Neonatal Hyperbilirubinemia, Types, Causes and Treatments: A Review Study

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ABSTRACT

Neonatal hyperbilirubinemia results from a readiness for the bilirubin production in neonates and limited their ability to excrete it. The diagnosis of hyperbilirubinemia based on yellow discoloration of the skin and whiteness of eyes, idle in the child’s movement and the lack of lactation. The baby seems sick or is difficult to awaken. Bilirubin is a tetrapyrrole pigment derived from breakdown product of normal heme catabolism in senescent red blood cells. Unconjugated bilirubin normal elevation is named newborn physiologic hyperbilirubinemia, whereas the level of bilirubin of infant normally to be a bit higher after birth, In the same context the placenta in the womb of the mother. Pathologic hyperbilirubinemia is defined as odd of bilirubin from the normal level so intervention required. Many reasons for this type of jaundice

Keywords: Hyperbilirubinemia, Bilirubin, Neonate Junduice

INTRODUCTION

Neonatal hyperbilirubinemia

It is the common condition of medical importance in newborns, also called Neonatal jaundice or Neonatal icterus, in which the yellow pigment bilirubin can accumulate in the extracellular fluids by the way in the skin, over the sclera in the membranes of conjunctival and in other membranes of mucous after birth within the first few days, as well as skin prevails in yellow almost in face and extending down onto the chest, jaundice of neonate in normal circumstances is harmless: this condition is often seen in infants around the second day after birth, and goes on till day 8 in normal births, or to about day 14 in premature births. Common reasons of neonatal jaundice include jaundice due to formula supplementation, normal physiologic and hemolytic jaundice that include deficiency of glucose-6-phosphate dehydrogenase, hereditary spherocytosis (Kaplan and Hammerman,2002; Watchko and Maisels,2003), deficiency of pyruvate kinase, autoantibodies of ABO/Rh blood type, or infantile pyknocytosis (Bertini et al.,2001;Huang et al.,2013).

The level of bilirubin have increased in all newborns at the first week of life with approximately 80% of preterm and 50-60% of term babies developing jaundice(NIHCE,2010; Rennie et al.,2010), when compared to full-term neonates, in the first week of life Preterm neonates are at greater risk of hospital readmission due to hyperbilirubinemia (Elizabete et al.,2011), the bilirubin reaches the highest point within 3–5 days of life and the problem usually resolved by two weeks, In contrast irreversible brain damage and kernicterus can cause by sever neonatal hyperbilirubinaemia (Cabra and Whitfield,2005). Sometimes, neonatal jaundice will either appear in First hours, for example, before 24 hours old of infant, or unexpectedly increase to a very high level in the second or the third day after birth; In this case there must be a special blood tests done to determine if the level

https://doi.org/10.46966/msjar.v2i2.23
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of bilirubin is abnormal and to identify the reasons behind the rise(Click et al.,2013).

Types of Neonatal Hyperbilirubinemia

*Physiologic Hyperbilirubinemia*

Unconjugated bilirubin normal elevation is named newborn physiologic hyperbilirubinemia (Mary,2010), whereas the level of bilirubin of infant normally to be a bit higher after birth. In the same context the placenta in the womb of the mother.

Eliminate bilirubin from body of baby then postnatal the liver of baby carries out this task and the liver may take some time until the competence to do so, this variance greatly depends on infant's genetic factors, feeding and race (Buescher and Bland,2011; NBAA,2012). Also, may be occurred for a number of reasons: An impaired ability to extract bilirubin, a high rate of bilirubin production and increased Enterohepatic circulation (Yang et al.,2013).

Production of bilirubin also increased when the red blood cell volume per body weight and hematocrit has been elevated in addition a shorter life span of the red blood cells (Bryon et al.,2011), whereas hepatic of infant has immature glucuronosyl transferase, essential enzyme of bilirubin conjugation since facilitates excretion of bilirubin from the body(Bryon et al.,2011), also it can decrease concentration of intestinal bacteria(Yang et al.,2013). Perhaps the reason for increased bilirubin load from relative polycythemia and immature hepatic uptake and processes of conjugation(Gartner and Herschel,2001). Wherefore, neonatal adaptation to bilirubin metabolism a reflection to indirect hyperbilirubinemia and is termed physiologic hyperbilirubinemia (Maisels,1999).

*Pathologic Hyperbilirubinemia*

Pathological jaundice is defined as odd of bilirubin from the normal level so intervention required. Many of the reasons for this type of jaundice such as deficiencies of erythrocyte-enzyme, incompatibilities of blood-group, the erythrocytes structural defects, deficient hepatic uptake of bilirubin, as occurs in Gilbert's syndrome, uridine diphosphate glucuronosyltransferase deficiency (UDGTD) and congenital liver disease one of the main reasons of neonatal hyperbilirubinemia, in any case this disease is much worse; infants have high yellowing color but not seeming well, unlike the rest of the types of jaundice so not resolved on its own. This problem cannot be solved by itself and perhaps lead to destruction (AAP,1994; Denney et al.,2001).

**Jaundice of Breastfeeding failure:**

Also called lack of breastfeeding jaundice is induced by intake of insufficient breast milk (Lynn et al.,2002; Mehta et al.,2005), so the removal of bilirubin is difficult; because, quantities of bowel movements inadequate (Kuzniewicz et al.,2008). This can usually be got better in sequencing.

**Jaundice Breast Milk**

A phenomenon also termed as (syndrome of BMJ) associated with breast milk feeding including prolonged unconjugated hyperbilirubinemia (Maruo et al.,2014). A common cause of indirect hyperbilirubinemia is a problem of mechanical which is a biochemical incident and the bilirubin with higher level may act as an antioxidant BMJ communally occurs in the later period of infants. So, the level of bilirubin may peak in the 6 to 14 days of infant life, whereas, these higher levels may persist for many weeks or in others the level decreased in 3rd to 4th weeks (Col et al.,2011).

Jaundice of breast milk is different than jaundice of breastfeeding (AAP,2004; Logeshwaran et al.,2013),whereas (BMJ) underlying cause is not entirely understood, suggesting that maternal milk contains substances such as β-glucuronidases can repress normal metabolism of bilirubin; therefore, enhancing increased glucuronide hydrolysis of bilirubin subsequently increase in the bilirubin enterohepatic circulation(Gartner and Herschel,2001).

Decreases of Hyperbilirubinemia occur when breast milk is replaced with artificial milk formula and even if continues feeding with maternal milk prolonged unconjugated hyperbilirubinemia over time may become better over time as well bilirubin encephalopathy (kernicterus), BMJ may be induced in severe unconjugated hyperbilirubinemia an occasional risk (PP,1994; Maruo et al.,2014).
Treatment of neonatal hyperbilirubinemia

Treatment of hyperbilirubinemia started with either phototherapy or exchange transfusion. Adequate hydration was also considered ways to reduce the high bilirubin in neonates. The levels of bilirubin start with phototherapy are different based on the health status and age of the neonates.

Treatment begins by phototherapy, but if this method fails to reduce the level of bilirubin in serum, symptoms of kernicterus will begin to appear, the next step for the treatment is exchange transfusion (Robert et al., 2011) which is considered the best way to treat hyperbilirubinemia, which affects neonates and protecting from kernicterus. It was the better successful treatment method for neonate's hyperbilirubinemia (Porter and Dennis, 2002).

CONCLUSION

Neonatal hyperbilirubinemia is known as icterus neonatorum, neonatal jaundice and infant jaundice is a very common medical condition but poorly understood problem and it is in many cases of uncertain clinical significance despite the seriousness of this disease. This unique problem of jaundiced newborn is harmful for developing central nervous system of infants, because, potentially toxic of serum bilirubin elevation there- after extreme jaundice can cause kernicterus due to permanent brain damage.

ACKNOWLEDGMENT

Not applicable.

REFERENCES

American Academy of Pediatrics (AAP), (1994). Practice Parameter: Management of Hyperbilirubinemia in the Healthy Term Newborn. Pediatrics, 94:558-566.

Bertini, G.; Dani, C.; Trochin, M. and Rubaltelli, F., (2001). Is breastfeeding really favoring early neonatal jaundice? Pediatrics, 107(3): 41.

Bryon, J.; Lauer, M.D.; Nancy, D. and Spector, M.D., (2011). Hyper- bilirubinemia in the Newborn. Pediatrics in Review, Vol.32(8).

Buescher, J.J.; Bland, H.,(2011) . Care of the newborn. In: Rakel RE, ed. Textbook of Family Medicine. 8th ed. Philadelphia, PA: Saunders Elsevier; chap 22.

Cabra, M.A.; Whitfield, J.M., (2005). The challenge of pre venting neonatal bilirubin encephalopathy: a new nursing protocol in the well newborn nursery. Proceedings (Baylor Univ Med Cent), 18: 217–9.

Click, R.; Dahl-Smith J.; Fowler, L.; DuBose, J.; Deneau-Saxton, M. and Herbert, J., (2013). An osteopathic approach to reduction of readmissions for neonatal jaundice. Osteopath Fam Physician,5:17-23.

Col, PV. Rama Mohan; Wg Cdr M Kumar; Maj R Pacharu,(2011). Mirizzi syndrome. MJAFI 67:280–281.

Dennery, PA.; Seidman, DS. and Stevenson, DK., (2001). Neonatal hyperbilirubinemia, N Engl J Med.,344:581–590.

Elizabete, P.; Maria, A M. and Fernando P. F., (2011). Systematic follow-up of hyperbilirubinemia in neonates with a gestational age of 35 to 37 weeks. J Pediatr.,87(4):301-306.

Gartner, L. M.; Herschel, M., (2001). Jaundice And Breastfeeding. Pediatric Clinics of North America, Vol.48(2): 389–400.

Huang, L.; Bao, Y.; Xu, Z.; Lei, X.; Chen, Y.; Zhang, Y.; Zhang, J., (2013). Neonatal bilirubin levels and childhood asthma in the US Collaborative Perinatal Project, 1959–1965. Am J Epidemiol. 178(12):1691–1697.

Kaplan, M.; Hammerman C., (2002). Glucose-6-phosphate dehydrogenase deficiency: a potential source of severe neonatal hyperbilirubinemia and kernicterus. Semin Neonatol.,7(2):121–128.

Kuzniewicz, M.W.; Escobar, G.J.; Wi, S.; Liljestrand, P.; McCulloch C.; Newman, T.B., (2008). Risk factors for severe hyperbilirubinemia among infants with borderline bilirubin levels: a nested case-control study. J Pediatr. 153(2):234–240.

Logeshwaran, R.; Siva, K. P.; Kuldeep, S.; Pravin K. S., (2013). A Novel Design of Low-Cost LED Photo Therapy Equipment. Bachelor of Engineering, Associate Professor - Department of Biomedical Engineering, Sri Sivasubramaniya Nadar College of Engineering, Tamil Nadu, India Master of
Science, Arizona State University, U.S.A. *International Journal of Engineering & Technology Research*, Vol (1), Issue 2: pp. 46-51.

**Lynn, C.; Garfunkel, J.; Cynthia C.,** (2002). Mosby's pediatric clinical advisor: instant diagnosis and treatment. *Elsevier Health Sciences*. pp. 200. ISBN 978-0-323-01049-8.

**Maisels, MJ.,** (1999). Jaundice. In Neonatology, Pathophysiology & Management of the Newborn, eds. Avery GB, Fletcher MA, & MacDonald MG, pp. 765-820. Lippincott Williams & Wilkins, Philadelphia.

**Maruo, Y.; Morioka, Y.; Fujito, H.; Nakahara, S.; Yanagi, T.; Matsui, K.; Mori, A.; Sato, H.; Tukey, R.H.; Takeuchi, Y.,** (2014). Bilirubin Uridine Diphosphate-Glucuronosyltransferase Variation Is a Genetic Basis of Breast Milk Jaundice. *J Pediatr*. 165:36-41.

**Mary Ann Liebert,** (2010). ABM Clinical Protocol #22: Guidelines for Management of Jaundice in the Breastfeeding Infant Equal to or Greater Than 35 Weeks’ Gestation. *The Academy of Breastfeeding Medicine Protocol Committee. Inc.*, vol.5(2).

**Mehta, S.; Kumar, P.; Narang, A.,** (2005). A randomized controlled trial of fluid supplementation in term neonates with severe hyperbilirubinemia. *J Pediatr*. 147:781-5.

**National Blood Authority Australia (NBAA),** (2012). Standing Council on Health. Criteria for the clinical use of intravenous immunoglobulin in Australia. Second edition.

**National Institute for Health and Clinical Excellence (NIHCE),** (2010). Neonatal jaundice. CG98. London: National Institute for Health and Clinical Excellence.

**Porter, ML. ; Dennis, BL.,**(2002). "Hyperbilirubinemia in the term newborn". Am Fam Physician, 65(4):599–606.

**Practice parameter (PP),** (1994)." management of hyperbilirubinemia in the healthy term newborn". *Pediatrics*. 944 pt 1:558–62.

**Rennie, J.; Burman-Roy, S. and Murphy, M.S.,** (2010). Neonatal jaundice: summary of NICE guidance. *BMJ.*, 340:c2409.

**Robert, M. ; Kliegman, Richard E. ;Behrman, Bal B. ;Jenson, S. Abramson, et al. ,**(2011). "Neonatal Jaundice". *Nelson text book of Pediatrics 19th Edition.Chapter96.3.page 603.*

**Watchko, J.F.; Maisels, MJ.,** (2003). Jaundice in low birthweight Infants: pathobiology and outcome. *Arch Dis Child Fetal Neonatal Ed.* 88: F455–F458.

**Yang, W.C.; Zhao, L.L.; Li, Y.C.; Chen, C.H.; Chang, Y.J; Fu, Y.C.; Wu, H.P. et al.** (2013). Bodyweight loss in predicting neonatal hyperbilirubinemia 72 hours after birth in term newborn infants. *BMC Pediatrics*, 13:154.