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Nosocomial Pneumonia in intensive care units

Szpitalne zapalenie płuc w oddziałach intensywnej terapii

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STRESZCZENIE

Szpitalne zapalenie płuc jest najczęstszym rodzajem infekcji stwierdzanym w oddziałach intensywnej terapii (OIT), a wysokie odsetki śmiertelności i umieralności z jego powodu nakazują uznać to schorzenie za najbardziej poważne w środowisku szpitalnym. Z tego względu właściwe rozpoznanie i zrozumienie wpływu szpitalnego zapalenia płuc na stan chorych hospitalizowanych w OIT odrywa ważną rolę. Ryzyko zachorowania uzależnione jest od stanu ogólnego pacjenta, rodzaju biologicznego czynnika chorobotwórczego, inwazyjnych i związanych z immunosupresją procedur diagnostyczno-leczniczych. W związku z powyższym zaleca się wdrożenie wielokierunkowych działań, obejmujących systematyczne kształcenie personelu, eliminację czynników ryzyka oraz wprowadzanie rekomendacji opracowanych przez odpowiednie instytucje naukowe. W niniejszym artykule przedstawiono problematykę zapadalności, patogenezy, czynników ryzyka oraz metod zapobiegania szpitalnemu zapaleniu płuc w oddziałach intensywnej terapii.

Słowa kluczowe: szpitalne zapalenie płuc, oddział intensywnej terapii, strategie zapobiegania.

ABSTRACT

Nosocomial pneumonia (NP) has been considered the most common infection in Intensive care units (ICUs) and due to its high morbidity and mortality rates it has been deemed the most serious hospital acquired infection. It is vital to recognize and understand the impact of nosocomial infections on ICU patients. The risk of NP is associated with patient's condition, infectious agents, invasive and immunosuppressive procedures. A multiprong approach should include staff education, minimizing risk factors, and implementing guidelines established by national committees. The aim of this article is to discuss incidence, pathogenesis, risk factors and prevention of nosocomial pneumonia in intensive care units.

Key words: nosocomial pneumonia, intensive care unit, prevention strategies

Introduction

Intensive care units (ICUs) have contributed greatly to the survival of patients with trauma, shock states, and other life-threatening conditions, but ICU admission is associated with increased risk of nosocomial (hospital-acquired) infection. Rates of nosocomial infection in patients requiring more than one week of advanced life support within an ICU are three to five times higher than in hospitalized patients who do not require ICU care. Infection is the most common direct or indirect cause of death of patients who survive the early period after major trauma or full-thickness burns and is the most commonly identified cause of multiple-organ dysfunction syndrome [1, 2, 3].

Managing infections in the intensive care unit can be a daunting challenge to any practitioner. In the United States, more than 5 million patients are admitted to ICUs every year [4]. Estimates from the National Nosocomial Infections Surveillance (NNIS), now the National Healthcare Safety Network (NHSN), found that approximately 1.7 million nosocomial infections occurred in the United States hospitals in 2002 with 24% of these infections in the ICU, a rate of 13 per 1000 patient days [5].

The past several decades have shown an increased effort in characterizing the epidemiology of healthcare–associated infections and in advancing the knowledge of infection prevention practices. The aim of this article is to discuss incidence, pathogenesis, risk factors and prevention of nosocomial pneumonia.

Impact and Incidence of Nosocomial Pneumonia

Nosocomial pneumonia (NP) is the second most common nosocomial infection after urinary tract infection and the most common nosocomial infection in ICUs. NP is the leading cause of mortality due to hospital-acquired infections [6, 7].

A nosocomial infection (derived from the Greek words nosos [disease] and komein [to care for], and later the Latin word for hospital nosocomium) is defined as an infection that is not present or incubating when the patient is admitted to hospital or other health-care facility [8].

Defined as pneumonia occurring 48 hours or more after hospital admission, NP also includes the subset of ventilator-associated pneumonia (VAP), often defined as pneumonia developing more than 48 to 72 hours after initiation of mechanical ventilation. Health care-associated pneumonia (HCAP), part of the continuum of NP, describes an increasingly common proportion of pneumonia developing outside the hospital [9, 10].

The incidence of NP depends on several factors. Patients with endotracheal tubes have rates that are up to 20-fold higher [11]. A chance of infection increases with duration of mechanical ventilation, and thus rates presented per 1000 ventilator days more accurately reflect the risk of VAP. Overall VAP rates of 7 to 14 per 1000 ventilator days have recently been reported [12]. Arabi et al [13] found that the rates of VAP varied from 10 to 41.7 per 1000 ventilator-days and were generally higher than the National Healthcare Safety Network (NHSN) benchmark rates for 2006 in their systematic review. VAP was associated with a crude mortality that ranged from 16% to 94% and with increased ICU length of stay (LOS). However, the data coming from National Healthcare Safety Network program showed that median rates of VAP were ranging from 0.7 cases per 1000 ventilator-days in pediatric units to 7.4 cases per 1000 ventilator-days in burn units (Table 1) [14].

Pathogenesis

For the development of pneumonia, virulent microorganisms should invade the lung parenchyma. This can happen either subsequent to a defect in defense mechanisms of the host or by an overwhelming inoculum. The normal human respiratory tract possesses a variety of defense mechanisms such as anatomic airway barriers, cough reflexes, mucus, mucociliary clearance, cell-mediated and humoral immunity and a dual phagocytic system that involves both alveolar macrophages and neutrophils [15, 16].

In the mechanically ventilated patient, numerous factors conspire to compromise host defenses: Critical illness, comorbidities, and malnutrition impair the immune system [17]. Endotracheal intubation thwarts the cough reflex, compromises mucociliary clearance;

Table 1. Ventilator-associated pneumonia rates* in selected wards
Tabela 1. Ryzyko wystąpienia zapalenia płuc związane ze sztuczną wentylacją

				Percentile				
Type of ICU	**No. of Units**	**Ventilator-days**	**Pooled Mean**	**10%**	**25%**	**50% (Median)**	**75%**	**90%**
Coronary	129	174,480	2.1	0.0	0.0	1.2	2.8	5.8
Cardiothoracic	109	214,373	3.9	0.0	0.9	2.6	5.4	9.7
Medical all others	80	181.102	2.2	0.0	0.0	1.3	3.5	6.1
Medical/surgical major teaching	115	383.068	2.9	0.0	0.9	2.0	3.1	5.6
Neurologic	15	25.528	6.7	-	-	-	-	-
Neurosurgical	42	76.763	5.3	0.0	2.6	4.0	5.6	8.2
Pediatric medical/ surgical	79	172.208	1.8	0.0	0.0	0.7	2.7	4.6
Surgical	127	311.739	4.9	0.0	1.8	3.8	6.5	9.9
Trauma	41	145.294	8.1	0.0	2.1	5.2	10.0	16.1
Burn	25	34.088	10.7	0.0	2.4	7.4	13.1	15.1
Respiratory	5	8748	0.5	-	-	-	-	-

*Number of ventilator–associated pneumonias × 1000
Number of ventilator–days

ICU, Intensive care unit
From National Healthcare Safety Network (NHSN) report: Data summary for 2006 through 2008, issued December 2009

injures the tracheal epithelial surface, and provides a direct conduit for bacteria from the mouth, hypopharynx, and stomach to gain direct access to the lower respiratory tract. Moreover, the cuff of the endotracheal tube allows pooling of oropharyngeal secretions in the subglottic region, forming an ideal medium for microbial growth, which periodically leaks around the cuff into the trachea. It would probably be more accurate pathogenically to rename VAP as "endotracheal intubation–related pneumonia." This combination of impaired host defenses and continuous exposure of the lower respiratory tract to large numbers of potential pathogens through the endotracheal tube puts the mechanically ventilated patient at great jeopardy of developing VAP [18, 19].

In order for microorganisms to cause VAP, they must first gain access to the normally sterile lower respiratory tract, where they can adhere to the mucosa and produce sustained infection. Microorganisms gain access by one of the following four mechanisms:

1. Aspiration of microbe-laden secretions, from the oropharynx directly or indirectly by the reflux from stomach into the oropharynx, then into the lower respiratory tract,
2. Inhalation of contaminated air or medical aerosols,
3. Direct extension of a contiguous infection such as a pleural space infection,
4. Hematogenous carriage of microorganisms to the lung from remote sites of local infection such as an intravascular device-related bloodstream infection (IVD-related BSI) [20, 21, 22, 23, 24].

By this route, aspiration of oropharyngeal contents comprising a large microbial inoculum overwhelms host defenses already compromised by critical illness and the presence of an endotracheal tube, readily leading to the development of nosocomial pneumonia [24].

Risk Factors for Nosocomial Pneumonia

A number of independent risk factors have been shown to increase the likelihood of developing VAP (Table 2). In general, these risk factors can be categorized as:

1. Factors that increase the likelihood or duration of mechanical ventilation,
2. Factors that increase colonization of the oropharynx and gastric mucosa,
3. Factors that increase the likelihood of aspiration,
4. Host factors that increase susceptibility to infection [25, 26, 27].

Prolonged mechanical ventilation or reintubation, or both, are the most powerful predictors of developing VAP. Cunnion et al [28] found that mechanical ventilation in excess of 24 hours was associated with a 12-fold increased risk of developing VAP, and Trouillet [29] found that ventilation longer than 7 days was associated with a six-fold increased risk. Emergent reintubation also carries a high risk of aspiration and was associated with a six-fold increased risk of VAP in a retrospective study [30].

Table 2. Risk factors for ventilator-associated pneumonia
Tabela 2. Czynniki ryzyka zapalenia płuc związanego ze sztuczną wentylacją

Host Factors	Intervention Factors
Serum albumin, <2.2 g/dL	H2 blockers ± antacids
Age, ≥60 yr	Paralytic agents, continuous intravenous sedation
Adult respiratory distress syndrome (ARDS)	Receipt of >4 units of blood
Chronic obstructive pulmonary disease or other chronic pulmonary diseases	Intracranial pressure monitoring
Coma or impaired consciousness	Mechanical ventilation in excess of 48 hr
Burns, trauma	Positive end-expiratory pressure
Organ failure	Frequent ventilator circuit changes
Advanced severity of illness	Reintubation
Large-volume gastric aspiration	Nasogastric tube
Gastric colonization and gastric pH	Supine head position
Upper respiratory tract colonization	Transport out of the intensive care unit
Sinusitis	Prior antibiotic therapy
Modifi ed from Chastre J, Fagon JY: Ventilator-associated pneumonia. Am J Resp Crit Care Med 2002; 165: 867-903.	

Endotracheal tubes allow pooling of hypopharyngeal secretions that can leak around the cuff directly into the trachea, and a supine position appears to increase the risk of aspiration around the cuff. Torres et al. [31] demonstrated the importance of body position in gastric reflux and tracheal aspiration. They instilled a colloid with technetium via nasogastric tube and, by placing patients in a recumbent position, they could demonstrate a significant reduction of the radioactivity in tracheal secretions, in comparison with patients in supine position. Additionally, Drakulovic et al [32] studied the impact of body position on the development of VAP in a randomized study. They placed 39 patients in a semi-recumbent (45°) or supine (0°) body position. Microbiologically confirmed pneumonia occurred in 5% of patients in the semi-recumbent position and in 23% in the supine group (P = 0.018).

Poor dental hygiene increases the bacterial burden in the oropharynx and is an independent risk factor for nosocomial pneumonia [33]. Likewise, a high gastric pH (>5) is associated with greatly increased bacterial colonization of the gastric contents, as well as an increased risk of VAP. Because low gastric pH prevents bacterial growth in the gastric chamber and bacterial migration from the small bowel [19]. The relationship between gastric pH and gastric colonization has been well established in several studies. The use of stress-ulcer prophylaxis agents that alter the gastric pH can increase the gastric colonization and VAP rates. Cook et al. [34] demonstrated a trend toward decreased clinically significant bleeding with H_2-antagonists and antacids in comparison with sucralfate. There was a trend toward an increased risk of pneumonia associated with H_2-antagonists as compared with no prophylaxis, and a significantly higher risk as compared with sucralfate.

Intubation, reintubation and tracheotomy causing local trauma and inflammation increase the risk of aspiration of nosocomnial microbes. It was shown, that endotracheal tubes were coated by bacteria from oropharynx partly or completely in 96 % and 84 % respectively [26].

Prevention

The clinical and financial consequences of NP justify the widespread pursuit of strategies aimed at preventing its development. Preventive strategies are either directed at reducing the overall incidence of infectious complications in hospitalized patients or they are specifically targeted at reducing the incidence of NP [27]. Prevention strategies can be classified as pharmacological or non-pharmacological:

Non-pharmacological Strategies

Education: Education should form part of all infection control programs with a view to educating staff about epidemiology and the procedures that have been shown to reduce the incidence of HCAP (level B) [35, 36].

Epidemiological surveillance: In the event of a HCAP outbreak, particularly in an ICU, causative pathogens and resistance patterns should be identified in clinically representative samples so that appropriate prevention strategies can be developed (level A) [37].

Nursing and kinesitherapy personnel: An increase in the number of professional nurses per patient and a higher level of academic qualification are factors associated with a reduction in the incidence of pneumonia and reintubations (level B) [38, 39]. A structure involving multidisciplinary teams favors a reduction in the incidence of HAP [40]. Inclusion of, a kinesiologist ensure better control and management of ventilation (level D).

Prevention iatrogenic spread: Careful handwashing both before and after contact with a patient has been shown to reduce the incidence of nosocomial infection [41, 42, 43]. Clusters of NP cases attributed to poor hand hygiene have been described, and careful handwashing remains an important defense against nosocomial infection spread (level A) [43, 44].

The quality of handwashing is important; hands should be washed with soap and water or a waterless antiseptic before and after touching patients, their secretions, or respiratory equipment, whether or not gloves are used (level B) [45, 46] In addition, the use of alcohol-based handwash in bedside dispensers is likely to improve compliance with hand hygiene recommendations. Because the use of artificial fingernails among health care providers has recently become known as a significant risk factor for colonization and subsequent spread of resistant organisms, guidelines recommend against their use by personnel with direct patient contact, especially in ICU and surgical settings [43]. ***Use of gloves and gown:*** The use of gloves and gown reduces the rate of nosocomial infection. This practice is more effective when directed against certain antibiotic-resistant agents (MRSA and vancomycin-resistant enterococci) [47].

Noninvasive ventilation: Endotracheal intubation is one of the most important risk factors contributing to pneumonia in patients requiring mechanical support for respiratory failure. Increasingly, the use of noninvasive ventilation (NIV) or positive-pressure mask ventilation in selected groups of patients has been effective in preventing endotracheal intubation. Noninvasive ventilation is recommended in selected cases where there are no contraindications (level B) [48, 49, 50].

In a randomized controlled trial of patients admitted with an acute exacerbation of chronic obstructive pulmonary disease (COPD), standard medical care combined with NIV was associated with significant reductions in endotracheal intubation, hospital length of stay (LOS), complications (including NP), and mortality when compared with standard medical care alone [49]. A subsequent case-control study of well-matched patients with acute exacerbations of COPD or cardiogenic

pulmonary edema (CHF) compared clinical outcomes between NIV and mechanical ventilation. The use of NIV was associated with lower rates of all nosocomial infections as well as NP (18% vs. 60%, P < .001, and 8% vs. 22%, P = .04, respectively). Patients treated with NIV also had shorter ICU LOS, lower mortality, and received less antibiotics [50].

Weaning: Shortening the duration of intubation reduces the principal risk factor of HCAP. The implementation of weaning protocols *(routine assessment aimed at identifying patients capable of breathing spontaneously, discontinuation of sedation, and use of other techniques)* [51] reduces the duration of invasive ventilation (level A) [52]. The implementation of weaning protocols has also been shown to significantly reduce the health care costs and institutional rates of VAP [53, 54, 55].

Patient positioning: Supine positioning of the mechanically ventilated patient's head has been shown to increase the risk of gastro-esophageal-pharyngeal aspiration [56]. Elevating the head of the bed at an angle of 30º to 45º is a simple cost-free measure that reduces the incidence of VAP (level B) [32, 56]. Three studies have evaluated the efficacy of semi-recumbent positioning (elevation of the head of the bed 45^0). Two small evaluations used surrogate outcomes of reflux and aspiration events, both of which were reduced with semi-recumbent positioning [56, 57]. A subsequent randomized controlled trial in 86 mechanically ventilated patients was stopped early after semi-recumbent positioning was associated with a significant reduction in VAP [32].

Aspiration of respiratory secretions: Two systems are used to aspirate secretions: *open systems*, in which all of the suctioned material is disposed of after each procedure; and *closed systems*, in which the equipment can be reused many times before emptying. There is no evidence that the closed system reduces the incidence of VAP (level B) [58]. The closed system does not depressurize the airway, maintains oxygenation, and facilitates the clearance of secretions. The apparatus should be changed when it no longer works properly or is visibly soiled. There are no recommendations regarding the use of sterile gloves in preference to clean gloves, nor in favor of continuous aspiration systems rather than conventional systems [59, 60]. Only sterile water should be used to flush secretions out of aspiration catheters if these are going to be reused (level B).

Subglottic secretion drainage: The pooling of contaminated secretions above the cuff of the endotracheal tube may predispose patients to aspiration and subsequently VAP. Removal of these secretions could theoretically reduce the risk of developing pneumonia. Kollef et al [60] found that the time to onset of VAP was delayed significantly (5.9 days versus 2.9 days, P= .006). However, in this latter study, a recent meta-analysis of the five studies that have evaluated this preventive strategy found that subglottic secretion drainage significantly reduces the incidence of VAP and should be considered for use in patients requiring more than 3 days of mechanical ventilation [61]. Recent evidence-based guidelines have recommended the use of endotracheal tubes that allow for suctioning of subglottic secretions [25, 62].

Oral hygiene: Given the potential role of oropharyngeal bacteria colonies in the development of NP, it would seem that improving oral hygiene could prevent many cases of NP. Binkley et al [63], found that although a majority of nurses caring for patients subject to mechanical ventilation recognized the importance of dental hygiene, the methods used to ensure it varied considerably. Improvement in oral hygiene in elderly nursing home residents – by the use of antiseptic mouthwash, brushing teeth after all meals, and weekly plaque removal – has been linked to reduced rates of aspiration pneumonia [64,65]. The use of chlorhexidine 0.12% oral rinse has been associated with reduced rates of VAP in surgical ICU patients [66].

Until more data are available on specific dental hygienic practices, it is recommended that mechanically ventilated patients have their teeth brushed daily, undergo oral cleansing every 2 to 4 hours, undergo routine suctioning to reduce accumulation of fluids in the oropharynx, and have a mouth moisturizer applied to their lips to prevent cracking [67].

Ventilator circuit management strategies: A decrease in the incidence of VAP has been observed when ventilator circuits are changed less frequently or only when mechanical ventilation has been withdrawn unless the quantity of secretions, blood, or water in the tubing is excessive (level A) [68]. The reusable components and circuits of respiratory support systems should be completely and carefully cleaned, sterilized or subject to high-level disinfection before being used for another patient. Condensation water should be eliminated regularly from the tubing to ensure that condensate does not flow towards the patient (level A) [68, 69].

Active humidifiers versus heat and moisture exchangers: Studies have demonstrated that the use of passive humidifiers (heat and moisture exchangers) as opposed to active humidifiers is associated with a significant reduction in the incidence of VAP. The use of heat and moisture exchangers (HMEs) has been recommended by authors of a systematic review and is currently recommended by the Canadian Critical Care Trials Group [62, 70]. Active humidifiers increase the resistive dead space load making the administration of aerosolized drugs more difficult. Since these humidifiers can also increase the risk of airway obstruction, patients must be monitored more often when these are being used. HMEs should only be changed when they are no longer functioning properly or are visibly soiled (level B) [71, 72].

Table 3. Recommendations for the prevention of ventilator-associated pneumonia

Tabela 3. Zalecane postępowanie w profilaktyce zapalenia płuc związanego ze sztuczną wentylacją

Preventive Measures	**HICPAC Grade***
a) General Measures	
■ Educate all health care workers involved with the care of mechanically ventilated patients on the risks and methods of preventing ventilator-associated pneumonia	IA
■ Perform adequate hand hygiene between contacts with patients	IA
■ Use gloves for handling respiratory secretions or objects contaminated with respiratory secretions	IB
■ Conduct surveillance for bacterial pneumonia in ICU patients using NNIS definitions. Include data on causative organisms and their antimicrobial susceptibility patterns. Express data as rates to assist intrahospital comparisons	IB
■ Do not routinely perform cultures of patients, equipment, or environment in the absence of an outbreak	II
■ Thoroughly clean all devices to be sterilized and disinfected	IA
■ Use steam sterilization or wet heat pasteurization for reprocessing of heat-stable semicritical devices and low-temperature sterilization for heat or moisture-sensitive devices	IA
■ Use sterile water for rinsing reusable semicritical devices	IB
■ Change ventilator circuit only when they become soiled	IA
■ Periodically drain and discard condensate from ventilator circuits	IB
■ Clean, disinfect, rinse with sterile water, and dry in-line nebulizers between treatments on the same patient	IB
■ When possible, use aerosolized medications in single-use vials	IB
b) Nonpharmacologic Measures to Reduce Pneumonia	
■ Oral (non-nasal) intubation	IB
■ Remove nasogastric and endotracheal tubes as soon as clinically feasible	IB
■ Avoid unnecessary reintubation	II
■ When feasible, use noninvasive ventilation to avoid the need for intubation or reintubation	II
■ Early tracheostomy	_
■ Semirecumbent positioning of the patient	II
■ Implement a comprehensive oral-hygiene program for mechanically ventilated patients	II
■ If feasible, use an endotracheal catheter that allows for continuous or frequent subglottic suctioning	II
■ Humidification with heat and moisture exchanger (HME)	NR
■ Closed multiuse catheters for airway secretion suctioning	NR
■ Kinetic bed therapy	NR
c) Pharmacologic Measure to Reduce Pneumonia	
■ Immunize all patients at risk for pneumococcal infection and influenza	IA
■ Routine use of chlorhexidine oral rinse	NR
■ Preferential use of sucralfate for stress bleeding prophylaxis	NR
■ Selective digestive decontamination	NR
■ Systemic antimicrobials to prevent development of pneumonia	NR
*Taken from **CDC/HICPAC** system of weighting recommendations based on scientific evidence. **IA,** strongly recommended for implementation and supported by well-designed experimental, clinical, or epidemiological studies. **IB,** strongly recommended for implementation and supported by some experimental, clinical, or epidemiological studies and a strong theoretical rationale. **IC,** required by state or federal regulations, rules or standards. **II,** suggested for implementation and supported by suggestive clinical or epidemiological trials or a theoretical rationale. **NR,** no recommendation for or against at this time.	
HICPAC, Healthcare Infection Control Practices Advisory Committee. Modified from; Tablan OC, Anderson LJ, Besser R, et al: Guidelines for preventing health-care-associated pneumonia, 2003: Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee. MMWR Recomm Rep 2004; 53(RR-3): 1-36 Dodek P, Keenan S, Cook D, et al: Evidence-based clinical practice guideline for the prevention of ventilator-associated pneumonia. Ann Intern Med 2004; 14: 305-313.	

Pharmacological Strategies

Stress ulcer prophylaxis: Antacids and H_2-blockers have been used extensively in the ICU setting to prevent stress ulcer bleeding but have been associated with an increased risk of developing of VAP because they lead to bacterial overgrowth of the gastric contents. Sucralfate prevents stress ulcer bleeding without reducing gastric pH but is more difficult to administer and is less effective than acid-reducing agents [73].

Seven meta-analyses of over 20 randomized trials have evaluated the risk of NP in critically ill patients associated with the use of stress ulcer prophylaxis.[70] Four showed significant reductions in the incidence of pneumonia, and 3 showed similar but non-significant trends toward reduced pneumonia in patients treated with sucralfate. Health care practitioners must weigh the potential benefit in reduction of pneumonia with the use of sucralfate against the increased risk of gastrointestinal bleeding when compared with H2-antagonists. Of note, a recent trial of 287 patients in a surgical ICU assessed the risk of both pneumonia and stress-related bleeding in patients randomized to omeprazole, famotidine, sucralfate, or placebo and found no statistically significant differences in bleeding or pneumonia rates between the 4 study groups [74].

Selective digestive decontamination: Selective digestive decontamination (SDD) involves sterilization of the oropharynx and gastrointestinal tract in mechanically ventilated patients to prevent aspiration of large numbers of potentially pathogenic organisms and subsequent VAP. Most evaluations of SDD have involved oral (and at times gastric) application of topical polymixin, aminoglycoside, and amphotericin. In many cases, investigators have added short courses of intravenous antibiotic therapy [27].

There have been 10 meta-analyses of over 40 randomized trials of SDD that have been recently summarized in systematic reviews [62,70]. The preponderance of evidence suggests that there is a significant reduction in the risk of VAP with the use of SDD. Several meta-analyses also suggest that, in addition to reductions in VAP, the combination of topical and IV antibiotics may provide a mortality benefit [70]. However, because of both the potential risk of inducing bacterial resistance and the laboriousness of the procedure, some experts are still opposed to this strategy. Consequently, the usefulness of routine selective decontamination as a way to prevent HAP is still an unresolved issue.

Conclusions

The intensive care unit, an essential component of modern hospital care is a complex system serving critically ill patients who depend on life support systems. However, as an environment, the ICU also puts patients at risk for the development of nosocomial infection. The risk is associated primarily with severity of patient's condition, type of infectious agents, use of invasive and immunosuppressive procedures, and quality f ICU environment. Pneumonia is the most common infection in ICUs, with morbidity and mortality rates so substantial that it is recognized the most serious infection acquired in the hospital environment. A multidirectional approach, including continuing staff education, minimizing risk factors, and implementing guidelines established by national committees, is needed. Infection-prevention committees can assist in implementing policies. This is an active area of research and we anticipate continued advancements to improve patient care.

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