Association between *Helicobacter pylori* Infection, ABO Blood Groups and Rhesus Factor in Peptic Ulcer Disease Patients, in Gezira, Central Sudan

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**Authors’ contributions**

This work was carried out in collaboration between all authors. Author OHS designed the study, wrote the protocol, and wrote the first draft of the manuscript. Author MEM managed the literature searches, analysis of the study data and managed the experimental process and author OK supervised the manuscript at all steps, and final editing and approval. All authors read and approved the final manuscript.

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**ABSTRACT**

Blood group antigens were associated with peptic ulcer disease, which is potentially caused by *Helicobacter pylori*. It was recently demonstrated that the receptor for *H. pylori* is the blood group antigen lewis*, which is exposed only in blood group O.

**Objectives:** To report the possible correlation among *H. Pylori* Infection, ABO and Rhesus (Rh) blood groups in patients with peptic ulcer disease.

**Methods:** This cross-sectional, prospective study was carried out between Jan 2010 and Dec 2010 among patients suffering from dyspeptic symptoms attending to Wad Medani Teaching Hospital-Endoscopy Unit. All patients had their blood group phenotype and Rhesus determined by

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1. INTRODUCTION

Helicobacter pylori (H. pylori) are Gram-negative, micro-aerophilic, spiral, rod-shaped bacteria, which are a major health problem worldwide [1]. Gastritis, peptic ulcer disease, gastric carcinoma and mucosa associated lymphoid tissue (MALT) lymphoma are recognized complications of H. pylori infection [2]. The prevalence of H. pylori is still high in most countries. In north European and North American populations, about one-third of adults are still infected, whereas in south and east Europe, South America, and Asia, the prevalence is often higher than 50% [3]. In developing countries, up to 93.6% of adults are infected with H. pylori [4]. In Sudan, the prevalence of infection was estimated to be between 65.8% and 80% [5,6].

An infected individual with H. pylori has an estimated lifetime of 10-20% for the development of peptic ulcer disease (PUD), which is at least 3-4 fold higher than in non-infected subjects. H. pylori infection can be diagnosed in 90-100% of duodenal ulcer (DU) patients and in 60-100% of gastric ulcer (GU) patients [7]. The most important factors of virulence of H. pylori are the cag Pathogenicity Island (cag PAI), vacuolating cytotoxin A (Vac A), urease, outer membrane proteins. Based on the degree of pathogenicity, H. pylori isolates are divided into two types: type I strains of H. pylori contain a 40 kb segment on their chromosome, termed cag PAI, they produce functional Vac A toxin, and the infection by the strains is associated with more severe disease forms; type II strains of H. pylori do not contain cag PAI, do not produce Vac A toxin, and induce only a mild form of gastritis [8]. The diagnosis of H. pylori infection is obtained by either invasive (urease test, culture, histology) or non-invasive tests (serology, urea breath test) [9].

The sensitivity of biopsy urease tests is approximately 90 to 95 percent, and specificity is 95 to 100 percent [10]. Thus, false positive tests are unusual.

Epidemiological studies have demonstrated higher frequencies of the O blood group and the nonsecretor phenotype of ABH antigens among patients suffering from peptic ulcers. Since Helicobacter pylori has been established as the main etiological factor in this disease, controversies about the associations of the ABO and Lewis blood group phenotypes and secretor and nonsecretor phenotypes in relation to susceptibility towards infection by this bacillus have been presented [11]. This study was done to report the possible correlation among H. pylori Infection, ABO and Rhesus (Rh) blood groups in Sudanese patients with peptic ulcer disease.

2. MATERIALS AND METHODS

2.1 Study Design

This was a descriptive, prospective cross-sectional hospital-based study. It was conducted in Wad Medani Teaching Hospital–Endoscopy Unit, Wad Medani, Gezira and Central Sudan, to report the possible correlation among H. pylori infection, ABO and Rhesus (Rh) blood groups in Sudanese patients with peptic ulcer disease (PUD).
2.2 Study Population

A total number of 40 consecutive patients who were diagnosed with PUD by oesophagogastroduodenoscopy (OGD) in Wad Medani Teaching Hospital—Endoscopy Unit and agreed to participate in the study, in the period from Jan 2010 to Dec 2010 were studied.

2.3 Inclusion Criteria

All patients who were diagnosed with PUD by oesophagogastroduodenoscopy during the study period who agreed to participate in the study by a written consent were recruited. For children consent was obtained from their parents.

2.4 Exclusion Criteria

Patients with gastric malignancy, those who recently had antibiotics (4 weeks before oesophagogastroduodenoscopy), antisecretory drugs, or proton pump inhibitors (2 weeks before oesophagogastroduodenoscopy) and those with a recent active gastrointestinal bleeding were excluded from the study.

2.5 Data Collection

40 patients with endoscopic evidence of peptic ulcer disease (cases of duodenal ulcer and gastric ulcer) were enrolled in this study. All patients underwent oesophagogastroduodenoscopy to prove the presence of PUD by using Pentax and Olympus videoscopes. All of them had their gastric antral biopsies examined for the presence of *H. pylori* by urease test (biopsy urease test, Pronto Dry: Manufacturer GASTREX, Warsaw Poland). A color change between pink or red from 5 to 30 minutes was considered as positive. Blood group phenotypes were determined by the slide hemagglutination method and correlated with *H. pylori* infection in those patients. The characteristic of patient and clinical data were collected by using a questionnaire.

2.6 Data Analysis

Data were analyzed by computer by using Statistical Package for Social Sciences (SPSS) program version 16. Data were analyzed by Nonparametric Binomial test based on Z approximation. The standard level of significance was taken at γ=5%. Data were then tabulated and presented in simple table forms.

2.7 Ethical Consideration and Clearance

A written consent was obtained from all patients. Ethical clearance was obtained from the Ethical Clearance Committee of the Faculty of Medicine, University of Gezira, before the start of the study.

3. RESULTS

With respect to the inclusion criteria, 40 cases were enrolled in the study. 29 (72.5%) were males and 11(26.5%) were females, with an age range between 14-85 years and a mean age of 50.75±18.8 years. 30 patients (75%) were seropositive for *H. pylori* by biopsy urease test, while 10 (25%) were negative. Blood groups distribution among the study subjects showed: Blood group O in 24 patients (60%), B in 13 patients (32.5%) and A in 3 patients (7.5%). None of them had blood group AB. See Table 1. 38 patients (95%) were Rhesus positive and 2(5%) were negative. Most patients 31 (77.5%) had D.U at oesophagogastroduodenoscopy, while 9(22.5%) had G.U.

| Table 1. Blood group distribution and rhesus factor (N=40) |
|-----------------------------------------------------------|
| Blood group | Rhesus | N | Observed prop | Test prop | Exact sig. (2-tailed) |
|-------------|--------|---|---------------|-----------|---------------------|
| A           | Positive | 3 | 1             | 0.5       |                     |
|             | Total   | 3 | 1             |           |                     |
| B           | Positive | 13| 1             | 0.5       |                     |
|             | Total   | 13| 1             |           |                     |
| O           | Positive | 22| 0.92          | 0.5       | 0.000               |
|             | Negative| 2 | 0.08          |           |                     |
|             | Total   | 24| 1             |           |                     |
| AB          | 0       | 0 | 0             | 0         |                     |
Among *H. Pylori* urease positive patients; 3 had Blood group A (10%), 9 had B (30%) and 18 (60%) had group O. See Table 2. Among patients who had D.U at oesophagastroduodenoscopy; 3 were Blood group A (9.7%), 10 were B (32.3%) and 18 were O (58.1%), while among those with G.U; 3 were Blood group B (33.3%) and 6 were O (66.7%). See Table 3.

With respect to Rhesus factor, it was found to be positive in 28 patients (93.3%) and it was negative in 2 patients (6.7%) among those who were urease positive for *H. Pylori*. Among patients with DU; 29 (93.5%) were Rhesus positive and 2 (6.5%) were negative. All patients with GU (9 patients) were Rhesus positive (100%). See Table 3. This implies that there was statistically significant correlation between the O blood group, positive Rhesus factor and *H. Pylori* infection in peptic ulcer disease patients. γ=0.023 and 0.024 respectively.

### 4. DISCUSSION

To our knowledge, the current study is the first one conducted in Gezira of central Sudan to report the possible correlation among *H. pylori* Infection, ABO and Rhesus (Rh) blood groups in Sudanese patients with peptic ulcer disease. The results of this study showed to a fair extent an association between the O blood group and *H. pylori* infection (γ=0.023), a finding which is supported by other studies [12,13]. Blood group A and B patients in this study were less prone to *H. pylori* infection (γ=0.25 and 0.26 respectively). Individuals with blood group AB were shown to be less prone to *H. pylori* infection in a different study [14].

Borén et al. [15] reported that the attachment of *H. pylori* to gastric mucosa is mediated by the Lewis b carbohydrate but receptors might therefore be reduced in individuals of blood groups A and B compared to people with blood group O. This was further supported by the findings of Alkout et al. [13] who showed that H antigen is an important gastroduodenal mucosal cell-receptor for *H. pylori* attachment [16]. Other ABO phenotypes were shown to express Lewis b carbohydrate but in a low level [17]. The findings of this present study support the hypothesis that, blood group O is more susceptible to *H. pylori* infection. A meta-analysis was conducted in Ethiopia by Shaweno et al. to verify the association between *H. pylori* infection and O blood group. Fourteen of the 18 included studies reported no significant association. Among ten reviewed studies which were conducted among dyspeptic patients, four showed statistically significant association [18]. The overall prevalence of *H. pylori* in the present study was 75% which is lower than that of 80% reported by Ahmed et al. [19] in patients suffering from gastro-oesophageal reflux disease. It is however higher than a prevalence of 48% reported by endoscopy in central Sudan [20]. The lower rate of infection in the present study may be due to improvement in standard of living. The prevalence of infection was low as compared to the neighboring countries such as Uganda (87%), Ethiopia (89%) and Libya (94%) [21,22,23], which might be explained by the difference in socioeconomic status, being higher among groups with lower socioeconomic status [24].

In this study, more males than females were positive for *H. pylori*. Patients who were suffering from duodenal ulcers were outnumbering those with gastric ulcers, as seen by other studies [25,26]. Others have shown predominance of females [27], while others have noticed no relation to gender [28,29].

| Blood group | Biopsy urease test | N   | Observed Prop | Test Prop | Exact Sig. (2-tailed) |
|-------------|-------------------|-----|---------------|-----------|----------------------|
| A           | Positive          | 3   | 1             | 0.5       | 0.250                |
|             | Total             | 3   | 1             |           |                      |
| B           | Positive          | 9   | 0.692308      | 0.5       | 0.267                |
|             | Negative          | 4   | 0.307692      | 0.5       |                      |
|             | Total             | 13  | 1             |           |                      |
| O           | Negative          | 6   | 0.25          | 0.5       | 0.023                |
|             | Positive          | 18  | 0.75          | 0.5       |                      |
|             | Total             | 24  | 1             |           |                      |
| AB          | 0                 | 0   | 0             | 0         | 0                    |

Table 2. Blood group correlation with biopsy urease test (N=40)
Table 3. Rhesus factor correlation with OGD and biopsy urease test (N=40)

| Rhesus | OGD   | Category | N   | Observed prop | Test prop | Asymp. sig. (2-tailed) | Exact sig. (2-tailed) |
|--------|-------|----------|-----|---------------|-----------|------------------------|-----------------------|
| Positive | DU    | Urease test | Positive | 21 | 0.73 | 0.5 | 0.024 | 0.024 |
|         |       |           | Negative | 8  | 0.27 | 0.5 | 0.180 |
|         |       |           | Total    | 29 | 1     |       |       |
| GU     | Urease test | Positive | 7    | 0.77 | 0.5 | 0.024 | 0.024 |
|         |       |           | Negative | 2  | 0.23 | 0.5 | 0.180 |
|         |       |           | Total    | 9  | 1     |       |       |
| Negative | DU    | Urease test | Positive | 2  | 1    | 0.5 | 0.024 | 0.024 |
|         |       |           | Total    | 2  | 1    |       |       |

Based on Z Approximation

5. CONCLUSION

This study suggets that can be a significant association between Rhesus positive group O and H. pylori infection.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Ruggiero P. Helicobacter pylori and inflammation. Curr Pharm Des. 2010;16(38):4225-4236.
2. Ruggiero P. Helicobacter pylori infection: What’s new. Curr Opin Infec Dis. 2012;25:337-344.
3. Eusebi LH, Zagari RM, Bazzoli F. Epidemiology of Helicobacter infection. Helicobacter. 2014;19(Suppl 1):1-5.
4. Olokoba A, Gashu W, Bwala S, Adamu A, Salawu FK. Helicobacter pylori infection in Nigerians with dyspepsia. Ghana Med J.2013;47:79-81.
5. Abdallah TM, Mohammed HB, Ali AA. Sero-prevalence and factors associated with Helicobacter pylori in Eastern Sudan. Asian Pac J Trop Dis. 2014;4:115-119.
6. Azim mirgahi YA, Ahmed M, Ismail MO, Fedil SS. Kamel M, Saidia H. Detection of Helicobacter pylori in endoscopic biopsies in Sudan. Trop Doct. 1994;24:161-163.
7. Kuipers EJ, Thijs JC, Festen HP. The prevalence of Helicobacter pylori in Peptic ulcer disease. Aliment Pharmcol Ther. 1995;9(Suppl 2):59-69.
8. Censini S, Lange C, Xiang Z, Crabtree JE, Ghira P, Borodovsky M, Rappuoli R, Covacci A. cag, a pathogenicity island of Helicobacter pylori, encodes type I specific and disease-associated virulence factors. Proc Natl Acad Sci USA. 1996;93:14648-53.
9. Fischbach W, Malfertheiner P, Hoffmann JC, Bolton W, Kist M, Koletzko S, et al. Helicobacter pylori and gastrodudenal ulcer disease. Dtsch Arztebl Int. 2009;106:801-808.
10. Howden CW, Hunt RH. Guidelines for the management of Helicobacter pylori infection. Am J Gastroenterol. 1998;93:2330.
11. Jaff MS. Relation between ABO blood groups and Helicobacter pylori infection in symptomatic patients. Clin Exp Gastroenterol. 2011;4:221-226.
12. Lin CW, Chang YS, Wu SC, Cheng KS. Helicobacter pylori in gastric biopsies of Taiwanese patients with gastroduodenal diseases. Jpn J Med Sci Biol. 1998;51:13-23.
13. Alcout AM, Blackwell CC, Weir DM, Poxton IR, Elton RA, Luman W, Palmer K. Isolation of cell surface component of Helicobacter pylori that binds H type 2, Lewis and Lewis B antigens. Gastroenterology.1997;112:1179-1187.
14. Kanbay M, Gur G, Arslan H, Yilmaz U, Boyacigil S. The relationship of ABO blood group, age, gender, smoking, and Helicobacter pylori infection. Dig Dis Sci. 2005;50:1214-1217.
15. Borén T, Falk P, Roth KA, Larson G, Normark S. Attachment of Helicobacter pylori to human gastric epithelium mediated by blood group antigens. Science. 1993;262:1892-3.
16. Alcout AM, Blackwell CC, Weir DM. Increased inflammatory responses of persons of blood group O to Helicobacter pylori. J Infect Dis. 2000;181:1364-1369.
17. Perry HE, Franklin RA, Bray SJ, Lo MK, Svensson LA, Henry SM. A novel study of association between Nisseria gonorrhoeae and the human carbohydrate blood group. Immunohematology. 2007;23:100-104.

18. Shameno D, Daka D. Association between O blood group and Helicobacter pylori infection: A systematic review and meta-analysis. Journal of Public Health and Epidemiology. 2013;5:471-478.

19. Ahmed HH, Mudawi HM, Fedail SS. Gastro-oesophageal reflux disease in Sudan: A clinical endoscopic and histopathological study. Trop Gastroenterol. 2004;25:135-138.

20. Abdalsadig NA, Adam AA, Abdul-Aziz H, Omer WH, Osman HA, Bolad AK. Comparison of different diagnostic methods of Helicobacter pylori infection in Sudanese patients. Alneelain Med J. 2012;2:27-34.

21. Newton R, Ziegle JL, Carpenter L, Gold BD, Owens M, Beral V, et al. Helicobacter pylori and cancer among adults in Uganda. Infect Agent Cancer. 2006;1:5.

22. Moges F, Kassu A, Mengistu G, Adugna S, Andualem B, Nishikawa T, et al. Seroprevalence of Helicobacter pylori in dyspeptic patients and its relationship with HIV infection, ABO blood groups and life style in a university hospital, northwest Ethiopia. World J Gastroenterol. 2006;12:1957-1961.

23. Bakka AS, El-Gariani AB, Abou Ghrara FM, Salih BA. Frequency of Helicobacter pylori infection in dyspeptic patients in Libya. Saudi Med J. 2002;23:1261-1265.

24. Graham DY, Malaty HM, Evans DG, Evans DJ, Jr, Klein PD, Adam E. Epidemiology of Helicobacter pylori in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. Gastroenterology. 1991;100:1495-1501.

25. Barik A Salih, M Fatih Abasiyanik, Nizamettin Bayyurt, Ersan Sander. H pylori infection and other risk factors associated with peptic ulcers in Turkish patients: A retrospective study. World J Gastroenterol 2007;21(13):3245-3248.

26. Vu C, Ng YY. Prevalence of Helicobacter pylori in peptic ulcer disease in a Singapore hospital. Singapore Med J. 2000;41:578-559.

27. Lacy B, Smore J. Helicobacter pylori: Ulcers and more: The beginning of an era. J Nutr. 2001;131:89-93.

28. Farshad SH, Japoni A, Alborzi A-V, Zarenezhad M, Ranjbar R. Changing prevalence of Helicobacter pylori in south of Iran Iranian. J Clin Infect Dis. 2010;5:65-69.

29. Alazmi WN, Siddique I, Alateeqi N, Al-Nakib B. Prevalence of Helicobacter pylori infection among new patients with dyspepsia in Kuwait. BMC Gastroenterol. 2010;10:14.