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Evaluation of *H. pylori* - Eradication Triple Therapy in Iraqi Peptic Ulcer Patients according to ABO Phenotypes: a New Study

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Abstract

Infection with *H. pylori* is an up growing public health problem that affects approximately 50% of people in industrialized nations, and up to 80% in developing countries. *Helicobacter pylori* (*H. pylori*) infection had been identified as the main causes of peptic ulcer disease (PUD). Blood group A phenotype was associated with gastric ulcer (GU) and gastric carcinoma, while blood group O phenotype found to be associated with duodenal ulcer DU predominantly; however, no explanation for this association was received. This study was conducted to, first, determine the relationship between ABO blood groups and *H. pylori* infection in peptic ulcer patients, and second, to study the response to the two weeks *H. pylori* eradication triple therapy in peptic ulcer patients carrying different blood groups. A total of 84 patients who presented with symptoms of PUD and showed positive endoscopic examination of PUD and evidence of *H. pylori* infection by histology and stool antigen test, were divided into four groups according to ABO blood group phenotype. All *H. pylori* infected patients received standard *H. pylori* eradication triple therapy for 14 days duration. Patients were followed up by re- endoscopic examination after 2 months of treatment course. The percentage of *H. pylori* infection in patients with peptic ulcer disease carrying blood group O was higher than other blood group phenotype. In *H. pylori*-infected peptic ulcerpatients , higher incidence of gastric ulcer (GU) was noticed among blood group A carriers, while higher incidence of duodenal ulcer (DU) was found among blood group O carriers when compare with other blood group phenotypes. Fourteen days triple therapy showed lower eradication rate in *H. pylori* infected blood group O peptic ulcer patients, while a higher response to the standard *H. pylori* eradication triple therapy was found among patients with blood group B phenotype.

1 Introduction

Infection with *H. pylori* is a substantial public health problem that affects 20-50% of people in developed nations, and up to 80% in less developed countries[1,] *H. pylori* infection is one of the widespread chronic bacterial infections in the world[2]. Most infected people by *H. pylori* remain asymptomatic and only minorities develop PUD[3]. It seems to be dependent on genetic factors of the host and virulence factors presented by this microorganism that determine the clinical significance of the infection [4]. It has been shown for many years, that blood group A had been associated with gastric carcinoma, while blood group O have been associated with DU; however, no explanation for this association was received[5]. Previously, it was found that infection by this gram-negative bacillus was associated with ABO blood groups in Brazilian patients performed upper gastrointestinal endoscopy[6].

In early 90s, Borén *et al*. reported that *H. pylori* bacterium

chooses to attach itself to the Lewis b antigen (Le^b), which is rich in fucose and is expressed on the surface of the epithelial cells of the gastric mucosa [7]. Also Alkout *et al*. demonstrated that H antigen represent an important receptor expressed in the gasteroduodenal mucosa to which *H. pylori* adhere[8]. Blood group apecific antigens (ABH-blood group) are complex fucosylated carbohydrates expressed on the surface of erythrocytes of all individuals of blood group A, B, or O, respectively. The ABH antigens are also found in other tissues such as gastric mucosal cells and their secretions, saliva, milk, and tear fluid [9]. Secretor status is the secretion of blood group antigens ABO (H) in fluids. In ABH secretors, people secrete antigens according to their blood groups (for example, group O people will secrete H antigen, group A people will secrete A and H antigens, etc)[10]. People who are ABH secretor probably have an ability to produce some biological decoys or metabolic chaff out into the gastric secretions that is very specific for *H. pylori* colonization. Also, in ABH non-secretors the immune response against *H. pylori* appears to be lower and *H. pylori* appears to attach with higher aggressiveness and cause more inflammation[11].

In chronic *H. pylori* infection, could be implicated in the induction of auto reactive antibodies in the pathogenesis of *H. pylori*- associated atrophic gastritis[12].The frequencies of different Lewis-secretor phenotypes vary markedly among different ethnic populations. It is well established that about 80% of the world's population are ABH antigens secretors, and only 20% are non secretors but with some racial differences[13].One of the studies in Kurds region (Kurdistan-Iraq) showed that 78% of ABH are secretors and 22% of them are non secretors[14].

Both ABH and Lewis antigens on the gastric and duodenal mucosa are synthesized through a specific enzyme (glycosyl transferase), which incorporates molecules of fucose in type I oligosaccharide precursor[15]. H antigen that is present on the cells of individuals with O blood group is the base for A and B antigens, but A and B antigens differ only in their added terminal sugars, which are controlled by specific enzymes called transferase enzymes[13,16]. The biologic significance of the A/B transferase has not been clearly demonstrated, but it would be expected that loss of this functional protein in group O patients would have some deleterious consequences for patients of this blood type[17]. The Lewis blood group antigen system composed of type 1 antigens, Le^a and Le^b and type 2 antigens, Le^x and Le^y, both biochemically related to the ABO blood group phenotypes, additionally, Lewis blood typing can identify both the Le antigen phenotype and secretor status of most people[18]. The Le^b is the predominant ABO blood group antigen expressed on epithelial cell surfaces and red cells of secretors,, whereas Le^a is expressed by non secretors, although some secretors have been found to express Le^a in variable amounts on their epithelial cells[8]. Surface and foveolar epithelia (columnar epithelia) coexpress either Le^a and Le^x in Le(a+,b−) individuals or Le^b and Le^y in Le(a−,b+) individuals, whereas a glandular epithelium lacks type 1 antigens (Le^a and Le^b) and expresses Le^x and Le^y irrespective of the secretor phenotype [19]. The O and Le (a-b+) phenotypes express a greater quantity of these fucosylated antigens in comparison with other groups and this difference predisposed these carriers to *H. pylori* infection[20]. *H. pylori* have several lipopolysaccharides such as O antigen on its outer membrane expressing Le^a and Le^b antigens. The Lewis antigen expression on the membrane of *H. pylori* for antigenic mimicry may create persistent colonization and surviving of bacteria in the stomach mucosa[21]. *H. pylori* bind to H and Le^b antigens (secretors) in the gastric mucosa[22,23].

Triple therapy has been the accepted standard of care for *H. pylori* eradication since the mid-1990s[24]. However, it may not be the most effective first-line treatment in certain regions, due to increasing antimicrobial resistance. There is controversy over the most effective duration of treatment for this regimen[25]. Evidence suggests that therapy is more successful if extended to >7 days[26,27], and many experts now recommend 10-14 days treatment[28]. The initial effectiveness of this regimen revealed that around 80–90% has progressively declined below 70–80%, in the last few years[29]. Resistance rates of *Helicobacter pylori* to antibiotics vary in different countries, and even in different regions of the same country. Choice of treatment can be modified according to antibiotic-resistance rates of *H. pylori*. The ideal target of therapeutic regimen for *H. pylori* infection should achieve an eradication rate of ≥ 80%[30].

In some countries, triple therapy with a proton-pump inhibitor, amoxicillin and clarithromycin is still the best option. In countries with clarithromycin resistance of more than 20%, bismuth-containing quadruple therapy, or non-bismuth sequential or concomitant therapies may be preferred option[31]. Studies conducted in Iraq demonstrated higher prevalence *H. pylori* positive than *H. pylori* negative among population and dyspeptic patients approximately (74-77%)[32.33].

Previous studies which showed that DUs were associated with blood group O, while GUs and gastric carcinoma were associated with blood group A[34,35]. Strong association was found among Iraqi patients as well [36] this study is another

attempt in this respect (though at a smaller scale), it may be (at least to our best knowledge); the first attempt to compare patients response to triple therapy in different blood groups or ABO phenotypes.

2 Materials and Methods

2.1 Study design

This is a prospective case control study performed on newly diagnosed patients with PUD (male and female), who attended the Endoscopy Unit from April 2013 to August 2014. Patients were registered in the study after signing a written consent, and the study was agreed by the Ethical Committee. Patients were enrolled if they showed positive endoscopic examination of PUD. Data were collected through the direct interview with the patient. Prior to endoscopy, questionnaire was prepared to take the history of the disease, and the characteristic of each patient including age, gender, blood group, the duration of complaint, smoking, alcohol intake, NSAIDs usage, presence of family history, signs, symptoms and previous treatment as well as the reason for requesting endoscopic assessment.

2.2 Patients

2.2.1 Inclusion criteria

- Patients aged between (15-77) years old.
- Patients with peptic ulcer disease (DU or GU) achieved by endoscopy.
- Patients positive for *H. pylori* by two methods: the histological examination and stool antigen test or UBT performed in the specialized gastroenterology laboratory.

2.2.2 Exclusion criteria

- Patients with drug history over the past month which mimic those used in the present study.
- Patients who had received proton pump inhibitors, H_2-blockers for a minimum of 2 weeks before test according to pervious study[37].
- Patients who had received bismuth compounds in the past 4 weeks[37].
- Patients who had severe concurrent disease that might affect the medical evaluation of this study.
- Pregnant and lactating Women.
- Patients who had severe gastroesophageal reflux disease (GERD), gastric tumors or history of gastrectomy.
- Patients with PUD who were smoker, NSAID users or alcoholics.
- Patients allergic to the study medications.

According to these criteria 116 patients whom presented with symptoms of PUD and showed positive endoscopic examination of PUD, 28 patients were *H. pylori* negative confirmed by two methods (histological examination and stool antigen test), and 88 patients present with *H. pylori* positive were included in the study, but 84 patients receive *H. pylori* eradication therapy continued throughout the study period, they were allocated into four groups:

- Blood group A**;** included 24 patients
- Blood group B**;** included 16 patients
- Blood group AB; included 12 patients
- Blood group O**;** included 32 patients

All *H. pylori* infected patients received *H. pylori* eradication triple therapy (clarithromycin (500 mg) capsules, amoxicillin (1g) capsules and esomeprazole (20mg) capsules) given twice daily for 14 days.

Patients were followed up by re-endoscopic examination after 2 months at end of treatment. Successful *H. pylori* eradication was represented as a negative Stool antigen test or UBT, and improved clinical symptoms. *H. pylori* considered positive if two tests (histological investigation plus stool antigen test or UBT) were positive. Three biopsies were taken from the antral part of the stomach of each patient for histopathological examination during the endoscopic investigation because *H. pylori* were not evenly distributed throughout the gastric mucosa[38].

Venous blood samples were taken from each patient after endoscopy for checking blood group at the beginning of the study (baseline samples). ABO phenotypes and Rh factor evaluations were carried out by standard hem agglutination assays[39]. *H. pylori* antigen in human fecal specimens, according to the technique of *H. pylori* antigen rapid test device (feces)[40]. Breath examination provides a rapid, non-invasive process of identifying the presence of active *H. pylori* infection and is frequently used to check whether eradication of the bacteria has been successful[41].

2.3 Statistical analysis

Data were analyzed using SAS 2012 (Statistical Analysis System) Version 9.1. Chi-square test was used to compare between parameters among different patients groups[42]. Analysis of *H. pylori* eradication efficacy was assessed via "per-protocol"analysis basis. Values with $P<0.05$ were considered to be significant.

3 Results and Discussions

3.1 Demographic data and disease characteristics:

Several studies demonstrated higher prevalence of *H. pylori* positive than *H. pylori* negative in peptic ulcer and dyspeptic patients[43,44,45]. Furthermore, the probability of *H. pylori* positive individuals to have any lesion in the gastric mucosa was found to be 10 folds greater than *H. pylori* negative individuals[44]. Studies conducted in Iraq demonstrated higher prevalence *H. pylori* positive than *H. pylori* negative among population and dyspeptic patients approximately (74-77)% [32,33]. Also similar finding was found in peptic ulcer patients enrolled in this study; where 75% of them showed positive *H. pylori* infection. Also previous studies presented variations of *H. pylori* infection among blood donors in different regions in the same place and in different countries[46]. Basic characteristics of the PUD patients involved in this study are shown in table 1.

The mean age of the studied patients, was 39.11 years, which was similar that reported a mean age of about 39.2 years[47]. On the contrary, other studies found a mean age of (46-50 years)[48,49,50].The percentages for *H. pylori* positive patients among males and females were 64.29% and 35.71%, respectively; these finding was compatible with other studies where male gender showed only a marginal predominance[51,52].This may be due to a significant higher infection rates in men than women and the literatures regarding the relationship between gender and *H. pylori* infection is conflicting. It is possible that women are more likely to have infection eradicated with antimicrobials used for other illnesses[53]. Other studies found that males, and females are infected at the same rate[54,55]. Additionally, the percentage of *H. pylori* positive among patients with GUs and DUs was higher than in *H. pylori* negative patients. This is consistent with other studies that reported a higher *H. pylori* infection incidence in both GUs and DUs.[56]. The distribution of ABO phenotypes in *H. pylori* positive PUD patients in this study was as follow: 28.57% for blood group A, 38.1% for blood group O, 19.04% for blood group B and 14.29% for blood group AB. This may be comparable with other studies that reported a higher incidence of *H. pylori* infection within blood group O of PUD patients[20,57]. Besides, Rh positivity in *H. pylori* positive patients was 96.43%, this finding was consistent with the study by Jaff *et al.* (2011), who reported that Rh positivity was 92.5% in *H. pylori* positive patients[20]. A similar study also found that Rh positivity were 92.9% in *H. pylori* positive patients [5].The main presenting symptom of PUD patients in the present study was epigastric pain (73.81%), heart burn (71.43% of patients) and vomiting (46.43%). Epigastric pain was the major complaint reported by several studies[49,58,59].

Table 1: Demographic data and disease characteristics in patients with peptic ulcer disease

Variables	*H. pylori* positive (No. = 84)	*H. pylori* negative (No. = 28)
Age (years)	39.11 ± 1.78	43.74 ± 2.62
Range(years)	15-77	20-72
Gender		
Female	30 (35.71)	15 (53.57)
Male	54 (64.29)	13(46.43)
Total	84(75.00)	28(25.00)
ABO phenotype		
A	24 (28.57)	11 (39.29)
O	32 (38.10)	8 (28.57)
B	16 (19.04)	6 (21.43)
AB	12(4.29)	3 (10.71)
Rh factor		
Positive	81(96.43)	26(92.86)
Negative	3(3.57)	2(7.14)
Type of ulcer		
Duodenal ulcer	53 (63.10)	16 (57.14)
Gastric ulcer	31 (36.90)	12 (42.86)
Family history of PUD		
Yes	26 (31.00)	7 (25.00)
No	58(69.0)	21(75.00)
Symptoms duration :(yr.)		
<1	29 (34.52)	11 (39.28)
1-5	27 (32.14)	8 (28.57)
>5	28 (33.33)	9 (32.14)
Presentation		
Epigastric pain	62 (73.81)	19 (67.86)
Vomiting	39(46.43)	14(50.00)
Heartburn	60(71.3)	21(75.00)
Melena	21(25.0)	4 (14.29)
Hematemesis	7 (8.33)	2 (7.14)
BMI(Kg/m^2)	25.87 ± 2.16	27.55 ± 2.92

Data presented as Mean± SE: (Age and BMI); BMI: body mass index;(n) number of patients and (%) percentage for other disease characteristics.

3.2 Distribution of H. pylori positive and negative in patients with peptic ulcer disease according to ABO phenotypes

The percentages of ABO phenotypes were analyzed in this study separately in accordance with *H. pylori* infection and non-infection (table 2). We observed that there was an association between *H. pylori* negative and blood group A, these finding was similar to that previously reported a higher

prevalence of blood group A among the uninfected patients[15]. Also the current study revealed a strong association of blood group O with *H. Pylori* infection, which was consistent with a recent study in Swedish population done by Ryberg *et al.* who found positive associations between the presence of blood group O, and *H. pylori* infection in PUD[60]. Furthermore, these results may be reinforced by data obtained from other researchers showing a greater susceptibility of blood group O patients to *H. pylori* infection[20,57,61,62].

Table 2: Distribution of *H. pylori* positive and negative in patients with peptic ulcer disease according to ABO

*H. pyori	ABO phenotypes										
	A		O		B		AB		Total		Chi-square (x^2)
	(n)	(%)	(n)	(%)	(n)	(%)	(n)	(%)	(n)	(%)	
H. pylori +ve	24	(28.57)	32	(38.10)	16	(19.04)	12	(14.29)	84	(75.00)	9.462 **
H. pylori -ve	11	(39.29)	8	(28.57)	6	(21.43)	3	(10.71)	28	(25.00)	9.462 **

Data presented as number (n) and percentage (%); ** (p< 0.01) highly significant **H. pylori* documented according to (histology; stool antigen or UBT)

The association of *H. Pylori* with blood group antigens fascinated doctors for few decades when an association was discovered between the infection of *H. Pylori* and blood group O[62]. In the early 1990s, Boren *et al.* demonstrated that Le^b antigen, which is found mostly in blood group O, functions as a receptor for *H. pylori* adhesions, mediating bacterial adherence to the gastric mucosal surface, which is essential for bacterial colonization[7]. This evidence was further supported by Alkout *et al.* who demonstrated that H-antigen, expressed on the gastroduodenal cells, acted as a receptor for *H. Pylori*[8]. This fucosylated antigen (H antigen) is not modified to A or B antigens in blood group O, which points to the fact that there is a positive correlation between blood group O and the infections caused by *H. Pylori*[62]. Furthermore, substitution of the Le^b antigen with blood group A and B determinants results in failure of *H. pylori* binding[7]. Reduced exposure of the Le^b antigen in blood groups A and B carriers could result in lower *H. pylori* infection rates, and a predominantly in persons with blood group O[63].A recent study in Iraq by Jaff *et al.* found that individuals with blood group O have significantly higher incidence of Le (a-b+) (secretor status) than in non-O blood group individuals[13]. Individuals with the Le (a–b+) phenotype (secretors) secrete Le^a as well as Le^b and ABH substances in body fluids. Thus, it is possible that the Le^b present in other body secretions such as gastric mucus may bind to specific glycoproteins of *H. pylori*, and hinder the binding of *H. pylori* to the gastric mucosa[21].The increased susceptibility of blood group O persons to peptic ulcer[8] might be partly due to blood group O individuals express a higher inflammatory responses to *H. pylori* with higher levels of lymphocyte infiltration in the gastrointestinal mucosa[64], a lower level of Von Willebrand's factor[65], higher density of colonized *H. pylori*[66], and a higher frequency of secretor status. All these together, may explain these individuals' increased susceptibility to peptic ulceration. However, Bhuiyan *et al.* showed that blood group A is found to be related with *H. Pylori* infection[67]. Earlier study demonstrated that *H. Pylori* infection is related to both O and A blood group type, and a negative relation with AB group[23]. On the contrary, only few studies have demonstrated that blood group O do not represent a risk factor for *H. pylori* infection[55,68].

Recent findings about *H. pylori* strains from different populations have revealed that *H. pylori* strains differed by approximately 1500-fold with respect to binding affinities, and there was considerable diversity related to the Bab A gene sequences[69,70]. There is heterogeneity in the expression of outer membrane proteins, especially Bab A, among different *H. pylori* strains, and there is heterogeneity in the capacity of *H. pylori* to bind to the (Le^b) antigen on the surface of gastric epithelial cells. This heterogeneity may be a factor that explains some of the differences in the clinical outcomes of this infection[69]. Not all strains are equally specific for O and (Le^b) antigen; many strains from outside South America can bind to A and (Le^b) antigen in addition to O and Le^b. For example, Peruvian strains are related to Spanish strains but not to Asian strains[70]. A study by Con *et al.* (2010) revealed higher frequency of babA2 in Japan (96.8%) than in Costa Rica (73.7%). In comparison, the frequency of babA2/B was higher in Costa Rica (11.6%) than in Japan (1.1%), the study also suggested that frequencies of babA2 and babA2/B exhibit geographic differences[71]. Another virulence factor characterized recently in *H. pylori* is sialic acid–binding adhesion (Sab A). The frequency of Sab A also exhibits geographic differences and is more common in European than Japanese[72,73]. This diversity in *H. pylori* strains may explain different findings due to

different geographic areas.

3.3 Distribution of gastric and duodenal ulcers according to ABO phenotypes

A higher incidence of GU (58.33%) in *H. pylori* positive was in blood group A when compared with other blood groups, while a higher incidence of DU (81.25%) in *H. pylori* positive was in blood group O when compared with other blood groups (Table 3).

These findings agree with previous studies, which showed that DUs were associated with blood group O, while GUs and gastric carcinoma were associated with blood group A[74,75,76].Furthermore, Syeda *et al* mentioned that the incidence of DU was 1.38 times in people belonging to blood group O as compared to other blood groups[77].This may be explained that DU were associated with acid hypersecrtion, predominantly in patients with blood group O, while GU in the body of the stomach occurring in patients with normal duodenum,were characterized by acid hyposecretion and this marked in patients with blood group A, the cause that blood type A is most likely to have gastric cancer[78]. Also Serum-pepsinogen level was found to be greater for individuals with blood group O than the blood group A. It is believed that the quantity of serum pepsinogen mass is in relation to the size of gastric secretory-cell. It is hypothesized that blood group culminates at the development of secretory cell mass, reinforcing that gastric peptic cell mass is larger in group O[77]. This might be reasons why blood group O is more susceptible to DU. While the association of blood group A with GU and carcinoma, which is also related with *H. pylori* infection. Many, other studies showed higher frequency of DU among patients with blood group O with significantly higher *H. pylori* positivity compared to other blood groups[5,36], including Iraqi population[36].

Table 3: Distribution of gastric and duodenal ulcers according to ABO phenotypes

ABO phenotypes	Patients (number)	Endoscopic findings			
		DU		GU	
		(n)	(%)	(n)	(%)
A	24	10	(41.67)	14	(58.33)
O	32	26	(81.25)	6	(18.75)
B	16	11	(68.75)	5	(31.25)
AB	12	6	(50.00)	6	(50.00)
Chi-square (x^2)	---	10.552 **		10.552 **	

Data presented as number (n) and percentage (%); ** (p< 0.01) highly significant

3.4 Ulcer-healing efficacy of H. pylori eradication triple therapy in peptic ulcer disease patients according to ABO phenotypes

Per-protocol analysis was performed to compare eradication efficacy for all patients with different blood groups who finished the course of treatment. The healing efficacy of triple therapy regimens in of peptic ulcer disease in ABO phenotypes as follows: (58.33%) for blood group A, (50%) for blood group O, (75%) for blood group B and (66.67%) for blood group AB, table 4**.** The total efficacy which was obtained in general in respect to the regimens was (59.52%). Statistically highly significant difference (P<0.01) was found among ABO phenotypes according to stool antigen or UBT after 8 weeks. Patients with blood group O showed less healing efficacy than patients with blood group A, B and AB. On the other hand, patients carrying blood group B had a higher healing efficacy than those with other groups phenotypes.

Individuals with blood group O were found to be more susceptible to peptic ulcer disease for decades without known cause until the relationship between Lewis b antigens and the attachment of *H. pylori* to gastric mucosa was observed[8]. As mentioned previously, the gastric mucosa of blood group O person are more prone for the attachment of *H. pylori*, because they had more receptors and Le^b antigens mediated the attachment of *H. pylori* to the mucosa [23].Taking together all these evidence may provide a possible explanation to the low response of group O patients to *H. pylori* eradication triple therapy.

3.5 Ulcer-healing efficacy of H. pylori eradication triple therapy in peptic ulcer disease patients according to location of ulcer in different ABO phenotype

The present study showed that patients with blood group O has the least statistically significant (P<0.01) ulcer healing efficacy in DU and GU compared with other blood groups after 2 months of *H. pylori* eradication triple regimen, table 5. No matched studies to our best knowledge that could interpret our result. However, since patients with blood group O show higher density of colonization by *H. pylori* compared to other blood groups where epithelial cells bound significantly to *H. pylori*[8]. Previous studies, have

demonstrated significant association between cagA-positive *H. pylori* strain, and the development of peptic ulcers among patients belonging to the blood group O[6,79].

Table 4: Ulcer-healing efficacy of *H. pylori* eradication triple therapy in peptic ulcer disease patients in ABO phenotypes after 2 months

ABO Phenotypes	Patients	Ulcer-healing efficacy			
		Healed	(%)	Un-healed	(%)
A	24	14	(58.33)	10	(41.67)
O	32	16	(50.00)	16	(50.00)
B	16	12	(75.00)	4	(25.00)
AB	12	8	(66.67)	4	(33.33)
Total	84	50	(59.52)	34	(40.48)
Chi-square (x^2)	---	8.637 **		8.637 **	

Data presented as number (n) and percentage (%); ** (P<0.01) high significant.

Table 5: Ulcer-healing efficacy of *H. pylori* eradication triple therapy in peptic ulcer disease patient according to location of ulcer in ABO phenotypes after 2 months

ABO phenotypes	Patients (n)	Location of ulcer (Ulcer healing efficacy)			
		DU		GU	
		(n)	(%)	(n)	(%)
A	24	6 / 10	(60.00)	8 / 14	(57.14)
O	32	13 / 26	(50.00)	3 / 6	(50.00)
B	16	8 /11	(73.00)	4 / 5	(80.00)
AB	12	4 / 6	(66.67)	4 / 6	(66.67)
Total	84	31 / 53	(58.50)	19 / 31	(61.30)
Chi-square (x^2)	----	7.613 **		9.482 **	

Data presented as number (n) and percentage (%); DU: duodenal ulcer; GU: gastric ulcer; ** (P<0.01) high significant.

Previous study demonstrated that bacterial load in patients with cag A positive was greater than in patients with cag A negative both in the antrum and corpus ($p < 0.01$)[80]. So patients with blood group O show higher density of colonization by *H. pylori*[8]. Furthermore, high antral density of *H. pylori* was associated with a significant reduction in the eradication rate after anti-*H. Pylori* treatment[81]. Also patients with blood group O have high gastric acidity as previously mentioned[77,78], and high gastric acidity was associated with reduced antibiotic therapy efficacy[82], or potentially has antibiotic resistance compared with other blood groups[83].

4 Conclusion

Association between *H. pylori* infection, ABO blood groups and Rh status in PUD has been widely evaluated over the past. This study is another attempt in this respect (though at a smaller scale), it may be to the best knowledge the first attempt to compare patients response to *H. pylori* eradication triple therapy in different blood groups or ABO phenotypes, which showed lower eradication rate after *H. pylori* eradication coarse in infected peptic ulcer patients carrying blood group O phenotype compared to those holding other blood groups, with higher percentage *of H. pylori*-infection, and higher incidence of duodenal ulcer (DU).Further study is warranted to assess the response to other *H. pylori* eradication protocols in patients carrying blood group O phenotype in an attempt to improve the response rate.

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6 Conflict of interest

The author declared none

7 Author's contributions

Manal Khalid Abdulridha(the corresponding author) brings the study design into it's applicable state along with drafting the manuscript. And the litreture review, result discussion, lab work, and data collection was carried out by Akram Ajeel Najeeb and Rana Hussein Kutaif. Finally Yassir Mustafa Kamal arranged the data into tabular form. All authers read and approved the final manuscript.

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