Prevalence of *Helicobacter Pylori* Serologically in Patients Who Presented With Dyspeptic Symptoms to Orotta Medical Surgical National Referral Hospital

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Abstract

Background: Optimum management of dyspepsia in primary care is a debatable subject. Testing and treatment for *Helicobacter pylori* has become widely accepted as the approach of choice for patients with chronic dyspepsia but no alarming features. We evaluated prevalence of *H. pylori* among outpatients with dyspepsia and serologic investigations for it in tertiary hospital Orotta Medical Surgical National Referral Hospital (OMSNRH) retrospectively.

Methods: A retrospectively collected data of *H. pylori* status among dyspeptic patients from Out Patient Department (OPD) and laboratory of OMSNRH, who had undergone serologic test for the infection, of the year 2012 was reviewed for the period from February 2013 to May 2013.

Results: The prevalence of dyspepsia visited OMSNRH was 6.08%, with female predominance, from a total visit of 30,035. Of all 1844 dyspeptic patients from the OPD, 20.93% were positive, 48.05% were negative and 31.02% were untested for *H. pylori.* From a total of 4136 of the laboratory results, the prevalence of *H. pylori* was 31%. In adults (>14 years), it was 34% and pediatric (<15 years), prevalence was 12.5%. Male sex preference was observed among the adults in this study (37.7% vs. 31.7%, P=0.034).

Conclusion: The prevalence of dyspepsia was 6.08% and the prevalence of *H. pylori* serologically in this study was 34% in adults and 12.5% in pediatric age group.

1. Background

Helicobacter pylori is one of the human pathogens with highest prevalence around the world; yet, its principal mode of transmission remains largely unknown.[1] It is one of the most common bacterial infections in humans. Two thirds of the world's population is infected with *H. pylori*, and primary infection occurs before the age of five years with permanent residence of the bacteria in the stomach afterwards.[2]The role of *H. pylori* in gastric disease and cancer has not been established until the end of the 20th century. Since then, its epidemiology has been extensively studied, and an accruing body of literature suggests that not all humans are equally at risk of infection by this gut pathogen. The epidemiology of *H. pylori* infection is characterized by marked differences between developing and developed countries, notably among children. In addition, congruent lines of evidence point out to socioeconomic factors and living standards as main determinants of the age-dependent acquisition rate of *H. pylori*, and consequently its prevalence. These data are alarming in the light of the changing global climate and birth rate, which are expected to change the demography of our planet, putting more children at risk of *H. pylori* and its complications for years to come.[1]

1.1 Literature Review

1.1.1 Introduction

The bacteria *H. pylori* are relatively small, gram-negative spiral-shaped or curved rods with 4 to 6 unipolar sheathed flagella which are of importance for bacterial motility and colonization of the stomach[2]. It was established in 1982 by Robin Warren and Barry Marshall as the causative agent of gastritis and peptic ulcer[3, 4], a discovery that revolutionized gastroenterology. Before Warren and Marshall, the human stomach was believed to be a sterile area. There are over 40 different species of *H. pylori* which differ in size and the number and location of flagella. But all the species have the same shape, i.e., curved rod, all possess flagella, all have GC content of their genome and a strong urease activity that has enabled diagnostic shift from invasive to non-invasive ones.[2] After the confirmed hypothesis that gastritis and peptic ulcer disease is caused by *H. pylori* in 1983, the diagnosis and treatment of peptic ulcer disease has changed dramatically. As more knowledge is added to what we already know about *H. pylori*, different diagnostic methods have come to view[2]. *H. pylori* produce the enzymes urease, catalase, and oxidase which can be observed by biochemical testing of the bacterial isolates.[2] It colonizes the gastric mucosa with the help of different structures in its cell wall. Why some individuals never develop the disease even though they harbor the organism is explained by the fact that bacterial virulence factors (like cag-PAI), genetic background of the individual and the environment all contribute to successful manifestation of the disease.[2] Today, *H. pylori* is recognized as the most common cause of gastritis, which in turn leads to the development of more gastrointestinal complications such as peptic and duodenal ulcers. Additionally, the organism is classified as a class 1 carcinogen because of its causal relationship to gastric adenocarcinoma, one of the world's deadliest cancers.[5,6]

1.1.2 Prevalence of *H. pylori* between developed and developing countries

The prevalence of infection seems to mostly depend on the rate of acquisition, but also on the rate of loss of infection[7] and the length of the persistence period between acquisition and loss.[8] Based on these factors, *H. pylori* prevalence differs from one country to another and may differ between different ethnic, social, or age groups within the same country.[1,7,9] Globally, the prevalence of *H. pylori* infection in developing countries is markedly higher than that in developed countries. [10–14] Sero-prevalence is higher in developing countries, with 80% of the adults *H. pylori*-positive in contrast to 40–50% prevalence rates in developed countries.[15] A plethora of studies reported and emphasized these differences within and between countries. The geographic differences in *H. pylori* prevalence have been attributed to the differential rate of acquisition of the bacterium during the first years of life.[7,16–17] In southern China, for example, the prevalence of *H. pylori* infection was shown to be significantly higher among Chinese subjects than that among Australians, a difference that was associated with the rate of acquisition of *H. pylori* under the age of ten years.[17,18] Acquisition of *H. pylori* is decreasing in developed countries at a faster rate than in developing countries, likely because of the faster improvement in hygiene practices in the developed world.[16] Moreover, infection during childhood in developed countries is not frequent.[19–22] In the United States, for example, the incidence of infection among children younger than five years is less than 5%, and only about 10% of the population is infected by adolescence.[19,20] By contrast, the

incidence of *H. pylori* infection in the developing world is higher and occurs at younger age.[21,23] By five years of age, about 50% of children in developing countries are already infected,[10,23] and the infection rates in adults can reach 90% or higher, with consequent acquisition of peptic ulcer disease in 20% and gastric cancer in less than 1%.[15]

Pounder and Ng classified the world into two groups according to the incidence of *H. pylori* infection.[8] Group One consisted of countries where the majority of children become infected with *H. pylori* during childhood, while chronic infection continues during adult life. These are mostly developing countries, e.g., Algeria, Nepal, South Africa, Saudi Arabia, Thailand, and Vietnam. In Group Two, mostly comprising developed countries, only a minority of children becomes infected during childhood, but the prevalence of infection rises with age during adulthood. Examples of Group Two countries are England, Finland, France, Japan, and the United States of America.[8] However, Pounder and Ng concluded their synthesis with an interesting question. Do the age-dependent prevalence data reflect that people in Group Two have more incidence of infection at older ages, or do the data rather reflect that the incidence of infection is declining in newer generations, which implies that the infected adults had been actually infected in their childhood? [8] This question was effectively answered later on, as longitudinal studies confirmed the birth cohort effect in the United States [7] and Russia,[24] for example.

Other studies indicate that in developed countries, the incidence of *H. pylori* is 40-50%, as compared to the almost 100%infection in developing countries. Better hygienic standards and widespread antibiotic use are believed to be the causes for this wide epidemiological difference.[25] Evidence points out that transmission are mainly fecal-oral, oral-oral, and vomitus-oral, with questionable transmission through contaminated water supplies. Environmental or animal reservoirs were investigated as sources of *H. pylori* infection. Food, animals, and water sources have been suggested as reservoirs outside the human gastrointestinal tract, and *H. pylori* or its DNA was detected in each of these sources. However, there is no definitive evidence that they are natural or primary vehicles of transmission.[1]

1.1.4 Risk factors

Studies have been shown that the prevalence of *H. pylori* infection is affected by factors like age, gender, ethnic and genetic predisposition, socioeconomic status, interfamilial relations and crowding index.[1]The effect of age on the prevalence of

H. pylori is one of the best-documented and least disputed aspects of *H. pylori* epidemiology. A positive correlation between age and prevalence has been reported in both developed and developing countries.[9,26–28] Consistently, the prevalence of infection was found to be higher in adults than that in children, and this pattern has been interpreted to partly reflect a birth cohort phenomenon caused by a higher incidence in the past due to poor living conditions and sanitation.[24,29,30]

A comprehensive longitudinal study undertaken by Fujisawa et al. evaluated changes in the seroepidemiological pattern of *H. pylori* in a group of Japanese people over a 20-year period.[30] Sera were

collected from 1015 subjects, and the overall prevalence of *H. pylori*-specific antibodies was 72.7% in 1974, 54.6% in 1984, and 39.3% in 1994. The prevalence of *H. pylori* was found to be positively correlated with age, suggesting that there was a clear cohort-shift in the seroepidemiological pattern of *H. pylori* during the 20 years studied.[30]

Marked differences in *H. pylori* seroprevalence have been observed and reported among various ethnic and racial groups.[12,31,32] For example, in Malaysia, the increased risk of *H. pylori* infection in Chinese and Indians was suggested as an inherent ethnic genetic predisposition.[31] In New Zealand, ethnicity was suggested as a risk factor among different groups in the populations. *H. pylori* infection was most prevalent in Pacific Islanders, intermediate in Maori, and least prevalent in Europeans. Even after the adjustment of confounding factors, such as age and socioeconomic status, ethnicity remained a significant covariate.[32]

By contrast, in a study conducted in USA, the prevalence of *H. pylori* infection was almost identical between Hispanic and African Americans, but significantly higher than that among Caucasians. However, ethnicity was ruled out as a major factor and the observed variance was attributed to socioeconomic conditions.[9] Finally, a study of monozygotic and dizygotic twins suggested also that genetic factors might have some influence on the incidence of *H. pylori* infection.[33]

An excess of *H. pylori* prevalence in one gender versus the other has been reported; [34–36] for instance, Woodward and colleagues observed a higher prevalence of *H. pylori* in men than in women.[36] Others found no gender-related difference in the prevalence of *H. pylori* infection.[31,32,37,38] A more recent, more comprehensive meta-analysis of large, population-based studies concluded a male predominance of *H. pylori*-related diseases in adults but not in children.[39]

Many studies reported an influence of interfamilial relation on the spreading of *H. pylori* infection and highlighted adult-child transmission.[40–42] Konno and coworkers suggested mother-to-child transmission as the single most probable cause of the interfamilial spreading of *H. pylori* infection after a five-year follow-up study. Among 44 children enrolled in that study, five children acquired *H. pylori* infection, and their bacterial isolates exhibited DNA fingerprinting patterns identical to those of their mothers.[42] Family size has also been shown to positively affect *H. pylori* infection incidence; the relative risk of infection has been shown to increase according to the number of children per household.[27,43]

Additionally, spouse-to-spouse transmission was suggested.[44,45] Georgopoulos et al. found a significant number of couples infected with indistinguishable strains of *H. pylori*.[44]

Finally, infected children were also proposed as a source of infection for parents or siblings.[27,46] However, a case-control study performed in Bangladesh found no difference in infection rates between parents of infected and non-infected infants, and concluded that in communities with high prevalence of *H. pylori* infection, interfamilial transmission might be masked by other environmental factors.[47]

Socioeconomic status was reported as one of the most important factors affecting the spreading of *H. pylori* infection.[9,12,48] In particular, the high age-specific prevalence of *H. pylori* infection in developing countries has been attributed to low socioeconomic level.[11] By contrast, the lower prevalence of *H. pylori* infection in developed countries may be a result of higher socioeconomic status. The overall prevalence of *H. pylori* among Swiss adolescents was found to be 9.7%. While this prevalence is among the lowest in Europe, further analysis indicated that subjects from foreign countries had higher rates of infection (30%) than natives (7.3%). This significant difference was largely attributed to higher living standards among natives.[49]

In USA, Malaty et al. classified children into five social classes. The prevalence of the infection was 82% in the lowest class, 52% in the two middle classes, and 11% in the two high classes, demonstrating an inverse correlation between *H. pylori* prevalence and socioeconomic status.[40]

Obviously, socioeconomic status is not restricted to income and social class but takes in consideration other factors, including living standards, sanitation, urbanization, and educational level.[18] Combined, these factors are likely to increase the risk for infectious diseases in general.

Educational level, in particular, has been used as a marker of socioeconomic status and has been considered as one of the important determinants of *H. pylori* prevalence in both developed[50] and developing countries.[28] Rosenstock et al. found that the short duration of schooling beside low socioeconomic status increases the likelihood of *H. pylori* infection in Denmark.[50]

Household crowding, sharing a bed, and increasing household contact have been identified as risk factors of *H. pylori* infection.[17,28,51,52] In a large community-based study, Torres and colleagues stated that density of living conditions is a prime determinant in the acquisition of *H. pylori*.[28] In childhood, crowded living conditions affect current *H. pylori* status, and the number of children in the present household increases the risk of infection for the adult family members.[27]

Mohammed M. Khalifa et al. in the article; *Helicobacter pylori: a poor man's gut pathogen*, conclude that, as with most infectious and non-infectious diseases, no one factor can be singled out as the major determinant of *H. pylori* incidence and prevalence. However, there is credible evidence that poverty-associated factors are major players.[1]

1.1.5 Dyspepsia, its prevalence and association with *H. pylori*

Dyspepsia is the main symptom for a multitude of esophageal, gastroduodenal, pancreatic, and hepatobiliary disorders. An international committee of clinical investigators (Rome III Committee) defined dyspepsia as one or more of the following symptoms: Postprandial fullness (termed postprandial distress syndrome), Early satiation (meaning inability to finish a normal sized meal or postprandial fullness) and Epigastric pain or burning (termed epigastric pain syndrome).[53] Its prevalence in western

countries is estimated to be 25% which accounts 2-5% of primary care consultation.[54] Up to one-fourth of cases of dyspepsia are due to peptic ulcer disease.[55] There is strong relationship between *H. pylori* infection and peptic ulcer disease which provides the rationale for *H. pylori* eradication in patients with known ulcers.[56] Several studies have compared strategies involving *H. pylori* testing with endoscopy for the initial investigation in patients with dyspepsia.[57] These studies have provided the parameters used in a number of decision analyses that have assessed the cost-effectiveness of dyspepsia evaluation, particularly the roles of endoscopy versus noninvasive strategies including *H. pylori* testing.[58,59]

In northern Nigeria at a tertiary hospital, a prospective study was done among dyspeptic patients in whom 77.1% are found to be positive for *H. pylori* with higher prevalence to males (51.9%). In the study endoscopic finding for majority of the patients was gastritis (42%) and 8.5% of the patients was diagnosed as having gastric and duodenal ulcers.[60]

Another study done in El-jamahiria Hospital to patients with dyspeptic symptoms attending endoscopic unit for different reasons in Bengazi, Libia, showed a detection 108 (82%) of 132 patients (86%) by rapid urease test, 77% by direct smear stain, 95% by histology) for *H. pylori*. The endoscopic findings revealed that 77 (77%) of 100 patients with non-ulcer dyspepsia, 26 (96%) of 27 with duodenal ulcer, 4 (100%) of 4 with gastric ulcer and in one patient with gastric cancer were *Helicobacter pylori* positive. The enzyme-linked immunosorbent assay test showed 94% sensitivity and 88% specificity.[61]

1.1.6 Diagnosis of *H. pylori*

Many studies have been performed to evaluate the best diagnostic test to set as a gold standard so far. Most of the diagnostic methods to detect *H. pylori* have high sensitivity and specificity. The investigation methods can be classified as invasive and noninvasive ones. The urease activity of *H. pylori* was exploited to be used in many diagnostic kits, like the rapid urease tests, or CLO tests. Other diagnostic studies include the urea breath test, antibody or antigen detection in serum, blood, saliva or stool samples, ELISA based serology tests, immunoblot based serology tests, bacterial culture and histology as well as PCR techniques.[2]

Noninvasive methods are indirect evidence of the presence of *H. pylori* and should be carefully validated with endoscopy-based techniques. Because of its high acid content, the gastric mucous layer and epithelium has low levels of colonization by other bacteria. In spite of this, *H. pylori* succeeds in colonizing the extracellular gastric mucosa with intracellular bacteria considered rare.[25]

In a study that compared locally made urease test with histology for the diagnosis of *H. pylori*, it was emphasized that, although a variety of techniques are available for the diagnosis of *H. pylori* infection, an ideal test needs to be simple, rapid, inexpensive, accurate and readily available for clinical application.[62]

Initial diagnostic tests usually begin with urease tests which are usually inexpensive when compared to histology, but to reach a cumulative diagnosis, several of these tests are required, which increases the expenses. And despite the presence of several invasive and non-invasive tests, histology remains to be

the gold standard,[62] and results of many studies are compared with histology taken as standard. In most studies, if the initial tests (most usually, urease tests) were negative, histological examinations were carried out to give the final diagnosis.

But the draw backs of histology are many. Some of which are, the expense and the time it takes for the results to be given to the patient. It is highly desirable for a test result to be given to the patient quickly so that the treatment can be started immediately. This is highly applicable for patients with peptic ulcer hemorrhage.[63]

Another disadvantage is as follows. Owing to its particular morphology, *H. pylori* is identified by routine haematoxylin and eosin stain. In this stain, the bacteria appear rose-colored. Additional stains, like Giemsa,Warthin-Starry stains can be used to clarify doubtful results. So, histological identification is time consuming and calls for an experienced pathologist. Other disadvantages include limited sensitivity when few organisms are present due to antibiotic treatment, confusion with other spiral shaped bacteria, like the *Campylobacters* from the oral cavity and *Coccoidal* forms of the bacteria which are difficult to identify histologically. But if chronic active gastritis is found during histology, then, *H. pylori* bacteria should be carefully sought as the two complement each other.[63]

In a study that evaluated the diagnostic performance of biopsy based methods for determination of *H. pylori* infection without a reference standard, it was found out that the competence of the pathologist and the time spent to examine the slides (inter- observer variability) and the variability of staining techniques were among the problems encountered during histology.[64] Giemsa stain was used in the study as it was believed to be the one most frequently used during clinical practice because of its easy handling and good diagnostic performance. Histological examination was believed to have an advantage over other tests because it provides information about gastric mucosal pathology.

The results of the afro mentioned article showed no significant difference whether one relies on culture, histology or CLO tests. The article stressed that neither of the tests is reliable if used alone to diagnose patients. If however used independently, these tests would, according to the study, show similar results, with a disagreement of only 14% among the studies in the test results. So, if one wishes to test the success of *H. pylori* treatment, one ought to have a combined reference standard, i.e., culture, histology and CLO test. The study reports a recurrence rate of 0% to assess the long term cure rate in comparison with some studies which reported recurrence rates as high as 5% because these studies based their conclusion on only one variable, be it urease test or even, biopsy tests. In another study that analyzed the cost effectiveness of biopsies of *H. pylori* detection in patients with functional dyspepsia, it was found out that patients subjected to histopathological studies after negative rapid urease tests had little additional benefit compared to the enormous amount of cure money they had to spend.[65]This is particularly important because functional dyspepsia, defined as dyspepsia without classical pathological finding occurs in about 60% of dyspeptic cases. In contrast, when gastric biopsy smear cytology was compared to culture, rapid urease test and immunohistochemistry, gastric biopsy smear cytology was found to be sufficient for detection of *H. pylori*, because of its high sensitivity, specificity and accurate

methods of diagnosis when compared to other tests.[66] Another study concluded after five year pediatric experience that gastric biopsies obtained during upper endoscopy showed a 100% sensitivity and specificity for Giemsa staining, compared to 88% specificity for the rapid urease test (CLO test) when these two were compared to evaluate *H. pylori* gastritis.[67]

Evidence also gave results that bleeding peptic ulcers can't be diagnosed reliably by rapid urease tests alone, as the sensitivity of the rapid urease test is decreased when there is blood in the gastric lumen. The study concluded that histology to be performed instead for the safe diagnosis of *H. pylori*.[68] This study was complemented by another one which investigated the best means of diagnosing *Helicobacter pylori* infection in acute upper gastrointestinal hemorrhage. Histology was found to be the best diagnostic test.[69]

Moreover, when invasive and non-invasive diagnostic tests were compared in a study attempting to find out the accurate diagnosis of *H. pylori* infection in children, histology, in combination with two other tests, i.e., biopsy urease test and Carbon 13 urea breath test was used as a gold standard and it had a diagnostic accuracy of 98.1%, Carbon 13 urea breath test the only test superior to it, scoring a diagnostic accuracy of 100% in pediatric population.[70] However, when the utility of diagnostic tests to determine the prevalence of *H. pylori* in children with recurrent abdominal pain was studies in North eastern Mexico, it was discovered that, "noninvasive methods had acceptable values in sensitivity and specificity in comparison with invasive tests".[71] Serology was found out to be the best study kit for these specific age groups. Another prospective cohort study evaluated invasive and non-invasive methods for the diagnosis of symptomatic children and adolescents. The diagnostic methods were endoscopy with gastric biopsies for 3 invasive tests, i.e., rapid urease test, histology and culture, and 2 non-invasive methods, i.e., ELISA serology and 13 carbon urea breath test-isotope ratio mass spectrometry.[72] The greatest sensitivity, considering *Helicobacter pylori*-positive cases, for any combination of 3 or more tests, was achieved by the rapid urease test (S=100%), followed by histology, serology and 13carbon-urea breath test (S=93.1%) and lastly by culture (S=79.3%). The highest specificity was obtained by histology (100%) and culture (100%), followed by the rapid urease test (84.2%), serology (78.9%) and 13carbon-urea breath test (78.9%).[72] This study concluded that if invasive methods were to be chosen for the evaluation of *Helicobacter pylori* infection in children, rapid urease test and histology were the best choice, but that serology was still preferable if the results of the two invasive tests show different results.

At last, in a study that examined noninvasive tests as substitutes for histology in the diagnosis of *H. pylori* infection when rapid urease tests gave negative results, histological examination of gastric biopsies was recognized as the, "Gold standard back-up", although non- invasive tests may offer the same results with less cost.[70] Whole blood or serum antibody testing could substitute histology with similar accuracy unless the patient has history of antibiotic treatment for *Helicobacter pylori* in which case histology should be taken instead. [73]

It is well known that invasive tests, like histological biopsies are time consuming, expensive and labor intensive. It is also known that they can't be applied to routine screening. But despite all these short comings, they are recognized to be the gold standard in the diagnosis of *Helicobacter pylori* infection.[74]

And though 5-10% of patients are misdiagnosed regardless of any biopsy based method used, [75], culture, the remaining option is difficult and time consuming and is only indicated in specific conditions, e.g., in antibiotic resistance testing. Thus, it is recommended that though non-invasive methods may save time and cost as well as being more applicable to certain age groups, e.g., children, invasive methods, with histology as one of the leading tests, offer more precise evaluation of the status of the mucosa in the quest for definitive diagnosis of *Helicobacter pylori* infection. In a study that aimed to assess the diagnostic value of different non-invasive tests to compare them with endoscopy based diagnostic methods, it was found out that after treatment, in presence of complaints, it is important to obtain samples for the investigation of gastric mucosa specimens to enhance the value of endoscopic examination.[76] This is so because patients with gastric mucosal erosion, glandular atrophy and intestinal metaplasia may be *Helicobacter pylori* negative with non-invasive tests, but may still have dyspeptic symptoms that can't be ignored. [76] The article concluded by declaring that patients with dyspepsia should not only be followed with non-invasive tests but also with classical histological tests as they have proven to have high concordance rate with other tests.

1.1.7 Regional prevalence of *H. pylori*

In Africa different studies have been done concerning the prevalence of *H. pylori.* The main diagnostic method used to find the incidence was mainly the non-invasive ones like serology and also urea breath test.[77]

For instance; among Algerian children, 43% were seropositive, and showed increase in seroprevalence with age peaked to 92% between age groups 40 and 49. At the same time in Ivory coast, the seroprevalence of *H. pylori* was around 54%, rising gradually to a plateau of 70-80% through adulthood.[77,78]

Another study done in Kenya 93% of 14 asymptomatic volunteer were found to have *H. pylori* during endoscopic examination.[77,79]

Neighboring countries of Eritrea, for instance Ethiopia, the seroprevalence is estimated to be >95% in adulthood.[80] Besides, a prospective study done in USA to the immigrants of East Africa mainly from Ethiopia and Somalia who visited to a hospital with a compliant of dyspepsia, 93% (42/45 patients) of them turned out to be positive for *H. pylori.*[81]

In Eritrea also like other developing countries the prevalence of *H. pylori* and gastric ulcer due to the bacteria is high. According to the data from HMIS the prevalence of the disease is increasing from time to time .The mortality rate associated with the disease is also increasing from year 1998 to 2007 but there is relative decrease in years 2008 and 2009 considering the prevalence of the disease in 1998 the number

of cases were about 27,355 and form that it increased progressively to about 45,910 by the year 2007 but in 2008 the prevalence fall slightly to about 44,981 but it raised again to 46,633 by the year 2009.when we deal with the mortality rate from the disease, in 1998 it was only 7 but it gradually increases from year to year and reached about 28 by the year 2007 and by the years 2008 and 2009 the rate fall to about 15 and 13 respectively .[82]

2. Objectives

As a third world country Eritrea is facing with many communicable and non-communicable diseases as well as socio economic problems. The infection of *H. pylori* is becoming concern as one of the communicable diseases and is believed to cause multiple gastrointestinal diseases with its complication. This fact had an important role on choosing this particular topic, and the study was aimed:

- To determine the prevalence of *Helicobacter pylori* among patients who come to the outpatient department (OPD) with dyspeptic symptoms in Orotta Medical Surgical National Referral Hospital (OMSNRH) in the year 2012.
- To determine the prevalence of *H. pylori* among patients who did serologic test at the laboratory of OMSNRH and analyse it by age and sex.
- To analyse the prevalence of the infection by age and sex.
- To determine the risk factors of the infection.
- To compare the prevalence with the regional and global prevalence.

2.1 Methodology

2.1.1 Study design

The research was a retrospective quantitative study. It was based on hospital document that required gathering of patient card of people who sought medical help for dyspeptic symptoms as a compliant, visited the Outpatient Department (OPD) and also collecting laboratory data of patients who did serologic investigations for *H. pylori* from January 2012 to December 2012 retrospectively.

2.1.2 Study area

The study was conducted at a tertiary referral hospital Orotta Medical Surgical National Referral Hospital (OMSNRH) in Asmara, which is found in Maekel region (one of the six regions of Eritrea). The time of study was from February 2013 to May 2013. Yearly more than 30,000 people visit the hospital from different regions of Eritrea.

2.1.3 Study Subjects

The study population or subjects were those patients who visited Out Patient Department (OPD) for dyspeptic symptoms as a compliant and as the same time patients or subjects who did serologic test for *H. pylori* from January 2012 to December 2012.

2.1.4 Data collection from the OPD

After acquiring permission from the higher authorities of OMSNRH for approval and ethical consideration, the researchers gathered the diagnosis book log, which is the Health Management Information System (HMIS) for clinic/outpatient department recording form, from January 2012 to December 2012 from the archive room of OMSNRH. The form contains all diagnosed diseases in the year 2012 which is represented by a specific code for a specific disease or syndrome with card number, age, sex and others in it, **see Annex-I**. In OMSNRM, the coding system is according to the International Classification of Diseases Tabulation List for Morbidity (ICDDIAG), **see Annex-II**. Since dyspepsia is a symptom of many gastrointestinal diseases, the selection of dyspeptic patients was from those patients who were diagnosed as having gastrointestinal abnormality. For that reason the research included all codes for Gastric/Duodenal Ulcer, Gastritis/Duodenitis and other oesophageal, stomach and duodenal diseases coded by ICDDIAG as 183, 184 and 185. So, the researchers selected all patients with a diagnosis code 183, 184 and 185 from the HMIS form for clinic/outpatient department. After selection, those patients with these codes were written in new form according their card number, age and sex. All card numbers were selected and entered in data form using Microsoft excel month by month. The card numbers were arranged sequentially and repeated numbers were removed. Following the arrangement of card numbers for each month, the cards were retrieved from the archive card room of OMSNRH.

The retrieved cards were reviewed and only patients with dyspeptic symptoms were included in this study; that is all patients with chief complain epigastric pain, heartburn, bloating, regurgitation, postprandial fullness, early satiation and also nausea and vomiting as optional symptom with the other mentioned symptoms. Otherwise all non-dyspeptic symptoms and other diagnosis were excluded from the study. All retrieved cards that filled the criteria for dyspepsia were collected month by month and each card was reviewed for the serologic test of *H. pylori.* Following, patient cards were categorized as those who turned out to be positive, those to be negative and those who did do the test for *H. pylori.* And for those who were positive for *H. pylori*, the study used several factors or variables to categorize it. The variables were card number or name, age, sex, address, history of triple treatment for *H. pylori*, month and any comorbid diseases specially diseases of gastrointestinal, **see Annex-III**. Ethnicity, life style and habitation were supposed to be included since these factors affect the prevalence of the infection; however the contents of the cards had limited information about these factors.

Using the above variables all the positives were categorized month by month through Microsoft Excel 2010 and repeated card numbers were eliminated. The age range in this study was 15 years and above, since OMSNRH only engages for adults 15 years and above. Address of the patients was also categorized into 5 zones of Eritrea accordingly. History of triple treatment for *H. pylori* was also evaluated and been recorded as Yes if there was history and No if there wasn't. Moreover, the research included other comorbid diseases as a variable, especially gastrointestinal disease that could be associated with the infection of *H. pylori.*

Those who were negative for *H. pylori* was counted and inserted as a Number in Microsoft Excel 2010. Besides, those patients who came for dyspeptic symptoms as a complaint or were diagnosed as having dyspepsia but never did any serologic test for *H. pylori* were counted and been inserted in Microsoft Excel 2010 as a number.

2.1.5 Data collection from the Laboratory

Besides the data of the OPD the researchers added the data from laboratory results for *H. pylori* solely. The reasons to add those lab results were:

- The retrieved cards were only of outpatient department (OPD) excluding inpatient, emergency ward and surgical ward. However, the data from laboratory includes inpatient department (IPD), outpatient departments (OPD) and also from pediatric and Maternity hospital.
- Since the recording system was not satisfactory the researchers decided to include laboratory results of *H. pylori* to find the unrecorded ones.

Gathering of data from the laboratory was using the data log book for the year 2012 used to record the results of all tests. The data log book contains all serologic results including *H. pylori* serologic results from January 2012 to December 2012. The serologic kit used to detect *H. pylori* was a qualitative one which can detect antibodies specific for *H. pylori* (IgG, IgM, IgA, and etc). It was manufactured in USA and was revised in April 25th, 2010.

The variables in the data log book are patient's name, age, sex, sample address (OPD, IPD, pediatric or Maternity Hospital) and results for different serologic investigations. The serologic results for *H. pylori* are written as either positive or negative. Therefore, the researchers counted all results (the positives and negatives) of serologic investigation done for *H. pylori* and recorded it as a number. Following, all the positives and negatives were isolated and were recorded using their name, age, sex, and month in Microsoft Excel 2010. From the given variables, the researchers took the first four variables only because there was limited information other than the above. Patient's name was reviewed and repeated patients were removed. With that, age and sex were reviewed also. The age range of patients in the laboratory data includes all ages since the laboratory accept samples even from Pediatric Hospital (age less than 15 years).

2.1.6 Data processing

After completing the recording and reviewing of the data in Microsoft Excel 2010, all the data from the OPD and laboratory that were entered in Microsoft Excel separately had to be analysed statistically separately. For that reason the research used a new edition of Statistical Package for Social Science version 18 (i.e. PASW statistics version 18) to analyse the data. Descriptive statistics (frequency, percentage, mean and others) were used to summarize the data using this software. Pearson's Chi square was used for detection of differences in categorical variables among groups. To find out any statistical significance p value was determined and $P \leq 0.05$ was considered to be significant.

3. Results

3.1. Data from Out Patient Department

Over the twelve months of the year 2012, a total of **1,844** patients presented with dyspepsia, from **30,335** patients, to the Out Patient Department (OPD) of Orotta Medical Surgical National Referral Hospital (OMSNRH), making the prevalence of dyspepsia in adult patients **6.08% (1,844/30,335)**. Over all there were **1103 (59.82%)** Females and **741 (40.18%)** males and their age range were from **15-90** years. Further analysis showed among the dyspeptic patients, **20.93% (386/1,844)** were found out to be positive for *H. pylori*, **48.05% (886/1,844)** were tested negative for the infection and also from that retrospective study **31.02% (572/1,844)** were sent with either of Aluminium hydroxide and Magnesium hydroxide (Antacids) or Omeprazole or H_2 blockers (like Ranitidine) without doing any serologic test for *H. pylori.* **(Table-1)**

Month	Number of *H. pylori* positives (%)	Number of *H. pylori* negatives (%)	Number of untested (%)	Number of dyspeptic patients (OPD)
January	29 (22.31)	60 (46.15)	41 (31.54)	130
February	43 (31.39)	48 (35.04)	46 (33.57)	137
March	56 (36.36)	54 (35.07)	44 (28.57)	154
April	33(22.3)	81 (54.73)	34 (22.97)	148
May	29 (20.71)	54 (38.57)	57 (40.72)	140
June	12 (15)	26 (32.5)	42 (52.5)	80
July	50 (24.51)	116 (56.86)	38 (18.63)	204
August	29 (16.11)	95 (52.78)	56 (31.11)	180
September	30 (18.87)	74 (46.54)	55(34.59)	159
October	34 (18.78)	93 (51.38)	54(29.84)	181
November	20 (11.63)	89 (51.74)	63(36.63)	172
December	21 (13.21)	96 (60.37)	42 (26.42)	159
Total	**386 (20.93%)**	**886 (48.05%)**	**572 (31.02%)**	**1844**

Table-1: Dyspeptic patients visited to the OPD in 2012 and their status about *H. pylori*

3.1.1 Gender of *H. pylori* positive respondents

Patients who were tested serologically for *H. pylori* were a total of 1272. From those who were tested, **386 (30.35%)** patients were resulted positive and **886 (69.65%)** were negative for the infection. Of all the 386 patients who were positive for the infection, majority of them were females **212 (54.9%)** and **174 (45.1%)** males. From a total of 1103 females who came for dyspepsia, **19.22% (212/1103)** were positive for *H.*

pylori and from 741 males **23.48% (174/742)** were positive for the infection. The age range in the study among the positive patients was from **15-90** years with a mean age of **38.02% (SD± 16)**. **(Figure-1)**

3.1.2 Age of study population

The age characteristics of the patients who resulted positive for the infection **(Table-2)**, showed higher positivity among the age groups less than 50 years which was 75.65% 5(292/386) with the p value 0.562. The peak age group with highest rate of seropositivity was among the age group 20-29 years. **(Figure-2)**

Table-2: Characteristics of the study population by age and sex

	Sex		
	Male (%)	**Female (%)**	**Total (%)**
Age			
Under 20	14 (41.2)	20(58.8)	34 (8.8)
20 - 29	46 (43.8)	59(56.2)	105 (27.2)
30-39	37 (44.0)	47 (56.0)	84 (21.8)
40-49	34(51.5)	32(48.5)	66 (17.1)
50-59	20 (41.7)	28(58.3)	48 (12.4)
60-69	14(45.2)	17(54.8)	31 (8.0)
70-79	6 (50.0)	6 (50.0)	12 (3.1)
80-90	3 (50.0)	3 (50.0)	6 (1.6)
Total	174(45.1)	212(54.9)	386 (100)

3.1.3 Previous history of triple treatment of study population

Among the patients who turned out to be positive for *H. pylori*, the history of previous triple treatment for the infection was 3.9% (15/386) and all of them were aged less than 50 years **(Table-3)**. Majority of the patients were between 30 and 39, 40% (6/15). However, there was no significant age difference among those patients (P-value was 0.35). Moreover, males had predominance over females which were 53.3% (8/15), and a P-value of 0.512.

Table-3: Number and percentage of patients who are treated for *H. pylori*

	History of Triple treatment		
	Yes (%)	No (%)	Total P-value
Sex			**0.512**
Male	8(4.6)	166(95.4)	174
Female	7(3.3)	205(96.7)	212
Age			**0.35**
Under 20	2(5.9)	32(94.1)	34
20 - 29	3(2.9)	103(97.1)	105
30-39	6(7.1)	78(92.9)	84
40-49	4(6.1)	62(93.9)	66
50-59		48(100)	48
60-69		31(100)	31
70-79		12(100)	12
80-90		6(100)	6
Total	15(3.9)	371(96.1)	386

The data patients who had history of previous treatment were only found on the first 7 months of the year 2012 and most of them had visited to hospital on the first month 13.8% (4/15). The P-value showed significance among the males (P= 0.018). **(Figure-3)**

3.1.4 Comorbid diseases of the respondents

Patients were also evaluated retrospectively for other comorbid diseases, especially gastrointestinal diseases. Out of 386 patients, 6.2% (24/386) had different comorbid diseases **(Table-4).** Gastrointestinal diseases like duodenal ulcer, gastric ulcer, gastric out let obstruction and upper gastrointestinal bleeding due to duodenal ulcer were the comorbidities diagnosed via endoscopy, which was 2.2% of the total. Majority of them were females 58.3% (16/24) but there was no significant sex difference on comorbidity (P=0.729).

On comparing Comorbidity to age groups **(Table-5)**, there was no significant age difference (P=0.224). Most of the diseases were seen among age groups 40 and 49, which is 33.3% (8/24).

	Comorbidity		
	Yes (Total %)	**No (Total %)**	**Total P value**
Sex			**0.729**
Male	8 (5.7)	166(94.3)	174
Female	16(6.6)	196(93.4)	212
Age			**0.224**
Under 20	1(2.9)	33(97.1)	34
20 – 29	2(1.9)	103(98.1)	105
30-39	5(6.0)	79(94.0)	84
40-49	8(12.1)	58(87.9)	66
50-59	4(8.3)	44(91.7)	48
60-69	3(9.7)	28(90.3)	31
70-79	1(8.3)	11(91.7)	12
80-90		6(100.0)	6
Total	6.2%	93.8%	386

Table-5: comorbidities compared to age among *H. pylori* patients

3.1.5 Distribution of study population by Address and Month

In addition to that, the patients who were positive for the infection came from all zones of Eritrea. Most of them were from Maekel zone, with a total of 70.5% (272/386) and 53.7% of them were females. **(Table-6)**

3.2 Data from the laboratory

The serologic results for *H. pylori* of the year 2012, which was gathered from the laboratory of OMSNRH over the 4 months period showed a total of 4136. Total positive results from the serologic test were **1281(31%)** and **2855 (69%)** were negative. Majority of the patients, 61.8% (2557/4136), were females with **746 (29.2%)** of them being positive whereas 1811 (70.8%) were negative. Of 1579 (38.2) male patients, **535 (34%)** were positive and 1044 (66%) were negative for the infection (**Table-7**). The prevalence of *H. pylori* is more on males **(34%)** than females **(29.2%)** from the tested patients. The observed level of significance was 0.001 (p value < 0.05).

Table-7: The prevalence of *H. pylori* serologically				
Sex	**Number of tested (%)**	H. pylori		**P value**
		Positive (%)	**Negative (%)**	**0.001**
Female	2557 (61.8)	746 (29.2)	1811 (70.8)	
Male	1579 (38.2)	535 (34)	1044 (66)	
Total	4136 (100)	1281 (31)	2855 (69)	

3.2.1 Age and gender of study population

The mean age of the patients who did the test was **34.02 (SD ± 18.3)** with age range **1-98** years. Majority 1088/4136 (26.3%) of the patients who did the test were among the age group **15-25.** Out of all of them approximately 14% (576/4136) were pediatric age group i.e. less than 15 years old **(Table-8).**

The prevalence of *H. pylori* had showed positive correlation with increment of age till age of 25 years and at the same time after the age of 25 years, there is a gradual decrement on the prevalence *H. pylori* on both sexes. The p value, except among the age group 26-35 (p= 0.017), showed non significance. **(Figure-4)**

3.2.2 Distribution of Study population by month

When comparing the prevalence of *H. pylori* by month, the peak positivity was in March 11.2% (143/1281). As we can see in **Figure-5**, in July and August there were higher visits comparing with others. Among females higher positivity was seen in July 6% (77/1281) and for male it was seen in March 5.7% (73/1281).

3.2.3 Prevalence of *H. pylori* in Adults (greater than 14 years)

In OMSNRH, Adults are considered starting from 15 years and above. In this study, there were a total of 3560 adult patients, which is 8.6% of the total visit. From a total 2214 (62.2%) female patients, **31.7% (701/2214)** were positive and 68.3% (1513/2214) were negative for *H. pylori.* Of **1346 (37.7%)** males, 37.7% (508/1346) were resulted positive and 62.3% (838/1346) were negative. The observed p value was 0.034 between sex groups. The overall prevalence of *H. pylori* was found out to be **34% (1209/3560) (Table-9)**. The mean age of the patients were **37.83** years **(SD±16.8).**

3.2.4 Prevalence of *H. pylori* in pediatric age group (less than 15 years)

Among the paediatric age group, the mean age was **10.5** years **(SD± 3.3)** with age range **1-14**. Of all 576 patients, 72 (12.5%) were positive and 504 (87.5%) were negative for *H. pylori*. From 343 (59.5%) females, 45 (13.1%) of them were tested positive and 298 (86.9%) were negative. Similarly, out 233 (40.5%) males, 27 (11.6%) were positive and 206 (88.4%) were negative **(Table-10)**. Females tend to be more infected than males among age group less than 15. The observed level of significance was 0.585.

Similar to the adult age groups, there is a positive correlation between increment of age and *H. pylori* prevalence. **(Figure-6)**

3.2.5 Distribution of pediatric age groups by month

The percentage of pediatric patients and their distribution by month for each sex is shown in **Figure-7**. The peak positivity for females was in February and for males it was on September. In May, June and July there higher number of visits comparing to the other months. (**Figure-8)**

4. Discussion

Dyspepsia remains the main gastrointestinal symptom which is costly, chronic condition and increasing in frequency.[83] In western population, it is estimated to have a prevalence of 25%, which accounts 2-5% of a primary care consultation.[54] Yearly, women tend to be more affected than men (14 per 1000 vs. 10 per 1000) as it is done in one study in USA.[84] In prospective study done in Malaysia at a primary care centre over a six period of time, showed a prevalence of dyspepsia to be 1.12%.[85] In this study, the prevalence of dyspepsia was 6.08% with female predominance (60% vs. 40%).

H. pylori is an organism with extremely high incidence in the world and there is strong association between the infection and gastro intestinal diseases like gastric or peptic ulcer.[56,83] Different studies showed different results in the prevalence of *H. pylori* among dyspeptic patients visited to primary care centres. There is marked difference in the prevalence between developing (>50%) and developed country (<50%). In Ibadan, Nigeria, a prospective study among dyspeptic patients in adults showed an overall prevalence of *H. pylori* to be 64%.[84] Similar study done in Malaysia, the prevalence was 23.5%.[85] In Kuwait also, among patients with dyspeptic symptoms ranged from 10-80 years, revealed a prevalence of 42.6% among Kuwaiti and 57.6% among expatriates.[83] On the other hand, the study done in OMSNRH revealed that among the dyspeptic patients 20.93% positive, 48.05% negative and at the same time 31.02% untested for *H. pylori*. However, the seroprevalence from the laboratory in adults (15-98 years) resulted to be a total of 34%. In different developing countries, seroprevalence of *H. pylori* is high. For instance, in Algeria the seropositivity with in age range 5-65 years was estimated to be 43%-92%. In South Africa, among age group ranging from 2 months to 87 years was 50-94%. Also in India (age ranged 3-81 years), seropositivity of *H. pylori* was from 60-81%.[77]

In pediatric age, there is higher prevalence in developing countries. The seropositivity in Nigeria aged 6 months to 2 years was to 57%, rising to 82% between 5 and 9 years. Likewise in Gambia, in rural children aged 5 months to 3yrs the seropositivity was 31%. In Ethiopia, the seropositivity in children aged 2-4

years was 48%, reached upto 80% by 6 years of age.[77,81] Comparatively, in this study in children aged 1-14 years, seropositivity was found out to be 12.5% i.e. it was 3.4% among age group 1-5 years, 9.8% among 6-10 years and 15.6% among 11-14 years. Similar to the other studies there is an increment of seropositivity with the rise of age, reaching peak in older age groups. In this studied pediatric age groups, female gender preference was seen (13.1% vs.11.6%, P=0.585).

Gender preference in this study from dyspeptic adult patients visited the OPD; though females to males ratio was 60–40%, males tend to be infected more than females (23.48% vs. 19.22%). However, there was no significant gender difference. On the other hand, seopositivity among adults from the laboratory showed male sex preference (37.7% vs. 31.7%) from a total of 58% females and 42% males. P value showed significance (p=0.034). While comparing to a similar study done in Northern Nigeria at a tertiary hospital, male predominance was shown (77.3% vs. 75.4%) but there was no significant sex difference (p>0.05).[60] Also in Kuwait among the expatriates, significant male predominance (66% vs. 40%, p=0.002) was shown.[83] Male predominance was seen in many studies but not in children.[36–39] There are varying reports of higher prevalence in either male or female, and also varying level of significant difference between infectivity and sex varies.[83,87]

Age distribution among different studies showed different results. At Lyari general hospital in Pakistan among adults, the peak age infectivity was among age group 21-30 years (28.58%, P value > 0.05) with 85% of the positives were less than 50 years old.[88] In Kuwait also, the peak age was among age group 30-49 years and majority of the positives were less 50 years (63.7%).[83] One study in Nigeria resulted the peak of infection was among 31-40 years. Yet no significant age difference occurred in these studies. In this study, the peak of infection in adults was 20-29 years (27.2%) from the OPD and 15-25 years (24.3%) from laboratory results. Majority (75%) of the patients were less than 50 years. Nonetheless no significant age difference is shown. There is positive correlation between age and *H. pylori* infection especially till the age of 50 years. Several studies showed a positive correlation between age and infection rate.[83,88,89] Unlike those other studies, in Tibetan refugees settled in Southern India, the prevalence of *H. pylori* was found not to rise with age but became lower in those older than age 40 years.[77] Similarly in this study, there is gradual regression in the rate of infectivity beyond 40 years of age.

H. pylori and other gastrointestinal disease especially peptic ulcer disease are significantly associated. Only about 15% of H. pylori-infected people develop an ulcer in their lifetime.[55] A study done in Nigeria at endoscopic centre, from 64% (55/86) *H. pylori* positive patients, 12.7% (7/55) patients were diagnosed as having serious Gastro duodenal comorbidities (Duodenal and Gastric ulcer, Gastric cancer). No significant level of difference was seen. In this study, 2.2% of the positives had different gastrointestinal comorbidities that could be associated with *H. pylori.* Further prospective study involving a larger number of patients is needed to establish the true association between the infection and comorbidities.

Address as a risk factor for *H. pylori* is documented in many studies. In a large cross-sectional seroprevalence study conducted in Southern China, the overall prevalence of *H. pylori* infection was

44.2%; a significantly higher prevalence was found in the urban areas (52.4%) than in the rural areas (38.6%).[77] Address of the study population in this study was mainly from Asmara, Maekel zone. The reason for this could be the location of the study area, OMSNRH which is found in this city (zone). And the positives are mainly from this region (70%). Maekel region is mainly considered as urban because most of its population is in the urban city Asmara.

From the study population, the highest visits to both (OPD and laboratory) were in July, making the total percent to be 11.1% for both of them. Following in August and October showed higher visits. The reason could be, since these months are in summer season, most our population especially students take vacation during this season.

In contrast, highest positivity rate was seen during the rainy season in March and July from both the OPD and laboratory. This could be explained that one of the main transmissions of *H. pylori* i.e. fecal-oral route, is high during this rainy season due to contamination of water supply. In Peru, a case-control study found that children using municipal water are three times more likely to acquire *H. pylori* than are children using an internal water source. Also the presence of *H. pylori* in their sewage system was reported. Another study in Chile, consumption of raw green vegetables was strongly correlated with *H. pylori* seropositivity.[77] Nonetheless; further detailed study is needed to explain these correlations.

Regarding the previous history of triple treatment, it is showed to be almost 4%. Nevertheless, the serologic test is an antibody test, it is not recommended to be a test of choice for those who were treated previously. Other tests like urea breath test or biopsy are recommended.[80]

5. Conclusion

The study was conducted in dyspeptic patients visited OMSNRH and also who did serology for *H. pylori* in laboratory of the hospital, to find their prevalence and whether dyspepsia was due to the infection of *H. pylori* or not. Besides, it was meant to identify the prevalence and risk factors of the infection at the same time compare by age and gender. The literature review revised the overall prevalence of the infection globally and regionally and its risk factors, and dyspepsia prevalence.

In this study, prevalence of dyspepsia (with female predominance) who visited the hospital is similar to other studies done. Though there were cases (one third of the total) who were untested for the infection, *H. pylori* related dyspepsia tested serologically is comparative with other studies. Unlike the developing countries, the overall prevalence of the infection among all age groups found from the laboratory was proportional with the prevalence of the developed countries.

Significant male gender preference over female was seen among the adults from laboratory data. In pediatric age, it was the other way round with no significant difference.

Age as a risk factor, in the study, were reviewed from documented data (cards) of the OPD retrospectively. It has been shown that, there was proportional *H. pylori* infection with age till the fourth and fifth decade

of life and then gradual regress after the fifth decade. The peak of infection was at younger ages in the third decade of life (twenty's).

The study population was mainly from urban area Asmara. This was due to the location of the study area in this city. The association of address and the infection as a risk factor is not conclusive based on this study.

It is also found that the highest seropositivity was seen during the rainy season, in March, July and August. However, conclusive ideas could not be given from this retrospective study.

Reinfection of previously treated patients could not be reliable with serologic investigation for *H. pylori*, since it only detects the specific antibody not the bacteria.

H. pylori related gastrointestinal comorbidities were also observed among the positives in this study.

5.1 Recommendation

- From this study, not all patients were tested for *H. pylori.* It would be more appropriate that all patients who came to a high level tertiary hospital for dyspepsia should have to be evaluated thoroughly.
- Diagnosis of *H. pylori* is difficult in our region. Update, more sensitive and specific, and cost effective diagnostic modalities like Stool antigen test (SAT), rapid urease test, $_{13}C$ urea breath test and $_{14}C$ urea breath test would be suitable for accurate diagnosis of the infection. Besides, endoscopy guided biopsy is the most specific diagnostic modality for the infection that could be instituted for appropriate patients.
- Computerized data registration for the diagnosed diseases and codes, at the same time registration of cards and laboratory results of patients, would be most suitable storing system.
- The diagnosis code using the ICDDIAG in the HMIS form is most of the time written by nurses or health assistances, thus diagnosis error is common. It would be advisable if every physician writes the code of his/her own diagnosis neatly in the HMIS form.
- There is a need to improve our understanding of the modes of transmission, risk factors, immune-pathogenesis of the associated Upper Gastro intestinal tract diseases, and diagnosis and treatment regimen of *H. pylori* infection in our region.
- Further Large-scale population-based prospective studies are needed in Eritrea to have accurate information about the infection.

5.2 Limitation of the study

- The study was a retrospective quantitative study which is prone to bias and less informative.

- There was limited information on the cards or data log books concerning address, ethnicity, past history, risk factors and also there were few missed cards.
- Among the dyspeptic patients from the OPD, there was significant number of patients who did not do any investigation for the infection, that limit the researchers to have accurate results.
- The only test kit used to detect an infection of *H. pylori* was serologic test kit which is less accurate, does not specify active infection, not recommended after therapy and with less positive predictive value (64%).[80]Moreover, the serologic test used in this study is not recommended for children.

Declarations

The authors have no conflict of interest to declare on this study.

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Tables 4, 6, 8, 9, And 10

Table 4, Table 6, Table 8, Table 9, and Table 10 are available in the Supplementary Files section.

Figures

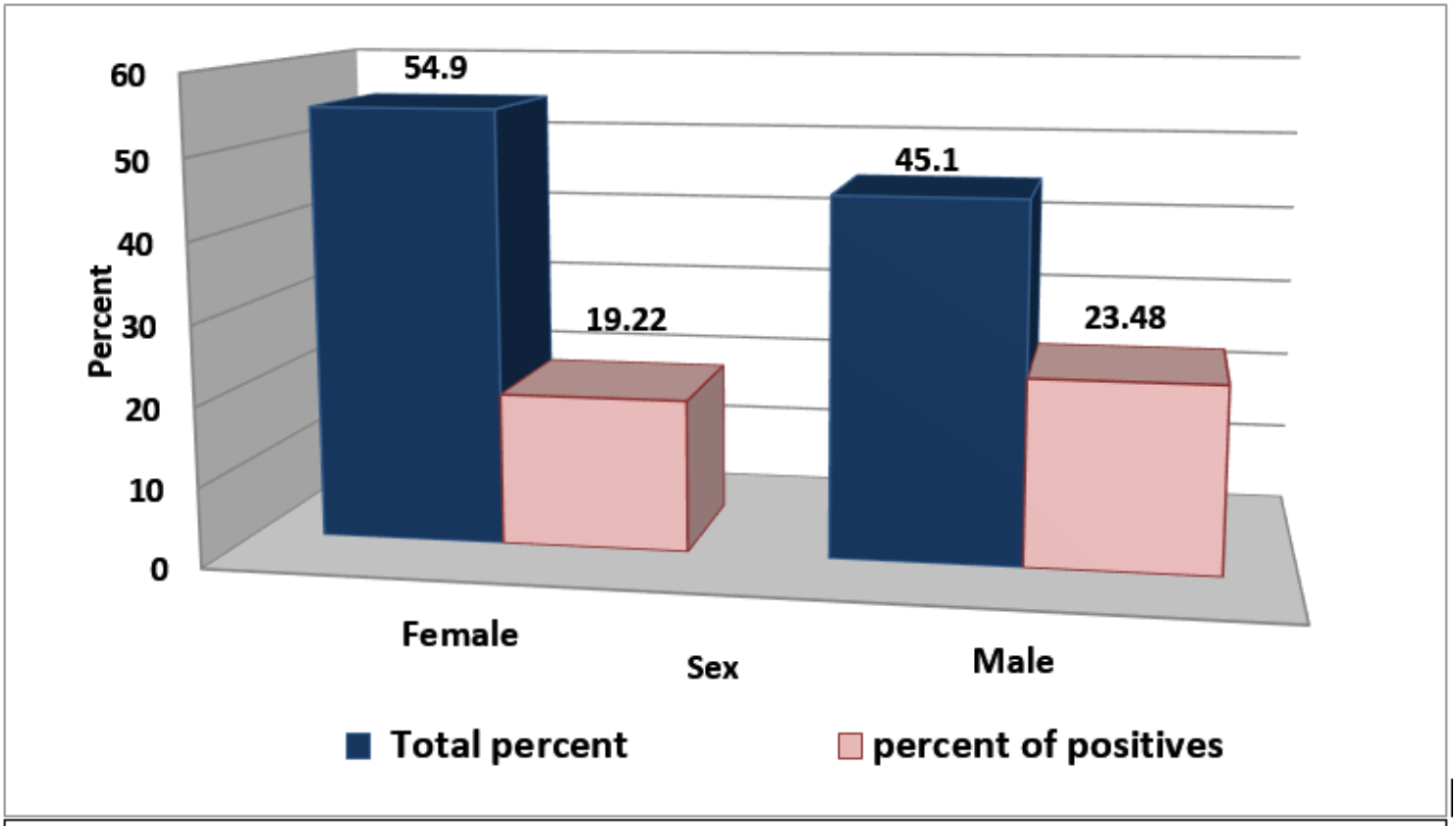

Figure-1 percent of *H. pylori* positive patients by sex

Figure 1

percent of *H. pylori* positive patients by sex

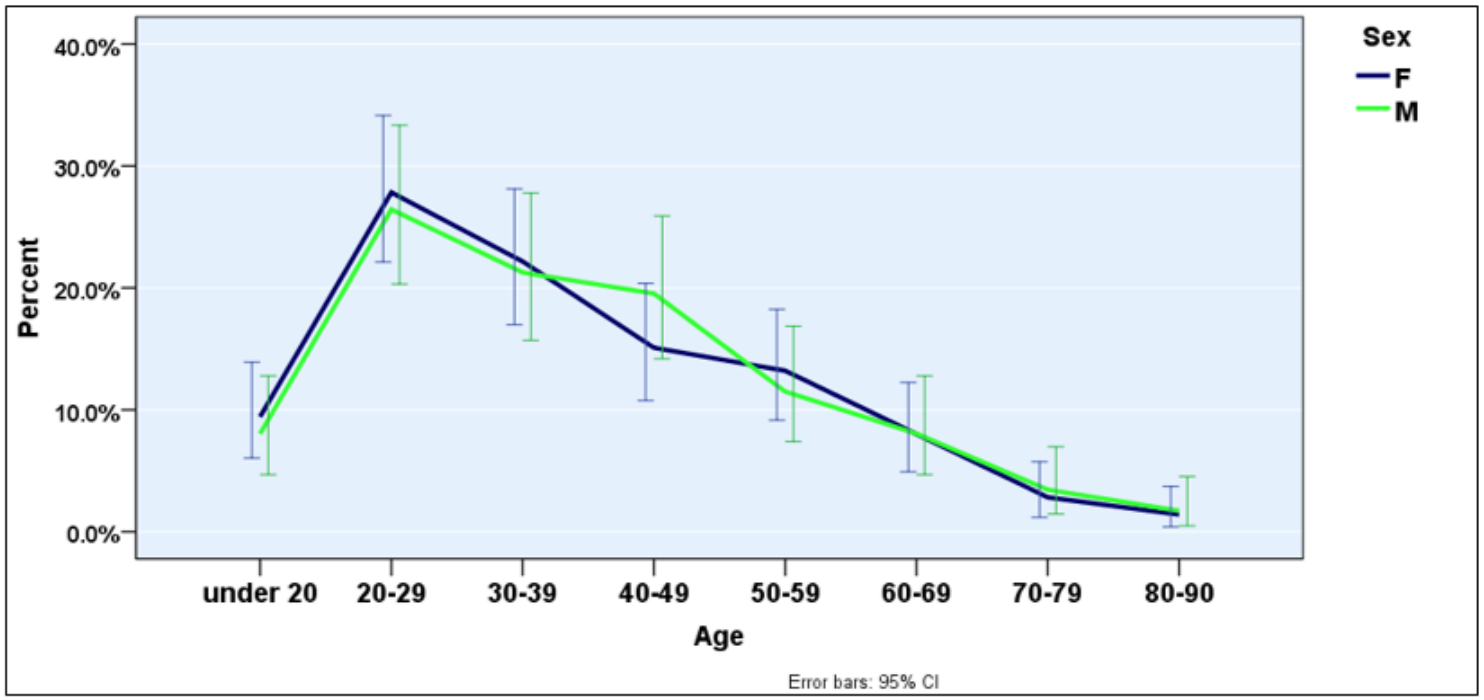

Figure-2: Distribution of *H. pylori* by age groups and sex

Figure 2

Distribution of *H. pylori* by age groups and sex

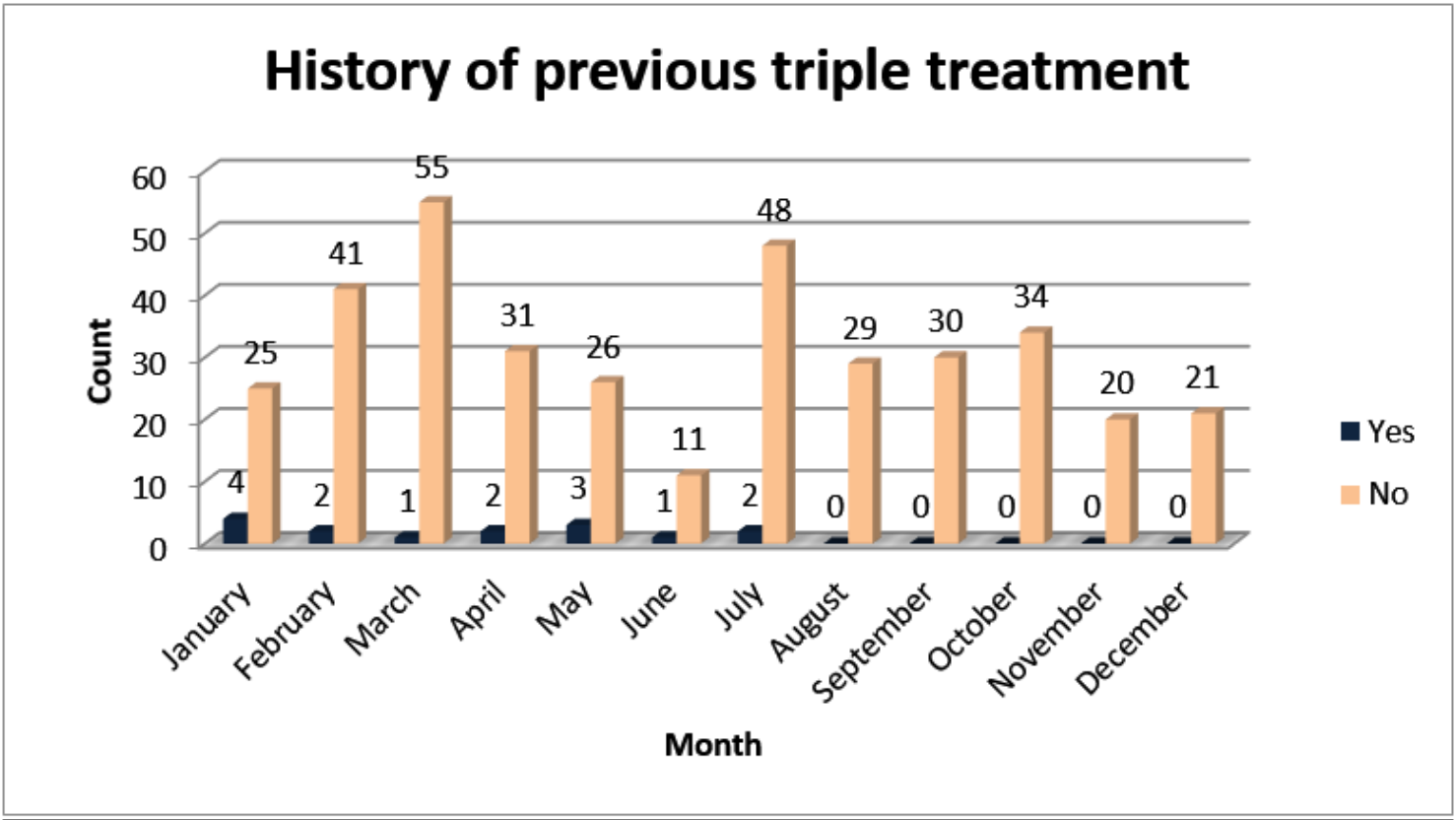

Figure-3: Distribution of patients who were treated for *H. pylori,* by month in the year 2012

Figure 3

Distribution of patients who were treated for *H. pylori,* by month in the year 2012

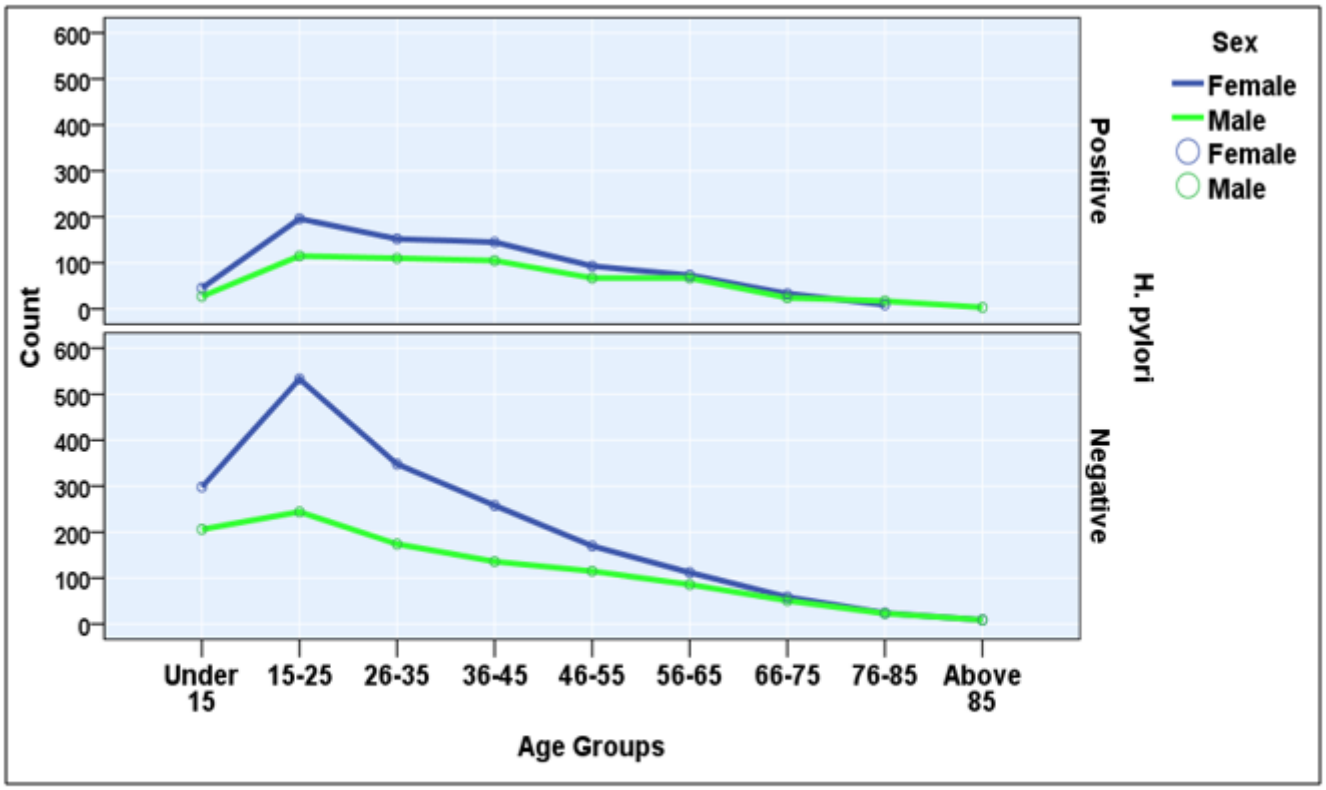

Figure-4: The prevalence of *H. pylori* among age groups by sex

Figure 4

The prevalence of *H. pylori* among age groups by sex

Figure 5

Distribution of *H. pylori* by Month

Figure 6

Distribution of *H. pylori* by sex in pediatric age groups

Figure 7

Distribution of *H. pylori* results in pediatric patients by sex and month in the year 2012

Figure 8

Total number of pediatric patients who did serology for *H. pylori* distributed by month

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Annexes.docx
- Table4.png
- Table6.png
- Table8.png
- Table9.png
- Table10.png