Neonatal Hyperbilirubinemia & Phototherapy

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Objectives
- Describe the mechanism of physiologic jaundice
- Identify the mechanisms predisposing the infant to physiological jaundice
- Discuss the use of phototherapy for hyperbilirubinemia
- Apply assessment criteria for the infant who is jaundiced
- Identify risk factors for polycythemia
- Identify the treatment and nursing interventions for polycythemia

What is Jaundice?
- Yellowish discoloration of skin and sclera of newborns due to bilirubin
- Also known as jaundice of the newborn...
Definitions:
- Hyperbilirubinemia- an elevated total serum bilirubin (TSB) level
  - Abnormal values vary by age, days of life, current illness and conditions
- Jaundice- the yellowish coloration of the skin and sclera caused by presence of bilirubin in elevated concentrations

Definitions:
- Conjugated- Direct Serum Hyperbilirubinemia
- Unconjugated- Indirect Hyperbilirubinemia
- Kernicterus- Irreversible, chronic sequelae of bilirubin toxicity

Bilirubin:
- By-product of the breakdown of red blood cells
- Presents as a higher than normal level of bilirubin in the blood for the gestational age and chronicle age in hours
- A breakdown of 1 gram of hemoglobin = 35mg of bilirubin
Pathophysiology

- Bilirubin binds to albumin to be transported to the liver
- Conjugation takes place in the liver
- Fat soluble unconjugated (indirect) bilirubin is converted to water soluble conjugated (direct) bilirubin
- This is necessary in order for the bilirubin to be metabolizes and removed by the body

Pathophysiology

- Unconjugated (indirect) bilirubin rides on albumin making its way to the liver where it becomes conjugated by two other enzymes and some glucuronic acid made from glucose. So, the interaction of unconjugated (indirect) bilirubin with these liver enzymes & glucuronic acid is what turns the unconjugated bilirubin into conjugated (direct) bilirubin

Bilirubin Breakdown
Pathophysiology

- In order for bilirubin to clear from the body it must be:
  Conjugated in liver, excreted in bile, and eliminated via urine and stool
- Most common reason that neonates need medical attention:
  "Physiologic jaundice" - a normal phenomenon during transition, but different from "Pathological jaundice"
  Becomes concerning when levels continue to rise
- Unconjugated bilirubin is NEUROTOXIC

Types of Jaundice

So, what's the difference between direct (conjugated) and indirect (unconjugated) bilirubin???
This form of bilirubin does not dissolve in water (it is insoluble). Indirect bilirubin travels through the bloodstream to the liver, where it is changed into a soluble form (conjugated or direct).

Unconjugated (Indirect Bilirubin)

- Elevated levels of bilirubin are caused by any of the following: imbalance in production, transport, uptake, conjugation, excretion, and reabsorption.
- This is most concerning due to risk for encephalopathy/kernicterus if not treated rapidly.

Conjugated (Direct Bilirubin)

- Considered elevated when:
  - Level > 2.0 mg/dL (severe > 5.0 mg/dL)
  - Level > 15% of total serum bilirubin
- Risk factors:
  - Low gestational age
  - Early and/or prolonged exposure to TPN
  - Lack of enteral feeding
  - Sepsis

Direct bilirubin dissolves in water (it is soluble) and is made by the liver from indirect bilirubin.
**Peak Jaundice Levels**
Affects nearly all newborns
Peak: varies by age and hours old

**Term Infant**
- DOL 3-4: Rising Bilirubin Levels
- DOL 5: Peak Bilirubin Levels
- After Day 5 bilirubin levels start decreasing

**Pre-term Infant**
- DOL 6-7 Bilirubin Levels Peak

**Risk factors for Severe Jaundice**
Major Risks:
- Jaundice in the first 24-36 hours of life (early onset)
- Rh/ABO incompatibility
- Exclusive breastfeeding
- Poor feeding/ineffective breastfeeding
- Bruising/cephalohematoma
- G6PD Deficiency
- Southeast Asian heritage
- Gestational age 35-36 weeks
- Prematurity
- Asian or Native American
- Bruising during birth process
- High altitude
- Infant of diabetic mother
- Induced labor

**Types of Jaundice**
Pathological Jaundice

- Definition:
  - Increase of bilirubin >0.2 mg/dL/hr
  - Jaundice in the first 24-36 hours of age
  - Persists > 7 days

- Underlying Causes:
  - Increased bilirubin production
  - Decreased bilirubin excretion
  - Combo of both

Physiological Jaundice

- Most infants develop visible jaundice due to elevation of unconjugated bilirubin concentration during their first week.
- This pattern of Hyperbilirubinemia has been classified into two functionally distinct periods.

  - **Phase one**
    - Term infants - jaundice lasts for about 10 days with a rapid rise of serum bilirubin.
    - Preterm infants - jaundice lasts for about two weeks, with a rapid rise of serum bilirubin.

  - **Phase two**
    - Bilirubin levels decline for two weeks, eventually mimicking adult values.
    - Preterm infants - phase two can last more than one month.
    - Exclusively breastfed infants - phase two can last more than one month.

Physiological Jaundice Causes

- Low enzyme activity
  - Glucuronosyltransferase which normally converts unconjugated bilirubin to conjugated bilirubin that can be excreted into the gastrointestinal tract.
  - After birth, this enzyme is actively down-regulated, since bilirubin needs to remain unconjugated in order to cross the placenta to avoid being accumulated in the fetus.
  - After birth, it takes some time for this enzyme to gain function.

- Shorter life span of fetal red blood cells

- Relatively low conversion of bilirubin to urobilinogen by the intestinal flora
  - Results in relatively high absorption of bilirubin back into the circulation.
Physiologic or Pathologic
Jaundice < 24 hrs is always pathologic!

Types of Jaundice

Breastfeeding Jaundice
- Breastfeeding jaundice or "lack of breastfeeding jaundice," is caused by insufficient breast milk intake, resulting in inadequate quantities of bowel movements to remove bilirubin from the body.
- Lack of milk intake which leads to lack of output
- Mechanical Problem with feeding

KEY POINTS
- Non-organic cause
- Early onset
- Related to ineffective breastfeeding
- Dehydration and poor nutrition
- More common among first time moms and preterm infants
Breast Milk Jaundice

- Where as ‘breast milk’ jaundice is a biochemical occurrence and the higher bilirubin possibly acts as an antioxidant. Breast milk jaundice occurs later in the newborn period, with the bilirubin level usually peaking in the sixth to 14th days of life. This late-onset jaundice may develop in up to one third of healthy breastfed infants.

KEY POINTS

- Non-hemolytic jaundice
- Late onset
- Begins after day 3-5 of life
- Increased bilirubin levels peak between 5-10mg/dL at about 2 weeks of life
- May persist for months
- Develops in about 2-4% of breastfed newborns
- Runs in families

Diagnosis

Transcutaneous (Tcb) Serum (SBR)

- Bilimeter being used by the Pediatric team on FMCC (MD's & NP)
- Cannot be used during or after phototherapy
- At risk infants should have a SBR drawn
- RN to draw SBR on all newborns at 24 hours of age with newborn screen

Laboratory Diagnosis

- Blood level varies by age in hours, size of infant and risk factors
- Blood levels usually does not exceed 17-18 mg/dL
- High levels of Bilirubin the greater risk for Kernicteris
Diagnosis: Bhutani Nomogram

- Scale used to measure risk
- Pathologic: TSB exceeds 95th percentile according to Bhutani nomogram

Clinical Signs and Symptoms

Jaundice
- The whites of the eyes become yellow or skin has a yellow or orange color
- Very sleepy or very floppy (like a rag doll)
- Low temperature
- Irritability/ fussiness
- Feeding problems (especially having trouble getting baby to awaken to feed)

Clinical Symptoms: Kernicterus
- Irritability, jitteriness, increased high-pitched crying
- Lethargy and poor feeding
- Back arching
- Apnea
- Seizures
- Long-term: Cerebral Palsy, upward gaze palsy, hearing loss, dental dysplasia
Jaundice to Kernicterus

Deposits in skin and nervous membranes → JAUNDICE

Unconjugated bilirubin deposits in the brain → ACUTE BILIRUBIN ENCEPHALOPATHY

Permanent neuronal damage → KERNICTERUS

Treatment

Goals

- Prevent severe hyperbilirubinemia that leads to kernicterus
- Tx of underlying conditions
- Maintenance of hydration and nutrition

Interventions

- Phototherapy
- Hydration
- Exchange transfusion
- IVIG

Phototherapy Treatment

- Mechanism: converts bilirubin to water soluble form that is easily excreted
- Forms:
  - Fluorescent lighting
  - Fiberoptic blankets
  - Bilibed
- Goal is to decrease TSB by 4-5 mg/dL or < 15 mg/dL total
- Breastfed infants are slower to recover
Phototherapy

Effectiveness: Important factors - Spectrum, irradiance, distance, surface area.

Phototherapy Treatment

- Primary treatment is Phototherapy
- Helps to breakdown bilirubin so that it can clear through the stool and urine
- Does not treat the underlying cause

Treatment: Prevent Kernicterus:

- Observe baby for jaundice
- Evaluate each baby’s risk factors for jaundice
- Thorough Handoff report & provider communication
- Educate families
- Bilicheck before discharge:
- At UCSD, all babies receive a serum bilirubin draw at the time of newborn screen. Results are plotted on the Hour Specific Bilirubin Nomogram
Treatment: Prevent Kernicterus:
• Damage from Kernicterus is Irreversible
• Prevention is Key!!!!!!

Nursing Care:
• Know the device you are working with distance between device and baby
• How do you check irradiance and what is the output range for the device hours of use
• Eye shields if necessary
• Monitor for hydration status/knowing what stools should look like
• Monitor time out for feeding and holding Thermoregulation
• What to do with a fussy baby in phototherapy

Nursing Assessment
• Assessment should be done q-shift, more often for high risk infants
• Evaluate risk factors
• Check Bilirubin levels on all infants before discharge
• Plot on Bhutani Curve nomogram and assess risk zone and any changes
APA Practice Guideline (2004): Hyperbilirubinemia

- Promote and support successful breastfeeding
- Perform a systematic assessment before discharge for the risk of severe hyperbilirubinemia
- Provide early and focused follow-up based on assessment
- When indicated, treat newborns with phototherapy or exchange transfusion to prevent the development of severe hyperbilirubinemia and, possibly, bibirubin encephalopathy (kernicterus).

(Pediatrics 2004; 114:297-316)

Discharge Precautions

- Early discharges (before 48hrs of life)
- Make sure adequate follow-up will be possible
- All babies should be seen 24-48 hours post discharge by HCP for follow-up and assessment
- Lactation support for breastfeeding families is important
- Educate families on what to look for, how to get help and what risk factors their child has

- Education, Education, Education!

Definitions

- **Polycythemia** is increased total RBC mass
  - Central venous hematocrit > 65%
  - Above 65% blood viscosity rises exponentially

- **Polycythemic hyperviscosity** is increased viscosity of the blood resulting from increased numbers of RBCs
  - Not all polycythemic infants have symptoms of hyperviscosity
• Altitude: increased RBC mass
• Neonatal age
  - Physiologic increase in hematocrit due to fluid shifts away from intravascular compartment with maximum at 2-4 hours of age
• Obstetric factors: delayed cord clamping or “stripping” of the umbilical cord
• High-risk delivery, especially if precipitous

Conditions that alter incidence

• Enhanced fetal erythropoiesis usually related to fetal hypoxia
  - Placental insufficiency
    • Maternal hypertension, abruption, post-dates, IUGR, maternal smoking
  - Endocrine disorders: due to increased oxygen consumption
    • IDM (>40% incidence), congenital thyrotoxicosis, CAH, Beckwith-Wiedemann syndrome (hyperinsulinism)

Perinatal processes

• Hypertransfusion
  - Delayed cord clamping
    • Placental vessels contain 1/3 of the fetal blood volume, half of which will be returned within 1 minute
  - Gravity: positioning below the placenta will increase placental transfusion
  - Meds: oxytocin can increase contractions and thus transfusion
    • Decreased in c-section b/c no contractions
  - Twin-twin transfusion
  - Maternal-fetal transfusion
  - Intrapartum asphyxia
    • Enhances net umbilical flow toward the infant, while acidosis increases capillary leak leading to reduced plasma volume
• Central venous hematocrit > 65%
  • ALWAYS draw a central venous sample if the capillary hematocrit is > 65%
    • Warmed capillary hematocrit > 65% only suggestive of polycythemia

**Diagnosis**

• Asymptomatic infants
  • Expectant observation unless central venous hematocrit >75% (consider partial exchange transfusion)
  • Can do a trial of rehydration over 6-8 hr if dehydrated
    • Usually at > 48 hours of age and weight loss > 8-10%
    • Give 130-150 ml/kg/d
    • Check central hematocrit q6 hours
      • Normal peak is at 2-4 hours of age for acute polycythemia

**Management**

• Symptomatic infants with central hct > 65%
  • Partial exchange transfusion is advisable but debatable
  • For exchange can use normal saline, Plasmanate, 5% albumin, or FFP
  • Volume exchanged =
    • (Weight (kg) x blood volume) x (hct - desired hct) / hct
    • Blood volume is 80 ml/kg
  • Exchange can be done via UVC that is not in the liver, low UAC, or PIV

**Management**
Summary

- Consider the risk factors, particularly prematurity and hemolysis.
- **Follow up is key!**
- Consider how well baby is feeding, parents’ ability to return to the doctors office.
- The higher the number of risk factors, the lower the level at which to intervene.
- Sometimes, you will be surprised. We can’t always prevent Hyperbilirubinemia, but we should always prevent kernicterus.

Any Questions?

References:

- Fanaroff and Martin chapters on hyperbilirubinemia

All images found on google images.