EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 5
Issue: 8
99-118
Year: 2018

STUDY OF DYSPEPSIA WITH SPECIAL REFERENCE TO HELICOBACTER PYLORI

Dr. Shailendra Pushpkar[1] and Dr. Y. R. Lamture[2*]

[1]Resident, Department of Surgery, JNMC, Sawangi, Wardha, MH, India.
[2]Professor, Department of Surgery, JNMC, Sawangi, Wardha, MH, India.

***Corresponding Author: Dr. Y.R. Lamture**
Department of Surgery. Jawaharlal Nehru Medical College, Sawangi Meghe, Wardha, Maharashtra, India.
Mail ID: dryrlamture@yahoo.co.in Phone (or Mobile) No.: 8308358648

Article Received on 07/06/2018 **Article Revised on 27/06/2018** **Article Accepted on 17/07/2018**

ABSTRACT

Background: Dyspepsia is a common condition that affects more than half of the population at some point during their lifetime. Oesophagogastroduodenoscopy (OGD) is recommended as the first investigation in the workup of a patient with dyspeptic symptoms and it is essential in the classification of the functional dyspepsia or organic causes. It has been shown that gastric *Helicobacter pylori (H. pylori)* infection is associated with dyspeptic symptoms. But cause and relationship has not yet been established beyond doubt. **Aim and Objectives: Aim:** To study the relation between *H. pylori* and dyspepsia.

Objectives:

1. To study oesophagogastroduodenoscopy findings in dyspeptic patients and its relation with *H. pylori.*
2. To study accuracy of rapid urease test.
3. To study the dyspepsia score.
4. To study efficacy of anti-*H. pylori* treatment

Methods: The present study entitled "Study of Dyspepsia with Special Reference to *Helicobacter pylori*" is a hospital based prospective interventional study conducted in Tertiary Health Care Centre Acharya Vinobha Bhave rural hospital attached to Jawaharlal Nehru medical college which comes under Datta Meghe Institute of Medical Science University. One hundred and eight cases inclusive of Outdoor and Indoor patients, presenting in the department of Surgery, Medicine and ENT with functional dyspepsia and have one or more out of three symptoms, epigastric pain or burning, early satiety & post-prandial fullness and willing to undergo endoscopy were enrolled in this study. **Results:** In this study, majority of the dyspeptic patients 48.15% belonged to the age group of 21-40 years. Whereas common age group among *H. pylori* positive patients was 21-40 years. Majority (55.56%) of the subjects were male. The most common endoscopic finding was normal study followed by Antral gastritis. The sensitivity of rapid urease test (RUT) was 100% and specificity of RUT came to be 96% and positive predictive value was 96.66% whereas negative predictive value was 100%. The diagnostic accuracy of RUT was 98.14 %. The frequency of *H. pylori* infection was 55.55% in our study population. In this study the triple drug therapy is the effective treatment for eradication of *H. pylori* infection. There is a significant decrease in dyspepsia score after treatment. The success rate of eradication therapy in our study was 81.67%. **Conclusion:** Oesophagogastroduodenoscopy (OGD) revealed normal finding in 33 out of 108 patients and on remaining 75 patients major abnormal finding found was antral gastritis. *H. pylori* infection was positive in 21.22% of patient with normal finding and all become negative after eradication therapy. Both Rapid Urease Test and Histopathological examination of biopsy specimen had almost similar results to detect *Helicobacter pylori* infection.

KEYWORDS: Dyspepsia, Oesophagogastroduodenoscopy, *H. pylori.*

INTRODUCTION

Dyspepsia is defined as chronic or recurrent pain or discomfort centered in the upper abdomen. Discomfort is defined as a subjective negative feeling that is non-painful and can incorporate a variety of symptoms including early satiety or upper abdominal fullness. Patients presenting with predominant or frequent (more than once a week) heartburn or acid regurgitation should be considered to have gastro esophageal reflux disease (GERD) until proven otherwise.[1] Regardless of its definition, the causes of dyspepsia are known to include peptic ulcer disease, gastro-esophageal reflux, and functional dyspepsia (FD). Functional dyspepsia, otherwise known as non-ulcer dyspepsia, is clearly the commonest cause of dyspeptic symptoms in the West and increasing in other parts of the world.[2] In Rome III guideline states that dyspepsia is non-reflux predominant pain or discomfort in the upper abdomen and the patients

must also have one or more of the following three symptoms: postprandial fullness, early satiety, epigastric pain or epigastric burning and no evidence of structural disease (including at upper endoscopy) that is likely to explain the symptoms Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis.[3]

Dyspepsia is a common condition that affects more than 50% of the population at some point during lifetime. Many studies have proven that the condition is experienced by approximately 20%-40% of the general adult population and accounts for 3%-4% of all consultations in primary care which causes huge economic costs to patients and to the economy. In Japan, India, and Turkey, the prevalence of dyspepsia has been estimated to be 17%, 30.4%, and 28.4%, respectively.[4,6]

Dyspepsia is a symptom and not a diagnosis. Symptoms may last for decades (even lifetime) and remissions and relapses are common. It is one of the commonest gastrointestinal complaint affecting at least 25% of the population during a year. Its prevalence varies in different population and countries, depending upon the prevalence of *Helicobacter pylori* (*H. pylori*) infection, obesity, drug - alcohol - tobacco intake and spices in diet.[7]

The pathophysiology of functional dyspepsia remains poorly understood. Number of potentially important abnormalities have been reported in functional dyspepsia patients, including impaired fundic accommodation, gastric hypersensitivity to distention, abnormal duodenojejunal motility, duodenal motor and sensory dysfunction, duodenal hypersensitivity and *Helicobacter pylori* infection.[8]

Most patients with functional dyspepsia and non-erosive reflux disease (NERD) complain of several symptoms related to meals, however the pathophysiology of these diseases remains poorly defined. 40-60% of patients with FD also have *H. pylori* gastritis[9], but whether *H. pylori* is the cause of the symptoms associated with FD is unclear.[10] Impaired gastric motility is associated with gastric emptying and gastric accommodation and has been implicated in the pathophysiology of functional dyspepsia, Delayed gastric emptying is reportedly present in 25% to 50% of patients with FD.[11]

It has been shown that gastric *H. pylori* infection is associated with dyspeptic symptoms. But cause and effect relationship has not yet been established beyond doubt.[12] Seroprevalence of *H. pylori* is high in developing countries such as India. 80% of Indian adults have antibodies against *H. pylori* in their sera.[12] *H. pylori* is a human pathogen that is transmitted from human to human, and causes chronic active gastritis in all colonized subjects. This can lead to peptic ulcer disease, atrophic gastritis, gastric adenocarcinoma, and MALT (mucosa-associated lymphoid tissue) lymphoma. *H. pylori* eradication therapies cures gastritis and can alter the progression to long-term complications, or recurrence of disease. For these reasons, *Helicobacter pylori* is considered an infectious disease irrespective of an individual's symptoms and stage of disease.[13]

H. pylori is a spiral, gram-negative, microaerophilic bacterium, which was established in 1982 by Robin Warren and Barry Marshall as the causative agent of gastritis and peptic ulcer.[14] Before Warren and Marshall, the human stomach was believed to be a sterile area. *Helicobacter pylori* formerly known as *Campylobacter pyloridis* then *Campylobacter pylori*, is one of the human pathogens with highest prevalence around the world; yet, its exact mode of transmission is still uncertain. This organism was isolated from the human stomach but has not been consistently isolated from any other species, therefore the mechanism by which it colonizes the human stomach remains largely unknown.

Today, *H. pylori* infection is recognized as the most common cause of gastritis, which leads to the development of more gastrointestinal (GI) complications such as peptic and duodenal ulcers. Additionally, the organism is classified as a class -1 carcinogen because of its causal relationship to gastric adenocarcinoma, one of the world's deadliest cancers.[15] *H. pylori* infection can be diagnosed either by invasive tests meaning that they require gastric tissue or mucous, which require endoscopy and gastric biopsy (e.g., rapid urease test) or by noninvasive tests. "Non-invasive" tests requiring only blood, breath or stool analysis (e.g. urea breathe test) all of them exhibiting both advantages and disadvantages regarding availability, rapidity of results, value, and diagnostic accuracy.

Oesophagogastroduodenoscopy (OGD) is the ideal procedure for detecting organic diseases of the foregut, but this procedure is yet to be widely available in developing countries. It has been estimated that approximately 50% of all upper oesophagogastroduodenoscopy referrals are dyspepsia related. One of the widely used invasive tests for dyspepsia is the rapid urease test (RUT). The RUT consists of urea rich agar gel medium with a pH sensitive dye. It is based on the principle that abundant urease enzyme produced by *H. pylori* hydrolyses urea to ammonia and carbon dioxide. The consequent rise in pH of the medium is detected by phenol red indicator. If urease is present in biopsy sample (mucosal) placed in the medium, it results in the hydrolysis of urea. Usually, the site of gastric biopsy used by many endoscopists is the antrum.

Till date, several modifications of Christensen's original urea medium have been developed to achieve quick results and to improve sensitivity and specificity. Although sensitivity and specificity of RUT are generally above 90%, the test results seem to be influenced by the

consumption of several drugs including proton pump inhibitors (PPI), antibiotics, H_2 receptor antagonists, and bismuth. The choice of test depends to a large extent on availability and cost. Other important factors are the clinical situation, population prevalence of infection, pretest probability of infection, differences in test performance, and factors that may influence the test results such as the use of anti-secretory treatment and antibiotics. In some regions where *H. pylori* prevalence is very high, diagnostic tests for the infection are not cost-effective and the decision to treat must then assume the presence of the infection.

Although the clinical picture of acute *H. pylori* infection has been described in detail in few reports, there is still considerable doubt as to whether chronic *H. pylori* infection causes Gastrointestinal symptoms in people without peptic ulcer disease. Some studies have shown that *H. pylori* infection rates are slightly higher in peoples with non-ulcer dyspepsia than in asymptomatic peoples.

Many trials evaluating the role of *H. pylori* in functional dyspepsia and the efficacy of *H. pylori* eradication treatment on the symptomatology of functional dyspepsia have been published in the past, but most of these have given conflicting results.[16]

In studies done Glasbrenner B et at[17] and Kyzekové J et al[18] they reported that in patients with dyspepsia the gastritis or other dyspeptic symptom was strongly associated with *H. pylori* infection. A population-based study in the United Kingdom has investigated that the association of *H. pylori* infection with functional dyspepsia.[19] Whereas Zanten et el[20] reported that *H. pylori* infection does cause any changes in gastric physiology and does not aggravate any of the dyspeptic symptoms. Other studies have, however, failed to demonstrate any relation between *H. pylori* infection and gastrointestinal symptoms and specific *H. pylori* related dyspepsia has not yet been identified.[21] [22]

Chiba N. et al[23] conducted randomized, placebo-controlled trial and found that, eradication therapy proved successful in a subset of patients with nonulcer dyspepsia. However, these findings were not confirmed in another trial of similar study done by Talley NJ et al.[24] This disparity suggests either that the relationship between *H. pylori* and nonulcer dyspepsia is weak or that dyspepsia is a heterogeneous disorder.

Chanyaswad J et al[25] in there study found that The sensitivity, specificity, Positive predictive values (PPV), Negative predictive values (NPV) and diagnostic accuracy of RUT was 57.9, 94.2, 67.9 and 74.7 which is very low compared to study done by Mojgan Foroutan et al[26] which was 97.37, 98.57 and 98.14%, respectively.

Talley et al[10] found that success of the anti *H. pylori* therapy was 24% that is in total 135 patient 32 patients were treated, In a study by Gilvarry J. et al[27] in there study they reported that the eradication of *H. pylori* was 85%.

Considering this confusing scenario regarding the role of *H. pylori* causing dyspepsia, efficacy of anti *H. pylori* treatment and accuracy of RUT test this study was conducted.

Thus the aim of this study is to determine the role of *H. pylori* in dyspepsia and efficacy of anti *H. pylori* therapy. This study would assist clinicians in choosing an optimal diagnostic and management approach to dyspeptic patients. It will also provide valuable information for similar studies in the future.

AIM AND OBJECTIVES

Aim

To study the relation between H. pylori and dyspepsia.

OBJECTIVES

1. To study oesophagogastroduodenoscopy findings in dyspeptic patients and its relation with H. pylori.
2. To study accuracy of rapid urease test.
3. To study the dyspepsia score.
4. To study efficacy of anti-H. pylori treatment.

MATERIAL AND METHODS

Study Setting

This study was conducted on patients presenting with functional dyspepsia at Acharya Vinoba Bhave Rural Hospital, Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha,

Study period: from 1st September 2015 to 31st august 2017.

Study Design: Prospective interventional study.

Participants, inclusion and exclusion criteria

In this study, we included outdoor and indoor patients visiting to this hospital. Patients presented with functional dyspepsia were included in the study. Functional dyspepsia is defined as "non-reflux predominant pain or discomfort in the upper abdomen and the patients must also have one or more of the following three symptoms: postprandial fullness, early satiety and epigastric pain or burning without evidence of structural disease".

Detailed symptomatology of all the patients was recorded. The symptoms were graded on the basis of frequency and severity, using visual analogue scale.[59] The total dyspepsia score was calculated for each patient by adding the scores for individual symptoms. All Patient undergoes ultrasonography (USG) abdomen to rule out other organic causes of dyspepsia like cholicystitis and appendicitis and if present they were excluded from study.

We obtained their informed consent and performed their oesophagogastroduodenoscopy (OGD). OGD was done by consultant in the department.

During OGD, endoscopic guided two antral biopsy specimens were collected from each patient, one antral biopsy specimens was subjected to Rapid Urease Test for detection of *H. pylori* and other was sent for histopathology evaluation. After endoscopy, endoscopes and biopsy forceps were sterilized with 2% gluteraldehyde for 30 minutes to prevent cross infection among patients.

In selected cases such as suspected malignancy, at least 4 biopsy specimens are recommended to be preserved in 10% formalin, labeled appropriately and dispatched for histological examination.

We divide these patients in 2 groups, first *H. pylori* positive, second group *H. pylori* negative. First group received treatment for *H. pylori* consisting of Amoxycillin 750 mg, Clarithromycin 250 mg and Omeprazole 20 mg twice daily for 14 days. The second group received treatment depending on clinical examination and finding of oesophagogastroduodenoscopy. This 14 days therapy is based on studies reported by J. Gilvarry et al[27] and S Arama et al 2016[60] with 14-day anti-HP regimen offered better *H. pylori* eradication compared to a similar, 7-day regimen.

These patients were clinically re-evaluated at an interval of one month and subjected for repeat endoscopy with antral biopsy to know status of *H. pylori* and to access improvement in total dyspepsia score.

Inclusion Criteria for the Study

1- Patient visited to AVBRH for dyspepsia and willing to undergo endoscopic evaluation.

Exclusion Criteria

1. Patients having organic diseases, leading to dyspepsia like symptom (Gall Bladder diseases, Appendicitis, Hiatus hernia).
2. Patients having history of chronic alcoholism, chronic smoking, use of steroids, PPI and NSAIDs.
3. Pregnancy.
4. Patient with Symptom having duration less than 3 month

The detailed history of all the patients was collected inclusive of their IPD No, name, age, sex, duration of complains (epigastric pain or burning, early satiety & post-prandial fullness) for at least more than 3 month, addiction (tobacco chewing and smoking history) and history of intake of steroid or NSAID.

After history and clinical examination, patient undergoes USG abdomen to rule out other organic causes of dyspepsia like cholicystitis and appendicitis which was done by consultant's radiologist in radiology department. After ultrasonography (USG) abdomen all the eligible patients were subjected to Upper GI Endoscopy/ oesophagogastroduodenoscopy (OGD) evaluation done by consultants having endoscopic expertise. Biopsy from antral part of the stomach from each patient was subjected for RUT test and histopathological examination to detect *H. pylori*. The oesophagogastroduodenoscopy was performed by Fujinon oesophagogastroduodenoscopy unit as follows:-

- Patient were asked to be nil by mouth (NBM) a day prior to the procedure.
- The procedure was explained to the patient and the attendant in their own language including possibility of biopsy or other related radiological/surgical interventions.
- Patients were asked to sign the consent form agreeing to the procedure and high risk consent was taken in the high risk group.
- They were also asked regarding the medications, any allergies in past.
- Patients were then asked to remove artificial denture, eye wares, if any, prior to the procedure.
- Local anesthesia spray (10% xylocaine) was given on posterior pharyngeal wall to prevent the gag reflex.
- Patients were asked to lie down in left lateral position and the plastic mouth guard was held between the teeth by the assistant to keep the mouth open and make it easier to pass endoscope.
- After lubricating the front part of the endoscope with 2% xylocaine jelly and adjusting the white contrast it was passed through the mouth guard and patient was asked to swallow it.
- Endoscope was guided under direct visualization through upper esophageal sphincter to the stomach.
- During this procedure, inspection of esophagus, stomach, pylorus, and first and second part of duodenum was done on the monitor and use of suction irrigation and insufflation was done whenever necessary.
- Reinspection was done while withdrawing the scope and with retroflection fundus and cardiac end of stomach was inspected.
- Procedures like biopsies, removal of polyps or foreign body was performed wherever indicated.
- During all this procedure, vital parameter of the patients and SpO2 (peripheral capillary oxygen saturation) were monitored with multipara.
- Patients were advised to take orally after one hour of the procedure when not contraindicated.

During oesophagogastroduodenoscopy two gastric biopsies sample of size 3 to 5 mm were obtained from the antrum of stomach using biopsy forceps first sample was immediately transferred to Rapid Urease Test dry kit (RUT) for detection of *Helicobacter pylori*. The second sample is sent for histopathlogy examination. This biopsy specimens is preserved in 10% formalin, labeled

with Name, Age/Sex, Hospital registration No. with date of examination.

A biopsy sample is taken from the antrum of the stomach, and is placed into a medium containing urea(as in RUT dry kit), the back of the RUT is peeled and biopsy was introduce in the exposed yellow part to media and adding one drop of distilled water then after covering with sticker as before. The color changes from yellow to pink or red then the test is called positive. Reading of the kit is done after 5 minute to 10 minute.(interval time is 4 Hour).

The urease produced by *H. pylori* hydrolyzes urea to ammonia, which raises the pH of the medium, and changes the color of the specimen from yellow (NEGATIVE) to red or pink (POSITIVE).

The biopsies taken from doubtful lesions were sent to department of Pathology for Histological examination.

OBSERVATION AND RESULTS

Table 1: Age wise distribution of patients in our study.

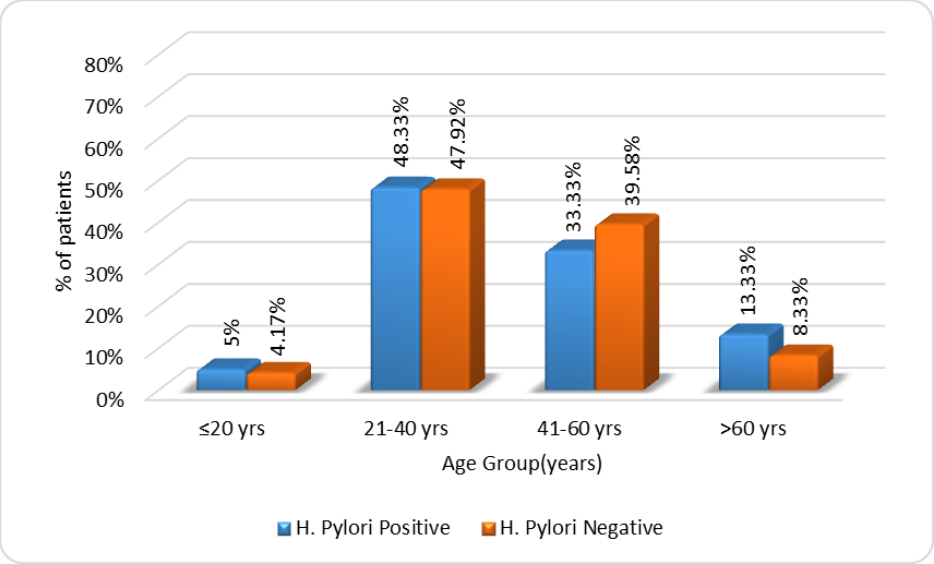
Age Group (years)	Total	*H. pylori* Positive	*H. pylori* Negative
≤20 years	5 (4.63%)	3 (5%)	2 (4.17%)
21-40 years	52 (48.15%)	29 (48.33%)	23 (47.92%)
41-60 years	39 (36.11%)	20 (33.33%)	19 (39.58%)
>60 years	12 (11.11%)	8 (13.33%)	4 (8.33%)
Total	**108 (100%)**	**60 (100%)**	**48 (100%)**
Mean ± SD	41.04±13.69	40.73±14.32	41.43±13.00
Range	16 - 68 years	17 - 70 years	16 - 68 years

Out of 108 patients studied, maximum patients (52 patients) were in the age group of 21 – 40 years, whereas 39 patients were in the age group of 41-60 years, 12 patients were of age more than 60 years and only 5 patients were below 20 years of age.

Twenty nine patients out of 52 were positive for *H. pylori* in the age group of 21-40 years, followed by 20 patients out of 39 in age group of 41-60 years, 8 out of 12 patients in age group >60 years and 3 out of 5 patients ≤ 20 years were positive for *H. pylori*.

The mean age of patients in *H. pylori* positive group was 40.73 years with a standard deviation of ±14.32 years and in *H. pylori* negative group mean age was 41.43 years with a standard deviation of ±13.00 years.

The mean age of this study was 41.04 years with standard deviation of ±13.69 years.



Graph 1: Age wise distribution of patients.

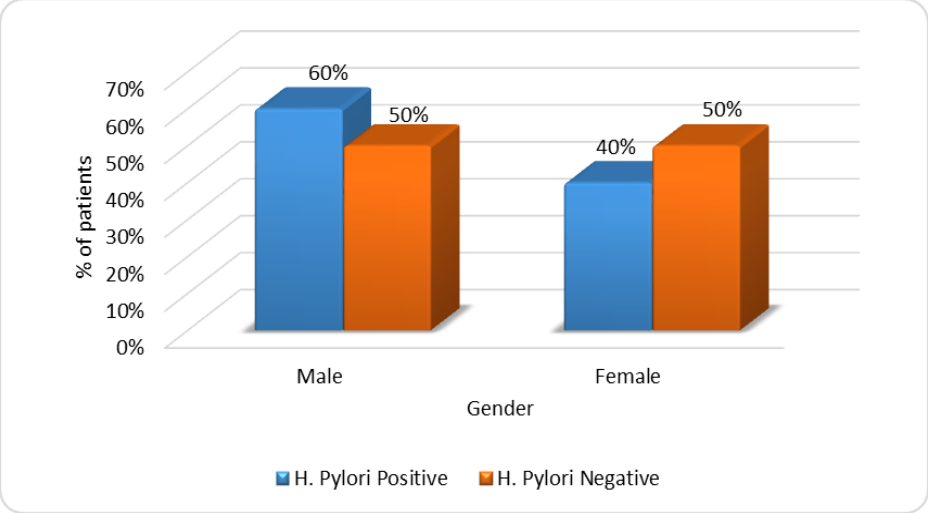
Table 2: Gender wise distribution of patients.

Gender	Total	*H. pylori* Positive	*H. pylori* Negative	p-value
Male	60(55.56%)	36(60%)	24(50%)	0.29,NS
Female	48(44.44%)	24(40%)	24(50%)	
Total	**108(100%)**	**60(100%)**	**48(100%)**	

Among 108 patients studied, 60 were males and 48 were females and M/F ratio in the present study was 1.25:1.

In *H. pylori* positive group out of 60 patients 36 were males and 24 were females and the M/F ratio was 1.5:1 and in *H. pylori* negative group out of 48 patients, 24 were males & 24 were females so the M/F ratio was 1:1.

Out of 60(55.56%) male patients, 36(60%) were found to be *H. pylori* positive, whereas among 48(44.44%) female patients, 24(40%) were positive for *H. pylori*.

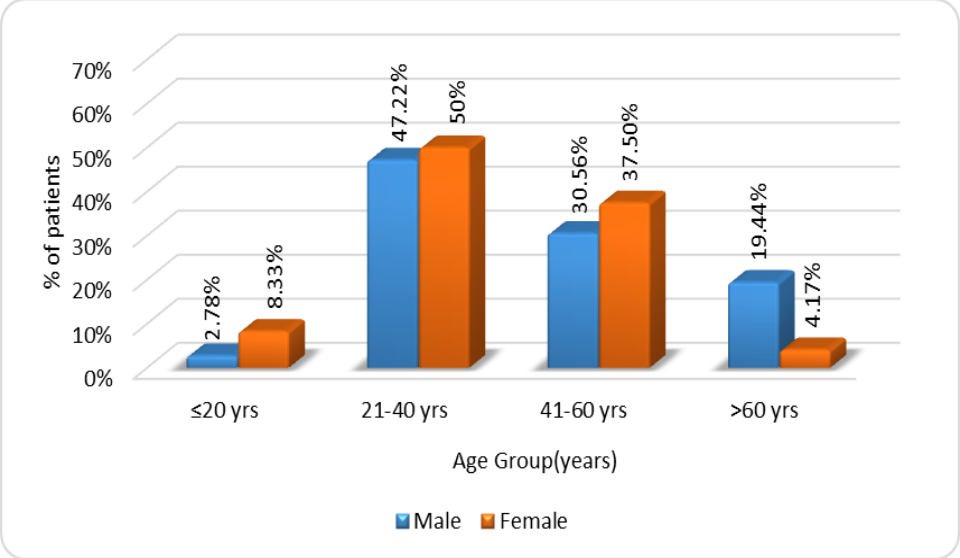


Graph 2: Gender wise distribution of patients.

Table 3: Age wise and Gender wise distribution of patients in our study.

Age Group(years)	Total	Male	Female	p-value
≤20 years	3(5%)	1(2.78%)	2(8.33%)	0.30,NS
21-40 years	29(48.33%)	17(47.22%)	12(50%)	
41-60 years	20(33.33%)	11(30.56%)	9(37.50%)	
>60 years	8(13.33%)	7(19.44%)	1(4.17%)	
Total	**60(100%)**	**36(100%)**	**24(100%)**	
Mean ±SD	40.73±14.32	41.66±15.70	39.33±12.14	
Range	17-70 years	20-70 years	17-65 years	

Majority of the patients 29 belonged to the age group of 21-40 years in which 17 male and 12 were females, followed by 20 patients belongs to the age group of 41-60 years, which includes 11 male and 9 female. 8 patients were above 60 years, which includes 7 male and 1 female, and 3 belonged to age group of ≤20 years which include 1 male and 2 female patients.



Graph 3: Age wise and Gender wise distribution of patients.

Table 4: Distribution of patients according to endoscopic findings and RUT result in our study.

Endoscopic Findings	Total	*H. pylori* Positive (RUT positive)	*H. pylori* Negative (RUT negative)
Acute Erosive Gastritis	2(1.85%)	1(1.67%)	1(2.08%)
Acute Erosive Gastritis with duodenitis	1(0.93%)	1(1.67%)	0(0.00%)
Antral Gastritis	29(26.85%)	18(30.00%)	11(22.92%)
Oesophagitis with Antral gastritis	19(17.59%)	14(23.33%)	5(10.42%)
Duodenal Ulcer	2(1.85%)	2(3.33%)	0(0.00%)
Oesophagitis with gastric polyp	3(2.78%)	1(1.67%)	2(4.17%)
Pangastritis	16(14.81%)	13(21.67%)	3(6.25%)
Pangastritis with duodenitis	3(2.78%)	3(5.00%)	0(0.00%)
Normal study	33(30.56%)	7(11.67%)	26(54.17%)
Total	**108(100%)**	**60(100%)**	**48(100%)**

On oesophagogastroduodenoscopy (OGD) 33 cases (30.56%) had normal study of which 7 (11.67%) cases were positive for *H. pylori*.

29 cases (26.85%) were diagnosed as antral gastritis of which 18 (30%) were positive for *H. pylori*.

Oesophagogastroduodenoscopy finding of 19 patients (17.59%) was suggestive of Oesophagitis with antral gastritis of which 14 (23.33%) were positive for *H. pylori*.

Out of 16 (14.81%) cases of pangastritis, 13 (21.67%) were positive for *H. pylori*.

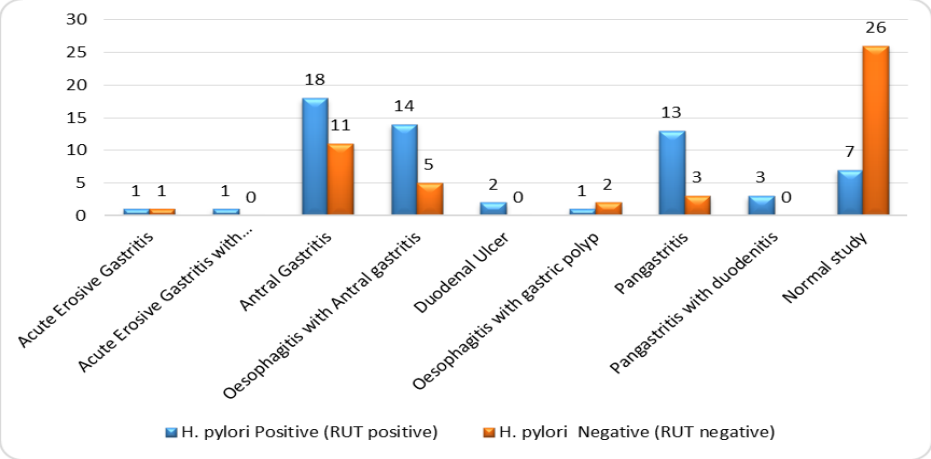
3 cases (2.78%) were diagnosed as pangastritis with duodenitis and all were positive for *H. pylori*.

On oesophagogastroduodenoscopy 3 cases(2.78%) had oesophagitis with gastric polyp of which 1 (1.67%) was positive for *H. pylori*.

Duodenal ulcer was found in 2 patient (1.85%) and both were positive for *H. pylori.*

Acute Erosive Gastritis was found in 2 cases and 1 was positive for *H. pylori*.

1 case shows Acute Erosive Gastritis with duodenitis and was positive for *H. pylori.*



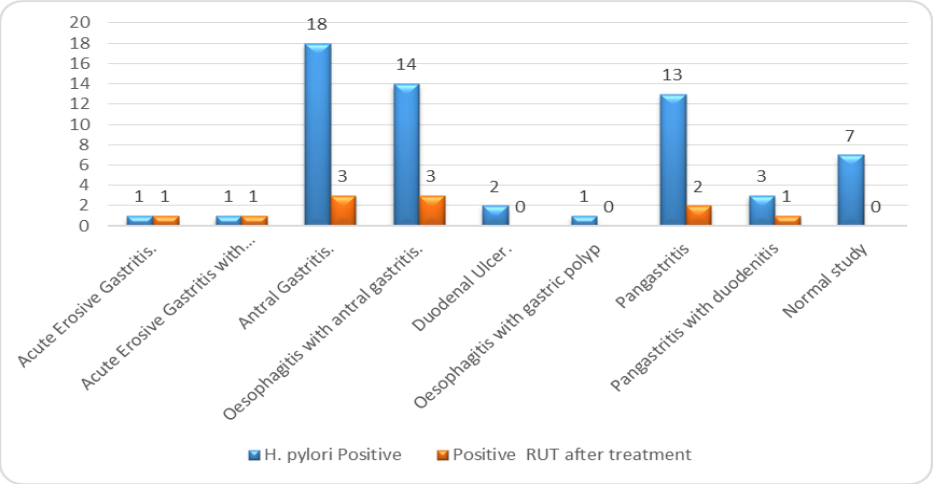
Graph 4: Distribution of patients according to endoscopic findings and RUT result in our study.

Table 5: Comparison of pre and post treatment endoscopic findings in *H. pylori* positive patients with their success rate.

Endoscopic Findings	*H. pylori* Positive	Positive RUT after treatment	Success rate of eradication of *H. pylori.*
Acute Erosive Gastritis.	1(1.67%)	1(9.09%)	0%
Acute Erosive Gastritis with duodenitis.	1(1.67%)	1(9.09%)	0%
Antral Gastritis.	18(30.00%)	3(27.27%)	83.33%
Oesophagitis with antral gastritis.	14(23.33%)	3(27.27%)	78.76%
Duodenal Ulcer.	2(3.33%)	0(0.0%)	100%
Oesophagitis with gastric polyp	1(1.67%)	0(0.00%)	100%
Pangastritis	13(21.67%)	2(18.18%)	84.67%
Pangastritis with duodenitis	3(5.00%)	1(9.09%)	66.67%
Normal study	7(11.67%)	0(0.00)	100%
Total	**60(100%)**	**11(100%)**	

Out of 60 *H. pylori* positive patients who receive anti *H. pylori* treatment, 11 patients remain positive post-treatment of which commonest finding was in 3 patients, which includes Antral Gastritis and 3 patients were of Oesophagitis with Antral gastritis, followed by 2 patients of Pangastritis.

The success rate of eradication of *H. pylori* by anti-*H. pylori* treatment was found 100% in Normal study, 100% in Oesophagitis with gastric polyp and 100% in patient with duodenal ulcer, 83.33% in Antral gastritis, 78.76% in Oesophagitis with antral gastritis and 0% in cases of Acute Erosive Gastritis and Acute Erosive Gastritis with duodenitis.



Graph 5: Comparison of pre and post treatment endoscopic findings in *H. pylori* positive patients with their success rate.

Table 6: Comparison of patients according to RUT and Histopathology result.

	RUT	Histopathology Result.	χ2-value	t-value
Positive For *H. pylori*	60	58	0.0747	*P*=0.78,NS
Negative For *H. pylori*	48	50		
TOTAL	108	108		

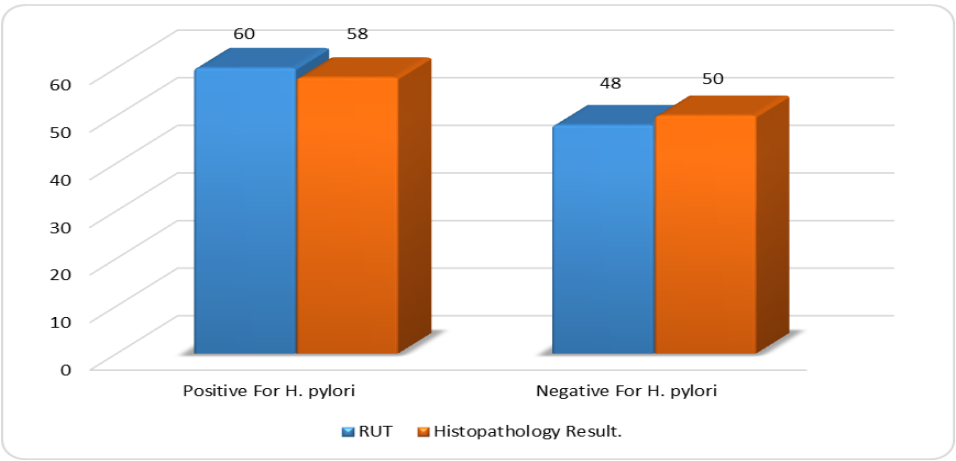
Among all endoscopic biopsy samples, rapid urease test was positive in 60 patients and 58 patients were positive on histopathology for *H. pylori.*

- True positive : 58 patients
- False negative : 0 patients
- False positive : 2 patients
- True negative : 48 patients

When compared with histopathology, the Sensitivity of RUT was 100% and Specificity of RUT came to be 96%.

The Positive predictive value was 96.66% whereas Negative predictive value came to be 100%.

- Sensitivity=100%(95% CI=93.84-100%)
- Specificity=96%(95% CI=86.29-99.51%)
- Positive Predictive Value=96.67% (CI=88.47-99.59%)
- Negative Predictive Value=100% (95% CI=92.60-100%)
- Diagnostic Accuracy=98.14%

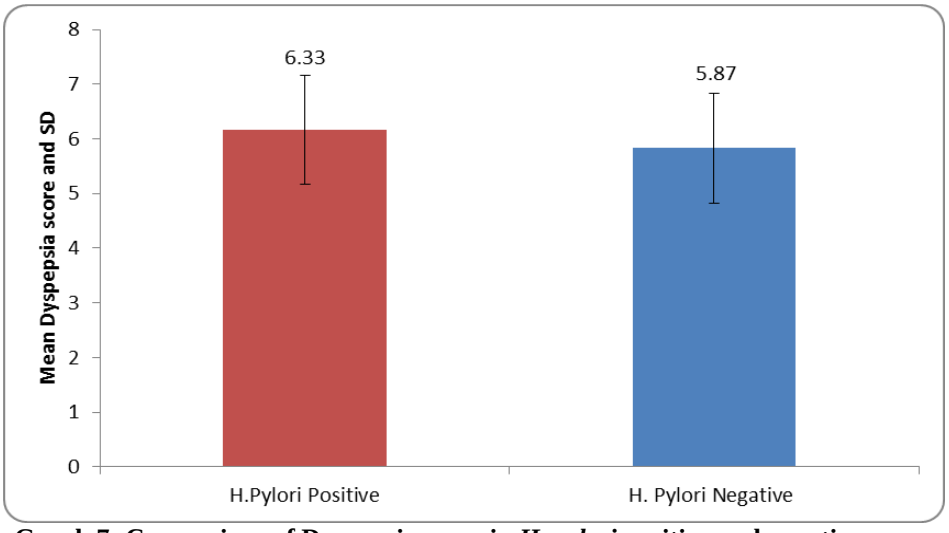


Graph 6: Comparison of patients according to RUT and Histopathology result.

Table 7: Comparison of Dyspepsia score in *H. pylori* positive and negative cases.
Student's paired t test.

	N (= no of patients)	Mean	Std. Deviation	Std. Error Mean	t-value
H. pylori Positive	60	6.33	1.48	0.19	1.53 p=0.12,NS
H. pylori Negative	48	5.87	1.61	0.23	

Mean dyspepsia score in *H. pylori* positive patients was 6.33 ± 1.48 and in *H. pylori* negative patients the mean dyspepsia score was 5.87 ± 1.61 and there was no significant difference found on mean dyspepsia score in both groups (p value = 0.12).

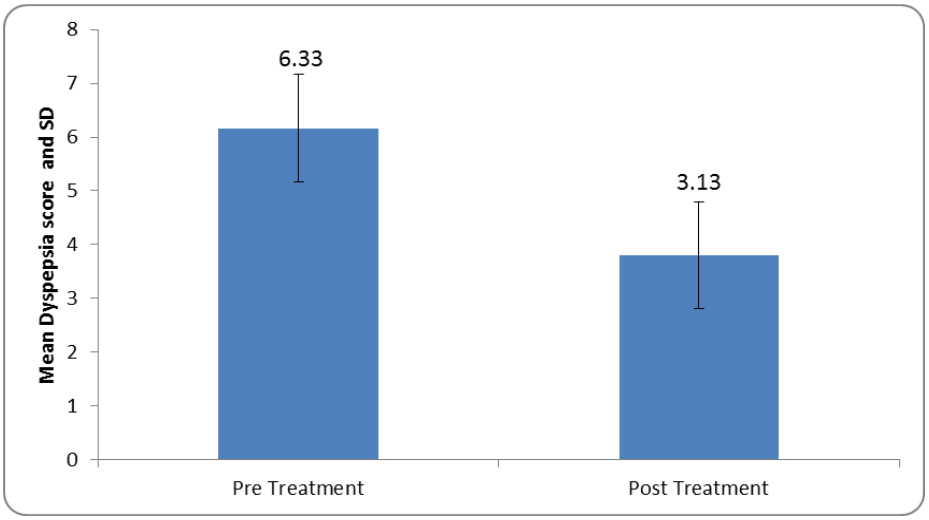


Graph 7: Comparison of Dyspepsia score in *H. pylori* positive and negative cases.

Table 8: Comparison of Mean Dyspepsia score in *H. pylori* positive case pretreatment and post treatment.
Student's paired t test

	N (= no of patients)	Mean	Std. Deviation	Std. Error Mean	t-value
Pre Treatment	60	6.33	1.48	0.19	14.62 p=0.0001,S
Post Treatment	60	3.13	1.96	0.25	

Out of 60 *H. pylori* positive patients mean dyspepsia score pretreatment was 6.33 with standard deviation of 1.48 and post treatment, mean dyspepsia score was 3.13 with standard deviation of 1.96 and this difference was statistically significant (p=0.0001).

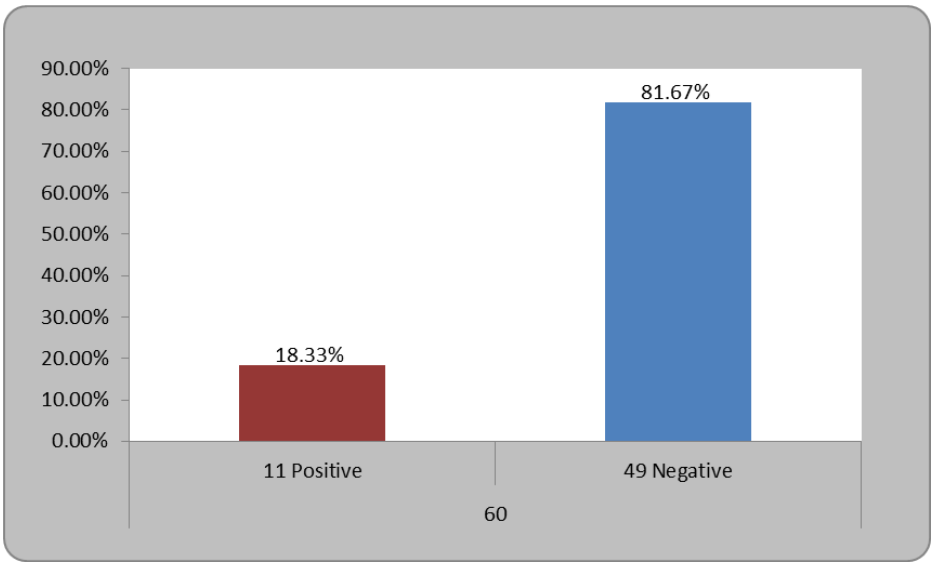


Graph 8: Comparison of Mean Dyspepsia score in H. pylori positive case pretreatment and post treatment.

Table 9: Result of rapid urease test in *H. pylori* positive patients after 1 month of anti *H. pylori* treatment.

Total No of *H. pylori* positive patient before treatment	Rapid urea test status After 1 month of anti *H. pylori* treatment.	Percentage (%)
60	11 Positive	18.33%
	49 Negative	81.67%

60 patients were *H. pylori* positive and received anti *H. pylori* treatment of which 49 patients (81.67%) showed complete eradication of *H. pylori* whereas 11 patients(18.33%) still remained positive for *H. pylori* on RUT.

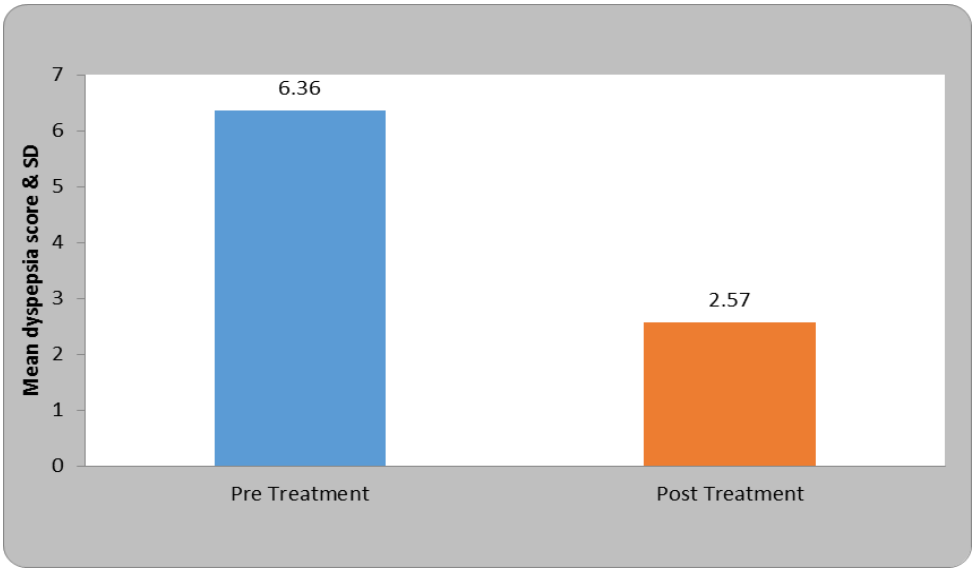


Graph 9: Result of rapid urease test in *H. pylori* positive patients after 1 month of anti *H. pylori* treatment.

Table 10: Comparison of dyspepsia score in sensitive patients after eradication treatment.

	Mean dyspepsia score	N	Std. Deviation	t-value	p-value
Pre Treatment	6.36	49	1.39	16.67	0.0001,S
Post Treatment	2.57	49	1.58		

Mean dyspepsia score pre treatment was 6.36 with standard deviation of 1.39 and post treatment mean dyspepsia score was 2.57 with standard deviation of 1.58 in patients sensitive to *H. pylori* treatment and this difference was statistically significant (p=0.0001).

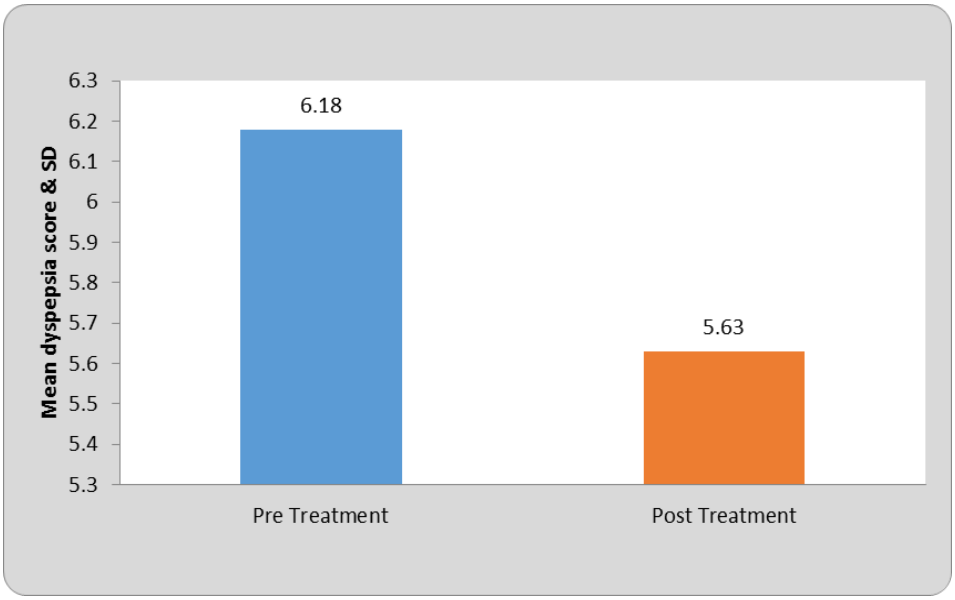


Graph 10: Comparison of dyspepsia score in sensitive patients after eradication treatment.

Table 11: Comparison of dyspepsia score in patients resistant to anti *H. pylori* treatment.

	Mean dyspepsia score	N	Std. Deviation	t-value	p-value
Pre Treatment	6.18	11	1.88	1.93	0.082,NS
Post Treatment	5.63	11	1.96		

Mean dyspepsia score pretreatment was 6.18 with standard deviation of 1.88 and post treatment mean dyspepsia score was 5.63 with standard deviation of 1.96 in patients resistant to anti *H. pylori* treatment and this difference was statistically non-significant (p=0.082).



Graph 11: Comparison of dyspepsia score in patients resistant to anti *H. pylori* treatment.

DISCUSSION

Age distribution

In the present study, 108 patients were included; the maximum patients were in the age group of 21-40 years that is 52 in number. Mean age of all dyspeptic patients in this study was 41.04years with standard deviation of ±13.69 years. Mean age of patients in *H. pylori* positive group was 40.73 years with a standard deviation of ±14.32 years and in *H. pylori* negative group mean age was 41.43 years with a standard deviation of ±13.00 years.

In studies done by Bashir T et al (2014)[28] and Sodhi et al (2013)[29], it was found that there was a significant increase in dyspepsia among the 30-55 years of age group.

Okello, T R et al(2015)[30] conducted study observed that dyspepsia was more seen in age group of 36-65yr followed by 19-35yrs.

A study conducted by Mohammed M (2014)[31] a total of 100 dyspeptic patients were taken in age ranged of 20 - 49 years with a mean of 34.2 ± 8.5 years which is similar to present study.

In our study younger population is more affected than the above mentioned study.

Author	Year	place	Mean age group
Olokoba AB et al[32]	2013	Nigeria	35.3 Years
Bashir T et al[33]	2014	Pakistan	39.31 Years
Blum A.L.et al[34]	2008	Global	47 years
Sodhi et al[29]	2013	India	44.5 years
Shrestha S et al[35]	2012	Nepal	20.12 Years
Miwa et al[36]	2000	japan	51.5 years
In present study	2017	India	41.04

Sex distribution

In our study, out of 108 dyspeptic patients majority were male accounting 60, while there were 48 female. The Male/Female ratio was 1.25:1 which shows the male predominance.

In *H. pylori* positive group 36 were males and 24 were females. So the M/F ratio in positive group was 1.5:1. In *H. pylori* negative group there were 24 male and 24 female so male female ratio was 1:1.

In a study conducted by Shrestha S et al in (2012)[35] the total 319 dyspeptic patients were studied of which 161 were male and 152 were female. They found that the mean age was 20.12years and M/F ratio was 1.05:1 hence shows male predominance and it correlates with our study.

In a similar study by Olokoba Ab et al (2013)[32] studied 125 dyspeptic patients and recorded that 49(39.2%) were males while 76(60.8%) were females, giving a male to female ratio of 1:1.6. Their ages ranged between 18 and 84 years with a mean age of 35.3± 12.7 years. which is not correlating with our study.

Author	Year	Place	No of patients (M/F)
Olokoba AB et al[32]	2013	Nigeria	125 (49/76)
Bashir T et al[33]	2014	Pakistan	116 (76/40)
Blum A.L.et al[34]	1998	Global	328(136/192)
Sodhi et al[29]	2013	India	519(169/350)
Shrestha S et al[35]	2012	Nepal	319 (161/152)
Miwa et al[36]	2000	Japan	85 (40/45)
In present study	2017	India	108(60/48)

Age and gender wise distribution

In our study majority of the patients, 29(48.33%) belonged to the age group of 21-40 years in which 17 were male and 12 were females.

Twenty (33.33%) belonged to the age group of 41-60 years, 11 were male and 9 were female**,** 8(13.33%) patient were in age group of 60 years of which 7 were male and 1 was females, and only 3 (5%) belonged to the age group of ≤20 years of which 1 was male and 2 were females.

In our study, common age group among *H. pylori* positive patient was 21-40 years and it includes 29 patients, followed by 20 patients in age group of 41-60 years, and while in negative group maximum patients were 23 belonged to age group of 21-40 years followed by 19 patients of age group 41-60 years.

A study conducted by Bashir T et al(2014)[28] in which 116 patient were included and majority 82 patients were between the age group 31– 45years. In their study 76(65.5%) patients were male and 40 (34.5%) were females. It is correlating with our study that is young adult (18-40 years) male are more infected.

Gurjeet et al (2012)[37] and Adlekha et al[38] reported that gender and age were not significant associated factors for *H. pylori* infection.

Adlekha et al[38] studied and found that *H. pylori* infection was diagnosed in 62.0% (329/530) of patients screened. There was no statistically significant difference in sex and age related distribution of *H. pylori* infection.

Frequency of *H. pylori* in dyspeptic patients

In our study, 108 dyspeptic patients were included and 60 were RUT positive.

Frequency of *H. pylori* infection in our study was 55.56%.

A study by Jemilohun A et al 2009[39] conducted a study in 86 dyspeptic patients and found the frequency of *H. pylori was 64%.*

Faintuch JJ et al(2014)[40] conducted a study for endoscopic findings in uninvestigated dyspepsia among 306 patients and the overall frequency of *H. pylori* in their study was 45.3%.

Yangchun Zhu et al (2011)[41] studied 5417 individuals in yangzhong city, China and found that 3435 (63.41%) were *H. pylori* positive.

In a similar study by Olokoba Ab et al (2013)[32] studied 125 dyspeptic patients with oesophagogastroduodenoscopy (OGD) guided biopsies. In their study *H. pylori* was detected in 80.0% of the histological samples. When compared to serological test the presence of *H. pylori* was 93.6%.

In a study of Shrestha S et al (2012)(35) of 319 dyspeptic patients was examined in which *H. pylori* was found in 67 (44%) patients.

Author	Year	*Frequency Of H. pylori*
Jemilohun A et al[39]	2009	64%
Faintuch JJ et al[40]	2014	45.3%
Yangchun Zhu et al[41]	2011	63.41%
Shrestha S et al[35]	2012	44 %
Olokoba Ab et al[32]	2013	80.0%
In present study	2017	55.56%

In a study conducted by Mbengue M et al (1997)[42] and found that *Helicobacter pylori* infection was detected in 82.8% of patients of 134 patients.

In our study out of 60 positive patients, 36 were male and 24 were female patients so the frequency in male was 60% and in female patients came to be 40%.

Shrestha S et al[35] studied total of 161 patients, and found frequency of *H. pylori* in male was 58% and 42% in female and concluded that the infection by *H. pylori* was significantly higher in males than females.

Oesophagogastroduodenoscopy (OGD) findings in dyspeptic patients and its relation with *H. pylori*

In our study the OGD finding was normal in 33 cases (30.56%), of which 7 cases were positive for *H. pylori* by RUT.

On OGD, Normal study was found in maximum number of patients that is 33 cases (30.56%).

Other abnormal findings were antral gastritis in 29 cases (26.85%) of which 18 were positive for *H. pylori* on RUT, Endoscopic finding of 19 (17.59%) patients was suggestive of oesophagitis with antral gastritis of which 14 were positive for *H. pylori*, there were 16(14.81%) cases of pangastritis and 13 were positive for *H. pylori*. 3(2.78%) case were diagnosed as pangastritis with duodenitis and all were positive for *H. pylori*. On oesophagogastroduodenoscopy 3(2.78%) cases had oesophagitis with gastric polyp of which 1 was positive for *H. pylori*, duodenal ulcer was found in 2(1.85%) patient and both were positive for *H. pylori*. Acute erosive gastritis was found in 2 (1.85%) cases and 1 was positive for *H. pylori*. 1(0.93%) case shows acute erosive gastritis with duodenitis, which was positive for *H. pylori*.

In our study no resistant case was found with normal finding on OGD. All 7 positive cases turn negative after anti *H. pylori* treatment.

Eighteen patients of Antral gastritis were found positive for *H. pylori* of which 3 patients were resistant for anti *H. pylori* treatment.

Out of 14 positive patients of oesophagitis with antral gastritis 3 were resistant for anti *H. pylori* treatment.

Thirteen cases of pangastritis were positive for *H. pylori* and after treatment 2 remain positive.

Three positive cases with pangastritis with duodenitis after treatment 1 remain positive for *H. pylori*.

Oesophagitis with gastric polyp with *H. pylori* positive was found in 1 patient and which came negative after treatment

One patient with acute erosive gastritis and duodenitis was positive for *H. pylori* remains positive after treatment.

One patient was diagnosed as *H. pylori* positive with pangastritis with duodenitis which remain positive after treatment.

In a study of Shrestha et al (2012)[35], 319 dyspeptic patients were studied. The most common endoscopic finding was gastritis (47.6%) followed by normal finding in 57 patients (17.87%). In our study, the normal finding is more and gastritis finding is less than this study so it is not correlated with our study. In cases of Gastritis, *H. pylori* was observed in 67 (44%) cases. 18 out of 57 cases (5.6%) were of Normal finding showed *H. pylori* and also all 3 cases of gastric cancer were positive for *H. pylori* infection. Duodenitis was present in 10 cases of which 40.0% were positive for *H. pylori* and 86.0% of the total Reflux Oesophagitis patients were *H. pylori* infected.

Faintuch JJ et al (2011)[40] conducted a study for endoscopic findings in uninvestigated dyspepsia among 306 patients and analyzed 282 subjects in the study and found Gastritis as endoscopic finding in 46% of cases. In 20% of the patient had normal endoscopic finding, 18% had GERD and 13% had Ulcers (duodenal in 9% and gastric in 4%). These result was not correlating with our study.

A study done by Jemilohun A et al 2009.[39] The most common abnormality at endoscopy was gastritis which was seen in 52 (60.5%) patients, followed by duodenitis 16(18.6%) and doudenogastric reflux 14(16.3%). Gastric ulcer (GU) was recorded in 8(9.3%) patients, 7(8.1%) patients had oesophagitis, while 3(3.5%) and 2(2.3%) patients had gastric cancer and duodenal ulcer (DU) respectively.

In a study by Mbengue M et al (1997).[42] *H. pylori* was identified in 76.2% of patients with normal endoscopic findings and in 100% presenting ulcers, erosions, or gastritis.

Jamil O et al 2013[43] studied 101 patients Positive endoscopic findings were gastritis in 48.5% and esophagitis in 21.8% patients.

Okello TR et al[30] studied 605 patients, and found gastritis (47.9%) to be the commonest condition followed by esophagitis (14.4%), cancer esophagus (5.1%), esophageal varicose (4%) and PUD (2.3%).

Mihara M et al[44] conducted study and concluded that among 642, 391(60.9%) patients were diagnosed as having gastritis, 251 patients with endoscopically normal finding.

Dhaliwal us et al 1997[45] visualized findings of the upper gastro-intestinal endoscopy performed on 100 dyspeptic patients and found that Reflux Oesophagitis was found in 43 cases having grade-1 Oesophagitis in 48.84%, grade-2 in 32.56%, grade-3 in 14.00% and grade-4 in 4.60% cases.

Korkmaz H et al[46] reported the endoscopic finding of 198 dyspeptic patients as esophagitis in 2%, gastric ulcer in 5%, duodenal ulcer in 2.5%, pangastritis in 5%, antral gastritis in 65.6%, atrophic gastritis in 2.5%, gastroduodenitis in 7%, gastritis and esophagitis together in 2%, gastric ulcer and duodenal ulcer together in 1%.

Accuracy of rapid urease test

Out of 108 dyspeptic patient, 60 patient were positive for RUT on endoscopic biopsy whereas 58 patients were histopathologically positive for *H. pylori*. 2 patients who were positive on RUT were found to be negative for *H. pylori* on histopathology.

Forty eight cases were negative for *H. pylori* on both RUT as well as histopathology. Considering histopathology as a gold standard method for the diagnosis of *H. pylori* infection, the sensitivity of RUT was 100%(95% CI=93.84-100%) and specificity of RUT came to be 96%(95% CI=86.29-99.51%) and positive predictive value was 96.67%(CI=88.47-99.59%) whereas negative predictive value was 100%(95% CI=92.60-100%), the diagnostic accuracy of RUT was 98.14%.

Various studies shows that the sensitivity and specificity of rapid urease test (RUT) and histological examination from gastric mucosal biopsies are almost similar.[48] [50]

Study done by Logan R. et al[47] and Lerang F et al[48] concluded that the sensitivity and specificity of RUT was 80–95% and 90–100%, respectively, Histological examination has a sensitivity of 83–95% and a specificity of 90–100%.

Diagnostic accuracy of RUT test in this study was 98% and when compared with histopathology there was no statistically difference in diagnostic accuracy hence we have used RUT to detect *H. pylori* in our study.

Though the accuracy is slightly higher in histopathology we preferred rapid urease test(RUT) in our study, because it gives quick result and there was no statistically significant difference in both diagnostic tools in studies done by various authors.[62][61][49]

AF Syam et al[61] out of 56 histopathology *H. pylori* positive cases, 39 patients were positive with dry rapid urease test and 17 patients were negative with dry rapid urease test. Overall sensitivity and specificity of dry rapid urease test were 69.7% and 95.7% respectively. Positive predictive value was 66.1% and negative predictive value was 96.4% and overall accurate rate was 92.9%. These results were lower than the present study.

Khean Lee et al[50] reported that sensitivity of the rapid urease test was 98.2%, specificity was 99.0%, positive predictive value came to be 99.0%, negative predictive value was 97.9% and the overall diagnostic accuracy for the rapid urease test was 98.5%. The result of these study correlate with our study.

Mojgan foroutan et al[26] concluded that among NSAID users, RUT sensitivity, specificity and accuracy rate were all 100%. The sensitivity, specificity and accuracy rate of RUT in patients without history of NSAID use were 97.37, 98.57 and 98.14%, respectively.

RUT	Present study	Khean Lee et al[50]	Said RM et al[51]	Mojgan foroutan et al[26]
sensitivity	100%	98.2%	98.1%,	97.37%
specificity	96%	99.0%	100%	98.57%
diagnostic accuracy	98%.	98.5%	99%	98.14%

Dyspepsia Score

This study provides information regarding dyspepsia score in *H. pylori* positive and *H. pylori* negative cases. Out of 108 dyspeptic patients 60 were *H. pylori* positive, the mean dyspepsia score in positive patients were 6.33 with a standard deviation of 1.48 and in *H. pylori* negative patients the mean dyspepsia score was 5.87 with a standard deviation of 1.61. the difference in mean dyspeptic score in two groups was statistically significant.

Out of 60 *H. pylori* positive patients 49 patients came negative after treatment, the mean dyspepsia score before treatment in these patients was 6.36 and after treatment it reduces to 2.57. The result of this reduction was statistically significant.

11 patients who were resistant to anti *H. pylori* treatment, mean dyspepsia score before treatment was 6.18 and after the eradication therapy it was 5.63. however this difference was statistically insignificant.

In our study the mean dyspepsia score before eradication therapy was more in *H. pylori* positive patient and less in *H. pylori* negative patient.

Shrivastava et al[16] in their study found that 80 patients with functional dyspepsia were randomly distributed into two groups to receive eradication or placebo therapy after taking biopsies for *H. pylori*. Symptom evaluation was done at baseline, at one and at three months to notice any improvement. There was no significant difference in

the total dyspepsia score between the *H. pylori* positive group and the *H. pylori* negative group.

Sarnelli G et al[63] concluded that there is no difference in the clinical presentation of patients with or without *H. pylori* infection.

Glasbrenner B et al[17] studied 88 patients with functional dyspepsia and found that the median symptom sum score was 8.5 in *H. pylori*-positive and 9.5 in *H. pylori*-negative patients with functional dyspepsia and they concluded that, there is no significant relationship with the severity of symptoms between the positive and negative group.

Efficacy of anti *H. pylori* treatment

In our study, we found that out of 108 patients, 60 patients were positive for *H. pylori* by RUT test. All these patients treated for eradication of *H. pylori*. Repeat RUT test after eradication therapy was performed at an interval of 1 month to know the status of *H. pylori*. Out of 60 patients 49 patients turn negative for RUT but 11 patients remain positive for RUT even after eradication therapy.

In *H. Pylori* positive patients, mean dyspepsia score before treatment was noted 6.33 with a standard deviation of 1.48, and after treatment mean dyspepsia score was 3.13 with a standard deviation of 1.96 there was a significant positive association was noted with mean dyspepsia score pre and post treatment (p value 0.0001). It shows that in patient with *H. pylori* infection after taking anti *H. pylori* treatment the symptoms were significantly improved.

Resistant 11 patients have a mean dyspepsia score of 6.74 before treatment and it reduces to 5.93 after treatment this reduction is not statistically significant. P<0.001.

As per results of RUT test the success rate of eradication therapy in our study was 81.6%.

Hyun Jeong Lee et al 2015[52] study was performed on 143 patients and the treatment was given similar to our study, i.e. proton pump inhibitor, amoxicillin and clarithromycin and found an Eradication rate of 76.2%.

In another study by Arama S et al 2016[145], includes 78 patient in which 52 were given 14 days anti-*H. pylori* treatment and found that an eradication rate of 84.6%.

In a study conducted by Shrivastava et al[16] found that there is a significant difference in dyspepsia score (P<0.001) pretreatment and post treatment. The finding of this study is correlating with our study.

Abdul A et al 2009[53] conducted a study in 23 *H. pylori* positive patients and graded their symptom using completed Modified Glasgow Dyspepsia Severity Score Questionnaire (MGDSSQ) before undergoing the treatment and at the completion of treatment and found mean Symptom score of 8.43 ± 1.56 pretreatment and post treatment was 0.87 ± 1.01.

Gilvarry J. et al[27] in their study, they reported that the eradication of *H. pylori* was 85% of those treated with triple therapy and in 14% of those who received bismuth plus placebo.

Shrestha S et al (2012)[35], One month after the completion of treatment, 132 of 150 patients (88 percent) in the group assigned to receive omeprazole and antibiotics had a negative test for *H. pylori.*

A prospective study done by García-Romeroe et al[54] including 99 patients with duodenal ulcer and found that, the eradication of *Helicobacter pylori* with triple therapy for 6 days in patients with duodenal ulcer is not satisfactory.

The study done by kiyota et al[55] reported that the eradication rate of the 2-week regimen was higher than that of the 1-week regimen. These study correlate with our study.

Author	Patients	Criteria for *H. pylori* diagnosis	*Helicobacter pylori* therapy	Success in the *H. pylori* therapy group
Talley et al.[10]	278	Serologic or urease breath test or histopathology test	Omeprazole; amoxicillin; clarithromycin, twice daily for x 7 d	32/135 (24%)
Blum et al.[34]	328	Ureasebreath test or histopathology test	Omeprazole; amoxicillin; clarithromycin twice daily x 7 d	45/164 (27%)
Talley et al.[24]	317	Urease breath test or histopathology test	Omeprazole; amoxicillin; clarithromycin, twice daily x 14 d	69/162(43%)
Mccoll et al.[56]	318	Urease breath test or histopathology test	Omeprazole twice daily; amoxicillin; metronidazole three times daily x 14 d	33/160(21%)
Greenberg et al.[57]	100	Histologic test	Omeprazole, twice daily clarithromycin three times daily x 14 d	55/89(62%)
In present study	108	Rapid ureas test and histology	Omeprazole; amoxicillin; clarithromycin twice daily for x 7 d	49/60 (81.6%)

Mean dyspepsia score in *H. pylori* positive and negative patient before and after eradication therapy

In our study mean dyspepsia score in *H. pylori* positive patients was 6.33 and in patients of *H. pylori* negative patients was 5.87 and after eradication therapy the mean dyspepsia score reduces to 3.13 in *H. pylori* positive group, whereas 4.33 in negative group. But these two groups are not comparable because the same treatment was not given in both the groups.

H. pylori positive patients consist of two groups; first group was sensitive to anti *H. pylori* therapy (49 patient turn negative after anti *H. pylori* therapy) whereas patient resistant to anti *H. pylori* therapy form a second group(11 patient remain positive after anti *H. pylori* therapy). The dyspepsia score in sensitive patients before treatment was 6.36 and it reduces to 2.57 after treatment. This reduction of score was statistically significant. Dyspepsia score in resistant 11 patients was 6.18 and reduces to 5.63 after treatment. This is not statistically significant.

This clearly suggests a strong correlation between *H. pylori* infection and severity of dyspepsia score.

Taddesse G et al[58] reported that *H. pylori* infection is associated with dyspepsia in their study. Glasbrenner B et at[17] and Kyzekové J et al[18] reported that in a patients with dyspepsia, the gastritis or other dyspeptic symptom was strongly associated with *H. pylori* infection.

SUMMARY

The present study entitled "Study of Dyspepsia with Special Reference to *Helicobacter pylori*" is a hospital based prospective interventional study conducted in Tertiary Health Care Centre Acharya Vinobha Bhave rural hospital attached to Jawaharlal Nehru medical college which comes under Datta Meghe Institute of Medical Science University. One hundred and eight cases inclusive of Outdoor and Indoor patients, presenting in the department of Surgery, Medicine and ENT with functional dyspepsia and have one or more out of three symptoms, epigastric pain or burning, early satiety & post-prandial fullness and willing to undergo endoscopy were enrolled in this study.

Detailed symptomatology of all the patients was recorded. The symptoms were graded on the basis of frequency and severity, using Visual Analogue Scale (0-10 for each symptom). The total dyspepsia score is calculated for each patient by adding the scores for individual symptoms. All patients had undergone ultrasonography abdomen to rule out other organic causes of dyspepsia like cholecystitis and appendicitis and if present they are excluded from the study.

All patients were screened for eligibility and underwent esophagogastroduodenoscopy (OGD) from October 2015 to October 2017. Subsequently, patients were divided into *H. pylori* positive[60] and *H. pylori* negative groups[48], based on the result of Rapid Urease Test.

- In this study, majority of the dyspeptic patients 52(48.15%) belonged to the age group of 21-40 years and only 5 (4.63%) were in the age group of ≤20 years whereas common age group among *H. pylori* positive patients was 21-40 years and it include 29 patients. No significant difference was noted between the age in two groups though mean age was found to be higher in RUT negative subjects.
- Majority of the subjects i.e. 60 (55.56%) were male and 48 (44.44%) were female.
- The most common endoscopic finding was normal study 33(30.56%) followed by Antral gastritis in 29(26.85%), followed by Oesophagitis with Antral gastritis in 19(17.59%), Pangastritis in 16(14.81%), Pangastritis with duodenitis in 3(2.78%), Oesophagitis with gastric polyp in 3(2.78%), duodenal ulcer in 2(1.85%) and acute erosive gastritis with duodenitis 1(0.93%).
- In present study, the sensitivity of RUT was 100% and specificity of RUT came to be 96% and positive predictive value was 96.66% whereas negative predictive value was 100%. The diagnostic accuracy of RUT was 98.14%.
- The frequency of *H. pylori* infection was 55.55% in our study population.
- The success rate of eradication therapy in our study was 81.67% in which 49 cases out of 60 were came to be negative after 1 month treatment.
- The triple therapy is the effective treatment for *H. pylori*. There is a significant decrease in dyspepsia score after treatment. The success rate of eradication therapy in our study was 81.67%. Whereas in resistant 18.4% patients there was reduction of total dyspepsia score but this reduction is not statistically significant.
- In present study, we concluded that upper abdominal pain is not due to acid peptic disease, but most of the upper abdominal pain is due acid peptic disease with *H. pylori* positivity.

In the view of the above results, patients attending hospital with epigastric pain are mostly due to high incidence of *Helicobacter pylori*. Hence all patients attending hospital with epigastric pain should be subjected to routine oesophagogastroduodenoscopy and endoscopic biopsy to perform RUT for screening of the *H. pylori*.

CONCLUSION

Our conclusion is based upon the observations and analysis of this study.

- Oesophagogastroduodenoscopy (OGD) revealed normal finding in 33 out of 108 patients and on remaining 75 patients major abnormal finding found was antral gastritis. *H. pylori* infection was positive in 21.22% of patient with normal finding and all

become negative after eradication therapy. Out of 75 patients with abnormal finding on oesophagogastroduodenoscopy, 80% were positive for *H. pylori* and of which 64 patient become negative after eradication therapy. The frequency of *H. pylori* infection was 55.55% in our study population.

- Both Rapid Urease Test and Histopathological examination of biopsy specimen had almost similar results to detect *Helicobacter pylori* infection, proving both tests are good for diagnosing *H. pylori* infections.
- There was no significant difference noted in total dyspepsia score of *H. pylori* positive group and the *H. pylori* negative group. In *H. pylori* positive patients, there was significant reduction in their post treatment dyspepsia score which was statistically significant. Whereas In resistant patients who remain positive after treatment the post treatment dyspepsia score reduces to some extent but this reduction is not statistically significant.
- The success rate of eradication therapy in our study was 81.6%. in which 49 patients out of 60 came to be negative after 1 month treatment and 11 remain positive.

In present study, we concluded that upper abdominal pain and dyspeptic symptom are not only due to acid peptic disease, but most of the upper abdominal pain was associated with *H. pylori* infection. Present study showed that dyspepsia is strongly associated with *H. pylori* infection.

Recommendation

Based on this study, it is recommended to undergo upper GI endoscopic evaluation along with *H. pylori* testing in all patients with functional dyspepsia.

H. pylori infection should be treated using triple therapy regimen and repeat Oesophagogastroduodenoscopy and RUT for detection of *H. pylori* is recommended after the completion of therapy.

We recommend further studies at multiple centers in this regard to reinforce hypothesis of strong relation in between *Helicobacter pylori* infection and dyspepsia.

BIBLIOGRAPHY

1. Talley NJ, Vakil N. Guidelines for the management of dyspepsia. Am J Gastroenterol, 2005; 100(10): 2324–37.
2. Locke GR. Prevalence, incidence and natural history of dyspepsia and functional dyspepsia. Baillieres Clin Gastroenterol, 1998; 12(3): 435–42.
3. Drossman DA. The Functional Gastrointestinal Disorders and the Rome III Process. Gastroenterology, 2006; 130(5): 1377–90.
4. Shah SS, Bhatia SJ, Mistry FP. Epidemiology of dyspepsia in Mumbai. Indian J Gastroenterol. 2001; (May 2001).
5. Hirakawa K, Adachi K, Amano K, Katsube T, Ishihara S, Fukuda R, et al. Prevalence of non-ulcer dyspepsia in the Japanese population. J Gastroenterol Hepatol., 1999; 14(11): 1083–7.
6. Kitapçioğlu G, Mandiracioğlu A, Caymaz Bor C, Bor S. Overlap of symptoms of dyspepsia and gastroesophageal reflux in the community. Turkish J Gastroenterol, 2007; 18(1): 14–9.
7. Desai H. Dyspepsia. Japi., 2012; 60(march): 2012.
8. Futagami S, Shimpuku M, Yin Y, Shindo T, Kodaka Y, Nagoya H, et al. Pathophysiology of Functional Dyspepsia. J Nippon Med Sch [Internet]. 2011; 78(5): 280–5. Available from: http://joi.jlc.jst.go.jp/JST.JSTAGE/jnms/78.280?from=CrossRef
9. Armstrong D. Helicobacter pylori infection and dyspepsia. Scand J Gastroenterol Suppl [Internet]., 1996; 215: 38–47. Available from: http://www.ncbi.nlm.nih.gov/pubmed/8722381
10. Talley NJ, Janssens J, Lauritsen K, Rácz I, Bolling-Sternevald E. Eradication of Helicobacter pylori in functional dyspepsia: randomised double blind placebo controlled trial with 12 months' follow up. The Optimal Regimen Cures Helicobacter Induced Dyspepsia (ORCHID) Study Group. BMJ [Internet]., 1999; 318(7187): 833–7. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=27795&tool=pmcentrez&rendertype=abstract
11. Quartero AO, De Wit NJ, Lodder AC, Numans ME, Smout AJPM, Hoes AW. Disturbed solid-phase gastric emptying in functional dyspepsia: A meta-analysis. Dig Dis Sci., 1998; 43(9): 2028–33.
12. Ghoshal UC, Chaturvedi R, Correa P. The enigma of Helicobacter pylori infection and gastric cancer. Indian J Gastroenterol [Internet]., 2010; 29(3): 95–100. Available from: http://www.ncbi.nlm.nih.gov/pubmed/20585917%5Cnhttp://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC3217495
13. Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, et al. Management of Helicobacter pylori infection—the Maastricht V/Florence Consensus Report. Gut [Internet]., 2017; 66(1): 6–30. Available from: http://gut.bmj.com/lookup/doi/10.1136/gutjnl-2016-312288
14. Robin Warren J, Marshall B. Unidentified Curved Bacilli On Gastric Epithelium In Active Chronic Gastritis. Vol. 321, The Lancet., 1983; 1273–5.
15. Peter S, Beglinger C. Helicobacter pylori and gastric cancer: The causal relationship. Digestion., 2007; 75(1): 25–35.
16. Upendra K. Shrivastava AG. Role of Helicobacter pylori in functional dyspepsia. Indian J Surg 2004 Vol 66 Issue 6 [Internet]., 2006; 66(6): 341–6. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=4130974&tool=pmcentrez&rendertype=abstract

17. Glasbrenner B, Weiler S, Ellenrieder V, Müller P, Adler G. Relationship between helicobacter pylori infection, histological gastritis, and functional dyspepsia. Hepatogastroenterology [Internet]., 1998 [cited 2017 Oct 2]; 45(24): 2238–43. Available from: http://www.ncbi.nlm.nih.gov/pubmed/9951902
18. Kyzekové J, Arlt J, Arltová M. Is there any relationship between functional dyspepsia and chronic gastritis associated with Helicobacter pylori infection? Hepatogastroenterology [Internet]. [cited 2017 Oct 9]; 48(38): 594–602. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11379362
19. Moayyedi P, Forman D, Braunholtz D, Feltbower R, Crocombe W, Liptrott M, et al. The proportion of upper gastrointestinal symptoms in the community associated with Helicobacter pylori, lifestyle factors, and nonsteroidal anti-inflammatory drugs. Leeds HELP Study Group. Am J Gastroenterol., 2000; 95(6): 1448–55.
20. ZANTEN SJOV VAN. The role of Helicobacter pylori infection in non-ulcer dyspepsia S. Blackwell Sci Ltd, Aliment Pharmacol Ther., 1997; 11(1): 63–9.
21. Bernersen B, Johnsen R, Bostad L, Straume B, Sommer a I, Burhol PG. Is Helicobacter pylori the cause of dyspepsia? BMJ., 1992; 304(6837): 1276–9.
22. Strauss RM, Wang TC, Kelsey PB, Compton CC, Ferraro MJ, Perez-Perez G, et al. Association of Helicobacter pylori infection with dyspeptic symptoms in patients undergoing gastroduodenoscopy [Internet]. Vol. 89, Am J Med., 1990. p. 464–9. Available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=2220879
23. Chiba N. Treating Helicobacter pylori infection in primary care patients with uninvestigated dyspepsia. BMJ., 2002; 324(7344): 1012.
24. Talley NJ, Vakil N, Ballard ED, Fennerty MB. Absence of benefit of eradicating Helicobacter pylori in patients with nonulcer dyspepsia. N Engl J Med [Internet]., 1999; 341(15): 1106–11. Available from: papers://5aecfcca-9729-4def-92fe-c46e5cd7cc81/Paper/p53718
25. J C. Comparison of the Results of Rapid Urease Test (Pronto dry®) at One and Twenty Four Hours in PPIs-Used Patients. Thai J Gastroenterol., 2009; 10(1): 39–42.
26. Foroutan M, Loloei B, Irvani S, Azargashb E. Accuracy of rapid urease test in diagnosing helicobacter pylori infection in patients using NSAIDs. Saudi J Gastroenterol [Internet]., 2010; 16(2): 110. Available from: http://www.saudijgastro.com/text.asp?2010/16/2/110/61238
27. Gilvarry J, Buckley MJM, Beattie S, Hamilton H, OMorain CA. Eradication of Helicobacter pylori affects symptoms in non-ulcer dyspepsia. Scand J Gastroenterol., 1997; 32(6): 535–40.
28. Bashir T, Nadeem MA, Randhawa FA, Nisar S, Randhawa FA, Pervaiz A. Frequency of helicobacter pylori in distal oesophageal mucosa of patients with dyspepsia. J Ayub Med Coll Abbottabad [Internet]., 2014 [cited 2017 Oct 2]; 26(3): 307–9. Available
29. Sodhi JS, Javid G, Zargar SA, Tufail S, Shah A, Khan BA, et al. Prevalence of Helicobacter pylori infection and the effect of its eradication on symptoms of functional dyspepsia in Kashmir, India. J Gastroenterol Hepatol [Internet]., 2013 May [cited 2017 Oct 5]; 28(5): 808–13. Available from: http://www.ncbi. nlm.nih.gov/pubmed/23432600
30. Okello TR, Ogwang DM, Alema NO, Pecorella I. An Evaluation of 605 Endoscopic Examination in a Rural Setting, Lacor Hospital in Northern Uganda. Br J Med Med Res., 2016; 15(10): 24412.
31. Mohammed MO. Correlation of Endoscopic Findings with Various Helicobacter pylori Tests among Dyspeptic Patients. Int J Clin Med [Internet]., 2014; 5(October): 1180–8. Available from: http://www.scirp.org/journal/PaperDownload.aspx?DOI=10.4236/ijcm.2014.519151
32. Olokoba AB, Gashau W, Bwala S, Adamu A, Salawu FK. Helicobacter pylori infection in Nigerians with dyspepsia. Ghana Med J., 2013; 47(2): 79–81.
33. Bashir T, Nadeem MA, Nisar S, Randhawa FA, Pervaiz A. Frequency of helicobacter pylori in distal oesophageal mucosa of patients with dyspepsia. J Ayub Med Coll Abbottabad., 2014; 26(3): 307–9.
34. Blum AL, Talley NJ, O'Morain C, Zanten Sjov Van. Lack of effect of treating helicobacter pylori infection in patients with nonulcer dyspepsia. N Engl J Med., 1998; 339(26): 1875–81.
35. Shrestha S, Paudel P, Pradhan GB, Shrestha L, Bhattachan CL. Prevalence study of H. pylori infection in dyspeptic patients coming to Nepal Medical College Teaching Hospital, Jorpati, Kathmandu. Nepal Med Coll J., 2012; 14(3): 229–33.
36. Miwa H, Hirai S, Nagahara A, Murai T, Nishira T, Kikuchi S, et al. Cure of Helicobacter pylori Infection Does not Improve Symptoms in Non-Ulcer Dyspepsia Patients-a Double-Blind Placebo-Controlled Study. Aliment Pharmacol Ther., 2000; 14: 317–24.
37. Kaur G, Raj SM. A Study of the Concordance Between Endoscopic Gastritis and Histological Gastritis in an Area with a Low Background Prevalence of Helicobacter Pylori Infection. Singapore Med J., 2002; 43(2): 090–2.
38. Adlekha S, Chadha T, Krishnan P, Sumangala B. Prevalence of helicobacter pylori infection among patients undergoing upper gastrointestinal endoscopy in a medical college hospital in kerala, India. Ann Med Health Sci Res [Internet]., 2013 Oct

[cited 2017 Oct 16]; 3(4): 559–63. Available from: http://www.ncbi. nlm.nih.gov/pubmed/24380008
39. Jemilohun AC, Otegbayo JA, Ola SO, Oluwasola OA, Akere A. Prevalence of Helicobacter pylori among Nigerian patients with dyspepsia in Ibadan. Pan Afr Med J [Internet]., 2010 [cited 2017 Oct 6]; 6: 18. Available from: http://www.ncbi. nlm.nih.gov/pubmed/21734925
40. Faintuch JJ, Silva FM, Navarro-Rodriguez T, Barbuti RC, Hashimoto CL, Rossini ARAL, et al. Endoscopic findings in uninvestigated dyspepsia. BMC Gastroenterol [Internet]., 2014; 14(1): 19. Available from: http://bmcgastroenterol.biomedcentral.com/articles/10.1186/1471-230X-14-19
41. Zhou X, Zhang G. Risk factors and prevalence of Helicobacter pylori infection in high incidence area of gastric carcinoma. J Gastroenterol Hepatol [Internet]., 2013; 28: 551. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L71213852%5Cnhttp://dx.doi.org/10.1111/jgh.12363_2
42. Mbengue M, Diouf ML, Dangou JM, Ka MM, Ba-Seck A, Ndiaye MF, et al. [Frequency of Helicobacter pylori infection in symptomatic patients in Senegal]. Med Trop (Mars) [Internet]. 1997 [cited 2017 Oct 13]; 57(3): 256–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/9513152
43. Jamil O, Sarwar S, Hussain Z, Fiaz RO, Chaudary RD. Association Between Functional Dyspepsia and Severity of Depression. J Coll Physicians Surg Pak [Internet]. 2016 Jun [cited 2017 Oct 2]; 26(6): 513–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27353991
44. Mihara M, Haruma K, Kamada T, Komoto K, Yoshihara M, Sumii K, et al. The role of endoscopic findings for the diagnosis of Helicobacter pylori infection: evaluation in a country with high prevalence of atrophic gastritis. Helicobacter [Internet]. 1999 Mar [cited 2017 Oct 2]; 4(1): 40–8.
45. Dhaliwal US, Dhawan S, Multani AS. Reflux oesophagitis in acid peptic disease (a fibre-optic endoscopic study). J Indian Med Assoc., 1997; 95(2): 35–6.
46. Korkmaz H, Kesli R, Karabagli P, Terzi Y. Comparison of the Diagnostic Accuracy of Five Different Stool Antigen Tests for the Diagnosis of Helicobacter pylori Infection. Helicobacter [Internet]. 2013 Oct [cited 2017 Oct 14]; 18(5): 384–91. Available from: http://doi.wiley.com/10.1111/hel.12053
47. Logan RP, Walker MM. ABC of the upper gastrointestinal tract: Epidemiology and diagnosis of Helicobacter pylori infection. BMJ [Internet]. 2001 [cited 2017 Oct 13]; 323(7318): 920–2. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1121445/pdf/920.pdf
48. Lerang F, Moum B, Mowinckel P, Haug JB, Ragnhildstveit E, Berge T, et al. Accuracy of seven different tests for the diagnosis of Helicobacter pylori infection and the impact of H2-receptor antagonists on test results. Scand J Gastroenterol [Internet]. 1998 Jan 8 [cited 2017 Oct 13]; 33(4): 364–9. Available from: http://www.tandfonline.com/doi/full/10.1080/00365529850170982
49. Talib SH, Sane SD, Gopanpallikar AM, Kumbhakarna NR. Effect of Helicobacter Pylori Eradication Therapy in Patients of Bronchial Asthma with Gastroesophageal Reflux Disease (GERD). Journal, Indian Acad Clin Med., 2003; 4(1): 39–49.
50. Goh KL, Cheah PL, Navaratnam P, Chin SC, Xiao SD. HUITAI rapid urease test: A new ultra-rapid biopsy urease test for the diagnosis of Helicobacter pylori infection. J Dig Dis., 2007; 8(3): 139–42.
51. Said RM, Cheah P-L, Chin S-C, Goh K-L. Evaluation of a new biopsy urease test: Pronto Dry, for the diagnosis of Helicobacter pylori infection. Eur J Gastroenterol Hepatol [Internet]. 2004; 16(2): 195–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/15075994
52. Lee HJ, Kim J Il, Lee JS, Jun EJ, Oh JH, Cheung DY, et al. Concomitant therapy achieved the best eradication rate for Helicobacter pylori among various treatment strategies. World J Gastroenterol, 2015; 21(1): 351–9.
53. Abdul Aziz A, Hamzah Z, Tong SF, Nadeson S, Wan Puteh S. Helicobacter pylori related dyspepsia: prevalence and treatment outcomes at University Kebangsaan Malaysia-Primary Care Centre. Asia Pac Fam Med [Internet]. 2009 May 12 [cited 2017 Oct 4]; 8(1): 4. Available from: http://apfmj.biomedcentral.com/articles/10.1186/1447-056X-8-4
54. García-Romero E, del Val A, Garrigues V, Cuquerella J, Higón MD, Barrachina M, et al. [Treatment with omeprazole, clarithromycin and amoxicillin over 6 days in patients with Helicobacter pylori-infected duodenal ulcer]. Gastroenterol Hepatol [Internet]. 1999 Jan [cited 2017 Oct 3]; 22(1): 1–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10089703
55. Kiyota K, Habu Y, Sugano Y, Inokuchi H, Mizuno S, Kimoto K, et al. Comparison of 1-week and 2-week triple therapy with omeprazole, amoxicillin, and clarithromycin in peptic ulcer patients with Helicobacter pylori infection: results of a randomized controlled trial. J Gastroenterol [Internet]. 1999 [cited 2017 Oct 3]; 34 Suppl 11: 76–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/10616771
56. McColl K et al. Symptomatic benefit from eradicating Helicobacter Pylori infection in patients with non-ulcer dyspepsia. New Engl. J. Med (1998) 339: 1869-74. Copyright Regulations 1969. Library (Lond)., 1998; 1968: 1869–74.
57. Greenberg PD, Cello JP. Lack of effect of treatment for Helicobacter pylori on symptoms of nonulcer

dyspepsia. Arch Intern Med., 1999; 159(19): 2283–8.
58. Taddesse G, Habteselassie A, Desta K, Esayas S, Bane A. Association of dyspepsia symptoms and Helicobacter pylori infections in private higher clinic, Addis Ababa, Ethiopia. Ethiop Med J [Internet]. 2011 Apr [cited 2017 Oct 14]; 49(2): 109–16. Available from: http://www.ncbi.nlm.nih.gov/pubmed/ 21796910
59. DeLoach LJ, Higgins MS, Caplan a B, Stiff JL. The visual analog scale in the immediate postoperative period: intrasubject variability and correlation with a numeric scale. Anesth Analg., 1998; 86(1): 102–6.
60. Arama SS, Tiliscan C, Negoita C, Croitoru A, Arama V, Mihai CM, et al. Efficacy of 7-day and 14-day triple therapy regimens for the eradication of helicobacter pylori: A comparative study in a cohort of romanian patients. Gastroenterol Res Pract., 2016; 2016.
61. Syam AF, Abdullah M, Rani AA, Nurdjanah S, Adi P, Djumhana A, et al. Evaluation of the use of rapid urease test: Pronto dryt o detect H. pylori in patients with dyspepsia in several cities in Indonesia. World J Gastroenterol, 2006; 12(38): 6216–8.
62. Siddique I, Al-Mekhaizeem K, Alateeqi N, Memon A, Hasan F. Diagnosis of Helicobacter pylori: improving the sensitivity of CLOtest by increasing the number of gastric antral biopsies. J Clin Gastroenterol [Internet]., 2008; 42(4): 356–60. Available from: http://www.ncbi.nlm.nih.gov/pubmed/ 18277905
63. Sarnelli G, Cuomo R, Janssens J, Tack J. Symptom patterns and pathophysiological mechanisms in dyspeptic patients with and without Helicobacter pylori. Dig Dis Sci., 2003; 48(12): 2229–36.