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Ventilator Associated Pneumonia in a Medical Intensive Care Unit: Incidence, Risk factors and Mortality at tertiary care centre of eastern Maharashtra

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Abstract

Background: Ventilator Associated Pneumonia (VAP) increases hospital costs, lengthens the hospital length of stay, and significantly contributes to patients discomfort, morbidity, and mortality. The aim was to look at the patient characteristics, microbiological profile and in-hospital mortality in adults receiving mechanical ventilation.

Methodology: In a prospective observational study we enrolled patients aged more than 12 years, with medical-illness and were receiving mechanical ventilation. Patients incubated for >48 hours were analyzed for VAP. Variables were collected, observed and analyzed. Tracheo-bronchial aspiration and microbiological analysis was done. We used Students t test to compare means, Chi-square test for proportions and Mann-Whitney test for medians.

Results: Of the 837 patients who underwent intubation and mechanical ventilation during the study period, 298 patients were ventilated for 48 hours or more. The incidence of VAP was 37.6%. Of the 298 patients, 241 (81% [95% CI 76%- 85%]) died in the hospital. The median length of stay was 6(IQR 4-12) days. The median APACHE II score of those who died was numerically and statistically significantly higher compared to those who survived [16 (IQR 11-20.5) vs. 12 (8-16)]; p=0.001.The most common pathogen associated with VAP was Acinetobacter baumannii. The independent predictors of mortality in mechanically ventilated patients were Age and APACHE II score.

Conclusion: The incidence and overall mortality due to VAP is high. VAP is an important problem in ICU settings and requires prompt care. The most common pathogens causing VAP were Acinetobacter baumannii

and Pseudomonas aeruginosa and were associated with a high fatality rate.

Keywords: Ventilator Associated Pneumonia, Acinetobacter baumannii, Pseudomonas aeruginosa, incidence, risk factor for VAP.

Introduction

Hospital-acquired pneumonia (HAP) is one of the important causes of death amongst patients with hospital acquired infections. Ventilator-associated pneumonia (VAP) has an incidence estimated of 10 to 25 percent and an all-cause mortality of 25 to 50 percent.[1]Early diagnosis of VAP is important because prompt and appropriate treatment can be lifesaving.[2]

About 50 % of the antibiotics administered in ICUs are for treatment of VAP.[3] VAP increases hospital costs, lengthens the hospital length of stay, and significantly contributes to patients discomfort, morbidity, and mortality.[4] As an example, two studies had estimated that VAP prolongs the length of mechanical ventilation by 7.6 to 11.5 days and prolongs hospitalization by 11.5 to 13.1 days compared with similar patients without VAP.[4, 5]

Currently there is no valid, reliable definition for VAP, and even the most widely-used VAP criteria and definitions lack accuracy. Clinical Pulmonary Infection Score (CPIS) have been suggested to identify mechanically ventilated patients at high risk of developing VAP.[6] A score of more than 8 was found to have a sensitivity of 80%, and specificity of 80%.[3] An another study comparing the Acute Physiology and Chronic Health Evaluation II (APACHE II) and CPIS scores for the predicting 30-day mortality in patients with VAP, APACHE II had better discrimination for predicting 30-day mortality in patients with VAP, with area under curve 0.808 (95% CI)0.704–0.912, $p < 0.001$). [7]

Previous studies have shown that appropriate antimicrobial treatment of patients with VAP significantly improves outcome.[8] Because local epidemiology, etiology, case mix, risk factors and resistance patterns are key determinants of the success or failure of treatment, it is important that the physicians caring for mechanically ventilated patients must use these data when they choose an antibiotic for their patients suspected to have VAP. Published data from India emphasizes the importance of identifying the local microbiological flora and sensitivity patterns for rational treatment of VAP.[9]

We designed this study to look at the patient characteristics, microbiological profile and in-hospital mortality in adults receiving mechanical ventilation for medical illnesses at a teaching hospital in Maharashtra.

Subjects and Methods

Setting

We conducted a prospective observational study in the adult Medical Intensive Care Unit (ICU) of a teaching not-for-profit hospital in Maharashtra. Patients more than 12 years of age with medical-illness as the cause of hospitalization, and were in receipt of mechanical ventilation were considered for the present study. Patients were excluded if they had any one of the following—patients intubated from another hospital and referred to us; patients receiving mechanical ventilation for less than 48 hours.

Data Collection

At enrolment, we collected data on the baseline variables: age, sex, date of hospital and ICU admission, the severity of illness score (Acute Physiology and Chronic Health Evaluation [APACHE II] score), and diagnosis at admission. Laboratory parameters included complete blood counts, serum creatinine, electrolytes, chest radiographs, Ultrasound of abdomen, Computed Tomography scans (wherever applicable) and arterial

blood gas (ABG) analysis was noted at the time of evaluation. We used the definition set by Centre for Disease Control (CDC) and Prevention National Healthcare Safety Network (NHSN) for defining VAP. APACHE II score was calculated by using the values of the following variables age, temperature, heart rate, respiratory rate, mean arterial pressure, serum sodium, potassium, creatinine, hematocrit, Arterial pH, white cell count, Glasgow coma scale, A-A gradient or PaO2. It was computed within 24 hours of admission. The score ranges from 0 to 71; the upper score, further severe disease and a privileged risk of death.

The Clinical Pulmonary Infection Score (CPIS) was calculated by collecting data on six variables: tracheal secretions, chest radiograph infiltrates, temperature, white cell count, PAO2/FIO2, and endotracheal aspirate cultures.(8) The clinical pulmonary infection score of >6 was considered as highly significant of VAP. Centers of Disease Control (CDC) Atlanta guidelines were used to calculate Ventilator Associated Condition (VAC), Infection Related Ventilator Associated Complication (IVAC), Possible and Probable Ventilator Associated Pneumonia.

As a part of our ICU protocol, we provided standard ventilator care which includes VAP bundle (head end elevation, prophylaxis for peptic ulcer, deep vein thrombosis prophylaxis, and daily sedation hold). Patients were ventilated for indications such as an acute respiratory failure, inadequate oxygenation or ventilation, and airway protection in a patient with depressed mental function.

Tracheo-bronchial aspiration was collected by inserting a catheter through the endotracheal tube until resistance was met and then by making gentle suction. Samples were sent to the microbiology laboratory within 1 hour for microbiological analysis. Gram-staining and culture were performed on all respiratory samples and antibiotic sensitivity and resistance patterns were noted. All incorporated patients were followed up each day to study their clinical advancement and results. The duration of mechanical ventilation and hospital stay, as well as the status on discharge, i.e., whether alive or dead, were noted.

Data Analysis

All the statistical analyses were performed using Stata (version 14, Stata Corporation, College Station, Texas).

Descriptive analysis

The data was initially summarized with the median and mean as measures of central tendency and standard deviations and interquartile ranges (IQR) as measures of spread for continuous variables. Categorical variables were reported as numbers and percentages and they were compared using the $\chi 2$ test or Fisher exact test. Continuous variables were reported as the mean (standard deviation) or median (interquartile range), and the variables were compared using the t test or the Mann-Whitney test. All tests were 2-sided, and a P value of less than 0.05 was measured statistically considerable.

Multivariate regression models

For the multivariable analysis, we built the models by including the factors that reflect demographics, APACHE II score, diagnosis, and CPIS. The analysis was performed using a mixture of continuous, categorical and binary candidate variables. Multivariable analysis was used to select the most influential variables in the final model.

Receiver operating characteristic (ROC) curves

To evaluate the performance of the different scales, we calculated the area under the Receiver operating characteristic (ROC) curves (8) by c-statistic and calibration (the p-value of Hosmer-Lemeshow goodness of fit test). An AUC of 0.5 indicates no discrimination, and an AUC of 1.0 indicates perfect discrimination. We

used lfit, lstat, lroc and roccomp commands in Stata to test the calibration and discrimination of the models.

Observation and Results

We conducted this study in a Medical ICU of Kasturba Hospital, Sevagram. Of the 837 patients who underwent intubation and mechanical ventilation during the study period of one year, 298 patients were ventilated for 48 hours or more. These patients were free of pneumonia at admission to the intensive care unit. Of the 298 patients, 241 (81% [95% CI 76%- 85%]) died in the hospital; 57 (19% [16%-28%]) survived to hospital discharge. The median length of stay was 6(IQR 4-12) days. Compared with survivors (6[3-12] days), non-survivors stayed a day short in the hospital (5[4-12] days; P=0.001).

The incidence of VAP was 37.6% (112 of 298; 95% CI 32.0-43.1%). Of the 112 patients who developed VAP, 38 (33%) patients developed early VAP, while 74 patients (67%) developed late VAP—calculated by using a cut point of > 4 days of mechanical ventilation. Pulmonary disorders (73, 24%), poisoning (46, 15%) and neurological disorders (48, 16%) accounted for 167 (56%) of 298 admissions.

The total duration of mechanical ventilation among patients with VAP was 10.83 ± 7.37 days (median, 8.5 days [interquartile range, 6 to 12 days]) compared with 5.03 ± 3.01 days (median, 4 days [interquartile range, 3 to 6 days]) in patients without VAP (P < 0.001). The median length of stay of the VAP patients was 5 (4-9) days. The median APACHE II score of the entire study patients was 15 (IQR 10-20). The proportion dying was higher with APACHE score > 15 (140 of 160, 87.5%, odds ratio [OR] 2.4, 95% CI 1.54- 4.64; p=0.002).The median APACHE II score of those who died was numerically and statistically significantly higher compared to those who survived [16 (IQR 11-20.5) vs. 12 (8-16)]; p=0.001.

On culture, the most common organisms grown were Acinetobacter baumannii (34 isolates, 30.4%), Klebsiella (17 isolates, 15.2%), Pseudomonas (8 isolates, 7.1%), and Escherichia coli (11 isolates, 9.8%). The most common pathogen associated with VAP was Acinetobacter baumannii. It was sensitive to Ampicillin sulbactam (54%), Imipenem (24%), Doxycycline (24%) and Ceftriaxone sulbactam (12%).

The independent predictors of mortality in mechanically ventilated patients were as follows: Age 40 to 60 years (Odds ratio: 2.77 [1.06-7.21]; p=0.036; Age 60 and above (OR 3.25[1.09-9.68; p=0.034]); APACHE II score cut point 15 (OR 2.21, [1.15-4.25]); p=0.016). However CPIS score (OR 1.20[0.59-2.44]); p=0.61 and Ventilator associated pneumonia (OR 1.11[0.54-2.28]), p=0.75) were not associated with mortality.

Discussion

Principle Finding

Our study shows that a third of patients ventilated for > 48 hours in medical intensive care unit developed Ventilator associated pneumonia (VAP) and 80% of them died. The incidence of VAP was 37.6% (95% CI 32.0-43.1%); the incidence density, 38.7 cases (95% CI 32.1-46.5) /1000 ventilator days and mortality, 82.1% (95% CI 74.9-89.3%).

The incidence of VAP was 37.6% (95% CI 32.0-43.1%); the incidence density, 38.7 cases (95% CI 32.1-46.5) /1000 ventilator days and mortality, 82.1% (95% CI 74.9-89.3%). The independent predictors of mortality in mechanically ventilated patients were as follows: Age 40 to 60 years (Odds ratio: 2.77 (1.06-7.21); p=0.036; Age 60 and above (OR 3.25(1.09-9.68; p=0.034); APACHE II score > 15 (OR 2.21, (1.15-4.25); p=0.016).

The following variables lacked statistically significant association with mortality: sex (OR 1.06[0.57-1.97]; p=0.19), CPIS score (OR 1.20[0.59-2.44]; p=0.61) and VAP (OR 1.11[0.54-2.28], p=0.75).

VAP is an important cause of morbidity and mortality in intensive care units, worldwide. VAP infection complicates the course of 8–20% of mechanically ventilated patient.[10] In contrast to urinary tract infections and cellulitis which are associated with low mortality, ranging from 1 to 4%, the death rate for VAP ranges from 24 to 50%. According to a systematic review of VAP infections among adult ICU patients in developing countries the rates of VAP infections ranged between 10 and 41.7/1000 MV-days and were generally higher than NHSN benchmark rates.[3, 11] Our study found no significant association between VAP and the demographic factors studied, except for age > 40 years. In contrast to the previous studies, we found that the VAP was not associated with male sex, younger age.[12] Our patients were younger ([median age, 50 years (interquartile range [IQR], 35–63; range=12 to 91 years] compared to western countries (mean age 59– 61 years). This finding is similar to earlier studies from India, where the mean age was found to be 41 and 43 years.[12, 13]

We found that poisoning, stroke and sepsis and ARDS accounted for nearly 40% of ICU admissions requiring mechanical ventilation. Our observations are in line with a recent study from South India [13] in which poisoning accounted for 40% of admissions. In high-income countries, most of the ICU patients requiring mechanical ventilation have pneumonia, chronic obstructive pulmonary disease, sepsis, heart failure, and neurological diseases.[14, 15]

The case-mix broadly reflects the high prevalence of poisoning, strokes, and infectious diseases among patients presenting to the ICU of our hospital.

Mortality

Patients with VAP emerge to have a 2- to 10-fold superior risk of death compared with patients devoid of pneumonia. The crude 30-day VAP mortality in Asian countries was reported as 44.8% (11.1%–66.7%).[16] Indian studies also show that ventilator associated pneumonia kill a large number of patients in ICU.[12, 17] In our study, of the 298 patients mechanically ventilated for > 2 days, 112 developed VAP— 92 (82.14%) of them died, indicating that our mortality numbers are at the top of the range. Ranjan and colleagues [17] reported VAP incidence of 57 % (60 out of 105); almost half of their study patients died with early VAP. Mathai and colleagues recently reported a mortality of 68.4% in their study.[18]

The in-hospital mortality in VAP positive patients varies considerably. For example, three Indian studies, published recently, have reported mortality of 68% (n= 250; VAP incidence 38%) (20); 37% (n=237; VAP incidence= 61%) (14) and 48% (n=105, VAP incidence 57%) [17] In a study from Brazil, the in-hospital mortality of patients on invasive mechanical ventilation was 43%, much lower than ours.[19] Thus, the in-hospital mortality in our setting (82%) was substantially higher comparable to previous studies. Our findings concur with historical studies from low-resource settings that had often documented mortality rates exceeding 70%.[20-22]

Our observations suggest that outcome of mechanically ventilated patients in our setting has failed to keep pace with impressive gains in other low-resource settings.

Length of Stay

In our study, the median (IQR) length of ventilator stay of those with VAP died was significantly higher compared to those who survived (11[7-18] days vs. 5[4-9] days, p=

0.001). Similarly, patients with VAP were ventilated longer 8.5 (IQR 6-12.5 days) vs. 4(3-6) days compared to those without. There was no difference in the mortality rates of patients with VAP compared to those without (p=0.18). In a retrospective matched cohort study from a large US in-patient database, the VAP significantly prolonged duration of MV (14.3 ± 15.5 days vs. 4.7 ± 7.0 days), ICU LOS (11.7 ± 11.0 days vs. 5.6 ± 6.1 days), and hospital LOS (25.5 ± 22.8 days vs. 14.0 ± 14.6 days).[23] The mean duration of mechanical ventilation in the present study was longer compared to the findings of the MV-ISG (5.9 days)[24] and a recent study from South India (10.9 days).[13]

Our data discovered analogous mortality rates stuck between patients with and without VAP infections. However, the duration of mechanical ventilation and hospital stay (10 days [6-16] vs. 5 days [3-8] were all significantly prolonged in patients with VAP infections. Our results are in line with the results of other study that showed that VAP prolongs hospital stay.[18]

Microbiology

In our study, Gram-negative organisms accounted for most cases of VAP infections (80%), a finding similar to that noted in Asian study.[12] Indian study reported that most cases of VAP found in their tertiary level ICU were caused by Gram-negative bacteria, (80.9%) such as Pseudomonas aeruginosa (21.3%) and Acinetobacter baumannii (21.3%).[25] A recent report presented by a panel of experts from ten Asian countries suggested that the prevalence of MDR pathogens is rising in Asian countries, and Acinetobacter baumannii– complex is emerging as a major pathogen in most of these ICUs.[12] Ranjan and colleagues reported a high prevalence of Gram-negative bacilli (96%) in their study.[17] We also found that Acinetobacter baumannii was isolated in one of six cases of VAP. Acinetobacter spp. (35% of VAP cases) and Pseudomonas aeruginosa (26%) were the major pathogens associated with VAP. Along with the whole 60 episodes of VAP reported, 10 episodes of VAP were polymicrobial and 50 episodes were monomicrobial. In the monomicrobial episodes, Gram-negative isolates accounted for 96% and even in polymicrobial episodes of VAP Gram-negative isolates were predominant accounting for 90%.

Indian tertiary care hospital reported prevalence of 48% of MDR Acinetobacter infections and 27% of MDR Pseudomonas infections. On culture, the most common organisms grown in patients with VAP was Acinetobacter baumannii (34 isolates, 30.3%), Klebsiella (17 isolates, 15.2%), Pseudomonas (8 isolates, 7.1%), and Escherichia coli (11 isolates, 9.8%).In contrast to several studies, we noted a relatively low proportion of our VAP infections caused by MDR pathogens, including carbapenem-resistant organisms. We found that Acinetobacter baumannii was sensitive to Ampicillin sulbactam (54%), Imipenem (24%), and Ceftriaxone sulbactam (12%) but resistant to many other antibiotics.

CPIS and APACHE II

The CPIS is calculated by clinical, physiological, microbiological and radiographic evidence to predict the presence or absence of VAP. Score ranges between 0 and 12 with a score of ≥ 6 showing high suspicion of VAP.[26] One meta-analysis of 13 studies which evaluated the accuracy of CPIS in diagnosing VAP reported pooled estimates for sensitivity and specificity for CPIS as 65 % (95 % CI 61-69 %) and 64 % (95 % CI 60-67 %), respectively. Of all the criteria used to calculate the CPIS, only time-dependent changes in the PaO2/FiO2 ratio early in VAP may provide some predictive power for VAP outcomes in clinical trials, namely clinical failure and mortality.[16]

Three studies have studied the performance of CPIS, with a total of 303 patients.[12, 18, 27] The observed mortality rates between them ranged from 28% to 50.8%, and the AUC ranged from 0.61 to 0.71. The AUC for CPIS in our study (0.88; 95% CI 0.84-0.91) indicates that it distinguished patients with VAP from those without, accurately. Singh and colleagues [28] showed that the CPIS is an effective clinical tool for determining whether to stop or continue antibiotics for longer than 3 days. Our data confirm these observations- the AROC for predicting the outcome for CPIS score was 0.53 (95% CI, 0.45 to 0.61)—indicating poor discriminative power.

The CPIS has been most successfully used in guiding treatment decisions for patients with a low likelihood of VAP, for whom CPIS guided therapy lowered costs and reduced the development of antimicrobial resistance.

APACHE II

According to a 2016 systematic review [29], tested APACHE II in predicting mortality in VAP patients. The AUC of APACHE II for predicting the mortality ranged from 0.53 to 0.87. APACHE II was both found to be an independent risk factor for VAP mortality but failed to predict hospital mortality.[30] APACHE II had a modest discriminatory ability for distinguishing survivors from non-survivors (AUC 0.653 [95% CI 0.57-0.72]. APACHE II as predictor of mortality impact and important in clinical decision making by helping in predicting the outcome, prolonging Ventilatory support, and anticipating clinical failure. APACHE II is a time consuming model and requires 15 variables.

Our data indicate that mechanical ventilation could be clinically effective in low-resource settings. We wish to sound a note of caution. We need to prevent nosocomial infections and improve care processes to reduce the mortality associated with VAP. To do so, we need to have more resources (nurses, technicians and residents and intensivist) in the ICU. Our findings would be applicable to settings where a similar case-mix is encountered. Although the case-mix differs, our conclusion might be valid to low-resource settings with material resources for mechanical ventilation, but not expert doctors, nurses, and additional paramedical human resources. We do not know if our findings could be applied to non-teaching hospitals in low-resource settings.

Strengths and Limitations

The study has several strengths. First, the sample size was large enough to obtain very precise estimates of mortality and to look for the risk factors for in-hospital mortality. Second, our study was prospectively conducted, from a clinician's viewpoint, with the diagnosis of VAP based on clinical criteria, and supplemented by microbiological results. Till today, most Indian studies on VAP infections are from a laboratory-based perception. Third, the admissions to the ICU reflect typical ICU admissions in a large teaching hospital in India. Our case mix represents a typical case-mix of Indian ICU with a variety of neurological, infectious, and poisoning acute illnesses. Fourth, all patients were followed up until discharge.

The study has some limitations. First, we did not attempt to verify the accuracy of a diagnosis of VAP. Notwithstanding, this metric reflects the real life situation, at least in our relatively resource limited setting. Second, we followed all patients only till discharge. We do not know if patients died soon after discharge, either at home or in another hospital. Third, the respiratory samples were obtained by blind endotracheal aspiration, and quantitative cultures could not be done on tracheobronchial microbiological aspirates, due to the limitation of resources. We might have overestimated the incidence of VAP infection.

Fourth, although all ventilated patients had their head elevated to 30°, we did not consistently use other components of VAP bundle (subglottic aspiration, silver-encoated tubes, and oropharyngeal care with chlorhexidine) in our mechanically ventilated patients. Our patients could have fared much better had they received all components of the VAP bundle.

VAP is associated with enormous adverse events: it triggers morbidity, prolongs ICU LOS, prolongs the length of stay on mechanical ventilation, and increases costs of hospitalization. To reduce these adverse events, intensive care units need design checklists, protocols, and standard operating procedures. The measures should include, but are not limited to, education, increased awareness of hand hygiene measures, reduction of the duration of mechanical ventilation and universal use of VAP bundles, all of which have been proven to reduce the risk of VAP infections. The International Nosocomial Infection Control Consortium (INICC) data which studied VAP infections from 44 adult ICUs from 14 developing countries, noted that implementation of a multi-dimensional approach which included, bundle of infection control interventions, education outcome surveillance, process surveillance, feedback of VAP rates and performance feedback of infection control practices resulted in a 55.8% decrease in the rate of VAP infection from 22.0 to 17.2/1000 MV days.[30] More specifically, the data from 21 ICUs across ten Indian cities demonstrated a 38% decrease in the VAP rates, from 17.43/1000 MV days to 10.81/1000 MV days (relative risk 0.62, 95% CI: 0.5–0.78, P = 0.0001) during the same study period, and using the same interventional measures.[31] We also believe that if nurses, residents, physicians, and technicians work together and adhere to the ICU check lists and SOPS, we could see more mechanically ventilated patients escape the VAP misery.

Table 1: Demographic characteristics

Characteristic	Survivors (n=57)	Non-Survivors (n=241)	P value	Total (n=298)
Age, mean, y	42.6 (19.6)	49.6(17.4)	0.04	25 (8.39)
12-20 y, n (%)	10(17.5)	15(6.22)		77 (25.4) 118(39.6)
21-40 y, n (%)	17 (29.8)	60 (24.9)	0.02	53 (24.4)
41-60 y, n (%)	20 (35.1)	98 (39.6)		78 (26.2)
>60 y, n (%)	10 (17.5)	68 (28.2)		
Sex, n (%)				
Male	39 (18.8)	168 (81.1)	0.84	207(73.3)
Female	18 (19.8)	73 (81.2)		91 (26.7)
Abbreviation: SD – Standard deviation; P value (two-sided) for t-test (means), Wilcoxon's Mann-Whitney U test or Fisher's exact test (medians) or Chi-square test (proportions) comparing characteristics of survivors and non-survivors.				

Figure 1: Receiver operating curves for predicting mortality in critically ill patients requiring mechanical ventilation according to the value of CPIS score and APACHE II

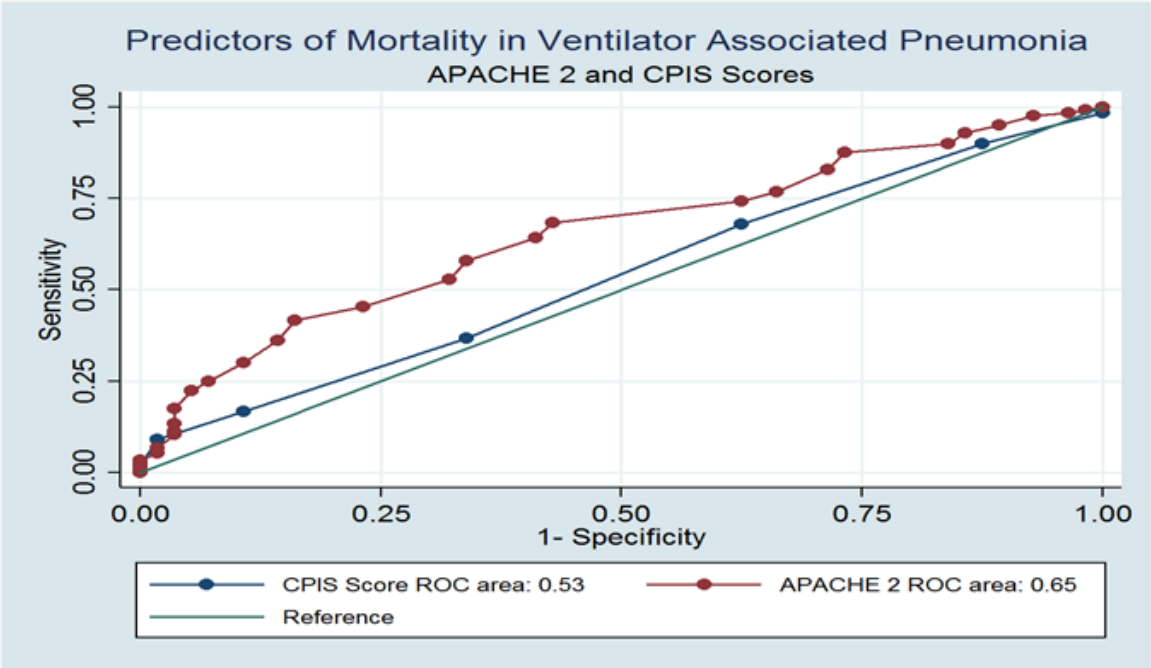

Figure 2: Acute Physiology and Chronic Health Evaluation (APACHE) II, and mortality in patients proven to have Ventilator Associated Pneumonia

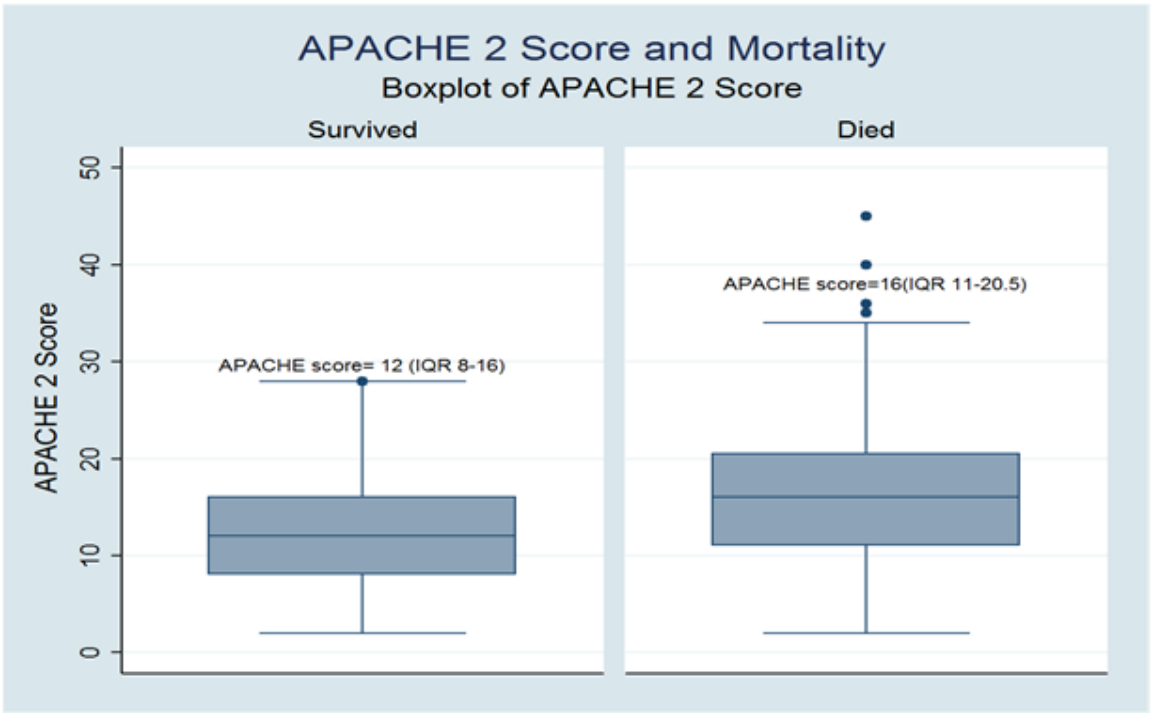

Figure 3: Boxplot of Length of stay on mechanical ventilator and hospital mortality

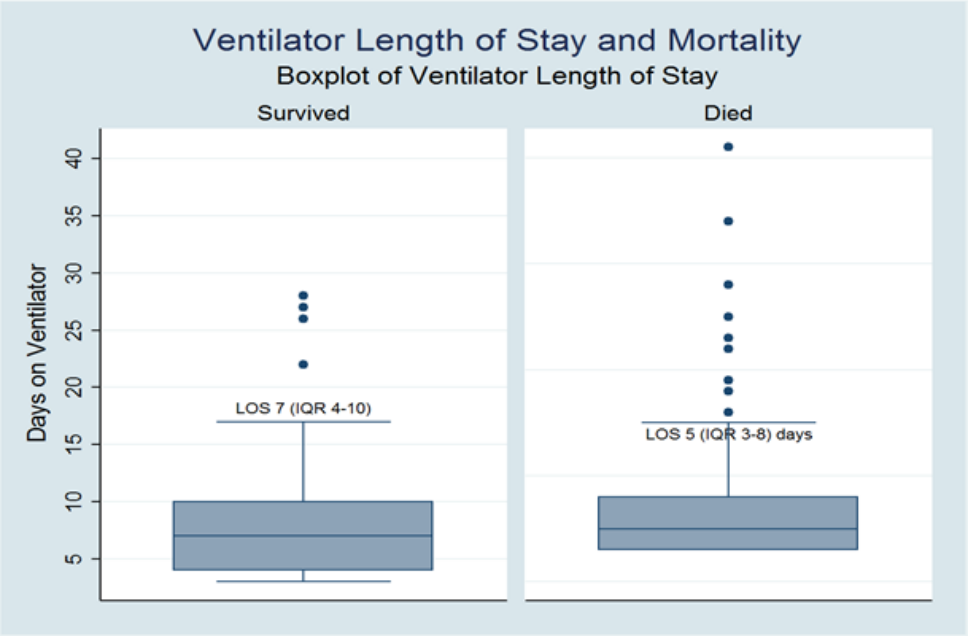

Table 2: Predictors of mortality: Multivariate logistic regression analysis

		Univariate Analysis			Multivariate Analysis		
Risk Factor	No. of Events	Odds Ratio	95% CI	P Value	Adjusted Odds Ratio	95% CI	P Value
Age, Years							
Reference	15/25						
20-40	60/77	2.35	0.89-6.75	0.082	2.38	0.89-6.37	0.08
40-60	98/118	3.26	1.28-8.30	0.013	2.78	1.06-7.23	0.03
>60	68/78	4.53	1.60-12.82	0.004	3.19	1.07-9.49	0.03
Sex							
Men	168/207						
Women	73/91	1.06	0.57-1.97	0.19			
VAP on ABG							
No	165/205	1/54	0.76-3.11	0.22			
Yes	70/82						
VAP on Chest x-ray							
No	51/61						
Yes	165/205	1.54	0.76-3.11	0.22			
VAC							
Yes	112/130	0.53	0.28-0.98	0.043			
No	129/168						

IVAC							
No	158/188	0.58	0.52-1.04	0.07			
Yes	83/110						
Probable VAP							
No	153/191	1.15	0.62-2.11	0.65			
Yes	88/107						
CPIS							
<3	77/98	1.24	0.68-1.29	0.24	1.11	0.84-1.48	0.43
>3	164/200						
VAP							
No	149/186	1.14	0.62-2.071.40-	0.66	0.99	0.44-2.22	0.99
Yes							
APACHE II Score>15	101/138	2.56	1.40-4.67	0.002	2.18	1.13-4.13	0.013

Abbreviations: ABG: Arterial blood gases

VAC: Ventilator Associated Condition

IVAC: Infection Ventilator Associated Condition

VAP: Ventilator Associated Pneumonia

APACHE II: Acute Physiology and Chronic Health Evaluation Score

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