

Prevalence of *Helicobacter pylori* in Northern Jordan

Endoscopy based study

Kamal E. Bani-Hani, MD, FRCS, Shadi M. Hammouri, MD, MBBS.

ABSTRACT

Objectives: *Helicobacter pylori* infection is considered the most common infection worldwide and is associated with many other disorders. The aim of this study is to determine the prevalence of this infection among patients undergoing endoscopy in Northern Jordan.

Methods: Between November 1998 and September 2000, all patients referred from the Gastro-esophageal Clinic to the Endoscopy Unit at Princess Basma Teaching Hospital, Irbid, Northern Jordan were enrolled in this prospective study. For each patient clinical and epidemiological data was collected and endoscopy was performed. At least 3 antral biopsies were obtained from each patient, and these were examined histologically for the presence of gastritis and stained for *Helicobacter pylori* using modified Giemsa stain.

Results: A total of 197 consecutive patients (113 females) with a mean age of 40.2 years (range 15-91 years) were studied. Abdominal pain was the highest presenting symptom. Gastritis 91% and esophagitis 42%

were the most frequent endoscopic findings. Gastritis was documented histologically in 183 (93%) of patients. *Helicobacter pylori* was found in 161 patients (82%), with all of these having histological gastritis. The 11 patients with gastric ulcer, compared to the 51 out of the 59 (86%) patients with duodenal ulcer, showed *Helicobacter pylori* in their biopsies.

Conclusions: The prevalence of *Helicobacter pylori* infection in patients subjected to an upper gastrointestinal endoscopy in Jordan is high. This study confirms that *Helicobacter pylori* is significantly associated with gastritis and peptic ulcer. Further studies are needed to determine the types of *Helicobacter pylori* strains present in Jordan.

Keywords: *Helicobacter pylori*, prevalence.

Saudi Med J 2001; Vol. 22 (10): 843-847

Helicobacter Pylori (*H.pylori*) was discovered in 1983 by the 2 Australian investigators Warren and Marshal.¹ Since then, it became the most common infection worldwide and by some estimates over one half of the world population are infected with this organism.² It is well recognized that *H.pylori* infection is associated with a wide range of digestive and other extra-gastrointestinal disorders,³ including chronic active gastritis,⁴⁻⁶ atrophic gastritis,⁷

duodenal ulcer,^{8,9} gastric ulcer,^{10,11} carcinoma of the stomach¹²⁻¹⁴ mucosa-associated lymphoid tissue lymphoma¹⁵⁻¹⁷ and skin diseases.¹⁸ Although *H.pylori* is associated strongly with peptic ulcer disease, it is also postulated as one of its causal factors. However, not all cases of peptic ulcers are due to *H.pylori* infection¹⁹ and the prevalence of *H.pylori* positive peptic ulcers is variable from one population to another ranging from 35%-60%.²⁰ It is estimated that

From the Surgical Department, Princess Basma Teaching Hospital, Faculty of Medicine, Jordan University of Science and Technology (JUST), Irbid, Jordan.

Received 31st March 2001. Accepted for publication in final form 6th June 2001.

Address correspondence and reprint request to: Dr. Kamal E. Bani-Hani, Assistant Professor of Surgery, Surgical Department, Faculty of Medicine, Jordan University of Science and Technology, PO Box 3030, Irbid, Jordan. Tel. +962 (2) 7060200. Fax. +962 (2) 7095010. E-mail: kamal@just.edu.jo

0%-1% of uninfected adults acquire *H.pylori* each year.^{4,21} However, detailed information on the prevalence of the bacteria in developing countries and on the factors that may influence the pattern of distribution remains scanty. The mode of *H.pylori* transmission is unknown but it is thought to be mainly through the fecal-oral route.⁴ Oral-oral and waterborne transmissions are other modes.²² In developing countries the prevalence of *H.pylori* ranges between 70%-90%,^{5,23} while in developed countries it is approximately 50%.⁴ Since the prevalence of *H.pylori* infection is much higher in the less developed nations the socio-economic status, low living standards and poor sanitation may be implicated. The prevalence data from the Middle East is scanty, especially from Jordan. This has stimulated us to conduct this prospective study to determine the prevalence of *H.pylori* in Northern Jordan.

Methods. We conducted a prospective study during the period November 1998 through to September 2000, involving 197 consecutive patients who were referred from the Gastro-esophageal Clinic to the Endoscopy Unit in Princess Basma Teaching Hospital in Northern Jordan. These patients were presented with upper gastrointestinal symptoms such as: Nausea, vomiting, heartburn, abdominal pain, and upper gastrointestinal bleeding. History of non-steroidal anti-inflammatory drug (NSAID) ingestion, and smoking habit was documented. None of the patients had been taking anti-ulcer drugs or antibiotics for at least 2 months prior to endoscopy. Upper gastrointestinal endoscopy was carried out with a short-acting sedative (5-10 mg midazolam) and local anesthetic spray. During endoscopy the endoscopic findings such as hiatus hernia, esophagitis and its grade, gastritis, gastric or duodenal ulcers and any gross esophageal, gastric or duodenal pathology were documented. At least 3 biopsies were taken from the antrum of the stomach and put in a formalin-containing tube. These biopsies were sent to the Pathology Department at the Faculty of Medicine, Jordan University of Science & Technology (JUST) for histological examination. The presence of *H.pylori* was determined by modified Giemsa stain.

Statistical analysis. The correlation analysis was used and the analysis was performed using the Statistical package for social science (SPSS) version (9.0) for windows. Statistical significance was accepted at $p < 0.05$.

Table 1 - Endoscopic findings in all patients.

Finding	n	%
Hiatus Hernia	76	(39)
Esophagitis	83	(42)
Grade (1)	72	(36.5)
Grade (2)	9	(5)
Grade (3)	2	(1)
Gastritis	179	(91)
Gastric Ulcer	11	(6)
Duodenal Ulcer	59	(30)
n=number		

Table 2 - Distribution of patients according to the age group and *Helicobacter pylori* (*H.pylori*) status.

Age Group	n	%	<i>H.pylori</i> ⁺ %
15-19	11	(6)	7 (64)
20-39	95	(48)	81 (85)
40-59	63	(32)	49 (78)
60-79	22	(11)	18 (82)
80-91	6	(3)	6 (100)
+=positive, n=number			

Table 3 - The clinical and histological characteristics of all patients.

Characterisites	n	%	<i>H.pylori</i> %
Gender			
Male	84	(43)	70 (83)
Female	113	(57)	91 (80.5)
Smoker	57	(29)	46 (81)
Non-smoker	140	(71)	115 (82)
NSAIDs (+)	30	(15)	26 (87)
NSAIDs (-)	167	(85)	135 (81)
Nausea	71	(36)	61 (86)
Vomiting	56	(28)	47 (84)
Heartburn	118	(60)	102 (86)
Abdominal pain	194	(98.5)	159 (82)
UGIB	28	(14)	25 (89)
Gastric Ulcer	11	(6)	11 (100)
Duodenal Ulcer	59	(30)	51 (86)
Histology			
Gastritis (+)	183	(93)	159 (87)
Gastritis (-)	14	(7)	2 (14)
<i>H.pylori</i> = <i>Helicobacter pylori</i> , NSAIDs=Non steroidal anti-inflammatory drugs, UGIB= upper gastrointestinal bleeding, n=number			

Table 4 - Correlations between histological gastritis and presence of *Helicobacter pylori* (*H.pylori*).

Variable	Test	Gastritis	<i>H.pylori</i>
Gastritis	Pearson Correlation	1.000	.483
	Sig. (2-tailed)		.000
<i>H.pylori</i>	Pearson Correlation	.483	1.000
	Sig. (2-tailed)	.000	
		N	N
		197	197
Sig=significance level, N=number of observations			

Results. Out of the 197 patients, 113 were females and 84 were males (female:male ratio was 1.35: 1). The mean age was 40.2 years, ranging from 15-91 years for the trial sample. The mean age for each gender was similar to the mean age of the sample. Abdominal pain was the most frequent symptom and was found in 194 patients (98.5%). Only 30 patients 15% gave history of NSAIDs ingestion and 57 patients 29% were smokers. The most frequent findings during endoscopy are shown in Table 1. Esophagitis was graded according to the modified Savary classification.²⁴ Histological gastritis was documented in 183 patients (93%) (mean Sydney Score was 4.8; range 2-9). *Helicobacter pylori* was found in 161 patients (82%), and all these patients had histological gastritis. While *H.pylori* was seen in all patients with gastric ulcer, only 51 (86%) of the patients with duodenal ulcer had *H.pylori*. The distribution of patients according to the age group and *H.pylori* status are shown in Table 2. The clinical and histological characteristics of all patients are shown in Table 3, and the correlation between histological gastritis and the presence of *H.pylori* is shown in Table 4.

Discussion. Studying the epidemiological data on *H.pylori* is essential as it provides necessary information regarding its prevalence and incidence, which will help in establishing public health actions that could halt transmission and therefore acquisition of the infection and aids the planning of community-wide and therapeutic programs to eradicate the bacterium.²⁵ Prevalence rates of *H.pylori* have shown a strong correlation with age, low socio-economic status, but not with gender.^{4,6,26} We have shown that the prevalence of *H.pylori* increases with age but is not related to gender (Tables 2 and 3), which is consistent with previous studies.^{4,6} The prevalence of *H.pylori* was higher in the subgroup that used NSAIDs, and this could be attributed to older age of this subgroup with 26 patients above 50 years of age. Both *H.pylori* and smoking are known risk factors for peptic ulcer disease, but studies on the relationship

between *H.pylori* and smoking have given conflicting results. For example, one study showed that *H.pylori* was more common in smokers than in non-smokers,²⁷ while another report suggested that smoking was negatively associated with *H.pylori* infection.²⁸ Other studies have shown that there was no association between *H.pylori* and smoking habits.²⁹ In our study there was no significant difference in the prevalence of *H.pylori* among smokers and non-smokers. Statistical analysis of the data revealed that there was a significant correlation between presence of histological gastritis and presence of *H.pylori*. Histological gastritis was present in all patients positive for *H.pylori* compared to 61% (22/36) in the *H.pylori* negative patients. The correlation between the 2 variables was significant ($p < 0.01$) (Table 4). As in other developing countries, *H.pylori* infection is common in Northern Jordan, as it affects 82% of the population involved in this study and this percentage is similar to reported data from other developing countries in different parts of the world. Two earlier studies concerning the prevalence of *H.pylori* infection in Jordan were published. The first one reported that *H.pylori* was present in 68% of patients regardless of the pathology found during endoscopy, and 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%, which is double that seen in developed countries.³⁰ The 2nd study reported that *H.pylori* was present in more than 90% of the dyspeptic patients.³¹ Reported prevalence data including our study was taken from symptomatic patients presented for endoscopy, and this cannot be regarded as a normal population and accordingly, these studies to date over-estimate the prevalence rate of *H.pylori* in the community. The prevalence of *H.pylori* in some developing countries is approximately 90%.²³ In India it was found that the frequency of *H.pylori* increases with age and it was greater than 80% by the age of 20 years.⁶ This high prevalence was also reported in the Kingdom of Saudi Arabia where it was found that *H.pylori* affects approximately 40% of those aged 5-10 years and 70% of those 20 years or older.⁵ Another study conducted in Brazil showed the organism in 78% of patients with a wide range of alimentary tract disorders presenting for endoscopy.³² These high percentages in developing countries differ from the developed countries in which only approximately 50% of adults in the industrialized countries are infected.^{4,23} *Helicobacter pylori* was seen in 52% of the asymptomatic population in United States of America (USA)⁴ and in approximately 52% of patients with different gastrointestinal symptoms in England.³³ In addition to the low prevalence rate, this rate is falling in western countries³⁴ and the rate of acquisition is only 0%-0.5% per patient per year.^{35,36} This variability in the prevalence rate is mainly due to inadequate living

conditions, poor sanitation, hygiene and overcrowding.^{4,6,26} Prevalence rates of *H.pylori* infection among our patients with duodenal ulcer is 86% and gastric ulcer 100%. These are similar to the rates reported elsewhere. The prevalence rate of *H.pylori* in duodenal ulcer patients is approximately 95% ranging from 78%-100%,³⁷ but a recent study found *H.pylori* in only 61% of duodenal ulcer patients.³⁸ The infection rates in patients with gastric ulcer is around 84% and has varied from 44%-100%.³⁷ These observations emphasize the importance of eradication of *H.pylori* in the management of peptic ulcer disease. Although most patients with peptic ulcer are *H.pylori*-positive, recent reports of *H.pylori*-negative ulcers are becoming more frequent particularly from countries where the prevalence of *H.pylori* is already low or rapidly decreasing.¹⁹ False negative tests and NSAIDs are the major causes of *H.pylori*-negative ulcers and patients should be carefully questioned regarding the use of NSAIDs. Approximately 50% of patients with *H.pylori*-negative duodenal ulcer have no obvious cause for their ulcer and some have increased gastric acid secretion and duodenal acid load, which is likely to be of pathogenic significance.³⁹ On the other hand, *H.pylori* may be an incidental finding in approximately one 3rd of all ulcer patients.⁴⁰

In conclusion, as in other developing countries *H.pylori* infection is common, and is found in 82% of the population studied in the North of Jordan. Our study confirms that *H.pylori* is significantly associated with gastritis and peptic ulcer disease. Further studies are needed to determine the types of *H.pylori* strains present in Jordan.

References

- Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1984; 1: 1311-1315.
- Pisani P, Parkin DM, Munoz N, Ferlay J. Cancer and infection: estimates of the attributable fraction in 1990. *Cancer Epidemiol Biomarkers Prev* 1997; 6: 387-400.
- Pakodi F, Abdel-Salam OM, Debreceni A, Mozsik G. *Helicobacter pylori*. One bacterium and a broad spectrum of human disease! An overview. *J Physiol Paris* 2000; 94: 139-152.
- Graham DY, Malaty HM, Evans DG, Evans DJ Jr, Klein PD, Adam E. Epidemiology of *Helicobacter Pylori* in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. *Gastroenterology* 1991; 100: 1495-1501.
- Al-Moagel MA, Evans DG, Abdulghani ME, Adam E, Evans DJ Jr, Malaty HM et al. Prevalence of *Helicobacter Pylori* (formerly *Campylobacter*) infection in Saudi Arabia, and comparison of those with and without upper gastrointestinal symptoms. *Am J Gastroenterol* 1990; 85: 944-948.
- Graham DY, Adam E, Reddy GT, Agarwal JP, Agarwal R, Evans DJ Jr et al. Seroepidemiology of *Helicobacter Pylori* infection in India. Comparison of developing and developed countries. *Dig Dis Sci* 1991; 36: 1084-1088.
- Ohkuma K, Okada M, Murayama H, Seo M, Maeda K, Kanda M et al. Association of *Helicobacter pylori* infection with atrophic gastritis and intestinal metaplasia. *J Gastroenterol Hepatol* 2000; 15: 1105-1112.
- Figura N, Guglielmetti P, Rossolini A, Barberi A, Cusi G, Musmanno RA et al. Cytotoxin production by *Campylobacter pylori* strains isolated from patients with peptic ulcers and from patients with chronic gastritis only. *J Clin Microbiol* 1989; 27: 225-226.
- Crabtree JE, Taylor JD, Wyatt JI, Heatley RV, Shallcross TM, Tompkins DS et al. Mucosal IgA recognition of *Helicobacter pylori* 120 kDa protein, peptic ulceration, and gastric pathology. *Lancet* 1991; 338: 332-335.
- Graham DY, Lew GM, Klein PD, Evans DG, Evans DJ Jr, Saeed ZA et al. Effect of treatment of *Helicobacter pylori* infection on the long term recurrence of gastric and duodenal ulcer. A randomized, controlled study. *Ann Intern Med* 1992; 116: 705-708.
- Karita M, Morshed MG, Ouchi K, Okita K. Bismuth-free triple therapy for eradicating *Helicobacter pylori* and reducing the gastric ulcer recurrence rate. *Am J Gastroenterol* 1994; 89: 1032-1035.
- Tokunaga Y, Shirahase H, Hoppou T, Kitaoka A, Tokuka A, Ohsumi K. Density of *Helicobacter pylori* infection evaluated semiquantitatively in gastric cancer. *J Clin Gastroenterol* 2000; 31: 217-221.
- Yamagata H, Kiyohara Y, Aoyagi K, Kato I, Iwamoto H, Nakayama K et al. Impact of *Helicobacter pylori* infection on gastric cancer incidence in a general Japanese population: the Hisayama study. *Arch Intern Med* 2000; 160: 1962-1968.
- Koshida Y, Koizumi W, Sasabe M, Katoh Y, Okayasu I. Association of *Helicobacter pylori*-dependent gastritis with gastric carcinomas in young Japanese patients: histopathological comparison of diffuse and intestinal type cancer cases. *Histopathology* 2000; 37: 124-130.
- Schmausser B, Eck M, Greiner A, Kraus M, Muller-Hermelink HK. Mucosal humoral immune response to CagA shows a high prevalence in patients with gastric MALT-type lymphoma. *Virchows Arch* 2000; 436: 115-118.
- Konturek PC, Konturek SJ, Starzyska T, Marlicz K, Bielanski W, Pierzchalski P et al. *Helicobacter pylori*-gastrin link in MALT lymphoma. *Aliment Pharmacol Ther* 2000; 14: 1311-1318.
- Parsonnet J, Hansen S, Rodriguez L, Gelb AB, Warnke RA, Jellum E et al. *Helicobacter Pylori* infection and gastric lymphoma. *N Engl J Med* 1994; 330: 1267-1271.
- Wedi B, Kapp A. *Helicobacter pylori* infection and skin diseases. *J Physiol Pharmacol* 1999; 50: 753-776.
- Peura DA. The problem of *Helicobacter pylori*-negative idiopathic ulcer disease. *Bailliere's Best Practice and Research in Clinical Gastroenterology* 2000; 14: 109-117.
- Kurata JH, Nogawa AN. Meta-analysis of risk factors for peptic ulcer. Nonsteroidal anti-inflammatory drugs, *Helicobacter pylori*, and smoking. *J Clin Gastroenterol* 1997; 24: 2-17.
- Parsonnet J. The incidence of *Helicobacter Pylori* infection. *Aliment Pharmacol Ther* 1995; 9 (Suppl. 2): 45-51.
- Deltenre M, de Koster E. How come I've got it? (A review of *Helicobacter pylori* transmission). *Eur J Gastroenterol Hepatol* 2000; 12: 479-482.
- Mégraud F, Brassens-Rabbe MP, Denis F, Belbouri A, Hoa DQ. Seroepidemiology of *Campylobacter pylori* infection in various populations. *J Clin Microbiol* 1989; 27: 1870-1873.
- Ollyo JB, Lang F, Fontollet C, Monnier PH. Savary's new endoscopic grading of reflux-oesophagitis: A simple, reproducible, logical, complete and useful classification [abstract]. *Gastroenterology* 1990; 89: 100.
- Pounder RE, NG D. The prevalence of *Helicobacter pylori* infection in different countries. *Aliment Pharmacol Ther* 1995; 9 (Suppl. 2): 33-39.

26. Castro L de, Coelho LG. Helicobacter pylori in South America. *Can J Gastroenterol* 1998; 12: 509-512.
27. Rajashekhar V, Bhasin DK, Ray P, Vaiphei K, Sharma BC, Singh K. Helicobacter pylori infection in chronic smokers with non ulcer dyspepsia. *Trop Gastroenterol* 2000; 21: 71-72.
28. Ogihara A, Kikuchi S, Hasegawa A, Kurosawa M, Miki K, Kaneko E et al. Relationship between Helicobacter pylori infection and smoking and drinking habits. *J Gastroenterol Hepatol* 2000; 15: 271-276.
29. Rosenstock SJ, Jorgensen T, Andersen LP, Bonnevie O. Association of Helicobacter pylori infection with lifestyle, chronic disease, body-indices, and age at menarche in Danish adults. *Scandinavian Journal of Public Health* 2000; 28: 32-40.
30. Latif AH, Shami SK, Batchoun R, Murad N, Sartawi O. Helicobacter pylori: a Jordanian study. *Postgrad Med J* 1991; 67: 994-998.
31. Shennak MM, Kilani AF. Helicobacter pylori in dyspeptic Jordanian patients. *Trop Gastroenterol* 1998; 19: 15-18.
32. Coelho LG, Das SS, Karim QN, Walker MM, Queiroz DM, Mendes EN et al. Campylobacter Pyloridis in the upper gastrointestinal tract: a Brazilian study. *Arq Gastroenterol* 1987; 24: 5-9.
33. Coelho LG, Das SS, Payne A, Karim QN, Baron JH, Walker MM. Campylobacter Pylori in esophagus, antrum, and duodenum. A histological and microbiological study. *Dig Dis Sci* 1989; 34: 445-448.
34. Banatvala N, Mayo K, Megraud F, Jennings R, Deeks JJ, Feldman RA. The cohort effect and Helicobacter Pylori. *J Infect Dis* 1993; 168: 219-221.
35. Kuipers EJ, Pena AS, Van Kamp G, Uytterlinde AM, Pals G, Pels NF et al. Seroconversion for Helicobacter Pylori. *Lancet* 1993; 342: 328-331.
36. Parsonnet J, Blaser MJ, Perez-Perez GI, Hargrett-Bean N, Tauxe RV. Symptoms and risk factors of Helicobacter Pylori infection in a cohort of epidemiologists. *Gastroenterology* 1992; 102: 41-46.
37. Kuipers EJ, Thijs JC, Festen HPM. The prevalence of Helicobacter pylori in peptic ulcer disease. *Aliment Pharmacol Ther* 1995; 9 (Suppl 2): 59-69.
38. Jyotheeswaran S, Shah AN, Jin HO, Potter G, Ona FV, Chey WY. Prevalence of Helicobacter pylori in peptic ulcer patients in greater Rochester, NY: is empirical triple therapy justified? *Am J Gastroenterol* 1998; 93: 574-578.
39. McColl KE. Helicobacter pylori-negative ulcer disease. *J Gastroenterol* 2000; 35 (Suppl 12): 47-50.
40. Arakawa T, Higuchi K, Fujiwara Y, Tominaga K, Watanabe T, Shiba M et al. Helicobacter pylori: criminal or innocent bystander? *J Gastroenterol* 2000; 35 (Suppl 12): 42-46.