Polmoniti Nosocomiali: HAP VAP

Dott. Davide F. Precone

Link
profitable solutions

Community Acquired (CAP)	Ventilator-Associated (VAP)	Hospital-Acquired (HAP)
		
Pneumonia that develops outside the hospital	Pneumonia that develops 48-72 hours after endotracheal intubation	Pneumonia that develops 48 hours after admission

Epidemiology of HAP and VAP

The epidemiology influenced by various factors, including differences in definitions, diagnostic limitations, and microbiological sampling methods across countries

Variability in Rates: The incidence of HAP varies, with estimated rates ranging from 5 to more than 20 cases per 1,000 admissions and from 2.5 to more than 6.1 cases per 1,000 non-ICU patients.

Hospital-acquired pneumonia (HAP) is a common nosocomial bacterial infection that is most prevalent in medical and surgical intensive care units (ICUs).

HAP remains *the second most common* inhospital infection after urinary tract infections, with considerable effects on both medical and economical aspects.

Pathogenesis

The most common pathogenetic route of HAP/VAP is micro-aspiration of pathogens colonizing the oropharyngeal and gastroenteric tract.

Risk factors for HAP and VAP

Risk Factors for Developing HAP/VAP
Invasive mechanical ventilation
Extreme age
Altered conscious level
Chronic lung disease, chronic kidney disease
Severe trauma
Malnutrition
Previous exposure to wide-spectrum antibiotics
Aspiration
Prolonged surgical procedures (thoracic/upper abdominal)
Use of glucocorticoids/opioids/neuromuscular blocking agents
Stress ulcer prophylaxis
Acute respiratory distress syndrome (ARDS)
Anemia

The risk of developing HAP is ten times higher in patients requiring mechanical ventilation.

Challenges in Diagnosis

Inaccuracy of the currently available diagnostic criteria. There is no generally accepted or documented **gold standard** diagnostic criterion for HAP/VAP

Using only **clinical diagnosis** of VAP may overlook approximately one-third of cases in the ICU

Recommended approach involves a combination of clinical scores, microbiological tools, and the kinetics of serum biomarkers to properly identify and evaluate patients with lower respiratory tract nosocomial infections.

Challenges in Diagnosis

A clinical score, known as the Clinical Pulmonary Infection Score (CPIS), has been developed to combine both clinical and paraclinical parameters. However, the sensitivity and specificity of this score remain a matter of controversy.

Parameter/Value	0 Points	1 Point	2 Points
Tracheal secretions *	Few	Moderate	Large
Chest radiography infiltrates	No new infiltrates	Diffuse new infiltrates	Localized new infiltrates
Temperature (°C)	36.5–38.4	38.5–38.9	>38.9 or <36
Hypoxemic index (P_aO_2/F_iO_2 mmHg)	>240 or ARDS		<240 and NO ARDS
White blood cell count ($\times 10^3/\mu L$)	4–11	<4 or >11	
Microbiological culture	Negative		Positive

* if tracheal secretions are purulent, clinicians should add one more point to the final score.

The kinetics of serum biomarkers

It can contribute to the proper identification and evaluation of patients with lower respiratory tract nosocomial infections, providing valuable prognostic and diagnostic information to guide clinical decision-making.

Procalcitonin

PCT kinetics can serve as a prognostic marker for VAP, and its measurement, in combination with clinical assessment, can be beneficial in specific clinical circumstances to reduce antibiotic treatment duration

C-reactive protein

Serial CRP measurements have been shown to be useful in predicting outcomes, with higher CRP levels associated with poor outcomes

Microbiological samples

Last guidelines, recommend qualitative or quantitative cultures from microbiological samples obtained from the lower respiratory tract as a cornerstone in identifying and diagnosing VAP

Invasive techniques	Bronchoalveolar lavage (BAL)
	Mini-bronchoalveolar lavage (mini-BAL)
	Protected specimen brush (PSB)
Non-invasive technique	Endotracheal aspirate

Microbiology and Etiology

The most common pathogens associated with HAP and VAP include ***Staphylococcus aureus*** (including methicillin-resistant ***MRSA*** and methicillin-susceptible MSSA), ***Pseudomonas aeruginosa***, ***Klebsiella pneumoniae***, Escherichia coli, Enterobacter species, Acinetobacter species, and Stenotrophomonas maltophilia.

It is important to note that the specific pathogens involved may vary based on geographic location, local ecology, and the prevalence of MDR organisms in different healthcare settings.

Prevention

Preventing Hospital-Acquired Pneumonia (HAP) and Ventilator-Associated Pneumonia (VAP) is crucial

Preventing pulmonary aspiration of oropharyngeal secretions by:

Raising the head of the bed to 30°–45°
Daily sedation interruption
Maintaining a constant airway cuff pressure of the endotracheal tube to a maximum of 30 cm of H2O
Applying optimal positive end-expiratory pressure (PEEP)
Subglottic secretions drainage with specially designed devices that permit continuous or intermittent aspiration.

Measurement of gastric residual volume, although its impact on aspiration risk is not well-documented.

Regular oropharyngeal care with chlorhexidine, which has been statistically correlated with a reduction in HAP incidence in selected cases.

Selective decontamination of the digestive tract (SDD) with specific antibiotics such as colistin, tobramycin, or nystatin.

Vaccination against specific pathogens such as H. influenzae and S. pneumoniae.

Treatment

antibiotherapy, with two main steps which should be followed:

Empirical treatment
which respects both the severity of the disease and the presence of risk factors for MDR pathogens

Target treatment
according to microbiological susceptibility reports or antibiograms.

Empiric Treatment

no MDR risk factors and low mortality risk

Monotherapy with narrow-spectrum drugs against methicillin-sensitive S. aureus (MSSA) and non-resistant Gram-negative bacilli is recommended.

ceftriaxone
cefotaxime
levofloxacin
moxifloxacin

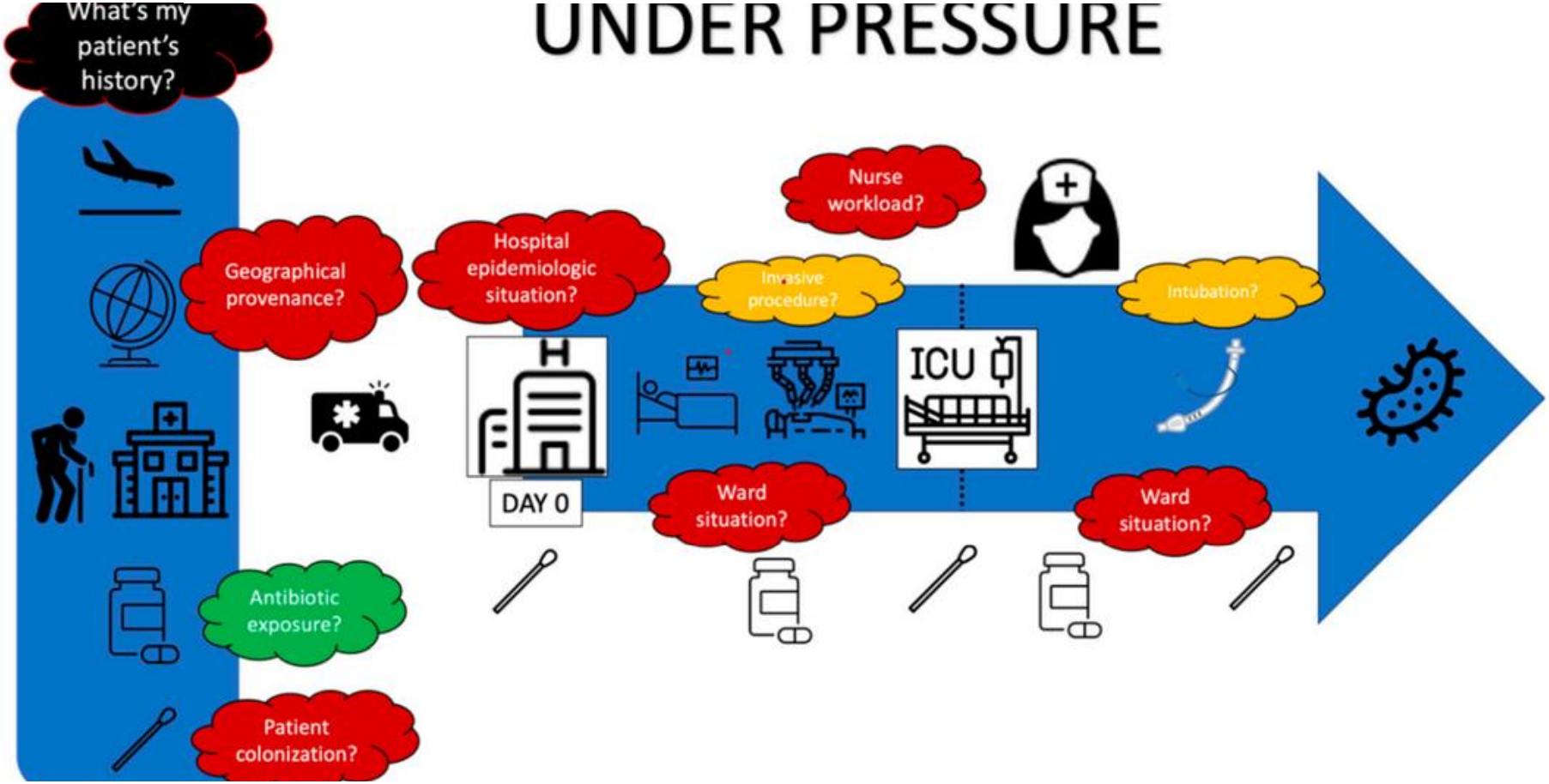
antibiotics

MDPI

Review

Empiric Treatment in HAP/VAP: "Don't You Want to Take a Leap of Faith?"

Khalil Chaïbi [1,2], Gauthier Péan de Ponfilly [3,4], Laurent Dortet [5,6], Jean-Ralph Zahar [7] and Benoît Pilmis [4,8,*]



Risk factors for MDR Pathogens

Risk Factors for MDR Pathogens
ARDS before VAP
Intravenous wide-spectrum antibiotic use in the last 3 months
Septic shock at the moment of suspecting VAP
At least 5 days length of hospitalization before suspecting VAP (late-onset subtype)
Renal replacement therapy before VAP

Empiric Treatment

high risk for MDR infections and a >15% mortality risk

Absence of septic shock

single agent against Gram-negative bacteria (especially P. aeruginosa)

imipenem, meropenem
ceftazidime, cefepime
levofloxacin
piperacillin–tazobactam

Empiric Treatment

high risk for MDR infections

Septic shock

should receive a combination
between two agents
and one agent against MRSA

imipenem, meropenem
ceftazidime, cefepime
levofloxacin
piperacillin–tazobactam
+
amikacin, gentamicin
ciprofloxacin, levofloxacin
+
vancomycin, teicoplanin
linezolid

Staph aerus

MSSA (mecA neg)

Antistaphylococcal Penicillins

- Oxacillin
  - 12g LD+CI

Cephalosporins

- Cefazolin
  - 6g LD+CI

MRSA (mecA pos)

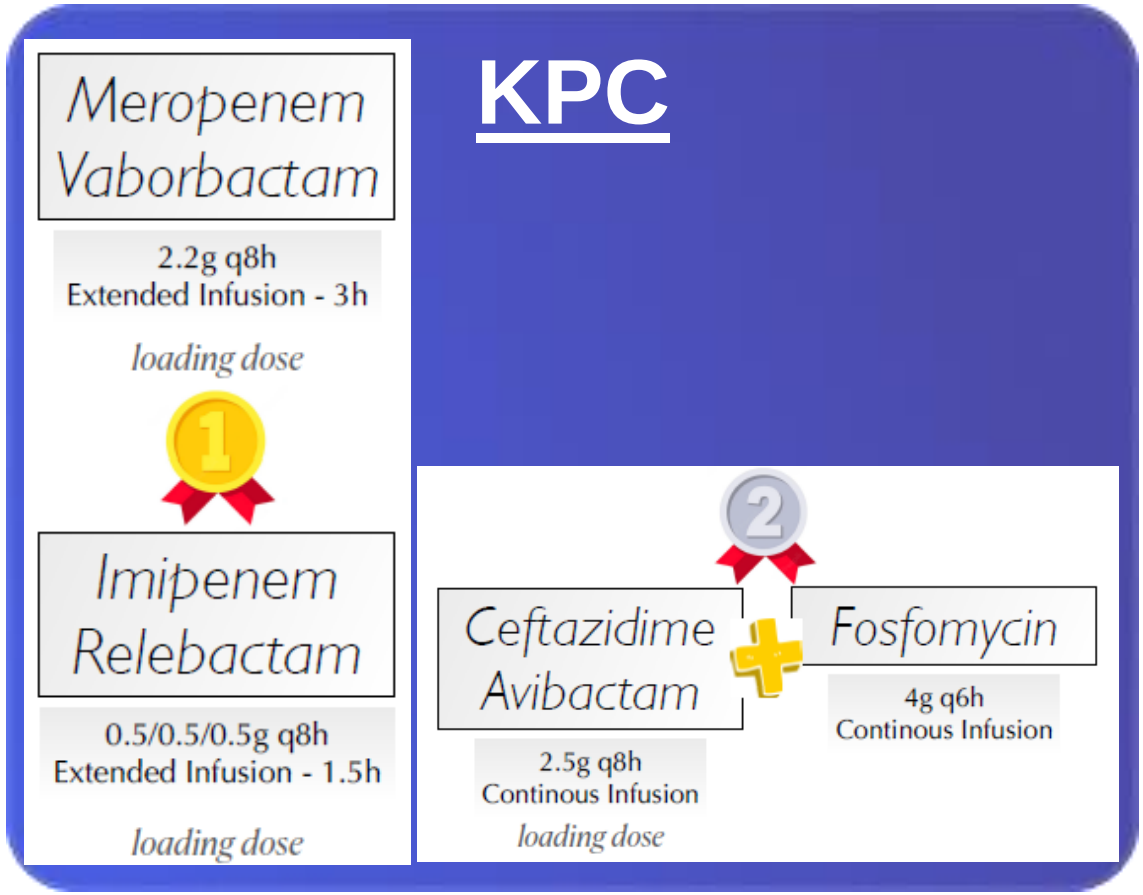
Linezolid

or

Linezolid
+
Fosfomycin
600 mg q12h
1200 mg CI
POSSIBLY
EVALUATE LD

Microorganisms **2023**; 11:394

klebsiella pneumoniae carbapenemase-producing



Exp Rev Anti Infect Ther Sep **2022**

Pseudomonas aeruginosa

Microorganisms **2023**; 11:394