DOI: https://doi.org/10.54393/pbmj.v5i1.284

PAKISTAN BIOMEDICAL JOURNAL

https://www.pakistanbmj.com/journal/index.php/pbmj/index

Volume 5, Issue 1 (January 2022)

Original Article

Role of Glucose Phosphate Deficiency in Neonatal Hyperbilirubinemia Among Local Population of Pakistan

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ARTICLE INFO

Key Words:
Neonates, Deficiency, Hyperbilirubinemia

How to Cite:
Shakoor, A. ., Bangash, S. ., & Hussain, S. . (2022). Role of Glucose Phosphate Deficiency in Neonatal Hyperbilirubinemia Among Local Population of Pakista: Glucose Phosphate Deficiency in Neonatal Hyperbilirubinemia. Pakistan BioMedical Journal, 5(1), 160–163. https://doi.org/10.54393/pbmj.v5i1.284

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ABSTRACT

Due to the prevalence of new-born jaundice (65%) and neonatal hyperbilirubinemia (85%) in the first 24 hours of life, Objectives: To investigate G6PD insufficiency in neonates Methods: The Punjab Health Department conducted a descriptive study from July to December 2021. The hospital's ethics committee approved this probe. The research included 197 patients among which there were infants aged 0-20 days. A survey was used to collect data. Gender, gestational age, age of presentation, and length of hospitalisation were all noted. Results: The study included 197 patients, 61 of whom were female and 136 males. Participants in this research had bilirubin levels between 10 and 30 mg/dl. The maximum age in this study was 18 days, while the minimum was a new-born infant. G6DP insufficiency was seen in 21 of the 197 patients. 176 individuals had normal G6PD levels. Conclusion: The study found that G6PD deficiency is common in new-borns. Severe hyperbilirubinemia has several risk factors.

INTRODUCTION

Neonatal hyperbilirubinemia occurs in 65 percent of full-term new-borns and 85 percent of preterm babies within the first 24 hours of life. Symptomless G6PD deficiency is common. Neonates with symptoms present to the hospital with new-born jaundice and/or acute haemolytic anaemia. Jaundice frequently begins at the same time or somewhat earlier than physiological jaundice [1,3]. As mentioned above, the pentose monophosphate shunt contains the G6PD enzyme. As a result of this enzyme, adenine dinucleotide (ADP) is converted into nicotinamide (NADP) (NADPH). Defending glutathione against oxidative destruction is a key role of NADPH. Oxidative stress affects erythrocytes because they are the only cells that can synthesise NADPH [4].Around 400 million people lack glucose-6-phosphate dehydrogenase (G6PD). The illness is more common among African, Asian, and Mediterranean populations. It is an X-linked recessive condition that runs in families. There are around 300 G6PD deficiency variations known thus far. A G6PD deficiency decreases malaria risk, possibly due to the disease's duration and frequency [5]. G6PD deficiency is the most prevalent red cell enzymopathy, affecting an estimated 400 million people globally. One study found that 4.9 percent of the world's population is G6PD deficient [6]. Immediate postnatal hyperbilirubinemia is associated with G6PD deficiency. Hyperbilirubinemia can cause lifelong brain damage or kernicterus [7]. Recent research suggests that rather than increased haemolysis, new-born hyperbilirubinemia is caused by a hepatic bilirubin metabolism deficit. A mutation in the UGT1A1 gene, either in the promoter or codon area of G6PD, can produce Gilbert-like syndrome [8].

METHODS

During the period July 2021 to December 2021, the Punjab Health Department conducted a descriptive study on its own. The hospital's ethical committee approved permission for this probe to be carried out, and the

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investigation began. Patients from a total of 197 different hospitals took part in the study. The research included the recruitment of new patients ranging in age from 0 to 20 days. A survey was used to collect the information. Patients' gender, gestational age, date of admission, and duration of stay were reported. Fluorescence spot testing was used to conduct the G6PD test (1 mL of whole blood in ethylenediamine tetra acetic acid tube). In our study, the bilirubin standard value was 10 to 30 mg/dl.The data was collected and analysed using Excel(2017).

RESULTS

There were 31 female infants (30.96%) and 68 male neonates in the 98-person sample(69.03%). Participants in this research had bilirubin levels between 10 and 30 mg/dl when recruited. The study's oldest participant was 18 days old, while the youngest was a new-born infant. 88 of the 98 individuals were normal at the time of selection, with just 11 G6DP deficient. 88 out of 90.345 patients had normal G6PD levels. Out of 11 patients (10%), just one was female, while the other ten were all male(90.47%).

No	Age (days of life)	Sex	G6PD level normal= 1,deficient= 2
1	4	m	1
2	3	m	1
3	18	m	1
4	8	m	1
5	2	m	2
6	8	f	1
7	5	m	1
8	4	f	2
9	8	m	1
10	5	f	1
11	4	m	1
12	5	m	1
13	5	m	1
14	5	m	1
15	6	m	1
16	13	m	1
17	2	m	1
18	2	f	1
19	6	m	2
20	4	m	2
21	4	m	2
22	4	m	2
23	3	m	1
24	10	m	2
25	10	m	1
26	4	m	1
27	5	m	1
28	5	m	1
29	3	f	1
30	1	m	2
31	6	m	1
32	5	f	1
33	2	m	1
34	8	m	2
35	5	m	1
36	5	f	1
37	3	m	1
38	10	m	2
39	10	m	2
40	6	f	1
41	5	m	1
42	3	f	1
43	7	m	1
44	5	m	2
45	3	m	2
46	4	m	1
47	5	m	2
48	3	f	1
49	7	m	2
50	8	m	1
51	5	m	1
52	10	m	2
53	4	m	1
54	5	m	1
55	4	m	2
56	4	f	2
57	8	m	1
58	4	m	2
59	3	f	1
60	4	m	1
61	5	f	1
62	6	f	1
63	8	m	1
64	7	m	1
65	5	m	1

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66	2	m	1
67	2	f	1
68	8	f	1
69	10	m	2
70	7	m	1
71	8	m	1
72	4	m	1
73	10	f	1
74	6	f	1
75	1	m	2
76	6	f	1
77	4	m	2
78	10	f	1
79	7	f	1
80	8	m	1
81	5	f	1
82	6	m	1
83	6	m	1
84	4	m	1
85	4	f	1
86	3	m	1
87	14	m	2

Table 1: Data of 98 selected patients of present study

DISCUSSION

G6PD deficiency increases the risk of oxidative damage to erythrocytes. Haemolytic anaemia, chronic haemolytic anaemia, and new-born hyperbilirubinemia are among clinical symptoms that may occur. [9-11] Illness may lead to death. Risk factors for neonatal G6PD deficiency have been identified. For G6PD normal new-borns, prematurity, with or without sepsis, is the most significant risk factor for jaundice. Preliminary study suggests that borderline preterm new-borns may have bilirubin production and conjugation that is inconsistent. Sepsis, the leading cause of new-born mortality in developing countries, raises the risk of jaundice in the child [12]. As a rule of thumb, preterm infants are more likely to suffer from this condition than healthy new-borns [13-14]. G6PD is a cell-protective antioxidant enzyme. G6PD deficiency affects approximately 200 million individuals worldwide. In the Middle East, India, China, and Africa, it is more common [15-17]. There is a link between high bilirubin levels (more than 18 mg/dL) and a G6PD deficiency. Enzyme deficiency is a common and serious health problem. Infections, hypoxia, and certain medicines, foods, and toxins may all cause acute haemolysis [18]. Early haemolysis is produced by oxidative stress, thus it's likely that something happened during pregnancy to induce it. Nonspherocytic haemolytic anaemia is a rare side effect [19-20].

CONCLUSION

The study found that G6PD deficiency is frequent in new-borns and children. Severe hyperbilirubinemia has various risk factors, many of which are relevant. Hyperbilirubinemia must be treated promptly in neonates with G6PD deficiency to avoid irreparable neurological consequences.

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