

Helicobacter pylori: a Jordanian study

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Summary: The prevalence of *Helicobacter pylori* in patients with upper gastrointestinal symptoms in the north of Jordan was studied prospectively. The occurrence of *H. pylori* was documented histologically and bacteriologically in 169 patients attending endoscopy for upper gastrointestinal symptoms. Our results showed that *H. pylori* was present in 70% of patients with acute gastritis, 73% of patients with chronic gastritis, 68% of patients with acute on chronic gastritis, 83% of patients with duodenal ulceration, 75% of the patients with gastric ulceration, 64% of patients with no pathology, and 68% of patients regardless of the pathology found. There was a sharp rise in the prevalence of *H. pylori* with age, up to the age of 40 years with an annual increase in the prevalence of 2%. This study shows that the prevalence of *H. pylori* in Jordan is similar to that seen in other developing countries with infections occurring at a lower age and with the annual infection rate being double that seen in developed countries.

Introduction

The presence of spiral bacteria in animal stomachs was reported by Bizzozero¹ in 1893. The presence of these organisms in human stomachs was confirmed by Krientz² in 1906. In 1938 Doenges³ found these organisms to be present in 43% of 242 human autopsy specimens of the stomach. They have also been detected in gastric mucosa in Meckel's diverticulum.⁴

These organisms became known as campylobacter-like organisms (CLO) because of their similarity to other members of the genus following the work of Marshall and Warren^{5,6} in 1983–4. Later the name *Campylobacter pyloridis* was used and shortly afterwards was changed to *Campylobacter pylori*. More recently the name *Helicobacter pylori* (HP) has been suggested⁷ due to the findings of fundamental structural differences between this organism and members of the *Campylobacter* genus.

HP is a spiral non-sporulating Gram-negative bacteria with flagellae at one end. It has the capability to split urea due to its ability to produce an urease enzyme.⁸ This facilitates its detection using rapid urease tests.^{9,10}

HP is rapidly gaining recognition as an important aetiological factor in peptic ulcer disease,^{5,11} gastritis^{5,12} and non-ulcer dyspepsia.¹³ However, it has also been detected in volunteers with no gastrointestinal symptoms,^{8,14} especially in association with asymptomatic gastritis¹⁵ and has even

been detected in children¹⁶ although its prevalence seems to increase with age.^{15,17}

No studies have been published concerning the prevalence of HP in patients with gastrointestinal symptoms in a Jordanian population. The aim of our study was to address this question.

Patients and method

This was a prospective study of 182 consecutive endoscopies on 169 patients complaining of upper gastrointestinal symptoms referred to the endoscopy unit of the Jordan University of Science and Technology. Of these, 88 were males and 81 were female. The age range was 17 to 86 years with a mean of 42.1 years (s.d. 13.9).

Endoscopic biopsies from within 5 cm of the pylorus were obtained for histological (10% formalin solution) and bacteriological examination. The histological specimens were processed using silver and haematoxylin and eosin staining and examined for the presence of HP and acute or chronic gastritis. Bacteriology specimens were transported in sterile vials containing 3 ml brain-heart infusion. Following mincing, the specimen was cultured in a blood agar plate at 37°C in anaerobic conditions for up to 10 days. The plates were then examined and the presence of HP determined by Gram staining, catalase production, oxidase production and urease production. The patient was considered to have HP if either the bacteriology or the histology specimens confirmed the presence of the organism. Occasionally patients were positive to one but not the other of the

investigations. This can be explained by the fact that there is often patchy distribution of the organism in the stomach¹⁸ and the site of the gastric biopsies for bacteriology and for histology were not always from the exact same location.

Results

Table I shows the breakdown of patients in respect to the pathology found and the rate of isolation of HP in each group. Table II shows the prevalence of HP and gastritis in patients in whom no visual abnormalities were detected on endoscopy.

HP was present in 70% of patients with acute gastritis, 73% of patients with chronic gastritis, 68% of patients with acute on chronic gastritis, 83% of patients with duodenal ulceration, 75% of the patients with gastric ulceration, 64% of patients with no pathology, and 68% of patients regardless of the pathology found. There were no significant differences in the prevalence of HP between the different pathologies found or between males and females.

Figure 1 shows the percentage of patients positive to HP against age group in increments of 5 years. It appears that the relationship between infection with HP and age is linear till the age of 40. A linear regression line fits these data with a coefficient of correlation of 0.99. This is significant at the 0.1% level and gives an increased prevalence of infection with HP of 2% for every year up to this age. After the age of 40, the increase in infection rate appears to flatten out.

Discussion

Neither the route of entry of *H. pylori* nor its pathogenicity in man has yet been fully established. One hypothesis suggests transmission from an exogenous source, and swine have been proposed due to similarities between human and swine HP.¹⁹ However, the high prevalences of HP found in patients from predominantly Moslem communities,^{20,21} including our own, would argue against this concept.

In developed countries, HP infection is rare in childhood but its prevalence appears to increase with age.^{15,17} Between the ages of 20–60, approximately 50% of the population become seropositive for HP. This increase occurs at a relatively linear rate with an annual increase in seropositivity of approximately 1%.¹⁹ In developing countries the situation is different. Infection seems to occur at a much younger age. In a study from Peru,²² over half of the children below the age of 5 years were already positive for HP, with children from poorer communities acquiring the infection earlier (first 12–18 months of life). In a study comparing the prevalence of HP in children under 10 years of age, in different populations,²⁰ 45.2% of Algerian children, 55.2% of children from Ivory Coast, and 13.1% of Vietnamese children were sero-positive to HP in comparison to 3.5% of French children. The prevalence increased up to the age of 50 reaching levels of 80–90% in the developing countries compared to 35% in France. In a study from Saudi Arabia,²¹ the prevalence of

Table I The prevalence of *Helicobacter pylori* (HP) in males and females categorized according to diagnosis on endoscopy and histology

	Males		Females		Total	
	HP Pos (%)	HP Neg (%)	HP Pos (%)	HP Neg (%)	HP Pos (%)	HP Neg (%)
Chronic gastritis	36 (75%)	12 (25%)	26 (70%)	11 (30%)	62 (73%)	23 (27%)
Acute gastritis	17 (68%)	8 (32%)	18 (72%)	7 (28%)	35 (70%)	15 (30%)
Acute on chronic gastritis	9 (53%)	8 (47%)	14 (82%)	3 (18%)	23 (68%)	11 (32%)
Gastric ulceration	2 (67%)	1 (33%)	1 (100%)	0 (0%)	3 (75%)	1 (25%)
Duodenal ulceration	20 (83%)	4 (17%)	10 (83%)	2 (17%)	30 (83%)	6 (17%)
Non-ulcer dyspepsia	6 (75%)	2 (25%)	3 (50%)	3 (50%)	9 (64%)	5 (36%)

Table II The prevalence of *Helicobacter pylori* (HP) in males and females categorized according to histology in patients with no visual findings on endoscopy

	Males		Females		Total	
	HP Pos (%)	HP Neg (%)	HP Pos (%)	HP Neg (%)	HP Pos (%)	HP Neg (%)
Chronic gastritis	3 (60%)	2 (40%)	3 (38%)	5 (62%)	6 (46%)	7 (54%)
Acute gastritis	0 (0%)	0 (0%)	2 (100%)	0 (0%)	2 (100%)	0 (0%)
Acute on chronic gastritis	8 (67%)	4 (33%)	12 (80%)	3 (20%)	20 (74%)	7 (26%)
No gastritis (N.U.D.)	6 (75%)	2 (25%)	3 (50%)	3 (50%)	9 (64%)	5 (36%)

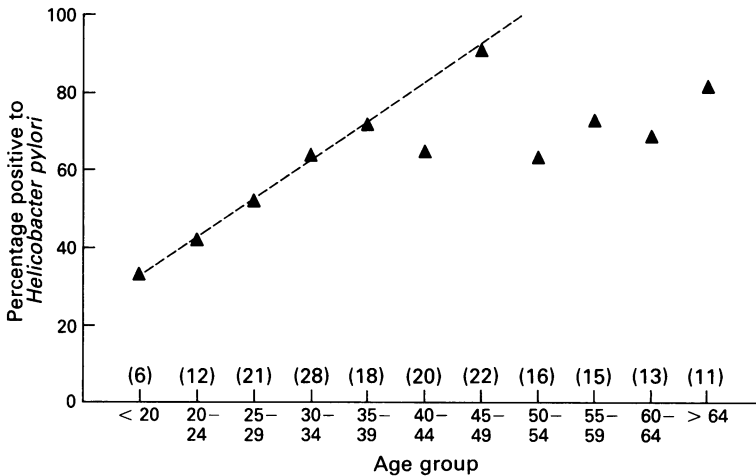


Figure 1 The percentage of patients found to be positive for *H. pylori* plotted against age group. The broken line represents a linear regression line drawn through the first five points and has a correlation coefficient of 0.99. The numbers in parentheses indicate the number of patients in each group.

HP infection in the first decade was 40% increasing rapidly to over 70% in those above the age of 20. The rate of increase was about 2.3% per annum. This is similar to our data which show an increase per annum of 2% up to the age of 40. There were no children in our study so it was not possible to determine the prevalence of HP infection in this group. However, it is possible to postulate that the prevalence of HP in children in our population is lower than that seen in the Saudi study since the prevalence of HP in our young adult population was lower (46% of patients aged 21–30 in our population compared to about 75% in the Saudi Arabian population). We found no significant sex differences in the prevalence of HP and this is a similar finding to other studies.^{19,21}

As well as age related differences, there are geographical and racial variations in the prevalence of HP. Generally, the prevalence in developed countries seems to be lower. However, several geographic and ethnic groups in developing countries have been shown to have a very low prevalence of HP infection. This includes Northern Australian Aborigines and Vietnamese populations.²³ Certainly in the Aborigine group, this correlates well with the lack of duodenal ulcer disease. Socioeconomic factors also appear to be important in this infection with a suggestion of a higher prevalence in the lower socioeconomic groups.^{21,22} Our population was a relatively homogeneous one and such comparisons could not be made.

Our findings of 64% of patients with normal gastric mucosa, 70% of patients with acute gastritis and 68% of patients with chronic gastritis being positive for HP is in accord with studies performed

in other countries.^{24–29} The finding of 75% of the patients with gastric ulceration and 83% of patients with duodenal ulceration being positive for HP is also similar to findings elsewhere.^{30–32}

Colin-Jones³³ has recently reviewed the role of HP in upper gastrointestinal disease. In duodenal ulcer disease it has been postulated that HP and endogenous acid production by gastric metaplasia of the duodenum play a synergistic role in the pathophysiology of ulceration³⁴ and there is little doubt that duodenal ulcers tend to recur more readily if this organism is not eradicated during treatment.³⁵ It is therefore desirable to ensure eradication of this organism in sufferers of duodenal ulceration. HP appears to be closely associated with acute and chronic gastritis and its eradication in these conditions is also desirable. Elimination of HP can be accomplished by a large number of antimicrobials³⁶ as well as by bismuth salts.^{37–39} The benefits of eradicating HP in asymptomatic carriers and in patients suffering from non-ulcer dyspepsia with no positive endoscopic findings have not been established. A number of these patients will have gastritis that can be detected histologically if biopsies are obtained at endoscopy.⁴⁰ It could be argued that a further portion of these patients may have had gastritis that subsided in the time between referral to endoscopy and execution of the procedure. The majority of these patients will be infected with HP and would probably benefit from its eradication.¹³ However, when evaluating the role of eradication of HP in non-ulcer dyspepsia or in a specific disease state, it is important to consider the effect of age⁴¹ and other epidemiological factors such as race and

geographical location.

This study shows that the prevalence of HP in Jordan is similar to that seen in other developing countries with infections occurring at a lower age and with the annual infection rate being double that seen in developed countries.

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