"PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS UNDERGOING UPPER GASTRO INTESTINAL ENDOSCOPY AT KLES DR. PRABHAKAR KORE HOSPITAL – A ONE YEAR CROSS-SECTIONAL STUDY"

REG NO. BH0110001

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M.S.)
in
GENERAL SURGERY

DEPARTMENT OF SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

APRIL - 2013

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ENDORSEMENT

This is to certify that the dissertation entitled **"PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS UNDERGOING UPPER GASTRO INTESTINAL ENDOSCOPY AT KLES DR. PRABHAKAR KORE HOSPITAL – A ONE YEAR CROSS-SECTIONAL STUDY"** is a bonafide research work done by **THE CANDIDATE REG NO. BH0110001.**

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LIST OF ABBREVIATIONS USED

CagA	- Cytotoxic associated gene A
dF	- Degree of Freedom
DPX	- Dibutyl phthalate and xylene
GERD	- Gastroesophageal reflux disease
H.Pylori	- Helicobacter pylori
HIV	- Human immunodeficiency virus
HPR	- Histopathological reporting
MALT	- Mucosa associated lymphoid tissue
M	- Moles
MW	- Molecular weight
NPV	- Negative predictive value
PCR	- Polymerase chain reaction
PPV	- Positive predictive value
RUT	- Rapid urease test
VacA	- Vacuolating cytotoxin A

ABSTRACT

Background and objectives

Helicobacter pylori (H. pylori) infection is the principal cause of chronic active gastritis and peptic ulcer disease and a major contributor for gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma. The present study was undertaken to estimate the prevalence of H. pylori and its association with gastritis.

Methodology

The present one year cross-sectional study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2011 to December 2011 A total of 175 patients undergoing upper gastro intestinal endoscopy were included in the study. Two biopsy specimens were obtained from the antrum. Biopsy was also taken from any suspicious lesions, if noted. One biopsy specimen from the antrum was used for rapid urease test and the remaining biopsy specimen sent for HPR.

Results

In the present study most of the patients (73.71%) were males. The male to female ratio was 2.80:1. Most of the patients were aged between 46 to 60 years (37.17%). The mean age of the study population was 49.47 ± 15.22 years. 94.86% of patients presented with pain abdomen. The next common complaint was vomiting which was present in 50.86%. The endoscopic findings revealed acute gastritis in 42.29% patients and chronic gastritis in 24% patients. Based on

histopathological findings, chronic gastritis was present among 69.14% patients and acute gastritis in 16%.

Conclusion and interpretation

Based on rapid urease test prevalence of *H. pylori* infection was 54.29% and histopathological reports revealed 29.14% prevalence of *H. pylori* infection. Rapid urease test showed 88.2% sensitivity and 59.6% specificity, 47.3% positive predictive value and 92.5% negative predictive value in diagnosing H. pylori infection when compared with histopathology. The association between *H. pylori* infection and gastritis could not be established in this study.

Keywords

Acute gastritis; Chronic gastritis; Helicobacter pylori infection; Rapid urease test.

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Chapter 1

Introduction

INTRODUCTION

Helicobacter pylori (H. pylori) infection is the principal cause of chronic active gastritis and peptic ulcer disease and a major contributor for gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma.[1]

Numerous studies have tried to assess the incidence and prevalence of *H. pylori* infection, its mode of transmission, and any risk factors contributing to development of the infection. The annual incidence reported in 3 adult studies in developed countries was between 0.3% and 0.5% per year.[2-4]

Recent studies have reported that, approximately 50% of the world's population is infected,[5] but only 10%–20% of infected persons become symptomatic.[6] The annual incidence rate of *H. pylori* infection is ≈4%–15% in developing countries, compared with ≈0.5% in industrialized countries.[7] Studies in the Republic of Georgia (ROG), a developing country with an economy in transition, suggested that >70% of adults are infected with *H. pylori*[8,9] and that the prevalence rate of gastric cancer is 18 per 100,000 population, ≈6- to 9-fold higher than in the United States.

Prevalence estimates vary greatly, depending on the location of the study group and the characteristics of the population studied. In general, prevalence increases with age[10] and correlates positively with a low socioeconomic status during childhood.[11] Worldwide, but especially in developed nations, infection with *H. pylori* is declining.[12]

The prevalence of H. Pylori infection in the west is less than 50%, while among developing countries this rate is high and ranges between 70 to 80%.[13] Helicobacter Pylori infection is common in the Indian subcontinent. Exposure occurs in childhood and approximately 80% of adults have been infected at sometime.[13]

The acquisition of *H. pylori* occurs during childhood, most often by a fecal-oral or oral-oral route. Some studies have also indicated a role for a gastrooral route of transmission.[14] The role played by other factors, including ABO blood group, alcohol and tobacco use, dietary and nutritional influences, and genetic predisposition to infection, has also been studied, but results have been inconsistent.[15]

In addition to producing local injury of gastric mucosa, *H. pylori* alters normal gastric secretion. Interestingly, the location and severity of the infection seem closely associated with the ultimate clinical outcome, most likely because of effects on gastric physiology. Many studies have shown that patients with a duodenal ulcer who are infected with *H. pylori* have an increased serum level of gastrin, which in turn leads to increased acid output.[16] These patients tend to have a milder phenotypic expression of their gastritis, with inflammation mostly in the antrum or distal part of the stomach.[17]

Currently, there are several popular methods for detecting the presence of *H. pylori* infection, each having its own advantages, disadvantages, and limitations. Basically, the tests available for diagnosis can be separated according to whether or not endoscopic biopsy is necessary. Histologic evaluation, culture,

polymerase chain reaction (PCR), and rapid urease tests are typically performed on tissue obtained at endoscopy. Alternatively, simple breath tests, serology, and stool assays are sometimes used, and trials investigating PCR amplification of saliva, feces, and dental plaque to detect the presence of *H. pylori* are ongoing.[18]

Despite the fact of high prevalence of *H. Pylori* infection there is scarcity of literature regarding prevalence of H. Pylori infection in this region. Hence the present study was undertaken to estimate the prevalence of H. pylori and its association with gastritis.

Chapter 2

Objectives

OBJECTIVES

The objectives of the present study were;

Primary

To determine the prevalence of *H. Pylori* infection in patients undergoing upper gastro intestinal endoscopy.

Secondary

To evaluate the association between *H. Pylori* infection and gastritis.

Chapter 3

Review of Literature

REVIEW OF LITERATURE

The discovery of Helicobacter pylori as a causative agent of peptic ulcer disease has revolutionized the understanding of the treatment of this condition.[19] Many patients still attribute symptoms of dyspepsia to an ulcer, and believe that ulcers are caused by diet, stress, and lifestyle factors; however, it is now clear that eradication of H. pylori is central to the management of this illness.[20]

Helicobacter pylori is a gram negative, microaerophilic bacterium that can inhabit various areas of the stomach, particularly the antrum. It causes a chronic low-level inflammation of the stomach lining and is strongly linked to the development of duodenal and gastric ulcers and stomach cancer.[21]

The bacterium was initially named Campylobacter pyloridis, then renamed H. pylori (pylori = genitive of pylorus). When 16S rRNA gene sequencing and other research showed in 1989 that the bacterium did not belong in the genus Campylobacter, it was placed in its own genus, Helicobacter. The genus derived from the ancient Greek "spiral" or "coil". The specific epithet pylōri means "of the pylorus" or pyloric valve (the circular opening leading from the stomach into the duodenum) from the Ancient Greek word which means gatekeeper.[22]

Historical aspects

Helicobacter pylori was first discovered in the stomachs of patients with gastritis and stomach ulcers in 1982 by Dr. Barry Marshall and Dr. Robin Warren of Perth, Western Australia. At the time the conventional thinking was that no

bacterium could live in the human stomach as the stomach produced extensive amounts of acid of strength comparable to the acid found in a car battery. Marshall and Warren rewrote the textbooks with reference to what causes gastritis and gastric ulcers. In recognition of their discovery, they were awarded the 2005 Nobel Prize in Physiology or Medicine. German scientists found spiral-shaped bacteria in the lining of the human stomach in 1875, but they were unable to culture it and the results were eventually forgotten. The Italian researcher Giulio Bizzozero described similarly shaped bacteria living in the acidic environment of the stomach of dogs in 1893. Professor Walery Jaworski of the Jagiellonian University in Krakow investigated sediments of gastric washings obtained from humans in 1899. Among some rod-like bacteria, he also found bacteria with a characteristic spiral shape, which he called Vibrio rugula. He was the first to suggest a possible role of this organism in the pathogenesis of gastric diseases. This work was included in the Handbook of Gastric Diseases, but it had little impact as it was written in Polish. Several small studies conducted in the early 1900s demonstrated the presence of curved rods in the stomach of many patients with peptic ulcers and stomach cancer.[23-25]

However interest in the bacteria waned when an American study published in 1954 failed to observe the bacteria in 1180 stomach biopsies.[26]

Interest in understanding the role of bacteria in stomach diseases was rekindled in the 1970s with the visualization of bacteria in the stomach of gastric ulcer patients. The bacterium had also been observed in 1979 by Robin Warren, who did further research on it with physician Barry Marshall beginning in 1981. After numerous unsuccessful attempts at culturing the bacteria from the stomach,

they finally succeeded in growing colonies in 1982 when they unintentionally left their Petri dishes incubating for 5 days over the Easter weekend. In their original paper, Warren and Marshall contended that most stomach ulcers and gastritis were caused by infection by this bacterium and not by stress or spicy food as had been assumed before.[19]

Although there was some skepticism initially, within several years numerous research groups verified the association of Helicobacter pylori with gastritis and to a lesser extent ulcer. To demonstrate that Helicobacter pylori caused gastritis and was not merely a bystander, Marshall drank a beaker of Helicobacter pylori culture. He became ill with nausea and vomiting several days later. An endoscopy ten days after inoculation revealed signs of gastritis and the presence of Helicobacter pylori. These results suggested that Helicobacter pylori was the causative agent of gastritis. Marshall and Warren went on to demonstrate that antibiotics are effective in the treatment of many cases of gastritis. In 1987 the Sydney gastroenterologist Thomas Borody invented the first triple therapy for the treatment of duodenal ulcers. In 1994, the National Institutes of Health (USA) published an opinion stating that most recurrent duodenal and gastric ulcers were caused by Helicobacter pylori and recommended that antibiotics be included in the treatment regimen.[27]

Recent research states that genetic diversity in Helicobacter pylori decreases with geographic distance from East Africa, the birthplace of modern humans. Using the genetic diversity data, the researchers have created simulations that indicate the bacterium seems to have spread from East Africa around 58,000 years ago. Their results indicate modern humans were already

infected by Helicobacter pylori before their migrations out of Africa, remaining associated with human hosts since that time.[28]

Epidemiology

Frequency

At least half the world's population is infected by the bacterium, making it the most widespread infection in the world. Actual infection rates vary from nation to nation, the people in under developed countries has much higher infection rates than the developed countries like North America, Australasia etc. where rates are estimated to be around 25%.[29] Infections are usually acquired in early childhood in all countries. However, the infection rate of children in developing nations is higher than in industrialized nations, probably due to poor sanitary conditions. In developed nations it is currently uncommon to find infected children, but the percentage of infected people increases with age, with about 50% infected over the age of 60 compared with around 10% between 18 and 30 years. The higher prevalence among the elderly reflects higher infection rates when they were children rather than infection at later ages.[29]

Prevalence appears to be higher in African-American and Hispanic populations, although this is likely related to socioeconomic rather than racial factors. The lower rate of infection in the developed countries is largely attributed to higher hygiene standards and widespread use of antibiotics. Despite high rates of infection in certain areas of the world, the overall frequency of Helicobacter pylori infection is declining. However, antibiotic resistance is appearing in

Helicobacter pylori; there are already many metronidazole and clarithromycin resistant strains in most parts of the world.[30]

Mortality/Morbidity

The mortality rate related to *H pylori* infection is not precisely known, but it seems to be minimal. Mortality is due to the complications of the infection, such as gastric ulcer perforation or MALTomas of the GI tract. Otherwise, the morbidity of *H pylori* infection can be very high.[31]

Race

The pathogenetic role of *H pylori* may differ depending on geography and race. White persons are infected with *H pylori* less frequently than persons of other racial groups. The prevalence rate is approximately 20% in white persons, 54% in African American persons, and 60% in Hispanic persons.[31]

Sex

No sex predilection is known; however, females have a higher incidence of reinfection (5-8%) than males.[31]

Age

H pylori infection may be acquired at any age. According to some epidemiologic studies, this infection is acquired most frequently during childhood. Children and females have a higher incidence of reinfection (5-8%) than adult males.[31]

The prevalence of *H. pylori* infection varies widely by geographic area, age, race, and socioeconomic status. It is not possible to ascertain when infection occurs, because most of the information on the rates of *H. pylori* in geographically and demographically diverse populations comes from seroprevalence studies. This has major disadvantages for epidemiologists, since it is generally not possible to distinguish between factors associated with acquiring versus maintaining *H. pylori* infection.[32]

A study reported that, approximately 30% of patients with dyspepsia in North America are infected with H. pylori compared with a prevalence of 80 to 90% in the developing world. The annual incidence of new H. pylori infections in industrialized countries is approximately 0.5 per 100 persons of the susceptible population compared with three or more per 100 persons in developing countries. In North America, the prevalence of H. pylori among Asian Americans, African Americans, and Hispanics is similar to that among persons in developing countries.[33]

In the United States, differences by race are evident, with Whites having a substantially lower seroprevalence of *H. pylori* than either Blacks or Hispanics.[32]

Ethnic differences were also evident in New Zealand, where *H. pylori* infection was most prevalent in Pacific Islanders, intermediate in Maori, and least prevalent in Europeans. After adjusting for age and socio economic status, the relative risks for Maori and Pacific Island subjects compared with European subjects were 1.4 (95 percent CI: 1.1,1.8) and 1.8 (95 percent CI: 1.4, 2.2), respectively. These differences in *H. pylori* prevalence by race/ethnicity and

nationality may reflect differences in social and/or hygiene factors or the widespread use of antimicrobials for treatment of other common infections, especially during childhood. This variability may also be explained by differences in ethnic or genetic predisposition to infections.[32]

Another study[33] on epidemiologic review on H. pylori from Singapore reported that, *H. pylori* seroprevalence rate in Bangladesh as 92%. In India, the reported overall seroprevalence rate was 79%. In Vietnam, the *H. pylori* seroprevalence rate was 74.6%. On the other hand, the seroprevalence rates in more developed countries were generally lower. In Australia, the overall seroprevalence rate was 15.1%. In Asian countries that became developed or industrialized in recent years, the seroprevalence rates were higher than Australia, but still considerably lower than less developed countries. In addition, a temporal effect was also evident with the younger population having low prevalence rates similar to developed Western countries. Among East Asian countries, the overall seroprevalence rate was 58.07% in China, 39.3% in Japan, 59.6% in South Korea and 54.5% in Taiwan. Among Southeast Asian countries, the reported seroprevalence rate was 35.9% in Malaysia, 31% in Singapore and 57% in Thailand.

In one of the Indian study[34] from chandigarh, two hundred and fifty-four individuals were screened for H. pylori. There were 80 symptomatic and 67 asymptomatic individuals. Helicobacter pylori was positive in 38 (56.7%) asymptomatic and 49 (61.3%) symptomatic individuals (P>0.05).Helicobacter pylori was present in 11/13 (84.6%) subjects with peptic ulcer.

Similarly in other Indian study,[35] H pylori prevalence in patients with dyspepsia and in control subjects was 65% and 46% respectively. The over all prevalence recorded in a study from Jammu was less in comparison to the recent studies from India[35,36] and outside India.[37]

A study from Jammu, India reported among H. pylori positive patients, 64.13% were males and 35.86% were females. Age wise distribution showed maximum prevalence of H. pylori infection in the age group of 36-45 years and minimum in the age group of 66-75 years.[34]

In another Indian study[35] age-related prevalence in the age groups of 10-19 years, 20-29 years, 30-39 years, 40-49 years and > or = 50 years were 52%, 70%, 69%, 60% and 59%, respectively.

Although some studies have reported an excess of *H. pylori* in one gender versus the other, no noteworthy gender differences exist in *H. pylori* prevalence overall. Differences in *H. pylori* prevalence by socio economic status factors can be striking.[32]

Risk factors

Risk factors for acquiring H. pylori infection include residence in a developing country, poor socioeconomic conditions, family overcrowding, and possibly an ethnic or genetic predisposition.[38] The other H. pylori associated risk factors include smoking, alcohol consumption, diet, occupational exposures, waterborne exposures, hygiene practices, density/crowding, social factors, and family history of gastric disease.

Smoking

Studies have assessed the possible association between *H. pylori* infection and smoking. Whereas some found that *H.* pylori-seropositive subjects were overall more likely than-seronegative subjects to be current smokers,[39-42] results were often not consistent by race or gender. Hamajima et al.[40] found an odds ratio of 7.8 for *H. pylori* infection for current male smokers but an odds ratio of only 1.2 for current female smokers. Conversely, Lin et al.[39] found a significant association with current smoking for females (OR = 2.8) but not for males. Lin et al.[39] found no association with intensity of smoking or age at which smoking began. Most of the recent studies found no significant association with current smoking or any other mode of tobacco use,[32] and one study from Japan[43] reported a significant negative association with current smoking. While one cannot rule out that an association between smoking and *H. pylori* infection may exist, such a hypothesis is not strongly supported by the current literature.

Alcohol consumption

None of several epidemiologic studies of the relation between alcohol consumption and *H. pylori* infection found a positive association, but many noted a nonstatistically significant reduction in risk.[32,39,42,43] It is difficult to evaluate whether alcohol consumption has a "protective" effect on the prevalence of *H. pylori. H. pylori* is better able to survive in the acid environment of the stomach than other bacteria are because of its production of urease. Therefore, it is not surprising that the reduction in pH that may accompany alcohol consumption would have little effect on the prevalence of *H. pylori*. However, alcohol is

known to have direct antimicrobial effects that appear to be more pronounced for wine than for other types of alcoholic beverages.[32]

Diet

Studies have also looked at dietary associations with *H. pylori.* Although the studies cover many different types of populations and include both adults and children, some consistent associations suggest that nutritional status may be related to *H. pylori* infection. Goodman et al.[44,45] and Fontham et al.[42] found significantly reduced odds ratios and negative gradients in risk of *H. pylori* infection with increased consumption of fruits and/or vegetables. An intervention study by Jarosz et al.[46] found that *H. pylori* infection was apparently eradicated in 30% of patients with chronic gastritis who were treated with vitamin C for 4 weeks compared with none in the control group.

Occupational exposures

Occupational exposures have been studied by several researchers to determine whether people working in certain occupations with potentially greater exposure to *H. pylori* had an increased prevalence of infection. Bohmer et al.,[47] in a study of inhabitants of institutes for the intellectually disabled in the Netherlands, found most of the intellectually disabled to be seropositive (83%). They also reported a higher rate of seropositivity (32%) among employees such as the nursing staff, who had intensive contact with institutionalized inhabitants, than among employees such as medical staff, speech trainers, secretarial staff, and drivers, who had little or no direct contact (14.1%). There has been conflicting data regarding the prevalence of *H. pylori* in endoscopy staff. Studies

in China and Taiwan found that medical staff who performed endoscopies had a higher prevalence of *H. pylori* than medical staff who did not perform these procedures,[48,49] and two studies in Australia reported the prevalence of *H. pylori* to be significantly higher in endoscopists compared with population controls.[50,51] In Germany, Braden et al.[52] found no increased risk of *H. pylori* infection in endoscopy staff (physicians and nurses) compared with the general medical staff (physicians and nurses) but did find a risk for all medical staff compared with controls. On the other hand, Rudi et al.[53] reported that exposure to neither patients in an acute care hospital nor to endoscopic procedures increased the rate of *H. pylori* infection.

Waterborne exposures

Water has been suggested as a possible source of *H. pylori* infection. Studies in Colombia, rural China, and Lima, Peru found that water source may be related to risk of *H. pylori* infection. Three waterborne factors were linked to higher risks of *H. pylori* infection in Colombian children: drinking water from a stream, swimming in a stream, and swimming in a swimming pool.[32]

Hygiene practices

Studies also have assessed the relation between *H. pylori* infection and various hygiene practice indicators in a number of countries. Overall, poor hygiene practices, especially during childhood, appear to be related to a higher seroprevalence of *H. pylori.* Some of these practices include having no water closet or bathroom or no hot water supply in the house when the subject was a child, sharing cups as children, having mothers who did not use soap when they

washed their hands, having mothers prechew the food for their young children, using chopsticks, not usually washing one's hands after going to the toilet, and living in a relatively small area with extremely limited sanitary facilities (e.g., submarine crews). Other hygiene practices during adulthood, such as sharing a toothbrush or cup and the type of toilet/bathroom facility, were not strongly related to *H. pylori* infection.[32]

Density/crowding

Studies have evaluated various density measures during both childhood and adulthood.[32] Some measure of overcrowding, such as living in a crowded environment, sibship size, number of persons or children in the home, number of persons per room, crowding index, having to share a room or bed with a parent, or living in an overcrowded space in a submarine, was consistently related to *H. pylori* positivity. The positive association of *H. pylori* with high-density environments, especially during childhood, suggests that crowded household quarters may facilitate transmission of infection among siblings and other family members. This finding is consistent with the data on intrafamilial clustering of *H. pylori*.

Social factors

In a variety of studies throughout the world, social factors have been independently associated with *H. pylori* status. The most commonly used measures were socio economic status based occupation, education and income.[32]

Occupation-based socio economic status was associated with *H. pylori* seroprevalence in studies in Ireland and South Wales and in a United Kingdom study by Webb et al.[54] but not in a study by Mendall et al.[55]

Income was related to *H. pylori* infection in Australia and Brazil and in Russia (in children but not adults) but not in Taiwan.[32]

Low educational level was significantly related to a higher risk of *H. pylori* in several studies.[32]

Low socio economic status, as defined differently by various investigators, also was associated with a higher seroprevalence of *H. pylori* in most studies in which it was evaluated.[32]

Family history of gastric disease

Studies have also evaluated the relation between *H. pylori* infection and family history of gastric disease. In a recent study by Brenner et al.,[56] the risk of being infected with *H. pylori* was significantly greater for adults with a parental history of stomach cancer than for those without such a history. The results for ulcer are somewhat inconsistent. Whereas the study in Germany by Brenner et al.[57] found a significantly elevated risk for children whose mothers, but not fathers, had ulcer disease, the study by Kikuchi et al. in Japan[43] reported a significantly elevated risk in public service workers whose fathers, but not mothers, had a history of ulcer disease.

Routes of transmission

Helicobacter pylori is contagious, although the exact route of transmission is not known. Person-to-person transmission by either the oral-oral or fecal-oral route is most likely. Consistent with these transmission routes, the bacteria have been isolated from feces, saliva and dental plaque of some infected people. Transmission occurs mainly within families in developed nations yet can also be acquired from the community in developing countries. Helicobacter pylori may also be transmitted orally by means of fecal matter through the ingestion of waste-tainted water, so a hygienic environment could help decrease the risk of Helicobacter pylori infection.[32]

Pathogenesis

The earliest descriptions of the organism classified it as predominately extracellular, gramnegative, flagellated, and motile. With the advancement of biochemical techniques, new information about the pathogenicity and virulence factors of Helicobacter pylori has emerged, indicating that infection by Helicobacter pylori requires a complex interaction of both bacterial and host factors.[21]

Investigators have identified several bacterial proteins necessary for colonization of the gastric mucosa by Helicobacter pylori, including proteins active in the transport of the organism to the surface of the mucosa (flagellin, which is encoded on genes flaA and flaB). Once in the presence of the gastric mucosa, bacteria induce a transient hypochlorhydria by an unknown mechanism.

The urease enzyme produced by the bacteria alters the microenvironment of the organism to facilitate colonization. Adherence then occurs via interaction between cell-surface glycolipids and adhesins specific to Helicobacter pylori. There also appears to be a role played by proteins called cecropins, which are produced by Helicobacter pylori and inhibit the growth of competing organisms, as well as by a P-type adenosine triphosphatase, which helps prevent excessive alkalinization of the microenvironment by urease. Once attached to gastric mucosa, Helicobacter pylori causes tissue injury by a complex cascade of events that depends on both the organism and the host. Helicobacter pylori, like all gram negative bacteria, has in its cell wall lipopolysaccharide, which acts to disrupt mucosal integrity.[21]

Furthermore, Helicobacter pylori release several pathogenic proteins that induce cell injury. For example, the CagA protein, produced by cytotoxic-associated gene A (cagA), is a highly immunogenic protein that may be associated with more severe clinical syndromes, such as duodenal ulcer and gastric adenocarcinoma (although this question is far from settled). There is increasing evidence that CagA positivity is associated with an increased risk for distal, but not proximal, gastric adenocarcinoma. In addition, protein products of the vacuolating cytotoxin A gene (vacA) and the A gene induced by contact with epithelium are known to be associated with mucosal injury.[21]

Once colonization of the gastric mucosa has taken place, the immunogenic properties of Helicobacter pylori induce an inflammatory reaction with neutrophilic gastritis that ultimately results in the clinical manifestations of the infection. This process is mediated by host factors, including interleukins 1, 2,

6, 8, and 12; interferon gamma, tumor necrosis factor, T and B lymphocytes and phagocytic cells. These factors mediate injury through release of reactive oxygen species and inflammatory cytokines. Helicobacter pylori additionally appear to increase the rate of mucosal-programmed cell death (also known as apoptosis).[21]

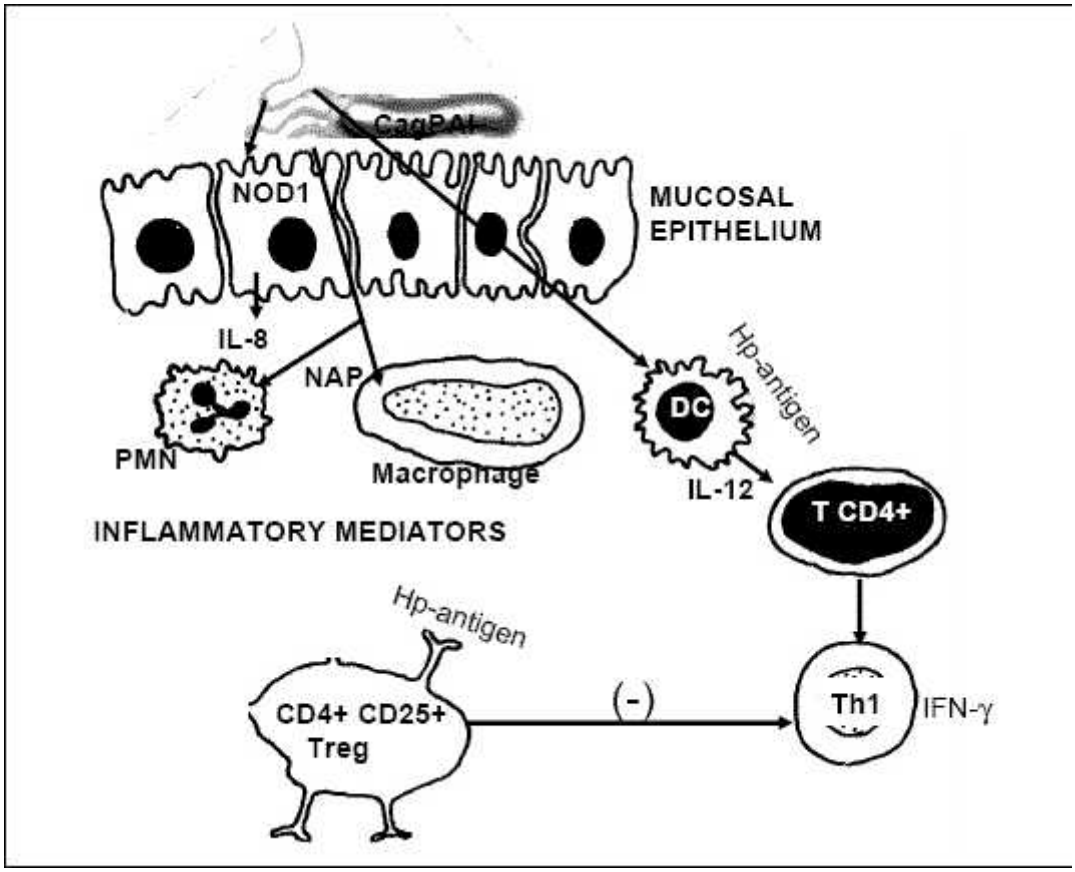


Figure 1. Interplay between *H. pylori* and the immune system. *H. pylori* induces both innate (macrophage, neutrophil and dendritic cell (DC) activation) and adaptive immune responses. Th1 effector cells of adaptive immunity are negatively regulated by T regulatory (Treg) cells. (Adapted from Mehmood A et al. IJABPT. 2010;1(3):1337-51).[21]

Effects on gastric physiology

In addition to producing local injury of gastric mucosa, *H. pylori* alters normal gastric secretion. Interestingly, the location and severity of the infection seem closely associated with the ultimate clinical outcome, most likely because

of effects on gastric physiology. Many studies have shown that patients with a duodenal ulcer who are infected with *H. pylori* have an increased serum level of gastrin, which in turn leads to increased acid output.[58] These patients tend to have a milder phenotypic expression of their gastritis, with inflammation mostly in the antrum or distal part of the stomach.[59] In contrast, patients with gastric adenocarcinoma, a known complication of *H. pylori* infection, tend to have pangastritis, with involvement of the acid-secreting body of the stomach as well as the antrum. This condition leads to atrophy of parietal cells (which are responsible for producing acid) and gastrin-producing cells of the antrum (which stimulate acid secretion) and eventually produces achlorhydria. Patients with gastric adenocarcinoma also have impaired acid secretion in response to stimulation with gastrin.[60]

Pathologic findings

Although extensive work has been performed to classify histopathologic changes seen with *H. pylori* infection, there is no consensus on classification; the Sydney system[61] and the Houston Gastritis Workshop system[62] have, however, been recognized as models. After colonization, there appears to be an intense neutrophilic infiltrate in the necks of the mucosal glands. Epithelial changes are common when there is irregularity of the surface architecture, and atrophy of the glands is typical of longstanding infection. Moreover, there is usually lymphocytic infiltration of the stroma and impaired mucus secretion. Finally, areas of patchy intestinal metaplasia may be seen, which are central to the development of neoplasia.[63]

Clinical manifestations

Once infected with Helicobacter pylori, most persons remain asymptomatic. Some infected persons may even clear the infection, with seroreversion rates commonly reported to be in the range of 5% to 10%; it is not known if this seroreversion is spontaneous or results from elimination of the organism by antibiotic agents used to treat other conditions. However, the typical course of disease in infected patients begins with chronic superficial gastritis, eventually progressing to atrophic gastritis. This progression appears to be a key event in the cellular cascade that results in the development of gastric carcinoma. The mechanism of tumorigenesis appears to involve DNA damage induced by different cytokines and free radicals released in the setting of chronic inflammation in susceptible persons. Although Helicobacter pylori is associated with the development of adenocarcinoma of the antrum and body of the stomach, it is also clearly linked with gastric mucosa–associated lymphoid tissue (MALT) lymphomas.[64]

Helicobacter pylori stimulates lymphocytic infiltration of the mucosal stroma; this infiltration may act as a focus for cellular alteration and proliferation, ultimately resulting in neoplastic transformation to lymphoma. It appears that Helicobacter pylori also produces proteins that stimulate growth of lymphocytes in the early stages of neoplasia. Most tellingly, it has been reported that regression of low-grade gastric MALT lymphoma can be achieved in 70% to 90% of patients with eradication of Helicobacter pylori infection. Recent work has shown endoscopic ultrasound examination to be invaluable in identifying the

grade of MALT lymphoma and in predicting the efficacy of treating the Helicobacter pylori infection to obtain regression of the lymphoma.[64]

Peptic ulcer disease

The relationship between Helicobacter pylori infection and peptic ulcer disease has been studied exhaustively, and it is now accepted that the organism is the major cause, but not the only cause, of peptic ulcer disease worldwide. Eradicating the infection can alter the natural course of peptic ulcer disease by dramatically reducing its recurrence rate in treated patients, compared with untreated patients. This reduction occurs in patients with duodenal and gastric ulcers that have no history of nonsteroidal anti-inflammatory drug use.[21]

The mechanism by which Helicobacter pylori induces peptic ulcer disease is incompletely understood but most likely involves a combination of genetic predisposition of the host, virulence factors of the organism (eg, VacA and CagA proteins), mechanical damage to the mucosa, and alterations of gastric and duodenal secretions.[21]

Non-ulcer dyspepsia

Non-ulcer dyspepsia comprises a constellation of varied symptoms, including dysmotility-like, ulcer-like, and reflux-like symptoms. Many possible causes have been suggested for non-ulcer dyspepsia, including lifestyle factors, stress, altered visceral sensation, increased serotonin sensitivity, alterations in gastric acid secretion and gastric emptying, and Helicobacter pylori infection. A

recent study also highlighted the role played by psychosocial impairment (depression, somatization, anxiety) in patients with non-ulcer dyspepsia.[21]

In a study linking[65] Helicobacter pylori infection to non-ulcer dyspepsia, patients with the latter condition were twice as likely to be positive for the organism. However, despite such epidemiologic evidence, treatment studies have failed to consistently show that eradication of Helicobacter pylori results in improvement of non-ulcer dyspepsia symptoms. Consequently, eradication of the organism can not be considered the standard of care in all patients with non-ulcer dyspepsia, because Helicobacter pylori infection is only a single part of the multi-factorial etiology of the disease.

Gastroesophageal reflux disease

Much attention has been focused on the possible relationship between infection with Helicobacter pylori and gastroesophageal reflux disease (GERD) in its various manifestations (esophagitis, Barrett's esophagus). Some investigators have suggested a link between the presence of Helicobacter pylori and a decreased risk for developing esophagitis and Barrett's esophagus; although this inverse association is supported by many prevalence studies, others fail to show it.

Studies have also indicated that certain strains of Helicobacter pylori, notably the CagA- positive strain, may be protective against the development of Barrett's esophagus. Moreover, Labenz and colleagues have shown that the incidence of esophagitis may in fact, increase after eradication of the organism. Treatment of Helicobacter pylori infection can lead to exacerbation of GERD in

many patients, prompting many gastroenterologists to defer endoscopic antral biopsies in patients with significant GERD and absent ulcer. Conversely, other studies using endoscopic findings, pH probe measurements, and histology to determine the presence of Helicobacter pylori did not find any association between GERD (in any of its manifestations) and infection with Helicobacter pylori. Clearly, more definitive studies are necessary to define the relationship, if any, between these 2 entities.[66]

Other disease associations

Investigators have further postulated a relationship between Helicobacter pylori infection and cardiovascular disease and iron-deficiency anemia. These associations, however, require much more study before a causal relationships is established.[67]

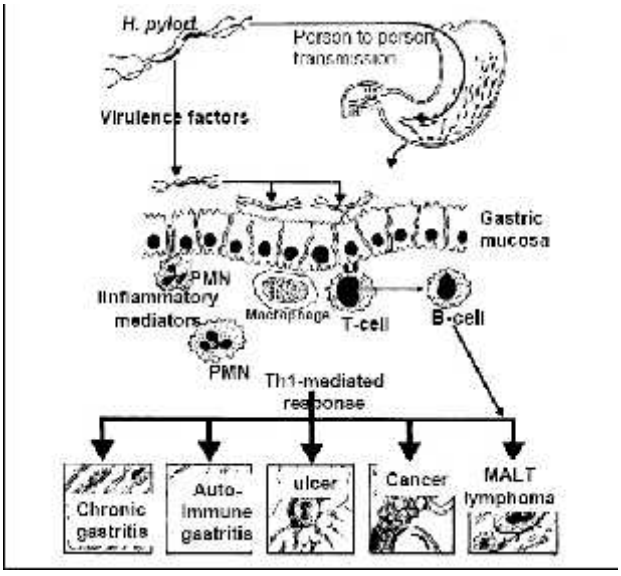


Figure 2. Immunopathology of *H. pylori* infection. Infection with *H. pylori* may result in: acute/chronic *gastritis*, autoimmune *gastritis*, peptic ulcer, gastric cancer or MALT lymphoma. (Adapted from Mehmood A et al. IJABPT. 2010;1(3):1337-51).[21]

Diagnostic testing

Currently, there are several popular methods for detecting the presence of Helicobacter pylori infection, each having its own advantages, disadvantages, and limitations. Basically, the tests available for diagnosis can be separated according to whether or not endoscopic biopsy is necessary. Histological evaluation, culture, polymerase chain reaction (PCR), and rapid urease tests are typically performed on tissue obtained at endoscopy. Alternatively, simple breath tests, serology, and stool assays are sometimes used, and trials investigating PCR amplification of saliva, feces, and dental plaque to detect the presence of Helicobacter pylori are ongoing.

Histology

Histologic evaluation has traditionally been the gold standard method for diagnosing Helicobacter pylori infection. The disadvantage of this technique is the need for endoscopy to obtain tissue. Limitations also arise at times because of an inadequate number of biopsy specimens obtained or failure to obtain specimens from different areas of the stomach. In some cases, different staining techniques may be necessary, which can involve longer processing times and higher costs.[21]

However, histologic sampling does allow for definitive diagnosis of infection, as well as of the degree of inflammation or metaplasia and the presence/absence of MALT lymphoma or other gastric cancers in high-risk patients.[21]

Culture

Because Helicobacter pylori is difficult to grow on culture media, the role of culture in diagnosis of the infection is limited mostly to research and epidemiologic considerations. Although costly, time-consuming, and labor intensive, culture does have a role in antibiotic susceptibility studies and studies of growth factors and metabolism.[21]

Polymerase chain reaction

With the advent of PCR, many exciting possibilities emerged for diagnosing and classifying Helicobacter pylori infection. PCR allows identification of the organism in small samples with few bacteria present and entails no special requirements in processing and transport. Moreover, PCR can be performed rapidly and cost- effectively, and it can be used to identify different strains of bacteria for pathogenic and epidemiologic studies. As suggested earlier, PCR also is being evaluated for its utility in identifying Helicobacter pylori in samples of dental plaque, saliva, and other easily sampled tissues. The major limitation of PCR is that relatively few laboratories currently have the capability to run the assay. In addition, because PCR can detect segments of Helicobacter pylori DNA in the gastric mucosa of previously treated patients, false-positive results can occur, and errors in human interpretation of bands on electrophoretic gels can likewise lead to false-negative results.[21]

Rapid urease testing

Rapid urease testing takes advantage of the fact that Helicobacter pylori is a urease producing organism. Samples obtained on endoscopy are placed in urea-containing medium; if urease is present, the urea will be broken down to carbon dioxide and ammonia, with a resultant increase in the pH of the medium and a subsequent color change in the pH dependent indicator. This test has the advantages of being inexpensive, fast, and widely available. It is limited, however, by the possibility of false negative results; decreased urease activity, caused either by recent ingestion of antibiotic agents, bismuth compounds, proton pump inhibitors, or sucralfate or by bile reflux, can contribute to these false-negative results.[21]

Urea breath test

A urea breath test similarly relies on the urease activity of Helicobacter pylori to detect the presence of active infection. In this test, a patient with suspected infection ingests either14Clabeled or 13C- labeled urea; 13C- labeled urea has the advantage of being non radioactive and thus safer (theoretically) for children and women of childbearing age. Urease, if present, splits the urea into ammonia and isotope-labeled carbon dioxide; the carbon dioxide is absorbed and eventually expired in the breath, where it is detected.[21]

Besides being excellent for documenting active infection, this test is also valuable for establishing absence of infection after treatment, an important consideration in patients with a history of complicated ulcer disease with

bleeding or perforation. In addition, a urea breath tests relatively inexpensive (whichever isotope is used), is easy to perform, and does not require endoscopy.[21]

However, if the patient has recently ingested proton pump inhibitors, antibiotic agents, or bismuth compounds, a urea breath test can be of limited value. Therefore, at least 1 week should separate the discontinuing of antisecretory medications and testing for active infection, and 4 weeks should separate treatment of Helicobacter pylori infection and testing for eradication of the organism. Moreover, except for major medical centers or tertiary referral centers where results are usually available in fewer than 24 hours, a urea breath test may be further limited by a turnaround time of several days (or longer) required for transport of samples and analysis by specialized laboratories not present in many community settings.[68]

Serologic tests

In response to Helicobacter pylori infection, the immune system typically mounts a response through production of immunoglobulins to organism-specific antigens. These antibodies can be detected in serum or whole-blood samples easily obtained in a physician's office. The presence of IgG antibodies to Helicobacter pylori can be detected by use of a biochemical assay, and many different ones are available. Serologic tests offer a fast, easy, and relatively inexpensive means of identifying patients who have been infected with the organism. However, this method is not a useful means of confirming eradication of Helicobacter pylori; several different samples and changes in titers of specified amounts over time would be needed. In addition, few patients become truly

seronegative, even after eradication of the organism. In low-prevalence populations, serologic tests should be a second-line methodology because of low positive predictive value and a tendency toward false-positive results. Serologic tests may be useful in identifying certain strains of more virulent Helicobacter pylori by detecting antibodies to virulence factors associated with more severe disease and complicated ulcers, gastric cancer, and lymphoma.[69]

Stool antigen testing

Stool antigen testing is a relatively new methodology that uses an enzyme immunoassay to detect the presence of Helicobacter pylori antigen in stool specimens. A cost effective and reliable means of diagnosing active infection and confirming cure, such testing has a sensitivity and specificity comparable to those of other noninvasive tests. Questions remain regarding possible cross reactivity with other Helicobacter species present in the intestines, but definitive studies are lacking.[21]

General diagnostic principles

The question, of which patients to test, when to test them and what test to use is still a troubling one for many physicians. Ultimately, the answer to these questions must be based on patient preference, cost, availability of different tests, and positive and negative predictive values of different tests (which depend on the individual patient population, including the prevalence of disorders caused by Helicobacter pylori infection in the community). Nevertheless, certain principles of testing seem universal. First, endoscopic methods of diagnosis should be used only if the procedure is necessary to detect some other condition besides

Helicobacter pylori infection. Second, only those patients in whom treatment will make a difference should be tested. Conclusive evidence does not exist that eradication of the infection in patients with simple dyspepsia will relieve symptoms, and testing of asymptomatic patients without a history of documented peptic ulcer disease is not warranted. Testing can be considered on a case by case basis in patients with symptoms suggestive of peptic ulcer disease.[21]

Because treatment of Helicobacter pylori infection is definitely indicated in patients with active or previously documented peptic ulcer disease, gastric MALT lymphoma, or family history of gastric cancer, their Helicobacter pylori status must be clarified. Urea breath and stool antigen tests are the most cost-efficient tests to identify active infection, but their limitations must be considered. Although serology is an excellent, inexpensive test to ascertain if someone with a history of peptic ulcer disease and unknown Helicobacter pylori status warrants treatment, endoscopy with tissue sampling in patients with a history of peptic ulcer disease can provide more definitive diagnosis of Helicobacter pylori infection, as well as information about the activity of peptic ulcer disease and possibly other factors at play (including gastric carcinoma).[21]

Diagnostic Tests for Helicobacter pylori[21]

Test	Sensitivity (%)	Specificity (%)	Usefulness
Invasive			
Endoscopy with biopsy			Diagnostic strategy of choice in children with persistent or severe upper abdominal symptoms
Histology	> 95	100	Sensitivity reduced by PPIs, antibiotics, and bismuth-containing compounds
Urease activity	93 to 97	> 95	Sensitivity reduced by PPIs, antibiotics, bismuth-containing compounds, and active bleeding
Culture	70 to 80	100	Technically demanding
Noninvasive			
Serology for immunoglobulin G	85	79	Sensitivity and specificity vary widely; positive result may persist for months after eradication; Reliability in children not adequately validated; not recommended
Urea breath test	95 to 100	91 to 98	Requires separate appointments; sensitivity reduced by PPIs, antibiotics, and bismuth-containing compounds; reliable test for cure
			Best available noninvasive test in children but higher false-positive rates in infants and children younger than six years compared with school-age children and adolescents
H. pylori stool antigen	91 to 98	94 to 99	Test for cure seven days after therapy is accurate; sensitivity reduced by PPIs, antibiotics, and bismuth-containing compounds;
			Easy to perform independent of age; possible alternative to urea breath test; monoclonal antibody-based test most reliable

Follow-up testing with urea breath or stool antigen tests both of which have sensitivities and specificities greater than 90% is necessary to document cure in patients with complicated peptic ulcer disease e.g. perforation, hemorrhage, obstruction or recurrent symptoms and should be performed 4 weeks after completion of treatment.[21]

Chapter 4

Methodology

METHODOLOGY

The present study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2011 to December 2011 on 100 patients undergoing upper gastro intestinal endoscopy.

Study design

The study design was cross-sectional study.

Study period and duration

The present study was conducted for one year from January 2011 to December 2011.

Place

The present study was carried done in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a teaching hospital attached to KLE University's Jawaharlal Nehru Medical College, Belgaum.

Source of Data

Cases undergoing upper gastro intestinal endoscopy in KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were included in the study.

Sample size

A total of 175 patients undergoing upper gastro intestinal endoscopy were included in the study.

Sampling procedure

After reviewing the literature the prevalence of H. Pylori infection was between 60 to 80% and average of the same was considered to calculate the sample size. The sample size "n" calculated by the following formula.

$n = (z^2 X p X q) / d^2$

Where, p = Prevalence (70%)

q = 100 – p (that is 100 – p = 30%)

d = Absolute error (10%)

Z = 1.96 ≈ 2 (Confidence interval - 95%)

Hence, n = 175

Selection criteria

Inclusion

- Patients aged between 18 to 70 years of both sexes.
- Patients undergoing upper gastro intestinal endoscopy and consenting for the participation in this study.

Exclusion

- Patients with HIV positive status.
- Patients in immuno-compromised states.

- Patients with gastric carcinoma.
- Patients receiving radiation therapy / anticancer chemotherapy.
- Patients with jaundice and upper gastro intestinal bleeding.
- Patient on the drugs such as Metronidazole, Tinidazole, Tetracycline, Amoxicillin, Clarithromycin, Tripotassium Dicitrato Bismuthate and Omeprezole

Ethical clearance

The study was approved from the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

The patients fulfilling selection criteria were informed in detail about the risks and benefits of the procedure and a written informed consent was obtained before enrollment (Annexure I).

Method of collection of data

Demographic data such as age and sex were recorded. Patients were interviewed for the clinical presentation such as pain abdomen, vomiting, haemetemesis and malena and history such as diet and personal habits. Through physical examination was conducted to assess built, nourishment and pallor. These findings were recorded on a predesigned and pretested proforma (Annexure II).

Procedure

Patient were kept nil by mouth six hours before the procedure. An upper gastro intestinal endoscopy was done and findings were noted. Two biopsy specimens were obtained from the antrum. Biopsy was also taken from any suspicious lesions, if noted. One biopsy specimen from the antrum was used for rapid urease test and the remaining biopsy specimen sent for HPR to the Department of pathology. Based on the rapid urease test and HPR findings the prevalence of H. pylori infection was determined.

Procedure of Rapid urease Test[70]

Biopsy specimen was crushed and put into 1ml Stuart's Urea Broth and incubated aerobically at 37 degree C. Colour change was observed over a period of several minutes. A colour change from yellow to reddish pink was considered positive. No colour change even after an hour was taken as negative test.

Composition of Stuart's Urea Broth (pH-6.8):

Yeast Extract	-	0.1 gm
Potassium Dihydrogen Orthophospate	-	9.1 gm
Anhydrous Disodium Hydrogen Phosphate	-	9.5 gm
Urea	-	20 gm
Phenol red	-	0.01gm

The media was prepared according to manufacturer instructions. The pH was adjusted to 6.8.The media was sterilized and dispensed into sterile tubes.

Photograph 1. Endoscopy unit

Photograph 2. Stuart's Urea Broth for Rapid Urease Test

Photograph 3. Rapid urease test – Pinkish red colour indicates positive for H pylori infection

The other biopsy specimen were sent for histopathological reporting.

Immediately after taking biopsy tissues were fixed into 10% formalin for a minimum of six hours. Afterwards paraffin blocks were prepared. From the paraffin blocks, sections of the tissue were cut with a thickness of 5 µm. Cut section were fixed to the slide.

Staining procedure[71]

1. H & E method

- Sections were deparaffinised
- Sections were stained with haematoxylein for four minutes.
- Sections were differentiated with 1% acid alcohol
- Sections were kept in running tap water for 5 to 10 minutes
- Sections were counterstained with eosin for 1.5 minutes
- Sections were washed in running tap water
- Sections were blotted three times, dehydrated in absolute alcohol for 1 to 2 minutes.
- Sections were blotted again put into xylene and blotted again and mounted with DPX

Result

- Helicobacter pylori – Rod shaped structure within the gland or above the mucous membrane

2.Giemsa Method

Composition

Azur II Eosin	-3.0gms
AzurII	-0.8gm
Glyserin	-250ml
Methyl alcohol	-250ml

Steps:

- Deparaffinise the section
- Put giemsa stain on the section for 1 minute
- Wash with tap water
- Dry and mount with DPX

Result

- Helicobacter pylori – Blue coloured rods within the glands
- Background – Bluish

3. Warthin Starry method

Step I – One litre of Walpole acetate – acetic buffer of pH 3.6 was prepared.

Substeps

- Preparation of stock solution
    - Stock A – 0.2 M acetic acid (MW 60.0) or 1.2 ml glacial acetic acid in 100 ml of distilled water.
    - Stock B – 0.2 M sodium acetate 1.64 gm sodium acetate anhydrous (MW 82.0) or 2.72 gm sodium acetate trihydrate (MW 136) in 100 mL of distilled water

- Composition of buffer at pH 3.6
    - 46.3 mL of stock A solution +
    - 3.7 mL of stock B solution +
    - Distilled water to make 100 CC

Step II

- Silver solution - Above prepared solution was used in preparing following solutions.

- Developer

    - 0.3 gm of hydroquinone + 10 mL of buffer. Of this solution 1 mL of solution was taken and to it 15 ml of warmed 5% gelatin was added and mixed and maintained at 40^0C.
    - 3 mL of 2% silver nitrate solution in pH 3.6 buffer was maintained at 55^0C
        - Solution x) and y) were mixed immediately before use.

Technique

- Sections were deparaffinized (Kept in xylene for five minutes and then with blotting paper excess xylene was removed sections were taken through absolute alcohol.
- Section hydrated with water and rinsed in pH 3.6 buffer.
- Sections stained with preheated 1% silver nitrate solution for 90 to 105 minutes at 55^0 to 60^0 C.

- Sections developed with developer for 3.5 minutes at 55^0C (sections turned golden brown).
- Developer poured off and sections rinsed in tap water for several minutes at 55^0 to 60^0 C and then in room temperature buffer.
- Sections dehydrated using alcohol and mounted.

Result

- Helicobacter pylori – Black curved rods
- Background – Yellowish brown

These methods were used for histopathological reporting.

The endoscopic findings were categorized as below:

Acute gastritis: Antral erosions, Acute erosive gastritis, Acute gastritis, Erosive gastritis, Diffuse haemorrhagic gastritis, Gastric erosions and Superficial gastritis.

Chronic gastritis: Chronic diffuse gastritis and Chronic gastritis

Gastric ulcer: Gastric ulcer

Duodenal ulcer: Duodenal ulcer

Others: Gastric polyp and Lax cardia

The association of H. pylori infection (according to rapid urease test and according to histopathological report) with gastritis (according to histopathological findings) was assessed.

Statistical analysis

The data obtained was coded and entered in Microsoft Excel Spreadsheet. The categorical data was expressed as rates, ratios and percentages and comparison was done using chi-square test. Continuous data was expressed as mean ± standard deviation. The diagnostic accuracy of the rapid urease test was determined by sensitivity, specificity, positive predictive value and negative predictive value. Kappa agreement was used to correlate the agreements between rapid urease test and histopatholigical findings. A ‘p’ value of less than or equal to 0.05 was considered as statistically significant.

Chapter 5

Results

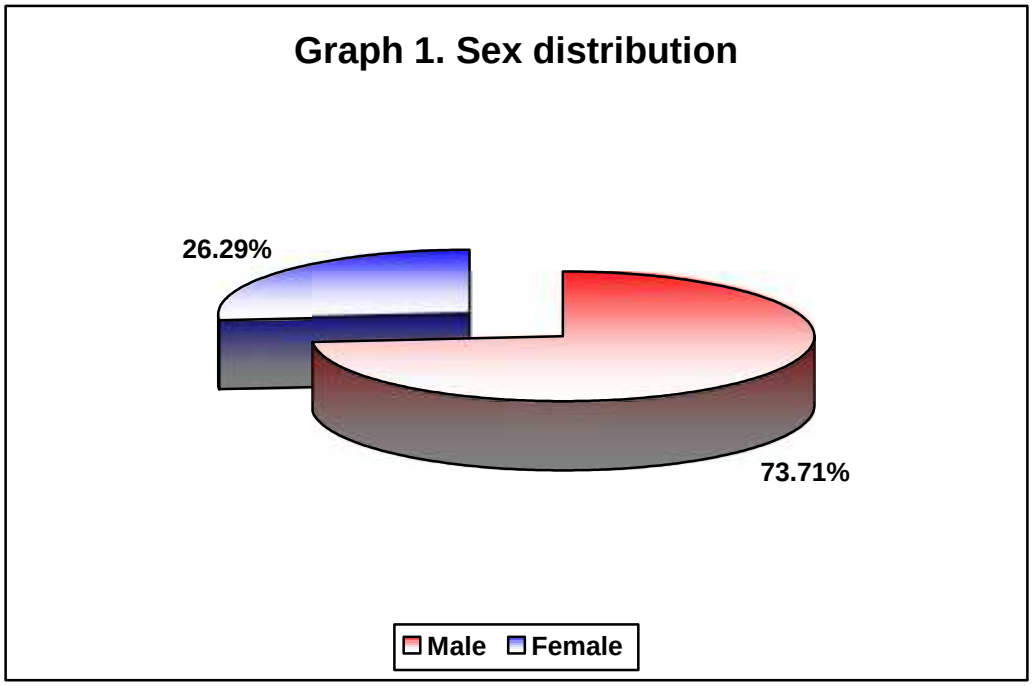
RESULTS

The present one year cross-sectional study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2011 to December 2011 A total of 175 patients undergoing upper gastro intestinal endoscopy were included in the study.

The data obtained was coded and entered in Microsoft Excel Spreadsheet. The analysis was done and final results were summarized as below.

Table 1. Sex distribution

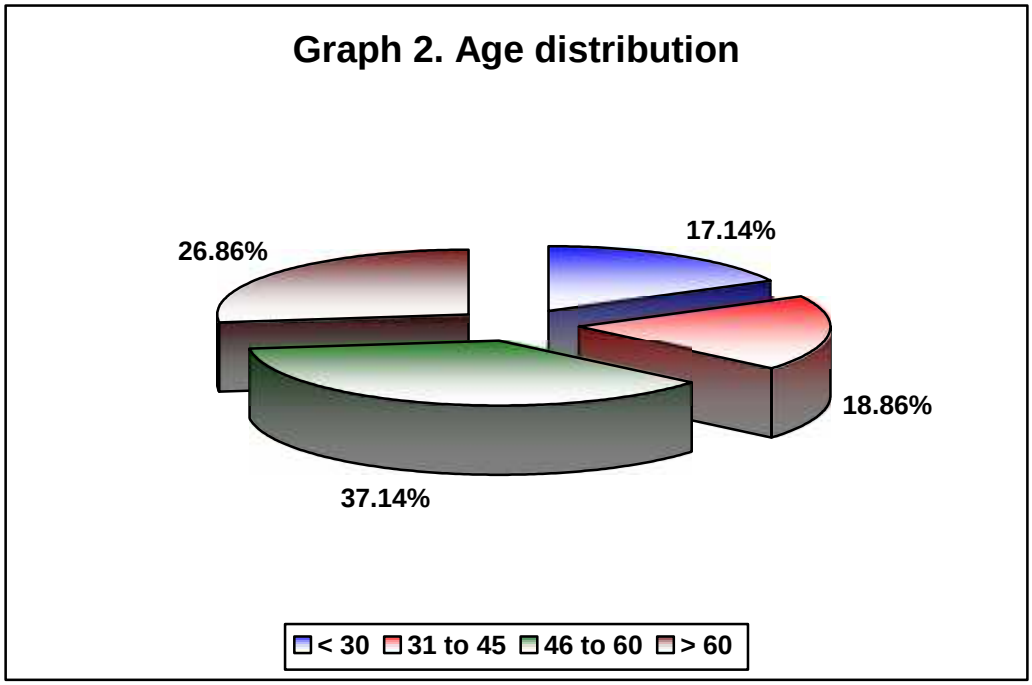
Sex	Distribution (n=175)	
	Number	**Percent**
Male	129	73.71
Female	46	26.29
Total	**175**	**100.00**



In the present study most of the patients (73.71%) were males. The male to female ratio was 2.80:1.

Table 2. Age distribution

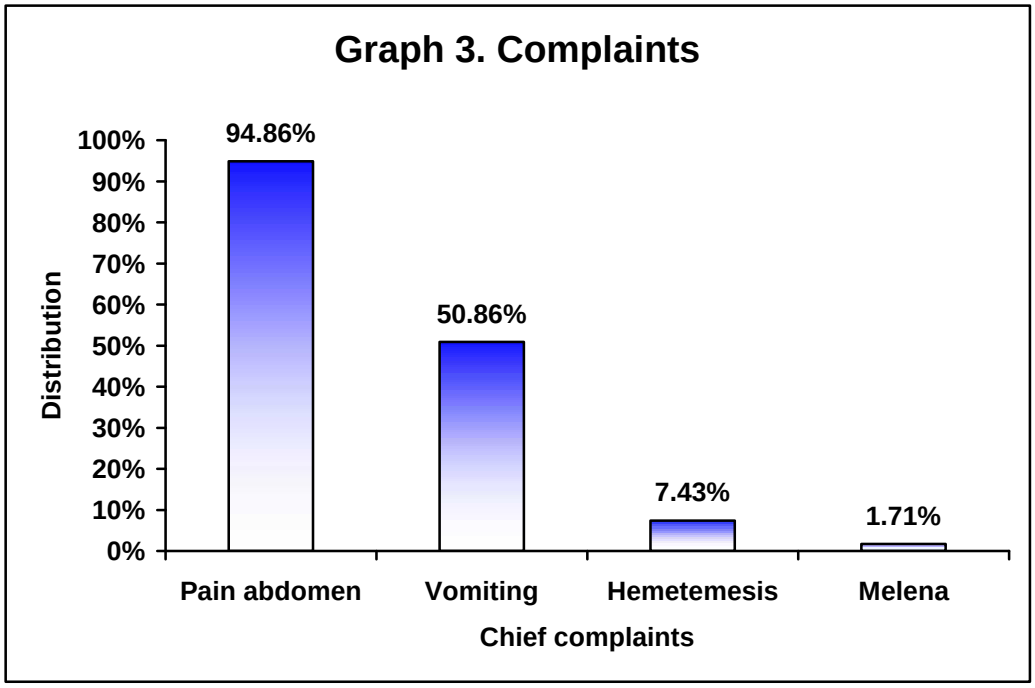
Age (Years)	Distribution (n=175)	
	Number	**Percent**
< 30	30	17.14
31 to 45	33	18.86
46 to 60	65	37.14
> 60	47	26.86
Total	**175**	**100.00**



In this study most of the patients were aged between 46 to 60 years (37.17%) followed by 26.86% were aged more than 60 years. However 18.86% patients were aged between 31 to 45 years and 17.14% had age less than 30 years. The mean age of the study population was 49.47 ± 15.22 years.

Table 3. Complaints

Chief complaints	Distribution (n=175)	
	Number	**Percent**
Pain abdomen	166	94.86
Vomiting	89	50.86
Hemetemesis	13	7.43
Melena	3	1.71



In the present study 94.86% of patients presented with pain abdomen. The next common complaint was vomiting which was present in 50.86%. However, hemetemesis and malaena were observed among 7.43% and 1.71% of patients respectively.

Table 4. Appetite

Appetite	Distribution (n=175)	
	Number	**Percent**
Normal	83	47.43
Increased	14	8.00
Decreased	78	44.57
Total	**175**	**100.00**

In this study normal appetite was recorded among 47.43% and it was decreased among 44.57% of patients. However increased appetite was present in 8% of patients.

Table 5. Diet

Diet	Distribution (n=175)	
	Number	**Percent**
Routine	92	52.57
Bland	36	20.57
Spicy	47	26.86
Total	**175**	**100.00**

In the present study The routine diet was reported by 52.57% patients whereas bland and spicy diet was reported by 20.57% and 26.86% patients respectively.

Table 6. Personal history

Personal history	Distribution (n=175)	
	Number	**Percent**
Tobacco chewing	36	20.57
Smoking	48	27.43
Alcoholism	62	35.43

In this study personal history of alcohol consumption was present in 35.43% patients, smoking was present in 27.43% and in 20.57% patients, history of tobacco chewing was noted.

Table 7. Built

Built	Distribution (n=175)	
	Number	**Percent**
Well	59	33.71
Moderate	92	52.57
Poor	24	13.71
Total	**175**	**100.00**

In the present study, more than half (52.57%) patients were built moderately whereas 33.71% were well built and 13.71% were poorly built.

Table 8. Nourishment

Nourishment	Distribution (n=175)	
	Number	**Percent**
Well nourished	126	72.00
Under nourished	49	28.00
Total	**175**	**100.00**

In this study 72% of the patients were well nourished and 28% were under nourished.

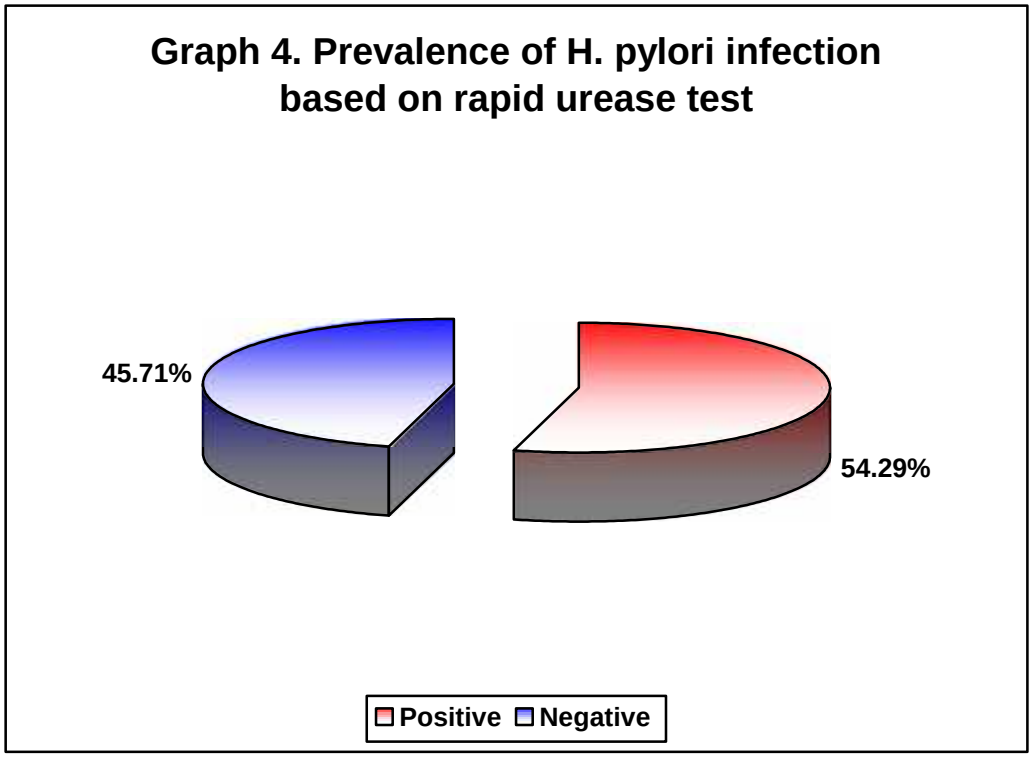
Table 9. Pallor

Pallor	Distribution (n=175)	
	Number	**Percent**
Present	52	29.71
Absent	123	70.29
Total	**175**	**100.00**

In this study pallor was present in 29.71% whereas 70.29% patients did not have pallor.

Table 10. Prevalence of *H. pylori* infection based on rapid urease test

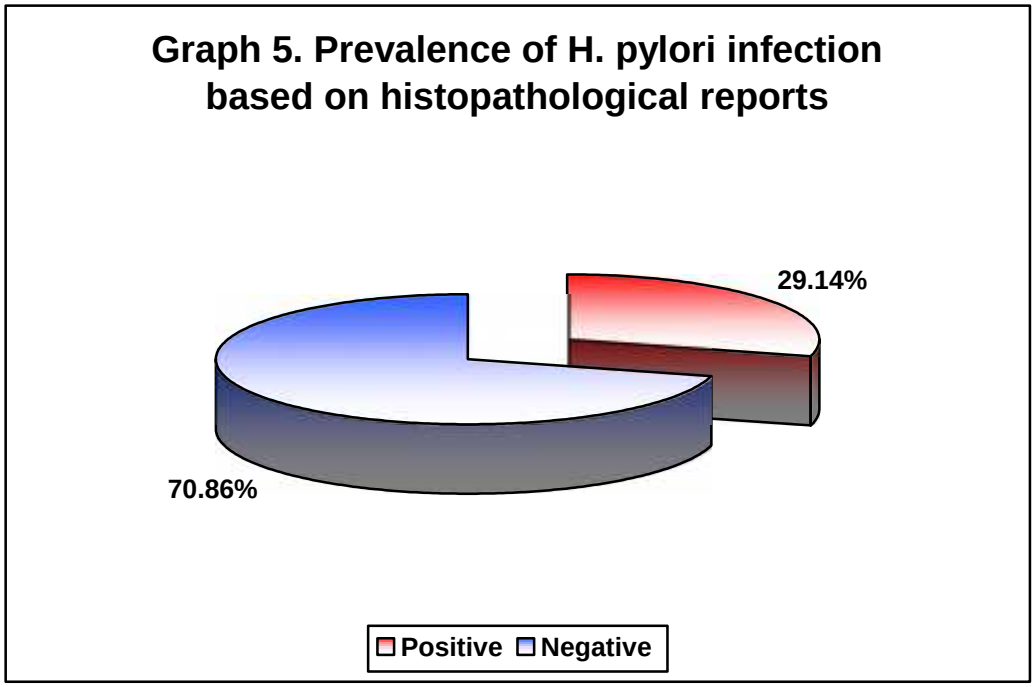
Results	Distribution (n=175)	
	Number	**Percent**
Positive	95	54.29
Negative	80	45.71
Total	**175**	**100.00**



In the present study based on rapid urease test prevalence of *H. pylori* infection was 54.29%.

Table 11. Prevalence of *H. pylori* infection based on histopathological reports

Infection	Distribution (n=175)	
	Number	Percent
Positive	51	29.14
Negative	124	70.86
Total	**175**	**100.00**

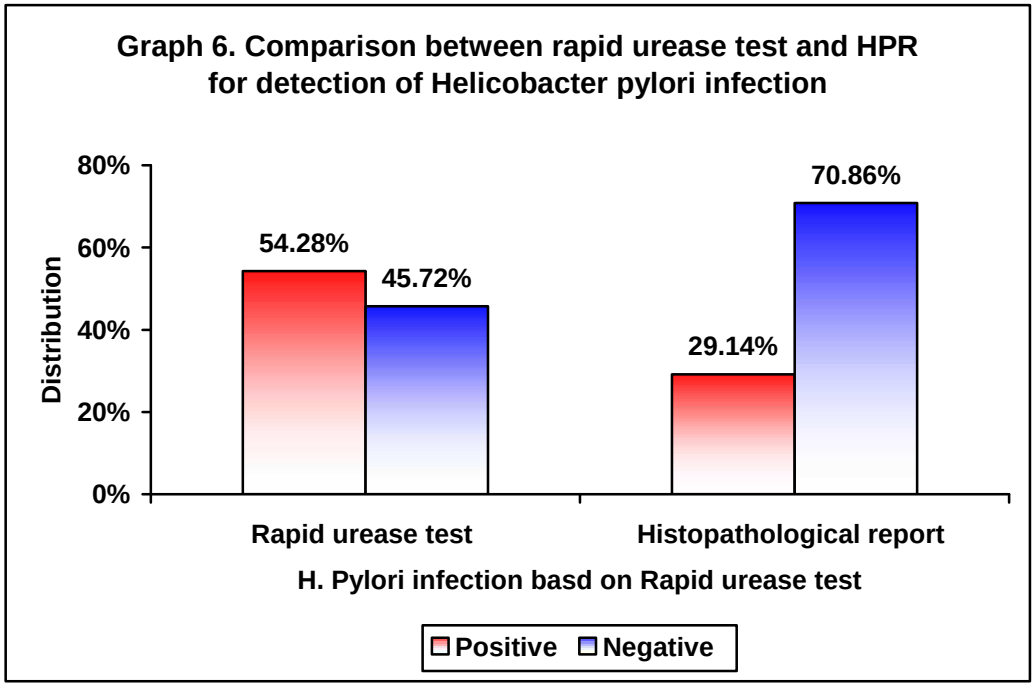


In this study based on histopathological reports the prevalence of *H. pylori* infection was 29.14%.

Table 12. Comparison between rapid urease test and HPR for detection of *H. pylori* infection

H. Pylori infection based on Rapid urease test	H. Pylori infection based on Histopathological report				Total	
	Present (n=51)		Absent (n=124)			
	Number	Percent	Number	Percent	Number	Percent
Positive (n=95)	45	88.24	50	40.32	95	54.28
Negative (n=80)	6	11.76	74	59.68	80	45.71
Total	**51**	**29.14**	**124**	**70.85**	**175**	**100**

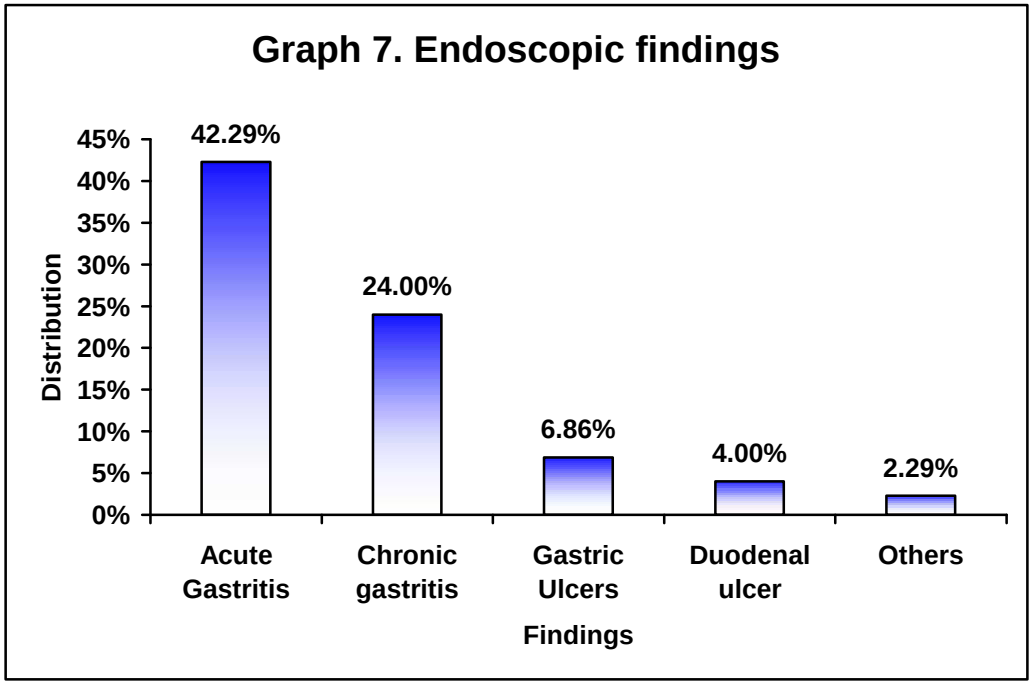
Sensitivity of rapid urease test =88.2%; Specificity=59.6%; PPV=47.3%; NPV=92.5%; Diagnostic Accuracy of rapid urease test =68%; k = 0.382; p < 0.001



In the present study rapid urease test showed 88.2% sensitivity and 59.6% specificity, 47.3% positive predictive value and 92.5% negative predictive value in diagnosing H. pylori infection when compared with histopathology.

Table 13. Endoscopic findings

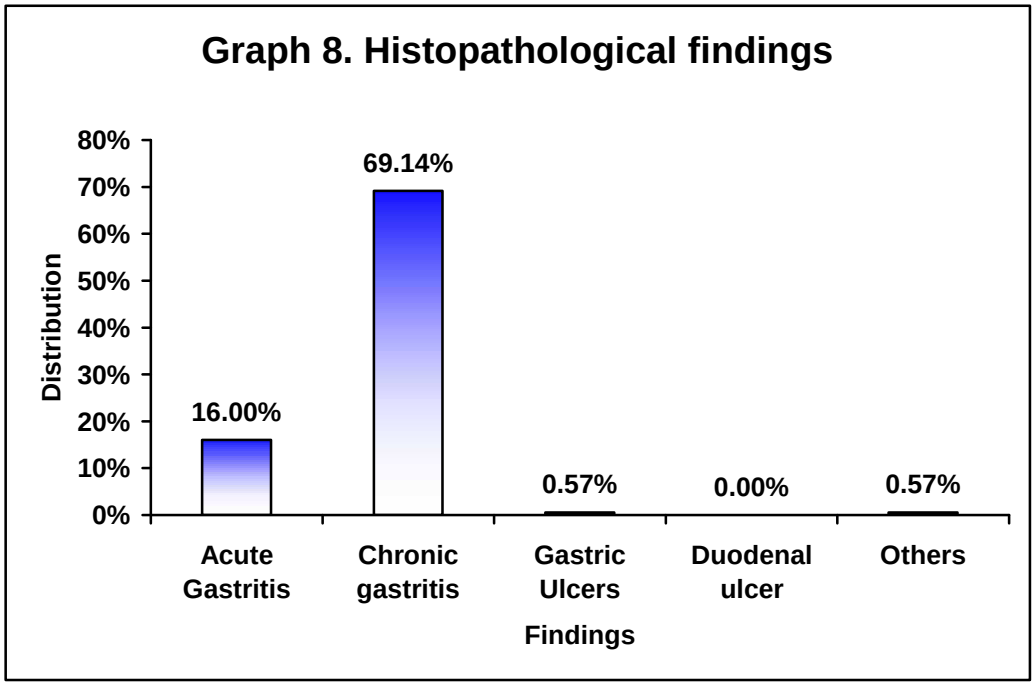
Findings	Distribution (n=175)	
	Number	**Percent**
Gastritis		
Acute Gastritis	74	42.29
Chronic gastritis	42	24.00
Ulcers		
Gastric Ulcers	12	6.86
Duodenal ulcer	7	4.00
Others	4	2.29
Normal	**47**	**26.86**



In the present study endoscopic findings revealed acute gastritis in 42.29% patients and chronic gastritis in 24% patients.

Table 14. Histopathological findings

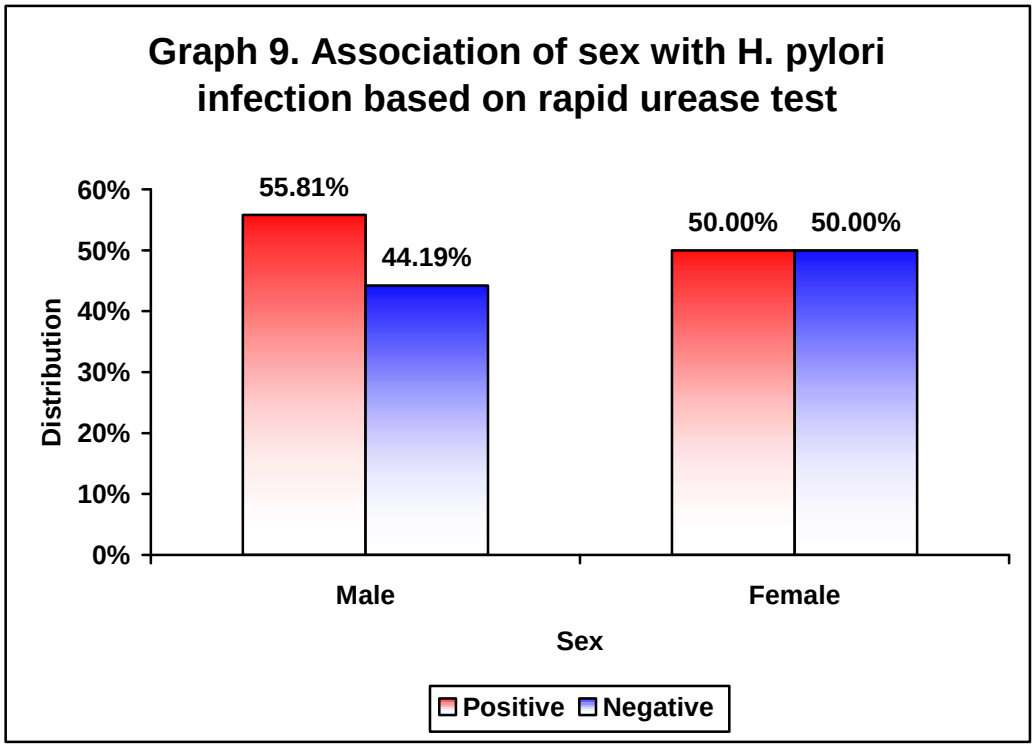
Findings	Distribution (n=175)	
	Number	**Percent**
Gastritis		
Acute Gastritis	28	16.00
Chronic gastritis	121	69.14
Ulcers		
Gastric Ulcers	1	0.57
Duodenal ulcer	0	0.00
Others	1	0.57
Normal	**26**	**14.86**



In this study histopathological findings showed chronic gastritis among 69.14% patients and acute gastritis in 16% of patients.

Table 15. Association of sex with *H. pylori* infection based on rapid urease test

Sex	H. Pylori infection based on Rapid urease test					
	Positive (n=95)		Negative (n=80)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
Male	72	55.81	57	44.19	129	100.00
Female	23	50.00	23	50.00	46	100.00
x^2=0.462			dF=1		p=0.497	

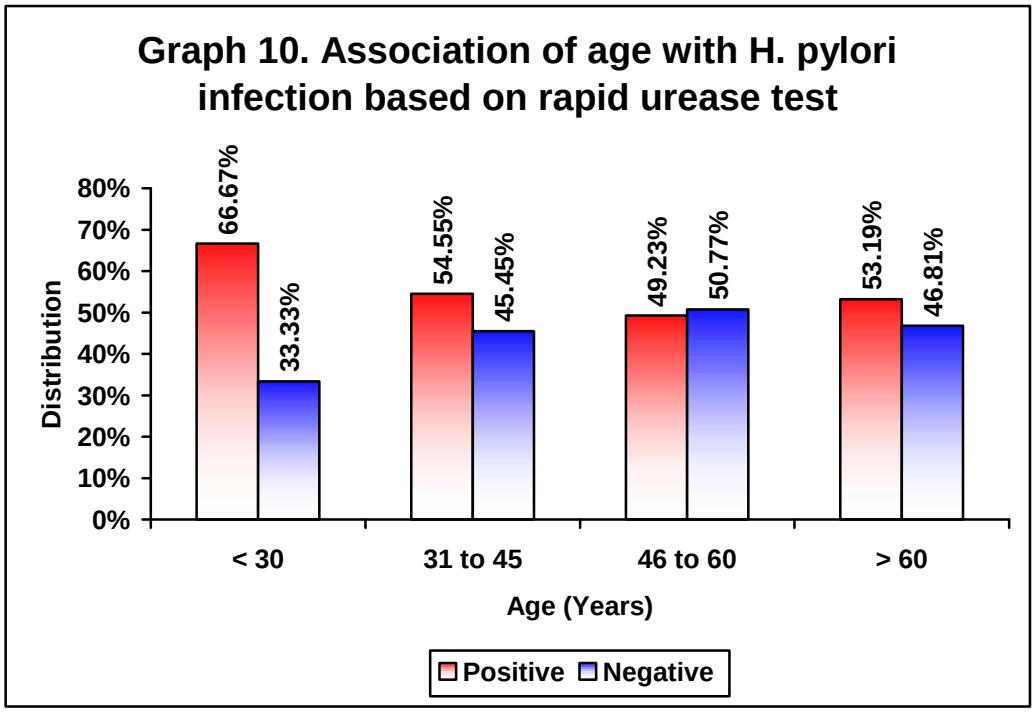


In the present study 55.81% of the males and 44.19% females had *H. pylori* infection based on rapid urease test. However this difference was statistically not significant (p=0.497).

Table 16. Association of age with *H. pylori* infection based on rapid urease test

Age (Years)	H. Pylori infection based on Rapid urease test					
	Positive (n=95)		Negative (n=80)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
< 30	20	66.67	10	33.33	30	100.00
31 to 45	18	54.55	15	45.45	33	100.00
46 to 60	32	49.23	33	50.77	65	100.00
> 60	25	53.19	22	46.81	47	100.00

$x^2 = 2.546$ dF = 3 p = 0.467

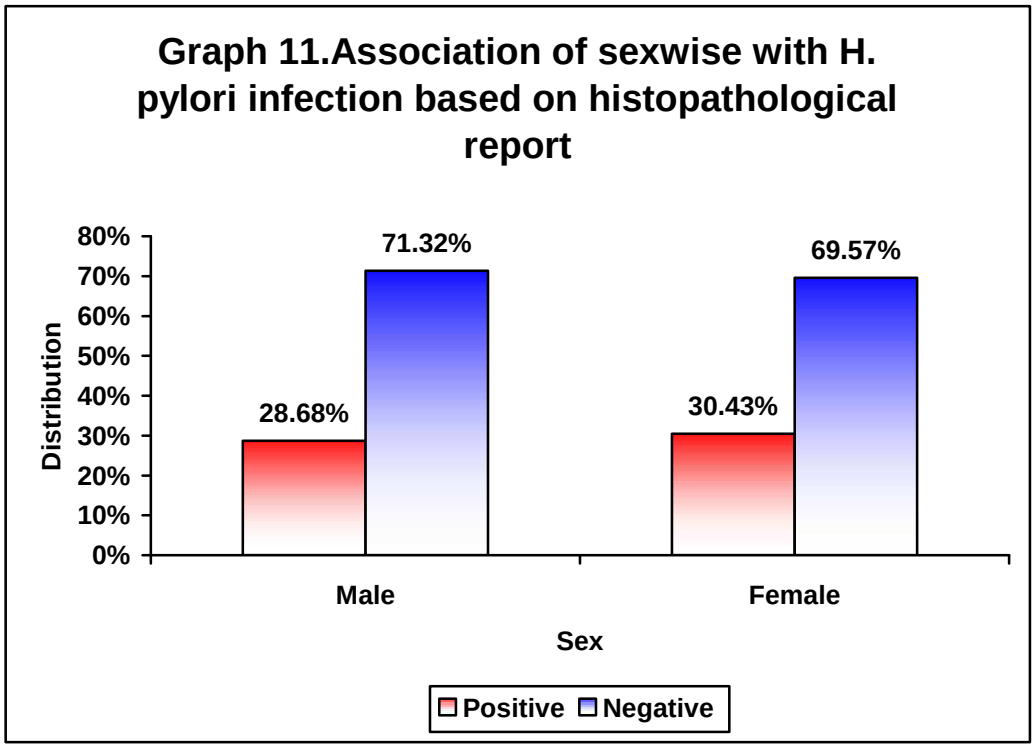


In the present study 66.67% patients with *H.pylori* infection were aged less than 30 years and 33.33% patients in the same age group did not have *H. pylori* infection based on rapid urease test. However, no statistically significant difference of *H. pylori* infection was observed between the different age groups.

Table 17. Association of sex with *H. pylori* infection based on histopathological report

Sex	H. Pylori infection based on Histopathological report					
	Positive (n=51)		Negative (n=124)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
Male	37	28.68	92	71.32	129	100.00
Female	14	30.43	32	69.57	46	100.00
Total	**51**	**59.12**	**124**	**140.88**	**175**	**200.00**

$x^2 = 0.050$ dF = 1 p = 0.822

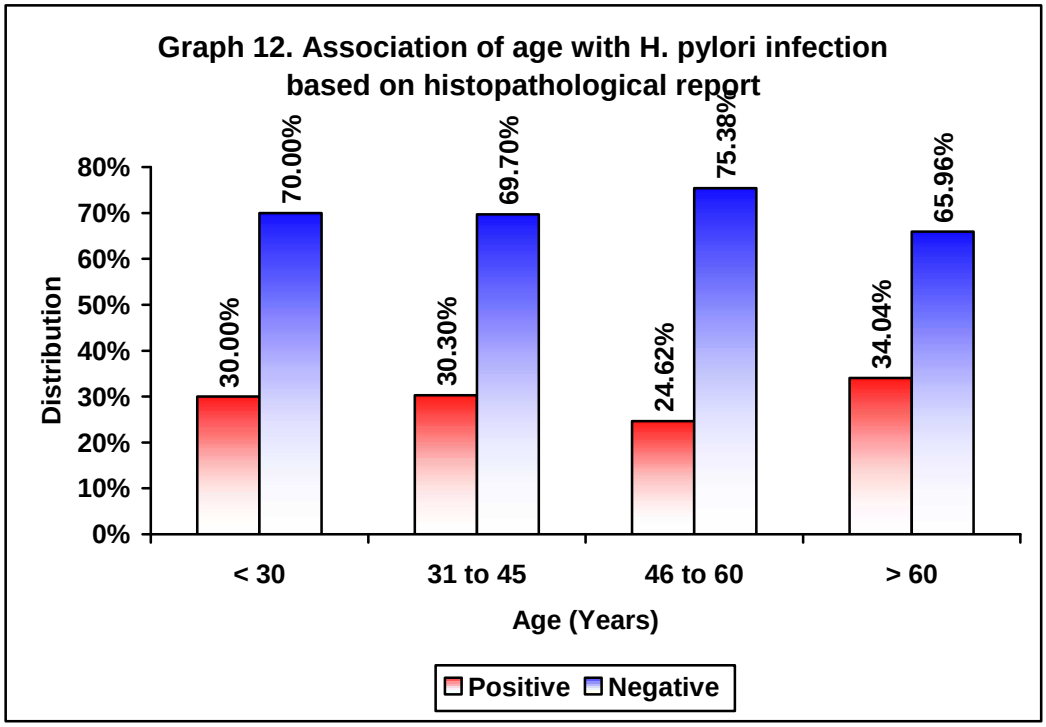


In the present study based on histopathological reports 28.68% of the males and 71.32% of females had *H. pylori* infection. However this difference was statistically not significant (p=0.822).

Table 18. Association of age with *H. pylori* infection based on histopathological report

Age (Years)	H. Pylori infection based on Histopathological report					
	Positive (n=51)		Negative (n=124)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
< 30	9	30.00	21	70.00	30	100.00
31 to 45	10	30.30	23	69.70	33	100.00
46 to 60	16	24.62	49	75.38	65	100.00
> 60	16	34.04	31	65.96	47	100.00

x^2 = 1.224 dF = 3 p = 0.747



In the present study based on histopathological report 30% of patients with *H.pylori* infection each were aged less than 30 years and 31 to 45 years whereas majority of the patients aged between 46 to 60 year did not have *H. pylori* infection. This difference between the prevalence among different age groups was statistically not significant (p=0.747).

Table 19. Endoscopic findings and prevalence of *H. pylori* according to rapid urease test

Findings	Positive (n=51)		Negative (n=124)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
Gastritis						
Acute Gastritis	42	56.76	32	43.24	74	100.00
Chronic gastritis	23	54.76	19	45.24	42	100.00
Ulcers						
Gastric Ulcers	6	50.00	6	50.00	12	100.00
Duodenal ulcer	6	85.71	1	14.29	7	100.00
Others	1	25.00	3	75.00	4	100.00
Normal	23	48.94	24	51.06	47	100.00

In this study on endoscopic findings revealed 74 patients with acute gastritis. Among them, *H. pylori* infection was present in 56.76% patients based on rapid urease test and of the 42 patients with chronic gastritis in 54.76% had *H. pylori* infection whereas, of 47 patients among the endoscopy revealed normal findings, 48.94% had *H. pylori* infection.

Table 20. Association of *H. pylori* infection based on rapid urease test with gastritis (Based on histopathological findings)

Findings	Positive (n=51)		Negative (n=124)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
Gastritis						
Acute Gastritis	12	42.86	16	57.14	28	100.00
Chronic gastritis	70	57.85	51	42.15	121	100.00
Ulcers			0			
Gastric Ulcers	0	0.00	1	100.00	1	100.00
Duodenal ulcer	0	0.00	0	0.00	0	0.00
Others	0	0.00	1	100.00	1	100.00
Normal	13	50.00	13	50.00	26	100.00
x^2 = 2.072					p = 0.150	

In the present study histopathological findings revealed 28 patients with acute gastritis. Among them, *H. pylori* infection was present in 42.86% patients based on rapid urease test and of the 121 patients with chronic gastritis in 57.85% had *H. pylori* infection. In 26 patients with normal histopathological report, 50% had *H. pylori* infection. However this variation in the prevalence of *H. pylori* infection among normal individuals and patients with gastritis was statistically not significant (p=0.150)

Table 21. Endoscopic findings and prevalence of H. pylori according to histopathological report

Findings	Positive (n=51)		Negative (n=124)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
Gastritis						
Acute Gastritis	21	28.38	53	71.62	74	100.00
Chronic gastritis	12	28.57	30	71.43	42	100.00
Ulcers						
Gastric Ulcers	4	33.33	8	66.67	12	100.00
Duodenal ulcer	4	57.14	3	42.86	7	100.00
Others	0	0.00	4	100.00	4	100.00
Normal	13	27.66	34	72.34	47	100.00

In this study on endoscopic findings revealed 74 patients with acute gastritis. Among them, *H. pylori* infection was present in 28.38% patients based on histopathological report and of the 42 patients with chronic gastritis in 28.57% had *H. pylori* infection whereas, in 47 patients, among whom the endoscopy revealed normal findings, 27.66% had *H. pylori* infection.

Table 22. Association of *H. pylori* infection based on histopathological report with gastritis (based on histopathological findings)

Findings	Positive (n=51)		Negative (n=124)		Total (n=175)	
	Number	Percent	Number	Percent	Number	Percent
Gastritis						
Acute Gastritis	9	32.14	19	67.86	28	100.00
Chronic gastritis	33	27.27	88	72.73	121	100.00
Ulcers						
Gastric Ulcers	0	0.00	1	100.00	1	100.00
Duodenal ulcer	0	0.00	0	0.00	0	0.00
Others	0	0.00	1	100.00	1	100.00
Normal	9	34.62	17	65.38	26	100.00
x^2=0.221			**dF=1**		**p=0.605**	

In the present study histopathological findings revealed 28 patients with acute gastritis. Among them, *H. pylori* infection was present in 32.14% patients based on histopathological reports and of the 121 patients with chronic gastritis, in 27.27% had *H. pylori* infection. In 26 patients with normal histopathological findings, 34.62% patients had *H. pylori* infection. However this difference in the prevalence of *H. pylori* infection among normal individuals and patients with gastritis was statistically not significant (p=0.605)

Chapter 6

Discussion

DISCUSSION

Helicobacter pylori is a gram negative, curved, microaerophilic and motile organism with multiple polar flagella. It resides in the stomach of man and other primates, lining up the gastric mucus secreting cells. Helicobacter pylori is a common bacterium infecting about half the world's population. The prevalence of H. pylori infection varies widely by geographic area, age, race, ethnicity, and socio-economic status. Rates appear to be higher in developing than in developed countries.[34]

Helicobacter pylori has proved to be of overwhelming importance in the aetiology of a number of common gastrointestinal diseases such as chronic gastritis, peptic ulceration (90% of duodenal ulcers and 80% of gastric ulcers) and gastric cancer and is a major cause of morbidity in infected patients. High prevalence of the acid peptic diseases as a result of H pylori has negative impact on patient quality of life and also poses economical strains. Loss of working days due to acid peptic disease adds to economical burden. Early detection of H pylori in any population and its eradication in such patients results in a significant reduction in usage of acid suppression and an improvement in overall quality and severity of dyspeptic symptoms.[34]

Thus, it is important to find out regional H pylori prevalence and identify high risk population infected with H pylori so that treatment strategies can be planned and implemented is such patients to reduce the menace of this disease. Despite the fact of high prevalence of *H. Pylori* infection there is scarcity of literature regarding prevalence of *H. Pylori* infection in this region. Hence the

present study was undertaken to estimate the prevalence of H. pylori and know its association with gastritis.

The present one year cross-sectional study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2011 to December 2011 on 175 patients undergoing upper gastro intestinal endoscopy. A total of 175 patients undergoing upper gastro intestinal endoscopy were included in the study. Presence of H. pylori infection was diagnosed by Rapid urease test and HPR.

In the present study most of the patients (73.71%) were males. The male to female ratio was 2.80:1 (Table 1, Graph 1). Most of the patients were aged between 46 to 60 years (37.17%) The mean age of the study population was 49.47 ± 15.22 years (Table 2, Graph 2). A similar study[34] from Jammu and Kashmir, India reported 64.13% of males and 35.86% females with a mean age of 43.07±11.76 but the maximum numbers of patients were in the age group of 36-45 years.

In this study 94.86% of patients presented with pain abdomen(Table 3, Graph 3). The next common complaint was vomiting which was present in 50.86%. Normal appetite was recorded among 47.43% and it was decreased among 44.57% of patients (Table 4). The bland and spicy diet was reported by 20.57% and 26.86% patients respectively (Table 5). History of alcohol consumption was present in 35.43% patients, smoking was present in 27.43% and in 20.57% patients, history of tobacco chewing was noted (Table 6). More than half (52.57%) patients were built moderately whereas 33.71% were well built

(Table 7). 72% of the patients were well nourished and 28% were under nourished (Table 8). Pallor was present in 29.71% (Table 9). A study[34] from Jammu and Kashmir, India reported pain in upper abdomen as the most frequent symptom in 54.20% of patients followed by vomiting in 11.80% patients.

In the present study the prevalence of *H. pylori* infection was 54.29% based on Rapid urease test (Table 10, Graph 4) and Histopathological studies revealed a prevalence of 29.14% (Table 11, Graph 5).

Globally, *H. pylori* infection affects 50% of the population. Rates have been suggested to be higher in developing than in developed countries in previous studies. In Asia, the prevalence of *Helicobacter pylori* (*H. pylori*) infection varies markedly in different countries. Higher prevalence rates are found in developing Asian countries while lower rates have been reported in more industrialized and developed countries. Within a country, the seroprevalence rates may vary between distinct geographic regions.[72]

However, recently even in developed countries like Northeastern Mexico high H. pylori prevalence in symptomatic patients has been reported.[37]

In one of the Indian study[36] from Chandigarh, 254 individuals were screened for H. pylori. There were 80 symptomatic and 67 asymptomatic individuals. Helicobacter pylori was positive in 38 (56.7%) asymptomatic and 49 (61.3%) symptomatic individuals ($p>0.05$). Helicobacter pylori was present in 11/13 (84.6%) subjects with peptic ulcer.

Similarly in other Indian study,[35] H pylori prevalence in patients with dyspepsia and in control subjects was 65% and 46% respectively. The prevalence based on Rapid urease test recorded in our study appears to be similar in comparison to the recent studies from India[36,35] and outside India.[37] Almost all the patients were symptomatic. However the prevalence based on Histopathological evaluation was less in this study

In the present study, rapid urease test showed diagnostic accuracy of 92.5% based on the sensitivity of 88.2%, 59.6% specificity, 47.3% positive predictive value and 92.5% negative predictive value in diagnosing H. pylori infection when compared with HPR (Table 12, Graph 6).

Histopathologic evaluation has traditionally been the gold standard method for diagnosing Helicobacter pylori infection. The disadvantage of this technique is the need for endoscopy to obtain tissue. Limitations also arise at times because of an inadequate number of biopsy specimens obtained or failure to obtain specimens from different areas of the stomach. In some cases, different staining techniques may be necessary, which can involve longer processing times and higher costs.[21] Some of these reasons have probably been the reason for low prevalence of H. pylori infection based on histopathological evaluation in this study.

Rapid urease testing takes advantage of the fact that Helicobacter pylori is a urease producing organism. Samples obtained on endoscopy are placed in urea-containing medium; if urease is present, the urea will be broken down to carbon dioxide and ammonia, with a resultant increase in the pH of the medium and a

subsequent color change in the pH dependent indicator. This test has the advantages of being inexpensive, fast, and widely available. It is limited, however, by the possibility of false positive results; decreased urease activity, caused either by recent ingestion of antibiotic agents, bismuth compounds, proton pump inhibitors, or sucralfate or by bile reflux, can contribute to these false-negative results.[21]

The rapid urease test is widely used in diagnosis of *H. pylori* infection all over the world due to a number of accompanying advantages, including less expense and more rapid results compared to histology or culture. Further, RUT has been shown to have high sensitivity, specificity and clinical accuracy. A study[73] reported a sensitivity, specificity, positive predictive value, negative predictive value and diagnostic accuracy of 98, 100, 100, 98 and 99%, respectively for RUT. However, the test has been shown to be less sensitive in case of concurrent use of proton pump inhibitors[74] bismuth and anti-helicobacter antibiotics. The test is also influenced by the pH of the gastric mucosa.[75]

In this study, 55.81% of the males and 44.19% females had *H. pylori* infection based on rapid urease test (Table 15, Graph 9). Similarly histopathological reports revealed, 28.68% of the males and 71.32% of females with *H. pylori* infection (Table 17, Graph 11). However this difference sex and H. pylori infection using both the tests was statistically not significant ($p>0.050$) lacking association of sex with *H. pylori* infection. Most of the patients (66.67%) with *H. pylori* infection were aged less than 30 years and 33.33% patients in the same age group did not have *H. pylori* infection based on rapid urease test (Table 16, Graph 10). Also, histopathological findings showed 30% of patients with *H.*

pylori infection each with age less than 30 years and between 31 to 45 years whereas majority of the patients aged between 46 to 60 year did not have *H. pylori* infection (Table 18, Graph 12). This difference between the prevalence of *H. pylori* infection by both rapid urease test and histopathology among different age groups was statistically not significant (p>0.050).

H. pylori infection has no sex predilection. *H pylori* infection may be acquired at any age. According to some epidemiologic studies, this infection is acquired most frequently during childhood. Children and females have a higher incidence of reinfection (5-8%) than adult males.[31] The findings of the present study also varied from studies from out side and from India as female/male ratio of 1.44:1 and mean age of 53 years was recorded in study from northeastern Mexico[37] whereas, in Indian study[35] age-related prevalence in the age groups of 10-19 years, 20-29 years, 30-39 years, 40-49 years and > or = 50 years were 52%, 70%, 69%, 60% and 59%, respectively. A study[34] from Jammu, India reported among H. pylori positive patients, 64.13% were males and 35.86% were females. Age wise distribution showed maximum prevalence of H. pylori infection in the age group of 36-45 years and minimum in the age group of 66-75 years.[34]

In this study endoscopic findings revealed acute gastritis in 42.29% patients and chronic gastritis in 24% patients (Table 12, Graph 7). The histopathological findings showed chronic gastritis among 69.14% patients and acute gastritis in 16% of patients (Table 14, Graph 8).

In the present study endoscopic findings revealed 74 patients with acute gastritis. Among them, based on rapid urease test *H. pylori* infection was present

in 56.76% patients and of the 42 patients with chronic gastritis 54.76% had *H. pylori* infection. Whereas of 47 patients who had normal endoscopic findings, 48.94% patients had *H. pylori* infection (Table 19). Similarly based on the histopathological report *H. pylori* infection was present in 28.38% and 28.57% of patients with acute gastritis and chronic gastritis respectively. Whereas in 47 patients among whom the endoscopy revealed normal findings, 27.66% had *H. pylori* infection (Table 21).

In this study histopathological findings revealed 28 patients with acute gastritis. Based on rapid urease test, *H. pylori* infection was present in 42.86% patients and of the 121 patients with chronic gastritis 57.85% patients had *H. pylori* infection. In 26 patients with normal histopathological report, 50% had *H. pylori* infection. However this variation in the prevalence of *H. pylori* infection among normal individuals and patients with gastritis was statistically not significant (p=0.150) (Table 20). Similarly based on histopathological report, H. pylori infection was present in 32.14% patients and of the 121 patients with chronic gastritis, in 27.27% patient *H. pylori* infection present. Among 26 patients with normal histopathological findings, 34.62% patients had *H. pylori* infection. However this difference in the prevalence of *H. pylori* infection among normal individuals and patients with gastritis was statistically not significant (p=0.605) (Table 22).

The primary disease caused by H pylori is gastritis. Not all infected persons have symptoms, but all show changes in the gastric mucosa ("chronic superficial gastritis"). H pylori are well adapted to survive in the hostile environment of the stomach. Their spiral shape allows them to corkscrew down

through the mucous layer to the gastric mucosa; they attach to mucous-secreting cells that line the stomach; they break down urea to produce ammonia that helps neutralize gastric acid in their immediate vicinity; and they produce various proteins that damage mucosal cells, attracting lymphocytes (which may be their primary source of nutrients) and causing persistent inflammation. After years or even decades of chronic superficial gastritis, carriers develop lesions ("atrophic gastritis") and eventually the stomach tissue can become abnormal and precancerous.[64]

In early studies, Warren and Marshall found H pylori in 65% of patients with gastritis, 85% of gastric ulcer patients, and all of duodenal ulcer patients. Marshall and Warren's discovery founded the concept that infection with H. pylori , and not (if at all, very indirectly) stress, can lead to a variety of upper gastrointestinal disorders such as gastric inflammation (gastritis), peptic ulcer disease (10%–20%), distal gastric adenocarcinoma (1%-2%), and gastric mucosal-associated lymphoid tissue (MALT) lymphoma (<1%).[76]

A study[34] from Jammu and Kashmir, India on 265 patients reported endoscopic and histopathological features of chronic superficial gastritis as the most common feature seen in 87 patients. Duodenitis and oesophagitis were the other common findings documented in 11 and 8 patients, respectively. A single chronic gastric ulcer was in two and acute duodenal ulcer in four patients.

In our study the association between H. pylori infection and Gastritis could not be established. Out of the 175 patients 149 patients had gastritis (Acute gastritis plus Chronic gastritis) according to HPR in which 82 had H. pylori

infection whereas 67 did show H. pylori infection (according to rapid urease test). Only 26 patients had normal finding on HPR among which, 13 patients had H. pylori infection whereas 13 did not show H pylori infection (according to rapid urease test).

Similarly out of the 175 patients 149 patients had gastritis (Acute gastritis plus Chronic gastritis) according to HPR in which in which 42 had H. pylori infection and 107 did not show H. pylori infection (based on HPR findings). Of the 26 patients with normal findings on HPR, 9 had H. pylori infection and 17 did not show H. pylori infection (based on HPR findings). Hence H. pylori as a cause for gastritis could not be established in this study.

Chapter 7

Conclusion

CONCLUSION

In the present study based on rapid urease test prevalence of *H. pylori* infection was 54.29% and histopathological studies revealed 29.14% of patients with *H. pylori* infection.

The association of *H. pylori* infection with gastritis could not be established in this study.

Chapter 8

Summary

SUMMARY

Helicobacter pylori (H. pylori) infection is the principal cause of chronic active gastritis and peptic ulcer disease and a major contributor for gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma. The present study was undertaken to estimate the prevalence of H. pylori and its association with gastritis.

The present one year cross-sectional study was conducted in the Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period, from January 2011 to December 2011 A total of 175 patients undergoing upper gastro intestinal endoscopy were included in the study. Two biopsy specimens were obtained from the antrum. Biopsy was also taken from any suspicious lesions, if noted. One biopsy specimen from the antrum was used for rapid urease test and the remaining biopsy specimen sent for HPR.

In the present study most of the patients (73.71%) were males. The male to female ratio was 2.80:1. Most of the patients were aged between 46 to 60 years (37.17%). The mean age of the study population was 49.47 ± 15.22 years. 94.86% of patients presented with pain abdomen. The next common complaint was vomiting which was present in 50.86%. Based on rapid urease test prevalence of *H. pylori* infection was 54.29% and histopathological reports revealed 29.14% prevalence of *H. pylori* infection. Rapid urease test showed 88.2% sensitivity and 59.6% specificity, 47.3% positive predictive value and 92.5% negative predictive value in diagnosing H. pylori infection when

compared with histopathology. The endoscopic findings revealed acute gastritis in 42.29% patients and chronic gastritis in 24% patients. Based on histopathological findings, chronic gastritis was present among 69.14% patients and acute gastritis in 16%.

The association of *H. pylori* infection with gastritis could not be established in this study.

Chapter 9

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Annexures

Annexure I

ANNEXURE I – CONSENT FORM

Mr / Mrs / Miss _____ we are requesting you to enrol yourself in study entitled, "**PREVALENCE OF H. PYLORI INFECTION IN PATIENTS UNDERGOING UPPER GASTRO INTESTINAL ENDOSCOPY AT KLES DR. PRABHAKAR KORE HOSPITAL – A ONE YEAR CROSS-SECTIONAL STUDY"** is being conducted by Dr. ***** ******, Post Graduate in Surgery at Jawaharlal Nehru Medical College Belgaum, Karnataka. Under guidance of Dr. ***** ***** Vice-Principal and Professor, Department of Surgery, Jawaharlal Nehru Medical College, Belgaum, under KLE University, Belgaum.

Respected Sir/Madam, we request you to enroll yourself to participate in our study as you are eligible for participating in this study. During the study you will be asked some questions regarding your present complaints and your are suppose to answer to the best of your knowledge.

Your participation in research is voluntary. If you decide to participate you are free to withdraw at any time.

The purpose of research is to determine the prevalence of H. Pylori infection in patients undergoing upper gastro intestinal endoscopy and to evaluate the relationship between H. Pylori infection and gastritis.

Procedure involved

If you agree to enroll yourself in this study, you will be informed in detail about the procedure. You will be interviewed regarding your present, past and

family history then you will be clinically examined in detail and investigated accordingly. You would be kept nil by mouth six hours before the procedure. An upper gastro intestinal endoscopy will be done and findings will be noted on predesigned and pretested proforma. Two biopsy specimens will be obtained from the antrum and/or suspicious lesions. One biopsy specimen will be used for rapid urease test and other biopsy specimen will be sent for HPR.

Benefits and Risks

The benefits of taking part in this research are you will have reduced post operative pain and post defecation pain after surgery. The no observable risks associated with this study.

Voluntary participation / Withdrawal

Taking part in the study is voluntary. You may choose not to enrol yourself in this study. Your decision will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Alternatives

Even if you decline the participation in the study, you will get the routine line of management.

Privacy and confidentiality

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you

during the research will be disclosed to other without your written permission except in emergency to protect your rights and welfare or if required by law.

Authorization to Publish Results

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

Financial Incentives for participation

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Compensation

In the event of injury, related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. There is compensation or payment for such medical treatment by law.

Questions/Contact details

If you have any queries, in future or in case of study related injury or illness, you may contact. Dr. **** ****** at Department of Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum Phone Number ***** *******or on ***** ******.

If you have any queries about your rights as a study subject, you may call

Principal and Chairman, J. N. Medical College Institutional Ethical Committee for Human Subjects Research, Ph. ***** ***** at J. N. Medical College, Belgaum.

CONSENT TO PARTICIPATE IN A RESEARCH STUDY:

I, Mr./Mrs. _____

voluntarily agree to take part in this study, by signing this consent form I am not giving up my legal rights. I may withdraw at any time. I am signing after having read, or been read to me in the vernacular language including risks and the benefits and having all queries cleared.

Subject Name: _____

Signature of the participant _____ Date _____
Or Left thumb print

Witness name: _____

Signature: _____ Date _____

Investigator's name: _____

Signature: _____ Date _____

Place: _____

Annexures

Annexure II

ANNEXURE II – PROFORMA

STUDY: PREVALENCE OF H. PYLORI INFECTION IN PATIENTS UNDERGOING UPPER GASTRO INTESTINAL ENDOSCOPY AT KLES DR. PRABHAKAR KORE HOSPITAL – A ONE YEAR CROSS-SECTIONAL STUDY.

PATIENT DETAILS

Name	:	IP/OP No	:
Sex	:	Age	:

Chief Complaints

Pain in abdomen	:	Yes / No
Vomiting	:	Yes / No
Haematemesis	:	Yes / No
Malena	:	Yes / No

Personal history

Appetite	:	Normal / Increased / Decreased
Diet	:	Routine / Bland / Spicy
Tobacco chewing	:	
Smoking	:	
Alcoholism	:	

On Examination

Built	:	Well nourished – 1/ Moderately built – 2/ Poorly built - 3
Nourishment	:	Well nourished - 1/ Under nourished - 2

Haemodynamic status: BP:

Pulse:

Pallor : Present - 1/Absent - 2

Investigations

Rapid urease test :

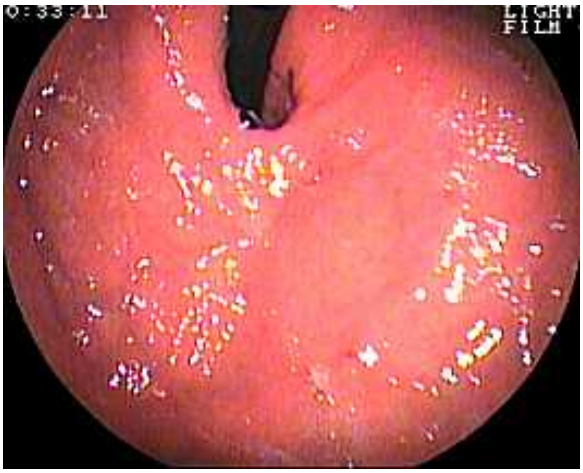
Endoscopic impression:

Histopathology Report:

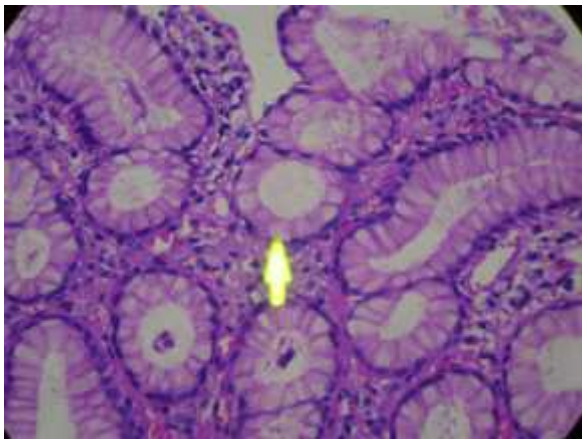
Annexures

Annexure III

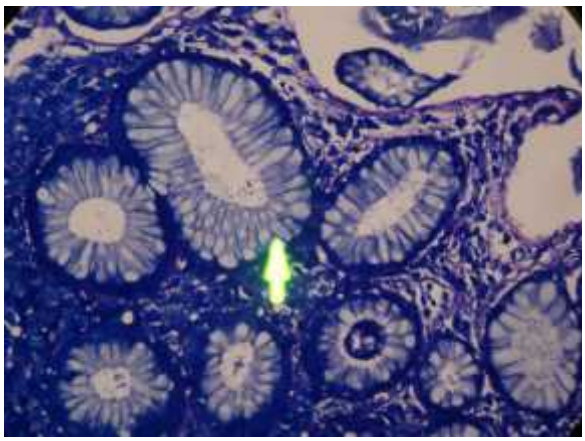
ANNEXURE III – PHOTOGRAPHS



Photograph 4. Endoscopic finding suggestive of chronic gastritis



Photograph 5. Rod shaped structure on H & E stain showing *H. Pylori* infection



Photograph 6. Blue coloured rods on Giemsa stain showing *H. pylori* infection

Annexures

Annexure IV

ANNEXURE IV – MASTER CHART

AB	- Absent
AE	- Antral erosions
AEG	- Acute erosive gastritis
AG	- Acute gastritis
BL	- Bland
CDG	- Chronic diffuse gastritis
CG	- Chronic gastritis
CI	- Chronic inflammation
DC	- Decreased
DE	- Duodenal erosion
DHG	- Diffuse haemorrhagic gastritis
DU	- Duodenal ulcer
EG	- Erosive gastritis
GE	- Gastric erosions
GP	- Gastric polyp
GU	- Gastric ulcer
GU	- Gastric ulcer
H.Pylori	- Helicobacter pylori
IN	- Increased
LC	- Lax cardia
MB	- Moderately built
NG	- Non specific gastritis
N	- No

NR	- Normal
PB	- Poorly built
PR	- Present
RT	- Routine
SG	- Superficial gastritis
SP	- Spicy
UN	- Under nourished
WB	- Well built
WN	- Well nourished
Y	- Yes

ANNEXURE IV - MASTER CHART

Serial Number	In Patient / Out Patient Number	Sex	Ae (Years)	Complaints				Personal History					Clinical exam			Investiatio		
				Pain abdomen	Vomiting	Haemetemesis	Melena	Appetite	Diet	Tobacco chewi	Smoki	Alcoholism	Built	Nourishment	Pallor	Rapid urease test	Endoscopic impression	Histopatholoical report
1	1648123	M	60	Y	N	N	N	NR	RT	N	N	N	PB	UN	PR	Y	CG	CDG
2	997046	M	65	Y	Y	N	N	NR	SP	N	Y	Y	PB	UN	AB	Y	CDG	CDG
3	998428	M	32	Y	Y	N	N	IN	SP	Y	N	Y	MB	WN	PR	Y	CG	EG
4	1005130	M	42	Y	N	N	N	NR	RT	N	N	Y	MB	UN	AB	N	EG	CDG
5	407061	M	68	Y	N	N	N	DC	RT	N	Y	N	MB	WN	AB	N	GU,EG,CDG	CDG
6	971075	F	65	Y	Y	N	N	DC	BL	N	N	N	WB	WN	AB	Y	CDG	CG
7	421884	M	52	Y	Y	N	N	IN	BL	N	Y	Y	MB	WN	AB	N	AE	CDG
8	971562	M	27	Y	Y	N	N	NR	SP	N	N	N	WB	WN	AB	Y	EG,DU	CDG
9	407042	F	20	Y	Y	Y	N	NR	RT	N	N	N	MB	WN	PR	N	DHG	CG
10	1580228	F	52	Y	Y	N	N	NR	RT	N	N	N	MB	UN	AB	Y	DU	CG
11	1587090	M	53	N	Y	Y	N	NR	SP	Y	Y	N	WB	WN	AB	N	GU,EG	CG
12	1649585	M	60	Y	Y	Y	N	DC	RT	Y	N	N	MB	UN	AB	Y	GE	CDG
13	1415487	F	52	Y	Y	Y	N	DC	RT	N	N	N	MB	WN	AB	Y	EG	CG
14	430211	M	30	N	Y	N	N	NR	RT	Y	N	N	MB	WN	PR	N	AEG	CG
15	431736	F	20	Y	Y	N	N	NR	RT	N	N	N	MB	UN	PR	N	NR	CG
16	431063	F	52	Y	N	N	N	NR	RT	N	N	N	MB	UN	AB	N	AG	CG
17	1770140	M	26	N	Y	N	N	NR	RT	N	Y	Y	WB	WN	AB	Y	EG	CG
18	431745	F	50	Y	N	N	N	DC	RT	N	N	N	WB	WN	AB	Y	CG	CG
19	430433	M	29	Y	Y	N	N	NR	SP	N	N	Y	WB	WN	AB	Y	CG	CG
20	1649619	F	30	Y	Y	N	N	NR	RT	N	Y	N	WB	WN	AB	Y	GU,EG	CG
21	1790958	M	65	Y	Y	N	N	DC	RT	N	N	Y	MB	WN	AB	Y	GE	CG
22	422748	M	65	Y	N	N	N	IN	SP	N	N	N	WB	WN	AB	Y	NR	CG
23	1683119	M	38	Y	Y	N	N	NR	RT	Y	N	N	MB	WN	AB	Y	DU	CG
24	403838	M	60	Y	N	N	N	DC	RT	N	Y	N	MB	WN	AB	Y	GE	CG
25	401916	M	36	Y	Y	Y	Y	DC	RT	N	Y	Y	MB	WN	PR	Y	DHG	DHG
26	1488684	F	55	Y	N	N	N	IN	SP	Y	N	N	PB	UN	PR	N	NR	CG
27	1771577	M	64	Y	Y	N	N	DC	RT	N	Y	N	PB	UN	AB	Y	DE,EG	CG
28	1752137	M	35	Y	N	N	N	DC	SP	N	Y	Y	MB	WN	AB	Y	GE	CG
29	1789196	F	70	Y	N	N	N	NR	RT	N	N	N	MB	WN	AB	N	CG	CG
30	1778994	M	45	Y	Y	N	N	NR	RT	N	N	Y	MB	WN	AB	N	GP	CI
31	1656218	M	49	Y	N	N	N	DC	SP	N	N	Y	MB	WN	AB	Y	CG	CG
32	1766787	M	58	Y	Y	N	N	NR	RT	Y	N	N	WB	WN	PR	Y	GE	CG
33	1135027	M	57	Y	N	N	N	NR	BL	N	N	Y	WB	WN	PR	Y	NR	CG
34	590928	F	51	Y	N	N	N	NR	RT	N	N	N	MB	WN	AB	N	NR	NR
35	433009	M	61	Y	N	N	N	DC	BL	N	N	Y	WB	WN	AB	N	NR	NR

ANNEXURE IV - MASTER CHART

				Complaints				Personal History					Clinical exam			Investiatio		
Serial Number	In Patient / Out Patient Number	Sex	Ae (Years)	Pain abdomen	Vomiting	Haemetemesis	Melena	Appetite	Diet	Tobacco chewi	Smoki	Alcoholism	Built	Nourishment	Pallor	Rapid urease test	Endoscopic impression	Histopatholoical report
36	1121	F	42	Y	Y	N	N	DC	RT	N	N	N	MB	WN	AB	Y	NR	CG
37	1693031	M	64	Y	N	N	N	DC	SP	N	Y	Y	WB	WN	AB	Y	NR	NR
38	449299	M	52	Y	N	N	N	DC	RT	N	N	N	MB	WN	AB	Y	NR	CG
39	739602	M	55	Y	N	Y	N	DC	RT	Y	Y	Y	PB	UN	PR	N	GE	AEG
40	423352	M	47	Y	N	N	N	NR	RT	N	Y	Y	MB	WN	AB	N	AG	AG
41	406039	M	70	Y	N	N	N	NR	RT	N	N	N	WB	WN	AB	N	GE	CG
42	1718890	F	70	Y	Y	N	N	NR	SP	Y	N	N	MB	UN	PR	Y	GU	CG
43	883387	M	50	Y	Y	N	N	IN	BL	Y	Y	Y	WB	WN	AB	N	CG	CG
44	1064274	F	70	Y	N	N	N	DC	RT	N	N	N	WB	WN	AB	N	CG,GU	CG
45	447793	M	62	N	Y	Y	N	DC	BL	N	N	N	MB	WN	AB	N	CG	AG
46	432351	M	65	Y	N	N	N	NR	RT	N	N	Y	MB	WN	AB	N	GE	CG
47	1138793	M	70	Y	N	N	N	NR	RT	N	N	N	WB	UN	AB	N	CG	NR
48	1424906	F	31	Y	Y	N	N	NR	RT	N	N	N	WB	WN	PR	N	EG	EG
49	449756	M	39	Y	N	N	N	NR	RT	N	Y	Y	WB	WN	AB	Y	NR	CG
50	1541869	M	40	Y	N	N	N	IN	BL	N	N	N	MB	WN	PR	Y	CG	CG
51	424229	M	70	Y	N	N	N	NR	RT	N	Y	N	MB	UN	PR	Y	EG	CG
52	1584685	M	33	Y	N	N	N	NR	RT	N	Y	Y	MB	UN	PR	Y	DU,EG	NR
53	400101	M	18	Y	Y	N	N	NR	RT	N	N	N	WB	WN	AB	Y	NR	CG
54	402990	F	65	Y	N	N	N	IN	RT	Y	N	N	WB	WN	AB	N	NR	NR
55	408903	M	32	Y	Y	N	N	DC	SP	N	Y	Y	WB	WN	AB	Y	NR	MG
56	404343	M	25	Y	N	N	N	NR	RT	N	Y	Y	MB	WN	PR	Y	NR	NR
57	1804943	M	36	Y	Y	N	N	NR	BL	N	N	N	MB	WN	PR	Y	EG	CG
58	1906287	M	60	Y	N	N	N	IN	SP	N	N	N	MB	WN	PR	Y	CG	CG
59	1120	M	18	Y	Y	N	N	DC	RT	N	N	N	WB	WN	AB	N	CG	CG
60	419432	M	40	Y	Y	N	N	DC	SP	N	N	Y	WB	WN	PR	N	NR	NG
61	412512	M	51	Y	N	N	N	DC	SP	Y	N	N	MB	WN	PR	N	NR	NG
62	441730	F	55	Y	Y	Y	N	DC	BL	N	N	N	MB	WN	AB	N	EG	CG
63	1064274	F	70	Y	N	N	N	IN	BL	N	N	N	MB	WN	PR	N	AEG	CG
64	1801921	M	66	Y	Y	N	N	NR	RT	N	N	Y	WB	WN	AB	N	AG	AG
65	1836297	M	52	Y	Y	N	N	NR	RT	N	N	N	PB	UN	PR	Y	NR	CG
66	443138	M	49	Y	N	N	N	NR	RT	Y	Y	Y	WB	WN	PR	N	EG	CG
67	1891246	M	59	Y	Y	N	N	NR	RT	N	N	Y	MB	WN	AB	N	CG	CDG
68	443754	M	27	Y	N	N	N	NR	RT	N	Y	Y	WB	WN	AB	Y	NR	CDG
69	445306	F	58	Y	N	N	N	NR	RT	N	N	N	WB	WN	AB	Y	NR	NR
70	1852126	M	50	Y	Y	N	N	IN	RT	N	Y	N	WB	WN	AB	Y	GU	CG

ANNEXURE IV - MASTER CHART

Serial Number	In Patient / Out Patient Number	Sex	Ae (Years)	Complaints				Personal History					Clinical exam			Investiatio		
				Pain abdomen	Vomiting	Haemetemesis	Melena	Appetite	Diet	Tobacco chewi	Smoki	Alcoholism	Built	Nourishment	Pallor	Rapid urease test	Endoscopic impression	Histopatholoical report
71	1885466	M	47	Y	Y	N	N	DC	SP	N	N	Y	PB	UN	PR	Y	AE	CDG
72	1902238	F	34	Y	Y	N	N	NR	BL	N	N	N	PB	UN	PR	Y	AG	AG
73	1899784	M	46	Y	Y	N	N	NR	RT	Y	Y	Y	WB	WN	AB	N	AG	CI
74	1897403	M	43	Y	N	N	N	IN	BL	N	N	N	WB	WN	PR	N	CG	CDG
75	1894268	M	57	Y	Y	N	Y	DC	RT	N	N	N	MB	UN	PR	N	EG	CDG
76	400893	M	67	Y	Y	Y	Y	DC	RT	N	Y	Y	PB	UN	PR	N	EG	NR
77	448756	M	24	Y	N	N	N	NR	BL	N	Y	N	WB	WN	AB	Y	CG	CDG
78	1884195	M	47	Y	N	N	N	DC	SP	N	Y	Y	WB	WN	AB	N	CG,GU	CDG
79	962289	M	53	Y	Y	N	N	DC	BL	N	Y	N	MB	UN	PR	Y	AEG	AG
80	1873233	M	60	Y	N	N	N	NR	RT	N	N	N	PB	UN	AB	Y	EG	CDG
81	15093	F	40	Y	N	N	N	NR	RT	N	N	N	MB	WN	AB	Y	NR	NR
82	1892635	F	60	Y	Y	N	N	DC	SP	Y	N	N	MB	UN	PR	Y	AG	AG
83	449954	F	60	Y	N	N	N	NR	RT	N	N	N	WB	WN	AB	N	NR	CG
84	1918971	M	36	Y	N	N	N	NR	SP	N	Y	Y	MB	WN	AB	Y	CDG	CG
85	452789	M	66	N	Y	N	N	NR	RT	N	N	Y	PB	UN	PR	Y	NR	NR
86	1900413	M	70	Y	N	N	N	IN	SP	N	Y	N	MB	WN	AB	N	CDG	CDG
87	453457	M	66	Y	N	N	N	IN	RT	N	N	Y	MB	WN	AB	Y	EG	CDG
88	1921746	M	28	Y	N	N	N	DC	SP	N	N	Y	PB	UN	AB	Y	GU	CDG
89	449941	M	53	Y	Y	N	N	NR	BL	N	Y	N	PB	UN	PR	Y	CDG	CG
90	1923507	M	46	Y	N	N	N	NR	RT	N	Y	Y	WB	WN	AB	Y	CG	CDG
91	1897403	M	44	Y	Y	N	N	NR	RT	Y	N	N	MB	WN	AB	N	CDG	CG
92	452724	M	38	Y	N	N	N	NR	SP	N	N	N	MB	WN	AB	N	CDG	CDG
93	443754	M	27	Y	Y	N	N	NR	SP	N	Y	Y	MB	WN	AB	N	EG	CDG
94	464772	F	29	Y	N	N	N	NR	RT	N	N	N	MB	WN	AB	Y	AG	CDG
95	2016225	M	18	Y	Y	N	N	NR	RT	N	N	N	MB	WN	AB	Y	EG	CDG
96	464454	M	49	Y	Y	N	N	DC	SP	Y	N	N	WB	WN	AB	N	EG	CI
97	464453	M	60	Y	N	Y	N	DC	BL	N	N	N	MB	WN	PR	Y	CDG	CDG
98	1994194	M	58	N	Y	N	N	DC	BL	N	Y	Y	WB	WN	AB	N	AE	CDG
99	461941	F	65	Y	Y	N	N	NR	SP	N	N	N	MB	WN	AB	Y	CDG	CG
100	1507305	M	47	Y	N	N	N	NR	SP	N	N	Y	WB	WN	AB	N	EG	CDG
101	1975322	M	29	Y	Y	Y	N	NR	RT	Y	Y	N	WB	WN	AB	N	DHG	CDG
102	1672113	F	70	Y	N	N	N	DC	BL	N	N	N	PB	UN	AB	Y	CDG	CG
103	1967064	M	64	Y	Y	N	N	DC	BL	N	N	N	MB	WN	AB	Y	CDG	CDG
104	458656	M	19	Y	Y	N	N	NR	SP	N	N	N	MB	WN	AB	Y	DHG	CG
105	424411	M	70	Y	N	N	N	DC	BL	N	N	N	MB	UN	AB	N	NR	NSG

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Serial Number	In Patient / Out Patient Number	Sex	Ae (Years)	Complaints				Personal History					Clinical exam			Investiatio		
				Pain abdomen	Vomiting	Haemetemesis	Melena	Appetite	Diet	Tobacco chewi	Smoki	Alcoholism	Built	Nourishment	Pallor	Rapid urease test	Endoscopic impression	Histopatholoical report
106	1962604	M	70	Y	N	N	N	DC	SP	N	N	Y	MB	UN	AB	Y	CDG	CG
107	455552	M	24	Y	Y	N	N	DC	RT	N	N	N	WB	WN	PR	Y	AG	CG
108	1953651	M	44	Y	N	N	N	NR	RT	N	N	Y	MB	UN	AB	N	EG	CDG
109	1927075	F	39	Y	Y	N	N	DC	SP	N	N	N	MB	WN	AB	N	CG	CDG
110	1926711	F	55	Y	N	N	N	DC	BL	Y	N	N	MB	WN	AB	Y	AG	CDG
111	440905	M	46	Y	N	N	N	DC	BL	N	Y	Y	MB	UN	AB	N	CDG	CG
112	1755272	F	55	Y	Y	N	N	NR	RT	N	N	N	MB	WN	AB	Y	CDG	CG
113	1830586	F	60	Y	N	N	N	NR	SP	N	N	N	MB	WN	AB	Y	CDG	CG
114	382161	F	60	Y	N	N	N	NR	BL	N	N	N	WB	WN	PR	N	EG	CG
115	1395462	F	58	Y	Y	N	N	DC	BL	N	N	N	PB	UN	AB	Y	DHG	CDG
116	1354230	M	62	Y	Y	N	N	NR	RT	N	Y	N	PB	UN	AB	Y	AEG	CG
117	1390406	M	70	Y	Y	N	N	DC	BL	N	N	N	WB	WN	AB	N	EG	CDG
118	1814110	M	60	Y	N	N	N	NR	RT	Y	N	N	WB	WN	AB	N	AG	CDG
119	1413286	M	65	Y	Y	N	N	NR	RT	N	N	N	WB	WN	AB	N	AG	CG
120	1426246	M	70	Y	N	N	N	DC	BL	N	N	N	PB	UN	AB	Y	EG	CG
121	387796	F	55	Y	Y	N	N	DC	RT	N	Y	Y	MB	WN	AB	N	EG	CI
122	1335901	M	22	Y	Y	N	N	DC	RT	N	N	N	MB	WN	AB	Y	GU,CG	CDG
123	1401540	F	62	Y	Y	N	N	NR	RT	N	N	N	WB	WN	PR	Y	CDG	CG
124	1402036	F	42	Y	N	N	N	DC	SP	Y	N	N	MB	WN	AB	N	NR	CDG
125	719529	F	62	Y	N	N	N	DC	SP	N	N	N	MB	UN	AB	Y	AE	CG
126	397276	M	68	Y	Y	N	N	NR	RT	N	N	Y	MB	UN	PR	N	NR	CDG
127	397879	M	70	Y	N	N	N	DC	BL	N	N	N	MB	UN	AB	N	CDG	CDG
128	1467082	M	61	Y	Y	N	N	DC	BL	Y	N	N	MB	WN	AB	Y	AE,GU	CDG
129	1344597	M	52	Y	N	N	N	NR	BL	Y	Y	Y	MB	WN	PR	Y	SG	CDG
130	1390964	M	67	Y	N	N	N	DC	RT	N	N	Y	WB	WN	PR	N	GU,CDG	CDG,GU
131	1435900	M	35	Y	N	Y	N	DC	SP	Y	Y	N	PB	UN	AB	N	NR	CDG
132	392268	M	18	Y	Y	N	N	DC	BL	N	N	N	WB	WN	AB	N	DU,EG	CG
133	1442438	M	56	Y	N	N	N	NR	RT	N	N	Y	MB	WN	AB	N	GU,CDG	CDG
134	581773	F	60	Y	Y	Y	N	DC	BL	N	N	N	MB	UN	PR	N	NR	CDG
135	1456971	M	48	N	Y	N	N	NR	RT	Y	N	N	WB	WN	AB	Y	NR	CG
136	453802	M	28	Y	N	N	N	NR	SP	N	Y	Y	WB	WN	AB	Y	EG	CG
137	453323	F	54	Y	N	N	N	NR	RT	N	N	N	MB	WN	AB	N	GP	GP,CG
138	1577159	M	55	Y	N	N	N	DC	BL	N	N	Y	PB	UN	PR	Y	DU,AG	AG
139	1928797	M	67	Y	N	N	N	NR	RT	N	N	N	MB	WN	AB	N	NR	CDG
140	868079	F	63	Y	Y	N	N	DC	RT	N	N	N	WB	WN	AB	Y	GP,EG	CG

ANNEXURE IV - MASTER CHART

				Complaints				Personal History					Clinical exam			Investiatio		
Serial Number	In Patient / Out Patient Number	Sex	Ae (Years)	Pain abdomen	Vomiting	Haemetemesis	Melena	Appetite	Diet	Tobacco chewi	Smoki	Alcoholism	Built	Nourishment	Pallor	Rapid urease test	Endoscopic impression	Histopatholoical report
141	454481	M	59	Y	N	N	N	NR	RT	N	N	Y	PB	UN	AB	N	NR	CDG
142	1584685	M	33	Y	Y	N	N	DC	RT	Y	N	Y	MB	WN	AB	Y	EG	CG
143	419432	M	40	N	N	N	N	DC	RT	Y	N	Y	PB	UN	PR	N	LC	CI
144	421173	M	28	Y	Y	N	N	NR	SP	N	N	N	WB	WN	AB	N	AG	AG
145	11062900	M	44	Y	Y	N	N	IN	RT	N	N	Y	MB	WN	PR	Y	NR	CG
146	1771717	M	64	N	Y	N	N	NR	RT	N	N	N	WB	WN	PR	Y	CDG	CG
147	110704001	M	60	Y	N	N	N	DC	RT	N	N	N	MB	WN	AB	N	NR	CG
148	421184	M	27	Y	Y	N	N	DC	SP	N	Y	N	WB	WN	AB	Y	AG	AG
149	420113	M	70	Y	Y	N	N	DC	RT	N	Y	N	MB	WN	AB	Y	NR	CG
150	431195	M	28	Y	Y	N	N	DC	SP	N	Y	N	WB	WN	AB	Y	AG	AG
151	416760	M	59	Y	Y	N	N	DC	RT	N	N	N	WB	WN	AB	Y	AG	CDG
152	425978	F	70	Y	N	N	N	NR	RT	N	N	N	WB	WN	AB	N	NR	NR
153	110606001	M	40	Y	N	N	N	DC	SP	Y	Y	Y	MB	WN	PR	Y	NR	CDG
154	1122	F	62	Y	Y	N	N	NR	BL	N	N	N	WB	WN	AB	Y	AE	AG
155	1120	M	18	Y	N	N	N	DC	SP	N	N	N	WB	WN	AB	Y	AG	AG
156	411908	M	60	Y	Y	N	N	DC	SP	N	N	Y	MB	UN	AB	Y	AG	CDG
157	421367	M	70	Y	Y	N	N	DC	SP	N	N	N	WB	WN	AB	N	AG	CDG
158	400562	M	60	Y	N	N	N	NR	SP	N	N	Y	MB	UN	PR	N	NR	NR
159	408915	M	49	Y	Y	N	N	DC	RT	Y	Y	Y	MB	UN	PR	Y	NR	NR
160	409138	F	43	Y	N	N	N	DC	RT	N	N	N	MB	WN	AB	N	NR	NR
161	410474	M	48	Y	N	N	N	NR	RT	Y	N	N	MB	WN	AB	N	NR	NR
162	419693	M	52	Y	N	N	N	NR	BL	N	N	N	MB	WN	AB	Y	NR	NR
163	420377	M	50	Y	Y	N	N	DC	SP	N	Y	Y	MB	WN	AB	N	NR	NR
164	421173	M	28	Y	Y	N	N	DC	RT	N	N	N	MB	WN	AB	Y	NR	NR
165	421734	F	20	Y	Y	N	N	DC	SP	N	N	N	PB	UN	PR	N	NR	NR
166	426139	M	43	Y	N	N	N	NR	BL	Y	N	N	MB	WN	AB	Y	NR	NR
167	432303	M	63	Y	Y	N	N	DC	BL	Y	N	N	MB	WN	AB	Y	NR	NR
168	437041	M	52	Y	N	N	N	NR	RT	N	N	Y	PB	UN	AB	N	NR	NR
169	429505	M	40	Y	N	N	N	NR	SP	Y	N	N	WB	WN	AB	N	AG	AG
170	427457	M	40	Y	Y	N	N	DC	SP	N	Y	Y	MB	UN	PR	Y	AG	AG
171	403003	M	30	Y	N	N	N	DC	RT	Y	Y	Y	MB	WN	AB	Y	AG	NR
172	403676	F	35	Y	N	N	N	NR	RT	N	N	N	MB	WN	PR	N	NR	NR
173	421184	M	27	Y	Y	N	N	DC	RT	Y	N	Y	MB	WN	AB	N	AG	AG
174	408182	F	70	Y	Y	N	N	DC	RT	Y	N	N	PB	UN	PR	Y	AG	NR
175	403675	F	48	Y	N	N	N	DC	SP	N	N	N	MB	UN	PR	N	CDG	CDG

ANNEXURE IV - MASTER CHART

Histopatholoical report for H. Pylori
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