ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
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 – 8, Issue – 2, March – 2023 , Page No. : 167 – 172

G6PD deficiency in neonatal jaundiced babies attending teaching institution.

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Citation this Article: Aakriti Khajuria, Sunny Babber, Ravinder K Gupta, Muskaan Nargotra, "G6PD deficiency in neonatal jaundiced babies attending teaching institution", IJMSIR- March - 2023, Vol – 8, Issue - 2, P. No. 167 – 172.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Neonatal jaundice is a common finding in newborns leading to high bilirubin levels in newborn which if not treated can lead to kernicterus and neonatal death. Neonatal jaundice can be pathological or physio logical. Glucose-6-phosphate dehydrogenase deficiency (G6PD) deficiency is an important cause of pathological jaundice which causes haemo lytic anaemia, hemo globin emia and haemo globinuria and is seen mainly in males due to X-linked inheritance.

Aim and objective: To study the G6PD status in neo natal jaundice and compare the clinical findings of neo natal jaundice cases with and without G6PD deficiency.

Materials and methods: This prospective and comparative study was conducted in Acharya Shri Chander College of Medical Sciences (ASCOMS) where 132 neonatal jaundice patients whose age varied from 2 to 12 days were taken for study over a period of one year. All these neonates were screened for G6PD deficiency. Data were collected regarding age of onset of jaundice, birth weight, gestational age, gender of neonate, family history of G6PD deficiency, duration of double photo therapy needed and investigations like Total serum bilirubin, white blood cells (WBC) count, red blood cells (RBC) count, Platelet count, haemo globin levels, reticulocyte counts and haema to crit percentage. All the findings were compared in the neonatal jaundice patients with and without G6PD deficiency.

Result: Out of 132 cases of neonatal jaundice, G6PD deficiency was present in 3.79% cases. On comparing cases of neonatal jaundice with and without deficiency of G6PD, we found that G6PD was present mainly in males and they had statistically significant higher total serum bilirubin levels. These G6PD deficient cases of neonatal jaundice needed longer duration of phototherapy and had decreased haemo globin levels with increased reticulo cyte counts ($p<0.05$).

Conclusion: Therefore, this study concludes that the G6PD deficiency is one of the important common cause of neonatal jaundice especially in case of male patients and therefore early screening should be done**.**

Keywords: Glucose – 6 - phosphate dehydrogenase, jaundice, neonates, phototherapy.

Introduction

Neonatal hyperbilirubinemia is an important problem in the first week of life. During the first week of life, if total

serum bilirubin (TSB) rises above the 95th percentile for age it is considered as hyperbilirubinemia [1].

Up to 60% of term neonates and up to 80% of preterm neonates develop clinical jaundice in first week of life [2]. Jaundice leads to high bilirubin levels which may be toxic to the developing brain and central nervous system [3]. All neonates should be screened and visually inspected for neonatal jaundice every 12 hour in the initial 3 to 5 days of life.

The newborn must be examined in good daylight [4]. Neonatal jaundice can be physiological or patho logical. The physio logical causes are more common and is of conjugated variety and improves within 2 weeks mostly [5]. Various causes of neonatal hyperbilirubinemia are physio logical jaundice, inadequate feeding, pre maturity, breast milk jaundice & various pathological causes are G6PD deficiency, Rh in comp ability, thalassemia, ABO in comp ability, biliary atresia, neo natal sepsis, poly cythaemia, neonatal hepatitis, hypo thyroidism and rare causes like Gilbert's syndrome [6].

The glucose -6- phosphate (G6PD) deficiency is one of the important causes of neonatal jaundice. It is a X-linked disease thus more common in males. It is one of the most common inherited enzymopathy [7]. The enzyme G6PD normally forms an integral part of pentose mono phosphate shunt.

Normally G6PD acts as an anti-oxidant and decreases free radical damage in the red blood cell. It thus protects haemo globin from getting oxidized. In cases of G6PD deficiency, there is no one to protect haemo globin from oxidation and thus haemo globin gets oxidized to metha emo globin and gets denatured and forms inclusion bodies known as Heinz bodies [8].

G6PD deficiency can occur on exposure to specific food fava beans or certain medications. Sulph on amides like sulpha metho xazole, antimalarials like primaquine and analgesics like aspirin are some of the drugs that have been found to cause oxidative stress and haemo lysis in patients with glucose-6- phosphate deficiency [9]. Due to this intravascular haemo lysis free haemo globin comes in blood and urine, thus making hemo globin emia and haemo globinuria as the main symptoms of G6PD deficiency.

Other findings are neonatal jaundice, spleno megaly and presence of Heinz bodies and bite cells on peripheral blood smear [10]. Family history and history of exposure to oxidative stress play a vital role in investigation other than the symptoms whereas con formation is done after techniques like molecular gene analysis or quantitative enzyme assay. Management is mainly supportive. Acute haemo lysis may be self-limiting or requiring blood transfusion.

Neonatal hyper bilirubinemia may sometimes require phototherapy or exchange transfusion in order to prevent kernicterus and other damaging effects on central nervous system [11].

Keeping this in background, this study was focused on finding the prevalence of G6PD deficiency in neonatal jaundice patients and then compare the findings in neonates having jaundice with and without deficiency of G6PD.

Aim of the study

To study the G6PD status in neonatal jaundice among neonates attending a tertiary care hospital in Jammu and to compare the findings in the patients of neonatal jaundice with and without G6PD deficiency.

Materials and methods

The study was conducted after getting approval from the Institute of Independent Ethical Committee with reference no. ASCOMS/ IEC/ RP&T/ 2020/ 380 dated 25/ 07/ 2020 in department of Pediatrics from Jan 2021 to Dec 2021 at Acharya Shri Chander College of Medical

Sciences and Hospital (ASCOMS & H) in Jammu over a period of one year. A total of 200 neonates with hyper bilirubinemia were admitted to our hospital. After using inclusive and exclusive criterion, we selected a sample size of 132 neonates (59 females and 73 males) at 95% confidence interval and 5% margin of error and by using 80% power of the test by G*POWER software.

Inclusive criterion

neonates with hyperbilirubinemia in age group of 2-12 days.

Exclusive criterion

neonates with hyperbilirubinemia not falling in the age group of 2-12 days.

Detailed history was taken from the parents of neonates and proper physical examination was done. Data was collected regarding Gestational Age, Gender, Age at onset of jaundice, Family history of G6PD deficiency and Birth weight.

Proper investigations were performed which included total serum bilirubin, haemo globin, reticulocyte counts, haema to crit, WBC, RBC and Platelets count. All the cases of neonatal jaundice were screened for G6PD deficiency by performing standard laboratory tests. The neonates with severe hyperbilirubinemia were given double phototherapy and its duration was recorded. After phototherapy, all the new-borns got cured and none of them needed exchange transfusion.

Statistical methods

Statistical analysis was done by using IBM SPSS VERSION 21. In this paper, we express the qualitative data in terms of percentages and was compared using non parametric tests such as chi-square test, adjusted chi-square test and fisher's exact test and the normality of data was checked by using box and whisker plot. The quantitative data was expressed in terms of mean and standard deviation and was compared by using parametric test i.e., student t-test (independent) and a value of $p<0.05$ was considered as statistically significant otherwise non-significant.

Result

Overall, 132 new-borns with neonatal jaundice were admitted to our neonatal ward during our study period. Out of these 132 new-borns, 5 had neonatal jaundice along with G6PD deficiency and 127 had neonatal jaundice with some other cause showing that prevalence of G6PD in patients of neonatal jaundice in our selected population is 3.79%.

On comparing the findings between the neonatal jaundice cases with and without G6PD deficiency, a statistically significant difference ($p<0.05$) is seen in relation to total serum bilirubin, haemo globin, reticulocyte counts, duration of double phototherapy and gender.

Table 1: Comparison of neonatal jaundice cases without and with G6PD deficiency

Variables	Neonatal jaundice (n=132)		
	Without G6PD deficiency(n=127)	With G6PD deficiency (n=5)	p-value
Age at onset of jaundice (in days), (Mean ±SD)	4.28 ± 1.80	5.32 ± 2.26	0.2113
Age group (in days)			
2-4 days	38 (29.92 %)	1 (20 %)	
5-9 days	65 (51.18 %)	3 (60 %)	

9-12 days	24 (18.89 %)	1 (20 %)	0.8887
Gestational age (in weeks) (Mean ±SD)	37.12 ± 2.26	37.52 ± 2.31	0.6987
Birth weight (in kg) (Mean ±SD)	2.73 ± 0.43	2.54 ± 0.36	0.3320
Gender, n (%) Female Male	 59 (46.46 %) 68 (53.54 %)	 0 (0) 5 (100 %)	 0.0486*
Family history	12 (9.44 %)	2 (40 %)	0.1511
Duration of double phototherapy (in days)	2.54 ± 1.56	4.02 ± 2.42	0.0437*

***Statistically significant**

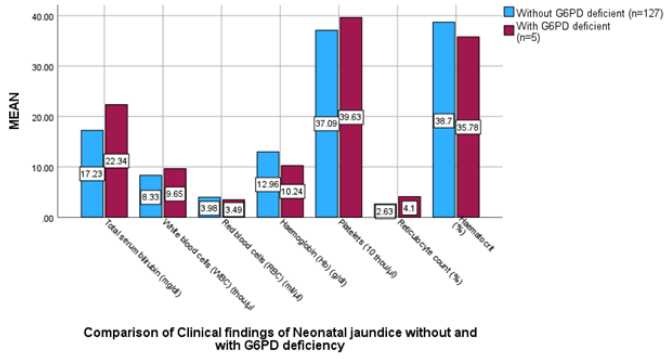
On comparing the cases of neonatal jaundice with and without G6PD deficiency, we find that there is no statistically significant difference ($p > 0.05$) in terms of Age at onset of jaundice, Gestational age, Birth weight and Family history while a statistical significant difference ($p < 0.05$) was seen in relation to Gender and Duration of double phototherapy given to the newborns as shown in table 1.

Table 2: Comparison of Clinical findings of Neonatal jaundice without and with G6PD deficiency

Variable	Without G6PD deficient (n=127) (Mean ±SD)	With G6PD deficient (n=5) (Mean ±SD)	p-value
Total serum bilirubin (mg/dl)	17.23 ± 2.45	22.34 ± 4.52	0.0001**
White blood cells (WBC) (thou/µl)	8.33 ± 2.87	9.65 ± 3.12	0.3163
Red blood cells (RBC) (mil/µl)	3.98 ± 0.86	3.49 ± 0.59	0.2099
Hemoglobin (Hb) (g/dl)	12.96 ± 2.32	10.24 ± 1.86	0.0108*
Platelets (thou/µl)	370.86 ± 120.56	396.32 ± 123.86	0.6443
Reticulocyte count (%)	2.63 ± 1.54	4.10 ± 2.12	0.0401*
Haematocrit (%)	38.70 ± 5.32	35.78 ± 4.43	0.2286

**** Highly statistically significant, *statistically significant**

Graph 1:



Comparison of Clinical findings of Neonatal jaundice without and with G6PD deficiency

In table 2, we compared the different Clinical findings of Neonatal jaundice without and with G6PD deficiency and we found that a statistically significant difference was seen in terms of total serum bilirubin, haemo globin and reticulocyte count where as a non-statistically significant difference was seen in other clinical findings such as WBC, RBC, Platelets and Haematocrit. Here, we can also appreciate that the total serum bilirubin and reticulocyte count is increased in case of neonatal jaundice patients with deficient G6PD whereas the haemo globin level were decreased in them due to haemo lytic anaemia caused by G6PD deficiency.

Discussion

Many studies have been done till now and almost all studies show G6PD deficiency as common cause of neonatal jaundice. This study shows that prevalence of

G6PD deficiency in neonates with hyper bilirubinemia in this population is 3.79%. Whereas a study done by Deshmukh Prabhakar Dattatray Rao [12] shows that the prevalence of G6PD Deficiency and its association with neonatal jaundice in babies born were found to be 1.8%. A study done in Indonesia by Dewi A. Wisnumurti [13] shows prevalence of G6PD deficiency in neonates with hyper bilirubinemia in Indonesian population was 1.72%. A similar study done by Mur Chana Khound [5] shows that G6PD deficiency contributes to 8% of pathological causes of neonatal hyperbilirubinemia.

Our results show significantly high total serum bilirubin in G6PD deficient neonates with jaundice. Similar to our results, a study by Hasan M. Isa [14] also shows that patients of neonatal jaundice with G6PD deficiency have significant higher levels of bilirubin as compared to the ones without deficiency of G6PD.

The present study shows male gender as risk factor for G6PD deficiency. A study by MD Cappellini [15] also shows males as risk factor.

In present study we didn't find any significant difference in presence of family history of G6PD deficiency whereas, a study by Zeinab A Kasemy [16] shows that family history of G6PD deficiency was significant risk factor for G6PD deficiency.

Similar to our results, a study by Richard O. Francis [17] showed that haemo globin levels decreased and reticulocyte count increased in patients of neonatal jaundice with G6PD deficiency.

Conclusion

The results show that the prevalence of G6PD deficiency in new-borns with neonatal jaundice is 3.78%. male gender of newborn poses a risk for G6PD deficiency. There is high total serum bilirubin in G6PD deficient neonates with jaundice and these newborns require more duration of double phototherapy. Results also showed that haemo globin levels decreased and reticulocyte count increased in patients of neonatal jaundice with G6PD deficiency.

Therefore, we conclude that the G6PD deficiency is one of the important common cause of neonatal jaundice especially in case of male patients and therefore early screening should be done in neonates and treatment must be done accordingly.

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