ISTANBUL UNIVERSITY PRESS

RESEARCH / ARAŞTIRMA
DOI: 10.26650/IUITFD.1003753
İst Tıp Fak Derg 2022 / J Ist Faculty Med 2022

EVALUATION OF HELICOBACTER PYLORI FREQUENCY AND GASTRIC ANTRUM PATHOLOGY FINDINGS IN PATIENTS WITH PORTAL HYPERTENSION

PORTAL HİPERTANSİYONU OLAN HASTALARDA HELİCOBACTER PYLORİ SIKLIĞININ VE MİDE ANTRUM PATOLOJİ BULGULARININ DEĞERLENDİRİLMESİ

Bilger ÇAVUŞ[1], Aslı ÇİFÇİBAŞI ÖRMECİ[1], Sami EVİRGEN[1], Raim İLİAZ[1], Alp ATASOY[1], Gülçin YEĞEN[2], Kadir DEMİR[1], Selman Fatih BEŞIŞIK[1], Sabahattin KAYMAKOĞLU[1], Filiz AKYÜZ[1]

[1]Istanbul University, İstanbul Faculty of Medicine, Department of Gastroenterohepatology, Istanbul, Turkiye
[2]Istanbul University, İstanbul Faculty of Medicine, Department of Pathology, Istanbul, Turkiye

ORCID IDs of the authors: B.Ç. 0000-0003-2203-4255; A.Ç.Ö. 0000-0001-6297-8045; S.E. 0000-0001-6920-777X; R.İ. 0000-0002-3635-1967; A.A. 0000-0003-1791-897X; G.Y. 0000-0003-2497-219X; K.D. 0000-0002-5226-3705; S.F.B. 0000-0001-5184-376X; S.K. 0000-0003-4910-249X; F.A. 0000-0001-7498-141X

Cite this article as: Cavus B, Cifcibasi Ormeci A, Evirgen S, İliaz R, Atasoy A, Yegen G, et al. Evaluation of helicobacter pylori frequency and gastric antrum pathology findings in patients with portal hypertension. J Ist Faculty Med 2022;85(2):211-5. doi: 10.26650/IUITFD.1003753

ABSTRACT

Objective: In our study, we aimed to evaluate the prevalence of Helicobacter pylori (H. Pylori) and the characteristics of antrum pathologies in patients with portal hypertensive gastropathy.

Materials and Methods: Antrum pathologies and the presence of H. Pylori evaluated with Giemsa stain in gastroscopies of 203 patients with portal hypertensive gastropathy.

Results: A total of 203 patients, 119 male, 84 female, with portal hypertensive gastropathy was included in our study. In patients with portal hypertension, the rate of H. Pylori positivity was 15.3%. This rate was determined as 18.5% under 65 years old and 8.8% over 65 years old. On the basis of gender, H. Pylori positivity rates were 21.8% in male patients and 6% in female patients. The most common antrum pathologies in the patients, in order of frequency, were reactive gastropathy with a rate of 41.4%, active gastritis with a rate of 23.6%, and edema with a rate of 22.2%. The rate of intestinal metaplasia was found to be 9.9%.

Conclusion: In the patients with portal hypertension included in our study, the H. Pylori positivity rate was found to be 15.3%. This rate was found to be below the H. Pylori positivity rate in our country. The most common antrum pathology in the patients was reactive gastropathy with a rate of 41.4%, while the rate of intestinal metaplasia was found to be 9.9%.

Keywords: Portal hypertensive gastropathy, H. pylori, gastroscopy, prevelance

ÖZET

Amaç: Çalışmamızda portal hipertansif gastropatisi (PHG) olan hastalarda Helicobacter pylori sıklığını ve antrum patolojilerinin özelliklerini değerlendirmeyi amaçladık.

Gereç ve Yöntem: Portal hipertansiyonu olan 203 hastanın endoskopi ünitemizde yapılmış olan gastroskopilerinde portal hipertansif gastropati saptanan hastaların antrum patolojileri ve Giemsa boyası ile değerlendirilen H. Pylori varlığı retrospektif olarak araştırılmıştır.

Bulgular: Çalışmaya, portal hipertansif gastropatisi olan, 119'u erkek, 84'ü kadın, toplam 203 hasta dahil edilmiştir. Portal hipertansif gastropatisi olan hastalarda, H. Pylori pozitiflik oranı %15,3 olarak saptandı. Bu oran 65 yaş altında %18,5, 65 yaş üzerinde ise %8,8 olarak saptanmıştır. Cinsiyet bazında, H. Pylori pozitiflik oranları erkek hastalarda %21,8, kadın hastalarda %6 olarak tespit edildi. Hastalarda en sık görülen antrum patolojileri sıklık sırasına göre: %41,4 oranı ile reaktif gastropati, %23,6 aktif gastrit ve %22,2'lik oran ile ödem olarak saptandı. İntestinal metaplazi oranı ise %9,9 olarak saptandı.

Sonuç: Çalışmamıza dahil ettiğimiz PHG'i olan hastalarda, H. Pylori pozitiflik oranı %15,3 olarak saptandı. Bu oranın ülkemiz genelindeki H. Pylori pozitiflik oranının altında olduğu görülmüştür. Hastalarda en sık görülen antrum patolojisi %41,4 oranı ile reaktif gastropati iken, intestinal metaplazi oranı %9,9 olarak saptandı.

Anahtar Kelimeler: Portal Hipertansif Gastropati (PHG), H. Pylori, gastroskopi, prevelans

Corresponding author/İletişim kurulacak yazar: dr_bilgercavus@yahoo.com

Submitted/Başvuru: 10.12.2021 • **Accepted/Kabul:** 01.02.2022 • **Published Online/Online Yayın:** 08.03.2022

INTRODUCTION

Portal hypertension (PHT), defined as an increase in blood pressure in the portal system, causes varicose veins in the gastrointestinal system as well as portal hypertensive gastropathy (PHG), enteropathy, and colopathy, which is characterized by characteristic mucosal pictures, primarily in the stomach (1, 2).

PHG is identified in patients with cirrhotic PHT and/or non-cirrhotic PHT by the presence of endoscopic findings in the stomach mucosa that appear as a mosaic pattern of erythematous, tiny, polygonal regions bordered by a reticular whitish border. Although cirrhosis is the most common cause of PHT, PHG can also arise in non-cirrhotic PHT instances. The pathophysiology of PHG is not well understood (3, 4). It is thought that the hemodynamic changes that develop on the basis of PHT are effective in the development of PHG by causing hyperdynamic congestion, which also leads to changes in gastric mucosal blood flow. Furthermore, alterations in this gastric mucosal microcirculation cause the release of several cytokines, hormones, and growth factors, resulting in a decrease in mucin secretion, making the stomach mucosa more sensitive and vulnerable to harmful elements (5, 6).

H. pylori, which is one of the most damaging factors in the gastric mucosa, is also accepted as an oncogenic bacterium. It has been proposed that bacterial virulence, host, and environmental factors all have a role in making H. pylori illness a complex process (7, 8). It advances and colonizes from the epithelial layer of the stomach mucosa to the basal layer by being influenced by numerous bacterial virulence factors such as flagella, as well as host gastric mucosal variables such as acidity and mucosal structure (8, 9). While PHG causes some hemodynamic and hormonal changes that may make the gastric mucosa vulnerable, there are conflicting results in the literature regarding how the relationship between this gastropathic stomach and H. pylori progresses and how the frequency of H. pylori changes in this patient group (3, 9). In this study, we aimed to investigate the frequency of H. pylori in patients with PHG in our patient group.

MATERIALS AND METHODS

A total of 203 patients with PHG were included in the study. The presence of H. pylori was explored retrospectively in patients who were reported to have esophageal varices and/or fundus varices in gastroscopic examinations performed in the endoscopy unit of Istanbul University, Istanbul Faculty of Medicine during 2014-2015 and whose antrum was biopsied. Patients whose biopsy specimens were unsuitable for histological analysis and/or H. pylori was not detected in the biopsy preparations, as well as those on proton pump inhibitors, were excluded from the study. The diagnosis of PHG was made based on the presence of typical endoscopic findings in patients with cirrhosis or non-cirrhotic PHT. Endoscopically, the typical findings of PHG were classified based on the presence of four main lesions which are mosaic-like pattern, presence of red spot lesions, presence of cherry-red spots, and presence of black-brown spots. Endoscopic evaluation was performed with Fujinon EG-450 WR5 flexible fiberoptic endoscopies under white light. The statistical significance level (a) was taken as 5% in the calculations and the SPSS (IBM SPSS for Windows, ver.22) statistical package program was used for the calculations. Power was obtained by taking at least 0.80 and Type-1 Error (a) 0.05 for each variable when computing the sample size for our investigation. Descriptive statistics for continuous (quantitative) variables are expressed as median, mean, standard deviation, minimum and maximum. Pearson correlation analysis was used to investigate the link between antrum diseases and H. pylori. The study was carried out in accordance with the Principles of the Declaration of Helsinki. Approval for the study was obtained from the ethics committee of Istanbul University, Istanbul Faculty of Medicine (Date: 25.06.2020, No: 104060).

RESULTS

A total of 203 patients with PHG, 119 male and 84 female, were included in our study. There were 135 patients under the age of 65 and 68 patients above the age of 65. In patients with PHG, the rate of H. pylori positivity was 15.3% (Table 1).

Table 1: H. pylori frequency

Groups	Frequency	Percentage
Negative	172	84.7%
Positive	31	15.3%

This rate was calculated to be 18.5% for those under the age of 65, and 8.8% for those over the age of 65. On the basis of gender, H. pylori positivity rates were 21.8% in male patients and 6% in female patients. In the patients' histological examinations, the most prevalent antrum diseases in order of frequency are: reactive gastropathy (41.4%), active gastritis (23.6%), and edema (22.2%) (Table 2).

The rate of intestinal metaplasia was found to be 9.9%. When the relationship between antrum pathologies and H. pylori was considered, it was discovered that the relationships between vascularectasis-vascular congestion, edema, reactive gastropathy, active gastritis, lymphoid-hyperplasia variables, and H. pylori were statistically significant because the sigma value was greater than 0.05. When the correlation coefficients are examined, it is seen that the highest correlation is between active gastritis and H. pylori (r=0.666). As a result, there is a strong and positive relationship between active gastritis and H. py-

Table 2: Evaluation of antrum pathologies frequency distribution

	Groups	Frequency	Percentage
Vascular ectasia-Vascular congestion	Negative	178	87.7%
	Positive	25	12.3%
Edema	Negative	158	77.8%
	Positive	45	22.2%
Reactive gastropathy	Negative	119	58.6%
	Positive	84	41.4%
Intestinal metaplasia	Negative	183	90.1%
	Positive	20	9.9%
Foveolar hyperplasia	Negative	167	82.3%
	Positive	36	17.7%
Active gastritis	Negative	155	76.4%
	Positive	48	23.6%
Inactive gastritis	Negative	181	89.2%
	Positive	22	10.8%
Lymphoid hyperplasia	Negative	193	95.1%
	Positive	10	4.9%

lori. Following that, the strongest link is shown between reactive gastropathy and H. pylori, with the correlation coefficient being negative, indicating that H. pylori drops while gastropathy increases, and H. pylori increases while gastropathy declines (Table 3).

Other antrum diseases (intestinal metaplasia, foveolar hyperplasia, inactive gastritis) and H. pylori were not statistically significant because their sigma values were greater than 0.05.

DISCUSSION

The link between PHG and H. pylori infection has been fascinating, both in terms of prevalence and the consequences of these two diseases on the development and the severity of each other's disorders. Various research studies have been reported in this regard, evaluating both the influence of H. pylori on PHG and cirrhosis, as well as the presence and effect of H. pylori on the backdrop of PHG (8-10).

In a study conducted by Ozaydın N. et al., who investigated the prevalence of H. pylori in 2186 individuals in our country, they discovered an 82.5% prevalence of H. pylori positivity. Korkmaz M. et al. discovered a 49.5% positivity rate for H. pylori in healthy persons in another investigation (11, 12). In the study of Karadag M et al., one of the studies evaluating the frequency of H. pylori in cirrhotic patients, the prevalence of H. pylori was found to be 47%. Again, in the same study, the prevalence of H. pylori was found to be 27% in cirrhotic patients with portal gastropathy, and H. pylori in cirrhotic patients without portal hypertensive gastropathy, its prevalence was found to be 60% (13). In this research article, the prevalence of H. pylori in cirrhotic patients was similar to those found in a previous study analyzing H. pylori prevalence rates in the general population, while the incidence of H. pylori positivity was much lower in cirrhotic and PHG patients. These findings are similar to the low prevalence of H. pylori that we found in patients with PHG in our study. In our study, we found the frequency of H. pylori to be 15% in patients with portal hypertensive gastropathy. Again, in one of the few studies in the literature analyzing the prevalence of H. pylori in PHG, Mc Cormack et al. discovered that the frequency of H. pylori was 26% in cirrhotic patients and 38% in the control group. They also discovered that H. pylori positive reduced as the severity of the PHG rose (14). One reason for the reduced H. pylori positive in PHG is the lack of a favorable milieu for H. pylori colonization in the congestive gastric mucosa (15).

In contrast to these studies, which found that the frequency of H. pylori was reduced in PHG, Sathar SA, et al. examined a total of 140 cirrhotic patients, 70 with PHG and 70 without PHG, and discovered that the overall H. pylori positivity rate was 35.7%, while the H. pylori positivity rate in cirrhotic patients with PHG was 44.3 percent. whereas 27.1% did not have PHG (16). Similarly, Puri S. et al. investigated the prevalence of H. pylori in 60 cirrhotic patients and discovered a rate of H. pylori positivity

Table 3: Correlation analysis results between antrum pathologies and H. pylori

		H. pylori	H. pylori (Male)	H. pylori (Female)	H. pylori (Under age 65)	H. pylori (65 and above)
Vascular ectasia Vascular congestion	Pearson Correlation	**-0.159(*)**	-0.169	-0.113	**-0.175(*)**	-0.121
	Sig. (2-tailed)	0.023	0.067	0.308	0.043	0.324
	n	203	119	84	135	68
Edema	Pearson Correlation	**-0.227(*)**	**-0.280(*)**	-0.136	**-0.260(*)**	-0.158
	Sig. (2-tailed)	0.001	0.002	0.217	0.002	0.197
	n	203	119	84	135	68
Reactive gastropathy	Pearson Correlation	**-0.357(*)**	**-0.412(*)**	**-0.234(*)**	**-0.395(*)**	**-0.268(*)**
	Sig. (2-tailed)	0.000	0.000	0.032	0.000	0.027
	n	203	119	84	135	68
Intestinal metaplasia	Pearson Correlation	-0.002	0.003	0.051	-0.120	**0.264(*)**
	Sig. (2-tailed)	0.972	0.978	0.642	0.167	0.030
	n	203	119	84	135	68
Foveolar hyperplasia	Pearson Correlation	-0.054	-0.042	-0.016	-0.047	-0.040
	Sig. (2-tailed)	0.447	0.654	0.887	0.590	0.743
	n	203	119	84	135	68
Active gastritis	Pearson Correlation	**0.666(**)**	**0.746(**)**	**0.428(*)**	**0.690(**)**	**0.611(**)**
	Sig. (2-tailed)	0.000	0.000	0.000	0.000	0.000
	n	203	119	84	135	68
Inactive gastritis	Pearson Correlation	-0.104	-0.169	0.051	-0.091	-0.121
	Sig. (2-tailed)	0.140	0.067	0.642	0.294	0.324
	n	203	119	84	135	68
Lymphoid hyperplasia	Pearson Correlation	**0.283(*)**	**0.264(*)**	**0.291(*)**	**0.365(*)**	-0.054
	Sig. (2-tailed)	0.000	0.004	0.007	0.000	0.661
	n	203	119	84	135	68

*: According to the Pearson correlation coefficient (r), there is a weak degree of statistically significant correlation at the sig. 0.005 significance level.
**: According to the Pearson correlation coefficient (r), there is a moderate/high degree of statistically significant correlation at the 0.005 significance level.

of 55%. While the positive rate was 67% in patients with PHG, it was found to be 33% in patients without PHG (17). The reason for the higher prevalence of H. pylori in patients with PHG is increased expression of Inducible Nitric Oxide Synthase resulting in high reactive oxygen species, congestion, increased secretion of cytokines such as TNF alpha, IL-8, etc., and it increases the virulence of H. pylori while creating a synergistic effect between H. pylori and PHG (18). Our study differs from other studies examining the prevalence of H. pylori in PHG in terms of both number and examination method, because it has the maximum number of cases and H. pylori is identified by staining with Giemsa in samples taken from stomach biopsies in all patients. We think that this is the reason for the lower rate of H. pylori positivity prevalence we found compared to other studies in the literature.

The most common pathological finding in the antral biopsies we acquired from PHG patients was reactive gastropathy, followed by active gastritis, edema, and vascular ectatic alterations. According to Chandanwale SS. et al., who examined the gastrointestinal pathologies of 30 patients with PHG, the most prevalent pathological result was dilated congestive capillaries and edema (19). PHG is mostly characterized by mucosal changes in the proximal stomach, and therefore, in our study evaluating antral biopsies, unlike Chandanwale SS et al., it was thought that one of the reasons we saw edema and vascular conges-

tion in the third frequency might be related to biopsy localizations (19). In our investigation, we discovered a negative connection between H. pylori and gastropathy, similar to the findings of Mc Cormack et al. There was a positive correlation between active gastritis and H. pylori.

The positive features of our study are that we analyzed more patients than previous studies on the frequency of H. pylori in patients with PHG, and that H. pylori was tested by biopsy as a standard in all patients. The limitations of our study are the fact that it is a retrospective study, and the absence of cirrhotic differentiation in the etiology of PHG can be considered.

In conclusion, PHG is a diagnosis that necessitates both clinical and endoscopic assessment. Despite the fact that H. pylori is associated with active gastritis in this patient population, the prevalence of H. pylori in gastropathy is decreasing.

Informed Consent: Written consent was obtained from the participants.

Ethics Committee Approval: This study was approved by the Clinical Research Ethical Committee of the Istanbul University, Istanbul Faculty of Medicine (Date: 25.06.2020, No: 104060).

Peer Review: Externally peer-reviewed.

Author Contributions: Conception/Design of Study- B.Ç., A.Ç.Ö., F.A., K.D., S.F.B., S.K.; Data Acquisition- A.A.; Data Analysis/Interpretation- G.Y., S.E.; Drafting Manuscript- A.Ç.Ö., S.E., R.I., A.A.; Critical Revision of Manuscript- B.Ç.; Approval and Accountability- B.Ç., A.Ç.Ö., S.E., R.İ., A.A., G.Y., K.D., F.B., S.K., F.A.

Conflict of Interest: Authors declared no conflict of interest

Financial Disclosure: This study was partially funded by Scientific Research Projects Coordination Unit of Istanbul University. Project number: 37697.

REFERENCES

1. Feldman M, Freidman LS, Brandt LJ. Editors Sleisenger and Fordtran's Gastrointestinal and Liver Disease Pathophysiology/ Diagnosis/ Management Volume 1 Eleventh Edition 2021; p. 801-2.
2. Franchis R. Faculty Expanding consensus in portal hypertension Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. J Hepatol 2015;63(3):743-52. [CrossRef]
3. Gjeorgjievski M, Cappell MS. Portal hypertensive gastropathy: A systematic review of the pathophysiology, clinical presentation, natural history and therapy. World J Hepatol 2016;8(4):231-62. [CrossRef]
4. Vaishnavi B, Kate V, Ananthakrishnan N. Prevalence of Helicobacter pylori in patients with portal hypertensive gastropathy - a study from south India. Med Sci Monit 2004;10(4):CR133-6.
5. Ohta M, Yamaguchi S, Gotoh N, Tomikawa M. Pathogenesis of portal hypertensive gastropathy: a clinical and experimental review. Surgery 2002;131(1 Suppl):S165-70. [CrossRef]
6. Payen JL, Cales P, Pienkowski P, Sozzani P, Kervran A, Frexinos J. et al. Weakness of mucosal barrier in portal hypertensive gastropathy of alcoholic cirrhosis. Effects of propranolol and enprostil. J Hepatol 1995;23(6):689-96. [CrossRef]
7. Kao CY, Sheu BS, Wu JJ. Helicobacter pylori infection: An overview ofbacterial virulence factors and pathogenesis. Biomed J 2016;39(1):14-23. [CrossRef]
8. Rossez Y, Gosset P, Boneca IG, Magalhães A, Ecobichon C, Reis CA, et al. The lacdiNAc-specific adhesin LabA mediates adhesion of Helicobacter pylori to human gastric mucosa. J Infect Dis 2014;210(8):1286-95. [CrossRef]
9. Mao T, Wang Y, Yin F, Zhao Q, Yang L, Ding X, et al. Association of endoscopic features of gastric mucosa with Helicobacter pylori infection in Chinese patients. Gastroenterol Res Pract 2016;2016:6539639. [CrossRef]
10. Zaigham A, Yakoob J, Usman MW, Shakir T, Hamid S, Jafri W. Effect of Helicobacter pylori and its virulence factors on portal hypertensive gastropathy and interleukin (IL)-8, IL-10, and tumor necrosis factor-alpha levels. Saudi J Gastroenterol 2014;20(2):120-7. [CrossRef]
11. Özaydın N, Türkyılmaz SA, Cali S. Prevalence and risk factors of Helicobacter pylori in Turkey: a nationally-representative, cross-sectional, screening with the 13C-Urea breath test. BMC Public Health 2013;13:1215. [CrossRef]
12. Korkmaz M, Sadıç M, Koca G, Demirel K, Atılgan Hİ, Özyurt S, et al. Helicobacter Pylori Enfeksiyonunun Demografik, Sosyoekonomik ve Çevresel Faktörler ile İlişkisi: Ankara Hastanesi Deneyimi Kafkas J Med Sci 2013;3(2):74-9. [CrossRef]
13. Karadağ M, Ünsal B, Yörük G, Akyol Z, Sadun Koşay S. Sirozlu hastalarda portal gastropati ve helicobacter pylori enfeksiyonu sıklığı. Türkiye Klinikleri J Gastroenterohepatol 1999;10(1):1-5.
14. Mc Cormack TT, Sims J, Eyre-Brook I. Gastric lesion in portal hypertension: Inflammatory gastritis or congestive gastropathy? Gut 1985;26(11):1226-32. [CrossRef]
15. Abbasi A, Bhutto AR, Butt N, Munir SM, Dhillo AK. Frequency of portal hypertensive gastropathy and its relationship with biochemical, haematological and endoscopic features in cirrhosis. J Coll Physicians Surg Pak 2011;21(12):723-6.
16. Sathar SA, Kunnathuparambil SG, Sreesh S, Narayanan P, Vinayakumar KR. Helicobacter pylori infection in patients with liver cirrhosis: prevalence and association with portal hypertensive gastropathy. Ann Gastroenterol 2014;27(1):48-52.
17. Puri S, Jain M, Narayan KS, Pandey S, Nijhawan S. Helicobacter pylori infection in patients with portal hypertensive gastropathy owing to liver cirrhosis: Prevalence and relation with severity of gastropathy. J Dig Endosc 2017;8(3):123-8. [CrossRef]
18. Elsebaey MA, Tawfik MA, Elshweikh SA, Negm MS, Elnaggar MH, Alghazaly GH, et al. Impact of Helicobacter pylori Infection on Gastric Variceal Bleeding among Patients with Liver Cirrhosis. Gastroenterol Res Pract 2019;2019:6529420. [CrossRef]
19. Chandanwale SS, Gupta N, Sheth J, Naragude P, Gambhir AS, Pathak P, et al. Histopathological study of portal hypertensive gastropathy using gastric biopsy. Med J DY Patil Vidyapeeth 2017;10(6):562-7.