SUMMARY AND CONCLUSION

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Erythrocyte glucose 6-phosphate dehydrogenase enzyme deficiency is widely distributed among many racial groups. The association between the enzyme deficiency and neonatal jaundice was first described by Doxiadis in 1964. Since then, reports from different localities of the world showed variable association rates between the enzyme deficiency and neonatal jaundice.

The present study aims at estimating the prevalence rate of the erythrocyte G6PD enzyme deficiency in a group of jaundiced newborn infants. Finding the association rate between the enzyme deficiency and neonatal hyperbilirubinemia. Finding the incidence of the erythrocyte G6PD enzyme deficiency among newborn infants presenting with hyperbilirubinemia. Description of the relevant historical, clinical and laboratory data among jaundiced babies with reduced enzyme activity. Estimates the course of neonatal hyperbilirubinemia in neonates with G6PD deficiency and those with normal enzyme level. Comparative study in the prognosis of neonatal hyperbilirubinemia in neonates with G6PD deficiency and those with normal enzyme level.

In our study we use to determine glucose-6-phosphate dehydrogenase level. (Modification of Boehringer Test Red-cell Hemolysate Method).
Instrument: COBAS FARA

This study was conducted in Security Forces Hospital, Riyadh, Saudi Arabia between the period from July 1996 to March 1997 and included two groups of neonates with hyperbilirubinaemia.

A) Group-I

This group included 100 neonates were delivered in Security Forces Hospital and had good apgar score over 1 & 5 minutes and discharged home with good general conditon and they presented back to emergency room with jaundice and were discharged home without any treatment as they fell in the ***white zone of infant jaundice graph*** (see before) and they were given an appointment to the outpatient clinic within (24-48) hours for follow up, as well as bilirubin determination.

B) Group-II

This group included 376 neonates were delivered in Security Forces Hospital. Some of them discharged home with good general condition and the others were admitted directly from observation nursery to the neonatal unit because of hyperbilirubinaemia. Those neonates who were discharged home presented back to the clinic or emergency room because of neonatal jaundice and they were admitted also to the neonatal unit.

N.B. The level of serum bilirubin of neonates in group II was high to require treatment (Phototherapy or exchange transfusion).

In our study the number of jaundiced babies with G6PD deficiency < 1200 U/L was 16 in the first group 16%, and 92 in the second group 24.5%.

* Total = 108 22.7%.

While the number of jaundiced babies with normal G6PD level 1200-3000 U/L was 84 in the first group 84%, and 284 in the second group 75.5%.

* Total = 368 77.3%

N.B: Level of G6PD was below 100 U/L in 16 jaundiced neonates, 2 of them in the first group and 14 in the second group.

In our study, in the second group of jaundiced babies the enzyme deficiency was common among males 67.4% than females 32.6% and in both groups together the enzyme deficiency was common among males 32% (This percentage from the total No. of male babies) than females 15% (This percentage from the total No. of female babies) ***and the differences between sexes is statistically significant.***

In the first group of our study the ***mean ± SD*** serum bilirubin levels at the time of presentation to the hospital with neonatal jaundice were (218 ± 40) m mol/L and the ***mean ± SD*** serum bilirubin levels in the second group at the time of admission to the hospital were (324 ± 57) mmol/L and there is statistical significant increase in the serum bilirubin levels at the time of admission to the hospital in the second group in comparison to the first group.

In the second group at the time of admission to the hospital the ***mean ± SD*** serum bilirubin levels for neonates with low enzyme level were (344 ± 55) mmol/L and for neonates with normal enzyme level were (318 ± 56) m mol/L. *There is statistical significant increase in the serum bilirubin level at the time of admission to the hospital in neonates with low G6PD level in the second group more than those with normal enzyme level.*

We find that ***mean ± SD*** age of neonates at the time of presentation to the hospital in the first group of jaundiced babies were (8.2 ± 3.4) days.

And in the second group of jaundiced babies who were admitted to the neonatal unit were between (7.2 ± 3.8) days.

There is statistical significant decrease in the age of neonates at the time of admission to the hospital with marked hyperbilirubinaemia (second group) in comparison to the age of those with mild hyperbilirubinaemia (first group) at the time of presentation to the hospital.

And the highest mean bilirubin level for jaundiced-G6PD deficient babies in the second group of our study was recorded on the 7th post natal day.

In our study in the first group all neonates were full term babies, and in the second group 352 neonates were full term babies (93.6%) and 24 neonates were preterm babies (6.4%).

That means all preterm babies were present in the second group and developed marked hyperbilirubinaemia because they were fallen in the area above white zone of infant jaundice graph. (see before)

N.B. Preterm babies are more liable to develop marked hyperbilirubinaemia.

Relation between gestational age in both groups together and G6PD level showed that the percentage of full term jaundiced neonates in both groups with low G6PD Level was 26% and with normal enzme level was 74%, on the other hand the percantage of preterm jaundiced

neonates in both groups with Low G6PD Level was 9% and with normal enzyme level was 91%.

Relation between gestational age and G6PD level in the second group of jaundiced babies showed that:

90 neonates with low enzyme level < 1200 U/L were full term babies 97.8%.

And 2 neonates with low enzyme level < 1200 U/L were preterm babies 2.2%.

On the other hand 262 neonates with normal enzyme level (1200 - 3000) U/L were full term babies 92.2%. And 22 neonates with normal enzyme level were preterm babies 7.8%.

Delivery methods in both groups showed that in the first group 68 neonates were delivered by SVD (69.4%) and 32 neonates were delivered by CS (30.6%). And in the second group 254 neonates were delivered by SVD (68.3%), and 122 neonates were delivered by CS (31.7%).

Relation between delivery method in both groups together and G6PD level showed that the percentage of jaundiced neonates in both groups delivered by NSVD with low G6PD Level was 28% and with normal enzyme level was 72% On the other hand the percentage of jaundiced neonates in both groups delivered by CS with low G6PD Level was 17.5% and with normal enzyme Level was 82.5%.

Relation between delivery methods and G6PD level in the second group of jaundiced babies showed that 72 neonates (78.3%) with low enzyme level <1200 U/L were delivered by SVD and 20 neonates (21.7%) with low enzyme level were delivered by CS.

On the other hand 182 neonates (65%) with normal enzyme level (1200-3000) U/L were delivered by SVD and 102 neonates (35%) with normal enzyme level were delivered by CS.

There is significant correlation in the number of neonates delivered by NSVD and associated with G6PD deficiency in comparison to those delivered by CS.

Parents of G6PD deficient jaundiced babies of both group I and II were questioned about the presence of acute hemolytic episodes among other members of the family (First and second degree consanguinity).

The results showed that a positive history of acute hemolytic episodes among family members of G6PD deficient jaundiced babies was present in 2 cases in the first group and 3 cases in the second group.

Statistical analysis showed no significant difference between +ve family history and -ve family history of acute hemolytic episodes.

Individual newborn infants of group I and group II were surveyed for possible exposure to the following presumable stress factors:

1. Maternal intake of hemolytic drug, local use of any oxidant agent capable of causing oxidative hemolysis or ingestion of fava beans late in pregnancy or during labour.
2. Presence of maternal infection by the time of delivery.
3. Evidence of fetal and/or early neonatal distress.

The surveillance for the first two factors (maternal factors) proved that none of the enzyme deficient jaundiced infants investigated was subjected to such a stressing effect.

Fetal and/or early neonatal distress were presumed present if one or more of the following conditions was present:

a) Prolonged labour.
b) Passage of meconium before or during labour.
c) Low 1 and 5 minutes Apgar score.
d) Need for resuscitation.
e) Signs of early neonatal distress e.g. cardio-pulmonary distress, acidosis, infection, sepsis... etc.

Results showed that 25% of G6PD deficient jaundiced infants in the second group with neonatal distress (signs of neonatal sepsis or infection) developed marked hyperbilirubinemia, whereas, only 30.4% of enzyme deficient jaundiced infants without distress developed such a degree of hyperbilirubinemia. The difference between the two percentages was not statistically significant.

In our study the ***mean ± SD*** haemoglobin level in the first group of jaundiced babies was (16.016 ± 3.48) g/dL and ***mean ± SD*** haemoglobin level in the second group jaundiced babies was (16.26 ± 2.44) g/dL.

Relation between haemoglobin and G6PD level in the second group of jaundiced babies showed that there is statistical significant decrease in the mean Hb in g/dl in the neonates with low enzyme level (15.19±2.87) than the mean Hb in g/dl in the neonates with normal enzyme level (16.66±2.13). **There was no evidence of hemolysis.** This can be explained by ***a majority of cases this jaundice may be not of hemolytic but of hepatic origin.***[114,115]

N.B: (1) Normal haemoglobin level for this age group is between (13.5-20) g/dL.

(2) We found drop in haemoglobin level (below 9gm/dL) in 2 neonates with low enzyme level < 100 U/L in the second group of jaundiced babies. One of them was transfused because of high serum bilirubin level.
(3) Reticulocytic count in 97.8% of the second group of jaundiced babies in our study were within normal value.

Phototherapy was highly effective in reducing bilirubin levels in the second group of jaundiced babies in our study. Neonates with normal enzyme level is significantly more affected than neonates with low enzyme level.

Exchange transfusion was done in 2 neonates with low G6PD level 2.2%, and transfusion was done in 6 neonates with normal enzyme level 2.1%.

Exchange transfusion was done because of severe hyper-bilirubinaemia.

Cases were fallen in the Red Zone (Zone of obligatory Exchange). In the infant jaundice Graph (See before).

It is likely that there are, and it may be also heuristically useful to consider, two different types of NNJ associated with G6PD deficiency. First, there would be a more common type, which can best be visualized as a marked exaggeration of "physiologic jaundice". This type is not greatly influenced by the environment, and it may result from G6PD deficiency being expressed in the liver. Second, there would be a rarer, frankly hemolytic type, which can be visualized as AHA (Acute Haemolytic Anaemia) occurring in a newborn because it happened to be exposed to one of the same agents that could cause AHA even in an adult.[75] Here, the exogenous agent may be a drug, an infection, or some particular local habit, for instance, the extensive use of naphthalene ("moth balls" or "comphor balls") in looking after babies. [132]