Outpatient Management of Common Respiratory Problems in a NICU graduate

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Objectives

- To review common respiratory problems of a NICU graduate
- To discuss outpatient interventions of respiratory disorders in infants with chronic lung disease of infancy
- To present outpatient diagnostic studies that will assist in the management of an infant with chronic lung disease
Definition

• Bronchopulmonary dysplasia (BPD)- pathologic process; infants still requiring oxygen (O2) at 28\textsuperscript{th} day of life.

• Chronic lung disease of infancy (CLDi) – continued requirement of O2 at 36 weeks post menstrual age (PCA)

• CLDi- better predictor of clinical respiratory difficulty in the first year of life.
Definition

NHLBI: infants <32 weeks

• Mild BPD – treatment with O2 of >21% for at least 28 days, but in room air at 36 weeks PCA
• Moderate - O2 of >21% for at least 28 days; O2 <30% at 36 weeks PCA
• Severe- treatment with O2 >21% for at least 28 days and require >30% O2 or positive pressure ventilation at 36 weeks.
Definition

• Common to both BPD and CLDi: lung immaturity, injury from O2, mechanical ventilation, lung inflammation
Statistics

• Approximately 1 out of every 9 live births occurs prematurely.
• 90% of preterm infants survive.
• BPD/CLDi remains the most common complication of extreme prematurity
• Increased morbidity in the first 2 years of life.
Common respiratory disorders in the outpatient

**Signs and Symptoms**
- Wheezing
- Apnea
- Increase work of breathing
- Desaturation

**Differential Diagnoses**
- Airway reactivity; Bronchiolitis; Aspiration syndromes; Airway obstruction; Pulmonary edema
- Apnea of prematurity; ALTE; Aspiration; Airway obstruction
- Subglottic damage; Pneumonia; Aspiration syndromes; Pulmonary edema
- Pulmonary hypertension; Airway obstruction; Pulmonary edema
Airway Reactivity

Episodic
Dramatic clinical response to bronchodilator.
Evidence of bronchodilator responsiveness seen in lung function testing:
- Infant pulmonary function testing if available – can be done in up to 2 years of age.
- Impulse oscillometry – for toddlers
- Spirometry for >= 5 years old
Airway Reactivity

Intervention:

1. Short acting beta agonist (SABA) PRN
2. +/- Inhaled corticosteroid

** Metered dose inhaler with mask spacer is as good or even better as nebulized form when correctly used. It is convenient and requires a lower dose.
** 1.25 to 2.5 mg nebulized Albuterol over 10-15 minutes per dose versus 216 mcg to 432 mcg (2-4 puffs of HFA form) per dose over a span of 30 seconds
BRONCHIOLITIS

- Etiology- any respiratory virus.
- Respiratory syncytial virus (RSV) bronchiolitis – potential severe complication in premature infants with CLDi.

- Diagnosis- History and physical examination.

- Intervention:
  - Hydration/Nutrition
  - Supplemental O2 or increase supplemental O2.
BRONCHIOLITIS

- **Prevention:** Palivizumab x 5 consecutive months during RSV season

- **Recommendation:**
  - Administer to infants with Mild to Severe BPD (NHLBI definition) in the 1st year of life.
  - Administer to infants with Mild to Severe BPD **IF** on chronic steroid use, diuretic therapy or supplemental O2.
ASPIRATION SYNDROMES

1. Aspiration from above (swallowing dysfunction)

2. Aspiration from below (gastroesophageal reflux disease- GERD)
Swallowing Dysfunction

- In premature infants, disorganized sucking persists up to 34 weeks gestational age.
- Exclude other gross anatomic abnormalities.
- Coordination of sucking, swallowing and breathing - a complex process.
- Dysphagia is from immaturity of this process.
Swallowing dysfunction

Intervention:
• 1. Referral to occupational/speech therapist for feeding strategy.
• 2. Video-fluoroscopic swallowing study.
• 3. Thickening of feeds.

** Risk of necrotizing enterocolitis on the use of xanthan gum for <37 weeks to 44 weeks PCA.
GERD

• “Clinically significant GER” – frequent vomiting, aspiration pneumonia, irritability; failure to thrive

• Silent aspiration worsens pulmonary status → recurrent airway inflammation → reflex bronchospasm.

• Prevalence is unknown.
GERD

Diagnostic studies:
  • Upper GI radiography
  • pH probe monitoring, impedance
  • Upper endoscopy
GERD

• Intervention

  • Choose Wisely campaign: “Avoid routine use of anti-reflux medications”.
  • 1. Reduce volume of feeding, increase frequency.
  • 2. Upright or prone position (only when awake)
  • 3. Acid suppressant: H2 antagonist or Proton pump inhibitor
  • 4. Fundoplication
Airway obstruction

1. Dynamic airway obstruction
   • Tracheomalacia
   • Tracheobronchomalacia

2. Fixed airway obstruction
   • Subglottic stenosis
   • Tracheobronchial stenosis
Tracheomalacia/Tracheobronchomalacia

- Tracheomalacia is the most common congenital anomaly of the trachea.
  - Primary (congenital)- faulty foregut division
  - Secondary (acquired, more common)- degeneration of normal tracheal cartilages.
- More commonly seen in premature infants.
Tracheomalacia/Tracheobronchomalacia

- Result of prolonged intubation, recurrent infection, increased airway pressure.
- Premature infants have immature supporting structures.
- Frequently associated with cardiovascular abnormalities.
- 50% of infants with tracheomalacia have GERD.
Tracheomalacia/Tracheobronchomalacia

• Signs and symptoms:
  • Wheezing not responding to SABA.
  • Recurrent croup. Characteristic barky cough.
  • “Dying spells” ; “BPD spells” - when patient is active, upset and more apparent with Valsalva maneuver.
  • Impaired mucus clearance, inefficient cough → mucus plugging → atelectasis
Tracheomalacia/Tracheobronchomalacia

Diagnostic studies:
- Fluoroscopy
- Chest CT
- Flexible Bronchoscopy – dynamic airway evaluation
Tracheomalacia/Tracheobronchomalacia

• Intervention:
  • Wait and see, feed and grow for mild cases.
  • Use of Ipratropium bromide (Gallagher et al 2011) for moderate cases.
  • Adjustable PEP (positive expiratory pressure) valve - improve efficiency of cough (Sirithangkul et al 2005)
  • Surgery for severe cases: tracheostomy; aortopexy; external splinting, internal stent
Subglottic stenosis/Tracheobronchial stenosis

- Acquired stenosis is more common than congenital form.
- History of prolonged or repeated endotracheal intubation.
- Subglottic stenosis occurs in 1.7 to 8% of neonates with history of intubation.
- Tracheobronchial stenosis – from repeated mucosal injury from suction catheters.
Subglottic/Tracheobronchial stenosis

• Diagnostic studies:
  • CT scan (neck and chest)
  • Rigid bronchoscopy

• Intervention:
  • Tracheostomy
  • Endoscopic dilation with steroid injection
  • Anterior cricoid split
  • Laryngotracheal reconstruction
Pulmonary Edema

• Factors contributing to lung edema in CLDi
  • Increased capillary permeability resulting from lung injury
  • Patent ductus arteriosus
  • Fluid overload

• Commonly used in the NICU for the following reasons:
  • Improve lung compliance.
  • Reduce number of mechanical ventilation days.
  • Furosemide up to 5 days. Thiazide- Spironolactone up to 42 days.
Pulmonary Edema

• Furosemide is used not more than 5 days. Nephrocalcinosis is a known complication.
• Chronic diuretic use: Thiazide- Spironolactone

• Cochrane review:
  • Chronic use of thiazide-spironolactone significantly decreased mortality rate.
  • Improvement of lung compliance at 4 weeks of treatment, but none thereafter.
  • Reduced the use of Furosemide.
Pulmonary Edema

- Intervention:
  - Treat underlying cardiac disease (refer to Cardiology) if hemodynamically unstable.
  - Fluid restriction.
  - Adjust dose for age, if still with recurrent signs and symptoms of pulmonary congestion.
  - Consider giving acute doses of Furosemide if with exacerbations. No chronic diuretics in between.
Apnea of Prematurity

- Occurs in less than 37 weeks PCA. Consider other diagnoses if patient’s PCA is term
- Secondary to immaturity of respiratory center.
- With concomitant upper airway obstruction in most cases.
Apnea of Prematurity

Diagnostic study:
Polysomnography (PSG) determines type of apnea.
- central, obstructive or mixed

Titration of O2 and/or non-invasive positive pressure ventilation (NIPPV) during PSG
Apnea of Prematurity

Intervention:

• Caffeine
• NIPPV in the form of either CPAP or BIPAP.
• Oxygen

** Choosing wisely campaign: “Avoid routine use of pneumograms for pre-discharge assessment of ongoing apnea of prematurity.”**
Apparent Life Threatening Event (ALTE)/SIDS

• Patients with CLDi are at high risk for ALTE/SIDS.

• Deaths are likely from unrecognized hypoxemia.

• Study of Bancalari et al on premature infants 26-33 weeks gestational, those given home O2 survived.

• Peripheral chemoreceptors function is abnormal in CLDi. Ventilatory and arousal responses to hypoxia are blunted.
Apparent Life Threatening Event/SIDS

- Diagnosis of ALTE is based on symptomatology.
- Diagnostic studies may be extensive to exclude primary etiology.

- Intervention:
  - Depends on what system is involved.
  - “Back to sleep campaign”
Pulmonary hypertension

- New BPD - reduced number of alveoli (alveolar simplification).

- Also reduced arteries and abnormal distribution in the lungs → impaired gas exchange → hypoxemia → pulmonary vasoconstriction → increase pulmonary vascular resistance → pulmonary hypertension (PH)
Pulmonary hypertension

- Pulmonary hypertension is about 27-37% of infants with BPD. (An, 2010, Slaughter 2011)

- Diagnostic studies:
  - Echocardiogram
  - Cardiac catheterization
Pulmonary hypertension

• Intervention:
  • Treat lung disease and other ongoing issues contributing to recurrent hypoxia.
  • Treat underlying cardiac lesions if present.
  • Pulmonary vasodilators:
    • Oxygen
    • Sildenafil
    • Bosentan
    • Calcium channel blockers
Pneumonia

• Most common etiology is viral.

• Premature infants follow a complicated course with RSV pneumonia.

• *Streptococcus pneumoniae* – common bacteria in community acquired pneumonia.
Pneumonia

• Diagnostic studies:
  • Check for Influenza/RSV from viral panel.
  • CXR if with moderate to severe respiratory distress and persistent hypoxemia.
Pneumonia

• Intervention:
  • Antibiotic (Amoxicillin) if stable to discharge home. Close follow up is important.
  • Hospitalize if + increasing respiratory distress, poor feeding, lethargy and O2 saturation < 91%.

Prevention:
- Keep immunization up to date.
Weaning Home Oxygen

If patient has documented pulmonary hypertension (PH) - DON’T WEAN!

- No consensus on the best way to wean.
- Consider weaning from O2, if infant is about 12 months old and:
  - keeping it in is not practical.
  - is moving a lot during sleep and strangulation from the O2 tubing becomes a hazard.
Weaning Home Oxygen

- Weaning criteria (Fitzgerald, 2012)
  - No increase work of breathing
  - No evidence of pulmonary hypertension.
  - Target minimum O2 saturation (SPO2) to at least 93-95%.
  - Adequate weight gain.
Weaning Home Oxygen

• ATS Statement 2002:
  • If patient is thriving start weaning at daytime → if SPO2 is adequate →
    wean night time O2 → if SPO2 adequate → Completely wean O2.

• Another option: PSG before weaning night time O2.
Traveling by air

- Aircraft cabins are pressurized up to 8000ft (equivalent FIO2 of 15%)

- High Altitude Simulation Test (HAST)

- If HAST is not accessible, increase O2 by 0.25LPM if flight is > 1 hour.
REFERENCES


Thank you.