Duffy Blood Group and Thalassemia Major Patients in Relation to Post Transfusion Malaria

N. Choudhury
J.G. Jolly
M.L. Dubey
R.C. Mahajan
N.K. Ganguly
S.K. Agnihotri

Thalassemias are a group of inherited disorders in which there are decreased production of one or more of the polypeptide chains of hemoglobin resulting in the production of erythrocytes which are microcytic and hypochromic(1). Due to repeated blood transfusions, patients are exposed to an increased threat of transmissible diseases like malaria(2). A high incidence of post transfusion malaria (PTM) in thalassemia patients is due to fresh blood and the high frequency of blood transfusions(3). Duffy blood group antigens on red cells and thalassemia disorder, both are genetically inherited. An attempt was made to find out whether the Duffy blood group system had any influence on the high incidence of PTM in thalassemia patients.

From the Department of Transfusion Medicine, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Post Box 375, Lucknow; and Departments of Parasitology, Experimental Medicine and Blood Transfusion, Post Graduate Institute of Medical Education and Research, Chandigarh 160 012.

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Material and Methods

A group of thalassemia major patients who visited the Blood Transfusion Department of Post Graduate Institute of Medical Education and Research, Chandigarh for transfusion therapy were studied for 16 months. They were screened for malaria infection by Giemsa(4) and Acridine Orange (AO) fluorescence staining(5). They were also tested for the presence of malaria antigen by commercially available monoclonal antibody (MAB), *i.e.*, Monofluo kit–*P. falciparum*(6). Duffy phenotyping was done in a group of randomly picked up thalassemia patients and in a group of blood donors as a control population(7). Chi-square test was applied to find out the statistical significance.

Results

A total of 125 thalassemia major patients were screened regularly during the study period and 8 (6.4%) patients were found to be positive for PTM. The diagnosis of PTM was established by following up blood donors retrospectively and testing with the help of smear examinations and/or antigen detection test. Among 8 PTM positive patients, 2 were suffering from *P. falciparum* infections and 4 had primary infection of *P. vivax* and in two patients species could not be identified (*Table I*). Duffy phenotyping was done in 42 patients as well as in 40 blood donors. The incidence of Duffy positive phenotype, *i.e.*, Fy (a^+ b^+), Fy (a^- b^+) and Fy (a^+ b^-) was found in 100% of patients and in 97.5% of healthy population (*Table II*). Among 42 thalassaemia patients, there were 4 PTM positive cases who were infected with *P. vivax*. With chi square test, the correlation between the incidence of Duffy positive

TABLE I—*Transfusion Induced P. Vivax Infection in Thalassemia Patients*

Patient No.	Giemsa	Acridine orange	Antigen detection	Remarks
1.	+ve	NA	NA*	*P. vivax*, treated outside
2.	+ve	+ve	+ve	Mixed infection with *P. vivax* and *P. falciparum*
3.	+ve	+ve	+ve	*P. vivax*
4.	+ve	+ve	+ve	*P. vivax*

*NA = Not available.

TABLE II— *Duffy Phenotypes in Thalassemia Patients and Blood Donors*

Duffy phenotype	Thalassemia patient (%)	Blood donors (%)
Fy (a^+ b^+)	36 (85.7)	26 (65.0)
Fy (a^+ b–)	6 (14.3)	12 (30.0)
Fy (a– b^+)	–	1 (2.5)
Fy (a–b–)	–	1 (2.5)
Total	42	40

phenotype and PTM positive (*P. vivax*) thalassemia patients in comparison to healthy population was insignificant ($p<0.5$).

Discussion

Blood transfusion is the conventional life supporting therapy for thalassemia major patients. Due to genetically inherited red cell disorder, these patients need repeated blood transfusions(3). Due to malaria endemicity of India, the patients who receive blood transfusion are facing the great danger of malaria transmission(8). But the viability of malaria parasites depends on that of their erythrocyte hosts(4). It is already established that *P. vivax* (*P. knowlessi*) gets entry to the red cells through Duffy positive antigens on the cell surface(9). The asexual form of malaria parasite which enters into the red cells continue to propagate with their own periodicities and results into attacks of clinical malaria(10). Thus, Duffy positive individuals in a malaria endemic population face higher chances of malaria (*P. vivax*) infection, if transmitted through blood.

In this study, 97.5% of healthy population and 100% of the thalassemia patients showed Duffy positive phenotype, including four PTM positive (*P. vivax*) cases. However, malaria infection was not found in blood donors. The occurrence of Duffy positive blood group and malaria infection in thalassemia patients was insignificant. There is a general relation between some genetic marker and disease susceptibility(11). Miller and Miller tried to find out the correlation between two genetically inherited factors, *i.e.,* Duffy phenotype and hypertension as well as Duffy phenotye and cancer in black population but no significant association was observed(12,13).

In this study, a correlation between the Duffy positive phenotype and the high incidence PTM was observed in thalassemia patients which was not significant. An elaborate study is needed to draw a concrete conclusion in relation to Duffy blood group system among thalassemia patients and their influence on malaria infection.

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REFERENCES

1. Brook CG, Thompson EN, Marshal WC. Growth in children and thalassemia major and effect of two different transfusion regimens. Arch Dis Child 1969, 44: 451-453.
2. Greenberg AE, Dinh PN, Mann JN. The association between malaria, blood transfusion and HIV seropositivity in a pediatric population in Kinshasa, Zaire, JAMA 1988, 259: 545-549.
3. Choudhury N, Dubey ML, Jolly JG, Kalra A, Mahajan RC, Ganguly NK. Post transfusion malaria in thalassemia patients. Blut 1990; 61: 314-316.
4. Bruce Chwatt LJ. Essential Malariology 2nd edn. London, William Heinemann, 1985, p 68.
5. Hare JD, Bahler DW. Analysis of *Plasmodium falciparum* in culture using Acridine Orange and flow cytometry. J Histochem Cytochem 1986, 34: 215-220.
6. Choudhury N, Jolly JG, Mahajan RC, *et al*. Selection of blood donors in malaria endemic countries. Lancet 1988, 11: 972-973.
7. Walker RH. Technical Manual, 10th edn. Virginia, American Association of Blood Banks, 1990, pp 236-237.
8. Bruce Chwatt LJ. International forum: Which are the appropriate modifications of existing regulation designed to prevent the transmission of malaria by blood transfusion, in view of the increasing frequency of travel to endemic areas? Vox Sang 1987, 52: 140-143.
9. Hadley TJ, Klotz FW, Miller LH. Invasion of erythrocytes by malaria parasites: A cellular and molecular overview. Ann Rev Microbiol 1986, 40: 451-452.
10. Manson Bahr PEC, Apted FIC. Manson's Tropical Diseases, 18th edn. London, Baillaire Tindall, 1982, pp 38-69.
11. Mollison PL. Blood Transfusion in Clinical Practice, 8th edn. Oxford, Blackwell Scientific, 1987, p 205.
12. Miller JM, Miller JM. Duffy and hypertension in black population. Am J Public Health 1985, 75: 558-559.
13. Miller JM, Miller JM. Duffy antigen and cancer in black population. Am J Surg 1983, 146: 639-640.