FEDERAL STATE BUDGETARY EDUCATIONAL INSTITUTION OF HIGHER EDUCATION
NORTH OSSETIAN STATE MEDICAL ACADEMY OF THE MINISTRY OF HEALTH OF THE RUSSIAN FEDERATION
(ΦFederal State Budgetary Educational Institution of Higher Education SOGMA of the Ministry of Health of the Russian Federation)

Department of Surgical Diseases No. 1

PURULENT SURGERY

uchebno-metodicheskoe posobie dlya studentov meditsinskikh vuzov

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The manual reflects the methods of performing the main manipulations and methods of treatment included in the list of skills that a student should master in accordance with the program of studying the discipline "General Surgery". The manual is illustrated with original drawings.

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General issues of surgical infection

I.Definition of the concept of infection. Classification.

Infection is a complex dynamic pathological process that involves the introduction of pathogenic microbes into the body and multiplication in it, followed by the development of various active and responsive protective and pathological reactionsй. Any infectious process consists of the specific impact of pathogenic factors of microorganisms (enzymes, exo-and endotoxins) on the tissues, organs and organsystems of the macroorganism, as well as a complex of responses of the latter (inflammation, phagocytosis, immunity, allergies, etc.). The concept of "surgical infection" includes 2 groups of pathological processes:

- Infectious diseases and complications of accidental injuriesй, in the treatment of which xandpypmedical intervention is crucialическоевмешательство.
- Infectious complications that develop in the postoperativeperiod due to surgical intervention.

Currently, infectious pathology accounts for at leasthalf of all surgical diseases, and infectious complications account for 20-40% ofpostoperative complications, they occupy a leading place in the structure of postoperative mortality.

The category "acute surgical infection" includes allinfectious processes of various etiologies that have an acute onset andcourse of the disease, the main group in this category is acute purulent infection.

This includes such serious infectious diseases as tetanus, rabies, anthrax, gas gangrene. These diseases- are specific wound complications, each ofwhich is caused by one specific pathogen, causing changes in the body that are characteristic only of this pathogen and require specific treatment and prevention. Unlike other diseases of the "acute" group, these infections are not always treated surgically. The category "chronic surgical infection" includes diseases with a long-term chronic course with periods of exacerbation and remission (forexample, osteomyelitis).

A "chronic non-specific infection" refers to a chronicpurulent inflammation. Diseases of this group are often the outcomeof acutediseases, that have developedas авresultнесвоевременофuntimely andinadequateихtreatment. The most importantdiseaseofthe "specialцифическойsurgicalinfection"group istuberculosis,хирургитhe surgicalformsof whichoftendevelopyin patientswith thisinfection. ДостаточноредкимивхирургическомаспектеявляютсяSyphilis, actinomycosis, etc. are quite rare in the surgical aspect актиномикозидр. ДляTheseзабodiseasesare characterizedby chronicinflammationwiththepresenceof specificgranulomas, which arethe criterion fortheir morphologicaldiagnosis. Theseтидiseasesare subjectto mandatory specifictherapy, хирургическоеand surgicalinterventionтisoneof the componentsinthe complextreatmentofsomeformsof these diseases .

Amonginfectioussurgicalpathology, the leadingrole isplayed by acutepurulentinfection, нawhichaccountsfor the mainpart of surgicalinfectiousdiseasesandcomplications.

Classificationof surgicalinfection.Depending on the nature of the causative agent of the disease and the reaction of the human body, surgical infection is divided into **chronic** and **sharp**.In a chronicinfection, there are two types: specificand non-specific.

Chronicspecificinfection – tuberculosis, syphilis, actinomycosis, leprosy.

Chronic H.especificinfection- as a primary disease caused by any pathogenic microorganism or as an outcome of an acute non-specific infection.

Acute surgical infection:

- acute purulent non-specific infection (the causative agent is Staphylococcus, Streptococcus, Escherichia coli, Pseudomonasaeruginosa, as a monoinfection, but more oftenpolyinfection and in association with anaerobes);
- acute anaerobic infection (gas phlegmon or gas gangrene);
- acute putrefactive infection;
- acute specific infection (tetanus, anthrax).

According to the ability to form a capsule - spore-forming (**clostridial**) and spore-forming (**non-clostridial**).

Depending on the source of infection – **exogenous** and **endogenous**.

The exogenous sources of contamination in the hospital include: bacteria carrying among patients and staff, microbial contamination of air or operating room (**droplet infection**) microbial contamination of the surgeon's hands and operating field instruments, dressing material (**contact infection**), infection transmitted through various kinds of implantant (sutures, vascular and orthopedic prostheses) – **implant.**

II**. Acute purulent infection.**

Etiologyandpathogenesis. Allthe varietyof acutepurulentdiseasesванийandcomplications ofvariouslocalizationis unitedby a commonморфологическийmorphological substrate- acutepurulentinflammation, causedby the introductionand vital activity of pathogenic гноеродноймpurulent microflora. Likeиany infectiousprocess, acutepurulentinfectioninvolvesдлясвоей реализацииthe presenceof three components for its implementationкомпонентов:

- the causativeagent of infection (pyogenicmicroflora);
- entrancegate (placeandmethodof introductionof microbes);

- a susceptibleorganismwithitsreactions: localandgeneral,

protectiveandpathological.

I.Pathogensof purulentsurgicalinfection. The sources of surgical infection are very diverse. Endogenous infection is caused by microorganisms that colonize various tissues and organs of the patient. Among them, there are both normal inhabitants of the skin, gastrointestinal tract, upper respiratory and genital tracts, and microflora of various pathological foci. Exogenous infection is caused by microorganisms that have entered the patient'sbody from the external environment.It is primarily divided into:

- home infection that occurred outside the hospital;

- hospital infection that occurred during the patient's stay in the hospital for more than 48 hours, when there is no evidence that contamination occurred before hospitalization, or occurred after discharge, when there is evidence of possible microbial contamination in the hospital.

Exogenous sources of contamination in the hospital include: bacterialinfection among patients, microbial contamination of the surgeon's hands and the operating field, instruments, suture and dressing materials, the air of operating rooms, wards, dressings, etc.д. Hospital infection is characterized by multi-resistance of microorganisms to most of the antibacterial drugs used in this medical institution, and the spectrum of pathogens depends more on the profile of the hospital than on the localization of the lesion. Thus, the prevalence of hospital infections reaches 25-30% in intensive care units.

The mostfrequentcausative agent of acutepurulentinfectionisStaphylococcus, хотяalthough по-следнееrecentlytherehas been a progressivedecreasein itsseedingrate frominfection foci - itgiveswayto gramфлоре-negative flora, withwhichpойit is oftenfoundinassociation. PathogenicStaphylococcusaureusis widespreadinnatureandonтелеthe human body, it formsa numberof toxinsновандфакторовpermeability factors (hemolysin, нnecrotoxin, plasmocoagulase, hyaluronidase,etc.), cwhichis associatedwith itshighвиру-virulenceandadaptability. It is resistanttomost modern antibacterialdrugs, втомincludingкcephalosporins, , since most ofitsstrainsproduce B-lactamases-enzymes, разрушающиеthat destroy antibioticsof the penicillin andcephalosporingroups.

Pseudomonasaeruginosa— gram-negative bacillus, which is widelysaprophyticonthe mucousmembrane of the respiratorytractanddigestivetract, hasa highresistancetoantibioticsandotherантибактери-antibacterialdrugsandфакторамenvironmental factorscреды. It is one ofthe mostизimportantвозбудителейsources ofhospitalinfection.

Streptococcus, Escherichiacoli, proteusandotherenterobacteriaare alsocommonpathogensof severeformsof purulentinfection of softtissuesandinternalorgans. They haveaerobicandанаэробные anaerobicstrains, and are oftenfoundinassociationwithotherpathogens, вincludingcnon-clostridialanaerobes. Suchassociationscausesв очагputrefactivегнилостныйdecayof necrotictissues, oftenwithgas formation.

Purulentinflammationdepends not only onthe virulenceofthe microflora, determinedby the ability to produce toxinsandtissue-destroying enzymes, butalsoonthe numberof microbes, that have invadedthe tissues: purulentinflammationdevelopsata concentration $10^{of}105$-106^{6}microbialbodiesper 1 gram ofcarcassorexudate (criticalconcentration). However, inthepresenceof foreign bodies and ишемиитissue ischemiадляразвитиягнойноговоспаления достаточно, 100 microbial bodies are sufficient for the development of purulent inflammation микробныхтел. Ifthe bacterialcontaminationof the purulentногоfocus, exceeds10^{10}-10^{11}microbialbodies per 1 gramof tissues, реальнathere is a realthreatof generalizationof infection.

2. The entrance gate is closed .воротаинфекцииThe entrancegatesformicrobesareoftenевсегоvariousповрежденияskinandmucosal injuries (wounds, scratchesдины, ожо, burns , scratching, укусыinsect bites,etc.), sometimesnotnoticed by humans.

The roleof entrance gates can be playedby the excretoryductsof the sebaceous, потosweat, milk, salivaryandotherglands, whileэтомthe developmentвof the inflammatory process inthemпроцессais facilitatedbystagnationof secretionswitha violation of the physiologicaldrainageof the gland. ВозможноиэндогенноеразвитиеIt is also possiblethat theendogenousdevelopmentof the foci of GI infectionis dueпереносyto its transferfromочаговthe dormant infectionинфекции, имеющихсяfoci present in the body.

The growthandreproductionof microbesintheentranceворотgatearea is promotedby allfactors, that violatethe ownprotectivepropertiesof tissues: the presenceof necrotictissues, ,кровообращения, circulatory-disorders, cooling, generalнарушенияhomeostasis disorders (anemia, beriberi, variousиммimmunosuppressivestates,etc.)

3.Reactionof the microorganism. The most importantlocalprotectivereaction of the bodytoa purulentinfectionisinflammation (a complexнеспецинon-specific complexreactionof the connectivetissueandзаключенногов

нейthe microcirculatory bed enclosed in itруслato thepathogeniceffectof stimuli of variousnature).

ВнедрившиесявтканиPyogenic microbes embedded in the tissuesмикробысвоимитоксинамивы-зываютcause tissue alteration with their toxinsтканей, вответвыделяются, and inflammatory mediators are released in responseвоспаления, the mainoneскоторыхbeingvasoactivesubstances-histamineandserotonin. The releaseof mediatorsisпусковыма triggerforдалььfurthercascadingdevelopmentof allmanifestationsof the inflammatory response: expansionмикроциркуляторногоof the microcirculatorybed witha violationof the rheological properties of blood and increased permeability of the vessel walls, withthe development of exudation of the liquid part of the blood and emigration of shaped elements. The nature of the exudate quickly becomes purulent due to the high content of dead white blood cells and tissue destruction products in it, which is due to the action of microbial toxins.

The speed and intensity of development of all elements of inflammation in the focus of purulent infection depends on the virulence of the microflora and the specificreactions of the macroorganism. In some cases, against the background of a highly virulent infection, there is a rapid development of the inflammatory process with sharp edema, vascular thrombosis, necrosisof ischemic tissues (hyperergic reaction). In other cases, when the virulence of microbes is low, the inflammatory process is sluggish,and the clinical signs of inflammation are weak (hypoergic reaction) or barely noticeable (allergic reaction).An intermediate position is occupied bythe normal allergic type of reaction, in which all manifestations of the inflammatory process in the focus are well expressed and quite adequate to the existing infection. This type of reaction is most common.

During the development of an inflammatory reaction, a white blood cell wall is formed around the purulent focus, and then a granulation shaft is formed, which separatesthe purulent process from the surrounding tissues and preventsthe spread of infection beyond the primary focus. This restrictive tissue reaction is usually well expressed in thenormal course of the inflammatory process; it maynot have time to develop in thehyperergic reaction and may be weakly expressed in thehypoergic types of reaction. In these cases, there is a high risk of generalization of the process.

In the focus of purulent inflammation, phagocytosis is actively occurring -an important protective cellular reaction of the body, during which cells-phagocytes, neutrophilic leukocytes, a large group ofmononuclearmacrophages, giant cells-absorb and destroy microbialcells and other foreign agents.

Serum factors are involved in the mechanism of phagocytosis. Phagocytosis is accompanied by intracellular digestion of microbial cell antigens and culminates in the formationof humoral and cellular immunity, the most important factor of specific protection of the organismfrom infection.

The inflammatory process in the focus of purulent infection goes through 3 stages of development.

1.The stage of infiltration is the formation of a dense inflammatory swelling (infiltrate) with the presence of serous or serous-fibrinousexudate, diffusely impregnating the infiltrate.

2. Suppuration stage - the first appearance of pus in a dense infiltrate around the foci of central necrosis.

3. The stage of sequestration and purulent melting is the formation of a softening focus in the infiltrate размягчениwith cavities filledwith free pus and tissue sequesters (rejectedparts of necrotic tissue).

Clinical picture and diagnosis of acute purulent infection.

1. Local symptoms. Local manifestations of the developing focus of acute purulent infection are characterized by all the classic signs of acute inflammation, which are: redness, local hyperthermia, swelling, pain, and impaired function.

Redness of the integument is determined visually only with the surfacelocalization of the focus and is associated with the expansionof the microcirculatory bed (arterioles, capillaries, venules) under the influence of inflammatory mediators and metabolic disorders (acidosis). With deep localization of the focus of infection, there are no external manifestations of a vascular reaction; they appear as the purulent process approaches the skin withprogressive tissue destruction.

Local hyperthermia, determined by palpation, is caused by a vascular reaction and increased metabolic processes (catabolic reaction) with the release of energy. With deep localization of the focus of inflammation, an increase in local temperature can be detected by thermometry and thermography.

The swelling is caused by edema and cellular infiltrationof the inflamed area and is a clinical manifestation of the processes of exudation and migration of shaped blood elements against the background of increased permeabilityof the microcirculatory bed. Cellular infiltration is mainly represented by neutrophilic leukocytes, which in largenumbers die in the focus of infection, which gives the exudate a purulent character.

Pain syndrome manifests itself as a spontaneous pain sensation that occurs and progresses as the inflammatory process increases, as well as as a reaction to palpation, which should be carried out very carefully, gradually moving the palpating hand (or instrument) from the periphery of the focus to the center. The zone of maximum palpatory soreness corresponds to the area of the center of the focus of infection, where destructive tissue changes and the formation of purulent exudate are usually more pronounced. Pain is associated with an increase in interstitial pressure in the focus of inflammation, as well as with irritation of pain receptorsof microbial waste products and metabolic disorders in the focus.

Impaired function is caused by pain syndrome anddeveloping anatomical changes in tissues (edema, infiltration, necrosis).

The most important clinical manifestation of the third stage of development of purulent inflammation is a symptom of fluctuation (swell), definedin the zone of softeningof the infiltrate (with its localization) or in the entire infiltrate (in the presence of a cavity with pus in its deep parts). Detection of symptoms of softening and fluctuationis an absolute sign of the presence of a purulent cavity in the focus, whichdetermines the indications for urgent surgical intervention.

Among the additional research methods that help to recognizethe purulent cavity in the focus of purulent infection, ultrasound scanning and computed tomography are used.

In some cases, the usual X-ray examination (X-ray and radiography) helps, revealing free gas in the abdomen and fluid in the purulent cavities of the chest.

In doubtful cases, a diagnostic puncture of the focus is indicated, which is performed with a thick needle

(pus can be thick) under local anesthesia. The appearance of even a fewdrops of pus in the suction syringe is of crucial diagnostic importance and determines further active treatment tactics. Deep ulcers are effectively punctured under the control of ultrasound.

Depending on the severity of delineation processes in the focus of purulentinfection purulent inflammation in soft tissues canoccur in two forms:

- Abscess (abscess) delineated accumulation of pus. It can develop in any tissues and organs.
- Phlegmon — diffuse (not delimited) purulent inflammation of the cellular spaces. It develops in those anatomical formations where there is a pronounced layer of fiber, along which purulent infiltration spreads without a tendency to delineate: subcutaneous, intermuscular adipose tissue on the trunk and limbs, cellular cases around the abdominal organs, cellular layers of the walls of the abdominal cavity organs, etc.

With any localization of the purulent focus, any form and stageof development of the inflammatory process, it is possible to spread the infection in various ways beyond the primary focus.

The contact pathway is the involvement of tissues adjacent to the focus in the pathological process if the local boundary reaction is imperfect. The contact pathway is most pronounced in the phlegmonous form of inflammation, whenthe inflammatory infiltration quickly spreads through the layers of fiber along the fascia, blood vessels and nerves.

The lymphogenic pathway is carried out when pathogenic flora spreads through the lymphatic slits through the lymphatic vessels to the regionallymph nodes closest to the focus and beyond, ifthe protective mechanisms of the lymph nodes do not block the infection. Along the course of the lymphatic vessels and in the lymph nodes, a picture of inflammation develops-lymphangitis and lymphadenitis.

The hematogenic pathway of infection occurs whenmicrobes enter the blood (bacteremia) through the venous kneeof the microcirculatory bed destroyed in the focus or by the development of acute thrombophlebitis in the focus. In the latter case, it is possibleto detach and circulate the blood of infected thrombotic masses with the development of a thromboembolic process with the formation of secondary (metastatic) purulent foci in various organs.

Any type of spread of infection beyond the primary focus can mean the beginning of generalization of the infection, so it requires the most intensive local and general treatment.

2. General reaction of the body. Common manifestations of the macroorganism's reaction to the presence of a purulent infection focus are the syndrome of a systemic inflammatory reaction (SIRS) caused by the circulation of cytokines, microbial toxins, numerous toxic products of tissue breakdown, and metabolic disorders in the purulent inflammation focus.

Clinical manifestations of general intoxication include weakness, poor appetite, headache, general malaise, and sleep disorders.

The most cardinal and constant symptom is fever-an increase in body temperature. A significantincrease in body temperature is most typical, especially in the evening hours with daily fluctuations of more than 1 ° C. In the case of processes in the infiltration stage, anincrease in temperature is usually not accompanied by large daily-fluctuations. With the appearance of free pus, the development of sequestration processes in the focus and purulent melting with the formation of cavities, the temperature takes on a hectic character with daily ranges of 1.5-2°C, with chills when the temperature rises and sweating when it falls to normal or subfebrile numbers.

Characteristic changes in blood tests:

Neutrophilic leukocytosis with a left shift in the leukocyte formula.

Relative or absolute lymphopenia and monocypenia.An absolute decrease in the number of mononuclears is prognostically unfavorable, as it indicates the depletion of protective immune mechanisms. Anemia.Increased ESR. Dysproteinemia - an increase in the number of globulins, especially the gammafraction,a relative decreasein the number of albumins.

Blood cultures for sterility - can reveal bacteremia,especially with repeated studies at the height of fever. An increase in the blood content of "medium molecules" - a universalmarker of the degree of intoxication. Medium-sized molecules - productsof incomplete cleavage of protein bodies of various nature-polypeptides with a molecular weight of 500-5000 . Changes in urine tests: proteinuria, cylindruria, sometimesleukocyturia.

General principles of treatment of acute purulent infection. Treatment of acute purulent infection consists of local treatment of the focus of infection and general treatment aimed at eliminating pathological changes in the macroorganism.

1. ***Local treatment*** of the focus of infection is determined by the stage of development of the inflammatory process.

At the stage of infiltration, when it is possible to completely reverse the development of the process by resorbing it, conservative treatment is carried out aimed at eliminating the infection and resorbing the infiltrate.

Local administration of antibiotics is used in the form of pricking the infiltrate with an antibiotic solution in 0.25-0.5% novocaine solution. The solution is injected into the soft tissues *around* the lesion, which can be punctured to detect the possible presence of pus.

For resorption of the infiltrate, physiotherapy procedures are used: a) UV radiation; b) UHF in wave

mode without heat sensation; c) iontophoresis withantiseptics, enzymes, anesthetics, etc.; d) semi-alcohol wet-drying bandages.

The above complex of conservative treatment canbe used to treat superficially located foci of infectionthat are accessible to visual and palpatory control. In the presence of deep foci of infection, when it is difficult or impossible to reliablyределение determine the stage of development of the process, local treatment consists inapplying cold against the background ofgeneral antibacterial therapy.

In all forms of acute purulent infection, the use of heatis contraindicated, являющего, which is a catalystfor enzymatic processes and contributes to the progression of destructivetissue changes in the focus, as well as purulent spread of pus beyond its borders.

In the stage of purulent melting and sequestration, the main mandatory component of local treatment is surgical intervention,performed in an emergency or urgent manner: opening and sanation of the purulent focus. The main principles of surgical treatment of the focus of octroi purulent infection are:

1) radical surgical rehabilitation of the lesion with the use of additional means that reduce its microbial contamination;

2) adequate drainage of the lesion in the postoperative period withlocal use of antibacterial agents;

3)an earlier closure of the surgical wound is possible. Surgical intervention involves:

- adequate analgesia (conduction or general anesthesia);
- rational wide access (incision), performed in compliance with the necessary anatomical and cosmetic requirements, ensuringcomplete evacuation of pus, opening of all pockets and congestion, full visual or palpatory revision of the cavity andperforming subsequent medical procedures in it;
- taking material for bacteriological examination;
- necrectomy, possibly a more complete removal of dead and non-viable tissue from the focus;
- treatment of the cavity with additional meansthat reduce microbial contamination (vacuuming,pulsingfluid treatment, ultrasoniccavitation through an antibiotic solution, UV radiation, laser irradiation, etc.);
- complete hemostasis.

Surgical intervention in the appropriate volume reducesthe microbial contamination of the focus to the level of 101 microbial bodies per I gram of tissue and below. For various reasons, it is not always possible to perform a single-stage radical necrectomy, which determines the lack of radicality of the entire operation.

2. **Generaltreatment**of purulentinfectionis carriedout inseveralниях: directions : antibacterialtherapy, detoxification, immunocorrection, and syndrome therapy

Antimicrobialtherapy isperformed withallmodernпрепаратамидrugs , the mainoneскоторыхbeingantibiotics, as well as thereare alsopreparations of the sulfonamide group, metronidazoleand sul-anilamidesаниламидами. Treatment with antibiotics is carriedcout taking into accountthe sensitivityof the microflora, the possibilityof side effectsandinteractionwithotherdrugs.

Beforedeterminingthe natureмикрофof the microfloraanditssensitivitytoantibacterialdrugs, it is advisableto conductempirical therapy with eitherантибиотикамиfirst- line antibiotics(полусинтетическиеsemi-synthetic penicillins and aminoglycosides),ormotor therapy withcephalosporins of the II-IIIgeneration. Лишьприособотяжелойинфекцииинеэффективности другихпрепаратовиспользуютсяReserve antibiotics (fluoroquinolines and carbopenems) are used only in cases of particularly severe infection and ineffectiveness of other drugsантибиотики (фторхинолиныи карбопенемы. Therapyis correctedafterreceivingданныхdatafrom a bacteriological study.

Detoxificationis most oftenвсегоperformed by intracorporeal administrationof drugsbased ondilutionandneutralizationof toxic substances in the patient's bodyc, followedвыведениемby their removalby naturalroutes. Thesemethodsincludeheavydrinking, infusiontherapy (transfusion of crystalloid, glucose-activated solutions and detoxifying blood substitutes), as well as forced diuresis against the background of controlled hemodilution. Methods of extracorporeal detoxification can also be used: hemosorption, lymphosorption, plasmapheresis, and blood UFO.

Immunocorrection is performed with drugs that stimulate the immune system (T-activin, thymoline. thymogen), as well as replacement therapy with passive immunization agents (hyperimmune plasma. y-globulins, interferon, blood components)

Syndrome therapy consists in the treatment of various pathological syndromes that develop against the background of purulent-resorptive phenomena. These syndromes are: respiratory failure, hepatic-renal failure, coagulopathy, etc.

PURULENT DISEASES OF THE FINGERS AND HAND.

Panaritium.

Panaritium is a purulent-inflammatory disease ofthe anatomical structures of the finger. Panaricium in ancient Greekmeans "edema", "leak", i.e. "suppuration".

Currently, the modified classification of panaritia by G. P. Zaitsev (1938), based on the anatomical principle, is

most widely used in clinical practice. Depending on the depth of palatal tissue damage, superficial and deep forms of panaritium are distinguished.

Surface forms of panaritium:

1. Skin panaritium
2. Paronychia
3. Subungual panaritium
4. Subcutaneous panaritium

Deep***forms of panaritium:***

1.Bone panaritium
   a) acute
   b) chronic (fistulous form)
2. Tendon panaritium (purulent tendovaginitis)
3. Articular panaritium
4. Osteoarticular panaritium
5. Pandactyl

The most common cause of panaritium is microtrauma.People of the most able - bodied age-20-50 years-are more likely to get sick.Men predominate among the patients, which is associated with a high frequency of injuries to the fingers of the hand.

Доминирующей инфекциейStaphylococcus aureus has been the dominant infection in the development of purulent diseases of the fingers andpussy in recent decades, although in recent years therehas been an increase in non-staphylococcal infections: Streptococcus, gram-negative flora, and aerobic-anaerobic associations.

Anatomy of the fingers and hand.

Features of the clinical course of panaritium are largely determined by the anatomical structures of the fingers of the hand.

The powerful development of all skin layers and stitching them in different directions withelastic fibers and collagen bundles well protect the deep layers of the finger from the penetration of microbes and thereby perform the function of a sewnbarrier. But this also has a negative side in the case of microtrauma penetration of microorganisms under the skin and the development of suppuration-dense, sharply thickened skin prevents the release of pus to the outside.

Dense strands in the subcutaneous fat tissue of the hand and fingers are located perpendicular to the surface of the skin of the fingers, attaching to the deep layers of the skin andperiosteum, forming almost closed cavities and significantly limiting the mobility of the skin. In connection with this purulent process in the subcutaneous fat layerspreads not on the surface of the fingers, but in depth. A well-developed network of lymphaticvessels contributes to the rapid spread of infection and the development of lymphangoitis and lymphadenitis.

The structure of the tendon sheaths of the middle fingers (II,III,IV) and extreme (Iand V) fingers is different. On the palmar surface, tendon sheaths IIof fingers II ,IIIand IV start from the base of the distal phalanges and extend inthe proximaldirection to the level of the metacarpophalangeal joints, ending afterin the area of the corresponding palmar elevations. Theseти tendon sheaths are isolated from each other. The tendons of the superficial and deep flexors of the V finger are surrounded by the vagina from the middle phalanx to the transverse ligament of the forearm, forming the ulnar synovial sac-bursa. An analogous-sheath is the radial one at the long flexor tendon of the first finger, which starts from the base of the distal phalanx and extends to the transverse ligament of the wrist, ending blindly in the Pirogov-Paron space. According tothe literature, these two vaginas in 10 - 15% of cases communicate with each other bymeans of a slit, which can lead to a rapid spread of the inflammatory process with the development of U-shaped phlegmon of the hand.

CLINIC, DIAGNOSIS AND TREATMENTSEPARATE FORMS OF PANARITIUM.

Surface forms of panaritia.

Cutaneous panaritium is the most favorable type of panaritium. The focus of inflammation is

localized intradermally, between the papillary layer and the epi-dermis. Patients complainof minor pain in the area of the affected finger. Onexamination, the presence of an epidermal bladder with purulent exudate is noted.

Treatment consists in excising the exfoliated epidermis with scissors along the border of the detachment. Anesthesia is not required, but it is necessary to avoid touching the instrument to the bottom of the wound, which is very painful. After treating the wound with an antiseptic, apply a bandage with a water-soluble ointment (levosin, levomekol, etc.).

Paronychia is an inflammation of the periarticular roller. The most common causeof occurrence is infectionof the skin of the periarticular roller damaged during manicure. Paronychia occurs in women 5 times more often than in men.

On examination, hyperemia and edema of the parotid roller, moderate overhanging of it over the nail plate are noted. With a chronic course, pathological granulations appear. When pressing on the periungual roller, a drop of pus is released from under it.

With early treatment of the patient and the absence of signs of purulentinflammation, conservative therapy is prescribed. Alcoholdressings are most effective 2-3 times a day, sometimes in combination with antibiotic therapy. If conservativetreatment is ineffective and the inflammatory process progresses, surgery is indicated.

Subungual panaritium. БольThe patient is disturbed by very intense bursting, throbbing pains inthe area of the nail phalanx of the affected finger. Instrumental palpation with a button probe of the palmar surface of the phalanx is painless, and pressing on the nail phalanx causes sharp pain.

The nail plate in the area of pus accumulation has a characteristic whitish-yellowish color, palpation shows its swell.

Treatment of subungual panaritium consists in partial or complete removal of the nail plate under anesthesia according to Lukashevich-Oberst.

Subcutaneous panaritium is the most common purulent behindsore fingers. In 85% of cases, the inflammatory processis localized on the palmar surface of the distal (nail) phalanges of the fingers.

Subcutaneous panaritium has a fairly vivid clinical picture. Themain complaintis severe, bursting pain in the affected phalanx of the finger. The intensity of pain increases, the patient loses rest and sleep. The painbecomes throbbing and increases when the arm is lowered down. Often only after the first "sleepless night" the patient turns to the surgeon.

Onexaminationpe, edema is noted
the affected phalanx, slight hyperemia of the skin in the area of the purulent focus, smoothness of the skin pattern. The generalcondition suffers moderately, and the temperature may rise to 37.5-38 degrees.

In the early stages after the onset of subcutaneous panaritium, in theabsence of symptoms of purulent inflammation, консерватившconservative therapy is possible. Alcohol bandages on the finger are effective 2-3 times a day, anti-biotic therapy. In the absence of a positive effect of therapyfor 1-2 days, surgical treatment is indicated.

When the nail phalanx is affected, the optimal access is a club-shaped incision. When the purulent focus is located on the middle or main phalanges, unilateral or bilateral mediolateral access is optimal, passing along the neutral line of the lateral surface of the finger.

Deep forms of panaritium.

Bone panaritium ranks second in frequency after subcutaneous and in 95% of cases develops secondarily, as a result of the transition of the purulent-necrotic process from the surrounding tissues to the bone

The clinical pictureof bone panaritium is largely similar to the clinic of subcutaneous panaritium, thepatient's general condition can suffer significantly, the body temperature sometimes rises to 39 degrees.

On examination, edema of the nail phalanx is detected, hyperemia may be absent, and the skin is often colored after multipleapplications of potassium permanganate solutions (iodine, diamond green). There is a purulent wound after previously performed surgicalintervention, often already transformed into a purulent fistula.

Theradiograph shows bone destruction. It should be noted that destructivechanges in bone tissue become visible on X-rays only for 9-14 days from the onset of the disease. Therefore, in the clinical picture of bone panaritium, despite the absence of radiological signs of bone destruction, surgical treatment is indicated.

Surgical treatmentof bone panaritium uses the samesurgicalapproaches as for subcutaneous panaritium. The volumeof surgical treatment of the purulent focus increases due to the need to removenecrotic areas of bone tissue.

Tendon panaritium-an inflammatory process affects the dryvein vagina and flexor tendons of the

fingers of the hand. There is a primarytendon panaritium (tendovaginitis), in which the tendon vagina is directly infected by stab or stab wounds that penetrate the vaginal cavity, and *a secondary* one that occurs as a complication of other forms of panaritium (bone, bone-joint).

Clinical picture. The disease begins acutely, with severe, painfulpains in the area of the affected finger. Sometimes the phenomenaof general intoxication are significant. On examination, diffuse edema of the entire finger is noted, sometimes more pronounced on its back surface. Hyperemia of the skin is insignificant, sometimes with a cyanotic tinge. The finger is inthe bent half-bent position. This is due to the fact that in thisposition the volume of the tendon sheath is maximal. Active movements in the finger are impossible, passive ones cause excruciating pain. A pathognomonic symptom is pain in the projection of the blind end of the tendon sheath in the area of the corresponding palmarelevation.

In the surgical treatment of tendonpanaritium, its timeliness is very important, since the delay in the operation leads toirreversible necrosis of the flexor tendon and irretrievable lossof palatal function. Incisions on the finger are made along a neutral mediolateral line on the middle and main phalanges.

With tendon panaritia of the II, III and IVfingers, an incision is required in the area of the corresponding palmarelevation with the opening of the blind end of the tendon sheath. Ifthere are purulent congestion in the subcutaneous tissue of the phalanges of the fingers, they are sanitized and drained. Under favorable conditions, the wounds can be sutured with rare primary sutures. An extremely important pointis the immobilization of the hand.

Purulent tendovaginitis of the second finger. Condition after surgery

When the tendon panaritiums 1 or V fingertips with the development of the radial or ulnar Toneburst tendon sheath reveal similar, but in some cases, during surgery detect non-viable or nekrotizirovanne the flexor tendon of a finger (no glitter, gray-green color, no bleeding, Rosvodokanal, etc.). In this case, it is necessary to excise nekrotizirovanne tendon, and after the reorganization of the wound suturing its primary suture. For a more adequate visual revision of the tendon, wider operative approaches are used (for example, a zigzag-like incision on the palmar surface of the finger according to Brunner).

Articular and osteoarticular panaritium is an acute purulent lesion of the interphalangeal and metacarpophalangeal joints of the hand. The clinical picture of articular panaritium is quite characteristic. Patients report severe pain in the area of the affected joint, first when moving in the joint, and then at rest. Pain leads to a violation of the function of not only the finger, but also the entire hand. On examination, there is a thickening of the finger in the areaof the gusset, which gives it a spindle-shaped shape. The skin above the joint is strained, the skin folds on the back surface are straightened, thereis a slight hyperemia of the skin. Palpation in the joint area, signs ofpassive movements are sharply painful. With axial load on the palec, pain in the joint is noted. Рент-Radiological changes in the joint usually appear 10-12 days later than the clinical symptoms.

With osteoarticular panaritia, a purulent fistula on the back of the joint (fistulous form) is often detected. The finger is half-bent, spindlythickened, active movements are absent, passive movements are sharplyrestricted. With passive movements in the joint, a characteristic bone crepitation is determined.

Pandactyl is a non-specific purulent-necrotic disease of the finger that spreads to at least two phalanges and affectsthe skin, subcutaneous tissue, joint, tendon and bone. Pandactyl is the most severe purulent-necrotic disease of the fingers of the hand.

The inflammatory process in pandactyl is more often characterized by wet necrosis with melting of tissues. The clinical pictureis characterized by a sharp thickening and enlargementof the finger in volume. The skin of the finger is sharply tense, cyanotic with a purple tint. The finger is half-bent (with necrosis and sequestrationof tendons, the finger straightens), there are no active movements,passive ones are sharply painful. Almost always, pandactyl is accompaniedby the development of regional lymphadenitis and lymphangitis. The pain is intense and excruciating for a long time. The general condition of patients due to intoxication worsens.

Body temperature rises, sleep and appetite are disturbed. The disease can be complicated by sepsis.

The scope of surgery for pandactyl depends on the affectedanatomical structures, i.e. surgical interventions described for individual forms of panaritium are used. As a surgical approach, you can use one mid-lateral (mediolateral) incision for the entire length of the finger with an arc-shaped extension to the palm in the area of the blind end of the tendon vagina. The principles of postoperative management are similar to those of other deep forms of panaritium, but the importance of adequate massiveantibacterial therapy is significantly increasing.

GENERAL PRINCIPLES OF TREATMENT OF PANARITIUM.

The key to success in the treatment of purulent infection of the fingers and hand is timely and adequate surgical intervention.

When performing operations to treat purulent diseases, the most optimal strategy is the following:поводу гнойных заболеваний наиболее опти:

1. The incision should provide a complete revision and rehabilitation of the purulent focus and at the same time be gentle, allowing you to get a good functional and aesthetic result in the end.

2. After evacuation of pus, it is necessary to perform a fullnecretomy, focusing on the color and structure of the tissues. Surgical treatment of the purulentfocus should be carried out taking into account the importantanatomical formations located nearby, in order to avoid their damage.

3. For a more radical removal of the purulent-necrotic focus during the operation, it is rational to vacuum the wound, treat it with low-frequency ultrasound, a pulsating stream of antiseptic, etc. methodsof additional self-assessment.

4. Purulent wound should be drained after surgical treatment. For this purpose, one or severalperforated PVC tubes of various diameters (depending on the size and configuration of the cavity) are placed on the bottom of the wound cavity. The free ends of the drains are removedthrough additional punctures of the skin to the outside. The drainage system, referred to in practice as the " drainage and flushing system "(DPS), operates from 3 to 14 days depending on the size of the cavity and the course of the wound process.

5. After a full-fledged surgical treatment of the abscess, in the absence of pronounced perifocal inflammation of the surrounding tissues, mobility of the wound edges and its active drainage, primary sutures can be applied to thewound.

Suturing the wound (primary, primary-delayed, early or late secondary) reduces the duration of treatment and improvesfunctional and aesthetic results. An important aspect of successful treatment of panaritium is the timeliness of surgical intervention. Only early surgical treatment prevents the progression of the purulent-destructive process and reduces the duration of treatment. It should be emphasized that the operation should be performed in strict compliance with all the rules of asepsis and antiseptic provisions, with a changeof surgical underwear, gloves and tools after surgical treatment of the purulent focus.Before the operation, it is necessary to wash household dirt under running water and soap for 3-5 minutes.

The surgical field is treated according toFilonchikov-Grossh with 96-degree alcohol and 10% alcohol tincture of iodine up to the elbow joint.

Pain relief. The most common typeof anesthesia for hand palsy operations is **Lukashevich-Oberst**anesthesia .In this type of anesthesia, 1-2 ml of 1% novocaine solution is injected into the base of the finger from both sides. Anesthesia occurs in an average of 5 minutes.

If the pathological process is localized on the main phalanx, Lukashevich-Oberst anesthesia is unacceptable and the method of choice is conduction-infiltration anesthesia according to E. V. Usoltseva or other types of conductionanesthesia. In some cases, the use of intravenous regionalanesthesia is acceptable. In case of intolerance to local anesthetics, intravenous anesthesia is indicated.

Online accesses. Over the last hundred years, there has been a significant evolution of views on-operational accesses in the opening of panaritia. If earlier it was considered optimal to make an incision in the projection of the abscess along the shortest path, regardless ofthe location of the abscess, now it is considered unacceptable to use incisions through the palmar surface of the fingers of the hand.

When opening an abscess on the nail phalanx of the finger, the most optimalis a semi-oval ("club-shaped") incision, sufficient toperform adequate necrectomy. На средней и основной фаланге чаще при-Paired or unilateral mid-lateral (mediolateral) incisions are more often used on the middle and main-phalanx. When these incisions are made in the middle of the lateral surface of the finger, there is lesslikely to be damage to the neurovascular bundle of the finger. Mediolateral incisions can be used both for opening thesubcutaneous and for opening the tendon panaritium, however, in the surgical treatment of tendon panaritium, it is necessary to open the "blind end", the tendonvagina in the area of the palmar elevation withtendonitis of the fingers II-IV.

The most optimal access for opening the blind end of the tendon sheaths of the II — IV fingers in the area of the palmarelevations is an arc-shaped incision no**Bunnell.**

Scope of the operational allowance. Surgical treatment of a purulent focus is the most responsible and difficult stage of the operation. Tissue remaining after necrectomy of questionable viability can cause a prolonged courseof the disease. At the same time, excessive radicalism in such a complex anatomicalarea as the fingers of the hand can cause irreparable damage to its function. The volume of surgical aid in the purulent focus of panaritiavaries depending on the localization of the lesion and the degree of destructionof anatomical structures.

With bone panaritium, excision of the purulent focus within healthy tissues is indicated. With total sequestration, it is even possible to remove the entire nail phalanx bone. Careful treatment of the skin andthe use of a primary suture with a drainage-washing system allows you to preservethe bone-free phalanx with a good aesthetic and functional result.

The proximity of the tendon sheath on the middle and main phalanges does notalways allow excising the purulent focus. However, additional methods of rehabilitation (vacuuming, low-frequency ultrasound treatment) and adequate drainageof the wound contribute to its rapid healing.

Postoperative management. An extremely important aspect of postoperative management is the immobilization of the hand, which is carried out at the end of the operation onthe operating table and continues until the inflammatory phenomena subside. The hand is fixed using a palm or back plaster splint, whichis applied to the hands after applying a bandage to the wound, in a functionalposition. The hand is given a slight back flexion, the fingers are slightly bent, and the first finger is opposed to the third finger. After the inflammatory phenomena subside, immobilization stops andthe development of the fingers of the hand begins to prevent stiffness and contractures of the hand. The development of the fingers of the hand is facilitated by thermal procedures (applications of paraffin, ozokerite) and physiotherapy procedures (electrophoresis withlyophase, potassium iodide, phonophoresis with hydrocortisone).An important aspect of postoperative management of the patient isthe possibility of washing the wound cavity through installed drainage-tubes (microirrigators) with antiseptic solutions. Washingis carried out daily in bandages. For washing wounds, 0.2-0.05% aqueous solution of chlorhexidine or 0.5-1% dioxidine solution is more often used. When the inflammatory phenomenon subsides, the drains are removed. Improving the results of surgical treatment can be achievedby rational antibiotic therapy. The main condition for successfulantibacterial therapy is the creation of a high concentration of an antibiotic and its prolonged stay in the focus of inflammation. An importantelement is selective antibiotic therapy, according to the data obtainedon the sensitivity of the identified microflora to antibiotics.

Pyoderma (folliculitis, furuncle, furunculosis).

Folliculitis *is* an acute purulent inflammation of the hair sac. The inflammatory process is limited to thehair sac and if it is localized only around the exitca, то называется site of the hair, it is called folliculitis. Like other superficial local pyoderma, the disease does not requiresurgery and is treated by a dermatologist

A boil *is* an acute purulent-necrotic inflammation of the hair follicle with all its appendages (the sebaceous gland and the muscle that lifts the hair). The closest parts of the skin and

subcutaneous tissue may be involved in the inflammatoryinfiltrate. The causative agent of the disease is more often Staphylococcusaureus.

Boils develop in any part of the body where there is a hairline. In men, boils occur about 10 times more often than in women, which is due to the peculiarities of metabolism.

In the initial stage of infiltration, a dense painful cone-shaped infiltrate is formed around the affected hair sac, which is the entrance gate of infection and the center of the purulent process. In the stage of suppuration, the necrotic hair follicle forms a purulent-necrotic "rod", clearly visible in the center of a dense inflammatory infiltrate. In the stage of sequestration and purulent melting, the rod is rejected, and a small purulent wound that has formed in its place healsby secondary tension, leaving behind a barely noticeable scar.

When a significant area of tissue around the hair sac is involved in the inflammatory infiltrate, a significant purulent cavity is formed. Such a boil is called an abscessed*boil*, and it is clinicallyassociated with more pronounced general symptoms of intoxication. Severe course of boils with severe pain and edema is observed when they are localized on the face, especially dangerous boils in the nasolabial triangle, nose and suborbital area, which can be complicated by purulent basal meningitis due to thrombosis of the cavernous sinus.

Treatment of boils in the stage of infiltration is conservative, under the influenceof which it can completely resolve. In the stage of suppuration and especially sequestration, puncturing the infiltrate with a solution of novocaine with antibiotics is effective. In the process of this manipulation, the necrotic rod usually either departs on its own, or is easily removed with a tool.

An abscessed boil is treated **promptly** and corresponds to the general principles of treatment of purulent processes: in the stage of purulent melting, an incision is performed, necrectomy, removal of pus withsubsequentopen wound management until healing. Residual infiltrate quickly resolves under the influence of physical therapy (UHF).

Furunculosis - multiple boils that occur simultaneouslyor sequentially on different parts of the body. It usually develops against the background of various general metabolic disorders in the body (beriberi, caxapdiabetes, hormonal disorders, anemia, etc.). Forsuccessful treatment of the disease, in addition to local effects on each boil, it is necessary to identify and eliminate the background pathology, without which the process is prone to relapse.

Treatment of boils and furunculosis. Uncomplicated forms of diseases can be treated on an outpatient basis, patients with facial boils and with the development of complications are subject to hospitalization. Local treatment consists in creating rest, applying stickers that protect the area of inflammation from mechanical impact, performing a toilet of the skin around the focus – wiping with 70: alcohol or 2% salicylic alcohol, smearing with 1-3% alcohol solution of methylene blue, diamond green, etc. It is strictly forbidden to squeeze out the contents of the boil and do a massage in the area of the focus of inflammation. If the patient has a high fever, systemic antibacterial and detoxification therapy is necessary.

With the development of complications, surgical intervention is indicated – opening the focus of infection.With furunculosis, each focus is treated according to the usual method. Use immunomodulators and subcutaneous administration of staphylococcal vaccine or immunization with staphylococcal toxoid.

The carbuncle.

Carbuncle is an acute purulent-necrotic inflammation of several adjacent hair follicles involving the surrounding soft tissues (skin, subcutaneous tissue, sometimes fascia and muscles). With a carbuncle, a single general inflammatory infiltrate is formed, and quite often the phenomena of lymphangitis and lymphadenitis are added. The causative agent of the disease is usually Staphylococcusaureus

Clinically, the carbuncle is much more severe than the boil: locally, there is a large accumulation of sharply painfulfoci of inflammatory infiltrate with hyperemia and cyanosis of the skin, followed by the formation of necrosis. Necrotic tissues partially undergo purulent melting and rejection. The superficial fascia covering the muscles in the area of inflammation may also be involved in necrosis and decay. After rejection of necrotic masses, the wound is granulated and heals by secondary tension with the formation of a rough scar.

The most frequent localization of the process is the back of the neck, back, occipital region, face. At the beginning of the disease, a small inflammatory infiltrate appears, rapidly increasing in size. There is marked soreness, tissue tension, the skin becomes purplish-bluish, the skin becomes thinner and breaks in several places, purulent contents enter through the breakouts - a symptom of"sieve".

Over time, the pinholes merge into one, forming an extensive purulent-necrotic wound with abundant purulent discharge. The disease is usually accompanied by intoxication phenomena, which regress after the breakthrough of the purulent focus.

Treatment. All medical measures are carried out only in a surgical hospital.

In connection with severe intoxication and the danger of early generalization of the process, early surgical intervention is indicated, which is carried out already at the stage of infiltration.

A wide cross - shaped or H-shaped incision is made through the entire thickness of the carbuncle, after which the entire skin-subcutaneous purulent-necrotic block is excised in layers. Then, the looseness is tamponized with ointments on a water-soluble basis and is kept open until the wound is completely cleaned and performed by granulation. Antibacterial drugs are prescribed, and infusion detoxification therapy is performed.

Hydradenite.

Hydradenitis isan acute purulentinflammation of the apocrine sweat glands. The disease is caused by staphylococci, predispose to hydradenitis hyperhidrosis (increased sweating), abrasions, diaper rash, endocrine disorders. The infection penetrates through the excretory ducts and causes inflammatory phenomena. In the vast majority of cases, the pathological process is localized in the axillary region.

Clinically, hydradenitis is manifested by a feeling of itching, soreness and a feeling of tension in the area of the formed dense node, 0.5 – 1.0 cm in diameter, the skin above it is purple-red. After 3-7 days, the node softens, a fluctuating abscess forms. When it is opened spontaneously or promptly, a creamy pus is released. The cycle lasts 10-15 days. With improper treatment or spontaneous autopsy, it often recurs.

Treatment. Conservative treatment is used in the initial stages of the development of the disease: intravenous or oral administration of cephalosparin antibiotics, vitamin therapy, immunomodulatory therapy (immunal, T-activin, thymalin, thymagen, etc.) and physiotherapy – UHF, UFO, laser therapy on the lesion site. In case of ineffectiveness of conservative measures or the development of purulent melting of the infiltrate, surgical treatment is indicated – opening the abscess. The operation is performed under general anesthesia, or under local anesthesia. Further, the wound is conducted in an open way according to generally accepted rules.

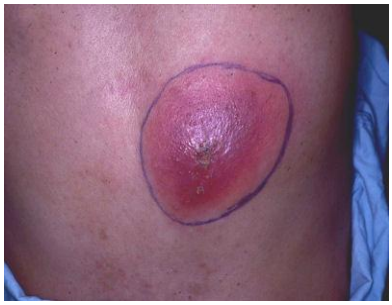
An abscess.

Abscess (abscess) – a limited accumulation of pus in tissues and organs with the formation of a cavity. The ways of infection penetration into tissues and organs are diverse: from the external environment through abrasions, wounds and injections, from other organs by hematogenic or lymphogenic routes. The causative agents of abscess in monoculture are more often staphylococcus or in association with any other infection.

With this disease, purulent inflammation develops with melting of tissues and the formation of a cavity containing tissue detritus and pus. In the circumference of the abscess, a pyogenic capsule develops, covered from the inside with granulations. The pyogenic envelope is an obstacle to the spread of infection to the surrounding tissues, which reduces the phenomenon of intoxication.

Manifestations of an abscess depend on its location, size, and duration of the disease. The general appearance of the abscess corresponds to signs of intoxication.

Local symptoms correspond to the classic signs of inflammation and are most clearly manifested in the superficial localization of the pathological process. One of the characteristic symptoms is fluctuation or swell, but it can be negative with small volumes of abscess.



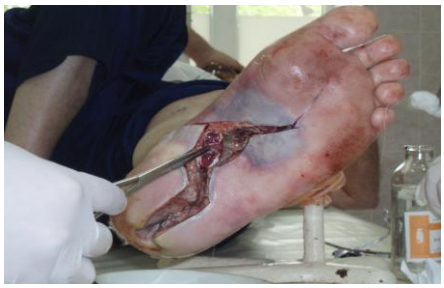
The following complications of abscesses are possible: an abscess bursts into neighboring organs and cavities with the development of peritonitis, purulent pleurisy or arthritis, in advanced cases, sepsis may develop.

Treatment. First of all, under general anesthesia, the abscess cavity is opened and drained. In the future, the wound is treated in an open way, as a purulent wound with the useofhyperosmolar preparations, enzymes and antiseptics. In case of severe intoxication, detoxification and antibacterial therapy (antibiotics and sulfonamides) is used. The selection of antibacterial drugs is carried out according to generally accepted rules, taking into account the sensitivity of microorganisms to antibiotics.

Phlegmon.

Phlegmon is an acute purulent inflammation of adipose tissue. Depending on the localization of the purulent-necrotic process, phlegmon of the limb is distinguished, phlegmon of the neck – adenophlegmon, phlegmon of the axillary region – axillary phlegmon, subpectoral phlegmon – inflammation of the fatty tissue of the pectoralis major muscle, purulent mediastinitis – damage to the mediastinal fiber, damage to the parotid fiber - paranephritis, involvement in the pathological process of pararectal fiber – paraproctitis.

Unlike an abscess, phlegmon does not develop delineation of the process due to the pyogenic capsule, pus permeates, inhibits tissues and can spread through the intermuscular, fascial and neurovascular spaces. Therefore, phlegmon in comparison with an abscess is more severe, with pronounced intoxication.



Phlegmon of the foot

Etiopathogenetic factors are similar to abscesses.

Clinical manifestations depend on the location of the purulent process and correspond to the classic signs of inflammation, but in this case the symptoms of intoxication prevail.

Treatment. Treatment of phlegmon is operative and only in the initial stages of the development of the disease is it permissible to use conservative methods of treatment. In the pre-and postoperative periods, antibacterial, anti-inflammatory and detoxification therapy is used.

The operation is performed under general anesthesia. Incisions are made over the site of the greatest fluctuation or infiltration. For the correct choice of surgical access, it is necessary to take into account the anatomical and topographic relations of the fascial beds. In addition, it is necessary to open all the intermuscular septa. Further, the wound is treated as a purulent wound and the use of active or passive aspiration is acceptable.

It is necessary to strictly monitor the condition of the wound in order to avoid the progression of the purulent process and timely conduct additional incisions.

Postinjection phlegmon is an acute purulent inflammationof the cell, which is a complication of injection therapy (violationof aseptics or injection technique). The process is localized in the places where the manipulation is performed: the buttocks, the outer surface of the shoulderand thigh, the anterior abdominal wall, and the elbow folds. It is more often observed after repeated injections, especially after the introduction of hypertensive and long-term absorbable solutions. It is treated according to the general principlesof phlegmon treatment.

Adenophlegmon *is* a purulent inflammation that develops as a result of the spread of the

inflammatory process from the lymph nodes (lymphadenitis) to the surrounding tissue. The most common localization is the neck, especially its medial triangle, where a large number of lymphnodes are concentrated, collecting lymph from the mouth, pharynx, pharynx (the most common entrance gate of infection), from the face and head. Less frequent localizations are inguinal and axillary areas, sub-sectoral zone.

Local clinical signs begin with the appearance of a deep painful dense inflammatory infiltrate without a skin reaction. But as the inflammatory process approaches the surface, skin hyperemia and edema appear, often without fluctuation. Diagnostic puncture in doubtful cases confirms the presence of a purulent process.

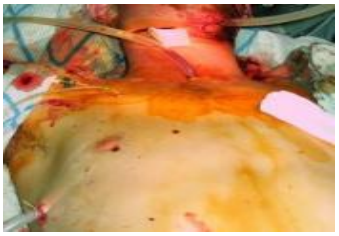
Treatment is carried out according to general principles.

Purulent mediastinitis is apurulent inflammationof the mediastinal fiber. More commonly, purulent mediastinitis is the result of foodwater damage or a complication after mediastinal surgery.

The mediastinum is conventionally divided into upper and lower, each of which includes an anterior and posterior regions.

The clinical picture usually consists of severalsymptoms: possible causes of the disease in the anamnesis; highfever (up to 39-40 S) with severe intoxication and other common signs of purulent infection; local manifestations of mediastinitis. Severe chest pains, respiratory disorders, cyanosis, dysphagia, and acute progressive heart failure are very characteristic. Anterior mediastinitis is characterized by painful beating, increased pain when the head is tilted back, bulging, hyperemia and swelling of the tissues above the jugular notch, sometimes torticollis. Posteriormetastinitis is characterized by radiating pain to the interscapular region,difficulty pressing on the spinous processes of the thoracic vertebrae, and rigidity of the long back muscles. Radiologically, it is possible to detect an expansion of the mediastinal boundaries, in the pleural and pericardial cavities.

Treatment of purulent mediastinitisconsists in active surgicalской tactics. For mediastinotomy, three main approaches are used: upper, lower transdiaphragmatic and extrapleural.



Mandatory in purulent surgery, opening and drainage of the abscess in case of esophageal injuries should be accompanied by the imposition of a gastrostomy forfurther feeding of the patient through a gastric fistula. Complex treatmentof purulent mediastinitis is a difficult and often unsuccessful task.

Paranephritis *is* a purulent-inflammatory process in the parotid fatty tissue. There is a distinction between primary (as a complication of kidney disease) and secondary paranephritis, which occurs in the absence of kidney disease. The source of infection can be inflammatory foci in neighboring organs (most often in the colon), or its spreadby hematogenic and lymphogenic routes from distant foci.

Depending on the location of the purulent-inflammatoryfocus, there are anterior, posterior (most common), upper, lower and total paranephritis.

Clinical manifestations characteristic of any purulent processhave some features in paranephritis: pronouncedhyperthermia (upto 39-40 degrees C), pain, pain in the lumbar region, protective contraction of the lumbar muscles on the side of the disease, forced position of the patient with the hip raised to the stomach andits painful extension. Radiologically, you can detect a limited excursion of the lungs, scoliosis of the spine and the absence of a contour of the lumbar muscle.

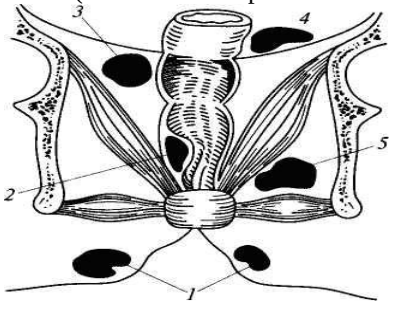
The treatment of purulent paranephritis is based on surgical intervention. It consists of lumbotomy (incision in the lumbar region), opening of the paranephralabscess, necrectomy and drainage of the focus.

Paraproctitis *is* an inflammation of the parotid intestinal tissue. There are acute purulent paraproctitis and, as its most frequent outcome, chronic paraproctitis (formation of a pararectal fistula).

НаибоThe most common clinical and anatomical classification of acute paraproctitis is based on the possible localization of the purulent focus:subcutaneous and submucosal (1), sciatico-rectal - (5)(ischiorectal),

pelvic-rectal –(3) (pelviorectal) and

post-rectal - (2) (retrorectal).

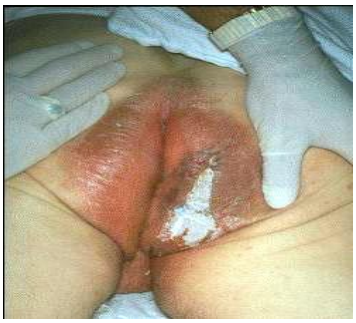


The etiopathogenesis of paraproctitis is based on theintroduction of infection from the lumen of the

rectum to the pararektal tissue through the ducts of the anal glands or mucosal defects caused bypain and damage to the intestine.

Clinic. Intoxication-related general clinical manifestations of purulent inflammation are accompanied by the following most persistentlocal symptoms: pain in the rectum, perineum or pelvis, stool retention, dysuria, and the presence of painful infiltration during palpationor digital rectal examination.

The most frequent(up to 50% of cases) subcutaneous paraproctitis is characterized by hyperemia of the skin, visually and palpation easily detectedstroke. The mildest form of submucosal paraproctitis (up to 6% of cases) is characterized by the bulging of the abscess into the intestinal lumen during its palsyexamination, flattening and infiltration of one of the intestinal walls



Diagnosis of ischiorectal paraproctitis (up to 40% of cases) is often delayed due to the long-term absence of external signs of the disease, similar to the manifestations of subcutaneous and submucosal foci.- A distinctive feature is the infiltration of the lower ampullary rectum and the anal canal above the scallop line. The rarest and most severe form of the disease is pelviorectal paraproctitis, which is manifested by a slow development of intoxication symptoms, severity of the condition, and indeterminate pain in the pelvis and lower abdomen. By the nature of the clinical course in the late stages ofpelviorectal paraproctitis is similar to ischiorectal. The retrorectallocalization of the abscess is characterized by severe pain in the rectum and sacrum from the very beginning of the disease, which increases during defecation and in a sitting position.

Treatment of acute paraproctitis should be surgical only. The operation is performed for emergency indications under general anesthesia. The main principle of surgical intervention is the opening and drainage of the purulent focus. Communication of the abscess cavity with the rectal lumen leads to the formation of pararectal fistulas and recurrent purulent paraproctitis.

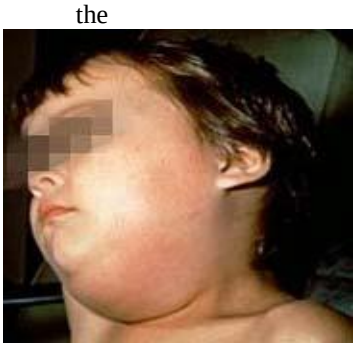
Purulent mumps.

Purulent mumps is a purulent inflammation of the parotidsalivary gland. There are serous, abscessed, phlegmonousand gangrenousforms of acute mumps. Purulent mumps include abscessed, phlegmonous and gangrenous forms.

The main causative agents of the disease are Staphylococcus and Streptococcus. The infection enters the parotid salivary gland more often from the oral cavity through its duct. A favorable factor for this is the reduction or cessation of salivation in weakened anddehydrated patients with infectious diseases or after extensive surgery. The disease is not contagious.

There are some peculiarities in the clinical manifestation of various forms of acute mumps, butecthere are also many similarities. In the area of the parotid salivary gland, pain appears, which increases when chewing and openingвании ртthemouth . The body temperature rises to Z9-40°. Locally, there is edema and hyperemia of the skin.

On palpation of the oculargland area, gum infiltration and sharp soreness are noted. Слизистая The oral mucosa is swollen and hyperemic near the opening of the salivary gland canal. With serous and abscessed formsof the disease, an enlarged, dense, painfulsebaceous gland can be palpated. When a **large** cavity is formed in the gland, it is possible to determine the softening site. Fluctuation is determined only when pacnpocextends the purulent process beyond the capsule of the gland.



Treatment. In the serous form of mumps, broad-spectrum antibiotics are prescribed. (cephalosporins, semi-synthetic penicillins, etc.). Also used semi-alcohol compresses, UHF, sollux. With purulent forms of the disease, urgent surgical treatment under general anesthesia is indicated. For an adequate opening of the abscess, 2 incisions are usually performed: a pair-behind and parallel to the vertical branch of the lower jaw, the second-in the transverse direction anteriorly and slightly indenting from the earlobe. Due to the risk of damage to the facial nerve during surgery, incisions are made radially from the earlobe parallel to its main branches. After opening the abscess, non-viable tissues are carefully removed and the wound is drained. In the postoperative period, antibacterial therapy is performed. Local wound treatment is performedin accordance with the phase of the wound process.

Prevention of mumps in operated patients consists in careful oral care, increased salivation (lemon,

chewinggum) and correction of water and electrolyte disorders in patients with severe diseases

Erysipelas.

Erysipelas is an infectious disease caused by B-hemolytic streptococcus and characterized by acute focal lesions of the skin, less often of the mucous membranes.

In recent years, the following classification of erysipelas has been increasingly used in practice:

I. By the nature of washing manifestations: a) erythematous b) erythematous-bullous, c) erythematous-hemorrhagic, d) bullous - hemorrhagic.

II. By the nature of the course: a) primary, b) repeated (occurs after 2 years, or a different localization), c) recurrent.

III. According to the prevalence of local manifestations: a) localized, b) widespread (migrating), c) metastatic (the appearance of distant foci of inflammation).

IV. According to the degree of intoxication (severity of the course): I easy, IImoderate severity, III heavy.

The pathogen enters the tissues more often by contact through smallskin lesions. In tissues, streptococcus spreadsmainly through the lymphatic vessels, which disrupts the lymph flow and causes edema. In the development of the disease, the airborne mechanism of streptococcus transmission withprimary nasopharyngeal lesion and subsequent spread of infection by lymphagenic or hematogenic pathways is also important. Most often, the inflammatoryprocess is localized on the lower extremities, less often on the face and upper extremities. It is noted that erysipelas most often occur in the summer-autumn period.

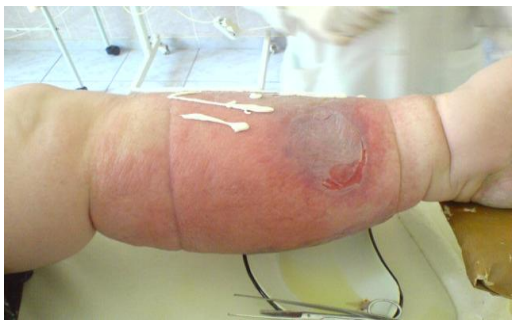
Erysipelas tend to be chronically recurrent in 25-35% of cases. After primary erysipelas, streptococcus may remain latent, forming chronic foci of endogenous infection in the skin, lymph vessels, and nodes. Under-certain conditions (hypothermia, stress, etc.), streptococcus goes from a latent state to an active one and another relapse of the disease occurs.

Clinical picture. The incubation period of erysipelas lasts fromseveral hours to 3-5 days. In the initial period of the disease, symptoms of intoxication develop rapidly, which in more than half of patients outstrip local manifestations of erysipelas from several hours to 2 knots. There is weakness, headache, chills, muscle aches, and a sharpincrease in body temperature. In the area of future local manifestationsof erysipelas, a burning or itching sensation occurs.

During the height of the disease, local symptoms of erysipelas appear.

Erythematous erysipelas are characterized by the presence of bright hyperemia of the skin with clear and uneven borders in the form of "flames", "contoursof a geographical map". The skin in the area of erythema is swollen, tense, painful on palpation (more along the periphery of hyperemia), more than 70% of patients develop regional lymphangitis.

With **bullous erysipelas**, after 2-5 days from the onset of the disease, against the background of skin hyperemia, a detachment of the epidermis appears, which is associated with increased exudation in the focus of inflammation. Puffins (bullae) are formed, containingeposyphilous exudate and a large numberof streptococci. Erosion occurs at the site of damaged bubbles. In place of the undamaged exfoliated epidermis, brownbones are formed.



Erythematous-hemorrhagic erysipelasdevelop up to 3 days after the onset of the disease. Against the background of hyperemia of the skin, hemorrhages of various sizes appear: from small petechiae to extensive draining-hemorrhages.

Bullous-hemorrhagicerysipelasoccur as a result of deepdamage to the capillaries and blood vessels of the dermis, and are most severe compared to other forms. Hemorrhagicexudate accumulates under the exfoliated epidermis, often with an admixture of fibrin.
With significant hemorrhages in the thickness of the skin, necrosis develops, ulcers will rejoice. With the addition of a secondaryinfection, the inflammatory process spreads to the subcutaneous fat, i.e. phlegmon develops.

For small-area lesions and a mild course of the disease, oral anti-bacterial therapy is prescribed (erythromycin, doxycycline. streptocide, rifampicin, ciprofloxacin)

In more severe cases of erysipelas, penicillin-type preparations are used in a daily dose of 6-12 million units for 7-10 days, and with the development of complications (necrosis, phlegmon,-abscess, etc.) - in combination with gentamicin (240 mg 1 time a day) or cephalosporinsare prescribed. For recurrent erysipelas, cephalosporin antibiotics of the III or IV generation are used. It is advisable to combine antibiotics with sulfonamide preparations (biseptol 960 mg 2 times a day.) Also detoxification and desensitizing (tavegil, suprastin) therapy, prescribe ascorbic acid and vitamins of group B. With severe skin infiltration in the focus of inflammation, non-steroidal anti-inflammatory drugs (butadion) are effective, in severe cases they resort to the use of corticosteroids (prednisone).

With bullous erysipelas, the blisters are opened, after which they are applied inmating with antiseptic solutions. With erythematous-hemorrhagic eruptionженаочаг, bandages are applied to the focus of inflammation with 5-10% liniment dibunola2 times a day for 5-7 days. UVfoci of inflammation and areas of regional lymph nodes, lasertherapy are effective, especially in hemorrhagic forms of erysipelas.

With the development of phlegmon and skin necrosis, incisions are made, non-viable tissues are excised, wounds are drained and bandages are applied with ointments on a water-soluble basis (levosin dioxicol, etc.). Inthe future, wounds are conducted in accordance with the basic principles of wound treatment.

Complications of erysipelas: skin necrosis, phlegmon, abscess, lymphangitis, thrombophlebitis, sepsis, toxicoanaphylacticshock.

Erysipeloid.

Erysipeloid is an infectious skin disease caused by swine erysipelas bacillus. Workers in the meat and fish industries are more likely to get erysipeloid. The infection penetrates through abrasions and cracks in the skin.

Clinic. At the site of infection, a red painful swelling with itching appears, which soon turns into a purple spot with clear contours - the "contourof a geographical map". The duration of the disease is 10-12 days, but the red spot remains for a long period of life.

Treatment. The use of antibiotics, sulfonamides, and physiotherapy.

Acute purulent mastitis.

Acute purulent mastitis is a purulent inflammation of the interstitium and parenchyma of the breast. There are lactation mastitis (80-95%) and non - lactation mastitis (10-15%). According to the nature of the inflammatory process, there are: non-purulent mastitis-serous and infiltrative; purulent mastitis – abscessing; phlegmonous and gangrenous. According to the localization of the abscess-subariolar, subcutaneous, intramammary, retromammary.

Predisposing factors are reduced immunological reactivity, complicated labor, pregnancy pathology, cracked nipples, and lactostasis. Extremely rare cases of mastitis were also observed in men, the development of the disease is

associated with a breast injury or a purulent complication of gynecomastia. In 80% of cases, mastitis develops in women after the first birth. Microorganisms, penetrating in various ways into the tissues of the mammary glands, cause milk clotting due to lactic acid fermentation and obturation of the excretory ducts. The number of microorganisms reaches a critical level and inflammation of the gland lobule occurs. With improper care and treatment, lactostasis turns into an infiltrative form of mastitis and spreads to the surrounding lobules.

The disease begins acutely: pains in the gland are disturbed, the gland increases in size, hyperemia appears, the amount of milk decreases, pumping and palpation of the gland become painful. This is how the serous form of lactation mastitis develops.

After a few days, the process turns into an infiltrative form, the temperature rises to 39-40 degrees. Symptoms of intoxication begin to appear. In the absence of treatment, after 2 to 3 days, the non-purulent form of mastitis turns into purulent – abscessing. General and local manifestations increase, and the symptom of fluctuation is clearly defined.

In the phlegmonous form of mastitis, the process spreads beyond the breast, developing lymphangitisand axillary lymphadenitis.

The most severe form of gangrenous mastitis occurs. As a result of late medical treatment due to the occurrence of thrombosis, compression of blood vessels, stagnation of blood in the vessels, gangrene of the breast develops. The condition of women is usually severe or extremely severe. Symptoms of intoxication are expressed, tachycardia up to 120-140 beats per minute, a decrease in blood pressure, shortness of breath, the temperature rises to 41 degrees. Locally, the skin is purplish-cyanotic in color, sometimes with areas of necrosis.

МаститNeonatal mastitisoccurs - inthe first 2-3 weeksжизниofthe child н's lifeagainst the backgroundof physiologicalcoarseningof the mammaryglands; it occursin bothgirls, так andyboys. The infectionpenetratesthетканьgland tissuethroughskin damageorhematogenicpathways. The diseaseis characterized by a veryrapiddevelopmentof purulentinflammation: bythe endof the firstto the beginning of the second dayfromthe momentof appearanceсимптомовof mastitis symptoms. Inмолочнойthe milkjelly, oftenclosertothe areola, формируетсяan abscess forms. Inthe future, the disease maybe complicatedby phlegmonпереднейof the anteriorchest.

Diagnosis of purulent mastitis usually does not cause difficulties, but in some cases, to clarify the stage of the disease, the depth of the purulent focus and the nature of exudate, it is possible to use ultrasound of the breast and puncture of the inflammatory focus.

Treatment. Treatment of lactostasis and initial forms of mastitis involves the use of conservative methods of treatment: elevated position of the gland, careful pumping of milk from both mammary glands, the use of antispasmodics (no-shpa, papaverine for 15-20 minutes). before pumping), antibacterial drugs, physical therapy (ultrasound, UHF therapy, UFR of the breast).

Treatment of purulent mastitis is only surgical. The autopsy is performed under general anesthesia.

When choosing access, the location and prevalence of the purulent process are taken into account. In subariolar mastitis, the incision is made in an arcuate manner, under the areola, retreating by 2-3 mm, but not more than ½ of its circumference. With subcutaneous or intramammary location of the abscess, radial incisions are used, taking into account the sectoral structure of the breast. With retromammary mastitis, the Bardenheyer approach is used – an arc-shaped incision along the transitional fold under the mammary gland.

Further treatment is carried out according to the generally accepted rules for the treatment of purulent wounds.

II. Purulent diseases of glandular organs, blood and lymphatic vessels.

Thrombophlebitis.

One of the most common diseases of the venous system is thrombophlebitis, which is observed in 20-40% of the population. Thrombophlebitis refers to inflammatory changes in the venous wall followed by the formation of a blood clot in the lumen of the vein, phlebothrombosis refers to the formation of a blood clot in the lumen of the vein with the addition of inflammatory changes in the venous wall.

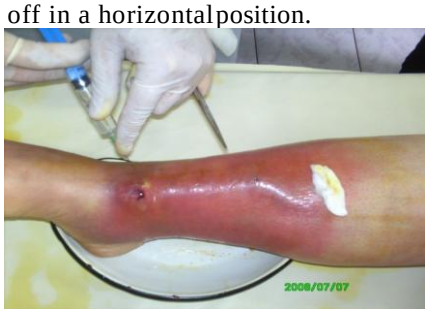
From the point of view of modern phlebology, there are no fundamental differences in the pathogenesis of thrombophlebitis and phlebothrombosis. With venous thrombosis , there are always inflammatory changes in the venous wall, and inflammation of the vein wall (phlebitis) is accompanied by its thrombosis. However, phlebologists, understanding the conventionality of such a division, use the term thrombophlebitis for subcutaneous lesions, and phlebothrombosis for deep veins; the only exceptions are extremely rare cases of purulent melting of thrombotic masses, which can occur in both the superficial and deep venous systems. In general, phlebitis is usually aseptic in nature, and the role of infection is probably limited to secondary changes in hemocoagulation.

Depending on the location of the thrombotic process, thrombophlebitis of the superficial and deep veins is distinguished. The term varicothrombophlebitis refers to the most common form of thrombophlebitis, in which the pathological process affects variously dilated superficial (subcutaneous) veins of the lower-extremities. In the vast majority of cases, it is a complication of varicose veins, less often occurs in post-thrombotic disease. Varicose veins are a "fertile ground" for the development of thrombosis, because changes in the vascular wall and slowing blood flow are important causes of thrombosis. With corresponding changes in the adhesive-aggregation properties of the formed blood elements and the plasma hemostasis link (which is facilitated by venous stagnation and turbulent blood flow), blood clots occur in them.

A number of factors play a role in the occurrence of venous thrombosis (Virchow's triad): venous endothelial damage (e.g., catheter insertion, obliterating thrombangiitis, septic phlebitis), hypercoagulation (e.g., blood diseases, oncological diseases), slowing of blood flow (postoperative period, varicose veins, etc.).

The clinical picture of acute varicothrombophlebitis and subcutaneous vein thrombophlebitis in most cases does not cause significant difficulties. Patients are concerned about pain of varying intensity in the-affected limb. On examination, there is a band of hyperemia in the projection of the affected, often varicose, vein. Palpation reveals a painful weight in this area. The contours and dimensions of the palpable infiltrate do not change when the patient is transferred from a vertical position to a horizontal one, while non-thrombosed venous nodes have a soft consistency and fall off in a horizontal position.

The progression of the inflammatory process and the addition of purulent microflora lead to the development of purulent thrombophlebitis, characterized by hyperemia of the skin along the course of thrombosed veins and often the appearance of fluctuations over the inflammatory infiltrate.



It is important to assess the presence and nature of edema of the affected limb. With thrombophlebitis of the subcutaneous veins, it usually has a local (not inflammatory) character. Pronounced widespread edema and cyanosis of the distal extremities indicate thrombotic damage to the deep veins. The most dangerous ascending forms of varicothrombophlebitis are when, as the disease develops, the process spreads from the distal parts of the limb in the proximal direction. At the same time, there is a real threat of a blood clot spreading through the sapheno-femoral fistula into the femoral vein, with its separation and the development of pulmonary embolism. It should be noted that in 30% of patients, the true border of the thrombus is located 15-20 cm proximal to the clinically determined signs of thrombophlebitis. The exact level of thrombosis spread can only be determined by phlebography or ultrasound duplex angioscanning (due to direct visualization of blood vessels).

Deep vein thrombosis, especially in the iliofemoral segment, is accompanied by a more pronounced pain syndrome, significant (often tense) edema of the affected lower limb, and the appearance of a compensatory network of subcutaneous venous collaterals.

Palpation of the calf muscles (Meyer's symptom) and dorsal flexion of the foot, which leads to tension

of the calf muscles (Pomais ' symptom), are sharply painful.

Treatment measures for acute thrombophlebitis shouldprevent the spread of thrombosis to the deep veins, prevent pulmonaryembolism, quickly stop inflammatory phenomena in the walls of the vein and surrounding tissues, and exclude the recurrence of thrombosisof varicose veins. Limited saphenousvein thrombophlebitis can be treated on an outpatient basis. Deep vein thrombophlebitis should only be treated in a hospital setting.

The need for emergency or urgent surgical treatment occurs with ascending thrombophlebitis of the large subcutaneous foam. In this case, Troyanov-Trendelenburg surgery is performed (ligation of the sapheno-femoral fistula), which prevents the spread of thrombosis to the deep veins. Patients withvaricotrombophlebitis who are not physically burdened can alsobe operated on in the first 2 weeks of the disease in order to reduce the duration of treatment. In this case, radical excision of subcutaneous, including thrombosedveins, is performed. The development of purulent thrombophlebitis of the subcutaneous veins requires urgent surgical treatment, including excision of the affected veins and drainage of purulent foci according to the general principles of purulent surgery. In-most cases, conservative treatment is performed. Prescribe an activeregimen to prevent deep vein thrombosis, mandatory elastic compression of the extremities with elastic bandages. In deep vein thrombophlebitis, bed rest with an elevatedposition of the limb is indicated.

Drug therapy should include the following classes of pharmaceutical drugs::

*non-steroidal anti-inflammatory drugs (diclofenac, ketoprofen, etc.)

- derived routines (troxevasin);
- disaggregants (reopoliglyukin, trental);
- polyenzyme mixtures (wobenzym, phlogenzym);

* phlebotonics of plant origin (diosmin, cyclo-3-fort, detralex);

* anticoagulants (direct-heparin, low-molecular-weight heparin - clexane, fraxiparin, indirect-phenylin).

The use of antibiotics is indicated for the development of purulent thrombophlebitisof the subcutaneous veins and venous gangrene due to deep vein thrombosis.

In the first hours of deep vein thrombosis, thrombolytic therapy in combination with anticoagulants can be effective:fibrinolizin 40-50 thousand units and streptokinase (urokinase, streptodornase) 500 thousand units each. Subsequently, indirect anticoagulants (phenylin) for several months under the control of the prothrombin index

Lymphadenitis,lymphangitis.

Lymphadenitis is an acute inflammation of the lymph nodes. The disease often develops against the background of a purulent process in one or another area of the body, i.e. secondary lymphadenitis. Microorganisms and their toxins with lymph from the primary focus enter the lymph nodes, causing an inflammatory process. The disease is caused by mixed microflora. There are serous, purulent and productive inflammation of the lymph nodes. Lymphadenitis is expressed in edema, swelling and purulent-necrotic melting of the node. Lymph nodes increase in size, often reach the size of a walnut, are sharply painful, have little movement, and are soldered to the skin. Around the hyperemia of the skin and soft tissue edema. When abscessed, a fluctuation is detected, which is an indication for its surgical treatment – opening the abscess. In the initial stages of the disease, antibiotics, sulfonamides, immunomodulators, and physiotherapy are used.

Lymphangitis is an acute inflammation of the lymphatic vessels. Lymphangitis is a secondary disease, a complication of infected skin lesions and local purulent processes. Acute lymphangitis of superficial and deep lymphatic vessels is distinguished.

With superficial lymphangitis, red stripes appear from the primary inflammatory focus, heading to the axillary fossa or inguinal region. Palpation along the course of the strip is determined by a dense, painful nodule. The body temperature rises to 39C and is accompanied by chills.

There are reticular lymphangitis with diffuse redness and stem-in the form of longitudinal stripes along the course of large lymphatic vessels.

Treatment should begin with the elimination of the primary focus (opening of ulcers), then prescribing antibiotics, sulfonamides, creating rest of the limb, applying a semi-alcoholic compress.

IV. Purulent diseases of bones and joints.

Acute purulent arthritis.

Acute purulent arthritis (arthritispurulcnta) is an acute purulent inflammation of the joint. The causative agents of the disease are pyogenicmicroflora, most often Staphylococcus. Purulent arthritis can be primary and secondary. Primary arthritis is more common and develops with opentraumatic injuries of the joints, when infection penetrates the joint through the wound. In secondary arthritis, the joints are infected by

hematogenic or lymphogenic pathways from various foci гнойного воспаof purulent inflammation (boils, sore throats, etc.), as well as when the purulent process spreads from the bones or surrounding soft tissues.

By the nature of the inflammatory process, all arthritis is divided into:serous, serous-fibrinous and purulent. Inflammation **of the knee** joint is called - gonitis**, hip**-coxitis, **shoulder**-plexitis.

The inflammatory process in the joint begins with the lesion of thesynovial membranes (acute synovitis). Exudate accumulates in the joint, first serous, then serous-fibrinous and purulent. With secondary aprub, the serous exudate stage immediately turns into purulent.When purulent inflammation spreads to the joint capsule, purulent arthritis occurs. If the bones that form the joint are involved in the purulent processразвивается , osteoarthritis develops. When the purulentprocess spreads to the tissues surrounding the joint, paraarthritis is formed-periarticular phlegmon, and возthere may also be swelling in far-located tissues.

The clinical picture of the disease is characterized by an acute onset, high body temperature, severe pain in the joint, which increaseswith movement. The contours of the joint are smoothed, the surrounding tissues are swollen, the skin is hyperemic Active and passive movements in the joint are sharply limited, the limb is in a forced semi-bent position. When palpation of the joint is sharp pain, often a symptom of fluctuation is determined. When fluid accumulates in the knee joint, a symptom occurs - balloting of the patella.

An X-ray of the joint reveals the expansion or narrowing of the joint gap, unevenness of the joint surfaces, and in osteoarthritis, foci of bone destruction. When performing a joint puncture for diagnostic purposes, the nature of the inflammatory process is clarified and exudate is examined for microflora and its sensitivity to antibiotics. With purulent arthritis, pus is evacuated from the joint cavity for therapeutic purposes.

Therapeutic tactics. The main therapeutic measures are: evacuation of inflammatory exudate, anti-inflammatory therapy (use of nonsteroidal harmons), general and local antibacterial therapy. In case of serous arthritis, the suture ispunctured, the contents are evacuated, and the cavity is washed with antiseptic solutions. The puncture is completed with the introduction of a broad-spectrum antibiotic solution into the joint cavity. In serous-fibrinous andpurulent forms of arthritis, the joint cavity is drained by a thin tube passed through the lumen of a puncture needle or a cannula of a special trocar. In these cases, the drainage tube is also used for washing thecavity with antiseptic solutions.

1. Osteomyelitis.

Osteomyelitis is a purulent inflammation of all anatomical structures of the bone, i.e. the compact substance (ostitis), the bone marrow (osteomyelitisproper) and the periosteum. Depending on the ways of penetration of microorganisms into the bone, hematogenous and post-traumatic osteomyelitis are distinguished. The latter, in turn, is divided into gunshotand postoperative. It is difficult to find a disease equal to osteomyelitis in terms of the duration of the clinical course. It can last for decades. In peacetime, hematogenous osteomyelitis is most common, during which acuteand chronicperiods are distinguished.

Acute hematogenous osteomyelitis in the vast majority of cases develops in childhood. The causative-agent of hematogenic osteomyelitis in 90-93% of cases is pathogenic staphylococcus, less often gram-negative flora and associations of gram (+), gram (-) and non-clostridial anaerobes. Until now, the question remains unclear-what is the basis of circulatory disorders of the bone with subsequent necrosis? Many theories of the pathogenesis of acute hematogenicosteomyelitis have been proposed. To date, the theory of M. V. Grinev (1977) - extravasal inflammatory edema with subsequent compression of bone vessels-has received the greatest support. It was found thatthe arteria of the spongy substance of the bone branch into capillaries, which passinto the wide venous capillaries (the so-called "venous sinuses") in the medullary canal. Blood flow in them is slower compared to capillaries of other tissues. This contributes to the sedimentation of microbes that haveentered the bloodstream from any primary purulent focus (angina, carbuncle, etc.), to the intima of the venous sinus. In the future, microbes enter the paravasal space, where theinflammatory process develops. Progressive edema compressesthe blood vessels of the bonemarrow and there is a shutdown of blood flow in the lesion, including collateral. No other theory explains the reasonfor switching off the collateral intraosseous blood flow and the therapeutic effect of bone trepanation.

Pathoanatomical picture of acute hematogenous osteomyelitis.The focus is initially edematous-hemorrhagic myelitis, and then purulent-necrotic destruction

bone marrow is accompanied by an increase in intraosseous pressure by tens of times (normally, intraosseous pressure in long tubular bones is 20-40 mm of water column), as a result of which the purulent process quickly spreads along the length of the medullary canal (bone marrow phlegmon) and through the channels of the compact substance of the bone subcostally (subperiosteal phlegmon). The inflammatory-altered periosteum peels off from the compact substance of the bone andbreaks, paraosal, and then intermuscular and subcutaneous phlegmons develop. In the future, when the thinned skin breaks through, a fistula is formed.

Clinic and diagnosis of acute hematogenous osteomyelitis. The importance of early diagnosis and, consequently, early treatment of acute hematogenicosteomyelitis, calculated not even days, but hours from the onset of the disease, is dictated by the features of inflammation in the bonemarrow canal, the rate and dynamics of

bone changes. According to statistics, if treatment is started before three days from the onset of the disease, then the transition of the disease to the chronic stage occurs in 8% of cases,and if from three to seven days, then in 25% of cases.

The most important common symptoms of acute hematogenous osteomyelitis include::

1. An increase in body temperature, especially in the septicform of the course of the disease, is the rule. It immediately reaches 39-40 degrees and lasts for a long time.

2. The general condition worsens-chills, weakness, lethargy, headache. Young children may have confusion, seizures. Symptoms of cardiovascular and respiratory failure oftenoccur. The liver and spleen increase, and symptoms of hepatic-renal failure appear.

The most important local signs are:

1. Pain in the affected limb, initially aching (for 1-2 hours), then quickly increases, especially withthe slightest movement, becomes bursting, drilling. The patient is restless, feelsthe affected limb. Pain increases even with careful palpation and percussion in the affected area. Pain is the earliest and most important symptom of acute hematogenous osteomyelitis.

2. KLocal early signs include the appearance of moderate edema in the affected area (edema can only be determined by comparativemeasurement of the circumference of the patient and a healthy limb).

3. Narrowing of theskin pattern of dilated cutaneous and later subcutaneousveins.

4. In later periods, with the development of intermuscular and subcutaneousphlegmon, local temperature increases, skin hyperemia andfluctuations appear, and regional lymph nodes increase.

Laboratory tests show leukocytosis, a shift ofthe blood leukophore to the left, an increase in ESR, hypochromic anemia, and bacteremia.

Additional diagnostic methods: computedtomography of the bone, measurement of intraosseous pressure, and thermography ofthe knee joint are used. Radiological signs of it: pronouncedcompactionof soft tissues in the immediate vicinity of the bone, rarefaction of the bone structure (osteoporosis), detachment of the periosteum and bone destruction with the formation of cavities (in the third or fourth week of the disease).

Treatment of acute hematogenous osteomyelitis.

Massive therapy with broad-spectrum antibiotics can interrupt inflammation in children and adults only at the stage of edematous-hemorrhagic myelitis, so treatment should certainly be continuous.

Hematogenous myelitis and purulent-necrotic myelitis do notrespond to general (including intravenous) drug therapy, and only direct exposure to the pathogen (administration of antibiotics through the osteoperforation opening into the medullary canal) is effective. After osteoperforation, the closed lumen of the medullary canal is drained, intraosseous pressure is reduced, andpain is eliminated. Through drainages, conditions are created for the outflow of pus, seeding for microflora and sanitizing the focus of inflammation with targeted antibiotics. The limb is immobilized for 4 to 5 days. In addition, the treatment package includes passive immunization of the patient (administration of antistaphylococcal gamma globulin I-3 ml ivfor 8-10 days, transfusion of antistaphylococcal plasma, detoxification, vitamin therapy, and physiotherapyprocedures).

Chronic hematogenous osteomyelitis, with the exception of primarychronic forms (Garre's sclerosing osteomyelitis, Ollier's albuminous osteomyelitis, and Brody's abscess) is the outcome of an acute process that is not timelydiagnosed and adequately treatedoc. The difference between chronic hematogenous osteomyelitis and other forms of chronic osteomyelitis is the large extent of the destructive process in the bone with the presence of bone sequesters, the multiplicity of purulent-necrotic foci, the long course of the disease with frequent exacerbations andthe formation of an external purulent fistula.

Modern principles of treatment of patients with chronic andposttraumatic osteomyelitis can be summarized in the following provisions:

a) preoperative examination of the patient should includeX-ray of the bone, fluorography, bacteriological examination ofthe fistula (wound) removed from the fistula (wound).);

b) it is necessary to prepare the skin of the limb, sanitize fistulas, washing them with antiseptic solutions;

c) general long-term preoperative antibiotic therapyis inappropriate (on the day of surgery, simultaneously with anesthesia, a single dose of a broad-spectrum antibiotic is administered intramuscularly or intravenously to the patient);

d) introduction of a dye (methylene blueor diamond green) into the fistula passage for intraoperative-diagnosis of the prevalence of purulent congestion in soft tissues and bones;

e) sufficient access to visually assess the required volume of necrectomy;

c) thorough necrosequestrectomy within the vitalbone tissue, which in chronic hematogenic osteo-myelitis is achieved after bone trepanation, and in post-traumatic osteomyelitis, resection of the affected area can be resorted to;

g) additional intraoperative treatment of bone tissue (vacuuming, low-frequency ultrasound, etc.). In addition, inpost-traumatic and postoperative osteomyelitis, it is necessary to remove all previously installed bone retainers, if theydo not ensure the stability of fragments.

After necrosequestrectomy or resection of the affected bone inpost-traumatic osteomyelitis, stable fixation ofbone fragments is carried out mainly with external fixation devices (rod and spoke). Inthe-postoperative period, targeted antibacterial therapy is performed. According to indications -

immunocorrection and administration of drugs that stimulate osteogenesis.

2. Acute purulent arthritis.

Acute purulentarthritisarthritisisan acute purulent inflammation of the joint. Acute purulent arthritis is a disease characterized by purulent-inflammatory and purulent-necrotic processes in the joint and surrounding tissues. Large joints are more often affected: hip, shoulder, knee. It is caused by staphylococci, streptococci, pneumococci or in association with other pyogenic bacteria. The causative agents of arthritis can also be tuberculosis, gonococcus, and typhoid fever. There are primary or exogenous arthritis associated directly with damage to the joint (injured, or polupansiona postoperative complications) and secondary or endogenous, in which the joints become infected by hematogenous or lymphogenous way from different foci of purulent Vespation (boil, sore throat and so on), as well as for distribution of a purulent process with bones or surrounding bustle" of the soft tissues or by contact.

By the nature of the inflammatory process, all arthritis is divided into:serous, serous-fibrinous and purulent. Inflammation **of the knee** joint is called - gonitis**, hip**-coxitis, **shoulder**-plexitis.

With purulent arthritis, inflammatory infiltration of the entire thickness of the articular sac is noted, and the process can pass to the ligamentous apparatus. In the initial stages of the disease, inflammation invades the synovial bags, acute synovitis develops, serous effusion appears in the joint cavity, and if left untreated, the effusion becomes serous-fibrinous, and then purulent. With secondary aprub, the serous exudate stage immediately turns into purulent.When purulent inflammation spreads to the joint capsule, purulent arthritis occurs. If the bones that form the joint are involved in the purulent processразвивается , osteoarthritis develops. When the purulentprocess spreads to the tissues surrounding the joint, paraarthritis is formed-periarticular phlegmon, and возthere may also be swelling in far-located tissues.

The clinical picture of the disease is characterized by an acute onset, high body temperature, severe pain in the joint, which increaseswith movement. The disease is usually severe. In the area of the affected joint, intense pain, often bursting in nature, joint swelling, tension, infiltration and bright hyperemia, smoothness of the joint contours are noted. The degree of smoothness of the joint contours and the increase in its volume depends on the amount of effusion in the joint cavity.

Active movements are completely absent, passive movements cause sharp pain. The limb takes on a forced position.

When palpation of the joint is sharp pain, often a symptom of fluctuation is determined. When fluid accumulates in the knee joint, a symptom occurs - balloting of the patella (with light pressure from the fingers, a floating patella is determined).

Radiologically, there are no pathological symptoms in the early stages. On

On days 7-10 of the disease, expansion of the joint gap, epiphysis osteoporosis, unevenness of the articular surfaces, and in osteoarthritis, foci of bone destruction are recorded.

If it is difficult to make a diagnosis, it is possible to perform a diagnostic puncture of the joint. The diagnosis is made based on the examination of the evacuated fluid. The resulting discharge is sent for bacteriological examination and determination of sensitivity to antibiotics.

Therapeutic tactics. Treatment should be general and local The main therapeutic measures are: evacuation of inflammatory exudate, anti-inflammatory therapy (use of nonsteroidal harmons), general and local antibacterial therapy.

General treatment includes antibacterial, anti-inflammatory and detoxification therapy (intravenous infusions, in severe cases, the use of extracorporeal detoxification methods).

In the treatment of purulent arthritis, complete immobilization of the limb with a plaster splint is necessary.

In the initial stages, multiple daily punctures of the joint are used to evacuate pathological exudate, and the introduction of antibacterial drugs into the joint cavity. Punctures are performed until the complete cessation of exudate intake.

If the moloinvasive method is ineffective or in advanced cases, arthrotomy is used with the adjustment of the flow-flushing system.

With the development of epiphyseal osteomyelitis, arthrotomy is performed, resection of damaged parts of the epiphysis is performed, and then conditions are created (by prolonged immobilization of the limb) for the development of ankylosis. In the future, you can resort to endoprosthetics (hip or knee) of the joint.

V. Surgical sepsis

Surgical sepsis is a severe general disease that usually occurs against the background of an existing local focus of infection, requiring surgical rehabilitation, as well as a decrease in the body's protective reactions and manifesting itself as dystrophic and degenerative changesin internal organs and tissues.

Modern research has significantly changed the understandingof the pathogenesis of sepsis. It turned out that systemic manifestations in sepsis are caused by inflammatory mediators that are released by macrophages and circulatory monocytes in THE BLOOD for the presence of a focus of infection. Macrophages and monocytes are activated by microbial toxins. Cytokine- induced damage to the vascular endothelium of various oporgans and tissues eventually leads to the development of multiple organ failure. Thus, it is not

the microbes themselves, but the substances activated by them that have a damaging effect on **organs** and tissues, and are responsible for the development of a "septic cascade" of pathophysiological phenomena.

Excessive microbial load (gram. positive, gram-negative flora, anaerobes, etc.) In the primary focus, the numberof microorganisms in 1 gram of tissues significantly exceeds the critical concentration (more than 105).

Interaction of bacteria and / or their fragmentswith macrophages and neutrophils

Blood supply of inflammatory mediators(prostaglandins, thromboxanes, leukotrienes, tumor necrosis factor, interleukins-1, -6, -2).

Systemic oresponse to inflammation (tissue inflammation, edema, bloodhypercoagulability, allergy, sensitization, vasodilation, hypovolemia,tissue ischemia, fever, leukocyte shift to the left, anemia)

Multiple organ failure (hypoperfusion of organs, multiple organ failure, fatal outcome)

The international terminology of sepsis was adopted at the conciliatory conference of the American College of Thoracic Surgeons and the Societyof Intensive Care Specialists (USA, 1991).) and it is increasingly used in domestic medical practice.

Sepsis is a non-specific infectious disease characterized by a pathological systemic reaction of the body to infection, which is of the same type with a wide variety of pathogens, and with progression leads to disruption of the functions of vital organs and systems.

The need to clarifythe terminology and classification of sepsis is primarily due to the need for its early diagnosis, which ensures timely initiation of treatment, таксince if the diagnosis of sepsis is made at the stage **of multipleorgan failure,** the mortality rate is extremely high, regardless of the therapy being undertaken. In addition, modern requirements forconducting scientific research imply obtaining comparabledata (effectiveness of new drugs, lethality, etc.) and require a unified classification and assessment of the severity of patients ' condition. In accordance with international terminology, the following concepts are distinguished::

Bacteremia (septicopyemia) - the presence of live bacteria and blood. Bacteremia is an optional sign of sepsis.

Septicemia – the presence of toxins in the bloodstream that cause intoxication.

Systemic inflammatory response syndrome (SSBBP) a term proposed for the clinical definition of common manifestationsof infection. The syndrome involves the presence of at least two of the following symptoms::

- температураThe body temperature ypa is Z8 ° - 40 ° C.
- heart rate 90 beats per 1 minute
- respiratory rate - 20 in 1 minute
- white blood cells – more than 12×10^{12} or the number of immature forms of neutrophils exceeds 10.

Sepsis systemic response to infectious inflammation. Identification of two or more of the above symptoms in the presence of a focus of infection (but not necessarily bacteremia) allowsus to formulate a clinical diagnosis of "sepsis".

Severe sepsis – sepsis with organ dysfunction, hypoperfusion, manifested by acidosis, oliguria, acute mental status disorder, and other signs of organ damage.

Septic shock (infectious and toxic shock) is the most severe form of sepsis, which is based on persistent arterial hypotension, which persists, despite adequate infusion therapy, systolic blood pressure in septic shock is below 90 mm Hg or decreases by more than 40 mm Hg from the initial one. With medical support, blood pressure may remain at normal levels, but there is always a violation of perfusion. The main cause of shock is considered to be refractive vasoplegia under the influence of nitric oxide. Disorders of microcirculation in it are caused not only by a violation of vascular tone, but also by a violation of the rheological properties of blood and the development of DIC-syndrome.

Multiple organ failure syndrome is a disorder of organ function that requires external intervention to maintain homeostasis.

Clinical criteria for the initial stage of sepsis: the presence of a focus of infection (not always), hyperthermia, tachycardia, shortness of breathand inadequate organ function.

Sepsis as a result of gangrene.

Manifestations of sepsis: disorders of mental status(psyche or consciousness), hypoxemia, metabolic

acidosis, oliguria.

Manifestations on the part of organs and systems:

Respiratory system - respiratory alkalosis, hyperventilation, adult respiratory distress syndrome (shock lung), diffuse infiltrates in the lungs, pulmonary edema.

Kidneys - hypoperfusion, damage to the renal tubules, azotemia and oliguria. Cardiovascular system - first, an increase in cardiacoutput, a decrease in peripheral resistance, vasodilation, endothelial damage, a drop in vascular tone and pressure, myocardial depression, a decrease in cardiac output, vasoconstriction, organ hypoperfusion, and refractory hypotension.

MeMental status - disorientation, drowsiness, confusion. agitation or lethargy, sopor, coma.

Liver - moderate dysfunction (an early symptom of sepsis), increased levels of bilirubin and transaminases-a poor prognostic sign.

Hematological parameters: neutrophilic leukocytosis, left shift (first, but not always), vacuolization and toxic granularity of neutrophils (always), thrombocytopenia, development of DIC syndrome, decrease in serum iron-the phenomenon of redistribution and binding to proteins (always).

Clinical picture of sepsis.

According to the clinical course, there are lightning-fast, acute, subacute and chronic sepsis.

With the lightning-fast form of the disease, the clinic proceeds extremely rapidly, and in the first or second day septic shock already develops, the disease usually lasts 5-7 days, the prognosis is usually unfavorable. The main pathogens are Pseudomonas aeruginosa, Staphylococcus, Streptococcus, anaerobes and gram-negative flora.

In acute sepsis, symptoms develop within a few days, the duration of the disease is 2-4 weeks. Acute sepsis is observed in 40% of patients and is accompanied by a high mortality rate.

In subacute sepsis, the duration of the disease is 6-12 weeks, and the outcome is usually favorable. Chronic sepsis sometimes lasts for years with exacerbations and remissions.

Gram-positive sepsis. Sepsis caused by gram-positive flora usually begins slowly, is accompanied by the development of a pronounced inflammatory component, and is prone to abscesses and prolonged course.

Gram-negative sepsis. Currently, the most common pathogens are Pseudomonas aeruginosa, proteus, klebsiella. Gram-negative sepsis is more often caused by an in-hospital (hospital) infection. The course of the disease is more aggressive and quickly leads to the development of multiple organ failure.

A characteristic feature of recent decades has been the growth of anaerobic and fungal sepsis and sepsis caused by mixed flora. Anaerobic sepsis is characterized by a violent, extremely severe course with a high probability of death.

Causative agents of sepsis are mainly opportunistic microorganisms. The flora is more often highly resistant to therapy, and the sensitivity to antibiotics in the sown strains is not more than 70%, even to the newest drugs.

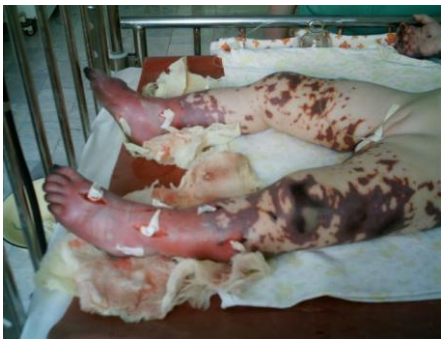
Sepsis has no pathognomonic symptoms. Clinical manifestations are diverse and depend on the phase of the disease, the pathogen that caused it, the form of the pathological process, the degree of compensation of organs and systems, the state of the immune system, the location of the primary focus, and a number of other factors.

In typical cases of acute sepsis, the clinic presents with symptoms of SIRS against the background of the presence of a primary infectious focus.

Complaints associated with the presence of a local focus of inflammation and local complications are characteristic. There is fever, general malaise, pallor of the skin, weakness, headache and muscle pain, loss of appetite, nausea, vomiting, decreased diuresis, intestinal disorders. On examination, dry skin and mucous membranes, tachypnea above 20 per minute, tachycardia above 100 per minute, blood pressure may decrease or remain at normal levels, confusion, enlarged liver and spleen. It is very important that in sepsis, these symptoms may persist even after opening and adequate drainage of the primary purulent focus.

In severe sepsis, symptoms of organ disorders come to the fore: tachycardia over 120 per minute, blood pressure decreases by more than 40 mmHg, tachypnea up to 26-30 per minute. It is possible to develop an abscess or gangrene of the lung. There is a clinic of septic endomyocarditis, toxic hepatitis, oliguria develops, intestinal dysfunction in the form of flatulence or dynamic obstruction is noted.

In more severe cases, anuria develops with edema on the extremities and in the serous cavities, symptoms of edema of the brain and lungs are added, and psychosis is possible. A picture of multiple organ failure develops.



Laboratory tests for sepsis show leukocytosis with a shift of the formula to the left, but later, with a decrease in the reactivity of the body and the development of hypoproteinemia (due to massive breakdown of blood proteins), leukopenia occurs. Anisocytosis, poikilocytosis, pathological granularity appear, ESR increases to 60 mm / h, anemia progresses, bilirubin, creatinine, and blood urea increase. There is hemocoagulopathy in the form of DIC syndrome. The immunogram shows signs of immunodeficiency.

Blood culture is mandatory, despite the fact that more than half of the patients do not have microflora growth. Blood culture should be taken three days in a row.

Treatment. If a primary purulent focus is detected, regardless of the localization, surgical treatment is performed, which consists in opening and adequate drainage of the focus cavity. During the operation, the tissues are dissected over the site of fluctuation or softening, purulent exudate is evacuated, necrotic tissues are removed, the cavity is revised with the separation of bridges, the wound is washed with antiseptics and adequate drainage is provided. In the future, the wound is carried out in an open way, taking into account the phase of the wound process.

If there is a purulent focus in the abdominal cavity, laparotomy is performed, the abscess is opened and drained. Opening of a lung abscess is performed either with a bronchoscope (during bronchoscopy, the cavity is opened and drained through the tracheobronchial tree, the drainage tube is removed through the nasal passages), or by puncture through the chest wall.

In all cases, it is necessary to conduct a microbiological study of the exudate with the determination of sensitivity to antibiotics.

Antibacterial therapy. If sepsis is suspected, blood samples and contaminated biological fluids should be collected for rapid microbiological examination before prescribing antibacterial therapy. It is recommended to start antibacterial therapy with broad-acting antibiotics, always in high doses and parenterally.

When the primary focus is located in soft tissues or in the abdominal cavity, the use of carbapenems or a combination of cefipime with metranidazole is recommended as first-line agents, reserve agents – cephalosporins of 3-4 generations or fluoroquinolones in combination with metranidazole (clindamycin), a combination of cefoperazone/sulbactam with amicocin, ticarcillin/clavunate or piperacillin/tazobactam with aminoglycosides. In staphylococcal sepsis, the most effective drugs are vancomycin and linezolid. In severe nosocomial sepsis, carbapenems are indicated, sometimes cefepime or protected anti-pseudomonas beta-lactams and ciprofloxacin. In anaerobic sepsis, a combination of synthetic penicillins with metranidazole, thienam is used, while in clostridial sepsis, natural penicillins with metranidazole are used. From superinfection with fungi, fluconazole, amphotericin B are indicated.

Infusion therapy. Detoxification includes infusions up to 3-6 liters per day, but always taking into account diuresis, forced diuresis, with the development of acute renal failure, the use of hemodialysis.

Infusion therapy is aimed at correcting homeostasis disorders and reducing the concentration of toxins, normalizing microcirculation and tissue perfusion. To maintain carbohydrate balance, 5%, 10% or 40% glucose solutions are administered intravenously together with insulin (for 3-4 mg of glucose, 1 unit. insulin).

Anaerobic gas infection.

Anaerobic clostridial (spore-forming) infection-gas gangrene-is a pathological process caused by anaerobic clostridia. It is characterized by the absence of an inflammatory reaction and a progressive increase in edema, gas formation, tissue necrosis, severe course of the disease, rapid progression of intoxication.

Anaerobic infection is caused by: Clostridiumperfringens, Cl. Oedematiens, Cl septicum, Cl. Histolyticum.

The percentage frequency of anaerobic infection in gas gangrene or phlegmon is expressed in the following terms: Clostridiumperfringens-in 50-90% of cases, Cl. Oedematiens – in 15-50%, Clsepticum-in10-30%, Cl. Histolyticum – in 2-6%.

Наиболее часто встречается Clostridium perfringens is most commonly foundperfringensalone or in symbiosis with aerobes. The features of its action include rapid gas formation in the tissues and severe intoxication.

Cl. Oedematiens is the second most common, with rapid development of edema in the tissues and very severe intoxication prevailing. Due to tissue edema and compression of vascular bundles, ischemia and tissue necrosis develop, which greatly increases the development of gangrene.

Cl. septicum is less likely to cause anaerobic infection and in these cases causes blood-serous edema and toxicosis in the tissues.

Cl. Histolyticum alone causes the development of the disease quite rarely, but in combination with other microbes leads to rapid melting of tissues and severe intoxication.

The toxic effect of anaerobes contributes to the damage and subsequent death of healthy tissues, which become a good breeding ground for microorganisms. The richness of glycogen makes muscles a particularly favorable environment for the development of anaerobes. All anaerobes have the ability to decompose sugar with the formation of gas, especially rapid gas formation occurs during the decomposition of glycogen and muscle proteins.

When anaerobes act on tissues, two phases are distinguished: 1) toxic edema and 2) gas formation and gangrene of the muscles and connective tissue.

Classification of anaerobic infection. There are three classifications of anaerobic infection:

1). **Pathoanatomic service**:

edematous-34.7 %

mixed -29.2 %
emphysematous -19.7 %
necrotic - 9.3 %
phlegmonous-7.1 %
fabric-melting – 0.6 %.

The highest mortality rate is observed in tissue-melting and reaches 90% of cases of damage.

2). **Clinical**: Lightning-fast, develops in the first hours, proceeds violently and at the same time has a high mortality rate.

Sharp shape. It should be noted that there is no chronic course of anaerobic infection.

3). **Anatomical:** epifascial (the infection has not yet penetrated the intermuscular and muscular spaces) and subfascial. Determining the anatomical distribution of the process is of great importance for solving the problem of the depth of surgical incisions.

Clinical picture. The incubation period of gas infection is quite short – on average 2-4 days. The shorter the incubation period, the more severe the disease is. Sometimes there are lightning-fast forms with the development of the disease in the first hours after the injury.

Locally, with gas gangrene, there are strong bursting pains in the limb, a feeling of tightness of the bandage. When examining the site of the lesion, edema, pallor of the skin and blue-purple spots and stripes are revealed.

There is subcutaneous emphysema and crepitation, determined by palpation or radiology.

Air in soft tissues
defined by the Rg-ci

When pressing on the edges of the wound, gas bubbles are often released from the depth. The rapid increase in edema is confirmed by the "ligature symptom": the ligature tied around the limb quickly cuts into the skin. This symptom can serve as a differential test between gas gangrene and phlegmon of the limb.

General symptoms are typical of intoxication, but intoxication increases rapidly and is much more severe than with any other infection. The pulse rate reaches 140-160 beats. in min. Breathing up to 30-40 per minute. Blood pressure drops to 80-60 mmHg. Soon enough, toxic shock develops. It is characteristic to maintain consciousness until death, but there are cases when the patient falls into a state of euphoria. In biochemical and clinical blood tests, a picture characteristic of severe intoxication develops.

Prevention. Early and thorough treatment of contaminated wounds in compliance with all the rules of asepsis and antiseptics. Prophylactically, patients are given anti-gangrenous serum -30,000 AU (antitoxic units). The antibody composition of the prophylactic dose of anti-gangrenous serum is as follows: against Clostridiumperfringens 10 000 AE, against Cl. septicum 10 000 AE, against Cl. Oedematiens 10,000 AU. Serum is best administered intravenously in a mixture with 100 ml of saline solution.

The use of anaerobic bacteriophage is also of preventive importance.

Treatment. 1. **Specific treatment** it consists in the use of anti-gangrenous serum. Prior to the establishment of a bacteriological diagnosis, polyvalent serum is used. The therapeutic dose of polyvalent serum is 150,000 AU (50,000 AU of each antibody). After clarifying the anaerobe, monovalent serum should be administered only against the isolated pathogen (50,000 AE).

The serum is administered intravenously in a slow drip on 400 ml of saline solution, it is permissible to pierce the soft tissues around the wound with serum to create a barrier of antibodies.

2. **Surgical treatment** consists of:

a) in the application of so-called lampas cuts – several deep and wide incisions on the limbs, reaching a length of up to 40-50 cm;

b) in guillotine amputation-amputation without applying a tourniquet and sutures.

After the operation, the wound is abundantly treated with oxidizing agents-potassium permanganate or hydrogen peroxide, and conditions are created for constant aeration of the wound. Good results were obtained with the use of hyperbaric oxygenation.

3. **Non-specific treatment** is used to eliminate toxicosis, for which they conduct infusion therapy in a volume of more than 5 liters, use antibacterial therapy, provide the patient with rest, high-calorie nutrition.

The outcome of gas gangrene depends on the timeliness of diagnosis, the state of the body before the disease, the virulence of the microflora and its dose, the localization and degree of development of the process.

Tetanus.

Tetanus is an acute specific infectious disease caused by the anaerobic spore-bearing bacillus Clostridiumtetani. Cl. tetani occurs as a saprophyte in the intestines of domestic animals as well as humans. Released with feces, it gets into the soil. In dry manure and soil, the tetanus spore can persist for up to 25-30 years. Thus, with any contaminated wound, a direct threat of tetanus disease is created. The optimal temperature for spore opening and reproduction of tetanus bacillus is 35-45°C., so in hot regions of the world, such as the Sahara Desert, tetanus disease is not present.

Classification.

I. By type of injury: wound, post-injection, post-burn, postoperative, postpartum, after abortions, after frostbite, after trauma, etc.

II. By prevalence:

1. General tetanus: a) the primary general form;

b) descending form; c) ascending form.

2. Local (limited) tetanus: a) limbs; b) head;

c) torso; d) a combination of limited localizations (arm, torso, etc.).

Local tetanus is characterized by a lighter course and delimited muscle damage with localization in the wound area.

The incubation period for tetanus is from 4 to 14 days, but the disease can develop at a later time. There are cases of tetanus with an incubation period of up to six months. It is noted that the shorter the incubation period, the more severe the disease is and the higher the mortality rate. A person is very sensitive to tetanus toxin and does not have a natural immune system. The selected Cl. tetani exotoxin consists of two components: tetanospasmin and tetanohemolysin. The first one acts on the nervous system, leads to the development of tonic and clonic spasms of striated muscles, and the second one destroys red blood cells. Tetanohemolysin has no practical significance in the pathogenesis of tetanus.

The disease occurs when tetanus bacillus enters the body through damaged integuments.

Tetanus Entrance Gate

Once in a favorable environment, tetanus bacillus at an optimal temperature of 36-37°C begins to multiply rapidly and release toxins. The blood stream delivers toxins to the brain and spinal cord and, affecting mainly the motor horns of the spinal cord, cause convulsions.

General tetanus clinic: symptoms of intoxication - headaches, pale skin, heavy sweating, weakness, tachycardia, tachypnea, fever, sometimes up to 40 ° C. the main symptoms of tetanus are clinical and tonic seizures, which can occur on any external stimulus. There is a spasm of the facial muscles - the so-called sardonic smile, and trismus (spasm of the masticatory muscles and neck muscles).

The most severe form of tetanus is **opisthotonus** (spasm of the neck muscles, long back muscles and abdominal muscles, which leads to overextension of the trunk), can lead to muscle tears and spinal fractures..

There is a delay in stool and urination. Convulsions involve the respiratory intercostal muscles and the diaphragm, leading to respiratory arrest.

In ascending tetanus, seizures begin in the lower extremities and gradually spread upward, while in descending tetanus, seizures spread from top to bottom.

Prevention. 1. early surgical treatment of the wound, the appointment of antibacterial therapy

2. Specific immunization: 1.0 ml of AS (tetanus toxoid) and 3000 IU of PSS (tetanus serum) according to Bezredko.

Treatment: hospitalization in the intensive care unit with isolation of the patient from visual, auditory and mechanical external stimuli. Place the patient in a separate darkened box.

Drug treatment: administration of sedatives, barbiturates, neuroleptics, infusion therapy up to 2.5 liters, correction of protein water-electrolyte balance, antibacterial therapy. If necessary, the use of muscle relaxants in

the presence of a ventilator.

Specific treatment – intravenous administration of tetanus serum for 150 thousand rubles.IU per day. The average therapeutic dose per course of treatment is 200-350 thousand IU. In critical cases, PSS can be administered at a dose of up to 1200 thousand IU.

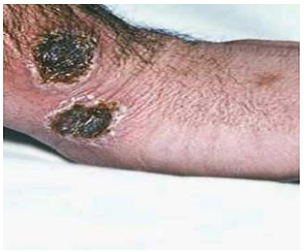
The fatal outcome can reach 50 %.

Anthrax.

Acute specific infectious disease caused by anthrax shelf. Infection is more likely to occur from a sick animal, the infection can also be carried by flies. The incubation period is 2-7 days. A person has three forms of anthrax-skin, intestinal and pulmonary. Of surgical interest is the cutaneous form, which is expressed in the formation of a specific carbuncle.

The place of carbuncle formation is painless, after 24-48 hours a blue-purple bubble appears.

After the pustule breaks through, the wound is covered with a solid, painless black scab with gangrenous skin areas.



Inflammation in anthrax is never purulent, the nearest lymph nodes increase, hemorrhagic lymphangitis develops.

In mild cases, the general signs of the disease are manifested by a short-term fever, in severe cases-by pronounced intoxication phenomena, and the mortality rate is 20-26 %.

Treatment of anthrax carbuncle should be carried out conservatively: rest, ointment dressings, immobilization. Incisions, probing, and tamponade are contraindicated.

Antibiotic therapy: anthrax bacteria are sensitive to penicillins-up to 10 million are administered. Units per day. Specific treatment consists in intravenous administration of anti-ulcer serum from 50 to 150 thousand units. In addition, intravenous administration of salvasan at 0.6-0.9 g is recommended.

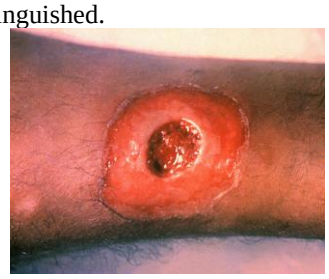
Diphtheria of wounds.

Diphtheria of wounds develops when Lefler's wand enters the wound.

Clinical data are characterized by the appearance of a dirty gray film that is difficult to remove on the wound surface.

Diphtheria infection of fresh and granulating wounds is distinguished.

In the first case, necrosis develops under a hard-to-remove film with severe inflammation of the wound, often with lymphangitis. General condition is serious.



Diphtheria lesion of granulating wounds proceeds much more easily, only film overlays predominate, which are easily formed again after removal. A feature of diphtheria of wounds is poor symptoms. The diagnosis is made on the basis of a bacteriological study.

Treatment consists of subcutaneous, intravenous or intravenous administration of anti-diphtheria serum in a dose of 3000-8000 immunizing units. The place is recommended to rest, dressings with antiseptic solutions in combination with anti-diphtheria serum.

Actinomycosis.

Chronic infectious non-contagious disease caused by radiant mycelium-actinomycetes. According to the endogenous theory, the causative agents of the disease are radiant mycelia that constantly saprophytize in the body (oral cavity, gastrointestinal tract). The exogenous theory has lost its dominant meaning.

Features of the development of the disease depend on the ways in which actinomycosis spreads in the patient's body. The predominant pathway is contact, and there are data on the lymphogenic pathway of spread.

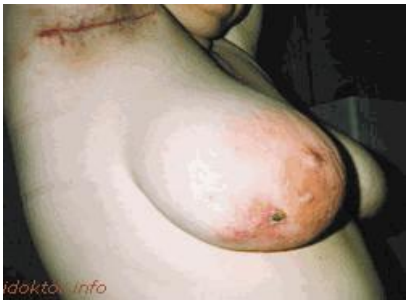
Thoracic actinomycosis. It is 10-20% among other localizations. The clinical picture of the disease is poor and usually proceeds unnoticed. Frequent colds and respiratory diseases, dull pains in the chest, shoulder, shoulder blade. The diagnosis is virificated by microscopic examination of sputum, druses or mycelia of actinomycetes are detected.

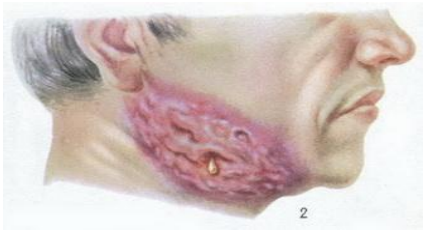
One of the complications is a specific lesion of the heart.

Abdominal actinomycosis (10-20%). In most cases, actinomycosis develops in the cecum. The disease

begins acutely, manifests itself with severe cramping abdominal pain, fever. The progression of the process is expressed in an increase in the infiltrate, which combines the actinomycosis infiltrate itself and the nearest organs and tissues into a common conglomerate.Most often, actinomycosis spreads from the primary focus to the anterior abdominal wall, forming an abscessed infiltrate in it.

In addition, there is actinomycosis of the skin, bones, genitourinary organs and kidneys, brain spinal cord, face.





For timely diagnosis, a comprehensive study is necessary, including X-ray examination of the lungs or bones, clinical and laboratory methods. A reliable diagnostic method is microscopy of the discharge from the lesion area in order to detect drusus.

Treatment of patients is complex, based on immunotherapy.

Specific immunotherapy – administration of actinolysate (iv 3.0 · 2 times a week for 3 months); APV (actinomycete polyvalent vaccine) - IV daily, starting from 0.1 ml to 1 ml. The course duration is 3 months.

Syphilis of bones and joints.

There are the main forms of bone lesions in syphilis: periostitis, ostitis, osteomyelitis. Specific syphilitic bone damage is less characteristic of early syphilis, more often manifested in the tertiary period. Early syphilis is characterized by specific polyarthritis: damage to the synovial membranes without damage to cartilage and bones. In the secondary period, joint damage occurs as polyarthritic synovitis. The joints are swollen, slightly hyperemic, and active movements are limited.there is a symmetrical lesion of the knee, ankle, sternoclavicular and small joints of the hands or feet. Patients complain of focal or diffuse bone soreness, which increases at night, is relieved by movement, and is aggravated by thermal procedures. The examination reveals a local swelling of the test consistency. Radiographs show a significant periosteal reaction, thickening of the periosteum, and signs of osteolysis.

In late syphilis, osteoperiostitis usually occurs simultaneously in several bones

The main manifestations of bone syphilis in the tertiary period are gum infiltrations. In gummous processes in the bone, granulation tissue causes ostitis with subsequent reactive changes. The predominance of proliferation processes over destruction distinguishes syphilis from tuberculosis.

The gummous process that occurs in the inner layer of the periosteum can penetrate through the vessels into the compact substance of the mucosa, into the spongy substance, which leads to destructive changes in these layers. Destructive changes depend on the localization of the process.

In syphilis, the diaphysis of the long tubular bones, usually the tibia, is more often affected. Flat bones of the skull and sternum are involved in the process in 5% of cases, the frontal and parietal bones are most often affected.

Syphilitic spondylitis (spinal lesion) occurs in 2-6% of cases, more often the body of one or two vertebrae is affected, usually in the cervical region. At the same time, immobility of the affected area develops, and spontaneous pain occurs.

The diagnosis is made on the basis of the clinical picture, X-ray data, serological blood tests (Wasserman reaction – RW) and the results of a trial anti-syphilitic treatment.

Surgical intervention (opening of abscesses) is performed only when a secondary infection is attached.

VI. SURGICAL DISEASES OF SEROUS CAVITIES

PERICARDITIS

PERICARDITIS is an acute or chronic inflammation of the pericardial sac. There are fibrinous,serous-fibrinous, hemorrhagic, xanthomatous, purulent, putrefactive pericarditis.

Etiology: infection (viruses, bacteria, rickettsiae, fungi, protozoa), rheumatism, rheumatoid arthritis, systemic lupus erythematosus, myocardial infarction, uremia, trauma, including surgery, ionizing radiation, tumors and hemoblastoses, parasitic infestations; for some pericarditis, the causes of their occurrence are not

established (idiopathic).

Pathogenesis - often allergic or autoimmune, with infectious pericarditis, infection can be a trigger; direct damage to the membranes of the heart by bacterial or other agents is not excluded.

Symptoms and course are determined by the underlying disease and the nature of the effusion, its amount (dry, effusive pericarditis) and the rate of accumulation. Initial symptoms: malaise, increased body temperature, retrosternal or precardial pain, often associated with respiratory phases, and sometimes resembling angina pectoris. Pericardial friction noise of varying intensity and prevalence is often heard. The accumulation of exudate is accompanied by the disappearance of precardial pain and pericardial friction noise, the appearance of shortness of breath, cyanosis, swelling of the cervical veins, weakening of the heartbeat, expansion of cardiac dullness, but with a moderate amount of effusion, heart failure is usually moderate. Due to a decrease in diastolic filling, the stroke volume of the heart decreases, heart sounds become deaf, the pulse is small and frequent, often paradoxical (a drop in filling and pulse tension during inspiration).

With constrictive (compressive) pericarditis, atrial fibrillation or atrial flutter often occurs as a result of deforming joints in the atrial region; at the beginning of diastole, loud pericardia is heard.

With rapid accumulation of exudate, cardiac tamponade may develop with cyanosis, tachycardia, weakening of the pulse, excruciating attacks of shortness of breath, sometimes with loss of consciousness, and rapidly increasing venous congestion. With constructive pericarditis with progressive Cicatricial compression of the heart, circulatory disorders in the liver and portal vein system increase. High central venous pressure, portal hypertension, ascites (pseudocyrrhosis of the Peak) are detected, peripheral edema appears; orthopnea, as a rule, is absent.

The spread of the inflammatory process to the mediastinal tissues and pleura leads to mediastinopericarditis or pleurisy, with the transition of inflammation from the epicardium to the myocardium (surface layers), myopericarditis develops.On the ECG in the first days of the disease, a concordant rise of the 5T segment in the standard and thoracic leads is noted, in the subsequent segment S T shifts to the isoelectric line, the T wave flattens or undergoes inversion; with a significant accumulation of effusion, the voltage of the QRS complex decreases.

X-ray examination reveals an increase in the diameter of the heart and a trapezoidal configuration of the heart shadow with a weakening of the pulsation of the heart contour. With prolonged pericarditis, calcification of the pericardium (carapace heart) is observed.

Echocardiography is a reliable method for detecting pericardial effusion, and jugular phlebography and phonocardiography are also used for diagnosis.

Differential diagnosis is made with the initial period of acute myocardial infarction and acute myocarditis.

Treatment. In case of allergic or infectious-allergic nature of pericarditis, corticosteroid drugs (prednisone 20-30 mg/day) and nonsteroidal anti-inflammatory drugs are used in the following daily doses:: acetylsalicylic acid 3-4 g, rheopyrin 3-4 tablets, ibuprofen (brufen) 0.8-1.2 g, indomethacin 75-150 mg.

For infectious and pyogenic pericarditis (staphylococcal, pneumococcal, etc.), antibiotics are used in accordance with the established or suspected pathogen (penicillins, aminoglycosides, cephalosporins, etc.).

For parasitic pericarditis, antiparasitic agents are prescribed. In case of a threat of cardiac tamponade, a therapeutic pericardial puncture is performed. In case of congestive phenomena, diuretics are used-furosemide (lasix) inside or in/m 40 mg or more, hypothiazide 50-100 mg inside, etc. A sharp increase in central venous pressure is an indication for bloodletting (up to 400 ml).

Surgical treatment (pericardectomy) is used for constructive pericarditis in the case of significant circulatory disorders and purulent pericarditis.

The prognosis is most unfavorable for tumor and purulent pericarditis.

PLEURAL EMPYEMA

Empyema of the pleura (purulent pleurisy)- accumulation of purulent exudate in the pleural cavity with secondary compression of the lung tissue in pleurisy.

Classification

- • By localization •• Unilateral or bilateral Restricted subtotal; total * * Basal or paramediastinal•
- Due to Metapneumonic, Parapneumonic, Postoperative,
- Downstream Acute Chronic
- • According to the amount of effusion, a small pyothorax is distinguished — an accumulation of exudate in the pleural sinuses (an amount of 200-500 ml); a medium pyothorax is an accumulation of exudate up to the scapula angle in the VII intercostal space (an amount of 500-1000 ml); a large pyothorax is an accumulation of exudate above the scapula angle (an amount of more than 1 liter).

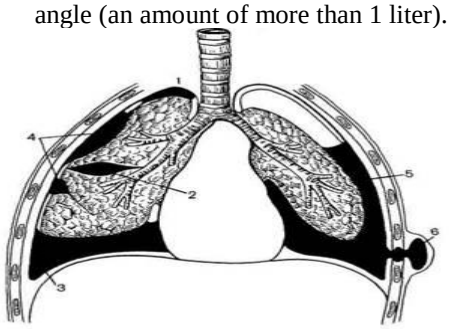
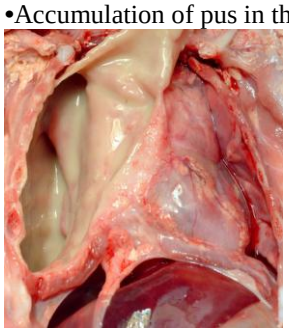


Рис. 6.16. Виды эмпиемы плевры.
1 — верхушечная (апикальная); 2 — междолевая (интерлобарная); 3 — базальная; 4 — пристеночная; 5 — пневмоторакс; 6 — прорыв гноя под кожу (осложнение эмпиемы плевры).

Etiology• Pathogens • * * staphylococci • * pneumococci • * facultative and obligate anaerobes • Direct route of infection •• Lung injury •• Chest injuries • * Esophageal rupture •• Breakthrough of abscess, gangrene of the lung, caverns •• Pneumonia • * Tuberculosis •• Progression of bacterial lung damage (abscess or bacterial destruction • * * Bronchiectasis •• Lung resection and other chest surgeries •• Pneumothorax * * Acute mediastinitis •• Osteomyelitis of the ribs and vertebrae * Indirect route of infection * • Subphragmatic abscess • * Acute pancreatitis • * Liver abscesses •• Inflammation of the soft tissues and bone framework of the chest wall *Idiopathic empyema.

Pathomorphology* Hyperemia and leukocytic infiltration of the pleura • Fibrin deposition •Accumulation of pus in the pleural cavity



Clinicalpicture

• Acute pleural empyema •• Cough with sputum discharge. Prolonged and frequent coughing attacks with the release of a large amount of sputum indicate the presence of a bronchopleural fistula • * Chest pain is minimally pronounced with calm breathing, sharply increases during a full deep breath •• Shortness **of breath** •• Voice tremor disorder. * * Dull or blunted percussion sound on the affected side, the upper limit of dullness corresponds to the Ellis-Damoiseau-Sokolov **line** • * Weakened or absent breathing during auscultation over the effusion **area** • * Bronchial breathing over the compressed lungs adjacent to the effusion •• Redness of the skin occurs only when pus breaks out from the empyema cavity under the skin •• The general condition progressively worsens: weakness, loss of appetite, weight loss, hectic body temperature, rapid pulse.

* Chronic pleural empyema • * Body temperature may be subfebrile or normal, with impaired outflow of pus becoming hectic •• Cough with purulent sputum • * Chest deformity on the affected side due to narrowing of intercostal spaces. Children develop scoliosis •• Percussion data depend on the degree of filling of the cavity with pus, respiratory noises above the cavity are not heard.

Laboratorytests* Leukocytosis, left shift of the leukocyte formula, hypo - and dysproteinemia, increased ESR • Analysis of pleural fluid — exudate (relative density above 1.015, protein above 30 g / l, albumin/globulin ratio-0.5-2.0, Rivalt test positive, white blood cells above 15).

Specialstudies• Thoracocentesis - pleural fluid is cloudy, thick, gradually turning into true pus, has a specific unpleasant smell * Laboratory examination of aspirated fluid •• Gram-based smear bacterioscopy •• Bacteriological research (often the results of these methods differ) •• pH determination — with an empyema pH of less than 7.2 •• Glucose concentration below the blood **glucose concentration** • X-ray examination •• The mediastinum is shifted to the side opposite to the side of effusion accumulation * * Basal darkening with a horizontal level in case of putrefactive infection or bronchopleural fistula * CT allows you to most accurately determine the presence of fluid in the pleural cavity and localize intrapleural osumkovanie • Pleurophistulography-contrast examination of the pleural cavity through fistulas *Ultrasound allows you to determine the amount of effusion, localize the site of puncture and drainage of the pleural cavity.

TREATMENT

Generalprinciples* Treatment of the underlying disease • Early complete removal of exudate from the pleural cavity by puncture or drainage • Expansion of the lung using constant aspiration, physical therapy •Rational antibiotic therapy.

Conservativetherapy* Early acute empyema — repeated pleural punctures with purulent exudate aspiration and adequate antibiotic therapy (clindamycin, ceftriaxone in combination with metronidazole; aminoglycosides, monobactams, carbapenems can be prescribed) •• Washing of the pleural cavity with the introduction of antibiotics, proteolytic enzymes * * Immunostimulating therapy •• Blood UFR • Infusion therapy and partial parenteral nutrition *Developed empyema with thick purulent exudate is an indication for prolonged closed drainage.

Surgicaltreatment

* Acute empyema • * Free empyema of the pleura-constant washing of the pleural cavity through two tubes, after 2-3 days the contents are sucked out through both tubes and the lung **is completely straightened** • * Wide thoracotomy with rib resection, pleural cavity toilet and subsequent drainage is indicated in the presence of large sequesters and clots in the pleural cavity •• In the presence of a bronchial fistula — tamponade of the corresponding bronchus •• If the above

measures are ineffective, early decortication of the lung is indicated.

***** Chronic empyema •• Empyema sanitation via active aspiration drainage •• In the presence of a bronchial fistula: the same + bronchial tamponade •• If ineffective-surgical treatment: repneumolysis, decortication of the lung, curettage of the pyogenic layer to the fibrous capsule, suturing of the bronchial fistula or resection of the affected area of the lung •• With tuberculous empyema, the volume of surgery is increased — a total parietal pleurectomy is performed.

Complications * Perforation • * Into the pulmonary parenchyma with the formation of bronchopleural fistulas • * Through the chest with accumulation of pus in the soft tissues of the chest wall * Septicopyemia • Secondary bronchiectasis • Amyloidosis.

The prognosis with timely treatment is favorable, with chronic empyema it may be unfavorable.

PERITONITIS

Peritonitis is ***an inflammation of the peritoneum, accompanied not only by local changes in the peritoneal cover, but also by a severe general reaction of the body to purulent intoxication.***

In the vast majority of cases, it develops secondarily as a complication of a purulent disease or violation of the integrity of any organ of the abdominal cavity (vermiform process, stomach, gallbladder, intestines, etc.). In rare cases, the primary cause is not found even at autopsy and such peritonitis is called cryptogenic.
Depending on the principle used as a basis, the following classifications of peritonitis are distinguished::

- By etiology: aseptic and infectious.
- By type of pathogen: staphylococcal, streptococcal, caused by Escherichia coli; caused by mixed flora, etc..
- By the prevalence of the process: **Common**: general (diffuse), when the entire peritoneum is affected, diffuse-part of the peritoneum is affected, but the process does not have a clear restriction.**Local** (delimited-the affected area of the peritoneum is isolated from the abdominal cavity by adhesions and non-delimited - the affected area of the peritoneum is not isolated from the abdominal cavity),.
- For reasons of occurrence: in inflammatory and destructive processes of the abdominal cavity, perforative, traumatic, postoperative, hematogenic, cryptogenic, etc.
- Peritonitis is also distinguished by its source of origin (appendicular, after perforation of a stomach ulcer, etc.)
- By clinical course (acute, chronic)
- By the nature of exudate (serous, serous-fibrinous, purulent, hemorrhagic, putrefactive, etc.).

Symptoms and course:
Peritonitis is secondary, so its clinical picture is layered on the symptoms of the primary disease. The patient's complaints are reduced to abdominal pain, nausea, vomiting, weakness, thirst, shortness of breath, etc. Examination allows you to notice pointed facial features of a gray-earthy color, sunken eyes, a difficult type of breathing, immobility of the abdominal wall, bloating, maintaining consciousness with some inhibition of reactions to various stimuli, a deaf voice.
There are also dry mucous membranes, dry, coated tongue, repeated vomiting, regurgitation. On palpation of the abdomen, tension and pain of the abdominal wall are observed, which are almost always expressed somewhat more in the area of the source of peritonitis.
Treatment:
Patients with purulent peritonitis need immediate hospitalization and emergency surgery. Comprehensive treatment is provided, including surgical and conservative methods. The operation aims to eliminate the primary focus of infection, remove pus and ensure not only a single, but also repeated administration of antibiotics into the abdominal cavity (through drainage). If it is impossible to eliminate the focus of infection, drainage is performed to create a reliable outflow of pus from the abdominal cavity.
Conservative tactics include:

1. fight against microflora and intoxication;
2. increasing the body's immunobiological strength;
3. improving the functions of the patient's organs and systems.

LIST OF LITERATURE

1. Clinical Surgery: A National Guide in 3 volumes (edited by V. S. Savelyev, Moscow, GEOTAR-Media, 2009).)
2. 80 lectures on surgery edited by S. V. Savelyev, Moscow: Lettera Publ., 2008, 912 articles.
3. V. K. Gostischev: A Guide for doctors, Moscow, GEOTAR-Media, 2007, 768 p.
4. Selected course of lectures on purulent surgery edited by V. D. Fedorov. Miklos Publishing House-Moscow: 2010

skinnecrosis develops, ulcers will rejoice. With

the addition of a secondaryinfection, the inflammatory process spreads to the subcutaneous fat, i.e. phlegmon develops.