HELICOBACTER PYLORI SEROLOGY AND EVALUATION OF GASTRODUODENAL DISEASE IN NIGERIANS WITH DYSPEPSIA

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Helicobacter pylori (H. pylori) has been strongly associated with various gastroduodenal diseases worldwide with only a few studies emanating from developing countries. The objectives of this study were to determine the prevalence of serum immunoglobulin G (IgG) and underlying gastroduodenal pathology in Nigerian patients with dyspepsia and ascertain the usefulness of H. pylori IgG screening in decreasing endoscopic workload in dyspeptics in Nigeria. Fifty-five patients with dyspepsia and 55 age and sex-matched apparently normal control were screened for H. pylori IgG using Immunocomb® kits. Each of the 55 patients was also examined endoscopically with biopsies taken appropriately. Serology was positive in 94.5% and 92.7% of dyspeptic patients and controls respectively. Gastroduodenal inflammation was the commonest endoscopic finding, 43 (78.18%). Other findings were malignant gastric tumour 6(10.9%), reflux oesophagitis 3 (5.4%), gastric ulcer 2 (3.64%), and duodenal ulcer in 1 (1.82%). Chronic gastritis was the main histopathologic finding in the dyspeptic patients. It is concluded that serum H. pylori IgG cannot be used as a screening procedure to reduce endoscopic workload in Nigerian patients with dyspepsia.

Keywords: dyspepsia; Helicobacter pylori serology; gastroduodenal disease

INTRODUCTION

Dyspepsia, which has been defined as pain or discomfort centered in the upper abdomen (1,2), is a common gastrointestinal complaint in Nigeria (3). In Ibadan, Nigeria, over 50% of dyspeptic patients have been shown to have non-ulcer dyspepsia (4). Since the landmark discovery by Barry and Marshal of the association between Helicobacter pylori and gastritis in 1982 (5), several studies have confirmed (6,7) or doubted its association with gastroduodenal disease (8) and yet some suggested its association with non-gastrointestinal disease (9). Serology for H. pylori infection has been found to be accurate, rapid, cost-effective (10,11) and capable of significantly decreasing endoscopic workload in
patients with dyspepsia (12). We investigated a total of 110 subjects at the University College Hospital Ibadan, Nigeria to determine the prevalence of H. pylori IgG serology and underlying gastroduodenal disease in patients with dyspepsia and normal controls.

MATERIAL AND METHODS

One hundred and ten adult subjects of both sexes, aged between 18 to 74 years consisting of 55 patients with dyspepsia (34 males; 21 females) with no previous treatment for H. pylori and 55 apparently normal control with no previous or present history of dyspeptic symptoms (33 males; 21 females) gave informed consent to participate in the study. Dyspeptic patients were consecutively selected from the pool of patients attending our Gastroenterology clinic, based on consent and fitness to undergo eosophaagogastroduodenoscopy (OGD). The control groups were apparently normal individuals selected from office workers and individuals attending the hospital for routine check ups and hypertension. The control group did not undergo OGD screening. Patients and control were aged between 18 and 74 years and the study period lasted for eighteen months. Excluded from the study were patients who have been on non-steroidal anti-inflammatory drugs (NSAIDs) in the last 3 months, chronic liver disease (all the dyspeptic patients were negative for hepatitis B and human immunodeficiency viruses 1 and 11) as well as pregnant women.

Five milliliters of venous blood was collected from each subject, into unheparinised bottles. The serum was separated after centrifugation and frozen immediately at -20°C till time of analyses after the study period. Sera were analyzed for H. pylori IgG using Immunocomb™ (Manufacturer- ORGENICS, Yvane, Israel. Website: http://www.orgenics.com) with sensitivity and specificity of 95.8% and 76% respectively. Upper gastrointestinal endoscopy was carried out in all the 55 patients with multiple biopsies taken from the gastric antrum, duodenum, upper and lower margins of cancerous lesions and other suspicious sites in the upper gastrointestinal tract. The control subjects did not undergo eosophaagogastroduodenoscopy. Tissue specimens were promptly
fixed in 10% formalin and subsequently processed in the histopathology laboratory where paraffin sections were stained with routine haematoxylin and eosin stain for histological examination.

RESULT

Serology

Fifty-two (94.5%) and 51 (92.7%) of the dyspeptic patients and normal controls were seropositive for H. pylori IgG receptively. Seropositivity was similar in both males and females. All the patients with gastric carcinoma were positive for H. pylori IgG antibody.

Endoscopy

Endoscopic findings were mainly gastroduodenitis 32(58.18%), chronic gastritis 9 (16.36), and chronic duodenitis 2 (3.6%) Table 1. Atrophic gastritis was observed in 11 patients with gastroduodenal inflammation. Six patients aged 35 to 67 years had gastric tumours (5 antral; 1 cardial) and consisted of 4 males and 1 female. The only female patient among them was 35 years old while the men were between 46 and 67 years with a mean age of 58 years. Reflux oesophagitis was found in 2 of the patients with gastric cancer.

Submucosal hemorrhage was found in 5 of the patients with gastritis. Three patients had oesophagitis while 12 (28%) of the 43 patients with gastroduodenal inflammation also had bile reflux.

Histopathology

Only 45 (18.8%) of the dyspeptic patients eventually had histopathologic assessment performed on the biopsy specimens (other specimens were lost in transit). Of these, 36 patients had gastric tissue present while 27 patients had duodenal tissue included in the biopsy specimen for assessment. Following histological assessment, there were 5 cases of gastric adenocarcinoma. Thirty-four of 36 (94.4%) gastric biopsies assessed had histological evidence of varying grades of chronic gastritis and one case of normal gastric mucosa was recorded. All duodenal biopsies examined show histological evidence of chronic, duodenitis, but none had H. pylori on histology. H. pylori colonization was observed at histology in 14 (41.2%) of the 34 cases of chronic gastritis, while one of the five cases of gastric adenocarcinoma showed H. pylori colonization in the background gastric tissue.
Table 1: Endoscopic features in patients with dyspepsia

<table>
<thead>
<tr>
<th>Endoscopic features</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastroduodenitis</td>
<td>32</td>
<td>58.18</td>
</tr>
<tr>
<td>Chronic gastritis</td>
<td>9</td>
<td>16.36</td>
</tr>
<tr>
<td>Gastric tumour</td>
<td>6</td>
<td>10.9</td>
</tr>
<tr>
<td>Chronic oesophagitis</td>
<td>3</td>
<td>5.45</td>
</tr>
<tr>
<td>Chronic duodenitis</td>
<td>2</td>
<td>3.64</td>
</tr>
<tr>
<td>Gastric ulcer</td>
<td>2</td>
<td>3.64</td>
</tr>
<tr>
<td>Duodenal ulcer</td>
<td>1</td>
<td>1.82</td>
</tr>
</tbody>
</table>

DISCUSSION

*Helicobacter pylori* has been shown by various studies to be causally linked with various gastro-duodenal disease (5,13,14,15). Most of these studies were done in developed countries of the world, with only a few studies emanating from developing countries. This cross-sectional study has shown a high seroprevalence of *H. pylori* both in dyspeptic patients and apparently normal control with no current or previous symptoms of dyspepsia. Previous studies in Nigeria and other parts of Africa have shown a similar high prevalence of *H. pylori* infection in normal and dyspeptic population (16,17,18). These findings further strengthen the multifactorial concept of aetiopathogenesis of dyspepsia as well as the fact that in a developing country like Nigeria where there is no significant difference in the infection in dyspeptic and normal populations, *H. pylori* may well be an innocent bystander or an opportunist that cashes in when other factors have rendered the mucosa susceptible to damage. Also, the well known fact that many people infected with *H. pylori* never show symptoms of disease (19) and finding of a similar prevalence of *H. pylori* infection in geographical area where incidence of dyspepsia is high in one and low in the other (20) tend to whittle down the significance of *H. pylori* infection in the pathogenesis of
acid peptic disease in these regions. Recently, it was shown that there has been an increase in peptic ulcer disease unrelated to *H. pylori* infection in developed countries (21). This finding also attests to polycausality of peptic ulcer disease. From the foregoing the magnitude of contribution of *H. pylori* to dyspepsia in the studied group is difficult to determine, as prevalence of infection in asymptomatic controls was not significantly different from those who had dyspepsia. The variation in host and microbial factors, which are known to determine the development of disease as well as the multifactorial nature of dyspepsia, may account for this. The extent of contribution of *H. pylori* in the population needs to be determined by further studies.

Dyspeptic patients in Nigeria could be safely presumed to be *H. pylori* positive until otherwise proven, as 94.5% of the dyspeptic patients were sero-positive. This prevalence in however slightly higher than 85% documented by Malu et al in blood donors in Jos, Northern Nigeria using a similar method. Endoscopic finding of mainly gastroduodenal inflammation in our study has been a typical finding among Nigerian patients with dyspepsia with incidence of frank ulceration much lower than findings in developed countries of the world (4, 22,23,24). This is most likely due to rampant abuse of over-the-counter drugs which in Nigeria include antibiotics, antacids and antisecretory drugs, which patients take without prescription and only presenting for hospital care if there is no improvement or with incomplete resolution of their symptoms. This abuse of therapy may cause some degree of ulcer healing before endoscopy is carried out and may account for the low incidence of ulcer recorded in these studies. Some of the patients with dyspepsia and gastroduodenal inflammation were found in addition to have duodenogastric reflux (bile reflux) but the degree of contribution of this to the dyspeptic pain is difficult to quantify. Endoscopic oesophagitis seen in 3 (5.5%) patients, 2 of whom had reflux in addition, suggest that gastric acid reflux may be contributory to dyspeptic pain in our patients. None of them, however, presented with classical symptoms of gastrooesophageal reflux disease (GORD)
before endoscopy. Six (10.9%) of the patients were found to have gastric carcinoma (5 antral; 1 cardiac) with age range 35 to 67 years. All the patients with gastric adenocarcinoma were positive for H. pylori serology but only one was positive on histology. The histological finding of H. pylori in 41.2% of the gastric tissue of patients with chronic gastritis is lower than IgG in the serum. This difference may be attributable to the patchy pattern of H. pylori colonization in gastric mucosa. Also previous studies have shown that identification of H. pylori is less sensitive when routine elementary and eosin preparation alone is applied [as was the case in this study] than when it is combined with special techniques such as Giemsa or Steiner staining (25). It is instructive that one of the patients who had a normal gastric histology was also negative for H. pylori IgG. This we consider as a case of non-ulcer dyspepsia type II.

CONCLUSION

It is concluded that H. pylori seropositivity is similarly high among both normal people and dyspeptic patients in the study group and therefore the test is not discriminatory between the two groups and as such cannot be used to confirm dyspepsia. Also, gastroduodenal inflammation remain the commonest cause of dyspepsia in Nigeria and Helicobacter pylori is commonly associated with chronic gastritis in Nigeria as is the case in the rest of the world.

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REFERENCE


dosc. 2001; 52: 20-26


