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BIOLOGICAL ROLE OF HUMAN BACTERICIDAL/PERMEABILITY-INCREASING PROTEIN IN HELICOBACTER PYLORI–ASSOCIATED UPPER GASTROINTESTINAL TRACT DISEASES IN CHILDREN

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Summary.

The prevalence of Helicobacter pylori infection (HP) varies across geographic regions and remains heterogeneous. Approximately one-third of children worldwide are infected with H. pylori, with primary acquisition occurring during childhood. The immune response to inflammatory diseases is mediated by the synthesis and release of antimicrobial peptides, which serve as protective factors. Human bactericidal/permeability-increasing protein (hBPI) is among these antimicrobial peptides, and its mechanisms of antimicrobial defense are exerted through multiple pathways. Investigation of the relationship between hBPI levels and morphological changes in the upper gastrointestinal mucosa in H. pylori–associated diseases in children may enhance understanding of the biological significance of this antimicrobial peptide in the host immune response.

Objectives of the study. *To determine the serum levels of human bactericidal/permeability-increasing protein in children with upper gastrointestinal tract diseases associated with H. pylori infection and to elucidate its role in the development and progression of the infectious process.*

Material and Methods. *A total of 60 patients aged 10 to 17 years were examined. Subgroup 1 comprised 30 children with gastroduodenal diseases associated with H. pylori. Subgroup 2 included 30 children with gastroduodenal diseases not associated with H. pylori. The control group consisted of 20 apparently healthy children matched for age and gender. Diagnosis was established by laboratory, endoscopic, and histological examinations. Determination of hBPI levels levels in serum was performed by enzyme-linked immunosorbent assay using a commercially available Human BPI ELISA Kit (Elabscience Biotechnology, Houston, TX, USA). The study protocol was approved by the Bioethics Committee of the Zaporizhzhia State Medical and Pharmaceutical University. The study was conducted in accordance with ethical standards and principles for medical research involving human subjects and adhered to fundamental bioethical norms. Informed consent was obtained from all participants or their legal guardians prior to enrollment (Protocol No. 3, dated October 28, 2024). Statistical analysis was performed using the licensed Statistica for Windows software package, version 13.0 (serial number JPZ8041382130ARCN10-J). Data are presented as arithmetic mean (M), standard deviation (σ), and standard error of the mean (m) for participants stratified into categories that met the criteria of normality. Associations between variables were evaluated using Spearman's rank correlation coefficient (r). To assess the significance of differences between the compared groups, the Student's t-test for small samples was used, supplemented by the nonparametric Mann–Whitney U test when appropriate. Differences were considered statistically significant at $p < 0.05$. The research was conducted under the departmental project entitled «Predicting the course of the most common inflammatory diseases of childhood» (registration number 01121U107520, 2020-2026).*

Results. *Serum levels of human bactericidal/permeability-increasing protein (hBPI) in the study group were 2.2 times higher than those in the control group ($p < 0.05$). In patients with erosive gastroduodenitis, hBPI levels were increased 3-fold compared with the control group ($p < 0.05$). Gender differences were observed: in girls with H. pylori infection, hBPI levels were elevated by 80% ($p < 0.05$), whereas in boys with H. pylori infection, serum hBPI levels were increased more markedly, by 300% ($p < 0.05$). In the presence of H. pylori infection, increased inflammatory activity was associated with elevated hBPI levels ($p < 0.05$). In the absence of H. pylori, the highest serum hBPI levels were observed in cases with grade 1 lymphoplasmacytic infiltrate ($p < 0.05$) and with minimal inflammatory changes ($p < 0.05$).*

Conclusions. *The course of upper gastrointestinal tract diseases associated with H. pylori in children is accompanied by increased hBPI levels, which correlate directly with the severity of mucosal lesions and the extent of mucosal involvement. Gender differences in hBPI levels were identified, with girls in the control group exhibiting higher hBPI levels than boys.*

Keywords: *Helicobacter pylori; Diseases, Gastroduodenal; Child; Adolescent; Diagnosis; Endoscopy; Bactericidal/Permeability-Increasing Protein.*

Introduction

Despite advances in modern medicine, the global prevalence of *Helicobacter pylori* infection remains heterogeneous across geographic regions, with no consistent downward trend in the pediatric population [1,2]. Approximately one-third of children worldwide are infected with *H. pylori*, and primary acquisition occurs predominantly during childhood [3]. Accordingly, elucidation of the pathogenesis of upper gastrointestinal tract diseases and the host immune response to this pathogen and associated inflammation continues to represent a priority in pediatric gastroenterology [4].

The immune response elicited by inflammatory diseases of the gastroduodenal region includes the synthesis and release of antimicrobial peptides (AMPs), which serve as protective effectors and facilitate eradication of pathogenic bacteria [5]. Human bactericidal/permeability-increasing protein (hBPI) constitutes one such AMP; its antimicrobial activity is exerted via bactericidal and permeability-increasing mechanisms [6,7]. The release of this antimicrobial peptide occurs from azurophilic granules of neutrophils [8] and from epithelial cells in response to tissue injury or inflammation [9]. Beyond its direct antimicrobial effects, hBPI possesses anti-inflammatory properties [10,11]; however, excessive synthesis and release may indirectly contribute to the perpetuation of chronic inflammation [12].

Evaluation of the associations between serum levels of hBPI and morphological changes in the upper

gastrointestinal mucosa in *Helicobacter pylori*–associated diseases in children may enhance understanding of the biological significance of this antimicrobial peptide in the host immune response.

Purpose: To determine serum levels of hBPI in children with upper gastrointestinal tract diseases associated with *H. pylori* to elucidate its role in the development and progression of the infectious process.

Materials and Methods: The study included 60 children aged 10 to 17 years, 11 months, and 29 days (25 girls and 35 boys) hospitalized between 2022 and 2024. All patients presented with relevant complaints and underwent fibroesophagogastroduodenoscopy (FEGDS), rapid urease test (RUT), and histological examination of gastric and duodenal mucosal biopsies.

Clinical diagnoses were established in accordance with the Standards of Medical Care «Peptic ulcer disease in adults and children» (Order of the Ministry of Health of Ukraine No. 1514 dated August 25, 2023) [13]. Endoscopic examinations were performed using an MTW-Endoskopie W. Haag KG endoscope, with five biopsy specimens obtained from different gastric sites for subsequent analysis [14].

H. pylori infection was detected by rapid urease test using a commercial kit (Ure Hp-test; Erba Lachema, Czech Republic) [15] and by histological examination [16].

The degree of *H. pylori* bacterial load, density of lymphoplasmacytic infiltrate, and inflammatory activity were graded histologically according to the updated Sydney system (1994) [17].

The study group comprised 60 children with gastroduodenal diseases: chronic gastroduodenitis was diagnosed in 34 patients (56.7%), and gastric or duodenal ulcers in 26 patients (43.3%).

Patients were stratified into two subgroups based on the presence of *H. pylori* infection. Subgroup 1 included 30 children (17 boys and 13 girls; mean age 14.72 ± 1.9 years) with *H. pylori*–associated gastroduodenal disease. In this subgroup, chronic gastroduodenitis was diagnosed in 23 cases (76.7%), and mucosal disruption in 7 children (23.3%): duodenal bulb ulcer in 1 child (3.3%) and erosive gastroduodenitis in 6 children (20.0%). Histological examination revealed mild chronic inflammation in 10 children (33.3%), moderate in 17 (56.7%), and severe in 3 (10.0%). Subgroup 2 consisted of 30 children (18 boys and 12 girls; mean age 14.76 ± 2.5 years) with gastroduodenal diseases not associated with *H. pylori.* In this subgroup, chronic gastroduodenitis was diagnosed in 11 cases (36.7%), and mucosal disruption in 19 children (63.3%): gastric or duodenal ulcer in 8 patients (26.7%) and erosive gastroduodenitis in 11 children (36.7%). Histological examination showed minimal inflammatory changes in 13 children (43.3%), mild chronic inflammation in 12 (40.0%), and moderate chronic inflammation in 5 (16.7%). The control group comprised 20 apparently healthy children matched for age and gender.

Determination of hBPI levels levels in serum was performed by enzyme-linked immunosorbent assay using a commercially available Human BPI ELISA Kit (Elabscience Biotechnology, Houston, TX, USA).

The study protocol was approved by the Bioethics Committee of Zaporizhzhia State Medical and Pharmaceutical University. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and adhered to fundamental bioethical norms. Informed consent was obtained from the legal guardians of all participating children and, where age-appropriate, from the children themselves prior to enrollment.

Statistical analysis was conducted using Statistica for Windows, version 13.0 (StatSoft Inc., Tulsa, OK, USA; serial number JPZ8041382130ARCN10-J). Normally distributed continuous variables are expressed as arithmetic mean (M) ± standard deviation (σ) or ± standard error of the mean (m) for participants stratified into categories that met the criteria of normality. Associations between variables were assessed using Spearman's rank correlation coefficient (r). To assess the significance of differences between the compared groups, the Student's t-test for small samples was used, supplemented by the nonparametric Mann–Whitney U test when appropriate. Differences were considered statistically significant at $p < 0.05$.

The research was conducted under the departmental project entitled «Predicting the course of the most common inflammatory diseases of childhood» (registration number 01121U107520, 2020-2026).

Results and discussion

Serum hBPI concentrations in children with upper gastrointestinal tract diseases are summarized in Table 1. As indicated in the table, the presence of these diseases was associated with a significant elevation in serum hBPI levels in the main group compared with the control group (2.2-fold increase, $p < 0.05$). Stratification by *Helicobacter pylori* status revealed no statistically significant difference in hBPI levels between the two subgroups ($p > 0.05$). Nevertheless, a consistent tendency toward higher hBPI concentrations was observed in the subgroup with *H. pylori*–associated disease.

Table 1

Serum hBPI concentrations in children with upper gastrointestinal tract diseases (M ± m).

Parameter	Main group n=60	Subgroup 1 n=30	Subgroup 2 n=30	Control group, n=20
Serum hBPI level (ng/ml)	13.2±1**	14.4±1.2**	10.9±1.5*	5.9±1
Serum hBPI level (ng/ml) in girls	13.6±1.5*	15.2±1.9*	11.2±1.9	8.4±1.4
Serum hBPI level (ng/ml) in boys	12.9±1.5**	13.6±1.7**	10.5±1.3**	3.4±0.5
Coefficient of variation (C_v, %)	45.6%	39.3%	57.5%	34%

Notes:

** – p<0,05 – compared with the parameters of control group*

***– p<0,01 – compared with the parameters of control group*

Given the heterogeneity of the study groups, including gender variability (C_v 34-57.5%), the sample was stratified by gender.

The findings, although somewhat unexpected, underscored the pathogenetic role of hBPI underlying gastroduodenal pathology. A clear gender difference in serum hBPI levels was observed in the control group ($p < 0.05$), whereas no such difference was present in the main group.

Analysis of serum hBPI levels in children with upper gastrointestinal tract diseases stratified by sex revealed the following patterns. Development of inflammatory pathology in the gastroduodenal region was associated with a statistically significant increase in serum hBPI in both sexes. Gender-specific features were evident. In particular, the highest hBPI levels were recorded in subgroups with inflammation associated with *Helicobacter pylori* infection. In girls with H. pylori–associated disease, serum hBPI levels increased by 80% compared with controls ($p < 0.05$). In girls without *H. pylori,* the increase was 33% and did not reach statistical significance ($p > 0.05$). A similar pattern was observed in boys; however, the elevation was more pronounced. In boys with *H. pylori*–associated gastroduodenal disease, serum hBPI levels rose by 300% ($p < 0.05$). In boys without *H. pylori*, the increase was 208%.

The relationship between serum hBPI levels and the degree of *H. pylori* bacterial load was evaluated. No significant correlation was found in the overall main group ($r = –0.09$, $p > 0.05$). However, gender-specific associations were identified: a strong positive correlation existed in girls ($r = +0.86$, $p < 0.05$), suggesting that hBPI levels may reflect the extent of *H. pylori* bacterial load in this subgroup. In boys, a significant but inverse correlation was observed ($r = –0.46$, $p < 0.05$), which may indicate gender-dependent differences in the immune response.

Serum hBPI levels were also analyzed in relation to the morphological state of the upper gastrointestinal mucosa. In children with overt gastric or duodenal ulceration, hBPI concentrations remained within the range of the control group (5.64 ± 1.3 ng/mL, $p > 0.05$). In contrast, erosive gastroduodenitis was associated with an almost threefold increase in serum hBPI (15.27 ± 1.2 ng/mL, $p < 0.05$), likely attributable to multiple superficial mucosal defects and consequent release of hBPI.

Serum hBPI concentrations in children with gastroduodenal inflammation were further evaluated according to the degree of inflammatory activity (Table 2). The data demonstrated that hBPI levels varied with the severity of inflammation, and the observed trends were consistent with those identified in the analysis of Table 1.

Table 2

Serum hBPI levels according to degree of inflammatory activity (based on morphological changes in the mucosa) in children with gastroduodenal diseases (M ± m).

Parameter	Main group n=60	Subgroup 1 n=30	Subgroup 2 n=30	Control group, n=20
Lymphoplasmacytic infiltrate (points)	1.5± 0.1	1.8 ± 0.1	1.3 ± 0.1*	-
Activity of inflammation (points)	1.3± 0.1	1.5±0.1	1±0.1*	-
Serum hBPI level (ng/ml)	13.2±1	14.4±1.2	10.9±1.5	5.91±1
Serum hBPI level /lymphoplasmacytic infiltrate ratio (a.u.).	8.8±0.5	8±0.7	8.4±0.8	-
Serum hBPI level /activity of inflammation ratio (a.u.)	10.2±0.5	9.6±0.7	10.9±0.8	-

*Note. * – p<0,05 – compared with the parameters of subgroup 1*

During exacerbation, serum levels of the studied peptide increased, which is consistent with the fact that hBPI is secreted by neutrophils, whose presence in biopsy specimens determines the degree of inflammatory activity. The obtained data demonstrate elevated hBPI levels in association with the development of chronic severe inflammation, as evaluated by the density of lymphoplasmacytic infiltrate (LPI). To ascertain whether this relationship reflects a direct artifact or indirect mechanistic interactions, hBPI concentrations were analyzed according to the severity of chronic inflammation. The results are presented in Figures 1 and 2.

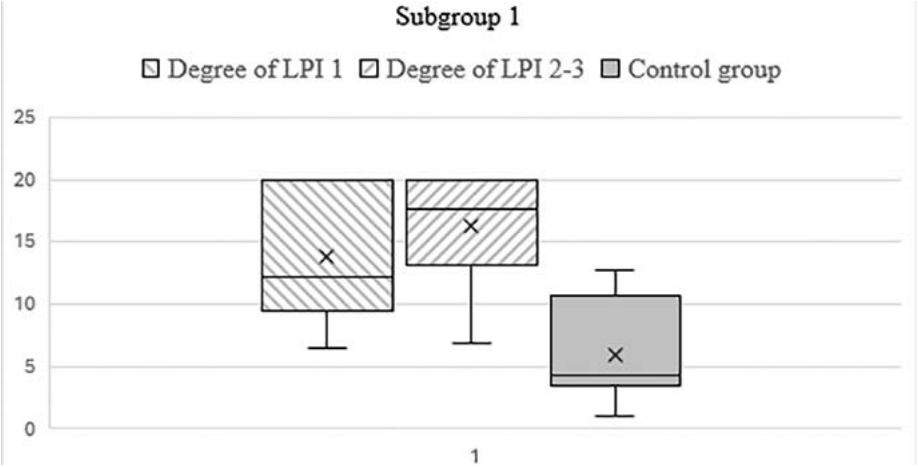

Figure 1. Serum hBPI concentration (ng/mL) in children with *Helicobacter pylori*–associated gastroduodenal diseases, Me (Q25; Q75).

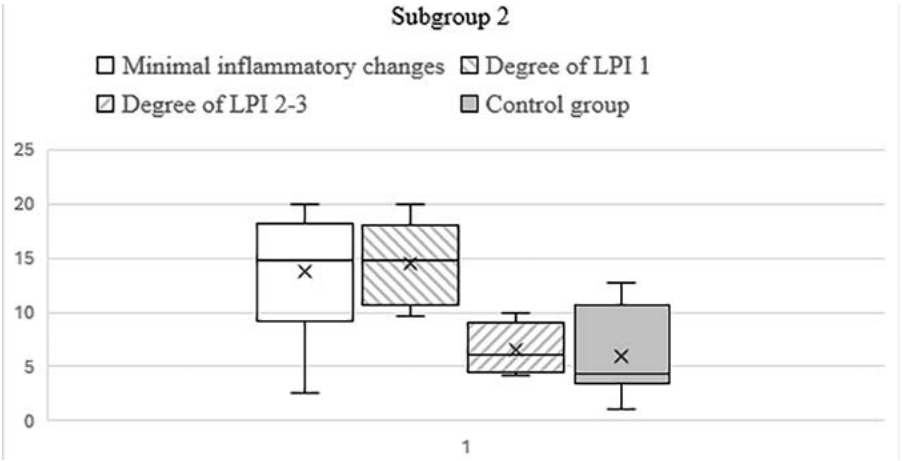

Figure 2. Serum hBPI concentration (ng/mL) in children with gastroduodenal diseases not associated with *H. pylori*, Me (Q25; Q75).

As shown in Figure 1, increasing severity of chronic inflammation (lymphocytic infiltrate) in subgroup 1 was accompanied by a statistically significant rise in serum hBPI levels, reaching 13.8 ± 1.9 ng/mL ($p < 0.05$). With grade 2-3 lymphoplasmacytic infiltrate, the mean hBPI level further increased to 16.2 ± 1.5 ng/mL, which was statistically higher than the group indicators ($p < 0.05$).

Serum hBPI concentrations in children with gastrointestinal diseases not associated with *H. pylori* exhibited an opposite pattern (Figure 2). The highest levels were recorded in the presence of grade 1 lymphoplasmacytic infiltrate (14.5 ± 1.8 ng/mL, $p < 0.05$) or minimal inflammatory changes in biopsy specimens (13.8 ± 1.8 ng/mL, $p < 0.05$). In cases with grade 2-3 lymphoplasmacytic infiltrate, hBPI levels did not differ from those in the control group (6.5 ± 1.2 ng/mL, $p > 0.05$). Thus, significant elevation of hBPI was observed even with minimal inflammatory changes accompanied by only limited immune cell infiltration. These findings suggest that the increase in hBPI levels may result from epithelial cell damage.

The mechanisms by which human bactericidal/permeability-increasing protein (hBPI) interacts with bacterial lipopolysaccharides provide insight into its role in host protective mechanisms [11,18]. hBPI is released from azurophilic granules of neutrophils [7] and from epithelial cells of the gastrointestinal tract [26]. This release is reflected in the observed association between serum hBPI levels and the activity of gastric mucosal inflammation ($p < 0.05$), as assessed by neutrophilic or lymphoplasmacytic infiltrate.

Elevated hBPI levels were detected in the presence of multiple gastric and duodenal erosions but not in cases of solitary ulcers ($p < 0.05$). This finding suggests increased hBPI synthesis in response to extensive epithelial cell damage in the gastroduodenal mucosa. Supporting evidence comes from Vllahu et al. (2024), who demonstrated that inflammation induces activation of epithelial cells, leading to synthesis and release of antimicrobial peptides, including hBPI, which contribute to mucosal protection [19].

Gender differences were evident: adolescent girls in the control group exhibited higher baseline hBPI secretion, whereas in gastroduodenal diseases not associated with *Helicobacter pylori*, the increase in this antimicrobial peptide was modest and did not reach statistical significance ($p > 0.05$). The literature describes modulatory effects of sex hormones on the synthesis and release of antimicrobial peptides [19,20]. Although data specific to hBPI are limited, sex hormones have been shown to influence antimicrobial peptide production by gastrointestinal epithelial cells [27]. Indirect regulatory mechanisms have also been proposed, involving sex hormone-mediated alterations in intestinal microbiota composition and proinflammatory cytokine levels [28].

The observed gender-specific differences in immune response may indicate a predisposition in girls toward more rapid onset and prolonged course of gastroduodenal disease. Overexpression of hBPI has been reported to indirectly sustain chronic inflammation [12].

The present findings align with and extend existing evidence on the involvement of this antimicrobial peptide in immune defense processes [7]. Published data elucidating the pathogenetic mechanisms of hBPI action on bacterial lipopolysaccharides have further clarified its contribution to host protective mechanisms [11,18]. Theprungsirikul et al. (2021) highlighted key aspects of hBPI's antimicrobial activity, including direct bactericidal effects mediated by binding of its positively charged N-terminal domain to negatively charged lipopolysaccharide molecules on Gram-negative bacteria, resulting in cell membrane disruption and enhanced phagocytosis [reference corresponding to cited work]. Kong et al. (2021) demonstrated that hBPI deficiency impairs mucosal protection, promotes inflammation, and increases bacterial load [11].

The release of human bactericidal/permeability-increasing protein (hBPI) from both azurophilic granules of neutrophils [7] and epithelial cells of the upper gastrointestinal tract [26] was clearly reflected in the observed associations between serum hBPI levels and the activity of mucosal inflammation ($p < 0.05$). Elevated hBPI levels were particularly evident in cases of severe chronic inflammation. Although an association between hBPI and the degree of lymphoplasmacytic infiltrate has not been previously described in the literature, the present relationship is most likely attributable to the chronicity of the inflammatory process and persistent epithelial cell damage, which sustains ongoing synthesis and release of hBPI [21].

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Histological examination revealed no *Helicobacter pylori* organisms in gastroduodenal mucosa without signs of chronic inflammation. In the absence of bacteria, low hBPI levels were observed in cases with grade 2-3 lymphoplasmacytic infiltrate. This raises the question of whether the development of chronic inflammation accompanied by epithelial cell damage constitutes a prerequisite for bacterial adhesion and active colonization of the gastric mucosa. Such conditions are characterized by increased expression of Lewis antigens on epithelial surfaces, providing additional binding sites for BabA and SabA adhesins [22], elevated pH, and mucin liquefaction [23], all of which facilitate extensive bacterial colonization [24,25]. Secondary *H. pylori* infection further exacerbates epithelial cell destruction through bacterial virulence factors and indirectly sustains a baseline level of chronic inflammation [24].

Conclusion

1. The course of upper gastrointestinal tract diseases associated with *Helicobacter pylori* in children is accompanied by elevated serum levels of human bactericidal/permeability-increasing protein, which correlate directly with the severity and extent of mucosal involvement.

2. Distinct gender differences in hBPI levels were identified, with girls in the control group exhibiting higher concentrations than boys.

Prospects for further research. Future studies should investigate antimicrobial peptides, particularly hBPI, and their interrelationships with other proteins, as well as changes in their serum levels in children with upper gastrointestinal tract diseases. Such research may deepen understanding of the pathogenesis of *H. pylori*–associated inflammatory gastroduodenal disorders, improve diagnostic approaches, and optimize treatment strategies in pediatric patients.

Authors' contribution: Savchenko D. S. – substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; references, agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved; Lezhenko H. O. – drafting the work or revising it critically for important intellectual content; Final approval of the version to be published;

Conflicts of interest: authors have no conflict of interest to declare.

Use of Artificial Intelligence. No artificial intelligence methods were used in the conduct of the research and in the preparation of the manuscript.

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БІОЛОГІЧНА РОЛЬ HUMAN BPI В ПЕРЕБІГУ ЗАХВОРЮВАНЬ ВЕРХНІХ ВІДДІЛІВ ШЛУНКОВО-КИШКОВОГО ТРАКТУ, АСОЦІЙОВАНИХ ІЗ HELICOBACTER PYLORI У ДІТЕЙ

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Summary.

Поширеність інфекції Helicobacter pylori (*HP*) варіює у різних регіонах світу залишається нерівномірною. Близько однієї третини дітей у світі мають *HP*, до того ж інфікування відбувається здебільшого у дитячому віці. Імунна відповідь при розвитку запальних захворювань здійснюється за рахунок синтезу та вивільнення антимікробних пептидів, які відіграють роль протективних факторів. Серед представників антимікробних пептидів humam BPI (hBPI), механізми протимікробного захисту якого реалізуються різними шляхами. Вивчення зв'язків між рівнем hBPI та морфологічними змінами слизової оболонки верхніх відділів шлунково-кишкового тракту при захворюваннях, асоційованих із *HP* у дітей, дасть змогу покращити розуміння біологічного значення даного антимікробного пептиду в імунній відповіді.

Мета. Дослідити рівень human BPI в сироватці крові дітей із захворюваннями верхніх відділів шлунково-кишкового тракту, асоційованими із *HP* та його роль у розвитку та перебігу інфекційного процесу.

Матеріали і методи. Обстежено 60 пацієнтів, віком від 10 до 17 років. Підгрупу 1 склали 30 дітей із захворюваннями гастродуоденальної зони, асоційованими з *HP.* До підгрупи 2 увійшли 30 дітей із захворюваннями гастродуоденальної зони, неасоційованими з *HP.* До групи контролю увійшли 20 умовно здорових дітей, репрезентативних за віком і статтю. Для діагностики

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було проведено лабораторне, ендоскопічне, гістологічне дослідження. Визначення рівнів hBPI в сироватці крові проводилося за допомогою імуноферментного аналізу з використанням комерційно доступного набору Human BPI ELISA Kit (Elabscience Biotechnology, Х'юстон, Техас, США). Протокол дослідження був затверджений Комітетом з біоетики Запорізького державного медико-фармацевтичного університету. Він був розроблений відповідно до етичних стандартів і принципів, що регулюють медичні дослідження на людях, і не порушував фундаментальних біоетичних норм. При включенні дітей у дослідження було отримано інформовану згоду. (Протокол № 3 від 28.10.2024 р.). Статистична обробка даних проводилася на ПК з використанням ліцензійного програмного пакету Statistica для Windows 13.0, серійний номер JPZ8041382130ARCN10-J, з використанням значень арифметичного середнього (M), середнього квадратичного середнього (σ) та середніх порівнянь (m) для учасників, розділених на категорії, що відповідали критеріям нормальності. Зв'язки між показниками оцінювали за допомогою додаткових методів рангової кореляції Спірмена (r). Для оцінки значущості показників у групах, що були рівними, використовували t-критерій Стьюдента для малих вибірок з використанням непараметричного U-критерію Манна-Уїтні. Значущість вважали істотною при $p<0,05$. Науково-дослідна робота кафедри на тему: «Прогнозування перебігу найбільш поширених запальних захворювань дитячого віку» (№ державної реєстрації 00121U107520, 2020-2026 рр.)

Результати. Встановлено, що рівень hBPI в сироватці крові дітей основної групи перевищував показники дітей групи контролю в 2,2 рази ($p<0,05$). Спостерігалось підвищення рівню hBPI при ерозивному гастродуоденіті у 3 рази порівняно з показниками контрольної групи ($p<0,05$). Виявлено гендерні особливості, а саме в підгрупі дівчат за наявності *HP* зростання рівню hBPI відбувалось на 80% ($p<0,05$), у підгрупі хлопчиків зростання hBPI у сироватці крові відбувалось більш виражено – на 300% ($p<0,05$). При наявності інфекції *HP* підвищення активності запалення відбувалось на фоні зростання hBPI ($p<0,05$). При відсутності *HP* найвищий вміст hBPI в сироватці крові реєструвався при лімфоплазматичному інфільтраті 1 ступеня ($p<0,05$) і при мінімальних запальних змінах ($p<0,05$).

Висновки. За результатами проведеного дослідження встановлено, що перебіг захворювань верхніх відділів шлунково-кишкового тракту, асоційованих із *HP,* у дітей відбувається на тлі зростання вмісту в сироватці крові hBPI, рівень якого прямо залежить від ступеню та площі уражень слизової оболонки. Відмічено гендерні відмінності у забезпеченні організму відповідним рівнем hBPI. Показано, що у дівчат контрольної групи рівень hBPI був вищим, порівняно з хлопчиками.

Ключові слова: Helicobacter pylori; гастродуоденальна зона; діти; підлітки; діагностика; ендоскопія; human Bactericidal/ Permeability-Increasing protein.

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