

G6PD Status in Patients with Presenile And Senile Cataract

Farzana Yasmin¹, Noorzahan Begum², Sultana Ferdousi³

Abstract

Background: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is one of the common enzymopathy and may be one of the risk factor for both presenile and senile cataract. **Objective:** To observe erythrocyte G6PD level in male patients with presenile and senile cataract in order to find out their enzyme status. **Methods:** This cross sectional study was carried out in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag Dhaka between 1st July 2009 and 30th June 2010. 60 male patients with presenile and senile cataract were included in the study group (Group-B). They were selected from Out patient Department (OPD) of Ophthalmology of BSMMU in Dhaka City. For comparison age matched 60 apparently healthy male without cataract (Group A) were also studied. According to age both study & control group were again subdivided in to Group B₁ & A₁(presenile,aged 40-60 years) and Group B₂ & A₂(senile, aged >60 years). Erythrocyte G6PD level was measured by Spectrophotometric method. Data were analyzed by independent sample t test, ANOVA, Chi-square test as applicable. **Results:** Mean erythrocyte G6PD level was significantly lower (P<0.01) in the presenile and senile cataractous groups compared to their corresponding noncataractous subjects. However 26.7% cataractous patients in presenile and 6.7% in senile group were G6PD deficient. **Conclusion:** Erythrocyte G6PD deficiency may be present in both presenile and senile cataract but more marked in presenile cataract patients.

Key words: G6PD, Cataract.

J Bangladesh Soc Physiol. 2011 June; 6(1): 1-4
For author affiliations, see end of text.

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Introduction

Cataract has been recognized by World Health Organization (WHO), as prime cause of impaired vision and blindness affecting more than 17 million people throughout world¹. Individuals with cataract at the age range of 40 to 60 years and above 60 year of age respectively was termed as presenile and senile cataract^{6,9}.

Pathophysiology of cataract involved denaturation and cross linking of lens proteins, with loss of transparency which is aggravated by its oxidative damage. Recently, it has been reported that raised H₂O₂ may cause development of cataract in G6PD deficient subjects¹⁰.

G6PD deficiency is predominantly a male syndrome. Since male possess only one copy of the gene encoding for G6PD, they are either normal or G6PD deficient. On the other hand, females are normal, heterozygous or homozygous. In the affected subject of the Mediterranean type, the enzyme deficiency occurs not only in the RBC but also in the lens in which this enzyme play an important role in maintaining transparency⁶.

Orzalesi, Sorcinelli and Gulso found that significantly higher prevalent G6PD deficiency among the patients with cataract⁶.

Bhatia, Patel and Dubley observed that the frequency of G6PD deficiency was higher in the presenile group but progressively lower in the senile group¹.

Again, Chen et al. reported that both in RBC and lens, presenile cataractous groups had lower G6PD level².

Cataract comprises 80% of avoidable blindness in Bangladesh⁴. In our country many people are suffering from cataract. But there is lack of information regarding G6PD enzyme status in these cataractous patients which may have some contribution in the disease. Therefore, the present study was carried out to observe erythrocyte G6PD status in presenile cataractous patients in order to find out the relationship between this enzyme deficiency and the development of presenile cataract.

Methods

This Cross-sectional study was carried out in the Department of Physiology, BSMMU, Dhaka between July 2009 and June 2010. Total number of 120 male subjects of 40-70 years of age were selected for the study. 60 male patients with cataract were enrolled for the study group (GroupB) from the Out Patients Department (OPD) of Bangabandhu Sheikh Mujib Medical University (BSMMU), Shahbag. 60 age, BMI, sex matched apparently healthy noncataractous subjects were randomly selected from the community and were taken as control (GroupA). Based on the age both study and control group were subdivided in to group B₁ (presenile cataractous, aged 40-60 years) and A₁ (presenile non cataractous, aged 40-60 years) and Group B₂ (senile cataractous, aged >60 years) & A₂ (senile noncataractous, aged >60 years). Subjects with traumatic eye, diabetes mellitus, radiation injured eye, hyperparathyroidism, hypertension, glaucoma and steroid users were excluded from the study. Protocol of this study was approved by ethical review committee of BSMMU. After selection of the subjects the purpose and benefits of the study were explained

to each subject and informed written consent was taken from them.

A detailed personal, medical, family, socioeconomic, drug history was recorded in a prefixed questionnaire. Thorough physical examinations including slit lamp examination of eye of all subjects were done to confirm the presence of cataract. Then 3 ml of venous blood was collected and erythrocyte G6PD level was measured by spectrophotometric method. The data were expressed as Mean \pm SD and was analyzed by unpaired Student's 't' test, ANOVA and Chi-square test as applicable.

Results

Anthropometric data of study subjects are presented in Table I. Both groups were similar in respective to age and BMI. Again mean erythrocyte G6PD level was significantly lower ($P < 0.01$) in group B₁ and B₂ than those of group A₁ and A₂ (Figure 1).

Table-I : Age and BMI in different groups of subjects (n=120).

Groups	n	Age (yrs)	BMI (kg/m ²)
A ₁	30	49.80 \pm 3.52	23.55 \pm 0.74
A ₂	30	62.87 \pm 1.36	23.64 \pm 0.77
B ₁	30	50.70 \pm 3.35	23.88 \pm 0.76
B ₂	30	63.47 \pm 1.55	23.68 \pm 0.84

Statistical Analysis:

Groups	p value
A ₁ vs A ₂ vs B ₁ vs B ₂	0.001** 0.402 ^{ns}
A ₁ vs B ₁	0.315 ^{ns} 0.089 ^{ns}
A ₂ vs B ₂	0.116 ^{ns} 0.867 ^{ns}

Data are expressed as Mean \pm SD.

Group A = Non cataractous subjects (Control group).

Group A₁ = Presenile

Group A₂ = Senile

Group B = Cataractous patients (Study group).

Group B₁ = Presenile

Group B₂ = Senile

ns= Non significant ($p > 0.05$)

No control subjects were found G6PD deficient. Again prevalence of G6PD deficiency is higher in presenile than that of senile cataractous group but difference was not significant. (Figure 2)

8(26.7%) of the presenile cataractous subjects had erythrocyte G6PD level below <245 mU/10⁹ RBC but no deficient subjects were found in senile cataractous group in these ranges. However, 2 (6.7%) of senile cataractous subjects had erythrocyte G6PD level below <200 mU/10⁹ RBC but no deficient subjects were found in presenile cataractous group in these ranges (Figure 3).

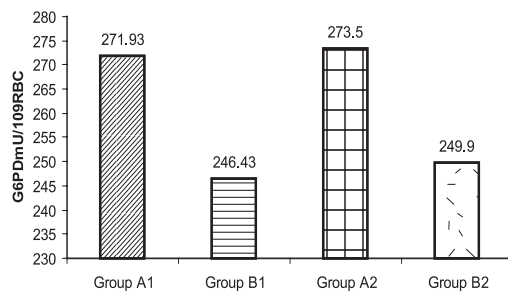


Figure 1: Mean erythrocyte G6PD level in different groups of subjects (n=120)

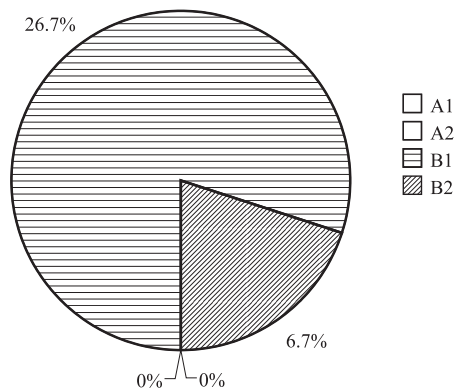


Figure 2 : Distribution of the subjects by G6PD deficiency in different groups (n=120).

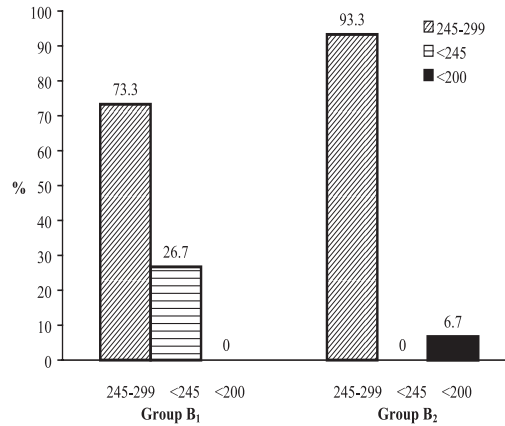


Figure 3 : Distribution of the subjects by erythrocyte G6PD level in cataractous groups (n=60).

Discussion

In the present study the erythrocyte G6PD status was studied in presenile and senile cataractous male patients. The values of erythrocyte G6PD level in noncataractous subjects in this study were almost similar to the findings of control subjects reported by various investigators of different countries ^{1, 11, 12}.

Again, mean erythrocyte G6PD level in cataractous patients were significantly lower than those of noncatarctous subjects which is also consistent with findings of other investigators ^{5,6,11-12}. In addition, the mean G6PD level was close to the lower limit of reference value.

In this study percentages of the erythrocyte G6PD deficiency in presenile and senile cataractous patients were also observed. Total 33.4% enzyme deficiency found in both the cataractous groups were almost similar to the findings reported by various investigators of different countries ^{6,11}. In addition, no G6PD deficiency was found in non cataractous healthy subjects.

Various researchers of different countries suggested that in cataract high molecular weight

proteins are formed following extensive oxidation which causes precipitation of lens proteins and thereby causing loss of transparency⁷.

It was also suggested that G6PD deficiency affects pentose phosphate pathway, resulting in inavailability of ribose for lens protein synthesis⁶.

Oxidation of ÉSH groups of Na⁺-K⁺ ATPase causes lenticular accumulation of Na⁺, Cl⁻, Ca⁺⁺ which leads to cellular hydration, Swelling, rupture and followed by opacity of lens ^{11,12}. In G6PD deficiency due to loss of NADPH and GSH production, there is increased susceptibility of the lens to oxidative insult and subsequent loss of lens transparency⁵.

In the present study, lowered erythrocyte G6PD level in presenile and senile cataractous patients may be suggestive of increased susceptibility of the lens to oxidative injury associated with deficiency of antioxidants in the ocular lens.

Conclusion

Therefore, this study concludes that G6PD deficiency may be a risk factor for development of cataract in presenile and senile age group.

Acknowledgement

Authors of this study are thankful to the of Department of Ophthalmology, BSMMU for their cooperation .

Author's affiliations

- *1. Farzana Yasmin, Lecturer Department of Physiology, Bangladesh Medical College, Dhaka. E mail:fyasminbmc@gmail.com
2. Noorzahan Begum, Professor Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Bangladesh, Email: noorzahan52@gmail.com
3. Sultana Ferdousi, Associate professor, Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Bangladesh, Email: sferdousiratna@gmail.com

**for correspondence*

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