Complications of Oropharyngeal Dysphagia: Aspiration Pneumonia

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Abstract

The incidence and prevalence of aspiration pneumonia (AP) are poorly defined. They increase in direct relation with age and underlying diseases. The pathogenesis of AP presumes the contribution of risk factors that alter swallowing function and predispose to the oropharyngeal bacterial colonization. The microbial etiology of AP involves Staphylococcus aureus, Haemophilus influenzae and Streptococcus pneumoniae for community-acquired AP and Gram-negative aerobic bacilli in nosocomial pneumonia. It is worth bearing in mind the relative unimportance of anaerobic bacteria in AP. When we choose the empirical antibiotic treatment, we have to consider some pathogens identified in oropharyngeal flora. Empirical treatment with antianaerobics should only be used in certain patients. According to some known risks factors, the prevention of AP should include measures in order to avoid it.

Introduction

Definition

We speak of aspiration pneumonia (AP) when we have radiological evidence of pulmonary condensation caused by a large quantity of secretions contaminated by pathogenic bacteria that have passed to the tracheobronchial tree in patients with impaired oropharyngeal or gastroesophageal motility [1]. The location of the radiological sign will depend on the posture of the patient at the time of aspiration. If the patient was upright or semi-recumbent, the affected part will be in the basal segments of the lower lobes, and if the patient was recumbent the
most commonly affected areas are in the posterior segment of the upper right lobe and/or the apical segment of the lower right lobe [2].

**Incidence and Prevalence of Aspiration Pneumonia**
The incidence and prevalence of AP is little known as most epidemiological studies on pneumonia consider cause from aspiration to be criteria for exclusion. In population-based studies, AP accounts for 1.2% of all community-acquired pneumonia (CAP) in persons >14 years of age, an incidence which increases with age [3]. Considering CAP patients who require hospitalization, AP is the cause of 6% of cases, and can reach 10% in patients over 80 years of age [4]. Nursing home residents and particularly those considered frail, however, are most at risk of pneumonia with an incidence of up to 10 times greater than in non-institutionalized older persons [5], with corresponding increased mortality. AP affects more than 30% of patients with stroke and is the main cause of mortality in the first year. About 60–80% of patients with neurodegenerative diseases suffer chronic aspiration of oropharyngeal secretions and the retention of secretions or pneumonia, and AP is the main cause of mortality. It is also the third cause of mortality in patients over 85 years of age [6].

**Pathophysiology and Risk Factors**
The material aspirated into the tracheobronchial tree must be colonized by bacteria for AP to occur [7]. Aspiration of sterile secretions may cause chemical pneumonitis [4] but not pneumonia. The pathogenesis of AP thus depends on the coexistence of two main groups of risk factors (fig. 1): (a) factors that affect oropharyngeal and/or gastroesophageal motility and (b) factors that favor bacterial colonization in oropharyngeal or gastroesophageal secretions. In the present chapter, we will mainly describe those factors that can influence oropharyngeal colonization.

**Risk Factors for Oropharyngeal Colonization**

**Age**
Age increases oropharyngeal colonization by certain bacteria such as *Staphylococcus aureus* and aerobic Gram-negative bacilli (GNB) such as *Klebsiella pneumoniae* and *Escherichia coli* associated with greater comorbidity [8].

**Poor Dental Hygiene**
The relation between dental and respiratory infection is well known. El-Solh et al. [7] found that there was an 80% coincidence between the bacteria found in the respiratory tract and in the dental plaque of patients from nursing homes who were admitted to the intensive care unit (ICU). ICU patients also had greater risk of nosocomial pneumonia when they had bacterial colonization of
dental plaque [9], and the risk was reduced with prophylactic antiseptic treatment. This suggests that dental plaque which has been colonized by aerobic micro-organisms acts as a microbe reservoir [10], and it follows that it is more evident in patients with dental prostheses than those with their own teeth.

**Malnutrition**
In addition to being directly related to dysphagia, malnutrition, evaluated from albumin levels and/or body mass index, can be an independent risk factor for pneumonia [11]. Malnutrition has also been described as a factor for poor prognosis associated with a higher mortality rate at 30 days, is clearly associated with mortality at 1 year and very closely associated with the presence of oropharyngeal dysphagia [12]. Thus, dependence, dysphagia, malnutrition and immune status must be considered as major prognostic indicators in elderly patients with pneumonia, as also indicated by other authors [11]. The influence nutrition, particularly vitamin content, has on the immune system could be the cause, although this hypothesis needs to be confirmed in further studies designed to this end.

**Smoking**
A direct relation, independent of other factors, has been found between smoking and the appearance of CAP [13], and this influence disappears when the habit is given up. The reason for this may be that smoking influences the mechanisms
of the defense system of the host and/or the surface of the mucosa in the respiratory tract by either increasing the number of abnormal cilia or impairing the permeability of the epithelium and thus weakening cilia clearing and favoring bacterial adhesion and consequent colonization.

Antibiotherapy
Several studies on patients diagnosed with CAP have found that antibiotherapy prior to diagnosis affected respiratory infection by some bacteria [14]. This effect was attributed to the selection of oropharyngeal flora with the appearance of more virulent microorganisms.

Inhalers and Aerosols
The devices used to administer treatment to patients with asthma or chronic bronchopathologies can be the source of oropharyngeal contamination as our group recently demonstrated in a case-control study [15]. We found that the role of the inhaled drugs as risk factors for CAP seemed to differ according to the baseline respiratory disease. Inhaled anticholinergics but not inhaled steroids favored CAP in patients with asthma, while inhaled steroids increased the risk for CAP in COPD patients but not in chronic bronchitis patients. This was in agreement with the hypothesis that poor hygiene may represent a causal component of the mechanism of infection. Indeed, the effect of inhalers on the development of CAP can be attributed to the active medication contained in the metered-dose inhaler.

Dehydration
Salivary flow and deglutition play a major role in preserving normal oropharyngeal flora by eliminating GNB. Insufficient liquid intake or decreased salivary production may impair this in older patients with functional incapacity [16] or under certain medication (diuretics, antihistamines, anticholinesterases, antidepressants and drugs for Parkinson's and hypertension), with the consequent increase in bacteria in the oropharyngeal cavity.

Reduction in the Effectiveness of the Immune System
Related to age, the peripheral T cells show signs of weakness when competing with antigens [17].

Special Situations
**Nasogastric Tubes.** The biofilm on the outer surface favors growth of microorganisms and increases the possibility of septic pseudoembolism in the oropharyngeal cavity, altering the oropharyngeal ecosystem and increasing colonization of the upper airways [18].

**Increased Gastric pH.** Although gastric juices are sterile due to their acidity, the pH can increase, favoring gastric and oropharyngeal colonization [19].
This has been described in patients with gastroparesis, small-bowel obstruction, enteral nutrition or those under treatment with anti-H₂ or proton pump inhibitors. However, we must be careful when relating acid suppressants, particularly proton pump inhibitors, with pneumonia as the study has been criticized for not using an appropriate control group.

Orotracheal Intubation. The risk of developing nosocomial pneumonia in intubated patients in the ICU is 5 times greater than those not intubated, and is directly related to the duration of intubation [20] and to the need for reintubation. The process can be explained in the following way: bacteria from the pharynx and stomach contaminate subglottic secretions, creating a reservoir which can be aspirated to the trachea, forming a biofilm around the endotracheal tube and which can then be disseminated in the lung through mechanical ventilation.

Microbiology of Aspiration Pneumonia

Basically, the pathogens that contaminate the nasopharynx and oropharynx are those responsible for AP [1]. Strong correlation has been found between cultures of dental plaque samples and those of bronchoalveolar lavage in patients with nosocomial pneumonia associated with mechanical ventilation. The most common are *Haemophilus influenzae* and *Streptococcus pneumoniae*, but in older persons the upper airways can be colonized by aerobic GNB (enterobacteria and *Pseudomonas aeruginosa*) and Gram-positive cocci such as *S. aureus*. Oropharyngeal colonization by aerobic GNB can affect 22–37% of nursing-home residents; the same occurs in patients admitted to the ICU where 60–73% of those over 65 years of age have been found to be contaminated with microorganisms [21]. These groups of patients also have a greater overall risk of infection by resistant *S. pneumoniae*. However, it is difficult to find large series of AP with microbiological diagnosis due to the difficulty of applying invasive fibrobronchoscopical techniques. In two recent studies on AP patients, where the authors used similar methodology [22] with cultures of bronchial secretions obtained with a telescopic catheter, 31 pathogens were isolated from a total of 77 patients. The most diagnosed were *S. aureus*, *H. influenzae* and *S. pneumoniae* in extra-hospital pneumonia, and aerobic GNB (*Klebsiella pneumoniae*, *Escherichia coli*, *Serratia narcenses* and *Proteus mirabilis*) in nosocomial pneumonia. It is worth noting the scarce implication of anaerobic pathogens contrary to earlier belief [22]. In a recent series by El-Sohl et al. [23] on 95 nursing home residents over 65 years of age that were admitted to the ICU for AP, microbiological diagnosis obtained for 54 patients revealed 20% were anaerobic (*6 Prevotella* sp, *3 Fusobacterium* sp, *1 Bacteroides* sp and *1 Peptostreptococcus* sp), but the majority also had enteric GNB, and the authors emphasized the clinical resolution within 72 h without the use of anaerobicides. This differs from
studies published in the 1970s in which anaerobic pathogens were given greater importance, but the methodology employed then is being questioned now [2]: microbiological cultures were made on samples obtained by transtracheal puncture which only revealed one contamination. They were also performed when the infection was advanced, often following complications such as abscesses, necrotizing pneumonia or empyema and on patients with chronic alcoholism most of whom presented purulent sputum, quite different from the patients we usually treat nowadays.

Antibiotic Treatment of Aspiration Pneumonia

The choice of antibiotic depends on the place of acquisition of AP and the patient’s prior state of health. Although AP is often treated empirically with penicillin or clindamycin, this may be inadequate for most AP given the normal microorganisms of the oropharyngeal flora such as *S. pneumoniae*, *H. influenzae* and GNB. The need for empirical treatment of anaerobic pathogens is not demonstrated as several studies have shown better clinical results without this antibiotic treatment. In any case, this treatment should be reserved for patients with major periodontal disease, purulent sputum or radiological evidence of necrotizing pneumonia or lung abscess. Nevertheless, the current guidelines for the treatment of suspected AP [24] recommend using intravenous amoxicillin-clavulanic acid (2 g amoxicillin/8 h) for 14 days. Moxifloxacin, ertapenem or clindamycin with a third-generation cephalosporin are alternatives. Cephalosporin should be replaced by piperacillin-tazobactam combination if ICU admission is needed. Above all, possible local resistances must be taken into account and treatment adjusted accordingly.

Prevention

Prevention and treatment of AP are currently possible. According to the two pathophysiological pillars on which the appearance of AP is based, we can intervene both in the prevention of oropharyngeal and gastropharyngeal colonization and in the treatment of oropharyngeal dysphagia which allow endobronchial aspiration (fig. 2). We can prevent oropharyngeal colonization in the following ways:

1. Administration of antipneumococccic and anti-influenza vaccines, preferably together.
2. Meal programs can improve nutritional risk and results suggest that such interventions may help reduce risk of CAP in older persons.
3. Advise giving up smoking.
4. Care of oral hygiene has been shown to reduce colonization by virulent pathogens and the incidence of pneumonia. Intensive oral hygiene improves
Fig. 2. The relationship between pathogenesis, risk factors, and preventive strategies for ventilator-associated pneumonia (VAP).
sensitivity to cough reflex as the changes produced by contaminating pathogens in the mucous and nerve endings are improved. Programs to clean dental prostheses should be established as well as the habit of wearing dentures during daytime only [25]. Oral hygiene should be maintained even among edentulous patients as ‘tongue cleaning’ is associated with decreased oropharyngeal bacterial load.

5 Nasogastric tubes do not prevent aspiration in patients with dysphagia and advanced dementia and so should only be used when strictly necessary. With regard to 24-hour enteral nutrition, interruptions of 6 h are recommended so the gastric pH can return to normal and eradicate bacteria that could otherwise cause oropharyngeal contamination [21].

6 Hand washing with antibacterial soap (chlorhexidine) followed by alcoholic solution before and after contact with the patient.

7 Avoid administration of sedatives in order to prevent relaxation of the oropharyngeal musculature. Antihistamines and anticholinergics should also be avoided.

8 Maintenance of good hydration of the oral cavity.

9 Avoid unnecessary antibiotic treatment although there is not enough evidence yet to show a reduction in the incidence of pneumonia.

10 Prophylaxis with anti-H₂ and proton bomb inhibitors should be restricted to patients who present recurrent pneumonia during treatment, at least until more scientific evidence is available [26].

11 Maintenance of good hygiene and dryness of pressurized devices and aerosols used in bronchodilator therapy.

12 Noninvasive mechanical ventilation in patients admitted for respiratory insufficiency is preferable as, among other advantages, it has a lower incidence of nosocomial pneumonia.

13 Subglottic aspirations are effective in patients that require more than 3 days’ orotracheal intubation. Selective digestive decontamination (mouth and stomach) with polymyxin + aminoglycoside + amphotericin is recommended to reduce the gastric bacterial load and avoid tracheobronchial colonization, thus reducing the incidence of pneumonia associated with mechanical ventilation. Similar results have been published on the decontamination of the oral cavity with chlorhexidine or chlorhexidine + colistin. Studies that question these results have prevented generalized use, but considering cost/benefits and the fact that they do not induce resistance to antibiotics, oral decontamination with chlorhexidine is an attractive method of reducing pneumonia.

14 Consider raising hospitalized patients in bed to a 45° angle [27], even though effectiveness has not been demonstrated in patients outside the ICU.
References
