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ORIGINAL ARTICLE

ABO, Rh Blood Groups and Haemoglobin Genotype Associations with *Helicobacter pylori* Infection amongst Indigenes of Wiyaakara, Rivers State

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Abstract

Introduction: This study aims to determine the association of ABO/Rh Blood groups and haemoglobin genotype with *Helicobacter pylori* infection among indigenes of Wiyaakara, Ogoni, Rivers State. This is a cross-sectional study carried out in Wiyaakara, Khana Local Government Area in Rivers State to determine the prevalence of *Helicobacter pylori* infection and its association with ABO/Rh blood group and haemoglobin genotype. The study was carried out amongst indigenes of Wiyaakara village in Khana LGA of Rivers State, Nigeria. Wiyaakara is part of Ogoni.

Materials and Methods: Determination of ABO/Rh blood group was done using tile method. Haemoglobin genotype was determined using cellulose acetate paper electrophoresis while determination of *Helicobacter pylori* infection was done using the rapid diagnostic test strip method.

Results: Out of 130 participants, 48.5% tested positive for *Helicobacter pylori* infection. Amongst those that tested positive, 68% were females while 32% were males. In ABO blood grouping, blood group O recorded the highest infectivity rate, followed by blood group A, blood group B and AB. Rh positive individuals recorded high frequency of occurrence than Rh negative. Haemoglobin genotype AA recorded high frequency of occurrence than that of AS. Based on odd ratios, the risk for the studied subjects to be infected with *Helicobacter pylori* was in the order of B>A>O (odd ratios: A = 1.78, B = 9.01, O = 0.54), considering the fact that AB blood group subjects was negligible due to fewer number of subjects (5). Haemoglobin AS subjects were more prone to having *Helicobacter pylori* than AA subjects.

Conclusion: The study has revealed that blood group B individual are more at risk of being infected with *Helicobacter pylori* in comparison to other ABO blood groups. Additionally, those with haemoglobin AS genotype were also at risk of being infected with *Helicobacter pylori* than those with haemoglobin AA genotype.

Introduction

Infection with *Helicobacter pylori* affects more than half of the world's population (1). The infection may begin in childhood and persist for a lifetime. *Helicobacter pylori*'s transmission mechanisms remain unknown, but the human stomach is the bacterium's reservoir. Most persons frequently complain of stomach ulcer and as such there is the need to carry out studies on *Helicobacter pylori* implicated as the cause of the disease.(1)

Helicobacter pylori has multiple virulence factors, including urease (UreA), outer membrane proteins, cytotoxin-associated gene A (cagA), vacuolating cytotoxin gene A (vacA), induced by contact with epithelium (IceA), blood group antigen binding adhesin (BabA), and outer inflammatory protein (OipA) (2). The cagA and vacA genes are the most frequently identified virulence factors in clinical isolates. CagA is a potent bacterial toxin linked to both gastritis and gastric cancer (2). The cagA gene is located on the cag pathogenesis island, which generates a type 4 secretory system. The cagA gene has been linked to conditions such as peptic ulcer and gastric cancer that are characterised by significant inflammation and infection in the gastrointestinal system (3,4).

Several diagnostic methods are used to identify H. pylori infections, such as invasive methods (e.g., endoscopy and culture) followed by histopathology and/or various molecular and nucleic acid amplification tests; and non-invasive endoscopy-independent methods such as urea breath tests, faecal antigen tests, and serological tests (5). Serological tests are simple, rapid, inexpensive, and convenient among non-invasive methods. For epidemiological research, numerous studies advocated the use of serological tests, such as screening of serum immunoglobulin G (IgG) antibodies by enzyme-linked immunosorbent assay (ELISA) (6,7).

The ABO blood group system continues to be the most significant factor in human blood transfusions. The ABO gene modifies the carbohydrate content of red blood cell antigens and regulates this system. A glycosyltransferase that catalyses the transfer of nucleotide sugars to the H antigen and coordinates the production of ABO blood group antigens which is encoded by the ABO gene on chromosome 9q34 (8). Multiple studies have suggested that ABO/Rh blood types may influence the development of infectious and non-infectious diseases, such as H. pylori infections and gastroduodenal ulcers (8,9). In a prior study, eleven patients with gastroduodenal ulcers were found to have a high prevalence of the blood group O. Despite the fact that associations between ABO blood groups and risk of H. pylori infection have been investigated in multiple studies, contradictory findings in this regard have been observed (10). To fully comprehend the relationship between blood groups and H. pylori infection, more research is required.

Haemoglobin (HB) is the protein found in red blood cells that carries oxygen to the tissues and organs of the body and gives blood its red colour (11). One normal haemoglobin (HB-AA) and five variants (HB-AS, HB-SS, HB-SC, HB-AC, and HB-CC) exist (11). The inheritance of HB-S from both parents results in sickle cell anaemia/disease (SCA/SCD) in the homozygous state (HB-SS). In contrast, the inheritance of HB-S from one parent and HB-A from the other produces HB-AS, also known as sickle cell trait (SCT) One of the variants, HB-C, is created by replacing glutamic acid with lysine at the sixth position of the -globin chain and causes mild chronic haemolytic anaemia. The inheritance of the HB-C gene from both parents results in homozygosity (HB-CC). At the point of inheritance, HB-C can also combine with HB-A and HB-S to form HB-AC and HB-SC, respectively (12,13). Nigeria has the world's highest incidence of sickle cell disease, with approximately 91,011 children born with complications. Approximately 2% of all newborns are affected (14). There is a paucity of data regarding the relationship between the

haemoglobin genotype and *H. pylori* infection.

Helicobacter pylori infection is causally related to peptic ulcer disease (15-17), and should be eradicated in such settings when detected. In addition, there is evidence that H. pylori can suppress the gastric acid barrier and this is associated with the disease occurrence (18). The prevalence of H. pylori infection varies widely between countries and racial groups within the same country, according to studies (19,20). There is paucity of scientific reports on the prevalence of H. pylori infection amongst the Ogonis, and this creates a loophole in associating the infection with blood group and haemoglobin genotype variations.

Materials and Method

Study Design

This was a study carried out on a cross-section of Wiyaakara indigenes in Ogoni, to determine the prevalence of Helicobacter pylori infection and its association with ABO/Rh blood group and haemoglobin gentotype. Non-indigenes of Wiyakaara were excluded from the study and only willing indigenes of Wiyakaara origin were enrolled for the study. Informed consent was obtained from all subjects prior to enrollment.

Study Area

The study was carried out amongst indigenes of Ogoni specifically from Wiiyaakara village in Khana local government area of Rivers State. Khana is a Local Government Area in Rivers State, Nigeria. It has a population estimated to be about 294,217 at the 2006 census. The people of Ogoni are predominantly farmers and fishermen, due to the nature of their work, they spend most hours of the day working without food, and hunger is a factor to consider physiologically as regarding Helicobacter pylori infection.

Study Population

Based on convenient sampling, a total of 130 subjects (35 males and 95 females; age 18 to 68 years) who volunteered, were enrolled in the study for the assessment of ABO/Rh blood groups, haemoglobin geneotype and H. pylori seroprevalence.

Sample Collection, Storage and Transportation 2.5 ml of blood was collected by venipuncture into a K_3EDTA anticoagulated concentration of 1.2mg/ml) sample container already labelled with subject's name, sex and age. EDTA anticoagulant was used to avoid coagulation of blood since the analysis were not done on the field. Analysis was carried out within 24 hours of sample collection. Collected samples were all transported under cold chain (ice packs/crushed ice in air tight and sealed thermocontainer, 2-8^{o}C), from Wiyakaara (site of collection) to Port Harcourt (where analysis was carried out).

Methodology

Determination of ABO and Rh Blood Group by Tube Method

For ABO blood grouping, five small (63x9.5 mm) tubes were labelled 1 to 5. In tube 1, one volume of anti-A serum and one volume of 3% patient's red cells were mixed together. In tube 2, one volume of anti-B serum and one volume of 3% patient's red cells were mixed together. In tube 3, one volume of patient's serum was mixed with one volume of 3% A cells. In tube 4, one volume of patient's serum was mixed with one volume of 3% B cells. In tube 5, one volume of patient's serum was mixed with one volume of patient's 3% control – O red cells. All contents of the tubes were mixed by gently tapping the base of each tube with the finger. The tubes were allowed to react at room temperature for 5 minutes. Thereafter they were centrifuged at 1000 g for 10 seconds. The results were read by tapping gently the base of each tube, checking for agglutination or haemolysis and confirmed under the light microscope using x10 objective

For Rh blood grouping, one drop of anti-D antiserum was added in a labelled test tube, followed by one drop of patient's 3% red cell suspension. The tube was mixed and incubat-

ed in a water bath for 30minutes. Thereafter, the tube was tapped gently to redispersed the cells. It was then observed microscopically. If no agglutination, it was further treated with 22% bovine albumin, centrifuged again at 1000 g for 10 seconds to confirm Rh D negative. Positive and negative controls were run simultaneously with the test.
Interpretation of Result: Visible agglutination or haemolysis indicated a positive reaction, while absence of agglutination or haemolysis indicated negative reaction.These results are confirmed with x10 objective under the light microscope

Determination of Haemoglobin Genotype using Cellulose Acetate Paper Electrophoresis at alkaline pH 8.4 – 8.6
Samples were centrifuged at 1200 g for 5 min. 20 microlitre of the packed red cells was diluted with 150 microlitre of the haemolysing reagent, mixed gently and left for at least 5 minutes. With the power supply disconnected, the electrophoresis tank was prepared by placing equal amounts of TEB buffer in each of the outer buffer compartments. two chamber wicks were wetted in the buffer and one was placed along each divider/bridge support ensuring that they made good contact with the buffer. The cellulose acetate was soaked by lowering it slowly into a reservoir of buffer. The cellulose acetate was left to soak for at least 5 min before use. The sample well plate was filled with 5 microlitre of each diluted sample or control and covered with a 50 mm coverslip or a 'short' glass slide to prevent evaporation. A second sample well plate was loaded with Zip-prep solution. The applicator tips were cleaned immediately prior to use by loading with Zip-prep solution and then applying them to a blotter. The cellulose acetate strip was removed from the buffer and blotted twice between two layers of clean blotting paper, ensuring that the cellulose acetate was not allowed to dry. The applicator was loaded by depressing the tips into the sample wells twice and apply this first loading onto some clean blotting paper. The applicator was reloaded and samples were applied to the cellulose acetate. The cellulose acetate plates were placed across the bridges, with the plastic side uppermost. Two glass slides were placed across the strip to maintain good contact and then electrophoresed at 350 V for 25 min. After 25 min electrophoresis, the cellulose acetate was immediately transfered to Ponceau S and fixed and stainned for 5 min. Excess stain was removed by washing for 5 min in the first acetic acid reservoir and for 10 min in each of the remaining two. It was subsequently blotted once, using clean blotting paper and left to dry.

***Helicobacter pylori* Screening**
Rapid Diagnostic Testing Procedures
The test, specimen and buffer were allowed to reach room temperature (15-30°C) prior to testing. The pouch was brought to room temperature before opening it and the test device was then removed from the sealed pouch and used immediately. The test device was placed on a clean and level surface. The dropper was held vertically and 2 drops of serum or plasma (approximately 50 µL) were transferred to the specimen well (S) of the test device, then 1 drop of buffer was added to the specimen well (S). The timer was started and results were read at 10 minutes.

Positive: The presence of a coloured line in the test line region and control line region indicates the presence of Helicobacter pylori antibodies.
Negative: The absence of a coloured line in the test line region indicates the absence of Helicobacter pylori antibodies.

Statistical Analysis
The data generated from this study were analyzed using descriptive statistics and represented in tables. Odds ratio, relative risks, likelihood ratios were used to determine the risk of being infected by Helicobacter pylori. Graph-pad prism was used for the analysis.

Results
A total of 130 subjects participated in the study. Males were 35 while females were 95. Details are shown in Table 1. 48.5% tested positive for Helicobacter pylori infection. Amongst those who tested

positive, 68% were females while 32% were males. (Table 2). In ABO blood grouping, blood group O recorded the highest number of infections, followed by blood group A, blood group B and AB. Rh positive individuals recorded high number of infection than Rh negative. Haemoglobin genotype AA recorded high infection than AS (Table 3).
The likelihood for the studied subjects to be infected with Helicobacter pylori was observed to be in the order of AB>B>A>O; but considering the fact that number of AB blood group subjects was 5 and negligible, B>A>O is the statistically significant order. Haemoglobin AS subjects were more prone to having *Helicobacter pylori* than AA subjects (Table 4).

Table 1. Demographic Details of Study Participants

Parameters	Number	Percentage (%)
Total number of subjects	130	100
Total number of males	35	27
Total number of females	95	73
Age Bracket	18 – 68years	

Table 2. Frequency Occurrence and Percentage Distribution of *Helicobacter pylori* Infection in the Studied Population

Parameter	Frequency	Percentage (%)
Helicobacter pylori Infection in Total Population	63	48.5
Helicobacter pylori Infection in Females from infected population	43	68.0
Helicobacter pylori Infection in Males from infected population	20	32.0

Table 3. Frequency Occurrence of Studied Parameters and Percentage Distribution of *Helicobacter pylori* positivity Based on the Studied Parameters

Parameter	Frequency of Occurrence (Frequency of Positivity)	Percentage % of Occurrence (% of Positivity)
Blood Group A	26 (8 positive)	20.0 (12.7)
Blood Group B	11 (5 positive)	8.50 (7.9)
Blood Group AB	5 (3 positive)	3.80 (4.8)
Blood Group O	88 (47 positive)	67.7 (74.6)
Blood Group Rh Positive	128 (61 positive)	98.5 (97)
Blood Group Rh Negative	2 (2 positive)	1.5 (3)
HB Genotype AA	99 (41 positive)	76.2
HB Genotype AS	31 (21 positive)	23.8

Table 4 Odd Ratios, Relative Risks and Likelihood Ratios of Studied Parameters in Relation to Risk of Having *Helicobacter pylori* Infection

Parameters	Odd Ratio	Relative Risk	Likelihood Ratio	p-value
Blood Group A	1.78 CI: 0.7 – 4.6	1.59 CI: 0.7 – 3.2	1.50	0.2963
Blood Group B	9.01 CI: 2.4 – 29	6.51 CI: 2.2 – 17	5.4	0.0031
Blood Group AB	37.5 CI: 5.9 – 225	23.8 CI: 5.0 – 103	15.6	0.0130
Blood Group O	0.54 CI: 0.3 – 0.9	0.70 CI: 0.5 – 09	0.78	0.0462
Blood Group Rh Positive	0.01 CI: 0.0 – 0.05	0.33 CI: 0.2 – 0.4	0.48	<0.0001
Blood Group Rh Negative	257 CI: 9.6 – 6859	128 CI: 6.8 - Infinity	65.0	0.0007 (S)
HB Genotype AA	0.16 CI: 0.0 – 0.2	0.41 CI: 0.3 – 0.5	0.45	<0.0001
HB Genotype AS	6.70 CI: 2.7 – 15.8	4.41 CI: 2.3 – 8.6	2.84	<0.0001

Discussion

This study investigated the association between Helicobacter pylori infection and the ABO blood group, Rh blood group, and haemoglobin genotypes among people of Wiyaakara, Khana LGA, Rivers State.

Forty-eight (48) percent of participants were infected with H. pylori in the study, this result is similar to what Al-Balushi et al. (21) reported in Oman with a slightly higher prevalence of 62.4%. Yordanov et al. (22) in Bulgaria reported H. pylori seropositivity rate of 72.4%, which is much higher than the prevalence found in the current study. Studies in Nigeria have found varied levels of H. pylori infection in various demographics, ranging from as low as 20% in children (23) to as high as 93.6% in Nigerians with dyspepsia (23,24); Christian et al. (25) in a study in Rivers State, Nigeria also reported high infection rate. However, the findings in this study were similar with the 30.9% prevalence found by Ikpeme et al. (26) in Uyo, South-South Nigeria but lower than values of 63.6% and 82% documented by Senbanjo et al.(27) and Holcombe et al. (28) in Lagos and Maiduguri, respectively. The high frequency of H. pylori revealed in this study and other investigations remains a significant problem in developing nations due to their isolation, poor socioeconomic situations, and inadequate hygiene standards (29).

However, the risk and likelihood for the studied subjects to be infected with Helicobacter pylori from this study is in the order of AB>B>A>O; but considering the number of AB blood group subjects which was negligible, B>A>O was the statistically significant order (blood group B having odd ratio of 9.01, relative risk of 6.51 and likelihood ratio 5.4; compared to that of A: odd ratio of 1.78, relative risk of 1.59 and likelihood ratio of 1.5; and that of blood group O: odd ratio of 0.54, relative risk of 0.7 and likelihood ratio of 0.78). The finding of this study takes cognizance of the fact that blood group O is highly distributed in the study population and this does not translate automatically to risk of infection despite having more of O subjects being positive for Helicobacter pylori. This observation was similar to Nwodo et al. (30) where the

order of H. pylori prevalence was B>O>A>AB with an odd ratio of 2.00. This observed pattern based on risk ratios from the current study varies from those of other studies by Inoue et al. (31) with the trend O>B>A>AB; Gasim et al. (32) with the trend O>A>B>AB, as blood group O posed a higher risk of infection with H. pylori. This discrepancy could be attributed to the imbalance in the distribution of ABO blood groups in the sampled population, and probably due to the receptor for H. pylori in Lewis b subjects exposed only in those who are blood group O (33), and considering the fact that Lewis b antigens are present amongst the Ogonis (34).

The study also reported the distribution of H. pylori based on Rh blood group with Rh D positive individuals recording higher frequency (98.5%) of occurrence than Rh D negative (1.5%) with odd ratios of 0.01 and 257 respectively. The odd ratios also do not translate to infectivity rate due to number of subjects with Rh negative individuals. This observation corroborated with the findings of Mbadiwe et al. (35) where a higher prevalence of H. pylori seropositivity was reported in Rh positive subjects than their Rh D negative counterpart. The present study was also in line with the observations by Khosravi et al. (36). This finding could be as a result of the dominance of Rh D blood group in the population. As regards haemoglobin genotype, haemoglobin genotype AA (76.2%) recorded high frequency of occurrence than that of AS (23.8%). There is a dearth of literature on the association of Helicobacter pylori infection with haemoglobin variants, however a similar study in Rivers State, Nigeria (25) showed that there was a higher frequency of haemoglobin genotype AA than AS and SS which corroborated with the present study, though the current study did not identify haemoglobin genotype SS or its association with H. pylori infection. The scientific and physiological rationale for the observed distribution remains to be seen as there is a paucity of literature on the aforementioned and also due to the fact the subjects with haemoglobin geneotype SS were not recruited for the study.

Conclusion

Rh D positive and Rh D negative patients did not yield a conclusive variation on the distribution of H. pylori infection, same as haemoglobin variants; whereas O blood group individuals had a greater percentage of Helicobacter pylori infection, blood group B subjects were more susceptible to Helicobacter pylori infection in comparison to other ABO blood groups. Additionally, those with haemoglobin AA were mostly afflicted with Helicobacter pylori infection in the study area however, AS individual were more susceptible. Based on odd ratios, relative risk and likelihood ratio, the probability of having H. pylori infection associating with ABO blood group, was observed to be in the order of B>A>O.

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