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Topic: SUPPURATIVE INFLAMMATORY DISEASES

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SUPPURATIVE INFLAMMATORY DISEASES

Symptoms of suppurative-inflammatory diseases can be local and general.

The local features of inflammation are characterized by the classical signs of inflammation:

- redness (rubor),
- swelling (tumor),
- pain (dolor),
- increase in local temperature (color),
- organ function disorder (functio laesa).

The spread and manifestation of the inflammatory process determine the extent of the local clinical presentation. Palpation of soft infiltrate, positive fluctuation sign indicate that the infiltrative phase has moved into the suppurative (purulent) phase. The local clinical signs of progressive suppuration are zones or stripes of redness in the skin (lymphangitis), firm tender strings of induration along the superficial veins (thrombophlebitis), appearance of firm tender indurations at the sites of the regional lymph nodes (lymphadenitis).

Progression of the inflammatory process is accompanied by the progress of both local and general inflammation and intoxication signs: body temperature rise, chills, agitation or alternatively weakness, and in extreme cases mental confusion, sometimes unconsciousness, headaches, general malaise, fatigue, a rapid pulse, extreme deviations in the blood picture, signs of liver and kidney function disorder, blood pressure reduction and congestion in the pulmonary blood circulation. The above symptoms can either be pronounced or latent depending on the character, type, extent, location and spread of the inflammation as well as the organism response to it.

In surgical infections the body temperature can rise to 40°C and over, there occur recurrent chills and a headache, hemoglobin level tends to decrease sharply as do the erythrocytes, leucocytes increase - in severe cases up to $20,0 - 25,0 \times 10^9/l$. Plasma globulins increase while albumins decrease, there is loss of appetite and intestinal disorders, constipation, presence of protein and cylinders appear in the urine. Intoxication leads to hemopoiesis distortion as a result of which the patient becomes anemic accompanied by marked changes in the white blood components: immature blood cells appear in the peripheral circulation, a shift of the leukocyte formula to the left is observed (a decrease in the mature and stab forms of neutrophils). A sharp increase in the erythrocyte sedimentation rate (ESR) occurs during the inflammatory process. It is notable that having once appeared at the onset of inflammation it tends to persist for a long time even after the inflammation is over. Sometimes the spleen and liver can enlarge, with yellowish sclera.

The clinical sign of endogenic intoxication is severity: the more pronounced the intoxication extent, the more vividly it manifests itself. In mild intoxications the

skin is pale, and in severe cases the skin is sallow; there is acrocyanosis, and hyperemic face. Pulse rate is fast-up to 100 - 110 beats per min, in severe conditions more than 130 beats per minute, blood pressure falls. The patient becomes dyspnoeic — breathing rate reaches 25 - 30 per min, and in severe intoxication more than 30 in a min. Change in the functions of the CNS are a vital feature of intoxication: from light euphoria at the beginning to depression or psychosis in the case of toxemia, in extreme cases of intoxication the patient can develop intoxication delirium. Assessment of diuresis plays a vital role in determining the severity of intoxication. There is a reduced output of urine in severe intoxications and in extreme cases acute renal failure can occur with associated oliguria and sometimes anuria.

Special methods of investigations are used to confirm the diagnosis of suppurative-inflammatory disease-puncture, roentgenography, endoscopic methods, clinical laboratory and biochemical tests of blood, urine and exudate. Microbiological analysis has the potential not only to isolate the causative agent, its pathogenic properties but also to determine the microorganism's sensitivity to the antibacterial medications that is about 30%. An important role in the comprehensive treatment of suppurative-inflammatory disease is assigned to the assessment of the patient's immune status, in order to select the appropriate and tailored immune therapy.

The adequate and timely surgical treatment of trauma and acute surgical disease in combination with rational antibiotic therapy have contributed to reduced incidence of suppurative infections and led to a change in the classical outcome of suppurative infections. These days when antibiotic therapy is widely used before the patients' admittance to the hospital, sometimes not even prescribed by a doctor, surgeons often have to deal with patients with advanced and critical forms of suppurative processes (purulent appendicitis, gall bladder empyema, peritonitis, purulent pleurisy, mastitis, phlegmon etc.). These patients are admitted without high body temperature, with moderate leucocytosis, minimal changes in ESR and minimal intoxications.

Abscess. An abscess is a collection of pus in any part of the body that, in most cases, causes swelling and inflammation around it.

Abscesses occur when an area of tissue becomes infected and the body's immune system tries to fight it. White blood cells move through the walls of the blood vessels into the area of the infection and collect in the damaged tissue. During this process, pus forms. Pus is the buildup of fluid, living and dead white blood cells, dead tissue, and bacteria or other foreign substances.

Abscesses can form in almost any part of the body. The skin, under the skin, and the teeth are the most common sites. Abscesses may be caused by bacteria, parasites, and foreign substances.

Abscesses in the skin are easy to see. They are red, raised, and painful. Abscesses in other areas of the body may not be seen, but they may cause organ damage.

Types of abscesses include: abdominal abscess, amoebic liver abscess, anorectal abscess, Bartholin abscess, brain abscess, epidural abscess, peritonsillar abscess, pyogenic liver abscess, skin abscess, spinal cord abscess, subcutaneous abscess, tooth abscess.

Major complications are spreading of the abscess material to adjacent or remote tissues and extensive regional tissue death (gangrene). The main symptoms and signs of a skin abscess are redness, heat, swelling, pain and loss of function. There may also be high temperature (fever) and chills. Internal abscess is more difficult to identify, but signs include pain in the affected area, a high temperature, and generally feeling unwell. Internal abscesses rarely heal themselves, so prompt medical attention is indicated if such an abscess is suspected. An abscess could potentially be fatal (although this is rare) if it compresses vital structures such as the trachea in the context of a deep neck abscess. If superficial, abscesses may be fluctuant when palpated. This is a wave-like motion which is caused by movement of the pus inside the abscess.

Risk factors for abscess formation include intravenous drug use. Another possible risk factor is a prior history of disc herniation or other spinal abnormality, though this has not been proven. Abscesses are caused by bacterial infection, parasites, or foreign substances. Bacterial infection is the most common cause. Often many different types of bacteria are involved in a single infection. In the United States and many other areas of the world the most common bacteria present is methicillin-resistant *Staphylococcus aureus*. Among spinal subdural abscesses, methicillin-sensitive *Staphylococcus aureus* is the most common organism involved. Rarely parasites can cause abscesses and this is more common in the developing world. Specific parasites known to do this include: dracunculiasis and myiasis.

An abscess is a defensive reaction of the tissue to prevent the spread of infectious materials to other parts of the body. The organisms or foreign materials kill the local cells, resulting in the release of cytokines. The cytokines trigger an inflammatory response, which draws large numbers of white blood cells to the area and increases the regional blood flow. The final structure of the abscess is an

abscess wall, or capsule, that is formed by the adjacent healthy cells in an attempt to keep the pus from infecting neighboring structures. However, such encapsulation tends to prevent immune cells from attacking bacteria in the pus, or from reaching the causative organism or foreign object.

An abscess has to be differentiated from haematoma, cysts and tumor degenerations. Diagnostic puncture can play a very important role here. The presence of gas - producing bacterial strains leads to the accumulation of gas in the abscess cavity, so-called gaseous abscess. Percussion of this kind of abscess gives a tympanic sound; X-ray films show the presence of gas bubbles with horizontal fluid levels beneath them (it is often observed in abscesses caused by agents of ichorous infections).

Treatment of an abscess is performed by incision, evacuation and drainage of the abscess cavity. Cold tuberculous abscesses are not to be incised since there is always danger of causing superinfections by suppurative strains. Small abscesses with well-formed capsules have to be removed in whole without opening them. An abscess has to be incised using the shortest surgical approach based on the anatomic and topographic peculiarities of the organ. Not infrequently an abscess is opened along a needle; the abscess is initially punctured with a needle, then the incision is made along the needle into the abscess cavity. The incision has to be made as much as possible towards the lower poles in order to create better conditions for drainage. In order to reduce contamination of the surrounding tissues during the process of incision, the surgical field is well isolated by gauze or napkins and after a small incision into the cavity an electric suction machine is used to evacuate the pus. After the pus has been suctioned, the incision is made longer, the cavity is explored by using the finger, breaking in the process the lacunae, and tissue sequestra are removed. All manipulations should be done carefully in order not to destroy the pyogenic membrane. The abscess cavity is washed with an antiseptic solution, and then drained with one or several plastic or PVC tubes, or gauze swabs soaked with some proteolytic enzymes, then antiseptics are packed into the cavity for drainage purposes. If drainage through the main incision is not adequate, another one is made on the opposite side — counter-aperture. Treatment of abscesses after incision and drainage is identical to that of infected wounds.

General therapeutic measures include body fortifying therapies, blood and plasma transfusions, and prescription of antibiotics taking into consideration the sensitivity results of the microbiologic analysis, specific therapeutic modalities (immunization with staphylococcal unatoxin, specific anatoxin etc.).

Phlegmon is a spreading diffuse inflammatory process with formation of suppurative/purulent exudate or pus. This is the result of acute purulent

inflammation which may be related to bacterial infection, however the term «phlegmon» mostly refers to a walled-off inflammatory mass without bacterial infection, one that may be palpable on physical examination.

Classifications:

1. By clinical course:
 - acute
 - subacute
2. By severity of condition:
 - mild
 - average
 - severe (with spreading to other location(s))
3. By location:
 - Superficial
 - cutaneous
 - subcutaneous
 - interstitial tissue
 - intramuscular
 - Deep
 - mediastinal
 - retroperitoneal
4. By etiology:
 - single
 - mix (e.g.: spore and non-spore forming anaerobes)
5. By pathogenesis:
 - per continuitatem (through neighbouring tissues)
 - hematogenous (through non-valvular veins like venous plexus of face e.g.: v. pterygoideus plexus → inflammation of veins (phlebitis) → thrombus formation in veins → embolization of thrombus into sinus venosus systems)
 - odontogenous
6. By exudative character:
 - purulent phlegmon
 - purulent-hemorrhagic phlegmon
 - putrefactive phlegmon
7. By presence of complications:

- with complications (disturbance of mastication, ingestion, speech, cardiovascular and respiratory system, peritonitis, lymphadenitis, loss of conscious if very severe, etc.)
- without complication

Signs of phlegmon are similar to the general symptoms of suppurative-inflammatory processes (a rise in the body temperature, weakness, malaise, and headache). These determine the patient's complaints, there is also pain and swelling at the site of infection, tenderness in movement as well as while changing position of the body.

Diagnostics:

- Complaints and clinical appearances
- Anamnesis
- Visual and Palpations
- Blood test – leukocytosis (up to $10-12 \times 10^9 /L$), decrease or absence eosinophils level, shift of white count differential to the left (neutrophilia), increase ESR (up to 35 – 40 mm/h).
- Urine test – presence of bacteria in urine, increase urinary leucocyte counts.
- X-ray test
- Ultrasound test

Treatment of phlegmon is surgical. At the initial can be allowed:

- bed rest and rest of the affected limb,
- antibiotic therapy,
- ultraviolet irradiation therapy as well as electrophoresis with chemotrypsin.
- novocain block of fascial case with antibiotics by the Vishnevsky method.

In the absence of positive effect within 12 - 24 hours or in the case of deterioration of symptoms the patient must be operated upon.

The phlegmon is incised under general anesthesia; pus and necrotic tissues are evacuated. Purulent tracts and pockets are opened; the wound is thoroughly washed with antiseptic solutions and drained. To ensure adequate drainage extra incisions (contra-apertures) are sometimes made. Treatment after surgery is similar to that of infected wounds.

A furuncle, also called a boil, is a deep folliculitis, infection of the hair follicle. It is most commonly caused by infection by the bacterium *Staphylococcus aureus*, resulting in a painful swollen area on the skin caused by an accumulation of pus and dead tissue. Boils which are expanded are basically pus-filled nodules. Individual boils clustered together are called carbuncles. Most human infections are caused by coagulase-positive *S. aureus* strains, notable for the bacteria's ability

to produce coagulase, an enzyme that can clot blood. Almost any organ system can be infected by *S. aureus*.

Boils are bumpy, red, pus-filled lumps around a hair follicle that are tender, warm, and very painful. They range from pea-sized to golf ball-sized. A yellow or white point at the center of the lump can be seen when the boil is ready to drain or discharge pus. In a severe infection, an individual may experience fever, swollen lymph nodes, and fatigue. A recurring boil is called chronic furunculosis. Skin infections tend to be recurrent in many patients and often spread to other family members. Systemic factors that lower resistance commonly are detectable, including: diabetes, obesity, and hematologic disorders. Boils can be caused by other skin conditions that cause the person to scratch and damage the skin. Boils may appear on the buttocks or near the anus, the back, the neck, the stomach, the chest, the arms or legs, or even in the ear canal. Boils may also appear around the eye, where they are called styes. A boil on the gum is called intraoral dental sinus, or more commonly, a gumboil.

Usually, the cause is bacteria such as staphylococci that are present on the skin. Bacterial colonisation begins in the hair follicles and can cause local cellulitis and inflammation. Additionally, myiasis caused by the Tumbu fly in Africa usually presents with cutaneous furuncles. Risk factors for furunculosis include bacterial carriage in the nostrils, diabetes mellitus, obesity, lymphoproliferative neoplasms, malnutrition, and use of immunosuppressive drugs.

The most common *complications* of boils are scarring and infection or abscess of the skin, spinal cord, brain, kidneys, or other organs. Infections may also spread to the bloodstream (bacteremia) and become life-threatening. *S. aureus* strains first infect the skin and its structures (for example, sebaceous glands, hair follicles) or invade damaged skin (cuts, abrasions). Sometimes the infections are relatively limited (such as a stye, boil, furuncle, or carbuncle), but other times they may spread to other skin areas (causing cellulitis, folliculitis, or impetigo). Unfortunately, these bacteria can reach the bloodstream (bacteremia) and end up in many different body sites, causing infections (wound infections, abscesses, osteomyelitis, endocarditis, pneumonia) that may severely harm or kill the infected person. *S. aureus* strains also produce enzymes and exotoxins that likely cause or increase the severity of certain diseases. Such diseases include food poisoning, septic shock, toxic shock syndrome, and scalded skin syndrome. Almost any organ system can be infected by *S. aureus*.

Patients with furuncles on the face (upper lips, eyelids, forehead) sometimes complain of severe headaches, and high temperature, which are signs of the furuncle complicated by suppurative thrombophlebitis of the facial veins. The

latter is associated with purulent meningitis as a result of the infection spreading to the veins of the cavernous sinus.

When such symptoms as remittent fever, intense chills, profuse sweating, delirium, confusion and skin pallor are encountered, it means the patient has developed sepsis, and the appearance of suppuration in other organs (metastatic abscess) confirms the diagnosis of septicemia (septicopyemia).

In the early stage of the process furuncles are *treated* conservatively, in the late stage they are to be operated. Antibacterial therapy is indicated in every stage of the disease. Patients are to be informed of the possibility of developing serious complications if they attempt to pick the lesions or cut them with a blade or even apply a hot compress. In the early stage of the process a 70% alcohol solution is used to clean the skin, 2% alcohol solution of salicylate, and ultraviolet radiation therapy and is used. After they have opened up, dressings are put on with proteolytic enzymes, hypertonic solutions of sodium chloride and ultraviolet irradiation is applied. When the core is evacuated, dressings with synthomycin ointments, or methyluracil are applied. When the furuncle is associated with such complications as lymphangitis and lymphadenitis, antibiotic therapy is indicated.

Patients with furuncles of the upper lip and above it have to be hospitalized as emergency in the surgical unit for general and topical therapy including antibiotic therapy. Furuncles of the maxillofacial area of the head are to be treated with antiplatelet drugs added to other methods due to complications they can cause (cavernous sinus thrombosis, purulent meningitis). The condition is treated by bed rest and a diet of mashed food.

The patient with recurrent furuncle at the same place has to be examined specially to exclude out any metabolic disorders (diabetes mellitus, vitamin deficiency). To boost the body's resistance to staphylococcal infections, immunization is done with staphylococcal anatoxin.



Fig. 1. Furuncle (A) and carbuncle (B)

A carbuncle is a cluster of boils, draining pus onto the skin. It is usually caused by bacterial infection, most commonly with *Staphylococcus aureus* or *Streptococcus pyogenes*, which can turn lethal. However, the presence of a

carbuncle is actually a sign that the immune system is working. The infection is contagious and may spread to other areas of the body, or other people; those living in the same residence may develop carbuncles at the same time. A carbuncle is made up of several skin boils. The infected mass is filled with fluid, pus and dead tissue. Fluid may drain out of the carbuncle, but sometimes the mass is so deep that it cannot drain on its own. Carbuncles may develop anywhere, but they are most common on the back and the nape of the neck. The carbuncle may be the size of a pea or as large as a golf ball. It may be red and irritated, and might hurt when touched. It may also grow very fast and have a white or yellow center. It may crust or spread to other skin areas. Sometimes other symptoms may occur, such as fatigue, fever and a general discomfort or sick feeling. Itching may occur before the carbuncle develops. The initial cause of a carbuncle can often not be determined. Triggers that make carbuncle infections more likely include rashes such as folliculitis; friction from clothing or shaving; having hair pulled out, such as sites where clothing or furniture grab at hairs; generally poor hygiene; poor nutrition; or weakening of immunity. Poor nutrition may be an important factor – for example, persons with diabetes and immune system diseases are more likely to develop infections (especially bacterial infections of the leg or foot).

Carbuncle has to be differentiated from Siberian ulcer carbuncle, which is characterized by the presence of hemorrhagic blisters, the absence of purulent discharge, painless infiltration, extreme tissue edema; the resulting necrotic tissue is black in colour and surrounded by tiny blisters with hemorrhagic contents. The blisters are found to contain the Siberian ulcer bacilli.

Treatment of a carbuncle at the initial stages is conservative. It involves rest for the affected organ. If it is on the face the patient should be put on bed rest. Patients are not allowed to talk, and are given liquid food. After treating the carbuncle with 70% ethyl spirit, aseptic dressing is applied, and ultraviolet radiation therapy is prescribed. Antibiotics are administered parenterally and long-acting sulphanilamides - orally. The treatment of carbuncles of the maxillofacial area is to be added with antiaggregant drugs. Uneffective conservative therapy for 2 - 3 days is an indication for surgical treatment, which is done under general anesthesia. A crosswise incision is made over the infiltration up to the fascia and the necrotic tissues are excised throughout, separating them from the fascia and skin, the whole area being cleaned of necrosis and pus. The postoperative period is managed according to the principles of treating infected wounds.

Hidradenitis is a common (though rarely diagnosed), chronic skin disease characterized by clusters of abscesses or subcutaneous boil-like "infections" (oftentimes free of actual bacteria) that most commonly affects apocrine sweat

gland bearing areas, such as the underarms, under the breasts, inner thighs, groin and buttocks. The disease is not contagious. There are indications that it is hereditary among certain ethnic groups and autoimmune in nature. Onset is most common in the late teens and early 20's. Hidradenitis outbreaks are painful in tender areas and may persist for years with interspersed periods of inflammation, often culminating in sudden drainage of pus. This process often forms open wounds that will not heal and frequently leads to significant scarring. For unknown reasons, people with hidradenitis develop plugging of their apocrine glands. Hidradenitis flares may be triggered by emotional stress, sweating, hormonal changes, heat and humidity, and the condition is exacerbated by friction from clothing. Persistent lesions may lead to the formation of sinus tracts, or tunnels connecting the abscesses or infections under the skin. At this stage, complete healing is usually not possible, and progression is variable, with some experiencing remission for months to years at a time, while others may worsen and require multiple surgeries. Bacterial infections and cellulitis (deep tissue inflammation) are other common complications of hidradenitis. Depression and physical pain are often seen with hidradenitis and can be difficult to manage.

Hydradenitis is treated with antibiotics, and long-acting sulfanilamides. Immunization with staphylococcal anatoxin is prescribed. Ultrasound and ultraviolet irradiation is used as a physical therapeutic measure. In the case of an abscess formation, surgery has to be performed — incision and drainage. If longterm treatment of hydradenitis proves unsuccessful and there is fear of ensuring sepsis, the whole subcutaneous fatty layer of the axillary fossa is excised such as with a carbuncle.

Lymphangitis is an inflammation or an infection of the lymphatic channels that occurs as a result of infection at a site distal to the channel.

The lymphatic system is a network of vessels (channels), nodes (glands), and organs. It is part of the immune system, which protects against and fights infections, inflammation, and cancers. The lymphatic system also participates in the transport of fluids, fats, proteins, and other substances throughout the body. The lymph nodes are small structures that filter the lymph fluid and contain many white blood cells to fight infections.

The most common cause of lymphangitis in humans is *Streptococcus pyogenes* (Group A strep), although it can also be caused by the fungus *Sporothrix schenckii*. This is characterized by certain inflammatory conditions of the skin caused by bacterial infections. Thin red lines may be observed running along the course of the lymphatic vessels in the affected area, accompanied by painful enlargement of the nearby lymph nodes. When the inferior limbs are affected, the

redness of the skin runs over the great saphenous vein location and confusion can be made with a thrombophlebitis. Chronic lymphangitis is a cutaneous condition that is the result of recurrent bouts of acute bacterial lymphangitis.

Signs and symptoms include a deep reddening of the skin, warmth, lymphadenitis, and a raised border around the affected area. The person may also have chills and a high fever along with moderate pain and swelling. A person with lymphangitis should be hospitalized and closely monitored by medical professionals.

Primarily treatment aims at eliminating the primary focus of infection and includes incision and drainage of abscess, phlegmon, pyoderma, and the rational drainage of purulent lesions. Antibiotic therapy is prescribed according to the type of microbes and their resistance to drug therapy. Rest is important, and the limb has to be elevated; for this purpose it can be immobilized.

Lymphadenitis is the inflammation of lymph nodes. It is often a complication of bacterial infections, although it can also be caused by viruses or other disease agents. Lymphadenitis may be either generalized, involving a number of lymph nodes, or limited to a few nodes in the area of a localized infection. Lymphadenitis is sometimes accompanied by lymphangitis, which is the inflammation of the lymphatic vessels that connect the lymph nodes. Lymphadenitis is marked by swollen lymph nodes that develop when the glands are overwhelmed by bacteria, virus, fungi, or other organisms. The nodes may be tender and hard or soft and "rubbery" if an abscess has formed. The skin over an inflamed node may be red and hot. The location of the affected nodes is usually associated with the site of an underlying infection, inflammation, or tumor. In most cases, the infectious organisms are Streptococci or Staphylococci. If the lymphatic vessels are also infected, in a condition referred to as lymphangitis, there will be red streaks extending from the wound in the direction of the lymph nodes, throbbing pain, and high fever and/or chills. The extensive network of lymphatic vessels throughout the body and their relation to the lymph nodes helps to explain why bacterial infection of the nodes can spread rapidly to or from other parts of the body. Lymphadenitis in children often occurs in the neck area because these lymph nodes are close to the ears and throat, which are frequent locations of bacterial infections in children. Lymphadenitis is also referred to as lymph node infection, lymph gland infection, or localized lymphadenopathy.

Streptococcal and staphylococcal bacteria are the most common causes of lymphadenitis, although viruses, protozoa, rickettsiae, fungi, and the tuberculosis bacillus can also infect the lymph nodes. Diseases or disorders that involve lymph nodes in specific areas of the body include rabbit fever (tularemia), cat-scratch

disease, lymphogranuloma venereum, chancroid, genital herpes, infected acne, dental abscesses, and bubonic plague. Lymphadenitis can also occur in conjunction with cellulitis, which is a deep, widespread tissue infection that develops from a cut or sore. In children, tonsillitis or bacterial sore throats are the most common causes of lymphadenitis in the neck area. Diseases that involve lymph nodes throughout the body include mononucleosis, cytomegalovirus infection, toxoplasmosis, and brucellosis.

The early symptoms of lymphadenitis are swelling of the nodes caused by a build-up of tissue fluid and an increased number of white blood cells resulting from the body's response to the infection. Further developments include fever with chills, loss of appetite, heavy perspiration, a rapid pulse, and general weakness.

Treatment of the initial stages of lymphadenitis is conservative. It involves rest of the affected organ, ultraviolet irradiation, active treatment of the primary focus of infection (timely incision and drainage of abscesses, phlegmon, proper drainage of purulent processes), and antibiotic therapy. Suppurative lymphadenitis is treated by surgery which includes incision of abscess, adenophlegmon, evacuation of pus and drainage. Postoperative therapy follows the methods of treatment for infected wounds.

Mastitis (also called mammitis) is the inflammation of breast tissue. *S. aureus* is the most common etiological organism responsible, but *S. epidermidis* and streptococci are occasionally isolated as well. Mastitis usually affects lactating women - women who are breastfeeding, producing milk. Hence, it is often referred to as lactation mastitis. The patient feels a hard, sore spot inside the breast. Mastitis can occur as a result of an infection or a blocked milk duct. According to studies, mastitis seems to affect approximately 10% of all breastfeeding mothers.

Classification of mastitis.

1. Edematous form.
2. Infiltrative form.
3. Suppurative-destructive forms:
 - a) breast abscess;
 - b) phlegmonous mastitis;
 - c) gangrenous mastitis.

The bacterial organisms invade the breast through cracks in the nipples, the exposed lymphatic ducts, or the milk ducts. Irregular nursing, which leads to

overfilling of the breasts, increases the effects of infections. The breasts become swollen, painful, reddened, hardened, and tender. The infection may be in one or both breasts; it can be localized or spread over an area. Purulent discharges may occur; frequently the discharge indicates abscess formation. Abscesses may remain internal or they may involve the skin. The lymphatic system's nodes and vessels are commonly enlarged and tender also. Acute mastitis accompanied by abscesses is often mistaken for acute inflammatory carcinoma (cancer) of the breasts. In a female child, after birth and during puberty, there may be brief episodes of breast inflammation; these are usually hormone induced and are not caused by bacterial infection.

Chronic mastitis is usually a secondary effect of systemic diseases such as tuberculosis, fungal infections, yeast infections, or syphilis. A relatively uncommon type of mastitis, called plasma cell mastitis, occurs most frequently in older women who have had a number of children and have a history of difficulty in nursing. It is sometimes difficult to distinguish from cancer of the breast. In this disease lymphatic fluids stagnate in the breast, and the stagnated fluids are treated by the body as foreign objects. Plasma cells, white blood cells, and fatty acid crystals accumulate, and fatty tissue suffers degeneration. A hard lump forms under part of the nipple; there may be distortion of the nipple because of the lesion. The nipple area is painful, tender, and inflamed and may exude a cloudy discharge. The milk ducts and lymph nodes are commonly thickened and enlarged. As the condition progresses, small areas of the breast become hardened as the original tissue is destroyed and replaced by fibrous or granular tissue. Injury to the breast tissue is sometimes followed by inflammation and necrosis (death) of the fatty tissue resulting in a hard fixed lump with no skin discoloration. If pyogenic microorganisms enter the congested breast, then after 2 - 4 days the breast becomes inflamed which sets the stage for the serous phase of mastitis sets. The condition has a sudden onset with a body temperature rise, sweating, weakness, fatigue and severe pain in the breast. The breast is found to be enlarged, tender on palpation, and the area of infiltration is not distinct. Milking is painful and does not bring any relief. Blood leucocytosis is up to $10 - 12 \times 10^9$, ESR up to 20 - 30 mm/hr. If treatment is delayed, the process can progress after 3 - 6 days to the infiltrative phase with pronounced clinical features of inflammation, and deterioration of the general condition. Body temperature may rise to 38 - 40°C. The palpated mass assumes a more distinct form. The inflammatory process in the breast changes the milk acidity by increasing the pH of milk, which is due to the increase of the activity of alkaline phosphatase. Microscopic analysis of the cellular contents of secretions from the breast shows large amounts of leucocytes. The transition of early forms of mastitis into purulent phase is characterized by an increase in intensity of both the

local and general symptoms of inflammation. Body temperature is constantly high or hectic. Infiltration in the breast increases, as does the skin hyperemia, fluctuation appears in one of the breast segments. An abscess can be localized either superficially or deeper with a spreading to the retromammary space. Patients with gangrenous mastitis are found to be critically sick. Body temperature is very high-up to 40 - 41 °C, pulse up to 120 - 130 beats per min, the breast is very swollen, skin is edematous with blisters containing hemorrhagic fluids and areas of necrosis. The edema spreads to the surrounding areas. High blood leucocytosis is noted with a shift to the left and toxic granular leucocytosis, protein is found in the urine.

Mastitis can be complicated by lymphangitis, lymphadenitis and rarely sepsis. After the abscess has burst, especially if it occurs spontaneously, breast fistula closing spontaneously long after it may appear.

Treatment. Treatment of the initial stages of mastitis is conservative, and that of the purulent forms is surgical. As soon as signs of breast congestion are noticed, the breasts should be supported in a raised position with either an immobilization bandage or brassiere that do not squeeze or press on the breasts but support them. Using a breast pump the breasts are evacuated of the milk; breast-feeding should be continued, fluid intake is limited, oxytocin and nospani are given. Antibiotics are used in case of serous and infiltrative mastitis (semisynthetic penicillins, aminoglycosides, cephalosporins and macrolides), sulfanilamides (in combination with antibiotics), infusion therapy, including plasma substitutes, hemodes, protein preparations, saline solutions. Substances that improve the body's resistance (gamma globulins, etc.) are also used. The breasts must be milked constantly to prevent congestion. All manipulations should be done after the breast has been emptied. In severe cases of mastitis it is recommended to suppress lactation by a combination of estrogen and androgen preparations.

Purulent mastitis is an indication for surgery, which is performed under general anesthesia; only in case of superficially located small abscesses surgery can be performed under local anesthesia with retromammal novocain block. Wide and fairly deep incisions are made on the breast; all the pus and necrotic tissues are evacuated. Intramammary lesions are opened through radial incisions. After the pus has been evacuated, the cavity is examined using a finger, opening at the same time the various lacunae, hydrogen peroxide solution is used to wash or irrigate the cavity and the latter is dried. Then under adequate lighting the cavity is examined visually with the wound edges held open by retractors, while pressing on the breast. If it is found that some pus is entering the wound from a deeper area, then that opening is widened up to join the main cavity. All necrotic tissue lying loose

in the abscess cavity is excised and removed. If there are several abscesses on the same breast, they are opened with separate incisions. Retromammal and deeply-seated intramammal abscesses are drained through semilunar incisions made through the lower inframammary fold. In this way the breast is separated from the pectoralis major muscle. Intramammal abscesses are drained from their back, the cavity is drained and the resulting wound is sutured leaving the drainage site with the tubes. This method of incision and drainage prevents damage to the intralobular milk ducts by providing a good drainage of pus and necrotic tissues and at the same time giving a good cosmetic result. In localized forms of acute mastitis and especially in cases of chronic mastitis, the focus of infection can be excised within healthy tissue and firm sutures are applied with a mall drain inserted for the instillation of antibiotics.

Treatment of the wound after incision and drainage is done taking into consideration the stage of the wound process. The use of secondary sutures cuts the healing time and improves the cosmetic results of the operation.

Suppurative hand infections

The hand is susceptible to infection by virtue of its intimate contact with the outside world, its great surface area and its propensity for injury. That is, the hand is exposed frequently to infectious organisms, and these organisms are frequently given a point of entry. The specialized anatomy of the hand, particularly the tendon sheaths and deep fascial spaces, create distinct pathways for infection to spread. In addition, even fully cleared infections of the hand can result in significant morbidity, including stiffness and weakness. For these reasons, early and aggressive treatment of hand infections is imperative.

In this section, specific hand infections will be considered:

- paronychia: infection of the folds of skin surrounding a fingernail;
- felon: a purulent collection on the palmar surface of the distal phalanx;
- flexor tenosynovitis: purulent material resides within the flexor tendon sheath;
- septic arthritis: infection in the joint space, often related to bite wounds.

Paronychia. The paronychium is a small band of epithelium that covers the medial and lateral borders of the nail. The eponychium is a small band of epithelium that covers the proximal aspect of the nail.

A paronychia is an infection of the paronychium or eponychium. It is caused by minor trauma such as nail biting, aggressive manicuring, hangnail picking or applying artificial nails. Immunodeficiency, poor glycemic control, and

occupations involving repeated hand exposure to water (e.g. dishwasher) are risk factors for the development of paronychia. Tenderness and erythema of the nail fold at the site of infection will become evident within a few days of the inciting trauma. Progression to abscess formation is common. Turkman et al described the "digital pressure test for paronychia": A paronychia will appear as a blanched area when light pressure is applied to the volar aspect of the affected digit.

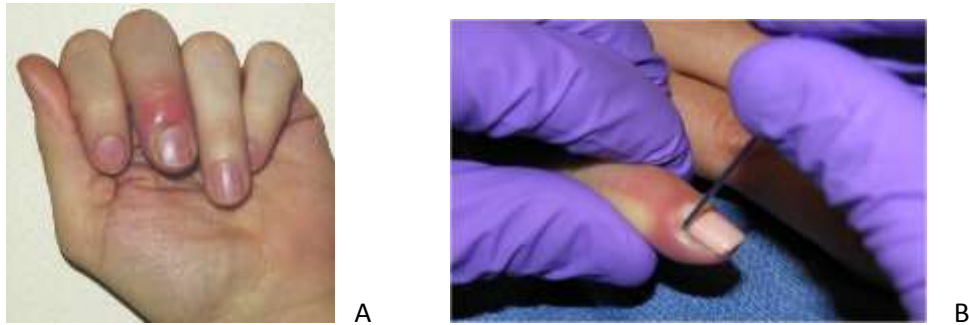


Fig. 2. (A) Paronychia; (B) The bevel of an 18 gauge needle is passed between the nail plate below and the nail fold above to allow for drainage of the pus.

Acute paronychia are usually caused by *Staphylococcus aureus* and are treated with a first-generation cephalosporin or anti-staphylococcal penicillin. Broader coverage is indicated if other pathogens are suspected. Chronic paronychia may be caused by *Candida albicans* or by exposure to irritants and allergens. Paronychia may be prevented by avoiding behaviors such as nail biting, finger sucking, and cuticle trimming. Patients with chronic paronychia should be advised to keep their nails short and to use gloves when exposed to known irritants.

Felon. A felon is an abscess on the palmar surface of the fingertip. Bacteria are normally introduced via minimal penetrating trauma, such as a splinter. The palmar aspect of the fingertip contains many osteocutaneous ligaments that connect the palmar skin of the fingertip to the distal phalanx. These ligaments prevent excessive mobility of the skin during pinch; they also maintain position of the cutaneous sensory endings and receptors to allow for identification of objects during grasp. The organization of these osteocutaneous ligaments form a relatively non-compliant compartment in the distal phalanx; thus, rather than expanding when pus is introduced, the compartment will simply increase in pressure. Elevated compartment pressure results in significant pain relative to the (small) amount of pus. In addition, the gradient between capillary pressure and tissue pressure is decreased; the resulting decrease in perfusion can lead to tissue necrosis. Furthermore, because the osteocutaneous ligaments attach to the distal phalanx itself, osteomyelitis (infection of the bone) can occur.

Treatment involves surgical drainage and antibiotics. Incision and drainage is performed at the most fluctuant point. The incision should not cross the distal interphalangeal joint flexion crease (to prevent formation of a flexion contracture from scar formation) or penetrate too deeply (to prevent spread of infection from violating the flexor tendon sheath). Potential complications of excessive dissection to drain a felon include an anesthetic fingertip or unstable finger pad. Antibiotic treatment should cover staphylococcal and streptococcal organisms. X-rays may be helpful to ensure that there is no retained foreign body.

Flexor tenosynovitis. In the fingers, a series of pulleys hold the tendons in close apposition to the bone, preventing bowstringing during flexion. There are a total of 8 pulleys overlying the finger flexor tendons and 3 pulleys overlying the thumb flexor tendon; these pulleys together are called the flexor tendon sheath. In flexor tenosynovitis, the infection is within the flexor tendon sheath. This infection is particularly harmful because bacterial exotoxins can destroy the paratenon (fatty tissue within the tendon sheath) and in turn damage the gliding surface of the tendon. In addition, inflammation can lead to adhesions and scarring, and infection can lead to overt necrosis of the tendon or the sheath. Although patients may not recall a specific history of trauma, flexor tenosynovitis is usually the product of penetrating trauma. Flexor tenosynovitis may be caused by inoculation and introduction of native skin flora (eg, *Staphylococcus* and *Streptococcus*) or by more unusual organisms (e.g., *Pasteurella* and *Eikenella*) when there is a bite wound. Flexor tenosynovitis can also have noninfectious causes such as chronic inflammation from diabetes mellitus, rheumatoid arthritis or other rheumatic conditions (eg, psoriatic arthritis, systemic lupus erythematosus, and sarcoidosis).

Kanavel described four classic signs of flexor tenosynovitis, as follows:

- Flexed posture of the digit.
- Fusiform (sausage-shaped, or tapering) swelling.
- Tenderness to palpation over the flexor tendon sheath.
- Pain over the flexor tendon sheath with passive extension of the finger.

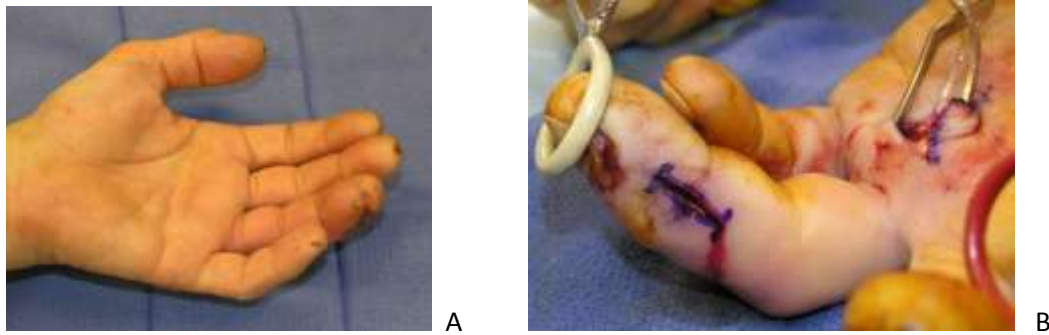


Fig. 3. (A) This patient's fourth digit exhibits erythema, fusiform swelling, and mild flexion compared to the adjacent digits. (B) Proximal and distal incisions have been made, allowing adequate drainage of the flexor tendon sheath.

Post-operative active and passive ROM exercises are recommended. Intravenous antibiotics should continue for an additional two or three days. (The duration of IV antibiotic administration as well as the need for oral antibiotics thereafter is determined by the intraoperative cultures and clinical response.) Post-operative adhesions damage gliding surfaces and decrease active range of motion, and thus require tenolysis. Soft tissue necrosis and flexor tendon rupture are other relatively common complications.

Joint infection. The metacarpophalangeal and interphalangeal joints are closed, relatively avascular spaces. Infection can reach the joint space via direct penetration or hematogenous spread. Small (and ring) finger metacarpophalangeal joint infections in particular may result from a "fight bite," where the patient strikes and an opponent in the mouth with a closed fist and the opponent's tooth penetrates the joint and seeds it with oral flora. As with flexor tenosynovitis, a major risk of joint space infection is destruction of the gliding surface by bacterial exotoxins, which can compromise recovery of motion after the infection resolves.

A fight bite is at particularly high risk for complications, for the following reasons:

- the puncher may underestimate the severity of the wound
- the puncher may have been intoxicated (and sufficiently "medicated" to not feel pain)
- the puncher may attribute initial symptoms to bone pain from punch and not present for care until cellulitis is rampant
- the initial examiner may underestimate the severity of the wound, as it is usually small (the size of an incisor tooth or smaller, eg 3mm) with clean edges
- the human mouth has a high concentration of nearly 200 species of bacteria, many "unusual" anaerobes

- motion of the MCP joint to "shake off the pain" may drive saliva deeper into the tissue
- the extensor tendon and joint capsule are fairly superficial and may be violated with seemingly shallow wounds
- the extensor tendon and joint capsule are fairly avascular and thus unable to fight infection

Fight bites should be meticulously irrigated, preferably with a formal debridement by a hand surgeon in the operating room. The laceration must not be closed in the ED.

Diagnosis of an established joint infection is often made by clinical examination. Patients will have swelling and erythema centered on the affected joint. Motion or axial loading of the joint will increase pain. Assessment of joint fluid for cell count, gram stain, and crystals (acute crystalline arthropathy such as gout can mimic a joint infection) can aid in the diagnosis, but it is often quite difficult to pass a needle into the narrow joint space and obtain an adequate sample. Serum markers of inflammation (such as white blood cell count, erythrocyte sedimentation rate, and C - reactive protein) are not typically elevated with an infection of a small joint of the hand. X-rays should be obtained to ensure that there is no fracture or retained tooth fragment.

Treatment consists of incision and drainage of the joint space. For the metacarpophalangeal joints of the fingers, the approach is normally dorsal through the long extensor tendon. In "fight bite" situations, there may be an indentation of the head of the metacarpal where it struck the tooth. For the interphalangeal joint, the approach is normally dorsolateral between the extensor mechanism dorsally and the collateral ligament laterally. Arthroscopic approaches have been described for the wrist and even the metacarpophalangeal joint, but an open approach is more commonly used.

General Principles:

- Open wounds must be irrigated to remove debris.
- Closed abscesses must be incised and drained
- Devitalized tissue should be debrided.
- Penetrating wounds require consideration of tetanus status.
- Splinting the hand may enhance healing.
- Patients with diabetes mellitus have more gram-negative infections and require broader antibiotic coverage.
- Patients in an immunocompromised state may develop a hand infection from hematogenous spread from another site.

- Unusual exposures lead to unusual bacteria: e.g., tropical fish aquarium workers, butchers, farmers.

Pandactilitis. This is a suppurative inflammation of all the finger tissues. Pandactilitis is a serious infection associated with severe intoxication (headache, elevated body temperature), regional lymphangitis, cubital and axillary lymphadenitis. Peripheral blood picture shows the changes typical for acute suppurative inflammation. Pandactilitis develops gradually. It is caused by the virulent infectious contamination through a hand injury. It can also result from a simple panaritium, especially the subcutaneous one. As pandactilitis develops, pain gradually increases in intensity, and the sick person experiences an intense, excruciating and distending pain. The edematous finger is of a bluish-violet colour. The infection can be either of a dry or wet necrosis. The discharge from the suppurative fistula or the postoperative wound is scanty, granulation is grey and dead. Palpation is tender all over the involved area and an attempt to move the finger may cause extreme pain. The patient's condition deteriorates, the body temperature increases, oedema and hyperemia of tissues increase and extend in the proximal direction. It is only an immediate surgical intervention that can stop the progression of the suppurative inflammatory process.

Hand phlegmons are infections that are localized to a hand lodge but can also be diffused. Often they are caused by a complicated panaritium, but they can also result from trauma to the hand or direct inoculation. Because of the gravely evolution the general manifestations of disease predominate over the local manifestations.

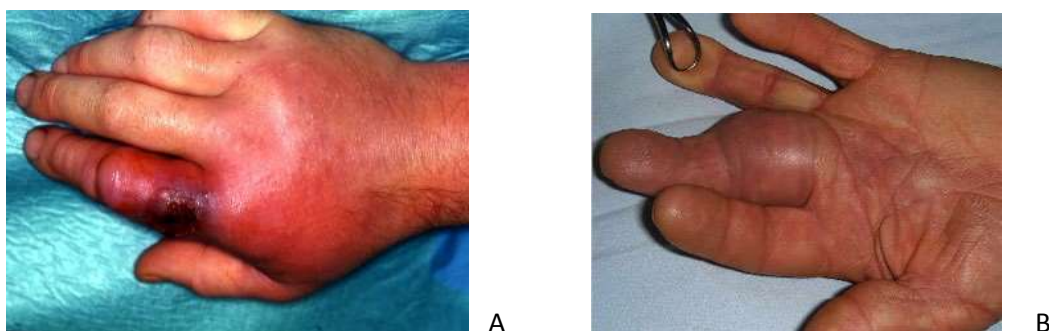


Fig. 4. Phlegmon of the hand (A, B)

Phlegmon of the ball of thumb (thenar eminence). This is accompanied by an extreme oedema of the thenar and the radial end of the dorsum of the hand. Severe pain on palpation, tense tissues, limitations in the mobility of the oedematous thenar tissues, smoothening of the palmar folds are all characteristic symptoms of thenar phlegmon. The purulent exudates can sometimes spread along the first interosteal muscles on the dorsum. In other cases the connective tissue

barriers separating divide the thenar from the mid palmar space disintegrate as a result of the suppurative process causing phlegmon of the mid palmar cavity.

Phlegmon of the hypothenar is often accompanied by mild intoxication. Minimal oedema, hyperemia and tension in the tissues, tenderness on palpation over the hypothenar and an increase in pain upon moving the little finger are the characteristic features.

Commissural phlegmon is localized at the distal part of the palm. The entry points of infection are usually deep skin fissures and skin callosity over the area of the 2nd-4th carpophalangeal joints of the palm. This phlegmon is also known as corn abscess. Phlegmons are associated with extreme pain, oedema on both sides of the hand. Fingers adjacent to the area of infection are somewhat spread apart and bent in their interphalangeal joints; extension is painful as a result of the tension on the inflamed palmar aponeurosis. It is possible for the pus to spread directly through the oval fissure of the aponeurosis to the dorsal surface of the hand, involving the tendons of the deep flexors, which are situated very closely. The spread of infection can also occur in the proximal direction along the canals of the vermiform muscles, involving the mid palmar space in the inflammation process.

Phlegmons of the mid palmar space are associated with the accumulation of pus between the palmar aponeurosis and the thin fascial sheath covering tendons of the flexor digitorum or between the fascia covering the palmar sides of the interosseal muscles and the posterior surface of the tendon of the long flexor digitorum. The disease is accompanied by pronounced intoxication, high body temperature, headache, as well as changes in the peripheral blood picture.

Examination of the hand reveals a swelling in the central palmar area, the skin is tense, skin folds are smooth and fluctuation is not possible to elicit. Palpation over the inflamed region may cause extreme pain. The dorsum of the hand is very edematous. The 2nd-5th fingers are slightly bent in the interphalangeal joints, attempts of passive or active movement cause extreme stretching of the inflamed palmar aponeurosis, which results in pain increase. Delayed or irrational treatment of mid palmar space phlegmon may cause a complication with the abscess bursting into the thenar fissure and the pus spreading along the canals of the vermiform muscles of the dorsum.

Crossed or U-form phlegmon is the most severe form of suppurative hand inflammation. The disease results from purulent tendovaginitis of the 1st or 5th finger with a spread of the purulent exudate to radial and ulna synovial sacs. U - form phlegmon is accompanied by pronounced intoxication, high body

temperature, headache, as well as body weakness. The hand is oedematous, bluish-violet in colour and palpation is extremely tender. The fingers are bent towards the palm, active movement is absent. Attempts of passive movement may cause extreme pain. By means of palpation the area of extreme tenderness is found to be around the projections of the 1st and 5th tendons and in the proximal parts of the hand, that is, at the site of the blunt ends of the radius and ulna synovial sacs. When the pus bursts into Pirogoff's space, diffuse tenderness and oedema of the distal parts of the forearm may result. The suppurative inflammatory process can spread to the mid palmar space, fissures of the thenar or hypothenar in the case of tenobursitis of the 1st and 5th fingers. Subsequently the pus spreads through the canals of the vermiform muscles to the dorsum of the hand to form an extensive purulent-necrotic area. Even with favourable resolution of U-type phlegmon the distant postoperative period is associated with limitations in the functions of the hand. Hence, it is very important to have a timely diagnosis and surgical treatment of this condition.

In subcutaneous phlegmon of the dorsum of the hand tissue edema and hyperemia are of diffuse nature, and it is difficult to determine the demarcation line of the infection. Thorough palpation can help to assess the area of tissue softening.

Subaponeurotic phlegmon of the dorsum of the hand occurs as a result of infection penetrating deep under the aponeurosis in case of stab wounds. In this kind of phlegmon a firm induration is observed which is associated with edema and hyperemia of the dorsum. Subcutaneous phlegmon also occurs, as a rule, secondary to damage of the skin covering of the dorsum of the hand. In suppurative affection of the palm there can be transmission of the infection to the dorsum through the lymphatic vessels or through the canals of the vermiform muscles. In such situations edema of the dorsum that is normally present in palmar lesions is accompanied by skin hyperemia, and the appearance of a diffusely tender area on palpation.

Treatment. Performing operations in case of palmar phlegmon one has to consider the fact that there is always oedema at the dorsum. Even in the presence of pronounced oedema at the dorsum one should never make an incision over this area before the presence of suppuration in the fingers and palm has been confirmed. However, if after an incision and drainage of a palmar or finger abscess the dorsum continues to swell, becomes more hyperemic and firmer, then there is a good reason to think that the contralateral edema has become suppurative.

Osteomyelitis is infection and inflammation of the bone or bone marrow. It can be usefully subclassified on the basis of the causative organism (pyogenic bacteria or mycobacteria) and the route, duration and anatomic location of the

infection. The definition of osteomyelitis is broad, and encompasses a wide variety of conditions. Traditionally, the length of time the infection has been present and whether there is suppuration (pus formation) or sclerosis (increased density of bone) is used to arbitrarily classify osteomyelitis. Chronic osteomyelitis is often defined as osteomyelitis that has been present for more than one month. In reality, there are no distinct subtypes, instead there is a spectrum of pathologic features that reflect balance between the type and severity of the cause of the inflammation, the immune system and local and systemic predisposing factors.

Classification

1. According to their etiological factors:
 - 1) non-specific osteomyelitis;
 - 2) specific osteomyelitis.
2. According to the mode of infection (transmission):
 - 1) hematogenic;
 - 2) non-hematogenic:
 - a) traumatic;
 - b) gunshot;
 - c) contact.
3. According to the clinical manifestation:
 - 1) hematogenic:
 - a) acute (toxic form, septicopyemia, localized form);
 - b) primary chronic;
 - c) secondary chronic;
 - 2) non-hematogenic:
 - a) acute;
 - b) chronic.

Osteomyelitis can also be typed according to the area of the skeleton in which it is present. For example, osteomyelitis of the jaws is different in several respects from osteomyelitis present in a long bone. Vertebral osteomyelitis is another possible presentation. Bone infections may occur at any age. Certain conditions increase the risk of developing such an infection, including sickle cell anemia,

injury, the presence of a foreign body (such as a bullet or a screw placed to hold together a broken bone), intravenous drug use (such as heroin), diabetes, kidney dialysis, surgical procedures to bony areas, untreated infections of tissue near a bone (for example, extreme cases of untreated sinus infections have led to osteomyelitis of the bones of the skull).

Two types of osteomyelitis are identified depending on the mode of penetration of the infection: hematogenic when an endogenous suppurative infection gains access to bone via the bloodstream, and non-hematogenic when the infection reaches the bone from the outside (exogenic). Non-haematogenic osteomyelitis occurs after open fractures, gunshots, internal fixation of bone (osteosynthesis) during healing of a fracture, in orthopedic operations, from the transfer of infection from the tissues surrounding the bone.

Acute hematogenic osteomyelitis. There are two main ways that infecting bacteria find their way to bone, resulting in the development of osteomyelitis. These include:

1. Spread via the bloodstream; 95% of these types of infections are due to *Staphylococcus aureus*. In this situation, the bacteria travels through the bloodstream to reach the bone. In children, the most likely site of infection is within one of the long bones, particularly the thigh bone (femur), one of the bones of the lower leg (tibia), or the bone of the upper arm (humerus). This is because in children these bones have particularly extensive blood circulation, making them more susceptible to invasion by bacteria. Different patterns of blood circulation in adults make the long bones less well-served by the circulatory system. These bones are therefore unlikely to develop osteomyelitis in adult patients. Instead, the bones of the spine (vertebrae) receive a lot of blood flow. Therefore, osteomyelitis in adults is most likely to affect a vertebra. Drug addicts may have osteomyelitis in the pubic bone or clavicle.

2. Spread from adjacent infected soft tissue; about 50% of all such cases are infected by *Staphylococcus aureus*. This often occurs in cases where recent surgery or injury has result in a soft tissue infection. The bacteria can then spread to nearby bone, resulting in osteomyelitis. Patients with diabetes are particularly susceptible to this source of osteomyelitis. The diabetes interferes with both nerve sensation and good blood flow to the feet. Diabetic patients are therefore prone to developing poorly healing wounds to their feet, which can then spread to bone, causing osteomyelitis.

There are three forms of acute hematogenic osteomyelitis: toxic form, septicopyemia, localized form.

Toxic form, or fulminant, is characterized by the development of extreme septic intoxication that starts in the early stage of the disease. The disease progresses rapidly and death ensues within the first few days. In this form the local pathological signs in the bone and its surrounding tissues fail to develop.

Septicopyemic form is characterized by the development of several suppurative-destructive foci in several bones simultaneously from the very onset of disease. Not often abscesses are observed in several parenchymatous organs such as the lungs, liver, and kidneys. All this adversely affects the disease process leading: to death in most instances. Bacteremia which is often encountered in this case contributes to the development of new osteomyelitis foci.

Localized form of acute hematogenic osteomyelitis manifests itself as a mild form of infection compared to the two previous ones: symptoms of suppurative intoxication are mild and often associated with a single suppurative-destructive focus in the bone. Local signs of infection are more common than the general signs and intoxication. This type of osteomyelitis often develops into a chronic form.

Most patients with acute hematogenic osteomyelitis present with a history of bone pain for several days. The hallmark of acute hematogenic osteomyelitis pain is its constant nature, with the level of pain increasing gradually. In young children, it is often difficult to elicit pain location, while in older children it is typically more localized. Pain generally leads to restricted use of the involved limb. As the sites most often involved are the long bones of the lower limbs, patients frequently present with a limp. In all cases, localized bone pain and fever should raise the clinical suspicion of acute hematogenic osteomyelitis. Exaggerated immobility of the joint and lack of point tenderness over the metaphysis suggest septic arthritis rather than (or in addition to) osteomyelitis. The classic signs of inflammation (redness, warmth and swelling) do not appear unless the infection has progressed through the metaphyseal cortex into the subperiosteal space. Such progression is more common in infants and young children who have a thinner bone cortex.

Elevated erythrocyte sedimentation rate (ESR), elevated C-reactive protein (CRP) and leukocytosis are often seen in acute hematogenic osteomyelitis, with an elevated CRP being the most sensitive laboratory parameter. However, this is also dependent on the offending agent.

Since physical examination and laboratory tests are suggestive rather than definitive, various imaging techniques have been used to facilitate the diagnosis of osteomyelitis. These include plain radiographs, skeletal scintigraphy, computed tomography (CT) and magnetic resonance imaging (MRI).

Plain radiographs are essential for excluding other diagnoses such as fracture. Although deep soft tissue swelling can be seen radiographically within the first few days of onset, osteopenia or osteolytic lesions from destruction of bone are usually not visible until 2 – 3 weeks after symptom onset. For this reason, the diagnostic utility of plain radiographs for diagnosing osteomyelitis is limited to those patients with prolonged symptoms and, as noted previously, this is generally not the case in patients. As a result, other imaging techniques are more commonly used to diagnose pediatric acute hematogenic osteomyelitis. Skeletal scintigraphy using technetium-99m diphosphonate allows for a whole-body survey, which is useful for those patients with poorly localized symptoms or if multifocal osteomyelitis is a concern, and this has a reported sensitivity of more than 90%. However, it necessitates exposure to ionizing radiation, and sensitivity is lower in neonates, making skeletal scintigraphy less useful in this age group. Differentiation of osteomyelitis from infarction associated with sickle cell disease and other disorders unrelated to infection, including neoplasms and fracture, can also be difficult. In addition, skeletal scintigraphy may have limited utility in diagnosing community-acquired *S. aureus* osteomyelitis. A recent study found that skeletal scintigraphy diagnosed osteomyelitis in only 53% of patients with community-acquired *S. aureus* osteomyelitis.

One benefit of CT scanning is that it provides specific anatomic information about the status of infection. CT can detect sequestra (indicative of chronic osteomyelitis) and intra-osseous gas, and can define subperiosteal abscesses, all of which are important considerations when designing the overall therapeutic approach. However, CT also requires exposure to radiation. There are studies suggesting that fluorodeoxyglucose positron emission tomography (FDG-PET) may be a useful alternative for diagnosis in adults, particularly when combined with CT. However, this issue has not been adequately examined in the specific context of the pediatric patient. MRI can be used to identify intra-osseous, subperiosteal and soft-tissue abscesses, thus enabling early abscess drainage without exposure to radiation. Edema and exudates within the medullary space are common findings of acute osteomyelitis and can be visualized by MRI. However, these findings can also be seen in other conditions such as fracture and infarct. Myositis is also readily identified by MRI. MRI yields better anatomic information, providing an anatomic atlas for orthopedists should surgery be indicated. For these reasons, MRI is becoming the imaging modality of choice for acute hematogenic osteomyelitis in patients. The sensitivity and specificity of MRI for the diagnosis of osteomyelitis range between 82 – 100% and 75 – 96%, respectively. Cost, availability and the need for sedation are important limitations to the use of MRI.

While patient presentation, laboratory testing and diagnostic imaging are all important, none are definitive with respect to a diagnosis of acute hematogenic osteomyelitis and, more importantly, none provide information about the antibiotic-resistance status of the offending organism. For this reason, isolation of the causative organism remains the diagnostic gold standard and is currently the only way to establish a definitive microbiologic diagnosis. In a significant number of cases, it is not possible to establish a definitive bacterial etiology, either because the offending organisms are difficult to cultivate or because empiric antimicrobial therapy has compromised microbiologic analysis. When the etiologic agent can be identified, empiric antimicrobial therapy should be adjusted based on the specific susceptibility profile of the offending bacterial strain. Cultured samples should include bone samples, which have a higher diagnostic yield in comparison with blood cultures. However, blood cultures should also be obtained, as an organism is recovered in approximately 50% of all acute hematogenic osteomyelitis infections. Needle aspiration of the affected bone can be performed using relatively noninvasive procedures in neonates and young children, while older children and adolescents often require more invasive surgical techniques such as drilling or cutting into the bone. Fungal and mycobacterial stains and cultures should also be obtained, particularly in cases with specific risk factors and in culture-negative cases of acute hematogenic osteomyelitis that are unresponsive to empiric therapy. Tissue samples should also be sent for histological examination to confirm the diagnosis of osteomyelitis. Acquiring cultures early in the course of acute hematogenic osteomyelitis is helpful because prolonged empiric antimicrobial therapy decreases the chance of recovering the causative agent. Indeed in a stable patient, if a plan is in place to rapidly obtain tissue cultures, it is our opinion that empiric antibiotic therapy should be delayed until tissue cultures are obtained.

Treatment. The treatment of acute hematogenic osteomyelitis demands appropriate antimicrobial therapy in all cases and may require surgical incision and drainage. For the reasons discussed earlier, appropriate drainage has become particularly important in recent years owing to the continued emergence of community-associated methicillin-resistant *S. aureus* (CA-MRSA). Incision and drainage should be performed whenever an abscess (intra-osseous, subperiosteal and/or soft-tissue) exists. Surgical removal of devitalized bone and debridement of affected soft tissues should be undertaken. It has been our experience that multiple incision and drainage procedures are often necessary in patients and adolescents with CA-MRSA osteomyelitis, even with appropriate antibiotic therapy. Surgical drainage should also be considered when a patient does not respond to empiric antibiotic therapy. In that case, surgical intervention may enhance treatment. In addition, surgical intervention allows for the collection of tissue which can be

microbiologically evaluated for unusual etiologies of osteomyelitis, and histologically examined to confirm the diagnosis.

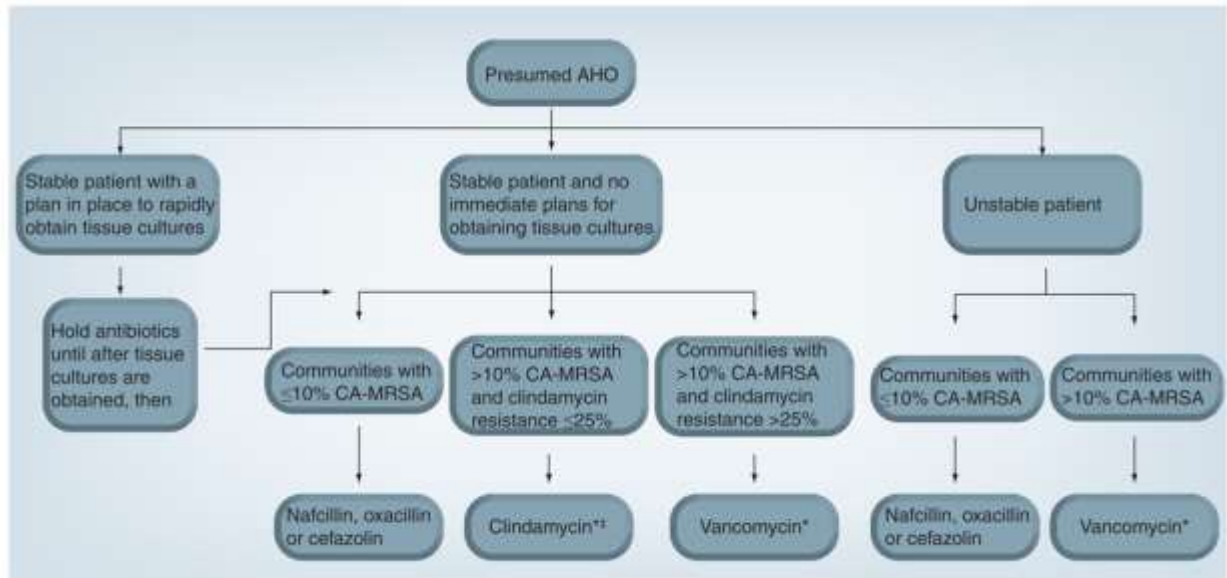


Fig. 5. Algorithm for the suggested initial antibiotic therapy of acute hematogenous osteomyelitis in children 3 months of age or older. If cultures yield an organism, antimicrobial treatment should be adjusted immediately (as needed). *If *Kingella kingae* is of particular concern, add therapy with cefazolin. ‡If clindamycin is considered for treatment, the inducible macrolide, lincosamide and streptogramin B resistance phenotype must be excluded by the D-test, as this phenotype is associated with treatment failure. AHO: Acute hematogenous osteomyelitis; CA-MRSA: Community-acquired methicillin-resistant *Staphylococcus aureus*.

Immunotherapy includes treatment by preparations for passive immunization (hyperimmune antistaphylococcal plasma, staphylococcal immunoglobulin), which is combined with staphylococcal anatoxin, bacteriophages, nonspecific immune therapy (prodigiosan, lizosim, methyluracyl etc.) as well as desensitization preparations.

Chronic osteomyelitis is a severe, persistent, and sometimes incapacitating infection of bone and bone marrow. It is often a recurring condition because it is difficult to treat definitively. This disease may result from the following:

- Inadequate treatment of acute osteomyelitis
- Hematogenous type of osteomyelitis
- Trauma
- Iatrogenic causes such as joint replacements and the internal fixation of fractures

- Compound fractures
- Infection with organisms, such as *Mb. tuberculosis* and *Tr. species* (syphilis)
- Contiguous spread from soft tissues, as may occur with diabetic ulcers or ulcers associated with peripheral vascular disease

Cierny and Mader proposed an anatomic classification of chronic osteomyelitis:

Type 1 — Endosteal or medullary lesion

Type 2 — Superficial osteomyelitis limited to the surface

Type 3 — Localized, well-marked lesion with sequestration and cavity formation

Type 4 — Diffuse osteomyelitis lesions

Chronic osteomyelitis is characterized by the following main triad of signs:

- 1) *relapsing trend*,
- 2) *the formation of sequestra (or osteomyelitis cavity)*,
- 3) *purulent fistula*.

Relapse of chronic osteomyelitis presents complaints of general malaise, weakness, headache, rise in body temperature, sweating, chills. There is pain in the limb, and a purulent fistula opens up. In some cases the skin over the focus of osteomyelitis becomes hyperemic, there is intensive pain and induration of the soft tissue occur followed by the fluctuation sign. The old fistula that has closed earlier opens up again, or a spontaneous opening of the phlegmon at a new site occurs. After the pus has been evacuated the patient's condition improves, intoxication reduces, temperature falls to subfebrile, local inflammation is gradually eliminated, the purulent fistula continues to function or also gradually closes up. The process enters into the remission phase, which can change into relapse at any time.

The clinical presentations of different kinds of chronic osteomyelitis are principally identical - there is an interchange of disease phases. In posttraumatic cases (including gunshot osteomyelitis), however, the area of bone infection is normally limited to the fracture site from which the purulent fistulas emanate. Chronic haematogenic osteomyelitis is characterized by the presence of extensive areas of osteomyelitis along the metaepiphysis and diaphysis with the purulent fistulas, sometimes several of them, situated at different sites. Accordingly, the patient shows pronounced signs of chronic suppurative intoxication, changes in the blood picture (leucocytosis, increased ESR, disproteinemia), renal disorders, etc.

The general symptoms of a relapse of chronic osteomyelitis are similar to those of any suppurative surgical disease, hence the body temperature is checked

as well as the necessary blood tests and urinalysis are made. In the presence of local signs, the extent of skin hyperemia, soft tissue induration and the presence of fluctuation signs are taken note of. It is important to assess the functional status of the fistula; a blunt probe is used to do the assessment, which in certain cases helps to establish the location of the focus of osteomyelitis. When an ulcer is found in the area of a long - standing purulent fistula, the edges and surface of the wound have to be thoroughly examined and if malignancy is suspected a biopsy is performed. To establish the extent of spread of the inflammatory process to the adjacent joint, the extent of movement, the presence of tenderness or effusion in it are assessed. X-ray methods help find out bone changes in acute osteomyelitis (periostitis or subperiosteal abscess) or chronic osteomyelitis (osteosclerosis, bone sequestrum or sequestrum box). Mild pathology may be detected by means of CT scan. Radiography is one of the most important diagnostic tools whereby the presence of sequestra, cavities, chronic periostitis is established and the extent of damage to the bone is determined. Fistulography is a very valuable method of investigation, it gives evidence of the direction of the fistula tract, its connection with the bone cavity, which is necessary in planning surgery, notably to determine the surgical approach.

The following types of primary chronic haematogenous osteomyelitis are distinguished:

1. *Brodie's abscess*, or intraosseous abscess, i.e. a circumscribed necrosis of the bone sponge with its subsequent lysis and cavity formation.
2. *Garre's disease*, or severe sclerosing osteitis with areas of rarefaction in the bone and a spindle-shaped thickening of bone diaphyses.
3. *Ollier's osteomyelitis*, or albuminous osteomyelitis, i.e. slow accumulation of serous fluid rich in protein in the bone rather than that of pus, which is occasionally followed by sequestration.

Treatment. Surgical treatment for chronic osteomyelitis is indicated when there are sequestra, purulent fistula, osteomyelitic cavity in the bone, osteomyelitic ulcers, malignancy, in pseudoarthrosis, in cases of frequent relapse with severe pain, intoxication and dysfunction of the locomotive system as well as in case of functional and morphological changes in the internal organs caused by the chronic suppurative infection.

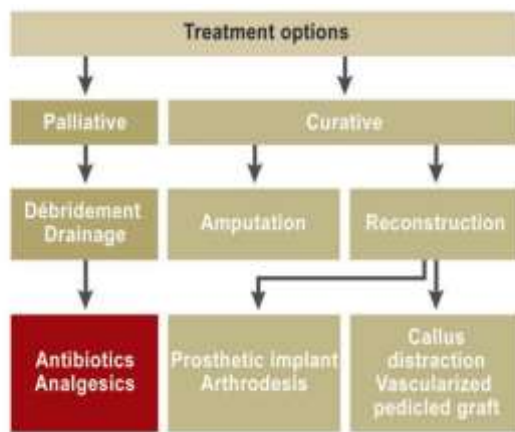


Fig. 6. Treatment options for chronic osteomyelitis.

The main component in the complex treatment of chronic osteomyelitis is radical surgery - necrectomy, which is often referred to as sequestrectomy. The aim of the operation is elimination of the chronic focus of infection in the bone and its surrounding soft tissues. In radical necrectomy the sequestrum is removed, all osteomyelitic cavities are incised and liquidated together with their internal wall granulations and detritus; ail purulent fistulas are excised. The next important step in the radical surgery is the sanitation and plasty of the bone cavity. More recently the plasty of bone cavity is achieved by using muscle pedicle flaps, bone plates (using autogenous or conserved bone tissue), chondroplasty (using conserved cartilage), and rarely, cutaneous flaps are used. Different biopolymer materials are used: collagen sponge impregnated with antibiotics, glue compositions with different ingredients and biopolymer plombs containing antiseptics. All these materials contain substances that enhance or activate bone tissue regeneration as well. Sanitation of the bone cavity after necrectomy is done by long - term methods of washing and drainage as well as vacuum drainage.

Acute suppurative arthritis is the purulent invasion of a joint by an infectious agent which produces arthritis. People with artificial joints are more at risk than the general population but have slightly different symptoms, are infected with different organisms and require different treatment. Septic arthritis is considered a medical emergency. If untreated, it may destroy the joint in a period of days. The infection may also spread to other parts of the body.

The term "suppurative arthritis" is a near synonym for septic arthritis. ("Suppurative" refers to the production of pus, without necessarily implying sepsis.) Reactive arthritis refers to arthritis caused by an immune consequence of an infection, but not directly attributable to the infection itself. Septic arthritis is usually caused by bacteria, but may be caused by viral, mycobacterial, and fungal pathogens as well. A broader term is "infectious arthritis", which describes arthritis

caused by any infectious organism. Viruses can cause arthritis, but it can be hard to determine if the arthritis is directly due to the virus or if the arthritis is reactive. Septic/suppurative arthritis and "bacterial arthritis" are sometimes considered equivalent, but there are exceptions. For example, *Borrelia burgdorferi* can cause infectious arthritis, but is not associated with suppurative arthritis.

Bacteria are carried by the bloodstream from an infectious focus elsewhere, introduced by a skin lesion that penetrates the joint, or by extension from adjacent tissue. Micro-organisms must reach the synovial membrane of a joint. This can happen in any of the following ways:

- dissemination of pathogens via the blood, from abscesses or wound infections, or from an unknown focus
- dissemination from an acute osteomyelitic focus,
- dissemination from adjacent soft tissue infection,
- entry via penetrating trauma
- entry via iatrogenic means.

Bacteria that are commonly found to cause septic arthritis are:

- *Staphylococcus aureus* - the most common cause in adults;
- *Streptococci* - the second most common cause;
- *Haemophilus influenzae* - was the most common cause in children but is now uncommon in areas where *Haemophilus* vaccination is practiced;
- *Neisseria gonorrhoea* - the most common cause of septic arthritis in young, sexually active adults. Multiple macules or vesicles seen over the trunk are a pathognomonic feature;
- *Escherichia coli* - in the elderly, IV drug users and the seriously ill;
- *M. tuberculosis*, *Salmonella* spp. and *Brucella* spp. - cause septic spinal arthritis.

In bacterial infection, *Pseudomonas aeruginosa* has been found to infect joints, especially in children who have sustained a puncture wound. This bacterium also causes endocarditis.

Septic arthritis can cause pain with any movement of the affected joint. Therefore, those affected by septic arthritis will often refuse to use the extremity and prefer to hold joint rigidly. Other common signs and symptoms are joint swelling, redness, and warmth. Septic arthritis should be considered whenever one is assessing a person with rapid onset of joint pain. Usually only one joint is affected however in seeding arthritis, several joints can be affected at the same time; this is especially the case when the infection is caused by *staphylococcus* or *gonococcus* bacteria. The diagnosis of septic arthritis is based on clinical

assessment and should prompt arthrocentesis. The diagnosis of septic arthritis can be difficult as no test is able to completely rule out the possibility. A number of factors should increase one's suspicion of the presence of an infection. Diagnosis is by aspiration (giving a turbid, non-viscous fluid), Gram stain and culture of fluid from the joint, as well as tell-tale signs in laboratory testing (such as a highly elevated neutrophils (approx. 90%), ESR or CRP). The ESR and CRP are almost always raised on admission, CRP being faster in diagnostics. X-ray in acute suppurative arthritis shows a widening of the joint spaces, osteoporosis at the epiphyseal bone ends of the affected joint. Puncture or a tap of the joint is crucial: the aspirate can be used to identify the type of inflammation (serous, purulent, purulo-hemorrhagic etc.). The aspirated fluid is sent for microbiological investigations to determine the type of pathogenic microorganisms and their sensitivity to antibiotics.

Treatment of acute suppurative arthritis combines both local and general therapeutic measures. Local measures include: a) puncture of the joint with aspiration of its contents, irrigation or washing of the joint cavity with antiseptic followed by infusion of antibiotics; therapeutic punctures are done daily until the accumulation of inflammatory exudates into the joint has stopped; b) immobilization of the joint with either POP slab or a therapeutic splint; c) physiotherapy, high-frequency therapy, quartz irradiation, electrophoresis with trypsin, antibiotics etc.; d) after the inflammation has subsided the patient is prescribed exercise therapy, massage and other manipulations to restore the joint functions. General therapeutic measures include antibiotic therapy, immunotherapy, blood transfusion, plasma, protein blood substitutes, detoxication therapy, rational nutrition rich in protein and vitamins. Arthrotomy is indicated when the puncture and aspiration, local and general antibiotic therapy prove unsuccessful. The joint cavity is cleared of all the purulent effusion and fibrinous deposit whereupon a drainage tube is placed for long-term washing sanitation. Paraarticular phlegmon has to be incised and drained.

Peritonitis - this is the inflammation of the parietal and visceral peritoneal layers accompanied by local changes and intoxication.

Aetiology Bacteria may enter the peritoneal cavity via four portals:

1. From the exterior: penetrating wound or infection at laparotomy.
2. From intra-abdominal viscera:

(a) gangrene of a viscus, e.g. acute appendicitis, acute cholecystitis, diverticulitis or infarction of the intestine;

(b) perforation of a viscus, e.g. perforated duodenal ulcer, perforated appendicitis, rupture of intestine from trauma;

(c) post-operative leakage of an intestinal suture line.

3. Via the blood stream: as part of a septicaemia (pneumococcal, streptococcal or staphylococcal). This has been badly termed primary peritonitis; in fact it is secondary to some initial source of infection.

4. Via the female genital tract: acute salpingitis or puerperal infection. Approximately 40 per cent of all cases of peritonitis are due to acute appendicitis, 20% are the result of post-operative complications and 20 per cent due to perforated peptic ulcer; these are the three headliners.

Classification

1. According to the extent of spread of the disease:

a) localized peritonitis: circumscribed (enclosed), unlimited (not enclosed);

b) generalized peritonitis: diffuse;

c) general (total) peritonitis.

2. According to the stage (phase) of development:

a) the reactive stage (1 - 24 hrs);

b) toxic phase (24 to 72 hrs);

c) the phase of polyorganic failure (after 72 hrs).

Pathology. Peritonitis of bowel origin usually shows a mixed flora (*Escherichia coli*, *Streptococcus faecalis*, *Pseudomonas*, and *Proteus*, together with the anaerobic *Clostridia* and *Bacteroides*). Gynaecological infections may be gonococcal or streptococcal. Blood borne peritonitis may be streptococcal, pneumococcal, staphylococcal or tuberculous. In young children a rare gynaecological infection is due to the pneumococcus.

The pathological effects of peritonitis are:

1. Widespread absorption of toxins from the large, inflamed surface.

2. The associated paralytic ileus with:

(a) loss of fluid

(b) loss of electrolytes

(c) loss of protein

3. Gross abdominal distension with elevation of the diaphragm, which produces a liability to lung collapse and pneumonia.

Clinical features. The *reactive phase* of peritonitis persists for 12 - 24 hrs and is characterized by intensive inflammatory changes in the peritoneum. Patients complain of pains in the stomach, which are intense and are initially located at the source of peritonitis, the pain later spreads to adjacent areas and can involve half or even the whole abdomen. Vomiting of stomach contents and later of bile is a common occurrence. Clinical symptoms include a rise in body temperature up to 38°C and above, tachycardia (pulse rate of up to 120 beats per min), increase in blood pressure and respiratory rate (up to 24 - 28 per min), restlessness, motor agitation. The face is initially flushed, then becomes pale. The abdomen is retracted or slightly distended; the abdominal wall or half of it is not involved in the act of breathing. On palpation the patient demonstrates severe tenderness and tensing of all the abdominal muscles depending on the spread of the process along the peritoneum. Bowel sounds are absent on auscultation. Laboratory blood tests show leucocytosis with a mild shift of the leukocyte formula to the left.

The *toxic phase* of peritonitis persists for 24 - 72 hrs and is characterized by severe intoxication and paresis of the GI (tract). Patients become adynamic, gaunted and hollow-eyed, skin is pale. Pulse rate is more than 120 beats per min and weak, blood pressure reduces. Body Temperature is high (39 - 40°C) hectic in nature, patients sometimes have chills. Abdomen is distended, tender on palpation but muscle rigidity is less than in the reactive period, abdominal percussion reveals meteorism, bowel sounds are absent. Vomiting is common of the intestinal contents. Blood test show leucocytosis with a marked shift of the leukocyte formula to the left (the appearance of immature forms) and toxic granular leucocytes.

The phase of polyorganic failure (after 72 hrs) is characterized by extreme intoxication of the organism. The patient is depressed, adynamic, apathetic to his surroundings, may be confused, quiet often develops toxic psychosis (inadequate behavior, agitation and hallucinations). The face is grayish-yellow, livid, bluish or sunken (Hippocratic face). There is profuse vomiting with the odour of faeces. The pulse is fast, weak and thready, blood pressure is low. Abdomen is very distended, tender to palpation all over. Peristaltic bowel sounds are absent («grave silence»). The body temperature reduces; the skin is covered with cold sweat. Blood tests show leucocytosis with a marked shift of the leukocyte formula to the left. Urine output is reduced, with a high proteinuria and cylinders present.

Investigations. These are of only limited value; diagnosis depends on the clinical features. X-ray of the abdomen may reveal free gas in cases of a perforated abdominal viscus (70% of perforated peptic ulcers show gas under the diaphragm). An X-ray of the chest is of aid in excluding pulmonary infection as a differential diagnosis and a serum amylase helps differentiate acute pancreatitis. There is usually a marked leucocytosis. Differential diagnosis This is from intestinal obstruction and from ureteric or biliary colic, in all of which the patient tends to be restless. Basal pneumonia, myocardial infarction, intraperitoneal haemorrhage or leakage of an aortic aneurysm are other fairly common misdiagnoses.

Principles of treatment. In this section an outline of treatment only is given, since specific causes of peritonitis may require specific therapy; these are dealt with in their appropriate chapters.

1. Relieve pain.
2. Gastric aspiration by means of naso-gastric tube; this reduces the risk of inhalation of vomit under anaesthesia and prevents further abdominal distension by removing swallowed air.
3. Fluid and electrolyte replacement by intravenous therapy; blood or blood substitutes may be required in the presence of shock.
4. Antibiotic therapy, usually to deal with the broad spectrum of bowel organisms, for example gentamicin and metronidazole, but therapy is guided, where possible, by checking the sensitivity of the responsible organisms.
5. Surgery is indicated if the source of infection can be removed or closed, for example, the repair of a perforated ulcer or removal of the gangrenous, perforated appendix. Purulent peritonitis is an indication for emergency operation. The objective of surgery is to liquidate the source of infection, sanitation of the abdominal cavity and evacuation of the purulent exudate in the abdominal cavity or the contents of the GIT in case of perforated viscus, drainage of the abdominal cavity for the infusion of antibiotics and evacuation of exudate. The preoperative assessment must be short- not more than 2 hours and aimed at the restoration of blood circulation, improvement of water-electrolyte imbalance and restoration of the circulating blood volume. Evaluation of the cardiovascular system during the preoperative period is especially important in patients who frail and elderly and who as a result of the severe intoxication easily develop cardiac decompensation. In spreading peritonitis the best surgical approach is the mid-line laparotomy incision. Liquidation of the source of infection involves the excision of the affected organ (appendectomy. cholecystectomy, removal of fallopian tubes, resection of

the intestine etc.), closure of the perforation in the case of stomach or duodenal ulcers. Sanitation (toileting) of the abdominal cavity is aimed at evacuation of the exudate with the help of electric suction machine or dry cleaning the abdominal cavity with gauze swabs, clearing of the fibrin deposits, washing the abdominal cavity with antiseptic solutions (dioxidin, soluble furagin, sodium hypochloride, ultrasonic cavitation). To combat the intestinal paresis the intestines are decompressed. In intestinal resection decompression is done through the open ends of the bowel: the bowel is brought out of the abdominal cavity, the clamps are removed and by pressing down the bowel contents and gas are emptied. In case one or both ends of the bowel are brought out in the form of a fistula, decompression will be achieved after the operation through this fistula (enterostomy or colostomy). During the postoperative period before the intestinal functions are fully restored the patient is placed on complete parenteral feeding. As the intestinal functions are restored, the patient is gradually returned to enteral feeding. To ensure detoxication, blood substitutes with detoxication properties are given as well as forced diuresis, haemadsorption, plasmapheresis etc. are used.

Suppurative pleurisy (pleural empyema) is the suppurative inflammation of the parietal and visceral pleura that is associated with local changes and intoxication. Pleural empyema, also known as pyothorax, is empyema (an accumulation of pus) in the pleural cavity that can develop when bacteria invade the pleural space, usually in the context of a pneumonia. It is one of various kinds of pleural effusion. There are three stages: exudative, when there is an increase in pleural fluid with or without the presence of pus; fibrinopurulent, when fibrous septa form localized pus pockets; and the final organizing stage, when there is scarring of the pleura membranes with possible inability of the lung to expand. Simple pleural effusions occur in up to 40% of bacterial pneumonias. They are usually small and resolve with appropriate antibiotic therapy. If however an empyema develops additional intervention is required.

Classification:

1. According to the etiology; streptococcal, pneumococcal, staphylococcal, diplococcal, mixed etc.
2. According to the pus distribution: free-total, average, minimal, encapsulated- single or multiple chambered (basal, attached in the pleural wall, paramedisternal, interlobar, and apical).
3. According to the character of the pathological changes: acute suppurative, ichorous, puruloichorous, pyopneumothorax and hemopyothorax.
4. According to the presenting clinical features: acute and chronic.

Epidemiology. The incidence of pleural empyema and the prevalence of specific causative microorganisms varies depending on the source of infection (community acquired vs. hospital acquired pneumonia), the age of the patient and host immune status. Risk factors include alcoholism, drug use, HIV infection, neoplasm and pre-existent pulmonary disease. The incidence of empyema seems to be rising in the adult population as well, albeit at a slower rate.

Symptoms. The clinical presentation of both the adult and pediatric patient with pleural empyema depends upon several factors, including the causative microorganism. Most cases present themselves in the setting of a pneumonia, although up to one third of patients do not have clinical signs of pneumonia and as many as 25% of cases is associated with trauma (including surgery). Typical symptoms include cough, chest pain, shortness of breath and fever. Body temperature rises to a high level (39 - 40°C), and is of constant or hectic character. Pulse rate reaches 120 - 130 beats/min, which indicates both suppurative intoxication and a shift of the heart and mediasternum vessels to the healthy side as a result of the fluid accumulation.

Diagnosis. The initial investigations for suspected empyema remains chest X-ray, although it cannot differentiate an empyema from uninfected parapneumonic effusion. Ultrasound must be used to confirm the presence of a pleural fluid collection and can be used to estimate the size of the effusion, differentiate between free and loculated pleural fluid and guide thoracentesis if necessary. Chest CT and MRI do not provide additional information in most cases and should therefore not be performed routinely. The most often used "golden" criteria for empyema are pleural effusion with macroscopic presence of pus, a positive Gram stain or culture of pleural fluid, or a pleural fluid pH under 7.2 with normal peripheral blood pH. Clinical guidelines for adult patients therefore advocate diagnostic pleural fluid aspiration in patients with pleural effusion in association with sepsis or pneumonic illness. Because pleural effusion in the pediatric population is almost always parapneumonic and the need for chest tube drainage can be made on clinical grounds, British guidelines for the management of pleural infection in children do not recommend diagnostic pleural fluid sampling. Blood and sputum culture has often already been performed in the setting of community acquired pneumonia needing hospitalization. It should however be noted that the micro-organism responsible for development of empyema is not necessarily the same as the organism causing the pneumonia, especially in adults. As already mentioned before, sensitivity of pleural fluid culture is generally low, often partly due to prior administration of antibiotics. It has been shown that culture yield can be increased from 44% to 69% if pleural fluid is injected into blood culture bottles (aerobic and anaerobic) immediately

after aspiration. Furthermore, diagnostic rates can be improved for specific pathogens using polymerase chain reaction or antigen detection, especially for *Streptococcus pneumoniae*, *Streptococcus pyogenes* and *Staphylococcus aureus*. Pneumococcal antigen detection in pleural fluid samples by latex agglutination can also be useful for rapid diagnosis of pneumococcal empyema. In the previously noted study, positive and negative predictive value of pneumococcal antigen detection was 95% and 90%, respectively. However, despite the additional diagnostic value of these tests, PCR and antigen detection have limited value in determining treatment choice because of the lack of information on antibiotic resistance.

Treatment. Proven empyema (as defined by the "golden" criteria mentioned earlier) is an indication for prompt chest tube drainage. This has been shown to improve resolution of the infection and shorten hospital admission. Data from a meta-analysis has shown that a pleural fluid pH of <7.2 is the most powerful indicator to predict the need for chest tube drainage in patients with non-purulent, culture negative fluid. Other indications for drainage include poor clinical progress during treatment with antibiotics alone and patients with a loculated pleural collection. Because of the viscous, lumpy nature of infected pleural fluid, in combination with possible septation and loculation, it has been proposed that intrapleural fibrinolytic or mucolytic therapy might improve drainage and therefore might have a positive effect on the clinical outcome. However, a well powered randomized controlled trial found no reduction in mortality or the need for surgery in patients who were given an intrapleural fibrinolytic agent (streptokinase) compared to placebo. Another recent randomized controlled trial did find a reduction in the need for surgery and a shorter hospital stay in patients treated with both a fibrinolytic agent (tissue plasminogen activator) and a mucolytic agent (DNase) compared to placebo or single use of either agent. Approximately 15 to 40 % of patients require surgical drainage of the infected pleural space because of inadequate drainage due to clogging of the chest tube or loculated empyema. Patients should thus be considered for surgery if they have ongoing signs of sepsis in association with a persistent pleural collection despite drainage and antibiotics. Video-assisted thoracoscopic surgery (vATs) is used as a first-line therapy in many hospitals, although open thoracic drainage remains a frequently used alternative technique. In rare cases when the closed method is ineffective, the open method is applied - thoracotomy for the evacuation of the thick pus, fibrin, sequestra of lung tissue which cannot be removed through the needle or drainage tube.

Antibiotic therapy. There is no readily available evidence on the route of administration and duration of antibiotics in patients with pleural empyema. Experts agree that all patients should be hospitalized and treated with antibiotics

intravenously. The specific antimicrobial agent should be chosen based on Gram stain and culture, or on local epidemiologic data when these are not available. Anaerobic coverage must be included in all adults, and in children if aspiration is likely. Good pleural fluid and empyema penetration has been reported in adults for penicillins, ceftriaxone, metronidazole, clindamycin, vancomycin, gentamycin and ciprofloxacin. Aminoglycosides should be avoided as they have poor penetration into the pleural space. There is no clear consensus on duration of intravenous and oral therapy. Switching to oral antibiotics can be considered upon clinical and objective improvement (adequate drainage and removal of chest tube, declining CRP, temperature normalization). Oral antibiotic treatment should then be continued for another 1-4 weeks, again based on clinical, biochemical and radiological response.

Prognosis. All patients with empyema require outpatient follow-up with a repeat chest X-ray and inflammatory biochemistry analysis within 4 weeks following discharge. Chest radiograph returns to normal in the majority of patients by 6 months. Patients should of course be advised to return sooner if symptoms redevelop. Long-term sequelae of pleural empyema are rare but include bronchopleural fistula formation, recurrent empyema and pleural thickening, which may lead to functional lung impairment needing surgical decortication. Approximately 15% of adult patients with pleural infection die within 1 year of the event, although deaths are usually due to comorbid conditions and not directly due to sepsis from the empyema.

SEPSIS

Sepsis is whole-body inflammation caused by an infection. Sepsis is caused by an immune response triggered by an infection. The infection is most commonly by bacteria, but can also be by fungi, viruses, or parasites. Common locations for the primary infection include: lungs, brain, urinary tract, skin, and abdominal organs. Risk factors include: the young, the old, people with poor immune systems such as from cancer or diabetes, as well as major trauma and burns.

Classification

1. According to the etiology: a) staphylococcal, b) streptococcal; c) pneumococcal; d) gonococcal; e) colibacillar; f) anaerobial; g) mixed.
2. According to the source: a) traumatic; b) from internal infections (tonsillitis, pneumonia etc.);
c) postoperative; d) cryptogenic.

3. According to the location of the primary focus: a) gynecological; b) urological; c) otogenic; d) odontogenic etc.
4. According to the clinical picture: a) fulminant; b) acute; c) subacute; d) recurrent; e) chronic.
5. According to the time of development; a) early (developing up to 10 - 14 days from the onset of disease or from the time of injury); b) late (developing after 2 weeks).
6. According to the reaction of the patient's organism: a) hyperergic form; b) normal reaction (normergy); c) hyperergic form.

The most common primary sources of infection resulting in sepsis are the lungs, the abdomen, and the urinary tract. Typically, 50% of all sepsis cases start as an infection in the lungs. No definitive source is found in one third to one half of cases. Infections leading to sepsis are usually bacterial but can also be fungal or viral. While gram-negative bacteria were previously the most common cause of sepsis, in the last decade gram-positive bacteria, most commonly staphylococci, are thought to cause more than 50% of cases of sepsis. Other commonly implicated bacteria include *Streptococcus pyogenes*, *Escherichia coli*, *Pseudomonas aeruginosa*, and *Klebsiella* species. Fungal sepsis accounts for approximately 5% of severe sepsis and septic shock cases; the most common cause of fungal sepsis is infection by *Candida* species of yeast.

In addition to symptoms related to the provoking cause, sepsis is frequently associated with either fever or low body temperature, rapid breathing, elevated heart rate, confusion, and edema. The increase in body temperature is a common feature of sepsis. At the beginning or during the peak of the disease the body temperature pattern is of three types: 1) remitting, in which the difference between the morning and evening temperatures is 2 - 3°C, this is evidenced in septicopyemia (sepsis with metastasis); 2) constant fever, when the temperature is persistently high and the difference between the morning and evening temperatures is 0,5°C rarely 1°C, it occurs in septicemia; 3) wave-like fever in septicopyemia: the period of subfebrile temperature after incision and drainage changes into a high temperature rise of up to 39 - 40°C, which indicates the appearance of new suppurative metastasis. In longer lasting septic conditions and with the process moving into a chronic one the temperature pattern becomes irregular. The febrile period can continue for several days to a few months, in the terminal stages when the patient is debilitated by disease the body temperature usually becomes normal. Early signs are a fast heart rate, decreased urination, and high blood sugar. Signs of established sepsis include confusion, metabolic acidosis (which may be

accompanied by faster breathing leading to a respiratory alkalosis), low blood pressure due to decreased systemic vascular resistance, higher cardiac output, and dysfunctions of blood coagulation (where clotting can lead to organ failure). The drop in blood pressure seen in sepsis may lead to shock. This may result in light-headedness. Bruising or intense bleeding may also occur.

Table. 1. Systemic inflammatory response syndrome (SIRS).

Finding	Value
Temperature	<36 °C (96.8 °F) or >38 °C (100.4 °F)
Heart rate	>90/min
Respiratory rate	>20/min or PaCO ₂ <32 mmHg (4.3 kPa)
WBC	<4x10 ⁹ /L (<4000/mm ³), >12x10 ⁹ /L (>12,000/mm ³), or 10% bands

Diagnosis is based on meeting at least two systemic inflammatory response syndrome (SIRS) criteria due to a presumed infection. Blood cultures are recommended preferably before antibiotics are started; however, infection of the blood is not required for the diagnosis. Medical imaging should be done looking for the possible location of infection. Other potential causes of similar signs and symptoms include: anaphylaxis, adrenal insufficiency, low blood volume, heart failure, and pulmonary embolism among others.

There are different levels of sepsis:

- Systemic inflammatory response syndrome (SIRS) is the presence of two or more of the following: abnormal body temperature, heart rate, respiratory rate or blood gas, and white blood cell count.
- Sepsis is defined as SIRS in response to an infectious process.
- Severe sepsis is defined as sepsis with sepsis-induced organ dysfunction or tissue hypoperfusion (manifesting as hypotension, elevated lactate, or decreased urine output)
- Septic shock is severe sepsis plus persistently low blood pressure despite the administration of intravenous fluids.

Treatment. Early recognition and focused management can improve the outcomes in sepsis. Surgical treatment of suppurative foci (abscess, phlegmon, infected wounds) involves the surgical debridement: a thorough excision of dead tissues, incision and drainage. Within the first three hours someone with sepsis should have received antibiotics, and intravenous fluids if there is evidence of either low blood pressure or other evidence for inadequate blood supply to organs (as evidenced by a raised level of lactate); blood cultures should also be obtained within this time period. After six hours the blood pressure should be adequate,

close monitoring of blood pressure and blood supply to organs should be in place, and the lactate should be measured again if it was initially raised. A related bundle, the "sepsis six", is in widespread use in the United Kingdom; this requires the administration of antibiotics within an hour of recognition, blood cultures, lactate and hemoglobin determination, urine output monitoring, high-flow oxygen, and intravenous fluids. Apart from the timely administration of fluids and antibiotics, the management of sepsis also involves surgical drainage of infected fluid collections, and appropriate support for organ dysfunction. This may include hemodialysis in kidney failure, mechanical ventilation in lung dysfunction, transfusion of blood products, and drug and fluid therapy for circulatory failure. Ensuring adequate nutrition - preferably by enteral feeding, but if necessary by parenteral nutrition - is important during prolonged illness. In those with high blood sugar levels, insulin to bring it down to 7.8 - 10 mmol/L (140 – 180 mg/dL) is recommended with lower levels potentially worsening outcomes. Medication to prevent deep vein thrombosis and gastric ulcers may also be used.

In severe sepsis and septic shock, broad-spectrum antibiotics (usually two or a β -lactam antibiotic with broad coverage) are recommended within 1 hour of making the diagnosis. For every hour delay in the administration of antibiotics, there is an associated 6% rise in mortality. Several factors determine the most appropriate choice for the initial antibiotic regimen. These factors include local patterns of bacterial sensitivity to antibiotics, whether the infection is thought to be a hospital or community-acquired infection, and which organ systems are thought to be infected. Antibiotic regimens should be reassessed daily and narrowed if appropriate. Treatment duration is typically 7–10 days with the type of antibiotic used directed by the results of cultures.

Intravenous fluids are titrated (measured and adjusted) in response to heart rate, blood pressure, and urine output; restoring large fluid deficits can require 6 to 10 liters of crystalloids. In cases of severe sepsis and septic shock where a central venous catheter is used to measure blood pressures dynamically, fluids should be administered until the central venous pressure (CVP) reaches 8 – 12mmHg. Once these goals are met, the central venous oxygen saturation (ScvO₂), i.e., the oxygen saturation of venous blood as it returns to the heart as measured at the vena cava, is optimized. If the ScvO₂ is less than 70%, blood may be given to reach a hemoglobin of 10 g/dL and then inotropes are added until the ScvO₂ is optimized. In those with acute respiratory distress syndrome (ARDS) and sufficient tissue blood fluid, more fluids should be given carefully. Crystalloid solutions are recommended initially. Crystalloid solutions and albumin are better than other fluids (such as hydroxyethyl starch) in terms of risk of death. Starches also carry an increased risk of acute kidney injury, and need for blood transfusion. Various

colloid solutions (such as modified gelatin) carry no advantage over crystalloid. Albumin also appears to be of no benefit over crystalloids. Packed red blood cells are recommended to keep the hemoglobin levels between 70 and 90 g/L. If the person has been sufficiently fluid resuscitated but the mean arterial pressure is not greater than 65 mmHg, vasopressors are recommended. Norepinephrine (noradrenaline) is recommended as the initial choice. If a single vasopressor is not enough to raise the blood pressure, epinephrine (adrenaline) or vasopressin may be added. Dopamine is typically not recommended. Dobutamine may be used if heart function is poor or blood flow is insufficient despite sufficient fluid volumes and blood pressure. The use of steroids in sepsis is controversial. The 2012 Surviving Sepsis Campaign recommends against their use in those with septic shock if intravenous fluids and vasopressors stabilize the person's cardiovascular function. During critical illness, a state of adrenal insufficiency and tissue resistance to corticosteroids may occur. This has been termed critical illness–related corticosteroid insufficiency. Treatment with corticosteroids might be most beneficial in those with septic shock and early severe ARDS, whereas its role in others such as those with pancreatitis or severe pneumonia is unclear. However, the exact way of determining corticosteroid insufficiency remains problematic. It should be suspected in those poorly responding to resuscitation with fluids and vasopressors. ACTH stimulation testing is not recommended to confirm the diagnosis. The method of stopping glucocorticoid drugs is variable, and it is unclear whether they should be slowly decreased or simply abruptly stopped. Early goal directed therapy (EGDT) is an approach to the management of severe sepsis during the initial 6 hours after diagnosis. A step-wise approach should be used, with the physiologic goal of optimizing cardiac preload, afterload, and contractility. It has been found to reduce mortality in those with sepsis. Urine output is also monitored, with a minimum goal of 0.5 ml/kg/hour. In the original trial, mortality was cut from 46.5% to 30.5%. An appropriate decrease in serum lactate however may be equivalent to SvO₂ and easier to obtain. Monoclonal and polyclonal preparations of intravenous immunoglobulin (IVIG) do not lower the rate of death in newborns and adults with sepsis. Evidence for the use of IgM-enriched polyclonal preparations of IVIG is inconsistent.