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Research Article

Effect of Helicobacter Pylori in Children with Nephrotic Syndrome to the Dysfunction of Stomach and Duodenum

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Abstract

In order to study the effect of helicobacter pylori on gastric and duodenal dysfunction in nephrotic syndrome in children, 126 children aged 7 to 17 years with various clinical variants of nephrotic syndrome were under our observation. The results of the study revealed that the detection rate of Helicobacter pylori in children with nephrotic syndrome largely depends on the depth of gastric and duodenal mucosa damage. It is detected in hypertrophic and erosive gastritis in 46 (71.8%) cases, in superficial changes in 18 (28.2%) cases. The HELIK-test is a simple and informative method for diagnosing Helicobacter pylori infection in gastric and duodenal disorders in children with nephrotic syndrome, it is convenient for medical practice and recommended for use in the primary examination of the patient, dynamic monitoring and monitoring of the effectiveness of therapy.

Keywords: Nephrotic Syndrome; Helicobacter Pylori; Children

Relevance of the topic

Nowadays, nephrotic syndrome (NS) in children is one of the topical topics of pediatrics, and the mechanisms of its development and the factors of its exacerbation are comprehensively studied by scientists [1,4,8-10]. In children, immunopathological processes in kidney diseases, hemostasiological and microcirculatory disorders, as well as long-term treatment with immunosuppressants and glucocorticosteroids lead to diffuse damage to cell membranes in the body, where the digestive system, including gastric and duodenal mucous membranes, is seriously affected, that is, the development of nephrogastrointestinal syndrome is serious. is attracting attention [2,3,11]. Analyzing the morphofunctional features of the digestive and excretory systems, the similar architecture of their histological structures, the transport system, the similar principles of functional management allow us to conclude about the generality of physiological, microbiological and immunological processes. In this regard, the structural-functional parallelism of both systems is reflected in the similarity of pathological processes [5,6,7]. In many cases, the origin of chronic diseases of the stomach and duodenum is related to the etiopathogenetic role of Helicobacter pylori (NR), and determining whether clinical and morphofunctional changes in the organs of the gastroduodenal tract in children with nephrotic syndrome is related to Helicobacter pylori (NR) is one of the urgent issues of medicine [12].

The purpose of the work

To study the effect of helicobacter pylori on gastric and duodenal dysfunction in nephrotic syndrome in children.

Research Materials and Methods

We observed 126 children aged 7 to 17 years with various clinical variants of nephrotic syndrome. Children were divided

into three groups. When dividing children into groups, first of all, clinical and laboratory characteristics of nephrotic syndrome were taken into account. The first group consisted of 34 people with the hormone-sensitive variant of nephrotic syndrome (GSNS). The second group consisted of 54 people with hormone-dependent and frequently recurrent (GB and frequently recurrent NS) variants The third group consisted of 38 children with hormone-resistant nephrotic syndrome (HRNS).

The control group was made up of 32 practically healthy children without chronic pathologies. The diagnosis of nephrotic syndrome was based on the international standards (proteinuria 1 g/m^2/milk, hypoalbuminemia less than 25 g/l, dysproteinemia, hypercholesterolemia, peripheral edema to anasarca) ISKDC, APN [(1974-2002)]. According to the guidelines of APN (Arbeitsgemeinchaft fur Paediatrisce Nephrology), ISKDC (International Study of Kidney Disease In Children) (1974-2009), nephrotic syndrome in children is diagnosed without biopsy if kidney function is preserved. We also formed a clinical diagnosis for the patients under our observation based on the above.

The level of infection with Helicobacter pylori was assessed using the HELIK test. This method is based on the assessment of the increase in the concentration of ammonia in the air in the patient's mouth after taking urea $^{12}C\ ^{1}X_4\ ^{14}H_2\ ^{16}O$ with a normal isotopic composition. Ingested urea, in the presence of NR in the stomach, is quickly hydrolyzed under the action of urease, which leads to an intensive formation of ammonia in exhaled air and an increase in its concentration. At first, the background concentration of ammonia in the air in the oral cavity of each patient was determined. For this, 2 liters of air was collected from the patient's oral cavity using an aspirator through a glass indicator tube filled with selective chemosorbent. Then the examined patient took 500 mg of urea with the specified composition in 15-20 ml of distilled water and rinsed his mouth with water. Air ammonia concentrations in the oral cavity were similarly reassessed for 10 minutes 2 minutes after urea ingestion. After administration of urea, an increase in the length of the stained column in the indicator tube was performed, where 1 mm column length is equal to 0.3 mg/m^3.

Results and Discussion

According to the results of the HELIK test, the study of the rate of endoscopic changes in the mucous membrane of the stomach and duodenum depending on the level of nephrotic syndrome activity showed that in the active stage of the disease, 110 (87.3%) of 126 children had pathologies of the gastroduodenal area, of which Endoscopic changes were detected in 78 (70.9%) cases, 8 (10.3%) in the first group of patients, 40 (51.3%) in the second group, 30 (38.4%) in the third group, and deep changes in 28 (16 observed in .7%) cases. The degree of increase in ammonia concentration in exhaled breath depends on the dose of urea taken, the higher the dose, the greater the increase. To evaluate this dependence, repeated studies were conducted in volunteer patients with repeated administration of 200 mg, 500 mg, 1 g and 2 doses of urea. A dose of 500 mg was found to be the minimum amount that provides a significant increase in the indicated effect without any unpleasant sensations in the patient. Repeat administration of the same dose of urea exactly reproduced the previous results.

Thus, it is important to have the exact dose of urea-500 mg when conducting the XELIK-test, because the test normative indicators were presented for this amount. In order to exclude other possible factors, in particular, the influence of food products on the test results, the studies were carried out at lunch.

In order to develop diagnostic criteria, as well as to evaluate the sensitivity and specificity of the method, we examined 110 children with nephrotic syndrome, aged 7 to 18 years, who received glucocorticoids for more than 6 months, had various disturbances in the stomach and duodenum, and gastrointestinal 32 children without clinical and endoscopic signs of damage in the upper parts of the tract formed the control group. In children in the control group, the initial concentration of ammonia in the air in the oral cavity S_1 averaged 0.34 ± 0.12 mg/m^3, the average increase in concentration after taking urea was 0.09 ± 0.02 mg/m^3, and the concentration of ammonia in S2 averaged 0.44 ± 0 equal to 18 mg/m^3. In 46 patients with HP (-) negative nephrotic syndrome and various gastroduodenal pathologies, the background concentration of ammonia C_1 in the oral cavity air was on average 0.36 ± 0.18 mg/m^3. After taking urea, the average value of S2 was 0.54 ± 0.21 mg/m^3, the average increase in concentration was DC - 0.18 ± 0.07 mg/m^3, which was little different from the values of the control group (P > 0,1). In 64 patients with NR (+) positive nephrotic syndrome and various gastroduodenal pathologies, the background concentration of ammonia (C_1) was 0.47 mg/m^3, which was significantly different from the values of the control group. After taking urea, a significant increase in ammonia concentration was noted: S 2 - 1.71 mg/m^3, DC - 1.2 mg/m^3. The difference between the control group and NR (+) positive patients had high reliability (R < 0.01) (Table 1).

N = 142	A F K		A O`O`	P
	C_1	C_2	DC	
Control group, n = 32	0,34 ± 0,12 мг/м^3	0,44 ± 0,18 мг/м^3	0,09 ± 0,02 мг/м^3	P > 0,1
HP (-) negative, n = 46	0,36 ± 0,18 мг/м^3	0,54 ± 0,21 мг/м^3	0,18 ± 0,07 мг/м^3	P > 0,1
HP (+) positive, n = 64	0,47 мг/м^3	1,71 мг/м^3	1,2 мг/м^3	P < 0,01

Table 1: HELIK test indicators in nephrotic syndrome M ± m.

Note: R-differences are significant compared to control group scores.

AFK is the background concentration of ammonia.

Average growth of AO'O'-ammonia

C1 AFK before taking urea

AFK after taking S2 Urea

DC- AO'O' after taking urea.

Helicobacter pylori was laboratory confirmed in 64 of the 78 children with NS and endoscopic changes in our study along with gastric and duodenal lesions. The rate of recording of Helicobacter pylori is directly related to the degree of damage to the gastric mucosa, and it is high in hypertrophic and erosive gastritis - 46 cases (71.8%), and low in 18 cases (28.2%) in superficial changes.

Conclusions

- The detection rate of Helicobacter pylori in children with nephrotic syndrome largely depends on the depth of damage to the mucosa of the stomach and duodenum. It was found in 46 (71.8%) cases of hypertrophic and erosive gastritis, and 18 (28.2%) cases of superficial changes.
- HELIK-test is a simple and informative method of diagnosing Helicobacter pylori infection in gastric and duodenal disorders in nephrotic syndrome in children, it is convenient for medical practice and is recommended for use in primary examination of the patient, dynamic monitoring and control of therapy effectiveness.

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