Original Article

Ventilator-associated pneumonia in a tertiary care hospital in India: incidence and risk factors

Noyal Mariya Joseph[1], Sujatha Sistla[1], Tarun Kumar Dutta[2], Ashok Shankar Badhe[3], Subhash Chandra Parija[1]

Departments of Microbiology[1], Medicine[2] and Anaesthesiology and Critical care[3], Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry, India

Abstract

Background: Knowledge of the incidence of ventilator-associated pneumonia (VAP) and its associated risk factors is imperative for the development and use of more effective preventive measures.

Methodology: We performed a prospective study over a period of 15 months to determine the incidence and the risk factors for development of VAP in critically ill adult patients admitted in different intensive care units (ICUs) of Jawaharlal Institute of Post-graduate Medical Education and Research (JIPMER), a tertiary care hospital in Pondicherry, India.

Results: The incidence of VAP was 30.67 and 15.87 per 1,000 ventilator days in the two different ICUs. In our study 58.3% of the cases were late-onset VAP, while 41.7% were early-onset VAP. Univariate analysis indicated that the following were significantly associated with VAP: impaired consciousness, tracheostomy, re-intubation, emergency intubation, and nasogastric tube. Emergency intubation and intravenous sedatives were found to be the specific risk factors for early onset VAP, while tracheostomy and re-intubation were the independent predictors of late-onset VAP by multivariate logistic regression analysis.

Conclusions: Knowledge of these risk factors may be useful in implementing simple and effective preventive measures including non-invasive ventilation, precaution during emergency intubation, minimizing the occurrence of reintubation, avoidance of tracheostomy as far as possible, and minimization of sedation.

Key words: ventilator-associated pneumonia, risk factors, emergency intubation**,** tracheostomy

J Infect Dev Ctries 2009; 3(10):771-777.

(Received August 6, 2009 – Accepted October 13, 2009)

Introduction

Ventilator-associated pneumonia (VAP) is defined as pneumonia occurring more than 48 hours after the initiation of endotracheal intubation and mechanical ventilation (MV) [1]. The incidence of VAP varies among different studies, depending on the definition, the type of hospital or ICU, the population studied, and the level of antibiotic exposure [2,3]. The lack of consensus regarding the most appropriate method to diagnose VAP also partly explains why incidence rates vary widely from one study to another. The incidence of VAP ranges from 13 to 51 per 1,000 ventilator days [4]. VAP is usually classified as either early onset, occurring within the first four days of MV or late onset, developing five or more days after initiation of MV [2].

Intubation and mechanical ventilation are associated with 6- to 21-fold increased risk of acquiring pneumonia in hospital settings [5]. In spite of the advances in the diagnosis, treatment, and prevention of VAP, it continues to be a major cause of morbidity and mortality among critically ill patients [6,7]. Several risk factors may predispose patients to either colonization of the respiratory tract with pathogenic microorganisms and/or aspiration of contaminated secretions [7-9]. Knowledge of the incidence of VAP and their associated risk factors are imperative for development and use of more effective preventive measures.

We performed a prospective study to determine the incidence of VAP in adult patients undergoing mechanical ventilation and to identify the main risk factors for development of VAP in critically ill patients admitted in different ICUs of the Jawaharlal Institute of Post-graduate Medical Education and Research (JIPMER), a tertiary care hospital in Pondicherry, India.

Table 1. Modified Clinical Pulmonary Infection Score (CPIS)*.

CPIS points	0	1	2
Temperature (^oC)	≥ 36.5and ≤ 38.4	≥ 38.5 and ≤ 38.9	≥ 39 or ≤ 36
Leucocyte count (per mm^3)	4,000 - 11,000	< 4,000 or > 11,000	< 4,000 or > 11,000 + band forms ≥ 500
Tracheal secretions	Rare	Abundant	Abundant + Purulent
PaO_2/ FiO_2 mm Hg	> 240 or ARDS	-	≤ 240 and no ARDS
Chest radiograph	No infiltrate	Diffuse infiltrate	Localized infiltrate
Culture of tracheal aspirate	Light growth or no growth	Moderate or heavy growth of pathogenic bacteria	Moderate or heavy growth of pathogenic bacteria and presence of the same bacteria in Gram stain

* Modified from Pugin *et al.* [10].

Materials and methods

Setting and Subjects

A prospective study was conducted over a period of 15 months from October 2006 to December 2007 in the departments of Microbiology, Medicine, and Anesthesiology and Critical Care at the Jawaharlal Institute of Post-graduate Medical Education and Research (JIPMER), a tertiary care hospital in Pondicherry, India. All the adult patients on mechanical ventilation (MV) for more than 48 hours in the Medicine Intensive Care Unit (MICU) and the Critical Care Unit (CCU) were included in this study. Patients with pneumonia prior to MV or within 48 hours of MV were excluded. This study was approved by the institute's research and ethical committees and informed consent was obtained from each patient's next of kin.

Study design and data collection

A group of attending physicians and nurses prospectively collected data on all patients who received mechanical ventilation. From each patient the following data were collected at ICU admission: name, age, gender, hospital number, primary diagnosis, date of admission in hospital and ICU. The presence or absence of the potential risk factors for the development of VAP was recorded. The study patients were monitored at every third day for the development of VAP using clinical and microbiological criteria until either discharge or death. The relevant data were recorded from medical records, bedside flow sheets, radiographic reports, and reports of microbiological studies of the patients.

Criteria for diagnosing VAP

The patients fulfilling both the clinical and microbiological criteria were diagnosed to be suffering from VAP. Clinical criteria included modified clinical pulmonary infection score (CPIS) > 6 (Table 1) [10] and microbiological criteria included positive Gram stain (> 10 polymorphonuclear cells/low power field and ≥ 1 bacteria/oil immersion field with or without the presence of intracellular bacteria) and quantitative endotracheal aspirate culture showing ≥ 10^5 CFU/ ml [11-13].

Identification of VAP pathogens

Quantitative culture of endotracheal aspirate (EA) was performed for identification of VAP pathogens. EA was serially diluted in sterile normal saline as 1/10, 1/100, 1/1,000, and 0.01 ml of 1/1,000 dilution was inoculated on 5% sheep blood agar. After incubation at 37^{o}C in a 5% CO_2 incubator for 24 hours, a colony count was done and expressed as number of colony forming units per ml (CFU/ml). The microorganisms isolated at a concentration of more than 10^5 CFU/ ml were considered as VAP pathogens and were identified based on standard bacteriological procedures including Gram's stain, colony morphology on blood agar and Mac Conkey agar, and biochemical reactions [14]. Non-glucose-fermenting, motile, oxidase positive, nitrate reducing, Gram-negative bacilli, with a characteristic sweet grape-like odour and distinctive blue-green pigment were identified *Pseudomonas aeruginosa*. Non-glucose-fermenting, non-motile, oxidase negative, nitrate non-reducing, Gram-negative coccobacilli, producing acid from glucose oxidatively, were identified as *Acinetobacter baumannii*. Oxidase-

negative, catalase positive, nitrate reducing, non-spore forming, Gram-negative bacilli, fermenting glucose and other carbohydrates, were considered as members of Enterobacteriaceae. Catalase-positive, mannitol fermenting, coagulase producing, Gram-positive cocci in clusters, with characteristic golden yellow pigment and hemolysis, were identified as *Staphylococcus aureus*. Methicillin-resistant *Staphylococcus aureus* (MRSA) were identified based on their ability to grow on oxacillin screen agar with 6 µg/ ml oxacillin and 4% NaCl.

Table 2. Patient characteristics.

Parameter	Non-VAP (n = 164)	VAP (n = 36)	*P* value (2-tailed)
Age (mean ± SD)	36.8 ± 16.3	41.4 ± 14.7	0.1770
Gender			
Male	95 (57.9%)	24 (66.7%)	0.4354
Female	69 (42.1%)	12 (33.3%)	
Primary diagnosis			
Poisoning[a]	55	10	0.6372
Neurological disorders (GBS, MND)	7	7	0.0046
Intra-abdominal diseases	12	4	0.4959
Snake bite	12	4	0.2694
CNS infections (encephalitis/ meningitis)	2	3	0.0249
Pregnancy-related disorders	12	2	1.0000
Fracture	3	1	0.4808
Tetanus	5	1	1.0000
Cardiovascular disease	9	1	1.0000
Subdural/ extradural hemorrhage	4	1	0.5603
Neuromuscular disorders	7	1	1.000
Leptospirosis	0	1	0.1800
Miscellaneous*	35	0	0.0050

[a] It includes organophosphorous (insecticide), yellow oleander and atropine poisoning. VAP – Ventilator-associated pneumonia; GBS – Guillain Barre syndrome; MND – Motor neuron Disease.
* Acute flaccid paralysis, frontotemporal intracranial space occupying lesion, cerebrovascular accident, multiple injury, hanging, sepsis, chondrosarcoma, renal cell carcinoma, chronic obstructive pulmonary disease with cardiac failure, CO_2 narcosis, diabetes mellitus with hypertension, diabetic nephropathy, neuroglycopenia, post hysterectomy, severe anaemia, chronic or acute renal failure.

Statistical Analysis

Results were expressed as mean ± SD. The chi-square test or Fisher's exact test was used to compare different groups. Univariate analysis was used to compare the variables for the outcome groups of interest (patients with VAP versus patients without VAP). Comparisons were unpaired and all tests of significance were two-tailed. Continuous variables were compared using Student's *t* test for normally distributed variables. We confirmed the results of these tests, with logistic regression analysis, using statistics software (SPSS 16.0, SPSS Inc, Chicago, Illinois). This step was necessary to avoid producing spuriously significant results with multiple comparisons. Results of the logistic regression analyses are reported as estimated odd ratios with their 95% confidence intervals. All *P* values < 0.05 were considered statistically significant and were based on univariate analysis.

Results

During a 15-month period (October 2006 to December 2007), 882 and 364 consecutive patients admitted to MICU and CCU respectively were prospectively evaluated. Of these patients, 607 (68.8%) in MICU and 101 (27.7%) in CCU were not intubated as there were no indications for MV. Among those requiring MV, 175 (19.8%) and 163 (44.8%) patients were mechanically ventilated for less than 48 hours in MICU and CCU respectively. One hundred patients (11.3%) from MICU and 100 patients from CCU (27.5%) received MV for more than 48 hours and comprised the study cohort.

Incidence

Of the 200 patients, 36 (18%) developed VAP during their ICU stay. The overall incidence of VAP was 22.94 per 1,000 ventilator days. The incidence of VAP in MICU and CCU were 30.67 and 15.87 per 1,000 ventilator days respectively. There was no statistically significant difference in the incidence of

VAP among MICU and CCU patients (two-tailed *P* value is 0.0976; Yates corrected Chi-square value is 2.74).

Table 3. Univariate analysis of risk factors for VAP.

S. No.	Risk factor	Non-VAP (n = 164) (%)	VAP (n = 36) (%)	Relative risk (95% confidence limits)	Attributable risk	*P* value
1.	Supine head position	8 (4.9)	1 (2.8)	0.61 (0.09 to 3.94)	-	1.0000
2.	Stress ulcer prophylaxis	151 (92.1)	36 (100.0)	Infinity	-	0.1308
3.	Impaired consciousness	14 (8.5)	8 (22.2)	2.31 (1.21 to 4.42)	56.7	0.0339
4.	Tracheostomy	25 (15.2)	11 (30.6)	2.00 (1.09 to 3.69)	50.0	0.0541
5.	Re-intubation	9 (5.5)	6 (16.7)	3.04 (1.15 to 8.0)	67.1	0.0327
6.	Emergency intubation	1 (0.6)	5 (13.9)	5.22 (3.22 to 8.44)	80.8	0.0008
7.	Nasogastric tube	35 (21.3)	15 (41.7)	2.14 (1.20 to 3.83)	53.3	0.0194
8.	Surgery	24 (14.6)	5 (13.9)	0.95 (0.40 to 2.24)	-	0.8836
9.	Burns	0 (0)	0 (0)		-	-
10.	Chronic renal failure	4 (2.4)	0 (0)	0.0 (-inf to inf)	-	1.0000
11.	Trauma	11 (6.7)	1 (2.8)	0.45 (0.07 to 3.0)	-	0.6976
12.	IV sedatives	33 (20.1)	6 (16.7)	0.83 (0.37 to 1.85)	-	0.8091
13.	Steroid therapy	32 (19.5)	8 (22.2)	1.14 (0.56 to 2.31)	-	0.8902
14.	Duration of MV ≥ 5 d	106 (64.6)	21 (58.3)	0.80 (0.44 to 1.46)	-	0.6031

Characteristics of patients with and without VAP

Of the 200 study patients, 119 were men (59.5%) and 81 (40.5%) were women. The mean ± SD age of patients receiving MV was 37.6 ± 16.1 years (range 14 to 80 years). There was no statistically significant difference in the age and sex distribution of the patients in VAP and non-VAP groups (Table 2). The most frequent cause of ICU admission was suicidal poisoning (32.5%). Majority of the cases of poisoning were due to organophosphorous (insecticide), followed by yellow oleander and atropine. The patients who had neurological disorders and CNS infections were significantly predisposed for the development of VAP (*P* value 0.0046 and 0.0249 respectively) (Table 2).

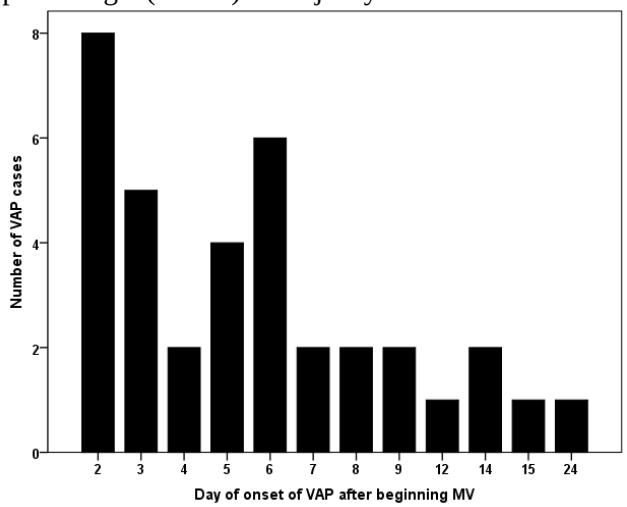

Fig. 1. Distribution of onset of VAP.

Time of onset of VAP

The onset of VAP was more likely to occur during the first two weeks of MV as 94% (34 out of 36) cases occurred during this period (Figure 1). In this study, 58.3% of the cases were late-onset VAP, while 41.7% were early-onset VAP.

Causative agents

Most cases of VAP were caused by Gram-negative bacteria, which accounted for 80.9% of causative organisms. *Pseudomonas aeruginosa* (21.3%) and *Acinetobacter baumannii* (21.3%) were the most common Gram-negative bacteria associated with VAP and *Staphylococcus aureus* (14.9%) was the most common Gram-positive bacteria among patients with VAP. MRSA accounted for 42.9% of the VAP due to *Staphylococcus aureus*. VAP was polymicrobial in 10 patients (27.8%).

Table 4. Multivariate logistic regression analysis of risk factors for VAP.

	***P* value**	**Estimated Odds ratio**	**95% confidence interval**	
			Lower	**Upper**
Impaired consciousness	0.170	2.158	.720	6.469
Tracheostomy	0.035	2.669	1.073	6.634
Re-intubation	0.089	3.075	.841	11.235
Emergency intubation	0.006	25.051	2.571	244.055
Nasogastric tube	0.061	2.282	.963	5.406

Table 5. Risk factors significantly associated with early-onset and late-onset VAP, by univariate analysis.

Risk factor	**Non-VAP (n = 58) (%)**	**VAP (n = 15) (%)**	**Relative risk (95% confidence limits)**	**Attributable risk**	***P* value**
Early-onset VAP					
Emergency intubation	1 (1.7)	4 (26.7)	4.95 (2.46 to 9.92)	79.8	0.0055
Intravenous sedatives	6 (10.3)	6 (40.0)	3.39 (1.48 to 7.75)	70.5	0.0127
Late-onset VAP					
Tracheostomy	25 (23.6)	11 (52.4)	2.78 (1.29 to 5.97)	64.0	0.0159
Re- intubation	1 (0.9)	3 (14.3)	5.13 (2.52 to 10.41)	80.5	0.0142

VAP – Ventilator-associated pneumonia.

Table 6. Risk factors significantly associated with early-onset and late-onsetVAP, by multivariate logistic regression.

	***P* value**	**Adjusted Odds ratio**	**95% confidence interval**	
			Lower	**Upper**
Early-onset VAP				
Emergency intubation	0.006	27.189	2.527	292.573
Intravenous sedatives	0.007	7.248	1.712	30.694
Late-onset VAP				
Tracheostomy	0.032	3.006	1.097	8.235
Emergency Intubation	0.043	11.853	1.087	129.289

VAP – Ventilator-associated pneumonia.

Risk factors

1) Risk factors for VAP: Univariate analysis indicated that the following were significantly associated with VAP: impaired consciousness, tracheostomy, re-intubation, emergency intubation, and nasogastric tube (Table 3). Selected risk factors were entered into a logistic regression model to perform the multivariate analysis, which revealed that the independent risk factor for VAP were emergency intubation and tracheostomy (Table 4).

2) Specific risk factors for early-onset VAP: Emergency intubation and intravenous sedatives were the specific risk factors for development of early-onset VAP by both univariate analysis and multivariate logistic regression (Table 5 and 6).

3) Specific risk factors for late-onset VAP: Tracheostomy and re-intubation were found to be the independent predictors of late-onset VAP by both univariate analysis and multivariate logistic regression (Table 5 and 6).

Outcome

In this study the crude mortality rate of patients with VAP was 16.2%. There was no statistically significant difference in mortality between VAP and

non-VAP groups (16.2% vs 20.5%; RR, 0.89; 95% CI, 0.40 to 1.95; *P* 0.9486).

Discussion

VAP is an important nosocomial infection among ICU patients receiving MV. The incidence of VAP (22.94 per 1,000 ventilator days) in our study was high, almost similar to another Indian study [15]. But in other Asian countries the incidence rate is relatively less, ranging from 9 to 12 per 1,000 ventilator days [16-18]. In this study there was no statistically significant difference in the incidence of VAP among MICU and CCU patients, in accordance with Torres *et al*., who found that the type of ICU population did not influence the incidence of VAP [19]. But, in general, the surgical ICUs have higher rates of VAP compared to the medical ICUs [20]. The incidence of nosocomial pneumonia was reported as 21.6% in patients admitted to a cardiothoracic ICU, 14% in another surgical ICU, and 9.3% in a medical ICU [21].

In the present study, 41.7% of cases were early-onset VAP, which is similar to other studies reporting early-onset VAP in almost half of all VAP episodes [3,22]. It was observed that majority of the VAP episodes occurred within the first two weeks of MV. The interaction of several risk factors during the initial days of MV put the patient at higher risk and also the exhaustion of most vulnerable patients during the first few weeks leads to the decline in the occurrence of VAP in later days [23].

Patients with neurological disorders and CNS infections in our study group were significantly predisposed for the development of VAP. These patients had impaired consciousness and inadequate cough reflexes which predisposed them for developing VAP. The other causes of ICU admission did not correspond to the incidence of VAP, although some studies have noted intra-abdominal diseases and multiple injury as significant predisposing factors for VAP [2,3,23]. The relatively fewer patients with these diagnoses in our study group could have failed to prove a significant association with VAP.

Identification of emergency intubation as an independent risk factor suggests that unplanned intubation may be associated with increased rates of aspiration of infected upper airway secretions. Tracheostomy was found to be another independent risk factor as it is probable that leakage of pooled secretions around the tracheostomy tube into the trachea increases tracheal colonization and leads to VAP. Administration of intravenous sedatives to patients on MV might impair their cough reflexes, increasing the risk of aspiration and subsequently predisposing them to development of VAP. Re-intubation also most often results in aspiration contributing to the development of VAP [7,24].

Supine head position, stress ulcer prophylaxis, surgery, burns, chronic renal failure, trauma, steroid therapy and duration of MV $\geq$ 5d were documented as independent risk factors for the development of VAP by multivariate analysis in different studies [2,3,7,9]. Supine head position and trauma were present only in a very few patients in both the VAP and non-VAP groups in our study. Similarly, burns were present in none of the study cohorts and stress ulcer prophylaxis was administered to almost all of them; therefore, a significant association of these factors with VAP could not be studied. Steroid therapy also did not show a significant association with VAP as steroid therapy was given only for a short duration in most of our patients. Awareness of the independent risk factors documented in this study may assist in identifying patients at higher risk for VAP, guide implementation of appropriate preventive measures, and modulate potential intervention measures during management.

Our analysis may not have the power to identify all important VAP risk factors in this study population. Despite those limitations, the findings of this study signify the importance of VAP in critically ill patients on MV. Further validation of the risk factors identified in this study is necessary. Additional studies on risk factors for VAP, combined with the knowledge of the causative pathogens, may guide development of more effective preventive strategies for VAP.

To conclude, VAP continues to be a major challenge to the critical care physicians in India and is a common nosocomial infection occurring in mechanically ventilated patients. Knowledge of the important risk factors predisposing to VAP may prove to be useful in implementing simple and effective preventive measures including non-invasive ventilation, precaution during emergency intubation, minimizing the occurrence of re-intubation, avoidance of tracheostomy as far as possible, and minimization of sedation.

References

1. Davis KA (2006) Ventilator-associated pneumonia: a review. J Intensive Care Med 21: 211-226.
2. Niederman MS and Craven DE (2005) Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med 171: 388-416.
3. Chastre J and Fagon JY (2002) Ventilator-associated pneumonia. Am J Respir Crit Care Med 165: 867-903.
4. Alp E and Voss A (2006) Ventilator associated pneumonia and infection control. Ann Clin Microbiol Antimicrob 5: 7.
5. Weber DJ, Rutala WA, Mayhall CG (1998) Nosocomial respiratory tract infections and Gram negative pneumonia. In: Fishman AP, Elias JA, Fishman JA, Grippi MA, Kaiser LR, Senior RM, editors. Pulmonary disease and disorders. New York: McGraw-Hill. 2213-2227.
6. Rello J, Ollendorf DA, Oster G, Vera-Llonch M, Bellm L, Redman R, Kollef MH (2002) Epidemiology and outcomes of ventilator-associated pneumonia in a large US database. Chest 122: 2115-2121.
7. Ibrahim EH, Tracy L, Hill C, Fraser VJ, Kollef MH (2001) The occurrence of ventilator-associated pneumonia in a community hospital: risk factors and clinical outcomes. Chest 120: 555-561.
8. afdar N, Crnich CJ, Maki DG (2005) The pathogenesis of ventilator-associated pneumonia: its relevance to developing effective strategies for prevention. Respir Care 50: 725-739.
9. Erbay RH, Yalcin AN, Zencir M, Serin S, Atalay H (2004) Costs and risk factors for ventilator-associated pneumonia in a Turkish university hospital's intensive care unit: a case-control study. BMC Pulm Med 4: 3.
10. Pugin J, Auckenthaler R, Mili N, Janssens JP, Lew PD, Suter PM (1991) Diagnosis of ventilator-associated pneumonia by bacteriologic analysis of bronchoscopic and nonbronchoscopic "blind" bronchoalveolar lavage fluid. Am Rev Respir Dis 143: 1121-1129.
11. Porzecanski I and Bowton DL (2006) Diagnosis and treatment of ventilator-associated pneumonia. Chest 130: 597-604.
12. Wu CL, Yang DI, Wang NY, Kuo HT, Chen PZ (2002) Quantitative culture of endotracheal aspirates in the diagnosis of ventilator-associated pneumonia in patients with treatment failure. Chest 122: 662-668.
13. Koenig SM and Truwit JD (2006) Ventilator-associated pneumonia: diagnosis, treatment, and prevention. Clin Microbiol Rev 19: 637-657.
14. Mackie TJ and McCartney JE (1996) Practical medical microbiology, 14th edition. New York: Churchill Livingstone 978p.
15. Rakshit P, Nagar VS, Deshpande AK (2005) Incidence, clinical outcome, and risk stratification of ventilator-associated pneumonia-a prospective cohort study. Indian J Crit Care Med 9: 211-216.
16. Aly NY, Al-Mousa HH, Al Asar el SM (2008) Nosocomial infections in a medical-surgical intensive care unit. Med Princ Pract 17: 373-377.
17. Suka M, Yoshida K, Uno H, Takezawa J (2007) Incidence and outcomes of ventilator-associated pneumonia in Japanese intensive care units: the Japanese nosocomial infection surveillance system. Infect Control Hosp Epidemiol 28: 307-313.
18. Thongpiyapoom S, Narong MN, Suwalak N, Jamulitrat S, Intaraksa P, Boonrat J, Kasatpibal N, Unahalekhaka A (2004) Device-associated infections and patterns of antimicrobial resistance in a medical-surgical intensive care unit in a university hospital in Thailand. J Med Assoc Thai 87: 819-824.
19. Torres A, Aznar R, Gatell JM, Jimenez P, Gonzalez J, Ferrer A, Celis R, Rodriguez-Roisin R (1990) Incidence, risk, and prognosis factors of nosocomial pneumonia in mechanically ventilated patients. Am Rev Respir Dis 142: 523-528.
20. Craven DE (2000) Epidemiology of ventilator-associated pneumonia. Chest 117: 186S-187S.
21. Kollef MH (1993) Ventilator-associated pneumonia. A multivariate analysis. JAMA; 270: 1965-1970.
22. Kollef MH (2005) What is ventilator-associated pneumonia and why is it important? Respir Care 50: 714-721.
23. Apostolopoulou E, Bakakos P, Katostaras T, Gregorakos L (2003) Incidence and risk factors for ventilator-associated pneumonia in 4 multidisciplinary intensive care units in Athens, Greece. Respir Care 48: 681-688.
24. Hunter JD (2006) Ventilator associated pneumonia. Postgrad Med J 82: 172-178.

Corresponding author

Noyal Mariya Joseph
Department of Microbiology
Mahatma Gandhi Medical College and Research Institute
Pillaiyarkuppam, Pondicherry – 607 402
India
Telephone: 9843094673
Email: noyaljoseph@yahoo.com

Conflict of Interest: No conflict of interest is declared.