Lecture – 04

Bacteria Causing Healthcare Associated Pneumonia

Microbiology Team - 430

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Health Care Associated Pneumonia

Definition of pneumonia: infection of the pulmonary parenchyma

Pneumonia: A. Community acquired pneumonia → caused by *Streptococcus pneumonia* < usually susceptible to antibiotic

B. Health care associated pneumonia → *caused by Pseudomonas aeruginosa* < usually resistant to antibiotics.

❖ **Nosocomial pneumonia**: is defined as hospital associated pneumonia (HAP) or health care associated pneumonia (HCAP).

- Occurring at least 48 hours after admission and not incubating at the time of hospitalization.

- Nosocomial pneumonia is the 2nd most common hospital-acquired infections after UTI (urinary tract infection).

- The incidence of Nosocomial pneumonia is highest in ICU (intensive care unit).

- The Nosocomial pneumonia is the leading cause of death from hospital-acquired infections.

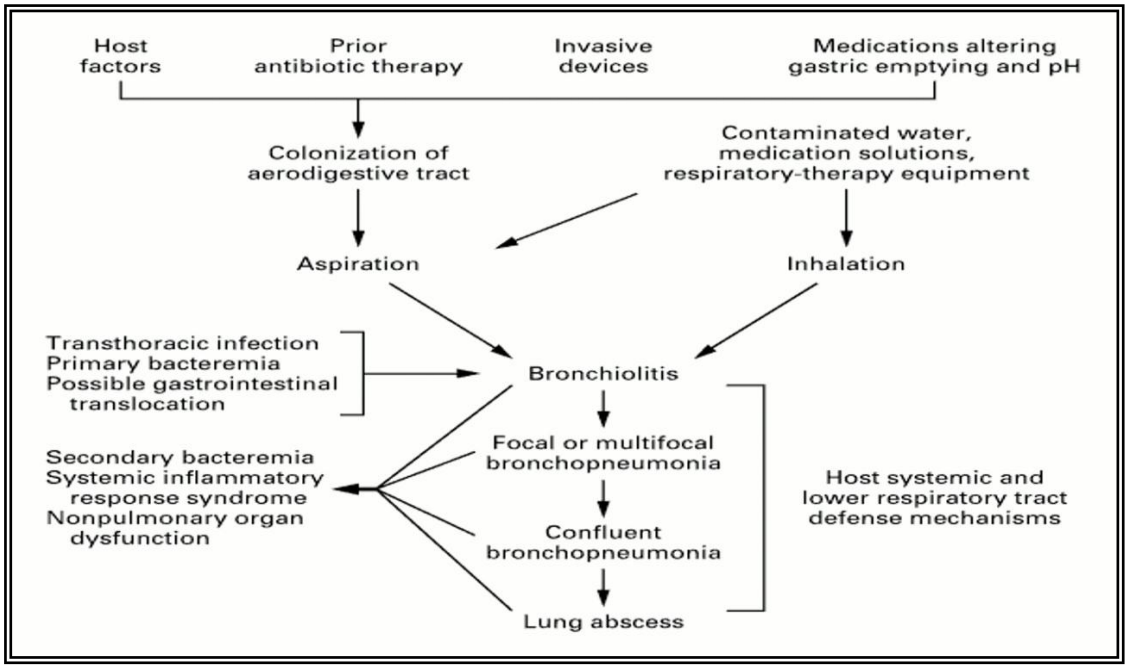
- The incidence of Nosocomial pneumonia in ventilated patients was **10-fold higher** than non-ventilated patients.

Pathogenesis:

- For pneumonia to occur, at least one of the following three conditions must occur:

1. Significant impairment of host defenses
2. Introduction of a sufficient-size inoculum to the overwhelm host's lower respiratory tract defenses.
3. The introduction of highly virulent organisms into the lower respiratory tract.

- Most common is microaspiration of oropharyngeal secretions colonized with pathogenic bacteria.



If the following factors (host factors, prior antibiotic therapy, invasive devices, contaminated water that we use in the ICU.) combined with colonization of aerodigestive tract and these colonization gets aspirated into the lower respiratory tract, it going to lead to bronchiolitis and it going to end with lung abscess and then pneumonia.

Classification:

1. Early-onset Nosocomial pneumonia:

Occurs during the first 4 days of admission usually is due to S.pneumoniae, MSSA (Methicilin sensitive S.aureus), H.influenza, or anaerobes.

2. Late-onset Nosocomial pneumonia:

Occurs more than 4 days of admission more commonly by Gram negative organisms, especially: **P.aeruginosa**, Acinetobacter, Enterobacteriaceae (organisms that live in the gut) (Klebsiella, Enterobacter, Serratia) or MRSA.

Causative Agents:

1. Enteric Gram negative bacilli are isolated most frequently particularly in patients with late-onset disease and in patients with serious underlying disease often already on broad-spectrum antibiotics
   (An antibiotic that is effective against both gram-negative and gram-positive bacteria species.)

2. Prior use of broad-spectrum antibiotics and an immunocompromised state make resistant Gram-negative organism more likely. → (Pseudomonas aeruginosa and Acinetobacter).

3. **P. aeruginosa** and **Acinetobacter**: are common causes of late-onset pneumonia, particularly in the ventilated patients.

4. ***S. aureus*:**
   - Ventilated patients after head trauma, neurosurgery, and wound infection**.**
   - Patients who has received prior antibiotics or in prolonged car in ICU.

5. **MRSA**:
   - Patients had received prior antibiotics thereby.
   - Undergo mechanical ventilation more than 5 days.
   - Received corticosteroids.
   - Presented with chronic lung disease.

6. **Anaerobes** :
   - Patients predisposed to aspiration.

7. **Ventilator associated pneumonia with anaerobes occurred more often with oropharyngeal intubation than nasopharyngeal intubation.**

Ventilator-Associated Pneumonia (VAP):

Is a Nosocomial pneumonia that has developed in patients who are receiving mechanical ventilation.

Classification:

- **Early-onset**: within 48-72 hours after tracheal intubation which complicates the intubation process.
- **Late-onset**: after 72 hours.

Pathogenesis:

- Require 2 important process:
  1. Bacterial colonization of the aerodigestive tract.
  2. Aspiration of contaminated secretion into the lower airway.
- Prevents mechanical clearance by cough and the mucociliary escalator.

Prevention of ***Ventilator Associated Pneumonia***:

The oral regimen (topical Gentamicin (there will be no absorption), Colistin, Vancomycin cream q6h (every 6 hours) for 3 weeks) treating oropharyngeal colonization could prevent VAP. (We use Vancomycin before lab results are available)

Non-pharmacological methods:

1. Continuous subglottic suctioning.
2. Avoidance of large gastric volume.
3. Effective hand washing and use of protective gowns and gloves.
4. Semi-recumbent positioning.
5. Oral (non-nasal) intubation.
6. Humidification with heat and moisture exchanger.
7. Posture change.

Pharmacological methods:

1. Stress-ulcer prophylaxis.
2. Combination antibiotics thereby
3. Prophylaxis antibiotics thereby.
4. Prophylactic treatment of neutropenic patients.
5. Chlorhexidine oral rinse.
6. Vaccines.

Treatment:

- Most initial therapy is empiric because no pathogen is identified or results are not available when antimicrobial decisions are made in most patients. Which means you should treat the patient with a broad-spectrum antibiotic regimen aimed at covering all likely bacterial pathogen. (Before the result of the culture are available).
- This regimen should subsequently be **narrowed**, according to the result of culture.
- The pathogen may be influenced by: - coexisting illnesses.
  - Prior treatment.
  - Length of hospitalization.
- The frequency of ICU –acquired P.aeruginosa carriage or colonization / infection was 23.4% at 7 days and 57.8% at 14 days.
- The mortality can be reduced with early appropriate empiric thereby.

(Form 30% with appropriate thereby to more than 90% with inappropriate thereby).

- Guidelines by American Thoracic Society has separated patients into three groups, each with a set of probable pathogens:

Group 1: mild to moderate HAP with no risk factor

Group 2: mild to moderate HAP with risk factors.

Group3a: sever HAP, early-onset with no risk factor.

Group3b: sever HAP, late-onset or early-onset with risk factor.

- For mild-to-moderate HAP, monotherapy (treatment of a condition by means of a single drug) has been shown to be effective.
- For sever HAP in which infections with resistant organisms is likely, combination therapy probably should be instituted until culture result are available.
- Patients for S. aureus infection, agents against this organism are necessary, including Vancomycin if MRSA is suspected.

Linezolid is comparable with Vancomycin. (Vancomycin causing nephrotoxicity, so it is contraindication with renal failure patients, we should use in this case Linezolid) The advantage of Linezolid is less possible nephrotoxicity.

Combination of antipseudomonal drugs is controversial:

1. Traditional:

Antipseudomonal beta-lactam with an Aminoglycoside .it is Synergy but potential nephrotoxicity. (This means it has an advantage and a disadvantage).

2. Another approach:

Antipseudomonal beta-lactam with a Fluoroquinolone. No benefit of synergy but reduces concern of nephrotoxicity, and quinolone gets into the lungs at higher concentrations.

Response to Therapy:

If no clinical response is noted or deterioration occurs, we need to consider:

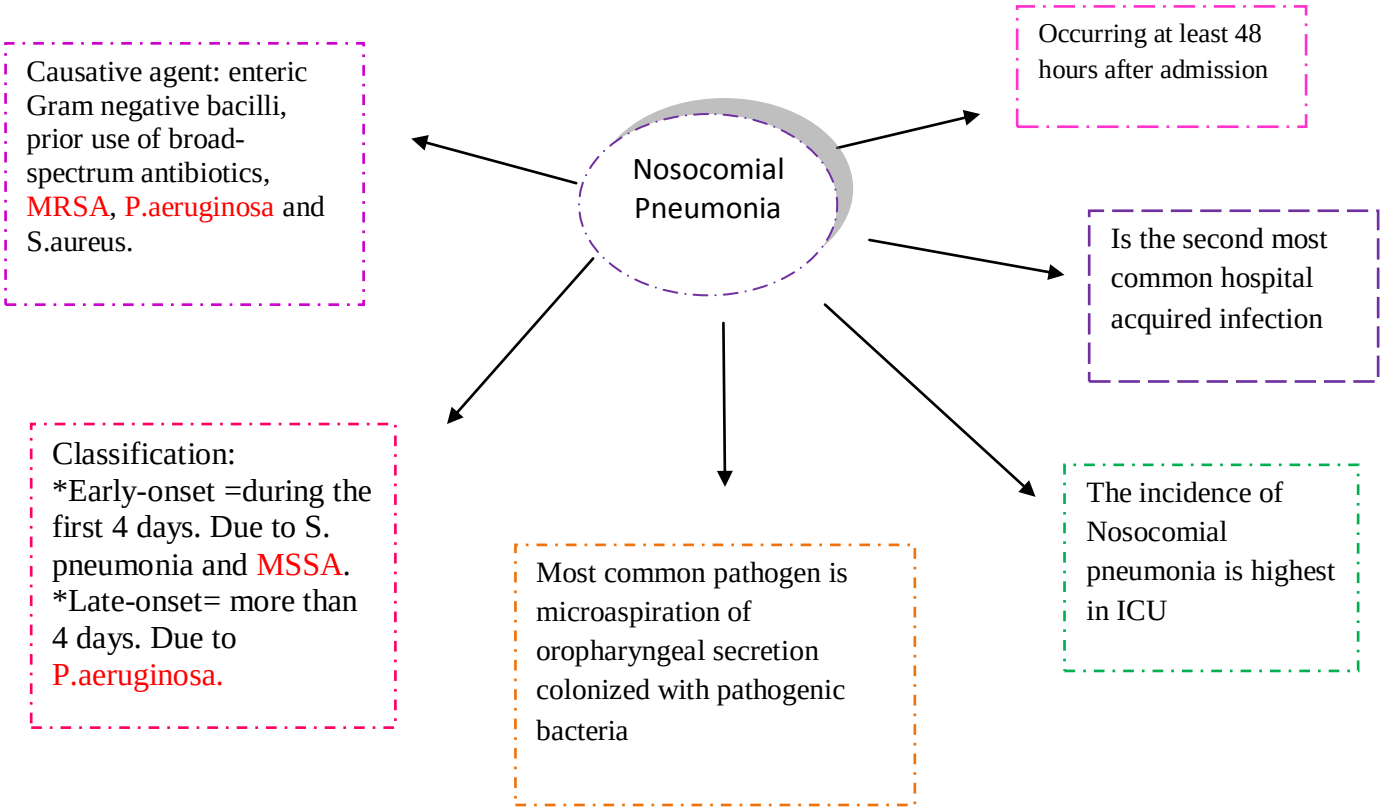
1. Infectious causes:

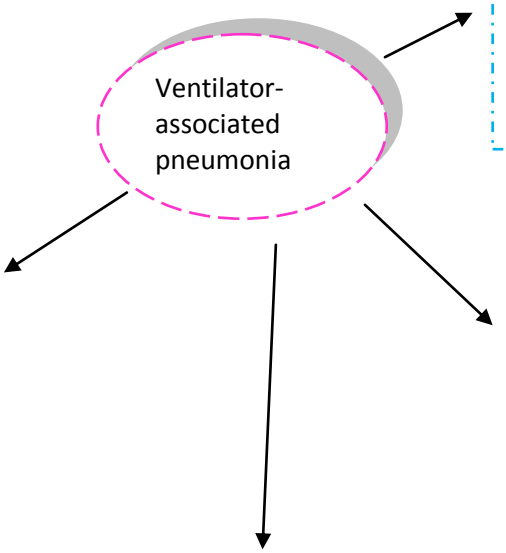
- Extrapulmonary infection
- Lung abscess
- Resistant pathogen
- Superinfection
- Unusual pathogens

2. Noninfectious events:

- Heart: congestive heart failure (CHF).
- Lung: fibroproliferative acute respiratory distress syndrome (ARDS).
- Pulmonary emboli.
- Atelectesis.

Summary


Nosocomial Pneumonia
Causative agent: enteric Gram negative bacilli, prior use of broad-spectrum antibiotics, MRSA, P.aeruginosa and S.aureus.
Occurring at least 48 hours after admission
Is the second most common hospital acquired infection
Classification:
*Early-onset =during the first 4 days. Due to S. pneumonia and MSSA.
*Late-onset= more than 4 days. Due to P.aeruginosa.
Most common pathogen is microaspiration of oropharyngeal secretion colonized with pathogenic bacteria
The incidence of Nosocomial pneumonia is highest in ICU



Classification:
*Early-onset= within 48-72 hours after tracheal intubation
*Late-onset= after72 hours.

Pathogenesis:

*Requires 2 important processes:
1- Bacterial colonization of aerodigestive tract.
2- Aspiration of contaminated secretion into the Lower airway.
*Prevents mechanical clearance by cough and the mucociliary.

Prevention:
The oral regimen (e.g. Vancomycin) treating oropharyngeal colonization could prevent VAP

Treatment:

*Most initial therapy is empiric. With broad-spectrum antibiotics regimen aimed at covering all likely bacterial pathogen.

*This regimen should subsequently be narrowed, according to result of culture.

* Patients for S.aureus infection, agent against this organism are necessary, including Vancomycin **if MRSA is suspected.**

***Linezolid, if the patient has kidney failure.

*combination of antipseudomonal drugs is controversial:

1- Traditional: **antipseudomonal beta-lactam with an Aminoglycoside (synergy but potential nephrotoxicity).**

2- Another approach: **Antipseudomonal beta-lactam with Fluoroquinolone (no benefit of synergy but reduce concern of nephrotoxicity and Quinolone gets into the lung at higher concentration**.