, diabetes.

Complications

Boils and carbuncles can leave scars.

Surrounding cellulitis or bacteraemia may develop if furunculosis or carbuncles extend.

Cavernous sinus thrombosis can complicate boils or carbuncles on the face but this is rare.

Metastatic infection is rare but can include osteomyelitis, acute endocarditis or brain abscess. Septicaemia is a very rare complication of both furuncles and carbuncles.

Prognosis

Over a course of two days to three weeks the boil becomes necrotic and develops into an abscess. It ruptures and discharges pus and often a core of necrotic material. Pain subsides as pressure is reduced; the redness and oedema diminish over days to weeks.

In people who have HIV, boils may coalesce into violaceous plaques.

A carbuncle grows in size for a few days to reach a diameter of 3-10 cm, occasionally more. After 5-7 days, suppuration occurs and multiple pustules soon appear on the surface, draining externally around multiple hair follicles:

A yellow-grey irregular crater develops at the centre. In some cases the necrosis develops more acutely without a follicular discharge and the entire central core is shed to leave a deep ulcer with a purulent floor.

Healing takes place slowly by granulation and the area may remain deeply violaceous for a prolonged period of time.

Death from toxaemia or from metastatic infection may occur in the frail and the ill.

PHLEGMONS AND ABSCESSES OF THE MAXILLARY REGION

An abscess is a collection of pus that has built up within the tissue of the body.

*Phlegmon (Cellulitis)*is an acute, diffuse inflammatory infiltration of the loose connective tissue found underneath the skin. It is believed today that cellulitis and phlegmon are interchangeable terms. The term cellulitis has prevailed and so the term phlegmon has just about been abandoned.

Phlegmons and abscesses may be the result of any infectedtooth and is usually due to a mixed infection. The microorganisms thought to be responsible are aerobic and anaerobic streptococci and staphylococci.

This diseasesarecharacterized by edema, headache, and reddish skin. The edema, whose margins are diffuse and not defined, may present in various areas of the face and its localization depends on the infected tooth responsible. For example, if the mandibular posterior teeth are involved, the edema presents as submandibular, and, in more severe cases, spreads towards the cheek or the opposite side, leading to grave disfigurement of the face. When the infection originates in the maxillary anterior teeth, the edema involves the upper lip, which presents with a characteristic protrusion. In the initial stage, cellulitis feels soft or doughy during palpation, without pus present, while in more advanced stages, a board-like induration appears, which may lead to suppuration. At this stage, the pus is localized in small focal sites in the deep tissue.

Abscess of Base of Upper Lip

Anatomic Location. This abscess develops at the loose connective tissue of the base of the upper lip atthe anterior region of the maxilla, beneath the pearshaped aperture.

Etiology. It is usually caused by infected root canals of maxillary anterior teeth.

Clinical Presentation. What characterizes this infection is the swelling and protrusion of the upper lip, which is accompanied by diffuse spreading and obliteration of the depth of the mucolabial fold.

Treatment. The incision for drainage is made at the mucolabial fold parallel to the alveolar process . A hemostat is then inserted inside the cavity, which reaches bone, aiming for the apex of the responsible tooth,

facilitating the evacuation of pus. After drainage of the abscess, a rubber drain is placed until the clinical symptoms of the infection subside.

Canine Fossa Abscess

Anatomic Location. The canine fossa, which is where this type of abscess develops, is a small space between the levator labii superioris and the levator anguli oris muscles.

Etiology. Infected root canals of premolars and especially those of canines of the maxilla are considered to be responsible for the development of abscesses of the canine fossa.

Clinical Presentation. This is characterized by edema, localized in the infraorbital region, which spreads towards the medial canthus of the eye, lower eyelid, and side of the nose as far as the corner of the mouth. There is also obliteration of the nasolabial fold, and somewhat of the mucolabial fold. The edema at the infraorbital region is painful during palpation, and later on the skin becomes taut and shiny due to suppuration, while its color is reddish.

Treatment. The incision for drainage is performed intraorally at the mucobuccal fold (parallel to the alveolar bone), in the canine region. A hemostat is then inserted, which is placed at the depth of the purulent accumulation until it comes into contact with bone, while the index finger of the nondominant hand palpates the infraorbital margin. Finally, a rubber drain is placed, which is stabilizedwith a suture on the mucosa.

Buccal Space Abscess

Anatomic Location. The space in which this abscess develops is between the buccinator and masseter muscles. Superiorly, it communicates with the pterygopalatine space; inferiorly with the pterygomandibular space. The spread of pus in the buccal space depends on the position of the apices of the responsible teeth relative to the attachment of the buccinator muscle.

Etiology. The buccal space abscess may originate from infected root canals of posterior teeth of the maxilla and mandible.

Clinical Presentation. It is characterized by swelling of the cheek, which extends from the zygomatic arch as far as the inferior border of the mandible, and from the anterior border of the ramus to the corner of the mouth. The skin appears taut and red, with or without fluctuation of the, which, if neglected, may result in spontaneous drainage.

Treatment. Access to the buccal space is usually intraoral for three main reasons: because the abscess fluctuates intraorally in themajority of cases; to avoid injuring the facial nerve; for esthetic reasons.

The intraoral incision is made at the posterior region of the mouth, in an anteroposterior direction and very carefully in order to avoid injury of the parotid duct. A hemostat is then used to explore the space thoroughly. An extraoral incision is made when intraoral access would not ensure adequate drainage, or when the pus is deep inside the space. The incision is made approximately 2 cm below and parallel to the inferior border of the mandible.

Infratemporal Abscess

Anatomic Location. The space in which this abscess develops is the superior extension of the pterygomandibular space. Laterally, this space is bounded by theramus of the mandible and the temporalis muscle, while medially, it is bounded by the medial and lateral pterygoid muscles, and is continuous with the temporal fossa. Important anatomic structures, such as the mandibular nerve, mylohyoid nerve, lingual nerve, buccal nerve, chorda tympani nerve, and the maxillary artery, are found in this space. Part of the pterygoid venous plexus is also found inside thisspace.

*Etiology.*Infections of the infratemporal space may be caused by infected root canals of posterior teeth of the maxilla and mandible, by way of the pterygomandibular space, and may also be the result of a posterior superior alveolar nerve block and an inferior alveolar nerve block.

Clinical Presentation. Trismus and pain during opening of the mouth with lateral deviation towards the affected side, edema at the region anterior to th ear which extends above the zygomatic arch, as well as edema of the eyelids are observed.

Treatment. The incision for drainage of the abscess is made intraorally, at the depth of the mucobuccal fold, and, more specifically, laterally (buccally) to the maxillary third molar and medially to the coronoid process, in a superoposterior direction. A hemostat is inserted into the suppurated space, in asuperior direction. Drainage of the abscess may be performed extraorally in certain cases. The incision is performed on the skin in a superior direction, and extends approximately 3 cm. The starting point of the incision is the angle created by the junction of the frontal and temporal processes of the zygomatic bone. Drainage of the abscess is achieved with a curved hemostat, which is inserted through the skin into the purulent accumulation.

Temporal Abscess

Anatomic Location. The temporal space is the superior continuation of the infratemporal space. This space is divided into superficial and deep temporal spaces. The superficial temporal space is bounded laterally by the

temporal fascia and medially by the temporalis muscle, while the deep temporal space is found between the medial surface of the temporalis muscle and the temporal bone.

Etiology. Infection of the temporal space is caused by the spread of infection from the infratemporal space, with which it communicates.

Clinical Presentation. It is characterized by painful edema of the temporal fascia, trismus (the temporalis and medial pterygoid muscles are involved), and pain during palpation of the edema.

Treatment. The incision for drainage is performed horizontally, at the margin of the scalp hair and approximately 3 cm above the zygomatic arch. It then continues carefully between the two layers of the temporal fascia as far as the temporalis muscle. A curvedhemostat is used to drain the abscess.

PHLEGMONS AND ABSCESSES OF THE MANDIBULAR REGION

Mental Abscess

Anatomic Location. The accumulation of pus in this space is located at the anterior region of the mandible, near the bone, and, more specifically, underneath the mentalis muscle, with spread of the infection towards the symphysis menti.

Etiology. The infection is usually the result of infected mandibular anterior teeth (incisors).

Clinical Presentation. Firm and painful swelling in the area of the chin is observed, while later the skin becomes shiny and red.

Treatment. The incision for drainage of the abscessmay be performed at the depth of the mucobuccal fold,if the abscess fluctuates intraorally. If the pus hasspread extraorally, though, an incision is made onthe skin, parallel to the inferior border of the chin,1–1,5 cm posteriorly. After drainage is complete, arubber drain is placed.

Submental Abscess

Anatomic Location. The submental space in whichthis abscess develops is bounded superiorly by the mylohyoid muscle, laterally and on both sides by the anterior belly of the digastric muscle, inferiorly by the superficial layer of the deep cervical fasciathat is above the hyoid bone, and finally, by the platysma muscle and overlying skin. This space contains the anterior jugular vein and the submental lymph nodes.

Etiology. Infection of the submental space usuallyoriginates in the mandibular anterior teeth or is theresult of spread of infection from other anatomic spaces (mental, sublingual, submandibular).

Clinical Presentation. The infection presents as anindurated and painful submental edema, which latermay fluctuate or may even spread as far as the hyoid bone.

*Treatment.*After local anesthesia is performedaround the abscess, an incision on the skinis made beneath the chin, in a horizontal direction andparallel to the anterior border of the chin.

Sublingual Abscess

There are two sublingual spaces above the mylohyoidmuscle, to the right and left of the midline. Thesespaces are divided by dense fascia. Abscesses formedin these spaces are known as sublingual abscesses.

Anatomic Location. The sublingual space is bounded superiorly by the mucosa of the floor of themouth, inferiorly by the mylohyoid muscle,

anteriorlyand laterally by the inner surface of the body of themandible, medially by the lingual septum, and posteriorly by the hyoid bone.This space contains the submandibular duct(Wharton's duct), the sublingual gland, the sublingualand lingual nerve, terminal branches of the lingualartery, and part of the submandibular gland.

Etiology. The teeth that are most commonly responsible for infection of the sublingual space are the mandibular anterior teeth, premolars and the first molar, whose apices are found above the attachment of themylohyoid muscle. Also, infection may spread to thisspace from other contiguous spaces with which it communicates (submandibular, submental, lateral pharyngeal).

Clinical Presentation. The abscess of the sublingualn space presents with characteristic swelling of the mucosa of the floor of the mouth, resulting in elevation of the tongue towards the palate and laterally. The mandibular lingual sulcus is obliterated and themucosa presents a bluish tinge. The patient speakswith difficulty, because of the edema, and movementsof the tongue are painful.

Treatment. The incision for drainage is performedintraorally, laterally, and along Wharton's duct andthe lingual nerve. In order to locate thepus, a hemostat is used to explore the space inferiorly,in an anteroposterior direction and beneath the gland. After drainage is complete, a rubber drain is placed.

Submandibular Abscess

Anatomic Location. The submandibular space isbounded laterally by the inferior border of the body ofthe mandible, medially by the anterior belly of the digastric muscle, posteriorly by the stylohyoid ligament and the posterior belly of the digastric muscle, superiorly by the mylohyoid and hyoglossus muscles, and inferiorly by the superficial layer of the deep cervical fascia. This space contains the submandibular salivary gland and the submandibular lymph nodes.

Etiology. Infection of this space may originate fromthe mandibular second and third molars, if their apices are found beneath the attachment of the mylohyoid muscle. It may also be the result of spread of infection from the sublingual or submental spaces.

Clinical Presentation. The infection presents as moderate swelling at the submandibular area, which spreads, creating greater edema that is indurated and redness of the overlying skin. Also, theangle of the mandible is obliterated, while pain duringpalpation and moderate trismus due to involvement ofthe medial pterygoid muscle are observed as well.

Treatment. The incision for drainage is performedon the skin, approximately 1 cm beneath and parallelto the inferior border of the mandible. During the incision, the course of the facial artery and vein (the incision should be made posterior to these) and the respective branch of the facial nerve should be taken into consideration. A hemostat is inserted into the cavity of the abscess to explore the space and an attempt is made to communicate with the infected spaces. Blunt dissection must be performedalong the medial surface of the mandibular bone also,because pus is often located in this area as well. Afterdrainage, a rubber drain is placed.

Submasseteric Abscess

Anatomic Location. The space in which this abscessdevelops is cleft-shaped and is located between themasseter muscle and the lateral surface of the ramus ofthe mandible. Posteriorly it is bounded bythe parotid gland, and anteriorly it is bounded by the mucosa of the retromolar area.

Etiology. Infection of this space originates in themandibular third molars (pericoronitis), and in rarecases because of migratory abscesses.

Clinical Presentation. It is characterized by a firmedema that is painful to pressure in the region of themasseter muscle, which extends from the posteriorborder of the ramus of the mandible as far as the anterior border of the masseter muscle. Also, severe trismus and an inability to palpate the angle of the mandible are observed. Intraorally, there is edemapresent at the retromolar area and at the anterior border of the ramus. This abscess rarely fluctuates, while it may present generalized symptoms.

Treatment. Treatment of this abscess is basically intraoral, with an incision that begins at the coronoid process and runs along the anterior border of the ramus towards the mucobuccal fold, approximately as far as the second molar. The incision may also be performed extraorally on the skin, beneath the angle ofthe mandible. In both cases, a hemostat is inserted, which proceeds as far as the center of suppuration and until it comes into contact with bone.Because access is distant from the purulent accumulation, often it is difficult to drain the area well, resulting in frequent relapse.

Pterygomandibular Abscess

Anatomic Location. This space is bounded laterallyby the medial surface of the ramus of the mandible,medially by the medial pterygoid muscle, superiorlyby the lateral pterygoid muscle, anteriorly by the pterygomandibular raphe, and posteriorly by the parotidgland. The pterygomandibular space contains the mandibular neurovascular bundle,

lingual nerve, and part of the buccal fat pad. It communicates with the pterygopalatal, infratemporal, submandibular, and lateral pharyngeal spaces.

Etiology. An abscess of this space is caused mainly byinfection of mandibular third molars or the result ofan inferior alveolar nerve block, if the penetration siteof the needle is infected (pericoronitis).

Clinical Presentation. Severe trismus and slight extraoral edema beneath the angle of the mandible are observed. Intraorally, edema of the soft palate of the affected side is present, as is displacement of the uvula and lateral pharyngeal wall, while there is difficulty inswallowing.

Treatment. The incision for drainage is performedon the mucosa of the oral cavity and, more specifically,along the mesial temporal crest. The incision must be 1.5 cm long and 3–4 mm deep. A curved hemostat is then inserted, which proceeds posteriorly and laterally until it comes into contact with the medial surface of the ramus. The abscess is drained, permitting the evacuation of pus along the shaft of the instrument.

Lateral Pharyngeal Abscess

Anatomic Location. The lateral pharyngeal space isconical shaped, with the base facing the skull while theapex faces the carotid sheath. It is bounded by the lateral wall of the pharynx, the medial pterygoid muscle, the styloid process and the associated attached muscles and ligaments, and the parotid gland. The lateral pharyngeal space contains the internal carotid artery, the internal jugular vein with the respective lymph nodes, the glossopharyngeal nerve, hypoglossal nerve, vagus nerve, and accessory nerve. It communicates directly with the submandibular space,as well as with the brain by way of foramina of theskull.

Etiology. Infections of this space originate in the region of the third molar and are the result of spread of infection from the submandibular and pterygomandibular spaces.

Clinical Presentation. Extraoral edema at the lateralregion of the neck that may spread as far as the tragusof the ear, displacement of the pharyngeal wall, tonsiland uvula towards the midline, pain that radiates tothe ear, trismus, difficulty in swallowing, significantlyelevated temperature, and generally malaise are noted.

*Treatment.*Drainage is performed extraorally (similar to that of the submandibular abscess) with an incision 2 cm long, inferior to or posterior to the posterior part of the body of the mandible. Access is achieved using a hemostat, which, after entering the centerof the purulent collection, proceeds towards themedial surface of the mandible, to the third molararea, and if possible, behind that area. The rubberdrain that is placed remains in

position for about2–3 days. Drainage of the abscess may also be performed intraorally, although it is difficult and risky, because there is a great chance of aspiration of pus, especially if the procedure is carried out under generalanesthesia.

Retropharyngeal Abscess

Anatomic Location. The retropharyngeal space is located posterior to the soft tissue of the posterior wallof the pharynx and is bounded anteriorly by the superior pharyngeal constrictor muscle and the associatedfascia, posteriorly by the prevertebral fascia, superiorlyby the base of the skull, and inferiorly by the posteriormediastinum.

Etiology. Infections of this space originate in thelateral pharyngeal space, which is close by.

Clinical Presentation. The same symptoms as thosepresent in the lateral pharyngeal abscess appear clinically, with even greater difficulty in swallowing though, due to edema at the posterior wall of the pharynx. If it is not treated in time, there is a risk of:obstruction of the upper respiratory tract, due todisplacement of the posterior wall of the pharynxanteriorly; rupture of the abscess and aspiration of pus intothe lungs, with asphyxiation resulting; spread of infection into the mediastinum.

Treatment. Therapy entails drainage through the lateral pharyngeal space, which is where the infection usually begins. Administration of antibiotics is mandatory.

Parotid Space Abscess

Anatomic Location. The space in which this abscessdevelops is located in the area of the ramusof the mandible and, more specifically, between thelayers of the fascia investing the parotid gland. It communicates with the lateral pharyngeal and the submandibular spaces. It contains the parotid gland and its duct, the external carotid artery, the superficialtemporal and facial artery, the retromandibular vein,the auriculotemporal nerve, and the facial nerve.Etiology. Infection of this space originates fromodontogenic migratory infections of the lateral pharyngeal and submandibular spaces.

Clinical Presentation. It presents with characteristicedema of the retromandibular and parotid region, difficulty in swallowing and pain mainly during chewing, which radiates to the ear and temporal region. In certain cases there is redness of the skin and subcutaneous fluctuation. Also, a purulent exudate may be noted from the papilla of the parotid duct after pressure is applied.

Treatment. Depending on the margins of the edema,therapy entails a broad incision posterior to the angleof the mandible, taking particular care not to injure the respective branch of the facial nerve. Drainage of pus is achieved after blunt dissection using a hemostat to explore the purulent collection.

Ludwig's angina

This is a feared condition in which there is bilateral involvement of the sublingual and submandibular spaces. This disorder results in elevation of the floor of the mouth and tongue and marked oedema of the soft tissues of the neck. Ludwig's angina is most commonly the consequence of dental sepsis but may complicate submandibular gland infection or infection of a mandibular fracture or of an intraoral wound. Patients have marked systemic upset and boardlike swelling of the neck and floor of mouth. Inability to swallow their own saliva is a major indicator of deterioration. The airway is at definite risk. Various oral commensal bacteria have been implicated.

*Treatment.*The airway must be maintained and secured at an early stage. This requires intubation to allow drainage of the compressed infected region usually by fibreoptic intubation. Tracheostomy or cricothyroidotomy should be procedures of last choice because a surgical airway under these conditions creates a high risk of mediastinitis and an approximate 50% mortality rate. The involved spaces should be decompressed, even though frequently little pus is obtained. High-dose intravenous antibiotics should be given to complement surgical decompression. Any infective focus should be removed.

Relations of Deep Spaces in Infections

Space	Likely Causes	Contents	Neighboring Spaces	Approach for Space Incision and Drainage
Buccal	Upper bicuspids Upper molars Lower bicuspids	Parotid duct Ant. facial a. and v. Transverse facial a. and v. Buccal fat pad	Infraorbital Pterygo-mandibular Infratemporal	Intraoral(small) Extraoral (large).
Infraorbital	Upper cuspid	Angular a. and v. Infraorbital n.	Buccal	Intraoral
Submandibular	Lower molars	Submandibular gland	Sublingual Submental	Extraoral

		Facial a. and v. Lymph nodes	Lateral pharyngeal Buccal	
Submental	Lower anteriors Fracture of symphysis	Ant. jugular v. Lymph nodes	Submandi-bular (on either side)	Extraoral
Sublingual	Lower bicuspids Lower molars Direct trauma	Sublingual glands Wharton's ducts Lingual n. Sublingual a. and v.	Submandi-bular Lateral pharyngeal Visceral (trachea and esophagus)	Intraoral Extraoral
Pterygo-mandibular	Lower third molars Fracture of angle of mandible	Mandibular div. of trigeminal n. Inf. alveolar a. and v.	Buccal Lateral pharyngeal Submassete-ric Deep temporal Parotid Peritonsillar	Intraoral Extraoral
Submasseteric	Lower third molars Fracture of angle of mandible	Masseteric a. and v.	Buccal Pterygomand ibular Superf. temporal Parotid	Intraoral Extraoral
Infratemporal deep temporal	Upper molars	Pterygoid plexus Internal maxillary a. and v. Mandibular div. of trigeminal n. Skull base foramina	Buccal Superf. temporal Inf. petrosal sinus	Intraoral Extraoral
Superfical temporal	Upper molars Lower molars	Temporal fat pad Temporal branch of facial n.	Buccal Deep temporal	Intraoral Extraoral
Lateral pharyngeal	Lower third molars Tonsillar infection in	Carotid a. Internal jugular v. Vagus n. Cervical sympathetic chain	Pterygo-mandibular Submandi-bular Sublingual Peritonsillar	Intraoral Extraoral

	neighbo-ring spaces		Retropha-ryngeal	

ACTINOMYCOSIS OF THE MAXILLOFACIAL AREA

Actinomycosis is an infectious disease that occurs as a result of the introduction into the body of actinomycetes (ray fungi). Disease in 80-85% of cases affects the maxillofacial area.

Etiology. Activators of actinomycosis are radiant fungi. The culture of actinomycetes can be aerobic and anaerobic. In the development of actinomycosis, a significant role is played by a mixed infection – streptococcs, staphylococcs, diplococcs and other coccs, as well as anaerobic microbes – bacteroides, anaerobic streptococcs, staphylococcs, and others.

Pathogenesis. Actinomycosis occurs as a result of autoinfection, when radiant fungi penetrate the tissues of the maxillofacial region from the oral cavity (through the hood over the wisdom tooth, the gingival pocket, the gangrenous tooth, mucosal ruptures, through the damaged skin, incisions in the oral cavity), and a specific actinomycotic granuloma.

Clinical forms of actinomycosis of the face, neck, jaws and oral cavity (classification by T. Robustova):

1) cutaneous;
2) subcutaneous;
3) submucosal;
4) mucous membranes;
5) odontogenic actinomycotic granuloma;
6) subcutaneous-intermuscular (deep);
7) actinomycosis of the lymph nodes;
8) actinomycosis of the periosteum of the jaw;
9) actinomycosis of jaws;
10) actinomycosis of the oral cavity organs – tongue, tonsils, salivary glands, maxillary sinus.

Cutaneous form of actinomycosis. Complaints of minor pain and denseness in the area of the skin of the cheek, submandibular area and other areas of the neck. There is a gradual increase and consolidation of foci.There is no increase in body temperature. Objectively– inflammatory skin infiltration, one or more outbreaks, thinning of the skin, change its color from bright red to brown-blue. On the skin of the face and neck are defined pustules filled with serous or purulent fluid, or tubercles containing granulation proliferation.

The subcutaneous form is characterized by the development of a pathological process in the subcutaneous tissue, as a rule, near the odontogenic focus. Patients complain of pain and swelling in the buccal, submandibular, parotid, mandibular areas. It occurs as a result of a previous odontogenic purulent disease or against the background of pathological damage to the premaxillary or buccal lymph nodes. Define the pain in the lesion and increase the body temperature within the subfebrile. Rounded infiltration is defined in the subcutaneous tissue, initially dense and painless. During the breakdown of a specific granuloma, the skin cools with the underlying tissues, becomes bright pink to red, a softening spot appears in the center of the focus.

The submucosal form of actinomycosis occurs when the integrity of the mucous membrane of the oral cavity is damaged – foreign bodies get caught, the injury is caused by sharp edges of the teeth or by biting. It develops without a rise in body temperature. Painful sensations in the lesion are moderate. Depending on the localization of the pain intensified when moving – opening the mouth, swallowing, talking, there is a feeling of embarrassment, foreign body.

Actinomycosis of the oral mucosa. Radiant fungi penetrate through the damaged and inflamed mucosa of the oral cavity. Traumatic factors: awns of grasses, blades of grass, fish bones. Sometimes the patient traumatizes the mucous membrane with sharp edges of the teeth. The mucosa of the lower lip and cheek, the sublingual region, the lower and lateral surfaces of the tongue are more often affected. Characterized by a slow flow, not accompanied by an increase in body temperature. Pain in the lesion is insignificant. When examining a patient, a superficially located inflammatory infiltrate with a bright red mucosa is marked above, the outbreak spreads outward, thinning of the mucous membrane, formation of small fistulous passages, from which the granulations swell.

Odontogenic actinomycosis granuloma. Distinguish odontogenic actinomycotic granuloma of the skin, subcutaneous tissue, submucosal tissue, periosteum of the jaw. When the actinomycotic odontogenic granuloma is localized in the skin and subcutaneous tissue, a transient fold is observed, proceeding from the tooth to the focus in soft tissues. Odontogenic granuloma in periosteum (periosteum) of jaws is characterized by a slow asymptomatic course, differing from similar manifestations of banal etiology in that the focus under the periosteum is intimately associated with the affected periodontium. The process often

spreads to the mucosa, with the next exacerbation it becomes thinner, forming a fistulous course.

The subcutaneous-intermuscular form of actinomycosis develops in the subcutaneous, intermuscular, interfascial fiber, extends to the skin, muscles, jaw and other bones of the face. It is localized in the submandibular, buccal and parotid-chewing areas, as well as affects the tissues of the temporal, infraorbital, zygomatic regions, the metamorphosis and pterygoid palatine fossae, the pterygo-mandibular spaces and other areas of the neck. Patients indicate the appearance of swelling due to inflammatory edema and subsequent infiltration of soft tissues. Progressive limitation of mouth opening Often the first symptom of actinomycosis is. Sprouting into surrounding tissues, radiant mushrooms affect the chewing and internal pterygoid muscles, and as a result, there arises a disturbing patient restriction of opening the mouth. The spread of the actinomycosis process on the bones of the facial skeleton manifests itself in the form of a destructive lesion, localized mainly in the lower jaw. Intraosseous abscesses, gums, cortical usuras, alternating small foci of osteoporosis and osteosclerosis are detected on the roentgenogram with a secondary destructive lesion of the bones of the facial skeleton with actinomycosis.

Actinomycosis of lymph nodes occurs with odontogenic, tonsillogenic, otogenic ways of spreading the infection. The clinical picture is diverse. The process manifests itself in the form of actinomycotic lymphangitis, abscessed lymphadenitis, adenophlegmons or chronic hyperplastic lymphadenitis. With actinomycosis of the lymph nodes, the process is localized in the facial, chin, submandibular, cervical lymph nodes; they are predominantly affected by abscess or hyperplastic lymphadenitis. The clinical picture of abscessed actinomycotic lymphadenitis is characterized by complaints of a limited, slightly painful, tight knot or infiltration in the relevant area. The disease develops slowly and sluggishly, not accompanied by an increase in body temperature. The lymph node is enlarged, it gradually cools with adjacent tissues, around it infiltration of tissues grows.

Actinomycosis of the jaws. When the primary lesion of the jaws is more often localized on the lower jaw and very rarely on the upper jaw in the form of an intraosseous abscess or intraosseous gum. With an intraosseous abscess, patients complain of pain in the affected area of the bone. In the immediate vicinity of the intraosteal focus with the canal of the lower jaw, sensitivity in the chin nerve branching region is impaired. In the

future, pain becomes intense, can take the character of neuralgic. Appear edema and infiltration of soft tissues adhering to the bone or periosteal thickening of it, inflammatory contracture of the masticatory muscles develops. The clinical picture of the intraosseous gum is characterized by a slow, calm course, with minor pain in the area of the affected bone; accompanied by a separate exacerbation, in which there is an inflammatory contracture of the masticatory muscles. Inflammatory changes in the adjacent soft tissues, periosteum are expressed only slightly and appear only when the process is exacerbated.

Radiographically, the primary destructive actinomycosis of individuals is characterized by the presence in the bone of one or more merged cavities of rounded shape, which are not always clearly contoured. With the intraosseous actinomycotic gum, the focus of resorption can be surrounded by a zone of sclerosis.

Actinomycosis of the oral cavity. Actinomycosis of the tongue arises after a chronic trauma with sharp edges of the teeth, improperly made dentures, and also due to ingress of foreign bodies, including a splinter of plant origin. The clinical picture of actinomycosis of the tongue can proceed in the form of a diffuse inflammatory process reminiscent of phlegmon or abscess. In such cases, the process is localized in the root region, as well as in the lateral parts of the tongue.

Actinomycosis of the tonsils. Patients complain of a feeling of embarrassment or a foreign body sensation in the throat. In the anamnesis there are separate exacerbations of the inflammatory process. There is an increase in the affected tonsil – its compaction to a cartilaginous consistency. The tonsil covering the mucous membrane is cloudy and soldered to the underlying tissues. Characteristic is the soldering of the enlarged tonsil with the bow, especially the anterior ones.

The forms of actinomycosis of the tonsils are:

1) exudative limited and diffuse actinomycosis;
2) productive limited and diffuse actinomycosis;
3) actinomycosis of deep lymph nodes in the parotid gland.

Actinomycosis of the salivary glands can be primary and secondary. The cause of the development of the actinomycosis process in the salivary glands is the penetration of infection through the gland duct during its injury and the ingress of foreign bodies into it, but mainly the subsidence of infection in respiratory, adenoviral diseases. Often, lymph nodes are the primary focus of the development of actinomycosis.

Actinomycosis of the maxillary sinus. Infection penetrates odontogenic, rarely rhinogenic pathway. The first manifestations of the disease are more often indistinguishable from acute or exacerbation of chronic sinusitis, but the disease can develop slowly and slowly. Clinically, there are difficult nasal breathing, sometimes purulent discharge from the nose. The anterior wall of the maxilla is thickened, the mucosa of the upper arch of the vestibule of the mouth is cloudy, infiltrated and somewhat soldered with a thickened periosteum. At the next exacerbation there are inflammatory swelling in the infraorbital area, swelling of the buccal and malar areas. On the X-ray the maxillary sinus is homogeneously darkened, the walls of the cavity are well defined.

Diagnostics. The sluggish and prolonged course of odontogenic inflammatory processes, the failure of the anti-inflammatory therapy being conducted are always alarming for actinomycosis. A check is needed to identify or exclude this disease. repeated, often multiple diagnostic studies are sometimes required.

1. Microbiological study of the detachable: study of the native preparation (definition of drusen and elements of radiant fungi), cytological examination of stained smears (according to Gram, Cilda and Noht) and isolation of pathogenic culture by sowing. the isolation of anaerobic culture is the most convincing for the diagnosis of actinomycosis.
2. Pathohistological study is important for the diagnosis of actinomycosis in the clinical course of the process, simulating tumor growth, is the main for confirmation of the diagnosis.
3. X-ray studies in the primary and secondary lesions of the bones of the face with actinomycosis.
4. Sialography.
5. Clinical examination of blood and urine. In the acute course of actinomycosis, the number of leukocytes was increased to 11.1-15 15 / l, neutrophilia, lymphocytopenia, monocytopenia; ESR increased to 15-35 mm / h and above. The chronic course is characterized by leukopenia, secondary anemia, a shift of the leukocyte form to the right due to lymphocytes; ESR increased from 30 to 60 mm / h.
6. Skin and allergic reaction with actinolysate and inhibition of migration of leukocytes with various antigens: standard streptococcal, staphylococcal, actinolysate.

Treatment should be comprehensive and include:

1) surgical methods of treatment with local effect on the wound process;

2) effects on specific immunity;

3) increase of the general reactivity of the organism;

4) the effect on concomitant purulent infection;

5) anti-inflammatory, desensitizing, symptomatic therapy, treatment of common co-morbidities;

6) physical methods of treatment and exercise therapy.

Surgical treatment of actinomycosis consists of:

• the removal of teeth, which were the entrance gates of actinomycosis;

• surgical treatment of actinomycotic foci in soft and bony tissues, removal of excessively newly formed bone and lymph nodes.

Care of the wound after the opening of the actinomycotic focus. Long drainage, subsequent scraping of granulations, treatment of affected tissues with 5% tincture of iodine, the introduction of iodoform powder are necessary. When attaching secondary pyogenic infection shows the deposited administration of antibiotics, drugs of the nitrofuran series, enzymes, bacteriophages, immune preparations.

With actinomycosis characterized by normal inflammatory reaction, actinolysate therapy is performed or specially selected immunomodulators are prescribed, as well as fortifying stimulants and, in some cases, biologically active drugs.

Therapy of actinomycosis with a hypergolic inflammatory reaction starts with detoxification, general restorative and stimulating treatment. In order to remove intoxication, intravenously drip gemodeza solutions, rheopoliglyukin with the addition of vitamins.

The use of physical methods of treatment (UHF, iontophoresis, phonophoresis of medicinal substances, helium-neon laser radiation, in some cases paraffin therapy) and exercise therapy are recommended in the general treatment complex of patients with actinomycosis.

The prognosis with actinomycosis of the maxillofacial region is favorable in most cases.

Prevention. Sanitation of the oral cavity and removal of odontogenic, dental pathological foci. The main thing in the prevention of actinomycosis is an increase in the overall anti-infection protection of the body.

SYPHILIS, TUBERCULOSIS, HIV-INFECTION OF THE MAXILLOFACIAL AREA

SYPHILIS – a chronic infectious venereal disease that affects all organs and tissues, including the maxillofacial region.

*Etiology.*The causative agent of syphilis is pale treponema (spirochete), in the human body develops as an optional anaerobic and is most often localized in the lymphatic system.

*Pathogenesis.*Syphilis infection occurs sexually. Treponema enters the mucous membrane or skin, more often if their integrity is impaired. Infection can also occur not through sexual contact (household syphilis) and in utero from a mother with a syphilis (congenital syphilis).

The clinical picture

The disease has several periods:

- incubation,
- primary
- secondary
- tertiary.

The incubation period (from infection to the appearance of the first clinical symptom – chancre) lasts an average of 20-40 days. It is possible both shortening it (15-7 days), and lengthening (3-5 months).

Syphilis manifestations in the maxillofacial region are observed in the primary, secondary, and tertiary periods of the disease. Separate changes are noted in congenital syphilis.

The primary period (from the appearance of hard chancre to the appearance of the first generalized rash) lasts 6-8 weeks. Three symptoms are typical: hard chancre, regional lymphadenitis, and regional lymphangitis.

A solid chancre is erosion or an ulcer that occurs at the site of introduction of pale treponemas. In case of sexual infection, the hard chancre is located on the genitals (in men it is more often on the head of the penis or foreskin, in women – on the labia, cervix). The following features are characteristic of hard chancres: small sizes (up to a small coin), regular rounded or oval outlines, gentle (saucer-shaped) edges, smooth bluish-red bottom with scanty detachable, densely elastic (cartilaginous) infiltrate at

the base. An uncomplicated hard chancre is painless, not accompanied by acute inflammation.

Regional lymphadenitis is manifested by specific changes in the lymph nodes closest to the solid chancre (usually inguinal).

Regional lymphangitis is not always detected. It is usually detected in men (when the chancre is located on the penis) in the form of a tightly elastic mobile painless subcutaneous cord on the back and at the root of the penis.

The appearance of primary syphilis or chancre on the mucous membrane of the mouth is characteristic. The solid chancre in the oral cavity is localized on the tongue, lip in the form of an ulcer or erosion, on the gum in the form of a half moon, in the area of the tonsils – in the form of its unilateral hypertrophy. Characteristic increase in cervical and submandibular lymph nodes.

Secondary period (from the first generalized rash before the appearance of tertiary syphilides – tubercles or gum) it lasts 3-4 years, is characterized by a wavy course, an abundance and a variety of clinical symptoms. All organs and systems can be affected, however, the main manifestations are represented by a rash on the skin and mucous membranes (syphilis of the secondary period). The appearance of syphilis is due to the penetration of pale treponemas into the skin and mucous membranes. The first generalized rash, which replaces the healing solid chancre, is the most abundant (secondary, fresh syphilis), it is accompanied by severe polydenitis. The rash lasts for several weeks (less often 2-3 months), then disappears for an indefinite time. Repeated episodes of rashes (secondary recurrent syphilis) alternate with periods of complete absence of manifestations (secondary latent syphilis). The rash with secondary recurrent syphilis is less abundant, prone to grouping. In the first half of the year it is accompanied by gradually resolving polyadenitis. Syphilis of the secondary period are usually not accompanied by subjective sensations.

In the secondary period, 5 groups of syphids are distinguished:

1. syphilitic roseola: pale pink rounded peeling spots of various sizes, usually located on the body;

2. syphilitic papules: cyanotic red smooth rounded nodules of various sizes, often changing their surface depending on localization - erosive on the mucous membranes, rejuvenated on the palms and soles, vegetating in the folds of the anus and genital organs - the so-called wide condylomas;

3. syphilitic pustules: of various sizes and depths of the abscess on a dense base, turning into ulcers or purulent crusts;

4. syphilitic baldness: a rapidly developing diffuse or small focal thinning of the hair on the head without inflammatory changes in the skin;

5. syphilitic leukoderma: spotty or lace hypopigmentation of the skin of the neck.

When the mucous membrane of the oral cavity is affected, pustular or roseolous elements are determined. More often the mucous membranes of the lips, cheeks, soft palate, tonsils are affected.

A rare manifestation of syphilis in the secondary period is damage to the periosteum. It captures a significant portion of the periosteum of the jaw, often the lower one, and is characterized by a slow and sluggish course. The thickened periosteum acquires a test-like consistency, but the subperiosteal abscess does not form. Gradually, the affected areas of the periosteum are compacted, flat elevations occur.

The tertiary period of syphilis begins most often in the 3-4th year of the disease and, if untreated, lasts until the patient's life. Its manifestations are most severe, lead to an indelible disfigurement of appearance, disability and, often, death. Due to advances in diagnosis and therapy, tertiary syphilis is currently rare.

The main features of tertiary syphilis:

• wave-like course with infrequent relapses and long-term latent states;

• a plurality of delimited powerful granules prone to disintegration with long periods of their existence (months, years);

• preferential localization of lesions at the site of injury;

• a small number of pale treponemas in the tissue and, in connection with this, a slight contagiousness of tertiary syphids;

• high level of infectious allergy with low immunity;

• often negative classical serological reactions to syphilis.

Syphilis of the Tertiary period are represented by two elements: tubercles and nodes (gum).

Tubercles can be localized in the mucous membrane, periosteum and bone tissue of the jaw. With the formation of syphilitic gum, initially a dense, painless node appears, which gradually opens with the gummy rod being rejected. The resulting gummy ulcer has a crater-like shape, painless on palpation. Its edges are even, dense, the bottom is covered with granulations.

The defeat of the periosteum in the tertiary period of syphilis is characterized by diffuse, dense infiltration of the periosteum of the body of the lower, less often the upper jaw. Further, the thickened periosteum gradually fuses with the mucous membrane, and in the area of the body of

the jaw – with the skin, the gum softens and opens out with the formation of a fistula or ulcer in the center. An ulcer on the periosteum of the jaw is gradually scarring, leaving thickenings on the surface, often of a roll-like shape. When the periosteum of the alveolar bone is affected, teeth can be involved in the process, they become painful and mobile. On the x-ray, foci of osteoporosis are found in areas of the bone corresponding to the location of the tubercles in the periosteum, as well as bone loss along the surface of the cortical layer in the form of a usura. When teeth are involved in the process, the compact plate of their cells is destroyed.

Changes in bone tissue in the tertiary period of syphilis are localized in the jaw, nasal bones, and nasal septum. The process begins with a thickening of the bone, which increases as gum develops. The patient is disturbed by severe pain, sometimes a violation of sensitivity in the area of branching of the chin, sub- and infraorbital, nasal palatine nerves. Subsequently, gum grows in one or more places to the periosteum, mucous membrane, or skin. The mucous membrane or skin turns red, becomes thinner, one or several foci of softening appear, gummous foci form fistulous passages. Sequestration is not always formed, in some patients they are small. Only the attachment of a secondary pyogenic infection leads to the necrosis of more significant areas of the bone and their rejection.

In case of defeat by the gummous process of the upper jaw, in case of secondary infection, a message is formed between the oral cavity and the nasal cavity or with the maxillary sinus.

After the collapse of gumma in the bone, a gradual healing of tissues occurs with the formation of coarse, dense, often constricting scars.

Congenital syphilis

With congenital syphilis, infection occurs in utero from a sick mother. Pale treponemas penetrate the fetus from the affected placenta through the umbilical vein or the umbilical cord fissures.

There are early (up to 4 years) and late (after 4 years) congenital syphilis.

Congenital syphilis of infancy is manifested by common dystrophic and febrile symptoms, visceral and bone lesions (hepatitis, nephritis, pneumonia, osteochondritis), the presence of an abundant rash on the skin and mucous membranes, similar to syphilis of the secondary period (various types of syphilitic roseola, papules and empty. Papules on the skin often have an erosive, wet surface. In the circumference of the mouth, the papules merge into continuous exudative plaques, streaked with deep radial cracks (diffuse Hochsinger infiltration), and subsequently leave characteristic radiant scars on the skin of the lips, neck and chin (Robinson-

Fournier radial scars). Erosive papules on the nasal mucosa lead to the formation of crusts that prevent nasal breathing, so the baby cannot suckle («syphilitic runny nose»). Large pustular rashes on the palms, soles, and other parts of the limbs characterize syphilitic pemphigus.

Osteochondritis often occurs in metaphyseal sections of the tubular bones, often ending in pathological fractures (Parro pseudoparalysis).

Congenital syphilis in early childhood is characterized by a predominant lesion of the skin, mucous membranes and bones. Rashes are less plentiful, localized, similar to those with secondary recurrent syphilis (widespread warts, syphilitic alopecia are common).

Late congenital syphilis is more often manifested at the age of 5-17 years. Its main symptoms are similar to manifestations of tertiary syphilis. On the skin and mucous membranes, tubercular and gummy changes occur. However, unlike tertiary syphilis in late congenital syphilis, there is a combination of persistent signs resulting from attacks of syphilis in the chest age. Distinguish between the unconditional and probable signs of late congenital syphilis.

Unconditional (pathognomonic) signs of late congenital syphilis include only the «Hetchinson triad»: Hetchinson teeth (barrel-shaped upper middle incisors with a notch along the free edge), parenchymal keratitis and labyrinth deafness. They often occur simultaneously.

Probable symptoms can occur with other diseases.

Diagnostics and treatment

1. Wasserman reaction and other serological reactions.
2. Microbiological research
3. Pathomorphological examination of affected tissues.

Syphilis treatment is carried out in a specialized venereological hospital or clinic.

Along with the general treatment of syphilis of the oral cavity and jaw, local therapy is carried out (washing of syphilitic elements, ulcerations, fistulous passages with various antiseptic solutions, most often with 2% chloramine solution). Every 3 days, excess granulation is cauterized with a 10% chromic acid solution.

In case of syphilis lesion of the jaw bone, a periodic study of the electric excitability of the pulp of the teeth is advisable, according to the indications - trepanation of teeth with dead pulp and treatment according to the principles of treatment of chronic periodontitis.

With the development of specific periodontitis, despite significant tooth mobility, they should not be removed. According to indications, teeth

are treated with canal filling, and after a specific treatment, they are quite well strengthened.

When a secondary pyogenic infection is attached, the general and local use of drugs affecting the microbial flora is shown.

Active surgical treatment for lesions of the periosteum of the jaw with syphilis is not indicated even in the case of sequestration. They are removed after specific treatment against the background of calming down and delimitation of the process.

The prognosis for timely diagnosis, proper treatment and further follow-up is generally favorable. After curing and removing patients from the register, defects on the face, in the oral cavity, hard palate, and other localization can be resolved operatively.

TUBERCULOSIS

Pathogen is Mycobacterium tuberculosis.

Pathogenesis. A person with tuberculosis is more often a source of infection, less often the disease develops in a zoologic way through milk from sick cows. Immunity and stability of the human body are of great importance in the development of tuberculosis. There are primary and secondary tuberculosis lesions. Primary lesion of the lymph nodes of the maxillofacial area occurs when a tuberculosis infection through the teeth, tonsils, mucous membrane of the mouth and nose, the skin of the face with their inflammation or damage. Secondary tuberculosis lesions of the maxillofacial area occurs when the tuberculosis process is active, when the primary affect is in the lung, intestines, bones, and also in the generalized forms of this pathological process.

Clinical picture. In the maxillofacial region, the lesions of the skin, mucous membranes, submucous base, subcutaneous tissue, salivary glands, jaws are distinguished.

Primary tuberculosis lesion is formed in the skin, mucous membrane of the oral cavity, lymph nodes. The primary lesion of lymph nodes with tuberculosis is characterized by the appearance of single or conjugated lymph nodes. The lymph nodes are dense, in the dynamics of the disease they are even more densified, reaching the cartilaginous or bone consistency. In some patients, the lymph node or nodes disintegrate with the appearance of a characteristic curdled secret.

Secondary tuberculosis of the skin of scrofuloderm, or collicative tuberculosis, is observed mainly in children and is localized in the skin, subcutaneous fatty tissue. The tuberculosis process is formed in the immediate vicinity of the tuberculosis, in the jaws or maxillary lymph

nodes, less often in the spread of infection from more distant tuberculosis foci, for example, in the osteoarticular specific process. For scrofulous disease, the development of an infiltrate in the subcutaneous tissue in the form of separate nodes or their chain, as well as fused gummy foci is characteristic. The foci are superficially covered with atrophic, often thin skin. Gradually the process spreads towards the skin, infiltrates are soldered to it and the foci are opened out with the formation of single fistulas or ulcers, as well as their combinations. After opening the foci, a bright red or red-violet color of the affected tissues is characteristic. When separating pus, a crust forms that closes the fistula or the surface of the ulcer. After healing of tuberculous lesions, typical atrophic scars of the stellate form remain on the skin and in the subcutaneous tissue. The general condition during the course of scrofuloderm is satisfactory. The disease can last many months and years.

Secondary tuberculous lymphadenitis develops in the tuberculous process in other organs: lungs, intestines, bones, etc. It proceeds chronically and is accompanied by subfibril temperature, general weakness, loss of appetite. In some patients, the process can have an acute onset, with a sharp increase in body temperature, with separate symptoms of intoxication. Clinically, the lymph nodes increase. They have a tuberous surface, clearly contour, palpation slightly painful, and sometimes painless. In some cases, a rapid breakdown of the focus is observed, in others - a slow suppuration of the lymph node with the formation of curdled tissue decay. After the contents come out, a fistula or a few fistulas remain.

Tuberculosis of jaws arises again as a result of the spread of tuberculous mycobacteria hematogenously or lymphogenically from other organs, mainly from the respiratory and digestive organs, and also because of the contact transition from the mucous membrane of the oral cavity.

There are the next forms of tuberculosis:

a) bone damage in the primary tuberculosis complex;

b) defeat of the bone with active pulmonary tuberculosis.

Tuberculosis of the jaws is more common with lung damage. It is characterized by the formation of a single site of bone resorption, often with a pronounced periosteal reaction. On the upper jaw, it is localized in the region of the infraorbital margin or zygomatic process, on the lower jaw – in the area of its body or branch.

Diagnostics.

1. Tuberculin diagnostics – allows to establish the presence of tuberculosis infection in the body. Tuberculin solutions are used in various techniques (Mantoux, Pirke, Koch tests).

2. X-ray methods of lung examination.

3. Investigation of smears of pus from the foci, imprints of cells from ulcers.

4. Pathohistological study of tissues.

5. Vaccination of guinea pigs pathogistological material from patients (in some cases).

Differential diagnosis. Primary and secondary tuberculosis lesions of regional lymph nodes should be differentiated from abscess, lymphadenitis, chronic osteomyelitis of the jaw, actinomycosis, syphilis, and also from malignant neoplasms. Scrofuloderm is differentiated from cutaneous and subcutaneous forms of actinomycosis, a disintegrating cancerous tumor. The defeat of the bones of the jawbone, lymph nodes must be differentiated from the same processes caused by pyogenic microbes, as well as malignant neoplasms.

Treatment in a specialized TB hospital. General treatment should be complemented by local measures: hygienic maintenance and sanitation of the oral cavity, toilet ulcers. Operative interventions are performed strictly according to the indications, namely, with the clinical effect of antituberculous treatment and the delimitation of the local process in the oral cavity, in the bone tissue. Opens the intraosseous foci, scraping out of them granulation, removing sequesters, excising fistulas and suturing ulcers or refreshing their edges for tissue healing by secondary tension under the tampon of iodine gauze. Teeth with a periodontal disease affected by tuberculosis must be removed.

Prognosis with a timely and properly conducted general antituberculous treatment is favorable.

Prevention. The use of modern methods of treating tuberculosis is the main one in the prevention of tuberculosis lesions of the maxillofacial region. Caries and its complications, diseases of the mucous membrane and periodontal should be treated, hygiene of the oral cavity should be observed.

HIV INFECTION

HIV infection is an infectious disease characterized by the development of immunodeficiency and subsequent opportunistic infections. All protective reactions and nervous regulation of the human body are

affected. HIV infection is not an independent disease, but a symptom complex.

Etiology. HIV infection is caused by the human immunodeficiency virus, which belongs to retroviruses. The virus affects mainly white blood cells, monocytes-macrophages, nerve cells, which play an important role in protecting the body from infectious agents. Destroying the main protective cells of the human body, it promotes the development of immunodeficiency and various pathological processes: infections, malignant neoplasms, etc.

Pathogenesis. Spread of HIV infection:

• Sexually. Of great importance are homosexual and heterosexual contacts;

• Transmission of HIV infection to a child from an infected mother (for example, with breastfeeding);

• With intravenous injection of infected blood. The spread of HIV infection is due to this among drug addicts due to sterilization of syringes. The length of the period from infection to the onset of clinical symptoms can range from a few weeks to 6 months. and even 4-5 years. Antibodies are formed after HIV infection in the human body. In some cases, they can neutralize the virus, in others – not. The virus is in the blood, urine, semen, saliva, etc. Once in the body, the virus does not always cause HIV infection. Viruses can develop symptoms of the disease.

Clinical picture. VI stages of HIV infection are identified by VI Pokrovsky:

I stage of incubation, from the moment of infection to clinical manifestations and development of antibodies;

II stage of primary manifestations - in the form of acute infection, asymptomatic infection, generalized lymphadenopathy;

III stage of secondary manifestations;

IV terminal phase.

Symptoms of HIV infection in the oral cavity:

1. Lymphadenopathy in the head and neck (occurs in 50% of AIDS patients). Differentiate follows from syphilis, tuberculosis, infectious mononucleosis.

2. Fungal lesions of the oral mucosa. Oral candidiasis is more often detected along with a similar manifestation in the gastrointestinal tract.

3. Signs of bacterial infections in the oral cavity in the form of necrotic gingivitis, generalized periodontitis.

4. Viral infections in the oral cavity as an opportunistic infection are manifested in the form of herpetic stomatitis, hairy leukoplakia, oral deprivation, condyloma, cytome virus rash and xerostomia, recurrent aphthous ulcers, idiopathic thrombocytopenic purpura, and an increase in all salivary glands.

5. Hairy «leukoplakia with the formation along the edges of the tongue of vertical seams of a whitish color.

6. Tumors and other diseases of the salivary glands, paralysis of the muscles of the tongue, Stevenson-Johnson syndrome.

7. Pointed kandilomy of the shell of the cavity of that cavity.

8. Petechia on the mucosa of the oral cavity.

9. «Geographical language».

10. Exfoliative cheilitis.

11. Other lesions of the oral mucosa of unexplained etiology.

Diagnosis. Detection of symptoms of HIV infection in the oral cavity allows the dentist to make a preliminary diagnosis, take precautions and send the patient for examination. The following groups must be examined: persons who have had sex with AIDS patients or with seropositive, drug addicts, homo and bisexuals, patients with venereal diseases, persons with promiscuous sexual relations, persons who have been abroad for more than 1 month, donors, pregnant women, recipients.

Treatment depends on the results of the examination and is aimed at stimulating the immune system, treating systemic diseases and tumors. Supportive treatment of HIV patients allows them to prolong their life; when manifested manifestations of HIV infected – to achieve their cure. Dental treatment is symptomatic.

MAXILLARY SINUSITIS

There are four paired paranasal sinuses, which are essentially extensions of the nose. These are the frontal sinus, the sphenoid sinus, the ethmoid air cells and the maxillary sinus.

The frontal sinus is not present at birth, but it pneumatizes throughout the frontal bone in life and drains through the frontonasal duct.

The sphenoid sinus rapidly enlarges around puberty. This is the deepest of the paranasal sinuses and is of great significance because of its close relationships with dura, the pituitary fossa and the cavernous sinus.

The ethmoid sinuses are a delicate labyrinth of cavities lying between the medial walls of the orbits. These are of particular importance in maxillofacial surgery with regard to medial wall blow-out injuries of the orbit.

The maxillary sinus is the paranasal sinus of greatest significance to oral and maxillofacial surgery because it lies in direct contact with the teeth in the upper jaw. This is of most direct relevance with regard to finding symptoms of pathology that may arise in the mouth and appear to manifest in the maxillary sinus (also known as the antrum) and *vice versa*.

Virtually all paranasal sinus disease will manifest through the nose because this is the final drainage site and the site of ventilation of the sinuses. Symptoms arise as a result of obstruction of this site.

Anatomy and Physiology of the Nose and Paranasal Sinuses

The pathophysiology of sinusitis must be understood in the context of the normal anatomy and physiology of the nose and paranasal sinuses. The paranasal sinuses are formed early in development as evaginations of respiratory mucosa from the nose into the facial bones. Cavity formation begins in utero, and pneumatization continues into early adolescent life.

The ethmoid sinus develops into a bony labyrinth of 3 to 15 small air cells on each side. In contrast, the other sinus cavities develop as a single bony cavity on each side of the facial skeleton, although variations may exist. The ostium of each sinus represents the point at which outpouching initiated.

The lateral nasal wall on each side is lined by three turbinate bones designated as inferior, middle, and superior. The space under each is known as either the inferior, middle, or superior meatus, respectively. The posterior ethmoid sinuses drain into the superior meatus. The sphenoid sinus drains into an area known as the sphenoethmoidal recess, which lies at the junction of the sphenoid and ethmoid bones in the superior portion of the posterior nasal cavity.

The paranasal sinuses and the majority of the nasal cavity itself are lined with pseudostratified columnar ciliated epithelium (respiratory type). The cilia suspend a mucous blanket, which is secreted by goblet cells in the mucous membrane. The cilia propel this blanket in a predetermined direction, in a manner similar to the «mucociliary escalator» of the tracheobronchial tree. This phenomenon is important because in the paranasal sinuses cilia propel mucus toward the natural ostium. This means that in the maxillary sinus cilia must propel mucus against gravitational forces. Any surgical procedures intended to promote sinus drainage must, however, be addressed to the natural ostium.

One or more of the following local factors may create a predisposition for sinusitis:

- mechanical obstruction of mucociliary flow,
- particularly in the osteomeatal complex,
- defects in ciliary capability to propel the mucous blanket,
- abnormal quantity or quality of secretions.

A combination of these factors results in the development of sinusitis by allowing stasis of secretions, resulting in bacterial colonization and infection with associated inflammation. 4 In turn, this results in further ostial obstruction, stasis, and exacerbation of the inflammatory process. Furthermore, impairment of sinus ventilation creates acidic anaerobic conditions that cause ciliary damage and ineffective mucus clearance. 5 A variety of local and systemic disease processes may promote sinusitis by influencing mucociliary clearance at the anatomic, histologic, immunologic, and biochemical levels

Diagnosis

Endoscopy and CT

Sinus infections are typically diagnosed based on clinical criteria described previously. Symptom severity and effect on quality of life can be scored on multiple different scales. Acute sinusitis is frequently diagnosed and managed by the primary care practitioner largely based on history, but recurrent acute sinusitis, chronic sinusitis, or that which has failed medical management requires endoscopic evaluation and radiographic imaging. This is important because over two-thirds of patients who meet the criteria for rhinosinusitis have negative results on endoscopy, and over 50% have negative results on CT scans.

Sinusitis can be diagnosed regardless of symptomatic criteria if pus is noted in the middle meatus during nasal endoscopy. In patients who have

had surgical antrostomy, pus may be seen within the maxillary sinus. This can be cultured during the examination, with the results being useful in antibiotic selection.

In addition to purulence, nasal endoscopy can detect mucosal inflammation, edema, polyposis and anatomic variations such as a deviated septum. A recent study demonstrated that the findings of purulence, polyps, or mucosal edema correlate with sinusitis by CT, but anatomic variation was not a significant predictor. Also, negative endoscopy was a good predictor for CT scan results that were normal or indicated minimal disease.46 Overall, these results underscore the need for endoscopy in the diagnostic evaluation of cases other than isolated episodes of uncomplicated acute sinusitis.

Clinical signs

Sinusitis can be acute or chronic, and it is defined as the inflammation of the lining of the paranasal sinuses. This may be restricted to one sinus (e.g. maxillary sinusitis) or may occur in all sinuses, as seen in pansinusitis.

Acute sinusitis

This is usually caused by relative obstruction to the ostea of the sinuses or in the presence of a foreign body. The mechanism of the pathology is usually fairly evident in that patients either have a recent history of viral upper respiratory tract infection that has led to defunctioning of the cilia and mucosal inflammation, which results in stagnation within the sinuses and bacterial superinfection, or they have had recent dental pathology or surgery of some description that has resulted in placement of a foreign body in the maxillary sinus.

In instances of pansinusitis, aggressive emergency treatment is indicated if the frontal sinus is involved because this gives rise to very severe frontal headache, and the proximity of the brain in the anterior cranial fossa can result in frontal abscess, cerebral abscess, encephalitis and meningitis. Treatment of acute sinusitis centres on reestablishing ventilation of the sinuses by using decongestants such as xylometazoline and oxymetazoline. The classical ephedrine is less effective and more difficult to deliver. Systemic antibiotics effective against Haemophilus influenzae and Staphylococcus aureus, usually co-fluampicil or Co-amoxyclav (amoxycillin and clavulanic acid) in high doses, and symptomatic relief using analgesia and possibly steam inhalations are essential. The advantages of inhalations are dubious. Most cases of acute sinusitis resolve with this medical therapy. It is comparatively rare to have to drain the sinuses surgically. Patients who fail to respond to outpatient

management or who have orbital cellulitis or acute frontal sinusitis should be managed by hospital admission, coronal computed tomography (CT) scanning, intravenous antibiotics and drainage if required, usually endoscopically. Children can develop a rapid form of orbital cellulitis secondary to pansinusitis and also need admission and a paediatric version of the foregoing.

Chronic sinusitis

Repeated bouts of acute sinusitis or failure to treat it adequately can result in chronic sinusitis, particularly in patients with established sinonasal disease. This can manifest as facial pain. The classical pain of sinusitis is worse when the head is moved forward and gives rise to a panmaxillary toothache. The management of chronic sinusitis has been revolutionized by the advent of functional endoscopic sinus surgery, which is a specialized subdiscipline of ear, nose and throat surgery, and patients with established chronic sinusitis should be referred for assessment for this kind of treatment. The one exception to this is patients who have repeated bouts of acute sinusitis secondary to a foreign body or a persisting oroantral fistula or a retained root.

The most common cause of an oroantral communication is the extraction of single standing multirooted upper molars. Endoscopic sinus surgery with antrostomy is the technique of choice in the treatment of chronic sinusitis. The canine fossa puncture technique has been advocated, but this has an element of reinventing the wheel because this was the site of the traditional Caldwell-Luc approach.

Retained roots in the maxillary antrum

This complication is often created when a root fractures during removal of a maxillary molar tooth, and rather than carry out transalveolar removal of the root, injudicious probing in the socket forces the root up into the antrum. Many roots are found actually lying underneath the antral mucosa and not lying loose in the antrum. In this case they tend to cause local infection rather than significant sinusitis. Most of these retained roots should be removed. Other pathology found in the maxillary antrum includes the entirely innocuous antral mucocele, antroliths and tumours.

There are basically two techniques for removing foreign bodies from the antrum. One is to use the point of entry, which is usually a tooth socket.

Complications of Sinusitis

Because of the proximity of the paranasal sinuses to the eyes and brain, complications of sinusitis are divided into two broad categories: orbital and intracranial.

Infection extending into the orbit and associated soft tissues usually originates from the ethmoids and occurs through one of two mechanisms: direct extension through the orbital wall or retrograde spread through veins between the sinuses and the orbit.

Lymphatic spread is not a significant factor because lymphatics are absent in the orbit.

Preseptal cellulitis, or periorbital cellulitis, is edema and inflammation of the skin and muscle anterior to the orbital septum secondary to impairment of venous drainage from these tissues.

There are no visual symptoms, restrictions of extraocular movement, or signs of chemosis as the infection has not invaded the intraconal soft tissues. In contrast, orbital cellulitis indicates edema and inflammation of the intraconal contents resulting in ophthalmoplegia, proptosis, and chemosis secondary to obstruction of venous outflow via the ophthalmic veins.

Subperiosteal abscess is a collection of purulent material between the bony orbital wall and the orbital periosteum, usually from direct spread of acute infection in the ethmoid sinuses through the lamina papyracea. Depending on the size of the abscess and the associated mass effect, and the degree of inflammation, ocular muscles and visual acuity are variably affected. Progression of this subperiosteal process may subsequently result in an abscess of the orbital tissues. An orbital abscess may also occur with progression of orbital cellulitis. At this stage, restriction of extraocular mobility, proptosis, chemosis, and visual loss are often observed. When orbital cellulitis or subperiosteal or orbital abscesses are suspected, contrast-enhanced CT examination is necessary.

Cavernous sinus thrombosis is a grave complication that occurs from direct extension or retrograde thrombophlebitis (via the ophthalmic vein) of ethmoid or sphenoid infections. In addition to restriction of extraocular mobility, proptosis, chemosis, and visual loss, cranial neuropathies and signs of meningitis may be observed. Given the frequency of ocular findings, this entity is often categorized with the orbital complications of sinusitis, but if this or another intracranial complication is suspected, magnetic resonance imaging must be performed. Lumbar puncture may also be indicated.

Intracranial complications occur less frequently than do orbital complications and are most commonly related to the frontal or sphenoid sinuses. These complications may occur via either direct spread or

retrograde *thrombophlebitis*. Pott's puffy tumor is a collection of pus under the forehead periosteum with inflammatory changes of the overlying skin and soft tissues.

This develops secondary to the spread of infection through emissary veins into the cranial bone marrow, and thus essentially represents osteomyelitis of the frontal bone.

An epidural abscess develops from osteitis of the posterior table of the frontal sinus extending into the space between the frontal bone and the dura. Patients present with low-grade fever and worsening headache from elevated intracranial pressure.

This complication may be surprisingly indolent because there are no focal neurologic signs and examination of the cerebrospinal fluid (CSF) is often normal. In a manner analogous to the orbital abscess, subdural and brain abscesses can occur from the direct spread of an epidural abscess or from retrograde thrombophlebitis. Increased intracranial pressure is significant in these cases and may lead to herniation and death. Subdural abscess may cause septic venous thrombosis and venous infarction. Brain abscess is associated with brain necrosis.

In contrast to the above intracranial conditions, which usually arise from the frontal sinus, meningitis typically arises from infection of the ethmoid or sphenoid sinus. The typical presenting symptoms and signs are high fever, headaches, seizures, and delirium. Lumbar puncture is necessary to establish the diagnosis and obtain culture results.

Treatment

Medical Management

The principle of therapy for sinusitis is to break the cycle of impaired mucociliary clearance, stasis, infection, and inflammation. Treatment for uncomplicated acute sinusitis is primarily medical, with antibiotics representing the mainstay of therapy. In most primary care settings, it is acceptable to initiat antibiotic therapy when the criteria for acute sinusitis are met. First-line drugs for acute rhinosinusitis recommended by the Agency for Health Care Policy and Research Institute include amoxicillin and trimethoprim/ sulfamethoxazole. It has been further recommended that cephalosporins, macrolides, penicillinase-resistant penicillins, and fluoroquinolones should be reserved for failures of first-line therapy or for complications.

However, some have questioned whether, given the high incidence of pneumococcal and *H. influenzae* resistance in many areas, this graduated antibiotic response is really appropriate.

Treatment duration should be at least 10 to 14 days, and antibiotic doses must be adjusted for patient weight (in children) and for hepatorenal function, where appropriate. Recent trends have included the use of culture-directed therapy, which, at least theoretically, allows longterm cost effective management. This can be performed safely and accurately usinga middle meatal swab under endoscopic guidance.

Oral decongestants such as pseudoephedrine and topical decongestants such as phenylephrine and oxymetazoline may be useful by decreasing tissue edema by adrenergic vasoconstriction. This allows sinus ventilation and symptomatic relief. Topical decongestants must be used judiciously, however, as continuance of these medications beyond 3 to 5 days is associated with reduced duration of action and rebound vasodilation, a condition known as rhinitis medicamentosa. The roles for antihistamines and topical nasal steroids in the management of acute infections are controversial. If allergy is thought to be a significant predisposing or coexisting factor, antihistamines may be indicated.

Topical steroids, although useful in chronic rhinosinusitis, have no proven efficacy in the treatment of acute sinusitis but may have a prophylactic effect in preventing recurrent acute episodes. Oral steroids (eg, prednisone or methylprednisolone) are not typically prescribed for acute sinusitis when a significant bacterial component is expected because the immunosuppressive effects may promote the development of complications. However, oral steroids are useful in the management of acute exacerbations of chronic sinusitis to control the baseline inflammatory tendencies of the sinonasal mucosa. Nasal saline irrigations and mucolytics may have a role in the treatment of both acute and chronic sinusitis by assisting the mobilization of secretions.

Antibiotic therapy is also a major component in the treatment of chronic (and subacute) sinusitis. The principles of treatment, however, differ from those for acute sinusitis. First, the appropriate duration of therapy may be as long as 3 to 6 weeks.

Commonly employed regimens include clindamycin (150 mg PO qid) plus either trimethoprim/sulfamethoxazole or a fluoroquinolone. Amoxicillin-clavulanate and selected oral second- and thirdgeneration cephalosporins may be useful as single-agent therapy. New-generation macrolides (clarithromycin, azithromycin) and other cephalosporins may be effective, depending on culture and sensitivity results. Each antibiotic has a unique profile of toxicities and side effects that must be considered. Recent trends have included the use of antibiotic-containing irrigations and nebulized aerosols, particularly in conjunction with endoscopic sinus surgery.

Steroids are also a mainstay in the treatment of chronic sinusitis. Steroids decrease inflammation nonspecifically via a variety of mechanisms. Primarily they inhibit cell-mediated immunity by blocking lymphocyte migration and proliferation.

Eosinophil and basophil counts are reduced, 68 and the release of histamine and leukotriene from basophils is inhibited.

Also, steroids decrease both vascular permeability and the secretory activity of submucosal glands.

Topical nasal steroids are effective in reducing mucosal inflammatory changes and are considered safe for long-term use.

With initiation of the medication, symptomatic improvement is not realized until > 1 week of use.Patients must be counseled in this regard because most patients expect the immediate relief provided by topical decongestants, which cannot be used long-term without rebound vasocongestion.

Potential risks associated with nasal steroids include epistaxis and septal perforation.

The complications of systemic steroid use, although possible, are rare with topical nasal steroids. Studies have demonstrated increased risk of acute open-angle glaucoma and ocular hypertension with inhaled but not intranasal steroid use.

Suppression of the adrenocortical axis has been observed with higher-than-recommended dosages, but other studies have shown that routine daily use is not associated with axis suppression.

Oral steroid therapy can be used intermittently in patients with chronic sinusitis to manage acute exacerbations. Several different steroid compounds are available, and each has its own relative potencies and side effects. Most often either prednisone or methylprednisolone is used. Doses usually begin at 30 mg daily (or equivalent) and are tapered over 2 to 3 weeks. Tapering doses are required after 5 to 7 days of therwww apy secondary to suppression of the adrenocortical axis. Severe acute exacerbations may require higher dosages, and some patients with recalcitrant chronic rhinosinusitis may necessitate long-term steroid regimens. Often, protracted steroid courses are necessary for management of coexisting asthma in this patient population.

Systemic steroid therapy is potentially associated with serious side effects. Long-term use may result in osteopenia or osteoporosis, which may be reversible in early phases. Patients on long-term oral steroids should therefore undergo bonedensity studies regularly. Steroid use is also associated with cataracts, hyperglycemia, glaucoma, sodium retention, fat accumulation, and psychosocial changes.

Patients with chronic sinusitis with significant atopic components may be difficult to manage. The most important strategy in this population is avoidance. Antihistamine use should be limited to those with documented allergy by testing or clear allergic stigmata such as frequent sneezing or itchy watery eyes. Antihistamines may cause drying and thickening of nasal secretion resulting in impaired mucociliary flow; therefore, they must be used judiciously. A full discussion of allergy management is beyond the scope of this chapter, but it may include topical and oral steroids, antihistamines, and mast cell stabilizers. There is also mounting evidence supporting the use of immunotherapy, particularly in cases with an allergic fungal component.

Antifungal agents may also have a role in the treatment of sinusitis. Invasive forms often require intravenous therapy with amphotericin B. Use of this medication is limited by renal toxicity. Chronic sinusitis with an allergic fungal component may also be treated with antifungal agents including itraconazole (200 mg PO bid).

Topical nasal irrigation with solutions containing amphotericin B or nystatin has also been employed in the treatment of fungal sinusitis. The efficacy of these treatments is an area of active research.

Surgery

Indications for surgery include:

– acute sinusitis with a pending or evolving complication,

– chronic sinusitis that has failed maximum medical management including at least 3 weeks of broadspectrum antibiotics,

– most forms of fungal sinusitis.

In cases of complicated acute sinusitis and invasive fungal disease, surgery should be performed on an urgent or emergent basis.

In uncomplicated chronic sinusitis the goals of surgery are to eliminate mechanical obstruction of mucociliary flow, remove chronically inflamed mucosa and bone, manage/prevent complications, and rule out other disorders such as neoplasia.

The determination that «maximal medical management» has failed must be individualized.

It should be noted that the indications for surgery are more stringent in the pediatric population, for whom some advocate 3 weeks of intravenous antibiotic therapy prior to consideration of surgery.

Procedure for removing retained roots

Give additional local anaesthesia if you are continuing from immediately fracturing the tooth. Use a vasoconstrictor for haemorrhage control. Mark out a broadly based two- or three-sided flap (a three-sided flap should be used if you wish to mobilize the mucosa for a buccal advancement flap). Elevate the flap subperiosteally. Retract the flap and remove the bone with a round burr. Remove any obstructing interdental septa that will obscure your view. It is essential that an adequate view is obtained; if the root is lying underneath the antral mucosa it will be comparatively easy to see and can be picked out. If the antral mucosa has been disrupted a fairly large amount of bone will have to be removed to create an adequate visual inspection hole through the socket. If this is the case use the buccal fat pad flap and an advancement flap to close the defect because it will be significant.

The second way to remove foreign bodies is to approach the maxillary antrum via the Caldwell-Luc operation. This has the advantage of providing a wide portal of entry into the maxillary antrum.

Caldwell-Luc procedure

The classical description is to create a linear incision above the maxillary premolar teeth where the bone lining the antrum is relatively thin. This is probably a mistake because it means that the scar is placed over an area with no bony support. One way of avoiding this is to ensure that you create an approach to avoid placing the incision line over a bony defect. Raise a classical three-sided flap by using the gingival sulcus and two relieving incisions. This allows wide exposure and also creates the option of raising an osteoplastic flap (where the bone is elevated intact with the periosteum and retaining vascularity). Another classical description is to gain access to the antrum for the Caldwell-Luc procedure by using a series of hand-held trocars thrust through the thin antral wall; this is a fairly brutal technique. Another way of doing this is to use a dental fissure burr and cut a bony window as an osteoplastic flap or simply a bony window that can be lifted out. The maxillary antrum is then opened, and the entire sinus can be inspected for foreign bodies, retained roots or any other pathology that happens to be present. Generally speaking, the antral lining should be left unless it is grossly diseased, in which case it should be completely stripped out If it is necessary to strip out the antral lining completely, significant bleeding may occur into the sinus, resulting in epistaxis. It is possible to minimize this bleeding by placing a pack in the antrum either via the Caldwell-Luc procedure or by bringing it out through the nose via an intranasal antrostomy. A simple way to do that is to use

balloon catheters in the antrum. The catheters are then simply deflated the day after and are taken out.

Oroantral communication

The most common cause of an oroantral communication is the removal of a single standing maxillary molar tooth with hypercementosis. It is worthwhile in the assessment of such teeth for exodontia to obtain a radiograph to see whether the roots of the tooth are in close proximity to the antrum. This does not mean that the tooth should not be removed or even that the patient has to be referred to a local oral surgeon; you simply should be aware when removing the tooth of the high chance of creating an oroantral communication.

Established oroantral fistulae

Once the fistula is established treatment will require formal excision of the fistulous tract to gain healing both of the antral lining and of the oral lining. There are various ways of achieving this.

Patients with established oroantral fistulae will suffer from bouts of recurrent maxillary sinusitis, complain of a foul taste in their mouth and may have fluids passing from the mouth through the fistula and out the nose. The fistula can be demonstrated by asking the patient to occlude the nose and blow out through the nose; air will be seen to bubble out of the fistula. In some instances hyperplastic antral lining may prolapse out through the established fistula.

Investigations. The only investigation required is a plain radiograph to ensure that there are no retained roots in the sinus. Small fragments of multirooted teeth are the likely cause of persisting postextraction sinusitis.

Treatment of a primary oroantral communication

Treatment of a primary oroantral communication consists simply of removing the tooth and any fragments of bone. Mobilize local gingivae and primarily suture the socket. This should allow a healthy clot to form within the area of communication. There is a high likelihood that this will heal. The patient should be advised of the communication and instructed to avoid nose blowing. The patient should also be provided with a nasal decongestant such as xylometazoline or oxymetazoline for the reasons given before and a broad-spectrum antibiotic (e.g. amoxycillin 250 to 500 mg three times daily). Review the patient to ensure that this site has healed and that an oroantral fistula (a fistula has occurred where there is an epithelial lining to the communication rather than a simple communication between the antrum and the mouth) has not formed.

Treatment of established oroantral fistula

The principles of treatment are as follows:

1. Complete excision of the epithelial lined tract
2. Removal of any foreign bodies.
3. Mobilization of tissue on the oral side and closure using an incision line that is capable of healing by primary intention. This means that it must rest on healthy bone or another layer of vascularized tissue. This can be in the following three basic ways.

Buccal advancement flap (as described by Rehrmann)

This procedure uses a three-sided advancement flap of buccal mucosa that is mobilized by making an incision through the periosteum to allow the flap to be stretched without tension over the defect. It is essential that the suture line rests on sound bone; otherwise, this flap will inevitably dehisce. The disadvantages of the buccal advancement flap are a relatively shallow sulcus postoperatively and the risk of flap dehiscence because it is a single-layer closure and must be closed over bone

Buccal fat pad flap

This procedure is approached using a similar incision to the buccal advancement flap, but when the periosteum is incised, an artery clip is passed into the submucosa to identify the buccal fat pad. This is an axial fat flap that lends itself beautifully to the closure of oroantral fistulae. It can be used as an isolated flap where the vascularized fat fills the defect and is held in place with vertical mattress sutures, thus allowing the raised buccal flap to be repositioned in the buccal sulcus to maintain sulcus depth

A combination of buccal fat pad and buccal advancement flap is probably the most effective way to close an oroantral fistula. The advantage of this technique is that the vascularized buccal fat pad fills the defect below the buccal advancement flap, which provides mucosal closure and thereby creates a double layered vascularized closure that has virtually no chance of dehiscence and breakdown

Palatal finger flap

This is the third technique for closing chronic or persisting oroantral fistula where buccal advancement flaps or fat pad flaps are not appropriate. The palatal finger flap is based on the greater palatine artery. The palatal flap is a very stiff and immobile flap, although it provides extremely robust, well-vascularized tissue. The disadvantages of the palatal flap are that it leaves a raw defect on the palate that requires some form of palate

coverage because it is extremely painful for weeks afterward and it is difficult to rotate the flap into position. This flap is one of those procedures that look very straightforward when it is drawn on a diagram but in reality, because of the stiffness of the palate, is actually extremely awkward to achieve.

As with closure on initial oroantral communications, the patient should avoid nose blowing and should be provided with a nasal decongestant and an antibiotic

DIFFERENTIAL DIAGNOSIS OF INFLAMMATORY PROCESSES OF THE MAXILLOFACIAL AREA

Symptom	Periodontitis	Periostitis	Osteomielitis
Local complaints	Pain of the tooth, aching nature, increasing with biting, feeling of «grown» tooth. Sharp pulpating tooth pain continuous or with short light intervals, amplified by heat and with a horizontal position of the body, the cold for a while calms her.	Pain spontaneous, extending to the entire jaw, irradiating along the branches of the trigeminal nerve: in the ear, temple, eyes. Pain is enhanced by the influence of thermal procedures.	Pain acute, intense, diffuse, extending to a row of teeth or half of the jaw, face and head
General complaints	No	General weakness, weakness, violation of appetite and sleep, fever up to 38	Remitting body temperature, chills, headache, pain throughout the body, poor sleep, insomnia, severe weakness, sweating, lack of appetite, constipation, or diarrhea.
Patient examination: by organs:	No changes, rapid pulse, satisfactory filling, weakness	Pulse frequent, weak filling, may be arrhythmic, heart sounds deaf	Weakness, malaise. Dysfunction of the intestine

Extraoral: examination, palpation	Inflammatory reaction of regional lymph nodes mouth opening is not limited	swelling of the soft tissues of the face of a soft consistency, painless, collected in a crease; skin color is not changed paresthesia of the skin of the lower lip and chin	swelling of the soft tissues of the face with signs of infiltration of subcutaneous tissue (malar, chin areas) mouth opening is limited
Intraoral; inspection. palpation	Reddening of the mucous membrane of the gum in the area of the causative tooth. Palpation along the root in the projection of the apex of the root is painful. There is a smooth alveolar elevation. The tongue is laid, the saliva is viscous, the viscous, unpleasant odor from the mouth	Reddening and swelling of the gum, smoothness, fluctuation, tenderness along the transitional fold of the vestibule of the oral cavity or from the oral side within the boundaries of the patient and a number of located dentists.	There is significant infiltration of soft tissues. As with the vestibular, and with the oral sides of the jaw.
Radiography	There is no change in the area of the «causal» tooth	The thickening of the periosteum is determined	There is a destruction of the bone tissue, a thickening of the periosteum of the jaw
Electric pulp test	Electroexcitability of the «causal»	The electric excitability of the	Electroexcitability of a

	tooth is absent	«causal» tooth is absent Electroexcitability of teeth in the inflammation focus is reduced to 30mA	group of teeth in the focus of inflammation is reduced to 130 μA-60 μA
C.B.C.	No changes	Leukocytosis, leukocyte shift left, increased ESR	Pronounced leukocytosis, leukocyte shift to the left with the appearance of young and myelocytes; absence of eosinophils; high ESR
C.U.A.	No changes	No changes	Protein from traces to 0.3% appearance of hyaline and granular cylinders, leukocytes and red blood cells
Bacteriological Study	A mixed microflora with a predominance of staphylococci	A mixed microflora with a predominance of staphylococci	A mixed microflora with a predominance of staphylococci

Abscesses and phlegmon of the face and neck of non-pediatric origin, as well as the resulting suppuration of the regional lymph nodes differ from the osteo-phlegmon characteristic beginning. Thus, adenophlegmons are preceded by an inflammatory process in the lymph nodes. Neodontogenic phlegmons develop with suppuration of hematomas, in case of complication of salivary stone disease, «malignant» course of fu-

runcles and carbuncles. For the so-called isolated phlegmon, the outlined inflammatory reaction of the periosteum is not characteristic.

Cysts of the maxillofacial region, as they grow, cause deformation of the soft tissues and jaws. Suppuration with the characteristic signs of an acute purulent process occurs again.

Differential diagnosis of chronic osteomyelitis

Chronic odontogenic osteomyelitis of the jaw is differentiated from specific jaw lesions (actinomycosis, tuberculosis, syphilis), benign and malignant tumors.

Disease of the bone with actinomycosis can be primary and secondary. Secondary bone lesions occur as a result of the spread of a specific infection from the infiltrated maxillary soft tissue. The infiltration is usually dense. Subsequently, multiple fistulas with crumbly pus are formed. It is much more difficult to distinguish from osteomyelitis, especially its hyperplastic form, the primary actinomycosis of the jaws. Bone with actinomycosis is swollen, has the appearance of a dense faith-tenon-like tumor, inside of which cystic spaces are revealed.

For tuberculous bone lesion, slow flow (months, years), sharp soreness, pronounced lymphadenitis are characteristic. In the pathological process, other bones of the face are involved, scars are formed.

Syphilis of jaws arises as a result of gummy lesions of the bone or periosteum. In cases of transition of the process from soft tissues to the jaw, diagnosis is not difficult. More often with syphilis, the bones of the nose, the central part of the palatine processes of the upper jaw, the alveolar process in the region of the front teeth are affected. For syphilitic damage to the bones, the formation of foci of softening (necrotic form) and ossifying periostitis (hyperplastic form) is characteristic. After the rejection of sequestration on a solid sky, there is a communication between the oral cavity and the nasal cavity or the maxillary sinus. As a result of sequestration of the septum of the nose and nasal bones, a characteristic deformation is formed – the saddle nose. In the differential diagnosis of specific bone lesions, laboratory research is crucial.

Chronic osteomyelitis of the jaw should be differentiated from benign tumors and tumor-like diseases (suppurative odontogenic cyst, osteoclastoma, osteoid-osteoma, eosinophilic granuloma, etc.), as well as malignant neoplasms. The growth of benign and malignant tumors is usually painless, not accompanied by acute inflammatory phenomena, symptoms of purulent-resorptive fever, especially at the onset of the disease. For neoplasms, the periodic increase or decrease in its volume is not typical. The exception is Ewing's sarcoma, originating from the

reticular tissue of the bone marrow. This tumor has symptoms similar to those in osteomyelitis (fever, leukocytosis, local bone tenderness, soft tissue swelling, sometimes skin hyperemia). Ewing's sarcoma initially develops slowly, then rapidly progresses. For a tumor, unlike osteomyelitis, acute, subacute and chronic course, formation of sequesters is not characteristic.

An important role in the differential diagnosis of chronic osteomyelitis of the penis is given to radiography, tomography, cytological research, and if necessary, a biopsy.

Differential diagnosis of lymphadenitis

Acute lymphadenitis must be differentiated from:

- periostitis and osteomyelitis of the jaws;
- odontogenic phlegmon;
- inflammation of the salivary glands.

Chronic productive lymphadenitis must be differentiated from:

- specific lesions of lymph nodes in actinomycosis, tuberculosis, syphilis;
- dermoid and brachyogenic cysts;
- benign tumors (mixed tumor of salivary glands, fibroma, neurinoma, etc.);
- primary malignant tumors of lymph nodes and metastases in them;
- lymphatic leukemia.

SEVERE COMPLICATIONS OF THE MAXILLOFACIAL INFECTION

Thrombophlebitis of facial veins. Thrombosis of cavernous sinus

More often thrombosis of face and cavernous sinus are complications of the face furuncles and carbuncles, acute sinusit, phlegmons of temporal and pterygoid-palatal fossa.

In the thrombophlebitis pathogenesis of facial veins and cavernous sinus is important the presence of a dense net of lymph and venous facial vessels with the multiple anastomosis, connection of facial veins, nose cavity and pterygoid-palatal fossa with the eyesocket veins of cavernous sinus, lowering of the organism reactivity after catarrhal and virus diseases, microbal allergy and autoallergy during the inflammatory processes in maxilla-facial region, mechanic damage of skin pustule. The main anastomosis, connecting the deep facial veins, pterygoid plexus with eyesocket veins, veins of hard brain-tunic, with the cavernous sinus, is lower orbital vein. In the anastomosis of facial veins with the cavities of hard brain-tunic seals are absent almost. The direction of veins blood flow could change during the inflammatory processes. In usual conditions a part of blood leaded from eyesocket along the angle vein into facial. During the inflammatory process, in the region of upper lip, blood through angle vein refluxes in eyesocket veins. It is known that thrombophlebitis of facial veins appears after stripping of skin pustules by patients or accidental pustule traumatizing more often. Mechanic trauma in the pustule region is accompanied by the damage of endothelium of small blood vessels, among them veins, which promote the thrombophlebitis development.

Thrombophlebitis of facial veins

The disease is characterized by the appearance along the angle and facial vein of painful «bands» of infiltrate tissue, skin hyperemia with the cyanotic color, and development of edema long after infiltrate. Subdermal veins are enlarged, radiate. It is obvious expressed intoxication, high body temperature, rigor, general sickness, leucocytosis with left formula shift, high ESR. Take place significant hemostasis change: shortens the time of dark blood clotting, rise the content of blood fibrinogen, appears fibrinogen B fraction, rise the factor XIII activity, inhibits fibrinolysis. During the abscess of thrombosed veins and infiltrates it is performed a surgical treatment with the active pustules drain. For prevention of thrombosis of cavernous sinus, it is recommended to ligate angle or facial vein depending on process localization. More difficult face thrombophlebitis complication is cavernous sinus thrombosis, which is concerned to intracranial

complications. Clinical picture. Appears a strong headache, sharply painfulness in eyes region, general weakness, and rigor. Body temperature achieves 38-40^{o}C. To the local manifestation belong edema and hyperemia of eyelid and front skin, infiltration of arcula soft tissue, exophtalm chemosis conjuctivitis, phthalmoplegia, mydriasis, and hyperemia of eye floor. In a circumferential blood the number of leucocytes achieves 15-20. 10^{9} /l, ESR increase till 40-60 mm/h.

Mediastinitis

Mediastinitis is a life-threatening condition that carries an extremely high mortality if recognized late or treated improperly. Although long recognized as a complication of certain infectious diseases, most cases of mediastinitis are associated with cardiac surgery. This complication affects approximately 1-2% of these patients. Although small in proportional terms, the actual number of patients affected by mediastinitis is substantial. This significantly increases mortality and cost.

After years of evolution, optimal therapy for mediastinitis is more clearly understood. Future directions for research should focus on prevention, including timely antibiotic administration, sterile technique, prophylactic measures such as topical bacitracin, and meticulous hemostasis. The focus should also include more accurate methods of diagnosis during the first 14 days after surgery, when computed tomography (CT) findings are not reliable.

However, the keys to successful management remain early recognition and aggressive treatment, including sternal reopening and debridement. Further research should also focus on the optimal timing and method of wound closure and the duration of antibiotic therapy required for optimal treatment.

Anatomy. The portion of the thorax defined as the mediastinum extends from the posterior aspect of the sternum to the anterior surface of the vertebral bodies and includes the paravertebral sulci when the locations of specific mediastinal masses are defined. It is limited bilaterally by the mediastinal parietal pleura and extends from the diaphragm inferiorly to the level of the thoracic inlet superiorly.

Traditionally, the mediastinum is artificially subdivided into three compartments (anterior, middle, and posterior) for better descriptive localization of specific lesions.

When the location or origin of specific masses or neoplasms is discussed, the compartments or spaces are most commonly defined as follows:

- Anterior – the anterior compartment extends from the posterior surface of the sternum to the anterior surface of the pericardium and great vessels, and it normally contains the thymus gland, adipose tissue, and lymph nodes; the physiology of the anterior mediastinum includes the lymphatics and thymus gland
- Middle – the physiology of the middle mediastinum includes the bronchi, the heart and pericardium, the hila of both lungs, the lymph nodes, the phrenic nerves, the great vessels, and the trachea
- Posterior – the physiology of the posterior mediastinum includes the azygos vein, the descending aorta, the esophagus, the lymph nodes, the thoracic duct, and the vagus and sympathetic nerves

*Pathophysiology.*Infection from either bacterial pathogens or more atypical organisms can inflame any of the mediastinal structures, causing physiologic compromise by compression, bleeding, systemic sepsis, or a combination of these.

The origin of infection following open heart operations is not known in most patients. Some believe that the process begins as an isolated area of sternal osteomyelitis that eventually leads to sternal separation. Others hold that sternal instability is the inciting event, and bacteria then migrate into deeper tissues. Inadequate mediastinal drainage in the operating room may also contribute to the development of a deeper chest infection.

The patient's own skin flora and the bacteria in the local surgical environment are possible sources of infection as well. Because some bacterial contamination of surgical wounds is inevitable, host risk factors are likely critical in promoting an active infection.

*Etiology.*Risk factors for the development of mediastinitis in this setting include the following:

- In general, the use of pedicled bilateral internal thoracic (mammary) artery (BITA) grafts carries increased risk for mediastinitis after coronary artery bypass grafting (CABG),and this risk is even higher among patients with diabetes, thus rendering many surgeons reluctant to use BITA grafting in this subgroup of patients; however, the use of skeletonized BITA grafts may reduce the risk, and this approach could be considered for patients with and without diabetes; there is growing evidence to suggest that diabetes is not necessarily a contraindication for BITA grafting.
- Emergency surgery
- External cardiac compression (conventional cardiopulmonary resuscitation)

- Obesity (>20% of ideal body weight)
- Postoperative shock, especially when multiple blood transfusions are required
- Prolonged bypass and operating room time
- Reoperation
- Reexploration following initial surgery
- Sternal wound dehiscence
- Surgical technical factors (eg, excessive use of electrocautery, bone wax, paramedian sternotomy)

Higher body mass index, higher creatinine level, the presence of peripheral vascular disease, preoperative corticosteroid use, and ventricular assist device or transplant surgery were all associated with an increased risk of mediastinal infection; in nondiabetic patients, postoperative hyperglycemia was associated with an increased infection risk.

Additional causes include the following:

- Esophageal perforation
- Trauma, especially blunt trauma to the chest or abdomen
- Tracheobronchial perforation, due to either penetrating or blunt trauma or instrumentation during bronchoscopy
- Descending infection following surgery of the head and neck, great vessels, or vertebrae
- Progressive odontogenic infection (Ludwig angina)
- Mediastinal extension of lung infection
- Chronic fibrosing mediastinitis due to granulomatous infections
- Ultrasonorgaphy (US)-guided transbronchial needle aspiration

Microbiology. Most mediastinitis cases involve gram-positive cocci, with *Staphylococcus aureus*and *Staphylococcus epidermidis* accounting for 70-80% of cases (see the image below).Mixed gram-positive and gram-negative infections account for approximately 40% of cases. Isolated gram-negative infections are rare causes. Postoperative mediastinitis caused by *Serratia marcescens* has been reported.

Acute mediastinitis has also been reported as a complication of Epstein-Barr virus infection.

*History.*Mediastinitis manifests within a spectrum that ranges from the subacute patient to the fulminant critically ill patient who requires immediate intervention in order to prevent death.

The typical postoperative patient presents with fever, high pulse, and report symptoms suggestive of a sternal wound infection (eg, sternal instability). Approximately two thirds of patients present within 14 days following surgery. Although a delay of months is occasionally observed, signs or symptoms typically develop within 1 month of the operation. Patients may report sternal pain that has increased since surgery, drainage from the wound site, an audible click due to sternal nonunion, and progressive redness over a variable period.

Physical Examination. Vital signs generally may show tachycardia and fever. In more advanced cases of sepsis, hypotension may be present, and the patient may require large volumes of crystalloid or vasopressor medication for support.

The Hamman sign is a crunching sound heard with a stethoscope over the precordium during systole. Its presence should alert the clinician to possible mediastinitis, though its absence does not change the probability of disease.

Direct signs of sternal infection may be among the initial presenting signs or may be delayed until after the diagnosis is already considered. Sternal pain, instability, or click; local cellulitis; and drainage can all be observed.

Distinguishing between a superficial wound infection and a deeper chest infection associated with mediastinitis can be challenging. Systemic signs of sepsis strongly suggest mediastinal involvement. Local wound exploration should be utilized as a mechanism to distinguish a superficial wound infection from a deep sternal wound infection.

*Laboratory Studies.*A complete blood count (CBC) shows leukocytosis, often with a left shift on the white blood cell (WBC) count differential. The hematocrit value decreases if bleeding has occurred. The platelet count increases in the early stages of sepsis or decreases as sepsis worsens or disseminated intravascular coagulation (DIC) occurs.

Bacteremia can be observed, and blood cultures should be obtained as clinically indicated. Results from properly collected blood cultures should be reflexive in the workup when mediastinitis is considered, especially in the postoperative patient several days after cardiothoracic surgery in the presence of sepsis.

Samples of any sternal drainage should be sent for Gram stain and culture. This is helpful for establishing a diagnosis and tailoring antimicrobial therapy. At operative exploration, additional cultures should be taken to direct antibiotic therapy.

*Imaging Studies.*Delays in the diagnosis of mediastinitis greatly increase morbidity and mortality. The condition is typically recognized because of high clinical awareness in susceptible populations. Occasionally, diagnostic imaging studies, including computed tomography (CT) of the chest, can be helpful if the diagnosis is in question.Local wound exploration is the predominant method of distinguishing between superficial wound infection and deep sternal wound infection.

Chest radiography. Findings include pneumomediastinum and air-fluid levels within the mediastinum. Air-fluid levels are often best seen on lateral films. Mediastinal widening is not a reliable sign of mediastinitis, especially postoperatively.

Computed tomography. CT is more accurate for helping to identify air-fluid levels and pneumomediastinum. A CT scan may demonstrate sternal separation and substernal fluid collections. These examinations can be helpful when the diagnosis is in question or in the late postoperative period. They should not take the place of prudent wound exploration to identify a deep sternal wound infection.

The later the scans are performed after surgery, the more accurate the results. If performed after postoperative week 2, CT scans have a sensitivity and specificity of almost 100%, though most wound infections occur before this time. The specificity of CT findings is clearly time-dependent.

CT findings consistent with mediastinitis can also be found in patients without sternal wound infections after cardiac surgery for as many as 21 days following the procedure. This makes integrating CT findings with clinical data and awareness critical.

Hosokawa et al, in a study aimed at assessing CT findings in pediatric patients diagnosed with mediastinitis after cardiovascular surgery, found that sternal destruction and capsular ring enhancement were observed more in patients with mediastinitis than in those without mediastinitis.

Magnetic resonance imaging. Magnetic resonance imaging (MRI) is poorly suited as a diagnostic modality in persons with mediastinitis. Postoperatively, patients may have sternal wires, vascular clips, metallic

valves, and pacing wires that contraindicate MRI. In addition, it is difficult to perform an MRI study on an intubated, critically ill patient.

Approach Considerations. Operative exploration includes reopening the previous sternotomy and debridement of necrotic and infected tissue. The sternum is carefully separated from the ventricle bypass grafts and the aorta so as not to cause bleeding. Cultures are sent to direct antibiotic therapy.

Wound closure is usually delayed until reasonable control of infection is achieved; however, some surgeons perform closure with muscle flaps at the initial debridement, with good results. Delayed closure is usually accomplished with muscle flaps (pectoralis, rectus) and may be aided by vacuum-assisted closure.

Sterile sternal dehiscence, which is described as a sternal nonunion, is usually not treated. Occasionally, patients have abrupt separation of the sternum in close proximity to cardiac surgery, necessitating sternal reclosure. Also, some have extreme pain or cannot tolerate the clicking and discomfort of the nonunion and require sternal reclosure.

For simple sternal dehiscence (postoperative mediastinitis), great care must be taken to exclude active infection before rewiring the sternum. Surgery is seldom recommended for cases of chronic fibrosing mediastinitis unless compression of the major mediastinal structures has occurred. In cases of sternal nonunion, surgery should be deferred except when patients have extreme pain.

Medical Therapy.

Appropriate, well-directed antibiotic therapy is crucial to successful treatment of mediastinitis.

Most patients have already received prophylactic antibiotics, usually a first-generation cephalosporin. Because as many as 20% of organisms cultured from infected sternotomy sites are methicillin-resistant *S aureus* (MRSA) and because another 20% are gram-negative organisms, it is vital to institute very broad and deep antibiotic coverage that includes *Pseudomonas* species. Culture results should then guide antibiotic use; multiple regimens are available for use with patients who have mediastinitis.

Therapy is usually prolonged, ranging from weeks to months. One study suggests that 4-6 weeks of therapy is adequate for most patients.

Enteral nutritional support should be instituted immediately, with a duodenal feeding tube, if necessary. Data suggest that the use of diets formulated with various anti-inflammatory compounds to include omega-3

long-chain fatty acids and arginine provide clinically important benefits for critically ill patients with sepsis. If enteral feedings are contraindicated, hyperalimentation should be considered.

Complications. Systemic sepsis is a major complication of mediastinitis and manifests with tachycardia, hypotension, poor urine output, and other signs of poor systemic perfusion. The aim of early aggressive therapy, both surgical and medical, is to prevent this often lethal complication.

Pneumoperitoneum and pneumothorax can produce serious local problems and eventual hemodynamic compromise.

If pleural effusions become infected and develop into empyema, systemic sepsis may occur.

Severe and life-threatening bleeding from ruptured vessels or the heart itself can occur when the chest is packed and left open to await definitive closure.

SVCS and compression of critical mediastinal structures are sometimes observed with chronic fibrosing mediastinitis. Endovascular management of SVCS occurring in this setting appears to be safe and feasible. Compression of the pulmonary vessels may give rise to pulmonary hypertension.

Sepsis

Sepsis is a life-threatening syndrome usually caused by bacterial infection. Sepsis is a response of the body's immune system that results in organ dysfunction or failure.

The systemic inflammatory response syndrome (SIRS) criteria were recently replaced by the quick Sequential Organ Failure Assessment (qSOFA) in 2016, allowing for quick bedside analysis of organ dysfunction in patients with suspected or documented infection.

The qSOFA score includes a respiratory rate of 22 breaths/minute or more, systolic blood pressure of 100 mm Hg or less, and altered level of consciousness. For completeness, severe sepsis is defined as sepsis complicated by organ dysfunction.

Multiple organ dysfunction syndrome (MODS) is characterized by progressive organ dysfunction in a severely ill patient, with failure to maintain homeostasis without intervention. It is the end stage in infectious conditions (sepsis, septic shock) and noninfectious conditions (eg, SIRS due to pancreatitis). The greater the number of organ failures, the higher the mortality risk, with the greatest risk associated with respiratory failure requiring mechanical ventilation.

MODS can be classified as primary or secondary.

Primary MODS is the direct result of identifiable injury or insult with early organ dysfunction (eg, renal failure due to a nephrotoxic agent or liver failure due to a hepatotoxic agent).

Secondary MODS is organ failure that has no attributable cause and is a consequence of the host's response (eg, acute respiratory distress syndrome [ARDS] in individuals with pancreatitis).

The following parameters are used to assess individual organ dysfunction:

- Respiratory system: Partial pressure of arterial oxygen (PaO_2)/fraction of inspired oxygen (FiO_2) ratio
- Hematology: Platelet count, coagulation panel (prothrombin time and partial thromboplastin time)
- Liver: Serum bilirubin
- Renal: Serum creatinine (or urine output)
- Brain: Glasgow coma score
- Cardiovascular: Hypotension and vasopressor requirement

Septic shock is defined as sepsis with hypotension requiring vasopressor therapy to maintain a mean blood pressure of more than 65 mm Hg and a serum lactate level exceeding 2 mmol/L (18 mg/dL) after adequate fluid resuscitation. This has a greater risk of mortality and long-term morbidity.

Pseudosepsis is defined as fever, leukocytosis, and hypotension due to causes other than sepsis. Examples might include the clinical picture seen with salicylate intoxication, methamphetamine overdose, or bilateral adrenal hemorrhage.

Etiology. Sepsis can be caused by an obvious injury or infection or a more complicated etiology such as perforation, compromise, or rupture of an intra-abdominal or pelvic structure. Other etiologies can include meningitis, head and neck infections, deep neck space infections, pyelonephritis, renal abscess (intrarenal or extrarenal), acute prostatitis/prostatic abscess, severe skin or skin structure infections (eg, necrotizing fasciitis), postsurgical infections, or systemic infections such as rickettsial infection.

*Clinical Presentation.*Individuals with sepsis may present with localizing symptoms related to a specific site or source of infection or may

present with nonspecific symptoms. Individuals with nonspecific symptoms are usually acutely ill with fever and may present with or without shaking chills. Mental status may be impaired in the setting of fever or hypotension. Patients with bacteremia from any source often display an increased breathing rate resulting in respiratory alkalosis. The skin of patients with sepsis may be warm or cold, depending on the adequacy of organ and skin perfusion.

Nonspecific signs and symptoms

The history and physical examination findings are nonspecific but may suggest the likely source of the septic process and thereby help determine the appropriate antimicrobial therapy and other interventions. General signs and symptoms of sepsis may include the following:

- Fever, with or without shaking chills (temperature >38,3ºC or < 36ºC)
- Impaired mental status (in the setting of fever or hypoperfusion)
- Increased breathing rate (>20 breaths/min) resulting in respiratory alkalosis
- Warm or cold skin, depending on the adequacy of organ perfusion and dilation of the superficial skin vessels
- Hypotension requiring pressor agents to maintain systolic blood pressure above 65 mm Hg

Systemic signs and symptoms

The clinical features depicted below may provide important diagnostic clues.

Diagnosis

A diagnosis of sepsis is based on a detailed history, physical examination, laboratory and microbiology testing, and imaging studies.

Laboratory studies that may be considered include the following:

- Complete blood (CBC) count – May show elevated or low white blood cell count, anemia, and/or thrombocytopenia
- Chemistry studies, such as markers of liver or kidney injury – May suggest organ dysfunction
- Bacterial cultures – Blood cultures and site-specific cultures based on clinical suspicion (eg, wound culture, sputum culture, or urine culture)
- Stained buffy coat smears or Gram staining of peripheral blood - May be helpful in certain infections

- Urine studies (urinalysis, microscopy, urine culture)
- Certain biomarkers, such as procalcitonin and presepsin – May be useful in diagnosing early sepsis and in determining prognosis

Imaging modalities should be focused on areas of clinical concern, based on the history and physical examination, and may include the following:

- Chest radiography (to rule out pneumonia and diagnose other causes of pulmonary infiltrates)
- Chest CT scanning (to further evaluate for pneumonia or other lung pathology)
- Abdominal ultrasonography (for suspected biliary tract obstruction)
- Abdominal CT scanning or MRI (for assessing a suspected non-biliary intra-abdominal source of infection or delineating intrarenal and extrarenal pathology)
- Site-specific soft tissue imaging, including ultrasonography, CT scanning, or MRI (to assess for possible abscess, fluid collection, or necrotizing skin infection)
- Contrast-enhanced CT scanning or MRI of the brain/neck (to assess for possible masses, abscess, fluid collection, or necrotizing infection)

The following cardiac studies may be useful if cardiac involvement or disease is suspected as a cause or complication of infection:

- Electrocardiography (ECG) to evaluate for conduction abnormalities or delays or arrhythmias; pericarditis may be a cause of «pseudosepsis».
- Cardiac enzyme levels.
- Echocardiography to evaluate for structural heart disease.

Invasive diagnostic procedures that may be considered include the following:

- Thoracentesis (in patients with pleural effusion)
- Paracentesis (in patients with ascites)
- Drainage of fluid collections/abscesses
- Bronchoscopy with washing, lavage, or other invasive sampling (in patients with suspected pneumonia)

Management

Initial management may include the following:

- Inpatient admission or ICU admission for monitoring and treatment.
- Initiation of empiric antibiotic therapy, to be followed by focused treatment based on culture, laboratory, and imaging data.
- Supportive therapy as necessary to maintain organ perfusion and respiration; timely intervention with infection source control, hemodynamic stabilization, and ventilatory support.
- Transfer if requisite facilities are not available at the admitting hospital.

Tying sites of infection to specific pathogens should occur, as follows:

- Intravenous line infections: Consider broad-spectrum coverage for gram-positive organisms, especially methicillin-resistant *Staphylococcus aureus* (MRSA) (linezolid, vancomycin, or daptomycin) and gram-negative nosocomial pathogens (especially *Pseudomonas* species and other Enterobacteriaceae [piperacillin-tazobactam, carbapenems, or cefepime]), and line removal. Some of these may be *Candida* infections.
- Biliary tract infections: Typical bacterial agents include Enterobacteriaceae, gut-associated anaerobes, and *Enterococcus*. Consider carbapenems, piperacillin-tazobactam, cephalosporins, or quinolones in combination with an anaerobic agent such as metronidazole.
- Intra-abdominal and pelvic infections: Typically Enterobacteriaceae, gut-associated anaerobes, or *Enterococcus*(carbapenems, piperacillin-tazobactam, or cephalosporins or quinolones in combination with an anaerobic agent such as metronidazole)
- Urosepsis: Typically Enterobacteriaceae or *Enterococcus* (carbapenems, piperacillin-tazobactam, cephalosporins, quinolones, or aminoglycosides)
- Pneumococcal sepsis: Third-generation cephalosporins, respiratory quinolone (levofloxacin or moxifloxacin), carbapenem, or vancomycin if resistance is suspected
- Sepsis of unknown origin: Meropenem, imipenem, piperacillin-tazobactam, or tigecycline; metronidazole plus levofloxacin, cefepime, or ceftriaxone may be alternatives

Early surgical evaluation for presumed intra-abdominal or pelvic sepsis is essential. Procedures that may be warranted depend on the source of the infection, the severity of sepsis, and the patient’s clinical status, among other factors. Once an etiologic pathogen is identified, typically via

culture, narrowed antibiotic therapy against the identified pathogen is appropriate (eg, penicillin for penicillin-susceptible *Streptococcus pneumoniae*).

TREATMENT OF THE MAXILLOFACIAL INFECTION

Step 1: Determine the Severity of Infection

Within the first few minutes of the presentation of a patient with a significant odontogenic infection, the surgeon should have above. A careful history and a brief but thorough physical examination should allow the treating surgeon to determine the anatomic location, rate of progression, and the potential for airway compromise of a given infection. The host defenses, including immune system competence and the level of systemic reserves that can be called upon by the patient to maintain homeostasis, are largely determined by history. Given this initial database the surgeon must then decide upon the setting of care, which will have a great influence on the outcome.

The clinical presentation and relevant surgical anatomy of infections of the various deep fascial spaces of the head and neck have been well described in other texts.

Three major factors must be considered in determining the severity of an infection of the head and neck: anatomic location, rate of progression, and airway compromise.

Rate of Progression

Upon interviewing the patient with an infection, the surgeon can appraise the rate of progression by inquiring about the onset of swelling and pain and comparing those times to the current signs and symptoms of swelling, pain, trismus, and airway compromise. In their study of hospitalized odontogenic infections, Flynn and colleagues found that the number of days of swelling prior to admission correlated negatively with the initial severity score. This is probably because patients with more severe and rapidly progressive infections were frightened enough to seek hospital care early on.

Airway Compromise

The most frequent cause of death in reported cases of odontogenic infection is airway obstruction. Therefore, the surgeon must assess current or impending airway obstruction within the first few moments of evaluating the patient with a head and neck infection.

Complete airway obstruction is, of course, a surgical emergency. In such cases insufficient or absent air movement in spite of inspiratory efforts will be apparent.

In highly skilled hands one brief attempt at endotracheal intubation may be made, but a direct surgical approach to the airway by

cricothyroidotomy or tracheotomy is more predictably successful. In such extreme circumstances the presence of infection overlying the trachea is less important than the absence of ventilation. Therefore, infection in the region of surgical airway access is not a contraindication to an emergency cricothyroidotomy or tracheotomy.

In partial airway obstruction, abnormal breath sounds will be evident, consisting of stridor or coarse airway sounds suggestive of fluid in the upper airways. The patient may assume a special posture that straightens the airway, such as the «sniffing position» in which the head is inclined forward and the chin is elevated, as if one were sniffing a rose. Other such postures include a sitting patient with the hands or elbows on the knees and the chest inclined forward with the head thrust anterior to the shoulders, which also straightens the airway and may allow secretions to drool outward onto the floor or into a pan. Occasionally a patient with a lateral pharyngeal space infection will incline the neck toward the opposite shoulder in order to position the upper airway over the laterally deviated trachea. patient suspected of odontogenic infection.

A maximum interincisal opening that has decreased to 20 mm or less in a patient with acute pain should be considered an infection of the masticator space until proved otherwise. Infections of the pterygomandibular space are sometimes missed because trismus hinders the examiner’s view of the oropharynx. Therefore, it is important for the examiner to position the patient’s occlusal plane parallel to the plane of vision and to orient a light coaxial to that plane of view. Then the patient is asked to maximally open the mouth in spite of pain, and the tongue is depressed with a mirror or tongue blade. This should allow the examiner to get at least a glimpse of the position of the uvula and the condition of the anterior tonsillar pillars. The affected tonsillar pillar will usually be edematous and reddened, and it will displace the uvula to the opposite side. If the suspected site of infection is touched with the mirror or tongue blade, acute pain may be elicited, especially as compared to the opposite side. The patient’s report of pain should be distinguished from the gagging that is likely to occur. Various clinical tests have been proposed with the aim of predicting difficult intubation. The Mallampati test has been correlated with difficult intubation by its available to the oral and maxillofacial surgeon is the pulse oximeter.

Step 2: Evaluate Host Defenses

Immune System Compromise

Lists the medical conditions that can interfere with proper function of the immune system, which is, of course, essential to the maintenance of host defense against infection.

- Diabetes
- Steroid therapy
- Organ transplants
- Malignancy
- Chemotherapy
- Chronic renal disease
- Malnutrition
- Alcoholism
- End-stage AIDS

Diabetes is listed first because it is the most common immune-compromising disease. Diabetics have the combination of a white blood cell migration defect, which inhibits successful chemotaxis of white blood cells to the infected site from the blood stream, and a vascular defect that impairs blood flow to small vessel tissue beds, especially in end organs such as the foot. Orally, diabetics have an increased susceptibility to periodontal infections. This disease also appears to decrease host resistance to more severe odontogenic infections such as necrotizing faciitis and deep fascial space infections.

The iatrogenic use of steroids has increased over recent years with the use of these medications to treat asthma, skin conditions, autoimmune diseases, cancer, and other inflammatory conditions. Corticosteroids appear to stabilize the cell membranes of immunocompetent cells, thereby decreasing the immune response. Patients with organ transplants are often treated with corticosteroids, as well as other immunosuppressive medications such as cyclosporine and azathioprine, to suppress organ rejection reactions.

It has been postulated that every patient with malignant disease has some defect of the immune system. The mechanisms of immune compromise in malignancy are variable and not well identified, but the surgeon treating the patient with ongoing cancer should assume that there is some defect of the immune system.

Cancer chemotherapy directly suppresses the immune system along with rapidly dividing cancer cells. Therefore, all patients who have

received cancer chemotherapy within the past year should be considered immunocompromised.

Other conditions that impair immune function include malnutrition, alcoholism, and chronic renal disease.

The role of human immunodeficiency virus (HIV) infection in diminishing host resistance to odontogenic infections is somewhat unclear and paradoxical.

HIV infection first and primarily damages the T cell. On the other hand, most odontogenic infections are due to extracellular bacteria, which are attacked by B cells, the white blood cells that elaborate antibodies. Although HIV infection may damage B cells early in the course of the disease, its most devastating effects are seen on the T cells, which explains the increased rate of cancers and infections by intracellular pathogens in patients with acquired immunodeficiency syndrome (AIDS) and pre-AIDS. Although patients with HIV seropositivity may suffer a more intense and/or prolonged hospital course than other patients, HIV seropositivity does not seem to increase the incidence of severe odontogenic infection

Step 3: Decide on the setting of care

As previously stated, an elevated fever increases metabolic needs and fluid losses, which can lead to dehydration. In addition to the clinical signs of dry skin, chapped lips, loss of skin turgor, and dry mucous membranes, dehydration can be assessed in the presence of normal serum creatinine by an elevated urine specific gravity (over 1.030) or an elevated blood urea nitrogen (BUN), which indicates prerenal azotemia.

Infections in deep spaces that have a severity score of 2 or greater can hinder access to the airway for intubation by causing trismus, directly compress the airway by swelling, or threaten ital structures directly. Thus, an odontogenic infection involving the masticator space, the perimandibular spaces, or deeper spaces indicates hospital admission.

Occasionally general anesthesia is required for patient management due to inability to achieve adequate local anesthesia, the need to secure the airway, or the inability of the patient to cooperate, as in a young child. Sometimes concurrent systemic disease indicates hospital admission and may even delay surgery, as in the need to reverse warfarin anticoagulation.

Step 4: Treat Surgically

Airway Security

The dramatic reduction in the mortality of Ludwig’s angina from 54 to 10% in only 3 years, afforded by Williams and Guralnick, was made possible by their changed surgical policy of immediate establishment of

airway security by early intubation or tracheotomy, followed by aggressive and early surgical intervention.

No antibiotics were used in their patients, except sulfa drugs in some cases. In the antibiotic era mortality has been further reduced to about 4%. It is therefore apparent that immediate establishment of airway security and early aggressive surgical therapy are the most important intervention steps in the management of severe odontogenic infections. Successful airway management in difficult situations requires a team approach. Preoperatively the surgeon should communicate with the anesthesiologist to establish the airway management plan. The anesthesiologist should be interested in understanding the anatomic location of the infection, as well as its implications for airway management. The anesthesiologist will value the opportunity to see any effacement, displacement, or deviation of the airway as demonstrated on clinical examination and CT. The airway management plan should include the projected initial management, as well as secondary procedures should the initial approach fail. An infrequently used surgical technique that may aid in protecting the airway during intubation or tracheotomy is needle decompression. In this technique, under local anesthesia an abscess of the pterygomandibular, lateral pharyngeal, submandibular, or sublingual space is aspirated with a large-bore needle in order to decompress the surrounding tissues.

This maneuver may decrease the risk of abscess rupture through taut, distended oropharyngeal tissues during instrumentation of the airway. Additional benefits of this procedure are the redirection of pus drainage into the oral cavity or onto the skin, where it can easily be removed, and obtaining an excellent specimen for culture and sensitivity testing.

Surgical Drainage

In general, surgery for management of severe odontogenic infections is not difficult. Given a thorough knowledge of the anatomy of the deep fascial spaces of the head and neck, the surgeon should be able, by using appropriate anatomic landmarks, to use small incisions and blunt dissection without direct exposure and visualization of the entire infected anatomic space. Lest the surgeon crush a vital structure within the beaks of a hemostat during blunt dissection, it is crucial to insert the instrument closed, then open it at the depth of penetration, and then withdraw the instrument in the open position.

A hemostat should never be blindly closed while it is inside a surgical wound. Another important principle of surgical incision and drainage is the need to dissect a pathway for the drain that includes the locations where pus is most likely to be found.

This can be guided by the preoperative CT examination and by knowledge of the pathways that odontogenic infection is most likely to take.

The most likely pathway for odontogenic infections to enter the submandibular space is through the thin lingual plate of the mandible, which also approximates the root apices of the lower molar teeth. By exploring this location, the surgeon may find a collection of pus that would otherwise have been missed. In order to pass a drain through the submandibular space effectively, the surgeon should therefore pass a large curved hemostat from one incision upward to the medial side of the mandible and then down to the other incision.

A Penrose drain can then be grasped in the tip of the hemostat and pulled through the dissected pathway from one incision to the other, thus draining the entire submandibular space Drains should be discontinued when the drainage ceases. They may be advanced gradually or removed all at once. There is no evidence in favor of either technique.

Pus usually stops flowing from surgically drained abscesses in 24 to 72 hour s, but this process may take somewhat longer when only cellulitis has been encountered.

It should be kept in mind however that latex Penrose drains can be antigenic, and after several days they may cause exudation due to foreign body reaction alone.

Timing of Incision and Drainage

Much of the surgical literature on the management of deep fascial space infections of the head and neck advocates an expectant approach to surgical drainage of deep neck infections. The overall strategy of this approach is to use parenteral antibiotic therapy as a means of controlling, localizing, or even eradicating the soft tissue infection. Failure of the medical approach is determined by patient deterioration, impending airway compromise, and the identification of an abscess by CT or clinical examination or both. Only then is surgical drainage undertaken. The expectant approach to management of severe odontogenic infections has not been supported by empiric investigation.

The alternative strategy, successfully demonstrated by Williams and Guralnick, is the immediate establishment of airway security as necessary, and aggressive early surgical intervention. Identification of an abscess is not required before surgical intervention. The approach by Williams and Guralnick is predicated on the concept that early incision and drainage aborts the spread of infection into deeper and more critical anatomic spaces, even when it is in the cellulitis stage

Culture and Sensitivity Testing

Infections that present in the low severity anatomic spaces are not in an anatomic position that is likely to threaten the airway or vital structures. In the absence of immunologic or systemic compromise, such infections are very unlikely to become serious or life threatening. Straightforward treatments, such as removal of the involved teeth, intraoral incision and drainage, and empiric antibiotic therapy, are almost always successful.

In this setting it can be hard to justify the increased cost of routine culture and antibiotic sensitivity testing. Furthermore, since most odontogenic pathogens are slow-growing species, identification can become an expensive and time-consuming task for the microbiology laboratory. This expense is hard to justify, given the fact that at least until recently, the oral flora is routinely sensitive to penicillin. Therefore, most microbiology laboratories, when given a specimen that grows out hemolytic streptococci mixed with short, anaerobic, weakly gram-negative rods, will report the growth of normal oral flora, thus avoiding the necessity for species identification and subsequent antibiotic sensitivity testing. For these reasons routine culture and sensitivity testing for minor oral infections does not appear to be justified. When an infection involves anatomic spaces of moderate or greater severity, or when there is significant medical or immune system compromise, culture and sensitivity testing as early as possible in the course of infection is important because the final result of antibiotic sensitivity testing can be delayed for as much as 2 weeks when fastidious or antibiotic-resistant organisms are involved. Culture and sensitivity testing is also justified when the surgeon is dealing with infections that have been subjected to multiple prior courses of antibiotic therapy or in chronic infections that are recalcitrant to therapy. Immunocompromised patients also tend to harbor unusual pathogens, such as *Klebsiella pneumonia* in diabetes, methicillin-resistant *Staphylococcus aureus* in intravenous-drug abusers, and intracellular pathogens, such as mycobacteria in HIV/AIDS. In summary, culture and sensitivity testing should be performed in unusual infections, the medically and immune compromised, and certainly in all cases severe enough to require hospitalization.

Proper culture technique involves the harvesting of the specimen in a manner that minimizes contamination by normal oral or skin flora. Ideally the skin or mucosa should be prepared with antiseptic and isolated, and the culture should be obtained by aspiration from the point of maximum inflammation, where abscess is most likely to be found. If this is not possible, then at surgery a swab and culturette system can be used, although the surgeon must be careful to avoid contamination of the specimen by saliva or skin flora. Furthermore the culture transport system should be designed to maintain the viability of anaerobic organisms, which do not survive in commonly available aerobic culturette systems. Even though the surgeon may not encounter pus during aspiration attempts or surgical drainage, fluid aspirates and swab cultures of infected sites do yield valid cultures with readily interpretable results. Therefore, specimens should be sent for culture and sensitivity testing even when pus is not obtained.

Step 5: Support Medically

Medical supportive care for the patient with a severe odontogenic infection is composed of hydration, nutrition, and control of fever in all patients. Maintenance or reestablishment of electrolyte balance and the control of systemic diseases may also be a crucial part of the necessary supportive medical care for some cases, and the reader is referred to appropriate texts for a more comprehensive discussion of these matters.

Initial temperature has been shown to be a significant predictor of the length of hospital stay with severe odontogenic infections. Fever below 39,4°C is probably beneficial. Mild temperature elevations promote phagocytosis, increase blood flow to the affected area, raise the metabolic rate, and enhance antibody function. Fever can become destructive by increasing metabolic and cardiovascular demands beyond physiologic reserve capacity. Energy stores can be rapidly depleted and the loss of fluid is significantly increased.

Adequate hydration is perhaps the best method for controlling fever. Daily sensible fluid loss, consisting primarily of sweat, is increased by 250 mL per degree of fever. Insensible fluid loss, consisting mainly of evaporation from lungs and skin, is increased by 50 to 75 mL per degree of fever per day. Therefore, a 70 kg patient with a fever of 102,2°F would have a daily fluid requirement of about 3,100 mL. This would translate to a required intravenous infusion rate of approximately 130 mL per hour, assuming no oral intake and no other extraordinary fluid losses. The next approach to controlling feve is usually taken by the administration of acetaminophen or aspirin. Fevers are often exaggerated in children and decreased in the elderly. Thus, an older patient with a relatively mild elevation of temperature may have a fairly significant infection. At the same time the surgeon may wish to control fever in the elderly at a lower temperature level than in the younger patient because of a fever's increased cardiovascular and metabolic demands. Fever can be controlled or reduced by a variety of other methods when necessary. These include cool water or alcohol sponge baths, chilled drinks when practical, or even an immersion bath using tepid water.

Fever also increases metabolic demand by 5 to 8% per degree of fever per day. Therefore, it may be necessary to supplement the infected patient's oral intake, which is likely to be significantly inhibited by the local effects of the infection and surgery, by using supplementary feedings or even enteral nutrition via a feeding tube.

Step 6: Choose and Prescribe

Antibiotic Therapy

The empiric antibiotics of choice for odontogenic infections are:

1. Outpatient
   - Penicillin
   - Clindamycin

– Cephalexin (only if the penicillin allergy was not the anaphylactoid type; use caution)

Penicillin allergy:

– Clindamycin
– Moxifloxacin
– Metronidazole alone

2. Inpatient
   – Clindamycin
   – Ampicillin + metronidazole
   – Ampicillin + sulbactam

   Penicillin allergy:
   – Clindamycin
   – Third-generation cephalosporin intravenous (only if the penicillin allergy was not the anaphylactoid type)
   – Moxifloxacin (especially for *Eikenella corrodens*)
   – Metronidazole alone (if neither clindamycin nor cephalosporins can be tolerated)

Empiric antibiotic therapy is used before culture and sensitivity reports are available. Cultures should be taken in severe infections that threaten vital structures.

These antibiotic choices are separated by severity of infection.Mild or outpatient infections have been shown in a number of studies to respond well to the oral penicillins.

There was no significant difference in pain or swelling at 7 days of therapy between penicillin and various other antibiotics, including clindamycin, amoxicillin, amoxicillin-clavulanate, and cephradine, although these parameters improved more rapidly during the first 48 hours of therapy with the alternative antibiotics.

In one pediatric study pain and swelling were significantly better at 7 days with amoxicillin.In all of the above referenced studies the involved tooth or teeth were treated with extraction or root canal therapy. Incision and drainage was performed as necessary. Therefore, penicillin continues to be a highly effective antibiotic for uncomplicated odontogenic infections, owing to its low cost and low incidence of unwanted side effects.

Most resistance to penicillin that occurs among the oral pathogens is due to synthesis of β-lactamase. Approximately 25% of the strains of the *Prevotella* and *Porphyromonas* genera are able to synthesize this enzyme. β-Lactamase can also be found in some strains of *Fusobacterium* and *Streptococcus* species. Importantly, however, the oral strains of streptococci that synthesize β-lactamase are generally among the *S. mitis, S. sanguis,* and *S. salivarius* species. These species are members of the *Streptococcus viridans* group that are responsible for many cases of endocarditis.

They are not frequently found in odontogenic abscesses. *Streptococcus anginosus, S. constellatus,* and *S. intermedius* are the viridans streptococci that comprise the *Streptococcus milleri* group. The *S. milleri* group is most commonly found in odontogenic abscesses, and fortunately it remains sensitive to the natural and semisynthetic penicillins, such as penicillin V and amoxicillin.

Therefore, it is reasonable to use penicillin plus a β -lactamase inhibitor such as ampicillin-sulbactam or a penicillin plus metronidazole as alternative antibiotics for serious odontogenic infections. The penicillins and metronidazole have the advantage of crossing the blood-brain barrier when the meninges are inflamed. Clindamycin, on the other hand, does not cross the blood-brain barrier. Therefore, it is appropriate to use penicillin plus metronidazole or ampicillin-sulbactam when there is a risk of an odontogenic infection entering the cranial cavity. Few cephalosporins are able to cross the blood-brain barrier. Some thirdgeneration cephalosporins, such as ceftadizime, can do so. In addition, ceftadizime is effective against the oral streptococci and most oral anaerobes.Among the cephalosporins, therefore, ceftadizime is the alternative antibiotic of choice. A new fluoroquinolone antibiotic, moxifloxacin has great promise in the treatment of head and neck infections. Its spectrum against oral streptococci and anaerobes is excellent. Its absorption is virtually complete via either the oral or intravenous routes, and it penetrates bone readily. Therefore, this new antibiotic may become a significant addition to the oral and maxillofacial surgeon's armamentarium. Even though metronidazole is active only against obligate anaerobic bacteria, its use alone in the treatment of odontogenic infections, when combined with appropriate surgical therapy, may be effective. In one study, ornidazole, a member of the nitroimidazole family, was effective when used alone in the management of odontogenic infections. Thus, the use of metronidazole alone may be an appropriate stratagem when all of the other appropriate antibiotics are contraindicated. As with all antibiotics, the surgeon should be aware of the side effects and drug interactions of the antibi antibiotics he or she uses. Metronidazole has a disulfiram-like reaction with alcohol, and should be used with caution in pregnancy.

Step 7: Administer the Antibiotic Properly

The tissue level of antibiotics determines their effectiveness. Those tissue levels are of course dependent on the antibiotic's level in serum, through which the antibiotic must pass in order to achieve therapeutic levels in soft tissues, bone, brain, and abscess cavities. Administration of antibiotics by the oral route requires that the drug successfully navigate the vagaries of the highly acidic stomach, the chemical qualities of ingested foods, and the basic intestinal tract. Once an antibiotic is absorbed by the gastric or intestinal mucosa, it may then be subject to firstpass metabolism in the liver and subsequent excretion though the bile. Part of the excreted antibiotic may then be reabsorbed by the intestine, resulting in enterohepatic recirculation. For these reasons orally administered antibiotics achieve much lower serum levels at a slower rate than when they are injected

directly into the vascular system intravenously. Some antibiotics, however, are equally well absorbed intravenously and orally. The fluoroquinolones, such as ciprofloxacin and moxifloxacin, are the best examples of this.

For this reason the fluoroquinolones are not given intravenously unless use of the oral route is contraindicated.

The minimum inhibitory concentration (MIC) is the concentration of an antibiotic that is required to kill a given percentage of the strains of a particular species, reported as 50% or 90% of strains (MIC50 or MIC90, respectively). The effectiveness of some antibiotics is determined by the ratio of the serum concentration of the antibiotic to the MIC required to kill a particular organism. For example, with the fluoroquinolones and the aminoglycosides, if the serum concentration achieved is three to four times the MIC for the organisms involved, then maximum killing power will be achieved. These are examples of concentration-dependent antibiotics. With time-dependent antibiotics,such as the β-lactams and vancomycin, antibiotic effectiveness is determined by the duration for which the serum concentration of the antibiotic remains above the MIC. With time-dependent antibiotics, it is necessary to know the serum elimination half-life (*t*1/2) of the antibiotic in order to determine its proper dosage interval.

The dosage interval can then be designed in order to maintain the serum concentration above the MIC for at least 40% of the dosage interval. Fortunately, the mathematics involved in these calculations have already been determined by the drug manufacturer. Dosage intervals should not be changed from published guidelines by the surgeon. Nonetheless, the surgeon must be aware of the greater effectiveness of intravenous antibiotics over their oral counterparts. For example, when penicillin G is given every 4 hours intravenously, a peak serum blood level of 20 μg/mL is achieved. Since the serum elimination half-life of penicillin G is 0,5 hours, after 3 hours (6 halflives) the serum concentration will be approximately 0,3 μg/mL. Since the MIC of S*treptococcus viridans* is 0,2 μg/mL, the serum concentration of penicillin G after an intravenous dose of 2 million units will remain above the MIC90 for approximately 75% of the dosage interval. Therefore, penicillin G, 2 million units given intravenously every 4 hours, should be highly effective against the viridans group of streptococci, especially the abscessforming *S. milleri* group. By the same method the peak serum level that can be achieved with an oral dose of 500 mg of amoxicillin is 7,5 μg/mL, and its *t*1/2 is only 1,2 hours. Since amoxicillin's MIC for viridans streptococci is 2 μg/mL, the serum concentration of amoxicillin will fall below the MIC90 at approximately 2 hours after the peak serum level has been achieved, which is only 25% of the 8-hour dosage interval. Therefore, oral amoxicillin, even though it is considered by many to be a more effective antibiotic, is less likely to be effective against the viridans streptococci than intravenous penicillin G.

Step 8: Evaluate the Patient Frequently

In outpatient infections that have been treated by tooth extraction and intraoral incision and drainage, the most appropriate initial follow-up appointment is usually at 2 days postoperatively for the following reasons:

1. Usually the drainage has ceased and the drain can be discontinued at this time.
2. There is usually a discernible improvement or deterioration in signs and symptoms allowing the next treatment decisions to be made.

For odontogenic deep fascial space infections that are serious enough for hospitalization, daily clinical evaluation and wound care are required. By 2 to 3 postoperative days the clinical signs of improvement should be apparent, such as decreasing swelling, defervescence, cessation of wound drainage, declining white blood cell count, decreased malaise, and a decrease in airway swelling such that extubation can be considered. Also at this time preliminary Gram's stains and/or culture reports should be available, which may provide some guidance as to the appropriateness of the empiric antibiotic therapy.

If the above signs of clinical improvement are not apparent, then it may be necessary to begin an investigation for possible treatment failure. One of the best methods of reevaluation is the postoperative CT. A postoperative CT can identify continued airway swelling that may preclude extubation, or further spread of the infection into previously undrained anatomic spaces, or it may confirm adequate surgical drainage of all the involved anatomic spaces by the visualization of radiopaque drains in all of the involved fascial spaces.Sometimes it is difficult to determine whether the inability to extubate a patient is due to antibiotic resistance or inadequate surgical drainage. The best available clinical test for the ability to extubate in the case of upper airway swelling is the air leak test.

The air leak test is performed in the following manner in the spontaneously ventilating patient:

1. The endotracheal tube and trachea are suctioned.
2. The oxygen supply is reconnected and any coughing that was stimulated by the tracheal suctioning is allowed to subside.
3. The oropharynx and oral cavity are suctioned free of debris, hemorrhage, and secretions.
4. The cuff of the endotracheal tube is deflated while the oxygen supply is maintained.
5. After waiting for any coughing to subside, the oxygen supply is disconnected and the surgeon's thumb is placed to occlude the opening of the endotracheal tube.
6. The patient is then instructed to breathe spontaneously around the endotracheal tube, and if this can be done, a positive air leak test isobtained. If the patient cannot breathe around the occluded endotracheal tube, then a negative result is obtained, and extubation should be delayed.

Given a positive air leak test result, the best method for patient extubation involves extubation over a stylet or preferably an endotracheal tube changer. Consideration may be given to performing the extubation procedure in an operating room,where the best facilities for handling an airway emergency are available.

Summary

Severe odontogenic infections can be the most challenging cases that an oral and maxillofacial surgeon will be called on to treat. Often the patient with a severe odontogenic infection has significant systemic or immune compromise, and the constant threat of airway obstruction due to infections in the maxillofacial region raises the risk of such cases incalculably. Furthermore, the increasing rarity of these cases and the ever-changing worlds of microbiology and antibiotic therapy make staying abreast of this field difficult for the busy surgeon. Therefore, the eight steps in the treatment of severe odontogenic infections remain the fundamental guiding principles that oral and maxillofacial surgeons must use in successful management of these cases. The application of the eight steps must be thorough and the surgeon's mind must always remain open to the possibility of treatment failure, an error in initial diagnosis, antibiotic resistance, and previously undiagnosed medically compromising conditions. Although adherence to these principles cannot always guarantee a successful result, it can assure the oral and maxillofacial surgeon that he or she is practicing at the highest standard of care.

TESTS AND TASKS

TOPIC: PERIAPICAL PERIODONTITIS

Tests

1. Clinical features of periapical periodontitis?
1) pulpitis in anamnesis
2) tender to percussion
3) localised, severe throbbing pain

2. What are the causes of apical periodontitis?
1) microbial
2) trauma
3) electrical
4) chemical

3. Odontogenic microbiology:
1) normal indigenous bacteria
2) polymicrobial
3) necrotic pulp, perio pocket

4. How do most often odontogenic infections begin?
1) pulpal necrosis
2) periapical infection
3) deep pockets

5. Symptoms of infection:
1) tumor (swelling)
2) dolor (pain)
3) calor (temp)
4) rubor (red)
5) functio laesa (loss function)
6) malaise
7) all answer

6. Components of host defense:
1) local
2) humoral
3) complement
4) cellular
5) all answer

7. What are the primary microorganisms that cause odontogenic infections?

1) aerobic 25%
2) anaerobic 75%
3) no right answer

8. Are odontogenic infections usually aerobic or anaerobic?

1) both anaerobes and aerobes
2) only anaerobes
3) only aerobes

9. Histological features of periapical periodontitis?

1) granulation tissue
2) mixed population of inflammatory cells
3) inflammation of PDL around apex
4) neutrophils
5) lymphocytes and plasma cells
6) periapical fibroblasts
7) osteoclastic resorption of bone

10. Radiological features of periapical periodontitis?

1) radiolucency around apex
2) loss of lamina dura
3) widening of PDL space

11. What is the purpose of radiograph examination of periodont?

1) assess presence or absence of signs of disease
2) determine extent and nature of pathologic change
3) establish baseline for measuring effect of intervention

12. What tissues of the periodontium can be evaluated radiographically?

1) periodontal ligament space
2) lamina dura
3) alveolar bone
4) palate
5) all of them

Answers: 1 – 1, 2, 3; 2 – 1, 2, 4; 3 – 1, 2, 3; 4 – 1, 2, 3; 5 – 7; 6 – 5; 7 – 1, 2; 8 – 1; 9 – 1-7; 10 – 1, 2, 3; 11 – 1-3; 12 – 5.

Tasks

Task №1. Define Periapical Periodontitis?

Task №2. What can Acute Periapical Periodontitis lead to?

Task №3. What can Chronic Periapical Periodontitis lead to?

Task №4. Management of Periapical Periodontitis?

Task №5. What are the components of an Apical Granuloma?

Task №6. What tissues of the periodontium can be evaluated radiographically?

TOPIC:WISDOM TEETH INFECTION

Tests

1.In which clinical cases the wisdom tooth extraction is indicated?

1) carious lesion
2) in any cases
3) wrong position and inflammatory processes.

2.Can we use radiography during examination patient with impacted tooth?

1) yes
2) no.

3. Indications for third tooth removal:

1) Pain
2) Inflomation
3) Periodontal disease
4) All answers are correct.

4. Choose indications for third tooth removal:

1) Syphilitis
2) Caries
3) Ludwig's angina
4) Sinusitis.

5. Should doctor extract impacted tooth before orthodontic treatment?

1) In most cases
2) Never
3) Always

6. What research should we done on a patient with impacted tooth?

1) Sialography
2) Ultrasound
3) X-ray
4) Angiography.

7. Pericoronaritis can be managed with:

1) Antibiotics
2) Root canal treatment
3) Osteotomy

8. Is general condition of the patient with pericoronaritis broken?

1) yes
2) no.

9. All patients having third molars (particularly lower) removed will have:

1) Some pain or discomfort
2) Root canal treatment
3) Abscess.

10. At what age do third molars extracted more often?

1) 3-7 years
2) 20-25 years
3) 35-45 years.

Answers: 1 – 3; 2 – 2; 3 –4; 4 –2; 5 – 1; 6 – 3; 7 – 1; 8 – 1; 9 – 1; 10 – 2.

Tasks

Task № 1. In patient P., 50 years old, during preparation for prosthetics of the oral cavity, an orthopantomogram was made on which a 3.8 tooth was found in the region of the angle of the lower jaw, located in horizontalposition in relation to the tooth 3.7. The patient does not

complain. Objectively: the configuration of the face is not changed, the skin of the physiological color, the regional lymph nodes are not palpable. The patient opens his mouth freely, within physiological capabilities. On examination of the oral cavity: the mucous membrane of the alveolar process inareas of uncrushed tooth 3.8 physiological coloration, palpationpainless. Partial secondary adentia (class II according toKennedy). Of the past diseases, the patient notes colds, hesuffering from diabetes mellitus (type II) of moderate severity.

Based on the data of the clinical examination and the results of radiation research methods, make a diagnosis.Determine the further tactics of the dentist-surgeon in the specified clinical situation.

Task № 2. Patient K., 19 years old, consulted a dental surgeon with complaints ofdull aching pains in the region of the lower jaw on the right, the presence of swelling of the right half of the face in the region of the angle of the lower jaw. The pain intensifies when swallowing and opening the mouth. The patient considers himself sick for 2 days, from the moment when pains appeared in the lower jawon right. The pains are increasing in nature. Body temperature in the evenings
reaches 37.3 ° C. Previously, the patient was not ill with anything. Objectively: face configuration is changed due to collateral soft tissue edema in the regionlower jaw on the right. The skin is physiological in color. Submandibular lymph nodesenlarged on the right (0.3 × 0.4 cm), painful on palpation, soft, elastic consistency, mobile, with skin and underlying tissues notsoldered. The patient opens his mouth within the physiological possibilities,while pain is noted. On examination of the oral cavity: 3.8 toothteething stage – two medial tubercles are visualized, the distal ones are covered with a hood of the mucous membrane, which is hyperemic,swollen, painful on palpation. When pressing on the hood of the mucous membrane covering the distal tubercles of the tooth 3.8, serous discharge is released from under it.

What additional examinationis necessary in this clinical situation?Based on the clinical examination, make a diagnosis. Determine the further tactics of the dentist-surgeon in the specified clinical situation and draw up a comprehensive treatment plan

Task №3. Patient S., 25 years old, came to the clinic to anoral surgeonwith complaints of persistent pain in the lower jaw on the right. Pains weardumb character, disturbed for two weeks. From domesticorgans and systems the patient does not note pathology. Objectively: the configuration was changed due to collateral soft tissue edema in the region

of the lower jaw on the right, physiological skin integuments. Submandibular and submentalhe lymph nodes on the right are enlarged (0.4 × 0.4 cm), painful whenpalpations, soft, elastic consistency, mobile, not soldered to the skin and underlying tissues. The patient opens his mouth painlessly, inphysiological limits. On examination of the oral cavity: the mucous membrane in the area of the tooth 4.8 is hyperemic, swollen. Tooth crown 4.8cut into ½ medial tubercles. Tooth 4.8 is distally inclined. Atpressure on the hood of the mucous membrane covering the distaltubercles of the tooth 4.8, serous discharge is allocated from under it. According toradiation research methods (dental x-ray) is determined by the superposition of the projection of the root apex 4.8 on the shadow of the mandibular canal.

Based on the clinical examination, make a diagnosis.What additional research methods are necessary in this clinical situation?What should be the further tactics of the dental surgeon inspecified clinical situation? Make a comprehensive treatment plan. In which health care institution (clinic or hospital) should the patient receive specialized care (performed tooth extraction 4.8)?What are the possible complications during complex surgery4.8 tooth extraction in the indicated clinical situation?

TOPIC:ACUTE DENTOALVEOLAR ABSCESS

Tests

1. Mandibular dentoalveolar abscess arise mainly from;

1) the canine of upper jaw
2) the last molar of the upper jaw
3) extraction of tooth 3.6
4) 1, 2
5) 2, 3

2. Mandibular dentoalveolar abscess is a result of:

1) blow to the mandible
2) injury to the mandible
3) tooth decay
4) from implants

3. Dentoalveolar abscess is:

1) inflammation of jaw bones
2) caries development of the tooth
3) abscess development under the periosteum

4) 1, 3

4. Dentoalveolar abscess can spread to:

1) nose
2) fascial spaces
3) pulp canal
4) jaw bones
5) all of the above.

5. Complications of mandibular dentoalveolar abscess is:

1) phlegmon
2) cancer
3) neck & mediastinal involvement
4) pleurisy
5) all of the above.

6. How can dentoalveolar abscess be treated:

1) extraction of the jaw
2) replacement of the jaw
3) antibiotic therapy
4) drainage of fluid in subperiosteal space and clean with antiseptics
5) extraction of tooth

7. Dentoalveolar abscess can be managed by:

1) dental therapist
2) dental surgeon
3) ear, nose and throat doctor
4) dental orthopedist
5) all of the above

8. Dentoalveolar abscess can be avoided by:

1) eating good diet
2) visit to the dentist regularly
3) medical follow up of extraction
4) all of the above

9. Clinical signs of a patient with dentoalveolar abscess include:

1) nausea
2) poor vision
3) lack of appetite
4) asymmetry of the face
5) swollen lymph nodes

10. Dentoalveolar abscess can lead to sepsis

1) true
2) false
3) 1 & 2

Answers: 1 – 3; 2 – 4; 3 – 3; 4 – 2, 4; 5 – 1, 3, 4; 6 – 3, 4, 5; 7 – 2; 8 – 4; 9 – 3, 4; 10 – 1.

Tasks

Task 1. Patient is a 32 years old, complains of pulsating pain and a sharply painful swelling in the left area of the hard palate.

Anamnesis: 3 days ago there was pain in a previously treated tooth Number 2.2. Then the toothache decreased in pain, but swelling appeared on the hard palate. The general condition is normal, and body temperature is 37,8 degrees.

Patient examination: there are no changes in the configuration of the face. The left and right submandibular lymph nodes are not palpable. Mouth opening is free without discomfort. On the left side of hard palate there is an oval-shaped bulging, the mucous membrane above it is red and in the center fluctuation is determined. Tooth Number 2.2 is filled, changed in color, percussion is painful.

Make a preliminary diagnosis.

Task 2.A 48 years old patient complains of swelling on the left cheek, pain in the jaw and the patient has a high body temperature (fever).

Anamnesis: five days ago, there was pain at tooth number 2.3, after two days there was a swelling of the soft tissues surrounding the tooth, and then the swelling was spread to the cheek. The patient didn't visit a doctor. Patient placed a heating pad on his cheek, and mouth washed with warm water.

Patient examination: there is significant swelling in the infra-orbital and anterior part of the buccal region on the left, the skin above it is not changed in color; deep palpation is painful. Mouth opening is free.

The muco-gingival fold in the region of tooth number 2.1, 2.2, 2.3, and 2.4 protrudes in the oral cavity; painful infiltration is determined on palpation.

2/3(two third) of the crown of tooth number 2.3 is destroyed by carious process, percussion is painful, the mobility is of a third degree, pus is released from under the gingival margin of a vestibular side.

Make a clinical diagnosis.

Task 3. What are the main symptoms of dentoalveolar abscess of the jaw?

Task 4. The patient is 39 years old, diagnosed with acute dentoalveolar abscess of the left body of the lower jaw. Chronic periodontitis of tooth Number 4.7

Describe the clinical picture of the disease. Make a treatment plan.

Task 5. What can dentoalveolarabscess lead to?

TOPIC:OSTEOMYELITIS OF THE JAWS

Tests

1. What are the main symptoms of acute odontogenic osteomyelitis of the jaw?

1) severe pain,
2) swelling and redness but no radiographic manifestations at first
3) decrease in trabeculae density after 10 days
4) significant radiological signs
5) no pain

2. What are sources of infection in the jaw?

1) most commonly the apex of non vital teeth
2) marginal or furcational periodontium
3) pericoronal space
4) open injury
5) hematogenous spread

3. Give the main clinical features of osteomyelitis

1) severe throbbing pain
2) tender, red, swelling gingiva
3) mobile teeth
4) lower lip paraesthesia
5) no symptoms

4. Aerobic bacteria which cause osteomyelitis include

1) Staphylococcus aureus
2) haemolytic streptococcus
3) E.coli
4) prevotella

5. What most affected jaw by osteomyelitis?

1) lower
2) upper
3) both

6. Anaerobic bacteria which cause osteomyelitis include

1) Porphyromonas
2) prevotella
3) Staphylococcus aureus

7. Osteomyelitis of maxilla is rare due to all of this except:

1) extensive blood supply to maxilla.
2) abundant medullary spaces
3) thick cortical plates
4) porous nature of membranous bone.
5) there is no correct answer

8. What symptoms of osteomyelitis?

1) fever
2) nausea
3) trismus (difficulty in opening mouth)
4) tenderness and redness
5) all answers above

9. Causes of odontogenic osteomyelitis:

1) trauma
2) virus
3) odontogenic infection
4) answers 1 and 2
5) furuncle

10. Treatment of osteomyelitis by:

1) antibiotics
2) improving oral hygiene
3) remove «causing» teeth in the area
4) all answers above
5) there is no correct answer

11. Types of osteomyelitis:

1) suppurative
2) focal sclerosing

3) diffuse sclerosing
4) fibrosis osteomyelitis
5) all answers.

12. Chronic osteomyelitis form as:
1) sequestra lying close to peripheral sclerosis
2) bleeding
3) new bone formation
4) answers 1 and 3
5) all answers

13. In which stage steomyelitis start formation of sequestra?
1) acute
2) subacute
3) chronic
4) acute and subacute
5) postacute

14. Pathological mobility and reaction on percussive groups of a teeth is defined at:
1) osteomyelitis of jaws
2) periostitis of jaws
3) abscess
4) pulpitis
5) phlegmon

15. The acute stage of an osteomyelitis of jaws proceeds:
1) 7-14 days
2) 6-8 weeks
3) 3-4 months
4) 2-3 years
5) 10-12 weeks

16. The sub-acute stage of jaw osteomyelitis proceeds:
1) 7-14 days
2) 6-8 weeks
3) 3-4 months
4) 4-8 days
5) 10-12 weeks

Answers: 1 – 1-4; 2 – 1-5; 3 – 1-4; 4 – 1- 3; 5 – 1; 6 – 1, 2; 7 – 3; 8 – 5; 9 – 3; 10 – 4; 11 – 5; 12 – 4; 13 – 3; 14 – 1; 15 – 1; 15 – 4.

Tasks

Task №1. What are the main symptoms of acute odontogenic osteomyelitis of the jaw?

Task №2. Define osteomyelitis.

Task №3. What are the inorganic bone components?

Task №4. Why does osteomyelitis occur more often in the mandible than in the maxilla?

Task №5. What are some systemic factors that can predispose to osteomyelitis?

Task №6. What are some local factors that can predispose to osteomyelitis?

TOPIC: LYMPHADENITIS OF THE MAXILLOFACIAL AREA

Tests

1. What are the main causes of lymphadenitis?

1) skin infections
2) bacteria such as streptococcus
3) bacteria such as staphylococcus
4) injury or trauma

2. Symptoms of acute purulent lymphadenitis:

1) swallowing and chewing difficult
2) complaints of throbbing pain in the lymph node
3) body temperature not exceeding 38 ^{0}C
4) the main complaint – the presence of the disease, «ball» under the skin
5) 1, 2, 3 are correct

3. What type of biopsy is indicated for the examine of the lymph nodespathology?
1) puncture
2) incisional biopsy
3) is not carried out
4) trepanation
5) 1, 4 are correct

4. The regional lymphatic drainage of the left side of the tip of the tongue is to the
1) left submental lymph node
2) left and right submental lymph nodes
3) left submandibular lymph node
4) left and right submandibular lymph nodes

5. Children up to 7 years suffer from lymphadenitis:
1) odontogenic
2) dermatogenic
3) 1,2are correct
4) hematogenic
5) 2,4are correct

6. When does formation of lymph nodes finish?
1) 2-3 years
2) 4-6 years
3) 6-8 years
4) 8-10 years
5) after 14 years

7. Role of lymphatic system at purulent diseases of an organism?
1) resorption of bacteria from surrounding tissue and their transportation in lymphatic vessels.
2) resorption of bacteria from surrounding tissue, clarification of blood by means of lymph nodes

8. How many lymph nodes does the superficial parotid group include?
1) 1-2
2) 2-3
3) 3-5
4) 5-8

5) 8-10

9. Direction of large lymphatic vessels:
1) conform to a direction of blood vessels
2) non conform a direction of blood vessels
3) conform to a direction of nerves

10. Specify the possible ways of infection in the salivary gland:
1) stomatogenous retrograde
2) hematogenous
3) lymphogenous

Answers: 1 – 1, 2, 3; 2 – 5; 3 – 1; 4 – 2; 5 – 2; 6 – 4; 7 – 1; 8 – 2; 9 – 1;10 – 1, 2, 3.

Tasks

Task 1. A 49-year-old male was referred to the Department of Oral Surgery by his general dental practitioner with a two-day history of a progressively increasing swelling in the right submandibular region.

Two weeks earlier, he noticed a broken part of lower second molar. There was an acute diffuse pain in the right lower second molar with development of swelling in the buccalvestibule. The patient decides to take antibiotic, but swelling persisted. There is no history of limited mouth opening, visual disturbances, difficulty breathing (all of them are signs of more facial space involvement).

Maxillofacial: Significant redness of the skin and right side, submandibular edema extending from inferior border of the mandible to the level of the neck. The swelling is firm on palpation without draining sinus tract formation. There is enlarged submandibular lymph nodes with tenderness.

Intraoral: The mouth opening is approximately 45 mm (limited mouth opening is not seen in this case, because it does not involve the muscles of mastication). Bimanual examination of the right intraoral site of the mandible reveals mucosal hyperemia with firm consistency. The mandibular second molar (tooth Number 4.7) was sensitive to palpation and percussion. The floor of the mouth is examined using direct and indirect vision followed by bimanual palpation of the entire area. The tissues appeared moist and very vascular and soft on palpation. We observed rough, lobular, and coral to light pink tissue of oropharynx, and the uvula area is normal.

Radiographic images: The orthopantomography evaluate odontogenic etiology of infections on different spaces. This image can reveal osseousanatomy of the maxillofacial region. The patient's postoperative orthopantomography shows a normal healing process.

What diagnosis is suggested?What would be the management?

Task 2.A 5 year-old boy was brought to the Department of Paediatric Dentistry, by his very concerned parents, with a complaint of pain and swelling in the child's lower right jaw that started 45 days ago. The pain was continuous and dull in nature, aggravated during mastication and temporarily relieved by medication. The parents reported that the facial swelling showed a gradual increase in size, and during the last 7 days, was associated with a rise in body temperature. The medical history elicited from the parents was non-contributory. Prior to the visit, a general medical practitioner, then a general dental practitioner, examined the child and finally by a paediatrician, each of them prescribed and subjected the child to a course of antibiotic therapy, with little effect. The last of these health care professionals further referred the child to Department of Paediatric Dentistry. The swelling was diffuse and soft, extending from the right corner of the mouth to almost the posterior border of the mandible, involving the entire submandibular region of the right side and also crossing over to the left side.

Intraoralexamination revealed poor oral hygiene and deep carious lesions in all of the lower primary molars with a sinus tract leading from the first primary molar of the left side. The molars in the area of concern, i.e., the right mandibular region exhibited a high degree ofmobility, such that they mimicked 'floating molars'. Obliteration of the vestibule was evident in the area of these teeth, together with marked gingival inflammation around the second molar

Task №3. What are the main symptoms of acute lymphadenitis?

Task №4.Define a treatment plan of chroniclymphadenitis?

Task №5. What can acute purulent lymphadenitis lead to?

TOPIC: FURUNCLES AND CARBUNCLES OF THE MAXILLOFACIAL AREA

Tests

1. Etiology of a boil:

1) skin contamination
2) damage of the skin of the face
3) extruding blackheads
4) skin disease (eczema)
5) complication of phlegmon of the maxillary tissue

2. Which statement is correct?

1) facial boil occurs due to infection of the hair follicle or sebaceous gland
2) in this case, a pustule is formed
3) follicle necrosis occurs
4) in the surrounding tissues – inflammatory infiltrate and edema
5) the disease is chronic

3. Furuncles inflammation occurs:

1) in the hair follicle
2) in the subcutaneous tissue
3) in the lymph nodes

4. Condition of the patient with carbuncle:

1) satisfactory
2) moderate
3) severe

5. The clinical picture of the carbuncle of the face is characterized:

1) infiltrate
2) the presence of several pustules in the center of the infiltrate
3) edema
4) pain
5) an increase in regional lymph nodes
6) hyperemia of the skin over the infiltrate

6. Facial carbuncle treatment:

1) antibacterial
2) desensitizing
3) detoxification therapy
4) anti-gangrenous serum

7. Methods for treating a furuncle of the face:

1) UV (ultraviolet) treatment
2) dressing with hypertonic solution
3) ointment dressings
4) linear section through the infiltrate

8. Treatment of furuncles and facial carbuncle should be carried out in:
1) clinics
2) the hospital

9. Furuncle of the buccal region may be complicated by thrombophlebitis of:
1) facial vein
2) superficial vein
3) angular vein

Answers:1 – 1, 2, 3; 2 – 1-4; 3 – 1; 4 – 2; 5 – 1-6; 6 – 1, 2, 3; 7 – 2, 3, 4; 8 – 2; 9 – 1.

Tasks

Task 1. A 41 years old patient was treated in the Department of Maxillofacial Surgery with a diagnosis of boils of the upper lip. On the seventh day after the course of complex treatment, including primary surgical treatment, postoperative drug and physiotherapeutic treatment, the patient was discharged in satisfactory condition under the supervision of a dental surgeon in a clinic to continue the course of treatment and rehabilitation measures.

Justify the decision behind patient's discharge under the supervision of the surgeon to continue the course of treatment and rehabilitation measures at home?

Task 2. A 43 yearold patientwith carbuncle on the upper lip was treated by dental surgeon at the dental clinic (primary surgical treatment of the purulent focus). After one day, the patient's condition worsened sharply: body temperature increased to 38 ºC, weakness, headache appeared, and edema spread to the periorbital region. The patient was transported to the hospital by an ambulance.

Objective examination: the configuration of the face is changed due to infiltration and edema of the upper lip, extending to the adjacent tissues of the buccal periorbital regions on the left side. Infiltrated pain upon

palpation was noticed. The skin in the area of infiltration is hyperemic, swollen, soldered to the underlying tissues.

What mistakes did the surgeon make during the initial management of the patient for first aid treatment?

Task 3. A 31 years old patient complains of a painful "elevation" in the left buccal region, headaches, general weakness. Symptoms appeared 3 days ago after a cosmetic facial cleansing procedure carried out by the patient at home. Body temperature is 37.9 ° C. The left buccal region is edematous, a cone-shaped painful infiltrate is determined in the center of the edema, the surrounding skin is hyperemic, in the middle a crater-like wound 0.2 x 0.3 cm, made by necrotic tissue, and purulent discharge.

Make a diagnosis. Make a treatment plan.

Task 4. A 48 years old patient developed a pustule on the skin of his chin, which quickly developed to a dense, sharply painful infiltrate measuring 3 x 5 cm. The covering skin has blue-red color. In the center there are three zones of necrosis near the hair follicles. The chin lymph nodes are enlarged, and painful upon palpation. What is the most possible diagnosis?

Task 5. A 20 years old patient has edema and infiltrate of 1.5 x 1.5 cm in the area of the upper lip on the right side. A necrotic trunk is in the center of the infiltrate. Establish a preliminary clinical diagnosis.

TOPIC: PHLEGMONS AND ABSCESSES OF THE MAXILLARY REGION

Tests

1. What is an abscess?

1) composed mainly of a central area of organisms and disintegrating polymorphonuclear leukocytes surrounded by viable leukocytes and some lymphocytes
2) a diffuse inflammatory reaction

2. What is cellulitis?

1) a local inflammatory reaction
2) a diffuse inflammatory reaction

3. Anaerobic gram-positive cocci are found in about 65% of cases, usually...

1) Streptococcus
2) Gonococcus
3) Gemiphorus
4) Peptostreptococcus

4. Odontogenic infections seem to pass through what four stages:

1) inoculation stage
2) cellulitis stage
3) abscess stage
4) Ludwig's angina
5) resolution stage

5. Pus spreads outside the alveolar bone and after perforating the bone it then spreads to the subperiosteal space

1) subperiosteal abscess
2) intraalveolar abscess
3) submucosa abscess
4) cellulitis

6. Firm, acute toxic and severe diffuse cellulitis that spreads rapidly, bilaterally affecting the submaxillary, sublingual, and submental spaces and resulting to an indurated swelling:

1) subperiosteal abscess
2) intraalveolar abscess
3) submucosa abscess
4) cellulitis
5) Ludwig's angina

7. Patient presents with «gum flaps» occuring over erupted wisdom teeth that are inflamed:

1) pericoronitis
2) calculus
3) periodontitis
4) Vincent's angina

8. Infections arising from maxillary teeth spread where?

1) into infraorbital space
2) into palatal space
3) into orbital space
4) into infratemporal space

5) into maxillary sinus
6) all answers

9. Where is the Canine Space?
1) between the buccinator and skin
2) between levator anguli oris and levator labii superioris
3) between lateral aspect of mandible and medial boundary of the masseter

10. Key to successful management of fascial spaces?
1) remove source of infection
2) inflammation treatment
3) degranulation

Answers: 1 – 1; 2 – 2, 3 – 1, 4; 4 – 1, 2, 3, 5; 5 – 1; 6 – 5; 7 – 1; 8 – 6; 9 – 2; 10 – 1.

Tasks

Task № 1. A 41 years old patient periodically suffered from pain at tooth Number 1.3. Five days ago, the patient again suffered from severe pain at tooth number 1.3, the next day there was a swelling of the upper lip and the infraorbital area to the right, the temperature rose 37.8^0C. The patient rinsed his mouth with a solution of sodium bicarbonate, and applied a heating pad. After this, the pain in the tooth subsided, but the swelling spread to the entire infraorbital area, continuing to increase in the following days. Body temperature rose to $38\ ^0$C.

Objectively: the state is satisfactory. With an extraoral examination, a swelling was determined in the infraorbital region, the upper lip and the lower eyelid on the right, which extended to the right cheek region. The nose is shifted to the left, and the right corner of the mouth is lowered. The skin above the swelling is tense and reddened. Opening the mouth is free. When palpated, it is determined by a painful compaction in the right buccal region. In the center of it there is a softening point, over which it is possible to determine the fluctuation (fluctuation).

The mucous membrane of the vestibule of the mouth in the region of tooth Number 1.5, 1.4, 1.3, 1.2 are bright red colored, palpation reveals a condensation with a softening and fluctuation above tooth Number 1.4, 1.3.

Make a diagnosis. Analyze the mistakes made by the patient.

Task № 2. A 43 years old patient complained of a limited swelling in the right side of the face.

Anamnesis: the patient addressed to the doctor that a week ago there were pains in tooth Number 1.7, amplifying at touching the tooth. The treatment was performed to tooth Number1.7. A few days after the treatment of tooth Number 1.7 the pain in this tooth resumed, a swelling appeared in the upper part of the right cheek, and the body temperature was 37.2 ^{0}C. When viewed below the zygomatic area on the right there is a swelling, the skin over it is thinned, and red. With palpation, the swelling is painful, limited, the skin is not going to a fold, a seal is determined up to 3 cm in diameter, in the center there is fluctuation. Opening the mouth is free. The mucous membrane of the vestibule of the mouth in the region of the upper molars on the right is of a red color, and during palpation a broad cord extends to the adjacent region of the cheek. When massing from the parotid duct, a transparent secret is obtained. Tooth Number 1.7 is filled, mobility of 2 degrees, and percussion is painful.

Make a clinical diagnosis. Schedule a comprehensive treatment plan.

Task №3. The patient complained of minor pain in the cheek area, which increased with touching. Earlier the patient complained of pain a tooth in the right posterior area of the upper jaw. Objectively: in the upper cheek on the right is an infiltrate of a rounded shape, there is edema of surrounding soft tissues. Infiltrate is soldered to the skin, when palpated it is determined by fluctuation. The mucous membrane of the cheek is sharply hyperemic, edematic, and the teeth are visible. Make a diagnosis. Make a treatment plan.

Task №4. A 42-year-old patient complains of severe spontaneous pains that increase with mouth opening and chewing. Objectively: there is infiltration of the left buccal region, pronounced edema of the surrounding tissue extending to the upper and lower eyelids, and the eye gap is narrowed. The skin in the cheek region is hyperemic and it is not going to a fold. There is edema and hyperemia of the left mucous membrane of the cheek, and the upper arch of the vestibule of the oral cavity. The prints of teeth are visible on the mucosa. Crown of tooth Number2.6,2.7 are destroyed above the level of the gum. Make a diagnosis. Make a treatment plan.

Task №5.A 50 years old patient complains of pain and swelling on the rightside of the cheek area and the upper lip.

Objectively: swelling of the tissues of the infraorbital, buccal areas and upper lip on the right. The right wing of the nose is elevated due to edema, and the nasolabial fold is smoothed. At palpation, the swelling is soft, slightly painless, the skin is not changed in color, and it is not going to fold. When examining the oral cavity: the upper vestibule of the mouth is smoothed, the mucous membrane over it is hyperemic and edematous and palpation is painful.The Crown of tooth Number 1.3 is destroyed above the level of the gum, and the tooth tissues are changed in color. Make a diagnosis. Make a treatment plan.

TOPIC: PHLEGMONS AND ABSCESSES OF THE MANDIBULAR REGION

Tests

1. Infections arising from mandibular teeth spread where?

1) into the submandibular space
2) into the sublingual space
3) into the submental space
4) into the masticator space
5) infraorbital space

2. Thin potential space between the levator anguli oris and the levator labii superioris muscles:

1) submental space
2) infraorbital space
3) submandibular space
4) sublingual space

3. What are the medial and superior borders of the infratemproal space?

1) bounded medially by lateral pterygoid plate of sphenoid bone
2) bounded superiorly by base of skul;
3) bounded medially by base of skull
4) bounded superiorly by lateral pterygoid plate of sphenoid bone

4. What space lies between the oral mucosa of the floor of the mouth and the mylohyoid muscle?

1) sublingual space
2) submental space
3) submandibular space

5. What space lies between the mylohyoid muscle and the overlying superficial layer of deep cervical fascia?
1) submandibular space
2) sublingual space
3) submental space

6. What space lies between the anterior bellies of the right and left digastric muscles and between the mylohyoid muscle and the overlying fascia?
1) submental space
2) sublingual space
3) submandibular

7. In severe odontogenic infections what space is most frequently invovled?
1) submental space
2) sublingual space
3) submandibular

8. What is border between the sublingual space and the submandibular space?
1) masseteric muscle
2) mylohyoid muscle
3) pterigoidal muscle
4) digastric muscles

9. What are the parts of mandibular teeth involved in odontogenic infections?
1) submental
2) sublingual
3) submandibular
4) buccal
5) all answers

10. Sublingual space abscess space communications:
1) submental space
2) lateral pharyngeal space (posteriorly)
3) submandibular space (inferiorly)
4) all answers

11. Submasseteric space location:

1) between the buccinator and skin
2) between lateral aspect of mandible and medial boundary of the masseter
3) between levator anguli oris and levator labii superioris

Answers: 1 – 1, 2, 3, 4; 2 – 2; 3 – 1, 2; 4 – 1; 5 – 1; 6 – 1; 7 – 3; 8 – 2; 9 – 5; 10 – 4; 11 – 2.

Tasks

Task №1. The patient, aged 42, complained of severe pain when swallowing, moving the tongue and opening the mouth. Objectively: there is a swelling in the posterior part of the submandibular triangle. Skin in this area is not changed. Palpation of lymph nodes is determines their increase and soreness. Opening the mouth by 2 cm.

In the run-up to the oral cavity, there are no inflammatory changes. The mucous membrane of the hyoid fold is sharply hyperemic, edematous and smoothed. The tissues on this site are sharply painful, infiltrated, and palpation is determined by fluctuation.

Make a diagnosis. Make a treatment plan.

Task №2. A 35 years old patient complained of pain in the area of the lower jaw to the left and swelling. Movement of the jaw, swallowing, and speaking aggravates pain. The patient has opening the mouth. When examined, there is a swelling in the area of the lower jaw on the left. The skin over the swelling is hyperemic, it is not collected in the fold, and there is a painful palpation in the left side of the lower jaw. Opening the mouth is somewhat limited because of soreness. When examining the oral cavity, the crown of tooth Number 35 teeth is destroyed to the level of the gum. There is a slight hyperemia of the mucosa of the hyoid area.

Make a diagnosis. Specify online access.

Task №3. Patient K. came to the doctor with complaints of swelling, soreness on the right area of the cheek, as well as pain while opening of the mouth. With palpation, there is a sharp pain, swelling with fluctuation and the skin of the cheek is not going to fold. From the oral cavity, there is hyperemia and swelling of the mucous cheek.

Make a preliminary diagnosis. What incision is carried out at the opening of this purulent formation?

Task №4. A 55-year-old patient complains of severe spontaneous pain in the lower jaw area. Objectively: diffuse swelling in the region of the entire submandibular triangle. The skin above it is infiltrated, and it is not going to a fold. A dense painful infiltration is palpable in the center. There is edema in the cheek and the parotid area. Opening the mouth is not limited.

Make a diagnosis. Define the scheme of treatment.

Task №5. A 48 years old patient complained of a slight swelling in the chin area, sharp pains in the tongue, and inability to swallow. Objectively: edema of the tissues of the subclavian area, the skin in this area is not changed, it is well assembled into the fold, and palpation in the middle line is painful. Opening the mouth is free and not ristricted, but painful. The tongue is enlarged due to uniform edema, its mobility is limited, and it's palpation is painful.

TOPIC: ACTINOMYCOSIS, SYPHILIS, TUBERCULOSIS, HIV-INFECTION OF THE MAXILLOFACIAL AREA

Tests

1. Name the causative agent of actinomycosis:
1) tubercle bacillus
2) radiate fungus
3) pale spirochete
4) yeast fungus

2. What environment promotes the development of actinomycotic infection?
1) anaerobic
2) aerobic
3) mixed
4) it doesn't matter

3. Indicate the ways of infection of actinomycetes tissues and organs of the maxillofacial area:
1) odontogenic
2) household
3) lymphogenic
4) the length
5) traumatic

4. Who first discovered the causative agent of actinomycosis in humans?
1) professor Berdygan K.I.
2) professor Israel
3) professor Robustova T.G.
4) professor Bernadsky Y.

5. When the periosteum of the jaw is involved in the pathological process when the actinomycosis affects the bone tissue?
1) primarily
2) secondarily
3) simultaneously
4) not involved

6. Indicate the most frequent localization of the submucosal form of actinomycosis in the oral cavity:
1) upper and lower lip
2) the cheek
3) the retromolar fossa
4) the lateral surface of the tongue
5) peritonsillar area.

7. Which of the large salivary glands are most often affected by ray fungi?
1) sublingual
2) submandibular
3) parotid
4) to the same extent

8. What are the forms of actinomycosis of jaw bones:
1) destructive
2) cystic
3) productively destructive
4) productive
5) lysing

9. Are antituberculosis drugs used for the medical treatment of actinomycosis?
1) yes
2) none

10. Actinomycosis is:
1) a developmental defect
2) a dystrophic process
3) tumor-like process
4) a specific inflammatory disease
5) nonspecific inflammatory disease

11. What develops with the long flow of actinomycosis in the maxillofacial region?

1) mediastenitis
2) pneumosclerosis
3) bronchial asthma
4) amyloidosis of internal organs

12. Pathomorphologic changes at a tuberculosis do not depend from:

1) forms of disease
2) stages of disease
3) localizations of process
4) seasonal prevalence of disease

13. Specific to a tuberculosis there are cells:

1) Swanowsky
2) Yavorsky cells
3) Ksantom.
4) Pirogov-Langhans
5) Hargrews

14. Constitutional Symptoms of Secondary Syphilis:

1) fever
2) sore throat
3) malaise
4) weight loss
5) headache
6) patchy alopecia

15. Neurological Complications of Tertiary Syphilis:

1) dementia
2) muscular reflex deterioration
3) argyll robertson pupil
4) seizures

16. What is the incubation period for syphilis?

1) 10-90 days
2) 1-2 days
3) 4-7 days
4) 100 days
5) 1 year

17. Early HIV Presentation:

1) seborreic keratitis
2) psoriasis
3) shingles (esp if >1 dermatome)
4) molluscum contagiousum
5) lymphadenopathy >1 extrainguinal site for > 3 months
6) thrombocytopenia – purpura
7) oral hairly leukoplakia
8) oral candidiasis
9) all answers

18. What are the three different ways for diagnosing AIDs?

1) CD4 count <200
2) CD4 percentage <14%
3) opportunistic infection
4) all answers

19. HIV Life Cycle:

1) binding, atachment
2) fusion
3) reverse transcriptase
4) replicaion
5) assembly
6) budding (protease)

20. What family and genus does the HIV virus belong to?

1) adenovirus
2) retrovirus family, lentivirus genus
3) koronavirus
4) z-virus

Answers: 1 – 2; 2 – 1; 3 – 1, 3, 4, 5; 4 – 2; 5 – 2; 6 – 1, 2, 3, 5; 7 – 3; 8 – 1, 3, 4; 9 – 1; 10 – 4; 11 – 4; 12 – 4; 13 – 4; 14 – 1-6; 15 – 1-4; 16 – 1; 17 – 9; 18 – 4; 19 – 1-6; 20 – 2.

Tasks

Task №1. A 27 years old patient complained of pain and swelling of soft tissues in the left submandibular region, an increase in body temperature to 37.3 ºC, malaise, and general weakness. Objectively: the configuration of the face is changed due to infiltration in the left

submandibular region. The skin above it is bright pink, it is soldered to surrounding tissues, and in the center is thinned. Infiltration is without clear boundaries. The patient opens his mouth within the physiological possibility. When examining the oral cavity: the mucosa in the region of tooth Number 3.7 is hyperemic, edematous. The tooth Number 3.7 is filled, and its percussion is positive.

Which preliminary diagnosis based on the data presented in the condition of the task can be delivered to the patient?What is the treatment plan for the patient in this clinical situation?

Task №2. A 39 years old patient, visited adental surgeon with complaints of pain and swelling of the soft tissues of the right side of the face, limiting mouth opening, raising the body temperature to 38.4 ° C, weakness, nausea, and malaise. Objectively: the configuration of the face is changed due to edema and infiltration of soft tissues in the submandibular, buccal, parotid-chewing and temporal regions on the right. In some areas of the infiltrate there are foci of softening. The skin above it is cyanotic in color and it is soldered to the underlying tissues. Through the fistulas, a viscous, suppurate discharge, containing small whitish grains. The opening of the mouth is limited to 1.5 cm between the central incisors. The mouth is not sanitized and there are multiple foci of chronic odontogenic infection.

What diagnosis on the basis of the data presented in the condition of the task can be delivered to the patient?What additional methods of examination in the specified clinical situation should the dental surgeon appoint to the patient?What is the treatment plan for the patient in this clinical situation?

Task №3. A 19 years old patient visited a dental-surgeon with complaints about the thickening of the lower jaw to the left, which slowly increases. In the history there were periodic pains tooth Number 3.6, there was an edema of soft tissues, sometimes the body temperature increased to 37,2 ° C. Objectively: the configuration of the face is changed due to the thickening of the body of the left side of lower jaw. Opening the mouth is somewhat difficult, but painless. When examining the oral cavity: tooth Number 3.6 is filled (treated). According to the radiation methods of investigation (orthopantomograms), the periodontal tooth gap was determined to be around tooth Number 3.6. Its root canals are sealed (filled/obturation/treated) to ⅔ of their lengths. In the projection of the teeth Number 3.6, and 3.7, bone stratifications from the periosteum are visualized. The compacting of the structure of a compact and spongy

substance, alternating with foci of bone resorption, is determined. The general condition of the patient is satisfactory.

What diagnosis on the basis of the data presented in the condition of the task can be delivered to the patient?What is the treatment plan for the patient in this clinical situation?

Task №4. A 24 years old patient visited a dental-surgeon with complaints about the presence of rounded formation (swelling) in the right submandibular region, which he discovered by accident. Objectively: in the right submandibular region palpation is painless, the size is increased to 2 cm in diameter, a mobile node of dense consistency. Skin over it is not changed. The patient opens his mouth within the physiological possibility. Tooth Number 4.6 was previously treated. According to the X-ray methods of examination (orthopantomograms) in the region of the tips of the roots of the tooth 4.6, a rounded focus of the discharge of bone tissue with distinct contours (0.3 × 0.4 cm) is determined.

What diagnosis on the basis of the data presented in the condition of the task can be delivered to the patient?What additional methods of examination in the specified clinical situation should the dentist-surgeon appoint to the patient?What is the treatment plan for the patient in this clinical situation?

Task №5. The doctor prescribed anti-tuberculosis medications to the patient with cutaneous actinomycosis of the right cheek. Did he do it right?

Task 6. A 28 years old patient has addressed with complaints to presence of an ulcer in tongue. Objectively: A painless oral ulceration of red color with a smooth polished bottom precisely outlined, and with a diameter of about 1,5 cm. Define the preliminary diagnosis

Task 7.A 45 years old patient, who suffers the open form of the tuberculosis, directed from general physician to the dental surgeon with the purpose of sanitation of an oral cavity. At examine: half of crowns of tooth Number 36, 37 are decayed, percussion of teeth weakly morbid, the mucosa is not changed aroundthe projection of apexes of roots. Tubercular periodontitis of tooth Number 36, 37 is diagnosed. What optimum tactics of treatment of the struck teeth?

Task 8. A 40 years old patient, upon examination the doctor found an ulcer located under the tongue with dimensions of 1,5 x 2 cm. Edges of it are undermined, the bottom has an acinose kind, and is coated with

yellowish granulations.

Define the preliminary diagnosis. Name disease with which it is necessary to carry out differential diagnostics. What additional methods are necessary for an establishment of the diagnosis?Appoint treatment. In what consultation of the expert requires the patient?

Task 9.A Patient has addressed dental surgeon with complaints to presence of an ulcer in tongue. At survey: a ulcer is of a spherical form, painless at a palpation, the dimension is 0,5 cm with equal edges which rise above the tissues a little, the surface of ulcer is smooth, and its of a shine red color.

Define the preliminary diagnosis.Make the plan of examine of the patient. Name diseases with which it is necessary to carry out differential diagnostics.

TOPIC: MAXILLARY SINUSITIS

Tests

1. Which imaging study is the most appropriate for the diagnosis of chronic sinusitis?

1) MRI
2) CT scan
3) X-ray
4) Ultrasound of the sinusitis

2. What percent of adult acute viral rhinitis/sinusitis develops into acute bacterial sinusitis?

1) 0,5-2%
2) 10-20%
3) 20-30%
4) 40-50%

3. Which is not an appropriate management of uncomplicated acute bacterial sinusitis?

1) oral antibiotics
2) intranasal steroid spray
3) irrigation
4) intravenous or intramuscular antibiotics

4. Which method is the most definitive method to detect the presence of nasal polyps?
1) CT scan of the sinus
2) MRI of the sinus
3) nasal endoscopy
4) anterior rhinoscopy

5. True or False: There are several FDA-approved medications for the treatment of chronic sinusitis without nasal polyps.
1) true
2) false

6. If a patient has a one-sided clear runny nose without significant other seasonal allergy symptoms such as sneezing and itch/watery eyes, what should the doctors be concerned about?
1) nasal polyps
2) mucocele
3) allergic fungal sinusitis
4) sinus or nasal tumor

7. If a patient has a unilateral sinus mass or involvement, the diagnosis is less likely to be:
1) allergic rhinitis
2) cold
3) gustatory rhinitis
4) cerebrospinal fluid leakage

8. Isfacial pressure/pain a symptom of sinusitis?
1) yes
2) no

9. Another term for a sinus infection is
1) sinusitis
2) arrhythmia
3) aplexia
4) mononucleosis

10. Which of the following is NOT a common symptom of a sinus infection? Pain/pressure in the face
1) Stuffy or runny nose

2) sore throat
3) coughing
4) all answers are common symptoms of sinus infections

Answers: 1 – 2; 2 – 1; 3 – 4; 4 – 3; 5 – 2; 6 - 1; 7 – 4; 8 – 1; 9 – 1; 10 – 5.

Tasks

Task 1. A patient complains of headache, purulent odoriferous discharge from the left side of the nose and difficulty breathing on the left side. These symptoms have been disturbing the patient for 3 years. Headache is diffused, accompanied by increase of discharge from the left side of the nose, dryness in the throat and the appearance of mucopurulent sputum in the throat in the morning. The general condition is satisfactory. The body temperature is 37.2^0C. Left lower eyelid is edematous. The left cheek is painful on palpation. The skin at the left nasal aperture is hyperemic and infiltrated with fissures covered with green crusts. The mucous membrane of the left half of the nose is hyperemic, swollen, covered with thick muco-purulent discharge. Pathological changes are not found in the right half of the nose. The 5th tooth of the left upper jaw is diagnosed with carious. Pharyngeal mucosa is hyperemic, swollen, and covered with thick muco-purulent discharge.Make a diagnosis. Propose methods of treatment.

Task 2. A 14 years old patient, complains of severe pain in the right side of the forehead, nasal congestion, and purulent discharge from the right half of the nose, and increased body temperature to 38.7^0C. All these symptoms started 4 days ago after influenza. Nasal mucosa is hyperemic and infiltrated. Pus is detected in the right middle and lower nasal passages. Breathing through the right half of the nose is difficult. There is tenderness of the right side of the forehead. X-ray of the para-nasal sinuses shows darkening in the right frontal sinus with horizontal level of liquid.Make a diagnosis. Propose methods of treatment

Task 3. A 62 years old patient complains of headache, weakness, lacrimation, and absence of nasal headache about a year ago. Restorative treatment was performed but improvement didn't follow. Facial asymmetry due to right exophthalmos and deformation of the right cheek is observed. The right naso-labial fold is flattened. Conjunctiva of the right eye is hyperemic. There is pus at the corners of his eyes. Mobility of the eyeball

is not disturbed. The right half of the nose is filled with a blue neoplasm displacing the nasal septum to the left. The left side of the nose is narrowed, and its mucous membrane is swollen. Left nasal passages are free. The back part of the neoplasm is hanging from the right choanae. The front wall of the right maxillary sinus is absent. The hard palate is deformed. Palpation determined bone destruction. Significant reduction of skin sensitivity of the right cheek is detected. Regional lymph nodes are not enlarged. X-ray of the paranasal sinuses shows that a shadow occupies the right half of the nose. There is bone destruction of the medial and orbital wall of the right maxillary sinus. The darkening in the right frontal and maxillary sinuses is homogeneous. There is bleeding from the right side of the nose. Make a diagnosis. Propose methods of treatment.

Task 4. A 17 years old patient suffers from chronic right-sided maxillary sinusitis. 10 days ago after hypothermia,its exacerbation has arisen. The patient did not go to the doctor, but was treated with thermal procedures and painkillers. Despite this treatment, there was a worsening of the condition, and two days ago the temperature rose to 39.5^0 C. There were bursting, with every day growing pains of the right eye, eyelids swelled, and eyes began to open badly. Objectively: the patient's condition is of moderate severity, the eyelids of the right eye are sharply infiltrated. The skin over them is hyperemic. The glottis is narrowed. Exophthalmos and chemosis of the right eye are determined. Palpation on the anterior wall of the upper jaw from the right is painful. The congestion of the right side of the nose is determined. With a rhinoscope in the middle nasal passage to the right, a purulent exudate is found. Make a diagnosis. Propose methods of treatment.

Task 5. A 43 years old patient, consulted about the installation of a dental implant on the upper jaw in the left side of missing tooth number 2.5-2.7.

From an anamnesis it is established that for 3 years,the patient suffers from chronic left-sided maxillary sinusitis, and in this connection repeatedly appealed to ENT-doctor during periods of exacerbations. Tooth number 2.5, 2.6, 2.7 teeth wereextracted due to complications of caries. On the orthopantomogram the pneumatic type of the maxillary sinus is determined. The distance from the crest of the alveolar process in the zone 2.6-2.7 to the sinus bottom is 5 mm.

Determine the possibility of dental implantation.

TOPIC: DIFFERENTIAL DIAGNOSIS OF INFLAMMATORY PROCESSES OF THE MAXILLOFACIAL AREA

Tests

1. In wich cases the Vincent'ssymptom is determined?
1) acute periodontitis
2) acute periostitis
3) chronic periodontitis
4) lower jaw osteomyelitis
5) acute pericoronoritis

2. What clinical sign is characteristic for jaw osteomyelitis?
1) edema of the mucous membrane of the alveolar process
2) thickening of the lower jaw from the vestibular and oral side
3) thickening of the lower jaw from the vestibular side
4) difficulty with mouth opening

3. Characteristics of the acute odontogenic osteomyelitis:
1) swelling of the alveolar mucous membrane
2) mobility of the causative tooth
3) inflammation in surrounding soft tissues in the form of an abscess or phlegmon
4) the presence of Vincent's symptom

4. Dentoalveolar abscess of the jaws should be differentiated with:
1) acute osteomyelitis of the jaws
2) acute sialoadenitis of the submaxillary gland
3) acute arthritis of the temporomandibular joint
4) actinomycosis of the jaws

5. A sharp pain only in the area of the causative tooth is characteristic for:
1) periodontitis
2) dentoalveolar abscess
3) osteomyelitis

6. The general condition of the patient is not changed when:
1) acute periodontitis
2) dentoalveolar abscess

3) acute osteomyelitis

7. Give a description of dentoalveolar abscess:

1) slight edema and hyperemia of the mucous gum in the area of the causative tooth

2) edema, hyperemia, infiltration of the mucosa in the area of several teeth on one side of the alveolar process

3) inflammatory infiltrate, covering the jaw from two sides

8. In case acute osteomyelitis of the jaw, in contrast to dentoalveolar abscess and periodontitis, the following changes in soft tissues are presens:

1) edema and infiltration are not determined

2) extensive edema of soft tissues and insignificant gingival infiltration in the area of «causal» and nearby teeth

3) dense painful infiltration, the skin is hyperemic, it does not gather into the fold

9. Asymmetry of the face due to soft tissues infiltration of the maxillofacial region is characteristic for:

1. periodontitis
2. dentoalveolar abscess
3. osteomyelitis

10. The general condition in case of acute osteomyelitis is:

1) satisfactory

2) satisfactory or moderate

3) moderate or heavy

Answers: 1 – 4; 2 – 2, 4; 3 – 1-4; 4 – 1; 5 – 1; 6 – 1; 7 – 1, 2; 8 – 3; 9 – 3; 10 – 3.

Tasks

Task 1. A 31 years old patient, came to the dentist with complaints about painful swelling in the right upper jaw, and increased body temperature up to 38 °C. From an anamnesis it is known, that 3 days ago there was toothache of the right upper jaw. The patient took analgesics. A day later the pain in the area of the causal tooth decreased, but there was a dull aching pain in the entire upper jaw. There was a swelling of the submaxillary soft tissues. Objectively: the configuration of the face is changed due to soft edema of the right buccal region. Examination the oral

cavity: edema and hyperemia of the alveolar mucosa in area of the tooth Number 1.3, 1.4, and 1.5. Palpation determines the fluctuation in the zone. Half of the Crown of tooth Number 1.4is destroyed, and percussion is positive.

What is the diagnosis?Carry out a differential diagnosis of this pathological condition.

Task 2. Mary is a 40 years old patient who is a dental phobic. She attended 4 weeks ago as a casual patient for extraction of a grossly decayed lower left first molar. She has reluctantly returned now complaining of awful pain on the lower left, swelling in that region and in the submandibular region, a numb lip on the lower left and a bad taste. Radiographs are taken. The radiographs show a 'mixed lesion' (i.e. radiolucent and radio-opaque) in the mandibular body.Describe your assessment, likely diagnosis and management.

Task 3. Chris is 25 years of age. He visiteda dental surgeon complaining that he has pain and swelling at the back of his mouth on the lower right side, a bad taste, badbreath and that he cannot open his mouth properly. He has lymphadenopathy of the right submandibular and cervical nodes. You know from a previous visit that he has a partially erupted lower third molar.Describe your assessment, likely diagnosis and management.

Task 4. A 75 years old woman complaining of 'an infection' in her left lower jaw that has been present for a few weeks, since she had her 35 extracted, and which now is causing her a lot of distress. She cannot wear her lower partial denture. She suffers from severe osteoporosis, having sustained several vertebral fractures over the last 5 years. On examination,there is swelling extra orally over the left jaw and some cervical lymphadenopathy. Intraoral, she has a partially dentate mouth, with missing molars on the lower left side of the jaw. Her alveolar ridge distal to tooth Number 3.5 is swollen and a draining sinus is visible buccally. The socket of tooth Number 3.5 is still partly open. Radiographic: there is a 2 × 3 cm area of radiolucency with a central area of radio-opacity located in region of tooth Number 3.5 and 3.6.

What diagnosis is suggested? What would be the management?

Task 5. A 20 years old male presents with pain and swelling at the 'back of his mouth' on the right side. It has been present for a couple of days. On examination, he has a tender, palpable, upper right cervical lymph node. There is some trismus. Intraoral, there is swelling of the gingivae

distal to tooth Number 4.7. During occlusion, erupted tooth Number 1.8 is traumatizing this swelling. Radio graphic examination: tooth Number 4.8 is present but mesioangularly impacted against tooth number 4.7.

What diagnosis is suggested? What would be the management?

Task 6. Define differential diagnosis of chronic osteomyelitis?

TOPIC: SEVERE COMPLICATIONS OF THE MAXILLOFACIAL INFECTION

Tests

1. What is Lemierre's syndrome?

1) fusobacterium infection
2) suppurative jugular venous thrombosis
3) aerobic infection

2. Infection spread with serious consequences

1) cavernous sinus thrombosis
2) mediastinitis
3) ludwig's angina
4) Abscess

3. What can spread by vascular system?

1) bacteraemia
2) infected thrombus
3) tooth

4. How can bacteraemia occur?

1) during dental treatment, bacteria can enter the blood circulation and can cause transient bacteraemia
2) in high risk patients – these bacteria may lodge in compromised tissues and can set up serious infections e.g., infective endocarditis
3) therefore antibiotic premedication before invasive dental procedures is essential in high risk patients

5. How can infected thrombus occur

1) dental infection can lead to thrombus formation and increase intra vascular blood pressure – can reverse the direction of blood flow
2) infected thrombi can break off and travel as an embolus (emboli), through veins to dural venous sinuses

6. Where are the cavernous sinuses located

1) located on the side of the sphenoid bone
2) located on the side of the temporal bone
3) located on the side of the occipital bone

7. What does the cavernous sinus communicate with

1) each other
2) pterygoid plexus of veins
3) superior ophthalmic vein (which anastomose with facial vein)

8. What do the cavernous sinuses drain

1) teeth drain through posterior superior alveolar arteria&inferior alveolarveins
2) lips drain through superior & inferior labial veins
3) pterygoid plexus of veins

9. Signs and symptoms of a cavernous sinus thrombosis

1) fever, drowsiness, rapid pulse, oedema in eyelids, tearing, diplopia
2) pressure headaches, generalized malaise
3) meningitis, septicaemia
4) tooth loss

10. Prevention of spread of dental infection

1) provision of regular, quality dentistry
2) early diagnosis & treatment
3) tooth lost

11. What is mediastinitis and consequences?

1) infection of the mediastinum can compress heart and lungs-also mess with nervous systemcontrol of heart beat and breathing rate
2) mortality rate is very high
3) can spread to the abdominal cavity
4) there is no right answer

12. What nerves can be affected by an infection of the cavernous sinus?

1) cranial nerve III, IV, V, VI
2) most common is VI
3) cranial nerve VII

13. What is sepsis?

1) immune system response to infection that has spread into the blood
2) can progress to septic shock and eventually multiple organ dysfunction
3) infection of the mediastinum

14. What is SIRS?

1) SIRS-systemic inflammatory response syndrome
2) clinical manifestation of sepsis but no infectious material or agent can be found
3) infection of the mediastinum

15. Why does necrotizing fasciitis occur?

1) due to superficial side of deep cervical fascia becoming infected and necrosed
2) clinical manifestation of sepsis
3) infection of the mediastinum

Answers: 1 – 1; 2 – 1, 2, 3; 3 – 1, 2; 4 – 1, 2, 3; 5 – 1, 2; 6 – 1;7 – 1, 2, 3; 8 – 2; 9 – 1, 2; 10 – 1, 2; 11 – 1, 2, 3; 12 – 1; 13 – 1, 2; 14 – 2; 15 – 1.

Tasks

Task 1. Infections can rise superiorly through the sinuses or vascular structures to invade where?

Task 2. Aggressive surgical exploration is still the primary method of therapy for serious odontogenic infection of the head & neck. Is it true?

Task 3. What is the retrovisceral space?

Task 4. Do infections of lateral pharyngeal space have access to retrovisceral spaces?

Task 5. Patient presents with extensive swelling in the neck. At first you may suspect meningitis due to swelling, rigidity of the neck and pain at the angle of the mandible, but the patient describes frequent dizziness and being light headed. This indicates there may be a restriction of blood flow to the brain. He also explains the swelling occurred in a matter of hours, and since then he has experienced fever and chills. Given these extra symptoms, what could it be?

Task 6. Patient presents with extensive swelling in the neck. At first you may suspect meningitis due to swelling, rigidity of the neck and pain at the angle of the mandible, but the patient describes frequent dizziness and being light headed. This indicates there may be a restriction of blood flow to the brain. He also explains the swelling occurred in a matter of hours, and since then he has experienced fever and chills. Given these extra symptoms, what could it be?

TOPIC: TREATMENT OF THE MAXILLOFACIAL INFECTION

Tests

1. There are principles in the management of odontogenic infections?

1) determine the severity
2) host defense
3) refer to specialist?
4) treat the infection surgically
5) support the patient medically
6) Choose and prescribe appropriate antibiotic
7) administer antibiotic properly
8) evaluate patient at least 2 days after surgical treatment

2. What are the indications for use of antibiotics?

1) acute - onset infection
2) diffuse swelling
3) compromised host defenses
4) fascial space involvement
5) severe pericoronitis (with purulence present)
6) osteomyelitis

3. What are situations in which antibiotics are not necessary?

1) chronic, well-localized abscess
2) minor vestibular abscess
3) dry socket (not an infection!)
4) root canal sterilization
5) mild pericoronitis
6) routine dental extractions in a nonimmunocompromised patient

4. What oral antibiotics are effective against odontogenic infections?

1) penicillin
2) amoxicillin
3) azithromycin
4) clindamycin
5) cephalexin
6) cefaclor
7) metronidazole

5. What is the usual recommended duration of antibiotic therapy?

1) 1 day after the infection has resolved
2) 2-3 days after the infection has resolved
3) mild-moderate infections with antibiotics will usually last 7 days

6. What are the reasons for treatment failure?

1) inadequate surgery
2) depressed host defenses
3) foreign body
4) antibiotic problems (non-compliance, drug not reaching site, dosage too low)
5) adequate surgery

7. Treatment of abscess

1) drainage
2) excision
3) antiseptic
4) irrigation
5) antibiotic treatment

8. Why provide incision and drainage?

1) drain pus
2) reduce tissue tension
3) improve local blood supply
4) provide a path of least resistance
5) alter the environment

9. What are the techniques for incision and drainage?

1) adequate anesthesia and pain control
2) obtain a specimen for Gm stain, culture, and antibiotic sensitivity test
3) incision into abscess cavity

4) blunt dissection
5) placement of penrose drain (leave 24-48 hours and pull after 2-3 days)
6) copious irrigation
7) all answers are right

10. Metronidozole is only effective against which type of bacteria?
1) aerobic
2) anaerobic
3) all answers are right

11. What is the adult dose for oral clindamycin?
1) 2g
2) 600mg
3) 500mg

12. What is the adult daily dose for oral cephalexin?
1) 1-2g
2) 600mg
3) 500mg

13. What is the adultdaily dose for oral azithromycin?
1) 2g
2) 600mg
3) 500mg

14. Systemic factors when assessing patient's need for antibiotics?
1) immunocompromised
2) diabetes
3) elderly
4) all answers are right

15. Anti-infectious agents:
1) antibacterials
2) antivirals
3) antifungals

Answers: 1 – 1-8; 2 – 1-6; 3 – 1-6; 4 – 1-7; 5 – 3; 6 - 1-4; 7 – 1-5; 8 – 1-4; 9 – 7; 10 – 2; 11 – 2; 12 – 1; 13 – 3; 14 – 4; 15 – 1-3.

Tasks

Tasks 1. What are the possible futures of infections once they gain access to the periapical bone?

Tasks 2. Give the stepwise pathophysiology of an odontogenic infection?

Tasks 3. Most commonly used antibiotics due to broad spectrum and low incidence of side effects?

Tasks 4. What is the easiest way to drain the buccal space? What do you need to beware of damaging?

Tasks 5. How do you treat a sublingual space infection?

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Maxillofacial infections

учебно-методическое пособие
на английском языке

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