Wheezing in infants

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Acute Bronchiolitis

Objectives

1. Understand the aetiology and natural history of bronchiolitis
2. Recognise and be able to describe the clinical features of bronchiolitis and be able to
   relate these to normal physiology.
3. Know how to treat acute bronchiolitis.
4. Be able to advise parents about how to care for a child with a bronchiolitis.

Pneumonia

Objectives

1. Know and understand the aetiology and natural history of pneumonia including
   knowledge of the common causative organisms.
2. Recognize and be able to describe the clinical features of pneumonia and be able to
   relate these to normal physiology.
3. Have knowledge of the treatments available to children with pneumonia including
   antibiotics, oxygen and physiotherapy.
4. Be able to advise parents about how to care for a child with a chest infection.

That entire wheeze is not asthma & asthma does not always wheeze:

1. A wheeze is a musical and continuous sound that originates from oscillations in
   narrowed airways.
2. Wheezing is heard mostly on expiration as a result of critical airway obstruction.
3. Infants are prone to wheeze due to a differing set of lung mechanics in comparison to
   older children and adults.
4. The obstruction to flow is affected by the airway caliber and compliance of the infant
   lung.
5. Resistance to airflow through a tube is inversely related to the radius of the tube to
   the 4th power.
6. In children <5 yr old, small caliber peripheral airways can contribute up to 50% of
   the total airway resistance.
7. Marginal additional narrowing can cause further flow limitation and a subsequent
   wheeze.
Acute bronchiolitis

Etiology:

1. Is predominantly a viral disease. Respiratory syncytial virus (RSV) is responsible for >50% of cases.
2. Other agents include parainfluenza, adenovirus, *Mycoplasma*, and, occasionally, other viruses.

Epidemiology

1. Bronchiolitis is more common in males, those who have not been breast-fed and in those who live in crowded conditions.
2. Older family members are a common source of infection; they may only experience minor respiratory symptoms.

Pathophysiology:

1. Acute bronchiolitis is characterized by bronchiolar obstruction with edema, mucus, and cellular debris.
2. Even minor bronchiolar wall thickening significantly affects airflow Resistance in the small air passages is increased during both inspiration and exhalation, but because the radius of an airway is smaller during expiration, the resultant respiratory obstruction leads to early air trapping and overinflation.
3. If obstruction becomes complete, there will be resorption of trapped distal air, and the child will develop atelectasis.
4. Hypoxemia is a consequence of ventilation-perfusion mismatch early in the course.
5. With severe obstructive disease and tiring of respiratory effort, hypercapnia may develop.

CLINICAL MANIFESTATIONS:

1. Acute bronchiolitis is usually preceded by exposure to an older contact with a minor respiratory syndrome within the previous wk.
2. The infant 1st develops a mild upper respiratory tract infection with sneezing and clear rhinorrhea.
3. This may be accompanied by diminished appetite and fever of 38.5–39°C.
4. Gradually, respiratory distress ensues, with paroxysmal wheezy cough, dyspnea, and irritability.
5. The infant is often tachypneic, which may interfere with feeding.
6. The child does not usually have other systemic complaints, such as diarrhea or vomiting.
7. Apnea may be more prominent than wheezing early in the course of the disease, particularly with very young infants (<2 mo old) or former premature infants.
8. The physical examination is characterized most prominently by wheezing.
9. The degree of tachypnea does not always correlate with the degree of hypoxemia or hypercarbia, so the use of pulse oximetry and noninvasive carbon dioxide determination is essential.

10. Work of breathing may be markedly increased, with nasal flaring and retractions.

11. Auscultation may reveal fine crackles or overt wheezes, with prolongation of the expiratory phase of breathing. Barely audible breath sounds suggest very severe disease with nearly complete bronchiolar obstruction.

12. Hyperinflation of the lungs may permit palpation of the liver and spleen.

**DIAGNOSTIC EVALUATION:**

1. In acute bronchiolitis: chest radiography reveals hyperinflated lungs with patchy atelectasis.
2. The white blood cell and differential counts are usually normal.
3. Viral testing (usually rapid immunofluorescence, polymerase chain reaction, or viral culture) is helpful if the diagnosis is uncertain or for epidemiologic purposes.
4. The diagnosis is clinical, particularly in a previously healthy infant presenting with a first-time wheezing episode during a community outbreak.

**TREATMENT:**

1. Infants with acute bronchiolitis who are experiencing respiratory distress should be hospitalized; the mainstay of treatment is supportive.
2. If hypoxemic, the child should receive cool humidified oxygen.
3. Sedatives are to be avoided because they may depress respiratory drive.
4. The infant is sometimes more comfortable if sitting with head and chest elevated at a 30-degree angle with neck extended.
5. The risk of aspiration of oral feedings may be high in infants with bronchiolitis, owing to tachypnea and the increased work of breathing. The infant may be fed through a nasogastric tube.
6. If there is any risk for further respiratory decompensation potentially necessitating tracheal intubation, however, the infant should not be fed orally but be maintained with parenteral fluids.
7. Frequent suctioning of nasal and oral secretions often provides relief of distress or cyanosis. Oxygen is indicated in all infants with hypoxia.
8. Bronchodilators produce modest short-term improvement in clinical features, but the statistical improvement in clinical scoring systems seen with them is not always clinically significant.
9. A trial dose of inhaled bronchodilator may be reasonable, with further therapy predicated on response in the individual patient.
10. Corticosteroids are not recommended in previously healthy infants with RSV.
11. Antibiotics have no value unless there is secondary bacterial pneumonia.
12. Likewise, there is no support for RSV immunoglobulin administration during acute episodes of RSV bronchiolitis.
13. Ribavirin, an antiviral agent administered by aerosol, has been used for infants with congenital heart disease or chronic lung disease.
PROGNOSIS:

1. Infants with acute bronchiolitis are at highest risk for further respiratory compromise in the 1st 48–72 hr after onset of cough and dyspnea; the child may be desperately ill with air hunger, apnea, and respiratory acidosis.
2. The case fatality rate is <1%, with death attributable to apnea, uncompensated respiratory acidosis, or severe dehydration.
3. After this critical period, symptoms may persist. The median duration of symptoms in ambulatory patients is ≈12 days.
4. Infants with conditions such as congenital heart disease, bronchopulmonary dysplasia, and immunodeficiency often have more severe disease, with higher morbidity and mortality.
5. There is a higher incidence of wheezing and asthma in children with a history of bronchiolitis unexplained by family history or other atopic syndromes.

PREVENTION:

1. Reduction in the severity and incidence of acute bronchiolitis due to RSV is possible through the administration palivizumab, an intramuscular monoclonal antibody to the RSV F protein, before and during RSV season. Palivizumab is recommended for infants <2 yr of age with chronic lung disease (bronchopulmonary dysplasia) or prematurity.
2. Meticulous hand washing is the best measure to prevent nosocomial transmission.

Other wheezing associated conditions:

1. **Allergy and asthma:** are important causes of wheezing and probably generate the most questions by the parents of a wheezing infant. Asthma is characterized by airway inflammation, bronchial hyperreactivity, and reversibility of obstruction.
2. **Cystic fibrosis:** should be considered in those infants who seem to fall out of the range of a normal clinical course. Cystic fibrosis is one such entity; suspicion increases in a patient with persistent respiratory symptoms, digital clubbing, malabsorption, failure to thrive, electrolyte abnormalities, or a resistance to bronchodilator treatment.
3. **Congenital malformations:** of the respiratory tract cause wheezing in early infancy. These findings can be diffuse or focal and can be from an external compression or an intrinsic abnormality. **External vascular compression** includes a vascular ring, in which the trachea and esophagus are surrounded completely by vascular structures, or a vascular sling, in which the trachea and esophagus are not completely encircled. **Cardiovascular causes** of wheezing include dilated chambers of the heart including massive cardiomegaly, left atrial enlargement, and dilated pulmonary arteries. Pulmonary edema caused by heart failure can also cause wheezing by lymphatic and bronchial vessel engorgement that leads to obstruction and edema of the bronchioles and further obstruction.
4. **Foreign body aspiration:** can cause acute or chronic wheezing. It is estimated that 78% of those who die from foreign body aspiration are between 2 mo and 4 yr old. Even in young infants, a foreign body can be ingested if given to the infant by another person such as an older sibling. Infants who have atypical histories or misleading clinical and radiologic findings may be misdiagnosed with asthma or another obstructive disorder as inflammation and granulation develop around the foreign body. Esophageal foreign body can transmit pressure to the membranous trachea, causing compromise of the airway lumen.

5. **Gastroesophageal reflux:** can cause wheezing with or without direct aspiration into the tracheobronchial tree. Without aspiration, the reflux is thought to trigger a vagal or neural reflex, causing increased airway resistance and airway reactivity. Aspiration from gastroesophageal reflux or from the direct aspiration from oral liquids can also cause wheezing.

6. **Trauma and tumors:** are much more rare causes of wheezing in infants. Trauma of any type to the tracheobronchial tree can cause an obstruction to airflow. Accidental or non-accidental aspirations, burns, or scalds of the tracheobronchial tree can cause inflammation of the airways and subsequent wheezing. Any space-occupying lesion either in the lung itself or extrinsic to the lung can cause tracheobronchial compression and obstruction to airflow.
Pneumonia

Community-acquired pneumonia (CAP):

Is defined as an acute infection of the pulmonary parenchyma in a patient who has acquired the infection in the community, as distinguished from hospital-acquired (nosocomial) pneumonia.

Viral pneumonia:

1. The onset of viral pneumonia is gradual and associated with preceding upper airway symptoms.
2. Auscultatory findings are usually diffuse and bilateral.
3. Infiltrates are usually interstitial.
4. Most children younger than 3 to 5 years of age who are admitted to the hospital with pneumonia have viral pneumonia (e.g., respiratory syncytial virus).
5. This is particularly true in the absence of lobar (or lobular) infiltrate and pleural effusion. Viral pneumonia does not require antibiotic therapy, unless a mixed infection or secondary bacterial infection is suspected.

Typical bacterial pneumonia:

Typical bacterial pneumonia may occur in children of all ages.

Clues to bacterial pneumonia include:

1. Alveolar infiltrate, lobar or segmental consolidation, large pleural effusion, elevated CRP, leukocytosis, signs of sepsis, and chills.
2. Other complications (pneumatoceles, cavitations, necrotizing processes) also are suggestive of typical bacterial etiology.

Streptococcus pneumoniae is the most common type of bacterial cause of pneumonia in children of all ages.

Other potential bacterial pathogens that may need to be included in empiric therapy for hospitalized children include S. aureus including methicillin-resistant S. aureus, Streptococcus pyogenes (Group A streptococcus), Haemophilus influenzae type b (if unimmunized), nontypeable H. influenzae, and Moraxella catarrhalis.

Treatment

1. Oral amoxicillin may be appropriate for children older than 6 months with uncomplicated pneumonia
2. Antibiotic regimens for uncomplicated bacterial pneumonia in hospitalized children when S. aureus is not a consideration include:
Ceftriaxone (50 to 75 mg/kg intravenously [IV] once daily up to a maximum dose of 4 g/day), or Cefotaxime (150 to 200 mg/kg per day IV in three or four divided doses up to a maximum of 8 to 10 g/day).

**Atypical bacterial pneumonia:**
Atypical bacterial pneumonia (M. pneumoniae and Chlamydia pneumoniae) is most common in children older than 5 years.

Clues to atypical bacterial pneumonia include:

1. Abrupt onset of constitutional findings (malaise and myalgia, headache, conjunctivitis, photophobia, sore throat),
2. Gradually worsening non-productive cough despite improvement of other symptoms, wheezing, rash, and interstitial infiltrates.

Atypical bacterial pathogens include C. trachomatis in afebrile infants, and M. pneumoniae and C. pneumoniae in older children and adolescents.

**Treatment:**
Antibiotic regimens for atypical bacterial pneumonia in hospitalized children include:

1. Erythromycin 40 mg/kg per day IV in four divided doses, maximum 4 g/day, or
2. Azithromycin 5 mg/kg once per day IV, maximum 500 mg/day, or
3. For children older than 8 years: Doxycycline (4 mg/kg per day IV in two divided doses; maximum 200 mg/day)

2. **Moderately severe CAP:** Children with moderately severe CAP may benefit from combination empiric therapy with a macrolide and a beta-lactam antibiotic
3. **Complicated CAP:** The spectrum of empiric coverage should be broadened for children with CAP who have complications, such as parapneumonic effusion and/or lung abscess. The expanded spectrum should include coverage for beta-lactam-resistant isolates, and community-associated methicillin-resistant S. aureus; coverage for anaerobes and gram-negative organisms also may be necessary for children with lung abscess.

Appropriate regimens include:

Clindamycin (30 to 40 mg/kg maximum of 1 to 2 g/day),

Vancomycin is an alternative (40 mg/kg per day IV in four divided doses up to a maximum of 2 to 4 g/day).
Nosocomial pneumonia:
Empiric treatment of nosocomial pneumonia should afford coverage for the usual nosocomial pathogens: S. aureus, Enterobacteriacea, Pseudomonas aeruginosa, and anaerobes.

1. Acceptable broad-spectrum empiric regimens include an aminoglycoside plus:

   Piperacillin-tazobactam or Meropenem.

2. Patients with true beta-lactam hypersensitivity (i.e., type 1 hypersensitivity reaction) can be treated with a combination of clindamycin plus an aminoglycoside.

Aspiration pneumonia:
Empiric antibiotic regimens for community-acquired aspiration pneumonia must cover oral anaerobes.

Appropriate antibiotics regimens for hospitalized children include:

1. Clindamycin or Meropenem
2. Immunocompromised host — vancomycin if methicillin-resistant staphylococcus is considered, and possibly trimethoprim-sulfamethoxazole for Pneumocystis jirovecii (formerly carinii).

Duration of therapy

1. **Parenteral therapy:** It is common to switch to oral therapy in patients who have received parenteral antibiotics when the patient has become afebrile for 24 to 48 hours and is able to keep down food.

2. **Uncomplicated cases:** Seven to 10 days of combined parenteral and oral therapy should be adequate for routine pathogens causing uncomplicated infection

3. **Complicated cases:** Treatment of complications, such as necrotizing pneumonia and lung abscess, requires a prolonged course of antibiotic therapy, usually initiated parenterally. The duration is determined by the clinical response, but is usually a total of four weeks or two weeks after the patient is afebrile and has improved clinically.

Prognosis:
Most otherwise healthy children with pneumonia recover without sequelae, even if the pneumonia is complicated.