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Collage of Medicine

PNEUMONIA IN CHILDREN

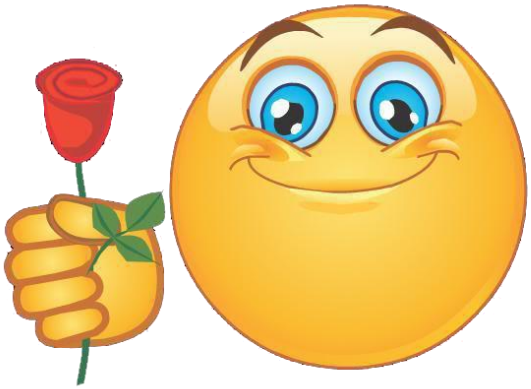
Research Submitted By:

Mohammed Raad Hadi

Sixth Year Student/ Medical Collage/ Diyala University

Supervised By:

Assistant Prof. Aseel Jasim MBChB.FICPS



شكر وتقدير...

اقدم شكري وامتناني بعد الله الى

عمادة جامعة ديالى ...

والى السيد العميد المحترم .....

والى الدكتورة أسيل جاسم محمد المحترمة ...

(قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا ۖ إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ)

اية 32 سورة البقرة

الاهداء

لن تكفي جُمل الشُّكر، وحتى لو بلغت ملء الأرض والسماء، أن تُعبِّر عن فضل أمي.

لن تستطيع كلماتي أن تصف مدى شعوري بالامتنان لصاحب الصدر الرحب والدي.

رُفقاء الدَّرب، والأهل، والخلَّان.

أُهديكم جميعًا بحثي المتواضع

Content

Pneumonia in children

Abstract

Respiratory disease is common in pediatrics and diagnosing pneumonia may be clinically challenging. Changes in pneumococcal resistance and immunization practices continue to change the incidence and etiologies of pneumonia. Careful attention to epidemiological, seasonal, and specific pediatric clinical factors and using adjunct radiographs and laboratory tests should guide the emergency physician in his or her management strategy, including selection of antibiotics and inpatient or outpatient disposition.

Introduction

Globally, pneumonia is an inflammation of the parenchyma of the lungs. Leading cause of morbidity and mortality in children younger than the age of 5 years. Although the majority of deaths attributed to pneumonia in children are mostly in the developing world, the burden of disease is substantial, and there are significant healthcare-associated costs related to pneumonia in the developed world. This activity reviews the cause, pathophysiology, and presentation of pediatric pneumonia and highlights the role of the inter-professional team in its management.[1]

Pneumonia and other lower respiratory tract infections are the leading causes of death worldwide. Because pneumonia is common and is associated with significant morbidity and mortality, promptly diagnosing pneumonia, correctly recognizing any complications or underlying conditions, and appropriately treating patients are all important. Although in developed countries the diagnosis is usually made on the basis of radiographic findings, the World Health Organization (WHO) has defined pneumonia solely on the basis of clinical findings obtained by visual inspection and on the timing of the respiratory rate.[1]

Pneumonia may originate in the lung or may be a focal complication of a contiguous or systemic inflammatory process. Abnormalities of airway potency as well as alveolar ventilation and perfusion occur frequently due to a range of possible mechanisms. These derangements often significantly alter gas exchange and dependent cellular metabolism in the many tissues and organs that determine survival and contribute to quality of life. Recognition, prevention, and treatment of these problems are major factors in the care of children with pneumonia. [1]

Etiology

Although most cases of pneumonia are caused by microorganisms, noninfectious causes include aspiration of food or gastric acid, foreign bodies, hydrocarbons. The cause of pneumonia in an individual patient is often difficult to determine because direct culture of lung tissue is invasive and rarely performed. **(2)**

Cultures performed on specimens obtained from the upper respiratory tract or "sputum" often do not accurately reflect the cause of lower respiratory tract infection. With the use of of-the-best diagnostic testing, a bacterial or viral cause of pneumonia can be identified in 40-80% of children with community-acquired pneumonia. (2)

Streptococcus pneumoniae (pneumococcus) is the most common bacterial pathogen in children 3 wk to 4 yr of age, whereas Mycoplasma pneumoniae and Chlamydial pneumoniae are the most frequent pathogens in children 5 yr and older. (2)

In addition to pneumococcus, other bacterial causes of pneumonia in previously healthy children include group A streptococcus (Streptococcus pyogenes), and Staphylococcus aureus,and H influenza . S. pneumoniae, H. influenzae, and S. aureus are the major causes of hospitalization and death from bacterial pneumonia among children in developing countries.

The incidence of H. influenzae has been significantly reduced in areas where routine Hib immunization has been implemented. (2)

Viral pathogens are a prominent cause of lower respiratory tract infections in infants and children <5 yr of age. Viruses are responsible for 45% of the episodes of pneumonia identified in hospitalized children in Dallas. Unlike bronchiolitis, for which the peak incidence is in the 1st yr of life, the highest frequency of viral pneumonia occurs between the ages of 2 and 3 yr, decreasing slowly thereafter. Of the respiratory viruses, influenza virus and respiratory syncytial virus (RSV) (are the major pathogens, especially in children <3 yr of age. [2]

Other common viruses causing pneumonia include parainfluenza viruses, adenoviruses, rhinoviruses, and human metapneumovirus. [2]

Epidemiology

Immunizations have markedly reduced the incidence of pneumonia caused by pertussis, diphtheria, measles, Haemophilus influenzae type b, and S. pneumoniae. Where used, bacille Calmette-Guérin (BCG) immunization for tuberculosis has also had some impact. Pneumonia is the single largest contributor of childhood mortality worldwide, killing an estimated 1 million children under 5 years of age annually. Risk factors for lower respiratory tract infections include gastroesophageal reflux, neurological impairment (aspiration), immunocompromised states, anatomical abnormalities of the respiratory tract, residence in residential care facilities, and hospitalization, especially in an intensive care unit. [3]

Pathophysiology

The lower respiratory tract is normally kept sterile by physiologic defense mechanisms, including mucociliary clearance, the properties of normal secretions such as secretory immunoglobulin A (IgA), and clearing of the airway by coughing. [4]

Immunologic defense mechanisms of the lung that limit invasion by pathogenic organisms include macrophages that are present in alveoli and bronchioles, secretory IgA, and other immunoglobulins. Additional factors that promote pulmonary infection include trauma, anesthesia, and aspiration.[4]

Viral pneumonia usually results from spread of infection along the airways, accompanied by direct injury of the respiratory epithelium, which results in airway obstruction from swelling, abnormal secretions, and cellular debris. [5]

The small caliber of airways in young infants makes such patients particularly susceptible to severe infection. Atelectasis, interstitial edema, and ventilation-perfusion mismatch causing significant hypoxemia often accompany airway obstruction. Viral infection of the respiratory tract can also predispose to secondary bacterial infection by disturbing normal host defense mechanisms, altering secretions, and modifying the bacterial flora. [5]

Bacterial pneumonia most often occurs when respiratory tract organisms colonize the trachea and subsequently gain access to the lungs, but pneumonia may also result from direct seeding of lung tissue after bacteremia When bacterial infection is established in the lung

parenchyma, the pathologic process varies according to the invading organism. M. pneumoniae attaches to the respiratory epithelium, inhibits ciliary action, and leads to cellular destruction and an inflammatory response in the submucosa. [5]

As the infection progresses, sloughed cellular debris, inflammatory cells, and mucus cause airway obstruction, with spread of infection occurring along the bronchial tree, as it does in viral pneumonia. [5]

S. pneumoniae produces local edema that aids in the proliferation of organisms and their spread into adjacent portions of lung, often resulting in the characteristic focal lobar involvement. [5]

Group A streptococcus infection of the lower respiratory tract results in more diffuse infection with interstitial pneumonia. The pathology includes necrosis of tracheobronchial mucosa; formation of large amounts of exudate, edema, and local hemorrhage, with extension into the interalveolar septa; and involvement of lymphatic vessels and the increased likelihood of pleural involvement. [5]

S. aureus pneumonia manifests in confluent bronchopneumonia, which is often unilateral and characterized by the presence of extensive areas of hemorrhagic necrosis and irregular areas of cavitation of the lung parenchyma, resulting in pneumatoceles, empyema, or, at times, bronchopulmonary fistulas. [5]

Clinical Manifestations

Viral and bacterial pneumonias are often preceded by several days of symptoms of an upper respiratory tract infection, typically rhinitis and cough. [6]

In viral pneumonia, fever is usually present; temperatures are generally lower than in bacterial pneumonia. Tachypnea is the most consistent clinical manifestation of pneumonia. Increased work of breathing accompanied by intercostal, subcostal, and suprasternal retractions, nasal flaring, and use of accessory muscles is common. Severe infection may be accompanied by cyanosis and respiratory fatigue, especially in infants. Auscultation of the chest usually reveal crackles and sometime wheezing.[6]

It is often not possible to distinguish viral pneumonia clinically from disease caused by Mycoplasma and other bacterial pathogens. Bacterial pneumonia in adults and older children typically begins suddenly with a shaking chill followed by a high fever, cough, and chest pain. Other symptoms that may be seen include drowsiness with intermittent periods of restlessness; rapid respirations; anxiety; and, occasionally, delirium. Circumoral cyanosis may be observed. In many children, splinting on the affected side to minimize pleuritic pain and improve ventilation is noted; such children may lie on one side with the knees drawn up to the chest.

Physical findings depend on the stage of pneumonia. Early in the course of illness, diminished breath sounds, scattered crackles, and rhonchi are commonly heard over the affected lung field. [6]

With the development of increasing consolidation or complications of pneumonia such as effusion, empyema, and pyopneumothorax, dullness on percussion is noted and breath sounds may be diminished. A lag in respiratory excursion often occurs on the affected side. Abdominal distention may be prominent because of gastric dilation from swallowed air or ileus. Abdominal pain is common in lower lobe pneumonia. The liver may seem enlarged because of downward displacement of the diaphragm secondary to hyperinflation of the lungs or superimposed congestive heart failure. Symptoms described in adults with pneumococcal pneumonia may be noted in older children but are rarely observed in infants and young children, in whom the clinical pattern is considerably more variable. [6]

In infants, there may be a prodrome of upper respiratory tract infection and diminished appetite, leading to the abrupt onset of fever, restlessness, apprehension, and respiratory distress. These infants appear ill, with respiratory distress manifested as grunting; nasal flaring; retractions of the supraclavicular, intercostal, and subcostal areas; tachypnea; tachycardia; air hunger; and often cyanosis. Results of physical examination may be misleading, particularly in young infants, with meager findings disproportionate to the degree of tachypnea. Some infants with bacterial pneumonia may have associated gastrointestinal disturbances characterized by vomiting, anorexia, diarrhea, and abdominal distention secondary to a paralytic ileus. Rapid progression of symptoms is characteristic in the most severe cases of bacterial pneumonia (staph pneumonia). [7]

Mycoplasma pneumonia presented mainly as debilitating cough that may be confused with pertussis, by clinical exam the clinical finding are minimal regarding the severity of symptoms .Unusual etiologies or recurrent pneumonias require special considerations [7]

Differential Diagnosis of Recurrent Pneumonia [8]

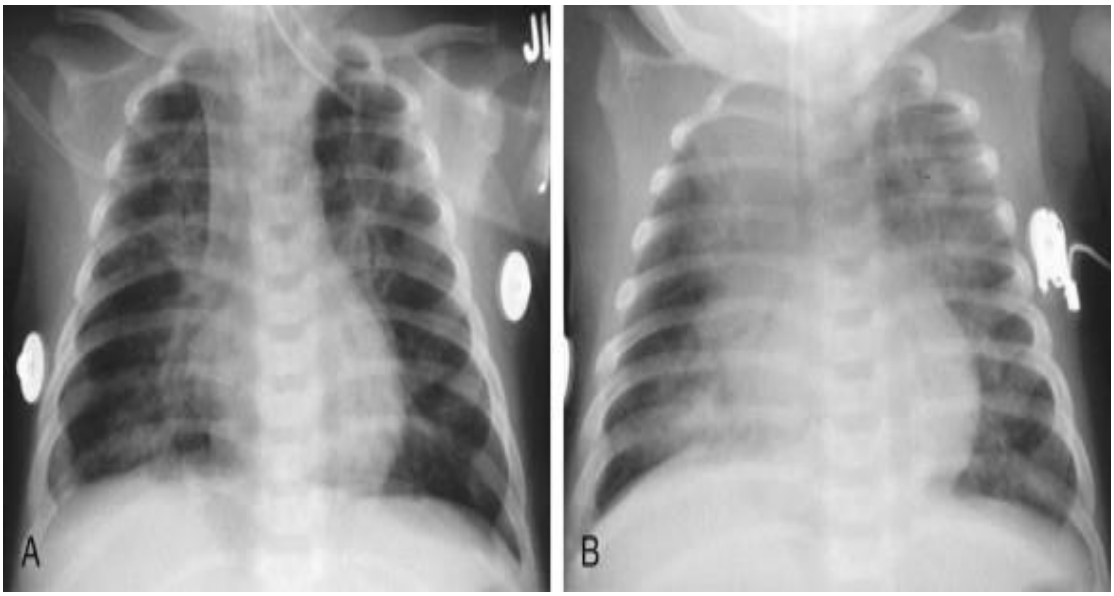
HEREDITARY DISORDERS
Cystic fibrosis
Sickle cell disease
DISORDERS OF IMMUNITY
AIDS
Bruton agammaglobulinemia
Complement deficiency
Selective IgG subclass deficiencies
Common variable immunodeficiency syndrome
Severe combined immunodeficiency syndrome
DISORDERS OF LEUKOCYTES
Chronic granulomatous disease
Hyperimmunoglobulin E syndrome (Job syndrome)
Leukocyte adhesion defect
DISORDERS OF CILIA
Primary ciliary dyskinesia
Kartagener syndrome

ANATOMICAL DISORDERS
Sequestration
Lobar emphysema
Foreign body
Tracheoesophageal fistula (H type)
Congenital pulmonary airway malformation (cystic adenomatoid malformation)
Gastroesophageal reflux
Bronchiectasis
Aspiration (oropharyngeal incoordination)
NONINFECTIOUS MIMICS OF PNEUMONIA
Autoimmune diseases (e.g., granulomatosis with polyangiitis)
Hypersensitivity pneumonitis

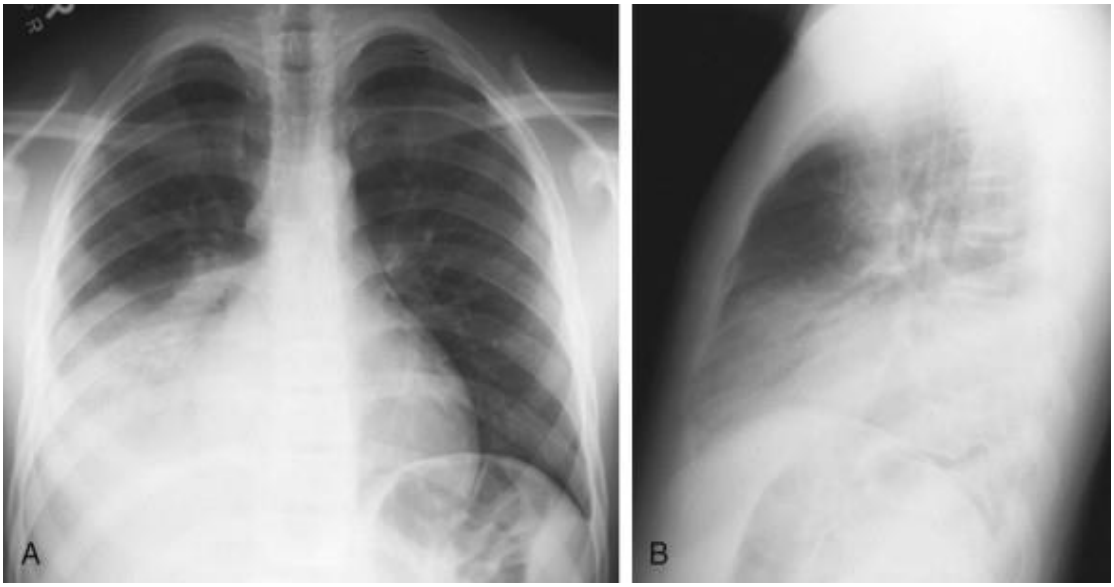
Diagnosis

1- An infiltrate on chest radiograph supports the diagnosis of pneumonia, the film may also indicate a complication such as a pleural effusion or empyema. [9]

Viral pneumonia is usually characterized by hyperinflation with bilateral interstitial infiltrates and peribronchial cuffing [9]



A, Radiographic findings characteristic of respiratory syncytial virus pneumonia in a 6 mo old infant with rapid respirations and fever. Anteroposterior radiograph of the chest shows hyperexpansion of the lungs with bilateral fine air space disease and streaks of density, indicating the presence of both pneumonia and atelectasis. An endotracheal tube is in place. B, One day later, the AP radiograph of the chest shows increased bilateral pneumonia. [9]



Radiographic findings characteristic of pneumococcal pneumonia in a 14 yr old boy with cough and fever. Posteroanterior (A) and lateral (B) chest radiographs reveal consolidation in the right lower lobe, strongly suggesting bacterial pneumonia. [9]

Confluent lobar consolidation is typically seen with pneumococcal pneumonia . The radiographic appearance alone is not diagnostic, and other clinical features must be considered**.** Repeat chest radiographs are not required for proof of cure for patients with uncomplicated pneumonia **.**[9]

2- Point-of-care use of portable or handheld ultrasonography is highly sensitive and specific in diagnosing pneumonia in children by determining lung consolidations and air Broncho grams or effusions. [10]

3- The peripheral white blood cell (WBC) count can be useful in differentiating viral from bacterial pneumonia , In viral pneumonia, the WBC count can be normal or elevated but is usually not higher than 20,000/mm3, with a lymphocyte predominance . Bacterial pneumonia is often associated with an elevated WBC count, in the range of 15,000-40,000/mm3, and a predominance of granulocytes. [10]

Atypical pneumonia due to C. pneumonia or M. pneumonia is difficult to distinguish from pneumococcal pneumonia on the basis of radiographic and laboratory findings.

Pneumococcal pneumonia is associated with a higher WBC count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) level

4- The definitive diagnosis of a viral infection rests on the isolation of a virus or detection of the viral genome or antigen in respiratory tract secretions .Growth of respiratory viruses in conventional viral culture usually requires 5-10 days, although shell vial cultures can reduce this to 2-3 days. (11)

Reliable DNA or RNA tests for the rapid detection of many respiratory pathogens, such as mycoplasma, pertussis, and viruses, including RSV, parainfluenza, influenza, and adenoviruses, are available and accurate. The definitive diagnosis of a bacterial infection requires isolation of an organism. (11)

5- Serologic techniques can also be used to diagnose a recent respiratory viral infection but generally require testing of acute and convalescent serum samples for a rise in antibodies to a specific viral agent. This diagnostic technique is laborious, slow, and not generally clinically useful because the infection usually resolves by the time it is confirmed serologically. Serologic testing may be valuable as an epidemiologic tool to define the incidence and prevalence of the various respiratory viral pathogens. (11)

6- Culture of sputum is of little value in the diagnosis of pneumonia in young children. Blood culture results are positive in only 10% of children with pneumococcal pneumonia. [12]

7- Cold agglutinins at titers >1: 64 are found in the blood in ≈50% of patients with M. pneumonia infections. Cold agglutinin findings are nonspecific because other pathogens such as influenza viruses may also cause increases. [12]

Treatment

Treatment of suspected bacterial pneumonia is based on the presumptive cause and the age and clinical appearance of the child.

For mildly ill children who do not require hospitalization, amoxicillin is recommended. In communities with a high percentage of penicillin-resistant pneumococci, high doses of amoxicillin (80-90 mg/kg/24 hr) should be prescribed. Therapeutic alternatives include cefuroxime axetil and amoxicillin/clavulanate .[13]

For school-aged children and in children in whom infection with M. pneumonia or C. pneumonia is suggested, a macrolide antibiotic such as azithromycin is an appropriate choice. [13]

The empiric treatment of suspected bacterial pneumonia in a hospitalized child requires an approach based on the clinical manifestations at the time of presentation. Parenteral cefotaxime or ceftriaxone is the mainstay of therapy when bacterial pneumonia is suggested. [13]

If clinical features suggest staphylococcal pneumonia (pneumatoceles, empyema), initial antimicrobial therapy should also include vancomycin or clindamycin. [13]

If viral pneumonia is suspected, it is reasonable to withhold antibiotic therapy, especially for those patients who are mildly ill, have clinical evidence suggesting viral infection, and are in no respiratory distress. Up to 30% of patients with known viral infection may have coexisting bacterial pathogens. Therefore, if the decision is made to withhold antibiotic therapy on the basis of presumptive diagnosis of a viral infection, deterioration in clinical status should signal the possibility of superimposed bacterial infection, and antibiotic therapy should be initiated. [13]

In developing countries, oral zinc (20 mg/day) helps accelerate recovery from severe pneumonia. [13]

The optimal duration of antibiotic treatment for pneumonia has not been well-established in controlled studies. For pneumococcal pneumonia, antibiotics should probably be continued until the patient has been afebrile for 72 hours and the total duration should not be less than 10 (or 5 days if azithromycin is used). Available data do not support prolonged courses of treatment for uncomplicated pneumonia. [13]

Antimicrobial Therapy for Pneumonia Caused by Specific Pathogens*

PATHOGEN	RECOMMENDED TREATMENT	ALTERNATIVE TREATMENT
Streptococcus pneumoniae with MIC for penicillin ≤2.0 µg/mL	Ampicillin or penicillin IV; amoxicillin PO	Ceftriaxone, cefotaxime, clindamycin, or vancomycin IV; Cefuroxime, cefpodoxime, levofloxacin,† or linezolid PO
S. pneumoniae with MIC for penicillin ≥4.0 µg/mL	Ceftriaxone IV; levofloxacin† or linezolid PO	Ampicillin, levofloxacin,† clindamycin, or vancomycin IV; clindamycin PO
Group A streptococcus	Penicillin or ampicillin IV; amoxicillin or penicillin PO	Ceftriaxone, cefotaxime, clindamycin, or vancomycin IV; clindamycin PO
Group B streptococcus	Penicillin or ampicillin IV; amoxicillin or penicillin PO	Ceftriaxone, cefotaxime, clindamycin, or vancomycin IV; clindamycin PO
Haemophilus influenzae	Ampicillin IV or amoxicillin PO if β-lactamase negative; ceftriaxone or cefotaxime IV or amoxicillin-clavulanate PO if β-lactamase positive	Ciprofloxacin† or levofloxacin† IV; cefdinir, cefixime, or cefpodoxime PO
Mycoplasma pneumoniae, *Chlamydophila pneumoniae*, or *Chlamydia trachomatis*	Azithromycin IV or PO	Erythromycin or levofloxacin IV; clarithromycin, erythromycin, doxycycline,† or a fluoroquinolone† PO
Staphylococcus aureus, methicillin susceptible (MSSA)	Cefazolin, oxacillin, or nafcillin IV; cephalexin PO	Clindamycin or vancomycin IV; clindamycin PO
S. aureus, methicillin resistant (MRSA)	Clindamycin or vancomycin IV; clindamycin PO	TMP-SMX or Linezolid IV or PO
Gram-negative aerobic bacilli (except *P. aeruginosa*)	Cefotaxime or ceftriaxone with or without an aminoglycoside IV; amoxicillin-clavulanate, cefdinir, or cefixime PO	Piperacillin-tazobactam plus an aminoglycoside‡; fluoroquinolone† PO
P. aeruginosa	Ceftazidime IV with or without an aminoglycoside‡; ciprofloxacin† if susceptible PO	Piperacillin-tazobactam IV with or without an aminoglycoside‡
Herpes simplex virus	Acyclovir IV	

Prognosis

Typically, patients with uncomplicated community-acquired bacterial pneumonial show response to therapy, with improvement in clinical symptoms (fever, cough, tachypnea, chest pain), within 48-96 hr of initiation of antibiotics . Radiographic evidence of improvement lags substantially behind clinical improvement. [14]

A number of factors must be considered when a patient does not improve with appropriate antibiotic therapy: [14]

(1) complications, such as empyema.

(2) Bacterial resistance.

(3) Nonbacterial etiologies such as viruses and aspiration of foreign bodies or food.

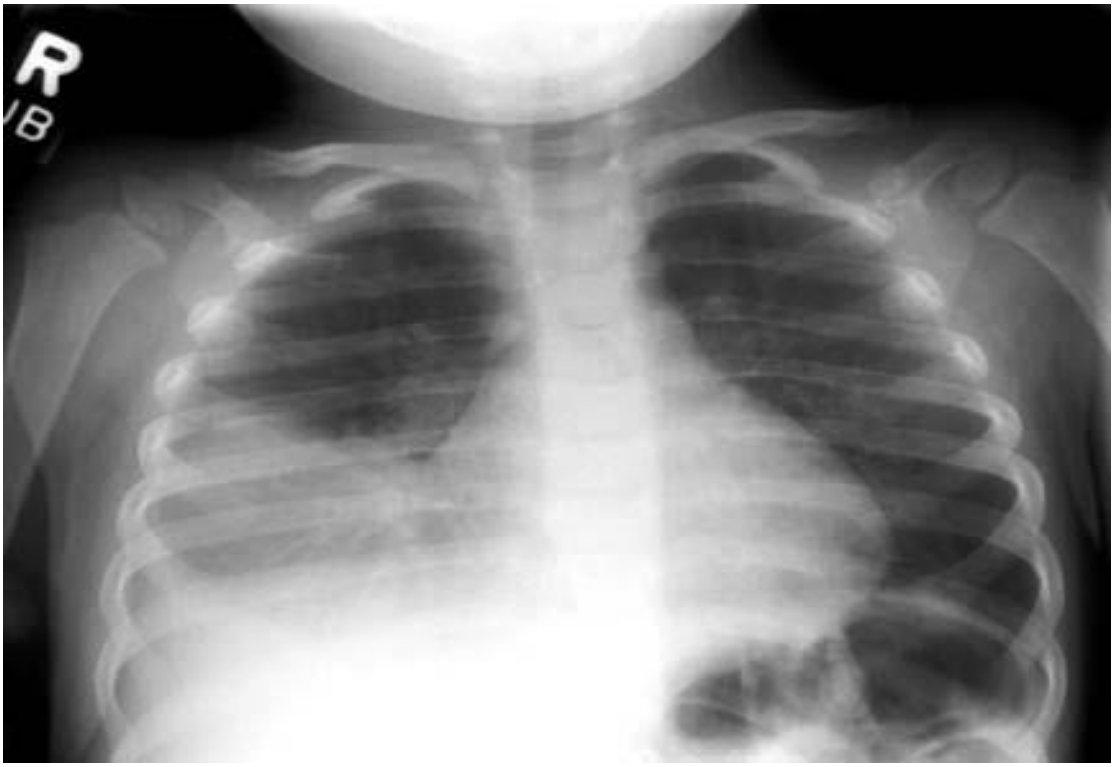
(4) Bronchial obstruction from endobronchial lesions, foreign body, or mucous plugs.

(5) Pre-existing diseases such as immunodeficiencies, ciliary dyskinesia, cystic fibrosis, pulmonary sequestration, or cystic adenomatoid malformation.

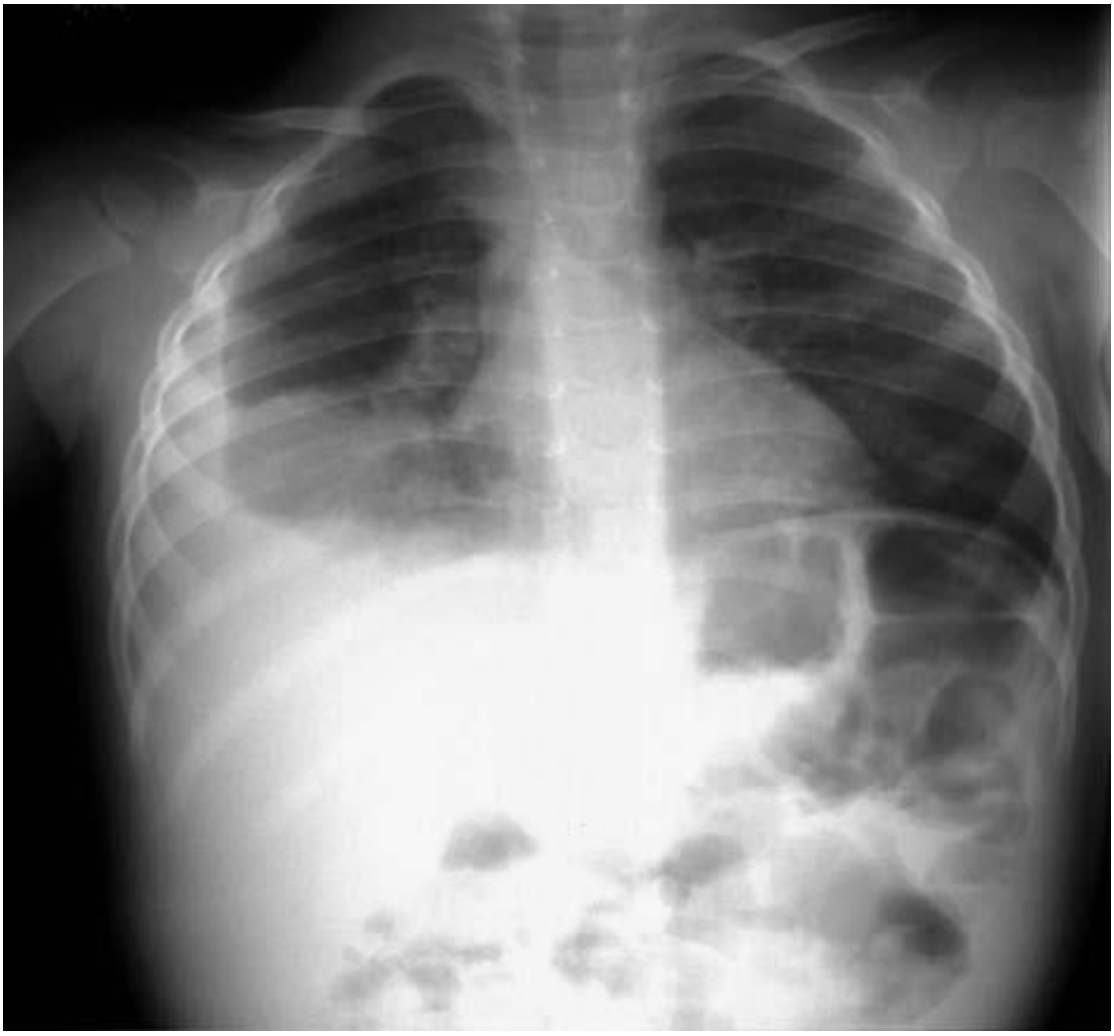
Complications

Complications of pneumonia are usually the result of direct spread of bacterial infection within the thoracic cavity (pleural effusion, empyema, and pericarditis) or bacteremia and hematologic spread. [6]

Meningitis, suppurative arthritis, and osteomyelitis are rare complications of hematologic spread of pneumococcal or H. influenzae type b infection.



Right lower lobe consolidation in a patient with bacterial pneumonia [6]



Radiograph from a patient with bacterial pneumonia (same patient as in the preceding image) a few days later. This radiograph reveals progression of pneumonia into the right middle lobe and the development of a large parapneumonic pleural effusion [6]

Prevention

Annual influenza vaccine is recommended for all children over 6 months of age. Trivalent or quadrivalent, inactivated influenza vaccines are licensed for use beginning at 6 months of age; live-attenuated vaccine can be used for persons 2-49 years of age. Universal childhood vaccination with conjugate vaccines for H. influenzae type b and S. pneumoniae has greatly diminished the incidence of these pneumonias. RSV infections can be prevented by use of palivizumab in some high-risk patients. Reducing the duration of mechanical ventilation and administering antibiotics judiciously reduces the incidence of ventilator-associated pneumonias. [14]

The head of the bed should be raised to 30-45 degrees for intubated patients to minimize risk of aspiration, and all suctioning equipment and saline should be sterile. Hand-washing before and after every patient contact and use of gloves for invasive procedures are important measures to prevent nosocomial transmission of infections. Hospital staff with respiratory illnesses or who are carriers of certain organisms, such as methicillin-resistant S. aureus, should comply with infection control policies to prevent transfer of organisms to patients. Treating sources of aerosols, such as air coolers, can prevent L. pneumonia. [14]

Conclusion

Despite advances in recent years, pediatric pneumonia continues to cause significant morbidity and mortality and poses diagnostic and therapeutic challenges. Vaccination against Haemophilus influenzae type b and Streptococcus pneumoniae has greatly reduced invasive disease rates caused by these pathogens, and the introduction of molecular diagnostics has highlighted the important role that respiratory viruses play in disease pathogenesis while also introducing new challenges. This updated understanding brings into question whether all children with CAP would benefit from antibiotic therapy, and if so, which therapies might be most effective. Limitations of current diagnostics, however, impede advances toward addressing these important questions. Biomarkers and host responses to infection are current areas of intense study that may facilitate a deeper understanding of pneumonia etiology and disease outcomes.

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