

JAUNDICE AND THE BREASTFED BABY

Lawrence M. Gartner, M.D.
The University of Chicago

Objectives:

1. Understand the physiology of bilirubin metabolism and transport in the newborn infant.
2. Understand the effects of breastfeeding on bilirubin metabolism and the mechanisms responsible for these alterations.
3. Differentiate between breastfeeding jaundice and breastmilk jaundice.
4. Understand the principles of management which preserve breastfeeding while minimizing risks of bilirubin toxicity.
5. Understand new concepts of possible benefits of neonatal jaundice and breastmilk jaundice.

Outline:

(1) Bilirubin Metabolism and Transport in the Newborn

Nearly every newborn infant has elevated serum unconjugated bilirubin concentrations during the early days of life due to the interactions of increased bilirubin synthesis, increased intestinal bilirubin absorption and decreased hepatic bilirubin uptake and conjugation as compared to the older child and adult. This is known as physiologic jaundice of the newborn. Prematurity and race alter these functions and result in different patterns of physiologic jaundice.

(2) Role of Breastfeeding in Physiologic Jaundice of the Newborn

In artificially-fed newborns, physiologic jaundice disappears after about 10 to 12 days of life and serum bilirubin concentrations decline to the adult normal levels of less than 1.5 mg/dl (23 μ M/L). In breastfed infants, jaundice and serum bilirubin concentrations remain elevated for a much longer period of time due to the effect of mature human milk in enhancing the intestinal absorption of bilirubin. During the third week of life, approximately one-third of all full-term, exclusively breastfed healthy infants will be clinically jaundiced and approximately two-thirds will have serum bilirubin concentrations which range from 1.5 mg/dl to about 20 mg/dl. This prolongation of physiologic jaundice in breastfed infants is known as Breastmilk Jaundice. Jaundice may persist for up to two months and elevated serum unconjugated bilirubin concentrations for up to 4 months.

During the first five days of life, optimally breastfed infants and artificially-fed infants have identical serum bilirubin concentrations and intensity of physiologic jaundice. Breastmilk jaundice does not begin until after the fifth day of life. Some breastfed infants who are not nursing frequently enough or who have problems with milk transfer due to maternal and/or newborn problems which are usually preventable or correctable, may have increased serum bilirubin concentrations and more intense clinical jaundice. This type of exaggerated early

jaundice is known as **Breastfeeding Jaundice** or, more appropriately, **Breast-non-feeding Jaundice**. It is the newborn equivalent of the adult phenomenon known as Starvation Jaundice. Breastfeeding jaundice results from inadequate frequency of breastfeeding and insufficient caloric intake which enhances intestinal bilirubin absorption. It can be prevented by initiating breastfeeding immediately after birth (first hour of life), by nursing the baby at least 10 to 12 times per day in the first week or two of life (with 24 hour rooming-in) and by not giving water or glucose water to breastfeeding infants. Early evaluation of breastfeeding technique and correction of position, latch and other problems will also prevent development of Breastfeeding Jaundice. Breastfeeding Jaundice or starvation jaundice may continue past the first five days of life into the second and later weeks of life if breastfeeding problems are not resolved and inadequate milk intake continues.

(3) Interaction of Breastfeeding Jaundice and Breastmilk Jaundice

Both *breastmilk* jaundice and *breastfeeding* jaundice are the result of an increase in intestinal absorption of unconjugated bilirubin. In the case of breastfeeding jaundice, insufficient caloric intake and diminished presence of milk in the intestine allows or promotes increased intestinal absorption. In the case of breastmilk jaundice, an as yet unidentified factor present in most mature human milk promotes increased intestinal bilirubin absorption.

The infant who develops exaggerated early-onset hyperbilirubinemia due to either hemolysis (ABO, Rh, etc.) or breastfeeding jaundice has a greater risk of developing higher levels of bilirubin when the breastmilk jaundice phenomenon begins after day 5. This is presumed to be due to the development of a larger bilirubin pool in the body early and then recycling this already larger pool via the intestinal absorption during the later weeks of neonatal life into an even larger pool. The prevention of early exaggerated hyperbilirubinemia, especially through effective breastfeeding, reduces the likelihood that breastmilk jaundice will rise to such high levels that it requires either further investigation or treatment.

(4) Management of Jaundice in the Breastfed Infant

Every effort should be made to maintain breastfeeding, and to reassure the mother that her breastmilk is good. Once having ruled out obvious causes of hemolysis (ABO, Rh, etc.) in a healthy, full-term breastfed infant, moderate levels of elevated serum unconjugated bilirubin do not require any treatment. After 72 hours of age, total serum bilirubin levels of up to 15 to 20 mg/dl in a healthy infant do not require the institution of supplementation with formula or interruption of breastfeeding, nor is phototherapy necessary. When serum bilirubin levels exceed 20 mg/dl, consideration may be given to continued observation to assess whether the elevated bilirubin is continuing to rise. If treatment is deemed necessary to lower the bilirubin, supplementation of breastfeeding with infant formula administered with a nursing supplementer device during each breastfeeding is preferable. If the infant's total serum bilirubin fails to

decline to 20 mg/dl or less, then interruption of breastfeeding for 24 hours and/or the institution of phototherapy may be considered. Exchange transfusions in full-term healthy infants are recommended only when total serum bilirubin exceeds 25 to 30 mg/dl.

(5) Theoretical Benefits of Jaundice

The nearly universal development of hyperbilirubinemia and jaundice in newborns and its prolongation in the great majority of breastfed infants may seem to be peculiar in view of the fact that bilirubin has the potential for causing bilirubin encephalopathy (kernicterus). There are many troubling aspects to bilirubin metabolism, as well, including the fact that the first step in heme degradation results in the production of biliverdin which is a non-toxic, water soluble compound that could be readily excreted without any further metabolism by both the liver and kidney. Why do all mammals convert this non-toxic biliverdin into insoluble, toxic bilirubin with the need for special transport mechanisms and potential for brain injury? The answer may lie in the observation that bilirubin is a very potent anti-oxidant. Many diseases of adults (cancer, cardiovascular disease) and infants (necrotising enterocolitis, retinopathy of the premature and bronchopulmonary dysplasia) are believed to be mediated by oxygen free radicals present in excess concentrations. Newborns are deficient in many of the naturally occurring antioxidants. Bilirubin may be the replacement for these deficient antioxidants. Recent animal studies have shown that elevated serum bilirubin concentrations can prevent bowel necrosis after an ischemic/hypoxic insult. In another study, the recycling of bilirubin to biliverdin and then back to bilirubin has been shown to provide protection against cell injury on a continuing basis, protecting cells from death even with a thousand-fold increase in oxygen free radicals. Further research is needed to prove that hyperbilirubinemia in newborns actually prevents development of disease or tissue injury.

REFERENCES

1. Gartner LM, Arias IM. Studies of prolonged neonatal jaundice in the breastfed infant. *J Pediatrics* 1966; 68:54
2. Dahms B, Krausse A, Gartner LM et al. Breast feeding and bilirubin during the first four days of life. *J Pediatrics* 1973; 83:1049
3. DeCarvalho M, Hall M, Harvey D. Effects of water supplementation on physiologic jaundice in breast-fed babies. *Arch Dis Child* 1981; 56:568
4. DeCarvalho M, Kalus MH, Merkatz RB. Frequency of breast-feeding and serum bilirubin concentration. *Am J Dis Child* 1982; 136:737
5. Schneider AP. Breast milk jaundice in the newborn. A real entity. *JAMA* 1986; 255:3270

6. Kuhr M, Paneth N. Feeding practices and early neonatal jaundice. *J Pediat Gastroent Nutr* 1982; 1:485
7. Gartner LM, Auerbach KG. Breastmilk and breastfeeding jaundice. *Advances in Pediatrics* 1987; 34:249
8. Gartner LM. Neonatal jaundice. *Pediatrics In Review* 1994; 15:422
9. Gartner LM. On the question of jaundice in the first five days of life. *Seminars in Perinatology* 1994; 18:502
10. American Academy of Pediatrics. Clinical Practice Guideline. Management of Hyperbilirubinemia in the Newborn Infant 35 or More Week of Gestation. *Pediatrics* 2004; 114:297-316
11. Gartner LM, Lee KS. Jaundice in the breastfed infant. *Clin Perinat* 1999; 26:431 - 445
12. Dixit R, Gartner LM. The jaundiced newborn: Minimizing the risks. *Contemporary Pediatrics* 1999; 16:166-183
13. Gartner LM, Herschel M. Jaundice and Breastfeeding. Breastfeeding 2001, Part II. The Management of Breastfeeding. *Pediatric Clinics of North America* 2001; 48:389
14. Baranano DE, Rao M, Ferris CD, Snyder SH. Biliverdin reductase: A major physiologic cytoprotectant. *PNAS* 2002;99:16093-16098
15. Hammerman C, Goldschmidt D, Caplan MS, Kaplan M, Bromiker R, Eidelman AI, Gartner LM, Hochman A. Protective Effect of Bilirubin in Ischemia-Reperfusion Injury in the Rat Intestine. *J Ped Gastro Nutr* 2002;35:344-349