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NEONATAL JAUNDICE

Saara Raj*

India.

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***Corresponding author: Saara Raj**
India.

BACKGROUND

- Jaundice is the most common condition that requires medical attention and hospital readmission in newborns. The yellow coloration of the skin and sclera in newborns with jaundice is the result of accumulation of unconjugated bilirubin. In most infants, unconjugated hyperbilirubinemia reflects a normal transitional phenomenon. However, in some infants, serum bilirubin levels may rise excessively which can be cause for concern because unconjugated bilirubin is neurotoxic and can cause death in newborns and lifelong neurologic sequelae in infants who survive. For these reasons, the presence of neonatal jaundice frequently results in diagnostic evaluation.
- The good news is that in most cases new born jaundice goes away on its own as a babies liver developes and as a baby beings to feed, which helps bilirubin pass through the body .

INTRODUCTION

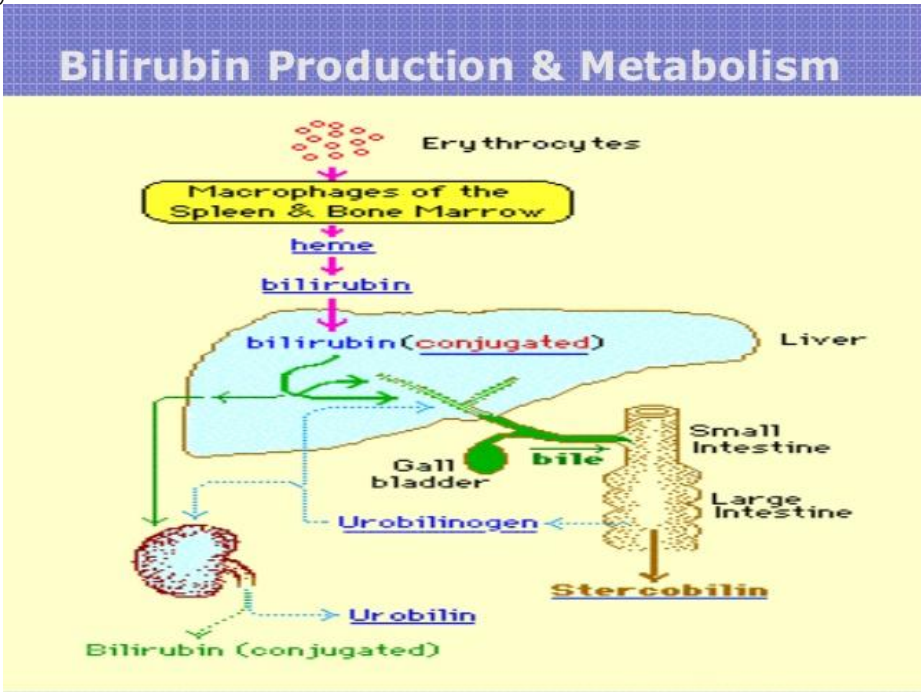
- The term jaundice is from the French word "jaune," which means yellow, Neonatal jaundice in most Newborn's is a mild and transient event. Neonatal jaundice may have first been described in a Chinese textbook 1000 years ago. Medical theses, essays, and textbooks from the 18th and 19th centuries contain discussions about the causes and treatment of neonatal jaundice

ETIOLOGY

- Neonates is due to either physiologic or pathologic causes, Over 75% of neonatal unconjugated hyperbilirubinemia is due to physiologic causes. Physiologic jaundice is also referred to as non-pathologic jaundice, and it is mild and transient. This occurs because of differences in the metabolism of bilirubin in the neonatal period leading to an increased bilirubin load. The increased bilirubin load in the New born arises from increased production of bilirubin due to a higher mass of red blood cells with a decreased lifespan in the neonate, a decreased bilirubin clearance from a deficiency of the uridine diphosphate glucuronosyltransferase enzyme, which in the New born has the activity of about 1% of the adult liver, and increased enterohepatic circulation.
- Causes of increased bilirubin production in pathologic jaundice are immune-mediated Hemolysis such as ABO and Rhesus incompatibility, non-immune mediated causes such as Cephalhematoma, red blood cell membrane defects like hereditary spherocytosis and elliptocytosis, enzyme defects like glucose-6-phosphate dehydrogenase deficiency and pyruvate kinase.
- ABO incompatibility occurs in mother's with blood group 'O' who have anti-A and anti-B IgG antibodies that cross the placenta and cause Hemolysis in newborns with blood group A or B. In Rhesus incompatibility, an Rh-negative mother who has been exposed to Rh-positive blood from a previous pregnancy becomes sensitized, causing hemolysis in the fetus with Rh-positive blood. Using RhoGAM(prescribed medicine that is used to prevent Rh immunizations the condition in which an individual blood developes anti bodies after exposure to Rh positive blood. RhoGAM is administered by intermuscular(IM) injection. RhoGAM is purified from human plasma containing anti-Rh(anti-D)).As prophylaxis in a mother with prior exposure has decreased the incidence of Rh hemolysis, which, although less common than ABO incompatibility, is more severe.
- Babies who aren't getting enough breast milk or formula either because there are having a hard time feeding or because their mothers milk is not in yet.
- Bruishing at birth or internal bleeding

- Un-infection and enzyme deficiency and abnormality in red plate cells.

Pathophysiology



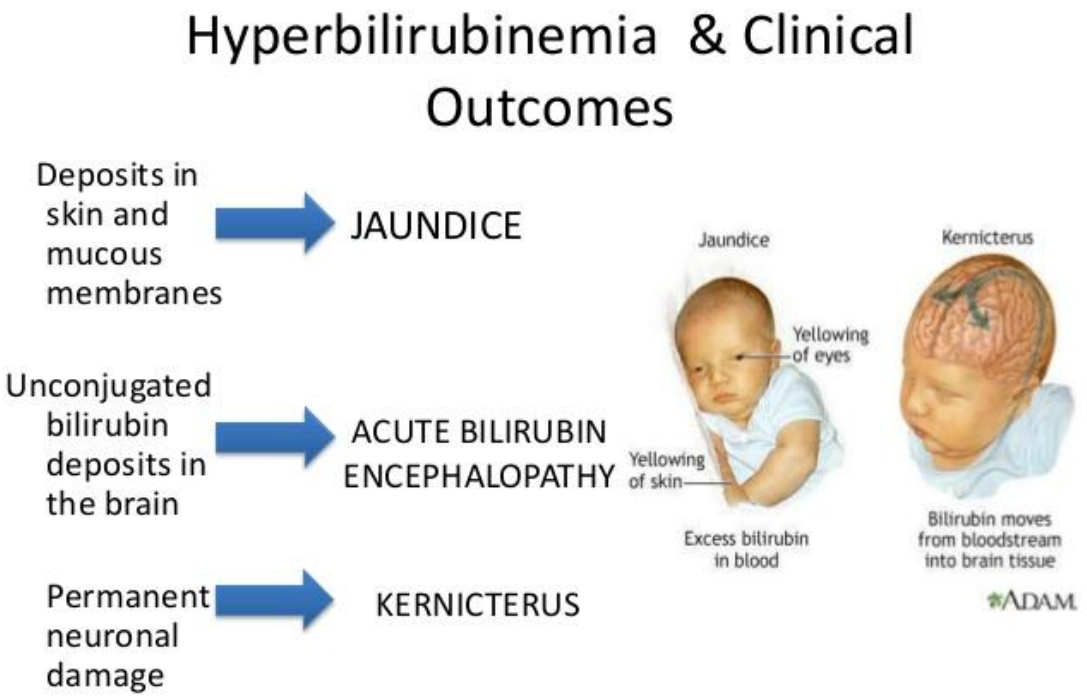
- Most bilirubin is produced when hemoglobin is broken down into unconjugated bilirubin (and other substances). Unconjugated bilirubin binds to albumin in the blood for transport to the liver, where it is taken up by hepatocytes and conjugated with glucuronic acid to make it water soluble. Conjugated bilirubin is excreted in bile into the duodenum. In the intestine, bacteria metabolize bilirubin to form urobilinogen. Some urobilinogen is eliminated in the feces, and some is reabsorbed, extracted by hepatocytes, reprocessed, and re-excreted in bile (enterohepatic circulation

Mechanisms of hyperbilirubinemia

- Hyperbilirubinemia may involve predominantly unconjugated or conjugated bilirubin.
  Unconjugated hyperbilirubinemia is most often caused by one of the following;
- Increased production
- Decreased hepatic uptake
- Decreased conjugation
  Conjugated hyperbilirubinemia is most often caused by ≥ 1 of the following:
- Dysfunction of hepatocytes (hepatocellular dysfunction)
- Slowing of bile egress from the liver (intrahepatic cholestasis)
- Obstruction of extrahepatic bile flow (extrahepatic cholestasis)
  Consequences
- Outcome is determined primarily by the cause of jaundice and the presence and severity of hepatic dysfunction. Hepatic dysfunction can result in coagulopathy, encephalopathy, and portal hypertension (which can lead to gastrointestinal bleeding).

Sings and symptoms of jaundice

- The first sigh of jaundice is a yellowing of a babies skin and eye the yellowing may begin within two to four days after birth and may start in the face before spreading down across the body.
- Bilirubin level typically peak between three to seven days after birth in a finger lightly pressed on a babies skin cause that area of skin become yellow it is likely a sign of jaundice.



- The jaundice spreads or become more intense baby develops fever over 100*F (38*c) babies yellow coloring deepens
- the baby feeds poorly appears listless or lethargic and makes high pitched cries

Diagnosis

- Urine test: It is used to measure levels of a substance called urobilinogen. Urobilinogen is produced when bacteria break down bilirubin inside the digestive system.
- Blood test : Blood tests include blood levels of enzymes found primarily from the liver, such as the aminotransferases (ALT, AST), and alkaline phosphatase (ALP), bilirubin (which causes the jaundice), and protein levels, specifically, total protein and albumin.
- Other primary lab tests for liver function include gamma glutamyl transferase (GGT) and prothrombin time (PT).

Treatment

➢ Mild infant jaundice often disappears on its own within two or three weeks. For moderate or severe jaundice, your baby may need to stay longer in the newborn nursery or be readmitted to the hospital.

Treatments to lower the level of bilirubin in your baby's blood may include.

➢ Enhanced nutrition. To prevent weight loss, your doctor may recommend more-frequent feeding or supplementation to ensure that your baby receives adequate nutrition.

Light therapy (phototherapy)

➢ Your baby may be placed under a special lamp that emits light in the blue-green spectrum. The light changes the shape and structure of bilirubin molecules in such a way that they can be excreted in both the urine and stool. During treatment, your baby will wear only a diaper and protective eye patches. Light therapy may be supplemented with the use of a light-emitting pad or mattress.

Intravenous immunoglobulin

➢ Jaundice may be related to blood type differences between mother and baby. This condition results in the baby carrying antibodies from the mother that contribute to the rapid breakdown of the baby's red blood cells. Intravenous transfusion of an immunoglobulin — a blood protein that can reduce levels of antibodies — may decrease jaundice and lessen the need for an exchange transfusion, although results are not conclusive.

Exchange transfusion

➢ Rarely, when severe jaundice doesn't respond to other treatments, a baby may need an exchange transfusion of blood. This involves repeatedly withdrawing small amounts of blood and replacing it with donor blood, thereby diluting the bilirubin and maternal antibodies — a procedure that's performed in a newborn intensive care unit.

Evaluation

- Bilirubin levels may be assessed using a transcutaneous measurement device or taking blood for total serum or plasma level determination. Transcutaneous measurement decreases the frequency of blood tests for bilirubin but is limited by dark skin tone and if the neonate has received phototherapy Also, if the transcutaneous bilirubin level exceeds the 95th percentile on the transcutaneous nomogram or 75% of the total serum bilirubin nomogram for phototherapy, the total serum bilirubin level should be measured.
- Recommended labs to identify the hemolytic disease as a cause of unconjugated hyperbilirubinemia are the neonate's blood group, Coombs test, complete blood cell (CBC), reticulocyte count, blood smear, and G6PD(it is a genetic disorder which commonly affects male's it happens when the body doesn't have enough of an emzyme). In patients with conjugated hyperbilirubinemia, the serum aminotransferases should be ordered for evidence of hepatocellular injury, gamma-glutamyl transferase (GGTP) levels for evidence of hepatobiliary disease and prothrombin time and serum albumin to evaluate for hepatic synthetic function.
- Imaging studies like ultrasonography and additional tests like TORCH titers(the TORCH screen is a group of blood test these test check for several different infections in a newborn the full form of TORCH is Toxoplasmosis, Rubella, Cytomegalovirus, Herpes simplex, and HIV. However it can also contain other newborn infections). Urine culture, viral cultures, serologic titers, amino acids, and the a-antitrypsin phenotype may be added depending on the suspected diagnosis for conjugated hyperbilirubinemia.

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