PREVALENCE OF HELICOBACTER PYLORI INFECTION
IN
PATIENTS WITH ENDOSCOPICALLY PROVEN PEPTIC ULCER
DISEASE
AT DR GEORGE MUKHARI HOSPITAL

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PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS WITH ENDOSCOPICALLY PROVEN PEPTIC ULCER DISEASE AT DR GEORGE MUKHARI HOSPITAL

By

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At the

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Supervisor: Dr RR Makinita
Co-supervisor: Prof MZ Koto
2013
DECLARATION

I declare that the…………………………………………….(mini-dissertation / dissertation / thesis) hereby submitted to the University of Limpopo, for the degree of …………………………… (degree & field of research) has not previously been submitted by me for a degree at this or any other University; that it is my work in design and in execution, and that all material contained herein has been duly acknowledged.

_____________________           _______________________
Initial & Surname (Title)            Date

____________________
Student Number
DEDICATION

To my loving fiancée Ntokozo and, to my parents Maria and Samuel Ndlovu
ABSTRACT

Introduction: Reports have shown prevalence of Helicobacter Pylori (H-Pylori) infection in developing countries is between 50% and 90%. Despite the higher incidence only less than 20% of those infected will manifest with gastro-duodenal symptoms. Despite recent reports of decline in prevalence of H-Pylori infection, sizable number of patients still present with bleeding, intractability, gastric outlet obstruction, and perforations. H-Pylori infection causes mucosa-associated lymphoid tissue (MALT) lymphoma of the stomach, and there is strong environmental factor related to development of chronic atrophic gastritis which leads to gastric adenocarcinoma.

Aim: To determine the incidence of H-Pylori infection in patients with endoscopically proven benign ulcer disease presenting at Dr George Mukhari Hospital and to determine the prevalence of smoking and non-steroidal anti-inflammatory (NSAIDs) use within the study sample.

Methods: We retrospectively collected records of all patients treated at our institution for benign peptic ulcer disease. We included all patients who were found to have benign peptic ulcer at gastroscopy. The patient’s demographic data, gastroscopy findings and CLO test results were looked at, from January 2009 to August 2012. We excluded all patients with malignant gastric and duodenal lesions.

Results: Total of 316 patients were included in our series (160 males, 153 females & 3 unknown gender), age ranges from 12-103 years, mean age 54.56 years, incidence of H-Pylori: n=155(49.05%) patients tested negative for H-Pylori; n=100 (31.65%) tested positive and the remaining 61 patients (19.30%) did not have a test result. Smoking and NSAIDs use not analyzed because the information was not recorded in 215 patients. Correlation between socioeconomic status and H-Pylori infection was not shown because of unrecorded information about living conditions at home like overcrowding, poor sanitation and lack of running water.

Conclusion: Prevalence of H-Pylori infection at Dr George Mukhari Hospital is lower than the reported incidence in developing countries. Probable because of improved water purification methods and improved sanitation.
ACKNOWLEDGEMENTS

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CHAPTER 1

INTRODUCTION

1.1. BACKGROUND

Prevalence of Helicobacter Pylori (H-Pylori) is well documented among patients with peptic ulcer disease. But the study of prevalence of H-pylori infection at Dr George Mukhari hospital has never been conducted. It appears that 90% to 95% of duodenal ulcer and 50% to 70% of gastric ulcer patients harbor H-Pylori infection; yet only 20% of the infected individuals develop peptic ulcer disease during their lifetime (Knigge 2001).

The prevalence also depends on the status of industrialization of the country, as the incidence in the United States is between 20% and 50% compared with 50% and 90% in most developing countries (Go 2002; Torres 2000). Infection with H-Pylori occurs in childhood when opportunities for transmission are higher and the host immune response to inoculum is relatively weaker than in adulthood (Suerbaum 2002). The human host is the chief reservoir of H-Pylori, but contaminated water is also implicated in infection transmission (Brown 2002; Moss 2003).

The fecal-oral and oral-oral routes of spread are most likely person-to-person transmission modes. Transmission rate increases when living condition are crowded and poor sanitation, as higher infection rates are found in the densely populated areas in developing countries (Suerbaum 2002; Moss 2003). In areas of low socioeconomic conditions where poor sanitation, contamination of water supplies, crowding, and poor personal hygiene are common, the likelihood of infection is higher, particularly among the children (Suerbaum 2002; Moss 2003).
In patients who are not infected by H-Pylori, yet have peptic ulcer disease, it is mandatory to do a thorough search for alternative causes of peptic ulcer disease. Use of non-steroidal anti-inflammatory drugs (NSAIDs) is implicated in the pathogenesis of peptic ulcer disease.

A population-based study by Rosenstock (2003, p. 186-93) is a 11-year prospective cohort study of more than 2400 Danish adults demonstrated the major risk factors for peptic ulcer disease to be H-Pylori infection and tobacco smoking. But in this study NSAIDs use did not affect the incidence of peptic ulcer disease. Despite recent reports of decline in prevalence of H-Pylori infection, patients with complicated peptic ulcer disease are still admitted at Dr George Mukhari hospital, with bleeding, intractability, gastric outlet obstruction, and perforations.

Helicobacter Pylori infection causes mucosa-associated lymphoid tissue (MALT) lymphoma of the stomach, and there is strong environmental factor related to development of chronic atrophic gastritis which leads to gastric adenocarcinoma (Hartmann 1987; Parsonnet 1994).

All patients with peptic ulcer disease proven at endoscopy are given eradication therapy, which entails Omeprazole, Amoxicillin, and Metronidazole or Clarithromycin. This treatment is given to all patients who have peptic ulcers, even without proof of infection with H-Pylori. A thorough literature search was conducted and there was no data, which looks at incidence of H-Pylori infection in an environment like Dr George Mukhari hospital, which provides services for both urban and rural communities.
1.2. AIM

The aim of the study is to determine the incidence of Helicobacter pylori infection in patients with endoscopically proven benign ulcer disease presenting at Dr George Mukhari Hospital and to determine the prevalence of smoking and non-steroidal anti-inflammatory use within the study sample.

1.3. ETHICAL ISSUES

This study was approved by MREC at the university of limpopo MEDUNSA Campus (Appendix 1)

1.3. OBJECTIVES

1. To determine the prevalence of Helicobacter Pylori in the above stated population of patients.
2. To determine the prevalence of smoking and non-steroidal anti-inflammatory use within the study sample.
3. To determine the incidence of poor socioeconomic status within the study sample.
1.4. RATIONALE OF THE STUDY

- We notice that all patients at Dr George Mukhari Hospital with endoscopically proven peptic ulcer disease are treated empirically for H-Pylori infection.
- The regimen used is:
  - Omeprazole 20mg daily per os for 6 weeks
  - Amoxycillin 1g 12 hourly per os for 14 days
  - Metronidazole 400mg 12 hourly per os for 14 days or Clarithromycin 500mg per os for 14 days.
- Despite the treatment a sizable number of patients remain symptomatic, and
- A number of patients are still admitted to surgical wards with complicated of peptic ulcer disease (perforations, bleeding, intractability and gastric outlet obstruction).
- The eradication therapy will be considered in patients with proven infection by H-Pylori, this will eliminate unnecessary use of antibiotics, and raising incidence of antibiotic resistance to H-Pylori.
CHAPTER 2

LITERATURE REVIEW

2.1. DEFINITION

Peptic ulcer is defined as an erosion of segment of mucosa of the gastrointestinal tract, which occurs in the stomach or first part of duodenum (Sarosi; 2005).

2.2. Historical Perspective

In 1984 Marshall and Warren published a seminal paper entitled “unidentified curved bacilli in the stomachs of patients with gastritis and peptic ulcer disease”, this overridden belief of human stomach environment is too acidic and hostile to allow microbial colonization or infection (Warren 1983; Marshall 1983). However earlier in 1893 Bizzozero presented to the royal Academy of medicine of Turin his observation of 3-to-8-μm spirilla that inhabited the superficial zones of pyloric glands.

2.3. AETIOLOGY

Originally Helicobacter pylori organism was referred to as Campylobacter Pyloriditis Helicobacter Pylori, a corkscrew shaped, micro-aerophilic gram-negative coccobacillus (0.5μm wide by 2-4μm long), equipped with two to six flagella that are lophotrichously positioned (fig 1). Currently over 50% of the world’s population is infected with Helicobacter Pylori and the prevalence varies from one population to another.
Socioeconomic status of a population is strongly related to Helicobacter Pylori infection rates. Over 80% of a population in developing countries is infected with Helicobacter Pylori. It has been observed that in developed countries the prevalence of Helicobacter Pylori infection has declined dramatically (Le 2007; Stabile 2005). Epidemiological data indicates that the prevalence of infection corresponds to improved sanitation, less overcrowding and advanced water purification methods.

The human host is the chief reservoir of Helicobacter Pylori, but contaminated water sources have also been implicated in infection transmission (Brown 2002; Moss 2003). Oral-oral and faeco-oral routes of spread are the most likely person-to-person transmission modes. The likelihoods of both are optimized when living conditions are crowded and unsanitary, and indeed the highest infection rates are found in the densely populated areas in non-industrialized countries (Moss 2003; Warren 1983).

FIG 1. H-Pylori organism
2.4. PATHOGENESIS

**H-Pylori**

Infection by *H-Pylori* is presumed to start from the stomach antrum and extends down to the corpus after extensive mucosal damage (Akada 2003). Colonization unavoidably stimulates nuclear factor-kappa B (NF-B) activation and interleukin-8 (IL-8) expression in gastric epithelial cells (Kim 2003).

Toll-like receptor 2 (TLR2) and 5 (TLR5) recognize *H. pylori* and initiate signaling pathways that result in enhanced activation of NF-B; IL-8 is secreted by the host cells to attract components of the innate and adaptive immune systems to the site of infection. This polarizes the immune response towards a Th110 response, further attracting inflammatory cells and T-lymphocytes. An effective CD4 +T-cell response is essential to clear *H-Pylori*, however this organism has been shown to inhibit CD4+T-cell proliferation and arresting IL-2 cell-cycle progression resulting in avoidance of clearance thereby staging an infection (Gebert 2003).

Initial infection by highly pathogenic strains possessing a cluster of genes known as the cag pathogenicity island result in altered expression of several genes associated with glycan biosynthesis especially _3GlcNAc T5, a GlcNAc transferase required for the biosynthesis of Lewis antigens (Sundrud 2004). Resultant over expression of _3GlcNAc T5 in human gastric carcinoma cell lines lead to increased sialyl–Lewis x expression, a specific kind of sugar molecule that these cells display on their surface as a flag to attract immune cells to the infection site (Marcos 2008; Nagorni 2008; Bor-Shyang 2006).

Among a number of adhesins, this organism uses bacterial adhesion protein called sialic-acid binding adhesin (SabA) to recognize a molecule associated with inflammation and a molecule known as Lewis B antigen binding adhesin (BabA) to adhere to the inflamed cells of the glandular lining (Bor-Shyang 2006).

The ability of *H-Pylori* to adjust its adherence properties to the level of inflammation it causes at the stomach surface could help explain how this bacterium maintains its persistence, decades-long infection in the stomach of millions worldwide.
NSAIDs

The anti-inflammatory effects of NSAIDs acts by inhibition of cyclo-oxygenase (COX) 1 and 2, it’s known that COX 1 inhibition will lead to decrease production of mucus barrier system and that promotes damage to the mucous membrane by acid (Pierre-Alain 2007).

2.5. CLINICAL PRESENTATION

Uncomplicated peptic ulcer usually present as: epigastric pain, with or without associated symptoms of bloating, early satiety, or nausea. Patient can present with either duodenal or gastric ulcer. Physiological explanation of duodenal ulcer develops in a hypersecretory gastric acid milieu appears to be related to the specific transitioning of H-Pylori gastritis to more serious localized antral gastritis (Go 2002; Suerbaum 2002). Minority of patients will eventually develop duodenal ulcers, because of associated increased gastric acid production. Development of antral gastritis as a result of H-Pylori infection will lead to attenuation of D-cell, hence acid overproduction since there impaired somatostatin release (el-Omar1993; el-Omar 1995).

The etiologic role of H-Pylori in gastric ulcers are less dramatic than duodenal ulcers, unlike duodenal ulcers gastric ulcer disease is associated with normal secretion of acid or even low secretion of acid as compared to hyper-secretion of acid seen in the duodenal ulcers.

Causative agent of gastric ulcers is less frequently associated with H-Pylori infection than duodenal ulcers; this means other etiologies are implicated in gastric ulceration (Knigge 2001). Classification of gastric ulcer disease is vital as far as pathogenesis and location is
concerned. The original Johnson classification system has been modified and widely used (Fig 2) (Johnson 1965; Lickstein 1997).

Type I gastric ulcer is situated along the lesser curvature of the stomach. Type I ulcers are the most common, they occur in a setting of acid hypo-secretion. Type II gastric ulcers combined gastric and duodenal ulceration, they are found on the lesser gastric curvature but may be located anywhere in the stomach and are thought to be related to gastric stasis and the hyper-secretory. Type III gastric ulcers occur in the pre-pyloric or pyloric area, like type II it occurs in a setting of acid hyper-secretion. Type IV gastric ulcers occurs in lesser curvature higher up, 1 to 2 cm within cardia of the stomach.

Type V gastric ulcers located anywhere in the stomach and are caused by NSAID intake, are superficial, sometimes multiple and with associated erosions of the stomach, and are infrequently symptomatic.

**FIG 2.** The 5 types of benign gastric ulcer
2.6. DIAGNOSIS

Diagnosis of Helicobacter Pylori infection can be made using invasive or non-invasive test. Among the array of diagnostic test for H-Pylori infection, presently there is no single gold standard test available. Nonetheless invasive test are more reliable but require endoscopic biopsy.

The urease test is invasive test, which can yield results within 3 hours; it makes the test an excellent modality for use by surgeons in their endoscopy suites. The urease agar test [CLO-test (Kimberley Clarke) ®] uses a prepared urea based agar containing a Ph-indicator. Gastric tissue taken from the antrum of the stomach is placed in the urea-based agar. In the presence of Helicobacter Pylori, which produce urease, a positive sample converts the urea to carbon dioxide and ammonia, and it changes the colour of the indicator (Fig 3).
Sensitivities ranges between 80% and 95% and specificities are as high as 95% to 100%. But specificities decreases substantially in a setting of proton pump inhibitor use, and achlorhydria (Laine 1997). Both sensitivity and specificity are decreased with bleeding ulcers and after medical therapy for Helicobacter Pylori (Tu 1999; Nishikawa 2000). Rapidity of urease agar test gives it an advantage over and above other tests, because in single visit a clinician is able to diagnose and treat H-Pylori infection.

Rapidity of urease agar test gives it an advantage over and above other tests, because in single visit a clinician is able to diagnose and treat H-Pylori infection.

Histological examination is another invasive modality for diagnosis of H-Pylori infection, which involve retrieval of antral mucosal specimen through endoscopy. Histological analysis involves the use of a number of staining techniques. However, data suggests that use of modified Giemsa stain is cost-effective and simple to perform (Rotimi 2000). Majority of laboratories use hematoxylin and eosin technique, this modality is associated with high false negative especially for low-density infection (Moayyedi 1998). Other types of stains available for histological analysis are Steiner, Warthin-starry, Gimenez, and Genta.

Polymerase chain reaction (PCR) was developed in the early 1990s, it has an ability to detect and amplify the presence of virtually any living organism. PCR allows detection of cagA, which helps to classify patients into high risk, warranting the need for prophylactic eradication or alternatively surveillance using endoscopy. Bacterial culture is type of invasive test, which involves culture of H-Pylori in vitro, its sensitivity ranges between 60% and 90% and specificity is as high as 100%(Rautelin 2003).

Polymerase chain reaction (PCR) was developed in the early 1990s, it has an ability to detect and amplify the presence of virtually any living organism. PCR allows detection of cagA, which helps to classify patients into high risk, warranting the need for prophylactic eradication or alternatively surveillance using endoscopy. Bacterial culture is type of invasive test, which involves culture of H-Pylori in vitro, its sensitivity ranges between 60% and 90% and specificity is as high as 100%(Rautelin 2003).

The most sensitive and specific non-invasive diagnostic test currently available is the urease breath test; it has an overall accuracy of more than 95%. Two versions of the test are available: a heavy $^{13}$C-urea and a radioactive $^{14}$C-urea. The action of urea by Helicobacter Pylori produces ammonia and labeled carbon dioxide derived from degradation of labeled urea, which is ingested orally by the patient. The labeled carbon dioxide is then measured in the patient’s expired breath (Fig 4).
Fig 4. Schematic diagram of the urea breath test

Serology is one of the earliest tests developed, which is cheap and widely used; it detects IgG against H-Pylori in blood. The major disadvantage of serology test is an inability to distinguish prior exposure from active infection. Antibodies formed in response to H-Pylori infection often remain in the blood for years, even after successful eradication therapy.

Stool antigen test uses intestinal bacterial by-product as a probe for H-Pylori, monoclonal and polyclonal antibodies are used to test stool specimens for H-Pylori bacterial antigens. String test is cost effective, and easy to perform but outdated test, which involves swallowing of a string by the patient, the string is then withdrawn from the stomach and tested for urease by a rapid urease test or cultured (Makristathis 2004).

2.7 TREATMENT

There are several therapeutic regimens that have been developed to eradicate Helicobacter pylori infection. The Maastricht Consensus Report of 1997 provided empirical treatment recommendations based on available clinical data, it was subsequently updated in 2002 (European Helicobacter Pylori Study Group 1997; Malfertheiner 2000). Treatment recommendations include an acid anti-secretory drug to be combined with two of the
several antibiotics, and the three commonly used antibiotics are amoxicillin, metronidazole and clarithromycin at recommended doses for duration of seven days.

The second line therapy should be initiated immediately whenever the first line triple therapy failure is demonstrated by follow-up testing. The second line therapy should consist of 4 drugs: proton pump inhibitor, Bismuth, metronidazole, and tetracycline for duration of 7 days. The second line therapy has an eradication rate which ranges from 81% to 88%, but when the second line therapy fails a clinician is required to acquire a stomach tissue for evaluation of drug sensitivities (Gene 2003).

Studies that have been conducted to explain eradication failures implicated two factors: antibiotic resistance or poor compliance and majority of treatment failures have remained unaccounted for (Ramirez-Ramos 1990).
CHAPTER 3

MATERIALS AND METHODS

3.1. PATIENT SELECTION

The study encompassed a total of n=316 patients (160 male, 153 female; mean age 55 years) who were found to have gastric or duodenal ulcers during Gastroscopy, from period of January 2009 to August 2012 were reviewed.

3.2. SETTING

Dr George Mukhari Hospital: Department of General Surgery, and department of Internal medicine, endoscopy unit.

3.3. STUDY POPULATION

3.3.1 INCLUSION CRITERIA

The study population is defined as all the patients who are done gastroscopy at the endoscopy unit of Dr George Mukhari Hospital, for symptoms suggestive of peptic ulcer disease, with endoscopically established gastric and duodenal ulcers.
3.3.2. EXCLUSION CRITERIA

1. Endoscopic features of malignant ulcer or inflammatory bowel disease.
2. A CLO-test (Kimberley Clarke) ® or histological staining should have been performed documenting the presence or absence of Helicobacter Pylori infection.
3. Patients must have no histologic evidence of malignancy from endoscopic biopsies taken during evaluation.

3.4 INSTRUMENTS AND PROCEDURES

3.4.1. Endoscopy

Endoscopy was done in all patients with suspected peptic ulcer disease, using the Olympus gastroscope to confirm the diagnosis of gastric and duodenal ulcer and to take several antral mucosal biopsies.

The esophagus, stomach and duodenum were thoroughly examined and findings noted. Several biopsy specimens were taken from gastric antrum of those patients who had ulcers. One specimen was put in urea based agar and other was placed in 10 % Formalin and sent for histological examination.

3.4.2. H-Pylori status

H-Pylori status was determined by rapid urease test and histological examination.
3.4.3. Data Collection

We reviewed all records of patients who were found to have gastric or duodenal ulcers during gastroscopy, from January 2009 till August 2012. The endoscopy unit procedure-recording book was used to get data of all the patients who met the criteria for the study population. All specimens were sent for histological evaluation to rule out malignancy, we reviewed all the histology reports in order to exclude patients with malignancy.

3.4.3. DATA ANALYSIS

Data was analyzed using the statistical package for social sciences (SPSS) version 18 software. Prevalence of Helicobacter Pylori infection amongst the sample group was assessed from frequency tables, pie graphs and bar charts.
CHAPTER 4

DATA ANALYSIS AND RESULTS

4.1 INTRODUCTION

This chapter consists of three sections, each section consists of, short notes, interpretation, frequency tables, pie graphs, and bar charts. The first section displays the demographics of the sample (n=313). In the second section the prevalence of smoking and NSAIDs usage is presented. Lastly section three shows the incidence of Helicobacter pylori infection and whether there is an association between socioeconomic status (SES) and infection with H-Pylori.

4.2 DESCRIPTION OF THE SAMPLE

A total of n=316 patients who were found to have gastric or duodenal ulcers during gastroscopy from January 2009 to August 2012 were reviewed. There was an equal distribution of gender in the sample with males comprising 50.63% and females 48.42%. The gender category of 3 (0.95%) patients was not filled in.
The mean age was 54.56 years (SD± 17.72) and ranged from 12 years to 103 years.

Fig 5. Distribution of gender

Fig 6. Distribution of age
Using Sturge’s formula the age category was grouped into 10 groups. The majority of patients fell in the 62-71 grouped age category. The age category of 4 (1.27%) was not filled in.

<table>
<thead>
<tr>
<th>Grouped age</th>
<th>Total (n=316)</th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>12-21</td>
<td>12</td>
<td>3.80</td>
</tr>
<tr>
<td>22-31</td>
<td>28</td>
<td>8.86</td>
</tr>
<tr>
<td>32-41</td>
<td>42</td>
<td>13.29</td>
</tr>
<tr>
<td>42-51</td>
<td>39</td>
<td>12.34</td>
</tr>
<tr>
<td>52-61</td>
<td>54</td>
<td>17.09</td>
</tr>
<tr>
<td><strong>62-71</strong></td>
<td><strong>88</strong></td>
<td><strong>27.85</strong></td>
</tr>
<tr>
<td>72-81</td>
<td>36</td>
<td>11.39</td>
</tr>
<tr>
<td>82-91</td>
<td>12</td>
<td>3.8</td>
</tr>
<tr>
<td>92-101</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>102-111</td>
<td>1</td>
<td>0.32</td>
</tr>
<tr>
<td>Missing data</td>
<td>4</td>
<td>1.27</td>
</tr>
</tbody>
</table>

**Table 1.** Grouped age of patients

Patients’ occupation is displayed in Figure 7. The occupation category of 3 (0.95%) was not completed. One patient listed his occupation as an operator, and was therefore coded as a labourer. Domestic workers were included in the cleaner category.
4.2 DRUG/SUBSTANCE USAGE

As mentioned in chapter one, smoking and non-steroidal anti-inflammatory drugs (NSAIDs) use are two important risk factors implicated in the pathogenesis of peptic ulcer disease. Unfortunately, both these categories were not completed in the same 215 patients. Approximately half of the patients diagnosed with peptic ulcers do not smoke and neither do they use NSAIDs. This is shown in Figure 8 and Figure 9 respectively.

Fig 7. Occupation of patients

Fig 8. Non-steroidal anti-inflammatory drugs usage
4.3 INCIDENCE OF HELICOBACTER PYLORI INFECTION

The CLO-test is performed in order to determine the presence or absence of Helicobacter Pylori infection. The majority (n=155; 49.05%) of patients were shown not to be infected; n=100 (31.65%) tested positive and the remaining 61 patients (19.30%) did not have a test result.
Socioeconomic status of a population is strongly related to Helicobacter Pylori infection rates (Ramirez-Ramos 1990; Graham 1991; Malaty 1996). As mentioned 3 patients’ occupation was not specified, and the CLO test was not done on 61 patients, hence a cross tabulation was executed on n=252. See Figure 11 below.

![H-pylori infection by employment status](image)

**Fig 11.** H-pylori infection by employment status

In order to ascertain the significance of socioeconomic status on the presence of H-Pylori infection, pensioners were then excluded and students were categorised as unemployed. The results are shown in Table 2 below.
Table 2. Measure of association of H-pylori infection by employment status

H-pylori infection

<table>
<thead>
<tr>
<th>Employment status</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employed</td>
<td>9</td>
<td>18</td>
<td>27</td>
</tr>
<tr>
<td>Unemployed</td>
<td>52</td>
<td>60</td>
<td>112</td>
</tr>
<tr>
<td>Total</td>
<td>61</td>
<td>78</td>
<td>139</td>
</tr>
</tbody>
</table>

χ² = 1.515
p = 0.218

The results of the chi-squared test above did not reach significance [p > 0.05; χ² < 3.84]. Therefore, it is concluded that the incidence of H-Pylori infection is not influenced by employment status.
CHAPTER 5

DISCUSSION, CONCLUSION AND RECOMMENDATIONS

5.1. DISCUSSION

Reports have shown that prevalence of H-Pylori infection in developing countries is between 50% and 90%. Despite the higher incidence only less than 20% of those infected will manifest with gastro-duodenal symptoms (Go 2002; Torres 2000). The prevalence of H-Pylori infection found in this study of 31.65% (100 of 316) is lower than the reported incidences found in similar studies conducted in third world countries (Pesler1997). Dr George Mukhari Hospital provides health care for both urban and rural communities, probable this is one of the reasons this study reports low infection rate. Our study also supports the findings of decline in H-Pylori infection rate.

Prior studies have shown the importance of age, poor household living conditions and low socioeconomic status as predictors of high H-Pylori infection rate in children (Bardhan 1997). In our study the frequency of infection increased with age from 3.8% in teenagers (12-21 years), 8.86% in young adults (22-31 years), 27.85% in old people (62-71 years). The age range and H-Pylori infection rate is similar to findings of other studies, which looked at prevalence of H-Pylori infection. It is reported by Moss et al. (2003, p.445-51), that 90% of children in developing countries are infected with H-Pylori infection, however not all infections persists beyond childhood because the organism is eliminated as the child grow, and the reasons are: maturing immune system and external factors such as exposure to antibiotics.
Association of poor socioeconomic status and H-Pylori infection is well documented in many studies, sub-group analysis of study done by Rupnow MF et al. (2000, p.228-37) documented that; overcrowding and low socioeconomic circumstances are responsible for higher rates of H-Pylori infection. Our study failed to demonstrate the association of poor socioeconomic status to higher H-Pylori infection because we only looked at employment status of patients, disregarding the household living condition like overcrowding, poor sanitation and lack of running water. The information about living conditions was neither documented in bed letters of patients nor gastroscopy request form. Improvement in sanitation and water purification methods has led to decreased infection rates of H-Pylori infection (Alborzia, 2006).

Information on history of smoking and NSAIDs use was not documented in 215 patients this has led to difficulties in analyzing the association of smoking and NSAID’s use within the study sample. The reason for the missing information on history of smoking and NSAIDs use is attributed to: attending clinicians disregarding this vital information and also lack of standardized Gastroscopy requesting forms.

5.2. CONCLUSION

From the results obtained in this study, the following conclusions can be drawn:

1. This study has revealed a low prevalence of H-Pylori infection(31.65%) as compared to other similar studies

2. Our hypothesis that poor socioeconomic status is associated with high H-Pylori infection rate was not confirmed. This however does not rule out association of low socioeconomic status and high infection rate, because information on living condition was not recorded.

3. Hypothesis that smoking and NSAIDs use is a risk factors for Peptic ulcer disease was also not confirmed. The reason was lack of documentation of these vital information on patients bed letters and gastroscopy forms.
5.3. RECOMMENDATIONS

1. Similar prospective studies need to be done at Dr George Mukhari hospital, to see the true prevalence of H-Pylori infection in our setting.

2. Avoid empirical treatment of every patient with Peptic ulcer disease as this has led to increasing incidence of drug resistance.

3. Gastroscopy request forms with the following information has been suggested (see Appendix 2):
   a. Patients Demographics
   b. Symptoms
   c. Risk factors: Smoking, and Use of NSAIDs
   d. Living Conditions at home: Overcrowding, Sanitation and Running water
REFERENCES


Sundrud M.S., Torres V.J. and Unutmaz D. Inhibition of primary human T cell proliferation by *Helicobacter pylori* vacuolating toxin (VacA) is independent of VacA effects on IL-2 secretion. *Proceedings of National Academy of Sciences USA*, 2004;101:7727-7732.

