

BRONCHIOLITIS

Acute bronchiolitis is a common disease of the lower respiratory tract (LRT) in infants, resulting from an inflammatory obstruction of the small airways. *The peak incidence is around 6 months of age ranging from 1 month –2 years, whereas the severe disease is more common infants aged 1-3 months.* Bronchiolitis is a *seasonal* disease, with a peak activity during winter & early spring.

Etiology & epidemiology

- ✚ Predominantly a viral disease. RSV is responsible for >50% of cases. Other agents include parainfluenza, adenovirus, and *Mycoplasma*. Human metapneumovirus and human bocavirus, which may be a primary cause of viral respiratory infection or occur as a co-infection with RSV.
- ✚ There is no evidence of bacterial cause for bronchiolitis, although bacterial pneumonia is sometimes confused clinically with bronchiolitis & bronchiolitis is rarely followed by bacterial super infection.
- ✚ Bronchiolitis is more common in boy in those who not have been breast fed, & in those who live in crowded conditions. Older family members are common source of infection but may experience only minor symptoms.

Pathophysiology

- ✚ Not all infected infants develop LRTI. Host anatomic and immunologic factors play a significant role in the severity of the clinical syndrome, as does the nature of the viral pathogen. Infants with pre-existent smaller airways and diminished lung function have a more-severe course. In addition, RSV infection incites a complex immune response. Eosinophils degranulate and release eosinophil cationic protein, which is cytotoxic to airway epithelium. Innate immunity plays a significant role and can depend on polymorphisms in toll-like receptor (TLR), interferon (IF), interleukins (IL), and nuclear factor κ B (NF κ B). Chemokines and cytokines such as tumor necrosis factor α (TNF- α) may be differentially expressed depending on the inciting virus.
- ✚ *Acute bronchiolitis is characterized by bronchiolar obstruction with edema , mucus, & cellular debris.* If obstruction is complete with resorption of the trapped air, the child will develop *atelectasis*.
- ✚ *Hypoxemia* is a consequence of ventilation-perfusion mismatch early in the course. With severe disease, *hypercapnia* develops.

Clinical Manifestations

- ✚ The illness is usually preceded by exposure to an older contact with a minor respiratory syndrome within the previous week.
- ✚ *The infants 1st develop a mild upper respiratory tract (URT) infection* with sneezing & clear rhinorrhea. This may be accompanied by ↓appetite & fever (ranging from subnormal to markedly ↑temperature).
- ✚ *Gradually, respiratory distress ensues with paroxysmal wheezy cough, dyspnea, & irritability.* The infants are often tachypneic which interferes with feeding. The child does not usually have other systemic complaints as diarrhea & vomiting. Apnea may be more prominent than wheezing early in the course of the disease, particularly in very young infants.

- ✚ The physical examination is characterized is often dominated by wheezing. The degree of tachypnea does not always correlate with the degree of hypoxemia or hypercarbia, so pulse oximetry and noninvasive determination of carbon dioxide is essential. Work of breathing may be markedly increased, with nasal flaring and retractions. Auscultation might 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. Hyperinflation of the lungs can permit palpation of the liver and spleen.
- ✚ *The single best indicator at the initial assessment appears to be the O₂ saturation which can be determined by pulse oximetry. SaO₂ <95% correlates with more severe disease. An arterial blood gas with PaO₂ of ≤65 mmHg or PaCO₂ of > 40 mmHg is particularly worrisome. Other predictors of ↑severity include: an ill or toxic appearance, history of prematurity (gestational age < 34 wk), atelectasis on CXR, respiratory rate (RR) > 70 beat per minute (bpm), infants <3 months old, & with underlying diseases as congenital heart diseases (CHD), bronchopulmonary dysplasia (BPD), immunodeficiency, & asthma.*

Diagnosis

- *Diagnosis is clinical.*
- *CXR →hyper inflated lungs with patchy atelectasis.*
- *WBC count & differential counts are usually normal without lymphopenia seen with other viral illnesses.*
- *Viral testing (usually rapid immunofluorescence, PCR, & viral culture).*

Differential diagnosis

- *Asthma : It is most commonly confused with acute bronchiolitis. The 2 conditions maynot be distinguished during the 1st episode, but repeated episodes of wheezing, absence ofthe viral prodrome, & presence of family history of atopy, or asthma support thediagnosis of asthma.*
- Foreign body in the trachea.
- Tracheomalacia, bronchomalacia, vascular ring.
- CHF, cystic fibrosis, pertussus, GER.

Treatment

■ Hospitalization

- Age < 12 wks.
- Preterm
- Cardiovascular, pulmonary, or immunologic disease.
- Extreme tachypnea
- Inability to take oral feedings
- Hypoxia.

Supportive measures.

- Hypoxemic children should receive cool humidified oxygen.
- Sedatives are to be avoided because they can depress respiratory drive.
- The infant is sometimes more comfortable if sitting with head and chest elevated at a 30-degree angle with neck extended.
- The infant may be fed through a nasogastric tube risk of aspiration of oral feedings may be high in infants with bronchiolitis, owing to tachypnea and the increased work of breathing.
- High-flow nasal cannula therapy can reduce the need for intubation in patients with impending respiratory failure.
- Frequent suctioning of nasal and oral secretions often provides relief of distress or cyanosis.
- Infant should not be fed orally but be maintained with parenteral fluids.

Medication

- **Bronchodilators** can produce modest short-term improvement in clinical features.

- **Corticosteroids**, whether parenteral, oral, or inhaled, have been used for bronchiolitis despite conflicting and often negative studies. Corticosteroids are not recommended in previously healthy infants with RSV.
- **Ribavirin**, an antiviral agent administered by aerosol, has been used for infants with congenital heart disease or chronic lung disease.
- **Antibiotics** have no value unless there is coexisting bacterial infection.
- No support for **RSV immunoglobulin** administration during acute episodes of RSV bronchiolitis in previously healthy children.
- Combined therapy with **nebulized epinephrine and dexamethasone** has been used but is not currently recommended.
- **Nebulized hypertonic saline** has also been reported to have some benefit.

Prognosis

The case fatality rate is <1%, with death attributable to apnea, respiratory arrest, or severe dehydration. After this critical period, symptoms can persist.

The median duration of symptoms in ambulatory patients is ~12 days.

There is a higher incidence of wheezing and asthma in children with a history of bronchiolitis unexplained by family history or other atopic syndromes.

It is unclear whether bronchiolitis incites an immune response that manifests as asthma later or whether those infants have an inherent predilection for asthma that is merely unmasked by their episode of RSV.

Approximately 60% of infants who wheeze will stop wheezing.

Prevention

- ✓ *RSV-IVIG (intravenous immunoglobulins) & IM monoclonal antibodies to RSV (palivizumab) are effective in preventing severe RSV disease in the high-risk infants when given before & during RSV season. Palivizumab is recommended for infants < 2 yrs of age with chronic lung disease (BPD) or prematurity.*
- ✓ *Meticulous hand washing is the best measure to prevent the nosocomial transmission.*