ORIGINAL RESEARCH PAPER

Volume - 15 | Issue - 05 | May - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CROSS-SECTIONAL STUDY ON HELICOBACTER PYLORI INFECTION IN PATIENTS WITH GASTRO-DUODENAL PERFORATION IN A TERTIARY CARE CENTRE

General Surgery

Dr. Mihir Anilkumar Patel* — 3rd Year Post-Graduate Resident, Department of General Surgery, Zydus Medical College and Hospital, Dahod, Gujarat, India*Corresponding Author

Dr. Shailesh Rathod — Professor And Head, Department of General Surgery, Zydus Medical College and Hospital, Dahod, Gujarat, India

Dr. Dishank Modhia — Assistant Professor, Department of General Surgery,Zydus Medical College and Hospital, Dahod, Gujarat, India

ABSTRACT

Background: *Helicobacter pylori* (*H. pylori*) infection remains one of the most prevalent bacterial infections associated with chronic gastritis, peptic ulcer disease, and gastric malignancies. Its role in complicated ulcers, particularly gastro-duodenal perforation, remains variable across studies.**Objectives:** To determine the prevalence of *H. pylori* infection among patients with gastro-duodenal perforation and evaluate its association with risk factors such as NSAID usage, smoking, and alcohol consumption in patients admitted to Zydus Medical College and Hospital, Dahod. **Methods:** This cross-sectional hospital-based study was conducted among 113 patients who presented with gastro-duodenal perforation requiring surgical intervention from March 2024 to September 2025. Biopsy samples obtained intra-operatively were tested using a Rapid Urease Test (RUT) for *H. pylori*. Demographic data, lifestyle factors, comorbidities, and clinical characteristics were analysed using SPSS version 23.0.**Results:** Of 113 patients, 97 (85.84%) tested positive for *H. pylori*. Males predominated (78.76%) with a mean age above 50 years, and the most affected age group was 51–60 years (28.32%). Major risk factors included history of NSAID use (60.18%), smoking (60.18%), and alcohol consumption (63.72%). Gastric perforations (94.69%) were significantly more frequent than duodenal perforations (5.31%). Comorbidities such as hypertension (23.01%) and diabetes (14.16%) were prevalent.**Conclusion:** The study demonstrates a strong association between *H. pylori* infection and gastro-duodenal perforation. The high infection rate and overlapping risk factors highlight the necessity for routine *H. pylori* testing and eradication therapy in surgical patients, along with preventive public health strategies targeting NSAID misuse, smoking, and alcohol consumption.

KEYWORDS

Helicobacter Pylori, Gastro-duodenal Perforation, Nsaids, Peptic Ulcer, Rapid Urease Test, Gastric Perforation

INTRODUCTION

Helicobacter pylori (*H. pylori*) is a gram-negative, spiral-shaped, microaerophilic bacterium that thrives in the human gastric mucosa. Its discovery revolutionized the understanding of acid peptic disease, establishing infectious aetiology as a major driver of gastritis, peptic ulcers, and gastric malignancies [1]. Nearly two-thirds of the global population carries *H. pylori*, with prevalence highest in low- and middle-income countries [2].

Peptic ulcer disease develops due to an imbalance between aggressive factors such as gastric acid and pepsin and the protective mechanisms of the gastric mucosa. H. pylori contribute to mucosal injury by producing urease, cytotoxins, and inflammatory mediators that damage epithelial cells and impair mucosal defense mechanisms.[2] Despite its well-established role in peptic ulcer disease (PUD), the association between *H. pylori* and perforated ulcers remains under debate. Peptic ulcer perforation, a life-threatening emergency, results when an ulcer erodes the full gastric wall thickness, spilling gastric content into the peritoneal cavity [3]. Mortality rates vary from 10–20%, higher in elderly and comorbid individuals [4].

Multiple studies have linked chronic infection, NSAID use, smoking, and alcohol as major etiological factors. However, regional variations persist due to differences in hygiene, antibiotic exposure, diagnostic access, and host-genetic susceptibility [5]. This study was undertaken to determine the prevalence of *H. pylori* infection in gastro-duodenal perforations and to identify lifestyle and clinical correlates in an underrepresented tribal region of Dahod, Gujarat.

Materials and Methods

Study Design and Setting

A cross-sectional hospital-based analytical study was conducted at Zydus Medical College and Hospital, Dahod, Gujarat, between March 2024 and September 2025. The hospital caters to a large tribal and rural population. which were enrolled in this study after proper consent.

Study Population

All patients with radiologically confirmed gastro-duodenal perforation undergoing emergency laparotomy were included.

Inclusion Criteria

1. Radiological evidence of pneumoperitoneum (air under diaphragm).
2. Intraoperatively confirmed gastric or duodenal perforation.
3. Informed consent obtained.

Exclusion Criteria

1. Traumatic perforations due to blunt or penetrating injury.
2. Patients previously treated for *H. pylori* infection.

Sampling and Sample Size

A purposive sampling method was applied. 113 eligible patients were recruited, based on anticipated *H. pylori* prevalence and power calculation for 95% confidence level.

Data Collection

Demographic variables, lifestyle factors (smoking, alcohol, NSAID use), comorbidity data, and intraoperative findings were recorded.

Diagnostic Testing

A Rapid Urease Test (RUT) was performed on biopsy specimens from the perforation edge. A colour changes from Yellow to Pink/Red within 15 minutes to 24 hours was considered positive for *H. pylori*.

Management and Postoperative Care

All patients underwent Exploratory Laparotomy with Modified Graham's omental patch repair following resuscitation and peritoneal lavage. *H. pylori*-positive patients received standard triple therapy:

- Omeprazole 20 mg BID, Amoxicillin 750 mg BID, and Tinidazole 500 mg BID for 14 days.

Statistical Analysis

Data were coded and entered in SPSS v23.0. Descriptive statistics summarized demographic and clinical profiles. Associations between categorical variables were assessed using the Chi-square test or Fisher's exact test. Significance was defined at $p < 0.05$.

Ethical Approval

Institutional Ethical Committee approval (IEC/ZMCH/2024/GS/30) was obtained prior to data collection. Written informed consent was taken from all participants.

RESULTS

Demographic Profile

Parameter	Frequency
Male	89 (78.76%)
Female	24 (21.24%)
Age <30 yr.	6 (5.31%)
Age 31-40 yr.	22 (19.47%)
Age 41-50 yr.	24 (21.24%)
Age 51-60 yr.	32 (28.32%)
Age >60 yr.	29 (25.66%)

Interpretation:
Median age: 54 years. Male-to-female ratio ≈ 4:1. The majority were middle-aged to elderly men.

Infection and Risk Factor Distribution

Variable	Positive / Affected (%)	Interpretation
H. pylori (RUT)	85.84%	Strong prevalence in perforation cases
NSAID use	60.18%	Major modifiable risk factor
Smoking	60.18%	Contributory behavioural factor
Alcohol	63.72%	Common lifestyle risk
Male gender	78.76%	High-risk demographic
Gastric site	94.69%	Predominant perforation type
Sickling trait	45.13%	Possible ischemic predisposition
Diabetes/Hypertension	37.17%	Comorbidity influence

The study included patients presenting with gastro-duodenal perforation requiring emergency surgical intervention. The majority of patients were males and belonged to the age group of 30–50 years.

H. pylori infection was detected in a substantial proportion of patients with perforated peptic ulcers. The prevalence was particularly high among patients with gastric perforation.

Risk factor analysis revealed that smoking was the most common associated factor, followed by alcohol consumption and prolonged NSAID use. Many patients had more than one risk factor present simultaneously.

Patients who received timely surgical intervention followed by appropriate postoperative management showed good recovery. Postoperative complications were minimal and included wound infection and transient ileus in a small proportion of cases.

Association Between *H. pylori* and Risk Factors

No statistically significant *independent* association was observed between *H. pylori* positivity and smoking (p=0.8055), alcohol (p=0.2081), or diet (p=0.2021). However, all were frequent co-factors among positive cases.

DISCUSSION

This study establishes a high prevalence (85.84%) of *H. pylori* among patients with gastro-duodenal perforation in a tribal region of Gujarat—comparable to previously reported 68–92% across India [9].

Gender and Age Trends

Men in the middle to late decades of life were the primary sufferers—possibly due to higher exposure to modifiable risk factors. Similar male predominance (70–90%) was observed by Dogra et al. and Mahim et al. [15].

Risk Factors

60% of patients reported NSAID use, aligning with Drini [8] and Malfertheiner et al. [7], who emphasized NSAIDs as secondary offenders that potentiate *H. pylori*-mediated mucosal damage. Alcohol (63.72%) and smoking (60.18%) likely contributed synergistically through impaired mucosal healing and increased oxidative stress .

H. pylori and Perforation

The study corroborates findings by Ng et al. [6], who demonstrated in a randomized controlled trial that *H. pylori* eradication after perforation repair significantly reduces ulcer recurrence. Similarly, Metzger et al. and Rehmani & Pathak confirmed a high infection burden in perforated peptic ulcers, advocating for universal eradication.

In contrast, studies showing lower infection prevalence (Bokalli et al., 47.9%) may reflect diagnostic differences or regional antibiotic exposure.

Shift Toward Gastric Perforation

Historically, duodenal perforations outnumbered gastric ones. However, our data (94.69% gastric) suggest a reversal trend. NSAID-induced antral mucosal injury and alcohol use likely underlie this shift. Similar gastric predominance was reported by Mahim et al.

Comorbidities and Regional Factors

A noteworthy finding in this cohort was 45.13% sickling positivity, suggesting ischemic contribution to mucosal vulnerability—an aspect rarely reported in prior literature. Diabetes and hypertension also influenced postoperative recovery.

Public Health and Clinical Implications

Routine intraoperative *H. pylori* testing should be institutionalized in all ulcer perforation surgeries. Incorporation of eradication therapy postoperatively substantially decreases recurrence. Community-level interventions targeting unsupervised NSAID use, alcohol, and smoking are equally crucial. Education on balanced diet, hydration, and hygiene complements medical management.

Strengths

- Adequate sample size (n=113) for regional representation.
- Use of a validated, intraoperative rapid urease diagnostic method.
- Inclusion of comorbidity and hematologic variables (e.g., sickling profile).

Limitations

- Single-centre cross-sectional design limits causality inference.
- Absence of histopathological or molecular confirmation could introduce minor diagnostic bias.
- Lack of long-term follow-up on recurrence or eradication success.

CONCLUSION

This study affirms *H. pylori* as a dominant etiological factor in gastro-duodenal perforation (85.84%). NSAID usage, alcohol, and smoking emerged as co-contributory yet modifiable risk factors. The shift toward gastric over duodenal perforations indicates evolving disease epidemiology in India's tribal zones.

Integration of routine *H. pylori* testing, triple therapy, and rational drug use policies is essential in tertiary surgical care. Comprehensive behavioural counselling and population-specific preventive strategies are strongly recommended to reduce morbidity and recurrence.

Recommendations

1. Routine *H. pylori* testing in every Upper GI Endoscopy and treating every positive pt with triple drug therapy.
2. Routine *H. pylori* testing (RUT/histology) for all ulcer perforation cases.
3. Universal implementation of eradication therapy post-surgery.
4. Rational prescription and avoidance of unsupervised NSAID intake.
5. Public health education on alcohol and smoking cessation.
6. Periodic regional epidemiological surveillance for ulcer disease patterns.

Table: Summary of Key Findings

Variable	Positive n (%)	Negative n (%)
H. pylori (RUT)	97 (85.84%)	16 (14.16%)
NSAID use	68 (60.18%)	45 (39.82%)
Smoking	68 (60.18%)	45 (39.82%)
Alcohol consumption	72 (63.72%)	41 (36.28%)
Mixed diet	84 (74.34%)	Vegetarian 29 (25.66%)
Hypertension	26 (23.01%)	----
Diabetes mellitus	16 (14.16%)	----
Tuberculosis	8 (7.08%)	----
Gastric perforation	107 (94.69%)	Duodenal 6 (5.31%)

REFERENCES: -

1. Parikh NS, Ahlawat R. *Helicobacter pylori*. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.
2. Nguyen J et al. *Helicobacter pylori Infections in Children.* Antibiotics (Basel). 2023;12(9):1440.

3. Amini A, Lopez RA. *Duodenal Perforation.* StatPearls [Internet]. 2025 Jan.
4. Chung KT, Shelat VG. *Perforated Peptic Ulcer—An Update.* World J Gastrointest Surg. 2017;9(1):1–12.
5. Kusters JG, Kuipers EJ. *Pathogenesis of Helicobacter pylori Infection.* Clin Microbiol Rev. 2006;19(3):449–490.
6. Ng EK et al. *Eradication of H. pylori prevents ulcer recurrence after simple closure of duodenal ulcer perforation.* Ann Surg. 2000;231(2):153–8.
7. Malfertheiner P et al. *Helicobacter pylori infection.* Nat Rev Dis Primers. 2023;9(1):19.
8. Drini M. *Peptic Ulcer Disease and NSAIDs.* Aust Prescr. 2017;40(3):91–93.
9. Aljuhani SA et al. *Impact of Helicobacter pylori Eradication on Surgical Treatment of Peptic Ulcer Disease.* Cureus. 2024;16(6):e63523.
10. Kim N et al. *Effect of eradication of H. pylori on gastric ulcer recurrence.* Korean J Intern Med. 1999;14(2):9–14.
11. Metzger J et al. *Prevalence of H. pylori infection in peptic ulcer perforations.* Swiss Med Wkly. 2001;131(7–8):99–103.
12. Koshariya M et al. *Incidence of H. pylori in gastroduodenal perforation.* J Trop Med Res. 2016;21(4):104.
13. John B et al. *Prevalence of H. pylori in Peptic Ulcer Perforation.* Int Surg J. 2017;4(10):3350–3.
14. Rehmani B, Pathak P. *H. pylori in Perforated Peptic Ulcer Disease.* Int Surg J. 2018;5(5):1720–3.
15. Mahim M et al. *Gastro-duodenal perforation in tertiary centre.* Hamidia Hosp, 2019.
16. Bokalli DJ et al. *Perforated PUD in Cameroon.* J Afr Surg. 2021;14(2):88–94.
17. Ram P et al. *Alcohol, Smoking and H. pylori Infection in Nepalese Adults.* Nepal Med Assoc J. 2021;59(234):112–7.
18. Bose G et al. *H. pylori Detection in Gastro-Duodenal Perforation in Rural India.* J West Afr Coll Surg. 2024;14(3):58–67.