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Research Article

To Study The Prevalence Of Helicobacter Pylori In Patients With Gastric Cancer Attending A Tertiary Care Centre: A Cross Sectional Study

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ABSTRACT

Abstract

Background: Gastric adenocarcinoma is a major global health threat. Gastric cancer remains a major cause of cancer-related mortality worldwide, with *Helicobacter pylori* (H. pylori) infection recognized as a key etiological factor. Regional prevalence data are essential to guide preventive and therapeutic strategies.

Aim and Objectives: To determine the prevalence of *H. pylori* infection among patients with histopathologically confirmed gastric carcinoma.

Material and Methods: This cross-sectional study included 112 consecutive patients with gastric adenocarcinoma confirmed by histopathology. Clinical details were recorded, and multiple biopsy specimens were obtained during endoscopy or surgery. *H. pylori* detection was carried out using rapid urease test (RUT) and histopathology with hematoxylin–eosin and modified Giemsa staining. Patients were considered positive if either test was positive.

Results: The mean age of patients was 51.2 years (range: 18–80), with a male predominance (60.7%). The peak incidence occurred in the 41–60 years age group. Distal gastric adenocarcinoma was the most common site (69.6%), followed by proximal (13.4%), body (8.9%), and diffuse carcinoma (8%). The prevalence of *H. pylori* was 43.8% (49/112). Site-specific analysis showed the highest prevalence in distal cancers (46.1%) and diffuse type (55.6%).

Conclusion: Nearly half of gastric carcinoma patients in this cohort were infected with *H. pylori*, highlighting its significant role in gastric carcinogenesis. These findings support early detection and eradication of *H. pylori* as a public health priority in high-risk populations.

Keywords*:Prevalence, H. Pylori, Infection, Histopathological, Gastric Carcinoma.*

INTRODUCTION

Gastric cancer is a major global health burden and remains one of the leading causes of cancer-related mortality worldwide. According to GLOBOCAN 2020 data, gastric cancer ranks as the fifth most common malignancy and the fourth leading cause of cancer death, accounting for more than one million new cases and approximately 769,000 deaths annually【1】. The disease shows marked geographical variation, with the highest prevalence in East Asia, Eastern Europe, and parts of South America, while relatively lower rates are seen in North America and Africa【2】. In India,

gastric cancer is a significant health concern, particularly in the southern and northeastern regions, where lifestyle, dietary, and environmental factors contribute to its increased incidence【3,4】.

This is one of the common causes of deaths and the etiological factors include salty food, low fibre diet, smoking, alcohol, previous history of gastric surgery, elderly high BMI people, low socio-economic class etc. More incidence of gastric cancers are seen with HNPCC, pernicious anaemia, FAP, Cowden syndrome, is seen with gastric cancers that happens in families.

Among the numerous risk factors for gastric cancer, infection with Helicobacter pylori has been identified as the most important and well-established etiological agent. H. pylori is a Gram-negative, spiral-shaped, microaerophilic bacterium that colonizes the gastric mucosa and induces chronic gastritis, which can progress through a cascade of precancerous lesions, including atrophic gastritis, intestinal metaplasia, dysplasia, and ultimately gastric adenocarcinoma【5,6】. The International Agency for Research on Cancer (IARC) has classified H. pylori as a Group I carcinogen, highlighting its strong causal relationship with non-cardia gastric cancer【7】.

Epidemiological studies suggest that approximately 50% of the world's population is infected with H. pylori, though only a fraction develops clinical disease【8】. Host genetic susceptibility, bacterial virulence factors such as cytotoxin-associated gene A (CagA) and vacuolatingcytotoxin A (VacA), and environmental influences including diet and smoking collectively determine disease progression【9,10】. In particular, CagA-positive strains are strongly associated with severe gastritis and a higher risk of carcinogenesis【11】.

The prevalence of H. pylori in gastric cancer varies widely across regions. A large international meta-analysis reported that nearly 60% of gastric adenocarcinomas are attributable to H. pylori infection【12】. In India, studies from Banaras, Chennai, and Kerala have demonstrated significant association between H. pylori infection and gastric malignancy, although regional differences in prevalence exist【13,14】. Despite improved sanitation and antibiotic use leading to a gradual decline in H. pylori infection rates in many developed countries, the burden remains substantial in developing regions【15】.

Understanding the prevalence and association of H. pylori in gastric cancer in specific populations is crucial, as it provides insights into local epidemiology and helps guide preventive strategies. Eradication of H. pylori infection has been shown to reduce the risk of gastric cancer, particularly when instituted before the development of precancerous changes【16】.

This study was undertaken to determine the prevalence of H. pylori infection in patients with gastric carcinoma attending a tertiary referral unit, thereby contributing that can aid in prevention and management strategies.

MATERIAL AND METHODS

This was a prospective cross-sectional study conducted in the Department of General Surgery and Department of Radiology with collaboration with Department of Microbiology, at a Tertiary care hospital for a period of 12 months i.e, April 2024 to April 2025.

Study Population

All consecutive patients suspected to have carcinoma stomach and undergoing diagnostic endoscopy or open surgery during the study period were screened for eligibility. A total of 112 patients who consented to participate and fulfilled the inclusion criteria were enrolled in the study.

Inclusion Criteria

1. Patients of either sex aged 18 years and above.
2. Patients suspected or diagnosed with carcinoma stomach on endoscopy.
3. Patients with histopathological confirmation of gastric adenocarcinoma.
4. Patients willing to provide informed consent for participation.

Exclusion Criteria

1. Patients with a history of neoadjuvant chemotherapy or radiotherapy, as these treatments may alter H. pylori colonization.
2. Patients with previous gastric surgery.
3. Patients with severe comorbidities or terminal illness not suitable for endoscopy/biopsy.

4. Patients who refused consent.

Data Collection
After obtaining informed consent, demographic details (age, sex, socioeconomic status) and clinical history (duration of symptoms, risk factors, dietary habits, smoking, alcohol use) were recorded. A thorough clinical examination was performed in all cases.

Diagnostic Evaluation
Endoscopy and Biopsy: All patients underwent upper gastrointestinal endoscopy. Multiple punch biopsy specimens were obtained from tumour margins and suspicious areas. In cases undergoing open surgery, intraoperative biopsies were collected.

Histopathology: Tissue samples were fixed in 10% formalin and processed for histopathological examination to confirm the diagnosis of gastric adenocarcinoma.
Detection of *H. pylori*: Presence of H. pylori was assessed using Rapid Urease Test (RUT) and confirmed by histopathological evaluation (Hematoxylin and Eosin and modified Giemsa staining). Patients were considered positive if either test demonstrated H. pylori.

Data Analysis
All collected data were entered into Microsoft Excel and analysed using descriptive statistics. Categorical variables were expressed as frequencies and percentages, while continuous variables such as age were expressed as mean, median, and range. The prevalence of H. pylori in gastric carcinoma was calculated as a proportion of total cases. Site-specific and age-wise associations of H. pylori were also analysed.

RESULTS
In the present study a total of 112 patients were diagnosed with carcinoma stomach on endoscopy and were sequentially included in the study. The mean age of presentation was 51.2 years, with a median of 54 years and a range between 18 and 80 years. The maximum number of cases was observed in the 41–60 years age group, which accounted for nearly half of the cases (46.4%). Patients younger than 40 years comprised 27.7% of the cohort, whereas those older than 40 years constituted 72.3%.

A male preponderance was noted, with 68 cases (60.7%) being males and 44 cases (39.3%) females, giving a male-to-female ratio of approximately 1.5:1.

Regarding the anatomical site of the tumour, distal gastric adenocarcinoma was the most common, accounting for 78 cases (69.6%). This was followed by proximal gastric carcinoma in 15 cases (13.4%), carcinoma involving the body of the stomach in 10 cases (8.9%), and diffuse gastric carcinoma in 9 cases (8%).

The prevalence of Helicobacter pylori infection in gastric carcinoma was found to be 43.8%. Out of 112 cases, H. pylori was detected in 49 patients, while 63 patients (56.2%) showed no evidence of the organism. On site-specific analysis, H. pylori positivity was observed in 36 of 78 cases of distal gastric adenocarcinoma, 3 of 10 cases involving the body of the stomach, 5 of 15 cases of proximal gastric carcinoma, and 5 of 9 cases of diffuse gastric carcinoma. Thus, although distal adenocarcinoma constituted the majority of tumours, a relatively higher proportion of H. pylori association was also noted in diffuse gastric cancers.

Age Distribution
The mean age of presentation was 51.2 years (median: 54, mode: 54, range: 18–80 years). The maximum number of gastric carcinoma cases occurred between 41–60 years.

Table 1. Age Distribution of Gastric Carcinoma Patients (n=112)

Age Group (years)	Frequency	Percentage
1–20	5	4.5%
21–40	26	23.2%
41–60	52	46.4%

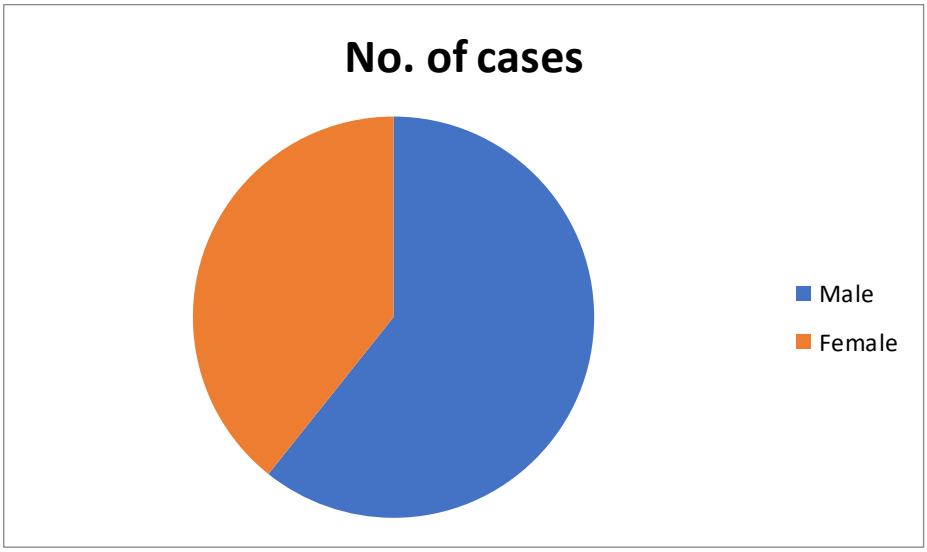
61–80	29	25.9%
Total	112	100%

Below 40 years: 27.7%

Above 40 years: 72.3%

Table 2. Gender Distribution of Gastric Carcinoma Patients (n=112)

Gender	Frequency	Percentage
Male	68	60.7%
Female	44	39.3%
Total	112	100%



Graph No. 1: Graphical Representation of Genderwise distribution of the cases

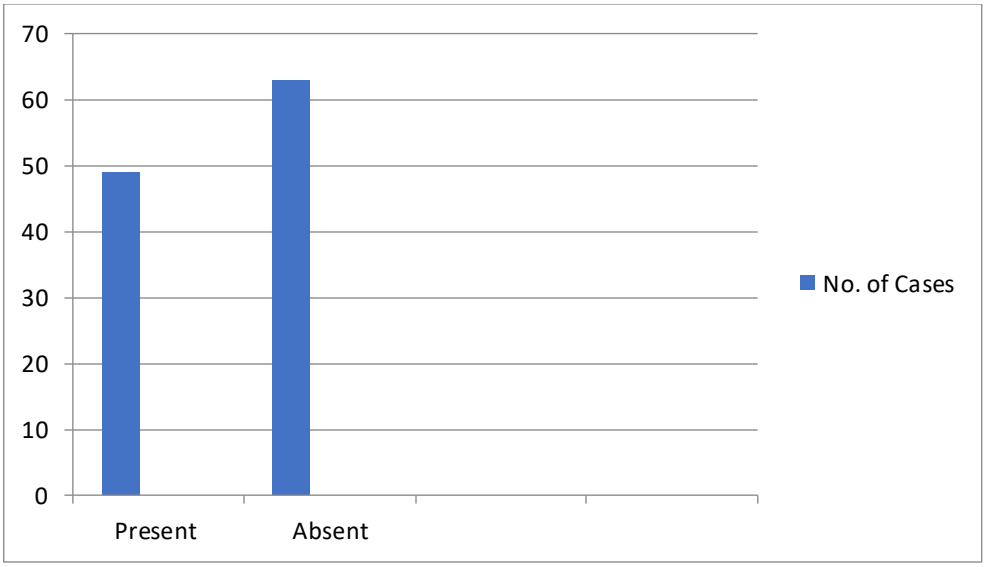
Table 3. Site-wise Distribution of Gastric Carcinoma (n=112)

Site	Frequency	Percentage
Distal Adenocarcinoma	78	69.6%
Body of Stomach	10	8.9%
Proximal Gastric CA	15	13.4%
Diffuse Gastric CA	9	8.0%
Total	112	100%

Prevalence of Helicobacter pylori
On analysis, H. pylori was detected in 49 of 112 cases.
Prevalence: 43.8%
Absent in 56.2%

Table 4. Prevalence of H. pylori in Gastric Carcinoma (n=112)

H. pylori	Frequency	Percentage
Present	49	43.8%
Absent	63	56.2%
Total	112	100%



Graph No. 2: Graphical Representation of cases

Association of H. pylori with Site of Tumour

Table 5. Distribution of H. pylori According to Tumour Site (n=112)

Site	Total Cases	H. pylori Positive
Distal Adenocarcinoma	78	36
Body of Stomach	10	3
Proximal Gastric CA	15	5
Diffuse Gastric CA	9	5
Total	112	49

DISCUSSION

In this study cohort of 112 patients with endoscopically confirmed gastric carcinoma, *Helicobacterpylori* was detected in 49 cases, giving a prevalence of 43.8%. This prevalence is comparable to previous Indian studies which reported H. pylori positivity in 40–50% of gastric carcinoma cases【13,14】, but slightly lower than international meta-analyses that suggest approximately 60% of gastric adenocarcinomas are attributable to H. pylori infection【12】. Variability in prevalence can be explained by geographical differences, diagnostic methods used, and prior treatment exposure.

The age and site distribution in our study peak the incidence in the 41–60 years age group and predominance of distal gastric adenocarcinoma — mirrors the classical Correa cascade of carcinogenesis from chronic gastritis to adenocarcinoma【5】. Distal tumours constituted nearly 70% of cases, and most H. pylori–positive tumours were also distal. This supports prior epidemiological data which demonstrate that long-standing infection drives intestinal-type gastric cancer, particularly in the distal stomach【6,12】.

Bacterial virulence factors contribute significantly to oncogenesis. The cytotoxin-associated gene A (CagA) protein alters intracellular signalling, while the vacuolatingcytotoxin A (VacA) induces immune evasion and epithelial injury. CagA-positive strains are consistently associated with more severe gastritis and higher cancer risk【9–11】. These findings explain why not all infected individuals develop carcinoma and why strain-specific differences exist between populations【10】.

Diagnostic limitations must also be considered. In our study, H. pylori was detected using the rapid urease test (RUT) and histopathology. Although both methods are widely available, their sensitivity can be reduced in patients with advanced atrophy, intestinal metaplasia, or those on proton pump inhibitors【8】. Studies have shown that combining histology with additional stains or molecular assays increases detection yield【15,16】. Therefore, our prevalence may represent an underestimation of prior exposure.

From a preventive perspective, evidence from randomized controlled trials and meta-analyses has shown that eradication of H. pylori significantly reduces gastric cancer risk, particularly when administered before the onset of premalignant changes【7,12,16】. A landmark Chinese trial demonstrated that eradication therapy reduced gastric cancer incidence in high-risk populations【16】. More recent systematic reviews confirm this benefit, although the absolute risk reduction varies by baseline prevalence and antibiotic resistance【15】.

Our study has several limitations. First, sampling was performed mainly on tumour or peri-tumour tissue, which may underestimate H. pylori colonization due to loss of mucosal niches in advanced disease. Second, strain typing (CagA, VacA, and other virulence markers) was not undertaken, which could have provided deeper mechanistic insights【9,10】. Third, the cross-sectional design limits causal inference. Finally, the study was single-centre with a modest sample size, and hence, larger multicentre studies are needed for generalizability.

In summary, the prevalence of H. pylori in gastric carcinoma patients in our cohort was 43.8%, reinforcing its significant role in gastric carcinogenesis. Early identification and eradication of H. pylori remain vital public health strategies to reduce the burden of gastric cancer, particularly in high-incidence regions like Kerala. In our study, H. pylori detection relied on rapid urease test (RUT) and histopathology, both of which are widely available but can underestimate prevalence. Advanced atrophy and intestinal metaplasia may reduce bacterial colonization, while prior proton pump inhibitor use may suppress growth. Studies have shown that combining RUT, histology with special stains, and molecular assays significantly improves sensitivity【8,15,16】. Hence, the prevalence reported here may underestimate true past exposure.

Prevention remains a cornerstone of gastric cancer control. Multiple randomized controlled trials and meta-analyses have confirmed that eradication of H. pylori significantly reduces the incidence of gastric carcinoma. A 2025 meta-analysis demonstrated a 36% reduction in gastric cancer risk (RR 0.64, 95% CI 0.48–0.84) following eradication therapy【1】. Another 2025 study confirmed that eradication reduced progression of intestinal metaplasia and improved gastric mucosal outcomes【17】. Furthermore, a large cluster-randomized trial in China (2025) showed that community-based eradication not only decreased cancer incidence but also proved more cost-effective than repeated endoscopic surveillance【19】.

Global burden estimates also highlight the importance of eradication: modeling studies predict that nearly 76% of gastric cancers arising in cohorts born between 2008–2017 could be prevented if H. pylori eradication strategies are widely implemented【20】. This supports the implementation of "screen-and-treat" policies in high-incidence populations and "test-and-treat" approaches in moderate-risk groups.

CONCLUSION

Our study demonstrates that H. pylori infection was present in 43.8% of gastric carcinoma patients. This underscores the significant contribution of the bacterium to gastric carcinogenesis in this region. When considered alongside recent global evidence, our findings reinforce the need for early detection and eradication of H. pylori as a feasible public health strategy to reduce gastric cancer incidence. Population-based eradication, particularly in high-risk groups, has the potential to substantially lower future cancer burden.

Limitations of the Study

1. Sampling bias – biopsies were taken from tumor/peri-tumor tissue, where bacterial colonization may be diminished, possibly underestimating prevalence.
2. Diagnostic constraints – only RUT and histology were used; more sensitive molecular methods were not applied.
3. Virulence markers – no testing for CagA or VacA status was performed, limiting mechanistic insights.
4. Study design – the cross-sectional, single-center design precludes causal inference and limits generalizability.
5. Sample size – though larger than earlier reports, the sample (n=112) is still modest compared to population-based cohorts.

DECLARATIONS:

Conflicts of interest: There is no any conflict of interest associated with this study

Consent to participate: There is consent to participate**.**

Consent for publication: There is consent for the publication of this paper.

Authors contributions: Author equally contributed the work.

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