09 - SECTION 2 Clinical Syndromes- Community-Acquired Infections

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Michael S. Niederman

Pneumonia DEFINITION Pneumonia is an infection of the pulmonary parenchyma. Despite significant morbidity and mortality, it is often misdiagnosed, mis treated, and underestimated. Pneumonia has usually been classified as community-acquired (CAP), hospital-acquired (HAP), or ventilatorassociated (VAP). A fourth category, healthcare-associated pneumonia, should be discontinued because it did not reliably predict infection with resistant pathogens and was associated with increased use of broad-spectrum antibiotics. Rather than relying on a predefined subset of pneumonia cases, it is better to assess patients individually based on risk factors for infection with a resistant organism, such as certain comorbid illnesses, recent hospitalization, or recent antibiotic therapy. Pneumonia caused by macroaspiration of oropharyngeal or gastric contents, usually referred to as aspiration pneumonia, is best thought of as a point on the continuum that includes CAP and HAP. Estimates suggest that aspiration pneumonia accounts for 5–15% of CAP cases, but reliable figures for HAP are unavailable. The airways or pulmonary parenchyma may be involved, and patients usually represent a clinical phenotype with risk factors for macroaspiration and involvement of characteristic anatomic pulmonary locations. In this chapter, we will not be dealing with pneumonia in immuno compromised hosts. PATHOPHYSIOLOGY Pneumonia is the result of the proliferation of microbial pathogens in the alveoli and the host’s response to them. Until recently, it was thought that the lungs were sterile and that pneumonia resulted from the introduction of potential pathogens into this sterile environment. Typically, this introduction occurred through microaspiration of oro pharyngeal organisms into the lower respiratory tract. The overcoming of innate and adaptive immunity by such microorganisms could result in the clinical syndrome of pneumonia. A complex and diverse community of bacteria in the

lungs consti tutes the lung microbiota. Awareness of this microbiota has prompted a rethinking of how pneumonia develops. Mechanical factors, such as the hairs and turbinates of the nares, the branching tracheobronchial tree, mucociliary clearance, and gag and cough reflexes, play roles in host defense but are insufficient to effectively block bacterial access to the lower airways. In the absence of a sufficient barrier, microorgan isms may reach the lower respiratory tract by a variety of pathways, including inhalation, microaspiration, and direct mucosal dispersion. The constitution of the lung microbiota is determined by three fac tors: microbial entry into the lungs, microbial elimination, and regional growth conditions for bacteria, such as pH, oxygen tension, and tem perature. The key question, however, is how a dynamic homeostasis among bacterial communities results in acute infection. Pneumonia therefore does not appear to be the result of the invasion of a sterile space by a particular microorganism but is more likely an emergent phenomenon dependent upon a number of mechanisms, including self-accelerating positive feedback loops. A possible model for pneumonia is as follows. An inflamma tory event resulting in epithelial and/or endothelial injury results in the release of cytokines, chemokines, and catecholamines, some of which may selectively promote the growth of certain bacteria, such as Streptococcus pneumoniae and Pseudomonas aeruginosa. This cycle of inflammation, enhanced nutrient availability, and release of potential

bacterial growth factors may result in a positive feedback loop that further accelerates inflammation and the growth of particular bacteria, which may then become dominant. In cases of CAP and HAP, the trigger may be a viral infection compounded by microaspiration of oropharyngeal organisms. In cases of true aspiration pneumonia, the trigger may possibly be the macroaspiration event itself.

Once triggered, innate and adaptive immune responses can help contain potential pathogens and prevent the development of pneumo nia. However, in the face of continuing inflammation (and especially if a positive feedback loop becomes sustainable), the process may proceed to a full-fledged pneumonia syndrome. Inflammatory mediators such as interleukin 6 and tumor necrosis factor result in fever, and chemo kines such as interleukin 8 and granulocyte colony-stimulating factor increase local neutrophil numbers. Mediators released by macrophages and neutrophils may create an alveolar capillary leak, resulting in impaired oxygenation, hypoxemia, and radiographic infiltrates. Bacte ria themselves may produce toxins that further amplify the inflamma tory response. Moreover, some bacterial pathogens appear to interfere with the hypoxic vasoconstriction that would normally occur with fluid-filled alveoli, possibly resulting in severe hypoxemia. Decreased compliance due to capillary leak, hypoxemia, increased respiratory drive, increased secretions, and occasionally infection-related bron chospasm all lead to worsening dyspnea. If severe enough, changes in lung mechanics secondary to reductions in lung volume, compliance, and intrapulmonary shunting of blood may cause respiratory failure. PATHOLOGY Classic pneumonia evolves through a series of stages. The initial stage is edema with a proteinaceous exudate and often bacteria in the alveoli. Next is a rapid transition to the red hepatization phase. Erythrocytes in this intraalveolar exudate give this stage its name. In the third phase, gray hepatization, erythrocytes have been lysed and degraded. The neutrophil is the predominant cell, fibrin deposition is abundant, and bacteria have disappeared. This phase corresponds with the successful containment of the infection and improvement in gas exchange. In the final phase, resolution, the macrophage reappears as the dominant cell in the alveolar space and the debris of neutrophils, bacteria and fibrin has been cleared, as has the inflammatory response. CHAPTER 131 Pneumonia This pattern has been described best for lobar pneumococcal

pneu monia but may not apply to pneumonia of all etiologies. In VAP, respi ratory bronchiolitis may precede the development of a radiologically apparent infiltrate. A bronchopneumonia pattern is most common in nosocomial pneumonias, whereas a lobar pattern is more common in bacterial CAP. Despite the radiographic appearance, viral and Pneumo cystis pneumonias represent alveolar rather than interstitial processes. COMMUNITY-ACQUIRED PNEUMONIA ■ ■ETIOLOGY Numerous microbes may cause CAP, including a variety of bacteria, viruses, fungi, and protozoa. Newer viral pathogens include metapneumoviruses, the coronavi ruses responsible for severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS), and the SARS-CoV-2 coronavirus. First described in December 2019, SARS-CoV-2 and its associated clinical disease, COVID-19, reached pandemic proportions and are a cause of significant morbidity and mortality. The COVID-19 pandemic has changed the etiologic profile of CAP, and the ultimate role that the SARS-CoV-2 virus will play as a cause of CAP remains to be seen. The virus and the disease are discussed in detail in Chap. 205. Although most CAP cases are caused by relatively few pathogens, an accurate determination of their prevalence is difficult because labora tory testing methods are often insensitive and indirect (Table 131-1). Separation of potential agents into “typical” bacterial pathogens and “atypical” organisms may be helpful, although both types of pathogens can lead to similar clinical syndromes. The former group includes S. pneumoniae, Haemophilus influenzae, and, in selected patients, Staphylo coccus aureus and gram-negative bacilli such as Klebsiella pneumoniae and P. aeruginosa. The “atypical” organisms include Mycoplasma pneumoniae,

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