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Community acquired pneumonia

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Abstract

Community-acquired pneumonia remains a major cause of morbidity and mortality worldwide, particularly among vulnerable populations. Despite advances in healthcare, it continues to challenge clinicians due to evolving pathogens, rising antibiotic resistance, and changing patient demographics. While *Streptococcus pneumoniae* is the commonest reported causative organism, polymicrobial infections and viral-bacterial co-infections are increasingly recognized, especially following influenza or SARS-CoV-2 infections.

In older adults, typical features of pneumonia such as fever and cough may be absent, thereby delaying the diagnosis and treatment. Chest radiographs often demonstrate air space consolidations, classically of a lobar pattern in pneumococcal infections, while interstitial patterns are seen in other causes. Pneumonia Severity Index (PSI) and CURB-65 are two validated tools for assessing the severity of community-acquired pneumonia. Treatment is usually empirical initially, based on likely pathogens and local resistance patterns. Patients not responding to antibiotics should be evaluated for complications of pneumonia or other pathogens like fungi.

Early diagnosis, appropriate empirical antibiotic therapy, antibiotic stewardship and supportive measures are the key to reducing mortality and improving patient outcomes.

Key words: community-acquired pneumonia, pneumonia, CAP, lower respiratory tract infection

Introduction

Community-acquired pneumonia (CAP) is a significant cause of morbidity and mortality worldwide, especially among vulnerable populations such as the elderly, patients with co-morbidities, and the immunocompromised. It is defined as an acute infection of the pulmonary parenchyma acquired outside of hospital[1] Despite advances in vaccination, antimicrobial therapy, and public health measures, CAP continues to challenge clinicians due to evolving microbial patterns, increasing antibiotic resistance, and changing patient demographics.[2]

The global burden of CAP is substantial. According to the World Health Organisation, lower respiratory tract infections, including CAP, remain among the leading causes of mortality worldwide, especially in low- and middle-income countries.[3] Hospitalisation rates and the health-care costs associated with CAP are significant, emphasising the need for timely diagnosis and appropriate management.[4] Sri Lanka faces specific challenges such as inappropriate use of antibiotics, inadequate vaccination coverage of high-risk groups and limited access to advanced diagnostic modalities, especially in peripheral health-care settings.

This article provides a comprehensive review of the epidemiology, clinical presentation, diagnostic strategies, and treatment approaches in line with antimicrobial stewardship in CAP management.

Epidemiology and risk factors

The incidence of CAP varies with age, geographic

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region, and healthcare accessibility. In developed countries, the annual incidence is estimated at 5 to 11 cases per 1,000 adults, with higher rates in individuals over 65 years of age.[5] In Sri Lanka and other South Asian countries, reliable population-based incidence data are limited, but hospital-based studies suggest a significant morbidity and mortality due to CAP. Common risk factors include advanced age, smoking, chronic obstructive pulmonary disease (COPD), diabetes mellitus, chronic kidney disease, immunosuppression, malnutrition and recent viral infections (e.g., influenza or COVID-19).[2]

Aetiology

The aetiology of CAP is diverse, with causative pathogens varying according to the patient's age, co-morbidities, and severity of illness.[1] Common causative organisms include *Streptococcus pneumoniae* (the most common cause), *Haemophilus influenzae*, *Moraxella catarrhalis, Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumophila,* and respiratory viruses (Influenza A/B, RSV, SARS-Cov-2).[6] In Sri Lanka, studies indicate that *Streptococcus pneumoniae* and *Haemophilus influenzae* remain predominant pathogens and emerging resistance is a concern.[7] Therefore, national-level surveillance and antibiotic stewardship programs are needed to address the problem of rising antibiotic resistance.

Polymicrobial infections and co-infections with viral and bacterial pathogens are increasingly recognised, particularly following influenza or SARS-CoV-2 infections. In the elderly, immunocompromised individuals and patients with recurrent hospital admissions, gram-negative bacilli such as *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*, along with *Staphylococcus aureus* (including MRSA), should be considered. Atypical bacterial pneumonia often originates from a range of environmental and zoonotic sources. Atypical organisms causing CAP include *Mycoplasma pneumoniae*, *Chlamydophila psittaci, Legionella pneumophila,* and *Coxiella burnetii*, which are not detectable by routine cultures and often require specialised investigations such as serology or PCR.[8]

Clinical features

Symptoms of pneumonia include acute onset of fever, productive cough with purulent sputum, pleuritic chest pain, dyspnoea and tachypnoea. However, atypical presentations, especially in the elderly and in immunocompromised individuals, may manifest with non-specific symptoms such as confusion or delirium, generalised weakness and anorexia. Typical features of pneumonia, such as fever and cough, may be absent in older adults, potentially delaying the diagnosis and initiation of treatment.[9]

Atypical pneumonia usually presents with more prolonged and nonspecific symptoms compared to typical bacterial pneumonia. Common symptoms include sore throat, headache, dry non-productive cough, low-grade fever and myalgia. Extrapulmonary manifestations, such as rash, gastrointestinal symptoms, or neurological signs, may also be present depending on the causative organism.

Physical examination in lobar pneumonia may reveal dullness to percussion, reduced breath sounds, coarse crepitations and bronchial breathing over affected areas of the lung. In bronchopneumonia, findings would be more diffuse with bilateral coarse crepitations on auscultation. Physical examination in atypical pneumonias may be unremarkable despite significant infiltrates on chest imaging.

Investigations and diagnosis

Timely diagnosis is crucial for initiating appropriate treatment and improving outcomes.[1] Neutrophil leucocytosis, elevated C-reactive protein (CRP) and raised blood urea levels are some of the expected laboratory findings.

Chest X-ray, an immediate and primary investigation for suspected CAP, will demonstrate the presence of pulmonary infiltrates, a hallmark of pneumonia. Chest X-ray has a high sensitivity, ranging from 70% to 90%, in detecting such infiltrates. However, its specificity is lower, as other conditions like atelectasis, pulmonary oedema, or malignancies may mimic changes of pneumonia, leading to potential false positives. The presence of cavities in the chest radiograph should raise suspicion of pulmonary tuberculosis,[10] *Klebsiella spp*, *Pseudomonas spp*, or *Staphylococcus aureus* infections. Further, CXR cannot reliably differentiate between bacterial and viral aetiologies.[11]

Sputum Gram stain offers rapid identification of bacterial morphology, while culture provides definitive identification and information on antimicrobial susceptibility. However, the sensitivity of sputum culture varies from 30 to 70% and is affected by sample quality and prior antibiotic use. Blood cultures, usually carried out in patients with severe CAP, though specific for detecting bacterial pathogens, have a relatively low sensitivity, with only 10-15% yielding positive results.

It should be noted that routine sputum Gram stain, sputum culture, and blood cultures are not recommended on all patients with CAP, unless they fall into one of the following categories.[1]

- Severe CAP[5]
- Being empirically treated for Methicillin-Resistant *Staphylococcus aureus* (MRSA) or *P. aeruginosa*
- Previously infected with MRSA or *P. aeruginosa*
- Presence of chronic respiratory co-morbidities

Routine testing of urinary legionella and pneumococcal antigen is also not advocated, unless indicated by epidemiological evidence, such as a legionella outbreak, recent travel, or in severe CAP. Testing for influenza can be performed during outbreaks using a rapid influenza antigen or molecular assay such as an influenza nucleic acid amplification test.[1]

Molecular diagnostics have revolutionised pathogen identification. The BioFire FilmArray Respiratory Panel is a multiplex PCR that can rapidly detect multiple viral and atypical bacterial pathogens from a nasopharyngeal swab with high sensitivity and specificity. Therefore, it has a significant impact on antimicrobial stewardship by guiding specific therapy.[12] However, the detection of genetic material does not always equate to active infection, as it may reflect colonisation.

The testing for galactomannan, a cell wall component of *Aspergillus* species, in the management of CAP is extremely limited and not recommended as a routine investigation. Galactomannan serves as a biomarker for invasive aspergillosis, a severe fungal infection that typically occurs in highly immunocompromised patients.

Severity assessment scores

In addition to clinical judgement, it is recommended that clinicians use a validated prediction tool such as Pneumonia Severity Index (PSI) or CURB-65 to assess the severity of pneumonia for prognostication and to make decisions on hospital admissions. Both tools were developed as prognostic models in immunocompetent patients with pneumonia. The PSI is more complex than CURB-65 but provides greater prognostic accuracy and better discriminative power in predicting mortality.[13]

CURB-65 comprises 5 criteria, with one point given for each positive criterion.

C – Confusion (new disorientation to person, place, or time)

U – Urea > 7 mmol/L (or BUN > 20 mg/dL)

R – Respiratory rate ≥ 30 breaths/min

B – Blood pressure: systolic < 90 mmHg or diastolic ≤ 60 mmHg

65 – Age ≥ 65 years

CURB-65 scores of 0-1, 2, and 3-5 indicate low, moderate and high risk, respectively.[14]

Further, CRB-65, a simplified version which exclude laboratory results has been introduced as a more practical score in the clinical setting. It has been shown that the CRB-65 has a high correlation to CURB 65 and widely recommended by the international authorities to be used in the routine clinical practice.[15] Scores of 0, 1-2 and above 3 correspond to low, moderate and high risk, respectively.

The CURB-65 score is valued for its simplicity and ease of use at the bedside but is limited by its reliance on only five binary variables. This can lead to inadequate risk stratification, particularly in intermediate-risk patients, and overlooks key factors like comorbidities, detailed lab values, and imaging findings. As a result, it may underestimate severity in some younger but seriously ill patients.

Pneumonia Severity Index (PSI) is more comprehensive, incorporating 20 variables across multiple domains. While better at identifying low-risk patients for outpatient care, PSI is complex and time-consuming, making it less practical in urgent settings. The criteria assessed, and their corresponding score is given in Table 1.

The risk stratification of CAP according to the PSI score, the 30-day mortality and recommended place of treatment is summarized in Table 2.[13]

Two multi-centre, cluster-randomised trials demonstrated that use of the PSI safely increases the proportion of patients who can be treated in the outpatient setting.[13,14] In comparison to the PSI, there is less evidence that CURB-65 is effective as a decision aid in guiding the initial facility of treatment, thus PSI score is preferred over CURB-65 in to be used as an aid to decide on in-patient versus outpatient treatment of CAP.[1,16]

Table 1. Pneumonia severity index (PSI) calculation

Factor	Score
Demographic factors	
Male	Age
Female	Age - 10
Long-term care	+10
Accompanying disease	
Neoplastic disease	+30
Liver disease	+20
Congestive heart failure	+10
Cerebrovascular disease	+10
Chronic kidney disease	+10
Symptoms at diagnosis	
Acute psychosis	+20
Breathing rate ≥30/min	+20
Systolic pressure < 90 mmHg	+15
Body temperature < 35°C or ≥40°C	+15
Heart rate ≥125/min	+10
Laboratory measurements	
Arterial blood pH < 7.35	+30
BUN ≥30 mg/dL	+20
Serum sodium < 130 mEq/L	+20
Serum glucose > 250 mg/dL	+10
Hb < 9 gm/dL (haematocrit < 30%)	+10
Atmospheric arterial blood gas (PaO2) < 60 mmHg (SaO2 < 90%)	+10

Treatment

Antibiotics: Treatment is usually initiated empirically, guided by likely pathogens and local resistance patterns. The empirical antibiotics according to the severity of CAP as recommended in the National Guideline for Empirical and Prophylactic Use of Antimicrobials 2024 (Sri Lanka) is summarized in Tables 3 and 4.[17]

Table 2. Risk stratification according to PSI

PSI Risk Class	Score Range	Risk Level	30 - Day Mortality	Recommended Place Treatment
I	Clinical criteria only (Age < 50, no comorbidities or abnormal vitals)	Very Low Risk	< 0.1%	Outpatient care
II	≤70	Low Risk	< 0.6%	Outpatient care
III	71-90	Moderate Risk	0.9%-2.8%	Observation or short inpatient stay
IV	91-130	High Risk	8.2%-9.3%	Inpatient care
V	> 130	Very High Risk	27%-31%	Inpatient care ± ICU

Table 3. Empirical antibiotic treatment for Mild and Moderate CAP (National Guideline for Empirical and Prophylactic Use of Antimicrobials 2024 (Sri Lanka))

Condition	Primary therapy	Alternative therapy / immediate beta-lactam hypersensitivity
• **Mild (CURB 65=0-1)** a) No comorbidities: (out-patient treatment)	Amoxicillin 500mg - 1g PO q8h or cefuroxime 500mg PO q12h Duration: 5 days	Clarithromycin 500mg PO q12h or erythromycin 500mg PO q6h (for use in pregnancy) or doxycycline 200mg PO (first dose) followed by 100mg PO q24h Duration: 5 days
• **Mild (CURB 65=0-1)** b) With comorbidities: alcoholism, COPD, bronchiectasis, DM (out-patient treatment)	Co-amoxiclav 625mg PO q8h/ cefuroxime 500mg PO q12h + clarithromycin 500mg PO q12h Duration: minimum of 5 days	Clarithromycin 500mg PO q12h Duration: minimum of 5 days
• **Moderate (CURB 65=2)** (requires hospitalization)	co-amoxiclav 1.2g IV q8h/ cefuroxime 1.5g IV q8h + clarithromycin 500mg PO q12h	levofloxacin 750mg PO/IV q24h (after exclusion of tuberculosis)

Table 4. Empirical antibiotic treatment for Severe CAP (National Guideline for Empirical and Prophylactic Use of Antimicrobials 2024 (Sri Lanka))

Condition	Primary therapy	Alternative therapy / immediate beta-lactam hypersensitivity
• **Severe (CURB 65=3-5)** May need ICU admission.	clarithromycin 500 mg IV/PO q12h or cefotaxime 1-2 g IV q8h ceftriaxone 1-2 g IV q24h + clarithromycin 500 mg IV/PO q12h	levofloxacin IV 500 mg q12h (after exclusion of tuberculosis)
• **For suspected community** acquired MRSA (CA - MRSA) pneumonia	Add vancomycin 1 g IV q12h	Add teicoplanin 400 mg IV q12h for 3 doses then 400 mg IV q24h
• **With risk factors for Pseudomonas:** • Cystic fibrosis • COPD • Bronchiectasis	piperacillin-tazobactam 4.5 g IV q6 - 8h or meropenem 1 g IV q8h or imipenem 500 mg IV q6h	ciprofloxacin 400 mg IV q12h + gentamicin 5-7 mg/kg IV q24h

A minimum of 5 days of treatment is recommended and the total duration of antibiotics should be guided by signs of clinical improvement and stability.[1] Prolonged antibiotic therapy may be required for slow responders, infection with resistant organisms or patients with complications.

It is important to note that quinolones such as levofloxacin and moxifloxacin are categorised as a 'reserve antibiotics' under Access, Watch, and Reserve (AWaRe) classification of antibiotics and should not be used unless absolutely indicated, This would help to minimize development of antimicrobial resistance.[18] Additionally, routine use of anaerobic cover for suspected aspiration pneumonia is not recommended unless there is a suspicion of lung abscess or empyema.[1]

For patients with CAP who test positive for influenza, treatment with oseltamivir is recommended regardless of duration of illness, although the greatest benefits are seen when therapy is started within 48 hours of symptom onset.[19] In addition, they should be given standard antibacterial treatment in both inpatient and outpatient settings to address the possibility of concomitant bacterial infection.[1]

Glucocorticoids: Routine administration of glucocorticoids as an adjunct therapy in the treatment of CAP is not recommended. Clinical Practice Guideline

of the American Thoracic Society and Infectious Diseases Society of America 2019 recommends consideration of addition of glucocorticoids for patients with CAP and refractory septic shock, endorsing the Surviving Sepsis Campaign recommendations.[1] However, 'Community-Acquired Pneumonia: Evaluation of Corticosteroids' (CAPE COD) trial has shown that early treatment with hydrocortisone at a dose of 200mg per day reduced the mortality at 28 days among patients admitted to intensive care unit (ICU) for severe CAP compared to the placebo.[20]

Other supportive measures: Adjunctive measures a such as oxygen therapy for hypoxaemia, IV fluids for dehydration, antipyretics for fever and chest physiotherapy may be indicated.[1]

Patients with retained secretions, impaired airway clearance, atelectasis, weak cough, or on mechanical ventilation may benefit from chest physiotherapy. Potential benefits may include slightly reduced hospital stay, duration of fever, and ICU days in select cases.[21] Nebulised saline may help to loosen thick, viscous secretions, particularly in ventilated or severely ill patients, though strong evidence is limited.[22] Nebulised mucolytics like N-acetyl cysteine might aid mucous clearance enabling easier ventilation management, especially in critical care setting, but also lack robust supporting data.[2] These therapies should be considered judiciously on a case-by-case basis, especially in critical care settings.

Complications

Complications of CAP include parapneumonic effusions, empyema, lung abscess, septic shock, sepsis with multi-organ failure and respiratory failure.[1] Patients who are not responding to antibiotics should be evaluated for empyema, lung abscess, pulmonary tuberculosis or invasive fungal infections. These complications require specific management which is beyond the scope of this article.

Prevention

Individuals who are at risk of CAP such as patients with chronic lung diseases (COPD, bron-chiectasis) will benefit from pneumococcal (PSV - 23), annual influenza and COVID-19 vaccinations. While pneumococcal vaccines have some efficacy in preventing vaccine-strain pneumococcal pneumonia and invasive disease, they will not prevent all types of CAP.[24] Annual influenza vaccination has been shown to reduce pneumonia, hospitalizations, and cardiac events in certain groups.[25]

Additional measures such as cessation of smoking, optimal control of underlying chronic illnesses like diabetes mellitus, maintaining proper hand hygiene and respiratory etiquette are helpful to reduce the incidence of CAP among vulnerable populations.[11]

Conclusion

CAP is a common and potentially life-threatening illness, particularly in the elderly and those with co-morbidities. Early diagnosis, appropriate empirical treatment, antibiotic stewardship and supportive care are essential in improving patient outcomes. With evolving pathogens and antibiotic resistance, continuous adaptation of clinical practice based on the current evidence and adhering to antibiotic stewardship principles are vital.

Key Messages

- CAP is a significant cause of morbidity and mortality, especially among vulnerable populations and typical clinical features can be absent in these groups leading to to delays in the diagnosis and treatment.
- Routine testing of sputum for Gram stain, sputum culture and blood culture on all patients with CAP is not recommended, unless they have severe CAP, chronic respiratory co-morbidities or being empirically treated for or previously infected with MRSA or *P. aeruginosa.*
- Pneumonia Severity Index (PSI) and CURB-65 are recommended to be used for severity assessment of CAP and to guide clinical decisions at the outset.
- Initial empirical antibiotic treatment should be based on likely pathogens and local resistance patterns.
- Respiratory quinolones like levofloxacin and moxifloxacin should not be used to treat CAP unless absolutely indicated.

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