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Prevalence of Glucose 6 Phosphate Dehydrogenase deficiency in Eastern India, a Study from a Tertiary Care Hospital

Sudeb Mukherjee*

Department of Cardiology, Institute of Cardiovascular Science, India

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***Corresponding author:** Sudeb Mukherjee Department of Cardiology, Institute of Cardiovascular Science, India, Email: drsumukherjee@gmail.com

Abstract

a. Background: Glucose 6 phosphate dehydrogenase (G6PD) deficiency is an X-linked recessive hereditary disease characterized by abnormally low levels of Glucose 6 phosphate dehydrogenase. Certain racial and ethnic groups have an increased incidence of this deficiency. This study destined to reveal the prevalence of G6PD deficiency in Eastern India population.

b. Methods: Total 250 patients (186 male and 64 female) were included in this study. They were subsequently categorized into various subgroups and analysed properly.

c. Results: G6PD deficiency was found in overall 10 % of the population in this part of the world. Prevalence was higher in tribal community compared to non tribal community.

d. Conclusion: Given the large proportion of the population suffering from G6PD deficiency mass screening programme should be implemented to prevent drugs (antimalarial and others) induced unwarranted catastrophe.

Materials and Methods: The present study on incidence of Glucose 6 phosphate dehydrogenase was carried out in individuals attending the Inpatient section of the Department of Medicine in R G KAR Medical College Hospital, Kolkata. The study of glucose 6 phosphate dehydrogenase enzyme on the 250 cases admitted in this Hospital for varying reasons in the Department of Medicine was done with the help of commercially available kit for detecting erythrocyte glucose 6 phosphate dehydrogenase enzymes. This enzyme study is based on the principle of method originally devised by Kornberg and Horecker and of Lohr and Waller modified on the recommendation of the ICSH. Besides routine investigation, following investigations were also done with special attention in all cases.

i. Complete blood count.

ii. Reticulocyte count.

iii. Comment on peripheral blood smear.

iv. Quantitative estimation of Glucose 6 phosphate dehydrogenase enzyme.

Quantitative Estimation of G6PD enzyme:

a. Laboratory Procedures: This was done with the help of commercially available kit. It is a sigma procedure and is a modification of the spectrophotometric methods of Kornberg and Horecker [1], and of Lohr and Waller [2]. This involves measurement of the rate of increase in reduction of NADP to NADPH and its spectrophotometric absorbance at 340 nm serves to quantify enzymatic activity.

Observation & Results: Table 1 shows that out of total no. of cases studied (n = 250), the maximum incidence was observed in 21-40 years of age group (49.2%). The age of cases studied varies from 14 years to 60 years. Table 2 shows that out of total no. of cases studied (n = 250), 74.4 % were males while 25.6 % were females. The maximum incidence was observed in 21-40 years of age (49.2 %). Table 3 shows that among the total no. of males 55% belonged to Tribal community while 45 % belonged to Non-tribal community. Among the females 36 % belonged to Tribal community while 64 % belonged to Non-tribal community. The incidence of G6PD deficiency in the selected sample frame of cases was 10%, details of which have been shown in (Table 4). Comparison of G6PD activity between the Tribal and Non-tribal population showed that 60% of G6PD deficient cases belong to the Tribal community while the rest 40 % belong to the Non-tribal community (Table 5). Further analysis regarding gender distribution in different communities has been shown in Table 6 and Table 7.

Discussion: In India Chatterjee et al. (1961) worked on mixed Indian population and investigated for enzyme Glucose 6 phosphate dehydrogenase deficiency and reported the incidence of 5% in Konkan Hindus and Parsis, 5% in Bengalis, 6% in Muslims, 8% in Nepalese and 11% in Uttar Pradesh and Bihar including Jharkhand [3]. Thirteen biochemically characterized variants have been reported from India. At the

molecular level, G6PD Mediterranean is the most common deficient variant in the caste groups whereas, G6PD Orissa is more prevalent among the tribal of India. The third common variant seen in India is G6PD Kerala-Kalyan.

Bhasin and Walter reviewed the prevalence and distribution of Glucose 6 phosphate dehydrogenase deficiency in India by pooling data from 224 different studies based on geographical, occupational, ethnic and linguistic categories. Higher prevalence was reported from North and West than South India [4]. Studies from the Eastern parts of India were few. In Southern India only tribals of Tamil Nadu and Andhra Pradesh show high prevalence. The occupational groups did not show any difference in the incidence of Glucose 6 phosphate dehydrogenase deficiency. The frequency is higher among the tribal than the caste populations. Generally the Austro-Asiatic and Indo-European language groups show higher prevalence compared to the Dravidian language speaking groups. The highest frequency (27.94%) of Glucose 6 phosphate dehydrogenase enzyme deficiency has been reported from Surat, Gujarat. The Parsi population of Mumbai also shows high frequency. High prevalence in tribes can be explained in terms of the geographical spread of malaria [5]. The age of subjects range from 14-60 years with maximum number of subjects in the age group of 21–40 years similar distribution was noted in some of the hospital based studies [6,7].

Sex distribution of subjects under study revealed 74.4% (n = 186) were male, while 25.6% were females. Such male bias is well known due to outdoor nature. The study shows that incidence of Glucose 6 phosphate dehydrogenase deficiency is 10%. It showed that 60% of G6PDdeficient cases belong to the tribal community while the rest 40% belong to the non-tribal community. This is consistent with the higher incidence of G6PD and other genetic disorders seen in tribal population of India according to several studies [8]. This clustering of genetic disorders in the tribal population may be due to the inbreeding that still occurs in such populations.

80 % of the G6PD deficient were males. This is consistent with several studies conducted for glucose 6 phosphate dehydrogenase enzyme deficiencies. It is due to the X-linked nature of this genetic disorder. Heterozygous males manifest the disorder while females who are homozygous usually manifest the disorder and heterozygous females remain carriers. Fortunately, most of the G6PD deficient person will remain clinically asymptomatic throughout their lives.

However, a proportion of glucose 6 phosphate dehydrogenase deficient individuals develop neonatal jaundice or acute hemolytic anemia, which, if managed inadequately, can cause death or permanent neurological damage. The highest frequencies of glucose 6 phosphate dehydrogenase deficiency are in tropical Africa and tropical and subtropical Asia, which are also malaria-endemic areas. In areas of high incidence, clinicians and patients must be alert and prepared to avoid any factors that might trigger severe clinical manifestations of the deficiency. In the present study conducted in my hospital it was seen that the incidence of glucose 6 phosphate dehydrogenase enzyme deficiency was 10%. On comparing the incidence between tribal and non-tribal population in the study, it was seen that the incidence was comparatively higher in the tribal population.

Therefore to conclude When clinical and hematological findings raise the suspicion of glucose 6 phosphate dehydrogenase deficiency, the disorder should be confirmed by quantitative spectrophotometric measurement of red blood cell enzyme activity. There is also need for a large screening programme, especially in malaria endemic zones, where due to natural selection of population, there seems to be a higher incidence of glucose 6 phosphate dehydrogenase enzyme deficiency, which gives protection against severe malaria. Such screening programmes can be done using the rapid fluorescent spot test initially, after which findings can be confirmed by a quantitative assay if necessary.

Keywords: Glucose 6 phosphate dehydrogenase (G6PD); Tribal and nontribal community; Malaria

Table 1: Age Distribution of Cases under Study (n=250).

Age group In years	Total no. of cases in different age Groups	Percentage %
14 - 20 YEARS	30	12%
21 - 40 YEARS	123	49.2%
41 - 60 YEARS	97	38.8%
TOTAL	250	100

Table 2: Sex Distribution of Cases under Study (n=250).

Age group In years	Total no. of cases In different age Groups	No. of Males	No. of Females	% of Males	% of Females
14-20 YEARS	30	19	11	10.22%	17.19%
21-40 YEARS	123	93	30	50.0%	46.88%
41-60 YEARS	97	74	23	39.78%	35.94%
TOTAL	250	186	64	100 (74.40%)	100 (25.60%)

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Table 3: Sex distribution of the community (tribal/non-tribal) to which the cases under study belong.

Community	Total no. of cases in each community	No. of Males	No. of Females	% of Males	% of Females
TRIBAL	125	102	23	54.84 %	35.94 %
NON-TRIBAL	125	84	41	45.16 %	64.06 %
TOTAL	250	186	64	74.40 %	25.60 %

Table 4: Incidence of glucose 6 phosphate dehydrogynase deficiency in the sample frame.

G6pd enzyme activity	Total no. of Cases	Percentage %
NORMAL ACTIVITY (4.6 – 13.5 U/gm Hb)	225	90.0%
LOW ACTIVITY (< 4.6 U/gm Hb)	25	10.0%
TOTAL	250	100

Table 5: Comparison between Different Communities (Tribal/Nontribal) Of Incidence Of Glucose 6 Phosphate Dehydrogenise Deficiency In The Sample Frame.

Community	Total no. of cases In each Community	Normal g6pd activity	Low g6pd activity	% With normal g6pd	% with low g6pd
TRIBAL	125	110	15	48.89%	60%
NON-TRIBAL	125	115	10	51.11%	40%
TOTAL	250	225	25	100	100

Table 6: Sex distribution of the tribal community under study with glucose 6 phosphate dehydrogenase deficiency. Sex Total no. of cases with low

Sex	Total no. of cases with low G6pd activity (<4.6 u/gm hb)	Percentage %
MALE	12	80%
FEMALE	3	20%
TOTAL	15	100

Table 7: Sex distribution of the non-tribal community under study with glucose 6 phosphate dehydrogenase deficiency.

Sex	Total no. of cases with low G6pd activity (<4.6 u/gm hb)	Percentage %
MALE	8	80%
FEMALE	2	20%
TOTAL	10	100

Introduction

Glucose 6 phosphate dehydrogenase (G6PD) deficiency is an X-linked recessive hereditary disease characterized by abnormally low levels of G6PD, a metabolic enzyme involved in the pentose phosphate pathway, which is especially important in erythrocyte metabolism. G6PD deficiency is the most common human enzyme defect being present in more than 400 million people worldwide. African, Middle Eastern, Indian Subcontinent and South East Asian people are affected the most along with those who are mixed with any of the above [9]. All humans have the Glucose 6 phosphate dehydrogenase gene. Some people are born with a mutation of the Glucose 6 phosphate dehydrogenase gene. Most of these individuals are asymptomatic but may exhibit non-immune hemolytic anemia, even severe anemia in response to exposure to certain environmental triggers, most commonly, infection or exposure to certain foods like fava beans (favism), medications or chemicals [10].

Glucose 6 phosphate dehydrogenase deficiency is an X-linked recessive genetic phenomenon. So the symptomatic patients are

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almost exclusively male, due to the X-linked recessive nature of inheritance, but female carriers can be clinically affected due to unfavorable lionization [11]. Certain racial and ethnic groups have an increased incidence of this deficiency. Glucose 6 phosphate dehydrogenase deficiency does differ among populations. It is found generally in people whose ancestors have come from areas where malaria has been common, such as the Mediterranean region, Africa and South East Asia [12]. Apart from study done by Baxim and Chatterjee et al. [3] there is no recent study to reveal the prevalence of G6PD deficiency in eastern India so far. In the present hospital based study - the aim is to make a comparative study of incidence of Glucose 6 phosphate dehydrogenase deficiency in West Bengal between tribals and non-tribals.

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