

Respiratory system

Lecture 1

م.د. اسيل جاسم محمد

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

- is predominantly a viral disease . RSV is responsible for more than 50% of cases. Other agents include parainfluenza, adenovirus, rhinovirus, and Mycoplasma. human metapneumovirus and human bocavirus, which may be a primary cause of viral respiratory infection or occur as a coinfection with RSV.
- there is no evidence of a bacterial cause for bronchiolitis and bronchiolitis is rarely followed by bacterial superinfection.
- 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 \downarrow appetite & fever (ranging from subnormal to markedly \uparrow 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. SpO₂ <95% correlates with more severe disease. An arterial blood gas with PaO₂ of \leq 65 mmHg or PaCO₂ of > 40 mmHg is particularly worrisome. Other predictors of \uparrow 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 is clinical. o CXR → hyper inflated lungs with patchy atelectasis. o WBC count & differential counts are usually normal without lymphopenia seen with other viral illnesses. o Viral testing (usually rapid immunofluorescence, PCR, & viral culture). Differential diagnosis □ Asthma : It is most commonly confused with acute bronchiolitis. The 2 conditions may not be distinguished during the 1st episode, but repeated episodes of wheezing, absence of the viral prodrome, & presence of family history of atopy, or asthma support the diagnosis of asthma. □ Foreign body in the trachea. □ Tracheomalacia, bronchomalacia, vascular ring. □ CHF, cystic fibrosis, pertussis, 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
.predisposition 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 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