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Features of the Course of Contagious Giardiasis Associated with Giardiasis

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Summary: Giardiasis is a widespread human protozoan disease. An invasive disease that occurs as an asymptomatic carrier, sometimes leading to impaired function of the small intestine. The fact is that often giardiasis occurs under the "mask" of other diseases. It can occur almost without clinical signs of the disease, as a result of which infected people do not seek medical help and giardiasis is detected in them by chance. In our article, we tried to give our point of view on contagious helminthiases associated with giardiasis.

Relevance. The causative agent of giardiasis Lamblia intestinalis (synonyms: Giardia lamblia, Giardia intestinalis, Giardia duodenalis) is a well-known protozoan organism isolated from the human intestine and is the cause of diarrhea in humans. In addition to humans, this type of Giardia can be found in various mammals: dogs, cats, sheep, cattle, bears, badgers, etc., as well as birds and reptiles. Some of the animals are probably the reservoir of this infection for humans. The causative agents of the disease exist in the form of two separate forms - the vegetative form and the cyst. If the first in the environment quickly die, then the cysts survive for a long time: in reservoirs, tap and waste water - up to several months, on food - from several hours to several days, in sand - up to 9 - 12 days. Giardia cysts (like helminth eggs) are resistant to the action of chlorine, ultraviolet radiation, can withstand freezing and heating up to 50 0C, but die when boiled.The source of giardiasis invasion is an infected person or animal, but the person has a leading role. Giardiasis refers to contagious protozooses. The transmission factors of the pathogen are dirty hands, water, food containing Giardia cysts. Insects (flies, cockroaches, mealworms, dung beetles) can also spread Giardia cysts. The mechanism of transmission of invasion is fecal-oral. The infectious dose is about 10-100 cysts.

Very favorable conditions for the transmission of giardiasis by contact and household can be created in the family, children's groups (nurseries, kindergartens, schools). If a child or an adult does not wash his hands well after going to the toilet, then he can transmit the infection through toys, furniture, dishes, and also when using a common towel, on which cysts remain viable for up to 2 days. Bad habits play a significant role in infection: keep your finger in your mouth, take it in your mouth, bite your nails, pencils, pens, etc. In children's institutions, when children affected by Giardia are identified, sanitary and epidemiological measures are taken, as with contact helminthiases. Given the high resistance of Giardia cysts to chlorine-containing preparations, the use of detergents and disinfectants and ultraviolet irradiation is recommended. Particular attention is paid to the personal hygiene of staff and children.

Diagnostics. The diagnosis of giardiasis is established on the basis of clinical signs of the disease, the results of a laboratory examination, and other data. The main laboratory diagnostics are parasitological studies of feces. Due to the intermittent, intermittent discharge of cysts (with interruptions from several days to 2 weeks), it is necessary to conduct multiple studies of feces - up to 6-7 times or more, at intervals of 1-2 days. Often Giardia cysts are detected already during the first studies. As additional diagnostic methods for giardiasis, immunological (serological) methods can be used. They are based on the detection in the blood of antibodies specific to Giardia antigens.The level of antibodies depends on the characteristics of the human immune system, the intensity of infection, the form of the course of the disease, etc. Antibodies can be detected both in people at the height of the disease and in those who have had an infection in the recent past. These circumstances limit the diagnostic value of antibodies.

Clinic: From the clinical symptoms of giardia infection, four main syndromes can be distinguished: pain, dyspeptic, asthenoneurotic and allergodermatological. According to the clinical course, acute and chronic forms of giardiasis are distinguished. Giardiasis can also occur in subclinical and asymptomatic forms. Acute giardiasis is characterized by fever, vomiting, diarrhea, rubella-like or measles-like rash, anorexia, sharp pains in the upper and middle epigastric region, bloating, and without a special examination for giardiasis, it is diagnosed as "intestinal infection of unknown etiology." The acute form is most common in young children. The acute period usually lasts a few days, after which giardiasis most often passes into a subacute or chronic stage.

Chronic giardiasis is accompanied by the main syndrome complex: chronic intoxication, hypovitaminosis, dyspepsia, dysbacteriosis.Typical symptoms are from the gastrointestinal tract, persistent diarrhea, alternating constipation and diarrhea, malabsorption, cramping abdominal pain, dyspepsia, nausea, loss of appetite, symptoms of general disorders (headaches, irritability, malaise, fatigue, emotional lability, malnutrition, in children - a lag in physical development, weight loss), skin symptoms - in addition to those described above, urticaria, strophulus, pruritus.

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In adult patients with giardiasis, pain in the right hypochondrium is most often noted, aggravated by eating fatty foods, bitterness or dryness in the mouth, nausea, unstable stools, pain in the epigastric region, less often pain around the navel, loss of appetite, headaches and dizziness. In most patients, the tongue is lined with a yellowish coating, there is pain on palpation at the projection point of the gallbladder, the epigastric region. Due to the rapid reproduction of Giardia in the intestine, especially with poor nutrition, the microbial landscape of the intestine changes dramatically. Microorganisms and fungi appear, which do not exist in the intestine under normal conditions and which, in turn, stimulate the process of reproduction of protozoa, the number of Escherichia coli, bifidumbacteria, lactobacilli, which provide the physiological process of digestion and absorption, is sharply reduced. There are also clinical forms of giardiasis with a predominance of allergic manifestations in the form of indomitable skin itching, urticaria, bronchial asthma and asthmatic bronchitis, eosinophilic pulmonary infiltrates, and sometimes persistent blepharitis. The most pronounced clinical manifestations coincide with periods of maximum intensity of cystic excretion. Pallor of the skin, especially the face, is observed in almost all patients, even with high levels of hemoglobin. With a long course of the disease and a high degree of intoxication, a sharp pallor of the skin of the nose ("marble nose") is released.In children with prolonged persistence of invasion, follicular hyperkeratosis (predominantly localized on the extensor surface of the arms, legs, lateral surfaces of the chest, abdomen), wavy pigmentation of the skin of the neck, pallor and subicteric shade of the nasolabial triangle are pathognomonic symptoms of giardiasis.

Clinical manifestations of the disease are detected in 80% of those infected, but asymptomatic carriage is also possible. Parasite carriers pose no less than a sick threat to the surrounding people, since they do not receive treatment and release the pathogen into the external environment for years. Giardiasis aggravates the course of many other infectious and non-infectious diseases, contributes to the allergization of the affected organism. With a long course, an organic pathology of the gastrointestinal, cardiovascular and other human systems is formed. Preventive vaccinations against the background of giardiasis may be ineffective. It is rather difficult to suspect giardiasis, it is not difficult to confuse it with other diseases, since most of its symptoms are also found in other diseases. Therefore, the diagnosis should be handled by specialists.

At the time of diagnosis, patients usually do not present any complaints.

1. General symptoms: occur due to the release of large amounts of cytokines by eosinophils - fatigue, fever and sweating, lack of appetite, weight loss.

2. Symptoms of the circulatory system (in ≈20%): associated with necrosis and fibrosis of the heart muscle and endocardium, as well as the occurrence of parietal thrombi in the heart cavities - symptoms of valve insufficiency, usually mitral and tricuspid, symptoms of restrictive cardiomyopathy, rhythm and conduction disturbances , thromboembolic complications, heart failure.

3. Symptoms from the respiratory system (in ≈50%): associated with eosinophilic infiltrates in the lungs, pulmonary fibrosis, heart failure or pulmonary embolism - chronic dry cough, shortness of breath.

4. Skin symptoms (in ≈60%): angioedema, skin redness, urticaria, papules and subcutaneous nodules, skin itching.

5. Gastrointestinal symptoms (in ≈30%): associated with mucosal ulcers, bleeding, perforation, cholecystitis, eosinophilic inflammation of the stomach or intestines; diarrhea, abdominal pain.

6. Neurological symptoms (in ≈55%): behavioral changes, memory disorders, ataxia, symptoms of peripheral polyneuropathy.

7. Others: enlargement of the liver or spleen, muscle and joint pain (caused by the release of pro-inflammatory cytokines by eosinophils), visual impairment (associated with retinal vascular thrombosis).

8.Natural course: chronic, sometimes mild, but most often progressive and can lead to death in a short time due to organ damage, usually heart failure, or as a result of transformation into acute leukemia.

Diagnostics

Additional research methods

1. Morphology of peripheral blood: eosinophilia (absolute number of eosinophils: mild 500–1500/μl; moderate 1500–5000/μl, severe >5000/μl), additional anemia (≈50%), thrombocytopenia (≈30%) or thrombocytosis (≈15%), moderate leukocytosis.

2. Bone marrow aspiration biopsy and trephine biopsy: indicated in patients with persistent HE of unknown etiology, eosinophilia >5000/μl, in case of suspected primary (neoplastic) HE/HES or lymphocytic variant of HES; aspiration biopsy - increased percentage of eosinophils, sometimes dysplastic changes in megakaryocytes and granulocytes; trepanobiopsy - hypercellularity, increased number of eosinophils, hyperplasia of a megakaryocytic or granulocytic germ, an increase in the number of reticular fibers.

3. Cytogenetic and molecular studies: in CEL, in most cases, the FIP1L1-PDGFRFA fusion gene is present, which arose as a result of a deletion of a fragment of chromosome 4 and encodes a protein with tyrosine kinase activity.

4. Other laboratory tests: increased concentration of IgE in idiopathic eosinophilia, in CEL-NIO - usually normal; increased concentrations of cardiac troponins, tryptase and vitamin B12 in neoplastic HES.

five.Histological examination of biopsy specimens of organs involved in the pathological process: eosinophilic infiltrates.

6. Other investigations: depending on the clinical picture (organs involved) and also to determine the cause of eosinophilia.

Diagnostic criteria

The diagnosis of HEL is made in accordance with the WHO criteria:

1) eosinophilia ≥1500/μl;

2) the presence of cytogenetic or molecular genetic abnormalities, or the number of blasts >5% in the bone marrow or >2% in the peripheral blood;

3) the absence of the Ph-chromosome and the chimeric BCR-ABL1 gene, as well as other myeloproliferative (IP, ET, PMF) or myelodysplastic/myeloproliferative (CMML, aCML) neoplasms;

Differential Diagnosis

1. HE/HES primary: eosinophils are part of a neoplastic clone (myeloproliferative tumors, AML).

2. HE/HES secondary (reactive): parasitic infestations (especially helminthiases), allergic reactions, drug reactions (allergic or toxic); rarer causes: pulmonary eosinophilia → Sect. 3.14.5, graft-versus-host disease, Hodgkin's lymphoma, peripheral T-cell lymphomas (PTCL), Langerhans cell histiocytosis, systemic indolent mastocytosis, solid tumor, allergic bronchopulmonary aspergillosis, chronic inflammatory diseases (eg, bowel), systemic connective tissue diseases (eosinophilic granulomatosis with vasculitis [formerly Churg-Strauss syndrome], other systemic vasculitis, deep eosinophilic fasciitis with eosinophilia).

3. Diseases affected by HPS

Treatment

1. Patients with an eosinophil count <5000/μl and no organ changes do not require emergency cytoreduction.

2. CEL with FIP1L1-PDGFRA gene and PDGFRB gene rearrangement: imatinib.In patients with heart failure, use glucocorticosteroids during the first 7 to 10 days of imatinib treatment to avoid deterioration in cardiac function caused by massive release of toxic proteins from eosinophil granules.

3. HES without swapping PDGFRA and PDGFRB: glucocorticosteroids, eg prednisone 1 mg/kg bw until eosinophils fall to normal and symptoms disappear, then slowly taper to the lowest effective maintenance dose. In reactive HPS, the underlying underlying disease should be treated.

4. In case of failure and in CEL-NOS: cytostatics (hydroxyurea, in case of failure of IFN-α, subsequently vincristine or etoposide; in case of failure, experimental treatment (mepolizumab, alemtuzumab) or allo-HSCT.

Prevention. To prevent infection with giardiasis, following the basic preventive measures will help:

-You should drink only boiled or factory-made bottled water.

- Washing raw fruits and vegetables is best finished by rinsing with boiled water or scalding with boiling water.

- It is recommended to use water filters capable of retaining particles with a diameter of 10 microns.

- You should not buy food from street vendors, eat food in establishments that are questionable in terms of compliance with hygiene rules.

- It is necessary to observe the rules of personal hygiene, to educate the necessary hygiene skills in children.

-In children's groups and common areas there should be special soap dishes, individual, and preferably disposable towels or napkins.-Given the high resistance of Giardia cysts to chlorine-containing preparations, it is recommended to use soap-soda solutions or detergent-disinfectants for cleaning (as in enterobiasis).

-Knowledge and compliance with preventive measures will help many people to prevent Giardia infection and the development of Giardiasis.

- In the absence of treatment, reliable self-healing is not observed in both acute and chronic forms of infection.

Literature

1. Bronshtejn A.M., Malyshev N.A. Gel'mintozy organov pishchevareniya: kishechnye nematodozy, trematodozy pecheni i larval'nye cestodozy (ekhinokokkozy) // Ross. med. zhurn. – 2004. – T. 12, №4.

2.Bronshtejn A.M., Luchev V.I., Malyshev N.A. Gel'mintozy organov pishchevareniya: problemy diagnostiki i lecheniya // Ros. med. zhurn. – 2005. – T. 7, №2.

3. Bodnya E.I. Problema profilaktiki parazitozov v sovremennyh usloviyah // Novosti mediciny i farmacii. – 2005. – №20–22. – S. 9.

4. Bodnya K.I. Diagnostika ta likuvannya gel'mintoziv u ditej // Suchasna gastroenterol. – 2006. – №6 (32).

5. Sergiev V.P. Parazitarnye bolezni segodnya i zavtra // Parazitarnye bolezni. – 2005. – №1.

6. Babak O.YA. Kishechnye gel'mintozy: situaciya i tendencii k ee izmeneniyu // Zdorov'ya Ukraïni. –2006. – №9/1.

7. Interv'yu s vedushchimi specialistami: professor Bodnya E.I., professor Kramarev S.A. i dr. //Zdorov'ya Ukraïni. – 2005. – №130.

8. Babak O.YA. Rol' i mesto tkanevyh parazitozov v patologii cheloveka // Zdorov'ya Ukraïni. –2007. – №7/1, 8, 9