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ULCER DISEASE OF THE STOMACH AND DUODENAL IN CHILDREN AND ADOLESCENTS

(Review article)

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Annotation: *Peptic ulcer disease (PU) is a chronic relapsing disease that occurs with alternating periods of exacerbation and remission, the leading manifestation of which is the formation of a defect (ulcer) in the wall of the stomach and duodenum.*

Key words: *gastric ulcer, duodenal ulcer, peptic ulcer, dyspeptic disorders, Helicobacter pylori, eradication therapy*

Etiology and pathogenesis. Peptic ulcer is a polyetiological, genetically and pathogenetically heterogeneous disease. Hereditary burden is of great importance (genetically determined high density of parietal cells, their increased sensitivity to gastrin, deficiency of trypsin inhibitors, congenital deficiency of antitrypsin, etc.)

When exposed to unfavorable factors (Helicobacter pylori infection, long-term poor nutrition, psycho-emotional stress, bad habits), a genetic predisposition to the development of ulcer is realized [13]. The pathogenesis of ulcer is based on an imbalance between the factors of acid-peptic aggression of the gastric contents and the elements of protection of the mucous membrane (MU) of the stomach and duodenum [3]. Strengthening aggressive factors or weakening protective factors leads to disruption of this balance and the occurrence of ulcers. Factors of aggression include hyperproduction of hydrochloric acid, increased excitability of parietal cells caused by vagotonia, infectious factors (Helicobacter pylori), impaired blood supply to the mucous membrane of the stomach and duodenum,

impaired antroduodenal acid brake, bile acids and lysolecithin. Protective factors are the mucous barrier, mucin, sialic acids, bicarbonates - reverse diffusion of hydrogen ions, regeneration, sufficient blood supply to the mucous membrane of the stomach and duodenum, antroduodenal acid brake. Ultimately, the formation of a peptic ulcer is caused by the action of hydrochloric acid on the mucous membrane of the stomach and duodenum, which allows antisecretory therapy to be considered the basis for the treatment of exacerbations of peptic ulcer disease [2]. The decisive etiological role in the development of ulcer is currently assigned to the microorganisms H. pylori. These bacteria produce a number of enzymes (urease, proteases, phospholipases) that damage the protective barrier of the mucous membrane, as well as various cytotoxins. Contamination of the gastric mucosa with H. pylori is accompanied by the development of superficial antral gastritis and duodenitis and leads to an increase in gastrin levels with a subsequent increase in the secretion of hydrochloric acid. Excessive intake of hydrochloric acid into the lumen of the duodenum under conditions of a relative deficiency of pancreatic bicarbonates contributes to increased duodenitis, the occurrence of intestinal metaplasia and the spread of H. pylori. In the presence of a hereditary predisposition and the action of additional etiological factors (nutrition errors, neuropsychic stress, etc.), an ulcerative defect is formed [15]. In children, unlike adults, H. pylori infection is much less often accompanied by ulceration of the stomach and duodenum. According to a large European multicenter study, in 1233 children with symptomatic H. pylori infection, PU was diagnosed in less than 5% of children under 12 years of age and only 10% of adolescents [4].

Epidemiology. Peptic ulcer disease is a fairly common disease, characterized by a chronic relapsing course and the possibility of life-threatening complications. In schoolchildren, PU occurs 7 times more often than in preschool children (2.7 per 1000 and 0.4 per 1000, respectively), in urban children - 2 times more often than in rural children. The ratio of boys to girls is 3:1. In the structure of pathology of the digestive organs, the share of peptic ulcer disease is 1.6%, and among diseases of the stomach and duodenum it is 2.7%. In the structure of PU

in children, duodenal ulcer (DU) is represented in 81% of cases, gastric PU is less common (13%) and combined localization of ulcerative defects in the stomach and duodenum is 6%. Classification of peptic ulcer according to A.V. Mazurin, 1984 with additions by V.F. Privorotsky and N.E. Luppova, 2005

Phases: 1. Exacerbation. 2. Incomplete clinical remission. 3. Clinical remission.

Course: 1. newly identified, 2. rarely recurrent (remission for more than 3 years), 3. continuously recurrent (remission less than 1 year) Localization: stomach, duodenum: - bulb - postbulbar sections dual localization

Form: 1. Uncomplicated. 2. Complicated: 1) bleeding 2) penetration 3) perforation 4) pyloric stenosis 5) perivisceritis

Infection with N. pylori 1.N.r. - positive, 2.N.r. - negative

Functional characteristics: The acidity of gastric contents and motility can be increased, decreased or normal. Clinical and endoscopic stages of peptic ulcer: Stage 1 - fresh ulcer Stage 2 - beginning of epithelization of the ulcerative defect Stage 3 - healing of the ulcerative defect with severe gastroduodenitis Stage 4 - clinical and endoscopic remission

Concomitant diseases: - pancreatitis - esophagitis - cholecystocholangitis

Diagnostics. Complaints and anamnesis. Features of the modern course of ulcer in children are an increase in the frequency of the disease, an increase in the frequency of complications and relapses, as well as leveling out the seasonality of exacerbations and their asymptomatic course in half of the patients [15]. The clinical manifestations of ulcer are diverse; the typical picture is not always observed, which significantly complicates diagnosis. Thus, in young children the disease often occurs atypically. Moreover, the younger the child, the less specific the complaints. At older ages, the symptoms of duodenal ulcer are similar to those in adults, although they may be more subtle.

When collecting anamnesis, it is recommended to pay attention to the presence of the following complaints: abdominal pain (epigastric or intragastric, nocturnal and “hungry” in nature, decreasing with food intake), heartburn,

belching, vomiting, nausea, decreased appetite, tendency to constipation or unstable stool, emotional lability, increased fatigue. Physical examination • During examination, it is recommended to pay attention to the presence of a white coating on the tongue • During palpation, it is recommended to pay attention to pain in the pyloroduodenal area. • It is recommended to evaluate for symptoms of muscle protection. • In the acute phase, it is recommended to evaluate the presence of a positive Mendelian symptom [2,12,15]. Laboratory diagnostics.

There are no laboratory markers pathognomonic for peptic ulcer.

Research is recommended to exclude complications. Patients undergo a complete complete blood count, fecal occult blood test, levels of total protein, albumin, amylase, lipase, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase, glucose, serum iron in the blood.

• It is recommended to determine Helicobacter pylori infection [7,14]

• H. pylori monoclonal fecal antigen and C13-carbon urease breath test (C13-UCT) are the main noninvasive tests.

• If it is not possible to perform the above non-invasive diagnostic methods, it is recommended to conduct a test for the presence of antibodies to H. pylori in the blood serum. Due to variability in the accuracy of various commercial serological tests, only validated IgG serological tests should be used. Blood serum tests for the presence of antibodies to H. pylori are most informative when conducting epidemiological studies. • In patients receiving proton pump inhibitors (PPIs), it is recommended, if possible, to withhold the PPI for 2 weeks before testing by bacteriology, histology, rapid urease test, UDT, or fecal H. pylori testing. [7]

Instrumental diagnostics • Fibroesophagogastroduodenoscopy (FEGDS) is recommended; if the ulcer is localized in the stomach, it is recommended to take 4-6 biopsies from the bottom and edges of the ulcer with histological examination. FEGDS allows you to reliably diagnose and characterize an ulcerative defect. Allows you to monitor its healing, conduct a cytological and histological assessment of the morphological structure of the gastric mucosa, and

exclude the malignant nature of ulceration. To exclude concomitant pathology, it is recommended to perform an ultrasound examination of the abdominal organs (liver, pancreas, gall bladder). • It is recommended to measure the acidity of the medium using the intragastric pH metry method • It is recommended to conduct a contrast X-ray examination of the upper gastrointestinal tract (according to indications: when complications of ulcer are detected or if there are contraindications to endoscopic examination) • Computed tomography is performed according to indications: when identifying penetrating ulcers to clarify the localization of the defect in relation to surrounding organs, when demonstrating a picture of pancreatitis and penetration into the pancreas, in case of pneumobilia with penetration into the bile ducts).

Differential diagnosis of peptic ulcer with functional disorders of the gastrointestinal tract, chronic gastroduodenitis, chronic diseases of the liver, biliary tract and pancreas is carried out according to anamnesis, examination, results of laboratory, endoscopic, x-ray and ultrasound examinations. Peptic ulcer disease must be differentiated from symptomatic ulcers, the pathogenesis of which is associated with certain underlying diseases or specific etiological factors. The clinical picture of exacerbation of these ulcers is erased, there is no seasonality or periodicity of the disease. Gastric and duodenal ulcers in Crohn's disease, which are sometimes also referred to as symptomatic gastroduodenal ulcers, are an independent form of Crohn's disease affecting the stomach and duodenum.

Treatment

Conservative treatment • Eradication therapy is recommended for gastric and duodenal ulcers associated with H. pylori. [7,14] • As first-line therapy, if resistance to clarithromycin does not exceed 15–20%, it is recommended to use standard triple therapy (according to the latest recommendations of the IV Maastricht Agreement (2010), ESPGHAN and NASPGHAN (2011)): PPI (esomeprazole, rabeprazole , omeprazole) 1-2 mg/kg/day + amoxicillin 50 mg/kg/day + clarithromycin 20 mg/kg/day PPI + clarithromycin + metronidazole

20 mg/kg/day. Duration of therapy is 10-14 days. In order to increase the acceptability of therapy, it is possible to use the so-called. “sequential” regimen, in which PPIs are prescribed for 14 days, and antibiotics are prescribed sequentially for 7 days each. If eradication therapy is ineffective, an individual selection of the drug is carried out based on the sensitivity of H. pylori to antibacterial drugs - third-line therapy. To assess the effectiveness of anti-Helicobacter therapy, standard non-invasive tests are used. Monitoring the effectiveness of eradication is determined after at least 6 weeks. after completion of treatment. • Due to the high level of resistance of H. pylori to metronidazole and poor tolerability of tetracycline in children, according to the recommendations of Russian experts, it is recommended to use the following regimens in children: First line therapy. - PPI + amoxicillin + clarithromycin - PPI + amoxicillin or clarithromycin + nifuratel (30 mg/kg/day) - PPI + amoxicillin + josamycin VC (50 mg/kg/day, no more than 2g/day). It is possible to use a “sequential” circuit. Quadruple therapy is used as second-line therapy: - bismuth subcitrate + PPI + amoxicillin + clarithromycin - bismuth subcitrate + PPI + amoxicillin or clarithromycin + nifuratel. The duration of treatment is 10-14 days [18]. • In order to overcome the resistance of H. pylori to clarithromycin and reduce side effects from the use of antibacterial drugs, it is recommended to use a regimen with sequential administration of antibiotics: PPI + bismuth subcitrate + amoxicillin - 5 days, then PPI + bismuth subcitrate + josamycin - 5 days [19]. The low detection rate of familial carriage of HP infection and the lack of a systematic approach to its eradication contributes to reinfection of HP and, as a consequence, recurrence of DU in children [20]. The highest effectiveness and safety of anti-Helicobacter therapy occurs against the background of prebiotic or probiotic therapy. • In case of peptic ulcer not associated with H . pylori, the goal of treatment is to relieve the clinical symptoms of the disease and scarring of the ulcer. In this regard, the prescription of antisecretory drugs is recommended. 17 • It is currently recommended to use proton pump inhibitors as the drugs of choice for peptic ulcers not associated with H. pylori: esomeprazole, omeprazole, rabeprazole,

which are prescribed at a dose of 1-2 mg/kg/day. The duration of the PPI course is 4 weeks for PUD, 8 weeks for PUD [21]. • H2-blockers have lost their position and are now rarely used, mainly, their use can be recommended for peptic ulcers not associated with H. pylori, when it is impossible to use PPIs (or in combination with them) in order to enhance the antisecretory effect [2]. • Antacids (aluminum hydroxide or phosphate, magnesium hydroxide) are recommended for use in complex therapy for symptomatic purposes to relieve dyspeptic complaints. To enhance cytoprotection, bismuth subcitrate 8 mg/kg/day is prescribed for up to 2-4 weeks. • For gastrointestinal motility disorders, it is recommended to prescribe prokinetics, antispasmodics • The effectiveness of treatment for gastric ulcers is recommended to be monitored endoscopically after 8 weeks, for duodenal ulcers - after 4 weeks [21]. Surgical treatment • Surgical treatment of gastric ulcer is recommended in the presence of complications of the disease: perforation of the ulcer, decompensated cicatricial ulcerative pyloric stenosis, accompanied by severe evacuation disorders; profuse gastrointestinal bleeding that cannot be stopped by conservative methods, including the use of endoscopic hemostasis. When choosing a method of surgical treatment, preference is given to organ-preserving operations. Rehabilitation • Physiotherapy and physical therapy (physical therapy) are recommended to improve blood and lymph circulation in the abdominal cavity; normalization of gastric and duodenal motility, secretory and neurohumoral regulation of digestive processes; creating favorable conditions for reparative processes in the mucous membrane of the stomach and duodenum.

Prevention and clinical observation. Prevention of ulcer disease involves eliminating factors that contribute to the formation of ulcers: combating bad habits (smoking), normalizing the daily routine and diet, eradicating HP infection in patients with functional dyspepsia, simultaneous prescription of PPIs if it is necessary to take NSAIDs and anticoagulants. An important place in the prevention of peptic ulcer disease and its relapses is occupied by sanatorium-resort treatment, carried out no earlier than 2-3 months after the exacerbation subsides.

Dispensary observation of a patient with a peptic ulcer is carried out for 5 years from the onset of the disease or exacerbation, even in the absence of the morphological substrate of the ulcer (clinical endoscopic remission). Complications of ulcer in childhood are observed in 7-10% of patients. In boys, complications are observed more often than in girls in the case of duodenal ulcer (with isolated duodenal ulcer - 2.3 times, with a combination of gastric and duodenal ulcer - 8 times). The proportion of complicated forms of ulcers is observed in children of all age groups with the same frequency. The structure of complications is dominated by bleeding (80%), less common are stenosis (11%), perforation (8%) and ulcer penetration (1.5%) [15,17]. Bleeding is characterized by blood in the vomit (scarlet or coffee-ground vomit), black tarry stools. With large blood loss, weakness, nausea, pallor, tachycardia, decreased blood pressure, and sometimes fainting are characteristic. With hidden bleeding in the stool, a positive reaction to occult blood is determined. Stenosis of the pylorobulbar zone usually develops during the healing process of the ulcer. As a result of food retention in the stomach, it expands with the subsequent development of intoxication and exhaustion. Clinically, this is manifested by vomiting of food eaten the day before, increased gastric motility, especially during palpation, and a "splashing noise" determined by jerky palpation of the abdominal wall. Penetration (penetration of an ulcer into neighboring organs) usually occurs against the background of a long and severe course of the disease and inadequate therapy. Accompanied by increased pain radiating to the back. There is vomiting, which does not bring relief, and fever is possible. Perforation of an ulcer is 2 times more common when the ulcer is localized in the stomach. The main clinical sign of perforation is a sharp, sudden ("dagger") pain in the epigastric region and in the right hypochondrium, often accompanied by a state of shock. There is a weak pulse, sharp pain in the pyloroduodenal area, and disappearance of hepatic dullness due to the release of air into the free abdominal cavity. Nausea, vomiting, and stool retention are observed [15,17]. Leading children. Indications for hospitalization: Peptic ulcer with a clinical picture of severe exacerbation (severe

pain). Signs of complications of ulcerative disease. Peptic ulcer with a history of complications. Peptic ulcer with concomitant diseases. Detection of ulcerations in the stomach, requiring differential diagnosis between benign ulcers and gastric cancer. Children with exacerbation of peptic ulcer disease are treated in pediatric or gastroenterological departments. The duration of hospital stay is on average 14-21 days at the onset and recurrence of peptic ulcer disease. Children with an uncomplicated course of peptic ulcer disease are subject to conservative treatment in an outpatient setting. Children in remission are observed on an outpatient basis. Children in remission are observed on an outpatient basis. Removal from dispensary registration is possible with complete remission for 5 years [12,13,17].

Outcomes and prognosis. The prognosis is favorable for uncomplicated peptic ulcer disease. In most children, the first course of treatment in a hospital leads to complete healing of the ulcer and recovery. In case of successful eradication, relapses of peptic ulcer occur in 6–7% of patients. The prognosis worsens with a long history of the disease in combination with frequent, prolonged relapses, with complicated forms of peptic ulcer disease.

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