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G6PD Deficiency in Dehradun Population

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***Abstract* - *Glucose-6-phosphate dehydrogenase (G6PD)* deficiency increases the vulnerability of erythrocytes to oxidative stress. Clinical presentations include acute hemolytic anemia, chronic hemolytic anaemia, neonatal hyperbilirubinemia, and an absence of clinical symptoms. The present study on incidence of *Glucose 6 phosphate dehydrogenase* was carried out in population of Dehradun region. The study of *glucose 6 phosphate dehydrogenase* enzyme on the 300 cases was done with the help of commercially available kit for detecting erythrocyte *glucose 6 phosphate dehydrogenase enzymes*. Present study concludes that *G6PD* deficiency is prevalent in population of Dehradun, region, it is of mild type. It is recommended that every subject should get tested for G6PD deficiency and carry G6PDstatus card while visiting doctor.**

Keywords: *Glucose-6-phosphate dehydrogenase (G6PD)* deficiency, oxidative stress, anaemia.

I. INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency increases the vulnerability of erythrocytes to oxidative stress. Clinical presentations include acute hemolytic anemia, chronic hemolytic anaemia, neonatal hyperbilirubinemia, and an absence of clinical symptoms. The disease is rarely fatal.

G6PD deficiency occurs with increased frequency throughout Africa, Asia, the Mediterranean, and the Middle East. In the United States, black males are most commonly affected, with a prevalence of approximately 10 percent. Prevalence of the deficiency is correlated with the geographic distribution of malaria, which has led to the theory that carriers of G6PD deficiency may incur partial protection against malarial infection. **(Ruwende C and Hill A; 1998; Mockenhaupt FP *et al*; 2003)** Cases of sporadic gene mutation occur in all populations.

The gene mutations affecting encoding of G6PD are found on the distal long arm of the X chromosome. More than 400 mutations. Have been identified, most being missense mutations. Most of the variants occur sporadically, although the G6PD Mediterranean and the G6PD A– variants occur with increased frequency in certain populations **(Beutler E; 1994).**

The diagnosis of G6PD deficiency is made by a quantitative spectrophotometric analysis or, more commonly, by a rapid fluorescent spot test detecting the generation of NADPH from NADP. The test is positive if the blood spot fails to fluoresce under ultraviolet light. In field research, where quick screening of a large number of patients is needed, other definitive testing to confirm an abnormal result. Tests based on polymerase chain reaction detect specific mutations and are used for population screening, family studies, or prenatal diagnosis.6In patients with acute hemolysis, testing for G6PD deficiency may be falsely negative because older erythrocytes with a higher enzyme deficiency have been hemolyzed. Young erythrocytes and reticulocytes have normal or near-normal enzyme activity. Female heterozygotes may be hard to diagnose because of X-chromosome mosaicism leading to a partial deficiency that will not be detected reliably with screening tests**.(Reclos GJ *et al*;2007)**. G6PD deficiency is one of a group of con-genital hemolytic anemias, and its diagnosis should be considered in children with a family history of jaundice, anemia, spleno-megaly, or cholelithiasis, especially in those of Mediterranean or African ancestry. 13Testing should be considered in children and adults (especially males of African, Mediterranean, or Asian descent) with an acute hemolytic reaction caused by infection, exposure to a known oxidative drug, or ingestion of fava beans. Although rare, G6PD deficiency should be considered as a cause of any chronic nonspherocytic hemolytic anemia across all population groups. Newborn screening for G6PD deficiency is not performed routinely in the United States, although it is done in countries with high disease prevalence. The World Health Organization recommends screening all newborns in populations with a prevalence of 3 to 5 percent or more in males.

Neonatal Hyperbilirubinemia: The prevalence of neonatal hyperbilirubinimia is twice that of the general populationin males who carry the defective gene and in homozygous females. It rarely occurs in heterozygous females**. (Corchia C *et al*; 1995).**

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Acute Hemolysis: Acute hemolysis is caused by infection, ingestion of fava beans, or exposure to an oxidative drug in G6PD deficient subjects.

Chronic Hemolysis: In chronic nonspherocytic hemolytic anemia, which usually is caused by a sporadic gene mutation, hemolysis occurs during normal erythrocyte metabolism. The severity of the hemolysis varies, causing mild hemolysis to transfusion-dependent anemia. Exposure to oxidative stress can cause acute hemolysis in these persons**.(Mason PJ;1996).**

Other clinical considerations: G6PD-deficient persons are predisposed to the development of sepsis and complications related to sepsis after a severe injury. Although research has failed to consistently show a clinically significant risk to patients receiving G6PD-deficient donor blood, blood banks generally do not accept G6PD-deficient blood donors. **(CBBS e-Network Forums; 2005).**

The main treatment for G6PD deficiency is avoidance of oxidative stressors. Rarely, anemia may be severe enough to warrant a blood transfusion. Splenectomy generally is not recommended. Folic acid and iron potentially are useful in hemolysis, although G6PD deficiency usually is asymptomatic and the associated hemolysis usually is short-lived. Antioxidants such as vitamin E and selenium have no proven benefit for the treatment of G6PD deficiency. 6, 31 researches is being done to identify medications that may inhibit oxidative induced hemolysis of G6PD-deficient red blood cells**. (Sharma SC *et al*; 2003).**

Considering the genetic nature of this disease, its complications and lack of adequate treatment, it should be widely screened in the general population. However studies in India are scanty and no study is reported in Dehradun region.

II. MATERIAL AND METHODS

The present study on incidence of *Glucose 6 phosphate dehydrogenase* was carried out in population of Dehradun region.. The study of *glucose 6 phosphate dehydrogenase* enzyme on the 300 cases 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.

III. RESULT AND DISCUSSION

Table 1 shows that out of total no. of subjects studied (n = 300), the age of subjects studied varies from 14 years to 60 years. The maximum number of subjects was in 21-40 years of age group.

Table 1: Age Distribution of Cases under Study (n=300)

Age group in years	Total no. of cases in different age Groups	%
14 - 20 YEARS	40	13.3%
21 - 40 YEARS	153	51%
41 - 60 YEARS	107	41.6
TOTAL	300	100

Table 2 shows that out of total no. of subjects studied (n = 300), 77.6 % were males while 22.3 % were females. The maximum subjects were in 21-40 years of age both in males (36.6%) and females (11%).

Table 2: Sex Distribution of Cases under Study (n=300)

Age group In years	Total no. of subjects in different age Groups	No. of Males	No. of Females	% of Males	% of Females
14-20 YEARS	40	24	16	8%	5.33%
21-40 YEARS	153	110	33	36.6%	11%
41-60 YEARS	107	99	18	33%	6%
TOTAL	300	233	77	100 (77.6%)	100 (22.33%)

The incidence of G6PD deficiency in the selected sample frame of cases was 15%, details of which have been shown in (Table 3).

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Table 3: 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)	255	85.0%
LOW ACTIVITY (< 4.6 U/gm Hb)	45	15.0%
TOTAL	300	100

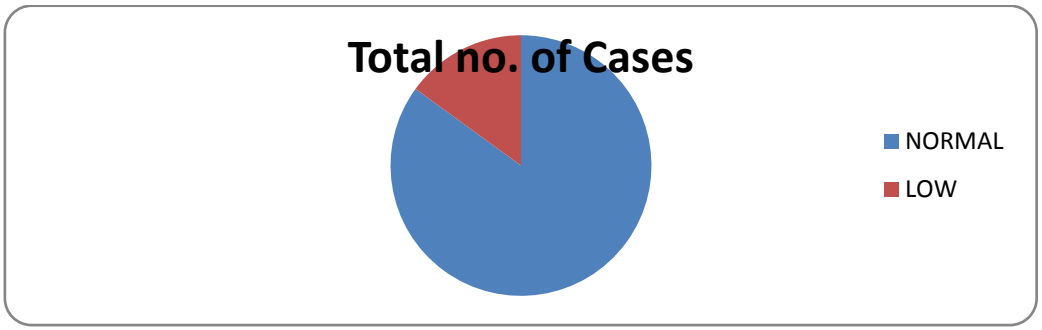

Figure 1: Incidence of *glucose 6 phosphate dehydrogynase* deficiency in the sample frame

Table 4: Distribution of G6PD deficient subjects among different sex and age groups

Age group In years	Total no. of *G6PD deficient* subjects in different age Groups	No. of Males	No. of Females
14-20 YEARS	10	11 (24.5%)	04 (8.5%)
21-40 YEARS	25	16 (35.5%)	05 (11.5%)
41-60 YEARS	10	07 (15%)	02(5%)
TOTAL	45	**34(75%)**	**11 (25%)**

The table 4 depicts the distribution of *G6PD* deficient subjects among different sex and age groups Out of 45 *G6PD* deficient subjects 34 (75%) were males and 11 were females (25%) Maximum incidence was observed in 21-40 years in both the sexes.

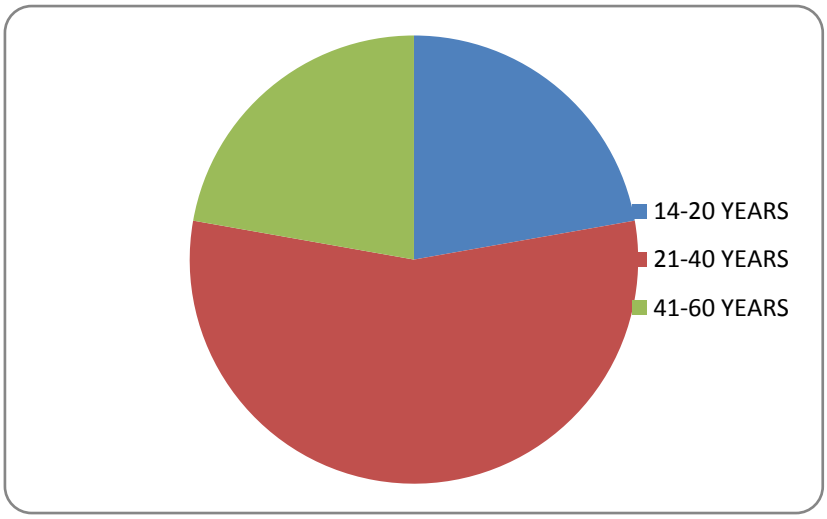

Figure 2: Age Wise Distribution of G6PD Patients

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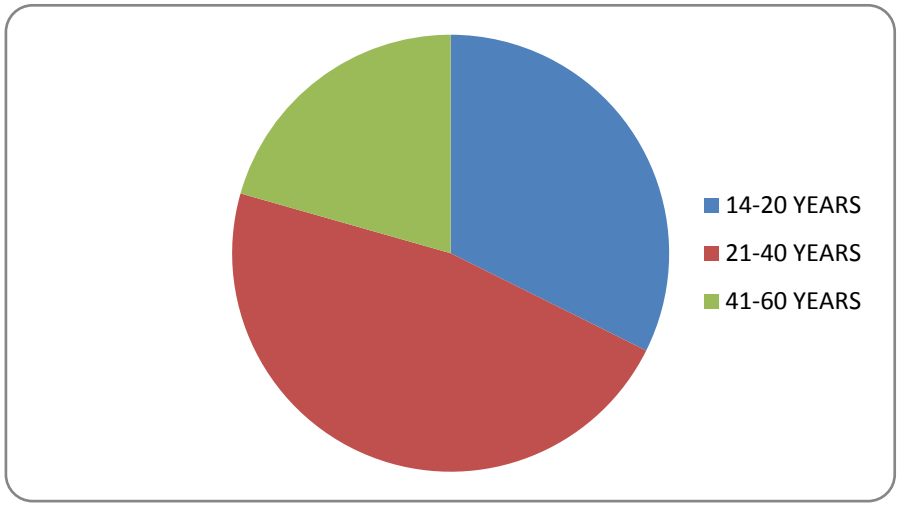

Figure 3: Age Wise Distribution of G6PD Patients in males

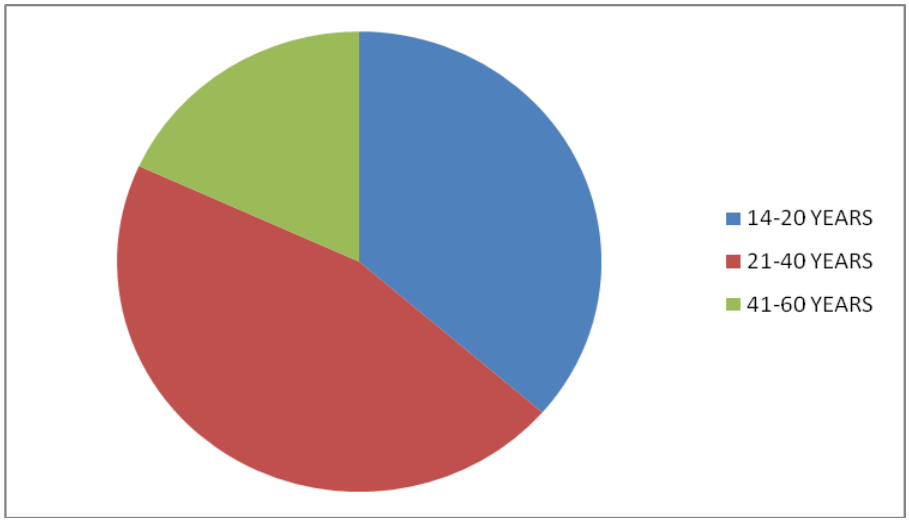

Figure 4: Age Wise Distribution of G6PD Patients in males

In India **Chatterjee *et al.* (**1963) 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. 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

The study shows that incidence of *Glucose 6 phosphate dehydrogenase* deficiency is 15%.**Bhasin and Walter (2001)** 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. 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 **[Chatterjee JB 1966].** The age of subjects range

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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 **[SC Gupte *et al;2005;*. TP Sharma, and Rajkumari 2010)].**

Sex distribution of subjects under study revealed 77.6% (n = 233) were male, while 22.33% were females. Such male bias is well known due to outdoor nature.

75 % 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.

IV. CONCLUSION

Present study concludes that G6PD deficiency is prevalent in population of Dehradun, region, it is of mild type. It is recommended that every subject should get tested for G6PD deficiency and carry G6PD status card while visiting doctor.

REFERENCES

[1] Ruwende C, Hill A. Glucose-6-phosphate dehydrogenase deficiency and malaria. J Mol Med 1998; 76:581-8.
[2] Mockenhaupt FP, Mandelkow J, Till H, Ehrhardt S, Eggelte TA, Bienzle U. Reduced prevalence of Plasmodium falciparum infection and of concomitant anaemia in pregnant women with heterozygous G6PD deficiency. Trop Med Int Health 2003; 8:118-24.
[3] Beutler E. G6PD deficiency. Blood. 1994; 84:3613–36.
[4] Reclos GJ, Hatzidakis CJ, Schulpis KH. Glucose-6-phos-phate dehydrogenase deficiency neonatal screening: preliminary evidence that a high percentage of partially deficient female neonates are missed during routine screening. J Med Screen 2000; 7:46-51.
[5] Carlo Corchia, Antonio Balata, Gian Franco Meloni and Tullio MeloniFavism in a female newborn infant whose mother ingested fava beans before delivery, The Journal of Pediatrics, 127 (5)1995, : 807-808.
[6] Mason PJ. New insights into G6PD deficiency. Br J Hae-matol 1996; 94:585-91.
[7] CBBS e-Network Forums. Eligibility of prospective blood donors known to have G6PD deficiency. Accessed online July 20, 2005, at: http://www.cbbsweb.org/enf/donor_g6pd.html
[8] Sharma SC, Sharma S, Gulati OP. Pycnogenol prevents haemolytic injury in G6PD deficient human erythro-cytes. Phytother Res 2003; 17:671-4.
[9] Kornberg A, Horecker BL. Glucose-6-phosphate dehydrogenase. In: Colowick SP, Kaplan NO, editors. Methods in Enzymology. New York, NY: Academic Press; 1959.
[10] Chatterji SC, Das PK Chloramphenicol induced haemolyticanaemia due to enzymatic deficiency of erythrocytes. J Indian MedAssoc 40: 172, 1963.
[11] Chatterjee JB (1966) Hemoglobinopathies, G6PD deficiency and allied problems in the Indian Sub-continent. Bull WHO 35(6): 837-856.
[12] TP Sharma, Rajkumari (2010) Glucose-6-phosphate dehydrogenase deficiency in Danguria Tharu settled in Baharaich district of Uttar Pradesh. The Anthropologist 12(1): 59-61.
[13] Sharma SC, Sharma S, Gulati OP. Pycnogenol prevents haemolytic injury in G6PD deficient human erythro-cytes. Phytother Res 2003; 17:671-4.

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