PODCAST No. Podcast 16 part 2

INTRODUCTIONS:

FEEDBACK:

TOPIC: neonatal jaundice part 2

TIP: Measure and plot levels according to baby' s gestation and age in hours/ days since birth

DISCUSSION:

Discussion: in our last podcast we covered the physiology and risk factors for neonatal jaundice. This time we are going to explore the differences between physiological and pathological jaundice And discuss differential diagnosis for unconjugated and conjugated jaundice.

We can break up the differential diagnosis into conjugated and unconjugated hyperbilirubinaemia. Conjugated hyperbilirubinaemia is always **pathological** however unconjugated can be divided into physiological and pathological.

Unconjugated hyperbilirubinaemia

Physiological

We briefly discussed physiological jaundice in the last podcast. It is due in part to the increased RBC breakdown and immature liver of the newborn. Presenting on day 2-3 beginning to disappear by a week and resolved by 10 days. The baby generally remains well. It can be exacerbated by bruising and is reaches a higher peak and may last longer in preterm infants.

Subtypes:

Breast feeding jaundice: common usually due to exaggeration of physiological hyperbilirubinaemia by dehydration caused by lack of milk production or intake. Common in mothers whose milk supply is small or late coming in or problems with initial feeding. Treatment is by rehydration- breast feeding support, supplementing with formula.

Breast MILK jaundice (less common) manifests as a persistent unconjugated jaundice starts around day 7 extending to 2-3 weeks after birth. Thought to be caused by substances in breast milk inhibit the conjugation activity of a key liver enzyme. Treatment is to continue breast feeding and treat jaundice if levels are high enough to require phototherapy.

Pathological unconjugated hyperbilirubinaemia

Caused by 2 major mechanisms:

1. Increased production of bilirubin
2. Decreased clearance of bilirubin

1. Increased production of bilirubin is caused by haemolysis so increased breakdown of rbcs. I.e. Haemolytic anaemia: eg haemolytic disease of the newborn (rhesus incompatibility); ABO incompatibility. Both caused by Mother and foetus do not have same blood type and mothers blood creates antibodies which destroy the newborns

rbcs. Simple blood type of both mother and child can elicit problem along with Direct Antiglutin test (DAT) or Coombs test.

Other cause of haemolytic disease are inherited red blood cell defects- membrane e.g. Spherocytosis, enzyme such as G6PD or pyruvate kinase deficiency or haemoglobin problem such as thalassaemia/sickle cell.

Investigating for these requires a fbc and peripheral blood smear for membrane or haemoglobin defects and specific metabolic testing will reveal enzyme deficiencies.

Sepsis is another cause (although it can also cause a conjugated picture). For unconjugated the baby will look Sick and likely to have fever and poor feeding.

ADMIT to hospital for septic screen. Fbc, urine and blood cultures with chest X-ray and LP to consider. Start the baby on empirical antibiotics. Think TORCH, GP B step, UTI, syphilis etc

Increased rbc breakdown due to haematoma. Likely cephalohaematoma by traumatic delivery. Worth noting the haematoma should not cross suture lines, if it does a subgaleal haematoma is present. This can be emergency due to significant blood loss. Treatment from cephalohaematoma is supportive as they usually self-resolve but may need phototherapy, fluid resuscitation and even blood transfusion.

2. Decreased clearance of bilirubin- usually caused by metabolic defects or endocrine disorders.

E.g. Crigler-Najjar syndrome (rare), Gilbert's syndrome (more common affecting between 4-16% of populations around the world). Both caused by genetic defects in the conjugation of bilirubin. Gilbert's is quite benign and largely asymptomatic.

Endocrine causes include maternal diabetes and congenital hypothyroidism. Usually picked up on metabolic screening programmes but can present before the results are available. Management appropriate resuscitation, treatment of jaundice with UV light, glucose monitoring for the former and potential thyroid hormone treatment for the latter.

Conjugated hyperbilirubinaemia

Less common but very important as always pathological. "Neonatal cholestasis" resulting from extrahepatic obstruction and intrahepatic disease.

Obstruction: Bilary atresia accounts for 25%. Must be differentiated from other causes of conjugated hyperbilirubinaemia as treatment is surgical and needs performing as soon as possible in order to increase success. Before 60 days of age. Babies usually healthy at birth but become progressively jaundiced within the first 2 months of life. Classically develop acolic (pale/ white) stools, dark urine and hepatosplenomegaly. They have raised aminotransferases and even higher GGT. They require USS and liver biopsy to rule out other causes in a centre able to take care of infants. Definitive diagnosis is with a cholangiogram usually performed in theatre so the Kasai (hepatoportoenterostomy) procedure can then be performed. 60-80% require a liver transplant.

Other causes of obstruction rare and management variable- biliary cysts, neonatal sclerosing cholangitis, tumours, gallstones, inspissated bile / plug syndrome.

Intrahepatic causes

- Congenital infections (as mentioned can cause both uncong and cong hyperbilirubinaemia) most common TORCH. E.coli UTI
- Metabolic/ genetic: extensive list but includes: cystic fibrosis, alpha-1- antitrypsin deficiency, galactosaemia, aminoacidutias. Early identification is necessary and prompt referral to a paediatric/ neonatal specialist centre for specific investigations.
- Toxins various toxins can cause conjugated hyperbilirubinaemia most common certainly in the U.K. is TPN induced (50% babies on tpn for >14 days will develop conj hyperbilirubinaemia. Or it can be drug induced.

In summary:

Majority of infants with unconjugated hyperbilirubinaemia do not have a serious underlying medical condition. Physiological jaundice resolves spontaneously.
Pathological unconjugated hyperbilirubinaemia has many causes but can be divided into 2 main categories: increased production and decreased clearance. Treatment is important to prevent kernicterus as unconjugated bilirubin can cross the blood- brain barrier.
Conjugated hyperbilirubinaemia is always pathological. Biliary atresia needs to be excluded as earlier surgical treatment results in better outcome.

In the part 3 we will talk about managing a jaundiced baby

GOODBYES: