academicJournals

Vol. 7(4), pp. 67-73, May 2015
DOI: 10.5897/JPVB2014.0180
Article No. : 16CA05E52220
ISSN 2141-2510

http://www.academicjournals.org/JPVB

Journal of Parasitology and Vector Biology

Full Length Research Paper

A study on correlation of malaria infection with A, B, O, RH blood group system

G. Singh*, A. D. Urhekar and R. Singh

Department of Microbiology, Mahatma Gandhi Memorial Medical College and Hospital, Mahatma Gandhi Memorial Institute of Health Sciences (Deemed University), Sector-1, Kamothe, Navi Mumbai-410209, Maharashtra, India.

Received 26 November, 2014; Accepted 10 April, 2015

The aim of this study was to find correlation of malaria infection with A, B, O and Rh blood grouping. A total of 200 blood samples were collected from suspected malaria cases out of which 40 samples were positive and 160 samples were negative for malaria. ABO and Rh blood grouping was done using Eryscreen®. Maximum numbers of malaria positive were seen in blood group 'O' positive followed by 'B' positive, 'A' positive and 'AB' positive. This study suggests that the person having blood group 'O' positive are more prone to malarial infection in endemic areas.

Key words: ABO and Rh blood group, malarial parasites, malaria, microscopy.

INTRODUCTION

Malaria is caused by an obligate, intracellular protozoan parasite of the genus *Plasmodium*. Of the four species that infect humans (*Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale* and *Plasmodium malariae*), *P. falciparum* is responsible for high mortality (Pathirana et al., 2005). The virulence of *P. falciparum* has been associated with the capacity of the infected red blood cells (RBCs) to adhere to uninfected RBCs, leading to rosetting of cells (Carlson et al., 1990; Ringwald et al., 1993). Previous studies have implicated the ABO blood group type in rosetting (Thakur and Verma, 1992). Blood group antigens A and B are trisaccharides attached to a variety of glycoproteins and glycolipids on the surface of erythrocytes, and these trisaccharides are thought to act as receptors for rosetting on uninfected erythrocytes and bind to parasite rosetting ligands such as PfEMP-1 and sequestrin (Martin et al., 1979; Ockenhouse et al., 1992). However, blood group antigens A and B are not expressed in blood group O individuals. As a result, rosettes formed by blood group O are suggested to be smaller and easily disrupted than rosettes formed by blood group A, B or AB erythrocytes (Daniel, 2005; Barragan et al., 2000).

The association of genetic markers with malaria has been the subject of numerous investigations, since the protection afforded by sickle-cell hemoglobin against infection by *falciparum* malaria parasite. A broad range of available evidence suggests that the origin, distribution and relative proportion of ABO blood groups in humans may have been directly influenced by selective genetic pressure from *Plasmodium falciparum* infection (Christine et al., 2007). Clinical reports of ABO blood groups and *P. falciparum* infection, reveals a correlation between disease severity and ABO groups. However, several studies undertaken have been unable to link ABO blood groups to the incidence of malaria or to the repeat attacks

*Corresponding author. E-mail: gurjeetsingh360@gmail.com

of malaria (Facer and Brown, 1979; Singh et al., 1995). Recent studies of the pathogenesis of malaria have shown that parasite triggered red blood cell rosette formation is associated with the severity of clinical disease and malaria (Treutiger et al., 1992; Pathirana et al., 2005). Rosetting was established as a *P. falciparum* virulence factor, the expression of which is modified by a variety of host factors. Anti-rosetting activity, presumably mediated by antibodies, was found in sera from patients in malaria endemic areas, and it was demonstrated that such activity was more abundant in individuals with uncomplicated malaria than in those with cerebral disease, suggesting that humoral immunity protects against rosette formation in vivo. Erythrocytes from individuals with sickle-cell trait, α-and ß-thalassemia trait or with HbE formed smaller and weaker rosettes than normal HbAA red blood cells. Recently, even *P. vivax* infection has been reported with clinical severity. There is a paucity of hospital- based, comparative studies to investigate the relationship between blood group types and severity of malarial infections (Deepa et al., 2011).

The ABO blood groups consist of A, B and H carbohydrate antigens which can regulate protein activities during infection and antibodies against these antigens. A number of studies were conducted to investigate the association between ABO blood group system and some disease conditions. 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 (Tewodros et al., 2011).

Aims and objectives

1. To assess the distribution of ABO blood group and their relationship with *P. falciparum* and *P. vivax* malaria among patients attending a tertiary care hospital, Navi Mumbai.
2. To correlate the blood groups and the clinical presentations including outcome in malaria patients.

MATERIALS AND METHODS

Ethical clearance

The study protocol was reviewed and approved by the Ethical Review Committee of Mahatma Gandhi Mission Institute of Health Sciences (Deemed University), Navi Mumbai. Written informed consent was obtained from all study participants and mothers/caretakers of children under 18 who participated in the study after explaining the purpose and objective of the study.

Place of study

The study was conducted in Department of Microbiology, Mahatma Gandhi Mission Medical College and Hospital, Navi Mumbai, Maharashtra, India.

Study area

The study area is highly endemic for malarial infection. During rainy season from July to October the incidence of the disease is much more and people visit the hospital in maximum numbers. Mahatma Gandhi Mission's (MGM) Medical College, Navi Mumbai is having 900 bedded attach tertiary hospital serving both as the tertiary care hospital as well as the MGM Medical College teaching hospital. It is located in the MGM Campus, Plot No. 1 and 2, NH-4 Junction, Mumbai-Pune Express Way, Sector-1, Kamothe, Navi Mumbai, Maharashtra, India. Navi Mumbai located in the eastern trans harbour in Mumbai, Maharashtra, India, which is endemic to malaria. This region has rainy seasons from June to October. However, urban and suburban areas of Navi Mumbai city have several swampy and unpopular slum areas with poor drainage and waste disposal facilities. These in turn provide fertile breeding grounds for the female *Anopheles* mosquito, the vector for *Plasmodium* species, leading to the burden of endemic malaria.

Study participants

A total of 200 blood samples of malaria suspected patients of outpatient department and inpatient department of medicine, tertiary care hospital at Navi Mumbai, India were included in this study.

Parasite density determination

Thick and thin blood film slides were prepared using Jaswant-Singh-Bhattacharji stain and Leishman's stain. The stained slides were examined under a light microscope using 100× oil immersions lens. Parasitaemia was calculated per 200 white blood cells (WBC) assuming 8000 WBC/µl of blood (Cheesebrough, 1998).

Determination of blood grouping

Blood group determination ABO blood groups were typed by agglutination using Eryscreen® (Anti-A, Anti-B and Anti-D) provided by Tulip Diagnostics (P) Ltd., India. Three drops of whole blood were placed in two different places of a grease-free clean glass slide on which a drop of antisera for blood group A, B and Rh was added. The blood cells and the antigen were mixed with applicator stick. The slide was then rotated to detect for agglutination and the result recorded accordingly (Barragan et al., 2000; Zoysa, 1985).

RESULTS AND DISCUSSION

This prospective and analytical study was conducted to find any correlation of malaria with blood group A, B, O and Rh. A total of 200 samples were included in this study out of which 40 (20%) samples were malaria positive and 160 (80%) samples were malaria negative. Malaria was confirmed by 3 methods light microscopy, Quantitative buffy coat (QBC) test and rapid malarial antigen test. Out of the 38 to 40 samples, 25 (62.50%) samples were *P. vivax,* 10 (25%) (13.16%) samples were *P. falciparum* and 5 (12.50%) samples were mixed species. There was blood grouping for all samples that is, positive as well as negative. Blood group distribution in malaria suspected patients was A positive 56 (28%), B positive 41 (20.5%), AB positive 23 (11.5%), O positive

Table 1. Showing distribution of blood groups in 200 malaria suspected cases.

Blood groups	Distribution n=200 (%)
A positive	56 (28)
B positive	41 (20.5)
AB positive	23 (11.5)
O positive	58 (29)
A negative	8 (4)
B negative	4 (2)
AB negative	2 (1)
O negative	8 (4)

Table 2. Showing malaria and blood groups.

Blood groups	Total no. of samples	Malaria positive	Percentages (%)
A positive	56	9	16.08
B positive	41	9	21.95
AB positive	23	3	13.04
O positive	58	15	25.86
A negative	8	1	12.5
B negative	4	0	0
AB negative	2	0	0
O negative	8	1	12.5

58 (29%), A negative 8 (4%), B negative 4 (2%), AB negative 2 (1%) and O negative 8 (4%). (Table 1).

The study suggests that *P. falciparum* malaria patients with blood group O, which is less prone to rosetting have a reduced chance of developing severe falciparum malaria as compared to patients with other blood groups (Zinaye and Beyene, 2010). 'O' group had an advantage over other groups (Deepa et al., 2011). The chance of having a *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, blood groups A, B and AB are more susceptible to *P. falciparum* infection as compared with individuals of blood group O (Zerihun et al., 2011). Parasitaemia seemed to be relatively high across all blood groups with groups O and AB subjects apparently recording the highest and least infection rates (Otajevwo, 2013). Patients with O blood group occur most in the clinic, but the prevalence of malaria was highest among those with B blood group (35.3%) and lowest in those with O blood group (17.7%) (Sule et al., 2014). A study reported that individuals of blood group A and B are more susceptible to malaria infection as compared with individuals of blood group O, however, the infection differs due to differential host susceptibility (Gayathri et al., 2013). The respective infective rates were 14.3, 11.1, 13.9 and 0.00% of the blood groups A, B, O and AB. The difference in infection percentage between the various blood groups was, however, not statistically significant (Muntaka and Opok, 2013) (Table 3). This study showed that maximum numbers of malaria cases were found in blood group 'O' positive that is, 25.86%, followed by B positive that is, 21.95%, A positive that is, 16.08% and AB positive that is, 13.04% (Table 2).

Maximum number of malaria suspected cases was seen in blood group 'O' positive in which 25.86% malaria was positive and 74.14% malaria was negative, followed by blood group 'B' positive in which 21.95% malaria was positive and 78.05% malaria was negative, A' positive in which 16.07% malaria was positive and 83.93% malaria was negative, blood group 'AB' positive in which 13.04% malaria was positive and 86.96% malaria was negative, and blood group 'A' negative in which 12.50% malaria was positive and 87.50% malaria was negative and blood group 'O' negative in which 12.50% malaria was positive and 87.50% malaria was negative (Table 2). In *P. vivax*, maximum number of malaria were seen in blood group 'O' positive that is, 40% followed by 'A' and 'B' positive that is, 22 and 20% respectively, 'A' negative and 'O' negative 4% each, however, no cases was seen in 'B' negative and 'AB' negative (Figure 1). In *P. falciparum,* maximum number of malaria were seen in blood group 'O' positive and 'A' positive that is, 40% each followed by 'B' and 'AB' positive, that is, 10% each, however no case was seen in 'A' negative, 'B' negative, 'AB' negative and 'O' negative (Figure 2).

Table 3. Showing correlation of prevalence of malaria in different blood group.

Workers	A (%)	B (%)	O (%)	AB (%)
Our study	16.08	21.95	25.86	13.04
Gayatri et al. (2013)	16.09	40.9	34.16	8.78
Deepa et al. (2011)	22	42	35	1
FD Olajevwo et al. (2013)	34.6	23.1	38.4	3.9
Sule Hussain et al. (2014)	32.3	35.3	17.7	24.2
Sing et al. (1995)	14.3	11.1	13.9	0
Tewodros et al. (2011)	23.5	21.9	51.3	3.3

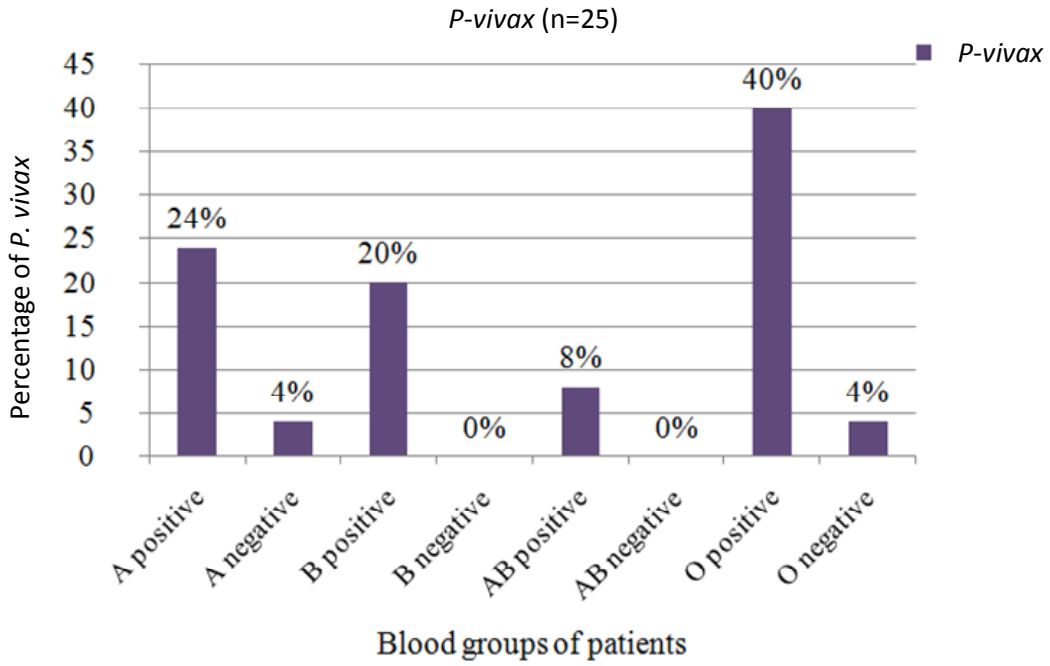

Figure 1. Showing distribution of blood groups in *P. vivax* infection.

In mixed species, maximum number of malaria were seen in blood group 'O' positive, that 40% followed by 'A' positive 'B' and 'AB' positive that is, 20% each, however no case was seen in 'A' negative, 'B' negative, 'AB' negative and 'O' negative (Figure 3). Malaria cases were found mostly in O positive blood group patients (25.86%), followed by B positive (21.95%), A positive (16.08%) and AB positive (13.04%) (Table 2). As regards correlation, there are some differences from other reports. High incidence of malaria in O positive blood group (25.86%) was found in the study of Olajevwo et al. (2013), Tewodros et al. (2011) also reported higher incidence in O blood group 38.4 and 51.3% respectively. Gayatri et al. (2013) and Deepa et al. (2011), however reported higher incidence in B blood group 40.97 and 42% respectively. Less incidence of malaria was reported in blood group A and AB by most workers. Similarly, less incidence was found in A Rh negative and O Rh negative blood group (12.5% each). Malaria was not found in B Rh negative and AB Rh negative. However, this number is small for any conclusion. Blood groups 'A' positive, 'B' positive, 'AB' positive, 'O' positive was statistically significant. (Table 4).

Regarding blood group and species distribution, it was observed that maximum incidence of *P. vivax, P. falciparum* and mixed species was in blood group O positive 40, 40 and 40% respectively (Table 5). Possible explanation for higher prevalence of malaria infection by earlier mentioned species, could be that there are no blood group antigens on the surface of O group red cells, and hence more number of receptors and chances of attachment of malarial parasites, where in blood group A, B and AB, the red cells are covered with respective blood group antigens and there is less number of receptor for malarial parasites and less chances for attachment of malaria parasite to these red cells.

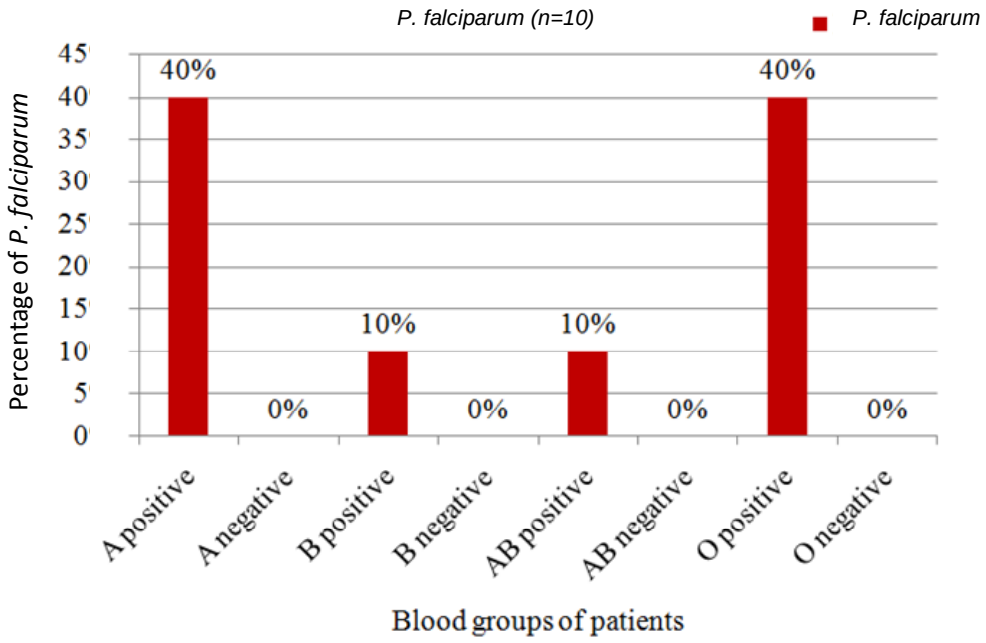

Figure 2. Showing distribution of blood groups in *P. falciparum* infection.

Table 4. Showing significant difference between Rh positive and Rh negative blood groups.

Blood groups	Negative	Positive	P value
A Positive	56	9	< 0.05*
A Negative	8	1	> 0.05
B Positive	41	9	< 0.05*
B Negative	4	0	> 0.05
AB Positive	23	3	< 0.05*
AB Negative	2	0	> 0.05
O Positive	58	15	< 0.05*
O Negative	8	1	< 0.05*

*Statistically significant (P value < 0.05).

Table 5. Showing blood group and overall malaria prevalence.

Parameters	Rh Positive				Rh Negative			
Malarial parasites	**A (%)**	**B (%)**	**AB (%)**	**O (%)**	**A (%)**	**B (%)**	**AB (%)**	**O (%)**
P. vivax (n=25)	24	20	8	40	4	0	0	4
P. falciparum (n=10)	40	10	10	40	0	0	0	0
Mixed species (n=5)	20	20	20	40	0	0	0	0

Conclusion

This study, found out that ABO and Rh blood groups of human beings may show differences in susceptibility to malarial infection, the total blood samples were 200 included in this study, out of which 40 samples were positive for malaria and 160 was negative. The A, B, O and Rh blood group was done using Eryscreen® of both positive as well as negative malaria cases. Maximum numbers of malaria positive cases were seen in blood group 'O' positive followed by 'A' 'B' positive 'A' and 'AB' positive. This study suggests that the person having

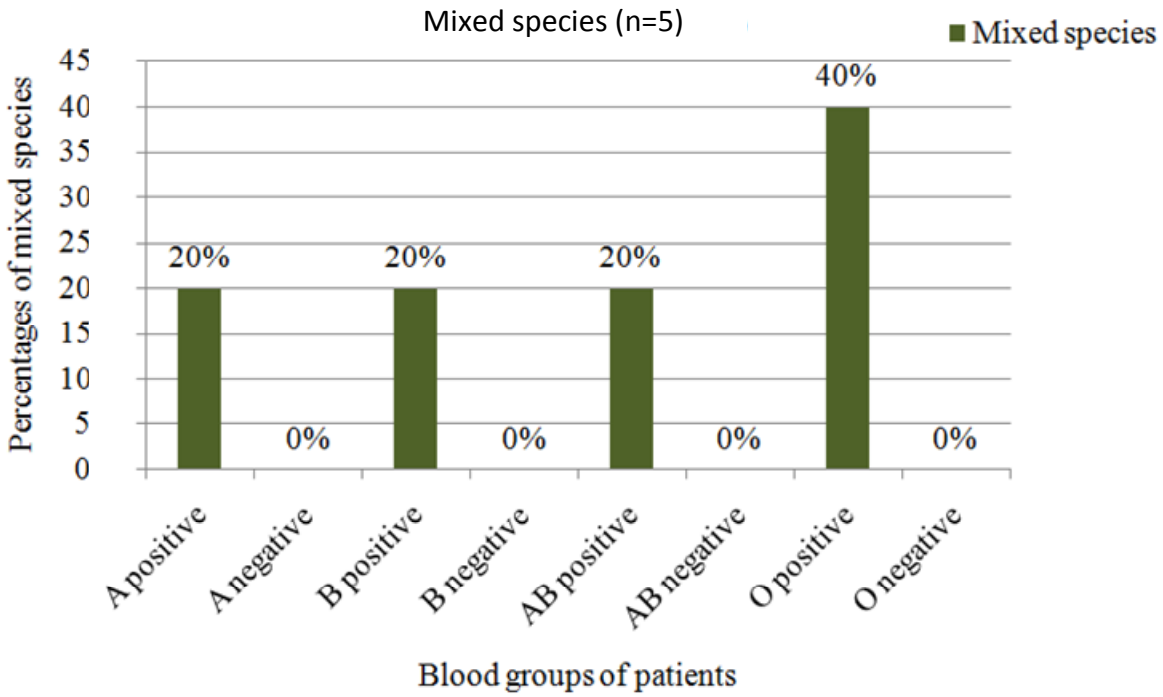

Figure 3. Showing distribution of blood groups in mixed infection.

blood group 'O' are more prone to malarial infection in endemic areas.

Conflict of Interest

The authors have not declared any conflict of interest.

REFERENCES

Pathirana SL, Alles HK, Bandara S, Phone KM, Perera MK, Wickremasinghe AR, Mendis KN, Handunnetti SM (2005). ABO-blood-group types and protection against severe, *Plasmodium falciparum* malaria. Ann. Trop. Med. Parasitol. 99:119-124.

Carlson J, Helmby H, Hill AV, Brewster D, Greenwood BM, Wahlgren M (1990). Human cerebral malaria: association with erythrocyte rosetting and lack of anti-rosetting antibodies. Lancet. 336:1457-1460.

Ringwald P, Peyron F, Lepers JP, Rabarison P, Rakotomalala C, Razanamparany M, Rabodonirina M, Roux J, Le BJ (1993). Parasite virulence factors during falciparum malaria: rosetting, cytoadherence, and modulation of cytoadherence by cytokines. Infect. Immun. 61:5198-5204.

Thakur A, Verma IC (1992). Malaria and ABO blood groups. Indian J. Malariol. 29:241-244.

Martin SK, Miller LH, Hicks CU, David WA, Ugbode C, Deane M (1979). Frequency of blood group antigens in Nigerian children with falciparum malaria. Trans. R. Soc. Trop. Med. Hyg. 73:216-218.

Ockenhouse CF, Tegoshi T, Maeno Y, Benjamin C, Ho M, Kan K E, Thway Y, Win K, Aikawa M, Lobb RR (1992). Human vascular endothelial cell adhesion receptors for Plasmodium falciparum-infected erythrocytes: roles for endothelial leukocyte adhesion molecule 1 and vascular cell adhesion molecule 1. J. Exp. Med. 176:1183-1189.

Daniel G (2005). The molecular genetics of blood group polymorphism. Transpl. Immunol. 14:143-153.

Barragan A, Kremsner PG, Wahlgren M, Carlson J (2000). Blood group A antigen is a coreceptor in Plasmodium falciparum rosetting. Infect. Immun. 68:2971-2975.

WHO (2002). Management of severe malaria Practical Hand Book, 2.

Cheesebrough M (1998). District laboratory practice in tropical countries Cambridge: Cambridge University press.

Zoysa D (1985). The distribution of ABO and Rhesus (Rh) blood groups in Sri Lanka. Ceylon Med. J. 30:37-41.

Zinaye T, Beyene P (2010). The ABO blood group and Plasmodium falciparum malaria in Awash, Metehara and Ziway areas, Ethiopia. Malar. J. 9:280.

Cserti CM, Dzik WH (2007). The ABO blood group system and *Plasmodium falciparum* malaria. Blood 110(7):2250–2258.

Facer CA, Brown J (1979). ABO blood groups and *falciparum* malaria. Trans. R. Soc. Trop. Med. Hyg. 73:599–600.

Singh N, Shukla MM, Uniyal VP, Sharma VP (1995). ABO blood groups among malaria cases from District Mandla, Madhya Pradesh. Indian J. Malariol. 32:59–63.

Treutiger CJ, Hedlund I, Helmby H, Carlson J, Jepson A, Twamasi P, Kwiatkowski D, Greenwood BM, Wahlgren M (1992). Rosette formation in *Plasmodium falciparum* isolates and antirosette activity of sera from Gambian with cerebral or uncomplicated malaria. Am. J. Trop. Med. Hyg. 46:503-510.

Pathirana SL, Alles HK, Bandara S, Phone-kyaw M, Perera MK, Wickremasinghe AR, Mendis KN, Handunnetti SM (2005). ABO blood group types and protection against severe *Plasmodium falciparum* malaria. Ann. Trop. Med. Parasitolol. 99:119-26.

Deepa, Vanamala A. Alwar, Karuna Ramesh kumar, Cecil Ross. ABO blood groups and malaria related clinical outcome. J. Vector Borne Dis. 48(1):7-11.

ABO blood group system. (2014). http://www.britannica.com/EBchecked/topic/1429/ABO-blood-group-system. ABO blood group system. http://en.wikipedia.org/wiki/ABO_blood_group_system

Tewodros Z, Abraham D, Berhanu E (2011). Association of ABO blood group and Plasmodium falciparum malaria in Dore Bafeno Area, Southern Ethiopia. Asian Pac. J .Trop. Biomed. 1(4):289-94.

Otajevwo FD (2013). Prevalence of malaria parasitaemia and its association with ABO blood grouping among students of igbinedion

University Okada, Nigeria. Br. J. Med. Med. Res. 3(4):1164-1177.
Sule HA, Idachaba SO, Idoko T (2014). Susceptibility of Humans of The ABO Blood Groups *to P. falciparum* infection Among Patients Attending Ahmadu Bello University Clinic (Sickbay), Samaru-Zaria, Kaduna State, Nigeria. Sch. J. App. Med. Sci. 2(4C):1305-1309.
Gayathri BN, Harendra KML, Gomathi N, Jeevan S, Reethesh RP (2013). Relationship between ABO blood groups and malaria with clinical outcome in rural area of South India. Glob. J. Med. Public Health 2(2):1-7.