Research Article

Screening of neonates for Glucose -6 - Phosphate Dehydrogenase deficiency in Adivasi area of Vasai Taluka

Raviraj Naik[1*], Sarita Dakhure[2], Rittu Chandel[3], Leela Abichandani[4]

[1,2]Assistant Professor, Department of Biochemistry, IIMSR, Warudi, Jalna, Maharashtra, INDIA.
[3]Assistant Professor, Department of Biochemistry, Dr. SGGS Medical College, Nanded, Maharashtra, INDIA.
[4]Professor and Head, Department of Biochemistry, Grant Medical College, Mumbai, Maharashtra, INDIA.
Email: raviraj_40@yahoo.com

Abstract

Introduction: Glucose – 6 – phosphate dehydrogenase (G-6-PD) is an enzyme that participates in the first step of Hexose monophosphate pathway of glucose metabolism. Deficiency of this enzyme is most common genetic disorder in India. Its deficiency causes hemolysis which eventually lead to acute haemolytic anemia and neonatal jaundice. The hemolysis in these deficient patients are triggered by bacterial, viral infections, drugs like aspirin and chloroquine, foods like fava beans Period from after birth to first 4 weeks is the neonatal period and the child is called a neonate. There is no cure for G-6-PD.Early detection and prevention of hemolytic episodes by avoiding the triggers is the only cure for this deficiency. **Aim:** To screen the neonates for glucose -6-Phosphate dehydrogenase deficiency so as to prevent the morbidity and mortality occurring due to this deficiency. **Material and Methods:** One Thousand neonatal blood sample were obtained from the Labour room, of the civil Hospital of vasai. The samples were analysed by Dye Decolourization method. **Results:** Of the 1000 samples, 10 was found to be G-6- PD deficient and 20 were found to be G-6-PD deficiency carriers. **Conclusion:** G-6-PD deficiency is common in Adivasi Population.G-6-PD deficiency testing should be done as a screening procedure at least in Adivasi residing areas as early diagnosis and prevention is the only way of treating this deficiency disorder and avoiding its complications.

Key-words: Glucose – 6 – phosphate dehydrogenase (G-6-PD) , deficiency, neonate, hemolysis, screening.

[*]**Address for Correspondence:**
Dr. Raviraj Naik, Assistant Professor, Department of Biochemistry, IIMSR,Warudi, Jalna, Maharashtra, INDIA.
Email: raviraj_40@yahoo.com

Received Date: 07/07/2015 Revised Date: 16/07/2015 Accepted Date: 18/07/2015

Access this article online

Quick Response Code:

Website: www.statperson.com

DOI: 01 August 2015

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is most common of all clinically significant enzyme defects.[1] This deficiency is X-linked which occurs due to mutations in coding region of G-6-PD gene. Allmost all of the 140 different mutations known are *single missense point* mutations. Exact incidence not known but several studies have reported incidence between 1-20%.[2] G-6-PD catalyzes first step in HMP shunt. NADPH produced keeps Glutathione in its reduced state which protects red blood cells from oxidativedamage;[3] so in G-6-PD deficiency reduced glutathione will not be formed and red blood cells will not be protected from oxidative stress which will lead to haemolytic crisis on exposure to different triggers.

Triggers for haemolytic crisis:-Infections [viralandbacterial],Drugs[Aspirinandchloroquine],Chemicals[naphthalene[4]] and certain foods [Fava beans[3]]. Early detection and prevention of haemolytic episodes by avoiding the triggers in newborn babies is the only cure.

MATERIAL AND METHODS

Site of collection of blood sample: From the labour room of rural hospital vasai, one thousand neonatal cord blood samples were collected in ethylene diamine tetracetic acid (EDTA) bulb. These samples were then analysed in the laboratory of Biochemistry department at GGMC, Mumbai; by the Dye decolorization method(Qualitative).

Principle: G-6-PD present in red cell haemolysate act on glucose-6-phosphate and reduces NADP to NADPH which with the help of PMS reduces blue coloured 2,6

How to site this article: Raviraj Naik, Sarita Dakhure, Rittu Chandel, Leela Abichandani. Screening of neonates for Glucose -6 -Phosphate Dehydrogenase deficiency in Adivasi area of Vasai Taluka. *International Journal of Recent Trends in Science and Technology* August 2015; 16(1): 20-21 http://www.statperson.com (accessed 02 August 2015).

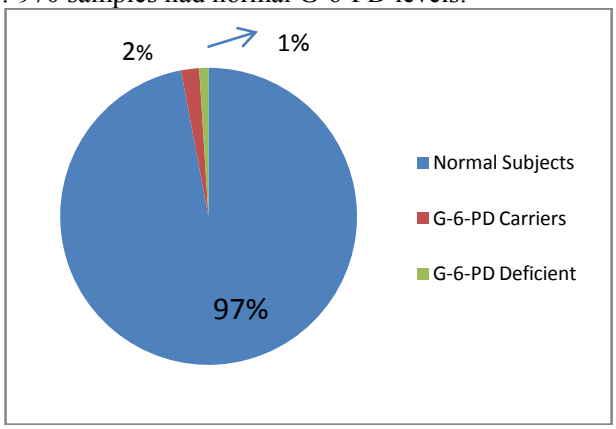
Dichlorophenol Indophenol into colorless form. Rate of decolorization is proportional to enzyme activity.
Reaction can be represented as : -
1]Glucose-6-phosphate + NADP --------> 6-Phosphogluconic acid + NADPH.
2]NADPH + 2,6-Dichlorophenolindophenol --------> NADP + Reduced 2,6-Dichlorophenolindophenol.
INTERPRETATION:- Time taken for colour change from initial deep blue to reddish purple is noted.

- Follow up to a maximum of 6 hours with 30 minute interval.
- Decolourization Time :- 1} Normal subjects : 30 -60 minutes.

2} G-6-PD deficient subjects : 140min - 24 hrs.
3} G-6-PD carriers :- 90 min.

OBSERVATION AND RESULTS

Total number of samples collected and examined = 1000.
Out of 1000 samples :- 10 sample was found to be G-6-PD deficient.{male newborn}
:-20 samples were found to be G-6-PD carriers.{female newborn}
:-970 samples had normal G-6-PD levels.



DISCUSSION

The gene for G-6-PD deficiency is located on terminal region of the long arm of the X-chromosome at position q28. It is a X-linked condition which usually manifest in males carrying mutant gene. The phenotype in females may be normal homozygote, G-6-PD deficient homozygote or heterozygous.[5,6] Random X -chromosome inactivation result in two RBC populations in female heterozygotes.[7]Several variants of G-6-PD deficiency were encountered in different regions of India during extensive screening programmes. Different groups of researchers have evaluated the scope of G-6-PD deficiency in different regions of country. WHO has quoted the incidence of G-6-PD deficiency in India from 0.2-19%. In other studies e.g; Deshmukh *et al.* study and Nishi Madan *et al.* study incidence of G-6-PD deficiency was 6% and 1.37% respectively.[8]Various incidences of G-6-PD deficiency in various subjects may be due to differences in Screening test solution and associated incidence of Malaria and Haemoglobinopathies in various regions. [9] Calculating the sensitivity and specificity of this screening method is out of scope of this study, but based on previous reports the neonatal cord blood G-6-PD deficiency screening had acceptable sensitivity{85.7} and high specificity{98.1%}. [10]

CONCLUSION

The early characterization of G-6-PD deficiency provides an etiological diagnosis for neonatal jaundice, as well as the opportunity to give the newborn's family information concerning the prevention of complications and mortality associated with G-6-PD deficiency.Considering its high incidence in our country, a neonatal screening programme for G-6-PD deficiency should therefore be taken into account in the National Health Schemes.

REFERENCES

1. World Health Organization. Report of the Working Group - Glucose-6-phosphate dehydrogenase Deficiency. Bull World Health Organ 1989;67: 601-611.
2. Handa R, Singh N, Chander J. Erythrocytic G-6-PD deficiency in Bania community in Punjab. J Associated physicians India 1992 Jan;40[1]:13-14.
3. DK, Neonatal Hyperbiblirubinemia. N Eng J Med 2001: 344:581-590.Dennery PA, Selden D S, Stevenson
4. Cappelini MD, Fiorelli G[January 2008]. G-6-PD deficiency. Lancet 371(9606):64-74.
5. Beutler E. The genetics of Glucose-6-phosphate dehydrogenase Deficiency. Semin Hematology 1990;27: 137-164
6. Luzzato L. Glucose-6-phosphate dehydrogenase Deficiency and hemolytic anemia .In Nathan DEC, Oski FA, eds. Hematology of Infancy and childhood. Philadelphia, PA; WB Saunders Co.,1993:674- 695
7. Beutler E, Yeh M, Fairbanks VF. The normal human female as a mosaic of X chromosome activity. Studies using the gene for G-6-PD deficiency as a marker. Proc Natl Acad Sci USA 1969;48:9-16.
8. World Health Organization. Standardization of procedures for the study of G-6-PD deficiency.Geneva.1967.
9. Nishi Madan, Talwar n, Maheshwari A, Sood SK. Incidence of G-6-PD deficiency in a hospital population of delhi. Indian J Med Res 1981;73:425-429.
10. Kher M, Solanki BR, Parande C, Junnarkar RV. G-6-PD deficiency at Nagpur and in surrounding areas. Indian Medical Gazette 1967; September; 34-39.

Source of Support: None Declared
Conflict of Interest: None Declared