THE EUROPEAN SOCIETY OF MEDICINE
Medical Research Archives, Volume 14 Issue 5

NARRATIVE LITERATURE REVIEW

Community-acquired Pneumonia: From Evidence to Clinical Practice in the Optimization of Care

Calsaverini Leal R[1*], Schimidt SC[2], Menezes LA[3], Leal HC[4]

[1]PhD in Health Sciences. Physiotherapist specialized in Pulmonary Rehabilitation. Faculty member at UNILAGO and FAMERP.
[2]PhD in Health Sciences. Specialist Physician. Faculty member at UNILAGO and FAMERP.
[3]Medical student at UNILAGO. Registered Nurse specialized in Urgent and Emergency Care.
[4]Medical degree from FAMECA.

[*]calsaverini@gmail.com

OPEN ACCESS

PUBLISHED
31 May 2026

CITATION
Calsaverini Leal, R., Schimidt, SC., et al., 2026. Community-acquired Pneumonia: From Evidence to Clinical Practice in the Optimization of Care. Medical Research Archives, [online] 14(5).

ISSN
2375-1924

ABSTRACT

Pneumonia is an acute inflammatory process of infectious origin that affects the terminal airways and the pulmonary parenchyma and may be caused by viruses, bacteria, and, less frequently, fungi and parasites. Community-acquired pneumonia (CAP) is defined as pneumonia occurring outside the hospital setting or manifesting within up to 48 hours after admission to healthcare services, representing a significant clinical challenge due to its high frequency and potential severity. Globally, CAP has an estimated incidence ranging from 1.5 to 14 cases per 1,000 inhabitants and remains one of the leading causes of morbidity and mortality, with an approximate mortality rate of 41.7 per 100,000 inhabitants. In Brazil, respiratory diseases are among the leading causes of death, with pneumonia being one of the most prevalent conditions and a major burden on the healthcare system. The etiology of CAP is broad, with more than one hundred microorganisms potentially involved; however, most cases with an identified pathogen are associated with specific agents, particularly bacteria and respiratory viruses. In recent decades, a reduction in the incidence of bacterial CAP has been observed, possibly related to the expansion of pneumococcal vaccination among at-risk groups and widespread childhood immunization. In contrast, viral infections have shown a progressive increase, altering the epidemiological profile of the disease. Despite advances in diagnosis and therapeutic management, CAP remains associated with a high burden of morbidity and mortality, highlighting the need for continuous updating of evidence-based clinical strategies. Therefore, the aim of this study is to analyze, through a literature review, the main causes of community-acquired pneumonia and to synthesize scientific evidence to support clinical practice in the recognition, management, and prevention of complications associated with this multifactorial condition.

Definition

Pneumonia is defined as an acute inflammatory process of infectious origin that affects the terminal airways and the pulmonary parenchyma and may be caused by viruses, bacteria, and, less commonly, fungi and parasites. Community-acquired pneumonia (CAP) affects patients outside the hospital setting or manifests within up to 48 hours after admission to a healthcare facility. Healthcare-associated pneumonia refers to pneumonia acquired by individuals in healthcare settings (e.g., nursing homes, hemodialysis centers) or following recent hospitalizations. It was previously used to identify patients at risk for infection by multidrug-resistant pathogens. However, this classification may have overestimated the severity of these patients, leading to increased inappropriate antibiotic use. Currently, its use is discouraged[1].

Epidemiology

Worldwide, community-acquired pneumonia (CAP) has an incidence ranging from 1.5 to 14 cases per 1,000 inhabitants and remains one of the leading causes of death globally, with high mortality rates reported across different populations[2,3]. In Brazil, respiratory diseases rank among the main causes of death, with pneumonia being one of the most frequent conditions. The highest incidence of CAP occurs at the extremes of age, particularly in children under 5 years and adults over 65 years, with a higher prevalence among males and during colder seasons such as autumn and winter (1). Mortality varies according to disease severity at diagnosis, being approximately 1% in outpatients, ranging from 4% to 18% among hospitalized patients, and reaching up to 50% in those requiring intensive care[2,3] .

Etiology

More than one hundred microorganisms—including bacteria, viruses, fungi, and parasites—have been implicated in the etiology of community-acquired pneumonia (CAP); however, most cases with an identified pathogen are associated with a limited group of agents, particularly *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, and atypical pathogens such as *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Legionella pneumophila*[4,5]. Historically, *S. pneumoniae* accounted for the majority of cases, reaching up to 95% in earlier reports; although it remains the most frequently identified pathogen, its relative incidence has declined over time, likely due to the widespread implementation of pneumococcal vaccination in both at-risk adults and pediatric populations[4]. Conversely, viral respiratory infections have shown a progressive increase in recent years, contributing to shifts in the epidemiological profile of CAP[4].

The distribution of etiologic agents varies according to disease severity and the clinical setting, and polymicrobial infections may occur, often involving combinations of pneumococcus, respiratory viruses, and atypical pathogens[4,5]. Specific epidemiological and clinical features should guide the etiological investigation. Anaerobic bacteria are commonly implicated in aspiration pneumonia, particularly in patients with alcohol use or poor oral hygiene. *Staphylococcus aureus* should be considered in intravenous drug users and in the presence of pneumatoceles on imaging, while community-acquired methicillin-resistant *S. aureus* (CA-MRSA) is associated with necrotizing forms of the disease [5].Additionally, pathogens such as *S. aureus*, Enterobacteriaceae, and *Pseudomonas aeruginosa* are more frequently observed following viral pneumonia, prior antibiotic exposure, or in patients with chronic lung diseases[5]. Although these pathogens are less common, they are associated with higher mortality and antimicrobial resistance, and their likelihood increases in the presence of risk factors such as advanced age, prior antibiotic use, comorbidities (e.g., COPD, bronchiectasis, and renal disease), and altered mental status[5].

Pathophysiology

The main mechanism of infection is the microaspiration of pathogenic organisms from the oropharynx. The lungs are naturally exposed to particulate matter and microorganisms present in the upper airways. During episodes of microaspiration, these organisms reach the lower respiratory tract, causing an imbalance in the normal lung flora, a phenomenon known as dysbiosis. This concept represents a paradigm shift in the study of the pulmonary microbiome and challenges the previous belief that the lower respiratory tract is sterile. Other mechanisms associated with

pneumonia include the direct inhalation of droplets containing microorganisms, aspiration of large amounts of gastric contents (Mendelson syndrome), and hematogenous spread (e.g., in intravenous drug users). Several host-related factors are associated with an increased risk of CAP[6].

Clinical Presentation

Patients generally present with cough, sputum production, dyspnea, chest pain, and fever. Cough is the most common finding and is present in up to 80% of cases. Systemic manifestations include prostration, fever, headache, sweating, chills, arthralgia, myalgia, and delirium. Physical examination findings include tachypnea, reduced chest expansion, increased tactile vocal fremitus, crackles, rhonchi, and dullness or subdullness to percussion. Extrapulmonary findings should be assessed to rule out infection at other sites (arthritis, endocarditis, meningitis, hepatitis, pericarditis)[7,8].

In elderly patients, fever and cough are less frequent, and the most common manifestations include tachypnea (usually with a respiratory rate greater than 24 breaths per minute), confusion, delirium, falls, functional decline, and decompensation of underlying diseases. Tachypnea may be the initial sign of pneumonia and may be present in more than 60% of elderly patients. Although nonspecific, it is a highly sensitive indicator of respiratory infection and has value for both diagnosis and prognosis[7,8].

Clinical Diagnosis

CAP presents as an acute lower respiratory tract disease, and the key clinical data for diagnosis include:

- Cough and one or more of the following symptoms: sputum production, dyspnea, and chest pain.
- At least one systemic finding: confusion, headache, sweating, chills, myalgia, fever (temperature ≥ 37.8°C).
- Focal findings on chest physical examination (the absence of abnormalities does not exclude the diagnosis).
- Compatible radiological changes on chest X-ray or computed tomography.
- CURB-65 is based on variables from which its name is derived: Confusion (score ≤ 8 according to the abbreviated mental test score); Urea > 50 mg/dL; Respiratory rate > 30 breaths/min; Blood pressure: systolic < 90 mmHg or diastolic < 60 mmHg; and Age ≥ 65 years.
- Comorbidities may increase the risk of complications in CAP, such as alcoholism, heart failure, liver failure, and neoplasms[9,10].
- SCAP: Major criteria include pH < 7.30 (13 points) and systolic blood pressure < 90 mmHg (11 points). Minor criteria include respiratory rate > 30 breaths/min (9 points); PaO_2/FiO_2 < 250 (6 points); urea > 30 mg/dL (5 points); altered level of consciousness (5 points); age ≥ 80 years (5 points); and the presence of multilobar or bilateral radiological infiltrates (5 points). A score ≥ 10 points predicts a higher risk of requiring mechanical ventilation and vasoactive drugs[10,11].
- SMART-COP: Systolic blood pressure < 90 mmHg (2 points); multilobar involvement (1 point); albumin < 3.5 g/dL (1 point); respiratory rate ≥ 25 breaths/min (1 point); heart rate > 125 bpm (1 point); confusion (1 point); SpO_2 < 93% or PaO_2 < 70 mmHg (2 points); and pH < 7.30 (2 points). A score greater than 3 identified 92% of patients who required invasive mechanical ventilation or vasoactive drugs during the course of CAP. The presence of a new radiological abnormality is essential to support the diagnosis of pneumonia, as symptoms are nonspecific and may also be present in upper respiratory tract infections. In elderly patients, the absence of fever and respiratory symptoms is associated with an increased risk of mortality, and interpretation of imaging studies may be impaired by the presence of comorbidities[9,11].

Etiological Diagnosis

The main mechanism of infection is the microaspiration of pathogenic organisms from the oropharynx. The lungs are naturally exposed to particulate matter and microorganisms present in the upper airways. During episodes of microaspiration, these organisms reach the lower respiratory tract, causing an imbalance in the normal lung flora, a phenomenon known as dysbiosis. This concept represents a paradigm shift in the study of the pulmonary microbiome and challenges the previous belief that the lower respiratory tract is sterile. Other mechanisms associated with pneumonia include the direct inhalation of droplets containing microorganisms, aspiration of large amounts of gastric contents (Mendelson syndrome), and hematogenous spread (e.g., in intravenous drug users)[12,13].

If you want, I can standardize all sections into a **single cohesive manuscript (journal-ready English)**

with consistent terminology and formatting.Several host-related factors are associated with an increased risk of CAP. The search for the etiological agent should be guided by clinical severity, epidemiological risk factors, and response to treatment. For outpatients, the identification of a specific pathogen is unnecessary, given the high efficacy of empirical antibiotic therapy and the low mortality rate[12,13].

In cases of severe CAP, targeted therapy reduces mortality; however, initiation of treatment should not be delayed while awaiting etiological diagnosis. In these cases, blood cultures (two samples), sputum examination with direct microscopy and culture for aerobic organisms are required, as well as tracheal aspirate or bronchoscopy with bronchoalveolar lavage (BAL) in selected cases. Urinary antigen testing for the identification of pneumococcal and *Legionella*-associated pneumonia is recommended, as it may modify the treatment regimen. The sensitivity of this test is not affected even after the initiation of antibiotic therapy[12,13].

Additional Tests

CHEST X-RAY

Chest X-ray (posteroanterior and lateral views) is the initial diagnostic method (demonstrating the appearance of interstitial opacities or consolidation). It assists in assessing disease severity (identifying multilobar involvement or associated pleural effusion), suggests differential diagnoses (such as abscess, tuberculosis, and pulmonary mass), and is also useful for monitoring response to treatment. It is not capable of determining the etiological agente. In most cases, complete resolution of radiological abnormalities occurs within two weeks after symptom onset and within six weeks in elderly patients, as well as in those with chronic obstructive pulmonary disease (COPD), immunosuppression, alcoholism, diabetes, and multilobar pneumonia[14,15].

Therefore, after antimicrobial treatment, chest X-ray should be repeated six weeks after symptom onset in smokers, individuals over fifty years of age, and/or those with chronic obstructive pulmonary disease (COPD). Evaluation of radiological resolution in this group is important due to their high risk of bronchogenic carcinoma. Persistent parenchymal abnormalities should be investigated. Persistence of imaging findings suggests that differential diagnoses, particularly neoplasia, should be considered. In patients with persistent clinical symptoms or abnormal physical examination findings, follow-up chest X-ray is also indicated[14,15].

CHEST ULTRASOUND

Chest ultrasound is useful for identifying parenchymal consolidation and for assessing the presence of associated complications. It may also assist in performing thoracentesis in cases of pleural effusion[14,15].

CHEST COMPUTED TOMOGRAPHY

Chest computed tomography (CT) should be performed when there is diagnostic uncertainty or suspicion of complications. The finding of mediastinal lymphadenopathy during an episode of CAP is common[14].

Laboratory Tests

Complementary tests should be performed only in patients with CAP who require hospitalization, as they provide information regarding organ dysfunction, inflammatory status, and disease severity. The following should be requested:

- Complete blood count, urea, creatinine, sodium, potassium, and blood glucose.
- **Arterial blood gas analysis**: should be performed only if peripheral oxygen saturation (pulse oximetry) is below 90% on room air, in cases of respiratory failure, or when there is suspicion of hypercapnia due to chronic obstructive pulmonary disease. Hypoxemia indicates the need for hospitalization.
- **HIV serology**: patients with atypical clinical, laboratory, or radiological findings should undergo HIV testing (after proper counseling and patient consent).
- **C-reactive protein (CRP)**: an inflammatory marker useful for assessing therapeutic response and with prognostic value. A worse prognosis or progression to complications is associated with elevated levels or the absence of at least a 50% reduction from baseline after the fourth day of antimicrobial treatment.
- **Procalcitonin (PCT)**: an inflammatory marker with high sensitivity but low specificity for differentiating bacterial from viral infection. It has prognostic value. PCT values above 0.25 µg/L support the use of antibiotic therapy, with stronger agreement when above 0.5 µg/L. Values below 0.1 µg/L support withholding antimicrobial therapy. A 90% reduction from baseline indicates treatment discontinuation. Studies have demonstrated

reduced duration of antibiotic use without increased mortality or treatment failure based on serial measurement of this marker.

- **AFB smear and culture for mycobacteria**: should be requested only in patients with imaging findings and clinical history compatible with tuberculosis, such as cavitary lung lesions and the presence of centrilobular nodules in a tree-in-bud pattern[15,16].

Risk Stratification

Assessment of disease severity should be performed in all patients diagnosed with CAP. Based on severity, decisions are made regarding the appropriate setting for treatment, the diagnostic strategy (including the need for etiological investigation), and initial antibiotic therapy. The most commonly used severity indices are the North American **PSI (Pneumonia Severity Index)** and the British **CURB-65** and **CRB-65**. The latter are simpler tools for severity stratification, and all are associated with 30-day mortality[10,17].

PSI is a scoring system that includes twenty variables and classifies patients into five severity categories associated with mortality risk. It has limitations due to the complexity of score calculation, underestimation of severity in younger patients without comorbidities, and overestimation of mortality in elderly individuals. CURB-65 is a scoring system that includes only five variables representative of acute disease in CAP, with each variable corresponding to 1 point. **The decision regarding the site of care** The decision about the site of treatment helps predict the likely etiologic agent and guides antibiotic selection[10].

Additionally, the presence of hypoxemia (SpO_2 < 90% on room air) on pulse oximetry; decompensated comorbidities; multilobar or bilateral consolidations; pleural effusion suspected of empyema; inability to take medications orally; poor socioeconomic conditions; and cognitive impairment are also indications for hospitalization. Patients with severe community-acquired pneumonia (CAP), according to the criteria suggested by Ewig, should be admitted to the intensive care unit (ICU)[10].

Differential Diagnoses

TRACHEOBRONCHITIS

Acute infection associated with productive cough, dyspnea, wheezing, and chest discomfort/pain, after exclusion of alternative diagnoses.

EXACERBATION OF PRE-EXISTING STRUCTURAL LUNG DISEASE (COPD/BRONCHIECTASIS)

May present with a similar clinical picture; however, there are no radiographic findings typical of pneumonia[17].

Treatment

Early antibiotic therapy (within up to four hours after diagnosis) is associated with reduced mortality in severe pneumonia. Treatment duration is determined according to disease severity, etiologic agent, associated complications, and the patient's clinical course[10,15].

OUTPATIENT TREATMENT

Patients may receive home treatment if there is assurance of adherence to the prescribed antibiotic. For otherwise healthy individuals, macrolides and beta-lactams are alternatives. Antibiotic coverage for atypical pathogens in mild CAP remains controversial, and several studies have not demonstrated advantages with this approach. North American, European, British, and Latin American guidelines differ regarding outpatient management due to varying positions on antimicrobial resistance[10,15].

The 2018 Brazilian guidelines for CAP management state that pneumococcal resistance to penicillin should not be a concern in less severe cases. They suggest monotherapy with either a beta-lactam or a macrolide for outpatients without comorbidities, no recent antibiotic use, no risk factors for resistance, and no contraindications or history of allergy to these drugs. Regarding macrolides, azithromycin is more effective *in vitro* against most strains of *Haemophilus influenzae* than clarithromycin and should therefore be preferred in patients with COPD. In patients without comorbidities, the guidelines reinforce avoiding fluoroquinolones due to recent warnings from the U.S. Food and Drug Administration about the potential risk of serious adverse effects[18,19].

For patients with comorbidities (COPD, liver or kidney disease, cancer, diabetes, congestive heart failure, alcoholism, or immunosuppression), those who have used antibiotics in the past three months, or those from regions where macrolide resistance exceeds 25% (e.g., the U.S. and some other countries), recommended options include either a beta-lactam combined with a macrolide or

monotherapy with a respiratory fluoroquinolone for at least five days. Amoxicillin-clavulanate may be used when Gram-negative infection (e.g., *Klebsiella*, *Haemophilus*) is suspected, although it has no greater activity against pneumococcus and does not cover atypical pathogens[10,18].

INPATIENT WARD TREATMENT

Monotherapy with a respiratory fluoroquinolone (levofloxacin, moxifloxacin, or gemifloxacin) or a beta-lactam combined with a macrolide is recommended by guidelines for hospitalized CAP patients due to good coverage and favorable clinical outcomes. However, concerns remain about the emergence of multidrug-resistant organisms.Macrolide monotherapy is contraindicated due to the high prevalence of *Streptococcus pneumoniae* resistance to this class. The need for atypical pathogen coverage remains controversial. Current recommendations favor a beta-lactam plus a macrolide or a respiratory fluoroquinolone alone. Beta-lactam monotherapy should only be used when *Legionella* infection has been excluded[10,18].

ICU TREATMENT

In severe CAP and septic shock, combination therapy provides greater benefit than monotherapy. Systematic reviews and meta-analyses suggest that beta-lactam plus macrolide therapy is superior to beta-lactam plus quinolone, although randomized trials are still needed. Possible explanations include coinfection with atypical pathogens, immunomodulatory effects of macrolides, and pharmacologic synergy. Therapy selection should consider risk factors for specific pathogens. Risk factors for *Pseudomonas aeruginosa* include:

- Structural lung disease (COPD with FEV_1 < 30%, bronchiectasis, or cystic fibrosis)
- Neutropenia (< 1,000/mm³)
- Chronic corticosteroid use (> 10 mg/day of prednisone or equivalent)
- Recent antibiotic use (> 7 days within the past 30 days)
- Recent hospitalization (within the past 7 days)

In the absence of risk factors for *Pseudomonas*, options include:

- Beta-lactam (ceftriaxone or cefuroxime) + macrolide (azithromycin or clarithromycin), or
- Beta-lactam + respiratory fluoroquinolone (levofloxacin or moxifloxacin)

In the presence of risk factors, preferred agents include respiratory fluoroquinolones, piperacillin-tazobactam, meropenem, and/or polymyxin B (as monotherapy or combination therapy)[10, 15, 17, 18].

DURATION OF TREATMENT

The optimal duration remains debated. Clinical parameters (afebrile for 48–72 hours, clinical improvement) and laboratory markers (decrease in leukocytosis, C-reactive protein, and procalcitonin) guide decisions.

For patients with good clinical response, therapy may be discontinued after seven days.

Patients with suspected viral pneumonia due to influenza A (H1N1) should receive antiviral therapy along with coverage for *Streptococcus pneumoniae* and *Staphylococcus aureus* if coinfection is suspected[10,18].

SYSTEMIC CORTICOSTEROIDS

The use of systemic corticosteroids in severe CAP has been associated with reduced length of hospital stay, lower rates of treatment failure, less radiologic progression, and reduced need for mechanical ventilation. Although recent studies support their use in severe CAP, further research is needed to clarify their effects on mortality, optimal dosing, and treatment duration[19].

CONVERSION FROM INTRAVENOUS TO ORAL ANTIBIOTICS

During this transition, preference should be given to continuing oral therapy with the same drug or within the same class, aiming to enable early discharge and reduce costs.

HOSPITAL DISCHARGE CRITERIA

After initial management of CAP, patients who demonstrate clinical improvement should be considered eligible for hospital discharge[10, 15, 17, 18].

COMPLICATIONS AND TREATMENT FAILURE

Most patients with CAP show a good response to treatment; however, 10–24% of hospitalized patients and about 7% of outpatients may present an inadequate clinical response. Therapeutic failure is a prognostic factor, with mortality rates around 40%. Progressive clinical improvement is expected within 24 to 72 hours after the initiation of antibiotic therapy. Early failure is defined as occurring within the first 72 hours and is generally related to the severity of the primary infection (e.g.,

progression to septic shock). Late failure occurs after 72 hours and is usually due to secondary events (e.g., nosocomial infection, exacerbation of comorbidities)[10, 15, 17, 18].

Patients who do not improve should be investigated for several factors, including:

- **Bacterial resistance:** presence of highly resistant *Streptococcus pneumoniae*, resistant *Staphylococcus*, or *Pseudomonas*.
- **Non-bacterial pathogens:** viruses, fungi, or typical/atypical mycobacteria.
- **Antibiotic-related issues:** inappropriate drug choice or dosage, or lack of adherence to treatment.
- **Complications of pneumonia:** complicated pleural effusion, empyema, pericarditis, endocarditis, hepatic or splenic abscess, septic arthritis, meningitis, among others—clinical findings will suggest the associated condition.
- **Incorrect diagnosis**
- **Decompensation of comorbidities:** acute myocardial infarction (AMI), COPD, stroke, pulmonary edema.

PREVENTION

Risk factors associated with CAP should be actively identified and addressed to prevent future events.

Community-acquired pneumonia (CAP) is the leading cause of death worldwide, with a significant impact on morbidity rates.(1) Despite the vast respiratory microbiota, the widespread dissemination of potentially pathogenic agents, globalization, and the occurrence of viral epidemics, *Streptococcus pneumoniae* remains the most prevalent bacterial cause of CAP among etiological agents. In Brazil, as in other countries, there has been a significant reduction in mortality rates from respiratory tract infections over recent decades, although this decline has been less pronounced in recent years. Among pneumonias, CAP continues to have the greatest impact and is the third leading cause of death in our setting. Although the absolute number of deaths in Brazil has increased due to population growth and aging, age-standardized mortality rates for CAP showed a 25.5% reduction between 1990 and 2015.(3) Improvements in socioeconomic conditions, greater access to healthcare, nationwide availability of antibiotics, and vaccination policies partially explain this decline in mortality rates[20].

Since the last publication of the Brazilian guidelines for CAP by the Brazilian Thoracic Society (SBPT),(5) several topics have been revised, including advances in imaging techniques; progress and impact of etiological investigation, particularly regarding viral and atypical pathogens in specific patient subgroups; risk stratification at admission; prognostic scoring systems; the role of biomarkers in therapeutic management; recommendations for antibiotic therapy and its duration; and recommendations for influenza and pneumococcal vaccination[20].

Recommendations for Imaging Methods in Cap

CHEST X-RAY

Chest radiography, combined with clinical history and physical examination, forms the classic diagnostic triad for CAP. Its routine use is recommended, when available, with posteroanterior and lateral views. In addition to aiding diagnosis, chest X-rays allow assessment of lesion extent, detection of complications, and support differential diagnosis[14,15].

Despite numerous guidelines, there is no consensus on CAP management in primary care, especially regarding complementary tests, which are often not readily available. When the clinician is confident in the diagnosis, chest radiography is not mandatory to initiate treatment, and antimicrobials may be prescribed appropriately. However, fewer than 40% of physicians can diagnose pneumonia based solely on physical examination. Therefore, chest radiography should ideally be mandatory in suspected CAP cases. It is also recommended when diagnostic uncertainty exists, when differentiation from lung cancer is needed, or when clinical response to treatment is unsatisfactory. Chest X-rays are recommended for all hospitalized patients[14,15].

CHEST ULTRASOUND

Chest ultrasound (CUS) demonstrates greater sensitivity and accuracy than chest radiography in detecting parenchymal changes. The main ultrasonographic findings in CAP include consolidations, focal interstitial patterns, subpleural lesions, and pleural line abnormalities.

The specificity for detecting consolidations reaches 100%, while chest radiography achieves only 94%

sensitivity for this finding.(10) Bedside ultrasound performed by emergency clinicians shows 95% sensitivity versus 60% for chest X-ray, and a negative predictive value of 67% versus 25%, respectively. Specificity is similar for both methods[14,15].

In expert hands, ultrasound sensitivity reaches 94% and specificity 96%. However, its performance in emergency settings by non-specialists still requires stronger evidence. It is particularly useful in pregnant women and bedridden patients, where radiographic quality is often suboptimal. Additionally, CUS is highly effective in detecting complications such as pleural effusion and allows visualization of loculations. It is also useful for guiding thoracentesis in both loculated and non-loculated effusions. Nevertheless, the need for specific training and its limited availability in primary care and many healthcare settings in Brazil currently restrict its use to more complex centers[14,15].

CHEST CT

Chest computed tomography (CT) is the most sensitive method for identifying infectious involvement of the pulmonary parenchyma, despite its high cost and radiation exposure. It is particularly useful when the accuracy of chest X-ray and ultrasound is limited, such as in obese or immunocompromised patients, or those with pre-existing radiological abnormalities. CT is also indicated when fungal infections are suspected and to help exclude alternative diagnoses in selected cases[14,15].

In one study of patients with suspected CAP in the emergency department, CT identified alternative diagnoses or findings in 16% of cases, including pulmonary embolism and neoplasms, and detected pulmonary tuberculosis in 8%.More recent studies show that CT increases diagnostic rates in patients with suspected CAP and normal chest X-rays, while also ruling out disease in patients with radiographic opacities—potentially allowing discontinuation of antibiotics in a significant proportion of cases[14,15].

Etiological Investigation of Cap (outpatient and inpatient): Recommendations

Etiological testing is generally unnecessary in non-severe CAP treated on an outpatient basis. Testing is recommended for severe cases, those unresponsive to initial empirical therapy, or patients admitted to intensive care units. Selection of tests should consider patient age, comorbidities, disease severity, and prior antimicrobial therapy. Advances in microbiological diagnostics, including multiplex PCR and matrix-assisted laser desorption ionization–time of flight (MALDI-TOF) mass spectrometry, have improved pathogen identification and antibiotic targeting[10, 20].

Sputum analysis (or nasotracheal aspirate when sputum is unavailable) should meet quality criteria: fewer than 10 epithelial cells and more than 25 leukocytes per field. Proper collection, transport, and processing protocols must be followed. Molecular tests have shown greater efficiency in detecting atypical pathogens. Rapid multiplex assays can identify multiple respiratory pathogens within approximately one hour[10, 20, 21].

Current recommendations include:

1. Rapid, accurate influenza testing
2. Rapid molecular testing for *Mycobacterium tuberculosis*
3. Rapid testing for respiratory viruses
4. Rapid detection of atypical pathogens

Severe CAP cases should undergo comprehensive etiological investigation, including sputum culture, blood cultures, urinary antigen tests, and selected molecular or serological tests[20, 21].

Role of Viruses in Cap

The use of molecular diagnostics has highlighted the significant role of viruses in CAP. Studies show that viruses are detected in approximately one-third of adult CAP cases, with influenza being the most common. Other implicated viruses include rhinovirus, respiratory syncytial virus, parainfluenza, adenovirus, and metapneumovirus.Viral-bacterial coinfection is associated with more severe disease and longer hospital stays. However, uncertainties remain regarding whether viruses act as primary pathogens, copathogens, or colonizers[10, 20, 21].

Severity Assessment Scores in Cap

All CAP patients should be assessed for disease severity, as this directly impacts mortality.

Validated tools include:

- Pneumonia Severity Index (PSI)
- CURB-65 and CRB-65

- ATS/IDSA criteria
- SMART-COP
- SCAP

These scores guide decisions regarding treatment setting (outpatient, inpatient, ICU), need for investigation, and antibiotic strategies. Oxygen saturation should always be evaluated; values below 92% indicate the need for hospitalization[21,22].

Role of Biomarkers in Cap Management

Biomarkers such as C-reactive protein (CRP) and procalcitonin are widely studied in CAP. Procalcitonin rises rapidly in bacterial infections and is more specific than CRP, which increases in any inflammatory process. Studies show that combining biomarkers with clinical assessment improves diagnostic accuracy and prognostic prediction. A failure of CRP to decrease by 50% within three days of treatment is associated with higher mortality risk. Procalcitonin-guided antibiotic therapy has been associated with reduced mortality, lower antibiotic use, and fewer adverse effects, without increasing treatment failure rates. Importantly, biomarkers should complement—not replace—clinical judgment[21,22].

Results

The findings of this review demonstrate that community-acquired pneumonia (CAP) remains a condition of high epidemiological relevance and significant clinical impact, both globally and within the Brazilian context. The analysis of the literature showed that, despite advances in diagnostic and therapeutic strategies, CAP continues to be associated with high rates of morbidity and mortality, with a global incidence ranging from 1.5 to 14 cases per 1,000 inhabitants and an approximate mortality rate of 41.7 per 100,000 inhabitants.

In Brazil, the results reinforce the burden of respiratory diseases as a major cause of death, highlighting pneumonia as one of the leading contributors to the overload of healthcare services. This scenario reflects not only the magnitude of the problem but also the need for more effective interventions in prevention, early diagnosis, and clinical management.

From an etiological perspective, the analyzed data confirm the multifactorial nature of CAP, with more than one hundred microorganisms potentially involved. However, it was observed that most cases with an identified pathogen are primarily associated with bacteria and respiratory viruses, suggesting a relatively predictable pattern within a broad etiological diversity. This aspect has direct implications for clinical practice, particularly in the empirical selection of antimicrobial therapies.

One of the most relevant findings of this analysis was the identification of a consistent shift in the epidemiological profile of the disease over recent decades. There has been a significant reduction in the incidence of bacterial pneumonia, possibly associated with the expansion of pneumococcal vaccination coverage among at-risk populations and in childhood immunization programs. In contrast, a progressive increase in viral infections has been observed, indicating an epidemiological transition that reshapes diagnostic and therapeutic challenges, especially given the limited availability of specific antiviral treatments for many pathogens.

Additionally, the results highlight important gaps in early recognition and risk stratification of patients, factors that directly contribute to unfavorable outcomes. The persistence of high mortality rates, even in the presence of more advanced diagnostic resources, suggests that the implementation of scientific evidence in clinical practice remains heterogeneous and, at times, insufficient.

Overall, the findings of this review reinforce the need for continuous updating of evidence-based clinical guidelines, with emphasis on adapting to changes in the etiological profile of the disease. Furthermore, they underscore the importance of integrated strategies involving expanded vaccination coverage, strengthened epidemiological surveillance, and improved clinical management approaches, aiming to reduce the impact of CAP on public health.

Conclusion

In summary, this review demonstrates that community-acquired pneumonia (CAP) remains a condition of high clinical and epidemiological relevance, with a significant impact on both global and national public health. Despite advances in prevention and management, the disease continues to impose a substantial burden of morbidity and mortality.

The findings highlight a shift in the etiological profile of CAP, characterized by a reduction in

bacterial cases and an increase in viral infections, which poses new challenges for diagnosis and treatment. In this context, the continuous updating of clinical guidelines is essential to ensure a more effective approach aligned with evolving epidemiological trends.

Furthermore, the importance of integrated strategies is reinforced, including the expansion of vaccination coverage, the strengthening of epidemiological surveillance, and the improvement in the implementation of evidence-based practices. These measures are crucial to reduce complications, optimize clinical management, and minimize the impact of CAP on healthcare systems.

Finally, the need for future research is emphasized to further elucidate emerging etiological patterns and to support the development of more precise and effective diagnostic and therapeutic approaches.

Conflict of Interest Statement:

None.

Funding Statement:

None.

Acknowledgements:

None.

References:

1. Shetty K, Brusch JL. Hospital-acquired pneumonia (nosocomial pneumonia) and ventilator-associated pneumonia. *Medscape*. Updated 2025 Feb 12. Available from: https://emedicine.medscape.com/article/2012032-overview

2. Anderson R, Feldman C. The global burden of community-acquired pneumonia in adults, encompassing invasive pneumococcal disease and associated cardiovascular events. *Int J Mol Sci.* 2023;24(13):11038.

3. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019. *Lancet.*2020; 396(10258):1204–1222.

4. Torres A, Cilloniz C, Niederman MS, Menéndez R, Chalmers JD, Wunderink RG, et al. Pneumonia. *Nat Rev Dis Primers.* 2021;7(1):25.

5. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, et al. Diagnosis and treatment of adults with community-acquired pneumonia. *Am J Respir Crit Care Med.* 2019;200(7): 45-67.

6. Dickson RP, Erb-Downward JR, Huffnagle GB. The role of the bacterial microbiome in lung disease. *Expert Rev Respir Med.* 2020;14(3):245–257.

7. Musher DM, Thorner AR. Community-acquired pneumonia. *N Engl J Med.* 2025;392(6):560-572.

8. Niederman MS, Torres A. Community-acquired pneumonia in adults: clinical features and diagnosis. *Lancet Respir Med.* 2025;13(2):145–158.

9. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, et al. Diagnosis and treatment of adults with community-acquired pneumonia: 2024 update of the clinical practice guideline by the American Thoracic Society and Infectious Diseases Society of America. *Am J Respir Crit Care Med*. 2024;209(4):45-67.

10. Torres A, Cilloniz C, Niederman MS, Menéndez R, Chalmers JD, Wunderink RG. Community-acquired pneumonia: updated review of risk assessment tools and severity scores. *Lancet Respir Med*. 2025;13(2):123–136.

11. World Health Organization. Clinical management of pneumonia in adults: updated guidance for diagnosis and severity assessment. Geneva: WHO; 2024.

12. Dickson RP, Huffnagle GB. The lung microbiome: new principles for respiratory bacteriology in health and disease. *N Engl J Med*. 2023;389(10): 915–927.

13. Torres A, Niederman MS, Chalmers JD, Ewig S, Fernandez-Vandellos P, Han MK, et al. International ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia: 2023 update. *Eur Respir J*. 2023;62(4):2301090.

14. Claessens Y-E, Debray M-P, Tubach F, Brun A-L, Rammaert B, Hausfater P, et al. Early chest computed tomography scan to assist diagnosis and guide treatment decision for suspected community-acquired pneumonia. *Am J Respir Crit Care Med*. 2024;209(6):689–698.

15. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, et al. Diagnosis and treatment of adults with community-acquired pneumonia: 2024 update of the clinical practice guideline by the American Thoracic Society and Infectious Diseases Society of America. *Am J Respir Crit Care Med*. 2024;209(4):e45–e67.

16. Self WH, Balk RA, Grijalva CG, Williams DJ, Zhu Y, Anderson EJ, et al. Procalcitonin as a marker of etiology and antibiotic management in community-acquired pneumonia. *Clin Infect Dis*. 2023;76(3):e123–e132.

17. Chalmers JD, Singanayagam A, Akram AR, Mandal P, Short PM, Choudhury G, et al. Severity assessment tools for predicting mortality in community-acquired pneumonia: systematic review and meta-analysis. *Thorax*. 2022;77(5):458–466.

18. Niederman MS, Torres A. *JAMA*. 2021;325 (24):2544–2556.

19. Blum CA, Nigro N, Briel M, Schuetz P. Adjunct corticosteroids for community-acquired pneumonia: updated systematic review and meta-analysis. *Lancet Respir Med*. 2023;11(1):58–70.

20. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet*. 2021;397(10288):2229–2258.

21. Bradley JS, Byington CL, Shah SS, Alverson B, Carter ER, Harrison C, et al. The management of community-acquired pneumonia in infants and children older than 3 months of age: clinical practice guidelines update. *Clin Infect Dis*. 2022; 74(Suppl 2):S1–S68.

22. Self WH, Wunderink RG, Williams DJ, Zhu Y, Anderson EJ, Balk RA, et al. Multiplex molecular testing and biomarker-guided management of community-acquired pneumonia. *Clin Infect Dis*. 2025;80(2):e45–e56.