Nosocomial pneumonia

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Abstract

Nosocomial pneumonia causes significant morbidity and mortality in hospital inpatients. The highest risk of nosocomial pneumonia is in Medical Intensive Care Units, particularly in intubated and mechanically ventilated patients (20-67%). Risk factors have been assessed for all hospital inpatients, both those admitted in Intensive Care Units and for those mechanically ventilated. The diagnosis typically requires the emergence of a new infiltrate on the chest X-ray, fever, leucocytosis and purulent secretions occurring more than 48 hours after admission. The differential diagnoses of fever and/or densities on chest radiographs have been considered. Pseudomonas aeruginosa is the most common pathogen (16-31%), with Staphylococcus aureus the second pathogen most often found (8-20%). The treatment is with supportive measures and antimicrobial therapy that is often empirical. If Pseudomonas aeruginosa is suspected, empirical therapy should be considered. The duration of antibiotic therapy can last for 21 days and it depends on the severity of illness, alterations on the chest X-ray and the pathogen.

Key words: nosocomial pneumonia, Pseudomonas aeruginosa, Medical Intensive Care Units.

Definition

Nosocomial infections are acquired infections in a hospital environment, which were not present, even in incubation stage, at the time of admission. [1] In the first place, there are the genital urinary tract infections (40%), followed by the surgical (25%) and only then pulmonary infections (15%). [2] Nosocomial pneumonia is defined as an infection of the lower airways which was not present, even in incubation stage, at the time of hospital admission. [1] The risk of acquiring nosocomial pneumonia is 0.5 – 1% in all hospitalized patients, that in patients admitted to intensive care units the risk increases to 7 – 20%. [1,3] In ventilated patients the risk ranges from 21 to 67%, depending on the patient's group: surgical or multi-traumatized patients are at a higher risk. For each day of assisted ventilation, the added risk is 1+/-0.76%. That is: on the 10th day of ventilation, 6.5% of patients acquire nosocomial pneumonia. [1]

Risk factors

The disease severity is well known as the determining factor whilst increasing the risk of infections acquired in the hospital environment.

The risk factors were considered for the hospital general population, for the intensive care unit patients and for ventilated patients and are as follows:

1) The hospital general population:
- Age above 70 years old;
- Chronic pulmonary disease;
- Thoracic-abdominal surgery;
- Changes of awareness;
- Orotracheal intubation;
- Big quantities of aspirate.

2) Intensive care unit
- Change in the airways reflexes;
- Apache II above 16;
- Gastrointestinal hemorrhage prophylaxis;
- Sedation;
- Mechanical ventilation for over 48 hours;

3) Pneumonia associated with the ventilator:
- Monitoring of intracranial pressure;
- Cimetidine;
- Change of the ventilator circuits every 24 hours.[1,4]

Pathogenesis

The retention of tracheobronchial secretion, the aspirate of the oropharynx content and the colonization of the upper airways by gram negative bacteria, as well as the change in all pulmonary defense mechanisms in critical patients, have an essential role in the pathogenesis of nosocomial pulmonary infections. Most of

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such infections are endogenous, being the source of the infection the patient's oral pharyngeal or gastrointestinal flora. The change in the pulmonary or humoral defense mechanisms is the main factor for pneumonia evolving or not to the microorganisms' aspiration.[1]

The hematogenous nosocomial pneumonia occurs more frequently as a result of catheter sepsis by Staphylococcus aureus.[3]

Pulmonary defenses are a mechanical barrier (epiglottis, coughing reflex, glottis closure reflex and mucociliary clearance), alveolar phagocytes, humoral factors (lipoprotein, glycoprotein, fibronectin, IgG and C3). Such defenses may be altered in conditions of alcoholism, diabetes mellitus, leukopenia, kidney failure, viral infection, primary respiratory disease, orotracheal intubation, nasogastric intubation, poor nutritional condition, hypoxemia and drugs (antibiotic, hypnotic, and non-steroids anti-inflammatory drugs, steroids…).[5]

Prophylaxis

In regard to the prophylaxis of nosocomial pneumonia, some arguably arguments focused and include: selective digestive decontamination, enteral nutrition, gastric protection and the change of ventilators trachea.

The selective digestive decontamination consists in the prophylactic use of antibiotic to eradicate pathogenic microorganisms from the gastrointestinal system, namely gram negative as enterobacteria and Pseudomonas aeruginosa, Staphylococcus aureus and fungi, in a way of reducing the incidence of infections in the respiratory system when there is aspiration of the gastric content. Several regimes can be used, using polymixin e with tobramycin and B amphotericin and tobramycin being replaced by gentamicin, norfloxacin or axtreonam and B amphotericin by nystatin.

Intermittent enteric nutrition seems to have benefits regarding permanent nutrition, because it reduces the gastric pH that in turn reduces the colonization of the gastrointestinal system by pathogenic organisms.

In the prophylaxis of digestive hemorrhage, sucralfate is used instead of cimetidine because it does not change the gastric pH and consequently does not predispose to colonization.

The change of the ventilators corrugated pipes should be made every 48 hours, instead of 24 hours, because it reduces handling and therefore the colonization. [2, 3]

Diagnosis

Nosocomial pneumonia diagnosis is made in the presence of new infiltrate in the thorax x-ray, fever, leukocytosis and purulent secretion, occurring 48 hours or more after hospital admission.[2,3,6]

The center for disease control definition for nosocomial pneumonia diagnosis combines the use of clinical, radiological, and microbial criteria.[6]

Purulent secretion are considered as adequate when revealing 25 or more polymorphonuclear leukocytes and less than 10 epithelial cells per small increase field, and diagnosis when there are over 5 – 7% of intracellular bacteria and a pathogenic agent is identified by Gram staining and cultural tests. [3] When finding 5 – 7% or more intracellular bacteria seems to be a pneumonia specific marker.[6] The presence of elastin fibers in the endotracheal aspirates are highly specific (100%) in the presence of necrotizing pneumonia.[5]

The low credibility of clinical criteria to detect nosocomial pneumonia drove the need of researching and using invasive and non-invasive techniques for the diagnosis of such pulmonary infections.[6]

In cooperating patients, it is possible to make a sputum collection or inducing secretions, if they have no expectoration. The aspiration of secretions by orotracheal tube or tracheostomy should not be performed due to the high probability of contaminating the oropharynx content and the culture low specificity.

The invasive diagnostic methods are:

1 - Bronchofiberscopy where it is possible to have a protected brushing, a Bronchoalveolar Washing and transbroncheal biopsy;

2 - Percutaneous pulmonary aspirate;

3 - Pulmonary biopsy.[6]

With the secretions obtained by protected brushing, the culture with over 10^3 colony forming units/ml (CFU /ml) is considered an infection.

The bronchoalveolar washing has low specificity but it is more sensitive. The threshold distinguishing colonization from infection is 10^5 colony-forming units/mL. If over 25% of cells found have intracellular microorganisms it can be said we are before a pneumonia. Such procedure is advantageous and should be used for the diagnosis of pneumonia by cytomegalovirus and Pneumocystis carinii, in this last case with a 90 – 95% sensitivity. [5,6]

Transbroncheal biopsy is useful for the diagnosis of

a central lesion.[5] Percutaneous pulmonary aspiration is contraindicated in ventilated patients. It is useful in peripheral cavity lesions and pulmonary abscesses by anaerobic agents. It has a high percentage of false negative.[5]

Pulmonary biopsy is the most invasive procedure. There is no risk of contamination and complications occur in 10% of cases (hemorrhage and pneumothorax).[5]

Differential diagnosis

Radiological evidence of pulmonary changes requires careful interpreting. For instance, the precision as the thorax x-ray can make a diagnosis of pneumonia in ventilated patients is only 52%. More precise results can be obtained when there is clinical evidence, mainly breathing symptoms.

The existence of pulmonary opacities can reflect pulmonary edema, adult respiratory distress syndrome, aspiration pneumonia, atelectasis, pulmonary contusion, embolism, neoplasms, uremic pneumonitis, chemical pneumonitis (e.g.: radiation) or any combination of such entities.[7]

It is still harder to distinguish from other processes characterized by fever and pulmonary densities as, for instance, atelectasis, catheter related infection, Clostridium difficile colitis, congestive cardiac insufficiency, deep vein thrombosis, peritonitis, reactions to drugs (e.g. amiodarone), primary bacteremia, pulmonary fibroproliferation, sinusitis and urinary tract infection.[8]

Etiology

Gram negative bacteria account for most nosocomial pneumonia, i.e., from 70 to 85% (*Table 1*).

However the range of microorganisms known to cause nosocomial pneumonia has been in the increase.

These include bacteria as: Staphylococcus epidermidis, Corynebacterium, non-rated Haemophilus influenzae, Moraxella catarrhalis and others.

Fungi including Candida and Aspergillus and virus including influenza and respiratory syncytial virus are capable of causing nosocomial infection and pneumonia.[5]

Gram negative bacilli are at present the main cause of bacteremia and nosocomial pneumonia. Pseudomonas aeruginosa is the most common pathogen agent, accounting for 16 – 31% of cases.[5,9] Pneumonia by Pseudomonas aeruginosa occurs more frequently in neutropenic patients with cystic fibrosis and in those receiving wide spectrum antibiotherapy. It can be acquired by aspiration of the oropharynx flora or, secondarily, by inhaling of nebulizer reservoirs. It is characterized by fever, severe dyspnea and hypoxemia. Thorax x-ray revealed a bilateral bronchopneumonia with infiltrate and small pleural effusions.[5]

Staphylococcus aureus is the second more common cause accounting for 8-20% of cases. Nosocomial pneumonia caused by this agent can be secondary to aspiration or hematogenous invasion, particularly by catheters or other infected vein access device. It produces a necrotizing pneumonia with cavities, abscesses or pneumatoceles.[5]

Streptococcus pneumonia and Haemophilus influenza, the most often implicated agents in community-acquired pneumonia accounts for 20 – 40% of nosocomial pneumonia.

Gram positive cocci were isolated in 20-30% of patients but simultaneously with Gram negative.[9]

Therapy

Treatment of nosocomial pneumonia includes support and antimicrobial therapy approaches.

Support approach: physiotherapy, oxygen therapy, use of painkillers and antipyretic drugs. Physiotherapy – including postural drainage, percussion and aspiration of secretions – is used to mobilize purulent secretions. Oxygen therapy is used in critical patients with documented pneumonia and hypoxemia. Often it is necessary to resort to mechanical ventilation. Analgesia is often needed for polypneic patients with intense coughing, being codeine usually adequate.

Some patients, particularly those with pneumococcus pneumonia may develop gastric and ileus distension, being necessary nasogastric intubation.

Antimicrobial approach, and as mentioned before, the first step to choose an antimicrobial agent is determined by the history and physical examination together with the Gram obtained by the culture of secretions. In intensive care units admitted patients is often necessary to resort to bronchoscopy.[5]

The initial treatment of a nosocomial pneumonia is often empirical, with therapy addressed to the suspected pathogenic agent, before the microbiology diagnosis is well established [9] (*Table 2*). When choosing a certain antibiotic as being optimum, it should be considered its efficacy, toxicity, cost and the potential of emerging resistant organisms.

The use of a wide spectrum antibiotic given by parenteral route as monotherapy is an attractive concept. Monotherapy with third-generation cephalosporin, wide spectrum penicillin, imipenem or fluorquinolone has shown to be effective for community-acquired moderate pneumonia or nosocomial pneumonia caused by susceptible agents. However it is not useful for severe nosocomial pneumonia and it can even potentiate an increase of microbial resistance.

Nosocomial pneumonia empiric therapy with aminoglycosides is controversial. However if associated to a better lactam, they have shown a synergistic effect in vitro. When Pseudomonas aeruginosa is suspected, empiric therapy with antipseudomonic penicillin (e.g.: piperacyllin- tazobactam), ceftazidime, imipenem or ciprofloxacin. If the agent is isolated, adding an aminoglycoside has a synergic action.[9]

When there is a suspicion of a Staphylococcus aureus pneumonia it can be considered as methicillin resistant, a case where the therapy should be carried out with vancomycin; or, still, it can be considered as methicillin sensitive where the therapy is carried out with oxacillin or nafcilin.[5]

Treatment Duration

The duration of the treatment should be individualized for each patient based on the severity, pathogenic agent and the drug clearance rate.

Prolonged therapy (for a minimum of 14 to 21 days), is reserved for severe pneumonia, multi-lobular or necrotizing or for infections caused by very virulent organisms, as Pseudomonas aeruginosa or Acinetobacter species.

A period of 7 to 10 days of antibiotherapy can be appropriate in moderate pneumonia caused by H. influenza, responding quickly to therapy.[9] ■

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