

## Original Research

# Exploring Ventilator-Associated Pneumonia in Neurocritical Care Settings

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### ABSTRACT

**Background:** Ventilator-associated pneumonia (VAP) represents the most frequently occurring hospital-acquired infection within the intensive care unit (ICU). This study aimed to determine the incidence of VAP, identify bacterial pathogens isolated from endotracheal aspirates, and assess their antibiotic sensitivity patterns in neurocritically ill patients.

**Materials and Methods:** A prospective cohort study was conducted on 145 neurocritically ill patients who developed VAP after they were admitted to the ICU of a tertiary care hospital. Endotracheal aspirates were collected under strict aseptic conditions using a 22-inch suction catheter attached to a mucus extractor. The aspirates were subjected to Gram staining, biochemical identification, and antimicrobial susceptibility testing.

**Results:** The study reported a 29.66% incidence of VAP. No significant association was found between the underlying neurological condition and the development of VAP. *Pseudomonas aeruginosa* was the most frequently isolated organism, followed by methicillin-resistant *Staphylococcus Aureus* (MRSA). Most isolates demonstrated resistance to commonly used antibiotics. Patients with VAP exhibited higher mortality rates, longer durations of mechanical ventilation, and extended hospital stays compared to non-VAP patients.

**Conclusion:** While VAP does not appear to have a definitive impact on mortality, it may contribute to prolonged ICU stays and extended periods of mechanical ventilation. Timely diagnosis and the initiation of targeted antibiotic therapy are essential to mitigate adverse outcomes associated with VAP.

**Key Words:** Ventilator, Pneumonia, Critical care, *Pseudomonas*.

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### INTRODUCTION

Ventilator-associated pneumonia (VAP) represents the most frequent hospital-acquired infection within intensive care units (ICUs). It is characterized as a lower respiratory tract infection that emerges at least 48 hours after the commencement of invasive mechanical ventilation (MV) [1]. Acute brain injury (ABI), particularly when accompanied by reduced consciousness, is a notable risk factor for respiratory complications, including VAP. Additional factors contributing to the risk of pneumonia include swallowing impairments, advanced age, and the use of sedation [2].

The prevalence of VAP ranges from 9% to 27% among intubated patients [3]. Prolonged mechanical ventilation

and reintubation following extubation are significant risk factors. Delay in administering appropriate antibiotic treatment can escalate mortality linked to VAP, underscoring the importance of initiating therapy without deferring for diagnostic confirmation [4].

Initial empirical antimicrobial therapy should be tailored according to local microbiological data, patient-specific factors, and the institutional sensitivity profile of potential pathogens. Studies have demonstrated that VAP is associated with higher mortality, extended ICU stays, and prolonged durations of mechanical ventilation [5].

This study aimed to assess the incidence of VAP, identify risk factors contributing to its development, and evaluate mortality and outcomes, the spectrum of

bacterial pathogens isolated from tracheal aspirates in VAP cases, and their respective antibiotic susceptibility profiles.

### MATERIAL AND METHODS

A prospective investigation was carried out on 145 individuals diagnosed with acute brain injury who were admitted to the intensive care unit (ICU) of a tertiary care hospital. None of the patients had pneumonia prior to initiation of mechanical ventilation. Elective tracheostomies were performed in selected cases where prolonged mechanical ventilation was anticipated, to reduce the risk of reintubation. Exclusion criteria included patients who either developed pneumonia within 48 hours of admission, presented with pneumonia upon admission, or had acute respiratory distress syndrome (ARDS).

The diagnosis of ventilator-associated pneumonia (VAP) was based on the clinical pulmonary infection score (CPIS), which was assessed daily throughout the duration of ventilator support. A CPIS score exceeding six was used to confirm VAP. Cases of early-onset VAP were defined as those occurring within 72 hours of initiating mechanical ventilation, while late-onset VAP referred to cases identified after 72 hours. For diagnostic sampling, endotracheal aspirates were collected under strict aseptic conditions using a 22-inch suction catheter attached to a mucus extractor. The catheter was introduced approximately 25 cm into the endotracheal tube, and aspiration was performed without the instillation of saline. Subsequently, 4 mL of sterile 0.9% saline was injected into the endotracheal tube to flush exudates into a sterile container. The collected samples were promptly transported to the laboratory for analysis. Precautions were taken to

prevent tracheal mucosal injury and hypoxia during the procedure.

A Gram stain of the aspirates was performed within one hour of collection. Samples were cultured on 5% blood agar and Mac Conkey agar, and the resultant colonies were subjected to Gram staining and biochemical identification. Antimicrobial susceptibility testing was conducted using Mueller-Hinton agar with the Kirby-Bauer disk diffusion method, adhering to Clinical Laboratory Standards Institute (CLSI) guidelines. Patients diagnosed with VAP were initiated on empirical antibiotic therapy based on the likelihood of multi-drug resistant pathogens. Treatment regimens were subsequently adjusted in response to culture sensitivity results.

Primary outcomes included patient mortality, while secondary outcomes comprised the incidence of VAP, identification and sensitivity profiles of isolated pathogens, duration of mechanical ventilation, and length of hospital stay. Associations between patient demographics, disease severity (evaluated via the APACHE II score), and care-related factors (e.g., reintubation) with VAP incidence and mortality rates were also analyzed. Data collected were organized into tables and subjected to statistical analysis using the chi-square test, Fisher's exact test, and paired t-test. A p-value of <0.05 was considered statistically significant.

### RESULTS

The distribution of gender between Non-VAP and VAP groups is presented in Table 1. Among the total participants (n = 145), a higher proportion of males (61.38%) compared to females (38.62%) were observed. However, the observed gender differences between the groups were not statistically significant (p = 0.21).

**Table 1: Gender frequency among study participants**

Gender	Non-VAP		VAP		Total		P Value
	n	%	n	%	n	%	
Male	66	45.52	23	15.86	89	61.38	0.21
Female	36	24.83	20	13.79	56	38.62	

Table 2 shows the distribution of participants across various age groups. The majority of the study population (26.90%) fell within the 21-30 age group, followed by 17.93% in the 51-60 age group. Notably, the percentage of VAP patients within the 21-30 age group (5.52%) was significantly lower than the Non-VAP group (21.38%). The p-value of 0.66 suggests no significant association between age group and VAP status.

**Table 2: Age frequency among study participants**

Age Groups (years)	Non-VAP		VAP		Total		P Value
	n	%	n	%	n	%	
15-20	13	8.97	9	6.21	22	15.17	0.66
21-30	31	21.38	8	5.52	39	26.90	
31-40	10	6.90	6	4.14	16	11.03	
41-50	15	10.34	6	4.14	21	14.48	
51-60	17	11.72	9	6.21	26	17.93	
61-70	12	8.28	4	2.76	16	11.03	
>70	4	2.76	1	0.69	5	3.45	

Table 3 presents the distribution of causative organisms in relation to mortality outcomes. *Pseudomonas aeruginosa* was the most frequently isolated pathogen (30.23%) in the total isolates, with a higher proportion found in the Late VAP group (13 isolates, 9 survivors, 4 non-survivors). The mortality rate for *Pseudomonas aeruginosa* was notable, with 9 survivors (20.93%) and 4 non-survivors (9.30%). MRSA and *Klebsiella pneumoniae* had similar frequencies (23.26%), with higher non-survivor rates for *Klebsiella pneumoniae* (7 non-survivors, 16.28%) compared to MRSA (3 non-survivors, 6.98%).

**Table 3: Causative Organisms and Mortality among study participants**

Organism	Total Isolates		Early VAP (n)	Late VAP (n)	Survivors n (%)	Non-survivors n (%)
	n	%				
<i>Pseudomonas aeruginosa</i>	13	30.23	0	13	9 (20.93)	4 (9.30)
MRSA	10	23.26	3	7	7 (16.28)	3 (6.98)
<i>K. pneumoniae</i>	10	23.26	3	7	3 (6.98)	7 (16.28)
<i>A. baumannii</i>	7	16.28	3	4	1 (2.33)	6 (13.95)
Enterococci	1	2.33	0	1	1 (2.33)	0 (0.00)
<i>S. pneumoniae</i>	1	2.33	1	0	1 (2.33)	0 (0.00)
<i>Candida</i>	1	2.33	0	1	1 (2.33)	0 (0.00)
Total	43	100	10	33	23 (53.49)	20 (46.51)

Table 4 illustrates the antibiotic resistance patterns of the microbial isolates. *Pseudomonas aeruginosa* exhibited high sensitivity to Colistin, Imipenem, and Meropenem, while showing resistance to Cefoperazone + Sulbactam and Ceftazidime. MRSA was highly sensitive to Linezolid and Vancomycin, but resistant to Methicillin and Oxacillin. *Klebsiella pneumoniae* showed sensitivity to Colistin and Polymyxin B, but exhibited intermediate resistance to Imipenem and Meropenem, with resistance to Cefotaxime and Ceftazidime.

**Table 4: Antibiogram pattern of the microbial isolates**

Organism	Highly Sensitive	Intermediate	Resistant
<b><i>Pseudomonas aeruginosa</i></b>	Colistin, Imipenem, Meropenem, Polymyxin	Gatifloxacin, Piperacillin + Tazobactam	Cefoperazone + Sulbactam, Ceftazidime, Levofloxacin
<b>MRSA</b>	Linezolid, Vancomycin	Clindamycin, Gatifloxacin, Levofloxacin	Amoxicillin + Clavulanate, Erythromycin, Methicillin, Oxacillin
<b><i>Klebsiella pneumoniae</i></b>	Colistin, Polymyxin B	Gatifloxacin, Imipenem, Meropenem,	Cefotaxime, Ceftriaxone, Ceftazidime
<b><i>Acinetobacter baumannii</i></b>	Colistin, Polymyxin B	Imipenem, Meropenem,	Cefoperazone + Sulbactam, Levofloxacin, Piperacillin + Tazobactam
<b><i>Streptococcus pneumoniae</i></b>	Imipenem, Meropenem, Vancomycin	Ceftazidime, Ceftriaxone, Penicillin	Chloramphenicol, Erythromycin, Ofloxacin, Tetracyclines,
<b>Enterococci</b>	Linezolid, Vancomycin	Cephalosporin, Penicillins	Gentamicin, Ofloxacin

## DISCUSSION

The incidence of VAP in this study was found to be 30%, which aligns closely with Gupta et al. [3], who reported an incidence of 28%. There was no significant association between gender, age, and VAP development, consistent with the findings of Gupta et al. [3]. A variety of clinical cases were included in the study. Patients requiring longer durations of mechanical ventilation were more likely to develop VAP. In contrast, patients who needed shorter periods of ventilation exhibited lower VAP rates. No significant correlation was found between primary diseases and the development of VAP, which corroborates the findings of Gupta et al. [3] and Awasthi et al. [4].

In our study, CVA patients contributed most significantly to the mortality rate, with sepsis as the second leading cause, although the relationship between diseases and mortality was not statistically significant. The CPIS system was employed as a diagnostic tool for identifying VAP, with a score >6 indicating pneumonia. This system was highly sensitive, as supported by studies by Luyt et al. [5] and Croce et al. [6]. Reintubation was found to be a significant risk factor for VAP. This finding is consistent with those of Gupta et al. [3], Panwar et al. [7], and Rit et al. [8]. The increased risk of VAP with reintubation may stem from repeated intubation and longer ventilation periods, as well as potential aspiration risk during the interval between extubation and reintubation.

Although early tracheostomy appeared to reduce VAP incidence, this was not statistically significant. *P. aeruginosa* was the most frequently isolated pathogen, all from late-onset VAP cases. The next most common pathogen was MRSA, with majority isolates from late-onset VAP cases. However, no significant correlation was observed between the infecting organism and VAP type. Other frequently isolated organisms included *Klebsiella pneumoniae* and *Acinetobacter baumannii*. This finding aligns with the study by Rit et al. [8].

The antibiotic resistance patterns revealed that most *P. aeruginosa* strains were resistant to commonly used beta-lactam antibiotics, but they remained highly sensitive to polymyxin B, colistin, meropenem, and imipenem. All isolated *S. aureus* strains were MRSA, sensitive to linezolid and vancomycin, but resistant to methicillin, oxacillin, amoxicillin + clavulanic acid, and erythromycin. Most *K. pneumoniae* isolates were ESBL-producing, with one isolate resistant to both carbapenems, but sensitive to polymyxin and colistin. *A. baumannii* demonstrated even higher carbapenem resistance, with about 50% of isolates resistant, although they remained sensitive to polymyxin B and colistin. These findings indicate an increasing prevalence of drug-resistant pathogens in our setting, contributing significantly to VAP, consistent with the

antibiogram profiles found in studies by Ijjaj et al. [10], Krishnamurthy et al. [11], and Gupta et al. [3].

The overall mortality rate in our study was 46.51%, with the VAP group showing higher mortality compared to the non-VAP group, though the difference was not statistically significant. Although VAP was not independently associated with mortality, the mortality rate was higher in VAP patients. Previous studies have shown mortality rates ranging from 30% to 50%, with VAP patients exhibiting significantly higher mortality than non-VAP patients. These results were similar to those of Gupta et al. [3] and Panwar et al. [8]. Naved et al. [12] and Gupta et al. [3] used the APACHE II score to assess patient condition at admission, finding that patients with higher scores had a higher mortality rate, supporting our study's findings. Mortality was further influenced by the type of organism isolated, with the highest mortality associated with *A. baumannii* and *K. pneumoniae*.

The mean duration of mechanical ventilation was significantly longer in VAP patients compared to non-VAP patients, confirming a highly significant difference between the two groups regarding ventilation duration. Gupta et al. [3] reported similar findings, with VAP patients requiring longer ventilation than non-VAP patients. Awasthi et al. [4] observed similar results in VAP patients aged 1 to 12 years. However, no significant difference in mechanical ventilation duration was found between survivors and non-survivors. VAP patients also had longer hospital stays compared to non-VAP patients, consistent with findings by Dubey et al. [13] and Gupta et al. [3], although there was no significant difference in hospital stay duration between survivors and non-survivors. The mean ICU stay was significantly longer in VAP patients, contributing to increased treatment costs, which is a crucial factor for patients' families in India.

VAP, though often transient and detectable early in ICU, complicates the acute phase of neurological illness, thus impacting mortality less significantly. Advancements in antibiotic stewardship in European settings have improved healthcare outcomes, as indicated by [14]. Additionally, interventions such as re-education of neuro-ICU staff and reducing brain imaging transports may help reduce infection rates [2]. VAP remains an independent risk factor for prolonged ICU stays and increased use of mechanical ventilation, emphasizing its impact on healthcare costs and resource utilization.

## CONCLUSION

VAP is frequently observed among neurocritically ill patients, though its prevalence varies significantly based on the specific type of brain injury. While VAP does not appear to have a definitive impact on mortality, it may contribute to prolonged ICU stays and

extended periods of mechanical ventilation. Future research involving larger cohorts of neurocritically ill patients should focus on evaluating the effects of antibiotic regimens and the role of isolated pathogens in the development and management of VAP.

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