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Department of Infectious diseases

METHODOLOGICAL GUIDE

AMEBIASIS

for students studying in the specialty
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Amebiasis - 2020

This manual contains material reflecting anthroponous intestinal protozoan disease - amebiasis. The manual reflects the etiology, epidemiology, pathogenesis, clinical picture, complications, differential and laboratory diagnostics, treatment. The manual contains test tasks, situational tasks. The proposed structure of the manual helps to highlight the main aspects of the studied nosology.

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Amebiasis

Amoebiasis is a protozoal disease caused by a histolytic amoeba, characterized by a tendency to protracted course, ulcerative lesions of the colon, the development of extraintestinal forms and complications from various organs and systems.

The relevance of studying the topic "Amebiasis" is due not only to the variety of forms and variants of the course, but also to the variety of clinical manifestations of the disease from asymptomatic, mild to severe forms with the development of complications. Amoebiasis should be able to distinguish among other diseases of the gastrointestinal tract, which have similar symptoms with it.

The purpose of the lesson: to learn modern diagnostics of both intestinal and extraintestinal forms of amebiasis for organizing timely adequate therapy, preventing the formation of protracted and chronic variants of the course of the disease and preventing possible complications that are life-threatening.

The student should be able to:

- to learn purposefully and consistently to collect an epidemiological history and anamnesis of the disease in dynamics by giving leading questions and taking into account the epidemiological clinical features of amebiasis;

- to learn to objectively examine a patient with amebiasis, paying special attention to those organs that are more affected by amoebiasis;

- plan a comprehensive examination of the patient, including laboratory, instrumental methods and methods using the existing equipment (ultrasound, radiography, colonoscopy, etc.);

- to learn the timely diagnosis of extraintestinal forms of amebiasis (acute amoebic hepatitis, liver abscess, amebiasis of the lungs, brain, skin);

- independently conduct sigmoidoscopy for patients with amebiasis and be able to read the pathomorphological picture characteristic of intestinal amebiasis;

- be able to take material from the feces of patients for a native smear and the coproscopy technique for detecting protozoa;

- to carry out sigmoidoscopic differential diagnosis of amebiasis with other diseases occurring with the defeat of the large intestine;

- assess the severity of the course of the disease, decipher the diagnosis in accordance with the existing classification;

- to organize specific and pathogenetic therapy in accordance with the deciphered diagnosis;

- to draw up an approximate plan of organizational measures for the prevention of amebiasis.

Etiology.

The causative agent - Entameba histolitica, belongs to the protozoa of the genus Entamoeba of the Sarcodina class. It exists in the form of quadrangular cysts and mononuclear vegetative forms (trophozoites) - precystic, luminal, large vegetative and tissue.

*Precystic form*inactive, has a homogeneous cytoplasm. It is determined in the feces of convalescents after an acute course of intestinal amebiasis and in cyst carriers after taking a laxative. The luminal form is Entameba histolitica, which lives in the lumen of the colon, larger than the precyst. It is found in the feces of patients with an acute form, in convalescents, in chronic recurrent disease, and in semi-formed stools in healthy carriers (or after giving a laxative). A significant number of them in an actively mobile state may indicate the initial stage of amoebic dysentery or an exacerbation of chronic amoebiasis in the recent past. In these cases, Charcot-Leiden crystals may also be found in the preparation. Both of these forms are non-pathogenic and in most cases transform back into cysts as they move through the colon.

In some cases, a large vegetative form is formed from the luminal form, and from it a tissue one. The large vegetative form is the largest. In the endoplasm in the digestive vacuoles, phagocytosed erythrocytes are determined, sometimes in large numbers. Such amoebas are called erythrophages or hematophages. The nucleus is not visible, which is an important differential sign that distinguishes them from similar in size and shape intestinal amoebas. The large vegetative form is found in acute intestinal amebiasis in freshly excreted feces of a sick person (see ex tempore). In addition to amoebas, the preparation may contain crystals of Charcot Leiden (fragments of eosinophils) of a characteristic elongated diamond shape. In a tissue form of amoeba under a microscope in a native preparation, the endoplasm is homogeneous, without any inclusions, the nucleus cannot be distinguished. The tissue form of amoeba differs from other vegetative forms in the fastest and most active movement of its body. The tissue form is found only in acute amebiasis directly in the affected tissues, and rarely in the patient's feces. These two forms are mobile, possess proteolytic enzymes and proteins that determine their virulence.

Standing out with feces into the external environment, vegetative forms quickly die. Cysts are quite stable: they persist in feces for up to 4 weeks, in water - up to 8 weeks, but quickly die when dried. For several days, cysts are capable of eating foods, vegetables, fruits and household items. At a temperature of 45 ° C, they die after 30 minutes, and at a temperature of 85 ° C, after a few seconds. Cysts are exposed to low temperatures (-20-21˚C) for several months. In a solution of mercuric chloride (1: 1000), they die within 4 hours, in a solution of cresol (1: 250) - in 5 - 10 minutes. 1% solution of lysol kills cysts in 1 min. In 5% solution of formalin, their viability lasts up to 5 days. Chlorination of water does not destroy cysts.

Epidemiology.

The reservoir and source of infection are people with acute or chronic amoebiasis, convalescents and asymptomatic cyst carriers. In endemic foci of invasion, in 90% of cases, it manifests itself in the form of prolonged (up to several years) asymptomatic carriage.

Transmission mechanism - fecal-oral :; the main routes of transmission are food (especially with vegetables and herbs), less often water. The household route of transmission (through hands contaminated with amoeba cysts) and sometimes contact (during oral and anal intercourse) are possible.

The natural susceptibility of people is high, the index of contagiousness reaches 20%. In the foci of invasion, clinically pronounced cases of amebiasis are more often observed in middle-aged people; children under 5 years of age rarely get sick.

Amoebiasis is widespread in many countries of the world. According to the WHO, 40-50 million cases of amoebic colitis and liver abscess are registered annually in the world, of which 40,000 - 110,000 cases end in death, which puts amoebiasis in second place after malaria in mortality from protozoal diseases.

Post-infectious immunity is relative and short-term. No specific prophylaxis has been developed.

Pathogenesis.

After swallowing, cysts overcome the acid barrier of the stomach, and then vegetate in the small intestine, successively transforming into a precystic and then into a luminal form. At these stages of development, pathogens are non-pathogenic, the invasion is manifested in the form of asymptomatic carriage. With a slow movement of feces along the cecum and the ascending colon, the luminal forms actively multiply, in most cases they are encystised, and cysts are then excreted with feces, and partially turn into large vegetative forms. Introduced into the intestinal wall, the large vegetative form turns into a tissue one.

Specific amoeba proteins form pores in target cells, inhibit motility and suppress the cytotoxic function of macrophages. Entameba histolitica is able to lyse neutrophils, and the mediators released during this process activate diarrhea and tissue damage. Secretory proteases of the pathogen and enzymes similar to bacterial ones cause tissue degradation. The fact of activation of apoptosis of epithelial cells by amoeba toxins was established. Damage to the mucous membrane and deeper layers of the intestinal wall occurs primarily in the blind, ascending and transverse colon and is accompanied by an inflammatory reaction typical of early invasive amoebiasis. Sub-

sequently, the phenomena of cytolysis of the epithelium, tissue necrosis with the formation of ulcers, and microcirculation disorders increase. Development of pancolitis with a severe course of the disease is possible.

With the disintegration of necrotic areas of the intestine, the concentration of biologically active substances in the blood increases, which causes mild or moderate intoxication.

The proliferation of granulation tissue in the intestinal wall can lead to its thickening and compaction, the formation of the so-called amoebas, narrowing the intestinal lumen, sometimes to complete obstruction. With the formation of deep ulcers, intestinal perforation, peritonitis (with the subsequent development of the adhesive process) are possible.

Extraintestinal amoebiasis is caused by hematogenous dissemination of amoebas along the systems of the portal and inferior vena cava and their subsequent fixation in the internal organs with the development of abscesses or ulcers. Most often, abscesses form in the right lobe of the liver, less often in the lungs, sometimes they can be in the brain, kidneys. The pancreas. Cases of lesions of the pericardium are described. Subphrenic liver abscess can be complicated by purulent fusion of the diaphragm, followed by purulent pleurisy.

Cutaneous amoebiasis occurs when pathogens are excreted with liquid feces and contaminate the skin of the perineum, where erosions and ulcers are formed.

Immunity in amebiasis is determined by the activity of macrophages. the production of secretory Ig A and lymphokines in the colon, as well as the formation of humoral antibodies that persist in the body during convalescence for up to 1 year or more.

The clinical picture.

The incubation period varies from 1 week to 2-3 (sometimes up to 6 and longer) months.

There are 3 main forms of amoebiasis: intestinal, extraintestinal and cutaneous amebiasis.

Intestinal amebiasis develops gradually and can only manifest itself as abdominal discomfort and mild diarrhea. For a mild course of the disease, short-term periods of mushy or liquid stools with mucus are characteristic, 2 to 3 times a day. Abdominal pains are moderate, have a periodic cramping character, or are often absent. The state of health of the patients remains satisfactory, the temperature does not rise. Episodes of diarrhea alternate with periods of normal stool or constipation with flatulence. This condition without treatment lasts from several days to several weeks. Such patients often become cyst carriers.

With moderate severity, the disease proceeds according to the type of food toxicoinfection: the frequency of bowel movements is 5-10 times a day, stool. Usually plentiful. Unlike shigellosis, with amoebiasis, the movement of the contents of the small intestines is not disturbed, the release of tissue fluids and electrolytes into the lumen of the large intestine is not disturbed, therefore, the fecal character of the stool is preserved. Intoxication is not expressed, cramping pains in the lower abdomen, nausea, headache, weakness, low-grade fever. In the absence of treatment, these phenomena gradually subside, and the disease can become chronic.

In severe form, the disease proceeds with pronounced symptoms: the temperature rises to 39 - 40 ° C, may be accompanied by chills, muscle pain, headache, and upset stool. The amount of faeces decreases. And the stool takes on a slimy jelly-like character. Only in rare cases is it possible to observe mucous-bloody stool ("raspberry jelly"). With the defeat of the nerve plexuses of the rectum, pain occurs during bowel movements and tenesmus. The abdomen is soft on palpation, there is pain in the ileocecal region and in the region of the sigmoid colon, rumbling in these areas.

With endoscopic examination, one can see swelling and focal hyperemia of the mucous membrane of the rectum and sigmoid colon, increased mucus production, small nodules of erosion and often ulcers with undermined, raised edges and an uneven sebaceous bottom, containing necrotic masses and surrounded by a belt of hyperemia. In patients with a predominant lesion of the proximal colon, only catarrhal proctosigmoiditis is found during colonoscopy. The mucous membrane, free from ulcers, looks little changed.

Acute manifestations of intestinal amebiasis persist for 4-6 days. Even without specific treatment, general well-being usually improves, the number of bowel movements decreases, and a temporary relief of colitis may occur. However, spontaneous cure does not occur, and the disease takes on a chronic course with a change in periods of exacerbations and remissions. Its duration without specific therapy reaches 10-15 years and often gives complications.

Chronic intestinal amebiasis proceeds in two clinical forms - recurrent and continuous.

In a recurrent form, periods of exacerbation alternate with periods of remission. In the period of exacerbation, patients complain of the appearance of low-grade fever, sometimes minor stool disorder, headache, loss of appetite, abdominal discomfort, general loss of strength, and weakness. On palpation of the abdomen, there is sensitivity or mild soreness in the ileum, rumbling of the intestine, swollen sigmoid colon. Periods of remission do not come for a long time, as a rule, they are short-term and repeated exacerbations are easily provoked by a violation of diet, stress and other acute diseases, especially of the gastrointestinal tract.

With a continuous course, there are no periods of remission, there is a weakening or strengthening of clinical manifestations.

Chronic intestinal amebiasis is accompanied by damage to the cardiovascular and nervous systems, asthenic syndrome, anemia, beriberi, exhaustion, cachexia develop. Patients complain of increased irritability, headache, sleep disturbance. Lability of the pulse, tachycardia, muffling of heart sounds are noted. The size of the liver and spleen does not change. The abdomen is retracted, there is a mild soreness in the iliac regions, less often along the colon. The ECG can show signs of myocardial dystrophy. In the hemogram, hypochromic anemia, eosinophilia, lymphocytosis and monocytosis are determined. Ulcers, polyps, amoebomas are detected endoscopically. In the analysis of feces, pre-cystic and cystic forms of amoeba can be determined.

Along with changes in the mucous membrane and necrosis, a regenerative process takes place in the intestinal wall, leading to the restoration of the defect through the formation of fibrous tissue. Such a process in chronic amebiasis can lead to the

formation of strictures and stenosis, usually in the ascending and descending parts of the colon. When the lesions are localized in the rectosigmoidal part of the large intestine, the clinical picture may correspond to the colitis syndrome in shigellosis. When the lesions are localized in the cecum, constipation with pain in the right ileal region, characteristic of the clinical picture of appendicitis, is usually noted.

It should be remembered that tests do not always give a positive result in a chronic course, and therefore it is necessary to carry out repeated studies or do provocation (laxatives) to accelerate the passage of the chyme through the intestines. This technique can also be carried out with suspected cyst carriers.

Extraintestinal amebiasis.

Extraintestinal amebiasis develops during the period of acute intestinal amebiasis, followed by it or a few months later, and sometimes even years. In some patients, it is not possible to establish a history of intestinal amebiasis. The most common amoebic liver disease. There are two known clinical forms of liver amebiasis: amoebic hepatitis and liver abscess.

Acute amoebic hepatitis usually develops in patients with acute intestinal amebiasis and is manifested by an enlarged liver (sometimes significant) for several days, pain in the right hypochondrium. On palpation, the liver is somewhat hardened and painful. Jaundice sometimes develops. The temperature is often subfebrile, with periodic rises to high numbers, but it can remain normal. In the hemogram, moderate leukocytosis.

Constant symptoms of amoebic liver abscess are enlargement of the liver and intense pain with their localization in the place of development of the pathological process. The pain is intense, radiating to the right shoulder, aggravated by deep breathing, palpation of the liver, changing the position of the patient in bed, coughing and sneezing. The temperature rises to 39˚C and above, the temperature curves are remitting, hectic or constant type. Fever is accompanied by chills, with a decrease in temperature, increased sweating is observed. The phenomena of intoxication are expressed.

Patients walk carefully, bent over to the side of the lesion and putting the elbow to the right hypochondrium, lie motionless in bed with their legs brought to the stomach. The appearance of patients is characterized by emaciation, sunken cheeks and eyes, pointed facial features, depression. The skin turgor is reduced, sometimes the skin becomes earthy. In severe cases, swelling of the skin, feet and legs appears. The abdomen is usually swollen, weakly involved in the act of breathing, muscle tension is often determined in the right hypochondrium. Pounding in the liver is painful.

With an amoebic liver abscess, the cardiovascular system suffers: heart sounds are muffled or deaf, maximum and diastolic are lowered, pulse is speeded up. X-ray examination reveals the high standing of the right and very rarely the left dome of the diaphragm with a decrease in its mobility. With subphrenic abscess localization, effusion in the right pleural sinus can be determined.

In the chronic course of amoebic liver abscess, the fever becomes abnormal, weakness, exhaustion increase, constant pressing pains in the right hypochondrium are noted. The liver is enlarged, painful, when an abscess is localized on its front surface, it can be palpated as a tumor-like formation with a diameter of up to 10 cm. Abscesses are single or multiple, localized, as a rule. In the right lobe of the liver. More often men are ill than women.

In the hemogram of patients with amoebic liver abscess, neutrophilic leukocytosis (15-50 × 109 in 1 μl) is found with a shift of the leukocyte formula to the left. ESR - accelerated. With a prolonged course, hypochromic anemia develops.

With hematogenous drift of amoebas into the lungs or with a breakthrough of a liver abscess into the pleural cavity, pulmonary amebiasis develops, clinically proceeding as a specific pleuropneumonia or lung abscess. Pneumonia presents with chest pain, cough, dry or scanty sputum, sometimes bloody. Body temperature is normal or subfebrile. With percussion of the lungs, dullness is detected. On auscultation - fine bubbling rales. X-ray examination reveals infiltrative changes in the lungs without signs of cavity formation. In peripheral blood, a small neutrophilic leukocytosis is sometimes determined. ESR-accelerated. Pneumonia is characterized by a

sluggish course and, if no specific treatment is carried out, they can turn into lung abscesses.

Amoebic abscesses of the lungs, as a rule, take a chronic course. Subfebrile temperature with periodic high rises. When coughing, a large amount of sputum is excreted in the blood ("chocolate" sputum), in which E. histolitica can be found. Ulcerative laryngitis and tracheitis develop. X-ray - a cavity in the affected lung with a horizontal level of the fluid contained in it is determined.

Cutaneous amebiasis is recorded mainly in the aborigines of tropical endemic zones. The disease is manifested by the formation of erosions and deep, slightly painful ulcers on the buttocks, in the perineum and in the perianal region. Perianal ulcers can lead to subsequent fistula formation.

Complications.

Among the complications from the intestine with amebiasis, the most important are appendicitis, pericolitis, perforation of the intestinal wall followed by peritonitis, intestinal strictures, amoeba, bleeding.

*Amebic appendicitis*the most frequent complication of the underlying disease (about 30%). The course is difficult with symptoms of common appendicitis against the background of amoebic intestinal lesions. Amebic appendicitis usually develops in an acute period or with an exacerbation of chronic amebiasis, so the symptoms of appendicitis are superimposed on the clinical picture of the current infection, which helps in the diagnosis and recognition of the etiology of appendicitis.

As a result of perforation of the intestinal wall, with an ulcerative necrotic process, purulent peritonitis occurs in it. Symptoms of amoebic peritonitis are usually the same as in perforated peritonitis of a different etiology, but often, especially with frequent prescription of antibiotics and in debilitated patients, it proceeds sluggishly with a mild pain syndrome, which makes it difficult to recognize and treat it in a timely manner. Amoebic peritonitis can be enclosed or spilled. In the latter case, it is especially difficult.

With the formation of scars at the site of deep ulcers, narrowing of the intestine may occur with symptoms of its relative obstruction, which, in turn, may occur dur-

ing the formation of adhesions between the organs of the abdominal cavity and, occasionally, with an amoeba. Cicatricial narrowing with predominant localization in the distal colon occurs in patients who have had a severe form of amebiasis with necrotizing ulcerative colitis. Cicatricial narrowing is often ring-shaped, due to the rejection of areas of the intestinal mucosa. In this case, stool retention, difficulty and soreness during bowel movements may occur. The feces look like narrow ribbons or pencils.

Amoeba is a tumor-like formation in the wall of the colon of an inflammatory nature, which occurs more often in the blind and ascending parts of the colon, less often in the hepatic and splenic flexures. The amoeba consists of fibroblasts, collagen and cellular elements, and contains a relatively small number of amoebas.

Intestinal bleeding is observed in the acute form and exacerbation of the chronic form of the disease. They occur with abdominal pain, growing anemia, cardiac disorders. As a rule, there is no need for urgent surgical intervention for this bleeding, patients respond well to conservative therapy.

Differential diagnostics.

Intestinal amoebiasis is differentiated from other protozoal diseases, shigellosis, salmonellosis, yersiniosis, campylobacteriosis, trichocephalosis. Intestinal schistosomiasis in the early in the early stage of the disease is distinguished from amoebiasis by possible exanthema and pruritus, in the stage of developed intensive invasion - myalgia and arthralgia, cough, hepatolienal syndrome, severe eosinophilia, excretion of eggs of schistosome with feces. When carrying out differential diagnosis of intestinal amebiasis with colon neoplasms, ulcerative colitis, Crohn's disease, methods of endoscopic studies play a decisive role.

Amebic liver abscess should be distinguished from echinococcosis, visceral leishmaniasis. With echinoccosis, high fever is not noted, intense pain in the liver and fluctuation are not characteristic, eosinophilia is often found in the hemogram. X-ray and ultrasound examination of the liver are of great importance. Visceral leishmaniasis is characterized by high prolonged wave-like fever, enlargement and induration of

the liver, and especially the spleen, their painlessness on palpation, leukopenia with a sharp increase in ESR.

Laboratory and instrumental diagnostics.

Typical changes in the hemogram with amebiasis: moderate leukocytosis and anemia, eosinophilia, monocytosis and lymphocytosis, increased ESR.

Microscopic examination of freshly excreted stool reveals the trophozoites of E. histolitica. The detection of a large vegetative form of amoeba containing phagocytosed erythrocytes confirms the diagnosis of intestinal amoebiasis, the tissue form is rarely detected. The presence in feces of luminal, precystic forms and quadruple cysts can also occur with amoebic carriage.

With extraintestinal amebiasis, it is rarely possible to detect an amoeba in the abscess pus, and therefore laboratory diagnostics is based on serological research methods - RNGA, ELISA, RIF, etc., which give positive results on average in 80% of cases. Serological methods are of little use in an endemic focus due to the long (up to 1 year or more) retention of "trace" antibodies (AT) in persons who have had amoebiasis. In recent years, PCR has been introduced into laboratory practice, as well as highly specific methods for detecting amoebic antigen (AG) using monoclonal antibodies.

Colonoscopy is essential in the diagnosis of intestinal amebiasis. In proximal colitis, only catarrhal and rarely erosive changes in the mucous membrane of the blind and ascending intestines can be seen. With a predominant lesion of the distal colon or with pancolitis, the intestinal mucosa is edematous, has foci of hyperemia, erosion and ulcers at different stages of development, there is little mucus in the intestinal lumen. With colonoscopy, it is possible to obtain a biopsy from the edges of the ulcers for subsequent parasitological and histological studies. The characteristic bulbous form of colon ulcers is noted. Biopsy is relatively contraindicated in fulminant colitis due to the increased risk of intestinal perforation.

In order to diagnose amoebic abscesses, X-ray, ultrasound, CT, MRI are used.

Treatment.

Treatment in most cases is outpatient. Hospitalization of patients is indicated for severe colitis and extraintestinal lesions. The diet is prescribed individually, depending on the tolerance of food.

With the localization of non-invasive forms of amoeba in the lumen of the intestine and the asymptomatic course of invasion, the risk of further development of the disease is possible. In these cases, luminal amoebicides are prescribed:

- etofamide at 20 mg / kg / day in 2 divided doses for 5 - 7 days;
- diloxanide furoate 500 mg 3 times a day for 10 days

In mild and moderate uncomplicated intestinal amoebiasis with the release of invasive forms of amoebas, tissue amoebicides from the group of nitroimidazoles are shown. Prescribe metronidazole (trichopolum) inside at 30 mg / kg / day in 3 divided doses for 7 to 10 days. Since, after treatment with metronidazole, parasites persist in the intestines in 40-60% of patients, after that, for complete release from luminal forms of amoebas, etofamide is prescribed at 20 mg / kg / day in 2 doses for 5 to 7 days.

To accelerate the elimination of amoebas and the repair of the intestinal mucosa simultaneously with metronidazole, it is useful to prescribe enterosidiv, 2 tablets 3 times a day for 10 days. The drug, acting on the pathogenic and conditionally pathogenic flora of the intestine, inhibits the transformation of commensal forms of amoebas into aggressive ones. In parallel with amoebicidal specific drugs, doxycycline can be prescribed at 100 mg per day.

With clinically pronounced intestinal amebiasis, a complex 10-day course of metronidazole in combination with doxycycline or enterosidive is recommended.

With extraintestinal amebiasis in adults, a 10-day course of metronidazole is combined with the appointment of chloroquine phosphate. During the first 2 days of treatment, 300 mg of chloroquine (base) is prescribed 2 times a day, then 300 mg once a day for 2 to 3 weeks. At the same time, it is possible to use broad-spectrum antibiotics. An alternative treatment regimen can also be used: tinidazole orally at 2.0 g / day in 1 dose for 5 to 10 days.

After the completion of these courses of treatment, etofamide is prescribed at 20 mg / kg / day in 2 divided doses for the elimination of the remaining amoebas in the intestine for 5 to 7 days.

Treatment of acute forms of amebiasis and exacerbations of chronic should be complex: along with the use of specific etiotropic therapy, pathogenetic, symptomatic and general strengthening are used. Pathogenetic therapy: for the correction of electrolytes and detoxification, saline solutions are prescribed orally - oralit, rehydron, etc. or intravenously - quartasol, trisol, acesol, etc. The use of desensitizing and antihistamines is shown - tavegil, suprastin, phenergan, etc. As anesthetic and antihistamines drugs are used antispasmodics - papaverine, platifillin, spazmalgon, etc.; astringent inside and in enemas. In some cases, weakened patients and dysproteinemia and anemia, transfusion of protein solutions and blood components (plasma, erythrocyte mass) is indicated.

Patient discharge rules.

In case of inpatient treatment, the discharge of convalescents is permissible after their clinical recovery and negative results in 6 parasitological studies of feces, taken at intervals of 1 - 2 days.

Dispensary observation.

Dispensary observation of those who have recovered is carried out for 12 months with parasitological examination of feces in 1.3, 6 and 12 months after discharge. Carriers of infection are not allowed to work in food factories.

Test tasks.

1. *With amoebiasis, the most commonly affected:*
    1. Stomach
    2. Duodenum
    3. Cecum
    4. Ascending gut
    5. Transverse colon
2. *Morphological changes in the colon:*
    1. Epithelial cytolysis

2. Microcirculation disorder
3. Tissue necrosis
4. Deep ulceration
5. Overgrowth of granulation tissue in the intestinal wall (amoeboma)

3. Symptoms of moderate intestinal amebiasis:

1. Diarrhea with vitreous, mucous stool
2. Pain along the colon
3. Stool in the form of "raspberry jelly"
4. Subfebrile temperature
5. Anemia

4. Manifestations of extraintestinal amebiasis:

1. Liver abscess
2. Lung abscess
3. Brain abscess
4. Myocarditis
5. Cutaneous amebiasis

5. Laboratory diagnostics of amebiasis:

1. Detection of vegetative forms of amoebas in feces
2. RNGA
3. ELISA
4. Immunofluorescence method
5. PCR

6. Complications of amebiasis:

1. Colon perforation
2. Appendicitis
3. Obstructive obstruction
4. Purulent pleurisy
5. Suppuration of amoeba

Answers:

1. - 3, 4, 5

2. - 1, 2, 3, 4, 5
3. - 1, 2, 3, 4, 5
4. - 1, 2, 3, 5
5. - 1, 2, 3, 4, 5
6. - 1, 2, 3, 4, 5

Situational task.

The patient is 31 years old. 1.5 months after arriving from Pakistan, where he was for about 2 years, malaise, sweating appeared, temperature fluctuations from 37.2 to 39 - 40 --C were observed during the day, which decreased after taking analgesics. Constantly disturbed by pain in the right hypochondrium, which intensified with movement or concussion, bitterness in the mouth. On the 5th day of illness, he turned to the doctor of the polyclinic and was referred to hospitalization in the infectious diseases department with a diagnosis of typhoid-paratyphoid disease.

A history of recurrent stool disorders for 5 years.

On examination - a state of moderate severity, the skin is pale, slight subicteric sclera and mucous membranes. The lymph nodes are not enlarged. Vesicular breathing in the lungs. Pulse 92 in 1 min. Heart sounds are weakened. HELL 115/75 mm RT st. The tongue is moist, coated with a grayish bloom. The abdomen is soft on palpation. In the right hypochondrium, a painful liver is palpated, protruding from under the edge of the costal arch by 3 - 4 cm. Liver dimensions: 12 × 17 × 10 cm. Ortner's symptom is positive. The spleen is not enlarged, the caecum is swollen, hums on palpation. Beating symptom - weakly positive on the right. Meningeal and focal symptoms could not be identified.

Make a preliminary diagnosis.

Make a plan of examination and treatment.

The answer to the problem.

1. Preliminary diagnosis: intestinal amebiasis, amoebic liver abscess.
2. Survey plan:

- general blood analysis;
- general urine analysis;
- scatological examination of feces for protozoa;
- blood for RNGA, RIF, PCR;
- chest x-ray;
- blood culture for sterility;
- survey radiography of the abdominal organs;
-biochemical blood test (bilirubin, cholesterol, transaminases);
- Ultrasound of the abdominal cavity.

3. Treatment plan:

- dihydroemetine hydrochloride - 1 mg / kg per day IM (no more than 60 mg) for 6 days.
- at the same time or immediately after completion of the course of dehydroemetine for amoebic liver abscesses, chloroquine is recommended - 600 mg of base per day for 2 days, then 300 mg of base for 2 to 3 days;
- after the completion of the course of dehydroemetine, in order to eliminate the remaining amoebas in the intestine, an amoebicide of universal action is used - metronidazole, 2 tablets 3 times a day (8 - 10 days)
- funds that affect the body's defenses and to eliminate intestinal dysbiosis.

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