

factors such as smoking, non-steroidal anti-inflammatory drugs (NSAIDs), and reflux of gastric juice (chemical gastritis) that are also implicated to cause chronic gastritis. *H. pylori*, though is regarded as the primary cause of gastritis, it can act as a synergist in addition with other etiological factors.^[7]

A wide range of laboratory investigations are available for diagnosis of *H. pylori*. The tests belong to non-invasive group and invasive group. Non-invasive tests include, urea breath test, serological Immunoglobulin G (IgG) and Immunoglobulin M (IgM) detection, saliva and urinary antibody test, and stool antigen test.^[8] The invasive tests are endoscopy based tests, which include histopathological examination, rapid urease test (RUT) and polymerase chain reaction. Whereas, invasive tests carry high sensitivity and specificity of >90%,^[9] the role of non-invasive tests such as serology is limited in areas of high prevalence, because of non-distinction between previous and current infection. There is wide variation in prevalence of *H. pylori* infection in different regions of the world. Though, many studies have already been conducted in the past on the current topic, still there is a paucity of information about *H. pylori* infection prevalence in this part of Southern India. The present study was undertaken to estimate the prevalence of *H. pylori* infection among patients presenting with the dyspepsia and compute its association with gastrointestinal lesions, in a medical college hospital in Kerala, India. The hospital serves as a reference center for rural and suburban population of Ernakulam district, Kerala, India.

Subjects and Methods

The study was conducted with 530 out patients and in-patients of Sree Narayana Institute of Medical Sciences-Ernakulam, Kerala, India. The study conducted was a cross-sectional prospective study, during the period of May 2010 to September 2012. The patients were selected on the basis of chief complaints of dyspepsia and the age of patients ranged from 14 to 86 years. Certain exclusion criteria were applied such as patients on proton pump inhibitor therapy or any antibiotic therapy within last 1 month. Endoscopy was carried out using the "Pentax" forward viewing esophago gastro duodenoscope. The patients were taken for upper gastrointestinal endoscopy after making them fast overnight. The endoscopy was considered normal on visualizing mucosa, which is pink in color, smooth, and lustrous. Two endoscopic biopsy fragments were obtained from each patient from the antrum. One biopsy fragment was sent to histopathology department in formalin container. Two sections of 4 μ thickness were cut from each block and mounted on two slides, one each on a slide. Slides were stained with normal H and E, stain and Giemsa stain. Histopathological assessment of gastric mucosa was carried out by a pathologist and grading was carried out for mononuclear cell infiltration, neutrophilic infiltration, atrophy, intestinal metaplasia, and density of *H. pylori* according to the visual analog of updated Sydney grading system^[10] for reporting gastric biopsies. Microscopic assessment of slides was carried out using labomed microscope vision 2000, India. In positive

cases, *H. pylori* appeared as light bluish rods in H and E stained slides with varying sizes (3-6 μ) on the luminal surface of mucosal cells. In Giemsa stain, *H. pylori* appeared dark blue in a light background. Diagnosis of reactive gastritis was based on the criteria laid by Dixon *et al.*,^[11] and Niemelä *et al.*,^[12] Another biopsy fragment was sent to microbiology department for RUT. RUT was performed by the following method: Urea (2 g) was dissolved in 20 ml double distilled water. 20 drops of phenol red were added to the solution and pH was adjusted between 6.8 and 6.9 by adding a drop of N/10 HCl, if pH was greater or N/10 NaOH if pH was less. Solution was faint yellow tint at this stage. This was transferred to sterile vial each containing 2 ml in each vial. Biopsy material was added and the temperature was kept constant at 35-37°C. Test was considered positive if color changed within 30 min and weekly positive if the change occurred after 2 h.

The study was approved and ethical clearance was obtained from institutional ethical and scientific research committee. The study involved the data analysis of routine procedure carried out in an institution and informed consent was obtained from each patient before the procedure.

Data analysis

Data analysis was carried out using the statistical package for social sciences, for Windows version 16.0 (SPSS 16; Chicago, IL, USA). Categorical variables were compared with Chi-square test. $P < 0.05$ was taken as statistically significant.

Results

Data of 530 patients were analyzed, out of which 61.9% (328/530) were males and 38.1% (202/530) were females. The mean (SD) for age was 48.8 (16.7) years with age ranging from 14 to 86 years. Histopathological examination showed *H. pylori* positivity in 59.4% (315/530) cases and RUT showed *H. pylori* positivity in 57.7% (306/530) cases. Diagnosis of *H. pylori* infection was made if both or either of the tests was positive. 62.0% (329/530) cases were found to be *H. pylori* positive, on combining both test results. The proportion of male cases positive for *H. pylori* 62.1% (204/328) was not statistically significant when compared to the proportion of females positive for *H. pylori* 61.8% (125/202) with P value of 1.00 [Table 1]. Similarly, comparison of *H. pylori* positive patients with age < 50 years –61.7% (176/285) and age > 50 years –62.4% (153/245) did not yield any statistically significant result with P value of 0.93. *H. pylori* prevalence was highest in the age group of 81-90 years [Table 2] with 75% (9/12) patients showed *H. pylori* positivity. Endoscopic abnormalities when compared with *H. pylori* presence yielded significant results, with 89.3% (294/329) of *H. pylori* positive cases revealing any of the abnormalities, with $P < 0.01$ [Table 3]. The most common endoscopic abnormality was gastritis 69% (306/443), followed by duodenitis –16.9% (75/443), esophagitis –11.9% (53/443), duodenogastric reflux –6.9% (31/443), hiatal hernia –6% (27/443) gastric

ulcer (GU) –2% (9/443), duodenal ulcer (DU) –2% (9/443) and Barrett’s esophagus 2% (9/443). Serious gastrointestinal pathology (GU, DU, and carcinoma) was only seen in 7.9% (42/530) patients. Patients showed varied presentation of clinical symptoms such as abdominal pain –60.9% (323/530), gastric fullness –16.9% (90/530), vomiting –12% (64/530), fatty food intolerance –9% (48/530), bloating –6.9% (37/530), belching –6% (32/530), melena –5% (27/530), early satiety –5% (27/530) and weight loss –3.9% (21/530).

Table 1: *Helicobacter pylori* sex distribution

<i>Helicobacter pylori</i>	Male (%)	Female (%)	Total (%)
Present	204 (62.0)	125 (38.0)	329 (100)
Absent	124 (61.7)	77 (38.3)	201 (100.0)
Total	328 (61.9)	202 (38.1)	530 (100.0)

df-1, χ^2 -0.005, P-1.00

Table 2: Age distribution of *Helicobacter pylori*

Age in years	<i>Helicobacter pylori</i> positive	<i>Helicobacter pylori</i> negative	Total
11-20	14 (58.3)	10 (41.7)	24
21-30	34 (58.6)	24 (41.4)	58
31-40	62 (65.2)	33 (34.8)	95
41-50	66 (61.1)	42 (38.9)	108
51-60	67 (62.6)	40 (37.4)	107
61-70	48 (58.5)	34 (41.5)	82
71-80	29 (65.9)	15 (34.1)	44
81-90	9 (75)	3 (25)	12

Table 3: Correlation of endoscopic abnormalities with *Helicobacter pylori* infection

Infection	Result	Endoscopic abnormalities		
		Present (%)	Absent (%)	Total (%)
<i>Helicobacter pylori</i>	Positive	294 (89.4)	35 (10.6)	329 (100)
	Negative	149 (74.1)	52 (25.9)	201 (100)

df-1, χ^2 -21.100, P<0.01

Table 4: Types of histopathological reporting

Types of pathological reporting	Percentage of total biopsy cases examined
<i>Helicobacter pylori</i> gastritis	(329/530) 62.0
Reactive gastritis	(56/530) 10.5
Non-specific gastritis	(58/530) 10.9
Ulcer (gastric, duodenal)	(21/530) 3.9
Dysplasia/carcinoma	(21/530) 3.9
Normal gastric mucosa	(45/530) 8.4

Table 5: Pathological diagnosis and associated histopathological features

Pathological diagnosis	<i>Helicobacter pylori</i> % positivity	% with acute inflammation	% with chronic inflammation	% with intestinal metaplasia	% with glandular atrophy
Gastritis (n=443)	(292/443) 65.9	(180/443) 40.6	(359/443) 81.0	(36/443) 8.1	(83/443) 18.7
Peptic ulcer (n=21)	(18/21) 85.7	(21/21) 100	(18/21) 85.7	(7/21) 33.3	(11/21) 52.3
Dysplasia or cancer (n=21)	(19/21) 90.5	(15/21) 71.4	(17/21) 80.9	(12/21) 57.1	(15/21) 71.4

df-1, χ^2 -13.11, P<0.01

Histopathological features when analyzed, 10.5% (56/530) patients were found to have reactive gastritis [Table 4]. *H. pylori* positivity was seen in 65.9% (292/443) patients [Table 5] with diagnosis of gastritis. Normal gastric mucosa was evident in 8.4% (45/530) cases. Histological features such as intestinal metaplasia and glandular atrophy were seen in 8.1% (36/443) and 18.7% (83/443) respectively in patients with gastritis, whereas these entities were seen in 57.1% (12/21) and 71.4% (15/21) of cases respectively in patients with dysplasia/cancer. The correlation of intestinal metaplasia with *H. pylori* infection was statistically non-significant, with P value of 0.58. The correlation of peptic ulcer and dysplasia/cancer was statistically highly significant with *H. pylori* infection, with P value of 0.02 and <0.01 respectively. The correlation of gastritis with *H. pylori* infection was statistically non-significant, with P value of 0.09.

Discussion

Gastritis, gastric ulceration, and gastric malignancies have many etiological factors, among which *H. pylori* infection is the principal cause. *H. pylori* infection is dependent upon many variables such as age, sex, socioeconomic status, dietary habits, genetic, and immunological factors. In the present study, we did not get a significant difference in *H. Pylori* prevalence according to gender. This is in concordance with the study results of Tarkhashvili et al.,^[13] and Shokrzadeh et al.,^[14] In contrast a study by Kaore et al.,^[15] showed higher prevalence in male gender.

Age distribution of *H. pylori* infection did not show any trend towards increase or decrease in infection with the advancing age. Though maximum percent of *H. pylori* positivity –75% (9/12) was seen in the age group of 81-90 years, this can be attributed to much less number of individuals^[12] studied in this group, compared to other age groups. There was no statistically significant difference in prevalence of *H. pylori* in the age group of <50 years and >50 years. This is similar to the observations laid by Tarkhashvili et al.,^[13] though few studies such as Shokrzadeh et al.,^[14] and Kaore, et al.,^[15] reported increased *H. pylori* infection in age groups of 20-40 years than the older age group.

In the present study, the commonest identifiable lesion at endoscopy was gastritis (69%). The correlation of endoscopic abnormality with *H. pylori* infection was statistically highly significant with a P < 0.01, proving endoscopic changes to be a sensitive indicator of *H. pylori* infection. This is in contrast to the observation laid by Jemilohun et al.,^[16] in which the correlation was not statistically significant. This can be

attributed to a lower number of cases (86) being evaluated in their study compared to the present study (530).

Pathological reporting of 530 cases showed only 7.2% (42/530) of cases having serious gastrointestinal complications like peptic ulcers (GU and DU) and dysplasia/carcinoma. The association of these lesions with *H. pylori* infection was found to be statistically significant with 85.7% (18/21) patients with peptic ulcer and 90.5% (19/21) with dysplasia/carcinoma. This is comparable to the study of Zapata-Colindres *et al.*,^[17] and Ahmad *et al.*,^[18] documenting *H. pylori* prevalence in gastric ulceration patients to be 80% and 84% respectively. Cotran *et al.*,^[19] reported the international association of *H. pylori* with gastric ulceration to be more than 70%.

The present study showed 10.5% of cases having reactive gastritis. There is rising incidence of reactive gastritis in rural and suburban population of developing nations like India due to increased duodenogastric reflux associated with changing life-style of the population, injudicious, and rampant use of drugs like NSAID, which are easily available and often being prescribed for musculoskeletal ailments.

The present study showed that dysplasia and carcinoma develop only in few of the cases having predisposing factors such as intestinal metaplasia and glandular atrophy. High *H. pylori* prevalence of 90.5% (19/21) in cases having dysplasia and carcinoma in this study, implies that the majority of gastric adenocarcinomas can be prevented with early stage detection of *H. pylori* and *H. pylori* eradication therapy. This is in concordance with the findings documented by an International agency for research on cancer, stating that 60% or more of gastric cancers world-wide can be prevented by the absence of *H. pylori* infection.^[20]

The present study has significant advantages like being conducted in a large patient group of 530 and computation of association of multiple variables with *H. pylori* infection. The present study has a major limitation that association of *H. pylori* infection with life-style related modifiable factors was not accessed. There is a need of another broader study in this region, assessing the association of different demographic and life-style factors and pre-existing conditions like diabetes mellitus with prevalence of *H. pylori* infection and follow-up of the patients after treatment and life-style modifications.

In conclusion, there is a high prevalence of *H. pylori* infection in rural population of Ernakulam district of Kerala, South India. Though, the prevalence of *H. pylori* gastritis and associated abdominal symptoms is high in number, serious gastrointestinal complications develop in few. Absolute prevention of these complications and relief from the distressing abdominal symptoms can be achieved through early detection by conventional and affordable diagnostic methods and empirical treatment with anti *H. pylori* therapy.

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