Diagnosis and Management of Pneumonia

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DR. ROBERT A. MARLOW (*Assistant Professor of Family Practice*): Today we will discuss a patient with pneumonia. The patient is a 27-year-old woman who developed fever, shaking chills, nonproductive cough, and right-sided pleuritic chest pain two days before her visit to the emergency room. She had some shortness of breath but no nasal congestion. She had vomited once and was not eating well, though she was taking some fluids. She had a previous episode of pneumonia when she was 13 years old.

She had a history of Down's syndrome with mental retardation, seizure disorder, and scoliosis treated with a Harrington rod. Her medications include phenytoin and oral contraceptives. She has no known allergies and does not smoke or use alcohol.

Family history was significant only in that the patient's mother had carcinoma of the breast. The patient lives in a foster group home with five other adults and a full-time caretaker, none of whom are ill. She had not traveled recently.

On examination, she looked ill and was grasping the right lower chest. Her temperature was 102.1 °F, pulse 112/min, respirations 24/min, and blood pressure 112/68 mmHg. Her head, eyes, ears, nose, and throat were normal. Her neck was supple without adenopathy, and her back showed marked scoliosis with an axial surgical scar. Her chest had a very small anteroposterior diameter. Her right anterior chest wall was very tender, and she had rales and decreased breath sounds at the right base. She had tachycardia, but no murmur or rub. Her abdomen was normal except for mild diffuse tenderness without rebound. Her extremities and skin were normal. She was mildly mentally retarded and had bilateral positive Babinski reflexes, but otherwise her neurologic examination was normal.

Laboratory test results included the following: hematocrit 0.45 (45 percent); white blood cell count 21.4×10^9/L ($21.4 \times 10^3/\mu L$) with 0.58 segmented neutrophils (58 percent), 0.22 band cells (22 percent), and 0.16 lymphocytes (16 percent) with toxic granulations noted; sodium 136 mmol/L (136 mEq/L); potassium 3.8 mmol/L (3.8 mEq/L); chloride 102 mmol/L (102 mEq/L); carbon dioxide content 24 mmol/L (24 mEq/L); blood urea nitrogen 3.6 mmol/L (10 mg/dL); creatinine 110 μmol/L (1.2 mg/dL); urinalysis was normal except for a specific gravity of 1.030. A chest x-ray film was ordered and Dr. Dixon will present those films.

DR. RAYMOND DIXON (*Radiologist in private practice*): The chest x-ray film shows a flocculent, nonsegmental, nonlobar infiltrate that involves many portions of the right lower lobe, certainly thought to be a pneumonia.

DR. MARLOW: I think you can also appreciate from the chest x-ray film the abnormality of the thorax, with a small anteroposterior diameter and scoliosis.

The patient could not produce sputum, so a Gram stain and culture were not done. Blood for two blood cultures and cold agglutinins was drawn.

Arterial blood gases were obtained on room air with the following results: pH 7.44 units, oxygen (pO_2) 5.2 kPa (39 mmHg), and carbon dioxide (pCO_2) 5.2 kPa (39 mmHg). Repeat blood gases on two liters per minute of oxygen revealed the following: pH 7.39 units, pO_2 7.5 kPa (56 mmHg), and pCO_2 5.6 kPa (42 mmHg).

Given this presentation in the emergency room, how would you initially manage this patient?

HOSPITAL COURSE

DR. RICHARD RATHE (*Second-year Family Practice Resident*): At this point, my management would be for a patient with a community-acquired pneumonia and no risk factors except for scoliosis. I would admit her to the hospital and probably put her on intravenous erythromycin. If she seemed really ill, I would probably put her on intravenous ampicillin and an aminoglycoside.

DR. MARLOW: The patient was placed on intravenous fluids for her dehydration. She was felt presumptively to have pneumococcal pneumonia and given intravenous penicillin. Ultrasonic mist inhalations were used, but she

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could not tolerate percussion or postural drainage. She was continued on her normal dose of phenytoin.

By the morning of the second hospital day, she was afebrile but otherwise clinically the same. Laboratory results of blood cultures were negative for growth at 24 hours, and cold agglutinin test results were not significant with a titer of only 1:4. Her blood urea nitrogen was 1.8 mmol/L (5 mg/dL) and her creatinine was 80 μmol/L (0.9 mg/dL). That afternoon, her temperature spiked to 102.0 °F, and a physician was called to see her. What would you do at this point?

DR. CATHERINE SCHELL (*Third-year Family Practice Resident*): I might consider switching her to erythromycin to cover for the possibility of Mycoplasma infection.

DR. RATHE: Especially since she is not producing any sputum.

DR. MARLOW: The decision was made to stop the penicillin and begin erythromycin, 500 mg intravenously, every six hours.

By the third hospital day, she was afebrile and slightly improved on physical examination. Her blood culture results were still negative. On the fourth hospital day, her temperature went back to 102.6 °F, and there was no change in her pain or her physical examination findings. Two more blood cultures were ordered, and a repeat white cell count was 16.1×10^9/L ($16.1 \times 10^3/\mu L$) with 0.60 segmented neutrophils, 0.14 band cells, and 0.12 lymphocytes. Chest x-ray film was repeated.

DR. DIXON: At this point a portion of the silhouette of the right hemi-diaphragm is lost by contiguous opacities, sometimes called the "silhouette sign." It should be called "loss-of-silhouette sign," as it means contiguity of two opacities. There has appeared a pleural effusion. Her lung remains clear elsewhere. Her other lung remains clear. An ipsilateral decubitus view indicates that the fluid is layering, thereby proving that it is free fluid.

DR. MARLOW: If you were called to see this patient at this point, what would you do?

DR. SCHELL: I would do skin testing for tuberculosis. I would also consider tapping the fluid in her chest.

DR. MARLOW: A thoracentesis was done, revealing the following results on the pleural fluid: pH 7.5; glucose 7.5 mmol/L (136 mg/dL); protein 0.03 g/L (2.7 mg/dL); white cell count 0.7×10^9/L ($0.7 \times 10^3/\mu L$) with 0.89 polymorphonuclear leukocytes and 0.11 lymphocytes, and red blood cell count 10.2×10^9/L ($10.2 \times 10^3/\mu L$); Gram stain showed moderate white blood cells but no bacteria. The fluid was cultured for aerobic and anaerobic bacteria. A chest x-ray film was obtained after thoracentesis.

DR. DIXON: The chest x-ray film demonstrates a small pneumothorax on the right.

DR. MARLOW: The patient was continued on the erythromycin, and intravenous ceftriaxone was added at

1 g every 12 hours. The patient's temperature was 99.4 °F on the fifth day of hospitalization. Her examination was essentially the same. Results of cold agglutinins remained unchanged at a titer of 1:4.

On the sixth hospital day, all culture results were negative, including four blood cultures and the pleural fluid cultures. Her temperature remained 99.1 °F to 99.8 °F over the next four days with some decrease in her pain. Examination of the chest showed less dullness on the right. Skin testing for tuberculosis was negative. An erythrocyte sedimentation rate was 50 mm/h. Another chest x-ray film was obtained.

DR. DIXON: A moderate-sized pneumothorax with fluid is present on the right. Of interest is differential collapse of the three lobes, which used to be important in tuberculosis. One can identify the upper lobe, the minor fissure, the middle lobe, and the lower lobe. The lower lobe collapse is quantitatively the greatest, and possibly relates to the fact that the lobe is abnormal—it is infiltrated.

DR. MARLOW: On the tenth hospital day, a surgeon was consulted and a right thoracostomy with chest tube drainage was performed. Pleural fluid obtained was sent for the following tests: aerobic bacteria culture, anaerobic bacteria culture, tuberculosis culture, and tuberculosis smear. The result of the smear for tuberculosis was negative. A chest x-ray film was ordered after the chest tube was placed.

DR. DIXON: As you can see on the film, the chest tube is in place. The lung is expanded, but there is still a modest amount of fluid.

DR. MARLOW: On the 12th hospital day, the patient was afebrile and she was improving clinically. All culture results were negative. The patient was switched from intravenous to oral erythromycin. On the 13th hospital day, the chest tube was removed, and the patient was scheduled for a follow-up chest x-ray examination.

DR. DIXON: The film, which is rotated somewhat, shows air in the axilla, not in the lung at all. There is a small pneumothorax over the apex of the right lung.

DR. MARLOW: The patient remained afebrile for the rest of the hospital course. On day 16 of hospitalization, the white cell count was down to 7.7×10^9/L (7.7×10^3/μL) with 0.45 segmented neutrophils, 0.03 band cells, and 0.32 lymphocytes. Ear oximetry showed she still needed some oxygen. On the 18th hospital day, the chest x-ray examination was repeated.

DR. DIXON: The final film shows a normal-appearing chest except for the baseline problems—full expansion and clearing of the infiltrate.

DR. MARLOW: Antibiotics were discontinued, and the patient was discharged home on the 21st hospital day on her usual medications. Her oxygen requirement had resolved. All culture results were negative on final report.

This case illustrates the difficulty of deciding how to manage a patient with pneumonia when a specific organism cannot be identified. I would like to discuss the diagnosis and empiric management of pneumonia. Pneumonia means inflammation in the lung parenchyma. The term *pneumonia* most commonly refers to an acute infection in the lung,[1] which is the leading cause of death from infectious disease in the United States.[2]

DIAGNOSIS OF PNEUMONIA

When evaluating a patient with an acute illness and a new pulmonary infiltrate, one must determine whether the disease is due to infection. Other noninfectious causes of parenchymal lung lesions can mimic or coexist with pneumonia, such as pulmonary embolus, congestive heart failure, carcinoma, uremia, and sarcoidosis.[3] It is not always easy to differentiate these lesions from pneumonia.

In obtaining the history from the patient, it is especially important to know the age of the patient and where the patient was located at the onset of the infection (community or hospital). Other important factors are the mode of onset (sudden or gradual), fever, chills, sputum production, and chest pain. The development of pneumonia from a bacterial infection often signals compromised pulmonary host defenses from other illnesses, so past medical history is important. Such things as smoking, alcohol abuse, and drug abuse are important. A history of recent family illnesses might be helpful. History of the patient's employment, travel, and sexual habits (especially if the patient is homosexual or a prostitute) are important.

Several findings on physical examination can be important. How sick or "septic" the patient appears is helpful. The temperature, pulse, and respiratory rate are important. Obviously, when examining the chest, looking for rales, dullness, or a pleural friction rub is necessary. Other signs on physical examination may also give helpful information.[3] When examining the skin, cyanosis would indicate hypoxia, petechiae might indicate bacteremia or endocarditis, and a maculopapular rash could indicate adenovirus or echovirus. On head, ears, eyes, nose, and throat examination, bullous myringitis is sometimes associated with infection by Mycoplasma pneumoniae, and conjunctivitis or nasopharyngitis could indicate adenovirus. A murmur or pericardial rub could indicate endocarditis or pericarditis, respectively. Upper abdominal tenderness is common in bacterial pneumonia, and gastric distention and ileus are common in severe bacterial pneumonia. Jaundice has been associated with severe pneumonococcal sepsis or Q fever. A stiff neck might indicate meningitis (Streptococcus pneumoniae or Hemophilus influenzae). Other neurologic findings are usually from an associated disorder.

TABLE 1. RELATIONSHIP BETWEEN CAUSATIVE AGENT AND RADIOGRAPHIC PATTERNS OF PNEUMONIA

Agent	Radiographic Pattern
Streptococcus pneumoniae	Localized or lobar consolidations; pleural effusion common
Mycoplasma pneumoniae	Lower lobes, often bilateral; pleural effusion in 15%
Staphylococcus aureus	Central patches of consolidation; multiple abscess formation; pleural effusion common
Klebsiella	Upper lobes with "bulging fissures," or nonspecific localized consolidation
Pseudomonas	Microabscesses and/or localized consolidations
Viral (adult)	Interstitial, often bilateral

Although very high white cell counts, greater than 12.0 $\times$ 10^9/L (12.0 $\times$ 10^3/μL), are indicative of bacterial infection, a normal white cell count does not rule out bacterial infection.[4] Mycoplasma pneumonia can cause a white cell count from normal to as high as 20.0 $\times$ 10^9/L (20.0 $\times$ 10^3/μL).[4]

A chest x-ray examination is indicated if there are clinical signs and symptoms of systemic illness with a lower respiratory tract focus.[5] A chest x-ray examination is probably not indicated if the patient is young or middle-aged, has no risk factors, is not coughing up sputum, does not appear to be septic, and does not have significant chest signs or symptoms.[5] The radiographic patterns of pneumonia are almost never diagnostic,[4] but they might help. The relationship between causative agent and radiographic patterns of pneumonia is shown in Table 1.[4]

The diagnosis of pneumonia is not made from results of the sputum culture, but the causative organism of a known pneumonia can sometimes be confirmed by analysis of the sputum.[6] Ideally a sputum specimen should reflect the organism in the lung; therefore, Gram stain of the sputum should be part of the initial evaluation of any patient with pneumonia.[4] As any clinician knows, however, there are problems with obtaining sputum. Many patients with fever are dehydrated and cannot produce adequate sputum.[4] Obtunded patients cannot voluntarily produce sputum.[4] One might obtain oral secretions rather than sputum. Analysis of oral secretions adds nothing and may in fact confuse the physician. Some rules of thumb have been addressed, such as needing to see greater than 25 polymorphonuclear leukocytes and fewer than 10 epithelial cells per oil-immersion field.[3] In a study at the Mayo Clinic, only 25 percent of the sputum specimens submitted to the laboratory met these minimum criteria.[7]

There are several methods of obtaining sputum. If a patient is able, he or she should actively attempt to cough up sputum. If rhonchi are present, but the patient is too weak to bring up sputum, consider nasotracheal suction with a trap.[3] There are some clinical situations for which transtracheal aspiration is indicated: an immunocompromised patient with pneumonia but no sputum, a patient being treated without good response, a patient with nosocomial pneumonia in which an unusual organism is possible.[4] One must consider whether the risk of empiric treatment outweighs the risk of transtracheal aspiration. Some situations might also dictate sputum be obtained at the time of bronchoscopy.

If sputum is obtained, at least perform a Gram stain on the sputum. If there is a predominate organism, it could guide you in the initial selection of antibiotic. Gram stain may well be more helpful than sputum culture, for Gram stain shows what organisms are present, whereas the sputum culture tends to show which organisms grow the fastest.

Blood specimens should be cultured prior to antibiotic therapy on all patients who are hospitalized with pneumonia. All patients hospitalized with pneumonia should have arterial blood gas determinations. Pleural effusions in association with pneumonia should be tapped for Gram stain and culture.

MANAGEMENT OF PNEUMONIA

A person with pneumonia should be considered for hospitalization when the following factors are present: the chest x-ray results show a significant infiltrate, the patient looks markedly ill, the patient is elderly, the patient is dehydrated, the patient complies poorly with oral medications, or the patient has no one at home to help with care.[5]

In selecting initial antibiotic therapy for pneumonia, it is useful to consider various pneumonia syndromes. The first group of syndromes comprises the community-acquired pneumonias. In a patient without risk factors, over 90 percent of community-acquired pneumonias are due to the following organisms: Mycoplasma pneumoniae, virus, Streptococcus pneumoniae, and in some locations Legionella.[1,2,4,5,8] It may be possible clinically to separate Streptococcus pneumoniae from Mycoplasma pneumoniae or virus. Factors associated with these two pneumonia syndromes are displayed in Table 2.[3,4,8]

Clinically, if one is certain that a patient has a Streptococcus pneumoniae infection, penicillin is the drug of choice,[3–6] either orally for outpatients or intravenously for inpatients. If one is not sure that a patient has Streptococcus pneumoniae, then erythromycin should be used.[3–6] In many pneumonias caused by Mycoplasma, results are negative for cold agglutinins, complement fix-

TABLE 2. FACTORS ASSOCIATED WITH PNEUMONIA CAUSED BY VIRUS OR MYCOPLASMA PNEUMONIAE VS STREPTOCOCCUS PNEUMONIAE

Factor	Virus or Mycoplasma pneumoniae	Streptococcus pneumoniae
Age	Younger	Elderly
Onset	Gradual	Sudden
Chills	Uncommon	Common
Fever	Low grade	High
Tachycardia (>120/min)	Rare	Common
Tachypnea (>30/min)	Rare	Common
Chest pain	Uncommon	Common
White cell count elevated	Uncommon	Common
Chest x-ray lobar or segmental	Rare	Common
Pleural effusion	Rare	Common
Sputum	Initially scant	Abundant
Sputum Gram stain	Rare polymorphonuclear leukocytes, no organisms	Many polymorphonuclear leukocytes, gram-positive diplococci

ation titers are normal during the first week of the illness, and white cell counts cannot reliably differentiate pneumonia caused by virus from that caused by Mycoplasma.[4]

In a patient who is a smoker or who has chronic lung disease, community-acquired pneumonias are commonly due to the following organisms: Streptococcus pneumoniae, Hemophilus influenzae, Staphylococcus aureus, Branhamella catarrhalis, and in some locations Legionella.[1,2,4,5,8,9] If a patient is treated as an outpatient, then amoxicillin-clavulanic acid would be a good choice for antibiotic, as many of the above organisms are β-lactamase producers. Another choice of antibiotic might be cefaclor. For inpatients who have chronic lung disease or who are smokers, base initial antibiotic therapy on the Gram stain (Table 3). If the presumptive organism is Streptococcus pneumoniae, use penicillin.[10,11] When Hemophilus influenzae is suspected, use a third-generation cephalosporin, trimethoprim-sulfamethoxazole, or chloramphenicol.[10,11] For presumed Staphylococcus aureus, use a first-generation cephalosporin or a penicillinase-resistant penicillin such as oxacillin; if the organisms are methicillin resistant, then use vancomycin.[10,11] For presumed Branhamella catarrhalis, use a second- or third-generation cephalosporin, trimethoprim-sulfamethoxazole, or chloramphenicol.[11] If Legionella is suspected, use erythromycin.[10,11]

In patients with community-acquired pneumonia who have a history of alcohol or drug abuse, the pneumonias are commonly caused by the following organisms: Streptococcus pneumoniae, Hemophilus influenzae, Staphylococcus aureus, and Klebsiella pneumoniae.[1,4,5,9] For outpatient treatment, amoxicillin-clavulanic acid or cefaclor would be reasonable choices. Initial inpatient treatment should be based on the Gram stain (Table 3). If Klebsiella is the presumed organism, then use a second- or third-generation cephalosporin; some authors would add an aminoglycoside.[10,11]

TABLE 3. ANTIBIOTIC CHOICE FOR SPECIFIC ORGANISMS

Organism	Antibiotic
Streptococcus pneumoniae	Penicillin
Hemophilus influenzae	Third-generation cephalosporin, trimethoprim-sulfamethoxazole, chloramphenicol
Staphylococcus aureus	First-generation cephalosporin, penicillinase-resistant penicillin (vancomycin if methicillin-resistant)
Branhamella catarrhalis	Second- or third-generation cephalosporin, trimethoprim-sulfamethoxazole, chloramphenicol
Legionella	Erythromycin
Klebsiella	Second- or third-generation cephalosporin (might add an aminoglycoside)

If a patient develops pneumonia following an influenza infection, the pneumonia is commonly due to the following organisms: Staphylococcus aureus, Hemophilus influenzae, or Streptococcus pneumoniae.[1,2,5,9] For outpatient treatment, amoxicillin-clavulanic acid or cefaclor would be good choices. If the patient is treated as an inpatient, then Gram stain should guide the initial choice of antibiotic (Table 3).

In a patient from the community who probably aspirated gastric contents or who has altered consciousness, pneumonia is commonly due to the following organisms: anaerobes, gram-negative rods, or Staphylococcus aureus.[1,4,9,11] These patients are usually treated as inpatients.[4,11] The drugs of choice would be penicillin, clindamycin, or cefoxitin.[4,11]

An elderly patient with a community-acquired pneumonia commonly is infected with one of the following organisms: Streptococcus pneumoniae, Hemophilus influenzae, gram-negative rods, Staphylococcus aureus, and in some locations Legionella.[10] These infections are generally treated on an inpatient basis with the Gram stain as a guide for the initial selection of antibiotic (Table 3). If the organism is presumably a gram-negative rod, use a second- or third-generation cephalosporin plus an aminoglycoside.[9]

A diabetic who develops a community-acquired pneumonia commonly is infected with one of the following organisms: Streptococcus pneumoniae, Staphylococcus aureus, or Klebsiella pneumoniae.[9] Generally, these pa-

tients are treated in the hospital with Gram-stain results indicating the initial choice of antibiotic (Table 3).

There are some less common special situations to consider in a patient with a community-acquired pneumonia. If the patient has been around cattle or sheep, consider Q fever (treated with tetracycline) or brucellosis (treated with tetracycline plus streptomycin).[1,11] When the patient has an association with parrots or other birds, consider psittacosis (treated with tetracycline).[1,11] If the patient has been in a desert environment, particularly the desert Southwest, consider coccidioidomycosis (treated with amphotericin B or maybe ketoconazole).[7,10] When the patient has been around chickens or caves, consider histoplasmosis (treated with amphotericin B or maybe ketoconazole).[7,10]

Should a patient present with a hospital-acquired pneumonia, the possible organisms involved change when compared with community-acquired pneumonia. The physician should be familiar with the organisms that are in his or her particular hospital.[11] Considering the variations from hospital to hospital, hospital-acquired pneumonias are commonly due to the following organisms: gram-negative rods including Pseudomonas, Staphylococcus aureus, anaerobes, and in some locations Legionella.[1,8,11] These patients are treated in the hospital, and obtaining sputum for Gram stain is especially important (Table 3). If the presumed organism is a gram-negative rod, treat with a third-generation cephalosporin plus an aminoglycoside.[10,11] When Pseudomonas is suspected, then use an antipseudomonal penicillin (piperacillin, ticarcillin, mezlocillin, azlocillin) plus an aminoglycoside.[11] If one suspects Staphylococcus aureus as well as gram-negative rods, use a first-generation cephalosporin plus an aminoglycoside[11] or imipenemcilastatin.

Should the patient be immunocompromised and develop pneumonia, then being able to identify the organism and treat specifically becomes even more important. One would do the usual workup of pneumonia as indicated above. In addition, some type of lung biopsy is usually indicated because too many of the possible organisms will not grow on sputum culture.[12] The specific type of immunodeficiency might give some indication of the possible organism. Decreased immunoglobulins are associated with infections caused by gram-positive cocci. Decreased granulocytes, fewer than 1.0×10^9/L ($1.0 \times 10^3/\mu$L), are associated with infections caused by gram-negative rods (including Pseudomonas) and Staphylococcus aureus. Decreased cell-mediated immunity, such as in acquired immune deficiency syndrome (AIDS), is associated with infections caused by Pneumocystis, Mycobacterium, fungus, virus, and Nocardia.

There are six possibilities to consider when the patient is not getting better in spite of antibiotic treatment.[3] First, the diagnosis may be wrong—the patient may not have pneumonia. Second, there may be undrained pus, such as an empyema or a lung abscess. Third, the patient may have developed a superinfection, which should be suspected if there is an initial response to antibiotic, followed three to five days later by more fever, a higher white cell count, more purulent sputum, and a new or worsening infiltrate on chest x-ray examination. Fourth, the fever may be drug-induced, which should be suspected if clinically the patient is improving out of proportion to the fever. When an equally effective antibiotic is substituted, the patient's fever should abate in 24 to 48 hours. Fifth, the patient may have compromised host defenses. Usually not much can be done in this case, but an attempt should be made to correct the underlying disease, if possible. Finally, the antibiotic used may not be appropriate. It is important to identify the infecting organism, if possible.

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