Original article

Detection of G6PD deficiency amongst healthy blood donors

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Abstract

Introduction: G6PD deficiency is the most common erythrocyte enzymopathy, being present in more than 400 million people worldwide. Blood donation from G6PD deficient donors might alter the quality of the donated blood during processing, storage or in the recipient circulatory system.

Aims & objectives: 1.To detect G6PD deficiency amongst healthy blood donors. 2.To find out the prevalence of G6PD deficiency.

Material & methods: A prospective study was carried out on 2012 healthy blood donors from February 2016 to December 2016 in the Department of IHBT, S.P. Medical College and Associated group of Hospitals, Bikaner. Blood donors were screened for G6PD deficiency using Methemoglobin Reduction Test.

Observations & results: Out of total 2012 healthy blood donors, 133(6.6%) donors were found to be G6PD deficient. Prevalence rate observed in the study was within the range of overall prevalence of India (0-27%). **Conclusion:** Prevalence of 6.6% should be taken as serious concern and further more studies are advised for screening of G6PD in healthy blood donors. As the methaemoglobin reduction test (MRT) used in this study is cost effective and convenient, it can be used as a screening method to identify G6PD deficient blood donors and it can be an aid-on extended medical checkup for healthy blood donors which will help to increase the safe and voluntary blood donations.

Keywords: G6PD deficiency, Methemoglobin Reduction Test(MRT), Blood donors

Introduction:

A blood bank plays an important role in ensuring the supply of safe blood as and when required. While it is important to ensure that there is an adequate supply of blood, it is also essential that the blood collection process does not harm either the blood donor or the recipient. This is achieved by doing stringent screening of pre and post donations for possible Transfusion Transmissible Infections (TTIs.) and other investigations.

G6PD is a cytoplasmic enzyme. It has a housekeeping role in all cells, and is particularly critical to the integrity and functioning of RBCs. The major function of G6PD is the prevention of oxidative damage to cells by promoting detoxification of free radicals[1]. The G6PD gene is present on the long arm of the X chromosome (Xq28) and consists of 13 exons with a length of 18 kb. The active form of G6PD enzyme is either a dimer or a tetramer of a single polypeptide subunit of about 59 kD[12]. Association of these subunits was NADP dependent[2,3].

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common erythrocyte enzymopathy, being present in more than 400 million people worldwide that may lead to neonatal jaundice or hemolytic crisis due to drugs or infections[4]. The study of G6PD deficiency among blood donors is important as WHO recommends deferring G6PD deficient blood donors with a history of hemolysis[5].

In India, G6PD deficiency was reported in 1963 by

Baxi et al, and the prevalence rate varied from 0 to 27% in different caste, ethnic, and linguistic groups. The frequency is higher among the tribals than the caste populations[6].

Early detection and prevention is the key strategy to successful management and control of G6PD deficiency. Genetic counseling, prenatal diagnosis, health education, and public awareness can provide benefits by way of preventive genetics to the affected individuals and their families[6].

Aims & Objectives:

1. Detection of Glucose-6-Phosphate dehydrogenase deficiency in healthy blood donors.
2. To study the prevalence of Glucose-6-phosphate dehydrogenase deficiency in healthy blood donors.

Material & Methods:

A total of 2012 samples from blood donors coming in the Department of IHBT, S.P. Medical College and Associated group of Hospital, Bikaner, were collected for screening of G6PD deficiency during a time period of 11 months from February 2016 to December 2016. Written consent was taken at the time of donor screening.

The method (Methaemoglobin Reduction Test/MRT) used in the this study was same as described in the WHO guidelines[7].

Principle of MRT: It involves the oxidation of haemoglobin to methaemoglobin by sodium nitrite and its subsequent enzymatic reconversion to haemoglobin in the presence of methylene blue. This redox dye affects the pentose phosphate pathway and activates reduced triphosphopyridine nucleotide-methaemoglobin reductase in normal, but not in G6PD deficient, erythrocytes. The rate of reduction of methaemoglobin to haemoglobin is proportional to the G6PD activity of the cell.

Procedure: 2 ml of the blood to be tested was added to the sample tube. 2 ml of blood were added to both the positive reference and normal reference tubes. Only one positive and one normal reference tubes are needed for each batch of tests performed. They were mixed well by inversion. Samples were incubated at 37° ± 1°C for three hours. After incubation, 0.1 ml of the test mixture was added to test tube containing 10 ml of distilled water. After 2 to 10 minutes, it was visually compared with the similarly diluted positive and normal references.

Interpretation: Normal G6PD status - Clear red, like the normal reference tube. G6PD deficiency - Brown, like the positive reference tube.

Observations & results:

It was observed that 133(6.6%) blood donors were G6PD deficient among 2012 healthy blood donors who came to donate blood in the Department of Immunohematology and Transfusion Medicine, S.P. Medical College and Associated Group of Hospitals, Bikaner. The prevalence rate observed in this study was 6.6%.

Table 1 shows the status of G6PD according to sex. All 133 G6PD deficient blood donors were male blood donors. All the female donors had normal G6PD status.

Table 2 shows the status of G6PD according to age group. Out of total 133 blood donors who had G6PD deficiency, maximum 75(56.4%) blood donors belonged to age group 21-30. Most common age group was 21-30 years where total 1137 blood donors were found.

Table 3 shows the status of G6PD according to ABO blood group. The most common ABO blood group in the study population was B blood group in both G6PD deficient(57, 42.8%) and G6PD normal(749, 39.8%) blood donors.

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Table 4 shows the status of G6PD according to Rhesus D blood group. Out of total 2012 blood donors, 1823 blood donors were Rh Positive and 189 blood donors were Rh Negative. Among 133 G6PD deficient blood donors, 120(90.2%) blood donors were Rh D Positive and 13(9.8%) blood donors were Rh D Negative.

Table 1

Status of G6PD according to Sex

Sex	Status of G6PD				Total	
	G6PD deficiency		Normal G6PD			
	No.	%	No.	%	No.	%
Male	133	100	1860	99.0	1993	99.1
Female	0	0	19	1.0	19	0.9
Total	133	100	1879	100	2012	100

Table 2

Status of G6PD according to age group

Age Group	Status of G6PD				Total	
	G6PD deficiency		Normal G6PD			
	No.	%	No.	%	No.	%
18-20	11	8.3	134	7.1	145	7.2
21-30	75	56.4	1062	56.5	1137	56.5
31-40	36	27.1	492	26.2	528	26.3
41-50	10	7.5	169	9.0	179	8.9
51-60	1	0.7	22	1.2	23	1.1
Total	133	100	1879	100	2012	100

Table 3

Status of G6PD according to ABO blood group

Blood Group	Status of G6PD				Total		χ^2	p
	G6PD deficiency		Normal G6PD					
	No.	%	No.	%	No.	%		
A	32	24.1	390	20.8	422	21.0	0.8183	>0.05
B	57	42.8	749	39.8	806	40.0	0.4642	>0.05
O	38	28.6	548	29.2	586	29.1	0.0212	>0.05
AB	6	4.5	192	10.2	198	9.9	4.9507	<0.01
Total	133	100	1879	100	2012	100		

Table 4

Status of G6PD according to Rhesus D blood group

Blood Group	Status of G6PD				Total	
	G6PD deficiency		Normal G6PD			
	No.	%	No.	%	No.	%
Rh D Positive	120	90.2	1703	90.6	1823	90.6
Rh D Negative	13	9.8	176	9.4	189	9.4
Total	133	100	1879	100	2012	100
χ^2	0.0243					
p	>0.05					

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Table 5

Comparison of studies for prevalence of G6PD Deficiency

Study	Year	Country	n	Prevalence
White et al[8]	1986	Yemen	146	6.2%
Choubisa et al[9]	1987	Rajasthan, India	1198	4.59%
Pant et al[10]	1992	Gujrat, India	414	5.9%
Ramadevi et al[11]	1994	South India	5140	7.8%
Kaeda[12]	1995	Orissa, India	49	6.12%
Hilmi et al[13]	2002	Iraq	758	6.1%
Sukumar et al[14]	2004	Mumbai, India	3166	10.5%
Matsuoka et al[15]	2004	Cambodia	670	7.0%
Nishank[16]	2008	Orissa, India	3480	6.41%
Present study	2016	Rajasthan, India	2012	6.6%

Discussion:

This study showed that out of total 2012 healthy blood donors, 133(6.6%) blood donors had G6PD deficiency and 1879(93.4%) blood donors had normal G6PD.

Table 5 shows the comparison of studies for prevalence of G6PD Deficiency. The prevalence rate observed in the present study was very similar to White et al[8] (6.2%), Pant et al[10] (5.9%), Kaeda et al[12] (6.12%), Hilmi et al[13] (6.1%), Matsuoka et al[15] (7.0%) and Nishank et al[16] (6.41%).

The present study showed that majority of the G6PD deficient blood donors were in the age group 21-30 years where total 75 blood donors were found G6PD deficient. Overall most common age group was also 21-30 years where total 1138 blood donors were found. Second most common age group was 31-40 years where total 528 blood donors were found. Our study is similar to Sidhu et al[17] observed that majority of the blood donors belonged to age group 18-30 years. There, was apparently decreased frequency of G6PD deficient blood donors with increasing age. Omisakin et al[18] observed that out of the 314 blood donors 80(25.5%) were G6PD deficient. Age group of 25-34 years had highest G6PD deficient donors 34(42.5%).

Out of total 2012 blood donors, only 19(0.9%) blood donors were females while remaining 1993(99.1%) blood donors were males with male to female ratio was 104.89:1. Out of 1993 males 133(6.7%) were G6PD deficient and 1860(93.3%)

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blood donors had normal G6PD status. All the 19 females had normal G6PD level. Our study is very similar to Alabdulaali et al[19] found that 1137(98.9%) blood donors were males among the study population of 1150 blood donors. Only 13(1.1%) blood donors belonged to female blood donors. Out of 1150 blood donors 9(0.78%), 23(2%) and 4(0.35%) blood donors had G6PD deficiency, Sickle cell trait and both conditions, respectively. Out of 1137 male blood donors, 13(1.14%) blood donors were G6PD deficient. Akanni et al[20] observed that out of 200 blood donors tested for G6PD, 163(81.5%) blood donors were males and 37(18.5%) blood donors were females. Out of 163 male blood donors 35(21.5%) were G6PD deficient and out of 37 female blood donors 4(10.8%) were G6PD deficient.

In our study most common ABO blood group was B blood group which is similar to a study done by Pant et al[10] who observed distribution of ABO blood groups and sickle cell haemoglobin on 783 blood samples in relation to malaria, from both the sexes of Muslim and Christian populations of Kheda district, Gujrat. Blood group B was dominant in both the communities. Significant association of ABO polymorphs with P. falciparum and total malaria cases was observed. 414 blood samples from male individuals were screened for G6PD deficiency. High frequency of G6PD deficiency was observed in Christians (5.9%) and low in Muslim (1.8%) population, whereas sickle cell haemoglobin in Muslim population was 1.5% and absent in Christians. Our study is similar to Sidhu et al[17] observed that majority of the blood donors belonged to B blood group. Out of total 500 blood donors, 155(31%) blood donors belonged to blood group B followed by O, AB and A blood groups having 129(25.8%), 110(22%) and 106(21.2%), respectively. Out of 155 blood donors who were of blood group B, 2(1.29%) blood donors were G6PD deficient.

Our study found that out of total 2012 blood donors, 1823(90.6%) blood donors were Rh D Positive and 189(9.4%) blood donors were Rh D Negative. Our study is similar to Sidhu et al[17] observed that out of total 500 blood donors 445(89%) of blood donors were Rh D Positive and 55(11%) of blood donors were Rh D Negative. Out of 445 Rh D Positive blood donors, 4(0.9%) were G6PD deficient. No blood donor were found G6PD deficient among Rh D Negative groups. Jeremiah et al[21] observed that out of 240 apparently healthy children, 219(91.3%) were Rh D positive and 21(8.7%) were Rh D negative. A significant association was found to exist between blood group O, Rh D negative and P. falciparum malaria. No association was found to exist between G6PD status and P. falciparum parasitaemia.

Conclusion:

The observation of 6.6% prevalence of G6PD deficient blood donors in the present study could be taken to consider that the problem of G6PD deficiency exists in the blood donors. It should be of concern as the G6PD deficiency remains obscure, there being no overt clinical manifestation.

As the study is conducted over a time period of eleven months from February 2016 to December 2016 involving 2012 blood donors, prevalence of 6.6% should be taken as serious concern and further more studies are advised for screening of G6PD in healthy blood donors.

As the methaemoglobin reduction test (MRT) used in this study is cost effective and convenient, it can be used as a screening method to identify G6PD deficient blood donors and it can be an aid-on extended medical checkup for healthy blood donors which will help to increase the safe and voluntary blood donations.

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