A Comparative Study Of The Prevalence Of Helicobacter Pylori Between The Inhabitants Of Areas Of Different Barometric Pressure

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Abstract

Background: There are significant geographical and social differences in the prevalence of Helicobacter pylori. Jordan Valley is located at 280-420 meters below, while Irbid city is at 600 meters above sea level. This study is designed to examine the prevalence of Helicobacter pylori infection in the below and above sea level environments.

Patients and Methods: A prospective comparative analysis of the prevalence of Helicobacter pylori in gastric biopsy specimens between patients living in Irbid city (group 1) and those living in the Jordan Valley (group 2) was performed. Another group of patients, originally from the Jordan Valley, who are currently residing in Irbid city for at least the last 10 years (group 3) was examined similarly to investigate the possible role of the genetic factor.

Results: The 3 groups were comparable in age, gender distribution, and duration of symptoms. The prevalence of Helicobacter pylori infection in group 1 was 82% (143/171). On the other hand, the prevalence of this infection in group 2 was 70% (66/96), which is significantly lower than that of group 1. The prevalence of this infection in group 3 was significantly indifferent from that of patients from Irbid city (80% (32/40)).

Conclusions: It is perhaps the environmental factors including the barometric pressure and seasonal temperature rather than the genetic background that is responsible for the difference of Helicobacter pylori infection between patients of the Jordan Valley and those of Irbid city.

INTRODUCTION

Helicobacter pylori (H. pylori) infect more than half of the world population making it the most prevalent infection worldwide [1]. It is regarded as the main etiological factor for peptic ulcer disease [2,3,4], as well as it is considered as an important risk factor for the pathogenesis of gastric polyps, MALT lymphoma and gastric cancer [5,6,7,8,9]. Additionally it might be involved in the pathogenesis of many extragastrointestinal tract diseases [10].

The prevalence rate of H. pylori infection in the West is less than 50% [$_{11}$, $_{12}$], while in the developing countries this rate is high and range between 70-90% [$_{12}$, $_{13}$, $_{14}$].

The main risk factors for H. pylori acquisition are childhood, low socio-economic status, low living standards, poor sanitation, and the presence of H. pylori-positive family members [$_{15,16,17,18,19,20,21,222,23,24$]. In addition there is a possible role for genetic factors [$_{25}$], and hereditability may explain up to 57% of H. pylori infection prevalence [$_{26}$]. Ethnicity was found to be an independent factor associated with eradication success [$_{27}$]. On the other hand, the high frequency of H. pylori infection in spouses suggests that environmental factors are more important than genetic factors for H. pylori infection [$_{28}$].

The role of these different environmental factors in the prevalence of H. pylori among different population groups seems to be significant. However, there is no available data on the effect of barometric pressure and seasonal temperatures on the prevalence of H. pylori infection. Additionally there are controversies regarding the importance of ethnic factors on such prevalence rate. This has prompted us to study the effect of these factors on the prevalence rate of H. pylori infection. Our experimental model of below sea level environment in the Jordan Valley (JV) versus above sea level environment in Irbid city seems to be a good model to look at the role of this group of environmental factors in H. pylori infection.

PATIENTS AND METHODS

JV is located at 280-420 meters below sea level. The Dead Sea in the JV is considered the lowest point on earth and located at about 420 meters below sea level. The original inhabitants of this area are Arabs with Negroid features. Irbid city (IC) is located at about 600 meters above sea level and the majority of its inhabitants are Arabs with Caucasian descent. The inhabitants of the JV come from marriages between Arabs with Caucasian descent and Blacks. The socioeconomic conditions of the people in the JV are lower than those conditions for people residing in IC. The seasonal temperatures in the JV are 3-8 0C higher than that of IC throughout the year. We conducted a prospective comparative analysis study during the period between November 1998 and November 2001, involving 320 patients who were referred from the gastro-esophageal clinic to the Endoscopy Unit at Princess Basma Teaching Hospital in IC. The patients were divided into three groups: Group 1 included 171 patients who are resident of IC; group 2 included 96 patients who are originally from and still live in the JV; group 3 included 40 patients who are originally from the JV, but currently residing in IC for at least the last 10 years. The remaining 13 patients (8 from the first group, 4 from the second group and one from the third group) were excluded from the study because of history of taking an antiulcer drugs or antibiotics within 2 months prior to endoscopy. All patients in the three groups presented with dyspeptic symptoms or upper gastrointestinal bleeding. History of non-steroidal anti-inflammatory drugs (NSAIDs) ingestion, smoking and alcohol intake for all patients were documented.

Our university research committee has approved the study proposal, as well as the manner in which informed consent was obtained from the patients. Upper gastrointestinal endoscopy was done with a short-acting sedative (5-10 mg midazolam) and local anesthetic spray. At least three antral gastric biopsies were obtained from each patient and these were examined for the presence of gastritis and stained for H. pylori using modified Giemsa stain.

Effect of ethnicity, age, sex, usage of alcohol, smoking and non-steroidal anti-inflammatory drugs, history of ulcer and

endoscopic diagnosis on prevalence of H. pylori infection were examined by univariate and multivariate analysis.

STATISTICAL ANALYSIS

The Correlation analysis and nonparametric Binomial Test were used. The analysis was performed using Statistical package for social science (SPSS) version (9.0) for windows. Statistical significant was accepted at a p<0.05.

RESULTS

Table 1 summarizes the clinical characteristics of the patients among the three groups. Group 1 included 171 patients; 99 males and 72 females, mean age is 38.9 years; range 13-91 years; group 2 included 96 patients; 56 males and 40 females, mean age is 40.3 years; range 12-74 years; and group 3 included 40 patients; 23 males, 17 females, mean age 40.1 years; range 15-68 years.

Figure 1

Table 1: The clinical characteristic of the patients among the three groups.

Group	Total	Sex		Age, mean	H. pylori	H. pylori
	Number	М	F	(range)/year	positive	negative
First	171	99	72	38.9 (13-91)	143	28
Second	96	56	40	40.3 (12-74)	66	30
Third	40	23	17	40.1 (15-68)	32	8
Total	307	17	129	39.3 (12-91)	241	66
		8				

Out of the 307 patients, 178 were males and 129 were females (male: female ratio was 1.38: 1). The mean age for the whole group was 39.3 years, ranging from 12-91 years. The mean age for each gender was similar to the mean age of the sample. Only 35 patients (15.5%) gave history of NSAIDs ingestion and 71 patients (29.7%) were smokers. The 3 groups were comparable in age, gender distribution, and duration of symptoms. There was no statistical difference between the 3 groups regarding history of NSAIDs ingestion, usage of alcohol or smoking. As seen in Table 1, the prevalence of H. pylori infection in group 1 was 82%, while the prevalence of this infection among patients in group 2 was 70%. The prevalence of H. pylori infection in patients originally from the JV and living in IC for, at least, the last 10 years (group 3) was 80%, which was significantly indifferent from that of patients of Irbid city.

To shed more light on the previous analysis, a nonparametric

Binomial test between two percentages was used. The results of the test are exhibited in Table 2. It is apparent that the difference between the percentage of group 1 and the percentage of group 2 is significant at 5% and 1% levels against the percentage of the unified group. While Table 3 shows that the difference between group 1 and group 3 is statistically insignificant as shown in the last column.

Figure 2

Table 2: A nonparametric Binomial Test; the difference between the percentage of group 1 and the percentage of group 2

		Category	Number	Observed	Test	Asymp. Sig
				Prop.	Prop.	(1-tailed)
Irbid	Group 1	1.00	143	.836257	.782	.052*
	Group 2	.00	28	.164		
	Total		171	1.000		
Jordan	Group 1	1.00	66	.6875	.782	.017†
Valley	Group 2	.00	30	.313		
	Total		96	1.000		

* Based on Z Approximation.

+ Alternative hypothesis states that the proportion of cases in the first group < .782.

Figure 3

Table 3: A nonparametric Binomial Test; the difference between the percentage of group 1 and the percentage of group 3

		Category	Number	Observed	Test	Asymp. Sig
				Prop.	Prop.	(1-tailed)
Irbid	Group 1	1.00	143	.836257	.829	.440*
	Group 3	.00	28	.164		
	Total		171	1.000		
Third	Group 1	1.00	32	.8	.829	.391†
group	Group 3	.00	8	.200		
	Total		40	1.000		

* Based on Z Approximation.

† Alternative hypothesis states that the proportion of cases in the first group < .829.</p>

DISCUSSION

There are wide geographical variations in the prevalence of H. pylori infection between different countries and between various regions within a given country [₂₉]. Additionally, variations were reported between various ethnic groups [₃₀, ₃₁]. There is no clear data to explain if these variations between and within population groups are the results of genetic factors of the different populations or the results of different environmental and socioeconomic factors acting in these different populations and ethnic groups. Data, reported

herein, suggest that the environmental factors, rather than the genetic background, have a significant role in the prevalence of H. pylori infection. This is based on the fact that this infection in patients originally from the JV and now living in IC (group 3), have a similar rate of infection to that in patients, who are originally and living in IC (group1). However, a more vivid conclusion is yet to be elucidated from a larger-scale study using a similar experimental model.

The differences in the environmental conditions such as the barometric pressure and the availability of oxygen and seasonal temperatures are, at least in part, responsible for this difference in the prevalence of H. pylori infection. However, the mechanism by which this occurs is yet to be investigated. Other studies from our institute had also suggested a significant role of environmental factors, such as barometric pressure and seasonal temperatures on the hormonal homeostasis in humans [32, 33]. Differences in leutinizing hormone and testosterone levels between exercising trained athletes in the above see level environment as compared to below see environment were suggested to be due, at least in part, to environmental conditions such as the barometric pressure and availability of oxygen [32]. The high temperature and high barometric pressure were also suggested to be responsible for differences in adrenocorticotropin and cortisol levels between people of the JV and those of IC [33].

Many studies reported on the variations in the prevalence of H. pylori infection among different racial or ethnic groups $[_{30}, _{31}]$. For example blacks and Hispanics in the USA has a higher prevalence than non- Hispanics whites [11, 25, 34,35,36]. In one study the age-adjusted prevalence was substantially higher among non-Hispanic blacks (52.7%) and Mexican Americans (61.6%) than among non-Hispanic whites (26.2%) [₃₆]. The reasons for this variation is not clear, but socioeconomic status during childhood and living in a crowded quarters plays an important factor [20]. The high prevalence of H. pylori infection among certain ethnic groups is partially explained by other factors associated with infection [36]. In one study the authors suggested that the difference in seroprevalence among blacks (16.8%), Hispanics (13.3%), and whites (8.3%) could be accounted for by differences in socioeconomic status [19]. They concluded that the socioeconomic status, not ethnic group, is the more important risk factor for acquisition of H. pylori infection during childhood [19]. This variability in the

prevalence rate is mainly due to inadequate living condition, poor sanitation and hygiene and overcrowding [11, 37, 38]. Group 3 in our experimental design included patients who are originally from the JV and now living in IC. Their genetic background is the same as those still living in the JV. Not only they are now living in the above sea level environment in IC for the last 10 or more years, but also their socioeconomic and sanitation situation is also similar to those in IC. Although these factors seem to be controlled, other environmental factors such as nutritional status were not considered, as it was concluded, from a questionnaire (data not shown), that they did not change their dietary habits since they moved to Irbid. It is suggested, here, that it is the environmental factors rather than the genetic factors are responsible for the lower prevalence in H. pylori infection in the JV compared to that in IC. In addition, it seems likely that the higher temperature and/or higher barometric pressure and perhaps other environmental factors of the JV, but not the lower socioeconomic and poor sanitary conditions of the JV (lower prevalence in group 2), nor the dietary habits and nutritional status of the people of the JV (similar prevalence of infection in groups 1 and 3), that are responsible for this difference in H. pylori infection between the JV and IC. However, this hypothesis needs further elucidation by studying this phenomenon at a larger scale using the same approach of comparison between the below sea level and above sea level environments.

Our study showed that the prevalence of H. pylori increases with age but is not related to gender, which is consistent with previous studies $[_{11}, _{13}, _{37}, _{38}]$.

As in other developing countries H. pylori infection is common in Northern Jordan, as it affects 78.5% of the population involved in this study and this percentage is more or less similar to reported data from other developing countries in different parts of the world [$_{12}$, $_{13}$, $_{14}$]. But it is much higher than the prevalence rate of lower than 50% reported from Western countries [$_{11}$, $_{12}$, $_{39}$].

CONCLUSIONS

H.pylori infection is common, and is found in 78.5% of the population studied in Northern Jordan. Our data suggest that it is perhaps the environmental factors (high temperature and high barometric pressure of the JV) rather that the genetic background that is responsible for the lower prevalence of H. pylori infection in the patients of the JV compared to those of IC.

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References

1. 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. 2. Crabtree JE, Taylor JD, Wyatt JI, et al. Mucosal IgA recognition of Helicobacter pylori 120 kDa protein, peptic ulceration, and gastric pathology. Lancet 1991; 338: 332-5. 3. Graham DY, Lew GM, Klein PD, 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: 705-8. 4. 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: 1032-5. 5. 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: 217-21. 6. Yamagata H, Kiyohara Y, Aoyagi 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: 1962-8. 7. 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: 124-30. 8. 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: 115-8. 9. Konturek PC, Konturek SJ, Starzyska T, et al. Helicobacter pylori-gastrin link in MALT lymphoma. Aliment Pharmacol Ther 2000; 14: 1311-8. 10. 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: 139-52. 11. 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:

1495-501.

12. Mégraud F, Brassens-Rabbe MP, Denis F, Belbouri A, Hoa DQ. Seroepidemiology of Campylobacter pylori infection in various populations. J Clin Microbiol 1989; 27: 1870-3. 13. Al-Moagel MA, Evans DG, Abdulghani ME, 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: 944-8. 14. Bani-Hani KE, Hammori SM. Prevalence of Helicobacter pylori in Northern Jordan. Endoscopy-based study. Saudi Med J 2001; 22: 843-7. 15. Rosenstock S, Jorgensen T, Andersen L, Bonnevie O. Seroconversion and seroreversion in IgG antibodies to Helicobacter pylori: a serology based prospective cohort study. J Epidemiol Community Health 2000; 54: 444-50. 16. Malaty HM, Graham DY. Importance of childhood socioeconomic status on the current prevalence of Helicobacter pylori infection. Gut 1994; 35: 742-5. 17. Pounder RE, Ng D. The prevalence of Helicobacter pylori infection in different countries. Aliment Pharmacol Ther 1995; 9(suppl 2): 33-9. 18. Vandenplas Y, Badriul H. Helicobacter pylori infection. Taiwan Erh Ko I Hsueh Hui Tsa Chih 1999; 40: 212-24. 19. Opekun AR, Gilger MA, Denyes SM, et al. Helicobacter pylori infection in children of Texas. J Pediatr Gastroenterol Nutr 2000; 31: 405-10. 20. Deltenre M, de Koster E. How come I've got it? (A review of Helicobacter pylori transmission). Eur J Gastroenterol Hepatol 2000; 12: 479-82. 21. Luzza F, Mancuso M, Imeneo M, et al. Evidence favouring the gastro-oral route in the transmission of Helicobacter pylori infection in children. Eu J Gastroenterol Hepatol 2000; 12: 623-7. 22. Drumm B, Perez-Perez GI, Blaser MJ, Sherman PM. Intrafamilial clustering of Helicobacter pylori infection. N Engl J Med 1990; 322: 359-63. 23. Malaty HM, Kumagai T, Tanaka E, et al. Evidence from a nine-year birth cohort study in Japan of transmission pathways of Helicobacter pylori infection. J Clin Microbiol 2000; 38: 1971-3. 24. Dominici P, Bellentani S, Di Biase AR, et al. Familial clustering of Helicobacter pylori infection: population based study. Br Med J 1999; 319: 537-41. 25. Replogle ML, Glaser SL, Hiatt RA, Parsonnet J. Biological sex as a risk factor for Helicobacter pylori infection in healthy young adults. Am J Epidemiol 1995; 142: 856-63.

26. Malaty HM, Engstrand L, Pedersen NL, Graham DY. Helicobacter pylori infection: genetic and environmental influences. A study of twins. Am Intern Med 1994; 120: 982-6.

27. Kaushik SP, Vu C. Helicobacter pylori eradication with lansoprazole, amoxycillin and clarithromycin: testing an ideal regimen in a multicultural south east Asian population and examining factors potentially influencing eradication. Aust N Z J Med 2000; 30: 231-5

28. Malaty HM, Graham DY, Klein PD, Evans DG, Adam E, Evans DJ. Transmission of Helicobacter pylori infection. Studies in families of healthy individuals. Scand J Gastroenterol 1991; 26: 927-32.

29. Parsonnet J. Helicobacter pylori: the size of the problem. Gut 1998; 43(suppl 1):S6-S9.

30. Teh BH, Lin JT, Pan WH, et al. Seroprevalence and associated risk factors of Helicobacter pylori infection in Taiwan. Anticancer Res 1994; 14: 1389-92.

31. Blecker U, Hauser B, Lanciers S, et al. The prevalence of Helicobacter pylori-positive serology in asymptomatic children. J Pediatr Gastroenterol Nutr 1993; 16: 252-6. 32. Bani Hani I, El-Migdadi F, Shotar A, Abudheese R, Bashir N. Stress from exercise in the below sea level

environment causes an increase in serum testosterone levels in trained athletes. Endocr Res 2001; 27: 19-23.

33. El-Migdadi, F, El-Akawi Z, Abudheese R and Bashir N. Effect of Ramadan fasting on serum levels of adrenocorticotropin and cortisol in people of the Jordan Valley. 2002. In preparation.

34. Smoak BL, Kelley PW, Taylor DN. Seroprevalence of Helicobacter pylori infection in a cohort of US Army recruits. Am J Epidemiol 1994; 139: 513-9.

35. Staat MA, Kruszon-Moran D, McQuillan GM, et al. A population-based serologic survey of Helicobacter pylori infection in children and adolescents in the United States. J Infect Dis 1996; 174: 1120-3.

36. Everhart JE, Kruszon-Moran D, Perez-Perez GI, Tralka TS, McQuillan G. Seroprevalence and ethnic differences in Helicobacter pylori infection among adults in the United States. J Infect Dis 2000; 181: 1359-63.

37. Graham DY, Adam E, Reddy GT, et al.

Seroepidemiology of Helicobacter Pylori infection in India. Comparison of developing and developed countries. Dig Dis Sci 1991; 36: 1084-8.

38. Castro L de, Coelho LG. Helicobacter pylori in South America. Can J Gastroenterol 1998; 12: 509-12.

39. Webb PM, Knight T, Greaves S, et al. Relation between infection with Helicobacter pylori and living conditions in childhood: evidence for person to person transmission in early life. Br Med J 1994; 308: 750-3.

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