VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Intestinal infections: cholera, typhoid fever, salmonellosis, dysentery, amoebiasis. Viral infections: herpes, cytomegaly, HIV infection. Anthropozoonotic and vector-borne infections. Echinococcosis.

Guidelines for students
III year of medical faculty
for practical exercises in pathological anatomy

Volgograd, 2020

1. **Purpose: To consider the main risk factors leading to the development of bacterial infections. and clinical signs, relevance to the body.**

2. Requirements for the level of the student in the development of the discipline - pathological anatomy.

1. Terms used on the topic “Intestinal infections: cholera, typhoid fever, salmonellosis, dysentery, amoebiasis. Viral infections: herpes, cytomegaly, HIV infection. Anthropozoonotic and vector-borne infections. Echinococcosis ”in the course of pathological anatomy, and the main objects studied by the pathologist, methods of pathological research.

2. The concept of the main risk factors for the development of intestinal infections: cholera, typhoid fever, salmonellosis, dysentery, amoebiasis.

3. The nomenclature and classification of intestinal infections.

4. The essence and basic patterns of intestinal infections.

5. Characteristic morphological differences of viral infections: herpes, cytomegaly, HIV infection.

6. Characteristic morphological signs of anthropozoonosis and vector-borne infections. echinococcosis, the principles of differential morphological diagnosis.

INFORMATION BLOCK

Infection is a biological phenomenon, the essence of which is the introduction into the human body and the multiplication of harmful prokaryotic and eukaryotic organisms in it, followed by the development of various forms of interaction - from asymptomatic carriage of invading agents to severe illness. In practical medicine, the term “infection” refers to an infectious disease, or an infectious process, that is, a complex of pathological changes and reactions to the introduction and reproduction of disease pathogens. Closely related to this is the concept of “infection” (infection) —the ingestion of the causative agent of an infectious disease into cells or tissues of the body. Infection can lead either to the occurrence of an infectious disease or to the carriage of pathogens in the human body (including endocytobiosis) without any signs of the disease. Despite the improvement of living conditions, mortality from infectious diseases remains quite high.

In order to attribute the disease to infections, it is necessary to observe the postulates of the outstanding German bacteriologist Robert Koch (R. Koch, 1843-1910):

• the causative agent of infection should be detected in all cases with this disease, but should not occur in other diseases or in healthy people;

• the pathogen can be isolated or isolated from the patient’s body in a pure culture;

• experimental introduction of a pure microbe culture into an organism that is sensitive to this pathogen causes this disease in an experimental animal.

When characterizing the pathogen, its virulence is taken into account - a measure of the pathogenic ™ pathogen ™ in relation to a given organism, which includes infectivity (ability to penetrate into the body), invasiveness (ability to spread in the organism) and toxicity.

Parasites are any organisms belonging either to prokaryotes or eukaryotes, i.e. viruses, microbes, protozoa, fungi, helminths, etc. that use another organism as a habitat and (or) a food source (in particular, the human body), causing harm in most cases. In such a broad interpretation, the concepts of "parasite" and "pathogen" are almost equal. Parasites (pathogens) are obligate (able to exist, or parasitize, only being in another organism) and optional, that is, capable of existing both inside the body (including inside its cells) and outside it. In practical medicine, the term "parasite" often refers only to eukaryotic organisms, considered separately from viruses and bacteria. They are involved in parasitology, which studies parasitic diseases caused by unicellular and multicellular eukaryotes. There are endoparasites living inside the cavities, tissues and cells of the macroorganism, and ectoparasites living on the surface of the body.

Infection mechanisms

Respiratory tract. Daily, city dwellers inhale an average of about 10,000 micro-beans, including viruses, bacteria and fungi. Alveoli reach only particles of 5 microns or less. They are attacked and, as a rule, absorbed by alveolar macrophages or neutrophils, attracted by cytokines. This purification system is normally quite effective, but its role can be reduced by smoking, secretion of a viscous substrate with hereditary cystic fibrosis, aspiration of gastric contents and traumatic intubation. It also "weakens" with viral infections. Some viruses of the respiratory group (for example, influenza pathogens) possess hemagglutinins, which attach carbohydrates to the surface of the epithelium and thereby prevent mucociliary clearance, that is, cleansing with mucus and cilia. Bacteria are found (from the hemophilic, pertussis, and pertussis groups) that produce toxins that paralyze the activity of epithelial cilia. Tubercle bacillus settles in the alveoli due to its resistance to the killer action of inactive macrophages.

Viruses can spread from cell to cell by fusion of the latter or by axon transport (poliovirus). They are also able to penetrate the bloodstream and move with the help of either vagrant macrophages (HIV-1) or red blood cells (the causative agent of tick-borne Colorado fever).

Thus, in some cases, the most important signs of the infection process are formed and appear in areas remote from the gates of infection. So, measles and chickenpox viruses enter the body through the respiratory tract, but the first signs of the disease appear in the form of a skin rash.

Intestinal lesions can be caused by almost any bacteria. The most common causative agents of intestinal infections are bacteria of the Enterobacteriaceae family. The latter includes the genera Shigella (the causative agent of dysentery), Salmonella (the causative agent of typhoid fever and other salmonellosis), Yersinia (the causative agent of yersiniosis), Esherichia (the causative agent of Escherichiosis, or coli infection) and others. The causative agent of cholera is the cholera vibrio (Vibrace family).

For the occurrence of any infectious disease, including bacterial intestinal infection, it is necessary:

- infection (the infecting dose may be small - from 10 bacterial bodies, for example, with dysentery), the most important is the fecal-oral route of infection;
- overcoming the mechanisms of combating infection in the mucous membranes of the gastrointestinal tract; in this case, insolvency of barriers, insufficiency of the phagocytic system, as well as specific immunity mechanisms (primarily IgA) are important.

Among the barrier mechanisms, the most important role is played by adequate pH and concentration of enzymes of the stomach and intestines, as well as colonization resistance. The latter is mainly associated with anaerobic bacteria that form a continuous biolayer on the surface of the intestinal mucosa. These microorganisms compete with pathogenic microbes for receptor fields on the surface of epithelial cells. In addition, the normal intestinal microflora produces antibiotic-like substances and induces the activation of specific immune mechanisms. In numerous experiments, the preliminary administration of antibiotics significantly reduced the dose of the pathogen necessary for the development of infection.

Dysentery

Dysentery is caused by bacteria of the genus Shigella. It includes 4 species: S. dysenteriae, S. flexneri, S. sonnei u S. boydii. Similar clinical and morphological manifestations of the disease can cause enteroinvasive Escherichia coli (Esherichia coli), however, these diseases should be classified as coli infection (Escherichiosis), and not dysentery.

Shigella multiply in the intestinal lumen (according to some data, also in the cytoplasm of epithelial cells). The dissemination of shigella occurs predominantly on the canal, lymphogenous and hematogenous dissemination are uncharacteristic.

With dysentery, to one degree or another, all parts of the gastrointestinal tract are affected (sometimes gastroenterocolitis develops). The most severe changes in most patients occur in the distal colon (proctosigmoiditis). Intestinal and extraintestinal lesions are caused by toxins of pathogens. Shigella dysenteriae has the most pronounced toxic properties, Shigella flexneri is less, Shigella sonnei is even less. In the same sequence, the depth and severity of structural changes in the intestine decreases. The

occurrence of tenesmus is determined by toxic damage to the intramural nervous system of the intestine.

In the colon, several types of changes (forms of lesions) are distinguished, with the development of more severe changes, these forms successively replace each other (stages): catarrhal colitis, fibrinous colitis, ulcerative colitis, ulcer healing. If the disease is caused by less pathogenic forms of Shigella (sonnei), then the development of fibrinous inflammation and ulcers occurs only when a secondary infection is stratified. Superinfection is most often caused by staphylococci or Pseudomonas aeruginosa, as well as fungi.

Catarrhal colitis. In the small and large intestines with this colitis, semi-liquid and gruel-like masses with a lot of mucus and streaks of blood are found. The mucous membrane is swollen (edematous), hyperemic, with overlapping mucus, sometimes slight superficial erosion at the tops of the folds. Intestinal changes are expressed unevenly (between the areas of the altered mucous membrane there are zones without clearly visible changes). Microscopically observed hyperemia and hypersecretion of mucus, focal and larger hemorrhages. Later, lymphoplasmacytic infiltration appears, the intensity of which increases as the exudative reactions subside. Leukocyte infiltration - from moderate to pronounced. In the intramural nerve plexuses (submucosal - Meissner and intermuscular - Auerbach) vacuolization and necrosis of nerve cells, decay of nerve fibers are observed.

Fibrinous colitis. Overlays can take the form of tender flakes that can easily separate from the surface of the intestine (croupous inflammation), or more dense rough gray or gray-brown films with a yellowish or greenish tint, often replacing significant areas of the mucous membrane that are tightly fixed to the intestinal wall (diphtheria inflammation). The film with fibrinous inflammation consists of fragments of necrotic tissue, fibrin and white blood cells. In the wall of the intestine, hyperemia, hemorrhages, diffuse leukocyte infiltration are noted. Sometimes colitis can have the nature of phlegmonous inflammation, against the background of pronounced plethora in the vessels of the intestine, stasis and fresh blood clots can be observed, in some cases with rapidly occurring.

Ulcerative colitis. Films (see above) usually do not tear into the lumen, but dissolve (melt), exposing the ulcerated surface of the mucous membrane. Ulcers have an irregular shape, their bottom is yellowish or reddish. They are usually shallow and do not extend beyond the mucous membrane, but can also be deep. In areas of ulceration, hyperemia of the intestinal wall is expressed. The mesenteric lymph nodes are enlarged, with areas of hemorrhage.

In some cases (especially in children) they describe a peculiar ulcerative lesion of the intestine. Against the background of catarrhal inflammation, hyperplasia of the lymphoid follicles of the intestine develops, later the latter undergo necrosis and purulent fusion. As a result, ulcers with overhanging edges (follicular ulcerative colitis) form in the intestine at the site of lymphoid follicles.

The abatement of inflammation and the regeneration of the mucous membrane during catarrhal inflammation develop quite rapidly. After 3-4 weeks, the structure of the mucous membrane does not differ from normal. With destructive lesions, complete anatomical recovery is achieved 5-6 months after the onset of the disease.

During regeneration, defects are filled with granulation tissue, which epithelizes and matures into fibrous tissue. If destructive changes are common, regeneration is slow. Sores heal slowly and with follicular-ulcerative colitis. During the formation of granulation tissue, foci of exudative inflammation can persist for a long time.

Extraintestinal changes result from intracanalicular dissemination of shigella (e.g. pneumonia), toxic effects and dehydration (especially in children). Often, liver damage (fatty degeneration) and kidney damage (tubular dystrophy and intracalillary glomerulonephritis) are often found.

Complications. Deep ulcers can be complicated by bleeding or perforation of the intestinal wall with the development of peritonitis (paraproctitis). Regeneration in conditions of ongoing inflammation with progressive deformation of the mucous membrane can lead to the formation and consolidation of chronic inflammation. In this case, two possible lesions are possible: chronic dysentery (accompanied by the release of shigella) and chronic nonspecific postdysenteric (ulcerative) colitis. The intestinal wall in both forms is thickened due to the growth of connective tissue, mainly in the submucosal layer of the mucous membrane. Defects with smooth edges and a grayish bottom are found in the mucous

membrane. Crypts are deformed, there are areas of atrophy. The defeat of the intestine is mosaic. Chronic inflammation can lead to the development of cicatricial stenosis of the colon.

Typhoid fever.

Typhoid fever is the most serious disease caused by Salmonella; its causative agent is Salmonella typhi (abdominalis). The pathogenesis of this disease remains not entirely clear. Salmonella multiply in the lumen of the intestine, penetrate the wall of the intestine and spread lymphogenously and hematogenously. All parts of the gastrointestinal tract are affected, however, the most severe changes develop in the small intestine. Early changes consist of a picture of catarrhal inflammation of the mucous membrane and peculiar changes in group lymphoid follicles of the small intestine.

Changes in the mucous membrane are most clearly defined in the terminal ileum. In the intestinal lumen are semi-liquid stool with an admixture of mucus (feces in patients looks like pea soup). The mucous membrane is full-blooded, swollen. Group and solitary lymphoid intestinal follicles are swollen, juicy, grayish-red in the section. The tissue of regional lymph nodes also looks like. The surface of the lymphoid formations of the wall of the small intestine [group lymphatic follicles (Peyer's plaques)] has a furrowed appearance. The surface features of Peyer's plaques made it possible to characterize these changes as "brain swelling."

Microscopic examination in the stage of cerebral swelling reveals plethora and swelling of the intestinal mucosa. There are few lymphocytes in the lymphoid tissue of the mucous membrane; they are replaced by larger cells with an oxyphilic cytoplasm. These cells are located in the lymphoid tissue diffusely or in the form of focal clusters ("typhoid nodules"). There are no causative agents of typhoid fever in these cells. Cerebral swelling of group lymphatic follicles (Peyer's patches) may be the only step in the morphogenesis of intestinal lesions in this infection. At least in some patients, the inflammation subsides, and the lymphoid apparatus of the intestine takes on a normal form. In a number of cases, however, destructive changes develop that are localized in the region of the brain-shaped Peyer's plaques. In these cases, there are 4 more stages of local changes in the intestine: necrosis, the formation of ulcers and clean ulcers, healing of ulcers. The duration of each stage is about 1 week. The reason for the development of destruction is one of the most controversial issues in the pathogenesis of typhoid fever. It is generally accepted that necrosis is allergic, but evidence of this situation has not yet been obtained.

At the stage of necrosis, tissue necrosis of lymphoid follicles occurs, starting from their surface sites. Necrosis most often does not reach the edges of plaques and follicles, but can spread beyond them. Necrotic areas are dirty gray, when saturated with bile pigments, greenish. The most common necrotic changes are plaques located at the ileocecal valve (Bauginia valve).

The stage of ulceration is characterized by the rejection of necrotic masses and the formation of ulcerative craters in the affected plaques and follicles. The edges of such craters hang over the resulting defects and have a “dirty” appearance due to the presence of detritus and exudate in them.

At the stage of clean ulcers, mucosal defects are usually shallow, with low edges. Necrotic masses are absent in them.

Then follows the healing of ulcers. A thin layer of granulation tissue forms at the bottom of the ulcer, and regenerating epithelium grows along with the edges of the defect. After healing, usually there are no gross cicatricial changes in the intestinal wall; the absence of lymphoid follicles and grayish pigmentation at the site of the former ulcers attract attention.

Extraintestinal manifestations of typhoid fever are associated with extensive dissemination of salmonella and toxic tissue damage. At the end of the 1st - beginning of the 2nd week of the disease, a typhoid rash (exanthema) appears. It is localized on the skin of the body, has a rose-papular character. Microscopy reveals plethora, edema, and lymphohistioplasmocytic vascular wall infiltration. In scrapings from the elements of the rash, the pathogen is found. In hematopoietic and lymphoid organs, diffuse and focal (in the form of granulomas) proliferation of macrophages and / or reticular cells are noted. The spleen is enlarged 3-4 times, in the section red, full-blooded. The liver is enlarged. Its fabric is yellowish, dull. Microscopic examination shows a picture of protein and fatty degeneration of hepatocytes.

Complications of typhoid fever. Bleeding occurs at the stage of ulceration (usually at the 3rd week of illness), perforation (perforation) of the intestine with the development of peritonitis - at the stage of clean ulcers (most often at the 4th week). Peritonitis may be due to necrosis of the mesenteric lymph nodes. Sometimes, at the 2-3rd week of illness, coagulation of waxy (cencker) skeletal muscle necrosis develops. They have the appearance of dense grayish-yellow foci with greasy sheen, localized most often in the muscles of the anterior abdominal wall.

In addition, the causative agents of typhoid fever after bacteremia can be detected in any organ, causing purulent inflammation. Possible meningitis, nephritis, prostatitis, perichondritis (for example, cartilage of the larynx), periostitis, osteomyelitis, arthritis, etc. Often pneumonia develops. It can have salmonella or a mixed (when another flora joins) etiology. Sometimes an infection-toxic shock occurs. Typhoid sepsis is also possible. At the same time, changes in the intestine can be mild and non-specific for typhoid fever, the disease proceeds in the form of septicopyemia.

Other salmonellosis. There is a large group of intestinal infections caused by salmonella. For humans, more than 700 species (antigenic variants) of salmonella are pathogenic. The most important are S. typhimurium, S. enteritidis. Less common are infections caused by S. paratyphi L, S. paratyphi B, and others.

There are 3 main clinical and anatomical forms of salmonellosis - gastrointestinal, septic and typhoid. Changes in salmonellosis resemble those in typhoid fever, but are much less pronounced.

With the **gastrointestinal form** of salmonellosis, a picture of catarrhal gastroenteritis is observed. In the lumen of the stomach and intestines, a large number of semi-liquid or liquid, often foamy masses with a pungent odor, a significant admixture of mucus are detected. The mucous membrane is edematous, full-blooded, often with hemorrhages and superficial erosion. There is always moderate hyperplasia of the lymphoid tissue of the intestinal wall and regional lymph nodes. In severe cases, inflammation in the intestine becomes hemorrhagic.

In the **septic form**, hematogenous generalization of the infection leads to the formation of numerous foci of purulent inflammation (abscesses) in the internal organs, more often in the liver, lungs and kidneys. Along with salmonella, other microorganisms are often found in these foci.

With **the typhoid form** of salmonellosis in the intestine and lymph nodes, changes occur similar to those in typhoid fever. However, they are much weaker

Cholera

Cholera is a quarantine infection caused by Vibrio cholerae (the Vibrionaceae family, which includes rods that live freely in fresh and sea water). The pathogenicity of the pathogen is associated with the presence of toxins, mainly exotoxin (cholerogen). By binding to enterocyte membranes, cholerogen stimulates intestinal secretion, which is accompanied by a massive loss of water and electrolytes (diarrhea, vomiting). With increasing dehydration, the main manifestations of cholera are associated. Systemic signs of an infectious disease are mild, there is no fever. There are 4 clinical and anatomical stages of the course of infection: enteritis, gastroenteritis, algide and typhoid.

The intestines are overflowed with a colorless or pinkish liquid that looks like a muddy soup (rice broth) with the smell of fish or raw grated potatoes. Bile is watery, transparent. With a macroscopic examination, pronounced edema and plethora are observed in the wall of the small intestine and stomach. There are minor hemorrhages. The intestinal epithelium is initially preserved, and as the swelling of the mucous membrane increases, it undergoes dystrophic changes and subsequently sloughs in places in the lumen. The stroma of the mucous membrane is infiltrated by lymphocytes and plasma cells. White blood cells are also found, they are usually not numerous. These changes correspond to the phase of cholera enteritis and gastroenteritis. Similar, but less severe changes are observed in the colon. A very severe course of cholera is accompanied by a pronounced paresis of the stomach and intestines, while vomiting and diarrhea may be absent ("dry" cholera).

Extraintestinal changes are due to increasing exsicosis and blood clotting. With the development of the algide phase, the face acquires a characteristic appearance - sharpened features, sunken eyes, earthy-colored skin (fades Hyppocratica). Contraction of the muscles of the limbs gives the body the

characteristic pose of a “boxer” (“gladiator”). The skin is dry, wrinkled ("laundresses hand"), skin folds do not straighten. In the dead, rigor mortis is pronounced and is not allowed for a long time. Blood is thick and dark ("currant jelly").

Complications. With a loss of fluid in a volume of 10% or more of body weight, hypovolemic shock develops. Hypo-potassiumemia leads to heart rhythm disturbances. Blood thickening, increasing its viscosity, hypotension cause the occurrence of cortical necrosis of the kidneys and renal failure. Cholera typhoid should also be considered as a complication of cholera. It is characterized by the development of diphtheria inflammation of the colon and is associated with the accession of a secondary (usually staphylococcal) infection.

3. Lesson plan

To study the following macropreparations, to describe them according to the scheme for the description of macropreparations.

Macropreparations.

1. To study fibrinous colitis by a macroscopic picture. Describe the macropreparation "Fibrinous colitis in dysentery." Characterize the thickness of the intestinal wall, localization, surface, color and density of attachment of the fibrinous film.
2. To study the cerebral swelling of group follicles of the small intestine with typhoid fever according to a macroscopic picture. Describe the macropreparation "Brain swelling of group lymphoid follicles of the small intestine with typhoid fever." Pay attention to size, surface, sectional view of group follicles; condition of the mucous membrane of the small intestine (thickness, color, luster).
3. To study ulcers of the small intestine with typhoid fever according to a macroscopic picture. Describe the macropreparation of "Ulcers of the small intestine in typhoid fever." Characterize to the number, location, shape, size, depth, condition of the edges and bottom of the defects of the mucous membrane of the small intestine.
4. To study liver echinococcosis by macroscopic picture. Describe macropreparation "Echinococcosis of the liver." Characterize the presence of blisters (cysts), in which the echinococcus embryo is located, outwardly resembles a cyst having a two-layer membrane consisting of a germinal and chitinous layer. Pay attention to the size, shape, localization of cysts.
5. Additional macropreparation “Fibrinous colitis in dysentery”. To study fibrinous colitis in dysentery using a macroscopic picture. Pay attention to the wall thickness of the intestine, localization, surface, color and density.

Examine the following micropreparations, sketch them, indicate and mark the pathological changes with arrows, using the atlas of micropreparations.

Micropreparations.

1. To study fibrinous colitis with dysentery according to the microscopic picture. Describe the micropreparation "Fibrinous colitis in dysentery" (hematoxylin and eosin staining). Pay attention to the localization and depth of necrosis, the type and composition of exudate; tissue change; adjacent to the area of inflammation (the cellular composition of the infiltrate, blood vessels, the state of the submucosal layer).
2. To study the cerebral swelling of group follicles of the small intestine with typhoid fever according to the microscopic picture. Describe micropreparation "Brain swelling of group lymphoid follicles of the small intestine with typhoid fever" (stained with hematoxylin and eosin). Pay attention to the state of the layers of the mucous membrane, the size and structure of group follicles; localization and cellular composition of typhoid granulomas.
3. To study the mesenteric lymph node in typhoid fever according to the microscopic picture. Describe the micropreparation "Mesenteric lymph node in typhoid fever" (hematoxylin and eosin staining). Pay attention to the safety of the picture of the structure of the lymph node, the number of lymphocytes, the localization and cellular composition of typhoid granulomas.

4. To study enteritis with cholera according to the microscopic picture. Describe the enteritis cholera micropreparation (hematoxylin and eosin staining). Pay attention to the state of the epithelium of the stroma of the villi, blood vessels, localization and cellular composition of the mucous membrane infiltrate.

5. To study typhoid encephalitis according to the microscopic picture. Describe the micropreparation Typhoid encephalitis (hematoxylin and eosin staining). Pay attention to the presence of typhoid granuloma in the wall of the vessel of the brain (blood vessels, localization and cellular composition). Indicate and mark the pathological changes with arrows.

Solve the following situational tasks using the tutorial.

Situational tasks

Situational task 1.

The death of a 3-year-old child occurred in the infectious diseases hospital on the 6th day of the disease, which began with a rise in temperature to 39 ° C, profuse diarrhea. Bacteriological examination made it possible to isolate Shigella Sonne type from the contents of the intestine. The immediate cause of death with progressive toxicosis was acute renal failure.

1. Give the name of the disease.
2. List the features of the localization of changes in the digestive tract that are characteristic of young children.
3. What are the options for the defeat of the digestive tract by type of inflammation.
4. Explain how the development of acute renal failure syndrome occurs.

Situational task 2.

An elderly patient was admitted to the intestinal infections department with abdominal pain, tenesmus. At the first sign of intestinal upset, she began to take high doses of antibiotics at home. Bacteriological examination of secretions from the intestine gave a negative result. When sigmoidoscopy revealed: the mucous membrane of the rectum and lower sigma is swollen, hyperemic, with spot hemorrhages and ulcerative defects on the tops of the folds, the form of ulcers is diverse. On the 5th day of hospitalization, signs of paraproctitis appeared.

1. Give the name of the disease.
2. What is the stage of the disease and the mechanism of ulcer formation?
3. Explain the cause of the development of paraptctitis.
4. Assume the cause of the negative bacteriological test result.

Situational task 3.

A 52-year-old patient was delivered to the infectious diseases clinic in the 2nd week after the onset of diarrhea and an increase in body temperature to 40 ° C. An exanthema typical of typhoid fever was detected. The diagnosis is confirmed bacteriologically.

1. What is the form of exanthema.
2. Describe macroscopic changes in the intestine, taking into account localization in the classical course of the disease.
3. Clarify the nature of microscopic changes in the intestinal wall at this stage. 4. List the possible intestinal complications of the disease.

Situational task 4.

A 40-year-old patient who was in the focus of an outbreak of cholera was hospitalized with typical manifestations of the disease, died in the third period (stage) of infection.

1. What is the causative agent of the disease.
2. List the external signs of the disease at this time and the clinical figurative names of these signs.
3. Give the name of the exotoxin secreted by the pathogen.
4. Describe the lesion of the gastrointestinal tract.

Situational task 5.

A patient who, due to circumstances, did not receive timely medical care in the outbreak of cholera, died in the clinical picture of acute renal failure.

1. Determine the approximate term (period) of death.
2. Specify the mechanism of development of pathological changes in the kidneys (pathogenesis).
3. List the extrarenal changes in the body of the deceased.

Answer the following questions of the current test control.

Select all correct answers.

1. The enterobacteriaceae family includes:
a) Salmonella typhi,
b) Shigella flexneri,
c) Yersinia pseudotuberculosis,
490
d) Vibrio cholerae,
e) Esherichia coli.

Select all correct answers.

2. Characterization of colonization resistance:
a) is associated mainly with aerobic bacteria,
b) forms a component of the barrier of the mucous membranes,
c) is disturbed with dysbiosis,
g) sensitive to the introduction of antibiotics,
e) based on competition for free cellular receptors.

Choose one correct answer

3. The causative agents of dysentery:
a) shigella
b) salmonella
c) yersinia,
d) Escherichia,
e) vibrios.

Select all correct answers.

4. Stages of colitis in dysentery:
a) catarrhal,
b) fibrinous,
c) ulcerative,
g) follicular-ulcerative,
e) purulent hemorrhagic.

Choose one correct answer

5. The intestine, which is most often affected by dysentery:
a) blind and direct,
b) colonic and sigmoid,
c) sigmoid and direct,
d) blind and sigmoid.

Choose one correct answer

6. The kind of Shigella that causes the most severe bowel damage:
a) Flexneri,
b) Dysenteriae,
c) Sonnei.

Choose one correct answer

7. The dissemination of dysentery pathogens from the primary focus most often occurs:
a) perineurally,
b) intracanalicular
c) lymphogenous,
g) hematogenous.

Select all correct answers.

8. Complications of dysentery:
a) fatty degeneration of the liver,
b) pneumonia,
c) paraproctitis,
g) chronic colitis,
e) purulent meningitis.

Choose one correct answer

9. A child of 5 years old in the summer has a fever, frequent painful stools, and in the feces an admixture of mucus with streaks of blood. Conclusion:

a) dysentery,
b) typhoid fever,
c) salmonellosis,
g) pseudotuberculosis,
d) cholera.

Choose one correct answer

10. Characteristic for children colitis with dysentery:

a) catarrhal,
b) fibrinous,
c) follicular-ulcerative,
g) ulcerative,
e) phlegmonous.

Select all correct answers.

11. The formation and type of ulcers with dysenteric ulcerative colitis:

a) are formed during the melting of fibrinous films,
b) usually do not go beyond the mucous membrane,
c) are formed at the site of lymphoid follicles,
g) have an oval shape,
e) hematin hydrochloride (hydrochloride) is determined at the bottom.

Choose one correct answer

12. The causative agents of typhoid fever:

a) shigella
b) salmonella
c) yersinia,
d) Escherichia,
e) vibrios.

Choose one correct answer

13. Intestine, in which with typhoid fever primary damage occurs:

a) direct
b) sigmoid,
c) duodenal,
d) iliac,
d) colonic.

Select all correct answers.

14. Stages of intestinal damage in typhoid fever:

a) cerebral swelling,
b) follicle formation,
c) necrosis,
g) the formation of ulcers,
e) clean sores.

Set the correct sequence

15. Stages of enteritis in typhoid fever:

a) necrosis,
b) the formation of ulcers,
c) cerebral swelling,
g) healing of ulcers,
e) clean sores.

Select all correct answers.

16. Complications of typhoid fever:

a) bleeding,
b) intestinal perforation,
c) coagulation (Tsenker) necrosis,
g) chronic colitis,
d) perichondritis of the larynx.

Choose one correct answer

17. A 30-year-old man fell ill acutely. Diagnosed with intestinal infection. At the 4th week of the disease, against the background of better health, a picture of peritonitis suddenly developed. In the operation, perforated holes were found along the free edge of the small intestine. Conclusion:

a) dysentery,
b) typhoid fever,
c) salmonellosis,
g) pseudotuberculosis,
d) cholera.

Select all correct answers.

18. The composition of granulomas with yersiniosis:

a) epithelioid cells,
b) white blood cells,
c) plasma cells,
d) Pirogov-Langhans cells,
e) macrophages.

Select all correct answers.

19. Extraintestinal manifestations of yersiniosis:

a) orchitis
b) hepatitis
c) polyarthritis,
g) scarlet fever-like exanthema,
d) flushing of the skin of the feet and hands.

Select all correct answers.

20. Complications of yersiniosis:

a) intestinal phlegmon,
b) gangrene of the intestine,
c) paraproctitis,
g) adhesive obstruction,
d) perforation of the intestinal wall.

Choose one correct answer

21. In the summer, an outbreak of the disease occurred in one of the Black Sea resorts, the leading sign of which was profuse diarrhea. No fever was noted. Conclusion:

a) dysentery,
b) typhoid fever,
c) salmonellosis,
g) pseudotuberculosis,
d) cholera.

Choose one correct answer

22. The group of quarantine infections includes:

a) cholera,
b) typhoid fever,
c) salmonellosis,
g) pseudotuberculosis,
d) dysentery.

Select all correct answers.

23. Abrupt dehydration (cholera algide) leads to:

a) "the washerwoman's hand",
b) "the face of Hippocrates,"
c) "boxer pose",
d) "raspberry tongue",
d) "blood type currant jelly."

Case

A patient with dysentery, confirmed by the results of a bacteriological study, revealed paraproctitis. After some time, back pain, pyuria, and fever appeared.

Choose one correct answer

24. The reason for paraproctitis:

a) hematogenous dissemination of infection,
b) rupture of the intestinal wall,
c) ulcer perforation,
d) lymphogenous dissemination of infection.

Choose one correct answer

25. Colitis at the indicated stage of dysentery:

a) catarrhal,
b) fibrinous,
c) purulent,
g) follicular
e) ulcerative.

Choose one correct answer

26. Pathological process in the kidneys.

a) glomerulonephritis,
b) pyelonephritis,
c) embolic purulent nephritis,
g) heart attack of the kidneys.

4. *List of recommended literature:*

Basic literature:

1. "Basic pathology" Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

1. "Pathology. Quick Review and MCQs" Harsh Mohan, 2004.
2. "Textbook of Pathology " Harsh Mohan, 2002.
3. "General and Systemic Pathology" Joseph Hunter, 2002.
4. "General and Systematic Pathology " Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2^{th}).
5. "Histology for Pathologist" Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2^{th}).
6. "Histopathology. A Color Atlas and Textbook" Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.
7. "Muir's Textbook of Pathology" Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14^{th}).
8. "Pathology" Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3^{th}).
9. "Pathology Illustrated" Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4^{th}).

10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).
11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).
12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10th).
13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7th).
14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.
15. “Pathology” Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.
16. “Short lectures on pathology” Zagoroulko A., 2002
17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.
18. “General pathology” Dr. Fatma Hafez, 1979.
19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

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