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"Neonatal Jaundice-An Overview"

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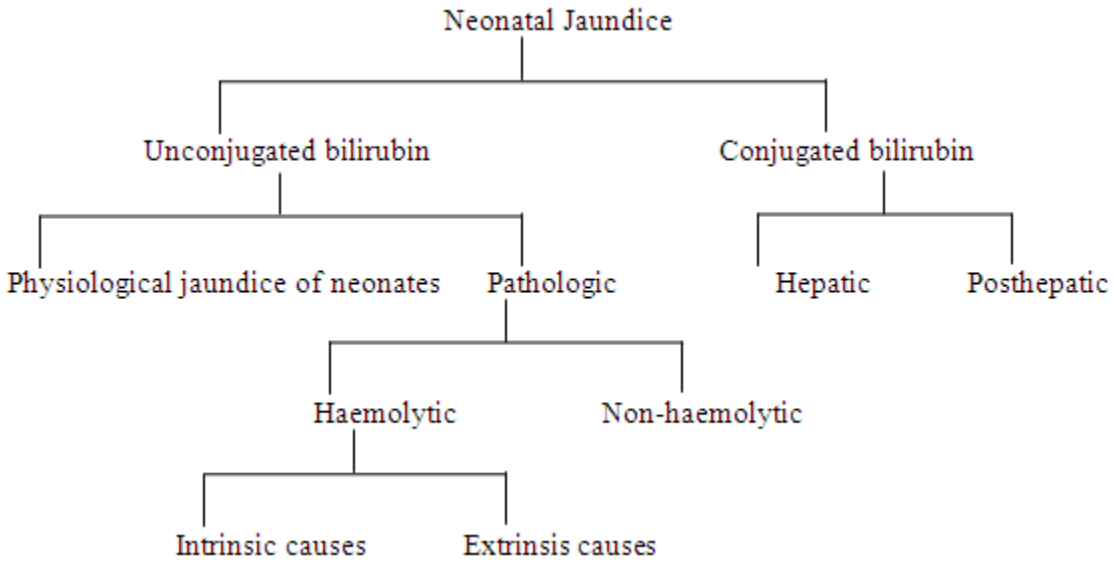
INTRODUCTION

Neonatal Jaundice is a yellowish discoloration of white part of the eyes and skin in a new born baby due to high bilirubin levels. It includes excess sleepiness and poor feeding. More than 80% of new borns will exhibit jaundice all new borns will have a serum bilirubin level that a higher than the adult norm, but the level of hyper bilirubinemia [bilirubin level more than 18mg/dl] requiring treatment is determined by age in hours and risk factors for developing reverse jaundice [1, 2, 3, 4].

Causes

In new borns jaundice tends to develop because of two factors the breakdown of fetal haemoglobin as it is replaced with adult haemoglobin which are unable to conjugate and so excrete bilirubin as qucikly as an adult this cause accumulation of bilirubin in the blood leading to symptoms of jaundice. If neonatal jaundice does not clear up with single therapy other causes such as biliary atresia, progressive familial intrarhepatic cholestasis, alpha 1 - antitrypsin deficiency and other Pediatrics liver disease should be considered. [5]

The causes of neonatal jaundice can be grouped into the following categories.



I. Unconjugated

(i) Hemolytic

Intrinsic causes of haemolysis

(a) Membrane conditions

- Spherocytosis
- Hereditary elliptrocytosis [33]

(b) Enzyme conditions

- Glucose - 6 - phosphate dehydrogenase deficiency (also called G6PD deficiency)
- Pyrurate kinase deficiency. [35]

(c) Globin synthesis defect

- Sickle cell Anaemia
- Alpha thalasemia [33]

(ii) Extrinsic causes of hemolysis

(a) Systemic conditions

- Sepsis
- Arteriovenous malformation

(b) Alloimmunity

(c) Hemolytic disease of new born (ABO) [33]

(d) RH disease [6]

Other blood type mismatches causing hemolytic disease of the new born. [7]

Non-Haemolytic causes [33]

- Breast feeding jaundice
- Breast milk jaundice
- Cephalhaematoma
- Polycythemia
- Urinary tract infection
- Sepsis
- Hypothyroidism
- Gilbert's Syndrome
- Crigler - Najjar Syndrome
- High G 1 Obstruction (Pyloric stenosis, Bowel obstruction) [8, 9]

Conjugated (Direct)

Liver causes

- Infections
- Sepsis
- Hepatitis A
- Hepatitis B
- TORCH infection
- Galactosemia
- Alphas - 1 - antitrypsin deficiency, which is commonly missed and must be considered in DDX.
- Cystic fibrosis
- Dubin Johnson Syndrome.
- Rotor Syndrome

Drugs

- Total parenteral nutrition
- Idiopathic

Post hepatic causes

- Biliary Atresia or bile duct obstruction
- Choledochal cyst. [33]

Physiological Jaundice

- Most infants develop visible jaundice due to the elevation of unconjugated bilirubin concentration during their first week. This is called physiological jaundice. This pattern of hyper bilirubinemia has been classified into two phases. [33]

Phase One

(1) Term infants: - Jaundice last for about 10 days with a rapid rise of serum bilirubin up to 12 mg/dl.

(2) Preterm infants :- Jaundice last for about two weeks with a rapid rise of serum bilirubin up to 15 mg/dl

Phase Two

Bilirubin levels decline to about 2 mg/dl for two weeks, eventually mimicking adult values.

1. Preterm infants: - Phase two can last more than one month.
2. Exclusively breast feed infants - phase two can last more than one month.

Breast feeding Jaundice

Or lack of breast feeding Jaundice is caused by insufficent breast milk intake [10] resulting in inadequate bowel movements to remove bilirubin from the body. This lead to increased enterohepatic circulation resulting in increased reabsorption of bilirubin from the intestine [11] occurring in the first week of life, most cases can be ameliorated by frequent breast feeding sessions of sufficient duration to stimulate adequate milk production.

Pathological Jaundice

Any of the following features suggests pathological jaundice:

(1) Clinical Jaundice appearing in the first 24 hours or greater than 14 days of life.
(2) Increased in the level of total bilirubin by more than 0.5 mg/dl/hour or 5 mg/dl/24 hour.
(3) Total bilirubin more than 19.5 mg/dl hyperbilirubinaemia.
(4) Direct bilirubin more than 2.0 mg/dl. [12]

"Pathophysiology of Neonatal Jaundice"

Bilirubin mainly originates from heme via the catalization of hemeoxygenize (HO) and biliverdin reductase and is conjugated into direct from by UDP - glucuronyl transferase for subsequent biliary secretion [13] Jaundice develops when conjugated and unconjugated bilirubin deposits onto the skin it is very common during neonatal period. Neonatal Jaundice is related to a verity of physiologic and pathologic conditions [29] physiologic aspects are comprised of increased bilirubin production, less hepatic conjugation and enhanced enterohepatic circulation [30]

Shorter life span of fetal red blood cell being approx 80 - 90 days in a full term infant [14] compared to 100 to 120 days in a full term.

In Pathologic condition there are two patterns of diseases: Hyperbilirubinaemia and cholestasis. Hyperbilirubinemia refers accumulation of Unconjugated bilirubin beyond the extent of physiological jaundice, while cholestasis results from the excretory obstruction of conjugated bilirubin. Neonatal hyperbilirubinemia carries a potential risk of Kernicterus, [15] proper management is of paramount important to avoid the complications. Etiological verification is essential because the underlying diseases are critical factors of neurological sequalae (16). There is a wide range of conditions that affect bilirubin levels including environmental and genetic origin. These events may lead to destruction of RBC delayed metabolism and increase absorption of bilirubin [17] The most important maternal effect on Neonatal hyperbilirubinaemia is isoimmune hemolytic disease. The clinical manifestation is early onset is early onset of hyperbilirubinemia with Anaemia [18]. In addition genetic interactions could enhance the severity of Neonatal hyperbilirubiaemia [19, 20]

Both enviromental and genetic factors are involved in the development of breast milk jaundice [21, 22]

DIAGNOSIS

(i.) Transcutacous bilirubin measurement provides more accurate information than clinical assessment this is done via a device (e.g. - Konica Minolta Drager Air shields JM - 103 or the Bilichek) which measures the amount of yellow color in subcutaneous tissue, converting it into an estimate of total serum bilirubin level. It is not invasive test that can be done at bedside.

(ii.) Diagnosis in often by measuring the serum bilirubin level in the blood [23] is the best method in predicting severe hyperbilirubinemia.

Umbilical Cord blood [Total Serum bilirubin] can also be measured, and should be sent for evaluation at birth, if mother was not tested for ABO and Rh blood types.

Treatment

Treatment depends upon the severity of hyperbilirubinemia, of etiology and risk of developing severe neurological complications.

Phototherapy

The mainitay of treatment for hyperbilirubinemia is phototherapy. It is effective within 24 - 36 hours of life and is effective in decreasing rates of exchange transfusion and in preventing progression to severe hyperbilirubinemia in infants with moderately elevated levels.

Phototherapy is initiated based on the individual risk of developing severe hyperbilirubinemia. In low risk infants the threshold to begin Phototherapy is at serum total bilirubin above 95th percentile. Treatment should be stopped once total bilirubin is below the treatment threshold.

Phototherapy is which works by changing transbilirubin into water soluble cis bilirubin [17,18, 25,31,32] generally quite safe and complications are very rare, but include burns, retinal damage, thermoregulatory instability, loose stools, dehydration skin rash and tanning of the skin.

Phototherapy should not be used in infants with conjugated hyperbilirubinemia since exertion is the issue and not conjugation. Bronze baby syndrome occurs in these cases. [8]

(2) Exchange transfusion

In exchange transfusion aliquots of the infants blood are removed and equal amounts of donor whole blood are transfused. This process aims to remove bilirubin in the serum, as well as partially hemolysed and antibody coated red blood cells. This treatment is considered in infants with total bilirubin concentration between 375 U mol/L and 425 U mol/L [24, 25] without response to intensive phototherapy in the presence of severe anaemia or hemolytic disease or rapid rise in bilirubin (> 17 U mol/L in less than 6 hours).

Exchange transfusion:

Indications: **-** All infants with symptoms and signs of Excessive bilirubin accumulation should receive immediate exchange transfusion.

- Additional investigation should be completed before exchange transfusion.
- Exchange transfusion is associated with significant morbidity. Complications include air embolism, vaso spasm, infarction, infection and death[24, 25].

COMPLICATIONS

Prolonged hyperbilirubinaemia (Severe Jaundice) can result in chronic bilirubin encephalopathy (Kernicterus) [26, 27]. Quick and accurate treatment of neonatal jaundice helps to reduce the risk of developing kern icterus [28].

Exchange transfusion performed to lower high bilirubin levels are an aggressive.

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