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ROLE OF AGGRESSIVE-PROTECTIVE FACTORS IN LESIONS OF THE GASTRODUODENAL ZONE IN ADOLESCENTS

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ABSTRACT

The high frequency of pathology of the digestive system in children, adolescents and adults, the absence of a tendency to decrease it remains one of the urgent problems of internal medicine. The aim of this study was to study the role of aggressive and protective factors in the occurrence of gastric ulcer and duodenal ulcer in adolescents.

The objective of the study. The study of the peculiarities of the relationship between aggressive and protective factors in the mucous membrane of the gastroduodenal zone with its various lesions in adolescents. Material and methods. Clinical studies were carried out in the City adolescent dispensary of the city of Tashkent on four groups of patients. The first group, which was the main one, consisted of 40 patients with erosive gastritis at the age from 14 to 18 years old, including 19 boys and 21 girls. The second group, also the main one, included 36 adolescents with duodenal ulcer at the age from 14 to 18 years old, including 19 boys and 17 girls. The third group, which served as a comparison group, included 24 adult patients with erosive gastritis at the age of 18 to 25 years, of which 23 were men and 23 women. The fourth group, also a comparison group, consisted of 24 adults with duodenal ulcer at the age from 18 to 25 years, including 18 men and 6 women.

Conclusion. A feature of damage to the protective mucous barrier of the gastroduodenal zone in gastric ulcer in adolescents with Helicobacter pylori is a lack of fucose, the main component of insoluble glycoproteins.

Key words: Peptic ulcer and duodenal ulcer, adolescents, Helicobacter pylori, protective mucous barrier.

INTRODUCTION

Introduction. Peptic ulcer is a polietiological, 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 (infection with Helicobacter pylori, long-term error in nutrition, psycho-emotional stress, bad habits) a genetic predisposition to the development of ulcer is realized [5,13].

As you know, damage to the mucous membrane of the gastroduodenal zone is a consequence of the violation of the relationship between the factors of "aggression" and "protection". Therefore, in damage to the mucous membrane of the gastroduodenal zone, along with HP, the state of other factors, such as the protective mucous barrier, lipid peroxidation of cell membranes, the state of regenerative processes in the mucosa, is of no small importance. [3,7,11]. The study of the peculiarities of changes in these factors in the presence of HP and the development of optimal schemes for etiopathogenetic therapy remain an urgent issue in gastroenterology. At this stage, we studied the state of the mucous barrier of the stomach, lipid peroxidation and regeneration in the gastric mucosa in different HP genotypes in adolescents with erosive gastritis and duodenal ulcer.

As noted earlier, the state of the gastric mucous barrier was studied by determining the content of insoluble mucous gel in gastric juice and carbohydrate and protein fractions of insoluble glycoproteins in the NSH suspension (Table 1).

Table 1
The content of NSH in gastric juice at
different HP genotypes in adolescents with erosive gastritis

Patient group	Number of patients	NSH, mg / ml
Control group	9	19,55±1,0
Teenagers with EG:		
HP type I	13	43,07±1,2*
HP type II	18	$30,06\pm1,3^{*a}$
Control group	8	25,68±1,2
Adults with EH:		
HP type I	18	65,63±2,1*
HP type II	4	63,27±2,7*

Note. * - P <0.05 compared to control; a - P <0.05 compared with the group of patients with type I HP.

As can be seen from Table 1, with erosive gastritis in adolescents, the content of NSH in the gastric juice significantly increases, and the revealed changes depend on the HP genotype. So, in adolescents with type I HP, an increase in the content of NSH was observed by 120.3% of that in the control group, and in patients with type II - by 53.7%. A more significant increase in NSH occurred in adults. In patients with type I HP, the content of NSH was higher by 155.6%, and in patients with type II HP - by 146.4%. No special differences in the content of NSH between types I and II were found.

The results of studying the content of GHP fractions in a suspension of a mucous gel for various HP genotypes in adolescents with erosive gastritis are shown in Table 4.2.

As can be seen from the table, in adolescents, the increase in the content of NSH in the gastric juice is due to the insufficient content of fucose in the insoluble glycoprotein. In patients with type I HP, the content of fucose was 53.6% lower than in the control group, while the content of sialic acids and total protein did not change. Similar, but less significant changes were observed in adolescents with type II EH. In this group, the fucose content was 35.6% lower than the control.

In adults, the content of fractions also varied depending on the type of HP, but they showed a decrease in the level of not only fucose, but also sialic acids and total protein.

Thus, a feature of damage to the protective mucous barrier of the gastroduodenal zone during erosive gastritis in adolescents with Helicobacter pylori is a lack of fucose, the main component of insoluble glycoproteins. In genotype I HP, the lack of fucose is more pronounced than in genotype II.

Table 2 presents the results of a comparative study of the content of NSH in gastric juice with various types of HP in adolescents with duodenal ulcer.

The content of NSH in gastric juice at different types of HP in adolescents with DU

Number of patients Patient group NSH, mg / ml Control $19,55\pm1,0$ Adolescents with DU: HP type I 22 54,62±2,0* 38,91±1,4* HP type II 8 8 Control $25,68\pm1,2$ Adults with duodenal ulcer: HP type I 19 84,44±2,1* HP type II 4 80,15±2,8*

Note. * - P < 0.05 compared to control.

Table 2

The presented data show that in adolescents with DU, the protective mucous barrier of the stomach undergoes more significant changes, depending on the HP genotype.

In adolescents with duodenal ulcer with genotype I HP, the content of NSH exceeded the control by 179.4%. In the case of genotype II HP, the content of NSH was higher by 99.0%. In adults, there was no particular dependence of the change in NSH on the HP genotype, and the revealed changes were much greater than in adolescents [2,8].

The results of studying the content of fractions of insoluble glycoproteins in different types of HP in adolescents with DU are presented in Table 3.

Table 3 MDA content in gastric juice in adolescents with erosive gastritis in association with different HP genotypes

Patient group	Number of patients	MDA, mg / ml
Control	9	0,211±0,060
Teenagers with EG:		
HP type I	13	0,441±0,013*
HP type II	18	0,389±0,012*
Control	8	0,323±0,010
Adults with EH:		
HP type I	18	0,783±0,013*
HP type II	4	0,753±0,015*

Note. * - P < 0.05 compared to control.

As can be seen from the table, the content of fractions of insoluble glycoproteins in DU in adolescents significantly differ from the changes in the group of patients with EH. In this group of patients, a significant decrease in the content of not only fucose, but also sialic acids was observed. In patients with genotype I HP, the level of sialic acids was lower than the control by 36.7%, and fucose by 67.2%. Some decrease in protein content turned out to be statistically insignificant. The results obtained in the group of adolescents with genotype II did not particularly differ from the results of the group with genotype I. In this group, the content of sialic acids decreased by 33.1%, and fucose by 57.7%. In contrast to adolescents, adults showed a more pronounced decrease in all fractions of insoluble glycoproteins, which also did not particularly depend on the HP genotype.

Thus, the mild severity of duodenal ulcer in adolescents is due to a moderate impairment of the protective function of the mucous barrier, which is more pronounced when associated with genotype I of HP. Violation of the protective function of the mucous barrier is characterized by a deficiency of sialic acids and fucose, the degree of decrease in which does not depend on the HP genotype. Table 3 shows the results of studying the content of malondialdehyde in gastric juice in adolescents with erosive gastritis with different HP genotypes [2,8].

As can be seen from the table, during EH in both adolescents and adults, the processes of lipid peroxidation in the mucous membrane of the gastroduodenal region are enhanced. The increase in LPO in adolescents was less intense and, like in adults, did not depend on the HP genotype.

In adolescents with EG with genotype I HP, we observed an increase in MDA content by 109.0%, and in adults - by 142.4%. In adolescents with genotype II HP, the MDA content exceeded the control by 84.3%, and in adults - by 133.1%. In both adolescents and adults, we did not observe a particular dependence of the increase in the MDA content on the HP genotype.

Table 4 shows the results of examination of patients with duodenal ulcer. As can be seen from the table, in adolescents with DU, the intensity of LPO was higher. In patients with type I HP, the MDA content was higher than the control by 194.3%, and in type II - by 188.1%, while in the EH these indicators were 109.0 and 84.3%, respectively. Similar changes were observed in adults with duodenal ulcer. The MDA content in patients with DU was significantly higher than in EG.

Table 4
MDA content in gastric juice in different HP genotypes
in adolescents with DU

Patient group	Number of patients	MDA, mg / ml
Control	9	0,211±0,060
Adolescents with DUA:		
HP type I	22	0,621±0,017*
HP type II	8	0,608±0,022*
Control	8	0,323±0,010
Adults with duodenal ulcer:		
HP type I	19	0,981±0,020*
HP type II	4	0,881±0,022*

Note. * - P < 0.05 compared to control.

Thus, along with Helicobacter pylori aggression, one of the reasons for the dysfunction of the protective mucous barrier is the intensification of LPO processes in the mucous membrane of the gastroduodenal zone. In adolescents with erosive gastritis and DU, LPO is less intense than in adults and does not depend on the HP genotype.

The results of studying the content of free hydroxyproline in gastric juice with various types of HP in adolescents with erosive gastritis and DU are shown in Tables 5 and 6.

Table 5
The content of free hydroxyproline in gastric juice at different HP genotypes in adolescents with EH

Patient group	Число больных	Free hydroxyproline, mg / ml
Control	9	4,14±0,16
Teenagers with EG:		
HP type I	13	5,17±0,21*
HP type II	18	4,92±0,14*
Control	8	6,16±0,19
Adults with EH:		
HP type I	18	11,53±0,64*
HP type II	4	9,46±0,44*

Note. * - P < 0.05 compared to control.

Table 6

The content of free hydroxyproline in gastric juice at different HP phenotypes in adolescents with DU

Patient group	Number of patients	Free hydroxyproline, mg / ml
Control	9	4,14±0,16
Adolescents with DUA:		
HP type I	22	5,29±0,30*
HP type II	8	5,14±0,41*
Control	8	6,16±0,19
Adults with duodenal ulcer:		
HP type I	19	15,34±0,63*
HP type II	4	16,37±0,89*

Note. * - P < 0.05 compared to control.

As can be seen from the table, in adolescents with EH in the mucous membrane of the gastroduodenal region, the regenerative processes do not change. In adolescents with EH with type I HP there was an increase in the content of free hydroxyproline by 24.8%, and in patients with type II HP only by 18.8%. These data did not differ significantly from the results of the control group.

A pronounced decrease in regeneration was noted in adults with EH. In this group, in patients with genotype I HP, the content of free oxyproline in gastric juice exceeded the control by 87.2%, and in patients with genotype II by 53.6%.

Similar changes were observed in the group of patients with DU. In adolescents with duodenal ulcer with both genotypes I and II HP, a slight change in the content of free hydroxyproline was recorded. Thus, in patients with genotype I HP, the content of oxyproline was higher than the control by 24.9%, and in patients with genotype II HP, by 24.1% (P <0.05). These results were fundamentally different from those of the group of adults. In adults with duodenal ulcer, there was a significant increase in the content of free hydroxyproline. At the same time, no particular dependence of these changes on the HP genotype was revealed.

Thus, one of the causes of erosive damage to the gastric mucosa in adolescents, along with the Helicobacter pylori association, is the insufficient functioning of the protective mucous barrier due to a fucose deficiency in the composition of insoluble glycoproteins. In genotype I HP, the lack of fucose is more pronounced than in genotype II. In contrast to adults, LPO as a factor of aggression increases to a moderate degree. Violation of the relationship between aggressive and protective factors in erosive gastritis is not accompanied by changes in regenerative processes in the mucous membrane of the gastroduodenal zone.

The mild severity of peptic ulcer disease in adolescents is due to moderate impairment of the protective function of the mucous barrier and intensification of lipid peroxidation. Dysfunction of the mucous barrier is a consequence of the deficiency of sialic acids and fucose in the composition of insoluble glycoproteins. The degree of decrease in carbohydrate components in the composition of the mucous barrier and the increase in LPO do not depend on the HP genotype. In contrast to adults, in adolescents with DU, the regenerative processes in the gastroduodenal mucosa remain within the normal range for a given age.

REFERENCES

- 1. Abdullaev U.R., Kayumov T.Kh. The effectiveness of various options for anti-Helicobacter pylori therapy in patients with gastroduodenal ulcers // Med. zhurn. Uzbekistan. 2008. No. 3. S. 31-33.
- 2. Abdullaeva D.A. Chronic gastroduodenal pathology in children (prevalence, risk factors, features of psychological status): Author's abstract. dis. ... Cand. honey. sciences. Tashkent, 2006 .-- P. 22.
- 3. Abdullaeva D.A., Akhmedova I.M. Assessment and correction of the quality of life of children with chronic gastroduodenal pathology // Pediatrics of monavii kirralari. Fan va amaliyot: Republic of ilmiy-amaliy anjumani materialari. Tashkent, 2008. S. 27-28.
- 4. Abdulkhakov R.A., Chernov V.M., Nasybullina E.R. and other Prevalence of various strains of Helicobacter pylori in patients with gastric ulcer and duodenal ulcer // Expert. and wedge. gastroenterol. 2003. No. 1. S. 78-79.
- 5. Alexandrova V.A., Kozlova I.P. Controversial and unresolved issues of Helicobacter pylori infection in children // Lech. doctor. 2002. No. 11. S. 70-74.
- 6. Alyavi A.L., Karimov M.M., Spiridonova A.Yu. Antisecretory activity of proton pump inhibitors of the first and second generation in the treatment of patients with duodenal ulcer // Ozbekiston tibbiyot jurnali. 2008. No. 3. S. 29-31.
- 7. Andersen L.P. New species of the genus Helicobacter in humans // Ros. zhurn. gastroenterol., hepatol., coloproctol. 2003. No. 2. S. 81-84.
- 8. Maev I.V., Kucheryavyy Yu.A., Gadzhieva M.R. New approaches to the diagnosis and treatment of chronic gastric erosions // Ros. zhurn. gastroenterol., hepatol., coloproctol. 2003. No. 1. S. 43-49.
- 9. Vostrikov G.P. The role of hereditary factors in the etiology of chronic duodenitis and duodenal ulcer // Expert. and wedge. gastroenterol. 2002. No. 2. S. 9-12.
- 10. Vostrikov G.P., Speransky M.D. The prevalence of gastritis, duodenitis, gastric ulcer and duodenal ulcer in Moscow over the past 5 years // Expert. and wedge. gastroenterol. 2002. No. 2. P. 56.
- 11. Gyulumyan O.N., Semin S.G., Safonov A.B. et al. Treatment regimens for familial Helicobacter pylori infection on an outpatient basis // Pediatrics. 2007 .-- T. 86 .-- S. 32-36.
- 12. Peptic ulcer disease Review Article. Paediatrics and Child Health, Volume 24, Issue 11, November 2014, Pages 485-490. Arun Nanjundaraje Urs, Priya Narula, Mike Thomson

- 13. Clinical guidelines for the diagnosis and treatment of peptic ulcer disease. Moscow 2013. 39 pages
- 14. Shay H., Sun D.C.H. Etiology and pathology of gastric and duodenal ulcer // In: H.L.Bockus "Gastroenterology" Vol.1. Philadelphia-London, 1968. P.420-465.
- 15. Koletzko S., Richy F., Bontems P., Crone J., Kalach N., Monteiro L. et al. Prospective multicenter study on antibiotic resistance of Helicbacter pylori strains obtained from children living in Europe // Gut. 2006; 55 (12): 1711–1716.
- 16. Jones N., Fallone C., Flook N., Sherman P., Smaill F., van Zanten S. J. et al. Consensus Conference: Update on the Management of Helicobacter pylori. An evidence-based evaluation of Helicobacter pylori infection and clinical sequelae in children and adolescents // Can J Gastroenterol. 2005; In Press.
- 17. Khurana R., Fischbach L., Chiba N., van Zanten S. V., Sherman P. M., George B. A. et al. Meta-analysis: Helicobacter pylori eradication treatment efficacy in children // Aliment Pharmacol Ther. 2007, Mar 1; 25 (5): 523–536.
- 18. Shcherbakov A.P., Shcherbakov P.L. Management of Helicobacter pylori infection in children (scientifically based recommendations of ESPGHAN and NASPGHAN 2010) / J. Attending physician, 2011, No. 6