Prevalence of *Helicobacter pylori* in asymptomatic adult patients in a tertiary care hospital: A cross sectional study.

Munish Rastogi¹, Dolly Rastogi², Shraddha Singh³, Asha Agarwal⁴, B.P. Priyadarshi⁵, Tanu Middha⁶

¹Department of Microbiology, C.S.J.M University, Uttar Pradesh, Kanpur, India
²Department of Physiology, G.S.V.M Medical College, Uttar Pradesh, Kanpur, India
³Department of Pathology, K.G Medical University, Uttar Pradesh, Lucknow, India
⁴Department of Medicine, G.S.V.M Medical College, Uttar Pradesh, Kanpur, India
⁵Department of Physiology, G.S.V.M Medical College, Uttar Pradesh, Kanpur, India
⁶Department of Community Medicine, G.S.V.M Medical College, Uttar Pradesh, Kanpur, India

Abstract

*Helicobacter pylori* is well recognized as major cause of gastro-intestinal diseases. The present cross-sectional study was done in the department of Medicine at G.S.V.M Medical College. A total of 208 adult patients attending outpatient department for symptoms other than those of gastrointestinal disorders were screened to find out the prevalence of *Helicobacter pylori* using Stool antigen card test. Out of these 208 patients, 92 were found positive for *Helicobacter pylori* by the test, giving a prevalence of 44.23%. A detailed proforma was filled, regarding the age and sex of the patient, education and occupation of the head of the family, sanitary practices, dietary habits and tobacco chewing. The patients were also examined for pallor. Among a total of 143 males, 61 were found positive for *Helicobacter pylori* (42.7%) and among 65 females, 31 were found positive (47.7%). Age wise distribution showed maximum prevalence of *Helicobacter pylori* in the age group of 30-39 years (50.7%) and minimum in the age group of more than 70 years (20%). A higher prevalence of *Helicobacter pylori* was found among non-vegetarians (53.3%) and among patients having pallor (52%), though both were not significant. The study of socio-economic status showed a prevalence of 42.3% in lower middle, 44.9% in upper lower and 50% in upper middle socio-economic groups. A prevalence of 51.7% was seen among subjects chewing tobacco.

Keywords: *H. pylori*, stool antigen card test, pallor, tobacco.

Introduction

*Helicobacter pylori*, formerly known as *Campylobacter pylori* is a gram negative, curved, microphilic and motile organism. It colonizes and grows in human epithelial tissue and mucus. It is a common bacterium infecting about half the world’s population [1]. There is substantial evidence that it causes chronic gastritis, peptic ulcers, and duodenal ulcers and is also involved in the development of gastric carcinoma [2-4]. *Helicobacter pylori* was identified in 1984 [5] and further it was classified as carcinogenic to humans by The International Agency for Research on Cancers in next 10 years [6]. Actual infection rates vary from nation to nation and the developing world having higher rates than the developed countries [1, 2]. The age at which this bacterium is acquired seems to influence the possible pathologic outcome of the infection. Infections are usually acquired in early childhood in most of the countries [7]. Acquisition at an older age brings different gastric changes more likely to lead to duodenal ulcers [8]. In industrialized countries almost 50% of adults are infected, although its prevalence seems to be decreasing [9]. However, in developing countries the prevalence is higher and as much as up to 90% figure has been reported [10,11]. Once acquired, *Helicobacter pylori* infection generally persists throughout life, unless treated by specific antimicrobial therapy [10]. *Helicobacter pylori* consist of a large diversity of strains and genomes [12-16]. The study of genome is focused to understand the ability of this organism to cause disease. Twenty nine percent of the loci are in the ‘Pathogenic’ category of genome database and two of the sequenced strains have an approximately 40 Kb long cytotoxin associated gene pathogenicity island (*cag PAI*), which is a common gene...
sequence responsible or pathogenesis is containing over 40 genes. This cag PAI is usually absent from Helicobacter pylori strains isolated from humans who are carriers of Helicobacter pylori but remain asymptomatic [17]. The cytotoxin associated gene (cagA) gene codes for one of the major Helicobacter pylori virulence proteins. Bacterial strains that have the cytotoxin associated gene ‘CagA’ gene are associated with an ability to cause ulcers [18]. It is important to state that many individuals who might be harboring the bacterium do not develop any clinically apparent disease.

Several modes of transmission of Helicobacter pylori are suspected and no single pathway has been clearly identified. It has been demonstrated that houseflies have the potential to transmit Helicobacter pylori mechanically [19] and thus poor sanitation may potentiate its spread. Person to person contact is considered to be the most likely transmission route. Another important mode of transmission is iatrogenic in which tubes or endoscopes that have been in contact with gastric mucosa of one individual are used for another patient [20]. Occupationally acquired infections have also been reported especially among, endoscopists and gastroenterologists [10,20-22]. Another possible route is orofecal and Helicobacter pylori have been isolated from faeces of infected young children [10,21].

There are also studies investigating the association between the seroprevalence of Helicobacter pylori and Hepatitis A virus [23-28]. Water contaminated with faeces may be a major source of infection as is consumption of uncooked vegetables irrigated with water contaminated with seropositivity [29]. Contaminated municipal water supply has greater chances of spreading Helicobacter pylori infection as compared to private water supply [30]. Sporadic isolates have been found from dental plaques and saliva [31,32]. Various socio-economic conditions comprising of high density crowding, poor sanitary practices, family income, educational level and occupation [33-35] have been held responsible in spreading of the pathogen. In developing countries, factors related to community and religion might also be responsible [36]. Early detection of Helicobacter pylori population and its eradication may result in significant improvement in severity of dyspeptic symptoms. It is important to find out Helicobacter pylori prevalence and identify high-risk population so that treatment strategies can be appropriately planned. This becomes even more important for those patients who are harbouring Helicobacter pylori but are asymptomatic.

Hence the present hospital-based cross-sectional study was done on patients attending OPD for conditions other than gastro-intestinal disorders and the prevalence of Helicobacter pylori was estimated. Other parameters like socio-economic status according to Kuppuswamy grading, age, sex, diet, pallor and tobacco consumption were also studied and association of Helicobacter pylori if any, was identified.

**Material and Methods**

The study was conducted in the department of Medicine at GSVM Medical College, Kanpur in collaboration with department of Physiology at KGMU, Lucknow during 2012-2013. A total of 208 patients of both sexes attending OPD for symptoms other than those of gastrointestinal disorders were screened for Helicobacter pylori. Written informed consent was taken from all the patients after explaining to them the nature and purpose of study. Ethical clearance was taken prior to the study from the ethical committee. Patients who had taken proton pump inhibitors or antibiotic for a month prior to study were excluded. Patients’ stool samples were collected in airtight containers and stool assay was performed using Immunocard STAT HpSA test (Standard diagnostics Inc).

The test device and the sample were brought to room temperature prior to testing. The test device was laid on a flat dry surface and about three drops of the prepared sample were poured into the sample well. Interpretation was done after fifteen minutes. Two colour bands, one at test band and another at control band indicated a positive result. Negative test results showed only control band. HpSA test is a non-invasive, accurate test especially useful for screening of asymptomatic subjects. Pallor was seen in the lower palpebral conjunctiva.

**Statistical Analysis**

Data were analysed by the chi-square test to compare the association between different variables and positive Helicobacter pylori rates. A value of $P < 0.05$ was considered statistically significant. Calculations were done using the software package SPSS 16.0.

**Results**

Out of total 208 patients, 92 patients were Helicobacter pylori positive by Immunocard STAT HpSA test, giving a hospital based prevalence of 44.23% (Table 1). Out of total 143 males, 61 were positive for Helicobacter pylori (42.7%) whereas out of 65 females 31 were positive (47.7%) (Table 2). The prevalence was estimated in different age groups. The maximum number of positive patients was found in the age group of 30-39 years (50.7%) and the minimum

<table>
<thead>
<tr>
<th>Table 1. Prevalence of Helicobacter pylori</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total subjects</td>
</tr>
<tr>
<td>Subjects positive for Helicobacter pylori</td>
</tr>
</tbody>
</table>

Prevalence of H. pylori = 92/208 X 100 =44.23 %
prevalence was in the age group of above 70 years (20%) (Table 3). For socio economic status, the groups were classified according to modified Kuppuswamy scale for urban families. Out of 118 patients belonging to upper lower socioeconomic group, 53 were positive for *Helicobacter pylori* (44.9%), out of 78 of lower middle group, 33 were positive (42.3%) and out of 12 of upper middle vegetarians with 43 positive (37.1%) (Table 5). Pallor was present in 98 patients with 51 positive (52.0 %) and absent in 110 patients with 41 positive for *H. pylori* (37.3%) (Table 6). Out of 58 tobacco-chewing subjects, 30 were positive for *Helicobacter pylori*, which indicated a prevalence of 51.7%. (Table 7).

**Discussion**

The prevalence of *Helicobacter pylori* infection varies worldwide, but higher colonization rates have been seen in

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**Table 2. Number of *Helicobacter pylori* positive patients according to sex**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Total subjects</th>
<th>Subjects positive for <em>Helicobacter pylori</em></th>
<th>Percentage %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>143</td>
<td>61</td>
<td>42.7%</td>
</tr>
<tr>
<td>Female</td>
<td>65</td>
<td>31</td>
<td>47.7%</td>
</tr>
</tbody>
</table>

**Table 3. Number of *Helicobacter pylori* positive patients according to age group**

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Total subjects</th>
<th>No. of positive patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>19-29</td>
<td>70</td>
<td>31</td>
<td>44.3%</td>
</tr>
<tr>
<td>30-39</td>
<td>69</td>
<td>35</td>
<td>50.7%</td>
</tr>
<tr>
<td>40-49</td>
<td>37</td>
<td>17</td>
<td>45.9%</td>
</tr>
<tr>
<td>50-59</td>
<td>18</td>
<td>5</td>
<td>27.8%</td>
</tr>
<tr>
<td>60-69</td>
<td>9</td>
<td>3</td>
<td>33.3%</td>
</tr>
<tr>
<td>&gt;70</td>
<td>5</td>
<td>1</td>
<td>20%</td>
</tr>
</tbody>
</table>

**Table 4. Number of *Helicobacter pylori* positive patients according to socio economic status**

<table>
<thead>
<tr>
<th>SE status</th>
<th>Total subjects</th>
<th>No. of positive patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper lower</td>
<td>118</td>
<td>53</td>
<td>44.9%</td>
</tr>
<tr>
<td>Lower middle</td>
<td>78</td>
<td>33</td>
<td>42.3%</td>
</tr>
<tr>
<td>Upper middle</td>
<td>12</td>
<td>6</td>
<td>50%</td>
</tr>
</tbody>
</table>

**Table 5. Number of *Helicobacter pylori* positive patients according to diet**

<table>
<thead>
<tr>
<th>Type of diet</th>
<th>Total subjects</th>
<th>No. of positive patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetarian</td>
<td>116</td>
<td>43</td>
<td>37.1%</td>
</tr>
<tr>
<td>Non- Vegetarian</td>
<td>92</td>
<td>49</td>
<td>53.3%</td>
</tr>
</tbody>
</table>

**Table 6. Number of *Helicobacter pylori* positive patients according to pallor**

<table>
<thead>
<tr>
<th>Pallor</th>
<th>Total subjects</th>
<th>No. of positive patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>98</td>
<td>51</td>
<td>52.0%</td>
</tr>
<tr>
<td>Absent</td>
<td>110</td>
<td>41</td>
<td>37.3%</td>
</tr>
</tbody>
</table>

**Table 7. Number of *Helicobacter pylori* positive patients according to tobacco chewing**

<table>
<thead>
<tr>
<th>Tobacco</th>
<th>Total subjects</th>
<th>No. of positive patients</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chewer</td>
<td>58</td>
<td>30</td>
<td>51.7%</td>
</tr>
<tr>
<td>Non-chewer</td>
<td>150</td>
<td>62</td>
<td>41.3%</td>
</tr>
</tbody>
</table>
This study was conducted to find out the prevalence of H. pylori among patients attending OPD for symptoms other than gastrointestinal disorders. These patients were screened for Helicobacter pylori by Immunocord STAT HpSA test. In a study from Chandigarh, 254 individuals were screened for Helicobacter pylori which was positive in 56.7% asymptomatic individuals [37]. The overall prevalence recorded in our study was 44.23%, which is less in comparison to the above study. This can be explained by the fact that prevalence of H. pylori varies widely by geographic area, age, race, and ethnicity and SE status. In another study, Helicobacter pylori prevalence was seen in patients with dyspepsia and in control subjects to be 65% and 46% respectively [38]. In a study conducted on healthy Omani blood donors, the overall prevalence for Helicobacter pylori was 69.5% [39]. Similar results were shown in a study from Turkey where 53% asymptomatic subjects were seropositive for Helicobacter pylori antibodies [40], whereas 51% asymptomatic subjects were seropositive in a study from Saudi Arabia [41]. In the present study, among Helicobacter pylori positive patients 42.7% were males and 47.7% were females. Although there is a slightly greater female preponderance but the difference between the genders was not significant which goes in accordance with a similar study from South India [42] and from other parts of the world [40, 43, 44]. However, in a study from Oman [39], Helicobacter pylori seropositivity has shown increasing tendency with age in females (55-65%) as compared to males of the same age group (35-55%). In another study, attention was given to gender differences indicating that prevalence of Helicobacter pylori infection was higher in men with upper and non-upper digestive tract symptoms than that in women [45]. In our study, age wise distribution showed maximum prevalence in the age group of 30-39 years (50.7%) and minimum in the age group of more than 70 years (20%). This goes in accordance with a similar Indian study in which the maximum prevalence was in the age group of 36-45 years (43.47%) and minimum in the age group of 66-75 years (3.26%) [46]. An early study from Saudi Arabia found an increase in seroprevalence of Helicobacter pylori with advancing age reaching to 70% for those who were 20 years old or more [47]. An increase in prevalence with age being maximum (74%) between 16-30 years and thereafter showing a slight decline has been reported in another Indian study[42]. In a study from Mumbai, age related prevalence of Helicobacter pylori showed similar results as ours with maximum prevalence of 58% in the age group of 30-39 years [38].

In the present study, out of 98 patients having pallor 51 patients were positive for H. pylori (55.4%) (p>0.05). Helicobacter pylori colonization appears to impair iron uptake and increase iron loss. Regarding the possible role of Helicobacter pylori in iron deficiency anaemia, a recent meta-analysis indicated that the infection is associated with depleted iron deposits. The mechanism by which Helicobacter pylori induces this alteration is not clear but it appears to involve GI blood loss, diminished iron absorption from diet and increased consumption of iron by the bacteria [48]. A study with Helicobacter pylori infected patients from Bangladesh has shown the prevalence of iron deficiency anaemia with a decrease in haemoglobin while serum ferritin was significantly higher in [49]. The prevalence of Helicobacter pylori in our study was found to be higher in low socioeconomic group being 57.6% in upper lower and 35.9% in lower middle groups. This is consistent with previous studies which have demonstrated that the prevalence of Helicobacter pylori as well as gastritis is more frequent in those who come from large families, have poor hygiene, low standards of living, poor sanitation practices and over crowded living conditions [50,51,52]. Socioeconomic status is not restricted to income and social class but also considers other factors such as living standards, urbanization and educational level [53]. Similar result was seen in a study among professional workers of Kashmir valley [54]. A prevalence of 46.7% was seen in vegetarians and 53.3% in non-vegetarian group, which was though higher in non-vegetarians but was not significant (p>0.05). This supports the fact that it is probably the food prepared under unhygienic conditions that plays a role in transmission of Helicobacter pylori in developing countries and not the type of food consumed [55]. Similarly, the prevalence of Helicobacter pylori among tobacco chewers was higher (51.7%) than among non-tobacco chewers (41.3%) but was not significant (p>0.05). Both non vegetarian diet and tobacco chewing have been studied as risk factors for peptic ulcer and gastric cancer among Helicobacter pylori infected individuals [56-58] but still more studies are required to establish a association between them.

**Conclusion**

The present study revealed substantial prevalence of Helicobacter pylori in asymptomatic patients with females being more affected than males and a maximum prevalence in the age group of 30-39 years. The prevalence is higher in low socioeconomic classes with poor sanitation practices and unhygienic water supply. A higher prevalence of Helicobacter pylori seen in subjects having pallor may be contributed to poor iron absorption. Similarly, a higher prevalence of Helicobacter pylori was noticed among non-vegetarians and tobacco consumers, which may be the contributing factors in the development of peptic ulcer and gastric cancer in patients harboring Helicobacter pylori. Identification of populations, who do not show symptoms of Helicobacter pylori infection, but still harbour it, is essential for controlling the infection and it still remains a challenge for the clinicians.

**Acknowledgement**

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References


Correspondence to:
Shraddha Singh
Department of Physiology
King George’s Medical University
Lucknow 226003, Uttar Pradesh
India.