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G6PD Deficiency & It`s Contribution to Neonatal Jaundice

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Abstract: Objectives: To detect the frequency of G6PD deficiency in 100 neonates admitted with jaundice to the neonatal unit, Sulaimanyah, pediatric teaching hospital. **Material and Methods:** This descriptive study was conducted in the Neonatal Unit of Sulaimanyah, a pediatric teaching hospital, from September 2011 to February 2012. One hundred cases of neonatal jaundice of both sexes admitted to the Neonatal Unit, Sulaimanyah, pediatric teaching hospital, were enrolled in the study. Detailed history and clinical examination were recorded. All the neonates were subjected to be an estimation of Serum Bilirubin levels (Total, Direct, and Indirect) G-6-PD detection via methemoglobin reduction test; normal blood yields color similar to that of normal reference tube, which is red, and blood from deficient subjects gives brown color similar to that in the deficient reference tube. Blood groups of both the mother and the baby besides retic count, Coomb`s test, looking at the peripheral smears, and other relevant investigations. **Results:** Out of the 100 icteric neonates, 72 (72 %) were males, while 28 (28%) were females. Sixteen (16%) babies were found to be G6PD deficient. 94(94%) cases presented with neonatal jaundice in the first week of life, while the age of presentation amongst the G6PD deficient neonates was between the 2nd to 4th day of life. An Indirect serum bilirubin level of >15mg\dl was found in these G6PD deficient neonates. **Conclusion:** G6PD deficiency is a common cause of neonatal jaundice and has more preponderance for the male sex. G6PD deficient babies present with jaundice in the 2nd – 4th days of life in 12 (75%) cases.

Keywords: knee arthroplasty; General anaesthesia; spinal anaesthesia; and complications.

INTRODUCTION

Glucose-6-phosphate dehydrogenase deficiency is the most common enzyme deficiency worldwide (Frank, J. *et al*., 2005). This X-linked deficiency affects more than 400 million people worldwide, representing an overall 4.9% global prevalence (George, B.S. *et al*., 2010). The highest frequencies are detected in Africa, Asia, the Mediterranean region, and in the Middle East, owing to recent migrations. However, the disorder is also found in North and South America and in northern European countries (Beutler, E. 1996). Glucose-6-phosphate dehydrogenase (G6PD) is an enzyme that catalyses the first reaction in the pentose phosphate pathway, providing reducing power to all cells in the form of NADPH (reduced form of nicotinamide adenine dinucleotide phosphate). NADPH enables cells to counterbalance oxidative stress that can be triggered by several oxidant agents and to preserve the reduced form of glutathione.

The most common clinical manifestations are neonatal jaundice and acute haemolytic anaemia, which in most patients is triggered by an exogenous agent (Rehman, H. *et al*., 2005). The likelihood of developing hemolysis and the severity of the disease are determined by the magnitude of the enzyme deficiency, which in turn is determined by the biochemical characteristics of the G6PD variant.

The World Health Organization has classified the different G6PD variants according to the magnitude of the enzyme deficiency and the severity of hemolysis (Vohr, B.R. et al., 2005).

And this study uses AIM To detect the frequency of G-6-PD deficiency in 100 consecutive neonates admitted with jaundice to the neonatal unit.

MATERIALS AND METHOD

This study was carried out in the Neonatal unit, sulaimany, pediatric teaching hospital from September 5th 2011, to February 25th 2012.

All jaundiced neonates who were full-term and had Serum Indirect Bilirubin levels of more than 15mg/dl were included in the study.

Premature, jaundiced neonates, those with neonatal sepsis, and neonates with direct hyperbilirubinaemia were excluded from the study.

Total of 100 admitted jaundiced neonates, aging from 1st day of life to 6 days, were Studied.

Detailed history, examination, and all the required investigations, e.g., serum bilirubin (Total, Direct, and Indirect), PCV, Peripheral blood smear,

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Reticulocyte count, coomb`s test and Blood grouping (both mother`s and neonate`s).

G6PD detection via Methemoglobin reduction test was used for the G6PD level, 2 ml of blood was used; normal blood yields a color similar to that in the normal reference tube, which is red. Blood from deficient subjects gives a brown color similar to that in the deficient reference tube.

RESULTS

One hundred neonates with criteria mentioned in the methods, among of which 16(16%) were found to be G6PD deficient and were enrolled in this study.

Of the total 100 cases of neonatal jaundiced studied, 72 were males, and 28 were females (Table 1).

Table 1: Sex-wide distribution of the total NNJ neonates included in the study (n=100)

Group	Number	Percentage	Male	Female	M: F ratio
Total Patients	100	100%	72	28	3:1
G6PD Normal	84	84%	60	24	2.5:1
G6PD Deficient	16	16%	12	4	3:1

Age ranged from 1 - 6 days with a mean age of 3 days. 12 (75%) with G6PD deficiency presented with jaundice between 2-4 days of birth (Table 2).

Table 2: Age of presentation with NNJ in G6PD deficient neonates (n=16)

Age at presentation	Number of cases	Percentage
1st day	2	12.5%
2nd day to 4th day of age	12	75%
After 4th day of life	2	12.5%
Total	16	100%

Serum indirect bilirubin level ranged from 15 - 22mg/dl. Nine babies developed severe hyperbilirubinaemia (Serum bilirubin level of > 20mg /dl) and were treated by exchange transfusion (Table 3).

Table 3: Total serum bilirubin level in G6PD deficient babies (n=16)

Grade	Serum bilirubin	No of patients	percentage
Mild to moderate	< 20 mg/dl	7	43.75%
Severe	> 20mg/dl	9	56.25%

All G6PD deficient jaundiced neonates received phototherapy. The duration of phototherapy was from 2 – 3 days. The total often exchange blood transfusions were performed in 9 babies. One baby required more than one exchange transfusion (Table 4).

Table 4: Treatment given to G6PD deficient jaundiced babies (n=16)

Treatment Modality	No of Patients	Percentage
Phototherapy	16	100%
Exchange transfusion	9	56.25%

In 18.75 % of G6PD-deficient neonates, there was a positive family history of G6PD deficiency. 10 (62.5%) of the G6PD deficient neonates had a PCV level on admission of more than 45%, and 6 (32.75 %) had a level of less than 45%. (Table 5).

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Table 5: The relationship of G6PD deficient to some variables

Variable	Number of cases	Percentage
PCV > 45%	10	62.5%
PCV < 45%	6	37.5%
Exclusive breastfeeding	12	75%
Mixed feeding	4	25%
Family history of G6PDD Yes No	 3 13	 18.75% 81.25

DISCUSSION

Of the 100 neonates studied, 72 (72%) were males, and only 28(28%) were females, and of the G6PD deficient neonates, 12 (75%) were male, and 4 (25%) cases were female, M: F ratio 3:1.

This male predominance may be attributed to the G6PD deficiency, as it frequently occurs in males than in females (17). Majority of the neonates with G6PD deficiency in our study presented with neonatal jaundice between the 2nd and 4th day of life. This is supported by other similar studies conducted internationally (Nair, P.A. *et al*., 2003).

The frequency of G6PD deficiency in this study was 16%. This figure correlates with other local studies, like Nikkhah. A. *et al*. observed 15.3% (38), and Amir Hussein *et al*. observed 13.6% (Nikkhah, A. *et al*., 2007).

This is also a relatively high occurrence rate as compared to studies from Ferda Ozlu (Turkey) (8), which observed 3%, and Farzaneh Eghbalian (Iran), which observed 4.4%. (Ozlu, F. et al., 2011) On the other hand, this frequency of G6PD deficiency in the jaundiced neonates is quite lower than the frequency reported from Al-naama (Eghabalian, F. 2007) (Basra), that was reported at 51%, and AL-Sowad (Baghdad) (Al-Naama, L.M. *et al*., 1987), who reported it to be 34%. These variations may be due to the frequency of carrier individuals, sample size, the method used for G6PD enzyme estimation, and detection rate.

In this study, Nine babies developed severe hyperbilirubinaemia (Serum bilirubin level of > 20mg/dl), and all of them had exchange transfusion; this result shows the incidence of significant hyperbilirubinemia in G6PD deficient neonates. This high figure of exchange transfusion among G6PD deficient neonates reported in this study may be due to lack of awareness, education, screening, and premarital counseling and referral timing for exchange, early neonatal discharge along with similarity to physiologic jaundice, and lack of parental knowledge, lead to late presentation and delayed treatment of these G6PD-deficient neonates (Mazzucchelli, I, 2005).

Both jaundiced neonates and their mothers had no significant history of drug ingestion. Injection of vitamin K was given to all of the babies. The hemolysis of G6PD is non-immune. Therefore, the direct Coombs test is negative; the results of our study did not show any positive Coomb`s test in the G6PD deficient group. In 50 % of G6PD deficiency neonates, there was a positive family history of neonatal jaundice. 62.5% of the G6PD deficient neonates had a PCV% level at the admission of more than 45%, and 32.75 % had a level less than 45%. Mean retic count was about 4.8%. These findings do not suggest significant hemolysis as a cause of jaundice in these neonates. As hemolysis in G6PD deficient newborns is spontaneous, without significant drug exposure. Moreover, in some G6PD deficient population groups, carboxy – haemoglobin studies have indicated exaggerated hemolysis, but in others, increased hemolysis has no correlation with total serum bilirubin level.

As hyperbilirubinaemia results from an imbalance between bilirubin production and its elimination, diminished bilirubin conjugation was suspected as a contributory factor in the pathogenesis of hyperbilirubinaemia. Serum- conjugated bilirubin fraction, reflecting intra-hepatic bilirubin conjugation, are low in G6PD deficient neonates (Kaplan, M *et al*., 1997).

In this study, the cause of jaundice could not be determined in 61% of the cases. Some of these cases were probably due to physiological jaundice. Others can be considered as nonspecific neonatal jaundice. It is also possible that some could have been G6PD deficient with normal levels during hemolysis.

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Phototherapy was given to all the babies and was found to be very effective in reducing serum bilirubin levels. The total often exchange blood transfusions were performed in 9 babies. One baby required more than an exchange transfusion. This procedure was highly effective in reversing indirect hyperbilirubinemia.

CONCLUSION

- G6PD deficiency is a common cause of neonatal jaundice, and screening for this condition should be included in the schedule of investigations for neonatal jaundice.
- Hyperbilirubinaemia in these patients is indirect and may lead to bilirubin encephalopathy.
- Phototherapy and exchange blood transfusion are effective treatments for these patients.
- G6PD deficient babies present with jaundice in the $2^{nd} - 4^{th}$ days of life in most cases.

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