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The ongoing challenge of ventilator-associated pneumonia: epidemiology, prevention, and risk factors for mortality in a secondary care hospital intensive care unit

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SUMMARY

Background: Ventilator-associated pneumonia (VAP) is a leading cause of morbidity and mortality among intensive care unit infections. Despite various preventive measures, the incidence of VAP remains high.

Aims: This study aimed to explore the epidemiology and risk factors for VAP associated mortality in a secondary care hospital, comparing outcomes before and after implementing a VAP prevention bundle.

Methods: This retrospective study was conducted from July 1, 2021, to June 30, 2023, at a secondary care hospital. Patients over 18 years old who underwent mechanical ventilation for more than 48 hours were included. The study compared the incidence, microbiological etiology, and outcomes of VAP before and after implementing the VAP prevention bundle and analyzed risk factors for mortality from VAP.

Results: A total of 83 patients diagnosed with VAP were included. Despite concerted efforts to implement the VAP prevention bundle, there was no significant decrease in the VAP rate per 1000 ventilator days, early-onset VAP, secondary bloodstream infections, acute respiratory distress syndrome, and 30-day mortality. The microbiological etiology of VAP remained consistent between the two periods. A decrease in lymphocyte count and albumin level were identified as independent risk factors for 30-day mortality.

Conclusions: Concerted efforts to implement a VAP prevention bundle did not significantly reduce the incidence or improve outcomes of VAP in this secondary care hospital setting. The microbiological etiology remained unchanged. Monitoring lymphocyte count and albumin level may help identify patients at high mortality risk. Further research is needed to develop more effective VAP prevention and management strategies.

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Introduction

Nosocomial infections occurring in intensive care units (ICUs) cause higher economic costs and increased mortality, prolonged hospital stay, the need for multiple antibiotic use, and the proliferation of resistant microorganisms [1]. Invasive mechanical ventilation (IMV) is ICUs' most commonly used respiratory support system. The risk of many nosocomial infection, especially ventilator-associated pneumonia (VAP), increases with IMV support. VAP is a subset of pneumonia that develops in the ICU and is defined as an infection of the lung parenchyma in patients who have been receiving invasive mechanical ventilation for at least 48 hours [2]. In the literature, the incidence of VAP ranges from 8.0% to 28.8% of the population at risk, with event rates ranging from 1.4 to 16.5 per 1000 ventilation days [3,4]. This wide range is due to different hospital and country conditions, VAP diagnosis and prevention methods, and diverse patient populations. *Staphylococcus aureus, Pseudomonas aeruginosa,* Candida species, *Klebsiella oxytoca* and *pneumoniae,* Streptococcus species, and Enterobacter species are among the pathogens frequently linked to VAP [5,6]. Host susceptibility is determined by local factors, such as underlying lung disease, or systemic factors, such as immunosuppression, neutropenia, age greater than 70, dysphagia, and recent abdominal or thoracic surgery [7]. Despite implementing various preventive measures, the incidence of VAP remains high. In some studies examining the risk factors associated with mortality in patients with VAP, underlying severe disease, high APACHE II scores, underlying chronic obstructive pulmonary disease (COPD), and infection with multidrug-resistant pathogens such as *Pseudomonas aeruginosa* and methicillin-resistant *Staphylococcus aureus* (MRSA) were frequently shown among the risk factors causing high mortality [8,9]. Early identification and understanding of the etiology of VAP in the ICU is essential to prevent mortality and morbidity. In this context, this article aims to explore the epidemiology and risk factors for mortality from VAP in a secondary care hospital. We will compare the situation before and after implementing a VAP prevention bundle, aiming to provide insights into the effectiveness of such interventions in reducing VAP incidence and improving patient outcomes. Through this analysis, we hope to contribute to the ongoing efforts to mitigate the burden of VAP in ICUs and enhance the quality of patient care.

Methods

This retrospective observational study was conducted in our adult medical-surgical ICU from July 1, 2021, to June 30, 2023. This study has been approved by the University of Health Sciences Bursa Yuksek Ihtisas Training and Research Hospital ethical committee with the following approval number and date: 2011-KAEK-25/06.01.2023.

This study enrolled adult participants over 18 years old who had undergone mechanical ventilation for longer than 48 hours. When analyzing the primary outcome, only the first case of bacterial VAP for each patient was included. VAP was defined as pneumonia that developed more than 48 hoursafter patients were intubated and received mechanical ventilation. For diagnosing VAP, the following criteria were used; new or deteriorating infiltrates on a chest radiograph, with at least two of the following criteria: (i) body temperature exceeding 38°C; (ii) leukocyte count surpassing 10,000/mm^3 or dropping below 4,000/mm3; (iii) purulent secretions from the bronchi (over 25 leukocytes) and 10 or fewer epithelial cells per gram stain of a deep endotracheal aspirate specimen; and (iv) decreased oxygenation in the blood [10,11]. VAP occurring within the first four days of mechanical ventilation was referred to as early-onset VAP, while VAP occurring on the fifth or later day of mechanical ventilation was referred to as late-onset VAP [3].

Sepsis was characterized by signs of infection along with systemic inflammation, which was indicated by at least two of the following: a heart rate over 90 beats per minute, respiratory rate more than 20 times per minute or low blood carbon dioxide levels, an abnormally high or low white blood cell count or normal count with more than 10% immature forms, and a body temperature above 38°C or below 36°C [12]. Septic shock referred to sepsis that resulted in persistent low blood pressure despite adequate fluid intake, as well as impaired tissue perfusion or organ dysfunction [12].

Microbiological samples were obtained from patients with suspected VAP or bloodstream infection (BSI). For suspected VAP, endotracheal aspirate samples were collected by passing a sterile suction catheter through the endotracheal tube and performing sterile saline lavage to obtain the sample. Quantitative cultures were done by plating serial dilutions on agar plates to determine bacterial concentration, with colonies counted after 24 hours of incubation. For suspected BSI, blood cultures were obtained by drawing blood from two separate peripheral venous sites and inoculating 10 mL into bottles. Bottles were incubated for 5 days or until positive, with isolates identified using standard laboratory techniques.

The Sequential Organ Failure Assessment (SOFA) score was calculated as described by Vincent *et al*. to assess degree of organ function or rate of failure during a patient's stay in an intensive care unit [13].

The APACHE II score was calculated within 24 hours after a patient entered the ICU. Similarly, the SOFA score was calculated within 24 hours of ICU admission, and a subsequent SOFA score was calculated on the day VAP was diagnosed.

The VAP prevention bundle applied in our hospital is the T.C. Ministry of Health national prevention bundle for healthcare-associated infections ventilator-associated pneumonia prevention bundle; ensuring hand hygiene in all interventions to be performed on the patient, daily assessment of endotracheal intubation and mechanical ventilation necessity, administration of a sedation "holiday", keping the head of the bed at 30—45 degrees and daily oral care with sterile water.

Before implementing the VAP prevention bundle, intensive care unit doctors and nurses received a oneweek training package consisting of power-point presentations and quizzes. The VAP prevention bundle, which was applied by ICU physicians and ICU nurses, was documented on the follow-up form. Compliance with the bundles was monitored daily by the nurse in charge of the intensive care unit with a checklist and these lists were checked monthly by the infection control nurse. Demographic, clinical, and biological findings on admission were obtained from the patient's medical charts and reviewed retrospectively.

Statistical analysis

The data underwent analysis through the SPSS software, version 28. To provide an overview of the data, descriptive statistics were employed. Qualitative data points were presented in percentage form, while quantitative ones were depicted as either mean values with standard deviations or

median values. The normality of continuous variables was verified using the Kolmogorov–Smirnov test. To discern differences among groups, the Chi-square or Fisher's exact test was utilized for categorical data, and depending on suitability, the t-test or Mann–Whitney test was used for continuous data.

Considering the numerous comparisons conducted in this research, a Bonferroni adjustment was incorporated to minimize the potential for Type I errors. To pinpoint independent factors influencing 30-day mortality, a multivariate logistic regression employing a forward stepwise approach was adopted, ensuring that there was no overlap of independent variables. The accuracy of the logistic regression model was gauged through the Hosmer-Lemeshow test for goodness-of-fit. All statistical evaluations were two-tailed, and a significance threshold was established at $P < 0.05$, considering multiple comparisons. Essential results were accompanied by their effect sizes, presenting odds ratios along with 95% confidence intervals.

Results

A total of 83 patients who developed VAP were included in the analysis. The mean age was 69.4 ± 6.9 years and 48.2% were female. The mean APACHE II score on ICU admission was 17.8 ± 3.0 and the mean SOFA score was 4.3 ± 1.6. The mean SOFA score on the day of VAP diagnosis was 5.9 ± 1.1. Early-onset VAP occurred in 22.9%, while 55.4% developed sepsis or septic shock. The mean duration of mechanical ventilation before VAP was 22.2 ± 16.7 days. The 30-day mortality rate was 59.0% (Table I).

When the pre-bundle and active-bundle periods were compared, the ventilator utilization rate and mean duration of mechanical ventilation before VAP were similar in both periods. The VAP incidence rate was 19.2% during the pre-bundle period compared to 17.5% during the active-bundle period. The VAP rate per 1000 ventilator days was 4.9 in the pre-bundle period compared to 4.0 in the active-bundle period (P=0.309). Sepsis/septic shock rates were slightly higher in the active-bundle period but the differences were not significant. The incidence of secondary BSIs and acute respiratory disctress syndrome (ARDS) were also similar in both periods. The distribution of VAP pathogens was similar for both periods. The mean length of stay was slightly longer in the active-bundle period but the difference was not significant. The 30-day mortality rate was higher in the active-bundle period but again the difference was not significant (Table II).

When VAP patients who survived and died within 30 days were compared, their mean ages were similar. The proportion of females was slightly higher in the death group, but not significantly. APACHE II and SOFA scores were significantly higher in the death group. The incidence of sepsis/septic shock and secondary BSIs were markedly higher in the death group. The duration of mechanical ventilation before VAP was significantly longer in the death group. The incidence of diabetes mellitus was higher in survivors. There were also significant differences in WBC counts, NLR, albumin, GFR and CRP levels. Presence of a central line was more frequent in the death group. *Acinetobacter baumannii* infection was more common in survivors (Table III).

Among the important risk factors for 30-day mortality determined on multivariate analysis, the lymphocyte count and albumin level were independent predictors. Duration of mechanical ventilation before VAP was borderline significant. The other variables were not significant on multivariate analysis (Table IV).

Table I
Patient characteristics

Characteristics	Total (N=83)
Age, years, mean ± SD	69.4 ± 6.9
Female, n (%)	40.0 (48.2%)
APACHE II score on ICU admission, mean ± SD	17.8 ± 3.0
SOFA on admission, mean ± SD	4.3 ± 1.6
SOFA on the day of the diagnosis of VAP, mean ± SD	5.9 ± 1.1
Early-onset VAP, n (%)	19 (22.9%)
Sepsis or septic shock, n (%)	46 (55.4%)
Duration of mechanical ventilation before VAP, day, mean ± SD	22.2 ± 16.7
Previous hospitalization within 3 months, n (%)	49 (59.0%)
Antimicrobial use within 3 months, n (%)	52 (62.7%)
Secondary BSI, n (%)	32 (38.5%)
Diabetes mellitus, n (%)	40 (48.2%)
Cerebrovascular disease, n (%)	39 (47.0%)
Hypertension, n (%)	51 (61.4%)
Chronic obstructive lung diaseses, n (%)	46 (55.4%)
Coronary artery disease, n (%)	39 (47.0%)
Chronic renal disease, n (%)	37 (44.6%)
Malignancy, n (%)	21 (25.3%)
Chronic steroid or immunsuppression therapy, n (%)	23 (27.7%)
ARDS, n (%)	46 (55.4%)
White blood cells (/mm^3), mean ± SD	16784.33 ± 6444.74
Lymphocytes (/mm^3), mean ± SD	1372.28 ± 495.66
Neutrophil-to-lymphocyte ratio, mean ± SD	15.57 ± 11.42
Platelets (/mm^3), mean ± SD	193602.41 ± 29791.93
Albumin, (g/L),mean ± SD	34.51 ± 1.88
Glomerular filtration rate calculated (mL/min/1.73 m^2), mean ± SD	35.21 ± 12.40
C-reactive protein (mg/L), mean ± SD	245.8 ± 123.8
Central-line catheter, n (%)	48 (57.8%)
Blood transfusion, n (%)	30 (36.1%)
Total parenteral nutrition, n (%)	42 (50.6%)
Hemodialysis, n (%)	17 (20.4%)
Escherichia coli, n (%)	35 (42.2%)
Klebsiella pneumoniae, n (%)	11 (13.3%)
Acinetobacter baumannii, n (%)	19 (22.9%)
Pseudomonas aeruginosa, n (%)	10 (12.0%)
Staphylococcus aureus, n (%)	10 (12.0%)
30 day mortality, n (%)	49 (59.0%)

ICU: intensive care unit, VAP: ventilator-associated pneumonia, BSI: bloodstream infection, ARDS: acute respiratory distress syndrome.

Discussion

VAP is a significant cause of morbidity and mortality in ICUs, with a wide range of incidence rates reported in the literature, reflecting the diversity of patient populations and hospital conditions [14,15].

Table II
Comparasion of pre-bundle and active-bundle periods

Characteristics	Pre-bundle period (*N*=46)	Active-bundle period (*N*=37)	*P* value
Age, years, mean ± SD	69.70 ± 7.35	69.24 ± 6.44	0.769
Female, n (%)	27 (58.70%)	13 (35.14%)	0.056
Ventilator utilization rate	78.3%	75.5%	0.071
Duration of mechanical ventilation, day, mean ± SD	47.76 ± 9.52	49.54 ± 10.00	0.408
VAP rate per 1000 ventilator day	4.9%	4.0%	0.309
Early-onset VAP, n (%)	12 (26.09%)	7 (18.92%)	0.610
Sepsis or septic shock, n (%)	24 (52.1%)	22 (59.4%)	0.469
Duration of mechanical ventilation before VAP, day, mean ± SD	27.1 ± 20.2	18.5 ± 15.1	0.537
Secondary BSI, n (%)	20 (43.48%)	22 (59.46%)	0.220
ARDS, n (%)	25 (54.3%)	21 (56.7%)	0.800
Escherichia coli, n (%)	21 (45.65%)	14 (37.84%)	0.622
Klebsiella pneumoniae, n (%)	4 (8.70%)	7 (18.92%)	0.298
Acinetobacter baumannii, n (%)	11 (23.91%)	8 (21.62%)	1.000
Pseudomonas aeruginosa, n (%)	5 (10.87%)	5 (13.51%)	0.977
Staphylococcus aureus, n (%)	6 (13.04%)	4 (10.81%)	1.000
Length of stay, day, mean ± SD	50.9 ± 8.9	53.51 ± 10.3	0.230
30 day mortality, n (%)	24 (52.17%)	25 (67.57%)	0.233

ICU: intensive care unit, VAP: ventilator-associated pneumonia, BSI: bloodstream infection, ARDS: acute respiratory distress syndrome.

This study showed a high burden of VAP in the ICU population studied, with an incidence of 22.9% for early-onset VAP and an overall 30-day mortality rate of 59.0%. These findings are consistent with other recent studies demonstrating VAP continued high morbidity and mortality. A multicenter observational study reported an incidence of early-onset VAP of 27.5% and associated 30-day mortality of 51.5% in a mixed ICU population [16]. Similarly, a meta-analysis found VAP-attributable mortality of 13% based on a systematic review of randomized controlled trials [17].

Sepsis and septic shock were observed in over half of the VAP cases in this study. The development of sepsis is known to increase mortality in VAP by 2—4 times compared to non-septic VAP [18]. Timely recognition and treatment of sepsis are crucial, and several studies have shown that implementing sepsis bundles improves survival in VAP [19]. Ongoing efforts to better prevent, diagnose, and manage sepsis are critical to reducing mortality from VAP.

Implementing a VAP prevention bundle did not significantly reduce the incidence of VAP or improve clinical outcomes in this study. The VAP rate per 1000 ventilator days was slightly lower in the active-bundle period compared to the pre-bundle period (4.0% vs. 4.9%, P=0.309), but this difference was not statistically significant. Similar findings of no significant reduction in VAP rates with prevention bundles have been reported in recent studies. A 2020 systematic review found that VAP bundles did not significantly decrease VAP incidence, length of ICU stay, or mortality [20]. Another meta-analysis in 2023 also found no significant differences in clinical outcomes between bundle and non-bundle groups [21].

The incidence of early-onset VAP was also similar between the two periods in this study (26.09% vs 18.92%, P=0.610). This suggests the VAP prevention bundle did not effectively reduce aspiration and intubation-related risk factors for early-onset VAP. A recent trial found that a multimodal VAP prevention program did not reduce early-onset VAP compared to standard care [22].

Interestingly, the incidence of sepsis/septic shock and secondary bloodstream infections was numerically higher in the active-bundle period compared to the pre-bundle period. However, these differences were not statistically significant. Some studies have reported increased antibiotic resistance with VAP bundles, which could explain higher rates of sepsis and bacteremia [23]. However, further research is needed to clarify these associations.

The distribution of VAP pathogens was similar between the two periods in this study. The most common pathogens were *E.coli, K. pneumoniae, A. baumannii, P.aeruginosa*, and *S. aureus*. This pattern of common VAP pathogens is consistent with recent literature [24]. The VAP bundle did not appear to alter the microbiological epidemiology in this study significantly.

Some potential reasons for the lack of efficacy of the VAP bundle include incomplete compliance, inadequate infection control practices, and antibiotic resistance. High compliance with all bundle elements is essential to reduce VAP effectively [25]. Adherence to hand hygiene, proper cuff pressure maintenance, and oral care protocols should be regularly audited. Prevention of cross-transmission through source control and environmental cleaning is also critical. Local antibiograms should guide appropriate empiric antibiotic selection.

The results of this study show several important differences between VAP patients who survived and those who died within 30 days. The death group had significantly higher severity of illness scores (APACHE II and SOFA) on ICU admission and the day of VAP diagnosis than the survival group. This is consistent with previous studies demonstrating that higher severity of illness is associated with increased mortality in VAP [26,18].

The sepsis/septic shock incidence was also significantly higher in the death group (71.4% vs 32.4%, P<0.001). Sepsis is a

Table III
Comparative Characteristics of VAP Patients: Death vs Survival

Characteristics	Death (*N*=49)	Survival (*N*=34)	*P* value
Age, years, mean ± SD	69.5 ± 7.2	69.4 ± 6.5	0.929
Female, n (%)	25.0 (51.0%)	15.0 (44.1%)	0.656
APACHE II score on ICU admission, mean ± SD	18.9 ± 2.5	16.2 ± 3.1	**< 0.001**
SOFA on admission, mean ± SD	4.8 ± 1.1	3.4 ± 1.7	**< 0.001**
SOFA on the day of the diagnosis of VAP, mean ± SD	6.2 ± 0.8	5.5 ± 1.3	**0.002**
Early-onset VAP, n (%)	8 (16.3%)	11 (32.4%)	0.113
Sepsis or septic shock, n (%)	35 (71.4%)	11 (32.4%)	**< 0.001**
Duration of mechanical ventilation before VAP, day, mean ± SD	25.2 ± 26.2	14.4 ± 12.7	**0.005**
Previous hospitalization within 3 months, n (%)	31 (63.2%)	18 (52.9%)	0.627
Antimicrobial use within 3 months, n (%)	32 (65.3%)	20 (58.8%)	0.646
Secondary BSI, n (%)	22 (44.9%)	10.0 (29.4%)	**0.002**
Diabetes mellitus, n (%)	20 (40.8%)	20 (58.8%)	**0.044**
Cerebrovascular disease, n (%)	25 (51.0%)	14 (41.2%)	0.503
Hypertension, n (%)	30 (61.2%)	21 (61.8%)	1.000
Chronic obstructive lung diaseses, n (%)	31 (63.3%)	15 (44.1%)	0.116
Coronary artery disease, n (%)	22 (44.9%)	17 (50.0%)	0.662
Chronic renal disease, n (%)	24 (48.9%)	13 (38.2%)	1.000
Malignancy, n (%)	12 (24.5%)	9 (26.5%)	0.492
Chronic steroid or immunsuppression therapy, n (%)	15 (30.6%)	8 (23.5%)	1.000
ARDS, n (%)	32 (65.3%)	14 (41.1%)	0.095
White blood cells (/mm^3), mean ± SD	17832.65 ± 6649.16	15273.52 ± 5909.02	0.075
Lymphocytes (/mm^3), mean ± SD	1179.59 ± 390.50	1650.00 ± 504.07	**< 0.001**
Neutrophil-to-lymphocyte ratio, mean ± SD	18.44 ± 12.07	11.42 ± 9.07	**0.005**
Platelets (/mm^3), mean ± SD	190653.06 ± 31478.53	197852.94 ± 27066.84	0.282
Albumin, (g/L),mean ± SD	33.95 ± 1.92	35.32 ± 1.51	**0.001**
Glomerular filtration rate calculated (mL/min/1.73 m^2), mean ± SD	30.49 ± 10.66	42.02 ± 11.66	**< 0.001**
C-reactive protein (mg/L), mean ± SD	290.5 ± 117.5	181.5 ± 103.7	**< 0.001**
Central-line catheter, n (%)	33 (67.3%)	15 (44.1%)	**0.044**
Blood transfusion, n (%)	16 (32.7%)	14 (41.2%)	0.490
Total parenteral nutrition, n (%)	25 (51.0%)	17 (50.0%)	1.000
Hemodialysis, n (%)	9 (18.4%)	8 (23.5%)	0.655
Escherichia coli, n (%)	23 (46.9%)	12 (35.3%)	0.368
Klebsiella pneumoniae, n (%)	7 (14.3%)	4 (11.8%)	1.000
Acinetobacter baumannii, n (%)	7 (14.3%)	12 (35.3%)	**0.034**
Pseudomonas aeruginosa, n (%)	5 (10.2%)	5 (14.7%)	0.733
Staphylococcus aureus, n (%)	8 (16.3%)	2 (5.9%)	0.187

ICU: intensive care unit, VAP: ventilator-associated pneumonia, BSI: bloodstream infection, ARDS: acute respiratory distress syndrome.

known risk factor for mortality in VAP, likely due to its effects on hemodynamic instability and organ dysfunction [27]. The duration of mechanical ventilation before VAP was significantly longer in the death group, which is also a recognized risk factor associated with increased exposure to antibiotics and the selection of drug-resistant pathogens [15].

Several laboratory parameters showed significant differences between the groups. The death group had lower lymphocyte counts, lower albumin levels, and higher C-reactive protein levels. Lymphopenia and hypoalbuminemia have been associated with higher mortality in critically ill patients, reflecting a state of immunosuppression and catabolism [28,29]. Elevated

Table IV
The significant risk factors for 30-day mortality in VAP

Characteristics	Multivariate analyse				
	Death (*N*=49)	Survival (*N*=34)	OR	95% CI	*P* value
Lymphocytes (/mm^3), mean ± SD	1179.59 ± 390.50	1650.00 ± 504.07	0.998	0.996, 1.000	**0.034**
Albumin, (g/L),mean ± SD	33.95 ± 1.92	35.32 ± 1.51	0.688	0.488, 0.971	**0.033**

C-reactive protein levels indicate exaggerated inflammation, which can contribute to multiorgan failure [30]. The death group also had a higher rate of secondary bloodstream infections, which are known to increase mortality risk in VAP [31].

In multivariate analysis, lymphocyte count and albumin level were identified as independent predictors of 30-day mortality. The impact of lymphopenia and hypoalbuminemia highlights the importance of immune dysfunction and nutritional status on outcomes. The borderline significance of pre-VAP ventilation duration reinforces the risks associated with prolonged intubation and mechanical ventilation [32]. White blood cell count, APACHE II score, SOFA score, secondary infections, diabetes mellitus, central line presence, and *A. baumannii* infection were not significant. Overall, these results provide important insights into prognostic factors for mortality in VAP patients. Preventive strategies targeting modifiable risk factors like nutrition and ventilation practices may help improve survival.

There are several limitations to this study that must be considered when evaluating the results. Firstly, this is a single-center retrospective observational study conducted in a secondary care hospital, which may limit the generalizability of our findings to other settings or larger tertiary care hospitals. The specific patient population, hospital practices, and local epidemiology might differ from other institutions, potentially influencing the incidence and outcomes of VAP. Secondly, the study relied on medical chart reviews for data collection, which may introduce potential biases due to incomplete or inaccurate documentation. There might be missing data or unrecorded clinical events that could influence the results. Thirdly, while we compared the pre-bundle and active-bundle periods, other unmeasured confounding factors could have influenced the outcomes, such as changes in ICU staffing, patient case mix, or other hospital-wide interventions during the study period. Furthermore, the study did not evaluate adherence to the VAP prevention bundle in detail. While the bundle implementation was described, the actual compliance with each bundle component was not assessed, which might have influenced its effectiveness.

Conclusions

In conclusion, this study sheds light on the persistent high morbidity and mortality linked to VAP, particularly in vulnerable ICU populations. While the VAP prevention bundle did not significantly improve outcomes compared to standard care, routine auditing of compliance, antimicrobial stewardship, and enhanced infection control practices might bolster the effectiveness of VAP bundles. The findings emphasize the significance of understanding prognostic factors for mortality, such as monitoring lymphocyte count and albumin levels in VAP patients. The VAP incidence remains high despite implementing a prevention bundle in a secondary care hospital. This underscores the urgent need for ongoing research into preventive strategies, improved diagnostic methods, and innovative treatments. Emphasis on modifiable risk factors like nutrition and ventilation practices and attention to the severity of illness, sepsis management, and evidence-based practices will be pivotal in enhancing patient outcomes. Further studies are essential to refine and optimize VAP prevention strategies globally.

Ethical approval

The study was granted ethical approval by the University of Health Sciences Bursa Yuksek İhtisas Training and Research Hospital Ethics Committee (Approval No. 2011- KAEK-25, Dated: 2023/06-01).

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No.

Author contribution

List the contributions of each author below. Example: Zhang San: Conceptualization, Methodology, Software.

Cihan Semet: Conceptualization, Methodology, Data curation, Writing- Original draft preparation, Writing- Reviewing and Editing.

Competing interests

The authors declare no competing interests.

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