Community-acquired pneumonia

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Abstract

Community-acquired pneumonia (CAP) is a common inflammatory process contained within the lungs in response to infection with non-hospital pathogens. Full resolution usually occurs with appropriate antimicrobial therapy. A significant proportion of patients develop severe CAP where there is failure to contain the local immune response and these patients may require admission to the intensive care unit (ICU). The CURB-65 severity score is a rapid, objective way of predicting mortality and can be used to guide site of care decisions in conjunction with clinical assessment. Microbiological investigations permit pathogen-specific antibiotic therapy and provide epidemiological data. Appropriate and timely administration of antibiotics is the mainstay of treatment. Complications include empyema, treatment failure, sepsis, respiratory failure and death.

Keywords Antimicrobial therapy; community-acquired pneumonia; intensive care; severity scores; microbiological investigation; treatment failure

Royal College of Anaesthetists CPD Matrix: 2A12, 2C01, 2C03, 3C00

Learning objectives

After reading this article, you should be able to:

- identify clinical markers in patients with severe community-acquired pneumonia (CAP) who may require intensive care admission
- list appropriate investigations
- describe specific treatment regimes
- describe complications of CAP

Community-acquired pneumonia (CAP) is managed as an outpatient in greater than 50% of cases. The incidence of hospitalization is approximately 1.1–4 per 1000 population. The proportion of admitted patients who require ICU management for CAP is approximately 6% and is increasing. In England, Wales and Northern Ireland there were 13 patients admitted annually per ICU in 1996. This increased to 30 patients per ICU in 2004. In the UK, mortality in the community is less than 1%, while the mortality of patients admitted to ICU is over 30%.[1]

CAP is usually caused by bacteria but primary viral infection may precede the bacterial pneumonia. The presentation and treatment of CAP in immunosuppressed patients (cystic fibrosis, HIV and malignancy) differs and will not be considered here.

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The defining features of CAP in hospital patients are:

- presentation with cough and new focal chest signs on examination
- at least one systemic feature of sweating, shivers, malaise and/or temperature of 38°C or more
- new shadowing on chest X-ray for which there is no other explanation (i.e. pulmonary oedema or infarction)
- patient presents with the above as the primary reason for attendance and is managed as pneumonia.[1]

Aetiology

The frequency with which different organisms cause CAP in hospital and in the ICU is shown in Figure 1. 'Atypical' pathogens are classically *Legionella pneumophiliae*, *Mycoplasma pneumonia* and *Chlamydia pneumonia*. Less common pathogens are *Haemophilus influenzae*, Gram-negative enteric bacilli, *Chlamydophila psittaci* and *Coxiella burnetii*. The term atypical is unhelpful and should be abandoned as it incorrectly implies that there is a characteristic clinical presentation for patients with these organisms.

Clinical features

In the majority of cases CAP presents as an acute illness with cough, purulent sputum and fever together with physical signs or radiological changes consistent with lung consolidation. Patients can rapidly deteriorate from respiratory failure and/or sepsis. This may not be recognized in younger patients due their ability to compensate for pending organ failure. Although certain symptoms and sign are more common with specific organisms none allow accurate diagnosis. Importantly, legionella can occur in outbreaks and mycoplasma in epidemics separated by 4 years.

Scoring systems

Several scoring systems have been developed to predict mortality. The CURB65 severity score is the simplest and has been validated (Table 1). British Thoracic Society guidelines recommend referral to ICU in patients presenting with a score of 4–5.[1]

The SMART-COP scoring system was developed in Australia to predict the need for invasive ventilation. This acronym represents systolic blood pressure below 90 mmHg, multilobar infiltrates, albumin less than 35 g/litre, raised respiratory rate (>25 for those age <50 years, and >30 for those age >50 years), tachycardia (>125/minute), confusion, low oxygen (<9 kPa (68 mmHg) if age <50 or <8 kPa (60 mmHg) if age >50), and arterial pH <7.35. The abnormalities in systolic blood pressure, oxygenation and arterial pH each received two points, while the

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Percentage frequency with which organisms cause community-acquired pneumonia in hospital and intensive care units in the UK

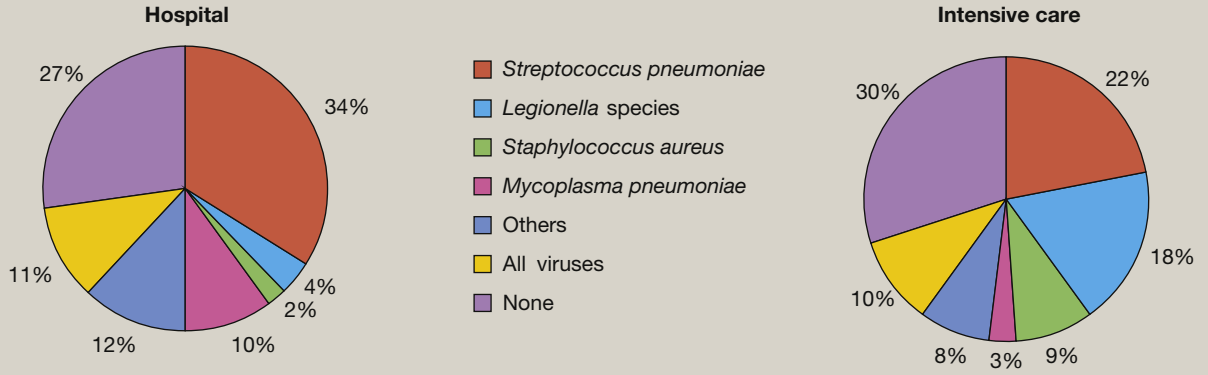

Figure 1

CURB65 severity score and predicted mortality

One point for each feature presenting:

- **C**onfusion
- **U**rea >7 mmol/litre
- **R**espiratory rate ≥30/minute
- **B**lood pressure (SBP <90 or DBP ≤60 mmHg)
- **A**ge >65

CURB65 score	Severity	Risk of death (%)
0–1	Low	<3
2	Moderate	9
3–5	High	15–40

DBP, diastolic blood pressure; SBP, systolic blood pressure.

Table 1

five other criteria received one point each. With three or more points the need for invasive ventilation was predicted with a sensitivity of 92% and specificity of 62%. The CURB65 was less sensitive (39%) but more specific (74%) at predicting the need for invasive ventilation.[2]

The PIRO score, (Predisposition, Insult, Response Organ dysfunction) is used to assess severity of illness in ICU patients. There are eight variables: comorbidities (chronic obstructive pulmonary disease, immunocompromise); age >70 years; multilobar opacities in chest radiograph; shock; severe hypoxaemia; acute renal failure; bacteraemia and acute respiratory distress syndrome (ARDS). One point is given for each variable. Very high scores (5–8) predict 28 mortality better than APACHE II.[3]

General investigations

Full blood count in bacterial CAP may show neutrophilia or neutropenia. Patients may have raised urea. Hyponatraemia can occur due to inappropriate antidiuretic hormone secretion mediated by lung inflammation. A falling C-reactive protein or procalcitonin (PCT) indicates response to treatment. An ECG and troponin level is necessary because acute coronary syndrome (ACS) occurs concurrently in 5% of patients. Chest X-ray shows consolidation, which can either be patchy or lobar. It can also show air bronchograms or interstitial shadowing which can be confused with interstitial oedema from heart failure. It is not possible to accurately predict the aetiology from radiological features. In one study, lung ultrasound was at least as sensitive as chest X-ray in diagnosing pneumonia: 92% vs. 87%.[4] In 8% of cases pneumonia the diagnosis could not be made due to consolidation not extending to pleura. The presence of heart and scapulae also limit the views and significant expertise are required.

Microbiological investigations

Blood and sputum are obtained for culture and sensitivity before prompt empirical antibiotics are administered. Gram stain of sputum is not universally available but in high-severity patients it can give an immediate indicator of the likely pathogen. Urine is sent for pneumococcal and legionella antigen testing. These antigen tests provide rapid results and are unaffected by prior antibiotic therapy. If positive for legionella antigen then culture of sputum for legionella should be requested.

Polymerase chain reaction (PCR) has replaced many of the serological tests for atypical organisms. PCR is used to diagnose *Mycoplasma pneumoniae*, *Chlamydophilia* species and a range of respiratory viruses.

Treatment

Basic care

Oxygen therapy should be used to achieve saturation targets appropriate for the individual.

Patients are also often dehydrated and require intravenous fluid therapy.

Analgesia is often required to target pleuritic pain and may help with expectoration.

Thromboprophylaxis should be considered where a period of immobility is likely.

Antimicrobial therapy

Empirical antibiotic therapy within 4 hours is advised[1] and this should be reduced to within 1 hour when severe sepsis accompanies CAP.[5]

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