Imaging of Pulmonary Infections

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Introduction

Pneumonia is one of the most frequent causes of morbidity and mortality throughout the world. It is the most prevalent community-acquired infection and the second most common nosocomial infectious disorder. Infections may occur in healthy people or individuals with concomitant intrapulmonary or extrathoracic diseases. Pneumonia may develop into a life-threatening condition, especially in immunocompromised patients, in children, and in the elderly.

In this contribution, the principles regarding epidemiology, pathogenesis, classification, and clinical and radiographic diagnoses of pneumonias are reviewed. Specific effort is directed toward formulating an integrated approach to the diagnosis of pneumonia that combines clinical and radiologic information. The course material focuses on: (1) community-acquired pneumonia (CAP); (2) nosocomial pneumonia (NP); and (3) pneumonia in immunocompromised patients.

Although the spectrum of organisms causing penumonia differs between these groups of patients, and there is considerable overlap with regard to their radiologic features, there may be findings on chest imaging that help to narrow the differential diagnosis.

Community-Acquired Pneumonia

Pathogenesis

CAPs are acquired in the community and most often seen in the offices of general practitioners, private radiologists, and in the outpatient department or the emergency room of the hospitals. CAP primarily affects children (15-35 of 1,000 children per year) and elderly people (30-40 per 1,000 persons per year). The mode of transmission of CAP is usually person-to-person via water or mucus droplets laden with viruses or bacteria. The most frequent pathogens are Gram-positive bacteria, such as *Streptococcus pneumoniae* (*Pneumococcus*) and *Staphylococcus aureus,* and Gram-negative bacteria, such as *Haemophilus influenzae* and atypical bacteria, including *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Legionella pneumophila*. In addition, respiratory viruses, such as the influenza viruses, the human metapneumovirus (hMPV), the respiratory syncytial virus (RSV), the rhinovirus, parainfluenza viruses, adenoviruses, and corona viruses may also cause CAP. It appears from the literature that the spectrum of organisms varies according to patient demographics, temporal, geographic, and diagnostic factors.

In CAP, the patient's health and socioeconomic status have a certain impact on the spectrum of causative organisms. Normally healthy people are most likely to contract mycoplasma pneumonia or a mild form of pneumococcal pneumonia. Debilitated patients, alcoholics, and chronically ill people, however, more often present with severe pneumococcal pneumonia or infections caused by *H. influenzae*, *S. aureus*, or Gram-negative bacilli. *L. pneumophila* and *Chlamydia* infections are more common in patients with some form of mild immunologic compromise. Patients with poor oral hygiene and occasional loss of consciousness (epilepsy, alcoholism) may suffer from anaerobic pulmonary infections. In addition, in these patients, *Mycobacterium tuberculosis* infection is more prevalent compared with in healthy persons without risk factors. Recurrent pneumonia in outpatients usually indicates some sort of underlying problem, such as congenital or acquired immunologic disorder; airway abnormalities, such as chronic bronchitis, bronchiectasis, and bronchogenic carcinoma; cardiac problems (congestive heart failure); or systemic diseases, such as diabetes, chronic alcoholism, and intravenous drug abuse. Up to 10% of CAP are aspiration pneumonias caused by aspiration of colonized oropharyngeal or gastric contents. The most frequently isolated pathogens in aspiration pneumonia are Gram-negative bacteria. Aspiration pneumonia must be differentiated from aspiration pneumonitis, which is a chemical pneumonitis that results from the aspiration of noncolonized gastric contents (Mendelson's syndrome).

The definition of CAP has been challenged over the last few years, as the definition of CAP also includes patients from nursing homes, rehabilitation hospitals, and outpatient-based surgical centers who routinely receive

invasive medical treatment. As a consequence, the bacteriology and outcome of these patients is more similar to those of NPs. Therefore, it has been proposed that pneumonia in outpatients who were hospitalized for more than 2 days over the previous 3 months or who reside in nursing homes or extended-care facilities should be categorized as health-care-associated pneumonia (HCAP).

Clinical Diagnosis

Patients suffering from CAP usually present with fever, cough, dyspnea, sputum production, and pleuritic chest pain, as well as laboratory signs, such as leucocytosis. Because the clinical symptoms are nonspecific (most people who have fever and cough do not have pneumonia), the chest radiograph is one of the most important tools in the diagnosis of CAP. The radiographic identification of pulmonary opacities is, in the appropriate clinical setting, indicative of pneumonia. Conversely, a patient who has fever and cough but does not have radiographic evidence of pneumonia can be considered to be free of pneumonia. In CAP, the causative organism is frequently not identified, because noninvasive tests such as sputum cultures correctly identify the offending organism in only 50% of cases, and invasive procedures are rarely used in these patients.

Radiographic Diagnosis

In patients with CAP, the primary role of the radiologist is to detect or to exclude pneumonia. A second task for the radiologist is to aid the clinician in determining the etiologic diagnosis. Categorization of the causative organism is sometimes possible by integrating clinical and laboratory information with radiographic pattern recognition. A specific etiologic diagnosis, however, is difficult to establish due to the increasing spectrum of causative organisms and their overlapping radiographic features. In a prospective study of 359 adults with CAP, Fang and coauthors compared the radiographic, clinical, and laboratory features of patients with bacterial pneumonia (caused by *H. influenzae*, *S. pneumoniae*, *S. aureus*, and aerobic Gram-negative bacilli) with features from patients with atypical pneumonia (caused by *M. pneumoniae* and *Chlamydia* spp.). The authors found no features that could reliably differentiate these groups. Another group prospectively compared the clinical and radiologic features of CAP caused by *L. pneumophila* to those of patients with pneumococcal infections. The authors concluded that *Legionella* infection clinically as well as radiologically may look like a typical bacterial pneumonia.

The chest radiograph is the first-line tool in evaluating patients with suspected CAP. Computed tomography (CT) is reserved for assessing complications or for guiding further diagnostic procedures. It is definitely indicated in investigating patients with recurrent or persistent pulmonary opacifications.

Nosocomial Pneumonia

NPs develop in a hospital environment. The incidence of NP ranges from 0.5 to 5 cases per 100 admissions, but, in the subgroup of ventilated patients in an intensive care setting, may reach 7-41%. Mortality rates reported for hospital-acquired pneumonia range from 20% in multi-hospital studies to 50% or higher in single referral centers and university hospitals. Apparently, mortality is related to the causative agent, such that the prognosis associated with aerobic Gram-negative pneumonias is considerably worse than that associated with Gram-positive or viral agents.

Pathogenesis

NP develops from bacterial colonization of the oropharynx and aspiration of oropharyngeal secretions and gastrointestinal contents into the lungs. In addition, the microbial contamination of inserted tubes, lines, and catheters is an important pathogenetic factor. Less commonly, NP is the result of bacteremia originating from right-sided endocarditis or septic pelvic thrombophlebitis. Risk factors are either patient-related (underlying illness, previous surgery, prolonged hospital care) or iatrogenic (intravascular catheters, tracheal tubes, indwelling catheters, respirator equipment). Sources of infections are hospital personnel and patients with active infections. Most notably, the inappropriate use of broad-spectrum and prophylactic antibiotics is an additional and important factor leading to an increased susceptibility to hospital-acquired pneumonia. The majority of NPs are caused by Gram-negative bacilli, including *Pseudomonas aeruginosa*, *Klebsiella* spp., *Enterobacteriaceae* spp., *Escherichia coli*, *Serratia marcescens*, and *Proteus.* In addition, Gram-positive cocci, atypical bacteria such as *L. pneumophila*, and viruses such as the respiratory syncytial virus, may play a role.

Clinical Diagnosis

Compared with CAP, it may be difficult for the clinician to diagnose pneumonia in a hospitalized patient. The classical findings for pneumonia, such as new fever, new pulmonary opacification on chest radiographs, cough, sputum production, and elevated leucocyte count may not be present in the hospitalized patient with NP. If present, these symptoms may not necessarily be caused by pneumonia. Microbiologic evaluation of the patient with suspected NP (sputum, bronchoalveolar lavage) may or may not be helpful because of the difficulties in differentiating contamination from true infection. In addition, pulmonary disease in a hospital environment may be caused by more than one agent. Therefore, identifying a pulmonary infection, using various methods to obtain a specimen, and the value of isolating potential pathogens are matters of constant discussion in the clinical diagnosis of NP.

Radiographic Diagnosis

Because of the potential difficulties in the clinical diagnosis of pneumonia in the hospitalized patient, the radiologist has an important role in detecting and classifying suspected pneumonia. However, the radiographic diagnosis of a pulmonary opacity in suspected NP is not as straightforward as it is in patients with CAP. The radiographic diagnosis of a pneumonia may be hampered by preexisting disorders or concomitant lung disease, such as fibrosing alveolitis, lupus pneumonitis, hemorrhage or contusion, acquired respiratory distress syndrome (ARDS), tumor, atelectasis, and embolic infarcts. These disorders may obscure or alter the otherwise characteristic radiographic appearance of a pulmonary opacification and also render the etiologic approach using pattern recognition difficult.

The difficulties in diagnosing NP are nicely demonstrated in two studies. Winer-Muram et al. assessed the diagnostic accuracy of bedside chest radiography for pneumonia in ARDS patients. The overall diagnostic accuracy in these patients was only 42% because of false-negative and false-positive results originating from diffuse parenchymal areas of increased opacity that obscured the radiographic features of pneumonia. Wunderink et al. compared the premortem chest radiographic findings with pulmonary autopsy studies in ventilated patients with NP. No radiographic findings had a diagnostic efficiency greater than 68%. The only radiographic abnormality that correlated with pneumonia, correctly predicting 60% of pneumonias, was the presence of an air bronchogram.

CT is used more often when an NP is suspected than in patients with CAP to detect early morphologic signs of infection (for example, ground-glass densities). CT can also identify a pulmonary opacification in areas of preexistent disease, detect complications such an empyema, and guide invasive diagnostic procedures, such as bronchoscopy or percutaneous biopsy, or thoracentesis.

Opportunistic Infections

Infectious agents that cause opportunistic pneumonia in humans include representatives from the classifications bacteria, virus, fungus, protozoa, and parasite. This chapter visits some of these pathogens and their appearance on chest film and CT. Usually, the chest radiograph will reveal the abnormality, but occasionally, the increased sensitivity of CT is necessary to see the pneumonia. Whereas the findings on chest imaging may not be totally pathognomonic of the underlying etiology of infection, they may be highly suggestive and will certainly lead to a reasonable differential diagnosis.

Human Immunodeficiency Syndrome

Since the first description of this disease and AIDS, *Pneumocystis jiroveci* (previously *carinii*) pneumonia (PCP) has been one of the most common complications.

PCP typically presents with increasing shortness of breath and may run a gradual or fulminant course. Chest films classically reveal a bilateral fine to medium reticulonodular pattern, generally bilateral but occasionally focal or unilateral. If the patient remains untreated, the radiograph progressively becomes more opaque and ultimately bilateral homogeneous opacities may be seen. Upper-lobe involvement was seen more commonly when inhaled pentamidine prophylactic therapy was used. The chest film may worsen within a few days of intravenous trimethoprim-sulfamethoxazole treatment secondary to overhydration and the production of pulmonary edema, but this can be treated with diuretics. Otherwise, with treatment, the radiographic course is one of steady improvement until by day 11, complete resolution may be seen. In approximately 10% of patients, pneumatoceles may develop. These are frequently in the upper lobes and will resolve within 2 months. However, they may also lead to pneumothorax, which can be extremely difficult to treat. Pneumothorax is seen in 5% of patients with PCP and AIDS. In about 10% of patients, the chest film may be normal. In some of these patients with normal radiographs, a CT scan will show the typical geographic appearance of ground-glass opacities associated with PCP. Lymphadenopathy and pleural effusions are not part of the PCP picture.

Cytomegalovirus may mimic the look of PCP on chest film, with diffuse bilateral fine to medium reticulonodular opacities. On CT, centrilobular nodules and ground-glass opacities are reported. and in a fairly large number of patients with CMV, the presence of discreet nodules, sometimes several centimeters in size, may help distinguish between these two entities.

Disseminated fungal infections, such as histoplasmosis and coccidiomycosis, generally produce bilateral, fairly symmetric, coarse, nodular opacities on chest radiographs. The nodules and occasionally reticular opacities are larger than those seen with PCP, and this may aid in distinguishing the processes.

Tuberculosis (TB) will appear differently depending on the patient's immune status. In patients with relatively normal CD-4 lymphocyte cell counts, TB will look much like it does in the general population. That is, with primary infection, patients will present with a homogeneous lobar opacity and ipsilateral hilar and/or mediastinal adenopathy. With postprimary infection, patients will present with apical and posterior upper-lobe and/or superior segment lower-lobe heterogeneous opacities with or without cavitation. In patients with low CD-4 cell counts and primary infection, the chest film may show homogeneous lobar opacities with adenopathy similar to immune competent hosts, but the chance of more adenopathy is present. With post primary disease, the organism disseminates more widely, creating a diffuse, coarse, nodular pattern on chest film similar to the pattern seen with fungal infections. Cavitation is not seen, as the body's response is weak, and well-formed granulomas and necrosis are not ususaly seen. If the organism is sensitive to appropriate therapy, then some resolution of the abnormal

findings on chest film should be observed within 1 week.

Ordinary bacterial infections occur with increased frequency in patients with HIV. They are perhaps being seen as a larger percentage of pulmonary infections in populations where antiretroviral therapy is available and in one Veteran's Administration (USA) cohort was seen more frequently than PCP as the initial pathogen. Common organisms are *S. pneumoniae*, *H. influenzae*, *S. aureus*, and *P. aeruginosa*. These usually appear as they do in normal hosts as homogeneous lobar opacities. Parapneumonic effusions may be present. The latter two organisms may present with some cavitation. They should begin to resolve within days of instituting antibacterial therapy, and complete resolution of abnormalities is seen in the majority of patients in about 2 weeks. Other bacterial etiologies, such as *Rhodococcus equi* and *Nocardia asteroides*, are less common and may present as nodules or masses with or without cavitation.

With the use of antiretroviral therapy, noninfectious etiologies of disease have become more prevalent, such as pulmonary hypertension and chronic obstructive pulmonary disease (COPD). Also, the immune reconstitution inflammatory syndrome occurring after antiretroviral treatment may cause confusing clinical and radiological findings.

Two neoplasms, related to infections, may also be seen in patients with AIDS and could cause confusion in generating a differential diagnosis. Non-Hodgkin's lymphoma (NHL) will produce well-defined nodules on chest films. The nodules range in size from about 1 cm to several centimeters in size. Solitary or multiple nodules may be noted. Lymphadenopathy and pleural effusions are also observed. The nodules have a tendency to grow extremely rapidly.

Whereas the nodules with NHL are very well defined, the nodules seen with Kaposi's sarcoma are not. This disease produces poorly marginated nodules that tend to coalesce. They tend to occur in the perihilar lung and lower lobes. On CT, the distribution is along bronchovascular pathways. In almost all cases of Kaposi's involvement in the lungs, cutaneous lesions will be seen. Pleural fluid is frequently seen as well.

Other Immune-Compromised Conditions

Increasing numbers of transplant procedures, both solid organ and bone marrow, have led to the widespread use of induced immunosuppression. Steroids are also being used with increased frequency for a number of medical conditions. With this, infectious complications in the setting of transplantation or steroid use have become a major problem. Whereas prophylactic treatment [with anti pneumocystis, cytomegalovirus (CMV), and occasionally fungal drugs] of these patients leads to some reduction in the number of infections, not all cases are prevented. The appearances on chest film of PCP and CMV will be similar to that in patients with HIV infection and are stated in the section above.

Other viral pathogens are also seen in this setting, including respiratory syncytial virus, parainfluenza virus, adenovirus, and influenza virus in lung transplant patients, and varicella-zoster, which may be seen in patients with lymphoma and those undergoing steroid therapy. On chest film, varicella pneumonia usually produces bilateral symmetric acinar opacities (nodules about 7-10 mm in diameter) that may coalesce as the disease worsens. CT shows similar-sized nodules and distribution as well as ground-glass opacities.

Among the fungal organisms seen with some regularity in this group of immunocompromised patients are *Cryptococcus* and *Aspergillus*. Other emerging agents include *Scedosporium apiospermum*, *Fusarium* spp., and *Mucorales* spp. *Cryptococcus* has numerous types of presentation on chest film. Perhaps most common is the appearance of well-defined nodules, usually solitary but sometimes multiple. If the nodules become masses, the margins may become indistinct. The nodules may cavitate. *Cryptococcus* may also manifest as a lobar pneumonia or diffuse heterogeneous reticulonodular opacities.

A. fumigatus is the usual species responsible for lung infections. The pattern of abnormality seen on chest film depends on the patient's immune status. In the setting of immunosuppression (neutropenia), the typical appearance is that of invasive aspergillosis. In this form of disease, the chest film initially demonstrates a poorly marginated area or areas of homogeneous opacity that may resemble ordinary bacterial pneumonia in appearance and distribution but is occasionally more round and distant from the subpleural lung than common community infections. In some cases, the disease is peripheral and wedge-shaped secondary to infarction caused by the angioinvasive obstruction of pulmonary vessels. In time, the lesions become more discreet and round, thus taking on the look of lung masses. As patients are treated and immune status improves, there may be cavitation within the masses with the formation of an air crescent. Wall thickness of the cavity is generally moderate. The air crescent is created by the contained necrotic debris within the cavity. On CT, initially, the areas of homogeneous opacity may have air bronchograms, and commonly, additional regions of involvement are identified. A halo sign may be observed, which is caused by ground-glass opacity that surrounds (frequently incompletely) a more opaque center of the lesion. The ground-glass portion is an area of hemorrhage and the central area is necrotic lung. The halo sign was thought to be pathognomonic of invasive aspergillosis but can be seen with other infections, neoplasms, and inflammatory diseases.

Take-Home Messages: Usefulness of Imaging Methods in Pulmonary Infections

Despite the increasing use of CT imaging for diagnosing chest disorders, plain film radiography is still the primary imaging modality for patients with suspected pneumonia. The presence of an appropriate lung opacity on a

chest radiograph is considered the gold standard for diagnosing pneumonia. Extensive knowledge of the radiographic appearances of pulmonary infections, their complications, and their course is essential in aiding the referring clinician and ultimately the patient. CT imaging is useful in patients with CAP and NP when there is an unresolving or complicated chest film and at times in immunocompromised patients with suspected pulmonary infections. CT can help differentiate infectious from non-infectious abnormalities. CT may detect empyema, cavitation, and lymphadenopathy when chest films cannot. CT should be performed in immunocompromised patients with a clinical suspicion of pneumonia when the chest film is normal. This is especially true when the early diagnosis of pneumonia is critical, as is the case with immunocompromised and severely ill patients.

To reiterate: No pattern of abnormality seen on chest films can be considered pathognomonic of a specific infection. However, the distribution and appearance of lung opacities, especially in conjunction with clinical information, should enable one to produce a useful, ordered list of most likely possibilities helpful to our clinical colleagues and, most importantly, the patients.

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