E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.48047/ijprt/16.01.743

Research Article

Prevalence of Helicobacter pylori Infection in Gastro-Duodenal Perforation and Its Clinical Implications: A Prospective Observational Study

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Received Date: 04/02//2026 Revised Date: 10/02/2026
Accepted: 15/02/2026 Published Date: 25/02/2026

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ABSTRACT

Background: Gastro-duodenal perforation is a surgical emergency with high morbidity and mortality, and peptic ulcer disease—frequently linked to Helicobacter pylori infection—is a common underlying cause. The role of H. pylori and the need for eradication therapy in perforation cases remain incompletely defined.

Aim: To determine the prevalence of H. pylori infection in gastro-duodenal perforation and to analyse its association with clinical variables and outcomes.

Materials and methods: A prospective observational study was conducted on 70 patients (aged 18–75 years) with gastro-duodenal perforation undergoing exploratory laparotomy. Two mucosal biopsies were taken from the perforation margin for rapid urease test and Giemsa staining. Demographic, clinical, intraoperative and postoperative data were recorded and analysed using SPSS; the chi-square test assessed associations ($p<0.05$ significant).

Results: H. pylori were detected in 45/70 patients (64.3%). Duodenal perforations predominated (47/70; 67.1%) and showed significantly higher positivity than gastric perforations (76.6% vs 39.1%; p=0.002). NSAID use was marginally associated with infection (67.5% vs 60.0%; p=0.042). Age, sex, smoking, alcohol, comorbidities, symptom profile, WBC count, perforation size and postoperative outcomes showed no significant association with H. pylori status. Overall complications occurred in 42.9%, mortality in 15.7%, and mean hospital stay was 9.47 ± 3.79 days.

Conclusion: H. pylori is a key etiological factor in gastro-duodenal perforation, particularly duodenal, supporting routine intraoperative testing and eradication therapy. However, immediate postoperative outcomes are driven mainly by surgical timing and patient status rather than infection status.

Keywords: Helicobacter pylori; Gastro-duodenal perforation; Peptic ulcer disease; Rapid urease test; Duodenal ulcer; Eradication therapy.

INTRODUCTION

Gastro-duodenal perforation is a surgical emergency requiring swift intervention due to its high morbidity and mortality. It occurs when a full-thickness defect in the gastro-duodenal wall allows luminal contents to leak into the peritoneal cavity, causing chemical and bacterial peritonitis. Despite advances in surgical techniques, antimicrobial therapy and critical care, its management remains complex. Peptic ulcer disease (PUD), a common cause of gastro-duodenal perforation, is often linked to Helicobacter pylori (H. pylori) infection.

PUD affects 5–10% of the global population, with regional variations influenced by socioeconomic status, hygiene, healthcare access and H. pylori prevalence. While its incidence has fallen in developed countries owing to improved medical management, it remains significant in developing nations where H. pylori are still prevalent. The lifetime risk of PUD is around 10%, with duodenal ulcers more common than gastric ulcers. Complications such as bleeding and perforation pose major risks, with 2–10% of PUD patients experiencing perforation, and mortality is high—particularly in delayed presentations or patients with comorbidities.

Peptic ulcer perforation results from a combination of environmental, microbial and host factors. H. pylori is the most significant contributor, found in about 78% of patients with perforated PUD. It causes chronic gastritis, damages the mucosal barrier and increases gastric acid secretion, leading to ulceration and perforation, while also triggering an acute inflammatory response that weakens mucosal defences. Additional risk factors include smoking, alcohol and NSAID use, all of which impair mucosal integrity and delay ulcer healing.

Perforation presents with sudden severe epigastric pain, abdominal rigidity and signs of peritoneal irritation. Diagnosis is largely clinical, supported by imaging (erect abdominal X-ray showing free air; CT for detailed visualisation) and laboratory findings of leucocytosis, metabolic acidosis and raised inflammatory markers. Surgical treatment—usually primary closure with an omental (Graham's) patch—is preferred for small perforations, while larger or recurrent ulcers may require definitive procedures; laparoscopic approaches are increasingly common. Postoperative care includes proton pump inhibitors, H. pylori eradication and lifestyle modification to prevent recurrence.

Despite aggressive management, mortality ranges from 5–25%, with worse outcomes in the elderly, those with severe comorbidities and delayed presentation. H. pylori eradication is proven to prevent recurrent ulcers, but its role in preventing perforation requires further study. Given the pathogen's high prevalence in perforation cases, screening and treatment should be prioritised in at-risk populations, though growing antibiotic resistance is a challenge. This study was undertaken to determine the prevalence of H. pylori infection in gastro-duodenal perforation and to analyse the outcome and the need for eradication therapy.

AIM AND OBJECTIVES

Aim: To determine the prevalence of H. pylori infection in gastro-duodenal perforation and to analyse the outcome and the need for H. pylori eradication therapy.

Objectives: (1) To study the site of ulcer and perforation; (2) to identify the prevalence of H. pylori infection.

MATERIALS AND METHODS

Study design and setting: A prospective observational study conducted in the Department of General Surgery, Government Thiruvarur Medical College and Hospital (GTMCH), Thiruvarur, among patients presenting with acute abdomen and suspected perforation who

were evaluated and managed surgically.

Study duration: One year, with consecutive enrolment of eligible patients.

Sample size and sampling: A total of 70 patients diagnosed with gastro-duodenal perforation and undergoing exploratory laparotomy were included, selected by simple random sampling from eligible admissions.

Inclusion criteria: Patients aged 18–75 years, of both sexes, diagnosed with gastro-duodenal perforation and undergoing exploratory laparotomy.

Exclusion criteria:

- Age less than 18 or more than 75 years
- Malignant perforation
- Traumatic perforation
- Perforations at sites other than the gastro-duodenal region

Study procedure: All patients underwent thorough clinical evaluation, imaging and preoperative stabilisation, followed by exploratory laparotomy under general anaesthesia. The perforation site was identified and repaired, usually with a Graham's omental patch. Two mucosal biopsies were collected from the margin of the perforation: the first was placed in a preformed detection kit for the rapid urease test (RUT), which indicates urease-producing bacteria by a colour change; the second was fixed in 10% formalin and sent for Giemsa staining to allow histological identification of H. pylori.

Study parameters: Demographic details (age, sex, smoking, alcohol and NSAID use); clinical presentation (symptoms, duration of illness); surgical findings (site and size of perforation, contamination); H. pylori status by RUT and Giemsa staining; and postoperative outcomes (complications, hospital stay, mortality).

Statistical analysis: Data were entered in Microsoft Excel and analysed using SPSS. Categorical variables (gender, smoking, alcohol, NSAID use, H. pylori positivity) were expressed as percentages, and continuous variables (age, perforation size, hospital stay) as mean ± standard deviation. The chi-square test assessed the association between perforation and H. pylori infection; a p-value <0.05 was considered statistically significant. The study was approved by the Institutional Ethics Committee of GTMCH, and informed consent was obtained from all participants.

RESULTS

Among the 70 patients, age ranged from 25 to 75 years (mean 51.26 ± 13.96), with a mid-adult predominance. The cohort was balanced by sex (37 males, 52.9%; 33 females, 47.1%). Smoking, alcohol and NSAID use were each present in 40/70 patients (57.1%). Comorbidities were common, most frequently diabetes mellitus alone (28.6%) and hypertension alone (20.0%), while 27.1% had none.

TABLE 1: BASELINE DEMOGRAPHIC AND CLINICAL CHARACTERISTICS (N=70).

Characteristic	Category	n (%)
Age (years)	Mean ± SD	51.26 ± 13.96
Sex	Male	37 (52.9)
	Female	33 (47.1)
Smoking	Yes	40 (57.1)
Alcohol use	Yes	40 (57.1)
NSAID use	Yes	40 (57.1)
Comorbidity	DM alone	20 (28.6)
	HTN alone	14 (20.0)
	HTN + DM	6 (8.6)
	HTN + IHD	5 (7.1)

	IHD alone	6 (8.6)
	None	19 (27.1)

Duodenal perforations dominated (47/70; 67.1%) over gastric (23/70; 32.9%). Rapid urease test with Giemsa staining detected H. pylori in 45/70 patients (64.3%). All 70 biopsies underwent dual testing. All H. pylori–positive patients received surgery plus eradication therapy, while all negative patients had surgery alone. Postoperative complications occurred in 30/70 (42.9%), mortality in 11/70 (15.7%), and 29/70 (41.4%) recovered uneventfully. Mean postoperative stay was 9.47 ± 3.79 days and mean symptom duration before presentation was 27.90 ± 13.20 hours
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TABLE 2: PERFORATION, INFECTION AND OUTCOME PROFILE (N=70).

Parameter	Category	n (%)
Site of perforation	Duodenal	47 (67.1)
	Gastric	23 (32.9)
H. pylori status	Positive	45 (64.3)
	Negative	25 (35.7)
Treatment	Surgery + eradication	45 (64.3)
	Surgery only	25 (35.7)
Outcome	Complications	30 (42.9)
	Recovered	29 (41.4)
	Mortality	11 (15.7)

Association of H. pylori status with clinical variables. On bivariate analysis, only the site of perforation and NSAID use were significantly associated with H. pylori positivity. H. pylori was found in 76.6% of duodenal versus 39.1% of gastric perforations (p=0.002), and in 67.5% of NSAID users versus 60.0% of non-users (p=0.042). Age, sex, smoking, alcohol, comorbidities, symptom complex, symptom duration and length of hospital stay showed no significant association
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TABLE 3: ASSOCIATION OF H. PYLORI STATUS WITH CLINICAL VARIABLES.

Variable	H. pylori positivity (%)	P value
Age across decades	44.4–80.0	0.370
Sex (female vs male)	66.7 vs 62.2	0.695
Smoking (yes vs no)	62.5 vs 66.7	0.719
Alcohol (yes vs no)	65.0 vs 63.3	0.885
NSAID use (yes vs no)	67.5 vs 60.0	0.042
Comorbidities	42.9–80.0	0.496
Symptom complex	40.0–66.7	0.431
Symptom duration	55.6–73.3	0.632
Site (duodenal vs gastric)	76.6 vs 39.1	0.002
Postoperative stay	60.7–100	0.859
Outcome (complications, +ve vs –ve)	46.7 vs 36.0	0.659

Continuous variables by H. pylori status. Mean WBC count (12.36 vs 12.31 $\times 10^9$/L;

p=0.940), perforation size (1.06 vs 0.85 cm; p=0.255), symptom duration (28.4 vs 27.1 h; p=0.701) and postoperative stay (9.56 vs 9.32 days; p=0.805) did not differ significantly between H. pylori–positive and –negative groups, indicating that acute inflammatory markers, defect severity and recovery time are independent of infection status.

DISCUSSION

This prospective study investigated the prevalence and clinical implications of H. pylori infection in 70 patients with gastro-duodenal perforation, using uniform intraoperative rapid urease testing and Giemsa staining. The overall prevalence of 64.3%—rising to 76.6% in duodenal perforations—underscores the bacterium's central etiological role and supports standardised perioperative testing and eradication to reduce recurrence risk. Importantly, H. pylori status did not impair short-term outcomes such as complication rates or hospital stay, attesting to the safe inclusion of eradication regimens without impairing recovery.

The mean age (51.26 ± 13.96 years) reflects a mid-adult predominance. This is younger than some series and older than others: Reinbach et al. reported a mean of 42 years with 47% positivity and no age association, whereas Sebastian et al. found a mean of 48.5 years with 82.8% positivity, and Alam et al. (2023) described first-time duodenal perforations at a mean of 52.3 years. In our cohort, positivity by age bracket ranged from 44.4% to 80.0% (p=0.370), indicating no significant variation across decades. Perforation is thus most common in mid-adulthood but can occur at any adult age, and age alone should not influence the decision to test or treat.

The near gender parity (52.9% male) contrasts with the classical male predominance in PUD, but our balanced distribution and the absence of a sex-based difference in positivity (66.7% female vs 62.2% male; p=0.695) mirror the findings of Gisbert et al. and Reinbach et al. Similarly, smoking (62.5% vs 66.7%; p=0.719) and alcohol use (65.0% vs 63.3%; p=0.885) showed no significant association with infection, consistent with Dogra et al. and others. While these lifestyle factors are relevant to ulcer formation, they do not appear to predict bacterial colonisation in the perforation setting.

NSAID use showed a marginally significant association with H. pylori positivity (67.5% vs 60.0%; p=0.042), suggesting a possible synergistic ulcerogenic effect when NSAID-induced mucosal injury coexists with infection. Gisbert et al. similarly identified NSAID exposure as a primary perforation risk factor, with H. pylori prevalence rising when NSAID users were excluded. Mechanistically, NSAIDs impair prostaglandin synthesis and disrupt the mucosal barrier, potentiating colonisation. Given the marginal p-value, larger studies are warranted, but the finding reinforces the need for gastroprotection and H. pylori testing in patients requiring NSAID therapy.

The strongest association was with perforation site: H. pylori was found in 76.6% of duodenal versus 39.1% of gastric perforations (p=0.002). This aligns with the bacterium's predilection for the duodenal bulb, as reported by Tokunaga et al. and Dogra et al., and motivates targeted testing and eradication specifically in duodenal perforation cases. By contrast, comorbidities, symptom complexes, symptom duration, WBC count and perforation size were all independent of infection status, indicating that the acuity and severity of presentation are driven primarily by peritoneal contamination and host inflammatory response rather than H. pylori alone.

Postoperative complications (42.9%),

mortality (15.7%) and mean hospital stay (9.47 ± 3.79 days) were comparable to other series (e.g. Dogra et al.: 40% morbidity, 12% mortality; Tokunaga et al.: 14% mortality) and did not differ significantly by H. pylori status. Our dual intraoperative biopsy approach avoids false negatives from preoperative acid suppression and provides immediate guidance for eradication therapy. Collectively, these findings indicate that prompt surgical intervention and standardised perioperative care drive short-term outcomes regardless of infection, while long-term management should focus on eradication to prevent recurrence.

STRENGTHS AND LIMITATIONS

The study's strengths lie in its prospective design, consecutive enrolment of 70 cases with minimal selection bias, and high diagnostic consistency through uniform perioperative testing across a broad age range and near-equal sex distribution. As a single-centre study, generalisability may be limited, and the sample—while adequate for descriptive analysis—may be underpowered to detect subtle interactions. Long-term follow-up was not performed; future studies incorporating molecular diagnostics, strain and resistance profiling, randomised comparison of eradication regimens, and health-economic evaluation of universal testing would further refine management.

CONCLUSION

This analysis of 70 consecutive gastro-duodenal perforation cases identifies H. pylori as a key etiological factor, with an overall prevalence of 64.3% and significantly higher positivity in duodenal perforations (76.6%). Once perforation occurs, however, patient demographics, lifestyle factors, comorbidities and postoperative recovery are largely independent of bacterial status. Duodenal site (p=0.002) and NSAID use (p=0.042) were the only variables significantly associated with infection, highlighting priorities for intervention: routine testing in duodenal perforations and gastroprotection for NSAID users. Intraoperative biopsy with rapid urease testing enables timely identification and eradication without delaying surgery or recovery. A dual approach of prompt surgical repair and systematic H. pylori eradication is recommended to prevent recurrence and reduce the long-term burden of peptic ulcer complications.

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