Bronchitis.doc

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Bronchitis refers to nonspecific bronchial inflammation and is associated with a number of childhood conditions. Acute bronchitis is a syndrome, usually viral in origin, with cough as a prominent feature.

Acute tracheobronchitis is a term used when the trachea is prominently involved. Nasopharyngitis may also be present, and a variety of viral and bacterial agents, such as those causing influenza, pertussis, and diphtheria, may be responsible. Isolation of common bacteria such as pneumococcus, Staphylococcus, and Streptococcus from the sputum may not imply a bacterial cause requiring antibiotic therapy.

Asthmatic bronchitis is an obsolete term; wheezing and bronchial inflammation are integral findings of asthma, and asthma exacerbations are commonly triggered by upper respiratory tract infections. Thus, use of the term asthmatic bronchitis may obscure the understanding on the part of patient and family that this is asthma.

ACUTE BRONCHITIS
Clinical Manifestations.
Acute bronchitis is commonly preceded by a viral upper respiratory tract infection. Thus, it is more common in the winter when respiratory viral syndromes predominate. The tracheobronchial epithelium is invaded by the infectious agent, leading to activation of inflammatory cells and release of cytokines. Constitutional symptoms, such as fever and malaise, follow. The tracheobronchial epithelium may become significantly damaged or hypersensitized, leading to a protracted cough lasting 1–3 wk.

Commonly, the child first presents with nonspecific upper respiratory infectious symptoms, such as rhinitis. Three to 4 days later, a frequent, dry, hacking cough develops, which may or may not be productive. After several days, the sputum may become purulent, but purulent sputum indicates leukocyte migration and does not necessarily imply bacterial infection. Many children swallow their sputum, and this may produce emesis. Chest pain may be a prominent complaint in older children, exacerbated by coughing. The mucus gradually thins, usually within 5–10 days, and then the cough gradually abates. The entire episode usually lasts about 2 wk and seldom longer than 3 wk.

Findings on physical examination vary with age of the patient and stage of the disease. Early findings are absent or low-grade fever and upper respiratory signs such as nasopharyngitis, conjunctivitis, and rhinitis. Auscultation of the chest may be unremarkable at this early phase. As the syndrome progresses and cough worsens, breath sounds become coarse, with coarse and fine crackles and scattered high-pitched wheezing. Chest radiographs are normal or may have increased bronchial markings.

The principal objective of the clinician is to exclude pneumonia, which is more likely caused by bacterial agents requiring antibiotic therapy. In at least one study of adults, absence of abnormality of vital signs (tachycardia, tachypnea, fever) and chest examination reduced the likelihood of pneumonia.

Criteria of acute bronchitis
I. Clinical:
- cough - dry and rough at the beginning of disease, gradually becoming productive;
- symptoms of intoxication are not expressed greatly and quickly disappear;
- symptoms of respiratory insufficiency are absent;
- physical signs: on percussion there is a slight tympanic resonance, on auscultation - dry and various bubbling rales, heard on both sides of lungs.

Obstructive bronchitis is a variant of acute bronchitis which proceeds with respiratory tract obstruction because of bronchospasm, mucous edema, hypersecretion and pressure from without.

Signs of respiratory tract obstruction: persistent, "spastic" cough, expiratory dyspnea, oral crepitations, dry and various bubbling rales.
II. X-ray:
- strengthened lung figure, at the same time absence of focal shadow;
- signs of disturbances of bronchial permeability: irregular pneumatisation of lungs (focus of hyper- and hypoventilation), lobular atelectasis.

III. Laboratory
- hematological: normal leukocyte count or leukopenia, lymphocytosis, monocytosis.
Red blood cells are not changed. ESR is not increased.

Differential Diagnosis.
Persistent or recurrent symptoms should lead the clinician to consider entities other than acute bronchitis. Many entities may manifest with cough as a prominent symptom.

Treatment.
There is no specific therapy for acute bronchitis. The disease is self-limited, and antibiotics, although frequently prescribed, do not hasten improvement in uncomplicated acute bronchitis. Frequent shifts in position may facilitate pulmonary drainage in infants. Older children are sometimes more comfortable with humidity, but this does not shorten the disease course. Cough suppressants may produce symptomatic relief but may also increase the risk of suppuration and inspissated secretions and therefore should be used judiciously. Antihistamines dry secretions and are not helpful, and expectorants are likewise not indicated.

CHRONIC BRONCHITIS

Chronic bronchitis is well recognized in adults, formally defined as 3 mo or more of productive cough each year for 2 yr or more. The disease may develop insidiously, with episodes of acute obstruction alternating with quiescent periods. A number of predisposing conditions may lead to progression of airflow obstruction or chronic obstructive pulmonary disease (COPD), with smoking as the major factor (up to 80% of patients have a smoking history). Other conditions include air pollution, occupational exposures, and repeated infections.

The applicability of this definition to children is unclear. The existence of chronic bronchitis as a distinct entity in children is controversial. However, like adults, children with chronic inflammatory diseases or those with toxic exposures may develop damaged pulmonary epithelium. Thus, chronic or recurring cough in children should guide the clinician to search for underlying pulmonary or systemic disorders.

Recurrent bronchitis is the disease with relapsing of acute bronchitis 3 and more times a year during 1-2 years. The absence of clinical obstruction and duration of clinical manifestation for 2 weeks and longer every relapse are common.

Phases of pathologic process: exacerbation, remission.

CIGARETTE SMOKING AND AIR POLLUTION

Exposure to environmental irritants, such as tobacco smoke and air pollution, can incite or aggravate cough. There is a well-established association between tobacco exposure and pulmonary disease, including bronchitis and wheezing. This may occur either through cigarette smoking or by exposure to passive smoke. Marijuana smoke is another irritant sometimes overlooked when eliciting a history.

A number of pollutants are also likely candidates as precipitants of lung disease, including particulate matter, ozone, and nitrogen dioxide. Because these substances coexist in the atmosphere, the relative contribution of any one to pulmonary symptoms is difficult to discern.

Bronchiolitis

Acute bronchiolitis is a common disease of the lower respiratory tract in infants, resulting from inflammatory obstruction of the small airways. By age 2 yr nearly all children have been infected, with severe disease more common among infants aged 1–3 mo. Bronchiolitis is seasonal, with peak activity during winter and early spring.

Etiology and Epidemiology.

Acute bronchiolitis is predominantly a viral disease. Respiratory syncytial virus (RSV) is responsible for more than 50% of cases. Other agents include parainfluenza, adenovirus, Mycoplasma, and occasionally other viruses. There is no evidence of a bacterial cause for bronchiolitis, although bacterial pneumonia is sometimes confused clinically with bronchiolitis and bronchiolitis may be followed by bacterial superinfection.

Estimates suggest that 50,000–80,000 of hospitalizations annually among children younger than 1 yr are attributable to RSV infection, representing an increase over the past decade. This increase may reflect increased attendance of infants in daycare centers, changes in criteria for hospital admission, and/or improved survival of premature infants and others at risk for severe RSV-associated disease.

Bronchiolitis is more common in males, in those who have not been breast-fed, and in those who live in crowded conditions. Older family members are a common source of infection but may experience only minor respiratory symptoms. The clinical manifestations of lower respiratory tract illness (LRTI) seen in young infants may be minimal in older patients, in whom bronchiolar edema is better tolerated.

Pathophysiology.

Not all infected infants develop LRTI. Host anatomic and immunologic factors seem to play a significant role in the severity of the clinical syndrome. 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. IgE antibody release may be related to wheezing. Other mediators invoked in the pathogenesis of airway inflammation include chemokines such as interleukin-8 (IL-8), macrophage-inflammatory protein (MIP) 1a, and RANTES (Regulated on Activation, Normal T-cell Expressed and Secreted). RSV-infected infants who wheeze express higher levels of interferon-a in the airway as well as leukotrienes. Additionally, altered regulation of surfactant proteins A and B may exacerbate the abnormal lung function of infants with bronchiolitis. These findings suggest a complex cellular
dysregulation producing the clinical syndrome.

Acute bronchiolitis is characterized by bronchiolar obstruction with edema, mucus, and cellular debris. Even minor bronchiolar wall thickening significantly affects airflow because resistance is inversely proportional to the fourth power of the radius of the bronchiolar passage. 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 ball valve respiratory obstruction leads to early air trapping and overinflation. If obstruction is complete with resorption of trapped air, the child will develop atelectasis.

These cellular and pathologic processes impair normal pulmonary gas exchange. 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 infant first develops a mild upper respiratory tract infection with sneezing and clear rhinorrhea. This may be accompanied by diminished appetite and fever of 38.5–39°C (101–102°F), although the temperature may range from subnormal to markedly elevated. Gradually, respiratory distress ensues, with paroxysmal wheezy cough, dyspnea, and irritability. The infant is often tachypneic, which interferes with feeding. The child does not usually have other systemic complaints, such as diarrhea or vomiting. Apnea may be more prominent than wheezing early in the course of the disease, particularly with very young infants.

The physical examination is characterized most prominently by wheezing. 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. Work of breathing may be markedly increased, with nasal flaring and retractions. 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. Hyperinflation of the lungs may permit palpation of the liver and spleen.

Chest radiography reveals hyperinflated lungs with patchy atelectasis. This may be difficult to distinguish from early bacterial pneumonia.

The white blood cell and differential counts are usually normal, without the lymphopenia seen with other viral illnesses. The utility of viral testing (usually rapid immunofluorescence, polymerase chain reaction, or viral culture) is debatable. The diagnosis is clinical, particularly in a previously healthy infant presenting with a first-time wheezing episode during a community outbreak. However, because concurrent bacterial infection is highly unlikely, confirmation of viral bronchiolitis may obviate the need for a sepsis evaluation in a febrile infant.

Differential Diagnosis.

The condition most commonly confused with acute bronchiolitis is asthma. The two conditions may not be distinguishable during the first episode, but repeated episodes of wheezing, absence of a viral prodrome, and presence of a family history of atopy or asthma supports a diagnosis of asthma. Other entities that may be confused with bronchiolitis in young infants include foreign body in the trachea, tracheo- or bronchomalacia, vascular rings, congestive heart failure, cystic fibrosis, or pertussis.

Course and Prognosis.

During the first 48–72hr after onset of cough and dyspnea the infant is at highest risk for further respiratory compromise; he or she may be desperately ill with air hunger, apnea, and respiratory acidosis. The case fatality rate is less than 1%, with death attributable to apnea, uncompensated respiratory acidosis, or severe dehydration. After this critical period, symptoms may persist. In one study of ambulatory children in South Africa, the median duration of symptoms was 12 days. Infants with conditions such as congenital heart disease, bronchopulmonary dysplasia, and immunodeficiency often have more severe disease, with higher morbidity and mortality.

Recurrent Wheezing after Bronchiolitis.

Epidemiologic studies with several year follow-up of index and control children show 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.

Treatment.

Infants with respiratory distress should be hospitalized; the mainstay of treatment is supportive. If hypoxemic, the child should receive cool humidified oxygen. Sedatives are to be avoided because they may 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 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. However, if there is any risk for further respiratory decompensation potentially necessitating tracheal intubation, the infant should be kept NPO and maintained with parenteral fluids.
A number of agents have been proposed as adjunctive therapies for bronchiolitis. 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. Several studies have included both infants with first-time and recurrent wheezing, complicating interpretation of the data. Nebulized epinephrine may be more effective than β-agonists. A trial dose of inhaled bronchodilator may be reasonable, with further therapy predicated on response in the individual patient.

Corticosteroids, whether parenteral, oral, or inhaled, are widely used despite conflicting studies. Differences of diagnostic criteria, measures of effect, timing and route of administration, and severity of illness complicate these studies. In a meta-analysis of steroid use, pooling of all studies and length-of-stay (LOS) plus duration-of-symptoms as outcomes yielded mean reduction in LOS of less than 1 day per patient. This effect disappeared if studies were used measuring LOS only or clearly excluding patients with previous episodes of wheezing. Thus, the theoretical benefits of corticosteroids do not outweigh their risks, side effects, and expense, and they are not indicated for 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. There is no convincing evidence of a positive impact on clinically important outcomes such as mortality and duration of hospitalization. Antibiotics have no value unless there is secondary bacterial pneumonia. Likewise, there is no support for RSV immune globulin administration during acute episodes of RSV bronchiolitis.

Prevention.

Pooled hyperimmune RSV intravenous immunoglobulin (RSV-IVIG, RespiGam) and palivizumab, (Synagis) an intramuscular monoclonal antibody to the RSV F protein are effective in preventing severe RSV disease in high-risk infants when given before and during RSV season. Palivizumab is recommended for infants younger than age 2 yr with chronic lung disease (bronchopulmonary dysplasia) or prematurity. Because of increased mortality after RespiGam administration to infants with symptomatic cyanotic congenital heart disease, the best approach to this population is still under investigation. Meticulous handwashing is the best measure to prevent nosocomial transmission.