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Original Article

Prevalence of Glucose-6-Phosphate Dehydrogenase Deficiency among Sickle Cell Patients of Chhattisgarh Region

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Abstract

Background: About 50% of world population of sickle cell disease (SCD) is found in India, predominant among the tribal population of central India. Sickle cell (SC) hemoglobinopathy and glucose-6-phosphate dehydrogenase (G6PD) enzyme deficiency are important genetic and public health problems in central-eastern part of India. This study aims to determine the prevalence of G6PD deficiency in SCD patients.

Materials and Methods: The study was carried out prospectively over a period of 1 year from September 2010 to August 2011 in the Department of Pathology at our institute. The material for the present study consisted of 100 cases including 76 patients with SCD and 24 of control group. Solubility test for sickling followed by hemoglobin (Hb) electrophoresis and qualitative dye decolorization test for G6PD deficiency were performed on all the samples.

Results: A total of 100 patients were studied, 76 were of SCD including 38 patients each of SC trait (AS) and SC anemia (SS) and remaining 24 patients were of the control group. The majority of AS patients were females (69.4%) while the SS and control patients had almost equal number of males and females. The prevalence of G6PD deficiency was observed to be 5%, predominantly in males i.e. 4 (9.1%) with only one female (1.79%). The prevalence of G6PD deficiency in SC patients (5.26%) was almost similar to that of control group (4.17%).

Conclusion: G6PD deficiency should be looked for in all subjects with SC anemia. Individual with HbSS is uniquely unfit to tolerate increased hemolysis and when the two problems coexist, particular care should be exercised in the administration of drugs known to initiate hemolysis in patients with G6PD deficiency.

Key words: Central India, Drugs, Hemoglobin electrophoresis, Hemolysis, Qualitative, Solubility test

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) and sickle cell disease (SCD) are two inherited red blood cell (RBC) disorders which could be encountered in the same person due to the following features common to both these disorders:

1. More commonly seen in certain geographical areas and ethnic groups
2. In stable conditions, both these disorders do not alter the hemoglobin (Hb) levels, RBC count, and indices
3. Patients usually remain asymptomatic in both.

Hence, detailed clinical history and screening tests are necessary to detect theses RBC disorders.[1]

The interaction between SCD and G6PD has been studied in different populations. Many studies have suggested the higher incidence of G6PD deficiency in SCD patients as compared to general population. However, other studies do not confirm this association.[2]

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The prevalence of sickle Hb varies from 15% to 30%, affecting nearly 3 million people of Chhattisgarh.[3] Hence, we performed this study to know the association and prevalence of G6PD in SCD patients so that to judiciously administer drugs during the general treatment or episodes of crisis.

MATERIALS AND METHODS

The present study was carried out in the Department of Pathology at our institute. The study was spread over a period of 1 year from September 2010 to August 2011.

The material for the present study consisted of 100 cases including 76 patients with SCD and 24 of control group.

With all standard aseptic precautions, blood was collected from anti-cubital vein using 21 or 22-gauge needle from each individual, after obtaining informed and written consent. Blood was delivered into ethylene diamine tetraacetic acid pilot, and solubility test for sickling was performed on all the samples, followed by Hb electrophoresis. In the entire cases, qualitative dye decolorization test for G6PD deficiency had been performed adjusting for the Hb content of the patient.

RESULTS

Of the total 100 patients studied, 76 were of SCD including 38 patients each of AS and SS. The 24 control patients selected had anemia but found to be negative for solubility test and Hb electrophoresis, of which 11 cases were of malaria, 8 cases of neonatal jaundice, 2 of β-thalassemia, and 3 cases of miscellaneous group. Of the 100 patients, 5 were found to be G6PD deficient showing the prevalence rate of 5%. In the individual group studied the prevalence was found to be 2 (5.3%), 2 (5.3%), and 1 (4.2%) in control, AS and SS patients, respectively (Table 1).

Most of the patients of AS were in age range of 11-20 years and 21-30 years, whereas most patients of SS were in between 0 and 10 years of age. Of the control patients, majority were in between 0 and 10 years. Both the patients of AS with G6PD deficiency were in age range of 11-20 years while both the SS patients in 0-10 years and the only control patient was above 40 years of age (Table 2).

Of the AS patients, females were more than males while among the SS patients equal number of males and females were present. Both the SS patients and the one control patient with G6PD deficiency were males (100%) while of the AS patients with G6PD deficiency one each (50%) was male and female (Table 3).

DISCUSSION

In the present series of 100 patients studied, the prevalence of G6PD deficiency was observed to be 5% (Table 1), predominantly in males i.e., 4 (9.1%) with only one female (0.79%) (Table 3).

Different studies conducted in the past indicate that the incidence of G6PD deficiency in India ranges from <1% to 27.94% (Table 4). The prevalence rate observed in the present study were similar to those of Swaroop *et al.*,[5] Kalra *et al.*,[12] Da Costa *et al.*,[10] Meera Khan,[7] Choubisa *et al.*,[17] Reddy *et al.*,[23] and Pant *et al.*[20] Discrepancies found with other may have resulted from different techniques used. The G6PD enzyme deficiency in the present study was detected using dichlorophenol indophenol dye as described by Bernstein.[35]

Table 1: Distribution of patients with and without G6PD deficiency according to pattern on hemoglobin electrophoresis

Haemoglobin pattern	Patients without G6PD deficiency		Patients with G6PD deficiency	
	n	%	*n*	%
AA	22	22	2	5.3
AS	38	38	2	5.3
SS	38	38	1	4.2
Thalassemia (HbA2)	2	2	0	0
Total	100		5	

G6PD: Glucose-6-phosphate dehydrogenase

Table 2: Age-wise distribution of patients with and without G6PD deficiency

Age range	Patients of AS				Patients of SS				Controls			
	Without G6PD deficiency		With G6PD deficiency		Without G6PD deficiency		With G6PD deficiency		Without G6PD deficiency		With G6PD deficiency	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
0-10	3	8.3	-	-	14	42.1	2	100	9	39.1	-	-
11-20	13	36.1	2	100	9	25	-	-	4	17.4	-	-
21-30	12	33.3	-	-	8	22.2	-	-	5	21.7	-	-
31-40	4	11.1	-	-	4	11.1	-	-	3	13.0	-	-
41-onwards	4	11.1	-	-	1	2.8	-	-	2	8.7	1	100
Total	36	100	2	100	36	100	2	100	23	100	1	100

G6PD: Glucose-6-phosphate dehydrogenase

Table 3: Gender-wise distribution of patients with and without G6PD deficiency

Gender	Patients of AS				Patients of SS				Controls			
	Without G6PD deficiency		With G6PD deficiency		Without G6PD deficiency		With G6PD deficiency		Without G6PD deficiency		With G6PD deficiency	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Male	11	30.6	1	50	18	50.0	2	100	11	47.8	1	100
Female	25	69.4	1	50	18	50.0	-	-	12	52.2	-	-
Total	36	100	2	100	36	100	2	100	23	100	01	100

G6PD: Glucose-6-phosphate dehydrogenase

Table 4: The prevalence of G6PD deficiency in patients without sickle cell disease in various studies in India

Name of author	Year	Place	Total number of patients without SCD	Total number of G6PD deficient patients	
				n	%
Baxi *et al*.[4]	1961	Mumbai	110	15	13.64
Swaroop *et al*.[5]	1961	Calcutta	60	3	5
Baxi *et al*.[6]	1963	Mumbai	216	34	15.7
Meera Khan[7]	1964	Andhra Pradesh	443	21	4.7
Khanduja *et al*.[8]	1966	Delhi	352	10	2.76
Deshmukh and Sharma[9]	1968	Aurangabad	100	6	6
Da Costa *et al*.[10]	1969	Mumbai	1865	95	5.1
Inamdar and Pohowalla[11]	1969	Indore	1000	62	6.2
Kalra *et al*.[12]	1973	Agra	100	5	5
Agarwal *et al*.[13]	1975	Lucknow	2560	44	1.7
Sharma *et al*.[14]	1975	Rohtak	500	3	0.6
Praharaj *et al*.[15]	1977	Orissa	200	22	11
Anand *et al*.[16]	1981	Rajasthan	300	36	12
Choubisa *et al*.[17]	1987	Udaipur, Rajasthan	1198	55	4.59
Verma *et al*.[18]	1990	Ludhiana			3.9
Choubisa[19]	1991	Dungarpur, Rajasthan	2922	329	11.25
Pant *et al*.[20]	1992	Kheda, Gujarat	414	25	5.9
Thakur and Verma[21]	1992	Bastar	473	118	25
Ramadevi *et al*.[22]	1994	South India	5140	400	7.8
Reddy *et al*.[23]	1995	Central India			4.57
Kaeda *et al*.[24]	1995	Orissa tribes	677	81	12
		Madhya Pradesh Baiga	263	11	4.2
Joshi *et al*.[25]	2001	Surat, Gujarat	272	80	27.94
			113	11	9.73
Shankarkumar[26]	2003	Dhule, Maharashtra	146	13	9
Balgir *et al*.[27]	2004	Orissa			4.3-17.4
Sukumar *et al*.[28]	2004	Mumbai	3166	332	10.5
Gupte *et al*.[29]	2005	Surat, Gujarat			22
Pao *et al*.[30]	2005				2.00
Balgir[31]	2006	Orissa			4.3-17.4
Nishank *et al*.[32]	2008	Bhubaneshwar, Orissa	3480	223	6.4
Choubisa[33]	2009	Sirohi, Rajasthan	368	56	15.21
Shanthala Devi *et al*.[34]	2010	South and West India	2005	16	0.8
Present study	2012	Raipur, Chhattisgarh	100	5	5

G6PD: Glucose-6-phosphate dehydrogenase, SCD: Sickle cell disease

In the present study, the prevalence of G6PD deficiency in sickle cell (SC) patients (5.26%) was almost similar to that of control group (4.17%). Similar findings of equal prevalence of G6PD deficiency among SC and control group was observed in studies by Heller *et al*.,[43] Naylor and associates[36] in Chicago, observed 14.3% G6PD deficiency in 56 SS males, as compared to 15% of 54 AA Afro-American males; Bouanga *et al*.,[2] also found equal prevalence in HbSS patients (22.2%) and in HbAA (22.5%) (Table 5).

However, an increase in the prevalence of G6PD deficiency among SC patients as compared to control patients was observed in the studies by Lewis *et al*.[37] in Ghana; Beutler *et al*.,[39] Bienzle.[40] Piomelli *et al*.[38] (33.3% among SC patients and 10.8% among controls); Diop *et al*.[46] (21.6%

in SC patients and 12.3% in normal subjects); Andoka *et al.*[44] (25.8% of in AA, 31.8% in AS and 45.2% in SS); Mohammad *et al.*[45] (47% in HbS and 19% in HbA). Nouraie *et al.*[47] studied 261 children and adolescents with HbSS and found that the prevalence of G6PD to be 13.6% in males and 3.3% in females with an overall prevalence of 8.7%.

Gibbs *et al.*[42] found the prevalence of G6PD deficiency was higher in males and lesser in females as compared to the general population, but the differences were not significant.

On the contrary, Nhonoli *et al.*[41] observed higher prevalence of 19.5% in AA and 14.0% in AS but this difference was not found to be significant.

Praharaj *et al.*[15] found 11% patients to be G6PD deficient, of which 10 were of SC anemia and 2 of SC trait. Kar *et al.*[48] screened 60 cases of malaria and observed that sickle Hb was found in 7 (11.5%) patients and G6PD deficiency in 3 (5%) cases. One patient with falciparum malaria had both SC trait and G6PD deficiency. Balgir[31] observed 12 cases showing compound heterozygosity for SC hemoglobinopathy and G6PD deficiency. The author demonstrated an inverse relationship of SC allele with G6PD deficiency and beta thalassemia in a cross-section of malaria endemic (*Plasmodium falciparum*) tribal communities in Orissa. Balgir[50] found 52.2% patients with SCD to be G6PD deficient in hemizygous/heterozygous/homozygous condition in Dhelki Kharia tribal community of Orissa (Table 6).

Variations in the results can also be due to racial, ethnic, and geographic distribution. Variable prevalence of erythrocyte G6PD enzyme deficiency can also be explained by the fact that Indian population is composed of many heterogeneous religion and caste groups.

A difference in the frequency of G6PD deficiency among SCD patients can occur by post-zygotic selection, indeed

Table 5: Prevalence of G6PD deficiency in patients with and without sickle cell disease in various studies outside India

Name of author	Year	Place	Total number patients of SCD	Total number of G6PD deficient patients with SCD		Total number of patients without SCD	Total number of G6PD deficient patients without SCD	
				n	%		*n*	%
Naylor *et al.* [36]	1960	Chicago	56	8	14.3	54	8	15
Lewis *et al.*[37]	1966	Ghana	95	41	43.1	109	16	14.7
Piomelli *et al.*[38]	1972	New York	15	5	33.3	102	11	10.8
Beutler *et al.*[39]	1974	Los Angeles	21	4	19.1	167	15	9
Bienzle *et al.*[40]	1975	Nigeria	100	16	16	1451	313	21.6
Nhonoli *et al.*[41]	1978	-	93	13	14	543	106	19.5
Gibbs *et al.*[42]	1980	Jamaica	Male-53	12	22.6	-	-	-
			Female-67	19	28.3			
Heller *et al.*[43]	1979	Illinois	5072	566	11.16	62,331	6964	11.17
Andoka *et al.*[44]	1988	Congo	AS-44	14	31.8	128	59	25.8
			SS-42	19	45.2			
Bouanga *et al.*[2]	1998	Congo	188	42	22.2	210	47	22.5
Mohammad *et al.*[45]	1998	Bahrain	125	59	47	185	35	19
Diop *et al.*[46]	2005	France	319	69	21.6	318	39	12.3
Nouraie *et al.*[47]	2010	USA	261		8.7			

G6PD: Glucose-6-phosphate dehydrogenase, SCD: Sickle cell disease

Table 6: Prevalence of G6PD deficiency in patients with sickle cell disease along with controls in various studies in India

Name of author	Year	Place	Total number patients of SCD	Total number of G6PD deficient patients with SCD		Total number of patients without SCD	Total number of G6PD deficient patients without SCD	
				n	%		*n*	%
Praharaj *et al.*[15]	1977	Orissa		12		200	22	11
Kar *et al.*[48]	1990	Western Orissa		1		60	3	5
Balgir[31]	2006	Orissa		12				4.3-17.4
Balgir[49]	2008	Bhubaneshwar, Orissa		29				
Balgir[50]	2010	Sundargarh, Orissa	23	12	52.2			
Present study	2012	Raipur, Chhattisgarh	76	4	5.26	24	1	4.17

SCD: Sickle cell disease, G6PD: Glucose-6-phosphate dehydrogenase

by postnatal selection, since the phenotypic expression of the homozygous S condition is not significant until at least a few months after birth.

CONCLUSION

The present study has been conducted in the tertiary care hospital, which may or may not represent a true cross-section of the normal population. Yet in a preliminary study of this nature, the observation of 5% could be taken to consider that the problem of G6PD deficiency exists in this region and should be of concern as the enzyme deficiency remains obscure, there being no overt clinical manifestation. It would be important to keep in mind that an intrinsic anomaly of enzyme would make these deficient subjects vulnerable to some of the common drugs, which in therapeutic doses are harmless for persons with normal enzyme; hence, the detection of this enzyme deficiency is important for protecting such individuals.

The number of patients screened in the present study is not sufficiently large to make the observation of the enzyme G6PD deficiency highly significant, and it would be proper to screen a larger and representative sample of this region to know the exact nature and magnitude of the problem.

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