Aspiration pneumonia and pneumonitis

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SYNOPSIS

The two major aspiration syndromes are bacterial pneumonia and chemical pneumonitis. They have distinct features but may overlap. For chemical pneumonitis, supportive care is the mainstay of treatment while bacterial infection requires antibiotics. The choice of antibiotic is confused by conflicting evidence about the organisms responsible for infection, the poor yield from cultures in clinical practice and the lack of data comparing antibiotic regimens. Preventive strategies may help to reduce aspiration in vulnerable patients.

Index words: respiratory tract infection, antibiotics, corticosteroids.

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Introduction

Aspiration of oropharyngeal or gastric contents is a common, but often unrecognised cause of pneumonia. Focusing the management of pneumonia on the distinction between community- and hospital-acquired disease means that clinicians may not appreciate that aspiration can be a significant factor in both presentations.

Classification

Aspiration can occur in healthy individuals without sequelae. The development of infection depends on other factors such as the size of the inoculum, virulence of the organisms and the state of host defences such as glottic closure, cough reflex and immune status. Pneumonia may arise following ‘micro’ aspiration of virulent micro-organisms. However, the term aspiration pneumonia is reserved for pneumonia arising when the size of the inoculum is large and/or host defences fail.

Aspiration can be divided into two broad categories. This has important management implications. Aspiration of oropharyngeal contents, for example due to swallowing difficulty, will cause bacterial pneumonia with mouth organisms predominating. Aspiration of gastric contents will cause a chemical pneumonitis (e.g. Mendelson’s syndrome) because the gastric contents are usually sterile, but their acidity results in the rapid development of inflammation in the lungs. There may be overlap between pneumonia and pneumonitis, but it is usually possible to make the distinction and tailor treatment accordingly.

Other aspiration syndromes include airway obstruction due to a foreign body and exogenous lipoid pneumonia.

Epidemiology

Table 1 lists common causes of aspiration. Patients with stroke or a critical illness requiring intensive care usually have several of these risk factors and make up a large proportion of cases. Poor dental hygiene, especially in elderly or debilitated patients, results in colonisation of the mouth with potentially pathogenic organisms and/or increased bacterial load. This increases the risk of infection should aspiration occur.

Clinical features

The history, examination and chest X-ray help to differentiate between pneumonia and pneumonitis.

Aspiration pneumonia

The clinical features are often indistinguishable from other causes of pneumonia, for example cough, chest pain, dyspnoea, fever and consolidation on chest X-ray. The presence of aspiration may be obvious, for example patients with motor

Table 1

<table>
<thead>
<tr>
<th>Conditions that predispose to aspiration</th>
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<tbody>
<tr>
<td>Altered level of consciousness</td>
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<tr>
<td>• stroke</td>
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<tr>
<td>• seizures</td>
</tr>
<tr>
<td>• intoxication (alcohol and other drugs)</td>
</tr>
<tr>
<td>• head trauma</td>
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<tr>
<td>• anaesthesia</td>
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<tr>
<td>Mechanical disruption of usual defences</td>
</tr>
<tr>
<td>• nasogastric tube</td>
</tr>
<tr>
<td>• endotracheal intubation</td>
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<tr>
<td>• tracheostomy</td>
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<tr>
<td>• upper gastrointestinal endoscopy</td>
</tr>
<tr>
<td>• bronchoscopy</td>
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<tr>
<td>Neuromuscular disease</td>
</tr>
<tr>
<td>• multiple sclerosis</td>
</tr>
<tr>
<td>• Parkinson’s disease</td>
</tr>
<tr>
<td>• myasthenia gravis</td>
</tr>
<tr>
<td>• bulbar or pseudobulbar palsy</td>
</tr>
<tr>
<td>Gastro-oesophageal disorders</td>
</tr>
<tr>
<td>• incompetent cardiac sphincter</td>
</tr>
<tr>
<td>• oesophageal stricture</td>
</tr>
<tr>
<td>• neoplasm</td>
</tr>
<tr>
<td>• gastric outlet obstruction</td>
</tr>
<tr>
<td>• protracted vomiting</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>• recumbent position</td>
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<td>• general debility</td>
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neurone disease who cough when swallowing. However, aspiration is usually not witnessed and detecting it requires a high degree of suspicion. Some patients will have a relatively sudden onset typical of infection with common organisms such as *Streptococcus pneumoniae*. However, compared with other causes of pneumonia, aspiration pneumonia tends to have a more indolent course, evolving over days to weeks rather than hours. Patients may present with late complications such as weight loss, anaemia, lung abscess or empyema.

Chest X-ray usually shows consolidation in the lung segments which were dependent at the time of aspiration. If the patient was supine the posterior segments of upper or apical segments of lower lobes are involved. The basal segments of lower lobes are involved if the patient was upright. The subsequent course on X-ray is similar to pneumonia from other causes. Without treatment, patients have a higher incidence of cavitation and abscess formation.

**Aspiration of gastric contents**

In contrast to the aspiration of oropharyngeal secretions, aspiration of gastric contents is more likely to be witnessed or inferred. The original description of Mendelson’s syndrome involved obstetric patients undergoing ether anaesthesia. Witnessed aspiration was followed by respiratory distress and cyanosis within two hours. The women’s X-rays showed infiltrates in one or both lower lobes. Despite the severity of the illness, all the patients had recovered within 36 hours and there was radiographic resolution within seven days.

Subsequent studies of aspiration pneumonitis have shown a more fulminant clinical course resulting in the adult respiratory distress syndrome. The different clinical course probably reflects different study populations. The original patients were young, previously healthy women, while later studies often involved elderly, debilitated patients or those burdened with serious comorbid conditions.

**Microbiology**

There is conflicting information about the range of organisms responsible for aspiration pneumonia. The role of anaerobic organisms from the mouth seemed to be established in the 1970s and 80s using transtracheal and pleural aspiration to obtain specimens from the lower respiratory tract, avoiding the problem of contamination of expectorated sputum by normal mouth flora. The organisms include *Peptostreptococcus*, *Fusobacterium* and *Bacteroides*.

The use of protected brush specimens obtained via a bronchoscope in more recent times has yielded a different range of organisms, the identity of which depends on where the infection is acquired. *Streptococcus pneumoniae* predominates in community-acquired cases, *Staphylococcus aureus* and Gram negative organisms predominate in hospital-acquired cases. Anaerobic organisms have been conspicuously absent in these series.

There are a number of reasons for these contradictory results. In the studies that isolated anaerobes, the specimens were usually obtained late in the course of the illness after complications such as empyema or lung abscess had developed. Many of the patients were alcoholics and had foul sputum. They presumably represent only part of the spectrum of aspiration pneumonia. The studies that isolated aerobic bacteria were performed at an earlier stage of the illness, usually before antibiotics were used, and were taken from a wider range of patients with aspiration pneumonia.

Identifying organism(s) responsible for pneumonia is often attempted but not achieved in clinical practice for a number of reasons. These include contamination of sputum specimens with oropharyngeal flora, previous treatment with antibiotics and the difficulties using invasive techniques (such as bronchoscopy, transtracheal and transthoracic and pleural aspiration) that are more reliable at isolating pathogens. Anaerobic pathogens are difficult to identify even with good laboratory expertise.

**Management**

All patients need supportive care as well as specific treatment.

**Aspiration (chemical) pneumonitis**

The airway should be cleared of fluids and particulate matter as soon as possible after the aspiration of gastric contents is witnessed. Endotracheal intubation should be considered for those who are unable to protect their airway. Although it is common practice, there is no evidence that prophylactic use of antibiotics improves outcomes and, theoretically, this may make things worse by selecting out resistant organisms. Conversely, there may be difficulty distinguishing between purely chemical pneumonitis and bacterial infection. In clinical studies, up to 25% of patients develop superimposed bacterial infection during the course of chemical pneumonitis.

A reasonable compromise is to withhold antibiotics where the diagnosis is clear. Empirical antibiotic treatment should be considered if there is no improvement after 48 hours. If the pneumonitis cannot be distinguished from bacterial pneumonia or if patients have conditions known to be associated with colonisation of gastric contents (for example small bowel obstruction, or being in intensive care) immediate empirical antibiotic treatment is appropriate.

Corticosteroids have been used, with varying degrees of enthusiasm, for decades in the management of aspiration pneumonitis, but there are limited data to support this practice. Studies in humans are generally unsuccessful and sometimes the outcomes are worse for those treated with corticosteroids. Large randomised controlled trials of high dose corticosteroids for adult respiratory distress syndrome (of which chemical pneumonitis is a subset) showed no benefit. As a result, this treatment is not recommended.

**Bacterial aspiration pneumonia**

In contrast to chemical pneumonitis, antibiotics are the most important component in the treatment of aspiration pneumonia. Early empirical treatment is required for cases that are severe enough to warrant hospitalisation. Waiting for the results of
culture is unwise and will disappoint because of the low yield. Where practical, samples of blood, sputum and pleural fluid should be taken for culture before antibiotic use. If antibiotics have already been used, cultures may still be helpful in severe cases with a large organism load.

**Which antibiotic?**

The choice of antibiotic is influenced by the clinical setting (community- versus hospital-acquired), culture results, previous antibiotic use, and disease characteristics. The recommendations of *Therapeutic Guidelines: Antibiotic* for empirical therapy (see box) emphasise the importance of anaerobic infection and minimise the role of aerobic organisms. I believe the emphasis should be tilted to at least giving equal importance to aerobic infection to antibiotic use.

Antibiotic regimens which only aim at anaerobic infection are indicated if there is evidence of anaerobic infection (for example lung abscess or empyema with putrid sputum) and reasonable confidence there is no aerobic infection. Metronidazole used alone results in a significant treatment failure rate and should be used with another drug, usually penicillin. If penicillin allergy is a problem, clindamycin alone is adequate. I prefer clindamycin for intravenous treatment as it is more effective than penicillin alone and comparable to the combination of penicillin and metronidazole. Clindamycin is more convenient to administer and overall is as well tolerated, although its adverse effect profile differs from that of penicillin and metronidazole. Amoxicillin-clavulanate alone is also effective. For oral therapy, I prefer either amoxicillin-clavulanate or clindamycin alone over penicillin with metronidazole for similar reasons.

The value of other drugs commonly prescribed to treat pneumonia has not been systematically studied in anaerobic infection. It is likely that some of these, for example macrolides, second and third generation cephalosporins, may be ineffective as they lack activity against some anaerobes including the *Bacteroides* group. Drugs such as ciprofloxacin, aminoglycosides and trimethoprim-sulfamethoxazole have poor activity against all anaerobes. Imipenem, meropenem or any combination beta lactam/beta lactamase inhibitor (e.g. ticarcillin and clavulanate or piperacillin and tazobactam) are effective against virtually all anaerobic bacteria and should be effective against anaerobic pulmonary infection. They are attractive if there is concern about aerobic Gram negative infection or infection due to the commonly recognised respiratory pathogens (*Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus* and *Moraxella* species) as well as anaerobic infection. *Therapeutic Guidelines: Antibiotic* recommends adding gentamicin to penicillin/metronidazole or clindamycin where Gram negative infection is suspected. I prefer to use one of the regimens mentioned above as they are generally easier to administer, less toxic and do not require measurement of drug concentrations.

Depending on the clinical setting, anaerobic infection may be relatively uncommon compared with aspiration pneumonia due to aerobic organisms. These especially include *Streptococcus pneumoniae* and *Haemophilus influenzae* in community-acquired cases, and *Staphylococcus aureus*, aerobic enteric Gram negative organisms and *Pseudomonas aeruginosa* in hospital-acquired cases or where there has been previous antibiotic use. There is an array of antibiotic regimens for hospital-acquired pneumonia (which is largely caused by aspiration).

**Which route?**

The decision to use intravenous rather than oral therapy will depend on a number of factors. More severe illness requires more aggressive treatment and greater certainty that adequate doses of antibiotic are delivered to the lungs. In these cases, especially if there is doubt about gastrointestinal absorption, intravenous therapy is required. In less severe illness, oral therapy will often suffice. Switching from intravenous to oral therapy will be determined by the individual patient’s progress. In those who respond rapidly, oral therapy can often be introduced within 2–3 days.

**Other interventions for aspiration syndromes**

A semirecumbent, as opposed to supine, body position reduces the frequency of nosocomial pneumonia in patients receiving mechanical ventilation presumably by minimising gastro-oesophageal reflux and subsequent aspiration. A systematic approach to improving oral care in nursing home patients has also been shown to reduce the rate of pneumonia, presumably by reducing the oral organism load in these patients who have a high rate of aspiration.

Assessment of cough and gag reflexes is an unreliable method to identify stroke and other patients at risk of aspiration. A more comprehensive evaluation of swallowing, usually by a speech pathologist, is required. This may include a videofluoroscopic swallowing study and/or endoscopic evaluation. In patients at risk, behavioural, dietary and medical interventions may reduce risk. For swallowing dysfunction, a soft diet is often used along with feeding strategies, for example keeping chin tucked, small bite size.

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**Therapeutic Guidelines: Antibiotic**

**recommendations for aspiration pneumonia**

For severe aspiration or lung abscess:

- benzylpenicillin 1.2 g intravenously 4–6 hourly + metronidazole 500 mg intravenously 12 hourly

If hypersensitive to penicillin:

- use clindamycin 600 mg intravenously eight hourly as a single drug

If Gram negative pneumonia is suspected:

- add gentamicin 4–6 mg/kg intravenously daily; alternatively, as a single agent use ticarcillin + clavulanate 3.1 g intravenously six hourly OR piperacillin + tazobactam 4.5 g intravenously eight hourly
There are contradictory aspects to the issue of tube feeding and aspiration pneumonia. It is reasonable, but unproven, to believe that tube feeding will reduce the risk of aspiration pneumonia in some patients with swallowing difficulties.

The contradiction lies in the role of tube feeding in causing aspiration pneumonia. Feeding tubes offer no protection from aspiration of oral secretions (and nasogastric tubes may make it worse). Aspiration of gastric contents can still occur and aspiration pneumonia remains a common cause of morbidity and death in patients fed this way. Percutaneous endoscopically-placed gastrostomy tubes are not superior to nasogastric tubes when it comes to preventing aspiration pneumonia. However, for long-term use they are more convenient and more acceptable to patients who cannot be adequately fed by conventional means. Tube feeding may be recommended for patients who continue to aspirate despite other preventive strategies (the bulk of these are stroke patients). This decision will also rest on patient preference, prognosis and other indications for tube feeding, such as nutritional maintenance.

Conclusion

Pulmonary aspiration is a significant cause of morbidity and mortality in a wide range of patients. Identifying and differentiating between the various aspiration syndromes is largely a clinical/epidemiological skill. Treatment is usually empirical, and therefore adequate differentiation between types of aspiration is needed to achieve best outcomes. Preventive strategies have recently been shown to reduce the rate of aspiration syndromes.

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REFERENCES


FURTHER READING


Conflict of interest: none declared

Self-test questions

The following statements are either true or false (answers on page 23)

7. Patients with aspiration pneumonitis should be given a bolus dose of corticosteroids as soon as possible after the aspiration.

8. Aspiration pneumonia does not occur in patients who are tube fed.