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ACUTE AND CHRONIC GASTRITIS AND GASTRODUODENITIS IN CHILDREN

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Abstract
Nowadays, among children, chronic gastritis and gastroduodenitis, as well as peptic ulcer disease of the stomach and duodenum, constitute a major part of gastrointestinal disorders. Therefore, having sufficient knowledge about these diseases, taking timely preventive measures, and starting treatment early make it possible to control these conditions completely and avoid potential complications.
This article describes the causes, clinical presentation, treatment, and prevention of chronic gastritis and gastroduodenitis, and peptic ulcer disease of the stomach and duodenum.

Keywords: Gastritis, secretion, infiltration, dyspeptic syndrome, meteorism, atrophy, gastroenteritis, gastroscopy, hyperthermia, metaplasia, intoxication, regeneration, fibro esophagogastroduodenoscopy (FEGDS), gastroduodenitis.

Introduction

Acute Gastritis

Acute gastritis is an acute inflammation of the gastric mucosa that occurs as a result of the short-term exposure of strong irritants.

Etiology

The development of acute gastritis may occur under the influence of various factors - exogenous and endogenous. Accordingly, the following types are distinguished:
I. **Primary (exogenous) acute gastritis**
II. **Secondary (endogenous) acute gastritis**
Primary acute gastritis may develop under the influence of the following factors:

1. The direct effect of infectious agents on the gastric mucosa.
This is usually associated with the consumption of poor-quality food, improper storage of mixtures and products, and non-compliance with hygiene rules. The most common pathogens are **staphylococci, salmonella, shigella, yersinia, klebsiella, and Escherichia coli**.

2. Chemical irritants and medications

These include acids and alkalis, alcohols, household chemical products, compounds of arsenic, iodine, phosphorus, acetone; medications such as **salicylates and other NSAIDs, glucocorticoids, reserpine, antimetabolites, nootropics, antibiotics, sulfonamides, iodine, bromine, iron, and potassium preparations**.

3. Food allergens - both protein and non-protein

In infants during the first months of life, acute gastritis may also develop as a result of a sudden transition to artificial feeding.

Secondary acute gastritis may develop against the background of general acute infectious diseases. In this case, gastric damage occurs due to the effects of toxins or the hematogenous spread of viruses and bacteria.

This type of acute gastritis may be observed in **diphtheria, influenza, measles, scarlet fever, typhus, pneumonia, and viral hepatitis**.

The most severe form of endogenous acute gastritis is **phlegmonous gastritis**, in which purulent inflammation develops not only in the mucosa but also in all layers of the stomach wall. Etiologically, phlegmonous gastritis is associated with the hematogenous penetration of **staphylococci or hemolytic streptococci**, and less commonly Escherichia coli, pneumococci, Proteus, or clostridia into the stomach wall.

The development of acute phlegmonous gastritis is facilitated by the following conditions:

- disruption of the integrity of the stomach wall (foreign body ingestion or trauma);
- purulent infectious foci in adjacent organs;
- peritonitis;
- septic conditions.

In acute renal failure, due to the rapid development of intoxication, an acute inflammatory process may also develop in the stomach.

Gastritis associated with **Helicobacter pylori** may also initially present as acute gastritis if massive infection with this microorganism occurs at once. This was convincingly demonstrated in self-infection experiments by B. Marshall and other volunteers. However, the acute gastritis observed in helicobacteriosis represents only the initial phase of the process, which later progresses into a chronic form.

Pathogenesis

The stomach has a powerful protective system that prevents the survival of microflora and damage to the mucosa. This system consists of:

- a low pH environment;
- the production of mucus, which increases in response to harmful influences.

Therefore, for **exogenous acute gastritis** to develop, the pathogen must be aggressive or present in high concentration. For example:

- small amounts of pathogenic microbes die in the acidic environment of the stomach;
- however, with massive invasion, the same microorganisms trigger acute inflammation.

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The duration of contact between the pathogen and the gastric mucosa is also important: if the stomach empties quickly, the pathological process may not develop in the stomach, and instead the lower gastrointestinal tract (acute enteritis, colitis) may be predominantly affected.

The most severe local damage is observed in **chemical burns (corrosive gastritis)**.

Another mechanism is the **endogenous pathway**, in which an acute inflammatory process in the mucosa develops due to hematogenous damage. In such cases, not only local but also systemic symptoms of the disease are present.

Sometimes a combined mechanism—both exogenous and endogenous-may occur, for example, when medications are taken during an acute infection.

Pathomorphology

Morphological changes observed in **acute gastritis** are not strongly dependent on its etiology and are characterized by:

- nonspecific inflammatory reaction,
- hyperemia and edema of the mucosa,
- neutrophilic infiltration,
- increased mucus production.

In some cases, small erosions with hemorrhages into the mucosal lamina propria may be detected.

Clinical Presentation

The disease typically begins with:

- restlessness of the child,
- loss of appetite,
- nausea,
- increasing general weakness.

Diffuse pain of moderate intensity may appear in the **epigastric region and around the navel**, described as aching, pressing, or cramping.

A typical sign is **recurrent vomiting**, sometimes containing mucus and occasionally blood. Vomiting brings some relief. Later, **diarrhea** (if acute enterocolitis develops), signs of **dehydration**, and **intoxication** may appear.

The abdomen is usually soft, with moderate tenderness in the **epigastrium and periumbilical area** on palpation.

Acute Allergic Gastritis

Symptoms are often accompanied by:

- rash (mostly urticarial),
- Quincke's edema,
- occasionally respiratory allergic manifestations.

Acute Corrosive Gastritis

Occurs after ingestion of **alkalis, acids, or disinfectants** and is considered the most severe form. Due to strong local effects, not only the stomach but also the **oral cavity and esophagus** are affected.

Clinical signs include:

- pain in the mouth,
- severe burning pain behind the sternum,

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- shortness of breath,
- pain and difficulty during swallowing,
- vomiting with blood.

Systemic signs include:

- tachycardia,
- decreased blood pressure.

On examination, burns may be detected in the oral cavity and pharyngeal mucosa; palpation reveals **epigastric tenderness** and sometimes **muscle tension**.

Acute Endogenous Gastritis

Symptoms are usually milder and occur against the background of **general intoxication** due to the underlying disease.

Typical signs include:

- moderate epigastric pain,
- nausea,
- anorexia,
- occasionally vomiting with blood.

Acute Phlegmonous Gastritis

Main symptoms:

- high fever,
- recurrent vomiting,
- severe epigastric pain.

The child's general condition may deteriorate rapidly:

- pallor,
- dry tongue,
- epigastric tenderness with muscle rigidity.

Laboratory findings:

- neutrophilic leukocytosis with left shift.

Urine analysis may show:

- mild proteinuria,
- microhematuria.

Diagnosis

The symptoms of **acute gastritis (AG)**, especially the exogenous type, are so characteristic that the diagnosis can often be made **without additional investigations**.

Important anamnesis factors include:

- sudden transition to artificial feeding,
- consumption of spoiled food or mixtures,

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- possible allergens, chemical substances, or medications.

Clinical signs aiding diagnosis:

- acute onset,
- epigastric tenderness,
- absence of peritoneal irritation signs.

Differential Diagnosis

Acute gastritis should be differentiated from:

- acute appendicitis,
- pancreatitis,
- cholecystitis.

Acute gastritis is not characterized by:

- very severe pain,
- strict localization,
- irradiation of pain.

Vomiting usually brings relief.

Endoscopy

Endoscopy is **not mandatory** for acute gastritis, but if performed, the following may be observed:

- hyperemia and edema of the gastric mucosa,
- occasional erosions,
- increased peristalsis,
- large amounts of mucus on the stomach walls and in its lumen.

Treatment

The most important aspect of **acute gastritis treatment** is to identify and eliminate the cause of the disease. In most cases, therapy is **symptomatic**.

Usually, it is sufficient to **empty the stomach**:

- Give the child 2–3 glasses of warm water to induce vomiting.

In **toxic-infectious or chemical acute gastritis**, gastric lavage with warm water through a thick tube is required during the first hours of the disease.

During the first day, **food should be withheld**, and a **water-tea diet** or **small warm drinks** is recommended (Oralit, Regidron, low-mineralized mineral water).

Afterward, the diet is gradually expanded, adhering to **mechanical, chemical, and thermal sparing principles**.

Allowed foods include:

- smooth broths,
- liquid pureed cereals,
- kissel,
- fruit jelly,
- meat soufflé and quenelles,

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- soft-boiled eggs,
- white bread toast (Diet Table No. 1a).

For pain relief:

- **Spasmolytics:** No-shpa, Papaverine – ½–1 tablet 3 times a day,
- **Cholinolytics:** Buscopan, Platyphyllin, Belladonna preparations – ½–1 tablet 3 times a day,
- **Antacids:** Almagel, Maalox, Phosphalugel – 1 teaspoon 4–6 times a day.

Adsorbents:

- Smecta, Polifepan, Cholestyramine – 5–20 g, 2–3 times a day, dissolved in plenty of water, between meals.

In case of vomiting – prokinetics:

- Cerucal, Motilium – 1 mg/kg/day in 3 doses, 30 minutes before meals.

In **acute toxic-infectious gastritis:**

- **Antibiotics** (aminoglycosides, fluoroquinolones, Biseptol) are given orally.

In severe cases, for **correction of water-electrolyte imbalance:**

- 5% glucose solution,
- physiological saline,
- potassium preparations are administered **parenterally**.

In **phlegmonous gastritis:**

- Laparotomy,
- Gastrotomy with drainage of the purulent focus,
- Antibiotics,
- Detoxification therapy are performed.

Prognosis

The course of **acute gastritis** is usually favorable and most often ends in complete recovery.

Exceptions:

- **Acute corrosive gastritis**, which may lead to **scar strictures and deformities**,
- **Acute Helicobacter pylori gastritis**, which later transforms into **chronic gastritis**.

Chronic Gastritis

Chronic gastritis is a recurrent inflammatory disease of the gastric mucosa and submucosa, characterized by cellular infiltration and disturbances in physiological regeneration. With inadequate treatment, chronic gastritis gradually leads to atrophy of the gastric glands and impairments in secretory, motor, and endocrine functions of the stomach.

In children, unlike in adults, only **10–15% of chronic gastritis cases** are considered a separate disease. Most often, **antral gastritis** occurs in combination with duodenitis or gastroduodenitis.

Chronic gastritis is **widely prevalent**, affecting **30–50% of the population**, and among pediatric gastrointestinal diseases, **gastric and duodenal disorders are the most common**, accounting for **58–65% of pediatric gastroenterological pathologies**, with **100–150 cases per 1,000 children**.

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Despite **Helicobacter pylori (HP)** being a leading etiological factor in chronic gastritis and ulcer disease in children, it is also necessary to consider **risk factors** that influence the onset and progression of the pathological process.

Exogenous Risk Factors for Chronic Gastritis

- **Dietary factors:** "fast food," spicy and fried foods, protein and vitamin deficiencies, use of food additives, irregular eating habits, etc.
- **Psychological-emotional factors:** stress, depression.
- **Environmental factors:** air pollution, nitrates in food, poor-quality drinking water.
- **Medications:** nonsteroidal anti-inflammatory drugs (NSAIDs) such as indomethacin, acetylsalicylic acid, corticosteroids, etc.
- **Parasitic infections:** mainly giardiasis.
- **Food allergies** and intolerance to certain products.
- **Oral and dental health problems.**
- **Hormonal dysfunctions.**

Endogenous Factors

- **HP infection.**
- **Bile reflux into the stomach.**
- **Endocrine disorders.**

The development of **modern therapeutic technologies, endoscopy, morphological examination of the gastric mucosa, and certain biochemical and bacteriological methods** has made it possible to classify chronic gastritis into independent types (Sydney classification):

Type A Gastritis (Endogenous, Autoimmune Gastritis)

- Autoimmune gastritis develops due to the production of **autoantibodies against parietal cells** of the stomach.
- Rare in children, accounting for **1–3% of cases**.
- Characterized by **primary atrophic changes** in the body and fundus of the stomach, **reduced gastric secretion**, and **increased gastrin levels** in the blood.

Type B Gastritis (Bacterial, HP-Associated Gastritis)

- **HP-associated gastritis** accounts for **80–85% of all gastroduodenal pathologies in children**.
- Chronic gastritis of this type is caused by **persistent HP infection**, usually localized in the **pyloric part of the stomach**.
- Infection can occur **orally** or during **endoscopic manipulations** such as gastric intubation.

Type C Gastritis (Reactive, Chemical, Reflux Gastritis)

- Type C gastritis develops due to **duodenogastric reflux** and the damaging effects of **bile acids on the gastric mucosa and epithelium**.
- A leading cause is **NSAID use**, which inhibits prostaglandin production and gastric mucus secretion, leading to erosions and microcirculatory disturbances.

Predisposing Factors in Children

- Chronic gastritis usually develops in children with **hereditary predisposition** and **exposure to the risk factors listed above**.
- Clinical manifestations depend on **the type of gastric secretion disturbance, the child's age, and individual characteristics**.

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Clinical Features Depending on Gastric Acid Secretion

Increased or Normal Acid Secretion (usually Type B gastritis)

- **Pain syndrome:** sharp, persistent, often associated with food intake. Morning pain is characteristic of fundal gastritis; evening pain, of antral gastritis. Night pain, seasonality, or diet have no clear correlation. Older children report **moderate epigastric and pyloroduodenal pain** during bowel movements.
- **Dyspeptic syndrome:** sour belching, bloating, intestinal rumbling, nausea, tendency to constipation.
- **Non-specific intoxication and asthenia:** variable; autonomic instability, irritability, rapid fatigue during mental and physical stress.

Reduced Acid Secretion (usually Type A gastritis)

- **Pain syndrome:** mild, diffuse, with epigastric discomfort. Postprandial fullness and heaviness are common; pain intensity depends on food type and volume. Mild diffuse pain may occur during bowel movements.
- **Dyspeptic syndrome:** dominant; belching, nausea, bitter taste in the mouth, reduced appetite, meteorism, variable diarrhea.
- Children may **avoid certain foods** (porridges, milk products).
- Non-specific intoxication is pronounced, with asthenia prevailing. Children may appear **pale**, have **impaired gastric digestion**, and in severe cases, develop **polyhypovitaminosis and malnutrition**.

Modern Assessment of Gastric Acid Function

- **Intragastric pH-metry** is the modern method to assess gastric acid secretion.
- Normal pH in the gastric body of children over 5 years is **1.7–2.5 when fasting**, and **1.5–2.5 after histamine stimulation**.
- The neutralizing pH in the antrum is **>5**, and the difference between body and antrum pH is normally **>2**.
- A **reduced pH difference** indicates impaired neutralizing function of the antrum and increased duodenal acidity (decompensated state).

Principles of Treating Type A Gastritis

The therapy is aimed at creating conditions for near-normal gastric function, serving as a compensatory treatment for atrophic processes in the gastric mucosa.

The main method of therapy is medicinal nutrition. During the acute stage, diet No. 1a is prescribed 5–6 times a day to ensure functional, mechanical, thermal, and chemical protection of the gastric mucosa. Foods that irritate the gastric mucosa (salty, smoked, fatty soups, marinades, spicy spices, fried meat and fish) should be excluded. Since patients often cannot tolerate sour milk, grape juice, or cream, these should be removed from the diet. Consumption of salt, strong tea, coffee, and their artificial substitutes is limited. Functional stimulation of the fundal glands is recommended along with anti-inflammatory measures. For this purpose, diet No. 2 or No. 15 is prescribed, which limits fatty types of meat and fish, hard-to-digest animal fats, fried potatoes, pastry-like foods, canned products, smoked products, and sweets. Milk is replaced by fresh dairy products (unsoured yogurt, cottage cheese, soft curd). Consumption of new and dark bread, pastries with added fat and sugar, cream, thick cream, cabbage, and gas-forming grapes is restricted.

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Anticholinergic and antacid agents are not prescribed for Type A gastritis. In the presence of pain and dyspeptic syndrome, medications such as metoclopramide, sulpiride, No-shpa, or butylscopolamine (Buscopan) can be taken orally or intramuscularly.

Herbal remedies for coating and soothing: infusions of plantain leaves, plantaglucid granules, chamomile, peppermint, red clover, and valerian root are widely used. Herbal decoctions are administered 1/3–1/2 cup 4–5 times a day, 2–4 weeks before meals.

To stimulate gastric secretory function, combined herbal remedies such as Gerbogastrin, Gerbion drops, plantain, and its medicinal products (pantaglucid) can be used.

For replacement therapy, hydrochloric acid, pepsin, and other agents can be used. To improve the trophicity of the gastric mucosa, agents that enhance microcirculation, protein synthesis, and reparative processes are prescribed: nicotinic acid preparations, vitamins B and C, methyluracil, and solcoseryl. In cases of concomitant megaloblast deficiency, vitamin B12 injections are additionally administered. During the resolution of the acute phase, physiotherapy methods and treatment with mineral waters can be applied. During remission, sanatorium-resort treatment is recommended.

Principles of Treating Type B Gastritis

In most cases, Type B gastritis is associated with Helicobacter pylori infection, so treatment is primarily aimed at eradicating this infection. At the same time, dietary nutrition remains important in the initial stages. Diet No. 1 is prescribed, which reduces mechanical and chemical irritation of the gastric mucosa. Meals are increased to 4–6 times per day.

If pain is pronounced during the acute phase, antispasmodics such as drotaverine (Drotaverine-KMP, No-shpa) or papaverine are prescribed. In some cases, cholinolytics such as atropine or Buscopan are effective. For increased gastric acidity, selective M-cholinolytic antisecretory agents such as pirenzepine (Gastrozepin) are prescribed for up to 4 weeks. Dosage: preschool children – 12.5 mg 2 times a day; school-age children – 25 mg 2 times a day.

H2-receptor blockers (famotidine, ranitidine) are given for up to 2 weeks. Children over 10 years receive famotidine 0.02–0.04 g at bedtime. After the antisecretory therapy course, complex antacids like Fosfalyugel or magnesium hydroxide-containing drugs (Almagel, Maalox) are used. Diosmectite (Smecta) can be prescribed at 6–9 g/day in water for children over 2 years.

Immediately after completing the course, residual dyspeptic and pain symptoms are treated with cytoprotective therapy, such as sucralfate (Antruksal, Venter) at a dose of 0.5–1.0 g 4 times a day (once at night) for 1 month.

To improve the trophicity of the gastric mucosa, cod liver oil and polyvitamin drugs may be prescribed for 3–4 weeks. In complex therapy, tranquilizers (diazepam, tazepam, etc.) can be used for 2–3 weeks. Herbal sedatives, such as valerian extract and Persen, are effective.

Principles of Treating Type C Gastritis

Type C gastritis (reflux gastritis), accompanied by motility disorders, duodenogastric, and gastroesophageal refluxes, is treated with metoclopramide (Reglan, Cerucal), which normalizes the closing function of the cardia. Metoclopramide also reduces gastroesophageal reflux, accelerates gastric emptying, and increases the resistance of the gastric mucosa to injury. Rare side effects include hyperkinesis, drowsiness, tinnitus, and dryness of the oral mucosa.

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Domperidone (Motilium) also normalizes gastric motility and has milder effects than Cerucal, with fewer side effects. Selective cholinomimetic cisapride is promising for gastroesophageal reflux (with caution in patients with cardiovascular disorders). To neutralize the aggressive effect of bile on the gastric mucosa, Fosfalyugel is prescribed; it adsorbs bile acids in addition to its antacid effect. Sucralfate (Ankrusal, Venter, Ulgastran, Sukreys) is another effective cytoprotective agent. Its mechanism in reflux gastritis involves forming complexes with the proteins of tissues adjacent to damaged mucosa, protecting the mucosa from acidic-pepsin effects. Diosmectite (Smecta) also has a cytoprotective effect.

Synthetic prostaglandin analogues (Cytotec, Arboprostil, etc.) are among the most effective cytoprotective agents. They reduce basal and stimulated gastric secretion and stimulate regeneration. However, these drugs can cause dyspeptic, reproductive, and allergic side effects and are thus used only in adolescents with erosive gastritis.

Chronic Gastro-Duodenitis

Chronic gastro-duodenitis is a persistent, recurrent disease with nonspecific structural changes (dystrophic, inflammatory, regenerative) in the gastric and duodenal mucosa, accompanied by various secretory and motor disturbances. It is the most common form of chronic gastroduodenal disease, comprising 58–74% of gastric and duodenal disorders.

Risk factors include predisposition and hereditary tendencies. Clinical presentation depends on the stage of inflammation, gastric secretory function, and motor-evacuatory disturbances of the stomach and duodenum. During acute phases, symptoms resemble peptic ulcer disease: pain, dyspeptic syndrome, and nonspecific chronic intoxication. Hepatobiliary, intestinal, and pancreatic pathologies may influence the clinical picture.

Pain Syndrome: Abdominal pain is characteristic, often stabbing, prolonged, and appearing on an empty stomach or 1.5–2 hours after a meal, located in the epigastrium, right costal margin, or around the navel. Pain increases after eating or physical activity.

Dyspeptic Syndrome: Frequent belching, bloating, prolonged nausea, postprandial heaviness, bitter taste, meteorism, constipation, or rarely diarrhea.

Nonspecific Intoxication Syndrome: Emotional lability, frequent headaches, irritability, general weakness, asthenia.

Modern treatment of chronic gastritis and gastro-duodenitis requires adequate diagnostics and paraclinical examination for optimal therapy.

Laboratory Examinations:

a) Mandatory (one-time):

- Complete blood count;
- Urinalysis;
- Total protein and blood protein fractions;

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- Tests for Helicobacter pylori (rapid urease test, bacteriological test, urea breath test, serological test (IFA), analysis of H. pylori antigen concentration in stool, PCR).

b) As needed:

- Occult blood test in stool (Gregersen reaction);
- Histological (cytological) examination of biopsy specimens using histopathological methods – "gold standard";
- Immunogram.

Instrumental Examinations and Diagnostic Criteria:

Mandatory:

- Fibroesophagogastroduodenoscopy with targeted biopsy and H. pylori express diagnostics (for erosive chronic gastroduodenitis – twice);
- Intragastric pH-metry (or fractional examination of gastric contents) – once;
- Abdominal ultrasound – once, to detect associated pathology.

As needed:

- X-ray examination of the stomach and duodenum (motor-evacuatory disorders, developmental anomalies);
- Reography;
- Other examinations according to the characteristics of associated pathology.

Principles of Treatment:

Treatment usually depends on the course of the disease, clinical-endoscopic changes, gastric secretory function, and the motor-evacuatory function of the stomach and duodenum. During exacerbations, treatment (inpatient or outpatient) should consider physical activity conditions. Dietary measures are prescribed (Diet No. 1 or No. 5) according to the patient's condition.

Complex therapy includes:

- **If** H. pylori **is present:** eradication anti-H. pylori therapy (usually for 7 days);
- **Antisecretory drugs:** H2 receptor antagonists for 2–3 weeks, selective M1 cholinolytics (pirenzepine) for 4 weeks. If necessary, cytoprotective and sorbent-effective antacids for 10–14 days (e.g., Smecta 1 packet 3–4 times/day);
- **Prokinetics** (domperidone) – prescribed in cases of reflux and duodenostasis for 10 days;
- **Spasmolytics** (drotaverine, papaverine, metasine) – for 7–10 days;
- **Sedatives and tranquilizers**, plant-based calming agents.

After stopping antisecretory drugs, reparants such as Smecta, sucralfate, likvitron, or flaxseed oil are prescribed for 4–6 weeks. Concurrent pancreatic pathology is treated with enzyme therapy. Adolescents may be prescribed intestinal spasmolytics (disetel, pinaverium bromide), laxatives (macrogol), and others if constipation is present.

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Physical therapy during exacerbations may include electrotherapy, heat therapy. Laser and magnet-laser therapy are used to normalize motor-evacuatory function of the stomach and duodenum and improve gastric mucosal trophism. Non-drug treatments include reflexotherapy.

During clinical remission: phytotherapy, balneotherapy, physiotherapy, therapeutic exercises (LFK), and non-traditional non-drug therapies.

Inpatient treatment duration is typically 21 days (for erosive chronic gastroduodenitis – 28 days). Inpatient treatment continuation is advisable in local gastroenterological sanatoriums.

Follow-up:

Children with chronic gastritis or chronic gastroduodenitis should be monitored for 5 years after the last exacerbation, with check-ups at least twice a year.

- Pediatrician: once every 6 months;
- Pediatric gastroenterologist: once a year;
- Fibrogastroduodenoscopy: once a year;
- For erosive chronic gastroduodenitis: examinations may increase to 3 times per year, endoscopic examinations – 2 times per year.

A patient can be discharged from dispensary observation after a 5-year clinical-radiological remission.

References

1. "Pediatrics" – T.A. Daminov, B.T. Khalmatova, U.R. Boboyeva – Tashkent, 2012.
2. Pediatric Gastroenterology: Guide / T.G. Avdeeva, Yu.V. Ryabukhin, L.P. Parmenova, N.Y. Krutikova, L.A. Zhlobnitskaya – Moscow: GEOTAR-Media, 2009.
3. Pediatrics: 2-volume textbook – 6th ed., revised – St. Petersburg: Piter, 2010.
4. Filimonov R.M. Adolescent Gastroenterology: Guide – 2nd ed., revised and supplemented – Moscow, 2008.
5. Shabalov N.P. Pediatrics. Textbook, vol. 2, 6th ed., revised – St. Petersburg, 2007.
6. Gastroenterology (National Guidelines) – GEOTAR-Media, 2008.

Internet sources:

1. www.tma.uz
2. www.zyonet.uz
3. info@minzdrav.uz
4. info@tma.uz
5. www.medbook.ru
6. www.medlincs.ru

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