ACTA FACULTATIS
MEDICAE NAISSENSIS

UDC: 613.25:[616.33-022.7:579.84
DOI: 10.5937/afmnai1904310A

Original article

Helicobacter Pylori in Obese Females

Abdulhadi Alrubaie[1], Maytham Al-Qanbar[2], Raed Rassam[3]

[1]*Head of Obesity Research and Therapy Unit, Alkindy College of Medicine/University of Baghdad, Iraq*
[2]*Departmentof Surgery, College of Medicine University of Kufa, Iraq*
[3]*Alkindy College of Medicine, University of Baghdad, Iraq*

SUMMARY

Obesity is an increasing health problem in developing countries and has grown into a major global epidemic. Recent studies suggest that colonization of the stomach with *Helicobacter pylori (H. pylori)* may affect gastric expression of appetite and satiety-related hormones, finding that patients cured of *H. pylori* infection have gained weight. Further exploration of the relationship between obesity and *H. pylori* infection is therefore warranted.

The objective of this study was to determine the prevalence of H. pylori infection in a sample of obese patients. A total of 69 obese females and 55 normal-weight females as a control group were included. Body mass indices (BMI) of all females were measured and tests for *H. pylori* performed. Subjects ranged in age from 20 to 59 years. The threshold for classification of obesity was a BMI of 30 kg/m² or higher. Fifty obese females (72.5%) were *H. pylori* positive. Thirty-two (58.2%) normal-weight females were seropositive for *H. pylori*, which was not significant ($p > 0.05$) when compared with obese group. All the obese above 45 years of age were found to be *H. pylori* positive.

The study showed that there was no significant relation between *H. pylori* infection and obesity. Rather, the prevalence of *H. pylori* infection increases with age.

Key words: **obesity, *Helicobacter pylori*, BMI**

Corresponding author:
Abdulhadi Alrubaie
E-mail: hadirubaie@yahoo.com

INTRODUCTION

Obesity is a major public health problem of concern globally. In the United States alone, more than 60% of adults are overweight or obese with significant impact on health and economy (1, 2). Obesity in adults is defined as a BMI of at least 30 kg/m²; overweight is defined as a BMI of 25 kg/m² or higher (3). Increases in calorie intake, diet composition and declining levels of physical activity may interact with changes in the gut microbiome to contribute to obesity and overweight (4). Obesity is linked to an increased risk of various health problems, including hypertension, diabetes mellitus type 2, dyslipidemia, ischemic heart disease, stroke, asthma, and arthritis (5, 6).

Helicobacter pylori, a gram-negative spiral-shaped bacteria found on gastric mucosa, was first isolated by the Nobel Prize winners Robin Warren and Barry Marshall (6). About half of the world population is predicted to be infected with *H. pylori* bacteria. Peptic ulcer diseases, atrophic gastritis, and gastric cancer could be caused by chronic infection with *H. pylori* bacteria (7, 8). Studies have suggested its role in the pathogenesis of systemic disorders like Reynaud phenomena, migraine and cardiovascular disease (9-11).

The interplay between *H. pylori* and obesity is not yet fully understood. While some data have revealed that eradication of this bacteria increases BMI in patients treated for peptic ulcer disease (12, 13), another study found that the risk of *H. pylori* infection does not increase in overweight young people (14). Obesity is considered a risk factor for gastroesophageal reflux disease (GERD), and the occurrence of GERD after *H. pylori* eradication may be due to the restoration of acid secretion by the gastric mucosa (15, 16).

Increase in appetite and weight gain has been reported after *H. pylori* eradication in asymptomatic subjects, and these findings may explain the increasing obesity seen in the western population where *H. pylori* prevalence is low (17).

METHODS

Between May 2014 and April 2018, 69 randomly selected females with obesity (BMI 30 kg/m² or higher) attended the obesity research and therapy unit at Alkindy College of Medicine University of Baghdad. Fifty-five normal-weight females were included in the study as a control group.

This study was approved ethically by the scientific affairs unit of Alkindy College of Medicine, University of Baghdad.

Laboratory assessment

The enzyme-linked immunosorbent assay kit (Acon, USA), a noninvasive serological assay with a sensitivity and specificity higher than 95% for the detection of *Helicobacter pylori* infection, was used in this study to determine the presence of *H. pylori*(18, 19).

Statistical analysis

Statistical analysis was performed by the use of IBM SPSS 23.0.

RESULTS

The age ranges for all female participants were 20 to 59 years. Fifty out of 82 (72.5%) obese females were *H. pylori* positive and 32 (58.2%) normal-weight females were seropositive for *H. pylori* (Table 1), a difference which was not statistically significant (P = 0.09).

Table 1. **Seroprevalence of *H. pylori* in relation to weight**

Weight	*H. pylori*		Total
	Negative	Positive	
Normal weight	23 (41.8%)	32 (58.2%)	55
Obese	19 (27.5%)	50 (72.5%)	69
Total	42 (33.9%)	82 (66.1%)	124

Table 2. **Seropositivity of *H. pylori* in respect to age and weight in normal and obese females**

Age /Years	Weight	*H. pylori*		Total
		negative	positive	
20-29	normal	10 (71.4%)	4 (28.6%)	14
	obese	14 (77.8%)	4 (22.2%)	18
	total	24 (75.0%)	8 (25.0%)	32
30-39	normal	6(37.5%)	10 (62.5%)	16
	obese	3 (15.0%)	17 (85.0%)	20
	total	9 (25.0%)	27 (75.0%)	36
40-49	normal	5 (35.7%)	9 (64.3%)	14
	obese	2 (11.1%)	16 (88.9%)	18
	total	7 (21.9%)	25 (78.1%)	32
50-59	normal	2 (18.2%)	9 (81.8%)	11
	obese	0 (0.0%)	13 (100.0%)	13
	total	2 (8.3%)	22 (91.7%)	24
Total	normal	23 (41.8%)	32 (58.2%)	55
	obese	19 (27.5%)	50 (72.5%)	69
	total	42 (33.9%)	82 (66.1%)	124

According to different age groups, the seropositivity of *H. pylori* for obese females was 25%, 75%, 78.1% and 91.7% for ages 20-29, 30-39, 40-49 and 50-59 years, respectively, which was not statistically significant (P > 0,05) when compared with the control group, but it is statistically significant when the acquisition of *H. pylori* with advancing age P < 0.05 (Table 2) is calculated.

DISCUSSION

Recent research has drawn attention to the role of *H. pylori*-associated gastritis in pathogenesis of nutritional problems and body weight, due to its supposed role and effects on the levels of gastric hormone leptin produced by chief cells (20).

Numerous studies have shown that persons infected with *H. pylori* produce lower amounts of ghrelin and its eradication lead to increase ghrelin production (21, 22).

Prevalence of *H. pylori* infection is related to multiple factors, such as age and socioeconomic status. Acquiring of infection in developing countries seems to be nearly universal (23).

In this study, we found that most of *H. pylori* positive cases (85%) were above 30 years of age, and 100% of obese patients above 50 years of age were *H. pylori* positive. If we compare the prevalence of *H. pylori* in obese females in this study with a study done by Nawafal et al. at North of Iraq, similar results were found in about 78% of the general population above the age of 40 being positive for *H. pylori* (24). In the nearby countries, such as Turkey and Iran, the seropositivity for *H. pylori* is 80.0% and 88.4% – 93%, respectively, while in Egypt seropositivity has been reported in 90.0% and in Algeria up to 92%.

The high seropositivity rates in developing countries are in stark contrast to rates in developed countries. For example, in the United States seropositivity has been reported upto 30.0%, in Canada 23.1% and in Germany 48.8%.

Helicobacter pylori through its potential effects on leptin hemostats may play a role in regulating the body weight. In the United States, where obesity is considered an important health problem, a large population-based study found no significant correlation between *H. pylori* status and being overweight (25). Our findings appear to confirm this in a population with much higher seropositivity rates.

The role of *H. pylori* infection in morbidly obese

patients is still controversial. Generally, the existing studies found that obese patients had a lower prevalence of *H. pylori* infection than the general population (26). Adipose tissue synthesizes the hormone leptin, which is now known to be produced by the stomach as well, and is involved in the regulation of satiety, food intake and body weight. *H.pylori* induces gastric inflammation, which may affect gastric leptin levels secretion and influence food intake and body weight (21).

Nweneka et al. stated that people infected with *H. pylori* have lower circulating ghrelin concentration than non-infected individuals. In addition, *H. pylori* eradication has no significant effects on ghrelin concentration (27).

Weight gain following *H. pylori* eradication has been demonstrated in numerous studies. Zuma et al. stated that eradication of *H. pylori* is associated with reduced gastric leptin levels followed by weight gain, though serum leptin levels remain unchanged (28).

CONCLUSION

In conclusion, our study found that there was no significant association between *H.pylori* infection and obesity, but rather that the acquiring of *H.pylori* bacterium was significantly related to the advancing age.

The authors declare that there is no conflict of interest.

References

1. Fryar CD, Carroll MD, Ogden CL. Prevalence of overweight, obesity, and extreme obesity among adults: United States, trends 1960-1962 through 2009-2010. Hyattsville, MD: National Center for Health Statistics. 2012 .

2. Flegal KM, Carroll MD, Ogden CL et al. Prevalence and trends in obesity among US adults, 1999-2008. JAMA 2010;20:235-41. https://doi.org/10.1001/jama.2009.2014

3. Ogden CL, Carroll MD, Curtin LR et al. Prevalence of overweight and obesity in the United States, 1999-2004. JAMA 2006; 13:1549-55. https://doi.org/10.1001/jama.295.13.1549

4. Prentice A, Jebb S. Energy intake/physical activity interactions in the homeostasis of body weight regulation. Nutrition Reviews 2004; 62:98-104. https://doi.org/10.1301/nr.2004.jul.S98-S104

5. Hammond RA, Levine R. The economic impact of obesity in the United States. Diabetes Metab Syndr Obes 2010; 3: 285-95. https://doi.org/10.2147/DMSO.S7384

6. Warren JR, Marshall B. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. The Lancet 1983 ;321:1273-5. https://doi.org/10.1016/S0140-6736(83)92719-8

7. Cover TL, Blaser MJ. Helicobacter pylori in health and disease. Gastroenterology 2009; 136:1863-73. https://doi.org/10.1053/j.gastro.2009.01.073

8. Parsonnet J. Helicobacter pylori and gastric cancer. Gastroenterol Clin N 1993; 22:89-104.

9. Saijo Y, Utsugi M, Yoshioka E et al. Relationship of Helicobacter pylori infection to arterial stiffness in Japanese subjects. Hypertension Research. 2005:28:283. https://doi.org/10.1291/hypres.28.283

10. Rogha M, Dadkhah D, Pourmoghaddas Z et al. Association of helicobacter pylori infection with severity of coronary heart disease. ARYA Atherosclerosis 2012; 7:138.

11. Fujiwara Y, Higuchi K, Arafa UA et al. Long-term effect of Helicobacter pylori eradication on

quality of life, body mass index, and newly developed diseases in Japanese patients with peptic ulcer disease. Hepato-Gastroenterology 2002; 49:1298-302.

12. Kamada T, Hata J, Kusunoki H et al. Eradication of Helicobacter pylori increases the incidence of hyperlipidaemia and obesity in peptic ulcer patients. Digest Live Dis 2005; 37:39-43. https://doi.org/10.1016/j.dld.2004.07.017

13. Labenz J, Blum AL, Bayerdorffer E et al. Curing Helicobacter pylori infection in patients with duodenal ulcer may provoke reflux esophagitis. Gastroenterology 1997; 112:1442-7. https://doi.org/10.1016/S0016-5085(97)70024-6

14. Kyriazanos ID, Sfiniadakis I, Gizaris V et al.The incidence of Helicobacter pylori infection is not increased among obese young individuals in Greece. J Clin Gastroenterol 2002; 34:541-6. https://doi.org/10.1097/00004836-200205000-00012

15. Locke III GR, Talley NJ, Fett SL et al. Risk factors associated with symptoms of gastroesophageal reflux. AM J Med 1999; 106:642-9. https://doi.org/10.1016/S0002-9343(99)00121-7

16. Koike T, Ohara S, Sekine H et al. Increased gastric acid secretion after Helicobacter pylori eradication may be a factor for developing reflux oesophagitis. Aliment Pharm Ther 2001; 15:813-20. https://doi.org/10.1046/j.1365-2036.2001.00988.x

17. Nwokolo CU, Freshwater DA, O'hare P et al. Plasma ghrelin following cure of Helicobacter pylori. Gut 2003; 52:637-40. https://doi.org/10.1136/gut.52.5.637

18. Talley NJ, Newell DG, Ormand JE et al. Serodiagnosis of Helicobacter pylori: comparison of enzyme-linked immunosorbent assays. J Clin Microbiol 1991; 29:1635-9.

19. Van de Wouw BA, de Boer WA, Jansz AR et al. Comparison of three commercially available enzyme-linked immunosorbent assays and biopsy-dependent diagnosis for detecting Helicobacter pylori infection. J Clin Microbiol 1996; 34:94-7.

20. Konturek PC, Czesnikiewicz-Guzik M, Bielanski W et al. Involvement of Helicobacter pylori infection in neuro-hormonal control of food intake. J Physiol Pharmacol 2006; 57Suppl:67-81.

21. Isomoto H, Nakazato M, Ueno H et al. Low plasma ghrelin levels in patients with Helicobacter pylori-associated gastritis. Am J Med 2004;117 :429-32. https://doi.org/10.1016/j.amjmed.2004.01.030

22. Nwokolo CU, Freshwater DA, O'hare P et al. Plasma ghrelin following cure of Helicobacter pylori. Gut 2003; 52:637-40. https://doi.org/10.1136/gut.52.5.637

23. Bardhan PK. Epidemiological features of Helicobacter pylori infection in developing countries. Clin Infect Dis 1997; 25:973-8. https://doi.org/10.1086/516067

24. Hussein NR, Robinson K, Atherton JC. A study of Age-Specific Helicobacter pylori Seropositivity Rates in Iraq. Helicobacter 2008; 13:306-7. https://doi.org/10.1111/j.1523-5378.2008.00618.x

25. Cho I, Blaser MJ, François F et al. Helicobacter pylori and overweight status in the United States: data from the Third National Health and Nutrition Examination Survey. Am J Epidemiol 2005; 162:579-84. https://doi.org/10.1093/aje/kwi237

26. Carabotti M, D'Ercole C, Iossa A et al. Helicobacter pylori infection in obesity and its clinical outcome after bariatric surgery. World J Gastroentero 2014; 20:647-53. https://doi.org/10.3748/wjg.v20.i3.647

27. Nweneka CV, Prentice AM. Helicobacter pylori infection and circulating ghrelin levels-a systematic review. BMC Gastroenterology 2011; 11:7. https://doi.org/10.1186/1471-230X-11-7

28. Azuma T, Suto H, Ito Y, Ohtani M et al. Gastric leptin and Helicobacter pylori infection. Gut 2001; 49:24-9. https://doi.org/10.1136/gut.49.3.324

Prisustvo bakterije *Helicobacter pylori* kod gojaznih žena

Abdulhadi Alrubaie[1], Maytham Al-Qanbar[2], Raed Rassam[3]

[1]*Načelnik odeljenja za ispitivanje gojaznosti i terapiju, Koledž medicinskih studija Alkindy, Univerzitet u Bagdadu, Irak*
[2]*Odeljenje za hirurgiju, Koledž medicinskih studija, Univerzitet u Kufi, Irak*
[3]*Koledž medicinskih studija Alkindy, Univerzitet u Bagdadu, Irak*

SAŽETAK

Gojaznost postaje sve veći problem u zemljama u razvoju i dobija razmere globalne epidemije. Rezultati nedavnih studija pokazali su da kolonizacija želuca bakterijom *Helicobacter pylori* (*H. pylori*) može da utiče na apetit i hormone koji regulišu osećaj sitosti, i da bolesnice koje se izleče od ove infekcije dobiju na težini. Dalje ispitivanje odnosa između gojaznosti i infekcije izazvane ovom bakterijom je neophodno.

Cilj ove studije bilo je određivanje prevalencije infekcije izazvane bakterijom *H. pylori* u grupi gojaznih bolesnica. U istraživanje je uključeno ukupno 69 gojaznih žena i 55 žena sa normalnom težinom, koje su činile kontrolnu grupu. Kod svih učesnica izmeren je indeks telesne mase i urađeno je testiranje na prisustvo ove bakterije. Bolesnice su bile starosne dobi od 20 do 59 godina. Prag za klasifikaciju gojaznosti bio je indeks telesne mase od 30 kg/m² ili iznad. Kod pedeset (72,5%) učesnica test na prisustvo bakterije *H. pylori* bio je pozitivan. Trideset dve (58,2%) učesnice bile su seropozitivne, što nije bilo značajno (p > 0,05) u poređenju sa grupom gojaznih žena. Sve bolesnice iznad 45 godina starosti bile su pozitivne na prisustvo bakterije *H. pylori*.

Rezulati studije nisu ukazali na značajan odnos između infekcije izazvane bakterijom *H. pylori* i gojaznosti. Utvrđeno je da prevalencija infekcije izazvane bakterijom *H. pylori* raste sa godinama.

Ključne reči: **gojaznost, *Helicobacter pylori*, indeks telesne mase**