# **Original Research Article**

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# Prevalence of *Helicobacter pylori* in patients with peptic ulcer disease

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## **ABSTRACT**

**Background:** Prevalence of *Helicobacter pylori* (*H. pylori*) emerges throughout the world and instigates peptic ulcer disease (PUD). The study was conducted with the aim to determine the prevalence of *H. pylori* in patients with PUD undergoing upper gastrointestinal endoscopy.

**Methods:** This prospective study was conducted on 150 cases of PUD from August 2009 to February 2011. Endoscopy was done in all cases. Biopsy was done and sent for histopathological examination and rapid urease test for confirmation of presence of *H. pylori*.

**Results:** Out of 150 patients with mean age of 45.76 years, 109 patients were diagnosed to have been infected with Helicobacter pylori (72.66%). Out of 89 patients with gastric ulcer, 61 patients were infected with Helicobacter pylori (68.53%). Forty two out of 51 patients (82.35%) with duodenal ulcers and 06 of 10 patients (60%) with carcinoma of storach were positive for *H. pylori*. The remaining patients were found to be negative for the *H. pylori* infection.

Conclusions: The findings of the study conclude that *H. pylori* was consistently associated with PUD.

Keywords: Helicobacter pylori, Giemsa stain, PUD, Urease test

# INTRODUCTION

The incidence of peptic ulcer disease (PUD) is majorly due to *Helicobacter pylori* infection. Many studies demonstrated that the prevalence of *H. pylori* is very high i.e., approximately 50% of the world's population is diseased with the organism.<sup>1,2</sup> The major risk factors associated with *H. pylori* infection was poor socioeconomic status, crowded living conditions, smoking, higher number of siblings and a lower consumption of fruits.<sup>3-5</sup>

Previous studies reveal stronger association between the gastric colonization of *H. pylori* and chronic gastritis and PUDs.<sup>6,7</sup> Its presence can be easily diagnosed by rapid urease test and histopathology at the time of upper gastrointestinal endoscopy. Rapid urease test is highly specific only when the bacteria density is high otherwise it produces false negative results. H&E stain can directly

identify *H. pylori* and its sensitivity and specificity has been reported as 69-93% and 87-90%, respectively. However, the specificity can be improved to 90-100% by using special stains such as modified Giemsa stain.<sup>8,9</sup>

The present study was done with the objective to study the prevalence of *H. pylori* in patients with PUD undergoing upper gastrointestinal endoscopy.

### **METHODS**

This was a prospective study conducted on 150 cases of PUD over a period of one and half years from August 2009 to February 2011 at KVG Medical College, Sullia, Karnataka. After getting approval from institutional ethics committee, patients of age between 18-70 years, patients having symptoms of PUD like upper abdominal pain, weight loss, poor appetite, nausea, vomiting, epigastric tenderness, hematemesis, malena, etc. and

patients having gastric/duodenal ulcers and ulceroproliferative growth on endoscopy were included in the study. Exclusion criteria were patients below 18 years and above 70 years of age, patients on NSAID's for more than one-month duration, patients who have received Anti-H. pylori treatment, patients with oesophageal growths on endoscopy and who are unwilling or unfit patients for gastroscopy.

After meeting the requirements of inclusion criteria, all the patients underwent upper gastro-intestinal endoscopy. According to the endoscopy findings the patients were divided into following groups. a. Duodenal ulcer, b. Gastric ulcer, and c. Carcinoma of stomach.

## Study procedure

All the patients in this study group, both in-patient as well as outpatient underwent upper gastro-intestinal endoscopy under topical aneasthesia. The patients were asked to fast for 12 hours prior to the procedure. The cases admitted with gastric outlet obstruction were given stomach wash the night before and the morning of the day on which the procedure was scheduled. Only a few patients were gives 5-10 mg diazepam intravenously for sedation depending on the preference of the consultants.

Oral lignocaine spray was used 5-10 minutes before the procedure for the local anaesthetic effect. The upper gastro-intestinal endoscopy was conducted with Pentax 29 P, flexible, fibreoptic endoscope with patients in left lateral positions.

On entering the oesophagus, any lesions/growths are the oesophagus were looked for. Cases of oesophageal carcinoma were excluded from the study as per the exclusion criteria. On entering the stomach, presence of any ulcers or growths was looked for.

Four endoscopic biopsies were taken. Two each from the gastric antrum and the edge of the ulcer crater depending on the findings. The biopsies from the antrum were randomly taken. Two biopsy specimens, one of the antral area and the other of the pathological finding were immediately inoculated into freshly prepared urea broth containing phenol red as the indicator. Positive test for *H. pylori* was indicated by change in colour of the medium from yellow to pink or red. The test was read as strongly positive when the change in colour occurred within 5-15 minutes following inoculation and weakly positive when the colour change occurred in first 6 hours. Any colour change in between was read as intermediate.

The other two biopsy specimens were sent in formalin solution for histopathology and special staining. Each of the biopsy specimens were fixed in 10% buffered formalin, routinely processed to paraffin and 3 um sections cut. One section of each biopsy specimen was routinely stained with haematoxylin and eosin (H&E) stain and examined microscopically for presence of *H*.

pylori organisms. The other sections of the biopsy specimen were dewaxed and taken to water and then incubated in 2% Giemsa solution in D-water for 30 minutes at room temperature. After rinsing in tap water, the sections are quickly dehydrated through ethanol solution before being cleared with xylene and examined for the presence of *H. pylori*.

The case was taken as *H. pylori* positive when the rapid urease test and/ or histopathological examination was positive.

#### **RESULTS**

Table 1 represents the demographic characteristics of the patients. Out of 150 patients there were 102 male patients and 48 female patients, age ranging from 28 years to 70 years (Mean-45-76 years). On endoscopy, it was noticed that 89 patients had gastric ulcer, 51 had duodenal ulcer and 10 had proliferative growth in the stomach on endoscopy.

**Table 1: Characteristics of the patients.** 

Characteristics	Gastric ulcer	Duodenal ulcer	Carcinoma stomach
No. of patients	89	51	10
Men/women	58/31	35/16	5/5
Mean age	42.76	42.82	60.2 (18-70)
(Range)	(18-70)	(18-70)	00.2 (18-70)

Table 2: Prevalence of *H. pylori* in various clinical presentations of PUD.

Clinical presentation	Number of cases	H. pylori positive	Percentage
Abdominal pain	150	109	72.66
Nausea/vomiting	37	24	64.86
Hematemesis	04	01	25
Malena	03	03	100
Loss of appetite/weight	50	34	68
Anemia	42	29	69.04
Epigastric mass	06	03	50
Epigastric tenderness	32	26	81.25

The signs and symptoms of PUD in patients are detailed in Table 2. Thirty-seven patients presented with nausea or vomiting out of which 24 had Helicobacter pylori infection. 04 patients had haematemesis, out of which 01 patients were positive for *H. pylori* infection. 03 patients had malena out of which 03 turned out to be *H. pylori* positive. 50 patients also had loss or weight or appetite on presentation. Of them, 34 patients were positive for *H. pylori*. On examination of these patients, 42 patients were anaemic out of whom 29 patients were positive for *H. pylori*. Of the total 150 patients, 32 patients had epigastric tenderness and 06 patients had an epigastric

mass on palpation. Of these 32 patients, 26 patients were tested positive for *H. pylori* and of those 06 patients, who had an epigastric mass, 03 patients were positive for *H. pylori*.

The most commonly affected age group with signs and symptoms of PUD was 31-40 years. Of them 29 were diagnosed with *H. pylori* infection. Males are commonly affected at the rate of 79-81%. On endoscopy most of the patients were identified with gastric ulcer (n=89). Of them 61 cases were *H. pylori* positive.

Table 3: Prevalence of *H. pylori* according to characteristics of the patients.

Characteristics	Total cases (n=150)	H. pylori positive (n=109)			
Age group		N	Percentage (%)		
18-30	24	17	15.59		
31-40	41	29	26.60		
41-50	31	23	21.10		
51-60	30	21	19.26		
61-70	24	19	17.43		
Sex					
Males	98	87	79.81		
Females	52	22	20.18		
Endoscopic findings					
Gastric ulcer	89	61	55.96		
Duodenal ulcer	51	42	38.53		
Carcinoma of stomach	10	06	5.50		

Out of the 109 patients who were *H. pylori* positive, 3 cases were positive for the rapid urease test and 106 cases were *H. pylori* positive by histopathology. Of them, 100 cases were positive for both H&E and Giemsa staining and 6 cases were positive on Giemsa staining (Figure 1).

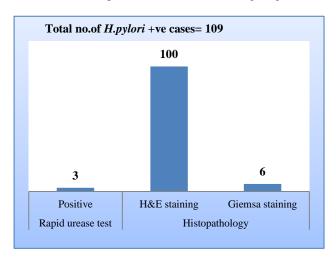


Figure 1: Detection of *H. pylori* according to different diagnostic procedures.

#### DISCUSSION

*H. pylori* are a spiral microorganism noted on the gastric mucosa and was first cultured by Marshall and Warren. <sup>10</sup> They identified a significant association between the presence of *H. pylori* on the gastric mucosa and antral gastritis. The present study was done to explore the possibility of this association between *H. pylori* and PUD.

The present study confirms that the prevalence rate of H. pylori was more in males (79.81%) compared to females (20.18%). These observations were in consistent with the findings of Naja et al.  $^{11}$ 

Marshall suggested that *H. pylori* were strongly associated with the duodenal ulcer and many studies supported the statement.<sup>10</sup> Our study also had the same findings.

In the present study, the overall positivity for *H. pylori* was 109 out of 150 patients (72.66%). This was comparable to the results of the study by Tytgat et al.<sup>12</sup> The prevalence of *H. pylori* is decreasing worldwide, probably due to improved hygiene, increased awareness regarding *H. pylori* and increase in consumption of antimicrobials. In a study by Rajachidambaram et al the positivity for *H. pylori* presence was at the rate of 44.21% out of 389 patients.<sup>13</sup>

In the present study, patients with duodenal ulcer have higher incidence of *H. pylori* when compared to patients with gastric ulcers. Out of 89 patients with gastric ulcer, 61 (68.53%) were found to be *H. pylori* positive and out of 51 patients with duodenal ulcers, 42 (82.35%) were found to be positive for *H. pylori*. This is in broad agreement with the findings of Dhinesh Babu et al.<sup>14</sup> In his study the prevalence rate was higher in cases of duodenal ulcers (88%) compared to gastric ulcers (71.42%).

In our study, we also found 10 cases of carcinoma stomach of which 06 cases (60%), proved to be *H. pylori* positive. This is in accordance with the results of the study by Van Zanten et al which showed only a moderate epidemiologic evidence of an association between chronic *H. pylori* infection and gastric cancer.<sup>15</sup>

The presence of *H. pylori* was confirmed by rapid urease test and histopathology by using H&E staining and Giemsa staining. In the present study, out of 150 cases 109 patients were found to be *H. pylori* positive. This was confirmed by rapid urease test in 3 cases and by histopathology in 106 cases. Similar findings were also made by Rajachidambaram et al.<sup>13</sup>

The increase in rapid urease negative in our study may be due to the increase in the usage of NSAIDs or proton pump inhibitors by study population.

#### **CONCLUSION**

The findings of the study reveal that *H. pylori* infections are more common in males than females. Seroprevalance of *H. pylori* increases with increasing age and the prevalence rate of *H. pylori* was higher in duodenal ulcer cases compared to gastric ulcers.

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Institutional Ethics Committee

#### **REFERENCES**

- Frenk RW, Jr, Clemens J. Helicobacter in developing world. Microbes Infect. 2003;5:705-13.
- 2. Torres J, Perez-Perez G, Goodman KJ. A comprehensive review of the natural history of *Helcobacter pylori* infection in children. Arch Med Res. 2000;31:431-69.
- 3. Moayyedi P, Axon AT, Feltbower R. Relation of adult lifestyle and socioeconomic factors to the prevalence of *Helicobacter pylori* infection. Int J Epidemiol. 2002;31:624-31.
- 4. Woodward M, Morrison C, McColl K. An investigation into factors associated with Helicobacter pylori infection. J Clin Epidemiol. 2000;53:175-81.
- 5. Olafsson S, Berstad A. Changes in food tolerance and lifestyle after eradication of Helicobacter pylori. Scand J Gastroenterol. 2003;38:268-76.
- 6. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. Clin Microbiol Rev. 2006;19(3):449-90.
- 7. Blaser MJ. Ecology of *Helicobacter pylori* in the human stomach. J Clin Invest. 1997;100:759-62.

- Yakoob J, Jafri W, Abid S, Jafri N, Abbas Z, Hamid S, et al. Role of rapid urease test and histopathology in the diagnosis of Helicobacter pylori infection in a developing country. BMC Gastroenterol. 2005;5:38.
- 9. Lee JY, Kim N. Diagnosis of *Helicobacter pylori* by invasive test: histology. Ann Transl Med. 2015;3(1):10.
- 10. Warren JR, Marshall B. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. Lancet. 1983;1(8336):1273-5.
- 11. Naja F, Kreiger N, Sullivan T. *Helicobacter pylori* infection in Ontario: prevalence and risk factors. Can J Gastroenterol. 2007;21(8):501-6.
- 12. Tytgat GN. *Helicobacter pylori*-reflections for the next millennium. Gut. 1999;45:145-7.
- 13. Rajachidambaram K, Kaarthesan D. Incidence of helicobacter pylori with acid peptic disease and malignant conditions of upper gastrointestinal tract in a tertiary centre a prospective study. JEMDS. 2016;5(35):1998-2004.
- 14. Babu DK, Bhaskar M. A comparative study of Helicobacter pylori in patients undergoing upper gastrointestine endoscopy in benign and malignant conditions of upper Gastrointestine tract in Karpaga Vinayaga Medical College and Hospital, Madhuranthagam. IOSR-JDMS. 2015;14(3):39-44.
- 15. Van Zanten SV, Sherman PM. *Helicobacter pylori* infection as a cause of gastritis, duodenal ulcer, gastric cancer and nonulcer dyspepsia: a systematic overview. CMAJ. 1994 Jan 15;150(2):177.

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