

## A commentary on Neonatal Jaundice (Icterus)

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### INTRODUCTION

Neonatal jaundice also called icterus; it is a yellow pigmentation of the skin and sclera that occurs when total bilirubin levels rise above the 95<sup>th</sup> percentile of age, usually about 2 mg/dL. Total hyperbilirubinemia can be caused mainly by indirect bilirubin or may be caused by conjugated or direct bilirubin and is highly dependent on where bilirubin metabolism is disrupted. Thus jaundice can be thought of as pre hepatic, hepatocellular, or post hepatic. If total bilirubin levels are elevated and total bilirubin levels are normal, that means there is an unresolved amount of bilirubin. Chronic hyperbilirubinemia is almost completely normal, called physiologic jaundice in the newborn. It is extremely common and grows in most infants, especially preterm infants, in the first two days to one week of life.

At birth, newborns have a high hematocrit, but red blood cells have a shorter lifespan. As a result, when the large numbers of red cells are converted, more hemoglobin is released. That hemoglobin is converted to unintended bilirubin. Now, newborn livers do not work well for bilirubin synthesis and in secreting this concentrated bilirubin in the intestinal tract, and that dysfunction leads to unintended hyperbilirubinemia. Generally, this is completely dangerous, as the total amount of bilirubin increases and is automatically resolved within one week in full-time babies or 2 weeks in premature babies.

If untreated, hyperbilirubinemia occurs in the first 24 hours after birth or lasts more than one week in older children or 2 weeks in premature babies or the baby shows signs or symptoms of serious illness, jaundice is considered pathologic and most likely due to pre hepatic cause. In Rh incompatibility, the mother should be Rh negative and the father should be Rh positive.

During the delivery of the first child with Rh, some of the baby's red blood cells may enter the mother's circulation. These red cells are found in the mother's immune system, and in response

the anti-rhesus antibodies of the mother are produced. When a second baby is born, if that baby is also Rh positive, then there are already Rh-produced anti-Rh antibodies from the previous pregnancy, which can cross the placenta and destroy the baby's red blood cells. To prevent this, all non-Rh mothers should be given anti-rhesus antibodies or gamma globulins, also called RhoGAM at 28 weeks of gestation and within 72 hours of giving birth. These anti-rhesus antibodies bind to any red Rh blood cells from the baby that enters the mother's circulation, preventing the mother's anti-rhesus antibodies from producing.

In ABO incompatibility, the most common condition is that the mother has group O and the baby has type A or B blood type. This is because A and B antigens are similar to normal natural antigens, such as viruses, dust, or pollen, present around them, so the exposure of these antigens triggers the production of anti-A and anti-B antigens. These antibodies therefore cross the placenta and cause hemolysis right from the first pregnancy. If the hemoglobin level is low, there may be blood clots outside the arteries, for example, cephalohematoma due to trauma during childbirth, which then decreases and is regenerated as bilirubin.

If hemolysis is due to a deficiency of red blood cells, such as hereditary spherocytosis or elliptocytosis, then a blood smear will show spherocytes or elliptocytes. Lower G6PD deficits and pyruvate kinase levels will be lower than pyruvate kinase deficits. Also, hemoglobin electrophoresis can produce hemoglobinopathies, such as thalassemia or sickle cell disease, especially hemoglobin electrophoresis in the newborn with two components: HbF 80% and HbA 20%. In the event that sickle cell HbA will be absent and infected with HbS, while in severe th-thalassemia HbA will also be absent but replaced. Now, when reticulocytes, LDH, and haptoglobin are all normal, jaundice is often associated with breastfeeding also called breastfeeding jaundice.

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## CONFLICT OF INTEREST

The author has declared that no competing interests exist.