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Bacterial Pneumonia

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Continuing Education Activity

Bacterial pneumonia, characterized by inflammation in lung parenchyma and alveolar spaces, is caused by various bacteria, each triggering an intricate interplay with the host immune response. Clinically, patients may experience symptoms such as fever, cough, dyspnea, and chest pain, with potential complications ranging from necrotizing pneumonia and empyema to meningitis, sepsis, and multiple organ failure. Long-term impacts on pulmonary function and quality of life can ensue.

This activity enhances knowledge of the disease's impact on pulmonary function and quality of life, recognizing the potential long-term consequences for patients. The focus on a multidisciplinary approach underscores the importance of collaboration among healthcare professionals in managing bacterial pneumonia. Clinicians will learn practical strategies for history-taking and physical examination to identify exposures and risk factors, enabling targeted and personalized patient management. Additionally, the activity provides insights into vaccination strategies, early mobilization, and rehabilitation, offering a holistic view of patient care. Overall, participation in this activity equips clinicians with the knowledge and skills needed to navigate the complexities of bacterial pneumonia and deliver optimal care within an interprofessional healthcare team.

Objectives:

- Identify the clinical manifestations and history features indicative of bacterial pneumonia to facilitate timely diagnosis.
- Select appropriate diagnostic tests, considering their sensitivity and specificity, to enhance diagnostic accuracy and guide effective therapeutic interventions.
- Implement evidence-based antimicrobial management strategies based on local epidemiology, susceptibility testing, and patient-specific factors to optimize treatment outcomes.
- Coordinate with various healthcare professionals to implement a cohesive and patient-centered approach to enhance the overall quality of care for individuals affected by bacterial pneumonia.

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Introduction

Bacterial pneumonia, characterized by inflammation in lung parenchyma and alveolar spaces, is caused by various bacteria, each triggering an intricate interplay with the host immune response. Clinically, patients may experience symptoms such as fever, cough, dyspnea, and chest pain, with potential complications ranging from necrotizing pneumonia and empyema to meningitis, sepsis, and multiple organ failure. Long-term impacts on pulmonary function and quality of life can ensue.

The word pneumonia is rooted in the ancient Greek word *pneumon* ("lung"). Therefore, pneumonia can be understood as "lung disease."[1] Clinically, it manifests as an inflammation of the parenchyma and alveolar spaces in 1 or both lungs, primarily attributed to infections, though not exclusively.[2]

Among the infectious causes of pneumonia, bacteria, viruses, fungi, and parasites are prominent. Bacterial pneumonia has a significant impact on the overall morbidity and mortality rates of those with pneumonia.[3][4] Various classification methods exist for organizing pneumonia, with the National Institutes of Health (NIH) system being the most widely embraced. This system categorizes pneumonia into community-acquired, hospital-acquired, and atypical pneumonia, further stratified based on severity.[5][6]

Additional classifications may include healthcare-acquired pneumonia (HCAP), which encompasses pneumonia acquired in hospitals, dialysis units, residential aged facilities, and ventilator-associated pneumonia (VAP).[2][7] However, HCAP has been omitted from American guidelines due to a lack of evidence regarding microbiological differences between community-acquired pneumonia (CAP) and HCAP.[8]

Bacterial Pneumonia Definitions

- **Community-acquired pneumonia:** CAP is the acute infection of lung tissue occurring in a patient who has acquired it from the community or within 48 hours of hospital admission.
- **Hospital-acquired pneumonia:** HAP is an acute infection of lung tissue in a nonintubated patient that develops after 48 hours of hospitalization.
- **Atypical pneumonia:** The acute infection caused by a pathogen not detectable by traditional Gram stain or standard cultures.
- **Ventilator-associated pneumonia:** VAP is a type of nosocomial infection of lung tissue that usually develops 48 hours or longer after intubation for mechanical ventilation.

Etiology

CAP can result from an extensive list of agents, including bacteria, viruses, fungi, and parasites. However, this activity will specifically concentrate on bacterial pneumonia and its causative factors. Bacteria have traditionally been classified into 2 categories based on etiology: typical and atypical organisms. Typical organisms can be cultured on standard media or identified through Gram stain, whereas atypical organisms lack such properties.[6]

- Typical pneumonia refers to pneumonia primarily caused by *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Staphylococcus aureus*, Group A *Streptococci*, *Moraxella catarrhalis*, anaerobes (often secondary to aspiration or oropharyngeal contents), and other aerobic Gram-negative bacteria.
- *Legionella* species, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Chlamydia psittaci* predominantly cause atypical pneumonia.
- Opportunistic infections can include organisms such as *Mycobacterium tuberculosis* and *Nocardia* species.

The primary cause of CAP globally is *S pneumoniae,* a trend observed in adults and children.[9][10][11][12] *H influenzae* is frequently cited as the second most prevalent bacterial pathogen. However, the prevalence of other commonly isolated pathogens can vary depending on

the geographical location and local epidemiology. These may include *Klebsiella pneumoniae*, *Legionella* species, *M pneumoniae*, *Chlamydia* species, *Pseudomonas aeruginosa*, and in some areas of the world, *Mycobacterium tuberculosis* and *Burkholderia pseudomallei.*[13] *Escherichia coli, Proteus mirabilis,* and other gram-negative coliform bacteria may be associated with aspiration events.[14] *Bordetella pertussis* may be relevant in unvaccinated populations.

Bacterial pathogens in HAP and HCAP globally include *K pneumoniae*, *E coli*, *P aeruginosa*, *S aureus,* including methicillin-resistant *S aureus* (MRSA), *Enterobacter* species, and *Acinetobacter baumannii* complex.[15][16] Many of these pathogens are also causative agents of VAP, encompassing non-multidrug-resistant (MDR) agents (eg, *S pneumoniae*, other *Streptococcus* species, *H influenzae*) and MDR (eg, *P aeruginosa*, MRSA, *A baumannii* complex and antibiotic-resistant *Enterobacteriaceae*) bacterial pathogens.[17][18]

Epidemiology

Globally, the annual incidence of CAP reaches up to 450 million cases, with approximately 95% occurring in developing countries, contributing to around 4 million deaths annually.[19] In Europe, the incidence varies from 206 to 470 cases per 100,000 patients per year,[20][21] while in China, reported incidence ranges from 298 to 2210 cases per 100 patient-years.[22] In the United States, lower respiratory tract infections surpass all other infections in terms of morbidity and mortality, with approximately 649 to 847 CAP cases per 100,000 patient years being hospitalized, disproportionately affecting low-income and minority groups.[23][24][25][26]

An elevated incidence of CAP is observed in specific age groups, specifically those younger than 4 years and 65 or older.[27][28] In the US, there are an estimated 2.2 to 8 million hospitalized cases annually, though other estimates suggest an annual incidence of 5 million or more CAP cases.[28] Stratified by bacterial predominance, the estimated annual frequency of *M pneumoniae*, *C pneumoniae,* and *Legionella* species cases in the US is estimated at 108,000, 49,700, and 18,000, respectively.[28]

Approximately 100,000 cases of CAP-related deaths occur annually in the US, with global mortality rates ranging from 2.6% to 18.5% at the time of hospitalization and up to 31% to 44.5% within a year of hospitalization.[29][30][31] Admission to the intensive care unit (ICU) is associated with increased mortality risk in CAP.[32] Despite the severity of the condition, up to 90% of CAP cases are successfully managed in the outpatient setting. Hospitalization is typically prompted by comorbidities and the progression of CAP, with a low mortality rate of 0.1% for outpatient-managed cases.[29][33]

Pathophysiology

The lower respiratory tract is not a sterile environment and is constantly exposed to environmental pathogens. Invasion and propagation of bacteria into lung parenchyma at the alveolar level can cause bacterial pneumonia. The body's inflammatory response against this invasion leads to the clinical symptoms of pneumonia.[34]

Various host defenses collaborate in the lungs to prevent microorganism proliferation. Mechanical defenses, such as nasal hair and mucus, and chemical defenses, including alveolar epithelial cell-produced proteins like surfactant proteins A and D with inherent opsonizing properties, contribute.[35] The early innate immune response involves neutrophilic infiltration triggered by toll-like receptor recognition.[36] Activated alveolar macrophages work to engulf and eliminate bacteria. However, if bacterial growth exceeds host defense capacity, their numbers rise.

Pathogenic bacteria have various virulence factors that facilitate the evading host immune responses. Examples include polysaccharide capsules in *S pneumoniae*, adhesion proteins in *M pneumoniae,* and pili associated with biofilm formation in *Legionella pneumophila.*[37][38][39][40]

Alveolar macrophages initiate host inflammatory responses upon bacterial invasion to limit bacterial spread within the human host. The host inflammatory responses are the primary drivers of the clinical manifestations observed in bacterial pneumonia. Cytokines are released in response to the inflammatory reaction, initiating a cascade that manifests as constitutional symptoms. For instance, interleukin-1 (IL-1) and tumor necrosis factor (TNF) are associated with fever, commonly observed in bacterial pneumonia.[34]

Chemokine-like interleukin-8 (IL-8) and colony-stimulating factors like granulocyte colony-stimulating factor (G-CSF) promote chemotaxis and neutrophil maturation, leading to leukocytosis. Other cytokines, such as interleukin-9 (IL-9) and interleukin-13 (IL-13), promote mucin production, forming purulent secretions that aid in bacterial inactivation.[41] These proinflammatory cytokines are responsible for the leakage of the alveolar-capillary membrane at the inflammation site, facilitating the migration of host leukocytes to the site of bacterial burden in the lungs. This leakage can result in decreased gas exchange and associated fibrosis, causing reduced compliance and manifesting as progressive and severe dyspnea.[42]

Inflammation within the pleura can trigger somatic receptors of the phrenic nerve within the parietal pleura, causing pleuritic chest pain.[43] Localized necrosis from damage to the lung parenchyma and tissue extravasation can result in hemoptysis.[44]

Histopathology

Pathologically, lobar pneumonia is characterized by the acute exudative inflammation of a lung lobe. If left untreated, it progresses through the following 4 advanced stages, which may overlap with one another:

1. **Congestion:** Pulmonary parenchyma is not fully consolidated, and microscopically, the alveoli contain serous exudates, including pathogens, some neutrophils, and macrophages.
2. **Red hepatization:** The affected lobe consolidates, acquiring a firm and liver-like texture. Microscopically, there is evidence of fibrin, serous exudate, pathogens, neutrophils, and macrophages. Capillaries are congested, and alveolar walls are thickened.
3. **Gray hepatization:** The lobe maintains a liver-like consistency but appears gray due to supportive and exudate-filled alveoli. This stage can be further subdivided based on the proportion of fibrin to leukocytes.
4. **Resolution:** Typically occurring after a week, this stage involves resolving the inflammatory process. Lymphatic drainage or a productive cough aids in clearing the exudate.[45]

History and Physical

When obtaining a patient's history, it is crucial to thoroughly investigate potential exposures, risks of aspiration, host factors, and presenting symptoms. Various comorbidities, such as cardiopulmonary conditions, neurological impairment, HIV, renal and liver disease, age, sex, smoking, alcohol misuse, and poor dental hygiene, can impact the risk for bacterial pneumonia, particularly CAP.[46][47] Assessing vaccination history becomes pertinent, particularly for infections caused by *H influenzae* and *B pertussis*.[48] Additionally, intravenous drug use can be a risk factor for bacterial pneumonia, including *S aureus* pneumonia.[49] In any

patient presenting with suspected bacterial pneumonia, it is essential to consider and evaluate all these potential risk factors.

Exposure

A comprehensive history of potential exposures is essential, as it aids in establishing possible etiologies. The following associations highlight specific exposures and their corresponding etiologies in bacterial pneumonia:

- *Legionella* pneumonia:
    - Associations: Smoking, contaminated air-conditioning, and water systems, such as in cruise ships and hotels [50][51][52]
- Pneumonia in crowded spaces:
    - Associations: Jails and shelters
    - Etiologies: *S pneumoniae*, *Mycobacteria* species, *M pneumoniae*, and *C pneumonia* [53][54][55][56]
        - Association
- Psittacosis:
    - Associations: Exposure to some birds, such as chickens, turkeys, and ducks
    - Etiology: *C psittaci* [57]
- Tularemia:
    - Associations: Exposure to infected rabbits, possums, arthropods, and rodents
    - Etiology: *Francisella tularensis* [58]*[59]*
- Leptospirosis and plague:
    - Associations: Exposure to infected rodents.
    - Etiologies: *Leptospira* species, *Yersinia pestis* [58][60][61]
- Q fever:
    - Associations: Exposure to macropods, cats, sheep, and cattle
    - Etiology: *Coxiella burnetii* [62][63]
- Melioidosis:
    - Associations: Inhalational proximity to disrupted soils in endemic areas.
    - Etiology: *B pseudomallei* [64]
- Immunocompromised pneumonia:
    - Etiologies: Hospitalization, corticosteroid or cytotoxic therapy, neutropenia
    - Etiology: *P aeruginosa* [65]

Risks of Aspiration

Patients with an increased aspiration risk are more susceptible to developing pneumonia secondary to aspiration. Associated risks include the following:[66][67][68]

- Altered mentation (including dementia. alcoholism, seizure disorders, and psychotropic drug use)
- Polypharmacy
- Dysphagia
- Gastroesophageal reflux disease (GERD)
- Daily supplemental oxygen therapy
- Dependency on oral care
- Decayed dentition
- Iatrogenic feeding supports dependency
- Sputum suctioning
- Smoking
- Urinary catheterization

Host Mechanisms

Obtaining a detailed history is of utmost importance to uncover clues to the etiology of pneumonia. For instance, a history of asthma, chronic obstructive pulmonary disease (COPD), smoking, and immunocompromised status can be indicative of *H influenzae* infection,[69] [70] which most commonly appears in the winter season. Similarly, social, sexual, medication, and family history can all help determine the cause of illness. For example, a history of advanced HIV with CD4 counts less than 100 cells/mm^3 can predispose to increased rates of bacterial pneumonia and opportunistic infections, including pulmonary tuberculosis and pulmonary cryptococcosis.[71]

Physical Manifestations

Features in the history of bacterial pneumonia may vary from indolent to fulminant. Clinical manifestations encompass constitutional findings and those resulting from lung and related tissue damage.[34] The following are significant findings obtained on a history:[72]

- Fever with tachycardia or chills and sweats
- Cough may be nonproductive or productive, with mucoid, purulent, or blood-tinged sputum
- Pleuritic chest pain if the pleura is involved
- Shortness of breath with normal daily routine work
- Fatigue, headache, myalgia, and arthralgia

A productive cough is the most common and significant presenting symptom. Some bacterial causes have historically been associated with particular sputum characteristics, such as:

- *S pneumoniae*: Rust-colored [73]
- *Pseudomonas*, *Hemophilus* species: Green [74]
- *K pneumoniae*: Red currant-jelly [75]
- Anaerobes: foul-smelling and bad-tasting [76]

More recent pooled analyses of randomized trials demonstrate that sputum color is not specific to one organism and may not correlate with culturable bacterial pathogens in up to 55% of cases.[74].

Atypical pneumonia presents with pulmonary and extrapulmonary manifestations. For instance, *Legionella* pneumonia often presents with altered mentation (including encephalitis, meningitis, and peripheral neuropathy), myocarditis, panniculitis, myositis, and cutaneous symptoms.[77][78]

Physical findings in bacterial pneumonia can vary among patients and are primarily influenced by the severity of lung consolidation, the type of organism, the extent of the infection, host factors, and the presence or absence of pleural effusion. The following are major clinical findings:[79][80][81][82]

- Fever (>38 °C or 100.4 °F)
- Hypothermia (<35 °C or 95 °F)
- Tachypnea (>18 breaths/min)
- Tachycardia (>100 bpm)
- Bradycardia (<60 bpm)
- Cyanosis
- Decreased chest expansion on the affected side
- Tracheal deviation in severe cases
- Percussion sounds vary from flat to dull
- Tactile fremitus
- Crackles, rales, and bronchial breath sounds are heard during auscultation
- Increased vocal resonance over the site of lobar consolidation
- Whispering pectoriloquy
- Lymphadenopathy
- Pleural rub
- Egophony (E to A changes)
- Hypoxia on pulse oximetry, which may be related to intrapulmonary shunting from perfused but poorly ventilated alveoli

Clinical findings that may be observed in infants include tachypnea, nasal flaring, intercostal retraction, abdominal distension, decreased feeding, and lethargy.[83] In children younger than 5 years, presentations may include tachypnea, chest indrawing, chest pain, abdominal pain, or pallor if unwell.[84] Children may not always exhibit typical examination findings consistent with pneumonia, necessitating a low threshold for chest radiography when history and examination alone cannot confirm pneumonia.[85]

In older patients, confusion may manifest earlier, and features such as fevers, rigors, and sputum production may not be as pronounced as in younger adults.[86] A critically ill patient may present with sepsis or multiple organ failure.[87]

Examination findings may be specific for certain etiologies, as outlined below:

- Relative bradycardia: *Legionella* species [88]
- Dental illnesses: Anaerobes [89]
- Impaired gag reflex and dysphagia: Aspiration pneumonia [67]
- Cutaneous nodules: Nocardiosis [90]
- Bullous myringitis: *Mycoplasma* species [91]
- Scrofula and spinal deformity: Tuberculosis [92][93]
- Osteomyelitis and lower lobe pneumonia: Actinomycosis [94][95]

Evaluation

The approach to evaluating and diagnosing pneumonia depends on the clinical status, laboratory parameters, and radiological evaluation.

Clinical Evaluation

The clinical evaluation involves a comprehensive patient history and a thorough physical examination to assess the clinical signs and symptoms mentioned previously. Prognostic scoring systems, such as the CURB-65, CORB (confusion [acute], oxygen saturation ≤90%, respiratory rate ≥30 breaths/min, and blood pressure <90 mm Hg [systolic] or ≤60 mm Hg [diastolic]), SMART-COP (systolic blood pressure, multilobar infiltrates, albumin, respiratory rate, tachycardia, confusion, oxygen, and pH), and the Pneumonia Severity Index (PSI) may be used as adequate predictors of medical complications such as ICU admission and death; however, prediction rules are often not robust enough to replace clinical judgment at the time of assessment.[32][96][97]

Illustratively, the CURB-65 score comprises:

- C = Confusion
- U = Uremia (BUN >20 mg/dL)
- R = Respiratory rate (>30 breaths/min)
- B = BP (BP <90/60 mm Hg)
- Age older than 65 years

A score of 0 to 1 advises outpatient treatment, 2 or greater suggests medical admission, and 3 or greater indicates ICU admission.[98]

Laboratory Evaluation

In the diagnostic process for pneumonia, laboratory values play a crucial role. This includes assessing a complete blood count (CBC) with differentials, inflammatory biomarkers like erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), blood cultures, sputum analysis or Gram staining and possibly urine antigen testing, or polymerase chain reaction (PCR) for nucleic acid detection of certain bacteria.[99][100][101]

Arterial blood gas analysis can reveal hypoxia and respiratory acidosis. Severe hypoxia is indicated by pulse oximetry readings of less than 92%, and an elevated CRP level predicts a severe infection.[102]

Obtaining blood cultures before administering antibiotics is recommended in high-severity cases.[103] However, they are positive in less than 10% of cases and may provide minimal clinical benefit in mild to moderate CAP.[104]

Good quality sputum evaluation may reveal more than 25 white blood cells (WBC) per low-power field and fewer than 10 squamous epithelial cells, allowing for visualization of bacteria through Gram stain techniques.[105][106] Microbiology tests can be performed to differentiate between similar microorganisms, such as distinguishing between *Nocardia* species and other anaerobic *Actinomyces* or between *Nocardia* species and the more acid-fast *M tuberculosi*s.[107][108] *S pneumoniae* can be differentiated from other Gram-positive cocci through optochin susceptibility and latex agglutination.[109][110] *Legionella* species can be differentiated from other pulmonary pathogens, except *Nocardia* and *Francisella* species, as it can be grown only buffered with charcoal yeast extraction.[111][112]

Antimicrobial susceptibility testing is crucial for targeting appropriate antibiotic treatment for bacterial pneumonia.[113] Additionally, it helps guide clinicians in adjusting treatment regimens based on the specific resistance patterns of the identified bacterial pathogens, ensuring optimal therapeutic outcomes for patients with bacterial pneumonia.

Specific biochemical evidence may also be present in some bacterial causes. For instance, *Legionella* may be associated with hyponatremia and microhematuria.[114][115] Recognizing these specific biochemical markers aids in the early identification and targeted management of bacterial infections, contributing to a more comprehensive understanding of the underlying etiology and facilitating prompt and effective therapeutic interventions.

Radiological Evaluation

Diagnostic imaging is pivotal in pneumonia evaluation, with a chest X-ray as an initial imaging test. Pulmonary infiltrates identified on plain film are considered a gold standard for diagnosis when supported by laboratory and clinical features.[3][116] The chest X-ray may uncover consolidations or parapneumonic effusions. *S aureus* pneumonia may manifest with multilobar involvement, patchy opacities, and cavitary lesions indicative of abscess formation.[117]

Transthoracic and transesophageal echocardiograms exclude endocarditis in cases of *S aureus* pneumonia with bacteremia.[118] Early identification of endocarditis through echocardiograms allows for prompt intervention and tailored treatment strategies, improving patient outcomes in *S aureus* pneumonia with bacteremia cases.

H influenzae infections are more likely to exhibit bronchopneumonia changes on chest radiography.[119] *P aeruginosa* manifestations include patchy opacities with abscess formation, a pattern also observed in *B pseudomallei* pneumonia.[120] A "bulging fissure" sign associated with pulmonary abscesses may be evident in *K pneumoniae. However,* it is also observed in *S pneumoniae, H influenzae, M tuberculosis,* and *Y pestis* pulmonary infections.[121][122]

Chest CT is reserved for complex cases with an unknown cause or those with structural lung disease.[123] Bronchoalveolar lavage is conducted in intubated patients, providing culture samples for further analysis.[124]

Treatment / Management

Once a pneumonia diagnosis is confirmed, the initial step in treatment involves a risk assessment to determine whether the patient should receive outpatient or inpatient care.[3][125][126]. In all cases of severe bacterial pneumonia, immediate initiation of empirical therapy is crucial to prevent sepsis, a common cause of ICU admission and mortality.[127]

While various scoring calculators can be used to risk stratify patients, clinical judgment remains essential in their application. The choice of empirical antibiotics depends on local epidemiology and the prevalent bacterial pathogens in the region.[3][128][129][130] Options may include

benzylpenicillin or third-generation cephalosporins for *S pneumoniae*, macrolides for *Legionella* species, and carbapenems or ceftazidime for *B pseudomallei*, varying by geographic location.

For administration, benzylpenicillin powder is reconstituted with water for injection, while ceftriaxone is mixed with 10 mL water for intravenous and intramuscular administration, often combined with lignocaine.[131] Respiratory fluoroquinolones or a combination of beta-lactam antibiotics with macrolides are recommended as first-line treatments in the US.[132]

Rationalizing antimicrobial therapy is advised when susceptibility testing results are available. For *P aeruginosa*–related pneumonia with sepsis, piperacillin and tazobactam may be used, and depending on resistance profiles, other options include ceftazidime, ceftazidime/avibactam (for difficult-to-treat *Pseudomonas*), cefepime, ciprofloxacin, and meropenem [133].

For outpatient management of patients lacking comorbidities, suitable antimicrobial options encompass those from penicillin, macrolide, and tetracycline classes. Patients with comorbidities may benefit from a respiratory fluoroquinolone or a combination of amoxicillin/clavulanate with a macrolide.[132] In cases requiring inpatient care, obtaining a microbiological specimen, such as sputum or blood cultures for severe cases, is advised to guide antimicrobial management. However, outpatient cases may not necessitate culture and susceptibility testing. The use of antipyretics is recommended to alleviate symptoms and reduce fevers in affected patients [134].

Following a positive culture result, treatment should be adjusted on the susceptibility results of the culture-specific pathogen. In cases of severe bacterial pneumonia, combination therapy may enhance survival, particularly in CAP patients with associated shock.[135] Patients with bacterial pneumonia without complications or underlying chronic respiratory illness, such as bronchiectasis, may be successfully treated with total antimicrobial durations as short as 5 days, aligning with guidelines established by the Infectious Diseases Society of America and the American Thoracic Society.[136]

The use of corticosteroids in bacterial pneumonia remains controversial and may be considered in patients experiencing persistent hypotension with presumed adrenal insufficiency.[137][138][139][140] Increasing evidence suggests the potential utility of corticosteroids in the intensive care population, showing benefits in preventing progression to mechanical ventilation and reducing mortality by 28 days when administered within 24 hours of severe CAP development.[141][142] The role of noninvasive ventilation is debated in bacterial pneumonia without respiratory failure cases, given high treatment failure rates, with consideration primarily given to those with preexisting COPD.[143] Bronchodilator therapy, without corticosteroid use, may offer limited benefit in moderate to severe exacerbations of COPD with pneumonia.[144]

In addition to pharmacological intervention, nonpharmacological management of bacterial pneumonia includes promoting smoking cessation, providing counseling support, and ensuring vaccination against influenza, COVID-19, and pneumococcus.[145][146][147][148] Encouraging the treatment and correction of dysphagia and addressing any intervening comorbidities are also crucial components of comprehensive patient care.[149]

Home-based treatment patients should be scheduled for a follow-up visit within 2 to 3 days to evaluate the potential development of pneumonia-related complications.[150] For patients discharged following hospitalization for bacterial pneumonia, a follow-up assessment within 7 days is recommended to determine the need for continued antibiotic management or potential rehydration.[151]

The evidence for chest physiotherapy in pneumonia is less clear than its effectiveness in bronchiectasis exacerbations. However, some evidence suggests it may reduce hospitalization duration and the need for mechanical ventilation.[152] As a result, various national guidelines have not recommended traditional airway clearance methods.[153] Instead, patients with

pneumonia are encouraged to sit out of bed for at least 20 minutes within the first 24 hours of hospitalization and increase mobility each subsequent day, with consideration for the use of positive expiratory pressure. Lateral decubitus positioning towards the lung affected by consolidation may alleviate symptom burden in bacterial pneumonia with respiratory failure.[154] Prone and upright positioning may play a role in reducing the incidence of VAP.[155][156]

Other measures demonstrated to be effective in bacterial pneumonia include:

- Hydration [152]
- Monitoring with pulse oximetry [157]
- Mechanical support if patients are in acute respiratory distress [158]
- Nutrition [159]
- Early mobilization [153]

Differential Diagnosis

Distinguishing pneumonia from other pulmonary diseases can be challenging, particularly in patients with coexisting pulmonary pathology. The differential diagnoses vary for children and adults:

Differential Diagnosis in Children

- Asthma or reactive airway disease exacerbation
- Bronchiolitis
- Croup
- Respiratory distress syndrome
- Epiglottitis

Differential Diagnosis in Adults

- Acute and chronic bronchitis
- Acute pulmonary edema
- Acute respiratory distress syndrome
- Aspiration of a foreign body
- Asthma exacerbation
- Atelectasis
- Bronchiectasis
- Bronchiolitis
- Chronic obstructive pulmonary disease exacerbation
- Fungal pulmonary infection (including *Pneumocystis jiroveci* pneumonia)
- Interstitial lung disease
- Lung abscess
- Organizing pneumonia

- Respiratory failure
- Viral pulmonary infection

Prognosis

The prognosis of pneumonia is influenced by various factors, including age, cognitive status, comorbidities (including malignancy and chronic respiratory diseases), immunosuppression, chronic alcohol misuse, hospital setting (inpatient or outpatient), and the need for ventilatory support.[160] The prognosis and survival for bacterial pneumonia are generally more favorable in otherwise healthy patients.[161]

Older patient groups tend to experience poorer clinical outcomes than their younger counterparts.[162] An observational study noted higher mortality rates within 30 days from the initial infection in patients aged 65 and older with *S pneumoniae* pulmonary infections, even after adjusting for vaccination status.[163] Mortality in bacterial pneumonia may also be associated with the type of pathogen involved. While *S pneumoniae* is a common cause of mortality, *Pseudomonas* species, *S aureus*, and polymicrobial infections exhibit higher mortality rates, possibly attributable to their virulence factors.[164][165][166][167]

If left untreated, pneumonia can result in an overall mortality rate of up to 30%.[162][168] A higher PSI score has been correlated with less favorable resolution of bacterial pneumonia by elevated inflammatory markers, anemia, confusion, and elevated blood urea nitrogen.[169] Early clinical failure is associated with persistent tachypnea, hypoxia, confusion, and arterial blood acidosis within the first 3 days of pneumonia treatment.[170] In VAP, a higher Acute Physiology and Chronic Health Evaluation II (APACHE II) score exceeding 21 or a Sequential Organ Failure Assessment (SOFA) score surpassing 6 increases the mortality risk, particularly if the elevated SOFA score persists a week after diagnosis.[171] When bacterial pneumonia coexists with comorbid viral respiratory infections such as influenza and COVID-19, there is an elevated mortality risk, underscoring the importance of vaccination in vulnerable and at-risk populations.[172][173]

Antimicrobial resistance is increasing globally, fueled by the extensive use of broad-spectrum antibiotics in human healthcare and the agricultural industry.[174][175] This trend restricts regional choices for antimicrobial treatment of bacterial pneumonia, escalates healthcare system expenses, and is linked to a poorer prognosis for patients with bacterial pneumonia [176].

Over the long term, nosocomial bacterial pneumonia has been linked to elevated mortality rates at 1 year, with a heightened risk among older patients residing in aged care institutions.[177] Among this demographic, factors such as compromised nutritional status, chronic steroid use, and preexisting comorbidities are associated with a poorer prognosis.[178] This underscores the significance of implementing regular and appropriate follow-up measures for at-risk population groups following bacterial pneumonia.

Complications

Bacterial pneumonia complications can be severe, leading to the exacerbation of preexisting comorbidities, respiratory failure, and sepsis associated with multiorgan failure and coagulopathy.[179][180][181] Other potential complications of bacterial pneumonia include:[48][161][182][183][184][185]

- Lung fibrosis
- Destruction of lung parenchyma
- Necrotizing pneumonia

- Impaired ventilatory function
- Cavitation
- Empyema
- Pulmonary abscess
- Meningitis
- Death

Deterrence and Patient Education

Patients should receive counseling to quit smoking, avoid alcohol intoxication, and maintain dental hygiene, as evidence suggests these behaviors may be risk factors for pneumonia.[146][186][187] Since bacterial pneumonia is transmitted by direct inoculation from fomites or respiratory droplets, emphasizing appropriate hand hygiene is crucial to prevent transmission.[188] Additionally, to prevent bacterial pneumonia, recommendations include:

- Vaccination against pneumococcus for older individuals and those who are immunocompromised [148]
- Annual vaccination against influenza for at-risk populations [189]
- Elderly and immunocompromised patients should be instructed to seek medical assistance as soon as they develop symptoms such as dyspnea, rigors, or fever [190]

Pearls and Other Issues

The pearls of bacterial pneumonia include:

- Most patients exhibit improvement within 48 to 72 hours [136][170]
- Chest X-ray findings lag behind clinical features and may take up to 8 weeks or longer to clear [191][192]
- If patients do not show improvement within 72 hours, an alternative cause should be suspected. Host factors need analysis, and considerations should include antibiotic resistance or the development of complications (eg, empyema) [170]

Enhancing Healthcare Team Outcomes

The management of pneumonia necessitates an interprofessional team to consider the patient's individual factors, comorbidities, and any resulting sequelae.[193] While most patients can be managed as outpatients, neglecting proper treatment for bacterial pneumonia may lead to elevated morbidity and mortality rates.[29][31]

In addition to pharmacotherapy, patients may require input from allied health professionals to address the underlying risk factors for bacterial pneumonia. This may involve speech pathology to manage dysphagia, psychological support for harm reduction in cases related to hazardous alcohol and intravenous drug use, consultation with a dietician for nutritional supplementation, and physiotherapy engagement for early mobilization.[153][159][194] A dental review and oral health management may reduce the incidence of pneumonia in residential aged-care facilities.[195] The treating clinician should continue to address underlying comorbidities, involve rehabilitation services when necessary, and provide regular encouragement for smoking cessation and alcohol abstinence.

Clinicians should actively promote adherence to appropriate influenza and pneumococcal vaccines.[148][189] Pharmacists play a crucial role in antimicrobial stewardship, ensuring responsible antimicrobial prescribing and educating patients about the importance of medication adherence while monitoring and addressing any adverse effects.[196][197] Antimicrobial stewardship pharmacists can guide antimicrobial choices, considering factors such as drug monitoring and drug-drug interactions, allergies, and renal and hepatic impairment.[198]

Nursing staff are critical for the daily nonpharmacological management of pneumonia in the inpatient setting, including cases of VAP. They may also participate in community vaccinations and provide education to at-risk patients.[199][200] Additionally, nurses can counsel patients on appropriate dosing and administration of medications, monitor treatment progress, and promptly report any issues to the managing clinician.

Finally, the treating clinician should encourage appropriate follow-up with patients to ensure the clinical resolution of their illness [150][151]. Successful management of pneumonia requires open communication and collaboration among the interprofessional team, working together to reduce the morbidity of pneumonia associated with this condition.

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