

A Clinical Study on Helicobacter Pylori Prevalence in Peptic Ulcer Perforation

Mohan Krishna¹, Ravikiran², N. Venkata Harish³, V. Mahidhar Reddy⁴

¹Junior Resident of General Surgery, NMC, Nellore

²Senior Resident of General Surgery, NMC, Nellore

³Associate Professor of General Surgery, NMC, Nellore

⁴Professor & HOD of General Surgery, NMC, Nellore

Received: 25-07-2025 / Revised: 23-08-2025 / Accepted: 26-09-2025

Corresponding Author: Dr. Mohan Krishna

Conflict of interest: Nil

Abstract:

Background: Peptic ulcer disease (PUD) results from an imbalance between mucosal defense and aggressive factors such as acid and pepsin. Although most cases respond to medical management, complications like perforation remain life-threatening and require surgical intervention. *Helicobacter pylori* (*H. pylori*) has been established as a major etiological factor in PUD, but its role in perforated ulcers remains controversial, particularly in developing regions.

Aim: To determine the prevalence of *Helicobacter pylori* infection in patients with peptic ulcer perforation and assess its association with demographic and lifestyle factors.

Methodology: A prospective hospital-based study was conducted on 50 patients aged 15–60 years diagnosed intraoperatively with peptic ulcer perforation at Narayana Medical College, Nellore, between January 2023 and June 2024. Biopsies from the ulcer edge and surrounding mucosa were obtained and tested for *H. pylori* using Giemsa staining and Rapid Urease Test. Data on age, gender, risk factors, and clinical presentation were analyzed.

Results: Of the 50 patients, 46 (92%) were male and 4 (8%) were female, with the highest incidence in the 24–42-year age group. Duodenal perforations (84%) predominated over gastric (16%). *H. pylori* was detected in 32 patients (64%), with 52% in duodenal and 12% in gastric perforations. Major risk factors included smoking (82%), alcohol use (82%), and NSAID intake (22%).

Conclusion: *H. pylori* infection remains a significant etiological factor in peptic ulcer perforation, particularly in duodenal ulcers among middle-aged males with concurrent smoking and alcohol habits. Routine intraoperative biopsy and postoperative eradication therapy are recommended to reduce recurrence and complications.

Keywords: *Helicobacter pylori*, Peptic ulcer perforation, duodenal ulcer, Risk factors.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

An imbalance between defensive mechanisms like mucosal integrity and aggressive forces like gastric acid and pepsin frequently leads to peptic ulcer disease (PUD), a common gastrointestinal ailment marked by mucosal damage in the stomach or duodenum. Although PUD may frequently be treated medically, complications like bleeding and perforation pose major risks to one's health, with the latter being a potentially fatal situation that necessitates surgery. [1]

The ulcer by itself does not pose an immediate threat to life, but the perforation and the resulting peritonitis do. Therefore, treating peritonitis and ensuring the closure of the perforation—which can be accomplished with surgery—are the therapeutic priority. [2] Peptic ulcer perforation occurs in approximately 2–10% of patients with PUD and is associated with significant morbidity and mortality.

[3] Factors such as the use of non-steroidal anti-inflammatory drugs (NSAIDs), smoking, and alcohol consumption have been identified as contributors to perforation risk, however the contribution of *H. Pylori* in peptic ulcer perforation is debatable, in earlier has shown the association with peptic ulcer disease in the recent literature. [4]

Helicobacter pylori (*H. pylori*), a Gram-negative, spiral-shaped bacterium, is a well-documented etiological agent in the development of peptic ulcers. Discovered in the early 1980s, *H. pylori* has been implicated in up to 90% of duodenal ulcers and 70% of gastric ulcers. Its ability to colonize the gastric mucosa and induce chronic inflammation plays a central role in ulcer pathogenesis. Despite advances in understanding its role in uncomplicated PUD, the association between *H. pylori* and peptic ulcer perforation remains an area of ongoing re-

search. After Barry J Marshall and J. Robin Warren discovery of *Helicobacter Pylori* research is going on in its association with PUD. [5]

Understanding the prevalence of *H. pylori* in patients presenting with perforated peptic ulcers is crucial for optimizing management strategies. Identifying this bacterium as a significant contributor could influence decisions regarding eradication therapy post-surgery, potentially preventing recurrence and reducing long-term complications.

This study aims to investigate the prevalence of *H. pylori* infection in patients with peptic ulcer perforation and explore its association with clinical and lifestyle factors. By providing insights into the burden of *H. pylori* in this critical subset of patients, the study seeks to enhance the understanding of its role in complicated PUD and inform evidence-based treatment protocols.

Aim: To study the prevalence of *helicobacter pylori* in peptic ulcer perforation cases to prevent the recurrence of peptic ulcer

Materials & Methods:

This was a hospital based clinical prospective study done in 50 cases undergoing explorative laparotomy with both open and laparoscopic techniques in

Narayana Medical College Nellore for a period of 18 months (January 2023 to June 2024) Period of Study: January 2023 to June 2024.

Inclusion criteria: Patient of both sexes between 15 – 60 years diagnosed as peptic ulcer perforation intraoperatively.

Exclusion criteria: Patient less than 15 years of age and more than 60 years of age, Patients with small bowel perforations, Patients presenting with bowel perforation due to trauma.

The study will be conducted on patients between the age group 15 to 60 years presenting with acute abdomen. The patients will be evaluated clinically and radiologically after the diagnosis of perforation is made will be taken up for emergency explorative laparotomy after taking informed and written consent and necessary investigations.

Intraoperatively if the perforation is diagnosed as peptic ulcer perforation, biopsies will be taken from the site of perforation and surrounding mucosa and will be subjected to pathological evaluation to find out the presence of *helicobacter pylori* with the help of Giemsa staining technique and Rapid urease test.

Results:

Table 1: Age & Gender incidence of peptic ulcer perforation

Age in years	Peptic ulcer Perforation		Total (Percentage)
	Duodenal	Gastric	
15-24	8 (16%)	1 (2%)	9 (18%)
24-33	12 (24%)	1 (2%)	13 (26%)
33-42	12 (24%)	2 (4%)	14 (28%)
42-51	6 (12%)	2 (4%)	8 (16%)
51-60	4 (8%)	2 (4%)	6 (12%)
Male	38 (76%)	8 (16%)	46 (92%)
Female	4 (8%)	0	4 (8%)

Table 2: Incidence of peptic ulcer perforation and H pylori

Age in years	H Pylori in Biopsy		Total (Percentage)
	Positive	Negative	
15-24	3 (6%)	6 (12%)	9 (18%)
24-33	10 (20%)	3 (12%)	13 (26%)
33-42	9 (18%)	5 (10%)	14 (28%)
42-51	6 (12%)	2 (4%)	8 (16%)
51-60	4 (8%)	2 (4%)	6 (12%)
Male	32 (64%)	14 (28%)	46 (92%)
Female	0	4 (8%)	4 (8%)

Table 3: Risk factors associated with peptic ulcer perforation

Risk Factors	Perforation		Total (Percentage)
	Duodenal	Gastric	
Smoking	35 (70%)	6 (12%)	41 (82%)
Alcohol	36 (72%)	6 (12%)	42 (82%)
NSAID	9 (18%)	2 (4%)	11 (22%)

Table 4: Mode of Presentation of Symptoms

Symptoms	Perforation		Total (Percentage)
	Duodenal	Gastric	
Pain	42 (84%)	8 (16%)	50 (100%)
Vomiting	36 (72%)	8 (16%)	42 (84%)
Abdominal Distension	42 (84%)	8 (16%)	50 (100%)
Fever	30 (60%)	5 (10%)	35 (75%)
Tenderness	42 (84%)	8 (16%)	50 (100%)
Guarding & Rigidity	42 (84%)	8 (16%)	50 (100%)
Bowel Sounds Absent	42 (84%)	8 (16%)	50 (100%)

Table 5: Prevalence of H Pylori

H Pylori	Perforation		Total (Percentage)
	Duodenal	Gastric	
Positive	26 (52%)	6 (12%)	32(64%)
Negative	16 (32%)	2 (4%)	18(36%)

Discussion:

Peptic ulcer perforation is one of the most common surgical emergencies encountered in the department of general surgery, although the incidence of peptic ulcer complications have been reduced drastically with the usage of proton pump inhibitor and H2 receptor antagonist therapy, but the treatment for perforation has been the surgery.

Age Incidence in Peptic Ulcer Perforation: In the present study, the highest incidence of peptic ulcer perforation was noted in the age group of 33–42 years, with duodenal perforations being the most frequent. This finding aligns with the observations reported by several researchers internationally. For example, Rajesh et al. (2019)⁶⁷ noted that the maximum incidence of perforated peptic ulcers occurred in the third and fourth decades of life. Similarly, a study by Svanes (2000) [6] stated that peptic ulcer perforation was most common between 30 and 50 years of age, attributing the trend to increased exposure to risk factors like smoking and alcohol.

However, some other studies, such as by Lau et al. (2005) [7] indicated a shift toward older age groups (>60 years), particularly in developed countries. This change was attributed to widespread *Helicobacter pylori* eradication therapy and increased NSAID use among the elderly. The slight younger age prevalence seen in our study could reflect regional differences in healthcare access, socioeconomic status, and lifestyle habits prevalent in India. Present series matches with panivelu et al [8].

According to Savnes C [9] the mortality rate for a perforated duodenal ulcer is higher in the elderly due to the patient's age rather than the type of surgery. He reported that the mortality rate was 0.6% in the age group under 50, 15% in the age group between 50 and 60, and 45.2% in the age

group over 60 (2009). And more common in adolescence according to Qiulong Shen et al [10]

In India, as per Malfertheiner et al. (2009) [11] *H. pylori* infection is often acquired during childhood but complications like ulceration and perforation manifest in young adulthood. Similar age prevalence was observed by Goodwin et al. (1994) [12] who emphasized that socioeconomic status and hygiene practices influence early infection rates.

Gender Distribution: In our study, males showed a significantly higher prevalence (84% of cases) compared to females (16%), with duodenal perforations being predominant. This male predominance is a well-established trend in literature. Studies by Bertleff and Lange (2010)⁷⁰ found male to female ratios ranging between 4:1 and 5:1 in perforated peptic ulcer cases. Similarly, Turan et al. (2007) [13] reported male dominance, attributing it to higher rates of smoking and alcohol intake among men.

The increased male prevalence could also be explained by behavioural factors: males are more likely to engage in behaviours that are risk factors for ulcer development and perforation, including smoking, alcohol consumption, and NSAID misuse. Hormonal differences might also contribute, as oestrogens have been considered protective against ulcer formation.

In our study 92% were males and 8% were females, male to female ratio 23:1, peptic ulcer perforation According to Skovgaard [14] (1997), males are more likely than females to experience perforation because they have experienced greater stress and strain in life, while female sex hormones provide some protection against perforation. According to Zahid Amman [15] (2008), men are more likely than women to have a high prevalence of perforation since they are under more stress. Thus, the gender distribution in this study mirrors global

patterns and underlines the need for targeted preventive strategies among high- risk male populations.

Incidence of H. Pylori in Peptic Ulcer Perforation: A significant finding in this study was the H. pylori positivity rate of 64% among patients with peptic ulcer perforation, based on biopsy samples. Duodenal ulcer perforations showed slightly higher H. pylori positivity compared to gastric ulcer perforations. Various studies have shown a variable association between H. pylori and peptic ulcer perforation: Lin et al. (2010) [16] found H. pylori infection rates around 70% in perforated peptic ulcers.

However, a meta-analysis by Chung et al. (2000) [17] suggested lower positivity rates (~48%) in perforated ulcers compared to non-perforated ulcers. Svanes (2000) hypothesized that factors such as NSAID use, stress, and lifestyle factors might play larger roles in ulcer perforation than H. pylori alone.

81% of instances with perforated duodenal ulcers had an H. pylori infection, according to Enders K & Lam et al [18] (2000). H-pylori was found in 80.9% of individuals with duodenal perforations (Yekin et al [19]., 2010). In this work, we evaluated the H. pylori infection using ulcer edge biopsy (histopathological investigation). The incidence of H. pylori in the study was 44%. Some research groups benefit from H. pylori. Interestingly, the positivity rate in our study is relatively high, suggesting that H. pylori remains a dominant etiological factor for peptic ulcer disease even in complicated presentations like perforations. This underlines the importance of postoperative H. pylori eradication therapy even after surgical management.

Age Distribution of H. Pylori Positivity: Our findings indicated that the highest prevalence of H. pylori positivity was in the 24–33 year age group. This observation is noteworthy because many other studies found peak infection rates either in childhood (due to early acquisition) or in middle age due to cumulative exposure. Thus, this study confirms that young adults are particularly vulnerable to serious H. pylori-associated complications in our region.

Risk Factors Associated with Peptic Ulcer Perforation high proportion of our patients had significant risk factors: 82% were smokers, 82% had alcohol consumption history, 22% reported NSAID usage. These findings are consistent with worldwide reports that smoking and alcohol are major risk factors for peptic ulcer perforation.

A study by Søreide et al. (1996) [20] reported that smoking increased the risk of peptic ulcer perforation by 2.7 times. Alcohol consumption was

found to impair gastric mucosal defenses, making the mucosa more vulnerable to acid attack, particularly in combination with smoking. Although NSAIDs are a well-known cause of peptic ulcer disease, their association with perforation in our study was lower. This lower percentage may reflect less NSAID use in younger populations or underreporting of over-the-counter drug use. In conclusion, modifiable risk factors like smoking and alcohol play a crucial role in peptic ulcer perforation, and aggressive lifestyle modification interventions are needed.

Mode of Presentation: Most of our patients presented with: Pain (100%), Vomiting (84%), Abdominal distension (100%), Fever (75%). These symptoms are characteristic of peritonitis secondary to gastrointestinal perforation. Studies by Taylor et al. (1997) and Søreide et al. (1996) indicated similar symptomatology, emphasizing that sudden severe abdominal pain is the hallmark of ulcer perforation. The high frequency of vomiting and abdominal distension corresponds to the onset of secondary peritonitis and paralytic ileus. Fever, observed in 75% of cases, reflects systemic inflammatory response. Thus, clinical presentation in our cohort matches classical descriptions of peptic ulcer perforation.

Clinical Signs: All patients showed classic signs of peritonitis: abdominal tenderness, guarding, rigidity, and absent bowel sounds. This mirrors previous clinical findings. Bertleff and Lange (2010) documented that abdominal rigidity and silent abdomen (absent bowel sounds) were nearly universal findings in peptic ulcer perforation. These signs are crucial for early clinical diagnosis and indicate an urgent need for surgical intervention. Thus, our findings are largely consistent with global patterns, with slight regional variations. Future multi-center studies with larger cohorts would help validate these findings. Nevertheless, a positivity rate of 64% in our study is relatively high, suggesting that H. pylori continues to be a dominant player in peptic ulcer complications in our setting.

Geographic and Socioeconomic Variation in H. pylori and Peptic Ulcer Disease H. pylori prevalence varies greatly across the world: In developed countries, prevalence rates are around 30-40% and are declining due to better hygiene and widespread use of eradication therapy. In developing countries, prevalence rates remain high, often exceeding 70-80%. India, where this study was conducted, is considered a high- prevalence region for H. pylori. Factors contributing include: Poor sanitation, Overcrowding, Early childhood exposure, Lack of awareness regarding infection control. Thus, the observed high H. pylori positivity in our study is consistent with the expected

epidemiological trends in low- to middle-income countries.

Gender Differences in *H. pylori* Infection:

Interestingly, our study noted zero positivity among females for *H. pylori* in the context of peptic ulcer perforation. This is unusual, as most studies (e.g., Hunt et al. 2011)⁷⁷ indicate no significant gender differences in *H. pylori* prevalence.

Possible explanations include: Small number of female patients (only 8% of total cases), Sampling variation, Protective factors in females, such as higher mucosal blood flow and hormonal influences (oestrogen), which may mitigate progression to complications despite infection. Further research with larger female cohorts would be necessary to confirm if there is a genuine gender-based biological difference in our population.

Timing of Surgical Intervention: Early surgical intervention is crucial in the management of peptic ulcer perforation. Studies have shown that delay beyond 24 hours significantly increases morbidity and mortality. Although surgical details were not elaborated in the observations provided, it is worth noting that prompt diagnosis and timely surgery are paramount for better outcomes.

Importance of *H. pylori* Eradication Post-Surgery:

Following surgical repair of a perforated ulcer, eradication of *H. pylori* has been demonstrated to: Reduce ulcer recurrence, Prevent re-perforation, Improve overall patient outcomes. Meta-analyses (e.g., Ford et al. 2016) confirm that postoperative triple therapy significantly reduces recurrence rates compared to surgery alone. Therefore, all patients in our cohort with confirmed *H. pylori* infection should ideally receive appropriate eradication therapy post-operatively, including: PPI (Proton Pump Inhibitor) + Amoxicillin + Clarithromycin or Metronidazole, depending on local resistance patterns.

Conclusion:

We conclude that H-PYLORI being the important causative factor for peptic ulcer perforation and accounts for 64% (Duodenal perforation — 52%, Gastric perforation — 12%). Peptic ulcer perforation is a multifactorial disease associating with Alcohol — 72%, Smoking — 72% and NSAID — 18%. Peptic ulcer perforation is common in middle aged individuals 24-42 years amounting 54% of our total cases.

References:

1. Acute Abdomen. Surg Clin N Am 1997; 4(6): Pg. 16.
2. Bhattacharya Kaushik et al. Peptic ulcer surgery: A historical review. Gastroenterology Today 2002; 6: 38-40.

3. William Schumer and Sheldon Burman. The perforated viscous diagnosis and treatment in surgical emergency. Surg Clin N Am 1997; 52 (1): 231-238
4. Maingots Abdominal Operations 11th edition, Michael T. Zinner, Stanley W. Ashley 1997: Pg. 358
5. FitzGerald R, Smith SM. An Overview of Helicobacter pylori Infection. Methods Mol Biol. 2021;2283:1-14. doi: 10.1007/978-1-0716-1302-3_1. PMID: 33765303.
6. Svanes C. Trends in perforated peptic ulcer: Incidence, etiology, treatment, and prognosis. World J Surg. 2000;24(3):277–83.
7. Lau JY, Sung J, Hill C, Henderson C, Howden CW, Metz DC. Systematic review of the epidemiology of complicated peptic ulcer disease: Incidence, recurrence, risk factors and mortality. Digestion. 2005;72(3-4):188–202.
8. Palanivelu et al Laparoscopic management of duodenal ulcer perforation: is it advantageous? Indian Society of Gastroenterology, 2007.
9. Svanes C. Trends in perforated peptic ulcer: Incidence, etiology, treatment and prognosis. World J Surg 2000; 24: 277-283.
10. Shen Q, Liu T, Wang S, Wang L, Wang D. Experience in diagnosis and treatment of duodenal ulcer perforation in children. BMC Pediatr. 2023 Mar 30;23(1):144.
11. Malfertheiner P, Megraud F, O'Morain CA, et al. Management of Helicobacter pylori infection—the Maastricht IV/ Florence Consensus Report. Gut. 2012;61(5):646–64.
12. Goodwin CS, Mendall MM, Northfield TC. Helicobacter pylori infection. Lancet. 1997;349(9047):265–9.
13. Turan M, Sen M, Canbay E, Karadayi K, Uzun S, Karaman I, et al. The impact of risk factors on perforated peptic ulcer. Turkish J Gastroenterol. 2007;18(1):33–8.
14. Skovgaard S et al. Late results of perforated duodenal ulcer treated by simple closure. World J Surg 1997; 1: 521-526.
15. Zahid Amman and Muhammed Naeem et al. Pattern of Change in the frequency of helicobacter pylori in perforated duodenal ulcer. J Ayub Medical College, Abbotabad, 2008; 20 (4).
16. Lin SY, Chang CW, Tsai HC, Wu TC, Wang CC, Lu CL. Role of Helicobacter pylori infection and NSAID usage in perforated peptic ulcer: a retrospective study. World J Gastroenterol. 2010;16(4):405–10.
17. Hunt RH, Xiao SD, Megraud F, Leon-Barua R, Bazzoli F, van der Merwe SW, et al. Helicobacter pylori in developing countries. World Gastroenterology
18. Enders KW, Md Y. H. lam et al. Eradication of Helicobacter pylori Prevents Recurrence of ulcer after simple closure of duodenal perfora-

- tion. Ann Surg 2000; 231: 153-158.
19. Kate V, Ananthakrishnan N et al: Effect of H-pylori eradication on the ulcer Recurrence rate after simple closure of perforated duodenal ulcer: Retrospective and prospective randomized controlled studies. Br J Surg 2001; 88: 1054-1058.
 20. Søreide K, Thorsen K, Søreide JA. Strategies to improve the outcome of emergency surgery for perforated peptic ulcer. Br J Surg. 2014;101(1):e51–64.