Original Article

Frequency of Helicobacter Pylori in Perforated Peptic Ulcer and Associated Risk Factors

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ABSTRACT

Objective: To determine the frequency and risk factors of Helicobacter pylori infection in patients diagnosed with perforated peptic ulcer disease.

Study Design: Cross-sectional study

Place and Duration of Study: This study was conducted at the Surgical Unit Bakhtawar Amin Medical & Dental College, from January 2023 to December 2023.

Methods: A total of 384 patients over the age of 12 with peptic ulcer perforation were included. Detailed histories were taken regarding potential causes of peptic ulcers, such as Helicobacter pylori infection, alcohol consumption, current or past significant illnesses, socioeconomic status and use of tobacco in betel nut or chewing form.

Results: In our study, the presence of helicobacter pylori was found in 230 (59.9%) patients. It was seen that the presence of helicobacter pylori was higher in males 146 (65.8%) than females 76 (34.2%), (p=0.006). Presence of helicobacter pylori was higher in smokers 56 (70.9%) than non-smokers, 23 (29.1%), (p=0.025). Similarly, presence of helicobacter pylori was higher in betel nut & pan chewing used patients 30 (78.9%), than 8 (21.1%) betel nut & pan chewing not used patients.

Conclusion: H. pylori infection was not significantly associated with PPU. However, factors such as alcohol intake and tobacco use were involved in perforation. Therefore, we can conclude that while H. pylori infection is not a risk factor for PPU, it is important to avoid other risk factors.

Key Words: H Pylori, Peptic ulcer disease, Risk factors, Perforation

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INTRODUCTION

Over the recent past, the incidence of peptic ulcer disease has reduced mainly by virtue of the PPI use and H pylori eradication[1]. However, complications such as perforation are still apart of the present picture and a cause for worry in the field of healthcare. This prolongation of complications may have an association with increasing proportion of geriatrics and excessive use of non-steroidal anti-inflammatory drugs (NSAIDs)[2].

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Iatrogenic duodenal perforations were formerly rare, but the growing incidence of endoscopic procedures like endoscopic retrograde cholangiopancreatography (ERCP) has led to this problem[3]. Thus, the treatment of choice for duodenal ulcers remains still a matter of controversy, and the diagnosis is frequently made at a rather late stage, which detrimental effect on the patient's survival[16].

Even till 1983 Waren & Marshall discovered the link between Helicobacter pylori and peptic ulcers, the consensus was that stress, dietary factors and increased ulcer acid secretion caused peptic ulcers[7]. Helicobacter pylori infection is estimated to affect about 50% of the world's population[7], with the distribution in the developing world being higher than in the western countries. More particularly, studies show that increased noise levels adversely affect task completion rates (92 % reduction), worker productivity (66. 6%, and 70% of duodenal perforation patient had history of H. pylori infection respectively [8].

Duodenal perforation though rare constitutes a serious threat to health and has a mortality index of between eight and twenty-five percent as has been witnessed in different research studies[9,10]. The first account of the perforated duodenal ulcer was given by Muralto in the year 1688 in the patient that was reported by

Lenepneau[1]. An omental patch which is not a new technique in such circumstances was for the first time described by Cellan-Jones in 1929[13]. Besides, the first laparoscopic surgery conducted for a perforated duodenal ulcer was in the year 1904[14].

As much as H. pylori infection is not rare in our community, published data indicates that incidence of this infection in cases of duodenal perforation might differ across regions. There is a dearth of literature regarding the correlation between H. pylori and duodenal perforation[15] and hence our work. Thus, identifying to what extent duodenal perforation patients treated in the local health care facility are infected with H. pylori, it will be possible to gain essential understanding of the extent of the problem as observed in the local setting. Altogether, these results will be reported to the gastroenterologists and surgeons in the area which in turn will enable them practice informed decision making in future research and practice.

METHODS

This cross-sectional study was conducted in Bakhtawar Amin Medical & Dental College, from January 2023 to December 2023. Patients presented at emergency and outpatient department of hospital with sign and symptoms of acute peritonitis due to perforation of peptic ulcer disease were enrolled. Sample size was calculated from previous study findings, H. pylori infection 52%, confidence interval 95% and margin of error 5%. Openepi software was used for estimation if sample. History of patients regarding use of tobacco (chewing or betel nut), alcohol use, socioeconomic status, diabetes, hypertension, smoking, and area of residence were taken.

Serology for H. pylori was performed immediately after diagnosing peptic ulcer perforation. Any silent perforations of peptic ulcers, including those of patients on medication. Patients with co-morbidities such as cardiovascular disease, arthritis, asthma, or any other condition for which the patients were taking medication were excluded. SPSS version 27.1 used for analysis of data. Test of significance were t test and chi square with significant p value of 0.05 or below was considered.

RESULTS

Overall, 384 patients were included in our study, with mean age 46.12±9.05 years. There 222 (57.8%) males and 162 (42.2%) females. Mean BMI of the study patients was 25.86±2.96 kg/m^2, and most of the patients 234 (60.9%) had BMI more than 25 kg/m^2. Majority of the patients 236 (62.5%) lived in urban areas, whereas 148 (38.5%) lived in rural areas. There were 311 (81.0%) patients had low socioeconomic status and 73 (19.0%) patients had high socioeconomic status. Diabetes mellitus was found in 105 (27.3%) patients, whereas hypertension was found in 100 (26.0%) patients. Furthermore, 79 (20.6%) patients were smokers. Mean peptic ulcer disease of the patients was 10.27±3.93 years, and majority of the patients had up to 11 years of peptic ulcer disease. Whereas use of betel nut & pan chewing was observed in 38 (9.9%) patients. (Table. No. 1).

In our study, the presence of helicobacter pylori was found in 230 (59.9%) patients. (Figure. I). Association of helicobacter pylori with demographics and baseline profile was shown in table. No. 2. It was seen that the presence of helicobacter pylori was higher in males 146 (65.8%) than females 76 (34.2%), (p=0.006). Presence of helicobacter pylori was higher in smokers 56 (70.9%) than non-smokers, 23 (29.1%), (p=0.025). Similarly, presence of helicobacter pylori was higher in betel nut & pan chewing used patients 30 (78.9%), than 8 (21.1%) betel nut & pan chewing not used patients. (Table. No. 2).

Table. No .1: Demographics and baseline profile

Variable	N (%)	Mean ± S.D
Age (years)		46.12±9.05
Up to 40	87 (22.7)	
More than 40	297 (77.3)	
BMI (kg/m^2)		25.86±2.96
Up to 25	150 (39.1)	
More than 25	234 (60.9)	
Gender		
Male	222 (57.8)	
Female	162 (42.2)	
Area of residence		
Urban	236 (62.5)	
Rural	148 (38.5)	
Socioeconomic status		
Low	311 (81.0)	
High	73 (19.0)	
Diabetes mellitus	105 (27.3)	
Hypertension	100 (26.0)	
Smoking	79 (20.6)	
Peptic ulcer disease (years)		10.27±3.93
Up to 11	237 (61.7)	
More than 11	147 (38.3)	
Use of betel nut & pan chewing	38 (9.9)	

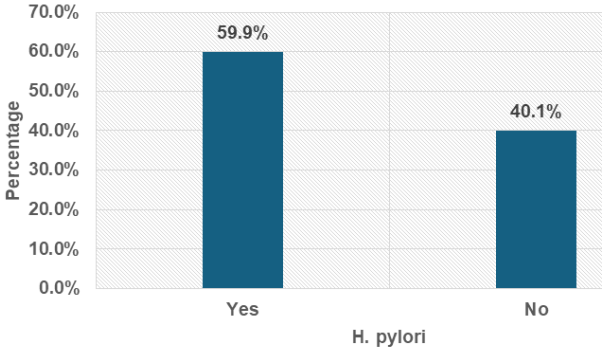

Figure No. 1:Presence of helicobacter pylori

Table. No. 2: Association of helicobacter pylori with demographics and baseline profile

Variable	Helicobacter pylori		p-value
	Yes	No	
Age (years)			
Up to 40 years	49 (56.3)	38 (43.7)	0.439
More than 40 years	181 (60.9)	116 (39.1)	
BMI (kg/m^2)			
Up to 25	97 (64.7)	53 (35.3)	0.127
More than 25	133 (56.8)	101 (43.2)	
Gender			
Male	146 (65.8)	76 (34.2)	0.006
Female	84 (51.9)	78 (48.1)	
Area of residence			
Urban	146 (61.9)	90 (38.1)	0.320
Rural	84 (56.8)	64 (43.2)	
Socioeconomic status			
Low	189 (60.8)	122 (39.2)	0.470
High	41 (56.2)	32 (43.8)	
Diabetes mellitus			
Yes	69 (65.7)	36 (34.3)	0.154
No	161 (57.7)	118 (42.3)	
Hypertension			
Yes	68 (68.0)	32 (32.0)	0.065
No	162 (57.0)	122 (43.0)	
Smoking			
Yes	56 (70.9)	23 (29.1)	0.025
No	174 (57.0)	131 (43.0)	
Peptic ulcer disease (years)			
Up to 11	144 (60.8)	93 (39.2)	0.661
More than 11	86 (58.5)	61 (41.5)	
Use of betel nut & pan chewing			
Yes	30 (78.9)	8 (21.1)	0.012
No	200 (57.8)	146 (42.2)	
N (%)			

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DISCUSSION

Perforated peptic ulcer is not just a local issue, but a prevalent emergency medical condition globally, characterized by a substantial risk of mortality, particularly among elderly patients[16]. Prompt surgical intervention to repair the perforation, coupled with comprehensive sepsis management, is critical for improving patient outcomes and reducing mortality rates. Rapid diagnosis and timely treatment are essential components of effective management, as delays can lead to complications such as peritonitis and systemic infection, which significantly worsen the prognosis. Consequently, healthcare providers must maintain a high index of suspicion and be prepared to initiate immediate surgical and medical treatment to optimize recovery and survival chances in affected individuals.

A study by Magsi et al[17] reported a 77.27% frequency of silent perforation, which refers to a perforation that occurs without the typical symptoms of a peptic ulcer, such as sudden severe abdominal pain. The significant risk factors identified were H. pylori infection and NSAID use. Screening for H. pylori and the use of antiulcer drugs can help reduce the risk of perforation in peptic ulcer disease (PUD).

The prevalence of H. pylori infection tends to increase with age and is influenced by socioeconomic status, as observed in both developed and developing countries. The prevalence among children can often indicate overall prevalence, given that initial H. pylori infections typically occur in early childhood and are rarely self-resolving. Hage et al[18] suggest that women infected with H. pylori can significantly influence their children's infection rates. Socioeconomic status and housing conditions during early childhood have been identified as critical factors affecting the infection rate in both adults and children.

In countries such as Saudi Arabia, India, and Vietnam, H. pylori infection rates range from 60% to 80%, which is significantly higher than the 20% to 25% infection

rates observed in developed nations like the United States, Australia, and France[19].

In our study, we found that 59.9% of cases with H. pylori infection, which is noticeably lower compared to the findings of Lowenthal et al,[20] who reported a prevalence of 73.9%, and Giannakis et al,[21] who reported 80.6%. Despite the ongoing debate over the relationship between H. pylori infection and perforated ulcers, the connection between the two is well-established for gastric ulcers.

CONCLUSION

H. pylori infection was not significantly associated with PPU. However, factors such as alcohol intake and tobacco use were involved in perforation. Therefore, we can conclude that while H. pylori infection is not a risk factor for PPU, it is important to avoid other risk factors.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Shabbir Ahmed, Muhammad Mumtaz Ather
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Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

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REFERENCES

1. Tadesse M, Musie E, Teklewold B, Hailu E. Prevalence of H. pylori in perforated peptic ulcer disease at Saint Paul's hospital millennium medical college, Addis Ababa, Ethiopia. Ethiopian J Health Sci 2021;31(5):969-974.
2. Seyoum N, Ethicha D, Assefa Z, Nega B. Risk factors that affect morbidity and mortality in patients with perforated peptic ulcer diseases in a teaching hospital. Ethiopian J Health Sci 2020 Jul 1;30(4):549-558.
3. Bokalli FA, McWright CF, Aseneh JB, Mbachan TM, Mokake ND, Nguedia JC, et al. Helicobacter pylori infections in peptic ulcer perforations: a retrospective analysis in two referral hospitals in Douala, Cameroon. Surg Sci 2020;11(10):298-311.
4. Gbenga OJ, Ayokunle DS, Ganiyu A, Adekova I. Pattern of presentation, management and early outcome in patients with perforated peptic ulcer disease in a semi-urban tertiary hospital. Ethiopian J Health Sci 2021;31(5):975-981.
5. Marshall B. Pylori in peptic ulcer disease. Germ theory. Med Pioneers Infectious Dis. 2023 Mar 29:295.
6. Sonnenberg A. Epidemiology of Helicobacter pylori. Aliment Pharmacol Therapeut 2022; 55:S1-3.
7. Fong IW, Fong IW. Helicobacter pylori infection: when should it be treated? Current Trends Concerns Infec Dis 2020:81-102.
8. Rahatullah HS. Prevalence and associated factors of peptic ulcer disease among dyspeptic patients at endoscopy unit in Kabul, Afghanistan. J Applied Pharm Sci Res 2023:6(2):19-24.
9. Singh LO, Maibam C, Singh TS, Singh SO. A clinical study of duodenal ulcer perforation. J Dent Med Sci 2020;19(8):07-29.
10. Khan SR, Alam J, Marwan M. Frequency of wound infection after duodenal ulcer perforation repair. Pak J Med Health Sci 2023;17(02):25-6.
11. Deshmukh SN, Parikh HP. Open versus laparoscopic repair of perforated duodenal ulcer: a comparative study. Int Surg J 2020;7(4):1004-8.
12. Pellicano R, Ribaldone DG, Fagoonee S, Astegiano M, Saracco GM, Mégraud F. A 2016 panorama of Helicobacter pylori infection: key messages for clinicians. Panminerva Med 2016; 58(4):304-17.
13. Alam M, Tahir R, Ahmad S, Amin S, Zareen A, Khan AG. Frequency of Helicobacter pylori (H. Pylori) among patients presenting with duodenal perforation. Pak J Med Health Sci 2023;17(03):142-4.
14. Crenner C. Operative innovation and surgical conservatism in twentieth-century ulcer surgery. J History Med Allied Sci 2023:jrad065.
15. Wang X, Qu J, Li K. Duodenal perforations secondary to a migrated biliary plastic stent successfully treated by endoscope: case-report and review of the literature. BMC Gastroenterol 2020;20:1-7.
16. Søreide K, Thorsen K, Harrison EM, Bingener J, Møller MH, et al. Perforated peptic ulcer. Lancet 2015;386:1288-98.
17. Magsi AM, Iqbal M, Malik M, Parveen S. Silent peptic ulcer disease perforation. J Surg Pak 2017 Apr;22:61-4.
18. Hage N, Renshaw JG, Winkler GS, Gellert P, Stolnik S, Falcone FH. Improved expression and purification of the Helicobacter pylori adhesion BabA through the incorporation of a hexa-lysine tag. Protein Expr Purif 2015;106: 25-30.
19. Dogra BB, Panchabhai S, Rejinthal S, Kalyan S, Priyadarshi S, Kandari A. Helicobacter pylori in gastroduodenal perforation. Med JDY Patil Univ 2014;7:170-2.
20. Lowenthal AC, Hill M, Sycuro LK, Mehmood K, Salama NR, Ottemann KM. Functional analysis of the Helicobacter pylori flagellar switch proteins. J Bacteriol 2009;191(23):7147-56.
21. Giannakis M, Chen SL, Karam SM, Engstrand L, Gordon JI. Helicobacter pylori evolution during progression from chronic atrophic gastritis to gastric cancer and its impact on gastric stem cells. Proc Natl Acad Sci 2008;105(11):4358-63.