Current Clinical and Medical Education

Received 10 Apl 2024 | Revised 12 Apl 2024 | Accepted 25 Apl 2024 | Published Online 20 May 2024

Published By: Vision Publisher

ISSN 2942-0792

CCME 02 (5), 125-133

Original Article

The Lifestyle of Pregnant Women and Its Impact on the Health of the Child after Birth TSB is a Model

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Supplementary information The online version of this article (https://doi.org/xx.xxx/xxx.xx) contains supplementary material,which is available to autho-rized users.

Introduction:

Worldwide, unconjugated hyperbilirubinemia still threatens the health of many newborn infants. Approximately 80% of term newborn infants develop physiologic unconjugated hyperbilirubinemia, which is transient and benign in the vast majority of these infants [1]. A small, but non-negligible proportion may develop severe neonatal hyperbilirubinemia (SNH). An estimated one million newborn infants worldwide suffer from SNH, necessitating intensive treatment to reduce the risk of death or kernicterus spectrum disorders (KSD) [2]. The incidence of SNH varies between 2 and 42 per 100,000 live-born infants in high-income countries (HIC) and depends, at least in part, on the predefined total serum bilirubin (TSB) concentration for this diagnosis [3].

The estimated incidence of SNH is higher in low-income and middle-income countries (LMIC) where limited access to health care facilities and appropriate treatment threatens the health of thousands of newborn infants [4]. The estimated contribution of SNH and/or Rhesus disease to KSD is 73 per 100,000 live births and the mortality rate is 119 per 100,000 live births in Eastern Europe, Latin America, sub-Saharan Africa, and Asia [2]. These numbers are surpassed by a mortality rate for neonatal jaundice of 730 per 100,000 live births in India [5]. Worldwide, 114,000 infants may die per year [2]. These data underline the clinical relevance of screening methods for SNH identification. The primary purpose of screening is timely detection of newborns at risk of developing KSD, while diagnostic tests can provide the quantitative confirmation of the extent of jaundice [6].

During the last centuries, neonatal jaundice had already been recognized as a risk to the health of newborns [7]. Nevertheless, it was not until 1969 that Kramer systematically correlated dermal zones of jaundice with actual TSB [8]. A number of studies evaluated the accuracy and reliability of visual assessment of jaundice, a long-standing way of estimating the severity of hyperbilirubinemia. Correlation coefficients between visual assessment and TSB concentrations vary between 0.35 and 0.75 [9]. Aim of the study Studying the relationship between maternal lifestyle and its effect on bilirubin levels in newborns. Studying the effect of the type of birth and breastfeeding method on the bilirubin level in newborns.

Bilirubin

Bilirubin is a yellow bile pigment produced through the breakdown of red blood cells, which is known as hemolysis. Bilirubin is metabolized prior to excretion through the faces and urine. Bilirubin exists in two forms; unconjugated and conjugated. Unconjugated bilirubin is insoluble in water. This means it can only travel in the bloodstream if bound to albumin and it cannot be directly excreted from the body. In contrast, conjugated bilirubin is water soluble. This allows it to travel through the bloodstream without requiring transport proteins like albumin, which means that it can also be excreted out of the body [10,11]. Reticuloendothelial cells are macrophages which are responsible for the maintenance of the blood, through the destruction of old or abnormal cells. They take up red blood cells and metabolism the hemoglobin present into its individual components; haem and globin. Globin is further broken down into amino acids which are subsequently recycled. Meanwhile, haem is broken down into iron and biliverdin, a process which is catalysed by haem oxygenase. The iron gets recycled, while biliverdin is reduced to create unconjugated bilirubin [12].

Bilirubin Conjugation

In the bloodstream, unconjugated bilirubin binds to albumin to facilitate its transport to the liver. Once in the liver, glucuronic acid is added to unconjugated bilirubin by the enzyme glucuronyl transferase. These forms conjugated bilirubin, which is soluble. This allows conjugated bilirubin to be excreted into the duodenum in bile [13].

Bilirubin Excretion

Once in the colon, colonic bacteria DE conjugate bilirubin and convert it into urobilinogen. Around 80% of this urobilinogen is further oxidized by intestinal bacteria and converted to stercobilin and then excreted through faces. It is stercobilin which gives faces their color. Around 20% of the urobilinogen is reabsorbed into the bloodstream as part of the enterohepatic circulation. It is carried to the liver where some is recycled for bile production, while a small percentage reaches the kidneys. Here, it is oxidized further into urobilin and then excreted into the urine [14].

Measurement of Bilirubin Levels

Bilirubin level can be checked through biochemical method, Bilirubin- meter or transcutaneous bilirubin meter [15].

• Biochemical

The gold standard method for bilirubin estimation is the total and conjugated bilirubin assessment based on the van den Bergh reaction

• Bili meter

Spectrophotometry is the base of Bili meter and it assesses total bilirubin in the serum. Because of the predominant unconjugated form of bilirubin, this method has been found a useful method in neonates.

• Transcutaneous Bilirubinometer

This method is noninvasive and is based on the principle of multi wavelength spectral reflectance from the bilirubin staining in the skin the accuracy of the instrument may be affected by variation of skin pigmentation and its thickness [16].

Normal bilirubin level for all ages:

Table 2-1: Levels of Bilirubin

Total (elderly, adalt, child)	0.2- 1.5 mg/dl
Newborn	8 - 12.0 mg/dl
Critical value (adult)	> 1.5 mg/dl
Critical value (newborn)	> 15 mg/dl
Pre-hepatic unconjugated, indirect	0.2 -0.7 mg/L
Post-hepatic, conjugated, direct	0.4-1 mg/l
Fecal urobilinogen	to 280 mg/day 40
Urine	mg/dL 0.02 10 0.0

Increased Bilirubin Production

➢ Immune-mediated hemolysis Includes blood group incompatibilities such as ABO and Rhesus incompatibility [17].

➢ Non-immune mediated hemolysis includes RBC membrane defects like hereditary spherocytosis and elliptocytosis; RBC enzyme defects like glucose-6-phosphate dehydrogenase (G6PD) deficiency; pyruvate kinase deficiency; sequestration like cephalohematoma, subgaleal hemorrhage, intracranial hemorrhage; polycythemia, and sepsis [18].

Neonatal jaundice

Neonatal jaundice or neonatal hyperbilirubinemia results from elevated total serum bilirubin (TSB) and clinically manifests as yellowish discoloration of the skin, sclera, and mucous membrane. The term jaundice derives from the French word "jaune," which means yellow. It is the most commonly encountered medical problem in the first two weeks of life and a common cause of readmission to the hospital after birth [19]. Approximately 60% of term and 80% of preterm newborns develop clinical jaundice in the first week after birth. In most cases, it is a mild, transient, and self-limiting condition and resolves without treatment referred to as "physiological jaundice [20]." However, it is imperative to distinguish this from a more severe form called "pathological jaundice." Failure to identify and treat this entity may result in bilirubin encephalopathy and associated neurological sequelae. Unconjugated hyperbilirubinemia (UHB) is the cause of clinical jaundice in most neonates, but some infants with jaundice have conjugated hyperbilirubinemia (CHB), which is always pathological and signifies an underlying medical or surgical cause. The etiology of pathological UHB and CHB is vast and varied [21]. Preterm infants and those born with congenital enzyme deficiencies are particularly prone to the harmful effects of unconjugated bilirubin on the central nervous system. Severe hyperbilirubinemia can cause bilirubin-induced neurological dysfunction (BIND) and, if not treated adequately, may lead to acute and chronic bilirubin encephalopathy. Phototherapy and exchange transfusions are the mainstay of treatment of UHB, and a subset of patients also respond to intravenous immunoglobulin (IVIG) [22]. Treatment of CHB is more complex and depends mainly on the etiology. Despite advances in care and management of hyperbilirubinemia, it remains a significant cause of morbidity and mortality [23].

Types of Hyperbilirubinemia

Several types of bilirubinemia have been reported in neonates including physiological jaundice, pathological jaundice, jaundice due to breastfeeding or breast milk and hemolytic jaundice including three subtypes due to Rh factor incompatibility, ABO blood group incompatibility and Jaundice associated with Glucose-6-phosphate dehydrogenase (G6PD) deficiency [24].

Physiological Jaundice

It is the most abundant type of newborn hyperbilirubinemia, having no serious consequences. Neurodevelopmental abnormalities including as athetosis, loss of hearing, and in rare cases intellectual deficits, may be related to high toxic level of bilirubin. Jaundice attributable to physiological immaturity which usually appears between 24-72 h of age and between 4th and 5th days can be considered as its peak in term neonates and in preterm at 7th day, it disappears by 10-14 days of life. Unconjugated bilirubin is the predominant form and usually its serum level is less than 15 mg/dl. Based on the recent recommendations of the AAP, bilirubin levels up to 17-18 mg/dl may be accepted as normal in term of healthy newborns [25, 26].

Breast Feeding and Breast Milk Jaundice

Exclusively infants with breastfeeding have a different physiological pattern for jaundice compared with artificially feed babies. Jaundice in breast fed babies usually appears between 24-72 h of age, peaks by 5-15 days of life and disappears by the third week of life. Higher bilirubin levels have been reported in these infants [28].

Hemolytic Jaundice

The most common causes of hemolytic jaundice include (a) Rh hemolytic disease, (b) ABO incompatibility and (c) Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency and minor blood group incompatibility [29].

Rh Factor Hemolytic Disease Rhesus hemolytic disease of the newborns (RHDN) results from maternal red-cell alloimmunization. Maternal antibodies are produced against the fetal red blood cells, when fetal red blood cells are positive for a certain antigen, usually at what time a baby having Rh positive born to a Rh-negative mother (and Rh-positive father), then maternal immunoglobulin (IgG) antibodies might cross the placenta into the fetal circulation and cause a wide variety of symptoms in the fetus, ranging from mild to severe hemolytic anemia and fetal hydrops. To facilitate early treatment in neonates who are dubitable to have Rh factor, a blood group and Rh typing, DCT, PCV (packed cell volume) and serum bilirubin on cord blood should be performed [30].

A reticulocyte count should be sent before the first exchange transfusion (ET). Vigorous phototherapy is required immediately after the birth and it should be continued until a level, which is 5 mg/dl less than the level estimated for exchange blood transfusion. In preterm babies, lower values of intervention for treatment of Rh hemolytic disease have been demonstrated. Phototherapy and exchange blood transfusion are recommended when a level is greater than 0.5% and 1% birth weight (kg) respectively. Eight intravenous immunoglobulins (IVIG) can be used in a dose of 500 mg/kg 12 hourly x 2 doses after the first ET [31]. After the first ET starting of Phenobarbitone 5 mg/kg/day x 5 may be recommended. (b) ABO Incompatibility the incidence of the incompatibility of the ABO blood groups of the mother and fetus, when the mother has the blood group O and the newborn has the A or B blood group, is 15-20% of all pregnancies [32]. Babies with O-blood group mothers should be closely checked for and discharged after 72 h. Routine cord blood screening is not recommended for newborns with O-group mothers. Jaundice

owing to ABO incompatibility usually appears 24 h after the birth. In the presence of significant jaundice or jaundice appearing within 24 h, the work up for pathological jaundice should be done. Intensive phototherapy is advised at SB 12-17 mg/dl depending upon postnatal age of the baby. Instructions and Precautionary Measure for Parents during Physiological Jaundice [33].

The benign nature of jaundice should be explained and demonstrated to the parents. The mother should be encouraged to breast-feed her baby frequently and exclusively, at least eight to twelve times per day for initial several days, with no top feeds or glucose water whatsoever. Mother should be told to bring the baby to the hospital if the color on the legs looks as yellow as the face. Any newborn discharged before 48 h of life should be evaluated again in the next 48 h for breastfeeding sufficiency and development of jaundice [34].

Pathological Jaundice

Bilirubin levels with a deviation from the normal range and requiring intervention would be described as pathological jaundice. Appearance of jaundice within 24 h due to increase in serum bilirubin beyond 5 mg/dl/day, peak levels higher than the expected normal range, presence of clinical jaundice more than 2 weeks and conjugated bilirubin (dark urine staining the clothes) would be categorized under this type of jaundice [27].

Symptoms of Jaundice

Jaundice initially causes the skin to become yellow. Later, the gums, palms of the hands, and soles of the feet, as well as the whites of the eyes, may also develop a yellow color. These changes may be hard to see in children with darker skin or if a baby is unable to open their eyelids. The color change [35]:

Is noticeable first in the face, and then the chest, stomach area, arms, and then finally the legs. However, in some babies, the head-to-toe progression of jaundice may not be seen, and the jaundice may appear over the entire body like a tan.

Can be checked by pressing one finger on a baby's forehead or nose (called "blanching" the skin). If the skin is jaundiced, it will appear yellow when the finger is removed.

Can be followed in some babies by pressing over their bony prominences of their chest, hips, and knees to check if the jaundice is worsening.

Should be checked more than once before a baby leaves the hospital after birth. If you bring your baby home sooner than three days after birth, you should check their skin color every day until the next scheduled appointment.

The baby should be taken to see a doctor or nurse for a checkup within one to three days after going home. Signs of worsening jaundice - Call your baby's doctor if any of the following occurs:

The yellow color is visible at the knee or lower, is darker in appearance (changing from a lemon yellow to an orange yellow color), or if the "whites" of the eyes appear yellow:

- Your baby has a fever
- Your baby has any difficulty eating
- Your baby is more sleepy than usual
- It is hard to wake up your baby
- Your baby is irritable and is difficult to console
- Your baby arches their neck or body backwards [36,37,38].

Causes of Jaundice

Jaundice is caused by the build-up of bilirubin in the blood. Bilirubin is formed and produced when red blood cells are broken down. Bilirubin (a yellow substance) is naturally removed by the liver and then excreted in stool and urine. Bilirubin levels become high when bilirubin is made faster than it can be removed. Jaundice is common in newborns since two to three times more bilirubin is made than in adults [39]. Newborn jaundice affects nearly all babies and is caused by a mild to moderate increase in bilirubin levels and is usually not harmful. It often reaches highest levels three to four days after birth and usually goes away by one to two weeks after birth. In babies who are born at 38 weeks or less and those who are significantly jaundiced, the jaundice may require more time to go away, as normal elimination processes develop with age [40]. Newborns with higher levels of bilirubin in the blood have what is called "severe hyperbilirubinemia," a more serious condition. Babies could develop severe hyperbilirubinemia within the first day after birth. If your baby becomes very yellow, it's very important to call or see your doctor immediately [41].

Complications of Jaundice

In babies whose blood bilirubin levels reach harmful levels, bilirubin may get into the brain and cause reversible damage (called acute bilirubin encephalopathy) or permanent damage (called kernicterus or chronic bilirubin encephalopathy). Frequent monitoring and urgent, early treatment of babies at high risk for jaundice helps to prevent severe hyperbilirubinemia [45].

Diagnosis of Jaundice

Newborn jaundice can be detected by examining the baby and testing bilirubin levels in the blood. The blood test

involves collecting a small amount (less than one-half teaspoon) of blood. Results of blood testing are available in most hospitals within a few hours. Jaundice after one week of age should be checked to see if it is from a serious condition (eg, a blockage of bilirubin removal in the liver). In some hospitals, screening for high bilirubin is at first performed by a device that measures bilirubin in the skin (referred to as "transcutaneous" screening). When the skin measurement exceeds a normal value, blood testing is done to make sure that level of bilirubin is accurate [46,47].

Prevention of Sever Hyperbilirubinemia

Prevention of severe hyperbilirubinemia is important in avoiding serious complications. Babies who are at risk for hyperbilirubinemia need to have regular follow-up visits with their doctor; these should be scheduled at the time of hospital discharge. The following information only applies to babies who are

healthy and born at term or late preterm (within a month of their due date). Screening Leading experts recommend that all newborns have their bilirubin levels tested before going home from the hospital, regardless of age. This is especially true for babies who are jaundiced before one day of age, in which case repeated testing is needed [48,49].

Monitoring Parents, other caregivers, and health care providers should watch babies closely if jaundice develops. Hyperbilirubinemia is usually easy to prevent and treat initially. However, complications can be serious and irreversible if treatment is delayed. You should contact your baby's health care provider immediately if you are concerned about worsening jaundice. Prompt treatment Babies with high bilirubin levels should be treated by a qualified health care provider to safely reduce bilirubin levels and prevent the risk of brain damage. Parents and health care providers should not delay treatment for any reason [50,51].

Treatments of Jaundice

The goal of treating jaundice is to efficiently and safely reduce the level of bilirubin. Babies with mild hyperbilirubinemia may need no treatment at all. Babies with higher bilirubin levels will need brief treatment, which is described below. Jaundice is common in premature babies (those born before 38 weeks). Premature babies are more vulnerable to hyperbilirubinemia because brain toxicity occurs at lower levels of bilirubin than in term babies. As a result, premature babies are treated at lower levels of bilirubin but with the same treatments discussed below [52].

Frequent feeding Providing adequate breast milk is an important part of preventing and treating jaundice because it helps in the removal of bilirubin in stools and urine. If your baby is not getting enough milk through breastfeeding, your doctor can talk to you about options such as supplementing with formula or donor breast milk. You will know that your baby is getting enough milk if they have at least six wet diapers per day, the color of their stool's changes from dark green to yellow, and they seems satisfied after feeding. Phototherapy ("light"therapy) is the most common medical treatment for hyperbilirubinemia in babies. In most cases, phototherapy is the only treatment required [53].

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