ORIGINAL ARTICLE

FREQUENCY AND FACTORS LEADING TO *HELICOBACTER PYLORI* INFECTION AMONG DYSPEPTIC PATIENTS

Saira Muhammad Ali, Syed Zea Ul Islam Farrukh, Syed Afzal ul Haq Haqqi, Arif Rasheed Siddiqui, Muneer Sadiq*, Saad Khalid Niaz

Patel Hospital Karachi, *Altibri Medical College and Hospital Karachi--Pakistan

Background: *Helicobacter pylori* is infecting 50 percent or more of the world's population, putting it the most ubiquitous infection on the world. This study is done with the objective to determine the frequency and risk factors of *Helicobacter pylori* infection among dyspepsia patients at Patel Hospital Karachi. **Methods:** This cross-sectional study was conducted at the gastroenterology department at the Patel Hospital in Karachi from 10th Jan to 10th July 2021. All patients with dyspepsia for at least 6 months having age 20-60 years of either gender were included. Three samples from stomach (2 from antrum,1 from corpus) for biopsies were collected from each patient. The specimen was sent to the microbiology department of the hospital and was reported as having histopathological confirmation of *Helicobacter pylori* infection. **Results:** Of 111 patients with dyspepsia, mean age of the patients was 44.19±16.41 years. Most of the patients (n=65, 58.6%) were males and 46 (41.4%) were females. The mean duration of dyspepsia was 11.48±5.53 months. *Helicobacter pylori* was discovered to be present in 93 percent of individuals (83.8 percent). The odds of *Helicobacter pylori* infection were found to be 7.99 times higher among patients over 40 years old (AOR: 7.99, 95 percent CI: 2.02-31.64, *p*: 0.003), 3.93 times higher among patients with >9 months of dyspepsia (AOR: 3.93, 95 percent CI: 1.09-14.16, p: 0.036), and 11.85 times higher among smokers as compared to non-smokers (AOR: 11.85, 95 percent CI: 1.42-99.08, *p*-value 0.023). **Conclusion:** The rate of *Helicobacter pylori* infection in patients with dyspepsia was found to be higher. Furthermore, increasing age, increase duration of dyspepsia and smoking is found to be independent risk factors.

Keywords: Dyspepsia; *Helicobacter pylori* Infection; Smoking

Citation: Ali SM, Farrukh ZUI, Haqqi SAH, Siddiqui AR, Sadiq M, Niaz SK. Frequency and factors leading to *helicobacter pylori* infection among dyspeptic patients. A single centre study. J Ayub Med Coll Abbottabad 2022;34(3):507–10.

DOI: 10.55519/JAMC-03-10745

INTRODUCTION

Helicobacter pylori infection is a global problem with a wide range of differences across the globe.[1,2] *Helicobacter pylori* is a gram-negative bacterium with strong links to elevated gastrointestinal problems, such as peptic ulcers and malignant conditions.[3] The causes of dyspepsia, ranging from esophagitis to cholelithiasis and gastro duodenitis to peptic ulcer disease, are widespread and severe.[4] *Helicobacter pylori* must be recognized a carcinogenic agent, according to a report issued by the World Health Organization.[5] According to published research, *Helicobacter pylori* is infecting 50 percent or more of the world's population, putting it the most ubiquitous infection on the world.[6] Another study has reported that *Helicobacter pylori* infection resulted in 75% of non-cardia gastric malignancy all over the world.[7] The aim of this study was to determine the burden and negative factors in histopathology of *Helicobacter pylori* infection in people with dyspepsia. The data from this study will provide the basis for estimating the frequency and magnitude of infection by *Helicobacter pylori* in our population so that better understanding and adequate management of the disease can be offered for dyspepsic symptoms to be reduced.

MATERIAL AND METHODS

This cross-sectional study was carried out in the gastroenterological unit of the Patel Hospital Karachi from 10th Jan to 10th July 2021. This study was carried out after approval by the institute's ethical review committee. Thos who consented were enrolled the Gastroenterology Department, Patel Hospital, Karachi.

Consecutive patients with dyspepsia for at least 6 months having age 20–60 years of either gender were included. Whereas non-consenting patients, pregnant females, patients with history of peptic ulcer disease, gastric malignancy or MALT lymphoma, or patients who had previously been treated for *Helicobacter pylori* infection were excluded.

Using the WHO software, this sample size was calculated. By taking the prevalence of *H pylori* in dyspeptic patients as 88.3 percent[8], margin of error as 6 percent and confidence level (C.I) as 95 percent. The required sample size came out to be 111. Brief history of dyspepsia and demographic data like age, gender was taken. All dyspeptic patients fulfilling inclusion criteria underwent upper gastrointestinal endoscopy performed by gastroenterologist with over five years of experience. Three samples from stomach (2 from antrum,1 from

corpus) for biopsies were collected from each patient. The specimen was sent to the microbiology department of the hospital and was reported as having histopathological confirmation of *Helicobacter pylori* infection. Dyspepsia is classified as positive if any combination of four symptoms is present: it is severe enough to interfere with daily activities and occurs at least three times per week over the last three months, with a start of at least six months in advance.

Patients having a histopathology finding proven *Helicobacter pylori* infection after endoscopic biopsy were used to label *Helicobacter pylori* infection positive. H&E staining was used to establish the presence of *Helicobacter pylori*, and Giemsa staining was used as necessary. *Helicobacter pylori* infection was detected by curved, spirochete-like bacteria in the superficial mucus layer and along the microvilli of epithelial cells. A smoker was defined as someone who smoked at least five cigarettes a day for at least a year.

The findings of quantitative variables like (age and duration of dyspepsia) and qualitative variable (age, gender, site of biopsy, hypertension, smoking, peptic ulcer disease history in family, and *Helicobacter pylori* infection) was noted. Statistical analysis was carried out using SPSS Version 21. Age and duration of dyspepsia were estimated using mean and standard deviations, whereas gender, smoking status, family medical history of peptic ulcer disease, and *Helicobacter pylori* infection (yes/no) were computed using frequency and percentages. Inferential statistic was explored using binary logistic regression taking active status of *Helicobacter pylori* as outcome variable.

RESULTS

The patients were 44.19±16.41 years old on average. There were 58 (52.3%) patients under the age of 40 and 53 (47.7%) patients over the age of 40. Majority of the patients (n=65, 58.6%) were males, while 46 (41.4%) were females. The mean duration of dyspepsia was 11.48±5.53 months. There were 60 (54.1%) patients with ≤9 months of duration of dyspepsia and 51 (45.9%) had >9 months of duration of dyspepsia. Smoking status was found positive in 33 (29.7%) patients whereas family history of peptic ulcer disease was found in 23 (20.7%) patients. The frequency of *Helicobacter pylori* was found to be 93 (83.8%). A significant association of *Helicobacter pylori* were found with age (p: 0.004), duration of dyspepsia (*p*: 0.027), and smoking (*p*: 0.014). (Table-1)

Regression analysis indicated that the probabilities of *Helicobacter pylori* infection were 5.81 times greater in patients over 40 years of age compared to individuals under 40 years of age (OR: 5.81, 95% CI: 1.58-21.44, p: 0.008). The risk of *Helicobacter pylori* infection was shown to be 3.57 times greater in individuals who had dyspepsia for more than 9 months compared to those who had dyspepsia for less than 9 months (OR: 3.57, 95% CI: 1.09-11.67, *p*: 0.035). In smokers, the risk of *Helicobacter pylori* infection is 8.92 times higher than in non-smokers (OR: 8.92, 95% CI: 1.13–70.08, *p*: 0.038). Somewhat similar findings were observed in multivariable analysis as well. The findings of multivariable analysis revealed that after adjustment of all other co-variates, the odds of *Helicobacter pylori* was found to be 7.99 times significantly higher among patients with >40 years of age as compared to ≤40 years of age (AOR: 7.99, 95% CI: 2.02–31.64, *p*: 0.003). The odds of *Helicobacter pylori* were found to be 3.93 times significantly higher among patients with >9 months of duration of dyspepsia as compared to ≤9 months of duration of dyspepsia (AOR: 3.93, 95% CI: 1.09–14.16, *p*: 0.036). The risk of *Helicobacter pylori* infection was shown to be 11.85 times greater in smokers than in non-smokers (AOR: 11.85, 95% CI: 1.42-99.08, *p*: 0.023). (Table-2)

Table-1: Comparison of *Helicobacter pylori* with general characteristics of the patients (n=111)

Variables	Total	*Helicobacter Pylori* Infection		
		Positive (n=93)	Negative (n=18)	*p*-value
Age, years				
≤40	58	43 (46.2)	15 (83.3)	0.004
>40	53	50 (53.8)	3 (16.7)	
Gender				
Male	65	54 (58.1)	11 (61.1)	0.810
Female	46	39 (41.9)	7 (38.9)	
Duration of dyspepsia, months				
≤9	60	46 (49.5)	14 (77.8)	0.027
>9	51	47 (50.5)	4 (22.2)	
Smoking status				
Non-smoker	33	32 (34.4)	1 (5.6)	0.014
Smoker	78	61 (65.6)	17 (94.4)	
Family history of PUD				
Yes	23	20 (21.5)	3 (16.7)	0.643
No	88	73 (78.5)	15 (83.3)	

Table 2: Regression analysis of variables associated with *H. pylori* infection (n=111)

	Active *H. pylori* infection				
	Positive (n=93)	**OR (95% CI)**	***p*-value**	**AOR (95% CI)**	***p*-value**
Age, years					
≤40	43	Ref		Ref	
>40	50	5.81 (1.58-21.44)	0.008	7.99 (2.02-31.64)	0.003
Gender					
Male	54	Ref		-	
Female	39	0.81 (0.31-2.47)	0.881		
Duration of dyspepsia, months					
≤9	46	Ref		Ref	
>9	47	3.57 (1.09-11.67)	0.035	3.93 (1.09-14.16)	0.036
Smoking status					
Non-smoker	32	Ref		Ref	
Smoker	61	8.92 (1.13-70.08)	0.038	11.85 (1.42-99.08)	0.023
Family history of PUD					
Yes	20	Ref		-	
No	73	1.37 (0.36-5.20)	0.644		
OR: Odds Ratio, CI: Confidence Interval					

DISCUSSION

The findings of this study have revealed a higher prevalence of *Helicobacter pylori* infection in our cohort. The prevalence of *Helicobacter pylori* in patients with dyspepsia was reported as 88% in the Mehmood et al study.[8] Similarly, Yasir et al found the prevalence in dyspepsia patients to be 69.3%.[9] However, a systematic review has reported prevalence of dyspepsia varied between 02–30%. While, Oling *et al* study showed the lower prevalence, i.e. 36%.[10] It is reported that extent of infection among developing nations is higher than in industrialized nation, probably due to poor sanitary conditions and standard of hygiene.[11] The global frequency of *Helicobacter pylori* has decreased, according to a recent systematic study, owing to improving living standards, including better socioeconomic situations and cleanliness levels.[12] Several studies have stated that drug-resistant *Helicobacter pylori* has grown in children in recent years, leading to increased eradication failure with triple first-line treatment.[13–16] A higher prevalence of *Helicobacter pylori* is also reported in other studies.[16,17]

Similar to our study findings, no association of *Helicobacter pylori* and gender is reported in a previous study.[17,18] However, the odds of *Helicobacter pylori* were found to be significantly higher among patients with increase age, increase months of duration of dyspepsia and smokers. In a study there were no apparent structural or biochemical abnormalities in most patients to explains their symptoms.[19] The pathophysiology behind this is complex and various pathophysiology and psychosocial factors have been suggested.[20,21] In addition, the dyspepsia may be episodic, recurrent or chronic, has also been reported in literature. Symptoms are frequently associated with food, but not always.[22] As a result, patients with dyspepsia are now empirically undergoing symptomatic care with prokinetics, antacids, and digestive enzymes that are vulnerable to adverse reactions and an extremely high risk of relapse.[23] Failure to diagnose or delay and the resulting inadequate management lead to greater morbidity, economic loss and even death in patients with malignancy.[24]

The limited sample size was one of the limitations. Secondly, the study lacks inclusion of certain important risk factors. Lastly, the study sample is based on a single private sector hospital. In spite of these limitations, this study is of great importance as it has determined the local burden of disease from the private sector hospital. The finding of this study will ultimately result in improved patient's outcome and quality of life.

CONCLUSION

The prevalence of *Helicobacter pylori* infection has been found to be higher. In addition, increased age, elevated dyspepsia duration and smoking are independent risk factors.

AUTHORS' CONTRIBUTION

SMA, SZUIF: Conceptualization of the study design, write-up. SAHH, ARS, MS, SKN: Data collection, data analysis, data interpretation, proof reading.

REFERENCES

1. Atherton JC, Blaser MJ. Coadaptation of *Helicobacter Pylori* and humans: Ancient history, modern implications. J Clin Invest 2009;119(9):2475–87.
2. Lehours P, Yilmaz O. Epidemiology of *Helicobacter Pylori* infection. Helicobacter 2007;12(Suppl 1):1–3.
3. Khan SS, Zulfiqar A, Danish KF, Sauwal M, Bashir S, Zaman S. Prevalence of H. *Pylori* infection in patients with

gastroduodenal disease in Pakistan. Rawal Med J 2008;33(1):88–9.
4. Talib A, Shujai M, Mahmood K, Farooqui AN, Mustufa G. Various methods to detect *Helicobacter pylori* and their respective yield. Ann Abbasi Shaheed Hosp Karachi Med Dent Coll 2005;10(2):698–3.
5. de Martel C, Ferlay J, Franceschi S, Vignat J, Bray F, Forman D, *et al*. Global burden of Cancer attributable to H. *pylori* infection: a review and synthetic analysis. Lancet Oncol 2013;3(6):607–15.
6. Essawi T, Hammoudeh W, Sabri I, Sweidan W, Farraj MA. Determination of *Helicobacter pylori* Virulence Genes in Gastric Biopsies by PCR. ISRN Gastroenterol 2013;2013:606258.
7. Dixon MF. Pathology of gasitritis and peptic ulceration. In: Mobley HLT, Mendz GI, Hazell, editors. H. pylori: Physiology and Genetics. Washington DC: ASM Press; 2001.
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(2):182–5.
9. Yasir S, Moin F, Akhtar SM. Frequency of *Helicobacter Pylori* Infection on histopathology in patients with dyspepsia. Am J Clin Med Res 2014;2(3):53–6.
10. Oling M, Odongo J, Kituuka O, Galukande M. Prevalence of *Helicobacter pylori* in dyspeptic patients at a tertiary hospital in a low resource setting. BMC Res Notes 2015;8:256.
11. Farshad S, Japoni A, Alborzi A, Zarenezhad M, Ranjbar R. Changing prevalence of *Helicobacter pylori* in south of Iran. Iran J Clin Infect Dis 2010;5(2):65–9.
12. Zamani M, Ebrahimtabar F, Zamani V, Miller WH, Alizadeh-Navaei R, Shokri-Shirvani J, *et al*. Systematic review with meta-analysis: the worldwide prevalence of *Helicobacter pylori* infection. Aliment Pharmacol Ther 2018;47(7):868–76.
13. Oleastro M, Cabral J, Ramalho PM, Lemos PS, Paixão E, Benoliel J, *et al*. Primary antibiotic resistance of *Helicobacter pylori* strains isolated from Portuguese children: a prospective multicentre study over a 10 year period. J Antimicrob Chemother 2011;66(10):2308–11.
14. Nguyen TV, Bengtsson C, Yin L, Nguyen GK, Hoang TT, Phung DC, *et al*. Eradication of *Helicobacter pylori* in children in Vietnam in relation to antibiotic resistance. Helicobacter 2012;17(4):319–25.
15. Butenko T, Jeverica S, Orel R, Homan M. Antibacterial resistance and the success of tailored triple therapy in *Helicobacter pylori* strains isolated from Slovenian children. Helicobacter 2017;22(5):e12400.
16. Shu X, Yin G, Liu M, Peng K, Zhao H, Jiang M. Antibiotics resistance of *Helicobacter pylori* in children with upper gastrointestinal symptoms in Hangzhou, China. Helicobacter 2018;23(3):e12481.
17. Tadesse E, Daka D, Yemane D, Shimelis T. Seroprevalence of *Helicobacter pylori* infection and its related risk factors in symptomatic patients in southern Ethiopia. BMC Res Notes 2014;7:834.
18. Rasheed F, Yameen A, Ahmad T, Bilal R. Rate of active *Helicobacter pylori* infection among symptomatic patients of Pakistan. Malay J Pathol 2017;39(1):69–72.
19. Ferrucci PF, Zucca E. Primary gastric lymphoma pathogenesis and treatment: what has changed over the past 10 years? Br J Haematol 2007;136:521–38.
20. Ola SO, Yakubu A, Otegbayo JA, Oluwasola AO, Ogunbiyi JO, Akang EE, *et al*. The most appropriate site for endoscopic biopsy for the detection of H. *Pylori* among Nigerians in Ibadan. West Afr J Med 2006;25:269–72.
21. Hashemi MR, Rahnavardi M, Bikdeli B, Zahedani M. H *Pylori* infection among 1000 southern Iranian dyspeptic patients. World J Gastroenterol 2006;12:5479–82.
22. Alazmi WM, Siddique I, Alateeqi N, Al-Nakib B. Prevalence of *Helicobacter pylori* infection among new outpatients with dyspepsia in Kuwait. BMC Gastroenterol 2010;10:14.
23. Galbán E, Arús E, Periles U. Endoscopic findings and associated risk factors in primary health care settings in Havana, Cuba. MEDICC Rev 2012;14(1):30–7.
24. Malik MF, Hussain T, Khan MN, Mirza SA, Faroo M. *Helicobacter Pylori* infection in patients with dyspeptic symptoms having normal endoscopy. Pak Armed Forces Med J 2010;60(1):30–2.

Submitted: March 3, 2022	*Revised: May 10, 2022*	*Accepted: May 10, 2022*

Address for Correspondence:
Syed Zea Ul Islam Farrukh, Patel Hospital Karachi-Pakistan
Email: dr_zea@hotmail.com