



ABO BLOOD GROUPS AND ABH ANTIGENS IN PULMONARY TUBERCULOSIS

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Article Received on 31/08/2016

Article Revised on 21/09/2016

Article Accepted on 11/10/2016

ABSTRACT

While there are conflicting reports on the association between ABO blood groups and pulmonary tuberculosis and non-secretion of ABH substances has been reported to be associated with tuberculosis, there is dearth of information on the association between ABO blood groups, secretion of ABH antigens and pulmonary tuberculosis. This study determined whether there was any relationship between ABO blood groups, secretor status and pulmonary tuberculosis in Osogbo, Southwestern Nigeria. A total of 295 individuals comprising 175 tuberculosis patients (test group) and 120 apparently healthy subjects (control group) participated in this study. ABO blood group was determined by tube and tile techniques and secretor status by haemagglutination inhibition test. There was no association between ABO blood groups and pulmonary tuberculosis ($\chi^2 = 2.25$, $df = 3$, $p = 0.52$). Non-secretion of ABH substances was significantly higher in tuberculosis patients than in control subjects ($\chi^2 = 8.93$, $df = 1$, $p = 0.003$). The frequency of non-secretor in blood group O individuals was significantly lower compared to blood groups A ($p < 0.0001$), B ($p = 0.02$) and A+B+AB ($p = 0.001$) individuals. Non-secretion of ABH antigens is associated with pulmonary tuberculosis and of the non-secretors; group O non-secretors were less associated with tuberculosis than non-O-non-secretors due to lower frequency of non-secretors among group O individuals.

KEYWORDS: ABO blood groups; ABH antigens; secretors; non-secretors; tuberculosis.

INTRODUCTION

The relationship between ABO blood group and a number of infectious diseases had been studied; blood group O phenotypes were reported to be more susceptible to severe form of cholera than non-O blood group phenotypes,^[1, 2] group O individuals were reported to be more prone to gastrointestinal infection caused by *Escherichia coli*^[3] but more resistant to severe malaria than non-O blood groups.^[4, 5] Reports on association between pulmonary tuberculosis and ABO blood groups had been conflicting. While some reported lack of association between pulmonary tuberculosis and ABO blood group system^[6, 7], others reported significant association between them.^[8-10]

Like ABO blood group, ABH secretor status had been related to the susceptibility or otherwise of a number of infectious diseases; non-secretors had been significantly associated with infections caused by bacteria such as *Haemophilus influenza*,^[11] *Neisseria meningitides*, *Streptococcus pneumonia*,^[12] *Escherichia coli*,^[13] fungal infection such as candidiasis^[14] and *Plasmodium* infection^[15] than secretors. On the otherhand, secretors

had been reported to be more prone to viral infections such as norovirus,^[16] influenza virus, rhinovirus, respiratory syncytial virus, echovirus^[17] and HIV^[18-20] than non-secretors.

Previous studies on ABH secretor status on apparently healthy individuals revealed that group O individuals were significantly more of secretors than non-O group individuals^[21, 22] which explained why O individuals are less susceptible to certain diseases or disorders.^[21] In this study, we hypothesized that group O individuals were less associated with pulmonary tuberculosis than non-O group due to lower frequency of non-secretor phenotype among the former than the latter.

MATERIALS AND METHODS

This study was carried out in Osogbo, Southwestern Nigeria. The test participants were drawn from patients attending tuberculosis clinics of the State teaching hospital, Asubiaro, Osogbo while the control subjects were apparently healthy members of staff and students of the hospital. A total of 295 individuals comprising 175 tuberculosis patients and 120 controls of age ≥ 16 years

participated in the study. Informed consent was obtained from each participant and ethical approval was obtained from the Ethical Committee of the College of Health Sciences, Ladoke Akintola University of Technology, Osogbo.

A sample of 2 mL of venous blood was collected from each participant into ethylenediaminetetraacetic acid (EDTA) bottle. ABO blood group antigens tests along with controls were performed by standard tube and tile techniques as described elsewhere^[22]. Also, 2 mL of saliva was collected from each participant for determination of secretor status. Secretor and non-secretor phenotypes were done using the haemagglutination inhibition test as described elsewhere.^[22] Laboratory investigations were carried out in the Research Laboratory, Department of Biomedical Sciences, College of Health Sciences, Ladoke Akintola University of Technology, Osogbo, Nigeria.

Statistical Analysis

The statistical package for social sciences software package (SPSS version 14) was used for statistical analysis. Differences between percentages and proportions were tested by Chi-square test. A p-value of < 0.05 was considered to be significant.

RESULTS

A total of 175 tuberculosis patients (84 males and 91 females) and 120 controls (62 males and 58 females) of age ≥ 16 years participated in this study. The sex and age distributions of the study population are given in Table 1. The distributions of males and females in the test and control groups were not significantly different ($\chi^2 = 0.38$, $df = 1$, $p = 0.54$). Similarly, there was no significant difference in the distribution of age between the test and control groups ($\chi^2 = 0.58$, $df = 3$, $p = 0.99$).

Also, the distributions of ABO blood groups and secretor status among the test and control subjects are given in Table 1. The distributions of ABO blood groups between the test and control groups were not significantly different ($\chi^2 = 2.25$, $df = 1$, $p = 0.52$). Of the 175 tuberculosis patients, 110 (62.9%) and 65 (37.1%) were secretors and non-secretors respectively while 95 (79.2%) and 25 (20.8%) of the controls were secretors and non-secretors respectively. Non-secretors in the test group were significantly higher than non-secretors in the control group ($\chi^2 = 8.93$, $df = 1$, $p = 0.003$, OR 2.55; 95% C.I 1.37-4.73). Therefore, non-secretion of ABH substances was significantly associated with pulmonary tuberculosis.

The distributions of non-secretors among the tuberculosis patients and control subjects are given in Table 2. The distributions of non-secretors varied significantly with ABO blood group both in the test ($\chi^2 = 17.0$, $df = 1$, $p < 0.0001$) and control ($\chi^2 = 7.88$, $df = 1$, $p = 0.049$) subjects. Also, analysis showed that group A non-secretors who had tuberculosis (18/27) were significantly higher than their control counterparts (08/25) ($\chi^2 = 6.24$, $df = 1$, $p = 0.01$). Similarly, group O non-secretors who had tuberculosis (25/98) were significantly higher than their control counterparts (07/63) ($\chi^2 = 4.04$, $df = 1$, $p = 0.04$).

In the tuberculosis group, there was a significant decrease in the incidence of non-secretor in blood group O individuals compared to blood groups A ($p < 0.0001$), B ($p = 0.02$) and A+B+AB ($p = 0.001$) individuals (Table 3). Similarly, in the control group, there was a significant decrease in the incidence of non-secretor in blood group O individuals compared to blood groups A ($p = 0.04$) and A+B+AB ($p = 0.006$) individuals (Table 4). Therefore group O individuals were significantly less associated with non-secretors compared to non-group O individuals in both the tuberculosis group and control group.

Table 1: Distribution of the Study Participants by Sex, Age, ABO blood group and Secretor Status

Variable	Test group (%) n=175	Control group (%) n=120	Total n=295	χ^2	p
Sex ^a				0.38	0.54
Female	91 (52.0)	58 (48.3)	149 (51.3)		
Male	84 (48.0)	62 (51.7)	146 (48.7)		
Age ^b (years)				0.58	0.99
16 – 20	20 (11.4)	11 (9.2)	31 (10.5)		
21 – 30	52 (29.7)	36 (30.0)	88 (29.8)		
31 – 40	39 (22.3)	28 (23.3)	67 (22.7)		
41 – 50	29 (16.6)	20 (16.7)	49 (16.6)		
51 – 60	22 (12.6)	17 (14.2)	39 (13.2)		
≥ 61	13 (7.4)	08 (6.7)	21 (7.1)		
ABO blood group ^c				2.25	0.52
A	27 (15.4)	24 (20.0)	51 (17.3)		
B	37 (21.1)	27 (22.5)	64 (21.7)		
AB	13 (7.4)	04 (3.3)	17 (5.8)		

O	98 (56.0)	65 (52.2)	163 (55.2)		
Secretor status ^d				8.93	0.003
Secretor	110 (62.9)	95 (79.2)	205 (69.5)		
Non-Secretor	65 (37.1)	25 (20.8)	90 (30.5)		

^a $\chi^2 = 0.38$, df = 1, p = 0.54, ^b $\chi^2 = 0.58$, df = 5, p = 0.99, ^c $\chi^2 = 2.25$, df = 3, p = 0.52, ^d $\chi^2 = 8.93$, df = 1, p = 0.003

Table 2: Distributions of Non-secretors among the tuberculosis and control Subjects by ABO Blood Group

ABO blood group	Tuberculosis Patients NS/NS+S (%)	Control Subjects NS/NS+S (%)	Total NS/NS+S (%)	χ^2	p-value
A	18/27 (66.7)	08/25 (32.0)	26/52 (50.0)	6.24	0.01
B	17/37 (45.9)	08/27 (29.6)	25/64 (39.1)	1.75	0.19
AB	05/13 (38.5)	02/05 (40.0)	07/18(38.9)	0.23	0.63
O	25/98 (25.5)	07/63 (11.1)	32/161(56.0)	4.04	0.04
Total	65/175(62.9)	25/120(20.8)	90/295(30.5)	8.93	0.003
p-value	<0.0001	0.049			

Table 3: Comparison of A, B, AB and O Non-Secretors among the Pulmonary Tuberculosis Patients

ABO non-secretor	A (18/27) (66.7%)	B (17/37) (45.9%)	AB (5/13) (38.5%)	A+B+AB (40/77) (51.9%)
A (18/27) (66.7%)	---	p = 0.10	p = 0.09	p = 0.18
B (17/37) (45.9%)	p = 0.10	---	p = 0.64	p = 0.55
AB (5/13) (38.5%)	p = 0.09	p = 0.64	---	p = 0.37
O (25/98) (25.5%)	p < 0.0001	p = 0.02	p = 0.51	p = 0.0003

Table 4: Comparison of A, B, AB and O Non-Secretors among the Control Subjects

ABO non-secretor	A (08/25) (32.0%)	B (08/27) (29.6%)	AB (02/05) (40.0%)	A+B+AB (18/57) (31.6%)
A (08/25) (32.0%)	---	p = 0.85	p = 0.86	p = 0.97
B (08/27) (29.6%)	p = 0.85	---	p = 0.95	p = 0.86
AB (02/05) (40.0%)	p = 0.86	p = 0.95	---	p = 0.91

O
(07/63)
(11.1%)

p < 0.04 p = 0.06 p = 0.25 p = 0.006

DISCUSSION

In Nigeria, we are not aware of any research findings relating ABO blood group and secretor status with tuberculosis. We tested the hypothesis that group O non-secretors were less associated with pulmonary tuberculosis than non-O group non-secretors due to low frequency of non-secretor phenotype among blood group O individuals.

The present study showed that the result of secretor status in the control group was in line with those of previous studies carried out in the same locality^[15, 20, 22]. The result of secretor status in the test group showed that non-secretors were more significantly associated with pulmonary tuberculosis than secretors. The susceptibility of non-secretors could be linked to the presence of Le^a antigens in greater amounts on the epithelial surfaces of non-secretor which might be receptors for *Mycobacterium tuberculosis*. On the other hand, the study showed lack of association between ABO blood group and pulmonary tuberculosis. Previous studies on whether or not there was association had been conflicting^[6-10]. These conflicting reports on ABO blood group and pulmonary tuberculosis might be a function of the secretor status of the participants.

While the present study showed a lack of association between ABO blood groups and tuberculosis and an association between non-secretors of ABH substances and tuberculosis; when combined, the non-O group, non-secretors were found to be more prone to tuberculosis compared to group O non-secretors. Group O individuals who were non-secretors were significantly lower in both the control and test groups and these supported the findings that group O individuals were significantly less associated with non-secretors compared to the other blood groups^[21,22]. This might be responsible for the significantly lower prevalence of tuberculosis observed in group O individuals who were non-secretors compared to non-O blood group individuals who were non-secretors. These findings suggest that the low frequency of non-secretion of ABH substances among group O individuals is responsible for the low occurrence of tuberculosis in this group compared to the non-O individuals.

CONCLUSION

This study shows that there is an association between non-secretions of ABH antigens and pulmonary tuberculosis. Of the non-secretors, group A non-secretors are the most associated with pulmonary tuberculosis followed by group B while group O non-secretors are the least associated. The protection afforded by group O individuals against pulmonary tuberculosis is linked to the low frequency of non-secretors among this group.

REFERENCES

1. Harris JB, Khan AI, LaRocque RC, Dorer DJ, Chowdhury F, Faruque AS, Ryan ET, Qadri F, Calderwood SB. Blood group, immunity, and risk of infection with *Vibrio cholerae* in an area of endemicity. *Infect Immun*, 2005; 73(11): 7422-7427.
2. Glass RI, Holmgren J, Haley CE, Khan MR, Svennerholm AM, Stoll BJ, Balayet-Hossain KM, Black RE, Yunus M, Barua D. Predisposition for cholera of individuals with O blood group. Possible evolutionary significance. *Am J Epidemiol*, 1985; 121(6): 791-796.
3. Blackwell CC, Dundas S, James VS, Mackenzie DAC, Braun JM, Alkout AM, Todd WTA, Elton RA, Weir DM. Blood group and susceptibility to disease caused by *Escherichia coli* O157. *J Infect Dis*, 2002; 185(3): 393-396.
4. Fry AE, Griffiths MJ, Auburn S, Diakite M, Forton JT, Green A, Richardson A, Wilson J, Jallow M, Sisay-Joof F, Pinder M, Peshu N, Williams TN, Marsh K, Molyneux ME, Taylor TE, Rockett KA, Kwiatkowski DP. Common variation in the ABO glycosyltransferase is associated with susceptibility to severe *Plasmodium falciparum* malaria. *Hum Mol Genet*, 2008; 17(4): 567-576.
5. Rowe JA, Opi DH, Williams TN. Blood groups and malaria: fresh insights into pathogenesis and identification of targets for intervention. *Curr Opin Hematol*, 2009; 16(6): 480-487.
6. Reddy VD, Usha T. The ABO blood groups and pulmonary tuberculosis in the Warangal district of the Telangana region. *J Indian Med Assoc*. 1990; 88(12): 337-38.
7. Reddy VD, Reddy M. Pulmonary tuberculosis and the ABO and the Rh blood groups in the Chittoor district of Rayalaseema in Andhra Pradesh. *J NTR UHS*. 1998; 3: 19-22.
8. Jain RC. The ABO blood groups and pulmonary tuberculosis. *Tubercule*, 1970; 51(3): 322-323.
9. Gondaliya ST, Makwana HH, Lakum NR, Agnihotri AS. Pulmonary tuberculosis and blood groups: any association? *Gujarat Med J*, 2012; 67(2): 39-41.
10. Rao BN, Reddy VD, Sahu PS, Veerendra KA, David MA, Yugandhar P, Muralishwar Rao J. the ABO blood group distribution and pulmonary tuberculosis. *J Clin Diag Res*. 2012; 6(6): 943-946.
11. Blackwell CC, Jonsdottir K, Hanson M, Weir DM. Non-secretion of ABO blood group antigens predisposing to infection by *Haemophilus influenzae*. *Lancet*, 1986a; 2: 687.
12. Blackwell CC, Jonsdottir K, Hanson M, Tood WT, Weir DM. Non-secretion of ABO blood group antigens predisposing to infection by *Neisseria meningitidis* and *Streptococcus pneumoniae*. *Lancet*, 1986b; 2: 284-285.

13. Sheinfeld J, Schaeffer AJ, Cordon-Cardo C, Rogatko A, Fair WR. Association of the Lewis blood group phenotype with recurrent urinary tract infections in women. *New Engl J Med*, 1989; 320: 773-777.
14. Thom SM, Blackwell CC, MacCallum CJ, Weir DM, Kinane DF, Wray D. Non-secretion of ABO blood group antigens and susceptibility to infection by *Candida* species. *FEMS Microbiol Immunol* 1989; 7: 401-406.
15. Igbeneghu C, Olisekodiaka MJ, Okanlawon BM, Onuegbu JA, Odaibo AB. (2015). Non-secretors of ABH Antigens are Susceptible to Falciparum Malaria. *SJAMS*, 3(5A): 1838-1841.
16. Thorven M, Grahn A, Hedlund K, Johansson H, Wahlfrid C, Larson G, Svensson L. Homozygous nonsense mutation (428G-A) in the human secretor (FUT2) gene provides resistance to symptomatic norovirus (CG11) infection. *J Virol*, 2005; 79: 15351-15355.
17. Raza MW, Blackwell CC, Molyneaux P, James VS, Ogilvie MM, Inglis JM, Weir DM. Association between secretor status and respiratory viral illness. *BMJ*, 1991; 303: 815-818.
18. Ali S, Niang MAF, N'doye I, Critchlow CW, Hawes SE, Hill AVS, Kiviat NB. Secretor polymorphism and human immunodeficiency virus infection in Senegalese women. *J Infect Dis*, 2000; 181: 737-739.
19. Kindberg E, Hejdeman B, Bratt G, Wahren B, Lindblom B, Hinkula J, Svensson L. A non-sense mutation (428G-A) in the fucosyltransferase FUT2 gene affects the progression of HIV 1 infection. *AIDS*, 2006; 20: 685-689.
20. Igbeneghu C, Odaibo GN, Olisekodiaka JM, Folarin OR, Oseni BSA. (2016). ABO Blood Group and Secretor Status in HIV Infection in Osogbo, Southwestern Nigeria. *EJBGE*, 3(1): 1-7.
21. Jaff MS. Higher frequency of secretor phenotype in O blood group-its benefits in prevention and /or treatment of some diseases. *Intern J Nanomed*, 2010; 5: 901-905.
22. Igbeneghu C, Olisekodiaka JM, Alabi T, Onuegbu JA, Oseni BA, Odaibo AB. (2015). ABH Secretors Status in Osogbo, Southwestern Nigeria. *Indian J Fund Appl Life Sci* 5(3): 42-47.