

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

SJIF Impact Factor 3.628

Research Article ISSN 3294-3211

EJPMR

PREVALENCE OF HELICOBACTER PYLORI INFECTION WITH PEPTIC ULCER **DISEASES IN IRAQI PATIENTS**

Essmaa H. Gutef*

Department of Basic Science, Collage of Dentistry, Wasit University, Iraq.

*Author for Correspondence: Dr. Essmaa H. Gutef

Department of Basic Science, Collage of Dentistry, Wasit University, Iraq.

Article Received on 19/02/2016

Article Revised on 10/03/2016

Article Accepted on 01/04/2016

ABSTRACT

OBJECTIVE: To determine the prevalence of *Helicobacter pylori* (*H. pylori*) infection among patients presenting with peptic ulcer disease (PUD). We aimed to examine the effect of age, gender, and types of peptic ulcer on the prevalence of the disease. METHODS: The study included 75 patients with PUD who underwent upper gastrointestinal endoscopy at Baghdad Teaching Hospital/Medical City from 1st of February to 30st of June 2015. All patients underwent upper gastrointestinal endoscopy for visual examination to distinguish between the gastric ulcer (GU) and duodenal ulcer (DU), and the stool antigen test (SAT) for the detection of H. pylori. RESULTS: The overall prevalence rate of *H. pylori* infection among 75 patients with PUD was 71.3%, while it was high among males (54.55%) as compared with females (45.45%). The H. pylori was high among males predominance with duodenal ulcer (56.1%) as compared with females (43.9), also high incidence of gastric ulcer was shown in this study in elderly patients with mean age (50.4 ± 3.07) years, compared to that of duodenal ulcers mean age (34.5 ± 2.69). Considering the location of ulcer, a lower prevalence of *H. pylori* infection was found in patients with gastric ulcer, at a rate of 66.7%, compared with 75.9% for those with duodenal ulcers, **CONCLUSION**: The high prevalence of H. pylori with gastric ulcer was found in the elderly and the high prevalence of H. pylori with duodenal ulcer was found in the males, also a lower prevalence of H. pylori infection was found in patients with gastric ulcer compared with those with duodenal ulcers.

KEYWORDS: gastroduodenal diseases, *Helicobacter pylori*, Peptic ulcer, Iraq.

1. INTRODUCTION

Helicobacter pylori is a gram-negative, curved and microaerophilic bacterium. It infects the stomach mucosa of more than 50% of the world's human populations. [1], it may cause variety of clinical symptoms such as chronic gastric inflammation, PUD, mucosa-associated lymphoid tissue (MALT) lymphoma and gastric adenocarcinoma. [4],[5] The majority of *H. pylori* infected persons are asymptomatic, and only a fraction (10-20%) of carriers' manifest clinical disease. [6],[7] The prevalence of *H. pylori* infection is variable in different countries.^[3] In 1983, Warren and Marshall described the presence of curved bacilli on gastric epithelia taken from patient with active chronic gastritis and latter on it have been proved that *H.pylori* are responsible for many gastrointestinal tract diseases including peptic ulcer. [8]

A peptic ulcer can be defined as a form of ulceration which develops for a reason in the epithelial lining surface expose to acid secretion of gastric glands. [9]

MATERIALS AND METHODS

Patients and samples: This study performs on newly diagnosed patients with peptic ulcer disease, who

attended the Endoscopy Unit of Baghdad Teaching Hospital/Medical City from 1st of February to 30st of June 2015. Patients were selected by a senior physician. Exclusion criteria if they were treated with antibiotics, proton pump inhibitors (ppIs) for the last 2 weeks, H2 receptor blockers, bismuth salts, and diarrhea at the time of sampling. Based on endoscopic examination patients were classified into the following groups: gastric ulcer (n = 14), duodenal ulcer (n = 41) and Stool specimens from each patient were collected and kept on -20°C until used for detection of *H. pylori* by SAT.

Diagnosis of PUD: The endoscopic examination was performed to verify the diagnosis of peptic ulcer disease distinguish between the gastric ulcer and duodenal ulcer.

Identification of H. pylori: Stool antigen test (SAT) identifies H.pylori antigen present in Stool. It's based immuno-chromatographic assay by a test (Helicobacter Antigen Quick Castte, Romania). Allow the test castte and samples to reach room temperature prior to testing. Place 1 ml (approximately 20 drops) of the sample diluents in a test tube. Add approximately a sample portion of 5 mm

diameter with a swab and shake gently in order to unstick and facilitate the sample dispersion. Shake the test tube well in order to assure good sample dispersion. Let the tube rest for at least 5 min for sedimentation. Dispense 4 drops of clear supernatant into the sample well of the cassette. Read the result after 10-15 minutes. In case of positive result colored line might existed, while its lack indicated a negative result.

Statistical Analysis: Data were analyzed using SAS 2012 (Statistical Analysis System). Chi-square test was utilized to compare between the results of the studied parameters among different patients groups. [10] Values with P<0.05 were considered to be significant.

RESULTS

A total 75 patients with endoscopically proved peptic ulcer were include in this study.fivety-five patients were *H.pylori* positive, 30 (54.55%) were male and 25 (45.45%) were female, patient with gender have significant difference * (P<0.05). Twenty were *H. pylori* negative, 10 (50%) were male and 10(50%) were female as shown in table (1).

Table (1):

Test	SAT				
Gender	(<i>H.pylori</i> +ve)		(H.pylori –ve)		Total
	N	%	N	%	
Male	30	54.55%	10	50.00%	40
Female	25	45.45%	10	50.00%	35
Total	55		10		75
Chi-square	4.68*		00.0 NS		3.25NS

distribution of *H.pylori* positive and negative in patients with peptic ulcer disease according to gender.

Ulcer type	Patients	A	Age	Gender		
	number	Range	Mean	Male	Female	
Gastric ulcer	14	42-70	50.4± 3.07	7 (50.00%)	7 (50.00%)	
Duodenal ulcer	41	30-70	34.5± 2.69	23(56.1%)	18 (43.9%)	
Total	55	30-70	36.6± 2.75	30 (53.1%)	25 (46.9%)	

P<0.05 *

The age of 14 patients with gastric ulcer range from 42-70 years (mean=50.4±3.07), 7male (50%) and 7 females (50%) while the ages of 41 patients with duodenal ulcer

range from 30-70 years (mean= 34.5 ± 2.69), 23 male (56.1%) and 18 female (43.9%) as shown in table (2).

Table (2): Age and gender distribution in peptic ulcer patients infected with *H.pylori* according to the type of ulcer

Data presented as mean ±SE (age)

Data presented as number and percentage (%)

The prevalence of *H.pylori* infection that determined by SAT shows that 55(73.3%) were *H.pylori* positive while 20(26.7%) of the patients were *H. Pylori* negative. patients with gastric ulcer high significant difference (P<0.01) in prevalence between *H.pylori* positive

(66.7%) and *H.pylori* negative (33.3%).patients with duodenal ulcer have high significant difference (P<0.01) in prevalence between *H.pylori* positive (75.9%) and *H.pylori* negative (24.1%) respectively as shown in table (3).

Table (3): Prevalence of *H.pylori* infection in patients with peptic ulcer disease.

Ulcer type	Patients	Positive result		Neg	ative result	Chi- square
	number	N	%	N	%	
Gastric ulcer	21	14	(66.7%)	7	(33.3%)	10.673**
Duodenal ulcer	54	41	(75.9%)	13	(24.1%)	12.094**

Total	75 55	(/1.5%)	20	(28.7%)	10.827**
-------	-------	---------	----	---------	----------

* (P<0.05), ** (P<0.01).

DISCUSSION

Helicobacter pylori (H. pylori) infection is a worldwide problem with a significant morbidity and mortality. [11, 12] Since the discovery of H. pylori, several studies have been undertaken to evaluate the prevalence of H.pylori among Gastroduodenal manifestations.

Regarding the gender, male and female for *H. pylori* positive patients were (30 males and 25females), this finding was close to that of Hajiani *et al.*, (2008)^[13] regarding *H. pylori* positivity, where male gender showed only a marginal predominance.^[13] A study by Yasir *et al.*, (2014)^[14] was found the predominance of male gender over female in *H. Pylori* positive patients.^[14] This may be due to a significant higher infection rates in men than women and the literatures regarding the relationship between sex and *H. pylori* infection is conflicting. It is possible that women are more likely to have infection eradicated with antimicrobials used for other illnesses.^[15]

The result in this study showed a male predominance with DU in *H. pylori* positive patients and this finding was in agreement with Marques *et al.*, (2011)^[16] who reported a high prevalence of duodenal ulcer in a male gender of Brazilian population with positive *H. pylori* infection. This was considered important determinants to gastrointestinal diseases outcome. ^[16] Chao Wu *et al.*, (2008)^[17] also reported a markedly lower prevalence of duodenal ulcer in females than in males. ^[17]

Studies had also reported a lower incidence of duodenal ulcer in young women until the onset of menopause and led to the idea that somehow female hormones protect against the development of duodenal ulcer. [18, 19] Tuo *et al.*, (2011)^[20], conclude that estrogen regulates human duodenal bicarbonate secretion, which could reduce the risk for duodenal ulcer in women and contribute to sex differences in prevalence of duodenal ulcer. [20]

A relatively high incidence of gastric ulcer was shown in this study in elderly people with mean age (50.4 ± 3.07) years, compared to that of duodenal ulcers mean age (34.5 ± 2.69) years, which was consistent with Dong *et al.*, $(2004)^{[21]}$ who reported an older patients with gastric ulcer than with duodenal ulcer. This may be attributable to lower defensive factors such as poor gastrin action capable of healing by its atrophic effect, decreased mucus, bicarbonate secretion and prostaglandins. [16]

In this study 73.3% of peptic ulcer patients were positive for H. pylori and 26.7% were negative. This was consistent with other studies reported higher percentage of H. pylori positive (69.3%-80.4%) compared to (19.6%-30.7%) H. pylori negative patients. [14, 22]

In one study conducted in Iraq showed that 78% of adults were infected with *H. pylori*. [23]

Considering the prevalence duodenal and gastric ulcers in *H. pylori* infected patients of in this study we found a higher prevalence of duodenal and gastric ulcers in *H. pylori* infected patients than in patients without *H. pylori* infection. Similar results were also reported by Chao Wu *et al.*, (2008). ^[17] This may indicate the role of *H. Pylori* infection was the main environmental risk factor in patients with gastric ulcer and duodenal ulcer. ^[24]

Considering the location of ulcer, a lower prevalence of *H. pylori* infection was found in patients with gastric ulcer, at a rate of 66.7%, compared with 75.9% for those with duodenal ulcers. This finding was in agreement with other studies. [25,26] Also comparable results of other studies reported *H. pylori* infection in about (42.9%-70%) in cases with gastric ulcers and (62.4%-90%) in those with duodenal ulcers.

REFERENCES

- Blaser MJ. Ecology of Helicobacter pylori in the human stomach. J Clin Invest, 1997; 100: 759-62.
- Blaser MJ. Helicobacter pylori and the pathogenesis of gastroduodenal inflammation. J Infect Dis, 1990; 161: 626-33.
- 3. Dunn BE, Cohen H, Blaser MJ. Helicobacter pylori. Clin Microbiol Rev. 1997; 10: 720-41.
- 4. Kumar S, Kumar A, Dixit VK. Direct detection and analysis of vacA genotypes and cagA gene of Helicobacter pylori from gastric biopsies by a novel multiplex polymerase chain reaction assay. Diagn Microbiol Infect Dis, 2008; 62: 366-73.
- 5. Forsyth MH, Atherton JC, Blaser MJ, Cover TL. Heterogeneity in levels of vacuolating cytotoxin gene (vacA) transcription among Helicobacter pylori strains. Infect Immun, 1998; 66: 3088-94.
- Boyanova L, Markovska R, Yordanov D, Marina M, Ivanova K, Panayotov S, et al. High prevalence of virulent Helicobacter pyloristrains in symptomatic Bulgarian patients. Diagn Microbiol Infect Dis, 2009; 64: 374-80.
- 7. Yamaota Y, Kato M, Asaka M. Geographic differences in gastric cancer incidence can be explained by differences between Helicobacter pylori strains. Intern Med, 2008; 47: 1077-83.
- 8. Pahwa R, Neeta, Kumar V, Kohli K. Clinical manifestations, causes and management strategies of peptic ulcer disease. Int. J. Pharm. Sci. Drug. Res., 2010; 2(2): 99-106.
- 9. Friedman S L, McQuaid K R, Grendell J H. Current Diagnosis & Treatment in Gastroenterology 2nd ed. McGraw-Hill/Appleton & Lange, 2002; 569-597.

- SAS. 2012. Statistical Analysis System, User's Guide. Statistical. Version 9.1th ed. SAS. Inst. Inc. Cary. N.C. USA.
- 11. Fuccio L, Eusebi LH and Bazzoli F. Gastric cancer, Helicobacter pylori infection and other risk factors. World J .Gastrointest. Oncol. 2010; 2: 342-347.
- 12. Alakkari A, Zullo Aand O'Connor HJ. Helicobacter pylori and non malignant diseases. Helicobacter 2011; 16(1): 33-37.
- 13. Hajiani e, hashemi j and vosoghi t. comparison of a 10 day triple and a two-week quadruple therapy in eradicating helicobacter pylori infection in patients referred to imam khomeini hospital clinics ahwaz, iran. jundishapur journal of natural pharmaceutical products, 2008; 3(1): 45-52.
- 14. Yasir S, Moin F and Akhtar S M.Frequency of Helicobacter Pylori Infection on Histopathology in Patients with Dyspepsia. American Journal of Clinical Medicine Research, 2014; 2(3): 53-56.
- Naja F, Kreiger N and Sullivan T. Helicobacter pylori infection in Ontario: Prevalence and risk factors. Can. J. Gastroenterol., 2007; 21(8): 501-506.
- 16. Marques SB, Mattar R, Artifon EL, et al. High prevalence of duodenal ulcer in a tertiary care hospital in the city of São Paulo, SP, Brazil. Arq. Gastroenterol, 2011; 48(3): 171-174.
- 17. Chao Wu H, Guang Tuo B, Min Wu W, et al. Prevalence of Peptic Ulcer in Dyspeptic Patients and the Influence of Age, Sex, and Helicobacter pylori Infection, Digestive diseases and sciences, 2008; 53(10): 2650-2656.
- 18. Javed M, Amin K, Muhammad D, et al. Prevalence of H. Pylori. Professional Med. J. 2010; 17(3): 431-439.
- 19. Andrecia V, Dumitras D, Sasca N, et al. H. Pylori like organisms in gastrodoudenal disease. Gastroenterol. Clin. Biol. 1990; 14(5): 437-41.

- Tuo B, Wen G and Wei J. Estrogen Regulation of Duodenal Bicarbonate Secretion and Sex-Specific Protection of Human Duodenum, Gastroenterology 2011; 141(3): 854–863.
- 21. Dong WG, Cheng CS, Liu SP, et al. Epidemiology of peptic ulcer disease in Wuhan area of China from 1997 to 2002. World J. Gastroenterol., 2004; 10(22): 3377-3379.
- 22. Nwodo E.N, Yakubu S.E ,Jatau E.D, et al. Seroprevalence of Helicobacter pylori Infection in Patients with Gastritis and Peptic Ulcer Disease in Kaduna, Kaduna State, Nigeria. African Journal of Basic & Applied Sciences, 2009; 1(5-6): 123-128.
- 23. Hussein NR, Robinson K, Atherton JC, et al. A study of age specific.
- 24. Helicobacter pylori seropositivity rates in Iraq. Helicobacter, 2008; 13(4): 306-307.
- 25. Barazandeh F, Yazdanbod A, Pourfarzi F, et al. Epidemiology of Peptic Ulcer Disease: Endoscopic Results of a Systematic Investigation in Iran. Middle East J. Dig. Dis., 2012; 4(2): 90-96.
- 26. Rosenstock S, JÄ, rgensen T, Bonnevie O, et al. Risk factors for peptic ulcer disease: a population based prospective cohort study comprising 2416 Danish adults. Gut., 2003; 52(2): 186-193.
- 27. Ciociola AA, McSorley DJ, Turner K, et al. Helicobacter pylori infection rates in duodenal ulcer patients in the United States may be lower than previously estimated. Am. J. Gastroenterol. 1999; 94(7): 1834-1840
- 28. Javed M, Amin K, Muhammad D, et al. Prevalence of H. Pylori. Professional Med. J. 2010; 17(3): 431-439.
- 29. 28.Lee SW, Chang CS, Lee TY, et al. Risk factors and therapeutic response in Chinese patients with peptic ulcer disease. World J. Gastroenterol. 2010; 16(16): 2017-2022.