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Chronic Gastritis, Gastroduodenitis in Children

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ABSTRACT

This article describes the origin, types and preventive measures of chronic gastritis, gastroduodenitis in children.

Chronic gastritis and gastroduodenitis (CG, CGD) is a chronic relapsing, progressive inflammatory-dystrophic lesion of the mucous membrane of the stomach and duodenum. In the structure of pediatric gastroenterological pathology, CG and CGD make up 50-65%. The incidence increases with age. Only 10-15% of children have an isolated lesion of the stomach or duodenum - gastritis or duodenitis, in 85-90% of cases the defeat of these organs is combined. The rise in incidence occurs at the age of 7-14 years. Girls get sick 1.5 times more often than boys [1,2].

Chronic gastritis and gastroduodenitis (CG, CGD) are multifactorial diseases. Matter:

- hereditary-constitutional predisposition to diseases of the digestive system - the indicator of family burden is 35-40%;
- infection with Helicobacter pylori (HP);
- nutritional errors (irregular, defective in composition, poor chewing, abuse of spicy food);
- chemical, including medicinal, effects;
- physical and psycho-emotional overload;
- food allergy;
- foci of infection, parasitosis and diseases of other digestive organs [3].

Against the background of hereditary predisposition and long-term exposure to these damaging factors, three main mechanisms of formation are possible, according to which CG is divided into three types:

A) endogenous autoimmune, due to the formation of antibodies to the lining cells of the stomach (gastritis A). This gastritis is in the structure of chronic hepatitis in children 1-3%.

B) exogenously infectious, which corresponds to HP-associated gastritis (gastritis B). In the structure of CG in children, it is about 85%;

C) exoendogenous, associated with irritation of the stomach with medications or severe duodenogastric reflux (gastritis C), in the structure of chronic hepatitis in children is 10-12%.

Classification of gastroduodenitis in children [Sydney system, 1990]

Gastro classification duodenitis in children [Sydney system, 1990] form	**Etiology**	**Localization**	**Endoscopy**	**Histology**	**Secretion**	**Period**
Spicy Chronic Special: granuloma-nauseous, eosinophil-ny	HP(-) HP(+) autoimmune Reactive Idiopathic	Antral fundamental pangastritis Duodenitis Antroduode-nit Common gastroduodenitis	Superficial Erosive Atrophic hyperplastic	Inflammation: a) weak b) moderate, c) severe atrophy: a) weak b) moderate c) expressed intestinal metaplasia	Normal Increased Reduced	Exacerbation Subremission Remission

Clinical picturedepends on the localization and prevalence of the inflammatory process. Unlike FRF, it is characteristic:

- ✓ periods of exacerbation lasting from several days to 2-3 weeks;
- ✓ stereotyped clinical manifestations during an exacerbation;
- ✓ the relationship of symptoms with the intake and nature of food;
- ✓ seasonal (spring-autumn) nature of exacerbations [4,5].

With antral gastritis and antroduodenitisthe disease proceeds according to the ulcer-like type. The leading symptom is abdominal pain:

A) occur on an empty stomach or 1.5-2 hours after a meal, sometimes at night;

B) decrease after eating;

C) are often accompanied by heartburn, sometimes sour belching, and occasionally vomiting, which brings relief [1].

Also typical:

1) pain on palpation in the epigastrium or pyloroduodenal zone;
2) tendency to constipation;
3) appetite is usually good;
4) the secretory function of the stomach is normal or increased;

5) at endoscopy - inflammatory-dystrophic lesion of the antrum of the stomach and duodenal bulb (antroduodenitis);

6) characteristic association with HP[7]. With fundic gastritis pain:

- ✓ occur after eating, especially after rich, fried and fatty foods;
- ✓ localized in the epigastrium and the navel;
- ✓ have a aching character;
- ✓ pass independently after 1 - 1.5 hours;
- ✓ accompanied by a feeling of heaviness, overflow in the epigastrium, belching with air, nausea, and occasionally vomiting of food eaten, bringing relief [6].

Of the other symptoms:

- ➢ the chair is unstable;
- ➢ reduced appetite and selective;
- ➢ on palpation, diffuse soreness in the epigastrium and the navel;
- ➢ the secretory function of the stomach is preserved or reduced;
- ➢ with endoscopy - damage to the fundus and body of the stomach, histologically, atrophic changes in the gastric mucosa can be detected;
- ➢ this type of CHD can be both autoimmune and associated with the condition of its long course [7,8].

Along with the main clinical forms of CGD, many atypical and asymptomatic ones are possible. Almost 40% of CHD is latent, the degree of morphological changes and clinical signs may not match.

Diagnosticsbased on the data of anamnesis and examination, includes the study of the secretory and motor functions of the stomach, endoscopy. Mandatory for CG and CGD is the definition of HP infection, histological examination of the gastric mucosa [2,3].

Endoscopy in CHD usually reveals focal or diffuse hyperemia of the mucous membrane, edema, hypertrophy of the folds, lymphofollicular hyperplasia, flat or elevated erosions can be detected. Sometimes pallor, thinning of the mucous membrane is detected, however, the presence or absence of atrophy and its severity can only be assessed histologically.

Differential Diagnosiscarried out with the same diseases as in the FRG, the secretory function of the stomach (according to pH-metry or fractional sounding) may be normal or impaired, more often elevated [9].

Motor disorders can be detected: spasm of sphincters, increased peristalsis, duodenogastric reflux, cardia insufficiency. The addition of a bacterial infection leads to the development of bacterial pneumonia against the background of the initial viral pneumonia and significantly aggravates the course of the disease [10].

Treatmentcomplex. During the period of exacerbation, physical and mental rest, therapeutic nutrition, medication and physiotherapy are necessary.

Nutrition is fractional, mechanically and chemically sparing with the exception of dishes that irritate the secretion.

With normal and increased acid-forming function, non-absorbable antacids are used: phosphalugel, megalac, maalox, protab. In cases of pronounced hyperproduction of hydrochloric acid, selective anticholinergics are used: gastrocepin, riabal. Other antisecretory agents (H2-histamine blockers, proton pump blockers) are used to a limited extent in children with CG and CGD.

In case of erosions, film-forming agents (sucralfate, denol) and synthetic analogues of prostaglandins PGE2 (misoprostol) are effective.

In cases of detecting helicobacteriosis, drug anti-infective therapy (see peptic ulcer disease) is indicated:

1) with erosive changes in the mucous membrane of the stomach and duodenum;
2) with persistent antral gastritis or gastroduodenitis;
3) in the presence of erosive and ulcerative diseases of the pyloroduodenal zone and stomach cancer in the family;
4) with proven HP virulence (serological detection of CagA and VacA proteins or morphologically - vacuolization of mucosal cells). Physiotherapeutic treatment includes inductothermia, electrophoresis of novocaine, papaverine, ozocerite or paraffin applications. Preventionconsists in creating conditions for rational nutrition, optimizing the daily regimen and the level of physical and psycho-emotional stress, sanitation of foci of infection, parasitosis [8].

Sanatorium treatment is indicated in the period of remission not earlier than 3 months after the exacerbation. Once a year, endoscopy and control of HP eradication are performed. Patients are removed from the dispensary registration after a complete clinical remission lasting for 3 years.

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