

RISK FACTORS FOR VENTILATOR-ASSOCIATED PNEUMONIA IN A TERTIARY HOSPITAL FROM JANUARY 2017 TO DECEMBER 2021

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ABSTRACT

Background: Ventilator-Associated Pneumonia (VAP) is recognized as a significant nosocomial infection globally contributing to higher mortality. Currently, there are no local studies to document VAP prevalence and risk factors.

Objective: To determine the prevalence of VAP and the risk factors for VAP among intubated patients in the Lung Center of the Philippines (LCP) from 2017 to 2021.

Methodology: This is a retrospective cohort study that reviewed the medical charts of adult intubated patients admitted to the critical care units of LCP from 2017–2021. The following data were extracted: the number of VAP cases and patient-, treatment-, and environment-related factors. Descriptive statistics were used to describe the baseline data. Binary Logistic Regression analysis was performed, reporting Odds Ratios (OR) with 95% Confidence Intervals. Multiple Regression analysis was utilized to assess predictors for continuous secondary outcomes, such as length of hospital stay. Chi-square test was used for determining associations between categorical variables where regression was not applicable. A p-value of < 0.05 was considered statistically significant. Data analysis was performed using Minitab statistical software.

Results and Discussion: Out of the 412 patients, 269 developed VAP (65.3%). The mean age was 61 years, with the majority being male (66%). Patient-related factors: sex, age, and smoking history were not associated with increased VAP risk, while Chronic Kidney Disease (CKD) was associated with lower risk of VAP (OR 0.11, $p = 0.03$). This inverse relationship was likely driven by the conservative fluid management and earlier extubation typical in renal patients. Admission diagnoses requiring intubation, specifically Community-Acquired Pneumonia High Risk (CAP-HR) (OR 2.67, $p = 0.01$) and Hospital-Acquired Pneumonia (HAP) (OR 3.84, $p = 0.04$), were associated with a higher risk of VAP, reflecting pre-existing lung inflammation and compromised local immunity. Treatment and environmental variables strongly influenced infection development; longer intubation days (OR 9.85, $p < 0.01$), tracheostomy (OR 6.86, $p < 0.01$), multiple antibiotic use (OR 1.13, $p < 0.01$), and a longer duration of hospital admission (OR 1.10, $p < 0.01$) were all linked to a higher risk of VAP, underscoring the hazards of prolonged device exposure and dysregulated microbial flora. Conversely, empiric antibiotic therapy (OR 0.37, $p < 0.01$) was associated with a lower risk, suggesting early suppression of initial colonizing pathogens. Regarding clinical outcomes, acute coronary syndrome, septic shock, and lung cancer were linked to higher mortality, whereas Chronic Obstructive Pulmonary Disease (COPD) was associated with lower mortality. Furthermore, HAP, cor pulmonale, late-onset VAP, multiple antibiotic use, dialysis, and tracheostomy significantly prolonged hospital stays. Notably, specific components of the VAP prevention bundle, including oral care and suctioning, correlated with increased mortality, a finding likely reflecting documentation bias among the most critically ill rather than direct causation.

Conclusions: The prevalence of VAP in the critical care units was high at 65.3%. Significant risk factors driving VAP development included admission for CAP-HR or HAP, prolonged intubation and hospitalization, tracheostomy, and multiple antibiotic use, while CKD and empiric antibiotic therapy were associated with a lower risk. We recommend standardizing VAP prevention bundles and strictly enforcing antimicrobial stewardship programs to mitigate these risks.

Keywords: Ventilator-Associated Pneumonia (VAP), Nosocomial infections, Intubation, Risk factors, Critical care

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Ventilator-Associated Pneumonia (VAP) is a form of pneumonia that develops more than 48 hours after mechanical ventilation and is a leading cause of hospital-acquired infections (HAI), contributing to high morbidity and mortality rates globally.¹ It affects approximately 5% to 40% of patients on a mechanical ventilator for over two days.² This variability may reflect differences in diagnostic criteria and hospital settings.¹ Understanding of VAP pathogenesis, risk factors, and prevention strategies has made notable strides. Identified risk factors include patient-specific variables like age and comorbidities, as well as treatment and procedural factors.³

However, challenges remain, such as inconsistent diagnosis and the rise of multidrug-resistant organisms (MDRO) due to improper antibiotic use. In regions like the WHO South-East Asia Region, VAP continues to be a major burden despite advancements in infection control practices.⁴ Prevention efforts focus on bundled interventions, including head-of-bed elevation, sedation management, secretion drainage, oral care, and maintaining endotracheal cuff pressure.^{5,6} While these measures have shown varying success, they remain promising in reducing VAP incidence.⁷

Thus, this study aimed to determine the VAP prevalence and the patient-related, treatment-related, and environment-related risk factors in adult intubated patients in the critical care units of Lung Center of the Philippines (LCP) from 2017 to 2021. Hospital-specific VAP incidences and risk factors are expected to inform tailored infection control strategies. Improved data collection and analysis can further refine prevention bundles and educational efforts, ultimately enhancing hospital policies and mitigating VAP's impact on patient outcomes.

METHODS

Study Design

This research utilized a retrospective cohort study design. Medical records of adult intubated patients admitted in critical care units of the LCP from 2017 to 2021 were reviewed to gather the target exposures, such as patient-related, treatment-related, and environment-related factors, and the outcome of interest, the occurrence of VAP, during their hospital stay.

Study Population and Duration

The target population included all admitted patients who were intubated (with a diagnosis of Acute Respiratory Failure [ARF] or Acute Respiratory Distress Syndrome [ARDS]) in LCP from January 1, 2017, to December 31, 2021. We included 1) intubated adult patients admitted to the Lung Center of the Philippines from January 1, 2017, to December 31, 2021, and 2) those who were admitted to a critical care unit or ward 3B. We excluded patients who 1) were admitted in the hospital for less than 48 hours, 2) signed limitation in medical care (with DNR or DNI

directives), 3) were discharged against medical advice, 4) were transferred out to another facility, 5) were admitted as a transfer from another facility, or 6) had incomplete records or charts (unable to view Chest X-ray result and endotracheal aspirate culture result).

Sample Size and Sampling Strategy

Using G*Power 3.1.9.7, a minimum of 190 patients was required for this study based on an odds ratio of 1.9 for patients with COPD developing Ventilator-Associated Pneumonia.⁸ This computation accounted for a 5% level of significance and 90% power. Total enumeration was used to achieve the desired sample size and maximize statistical power, with all charts screened and assessed for eligibility.

Study Procedure

A comprehensive roster of patients diagnosed with ARF and/or ARDS was retrieved from the medical records of the Lung Center of the Philippines. Each patient's chart underwent a detailed review by the principal investigator based on predefined inclusion and exclusion criteria. A final cohort was then identified, with each participant assigned a unique identifier code. Data were extracted by the principal investigator and a trained research assistant using a structured data collection tool from hospital charts and the WEB laboratory information system online database. VAP identification was based on the diagnosis of the attending physician and/or LCP-Infection Control Committee (ICC) records.

Data extracted included patient-related factors (age, sex, smoking history, comorbidities, reason for intubation, and etiologic agents), treatment-related factors (VAP bundle orders, duration of mechanical ventilation, antibiotic use history, and tracheostomy), and environment-related factors (ward of admission and duration of admission before VAP). Data were encoded in an MS Excel Worksheet by the trained research assistant. Data privacy and confidentiality were observed, and data protection measures (restricted access, de-identified data) were implemented. Regarding missing data, charts with incomplete critical records (missing Chest X-ray or endotracheal aspirate culture results) were excluded from the study (listwise deletion) to ensure data integrity.

Statistical Analysis

Descriptive statistics were used to summarize the data; categorical variables were reported as counts and percentages, while continuous variables were expressed as means and standard deviations. To determine the association between risk factors and dichotomous outcomes (such as VAP development and mortality), binary logistic regression analysis was performed, reporting Odds Ratios (OR) with 95% Confidence Intervals. Multiple regression analysis was utilized to assess predictors for continuous secondary outcomes, such as length of hospital stay. Chi-square test was used for determining associations between categorical variables where regression was not applicable. A p-value of < 0.05

was considered statistically significant. Data analysis was performed using Minitab statistical software.

Ethical considerations

This study was approved by and was under the oversight of the LCP-Institutional Ethics and Review Board (LCP-PF-009-2023) before implementation. A waiver of informed consent was secured from the LCP-IERB.

RESULTS

Recruitment

From January 2017 to December 2021, a total of 3,462 patients were identified with a diagnosis of ARF and/or ARDS (Figure 1). A total of 1,762 charts were successfully retrieved, and a total of 412 patients met the criteria for inclusion. A total of 1,350 patients were excluded for the following reasons: 535 were admitted for less than 48 hours, 367 had limitations of care such as do-not-intubate (DNI) and/or do-not-resuscitate (DNR) directives, 169 refused ICU admission, 117 were on non-invasive ventilation (NIV), 102 were intubated for less than 48 hours, 19 were transferred from another hospital, and 39 were discharged against medical advice. Of the 412 included patients, 269 developed VAP while 143 did not have VAP during admission.

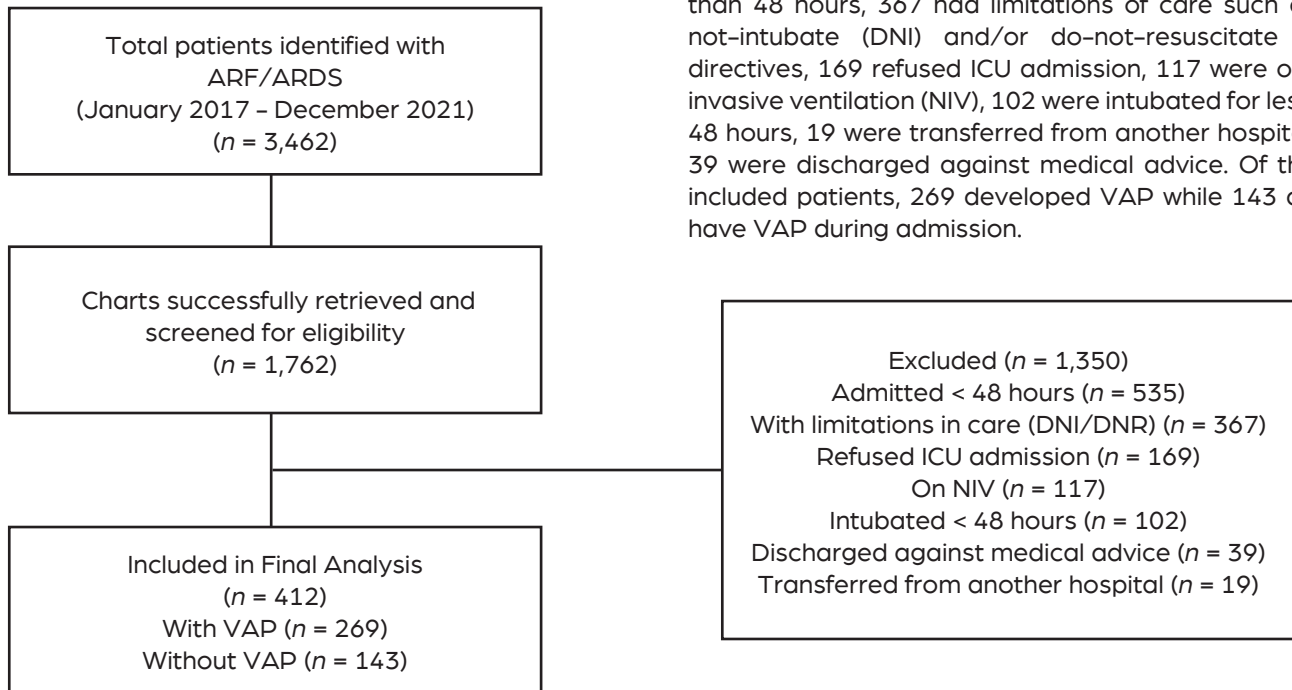


Figure 1. Flow diagram showing patient selection, exclusion and final study population



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VENTILATOR-ASSOCIATED PNEUMONIA

Bundles of Care

APPLY FOR ALL INTUBATED/MECHANICALLY VENTILATED PATIENTS

To strengthen our infection prevention measures, especially against ventilator-associated pneumonia (VAP), all units must implement the updated VAP Bundles of Care. This bundle is based on strong current evidence supporting practices that significantly lower the risk of VAP.

Demographic and Clinical Characteristics

A total of 412 intubated patients were included in the study (Table 1). The mean age was 61.21 years (SD 15.00), with a male predominance (66.01%). Hypertension

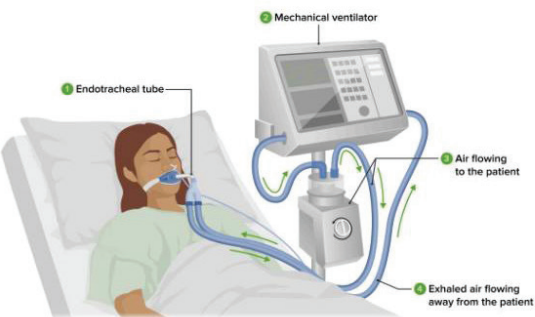
(40.00%) was the most common comorbidity, followed by pulmonary tuberculosis (21.05%) and diabetes mellitus (20.53%). Demographic factors and comorbidities were generally similar between patients with and without VAP.

Table 1. Baseline characteristics and presence of VAP

	Ventilator-Associated Pneumonia		
	Total (n = 412)	With (n = 269)	Without (n = 143)
	Frequency (%); Mean $\pm$ SD		
Age, years	61.21 $\pm$ 15	61.52 $\pm$ 16.74	60 $\pm$ 14.57
Sex: Male	272 (66.01)	169 (62.82)	103 (72.02)
Female	140 (33.99)	100 (37.17)	40 (27.07)
Smoking history: Non-smoker	190 (46.11)	152 (56.50)	38 (26.57)
Smoker	170 (41.26)	115 (42.75)	55 (53.39)
Previous smoker	52 (12.62)	37 (13.75)	15 (10.48)
Comorbidity			
Hypertension	169 (40)	94 (35.83)	75 (46.38)
Pulmonary tuberculosis	88 (21.05)	82 (20)	6 (23.19)
Diabetes mellitus	82 (20.53)	54 (20.66)	28 (20.29)
COPD	81 (20.53)	45 (17.36)	36 (26.09)
Bronchial asthma	58 (14.21)	37 (14.05)	21 (14.49)
Post TB bronchiectasis	41 (10.53)	31 (12.4)	10 (7.25)
CVA/CVD	20 (4.74)	13 (5.79)	7 (2.9)
CKD	6 (2.63)	0	6 (7.25)
Lung Cancer	6 (2.63)	4 (3.31)	2 (1.45)
Malignancy other than lung	6 (2.63)	3 (2.48)	3 (2.9)
Other comorbidities	9 (4.74)	6 (4.96)	3 (4.35)

VAP = Ventilator-Associated Pneumonia, COPD = Chronic Obstructive Pulmonary Disease, CVA/CVD = Cerebrovascular Accident/Disease, CAD = Coronary Artery Disease, CKD = Chronic Kidney Disease, Other comorbidities = include Neuromuscular dystrophy, Cor pulmonale, Polycythemia, Kyphoscoliosis, Thymoma, Parkinson's Disease

Apply the VAP bundle to all patients who are intubated or on mechanical ventilation, unless specific contraindications are present. The following bundle elements must be consistently implemented:



HEAD OF BED ELEVATION (>30 DEGREES)

2 9-10-2-30° **Action:** Keep the head of the bed elevated between 30–45 degrees, unless contraindicated. **Rationale:** Prevents aspiration of secretions and gastric contents.

ORAL CARE (EVERY 6 HOURS)

3 ORAL CARE **Action:** Perform oral care using toothbrushing and sterile water or appropriate antiseptic every six hours. **Rationale:** Reduces oral bacterial colonization and microaspiration risk.

Note: Routine use of chlorhexidine is **optional** and should be guided by physician orders and patient risk factors.

CUFF PRESSURE MONITORING

6 CUFF PRESSURE **Action:** Check endotracheal tube cuff pressure each shift and after patient repositioning or transport. Target pressure: 20–30 cm H₂O. **Rationale:** Ensures a proper seal to minimize aspiration.

DEEP VEIN THROMBOSIS (DVT) PROPHYLAXIS

Action: Ensure DVT prophylaxis is in place. **Rationale:** Reduces the risk of thromboembolic events in immobilized patients.

PEPTIC ULCER DISEASE (PUD) PROPHYLAXIS

Action: Confirm that PUD prophylaxis is ordered and administered. **Rationale:** Prevents stress ulcers and related complications.

DAILY SEDATION VACATION

8 WEANING **Action:** Conduct daily sedation interruption per physician order. **Rationale:** Assesses neurological status and readiness for ventilator weaning.

DAILY ASSESSMENT OF READINESS TO EXTUBATE

Action: Evaluate daily if the patient is ready for ventilator weaning and extubation. **Rationale:** Reduces mechanical ventilation time, decreasing VAP risk.

DON'T FORGET

CLEAN HANDS SAVE LIVES!

Follow WHO's **5 Moments for Hand Hygiene** to prevent infection.



Patient-Related Risk Factors for VAP

Among the comorbidities, CKD significantly reduced the odds of developing VAP by approximately 89% (Table 2). In contrast, specific reasons for intubation were strong

predictors of VAP. Intubation due to CAP-HR more than doubled the risk of VAP, while HAP nearly quadrupled the odds compared to patients intubated for other reasons.

Table 2. Patient-Related Factors and Risk of VAP Development

	Coefficient	SE Coefficient	P-Value	Odds Ratio (OR)	95% CI for OR
Comorbidities					
Asthma	-0.26	0.37	0.48	0.77	(0.37, 1.59)
CAD	0.556	0.66	0.40	1.74	(0.47, 6.35)
CKD	-2.24	1.01	0.03*	0.11	(0.01, 0.77)
COPD	-0.10	0.41	0.81	0.90	(0.40, 2.03)
Cor Pulmonale	-15	325	0.96	0.00	(0.00, 6.01E+27)
CVA/CVD	0.28	0.64	0.66	1.32	(0.38, 4.67)
Diabetes Mellitus Type 2	0.30	0.35	0.39	1.35	(0.68, 2.70)
Hypertension	-0.01	0.29	0.98	0.99	(0.56, 1.74)
Lung Cancer	1.60	0.91	0.08	4.95	(0.83, 29.55)
Malignancy other than lung	-0.17	0.87	0.84	0.84	(0.15, 4.67)
Neuromuscular dystrophy	11.00	325	0.98	58384.71	(0.00, 1.55E+28)
Post TB bronchiectasis	0.34	0.67	0.62	1.40	(0.38, 5.25)
Others	1.64	1.19	0.17	5.18	(0.50, 53.72)
Reason for Intubation					
ACS/NSTEMI/STEMI	-0.19	0.39	0.64	0.83	(0.38, 1.80)
BAIAE	-0.33	1.57	0.83	0.72	(0.03, 15.66)
Central airway obstruction	12	325	0.971	118488.19	(0, 3.15E+28)
CAP HR	0.98	0.39	0.011 *	2.67	(1.25, 5.71)
COPDIAE	-0.04	0.44	0.93	0.96	(0.40, 2.29)
Cor Pulmonale	-1.04	0.69	0.13	0.35	(0.09, 1.36)
COVID 19	-0.22	0.66	0.74	0.81	(0.22, 2.93)
CVA/CVD	0	0.68	0.99	0.99	(0.26, 3.79)
Decompression sickness	12	325	0.97	143771.60	(0, 3.81E+28)
DRTB	1.25	1.16	0.28	3.50	(0.36, 33.82)
HAP	1.35	0.66	0.042*	3.84	(1.05, 14.05)
Lung Cancer	-0.59	0.65	0.36	0.55	(0.15, 1.98)
Malignancy other than lung	0.65	0.76	0.40	1.91	(0.43, 8.52)
Massive Hemoptysis	1.72	1.53	0.26	5.56	(0.28, 110.94)
Myasthenia gravis	-1	354	1	0.31	(0, 5.12E+30)
Pericardial effusion	-1.24	1.49	0.41	0.29	(0.02, 5.42)
Pleural Effusion	1.42	1.54	0.36	4.16	(0.20, 85.78)
Pneumohydrothorax	-12	325	0.97	0.00	(0, 1.66E+27)
Pneumothorax	0.57	1.63	0.73	1.78	(0.07, 43.74)
Post TB bronchiectasis IE	-0.61	0.58	0.30	0.54	(0.17, 1.71)
PTB	-0.91	0.72	0.21	0.40	(0.10, 1.66)
Pulmonary Congestion	0.06	1.42	0.97	1.06	(0.06, 17.26)
Pulmonary Embolism	-14.00	325	0.97	0	(0, 2.65E+27)
Sepsis/Septic Shock	0.36	0.39	0.36	1.43	(0.66, 3.06)

Note: *Significant at $p < 0.05$ using Binary Logistic Regression Analysis; Deviance R-Sq = 14.59%; Deviance R-Sq(adj) = 2.23%
VAP = Ventilator-Associated Pneumonia, CKD = Chronic Kidney Disease, CAP-HR = Community-Acquired Pneumonia High Risk, HAP = Hospital-Acquired Pneumonia, ACS = Acute Coronary Syndrome, COPDIAE = chronic obstructive pulmonary disease in acute exacerbation, NSTEMI = non-ST-segment elevation myocardial infarction, STEMI = ST-segment elevation myocardial infarction.

Microbiological Profile of Endotracheal Aspirates

Analysis of the 412 endotracheal aspirates revealed a predominantly gram-negative microbial landscape (Table 3). *Acinetobacter baumannii* was the most frequently isolated organism overall (12.63%), maintaining a consistent presence in both the VAP (12.40%) and non-VAP (13.04%) groups. This was followed by *Candida* species (11.05%) and *Pseudomonas aeruginosa* (10.53%). A

distinct divergence in pathogen distribution was observed between the two cohorts. Specifically, *Streptococcus pneumoniae* was notably more prevalent among patients who did not develop VAP (10.14%) compared to those who did (2.48%). Conversely, opportunistic nosocomial pathogens, including *Acinetobacter lwoffii* (6.61%) and *Enterococcus* species (4.13%), were isolated exclusively in the VAP cohort and were absent in the non-VAP group.

Table 3. Microbiological Profile of Endotracheal Aspirates

	Total (n = 412)	With VAP (n = 269)	Without VAP (n = 143)
Frequency (%); Mean + SD			
ETA organism isolated			
<i>Acinetobacter baumannii</i>	49 (12.63)	32 (12.4)	17 (13.04)
<i>Candida</i> sp	45 (11.05)	29 (11.57)	16 (10.14)
<i>Pseudomonas aeruginosa</i>	41 (10.53)	24 (9.92)	17 (11.59)
<i>Klebsiella pneumoniae</i>	24 (6.84)	13 (4.96)	11 (10.14)
<i>Streptococcus viridans</i>	24 (6.32)	15 (5.79)	9 (7.25)
<i>Streptococcus pneumoniae</i>	20 (5.26)	5 (2.48)	15 (10.14)
<i>Acinetobacter lwoffii</i>	16 (4.21)	16 (6.61)	0
Coagulase Negative <i>Staphylococcus</i>	16 (4.21)	10 (4.13)	6 (4.35)
<i>Escherichia coli</i>	12 (3.16)	10 (4.13)	2 (1.45)
<i>Enterococcus</i>	10 (2.63)	10 (4.13)	0
<i>Enterobacter cloacae</i>	8 (2.11)	5 (2.48)	3 (1.45)
<i>Staphylococcus aureus</i>	8 (2.11)	4 (1.65)	4 (2.9)
<i>Stenotrophomonas maltophilia</i>	8 (2.11)	5 (2.48)	3 (1.45)
<i>Sphingomonas paucimobilis</i>	8 (2.11)	4 (1.65)	4 (2.9)
<i>Serratia marcescens</i>	6 (1.58)	4 (1.65)	2 (1.45)
<i>Burkholderia cepacia</i>	6 (1.58)	4 (1.65)	2 (1.45)
<i>Enterococcus faecalis</i>	6 (1.58)	4 (1.65)	2 (1.45)
<i>Klebsiella oxytoca</i>	6 (1.58)	6 (2.48)	0
<i>Elizabethkingia Meningoseptica</i>	6 (1.05)	3 (0.83)	3 (1.45)
<i>Staphylococcus warneri</i>	6 (1.05)	3 (0.83)	3 (1.45)
<i>Pseudomonas fluorescens</i>	6 (1.05)	3 (0.83)	3 (1.45)
<i>Moraxella</i> sp	6 (1.05)	6 (1.65)	0
<i>Staphylococcus saprophyticus</i>	2 (0.53)	2 (0.83)	0
<i>Pseudomonas luteola</i>	2 (0.53)	2 (0.83)	0
<i>Streptococcus sanguinis</i>	2 (0.53)	2 (0.83)	0
<i>Pseudomonas stutzeri</i>	2 (0.53)	2 (0.83)	0

Treatment and Environmental Risk Factors

Tracheostomy was the strongest independent risk factor, increasing VAP risk nearly sevenfold (Table 4). Similarly, prolonged mechanical ventilation (>4 days) increased the likelihood of VAP nearly tenfold. The use of multiple antibiotics during hospitalization quadrupled the odds of developing VAP, whereas the use of empiric antibiotic

therapy was associated with a 63% reduction in risk compared to targeted therapy. Additionally, every day of hospital admission before intubation increased the odds of VAP by 10%. Conversely, neither the specific ward of admission nor individual VAP bundle components ordered at intubation significantly influenced VAP development.

Table 4. Treatment and Environmental Factors and VAP Risk

	Coefficient	SE Coefficient	P-Value	Odds Ratio (OR)	95% CI for OR
Treatment-Related Factors					
Use of Bundle of Care	0.24	0.22	0.29	1.27	(0.82, 1.97)
Head of Bed Elevation (30°–45°)					
Oral Care	0.18	0.22	0.42	1.20	(0.77, 1.85)
Proton Pump Inhibitor (PPI) Use	0.48	0.35	0.17	1.62	(0.81, 3.22)
Suctioning	0.56	0.3	0.07	1.75	(0.96, 3.17)
Duration of Mechanical Ventilator Use					
Intubation > 4 days	—	—	< 0.01*	9.85	—
MDRO Risk Factors					
Multiple Antibiotic Use	1.48	0.26	<0.001*	4.40	(2.64, 7.34)
Empiric Antibiotic Therapy	-0.98	0.21	<0.001*	0.37	(0.25, 0.57)
Chemotherapy/Radiotherapy	0.67	0.84	0.42	1.96	(0.38, 10.08)
Current Hospitalization > 5 days	0.43	0.4	0.28	1.54	(0.70, 3.40)
Dialysis Use	0.04	0.31	0.89	1.04	(0.57, 1.91)
Immunocompromised State	0.61	0.52	0.24	1.83	(0.66, 5.05)
Previous Antibiotic Use	0.03	0.32	0.92	1.03	(0.55, 1.94)
Tracheostomy	1.93	0.53	<0.001*	6.86	(2.41, 19.52)
Environment-Related Factors					
Ward of Admission					
MICU / RICU / SICU / 3B / STU	—	—	>0.05	NS	NS
Duration of Admission					
Duration before VAP	0.09	0.01	<0.001*	1.1	(1.07, 1.13)

*Significant at $p < 0.05$

Factors Associated with Mortality

Among patient-related factors, the reason for intubation was a critical predictor for mortality (Table 5). Patients intubated due to Sepsis or Septic Shock had nearly a 12-fold increase in mortality odds, while those with Lung Cancer had a fivefold increase. Acute Coronary Syndrome (ACS) also significantly tripled the risk of death. Conversely, intubation due to COPD exacerbation was associated with an 80% reduction in mortality risk compared to other causes.

Regarding treatment-related factors, interventions intended for VAP prevention were associated with higher mortality in this cohort. Orders for suctioning and oral care were associated with a twofold increase in mortality risk. Additionally, chronic dialysis was a strong predictor, increasing mortality odds by nearly five times. Interestingly, a current hospitalization duration of more than five days before VAP onset was associated with a 68% reduction in mortality, possibly reflecting a survival bias where early mortality cases were excluded. Environmental factors, including the specific ward of admission, did not significantly impact mortality outcomes.

Table 5. Factors Associated with Mortality

	Coefficient	SE Coefficient	P-Value	Odds Ratio (OR)	95% CI for OR
I. Patient-Related Factors					
Demographics					
Age	0.01	0.01	0.37	1.01	(0.99, 1.03)
Sex (Female vs Male)	-0.47	0.39	0.23	0.63	(0.29, 1.34)
Smoking (Non-Smoker)	-0.08	0.43	0.85	0.92	(0.39, 2.16)
Comorbid Conditions					
COPD	-1.02	0.54	0.06	0.36	(0.13, 1.04)
Lung Cancer	-0.79	0.87	0.37	0.45	(0.08, 2.52)
Diabetes Mellitus	0.2	0.41	0.62	1.22	(0.55, 2.70)
Hypertension	-0.05	0.34	0.88	0.95	(0.48, 1.85)
Asthma	-0.45	0.47	0.34	0.64	(0.25, 1.61)
Reason for Intubation					
Sepsis / Septic Shock	2.47	0.54	<0.001*	11.77	(4.06, 34.11)
Lung Cancer	1.63	0.7	0.02*	5.12	(1.31, 19.99)
ACS / NSTEMI / STEMI	1.22	0.45	<0.01*	3.37	(1.41, 8.06)
COPD Exacerbation	-1.6	0.78	0.04*	0.2	(0.04, 0.93)
CVA / CVD	1.43	0.83	0.08	4.19	(0.83, 21.19)
COVID-19	0.66	0.78	0.4	1.93	(0.42, 8.83)
CAP-HR	-0.01	0.44	0.97	0.99	(0.42, 2.33)
HAP	-0.09	0.77	0.91	0.91	(0.20, 4.14)
II. Treatment-Related Factors					
VAP Bundle Orders					
Suctioning	0.78	0.35	0.03*	2.18	(1.10, 4.34)
Oral Care	0.66	0.23	<0.01*	1.93	(1.23, 3.02)
PPI Use	-0.25	0.37	0.49	0.78	(0.38, 1.59)
Head of Bed Elevation	-0.32	0.22	0.16	0.73	(0.47, 1.13)
Interventions & MDRO Risks					
Dialysis Use	1.57	0.3	<0.001*	4.78	(2.63, 8.68)
Current Hosp > 5 Days	-1.14	0.43	<0.01*	0.32	(0.14, 0.74)
Immunocompromised State	0.86	0.46	0.06	2.37	(0.96, 5.86)
Immunosuppressive Drug	0.5	0.29	0.09	1.65	(0.93, 2.92)
Multiple Antibiotic Use	0.13	0.28	0.66	1.13	(0.65, 1.98)
Empiric Antibiotics	0.01	0.2	0.97	1.01	(0.68, 1.50)
Intubation > 4 Days	-0.29	0.26	0.27	0.75	(0.45, 1.26)
III. Environment-Related Factors					
Ward of Admission					
MICU (Reference)	—	—	—	1	—
RICU	-0.68	1.12	0.54	0.51	(0.06, 4.54)
SICU	0.45	0.89	0.61	1.57	(0.27, 8.99)
Ward 3B	-12.5	450	0.98	0	(0.00, >100)
STU (Special Treatment Unit)	13.5	215	0.95	>100	(0.00, >100)

*Significant at $p < 0.05$

Note: Coefficients, Standard Errors (SE), and Odds Ratios (OR) are rounded to 2 decimal places.

Factors Associated with Length of Hospital Stay

Among the reasons for intubation, Hospital-Acquired Pneumonia (HAP) and Cor Pulmonale were significant predictors of prolonged stay, showing substantial

positive coefficients. Environmental factors also played a role; notably, late-onset VAP and a longer duration of admission before VAP diagnosis were strongly associated with extended hospital stays. Treatment-related factors

such as tracheostomy and multiple antibiotic use were also linked to longer admissions. Conversely, intubation

due to lung cancer or CKD did not significantly extend the length of stay in this cohort.

Table 6. Factors Associated with Duration of Hospitalization

	Coefficient	SE Coefficient	P-Value
I. Patient-Related Factors			
<i>Demographics</i>			
Age	0.03	0.07	0.63
Sex (Female)	2.23	2.5	0.37
Smoking (Non-Smoker)	-1.03	2.82	0.72
Previous Smoker	-1.72	2.65	0.52
<i>Comorbid Conditions</i>			
Asthma	4.18	2.9	0.15
CAD	-2.93	5.12	0.57
CKD	-6.26	7.46	0.4
COPD	-1.37	3.3	0.68
Cor Pulmonale	-7.2	19.7	0.72
CVA/CVD	-3.38	4.87	0.49
Diabetes Mellitus	-0.1	2.68	0.97
Hypertension	1.04	2.24	0.64
Lung Cancer	11.43	6.59	0.08
<i>Reason for Intubation</i>			
HAP	13.17	5.07	0.01*
Cor Pulmonale	12.45	5.47	0.02*
Sepsis/Septic Shock	5.16	3.03	0.09
Lung Cancer	-7.72	5.23	0.14
COVID-19	-5.99	5.15	0.25
CAO (Central Airway Obstruction)	-21.3	19.2	0.27
Pulmonary Congestion	-12	11	0.28
CAD	6.22	6.05	0.31
Pulmonary Embolism	-18.5	19.5	0.34
Pericardial Effusion	-10.8	11.9	0.37
Massive Hemoptysis	6.74	8.65	0.44
Obstructive Pneumonia	4.82	7.62	0.53
COPD Exacerbation	-1.6	3.49	0.65
Pneumothorax	6	13.3	0.65
ACS/NSTEMI/STEMI	-0.81	3.07	0.79
CAP-HR	0.18	2.8	0.95
II. Treatment-Related Factors			
<i>Interventions & MDRO Risks</i>			
Tracheostomy	–	–	<0.001*
Multiple Antibiotic Use	–	–	<0.001*
Dialysis Use	–	–	<0.001*
Late-Onset VAP	–	–	<0.001*
Duration of Admission	1.1	–	<0.001*
III. Environmental-Related Factors			
<i>Ward of Admission (Ref: MICU)</i>			
RICU	–	–	>0.05
SICU	–	–	>0.05
Ward 3B	–	–	>0.05
STU (Special Treatment Unit)	–	–	>0.05

*Significant at $p < 0.05$

DISCUSSION

This study at our institution identifies key risk factors for the development of VAP in critical care units. In this study, baseline demographic characteristics such as age, sex, smoking history, and general systemic comorbidities were not significantly associated with the development of VAP. While ARF and ARDS were highly prevalent among elderly males with comorbid conditions, these clinical profiles were equally distributed between the VAP and non-VAP groups and did not correlate with increased mortality or prolonged hospital length of stay. While these findings challenge several historically established cohorts that identified advanced age and systemic comorbidities as primary drivers of VAP,^{9,10} they strongly align with a growing body of contemporary literature. Recent investigations have similarly demonstrated that baseline demographics are frequently overshadowed by device-related variables and the acute severity of the underlying illness. This paradigm shift suggests that in modern critical care settings, a patient's susceptibility to VAP is driven less by their intrinsic baseline characteristics and more by the profound environmental disruption and duration of invasive mechanical ventilation itself.

Notably, CKD was associated with a reduced incidence of VAP. This inverse relationship may reflect clinical management differences and selection bias rather than true physiological protection. CKD patients often require aggressive fluid restriction and closer hemodynamic monitoring; literature confirms that such conservative fluid strategies promote negative fluid balance, shorter ventilator days, and earlier extubation, thereby narrowing the risk window for VAP.¹¹ However, when renal impairment progressed to require dialysis, this intervention was associated with both VAP development and mortality, likely reflecting the profound vulnerability, immune dysregulation, and overall severity of illness in patients requiring invasive organ support. Furthermore, this apparent protection may be compounded by selection bias, such as the earlier transfer of these complex patients, and the routine administration of broad-spectrum, renal-adjusted antibiotic regimens that incidentally suppress early airway colonization.

The microbiological profile of endotracheal aspirates underscores a distinct ecological shift in the airways of intubated patients. The dominance of gram-negative organisms, specifically *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae*, aligns with the known pathogenesis of VAP. These resilient pathogens rapidly form protective biofilms on endotracheal tubes, creating a persistent reservoir for micro-aspiration and subsequent infection.¹² Comparing the VAP and non-VAP cohorts clarifies the crucial distinction between airway colonization and nosocomial superinfection. The higher prevalence of *Streptococcus pneumoniae* in the non-VAP group likely reflects residual colonization from their initial community-acquired pneumonia (CAP) after

targeted therapy. Conversely, the exclusive isolation of *Acinetobacter lwoffii* and *Enterococcus* species in the VAP cohort highlights their role as true opportunistic, device-associated pathogens that emerge under the selective pressure of broad-spectrum empiric antibiotics.¹³ Finally, the prominent isolation of *Candida* species (11.05% overall) and its independent association with mortality warrants attention. Often dismissed as benign airway colonization, its heavy presence is clinically significant. Extensive *Candida* colonization serves as a surrogate marker for profound host immunosuppression, prolonged ICU stays, and fungal overgrowth following heavy antibiotic exposure.¹⁴ Furthermore, dense *Candida* colonization can promote the growth and virulence of co-existing bacteria like *Pseudomonas aeruginosa*, complicating clinical recovery and driving up mortality.

Conversely, admission diagnoses of high-risk community-acquired pneumonia and hospital-acquired pneumonia significantly increased the odds of developing ventilator-associated pneumonia. This predisposition is likely driven by a combination of pre-existing lung inflammation, prior heavy antibiotic exposure, and the subsequent disruption of normal respiratory flora. The initial pneumonic insult causes diffuse epithelial damage and impairs ciliary clearance.¹⁵ When combined with an endotracheal tube, this compromised microenvironment facilitates a clear pathogenic sequence. Specifically, the pre-existing pneumonia necessitates prolonged mechanical ventilation, which in turn provides an extended opportunity for bacterial colonization and biofilm formation, ultimately resulting in the development of ventilator-associated pneumonia.¹²

Therapeutic interventions and device utilization heavily influenced both the development of ventilator-associated pneumonia and patient mortality. The duration of mechanical ventilation directly correlated with disease incidence and extended hospital stays, reinforcing the fundamental critical care principle that limiting intubation time is paramount. Similarly, tracheostomy was significantly associated with both the development of the infection and mortality. However, our retrospective analysis did not stratify tracheostomy by timing, such as early versus late intervention. This is a critical distinction, as literature demonstrates that delayed tracheostomy prolongs endotracheal intubation and increases colonization risk. Early tracheostomy, typically performed within the first seven to ten days, has been shown to reduce overall disease incidence and facilitate faster weaning.^{16,17}

Antimicrobial strategies also yielded complex outcomes. While multiple antibiotic use was strongly tied to the development of ventilator-associated pneumonia and mortality, an observed inverse association between empiric antibiotic therapy and disease development requires careful contextualization. This apparent protective effect may stem from early broad-spectrum

coverage effectively suppressing early-onset pathogens. Alternatively, it may reflect confounding by indication, wherein patients presenting with profound sepsis receive immediate empiric therapy, and their clinical outcomes, including mortality, are determined rapidly by their primary illness before the pneumonia has the opportunity to develop.¹⁸ Consequently, critical care practitioners must continuously balance the immediate life-saving benefits of early empiric therapy against the vital principles of antimicrobial stewardship to prevent the subsequent selection of multidrug-resistant organisms.

Analysis of prevention bundles revealed inconsistent implementation across the institution. Individual components, such as proton pump inhibitors, head of bed elevation, and venous thromboembolism prophylaxis, did not significantly alter the risk of developing ventilator-associated pneumonia. Strikingly, orders for oral care and secretion suctioning were associated with increased mortality. This association likely reflects documentation and selection bias; these specific orders are frequently documented more rigorously in the most critically ill patients who inherently carry a higher mortality risk, rather than suggesting a direct causal harm from oral hygiene. Environment-related factors, specifically the specific ward or intensive care unit of admission, showed no significant association with VAP development. This indicates a uniform standard of critical care delivery and baseline infection control practices for mechanically ventilated patients across the hospital's various departments.

LIMITATIONS

Several limitations must be considered when interpreting these findings. First, the study is constrained by its retrospective design, which is inherently vulnerable to missing documentation, unstratified variables (such as the exact timing of tracheostomies), and difficulties in verifying non-standardized bundle adherence. Second, there is a potential for misclassification of VAP due to diagnostic heterogeneity, as clinical and radiographic criteria frequently overlap with other underlying pulmonary

pathologies. Finally, the single-center design limits the broad generalizability of our results, as local microbial ecologies and institutional protocols heavily dictate VAP dynamics.

RECOMMENDATIONS

Given that baseline diagnoses of CAP-HR and HAP directly increased the odds of developing VAP, we recommend enhanced surveillance and targeted prevention strategies for these specific patient populations upon admission. Additionally, future prospective studies should investigate the potentially protective patient-level clinical mechanisms, such as conservative fluid management, observed in those with CKD. Regarding treatment-related factors, the significant associations of prolonged mechanical ventilation, tracheostomy, and multiple antibiotic exposure with both VAP development and subsequent mortality highlight the need for the institutional implementation of protocolized weaning strategies, early tracheostomy evaluations, and stringent antimicrobial stewardship programs. Furthermore, because our institution lacked a formalized VAP bundle checklist during the study period, relying instead on individual orders, and because specific interventions like oral care were unexpectedly associated with higher mortality, we recommend that healthcare facilities transition to unified, protocolized VAP prevention bundles. Regular audits of these formalized bundles are highly encouraged to evaluate true bedside implementation fidelity, while future research should explore optimal antibiotic regimens and durations to minimize multidrug-resistant organism development. Finally, to address environmental and institutional variables, healthcare facilities should consider standardizing VAP diagnostic criteria across all critical care units to ensure consistent reporting and infection control. To comprehensively advance this field, future multi-center, prospective studies with larger sample sizes and longer observational periods are essential to mitigate the inherent biases of this single-center retrospective design and validate these findings across diverse clinical settings.

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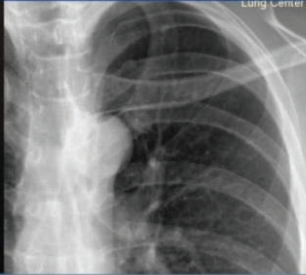
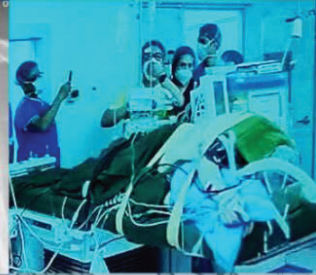
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