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Clinico-Functional Parallels And Mechanisms Of Co-Adjacency Between Chronic Gastroduodenal And Hepatobiliary Pathology In Children

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Abstract. Chronic digestive disorders in pediatric patients are characterized by a high frequency of concomitant lesions across various gastrointestinal tracts, among which interconnected impairments of the gastroduodenal zone and the hepatobiliary system hold a distinct position. This article presents the results of an analysis investigating the pathogenic co-adjacency of chronic gastroduodenitis and biliary tract dysfunction in the pediatric population. Key neurohumoral and motor-evacuation mechanisms supporting the mutual aggravation of inflammatory-destructive processes in the upper gastrointestinal mucosa and motor-tonic disorders of the biliary tract are thoroughly reviewed. The clinical necessity for a comprehensive, coordinated therapeutic approach to managing this combined pathology is substantiated in order to prevent disease chronicity and improve the overall quality of life in young patients.

Keywords: children, chronic gastroduodenitis, hepatobiliary system, gallbladder dysfunction, motor-evacuation disorders, duodenogastric reflux.

Introduction. In the structure of somatic pathology of childhood, diseases of the digestive system consistently occupy one of the leading places, while in recent years there has been a steady increase in the number of chronic, recurrent and combined forms of diseases [1,2,6]. The anatomical and functional unity of the organs of the upper digestive tract determines the systemic nature of the lesion: isolated involvement in the pathological process of only the stomach, duodenum (duodenum) or hepatobiliary system (GBS) is extremely rare in pediatric practice. Most researchers consider this region as a single gastrohepathoduodenal system, where the duodenum acts as a central "switchboard" and the main coordinator of digestive processes [3,4,8].

The close pathogenetic relationship between chronic gastroduodenitis (CGD) and biliary tract pathology is due to the commonality of their embryonic origin, innervation, and humoral regulation. The mucous membrane of the duodenum is a powerful endocrine organ that secretes cholecystokinin, secretin, motilin and somatostatin. Violation of the architecture and inflammation of the duodenal mucosa in HCG inevitably lead to a discoordination of the production of these regulatory peptides. Deficiency of cholecystokinin and secretin reduces the tone of the gallbladder and disrupts the coordinated work of the Oddi sphincter, inducing the development of dysfunctional disorders of the biliary tract (DRBT) [5,7,9].

In turn, motor-tonic disorders of the hepatobiliary system, accompanied by untimely or inadequate intake of bile into the intestinal lumen, cause duodenal hypertension, duodenostasis and, as a result, pathological duodenogastric reflux (DHR). The ingestion of bile acids and lysolecithin into the pyloric part of the stomach has a pronounced cytotoxic and detergent effect on the mucous membrane, destroying the mucosal-bicarbonate barrier and inducing chemical reflux gastritis [10,12,14]. Thus, a pathological vicious circle is forming, requiring a pediatrician and a pediatric gastroenterologist to have a deep understanding of the syntropy of these conditions in order to develop effective complex therapy regimens [11,13].

Materials and methods of research. A comprehensive examination of 120 children aged 7 to 17 years (average age - 12.4±1.8 years) with a verified diagnosis of chronic gastroduodenitis (CGD) in the acute phase was conducted on the basis of the pediatric gastroenterological hospital (or outpatient polyclinic department).

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Criteria for inclusion in the study: documented diagnosis of HCG (clinically, endoscopically and histologically); age from 7 to 17 years inclusive; informed voluntary consent of parents and children over 14 years old.

Exclusion criteria: peptic ulcer of the stomach and duodenum (duodenal ulcer); acute infectious diseases; severe concomitant somatic and congenital pathology of the digestive system (GBS malformations, liver cirrhosis, Crohn's disease); taking antisecretory, antibacterial drugs or choleretics during the last 4 weeks before the start of the examination.

All patients underwent a standardized examination, which included a clinical and anamnestic method - an assessment of the nature, intensity and localization of pain and dyspeptic syndromes. Esophagogastroduodenoscopy (EGDS) was performed on an Olympus device (Japan) to visually assess the nature of mucosal inflammation, the presence of duodenogastric reflux (DHR) and bile discharge. The diagnosis of Helicobacter pylori infection was carried out by rapid urease tests and histological method. Ultrasound examination (ultrasound) of the abdominal organs was performed using the Mindray ultrasound system (China). To assess the motor evacuation function of the hepatobiliary system (HBS), dynamic echocholecystography was performed using a standardized choleretic breakfast (sorbitol solution or egg yolks). Gallbladder volume was calculated before and 30, 45, and 60 minutes after stimulation. Gallbladder dysfunction was diagnosed with a change in its contractility - hypokinesia (reduction of less than 40% of the initial volume) or hyperkinesia (reduction of more than 60%). Laboratory methods included a detailed clinical blood test, a biochemical blood test to determine the activity of liver enzymes (ALT, AST, GGT, alkaline phosphatase) and levels of bilirubin fractions, cholesterol.

Statistical processing of the results was carried out using the StatSoft Statistica v application software package. 10.0 and IBM SPSS Statistics 26.0. Quantitative indicators are presented as M±m. Pearson's chi-squared ($\chi 2$) criterion was used to compare qualitative features, and the Student's t—test or the Mann-Whitney U-test were used for quantitative features. Spearman correlation analysis (rs) was used to assess the strength of the relationship. The critical significance level is assumed to be $p<0.05$.

The results and their discussion. According to the results of a comprehensive examination, concomitant pathology of the hepatobiliary system was verified in 86 out of 120 children with HCG, which accounted for 71.7% of cases. This confirms the systemic nature of the lesion of the gastrohepathoduodenal zone in the pediatric population. The structure of hepatobiliary disorders was dominated by dysfunctional disorders of the biliary tract (DRBT) — 62.5% (75 children), among which hypotonic-hypokinetic type was visualized in 64.0% (48 patients), and hypertensive-hyperkinetic type in 36.0% (27 patients). Chronic non-calculous cholecystitis in the subremission stage was detected in 9.2% (11 children).

Table No. 1.

№	Endoscopic markers of CGD	Patients with concomitant DRBT (n=75)	Patients without GBS pathology (n=34)	Pearson's criterion ($\chi 2$)	Significance level (p)
1.	Erosive antrum gastritis / duodenitis	32,0% (24 детей)	11,8% (4 детей)	5,12	p<0,024
2.	Duodenogastric reflux (DHR)	58,7% (44 детей)	17,6% (6 детей)	15,83	p<0,001
3.	Focal hyperplasia of the duodenal mucosa	26,7% (20 детей)	8,8% (3 детей)	4,52	p=0,033

The analysis revealed clear pathogenetic parallels. In children with concomitant hypokinetic disorders of the gallbladder, torpid, continuously recurrent course of HCG with severe dyspeptic syndrome (feeling of early satiety, heaviness in the epigastrium, bitter belching) was significantly more often recorded. On the contrary, with the hyperkinetic type of DRBT, severe pain prevailed in

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the clinic (acute cramping pains in the right hypochondrium and pyloroduodenal zone 20-30 minutes after eating).

Of particular interest is the high detection rate of duodenogastric reflux (58.7%) in children with biliary tract pathology. A decrease in the contractility of the gallbladder and discoordination of the Oddi sphincter lead to an untimely flow of bile into the duodenum. This initiates duodenal dyskinesia, intestinal paresis, and motor inversion. Aggressive components of bile (bile acids, lysolecithin), thrown into the stomach, damage the protective mucosal barrier, induce epithelial metaplasia and support chronic inflammation, independent of H. pylori infection.

Correlation analysis confirmed the presence of a direct moderate association between a decrease in the gallbladder ejection fraction according to ultrasound and the severity of endoscopic signs of inflammation of the antrum gastric mucosa ($r_s=0.46$; $p<0.01$). Thus, biliary insufficiency and motor abnormalities of GBS act as a powerful pro-visual factor aggravating the course of chronic gastroduodenitis in children.

Conclusion and practical recommendations. The conducted study convincingly proves the high incidence of syntropia and the close pathogenetic association of lesions of the upper digestive tract and the hepatobiliary system (GBS) in the pediatric population. The detection of concomitant biliary dysfunction in 71.7% of children with chronic gastroduodenitis (CGD) indicates a systemic nature of disorders within a single gastrohepathoduodenal zone. Motor-tonic disorders of the biliary tract do not just accompany inflammation of the mucous membrane, but act as a powerful factor in its maintenance and prolongation due to the induction of pathological duodenogastric reflux. The presence of a direct correlation between a decrease in the motor function of the gallbladder and the severity of inflammatory and erosive changes in the antrum of the stomach and duodenum dictates the need to review traditional isolated treatment approaches. The use of only standard regimens (for example, antihelicobacterium or isolated antacid therapy) for combined pathology is ineffective and leads to the formation of continuously recurrent forms of the disease.

The algorithm of complex therapy of combined pathology in children should include correction of motor evacuation disorders. With concomitant hypokinetic disorders of the biliary tract and the presence of duodenogastric reflux (DHR), it is a priority to prescribe prokinetics that restore physiological anterograde peristalsis, eliminate duodenostasis and prevent aggressive contents from being thrown into the stomach. In hyperkinetic type of biliary tract dysfunction, the use of selective myotropic antispasmodics is justified to relieve spastic pain syndrome and normalize the tone of the Oddi sphincter.

Since the components of bile have a pronounced detergent and cytotoxic effect on the gastric epithelium, cytoprotection of the mucous membrane and neutralization of bile acids should be performed, and therefore antacids and alginates should be included in therapy, which help reduce acidity and effectively adsorb bile acids and lysolecithin, creating a mechanical protective barrier.

As a pathogenetic therapy aimed at changing the physico-chemical properties of bile and reducing its aggressiveness to the mucous membranes, the use of ursodeoxycholic acid (UDCA) preparations is recommended, which replaces toxic hydrophobic bile acids with hydrophilic non-toxic fractions, reduces duodenal inflammation and improves the overall choleretic function of the liver. The dosage is calculated individually at the rate of 10 mg / kg of body weight once a day (before bedtime).

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