ORIGINAL ARTICLE

Association of *Helicobacter pylori* infection with metabolic parameters and dietary habits among medical undergraduate students in southeastern of Iran

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Background: To date, there is still inconclusive evidence on the extra-gastric manifestations of Helicobacter pylori (*H.pylori*) infection. This study aimed to determine whether there is an association between *H.pylori* infection with metabolic syndrome and dietary habits among medical undergraduate students in south-eastern of Iran, Zahedan. **Materials and Methods:** This cross-sectional study was done among 363 undergraduate students in Zahedan University of Medical Sciences during spring 2014. All subjects completed a questionnaire including demographic factors and dietary habits. Serum *H. pylori*-specific IgG antibodies, total cholesterol (TC), triglycerides (TG), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), and fasting blood sugar (FBS) levels were measured after an overnight fast. **Results:** The seroprevalence of *H. pylori* infection was 45.7%. *H. pylori*-positive subjects had lower mean levels of TC and TG and higher levels of HDL-C compared to *H. pylori*-negative subjects. In addition, lower levels of LDL-C ($P = 0.044$) and FBS ($P = 0.05$) were observed among subjects with positive *H pylori* infection. Only rare consumption of raw vegetables (odds ratio [OR] =3.74, 95% confidence interval [CI] =1.37–5.24) as well as higher levels of FBS (OR = 1.031, 95% CI = 1.001–1.99) were significantly associated with higher odds of *H. pylori* infection in both the univariate and multiple logistic regression analysis. **Conclusion:** In a small population of young students in southeastern of Iran, *H. pylori* infection was associated with low consumption of raw vegetables and higher serum blood glucose.

Key words: Dietary habits, *Helicobacter pylori*, metabolic parameters, southeastern of Iran, undergraduate students

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INTRODUCTION

Helicobacter pylori is a Gram-negative, microaerophilic, spiral-shaped bacterium which is categorized as a Class I carcinogen in 1994 by the World Health Organization.[1,2] The infection rate varies by country and is higher in developing countries than in developed nations.[3] In Iran, it is estimated about half the population is infected with *H. pylori*.[4,5]

The role of *H. pylori* infection in gastric-related diseases, such as peptic ulcers, mucosa-associated lymphoid tissue - lymphoma, and gastric cancer has been well documented.[6] In recent years, researchers have investigated the extragastric manifestations of *H. pylori*. It is hypothesized that chronic inflammation induced by *H. pylori* may alter lipid and glucose metabolism through releasing inflammatory mediators including cytokines (e.g., interleukin [IL]-1, IL-6, tumor necrosis factor-α [TNF-α], interferon-α], and acute phase proteins (e.g., C-reactive protein [CRP]). Moreover, *H. pylori* colonization can influence on releasing gastrointestinal hormones such as ghrelin, leptin, gastrin, and somatostatin which may result in alteration in glucose homeostasis. Therefore, chronic *H. pylori* infection may increase the risk of

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the development of dyslipidemia, insulin resistance, and related morbidities.[7-9] This hypothesize was confirmed in a large population of Taiwanese adults by Chen *et al*. which showed *H. pylori* infected participants had significantly higher fasting blood sugar (FBS), low-density lipoprotein-cholesterol (LDL-C) and triglycerides (TG), and lower high-density lipoprotein-cholesterol (HDL-C) levels compared to uninfected ones.[10] Although some researches did not report such a relation.[11,12]

It is believed that environmental factors can potentially affect the development of *H. pylori* infection in different population.[13] In comparison with other environmental factors, less attention has been paid to the relationship of diet with *H. pylori* infection. It is suggested that some dietary factors such as fruits and vegetables may have a protective effect against *H. pylori* infection likely due to their antioxidants-rich contents.[14,15] On the other hand, some reports indicated that diet may increase the susceptibility to *H. pylori* infection through contamination of foods with *H. pylori* bacterium.[16,17] The relationship of diet-infection is still unclear and providing further evidences to elucidate this association seems to be crucial.

Although there are many epidemiological studies in Iran on the prevalence of *H. pylori* infection in different geographical regions and subpopulations, but only a few of them have investigated the extragastric manifestations of *H. pylori*. For this reason, the aim of this study was to investigate the prevalence of *H. pylori* infection and its relationship with serum lipid profile, FBS, and dietary habits among undergraduate students of Zahedan University of Medical Sciences.

MATERIALS AND METHODS

Study subjects, geographical area, inclusion and exclusion criteria

This cross-sectional descriptive study was performed on undergraduate students of Zahedan University of Medical Sciences during spring 2014 at Zahedan City, Center of Sistan and Baluchestan Province, Iran. All undergraduate students aged 18–24-year-old were enrolled at the study. Subjects with previous or current treatment with proton pump inhibitors or prior *H. pylori* eradication therapy or serious illness were excluded from the study. Finally, a total of 363 students were recruited into the study. Informed consent was obtained from each subject. The study protocol was approved by the Ethics Committee of Zahedan University of Medical Sciences (Research Project Number: 6751) and Research Center for Children and Adolescent Health, Zahedan, Islamic Republic of Iran.

Data collection

Data were collected by a self-questionnaire including demographic characteristics and dietary habits. Age, sex, parental education, marital status, family size, place of living, drinking water source, consumption of tea, cola, raw vegetables, fruits, and dairy foods of study subjects were recorded in a database.

Serological detection of *Helicobacter pylori*-specific IgG antibodies

Blood samples were collected from the subjects by taking 5 ml of venous blood between 07:00 and 10:00 am after an overnight fast (12 h of fasting). *H. pylori*-specific IgG antibodies were determined by an enzyme-linked immunosorbent assay (ELISA) using PishtazTeb Kits (PhishtazTeb Diagnostic Co., Tehran, Iran) with reported a sensitivity of 97% and a specificity of 91%. A cutoff antibody titers ≥20 were interpreted as positive according to the manufacturer's instructions.

Evaluation of serum lipid profile and fasting blood sugar

Total cholesterol (TC) and TG concentrations were measured by enzymatic methods with cholesterol esterase, cholesterol oxidase, and glycerol phosphate oxidase using ParsAzmoon Kits (ParsAzmoon Co., Tehran, Iran).The interassay coefficients of variability (inter-assay %CV) of TC and TG kits were reported 2% and 1.6%. The HDL-C levels were measured by use of a phosphotungstic acid and magnesium chloride fluid for the precipitation of the apolipoprotein B-containing lipoproteins. The LDL-C levels were calculated according to the Friedewald formula.[18] Serum FBS was assayed using an enzymatic method with glucose oxidase (ParsAzmoon Kit, inter-assay %CV = 3%).

Statistical analysis

All data were analyzed using Stata version 12 software (Stata Corporation, College Station, Texas, USA). Categorical variables were compared using the Chi-square test. The normality of parameters was determined using the Kolmogorov–Smirnov test. The independent sample *t*-test or Mann–Whitney U-test were used to compare continuous variables between two groups as appropriate. Logistic regression analysis was done to evaluate the adjusted association of *H. pylori* infection as a binary dependent variable with other study variables. Model estimation and the goodness-of-fit of the logistic regression model were evaluated by the Hosmer and Lemeshow test. The test showed a goodness-of-fit for the model adjustment ($P = 0.51$), obtaining a sensitivity of 76%, and a specificity of 81% with a cutoff of 49%. Variance inflation factor was used to assess the multicollinearity, and there was no collinearity between independent variables. Hence, all independent variables were entered to the final multiple logistic regression model. $P < 0.05$ was defined as statistically significant.

RESULTS

Among 363 students, 166 (45.7%) had *H. pylori* infection. Table 1 shows the demographic and lifestyle characteristics of students. The subjects of study were consisted of 52.9% male and 47.1% female with the mean age of 21.9 ± 2.12, respectively. The majority were unmarried, lived in urban regions, and came from at least four member families. On the basis of parental education level, almost one-third (35.5%) had a father with university degree or a mother with <8 years of education. The piped water was the main drinking water source of study subjects. Consumption of tea, cola, raw vegetables, fruits, and dairy foods were reported one to six times per week by half of the students, respectively. The Chi-square test showed no statistically significant difference in mentioned demographic variables and dietary habits between *H. pylori*-positive and *H. pylori*-negative subjects.

Table 2 compared the serum blood lipids and glucose between *H. pylori*-positive and *H. pylori*-negative subjects. *H. pylori*-positive subjects had significantly lower mean levels of LDL-C (P = 0.044) and FBS (P = 0.05) compared to *H. pylori*-negative ones. No significant difference was found in mean levels of TC, TG and HDL-C between the two groups.

As it shown in Table 3, univariate analysis showed that male gender (odds ratio [OR] =1.4, 95% confidence interval [CI] =1.05–2.67) and higher level of LDL-C (OR = 1.11, 95% CI = 1.05–1.98) were significantly associated with higher odds of *H. pylori* infection. While such an association was not maintained in multiple logistic regression model. Also, living in families with four family members or less was significantly associated with increased odds of *H. pylori* infection in logistic regression analysis (OR = 1.95, 95% CI = 1.17–4.23).Only rare consumption of raw vegetables (OR = 3.74, 95% CI = 1.37–5.24), as well as higher levels of FBS (OR = 1.031, 95% CI = 1.001–1.99) were significantly correlated with higher odds of *H. pylori* infection in both the univariate and multiple logistic regression analysis.

DISCUSSION

With respect to the high prevalence of both *H. pylori* infection and metabolic syndrome in Iranian population,[4,19] the present study aimed to examine the relationship of *H. pylori* infection and metabolic parameters among a group of young individuals. Despite the lower mean levels of LDL-C and FBS among *H. pylori*-positive subjects in bivariate analysis, a higher odds of *H. pylori* infection was observed with elevated levels of LDL-C in the univariate analysis as well as elevated levels of FBS in both univariate and multiple logistic regression analysis. One possible explanation for these differences is that such adjusted associations might be mediated by the negative effect of confounding variables which were not considered in the univariate analysis model. Therefore, we used the results of logistic regression analysis to interpret our study findings.

In terms of *H. pylori* infection and lipid profile, studies reported contradictory findings. It is believed that proinflammatory cytokines activated by chronic *H. pylori* infection can create an atherogenic lipid profile through multiple ways such as stimulating lipoprotein lipase activity in adipose tissue, increasing hepatic fatty acid synthesis, and affecting lipolysis.[20] With this regard, some authors considered *H. pylori* as a possible risk factor for dyslipidemia due to increasing TC, TG, LDL, and decreasing HDL levels.[9,10,21] In the present study, *H. pylori*-positive subjects had lower mean levels of TC, TG, LDL-C, and higher levels of HDL-C compared to *H. pylori*-negative subjects, but it was only significant for LDL-C levels in bivariate analysis. Although in univariate analysis, we observed higher odds of *H. pylori* infection with elevated levels of LDL-C, which was not sustained significant in logistic regression analysis. Similar results were reported by Sotuneh *et al*. and Naja *et al*. They suggested that the strain type of *H. pylori* or the host genetic factors could affect inflammatory response to *H. pylori* and by this way, *H. pylori* might act differently in blood lipids modifications.[11,12] The variation observed in blood lipid levels in the present study in comparison with above-mentioned reports may be the result of different lifestyle habits of study population such as differences in dietary factors, physical activity, and smoking status. These factors can potentially affect blood lipids and thus should be regarded alongside with *H. pylori* infection when interpreting the results.[22]

The association between glycemic status and *H. pylori* infection remains unclear. In this study, logistic regression analysis revealed that *H. pylori* infection was significantly associated with elevated FBS levels. Some authors have considered *H. pylori* infection as a risk factor for the development of insulin resistance. They suggested that proinflammatory cytokines produced by *H. pylori* infection such as CRP, IL-6, and TNF-α may shift glucose metabolism toward a diabetogenic direction by multiple mechanisms including stimulatory effect on secretion of insulin counter-regulatory hormones or cytokines, increasing free fatty acids oxidation, interfering with the action of glucose transporter protein GLUT4, altering the structure of insulin receptor substrate, and thus impairing action of insulin. In addition, *H. pylori* decreases ghrelin levels, which related to higher fasting insulin concentrations, and increases leptin levels, which may result in impairment of glucose-stimulated insulin secretion and apoptosis of pancreatic β-cells. On the other hand, *H. pylori* can increases

Table 1: Bivariate analysis of the demographic characteristics and dietary habits (as independent variables) with *Helicobacter pylori* infection among medical undergraduate students in southeastern of Iran

Independent variable	Total (*n*=363)	*Helicobacter pylori*		*P*†
		Positive (*n*=166)	Negative (*n*=197)	
Age (years)				
<20	147 (40.5)	61 (41.5)	86 (58.5)	0.18
≥20	216 (59.5)	105 (48.6)	111 (51.4)	
Sex				
Male	192 (52.9)	95 (49.5)	97 (50.5)	0.05
Female	171 (47.1)	71 (41.5)	100 (58.5)	
Educational status of father				
Illiterate	35 (9.6)	15 (42.9)	20 (57.1)	0.93
<8 years of schooling	77 (21.2)	36 (46.8)	41 (53.2)	
8-12 years of schooling	122 (33.6)	58 (47.5)	64 (52.5)	
University	129 (35.5)	57 (44.2)	72 (55.8)	
Educational status of mother				
Illiterate	94 (25.9)	49 (52.1)	45 (47.9)	0.17
<8 years of schooling	129 (35.5)	63 (48.8)	66 (51.2)	
8-12 years of schooling	76 (20.9)	29 (38.2)	47 (61.8)	
University	64 (17.6)	25 (39.1)	39 (60.9)	
Marital status				
Unmarried	336 (92.6)	154 (45.8)	182 (54.2)	0.88
Married	27 (7.4)	12 (44.4)	15 (55.6)	
Place of living				
Urban	299 (82.4)	132 (44.1)	167 (55.9)	0.19
Rural	64 (17.6)	34 (53.1)	30 (46.9)	
Family size				
≤4	84 (23.1)	40 (47.6)	44 (52.4)	0.69
>4	279 (76.9)	126 (45.2)	153 (54.8)	
Drinking water source				
Piped water	313 (86.2)	148 (47.3)	165 (52.7)	0.30
Bottled water	13 (3.6)	4 (30.8)	9 (69.2)	
Piped and bottled water	37 (10.2)	14 (37.8)	23 (62.2)	
Tea intake (times weekly)[a]				
Rare	66 (18.2)	28 (42.4)	38 (57.6)	0.74
1-6	172 (47.4)	82 (47.7)	90 (52.3)	
7	125 (34.4)	56 (44.8)	69 (55.2)	
Cola intake (times weekly)[b]				
Rare	141 (38.8)	63 (44.7)	78 (55.3)	0.93
1-6	193 (53.2)	90 (46.6)	103 (53.4)	
7	29 (8.0)	13 (44.8)	16 (55.2)	
Raw vegetable intake (times weekly)[c]				
Rare	117 (32.2)	44 (37.6)	73 (62.4)	0.05
1-6	199 (54.8)	97 (48.7)	102 (51.3)	
7	47 (12.9)	25 (53.2)	22 (46.8)	
Fruits intake (times weekly)[d]				
Rare	62 (17.1)	25 (40.3)	37 (59.7)	0.36
1-6	233 (64.2)	113 (48.5)	120 (51.5)	
7	68 (18.7)	28 (41.2)	40 (58.8)	
Dairy foods intake (times weekly)[e]				
Rare	40 (11.0)	22 (55.0)	18 (45.0)	0.44
1-6	204 (56.2)	90 (44.1)	114 (55.9)	
7	119 (32.8)	54 (45.4)	65 (54.6)	

†*P* value was derived from Chi-square test; [a]How often the student has consumed tea?; [b]How often the student has consumed cola?; [c]How often the student has consumed raw vegetables?; [d]How often the student has consumed fruits?; [e]How often the student has consumed dairy foods?

gastrin levels which increase glucose-stimulated insulin secretion, and decreases somatostatin levels, which inhibits insulin secretion. Therefore, *H. pylori* infection may lead to the development of insulin resistance.[7,9,23] However, other

Table 2: The mean and standard deviation of laboratory data for *Helicobacter pylori*-positive and *Helicobacter pylori*-Negative students in southeastern of Iran

Variable	Total (*n*=363)	*Helicobacter pylori*		*P*†
		Positive (*n*=166)	Negative (*n*=197)	
TG (mg/dL)	86.1±48.5 (79, 30-711)‡	83.3±35.3 (77.5, 30-279)	88.4±57.3 (80, 31-711)	0.52
TC (mg/dL)	132.5±29.8 (131, 75-233)	131.5±29.1 (126.5, 79-209)	133.4±30.5 (133, 75-233)	0.55
LDL-C (mg/dL)	66.7±20.06 (65, 17-122)	64.5±14.1 (64, 21-118)	67.4±13.1 (66, 17-122)	0.044*
HDL-C (mg/dL)	43.6±12.9 (40., 25-77)	44.7±13.1 (42, 25-76)	42.7±12.7 (39, 25-77)	0.14
FBS (mg/dL)	68.7±10.4 (68, 27-103)	66.4±8.3 (67, 43-103)	69.5±9.4 (70, 27-99)	0.05*

†TC, LDL-C, FBS: *P* values by independent sample *t*-test; TG, HDL-C: *P* values by Mann-Whitney U-test; ‡Mean±SD (median, minimum-maximum); *Significant variables.
TG = Triglycerides; TC = Total cholesterol; LDL-C = Low-density lipoprotein-cholesterol; HDL-C = High-density lipoprotein-cholesterol; FBS = Fasting blood sugar; SD = Standard deviation

Table 3: Results of univariate and multiple logistic regression analysis between dependent and independent variables (dependent variable is *Helicobacter pylori* infection status)

Variable	*B*	Univariate OR (95% CI)	Adjusted OR† (95% CI)
Age	−0.029	0.97 (0.88-1.07)	0.92 (0.77-1.11)
Sex			
Male	0.32	1.4 (1.05-2.67)*	1.24 (0.85-1.31)
Female‡		1	
Educational status of father			
Illiterate	0.48	1.05 (0.49-2.24)	2.05 (0.71-6.13)
<8 years of schooling	−0.1	0.9 (0.51-1.58)	1.22 (0.83-4.21)
8-12 years of schooling	−0.14	0.87 (0.53-1.43)	1.02 (0.71-3.2)
University‡		1	
Educational status of mother			
Illiterate	−0.54	0.58 (0.3-1.12)	0.54 (0.23-3.18)
<8 years of schooling	−0.4	0.67 (0.36-1.23)	0.62 (0.45-1.98)
8-12 years of schooling	0.029	1.03 (0.52-2.05)	0.82 (0.55-4.98)
University‡		1	
Marital status			
Unmarried	0.058	1.06 (0.48-2.32)	1.92 (0.98-2.48)
Married‡		1	
Place of living			
Urban	−0.37	0.69 (0.4-1.19)	0.42 (0.22-2.08)
Rural‡		1	
Family size			
≤4	0.095	1.10 (0.67-1.8)	1.95 (1.17-4.23)*
>4‡		1	1
Drinking water source			
Piped water	−0.4	0.67 (0.34-1.36)	0.84 (0.61-2.7)
Bottled water	0.31	1.37 (0.35-5.29)	1.50 (0.65-3.43)
Piped and bottled water‡		1	
Tea intake (times weekly)			
Rare	0.095	1.10 (0.6-2.1)	0.72 (0.25-1.08)
1-6	−0.11	0.89 (0.56-1.41)	0.78 (0.61-1.29)
7‡		1	
Cola intake (times weekly)			
Rare	0.006	1.006 (0.45-2.24)	0.66 (0.31-1.89)
1-6	−0.072	0.93 (0.42-2.03)	0.88 (0.43-2.29)
7‡		1	
Raw vegetables intake (times weekly)			
Rare	0.64	1.90 (1.01-3.73)*	3.74 (1.37-5.24)*
1-6	0.17	1.19 (0.63-2.25)	2.02 (0.46-3.78)
7‡		1	
Fruits intake (times weekly)			
Rare	0.029	1.03 (0.51-2.08)	0.99 (0.21-2.99)
1-6	−0.3	0.74 (0.43-1.30)	0.63 (0.3-2.09)
7‡		1	

Contd...

Table 3: Contd...

Variable	*B*	Univariate OR (95% CI)	Adjusted OR† (95% CI)
Dairy foods intake (times weekly)			
Rare	−0.38	0.68 (0.33-1.36)	0.81 (0.41-2.01)
1-6	0.048	1.05 (0.66-1.65)	0.71 (0.44-2.31)
7‡		1	
TG (mg/dL)	0.001	1.001 (0.99-1.10)	1.21 (0.57-3.97)
TC (mg/dL)	−0.01	0.99 (0.97-1.11)	1.07 (0.55-2.08)
LDL-C (mg/dL)	0.01	1.11 (1.05-1.98)*	1.55 (0.79-3.05)
HDL-C (mg/dL)	−0.01	0.99 (0.96-1.01)	0.85 (0.66-1.91)
FBS (mg/dL)	0.02	1.022 (1-1.14)*	1.031 (1.001-1.99)*

†Adjusted OR, each variable adjusted for the other variables based on Hosmer-Lemeshow method to model building; ‡Reference group; *Significant at level $P<0.05$. TG = Triglyceride; TC = Total cholesterol; LDL-C = Low-density lipoprotein-cholesterol; HDL-C = High-density lipoprotein-cholesterol; FBS = Fasting blood sugar; OR = Odds ratio

studies found no association between *H. pylori* infection and glycemic status.[11,12] The inconsistent results reported by studies may be due to the differences in study population characteristics and their methodology.

In terms of *H. pylori* infection and dietary habits, we found an inverse association between *H. pylori* infection and consumption of raw vegetables. This is in line with findings of some previous reports.[14,15] New evidence suggested that phytochemical components found in vegetables such as isothiocyanate sulforaphane and glucosinolate can inhibit *H. pylori* colonization and gastric mucosa inflammation and thus, they may have antibacterial activity against *H. pylori*.[24] In contrast, some studies reported that *H. pylori*-positive individuals had significantly higher intake of raw vegetables compared to negative ones which may be contributed intransmission of bacteria through contaminated foods probably due to poor hygiene conditions of study population.[16,17] Moreover, the results of some clinical trials represented the beneficial effects of dietary factors such as Omega-3 polyunsaturated fatty acids or sulforaphane-rich broccoli sprouts in inhibition of *H. pylori* colonization, attenuation of *H. pylori*-induced inflammation, and modifying blood lipid distortion among *H. pylori*-infected patients.[25,26] These finding may strengthen the possible role of diet in *H. pylori* infection. However, more researches especially longitudinal ones are needed to clarify the relationship of diet-infection.

A potential limitation of this study is the small sample size of study which might limited the ability to detect significant results. Increased sample size would be needed to confirm the observed tendency toward metabolic parameters in *H. pylori*-infected subjects. Another limitation is the lack of ability of serological tests to distinguish a current *H. pylori* infection from a past one. As a result, it might lead to false positive results particularly in those with a history of *H. pylori* eradication treatment.[27] However, to reduce this error, we had already excluded individuals with a history of *H. pylori* eradication treatment. In addition, the fact that we did not consider some confounding factors such as physical activity and smoking status in this study might weakened our findings regarding to metabolic parameters and therefore should be regarded as a limitation.

CONCLUSION

The present study showed that in a small population of young students in southeastern of Iran, *H. pylori* infection was only associated with low consumption of raw vegetables and higher serum blood glucose levels after adjusting for other parameters. It is recommended that future researches examine the interaction between *H. pylori* infection and metabolic parameters according to different genotypes of *H. pylori* in Iran as well as the host genetic factors.

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Conflicts of interest

There are no conflicts of interest.

AUTHORS' CONTRIBUTION

- OE contributed in the conception of the work, conducting the study, writing the manuscript, approval of the final version of the manuscript, and agreed for all aspects of the work,
- MSh contributed in the conception and design of the work, revising the draft,approval of the final version of the manuscript, and agreed for all aspects of the work,
- TSh contributed in the conception of the work , revising the draft, approval of the final version of the manuscript, and agreed for all aspects of the work,

- HA contributed in the conception of the work, analyzing the data, approval of the final version of the manuscript, and agreed for all aspects of the work.

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