PODCAST No. 16

INTRODUCTIONS:

FEEDBACK:

Topic: Neonatal Jaundice part 1

Tip: Consider risk factors for jaundice

DISCUSSION: This part 1 of 2 parts discussing neonatal jaundice. First a refresher with definitions, a brief review of bilirubin metabolism and then the consequences and risk factors for developing hyperbilirubinaemia.

- What is neonatal jaundice?

It is jaundice, a yellow discoloration of the skin caused by the deposition of bilirubin in neonates, neonates refers to a newborn up to the age of 28 days. Raised levels of bilirubin causing jaundice are known as hyperbilirubinaemia.

- Why discuss neonatal jaundice?

It is a common problem worldwide. It can affect up to 60% of term neonates and 80% of preterm neonates. In the majority of cases, physiological jaundice resolves without intervention. This presents at 2-3 days of age and disappears at the end of the first week and resolved by 10 days. The bilirubin level does not exceed 200micromol/L and the baby remains well. 10 % of breast fed babies are still jaundiced at 1 month of age.

However, some neonates can develop high levels of jaundice that can result in irreversible and permanent brain damage if not treated this includes deafness, kernicterus (acute bilirubin encephalopathy) and athetoid cerebral palsy. The bilirubin level at which this damage occurs is lower in preterm neonates.

Thankfully the incidence of severe hyperbilirubinaemia is much less common, only around 2% of term infants have this, however a systematic review and meta-analysis carried out last year and published in the BMJ looking at the burden of severe neonatal jaundice found that The African region had the highest incidence of severe Neonatal Jaundice per 10,000 live births globally at 667/10000 live births. (SNJ defined by clinical outcome of acute bilirubin encephalopathy/kernicterus and/or exchange transfusion and/or jaundice-related death.

- What is hyperbilirubinaemia?

In neonates bilirubin levels are higher than adults as they have a higher concentration of RBC which also have a shorter life span. RBC breakdown creates unconjugated bilirubin

which circulates mostly bound to albumin. From here it travels to the liver where it is metabolised to produce conjugated bilirubin which is excreted in stool.

In babies there is a process called enterohepatic circulation of bilirubin whereby an enzyme in the intestines converts some of the conjugated bilirubin back into unconjugated so that it is reabsorbed and recycled in to the circulation. This makes the level on conjugated bilirubin very low and unconjugated bilirubin very high in comparison. So hyperbilirubinaemia is based on a type of a special graph which shows standardised bilirubin levels in correlation to the neonate's gestational age and hours of life. This also helps guide treatment. (Will attach graph to transcript)

Hyperbilirubinaemia can be considered severe when:

1. Onset of jaundice within the first 24 hours of life
2. The rate of increase in total bilirubin is too high
3. The level of conjugated bilirubin is too high compared with total
4. The neonate has signs and symptoms suggestive of serious illness

Reasons for treating hyperbilirubinaemia

When there is too much bilirubin in the infant's blood it overwhelms the albumin binding capacity and is not transported to the liver. The bilirubin builds up and is free to cross the blood-brain barrier. Bilirubin is neurotoxic and high levels result in bilirubin-induced neurologic dysfunction (BIND). This produces a spectrum of neurological findings which can be subtle or severe and is related to the level of bilirubin and length of elevation.

- Acute bilirubin encephalopathy is a Clinical syndrome: First baby may be mildly hypotonic (floppy), increased sleepiness or poor suck. As the hyperbiliubinaemia progresses the infant can develop a high pitched cry, be difficult to console, become febrile and hypertonic. In the advanced phase the baby develops apnoeas, inability to feed, persistent retrocollis (backward tilting of the neck due to muscle contraction) and opisthotonos (abnormal posturing of severe back arching), seizures, coma and even death. It can be reversible if treated appropriately but otherwise can lead to kernicterus

Kernicterus- occurs from the deposition of bilirubin in the brain causing staining and necrosis. Often affects basal ganglia and brain stem and most effects come from damage to these areas. This includes cerebral palsy, hearing loss, gaze abnormalities, dental dysplasia, developmental delay and learning difficulties.

- Risk factors for developing significant neonatal jaundice
    1. Low birth weight: premature or small for dates
    2. Breast-fed babies
    3. A previous sibling with neonatal jaundice requiring phototherapy

4. Visible jaundice in first 24 hours
5. Infants of diabetic mothers
6. Asian ethnicity
7. ABO or RH incompatibility (leading to haemolytic jaundice)
8. Birth trauma (cephalohaematoma/ instrumental delivery)
9. TORCH infections

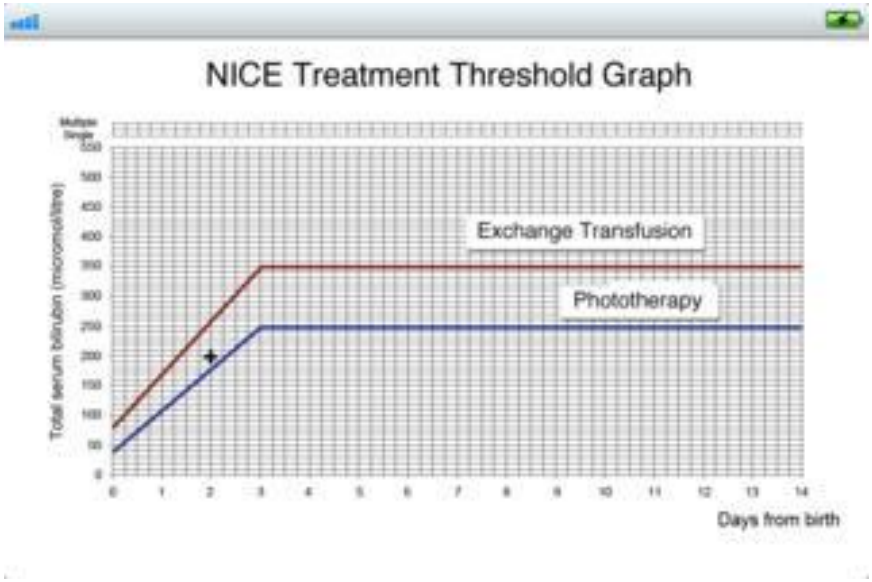
In summary: Jaundice (also known as hyperbilirubinaemia) is common and affects over half of term babies and nearly all premature neonates

Severe hyperbilirubinaemia must be treated in order to prevent bilirubin induced neurologic dysfunction, the long term consequence being Kernicterus

Plot bilirubin level to quantify degree of hyperbilirubinaemia, to determine whether treatment is needed.

In part 2 we will discuss the difference between physiological and pathological jaundice, discuss a clinical approach to neonatal jaundice and discuss treatment options.

Keep listening!



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