Acute management of pneumonia in adult patients

Opening Vignette

A 40-year-old woman presented to her general practitioner's clinic with 3 days of cough, yellow phlegm, dyspnoea and fever. She had diabetes mellitus, which was under good control with insulin and metformin. She had no sick contacts and was fully vaccinated for COVID-19. Vital signs were as follows: temperature 39°C, respiratory rate 25 breaths/min, heart rate 120 beats/min, blood pressure 80/50 mmHg and peripheral oxygen saturation of 89% in room air. Physical examination additionally revealed right-sided lung crepitations.

IDENTIFICATION AND DIFFERENTIAL DIAGNOSIS OF PNEUMONIA

The patient has clinical features of pneumonia, marked by a combination of cough, phlegm production, fever and lung crepitations, which suggest infection of the lung parenchyma. Not all of these clinical features may be present; for example, fever may be absent in older adults. Radiological confirmation of pneumonia requires demonstration of consolidation on chest radiography. In the absence of chest radiography, point-of-care lung ultrasound may be used to identify consolidation that abuts the visceral pleura, though consolidation that does not reach the pleura may be missed. Besides consolidation, other ultrasonographic signs of pneumonia include hepatisation of the lung, dynamic air bronchograms and shred sign (irregular junction between consolidated and aerated lung). For the diagnosis of community-acquired pneumonia, lung ultrasonography in the hands of non-imaging specialists (e.g. emergency physicians, internal medicine physicians, intensivists) achieved a sensitivity of 68%–100% and a specificity of 61%–99%, compared to a chest computed tomography reference standard.[1] Blood and sputum cultures — preferably before antibiotic administration — can help identify the pathogen and streamline antimicrobial therapy. Additionally, genotyping for the detection of antibiotic resistance-related mutations may be available, though culture remains an important reference standard for drug susceptibility testing.

Chest radiography, computed tomography and culture-based tests are often taken as the reference tests for pneumonia diagnosis, particularly for severe pneumonia. Beyond these reference tests, other tests are available to diagnose pneumonia and its aetiology, with widely varying sensitivities and specificities [Table 1].[1-10] For a test to be useful in ruling *out* a diagnosis/aetiology, its *sensitivity* should be high (e.g. ≥90%). Similarly, for a test to be useful in ruling *in* a diagnosis/aetiology, its *specificity* should be high (e.g. ≥90%). For low-risk community-acquired pneumonia which is managed on an outpatient basis, routine blood cultures have low yield. Depending on clinical suspicion, rapid antigen or molecular tests for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) or sputum testing for tuberculosis may be considered. Nonetheless, even without any chest imaging or microbiological testing, *presumed* pneumonia based on clinical features alone needs prompt attention from frontline clinicians.

Frontline clinicians would encounter pneumonia often [Table 2]. Pneumonia is a leading infectious cause of disease and death globally, with >2 million deaths reported from lower respiratory tract infections in 2016.[11] Pneumonia can also occur within healthcare settings like nursing homes, hospital wards and intensive care units. Patients with pneumonia may also present repeatedly if the disease is non-resolving. Guidelines for pneumonia have been published in the USA[12,13] and the UK.[14] The principles of management as outlined in this article are consistent with these guidelines. Additionally, while some guidelines differentiate between community-acquired and hospital-acquired pneumonia,[13,14] the clinical impact of such classification seems to be minimal compared to local epidemiology.[12]

Determination of pneumonia depends on the clinical criteria and does not seem to benefit from more complicated scoring methods (e.g. Clinical Pulmonary Infection Score for diagnosis of ventilator-associated pneumonia).[13] It is important to avoid diagnostic closure, and the general approach to pneumonia in adult patients requires consideration of differential diagnoses [Figure 1]. Mimics of pneumonia can present with the same constellation of respiratory symptoms, but require other specific treatment. For instance, extrapulmonary infection can seed the lungs via haematogenous spread and requires either intensified antibiotic dosing in the case of infective endocarditis or pus drainage in the case of liver abscess. Non-infective pneumonia (e.g. organising pneumonia, eosinophilic pneumonia) responds well to steroids, while pulmonary embolism requires anticoagulation.

RISK STRATIFICATION OF PNEUMONIA

The relatively high prevalence and mortality burden of pneumonia has driven development of several early warning and risk stratification systems. If available, machine learning can continuously draw upon big data to accurately identify and prognosticate pneumonia. Nonetheless, such a complex approach may not be accessible or necessary for frontline

Table 1. Selected diagnostic test characteristics and requirements.

Diagnostic test	Diagnostic outcome	Test characteristics
Lung ultrasound by non-imaging specialists, looking for subpleural or alveolar consolidation[1]	Presence of CAP	Sensitivity: 68%–100% Specificity: 57%–100%
Serum CRP>10 mg/L[2]	Presence of CAP	Sensitivity: 90% (CI 52%–99%) Specificity: 48% (CI 27%–70%)
Serum PCT>0.5 mcg/L[2]	Presence of CAP	Sensitivity: 28% (CI 11%–53%) Specificity: 96% (CI 80%–99%)
Serum PCT>0.5 mcg/L[3]	Bacterial CAP (vs. viral CAP)	Sensitivity: 10%–78% Specificity: 47%–100%
Serum mycoplasma IgM Ab[4]	Mycoplasma LRTI	Sensitivity: 85% (CI 63%–95%) Specificity: 90% (CI 75%–97%)
Urine streptococcal antigen[4]	Pneumococcal LRTI	Sensitivity: 70% (CI 60%–79%) Specificity: 83% (CI 63%–93%)
Urine *Legionella* antigen[6]	Legionellosis (mostly serotype 1)	Sensitivity: 74% (CI 68%–81%) Specificity: 99% (CI 98%–99.7%)
Sputum bacterial culture[7]	Non-TB bacteria	*Streptococcus pneumoniae* Sensitivity: 59% (CI 56%–62%) Specificity: 87% (CI 86%–89%) *Haemophilus influenzae* Sensitivity: 78% (CI 72%–84%) Specificity: 96% (CI 94%–97%) *Staphylococcus aureus* Sensitivity: 72% (CI 53%–87%) Specificity: 97% (CI 95%–99%) Gram-negative bacilli Sensitivity: 64% (CI 49%–77%) Specificity: 99% (CI 97%–99%)
Direct sputum smear for TB[8]	Pulmonary TB	Sensitivity: 31%–80% Specificity: 93%–100%
Immunochromatographic assay for influenza A/B Ag using respiratory samples[4]	Influenza LRTI	Sensitivity: 69% (CI 64%–74%) Specificity: 97% (CI 96%–98%)
TB PCR test using sputum[9]	Pulmonary TB	Smear-negative, culture-positive Sensitivity: 78% (CI 68%–86%) Specificity: 96% (CI 93%–98%) Smear-positive, culture-positive Sensitivity: 99% (CI 98%–100%) Specificity: 100%
Pneumococcus PCR test using respiratory samples[4]	Pneumococcal LRTI	Sensitivity: 96% (CI 93%–98%) Specificity: 91% (CI 71%–98%)
Mycoplasma PCR using respiratory samples[4]	*Mycoplasma pneumoniae* LRTI	Sensitivity: 87% (CI 73%–95%) Specificity: 98% (CI 97%–99%)
Chlamydia PCR using respiratory samples[4]	*Chlamydophila pneumoniae* LRTI	Sensitivity: 83% (CI 58%–94%) Specificity: 99% (CI 96%–100%)
COVID-19 quantitative PCR test on respiratory samples[10]	COVID-19 infection	Sensitivity: 93% (CI 88%–96%) Specificity: 93% (CI 87%–96%)
Influenza A/B PCR test using respiratory samples[4]	Influenza LRTI	Sensitivity: 94% (90%–96%) Specificity: 98% (97%–99%)
Pneumocystis PCR using BAL or induced sputum[5]	PCP	Sensitivity: 94%–99% Specificity: 89%–91%
Pneumocystis cytology using BAL or induced sputum[5]	PCP	Sensitivity: 55–90% (lower in HIV-negative patients) Specificity: 100%

Ab: antibody, Ag: antigen, BAL: bronchoalveolar lavage, CAP: community-acquired pneumonia, CI: 95% confidence interval, COVID-19: coronavirus disease 2019, CRP: C-reactive protein, LRTI: lower respiratory tract infection, PCP: *Pneumocystis jirovecii* pneumonia, PCR: polymerase chain reaction (also known as nucleic acid amplification test), PCT: procalcitonin, TB: tuberculosis

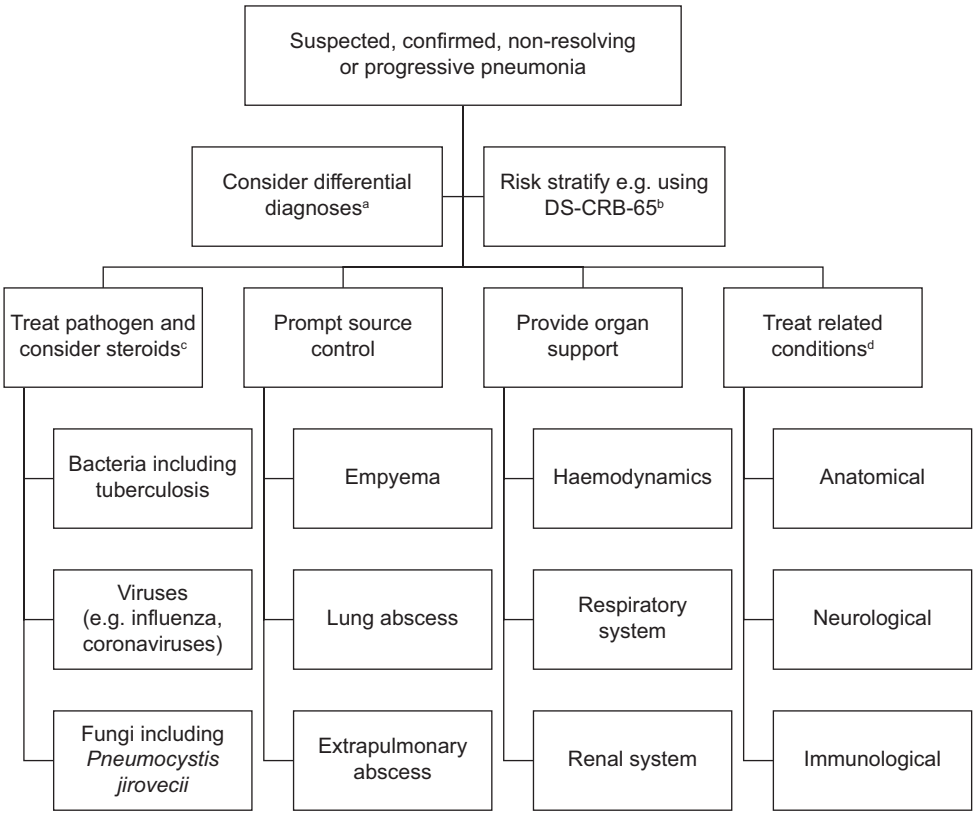

Figure 1: Chart shows the approach to pneumonia in adult patients. [a]Differential diagnoses of pneumonia: extrapulmonary infection (e.g. infective endocarditis, liver abscess), heart failure, cancer, interstitial lung disease, non-infective pneumonia (e.g. organising pneumonia, eosinophilic pneumonia), pulmonary embolism. [b]DS-CRB-65: D-criterion (comorbid diseases) defined as the presence of one or more of the following: congestive heart failure, chronic renal disease, chronic liver disease, cerebrovascular disease, other chronic neurological disease or active malignancy; S-criterion defined as oxygen saturation of <90% measured by pulse oximetry at admission or on first physician contact without oxygen supplementation; C-criterion defined as confusion; R-criterion defined as respiratory rate ≥30/min; B-criterion defined as systolic blood pressure <90 mmHg or diastolic blood pressure ≤60 mmHg; and 65-criterion defined as age ≥65 years. [c]Consider steroids for (a) patients with an ongoing need for vasopressor therapy; (b) patients with *Pneumocystis jirovecii* pneumonia who have arterial oxygen partial pressure <70 mmHg or peripheral oxygen saturation of <92% in room air and (c) patients with COVID-19 pneumonia who require either invasive mechanical ventilation or oxygen. [d]Related conditions leading to pneumonia: (a) anatomical (e.g. oral hygiene, head/neck cancer, tracheoesophageal fistula, endobronchial obstruction, abdominal distension); (b) neurological (e.g. stroke, neuromuscular disease, alcohol/drug intoxication); and (c) immunological (e.g. poorly controlled diabetes mellitus, acquired immunodeficiency syndrome, leucopenia, immunoglobulin deficiency).

clinicians to provide good care. Rapid triaging of pneumonia can be done with one of several risk scores (e.g. Pneumonia Severity Index, CURB-65 and the Infectious Diseases Society of America/American Thoracic Society [IDSA/ATS] 2007 criteria).[15,16] It must be emphasised that risk stratification using any score is imperfect[15-18] and requires clinician gestalt as a safety net. Additionally, risk stratification by itself works only when its result is promptly linked to appropriate treatment and disposition.

Examples of risk stratification tools for pneumonia that can be used without the need for laboratory testing are the CRB-65[19] and the DS-CRB-65[20] scores [Table 3]. One point is assigned to each of the following six criteria for the DS-CRB-65 score: D-criterion (comorbid diseases) defined as the presence of one or more of the following — congestive heart failure, chronic renal disease, chronic liver disease, cerebrovascular disease, other chronic neurological disease or active malignancy; S-criterion defined as oxygen saturation of <90% as measured by pulse oximetry at admission or on first physician contact without oxygen supplementation; C-criterion defined as confusion; R-criterion defined as respiratory rate ≥30/min; B-criterion defined as systolic blood pressure <90 mmHg or diastolic blood pressure ≤60 mmHg; and 65-criterion defined as age ≥65 years. With wider availability of home-based pulse oximetry and digital blood pressure monitors, this score may even be applied via telemedicine. CRB-65 ≥1 or DS-CRB-65 ≥2 predicts increased 30-day mortality and necessitates closely monitored management, most commonly in an inpatient or, more recently, in a hospital-at-home setting.

ANTIMICROBIAL CHOICE FOR PNEUMONIA

Pneumonia is caused by one or more pathogens which can directly damage the lung tissue. Early administration of antimicrobials reduces the microbial load and blunts direct

Table 2. Selected clinical scenarios for pneumonia in adult patients.

Clinical scenario	Patient characteristics	Pathogen considerations
Immunocompetent	Absence of immunosuppressive conditions and medications	Pneumococcus; *Mycoplasma*; *Legionella*; influenza; SARS-CoV-2; *Pseudomonas* (especially for severe pneumonia); Melioidosis (especially for severe pneumonia in the tropics); tuberculosis
Immunocompromised	Poorly controlled diabetes mellitus; solid-organ transplant; haematopoietic stem cell transplant; high-dose steroids and other immunosuppressive drugs; chemotherapy HIV patients with low CD4 counts	Melioidosis; tuberculosis; *Pneumocystis jirovecii*; Aspergillosis; moulds; Cytomegalovirus
Previous and recent exposure to broad-spectrum antimicrobials	Patients can be from the community or from healthcare-associated settings (e.g. nursing home and hospitals)	Methicillin-resistant; *Staphylococcus aureus*; drug-resistant *Pseudomonas*
Large volume aspiration (macroaspiration)	Impaired consciousness; cardiac arrest; neurological disorders with bulbar dysfunction; head-and-neck cancer; poor cough; impaired cough reflex; aspiration pneumonitis affects the most dependent regions of the lung and secondary bacterial infection may then set in	Polymicrobial infection including anaerobes; check for concomitant foreign body aspiration, which would need removal via interventional bronchoscopy; for mild illness due to chemical pneumonitis, antibiotics may be withheld and the patient reviewed in 48 h
Postoperative	Inadequate fasting leading to aspiration risk; premature extubation before return of protective airway reflexes	For mild illness due to chemical pneumonitis, antibiotics may be withheld and the patient reviewed in 48 h
Ventilator acquired	Invasive mechanical ventilation >48 h, via endotracheal tube or tracheostomy	Methicillin-resistant *S. aureus*; *Acinetobacter*; *Pseudomonas*; *Stenotrophomonas*

HIV: human immunodeficiency virus, SARS-CoV-2: severe acute respiratory syndrome coronavirus 2

pathogenic attack. Empirical broad-spectrum antimicrobials should be administered based on best available data. Such data include knowledge on local epidemiology and hospital antibiograms.

Studies conducted before the coronavirus disease 2019 (COVID-19) pandemic in the Asia-Pacific showed that community-acquired bacterial pneumonia was predominantly due to *Streptococcus pneumoniae*, *Mycoplasma pneumoniae*, *Haemophilus influenzae*, *Chlamydophila pneumoniae* and *Staphylococcus aureus*, though community-acquired methicillin-resistant *S. aureus* remained uncommon.[21] For severe cases, *Klebsiella pneumoniae*, *Burkholderia pseudomallei* and severe influenza pneumonia were additional aetiologic considerations. Therefore, to empirically treat community-acquired bacterial pneumonia, β-lactam and macrolide combination therapy would generally be sufficient, with broader gram-negative cover and an anti-influenza agent also required for severe cases.[12]

During the COVID-19 pandemic, many cases of community-acquired pneumonia were due to SARS-CoV-2. While treatment with antibiotics is commonly recommended for bacterial pneumonia, it would not be necessary in most instances of mild-to-moderate COVID-19 pneumonia. Another instance where antibiotics may be withheld is mild aspiration pneumonia due to chemical pneumonitis.[22]

Certain clinical scenarios deserve special consideration to guide the identification of possible pathogens and drug resistance [Table 2]. In countries with mid-to-high tuberculosis prevalence, careful attention to the chest radiograph is required to look for active pulmonary tuberculosis. The presence of patchy infiltrates or cavitation in the upper lobes necessitates early referral for sputum testing. This not only benefits the patient clinically, but also improves public health by limiting tuberculosis spread. At the same time, in such countries, empirical use of respiratory fluoroquinolones (e.g. levofloxacin, moxifloxacin) should be avoided as these medications are also used to treat tuberculosis. Using a single antituberculosis agent leads to drug resistance and delays culture positivity should subsequent sputum testing be done.

Immunocompromised patients are susceptible to myriad pathogens. For example, patients with poorly controlled diabetes mellitus living in the tropics may be infected by melioidosis. Patients with human immunodeficiency virus (HIV) and low CD4 counts may get *Pneumocystis jirovecii* pneumonia. In non-HIV patients on long-term steroids and other immunomodulatory agents (e.g. biological agents), both tuberculosis and *P. jirovecii* pneumonia should be considered. Highly immunocompromised patients, like those who have recently undergone haematopoietic stem cell transplantation, may even be infected by fungi/moulds (e.g. *Candida*, *Aspergillus*, *Mucor*) and parasites (e.g. *Strongyloides*).

STEROID USE FOR PNEUMONIA

The immune response to infection is an overlap of proinflammatory and anti-inflammatory processes, and the net effect could be hyperinflammatory or immunosuppressive.[23] Apart from direct pathogenic attack, excessive inflammation can also lead to organ damage. Steroids and other anti-inflammatory agents (e.g. interleukin-6 inhibitors) may have a role to mitigate infection-induced inflammation. However, these drugs are double-edged swords. Excessive anti-inflammatory agent use raises secondary infection risk and worsens overall clinical outcomes, especially with concurrent infection-induced immunosuppression.

Nonetheless, appropriate patient selection and steroid dosing can improve clinical outcomes. A recommended indication for steroids is pneumonia with refractory septic shock;[12] patients

Table 3. Prognostic scores (not requiring blood tests) for pneumonia in adult patients.

Condition	Score components	Interpretation
CRB-65	C: Score 1 if confusion is present R: Score 1 if the respiratory rate is ≥30/min B: Score 1 if the systolic blood pressure is <90 mmHg or diastolic blood pressure is ≤60 mmHg 65: Score 1 if age is ≥65 years	Total score 0: Low risk 1–2: Intermediate risk 3–4: High risk Mortality over ~30 days in hospital-based studies Low risk: 2.4% Intermediate risk: 13.3% High risk: 34.3% Mortality over ~30 days in community-based studies Low risk: 0% Intermediate risk: 1.6% High risk: 18.5%
DS-CRB-65	D: Score 1 if one or more comorbid diseases is present. Comorbid diseases include congestive heart failure, chronic kidney disease, chronic liver disease, cerebrovascular disease, other chronic neurological diseases or active malignancy S: Score 1 if oxygen saturation is <90% measured by pulse oximetry at admission or on first physician contact without oxygen supplementation C: As in CRB-65 R: As in CRB-65 B: As in CRB-65 65: As in CRB-65	Total score 0–1: Low risk 2: Intermediate risk 3–6: High risk Mortality over ~30 days in hospital–based studies Low risk: 0–1% Intermediate risk: 5% High risk: 10%–50%

with an ongoing need for vasopressor therapy should receive 200 mg/day of intravenous hydrocortisone, which speeds up shock resolution.[24] For *P. jirovecii* pneumonia with hypoxaemia (arterial oxygen partial pressure <70 mmHg or peripheral oxygen saturation <92% in room air), adjunctive corticosteroid has mortality benefit for HIV-positive patients (admittedly less so for HIV-negative patients).[25] The usual steroid regimen for *P. jirovecii* pneumonia with hypoxaemia consists of prednisolone 40 mg twice daily (days 1–5), followed by 40 mg/day (days 6–10) and then 20 mg/day (days 11–21).

More recently, another accepted indication for steroids is COVID-19 pneumonia. Patients with COVID-19 pneumonia who require either invasive mechanical ventilation or supplemental oxygen benefit from oral or intravenous dexamethasone 6 mg once daily for up to 10 days, which decreases 28-day mortality.[26] In the DEXA-ARDS trial, which requires further validation, non-COVID-19 patients with persistent moderate-to-severe acute respiratory distress syndrome (ARDS) (defined by a ratio of partial pressure of arterial oxygen to the fraction of inspired oxygen [FiO_2] of ≤200 mmHg assessed with a positive end-expiratory pressure of ≥10 cmH_2O and FiO_2 of ≥0.5 at 24 h after ARDS onset), benefited from intravenous dexamethasone 20 mg once daily (days 1–5), followed by 10 mg once daily (days 6–10), which decreased 60-day mortality.[27]

SOURCE CONTROL AND ORGAN SUPPORT FOR PNEUMONIA

In most cases of pneumonia, a short course of antibiotics over 5–7 days[12,13] brings about clinical improvement and eventual resolution of symptoms over the next 2–3 weeks. Complicated forms of pneumonia respond less favourably to short courses of antibiotics alone. Pneumonia with abscess formation, which can be diagnosed using computed tomography, requires an extended course over 4–6 weeks. For cases of parapneumonic effusion or empyema, source control with drainage of infected pleural collections may be most expediently done using ultrasound-guided chest tube drainage. Poorly draining pleural collections may be due to viscous pus or pleural septations, which can be overcome by intrapleural dual lysis (comprising DNase and tissue plasminogen activator) or by video-assisted thoracic surgery. Occasionally, pneumonia may be secondary to spread from an extrapulmonary source, such as from infective endocarditis or liver. Infective endocarditis requires prolonged high-dose antibiotics, while large liver abscesses may require percutaneous or surgical drainage.

In patients with pneumonia, support of key organs involves reversal of shock, acute respiratory failure and acute kidney failure. Shock is most readily identified by delayed capillary refill >3 s and necessitates a rapid fluid challenge, even in the absence of hypotension (i.e. shock can exist even if the mean arterial pressure is <65 mmHg, where the mean arterial pressure is the sum of one-third of the systolic blood pressure and two-thirds of the diastolic blood pressure). Concomitant hypotension, if not improved by fluid challenge, usually requires administration of vasopressors, for example, noradrenaline starting at a dose of 0.05 mcg/kg/min or dopamine starting at 5 mcg/kg/min. Acute respiratory failure can be readily detected by a peripheral oxygen saturation of <90% using routine pulse oximetry. Supplemental oxygen using nasal prongs or face masks should be quickly instituted pending further investigation (e.g. arterial blood gas analysis) and before more advanced forms of respiratory support are used (e.g. non-invasive or invasive mechanical ventilation). Renal failure may not be apparent initially and may be demonstrated clinically by oliguria (i.e. urine production <0.5 mL/kg body weight/h). Blood urea should be measured, and kidney replacement therapy is indicated when the blood urea exceeds 39.9 mmol/L or if oliguria persists for >72 h.[28]

RELATED CONDITIONS LEADING TO PNEUMONIA

When pneumonia is confirmed, identification and treatment of underlying causes can help reduce recurrence. Bacterial burden is increased with poor oral hygiene, which is readily apparent. Aspiration risk is increased in the presence of anatomical

or neurological anomalies. Anatomical anomalies include head/neck cancer (before or after treatment with radiotherapy), tracheoesophageal fistula (e.g. due to oesophageal cancer) and endobronchial obstruction (e.g. due to foreign body aspiration or endobronchial cancer). For the latter, a fixed and localised wheeze may be detected on physical examination. Neurological anomalies affecting bulbar function (e.g. stroke, neuromuscular disease, alcohol/drug intoxication) can present with audible gurgling.

Both anatomical and neurological anomalies exist in post-operative pneumonia, which occurs after 0.5%–28% of general surgical operations and 2%–54% of cardiothoracic surgical procedures.[29] High-risk patients may be identified before non-cardiothoracic surgery using a variety of clinical tools.[30,31] Anatomically, abdominal splinting due to ascites or bowel obstruction may result in macroaspiration if not adequately decompressed. Neurologically, post-operative analgesia may impair cough and secretion clearance, leading to atelectasis and pneumonia in patients undergoing prolonged bedrest.

Immunological suppression not only makes the patient vulnerable to pneumonia, but also increases the chance of less-common infections. Some conditions leading to immunological suppression are modifiable. Poorly controlled diabetes mellitus requires glucose control while avoiding hypoglycaemia, and a glucose target range of 7.8–10 mmol/L is reasonable in critically ill diabetic patients with pneumonia.[32] Acquired immunodeficiency syndrome requires early institution of antiretroviral therapy. Leucopenia may be reversed using granulocyte colony-stimulating factor, while immunoglobulin deficiency may be overcome with immunoglobulin infusion.

FOLLOW-UP AFTER THE ACUTE EPISODE OF PNEUMONIA

Patients who receive appropriate therapy for pneumonia should improve clinically within 48–72 h,[12,14] though complete disease resolution takes a longer time. Post-pneumonia cough may persist for several weeks. Decreased physical function may continue for months, necessitating graduated return to pre-illness exercise levels. Radiological resolution takes 2–3 months for most patients, and follow-up chest radiographs may be done then.[33] Persistent radiological abnormalities should prompt consideration of pulmonary tuberculosis, which is endemic in Singapore, and alternative diagnoses like lung cancer in a patient with risk factors (e.g. smoking/elderly).

Patients who improve clinically should complete a short course of antibiotics, typically over 5–7 days.[12-14] If culture results are available, these should be used to de-escalate broad-spectrum antibiotics. Patients who do not improve within 48–72 h of receiving appropriate empirical treatment should be re-evaluated for differential diagnoses and for the development of complications of pneumonia. The concepts outlined in Figure 1 can be revisited. For instance, empyema could develop despite appropriate antibiotics, and the patient may require further chest imaging and drainage.

TAKE-HOME MESSAGES

1. Pneumonia is a clinical diagnosis marked by a combination of cough, phlegm production, fever and lung crepitations, which suggest infection of the lung parenchyma.
2. Even without any chest imaging or microbiological testing, *presumed* pneumonia based on clinical features alone needs prompt attention from frontline clinicians.
3. The general approach to pneumonia in adult patients requires consideration of differential diagnoses, risk stratification, appropriate antimicrobials, source control, organ support and treating related conditions (e.g. anatomical, neurological and immunological conditions).
4. Rapid triaging of pneumonia can be done with one of several risk scores (e.g. CRB-65, DS-CRB-65).
5. Patients with uncomplicated pneumonia should complete a short course of antibiotics, typically over 5–7 days.

Closing Vignette

The patient was diagnosed with community-acquired pneumonia. Based on her low blood pressure of 80/50 mmHg and peripheral oxygen saturation of 89% in room air, her DS-CRB-65 score was 2. Intravenous normal saline 500 mL and supplemental oxygen via nasal prongs were administered. An ambulance was called to bring her to the nearest emergency department. Her blood pressure improved to 120/90 mmHg after a further 1,000 mL of normal saline was administered. She was also given intravenous amoxicillin–clavulanic acid and oral azithromycin and admitted to the general ward. Her initial chest X-ray showed right-sided consolidation and no pleural effusion. Blood cultures did not have any bacterial growth. Polymerase chain reaction tests for COVID-19, influenza and tuberculosis were negative. Her fever resolved on the third day of hospitalisation, and she was discharged from the hospital with another 4 days of oral amoxicillin–clavulanic acid. Follow-up chest X-ray at 2 months showed complete resolution of the right-sided consolidation.

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Conflicts of interest

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SMC CATEGORY 3B CME PROGRAMME

Online Quiz: https://www.sma.org.sg/cme-programme

Deadline for submission: 6 pm, 10 April 2023

Question	True	False
1. In the absence of chest radiography, point-of-care lung ultrasound may be used to diagnose pneumonia.		
2. Genotyping for the detection of antibiotic resistance-related mutations may replace culture as the reference standard for drug susceptibility testing.		
3. For a test to be useful in ruling *out* a diagnosis/aetiology, its *specificity* should be high (e.g. ≥90%).		
4. Risk scores such as Pneumonia Severity Index, CURB-65 and the Infectious Diseases Society of America/ American Thoracic Society (IDSA/ATS) 2007 criteria require laboratory testing.		
5. In countries with mid-to-high tuberculosis prevalence, empirical use of respiratory fluoroquinolones for pneumonia should be avoided.		
6. A recommended indication for steroids is pneumonia with refractory septic shock.		
7. Adjunctive steroids can improve survival for all patients with *Pneumocystis jirovecii* pneumonia.		
8. Adjunctive steroids can improve survival for all patients with COVID-19 pneumonia.		
9. Most cases of bacterial pneumonia can be successfully treated with 5–7 days of antibiotics.		
10. Pneumonia with abscess formation, which can be diagnosed using computed tomography, requires an extended course over 4–6 weeks.		
11. Treatment of lung abscesses generally requires percutaneous drainage or surgery.		
12. Poorly draining pleural collections, which may be due to viscous pus or pleural septations, can be treated with intrapleural dual lysis (comprising DAase and tissue plasminogen activator).		
13. For adult patients with pneumonia, kidney replacement therapy is indicated when the blood urea exceeds 39.9 mmol/L or if oliguria persists for >72 h.		
14. A glucose target range of 4.4–6.1 mmol/L is reasonable in critically ill diabetic patients with pneumonia.		
15. Patients who receive appropriate therapy for pneumonia should improve clinically within 48–72 h.		
16. Radiological resolution of pneumonia takes 2–3 weeks for most patients.		
17. Serum C-reactive protein >10 mg/L is more specific than sensitive for diagnosis of community-acquired pneumonia.		
18. Serum procalcitonin >0.5 mcg/L is more specific than sensitive for diagnosis of community-acquired pneumonia.		
19. Serum mycoplasma IgG Ab may be used to diagnose acute mycoplasma lower respiratory tract infection.		
20. Influenza A/B polymerase chain reaction test using respiratory sample is highly sensitive and specific for the diagnosis of influenza lower respiratory tract infection.		