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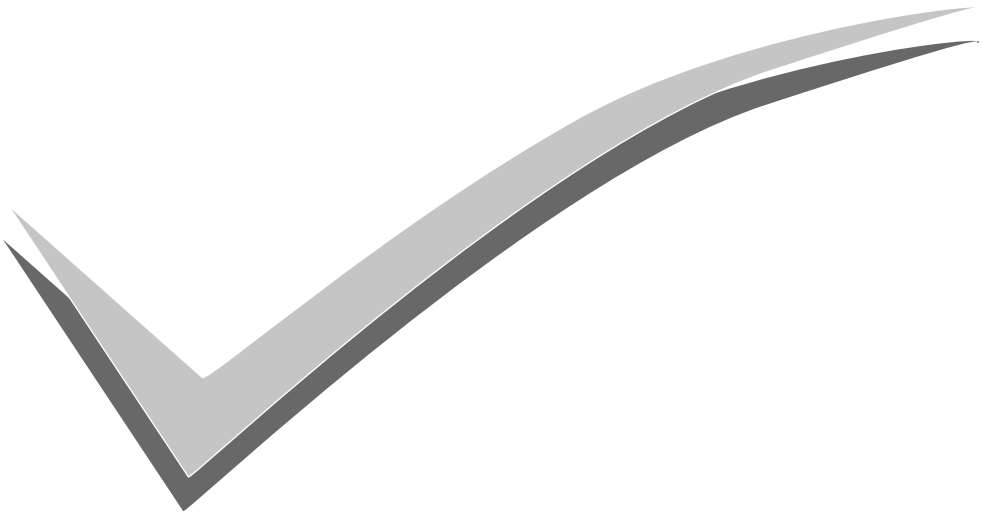

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ABO/Rho(D) Forward Grouping Reagents

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Original Article

Distribution of ABO and Rh-D Blood Groups Among Blood Donors: Western India Data

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Abstract

Introduction: The distribution of ABO and Rhesus (Rh) - D blood groups varies from one population to another. The ABO and Rh blood groups play an integral role in blood transfusion service. They also have an important and useful role in population genetic studies and certain medico-legal cases. We carried out this study with the aim of determining the distribution of ABO and Rh blood groups among blood donors. This would also help in the planning of the ever increasing demand for safe blood and blood products.

Aim: This study was undertaken with the aim of determining the distribution of ABO and Rhesus blood groups among blood donors in a tertiary care hospital in Western India.

Materials and Methods: A retrospective study was performed over 5 years. The ABO blood grouping and Rhesus typing were done by tube method using venous blood samples.

Results: The most common blood group was "O" (32.38%), followed by "B" (31.40%), "A" (26.70%), and "AB" (9.51%). The frequency of Rh positive group (94.62%) is more than Rh negative group (5.38%).

Key words: ABO groups, Blood donors, Blood transfusion, Rhesus groups

INTRODUCTION

The ABO blood groups and Rhesus (Rh) blood group antigens are the most frequently studied genetic markers. Although the antigens involved in ABO and Rh blood groups are stable throughout the life, ABO and Rh genes and phenotype vary widely across different races and geographical areas.[1] The aim of any blood transfusion to the patient is that it should be beneficial to the patient. This is possible when we provide the patient with donor red cells that optimally survive after transfusion and serve their function.[2] The distribution of ABO and Rh blood groups is important for the effective management of blood banks.[3] The ABO blood group system was the first human blood group system discovered by Landsteiner in 1900.[4] The ABO blood group system is the only system in which antibodies are constantly present in the serum of human beings whose red cells lack the antigens. Depending on whether Rh antigen is present on red cells or not, Rh phenotype is classified as Rh - D positive and Rh-D negative. Although all individuals share the same blood group system, they differ in the frequencies of a specific type.[5] ABO and Rhesus (Rh) groups varies markedly in different parts of the world. Karl Landsteiner discovered the blood groups ABO and classified it into A, B, and O groups. Blood group AB was discovered by Landsteiner's associate, Von Decastello and Sturli in 1902. The Rh (D) antigens have greater immunogenicity than all other red cell antigens except A and B antigens. Transfusion of ABO-incompatible blood can be associated with acute intravascular hemolysis, renal failure and death. So in the blood bank, every blood donation is screened for ABO and Rhesus factor. This

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study was conducted with the aim of determining the distribution of ABO and Rhesus blood groups among blood donors.

MATERIALS AND METHODS

This was a retrospective study conducted over a period of 5 years. Venous blood was collected in EDTA and plain clean vacutainer tubes and allowed to clot naturally at room temperature. The ABO blood Grouping and Rhesus typing were determined by tube method. Forward grouping was carried out using monoclonal anti- sera; anti-A, anti-B, anti-AB, and anti-D (Eryclone, tulip diagnostics Ltd.). Results of ABO grouping were confirmed by reverse grouping using known A and B red cells. Rhesus negativity was confirmed by repeat testing and by Du-gel cards (DiaMed - ID, Coombs Anti-IgG cards).

Each antiserum was validated before using it including titer and avidity of each new lot. For reverse group testing, cells were pooled from three different known donor samples. These pooled cells were prepared daily using pretested known blood group samples.

Statistics

After data collection, data entry was done in Excel. Data analysis was performed with the help of EpI Info version 8. Qualitative data have been presented with the help of frequency, and percentage table and association among various study parameters was assessed with Chi-square test. $P < 0.05$ was taken as significant.

Table 1: Total blood collection and sex distribution of donors

Year	Total donors (%)	Male (%)	Female (%)
2007	14810 (19.32)	13736 (17.92)	1074 (1.40)
2008	15394 (20.08)	14128 (18.43)	1266 (1.65)
2009	14849 (19.37)	13289 (17.34)	1560 (2.04)
2010	15820 (20.64)	14703 (19.18)	1117 (1.46)
2011	15780 (20.59)	14507 (18.93)	1273 (1.66)
Total	76653	70363 (91.79)	6290 (8.21)

RESULTS

We studied ABO and Rh blood groups in 76,653 donors composed of 91.79% male and 8.21% female donors as shown in Table 1. They were in the age group of 18-60 years. The distribution of ABO and Rh groups is shown in Table 2. Out of 76,653 donors, the most common blood group was "O" (32.38%), followed by "B" (31.40%), "A" (26.70%), and "AB" (9.51%) which was found to be statistically significant ($P < 0.05\%$). Rh positivity was found in 72,526 (94.62%) donors while 4,127 (5.38%) donors were Rhesus negative.

DISCUSSION

Although ABO and Rh genes and phenotypes are stable throughout the life, they vary widely across races and various geographical boundaries. These genes and phenotypes also have different biochemical compositions.[6] The polymorphism in these blood group system is important in population genetic studies, in evaluating the probability of hemolytic disease in the newborn, resolving paternity dispute cases and for forensic purposes. The distribution of ABO and RH-D phenotypes in different populations has been extensively studied. These blood group systems are not only important in blood transfusions but also associated with different diseases including cardiovascular diseases, organ transplantation, and erythroblastosis in neonates. Blood transfusion is a life-saving procedure but can cause acute and delayed complications. Complications of blood transfusions with wrongly labeled blood groups may be mild or can be life-threatening. Rh system found to be second most important blood group system due to hemolytic disease of newborn. The importance of Rh system has found in Rh-D negative individuals in subsequent transfusions once they develop Rh antibodies. This D antigen is the most important in transfusion practice in which the person whose red cell lacks the D antigen do not regularly have anti-D in their serum. The aim of this study was to determine the distribution

Table 2: Group wise distribution of blood donors

Year	Blood Group A (%)	Blood Group B (%)	Blood Group AB (%)	Blood Group O (%)	Rhesus positive (%)	Rhesus negative (%)	Total donors (%)
2007	3971 (5.18)	4623 (6.03)	1363 (1.78)	4853 (6.33)	14005 (18.27)	805 (1.05)	14810 (19.32)
2008	4077 (5.32)	4843 (6.32)	1512 (1.97)	4962 (6.47)	14534 (18.96)	860 (1.12)	15394 (20.08)
2009	4003 (5.22)	4641 (6.05)	1427 (1.86)	4778 (6.23)	14033 (18.31)	816 (1.06)	14849 (19.37)
2010	4189 (5.46)	5006 (6.53)	1382 (1.80)	5243 (6.84)	14990 (19.56)	830 (1.08)	15820 (20.64)
2011	4229 (5.52)	4955 (6.46)	1609 (2.10)	4987 (6.51)	14964 (19.52)	816 (1.06)	15780 (20.59)
Total	20469 (26.70)	24068 (31.40)	7293 (9.51)	24823 (32.38)	72526 (94.62)	4127 (5.38)	76653

of ABO and Rhesus blood group among blood donors. In our study, the most common blood group was "O" (32.38%) followed by "B" (31.40%), "A" (26.70%), and "AB" (9.51%) which is comparable with other studies. The study by Nag and Das[5] in West Bengal population were also observed that "O" blood group (34.8%) was common followed by blood groups "B" (33.6%), "A" (23.9%), and "AB" (7.7%) which is comparable with our study. Studies by Anjali *et al*.,[7] Periyavan *et al*.,[8] Enosolease and Bazuaye,[9] Das *et al*.,[10] Mwangi[11] also showed that blood group "O" was the most common followed by group "B," "A" and "AB" which is comparable with this study and also the findings regarding occurrence of Rh typing was almost in agreement to that from our study. In our study, Rh positivity was found in 72,526 (94.62%) donors while 4,127 (5.38%) donors were Rhesus negative. The study by Randriamanantany *et al*.[12] also showed that Rh positive was by far the most prevalent which is comparable with our study. Other studies conducted by Hamed *et al*.[13] showed 94.23% Rh (D) positivity and 5.77% Rh (D) negativity while Thakral *et al*.[14] showed 93.39% Rh (D) positivity and 5.56% Rh (D) negativity which is comparable with our study.

CONCLUSION

We established that among the various ABO and Rh-D blood groups, blood group "O" is the most common followed by blood groups "B," "A" and "AB" with a predominance of Rh positivity. In addition to compatibility test in blood transfusion practice, knowledge of the blood group distribution is also important for geographical information, genetic studies and for forensic studies in the population.

REFERENCES

1. Agrawal A, Tiwari AK, Mehta N, Bhattacharya P, Wankhede R, Tulsiani S, *et al*. ABO and Rh (D) group distribution and gene frequency; the first multicentric study in India. Asian J Transfus Sci 2014;8:121-5.
2. Kahar MA, Patel RD. Phenotype frequencies of blood group systems (Rh, Kell, Kidd, Duffy, MNS, P, Lewis, and Lutheran) in blood donors of south Gujarat, India. Asian J Transfus Sci 2014;8:51-5.
3. Kaur H, Khanna A, Manjari M, Khanna M. Prevalence of ABO blood groups and rhesus (Rh) factor in the population residing in and around Amritsar, Punjab (a 4-year study from June 2007 to June 2011). Asian J Transfus Sci 2013;7:159.
4. Chandra T, Gupta A. Frequency of ABO and rhesus blood groups in blood donors. Asian J Transfus Sci 2012;6:52-3.
5. Nag I, Das SS. ABO and Rhesus blood groups in potential blood donors at Durgapur Steel city of the district of Burdwan, West Bengal. Asian J Transfus Sci 2012;6:54-5.
6. Agarwal N, Thapliyal RM, Chatterjee K. Blood group phenotype frequencies in blood donors from a tertiary care hospital in north India. Blood Res 2013;48:51-4.
7. Anjali H, Issac A, Anjali MR, Anish TS. Transfusion-transmissible infections among voluntary blood donors at government medical college Thiruvananthapuram, Kerala, India. Asian J Transfus Sci 2012;6:55-6.
8. Periyavan S, Sangeetha SK, Marimuthu P, Manjunath BK, Seema DM. Distribution of ABO and Rhesus-D blood groups in and around Bangalore. Asian J Transfus Sci 2010;4:41.
9. Enosolease ME, Bazuaye GN. Distribution of ABO and Rh-D blood group in the Benin area of Niger-delta: Implication for regional blood transfusion. Asian J Transfus Sci 2008;2:3-5.
10. Das PK, Nair SC, Harris VK, Rose D, Mammen JJ, Bose YN, *et al*. Distribution of ABO and Rh-D blood groups among blood donors in a tertiary care centre in South India. Trop Doct 2001;31:47-8.
11. Mwangi J. Blood group distribution in an urban population of patient targeted blood donors. East Afr Med J 1999;76(11):615-8.
12. Randriamanantany ZA, Rajaonatahina DH, Razafimanantsoa FE, Rasamindrakotroka MT, Andriamahenina R, Rasoarilalamanarivo FB, *et al*. Phenotypic and allelic profile of ABO and Rhésus D blood group system among blood donor in Antananarivo. Int J Immunogenet 2012;39:477-9.
13. Hamed CT, Bollahi MA, Abdelhamid I, Med Mahmoud MA, Ba B, Ghaber S, *et al*. Frequencies and ethnic distribution of ABO and Rh(D) blood groups in Mauritania: Results of first nationwide study. Int J Immunogenet 2012;39:151-4.
14. Thakral B, Saluja K, Sharma RR, Marwaha N. Phenotype frequencies of blood group systems (Rh, Kell, Kidd, Duffy, MNS, P, Lewis, and Lutheran) in north Indian blood donors. Transfus Apher Sci 2010;43:17-22.

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Frequency of ABO and Rhesus Blood Groups: A Study among the donors of Sir.T.Hospital Bhavnagar, Gujarat

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ABSTRACT

Background & Objectives: *The ABO blood group system was the first human blood group system to be discovered by Landsteiner in 1900. The second type of blood group is the rhesus system. There are only two Rh phenotype such as Rh positive and Rh negative, depending on whether Rh antigen is present on the red cell or not. The frequency of ABO and Rh phenotypes in different populations has been extensively studied. The present study was done to assess the prevalence of blood groups in different categories of bhavnagar and to compare our results with other studies conducted in India and elsewhere in the world and its multipurpose future utilities for the health planner.*

Materials and Method: *A retrospective study was carried out on 40416 blood donors (male and female) during a period of three year from 1st January2012 to 31st December 2014, donors were selected and screened for study in the sir. T. Hospital Blood Bank, Bhavnagar Gujrat. India. . Each sample of donors was tested for ABO and Rhesus group status using antisera (Eryclone Monoclonal ABO/Rh, Tulip Diagnostic Ltd. Goa, India) combined slide and tube method*

Results: *The frequency of various blood group according to present study, from table -1: B+ve 33.4%, O+ve 29.4%, A+ve 21.86%, AB+VE 8.9%, B-ve 2.3%, O-ve 2%, A-ve 1.46% & rarest being AB-ve 0.7% Also from Table 2: Rh-Group positive are 93.57% and Rh –Neg less common with 6.42% prevalence Conclusion group "B" is most common Blood group in Bhavnagar population followed by "O"A,and AB Blood Group, also Rh-Group positive are 93.57% and Rh –Neg less common with 6.42% prevalence*

Keywords: *blood donor ,blood group ,ABO,Rhesus (Rh)*

INTRODUCTION

The ABO blood group system was the first human blood group system to be discovered by Landsteiner in 1900. The ABO blood group system is the only system in which antibodies are consistently and predictably present in the serum of normal individuals whose red cells lack the antigens[1] The second type of blood group is the rhesus system. There are only two Rh phenotype such as Rh positive and Rh negative, depending on whether Rh antigen is present on the red cell or not. Determination of ABO blood groups is done by detecting A and B antigens. In addition, known red cells are used to detect anti-A and anti-B in

the serum, by a process called 'reverse' grouping. ABO and Rh gene phenotypes vary widely across races and geographical boundaries [2,3,4] Together these two systems have proved to be the most important for blood transfusion purposes. In modern medicine, the need for blood group frequency and prevalence studies is multipurpose, as besides their importance in evolution, their relation to disease and environment is being increasingly important [5,6]. Blood groups are genetically determined. The vast majority are inherited in a simple Mendelian fashion and are stable characteristics which are useful in paternity testing.[7] group is essential for effective management of blood banks inventory, be it a facility of a smaller local transfusion service or a regional or national transfusion service. It is, therefore, imperative to have information on the distribution of these blood groups in any population.[8]

The present study was done to assess the prevalence of blood groups in different categories of Sir Takhtasinhji general hospital, bhavnagar India and to compare our results with other studies conducted in India and elsewhere in the world and its multipurpose future utilities for the health planners.

OBJECTIVES

This study is aimed to determine frequency and distribution ABO and Rh blood group patterns among blood donors in the sir T. Hospital Blood Bank, Bhavnagar, Gujarat and compare with other data from similar studies within the India and all over the world.

MATERIALS AND METHOD

A retrospective study was carried out on 40416 blood donors (male and female) during a period of one year from 1st January2012 to 31st December 2014 in the sir. T. Hospital Blood Bank, Bhavnagar Gujrat. India the blood donors were selected after taking a detailed history and a complete examination regarding their eligibility criteria for blood donation. Donor's name, age, sex, occupation, caste, complete postal address and contact number was taken. Donors were deferred or accepted according to their medical history regarding chronic or acute diseases. Findings were further confirmed by physical examination of the patient. Blood was taken from a donor only after fulfilling all the eligibility criteria of a healthy donor. Blood was taken for donors who were between 18-60 years of age, more than 45 kg weight with hemoglobin more than 12.5 g%. The donors have no history of any disease, infection or recent treatment. Written consent was also taken from them prior to donation regarding their acceptability for the tests to be carried out for the transfusion transmitted diseases. The Blood samples were obtained by standard procedures of venupuncture and subjected to determination of ABO and Rhesus blood group using antisera (Eryclone Monoclonal ABO/Rh, Tulip Diagnostic Ltd. Goa, India) by combined slide and test tube method. Each sample of donors was tested for ABO and Rhesus status

OBSERVATION AND RESULTS

Table -1: Distribution of ABO and Rh Blood group system

Group	Total Donors 2012	%	Total Donors 2013	%	Total Donors 2014	%
A+VE	2535	22.4	2932	21.4	3385	21.8
A-VE	164	1.4	205	33.3	240	1.5
B+VE	3769	33.2	4554	8.7	5177	33.7
B-VE	277	2.5	306	30.0	346	2.1
AB+VE	1002	8.8	1192	1.5	1412	9.2
AB-VE	66	0.9	97	2.3	86	0.5
O+VE	3268	29.0	4110	0.7	4533	29.1
O-VE	207	1.8	282	2.1	326	2.1
Total	**11288**	**100**	**13678**	**100**	**15505**	**100**

Table- 2: Rh blood group frequency in present study

	Rh +ve	**Rh –ve**	
2012	10574	714	
2013	12788	890	
2014	14507	998 = 40471	
Total	37869 (93.57%)	2602 (6.42%)	

Figure 1: Bar diagram showing number of blood donors in group wise distribution in Bhavnagar population in 3 years 2012, 2013 and 2014

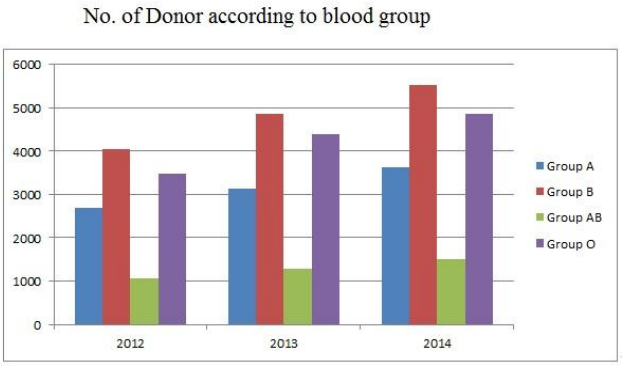
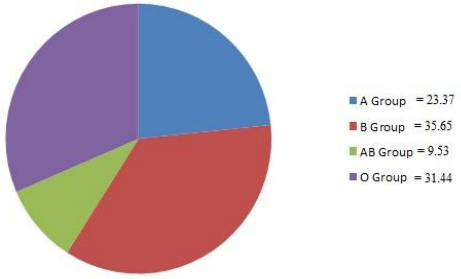


Figure 2: Pie Diagram showing frequency of different blood groups



From above result our study has determind the distribution & frequency of ABO & Rh Blood group among the – 40471 Donor, coming to Sir T. Hospital, Blood bank, Bhavnagar in Year Jan -2012 To Dec -2014. Our Result state that blood group "B" is most common followed by "O" Blood Group.

The frequency of various blood group according to present study, from table -1: B+ve 33.4%, O+ve 29.4%, A+ve 21.86%, AB+VE 8.9%, B-ve 2.3%, O-ve 2%, A-ve 1.46% & rarest being AB-ve 0.7% Also from Table 2: Rh-Group positive are 93.57% and Rh –Neg less common with 6.42% prevalence.

Table -3: Comparison study on frequency of ABO & Rhesus Blood Group in it deferent geographical areas

Region	A (%)	B (%)	O (%)	AB (%)	Rh +ve	Rh -ve
Present study	23.33	35.7	31.4	9.6	93.5	6.4
Eastern-ahemdabad[9]	23.3	35.5	32.5	8.8	94.2	5.8
Vellore [11]	18.85	32.69	38.75	5.27	94.5	5.47
Bangalore [12]	23.85	29.95	39.82	6.37	94.3	6.7
Chittor [13]	18.95	25.79	47.37	7.89	90.6	8.4
Pakistan [10]	23.8	38	10	10	89.1	10.9
Nepal [14]	34	29	33	4	96.7	3.3
Niger delta[15]	23.8	20.7	52.7	2.8	93.9	6.12
USA [16]	41	9	46	4	85	15
Britain [17]	41.7	8.6	46.7	3	83	17

The comparison of frequency and distribution of ABO and Rh Group in blood donor at present study with the similar studies carried out within and outside India is describe in table-3, Distribution of ABO and Rh grouping was amparable to the studies done at Eastern Ahmedabad 9, and Pakistan 10, All these studies have described "B" as most frequent and "AB" as least common Blood Group.

The second most common is "O" in resent study as well as in Eastern Ahmedabad [9],

Whereas in southern India [(11,12,13)] have described contras finding with "O" being the most common followed by "B", "A" and "AB"

In Nepal [14], Britain [17], USA [16], "O" and "A" are the common blood group that are followed by "B" and "AB".

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In Nepal [14], Britain [17], USA [16], "O" and "A" are the common blood group that are followed by "B" and "AB".

In Nigeria [15] "O" is the predominantly encountered blood group for more than 50% of Donors and AB has least common accordance.

This difference in Phenotype of Blood group is due to the prevalence of autosomal genes at various geographer regions. Due to autosomal inheritance male and female data does not affect the frequency of blood group phenotypes 18.From our observation we can state that blood group system not only help in transfusion service, but also help to take preventative measure against disease which are associated with different blood group. There is known genetic association of specific blood group to certain disease in certain population. Group "A" more frequency with coronary heart disease, IHD, Venous Thrombus, and atherosclerosis with its low in people with blood group "O" which state to have protective

effect against this disease (19,20,21)The "B" antigen increase risk of ovarian ca.22, Gastric carcinoma. More common in blood group "A" and least in group "O" 23,

In short, generation of simple database of blood group not only provide date about the availability of human blood in case of emergency regional natural comities but also serve to enable insight into possibilities to future burden of disease, it useful to health planner while making efforts to face the future health challengers' in the region

References

1. Lo YM, Hjelm NM, Fidler C, Sargent IL, Murphy MF, et al. (1998) Prenatal diagnosis of fetal RhD status by molecular analysis of maternal plasma. N Engl J Med 339: 1734-1738. Lasky LC, Lane TA, Miller JP, Lindgren B, Patterson HA, et al. (2002) In utero or ex utero cord blood collection: which is better? Transfusion 42: 1261-1267.
2. Lasky LC, Lane TA, Miller JP, Lindgren B, Patterson HA, et al. (2002) In utero or ex utero cord blood collection: which is better? Transfusion 42: 1261-1267
3. Wall DA, Noffsinger JM, Mueckl KA, Alonso JM 3rd, Regan DM, et al. (1997) Feasibility of an obstetrician-based cord blood collection network for unrelated donor umbilical cord blood banking. J Matern Fetal Med 6: 320-323.
4. Dhot PS, Nair V, Swarup D, Sirohi D, Ganguli P (2003) Cord blood stem cell banking and transplantation. Indian J Pediatr 70: 989-992.
5. Khan MS, Subhan F, Tahir F, Kazi BM, Dil AS, Sultan S. Prevalence of blood groups and Rh factor in Bannu region NWFP (Pakistan). Pak J Med Res 2004; 43(1):8–10.
6. Khaliq MA, Khan JA, Shah H, Khan SP. Frequency of ABO and Rh (D) blood group in Hazara division (Abbottabad). Pak J Med Res 1984; 23:102–3.
7. Hoffbrand A V and Pettit J E. Blood Transfusion in "Essential Haematology", Oxford UK, Black well scientific Publication. 2006, 5th Edition: 307–9.
8. Enosolease M E, Bazuaye G N. Distribution of ABO and Rh- D blood groups in the Benin area of Niger-Delta: Implication for regional blood transfusion. Asian J Transf Sci. 2008; 2 (1): 3–5
9. Wadhwa M K, Patel S M, Kothari D C, Pandey M, Patel D D. Distribution of ABO and Rhesus-D groups in Gujarat, India: a hospital based study. Indan J Ped Oncol.1998; 19 (4): 137–141.
10. Hammed A, Hussain W, Ahmed J, Rabbi F, Qureshi J A. Prevalence of Phenotypes and Genes of ABO and Rhesus (Rh) Blood Groups in Faisalabad, Pakistan. Pak J Biol Sci. 2002; 5: 722–724.
11. Das P K, Nair S C, Harris V K, Rose D, Mammen J J, Bose Y N, Sudarsanam A. Distribution of ABO and Rh-D blood groups among blood donors in a tertiary care centre in South India. Trop Doct. 2001; 31 (1): 47–8.
12. Periyavan A, Sangeetha S K, Marimuthu P, Manjunath B K, Seema. Distribution of ABO and Rhesus-D groups in and around Bangalore. Asian J Transfus Sci. 2010; 4 (1): 41.
13. Reddy K S N, Sudha G. and Rh (D) blood groups among the desuri Reddis of Chittoor District, Andhra Pradesh. Anthropologist. 2009; 11 (3): 237-238.
14. Pramanik T, Pramanik S. Distribution of ABO and Rh blood groups in Nepalese medical students: a report. East Mediterr Health J. 2000 Jan; 6 (1): 156-8
15. Enosolease M E, Bazuaye G N. Distribution of ABO and Rh- D blood groups in the Benin area of Niger-Delta: Implication for regional blood transfusion. Asian J Transf Sci. 2008; 2 (1) 3-5
16. Mollison P L, Engelfriet C P, Conteras M. The Rh blood group system. In Blood Transfusion in Clinical Medicine, 9th

Edition. Oxford: Black well Scientific Publication.1993; 2008–9.
17. Frances TF: Blood groups (ABO groups). In: Common Laboratory and Diagnostic Tests. Philadelphia: Lippincott. 2002, 3rd Edition: 19–5.
18. saran R.k..transfusion medicine ,technical manual ,Second edition- new delhi India:2003 Page 56.
19. Khan M I, Micheal S, Akhtar F, Naveed A, Ahmed A & Qamar R. Association of ABO blood groups with glaucoma in the Pakistani population. Canadian Journal of Ophthalmology 2009; 44: 582–586.
20. Alam M. ABO and Rhesus blood groups in potential blood donors at Skardu (Northern Areas). Pakistan Journal of Pathology. 2005; 16: 94–97.
21. Khan M S, Subhan F, Tahir F, Kazi B M, Dil A S, Sultan S, Deepa F, Khan F & Sheikh M A. Prevalence of blood groups and Rh factor in Bannu District (NWFP) Pakistan.
22. Gates M A, Wolpin B M, Cramer D W, Hankinson S E, Tworoger S S. ABO blood group and incidence of epithelial ovarian cancer. Int J Cancer. 2010; 128 (2): 482–6.
23. Aird I, Bentall H H, Roberts J A. (1953). A relationship between cancer of stomach and the ABO blood groups. Br Med J. 2011 Apr; 1 (4814): 799–801.

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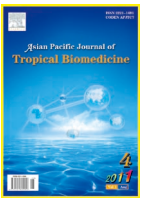

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Association of ABO blood group and *Plasmodium falciparum* malaria in Dore Bafeno Area, Southern Ethiopia

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ABSTRACT

Objective: To assess the distribution of ABO blood group and their relationship with *Plasmodium falciparum (P. falciparum)* malaria among febrile outpatients who sought medical attention at Dore Bafeno Health Center, Southern Ethiopia. **Methods:** A total of 269 febrile outpatients who visited Dore Bafeno Health Center, Southern Ethiopia, were examined for malaria and also tested for ABO blood groups in January 2010. The blood specimens were collected by finger pricking, stained with Geimsa, and examined microscopically. Positive cases of the parasitemia were counted. CareStart™ Malaria *Pf/Pv* Combo was also used to test the blood specimens for malaria. ABO blood groups were determined by agglutination test using ERYCLONE® antisera. Data on socio-demographic characteristics and treatment status of the participants were also collected. *Chi*-square and ANOVA tests were used to assess the difference between frequencies and means, respectively. **Results:** Out of a total of 269 participants, 178 (66.2%) febrile patients were found to be infected with *Plasmodium* parasites, among which 146 (54.3%), 28 (10.4%), and 4 (1.5%) belonged to *P. falciparum*, *P. vivax*, and mixed infections, respectively. All febrile patients were also tested for ABO blood groups and 51.3%, 23.5%, 21.9% and 3.3% were found to be blood types of O, A, B and AB, respectively. Both total malaria infection and *P. falciparum* infection showed significant association with blood types ($P<0.05$). The proportion of A or B but not O phenotypes was higher ($P<0.05$) in individuals with *P. falciparum* as compared with non-infected individuals. The chance of having *P. falciparum* infection in patients with blood groups A, B and AB was 2.5, 2.5 and 3.3 times more than individuals showing blood O phenotypes, respectively. The mean *P. falciparum* malaria parasitaemia for blood groups A, B, AB, and O were 3 744/ μ L, 1 805/ μ L, 5 331/ μ L, and 1 515/ μ L, respectively ($P<0.01$). **Conclusions:** The present findings indicate that individuals of blood groups A, B and AB are more susceptible to *P. falciparum* infection as compared with individuals of blood group O. Nevertheless, further in depth studies are required to clearly establish the role that ABO blood group plays in *P. falciparum* malaria.

1. Introduction

The ABO blood groups consist of A, B and H carbohydrate antigens which can regulate protein activities during infection and antibodies against these antigens[1,2]. A number of studies were conducted to investigate the association between ABO blood group system and some disease conditions[3-8]. Some of these studies reported significant associations, suggesting that ABO blood groups have an impact on infection status of the individuals possessing a particular ABO blood group[5-8].

In view of a heavy burden placed on human health due to malaria, a good many investigations have been conducted to find out whether or not ABO blood groups antigens are associated with susceptibility, resistance, or severity of *P. falciparum* malaria. Nonetheless, these studies have reported contradictory results. Some studies reported the absence of significant association between *P. falciparum* (prevalence, parasitaemia or antibody titer) and ABO antigens[4,9-14]. On the other hand, other studies have shown that high frequency of malaria episodes has been observed among blood group 'A' individuals as compared with other blood groups individuals[15]. Large numbers of severe malaria cases were also reported among blood group 'A'

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individuals[16,17]. Furthermore, Migot-Nabias and Pathirana *et al*[18,19] observed low parasitaemia and uncomplicated malaria cases among blood group 'O' individuals, respectively.

Variations in reports on the association of ABO blood groups and disease progression of *P. falciparum* malaria show the complexity of the interaction between the parasite and host immune responses. In addition, studies have shown the impact of other red blood cells (RBC) polymorphisms including haemoglobin (Hb) abnormalities such as HbS, HbC, thalassemia and deficiency in erythrocyte complement receptor (CR) or glucose-6-phosphate dehydrogenas on *P. falciparum* malaria susceptibility and severity[18,20-23]. This makes it difficult to make a clear analysis on the association of ABO blood groups and *P. falciparum* because so far most of the study designs have been conducted *in vivo*. Therefore, this study aims to assess the distribution of ABO blood groups and their relationship with *P. falciparum* malaria among febrile outpatients who sought medical attention at Dore Bafeno Health Center, Southern Ethiopia.

2. Materials and methods

2.1. Study area and population

A cross-sectional study was conducted at Dore Bafeno Health Center, Southern Ethiopia, to assess the association of ABO blood groups antigens with malaria in January 2010. Dore Bafeno is located at about 23 km to the southwest of Hawassa town. The area has an elevation of about 1 708 m above sea level. It has an estimated total population of 139891, consisting of 70 503 males and 69 388 females.

The study population was composed of febrile outpatients who sought medical attention at Dore Bafeno Health Center in January 2010. A total of 269 febrile outpatients were selected as study participants, excluding individuals who took antimalarial drugs within two weeks before blood test and who refused to participate in the study. The target populations were almost from the same ethnic (Sidama) group.

2.2. Ethical approval

The study obtained ethical clearance from the Institutional Research Board (IRB) of the Aklilu Lemma Institute of Pathobiology, Addis Ababa University, and from the Health Bureau of South Nations Nationalities and Peoples Region (SNNPR). Malaria positive cases were treated with antimalarial drugs based on the current national treatment guideline of Ethiopia.

2.3. Clinical and laboratory diagnosis

Before collecting blood sample, explanation about the study was given and a written informed consent was obtained from every study participant including the guardians of children. Capillary blood was collected by finger pricking using 70% isopropanol and sterile disposable lancet. Heel puncture was used for infants. Immediately, thin film was spread on grease free, frosted end, labeled slide using a smooth edged slide spreader. Thick film was also prepared on the same slide. Thin film was then fixed with methanol. The blood film was stained with 10% Giemsa for 10 minutes. Finally, the films were examined under an oil immersion microscope objective (100×). Parasitemia was determined for febrile patients who tested positive for *P. falciparum* by counting the number of parasites (asexual forms only) against 200 white blood cells (WBC). This counting was done by using hand tally counters. Then, the number of parasites per microliter of blood was calculated[24]. CareStart™ Malaria *Pf/Pv* Combo test was also performed parallel with blood film examination using the same blood sample following manufacturer's instruction (Access Bio, Inc. NJ USA). Similarly, the blood group of the study participants was determined by direct slide method, using agglutinating A and B Monoclonal ERYCLONE® anti-sera alongside with the former procedures.

2.4. Data analysis

Data were entered in Microsoft Excel, checked for its correctness, and exported to and analyzed using SPSS version 13 (SPSS Inc, Chicago, IL). *Chi*-square test was used to assess the difference between frequencies (the associations between blood groups and *P. falciparum* malaria cases). ANOVA was used to test the difference between parasitemia means. Observed difference was considered to be significant for $P<0.05$.

3. Result

3.1. Malaria infection

Out of a total of 269 febrile patients who visited Dore Bafeno Health Center for medical attention, 178 (66.2%) were found to be infected with *Plasmodium* parasites as determined by microscopy. The prevalence of malaria was found to be the highest among under-five children as compared with older age groups but the difference was not significant ($P>0.05$). Similarly, the prevalence was higher among females (67.2%) than males (65.3%) and this difference was not statistically significant (Table 1).

3.2. ABO blood groups and malaria infection

All febrile patients examined for malaria were also tested for ABO blood groups. Accordingly, 51.3%, 23.5%, 21.9% and 3.3% were found to be blood types of O, A, B and AB,

Table 1
Prevalence of malaria by age and sex among the study participants at Dore Bafeno Health Center, Southern Ethiopia, January 2010 [*n* (%)].

Age	Number examined	*P. falciparum*	*P. vivax*	Mixed*	Total
≤5	67	43 (64.2)	7 (10.4)	0 (0.0)	50 (74.6)
6-15	112	61 (54.5)	11 (9.8)	4 (3.6)	76 (67.8)
>15	90	42 (46.7)	10 (11.1)	0 (0.0)	52 (57.8)
Total	269	146 (54.3)	28 (10.4)	4 (1.5)	178 (66.2)
χ^2, *P*		4.75, 0.09	0.89, 0.96	5.69, 0.06	5.11, 0.08
Sex					
Male	144	80 (55.5)	11 (7.6)	3 (2.0)	94 (65.3)
Female	125	66 (52.8)	17 (13.6)	1 (0.8)	84 (67.2)
χ^2, *P*		0.21, 0.65	2.55, 0.11	0.75, 0.39	0.11, 0.74

*Mixed infection of *P. falciparum* and *P. vivax*.

Table 2
Frequency of ABO blood groups among *Plasmodium*-infected cases at Dore Bafeno Health Center, Southern Ethiopia, January 2010 [*n* (%)].

Blood group	Number examined	Infection type				Total	χ^2, *P*
		P. falciparum	*P. vivax*	Mixed*	Non-infected		
A	63	41 (65.1)	5 (7.9)	1 (1.6)	16 (25.4)	63 (23.5)	61.6, <0.0100
B	59	38 (64.4)	7 (11.9)	0 (0.0)	14 (23.7)	59 (21.9)	55.5, <0.0100
AB	9	7 (77.8)	0 (0.0)	0 (0.0)	2 (22.2)	9 (3.3)	14.6, <0.0100
O	138	60 (43.5)	16 (11.6)	3 (2.2)	59 (42.8)	138 (51.3)	74.9, <0.0100
Total	269	146 (54.3)	28 (10.4)	4 (1.5)	91 (33.8)	269 (100)	183.0, <0.0001
χ^2, *P*		13.9, 0.0030	1.8, 0.6150	1.5, 0.6880	10.4, 0.0150	126.2, 0.0001	

Table 3
Comparison of frequency of ABO blood groups among *P. falciparum*-infected individuals with that of non-*Plasmodium*-infected individuals at Dore Bafeno Health Center, Southern Ethiopia, January 2010 [*n* (%)].

Blood group	Age group	Numbers with blood type	*P. falciparum* infected	Non-*Plasmodium*-infected	χ^2, *P*	χ^2, *P*
A						
	≤5	16	13 (81.2)	1 (6.3)	8.64, 0.003	
	6-15	21	16 (76.2)	5 (23.8)	4.76, 0.029	
	>15	26	13 (50.0)	10 (38.5)	0.18, 0.670	
	Total	63	42 (66.7)	16 (25.4)	10.79, 0.001	5.99, 0.050
B						
	≤5	14	10 (71.4)	3 (21.4)	2.76, 0.090	
	6-15	28	20 (71.4)	6 (21.4)	6.50, 0.010	
	>15	17	8 (47.1)	5 (29.4)	0.30, 0.580	
	Total	59	38 (64.4)	14 (23.7)	10.18, 0.001	1.17, 0.556
AB						
	≤5	1	1 (100.0)	0 (0.0)	0.00, 1.000	
	6-15	4	3 (75.0)	1 (25.0)	0.26, 0.610	
	>15	4	3 (75.0)	1 (25.0)	0.26, 0.610	
	Total	9	7 (77.8)	2 (22.2)	1.78, 0.182	0.32, 0.852
O						
	≤5	36	19 (52.8)	13 (36.1)	0.78, 0.370	
	6-15	59	26 (44.1)	14 (23.7)	0.02, 0.887	
	>15	43	18 (41.9)	22 (51.2)	0.22, 0.639	
	Total	138	63 (45.7)	59 (42.8)	0.08, 0.777	1.48, 0.478

respectively(χ^2=126.2, *P*<0.01). The highest proportion of individuals in all blood groups were infected with *P. falciparum* as compared with other groups (*P. vivax*, mixed and non-infected), and this difference was statistically significant (*P*<0.01). In general, malaria infection showed significant association with blood group (χ^2=10.4, *P*=0.015) with the highest proportion (77.8%) observed among individuals with AB blood group, followed by those with blood group B (64.4%). Prevalence of *P. falciparum* infection also showed similar pattern (χ^2=13.9, *P*<0.01) with the highest proportion (77.8%) observed among individuals with AB blood group, followed by those with blood group A (65.1%) (Table2).

65.1%, 64.4%, 77.8% and 43.5% of individuals with A, B, AB and O blood types, respectively had *P. falciparum* infections. The proportion of A or B but not O phenotypes was higher

(P<0.05) in individuals with *P. falciparum* compared with non-infected individuals. *P. falciparum* infection do not show association (P>0.05) with age in all the 4 blood groups (Table 3).

The chance of having *P. falciparum* infection in patients with blood groups A, B and AB was 2.5 (χ^2=6.941, P=0.008, OR=2.46, 95% confidence interval (CI)=1.256-4.804), 2.5 (χ^2=6.841, P=0.009, OR=2.54, 95% CI=1.260-5.119) and 3.3 (χ^2=2.284, P=0.131, OR=3.28, 95% CI=0.737-14.380) times higher than individuals showing blood O phenotypes, respectively. Also, this probability of *P. falciparum* infection for patients with blood group A, B and AB was found higher (OR>1.5) when compared with blood group O individuals for each age groups.

3.3. ABO blood type and P. falciparum parasitaemia

About 38.1% (16/42), 47.4% (18/38), 22.2% (2/9) and 66.7% (42/63) *P. falciparum* infected individuals of blood group A, B, AB and O, respectively had parasite density of less than 1000 parasites/μL of blood. In contrast, 59.5% (25/42), 52.6% (20/38), 77.8% (7/9) and 33.3% (21/63) *P. falciparum* infected individuals of blood group A, B, AB and O, respectively had parasite density of greater than 1000 parasites/μL of blood. Only 2.4% (1/42) individuals of *P. falciparum* infected patients with blood group A had a parasite density of greater than 100 000 parasites/μL of blood. Mean *P. falciparum* malaria parasitaemia for blood groups A, B, AB and O were 3 744/μL, 1 805/μL, 5 331/μL, and 1 515/μL respectively. This difference was statistically significant (P<0.01, F= 11.510).

4. Discussion

In this study, high percentage of O blood group (51.3%) phenotype was observed among the study participants followed by A (23.5%), B (21.6%), and AB (3.3%). This agrees with some previous studies that also reported high frequency of group 'O' and low frequency of group 'A' phenotypes in tropical regions where malaria is rampant[14,25,26]. On the other hand, other studies reported high prevalence of blood group 'A' and low prevalence of blood group 'O' phenotypes in colder regions where malaria has not been endemic [14,27]. Hence, the present finding seems to substantiate the hypothesis about a selective (survival) evolutionary advantage of *P. falciparum* infection on blood group O cells compared with other blood group types (A, B or AB) in areas where malaria is endemic[28].

In this study, significantly higher proportions of individuals with blood group A, B and AB but not O were found to be infected with *P. falciparum* as compared with non-*P. falciparum* infected individuals. This is also consistent with previous reports[29,30], suggesting that individuals with blood groups A, B and AB are more susceptible to *P. falciparum* infection than those with O group. Beiguelman and Santos *et al*[15,31] similarly observed significant association between the presence of A or B antigen and the number of malarial episodes in Brazil. Several mechanisms relate to these associations, including affinity for *Anopheles* species, shared ABO antigens with *P. falciparum*, impairment of merozoite penetration of RBCs, as well as cytoadherence, endothelial activation and rosetting[32]. On the other hand, absence of association between ABO system and malaria infection was also observed in other populations[10,13,29,33,34]. Also, in contrast to our observation, Rowe and Tekeste *et al*[35,36] documented absence of difference in the frequency of ABO blood groups between healthy controls and those with uncomplicated malaria, suggesting insignificant effect of the ABO blood groups on uncomplicated clinical malaria disease. However, in accordance with our findings, Rowe *et al*[35] observed the absence of significance difference in the frequency of group O between uncomplicated malaria cases and the healthy controls.

The lowest mean parasitaemia was also observed among individuals with blood group O as compared to blood groups A, B and AB. Similarly, Migot-Nabias *et al*[18] observed lower *P. falciparum* parasitaemia in those with blood group O as compared to non-O subjects. In addition, other studies also reported a high chance of severe malaria or high parasitaemia cases in individuals possessing blood group A or AB cells than among individuals with blood group 'O'[16, 17,19,29].

The mechanism by which 'A' promotes susceptibility and 'O' confers a relative protective effect against high *P. falciparum* parasitaemia is not well understood. Nevertheless, different studies have come up with their reasonable explanations on the basis of rosette formation. Several reports support the hypothesis that blood group A represents a risk factor for high chance of rosette, which is usually characterized by high *P. falciparum* parasitaemia during malaria infection and a reducing effect of blood group 'O' on rosette[37-45]. The presence of several glycosylated adhesion molecules such as intracellular adhesion molecule 1[38], complement receptor 1[37], heparin sulfate-like glycosaminoglycan[40,41], platelet glycoprotein CD36[46-54], high level von Willebrand factor[43], low arginine and nitrate levels[42], presence of cellular micro-particles [44] and the nature of sugar molecules (trisacchrides)[55] in blood group 'A' cells promote a high chance of binding with the rosette-forming surface molecules of the *P. falciparum* such as Duffy binding-like domain 1 alpha of *P. falciparum* erythrocyte membrane protein-1[21,56], rifs[57,58]. On the other hand, blood group 'O' cells show deficiency of most of the above adhesive molecules and contain disaccharides sugar molecules which reduce the rate, size and stability of rosette formed during *P. falciparum* infection[44,55].

The present study only employed parasitaemia as a laboratory marker to determine the association of ABO blood groups and *P. falciparum* malaria. The study also did not consider factors like HbS, HbC, CR, iron status of the host,

place of residence of the study population which could affect the nature of *P. falciparum* infection among the study population. Had more laboratory markers or clinical features (*e.g.* cerebral malaria) been used, more information would have been generated on the associations. Nevertheless, the findings indicate that individuals of blood group A, B and AB are more susceptible to *P. falciparum* infection as compared with individuals with the blood type O. Further in-depth studies are required to clearly establish the role of ABO blood groups in the *P. falciparum* malaria.

Conflict of interest statement

We declare that we have no conflict of interest.

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References

[1] Chung WY, Gardiner DL, Hyland C, Gatton M, Kemp DJ, Trenholme KR. Enhanced invasion of blood group A1 erythrocytes by *Plasmodium falciparum*. *Mol Biochem Parasitol* 2005; **144**: 128-130.

[2] Greenwell P. Blood group antigens: molecules seeking a function? *Glycoconj J* 1997; **14**(2): 159-173.

[3] Tursen U, Tiftik EN, Unal S, Gunduz O, Kaya TI, Camdeviren H, et al. Relationship between ABO blood groups and skin cancers. *Dermatol Online J* 2005; **11**: 44.

[4] Kassim OO, Ejezie GC. ABO blood groups in malaria and schistosomiasis haematobium. *Acta Trop* 1982; **39**: 179-184.

[5] Opera KN. Onchocerciasis and ABO blood group status: a field based study. *Int J Trop Med* 2007; **2**(4): 123-125.

[6] Abdulazeez AA, Alo EB, Rebecca SN. Carriage rate of Human Immunodeficiency Virus (HIV) infection among different ABO and Rhesus blood groups in Adamawa state, Nigeria. *Biomed Res* 2008; **19**: 41-44.

[7] Ndambaa J, Gomoa E, Nyazemab N, Makazaa N, Kaondera KC. Schistosomiasis infection in relation to the ABO blood groups among school children in Zimbabwe. *Acta Trop* 1997; **65**: 181-190.

[8] Blackwell CC, Dundas S, James VS, Mackenzie AC, Braun JM, Alkout AM, et al. Blood group and susceptibility to disease caused by *Escherichia coli* O157. *J Infect Dis* 2002; **185**(3): 393-396.

[9] Martin SK, Miller LH, Hicks CU, David-West A, Ugbode C, Deane M. Frequency of blood group antigens in Nigerian children with falciparum malaria. *Trans R Soc Trop Med Hyg* 1979; **73**: 216-218.

[10] Bayoumi RA, Bashir AH, Abdulhadi NH. Resistance to falciparum malaria among adults in central Sudan. *Am J Trop Med Hyg* 1986; **35**: 45-55.

[11] Akinboye DO, Ogunrinade AF. Malaria and loaisis among blood donors at Ibadan, Nigeria. *Trans R Soc Trop Med Hyg* 1987; **81**: 398-399.

[12] Thakur A, Verma IC. Malaria and ABO blood groups. *Indian J Malariol* 1992; **29**: 241-244.

[13] Montoya F, Restrepo M, Montoya AE, Rojas W. Blood groups and malaria. *Rev Inst Med Trop São Paulo* 1994; **36**: 33-38.

[14] Uneke CJ, Ogbu O, Nwojiji V. Potential risk of induced malaria by blood transfusion in South-eastern Nigeria. *Mcgill J Med* 2006; **9**: 8-13.

[15] Beiguelman B, Alves FP, Moura MM, Engracia V, Nunes AC, Heckmann MI, et al. The association of genetic markers and malaria infection in the Brazilian Western Amazonian Region. *Mem Inst Oswaldo Cruz* 2003; **98**: 455-460.

[16] Fischer PR, Boone P. Short report: severe malaria associated with blood group. *Am J Trop Med Hyg* 1998; **58**: 122-123.

[17] Lell B, May J, Schmidt-Ott RJ, Lehman LG, Luckner D, Greve B, et al. The role of red blood cell polymorphisms in resistance and susceptibility to malaria. *Clin Infect Dis* 1999; **28**: 794-799.

[18] Migot-Nabias F, Mombo LE, Luty AJ, Dubois B, Nabias R, Bisseye C, et al. Human genetic factors related to susceptibility to mild malaria in Gabon. *Genes Immun* 2000; **1**: 435-441.

[19] Pathirana SL, Alles HK, Bandara S, Phone-Kyaw M, Perera MK, Wickremasinghe AR, et al. ABO-blood-group types and protection against severe, *Plasmodium falciparum* malaria. *Ann Trop Med Parasitol* 2005; **99**: 119-124.

[20] Carlson J, Nash GB, Gabutti V, Al-Yaman F, Wahlgren M. Natural protection against severe *Plasmodium falciparum* malaria due to impaired rosette formation. *Blood* 1994; **84**: 3909-3914.

[21] Rowe JA, Moulds JM, Newbold CI, Miller LH. *P. falciparum* rosetting mediated by a parasite-variant erythrocyte membrane protein and complement-receptor 1. *Nature* 1997; **388**: 292-295.

[22] Weatherall DJ, Miller LH, Baruch DI, Marsh K, Doumbo OK, Casals-Pascual C, et al. Malaria and the red cell. *Hematology Am Soc Hematol Educ Program* 2002; 35-57.

[23] Cockburn IA, Mackinnon MJ, O'Donnell A, Allen SJ, Moulds JM, Baisor M, et al. A human complement receptor 1 polymorphism that reduces *Plasmodium falciparum* rosetting confers protection against severe malaria. *Proc Natl Acad Sci USA* 2004; **101**: 272-277.

[24] Cheesbrough M. Parasitological tests. *District laboratory practices in tropical countries, part 1*. 2nd ed. England: Cambridge University Press; 1998, p. 220-221.

[25] Seyoum S, Dagne K. ABO and rhesus blood type frequencies in data from hospitals and the Red Cross in Ethiopia. *Ethiop Med J* 1985; **23**: 1-6.

[26] Tadege T, Mengistu Y, Desta K, Asrat D. Seroprevalence of *Helicobacter pylori* infection in and its relationship with ABO blood groups. *Ethiop J Health Dev* 2005; **19**(1): 55-59.

[27] Mourant AE, Kopec AC, Domaniewska-Sobczak K. *The distribution of the human blood groups and other polymorphisms*.

London: Oxford University Press; 1976.

[28] Cserti CM, Dzik WH. The ABO blood group system and *Plasmodium falciparum* malaria. *Blood* 2007; **110**: 2250-2258.

[29] Singh N, Shukla MM, Uniyal VP, Sharma VP. ABO blood groups among malaria cases from district Mandla, Madhya Pradesh. *Indian J Malariol* 1995; **32**: 59-63.

[30] Fry AE, Griffiths MJ, Auburn S, Diakite M, Forton JT, Green A, et al. Common variation in the ABO glycosyltransferase is associated with susceptibility to severe *Plasmodium falciparum* malaria. *Hum Mol Genet* 2008; **17**: 567-576.

[31] Santos SEB, Salzano FM, Franco MHLP, Freitas MJM. Mobility, genetic markers, susceptibility to malaria and race mixture in Manaus, Brazil. *J Hum Evol* 1983; **12**: 373-381.

[32] Loscertales MP, Owens S, O'Donnell J, Bunn J, Bosch-Capblanch X, Brabin BJ. ABO blood group phenotypes and *Plasmodium falciparum* malaria: unlocking a pivotal mechanism. *Adv Parasitol* 2007; **65**: 1-50.

[33] Osisanyia JO. ABO blood groups and infections with human malarial parasites *in-vivo* and *in-vitro*. *East Afr Med J* 1983; **60**: 616-621.

[34] Singh IP, Walter H, Bhasin MK, Veena B, Sudhakar K. Genetic markers and malaria observations in Gujarat, India. *Hum Hered* 1986; **36**: 31-36.

[35] Rowe JA, Handel IG, Thera MA, Deans AM, Lyke KE, Kone A, et al. Blood group O protects against severe *Plasmodium falciparum* malaria through the mechanism of reduced rosetting. *Proc Natl Acad Sci USA* 2007; **104**: 17471-17476.

[36] Tekeste Z, Petros B. The ABO blood group and *Plasmodium falciparum* malaria in Awash, Metehara and Ziway areas, Ethiopia. *Malar J* 2010; **9**: 280.

[37] Lublin DM, Griffith RC, Atkinson JP. Influence of glycosylation on allelic and cell-specific Mr variation, receptor processing, and ligand binding of the human complement C3b/C4b receptor. *J Biol Chem* 1986; **261**: 5736-5744.

[38] Carlson J, Holmquist G, Taylor DW, Perlmann P, Wahlgren M. Antibodies to a histidine-rich protein (PfHRP1) disrupt spontaneously formed *Plasmodium falciparum* erythrocyte rosettes. *Proc Natl Acad Sci USA* 1990; **87**: 2511-2515.

[39] Carlson J, Wahlgren M. *Plasmodium falciparum* erythrocyte rosetting is mediated by promiscuous lectin-like interactions. *J Exp Med* 1992; **176**: 1311-1317.

[40] Chen Q, Heddini A, Barragan A, Fernandex V, Pearce SF, Wahlgren M. The semi-conserved head structure of *Plasmodium falciparum* erythrocyte membrane protein 1 mediates binding to multiple independent host receptors. *J Exp Med* 2000a; **192**: 1-9.

[41] Chen Q, Schlichtherle M, Wahlgren M. Molecular aspects of severe malaria. *Clin Micro Rev* 2000b; **13**: 439-450.

[42] Lopansri BK, Anstey NM, Weinberg JB, Stoddard GJ, Hobbs MR, Levesque MC, et al. Low plasma arginine concentrations in children with cerebral malaria and decreased nitric oxide production. *Lancet* 2003; **361**: 676-678.

[43] Jenkins PV, O'Donnell JS. ABO blood group determines plasma von Willebrand factor levels: a biologic function after all? *Transfusion* 2006; **46**: 1836-1844.

[44] Van der Heyde HC, Nolan J, Combes V, Gramaglia I, Grau GE. A unified hypothesis for the genesis of cerebral malaria: sequestration, inflammation and hemostasis leading to microcirculatory dysfunction. *Trends Parasitol* 2006; **22**: 503-508.

[45] Rowe JA, Handel IG, Thera MA, Deans AM, Lyke KE, Kone A, et al. Blood group O protects against severe *Plasmodium falciparum* malaria through the mechanism of reduced rosetting. *Proc Natl Acad Sci USA* 2007; **104**: 17471-17476.

[46] Stockelberg D, Hou M, Rydberg L, Kutti J, Wadenvik H. Evidence for an expression of blood group A antigen on platelet glycoproteins IV and V. *Transfus Med* 1996; **6**: 243-248.

[47] Fan ZG, Zhang LM, Yan GG, Wu Q, Gan XF, Zhong SF, et al. Bioinformatics analysis for structure and function of CPR of *Plasmodium falciparum*. *Asian Pac J Trop Med* 2011; **4**(2): 85-87.

[48] Tangpukdee N, Wai KM, Muangnoicharoen S, Kano S, Phophak N, Tiemprasert J, et al. Indicators of fatal outcome in severe *Plasmodium falciparum* malaria: a study in a tertiary-care hospital in Thailand. *Asian Pac J Trop Med* 2010; **3**(11): 855-859.

[49]Wisedpanichkij R, Chaicharoenkul W, Mahamad P, Prompradit P, Na-Bangchang K. Polymorphisms of the oxidant enzymes glutathione S-transferase and glutathione reductase and their association with resistance of *Plasmodium falciparum* isolates to antimalarial drugs. *Asian Pac J Trop Med* 2010; **3**(9): 673-677.

[50] Nmorsi OPG, Isaac C, Ukwandu NCD, Ohaneme BA. Pro-and anti-inflammatory cytokines profiles among Nigerian children infected with *Plasmodium falciparum* malaria. *Asian Pac J Trop Med* 2010; **3**(1): 41-44.

[51] Nmorsi OPG, Isaac C, Ukwandu NCD, Ekundayo AO, Ekozien MI. Schistoma haematobium and *Plasmodium falciparum* coinfection with protection against *Plasmodium falciparum* malaria in Nigerian children. *Asian Pac J Trop Med* 2009; **2**(2): 16-20.

[52] Peter G, Manuel AL, Anil S. Study comparing the clinical profile of complicated cases of *Plasmodium falciparum* malaria among adults and children. *Asian Pac J Trop Dis* 2011; **1**(1): 35-37.

[53] Krungkrai SR, Krungkrai J. Malaria parasite carbonic anhydrase: inhibition of aromatic/heterocyclic sulfonamides and its therapeutic potential. *Asian Pac J Trop Biomed* 2011; **1**(3): 233-242.

[54] Rout R, Dhangadamajhi G, Mohapatra BN, Kar SK, Ranji M. Genetic diversity of PfEMP1-DBL 1-α and its association with severe malaria in a hyperendemic state of India. *Asian Pac J Trop Med* 2010; **3**(7): 505-509.

[55] Daniels G. The molecular genetics of blood group polymorphism. *Transpl Immunol* 2005; **14**: 143-153.

[56] Chen Q, Barragan A, Fernandez V, Sundstrom A, Schlichtherle M, Sahlen A, et al. Identification of *Plasmodium falciparum* erythrocyte membrane protein1(PfEMP1) as the rosetting ligand of the malaria parasite *P. falciparum*. *J Exp Med* 1998; **187**: 15-23.

[57] Helmby H, Cavelier L, Pettersson U, Wahlgren M. Rosetting *Plasmodium falciparum*-infected erythrocytes express unique strain-specific antigens on their surface. *Infect Immun* 1993; **61**(1): 284-288.

[58] Fernandez V, Hommel M, Chen Q, Hagblom P, Wahlgren M. Small, clonally variant antigens expressed on the surface of the *Plasmodium falciparum*-infected erythrocyte are encoded by the rif gene family and are the target of human immune responses. *J Exp Med* 1999; **190**: 1393-1404.

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Assessing the association of severe malaria infection and ABO blood groups in northwestern Ethiopia

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ABSTRACT

Background & objectives: There is lack of adequate information on the association between severe malaria and some human genetic markers like ABO blood types. The study was undertaken to evaluate the association between severe malaria infection and ABO blood types among febrile patients attending Felegeselam Health Center, northwestern Ethiopia.

Methods: A total of 398 febrile patients were examined for malaria and tested for ABO blood groups in December 2011. The blood samples were collected by finger pricking, stained with Giemsa and slides were examined microscopically. ABO blood group was determined by agglutination test using agglutinating A and B monoclonal anti-sera together with parasite load count. Chi-square and ANOVA tests were used to assess the difference between frequencies and means, respectively.

Results: Out of 398 acute febrile patients, 201 (50.5%) were found to be infected with *Plasmodium* parasites. Of which 194 (48.74%) and 7 (1.76%) belong to *Plasmodium falciparum* and *P. vivax*, respectively. The distribution of ABO blood groups was O (46%), A (27.1%), B (23.1%) and AB (3.8%). The percentage of severe malaria with respect to blood group A, B, AB and O was found to be 40, 34.1, 14.3 and 5.1%, respectively. The association of severe malaria with non 'O' blood types was statistically significant ($\chi^2 = 31.246$, $p < 0.01$).

Interpretation & conclusion: The present findings indicate that individuals with blood groups A, B and AB are more susceptible for severe malaria infection than blood group O.

Key words ABO blood group; acute febrile illness; Ethiopia*; Plasmodium falciparum; P. vivax;* severe malaria

INTRODUCTION

The ABO blood grouping system consists of the A, B and H carbohydrate antigens produced by a series of enzymatic reactions catalyzed by glycosyltransferase and antibodies against these antigens. ABO blood grouping is based on the presence or absence of A and B blood group antigens on the surface of red blood cells (RBC) derived from inherited gene[1].

In clinical practice, ABO is the most important system for blood group compatibility and ABO antigen associations with infections[2]. The relationship between ABO and malaria was first suggested 40 years ago[3]. There is a hypothesis that *Plasmodium falciparum* malaria has shaped the distribution of ABO blood groups in humans[3].

Malaria has been the most important selective force on the human population, and several erythrocyte polymorphisms have evolved that confer the resistance to severe malaria. *Plasmodium falciparum* rosetting is reduced in blood group 'O' but the contribution of the ABO blood group system to protect severe malaria has received little consideration[4–5]. Most of the time individuals great parasite density in their peripheral blood have a higher risk of developing severe malarial disease. However, some individuals develop severe and even fatal malaria with a very low peripheral parasitaemia due to sequestration of the parasite in the deep tissue capillaries[6].

Many investigations have been conducted to find out whether or not ABO blood group antigens are associated with susceptibility, resistance, or severity of *P. falciparum* malaria. *Plasmodium falciparum* infections can be linked to the most severe forms of human malaria and virulence is associated with parasite reproduction rate and erythrocyte invasion mechanism[7].

However, some studies reported the absence of significant association between severe malaria and ABO antigens[8]. On the other hand, few studies have shown that high frequency of malaria episodes has been observed among blood group 'O' individuals as compared to other blood group individuals[9]. Large numbers of severe malaria cases were also reported among blood group 'A' individuals[10]. Furthermore, low parasitaemia was observed among uncomplicated malaria cases and blood group 'O' individuals[4, 10–11].

Variations in reports on the association of ABO blood groups and disease progression of *P. falciparum* malaria show the complexity of the interaction between the parasites and host immune responses. In addition, studies have shown the impact of other RBC polymorphisms including hemoglobin (Hb) abnormalities such as HbS, HbC and thalassemia[9, 12–14], deficiency in erythrocyte complement receptor (CR) or glucose-6-phosphate dehydrogenase on *P. falciparum* malaria susceptibility and severity[15–16] and lack of Duffy antigen[17] protection developing severe malaria in endemic areas. This makes it difficult to make a clear analysis on the association of severe malaria and ABO blood groups. Therefore, this study aims to assess the relationship of severe malaria infection with their ABO blood types among acute febrile patients who sought medical attention at Felegeselam Health Center, northwestern Ethiopia where *P. falciparum* is the dominant species.

MATERIAL & METHODS

Study area

A cross-sectional study was conducted at Felegeselam Health Center, northwestren Ethiopia, to assess the association of ABO blood group antigens with severe malaria in December 2011. Felegeselam is located at about 570 km away from Addis Ababa. The area has an elevation between 1000 and 1050 m above sea level. The study area has annual temperature ranging from 28–43°C with annual rainfall of 1050 mm. The total population of the study area is 50,307; of which 26,984 are males and 25,323 are females[18]. The study population comprised of febrile outpatients who sought medical attention at Felegeselam Health Center in December 2011. A total of 398 acute febrile patients were selected as study participants, excluding individuals who took antimalarial drugs within two weeks before the blood test, patients who were not living permanently in the area and who refused to participate in the study.

Clinical and laboratory diagnosis

Health staff members were trained about how to collect sample and explanation was given prior to the data collection. Capillary blood was collected by finger pricking using 70% alcohol and sterile disposable lancet. Thick and thin films were prepared on the same slide. Thin films were fixed with methanol. The blood films were stained with 6% Giemsa for 10 min. Finally, the films were examined under an oil immersion microscope objective (100×). Parasitaemia was determined for febrile patients who tested positive for *P. falciparum* and *P. vivax* by counting the number of parasites (asexual forms only) against 200 WBCs using hand tally counters. Then, the number of parasites per microliter (µl) of blood was calculated. If the parasitic load was greater than 10,000 parasites/µl of blood, the infection was said to be severe. On the other hand, the infection was considered as uncomplicated if the parasite load was lesser than 10,000 parasites/µl. This can be used to give a more accurate figure with appropriate adjustment of the multiplication factor[19]. In addition, the blood group of the study participants was determined by direct slide method, using agglutinating A and B monoclonal Eryclone® antisera together with the former procedures.

Data analysis

Data were entered into Microsoft Excel, exported to SPSS version 16 and analyzed. Chi-square test was used to assess the difference between frequencies (the associations between blood groups and *P. falciparum* malaria cases). ANOVA was used to test the difference between parasitaemia means. Observed difference was considered to be significant for $p < 0.05$.

Ethical approval

The study obtained ethical clearance from Microbiology, Immunology and Parasitology Department Ethical Review Committee, College of Health Science, Addis Ababa University and from the Pawe Woreda Health Office. Written informed consent was obtained from every study participant and guardians in case of children. Malaria positive cases were treated with antimalarial drugs based on the current national treatment guidelines of Ethiopia.

RESULTS

Malaria infection

Out of 398 acute febrile cases who visited Felegeselam Health Center for medical attention, 201 (50.5%) were found to be infected with *Plasmodium* parasites as determined by microscopy. Of which *P. falciparum* and *P. vivax* accounted for 194 (48.7%) and 7 (1.8%), respectively (Table 1). The prevalence of malaria

Table 1. Prevalence of malaria by age and sex among acute febrile cases

Age (yr)/ Sex	No. of cases examined	*Pf*	*Pv*	Total	χ^2, *p*-value
1–4	52	23 (44.2)	1 (1.9)	28 (53.8)	1.26, 0.87
5–17	166	86 (51.8)	3 (1.8)	77 (46.4)	
≥ 18	180	85 (47.2)	3 (1.7)	92 (51.1)	
M	176	83 (47.2)	3 (1.7)	90 (51.1)	0.22, 0.896
F	222	111 (50)	4 (1.8)	107 (48.2)	
Total	398	194 (48.7)	7 (1.8)	197 (49.5)	

Figures in parentheses indicate percentages.

was highest among 5 to 17 yr as compared with under five and older age groups but the difference was not significant (p >0.05). Similarly, the prevalence was higher among females (51.8%) than males (48.9%) and this difference was not statistically significant (χ^2=0.22, p >0.05) (Table 1).

ABO blood groups and malaria infection

All febrile patients examined for malaria were also tested for ABO blood groups. Accordingly, 46, 27.1, 23.1 and 3.8% were found to be blood types of O, A, B and AB (Table 2). All of them were Rh positive. There were 200 volunteer blood donors used as a control to assess the distribution of blood types among the community. The donors who lived in that specific community and donated blood in Pawe Hospital were selected based on their address. The distribution of ABO phenotypes among blood donors was O (60%), A (26%), B (12.5%) and AB (1.5%). Only one blood donor with blood group O was Rh (–)ve.

In general, malaria infection was observed with the highest proportion (53.6%) among individuals with O blood group, followed by those with blood group A (50.9%) (Table 2). In all blood groups of acute febrile illness (AFI) cases that have body temperature above normal, the prevalence of *P. falciparum* malarial infection was higher than that of *P. vivax* infection. Prevalence of *P. falciparum* infection was high (51.9%) among individuals with O blood group, followed by individuals with blood group A (50.9%) (Table 2). The percentage of *P. falciparum* infection among blood groups A, B, AB and O was 50.9, 41.3, 40 and 51.9%, respectively (Table 2). The proportion of A or O was higher in individuals with *P. falciparum* infection compared with non-infected individuals (Table 2) who were screened. *Plasmodium falciparum* infection did not show significant association (p >0.05) with the age among different blood groups.

Table 2. Prevalence of malaria among the study participants based on their blood types

Blood group	No. of cases examined	*Pf* (%)	*Pv* (%)	Non-infected (%)	Total (%)	χ^2, *p*-value
A	108	55 (50.9)	0 (0)	53 (49.1)	55 (50.9)	7.96, 0.24
B	92	38 (41.3)	3 (3.3)	51 (55.4)	41 (44.6)	
AB	15	6 (40)	1 (6.7)	8 (53.3)	7 (46.7)	
O	183	95 (51.9)	3 (1.6)	85 (46.4)	98 (53.6)	
Total	398	194 (48.7)	7 (1.8)	197 (49.5)	201 (50.5)	

Figures in parentheses indicate percentages.

ABO blood type and P. falciparum parasitaemia

There were 42 severe malaria (parasitic load >10,000 parasites/µl) and 159 uncomplicated malaria (parasitic load <10,000 parasites/µl) cases isolated in this study. Parasitic load was used as a marker of differentiation between severe and uncomplicated malaria. Prevalence of severe malaria among blood groups A, B, AB and O was 39.3, 35, 14.3 and 5.1%, respectively (Table 3). All severe malaria cases were caused by *P. falciparum*. In general, severe malaria infection showed significant association (χ^2= 30.54, p <0.01) with non 'O' blood groups. The highest proportion of severe malaria was observed among participants with blood group A (39.3%) but the least proportion was found to be in O blood group (5.1%) (Table 3).

The median parasitic count for all positive, uncomplicated malaria and severe malaria cases were 82, 54 and 396 parasites/µl, respectively. Similarly, the median parasitic count for each blood group A, B, AB and O was 128.5, 140, 84 and 56 parasites/µl, respectively. On the other hand, the mean parasitic count for all positive, mild malaria and severe malaria cases were 144, 169.2 and 539.4 parasites/µl of blood, respectively. The mean parasitic count for each blood group A, B, AB and O was 228.9, 125, 108.7 and 55.7 parasites/µl of blood, respectively.

There were also three severe malaria cases whose parasitic load was greater than 100,000 parasites/µl of blood and two of them had the blood type 'A' where as one had blood group 'O'. The frequencies of A, B, AB and O blood groups in uncomplicated malaria cases was 21.4, 16.5, 3.8 and 58.9%, respectively (Table 3). The prevalence of uncomplicated malaria among blood groups A, B, AB and O was 60.7, 65, 85.7 and 94.9%, respectively (Table 3). This indicated that most uncomplicated malaria cases had blood group O followed by blood group AB.

The chance of having severe malaria infection in patients with blood groups A, B and AB was 12 (χ^2= 28.801,

Table 3. Percentage distribution of malaria characters based on their ABO blood types

Malaria characteristics	Blood group types				Total	χ^2, *p*-value
	A	B	AB	O		
Severe	22 (39.3)	14 (35)	1 (14.3)	5 (5.1)	42	30.54, 0.0
Uncomplicated	34 (21.4)	26 (16.4)	6 (3.8)	93 (58.5)	159	
Total	56 (27.9)	40 (19.9)	7 (3.5)	98 (48.6)	201	

Figures in parentheses indicate percentages.

$p = 0.0$, OR=12.04, 95% confidence interval (CI) = 4.222–34.306), 10 ($\chi^2 = 21.387$, $p = 0$, OR = 10.02, 95% CI = 3.301–30.385) and 3.1 ($\chi^2 = 2.284$, $p = 0.335$, OR = 3.10, 95% CI = 0.311–30.929) times higher than individuals showing blood group O phenotypes, respectively.

The parasite load for all malaria positive cases was ≥ 100 parasites/µl. About 21.8% of *P. falciparum* infected study participants of blood group A, 12.2% of blood group B, 28.6% of blood group AB and 21.4% blood group O had shown parasitic load of ≤ 1000 parasites/µl of blood. On the other hand, 78.2% (43/55), 85.4% (35/41), 71.4% (5/7) and 77.6% (76/98) *P. falciparum* infected individuals of blood group A, B, AB and O, respectively had parasite density of ≥1000 parasites/µl of blood. Only 3.6% (2/55) and 1% (1/98) of *P. falciparum* infected patients with blood group A and O respectively had a parasite density of ≥100,000 parasites/µl of blood.

DISCUSSION

In the present study, high proportion of blood group O (46%) phenotype was observed among the study participants. This agrees with some previous reports (51.3%) in southern Ethiopia[10], (45.7%) in Awash, Metehara and Ziway areas of Ethiopia[11], (55.83%) in Amazon region of Brazil[20] and 54.4% in Zimbabwe[21] which showed high frequency of group 'O' than non 'O' phenotypes in tropical regions where malaria is prevalent.

Significantly higher proportion of individuals with blood groups A, B and AB were found to have severe *P. falciparum* infection than blood group O. This was consistent with previous reports[4, 10–11, 21] which emphasised that non O blood groups were more susceptible to *P. falciparum* infection than those with O blood group.

The lowest mean parasitaemia was observed among individuals with blood group O as compared to blood groups A, B and AB in this study. The results of the present study is in agreement with reports from southern Ethiopia[10] and Awash, Metehara and Ziway areas of Ethiopia[11] which showed that blood group O had the lowest mean parasitaemia.

In contrast to the present study, previous reports indicated high prevalence of blood group A and low prevalence of blood group O phenotypes in colder regions where malaria has not been endemic[22]. Hence, the present finding seems to confirm the hypothesis about a selective survival evolutionary advantage of *P. falciparum* infection in blood group O compared with non O blood groups in malaria endemic areas.

The present study revealed that there was a difference in the frequency of ABO blood groups between controls and those with uncomplicated malaria cases. Similarly, individuals with severe malaria also had significantly higher parasite count than patients with uncomplicated malaria. This study is consistent with previous reports[5, 10]. In contrast to the present study, other authors reported the absence of significant difference in the frequency of parasitic load between severe and uncomplicated malaria cases[11]. However, this finding can be explained by the fact that in severe falciparum malaria infection, parasitized erythrocytes at schizont stage are known to be sequestered in deep tissue capillaries and may result in low parasite count in the peripheral blood[23]. In the present study, the severity of infection with falciparum malaria may have been greater.

The mechanism by which blood group 'A' promote susceptibility and blood group 'O' confers a relative protective effect against severe malaria is not well understood; however, different studies have done on the basis of rosette formation. Several reports supported that blood group A stands for a risk factor for high chance of rosetting, which is usually characterized by high *P. falciparum* parasitaemia during malaria infection and a reducing effect of blood group 'O' on rosetting[5, 24–25]. The presence of several glycosylated intracellular adhesion molecules and chondroitin sulfate A[26], CD36[27] and Duffy antigen[17] in blood group 'A' cells promote a high chance of binding with the rosette-forming surface molecules of *P. falciparum* and leads to development of severe malaria.

The present study only employed parasitic load as a laboratory marker to determine the association of ABO blood groups and severe malaria. The study also did not consider factors like hemoglobin S, hemoglobin C, iron status of the host, place of residence of the study population which could affect the nature of *P. falciparum* infection among the study population. Nevertheless, the findings indicated that severe malaria is associated with blood groups and individuals of blood group A, B and AB are more susceptible to severe malaria infection as compared with individuals with the blood type O. Further, indepth studies are needed to clearly assess the role of ABO blood groups in severe malaria cases to minimize mortality and morbidity of malaria in endemic areas.

REFERENCES

1. Yamamoto F, McNeill PD, Hakomori S. Genomic organization of human histo-blood group ABO genes. *Glycobiology* 1995; *5:* 51–8.
2. Cserti CM, Dzik WH. The ABO blood group system and *Plasmodium falciparum* malaria. *Blood* 2007; *110:* 2250–8.

3. Athreya BH, Coriell LL. Relation of blood groups to infection a survey and review of data suggesting possible relationship between malaria and blood groups. *Am J Epidemiol* 1966; *88:* 2.
4. Migot-Nabias F, Mombo LE, Luty AJ, Dubois B, Nabias R, Bisseye C. Human genetic factors related to susceptibility to mild malaria in Gabon. *Genes Immun* 2000; *1:* 435–41.
5. Rowe JA, Handel IG, Thera MA, Deans AM, Lyke KE, Kone A, *et al*. Blood group O protects against severe *Plasmodium falciparum* malaria through the mechanism of reduced resetting. *Proc Natl Acad Sci USA* 2007; *104:* 17471–6.
6. Gay F, Zougbede S, Dilimabaka N, Rebollo A, Mazier, DMoreno A. Cerebral malaria: What is known and what is on research? *Rev Neurol* 2012; *168:* 239–56.
7. Chotivanich K, Udomsangpetch R, Simpson J, Newton P. Parasite multiplication potential and the severity of falciparum malaria. *J Infect Dis* 2000; *181:* 1206–9.
8. Kuadzi JT, Badu GA, Addae MM. *Plasmodium falciparum* malaria in children at a tertiary teaching hospital: ABO blood group is a risk factor. *Pan Afr Med J* 2011; *10:* 2.
9. Theresa KN, Paul W, Jane-Frances A. Effective of ABO/RH blood groups, G-6-PD enzyme activity and hemoglobin genotypes on malaria parasitaemia and parasite density. *Afr J Health Sci* 2004; *11:* 93–7.
10. Zerihun T, Degarege A, Erko B. Association of ABO blood group and *Plasmodium falciparum* malaria in Dore Bafeno Area, southern Ethiopia. *Asian Pacific J Trop Biomed* 2011; *1:* 289–94.
11. Tekeste Z, Petros B. The ABO blood group and *Plasmodium falciparum* malaria in Awash, Metehara and Ziway areas of Ethiopia. *Malar J* 2010; *9:* 280.
12. Butthep P, Wanram S, Pattanapanyasat K, Vattanaviboon P, Fucharoen S, Wilairat P. Cytoadherence between endothelial cells and *P. falciparum* infected and noninfected normal and thalassemic red blood cells. *Cytometry Part B* (Clin Cytom) 2006; *70:* 432–42.
13. Hill AV, Allsopp CEM, Kwiatkowski D, Anstey NM, Twumasi P, Rowe PA, *et al.* Common west African HLA antigens are associated with protection from severe malaria. *Nature* 1991; *352:* 595–600.
14. Karyakarte RP, Damel AS. *Medical parasitology*. India: Runabha Books and Allied (P) Ltd. 2003; p. 62–87.
15. Beutler E, Vulliamy TJ. Hematologically important mutations: Glucose-6-phosphate dehydrogenase. *Blood Cells Mol Dis* 2002; *28:* 93–103.
16. Greene LS. G6PD deficiency as protection against falciparum malaria: An epidemiologic critique of population and experimental studies. *Year B Phys Anthropol* 1993; *36:* 153–78.
17. Singh SK, Singh AP, Pandey S, Yazdani SS, Chitnis CE, Sharma A. Definition of structural elements in *Plasmodium vivax* and *P. knowlesi* duffy-binding domains necessary for erythrocyte invasion. *Biochem J* 2003; *374:* 193–8.
18. Federal Democratic Republic of Ethiopia population census commission 2007. Summary and statistical reports of the 2007 population and housing census, summary and statistical report of the 2007-CSA. Available from: *http//www.csa.gov.et/pdf/Cen2007_firstdraft.pdf.*
19. Moody A. Rapid diagnostic tests for malaria parasites. *Clin Microbiol Rev* 2002; *15:* 66–78.
20. Carvalho DB, Mattos LC, de Souza-Neiras WC, Bonini-Domingos CR, Cósimo AB, Storti-Melo LM, *et al*. Frequency of ABO blood group system polymorphisms in *Plasmodium falciparum* malaria patients and blood donors from the Brazilian Amazon region. *Genet Mol Res* 2010; *9:* 1443–9.
21. Fischer PR, Boone P. Short report: Severe malaria associated with blood group. *Am J Trop Med Hyg* 1998; *58:* 122–3.
22. *Implementation of the global malaria control strategy: Report of a WHO Study Group*. Geneva: World Health Organization 1993.
23. Dondorp AM, Ince C, Charunwatthana P, Hanson J, van Kuijen A, Faiz MA. Direct *in vivo* assessment of microcirculatory dysfunction in severe falciparum malaria. *J Infect Dis* 2008; *197:* 79–84.
24. Carlson J, Helmby H, Hill A, Brewster D, Greenwood B, Wahlgren M. Human cerebral malaria: Association with erythrocyte rosetting and lack of anti-rosetting antibodies. *Lancet* 1990; *336:* 1457–60.
25. Deepa, Alwar VA, Rameshkumar K, Ross C. ABO blood groups and malaria related clinical outcome. *J Vector Borne Dis* 2011: *48:* 7–11.
26. Brendan SC, Alen FC. *Plasmodium falciparum* virulence determinants unveiled genome. *Biology* 2002; *3:* 11.
27. Udomsangpetch R, Webster HK, Pattanapanyasat K, Pitchayangkul S, Thaithong S. Cytoadherence characteristics of rosette-forming *Plasmodium falciparum*. *Infect Immun* 1992; *60:* 4483–90.

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Relationship between ABO blood groups and skin cancers

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Main Content

Relationship between ABO blood groups and skin cancers

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Abstract

Studies of associations between various cancers and the ABO blood groups have shown elevated relative risks for some categories of disease. To date, no report has evaluated the relationship between the ABO blood groups and the skin cancers. To investigate this association, we conducted a retrospective study of premalignant and malignant tumors diagnosed in Turkey. All tumors were histologically confirmed. Blood information was obtained for 98 individuals with premalignant and malignant skin tumors, and the distribution of ABO and Rh blood type for cases was compared with that of 419 healthy blood donors from the same geographic area. Although patients with blood group A were higher, group 0 lower than in controls, the differences were not significant. The distribution of Rh factor, blood group B and AB among cases and controls also did not differ significantly. We found a significant relationship between age and skin cancer (p=0.0001). Old patients had 1.238 times higher risk for skin cancer. Further studies in larger series on blood group antigens are needed to elucidate the relationship between these antigens and skin cancer.

Introduction

Blood group antigens, which are the major alloantigens in humans, are present on the surface of red blood cells and various epithelial cells. As the majority of human cancers are derived from epithelial cells, changes in blood group antigens are an important aspect of human tumor [1, 2, 3, 4]. In some tumors, alteration of ABO/Lewis-related antigens is associated with malignant transformation [5-19].

The relationship with blood groups and incidence, clinicopathologic parameter and prognosis had been studied in many cancers such as esophagus, cardiac, gastric, lung, laryngeal, hypopharyngeal, salivary gland, gynecologic, colorectal, pancreatic, bone, urinary bladder, ureter, renal, breast, prostate, testicular tumors and uveal melanoma [5-19]. Additionally, ABO genes are distributed differently among socioeconomic groups and we know that socioeconomic status is one of the risk factors for disease [20]. Therefore, we hypothesized that analysis of blood group and related antigens on skin cancers would provide useful information on the risk factors. To date, no report has evaluated the relationship between the ABO blood groups and the skin cancers. Thus, a retrospective assessment of the relation between blood groups and malignant and premalignant skin lesions was performed with a view to start the study of the genetics of these cancers in Turkey.

Material and methods

The study was approved by the Ethics Committee of the Medical Faculty of the University of Mersin. All the patients and controls accepted blood examination. Ninety eight patients with skin cancer including 23 squamous cell carcinoma (SCC), 42 basal cell carcinoma (BCC), 33 *in situ* squamous cell carcinoma (ISCC) and 419 control subjects were enrolled in this study. Control subjects were selected among healthy people with no history of cardiovascular disease, cancer, chronic degenerative neurologic disease, chronic obstructive pulmonary disease, hepatitis, allergies in general or alcohol abuse. The diagnosis of skin cancer was based on dermatopathologic examination. Documentation of clinical findings included: (a)Gender (b)Age (c)Clinic types of the skin cancer including SCC, BCC and ISCC and (d) tumor location.

Blood samples were obtained into vacuum tubes containing EDTA (vacutainer, Becton Dickinsen, Marseilles, France) from each donor's venous circulation. ABO and Rh blood typing were carried out with tube method and gel method.

Tube method: One drop of anti-A, anti-B, or anti-D (Eryclone, Tulip Diagnostics, Bambolim, India) was added to the appropriately labeled tube. A 5 percent suspension of red blood cells (RBC) was made in isotonic saline. 1 drop was added to tubes containing anti-A, anti-B, or anti-D. The contests of the tubes were mixed thoroughly, and the tubes were centrifuged for 20 seconds at 3400 rpm. Tubes were read macroscopically for agglutination.

Gel method: A 5 percent RBC suspension was prepared in diluent (modified bromelin solution for red cell suspensions). Gel cards (Diaclon ID, Diamed AG, Cressier, Switzerland) were used for ABO and Rh typing. 10 µL of RBC suspension was added to the gel microtubes containing anti-A, anti-B, anti-D, and control reagents, respectively. 50 µL of donor plasma were added to microtubes for reverse ABO group testing. The ID cards were centrifuged at 895 rpm 10 minutes in the centrifuges (ID-centrifuge). A positive reaction (4+) was determined by the formation of a red line on the gel surface, whereas intermediate reactions were characterized by red agglutinates distributed throughout the gel. With a negative reaction, a compact button of cells formed on the bottom of the microtube.

Multivariate analysis was carried out by using a logistic regression model [21]. Logistic regression analysis was chosen to study the predictive value of each risk factor such as sex, age and blood types. *P* values < 0.05 were regarded as statistically significant.

Results

Among 98 subjects with skin cancer, the ABO blood types of A, B, 0 or AB were 45 (% 45), 15 (15 %), 30 (30 %) and 8 (8 %) respectively. For controls, the ABO blood types were 147 (35 %), 66(15 %), 188 (44 %) and 18 (4 %) respectively. The mean age was 53±6 (303 male, 116 female) for controls, and 63±14 (70 male, 28 female) for patients.

Although patients with blood group A were higher, group 0 lower than in controls, the differences were not significant. In our study, we could not find any significant relationship according to blood types in total patient group, and also among skin cancer types. (Tables 1-4). The distribution of Rh factor among cases and controls did not differ significantly. There was no difference in ABO blood group or Rh factor and tumor location and sex. We found a significant relationship between age and skin cancer (p = 0.0001). Old patients had 1.238 times higher risk for skin cancer.

Discussion

Skin cancer is the most common type of cancer in humans. The etiopathogenesis of skin cancers is still unknown. Exposure to sunlight, particularly ultraviolet B radiation is a strong risk factor associated with skin cancer development. Other known risk factors include exposure to ionizing radiation, arsenicals, polyaromatic hydrocarbons, transplantation-associated immunosuppression and psoralen plus ultraviolet A therapy [22]. The explanation for the association between ABO blood groups and some special diseases was still unclear. Many reports have shown that blood group antigen expression in tumor is correlated with metastasis and prognosis [23, 24]. The loss or presence of blood group antigens can increase cellular motility or facilitate the interaction between tumor cells and the endothelium of distant organs [25].

In our patients, we did not find any significant association with blood types. Although patients with blood group A were higher than in controls, there was no statistically significant. Some authors observed that A blood type was significantly more frequent in patients with laryngeal, hypopharinx, pancreatic, breast, testicular and bone cancers [9, 10, 13, 14, 16, 18]. To our knowledge, no report has evaluated the relationship between the ABO blood groups and the skin cancers. Jager et al. observed that no significant differences in survival or the development of metastases in patients with uveal melanoma regard to the ABO antigens [19]. ABO blood group genes are map at 9q in which the genetic alteration is common in many cancers [26]. Thus, ABO blood group antigen expression may be effected by the genetic change of tumors [22]. On the other hand, it is possible the observed associations are not due to the blood group antigens themselves, but to the effects of genes closely associated with them. Additionally it might have nothing to do with molecular mechanisms or genetics. It is merely the result of population history, environment, diet and customs [22]. In conclusion, our study shows no significant association of ABO blood groups with skin cancers. Further studies on blood group antigens in larger series are needed to elucidate the relationship between blood group antigens and skin cancers.

References

1. Dabelsteen E. Cell surface carbohydrates as prognostic markers in human carcinomas. J Pathol 1996; 179:358-69.

2. Hakomori S. Tumor-associated carbonhydrate antigens. Ann Rev Immunol 1984; 2:103-26.

3. Lee JS, Ro JY, Sahin AA, Hong WK, Brown BW, Mountain CF, et al. Expression of blood-group antigen A-a favorable prognostic factor in non-small-cell lung cancer. N Engl J Med 1991; 324:1084-90.

4. Coon JS, Weinstein RS. Blood group-related antigens as markers of malignant potential and heterogeneity in human carcinomas. Hum Pathol 1986; 17:1089-106.

5. Su M, Lu SM, Tian DP, Zhao H, Li XY, Li DR, Zheng ZC. Relationship between ABO blood groups and carcinoma of esophagus and cardia in Chaosan inhabitants of China. World J Gastroenterol 2001; 7:657-661.

6. Nakagoe T, Fukushima K, Nanashima A, Sawai T, Tsuji T, Jibiki MA, et al. Comparison of the expression of ABH/Lewis-related antigens in polypoid and non-polypoid growth types of colorectal carcinoma. J Gastroenterol Hepatol 2001; 16:176-183.

7. You WC, Ma JL, Liu WD, Gail MH, Chang YS, Zhang L, et al. Blood type and family cancer history in relation to precancerous gastic lesions. Int J Epidemiol 2000; 29:405-407.

8. Graziano SL, Tatum AH, Gonchoroff NJ, Newman NB, Kohman LJ. Blod group antigen A, and flow cytometric analysis in resected early-stage non-small cell lung cancer. Clin Cancer Res 1997; 3:87-93.

9. Pyd M, Rzewnicki I, Suwayach U. ABO blood groups in patients with laryngeal and hypopharyngeal cancer. Otolaryngol Pol 1995; 49:396-8.

10. Pinkston JA, Cole P. ABO blood groups and salivary gland tumors (Alabama, United States). Cancer Causes Control 1996; 7:572-4.

11. Marinaccio M, Traversa A, Carioggia E, Valentino L, Coviella M, Salamanna S, et al. Blood groups of the ABO system and survival rate in gynecologic tumors. Minerva Ginecol 1995; 47:69-76.

12. Juhl BR. Blood group antigens in transitional cell tumors of the urinary bladder. An immunohistochemical study. Dan Med Bull 1994; 41:1-11.

13. Vioque J, Walker AM. Pancreatic cancer and ABO blood types: a study of cases and controls. Med Clin (Barc.) 1991; 96:761-4.

14. Jia DX. Bone tumor and ABO blood type. Zhonghua Zhong Liu Za Zhia 1991; 13:220-2.

15. Cordon-Cardo C, Reuter VE, Finstad CL, Sheinfeld J, Lloyd KO, et al. Blood group-related antigens in human kidney: modulation of Lewis determinants in renal cell carcinoma. Cancer Res 1989; 49:212-8.

16. Anderson DE, Haas C. Blood type A and familial breast cancer. Cancer 1984; 54:1845-9.

17. Walker PD, Karnik S, de Kerion JB, Pramberg JC. Cell surface blood group antigens in prostatic carcinoma. Am J Clin Pathol 1984; 81:503-6.

18. Jordan GH, Lynch DF. Relationship of blood group to testicular carcinoma. Urology 1983; 22:265-7.

19. Jager MJ, Völker-Dieben HJ, De Wolff-Roundaal D, Kakebeeke-Kemme H, DÕAmaro J. Possible relation between HLA and ABO type and prognosis of uveal melanoma. Documente Ophthalmologica 1992; 82:43-47.

20. Petrakis NL, King MC. Genetic markers and cancer epidemiology. Cancer 1977; 39:1861-1866.

21. Hosmer DW, Lemeshow S. Applied Logistic Regression. New York: John Wiley & Sons, 1990.

22. Armstrong BK, Kricker A. Skin Cancer. Dermatol Clin 1995; 13:583-594.

23. Nakagoe T, Nanashima A, Sawai T, Tuji T, Ohbatake M, Jibiki M, Yamaguchi H, et al. Expression of blood group antigens A, B, and H in carcinoma tissue correlates with a poor prognosis for colorectal cancer patients. J Cancer Res Clin Oncol 2000; 126:375-382.

24. Moldvay J, Scheid P, Wild P, Nabil K, Siat J, Borrelly J, et al. Predictive survival markers in patients with surgically resected non-small cell lung carcinoma. Clin Cancer Res 2000; 6:1125-1134.

25. Ichikawa D, Handa K, Hakomori S. Histo-blood group A/B antigen deletion /reduction vs. continuous expression in human tumor cells as correlated with their malignancy. Int J Cancer 1998; 76:284-289.

26. Hu N, Roth MJ, Polymeropolous M, Tang ZZ, Emmert-Buck MR, Wanf QH, et al. Identification of novel regions of allelic loss from a genome wide scan of esophageal squamous cell carcinoma in a high risk Chinese population. Genes Chromosomes Cancer 2000; 27:217-228.

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Research Article

RELATION BETWEEN PRAKRITI (AYURVEDIC CONCEPT OF CONSTITUTION) AND BLOOD GROUP AMONG AYURVEDIC STUDENTS

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ABSTRACT

Background: The word 'Prakriti' means 'nature' and this reflects the natural state of human beings on an anatomical, physiological, and psychological level. Prakriti or Constitution is an important concept of Ayurved. Maintenance of health, prevention of disease, achieving longevity & treatment of diseases depends on this fundamental theory of understanding human being. Ayurveda considers Vata, Pitta and Kapha or Tridoshas (Ayurvedic biological constituents) as main determinants of human prakriti. Ayurveda classifies entire human population into seven constitutional types (Prakriti), based on the dominance of any single or a combination of two or three Doshas The present study was carried out to study the relation between blood groups (A B O system) varied in the different *prakriti* subtypes was studied.

Aims: To study the Relation between prakriti & blood group.

Objectives of the Study: 1)To study the prakriti with the subjective parameters mentioned in the Text in Ayurvedic Students.2)To study the blood group of each student .3) To study the Relation between prakriti & blood group.

Material & method: After obtaining Institutional Ethics Committee permission, normal healthy individuals of either sex between the age group 17 to 22 years were recruited in the study. Their prakriti evaluation was done using a standardized validated questionnaire 1) Assessment of Prakriti among the Volunteers.2) Assessment of Blood Group.

Result: It was seen that Vata Prakriti was associated with blood group A, Pitta Prakriti was associated with blood group O and Kapha Prakriti was associated with blood group B.

Conclusion: Association was found between *Prakriti* and Blood group in some extent.

Keywords: *Prakriti,* Blood groups, Tridosha, Vata, Pitta, Kapha.

INTRODUCTION

The word 'Prakriti' means 'nature' and this reflects the natural state of human beings on an physical, physiological, and psychological level. The concept is claimed to be useful in predicting an individual's susceptibility to a particular disease, prognosis of that illness and selection of therapy[1-3]. Ayurveda attributes these constitutional characteristics of an individual to the preponderance of certain "doshas". Three main doshas are described, viz. vata, pitta and kapha. Kapha dosha is the "anabolic", synthetic dosha, responsible for growth and maintenance of structure . The pitta dosha is the one responsible for metabolism, including digestion in the gut, and cellular or sub-cellular metabolism[2]. Vata dosha is responsible for movement (muscular, nervous energy etc.). Based on the predominance of individual doshas, there are three major types of prakriti named after predominant dosha, viz., vata, pitta and kapha. The prakriti is believed to be determined at the time of conception and is influenced by the milieu interior of the womb and the dietary habits and lifestyle of the mother[3]. These prakritis exhibit attributes of the dominant Dosha in physical, physiological and psychological characteristics. The disturbance in equilibrium of these doshas can lead to disease according to the prakriti of the person for example; a pitta prakriti person is described to be more prone to peptic ulcers, hypertension, and skin diseases, a vata prakriti person to backache, joint aches and crackling joints while individuals with kapha prakriti are prone to obesity, diabetes and atherosclerosis[1-6] . Since prakriti has been described to have genetic origin in Ayurvedic texts[3].

There is certain relation between parents and inborn child in blood grouping. The heredity gene is directly concern with the inborn child's metabolic pattern too. The inborn metabolic pattern of an individual is the continuation of metabolic

pattern of the parents. So these relations according to modern discovery are due to gene. But around thousands of years ago no word called "gene was used. In fact the pitrij bhav (Father's character) an matrij bhav (Matrij character) refer to gene XX and XY, it is not so direct but pointing towards these genes. Still re discovery of prakriti is not there in modern techniques[5].

A blood type (also called a blood group) is a classification of blood based on the presence or absence of inherited antigenic substances on the surface of red blood cells (RBCs). These antigens may be proteins, carbohydrates, glycoproteins, or glycolipids, depending on the blood group system. Some of these antigens are also present on the surface of other types of cells of various tissues. The blood type (or blood group) depends on the types that are been passed down to you from your parents. So Blood group & Prakriti both are inborn, constant in nature, & there are no change .On the basis of this ,we studied the Relation between prakriti & blood group among Ayurvedic students.

Aims

To study the Relation between prakriti & blood group among Ayurvedic students.

Objectives of the Study

1) To study the prakriti with the subjective parameters mentioned in the Text in Ayurvedic Students
2) To study the variability of blood group of each student
3) To study the Relation between prakriti & blood group Among Ayurvedic students

MATERIALS AND METHODS

100 randomly selected clinically healthy volunteers belonging to the Single constitutional types were studied for the variability of Blood Group.

Observational cross sectional study was conducted among first year ayurvedic students at Mahatma Gandhi Ayurvedic College and research centre, Salod (H), Wardha. Study was conducted from April 2014 to March 2015. All the 100 Healthy Students between 17 to 22 year age group were selected for the study.

Approval was taken from the Institutional ethics committee (Datta Meghe Institute of Medical sciences, DU, Wardha) on 5.3.2014 with Ref.No. DMIMS (DU)/IEC/2013-14/565. Informed written consent was taken from the study subjects.

Students were interviewed by predesigned questionnaire that contains the information about Prakriti and blood groups were determined by tile agglutination method, by using commercially available Antiserum A and Antiserum B (Tulip Diagnostics (p) Ltd).

Assessment Criteria

1) Assessment of Prakriti among the Volunteers- Preparation of the Questionnaire

The Prakriti (constitution) was determined according to the description given in Ayurvedic Text Books viz. the Charak Samhita (1), Sushrut Samhita (2) and Sartha Vagbhatta (3). For the study, the prakriti of each volunteer was assessed using the validated questionnaire. It was further confirmed by an Ayurvedic physician to assess various physical, physiological and psychological characters as described in Ayurvedic texts[5]. The subjects were given a proforma which contained subjective and objective parameters. The subjective parameters were of psychological and physiological types and the objective parameters were of physical and physiological types. More importance was given to the objective parameters than the subjective ones.

These volunteers were enrolled in the study only after undergoing the clinical examination and being declared clinically healthy and physically fit. A written consent was obtained from them to participate in the study. These 100 volunteers were then subjected to some simple experiments in the human physiology laboratory of the department of Kriya Sharir as described in the following paragraphs.

2) Assessment of Blood Group:

Blood group was also determine with a kit from Sera Eryclone, manufactured by Tulip Diagnostics (P) Ltd. Blood is drawn from by capillary method. The test to determine your blood group is called ABO typing. Blood sample is mixed with antibodies against type A and B blood, and the sample was checked to see whether or not the blood cells stick together (agglutinate). If blood cells stick together, it means the blood reacted with one of the antibodies.

OBSERVATIONS AND RESULTS

The correlation between the Prakriti and the blood groups was studied. It was seen that Vata Prakriti was associated with blood group A, Pitta Prakriti was associated with blood group O and Kapha Prakriti was associated with blood group B. Blood group AB could not be associated with any Doshas (Ayurvedic Biological Constituents) probably because of its small sample size.

Table: Association between blood groups and the Prakriti

Blood Group	Vata	Pitta	Kapha	Total
A	10	4	5	19
B	6	8	13	27
AB	1	7	5	13
O	6	25	10	41
Total	23	44	33	100

We found that the number of Pitta predominant persons was maximum and that of vata predominant was minimum. Out of 100 subjects, 23 % were Vata predominant, Pitta predominant were 44 % and Kapha predominant were 33%.

DISCUSSION

Many research workers have tried to find out Prakriti (Ayurvedic constitution) of different groups of people. Some

have tried to find out association between Prakriti (Ayurvedic constitution) and different diseases. Some research workers have studied association between psychological inclinations and Prakriti (Ayurvedic constitution). Some have tried to find out whether parameters of Prakriti (Ayurvedic constitution) can be helpful in doing differential diagnosis of different diseases without taking help of pathological investigations[7-8].
The present study was designed to find out whether there is any correlation between Tridoshas (Ayurvedic biological constituents) and blood groups.
Studies conducted by Dr. Mrs. Purandare V R "Correlation Between ABO Blood Groups And Tridoshas (Ayurvedic Biological Constituents)"in 447 volunteers, the significant correlation was found. Kapha (Ayurvedic biological constituent) was found to be correlated with blood group A, Pitta (Ayurvedic biological constituent) was correlated with blood group B and Vata (Ayurvedic biological constituent) was correlated with blood group O[9].
Dr. Gaikwad, Tilak Ayurved Mahavidyalaya, Pune. tried to find out relation between blood group & constitution. 500 volunteers kapha constitution people have tendency to be 'B' positive blood groups, Pitta constitution people have tendency of 'O' positive blood Groups, Vata constitution people have tendency of 'A' positive blood groups.
Dr. Mohinder Pal Singh studied ratio between Kapha pradhan prakruti & blood group with resct to sex in Bharati Vidyapeeth University, Pune. The correlation was found that Kapha biotype male patient have predominantly B positive blood group & Kapha biotype female patient have predominantly O positive blood group.
Dr.Supriya Bhalerao studied prakriti (Ayurvedic concept of constitution) and variations in platelet aggregation. The findings of study however can have implications with respect to pharmacogenomics & study of dose-response relationships. These findings can also prove useful for the randomization in clinical trial design, as randomization would be best within the specific prakriti or dosha predominant sub-groups than across an aggregated population[10].
In the present study correlation between Prakriti (Ayurvedic Biological Constituents) and blood groups was found, Blood group A with Vata predominance, Blood group B with kapha predominance, Blood group O with Pitta predominance & Blood group AB could not be correlated with any Prakriti probably because of its small sample size.

CONCLUSION

In the present study, an attempt has been made to associate A B O blood groups with the Prakriti predominant Doshas. We got association between group A and vata, group O with Pitta and B with Kapha. As there is association between the blood groups and Prakriti . Blood groups are genetic markers; the association may be further extended to use Prakriti for genetic study. A large sample size is necessary for the derivation of positive conclusion.

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REFERENCES

1. Sutra S, Charak S (Eds): '11th Adhyaya' 5th edition. Varanasi: Chaukhamba Sanskrit Sansthan; 2001; 66.
2. Dahanukar SA, Thatte UM:'The rulers:Doshas' Chapter 3 in Ayurveda Unraveled.. 1st edition. 1996; 13-25.
3. Viman S, Charak S (Eds): '8th Adhyaya' 5th edition. Varanasi: Chaukhamba Sanskrit Sansthan; 2001; 277.
4. Murthy KRS. Sushruta Samhita, Sharira Sthana. chapter 4. Varanasi, India: Chaukhambha Orientalia; 2008. (Jaikrishnadas Ayurveda Series No.102).
5. http://roshanbaskota.com.np, September 21, 2011, Prakriti (Personal Nature) in Ayurveda, Roshan Baskota.
6. Gurunatham T, Chandrasekaran PV, Usha SP and Sarangan R, preliminary study of the assessment of Prakriti. JRIM (Journal Of Research in Indian Medicine) 1967; 2 (1): 105-112.
7. Svoboda RE: Constitutional characteristics'; Chapter 2 in Prakriti: Your Ayurvedic constitution. 1st edition. 1996; 49-54.
8. Bhalerao SS, Pawse P: Chapter 2 "Concept of Prakriti.Dr. Sharadini Dahanukar-Ayurveda Centre for Research, Training and Services 1st edition. 1996; 15.
9. Dr. Mrs. Purandare v r correlation between abo blood groups and tridoshas (ayurvedic biological constituents) International journal of ayurvedic & herbal medicine, 2013; 3(1): 1053-1056.
10. Supriya Bhalerao, Tejashree Deshpande Prakriti (Ayurvedic concept of constitution) and variations in platelet aggregation, Evid Based Complement Alternat Med, Dec.2012.

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