ORIGINAL ARTICLE ÖZGÜN ARAŞTIRMA

Relationship between Helicobacter Pylori Gastritis and Plasma Amino Acids

Helicobacter Pylori Gastriti ile Plazma Amino Asitleri İlişkisi

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Abstract

Introduction: This study investigated the relationship between plasma amino acid levels and the presence of *Helicobacter pylori* (*H. pylori*) in children.

Materials and Methods: The study included 88 pediatric patients with gastritis (30 *H. pylori*-positive and 58 *H. pylori*-negative) and a control group of 32 children. Plasma amino acid levels were assessed using liquid chromatography-tandem mass spectrometry, and dietary amino acid intake for all participants was estimated based on a three-day dietary questionnaire.

Results: Plasma glycine, alanine and histidine levels were significantly high in the *H. pylori*-negative group compared to the other groups ($p<0.05$). Plasma cysteine, homocysteine and hydroxylysine levels were significantly higher in the control group compared to both gastritis groups ($p<0.05$). Plasma asparagine, glutamine and histidine levels were found significantly lower in the *H. pylori* positive group than in the *H. pylori* negative group ($p<0.05$).

Conclusion: This study identified a moderate correlation between plasma levels of asparagine, glutamine, and histidine and the presence of *H. pylori* gastritis in children.

Keywords
Helicobacter pylori, plasma amino acids, gastritis, pediatric

Öz

Giriş: Bu çalışmada plazma amino asit düzeyleri ile çocuklarda *Helicobacter pylori* (*H. pylori*) varlığı arasındaki ilişki araştırıldı.

Gereç ve Yöntem: Çalışmaya gastriti olan 88 çocuk hasta (30 *H. pylori* pozitif ve 58 *H. pylori* negatif) ve 32 çocuktan oluşan bir kontrol grubu dahil edildi. Plazma amino asit düzeyleri sıvı kromatografisi-tandem kütle spektrometrisi kullanılarak değerlendirildi ve tüm katılımcıların diyetle amino asit alımı üç günlük diyet anketine dayanarak tahmin edildi.

Bulgular: Plazma glisin, alanin ve histidin düzeyleri *H. pylori* negatif grupta diğer gruplara kıyasla anlamlı derecede yüksekti ($p<0,05$). Plazma sistein, homosistein ve hidroksilizin düzeyleri kontrol grubunda her iki gastrit grubuna kıyasla anlamlı derecede yüksekti ($p<0,05$). Plazma asparagin, glutamin ve histidin düzeyleri *H. pylori* pozitif grupta *H. pylori* negatif gruba kıyasla anlamlı derecede düşük bulundu ($p<0,05$).

Sonuç: Bu çalışmada, asparagin, glutamin ve histidin plazma düzeyleri ile çocuklarda *H. pylori* gastriti varlığı arasında orta düzeyde bir korelasyon tespit edildi.

Anahtar kelimeler
Helicobacter pylori, plazma amino asitleri, gastrit, çocuk

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Introduction

Helicobacter pylori (*H. pylori*) is a prevalent bacterial infection, commonly acquired in childhood, with reported prevalence rates of 47.2% and 75.8% among children in western and eastern Türkiye, respectively (1,2). Early acquisition of *H. pylori* is a substantial risk factor for complications later in life, including acute and chronic gastritis, peptic ulcers, and gastric cancer (3,4). However, not all individuals infected with *H. pylori* develop gastritis symptoms, and certain aspects of its pathogenesis, particularly during childhood, remain unclear.

Amino acids are essential in sustaining physiological functions and supporting healthy growth. Elevated amino acid levels and their derivatives may contribute to conditions like neurological diseases, oxidative stress, and cardiovascular disorders, while dietary amino acid supplementation has shown therapeutic potential for obesity, metabolic syndrome, and cardiovascular diseases (5). Limited studies indicate that melatonin and its precursor, L-tryptophan, can protect the gastric mucosa and enhance healing of *H. pylori*-associated gastroduodenal ulcers (6). Additionally, histidine, glutamine, glycine, and arginine have been identified as strong chemoattractants for *H. pylori* (7). This cross-sectional study aims to investigate the effect of plasma amino acids on *H. pylori* colonization in gastric mucosa and the changes that occur after *H. pylori* treatment.

Materials and Methods

Study Population

The study enrolled patients aged 6-18 years who presented with dyspeptic complaints at the Department of Pediatric Gastroenterology at Erciyes University between January and October 2019. Prior to endoscopy, 2 mL of venous blood was collected for plasma amino acid analysis. Upper gastrointestinal endoscopy was conducted using a Fujinon 4400-HD-EG530FP system, with biopsies taken from the antrum and corpus for histopathological assessment (8). Patients were classified as *H. pylori*-positive or -negative based on biopsy results. *H. pylori*-positive patients received a two-week eradication therapy regimen comprising amoxicillin, clarithromycin, and lansoprazole. Following four weeks of therapy, it was confirmed to be eradicated with negative stool antigen test for *H. pylori*, and blood samples were drawn again for amino acid analysis. The control group included children aged 6-18 years without dyspeptic symptoms and also negative for *H. pylori* antigen in stool. Exclusion criteria included patients with previously treated gastritis.

The study received approval from the Erciyes University Ethics Committee (date: 09.01.2019, approval number: 2019/25) and was funded by the Erciyes University Scientific Research Project Unit (Project No.: TSA-2019-8825). Informed consent was obtained from the parents of the patients.

Amino Acid Analysis

For plasma amino acid analysis, 2 mL of venous blood was collected from each patient into an EDTA tube following at least 8 hours of fasting. Samples were centrifuged at 5000 rpm for 5 minutes to separate the plasma, which was then stored at -20 °C until analysis. Upon completion of sample collection, all plasma samples were analyzed collectively. From each sample, 100 µL of plasma was prepared by removing nitrogen with a blow tube, and a 10 µL aliquot was processed for amino acid quantification using liquid chromatography-tandem mass spectrometry.

Dietary Analysis

Participants recorded their dietary intake over three days, detailing each meal and portion size. These records were evaluated by a dietitian using the BeBIS 7.2 nutrition information software, enabling calculation of both essential and non-essential amino acid intake. Growth parameters, including z-scores for body weight and height, were also documented for each patient.

Statistical Analysis

Statistical analyses were conducted using IBM SPSS for Windows (version 26.0; IBM Corp., Armonk, NY) and R Statistical Software (version 4.3.0; R Core Team, 2023). Descriptive statistics for continuous variables were reported as means with standard deviations or as medians with interquartile ranges. Categorical variables were summarized as frequencies and percentages. Normality was assessed visually via QQ-plots and histograms, as well as with the Shapiro-Wilk test. Independent group comparisons were made using either the One-Way ANOVA or Kruskal-Wallis test, depending on normality assumptions. For significant group differences, post-hoc tests were performed to identify specific group contrasts. Time-based changes were analyzed using paired t-tests or Wilcoxon signed-rank tests, as appropriate. Chi-Square tests (including Pearson, Yates, and Fisher's exact tests) were applied to compare categorical data. Receiver Operating Characteristic (ROC) analysis was used to evaluate the discriminatory power of variables, with Area Under the Curve (AUC) values and confidence intervals reported. Statistical significance was set at $p<0.05$.

Results

The study included 88 pediatric patients diagnosed with gastritis, of whom 30 (34.1%) were *H. pylori*-positive and 58 (65.9%) were *H. pylori*-negative, as well as a control group of 32 children. The mean age of the patients was 16 ± 2.1 years.

Comparison of plasma amino acid levels of patients and control group is shown in Table 1. Glycine, alanine and histidine levels were significantly high in the *H. pylori*-negative group compared to the other groups ($p<0.05$). Significant differences were observed in plasma asparagine, aspartic acid and glutamine levels between the groups ($p<0.05$). Levels of cystine, homocysteine and hydroxylysine were significantly higher in the control group than in the gastritis groups ($p<0.05$).

Table 2 describes the changes in plasma amino acid levels among *H. pylori*-positive patients before and after *H. pylori* eradication treatment. Significant reductions

Table 1. Comparison of the patients and the control group in terms of plasma amino acid levels

Amino acids	Normal levels (min - max) (nmol/mL)	*H. pylori*-positive (n=30)	*H. pylori*-negative (n=58)	Control (n=32)	p-value
Alanine	157-481	505 (398-573)[a]	549 (476-687)[b]	456 (366-582.)[a]	**0.005**
Arginine	38-122	76.67 ± 36.01[ab]	80.98 ± 34.27[a]	59.03 ± 21.81[b]	**0.008**
Argininosuccinic acid	0-1	4.7 ± 2.12[ab]	4.91 ± 2.47[a]	3.34 ± 3.04 [b]	**0.019**
Asparagine	23-70	62 (52-70)[a]	73 (62-83)[b]	49.5 (40.5-61) [c]	**<0.001**
Aspartic acid	1-8	36.5 (12-155)[a]	15 (10-67)[b]	8 (2-17) [c]	**<0.001**
Cystine	33-57	17 (14-28)[a]	15.5 (6-23)[a]	54 (48.5-61) [b]	**<0.001**
Citrulline	9-52	42.83 ± 2.10	43.88 ± 3.23	38.81± 11.26	0.179
Glutamine	405-923	446 (361-566)[a]	672 (520-793)[b]	949 (882.5-1096) [c]	<0.001
Glutamic acid	9-109	45.5 (39-55)	37.5 (24-56)	48.5 (28.5-65)	0.081
Glycine	138-349	282 (212-317)[ab]	305 (262-362)[b]	245 (206.5-314.5) [a]	**0.004**
Homocysteine	0-1	0.001 (0-0.001)[a]	0.001 (0-0.002)[a]	2 (1-4.5) [b]	**<0.001**
Hydroxylysine	0-1	2 (2-3)[a]	3 (2-4)[a]	5 (3-6) [b]	**0.001**
Hydroxyproline	6-32	21.5 (14-83)	19.5 (14-27)	17.5 (15-25.5)	0.163
Isoleucine	33-97	108.5 (90-124)[a]	99.5 (91-118)[a]	59.5 (52-65)[b]	**<0.001**
Leucine	65-179	186.4 ± 48.56[a]	173.41 ± 32.24[a]	129.19 ± 22.50[b]	**<0.001**
Lysine	98-231	201.53 ± 47.55[a]	224.59 ± 48.94[a]	147.0 ± 29.46[b]	**<0.001**
Methionine	14-37	33.5 (25-46)[a]	36 (30-43)[a]	23.5 (18-27.5) [b]	**<0.001**
Ornithine	33-103	70.5 (64-109)[a]	90 (70-115)[a]	47.5 (36.5-63.5) [b]	**<0.001**
Phenylalanine	38-86	83.5 (73-98)[a]	79.5 (71-99)[a]	59 (51.5-70) [b]	**<0.001**
Proline	99-351	290.5 (216-395)[a]	280.5 (217-396)[a]	171 (135.5-252) [b]	**<0.001**
Serine	85-185	178 (154-197)[ab]	182 (158-202)[a]	159.5 (120.5-184.5)[b]	**0.034**
Threonine	59-195	211.5 (177-254)[a]	241.5 (201-293)[a]	138 (116.5-153) [b]	**<0.001**
Tryptophan	30-94	72.53 ± 16.52[a]	69.98 ± 18.64[a]	47.78 ± 6.62[b]	**<0.001**
Tyrosine	31-108	69.5 (59-86)[ab]	74.5 (65-90)[a]	65 (56-74)[b]	**0.016**
Valine	130-307	276.5 (223-304)[a]	281.5 (234-334)[a]	198.5 (182-228.5) [b]	**<0.001**
Histidine	54-113	95 (85-108)[a]	110.5 (100-135)[b]	89 (81-104)[a]	**<0.001**

Variables are summarized with mean ± standard deviation or median (Q1- Q3). Post-hoc test results were shown using letters (a, b, c). Different letters (a-b, a-c, b-c) represented that there was a statistically significant difference between groups

were observed after treatment for levels of alanine, arginine, asparagine, aspartic acid, citrulline, glutamic acid, hydroxyproline, isoleucine, leucine, phenylalanine, tryptophan, tyrosine and valine ($p<0.05$).

In contrast, levels of homocysteine and glutamine were found to increase following treatment ($p<0.05$).

Table 3 compares the dietary content among the groups. No significant differences were observed between the *H. pylori*-positive and *H. pylori*-negative groups for these dietary components.

The area under the ROC curve (AUC) was used to evaluate the discriminatory ability of amino acids in identifying *H. pylori* positive patients (Table 4). Among the amino acids analyzed, asparagine, glutamine, and histidine showed moderate AUC levels for distinguishing between *H. pylori*-positive and *H. pylori*-negative patients. Performance was further evaluated by combining pairs and trios of these amino acids. When asparagine, glutamine, and histidine were used together, the AUC (95% CI) reached 0.80 (0.71 – 0.91). In contrast, using only glutamine and histidine without asparagine yielded an AUC (95% CI) of 0.79 (0.69 – 0.90). ROC analysis of dietary components indicated non-significant discriminatory power (AUC < 0.70).

Table 2. Comparison of *H. pylori* positive patients and the same patients after eradication treatment of plasma amino acid levels

Amino acids	Normal levels (min - max) (nmol/mL)	Before	After	p-value
Alanine	157-481	505.0 (394.0 – 574.0)	428.5 (356.0 – 516.25)	**0.041**
Arginine	38-122	76.67 ± 36.01	55.27 ± 27.08	**0.007**
Argininosuccinic acid	0-1	4.7 ± 2.12	4.83 ± 2.18	0.769
Asparagine	23-70	62.0 (50.75 – 70.25)	68.0 (59.25-77.0)	**0.024**
Aspartic acid	1-8	36.5 (12.0 – 157.5)	12.5 (4.0-44.5)	**0.003**
Cystine	33-57	17 (14.0-29.0)	13.5 (4.75-25.25)	0.266
Citrulline	9-52	42.83 ± 12.10	36.47 ± 12.23	**<0.001**
Glutamine	405-923	446 (352.25-572.25)	741.5(707.75-827.5)	**<0.001**
Glutamic acid	9-109	45.5 (38.75-56.25)	12.0 (8.5-63.25)	**0.003**
Glycine	138-349	282 (211.25-318.25)	249.5 (227.0-317.75)	0.371
Homocysteine	0-1	0.001 (0.0-0.001)	1.0 (0.0-1.25)	**0.001**
Hydroxylysine	0-1	2.0 (2.0-3.25)	3.5 (1.0-5.0)	0.091
Hydroxyproline	6-32	21.5 (14.0-86.5)	13.5 (11.0-23.5)	**0.009**
Isoleucine	33-97	107.9 ± 21.44	92.47 ± 14.75	**0.003**
Leucine	65-179	174.0 (147.0-218.5)	139.0 (126.0-158.0)	**0.004**
Lysine	98-231	190.5 (162.25-238.25)	166.5 (151.0-201.5)	0.150
Methionine	14-37	33.5 (25.0-46.75)	31.0 (27.0-35.25)	0.096
Ornithine	33-103	70.5 (63.5-116.0)	93.5 (71.75-114.0)	0.258
Phenylalanine	38-86	85.4 ± 19.40	69.87 ± 14.09	**0.001**
Proline	99-351	290.0 (210.5-396.75)	261.0 (215.25-341.25)	0.250
Serine	85-185	178.0 (154.0-197.0)	151.5 (127.25-180.75)	0.165
Threonine	59-195	213.77 ± 58.34	213.17 ± 57.75	0.969
Tryptophan	30-94	72.53 ± 16.52	55.4 ± 11.36	**<0.001**
Tyrosine	31-108	76.77 ± 27.79	59.63 ± 13.98	**0.004**
Valine	130-307	271.57 ± 65.62	232.33 ± 50.84	**0.015**
Histidine	54-113	98.5 ± 20.72	94.57 ± 20.53	0.471

Variables are summarized with mean ± standard deviation or median (Q1- Q3)

Table 3. Comparison of the dietary ingredients of the groups				
	***H. pylori*-positive (n=30)**	***H. pylori*-negative (n=58)**	**Control (n=32)**	**p-value**
Energy (kcal/day)	2657.1 (1915.8-3938.5)[a]	2434.2 (1805.6-3864.0)[a]	1656.2 (1427.5-2124.7)[b]	**<0.001**
Protein (g)	95.6 (66-118.4)[a]	92.6 (64-117.5)[a]	68.5 (39-76)[b]	**<0.001**
Isoleucine (mg)	4771.9 (3124.5-5465.7)[a]	4633.5 (2919.5-5378.8)[a]	3170.1 (2646.4-3802.3)[b]	**<0.001**
Leucine (mg)	7572 (5033.7-8760.9)[a]	7398 (4923.6-8597.7)[a]	5269.3 (4286.2-6081.4)[b]	**<0.001**
Lysine (mg)	5487.9 (3441.3-6302)[a]	5550.5 (3251.3-6312)[a]	3693.6 (2994.8-4418.8)[b]	**<0.001**
Methionine (mg)	1917.9 (1365.9-2209.7)[a]	1843.4 (1465.9-2312.7)[a]	1258.9 (1091.3-1495.5)[b]	**<0.001**
Cystine (mg)	1318.7 (971.3-1854.1)[a]	1226.2 (969.3-1506.2)[a]	945.2 (783.3-1191.8)[b]	**<0.001**
Phenylalanine (mg)	4297.3 (3080.3-5218.1)[a]	4125.7 (3078.3-5072.6)[a]	3140.6 (2546.8-3749.5)[b]	**0.003**
Tyrosine (mg)	3578.7 (2409.8-4162.8)[a]	3576.4 (2403.8-3888.4)[a]	2512 (1955.8-2847.6)[b]	**0.001**
Threonine (mg)	3829.1 (2791.8-4245.1)[a]	3729.1 (2691.4-4181.6)[a]	2492.2 (2078.9-3038.8)[b]	**<0.001**
Tryptophan (mg)	1122.5 (769.6-1302.3)[a]	1119.5 (767.3-1270.3)[a]	788 (647.2-912.6)[b]	**<0.001**
Valine (mg)	5516.2 (3807.3-6164.7)[a]	5428.9 (3797.3-6109.2)[a]	3588.9 (2975.2-4245.7)[b]	**<0.001**
Histidine (mg)	2352.5 (1439.6-2664.8)[a]	2042.5 (1429.2-2464.6)[a]	1615.3 (1364.6-1832.9)[b]	**0.002**
Essential aa (g)	46.1 (30.9-52)[a]	44.8 (29.9-50.8)[a]	32.7 (27.1-37.6)[b]	0.001
Aspartic acid (mg)	7726.6 (5507.6-8845.4)[a]	7599.9 (5497.6-8701.2)[a]	5552.9 (4543.5-7032.9)[b]	**0.002**
Glutamic acid (mg)	18686 (13393-27278)[a]	17929 (13373-24792)[a]	14568.5 (11748-17065.5)[b]	**0.008**
Glycine (mg)	3348.1 (2330.4-4240.8)[a]	3138.1 (2329.4-4071.1)[a]	2565.7 (1916.3-3084.8)[b]	**0.016**
Proline (mg)	6578.7 (4389.4-9405.9)[a]	6321.6 (4372.4-8995)[a]	5089.7 (3996.9-5870.7)[b]	**0.002**
Non-essential aa (g)	43.4 (30.6-60.3)[a]	45.4 (30.8-55.2)[a]	33.7 (27.9-39)[b]	**0.005**
Plant proteins (g)	41.2 (29.9-72.1)	39.9 (28.9-63.3)	34.2 (22.3-50.7)	0.242
Uric acid (mg)	460.2 (356.5-598.4)[a]	456.3 (366.3-579.9)[a]	376.5 (287.2-451.8)[b]	**0.014**
Arginine (mg)	4492.8 (3420.9-5709.4)	4362.9 (3419.9-5655.1)	3237.3 (2319.9-5475.6)	**0.091**
Alanine (mg)	3939.6 (3121.1-4815.1)[a]	3729.6 (3120.1-4521.4)[a]	2674.8 (2251.3-3172.5)[b]	**<0.001**
Purine (mg)	151.8 (118.7-200.4)[a]	153.5 (118.7-192.5)[a]	125.5 (96.5-151.2)[b]	**0.016**
Serine (mg)	4836.3 (3325.2-5842.9)[a]	4679.9 (3315.2-5737.8)[a]	3354.6 (2737.1-4010.3)[b]	**0.001**

Variables are summarized with median (Q1- Q3). Post-hoc test results were shown using letters (a, b). Different letters (a-b) represented that there was a statistically significant difference between groups

Table 4. Results of ROC curve analysis for Asparagine, Glutamine and Histidine			
Amino acids	**AUC**	**95 % CI**	**p-value**
Asparagine	0.73	0.63 – 0.84	**<0.001**
Glutamine	0.76	0.66 – 0.86	**<0.001**
Histidine	0.72	0.61 – 0.83	**0.001**
Asparagine and glutamine	0.79	0.69 – 0.88	**<0.001**
Asparagine and histidine	0.77	0.67 – 0.87	**<0.001**
Glutamine and histidine	0.79	0.69 – 0.90	**<0.001**
Asparagine, glutamine and histidine	0.81	0.71 – 0.91	**<0.001**

AUC: Area under the ROC curve, CI: Confidence interval

Discussion

Amino acids play a fundamental role in maintaining body functions and exist in various compositions across living organisms. They are generally classified as essential or non-essential. Essential amino acids, such as arginine, isoleucine, histidine, leucine, lysine, methionine, phenylalanine, tryptophan, threonine, and valine, are vital not only for protein synthesis but also as precursors for non-essential amino acids. Essential amino acids must be obtained through diet, as they are crucial for bodily processes and non-essential amino acid synthesis (9).

H. pylori survives within the gastric epithelial cells of its host by leveraging urease activity and outer membrane proteins. It utilizes urea in amino acid synthesis and can metabolize L-arginine and L-ornithine, facilitating their breakdown into urea (10). The simplest amino acid, glycine, is known to exhibit antibacterial properties by inhibiting cell wall synthesis. In a study by Minami et al. (11), glycine was shown to work effectively with other antimicrobial agents in treating clarithromycin-resistant *H. pylori* infections. Additionally, glycine inhibits NF-kB and reduces the expression of proinflammatory cytokines, exhibiting anti-inflammatory effects (12). In this study, plasma levels of glycine, alanine, and histidine were notably higher in *H. pylori*-negative gastritis patients compared to other groups, suggesting a possible differential metabolic profile in these cases. Abdollahi et al. (13) reported that phenylalanine, aspartic acid, glutamic acid, leucine, and isoleucine act as positive chemotactic agents for *H. pylori*, while tyrosine has a negative effect. In the current study, plasma aspartic acid levels were elevated in *H. pylori*-positive gastritis cases compared to the control group, highlighting a potential link between amino acid levels and bacterial colonization or activity.

Glutathione, a potent antioxidant, is synthesized from cysteine and plays a crucial role in inhibiting inflammation by promoting leukotriene synthesis, a key factor in the body's immune response (14). In this study, plasma cystine levels in gastritis patients were lower than those in the control group. While dietary cystine intake was higher in patients than in the control group, low plasma cystine may act as a contributing factor in gastritis development. Hellström et al. (15) noted that slow-release cysteine formulations can neutralize carcinogenic acetaldehyde in the stomachs of individuals with *H. pylori* infection or chronic atrophic gastritis. Additionally, Di Mario et al. (16) reported that in chronic atrophic gastritis patients, cysteine supplementation improved gastric health by increasing pepsinogen levels and reducing gastrin levels.

H. pylori requires arginine, leucine, isoleucine, histidine, methionine, phenylalanine, and valine for growth, which may influence amino acid levels in infected individuals (17). In addition to elevated cystine and hydroxylysine levels, control group participants also had higher homocysteine levels. Although plasma homocysteine levels exceeded the reference range, they remained below the hyperhomocysteinemia threshold of 15 micromol/L (or 15 nmol/mL), rendering them statistically insignificant (18). However, a notable increase in aspartic acid levels was observed in the presence of *H. pylori*. *H. pylori* employs asparaginase and glutaminase activity for ammonia production, leading to substantial depletion of aspartate and glutamate (19). Leduc et al. (20) demonstrated that L-asparaginase and γ-glutamyltranspeptidase serve as key periplasmic deamidases in *H. pylori*. In this study, elevated aspartic acid levels in the *H. pylori*-positive group may support *H. pylori* activity. After eradication therapy, a marked decrease in plasma aspartic acid levels was observed, while glutamine levels increased in the same group.

Among the amino acids, asparagine, glutamine, and histidine displayed moderate AUC values in differentiating *H. pylori*-positive from *H. pylori*-negative children. The combined analysis of these three amino acids proved more valuable than individual or paired assessments. Asparagine and glutamine, non-essential amino acids, possess immunological and growth modulation effects (21). Histidine, an essential amino acid, has notable antioxidant properties (22). The *H. pylori*-positive group exhibited significantly lower levels of these three amino acids compared to the *H. pylori*-negative group.

Certain foods, including bovine milk, broccoli sprouts, cranberry, highbush blueberry juice, and plant oils, are known to have protective effects against *H. pylori* infection (23,24). Xia et al. (25) reported that diets high in carbohydrates and sweets were associated with *H. pylori* infection, while diets rich in animal offal, fish, poultry, and seafood showed a negative association. In this study, dietary protein intake in the *H. pylo*ri-positive group was higher than in the control group. Despite dietary differences between gastritis patients and the control group, no significant dietary variations were noted between the *H. pylo*ri-positive and *H. pylo*ri-negative groups.

Study Limitations

This study has some limitations. First, there is limited comparable data in the existing literature. Additionally, since plasma amino acid levels are closely influenced by diet, interpreting our plasma amino acid measurements in patients proved challenging. Nevertheless, we observed

changes in plasma levels of certain amino acids that align with findings from patient groups. On the other hand, since gastric juice amino acid levels of the patients could not be studied, comparison of plasma amino acid levels and gastric juice amino acid levels was not possible.

Conclusion

In conclusion, this study is the first to explore the relationship between plasma amino acids and *H. pylori*. Plasma levels of asparagine, glutamine, and histidine displayed moderate discriminatory potential for the presence of *H. pylori*. Further clinical studies are needed to investigate the amino acids that may facilitate *H. pylori* colonization.

Ethics

Ethics Committee Approval: The study received approval from the Erciyes University Ethics Committee (date: 09.01.2019, approval number: 2019/25) and was funded by the Erciyes University Scientific Research Project Unit (Project No.: TSA-2019-8825).

Footnotes

Conflict of Interest: No conflict of interest was declared by the authors.

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