

Cord Blood Albumin as A Predictor of Neonatal Jaundice

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Abstract

Jaundice is one of the commonest problems that can occur in a newborn. Many a times it is physiological in the newborn because liver is not mature enough to handle the bilirubin and there is an increased load of bilirubin due to a higher circulating erythrocyte volume, a shorter erythrocyte life span and a larger early labeled bilirubin peak. Early prediction will help in early discharge and prevent hospitalization of babies and mothers. Albumin is synthesized by liver and it helps in transport of unconjugated bilirubin. Forty newborns were inducted into the study with the following inclusion criteria: sequentially born term babies (gestational age > 37 weeks) from any mode of delivery, both genders, any birth weight, APGAR score of more than 7 at first and fifth minutes of life, and without Rh incompatibility between mother and child. They were estimated for cord blood serum albumin. Wherever necessary further laboratory tests were done for bilirubin and managed accordingly. Our study showed that 82 % of neonates who had albumin levels less than 2.8 gm/dl developed hyperbilirubinemia requiring phototherapy (PT) and about 12% needed exchange transfusion. At higher levels of albumin that is 2.8 - 3.3 gm/dl, 40% needed PT and with cord blood albumin > 3.3 gm/dl did not need any intervention for hyperbilirubinemia. Hence we can conclude that cord blood albumin levels more than 3.3 gm/dl is probably safe for early discharge of baby. Umbilical cord albumin levels are useful in predicting the development of jaundice in healthy term infants.

E-mail: Jaundice; Umbilical cord albumin; Phototherapy.

Introduction

Jaundice is one of the commonest problems that can occur in a newborn. Many a times it is physiological in the newborn because liver is not mature enough to handle the bilirubin. The neonates have about 1% of uridine diphosphoglucuronosyl transferase (UDPGT) activity as that of an adult [1]. Apart from this there is an increased load of bilirubin in neonates as they have a higher circulating erythrocyte volume, a shorter mean erythrocyte life span and a larger early labeled bilirubin peak [2]. This hyperbilirubinemia is due to unconjugated bilirubin which is toxic to central nervous system. More than two thirds of all newborns appear

jaundiced clinically because at some point during the first week of life almost every newborn has a total serum bilirubin (TSB) level of > 1 mg/dl, the upper limit of normal for an adult. There are significant differences in TSB levels in different populations and it is difficult to define as normal or abnormal or obtain diagnostic and therapeutic cut off levels [3]. Defining a certain bilirubin level as physiological can be misleading and potentially dangerous. It is difficult to predict the course of bilirubinemia on day one of a neonate. There have been reports of cord blood bilirubin as predictor of hyperbilirubinemia that would require phototherapy (PT) [4, 5]. Early prediction will help in early discharge and prevent unnecessary hospitalization of babies and mothers. Albumin is

synthesized by liver and it helps in transport of unconjugated bilirubin. There is paucity of reports on serum albumin or cord blood albumin levels as a predictor of hyperbilirubinemia. Keeping the aforesaid in mind we studied umbilical cord serum albumin levels and followed the babies for hyperbilirubinemia and those requiring PT.

Methods

This study was conducted in the departments of Pediatrics and Biochemistry at our Institute. Participants: Forty newborns were inducted into the study with the following *inclusion* criteria: sequentially born term babies (gestational age > 37 weeks) from any mode of delivery both genders, any birth weight, APGAR score of more than 7 at first and fifth minutes of life, without Rh incompatibility between mother and child. The exclusion criteria were preterm babies (gestational age < 37 weeks), neonatal sepsis and any complications arising during the hospital stay.

Biochemical investigations

With the informed consent of the parents 2 ml of cord blood was collected from newborns and serum separated. They were estimated for serum albumin by BCG method [6] using Dade Behring, UK. From then on the newborns were followed up clinically for jaundice according to the evaluation of the Kramer dermal zones [7]. Wherever necessary further laboratory tests were done for bilirubin and managed

accordingly.

Statistical Analysis

The qualitative variables were represented as absolute (n) and relative (%) frequency values. Quantitative variables were represented as mean and standard deviation. Correlation between birth weight and cord blood albumin was obtained using Pearson's correlation.

Results

The lower limit of normal for serum albumin in term babies is 2.8 gm/dl at birth [8]. We grouped our subjects according to their cord blood albumin levels upto 2.8 gm/dl, 2.8 - 3.3 and > 3.3 gm/dl. Table 1 shows the general characteristics of all the participants. 82.35% of babies with cord blood albumin < 2.8 gm/dl developed hyperbilirubinemia, all of them requiring phototherapy and 2 of them (11.8%) requiring exchange transfusion. The group with cord blood albumin between 2.8 - 3.3 gm/dl had a lesser incidence that is 40% babies developed hyperbilirubinemia and they were all treated with PT. Whereas none of the babies with cord blood albumin > 3.3 gm/dl developed hyperbilirubinemia requiring any intervention. Table 2 shows that the Pearson's correlation 'r' between birth weight and cord blood albumin. It was positive in the lower group that is < 2.8 and 2.8 - 3.3 gm/dl and was negative when albumin levels were > 3.3 gm/dl. However, none of them were statistically significant. ANOVA done of cord blood albumin levels between groups and

Table 1: General characteristics of participants

	< 2.8 gm/dl	2.8 - 3.3 g/dl	> 3.3 gm/dl
Number of neonates (n)	17	15	8
Neonates developing hyperbilirubinemia (%)	14 (82.35 %)	6 (40 %)	0
Number of newborns requiring phototherapy (%)	14 (82.35 %)	6 (40 %)	0
Number of newborns requiring exchange transfusion (%)	2 (11.8%)	0	0
Weight in kg (mean ± SD)	2.87± 0.48	2.97± 0.36	3.12± 0.27
Cord blood albumin in gm/dl (mean ± SD)	2.49± 0.19	3.2 ± 0.1	3.6± 0.19

Table 2: Pearsons correlation (r) between weight of neonate and cord blood albumin

Cord blood albumin (gm/dl)	r	P
< 2.8	0.04	NS
2.8-3.3	0.24	NS
>3.3	-0.3	NS

within groups showed significant differences ($p=0.000$) whereas the difference for birth weight was not statistically significant ($p=0.541$).

Discussion

Albumin is the major binding protein in the human neonate. Low production of albumin will lower its transport and binding capacity [9]. Albumin binds to potentially toxic products like bilirubin and antibiotics. Bilirubin binds to albumin in an equimolar ratio. Free bilirubin is anticipated when the molar bilirubin-to-albumin (B:A) ratio is >0.8 . It is the free bilirubin which crosses the blood brain barrier. There are no precise data to correlate a specific bilirubin value or albumin value with neurotoxicity. The clinical manifestations of acute bilirubin encephalopathy can be insidious and progress rapidly to severe and life threatening illness. Kernicterus is the chronic sequelae of acute bilirubin encephalopathy. The incidence of kernicterus is unknown [10]. Unconjugated hyperbilirubinemia is a potentially correctable cause and kernicterus is preventable [11]. The decision to treat hyperbilirubinemia is based on the infant's history course, physical findings serum bilirubin levels and risk benefit analysis [12]. Our study showed that 82% of neonates who had albumin levels less than 2.8 gm/dl developed hyperbilirubinemia requiring PT and about 12% needed exchange transfusion. At higher levels of albumin that is 2.8 - 3.3 gm/dl 40% needed PT and neonates with cord blood albumin >3.3 gm/dl did not need any intervention for hyperbilirubinemia. Hence we can conclude that cord blood albumin levels more than 3.3 gm/dl is probably safe for early discharge of baby. Similar work has been reported using cord blood bilirubin [4, 5, 13] and umbilical cord haptoglobin [14].

Conclusion

Umbilical cord albumin levels are useful in predicting the development of jaundice in healthy term infants. It will help detect infants at low or high risk for hyperbilirubinemia. This will minimize hospitalization and prevent readmission of infants with jaundice.

References

1. Kawade N, Onishi S. The prenatal and postnatal

development of UDP-glucuronyltransferase activity toward bilirubin and the effect of premature birth on this activity in the human liver. *Biochem J.* 1981; 196: 257-260.

2. MacDonald MG, Mullet MD, Seshia MMK. *Avery's Neonatology: Pathophysiology and Management of the Newborn.* 6th ed. Lippincott Williams & Wilkins, Philadelphia. 2005; pp 773
3. Sackett DL, Hayes RB, Guyatt GH. *Clinical Epidemiology: A Basic Science For Clinical Medicine* 2nd ed. Boston: Little Brown, 1991.
4. Sun G, Wang YL, Liang JF, Dn LZ. Predictive value of umbilical cord blood bilirubin level for subsequent neonatal jaundice. *Chinese Journal of Pediatrics.* 2007; 45(11): 848-852.
5. Suchonsker b, Weelgos M, Bobroska K, Marianowski L. Concentration of bilirubin in the umbilical blood as an indicator of hyperbilirubinemia in newborn. *Ginekol Pol.* 2004; 75(10): 749-753.
6. Dumas BT, Peters T, Jr. Serum and urine albumin: A progress report on their measurement and clinical significance. *Clin Chem Acta.* 1997; 258: 3-20.
7. Szabo P, Wolf M, Bucher HU, Fauchere JC, Haensse D, Arlettaz R. Detection of hyperbilirubinemia in jaundiced full term neonates by eye or by bilirubinometer? *Eur J Pediatrics.* 2004; 163(12): 722-727.
8. Burtis CA, Ashwood AR, Bruns DE. *Tietz Textbook of Clinical Chemistry and Molecular Diagnostics.* 4th ed. Elsevier, Missouri. 2008; pp 2254.
9. Sgro M, Campbell D, Shah V. Incidence and causes of severe neonatal hyperbilirubinemia in Canada. *Canadian Medical Association Journal.* 2006; 175 (6): 561.
10. Bunt JE, Rieneld T, Schier Beek H, Wattimena JL, Zimmermann LJ, Van Goudoever JB. Albumin synthesis in preterm infants on the first day of life studied with [3 H] leucine. *Am J Physiol Gastrointest Liver Physiol.* 2007; 292(4): 1157-1161.
11. American academy of pediatrics. Subcommittee on Hyperbilirubinemia: Neonatal Jaundice and Kernicterus *Pediatrics.* 2001; 108: 763-765.
12. American academy of Pediatrics. Subcommittee on Hyperbilirubinemia: Management of Hyperbilirubinemia in the newborn infant 35 or more weeks gestation. *Pediatrics* 2004; 114: 297-316.
13. Bernaldo AJN, Segre CAM. Bilirubin dosage in Cord blood: could it predict neonatal hyperbilirubinemia? *Sao Paulo Med J.* 2004; 122(3): 99-103.
14. Cakmak A, Calik M, Atas A, Hirfanoglu I, Erel O. Can haptoglobin be an indicator for the early diagnosis of neonatal jaundice? *J clin Lab Anal.* 2008; 22(6): 409-414.