Middle-East Journal of Scientific Research 28 (3): 193-198, 2020
ISSN 1990-9233

DOI: 10.5829/idosi.mejsr.2020.193.198

Helicobacter Pylori Infection and Body Mass Index in Fayoum Governorate, Egypt

Safaa Khamis Hassan

Public Health and Community Medicine Department,
Faculty of Medicine, Fayoum University, Egypt

Abstract: *Helicobacter pylori* is a highly recurrent infection all over the world, more than half of the population worldwide is infected with this microorganism. Most infections are not associated with the clinical disease; a significant proportion will go on to develop chronic gastritis, peptic ulcer diseases. The study aimed to identify a possible association of *H. pylori* infection and body mass index. A descriptive cross-section study was conducted using a pre-designed interviewer-administered questionnaire. Results: showed that there was a significant difference between *H.pylori* positive and negative antigen in relation to age, residence, BMI, (p- value= 0.034, p-value =0.005, p- value=0.000 respectively). *H. pylori* positive antigen showed an association with, rural residence, increasing total cholesterol and underweight and body mass index ≥ 35 kg/m^2 Conclusion: There were a high *H. pylori* infection in adults with low BMI and very high body mass index compared to those with a normal BMI and the overweight.

Key words: *H. pylori* · Body Mass Index · Fayoum · Egypt

INTRODUCTION

Helicobacter pylori is a Gram-negative organism living in the human stomach and linked to many gastrointestinal diseases [1]. It is a highly prevalent infection all over the world; more than half of the population worldwide is infected with this microorganism [2, 3].

Many people acquired *H. pylori* infection during their early childhood and this is depending on the country, age, socioeconomic background, nutritional status, urbanization and hygiene [4, 5]. Most cases of *H. pylori* infection are not associated with clinical disease, but a significant proportion will develop chronic gastritis, peptic ulcer diseases and less than 1% of infected patients will develop gastric cancer [6, 7].

In EMRO countries, the overall prevalence of *H. pylori* infection irrespective of time and age ranged from 22 to 87.6% [8]. Egypt had the highest prevalence of *H. pylori* in the healthy asymptomatic population both in adults and the pediatric population [9, 10]. A high prevalence of *H. pylori* infections has been reported, ranging from 70% in the general population [11], 73% among school children [9], up to 88% in patients with chronic active HCV [12].

Obesity is a public health concern worldwide. The World Health Organization (WHO), estimated that over 600 million people in the world were obese in the year 2014. Obesity is associated with many infectious diseases, such as human adenovirus 36, H1N1/influenza virus, human immunodeficiency virus, and *H. pylori* [13]. The association between obesity and *H. pylori* infection is controversial; a lower prevalence of *H. pylori* infection was found in obese patients than in the general population [14]. Other studies have reported a higher *H. pylori* prevalence in obese patients [15, 16]. The study aimed to determine the possible association between *H. pylori* and BMI.

MATERIALS AND METHODS

Study Design and Setting: A descriptive cross-sectional study was conducted among individuals aged from 17 to 65 years who came to modify their bodies and lifestyles without a history of past *H. pylori* infection or taking medication for *H. pylori* infection at the outpatient food clinic in Fayoum governorate from April 2018 to May 2019. Informed consent was obtained from each subject. A purposive sampling technique was used to select our participants.

Corresponding Author: Safaa Khamis Hassan, Public Health and Community Medicine Department,
Faculty of Medicine, Fayoum University, Egypt.

Data Collection Tools: All subjects were interviewed by a pre-designed interviewer-administered questionnaire including the following items; sociodemographic data (age, sex, residence, occupation, and family income level meets the expenses (yes and save considered high-income level), yes considered moderate-income level, (sometimes or no considered low-income level) [17].

Anthropometric measurements (weight and height) were measured for each individual who completed the questionnaire. Weight was measured to the nearest 0.1 kg through an electronic scale, the participant was wearing light clothing and without shoes. The height was measured to the nearest 0.1 cm using a wooden stadiometer placed on a flat surface.

BMI was calculated depending on the following equation; weight in kilograms divided by the square of height in meters.` Underweight" was defined as BMI lower than 18.5 kg/m^2; "normal" 18.5 to 24.9 kg/m^2; "Overweight 25 to 29.9 kg/m^2, class1 obesity 30 to 34.9 kg/m^2 class11 obesity from 35 to 39. 9 kg/m^2 and class111 obesity (morbid) ≥ 40 kg/m^2

[18]. Data was collected on; fasting blood lipid profile i.e. serum cholesterol & triglycerides and the results of the *H. pylori* test. *H. pylori* were diagnosed using a stool antigen test which is rapid, non-invasive, reliable and easy to perform and can be used to detect an existing infection [19].

Sample Size Calculation: The sample size was determined for the study using Open Epi, Version 3, It was calculated based on the prevalence of 50% and a design effect of 2 and confidence limit 80% ±5%. Thus the least required sample size was 329 increased by 10% to avoid unresponsive rate 362.

Ethical Approval and Consent to Participate: The study protocol was approved by the ethics committee of the Faculty of Medicine, Fayoum University that complies with guidelines of the World Medical Association Declaration of Helsinki. Informed written consents were obtained from all patients or their legal guardians

Statistical Analysis: Data were collected, coded and analyzed using Statistical Package for Social Science (SPSS) software version 16. Simple descriptive analysis in the form frequencies and percentages were calculated for numerical data. Non-parametric tests (chi-square and Mann-Whitney U) and Binary logistic regression were used to find its association with other factors. $P < 0.05$ was considered statistically significant.

Table 1: Clinical and demographic characteristics of the studied participants (N=362)

Characters	N (362)	%
Sex		
Male	129	35.6
Female	233	64.4
Age		
≤20	66	18.2
21-40 years old	174	48.1
41-60	101	27.9
>60 years old	21	5.8
Residence		
Rural	156	43.1
Urban	206	56.9
Family income status (meets the expenses)		
Low	62	17.1
Moderate	212	58.6
High	33	24.3
BMI		
Less than 18.5	46	12.7
18.5 to 24.9	84	23.2
25-29.0	33	9.1
30-34.9	90	24.9
35-39.9	84	23.2
≥40	25	6.9
Total cholesterol		
≤200	273	75.4
>200	89	24.6
Triglycerides		
≤150	327	90.3
>150	35	9.7
Fasting blood glucose		
<126	334	92.3
≥126	28	7.7
H.pylori infection	137	37.8

RESULTS

A total of 362 individuals were included in the study. Of these 233 (64.5) were females and 129 (35.6%) were males, near half of them 174 (48.1%) were between the age of 21 to 40 years old. More than half of the 206 (56.9%) were urban people. 46 (12.7%) were underweight BMI ≥18.5 kg/m^2 and 25 (6.9%) were with morbid obesity BMI ≥40 kg/m^2, 89 (24.6%) with a high cholesterol level, 35 (9.7) with increased TG level and 28 (7.7%) were diabetic (Table 1).

There was no statistically significant difference between *H. pylori*-negative and *H. pylori*-positive concerning sex and family income level. While there was a significant difference in relation to age, residence, BMI, (p value= 0.034, p-value =0.005, p value=0.000 respectively) (Table 2). The HPPA showed a higher median level of cholesterol, FBS and TG compared to HPNA. There was a significant association with cholesterol level only p value=0.001 (Table 3).

Table 2: Demographic characteristics and BMI of the participants according to *H. pylori* antigen state

Characters	Total number	Negative HP	Positive HP	X^2	P-value
Sex					
Males	129	86 (66.7)	43(33.3)		
Females	233	139(59.7)	94(40.3)	1.735	0.188
Age					
≤20 years	66	32 (48.1)	34(51.5)		
21-40	174	112 (64.4)	62 (35.6)		
41-60	101	70 (69.3)	31 (30.7)		
>60 years old	21	11(52.4)	10 (47.6)	8.655	0.034*
Residence					
Rural	156	84(53.8)	72(46.2)		
Urban	206	141(68.4)	65 (31.6)	8.045	0.005*
Economic level					
Low	62	36(58.1)	26(41.9)		
Moderate	212	134(63.2)	78 (36.8)		
High	88	55(62.5)	33 (37.5)	0.545	0.761
BMI kg/m^2					
<18.5	35	7(20.0)	28(80.0)		
18.5-24.9	83	74(89.2)	9(10.8)		
25-29.9	32	30(93.8)	2(6.2)		
30-34.9	90	72(80)	18(20)		
35-39.9	94	38(40.4)	56(59.6)		
≥40	28	4(14.3)	24(85.7)	1.241	0.000*

Statistically significant*

Table 3: Comparison between HPPA and HPNA according to the median level of total cholesterol, TG and FBG

	Negative HP		Positive HP			
	Median	IQR	Median	IQ range	Z	P-value Mann-Whitney U
Cholesterol	160	58 (186-129)	168	72 (210-138)	-3.176	0.001*
FBG	79	14(90-76)	87	38 (69-107)	-0.967	0.333
Triglycerides	70	35(91-57)	74	43(103-60)	-1.285	0.199

Statistically significant*

Table 4: Logistic regression of possible predictors of H.pylori positive antigen

Predicators	P- value	OR (95% CI)
Sex	0.866	1.051 (0.591-1.867)
Age	0.496	0.993 (0.972-1.014)
Residence (rural)	0.001*	2.562(1.461-4.493)
Socioeconomic	0.990	0.997(0.653-1.523)
Fasting blood glucose	0.067	1.010 (0.999-1.020)
Total Cholesterol	0.021*	1.008 (1.001-1.015)
Increasing triglycerides	0.757	1.001(0.994-1.008)
Body mass index	0.000*	
<18.5	0.000	26.040(9.540-71.079)
≥35	0.000	11.146 (6.242-19.901)

Statistically significant*

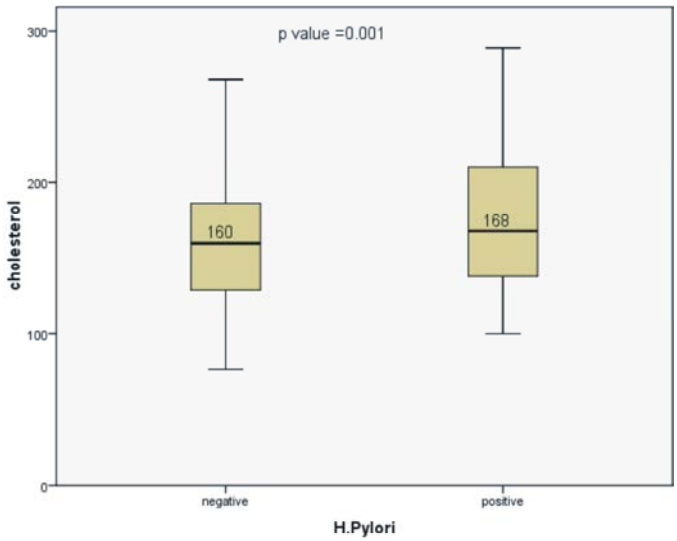

Fig. 1: Cholesterol level according to HP antigen state

H. pylori positive antigen showed an association with, rural residence, increasing total cholesterol and underweight and body mass index ≥ 35 kg/m^2 were reported as positive H pylori risk factors with OR (95% CI); 2.562(1.461-4.493), 1.008 (1.001-1.015)), 1.011 (1.002-1.019)and26.040(9.540-71.079),11.146(6.242-19.901) respectively (Table 4). Fig. 1 shows cholesterol level in relation to H. pylori positive (median and IQ 168, IQ= 72) and H.pylori negative (median =160, IQ=58).

DISCUSSION

The infection of *H. pylori* is largely diffused, which may reach in developing countries (up to 80%) only 10 -20% of patients develop clinical manifestations [20]. In Egypt, a study in a rural area revealed a seropositivity rate of 91.7% of the rural population. The rate of infection was different in different age groups with an increasing trend in older ages [21].

In the following study, the proportion of the individuals with *H. pylori* positive antigen was 137/362 (37.8%), with more prevalence among rural people compared to urban and with different distribution among different age groups with more frequency among young people ≤20 y and older population >60 years with significant difference (p=0.034) this may be attributed to the conditions that increase the risk of infections such as, bad hygiene, lack of sanitation especially many of the rural areas in Fayoum governorate are lacking waste disposal system or access for sanitary water supply or a system of infection control procedures especially for food shops.

The proportion of HPPA was higher among low socioeconomics but without a significant association and among females than males, however, several studies reported an association between low socioeconomic, rural residence, increasing age and male gender with HPPA infection [22-24], This may be attributed to the different methodology and environment and that most clients to adjust their body weight, are female.

The results of the association of HPPA and body mass index in many studies are controversial. Our findings of the study associated the *H. pylori* infection to the very low body mass index by (80%) and the very high body mass index among class 111 obesity (59.6%) and morbid obesity (85.7%), compared to normal and high body mass index with significance p-value =0.000, while In Turkey prevalence of 57.2% of HPPI in Turkish obese subjects compared to 27.0% in normal body weight [25].

Other studies, revealed that the prevalence of *H. pylori* positive antigens was higher in obese patients 208/399 (52%) compared to overweight 111/399; 28% (p=0.001) [26]. However, a recent review showed a significant inverse correlation between H. pylori prevalence and the rate of overweight/obesity in countries of the developed world. Thus, the gradual decrease of the *H. pylori* colonization was attributed to obesity endemic observed in the western world [27].

H. pylori infection could induce insulin resistance; disturb glucose and lipid homeostasis and metabolism of adipocytokines [28, 29]. In our findings, the HPPA was associated with a higher level of cholesterol, fasting blood sugar and triglycerides but with a significant difference concerning cholesterol level only (p value=0.001). In previous studies regarding the

association of *H. pylori*, infection with lipid metabolism showed relatively consistent evidence, but conflicting results also exist [30, 31]. An association between *H. pylori* infection and type 2 diabetes (P = 0.001) was reported [24] while others provided evidence that *H. pylori* infection is associated with dyslipidemia such as higher total cholesterol and LDL-C, as well as lower HDL-C, but with no association with glucose level [32].

Low socioeconomic status, low body weight and height, living in rural areas and lower educational status were risk factors for the acquisition of *H. pylori* in Egyptian studies [9]. In our study the predictors of HPPA were a rural residence, high cholesterol level, low body mass index<18.5 kg/m^2 and high body mass index ≥ 35 kg/m^2, others reported higher total cholesterol level, lower HDL-C level and higher diastolic blood pressure were the predicators of HPPA [31], Others reported the age 31-50 years, BMI > 23.1 kg/m^2 and type 2 diabetes were risk factors for *H. pylori* [24].

CONCLUSIONS

There was a high *H. pylori* infection in adults with low BMI and a very high body mass index compared to those with a normal BMI and the overweight. *H. pylori* were significantly associated with rural residences, younger age and higher cholesterol levels in our population. A large community-based study is recommended to further understand these associations at a general population level.

Limitation:

- It's not a community-based study.
- Purposive and relatively small sample size.
- Waist circumference, presenting symptoms of H pylori infection and LDL, HDL was not evaluated.

REFERENCES

1. Kuo, F.C., C.Y. Wu, C.H. Kuo, C.F. Wu, Y.H. Chen, C.Y. Chen, Y.C. Lo, M.T. Wu and H.M. Hu, 2014. The utilization of a new immunochromatographic test in the detection of Helicobacter pylori antibody from maternal and umbilical cord serum," BioMed Res. Int. Article ID 568410/6 pages/ https:// doi.org/ 10.1155/ 2014/ 568410
2. Darko, R., A.E. Yawson, V. Osei, J. Owusu-Ansah and S. Aluze-Ele, 2015. Changing patterns of the prevalence of Helicobacter pylori among patients at a corporate hospital in Ghana, Ghana. J. Medical., 49(3): 147-153.
3. Dane, A. and T. Gurbuz, 2016. Clinical comparative study of the effects of Helicobacter pylori colonization on oral health in children. Pakistan J. Medical Sciences, 32(4): 969-973.
4. Nagy, P., S. Johansson and M. Molloy-Bland, 2016. Systematic review of time trends in the prevalence of Helicobacter pylori infection in China and the USA. Gut Pathog. 8:8. doi:10.1186/s13099-016-0091-7
5. Malfertheiner, P., F. Megraud, C. O'morain, F. Bazzoli, E. El-Omar, D. Graham, R. Hunt, T. Rokkas, N. Vakil and E.J. Kuipers, 2007. Current concepts in the management of Helicobacter pylori infection: the Maastricht III Consensus Report. Gut., 56: 772-781.
6. Mandeville, K.L., J. Krabshuis, N.G. Ladep, C.J.J. Mulder, E.M.M. Quigley and S.A. Khan, 2009. Gastroenterology in developing countries: issues and advances. World J. Gastroenterol., 15: 2839-2854.
7. Delgado, J.S., E. Gené, D. Suárez, P. Garcia-Iglesias, E. Brullet and M. Gallach, 2011. Has *H. pylori* prevalence in bleeding peptic ulcer been underestimated? A meta-regression. Am. J. Gastroenterol., 106: 398-405.
8. Eshraghian, A., 2014. Epidemiology of Helicobacter pylori infection among the healthy population in Iran and countries of the Eastern Mediterranean Region: A systematic review of prevalence and risk factors, World J. Gastroenterol., 20(46): 17618-17625.
9. Mohammad, M.A., L. Hussein, A. Coward and S.J. Jackson, 2008. Prevalence of Helicobacter pylori infection among Egyptian children: impact of social background and effect on growth. Public Health Nutr., 11: 230-236.
10. Bassily, S., R.W. Frenck, E.W. Mohareb, T. Wierzba, S. Savarino, E. Hall, A. Kotkat, A. Naficy, K.C. Hyams and J. Clemens, 1999. Seroprevalence of Helicobacter pylori among Egyptian newborns and their mothers: a preliminary report. Am J. Trop. Med. Hyg., 61: 37-40.
11. Ghaith, D., M. Elzahry, G. Mostafa, S. Mostafa, R. Elsherif and I. Ramzy, 2016. Mutations affecting domain V of the 23S rRNA gene in *Helicobacter pylori* from Cairo, Egypt. J. Chemother., 28: 367-370.
12. Hanafy, A.S., A.T. El Hawary, E.F. Hamed and A.M. Hassaneen, 2016. Impact of Helicobacter pylori eradication on refractory thrombocytopenia in patients with chronic HCV awaiting antiviral therapy. Eur. J. Clin. Microbiol. Infect. Dis. [Internet]. Springer Berlin Heidelberg, 35: 1171-6.
13. Dhurandhar, N.V., D. Bailey and D. Thomas, 2015. Interaction of obesity and infections. Obes. Rev., 16: 1017-1029.

14. Carabotti, M., C. D'Ercole, A. Iossa, E. Corazziari, G. Silecchia and C. Severi, 2014. Helicobacter pylori infection in obesity and its clinical outcome after bariatric surgery. World J. Gastroenterol., 20: 647-653.
15. Arslan, E., H. Atilgan and I. Yavaşoğlu, 2009. The prevalence of Helicobacter pylori in obese subjects. Eur J. Intern. Med., 20: 695-697.
16. Dutta, S.K., M. Arora, A. Kireet, H. Bashandy and A. Gandsas, 2009. Upper gastrointestinal symptoms and associated disorders in morbidly obese patients: a prospective study. Dig. Dis. Sci., 54: 1243-1246.
17. El-Gilany, A., A. El-Wehady and M. El-Wasify, 2012. Updating and validation of the socioeconomic status scale for health research in Egypt. East. Mediterr. Health J., 18(9): 962-968.
18. Sturm, R., 2007. Increases in morbid obesity in the USA: 2000-2005. Public Health, 121(7): 492-6.
19. Kazemi, S., H. Tavakkoli, M.R. Habizadeh and M.H. Emami, 2011. Diagnostic values of Helicobacter pylori diagnostic tests: stool antigen test, urea breath test, rapid urease test, serology and histology. J. Res. Med. Sci., 16(9): 1097-104.
20. Malfertheiner, P., F.K. Chan and K.E. McColl, 2009. Peptic ulcer disease. Lancet., 374: 1449-1461.
21. El Dine, S.S., M. Mubarak, R. Salama, M.E. Raziky, E. El Sherbiny, S. Zakaria and M.S. Zakaria, 2008. Low seroprevalence of anti-CagA antibodies in spite of high seroprevalence of anti-H pylori antibodies in the rural Egyptian community. Res. J. Med. Sci., 3: 118-123.
22. Moayyedi, P., A.T.R. Axon, R. Feltbower, S. Duffett, W. Crocombe, D. Braunholtz, G. Richards and D. Forman, 2002. Relation of adult lifestyle and socioeconomic factors to the prevalence of Helicobacter pylori infection international J. Epidemiol., 31(3): 624-631.
23. Chen, H.L., M.J. Chen, S.C. Shih, H.Y. Wang, L.T. Lin and M.J. Bair, 2014. Socioeconomic status, personal habits and prevalence of Helicobacter pylori infection in the inhabitants of Lanyu. The Formosan J. Medical. Associ., 113(5): 278-283.
24. Siddiqui, B., J. Yakoob, Z. Abbas, R. Azmat, S.S. Fatima and S. Awan, 2018. Distribution of Helicobacter pylori infection and abnormal body-mass index (BMI) in a developing country J. Infect Dev. Ctrie., 12(5): 342-346.
25. Arslan, E., H. Atilgan and I. Yavasoglu, 2009. The prevalence of Helicobacter pylori in obese subjects. Eur. J. Intern. Med., 20: 695-697.
26. Al-Zubaidi, A.M., A.H. Alzobydi, S.A. Alsareii, A.T. Al-Shahrani, N. Alzaman and S. Kassim, 2018. Body Mass Index and Helicobacter pylori among Obese and Non-Obese Patients in Najran, Saudi Arabia: A Case-Control StudyInt J Environ Res Public Health 15(11): 2586.
27. Lender, N., N.J. Talley, P. Enck, S. Haag, S. Zipfel, M. Morrison and G.J. Holtmann, 2014. Review article: Associations between Helicobacter pylori and obesity-An ecological study. Aliment. Pharmacol. Ther., 40: 24-31.
28. Gunji, T., N. Matsuhashi, H. Sato, K. Fujibayashi, M. Okumura, N. Sasabe and A. Urabe, 2009. Helicobacter pylori infection significantly increases insulin resistance in the asymptomatic Japanese population. Helicobacter., 14: 144-150.
29. Gen, R., M. Demir and H. Ataseven, 2010. Effect of Helicobacter pylori eradication on insulin resistance, serum lipids and low-grade inflammation. South Med. J., 103: 190-196.
30. Chen, T.P., H.F. Hung, M.K. Chen, H.H. Lai, W.F. Hsu, K.C. Huang and K.C. Yang, 2015. Helicobacter Pylori Infection is Positively Associated with Metabolic Syndrome in Taiwanese Adults: a Cross-Sectional Study. Helicobacter, 20(3): 184-191.
31. Satoh, H., Y. Saijo, E. Yoshioka and H. Tsutsui, 2010. Helicobacter Pylori infection is a significant risk for modified lipid profile in Japanese male subjects. J. Atheroscler Thromb., 17: 1041-1048.
32. Kim, T.J., H. Lee, M. Kang, J.E. Kim, Y.H. Choi, Y.W. Min, B.H. Min, J.H. Lee, H.J. Son, P.L. Rhee, S.Y. Baek, S.H. Ahn and J.J. Kim, 2016. Helicobacter pylori is associated with dyslipidemia but not with other risk factors of cardiovascular disease Scientific Repo Rts., 6: 38015:1-8.