# 5
# Pneumonia

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**Abstract** Despite antibiotic therapy, pneumonia remains a significant worldwide cause of morbidity and mortality. The term pneumonia covers several distinct clinical entities, and correct classification is vital as the aetiology, infective organism, antibiotic management and outcome are determined by how and where pneumonia was contracted. Early recognition and appropriate treatment improve outcome. Critical care physicians must be familiar with all aspects of pneumonia, as they will be expected to advise on and manage severe community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP) and opportunistic pneumonias in immuno-compromised patients in the wards, high dependency units (HDUs) and intensive care units (ICUs). Differences in the recently published antibiotic guidelines between the British and American Thoracic Societies are highlighted in this chapter.

## General Definition of Pneumonia

Pneumonia describes an *acute lower respiratory tract* (*LRT*) *illness*, usually but not always due to *infection*, associated with *fever*, *focal chest symptoms* (*with or without clinical signs*) and *new shadowing on chest radiography* (Figure 5.1). The many infective micro-organisms and pathological insults that cause pneumonia are listed in Table 5.1.

## Classification of Pneumonia

In the clinical situation, *microbiological classification* of pneumonia is not practical, since identification of the organism, even when possible, may take several days. Likewise, *anatomical* (*radiographic*) appearance (e.g. lobar pneumonia, which describes consolidation localised to one lobe; or bronchopneumonia, which describes widespread, patchy consolidation) gives little practical information about the cause, course or prognosis of the infection. The following classification is widely accepted:

- **Community-acquired pneumonia (CAP)** describes LRT infections occurring within 48 h of hospital admission in patients who have not been hospitalised for more than 14 days. The most likely infective organisms are *Streptococcus pneumoniae* (20–75%), *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*.

- **Hospital-acquired (nosocomial) pneumonia (HAP)** is defined as any LRT infection developing greater than 48 h after hospital admission, which was not incubating at the time of admission. HAP includes: (i) *ventilator-associated pneumonia* (*VAP*), which refers to pneumonia developing greater than 48–72 h after endotracheal intubation; and (ii) *health-care-associated pneumonia* (*HCAP*), which includes any patient admitted to hospital for more than 2 days within 90 days of the infection, residing in a nursing home, receiving therapy (e.g. wound care, intravenous [iv] therapy) within 30 days of the current infection, or attending a hospital or haemodialysis clinic. Likely, causative organisms differ from CAP and include Gram-negative bacilli (~70%) or *Staphylococcus* (~15%).

- **Aspiration/anaerobic pneumonia** occurs due to *bacteroides* and other anaerobic infections, which follow aspiration of oropharyngeal contents due

**FIGURE 5.1.** Pneumonia chest x-ray (CXR).

**TABLE 5.1.** Micro-organisms and pathological insults that cause pneumonia

| Bacterial infections | Atypical infections | Fungal infection |
|---|---|---|
| Streptococcus pneumoniae | Mycoplasma pneumoniae | Aspergillus |
| Haemophilus influenzae | Coxiella burnetti | Candida |
| Escherichia Coli | Chlamydia psittaci | Nocardia |
| Klebsiella pneumoniae | Legionella pneumophila | Histoplasmosis |
| Pseudomonas aeruginosa | | |
| **Viral infections** | **Protozoal infections** | **Others** |
| Influenza | Pneumocystis carinii | Aspiration |
| Coxsackie | Toxoplasmosis | Bronchiectasis |
| Respiratory syncytial | Amoeboesis | Cystic fibrosis |
| Cytomegalovirus | | Lipoid pneumonia |
| Adenovirus | | Radiation |

to impaired laryngeal competence (e.g. cerebrovascular accident [CVA]) or reduced consciousness (e.g. drugs, alcohol, postoperative).

- **Opportunistic pneumonia** occurs in immunosuppressed patients taking high-dose steroids, chemotherapy or who are HIV positive. They are susceptible to viral, fungal and mycobacterial infections, in addition to the normal range of bacterial organisms.
- **Recurrent pneumonia** occurs due to aerobic and anaerobic organisms in cystic fibrosis and bronchiectasis.

# Community–Acquired Pneumonia

## Epidemiology: Incidence

Prospective population studies report an annual CAP incidence of 5–11 cases per 1,000 adults and ~15–45% require hospitalisation (1–4 cases per 1,000) of whom 5–10% are treated in ICU. Incidence is highest in the very young and elderly (~35 per 1,000 in patients aged >75 years). *Mortality* is <1% in patients treated at home, 5–12% in hospitalised patients and 25–50% in ICU patients. *Seasonal variation* occurs with individual pathogens; *M. pneumonia*, *Staphylococcus* and influenza virus occur in winter, *Legionella* spp. in September–October and *Coxiella burnetii* in spring. Annual cycles are also reported (e.g. 4 yearly *mycoplasma* epidemics). Frequent viral infections increase CAP in winter.

## Aetiology

Identifying the causative pathogen in CAP is difficult and results vary with methodology (e.g. bacterial quantification), geography (e.g. the UK, Europe, the USA) and illness severity (e.g. community, hospital or ICU). No infective cause is found in 30–50% of cases, but in virtually all studies the most frequently identified organism is *S. pneumoniae* (20–75%). The 'atypical' bacterial pathogens including *M. pneumonaie, Chlamydia pneumoniae and Legionalla* spp. (2–25%) and viral infections including influenza A and B (8–12%) are relatively common causes of CAP. *Haemophilus influenza* and *Moraxella catarrhalis* are often associated with COPD exacerbations and staphylococcal infection may follow influenza. *Respiratory syncytial virus* affects children <2 years old. Alcoholic, diabetic, heart failure and nursing-home patients are prone to infection with staphylococcal, anaerobic and Gram-negative organisms.

## Risk Factors

Table 5.2 lists factors associated with increased risk of CAP. *Specific risk factors* include *age* (e.g. mycoplasma in young adults), *occupation* (e.g. brucellosis in abattoir workers, *C. burnetii* [Q fever] in sheep workers), *environment* (e.g. psittacosis with pet birds, tularaemia and erlichiosis due to tick bites) or *geographical* (e.g. coccidomycosis

**TABLE 5.2.** Risk factors for pneumonia

- Age: >65, <5 years old
- Diabetes mellitus
- Chronic disease (e.g. renal, lung)
- Alcohol dependency
- Aspiration (e.g. epilepsy)
- Recent viral illness (e.g. influenza)
- Malnutrition
- Immunosuppression: (e.g. drugs, HIV)
- Environmental (e.g. psitticosis)
- Occupational (e.g. Q fever)
- Travel abroad (e.g. paragonomiasis)
- Air conditioning (e.g. Legionella)
- Mechanical ventilation
- Postoperative (e.g. obesity, smoking)

in southwest USA). Epidemics of *C. burnetti* (Q fever) or *Legionella pneumophila* are often localised. For example, patients developing Legionnaires disease may have been exposed to a contaminated air conditioner at a specific hotel.

## Diagnosis

Diagnosis of CAP on the basis of history and clinical findings is inaccurate without a chest radiograph, and several studies have demonstrated that the causative organism cannot be predicted from clinical features. In particular, 'atypical' pathogens do not have a characteristic clinical presentation. The diagnostic aims are to (1) establish the *diagnosis of pneumonia*, (2) determine *classification* (*aetiology*), which aids initial antibiotic choice, (3) assess *severity*, which determines the most appropriate ward placement (e.g. ward, HDU or ICU), (4) *adjust antibiotic therapy* when microbiological results are available, and (5) identify and treat *complications*.

- **Clinical features**: *symptoms* may be general (e.g. malaise, fever, rigors, myalgia) or chest-specific (e.g. dyspnoea, pleurisy, cough, discoloured sputum, haemoptysis). *Signs* include cyanosis, tachycardia and tachypnoea; with focal dullness, crepitations, bronchial breathing and pleuritic rub on chest examination. In young or old patients and in those with atypical pneumonias (e.g. mycoplasma, legionella) *non-respiratory features* (e.g. headache, confusion, rashes, diarrhoea) may predominate. *Complications* are shown in Figure 5.3.
- **Investigations**: *routine blood tests* should be included. White cell count (WCC) and C-reactive protein (CRP) confirm infection, abnormal liver

function tests suggest *Legionella* spp. or *M. pneumoniae* infection and haemolysis and cold agglutinins occur in ~50% of mycoplasma infections. *Blood gases*: are performed to identify respiratory failure. *Microbiology*: is essential in all cases but no micro-organism is isolated in ~33–50% of patients due to previous antibiotic therapy or inadequate specimen collection. Blood cultures are recommended in severe pneumonia and may be positive in ~25%. Sputum, pleural fluid and bronchoalveolar lavage samples, with appropriate staining (e.g. Gram stain), culture and assessment of antibiotic sensitivity may determine the pathogen (~30–50%) and effective therapy. *Serology*: identifies *M. pneumoniae* infection, but long processing times limit clinical value. Recently developed rapid antigen detection tests for *Legionella* spp. (e.g. in urine) and *S. pneumoniae* (e.g. in serum, pleural fluid, urine) are more useful. *Radiological*: chest x-ray (CXR) and computed tomography (CT) scans aid diagnosis, monitor deterioration, indicate severity and aid early detection of complications (Figure 5.3). Resolution of radiological changes may be slow and should not lead to further investigation during clinical improvement.

- **Severity assessment**: the following features are associated with increased CAP mortality and indicate the need for monitoring on an HDU/ICU. (a) *Clinical*: age > 60 years, respiratory rate > 30/min, diastolic blood pressure < 60 mmHg, new atrial fibrillation, confusion, multilobar involvement and coexisting illness. (b) *Laboratory*: urea > 7 mmol/L, albumin < 35 g/L, hypoxaemia $PO_2$ < 8 kPa, leucopenia (WCC < 4 × 10⁹/L), leucocytosis (WCC > 20 × 10⁹/L) and bacteraemia. *Severity scoring*: the recently validated CURB-65 score, which allocates one points for each of confusion; urea > 7 mmol/L; respiratory rate > 30/min; low systolic (<90 mmHg) or diastolic (<60 mmHg) blood pressure and age > 65 years, stratifies patients into mortality groups suitable for different management pathways. A score greater than 4 indicates the need for admission to a monitored bed in an HDU or ICU.

## Management

### Supportive Measures

These include oxygen to maintain $PaO_2$ > 8 kPa ($SaO_2$ > 90%) and intravenous (iv) fluid and if

necessary inotropic support to maintain a mean blood pressure > 65 mmHg and urine output 0.5–1 mL/kg/min. *Ventilatory support:* non-invasive (e.g. CPAP, NIPPV) or mechanical ventilation (MV) may be required in respiratory failure. Persisting hypoxia with $PaO_2 < 8$ kPa despite maximal oxygen administration, progressive hypercapnia, severe acidosis (pH < 7.2), shock and depressed consciousness are indications for mechanical ventilation. Alveolar recruitment strategies using PEEP aid oxygenation and ventilator modes that avoid high peak pressures and alveolar hyperinflation are optimal. *Physiotherapy* and *bronchoscopy* may aid sputum clearance and sample collection for further microbiology. Steroids do not improve pneumonia resolution, but may be indicted (hydrocortisone 8 mg/h) in patients with septic shock who are poorly responsive to fluid and inotropic therapy.

### Initial Antibiotic Therapy

Initial antibiotic therapy represents the 'best guess', according to pneumonia classification and likely organisms, as microbiological results are often not available for >24h. Therapy is adjusted when results and antibiotic sensitivities are available. The American and British Thoracic Societies (ATS, BTS) recommendations for initial antibiotic therapy differ slightly and are as follows:

- **Non-hospitalised patients:** with less severe symptoms usually respond to oral monotherapy with amoxicillin 0.5–1 g tds (BTS), or a macrolide (e.g. clarithromycin 500 mg bd) (ATS/BTS) *or* doxycycline 100 mg od (ATS). Patients with severe symptoms or at risk for drug-resistant *S. pneumoniae* (e.g. recent antibiotics, comorbidity, alcoholism, immunosuppressive illness, recent travel to areas with high levels of *S. pneumoniae* resistance [e.g. Portugal, Spain]) are treated with a β-lactam (e.g. co-amoxiclav 625 mg tds) *plus* a macrolide or doxycycline; *or* an oral antipneumococcal fluoroquinolone alone (e.g. levofloxacin 500 mg od, moxifloxacin) (ATS).
- **Hospitalised patients:** antibiotic therapy must cover 'atypical' organisms and *S. pneumoniae* at admission. If not severe, the BTS suggests that combined ampicillin *plus* macrolide (oral or iv) may be adequate. In severe CAP, the BTS recommends intravenous therapy with co-amoxiclav

1.2 g tds *or* a second- or third-generation cephalosporin (e.g. cefuroxime 1.5 g tds iv) *plus* a macrolide (e.g. clarithromycin 500 mg bd). The ATS guidelines recommend intravenous therapy with a β-lactam (e.g. ceftriaxone 1–2 g od) *plus* an advanced macrolide (clarithromycin 500 mg bd) *or* an antipneumoccocal fluoroquinolone (e.g. levofloxacin 500 mg bd or moxafloxacin). Staphylococcal infection following influenza and *H. influenzae* in COPD should also be appropriately covered. An antipseudomonal agent may be required when there is underlying structural lung disease.

## Hospital-Acquired (Nosocomial) Pneumonia

*Hospital-acquired pneumonia* (HAP) including *ventilator-associated pneumonia* (VAP) and *health-care associated pneumonia* (HCAP) affects 0.5–2% of hospitalised patients. Pathogenesis, causative organisms and outcome differ from community-acquired pneumonia (CAP). Preventative measures and early antibiotic therapy guided by awareness of the role of potential multidrug-resistant (MDR) pathogens improve outcome (Table 5.3). **Definitions:** for HAP, VAP and HCAP see Classification of Pneumonia.

## Epidemiology

HAP is the second commonest nosocomial (hospital-acquired) infection in the USA (after wound infection) and the most important in terms of mortality. Available data suggest that it affects between 5 and 10 per 1,000 hospital admissions

**TABLE 5.3.** Risk factors for multidrug-resistant pathogens causing hospital-acquired pneumonia

- Antimicrobial therapy in the previous 90 days
- Current hospitalisation of >5 days
- High frequency of local antibiotic resistance
- Presence of risk factors for HCAP
  - Hospitalisation for >2 days in the previous 90 days
  - Residence in a nursing home
  - Home wound care or intravenous therapy
  - Chronic dialysis within 30 days
  - Family member with MDR pathogen
- Immunosuppressive disease and/or therapy

and lengthens hospital stay by 3–14 days/patient. Incidence is highest in surgical and ICU wards and in teaching hospitals. In ICU, HAP accounts for 25% of all infections and ~50% of prescribed antibiotics. The risk of HAP increases 6–20-fold during mechanical ventilation (MV) and occurs in 9–27% of intubated patients. VAP accounts for >80% of all HAPs. The risk of VAP is highest early in the course of hospital stay and is estimated to be 3%/day during the first 5 days of MV, 2%/day during days 5–10 of MV and 1%/day after this. As most MV is short term about 50% of all episodes of VAP occur during the first 4 days of MV.

## Aetiology

HAP is caused by a wide spectrum of bacterial pathogens. Time of onset (i.e. early or late-onset HAP/VAP) and risk factors for infection with MDR organisms determine potential pathogens (Table 5.3). Aerobic Gram-negative bacilli (e.g. *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, *Escherichia coli*, *Acinetobacter* spp.) cause ~60–70% of infections and *Staphylococcus aureus* causes ~10–15%. *S. pneumoniae* and *H. influenzae* may be isolated in early onset HAP/VAP. In ICU, >50% *S. aureus* infections are methicillin-resistant (MRSA). In general, the bacteriology in non-ventilated and ventilated patients is similar. Pneumonia due to *S. aureus* is more common in

diabetics, head trauma and ICU patients. Reported rates of polymicrobial infections vary widely but are increasing and are especially high in patients with ARDS. Viral and fungal infections are rare in immunocompetent hosts.

## Pathogenesis

Oropharyneal colonisation with enteric Gram-negative bacteria occurs in most hospitalised patients within a few days of admission due to immobility, impaired consciousness, instrumentation (e.g. nasogastric tubes, gastroscopy), inadequate attendant hygiene with cross infection or inhibition of gastric acid secretion (e.g. proton pump inhibitor therapy) (Figure 5.2). Subsequent aspiration of nasopharyngeal secretions (or gastric contents) due to supine positioning, reduced consciousness, difficulty swallowing, leakage past endotracheal-tube cuffs, infected biofilm in the endotracheal tube with subsequent embolisation to distal airways or direct inoculation of the airways during suctioning introduces these organisms into the LRT. Impaired mechanical, cellular and humoral host defences and inability to clear secretions (e.g. structural lung disease, sedation, MV, postoperative pain) promote ensuing infection. Haematogenous spread from distant infected sites (e.g. infected central lines) may also cause HAP.
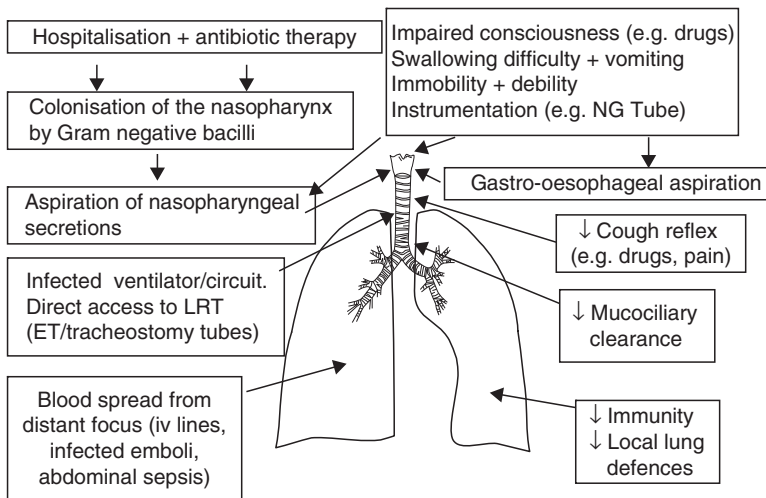


**FIGURE 5.2.** Pathogenesis of hospital-acquired pneumonia (HAP).

## Risk Factors

Risk factors include those that predispose to CAP and factors associated with the development of MDR organisms and HAP pathogenesis (Tables 5.2 and 5.3, Figure 5.2). Critical risk factors include prolonged (>48 h) mechanical ventilation, duration of hospital or ICU stay, severity of illness (including APACHE score), presence of acute respiratory distress syndrome and medical comorbidity. In ventilated patients prior use of antibiotics appears protective, whereas continuous sedation, cardiopulmonary resuscitation, high ventilatory pressures, upper airway colonisation and duration of ventilation are independent risk factors. *Prevention*: Table 5.4 lists modifiable risk factors that reduce HAP incidence.

## Diagnosis

Diagnosis of HAP is based on *both clinical and microbiological assessments* but may be difficult (especially in ventilated patients) as clinical features are often non-specific, patients may have concurrent illness (e.g. ARDS) and previous antibiotics limit microbiological evaluation. *Clinical criteria*: HAP is suspected if a patient develops new radiographic infiltrates (± hypoxaemia) with at least two of three clinical features suggestive of infection (e.g. temperature > 38°C, purulent sputum, leukocytosis or leukopaenia). Purulent tracheobronchitis may mimic many of the clinical features of HAP. *Diagnostic tests*: verify infection and determine the causative organism and antibiotic sensitivity. They include all the routine blood and radiographic tests used in CAP, blood cultures, CRP monitoring, diagnostic thoracocentesis of pleural effusions and LRT cultures including endotracheal aspirates and bronchioalveolar lavage in intubated patients. CT scanning aids diagnosis and detection of complications. *Complications:* are frequent with cavitation, abscesses, effusions and haemoptysis (Figure 5.3).

## Management

*Early diagnosis and treatment of HAP improves morbidity and mortality* and requires constant vigilance in hospital patients. Antibiotic therapy must not be delayed while awaiting diagnostic tests and microbiological results.
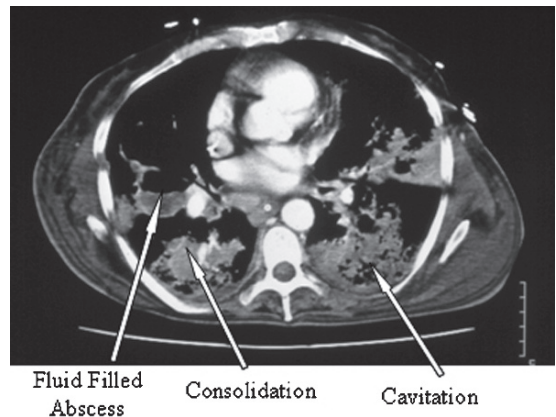
**TABLE 5.4.** Risk factors for HAP and VAP

| Unmodifiable risk factors | Modifiable risk factors |
|---|---|
| *Host-related* | *Host-related* |
| • Malnutrition | • Nutrition (e.g. enteral feeding) |
| • Age: >65, <5 years old | • Pain control, physiotherapy |
| • Diabetes | • Posture, kinetic beds |
| • Chronic disease (e.g. renal) | • Limit immunosuppressive therapy |
| • Alcohol dependency | • Preoperative smoking cessation |
| • Aspiration (e.g. epilepsy) | |
| • Immunosuppression (e.g. SLE) | *Therapy-related* |
| • Recent viral illness | • Semi-recumbent position (30° head up) |
| • Smoking | • Early removal of iv lines, ET and NG tubes |
| • Obesity | • Avoid intubation + re-intubation |
| *Epidemiological factors* | • Minimise sedative use |
| • Environmental (e.g. psitticosis) | • Avoid gastric overdistention |
| • Occupational (e.g. Q fever) | • Maintain ET cuff pressure >20 cmH$_2$O |
| • Travel abroad (e.g. paragonomiasis) | • Subglottic aspiration during intubation |
| • Air conditioning (e.g. Legionella) | • Drain and change ventilator circuits |
| *Therapy-related* | *Infection control* |
| • Mechanical ventilation | • Hand washing, sterile technique |
| • Postoperative | • Microbiological surveillance |
| | • Patient isolation |



**FIGURE 5.3.** Computed tomography (CT) scan from a patient with hospital-acquired pneumonia showing consolidation, cavitation and abscess formation.

## Supportive Therapy

Supportive therapy includes supplemental oxygen to maintain $PaO_2 > 8\,kPa$ (saturation > 90%) and intravenous fluids (± vasopressors or inotropes) to maintain a mean blood pressure >65 mmHg and urine output 0.5–1 mL/kg/min. *Ventilatory support* including non-invasive (e.g. CPAP, NIPPV) or mechanical ventilation may be required in respiratory failure (see CAP management). *Intensive insulin therapy* to maintain serum glucose levels between 4 and 6.5 mmol/L improves outcome. Vigorous *physiotherapy* and adequate *analgesia* aid sputum clearance postoperatively and in the immobilised patient. Avoid heavy sedation and paralytic agents that may depress cough. *Semi-recumbent nursing* of bed-bound patients with elevation of the bedhead to 30° reduces aspiration risk. *Continuous subglottic aspiration* using specially designed endotracheal tubes may reduce early onset VAP but frequency of ventilator circuit changes does not affect VAP incidence. *Stress ulcer prophylaxis* with either sucrulfate or proton pump inhibitors is acceptable although there is a trend to reduce VAP with sucrulfate. It is essential that modifiable risk factors are addressed (Table 5.4).

## Antibiotic Therapy

Antibiotic therapy is empirical (as with CAP) while awaiting microbiological guidance. However, the causative organisms and suitable antibiotics differ from CAP. The initial key decisions are (i) whether the patient has risk factors for MDR organisms (Table 5.3), and (ii) whether the patient has early or late-onset HAP/VAP or HCAP, which determines the need for broad-spectrum (i.e. combination) antibiotic therapy. Figure 5.4 illustrates the recently published American Thoracic Society guidelines for initial, empiric antibiotic therapy. Initial therapy should be intravenous in all patients. About 50% of the initial, empiric treatment regimes will need modification due to either resistant organisms or failure to respond. However, it is important to try and get the antibiotic treatment 'right the first time' because mortality is lower in patients receiving effective initial antibiotic therapy compared to those that require a treatment change. Consequently, *local*
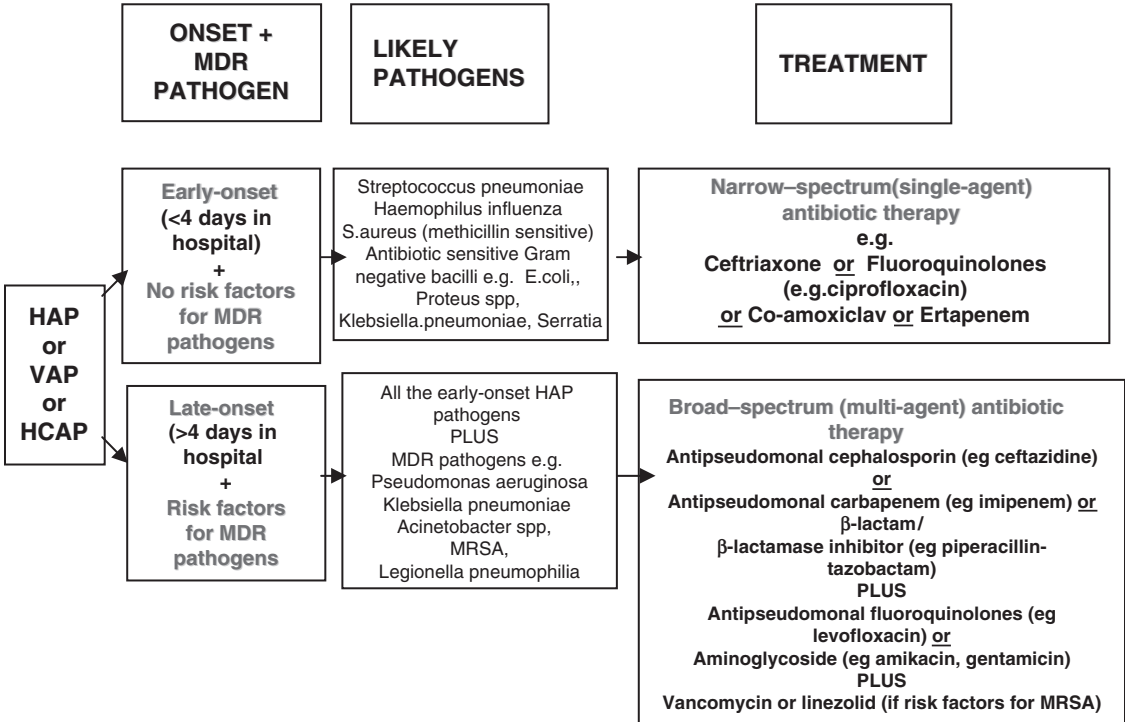


FIGURE 5.4. Likely pathogens and empirical antibiotic treatment of hospital-acquired pneumonia (HAP).

patterns of bacterial infection and antibiotic resistance should be used to establish the 'best empiric therapy regimen' for an individual hospital.

- In *early onset HAP/VAP* (<4 days in hospital) with no risk factors for MDR organisms, antibiotic *monotherapy* is advised with:

  - A β-lactam/β-lactamase inhibitor (e.g. piperacillin-tazobactam 4.5 g qds iv)
  - A third-generation cephalosporin (e.g. ceftazidime 2 g tds iv) or
  - A floroquinolone (e.g. ciprofloxacin 400 mg tds iv)

- In *late-onset HAP/VAP* (>4 days in hospital) or with risk factors for MDR pathogens (Table 5.4) and most HCAP *combination therapy*, with broad-spectrum antibiotics that cover MDR Gram-negative bacilli and MRSA, may be required. For example:

  - An antipseudomonal cephalosporin (e.g. ceftazidine 2 g tds iv), an antipseudomonal carbepenem (e.g. imipenem 0.5–1 g qds, meropenem 1 g tds iv), or a β-lactam/β-lactamase inhibitor (e.g. piperacillin-tazobactam 4.5 g qds iv).
  - *Plus* an antipseudomonal fluoroquinolone (e.g. ciprofloxacin 400 mg tds iv, levofloxacin 750 mg od iv) or aminoglycoside (e.g. gentamicin 7 mg/kg od iv, adjusted to maintain monitored trough levels <1 μg/mL).
  - *Plus* vancomycin 15 mg/kg bd (adjusted to maintain monitored trough levels 15–20 μg/mL) or linezolid 600 mg bd iv.
  - Adjunctive therapy with inhaled aerosolised aminoglycosides or polymyxin should be considered in patients not improving with systemic antibiotic therapy or who have VAP due to MDR Gram-negative pathogens and/or carbapenem-resistant *Acinetobacter* spp.

A short course of therapy (e.g. 7 days) is appropriate, if the clinical response is good. However, aggressive or resistant pathogens (e.g. *P. aeruginosa*, *S. Aureus*) may require treatment for 14–21 days, which improves subsequent survival. Overuse of antibiotics is avoided by tailoring antibiotic therapy to the results of lower respiratory tract cultures, withdrawing unnecessary antibiotics and shortening duration of therapy to the minimum effective period. Sterile cultures (in the absence of new antibiotics in the previous 72 h) virtually rules out the presence of HAP (94% negative predictive value for VAP) and withdrawal of antibiotic therapy should be considered. Although there is some evidence for a reduction in ICU-acquired HAP/VAP following prophylactic antibiotic administration, routine use is *not* recommended.

## Mortality

The crude mortality rate for HAP may be as high as 30–70%, but many of these critically ill patients die of their underlying disease rather than pneumonia. The directly 'attributable mortality' due to HAP is estimated to be 33–50% in case-matched VAP studies. Delayed or ineffective antibiotic therapy, bacteraemia (especially with *P. aeruginosa* or *Acinetobacter* spp.), medical rather than surgical illness and VAP also increase mortality. *Early onset* HAP/VAP (defined as occurring within the first 4 days of hospitalisation) is usually caused by antibiotic-sensitive bacteria and carries a better prognosis than *late-onset* HAP/VAP (defined as occurring 5 days or more after hospitalisation), which is associated with MDR pathogens. However, in early onset HAP/VAP, prior antibiotic therapy or hospitalisation predisposes to MDR pathogens and is treated as late-onset HAP/VAP.

## Other Pneumonias

### Aspiration/Anaerobic Pneumonia

*Bacteroides* and other anaerobic infections follow aspiration of oropharyngeal contents due to impaired laryngeal competence (e.g. CVA) or reduced consciousness (e.g. drugs, alcohol) (Figure 5.5). Lung abscesses are common. Antibiotic therapy should include anaerobic coverage (e.g. metronidazole 500 mg tds iv). Large lung abscesses may require oral antibiotic therapy for several weeks.

### Pneumonia During Immunosuppression

HIV, bone marrow transplant and chemotherapy patients are susceptible to viral (e.g. cytomegalovirus), fungal (e.g. aspergillus) and mycobacterial infections, in addition to the normal range of organisms. Severely immunocompromised patients require isolation, barrier nursing
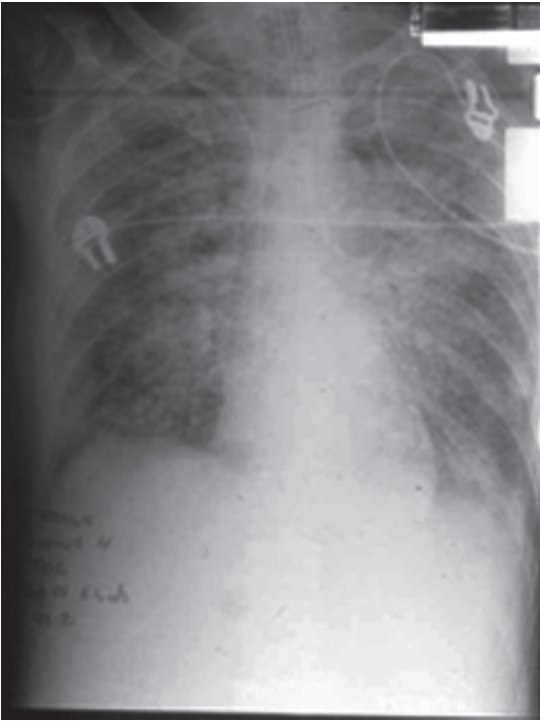
**FIGURE 5.5.** Aspiration pneumonia; chest x-ray (CXR) 6 h after aspiration of gastric contents.

and combined broad-spectrum antibiotic, antifungal (e.g. amphotericin) and antiviral (e.g. acyclovir) regimes. HIV patients with $CD_4$ counts <200/$mm^3$ are at high risk of opportunistic infections including *Pneumocystis carinii* pneumonia (PCP), toxoplasmosis and mycobacterium avian intracellulare. PCP is treated with steroids and high-dose co-trimoxazole.

## Recurrent Pneumonia

Recurrent pneumonia with aerobic and anaerobic organisms occurs in cystic fibrosis and bronchiectasis. Pulmonary fibrosis with distorted architecture and frequent antibiotic usage result in MDR organisms and the need for broad-spectrum regimens adjusted according to microbiological results and antibiotic sensitivities.

## Recommended Reading

1. BTS Guidelines for the management of community acquired pneumonia in adults. *Thorax*. 2001;56 (Suppl 4):1–68.
2. American Thoracic Society: Guidelines for the management of adults with community-acquired pneumonia; diagnosis, assessment of severity, microbial therapy and prevention. *Am J Respir Crit Care Med*. 2001;163:1730–1754.
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5. P. Tarsia, S. Aliberti, R. Cosentini, F. Blasi. Hospital-acquired pneumonia. *Breathe*. 2005;1:297–301.