MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty *Medicine*
Department *Surgery, Radiological Diagnostics, Radiation Medicine, Therapy and Oncology*

APPROVED BY
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METHODOLOGICAL RECOMMENDATION FOR PRACTICAL CLASSES OF THE ACADEMIC DISCIPLINE

Faculty, course Medical 6th year

Academic discipline Surgery
(name of the discipline)

PRACTICAL CLASSES

Practical class № 16

Topic: "Surgical treatment of infectious diseases. Causes, diagnosis and differential diagnosis, treatment tactics"

Approved:

At the meeting of the Department of Surgery, Radiation Diagnostics, Radiation Medicine, Therapy and Oncology of Odesa National Medical University

Odesa National Medical University

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PRACTICAL CLASSES

Practical class № 16

1. **Topic: "Surgical treatment of infectious diseases. Causes, diagnosis and differential diagnosis, treatment tactics"**
2. **Relevance of the topic.**

Surgical complications of infectious diseases occupy one of the main places in surgical practice. The history of surgery is inextricably linked with the fight against infection. The widespread use of antibiotics due to their mutagenic effect led to a change in the species composition and properties of the pus-forming microbial flora, which reduced the effectiveness of antibiotic therapy. Of particular importance are the issues of surgical treatment of complications of infectious diseases, where conservative methods should be rationally combined with timely surgical intervention, determination of indications for hospitalization of such patients.

Existing methods of treating infectious diseases do not always cause the suppression and destruction of the infection. The most important point is surgical complications of infectious diseases, which have one of the main places for the treatment of complications of infectious diseases.

3. Objectives:

3.1. General objectives: A student of higher education must learn:

1.	Identify anamnestic and clinical objective signs of surgical complications in infectious diseases	II level
2.	Basic principles of diagnosis, differential diagnosis	II level
3.	Assign an examination plan using laboratory and X-ray examinations.	III level
4.	Provide emergency conservative care to patients with surgical complications in infectious diseases.	III level
5.	Determine indications for surgical intervention and theoretically know the methodology of their implementation.	II level

3.2. <u>Educational objectives:</u>

1. Formation of a professionally significant personality of the doctor.

To emphasize the achievements of the national surgical school in the development of modern methods of treatment of surgical complications in infectious diseases.

3.3. <u>Specific objectives</u>:

to know:

- Etiology, pathogenesis.
- Clinical picture of surgical complications in infectious diseases;
- Differential diagnostic criteria of surgical complications in infectious diseases;
- Methods of instrumental and laboratory examination of patients with surgical complications in infectious diseases;
- Conservative and operative treatment of patients with surgical complications in infectious diseases;

3.4. Based on theoretical knowledge of the topic:

Be able to (master techniques):

- Collect medical history.
- Conduct differential diagnosis between purulent diseases of various genesis and other infectious diseases;
- Determine the diagnosis of the disease.
- Prescribe conservative or operative tactics for the treatment of the disease.

3. (interdisciplinary integration).

№	Disciplines	To know	To be able to
1	**2**	**3**	**4**
I. Previous disciplines			
1.	Anatomy, topographical anatomy	The structure of the skin, lungs, liver, spleen, stomach, intestines, ways of possible movement of purulent exudate through anatomical channels	To establish the source of inflammation, to carry out differential diagnosis between purulent diseases of various genesis and other infectious diseases; place of incision.
2.	Pharmacology	Mechanism of action of antibacterial drugs	

4. Content of the class:

Etiology and pathogenesis of infectious diseases that may have surgical complications. Classification. Diagnostics. Differential diagnosis.

Etiology, pathogenesis.

Surgical complications of infectious diseases occupy one of the most important places in surgical practice. Most often, these complications occur in patients with specific infections: tuberculosis, HIV infection, typhoid fever, cholera, Crohn's disease, meningococcal infection, helminthiasis (hookworm, echinococcosis, giardiasis, nematodes), malaria, anthrax, blood diseases, syphilis, and poisoning.

However, a specific infection is often accompanied by other infections *such as staphylococci*, *streptococci*, *pneumococci*, *gonococci*, *Escherichia coli and Klebsiella pneumoniae*, etc., often in symbiosis with anaerobic microorganisms. Bacteria that get into the wound begin to show their vital activity and multiply in it on average after 6 to 12 hours.

The introduction of infection and the development of the disease, which contribute to their development, are:

a) the presence of a nutritious environment for them in the area of injury (hemorrhage, necrotic tissue);

b) simultaneous coexistence of several types of microbes (polyinfection);

c) penetration of germs of increased virulence, for example, contamination of the injury site with purulent discharge from another patient;

d) weakness of immunological reactions;

e) disorders of local and general blood circulation of the patient.

The body responds to the appearance of bacteria with a local and general reaction. The local tissue reaction is expressed primarily by changes in blood circulation of a neuro-reflex nature. Arterial hyperemia develops, followed by venous stasis with the formation of

edema, pain, local fever, etc. A large number of neutrophilic leukocytes accumulate in the inflammatory focus.

The general reaction of the body to the introduction of specific and pus-forming microbes occurs simultaneously with the local reaction. Its degree depends on the amount of bacterial toxins and tissue breakdown products, as well as the body's resistance. Particularly virulent microbes that secrete toxins usually cause a strong general reaction of the body. Its manifestations are: fever, blackouts, and sometimes fainting, headache, general malaise, tachycardia, sharply pronounced changes in blood counts, liver function, blood pressure, and stagnation in the small circulation. Patients need a thorough examination to identify the primary purulent focus and the entrance gate.

There are hyperergic, normergic, and hypoergic reactions.

Hyperergic – is a rapidly developing process that, despite timely, rational treatment, often ends in death.

Normergic – the process develops less rapidly, fewer tissues are involved in the inflammatory process, and changes in the blood are not evident. And this process is easier to treat.

Hypoergic – the inflammatory process is limited to a small area, with less swelling. These processes are easily treatable, and in some cases, you can do without treatment, but the latter is possible only if the body has good defenses, otherwise the infection becomes protracted.

Classification. Diagnostics. Treatment

Taking into account the peculiarities of the clinical course and the nature of changes in the inflammatory focus, acute and chronic forms are distinguished from all types of surgical infection.

1. Acute surgical infection:

a) purulent;
b) putrid;
c) anaerobic;
d) Specific (tetanus, anthrax, tuberculosis, HIV, typhoid, cholera, Crohn's disease, meningococcal infection, helminthic diseases (ascaris, echinococcosis, giardiasis, nematodes), malaria, blood diseases, poisoning.

2. Chronic surgical infection:

a) nonspecific (pus-forming);
b) Specific (tuberculosis, syphilis, actinomycosis, etc.).

In each of these forms, there can be forms with a predominance of local manifestations (local surgical infection) or with a predominance of general phenomena with a septic course (general surgical infection).

5.1 Tuberculosis.

The term “tuberculosis” was introduced by Laennek, derived from the Latin word for “tubercle”.

In 1882, German researcher Robert Koch provided comprehensive evidence of the infectious nature of tuberculosis through his scientific work. He identified and described the causative agent of the disease. The causative agent of tuberculosis is commonly referred to as the Koch bacterium (KB) or Mycobacterium tuberculosis (MTB). It is a representative of a large group of mycobacteria related to lower plant organisms - the radiating fungi.

There are several types of tuberculosis mycobacteria that can cause disease in humans: human, bovine, avian, mouse and African species.

In men, the disease is caused by the human species in 92-95% of cases, and by the

bovine species in 3-5% of cases. The other two - avian and mouse - are almost safe for humans. In 1969, a subspecies of mycobacterium tuberculosis, called African tuberculosis, was first isolated from humans in Central Africa.

In our country, there are currently 3 methods for detecting tuberculosis: tuberculin diagnostics, fluorographic method and bacteriological examination of sputum.

Tuberculin diagnostics is used for children and adolescents under 15 years of age. For these purposes, a single intradermal tuberculin Mantoux test is used. The test result is evaluated after 72 hours, the size of the infiltrate is determined using a transparent ruler. The reaction can be negative, doubtful, positive, weakly positive, of medium intensity and pronounced. Negative tests are observed in healthy people who are not infected with TB.

The main method of preventive examinations of the population aged 15 years and older is a fluorographic examination.

Chemotherapy is the treatment of patients with anti-tuberculosis drugs. It is the leading treatment for patients with tuberculosis.

1.2. CLASSIFICATION

Respiratory tuberculosis:

- Focal pulmonary tuberculosis
- Intrathoracic tuberculous lymphadenitis
- Tuberculosis of the lungs
- Tuberculosis intoxication
- Infiltrative pulmonary tuberculosis
- Cavernous tuberculosis of the lungs
- Pulmonary tuberculosis fibrous-cavitary
- Cirrhotic pulmonary tuberculosis
- Tuberculosis of the upper respiratory tract, trachea, bronchi.

Respiratory tuberculosis combined with dust-related occupational lung diseases.

Tuberculous Lymphadenitis:

- Tuberculosis of the peripheral lymph nodes
- Tuberculosis of the mesenteric lymph nodes

Bone and joint tuberculosis:

- shoulder joint
- elbow joint
- hip joint
- knee joint
- Tuberculosis of the brain
- Tuberculosis of the eye
- Tuberculosis of the larynx
- Tuberculosis of the ureter and genitals
- Tuberculosis of the adrenal glands
- Tuberculosis of the intestines
- Tuberculosis of the skin

5.3. CLINICAL PICTURE

- Primary tuberculosis.

Primary tuberculosis develops after mycobacteria come into contact with tuberculosis mycobacteria. It is mainly pulmonary tuberculosis.

Secondary tuberculosis.

Secondary tuberculosis, i.e. tuberculosis in people who have had primary tuberculosis in the past, can occur both endogenously and as a result of repeated (exogenous) infection of the body.

Clinical signs of tuberculosis.

Of all the organs and systems, the lungs are most often affected by tuberculosis, and damage to other organs often develops as a complication of the pulmonary process. Early detection of tuberculosis is one of the most important tasks of a doctor. In children, external lymph nodes (cervical, submandibular, axillary, inguinal), as well as lymph nodes of the chest and abdominal cavities, are often affected by tuberculosis. When the tuberculosis process develops in the lung, one of the signs of the disease may be a fever. The high temperature can last for 2-3 weeks and then drop to 37.2-37.4°. Unlike pneumonia, a decrease in temperature does not lead to recovery in tuberculosis, and the patient continues to have all or part of the previously mentioned symptoms. Profound metabolic disorders in the body, digestive disorders, the breakdown of proteins to their final products and tissue death cause weight loss and weakening of the body in pulmonary tuberculosis.

6. Laboratory diagnostics.

Laboratory diagnosis of tuberculosis includes bacteriological and bacterioscopic methods of examination, biological and allergic reactions. Serologic reactions have also been proposed, but have not been used in practice.

Typhoid fever. Paratyphoid A and B

Acute infectious diseases caused by Salmonella bacteria, which, affecting the lymphatic formations of the small intestine, enter the bloodstream and, spreading throughout the body, cause intoxication, enlargement of the liver and spleen, the appearance of a rosaceous rash and central nervous system depression.

Causes of occurrence

Drinking contaminated water or food. Transmitted through dirty hands or objects contaminated with the secretions of a patient or carrier.

Disease development

When entering the human body through the mouth, the pathogens of typhoid fever and paratyphoid fever A and B penetrate the lumen of the small intestine, where they invade its lymphoid elements, multiply, break through the lymphatic protective barrier and enter the bloodstream. They are carried by the bloodstream through the body, settle in various organs and cause dystrophic processes, and their toxins affect the central nervous system. When pathogens enter the skin vessels, a rosaceous rash appears. Part of the pathogens from the liver, through the gallbladder with bile, re-enters the lumen of the small intestine, where it again penetrates the lymphoid elements, causing very rapid cell death, destruction and formation of ulcers in the intestinal wall due to an allergic reaction. Due to the formation of ulcers in the wall, perforation and development of peritonitis are possible, and bleeding is possible in the case of destruction of blood vessels in the area of ulceration.

Symptoms

The latent period of the disease (incubation) before the first symptoms appear can last from 7 to 25 days in typhoid fever and somewhat less in paratyphoid fever. Most often it lasts 9-14 days.

In the typical form, the disease is cyclical. The initial period of the disease is characterized by rapid or slow development of intoxication syndrome (weakness, fatigue, chills, headache, sleep disturbance, gradual increase in temperature up to 40 °C), the

appearance of flatulence, which is expressed more often by stool retention than diarrhea. The patient is lethargic and pale. Slow and monosyllabic answers to questions. There is a discrepancy between the pulse and high temperature, its frequency is less than it should be. The liver and spleen become enlarged. By the end of the first and beginning of the second week, the period of the disease's peak occurs, when all symptoms reach their maximum development. On the 8th-10th day of the disease, a pale, rosaceous rash appears on the skin of the abdomen and chest. The abdomen is swollen, and there is pain in the right side. This is the most dangerous period of the disease, when complications are possible. Then comes the recovery period, when the impaired body functions are restored and the infection is released.

Diagnostics

The diagnosis of typhoid fever and paratyphoid fever A and B is made on the basis of clinical symptoms, dynamics of the disease and laboratory data (bacteriological culture of feces, urine, blood and bile on special environments, as well as the detection of specific antibodies against typhoid fever and paratyphoid fever in the blood).

Treatment

Patients are required to be hospitalized in an infectious disease hospital due to the severity of the condition, the danger to others, and the need for timely treatment. A special regimen and diet are required. For treatment, antibiotics and antibacterial agents (levomycetin, tarevit, dordum, etc.) are used. In case of complications, special medications are used up to the surgery.

Preventive measures

Early detection of patients and their hospitalization is of great importance, which reduces the likelihood of bacterial carriage and the risk of infecting others. All persons who have been in contact with a typhoid patient are subjected to a stool culture and daily temperature measurements and examinations for 21 days. Personal hygiene is required. A vaccine is used to protect against the disease for 5 years.

Cholera - is an acute anthropogenic fecal-oral infection caused by cholera vibrio, which manifests with symptoms of watery diarrhea, vomiting with possible development of dehydration shock. It is a particularly dangerous infection.

Etiology. The causative agent of cholera is cholera vibrio.

Epidemiology.

The mechanism of cholera infection is fecal-oral. The transmission routes are water, nutritional, and household contact. As with all intestinal infections, cholera is characterized by summer-autumn seasonality.

Pathogenesis.

After crossing the gastric barrier, cholera vibrio quickly contaminates the mucous membrane of the small intestine. This infection is not an invasive one - vibrio are localized on the surface of the mucous membrane and in its lumen. The main role in the pathogenesis of cholera is played by large amounts of exotoxin secreted by vibrio during their vital activity.

Clinical picture.

The incubation period varies from several hours to 5 days, with an average of 2 days. There are typical and atypical forms of cholera. In typical cholera, there are mild, moderate, and severe cases.

The atypical form can occur as erased, “dry” and lightning cholera.

The stool is watery, in typical cases it looks like rice broth. There are 4 degrees of dehydration.

Dehydration of the first degree is a loss of fluid in the amount of 1-3% of body weight. The condition of patients during this period suffers little. The main complaint is thirst. Dehydration of the second degree - loss of 4-6% of body weight is characterized by a moderate decrease in the volume of circulating plasma. This is accompanied by increased thirst, weakness, dry mucous membranes, tachycardia, a tendency to lower systolic blood pressure and diuresis. Dehydration of the third degree is characterized by a loss of 7-9% of body weight. At the same time, the volume of circulating plasma and intercellular fluid significantly decreases, renal circulation is impaired, and metabolic disorders appear: acidosis with accumulation of lactic acid. There are cramps of the calf muscles, feet and hands, reduced skin turgor, tachycardia, hoarseness of the voice, cyanosis. Due to severe dehydration, facial features become sharpened, eyes droop, the “dark glasses symptom”, “fades cholerica” is noted, and wrinkling of the skin of the hands determines the symptom of “laundress's hands”.

Hypotension, hypokalemia, acidosis, oliguria, characteristic of the third degree of dehydration, can be reversed by adequate therapy. In its absence, the IV degree of dehydration (loss of more than 10% of body weight) leads to the development of deep dehydration shock. The body temperature drops below normal (choleric algid), shortness of breath increases, aphonia, severe hypotension, anuria, and muscle fibrillation appear. Decompensated metabolic acidosis and signs of severe tissue hypoxia develop. Only emergency care at the prehospital stage and hospital therapy can save the patient.

In cases where dehydration shock develops within a few hours (one day), the form of the disease is called lightning. Dry cholera occurs without diarrhea and vomiting, but with signs of rapid development of dehydration shock - a sharp drop in blood pressure, development of tachypnea, shortness of breath, aphonia, anuria, and convulsions.

Diagnostics. In laboratory diagnostics, bacterioscopic examination of feces and vomit is possible, which is of indicative value. Among the rapid diagnostic methods: RIF, ELISA, etc.

Treatment. All patients with or suspected of having cholera are subject to mandatory hospitalization. The immediate treatment measure is to replenish water and electrolyte deficiencies with oral rehydration solutions. In the presence of vomiting, as well as in patients with severe disease, intravenous polyionic solutions are administered.

The main principle of treatment of patients with cholera is immediate rehydration at home, in an ambulance and in a hospital upon first contact with the patient. In mild and moderate cases, oral rehydration should be performed.

The WHO Expert Committee recommends the following composition for oral rehydration: sodium chloride - 3.5 g, sodium bicarbonate - 2.5 g, potassium chloride - 1.5 g, glucose - 20 g, boiled water - 1 litre.

Cholesterol-anatoxin is used for specific prophylaxis.

Differential diagnosis

Cholera is distinguished from salmonellosis, foodborne toxicity, shigellosis, poisoning with pesticides, heavy metal salts and fungi, rotavirus gastroenteritis, and escherichiosis.

Laboratory diagnostics

In severe cholera, a preliminary diagnosis is made on the basis of clinical findings and epidemiologic history. However, the final diagnosis is possible only after obtaining the results of a bacteriological test, which takes 36-48 hours.

Complications of cholera can be caused by secondary infection with the development of

pneumonia, abscesses and phlegmon. Prolonged intravenous manipulations can cause pyrogenic reactions, phlebitis and thrombophlebitis. Acute cerebrovascular accident, myocardial infarction, and mesenteric vascular thrombosis are also possible.

Anthrax

Anthrax (synonyms: malignant carbuncle; anthrax - English; Milzbrand - German; Charbon, anthrax carbon - French) is an acute infectious disease, a zoonosis from the group of infections of the external integument. It is included in the group of particularly dangerous infections. The name of the microbe comes from the Greek “anthracis” - coal, which is explained by the formation of similarly colored ulcers on the skin during infection.

Characteristics of the pathogen

Pathogen - Bacillus anthraxis, aerobus, facultative anaerobe, is a Gram-positive, non-motile, rather large rod 6-10 μm long and 1-2 μm wide; Gram stain. In susceptible organisms, the vegetative form produces a capsule, which is a polypeptide with antiphagocytic activity, prevents opsonization and phagocytosis of bacilli and simultaneously promotes their fixation on host cells, when exposed to free oxygen and at a temperature of 15-42 °C. The presence of the capsule distinguishes virulent anthrax strains from vaccine anthrax.

The aggressiveness of the microbe in the body is largely due to the capsule substance, which is a polymer of D-glutamic acid. It is the capsule that inhibits phagocytosis, preventing the death of the bacillus, and protects it from the bactericidal effects of lymph and blood.

Bacillus is sensitive to most conventional antibiotics of the penicillin and tetracycline groups, levomycetin, streptomycin, and neomycin.

The causative agent of anthrax was discovered and isolated in pure culture in 1876. by R. Koch. He also grew the bacterium in an artificial nutrient medium, detected spore formation, and reproduced anthrax infection in an experiment on mice. Only 5 years later, L. Pasteur obtained and tested a live anthrax vaccine on animals.

Epidemiology

Anthrax is a unique infectious disease of animals and humans. Once it occurs in any area, it can take root, keeping the threat of repeated outbreaks alive for many years.

Ways of infection

The entry point for the cutaneous (localized) form is any area of the skin or mucous membranes. In this case, the pathogens form capsules and secrete an exotoxin that causes dense edema and necrosis. After 2 to 14 days, an anthrax carbuncle develops at the site of introduction.

For alimentary infection, mechanical damage to the intestinal mucosa is required. When eating infected (and insufficiently processed) meat, spores penetrate the submucosa and regional lymph nodes. The intestinal form of anthrax develops, in which the pathogens also penetrate the bloodstream and the disease generalizes and becomes septic. Primary intestinal anthrax does not develop.

Anthrax is highly treatable if diagnosed in the early stages of the disease.

Diagnosis and differential diagnosis. Identification of the causative agent.

Laboratory confirmation of the diagnosis is the isolation of an anthrax bacillus culture and its identification. For the study, the contents of the pustule, vesicles, and tissue effusion from under the scab are taken. In case of suspected pulmonary form, blood, sputum, and feces are taken. In cutaneous forms, hemoculture is rarely performed. To examine the material (skins, wool), a thermoprecipitation reaction (Ascoli reaction) is used. The immunofluorescent method is also

used to detect the pathogen.

It is necessary to differentiate from furuncle, carbuncle, erysipelas, in particular from the bullous form. Pulmonary (inhalation) anthrax is differentiated from the pulmonary form of plague, tularemia, melioidosis, legionellosis and severe pneumonia of other etiologies.

It affects more adults than children and men than women.

Anthrax sepsis

After lymphohematogenous spread of B. anthracis from the primary lesions (skin, intestinal tract, and lungs), sepsis develops. Clinical features include fever, toxemia, and shock, followed by death in a short time.

In the differential diagnosis, sepsis caused by other bacteria should be considered. The final diagnosis is made after isolation of B. anthracis from the primary lesions and blood culture.

Prognosis

Before the introduction of antibiotics, the mortality rate in the skin form reached 20%, with modern antibiotic treatment started on time, it does not exceed 1%. In pulmonary, intestinal and septic forms, the prognosis is unfavourable.

Complications of anthrax can include anthrax sepsis, purulent meningitis, purulent toxic damage to the kidneys and liver, and intestines.

Preventive measures

Prevention consists in reducing and eliminating the incidence of the disease among pets.

Prevention should be carried out as soon as possible after possible infection (up to 5 days). In these situations, antibiotics are used - orally phenoxymethyl-penicillin 1.0 g - 2 times a day for five days or tetracycline 0.5 g - 2 times a day for five days. It is allowed to use ampicillin 1.0 g - 3 times a day, oxacillin 0.2 g - 1 time a day, rifampicin 0.3 g - 2 times a day.

Vaccines are used to create active artificial immunity to the anthrax pathogen. The founder of the development of a live anthrax vaccine is L. Pasteur.

Ascaridosis

Ascaridosis is a helminthic disease that has been known since ancient times in the population of countries with temperate, warm and hot climates with sufficient humidity throughout the year. Ascaridosis is the most common helminthic disease, spreading all over the world. It is rare in countries with dry climates and is absent above the Arctic Circle.

Etiology. The causative agent of ascariasis is a roundworm - human hookworm (Ascaris lumbricoides). Adults have a spindle-shaped form.

Epidemiology.

Humans, whose intestines are parasitized by female and male roundworms, are the only source of infection. A mature female can lay up to 245,000 eggs per day, and both fertilized and unfertilized eggs can be laid.

Pathogenesis.

Larvae hatch from mature eggs ingested by humans in the small intestine, penetrate the intestinal wall and penetrate blood capillaries, then hematogenously migrate to the liver and lungs. In addition to the intestines, liver and lungs, hookworm larvae were found in the brain, eye and other organs. They feed intensively on blood serum and red blood cells. In the lungs, the larva actively enters the alveoli and bronchioles, moves through the small and large bronchi with the help of ciliated epithelium to the oropharynx, where sputum with

larvae is swallowed. Once in the intestine, the larva reaches sexual maturity within 70-75 days. The lifespan of an adult roundworm reaches one year, after which it dies and is excreted in the feces.

Symptoms and course. Clinical manifestations of ascariasis depend on the localization of the parasites and the intensity of the invasion. In the clinical course of ascariasis, two phases are distinguished - early (migratory) and late (intestinal). The first phase coincides with the period of larval migration, while the second is due to the parasitism of helminths in the intestines and possible complications.

Complication.

A frequent complication of ascariasis is intestinal obstruction, which is caused by the closure of the intestinal lumen with a ball of ascaris or due to a violation of the neuromuscular regulation of the tone of the intestine. A serious complication of ascariasis is the penetration of helminths into the bile ducts and gall bladder. Jaundice occurs in cases of cholangiohepatitis and mechanical blockage of the common bile duct with ascaris. The temperature during the development of complications can be septic in nature with chills. As a result of joining a bacterial infection, purulent cholangitis and multiple liver abscesses often occur, which in turn can be complicated by peritonitis, purulent pleurisy, sepsis, and abscesses in the abdominal cavity. Penetration of roundworms into the ducts of the pancreas causes acute pancreatitis. Their entry into the appendix causes appendicitis or appendicular colic without inflammatory manifestations.

Diagnosis and differential diagnosis.

X-ray picture of infiltrates can simulate tuberculosis, pneumonia, lung tumor. The main difference between infiltrates in ascariasis is their rapid disappearance without any residual effects. Similar infiltrates can be found in other helminth infections - hookworm and strongyloidosis.

The reliable establishment of ascariasis in the first phase is based on the detection of ascaris larvae in sputum and the establishment of immunological reactions that detect specific antibodies in the blood of patients. In the intestinal stage of the disease, the main method is examination of feces for ascaris eggs.

Liver echinococcosis

There are two forms of echinococcosis: cystic (hydatid) and alveolar.

The hydatidic form of echinococcosis is a disease caused by the cystic or larval stage of development of the echinococcal tapeworm Echinococcus granulosus. The main host of the worm is a dog, and intermediate hosts are humans, sheep, and cattle. When worm eggs enter the human body with water and vegetables, they penetrate the wall of the stomach or small intestine and then reach the liver or lungs (the most common sites of damage) via the blood and lymphatic pathways. At the beginning of the parasite's development in the human body, it is filled with fluid; it looks like a colourless bubble with a diameter of about 1 mm, which increases in size over time. The wall of the hydatid consists of an inner (germinative) and outer (chitinous or cuticular) membrane. Externally, such an echinococcal cyst has a dense fibrous membrane, which is the result of the reaction of the liver tissue in response to the presence of the parasite. This membrane is very dense and almost inseparable from the healthy liver parenchyma, but can be separated from the chitinous membrane. In more than 80% of patients, the right lobe of the liver is affected, and in 1/2 of patients, multiple cysts are detected.

Clinic and diagnostics: for a long time (sometimes for many years), starting from the

moment of infection, there are no “clinical signs of the disease, and the person feels practically healthy. The most common complications of the hydatidiform form of echinococcus are jaundice, rupture of the hydatid cyst, suppuration of the hydatid cyst. The most serious complication is perforation of the cyst into the free abdominal cavity. Symptoms of anaphylactic shock and widespread peritonitis occur. In a complete blood count, eosinophilia is often detected (up to 20% and above). For diagnostics, the Casoni skin test with sterile echinococcal bladder fluid is used. The mechanism of this test is similar to the reaction to tuberculin in tuberculosis. The Casoni test is positive in 50% of patients. Approximately 1 year after the death of the parasite, the reaction becomes negative. Ultrasound echolocation and computed tomography are the most reliable and simple. Laparoscopy and angiography are widely used among invasive methods of examination.

Treatment: There is no single drug that has a therapeutic effect on cystic forms of echinococcosis. The optimal treatment is echinococcectomy. In case of large cysts located in the liver tissue, this method is fraught with damage to large vessels and bile ducts. The most commonly used method is removal of the cyst with its germinal and chitinous membranes after a preliminary puncture of the cyst cavity, with suction of its contents. This technique allows avoiding cyst rupture and dissemination of the parasite during cyst extraction. After removal of the cyst, the fibrous membrane is treated with a 2% formalin solution and sutured with separate sutures from the inside (capitonage). If it is not possible to suture the cavity, tamponade over its omentum is used. In case of suppuration of the cyst contents after the main stage of the operation, the remaining cavity is drained.

Treatment of pulmonary echinococcosis

Treatment of pulmonary echinococcosis is only surgical. The optimal operation, which should be sought in all cases, is an “ideal echinococcectomy” - this is the removal, exfoliation of the parasite in a chitinous capsule from the fibrous capsule without violating its integrity (without opening the bubble).

The following procedures can be performed:

- - echinococcectomy after preliminary suction of the contents of the echinococcal cyst. With this method, after fencing off with napkins, the cyst is punctured with a thick needle, the contents are sucked out of it and the fibrous capsule is dissected.
- - Lung resection for echinococcus is performed mainly in case of extensive secondary inflammatory processes or in combination with other diseases requiring lung resection**.**

Crohn's disease

Crohn's disease is a chronic recurrent disease, a specific feature of which is segmental transmural granulomatous inflammation of any part of the digestive tract from the oral cavity to the anus. Most often, the small and large intestines are affected simultaneously (ileocolitis - 40-55%) and the anorectal region (30-40%). Somewhat less commonly, the disease is limited to the small (25-30%), large (20-25%) or rectum (11-26%) intestine, in 3-5% of cases the upper parts are affected: esophagus, stomach and duodenum.

The prevalence in the world is 50-70 cases per 100 thousand people and has increased several times in recent decades. The highest incidence is observed in the Scandinavian countries. The first onset of the disease usually occurs at the age of 15-35 years. Women and men are equally affected.

Etiology

Not known to date.

Clinical picture

Polymorphism of clinical symptoms, gradual onset of the disease, similarity of the

clinic to other inflammatory bowel diseases. The characteristic symptoms of the disease are persistent or nocturnal diarrhea, abdominal pain, weight loss, fever and rectal bleeding. Rarely does the disease begin acutely, it usually has a fulminant course and even debuts with toxic **Megacolon.**

The main factor affecting the clinic and course of the disease is the localization of the lesion. Most often, the cecum and colon are involved in the inflammatory process, which can be complicated by intestinal obstruction, inflammatory infiltrates and abscesses.

The long course of CD can also be complicated by gastrointestinal adenocarcinoma and rarely lymphoma.

Clinical classification

By localization of the process:

- terminal ileitis,
- ileocolitis,
- predominant lesion of the large intestine,
- damage to the upper parts of the gastrointestinal tract (esophagus, stomach, small intestine).

By form:

• inflammatory,
• fibrostenotic,
• with the formation of fistulas.

By stages of the disease:

• active,
• remission

By degree of severity:

- severe form:
- medium form:
- light form

The degree of CD activity is determined by the Best index

Diagnostics

Physical examination methods

• survey - chronic diarrhea (sometimes nocturnal), abdominal pain, weight loss, fever, presence of blood in stool;
• examination - palpable compaction of the intestine (most often in the right lower quadrant of the abdomen), perianal fissures, fistulas and abscesses.

Extraintestinal symptoms:

• inflammatory eye diseases (iridocyclitis, episcleritis, etc.);
• arthritis (large joints are affected);
• lesions of the skin and mucous membranes (erythema nodosa, etc.);
• kidney damage (appearance of oxalates, obstructive hydronephrosis);
• liver damage (fatty hepatosis, hepatitis);
• inflammatory manifestations (B12 deficiency anemia, amyloidosis, hypoalbuminemia, electrolyte deficiency).

Complication:

• intestinal obstruction;
• occurrence of fistulas, abscesses, cracks, intestinal strictures;
• •toxic dilatation of the intestine (very rare);

- bowel cancer (less common than ulcerative colitis).

Instrumental and other diagnostic methods

- endoscopic examination with morphological examination of biopsies (carried out in all cases to verify the diagnosis - characteristically a focal, asymmetric, transmural granulomatous inflammatory lesion of any part of the intestinal tube in the "cobblestone" type);
- morphological study of biopsies - reveals specific granulomatous inflammation.
- X-ray examination - irrigoscopy and passage of barium through the small intestine (carried out to determine the prevalence of the process and the presence of complications - fistulas, intestinal obstruction, etc.);

Treatment

If indicated, surgical treatment:

- Complications of CD (intestinal bleeding, intestinal obstruction with signs of obstruction, abscesses, fistulas), grade II-III dysplasia, malignization, growth and development retardation in children; refractoriness of CD to drug therapy (including steroid-dependent course). In contrast to UC, in the chronic course of CD, surgery is necessary in most cases. Approximately 70% of patients with CD undergo surgery at least once during their lifetime.

Criteria of treatment efficiency

Elimination (reduction) of disease symptoms and achievement of clinical, laboratory and endoscopic remission (the effectiveness of aminosalicylates is evaluated on the 14th-21st day of therapy, corticosteroids - on the 7th-21st day, azathioprine - after 2-3 months).

Full recovery does not occur, although with adequate treatment, patients can live a full life.

Hodgkin lymphoma

Hodgkin lymphoma - it is a tumour disease of the lymphatic system.

Lymphogranulomatosis occurs 3 times more often in families where such patients were already registered, compared to families where they were not.

Causes of lymphogranulomatosis

The causes of lymphogranulomatosis have not been finally clarified. Some experts believe that lymphogranulomatosis is associated with the Epstein-Barr virus.

Manifestations of lymphogranulomatosis

Manifestations of lymphogranulomatosis are very diverse. Starting in the lymph nodes, the painful process can spread to almost all organs, accompanied by pronounced manifestations of intoxication (weakness, lethargy, drowsiness, headaches). The first manifestation of lymphogranulomatosis is usually an increase in lymph nodes, or in 60-75% of cases, the process begins in the cervical-supraclavicular lymph nodes, a little more often on the right. Enlarged lymph nodes are mobile, not fused to the skin, and in some cases painful. In some patients, the disease begins with an increase in mediastinal lymph nodes. In isolated cases, the disease begins with an isolated lesion of the colo-aortic lymph nodes. The patient complains of pain in the lower back, which occurs mainly at night.

The most frequent localization of lymphogranulomatosis is lung tissue. A tumor in the lymph nodes of the mediastinum can grow into the heart, esophagus, and trachea.

Human bones are as common as lung tissue, the localization of the disease in all variants of the disease. Vertebrae are most often affected, then the sternum, pelvic bones, ribs, less often - tubular bones.

Involvement in the bone process is manifested by pain, radiological diagnosis is usually delayed. In isolated cases, a bone (sternum) lesion may be the first visible sign of

lymphogranulomatosis.

Liver damage is detected late due to the great compensatory capabilities of this organ. There are no characteristic signs of specific liver damage.

The gastrointestinal tract, as a rule, suffers secondarily in connection with the compression or germination of the tumour from the affected lymph nodes. However, in some cases there is a lymphogranulomatous lesion of the stomach and small intestine. The process usually affects the submucous layer, ulcers are not formed.

Diagnosis of lymphogranulomatosis

The morphological diagnosis can be considered reliable only if lacunar histiocytes are present in the histological variant. Histological analysis not only confirms and establishes the disease, but also determines its morphological variant.

Treatment of lymphogranulomatosis

Modern methods of treatment of lymphogranulomatosis are based on the concept of curability of this disease. While lymphogranulomatosis remains a local lesion of several groups of lymph nodes (stage 1-2), it can be cured by radiation. The results of long-term use of polychemotherapy "up to the limit of tolerance of healthy tissues" allow us to assume treatment in the case of a widespread process.

Chemotherapy is given at the time of diagnosis. Radiation therapy is also used. Many hematologists believe that it is necessary to combine chemotherapy and radiation therapy. Correct treatment in the first stage can lead to a full recovery.

Chemotherapy and irradiation of all groups of lymph nodes are very toxic. Patients can hardly tolerate treatment due to frequent adverse reactions, including nausea and vomiting, hypothyroidism, infertility, secondary bone marrow lesions, including acute leukemia.

5. Plan and organizational structure of the class.

№1	Preparatory stage	to assess the advanced level of knowledge and skills of advanced level higher education applicants	Questions for control, tests.	Mastery level I-II	15%
№2	Basic stage	formation of professional mastery skills skills and knowledge, curation of patients	professiona l algorithm, treatment scheme, orienting map, physical methods	educational tasks of research, interpretation laboratory and instrumental methods.	65%
№3	Final stage	Control of mastering the material, the level of skills and abilities.	tasks and tests of the final knowledge level	tasks of the rector's control, tests Krok – II	20%

Materials on methodical provision of classes.

6.1. Control questions for the initial level of knowledge:

1. Peculiarities of anatomical spread of infection.
2. The main clinical signs of specific and purulent diseases.
3. Definition, diagnosis, clinic, treatment of specific and purulent diseases.
4. Development of specific and purulent processes in the lungs, liver, kidneys, gastrointestinal tract, brain, in cellular spaces, skin.
5. Specific diseases of the lungs, liver, kidneys, gastrointestinal tract, brain, skin.
6. Specific diseases and immunological responses of the body.
7. Tactics of the surgeon at different stages of the development of purulent tissue disease.
8. Indications for operative interventions
9. Methods of operative interventions depending on the localization of purulent inflammation.

6.2. Orientation map for independent work with literature on the subject of the class.

№	Basic tasks (to learn)	Instructions (to name)
1.	Anatomy and physiology of lungs, liver, spleen, stomach, intestines. Ways of the possible movement of purulent exudate along the anatomical channels of the skin structure.	- structure of lungs, liver, spleen, stomach, intestines, skin. - function of the lungs, liver, spleen, stomach, intestines, skin.
2.	Clinical and objective signs of surgical complications in infectious diseases of the lungs, liver, spleen, stomach, intestines, skin.	- clinical picture: a) surgical complications in infectious lung diseases. b) surgical complications in infectious diseases of the liver, spleen, stomach and intestines, purulent diseases of the skin and subcutaneous tissue; c) features of the clinic of anaerobic infection. d) peculiarities of the clinic of a specific infection. e) features of the clinic of a specific infection with damage to the skin and subcutaneous tissue;
3.	Methods of examination of patients with surgical complications in infectious diseases of the lungs, liver, spleen, stomach, intestines, skin.	- laboratory research; - immunological studies; - bacteriological research; - puncture of infiltrate; - ultrasound examination; - X-ray examination;
		- CT

4.	Features of treatment of patients with surgical complications in infectious diseases of the lungs, liver, spleen, stomach, intestines, skin	- Antibacterial therapy; - Peculiarities of treatment of patients with surgical complications for infectious diseases of the lungs, liver, spleen, stomach, intestines, skin and subcutaneous tissue of the face, neck, limbs, hands and fingers;
5.	Methods of dissection and drainage of purulent cavities in case of surgical complications in infectious diseases of the lungs, liver, spleen, stomach, intestines, skin.	- Peculiarities of autopsy of patients with purulent complications in infectious diseases, lungs, stomach, liver, spleen, intestines, skin and subcutaneous tissue, face, neck, limbs, hand and fingers;
6.	Prevention of surgical complications in infectious diseases of the lungs, liver, spleen, stomach, intestines, skin, and soft tissues.	- compliance with hygiene rules; - consumption of good quality food and drinking water; - prevention of injuries, tissue destruction; - prevention of skin damage; - preventive clutches;

6.3. <u>Situational tasks</u>

1. Patient S., 38 years old, turned to the surgeon with complaints of neck deformity, painless nodes of rounded and irregular shape. The skin above them is of normal color, the fever is up to 39^0C. The most probable diagnosis? The most correct tactics?

A.Cervicallymphadenitis
B.Lymphagranulomatosi
C. Tuberculosis of the neck.
D. Lipomatosis of the neck with suppuration.
E. Manifestation of the metastatic process.
Answer standard: Lymphogranulomatosis.

2. The patient, 40 years old, has several painful isolated infiltrates of an irregular shape with a focus of softening of one of them. Along the edges of this softening, there are several narrow holes from which pus is released. The edges are cut. The fever is palpable. He has been ill for about 14 days since he returned from hunting a wolf. The most likely diagnosis?

A. Anthrax Ulcerated carbuncle.
B. Skin tuberculosis.
C. Lipoma with suppuration.
D. Manifestation of the metastatic process.
E. All rabies manifestations.
Answer standard: Anthrax Ulcerated carbuncle.

3. The patient, 31 years old, had painless hemorrhages in the skin, mucous membranes and nosebleeds, weakness, dizziness after sleeping. The patient has had small and large hemorrhages in the skin and mucous membranes for several months. The most likely diagnosis?

A. Multiple myeloma.
B. Erysipelas.

C. Werlhof's disease.
D. Erysipeloid.
E. Thrombosis of the superior vena cava.

Answer standard: Werlhof's disease.

4. Manifestations of lymphogranulomatosis are very diverse. Starting in the lymph nodes, the process can spread to almost all organs, accompanied by various manifestations of intoxication (weakness, drowsiness, headache). The first manifestation of lymphogranulomatosis is an increase in lymph nodes. Where does this process most often begin?

A. In the lymph nodes of the mediastinum.
B. In cervical-supraclavicular lymph nodes.
C. In the inguinal lymph nodes.
D. In the intestinal lymph nodes.
E. In the lymph nodes of the skin.
Answer standard: In the lymph nodes of the skin

Which of the following can be the cause of infectious diseases?
A. All of the above.
B. Viruses.
C. Bacteria.
D. Fungi.

What factor most often contributes to the development of postoperative infection?
A. All of the above.
B. Low immunity of the patient.
C. Non-observance of asepsis and antiseptics.
D. Prolonged surgical intervention.

What diagnostic method is the “gold standard” for detecting a bacterial infection?
A. Bacteria culture.
B. Ultrasound.
C. Laboratory examination of blood.
D. X-ray.

Which indicator in the general blood test most often indicates the presence of an infection?
A. Increase in leukocyte level.
B. Decreased hemoglobin.
C. Increase in hematocrit.
D. Increased level of platelets.

What infectious disease is most often differentiated from appendicitis?
A. Renal colic.
B. Acute pancreatitis.
C. Peritonitis.
D. Intestinal obstruction.

Which symptom is most specific for acute appendicitis?

A. Localization of pain in the right iliac region.
B. Backache.
C. Diarrhea.
D. Dry mouth.

What is the main treatment for bacterial infections?
A. Antibiotic therapy.
B. Chemotherapy.
C. Antiviral therapy.
D. Vitamin therapy.

What is the first step in treating an infected wound?
A. Surgical treatment.
B. Use of antiseptics.
C. Applying a bandage.
D. Prescribing antibiotics.

Which of the following is a typical complication of purulent inflammation?
A. Abscess.
B. Allergic rhinitis.
C. Arthritis.
D. Diabetic nephropathy.

What is the greatest risk for infections in immunocompromised patients?
A. Sepsis development.
B. The occurrence of allergies.
C. Appearance of hemorrhagic syndrome.
D. Development of diabetes.

Literature:

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