Helicobacter Pylori, Association with Upper Gastrointestinal Disease in Bahrain

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ABSTRACT

One hundred and fifty consecutive patients with either mild or severe dyspepsia, were investigated for the presence of Helicobacter Pylori (HP). HP were found in 51% of the patients in the gastric antrum and 26% in the duodenum. Antral HP occurred significantly more often in patients with superficial active gastritis compared to those with no evidence of inflammatory changes (P<0.01), strongly suggesting its role in the pathogenesis of upper gastrointestinal diseases in our patients.

There has been worldwide interest in HP and its role in gastroduodenitis, since Warren & Marshal's publication in 1983. Accumulating evidence suggests that HP is the aetiological agent responsible for gastritis[9,12,15] and also the cause of dyspepsia although still disputed[2,17]. Part of the interest has also been based on the probability that HP may be causally related to peptic ulcer disease[21].

The epidemiological pattern and the nature of HP infection of upper gastrointestinal disease has shown differences in western and developing countries[1,5,18,23]. The route of entry and the method of transmission are not fully understood although there is a report from Chili implicating uncooked vegetables[14] which is supported by a study that isolated HP from faces[13,22].

Gastritis and peptic ulcer diseases have been recognised to be common in Bahrain, but there is no reliable epidemiological data to support the hypothesis except health statistics surveys of the Ministry of Health Bahrain in 1991[20]. We undertook this prospective study to determine the prevalence of HP among dyspeptic population and to investigate the possible role of HP in gastritis and peptic ulcer.

METHODS

Patients

The study was conducted on 150 consecutive patients with upper gastrointestinal symptoms requiring endoscopy and attended Salmaniya Medical Centre during the period between August 1990 to December 1992.

Endoscopy

Endoscopy was performed using Olympus Type XQ20GIF Endoscope. Informed consent had been obtained. The biopsies were taken systematically from the gastric antrum and duodenal bulb. Two adjacent areas were sampled from each part, one placed in brucella broth for microbiological techniques, and the other in 10% formal saline for histology.

Microbiology

Specimens were processed immediately or kept at 4°C for no longer than 4 hours and were inoculated on to

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blood agar. A smear for Gram stain was also made. The plates were incubated at 37°C micro-aerophilically for up to six days. HP was identified by its typical colony and gram staining appearance, positive oxidase and urease tests.

Histology

The biopsies were routinely processed and stained by Haematoxylin and Eosin. The duodenal biopsies were stained by Period Acid Schiff (PAS) reagent to detect focal gastric metaplasia. Light microscopy was performed without prior knowledge of microbiological results. HP was searched for in all specimens. Pathological findings were assessed and histological grading of inflammation was carried out using the classification of Whitehead et al[25].

Analytical Procedures

Specimens were considered positive for HP if the organism was identified by culture. Statistical comparisons were performed using X^2 test.

RESULTS

150 antral and 123 duodenal biopsies were obtained from 150 patients. From 27 patients duodenal biopsy was not taken because the duodenum appeared normal on endoscopy. There were 104 men and 46 women. The mean age was 39.2 years old and ranged between 14 to 80 years old. HP was diagnosed in 77 (51%) of antral and 34 (28%) of duodenal biopsies.

118 patients (79%) showed some form of gastritis in the antral region, ulcer or lymphoma on endoscopy. The remaining 32 (21%) were normal on endoscopy. Only 3 (9.4%) out of these 32 were positive for HP as compared with 74 (62%) out of the 118 endoscopically proven gastritis, ulcer patients, a statistically highly significant difference (X^2 = 25.7, P<0.001).

HP infection and relation to age

The maximum rate of infection was found among the age group of 40 to 49 years old and the lowest was among the 50 years old and over. There was a significant association between HP infection and age of patients (Table 1).

Table 1
Age distribution and the presence of H.pylori in the antrum

Age in years	*HP positive*		*HP negative*		*Total*	
	No	*%*	*No*	*%*	*No*	*%*
< 30	19	54.5	16	45.7	35	23.3
30-39	28	51.9	26	48.1	54	36.0
40-49	23	63.9	13	36.9	36	24.0
> 50	7	28.0	18	72.0	25	16.7
Total	77	51.3	73	48.7	150	100.0

HP infection and the antral inflammatory changes

Of the 54 patients with active superficial gastritis 40 (74%) had HP. It was also found in 18 (64%) out of 28 patients with inactive superficial gastritis. The number of other forms of inflammatory changes were too small to be analysed. Nevertheless, 3 of 5 patients with gastric lymphoma had HP infection (Table 2).

HP and inflammatory changes in the duodenum

Of the 11 patients with active duodenitis, 7 (64%) had HP. It was also found in 8 (61%) of the 13 patients with duodenal ulcer. In the remaining 76 patients with normal endoscopy, only 13 (17%) had HP. If taken together the prevalence of HP in the active duodenitis and duodenal ulcer groups (15 out of 24) would be significantly

Table 2
Inflammatory changes and presence of H.pylori in the antrum

Pathological changes	*No of patients*	*HP positive*	
		No	*%*
Active superficial gastritis	54	40	74
Inactive superficial gastritis	28	18	64
Mild inflammation	16	6	37
Active erosive gastritis	7	5	71
Lymphoma	5	3	60
Ulcer	8	25	40
Normal	32	3	10
Total	150	77	51

Table 3
Inflammatory changes and presence of H.pylori in the duodenum

Inflammatory changes	*No of patients*	*Positive*	
		No	*%*
Active duodenitis	11	7	64
Duodenal ulcer	13	8	61
Mild inflammation	23	6	26
Normal	76	13	17
Total	123	34	28

higher ($X^2 = 19.3$, $P<0.001$) than in patients with normal duodenum on endoscopy (Table 3). Gastric metaplasia was not seen in duodenal biopsies examined.

DISCUSSION

HP infection is prevalent worldwide, but knowledge of it's epidemiology is still not clear. Many early reports associated HP to symptomatic patients presenting for endoscopy. During the last ten years, HP has been rapidly recognised as an important aetiological factor in peptic ulcer disease, gastritis, and non-ulcer dyspepsia, and has also been found in healthy carriers[3].

The methods available for the detection of HP infection are microbiological culture, histology and quick urease tests. These methods are all dependent on endoscopy and biopsy, which are the methods used in this study. Alternatively HP infection can be detected non-invasively with urease breath test and with serology[19].

This study shows that 51% of a representative smaple of 150 patients with upper gastrointestinal symptoms were positive for HP infection, 123 patients had both antral and duodenal biopsies and 27 had only an antral biopsy. Of these, 32 antral biopsies and 76 duodenal biopsies were normal. The prevalence of HP infection was statistically significantly being higher in both the antral and duodenal biopsies. These results conform with the general suggestions in the literature that HP infection has an aetiological role in upper gastrointestinal diseases and in particular, in patients with duodenal ulcer. Moreover, HP infection may play a role in the pathogenesis of non-ulcer dyspepsia. Therefore classifying the subjects into HP infection positive and negative may be useful to highlight the heterogeneity of subjects with this disease[16].

The frequency of HP infection was 54% in young adult patient and increased to 64% in the age group between 40 to 49 years old.

Although there was a preponderance of males over females among the population of the present study, the percentage of infection with HP showed no sex related difference. This is in accordance with other studies[5,10]. Furthermore, there is no significant correlation between the presence of HP and the endoscopic findings of gastritis confirming that endoscopy alone is not the best tool for the diagnosis of HP infection.

This study also suggest the possible association of HP infection in the causation of gastritis. However, the relationship with peptic ulceration is more complex and controversial, yet evidence is now accumulating to suggest that it is responsible for ulcer relapse although its concomitant presence does not prevent ulcer healing. Controversy regarding the role of HP in the pathogenesis of peptic ulcer disease still continues and its exact role remains uncertain. Some authors, however have suggested that colonisation of gastric mucosa by HP is associated with antral gastritis, peptic ulcer disease and non-ulcer dyspepsia[15]. One study from Norway concluded that HP seems to have a pathogenetic role in gastritis and may be a contributing factor but not a cause of peptic ulcer[4].

In one of the largest studies, HP infection was proved by positive culture in 19.4% of patients, whereas serology was positive in 33.7%[8]. Healthy volunteers who were tested with urease test showed 45% positive results[11]. It has been suggested in this respect that serology might replace endoscopy in the diagnosis of HP associated gastritis[13].

However the excellent sensitivity (97%) and specificity (100%) of the non-invasive urease breath test and it's simplicity makes it an ideal method for screening people for HP colonisation[7].

CONCLUSION

This is the first study to describe the prevalencve and the pattern of HP associated upper gastrointestinal tract diseases in Bahrain. Future studies are needed especially for eradication trials. The limited practice of triple therapy (ie. Bismath – metronidazole – amoxicillin) needs further investigation and evaluation. Preliminary results from an on-going sensitivity

study has shown that most strains of HP are resistant to metronidazole. Therefore in our part of the world treatment using metronidazole in the triple therapy may not be very effective. Other drugs such as Rifampcin could be used instead. With the new development in understanding the pathogenesis of gastritis and the emergence of HP as a cause and contributing factor to peptic ulcer, the diagnosis and the management have to be reassessed.

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