ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 49. 23
PubMed - National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 3, Issue – 6, December - 2018, Page No. : 177 – 180

Prevalence of G6PD deficiency in patients of a Tertiary Care Hospital, Bhubaneswar, Odisha.

[1]Karmajeet Rath, Associate professor, Department of Pharmacology, IMS & SUM Hospital, Bhubaneswar, Odisha
[2]Suman Mandal, Post Graduate student of MD Pharmacology, Department of Pharmacology, IMS & SUM Hospital, Bhubaneswar, Odisha
[3]Sarthak Ranjan Nayak, Associate professor, Department of Biochemistry, IMS & SUM Hospital, Bhubaneswar, Odisha
Corresponding Author: Suman Mandal, Post Graduate student of MD Pharmacology, Department of Pharmacology, IMS & SUM Hospital, Bhubaneswar, Odisha

Type of Publication: Original Research Paper
Conflicts of Interest: Nil

Abstract

Introduction: G6PD is extremely essential housekeeping gene and its deficiency increases haemoglobin [Hb] vulnerability, leading to Hb instability. Various factors can trigger the sign and symptoms of G6PD deficiency. Early recognition of G6PD deficiency can prevent serious complications.

Objectives: To find occurrence of G6PD deficiency in 'symptomatic patients' admitted in IMS & SUM Hospital & also to find which gender has more prevalence of G6PD deficiency.

Materials and Methods: This study was carried out among 183 patients (admitted in Hospital) at Biochemistry Department of IMS & SUM Hospital between September 2017 to August 2018.

Results: Out of 183 patients, 90 were males, and 93 were females, and 15 patients showed deficiency of G6PD (prevalence 8.19%). Out of 15 G6PD deficient patients, 12 were within 10 yrs age, and 3 were between 21- 30 yrs age.

Conclusion: G6PD deficiency should be looked for all suspected children from the high prevalence area of Odisha before giving them any oxidant drugs. Government should take initiatives to save life from the fatal complications of G6PD deficiency.

Keywords: G6PD deficiency, symptomatic, prevalence.

Introduction

Glucose – 6 – phosphate dehydrogenase (G6PD) is extremely essential housekeeping gene expressed in all the tissues. Mutants having 100% absence of G6PD enzymes will be unsuited with life and are thus not reported [1]. G6PD deficiency causing gene found in approx 7.5% of world's population and highest proportion (35%) found in parts of Africa & lowest (0.1%) proportion found in Japan & parts of Europe [2]. The allocation of G6PD deficient population geographically overlaps very much with the prevalence of malaria suggesting that G6PD deficiency may take part in a defensive role against malaria [2]. Due to oxidative harm, G6PD deficiency increases haemoglobin (Hb) vulnerability, leading to Hb instability & precipitation of Heinz bodies [3]. It is genetically inherited sex linked defect. The gene encoding G6PD located on the X-chromosome (Xq28) contains 13 exons & is more than 20 kb in length [4]. The occurrence of G6PD deficiency in any particular population is determined by the number of deficient male due to its sex

linked condition [5]. Stress, various infections, various chemicals in medicines & foods can trigger various sign & symptoms of G6PD deficiency. So, early recognition of G6PD deficiency can help to prevent serious complications [6]. G6PD enzymes catalyze the first step in the HMP pathways converting G6PD to 6 phosphogluconolactone & reducing the co-factor nicotinamide adenine dinucleotide phosphate (NADP) to NADPH [7]. Glutathione (GSH) is important for protecting red cells from oxidative harm [8].

This study tries to bridge the gap of knowledge towards health care.

The study was aimed

1. To find occurrence of G6PD deficiency in 'symptomatic Patients' admitted in IMS & SUM Hospital.
2. To find which Gender has more prevalence of G6PD deficiency.

Material And Methods

Haemoglobin estimation was carried out by calorimetric cyanmethemoglobin method [9]. G6PD deficiency was screened using the fluorescence spot method [10], which is an accepted protocol worldwide. This method depends upon the fluorescence of NADPH formed through the action of G6PD when illuminated under UV light [11]. Informed consent was obtained from all subjects and 5ml blood was withdrawn by aseptic technique in K3 EDTA vacutainer [12]. The period of the study was 1 year. All the samples were checked for G6PD activity by methemoglobin reduction test. After carrying out these procedures, all samples were interpreted as G6PD present, which demonstrate clear red colour, and as G6PD deficient, which demonstrate brown colour [12]. G6PD enzyme activity of ≥6.97 U/g Hb was regarded as normal, while value <6.97 U/g Hb were regarded as deficient at 37°C [12]. Hb <12g/dl were considered as anaemic [13].

Observations And Results

Table 1: Prevalence of G6PD deficiency.

	Overall prevalence	Sex distribution		Association
		Male	Female	Pearson's Chi-square test Between male & female
Total subjects	183	90	93	χ^2= 2.70.
Deficient subjects	15	9	6	And p value=0.1 Non-significant
Prevalence	8.19%	10%	6.45%	

Table 2: Age-wise distribution of patients with or without G6PD deficiency.

Age Range	Without G6PD deficiency	Percentage	With G6PD deficiency	Percentage
0-10	99	58.93%	12	80%
11-20	3	1.78%	0	0
21-30	30	17.85%	3	20%
31-40	15	8.93%	0	0
41 onwards	21	12.5%	0	0
Total	168	100%	15	100%

Table 3: Anaemic prevalence among population [13].

Total number of Patient	Male	Female
Normal (Hb% ≥12gm/dL)	75	72
Anaemic (Hb% <12gm/dL)	15	21
Mild (Hb% 11-11.9gm/dL)	0	3
Moderate (Hb% 8-10.9gm/dL)	9	12
Severe (Hb% <8gm/dL)	6	6

Table 4: Drugs to be avoided by persons with G6PD deficiency (Beutler Ernest, 2011) [12].

Acetanilide	Dapsone	Diaminodiphenylsulfone	Cotrimoxazole
Furazolidone(Furoxone)	Glibenclamide	Isobutyl nitrite	Methylene Blue
Naphthalene	Niridazole	Nitrofurantoin	Nalidixic acid
Phenazopyridine	Phenylphdrazine	Primaquine	Sulfamithoxyzole
Sulfanilamide	Sulfapyridine		

Table 5: Drugs that carry possible risk of haemolysis in G6PD deficient individuals.

Acetaminophen	Acetylsalicytic acid	Ascorbic acid	Aminopyrine
Chloramphenicol	Proguanil	Chloroquine	Ciprofloxacin
Colchicine	Isoniazid	L-Dopa	Norfloxacin
Phenytoin	Quinine	Streptomycin	Sulfadiazine
Vitamin K			

Table 1 shows the prevalence of G6PD deficiency among the patients of IMS & SUM Hospital was 8.19% and the G6PD deficiency was non-significantly concentrated in males. Table 2 shows age-wise distribution among the

patients. Table 3 shows anaemic prevalence among the patient population.

Table 4 & 5 shows the list of drugs which are avoided in G6PD deficient patients.

Discussion

Our country is endemic region for malaria. 1.5 to 2 million confirmed cases are reported annually. Also, infectious diseases contribute about 30% of the disease burden in India [12]. In management of these diseases many drugs are used, which may cause severe haemolysis (Table 4 and 5) and leads various degree of morbidity and mortality in G6PD deficient individuals. Prevalence of G6PD deficiency in our country varies between 0-28% [15]. Variation in the results can also be due to racial, ethnic, and geographic distribution [16]. In Odisha, the prevalence of G6PD deficiency varied from 1.3 to 17.4% [14]. In our study, we found prevalence of G6PD deficiency was 8.19%. Our finding was in accordance with Balgir RS [17] who shows prevalence of 4.3-17.4% in Odisha and closely similar with the study done by Ramadevi et al [18] (7.8%) and Shankarkumar [19](9%). In G6PD deficiency disease males are predominantly involves, since disease is X- linked recessive [12]. In our study G6PD deficiency was more concentrated in males (10%). But Wang et al indicated that in his study sex was not a significant predictor associated with actual G6PD enzyme level [10].

So, in area where prevalence of G6PD deficiency is high, before giving drugs (mentioned in table 4 and 5), it is necessary to investigate the patient for G6PD deficiency.

Conclusion

Because of high prevalence of G6PD deficiency in some region of Odisha, all suspected children from those region should be investigated for G6PD enzyme deficiency before administration of any Anti-malarial or Antibacterial drugs or Oxidants. To achieve this level, government should adopt some effective policies; like start of educational awareness program to ensure complete avoidance of oxidant drugs and prevention of infections in previously diagnosed cases. Government should include hepatitis A and B vaccination in National Immunization schedule. Government should establish nationwide program for screening of infants born in high prevalence area. For it, government should start facility of investigating the disease at all community health centre (CHC) level. If, limited resources come in way, direct above policies towards only suspected children.

References

1. Patel H, Patel N, Maniyar A, Gandhi K, Patil R. A study of G6PD deficiency in neonatal hyperbilirubinemia; IJMSPH: 2015, vol 4, issue 5, 621-623.

2. Ruwende C, Khoo SC, Snow RW, Yates SN, Kwiatkowski D, Gupta S, et al. Natural selection of hemi and heterozygotes for G6PD deficiency in Africa by resistance to severe malaria. Nature 1995; 376: 246-249.

3. Ronquist G, Theodorsson E. Inherited, non-spherocytic haemolysis due to deficiency of G6PD. Scand J Clin Lab Invest 2007; 67: 105-111.

4. Takizawa T, Huang IY, Ikuta T, Yoshida A. Human G6PD: Primary structure and cDNA cloning. Proc Natl Acad Sci USA, 1986; 83; 4157-4161.

5. Mallouh AA, Imseeh G, Abu- osba YK, Hamdan JA. Screening for G6PD can prevent severe neonatal jaundice. Ann Trop Paediatric 1992; 12: 391-395.

6. Shah I, Jarullah J, Jaouni SA, Jamal MS, Jarullah B. Higher prevalence of G6PD deficiency in tribal population against urban population: a signal to natural selection; Biomedical Research: 28(1): 385-388.

7. G6PD deficiency. WHO Bulletin OMS 1989.

8. Beutler E, G6PD deficiency. Blood 1994; 84: 3613-3636.

9. Beutler E, West C: Clinical Chemistry: 1984, 30(6), 871-874.

10. Wang FL, Boo NY, Ainoon O, Wong MK; Singapore Medical Journal, 2009, 50(1), 62-67.

11. Sivaraj B, Radha R, Vidya C, Sanil R; Prevalence of G6PD deficiency among the Badagas of Nilgiris; AJBPR: issue 1, vol-5: 2015: 295-299.

12. Singh UR, Singh AB, Singh Uike GP. Comparison of prevalence of G6PD deficiency in General Population and admitted symptomatic children of GM Hospital associated to SSMC, Rewa, MP India; IJBARD, December 2014: vol4, issue 1, 61-69.

13. WHO, UNICEF, UNU. Iron deficiency anaemia: assessment, prevention and control, a guide for programme managers. Geneva, World Health Organization, 2001.

14. Report of the ICMR Multicentric Task Force on Intervention programme for nutritional anemia and haemoglobinopathies amongst some primitive tribal populations of India. New Delhi: Indian Council of Medical Research; 2010.

15. Basin MK, Walter H. Genetics of castes and tribes in India. Delhi: Kamla- Raj Enterprises;2001.

16.Menon MJ, Gahine R, Hussain N, Gupta Y. Prevalence of G6PD deficiency among sikle cell patients of Chhattisgarh region. IJSS; Nov 2016; Vol 4;Issue 8.

17. Balgir RS. Do tribal communities show an inverse relationship between sickle cell disorders and glucose-6-phosphate dehydrogenase deficiency in malaria endemic areas of central-Eastern India? Homo 2006; 57:163-76.

18. Ramadevi R, Savithri HS, Devi AR, Bittles AH, Rao NA. An unusual distribution of G6PD deficiency of south Indian newborn population. IJBB 1994; 31: 358-360.

19. Shankarkumar U. A correlative study of HLA, sikle cell gene and G6PD deficiency with splenomegaly and malaria incidence among Bhils and Pawra tribes from Dhadgon, Dhule, Maharastra. Stud Tribes Tribals 2003; 1: 91-4.