

**MINISTRY OF HEALTH OF UKRAINE
POLTAVA STATE MEDICAL UNIVERSITY**

Department general surgery with care of the patient

“APPROVED”

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**METHODICAL INSTRUCTIONS
FOR STUDENT SELF-DIRECTED WORK
WHEN PREPARING FOR AND DURING PRACTICAL CLASS**

Study discipline	General surgery
Module №2	Surgical infection. Necrosis. Bases of transplantology and clinical oncology. Methods of examination of surgical patients
Content module 1.	Surgical infection. necrosis
Lesson theme №24	Putrefactive surgical infection: pathogens, features of clinical symptoms and course, principles of treatment. Anaerobic gas gangrene: etiology, pathogenesis, clinic, treatment, specific and nonspecific prevention.
Years of study	<i>III</i>
Faculty	Medicine, Foreign students training faculty

Poltava 2021

Content module 1.	<i>Surgical infection. Necrosis.</i>
<i>Lesson theme №25</i>	Tetanus. Anthrax. Diphtheria of a wound. Etiology, pathogenesis, clinic, diagnosis, treatment, prevention. Diagnosis, prevention and treatment of tetanus in conditions of military operations and extreme situations.

1. Relevance of the topic:

The relevance of the problem lies in the fact that the prognosis of the disease is always very serious, anaerobic infection tends to spread rapidly, causes a pronounced general intoxication of the body. The most likely development of putrefactive infection and gas gangrene during transport and shaft injuries with localization of wounds in the thighs, buttocks; in persons serving cattle, pigs, sheep, goats, especially if an accident occurred in the place where animals are kept, and the wound is contaminated with manure. In addition, contamination of the wound can last as long as open lesions exist. Dirty surgical or traumatic wounds with significant tissue damage and foreign bodies are risk factors for the disease. Non-traumatic gas gangrene sometimes occurs through clostridia that enter the patient's intestines.

2. Specific objectives:

1. To explain the features of the course of putrefactive surgical infection and anaerobic gas gangrene.
2. Interpret the principles of diagnosis and differential diagnosis of putrefactive surgical infection and anaerobic gas gangrene.
3. Explain the features of surgical treatment of putrefactive surgical infection and anaerobic gas gangrene.
4. Explain the features of the conservative treatment of putrefactive surgical infection and anaerobic gas gangrene.
5. Classify the main methods for the prevention of putrefactive surgical infection and anaerobic gas gangrene.

3. Basic knowledge, skills needed to study the topic (interdisciplinary integration)

Name of previous disciplines	acquired skills
microbiology	Principles of microbiological research. Etiology of putrefactive surgical infection and anaerobic gas gangrene.
pathophysiology	Signs of an inflammatory process
pharmacology	Principles of modern rational treatment of putrefactive surgical infection and anaerobic gas gangrene.
pathanatomy	The nature of inflammation, especially the pathanatomical picture of gas gangrene

The student must have an idea:

- about the etiology, pathogenesis, classification, clinic and structure of putrefactive surgical infection and anaerobic gas gangrene;
- a modern classification of the course of the wound process of putrefactive surgical infection and anaerobic gas gangrene;
- types of wound healing in case of putrefactive surgical infection and anaerobic gas gangrene;
- about the general reaction of the body to the causative agent of putrefactive surgical infection and anaerobic gas gangrene;
- On special methods for clinical examination of patients with putrefactive surgical infection and anaerobic gas gangrene.

The student must know:

- the basics of asepsis and antiseptics;
- Definition of putrefactive surgical infection and anaerobic gas gangrene;
- etiology and pathogenesis of putrefactive surgical infection and anaerobic gas gangrene;
- histological structure of the skin, adipose tissue, muscles, bones;
- possible general and local complications;
- the main stages of surgical intervention
- Features of the management of putrefactive surgical infection and anaerobic gas gangrene;
- methods of putrefactive surgical infection and anaerobic gas gangrene;
- various types of dressings that can be used;
- principles of care for patients with putrefactive surgical infection and anaerobic gas gangrene;
- The main mechanisms and timing of the development of putrefactive surgical infection and anaerobic gas gangrene.

The student must be able to:

- apply the principles of care for surgical patients (patient hygiene taking into account the localization of the disease)
- examine the patient and the place of the disease;
- be able to give a clinical interpretation of the identified symptoms
- form a clinical diagnosis;
- prescribe conservative treatment;
- substantiate the indications for surgical intervention;
- conduct postoperative monitoring of patients and provide care;
- apply preventive measures for the development of putrefactive surgical infection and anaerobic gas gangrene;

Mastering practical skills by a student:

- master the technique of palpation in determining the boundaries of the impression of tissues, organs;
- identify the symptoms of inflammation;
- learn to detect fluctuations or softening in the focus of inflammation;

- Learn how to flush wounds with antiseptics;
- determine the clinical and morphological signs of wound healing by primary and secondary intention;
- improve the technique of applying various types of dressings, depending on the localisation of the pathological process;
- master the technique of sampling the material on the sensitivity of microflora to antibiotics.

4. The task for independent work in preparation for the lesson.

4.1. The list of basic terms, parameters, characteristics that a student must learn in preparation for the lesson:

Term	definition
anaerobic infection	Severe wound infection caused by anaerobic microorganisms and is characterized mainly by damage to the connective and muscle tissues.
Gas gangrene (anaerobic gangrene or clostridial myonecrosis)	Complications of a wound arising from infection with anaerobic spore-forming microorganisms (clostridia).
Clostridium perfringens Clostridium oedematiens Clostridium septicum Clostridium histolyticum	The main representatives of the genus that cause gas gangrene
α -toxin	Lecithinase-C, has a necrotizing and hemolytic effect;
β -toxin	Hemolysin, which in addition to hemolysis has a cardiotoxic effect
v toxin	Neuraminidase, causes new foci of necrosis and destroys the immune receptors of red blood cells
putrefactive infection	The pathological process proceeds with the active participation of putrefactive microflora.
Fournier syndrome	Idiopathic gangrene of the scrotum.

4.2. Theoretical questions for the lesson:

1. A modern view of the etiology, pathogenesis, clinical and morphological characteristics of putrefactive surgical infections and anaerobic gas gangrene.
2. Clinic diagnosis of the wound process with putrefactive surgical infection and anaerobic gas gangrene.
3. Methods of diagnosis and control of the course of the wound process with putrefactive surgical infection and anaerobic gas gangrene.
4. Modern principles and methods of treating putrefactive surgical infection and anaerobic gas gangrene;

5. Types of wound healing after surgical treatment of putrefactive surgical infection and anaerobic gas gangrene;

6. Technique for surgical treatment of putrefactive surgical infection and anaerobic gas gangrene;

7. General characteristics of drugs used for local treatment and prevention of infectious complications.

8. Therapy depending on the phase of the wound process.

9. Preventive measures for the further progression of the disease.

4.3. Practical tasks used in the lesson

1 Transportation of patients to the operating room.

2. Laying the patient on the operating table in accordance with the area of the manipulation.

3. Hand treatment to perform the intervention.

4. Be able to wear a sterile bathrobe.

5. To collect anamnesis in patients.

6. Assess the results of laboratory research methods and draw up an additional examination plan.

7. Make a treatment plan for a specific patient.

8. Drawing up a stage epicrisis.

9. Filling in the primary documentation (case of history).

10. Analysis of archival material.

11. Prepare a wound wash kit.

12. Prepare a kit for ligation.

13. Taking material for bacteriological control.

14. Various types of cleaning in a purulent dressing room.

15. Disposal of dressings.

16. Production of tables and other illustrative material (photos, slides, preparations).

5. Theme content

PUTRID (PYOGENIC) INFECTION

Putrefactive infection is a pathological process, proceeds with the active participation of putrefactive microflora.

Bacteriology. Putrefactive microflora are obligate and facultative anaerobes that realize their pathogenic properties only when conditions arise for them to metabolize without oxygen. Most often, non-clostridial anaerobes (bacteroids, anaerobic cocci (peptococci, peptostreptococci), etc.), *Proteus vulgaris*, *E. coli*, *Streptococcus putrificus* are sown. *Pseudomonas aeruginosa*, *B. sporogenes*, *Streptococcus fecalis*, *B. emphysematicus* and others are also seeded. Usually these are gastrointestinal saprophytes. It is believed that up to 90% of all surgical infections are endogenous. As a rule, there are

associations of aerobic and anaerobic flora. With putrefactive infection, microflora changes are observed at different periods of the wound process. Already at the beginning, among other microorganisms, putrefactive, predominantly proteolysing, microbes (anaerobic and aerobic, spore and nespory) are present, but they are few in the first hours. In the following days, their number increases.

In purulent and putrefactive forms, the nature of the microflora does not play a decisive role in determining the clinical manifestations and course of a surgical infection. Of paramount importance is the general state of the macroorganism (defense and detoxification systems), local and regional changes (hemo- and lymph disorders), structural features and organ damage functions. Depending on the specific combination of general and local changes, one or another variant of surgical infection occurs. In the presence of the same pathogens, the course of surgical infection in different patients is not the same.

The conditions necessary for the development of putrefactive infection are severe wound contamination, extensive necrosis (wounds with a large array of broken tissues), anaerobic conditions in the deep pockets of the wound, and a decrease in the reactivity of the body. Such conditions occur with diabetic gangrene, urinary phlegmon with pelvic fracture, umbilical and fecal phlegmon, (phlegmon of the anterior abdominal wall after damage to the colon), putrefactive peritonitis, bite and gunshot wound.

Pathogenesis. Due to trauma, surgery, tumor decay, penetration (translocation of the digestive tract flora) of putrefactive flora into the bloodstream and tissues can occur. Anaerobes in an environment unusual for them become pathogens. The main condition for the life of putrefactive flora is an oxygen-free atmosphere, a negative redox potential and the presence of growth factors. Normally, the redox potential is +150 mV, in dead tissue and abscesses it decreases to - (110-150) mV.

Putrefactive pathogenicity factors

pathogenicity factors	The action of pathogenicity factors
Enzymes - collagenase and other proteases, deoxyribonuclease, hyaluronidase	tissue destruction
endotoxins	Antigenicity, toxigenicity, activation of white blood cells and other phagocytes and immunocytes
capsule	Weakening phagocytosis and antigenicity
Metabolites - fat acids, indole, hydrogen sulfide, ammonia.	Inhibition of other bacteria, toxic effect on macroorganism cells.

In tissues, pathogens realize their proteolytic effect and contribute to the melting of tissues. The processes of tissue disintegration are accompanied by the release of a significant amount of purulent-hemorrhagic exudate and stench gas. Under the influence of putrefactive flora, the processes of fermentation and the breakdown of proteins intensify (with the formation of indole, skatol, cadaverine).

With further progression of putrefactive infection, exudation decreases, reparative processes cease, tissue necrosis progresses, there is a risk of secondary bleeding when the wound is located near the main vessels.

Toxins cause endotoxemia, disturbances in water-electrolyte balance, hypo- and dysproteinemia, changes in peripheral blood, digestive disorders and excretion.

Pathological anatomy. Necrosis and inflammation (with a predominance of necrosis over inflammation). Gas formation. Hemorrhagic exudate with droplets of fat. The tissues are gray or dirty red.

Classification.

By etiology - bacteroid, peptostreptococcal, fusobacterial, etc.

Localization - soft tissues (Wound infection, mastitis, paraproctitis, urinary and umbilical phlegmon, diabetic gangrene, fecal phlegmon of the anterior abdominal wall), internal organs (necrotic pneumonia and lung abscesses, brain abscess, endocarditis), serous cavities (peritonitis, pleural empyema)

By prevalence - local (limited), limited, systemic (generalized).

By origin - nosocomial, community-acquired.

For reasons of occurrence - traumatic, iatrogenic, spontaneous.

Examples of clinical forms are putrefactive wound infection (anaerobic cellulitis, anaerobic myositis (myonecrosis), anaerobic fasciitis), diabetic foot phlegmon, putrefactive paraproctitis, odontogenic phlegmon, appendicular abscess, Fournier syndrome.

Fournier syndrome - idiopathic scrotum gangrene. It occurs suddenly and spreads rapidly. Severe septic course. The testicles are not impressive (should not be removed). Sometimes it begins with symptoms of an acute abdomen and intestinal obstruction.

Clinic. Symptoms of endotoxemia are common symptoms (see “General issues of surgical insectology”). Local symptoms. The walls and bottom of the wound are gray, dirty red with patches of necrosis, generous purulent-hemorrhagic, gray-green or dirty brown, fetid exudate with gas bubbles and drops of fat, structureless detritus of gray or gray-green color (sometimes with black or brown patches), swelling and crepitus of tissues, skin hyperemia in the wound area. In case of adverse course, the amount of exudate decreases, the area of patches increases Crozat (ris.2.13) [1].

Gangrenous-putrefactive paraproctitis - gangrenous-putrefactive breakdown of the tissue of the sciatic-rectal and pelvic rectal cavities with the spread of the process into the subcutaneous tissue and muscles of the gluteal region. It is accompanied by severe endotoxemia. Lightning current.

Anaerobic cellulite. Local - serous inflammation, rapid progression of inflammation and necrosis in adipose tissue. The transition of necrosis to surrounding formations is possible. Severe endotoxemia. The skin is hyperemic with bluish-purple spots. "Skin necrosis. Similar changes occur in diabetic foot phlegmon.

Anaerobic (necrotic) fasciitis. A rapidly progressive purulent-putrefactive inflammatory process of the superficial fascia and secondary inflammatory and necrotic changes in fiber and skin. External signs are weak and do not correspond to the breadth of internal destruction. Toxemia is 1–2 days ahead of local symptoms. Similar changes occur in diabetic foot phlegmon.

Anaerobic myositis (myonecrosis). It begins imperceptibly after an injury or surgery. Local symptoms may be clear or unexpressed. Exudate - serous or serous-hemorrhagic. Endotoxemia. The focus of necrosis is small. The rapid spread of inflammation to the connective tissue layers of the muscles. Similar changes occur in diabetic foot phlegmon.

Differences in putrefactive phlegmon and anaerobic clostridial infection (F.Kh. Katushev (1978) in modification)

	<i>putrefactive phlegmon</i>	<i>Clostridial gas anaerobic infection</i>
Pathomorphology	Putrid tissue fusion only in the wound area	Progressive muscle breakdown without inflammatory reaction or suppuration
gas	Limited accumulation of gas in fiber. X-ray - a significant accumulation of gas.	Gas in the muscles spreads far. X-ray - striation along the muscles
exudate	Purulent-hemorrhagic, fetid, gray-green or dirty brown exudate with gas bubbles and drops of fat, rich in cells.	Serous-bloody exudate without cellular elements.
local manifestations	Inflammatory changes, necrosis of the edges of the wound, soreness in the wound area.	No signs of inflammation. The skin is pale, later crimson and deflated. Swelling is rapidly increasing.

In clinical manifestations, putrefactive infection often resembles gas gangrene, but differs from the latter in a number of signs

complications - arosive bleeding (with the proximity of the great vessels).

diagnostics - assessment of clinical manifestations, circumstances of occurrence, course of bacterioscopy (Gram smear), gas chromatography (non-volatile fatty acids), excretion of pathogens (anaerobes) in the laboratory.

treatment should be started immediately and carried out comprehensively, as in sepsis. Radical surgical treatment of the lesion includes excision of non-viable tissues, elimination of pockets, swabs, and adequate drainage. Local treatment in the postoperative period: drainage, continuous irrigation and prolonged washing with antiseptics (especially oxidizing agents), the use of sorbents and osmotically active substances. **General treatment** (see "General issues of surgical infection"). Often (when the infection covers vital organs or several anatomical sites and does not tend to be limited), palliative operations are performed - opening and decompressing tissues, limiting incisions, removal and venous ligation, drainage ulcerous strip incisions have an auxiliary value. With the progression of putrefactive decay, the spread of the process beyond the primary wound amputation is shown. With cellulite and myositis, when long absent pronounced necrosis, shown earlier operation (incision and drainage of limiting or "blocking" section). This state is the only indication to obkalyvanie antibiotics.

the forecast is always serious. A successful outcome is possible only in the conditions at the time of the begun therapy.

prevention - the proper organization of emergency care for injuries and acute surgical diseases, early and complete surgical treatment, the proper organization of treatment for patients with diabetes and obliterating vascular diseases.

GAS GANGRENE. Also known under the names: anaerobic clostridial infection, anaerobic infection, anaerobic gangrene, gas infection, acute malignant edema, bronze erysipelas, white erysipelas, military gangrene, anaerobic myonecrosis, Antonov fire. The disease has been known since the time of Hippocrates. The disease was first described in 1562 by the French surgeon Ambroise Pare.

Most often found during wars. The first information about it dates back to antiquity. During the First World War, gas infection was observed in 5-13%, World War II - in 0.5-1% of the wounded. In peacetime, sometimes it occurs after open injuries, especially agricultural and railway ones, some of its cases in this operation on the colon, removal of foreign bodies from tissues, amputation of limbs, criminal abortions and even cutting abscesses.

Mortality with gas gangrene and today is 20-50%.

Etiology and epidemiology.

The causative agents of anaerobic gangrene include:

- Clostridia perfringens (the one that forms gas) - occurs in 50-100% of cases of the disease;
- clostridia edematiens (and that forms edema) - occurs in 15-50% of cases;
- clostridia histolyticum (the one that melts the tissue)
- clostridia vibrio-septicum (septic).

The development of the disease is caused by the associations of these microorganisms. Other clostridia do not participate in the development of this process.

The source of anaerobic clostridial infection is the digestive canal (mainly the colon) of humans and animals, where they live in a vegetative form as saprophytes. With feces of clostridia, they enter the environment and exist there mainly in the form of spores,

they live for a long time. They are found in soil at a depth of 1 m, especially flavored with feces and along railway strips, in dust, on skin, hair, clothes, and linen. Clostridia are excreted from the digestive tract of 25-30% of healthy people, they are found on the skin of 44% of hospital patients

Classification of anaerobic gangrene.

1. According to clinical and morphological characteristics:

a) N.I. Pirogov distributed gas gangrene into epifascial - superficial (found in 6% of patients) and subfascial - deep (found in 94% of patients) forms;

b) M. Weinberg and Sechuin (1918) identified the following forms:

- emphysematous (classical)
- swollen (toxic)
- mixed;
- putrid;
- phlegmonous.

c) O.M. Berkutov proposed a simplified classification, according to which the following forms are distinguished:

- gas;
- gas-edematous;
- purulent-putrefactive.

The first two have the character of a deep form (subfascial), the latter of a superficial (epifascial) form in accordance with the Pirogov classification.

2. According to the clinical course, there are: lightning fast, rapidly and slowly progressing anaerobic gangrene.

3. In severity: very severe, severe and moderate.

4. During the disease, 4 phases are distinguished:

- early signs or limited gas phlegmon;
- common gas phlegmon;
- development of gangrene;
- arterial thrombosis.

Pathogenesis.

Infection occurs exclusively by the wound route, although after open, for example, railroad injuries, wound contamination with clostridia is observed in 100%. Gas gangrene occurs in no more than 1% of cases. That is, certain conditions are necessary for its development.

Factors contributing to the development of anaerobic clostridial infection:

- significant contamination of the wound with feces or land (agricultural and railway injuries are especially dangerous)
- the presence in the wound of significant areas of death, foreign bodies, "blind pockets", non-aerobic zones (gunshot fragmentation wounds are especially dangerous)
- getting into the wound with soil, calcium and silicon;
- damage to the great vessels and nerves;
- open damage to large muscle masses;
- open bone fractures;
- injuries to the legs, buttocks;

- belated, poor-quality surgical treatment of the wound (overlay without indications of primary sutures is especially dangerous)
- accession of purulent and putrefactive infection;
- the presence of severe concomitant pathology, cardiovascular failure, diabetes, anemia, hypoproteinemia, etc.
- shock, blood loss, overwork, hypothermia, etc.

In a wound, in the presence of combined favorable conditions, spores of clostridia begin to sprout and multiply rapidly. They secrete very powerful toxins: necrotoxin and hemolysin, as well as toxin enzymes: lecithinase, hyaluronidase, collagenase, proteases, etc. Enzymes and toxins increase vascular permeability, destroy cell membranes, which leads to local development of edema, hemorrhage, hemolysis and death. The muscles, connective tissue, blood vessels, nerves are affected. Vascular thrombosis contributes to the accelerated spread of necrosis.

Fermentation of glycogen and other carbohydrates causes the formation and accumulation in the tissues of lactic acid and gases: methane, hydrogen, carbon dioxide.

In the presence of putrefactive infection, ammonia and hydrogen sulfide are formed. The nature of local changes - the advantage of the development of processes of gas formation, edema, or tissue melting depends on the presence of certain associations of clostridia - pathogens of infection.

The development of anaerobic infection, as a rule, is ahead of the occurrence of a purulent process. At the same time, the imagination existing among some surgeons, the initial development of purulent infection excludes the possibility of joining clostridial does not have sufficient grounds.

Morphologically, local changes at first, as a rule, have the character of limited gas phlegmon, rapidly progressing, spreading, and passing into moist gangrene.

Inflammatory-reactive reactions in most cases are completely absent. Only with a surface purulent-putrefactive form around the affected area can leukocyte infiltration be observed.

The unlimited rapid spread of wet clostridial necrosis leads to very severe intoxication with microbial enzymes and toxins, as well as tissue breakdown products, invasion of microorganisms into the circulatory or lymphatic system. As a result, anaerobic sepsis, multiple organ failure, or bacteria-toxic shock develop.

Clinic of anaerobic gangrene.

The course of the disease is inherent in phase nature.

The first manifestation is the incubation period, which lasts from 3 (mainly) to 7 days.

local changes

In the **1st phase** - early signs (limited gas phlegmon) - dull, bursting pains in the wound intensifies or replenishes, sensations of bandage pressure, proximal edema appear, exudation from the wound decreases, its color changes from pink to gray.

In the **2nd phase** - a common gas phlegmon - the wound acquires a gray-brown color, the exudate becomes cloudy, smelly, smells like peroxide of cabbage or stale cheese, swelling spreads, pallor with blue vein contours, crepitus appears.

In the **3rd phase** - the development of gangrene on the surface of the skin - blisters appear, spots of crimson-blue and yellow-green color and the color of the "autumn forest" (symptom of "land card").

In the 4th phase - arterial thrombosis - the ending becomes cold as ice, black and blue, dirty brown ("boiled") muscles are visible from the wound, there is no pulsation of the peripheral arteries, sensitivity, limb movements.

common manifestations

In the early stages, patients observed: weakness, anxiety, agitation, fever, tachycardia.

Further, the general condition quickly worsens, excitement is replaced by emotional suppression and depression, appetite disappears, mucous membranes and tongue become dry, become covered with a dirty coating, the temperature rises to 39-40 ° C, tachycardia increases, blood pressure decreases. In blood tests, an increase in the number of leukocytes, a significant shift of the leukocyte formula to the left, acceleration of ESR, and the progression of anemia are determined.

With the development of sepsis, symptoms of general intoxication intensify, there are signs of multiple organ failure and a decrease in diuresis, jaundice with a corresponding increase in the level of nitrogenemia, bilirubinemia.

Toxic shock - is accompanied by a rapid drop in hemodynamics and other vital functions and can quickly lead to the death of the patient.

Treatment of gas gangrene should be comprehensive. At the same time, surgical intervention is crucial. The operation is performed urgently after short-term preparation aimed at correcting hemostasis, under endotracheal anesthesia or under certain conditions - epidural anesthesia.

In cases of phlegmon or limited muscle necrosis with the preservation of the pulse in the distal part of the limb, a wide cut of the skin is possible with complete removal of necrotic tissue within the limits of visually unchanged. It is necessary to try to make a radical surgical treatment of the site of infection and, if possible, get a "clean" wound. In addition, some authors recommend that the edges of the wound be widely distracted and fixed with provisional sutures to unaffected skin.

Do not be afraid of the formation of a large tissue defect. Only a radical removal of all areas of necrosis gives a chance to save the patient's life.

Tape incisions is a palliative operation, they lead to the formation of a large purulent wound, therefore they are not very favorable and have only auxiliary value.

With the development of gangrene of the extremity, as evidenced by the coldness of the skin, the absence of pulsation of the artery in the distal parts, the disappearance of sensitivity, contracture, etc., Guillotine amputation is shown, as proximal to the affected area. Tissues intersect at one level. The stump is NOT sutured, the wound is permanently fused with oxidizing agents: solutions of 3% hydrogen peroxide or potassium permanganate. Local application of dioxidine and octenisept is possible, to which anaerobes are sensitive. With a favorable course, the wound is sutured with repeated sutures for drainage. A mandatory component should be antibiotic therapy. Since the isolation and identification of anaerobes, as well as the determination of their sensitivity to

antibiotics, are complex and require 2-4 days, the initial administration of antibiotics is of an imperial nature. They should be prescribed given the spectrum of action of the drugs,

It is necessary to start antibiotic therapy simultaneously with the operation. Drugs are administered in maximum doses. The most active antibiotics of a wide spectrum of activity against anaerobes are: lincosamides-clindamycin (dalacin), which is advisable to be prescribed together with aminoglycosides; carbapenemiv derivatives - meronem, thienam, and mini; some 3rd generation cephalosporins - cefotaxime (cefantal), cefobid, etc. beta-lactamase - mefoxin; Fortum, rifampian drugs, as well as antimicrobials of the imidazole group - metranidazole, metragil, metrid, flagin, etc.

Hyperbaric oxygenation is the pathogenetic means of postoperative treatment (patients stay in special chambers with high oxygen pressure - pressure chambers).

It is also necessary to apply massive infusion therapy aimed at detoxification (transfusion of crystalloid solutions, blood substitutes - neo-hemodesis, rheosarbylact, etc.) and providing the body with energy substances (10% glucose solution with potassium and insulin) and proteins (plasma, albumin) with correction of water-electrolyte. If appropriate: forced diuresis, blood ultraviolet radiation and extracorporeal detoxification by lymph and plasma sorption methods.

Important treatments include red blood cell transfusion, direct-acting anticoagulants, proteolysis inhibitors, corticosteroids.

Patients need a high-calorie and high-vitamin diet. If necessary, the food mixture is introduced into the stomach using a probe.

The decisive means of preventing anaerobic gangrene is timely and adequate surgical treatment of the wound with a wide incision and the most complete removal of non-viable tissues. Particular attention should be paid to the use of primary joints. At the lowest risk of infection, it is better to throw the wound after surgery not wired, but in case of a favorable course

6. Materials for self-control.

- 1) The specificity of pathogens of putrefactive surgical infection and gas gangrene;
- 2) Causes of putrefactive surgical infection and gas gangrene;
- 3) Definition of putrefactive surgical infection and gas gangrene;
- 4) Clinical symptoms of putrefactive surgical infection and gas gangrene;
- 5) Diagnostic methods for putrefactive surgical infection and gas gangrene;

6.1. The task for self-control.

question:

1. The specificity of pathogens of putrefactive surgical infection and gas gangrene;
2. Causes of putrefactive surgical infection and gas gangrene;
3. Definition of putrefactive surgical infection and gas gangrene;
4. Clinical symptoms of putrefactive surgical infection and gas gangrene;
5. Diagnostic methods for putrefactive surgical infection and gas gangrene;
6. Conservative and surgical treatment of putrefactive surgical infection and gas gangrene;
7. Sanitary-hygienic regimen for caring for patients with putrefactive surgical infection and gas gangrene;

8. Specific and non-specific prophylaxis of putrefactive surgical infection and gas gangrene.

Tasks:

- 1) Features of the course of putrefactive surgical infection and gas gangrene;
- 2) Principles of management of patients with putrefactive surgical infection and gas gangrene;
- 3) Surgical treatment of putrefactive surgical infection and gas gangrene;
- 4) Prevention of gas putrefactive surgical infection and gas gangrene;
- 5) The sanitary-hygienic regime in the departments in the treatment of putrefactive surgical infection and gas gangrene

Test tasks.

6.3 Tests for self-control (initial level of knowledge)

Tests and tasks of checking the initial level of knowledge.

1. Optimal conditions for the development of gas gangrene arise when:

- a) closed fractures;
- b) mechanical damage to the skin;
- c) burns of the II-IV degree;
- d) frostbite II-IV degree;
- e) insect bites.

2. The characteristic local signs of gas gangrene are:

- a) inflammatory reaction, necrosis, edema, intoxication;
- b) lack of inflammatory reaction, edema, necrosis
- c) edema, lymphangitis;
- d) elephantiasis;
- e) white blood cell an increase, bacteremia, subfascial phlegmon.

3. The preferred localization of the process with gas gangrene are:

- a) head, neck;
- b) limbs;
- c) the body;
- d) perineum;
- e) the intestines.

4. When exposed to pathogens of gas gangrene, the following develops:

- a) numerous abscesses;
- b) detachment of the epidermis with necrosis of the subcutaneous tissue;
- c) gas formation with necrosis of muscles and connective tissue;
- d) traumatic shock
- e) necrosis of the skin, muscles, and bone tissue.

5. Clinically distinguish the following forms of gas gangrene:

- a) lightning fast, sluggish;
- b) acute, subacute;
- c) acute, chronic;
- d) chronic, recurrent;
- e) acute, recurrent.

6. Pathanomic distinguish the following forms of gas gangrene:

- a) erythematous, bullous, phlegmonous, necrotic;
- b) metastatic, septic;
- c) septic, septicopyemic;
- d) emphysematous, necrotic, phlegmonous, edematous;
- e) catarrhal, septic, tissue fusion.

7. According to the anatomical classification, the following forms of gas gangrene are distinguished:

- a) epifascial, subfascial;
- b) intraarticularis;
- c) intraosseal;
- d) epidural, subdural;
- e) skin, subcutaneous, muscle.

14. The characteristic features of gas gangrene when examining a wound are:

- a) edema, hyperemia, purulent discharge;
- b) swelling, red spots and streaks on the skin
- c) fetid discharge from the wound, pallor of the skin;
- d) emphysema, rapid increase in edema;
- d) pulling pain in the wound, swelling, muscle twitching.

15. Non-specific prophylaxis of gas gangrene includes:

- a) primary surgical treatment of the wound;
- b) massive antibiotic therapy;
- c) the introduction of antigangrenous serum;
- g) chipping the wound with antibiotics
- d) determination of the sensitivity of the pathogen to antibiotics.

16. The most important treatment measures for gas gangrene are:

- a) antyshock therapy;
- b) the introduction of therapeutic doses of tetanus toxoid;
- c) desensitizing and antibacterial therapy;
- d) opening the focus of infection with necrectomy and oxybarotherapy;
- e) chipping of lesions with antibiotics.

17. What is the minimum dose of antigangrenous serum in the treatment of gas gangrene:

- a) 30,000 IU;

- b) 90,000 IU;
- c) 150,000 IU;
- d) 300,000 IU;
- d) 600,000 IU.

18. The characteristic clinical signs of non-clostridial soft tissue infection are:

- a) edema, marble pattern of the skin, gas formation;
- b) hyperemia of the skin with clear boundaries;
- c) necrosis of muscles and connective tissue, gas formation;
- d) abscess formation with purulent hemorrhagic fetid discharge;
- e) anemia, white blood cell an increase, muscle twitching in the area of inflammation.

Situational tasks for the initial level of knowledge

1. A patient with gas gangrene of the lower leg was admitted to the department. Given the high contagiousness of anaerobic clostridial infection, it is necessary to organize and monitor compliance with the sanitary-epidemiological regime in the department. What does this consist of?

2. Patient Ch., 24years old, operated for acute appendicitis. On the fifth day, severe hyperemia of the wound edges, edema of the subcutaneous tissue in the hypochondrium and upper third of the thigh were noted. Preliminary diagnosis? What methods can be verified?

3. Patient K., due to anaerobic gas gangrene of the right lower leg, underwent a high amputation of the right leg. How is the used dressing handled?

4. Patient A. was subjected to exarticulation of the right upper limb due to anaerobic gas gangrene of the right shoulder. How is the processing of used tools performed?

Tests of difficulty level III

1. A 65-year-old patient received a superficial wound (abrasion) of his lower leg during agricultural work. I did not seek medical help for the injury. There is no evidence of tetanus vaccination. After 10 days, there were pains in the wound area, pain when swallowing, sweating. After 2 days, there was stiffness of the masticatory muscles and muscles of the neck, sweating. Tonic and clonic convulsions after 10-20 minutes with a tendency to increase in frequency, impaired swallowing, breathing, profuse sweating, temperature 40°C Determine the diagnosis, form, degree, prognosis, disease, treatment principles.

2. A 30-year-old patient is a livestock procurer, hospitalized in the surgical department 5 days after the onset of the disease with a diagnosis of erysipelas. There is a

rapidly progressing widespread, dense, painless swelling of the forearm, covered with small vesicles, patches of necrosis. Part of the vesicles burst, a large amount of serous fluid is released. The patient's condition is severe, severe headache, delirium, and temperature reaches 40°C. What disease should be suspected, what should be done to clarify it, what measures to cause. Determine the prognosis and treatment.

3. In a patient of 20 years, after opening the phlegmon of the thigh, the wound was cleaned, partially filled with granulations, the general condition and temperature returned to normal. For no apparent reason, the general condition again worsened significantly, the temperature rose to 39 ° C, tachycardia arose, sleep, appetite worsened. The wound was covered with a dirty, tightly soldered plaque of fibrin, serous hemorrhagic discharge, edema and hyperemia of surrounding tissues, regional lymphadenitis appeared. What complication takes place, what needs to be done to clarify it, the necessary organizational and therapeutic measures?

4. 44-year-old Man, fell into an open sewer pit. Got an open fracture of the right lower leg. The surgical department conducted the initial surgical treatment of the wound using blind joints and skeletal traction. On the second day, the patient appeared euphoric, pain in the wound, a feeling of fullness and embossing of the dressing. What is the complication? What local changes are characteristic for such a complication?

5. Three patients with fractures of the lower leg bones and significant contamination of the ground and pieces of clothing with lacerations of the lower extremities were delivered to the district hospital from the scene of the traffic accident. Your actions?

6. After administering antigangrenous serum for prophylactic purposes to the patient, fever, pain in the ore cell, sharp abdominal pain, vomiting, drop in blood pressure, cold sweat, cyanosis, stupefaction appeared. What complication arose? Your actions?

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The distribution points are awarded to students:

At mastering topic number 24 to number 2 module for training activities for students rated a 4-point scale (traditional) scale, which is then converted into points as follows:

rating	Points
5 (excellent)	5
4 (good)	4
3 (satisfactory)	3
2 (poor)	0

Guidelines prepared

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