



A Study on Incidence of *Helicobacter Pylori* Infection in Dyspeptic Patients in Sree Balaji Medical College and Hospital

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ABSTRACT

Objective: *Helicobacter pylori* (*H. pylori*) infection is a major cause of various upper gastrointestinal (UGI) disorders. The aim of this study was to determine the prevalence of *H. pylori* among patients with dyspepsia admitted to Sree Balaji Medical college and Hospital, Chennai.

Methods: Totally, 180 patients were enrolled for the study. Their premedical history, demographic data, lifestyle habits and socio-economic status were carefully documented. The specimen bile ducts were analysed using biochemically and microbiologically. The results were expressed using statistical Softwares.

Results: The present study showed that revealed that the infection was based on the aging and the patients and more prone in male (67%). The patients from upper middle class had higher infections (35%). Our results showed a strong correlation with *H. pylori* infection, urease test and biopsy. The results were statistically significant.

Conclusion: The well-described association of *H. pylori* with dyspepsia was confirmed by our study.

Key words: Asymptomatic gallstone, Prevalence of gallstone disease, Urease test, Dyspepsia

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INTRODUCTION

Brown et al. of Perth, Western Australia discovered *H. pylori* in 1983. Originally, the organism was named *Campylobacter pylori* because it was structurally like other *Campylobacter* species, such as *C. jejuni* [1]. Signs of *H. pylori* infection such as gram-negative gastric bacilli, gastric urease and epidemics of hypochlorhydria have been described since the late nineteenth century [2]. These observations could be better explained after Warren and Marshall in the early 1980's managed to culture a bacterium that was to be designated *Campylobacter pyloridis* [3]. In 1989, the genus *Helicobacter* was created, and the bacterium received the name *H. pylori* [1]. *H. pylori* is a small (0.5 - 1.0 µm in width and 2.5 to 5.0 µm in length), spiral shaped, highly mobile, gram

negative rod with 4 - 6 unipolar sheathed flagella [4]. The microorganism grows slowly invitro and requires special media and a microaerophilic (5% O₂, 10% CO₂, and 85% N₂) environment [5]. The most striking biochemical characteristic is the production of large quantities of urease. This enzyme digests urea to produce carbon dioxide and ammonia. In the presence of water this leads to the formation of ammonium hydroxide [6]. In this way, *H. Pylori* is able to neutralize the acid in its direct environment.

H. pylori, colonizes the human stomach, can cause gastritis, is strongly associated with gastric and duodenal ulceration (DU) and has been implicated in the causation of gastric carcinoma and mucosa-associated lymphoid tissue (MALT) lymphomas. It has been reported that there is relationship between *H. pylori* infection and children's gastroenterological diseases [7]. However, only 10-20% of infected individuals manifests severe complications and this selectivity in disease progression is inadequately understood [8,9]. Epidemiological studies

have shown that a weakly positive correlation exists between chronic gastric infection with *H. pylori* and coronary heart disease [10]. Urease, vacuolating cytotoxin VacA, and the pathogenicity island (cagPAI) gene products, are the main factors of virulence of this organism. *H. pylori* LPS may have an important role in autoimmune-mediated damage in the gastric mucosa [11]. Half of the world's population is estimated to be infected with *H. pylori*, which makes it one of the most common bacterial pathogens in humans. The prevalence of *H. pylori* infection worldwide is approximately 50%, it reaches as high as 80%–90% in developing countries, and about 35%–40% in the United States. Approximately 20% of persons infected with *H. pylori* develop related gastro duodenal disorders during their lifetime. The annual incidence of *H. pylori* infection is about 4%–15% in developing countries, compared with approximately 0.5% in industrialized countries. Documented risk factors include low socioeconomic status, overcrowding, poor sanitation, or hygiene, and living in a developing country.

In "Israel", the prevalence of *H. pylori* infection is about 60%, and the annual incidence of gastric cancer is about 15 per 100,000 populations. The prevalence of *H. pylori* infection was 10% among children in Egypt. In crude analyses, prevalence was associated with increasing age, non-white skin color, lower current family income, lower education level, higher size of the family, low socioeconomic conditions in childhood, higher number of siblings and attendance to day-care centers in childhood, and presence of dyspeptic symptoms. Socioeconomic conditions in childhood besides ethnicity and presence of dyspeptic symptoms were the factors significantly associated with the infection. There are several methods of diagnosing *H. pylori* infection including invasive procedures using mucosa biopsies taken during endoscopy (mainly culture, histology, and the rapid urease test) and non-invasive procedures. Non-invasive testing methods for detection of *H. pylori* or confirmation of eradication include: 1) Antibody tests (in serum, saliva, or blood); 2) Antigen tests (in stools, saliva, or urine); and 3) Radioactive or non-radioactive urea breath tests. The most interesting of the non-invasive tests is the detection of antigens in stool samples by enzyme immunoassay technique. While this test has good performance at a reasonable cost,

doubts exist regarding patient and clinician compliance and actual performance, particularly about inter laboratory variability. The urea breath test is based on analysis of samples of exhaled air before and after ingestion of urea containing specially labelled carbo.

PCR is a powerful technique for the detection of target DNA in various clinical specimens, but its application to fecal specimens has been limited due to the presence of substances inhibiting the reaction. Eradication therapy of symptomatic *H. pylori* infection substantially reduces the recurrence of associated gastro duodenal diseases. Therapy entails complicated regimens of several antimicrobial agents for at least 2 weeks. In general, triple therapy regimens usually entail two of the following antimicrobial agents: metronidazole, amoxicillin, tetracycline, or clarithromycin in combination with a proton pump inhibitor or bismuth salts. The most common causes of treatment failure are patient noncompliance and antimicrobial resistance of the infecting *H. pylori* strain. Quadruple regimens are used as a salvage therapy when triple therapy regimens have failed. However, the success of treatment is usually dependent on early detection. Moreover, prevention of *H. pylori* infection seems to be a wise strategy. Prevention strategies require deep understanding of the transmission risks. *H. pylori* infection can be prevented by interrupting the transmission of the infection by improving environment, socioeconomic status, and personal hygiene. It has been shown convincingly that with the improvement of socioeconomic status in developed countries, *H. pylori* infection has gone down by 50% over the last 3 decades. Further studies have shown that superficial gastric ulcers in a mouse model infected with *H. pylori* can be prevented by the administration of a recombinant oral vaccine with E. coli heat labile toxin (LT) given as adjuvant.

AIM OF THE STUDY

- ✓ To study the age incidence of patient with *H. pylori* in dyspepsia.
- ✓ To study the sex incidence of patient with *H. pylori* in dyspepsia.
- ✓ To study the incidence of *H. pylori* with presenting complaints of dyspepsia.

- ✓ To study the incidence and duration of complaints patient with *H. pylori* presenting has dyspepsia.
- ✓ To study the incidence of *H. pylori* in endoscopic findings.
- ✓ To study the incidence of *H. pylori* in endoscopic biopsy.
- ✓ To study the incidence of *H. pylori* in rapid urease test.
- ✓ To study the treatment of *helicobacter pylori* infection and eradication.

MATERIALS AND METHODS

The materials for this study were 180 patients who were admitted in surgical wards at Sree Balaji Medical College and Hospital, Chennai from October 2013 to October 2015 (period of 2yrs). All patients with dyspeptic symptoms who were referred to the Department of Gastroenterology were assessed by a consultant Surgical Gastroenterologist. Patients requiring upper gastrointestinal endoscopy were eligible for study. The procedure was explained to the patient and the need to check for *Helicobacter pylori* infection was explained. Informed consent was obtained from all patients.

Endoscopy was carried out with Olympus forward viewing flexible endoscope. Local pharyngeal anesthesia with 10% lignocaine spray was used in all patients as well as sedation with midazolam intravenously. All patients had evaluation of the oesophagus, stomach and proximal duodenum and abnormalities were documented. Gastric biopsies for histological examination and Rapid urease test for the presence of *Helicobacter pylori* organisms were performed on all patients. Five samples were taken from the stomach: two from the antrum, two from the body and one from the incisura angularis. Gastric samples for histological evaluation were sent to Department of pathology, Sree Balaji medical college and hospital. Data obtained included age, gender, occupation, socio economic status, presenting complaints, duration of symptoms, findings on endoscopy and results of gastric biopsies and rapid urease test.

RESULTS

This study was conducted with the patients

admitted to Sree Balaji Medical College and Hospital, Chennai during the period from October 2013 to October 2015. Totally, 180 cases of incidence of *H. pylori* in dyspepsia studied and all of them were undergone for upper gastrointestinal endoscopy, rapid urease test and biopsy. The present study revealed that the infection was based on the aging and the patients between 40-50 years were prone (39%) for the infection (Table 1 and Figure 1). The results (Table 2 and Figure 2) also showed that the male was having higher infection rate (67%)

Table 1: Relationship between aging and *H. pylori* infection in studied patients.

Age	H. Pylori Positive	%	H. Pylori Negative	%	Total no of cases	%
21-30	3	2	1	2	4	2
31-40	16	13	10	18	26	14
41-50	49	40	20	36	69	39
51-60	37	30	20	36	57	32
> 60	19	15	5	10	24	13
Total	124		56		180	100

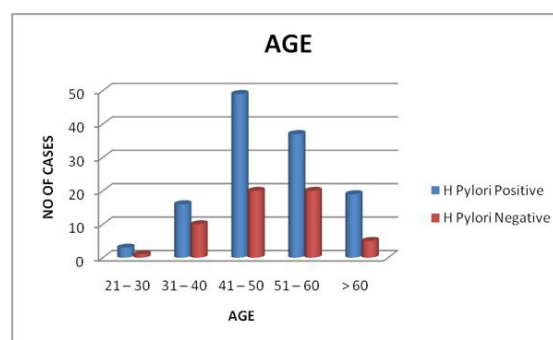


Figure 1: Sex Based variation in GSD prevalence among patients.

Table 2: Relationship between sex and *H. pylori* infection in studied patients.

Gender	H. Pylori Positive	%	H. Pylori Negative	%	No of cases	%
Male	95	77	27	48	122	67
Female	29	23	29	52	58	33
TOTAL	124		56		180	100

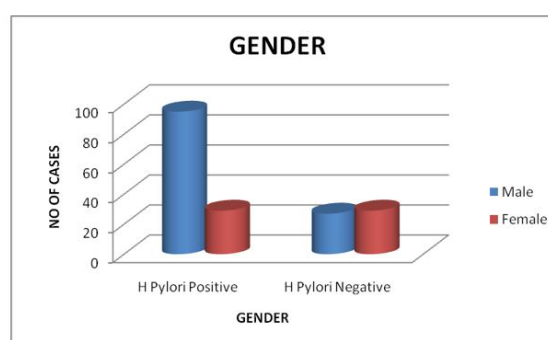


Figure 2: Relationship between Sex incidence and *H. pylori* infection in studied patients.

than the females (33%). Our results further showed that the occupation play a pivotal role in infection rate (Table 3 and Figure 3). The persons working in skilled area had higher rate (38%) followed by unskilled (30%), professional (25%) and unemployed (7%). The present study demonstrated the significance of socio-economic status and infection rate (Table 4 and Figure 4). The upper middle class had higher infections (35%) than middle class (25%), followed by lower middle class (14%) and lower class (6%-20%).

The smokers were found to be with higher infection rate (41%) than alcoholics (32%) and interestingly, our results showed that the as both smokers + alcoholics had lower rate (7%). These

Table 3: Relationship between occupation and *H. pylori* infection in studied patients.

Occupation	<i>H. Pylori</i> Positive	%	<i>H. Pylori</i> Negative	%	No of cases	%
Professional	32	26	13	23	45	25
Skilled	51	41	18	33	69	38
Unskilled	33	27	21	37	54	30
Unemployed	8	6	4	7	12	7
TOTAL	124		56		180	100
Total	124		56		180	100

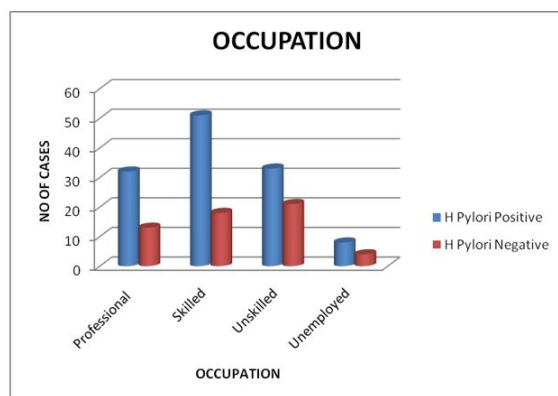


Figure 3: Relationship between Occupation and *H. pylori* infection in studied patients.

Table 4: Socio economic status and *H. pylori* infection in studied patients.

Socio economic status	<i>H. Pylori</i> Positive	%	<i>H. Pylori</i> Negative	%	No of cases	%
Upper Class	8	6	4	7	12	6
Upper Middle Class	49	40	13	23	62	35
Middle Class	28	23	16	29	44	25
Lower Middle Class	18	14	8	14	26	14
Lower Class	21	17	15	27	36	20
Total	124		56		180	100

results showed that the sedentary lifestyles had significant role in determining the infection rate (Table 5 and Figure 5). The patients felt the varied duration in disease symptoms (Table 6 and Figure 6). 36% of them told that the symptoms were lasting for at least 3 weeks and 37% of them felt it for 3-4 weeks. 27% registered

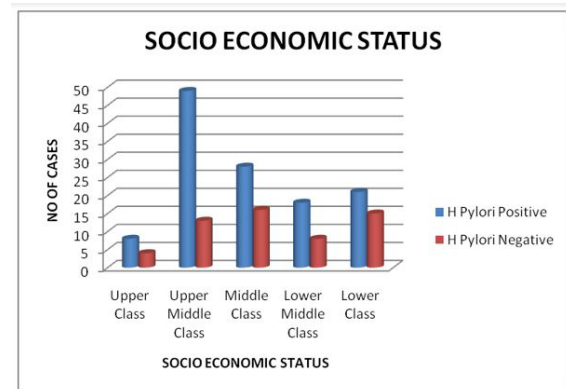


Figure 4: Relationship between: Socio economic status and *H. pylori* infection in studied patients.

Table 5: Sedentary lifestyle and *H. pylori* infection in studied patients.

Sedentary lifestyle	<i>H. Pylori</i> Positive	%	<i>H. Pylori</i> Negative	%	No of cases	%
Smoking	55	44	19	34	74	41
Alcohol	53	43	5	9	58	32
Smoking+ Alcohol	9	7	4	7	13	7
Tea Totaller	7	6	28	50	35	20
Total	124		56		180	100

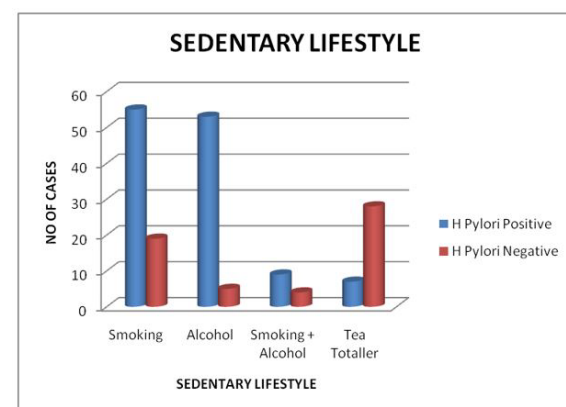


Figure 5: Relationship between: Sedentary lifestyle and *H. pylori* infection in studied patients.

Table 6: Symptom duration and *H. pylori* infection in studied patients.

Duration of symptoms	<i>H. Pylori</i> Positive	%	<i>H. Pylori</i> Negative	%	No of cases	%
< 3 weeks	49	40	16	29	65	36
3 to 4 weeks	49	40	18	32	67	37
> 4 weeks	26	20	22	39	48	27
Total	124		56		180	100

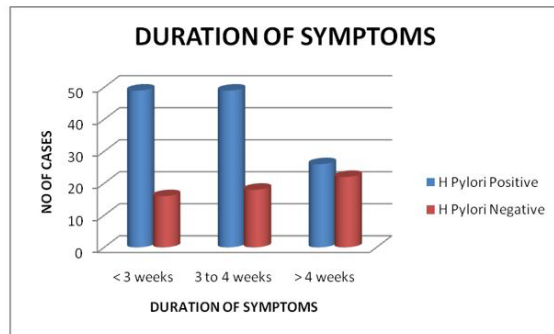


Figure 6: Relationship between: Duration of symptoms and *H. pylori* infection in studied patients.

as more than 4 weeks. The present study showed that the most common symptoms (Table 7 and Figure 7) presented by *H. pylori* positive was epigastric pain (30%) followed by epigastric burning (11%), early satiety (13%), nausea (11%), vomiting (16%), bloating (06%), belching (07%) Post Prandial Fullness (06%).

The endoscopic finding revealed that the patients with antral erosions had higher chances (37%) of *H. pylori* infections. Next to them, patients with peptic ulcer (27%) were prone

Table 7: Symptoms and *H. pylori* infection in studied patients.

Symptoms	Male	%	Female	%	No of cases	%
Epigastric Pain	40	73	15	27	55	30
Epigastric Burning	14	70	6	30	20	11
Early Satiety	13	57	10	43	23	13
Nausea	14	70	6	30	20	11
Vomiting	17	61	11	39	28	16
Bloating	8	72	3	28	11	6
Bleching	9	75	3	25	12	7
Post Prandial Fullness	7	64	4	36	11	6
Total	122		58		180	100

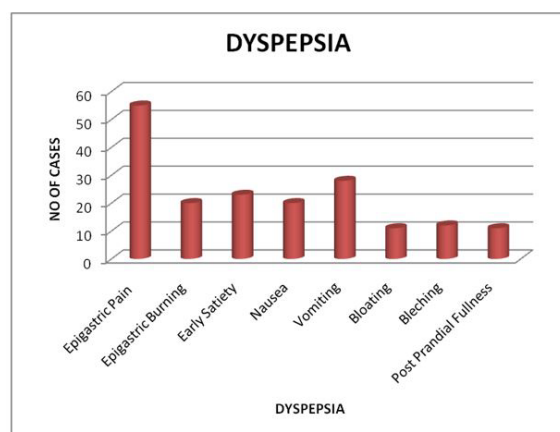


Figure 7: Relationship between Dyspepsia and *H. pylori* infection in studied patients.

for infections followed by reflex oesophagitis (25%), Gastroesophageal Reflex Disease (10%). Surprisingly, a single patient with gastric cancer showed lowest infection rate in the study. The results were summarized in Table. 8 and Figure. 8. A comparative analysis of *H. pylori* infection and Rapid Urease Test in the present study (Table 9 and Figure 9) revealed that the urease positivity

Table 8: Endoscopic findings and *H. pylori* infection in studied patients.

Endoscopic findings	Male	%	Female	%	Overall, no of cases	%
Peptic Ulcer Disease	27	55	22	45	49	27
Reflex Oesophagitis	39	87	6	13	45	25
Antral Erosions	45	69	20	31	65	37
Gastroesophageal Reflex Disease	10	55	8	45	18	10
Gastric Malignancy	1	33	2	67	3	1
Total	122		58		180	100
Bleching	9	75	3	25	12	7
Post Prandial Fullness	7	64	4	36	11	6
Total	122		58		180	100

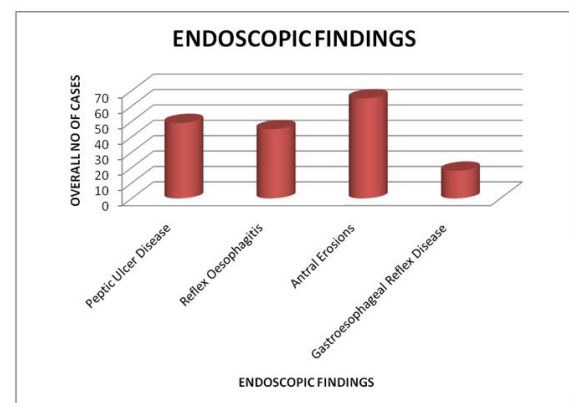


Figure 8: Relationship between Dyspepsia and *H. pylori* infection in studied patients.

Table 9: Rapid Urease Test and *H. pylori* infection in studied patients.

	<i>H. Pylori</i> infection Positive		<i>H. Pylori</i> infection Negative		Total	
	N	%	N	%	N	%
Rapid Urease Test						
Epigastric Pain	41	75	14	25	55	30
Epigastric Burning	11	55	9	45	20	11
Early Satiety	13	57	10	43	23	13
Nausea	12	60	8	40	20	11
Vomiting	18	64	10	36	28	16
Bloating	6	55	5	45	11	6
Bleching	7	58	5	42	12	7
Post Prandial Fullness	6	55	5	45	11	6
Total	114		66		180	100
Chi-square Value ($\chi^2=17.34$) P value=0.0000 (< 0.05)						

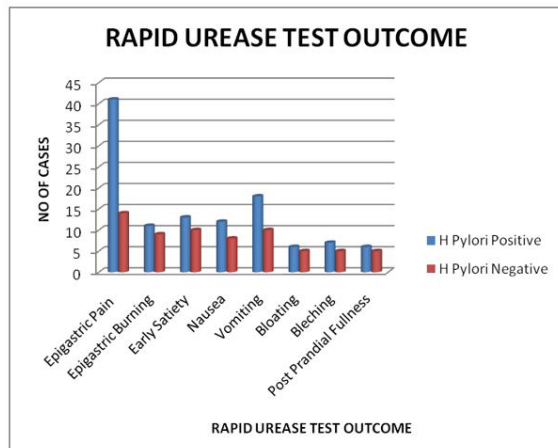


Figure 9: Relationship between Rapid Urease test Outcome and *H. pylori* infection in studied patients.

could be a strong indicator for the confirmation of the infection and statistically significant ($p < 0.05$, Chi-square Value ($\chi^2=17.34$). In addition to, our study results also showed that biopsy could be another indicator the infection in patients (Table 10 and Figure 10). The results were statistically significant. In this study the patients who were confirmed with *H. Pylori* positive, treatment with Triple regimen was given for a duration of 2 weeks and when they were followed up after 3 months. at the end of drug course, 95% patients with Gastroesophageal Reflex Disease and 93% of patients with Antral Erosions were cured

Table 10: Biopsy and *H. pylori* infection in studied patients.

	H. Pylori infection Positive		H. Pylori infection Negative		Total	
	N	%	N	%	N	%
Peptic Ulcer Disease	32	65	17	35	49	27
Reflex Oesophagitis	29	64	16	36	45	25
Antral Erosions	48	74	17	26	65	37
Gastroesophageal Reflex Disease	12	67	6	33	18	10
Gastric Malignancy	3	0	0	0	3	1
Total	124	100	56	100	180	

Chi-square Value ($\chi^2=14.25$) P value=0.0015 (<0.05)

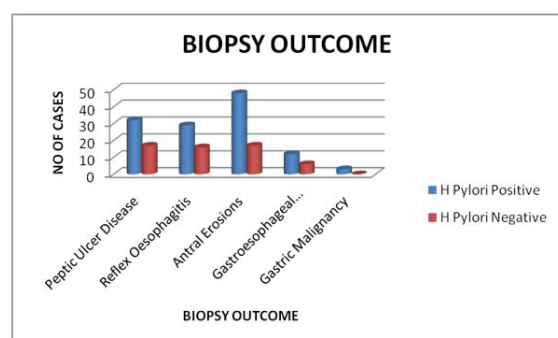


Figure 10: Relationship between Biopsy outcome and *H. pylori* infection in studied patients.

followed by Peptic Ulcer Disease (91%), Reflex Oesophagitis (89%), of the cases were cured with respect to. However, the Gastric Malignant cases were referred to the Oncology department for further treatment (Table 11 and Figure 11).

Table 11: *H. pylori* treatment and its eradication.

	H. Pylori +ve	Rx for H. Pylori Triple regimen (Clarithromycin /Amoxycillin) PP1	Duration	Follow up after 3 months	
				Eradicated %	Not Eradicated %
Peptic Ulcer	32	32	14 days	91	9
Disease Reflex Oesophagitis	29	29	14 days	89	11
Antral Erosions	48	48	14 days	93	7
Gastroesophageal Reflex Disease	12	12	14 days	95	5

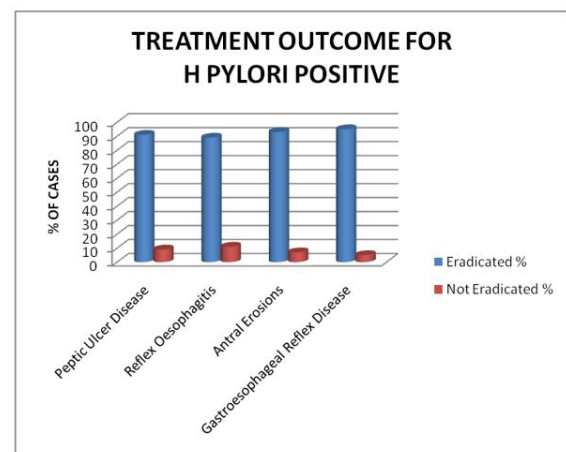


Figure 11: Relationship between treatment outcome for *H. pylori* positive.

DISCUSSION

H. pylori is a spiral-shaped microaerophilic Gram-negative bacterium that colonizes the gastric mucosa of human beings. The microorganism is the major agent of gastritis and plays an important role in the pathogenesis of peptic ulcer and gastric cancer. *H. pylori* is believed to infect the host by the fecal-oral route and home to the gastric mucosa. Although it is acid sensitive, *H. pylori* can survive in the stomach for short periods by neutralizing the gastric acid. The current therapy for *H. pylori* infection is efficacious but the treatment regimen is complex and demanding on the patient and it does not provide resistance to future infections. The aim of this study is to determine the incidence of *H. pylori* infection in dyspeptic patient. In this study 180 patients undergoing upper gastrointestinal endoscopy were interviewed. Data concerning age, sex, occupational status, socio economic

status, and sedentary lifestyle, duration of symptoms of dyspepsia, endoscopic findings, rapid urease test and biopsy were obtained during interviews.

The present study demonstrated that highest infection occurred at the age of 50 (39%) and this finding is agreed with similar studies in developed countries which showed that the infection begin in younger age and increasing annually with age. This may be due to the reason that the number of participant of the older age in this study. In another study conducted in south of Brazil (2005), the author showed totally different results. Among the 563 eligible individuals, the prevalence rate of *H. pylori* infection was 63.4%. In crude analyses, prevalence was associated with increasing age [11]. Our results showed that the male were more prone for *H. pylori* infection and this accordance with previous study [11]. Several studies reported the strong correlation between *H. pylori* infection and occupation [3]. The prevalence of HP infection varies among countries and within a country. The risk factors of HP infection confirmed that low socioeconomic status, crowded living conditions in childhood, low educational level of parents and unreliable well-water supply in the childhood household are associated with the infection [1]. In this work, there was significant correlation detected between socioeconomic level and HP infection. This could be attributed to a higher admission rate of patients of upper medium socioeconomic level 35%.

The present study demonstrated that the highest positive results were in 41 % are reported as smokers. In another study which agree with our result, El- Barrawy et al. [4], demonstrated that infection prevailed mostly (70%) in smokers (113 out of 161). According to odds ratio, the risk of infection was 5.3 times higher for smokers than nonsmokers, which was significant. They attributed their finding to the destructive effect of smoking on the immunity of gastric mucosa and lining layers and hence increasing its susceptibility to infection by *H. pylori*. Communal Shisha smoking might carry the risk of passing the infection from a diseased person to an uninfected one, as oral-to-oral infection. Tables 6 and 7 In this study most common symptoms presented by *H. pylori* infection were more common in patients suffering from

epigastric pain (30%). Other studies added that the presence of epigastric pain, especially if associated with dyspeptic symptoms is more associated with abnormal gastric pathology. Endoscopic findings in dyspeptic patients' Antral mucosa is the main target for HP colonization of 37% in this study. Followed by peptic ulcer disease 27%, reflux oesophagitis 25%, Gastroesophageal Reflux Disease 10% and one case of gastric cancer were positive for *H. pylori*. Gastritis occurs due to the inflammation of the gastric mucosa that can be caused by HP. Blaser reported that chronic active gastritis is mainly induced by HP. We have discovered a significant difference ($p=0.027$) between positive HP histopathology and grading of gastritis was discovered similar to Akbar et al. [9]. In healthy population, HP seroprevalence ranges from 25% in developed countries up to 80% in developing countries [6]. In this setting, it was 73.3%. This result is comparable to an earlier study by Al-Moagel et al. [8]. We must mention that our small control number (15 healthy population) should be reported as a limitation in this study. In addition, the HP seroprevalence among those individuals was mostly an indication of past HP exposure not an active infection. In this study when the comparison was made between the *H. Pylori* patients in both the classification (positive and negative) with respect to Rapid Urease Test, P value is 0.0000 (< 0.05) which concludes the test that the *H. Pylori* positive cases are significant. The test which was performed on biopsy collected from patients during upper gastroscopy proved to be rapid and simple. In our study two tests were used to evaluate *H. pylori* infection. 1-Invasive test required endoscopy for (URUT, MB staining). Unfortunately, at present, no single test can be relied upon to detect *H. pylori* infection and a combination of tests is recommended as gold standard. A patient was considered an *H. pylori* positive if culture alone or histology plus rapid urease test (RUT) were positive. The decision by the physician to select the proper part to collect a biopsy during gastroscopy would greatly affect the outcome of the invasive tests such as methylene blue staining and Ultra rapid urease tests. A study in Yemen showed that the prevalence of *H. pylori* infection among 275 dyspeptic patients was 82.2%. In their study they depended on URUT for detect the infection [7]. The present study demonstrated the systematic

need of diagnosis and follow up to eradicate the *H. pylori* infections.

CONCLUSION

The present study aimed to detect incidence of *H. pylori* infection in dyspeptic patients and attempt to determine possible risk factors is acquisition of the microorganism. Our results showed that age, gender and socioeconomic status play a pivotal role in determining the severity of the disease. Further, we found a strong correlation with urease positivity and *H. pylori*. These findings certainly would be useful to prevent and eradicate the *H. pylori* infections.

FUNDING

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ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1. Brown L. *Helicobacter pylori*: Epidemiology and routes of transmission. Epidemiol Rev 2000; 22:83-97.
2. Zmira S, Haim S, Yaron N, et al. Resistance of *Helicobacter pylori* isolated in Israel to metronidazole, clarithromycin, tetracycline, amoxicillin and cefixime. J Antimicrob Chemother 2002; 49:1023 -1026.
3. Peters C, Schablon A, Harling M, et al. The occupational risk of *Helicobacter pylori* infection among gastroenterologists and their assistants. BMC Infect Dis 2011; 11:154.
4. El Barrawy MA, Morad MI, Gaber M. Role of *Helicobacter pylori* in the genesis of gastric ulcerations among smokers and nonsmokers. Eastern Mediterranean Health J 1997; 3:316-321.
5. Abo-Shadi MA, El-Shazly TA, Al-Johani MS. Clinical, endoscopic, pathological and serological findings of *Helicobacter pylori* infection in Saudi patients with upper gastrointestinal diseases. J Adv Med Med Res 2013; 1109-1124.
6. Bardhan PK. Epidemiological features of *Helicobacter pylori* infection in developing countries. Clinical Infect Dis 1997; 25:973-978.
7. Gunaid AA, Hassan NA, Murray-Lyon I. Prevalence and risk factors for *Helicobacter pylori* infection among Yemeni dyspeptic patients. Saudi Med J 2003; 24:512-517.
8. Al-Moagel MA, Evans DG, Abdulghani ME, et al. Prevalence of *Helicobacter* (formerly *Campylobacter*) *pylori* infection in Saudia Arabia, and comparison of those with and without upper gastrointestinal symptoms. Am J Gastroenterol 1990; 85.
9. Akbar DH, Eltahawy AT. *Helicobacter pylori* infection at a university hospital in Saudi Arabia: prevalence, comparison of diagnostic modalities and endoscopic findings. Indian J Pathol Microbiol 2005; 48:181.
10. Abdollah B, Robert W, Remon A, et al. Seroepidemiology of *Helicobacter pylori* infection in a population of Egyptian children. Inter J Epidemiol 2000; 29:928 -932.
11. Ina S, Jose B, Ari S, et al. Prevalence of *Helicobacter pylori* infection and associated factors among adults in Southern Brazil: a population-based cross-sectional Study. BMC Public Health. 2005; 5:1-10.