A STUDY OF VENTILATOR ASSOCIATED PNEUMONIA –RISK FACTORS AND OUTCOMES

By

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DISSERTATION SUBMITTED TO BLDE DEEMED UNIVERSITY,

VIJAYAPURA

In partial fulfillment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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ACKNOWLEDGEMENT

I have no words to express my deep sense of gratitude and regard to my guide, **Dr. SANJEEVKUMAR N. BENTOOR M.D**, Professor of Medicine, Head of the Department of General Medicine, under whose inspiring guidance & supervision, I am studying and continuing to learn the art of medicine. His deep knowledge, devotion to work and zeal for scientific research makes him a source of inspiration for me and others. It is because of his generous help, expert and vigilant supervision, that has guided& helped me to bring out this work in the present form.

I sincerely thank **Dr. ARAVIND V PATIL** Principal, Shri B.M. Patil Medical College and Research Centre, Vijayapura, for permitting me to conduct this study.

Dr. GUJJA RAGHAV

ABBREVIATIONS

APACHE	–	Acute Physiology and Chronic Health Evaluation
ARDS	–	Acute Respiratory Distress Syndrome
BAL	–	Bronchoalveolar Lavage
CAP	–	Community-Acquired Pneumonia
CDC	–	Centers for Disease Control and Prevention
CFU	–	Colony Forming Units
COPD	–	Chronic Obstructive Pulmonary Disease
CT	–	Computed Tomography
CTTI	–	Clinical Trials Transformation Initiative
ECDC	–	European Centre for Disease Prevention and Control
FiO_2	–	Fraction of Inspired Oxygen
HAP	–	Hospital-Acquired Pneumonia
HCAP	–	Health Care-Associated Pneumonia (Retired Term)
HR	–	Hazard Ratio
ICU	–	Intensive Care Unit
ICU-HAP	–	Intensive Care Unit-Hospital Acquired Pneumonia
IVAC	–	Infection-related Ventilator-Associated Complication
MDR	–	Multidrug-Resistant
MODS	–	Multi-Organ Dysfunction Syndrome
MRSA	–	Methicillin-Resistant Staphylococcus aureus
MSSA	–	Methicillin-Susceptible Staphylococcus aureus
NHSN	–	National Healthcare Safety Network
PCR	–	Polymerase Chain Reaction
PDR	–	Pandrug-Resistant

PEEP	–	Positive End-Expiratory Pressure
PSB	–	Protected Specimen Brush
SOFA	–	Sequential Organ Failure Assessment
VAC	–	Ventilator-Associated Condition
VAE	–	Ventilator-Associated Event
VAP	–	Ventilator-Associated Pneumonia
VHAP	–	Ventilated Hospital-Acquired Pneumonia
XDR	–	Extensively Drug-Resistant

ABSTRACT

Background:

Ventilator-associated pneumonia (VAP) is a serious nosocomial infection that occurs in mechanically ventilated patients, increasing morbidity, mortality, and healthcare costs. It is one of the most common ICU-acquired infections, with a significant impact on patient outcomes. Understanding the risk factors and microbial profile of VAP is crucial for improving prevention and treatment strategies.

Aim:

This study aimed to assess the prevalence, risk factors, microbiological profile, and outcomes of VAP in critically ill patients requiring mechanical ventilation.

Materials and Methods:

A prospective observational study was conducted on 175 mechanically ventilated patients in the medical ICU, fulfilling the inclusion criteria. Clinical data, risk factors, microbiological findings, and patient outcomes were analysed. Endotracheal cultures were performed, and antimicrobial susceptibility patterns were evaluated. Statistical analysis was conducted using SPSS version 20, with significance set at $p<0.05$.

Results:

The mean age of patients was 48.71 years, with a male preponderance (70.9%). The mean duration of mechanical ventilation was 11.1 days, and VAP was present in 74.3% of cases, with 44% classified as late-onset and 30.3% as early-onset VAP. The most common risk factors included emergency intubation (68.6%), impaired consciousness (64.6%), and prolonged mechanical ventilation (>7 days) (58.3%). The predominant pathogens were *Acinetobacter baumannii* and *Klebsiella*

pneumoniae (20% each), with multidrug-resistant strains posing a significant challenge. High resistance was noted against cephalosporins, fluoroquinolones, and carbapenems, while Tigecycline remained highly effective (97.4% sensitivity). No significant difference in patient outcomes was observed between VAP and non-VAP groups, but prolonged ventilation, emergency intubation, and tracheostomy were significantly associated with VAP development ($p<0.05$).

Conclusion:

VAP remains a major ICU concern, with emergency intubation, prolonged ventilation, and tracheostomy being key risk factors. The high prevalence of multidrug-resistant organisms highlights the need for strict infection control measures and antimicrobial stewardship programs to improve patient outcomes.

Keywords:

Ventilator-associated pneumonia, risk factors, mechanical ventilation, ICU infections, antimicrobial resistance, multidrug-resistant organisms.

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INTRODUCTION

"Ventilator associated penumonia (VAP) is a nosocomial infection which develops after 48 hours of mechanical ventilation. It is one of the most important complications of the modern day intensive care units (ICUs). The risk of pneumonia for patients on ventilator increases by 3-10 times. Ventilator-associated pneumonia (VAP) is the most common nosocomial infection in people receiving mechanical ventilation. VAP is defined as pneumonia occurring more than 48 h after endotracheal intubation/initiation of mechanical ventilation or pneumonia developing even after extubation. [1,2]

VAP is the most common ICU-acquired infection among mechanically ventilated patients.[3] VAP is a kind of hospital-acquired pneumonia. It affects 9-27 percent of ventilated patients.[4] In ICU patients with pneumonia in India, the total crude death rate is 67.4 percent, with infection accounting for 40 percent of the mortality."[5]

Intensive care facilities, length of hospital stay, and previous antibiotic use all affect the frequency of VAP and the organisms that cause it. "The onset of ventilator-associated pneumonia was found to be significantly influenced by the presence of organ failure, COPD, emergency intubation, and re-intubation.[6–9]

Notably, the most frequent etiological agents of VAP in both early and late groups have been found as Acinetobacter species, Klebsiella pneumoniae, and Pseudomonas aeruginosa." The morbidity and mortality rates associated with ventilator-associated pneumonia are considerably higher.[10,11]

Present study aimed to assess the various risk factors and outcome in patients with ventilator associated pneumonia.

REVIEW OF LITERATURE

Pneumonia is often classified based on the location where it was acquired. "Hospital-acquired pneumonia (HAP), also known as nosocomial pneumonia, occurs 48 hours or more after hospital admission and is not present or incubating at the time of admission. Ventilator-associated pneumonia (VAP) is a specific form of HAP that develops 48 hours or more after endotracheal intubation and mechanical ventilation."[12] "Ventilator-associated pneumonia (VAP) is a significant concern in intensive care units, as it is associated with a higher risk of mortality. Prompt and accurate diagnosis is essential to initiate timely and appropriate treatment while minimizing antibiotic overuse, which could contribute to antibiotic resistance. Notably, patients with severe hospital-acquired pneumonia (HAP) who require mechanical ventilation after the onset of infection do not fall under the VAP category; this condition is referred to as ventilated hospital-acquired pneumonia (VHAP). Despite this distinction, VHAP shares similar microbiology, diagnostic approaches, and clinical outcomes with VAP rather than with HAP."[12–15]

"Term	**Definition**
Classification by site of acquisition	
Community-acquired pneumonia (CAP)	Acute pulmonary parenchymal infection obtained outside of a health-care environment.
Nosocomial pneumonia	An acute infection of the pulmonary parenchyma acquired in hospital settings, which encompasses hospital-acquired pneumonia and ventilator-associated pneumonia
Hospital-acquired pneumonia (HAP)	Pneumonia acquired ≥48 hours after hospital admission; includes both HAP and VAP

Ventilator-associated pneumonia (VAP)	Pneumonia acquired ≥48 hours after endotracheal intubation
Health care-associated pneumonia (HCAP)	Retired term, which referred to pneumonia acquired in health care facilities (eg, nursing homes, hemodialysis centers) or after recent hospitalization*
Classification by etiology	
Atypical pneumonia	Pneumonia caused by "atypical"¶ bacterial pathogens including , *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Legionella* spp, *Chlamydia psittaci*, and *Coxiella burnetii*
Aspiration pneumonia	Adverse pulmonary effects caused by the admission of stomach or oropharyngeal fluids, which may include germs and/or have a low pH, or exogenous substances (for example, ingested food particles or liquids, mineral oil, salt, or fresh water) into the lower airways.
Chemical pneumonitis	Aspiration of substances (eg, acidic gastric fluid) that cause an inflammatory reaction in the lower airways, independent of bacterial infection
Bacterial aspiration pneumonia	An active infection caused by huge numbers of microorganisms being inoculated into the lungs via orogastric contents.”

“The term "health care-associated pneumonia" (HCAP) was added to the American Thoracic Society/Infectious Diseases Society of America (ATS/IDSA) guidelines in 2005, and it referred to pneumonia acquired in health care facilities such as nursing homes, hemodialysis centres, outpatient clinics, or during a hospitalisation

within the previous three months. This category was used to identify patients who were at risk of infection with multidrug-resistant (MDR) pathogens based on their specific risk factors and illness severity."[16]

Antimicrobial resistance: "The Centers for Disease Control and Prevention (CDC) in the United States and the European Centre for Disease Prevention and Control (ECDC) in Europe have established standardized terminology for antimicrobial-resistant gram-negative bacilli, which are significant pathogens responsible for hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP)."[17]

"Multidrug resistant (MDR) refers to acquired non-susceptibility to at least one agent in three different antimicrobial classes.

Extensively drug resistant (XDR) refers to non-susceptibility to at least one agent in all but two antimicrobial classes.

Pandrug resistant (PDR) refers to non-susceptibility to all antimicrobial agents that can be used for treatment."

Epidemiology:

"The National Healthcare Safety Network (NHSN) of the Centers for Disease Control and Prevention (CDC) reports a consistent decline in ventilator-associated pneumonia (VAP) rates in the United States. Between 2006 and 2012, the incidence of VAP per 1,000 ventilator-days dropped from 3.1 to 0.9 in medical intensive care units (ICUs) and from 5.2 to 2.0 in surgical ICUs.[18,19]

The NHSN definition of ventilator-associated pneumonia (VAP) incorporates qualitative criteria, such as increased secretions or worsening oxygenation. As a result, it remains uncertain whether the reported decline in VAP incidence reflects an

actual reduction in cases or is attributable to stricter adherence to these subjective criteria."[20]

"Long hospital stays and high expenses are related with VAP.[12] Two studies found that VAP increases the time of mechanical ventilation by 7.6 to 11.5 days and hospitalisation by 11.5 to 13.1 days when compared to identical patients who did not have VAP; the extra expense associated with VAP has been estimated to be over USD $40,000 per patient."[21,22]

Pathogenesis:

"The pathophysiology of hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) involves the interplay between the quantity and virulence of microorganisms entering the lower respiratory tract and the host's immune defenses, including humoral, mechanical, and cellular mechanisms. The primary route of lung infection is the microaspiration of pathogens colonizing the oropharyngeal tract, with the gastrointestinal tract serving as a less common source." Aspiration occurs in approximately 45% of healthy individuals during sleep and is even more frequent among critically ill patients, where it occurs regularly.[23] "Although it is commonly thought to be largely protective, the placement of an endotracheal tube increases the aspiration of oropharyngeal secretions and microorganisms into the lungs. Pneumonia may result depending on the amount and aggressiveness of organisms that enter the lung, as well as the human response.[24,25]

Clinical presentation:

More than 48 hours after intubation, the majority of patients with VAP experience a gradual or sudden onset of the following symptoms.[26]

Symptoms: dyspnea

Signs:

Fever

Hemoptysis

Tachypnea,

Purulent secretion

Rhonchi

Reduced breath sounds

Crackles

Bronchospasm

Ventilator mechanics: reduced tidal volume, increased inspiratory pressure

Laboratory findings: worsening hypoxemia, leukocytosis

Microbiology:

Hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) are often polymicrobial infections caused by a diverse range of pathogens. Common causative agents include aerobic gram-negative bacilli such as *Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter* species, *Pseudomonas aeruginosa*, and *Acinetobacter* species. Additionally, gram-positive cocci, including *Staphylococcus aureus* (notably methicillin-resistant *S. aureus* [MRSA]) and various *Streptococcus* species, are frequently implicated."[27,28] "There is growing realisation that viruses may cause a significant proportion of nosocomial pneumonias in regular medical and surgical patients, as well as viruses and fungi in immunocompromised patients."

"Methicillin-susceptible S. aureus (MSSA; 9 percent), MRSA (18 percent), P. aeruginosa (18 percent), Stenotrophomonas maltophilia (7 percent), Acinetobacter spp (8 percent), and other species were among the infecting flora in VAP patients (9 percent)."

"In nonventilated patients with HAP, the infecting flora was comparable, with the exception that non-Enterobacteriaceae gram-negative bacilli (P. aeruginosa, Acinetobacter, and S. maltophilia) were less common. It specifically contained MSSA (13%), MRSA (20%), P. aeruginosa (9%), S. maltophilia (1%), Acinetobacter spp (3%), and other species (18 percent)."

Risk factors for MDR:

"The pathogenesis of hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) is significantly shaped by the patient's vulnerability to multidrug-resistant (MDR) pathogens. The prevalence of MDR infections differs across hospitals, within different hospital units, and among patient populations. Key risk factors for acquiring MDR pathogens include prolonged hospital stays and recent exposure to antibiotics. Understanding the local susceptibility patterns of nosocomial infections within a specific healthcare setting is essential for selecting appropriate empiric antibiotic therapy and optimizing patient outcomes.[12]

Table 1: Risk factors for multidrug resistant ventilator associated pneumonia

Risk factors for MDR pathogens:
IV antibiotic use within the previous 90 days
Septic shock at the time of VAP
ARDS preceding VAP
Equal or more than 5 days of hospitalization prior to the occurrence of VAP
Acute renal replacement therapy prior to VAP onset
Risk factors for MDR *Pseudomonas* and other gram-negative bacilli:
Treatment in an ICU in which more than 10 percent of gram-negative isolates are resistant to an agent being considered for monotherapy
ICU Treatment in which local antimicrobial susceptibility rates are not known

Colonization with OR prior isolation of MDR *Pseudomonas* or other gram-negative bacilli
Risk factors for MRSA:
Treatment in a unit in which >10 to 20 percent of *Staphylococcus aureus* isolates are methicillin resistant
Treatment in a unit in which the prevalence of MRSA is not known
Colonization with OR prior isolation of MRSA

Diagnostic evaluation:

VAP should be considered in individuals who have a new or increasing pulmonary infiltrate on imaging, as well as supporting clinical indications of infection (eg, fever, secretions, leukocytosis). When a pathogen is identified in lower respiratory tract sample, the diagnosis is confirmed."

Computed tomography:

Chest computed tomography (CT) "without contrast is not routinely utilized for patients with suspected ventilator-associated pneumonia (VAP). However, it can be useful in cases where patients present with clinical signs of respiratory infection, such as fever, leukocytosis, and purulent tracheobronchial secretions, but have a normal chest radiograph. CT may also help identify a specific lobe for targeted sampling. Additionally, chest CT can be indicated for patients with a prior CT diagnosis of pneumonia to assess for new or worsening abnormalities, including the development of pleural effusions. Nonetheless, pulmonary infiltrates are frequently observed in mechanically ventilated patients and may result from various causes, making imaging-based assessment of VAP in critical care settings challenging and often inconclusive."[29–31]

Respiratory tract sampling:

"Because antibiotic therapy lowers the sensitivity of both microscopic analysis and culture, respiratory samples are preferably acquired prior to the commencement of medications or modification of antibiotic therapy (in those currently receiving antibiotics).[32–34] However, it is not unusual for severe sickness or sampling delays to necessitate the administration of empiric antibiotics prior to diagnostic sampling."

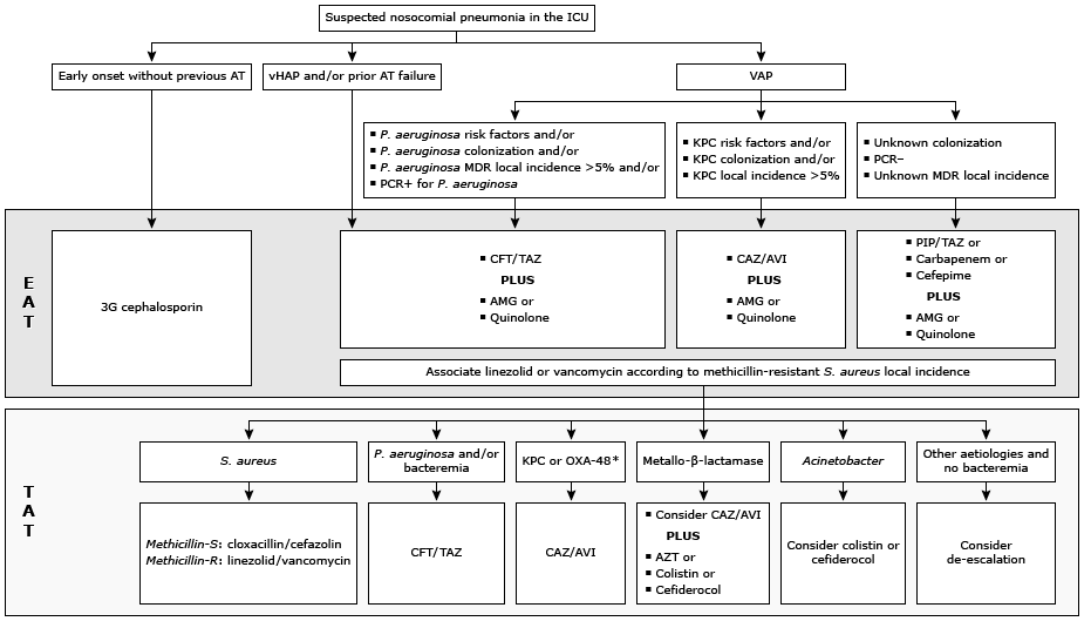

Figure 1: Suspected nosocomial pneumonia in the intensive care unit

"Invasive sampling methods for suspected VAP include nonbronchoscopic techniques, such as mini-bronchoalveolar lavage (mini-BAL), and bronchoscopic techniques, including bronchoscopic BAL and protected specimen brush (PSB). Among these, bronchoscopic BAL is the preferred method for sampling the lower respiratory tract. This preference is due to the larger sample size obtained with BAL compared to PSB (and potentially mini-BAL), which provides a dominant alveolar sample with minimal contamination from the upper airways. Several studies have shown that bronchoscopic sampling can reduce inappropriate antibiotic use and enable quicker de-escalation of antimicrobial therapy without negatively impacting

mortality or hospital stay duration, as compared to noninvasive methods like endotracheal aspirates."[35,36]

"Mini-BAL is performed by blindly advancing a catheter through the endotracheal tube until resistance is met, then infusing sterile saline through the catheter (typically three 50 mL aliquots), and aspirating with the syringe (the catheter is estimated to be located in the distal endobronchial airway (eg, second or third order bronchus)."

Microscopic analysis and quantitative culture:

"All respiratory tract samples should be sent for microscopic analysis, and it is preferred to obtain quantitative cultures. Microscopic examination typically involves a semi-quantitative assessment of polymorphonuclear leukocytes and other cell types, along with Gram staining. Although microscopy is not definitive for diagnosing VAP, the data from this examination are available before culture results and can help identify a likely pathogen. This early information can guide the adjustment of antibiotic therapy to better target the infection.[37] The presence of a high number of neutrophils in respiratory samples is consistent with VAP, and the bacterial morphology can help identify potential pathogens, such as Gram-negative rods. A prospective cohort analysis of 39 patients with BAL found that VAP could be confidently ruled out in those who had fewer than 50% neutrophils in their total nucleated cells." Quantitative cultures can be used to enumerate bacteria in respiratory samples. When bacterial growth exceeds a specific threshold, VAP is considered to be present.[38] "Only pulmonary pathogen bacteria should be counted. Staphylococcus epidermidis and most Gram-positive bacilli (excluding actinomycosis and nocardia) are examples of organisms that should not be counted.

Typical thresholds include the following:

- Endotracheal aspirates – ≥1,000,000 colony forming units (cfu)/mL
- Bronchoscopic- or mini-BAL – 10,000 cfu/mL
- PSB – 1000 cfu/mL

The thresholds used in quantitative cultures are high enough to reduce the likelihood of misdiagnosing tracheobronchial colonization as VAP. However, quantitative cultures are not routinely performed in most laboratories unless specifically requested, as they are considered more labor-intensive and costly compared to qualitative or semi-quantitative cultures. Similarly, anaerobe quantification generally follows the same guidelines but is more time-consuming and requires specialized laboratory expertise, which means it is only conducted in select facilities."

Non-invasive respiratory sampling:

"Tracheobronchial aspiration (ie, endotracheal aspirate) is performed by advancing a catheter through the endotracheal tube until resistance is met and suction is applied (likely located in trachea or main stem bronchus. The sample is directly aspirated into a sterile specimen trap that can be sent for microbiologic analysis."

Lung biopsy criteria:

Lung biopsy is not commonly performed in patients with suspected VAP because most cases can be diagnosed through lower respiratory tract samples and cultures. "It is typically reserved for patients whose infiltrates persist despite antibacterial treatment or when the cause is suspected to be non-infectious. The purpose of obtaining tissue in these cases is to identify a pathogen that may have been overlooked in earlier samples, such as hard-to-culture organisms like fungi or herpes viruses, or to uncover a non-infectious condition that mimics an infection, such as

cancer, cryptogenic organizing pneumonitis, lymphangitis, interstitial pneumonitis, or vasculitis.

Polymerase chain reaction technique role:

Molecular approaches have emerged to aid in the fast detection and antibiotic therapy of infections, including VAP, in patients with pneumonia.[39] Polymerase chain reaction (PCR) testing, while not routinely performed or universally available, can be challenging to interpret. PCR is a rapid and cost-effective technique that amplifies small portions of microbial DNA for pathogen identification. Multiplex PCR assays, which allow multiple tests to be conducted simultaneously, are particularly useful in critically ill patients with a wide range of potential pathogens. These PCR methods can quickly detect specific bacteria in respiratory samples, enabling timely empiric antibiotic treatment and adjustments as needed. Commercially available multiplex PCR systems have demonstrated fast and relatively accurate microorganism identification in suspected VAP cases, helping to guide antibiotic therapy. However, more research is necessary to help clinicians determine the optimal use and timing of PCR in clinical practice."

Diagnosis:

"VAP is a clinical diagnosis made in a patient who has been mechanically ventilated for ≥48 hours who develops a new or progressive lung infiltrate on imaging with clinical evidence that the infiltrate is of infectious origin (eg, fever, purulent sputum, leukocytosis, and decline in oxygenation), together with a positive pathogen identified on microbiologic respiratory sample.[12]

Staphylococcus aureus, Pseudomonas aeruginosa, and other gram-negative bacilli are common pathogens recovered from VAP patients. At 2016, the Clinical Trials Transformation Initiative (CTTI) conducted a prospective trial in US hospitals.

The VAE system is a three-tiered monitoring definition that uses objective, publicly available data to identify problems, such as VAP, in mechanically ventilated adult patients."

"Ventilator-associated condition (VAC) – The first tier definition, VAC, identifies patients with a period of sustained respiratory deterioration (changes in positive end-expiratory pressure [PEEP] ≥3 cm H2O or fraction of inspired oxygen [FiO2] ≥0.2 [ie, 20 points] for two days) following a sustained period of stability or improvement on the ventilator (greater than or equal to two days)"

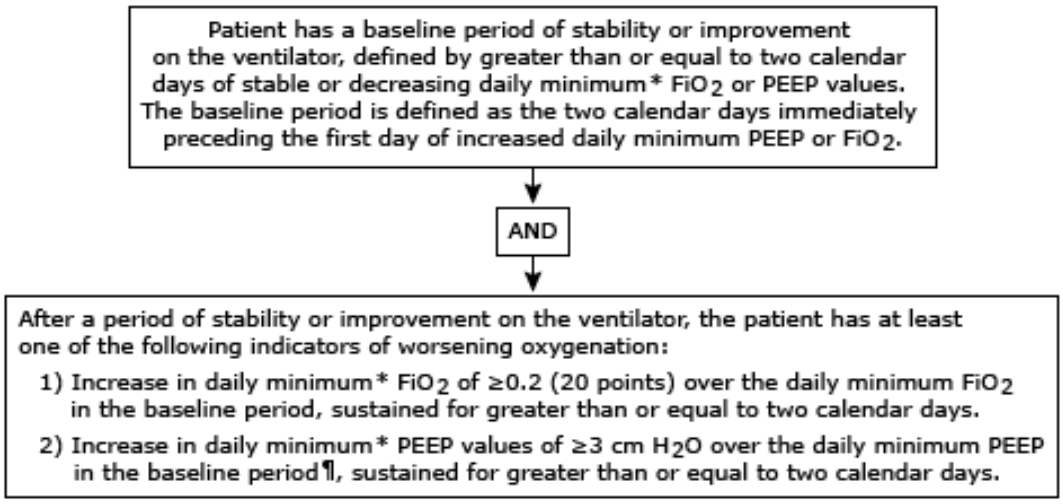

Figure 2: Ventilator associated condition

Infection-related ventilator-associated complication (IVAC) is a classification that applies to patients who exhibit ventilator-associated conditions (VAC) and meet additional criteria. Specifically, IVAC requires the patient to have an abnormal temperature (below 36°C or above 38°C) or a white blood cell count outside the normal range (≤4000 or ≥12,000 cells/mm³). Additionally, the patient must be started on one or more new antibiotics for at least four days. This definition helps to identify patients with potential infections that are complicating their ventilator use.

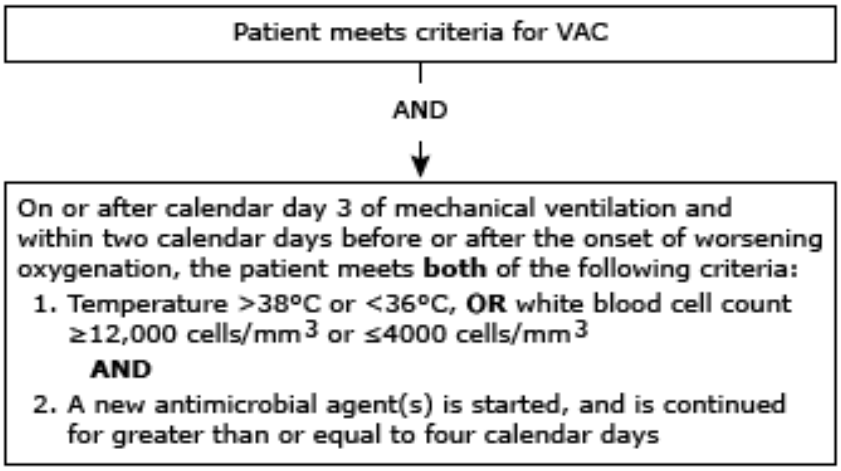

Figure 3: Infection related ventilator associated complication (IVAC)

Potential and likely VAP — "The third-tier classifications, possible and probable VAP, require IVAC patients to have laboratory and/or microbiological evidence of respiratory infection. Gram stain evidence of purulent pulmonary secretions or a pathogenic pulmonary culture in an IVAC patient is considered possible VAP."

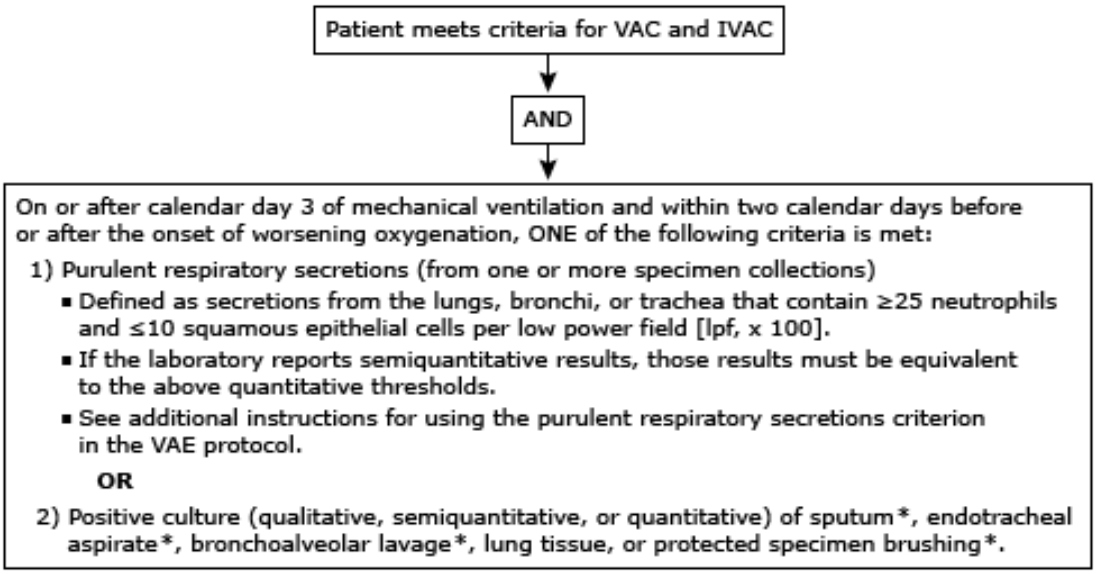

Figure 4: Possible ventilatory associated pneumonia (VAP).

Various articles;

In a study conducted by Safdar N et al., (2005) to "assess the clinical and economic consequence of VAP. The findings show that 10-20% of patients on mechanical ventilation for over 48 hours develop ventilator-associated pneumonia (VAP), which significantly increases the risk of death, with critically ill patients being twice as likely to die. VAP also leads to longer ICU stays, averaging 6.10 days, and incurs additional costs exceeding $10,019. VAP is common in ventilated patients and is linked to higher morbidity, mortality, and financial burden, highlighting the urgent need for effective prevention strategies."[40].

In a study conducted by Hugonnet S et al., (2007) to assess "the staffing level a determinant of late onset ventilator associated pneumonia. In a study of 2,470 ICU patients, 262 episodes of ventilator-associated pneumonia (VAP) were diagnosed, with 22.3% of mechanically ventilated patients developing VAP. The median duration of mechanical ventilation was 3 days for patients without VAP and 11 days for those with VAP, with late-onset VAP accounting for 61% of cases. The VAP rate was 37.6 episodes per 1,000 days at risk. A higher nurse-to-patient ratio was associated with a reduced risk of late-onset VAP (hazard ratio 0.42), but no association was found for early-onset VAP. In conclusion, a lower nurse-to-patient ratio increases the risk of late-onset VAP."[41]

In a study conducted by Bouadma L et al., (2015) to assess "the VAP in prevalence, outcome and relationship. In a study of 3,028 patients, 77% experienced at least one ventilator-associated condition, and 29% had one infection-related ventilator-associated complication episode. Nosocomial infections, including ventilator-associated pneumonia (VAP), were the leading causes of both conditions, accounting for 27.3% and 43.8% of cases, respectively. The sensitivity and specificity

for diagnosing VAP were 0.92 and 0.28 for ventilator-associated conditions, and 0.67 and 0.75 for infection-related ventilator-associated complications. Strong correlations were found between ventilator-associated conditions, infection-related ventilator-associated complications, and VAP occurrence (R2 = 0.69 and 0.82). Patients without any ventilator-associated events had a significantly higher median number of days alive without antibiotics and mechanical ventilation by day 28. Rates of ventilator-associated events were closely associated with antibiotic use within each ICU (R2 = 0.987 and 0.99). These events are common among at-risk populations and are closely linked to antibiotic consumption, suggesting they could serve as a quality indicator for improvement programs."[42]

In a study conducted by Inchai J et al., (2015) to "assess the VAP epidemiology and prognostic indicator in 30 day mortality. The study revealed a high 30-day mortality rate of 44.4% among patients with ventilator-associated pneumonia (VAP). The primary pathogens were *Acinetobacter baumannii* (54.3%), *Pseudomonas aeruginosa* (35.2%), and methicillin-resistant *Staphylococcus aureus* (15.1%). Most *A. baumannii* strains were drug-resistant (90.2%). Key prognostic factors included co-morbid malignancy (HR = 1.60), septic shock (HR = 2.51), a Simplified Acute Physiology Score II >45 (HR = 1.62), a Sequential Organ Failure Assessment score >5 (HR = 3.40), and delayed inappropriate antibiotic treatment (HR = 2.23). The study emphasized that early detection and surveillance of VAP in mechanically ventilated patients, along with timely treatment and appropriate empirical antibiotic use based on local resistance patterns, could improve outcomes."[43]

In a study conducted by Walaszek MZ et al., (2016) to "assess the risk factor for hospital acquired pneumonia in ICU. In the analyzed unit, 58 cases of ventilator-associated pneumonia (VAP) were identified in patients on mechanical ventilation,

with a higher incidence in men (6%) compared to women (3%). Mechanical ventilation lasting more than 20 days was a significant factor contributing to VAP ($p < 0.001$). Underlying diseases, such as multiple trauma, sepsis, central nervous system diseases, endocrine disorders, and respiratory diseases, influenced VAP incidence, with the highest rates observed in trauma patients (9.2%) and those with sepsis (9.7%). Invasive procedures like reintubation, tracheostomy, and bronchoscopy were significant risk factors ($p < 0.001$) for VAP development. Between 2010 and 2014, the VAP incidence was 4.7%, with an incidence density of 10.5 per 1000 ventilation-days and a mortality rate of 32.8%. The most common pathogens identified were Acinetobacter baumannii (36.4%), Pseudomonas aeruginosa (13.8%), and Escherichia coli (12%)."[44]

In a study conducted by Saied W et al., (2019) to "assess the mortality risk associated with VAP. In a study of 14,212 ICU patients who stayed for more than 48 hours, 7,735 were at risk for ventilator-associated pneumonia (VAP) and 9,747 for ICU-hospital-acquired pneumonia (ICU-HAP). VAP occurred in 15% of at-risk patients (1,161 patients), while ICU-HAP affected 2% (176 patients). After adjusting for prognostic factors, both VAP (hazard ratio 1.38) and ICU-HAP (hazard ratio 1.82) were linked to a significant increase in 30-day mortality. The adequacy of early antibiotic therapy did not improve prognosis, especially for ICU-HAP. The mortality impact was similar for infections caused by P. aeruginosa and the ESKAPE group of pathogens. The study concluded that both types of pneumonia increased 30-day mortality by 82% and 38%, respectively, highlighting the need for effective prevention strategies for ICU-HAP in non-ventilated patients."[45]

In a review study conducted by Wu D et al., (2019) to "assess the risk factors fo VAP in critically ill patients. Patients with disorders of consciousness experience

significantly longer hospital stays and mechanical ventilation durations, leading to increased exposure to invasive procedures and the bacterial environment in the ICU.

This heightened exposure significantly raises the risk of developing ventilator-associated pneumonia (VAP). Identifying the risk factors for VAP is crucial for effective clinical prevention. This review examined recent retrospective and prospective clinical trials from various global centers on VAP risk factors, but noted variability in study design, sample size, patient demographics, and geography, which can result in inconsistent findings. Additionally, the lack of standardized diagnostic criteria and treatment protocols for VAP affects the accuracy of the results. Therefore, further research with larger sample sizes and unified definitions is essential to improve the understanding of VAP's global epidemiological characteristics and enhance prevention and control strategies."[6]

In study by Rao S et al., (2021) to assess the "incidence, determinants and outcome of VAP in medical intensive care. in 166 patients in a medical ICU who were getting mechanical ventilation were observed. For 1000 days of mechanical ventilation, there were 43.5 cases of VAP in the current research. Organ failure, emergency intubation, reintubation, and COPD are risk factors that were found to be significant in the research. Acinetobacter (30%), Klebsiella pneumoniae (27.1%), and Pseudomonas aeruginosa (20%) were the most prevalent pathogens linked to VAP. Compared to the non-VAP group (15.7%), the mortality was greater in the VAP group (31.3%). The incidence of ventilator-associated pneumonia (VAP) is notably high in developing countries. In a recent study, several risk factors were identified as being associated with VAP, including the presence of chronic obstructive pulmonary disease (COPD), reintubation, organ failure, and emergency intubation. VAP is linked to significantly longer hospital stays, increased morbidity, and higher mortality rates,

highlighting the importance of early detection and management in reducing these adverse outcomes."[46]

AIMS & OBJECTIVES

Objective

1. To figure out how common VAP is in medical ICUs.
2. To identify the risk factors in patients with VAP and to compare with those without VAP.
3. To identify the organisms causing VAP.
4. To evaluate the individuals with VAP's clinical results and compare with those without VAP.

MATERIAL & METHOD

Type of study: Prospective observational study.

Inclusion Criteria:

- Mechanically ventilated patients for more than 48 hours in medical ICUs'.
- Patients aged more than 18 years.

Exclusion Criteria

- Prior to or within 48 hours of mechanical breathing, patients with pneumonia.
- Presence of a previously established permanent artificial airway.
- Patients intubated outside our hospital.

Sample size

With anticipated Proportion of ventilator associated pneumonia 43.5%,[9] the study would require a sample size of 175 subjects with 95% level of confidence and 3% absolute precision.

Formula used

$$n=z^2p*q/d^2$$

Where Z= Z statistic at α level of significance

d^2 = Absolute error

P= Proportion rate

q= 100-p

Investigations:

After applying inclusion and exclusion criteria, a randomly selected group of patients underwent detailed history, clinical examination and following set of investigations.

- Endotracheal tube culture
- Chest X ray
- Complete blood count

STATISTICAL ANALYSIS

The data obtained were entered in a Microsoft Excel sheet, and statistical analysis was performed using statistical package for the social sciences (SPSS Verson 20). Results were presented as Mean (Median) ±SD, Inter quartile range counts and percentages and diagrams. For normally distributed continuous variables was compared using Independent t test. For not normally distributed variables Mann Whitney U test was used. Association between Categorical variables was compared using Chi square test. Regression analysis used to find risk factors. (If necessary). A $p < 0.05$ was considered statistically significant.

RESULTS

Present study included total of 175 patients with fulfilling inclusion criteria, with mean age of 48.71yrs.

Table 2: Showing the mean age of patients

	N	Minimum	Maximum	Mean	SD
Age	175	18.0	85.0	48.714	18.16

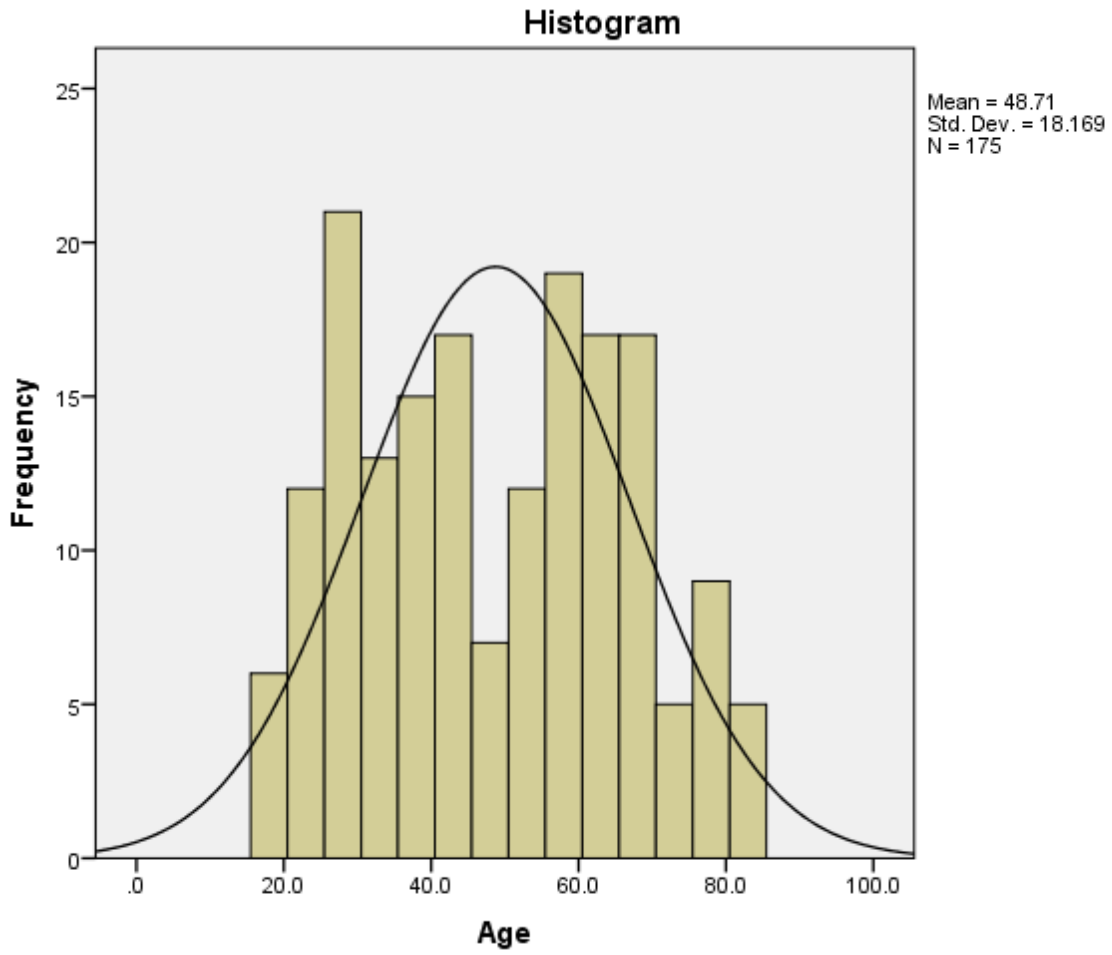

Figure 5: Showing the mean age of patients

Table 3: Gender distribution

		Count	N %
Gender	Female	51	29.1%
	Male	124	70.9%

Among the patients 70.9% were male and 29.1% were female with male preponderance in the study.

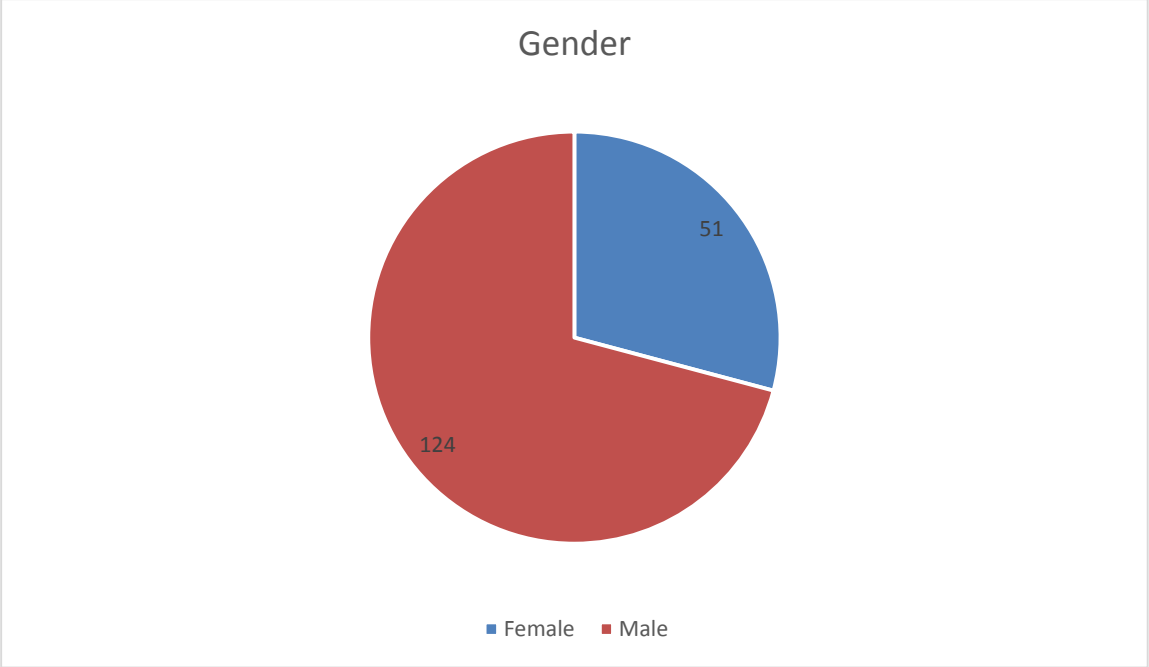

Figure 6: Gender distribution

Table 4: Duration of the ventilator and hospital stay

	Mean	SD
Duration of MV	11.1	9.4
Duration of onset VAP	6.3	4.5
Duration of MV after VAP	5.8	7.8
Duration of hospital stay	15.3	13.6

The mean duration of mechanical ventilation was for 11.1days, duration of onset of VAP was 6.3days, the duration of MV after VAP was 5.8days. the overall mean duration of hospital stay among patients was 15.3%.

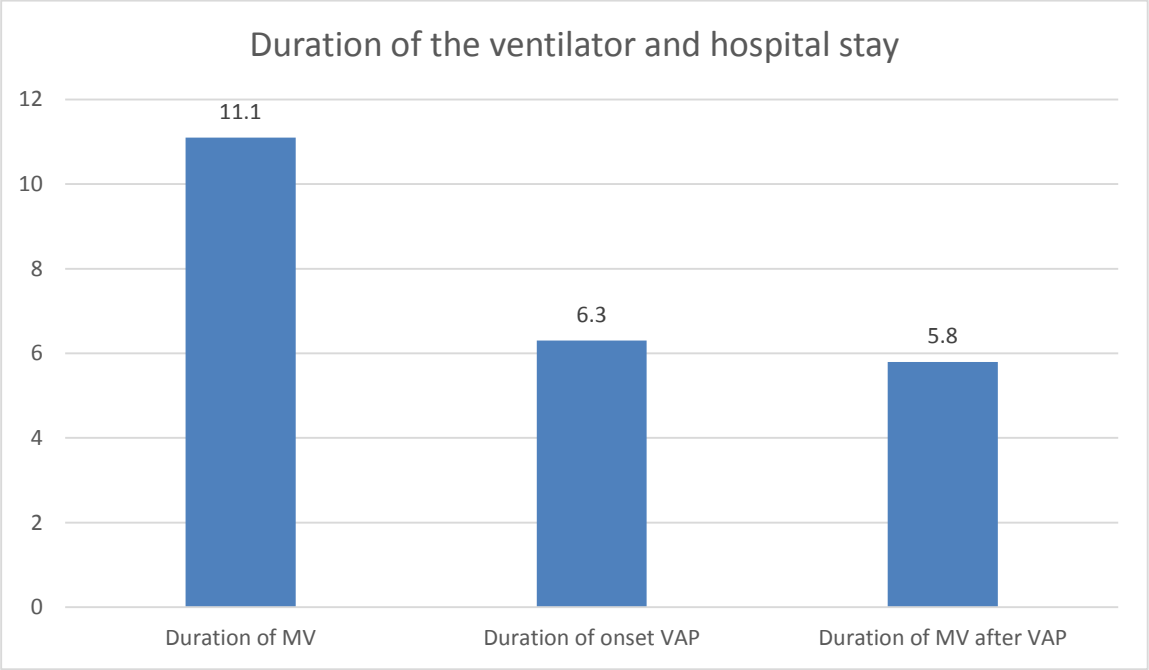

Figure 7: Duration of the ventilator and hospital stay

Table 5: Showing the type of VAP

		Count	N %
Type of VAP	Early onset	53	30.3%
	Late onset	77	44.0%
	No VAP	45	25.7%

VAP was not needed in 25.7%, other were with 44% late onset type of VAP and 30.3% with early onset VAP.

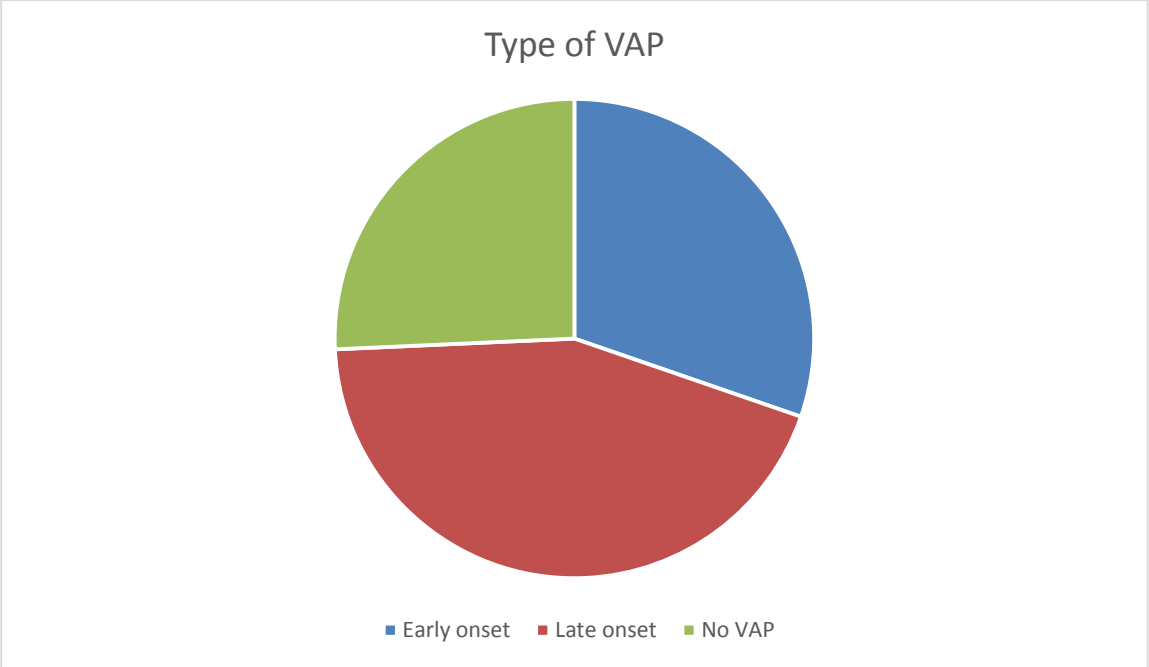

Figure 8: Showing the type of VAP

Table 6: Showing presence of VAP among patients

		Count	N %
VAP	No VAP	45	25.7%
	VAP	130	74.3%

VAP was present in 74.3% cases and not on VAP were 25.7% of the cases.

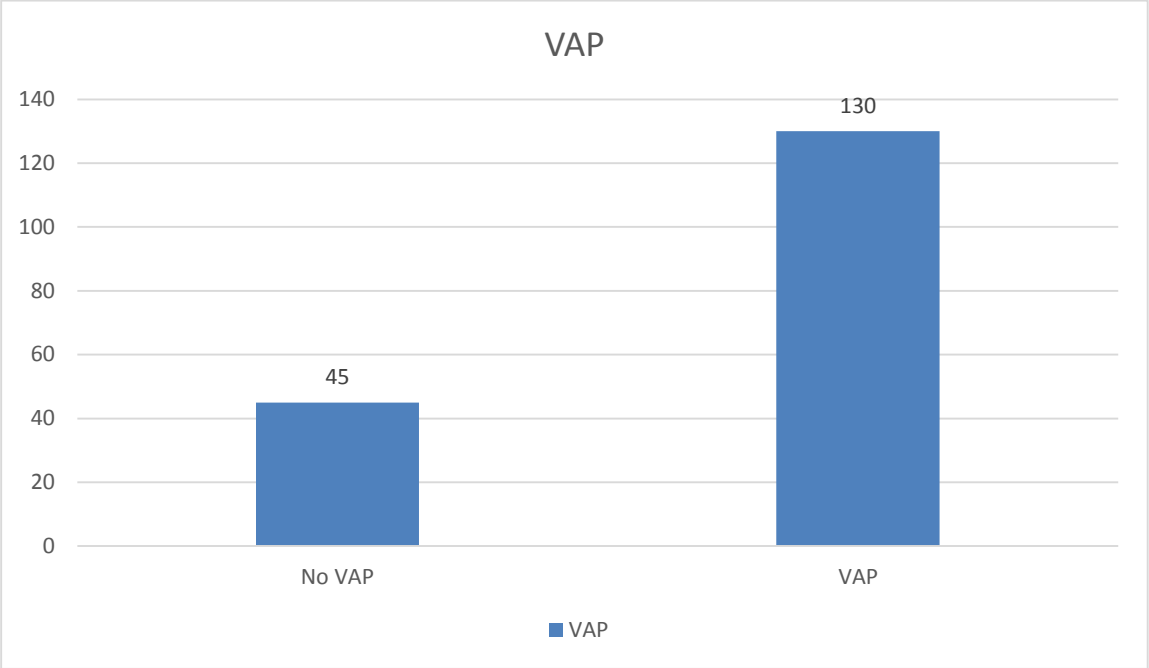

Figure 9: Showing presence of VAP among patients

Table 7: Showing the outcome of patients

		Count	N %
Outcome	DAMA	55	31.4%
	Recovered	60	34.3%
	Worsened	60	34.3%

Among the patients, 34.3% worsened in their condition and 34.3% recovered, and 31.4% were discharge against medical advice.

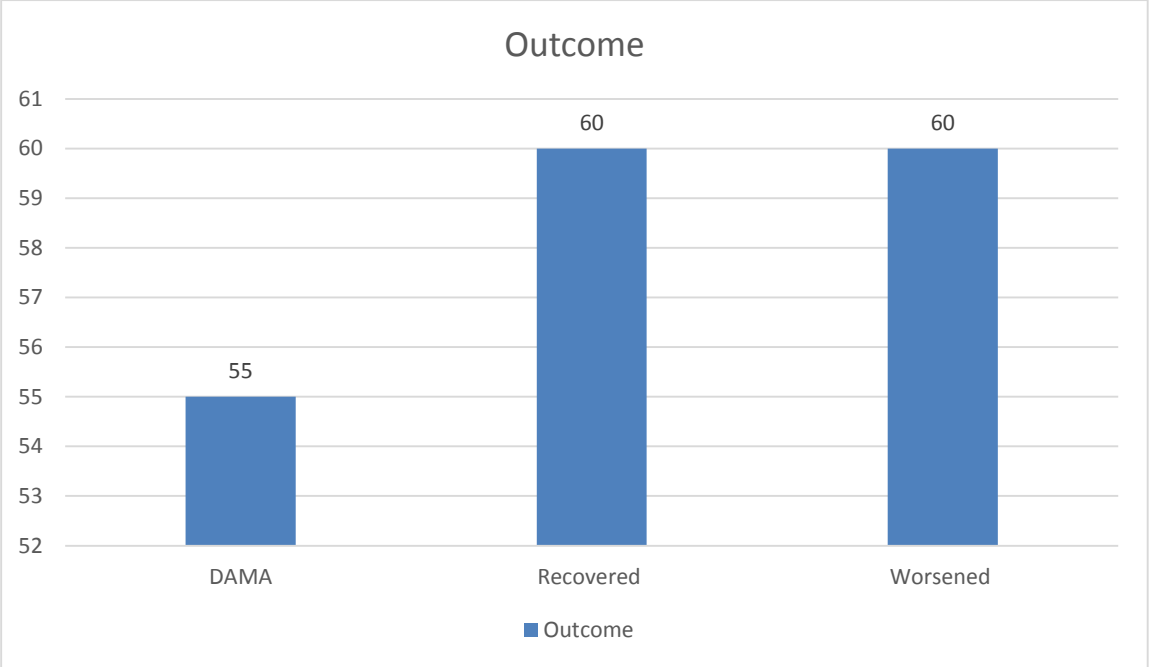

Figure 10: Showing the outcome of patients

Table 8: Showing the cause of MV

		Count	N %
Cause of MV	Airway protection	107	61.1%
	Type 1	26	14.9%
	Type 2	42	24.0%

The most common cause of MV was airway protection (61.1%) followed by 24% with type 2 and 14.9% with type 1.

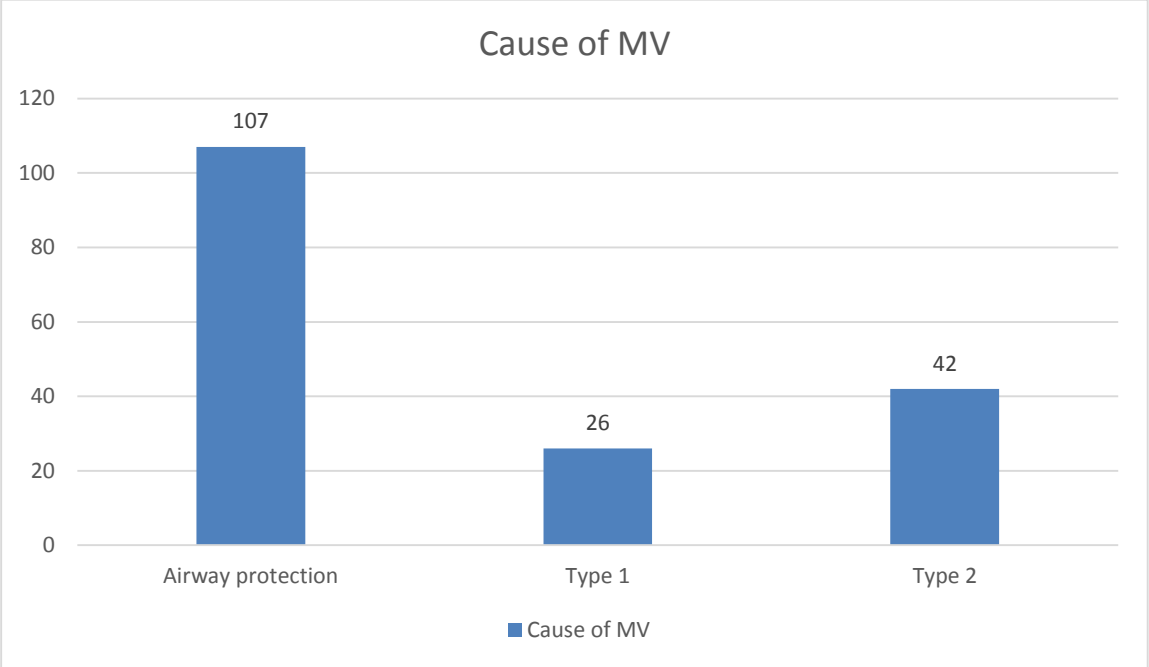

Figure 11: Showing the cause of MV

Table 9: Showing the frequency of risk factors among patients

		Count	N %
Age >60years	No	123	70.3%
	Yes	52	29.7%
Impaired Consiousness	No	62	35.4%
	Yes	113	64.6%
COPD	No	163	93.1%
	Yes	12	6.9%
Diabetes Mellitus	No	139	79.4%
	Yes	36	20.6%
Alcoholism	No	108	61.7%
	Yes	67	38.3%
Smoking	No	116	66.3%
	Yes	59	33.7%
MVgt7days	No	73	41.7%
	Yes	102	58.3%
Immunosupressive therapy	No	118	67.4%
	Yes	57	32.6%
Organ Failure	No	89	50.9%
	Yes	86	49.1%
Reintubation	No	163	93.1%
	Yes	12	6.9%
Emergency intubation	No	55	31.4%
	Yes	120	68.6%
Tracheostomy	No	152	86.9%
	Yes	23	13.1%

The most common risk factors identified were emergency intubation (68.6%), impaired consciousness (64.6%), and mechanical ventilation for more than 7 days (58.3%), all of which are critical contributors to respiratory complications. Additionally, organ failure (49.1%), alcoholism (38.3%), and immunosuppressive therapy (32.6%) were also notable risk factors. Smoking (33.7%), diabetes mellitus

(20.6%), and tracheostomy (13.1%) were present in a smaller proportion of cases. Less frequent but significant factors included COPD (6.9%) and reintubation (6.9%), indicating that while these conditions were less common, they could still play a role in disease severity. The data highlights the importance of monitoring high-risk patients, particularly those requiring prolonged ventilation, emergency intubation, or with altered consciousness, to prevent complications and improve outcomes.

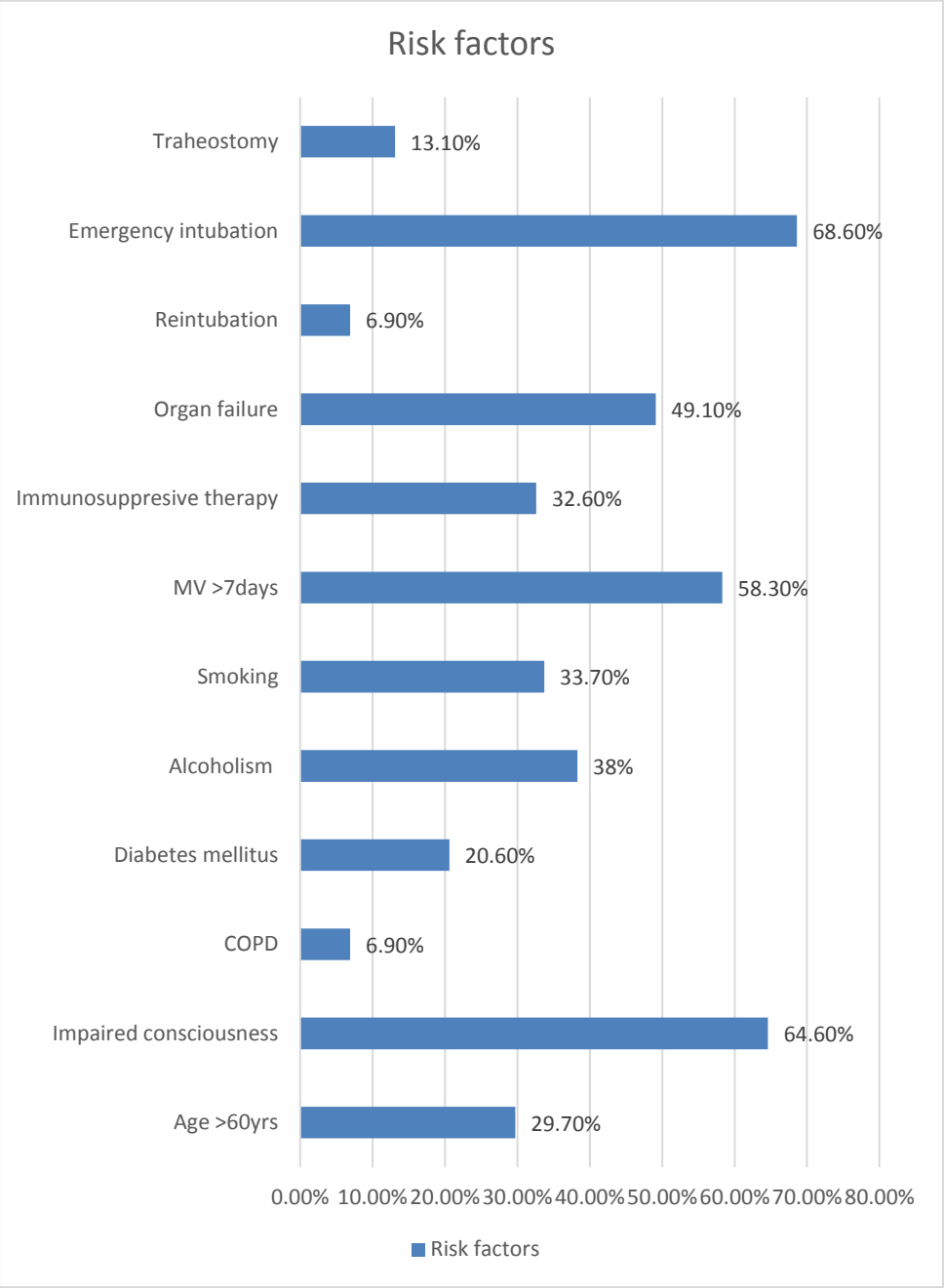

Figure 12: Showing the frequency of risk factors among patients

Table 10: Showing the distribution of ET culture report among patients

		Count	N %
ET culture	Nil	45	25.7%
	Acinetobacter baumannii (MDR)	3	1.7%
	Acinetobacter baumannii complex	35	20.0%
	Acinetobacter spp	2	1.1%
	Citrobacter freundii	4	2.3%
	citrobacter koseri	1	0.6%
	Enterobacter aerogenes	2	1.1%
	Escherichia coli	4	2.3%
	Escherichia coli (CRE)	3	1.7%
	Klebsiella oxytoca	3	1.7%
	Klebsiella pneumoniae	35	20.0%
	Klebsiella pneumoniae ssp pneumoniae (MDRO)	11	6.3%
	Pseudomonas aeruginosa	12	6.9%
	Pseudomonas aeruginosa(MDR)	2	1.1%
	Serratia marcescens	3	1.7%
	Staphylococcus aureus	5	2.9%
	Staphylococcus aureus (MRSA)	5	2.9%

The most commonly isolated organisms from the ET cultures were Acinetobacter baumannii complex and Klebsiella pneumoniae, each accounting for 20.0% (35 isolates). Among the Klebsiella pneumoniae isolates, 6.3% (11 isolates) were classified as multidrug-resistant organisms (MDRO), highlighting concerns regarding antimicrobial resistance. Additionally, Acinetobacter baumannii (MDR) was detected in 1.7% (3 isolates), indicating a smaller but significant presence of multidrug-resistant strains. Other frequently identified organisms included Pseudomonas aeruginosa (6.9%, 12 isolates), with 1.1% (2 isolates) being multidrug-resistant (MDR), and Citrobacter freundii and Escherichia coli, each contributing 2.3% (4 isolates). Notably, Escherichia coli (CRE), a carbapenem-resistant strain, was

found in 1.7% (3 isolates), signaling a potential challenge for treatment. Klebsiella oxytoca, Serratia marcescens, and Enterobacter aerogenes were detected in smaller proportions, ranging from 1.1% to 1.7%. The high prevalence of Acinetobacter and Klebsiella species, especially their resistant variants, underscores the need for strict infection control measures and targeted antimicrobial stewardship programs to curb the spread of resistant pathogens.

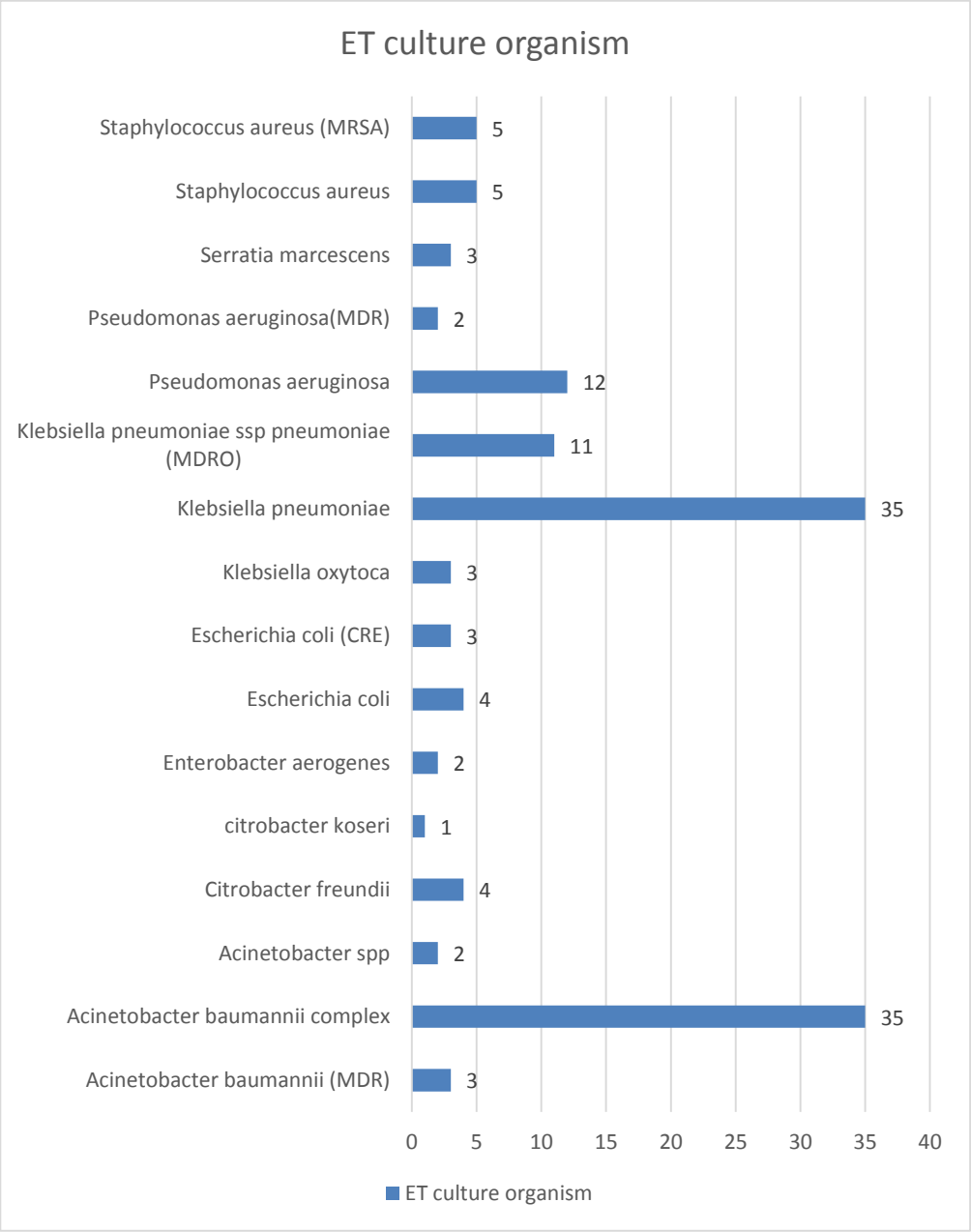

Figure 13: Showing the distribution of ET culture report among patients

Table 11: Showing the sensitivity and resistance pattern of organism

		Count	N %
Amoxicillin Clavulanic acid	R	48	81.3%
	S	11	18.7%
Piperacillin/Tazobactam	R	105	89.8%
	S	12	10.2%
Ceftriaxone	R	104	94.6%
	S	6	5.4%
Cefoperazone/Sulbactam	R	33	46.5%
	S	38	53.5%
Imipenem	R	88	80.0%
	S	22	20.0%
Meropenem	R	98	80.4%
	S	24	19.6%
Amikacin	R	81	67.5%
	S	39	32.5%
Gentamicin	R	35	53.5%
	S	33	46.5%
Ciprofloxacin	R	105	87.5%
	S	15	12.5%
Tigecycline	R	3	2.6%
	S	111	97.4%
Trimethopriim Sulfamethoxazole	R	70	61.5%
	S	44	38.5%
Levofloxacin	R	100	88.5%
	S	13	11.5%

The most common resistance pattern observed in the data is against Ceftriaxone, with 94.6% (104 isolates) showing resistance and only 5.4% (6 isolates) being sensitive. Similarly, Piperacillin/Tazobactam (89.8%), Levofloxacin (88.5%), and Ciprofloxacin (87.5%) exhibit high resistance rates, indicating significant antimicrobial resistance among the isolates tested. Carbapenems, including Imipenem

(80.0%) and Meropenem (80.4%), also show high resistance rates, which is concerning given their use in treating multidrug-resistant infections. On the other hand, the most common sensitive pattern is seen with Tigecycline, where 97.4% (111 isolates) remain susceptible, indicating its potential efficacy against these resistant strains. Cefoperazone/Sulbactam shows a nearly balanced resistance and sensitivity profile, with 53.5% of isolates being sensitive. Amikacin (32.5% sensitivity) and Trimethoprim/Sulfamethoxazole (38.5% sensitivity) also demonstrate some degree of effectiveness but have notable resistance rates. Overall, the data highlights a high prevalence of resistance to commonly used antibiotics, particularly cephalosporins, fluoroquinolones, and carbapenems, while Tigecycline remains highly effective. This underscores the need for judicious antibiotic use and antimicrobial stewardship programs to combat resistance trends.

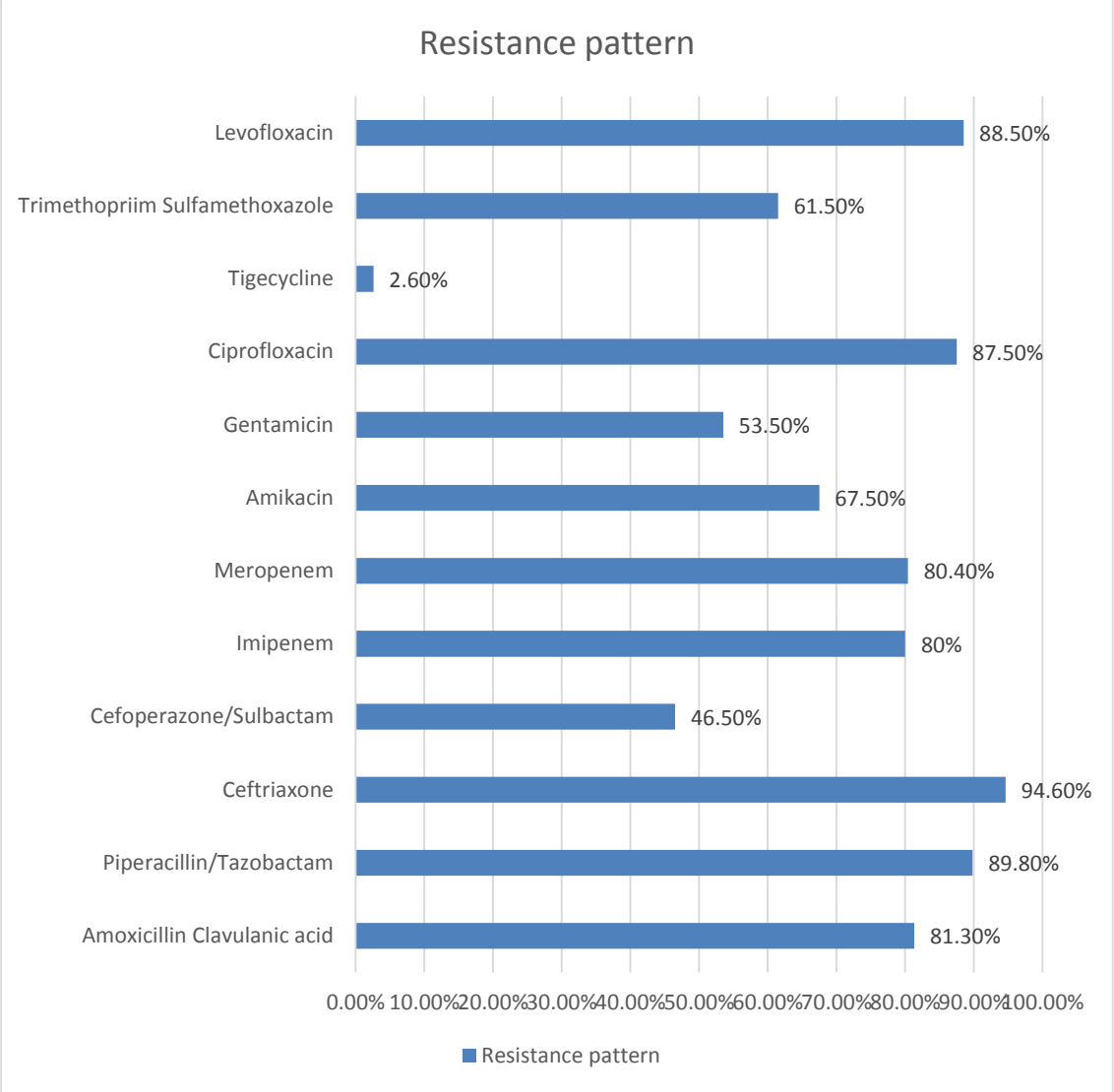

Figure 14: Showing the sensitivity and resistance pattern of organism

Table 12: Association of outcome with VAP among patients

		No VAP		VAP		Chi-square (p-value)
		Count	N %	Count	N %	
Outcome	DAMA	16	35.6%	39	30.0%	0.522 (0.77)
	Recovered	15	33.3%	45	34.6%	
	Worsened	14	31.1%	46	35.4%	

There is no significant difference noted in the VAP requirement with outcome of the patient.

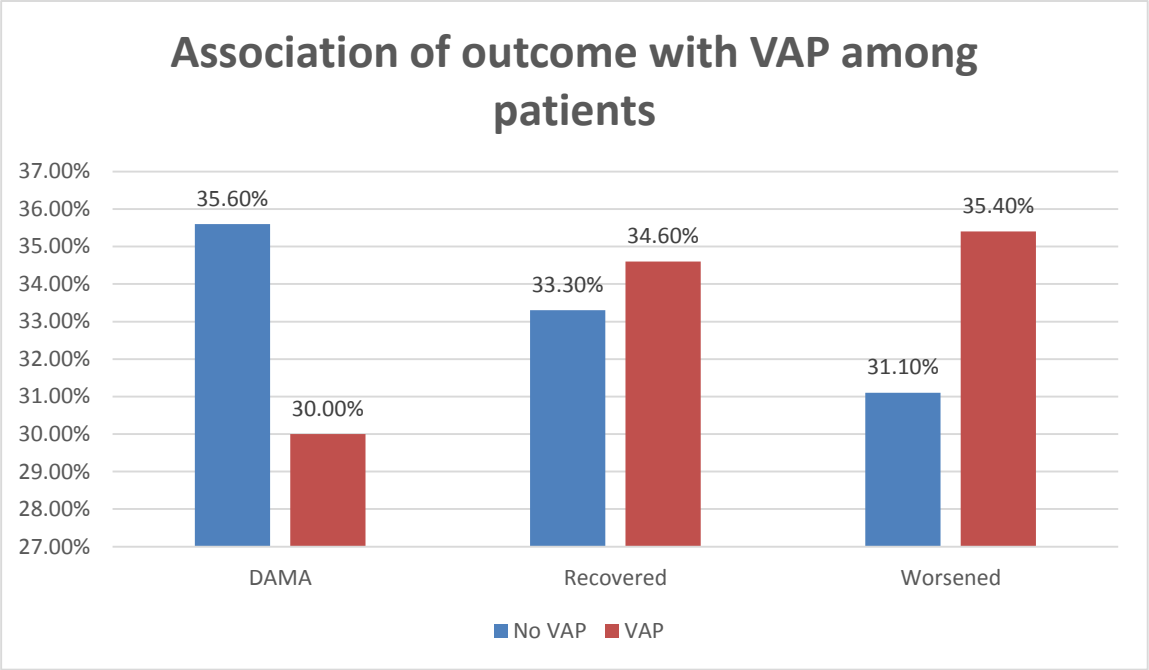

Figure 15: Association of outcome with VAP among patients

Table 13: Association of the risk factors with VAP among patients

		No VAP		VAP		Chi-square (p-value)
		Count	N %	Count	N %	
Age >60years	No	33	73.3%	90	69.2%	0.26 (0.60)
	Yes	12	26.7%	40	30.8%	
Impaired Consciousness	No	19	42.2%	43	33.1%	1.22 (0.26)
	Yes	26	57.8%	87	66.9%	
COPD	No	40	88.9%	123	94.6%	1.71 (0.19)
	Yes	5	11.1%	7	5.4%	
Diabetes Mellitus	No	38	84.4%	101	77.7%	0.93 (0.33)
	Yes	7	15.6%	29	22.3%	
Alcoholism	No	27	60.0%	81	62.3%	0.075 (0.78)
	Yes	18	40.0%	49	37.7%	
Smoking	No	30	66.7%	86	66.2%	0.004 (0.95)
	Yes	15	33.3%	44	33.8%	
MV >7days	No	24	53.3%	49	37.7%	3.36 (0.05)*
	Yes	21	46.7%	81	62.3%	
Immunosupressive therapy	No	34	75.6%	84	64.6%	1.82 (0.177)
	Yes	11	24.4%	46	35.4%	
Organ Failure	No	24	53.3%	65	50.0%	0.14 (0.7)
	Yes	21	46.7%	65	50.0%	
Reintubation	No	43	95.6%	120	92.3%	0.55 (0.45)
	Yes	2	4.4%	10	7.7%	
Emergency intubation	No	20	44.4%	35	26.9%	4.72 (0.02)*
	Yes	25	55.6%	95	73.1%	
Tracheostomy	No	43	95.6%	109	83.8%	4.01 (0.01)*
	Yes	2	4.4%	21	16.2%	

On association of various risk factors with VAP, there is significant higher incidence of VAP among the cases with MV >7days, emergency intubation and

tracheostomy.($p<0.05$) Other risk factors with higher incidence of VAP were the presence of diabetes mellitus, immunosuppressive therapy, and reintubation.

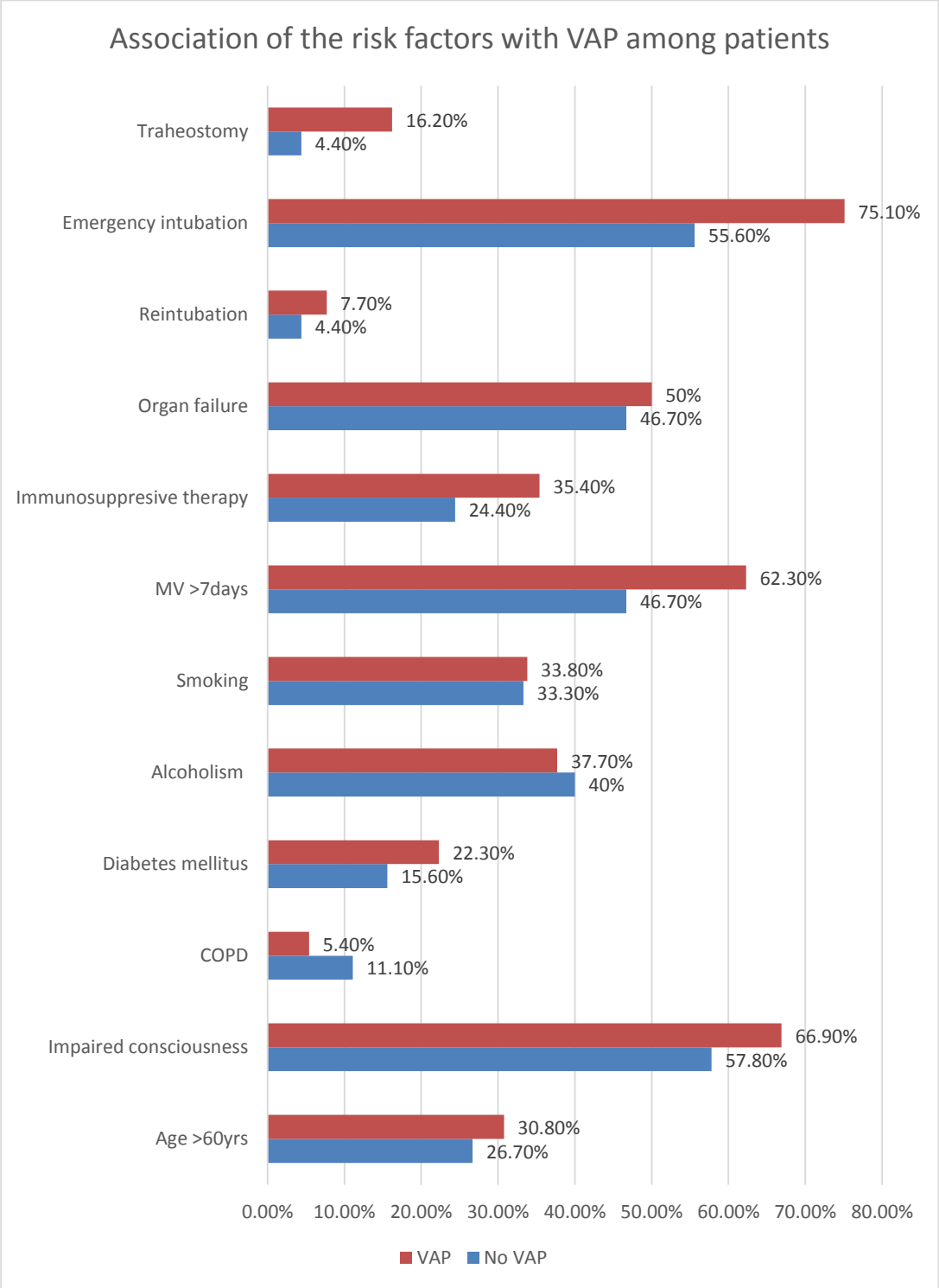

Figure 16: Association of the risk factors with VAP among patients

DISCUSSION

Ventilator-associated pneumonia (VAP) remains a significant challenge in intensive care units (ICUs), contributing to increased morbidity, mortality, prolonged hospital stays, and higher healthcare costs. It is a subtype of hospital-acquired pneumonia that occurs after 48 hours of mechanical ventilation and is associated with a high incidence in critically ill patients. The pathogenesis of VAP is multifactorial, primarily involving microaspiration of oropharyngeal secretions, biofilm formation in endotracheal tubes, and impaired host defenses. Early identification of risk factors and effective preventive strategies are crucial to reducing the burden of VAP and improving patient outcomes. This study aimed to evaluate the prevalence, risk factors, microbial profile, and outcomes of VAP in critically ill patients

Present study included total of 175 patients with fulfilling inclusion criteria, with mean age of 48.71yrs, 70.9% male and 29.1% female patients (male preponderance). The mean duration of mechanical ventilation was for 11.1days, duration of onset of VAP was 6.3days, the duration of MV after VAP was 5.8days. the overall mean duration of hospital stay among patients was 15.3%. VAP was present in 74.3% cases and not on VAP were 25.7% of the cases. VAP was not needed in 25.7%, other were with 44% late onset type of VAP and 30.3% with early onset VAP.

In similar stud by Safdar N et al., findings show that “10-20% of patients on mechanical ventilation for over 48 hours develop ventilator-associated pneumonia (VAP), which significantly increases the risk of death, with critically ill patients being twice as likely to die. VAP is common in ventilated patients and is linked to higher morbidity, mortality, and financial burden, highlighting the urgent need for effective prevention strategies.”[40]. Another study by Hugonnet S et al., of the “2,470 ICU

patients, 262 episodes of ventilator-associated pneumonia (VAP) were diagnosed, with 22.3% of mechanically ventilated patients developing VAP. The median duration of mechanical ventilation was 3 days for patients without VAP and 11 days for those with VAP, with late-onset VAP accounting for 61% of cases." [41]

Saied W et al., found that VAP occurred in 15% of at-risk patients (1,161 patients), while ICU-HAP affected 2% (176 patients). After adjusting for prognostic factors, both VAP (hazard ratio 1.38) and ICU-HAP (hazard ratio 1.82) were linked to a significant increase in 30-day mortality. [45]

The most common risk factors identified were emergency intubation (68.6%), impaired consciousness (64.6%), and mechanical ventilation for more than 7 days (58.3%), all of which are critical contributors to respiratory complications. Additionally, organ failure (49.1%), alcoholism (38.3%), and immunosuppressive therapy (32.6%) were also notable risk factors. Smoking (33.7%), diabetes mellitus (20.6%), and tracheostomy (13.1%) were present in a smaller proportion of cases. Less frequent but significant factors included COPD (6.9%) and reintubation (6.9%), indicating that while these conditions were less common, they could still play a role in disease severity. The data highlights the importance of monitoring high-risk patients, particularly those requiring prolonged ventilation, emergency intubation, or with altered consciousness, to prevent complications and improve outcomes.

In study by Rao S et al., the Organ failure, emergency intubation, reintubation, and COPD are risk factors that were found to be significant. [46]

The most commonly isolated organisms from the ET cultures were Acinetobacter baumannii complex and Klebsiella pneumoniae, each accounting for 20.0% (35 isolates). Among the Klebsiella pneumoniae isolates, 6.3% (11 isolates) were classified as multidrug-resistant organisms (MDRO), highlighting concerns

regarding antimicrobial resistance. Additionally, Acinetobacter baumannii (MDR) was detected in 1.7% (3 isolates), indicating a smaller but significant presence of multidrug-resistant strains. Other frequently identified organisms included Pseudomonas aeruginosa (6.9%, 12 isolates), with 1.1% (2 isolates) being multidrug-resistant (MDR), and Citrobacter freundii and Escherichia coli, each contributing 2.3% (4 isolates). Notably, Escherichia coli (CRE), a carbapenem-resistant strain, was found in 1.7% (3 isolates), signaling a potential challenge for treatment. Klebsiella oxytoca, Serratia marcescens, and Enterobacter aerogenes were detected in smaller proportions, ranging from 1.1% to 1.7%. The high prevalence of Acinetobacter and Klebsiella species, especially their resistant variants, underscores the need for strict infection control measures and targeted antimicrobial stewardship programs to curb the spread of resistant pathogens.

In similar study by Inchai J et al., the primary pathogens were *Acinetobacter baumannii* (54.3%), *Pseudomonas aeruginosa* (35.2%), and methicillin-resistant *Staphylococcus aureus* (15.1%). Most *A. baumannii* strains were drug-resistant (90.2%).[43] Walaszek MZ et al., found most common pathogens identified were Acinetobacter baumannii (36.4%), Pseudomonas aeruginosa (13.8%), and Escherichia coli (12%).”[44]

Rao S et al., documented that Acinetobacter (30%), Klebsiella pneumoniae (27.1%), and Pseudomonas aeruginosa (20%) were the most prevalent pathogens linked to VAP. Compared to the non-VAP group (15.7%), the mortality was greater in the VAP group (31.3%).[46]

The most common resistance pattern observed in the data is against Ceftriaxone, with 94.6% (104 isolates) showing resistance and only 5.4% (6 isolates) being sensitive. Similarly, Piperacillin/Tazobactam (89.8%), Levofloxacin (88.5%),

and Ciprofloxacin (87.5%) exhibit high resistance rates, indicating significant antimicrobial resistance among the isolates tested. Carbapenems, including Imipenem (80.0%) and Meropenem (80.4%), also show high resistance rates, which is concerning given their use in treating multidrug-resistant infections. On the other hand, the most common sensitive pattern is seen with Tigecycline, where 97.4% (111 isolates) remain susceptible, indicating its potential efficacy against these resistant strains. Cefoperazone/Sulbactam shows a nearly balanced resistance and sensitivity profile, with 53.5% of isolates being sensitive. Amikacin (32.5% sensitivity) and Trimethoprim/Sulfamethoxazole (38.5% sensitivity) also demonstrate some degree of effectiveness but have notable resistance rates. Overall, the data highlights a high prevalence of resistance to commonly used antibiotics, particularly cephalosporins, fluoroquinolones, and carbapenems, while Tigecycline remains highly effective. This underscores the need for judicious antibiotic use and antimicrobial stewardship programs to combat resistance trends.

On association of various risk factors with VAP, there is significant higher incidence of VAP among the cases with MV >7days, emergency intubation and tracheostomy.($p<0.05$) Other risk factors with higher incidence of VAP were the presence of diabetes mellitus, immunosuppressive therapy, and reintubation.

Bouadma L et al., found that the Nosocomial infections, including ventilator-associated pneumonia (VAP), were the leading causes of both conditions, accounting for 27.3% and 43.8% of cases, respectively. The sensitivity and specificity for diagnosing VAP were 0.92 and 0.28 for ventilator-associated conditions, and 0.67 and 0.75 for infection-related ventilator-associated complications. Strong correlations were found between ventilator-associated conditions, infection-related ventilator-associated complications, and VAP occurrence ($R2 = 0.69$ and 0.82). Patients without

any ventilator-associated events had a significantly higher median number of days alive without antibiotics and mechanical ventilation by day 28. Rates of ventilator-associated events were closely associated with antibiotic use within each ICU (R2 = 0.987 and 0.99). These events are common among at-risk populations and are closely linked to antibiotic consumption, suggesting they could serve as a quality indicator for improvement programs.[42] Another study by Rao S et al., the "several risk factors were identified as being associated with VAP, including the presence of chronic obstructive pulmonary disease (COPD), reintubation, organ failure, and emergency intubation. VAP is linked to significantly longer hospital stays, increased morbidity, and higher mortality rates, highlighting the importance of early detection and management in reducing these adverse outcomes."[46]

Recommendations

Based on the findings of this study on ventilator-associated pneumonia (VAP), its risk factors, and outcomes, the following recommendations are proposed to reduce VAP incidence, improve patient outcomes, and address antimicrobial resistance in ICU settings:

1. Prevention and Early Detection of VAP

- Implement strict **ventilator care bundles**, including head-of-bed elevation (30–45 degrees), daily sedation vacations, and early assessment for extubation.
- Minimize **unnecessary mechanical ventilation** by promoting early weaning protocols and non-invasive ventilation when feasible.
- Avoid **emergency intubation whenever possible** by ensuring timely elective intubation under controlled conditions.
- Conduct regular **oral hygiene with chlorhexidine** to reduce oropharyngeal colonization and lower the risk of microaspiration.

2. Identifying and Managing High-Risk Patients

- Closely monitor patients with **emergency intubation, prolonged mechanical ventilation (>7 days), tracheostomy, diabetes mellitus, and immunosuppressive therapy**, as they are at a higher risk of developing VAP.
- Implement early **risk stratification protocols** for ICU patients to identify those needing enhanced monitoring and preventive measures.
- Ensure **appropriate sedation management** and spontaneous breathing trials to reduce unnecessary ventilator dependency.

3. Infection Control and Antimicrobial Stewardship

- Strengthen **infection control practices**, including hand hygiene, sterilization of respiratory equipment, and strict adherence to aseptic techniques during suctioning and intubation.
- Implement **antimicrobial stewardship programs** to prevent the emergence of multidrug-resistant organisms (MDROs) by optimizing antibiotic prescribing practices.
- Conduct **routine microbial surveillance** to monitor prevalent organisms and resistance patterns to guide empirical therapy.
- Encourage **de-escalation of antibiotics** based on culture reports to avoid unnecessary use of broad-spectrum antibiotics.

4. Addressing Antimicrobial Resistance

- Given the **high resistance rates** to cephalosporins, fluoroquinolones, and carbapenems, the use of these antibiotics should be restricted and **guided by culture sensitivity results**.
- Promote the use of **Tigecycline and Cefoperazone/Sulbactam**, which showed better sensitivity patterns in this study, for treating MDRO infections.

- Encourage research and implementation of **alternative treatment strategies**, including combination therapy, to manage resistant infections effectively.

5. Enhancing ICU Protocols and Staff Training

- Conduct **regular training programs** for ICU staff on ventilator-associated complications, VAP prevention strategies, and infection control measures.
- Encourage **multidisciplinary collaboration** among intensivists, infectious disease specialists, respiratory therapists, and nursing staff to optimize patient management.
- Establish **audit and feedback systems** to evaluate adherence to VAP prevention protocols and improve compliance.

By implementing these recommendations, ICU teams can reduce the burden of VAP, improve patient outcomes, and mitigate the challenge of antimicrobial resistance.

SUMMARY

Present study included total of 175 patients with fulfilling inclusion criteria, with mean age of 48.71yrs.

Among the patients 70.9% were male and 29.1% were female with male preponderance in the study.

The mean duration of mechanical ventilation was for 11.1days, duration of onset of VAP was 6.3days, the duration of MV after VAP was 5.8days. the overall mean duration of hospital stay among patients was 15.3%.

VAP was not needed in 25.7%, other were with 44% late onset type of VAP and 30.3% with early onset VAP.

VAP was present in 74.3% cases and not on VAP were 25.7% of the cases.

Among the patients, 34.3% worsened in their condition and 34.3% recovered, and 31.4% were discharge against medical advice.

The most common cause of MV was airway protection (61.1%) followed by 24% with type 2 and 14.9% with type 1.

The most common risk factors identified were emergency intubation (68.6%), impaired consciousness (64.6%), and mechanical ventilation for more than 7 days (58.3%), all of which are critical contributors to respiratory complications. Additionally, organ failure (49.1%), alcoholism (38.3%), and immunosuppressive therapy (32.6%) were also notable risk factors. Smoking (33.7%), diabetes mellitus (20.6%), and tracheostomy (13.1%) were present in a smaller proportion of cases. Less frequent but significant factors included COPD (6.9%) and reintubation (6.9%), indicating that while these conditions were less common, they could still play a role in disease severity. The data highlights the importance of monitoring high-risk patients,

particularly those requiring prolonged ventilation, emergency intubation, or with altered consciousness, to prevent complications and improve outcomes.

The most commonly isolated organisms from the ET cultures were Acinetobacter baumannii complex and Klebsiella pneumoniae, each accounting for 20.0% (35 isolates). Among the Klebsiella pneumoniae isolates, 6.3% (11 isolates) were classified as multidrug-resistant organisms (MDRO), highlighting concerns regarding antimicrobial resistance. Additionally, Acinetobacter baumannii (MDR) was detected in 1.7% (3 isolates), indicating a smaller but significant presence of multidrug-resistant strains. Other frequently identified organisms included Pseudomonas aeruginosa (6.9%, 12 isolates), with 1.1% (2 isolates) being multidrug-resistant (MDR), and Citrobacter freundii and Escherichia coli, each contributing 2.3% (4 isolates). Notably, Escherichia coli (CRE), a carbapenem-resistant strain, was found in 1.7% (3 isolates), signaling a potential challenge for treatment. Klebsiella oxytoca, Serratia marcescens, and Enterobacter aerogenes were detected in smaller proportions, ranging from 1.1% to 1.7%. The high prevalence of Acinetobacter and Klebsiella species, especially their resistant variants, underscores the need for strict infection control measures and targeted antimicrobial stewardship programs to curb the spread of resistant pathogens.

The most common resistance pattern observed in the data is against Ceftriaxone, with 94.6% (104 isolates) showing resistance and only 5.4% (6 isolates) being sensitive. Similarly, Piperacillin/Tazobactam (89.8%), Levofloxacin (88.5%), and Ciprofloxacin (87.5%) exhibit high resistance rates, indicating significant antimicrobial resistance among the isolates tested. Carbapenems, including Imipenem (80.0%) and Meropenem (80.4%), also show high resistance rates, which is concerning given their use in treating multidrug-resistant infections. On the other

hand, the most common sensitive pattern is seen with Tigecycline, where 97.4% (111 isolates) remain susceptible, indicating its potential efficacy against these resistant strains. Cefoperazone/Sulbactam shows a nearly balanced resistance and sensitivity profile, with 53.5% of isolates being sensitive. Amikacin (32.5% sensitivity) and Trimethoprim/Sulfamethoxazole (38.5% sensitivity) also demonstrate some degree of effectiveness but have notable resistance rates. Overall, the data highlights a high prevalence of resistance to commonly used antibiotics, particularly cephalosporins, fluoroquinolones, and carbapenems, while Tigecycline remains highly effective. This underscores the need for judicious antibiotic use and antimicrobial stewardship programs to combat resistance trends.

There is no significant difference noted in the VAP requirement with outcome of the patient.

On association of various risk factors with VAP, there is significant higher incidence of VAP among the cases with MV >7days, emergency intubation and tracheostomy.($p<0.05$) Other risk factors with higher incidence of VAP were the presence of diabetes mellitus, immunosuppressive therapy, and reintubation.

CONCLUSION

The present study highlights the significant burden of ventilator-associated pneumonia (VAP) in critically ill patients requiring mechanical ventilation. Among the 175 patients included, VAP was present in 74.3% of cases, with a higher prevalence of late-onset VAP (44%). The study identified key risk factors contributing to the development of VAP, including prolonged mechanical ventilation (>7 days), emergency intubation, impaired consciousness, and tracheostomy, emphasizing the need for close monitoring and preventive measures in high-risk patients.

Microbiological analysis revealed a predominance of *Acinetobacter baumannii* and *Klebsiella pneumoniae*, with a significant proportion of multidrug-resistant (MDR) strains. The study also identified alarming antimicrobial resistance patterns, with high resistance rates to cephalosporins, fluoroquinolones, and carbapenems, while Tigecycline remained the most effective antibiotic. These findings underscore the urgent need for strict infection control measures and antimicrobial stewardship programs to mitigate resistance trends and optimize treatment strategies.

Patient outcomes varied, with 34.3% of cases worsening, 34.3% recovering, and 31.4% being discharged against medical advice. Importantly, no significant difference was observed between VAP presence and patient outcomes, suggesting that other clinical factors may play a role in disease progression. However, the strong association between prolonged mechanical ventilation, emergency intubation, and tracheostomy with VAP incidence ($p<0.05$) reinforces the importance of early preventive strategies.

In conclusion, this study emphasizes the critical role of early identification and management of risk factors, strict infection control protocols, and judicious antibiotic use to improve patient outcomes and curb antimicrobial resistance in ventilated patients. Further research is needed to refine VAP prevention strategies and optimize treatment approaches in critically ill populations.

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ANNEXURE I

Azadi Ka Amrit Mahotsav

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 886/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "A STUDY OF VENTILATOR ASSOCIATED PNEUMONIA –RISK FACTORS AND OUTCOMES".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.GUJJA RAGHAV

NAME OF THE GUIDE: DR.SANJEEVKUMAR N.BENTOOR, PROFESSOR, DEPT. OF GENERAL MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
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ANNEXURE II

CONSENT FORM

BLDEDU'S SHRI B.M.PATIL MEDICALCOLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR- 586103

TITLE OF THE PROJECT - **A STUDY OF VENTILATOR ASSOCIATED PNEUMONIA –RISK FACTORS AND OUTCOMES**

PRINCIPAL INVESTIGATOR - **Dr. GUJJA RAGHAV**
+91 7013785398

P.G. GUIDE NAME - **Dr. SANJEEVKUMAR N. BENTOOR**
PROFFESSOR AND HEAD
DEPARTMENT OF MEDICINE.
08352-, Ext-2148

All aspects of this consent form are explained to the patient in the language understood by him/her.

INFORMED PART PURPOSE OF RESEARCH: I have been informed about this study. I have also been given a free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will help to patient's survival and better outcome.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime. Dr. GUJJA RAGHAV is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. GUJJA RAGHAV may terminate my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Dr. GUJJA RAGHAV Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. GUJJA RAGHAV** has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

_____ _____

Participant / Guardian Date

_____ _____

Witness to signature

B.L.D.E. (DEEMED TO BE UNIVERSITY)

SHRI B.M PATIL MEDICAL COLLEGE VIJAYAPURA,

KARNATAKA

SCHEME OF CASE TAKING

Informant :

Name: CASE NO:

Age: IP NO:

Sex: D.O.A:

Religion: D.O.D:

Past Occupation:

Present Occupation:

Residence:

Chief complaints:

History of present illness:

Past History:

Personal History:

Family History:

Treatment History:

General Physical Examination

Height:

Weight:

Body Mass Index:

Vitals

PR:

B.P.:

R.R.:

Temp:

Head-to-toe examination:

SYSTEMIC EXAMINATION:

GASTRO INTESTINAL SYSTEM:

CENTRAL NERVOUS SYSTEM:

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

DIAGNOSIS AT THE TIME OF ADMISSION:

1. INDICATION FOR MECHANICAL VENTILATION:

2. RISK FACTORS:

AGE >60YEARS,ORGAN FAILURE,ARDS ,CHRONIC LUNG FAILURE, IMPAIRED CONSCIOUSNESS, TRACHEOSTOMY ,SUPINE HEAD POSITION, IMMUNE SUPPRESSIVE THERAPY, EMERGENCY INTUBATION, CHRONIC RENAL FAILURE, REINTUBATION, DURATION OF MECHANICAL VENTILATION>7DAYS

3. EMPIRICAL ANTIBIOTIC THERAPY:

DURATION AND DOSE OF THEIR USE BEFORE ONSET OF VAP

4. ONSET OF VAP: A) EARLY B) LATE

5. CHEST X-RAY FINDINGS:

A) AT THE TIME OF DIAGNOSIS

B) FOLLOW UP X-RAY

6. ENDOTRACHEAL TUBE CULTURE:

A) BACTERIA GROWN

B) SENSITIVITY

7). Ventilatory mode: BIPAP SIMV a) volume control b) pressure control , PSV,PRVC,APRV

a) VENTILATORY SETTINGS (Day 1 after initial settlement)

FiO_2

Rate

PEEP

Pressure support

I: E ratio

b) On the day of diagnosis

8) DURATION OF MECHANICAL VENTILATION:

9) COMPLICATIONS:

10) OUTCOME: RECOVERED/EXPIRED/LOST TO FOLLOW UP

LABS:

TEST NAME	RESULT	UNITS	NORMAL RANGE
HB		g/dl	13-17
PCV		%	36-46
MCV		fl	83-101
MCH		pg	27-32
MCHC		g/dl	32-35
RDW		%	11.6-14
RBC		Million/cumm	3.8-4.8
TC		$10^3/\mu l$	4-10
NEUTROPHILS		%	40-80
LYMPHOCYTES		%	20-40
EOSINOPHILS		%	1-6
MONOCYTES		%	2-10
BASOPHILS		%	0-2
PLATELET COUNT		$10^3/\mu l$	150-410
PCT		%	0.22-0.24
MPV		fl	7.5-12
PDW		fl	10-25
P-LCR		%	15-35

TEST NAME	RESULT	UNITS
PH		
PCO2		mmHg
PO2		mmHg
HCO3		mmol/L
SBC		mmol/L
BEB		mmol/L
BEECF		mmol/L
TCO2		mmol/L
A-ADO2		mmHg
SO2C		%
RI		
FIO2		%
LACTATE		mmol/L

TEST NAME	RESULT	UNITS	NORMAL RANGE
SERUM BILIRUBIN (TOTAL)		mg/dl	Adults: 0.2-1.2 Neonates:<5.5
SERUM BILIRUBIN (CONJUGATED)		mg/dl	Adults:0.0-0.3
SERUM BILIRUBIN (UNCONJUGATED)		mg/dl	Adults:0.0-1.1 Neonates:<5.2
SGPT		U/L	17-59
SGOT		U/L	21-72
SERUM PROTEIN		g/dl	6.3-8.2
SERUM ALBUMIN		g/dl	3.5-5.5
GLOBULIN		g/dl	2-3.5
AG RATIO			0.8-1.2
ALP(ALKALINE PHOSPHATASE)		U/L	38-126

TEST NAME	RESULT	UNIT	NORMAL RANGE
BLOOD UREA		mg/dl	19-43
SERUM CREATININE		mg/dl	0.6-1.1
URIC ACID		mg/dl	4-7
SERUM CALCIUM		mg/dl	8.4-10.2
SERUM PHOSPHORUS		mg/dl	2.5-4.5
SERUM SODIUM		mEq/L	135-145
SERUM POTASSIUM		mEq/L	3.5-5.1
SERUM CHLORIDE		mmol/L	98-107

MASTERCHART

Sl No	UHID No	Name	Age	Sex	Duration of MV	Duration of onset VAP	Duration of MV after VAP	Duration of hospital stay	Type of VAP	Outcome	Diagnosis at the time of admission	Cause of MV	Age>60 years	Impaired Consiousness	COPD	Diabetes Mellitus	Alcoholism	Smoking	MV>7days
1	124904	LAXMAN B BIRADAR	83	M	9	5	3	13	late onset	dama	squamous cell carcinoma of right lung	airway protection	yes	yes	no	no	no	yes	yes
2	138260	nirmala basavaraj naikodi	31	f	10			20	no vap	recovered	P3L2D1 ON POD 2 FOLLLOWING EMERGENCY LSCS(OUTSIDE)	airway protection	no	no	no	no	no	no	no
3	138169	basanna gangappa halagunaki	66	m	8	3	5	8	early onset	worsened	cardiogenic shock/heart failure	airway protection	yes	yes	no	yes	yes	yes	yes
4	132540	shreedevi RAMESH CHALAWADI	31	f	12	5	7	23	late onset	recovered	edh,sdh	airway protection	no	yes	no	no	yes	yes	yes
5	141396	hanamanth ogeppa teli	75	m	9	4	4	13	early onset	recovered	left subtrochanteric fracture with copd with anemia	airway protection	yes	yes	yes	no	no	yes	yes
6	143624	raju k nadaf	55	m	13	8	5	13	late onset	worsened	dka,sepsis	type 1	no	no	no	yes	yes	no	yes
7	149240	umar faruk adavani	42	m	12	6	6	12	late onset	dama	acute ischemic stroke with bilateral pontine infarct	airway protection	no	yes	no	no	no	no	yes
8	150542	ashok n patarotti	41	m	13	7	6	22	late onset	recovered	sdh,edh	airway protection	no	yes	no	no	no	no	yes
9	153028	prashant chandrakanth dalawai	30	m	6	3	3	9	early onset	recovered	altered sensorium	airway protection	no	yes	no	no	no	no	no
10	155557	basavaraj shivakantappa madar	51	m	3	2	1	3	early onset	worsened	dka,pancreatitis,sepsis	type 1	no	yes	no	yes	yes	no	no
11	154343	jetteppa d kachakanur	36	m	7	4	3	7	early onset	worsened	op poisoning,sepsis	type 1	no	yes	no	no	yes	yes	no
12	154347	prakash bhimanna badiger	27	m	4	3	1	4	early onset	worsened	ihd,heart failure	type 1	no	yes	no	no	yes	yes	no
13	34235	rehmatbee khatik	68	f	8	4	4	8	early onset	dama	cardiogenic shock/heart failure	airway protection	yes	yes	no	yes	no	no	yes
14	161931	sheshu basantrao kulkarni	56	m	3	3	0	3	early onset	dama	dka,sepsis	airway protection	no	no	no	yes	yes	no	no
15	167374	sadappa basappa badiger	55	m	14	10	4	14	late onset	worsened	rta,sdh	airway protection	no	yes	no	yes	yes	yes	yes
16	111955	anil mallappa janai	25	m	85	20	65	121	late onset	recovered	sah,sdh	airway protection	no	yes	no	no	yes	no	yes
17	174290	prabhugouda sharanagouda biradar	28	m	10			16	no vap	recovered	rta	airway protection	no	yes	no	no	yes	no	yes
18	175552	bhimappa basappa chigari	50	m	5			9	no vap	recovered	neurotoxic snake bite	airway protection	no	yes	no	no	no	no	no
19	167412	siddu ramanna jidgi	37	m	13			13	no vap	worsened	septic shock,mods	type 2	no	no	no	yes	yes	no	yes
20	189260	narasappa	76	m	3	3	0	3	no vap	worsened	cardiogenic shock/heart failure	type 2	yes	yes	no	yes	yes	yes	no
21	190065	ashok mahadevappa jirli	55	m	6	4	2	6	early onset	dama	cp arrest(reverted) aspirational pneumonitis	airway protection	no	yes	no	no	no	no	no
22	193402	a b kumbar	50	m	21	4	17	21	early onset	dama	cva,ckd,t2dm,htn	airway protection	no	yes	no	yes	yes	yes	yes

23	193930	shivaray ambanna pujari	45	m	8	5	3	13	late onset	recovered	snake bite	airway protection	no	yes	no	no	no	no	yes
24	195239	rahamatbee imamsab bamadenmadu	60	f	15	3	12	23	early onset	recovered	hollow viscous perforation	airway protection	no	no	no	no	no	no	yes
25	196703	shankreppa irasangappa reshami	85	m	13	4	9	13	early onset	dama	ic bleed	airway protection	yes	yes	no	no	no	yes	yes
26	197892	dilip shivappa haladakeri	43	m	10			10	no vap	worsened	septic shock,mods	airway protection	no	yes	no	no	no	yes	yes
27	205229	metabai pulsingh nayak	65	f	20	12	8	20	late onset	dama	cva	airway protection	yes	yes	no	yes	no	no	yes
28	208522	nagawwa sayabanna athanur	80	f	3	1	0	3	early onset	dama	anemia/aki/lrti	type 2	yes	no	yes	yes	no	no	no
29	203854	kashinath shivappa narale	27	m	48	15	33	48	late onset	worsened	septic shock,mods	type 2	no	yes	no	no	yes	no	yes
30	208526	rajendra yankappa ghorpade	41	m	16	5	11	28	late onset	recovered	rta	airway protection	no	no	no	no	yes	no	yes
31	211707	dodappa chandramappa chittapur	57	m	3			6	no vap	dama	op poisoning	type 1	no	no	no	no	no	no	no
32	210887	tarabai hanamant chavan	60	f	14	4	10	21	early onset	recovered	rta	airway protection	no	yes	no	no	no	no	yes
33	213965	maheboob a donur	26	m	5	3	1	5	early onset	dama	rta	airway protection	no	yes	no	no	yes	no	no
34	221196	mehaboob	27	m	5	3	2	5	early onset	dama	rta	airway protection	no	yes	no	no	no	no	no
35	216645	savita balavant kanti	26	f	14	7	7	14	late onset	worsened	meningoencephalitis,sepsis,mods	type 2	no	yes	no	no	no	no	yes
36	227002	mallappa shivappa myageri	38	m	25			35	no vap	recovered	rta	airway protection	no	yes	no	no	yes	no	yes
37	234173	rahul devarmani	25	m	3	3	0	3	early onset	dama	rta,sdh,sepsis,mods	airway protection	no	yes	no	no	yes	yes	no
38	230150	satish shivappa nagaradi	26	m	12	7	5	22	late onset	dama	rta,sdh	airway protection	no	yes	no	no	yes	no	yes
39	239851	siddamma s kakkalameli	71	f	4	3	1	4	early onset	worsened	septic shock,mods	type 2	yes	no	no	no	no	no	no
40	245084	iranna bhimappa talikoti	35	m	12			12	no vap	dama	cld	airway protection	no	yes	no	no	yes	no	yes
41	247358	suresh yallappa hosamani	37	m	4	3	1	4	early onset	dama	dka,left pleural effusion	airway protection	no	yes	no	yes	yes	no	no
42	253384	jaya shrikanth rathod	19	f	4			4	no vap	dama	status epilepticus	airway protection	no	yes	no	no	no	no	no
43	232177	ogappa shakrappa honnutagi	78	f	10	6	4	21	late onset	dama	carcinoma esophagus	type 2	yes	no	no	no	no	no	no
44	252329	ramesh laxman hadapad	49	m	3			3	no vap	dama	cld	airway protection	no	yes	no	yes	yes	no	no
45	253503	raghavendra halleppa minjagi	32	m	12	3	9	12	early onset	dama	cva		no	no	no	no	no	yes	yes
46	664	shantabai biradar	65	f	13			13	no vap	worsened	Ihd	type 2	yes	yes	yes	yes	no	no	yes
47	257700	jyotis L mahto	20	m	7	5	2	10	late onset	recovered	neurotoxic snake bite	airway protection	no	yes	no	no	no	no	no
48	259448	basangouda hachreddy	78	m	8			10	no vap	dama	Ihd,copd	type 2	yes	no	yes	no	no	no	yes
49	259855	manjunath sidaray kadimani	29	m	4	3	1	4	early onset	worsened	op poisoning	airway protection	no	yes	no	no	yes	yes	no
50	258733	prabhavati annaray gabasavalagi	32	f	5			8	no vap	recovered	neurotoxic snake bite	type 2	no	no	no	no	no	no	no
51	258664	shobhagani bemal	23	f	4			4	no vap	worsened	seizure disorder	type 2	no	yes	no	no	no	no	no
52	259962	nashina sohan bagade	30	f	6			6	no vap	dama	burns	airway protection	no	yes	no	no	no	no	no

53	263020	kusheppa adiveppa nagaral	55	m	17	4	13	29	early onset	recovered	sdh,edh	airway protection	no	yes	no	yes	yes	yes	yes
54	223648	abdulraheman takkalaki	24	m	6			20	no vap	recovered	right cp angle tumor	airway protection	no	no	no	no	no	no	no
55	270330	jyoti adiyappa kinagi	36	f	4	3	1	4	early onset	worsened	rta,sdh	airway protection	no	yes	no	no	no	no	no
56	265840	bhagyashree I gareeb	21	f	11	6	5	24	late onset	recovered	op poisoning	type 2	no	no	no	no	no	no	yes
57	271924	irappa mahantappa kumbar	45	m	5	4	1	10	early onset	worsened	meningoencephalitis,cva	airway protection	no	yes	no	no	yes	yes	no
58	231747	laxman janappa kannur	58	m	5			5	no vap	dama	dcld,hrs,hepatic encephalopathy	airway protection	no	yes	no	no	yes	yes	no
59	271943	seema m kharoshi	18	f	7			12	no vap	recovered	neurotoxic snake bite	airway protection	no	yes	no	no	no	no	no
60	273815	santosh sayavva chalawadi	23	m	8			32	no vap	recovered	left radius fracture	airway protection	no	no	no	no	no	no	yes
61	278533	siddu yallappa doddamani	30	m	6			9	no vap	recovered	op poisoning	airway protection	no	yes	no	no	yes	no	yes
62	383451	mallayya g mathpati	50	m	10	5	5	10	late onset	worsened	post tb sequalae	type 2	no	no	yes	yes	no	yes	yes
63	283905	savitri s ginni	64	f	13	7	5	24	late onset	recovered	cva,meningoencephalitis	airway protection	yes	yes	no	no	no	no	yes
64	295592	savita shrishail doddi	26	f	8	5	3	13	late onset	recovered	neurotoxic snake bite	type 1	no	no	no	no	no	no	yes
65	288927	nirpad mahadev muchandi	35	m	20	12	8	29	late onset	worsened	cld,sepsis,mods	type 2	no	yes	no	no	yes	yes	yes
66	304807	basavaraj sharanappa malaghan	61	m	14			14	no vap	dama	jejunal perforation	airway protection	yes	no	no	no	no	yes	yes
67	317848	seema koushar abadulmujib halli	46	f	7	5	2	7	late onset	worsened	septic shock,mods	type 1	no	no	no	no	no	no	no
68	325255	mallu gurappa nagaral	30	m	7	4	3	7	early onset	dama	ileal perforation,sepsis,septic shock	airway protection	no	no	no	no	yes	no	no
69	331432	shabir ahmed	39	m	6			11	no vap	recovered	sdh	airway protection	no	yes	no	no	no	no	no
70	334239	vijamma sahebgouda sasanur	31	f	6	4	2	6	early onset	worsened	septic shock,mods	type 2	no	no	no	no	no	no	no
71	206589	babu kavalgi	57	m	6	4	2	15	early onset	worsened	septic shock,mods	airway protection	no	no	no	no	no	no	yes
72	91680	chetan gangadar badiger	33	m	8	4	4	12	early onset	recovered	right frontotemporal lobe decompression	type 2	no	yes	no	no	no	no	yes
73	334969	raju gopal karadi	36	m	40	12	28	60	late onset	recovered	sdh,edh	type 2	no	yes	no	no	yes	yes	yes
74	346189	shrikanth huchappa doddamani	56	m	5	3	2	5	late onset	dama	septic shock,mods	type 2	no	no	no	no	no	no	no
75	152886	nalini g kulkarni	80	f	4	4	0	4	early onset	worsened	septic shock,mods	type 2	yes	no	no	yes	no	no	no
76	347948	vitthal jotteppa waliker	26	m	24	10	14	36	late onset	worsened	septic shock,mods	type 2	no	yes	no	no	no	no	yes
77	361455	basavaraj basalingappa navadagi	35	m	9			14	no vap	recovered	sdh,sah	airway protection	no	yes	no	no	yes	yes	yes
78	365673	manappa kasturappa rathod	47	m	4	4	0	4	early onset	worsened	septic shock,mods	airway protection	no	no	no	no	no	no	no
79	368904	nagamma huchappa kodekal	66	f	4	3	1	6	early onset	worsened	septic shock,mods	airway protection	yes	yes	no	no	no	no	no
80	371311	ashok basappa sugur	59	m	10	4	6	19	early onset	recovered	right pneumothorax post TB sequalae	type 1	no	no	no	no	no	no	yes
81	369470	akshay kumar shivaray dalawi	25	m	10	6	4	17	late onset	recovered	op poisoning	type 2	no	no	no	no	no	no	yes
82	371271	gurubai meghu rathod	65	f	20	13	7	43	late onset	recovered	bilateral frontal contusion	airway protection	yes	yes	no	no	no	no	yes
83	377318	bharathi muragayya jeeragalamath	56	f	9	5	4	9	late onset	worsened	sepsis , septic shock ,sah , sdh	airway protection	no	yes	no	no	no	no	yes

84	381530	kavita sidlappanavar	26	f	23	13	10	23	late onset	dama	sdh,edh	airway protection	no	yes	no	no	yes	yes	yes
85	380249	renuka ambresh inachagal	31	f	28	15	13	42	late onset	dama	sdh,sah,diffuse axonal injury	airway protection	no	yes	no	no	no	no	yes
86	409054	siddalingappa manappa mudigal	66	m	10	5	5	22	late onset	recovered	right thalamic bleed,hydrocephalus	type 2	yes	no	no	no	no	no	yes
87	3501	malasiddappa kallappa navi	51	m	6	3	3	10	early onset	dama	cva	type 2	no	no	no	no	yes	yes	no
88	1722	shivanna narasappa badiger	56	m	9	6	3	9	late onset	dama	ic bleed	airway protection	no	yes	no	no	yes	yes	yes
89	9106	karishma irappa yaranal	26	m	6	4	2	10	early onset	recovered	op poisoning	airway protection	no	yes	no	no	no	no	no
90	38033	dundavva appasab gani	65	f	3			3	no vap	dama	septic shock,mods	airway protection	yes	yes	no	no	no	no	no
91	36198	bhagyashree holeppa	27	f	11	6	5	11	late onset	dama	septic shock,mods	airway protection	no	no	no	no	no	no	yes
92	34785	irappa gurappa belavadaggi	22	m	18	11	7	21	late onset	recovered	op poisoning	airway protection	no	yes	no	no	no	no	yes
93	264310	irappa denakkanavar	57	m	5	3	2	8	early onset	worsened	ihd,cardiogenic shock	type 2	no	no	no	no	no	yes	no
94	42153	ramesh b kalaburagi	42	m	10			23	no vap	recovered	ic bleed	type 2	no	no	no	no	yes	yes	yes
95	43373	nagappa kolli	62	m	12	7	5	26	late onset	recovered	spinal injury	type 1	yes	yes	no	no	yes	no	yes
96	50912	ningayya bhimayya pujari	45	m	16	8	8	24	late onset	recovered	sdh	airway protection	no	yes	no	no	yes	no	yes
97	52007	rahul sangappa parit	29	m	11	5	6	25	late onset	recovered	rta,sdh	airway protection	no	yes	no	no	yes	yes	yes
98	324106	mallappa d hullur	45	m	25	5	20	25	late onset	worsened	ic bleed	airway protection	no	yes	yes	no	yes	yes	yes
99	55398	rajendra chandrashekar hakkapaki	60	m	20	13	7	45	late onset	recovered	cva,htn	airway protection	yes	yes	no	no	no	no	yes
100	69289	faisal bangi	28	m	16	10	6	26	late onset	recovered	seizure disorder	airway protection	no	yes	no	no	no	no	yes
101	60919	bhimaraya gurabala makani	69	m	24	20	4	28	late onset	recovered	cva	airway protection	yes	yes	no	no	no	yes	yes
102	83685	trimurti mohan tele	26	m	13	3	10	33	early onset	recovered	edh,sdh		no	no	no	no	no	no	yes
103	88045	musthafa m mujawar	60	m	22	17	5	28	late onset	recovered	cva,aki,sepsis,mods	airway protection	no	yes	no	no	no	no	yes
104	18818	mallangouda shankargouda patil	69	m	7	7	0	7	late onset	dama	copd	type 2	yes	no	yes	no	no	yes	no
105	83534	fatima maibubsab aigali	77	f	12	10	2	12	late onset	worsened	septic shock,mods	type 2	yes	no	no	no	no	no	yes
106	91620	aravind ranganagouda patil	56	m	7	5	2	7	late onset	dama	rta	airway protection	no	yes	no	no	no	no	no
107	112234	laxmibai ganapati durve	65	f	5	3	2	25	late onset	dama	left tibia ,fibula compound fracture	type 2	yes	yes	no	no	no	no	no
108	116107	mahantgouda mullimani	65	m	16	9	7	16	late onset	worsened	septic shock,mods	type 1	yes	no	no	yes	no	no	yes
109	126154	mahadev gangappa bellannavar	27	m	16	10	6	32	late onset	recovered	sah,sdh	type 2	no	no	no	no	yes	yes	yes
110	132161	salman raza	33	m	4			4	no vap	dama	rta, ileal perforation	airway protection	no	yes	no	no	no	no	no
111	132171	harish s teggelli	38	m	10	3	7	24	early onset	recovered	edh,hie	airway protection	no	yes	no	no	yes	yes	yes
112	134489	basanna ningayya guruvin	63	m	35	8	27	35	late onset	dama	septic shock,mods	type 1	yes	no	no	yes	yes	yes	yes
113	146887	asma m antaragangi	19	f	3			3	no vap	dama	septic shock,mods	type 1	no	no	no	no	no	no	no
114	147746	bhamu kheshu pawar	40	m	4	4	0	4	early onset	dama	meningoencephalitis,sepsis	airway protection	no	yes	no	no	yes	no	no

115	150723	gurulingappa s biradar	77	m	13	5	8	13	late onset	dama	meningoencephalitis,sepsis	airway protection	yes	yes	no	no	no	no	yes
116	125592	ramesh baburao	44	m	7	4	3	10	early onset	recovered	sepsis , septic shock ,lrti	type 2	no	no	no	no	no	no	no
117	156625	ravikant ghalappa birajdar	45	m	11	11	0	11	late onset	worsened	septic shock,mods	type 1	no	no	no	no	no	no	no
118	168415	abdul rajak m jamadar	75	m	5	2	3	5	early onset	dama	pleural effusion,parkinson disease,hypothyroid	airway protection	yes	yes	no	yes	yes	yes	no
119	168398	esubai sidaray honamore	78	f	7	4	3	11	early onset	worsened	copd	type 2	yes	no	yes	yes	no	no	no
120	167006	venkappa narasagond	60	m	2	2	0	2	early onset	dama	meningoencephalitis,cva	airway protection	no	yes	no	yes	no	no	no
121	178373	gurappa sangappa metri	42	m	12	6	6	53	late onset	recovered	sdh,sah,hemothorax	airway protection	no	no	no	no	no	no	yes
122	186900	ravi mahadevappa kolar	25	m	9	8	1	9	late onset	worsened	rta	airway protection	no	yes	no	no	yes	yes	yes
123	189703	pandit zalaki	42	m	5	3	2	11	late onset	recovered	pulmonary tb	type 2	no	no	no	yes	no	yes	no
124	190591	basavaraj h kunjoti	40	m	40	37	3	52	late onset	recovered	sah,sdh	airway protection	no	yes	no	no	yes	yes	yes
125	194970	mahadevi madar	40	f	13	8	5	26	late onset	recovered	cva	airway protection	no	yes	no	no	no	no	yes
126	200561	sakkubai ramu jadhav	64	f	10	3	7	10	early onset	dama	sah,ards	airway protection	yes	yes	no	no	no	no	yes
127	212867	sangappa ramappa talawar	40	m	44	8	36	52	late onset	recovered	sdh	airway protection	no	yes	no	no	yes	yes	yes
128	214365	adappa amrappa ganganagoudar	73	m	12	7	5	21	late onset	recovered	cva,myasthenia gravis	airway protection	yes	yes	no	yes	no	no	yes
129	213516	gurappa ramappa chalawadi	66	m	3	2	1	14	early onset	worsened	motor neuron disease	airway protection	yes	yes	no	no	no	no	no
130	106361	iranagouda sahebgouda patil	54	m	10	3	7	20	early onset	recovered	sepsis , septic shock ,sah , sdh	airway protection	no	yes	no	no	no	no	yes
131	223374	kusuma ramachandra almale	69	f	12			19	no vap	recovered	brain stem bleed	airway protection	yes	yes	no	no	no	no	yes
132	227198	prabhakar siddappa pasodi	55	m	15	10	5	21	late onset	recovered	cva,aki,meningoencephalitis	airway protection	no	yes	no	yes	yes	yes	yes
133	232524	kanakappa bhimappa myageri	67	m	6			6	no vap	dama	septic shock,mods	airway protection	yes	yes	no	no	no	no	no
134	239650	keshav jadhav	64	m	11	7	4	11	late onset	worsened	ic bleed	airway protection	yes	yes	no	yes	yes	yes	no
135	242883	basangouda n metipatil	58	m	15	13	2	15	late onset	worsened	ic bleed	airway protection	no	yes	no	no	yes	no	yes
136	122134	vijayalaxmi hanamant talawar	60	f	5	3	2	8	early onset	worsened	septic shock,mods	type 1	no	yes	no	yes	no	no	no
137	249074	nilavati	70	f	12	4	8	18	late onset	recovered	cva,sdh,status epilepticus	airway protection	yes	yes	no	no	no	no	yes
138	249882	devindrappa sangappa pattar	55	m	6	4	2	8	early onset	recovered	rta	airway protection	no	yes	no	no	no	no	no
139	249871	mallikarjun shivappa pujari	40	m	15	4	11	15	early onset	worsened	brain stem injury	airway protection	no	yes	no	no	yes	yes	yes
140	251391	savitri tenkali	68	f	5	3	2	8	early onset	dama	cva,htn	airway protection	yes	yes	no	no	no	no	no
141	253646	suvarna lata	76	f	11	5	6	11	late onset	dama	cva	airway protection	yes	yes	no	yes	no	no	yes
142	253694	bhaganna yashavant wagha	39	m	18	12	6	20	late onset	dama	spinal shock,hie	airway protection	no	yes	no	no	yes	no	yes
143	261416	laxmibai bhimanna madakar	81	f	12			16	no vap	worsened	septic shock,mods	type 1	yes	no	no	yes	no	no	yes

144	262218	mallavva madiwalappa chokkavi	69	f	12	8	4	12	late onset	worsened	ihd,cardiogenic shock	type 2	yes	no	no	no	no	no	yes
145	192060	gangabai malkappa bajantri	60	f	9	8	1	9	late onset	worsened	septic shock,mods	type 1	no	no	no	yes	no	no	yes
146	269269	kaveri pujari	19	f	18	10	8	24	late onset	recovered	op poisoning	type 2	no	no	no	no	no	no	yes
147	279189	riyaz meti	43	m	3			3	no vap	dama	carcinoma lung	type 1	no	no	no	no	yes	yes	no
148	275321	marlinga siddappa kiranagi	23	m	11			11	no vap	dama	op poisoning,sepsis,mods	type 2	no	yes	no	no	yes	yes	yes
149	269285	kasturibai kumbar	73	f	15	10	5	15	late onset	worsened	sepsis , septic shock ,sah , sdh	type 2	yes	yes	no	no	no	no	no
150	294298	dayanand kallappa ghote	60	m	7	7	0	7	late onset	dama	septic shock,mods	type 2	yes	yes	no	no	no	no	no
151	242676	siddu talevada	41	m	8			13	no vap	recovered	op poisoning	type 2	no	no	no	no	yes	yes	yes
152	300678	prashant waghmore	32	m	21	11	10	21	late onset	worsened	septic shock,mods	airway protection	no	yes	no	no	yes	yes	yes
153	299198	annaraya patil	18	m	18	5	13	18	late onset	worsened	meningoencephalitis,sepsis	airway protection	no	yes	no	no	no	no	yes
154	212057	bhagyashree n	25	f	6	4	2	6	late onset	worsened	meningoencephalitis,sepsis	type 1	no	no	no	no	no	no	no
155	220201	hajimalang	42	m	10	5	5	10	late onset	dama	seizure disorder	airway protection	no	yes	no	no	no	no	yes
156	235522	revansidda yalagi	70	m	12			12	no vap	worsened	edh,sdh	airway protection	yes	yes	yes	no	yes	yes	yes
157	225103	yallavva mali	45	m	7			7	no vap	worsened	neurotoxic snake bite	type 1	no	no	no	no	yes	yes	no
158	227527	sunil koppa	52	m	7			7	no vap	worsened	carcinoma buccal mucosa,ards,sepsis	type 1	no	no	no	yes	yes	yes	no
159	2024/291	roopa more	35	f	10			10	no vap	worsened	pres syndrome,hydrocephalus,sepsis	type 2	no	no	no	no	no	no	yes
160	222711	bandenawaaz mulla	61	m	10	6	4	15	late onset	recovered	seizure disorder,sdh	type 1	yes	yes	no	yes	yes	yes	yes
161	244266	arun kumar	85	m	8			12	no vap	recovered	meningoencephalitis,sepsis	airway protection	yes	yes	yes	no	no	yes	yes
162	244273	alfiya shaikh	21	f	7			7	no vap	dama	status epilepticus	airway protection	no	yes	no	no	no	no	no
163	245628	parameshwar waliker	63	m	6	4	2	6	early onset	worsened	sdh	airway protection	yes	yes	no	no	yes	no	no
164	261834	arjun nidoni	85	m	5	3	2	10	early onset	worsened	meningoencephalitis,sepsis,copd,mods	airway protection	yes	yes	yes	no	no	yes	no
165	288885	guruling billur	51	m	4			4	no vap	worsened	septic shock,mods	type 1	no	no	no	no	no	no	no
166	297579	guraddi bhimanagouda	53	m	10	6	4	10	late onset	worsened	op poisoning	type 2	no	no	no	no	yes	yes	yes
167	345306	dasharat	70	m	5			5	no vap	worsened	septic shock,mods	type 1	yes	no	no	yes	yes	yes	no
168	348279	Rangavva	68	f	10	5	5	10	late onset	worsened	septic shock,mods	type 1	no	yes	no	no	no	no	yes
169	349884	heeru laalu chavan	65	m	5	3	2	5	early onset	dama	cardiogenic shock/heart failure	type 1	yes	no	no	yes	no	yes	no
170	355800	manohar	66	m	7	4	3	7	early onset	worsened	septic shock,mods	type 2	no	no	no	yes	yes	yes	no
171	360468	ningondappa mathali	50	m	8	5	3	8	late onset	worsened	sdh,sah	airway protection	no	yes	no	no	yes	yes	yes
172	441009	laxman gotyal	70	m	10	6	4	10	late onset	dama	dka,sepsis	type 1	yes	no	no	yes	yes	yes	yes
173	465344	shivanand s hadapad	38	m	12			12	no vap	dama	seizure disorder,sdh	airway protection	no	yes	no	no	no	no	yes
174	465333	sanganabasava gujjar	65	f	10			10	no vap	worsened	edh,sdh	airway protection	yes	yes	no	no	no	no	yes
175	475720	lalesab kadarasab nashi	65	m	4			4	no vap	worsened	post tb sequalae	type 2	no	no	yes	no	yes	yes	no

Sl No	Immunosupressive therapy	Organ Failure	Reintubation	Emergency intubation	Tracheostomy	ET culture	AMOXICILLIN - CLAVULANIC ACID	PIPERACILLIN-TAZOBACTAM	CEFTRIAXONE	CEFOPERAZONE-SULBACTAM	IMIPENEM	MEROPENEM	AMIKACIN	GENTAMICIN	CIPROFLOXACIN	TIGECYCLINE	TRIMETHOPRIIM-SULFAMETHOXAZOLE	LEVOFLOXACIN
1	yes	yes	no	yes	no	Acinetobacter spp				S	R	R	R	R	S	S	S	
2	no	no	no	yes	no													
3	no	yes	no	yes	no	Citrobacter freundii				S	S	S	S	S		S	S	
4	no	no	no	yes	yes	Klebsiella pneumoniae	R	R	R			R	R	S	R	S	S	R
5	no	yes	no	yes	no	Acinetobacter spp		R				R	R	R	R	S		R
6	no	yes	no	yes	no	Klebsiella pneumoniae	R	R	R			R	R	S	R	S	S	R
7	no	yes	no	yes	no	Klebsiella pneumoniae	R	R	R	S		R	R		R	S		R
8	no	no	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
9	no	no	no	yes	no	Acinetobacter baumannii (MDR)		R	R	S	R	R	R		R	S	R	R
10	no	yes	no	yes	no	Escherichia coli	R	R	R	S	S	S	S	S	R	S	S	
11	no	yes	no	yes	no	Acinetobacter baumannii (MDR)		R	R	R		R	R				S	
12	no	yes	no	yes	no	Pseudomonas aeruginosa		S		S	S	S						
13	yes	yes	no	yes	no	Acinetobacter baumannii (MDR)		R	R	S		R	R		R	S	R	R
14	yes	no	no	yes	no	Klebsiella pneumoniae	S	R	R	S	R	R	S	S	S	S	S	
15	no	yes	yes	yes	no	Klebsiella pneumoniae	R	R	R			R	S	S		S	R	
16	yes	no	yes	yes	yes	Klebsiella pneumoniae	S	R	R	S	R	R	S	S	S	S	S	
17	no	no	no	yes	no													
18	no	no	no	yes	no													
19	no	yes	no	no	no													
20	no	yes	no	no	no	Pseudomonas aeruginosa		S		S	S	S	S	S				S
21	no	yes	no	yes	no	Klebsiella pneumoniae	R	R	R			R	S	S		S	R	
22	yes	yes	no	yes	yes	Staphylococcus aureus									S	S	S	S
23	no	no	no	yes	no	Klebsiella pneumoniae	S	R	R	S	R	R	S	S	S	S	S	S

24	no	no	no	no	no	Escherichia coli (CRE)		R	R		R	R	R		R	S	R	R
25	no	no	no	yes	no	Acinetobacter baumannii complex					R	R						
26	no	yes	no	yes	no													
27	no	no	no	yes	no	Pseudomonas aeruginosa		S		S		S	S	S				S
28	no	no	no	no	no	Staphylococcus aureus (MRSA)									R	S	S	R
29	no	yes	no	yes	no	Klebsiella pneumoniae	S	R	S	S	S	S	S	R	R	S	S	
30	no	no	no	no	no	Serratia marcescens		R	R		R	R	R		R	S	R	R
31	no	no	no	no	no													
32	no	no	no	yes	no	Serratia marcescens	R	R	R		S	S	R		R	S	S	R
33	no	no	no	yes	no	Klebsiella oxytoca	R	R	R		S		R	S	R	S	S	R
34	no	no	no	yes	no	Klebsiella pneumoniae		R	R			R	R		R	S	R	R
35	yes	yes	no	no	no	Acinetobacter baumannii complex		R	R			R	R		R	S	R	R
36	no	no	no	yes	no													
37	no	yes	no	yes	no	Escherichia coli	S	R	R	S	S	S	S	S	R	S	S	
38	no	no	no	yes	no	Pseudomonas aeruginosa		S	S	S	S	S	S		S		S	S
39	yes	yes	no	no	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
40	no	yes	no	yes	no	Staphylococcus aureus (MRSA)									R	S	S	R
41	yes	yes	no	yes	no	**Citrobacter freundii**	R	R	R		R	R	R	R	R	S	S	R
42	no	no	no	no	no													
43	yes	no	no	no	no	Klebsiella pneumoniae		R	R	S	R	R	S	S	S	S	S	S
44	no	no	no	yes	no													
45	no	no	no	no	no	Staphylococcus aureus								S	S	S	S	S
46	yes	yes	no	no	no													
47	no	no	no	yes	no	Klebsiella pneumoniae	S	R	R	S	S	S	S	R	R	S	S	
48	no	no	no	no	no													
49	no	no	no	yes	no	**Staphylococcus aureus (MRSA)**									R	S	S	R
50	no	no	no	no	no													
51	no	yes	no	no	no													
52	yes	yes	no	yes	no													

53	no	no	no	no	no	citrobacter koseri	R	R	R		R	R	R	R	R	S	S	R
54	no	no	no	no	no													
55	no	no	no	yes	no	**Pseudomonas aeruginosa**	S	S	S	S	S	S	S		S			S
56	no	no	no	no	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
57	yes	yes	no	no	no	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	R	S	R
58	yes	yes	no	yes	no													
59	no	no	no	yes	no													
60	no	no	no	no	no													
61	no	no	no	yes	no													
62	yes	yes	no	no	no	Klebsiella pneumoniae	R	R	R	R	R	R	R	R	R	S	R	R
63	no	no	no	yes	no	**Pseudomonas aeruginosa**	S	S	S	S	S	S	S		S			S
64	no	no	no	no	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
65	yes	yes	yes	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
66	no	yes	no	no	no													
67	yes	yes	no	yes	no	Staphylococcus aureus									S	S	S	S
68	yes	yes	no	yes	no	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	S	S	R
69	no	no	no	no	yes													
70	no	yes	no	yes	no	Citrobacter freundii		R	R		R	R	R		R	S	R	R
71	no	no	no	no	no	Klebsiella pneumoniae		R	R		R	R	R		R	S	R	R
72	no	no	no	no	yes	Klebsiella pneumoniae ssp pneumoniae (MDRO)		R	R		R	R	R		R	S	R	R
73	yes	no	yes	yes	yes	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
74	no	yes	no	no	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
75	no	yes	no	no	no	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	S	S	R
76	yes	yes	no	yes	yes	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	S	S	R
77	no	no	no	yes	no													
78	yes	yes	no	yes	no	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	S	S	R
79	no	yes	no	yes	no	Escherichia coli (CRE)		R	R		R	R	R		R	S	R	R
80	yes	yes	no	no	no	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	S	S	R
81	no	no	no	no	no	Pseudomonas aeruginosa				R		R	S		S			R
82	no	no	no	yes	yes	Staphylococcus aureus (MRSA)									R	S	S	R

83	no	yes	no	no	no	Klebsiella pneumoniae		R	R		R	R	R		R	S	R	R
84	no	no	no	yes	yes	Klebsiella pneumoniae				S	S	S	S	S		S	S	
85	yes	yes	yes	yes	yes	Klebsiella pneumoniae	R	R	R	R	R	R	S	S	R	S	S	R
86	no	no	no	no	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)		R	R		R	R	R		R	S	R	R
87	no	no	no	yes	no	Klebsiella pneumoniae	R	R	R	S			R		R	S	S	R
88	no	no	no	yes	no	Staphylococcus aureus (MRSA)	S	S	S	S	S	S	S	S		S	S	
89	no	no	no	yes	no	Staphylococcus aureus		R	R					R	R	S		
90	no	yes	no	yes	no													
91	yes	yes	no	yes	no	Klebsiella pneumoniae		R	R		R	R	R		R		R	R
92	no	yes	no	yes	yes	Pseudomonas aeruginosa				R		R	S		S			R
93	no	yes	no	no	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
94	no	no	no	no	no													
95	no	no	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
96	no	no	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
97	no	no	no	yes	no	Klebsiella pneumoniae		R	R		R	R	R		R	S	R	R
98	no	no	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
99	no	no	no	yes	yes	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
100	no	no	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
101	no	yes	no	yes	yes	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
102	yes	no	no	no	yes	**Klebsiella pneumoniae ssp pneumoniae (MDRO)**	R	R	R	R	R	R	R	R	R	S	R	R
103	yes	yes	yes	yes	no	Klebsiella pneumoniae	R	R	R	R	R	R	R	R	R	S	R	R
104	no	yes	no	yes	no	Citrobacter freundii		R	R		R	R	R		R	S	R	R
105	yes	no	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
106	no	no	no	yes	no	Klebsiella pneumoniae		R	R		R	R	R		R	S	R	R
107	yes	yes	no	no	no	Acinetobacter baumannii complex	R	R	R	R	R	R	R	R	R	S	R	R
108	yes	yes	no	yes	no	Klebsiella pneumoniae		R	R		R	R	R		R	S	R	R
109	no	no	no	no	yes	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
110	no	no	no	yes	no													
111	yes	yes	no	yes	yes	Acinetobacter baumannii complex	R	R	R	R	R	R	R	R	R	S	R	R

112	yes	yes	no	no	yes	Acinetobacter baumannii complex	R	R	R	R	R	R	R	R	R	S	R	R
113	no	no	no	yes	no													
114	no	no	no	yes	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)		R	R		R	R	R		R	S	S	R
115	no	no	no	yes	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)	R	R	R	R	R	R	R	R	R	S	R	R
116	yes	yes	no	no	yes	Klebsiella pneumoniae		R	R		R	R	R		R	S	R	R
117	yes	yes	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
118	no	no	no	yes	no	Klebsiella pneumoniae	R	R	R	S	R	R	S	S	R	S	R	R
119	yes	yes	no	no	no	Acinetobacter baumannii complex	R	R	R	R	R	R	R	R	R	S	S	R
120	no	no	no	yes	no	Klebsiella pneumoniae	S	S	R	S	S	S	S	S	S	S	S	S
121	no	no	no	no	no	Klebsiella oxytoca	R	R	R	R	R	R	R	R	R	S	R	R
122	no	no	no	no	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)	R	R	R	R	R	S	S	R	R	S	S	R
123	yes	yes	no	no	no	Klebsiella pneumoniae	R	R	R	R	R	R	R	R	R	S	R	R
124	yes	no	no	yes	yes	Klebsiella pneumoniae ssp pneumoniae (MDRO)	R	R	R	R	R	R	R	R	R	R	R	R
125	yes	no	no	yes	no	Pseudomonas aeruginosa		R		S	S	S	S		R			R
126	no	no	no	yes	no	Acinetobacter baumannii complex	R	R	R	S	R	R	R	R	R	S	S	R
127	yes	no	yes	yes	yes	**Enterobacter aerogenes**	R	R	R	R	R	R	R	R	R	S	S	R
128	yes	yes	no	yes	no	Acinetobacter baumannii complex	R	R	R	R	R	R	R	R	R	S	R	R
129	no	no	no	yes	no	Acinetobacter baumannii complex	R	R	R	R	R	R	R	R	R	S	R	R
130	yes	yes	no	yes	no	Staphylococcus aureus (MRSA)	S	S	S	S	S	S	S	S	R	S	S	R
131	no	no	no	yes	no													
132	yes	no	no	yes	no	Klebsiella pneumoniae	R	R	R	R	R	R	R	R	R	S	R	R
133	yes	yes	no	yes	no													
134	no	yes	no	yes	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)	R	R	R	R	R	R	R	R	R	S	R	R
135	no	yes	no	yes	no	Klebsiella pneumoniae	R	R	R	R	R	R	R	R	R	S	R	R
136	yes	yes	no	no	no	Escherichia coli	R	R	R	R	R	S	S	S	R	S	S	R
137	no	no	no	yes	no	Acinetobacter baumannii complex	R	R	R	S	R	R	R	R	R	S	R	R
138	no	no	no	yes	no	Acinetobacter baumannii complex	R	R	R	S	R	R	R	R	R	S	R	R
139	no	yes	no	yes	no	Acinetobacter baumannii complex	R	R	R	S	R	R	R	R	R	S	R	R
140	no	yes	no	yes	no	Acinetobacter baumannii complex	R	R	R	S	R	R	R	R	R	S	R	R

141	yes	no	no	yes	yes	Acinetobacter baumannii complex	R	R	R	S	R	R	R	R	R	S	R	R
142	yes	yes	yes	yes	no	Pseudomonas aeruginosa(MDR)	R	R	R	R	R	R	R	R	R	R	R	R
143	no	yes	no	no	no													
144	yes	yes	yes	yes	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)	R	R	R	R	R	R	R	R	R	S	R	R
145	yes	yes	no	no	no	Pseudomonas aeruginosa(MDR)		R		S	S	S	S		R			R
146	no	no	yes	yes	yes	Pseudomonas aeruginosa		R	R		R	R	S	S			S	
147	yes	yes	no	no	no													
148	yes	yes	yes	yes	no													
149	no	yes	no	yes	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)	R	R	R	R	R	R	R	R	R		R	R
150	yes	yes	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
151	no	no	no	no	no													
152	yes	yes	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
153	yes	yes	no	yes	no	Escherichia coli		S	R	S	S	S		R	R			
154	no	yes	no	no	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S		R
155	no	no	no	yes	no	Pseudomonas aeruginosa		S	R	R	R	R			S			S
156	no	no	no	yes	no													
157	no	yes	no	no	no													
158	yes	yes	no	no	no													
159	yes	yes	no	no	no													
160	no	no	no	yes	no	Staphylococcus aureus				S	S	S	S	S	R	S	R	R
161	no	no	no	yes	no													
162	no	no	no	yes	no													
163	no	no	no	yes	no	Klebsiella oxytoca		R	R		R	R	R		R	S	R	R
164	yes	yes	no	yes	no	Serratia marcescens		R	R		R	R	R		R	S	R	R
165	yes	yes	no	no	no													
166	no	yes	no	yes	no	Acinetobacter baumannii complex		R	R		R	R	R		R	S	R	R
167	yes	yes	no	yes	no													
168	no	yes	no	yes	no	Pseudomonas aeruginosa		R	R		R	R	R		R	S	R	R
169	no	yes	no	no	no	Enterobacter aerogenes		S		S	S	S	S	S				S
170	no	yes	no	no	no	Pseudomonas aeruginosa		R	R		R	R	R		R	S	R	R
171	no	no	no	yes	no	Klebsiella pneumoniae ssp pneumoniae (MDRO)				S	S	S	S	S	R	S	R	R
172	no	no	no	yes	no	Escherichia coli (CRE)	R	R	R	S	R	R	S	S	R	S		
173	no	no	yes	yes	yes													
174	no	yes	no	yes	no													
175	yes	yes	no	yes	no													