

PedsCases Podcast Scripts

This is a text version of a podcast from PedsCases.com on “**Neonatal Jaundice.**” These podcasts are designed to give medical students an overview of key topics in pediatrics. The audio versions are accessible on iTunes or at www.pedcases.com/podcasts.

Neonatal Jaundice

Developed by Dr. Brittnee Kegler and Dr. Melanie Lewis for PedsCases.com.
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PART 1

Hello! My name is Brittnee Kegler and I am a first year family medicine resident at Queen’s University. This podcast was reviewed by Dr. Melanie Lewis, a general pediatrician and Professor at the University of Alberta and the Stollery Children’s Hospital in Edmonton.

This is the first of a three part series focusing on neonatal jaundice. This first part will focus on building a definition of neonatal jaundice, along with its consequences and risk factors. The second will focus on creating a broad differential diagnosis. Finally, the third part will help generate a clinical approach to jaundice through the use of history, physical exam, and investigations, then wrap up with treatment.

In this podcast, I will be focussing on the following learning objectives:

1. Discuss the definition, measurement, and epidemiology of hyperbilirubinemia
2. Review bilirubin metabolism in neonates
3. Examine the consequences, and risk factors, for developing hyperbilirubinemia

Let’s begin!

Case

You are a third year medical student on your pediatric rotation, currently at an academic day with your peers. Today, the preceptor wishes to tackle the topic of neonatal jaundice. She says, “Neonatal jaundice is a common occurrence, yet can be very confusing to medical students and residents alike. So let’s start by breaking it down. First, how do you define this condition? Do we know how many babies it actually affects? What is the physiology of this process?”

Let’s answer these questions together.

Definitions

So what exactly is neonatal jaundice? This can be defined based upon its name: Neonatal, referring to a newborn up to 28 days of life, and jaundice, a yellow discoloration of the skin caused by the deposition of bilirubin. Since jaundice is caused by increased bilirubin, the clinical word for this is hyperbilirubinemia.

Hyperbilirubinemia has a more specific definition, and is based upon a type of graph called the Bhuntani nomogram. These graphs show standardized bilirubin levels in correlation to the neonate's gestational age and hours of life. Therefore, hyperbilirubinemia is defined as a total serum bilirubin greater than the 95th percentile on the neonate's given nomogram. This also guides clinicians in treatment of the baby (discussed more in Part 3).

Hyperbilirubinemia can then be considered severe when:

1. The neonate has 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 when compared to the total bilirubin
4. The neonate has signs or symptoms suggestive of a serious illness

Severe hyperbilirubinemia can lead to critical hyperbilirubinemia which requires immediate action in order to prevent complications.

Epidemiology

So how many babies does this disease effect? Well, up to 60% of term newborns will have clinical jaundice in the first week of life and almost all preterm newborns will have jaundice at some point. It is very common. Thankfully, severe hyperbilirubinemia is not so common - only 2% of term newborns will have this. The incidence of acute bilirubin encephalopathy is even lower being estimated at 1 in 10 000 births and chronic bilirubin encephalopathy at 1 in 100 000.

Bilirubin Metabolism

Let's move on to a bit about how bilirubin is made and circulated through the body. This is important to note as changes within this system can cause bilirubin to build up causing jaundice.

Bilirubin is produced from the breakdown of hemoglobin from red blood cells. It is then bound to albumin and transported to the liver where it is taken up by hepatocytes. These hepatocytes catalyze the unconjugated bilirubin into conjugated bilirubin, making it more water-soluble. This allows the bilirubin to be secreted into the bile, which travels via the bile duct into the digestive tract. It is then excreted.

In babies, there is a special enzyme in the intestines that allows deconjugation of the conjugated bilirubin, turning it back into unconjugated. This allows it to be reabsorbed

and recycled into circulation. This whole process is known as “enterohepatic circulation of bilirubin”. This makes the level of conjugated bilirubin very low in babies, and subsequently, the unconjugated bilirubin very high in comparison.

Case

Let’s move back to the classroom. “Excellent!” your preceptor says. “This group knows quite a bit on the background of jaundice. But why is jaundice bad? What problems can it cause?”

Let’s go through the consequences of hyperbilirubinemia together.

Consequences of Hyperbilirubinemia

What can happen if the bilirubin starts to increase in a baby?

When there is too much bilirubin in the infant’s blood, it overwhelms the albumin binding capacity and is not able to be transported to the liver. This bilirubin builds up and is now free to cross the blood-brain barrier. Since bilirubin is a potential neurotoxin, high levels result in bilirubin-induced neurologic dysfunction (or BIND for short).

BIND is a spectrum of neurologic findings which can manifest from subtle to severe, correlated with how high the level of bilirubin is, and how long it stays elevated. In the acute phase, this is known as acute bilirubin encephalopathy, and chronically as chronic bilirubin encephalopathy, the latter of which corresponds with the pathological finding of kernicterus.

Acute bilirubin encephalopathy is a clinical syndrome that progresses through three phases. In the first phase, there are only subtle signs in the infant, such as a mild hypotonia, increased sleepiness, or poor suck. As hyperbilirubinemia progresses into the intermediate phase, the infant can develop a high-pitched cry and become difficult to console, febrile, and hypertonic (with retrocollis and opisthotonos upon stimulation). Into the advanced phase, the infant now develops apnea, inability to feed, persistent retrocollis and opisthotonos, seizures, coma, and even death. It should be noted the ABE can be reversible if treated appropriately but otherwise will lead to the permanent state of kernicterus.

Kernicterus is the chronic outcome of BIND and comes from deposition of bilirubin in the brain causing staining and necrosis. This corresponds to the clinical sequelae known as chronic bilirubin encephalopathy - most infants will develop this within the first year of life after ABE if not treated. Since kernicterus often affects the basal ganglia and brainstem, most effects come from damage to these areas. This includes cerebral palsy, hearing loss, gaze abnormalities, dental dysplasia, developmental delay and mental deficiency.

Now that we know why hyperbilirubinemia is so serious, let’s move back to our class.

Case

“Good!” your preceptor says. “As you can see, hyperbilirubinemia can be very serious, which is why we need to catch it early. Do you know the risk factors for jaundice? Let’s review that together.”

Risk Factors

So what are the risk factors that contribute to jaundice? These can be broken down into both maternal and neonatal factors.

For the mother, there can be an ABO or Rh incompatibility leading to a hemolytic jaundice. Other risk factors include drugs (common ones being diazepam and oxytocin), maternal illness (such as gestational diabetes), difficulty with breastfeeding, or maternal age older than 25. Also, mothers of Asian, European, or Native American ethnicity are also more at risk.

Neonatal risk factors are as follows: birth trauma (such as an instrumented delivery or development of a cephalohematoma), drugs (with common ones being Pediazole or chloramphenicol), excessive weight loss after birth, TORCH infections, infrequent feedings and dehydration, male gender, polycythemia, prematurity, previous sibling with hyperbilirubinemia, and delayed meconium.

In general, the most common risk factors are blood group incompatibility of the mother, prematurity of the infant or a previously jaundiced sibling.

Case

“Excellent work class.” your preceptor says. “Thank-you for taking the time to work through the pathophysiology of neonatal jaundice. Before we close, let’s go through a few take-away points from this podcast.”

Take-Away Points

1. Jaundice (also known as hyperbilirubinemia) is COMMON and affects over half of term babies and almost ALL premature neonates.
2. Severe hyperbilirubinemia must be treated in order to prevent bilirubin-induced neurologic dysfunction, the long term result which is kernicterus.
3. When suspecting neonatal jaundice, use a Bhuntani nomogram to quantify the degree of hyperbilirubinemia, to determine whether treatment is needed.

This concludes our podcast. Thanks for listening, and we hope to see you back for Part 2!

PART 2

Hello! My name is Brittnee Kegler and I am a first year family medicine resident at Queen's University. This podcast was reviewed by Dr. Melanie Lewis, a general pediatrician and Professor at the University of Alberta and the Stollery Children's Hospital in Edmonton.

This is the second of a three part series focusing on neonatal jaundice. The first part focused on building a definition of neonatal jaundice, along with its consequences and risk factors. This second will focus on creating a broad differential diagnosis. Finally, the third part will help generate a clinical approach to jaundice through the use of history, physical exam, and investigations, then wrap up with treatment.

In this podcast, I will be focussing on the following learning objectives:

1. Characterize the differences between physiologic and pathologic jaundice
2. Create a differential diagnosis for unconjugated hyperbilirubinemia
3. Create a differential diagnosis for conjugated hyperbilirubinemia

Let's begin!

Case

You are a fourth year medical student studying for your year end exams. While studying pediatrics with your friends, you decide to review neonatal jaundice, since it seems like a high yield topic. One of your colleagues suggests generating a differential diagnosis on the white board to go back over the main causes.

Let's work on this together!

Differential Diagnosis

The differential for hyperbilirubinemia can be broken down into two main categories: unconjugated hyperbilirubinemia and conjugated hyperbilirubinemia. Conjugated hyperbilirubinemia is ALWAYS pathologic, whereas unconjugated hyperbilirubinemia can be broken down into both pathologic and physiologic. Let's focus on the latter first.

Physiologic Jaundice

Physiologic jaundice usually manifests itself at the 2nd or 3rd day of life and typically resolves by about the 7th day. This is due to an increased bilirubin production from breakdown of red blood cells and the neonate's immature liver being unable to conjugate the bilirubin at a fast enough pace. Combined with an increased enterohepatic circulation, the baby's system is simply not able to keep up with the influx of bilirubin which manifests as jaundice. It should be noted that if the baby is premature, there will be a higher peak of bilirubin as well as a longer time for it to resolve. Physiologic jaundice can also be exacerbated by bruising.

Other physiologic subtypes of jaundice include breastFEEDING jaundice and breast MILK jaundice. While the former is common, the latter is quite rare.

In breastfeeding jaundice, the physiologic unconjugated hyperbilirubinemia is exaggerated by dehydration caused by a lack of milk production or intake. This is common in mothers whose milk supply is small or late coming in. It is common to peak at the same time as normal physiologic jaundice.

In breast milk jaundice, this manifests as a persistent unconjugated hyperbilirubinemia that starts at around the 7th day of life and extends to 2-3 weeks after birth. This is caused when natural substances found in the breastmilk begin inhibiting the conjugation activity of a key enzyme in the liver.

The treatment of both of these subtypes is different. Since breastfeeding jaundice is caused by dehydration of the infant, it's treatment is via rehydration. Depending on the severity, this can range from a referral to a lactation consultant, to supplementing with formula depending on milk supply. The treatment of breast milk jaundice is to continue breastfeeding. Overall, breastfeeding support is very important to provide to these new mothers as well.

Pathologic Jaundice

Now let's get into the different types of pathologic jaundice. We'll begin with causes of pathologic unconjugated hyperbilirubinemia. This can be caused by two major mechanisms: increased production or decreased clearance. Let's start with the first one.

Increased Production

Overall, increased production of bilirubin can be traced back to hemolysis of red blood cells. As more erythrocytes become broken down, the more the unconjugated bilirubin will rise.

The most common cause of this hemolysis is by ABO or Rh incompatibility. This is a condition that arises when mother and fetus do not have the same blood type and the mother creates antibodies which destroy the red blood cells of the newborn. A simple blood type of both mother and baby can elicit the problem, along with a direct antiglobulin test, also known as a Coomb's test. All infants with a positive DAT and at increased risk of severe disease should receive IVIG. If further treatment is required, a transfusion of the appropriate cross-matched blood product to the babe is recommended. Guidelines state that ALL mothers be tested for ABO and Rh factor, and screened for red cell antibodies during pregnancy so that this can be mitigated.

Another cause of hemolysis is inherited red blood cell defects. There are three main categories these defects can occur in: membrane (such as spherocytosis or elliptocytosis), enzyme (such as G6PD or pyruvate kinase deficiency or congenital erythropoietic porphyria), or hemoglobin (such as thalassemia). These causes are quite rare but can be hereditary, and selected at-risk infants of certain ethnicities may be screened for this. For investigations, a CBCd and peripheral blood smear will reveal any

membrane or hemoglobin defects, whereas specific metabolic testing will reveal enzyme deficiencies. The treatment is beyond the scope of this podcast.

Sepsis is another cause of jaundice, although this can cause a conjugated as well as unconjugated picture. For unconjugated, sepsis is known to cause hemolysis of red blood cells leading to hyperbilirubinemia. This baby will look SICK and likely have a fever! On investigations, a CBCd, urine and blood cultures MUST be performed, with the option of a chest x-ray and an LP as well. Start the babe on empiric antibiotics, replace fluids if necessary, and admit to hospital.

Lastly, another cause of increased RBC breakdown can be found when this blood is sequestered within a closed space. In a baby, this is likely from a cephalohematoma caused by a traumatic delivery. This diagnosis can be made clinically, or with imaging of the head via CT scan. Note that the hematoma should NOT cross suture lines, and if it does, a subgaleal hematoma is present, and this is can be an EMERGENCY due to significant blood loss. For a cephalohematoma, this is likely to resolve on its own over a couple of months. Until then, supportive care/observation is necessary depending on its severity and may require UV light therapy, fluid resuscitation, and even a blood transfusion.

Decreased Clearance

Now we move on to the other cause of unconjugated hyperbilirubinemia: decreased clearance. These are usually caused by metabolic defects or endocrine disorders.

Of the disorders that cause decreased clearance, we have Creeg-ler-Na-jhar syndrome and Gilbert syndrome. Both of these are caused by genetic defects involving catalysts in the conjugation of bilirubin. Although Crigler-Niger is quite rare, Gilbert's syndrome affects between 4 and 16% of populations around the world. However, the disease is overall quite benign and largely asymptomatic. Patients that do have manifestations usually present in adolescence with mild intermittent jaundice. Neonates may have breast milk jaundice during the second week of birth if anything.

The remaining causes of decreased clearance include maternal diabetes and congenital hypothyroidism. These are usually picked up on metabolic screening programs but can present before results come in. Management involves appropriate resuscitation, treatment of the jaundice via UV light, and then glucose monitoring for the former and potential thyroid hormone treatment in the latter.

Conjugated Hyperbilirubinemia

Now, we move into conjugated hyperbilirubinemia. This is less common as compared to unconjugated causes of hyperbilirubinemia but no less important. It is often referred to as "neonatal cholestasis" and results from extrahepatic obstruction and intrahepatic disease.

Obstruction

Biliary atresia **MUST** be differentiated from other causes of conjugated hyperbilirubinemia as treatment is **SURGICAL** and **MUST** be performed as soon as possible in order to increase success. Babies with this condition will usually be healthy at birth, but will become progressively jaundiced within their first 2 months of life. They may also develop acolic (or pale) stools, dark urine, and hepatosplenomegaly. Lab tests will show elevations in aminotransferases and even higher GGT. U/S and liver biopsy are performed to rule out other causes of increased conjugated bilirubin. Definitive diagnosis is made via cholangiogram and is usually done in the OR. The Kasai procedure (or hepatoportoenterostomy) should then be performed. It should be noted that timing is of the essence with this disease as success of surgery diminishes as the babe becomes older – it is preferable before 60 days of age. Eventually, 60-80% of patients will also require a liver transplant. This condition affects one-quarter of all babies with neonatal cholestasis.

Other causes of obstruction include biliary cysts, neonatal sclerosing cholangitis, tumors, gallstones, or inspissated bile/plug syndrome. It should be noted that these causes are much more rare and their management is variable. Ultrasound is a good first step, along with liver enzymes and liver function tests. These conditions will likely need consultation to a pediatric surgeon.

Now, let's move on to the causes of intrahepatic diseases.

Infectious

Congenital infections can also lead to conjugated hyperbilirubinemia. The most common include the TORCH infections – toxoplasmosis, others, rubella, cytomegalovirus, and herpes. Sepsis can also present as a conjugated jaundice, for example, an E. coli UTI. As mentioned in the previous section, appropriate septic work-up must take place, and treatment with fluids and empiric antibiotics.

Metabolic/Genetic

Various metabolic and genetic disorders can also lead to a conjugated picture. The list is quite extensive, but the most common of these is cystic fibrosis, alpha-1 antitrypsin deficiency, galactosemia, and tyrosinemia. Early identification of any metabolic or genetic disorder is necessary, as specific treatment is usually available. The newborn screen will usually pick up the more common of these.

Toxic

Various toxins to the babe can also cause neonatal cholestasis. This can be either drug induced or TPN induced, with TPN being much more common. This is important to note as about 50% of infants treated with TPN for greater than 14 days will develop a conjugated hyperbilirubinemia. This can be treated by weaning the babe from TPN and increasing enteral feeds.

Case

You and your colleagues lean back from the white board where you've just created a comprehensive list of all you've been talking about. You're content with your work, and decide to finish up your day with a celebratory coffee before heading home.

Now, before we leave, let's reiterate a few take-away points.

Take-Away Points

1. The majority of term infants with hyperbilirubinemia do NOT have an underlying serious medical condition. Physiologic jaundice resolves spontaneously as the babe's hepatic system matures.
2. Pathologic unconjugated hyperbilirubinemia has a multitude of causes but can simply be divided into two main categories of increased production and decreased clearance.
3. Conjugated hyperbilirubinemia is ALWAYS pathologic. Biliary atresia must be differentiated from the other causes as the treatment is surgical rather than medical, and has a better outcome when performed earlier.

This concludes our podcast. Thanks for listening, and we hope to see you back for Part 3!

PART 3

Hello! My name is Brittnee Kegler and I am a first year family medicine resident at Queen's University. This podcast was reviewed by Dr. Melanie Lewis, a general pediatrician and Professor at the University of Alberta and the Stollery Children's Hospital in Edmonton.

This is the third of a three part series focusing on neonatal jaundice. The first part focused on building a definition of neonatal jaundice, along with its consequences and risk factors. The second part focused on creating a broad differential diagnosis. Finally, this third part will help generate a clinical approach to jaundice through the use of history, physical exam, and investigations, then wrap up with treatment.

In this podcast, I will be focussing on the following learning objectives:

1. Develop a clinical approach to neonatal jaundice through history, physical exam and investigations
2. Discuss treatment options for hyperbilirubinemia

Let's begin!

Case

You are a first year family medicine resident, currently working in a community office. Your preceptor approaches you and tells you that you're in luck! There is a new patient to be seen today for a newborn check-up. Your preceptor tells you that you will be seeing a 4 day old newborn male named Oliver.

You enter the room, introduce yourself to Oliver's parents, and ask if they have any concerns for you today. The mother says she's worried since she's noticed Oliver's skin is a bit more yellow ever since she's brought him home.

First, let's take a proper history around jaundice. There are several questions that should be asked at this time.

History

With any neonatal presentation, we first must confirm the baby's age and then can ask about the history of presenting illness. When did the caregiver start to notice the jaundice? Was it immediately after birth, after coming home from the hospital, or a week later?

Ask about other signs and symptoms the baby may have. Has the baby been increasingly sleepy? Has their cry changed? Has the babe had a fever, or been coughing? Have they seemed "floppy" or more jittery?

Ask a quick nutritional history: is the baby breastfed or bottlefed? If breastfed, does the mom feel she have good milk supply? Ask if she can hear the baby sucking AND swallowing. Does she feel the baby latches well onto the breast? Does the baby seem content after a feed? If the baby is bottle-fed, confirm what formula the caregiver uses and how they mix the formula. Ask how much the baby is feeding, and whether they spit up after meals. A normal newborn will feed 7 or more times in a 24 hour period, or roughly every 2-3 hours.

Then ask about diapers. How often is the babe urinating and stooling? Is the urine excessively smelly? Quantify the texture of the stools – are they hard or soft? Also ask about the color of the poop – answers could vary from yellow, green, or brown to more concerning extremes such as dark black or sandy white. Is there any blood in the diaper, whether from the urine or the stool? In general, a normal newborn should have 6 or more wet diapers per day after the first week of life, and approximately 3-4 stools per day.

Once we've finished our nutritional history, move onto the birth history. Make sure to quantify the gestational age of the neonate when it was born – was it preterm? Ask about the pregnancy – did the mother have routine prenatal care with appropriate blood tests and urine tests throughout? Were there any complications during the pregnancy?

Also, does the mom have any medical conditions that we should be aware of? Has the mother been pregnant before?

During the birthing process itself, how long was the labor? How long were the membranes ruptured for? Does the mother know her blood type and Group B Strep status? Was the birth vaginal or cesarian? Was there any forceps or vacuum used during the delivery? How much did the baby weigh at birth? Were there any complications during the birth? Did the baby cry right away? Did they spend any time in the NICU? Where was the baby born?

Moving into family history, make sure to ask if there are any genetic anomalies that run in the family. Ask specifically about any blood anomalies or problems with metabolism. Also ask if any other neonates had jaundice, and if so, if they had to spend time under a UV light.

Lastly, quickly touch on any medications the baby is taking, as well as any allergies the family has noticed. Since we are focusing on neonates up to 28 days of life, immunizations would not be given.

Case

When you ask these questions to Oliver's parents, you receive the following information. The mother first started noticing the jaundice a day or two ago, and does not think this was present at birth. Oliver has been breastfeeding about every 2 hours although mom is worried that she doesn't have good milk supply as he seems to not be sucking for very long. He pees after every feed and has 2-3 yellow seedy poops every day. In terms of the birth, Oliver was born 2 weeks early via a normal vaginal birth with no complications. He cried right away, and weighed 5lbs 10oz. The parents don't think there are any genetic problems in the family, and the mother states that she was "a bit more yellow" as a baby and had a "special blanket" for a couple of hours. The mother confirms that she gives Oliver D-drops daily and hasn't noticed any allergies.

Physical

Now that a full history has been elicited, we can move onto the physical exam.

First, we must inspect the baby to see where the jaundice is located. Although the baby may not have much jaundice in its skin, make sure to also look at the whites of the baby's eyes, the mucous membranes, the palmar creases, and the frenulum. Also check the skin for any signs of trauma or bruising such as a cephalohematoma.

After inspecting the skin, a full screening exam should be performed, with a special emphasis on the abdominal exam and the neurologic exam. Check the baby's tone and reflexes and how well it interacts with its environment. Does it seem excessively sleepy? Does it cry during the exam but calm when the caregiver cuddles them? On

abdominal exam, look for any masses and any signs of hepatosplenomegaly. Lastly, check for any other gross malformations the baby may have.

Case

Let's go back to our case.

On examination of Oliver, it is clear the skin is jaundiced but there are no other bruises or marks on the skin. He does have a bit of flaking on his scalp. Breath sounds and heart sounds are normal, and both fontanelles feel soft. His belly is soft and there are no obvious masses. An examination of the genitalia reveals a normal non-circumcised penis and palpable testicles. For the majority of the exam, Oliver wiggles around, and keeps his hips and knees flexed. All reflexes are normal, and he cries after you perform the Moro reflex on him. Once the father picks him and starts rocking him, he becomes calmer.

After the physical exams, you go to report back to your preceptor. She listens intently to your story.

"I am not too worried about Oliver," she says. "It seems like Oliver's story is in keeping with physiologic jaundice, specifically breastfeeding jaundice. Are there any investigations we should order?"

Let's chat about this next.

Investigations

The first and most important test, is to QUANTIFY the level of bilirubin in order to determine if it needs to be treated or not. Bilirubin can be measured two ways: through a blood sample or transcutaneously (ie through the skin). The transcutaneous bilirubin is commonly used as a screening test for infants when they are first born. It is not as reliable as a blood test, as changes in the skin color and thickness can affect bilirubin levels along with the initiation of phototherapy. Current guidelines state that either should be measured in all infants within the first 72 hours of life, sometimes with the metabolic screening test (heel poke). The Bhutani nomogram can then be used to compare the baby's bilirubin levels to the thresholds for treatment, and determine the risk of progression to severe hyperbilirubinemia. A copy should be given to the parents, and appropriate follow-up put in place (especially if the baby is discharged before 24 hours of life).

In infants that have severe or prolonged hyperbilirubinemia, they should be further investigated with a conjugated bilirubin in order to determine whether this is a conjugated or unconjugated hyperbilirubinemia.

If the hyperbilirubinemia is unconjugated, further work-up may be necessary. If hemolysis is suspected, a CBC/dif, blood group of mother and infant, a peripheral blood

smear, and a Coombs test should be performed. If the baby is unwell or has a fever, a septic work-up should take place with a CBCdif, blood & urine cultures, and possibly a lumbar puncture. If neonatal metabolic screening has not been undertaken, a TSH and G6PD screen should also occur.

If the hyperbilirubinemia is determined to be conjugated, the work-up should be focused on determining whether the cause is hepatic or extra-hepatic in nature. Liver enzymes (in the form of AST & ALT), coagulation studies (done with a PT & PTT) and an albumin should all be drawn. Suspected infection should elicit a CBCdif, TORCH screen and septic work-up. An abdominal U/S and/or HIDA scan can be used to rule out biliary atresia or cysts, and lastly, a metabolic screen (if not already performed) should be done to rule out hypothyroidism and cystic fibrosis.

Let's return to our case.

Case

“What tests would be appropriate for Oliver? In this case, the only test we should do is a transcutaneous bilirubin. This will enable us to quantify the jaundice and see if treatment is necessary or not. Before we go in and tell the parents what is going on, let's quickly go over treatment to wrap this up.”

Treatment

Treatment of neonatal jaundice is focused on two main points: symptomatic control and treatment of the underlying cause. This second point has already been covered when we discussed the differential diagnosis. Now, let's focus on how to treat the jaundice itself.

Hyperbilirubinemia that is unconjugated has the same symptomatic treatment regardless of underlying cause. This involves phototherapy via the use of a special wavelength of light. When this light hits the bilirubin molecules in the skin, it is able to break it down and convert it into a form that is water-soluble and can therefore be excreted. It is largely inexpensive and non-invasive, and is available in two forms: a blanket, or a direct light source. These babies are then placed inside a special cot with this light source, along with eye protection so that it does not damage the retina. Treatment can occur for a variable amount of time and bili concentration should be rechecked w/in 2-6 hours to confirm response. Intensive phototherapy is recommended for infants with, or at risk for, severe hyperbilirubinemia, and is accomplished with higher intensities of light and increased exposed area of the infant. Breastfeeding should be continued during phototherapy, and fluids given if the babe is at high risk of progressing.

If the babe's bilirubin levels are too high for phototherapy, or if phototherapy fails to control them, an exchange transfusion can be performed. This is very rare, and usually only done in severe cases of jaundice in order to prevent those serious consequences mentioned before. It involves removing the infant's blood and replacing with a donor's

blood in order to prevent the buildup of toxins (in this case, bilirubin). A baby with signs of acute bilirubin encephalopathy should have an exchange transfusion performed immediately.

Let's head back to the clinic.

Case

“Good work!” your preceptor states. “For Oliver, I think it would be our best bet to get a lactation consult to make sure the mother’s technique is appropriate. Let’s also get a transcutaneous bilirubin to make sure it’s not too high. If it is, we can call ahead to the hospital for phototherapy. But I have a feeling that little Oliver will be just fine.”

You enter the room with your preceptor and catch the parents up to date on everything that was discussed. The parents are relieved that Oliver will be okay and agree with the current plan. They thank the both of you as they leave.

Excellent work today! Before we close, let’s go through a few take-away points from this podcast.

Take-Away Points

1. When taking a history around jaundice, always ask about birth history, nutritional history and family history to help rule out underlying pathologic causes.
2. Any neurological signs or symptoms found on physical exam are a huge red flag and warrants immediate investigation.
3. Current guidelines encourage screening for hyperbilirubinemia within the first 72 hours of life with either a transcutaneous bilirubin or a blood draw. Other investigations for jaundice are only warranted when a pathologic cause is suspected or the hyperbilirubinemia is considered prolonged or severe.
4. Hyperbilirubinemia is treated by addressing the underlying cause. In unconjugated hyperbilirubinemia, symptomatic treatment involves phototherapy.
5. Exchange transfusion should be started immediately if any infant presents with signs of acute bilirubin encephalopathy.

This concludes our podcast. Thanks for listening!

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