# THE PREVALENCE OF HELICBACTER PYLORI AMONG PATIENTS COMPLAINING FROM **ABDOMINAL PAIN**

Ahed J. Al-Khatib Jordan University of Science and Technology, Jordan Ahmed Saber Abu-zaiton Al-albayt University

#### Abstract

Abstract Helicobacter pylori has been associated with a number of gastrointestinal disorders including gastritis, peptic ulcer disease, gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphoma. The present study was conducted to determine the prevalence of Helicobacter pylori infection among patients complaining from abdominal pain and visiting internal medicine clinics. It was also purposed to investigate the relationship between both age and sex with Helicobacter pylori infection. A retrospective study was conducted and included 101patients with abdominal pain. The results showed that the prevalence of IgG against Helicobacter pylori infection is 85%, while about 38.6% of cases were positive for IgA against Helicobacter pylori. No significant relationships were found for IgG, IgA with age and sex (p value >0.05). Taken together, the study showed high prevalence of H. pylori infection among patients complaining of abdominal pain. H.pylori infection is not associated significantly with age and sex.

## Keywords: H.pylori, IgA, IgG, abdominal pain

## Introduction

From a microbiologic point of view, *Helicobacter pylori* (*H. pylori*) is classified as a gram negative, spiral shaped organism that colonizes the human gastric mucosa. *H. pylori* has a ubiquitous, worldwide distribution as it is claimed to be the commonest bacterial infection worldwide (Hunt, 1996). It has the ability to colonize the acid secreting portion of the stomach where it remains for long period, possibly for life (Stein, 2002; Appelmelk, 1998).

Helicobacter pylori infects over half the world's population making it one of the most successful bacterial pathogens (Giudice, 2001). Several

studies have indicated that infection rates to be highest in developing countries and gastric colonization is usually lifelong unless infection is treated (Frenck, 2003; Miehlke, 1999).

treated (Frenck, 2003; Miehlke, 1999). Several studies suggested that persisting H.pylori infection in spite of a strong cellular and humoral immune response both locally and systemically is due to the fact that natural infection does not induce protective immunity. Furthermore, infections are asymptomatic in approximately 80% of individuals, however in the remaining 20% H. pylori infection is associated with a number of gastrointestinal disorders including gastritis, peptic ulcer disease, gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphoma (Enno, 1998; Ernst, 2000; Correa; Uemura, 2001). The H. pylori organism has the ability to colonize the human stomach and induces long-term infection of the gastric and duodenal mucosa in children and adults. It has been estimated that the majority of peptic ulcers and 85% of gastric adenocarcinomas and in over 95% of cases of gastric mucosa-associated lymphoid tissue (MALT) lymphoma are attributed to the infection by H.pylori (Tolia, 1999). Furthermore, several researchers have conducted several

Furthermore. several researchers have conducted several epidemiologic studies in various geographical regions and found the early acquirement of this infection in life especially in developing countries

(Ashorn, 1996; Thomas, 1993; Bitzan, 1998). According to studies by Wu (2001) and Rowland (1999), infection with H. pylori occurs via oral ingestion of the bacterium and is transmitted primarily within families during early childhood. Other routes of infection include direct transmission from person to person by saliva, vomit, or feces in industrialized countries (Wu, 2001; Parsonnet, 1999).

## **Treatment of H.pylori**

**Treatment of H.pylori** There are certain therapeutic options for treatment of H. pylori infection including a combination therapy with two different antibiotics together with a proton-pump inhibitor, which is in most cases successful in eradicating the bacteria and healing ulcers (Wong, 1999). In several conditions, it required to test for H. pylori if there is peptic ulcer disease, low grade gastric MALT lymphoma, after endoscopic resection of early gastric cancer, if there are first degree relatives with gastric cancer, and in certain cases of dyspepsia (Stenström, 2008) not routinely. Several ways of testing exist. One can test noninvasively for H. pylori infection with a blood antibody test, stool antigen test, or with the carbon urea breath test (in which the patient drinks 14C- or 13C-labelled urea, which the bacterium metabolizes, producing labelled carbon dioxide that can be detected in the breath). However, the most reliable method for detecting H. pylori infection is a biopsy check during endoscopy with a rapid urease

test, histological examination, and microbial culture. There is also a urine ELISA test with a 96% sensitivity and 79% specificity. None of the test methods are completely failsafe. Even biopsy is dependent on the location of the biopsy. Blood antibody tests, for example, range from 76% to 84% sensitivity. Some drugs can affect H. pylori urease activity and give false negatives with the urea-based tests (Stenström, 2008; Kusters, 2006).

## **Study objectives**

1- To determine the prevalence of H.pylori among patients complaining from abdominal pain.

2- To investigate the relationship between the IgA and IgG with sex and age.

#### **Methods and Subjects**

This is a retrospective study. We referred to files of patients visiting internal medicine clinics. Study sample included 101 patients complaining from abdominal pain. patients were screened for H.pylori using ELIZA. Both IgG and IgA were screened for all patients. Cases were considered positive

for H.pylori if the titer for IgG or IgA is  $\geq 20$ . Statistical analysis included descriptive methods such as frequencies and percentages to describe general characteristics of participants. Chi-square test was used to explore the relationship between study variable with both IgG and IgA. Statistical significance was tested at alpha level  $\leq 0.05$ .

#### Results

#### **Characteristics of participants**

As shown in data presented in table 1, the study included 101 participants. About 56% of participants were under the age of 35 years whereas about 44% of participants were more than 35 years. About 65% of participants were males while about 35% of participants were females. About 39% of cases were positive for IgA and about 85% of cases were positive for IgG (table 1).

Variable	Frequency	Percentage	
Age (years)			
<35	57	56.4	
<u>&gt;</u> 35	44	43.6	
Sex			
Male	66	65.3	
female	35	34.7	
IgA			
Positive	39	38.6	
Negative	62	61.4	
IgG			
Positive	86	85.1	
Negative	15	14.9	

Table 1: General characteristics of participants

## The relationship between IgA and study variables

The data presented in table 2 showed the relationship between IgA and both of age and sex. There were 22 cases (38.6%) positive for IgA under the age 35 years while there were 35 cases (61.4%) negative for IgA under 35 years. The data showed there were 17 cases (38.6%) positive for IgA over 35 years while there were 27 cases (61.4%) positive for IgA over 35 years. The relationship between IgA and age is not statistically significant (p value 0.990).

The data showed that about 41% of males were positive for IgA while about 59% of males were negative for IgA. About 34% of females were positive for IgA while about 66% of females were negative for IgA. The relationship between IgA and sex is not statistically significant (p value 0.515) (table 2).

variable	IgA		P value
	Positive N (%)	Negative N (%)	
<35	22 (38.6)	35 (61.4)	
<u>&gt;</u> 35	17 (38.6)		
		27 (61.4)	
Sex			0.515
Males	27 (40.9)	39 (59.1)	
Females	12 (34.3)	23 (65.7)	

Table 2: the relationship between IgA and study variables

## The relationship between IgG and study variables

As shown in table 3, 86% of participants under the age 35 years were As shown in table 3, 86% of participants under the age 35 years were positive for IgG, and 14% were negative for IgG under the same group. About 84% of participants were positive for IgG over 35 years and about 16% were negative under the same age group. The relationship between IgG and age is not statistically significant (p value 0.793). In regard to sex, about 85% of males and about 86% of females were positive for IgG. The relationship between sex and IgG is not statistically

significant (p value 0.907).

variable	IgG		P value
	Positive	Negative	
	N (%)	N (%)	
Age (years)			0.793
<35	49 (86)	8(14)	
<u>&gt;</u> 35	37 (84.1)	7 (15.9)	
Sex			0.907
Males	56 (84.8)	10 (15.2)	
Females	30 (85.7)	5 (14.3)	

Table 3: The relationship between IgG and study variables

## Discussion

Of 101 patient visiting internal medicine clinics of Prince Hamza ibn Hashim Hospital complaining from abdominal pain, 86 (85.1%) were positive for H.pylori (IgG). This finding is consistent with other studies (Tolia, 1999).

The data of the present study showed 39 cases (38.6%) were positive for IgA of H.pylori. our results are in line with other studies in which it has repeatedly been shown that the titers of IgA and IgG antibodies to H pylon in serum can be used as non-invasive tests for the presence of gastric Hpylon infection and gastritis (Kreuning, 1994).

It is not surprising to have no significant correlation between the titers of both IgG and IgA with age and sex (p value >0.05 for all). The reason is thought to be attributed to the clinical outcomes of the patients which are independent of age and sex.

**Conclusions:** the study showed high prevalence of H.pylori infection among patients complaining of abdominal pain. H.pylori infection is not associated significantly with age and sex.

## **References:**

Appelmelk BJ, Faller G, Claeys D, Kirchner T, Vandenbroucke-Grauls C on trial: the case for Helicobacter pylori (1998). Bugs and autoimmunity.Trends Immunol Today, 296: 7-11.

Ashorn M, Miettinen A, Ruuska T, Liappala P, Maki M (1996). Seroepidemiological study of Helicobacter pylori infection in infancy. Arch Dis Child, 74:141–2.

Bitzan MM, Gold BD, Philpott DJ, et al (1998). Inhibition of Helicobacter pylori and Helicobacter mustelae binding to lipid receptor, by bovine colostrum. J Infect Dis; 177(4): 955–61.

B.C. Wong, S.K. Lam, K.C. Lai, W.H. Hu, C.K. Ching, J. Ho, S.T.Yuen, C.K. Chan, G.K. Lau, C.L. Lai (1999), Triple therapy for Helicobacter pylori eradication is more effective than long-term maintenance antisecretory treatment in the prevention of recurrence of duodenal ulcer: a prospective long-term follow-up study, Aliment. Pharmacol. Ther. 13, 303–309. Correa P (2003). Helicobacter pylori infection and gastric cancer. Cancer

Epidemiol Biomarkers Prev, 12(3):238s-41s.

Del Giudice G, Covacci A, Telford JL (2001). Montecucco C, Rappuoli R. The design of vaccines against Helicobacter pylori and their development. Annu Rev Immunol, 19:523–63.

Enno A, O'Rourke J, Braye S, Howlett R, Lee A (1998). Antigen-dependent progression of mucosa-associated lymphoid tissue (MALT)-type lymphoma in the stomach, Effects of antimicrobial therapy on gastricMALTlymphoma in mice.AmJ Pathol, 152(6):1625-32.

Ernst P, Gold B (2000). The disease spectrum of Helicobacter pylori: the immunopathogenesis of gastroduodenal ulcer and gastric cancer. Annu Rev Microbiol, 54:615–40.

Frenck Jr RW, Clemens J (2003). Helicobacter in the developing world. Microbes Infect 5(8.): 705–13.

Hunt RH (1996). The role of Helicobacter pylori in pathogenesis: the spectrum of clinical outcomes. Scand J Gastroenterol Suppl, 220: 3–9. J Kreuning, J Lindeman, I Biemond, C B H W Lamers (1994). Relation

J Kreuning, J Lindeman, I Biemond, C B H W Lamers (1994). Relation between IgG and IgA antibody titres against Helicobacter pylori in serum and severity of gastritis in asymptomatic subjects. J Clin Pathol, 47:227-231 Kusters JG, van Vliet AH, Kuipers EJ (July 2006). Pathogenesis of Helicobacter pylori Infection. Clin Microbiol Rev 19 (3): 449–90. Miehlke S, Thomas R, Guiterrez O, Graham D, Go M (1999). DNA fingerprinting of single colonies of Helicobacter pylori from gastric cancer patients suggests infection with a single predominant strain. J Clin Microbiol 27(1):245–7 Microbiol, 37(1):245–7.

Parsonnet J, Shmuely H, Haggerty T (1999). Fecal and oral shedding of Helicobacter pylori from healthy infected adults. JAMA, 282: 2240-5. Rowland M, Kumar D, Daly L, O'Connor P, Vaughan D, Drumm B (1999). Low rates of Helicobacter pylori reinfection in children. Gastroenterology

Stein M, Bagnoli F, Halenbeck R, Rappuoli R, Fantl WJ, Covacci A (2002). c-Src/Lyn kinases activate Helicobacter pylori cagA through tyrosine

phosphorylation of the EPIYA motifs. Mol Microbiol, 43:971–80. Stenström B, Mendis A, Marshall B (2008). "Helicobacter pylori - The latest in diagnosis and treatment". Aust Fam Physician, 37 (8): 608–12. PMID 18704207.

Thomas JE, Austin S, Dale A, McClean P, Harding M, Coward WA (1993). Protection by human milk IgA against Helicobacter pylori infection in infancy. Lancet, 342:121-2.

Tolia V (1999). Helicobacter pylori Infection in pediatric patients. Curr Gastroenterol Rep, 1:308–13.

Uemura N, Okamoto S, Yamamoto S, Matsumura N, Yamaguchi S, Yamakido M, et al (2001). Helicobacter pylori infection and the development of gastric cancer. N Engl J Med, 345(11):784–9. Wu ML, Lewin KJ (2001). Understanding Helicobacter pylori. HUM

PATHOL, 32:247-9.