



Original Research Article

The prevalence of *Helicobacter pylori* and risk factors infection associated in Taiz city, Yemen

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A B S T R A C T

Keywords

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IGM test;
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H. pylori infection is associated with several upper gastrointestinal disorders. There are no previous studies on the epidemiology of the infection in Yemen. The purpose of this study is to measure the rate and to explore the associated factors among the some patients in Taiz city, Yemen. This study included 83 specimens isolated from patients that chosen from five different hospitals (Al-Thawra 20, Al-Jumhoori 18, Al-Kanadi 15, Al-Rafaei 15 and Al-Ta'won 15) containing 43 male and 40 female, their aged ranged from 15 to 80 years. The data were obtained by questionnaire. *H. pylori* positive was checked by assessed IgM antibody in blood using ELISA technique. Results of this study showed that prevalence of the infection increased with age greater than 35 years. Smoking, drinking water resources, coffee drinking status were asked by a self-administered questionnaire. Results of this study showed that there was a significant correlation between smoking, drinking coffee also the type of drinking water consumed during childhood and *H. pylori* infection. *H. pylori* infection showed no significant correlation with sex, weight and consumption antibiotics.

Introduction

Gastritis has become a common health problem in over the entire world. Young and adult populations have complaints of various gastrointestinal problems. Early detection and treatment could help to maintain their good health. In developing countries, more than 80% of the population is *H. pylori* positive (Perez-Perez *et al.*, 2003). Oral ingestion of the bacterium causes infection; evidence suggests that oral-oral, gastric-oral, and fecal oral routes of transmission of *H. pylori* infection exist (Czinn, 2005).

H. pylori are urease, catalase, and oxidase positive; it can catabolic glucose (Kusters *et al.*, 2006). *H. pylori* infection can be diagnosed by several techniques such as Gram-stained smears of biopsy specimen of the gastric mucosa, culture on Skirrow's medium. It can also be diagnosed by noninvasive diagnostic tests such as urea breath test, detection of Helicobacter antigen in the stool for confirmation, and detection of IgM antibodies in the patient's serum (Levinson, 2006). Lower socioeconomic status, lower levels of

education, poor hygiene and sanitation, household crowding were associated with a higher prevalence of *H. pylori* infection (Bardhan, 1997). Lack of food hygiene and indecent sanitation are very common which create favorable environment for getting infection with *H. pylori* in crowded places like market, hospitals, restaurants etc.

Although understanding the epidemiology of the infection is important to define the scope and magnitude of its impact on the public health, parallel effort should be invested in understanding the pathogenesis of diseases associated with *H. pylori* infection. On one hand, it has been suggested that reactive oxygen species (ROS) production with damage to cell macromolecules, including DNA, may be an important mechanism of *H. pylori* action (Khanzode *et al.*, 2003). On the other hand, diet has been linked with the etiology of gastric cancer in numerous international studies. Vitamins E and C intakes are postulated to protect against development of gastric cancer (Flora, 2007).

Little is known about the factors, apart from poor living conditions during childhood that affect either acquisition or elimination of the organism (Webb *et al.*, 1994). Lifestyle factors operating during adulthood such as smoking and alcohol consumption may influence spontaneous eradication of the organism. Studies investigating the relationship between antibody evidence of *H. pylori* infection and these factors have provided inconsistent findings (Murray *et al.*, 1997; Eurogast Study Group, 1993; Brenner *et al.*, 1999; Ogihara *et al.*, 2000), but serological tests may misclassify as still infected those individuals in which the organism has been eradicated.

The main objective of this study is to estimate the prevalence of *H. pylori* infection and to identify risk factors associated with the infection in the Taiz city of Yemen population such as ethnicity, socioeconomic status and lifestyle habits.

Materials and Methods

Study population

A cross-sectional sero epidemiologic study was carried out in Taiz city. The study period was January 1st, 2012 to June 31, 2012. The total study population was 83 patients that chosen from five different hospitals (Al-Thawra 20, Al-Jumhoori 18, Al-Kanadi 15, Al-Rafaei 15 and Al-Ta'won 15) containing 43 male and 40 female, their aged ranged from 15 to 80 years. The samples have been collected randomly from patients and the total sample was 83. The history of the patients was recorded in a predesigned data collection sheet.

Questionnaire

A trained physician interviewed each volunteer and completed a detailed questionnaire. The questionnaire was designed to obtain demographic data such as age, sex, occupation, educational level, and place of birth. Socioeconomic status was also assessed.

Sample size

Eighty three randomly selected eligible patients were subjected for blood sample collection. All patients were interviewed. Verbal consent was obtained from all patients.

Sample collection and Serologic Methods

About 5 ml of venous blood was collected for *H. pylori* IgM serum determination. Purified *H. pylori* antigen (DRG, Germany) coated on the surface of micro-wells. 1:40 dilution of patient's serum was added to the wells, and the *H. pylori* IgM-specific antibody, if present, binds to the antigen. Enzyme conjugate was added, which binds to the antibody-antigen complex. The enzyme conjugate catalytic reaction is stopped at a specific time. Reading of color absorbance and result interpretation was done according to the manufacturer recommendations.

Result and Discussion

Our study included eighty three patients (43 males and 40 females) with a mean age of 40.0±16.8 years (age range, 15 to 80 years) participated in the study. Among 83 patients, the *H. pylori* positive results determined by Serum IgM Test 53.0% (44 of 83) and negative result 47.0% (39 of 83) (Table1) .

Table.1 True *H. pylori* Positive

<i>H.pylori</i>	Frequency	Percent
Negative	39	47.0
positive	44	53.0
total	83	100.0

The association of *H. pylori* infection with demographic data and drug uses of patient

The prevalence of *H. pylori* infection was defined according to the different demographic data of the patients, including gender, age, weight and drugs. Among the 83 patients who completed

data, the highest positive result was found in the age group of 36-50yr (57.7%) while the highest negative result was found in the age group of 15-20yr (60.0%) table 2.

The highest positive result was in female and it constituted 52.5%, while in male it constituted 41.9%. There were no significant statistical results related to age or sex to be considered as a risk factor. The highest positivity result was found in weight group 40-55kg (66.7%) but the lowest results were in weight group 71-85k (45.1%) table 2.

The association of *H. pylori* infection with previous patient history

The quality of water had a strong effect on the prevalence of *H. pylori* infection. The prevalence of infection among patients who usually consumed municipality or well water during childhood was 48.3% compared with 42.9% among those who usually consumed filtered water. However, the prevalence of infection among the patients who usually smoking was 55.6% compare with 36.9% among those who nonsmoking. Even the number of cigarettes seems were affect or increases the possibility of infection. The drink of coffee had a strong effect on the prevalence of *H. pylori* infection. The prevalence of infection among patients who drink coffee was 50.0% compared with 45.5% among those who not drink coffee. However the drink more than five cups per day of coffee seems are affect or increase the possibility of infection. It was found that the prevalence of infection among patients who drink more than five cups per day was 85.8% (Table 4). As shown in table.5 the history of antibiotics intake in the last month was evaluated and the results showed no significant results.

Table.2 Risk factors for *H. pylori* Infection

Variable		<i>H.pylori</i> infection			
		Negative		Positive	
		No.	%	No.	%
Age	15-20 y	6	60.0	4	40.0
	21-35 y	16	59.2	11	40.8
	36-50 y	11	42.3	15	57.7
	>50y	11	55.0	9	45.0
Sex	Male	25	58.1	18	41.9
	Female	19	47.5	21	52.5
weight	40-55	2	33.3	4	66.7
	56-70	25	54.4	21	45.6
	71-85	17	54.9	14	45.1

Table.3 Life style variables

Variable		<i>H. pylori</i> infection			
		Negative		Positive	
		No.	%	No.	%
Smoking	No	24	63.1	14	36.9
	Yes	20	44.4	25	55.6
No. of cigarettes/day	1--20	13	37.1	22	62.9
	>20	7	70	3	30
Drink of coffee	No	13	50	13	50
	Yes	31	54.3	26	45.7
How many cups per day	1--5	29	67.4	14	32.6
	>5	2	14.2	12	85.8
Type of drinking water during adulthood	Municipality or well water	32	51.7	30	48.3
	Filtered water	12	57.1	9	42.9
Type of drinking water during adulthood	Municipality or well water	14	56	11	44
During adulthood	Filtered water	30	51.8	28	48.2

Table.4 Antibiotics and drugs intake relation to *H. pylori* infection

Total		Positive		Negative		Drugs	
No	%	No	%	No	%	No yes	antibiotics in the last month
100.0	26	53.9	14	46.1	12		
100.0	57	43.9	25	56.1	32		
100.0	83	47.0	39	53.0	44		

H.pylori is a spiral-shaped microaerophilic Gram-negative bacterium that colonizes the gastric mucosa of human beings. The microorganism is the major agent of gastritis and plays an important role in the pathogenesis of peptic ulcer and gastric cancer (McGuigan, 1996). *H. pylori* are believed to infect the host by the fecal-oral route and home to the gastric mucosa. Although it is acid sensitive, *H. pylori* can survive in the stomach for short periods by neutralizing the gastric acid. The current therapy for *H. pylori* infection is efficacious but the treatment regimen is complex and demanding on the patient and it does not provide resistance to future infections (Soll, 1996). Current data suggest that the overall prevalence of *H. pylori* infection is higher both in developing countries and in lower socioeconomic groups in the developed world. Probably these populations are exposed to conditions that favor the acquisition of the microorganism such as precarious hygiene, crowded house hold conditions and deficient sanitation (de Oliveira *et al.*, 1999). The major risk factor for acquiring *H. pylori* infection is poor socioeconomic conditions during childhood. The rate of *H. pylori* infection in developed countries ranges from 10% in children to 60% in 60 year olds (Imrie *et al.*, 2001). Various methods are available for detecting *H. pylori*, but all have limitations. *H. pylori* infection can be diagnosed by invasive tests requiring (IGM).

In the present study the population age ranged between 15-80 years, with mean age of 40.0 ± 16.8 years (48.1%) are females and (51.9%) are males. In our study the rate of *H. pylori* infection according the positive results was 48.3%. Our results do not agree with the prevalence of *H. pylori* infection among

patients in other countries in the Middle East. These differences in the prevalence is might be due to the fact that the number of patients included in our study was limited, different tests used in other countries, different possible risk factor, source of drinking water, eating habits among the people. A study in Yemen (1998) showed that the prevalence of *H. pylori* infection among 275 dyspeptic patients was 82.2%. In their study they depended on URUT for detect the infection (Gunaid *et al.*, 2003). Another study in Saudi Arabia (2005) showed that the prevalence among 120 volunteer students was low (35%) in comparison to other study conducted on a larger number of the Saudi population which showed prevalence of 80% (Almadi *et al.*, 2007).

In the current study, found that the highest frequency was in the age group of 36-50yr (57.7%), which considered as a risk factor for *H. pylori* infection while the negative results was observed in the age group of 15-20yr (40.0%) with significant difference. Our study confirmed with many studies like Sari and Taiwan, which found that the frequency of *H. pylori* infection increased with age, while in the other a study in Kerman, which found that the frequency of infection did not differ among various age groups (Lin, 1999; Babamahmoodi *et al.*, 2003; Zahedi *et al.*, 2000).

The highest positive result was found in weight group 40-55kg it was 66.7% but the lowest results were in weight group 71-85kg. With regard to marital status, there were no significant differences in table 2. Epidemiologic studies indicate inconsistent result about the relation between cigarettes smoking, which is accepted as risk factor for *H.pylori* infection. Recent studies which have

analyzed the relation between cigarettes smoking and *H.pylori* risk produced controversial result while some studies suggested that *H.pylori* risk significantly rose with cigarette smoking, another study has suggested that it decreased the risk (Ozden *et al.*, 2004; Kanbay *et al.*, 2005). In the present study, found that Smoking is considered as a risk factor for many diseases and is implicated by several studies in the literature as a risk factor for *H. pylori* infection. The frequency of *H. pylori* infection in the smoker (55.6%) and non-smoker (36.9%) was significantly different in our study; this is compatible with results obtained in studies performed in Glasgow, U.K., which found that smokers (73%) have higher prevalence's than never-smokers (59%) (24). However, other reports from the literature showed no association of smoking with *H. pylori* infection (Woodward *et al.*, 2000; Akin *et al.*, 2004; Constanza *et al.*, 2004). There are reasons why smoking might have little effect on, or even increase, the hostility of the gastric environment to *H. pylori*. The acid gastric pH prevents most organisms from thriving or even surviving in the stomach. *H.pylori*, however, has an electropositive internal milieu; twice the number of basic amino acids, arginine and lysine, as *Haemophilus influenza* and *Escherichia coli*; and powerful urease activity, with the ability to produce both ammonia and factors that inhibit parietal cell acid production (Everhart *et al.*, 2000). All these attributes make survival of *H. pylori* in the stomach less influenced by the reduction in pH which may accompany with smoking consumption (Tomb *et al.*, 1997).

Recent studies which have analyzed the relation between coffee consumption and *H.pylori* infection suggested that *H.pylori* infection significantly rose with coffee

consumption (Endoh and Leung, 1994; Brenner *et al.*, 1997).

In the present study, found that coffee consumption is considered as a risk factor for *H. pylori* infection. The frequency of *H. pylori* infection in the coffee consumer (50.0%) and non-coffee consumer (45.7%) was significantly different in our study; this is compatible with results obtained in the above studies.

A survey in Germany in 1997 on 447 patients with an overall prevalence of 21%, coffee consumption showed a positive dose-response relation with active infection. The positive relation between coffee consumption and *H.pylori* infection identified in that study is consistent with results from a cohort study among epidemiologists in which the risk of sero conversion (change from negative to positive results for antibodies to *H. pylori* in serum) was 4.6 times higher among those who drank more than 2 cups of caffeinated drinks a day than among the others. The mechanisms underlying this association require further research (Brenner *et al.*, 1999).

In recent studies, it has been suggested that *H.pylori* prevalence has been decreasing over the years. It was thought that better health, hygiene and socioeconomic condition and use of antibiotics could be the reason for decrease (Reshetnikov *et al.*, 2003; Almad *et al.*, 2007). In present study noted that among the study patients, 57 took antibiotics one day to one month prior sample collection, 56.1% of them were shown to be negative while among the 26 who did not consume antibiotics, only 46.1 % of them were negative for *H. pylori*. This reduction could be explained by the fact that the antibiotic consumption may reduce the number and activity of the

organism, therefore interfering with the result outcome producing false negative results (table5).

The frequency and risk factors for contamination of *Helicobacter pylori* infection was investigated among patients at Taiz city of yemen. Eighty three patients randomly selected from different hospitals in Taiz, namely Al-Thawrah, Al-Jumhori, Al-Kanadi, Al-Ta'awon, and Al-Rafaei. The age of the patients was ranging between the ages of 15-80. The data were obtained by questionnaire. *H.pylori* positive was checked by assessed IgM antibody in blood using ELISA technique. Results of this study showed that prevalence of the infection increased with age greater than 35 years. Smoking, drinking water resources, coffee drinking status were asked by a self-administered questionnaire. Results of this study showed that there was a significant correlation between smoking, drinking coffee also the type of drinking water consumed during childhood and *H. pylori* infection. *H. pylori* infection showed no significant correlation with sex, weight and consumption antibiotics.

References

- Akin, L., S. Tezcan, S, et al., 2004. Seroprevalence and some correlates of *Helicobacter pylori* at adult ages in Gulveren Health District, Ankara, Turkey. *Epidemiol. Infect.* 132: 847-856.
- Almad, M.A., A.M. Aliebreem, F.A. Tounesi, et al., 2007. *Helicobacter pylori* prevalence among medical students in high endemic area. *Saudi. Med.J.* 28: 896-898.
- Almadi, M., A. Aljebreen, F. Tounesi and Abdo, A. 2007. *Helicobacter pylori* prevalence among medical students in a high endemic area. *Saud.Med. J.* 28(6) :896-898.
- Babamahmoodi, F., A. Ajemi, M. Kalhor, R. ShfieiGh and Khalilian, A.R.2003. Seroepidemiologic study of infection with *Helicobacter pylori* in Sari, Iran in 2001-02. *J. Mazandaran Uni. Medi. Sci.* 14: 39-47.
- Bardhan, P.K., 1997. Epidemiological features of *Helicobacter pylori* infection in developing countries. *Clin. Infect. Dis.* 25: 973-78.
- Brenner, H., D. Rothenbacher, G. Bod and Adler, G.1999. Inverse gradedrelationship between alcohol consumption and active infectionwith *Helicobacter pylori*. *Am. J. Epidemiol.* 149:571-6.
- Brenner, H., D. Rothenbacher, G. Bode and Adler, G. 1997. Relation ofsmoking and alcohol and coffee consumption to active *Helicobacterpylori* infection: Cross sectional study. *Br. Med. J.*315:1489-92.
- Brenner, H., G. Berg, N. Lappus, et al., 1999. Alcohol consumption and*Helicobacter pylori* infection; results from the German National Health and Nutrition Survey. *Epidemiol.* 10:214-8.
- Constanza, C., L. Eduardo, et al., 2004. Determinants of *Helicobacter pylori* seroprevalence inMexican adolescents. *Helicobacter.*9 (2): 106-114.
- Czinn, S.J., 2005. *Helicobacter pylori* infection: Detection, investigation, and management. *J. Pediatr.* 146(3 Suppl): S21-6.
- De Oliveira, A., G. Rocha, D. Queiroz, M. Barbosa and Silva, S. 1999-prevalence of *H.pylori* infection in a population from the rural area of aracua, MG,Brazil. *Revista de Microbiol.* 30(1):59-61.
- Endoh, K., and Leung, F. 1994. Effects of smoking and nicotine on the gastric mucosa: A review of clinical and experimental evidence. *Gastroenterol.* 107(3):864-878.
- Eurogast Study Group. 1993. Epidemiology of, and risk factors for, *Helicobacter pylori* infection among 3194 asymptomatic subjectsin 17 populations. *Gut.* 34:1672-6.
- Everhart, J., D. Moran, et al., 2000. Seroprevalence and ethnic differences in *H. pylori* infection among adults in the

- United States. J. Infect. Dis. 171: 1359-1363.
- Flora, S.J., 2007. Role of free radicals and antioxidants in health and disease. Cell MolBiol; 53(1):1-2.
- Gunaid, A., N. Hassan and Murray-Lyon, I. 2003. Prevalence and risk factors for *Helicobacter pylori* infection among Yemeni dyspeptic patients. Saud. Med. J. 24(5) :512-517
- Imrie, C., FRACP, M. Rowland, M. Billy Bourke and Brendan Drumm, M. 2001. Is *Helicobacter pylori* Infection in Childhood a Risk Factor for Gastric Cancer?. *Pediatrics*. 107(2): 373-380
- Kanbay, M., Gur, G., Arslan, H., et al.(2005): The relationship of ABO blood group, age, Gender, smoking, and *Helicobacter pylori* infection *Dig. Dis.*, 50, 1214-1217.
- Khanzode, S., Khanzode, D, et al., 2003. Serum and plasma concentration of oxidants and antioxidants in patients of *Helicobacter pylori* gastritis and its correlation with gastric cancer. *Cancer let.* 195: 27-31.
- Kusters, J.G., A.H.M.V. Vliet and Kuipers, E.J. 2006. Pathogenesis of *Helicobacter pylori* Infection. *Clin. Microbiol. Rev.* 19(3): 449-490.
- Levinson, W., 2006. Gram-negative rods related to the enteric tracts. In: Review of Medical Microbiology and Immunology, 9th ed. Lange Medical Books/McGraw-Hill Inc, pp. 133-151.
- Lin, D.B., 1999. Seroepidemiology of *Helicobacter pylori* infection among preschool children in Taiwan. *Am J. Trop Med Hyg.* 61: 554-558.
- McGuigan, J., 1996. *Helicobacter pylori*: the versatile pathogen. *Dig.Dis.* 14(5) :284-303.
- Murray, L.J., K.B. Bamford, E.E. McCrum and Evans, A.E.1997. Epidemiology of *Helicobacter pylori* infection in 4742 randomly selected subjects from Northern Ireland. *Int. J. Epidemiol.* 26:880-7.
- Ogihara, A., S. Kikuchi, A. Hasegawa, et al., 2000. Relationship between *Helicobacter pylori* infection and smoking and drinking habits. *J. Gastroenterol.Hepatol.* 15:271-6.
- Ozden, A., G. Bozday, Ozkan, M., et al., 2004. Changes in the seroepidemiological Pattern of *Helicobacter pylori* infection over the last 10 years in Turkey. *Turk.J. Gastroenterol.*, 15, 156-158.
- Perez-Perez, G.I., R.B. Sack, R.Reid, M. Santosham, J. Croll and Blaser, M.J. 2003. Transient and persistent *Helicobacter pylori* colonization Native American children. *J. Clin. Microbiol.* 41(6): 2401-2407.
- Reshetnikov, O.V., D.V. Deniscva, L.G. Zavyalova, et al., 2003. *Helicobacter pylori* seropositive among adolescents in Novosibirsk, Russia prevalence and associated factors. *J. pediatr. Gastroenterol. Nutr.* 36:72-76.
- Soll, A., 1996. Consensus conference. Medical treatment of peptic ulcer disease. Practice guidelines. Practice Parameters Committee of the American College of Gastroenterology. *Jama.* 275(8):622-629
- Tomb, J., O. White, A. Kerlavage, R. Clayton, G.Sutton *et al.*, 1997. The complete genome sequence of the gastric pathogen *Helicobacter pylori*. *Nature.* 388(6642):539-47
- Webb., P.M., T. Knight, S. Greaves, et al., 1994. Relation between infection with *Helicobacter pylori* and living conditions in childhood: Evidence for person to person transmission in early life. *Br. Med. J.* 308:750-3.
- Woodward, M., C. Morrison and McColl, K. 2000. An investigation into factors associated with *Helicobacter pylori* infection. *J. Clin.Epidemiol.* 53:175-81.
- Zahedi, M.J., S. Darvish Moghadam, M. Atapour and Jahanbakhsh Absasi, M. 2000. Relative frequency of *Helicobacter pylori* infection in patients referring to health centers in 2000, in Kerman, Iran. *J. Med. Uni. Kerman.* 3: 140-144.