International Journal of Research and Development in Pharmacy and Life Sciences
Available online at http//www.ijrdpl.com
June - July, 2016, Vol. 5, No.4, pp 2198-2200
ISSN (P): 2393-932X, ISSN (E): 2278-0238

Review Article

NEONATAL JAUNDICE - A REVIEW

Biswajit Batabyal [1]*, Sudipta Chakraborty[2]

1. Midland Diagnostic Lab, Belgharia, Kolkata, West Bengal, India.
2. Gouridevi Institute of Medical Science, Durgapur, Burdwan, West Bengal, India.

***Corresponding author's Email:** biswajit.batabyal@gmail.com

(Received: April 10, 2016; **Accepted:** May 14, 2016**)**

ABSTRACT

Newborn jaundice occurs when a baby has a high level of bilirubin in the blood. Bilirubin is a yellow substance that the body creates when it replaces old red blood cells. The liver helps break down the substance so it can be removed from the body in the stool. Neonatal jaundice is common, and usually harmless, because of physiological jaundice or breast-feeding. In some neonates unconjugated bilirubin concentration, coupled with other risk factors, is sufficient to allow free bilirubin to cross the blood-brain barrier and cause kernicterus. Another subgroup of infants is jaundiced because of elevated conjugated bilirubin; a marker for a number of pathological conditions. Bilirubin measurement must identify those infants at risk. Transcutaneous bilirubin measurement is increasingly used in healthy infants, especially before early discharge or at home, to assess the need for laboratory bilirubin measurement. Transcutaneous measurements are not covered by laboratory quality assessment schemes. Guidelines on management of neonatal jaundice utilize age in hours and other risk factors to define bilirubin action thresholds, which may be as low as 100 µmol/L for sick premature infants, whereas early discharged babies may only present after bilirubin concentrations are extremely high. Hence, there is a requirement for accurate total bilirubin measurement from <100 to >500 µmol/L, with sufficient precision to assess the rate of bilirubin change with time. Babies presenting with late jaundice always require conjugated bilirubin measurement. It is of concern that many total and direct bilirubin automated kit methods suffer from haemolysis interference, while use of in-house methods or modification of commercial methods has virtually disappeared. External quality assessment has a vital role in providing data on different methods' performance, including accuracy, precision and susceptibility to interference. Laboratories should consider whether their adult bilirubin methods are suitable for neonates.

Keywords: Neonatal jaundice, Cause, Treatment.

INTRODUCTION

Neonatal jaundice or neonatal hyperbilirubinemia, or neonatal icterus (from the Greek word Ἴκτερος), attributive adjective: icteric, is a yellowing of the skin and other tissues of a newborn infant. A bilirubin level of more than 85 µmol/l (5mg/dL) leads to a jaundiced appearance in neonates whereas in adults a level of 34 µmol/l (2 mg/dL) is needed for this to occur. In newborns, jaundice is detected by blanching the skin with pressure applied by a finger so that it reveals underlying skin and subcutaneous tissue.[1] Jaundiced newborns have yellow discoloration of the white part of the eye, and yellowing of the face, extending down onto the chest. Neonatal jaundice can make the newborn sleepy and interfere with feeding. Extreme jaundice can cause permanent brain damage from kernicterus. In neonates, the yellow discoloration of the skin is first noted in the face and as the bilirubin level rises proceeds caudal to the trunk and then to the extremities.[2] This condition is common in newborns affecting over half (50–60%) of all babies in the first week of life.[3]

Infants whose palms and soles are yellow, have serum bilirubin level over 255 µmol/l (15 mg/dL) (more serious level). Studies have shown that trained examiners assessment of levels of jaundice show moderate agreement with

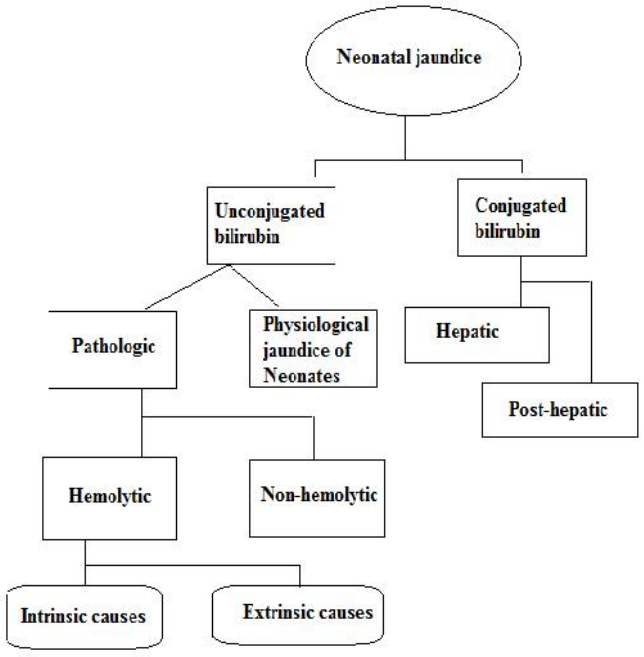
icterometer bilirubin measurements.[2] In infants, jaundice can be measured using invasive or non-invasive methods.

CAUSES

In neonates, jaundice tends to develop because of two factors - the breakdown of fetal hemoglobin as it is replaced with adult hemoglobin and the relatively immature metabolic pathways of the liver, which are unable to conjugate and so excrete bilirubin as quickly as an adult. This causes an accumulation of bilirubin in the blood (hyperbilirubinemia), leading to the symptoms of jaundice.

If the neonatal jaundice does not clear up with simple phototherapy, other causes such as biliary atresia, Progressive familial intrahepatic cholestasis, bile duct paucity, Alagille syndrome, alpha 1-antitrypsin deficiency, and other pediatric liver diseases should be considered. The evaluation for these will include blood work and a variety of diagnostic tests. Prolonged neonatal jaundice is serious and should be followed up promptly.

Severe neonatal jaundice may indicate the presence of other conditions contributing to the elevated bilirubin levels, of which there are a large variety of possibilities (see below). These should be detected or excluded as part of the differential diagnosis to prevent the development of complications. They can be grouped into the following categories:



Physiological jaundice

Most infants develop visible jaundice due to elevation of unconjugated bilirubin concentration during their first week. This common condition is called physiological jaundice. This pattern of hyperbilirubinemia has been classified into two functionally distinct periods.

Phase I

1. Term infants - jaundice lasts for about 10 days with a rapid rise of serum bilirubin up to 204 µmol/l (12 mg/dL).

2. Preterm infants - jaundice lasts for about two weeks, with a rapid rise of serum bilirubin up to 255 µmol/l (15 mg/dL).

Phase II:

Bilirubin levels decline to about 34 µmol/l (2 mg/dL) for two weeks, eventually mimicking adult values.

1. Preterm infants - phase two can last more than one month.

2. Exclusively breastfed infants - phase two can last more than one month.

Mechanisms involved in physiological jaundice are mainly:

• Relatively low activity of the enzyme glucuronosyltransferase which normally converts unconjugated bilirubin to conjugated bilirubin that can be excreted into the gastrointestinal tract.[4] Before birth, this enzyme is actively down-regulated, since bilirubin needs to remain unconjugated in order to cross the placenta to avoid being accumulated in the fetus.[5] After birth, it takes some time for this enzyme to gain function.

• Shorter life span of fetal red blood cells,[4] being approximately 80 to 90 days in a full term infant,[6] compared to 100 to 120 days in adults.

• Relatively low conversion of bilirubin to urobilinogen by the intestinal flora, resulting in relatively high absorption of bilirubin back into the circulation.[4]

Diagnosis

Clinical Assessment

This method is less accurate and more subjective in estimating jaundice.

Ingram icterometer: In this method a piece of transparent plastic known as Ingram icterometer is used. Ingram icterometer is painted in five transverse strips of graded yellow lines. The instrument is pressed against the nose and the yellow colour of the blanched skin is matched with the graded yellow lines and bilirubin level is assigned.

Transcutaneous bilirubinometer: This is hand held, portable and rechargeable but expensive and sophisticated. When pressure is applied to the photoprobe, a xenon tube generates a strobe light, and this light passes through the subcutaneous tissue. The reflected light returns through the second fibre optic bundle to the spectrophotometric module. The intensity of the yellow color in this light, after correcting for the haemoglobin, is measured and instantly displayed in arbitrary units.

Any of the following features characterizes pathological jaundice:

1. Clinical jaundice appearing in the first 24 hours or greater than 14 days of life.
2. Increases in the level of total bilirubin by more than 8.5 µmol/l (0.5 mg/dL) per hour or (85 µmol/l) 5 mg/dL per 24 hours.
3. Total bilirubin more than 331.5 µmol/l (19.5 mg/dL) (hyperbilirubinemia).
4. Direct bilirubin more than 34 µmol/l (2.0 mg/dL).

The aim of clinical assessment is to distinguish physiological from pathological jaundice. The signs which help to differentiate pathological jaundice of neonates from physiological jaundice of neonates are the presence of intrauterine growth restriction, stigma of intrauterine infections (e.g. cataracts, small head, and enlargement of the liver and spleen), cephalohematoma, bruising, signs of bleeding in the brain's ventricles. History of illness is noteworthy. Family history of jaundice and anemia, family history of neonatal or early infant death due to liver disease, maternal illness suggestive of viral infection (fever, rash or lymphadenopathy), maternal drugs (e.g. sulphonamides, anti-malarials causing red blood cell destruction in G6PD deficiency) are suggestive of pathological jaundice in neonates. [7]

TREATMENT

The bilirubin levels for initiative of phototherapy varies depends on the age and health status of the newborn. However, any newborn with a total serum bilirubin greater than 359 µmol/l (21 mg/dL) should receive phototherapy.[8]

CONCLUSION

The high incidence of neonatal jaundice combined with the shortening of post natal stay at hospital make the early screening and surveillance for neonatal hyperbilirubinemia essential to ensure that these infants are not missed, as it is still not known at what level bilirubin can cause a significant risk of brain damage. So before discharge, every newborn infant should be assessed for the risk of hyperbilirubinemia. The predischarge bilirubin measurements using TSB or TCB and/or assessment of clinical risk factors are used as screening tools for predicting the neonatal hyperbilirubinemia.

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