NEW TRENDS FOR PROPHYLAXIS AGAINST INTENSIVE CARE UNIT-ACQUIRED PNEUMONIA

An Essay
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INTRODUCTION

Pneumonia is one of the most common nosocomial infections occurring in hospitalized patients especially in ICU patients that occurs more than 48 hours after admission and without any antecedent signs of infection at the time of ICU admission **(American Thoracic Society, 2005).**

Many studies have investigated about the risk factors for development of pneumonia in intensive care units and its consequences these include old age, male gender, hospitalization prior to ICU admission, length of ICU stay, treatment in large hospitals, a low Glasgow Coma Scale (GCS), respiratory failure, congestive heart failure, acute renal failure and dialysis, bronchoscopy, tracheostomy, re-intubation, duration of mechanical ventilation, detection of certain multi drug resistant pathogens, use of central vein catheters, bacteraemia, enteral feeding, and application of sucralfat or corticosteroids **(Martin et al., 2008)**.

The most important factor influencing the mortality of ICU acquired pneumonia is prompt and adequate empiric treatment. Multiple studies have demonstrated that delays in appropriate antibiotic therapy are associated with increased mortality **(Garcia et al., 2007)**.

Data have accumulated to support interventions for prevention of ICU acquired Pneumonia, yet translation into

practice is lacking. The focus should be addressing modifiable risk factors such as endotracheal and nasogastric tubes, tracheostomy, reintubation, enteral nutrition, corticosteroid administration, gastric pH-modifying agents and prophylatic antibiotic use **(Steven et al., 2006)**.

There have been several efforts to reduce the development of ICU-acquired pneumonia using various strategies including selective bowel decontamination and administration of local antimicrobial agents via the respiratory tract **(Matthew et al., 2006).**

AIM OF THE WORK

It is to study the new prophylactic measures which could be used for prevention of development of ICU-acquired pneumonia.

Chapter (1)
PATHOPHYSIOLOGY AND RISK FACTORS

Definitions

ICU-acquired pneumonia

It is a respiratory infection developing more than 48 hours after intensive care unit admission with new and persistent infiltrate (radiographically present for > 48 hours), plus at least two of the following criteria: [1] Core temperature > 38.5 or < 36°C, [2] Blood leukocytes > 10/µl or < 4/µl or [3] Purulent tracheal secretions **(Masterton et al., 2008).**

Ventilator associated pneumonia (VAP):

VAP is a common type of ICU acquired pneumonia which includes all patients receiving mechanical ventilation at the time of infection **(Richards et al., 1999).**

According to its onset VAP can be divided into 2 types: early and late. Early-onset VAP occurs 48 to 96 hours after intubation and is associated with antibiotic-susceptible organisms. Late-onset VAP occurs more than 96 hours after intubation and is associated with antibiotic-resistant organisms **(Kollef et al., 1999).**

Epidemiology:

- *Incidence of ICU-acquired pneumonia*
  - It is the second most common nosocomial infection.
  - Rate 5-15 cases/ 1000 ICU admission.
  - Increase 6 to 20 folds in mechanically ventilated patients.
  - Increases hospital stay by 7 to 9 days / patient.
  - Excess cost for every patient.
  - Mortality rate is seriously high (about30- 50 %).

(Coleman et al., 2008)

- *Ventilator Associated Pneumonia (VAP)*
  - Most vigorous type of ICU acquired pneumonia occurs 9–27% of all intubated patients.
  - Crude rate of VAP is usually 1-3% per day of intubation & mechanical ventilation.
  - Rates are greatly higher in surgical ICU patients than in medical ICU ones. There are two possible explanations for this: firstly, there is an effect independent of whether they acquire pneumonia during their ICU stay, and secondly via a pneumonia which extends their ICU stay as well (**Chastre et al., 2002**).

Mortality of VAP

- Crude mortality rates for VAP ranges from 20-71%, but deaths are often due to other causes in critically ill patients.
- A preferred measure is “Attributable mortality”, defined as percentage of deaths that would not have occurred in the absence of infection.
- Attributable mortality of VAP ranges from 27-43% (**Beth Augustyn, 2007**).

Risk factors for ICU - acquired pneumonia

1- Risk factors in ventilated patient:

Although any patient with an endotracheal tube in place for more than 48 hours is at risk for VAP, certain patients are at higher risk (Figure 1) **(Masterton et al., 2008).**

The risk factors for VAP can be divided into 3 categories: host related, device related, and personnel related **(Kollef et al., 2004).**

Host-related risk factors include:

- Pre-existing conditions such as:
  - Immunosuppression.
  - Chronic obstructive lung disease
  - Acute respiratory distress syndrome **(Torres et al., 1992)**.

- Patients' body positioning.

  Bacterial contamination of endotracheal secretions is higher in patients in the supine position than in patients in the semirecumbent position **(Torres et al., 1992)**.

- Level of consciousness.

  Whether due to a pathophysiological process, medication, or injury, decreased level of consciousness resulting in loss of cough and gag reflexes contributes to the risk of aspiration and therefore increased risk for VAP **(Schleder, 2003)**.

- Number of intubations.

  Reintubation and subsequent aspiration can increase the likelihood of VAP 6-fold **(Torres et al., 1995).**

- Medications, including sedative agents and antibiotics.

(Sopena et al., 2005)

Device-related risk factors:

1) Endotracheal tube, increase risk is due to:
   - Secretions pool above the cuff of an endotracheal tube,
   - Low cuff pressures can lead to microaspiration
   - Leakage of bacteria around the cuff into the trachea
2) Ventilator circuit
3) Nasogastric or an orogastric tube.

(Ferrer et al., 2001)

Personnel related risk factors:

- Improper hand washing resulting in cross-contamination of patients is the biggest personnel-related risk factor for VAP. Patients who are intubated and receiving mechanical ventilation often need interventions such as suctioning or manipulation of ventilator circuit. These interventions increase the likelihood of cross-contamination between patients if healthcare staff does not use proper hand-washing techniques. Failure to wash hands and change gloves between contaminated patients has been associated with an increased incidence of VAP **(Kollef et al., 2004).**
- In addition, failure to wear proper personal protec- resistant organisms have been identified increases the risk of cross-contamination between patients **(Tablan et al., 2004).**

2- Risk factors in non ventilated patient:

There is a wide variety of risk factors for Non-Ventilator Associated Hospital-Acquired Pneumonia (NVHAP). They vary according to the population from which cases are selected. For instance, risk factors among patients who were not submitted to surgical procedures differ from those reported for post-operative NVHAP**: (Windsor et al., 1988**)

1. Age was a significant risk factor for NVHAP the greater incidence of pneumonia among elderly people.

2. Nutritional status.
3. Smoking and / or alcoholism.
4. Comorbid illnesses (Renal and Central Nervous System [CNS] diseases. CNS diseases may depress cough reflexes, impair swallowing mechanisms and affect respiratory patterns. All these alterations facilitate the access of microorganisms to the lower airways**.**
5. Invasive devices (Central Venous Catheter, Urinary Catheter, Naso-enteral tube).
6. Longer hospital stay.
7. Previous intensive care unit (ICU) stay.
8. Surgical prcedures.
9. Bed restriction.
10. Medical prescriptions such as Antacids (Ranitidine or Omeprazole), Sedatives, Steroids (or other immune-suppressing drugs) and Antimicrobials. There is a strong association between use of Antacids and increased risk for NVHAP this may be due to alkalinization of the stomach which provides an ideal environment for bacterial overgrowth-and, subsequently, to contamination of the lower airways. Among nonventilated patients, contaminated gastric content could reach lower airways through reflux **(Sopena et al., 2005).**

Risk factors for resistance include antimicrobial therapy in the past 90 days and late-onset during hospitalization (>5 days), Mild to moderate presentation: no hypotension, intubation, sepsis syndrome, rapid progression of infiltrates or end-organ dysfunction, Severe presentation: hypotension, intubation, sepsis syndrome, rapid progression of infiltrates or end-organ dysfunction. MRSA Methicillin-resistant Staph aureus (Figure 1) **(Coleman et al., 2008).**

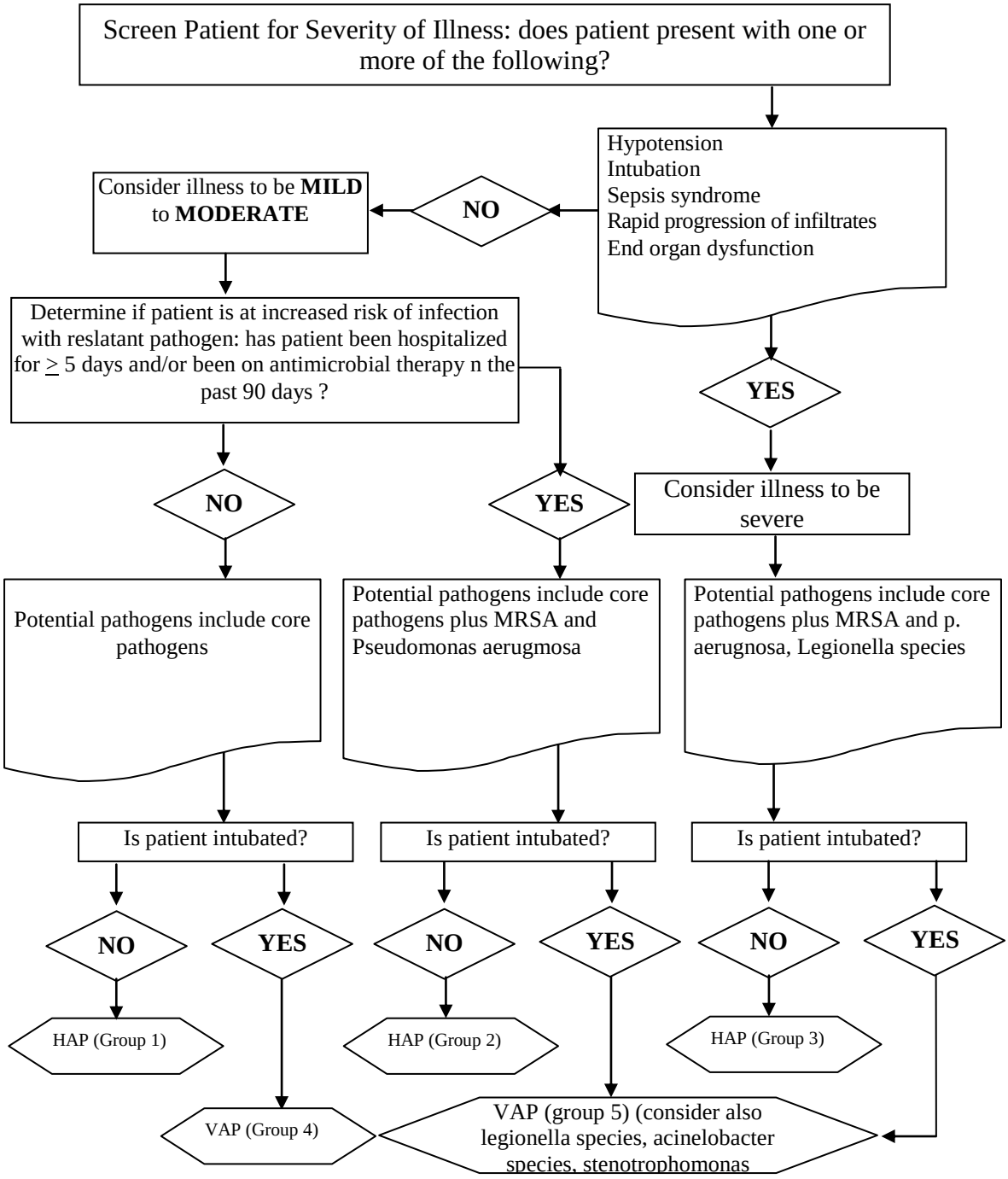

Fig. (1): Algorithm for determining the microbiological cause of hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) using risk factors for resistance and severity of illness **(Coleman et al., 2008).**

Core pathogens are:

- Enteric gram-negative bacilli (Non-Pseudomonal, Enterobocter species, Escherichia coli, Klebsiella species and Proteus species).
- Serrotio morcescens.
- Hemophilus influenzae.
- Methicillin-sensitive Staphylococcus oreus.
- Streptococcus pneumonae.

(**American Thoracic Society, 1995)**

Pathophysiology for ICU-acquired pneumonia:

Pathophysiology of ICU- acquired pneumonia involves 2 main processes: colonization of the respiratory and digestive tracts and microaspiration of secretions of the upper and lower parts of the airway **(Livingston et al., 2000**).

Colonization of bacteria refers to the presence of bacteria without an active host response. Bacterial colonization of the lungs can be due to spread of organisms from many different sources, including the oropharynx, sinus cavities, nares, dental plaque, gastrointestinal tract, patient-to-patient contact, and the ventilator circuit. Inhalation of colonized bacteria from any of these sources can cause an active host response and, ultimately, VAP **(Kunis et al., 2003).**

The presence of an endotracheal tube provides a direct route for colonized bacteria to enter the lower respiratory tract. Upper airway and oral secretions can pool above the cuff of an endotracheal tube and line the tube, forming a biofilm. Starting as early as 12 hours after intubation, the biofilm contains large amounts of bacteria that can be disseminated into the lungs by ventilator-induced breaths, In addition, the biofilm may become dislodged by instillation of saline into the endotracheal tube, suctioning, coughing, or repositioning of the endotracheal tube **(Morehead et al., 2002).**

Endotracheal tubes cause an abnormal interruption between the upper airway and the trachea, bypassing the structures in the upper airway and providing bacteria a direct route into the lower airway **(Kunis et al., 2003).**

Because the upper airway is bypassed, there is decrease in the body's ability to filter and humidify air. In addition, the cough reflex is often eliminated and/or decreased by the presence of an endotracheal tube, and mucociliary clearance can be impaired because of mucosal injury during intubation. An endotracheal tube provides a place for bacteria to bind in the trachea, a situation that further increases production and secretion of mucus. The impairment of these natural host defense mechanisms increases the likelihood of bacterial colonization and subsequent aspiration of the colonized organisms **(De Rosa et al., 2003).**