Glucose 6 phosphate dehydrogenase deficiency among neonates with hyperbilirubinaemia in western Iraq

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Summary

Background: Glucose -6-phosphate (G6PD) deficiency seems to be a major cause of neonatal hyperbilirubinaemia. This study was carried out to determine the prevalence of G6PD deficiency among icteric neonates in western Iraq and to evaluate its association with hemolysis in neonatal jaundice.

Patients and Methods: All icteric neonates admitted to Al-Ramadi Maternity and Paediatrics hospital, Al-Anbar governorat, for the period from 1st Feb. to 1st Dec. 2006 were included in the study. Data collected from case records and includes age, sex, total serum bilirubin hemoglobin level, reticulocyte count, blood group and Rh of the mothers and neonates, direct coomb's test and peripheral smear. G6PD enzymewas measured also.

Results: eight out of 100 icteric neonates had G6PD deficiency, with male to female ratio of 7:1. A significant higher total serum bilrubin (TSB) level was among G6PD deficient icteric neonates than that among non deficient icteric neonates. Reticulocytes count and haemoglobin level was not significantly differ between G6PD deficient and non deficient icteric neonates.

Conclusion: Neonatal screening for G6PD deficiency is a need in order to control genetic blood diseases.

Key words: G6PD deficiency, haemolysis, TSB

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Introduction

Glucose- 6- phosphate dehydrogenase (G6PD) enzyme deficiency is an X linked genetically determined enzyme disorder (1). It is found in several countries among widely ethnic groups especially in Miditerranean region, Middle East and Africa(2) with a high frequency in areas endemic with malaria (3,4). There is a risk of acute hemolysis and neonatal hyperbilirubinaemia in affected neonates which may be so sever to cause kernicterus and death (1,5).

The prevalence of neonatal jaundice is twice that of general population in male carrying the defective gene and in homozygous female 6. Hemolysis as well as impairement of bilirubin conjucation was implicated in pathogensis of neonatal jaundice in G6PD enzyme deficient neonates (7.8). In Iraq, G6PD deficiency seems to be a major cause of naonatal hyperbilirubinaemia (9-12). This study was conducted to determine the prevalence of G6PD deficiency among icteric neonates in western Iraq and to evaluate its association with hemolysis in neonatal jaundice.

Materials and methods

All the icteric neonates (term neonates had total serum bilirubin, TSB, > 12 mg /dl while preterm neonates had TSB > 10 mg / dl) admitted to Al-Ramadi Maternity and Paediatrics hospital, Al-anbar governorate, for the period from 1st Feb. To 1st Dec. 2006 were included in the study.

Data were collected from case records and include age, sex, TSB, hemoglobin level, reticulocyte count, blood group and Rh of the mothers and neonates, direct coombs test and peripheral blood smear. G6PD enzyme was measured by flourscent spot method (Boch ringer manuheim GmGH, West Germany).

Hemolysis was determined by hemoglobin level (< 14.2 ± 2.1 gm / dl and according to age) and reticulocyte count (> 3.2 ± 1.4) 13 with a blood film suggesting hemolysis. Phototherapy and exchange transfusion (ET) were applied when TSB level was > 15 mg / dl and > 20 mg / dl, respectively, in term neonates. Lower levers of TSB were used in preterm infants.

Student's t test were used to examine the effect of G6PD deficiency on TSB, reticulocyte count and hemoglobin level. Chi square was done to examine the effect of G6PD deficiency on the type of management of hyperbilirubinaemia. P value less than 0.05 was considered as significant.

Results:

Eight out of 100 icteric neonates had G6PD deficiency. Male to female ratio was 7:1.

A significant higher TSB level (21.75 ± 1.25) was observed among G6PD deficient icteric neonates than that among non deficient icteric neonates (15.83 ± 2.43) ($p < 0.05$). No significant difference in reticulocyte count and hemoglobin level between G6PD deficient icteric neonates (2.75 ± 0.71, 14.51 ± 1.2; respectively) and non deficient icteric neonates

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(2.82 ± 1.16, 14.45 ± 1.54; respectively). These findings are shown in Table 1.
No significant difference in the types of treatment of hyperbilirubinaemia (phototherapy and exchange transfusion) between icteric neonates with G6PD enzyme deficiency and those without. (Table 2).

Discussion:

The finding that 8% of the jaundiced neonates had G_6PD deficiency is similar to that reported in Baghdad (8.8%) [9] and much lower than that reported in Basrah (51%) 10. The prevalence of G_6PD deficiency among asymptomatic healthy persons in western Iraq (Al- Anbar) (5%) 14 was much lower than the rate in southern Iraq (Basrah) (12.5%) 11. High prevalence of sickle cell disease in Basrah 11,15 may be attribut for this difference in the rate of G_6PD deficiency. Several reports demonstrated higher prevalence of G_6PD deficiency among patients with sickle cell disease than in normal subjects16,20 . However, other reports showed no association between the prevalence of sickle cell disease and G_6PD deficiency 21,22. The G_6PD deficiency occurs with increased frequency throughout Africa, Asia, the Mediterranean and the Middle East. The prevalence of G_6PD deficiency is correlated with distribution of malaria, which led to the theory that carriers of G_6PD deficiency may incur partial protection against malaria infection 3,4.
This study revealed a male to female ratio of 7:1. G_6PD deficiency is X- linked inherited disorder [1]. In addition, female heterozygote may be hard to diagnose because of X- chromosome mosaicism leading to partial deficiency that will not be detected reliably with screening or diagnostic tests 23,24 and neonatal hyperbilirubinemia rarely occur in heterozygous females 5.
A significant higher level of TSB in G_6PD deficient neonates than in non G_6PD deficient jaundiced neonates was observed. This finding is inconsistent with that of other workers (6). However, the prevalence of neonatal hyperbilirubinemia is twice than of general population in G_6PD deficient males and in homozygous female (25).
This study showed similar figures of haemoglobin level and reticulocyte count in G_6PD deficient and non deficient jaundiced neonates. Haemolysis is not the main determinant of neonatal hyperbilirubinemia in G_6PD deficient neonates (7,26). Hyperbilirubinemia in G_6PD deficient neonates is the result of an imbalance between production and conjugation of bilirubin with a tendency for insufficient bilirubin conjugation over increased haemolysis in its pathogensis (8). Prematurity is an important contributing factor to hyperbilirubinemia (27). Recently, high prevalence of prematurity was reported in Iraq (28,29). Acute haemolysis could be caused by infection. A high prevalence of neonatal sepsis (9.2 per 1000 live birth) was reported in Al-Anbar governorate (30).
The finding that 62.5% of G_6PD deficient jaundiced neonates underwent exchange transfusion (ET) is much higher than that reported in Basrah (28.4%) [10] and in Saudi Arabia (7%) (31). This might be attributable to the deterioration of health services during and after the Gulf wars and the sanctions in Iraq (12,32). In Bahrain, where G_6PD deficiency is a problem (33), a campaign to control genetic blood diseases was started (34). With continuation of education, awareness and a premarital counseling service, the expected number of affected children born to be reduced tremendously over the next years.
A high rate of neonatal hyperbilirubinemia was due to G_6PD deficiency, and a high rate of them required ET. Neonatal screening for G_6PD deficiency is a need to build a control program of genetic blood diseases

Table 1. Distribution of TSB, reticulocyte count and hemoglobin level among icteric neonates

Variable	G6PD				
(mean ± SD)	deficenct neonates		Non deficient neonates		P value
TSB	21.75 ± 1.25		15.83 ± 2.43		< 0.05
Reticulocyte count	2.75 ± 0.71		2.82 ± 1.16		> 0.05
Hemoglobin level	14.51 ± 1.2		14.45 ± 1.54		> 0.05

Table 2. Management of neonatal hyperbilirubinaemia

	G6PD		
Treatment	Deficient neonates		Non Deficient neonates
Phototherapy	8 (100)		54 (79.1)
Exchange blood transfusion	5 (62.5)		14 (20.9)

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