ORIGINAL ARTICLE

PREVALENCE OF G6PD DEFICIENCY IN SCHOOL GOING ENROLLED CHILDREN IN DISTRICT MARDAN

Mohtasimbillah[1], Sardar Ahmad[2], Imtiaz Ahmad[3], Hiba Khan[1], Hifsa Gul[1], Abid Sohail Taj[4]

[1]Department of Pathology, Bacha Khan Medical College Mardan - Pakistan

[2]Department of Pathology, Saidu Medical College, Swat - Pakistan

[3]Department of Pathology, BKMC Mardan - Pakistan

[4]Department of Pathology, IBMS Peshawar - Pakistan

ABSTRACT

Objective: To find out prevalence of Glucose 6 Phosphate Dehydrogenase (G6PD) deficiency in school going enrolled children of District Mardan.

Material and Methods: The study was carried out in community based primary schools, where 400 male apparently healthy children in the age range of 5-10 years with a mean age of 8.55 ± 1.23 yrs. were randomly selected. The activity of G6PD was determined by a dye reduction method screening test devised by Sigma Diagnostics USA.

Results: The overall prevalence of G6PD deficiency in our study was 10%. Further analysis showed that distribution in two tehsils of the district i.e., Tehsil Mardan was 8.5 % and in Tehsil Takhtbhai was 11.5 %.

Conclusion: Glucose 6 phosphate Dehydrogenase deficiency is one of the most commonly occurring congenital red cell enzymopathy in our province. Being highly prevalent, community screening for early detection of susceptible cases will not only prevent the development of hemolytic crises by avoiding oxidative stress but will also improve the quality of life.

Key Words: Glucose 6 phosphate Dehydrogenase Deficiency (G6PD), Oxidative stress.

This article may be cited as: Mohtasimbillah, Ahmad S, Ahmad I, Khan H, Gul H, Taj AS. Prevalence of G6PD deficiency in School going enrolled children in District Mardan. J Med Sci 2017; 25: (3) 308-312.

INTRODUCTION

Glucose-6-Phosphate-Dehydrogenase (G6PD) deficiency is one of the most common congenital red cell enzymopathy, affecting approximately 400 million people, expressed as X linked disorder[1,3]. It is one of the most common clinically significant among the enzymatic defects not only in haematology, but also in human biology as a whole[2]. The disease was discovered fifty years ago and gained the attention of scientific community for its severe effect on RBCs[3,4].

G6PD deficiency is widespread but is most common in Africa and Arabian Peninsula with a reported prevalence of 32.5%[3]. Rai V et al reported 9.3% and 10.7% in females and males respectively[4]. G6PD produces NADPH during glucose metabolism, maintaining RBC integrity[5]. G6PD catalyze the first step of glucose metabolism converting glucose 6 phosphates to 6 phosphogluconate. NADPH is converted to NADP+ with concurrent conversion of glutathione to reduced glutathione. Reduced glutathione in turn prevents oxidation of hemoglobin and other cellular protein hence has a protective effect of RBC by protecting it from oxidative damage of drugs, infections and environmental stresses[6]. The prevalence rate varies from as high as 62% among Kurdish Jews to as low as 0.1% in Japan and Europe, while it ranges from 3 to 6.9% in Pakistan southern china and southern Russia[1,7]. According to WHO the prevalence of G6PD is 10 and14% in Iran and Bengal respectively[8]. The frequent occurrence of consanguineal marriages and homozygous female add more to the frequency of disease. In Pakistan there is no large scale study on prevalence of G6PD deficiency. Husain et al reported 16% in neonates admitted in nursery unit Lady Reading hospital Peshawar with jaundice[9]. In other study conducted by Habeeb ur Rehman et al in nursery unit of Khyber teaching hospital as 30.1%[10].

Dr. Mohtasimbillah (Corresponding Author)
Department of Pathology, Bacha Khan Medical College Mardan, Khyber Pakhtunkhwa, Pakistan
Cell: 03005772024
Email: ikrambkmc@gmail.com

Date Received: March 3, 2017
Date Revised: June 10, 2017
Date Accepted: September 20, 2017

MATERIAL AND METHODS

This study was one of the first which was conducted in healthy school going children. A total 400 male primary school going children with age range from 5 to 10 years with a mean age of 8.55 ± 1.23 years were included in this study.200 students from Tehsil Mardan and 200 from Tehsil Takhtbhai without any racial discrimination were included. Different experimental models can be envisaged for an effective prevalence based study. Lau et al reported a community based screening program as a feasible option for screening of thalassemia trait in Hong Kong by screening school going children. We applied similar experimental model for prevalence studies of G6PD in Mardan. District Mardan which comprises a heterogeneous population has not been previously screened for the disease. A study so conducted here was expected to yield newer findings depicting a gross general prevalence of the region and stratified specific prevalence for the inhabitant tribes. Such experimental models can be adopted for prevalence studies in a low socio-economic background for scientifically valid and socially effective demographic studies. The school going children were selected because they were true representative of the resident communities. Total population of children between the ages of 05 - 10 is approximately 0.28 million. Epidemiological studies in above population thus become statistically viable analysis.

Male Primary school children, age 5 to 10 years were selected because of lower rate of dropout in these children. Out of 75 Union councils of the district, we randomly selected ten union councils from two Tehsils i.e. Mardan and Tehsil Takhtbhai. Random selection of Schools both from public and private sectors was made through draw from each of ten Union councils. A total of 400 hundred students, forty from each school were randomly selected.

Study Setting

This study was carried out in Hematology departments of Bacha Khan Medical College (BKMC) Mardan.

Sample collection

Prior written parental consent was obtained through school administration. Proper protocol for collection of blood samples by aseptic techniques was followed. After selection of students from different classes using inclusion and exclusion criteria, blood sample were collected. Basic information was recorded on a structured Proforma. Venous blood samples were collected in K+ EDTA tube with proper shaking of blood. EDTA tube and Proforma of each student was labeled separately and EDTA tube was put in sample container. Different methodologies are used for G6PD estimation; the common types are fluorescent spot, methaemoglobin, qualitative and quantitative estimation. We selected the colour decolarization qualitative method using commercially available sigma kit.

RESULTS

The study was conducted in male primary school going children in Mardan district from March 2012 to December 2012. Out of 400 hundred students 40 forty students (10%) were found to be glucose 6 phosphate Dehydrogenase (G6PD) deficient.

The study population was heterogeneous and was found to comprise several major and minor tribes. Table 2 depicts the prevalence of G6PD deficiency in these tribes. The miscellaneous group consists of several minor tribes.

DISCUSSION

This study was conducted in Mardan district. The district comprises of two tehsils, with a diverse group of population represented in study sampling. The district is home to the major tribes of Yousafzai Pathans and emigrants from tribal areas of Mohmand, Bajaur, Dir, and Khyber Agency. In order to obtain a representative sample of children of various groups, we visited the primary level schools in urban and rural set-up of both tehsils.

Glucose-6- Phosphate dehydrogenase deficiency was first reported in a study on Pathans by Stern et al.,[11]. The main stay of our study was to find the prevalence of G6PD deficiency in Mardan district. A number of hospital based studies have been reported in the

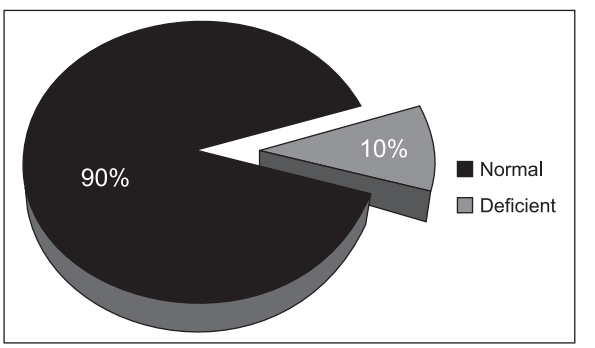

Figure 1. Prevalence of G6PD deficiency in District

Table 1. Tehsil Wise Distribution of G6PD Deficiency

Tehsils	Total Number of Children	G6PD deficient	%age
Mardan	200	17	8.5%
Takhtbhai	200	23	11.5%

Table 2. Showing Distribution of G6PD deficiency in different tribes residing in Mardan

		G6PD		Total
		Normal	Deficient	
Bajauri	Count	67	13	80
	% within Tribes	83.8%	16.2%	100.0%
	% within G6PD	18.6%	32.5%	20.0%
	% of Total	16.8%	3.2%	20.0%
Mohmand	Count	80	13	93
	% within Tribes	86.0%	14.0%	100.0%
	% within G6PD	22.2%	32.5%	23.2%
	% of Total	20.0%	3.2%	23.2%
Malizai	Count	70	5	75
	% within Tribes	93.3%	6.7%	100.0%
	% within G6PD	19.4%	12.5%	18.8%
	% of Total	17.5%	1.2%	18.8%
Afridi	Count	27	1	28
	% within Tribes	96.4%	3.6%	100.0%
	% within G6PD	7.5%	2.5%	7.0%
	% of Total	6.8%	0.2%	7.0%
Khattak	Count	31	3	34
	% within Tribes	91.2%	8.8%	100.0%
	% within G6PD	8.6%	7.5%	8.5%
	% of Total	7.8%	0.8%	8.5%
Yousafzai	Count	77	3	80
	% within Tribes	96.2%	3.8%	100.0%
	% within G6PD	21.4%	7.5%	20.0%
	% of Total	19.2%	0.8%	20.0%
Miscellaneous	Count	8	2	10
	% within Tribes	80.0%	20.0%	100.0%
	% within G6PD	2.2%	5.0%	2.5%
	% of Total	2.0%	0.5%	2.5%
Total	Count	360	40	400
	% within Tribes	90.0%	10.0%	100.0%
	% within G6PD	100.0%	100.0%	100.0%
	% of Total	90.0%	10.0%	100.0%

province, where the true picture of prevalence could not be derived at. In this study, population was drawn from community based primary schools on the analogy of similar studies of established repute[12,13]. Proper selections of schools of different union councils of both tehsils were done. The study population included apparently healthy school going children aged 5-10 years. The main finding of our study was significantly high prevalence of G6PD deficiency that was 10.0%. This study is in agreement with some of the previous studies[1,3,14,15,16].

The discrepancy between our results and some of the previous studies could be due to actual design of the studies. Most of studies reported in Pakistan are institutional based. The sample selection in the studies thus affects the ultimate result. In our study the Mohmand and Bajaur tribes have the highest frequency of G6PD deficiency. As both tribes belong to the tribal areas of KPK, one can hypothesize a number of reasons for it. Tribals are known to strictly adhere to their traditions and customs, which after all are very conservatives. Consanguinity of marriages and marrying within the tribes is closely followed; hence genetic disorders are restricted and compounded in tribal community with passage of time. The analysis showed that Mohmand and Bajaur tribes were having high prevalence of G6PD deficiency as compared to other ethnic groups in Mardan, which is partially consistent with Khan et al[17,18]. The possibility of founder members of Mohmand and Bajauar might have been deficient in G6PD enzyme, which may be the reason of developing high prevalence of G6PD deficiency in these tribes residing in district Mardan. Ali et al, reported 8% frequency in Pathans[19]. Bauma et al, reported that the prevalence of G6PD deficiency in Pukhtoons adult male was 11.4%[20]. Our study did not agree with the report of Moiz et al., conducted in Karachi where she has shown the Prevalence of 6.2%[21]. This difference could be attributed to small sample size and higher mean age of sample population than our study. Hashmi et al, reported 7% G6PD deficiency in young male[22]. Marked difference has been reported from other regions with low prevalence by Ronald et al., in which he included soldiers in his study and the prevalence was 2.6% which greatly contradicts our findings[23]. Though the sample size was adequate in the afore-mentioned study, the difference may be attributed to the fact that army soldiers from all over Pakistan were included whereas our study was in a single distinguished district of the province. Alvi et al, and Imran et al., study on Pakistani neonates has reported 10 % and 12 % deficiency[24,25]. Khattak et al., observed 12% G6PD deficiency in adults in KPK[26]. Lal et al., found 14% G6PD deficiency in neonates[15]. In a recent study by Assadullah et al., and Khan et al.,reported as 16%

and 13 % prevalence in neonates with jaundice[16,27]. Ali et al., reported 29.3% G6PD deficiency in jaundiced infants[28]. Jan found 9% neonates to be G6PD deficient presenting with jaundice[29].

CONCLUSION

G6PD deficiency, being a highly prevalent disease, is a major cause of neonatal jaundice and haemolytic anemia. It is recommended to devise a policy to screen local population for the disease. Parents of G6PD deficient children should be educated to avoid anti-oxidant drugs and a list of antioxidant drugs should be provided to them.

RECOMMENDATIONS

It is recommended that a detailed mutational analysis' study be conducted to know the spectrum of mutations in local population. The study design, we presented, can be projected to screen larger study groups for better generalization of the findings.

REFERENCES

1. Nkhoma ET, Poole C, Vannappagari V, Hall SA, Beutler E. The global prevalence of glucose-6-phosphate dehydrogenase deficiency: a systematic review and meta-analysis. Blood Cells Mol Dis. 2009; 42(3):267-78.
2. Beutler E. Glucose-6-phosphate dehydrogenase deficiency: a historical perspective. Blood. 2008;111:16-24.
3. Rosalind E.H, Frederic BP,Anand PP ,Oscar A.N, Peter W G et al G6PD deficiency prevalence and estimates of affected populations in malaria endemic countries: A geostatistical model-based map.2012, 9 ,(11) :1-15
4. Rai V, Kumar P. Epidemiological study of glucose-6-phosphate dehydrogenase deficiency in scheduled caste population of India. Journal of Anthropology.2012, article id 984180, doi:10.1155/2012/984180.
5. Beutler E .G6PD Deficiency. Blood: 1994, 84 (11) 3613---3636.
6. http://en.wikipedia.org/wiki/glucose-6 phosphate dehydrogenase deficiency, date accessed 9-2014.
7. Beutler E. Hemolytic anemia in disorders of red cell metabolism. New York: Plenum medical book company, 1978: 23-169.
8. Khan M: Glucose 6 phosphate dehydrogenase deficiency in adults. J Coll Physicians surg Pak 2004, 14(7):400-403.
9. Hussain M, Irshad M, Kalim M, Ali L, Ali L. Glucose-6-Phosphate dehydrogenase deficiency in jaundiced neonates. J Postgrad Med Inst 2010; 24(2): 122-126
10. Rehman H ,Khan, Roghani MT et al. Erythrocyte glucose 6-phosphate dehydrogenase deficiency and neonatal jaundice. JPMA.1995; 45:259—264.
11. Stern MA, Kynoch PA, Lehmann H. Beta-thalassemia, glucose-6-phosphate-dehydrogenasedeficiency, and hemoglobin D-in Punjabi and Pathans. Lancet 1968; 1: 1284-5.
12. Khattak MI, Ishaq T, Subhan M, Afridi J. The frequency and age presentation of G6PD deficiency in 200 patients of hemolytic anemia. J Postgrad Med Inst 2006; 20: 170-3.
13. LAU YL, Chan LC, Yin YA, Ha SY, Yeung CY et al. Prevalence and genotype of α and β thalassemia. NEJM 1997,336,18:1229-1301
14. Mohanty D, Mukherjee MB & Colah RB.Glucose-6-phosphatedehydrogenase deficiency in India. Indian J Pediatr; 2004; 71 (6): 525-529.
15. Amreek L, Neem S, A.Ashfaq et al, Frequency of glucose phosphate dehydrogenase deficiency in swat. JSMC 2013;3: 145-153.
16. Assad U, Afzal S, Yar M et al,Glucose 6 phosphate dehydrogenase deficiency International journal of advance research 2014;2:(11): 888-899.
17. Khan TA, Ahmed S, Anwar M, Ayyub M. The frequency of Glucose 6 phosphate deficiency in Punjabis and Pathans. JPMI 2005; 18 (4): 592-97.
18. Brian Glyn Williams, Professor of Islamic studies, U. Mass Dartmouth; author, 'Predators' and 'The Last Warlord 'The Lost Children of Alexander the Great: A Journey to the Pagan Kalash People of Pakistan. The New York Times Posted: 02/21/2014 10:28 pm EST.
19. Ali N, Anwar M, Ayyub M, Bhatti FA, Nadeem M, Nadeem A. Frequency of glucose-6-phosphate dehydrogenase deficiency in some ethnic groups of Pakistan. J Coll Physicians surg Pak 2005; 15: 137-41.
20. Bouma MJ, Goris M, Akhtar T,Khan N, Khan N, Kita E: Prevalence and clinical presentation of glucose-6-phosphate dehydrogenase deficiency in Pakistani pathans and afghan refugee communities in Pakistan; implications for the use of Primaquine in regional malaria control programmes. Trans R Soc Trop Med Hyg 1995, 89(1) 62—64.
21. Moiz B, Nasir A, Moatter T, Naqvi ZA, Khurshid M. Molecular characterization of glucose-6-phosphate dehydrogenase deficiency in Pakistani population. Int J Lab Hematol 2011; 33: 5708
22. Hashmi JA, Farzana F, Ahmad M. Abnormal Hemoglobin, Thalassemic trait. J Lab clinical Med 1970; 76: 8-9.

23. Ronald AR, Underwood BA, Woodward TE. Glucose-6-phosphate dehydrogenase deficiency in Pakistani males. Trans R Soc Trop Med Hyg 1968; 62: 531-3.

24. Alvi MY, Laeeq A, Khan MA, Iqbal MA. Glucose-6-Phosphate Dehydrogenase deficiency associated with neonatal jaundice Pak Paed J 2006; 30: 28-33.

25. Imran M, Rashid, Akbar A. Neonatal jaundice due to G6PD deficiency. Pak Ped J 1984; 8: 126-28

26. Khattak MF, Dawood MM, Saleem M. The prevalence of glucose 6 phosphate dehydrogenase deficiency in Northern Pakistan. Pak Armed Forces Med J 1992; 42: 35-8.

27. Khan A, Khawar N Glucose 6 phosphate dehydrogenase deficiency with neonatal hyperbilirubinemia 1988 JPMI 16 (1) 33-38.

28. Ali Q, Aqeel M, Iqbal H frequency of glucose 6 phosphate dehydrogenase in neonatal jaundice JMS 201;1 (4):167-170.

29. Jan AZ, Zahid SB, Ahmad S. Frequency of G6PD deficiency and its severity in neonatal jaundice in Rehman Medical Institute, Peshawar. Khyber Med Univ J 2013; 5(1): 36-39

CONFLICT OF INTEREST: Authors declare no conflict of interest

GRANT SUPPORT AND FINANCIAL DISCLOSURE NIL

AUTHOR'S CONTRIBUTION

Following authors have made substantial contributions to the manuscript as under:

Mohtasimbillah: Literature Review

Ahmad S: Data Analysis

Ahmad I: Laboratory Works

Khan H: Data Collection

Gul H: Statistical Analysis

Taj AS: Proof Reading

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.