Original Article

Relationship between Upper GI symptoms and Endoscopic findings with Gastric H Pylori density.

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Abstract:

Objective: Helicobacter pylori affect many individuals in developed and developing countries. Inflammation caused by H pylori differs depending on the virulence factors, bacterial density and host response of bacteria. This study is designed to investigate the association between density of H Pylori colonization in gastric mucosa in biopsy specimens and severity of gastric mucosal inflammation.

Methods: This study of 75 patients was done at Department of Gastroenterology, Ghurki Trust Teaching Hospital Lahore. These patients presented with GI symptoms and got endoscopy done between June 2023 to June 2024. Their histopathology reports were retrospectively screened and severity of inflammation and H Pylori density were analyzed by Sydney scoring. Data analysis was done using SPSS software version24. **Results:** The analysis of H. pylori density and its association with gastrointestinal symptoms and endoscopic findings reveals that most gastrointestinal symptoms, including epigastric pain, nausea, and retrosternal burning, show no significant association with H. pylori density. On endoscopy findings evaluation only esophagitis is associated with higher H. pylori densities (p < 0.001), indicating a potential link between the bacterium and this condition. Other findings, such as moderate gastritis and duodenitis, show trends toward association but are not statistically significant.

Conclusion: Our study shows that density of H Pylori infection has no influence over Upper GI symptoms and also endoscopic findings cannot be taken as evidence of H pylori infection.

Keywords: Endoscopy, H Pylori, Esophagitis, Gastritis, Duodenitis.

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Introduction:

Helicobacter pylori are flagellated, spiral-shaped, microaerophilic, Gram-negative bacteria, which infect Gastric mucosa of almost half of the world's population. Poor sanitation conditions and non-availability of safe and clean water make it a common infection in the under-developed world. Its colonization in gastric Mucosa leads to mucosal inflammation, which can lead to a wide variety of diseases ranging from Gastritis, Peptic

Ulcer Disease to Gastric Carcinoma and MALT Lymphoma.² Along with H Pylori infection, several host factors such as life habits, Genotype and immunological response also contribute towards disease severity. This finding is based on the fact that prevalence of H Pylori in PUD varies in different geographical areas and only 10 % of infected people develop clinically significant disease.³ Furthermore, it has been seen that eradication of H pylori leads to worsening or

development of Gastroesophageal Reflux disease.

H. Pylori induced gastritis is highly prevalent in developing countries. In Pakistan as well, a high prevalence is reported. According to a local research, 88 percent of dyspeptic gastritis patients had H. Pylori infection. 4 Inflammation of Gastric mucosa leads to atrophy, intestinal metaplasia, dysplasia and gastric carcinoma.⁵ In the data from developed world as well, intestinal metaplasia and atrophy are considered as premalignant disorders associated with H. Pylori induced chronic gastritis.6 So, it means that fundamental step which leads to complications is Gastric atrophy. Hence, the role of H. Pylori should be studied at all levels, i.e., initial infection, atrophy and its associated symptoms, and the intestinal metaplasia and possible carcinoma or lymphoma. Moreover, the bacterial density has been correlated with gastric inflammation. Another local study showed that the density of H. Pylori on biopsy proven gastritis is positively correlated histological evidence with of chronic inflammatory infiltrate.7 In another local study it was found that greater the load of H. Pylori infection, the higher is the degree of neutrophilic activity, atrophy and intestinal metaplasia.8

The objectives of present study were not limited to chronic gastritis alone, but also included evaluating the bacterial density in relation to the severity of all endoscopic findings, as well as the common GI symptoms with which patients presented for upper GI endoscopy.

Methods:

This cross-sectional correlational study was carried out at Gastroenterology and Endoscopy department of Ghurki Trust Teaching Hospital in collaboration with Pathology department of LMDC/GTTH. A total of 75 patients who presented with upper GI symptoms and underwent endoscopy with biopsy were evaluated from July 2024 to December 2024.

Indications of endoscopy like pain epigastrium, nausea, retrosternal Burning etc. were noted (Table 1) Endoscopy was performed by same Endoscopist using Olympus Endoscope XQ160 series to clear the interobserver variation. Seventy-five Gastric antral biopsies of chronic

gastritis patients were included in the study. Gastric biopsies of patients who were on anti H Pylori therapy or had received H. Pylori eradication treatment in past were excluded.

Baseline data which included age, gender, symptoms, history, concomitant medication (especially antibiotics) and endoscopic findings were entered in patient's proforma. Gastric biopsy tissue was processed by same histopathologist after staining with hematoxylin and eosin. Giemsa stain was then used for H. Pylori demonstration. The Sydney System of classification of Histopathological features of Chronic Gastritis was used and gastritis was classified as none, mild, moderate and severe on a scale of 0-3. Similarly, density of H pylori was evaluated based upon histopathological evaluation.

The H. Pylori density was graded as follows:

0: none

1: H. Pylori seen only in one place

2: just a few H. Pylori seen

3: dispersed H. Pylori seen in separate foci

4: numerous H. Pylori in separate foci

5: almost complete coverage of gastric surface by layer of H. Pylori

6: uninterrupted coverage of gastric surface by a dense layer of H. Pylori

None was considered when no H Pylori was seen. Mild was 1-2, Moderate was 3-4 and severe was 5-6.

This classification provided numerical data for statistical analysis and is widely used. Before grading these specimens, two pathologists agreed with a consensus on the scoring of gastritis.

SPSS (Statistical Package for Social Sciences) for Windows 20.0 program was used for the statistical analysis. Descriptive statistics for continuous variables was summarized as mean and standard deviation, and descriptive statistics for categorical data was summarized as percentage. The Chi-Square test was used to compare the data in the categorical structure.

Correlation between parameters was done by using Pearson correlation analysis. The results were evaluated with a confidence interval of 95% and in a significance level of p < 0.05.

Results:

In this study, a total of 75 patients were diagnosed with upper gastrointestinal symptoms; among these, more than half of the cases, 47(62.7%), were females, while fewer were males, 28(37.3%), with a mean age of 38.84 ± 14.52 ranging from 17-84 years. The mean age of male and female patients was 36.68 ± 14.32 and 40.13 ± 14.64 years, respectively.

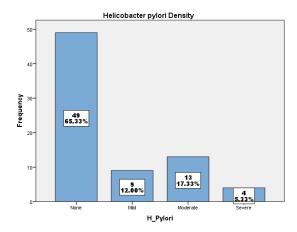
Table 1: Distribution of patients according to gastrointestinal symptoms & Endoscopic Findings

Parameters	N(%)				
Gastrointestinal symptoms	Yes	No			
Epigastric_pain	60(80)	15(20)			
Nausea	35(46.7)	40(53.3)			
Vomiting	14(18.7)	61(81.3)			
Retrosternal burning	36(48)	39(52)			
Malena	1(1.3)	74(98.7)			
Hematemesis	1(1.3)	74(98.7)			
Burping	3(4)	72(96)			
Belching	3(4)	72(96)			
Endoscopic Findings					
Esophagitis	54(72)	21(28)			
Gastritis Mild	3(4)	72(96)			
Gastritis Moderate	25(33.3)	50(66.7)			
Gastritis Severe	12(16)	63(84)			
Duodenitis	28(34.7)	49(65.3)			
Antral Gastritis	33(44)	42(56)			
Gastric Ulcer	1(1.3)	74(98.7)			
Duodenal Ulcer	5(6.7)	70(93.3)			
Evaluation of H. pylori	Positive	Negative			
Esophagitis	54(72)	21(28)			
	26(34.7)	49(65.3)			

The above table evaluates the distribution of gastrointestinal symptoms and endoscopic findings in patients, aiming to understand their correlation with H. pylori density. The most common gastrointestinal symptoms reported

among patients were epigastric pain (80%), retrosternal burning (48%), and nausea (46.7%). Less frequently observed symptoms included vomiting (18.7%), melena (1.3%), hematemesis (1.3%), burping (4%), and belching (4%). Endoscopic examination revealed esophagitis was the most prevalent finding, affecting 72% of the patients. Other significant findings included Antral Gastritis (44%), Duodenitis (34.7%), Moderate Gastritis (33.3%), and Severe Gastritis (16%). Mild gastritis was observed in only 4% of the cases, while gastric ulcers and duodenal ulcers were found in 1.3% and 6.7% of the patients, respectively. The evaluation for H. pylori presence showed that 34.7% of the patients tested positive for the bacterium. This significant presence indicates that H. pylori plays a notable role in the development of gastrointestinal symptoms and associated pathologies.

Figure 1 shows the distribution of patients according to H. pylori density.



The figure presents the distribution of H. pylori density among 75 patients, categorized into four levels: None, Mild, Moderate, and Severe. The majority of patients, 49 out of 75 (65.3%), showed no detectable H. pylori presence, indicating that a significant portion of the patient cohort does not have an active H. pylori infection. H. pylori density was classified as mild in 9 patients, representing 12.0% of the total sample, suggesting that a smaller segment of the population has a low level of H. pylori infection. A moderate density of H. pylori was found in 13 patients, accounting for 17.3% of the cohort, indicating a clear prevalence of moderate

infection levels among the patients. Only 4 patients (5.3%) had a severe density of H. pylori, reflecting a relatively low prevalence of high-density infections in the studied population.

Table 2: Association between H. pylori density, gastrointestinal symptoms & Endoscopic Findings

	H. pylori density					
Parameters		None	Mild	Moderate	Severe	p-value
Epigastric pain	Yes	39(65.0)	7(11.7)	11(18.3)	3(5.0)	.966
	No	10(66.7)	2(13.3)	2(13.3)	1(6.7)	
Nausea	Yes	22(62.9)	5(14.3)	6(17.1)	2(5.7)	.947
	No	27(67.5)	4(10.0)	7(17.5)	2(5.0)	
Vomiting	Yes	7(50.0)	3(21.4)	2(14.3)	2(14.3)	.206
	No	42(68.9)	6(9.8)	11(18.0)	2(3.3)	
Retrosternal burning	Yes	21 (58.3)	5(13.9)	9(25.0)	1(2.8)	.270
~u.m.g	No	28(71.8)	4(10.3)	4(10.3)	3(7.7)	
Malena	Yes	1(100)	-	-	-	.911
	No	48(64.9)	9(12.2)	13(17.6)	4(5.4)	
Hematemesis	Yes	1(100.0)	-	-	-	
	No	48(64.9)	9(12.2)	13(17.6)	4(5.4)	.911
Burping	Yes	2(66.7)	-	1(33.3)	-	.800
	No	47(65.3)	9(12.5)	12(16.7)	4(5.6	
Belching	Yes	2(66.7)	-	1(33.3)	-	.800
	No	47(65.3)	9(12.5)	12(16.7)	4(5.6)	
Esophagitis	Yes	33(61.1)	9(16.7)	12(22.2)	-	**<.001
	No	16(76.2)	-	1(4.8)	4(19.0)	
Gastritis Mild	Yes	3(100)	-	-	-	.646
	No	46(63.9)	9(12.5)	13(18.1)	4(5.6)	
Gastritis	Yes	20	-	3(12.0)	2(8.0)	.077
Moderate	No	(80.0) 29(58.0)	9(18.0)	10(20.0)	2(4.0)	
Gastritis	Yes	7(58.3)	2(16.7)	3(25.0)	-	.657
Severe	No	42(66.7)	7(11.1)	10(15.9)	4(6.3)	
Duodenitis	Yes	14(53.8)	5(19.2)	6(23.1)	1(3.8)	.326
	No	35(71.4)	4(8.2)	7(14.3)	3(6.1)	
Antro gastritis	Yes	20(60.6)	6(18.2)	6(18.2)	1(3.0)	.442
	No	20(64.5)	1(3.2)	7(22.6)	3(9.7)	
Gastric Ulcer	Yes	1(100)	-	-	-	.911
	No	48(64.9)	9(12.2)	13(17.6)	4(5.4)	
Duodenal Ulcer	Yes	3(60)	-	2(40)	-	460
	No	48(65.7)	9(12.9)	11(15.7)	4(5.7)	.468

**statistically significant at 0.01 level of significance

The analysis of H. pylori density and its association with gastrointestinal symptoms and endoscopic findings reveals that gastrointestinal symptoms, including epigastric pain, nausea, and retrosternal burning, show no significant association with H. pylori density. However, vomiting is more common in patients with severe H. pylori density, though not significantly. Notably, esophagitis is significantly associated with higher H. pylori densities (p < 0.001), indicating a potential link between the bacterium and this condition. Other findings, such as moderate gastritis and duodenitis, show trends toward association but are not statistically significant. These insights highlight importance of considering H. pylori density in the context of esophagitis and potentially other gastrointestinal conditions. (Table 2)

Table 3: Association between H. pylori & gastrointestinal symptoms

Parameters	N(%)			
Gastrointestinal	Positive	Negative	p-value	
symptoms				
Epigastric_pain	21(35)	39(65)		
Yes No	5(33.3)	10(66.7)	.903	
NO	- ()	1(111)		
Nausea	12(27.1)	22((2.0)		
Yes	13(37.1)	22(62.9)	.673	
No	13(37.1)	27(67.5)		
Vomiting				
Yes	7(50.0)	7(50.0)	.220	
No	19(31.1)	42(68.9)		
Malena				
Yes	-	1(100)	1.000	
No	26(35.1)	48(64.9)	1.000	
Retrosternal burning				
Yes	15(41.7)	21(58.3)	.221	
No	11(28.2)	28(71.8)		
Hematemesis				
Yes	-	1(100)	1.000	
No	26(35.1)	48(64.9)	1.000	
Burping				
Yes	1(33.3)	2(66.7)	1.000	
No	25(34.7)	47(65.3)		
Belching				
Yes	1(33.3)	2(66.7)	1.000	
No	25(34.7)	47(65.3)		

Table 3 presents the association between various gastrointestinal symptoms and the presence of H. pylori. The parameters considered include epigastric pain, nausea, vomiting, melena, retrosternal burning, hematemesis, burping, and belching. For each symptom, the table provides the number and percentage of positive and negative H. pylori cases, along with the p-value, to indicate statistical significance.

For epigastric pain, 35% of positive cases and 65% of negative cases were reported, with a p-value of .903, indicating no significant association with H. pylori. Similarly, nausea showed no significant association, as 37.1% of positive cases and 62.9% of negative cases had a p-value of .673. Vomiting had a higher percentage of positive cases at 50%, compared to 31.1% of negative cases, but the association was not statistically significant (p = .220). For melena, data was missing for positive cases, and the p-value was 1.000, suggesting no significant association.

Retrosternal burning was reported in 41.7% of positive cases and 58.3% of negative cases, with a p-value of .221, showing no significant association. Hematemesis also had missing data for positive cases and a p-value of 1.000, indicating no significant association. Both burping and belching showed similar results, with 33.3% positive cases and 66.7% negative cases and a p-value of 1.000, suggesting no significant association with H. pylori.

DISCUSSION

H. Pylori is a gram negative, spiral-shaped bacterium which is responsible for Gastric inflammation and possible complications like Gastric Atrophy, Intestinal metaplasia and MALT lymphoma¹². A lot of studies have been done to establish the relationship of these findings and density of H Pylori with variable results.^{3,4,5} Similarly, it has been attempted in past to predict the presence of H pylori based upon the symptomatology but no clear association has been established.⁷ Our study had two objectives. First, to find out symptoms associated with presence of H Pylori and secondly the possible association of severity of endoscopic findings and degree of colonization of H pylori.

Detection of H. pylori by optical microscopy is considered to be an efficient method This method is highly rated because of its potential of definite diagnosis of H. pylori infection, thus indicating gastric inflammation.8 In the study by Lobo Gatti and collaborators, histological test was found to be most sensitive for H. pylori detection, compared to others like urease test and culture tests. This is the reason that we used this test for evaluation of H pylori. Our results show that 34 % of patients were found to be positive for H pylori. This value is far less than the values seen in other studies of third world countries. Two studies from Brazil showed an incidence of 85% and 78 %, 10 while a Jordanian study 11 showed a value of 82 %. Global prevalence of H pylori has been estimated by WHO to be around 35 % which is guite close to our numbers. While another local data from Pakistan shows a value of 73%8. So, it can be concluded that exact value varies amongst various communities, sample cohort and symptomatology.

H. pylori do play a role in the pathogenesis of various gastric diseases (gastritis, ulcer, cancer) because it leads to mucosal destruction. Exact mechanism of this mucosal damage is unknown, but it is found out that proteases released by H Pylori damage the mucus structure by increasing the gastric acid secretion.

The most common gastrointestinal symptoms reported among patients were epigastric pain (80%) followed by retrosternal burning (48%) but only 35% of patients were H pylori positive, which was statistically in-significant. Majority of those patients who experienced epigastric pain did not have H Pylori infection. So, it means that merely pain is not a positive indicator for presence of H Pylori infection. This symptom correlation has been studied in various studies. According to local data, pain has been related to H Pylori infection.^{7,8} Main reason for this difference of result may be due to multiple reasons, especially, NSAIDs intake and various different cohort of patients. When it comes to most common endoscopic finding in patients having infection, only esophagitis was found to be statistically significant while gastritis, duodenitis and presence of ulcer were not found to be related to H Pylori infection. It is, in contrast

to previous work done in the region¹⁹ where gastritis was found to be the major endoscopic finding.

Although amongst the available data there was a statistically significant relation between the intensity of H. pylori and the severity of inflammation in study by sarin. This study revealed that as the intensity of H. pylori increases, there is increase in the severity of inflammation as well.¹³ In another study done by Yakoob, et al. a significant relationship was found between intensity of H. pylori colonization and chronic inflammatory gastric activity. 14 In a performed on Histopathological examination of endoscopic biopsy specimens of 461 patients, Türkay, et al. also concluded that as the intensity of H. pylori increased, the intensity of inflammation also increased proportionately.¹⁵ Similarly, in another work done by Alagöz, et al. a significant correlation was observed between lymphoplasmacytic cell infiltration inflammation activation and severity of H Pylori infestation¹⁶.In contrast, to these positive associations in a study of 272 gastric biopsy specimens by Ardakani, et al. no significant relationship was found between the density of H. pylori and the severity of chronic gastritis activity. These findings are in strong agreement to our findings that there is no well-defined association of H pylori infection and degree of gastric inflammation.¹⁷ In a study done in India Choudhary, et al. also found no statistically significant relationship between H. pylori density and chronic gastritis. 18 This is also in agreement to our findings. But variation between studies is because of variation in genomic structure of h pylori and life style differences and antimicrobial resistance of organism in different strains.

CONCLUSION:

Our study concludes that H pylori infection has a variable symptom profile depending upon demographics and concomitant risk factors. Similarly, endoscopic findings cannot be taken as evidence of H pylori infection. For proper detection we have to rely on investigations of H Pylori.

REFERENCES:

- 1. Chen YC, Malfertheiner P, Yu HT, Kuo CL, Chang YY, Meng FT et al. Global Prevalence of Helicobacter pylori Infection and Incidence of Gastric Cancer Between 1980 and 2022. Gastroenterology. 2024 Apr;166(4):605-619. doi: 10.1053/j.gastro.2023.12.022.
- 2. Sardar M, Kumar D, Aakash F, Partab F, Kumar S, Barkha F, Danesh F et al. Prevalence and etiology of *Helicobacter pylori* infection in dyspepsia patients: a hospital-based cross-sectional study. Ann Med Surg (Lond). 2023 Apr 4;85(4):665-669. doi: 10.1097/MS9.00000000000000120.
- 3. Khalid H, Zubair A, Malik TM, Ayyub M, Muhammad I. A. histopathological analysis of chronic inflammatory infiltrate in patients of H pylori associated chronic gastritis. Pak Armed Forces Med J. 2015; 65: 36-41.
- 4. Khalid H, Zubair A, Kiran N et al. Histopathological evaluation of H pylori density and its correlation with activity, atrophy and intestinal metaplasia. JIIMC 2018;13(1): 26 -32
- 5. Zhang C, Yamada N, Wu YL, Wen M, Matsuhisa T, Matsukura N. Helicobacter Pylori infection, glandular atrophy and intestinal metaplasia in superficial gastritis, gastric erosion, erosive gastritis, gastric ulcer and early gastric cancer. World J Gastroenterol. 2005; 11: 791–6.
- Ozdil K, Sahin A, Kahraman R, Yuzbasioglu B, Demirdag H, Calhan T, et al. Current prevalence of intestinal metaplasia and Helicobacter pylori infection in dyspeptic adult patients from Turkey. Hepatogastroenterol. 2010; 57: 1563-6.
- 7. Fareed R, Abbas Z, Shah MA. Effect of Helicobacter pylori density on inflammatory activity in stomach. J Pak Med Assoc. 2000; 50: 148-5
- 8. Mehmood K, Awan AA, Muhammad N, Hasan F, Nadir A. Helicobacter pylori

- prevalence and histopathological findings in dyspeptic patients. J Ayub Med Coll Abbottabad. 2014; 26: 182-5.
- Lobo Gatti L., Agostinho J.N.F., De Lábio R., et al. Helicobacter pylori and cag A and vac A gene status in children from Brazil with chronic gastritis. Clin Exp Med 2003;3:166-72
- 10. Almeida Cunha R.P., Alves F.P., Rocha A.M., et al. Prevalence and risk factors associated with Helicobacter pylori infection in native populations from Brazilian Western Amazon. Trans R Soc of Trop Med Hyg 2003;97:382-6.
- Bani-Hani K.E., Hammouri S.M. Prevalence of Helicobacter pylori in Northern Jordan. Saudi Med J 2001; 22:843-7.
- 12. Hui PK, Chan WY, Cheung PS, et al. Pathologic changes of gastric mucosa colonized by Helicobacter pylori. Hum Pathol 1992;23: 548-556.
- 13. Sayin S. The Relation between Helicobacter Pylori Density and Gastritis Severity. Int Arch Intern Med 3:019. doi.org/10.23937/2643-4466/1710019
- 14. Yakoob MY, Hussainy AS. Chronic gastritis and helicobacter pylori: A histopathological study of gastric mucosal biopsies. J Coll Physicians Surg Pak 2010;20: 773-775
- 15. Turkay C, Erbayrak M, Bavbek N, et al. Helicobacter pylori and histopathological findings in patients with dyspepsia. Turk J Gastroenterol 2011;22: 122-127.
- 16. Alagoz S, Turkay C, Yonem O et al. The relationship between helicobacter pylori intensity and histopathological findings in cases with chronic gastritis and duodenal ulcer. Turk J Gastroenterol 2002;13: 98-102.
- 17. Ardakani A, Mohammadizadeh F. The study of relationship between helicobacter pylori density in gastric mucosa and the severity and activity of chronic gastritis. JRMS 2006;11: 282.
- 18. Choudhary CK, Bhanot UK, Agarwal A, et al. Correlation of h. pylori density with grading of chronic gastritis. Indian J Pathol Microbiol 2001;44: 325-328.

19. Tanni NN, Ahmad S, Anwer S et al. Endoscopic and histopathological findings in adult patients and their association with Helicobacter pylori infection in Dhaka Bangladesh. IJID Regions 2 2022; 30–34

Author's Contribution:

AM: Conceived and designed the study, involved in data collection, performed statistical analysis and writing the manuscript.

II, UA, SS, MF, SSQ, UH: Collected the data, critical review and preparation of manuscript.

All authors have read, approved the final manuscript and are responsible for the integrity of the study.