"COMPARISON OF VENTILATOR ASSOCIATED PNEUMONIA BETWEEN PULMONARY AND NON-PULMONARY INDICATIONS FOR MECHANICAL VENTILATION"

Submitted by

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In partial fulfilment of the requirements for the degree of

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IN

RESPIRATORY MEDICINE

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I hereby declare that this dissertation/thesis entitled **"COMPARISON OF VENTILATOR ASSOCIATED PNEUMONIA BETWEEN PULMONARY AND NON-PULMONARY INDICATIONS FOR MECHANICAL VENTILATION"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. KEERTIVARDHAN D KULKARNI**, Professor and Head, Department of Respiratory Medicine, BLDE (DU) and DR. **SANJEEVKUMAR N. BENTOOR,** Professor and Head, Department of Medicine, BLDE (DU) Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura, Karnataka.

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LIST OF ABBREVIATIONS

VAP: Ventilator Associated Pneumonia

MV: Mechanical Ventilation

COPD: Chronic obstructive pulmonary disease

TBI: Traumatic brain injury

ILD: Interstitial Lung disease

ICU: Intensive Care Unit

APACHE II: Acute Physiology and Chronic Health Evaluation II

CPIS: Clinical Pulmonary Infection Score

SOFA: Sequential Organ Assessment Score

VAC: Ventilator associated Condition

VAE: Ventilator associated event

HAP: Hospital acquired pneumonia

CAP: Community acquired pneumonia

HCAP: Health Care associated pneumonia

CLD: Chronic liver disease

CKD: Chronic kidney disease

IHD: Interstitial heart disease

MDR: Multi drug resistance

MDRO: Multi drug resistant organisms

MRSA: Methicillin Resistant Staphylococcus Aureus

MSSA: Methicillin sensitive Staphylococcus Aureus

CHF: Chronic heart failure

%: Percentage

ROS: Reactive oxygen species

PaCO2: Partial pressure of carbon dioxide

PaO2: Partial pressure of oxygen

FiO2: Fractional inspired oxygen

HIV: Human Immunodeficiency Virus

NIV: Non-invasive ventilation

VHAP: Ventilator hospital acquired pneumonia

MAP: Mean arterial pressure

SPP: Species

ATS: American thoracic society

IDSA: Infectious diseases society of America

CDC: Centre of disease control

XDR: Extensive drug resistance

PDR: Pan drug resistance

ARDS: Acute respiratory distress syndrome

VAT: Ventilator associated tracheobronchitis

ECBL: Extended spectrum beta lactamase

CRE: Colistin resistant enterobacteriacae

BAL: Bronchoalveolar lavage

PSB: Protected specimen brush

PCR: Polymerase chain reaction

VAC mode: Volume limited assist control mode

PAC Mode: Pressure limited assist control mode

SIMV: Synchronised intermittent mandatory ventilation

PSV: Pressure support ventilation

E. coli: Escherichia Coli

P. aeruginosa: Pseudomonas aeruginos

ABSTRACT

BACKGROUND:

Ventilator associated pneumonia (VAP) is among the most prevalent illnesses that are acquired in hospitals. VAP develops in about 10% of patients who need mechanical breathing, with a 20–50% fatality rate world wide. In India, the incidence has been estimated to be around 27%. VAP is defined as pneumonia or infection in lung parenchyma acquired in patients after invasive mechanical ventilation after 48–72 hours. It is associated with high morbidity, mortality, prolonged hospital stay, and cost of treatment. The indications for mechanical ventilation could be due to pulmonary or non-pulmonary causes like, acute hypoxemic respiratory failure, hypoventilation, hemodynamic compromise, cardiorespiratory arrest, stroke, traumatic brain injury, and the incidence of VAP in these conditions differs accordingly. There are various scores available for early diagnosis of VAP and to predict the outcomes and mortality of the patients on mechanical ventilation. While the Clinical Pulmonary Infection Score (CPIS) aids in diagnosis of VAP, Sequential organ function assessment (SOFA) score is a widely used tool for predicting mortality in septic ICU patients, whereas Acute physiology and chronic health evaluation (APACHE II) score predicts the outcome and duration of Mechanical ventilation

AIM OF THE STUDY:

To compare ventilated associated pneumonia between pulmonary indications and non-pulmonary indications of Mechanical ventilation.

OBJECTIVES OF THE STUDY:

1. To compare Incidence of VAP between pulmonary and non-pulmonary indications for Mechanical ventilation.

2. To detect the organism and its resistance pattern causing VAP in ICU.

3. To compare the outcome of VAP between pulmonary and non-pulmonary indications for Mechanical ventilation.

4.To find out the predictive value of the following at diagnosis towards outcome and prognosis:

- Modified Clinical Pulmonary Infection Score (modified CPIS)
- Acute physiology and Chronic Health Evaluation (APACHE-II)
- Sequential Organ Failure Assessment Score (SOFA score)

MATERIALS AND METHODS:

Patients included in the study will be evaluated daily in the ICU. Baseline Chest X-ray will be done immediately after Intubation or Tracheostomy and Chest X-ray after 48 hours will be repeated and compared. Any new pulmonary lesion will be considered as VAP according to the ATS/IDSA Guidelines. Patients admitted in Shri. B. M. Patil Medical College and Hospital, and developing VAP post mechanical ventilation for various causes (pulmonary vs non pulmonary) were enrolled in the study. Course in the hospital of all patients developing VAP followed up till the discharge of the patients. ET tube secretions, Tracheostomy tube secretions will be sent for Gram's stain and Culture and Sensitivity for isolation of organism and resistance pattern.

Predictors of severity like Apache-II score, SOFA score, and CPIS will be calculated and analyzed. Data collected will be analysed by comparison of VAP between pulmonary and non-pulmonary indications for Mechanical Ventilation. Incidence, organism, resistance pattern, outcomes like resolution of VAP/ death/progression will be analyzed.

RESULTS

In this study, the mean age in pulmonary group is 58 ± 16.4 and in Non-pulmonary group is 49.5 ± 18.03 respectively and male predominance was dominated in our study.

Out of 254 Mechanically ventilated patients, the incidence of Ventilator associated pneumonia (VAP) in pulmonary group is 47% in pulmonary group and 52.5% in non-pulmonary group.

Patients who didn't develop VAP were not included in the study. VAP is diagnosed based on new infiltrates on Chest X-ray after 48 hours of mechanical ventilation and positive ET/Tracheostomy secretion culture. Among 126 VAP patients (63 in each group) COPD patients in pulmonary group and traumatic brain injury (TBI) patients in non-pulmonary group patients have a higher rate of VAP development.

Among the pulmonary cases, the three most frequently isolated pathogens were the gram-negative organisms which are *Acinetobacter baumannii Complex*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa* and the most common gram-positive organism is *Staphylococcus aureus.*

In the non-pulmonary group, *Acinetobacter baumannii Complex* was again the most prevalent pathogen, followed closely by *Klebsiella pneumoniae* and *Klebsiella pneumoniae* (MDRO) which are the most common gram-negative organisms. and most common gram-positive organism is again the *Staphylococcus aureus.*

Most of the organisms in pulmonary group are resistant to Carbapenems > Ceftriaxone > Cefuroxime > Piperacillin/Tazobactam > Ciprofloxacin = Amikacin > Cefoperazone/Sulbactam.

Most of the organisms in Non-pulmonary group are resistant to Fluoroquinolones > Piperacillin/ Tazobactam > Ceftriaxone > Carbapenems (Meropenem > Imipenem) > Amoxicillin/Clavulunic acid > Aminoglycosides > Cefoperazone/Sulbactam

Most of the organisms are sensitive to Tigecycline followed by Trimethoprim/Sulfamethoxazole, Cefoperozone/sulbactam and Aminoglycosides in both the groups.

A total of 35 patients are excluded from our study due to various reasons like Discharge

against medical advice (DAMA) due to financial issues, family issues and referral to higher centres. Out of 91 patients, overall mortality rate in our study in VAP patients is 25.3% and improvement is seen in 46.8% patients. Our study shows high mortality in pulmonary group 30.1% compared to Non pulmonary group. Improvement Rates were significantly higher in pulmonary cases 55.6% compared to non-pulmonary cases 38.1% which is statistically significant.

There is a strong association between higher modified CPIS scores and adverse patient outcomes in pulmonary group. Among non-pulmonary group Modified CPIS has moderate predictive ability for mortality in VAP.

APACHE 2 score is a moderate predictor for mortality in pulmonary cases with moderate sensitivity and specificity. Among non-pulmonary group, APACHE II score demonstrated a strong ability to discriminate between survivors and non-survivors. A higher sensitivity ensures that no high-risk patients are missed, which is crucial in clinical settings where early intervention can significantly impact patient outcomes.

SOFA score is a moderate predictor for mortality in pulmonary cases with moderate sensitivity and specificity. Among non-pulmonary group, the findings in our study suggest that the SOFA score is a valuable prognostic tool, with a high discriminatory ability.

CONCLUSION

Incidence of Ventilator associated pneumonia (VAP) in pulmonary group is 47% in pulmonary group and 52.5% in non-pulmonary group. The most common organisms causing VAP are *Acinetobacter baumannii Complex* followed closely by *Klebsiella pneumoniae* in both groups. Most of the organisms in pulmonary group are resistant to Carbapenems > Ceftriaxone and in non-pulmonary group, high resistance is to fluoroquinolones and Piperacillin/Tazobactam. The overall mortality and improvement is more in pulmonary group compared to non pulmonary group. Modified CPIS is the strong predictor of mortality in

pulmonary group whereas APACHE 2 and SOFA score are the strong predictors of outcome in the non-pulmonary group.

KEYWORDS: VAP, COPD, TBI, SOFA, APACHE 2, MODIFIED CPIS

INTRODUCTION

In hospitals, ventilator-associated pneumonia (VAP) is one of the most common infections. Roughly, 10% of patients requiring Mechanical ventilation develop VAP, with a mortality rate of 20-50% globally [(1)]. In India, the incidence has been estimated to be around 27% [(2)].

VAP is defined as pneumonia or infection in lung parenchyma acquired in patients after invasive mechanical ventilation after 48–72 hours [(3)]. It is associated with high morbidity, mortality, prolonged hospital stay, and cost of treatment. The indications for mechanical ventilation could be due to pulmonary or non-pulmonary causes like, acute hypoxemic respiratory failure, hypoventilation, hemodynamic compromise, cardiorespiratory arrest, stroke, traumatic brain injury etc, and the incidence of VAP in these conditions differs accordingly [(3)]. There are various scores available for early diagnosis of VAP and to predict the outcomes and mortality of the patients on mechanical ventilation. While the Clinical Pulmonary Infection Score (CPIS) aids in diagnosis of VAP, Sequential organ function assessment (SOFA) score is a widely used tool for predicting mortality in septic ICU patients, whereas Acute physiology and chronic health evaluation (APACHE II) score predicts the outcome and duration of Mechanical ventilation [(4)]

It has also been reported in several studies that a third to a half of all VAP-related deaths are the direct result of infection [(5)]. Although the causative organisms differ in each ICU set up, the most common with a higher mortality rate in cases are Pseudomonas aeruginosa and Acinetobacter species, thus implying the need to identify the causative organism for targeted therapy with antibiotics and to enable early recovery [(5)].

Ventilator associated pneumonia (VAP) is a nosocomial infection which develops after 48 hours of mechanical ventilation. It is one of the most important complications of the 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 [1,2].

VAP is defined as pneumonia or infection in lung parenchyma acquired in patients after invasive mechanical ventilation after 48–72 hours [3].

The indications for mechanical ventilation could be due to pulmonary or non-pulmonary causes like, acute hypoxemic respiratory failure, hypoventilation, hemodynamic compromise, cardiorespiratory arrest, stroke, traumatic brain injury etc, and the incidence of VAP in these conditions differs accordingly [3].

The most frequent ICU-acquired infection in patients on mechanical ventilation is VAP[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].

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[7,8]. The morbidity and mortality rates associated with ventilator-associated pneumonia are considerably higher.[10,11]

There are various scores available for early diagnosis of VAP and to predict the outcomes and mortality of the patients on mechanical ventilation. Clinical Pulmonary Infection Score (CPIS) aids in diagnosis of VAP, Sequential organ function assessment (SOFA) score is a widely used tool for predicting mortality in septic ICU patients, whereas Acute physiology

and chronic health evaluation (APACHE II) score predicts the outcome and duration of Mechanical ventilation. [12]

This study is undertaken to assess the incidence and outcomes of VAP in pulmonary and non-pulmonary indications of mechanical ventilation, and also to identify the most common organisms causing the infection and their resistance pattern.

AIMS AND OBJECTIVES OF THE STUDY

AIM OF THE STUDY:

To compare ventilated associated pneumonia between pulmonary indications and non-pulmonary indications of Mechanical ventilation.

OBJECTIVES:

1. To compare Incidence of VAP between pulmonary and non-pulmonary indications for Mechanical ventilation.

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- Sequential Organ Failure Assessment Score (SOFA score)

REVIEW OF LITERATURE

HISTORY:

Since the late 1950s Ventilator-associated pneumonia (VAP) has been a known complication in the intensive care unit (ICU) [13]. Critically ill patients had respiratory tract colonization, by their own Gram-negative flora, and these organisms often proliferated in endotracheal tube biofilm, and condensated in ventilator circuits, and often re-inoculated into patients during endotracheal suctioning and tubing circuit changes [14].

Since late 1960s-1970s VAP was increasingly recognized as a significant complication of mechanical ventilation. The incidence of pneumonia in intubated patients was higher than in those who were not ventilated.

The development of diagnostic criteria and definitions for VAP took place in late 1980s where it occurred in up to 28% of mechanically ventilated patients, with the highest rates early in the course of intubation (3% per day risk up to day 5) [13].

In late 1990s the emergence of antibiotic resistance in pathogens associated with VAP became a significant concern. This led to studies on the microbiology of VAP and the development of guidelines for its prevention and management. This information was used in the early part of this century to develop "ventilator bundles", which dramatically reduced the reported rates of VAP.

2010s-Present: Ongoing research has focused on improving outcomes through enhanced infection control practices, the use of non-invasive ventilation (NIV) where appropriate, and the investigation of new antimicrobial agents. The role of the microbiome in respiratory infections has also gained attention [13].

The ongoing issue of multi-drugresistant organisms (MDRO) complicates the treatment of VAP, necessitating continuous monitoring and research into new therapeutic options [15]. The COVID-19 pandemic brought renewed focus on VAP, particularly in mechanically ventilated

patients, highlighting both the challenges and the need for updated protocols [12].

Pneumonia is often classified based on the location where it was acquired [7]. "Hospital-acquired pneumonia (HAP), also known as nosocomial pneumonia, occurs 48 hours or more after hospital admission and is not present at the time of admission [8]. Ventilator-associated pneumonia (VAP) is a specific form of HAP that develops 48 hours or more after endotracheal intubation and mechanical ventilation"[9-12]. "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. However, 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, ventilated hospital-acquired pneumonia (VHAP) shares similar microbiology, diagnostic approaches, and clinical outcomes with VAP rather than with HAP"[16–20].

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 (for example, 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 aspiration 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 (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.

Table 1: Pneumonia definitions

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 hospitalization within the previous three months. This category is abandoned from recent 2019 American Thoracic Society/Infectious Diseases Society of America (ATS/IDSA) guidelines. This 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[21].

Antimicrobial resistance: "The Centres 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)" [22].

"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.

Pan drug 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 (CDC) and Prevention 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.[22,23].

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[24].

VAP rate was higher in patients with ARDS than in other ventilated patients which leads to sepsis, multiple organ failure, and death. Burden of HAP is estimated at around 5-10 cases per 1000 hospital admissions with a 6-fold to 20-fold increased risk of VAP in mechanically ventilated patients. VAP appear to have a 2-fold to 10-fold higher risk of death than those without pneumonia.

"Long hospital stays and high expenses are related with VAP[12] .VAP increases the time of mechanical ventilation by 7.6 to 11.5 days and hospitalization 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"[25,26].

Pathogenesis:

Independent predictors of Ventilator associated Pneumonia (VAP):

- Burns, trauma, CNS disease, respiratory disease, or cardiac disease
- Mechanical ventilation during the preceding 24 hours
- Witnessed aspiration
- Use of paralytic agents.

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 defences, including humoral, mechanical, and cellular mechanisms. The organisms causing VAP vary according to case mix, prior antibiotic exposure, the length of stay in the ICU, length of mechanical ventilation, patient characteristics, clinical circumstances, and geographic location even between units in the same hospital [(8)].

The primary route of lung infection is the micro aspiration 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[(27)]. "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."[(28,29)].

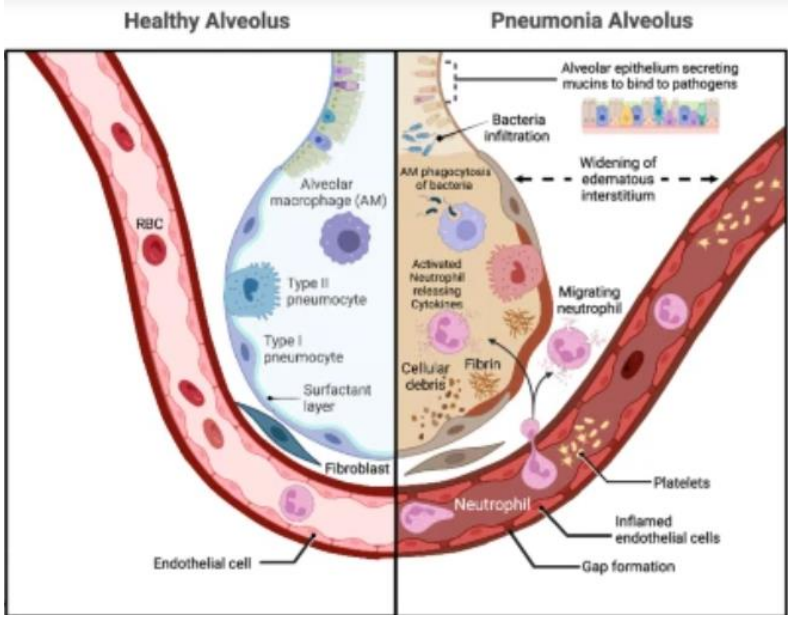


Figure 1: In response to invading pathogens, alveolar macrophages and neutrophils' immune and inflammatory response lead to inflamed, edematous and infected alveoli [(7)].

Ventilator-associated tracheobronchitis (VAT) refers to a lower respiratory infection of intubated mechanically ventilated patients with no radiological infiltrate present. The definition of VAT shares the same criteria as VAP, except without the presence of new pulmonary infiltrates on portable chest radiograph.

Clinical presentation:

More than 48 hours after intubation, the majority of patients with VAP experience a gradual or sudden onset of the following symptoms[30].

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, leucocytosis

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[31,32]. "There is growing realization 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 (Pseudomonas aeruginosa, Acinetobacter, and Stenotrophomonas maltophilia) were less common. It specifically contained MSSA (13%), MRSA (20%), P. aeruginosa (9%), Stenotrophonas 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[13].

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

Table 2: Risk factors for multidrug resistant ventilator associated pneumonia

Extended-spectrum β-lactamase–producing **Enterobacteriaceae** (ESBL-PE) are increasingly encountered in patients with HAP, including VAP, with additional mortality. They now represent 19–61% of the episodes caused by Enterobacteriaceae.

Systematic screening of ESBL-PE **fecal carriage** may help to guide initial therapy in patients with VAP when cultures are negative because they have a very good negative predictive value for subsequent ESBL-PE infections

Carbapenem resistant Enterobacteriaceae (CRE) - The emergence of infections caused by worldwide represents another risk for VAP

ICU Location	**VAP cases**	**Ventilator Days**	**Rate**
Burn	86	19,503	4.4
Medical	396	419,123	0.9
Medical/surgical	1398	1,330,178	1.0
Neurologic	62	20,859	3.0
Neurosurgical	210	98,026	2.1
Surgical	472	223,639	2.1
Cardiothoracic	319	190,785	1.7
Trauma	508	141,314	3.6

Table 3: National Healthcare Safety Network VAP Rates Based on ICU Location in Major Teaching Hospitals

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 (e.g., fever, secretions, leukocytosis). When a pathogen is identified in lower respiratory tract sample, the diagnosis is confirmed."

Criteria	**Description**
Clinical	• Fever > 38 °C with no other cause AND • Leucocytosis or leukopenia AND at least one of the following; • New onset or change in sputum • Cough, dyspnoea or tachypnoea • Worsening gas exchange
Radiological	• Chest radiographs or computed tomograms with evidence of pulmonary infiltrates OR air bronchograms. If there is a pulmonary disease history, compare serial images.

Microbiological	• Positive quantitative culture from minimally contaminated lower respiratory tract specimen OR • Positive sputum culture or non-quantitative lower respiratory tract culture

Table 4: Clinical, Radiological and Microbiological Criteria for diagnosing VAP [7].

PNEU Type	Definition
PNU 1	Two or more serial chest radiographs with at least one of the following: new or progressive and persistent infiltrate, consolidation, or cavitation and at least one of the following: • Fever (>38°C) • Leukopenia (<4000 WBC/mm3) or leukocytosis (>12000 WBC/mm3) • Altered mental status in an adult ≥70 years of age without an alternative etiology and At least two of the following:

	• New onset of purulent sputum or change in character of sputum or increased secretions/suction requirements • New-onset or worsening cough, dyspnea, or tachypnea • Rales or bronchial breath sounds • Worsening gas exchange
PNU 2	Two or more serial chest radiographs with at least one of the following: new or progressive and persistent infiltrate, consolidation, or cavitation At least one of the following: • Fever (>38°C) • Leukopenia (<4000 WBC/mm3) or leukocytosis (>12000 WBC/mm3) and At least one of the following: • New onset of purulent sputum or change in character of sputum or increased secretions/suction requirements

	• New-onset or worsening cough, dyspnea, or tachypnea • Rales or bronchial breath sounds • Worsening gas exchange And At least one of the following: • Positive blood culture not related to another source of infection • Positive pleural fluid culture • Positive quantitative culture from minimally contaminated LRT specimen • ≥5% of BAL cells containing intracellular bacteria • Histopathologic examination revealing one of the following: abscess formation, positive quantitative culture, or invasion of lung parenchyma by fungal hyphae or pseudo hyphae

Table 5: Centers for Disease Control and Prevention National Healthcare Safety Network

The absence of lung infiltrates does not exclude the possibility that a percentage of VAT could be actual VAP [33]. The main pathogenetic theories are: a) colonization leads to (Ventilator associated tracheobronchitis) VAT and VAT leads to VAP, i.e., VAP is preceded by VAT, b) colonization may lead to either VAT or VAP, without VAT being a precursor of VAP, and c) colonization leads to ventilator-associated respiratory infections with some overlap between VAT and early-VAP.

Computed tomography:

Chest computed tomography (CT) without contrast is not routinely utilized for patients with suspected ventilator-associated pneumonia (VAP) [34]. 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 [35]. 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[36].

Respiratory tract sampling:

"Antibiotic therapy lowers the sensitivity of both microscopic analysis and culture, so respiratory samples are preferably acquired prior to the commencement of medications or modification of antibiotic therapy (in those currently receiving antibiotics)[37–39]. However, it is not unusual for severe sickness or sampling delays to necessitate the administration of empiric antibiotics prior to diagnostic sampling."

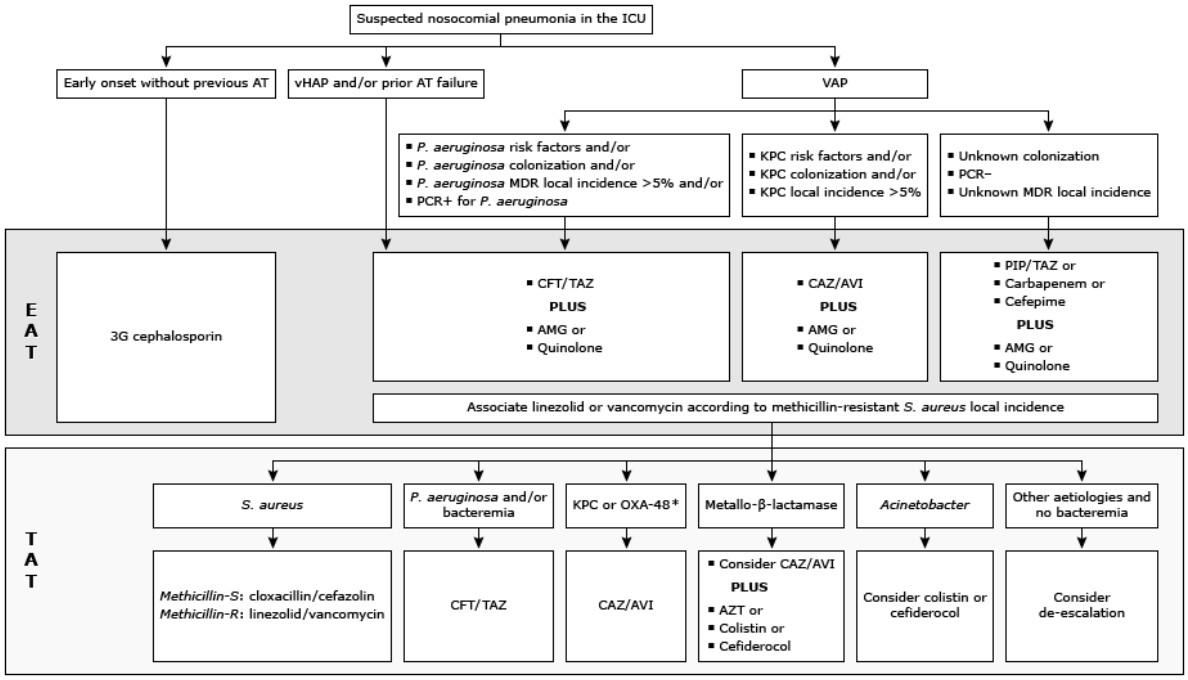


Figure 2: Suspected nosocomial pneumonia in the intensive care unit

Invasive sampling methods for suspected VAP include non-bronchoscopy techniques, such as mini-bronchoalveolar lavage (mini-BAL), and bronchoscopy techniques, including bronchoscopy Bronchoalveolar lavage (BAL) and protected specimen brush (PSB). Among these, bronchoscopy 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 protected specimen brush PSB (and potentially mini-BAL), which provides a dominant alveolar sample with minimal contamination from the upper airways. Several studies have shown that bronchoscopy 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 non-invasive methods like endotracheal aspirates[40,41].

“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 (for example 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[(42)]. 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[(41)]. "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."

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 (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 [(43)].

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.[(44)] 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 [(44)]. 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 (fever, purulent sputum, leukocytosis, and decline in oxygenation), together with a positive pathogen identified on microbiologic respiratory sample[16].

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 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 for two days) following a sustained period of stability or improvement on the ventilator (greater than or equal to two days)" [45].

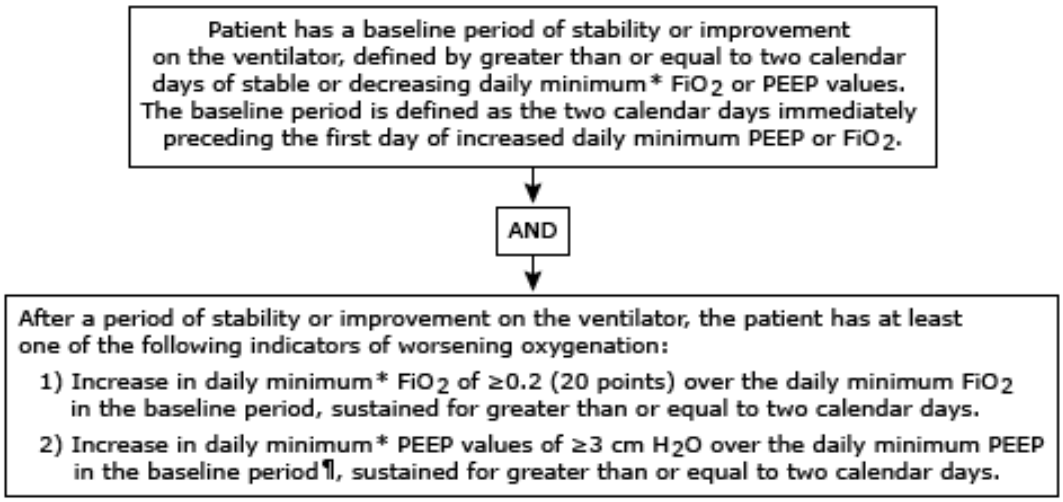


Figure 3: Ventilator associated condition

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 [(44)]. This definition helps to identify patients with potential infections that are complicating their ventilator use.

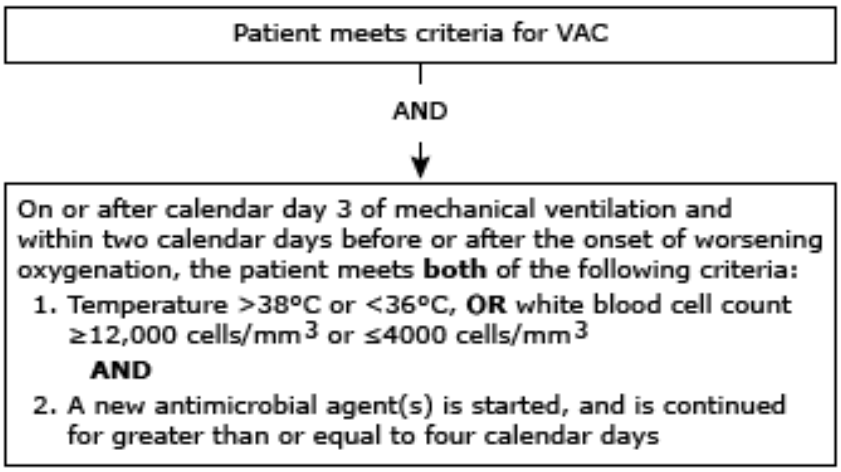


Figure 4: Infection related ventilator associated complication (IVAC)

Potential and likely VAP — The third-tier classifications, possible and probable VAP, require Infection related ventilator associated complication (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.

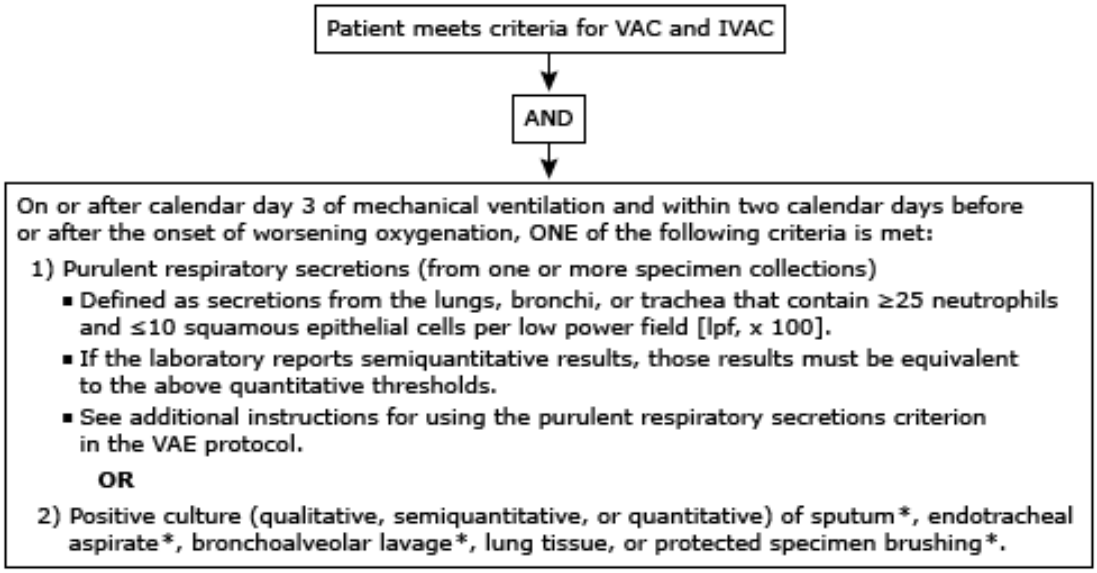


Figure 5: Possible ventilatory associated pneumonia (VAP)

SCORING SYSTEMS USED IN VENTILATOR ASSOCIATED PNEUMONIA (VAP):

Ventilator-associated pneumonia (VAP) is the most frequent infection with high mortality rates in intensive care units (ICUs) and the prediction of outcome is important in the decision-making process.

CLINICAL PULMONARY INFECTION SCORE (CPIS):

A simple tool for the diagnosis of VAP was needed, thus, a scoring system was developed in 1991, which included 7 clinical parameters for VAP diagnosis and it was named as Clinical Pulmonary Infection Score (CPIS) [(10)].

The CPIS is a popular VAP diagnosis method incorporates readily available clinical information. A subsequent study found that the CPIS has a sensitivity of 72–77% and a specificity of 42–85% for diagnosing VAP [(46)].

CPIS points	0	1	2
1. Tracheal secretions	Rare	Abundant	Abundant + Purulent
2. Chest X-ray infiltrates	No infiltrate	Diffuse	Localized
3.Temperature	≥ 36.5 and ≤ 38.4	≥38.5 and ≤38.9	≥39 or ≤36
4. Leukocyte count	≥4000 and ≤ 11,000	<4000 or >11000	< 4000 or > 11000 + band forms ≥500
5. Pa02/FiO2 mmHg	>240 or ARDS		≤ 240 and no evidence of ARDS

Table 6: CPIS scoring system – screening tool for early diagnosis of VAP

In this scoring system, the clinic is evaluated with radiological and endotracheal aspirate (ETA) culture results. The diagnosis of VAP was made using body temperature, leucocyte count and morphology, tracheal secretion amount and character, PaO2 / FiO2 ratio, presence of pulmonary infiltration and its progression and microbiological culture results. A score of 6 or more suggests VAP [(46)].

Clinical management employs modified CPIS in an effort to minimize the needless use of antibiotics in patients with suspected VAP. In this patient series, stopping antibiotics was safe if the modified score remained less than 6 at baseline and after three days.

CPIS points	0	1	2
1. Tracheal secretions	Rare	Abundant	Abundant +

			Purulent
2. Chest X-ray infiltrates	No infiltrate	Diffuse	Localized
3.Temperature	≥ 36.5 and ≤ 38.4	≥38.5 and ≤38.9	≥39 or ≤36
4. Leukocyte count	≥4000 and ≤ 11,000	<4000 or >11000	< 4000 or > 11000 + band forms ≥500
5. Pa02/FiO2 mmHg	>240 or ARDS		≤ 240 and no evidence of ARDS
6. Microbiology	Negative		Positive

Table 7: Modified CPIS scoring system

ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION II (APACHE II):

APACHE II ("Acute Physiology and Chronic Health Evaluation II") score is a disease severity classification system, which is one of the most widely used scores in medical or surgical intensive care unit (ICU) [(47)]. The severity of disease at the time of intubation, measured by APACHE II, is used to define risk for future development of VAP [(48)].

When it came to predicting 30-day mortality in patients with VAP, APACHE II demonstrated strong discrimination and calibration. We think that the primary cause of this is because the CPIS was created for the clinical setting, while the APACHE II was intended as a severity-of-disease categorization.

Age points

Age	Points

<44y	0
45-54y	2
55-64y	3
65-74y	5
>75y	6

Chronic health points

Non-operative or emergency postop and any conditions below	5
Elective operation and any conditions below*	2
**cirrhosis with portal hypertension or encephalopathy; class IV angina, chronic hypoxia, increaseCO2; Chronic dialysis; immunocompromised	

Acute physiologic score (0-4 points)

1.Temperature

2. Mean arterial pressure

3. Heart rate

4. Oxygenation

5. Respiratory rate

6. Arterial pH

7. HC03

8. Potassium

9. Sodium

10. Serum creatinine

11. Hematocrit

12. TLC

13. GCS

Score	Mortality
0-4	4%
5-9	4%
10-14	15%
15-19	25%
20-24	40%
25-29	55%
30-34	75%
>34	85%

Chronic Health points: If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

- 5 points for nonoperative or emergency postoperative patients
- 2 points for elective postoperative patients

To compute APACHE II Score: Sum points: AP + APS + CHP

ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION IV (APACHE IV):

The length of stay (LOS) in the intensive care unit (ICU) for critically sick patients can be predicted using the Acute Physiology and Chronic Health Evaluation (APACHE) IV model[51] helpful in anticipating intensive care unit length of stay for sepsis patients. For forecasting ICU length of stay in critically ill patients, it has been updated [52]. APACHE IV predicts each patient's mortality and intensive care unit length of stay using multivariate linear regression and demographic information, entrance diagnosis, and physiological abnormalities [52].

Acute physiologic score

Age

Temperature (C)

MAP (mmHg)

HR (/min)

RR(/min)

Mechanical Ventilation

Fio2 (%)

Po2 (mmHg)

Pco2 (mmHg)

Arterial PH

Sodium (mEq/L)

Urine Output (mL/24hrs)

Creatinine (mg/dL)

Urea (mEq/L)

BSL (mg/dl)

Albumin (g/L)

Bilirubin (mg/dl)

Hematocrit (%)

WBC (X1000/mm3)

GCS

Admission information

Pre – ICU LOS (days)

Origin

Readmission

Emergency Surgery

Admission diagnosis

Postoperative

Nonoperative

Chronic Health Condition

- CRF
- Cirrhosis
- Hepatic failure
- Metastatic carcinoma
- Lymphoma
- Leukaemia / myeloma
- Immunosuppression
- AIDS

SEQUENTIAL ORGAN ASSESSMENT (SOFA) SCORE:

The Sequential Organ Failure Assessment (SOFA) score is based on the degree of dysfunction in six organ systems—respiratory system, coagulation, hepatic, cardiovascular system, central nervous system, and renal [53]. The Sequential Organ Failure Assessment (SOFA) score was developed to provide a simple method of assessing and monitoring organ dysfunction in critically ill patients.

	0	1	2	3	4
Respiratory Pao2/Fio2 mmHg	>400	≤ 400	≤ 300	≤ 200	≤ 100
Coagulation Platelets x	>150	≤ 150	≤ 100	≤ 50	≤ 20

1000/mm3					
Liver Bilirubin mg/dl	< 1.2 (< 20)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	> 12.0 (> 204)
Cardiovascular Hypotension	No hypotension	MAP < 70 m mHg	Dopamine ≤ 5 or dobutamine (any dose) *	Dopamine > 5 or epinephrine ≤ 0.1 or norepinephrine ≤ 0.1*	Dopamine > 15 or epinephrine > 0.1 or norepinephrine > 0.1*
Central nervous system GCS	15	13-14	10-12	6-9	<6
Renal Creatinine	< 1.2 (< 110)	1.2–1.9 (110–170)	2.0–3.4 (171–299)	2.0–3.4 (171–299)	> 5.0 (> 440)
OR urine output				< 500 ml/d	< 200 ml/d

*Adrenergic agents administered for at least one hour

Table 8: Sequential organ assessment (SOFA) scoring system

MECHANICAL VENTILATION:

For patients with hypoxemic respiratory failure, compromised airways, or impaired ventilation, mechanical ventilation is an essential emergency intervention. Positive pressure breathing is used in this procedure, which depends on the airway system's compliance and opposition. Airway compromise, or individuals with dynamic airways, airway blockage, hypoventilation, and hypoxemia as a result of numerous pulmonary and systemic diseases, are

important indications for invasive mechanical ventilation.

Objectives of Mechanical Ventilation:

- To identify key indications for invasive mechanical ventilation in patients with compromised airways, impaired ventilation and respiratory failure.
- To implement safe and effective mechanical ventilation strategies.
- Depending on the patient's state, choose the right ventilator modes, tidal volumes, respiratory rates, and positive end-expiratory pressure levels [54,55].

Indications for Invasive Mechanical Ventilation:

- Airway compromise
    1. Patients with dynamic airways, such as trauma or oropharyngeal infection to protect airway.
    2. An acute exacerbation of chronic obstructive pulmonary disease or proximal involvement such as angioedema or distal involvement such as bronchospasm can occur in patients with airway blockage.[55].
- Hypoventilation, which can be brought on by inadequate drive, pump failure, or issues with gas exchange, can result in hypercapnic respiratory failure. This can be divided into the following groups:
    1. Drug overdose-related central drive impairment
    2. Respiratory muscle weakness (muscular dystrophy and myositis)
    3. Peripheral nerve system abnormalities, including Guillain-Barré syndrome or myasthenic crises

4. Restrictive defects (chest wall disease, pneumothorax, or pleural effusion)

- Hypoxemic respiratory failure brought on by ineffective oxygen exchange or delivery to peripheral tissues because of: o Defects in alveolar filling, such as acute respiratory distress syndrome and pneumonia (ARDS), or edema of the lungs.
    1. Pulmonary vascular abnormalities, such as major pulmonary embolism or air emboli, that result in ventilation-perfusion mismatch (V/Q).
    2. Advanced pulmonary fibrosis is one example of a diffusion deficiency [56,57].
- Increased ventilatory demand like severe sepsis, shock, or severe metabolic acidosis

The amount of air exchanged during each respiratory cycle is known as the tidal volume (VT) [58]. Height and gender determine VT, which typically ranges between 8 and 10 mL/kg of ideal weight of the body (IBW).

Mechanical ventilation can be applied through different modes, mandatory or assisted modes. In the assisted mode, the patient's inspiratory effort will trigger the mechanical ventilation to deliver the breath.

The most frequent modes of mechanical ventilation:

- Volume-limited assist control (VAC) ventilation
- Pressure-limited assist control (PAC) ventilation
- Synchronized intermittent mandatory ventilation (SIMV) with pressure support ventilation (PSV)

Other different types of modes are controlled mechanical ventilation, which can be volume-limited or pressure-limited, or IMV (intermittent mandatory ventilation) [59].

Tidal volume is adjusted to a fixed amount in volume limited-assist control mode (VAC), with the static airway pressure influenced by lung compliance [60]. In Pressure limited assist control (PAC) mode, the driving pressure is fixed, which results in variable Tidal volume V_T. Higher lung compliance leads to higher V_T, and lower lung compliance leads to lower V_T [61].

Mechanical ventilation has four stages:

1. Trigger phase,
2. Inspiratory phase,
3. Cycling phase and the
4. Expiratory phase

Trigger phase initiates inspiration, by the patient's effort or preset parameters by the mechanical ventilator. The inspiratory phase involves intake of air into the lungs. Following inspiration, in the cycling phase cessation of inspiration takes place but precedes the onset of exhalation. At last, the expiratory phase signifies the passive expiration of air from the patient's lungs.

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 [62].

In a study conducted by Hugonnet S et al., (2007) to assess the staffing level a determinant of

late onset ventilator associated pneumonia 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 [(63)].

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 [(64)].

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 [(65)].

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 traumas, 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%) [(66)].

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 [(67)].

In a review study conducted by Wu D et al., (2019) to assess the risk factors for 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 [68].

MATERIALS AND METHODS

SOURCE OF DATA:

All patients admitted in the RICU, MICU and SICU who were Mechanically ventilated in B.L.D.E(DEEMED TO BE UNIVERSITY)'s, Shri B. M. Patil Medical College, Hospital and Research Centre, Vijayapura were included in the study.

METHOD OF COLLECTION OF DATA:

Study Design: Cross sectional study

Study Period: Two Years

Study Sample :126 VAP Patients (63 patients in Pulmonary group, 63 patients in Non-pulmonary group)

Inclusion criteria

- Patients willing to give informed consent.
- Patients aged above 12 years
- Patients who were mechanically ventilated

Exclusion criteria

- Patient unwilling to give informed consent.
- Pregnant and lactating women.
- Patient aged <12 years

Patients included in the study will be evaluated daily in the ICU. Baseline Chest X-ray will be done immediately after Intubation or Tracheostomy and Chest X-ray after 48 hours will be repeated and compared. Any new pulmonary lesion will be considered as VAP according to the ATS/IDSA Guidelines. Patients admitted in Shri. B. M. Patil Medical College and Hospital, and developing VAP post mechanical ventilation for various causes (pulmonary vs non pulmonary) were enrolled in the study. Course in the hospital of all patients developing VAP followed up till the discharge of the patients. ET tube secretions, Tracheostomy tube secretions will be sent for Gram's stain and Culture and Sensitivity for isolation of organism and resistance pattern.

Predictors of severity like Apache-II score, SOFA score, and CPIS will be calculated

and analyzed. Data collected will be analyzed by comparison of VAP between pulmonary and non-pulmonary indications for Mechanical Ventilation. Incidence, organism, resistance pattern, outcomes like improvement/ death/progression will be analyzed. Apache-II score, SOFA score, and CPIS will be compared and outcomes will be analyzed.

Statistical Analysis

- The data obtained is entered in a Microsoft Excel sheet, and statistical analysis is performed using a statistical package for the social sciences (Version 20).
- Results will be presented as Mean ±SD, Median and interquartile range, frequency, percentages and diagrams.
- For normally distributed continuous variables between two groups will be compared using independent t-test and for not normally distributed variables, Mann Whitney U test will be used.
- Categorical variables between two groups will be compared using the Chi-square test/Fisher\s Exact test.
- $P < 0.05$ will be considered statistically significant. All statistical tests will perform two-tailed.

RESULTS

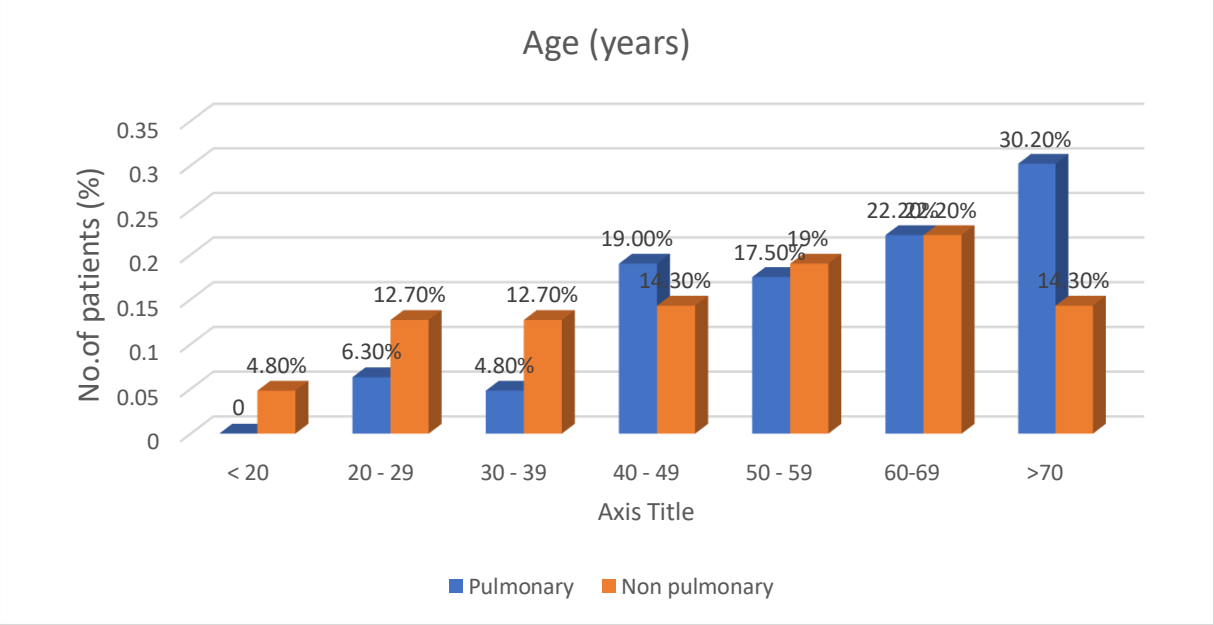
NUMBER OF PATIENTS IN EACH GROUP:

	Pulmonary	Non pulmonary	Total
NUMBER OF PATIENTS IN EACH GROUP	63	63	126

AGE DISTRIBUTION:

The distribution of patients according to different age groups in two indications is depicted below with majority of patients in both the groups being between the age group of above 60. The mean age in pulmonary indication is 58 ± 16.4 and in Non-pulmonary indication is 49.5 ± 18.03 respectively

GRAPH 1: AGE DISTRIBUTION BETWEEN PULMONARY AND NON PULMONARY INDICATIONS OF MV



GENDER DISTRIBUTION

Majority of the patients in the study were male and gender distribution in both groups was similar to each other.

TABLE 9: GENDER DISTRIBUTION OF VAP AMONG PULMONARY AND NON-PULMONARY INDICATIONS OF MV

	Pulmonary	Non pulmonary	Total
MALE	45(71.4%)	48(76.1%)	93(73.8%)
FEMALE	18(28.5%)	15(23.8%)	33(26.1%)

INCIDENCE OF VAP

Of the 254 patients who were on mechanical ventilation for more than 48 hours in ICU, the incidence of VAP in pulmonary group was 47% and Non pulmonary group was 52.5%.

TABLE 10: DISTRIBUTION OF INCIDENCE OF VAP AMONG PULMONARY AND NON-PULMONARY INDICATIONS OF MV

	No. of patients with VAP n %	No. of patients without VAP n %	TOTAL
NON-PULMONARY	63(47%)	57	120
PULMONARY	63(52.5%)	71	134
TOTAL	126	128	254

Non pulmonary group were nearly 1.3 times more likely to develop VAP than pulmonary group (odds ratio: 1.246; 95% CI: 0.7605 - 2.04; p = 0.1933).

GRAPH 2: DISTRIBUTION OF DIAGNOSIS AT THE TIME OF ADMISSION IN PULMONARY INDICATIONS OF MV

Among the 63 cases of pulmonary group, COPD is the most frequent etiology at 25.39%, followed by CAP at 19.04%, Post TB sequelae and Pulmonary TB each at 14.28%, with lower frequencies for ILD 9.52%, Carcinoma lung 6.34%, Asthma 4.76%, MDRTB 3.17%, Kyphoscoliosis and OSA each at 1.58%. The distribution is given below in the graph

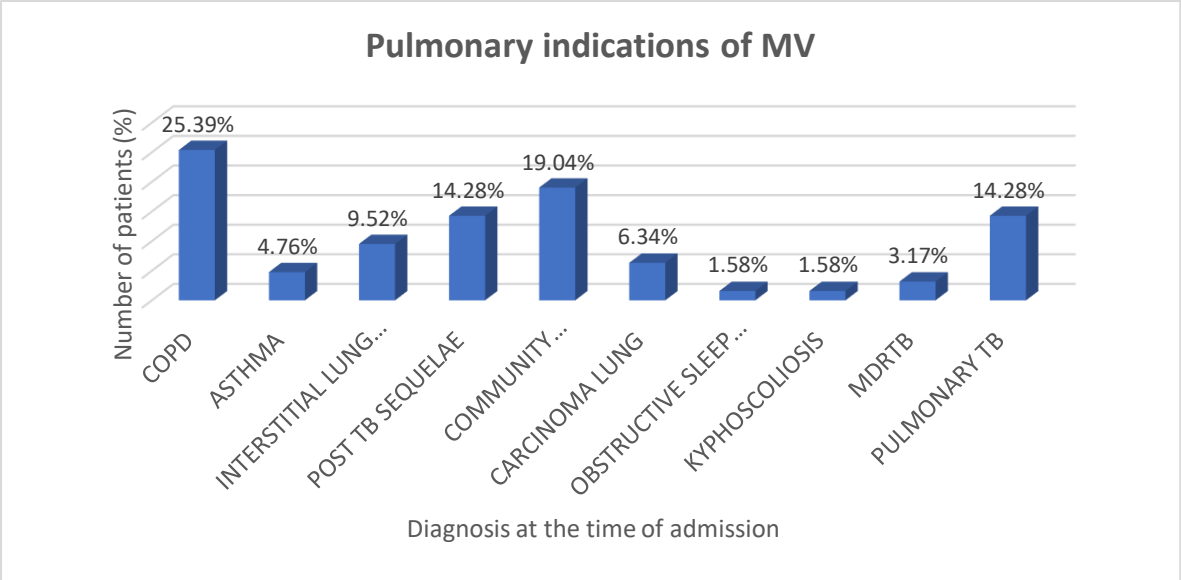


TABLE 11: DIAGNOSIS AT THE TIME OF ADMISSION IN PULMONARY INDICATION OF MV

Diagnosis the time of admission	No. of patients in pulmonary indications of MV n (%)
COPD	16 (25.39%)
Asthma	3 (4.76%)
Interstitial lung disease	6 (9.52%)
Post TB sequelae	9 (14.28%)
Community acquired Pneumonia (CAP)	12 (19.04%)
Carcinoma lung	4 (6.34%)
Obstructive Sleep Apnea	1 (1.58%)
Kyphoscoliosis	1 (1.58%)
MDRTB	2 (3.17%)
Pulmonary Tuberculosis	9 (14.28%)

GRAPH 3: DISTRIBUTION OF DIAGNOSIS AT THE TIME OF ADMISSION IN PULMONARY INDICATIONS OF MV

Among the 63 cases of Non pulmonary group, Traumatic brain injury is the most frequent

etiology at 28.57%, followed by Sepsis at 17.46% and Stroke 12.70%, Burns, OP poisoning and Chronic liver disease each at 6.35% and with lower frequencies for CKD, IHD, Meningoencephalitis, Neurotoxic snake bite each at 4.76%, Jejunal perforation and Parkinsons disease each at 1.59%. The distribution is given below in the graph

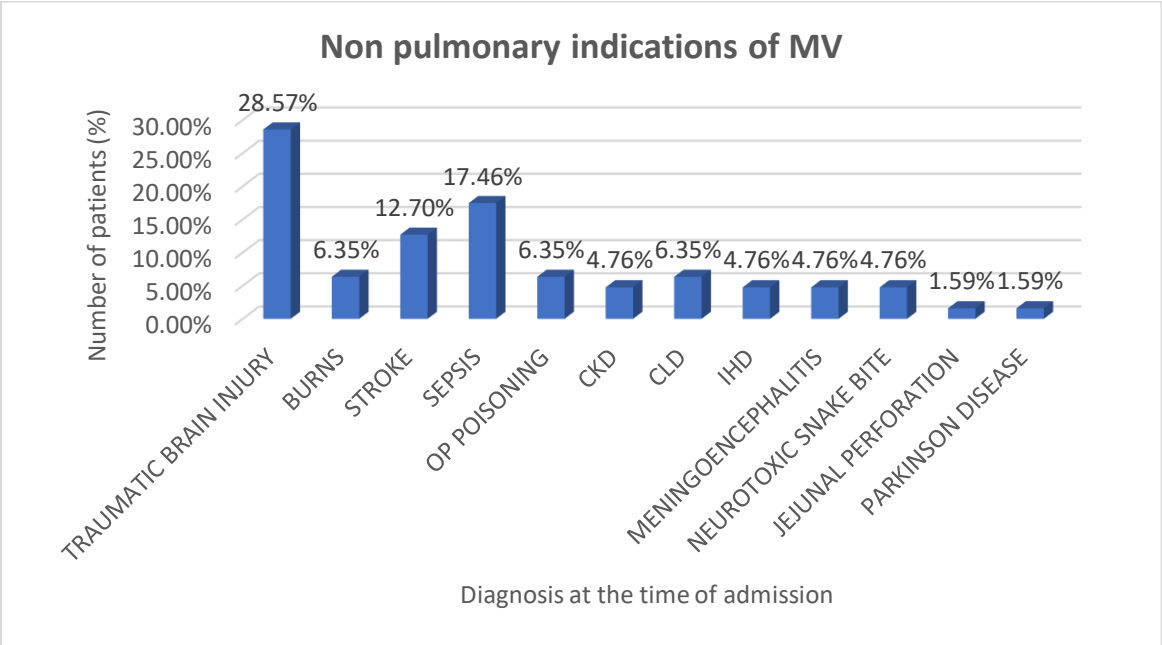


TABLE 12: DIAGNOSIS AT THE TIME OF ADMISSION IN NON-PULMONARY INDICATIONS OF MV

Diagnosis the time of admission	No. of patients in pulmonary group n (%)
Traumatic brain injury (TBI)	18 (28.57%)
Burns	4 (6.35 %)
Stroke	8 (12.70%)
Sepsis	11 (17.46%)
OP poisoning	4 (6.35%)
CKD	3 (4.76%)
CLD	4 (6.35%)
Congestive heart failure	3 (4.76%)
Meningoencephalitis	3 (4.76%)
Neurotoxic snake bite	3 (4.76%)
Jejunal perforation	1(1.59%)
Parkinson disease	1 (1.59%)

TABLE 13: DISTRIBUTION OF RIGHT LUNG INVOLVEMENT IN CHEST X-RAY FOR DIAGNOSING VAP IN BOTH GROUPS

Out of 126 cases of VAP, 29 cases (46%) in pulmonary group and 36 cases (57.1%) in Non-pulmonary group shows right lung involvement in diagnosing VAP.

Right	Pulmonary	Non pulmonary	Total	Chi square test	P value
UPPER ZONE	10(15.8%)	4(6.3%)	14(11.1%)	2.8699	P=0.0903
MID ZONE	8(12.6%)	15(23.8%)	23(18.2%)	2.5855	P=0.1078
LOWER ZONE	11(17.4%)	17(26.9%)	28(22.2%)	1.6399	P = 0.2003
	29(46%)	**36(57.1%)**	**65(51.5%)**	**1.5448**	**P = 0.2139**

TABLE 14: DISTRIBUTION OF LEFT LUNG INVOLVEMENT IN CHEST X-RAY FOR DIAGNOSING VAP IN BOTH GROUPS

Out of 126 cases of VAP, 36 cases (57.1%) in pulmonary group and 30 cases (47.6%) in Non-pulmonary group shows left lung involvement in diagnosing VAP.

Left	Pulmonary	Non pulmonary	Total	Chi square test	P value
UPPER ZONE	11(17.4%)	4(6.3%)	15(11.9%)	3.6787	P = 0.05*
MID ZONE	8(12.6%)	4(6.3%)	12(9.5%)	1.4620	P = 0.2266
LOWER ZONE	17(26.9%)	22(34.9%)	39(30.9%)	0.9210	P = 0.3372
	36(57.1%)	30(47.6%)	66(52.3%)	1.1364	P=0.2864
*Statistically significant					

There is positive correlation between left upper zone involvement in diagnosing VAP between two groups with more number of cases in left upper zone involvement in pulmonary group than Non pulmonary group and is statistically significant with p value of 0.05 from chi square test.

TABLE 15: DISTRIBUTION OF BILATERAL INVOLVEMENT OF LUNGS IN CHEST X-RAY FOR DIAGNOSING VAP IN BOTH GROUPS

	Pulmonary	Non pulmonary	Total	Chi square test	P value

BILATERAL INVOLVEMENT	18(28.5%)	8(12.6%)	26(20.6%)	4.8077	P = 0.0283*
*Statistically significant					

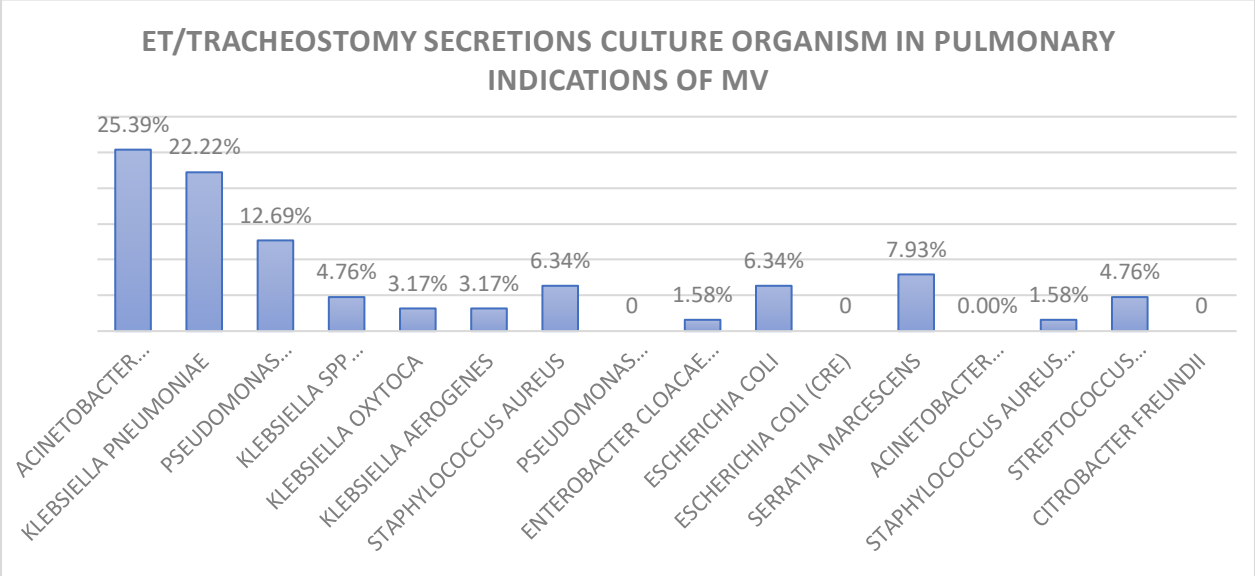
There is positive correlation between bilateral involvement of lungs in diagnosing VAP between two groups with more number of cases in pulmonary group than Non pulmonary group and is statistically significant with p value of 0.0283 from chi square test.

GRAPH 4 : DISTRIBUTION OF ET/TRACHEOSTOMY SECRETIONS CULTURE ORGANISM AMONG VAP CASES IN PULMONARY INDICATIONS OF MV

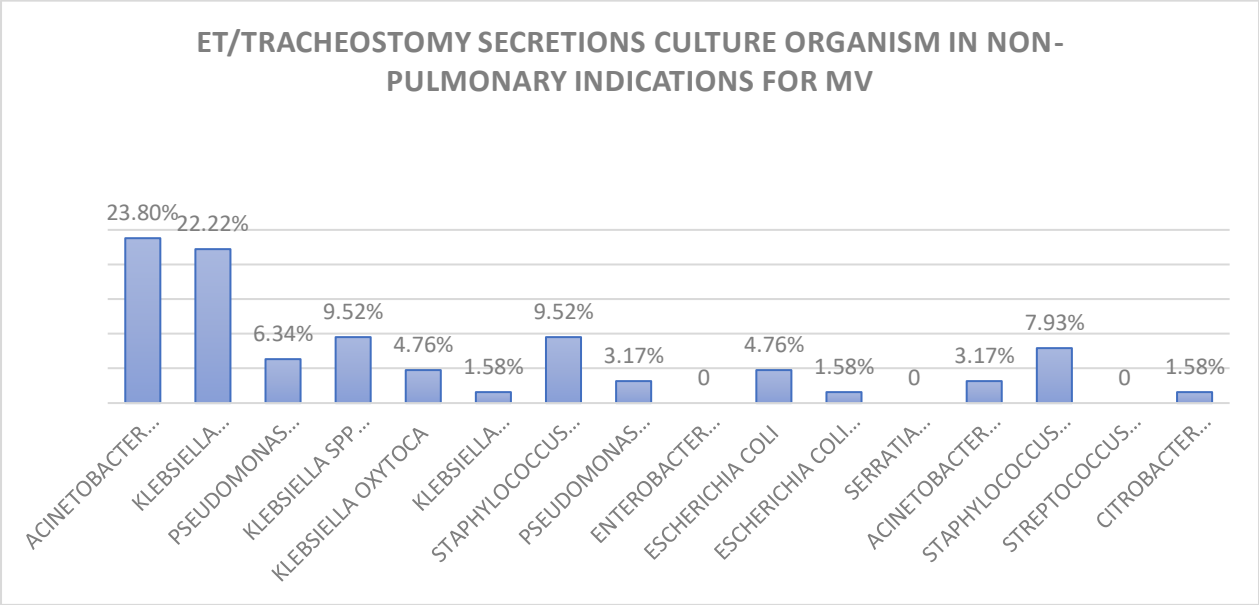
Most Common VAP-Causing Organisms in Pulmonary Group (n=63)

Among the pulmonary cases, the three most frequently isolated pathogens were Acinetobacter baumannii Complex (16 cases, 25.39%), Klebsiella pneumoniae (14 cases, 22.2%), and Pseudomonas aeruginosa (8 cases, 12.69%).

There is positive correlation between Serratia marcescens between two groups with more number of organisms isolated in pulmonary group and is statistically significant with p value of 0.0230 from chi square test.



GRAPH 5 : DISTRIBUTION OF ET/TRACHEOSTOMY SECRETIONS CULTURE ORGANISM AMONG VAP CASES IN PULMONARY INDICATIONS OF MV



Most Common VAP-Causing Organisms in Non-Pulmonary Group (n=63)

In the non-pulmonary group, Acinetobacter baumannii Complex was again the most prevalent pathogen, accounting for 15 cases (23.8%), followed closely by Klebsiella pneumoniae with 14 cases (22.2%). Klebsiella pneumoniae (MDRO) and Staphylococcus aureus were each found in 6 cases (9.52%), while Staphylococcus aureus (MRSA) was present in 5 cases (7.93%).

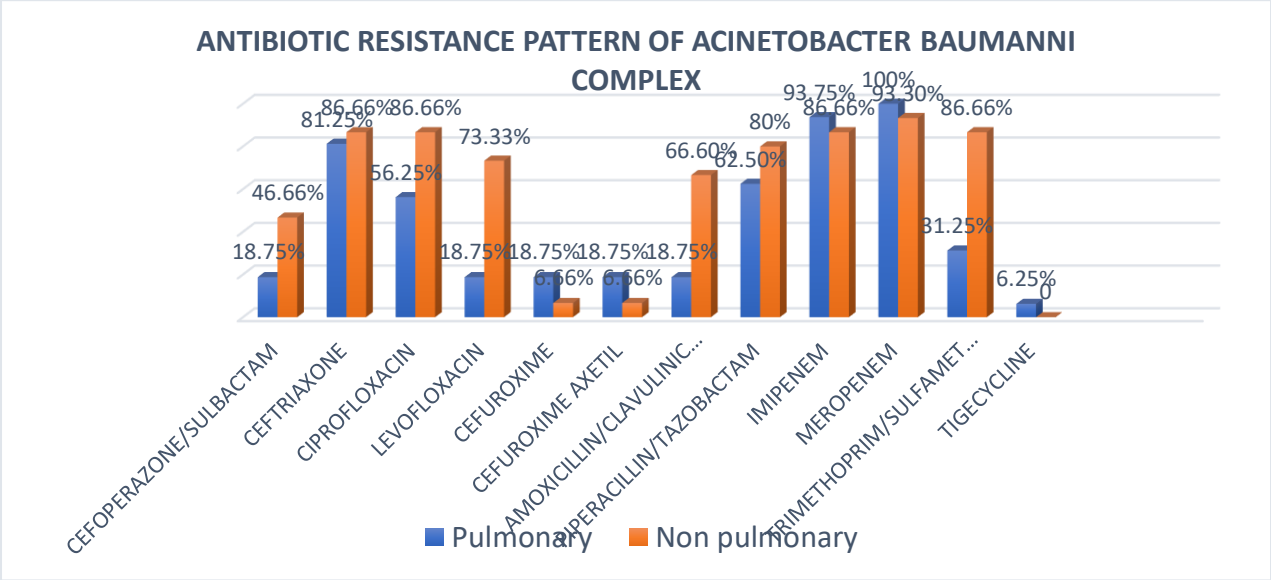
TABLE 16: DISTRIBUTION OF ET/TRACHEOSTOMY SECRETIONS CULTURE ORGANISM AMONG VAP CASES IN PULMONARY AND NON PULMONARY INDICATIONS OF MV

ENDOTRACHEAL TUBE SECRETIONS/TRACHEOSTOMY TUBE SECRETIONS CULTURE ORGANISM	Pulmonary	Non pulmonary	Total	Chi square test	P value
ACINETOBACTER BAUMANNI COMPLEX	16(25.39%)	15(23.80%)	31(24.60%)	0.0424	P=0.8368
ACINETOBACTER BAUMANNII (MDR)	0	2(3.17%)	2(2.38%)	2.0161	P=0.1556
KLEBSIELLA PNEUMONIAE	14(22.22%)	14(22.22%)	28(22.22%)	0	P=1.000
KLEBSIELLA SPP PNEUMONIAE (MDRO)	3(4.76%)	6(9.52%)	9(7.14%)	1.0684	P=0.3013
KLEBSIELLA OXYTOCA	2(3.17%)	3(4.76%)	5(3.96%)	0.2066	P=0.6494

KLEBSIELLA AEROGENES	2(3.17%)	1(1.58%)	3(2.38%)	0.1501	P=0.6985
PSEUDOMONAS AERUGINOSA	8(12.69%)	4(6.34%)	12(9.52%)	1.462	P=0.2266
PSEUDOMONAS AERUGINOSA (MDR)	0	2(3.17%)	2(1.58%)	2.0161	P=0.1556
ENTEROBACTER CLOACAE COMPLEX	1(1.58%)	0	1(0.79%)	1	P=0.3173
ESCHERICHIA COLI	4(6.34%)	3(4.76%)	7(5.55%)	0.1501	P=0.6985
ESCHERICHIA COLI (CRE)	0	1(1.58%)	1(0.79%)	1	P=0.3173
SERRATIA MARCESCENS	5(7.93%)	0	5(3.96%)	5.1653	**P=0.0230***
STAPHYLOCOCCUS AUREUS	4(6.34%)	6(9.52%)	10(7.93%)	0.431	P=0.5115
STAPHYLOCOCCUS AUREUS (MRSA)	1(1.58%)	5(7.93%)	6(4.76%)	2.7778	P=0.0956
STREPTOCOCCUS PNEUMONIAE	3(4.76%)	0	3(2.38%)	3.0488	P=0.0808
CITROBACTER FREUNDII	0	1(1.58%)	1(0.79%)	1	P=0.3173
TOTAL	63(100%)	63(100%)	126(100%)		
*Statistically significant					

GRAPH 6: DISTRIBUTION OF ACINETOBACTER BAUMANNI COMPLEX AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY & NON-PULMONARY INDICATIONS OF MV

Levofloxacin, Amoxicillin/Clavulanic Acid and Trimethoprim/Sulfamethoxazole resistance was significantly higher in the non-pulmonary group with p = 0.0023, p = 0.0020 and p = 0.0018. Tigecycline and Trimethoprim/Sulfamethoxazole sensitivity was significantly higher in the non-pulmonary group p = 0.0082 and p = 0.0181 as shown below



GRAPH 7: DISTRIBUTION OF ACINETOBACTER BAUMANNI COMPLEX AND

ITS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY & NON-PULMONARY GROUPS

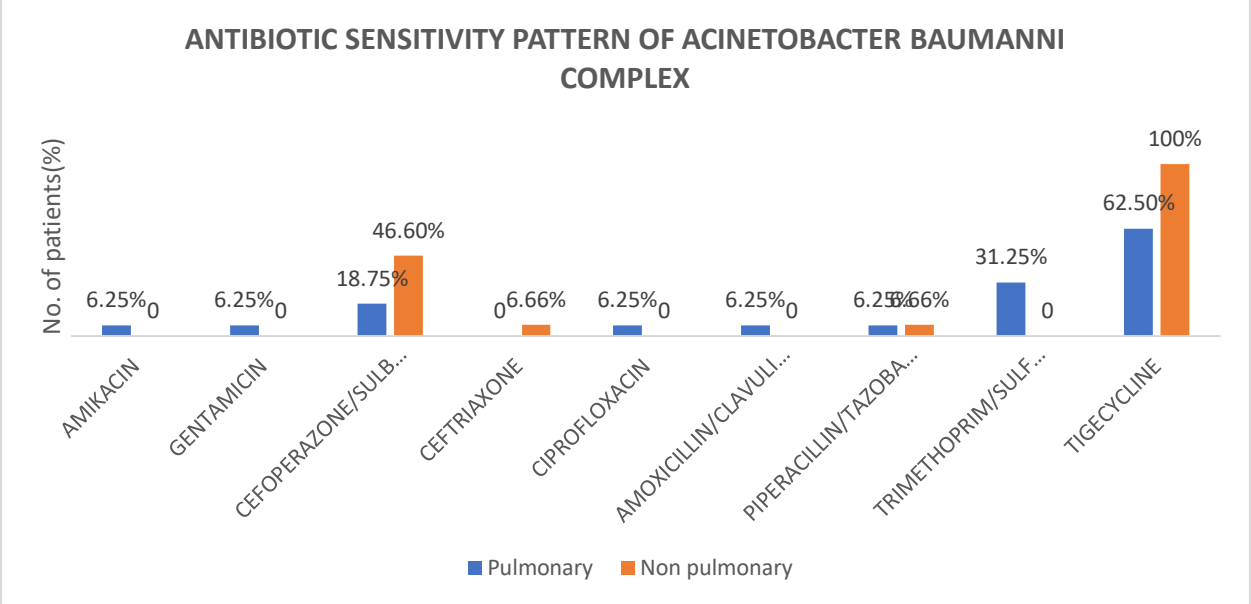


TABLE 17: DISTRIBUTION OF ACINETOBACTER BAUMANNI COMPLEX AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

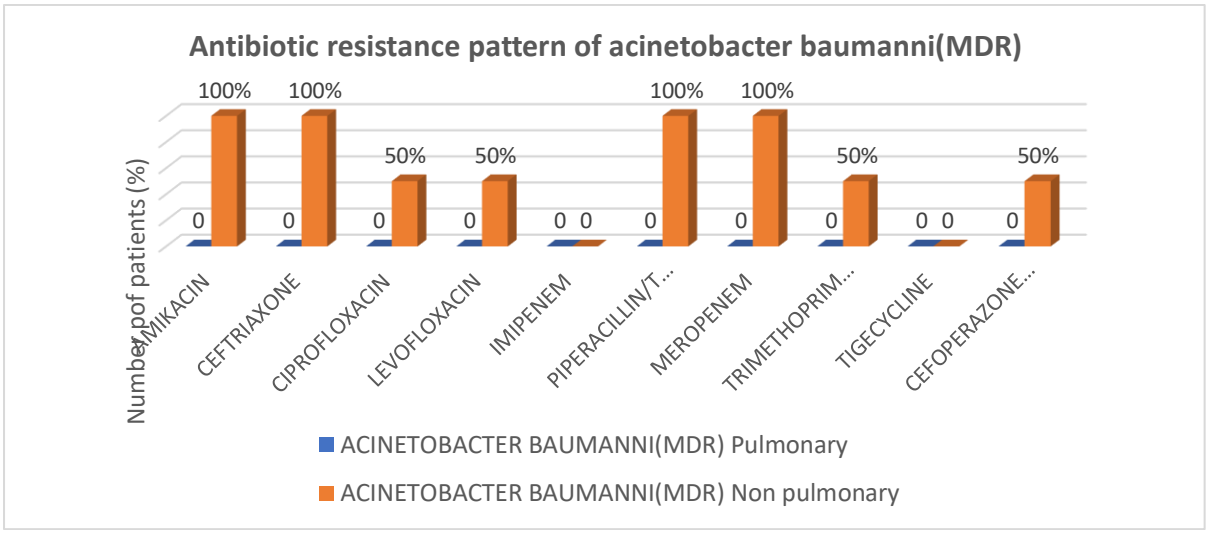
ACINETOBACTER BAUMANNI COMPLEX	Pulmonary	Non-pulmonary		Total		P value
	16	15		31		
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary	P value	Pulmonary	Non pulmonary	
AMIKACIN	11(68.75%)	13(86.66%)	P=0.2331	1(6.25%)	0	P=0.3250
GENTAMICIN	11(68.75%)	13(86.66%)	P=0.2331	1(6.25%)	0	P=0.3250
CEFOPERAZONE/SULBACTAM	3(18.75%)	7(46.66%)	P=0.0966	3(18.75%)	7(46.6%)	P=0.0966
CEFTRIAXONE	13(81.25%)	13(86.66%)	P=0.6820	0	1(6.66%)	P=0.3096
CIPROFLOXACIN	9(56.25%)	13(86.66%)	P=0.0622	1(6.25%)	0	P=0.3250
LEVOFLOXACIN	3(18.75%)	11(73.33%)	**P=0.0023***	0	0	NA
CEFUROXIME	3(18.75%)	1(6.66%)	P=0.3159	0	0	NA
CEFUROXIME AXETIL	3(18.75%)	1(6.66%)	P=0.3159	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	2(18.75%)	10(66.6%)	**P=0.0020***	1(6.25%)	0	P=0.3250
PIPERACILLIN/TAZOBACTAM	10(62.5%)	12(80%)	P=0.2834	1(6.25%)	1(6.66%)	P=0.9624
IMIPENEM	15(93.75%)	13(86.66%)	P=0.5050	0	0	NA
MEROPENEM	16(100%)	14(93.3%)	P=0.2938	0	0	NA
TRIMETHOPRIM/SULFAMETHOXAZOLE	5(31.25%)	13(86.66%)	**P=0.0018***	5(31.25%)	0	**P=0.0181***

TIGECYCLINE	1(6.25%)	0	P=0.3250	10(62.5%)	15(100%)	**P=0.0082***
*Statistically significant						

GRAPH 8: DISTRIBUTION OF ACINETOBACTER BAUMANNI MDR AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY & NON-PULMONARY INDICATIONS OF VAP

Among 126 cases of VAP, 2 cases (6.45%) were identified as Acinetobacter baumanni MDR strains, both of which were from the **non-pulmonary group**.

Both MDR cases were completely resistant to Amikacin (100%), Ceftriaxone (100%), Piperacillin/Tazobactam (100%) and Meropenem (100%).



GRAPH 9: DISTRIBUTION OF ACINETOBACTER BAUMANNI MDR AND ITS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY & NON-PULMONARY INDICATIONS OF MV

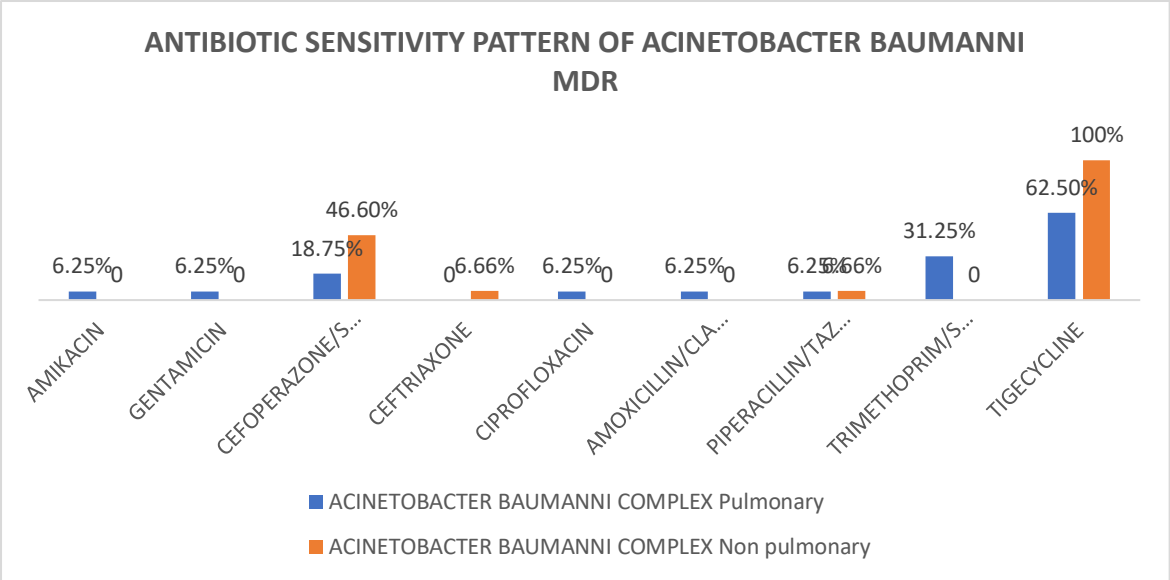


TABLE 18: DISTRIBUTION OF ACINETOBACTER BAUMANNI MDR AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

Since all MDR cases were from the non-pulmonary group, a chi-square test for significance was not applicable for comparing pulmonary and non-pulmonary cases

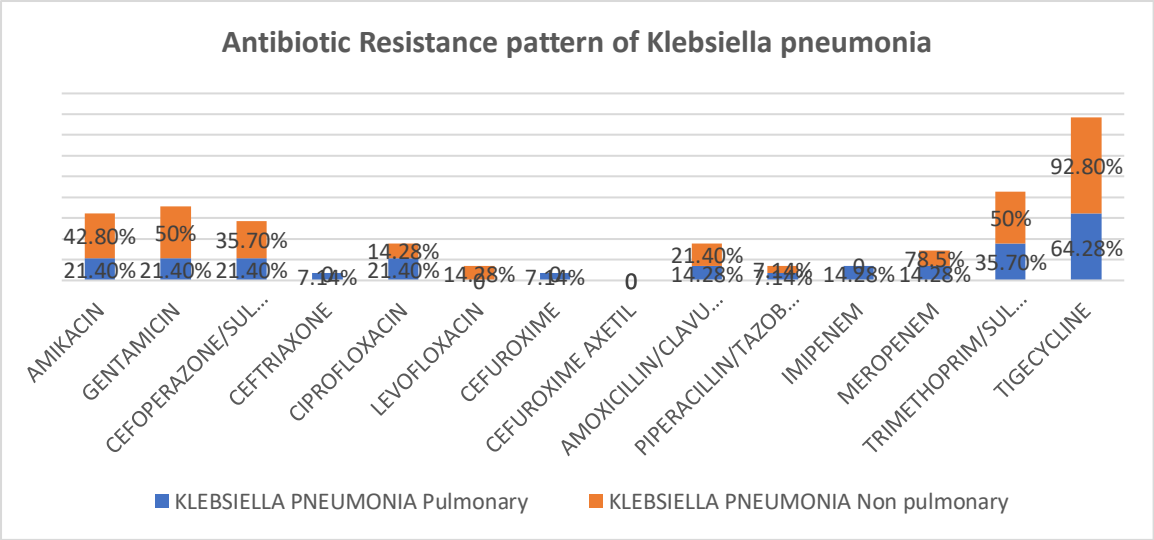
ACINETOBACTER BAUMANNI(MDR)	Pulmonary	Non pulmonary	Total			Chi square test p value
	0	2	2			
	ANTIBIOTIC RESISTANCE		ANTIBIOTIC SENSITIVITY			
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	0	2(100%)	NA	0	0	NA
CEFTRIAXONE		2(100%)			0	
CIPROFLOXACIN		1(50%)			0	
LEVOFLOXACIN		1(50%)			0	
IMIPENEM		0			0	
PIPERACILLIN/TAZOBACTAM		2(100%)			0	
MEROPENEM		2(100%)			0	
TRIMETHOPRIM/SULFAMETHOXAZOLE		1(50%)			1(50%)	
TIGECYCLINE		0			1(50%)	
CEFOPERAZONE/SULBACTAM		1(50%)			1(50%)	

TABLE 19: DISTRIBUTION OF KLEBSIELLA PNEUMONIA AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

A total of 28 cases of *Klebsiella pneumoniae* infections were analyzed, with 14 cases in each group. Antibiotic resistance and sensitivity patterns were compared between these groups, and statistical significance was assessed using the Chi-square test.

KLEBSIELLA PNEUMONIA	PULMONARY	NON-PULMONARY		TOTAL		Chi square test p value
	14	14		28		
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	6(42.8%)	7(50%)	p=0.701	3(21.4%)	6(42.8%)	P=0.2332
GENTAMICIN	5(35.7%)	5(35.7%)	p=1.001	3(21.4%)	7(50%)	P=0.1233
CEFOPERAZONE/SULBACTAM	8(57.14%)	4(28.57%)	p=0.132	3(21.4%)	5(35.7%)	P=0.2132
CEFTRIAXONE	13(92.85%)	13(92.85%)	p=1.000	1(7.14%)	0	P=0.2131
CIPROFLOXACIN	5(35.7%)	10(71.42%)	p=0.061	3(21.4%)	2(14.28%)	P=0.612
LEVOFLOXACIN	3(21.4%)	9(64.28%)	p=0.2312	0	2(14.28%)	P=0.4213
CEFUROXIME	8(57.14%)	0	**p=0.001***	1(7.14%)	0	P=0.213
CEFUROXIME AXETIL	8(57.14%)	0	**p=0.001***	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	3(21.4%)	10(71.42%)	**p=0.009***	2(14.28%)	3(21.4%)	P=0.321
PIPERACILLIN/TAZOBACTAM	11(78.5%)	1(7.14%)	**p=0.0002***	1(7.14%)	1(7.14%)	P=1.00
IMIPENEM	1(7.14%)	7(50%)	**p=0.01***	2(14.28%)	0	P=0.432
MEROPENEM	12(85.71%)	11(78.5%)	p=0.701	2(14.28%)	2(14.28%)	P=1.00
TRIMETHOPRIM/SULFAMETHOXAZOLE	6(42.8%)	5(35.7%)	p=0.612	5(35.7%)	7(50%)	P=0.623
TIGECYCLINE	1(7.14%)	0	p=0.213	9(64.28%)	13(92.85%)	P=0.07
*Statistically significant						

GRAPH 10: DISTRIBUTION OF KLEBSIELLA PNEUMONIA AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY & NON-PULMONARY INDICATIONS OF MV



GRAPH 11: DISTRIBUTION OF KLEBSIELLA PNEUMONIA AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY & NON-PULMONARY INDICATIONS OF MV

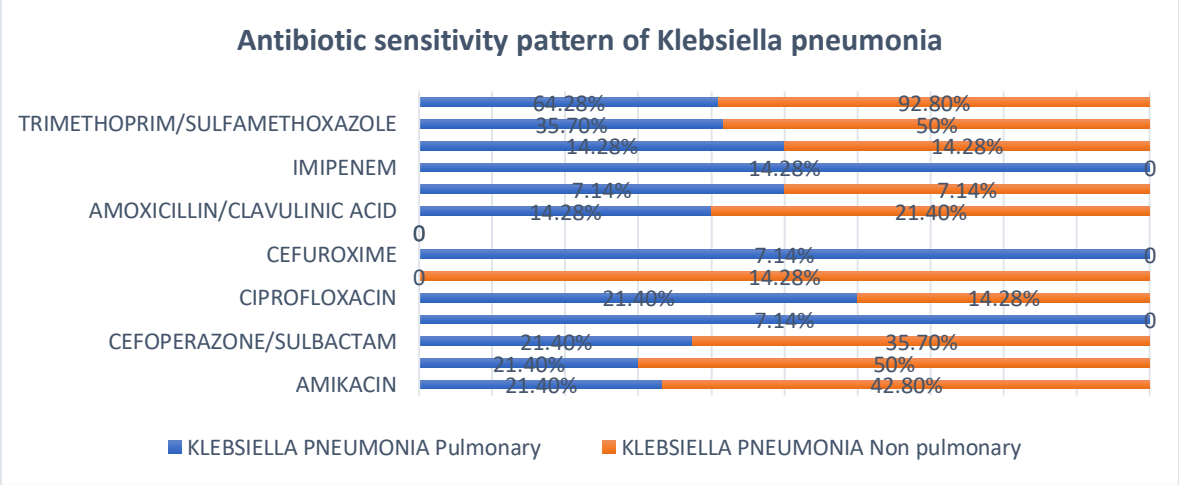
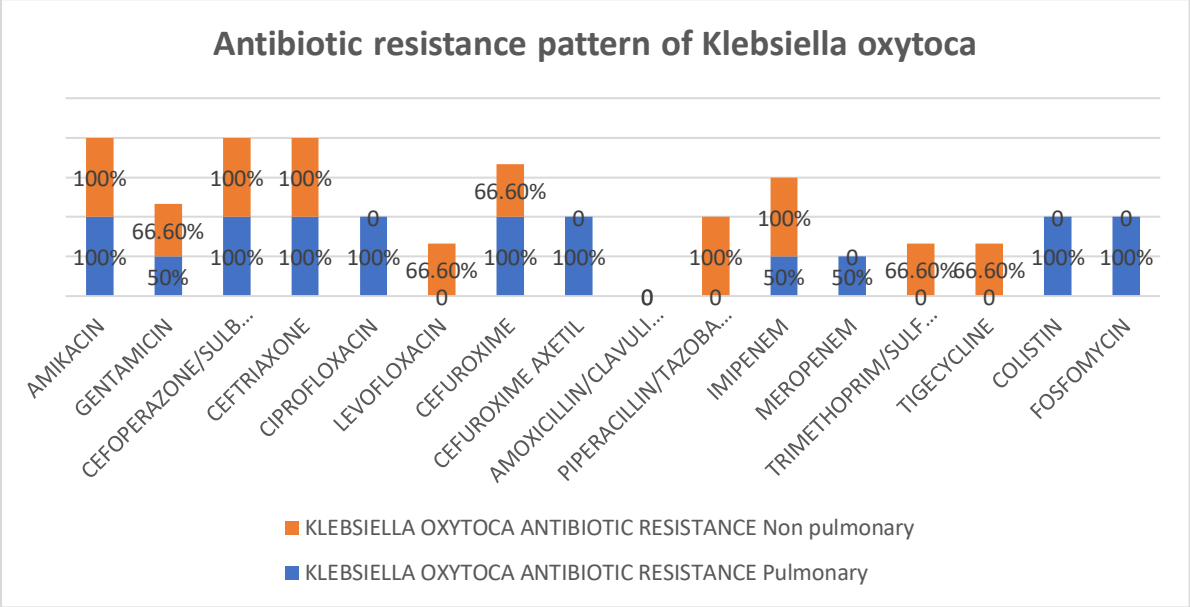


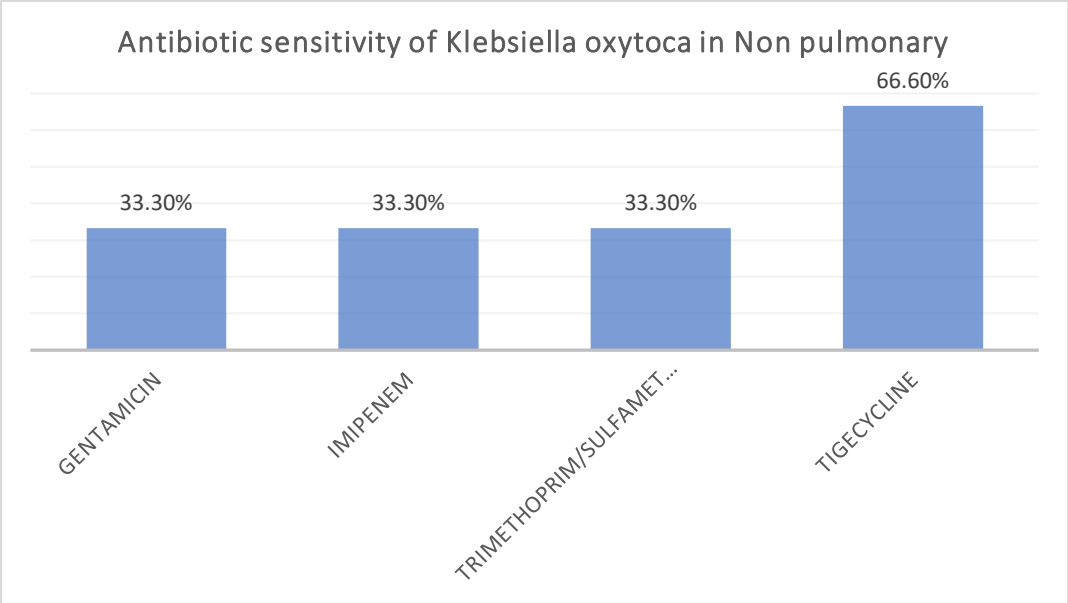
TABLE 20: DISTRIBUTION OF KLEBSIELLA OXYTOCA AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

KLEBSIELLA OXYTOCA	**Pulmonary**	**Non pulmonary**	**TOTAL**			**Chi square test p value**
	2	3	5			
	ANTIBIOTIC RESISTANCE			**ANTIBIOTIC SENSITIVITY**		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	

AMIKACIN	2(100%)	3(100%)	p=1.000	0	0	NA
GENTAMICIN	1(50%)	2(66.6%)	p=1.000	0	1(33.3%)	P=0.786
CEFOPERAZONE/SULBACTAM	2(100%)	3(100%)	p=1.000	0	0	NA
CEFTRIAXONE	2(100%)	3(100%)	p=1.000	0	0	NA
CIPROFLOXACIN	2(100%)	0	p=0.181	0	0	NA
LEVOFLOXACIN	0	2(66.6%)	p=0.176	0	0	NA
CEFUROXIME	2(100%)	2(66.6%)	p=1.000	0	0	NA
CEFUROXIME AXETIL	2(100%)	0	p=0.181	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	0	0	NA	0	0	NA
PIPERACILLIN/TAZOBACTAM	0	3(100%)	**P=0.045***	0	0	NA
IMIPENEM	1(50%)	3(100%)	P=0.220	0	1(33.3%)	P=0.786
MEROPENEM	1(50%)	0	P=0.786	0	0	NA
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	2(66.6%)	P=0.176	0	1(33.3%)	P=0.786
TIGECYCLINE	0	2(66.6%)	P=0.176	0	2(66.6%)	P=0.176
COLISTIN	2(100%)	0	p=0.181	0	0	NA
FOSFOMYCIN	2(100%)	0	p=0.181	0	0	A
*Statistically significant						



GRAPH 12: DISTRIBUTION OF KLEBSIELLA OXYTOCA AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS FOR MV



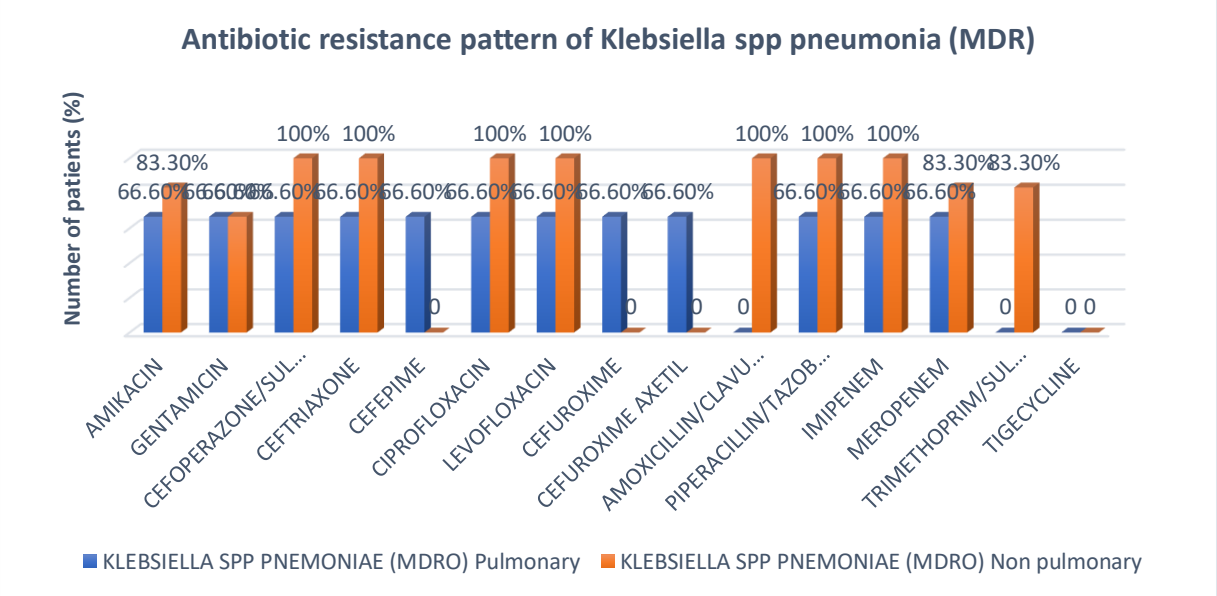
GRAPH 13: DISTRIBUTION OF KLEBSIELLA OXYTOCA AND ITS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS FOR MV

TABLE 21: DISTRIBUTION OF KLEBSIELLA SPP PNEUMONIAE (MDRO) AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

KLEBSIELLA SPP PNEMONIAE (MDRO)	Pulmonary	Non pulmonary		Total		Chi square test p value
	3	6		9		
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	2(66.6%)	5(83.3%)	P=0.156	3(100%)	1(16.6%)	**P=0.02***
GENTAMICIN	2(66.6%)	4(66.6%)	p=0.765	0	0	NA
CEFOPERAZONE/SULBACTAM	2(66.6%)	6(100%)	**p=0.033***	0	0	NA
CEFTRIAXONE	2(66.6%)	6(100%)	**p=0.033***	0	0	NA
CEFEPIME	2(66.6%)	0	p=0.181	0	0	NA
CIPROFLOXACIN	2(66.6%)	6(100%)	**p=0.033***	0	0	NA

LEVOFLOXACIN	2(66.6%)	6(100%)	**p=0.033***	0	0	NA
CEFUROXIME	2(66.6%)	0	p=0.181	0	0	NA
CEFUROXIME AXETIL	2(66.6%)	0	NA	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	0	6(100%)	**P=0.045 ***	0	0	NA
PIPERACILLIN/TAZOBACTAM	2(66.6%)	6(100%)	**p=0.033***	0	0	NA
IMIPENEM	2(66.6%)	6(100%)	**p=0.033***	0	0	NA
MEROPENEM	2(66.6%)	5(83.3%)	P=0.156	0	1(16.6%)	P=0.176
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	5(83.3%)	P=0.02*	0	1(16.6%)	P=0.176
TIGECYCLINE	0	0	NA	2(66.6%)	5(83.3%)	P=0.156
*Statistically significant						

GRAPH 14: DISTRIBUTION OF KLEBSIELLA SPP PNEUMONIAE (MDRO) AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY AND NON PULMONARY INDICATIONS OF MV



GRAPH 15: DISTRIBUTION OF KLEBSIELLA SPP PNEUMONIAE (MDRO) AND ITS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY AND NON PULMONARY INDICATIONS OF MV

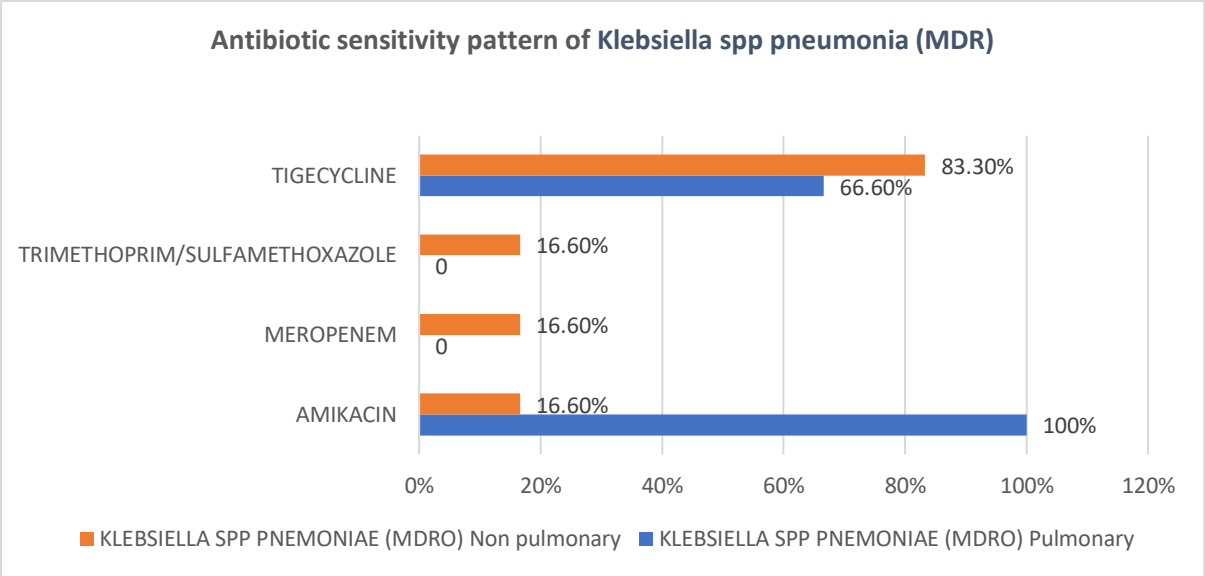


TABLE 22: DISTRIBUTION OF KLEBSIELLA AEROGENES AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

KLEBSIELLA AEROGENES	Pulmonary	Non pulmonary	Total			Chi square test	P value
	2	1	3				
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY			
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary		
AMIKACIN	0	0	NA	2(100%)	1(100%)	0.5556	P = 0.456
GENTAMICIN				2(100%)	1(100%)		
CEFOPERAZONE/SULBACTAM				2(100%)	1(100%)		
CEFTRIAXONE				2(100%)	1(100%)		
CEFEPIME				2(100%)	1(100%)		
CIPROFLOXACIN				2(100%)	1(100%)		
LEVOFLOXACIN				2(100%)	1(100%)		
PIPERACILLIN/TAZOBACTAM				2(100%)	1(100%)		
IMIPENEM				2(100%)	1(100%)		
MEROPENEM				2(100%)	1(100%)		
FOSFOMYCIN				2(100%)	1(100%)		
TIGECYCLINE				2(100%)	1(100%)		

GRAPH 16: DISTRIBUTION OF KLEBSIELLA AEROGENES AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY

AND NON-PULMONARY INDICATIONS OF MV

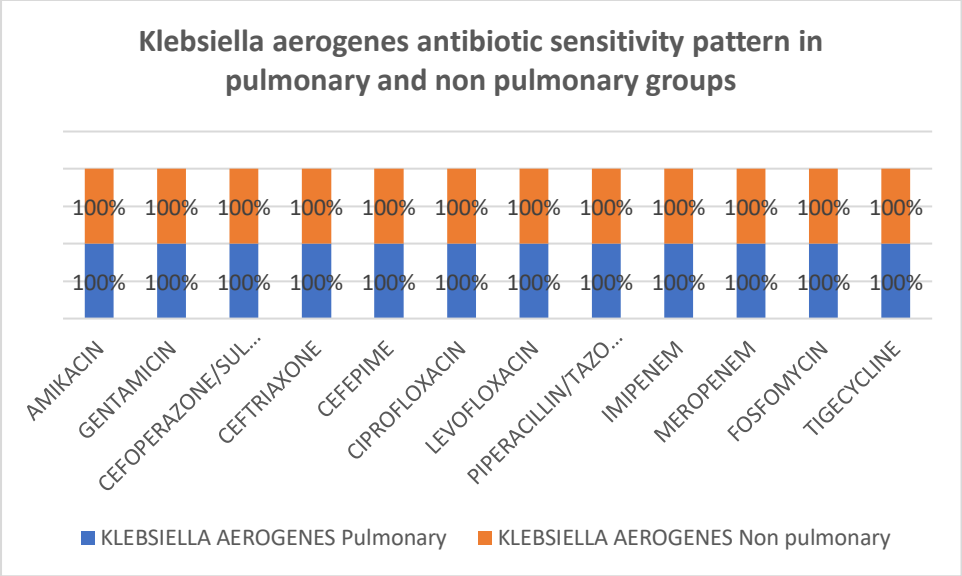
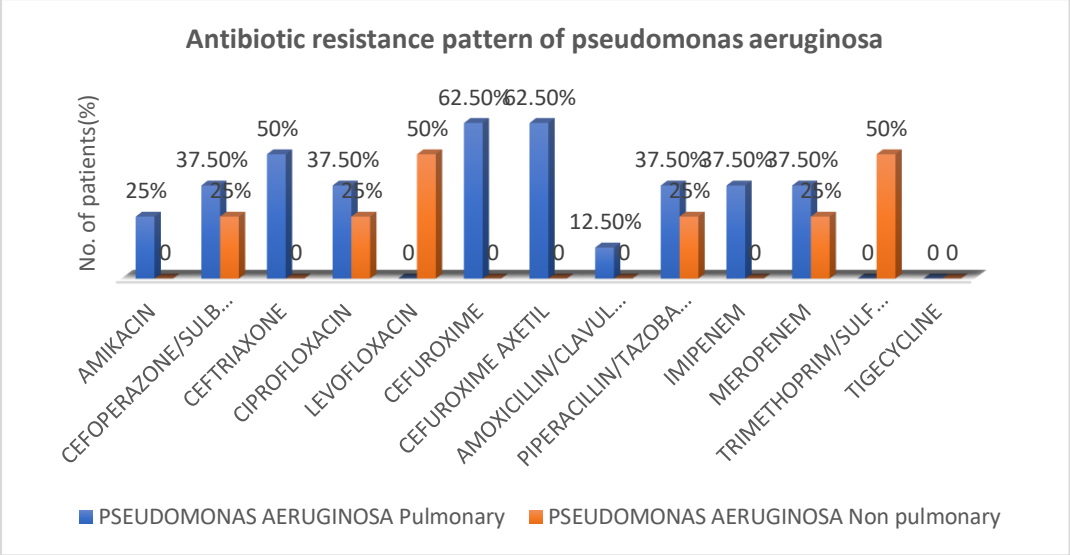


TABLE 23: DISTRIBUTION OF PSEUDOMONAS AERUGINOSA AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON PULMONARY INDICATIONS OF MV

PSEUDOMONAS AERUGINOSA	Pulmonary	Non pulmonary	Total			Chi square test p value
	8	4	12			
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	2(25%)	0	P=0.294	3(37.5%)	1(25%)	P=0.268
CEFOPERAZONE/SULBACTAM	3(37.5%)	1(25%)	P=0.268	5(62.5%)	3(75%)	P=0.342
CEFTRIAXONE	4(50%)	0	P=0.09	0	0	NA
CIPROFLOXACIN	3(37.5%)	1(25%)	P=0.268	0	1(25%)	P=0.06
LEVOFLOXACIN	0	2(50%)	**P=0.03***	0	0	NA
CEFUROXIME	5(62.5%)	0	**P=0.04***	0	0	NA
CEFUROXIME AXETIL	5(62.5%)	0	**P=0.04***	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	1(12.5%)	0	NA	0	0	NA
PIPERACILLIN/TAZOBACTAM	3(37.5%)	1(25%)	P=0.268	4(50%)	0	**P=0.05***
IMIPENEM	3(37.5%)	0	P=0.172	5(62.5%)	2(50%)	P=0.698
MEROPENEM	3(37.5%)	1(25%)	P=0.268	5(62.5%)	3(75%)	P=0.342
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	2(50%)	**P=0.03***	0	1(25%)	P=0.06
TIGECYCLINE	0	0	NA	2(25%)	2(50%)	P=0.08

*Statistically significant						

GRAPH 17: DISTRIBUTION OF PSEUDOMONAS AERUGINOSA AND ITS ANTIBIOTIC RESISTANCE PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV



GRAPH 18: DISTRIBUTION OF PSEUDOMONAS AERUGINOSA AND ITS ANTIBIOTIC SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

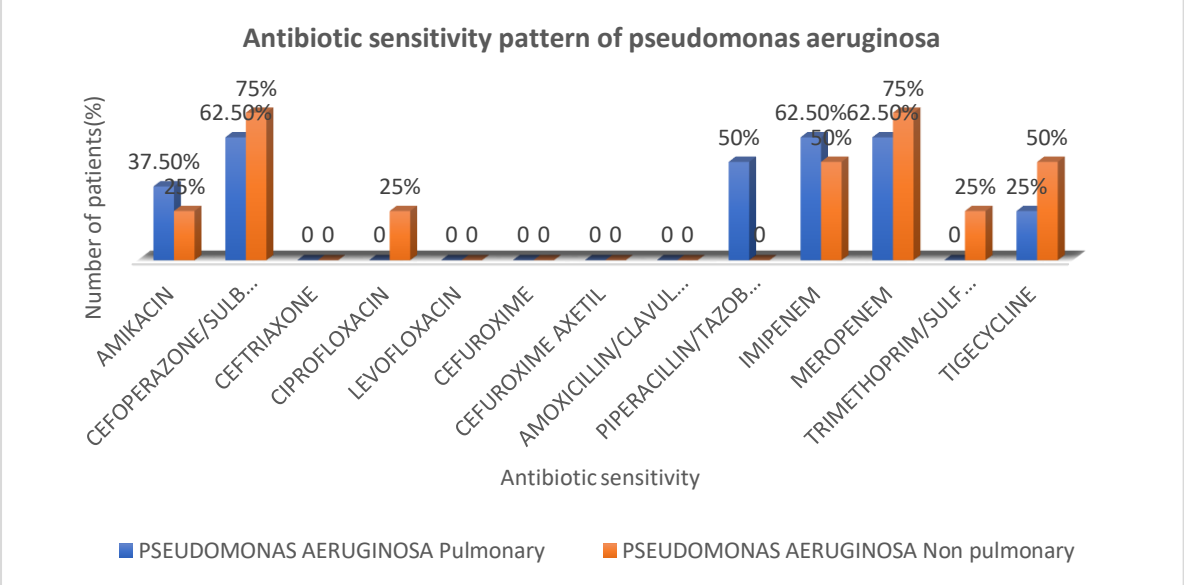
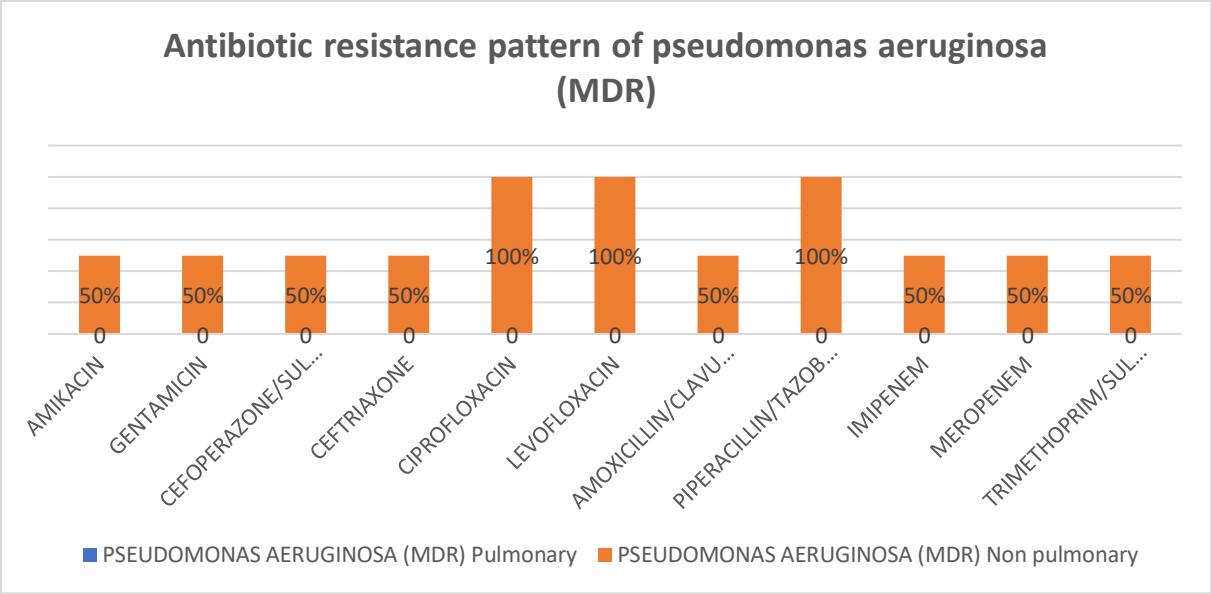


TABLE 24: DISTRIBUTION OF PSEUDOMONAS AERUGINOSA (MDR) AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

PSEUDOMONAS AERUGINOSA (MDR)	Pulmonary	Non pulmonary		Total		Chi square test p value
	0	2		2		
	ANTIBIOTIC RESISTANCE		Chi square test p value	ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	0	1(50%)	NA	0	1(50%)	NA
GENTAMICIN	0	1(50%)		0	0	
CEFOPERAZONE/SULBACTAM	0	1(50%)		0	1(50%)	
CEFTRIAXONE	0	1(50%)		0	0	
CEFEPIME	0	0		0	0	
CIPROFLOXACIN	0	2(100%)		0	0	
LEVOFLOXACIN	0	2(100%)		0	0	
CEFUROXIME	0	0		0	0	
CEFUROXIME AXETIL	0	0		0	0	
AMOXICILLIN/CLAVULINIC ACID	0	1(50%)		0	0	
PIPERACILLIN/TAZOBACTAM	0	2(100%)		0	0	
IMIPENEM	0	1(50%)		0	1(50%)	
MEROPENEM	0	1(50%)		0	1(50%)	
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	1(50%)		0	0	

GRAPH 19: DISTRIBUTION OF PSEUDOMONAS AERUGINOSA (MDR) AND ITS ANTIBIOTIC RESISTANCE PATTERN IN NON-PULMONARY INDICATIONS FOR MV



GRAPH 20: DISTRIBUTION OF PSEUDOMONAS AERUGINOSA (MDR) AND ITS ANTIBIOTIC SENSITIVITY PATTERN IN NON-PULMONARY INDICATIONS OF MV

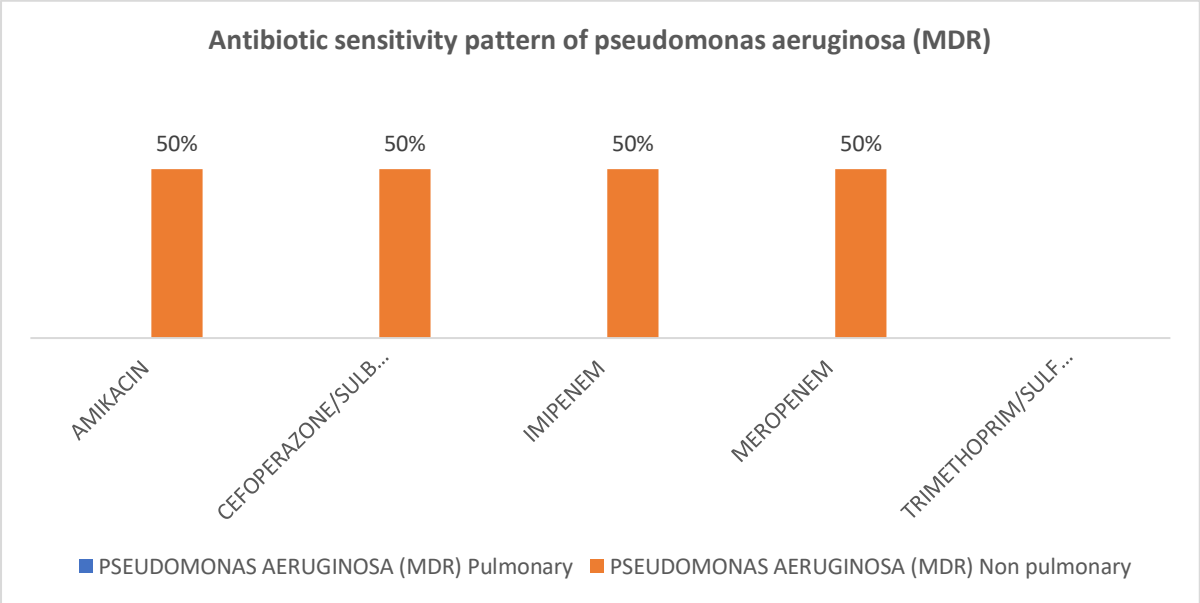
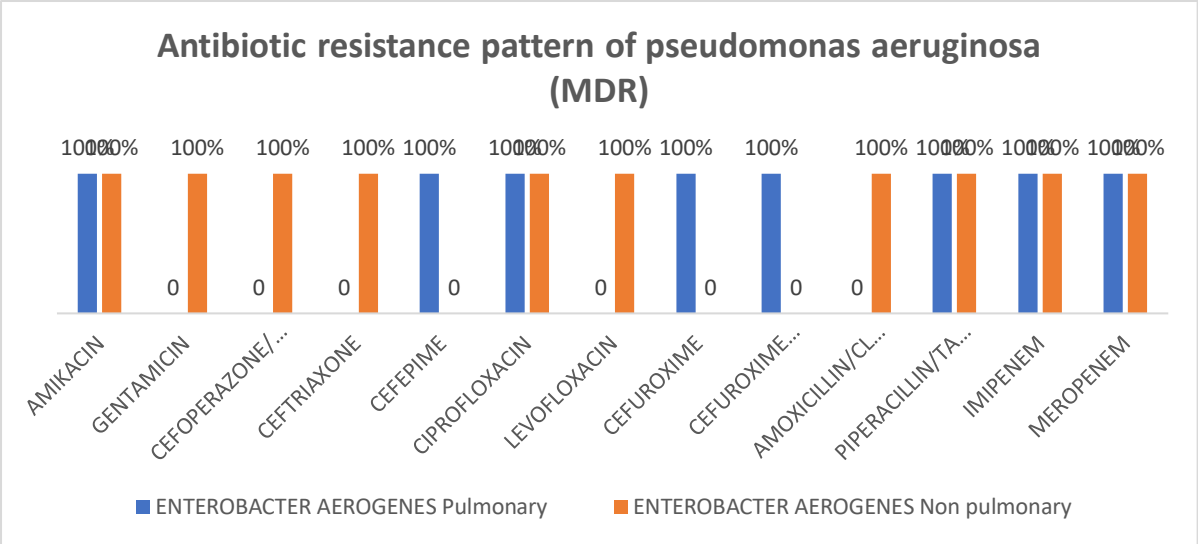


TABLE 25: DISTRIBUTION OF ENTEROBACTER AEROGENES AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV

ENTEROBACTER AEROGENES	Pulmonary	Non pulmonary	Total		Chi square test p value
	1	1	2		
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY	
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	1(100%)	1(100%)		0	0	
GENTAMICIN	0	1(100%)		1(100%)	0	
CEFOPERAZONE/SULBACTAM	0	1(100%)	NA	0	0	
CEFTRIAXONE	0	1(100%)		0	0	
CEFEPIME	1(100%)	0		0	0	
CIPROFLOXACIN	1(100%)	1(100%)		0	0	
LEVOFLOXACIN	0	1(100%)		0	0	NA
CEFUROXIME	1(100%)	0		0	0	
CEFUROXIME AXETIL	1(100%)	0		0	0	
AMOXICILLIN/CLAVULINIC ACID	0	1(100%)		0	0	
PIPERACILLIN/TAZOBACTAM	1(100%)	1(100%)		0	0	
IMIPENEM	1(100%)	1(100%)		0	0	
MEROPENEM	1(100%)	1(100%)		0	0	
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	0		1(100%)	1(100%)	
TIGECYCLINE	0	0		1(100%)	1(100%)	
ERTAPENEM	0	0		1(100%)	0	
FOSFOMYCIN	0	0		1(100%)	0	

GRAPH 21: DISTRIBUTION OF ENTEROBACTER AEROGENES AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS OF MV



GRAPH 22: DISTRIBUTION OF ENTEROBACTER AEROGENES AND ITS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS FOR MV

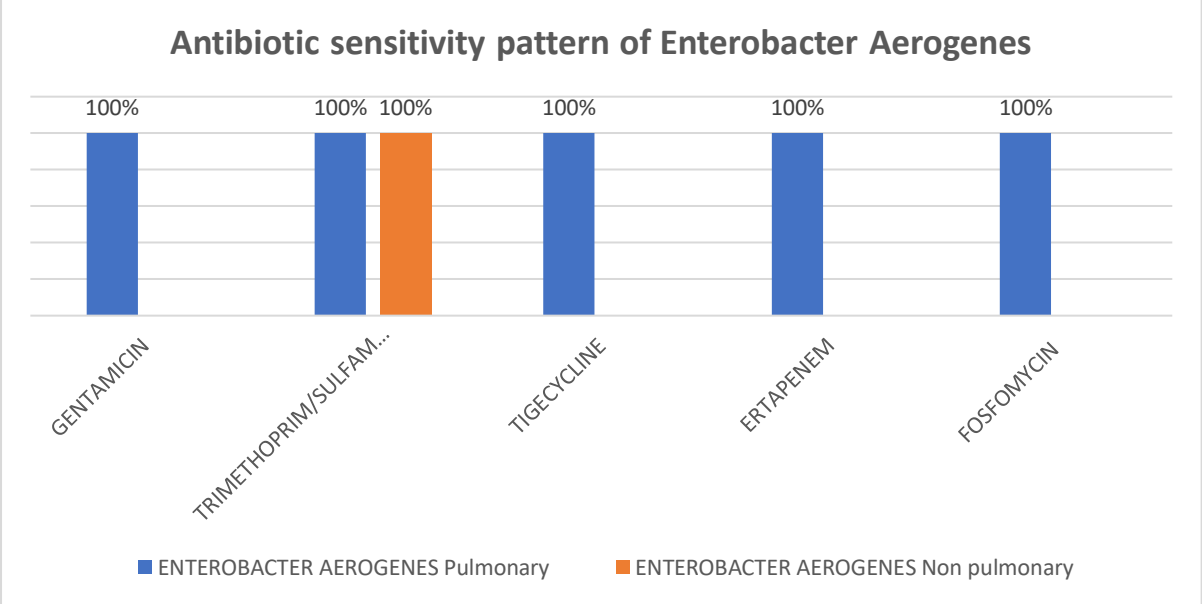
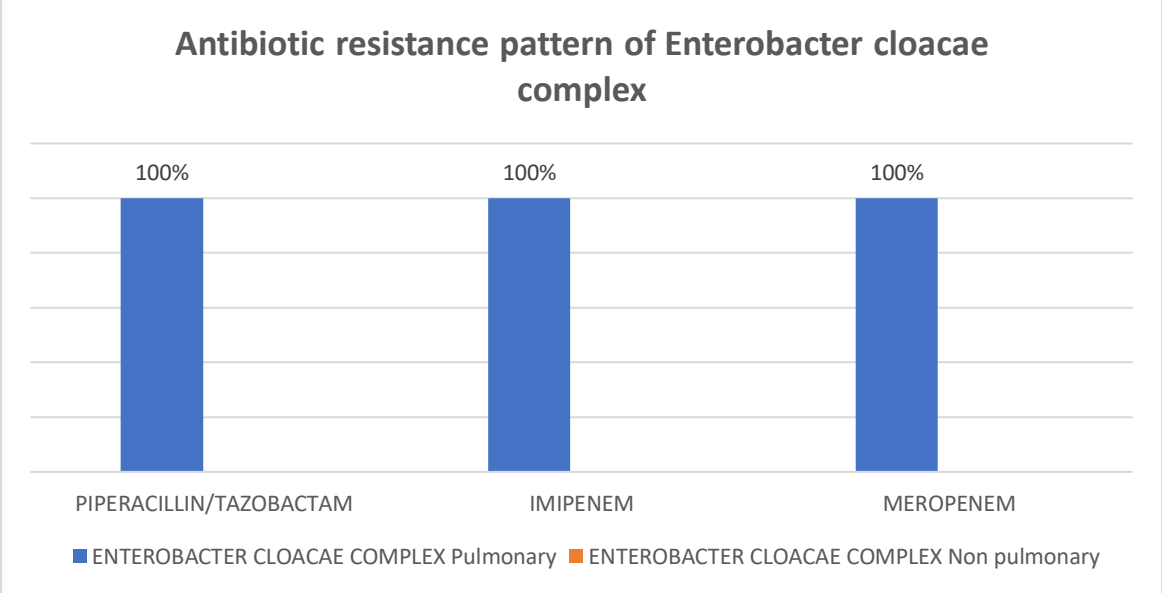


TABLE 26: DISTRIBUTION OF ENTEROBACTER CLOACAE COMPLEX AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY ANTIBIOTIC RESISTANCE PATTERN OF PSEUDOMONAS AERUGINOSA (MDR)

ENTEROBACTER CLOACAE COMPLEX	Pulmonary	Non pulmonary		Total		Chi square test p value

	1	0	1		
	ANTIBIOTIC RESISTANCE			**ANTIBIOTIC SENSITIVITY**		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	0	0		1(100%)	0	
GENTAMICIN	0	0		1(100%)	0	
CEFOPERAZONE/SULBACTAM	0	0		1(100%)	0	
CEFTRIAXONE	0	0		1(100%)	0	
CIPROFLOXACIN	0	0		1(100%)	0	
CEFUROXIME	1(100%)	0	NA	0	0	NA
CEFUROXIME AXETIL	1(100%)	0		0	0	
PIPERACILLIN/TAZOBACTAM	0	0		1(100%)	0	
IMIPENEM	0	0		1(100%)	0	
MEROPENEM	0	0		1(100%)	0	
COLISTIN	1(100%)	0		0	0	
FOSFOMYCIN	1(100%)	0		0	0	

GRAPH 23: DISTRIBUTION OF ENTEROBACTER CLOACAE COMPLEX AND ITS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY INDICATIONS OF VAP



GRAPH 24: DISTRIBUTION OF ENTEROBACTER CLOACAE COMPLEX AND ITS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY INDICATIONS OF VAP

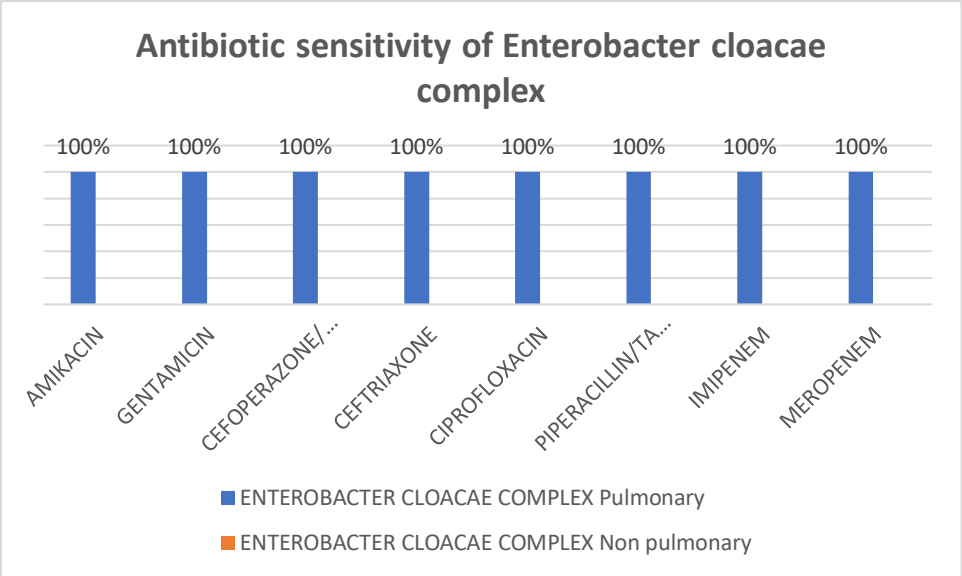
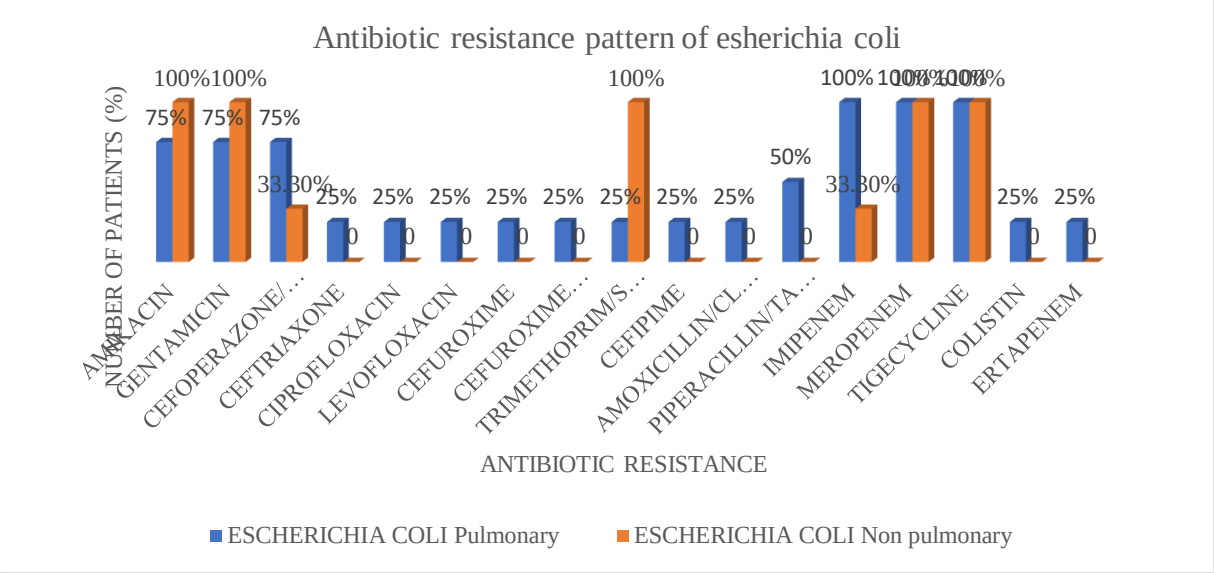


TABLE 27: DISTRIBUTION OF ESCHERICHIA COLI AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

ESCHERICHIA COLI	Pulmonary	Pon pulmonary		Total		Chi square test p value
	4	3		7		
	ANTIBIOTIC RESISTANCE			ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	0	0	NA	3(75%)	3(100%)	P=1.000
GENTAMICIN	0	0	NA	3(75%)	3(100%)	P=1.000
CEFOPERAZONE/SULBACTAM	0	2(66.6%)	P=0.0712	3(75%)	1(33.3%)	P=0.0967
CEFTRIAXONE	2(50%)	3(100%)	P=0.1797	1(25%)	0	P=0.3865
CIPROFLOXACIN	1(25%)	3(100%)	P=0.0612	1(25%)	0	P=0.3866
LEVOFLOXACIN	0	0	NA	1(25%)	0	P=0.3867
CEFUROXIME	2(50%)	0	P=0.0654	1(25%)	0	P=0.3868
CEFUROXIME AXETIL	2(50%)	0	P=0.0654	1(25%)	0	P=0.3869
TRIMETHOPRIM/SULFAMETHOXAZOLE	1(25%)	0	P=0.3865	1(25%)	3(100%)	P=0.0612
CEFIPIME	1(25%)	0	P=0.3865	1(25%)	0	P=0.3865
AMOXICILLIN/CLAVULINIC ACID	3(75%)	3(100%)	P=0.9876	1(25%)	0	P=0.3865
PIPERACILLIN/TAZOBACTAM	2(50%)	3(100%)	P=0.1797	2(50%)	0	P=0.0654
IMIPENEM	0	2(66.6%)	P=0.0712	4(100%)	1(33.3%)	P=0.0736
MEROPENEM	0	0	NA	4(100%)	3(100%)	P=0.9121
TIGECYCLINE	0	0	NA	2(100%)	3(100%)	P=0.1797
COLISTIN	0	0	NA	1(25%)	0	P=0.3869

ERTAPENEM	0	0	NA	1(25%)	0	P=0.3870
FOSFOMYCIN	0	0	NA	1(25%)	0	P=0.3871

GRAPH 25: DISTRIBUTION OF ESCHERICHIA COLI AND ITS ANTIBIOTIC RESISTANCE PATTERNSIN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP



GRAPH 26: DISTRIBUTION OF ESCHERICHIA COLI AND ITS ANTIBIOTIC SENSITIVITY PATTERNSIN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

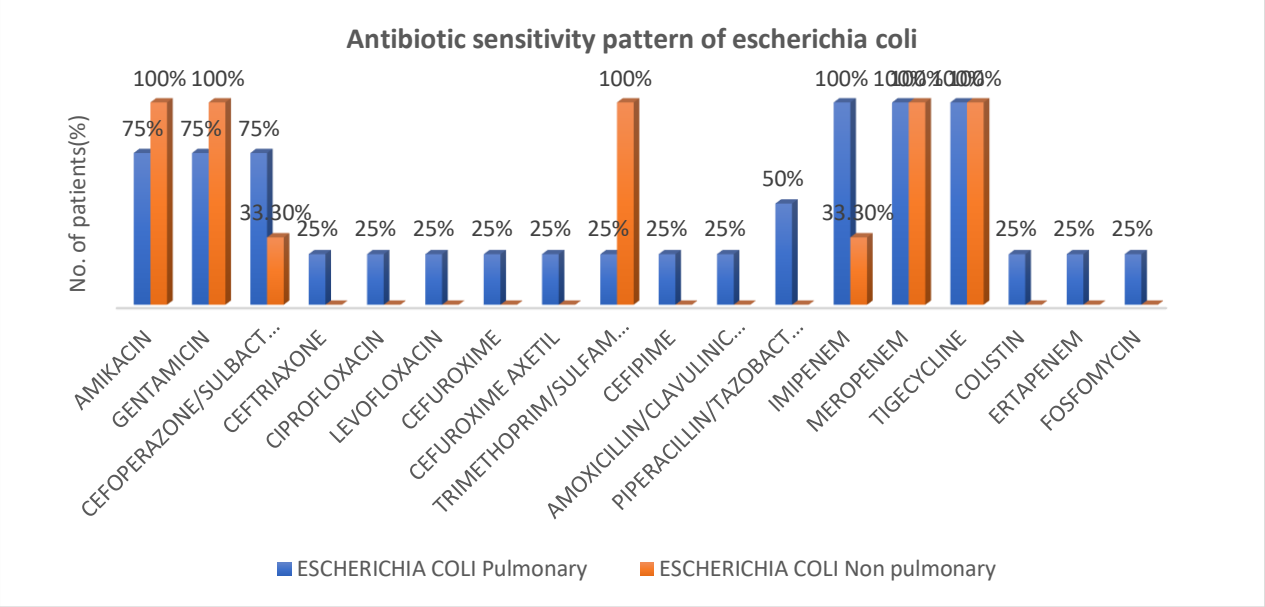


TABLE 28: DISTRIBUTION OF SERRATIA MARCESCENS AND ITS ANTIBIOTIC

RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

SERRATIA MARCESCENS	PULMONARY	NON PULMONARY	TOTAL	
	5	0	5	
	ANTIBIOTIC RESISTANCE		ANTIBIOTIC SENSITIVITY	
	Pulmonary	Non pulmonary	Pulmonary	Non pulmonary
AMIKACIN	0	0	4(80%)	0
GENTAMICIN	1(20%)	0	1(20%)	0
CEFOPERAZONE/SULBACTAM	4(80%)	0	1(20%)	0
CEFTRIAXONE	4(80%)	0	0	0
CIPROFLOXACIN	1(20%)	0	0	0
CEFUROXIME	5(100%)	0	0	0
CEFUROXIME AXETIL	5(100%)	0	NA 0	0
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	0	5(100%)	0
CEFIPIME	4(80%)	0	0	0
AMOXICILLIN/CLAVULINIC ACID	5(100%)	0	0	0
IMIPENEM	4(80%)	0	1(20%)	0
MEROPENEM	4(80%)	0	1(20%)	0
TIGECYCLINE	0	0	5(100%)	0
ERTAPENEM	4(80%)	0	1(20%)	0

GRAPH 27: DISTRIBUTION OF ESCHERICHIA COLI AND ITS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY INDICATIONS OF VAP

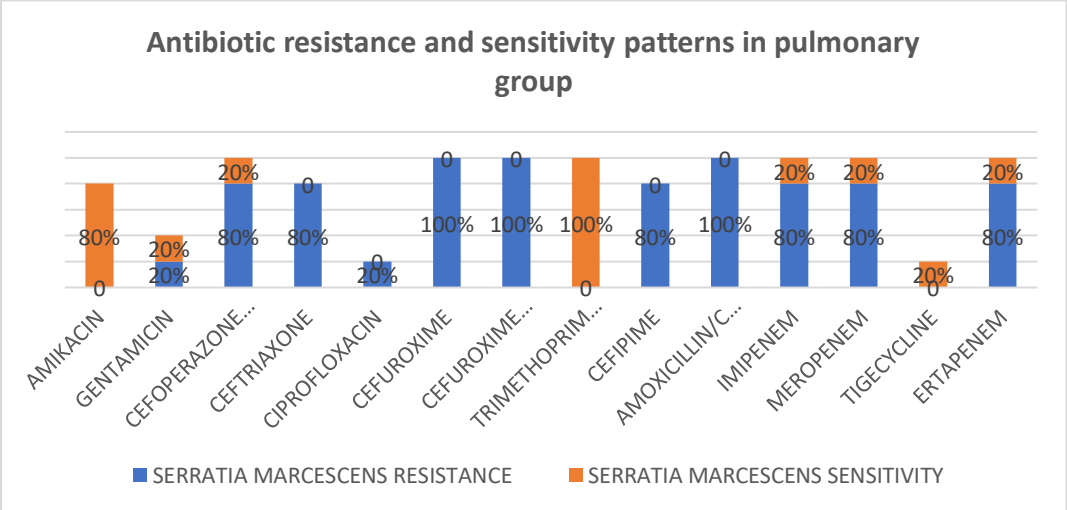
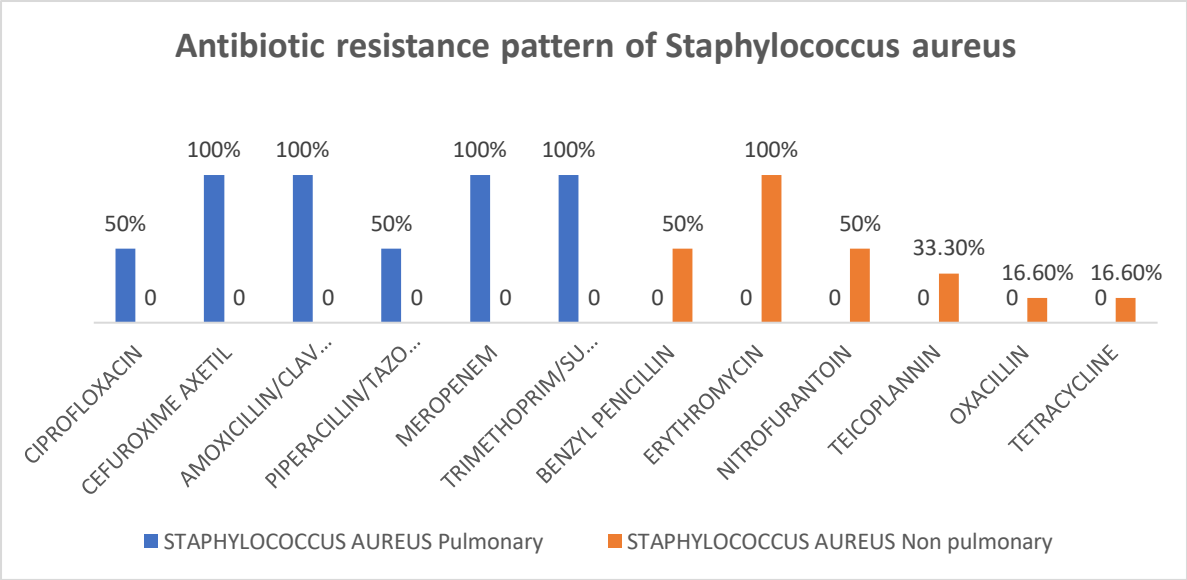


TABLE 29: DISTRIBUTION OF AND ITS STAPHYLOCOCCUS AUREUS ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY

AND NON-PULMONARY INDICATIONS OF VAP

STAPHYLOCOCCUS AUREUS	Pulmonary	Non pulmonary	Total			Chi square test p value
	4	6	10			
	ANTIBIOTIC RESISTANCE		ANTIBIOTIC SENSITIVITY			
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
CEFOPERAZONE/SULBACTAM	0	0	NA	0	4(66.6%)	**P=0.0455***
CEFTRIAXONE	0	0	NA	2(50%)	0	P=0.0662
CIPROFLOXACIN	2(50%)	0	P=0.0662	2(50%)	0	P=0.0662
LEVOFLOXACIN	0	0	NA	0	6(100%)	**P=0.0027***
CEFUROXIME	0	0	NA	0	0	NA
CEFUROXIME AXETIL	4(100%)	0	**P=0.0027***	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	4(100%)	0	**P=0.0027***	0	0	NA
PIPERACILLIN/TAZOBACTAM	2(50%)	0	P=0.0662	0	0	NA
IMIPENEM	0	0	NA	0	0	NA
MEROPENEM	4(100%)	0	**P=0.0027***	0	0	NA
TRIMETHOPRIM/SULFAMETHOXAZOLE	4(100%)	0	**P=0.0027***	0	0	NA
ERTAPENEM	0	0	NA	2(50%)	5(83.3%)	P=0.0843
TIGECYCLINE	0	0	NA	2(50%)	0	P=0.0662
CLINDAMYCIN	0	0	NA	2(50%)	6(100%)	P=0.0662
BENZYL PENICILLIN	0	3(50%)	P=0.1088	0	0	NA
ERYTHROMYCIN	0	6(100%)	**P=0.0027***	0	0	NA
NITROFURANTOIN	0	3(50%)	P=0.1088	0	0	NA
TEICOPLANNIN	0	2(33.3%)	P=0.0812	0	0	NA
OXACILLIN	0	1(16.6%)	P=0.4142	0	0	NA
TETRACYCLINE	0	1(16.6%)	P=0.4142	0	0	NA
*Statistically significant						

GRAPH 28: DISTRIBUTION OF AND ITS STAPHYLOCOCCUS AUREUS ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP



GRAPH 29: DISTRIBUTION OF AND ITS STAPHYLOCOCCUS AUREUS ANTIBIOTIC SENSITIVITY PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

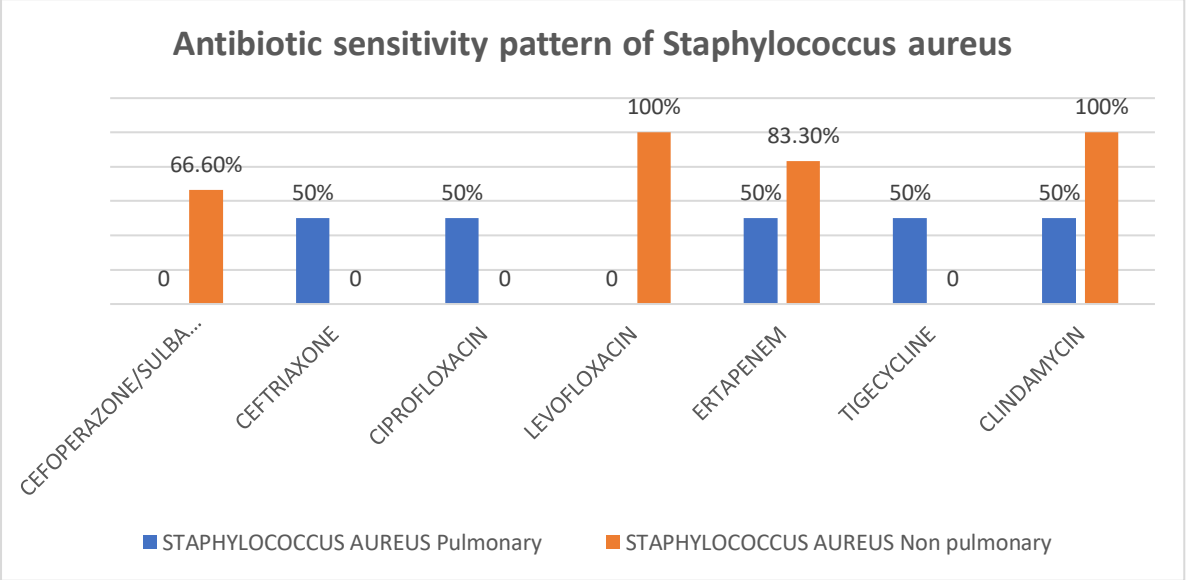
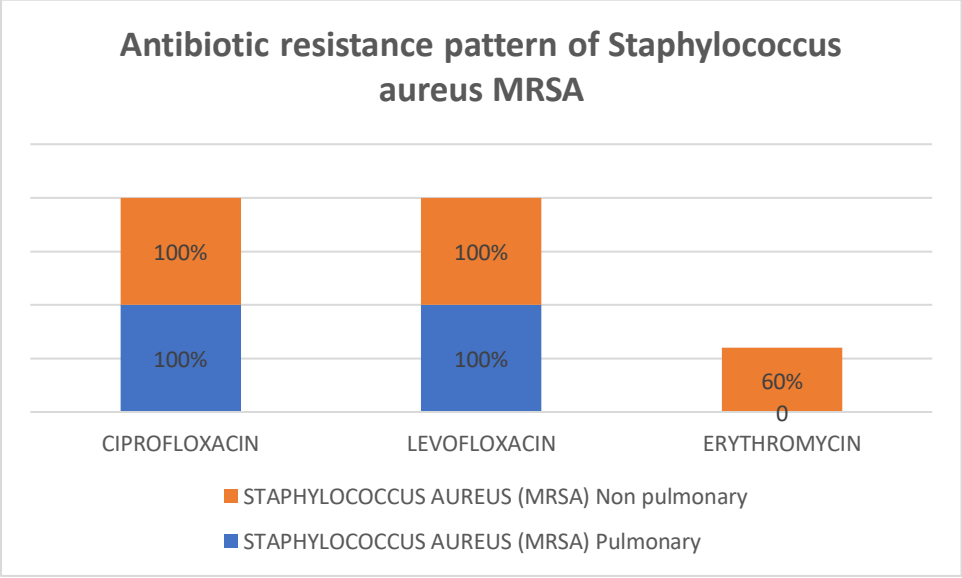


TABLE 30: DISTRIBUTION OF AND ITS STAPHYLOCOCCUS AUREUS (MRSA) ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

STAPHYLOCOCCUS AUREUS (MRSA)	Pulmonary	Non pulmonary	Total			Chi square test p value
	1	5	6			

	ANTIBIOTIC RESISTANCE		ANTIBIOTIC SENSITIVITY			
	Pulmonary	**Non pulmonary**		**Pulmonary**	**Non pulmonary**	
AMIKACIN	0	0	NA	0	2(40%)	P=0.5271
GENTAMICIN	0	0	NA	0	2(40%)	P=0.5271
CEFOPERAZONE/SULBACTAM	0	0	NA	1(100%)	2(40%)	P=0.2431
CIPROFLOXACIN	1(100%)	5(100%)	P=0.0463*	0	0	P=0.0143*
LEVOFLOXACIN	1(100%)	5(100%)	P=0.0463*	0	0	NA
ERYTHROMYCIN	0	3(60%)	P=0.1237	0	0	NA
AMOXICILLIN/CLAVULINIC ACID	0	0	NA	0	2(40%)	P=0.5271
PIPERACILLIN/TAZOBACTAM	0	0	NA	0	2(40%)	P=0.5272
IMIPENEM	0	0	NA	0	2(40%)	P=0.5273
MEROPENEM	0	0	NA	0	2(40%)	P=0.5274
TRIMETHOPRIM/SULFAMETHOXAZOLE	0	0	NA	1(100%)	5(100%)	P=0.0463*
TIGECYCLINE	0	0	NA	1(100%)	5(100%)	P=0.0463*
VANCOMYCIN	0	0	NA	1(100%)	3(60%)	P=0.6213
LINEZOLID	0	0	NA	1(100%)	3(60%)	P=0.6214
TETRACYCLINE	0	0	NA	1(100%)	3(60%)	P=0.6215
*Statistically significant						

GRAPH 30: DISTRIBUTION OF AND ITS STAPHYLOCOCCUS AUREUS (MRSA) ANTIBIOTIC RESISTANCE PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP



GRAPH 31: DISTRIBUTION OF AND ITS STAPHYLOCOCCUS AUREUS (MRSA) ANTIBIOTIC SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

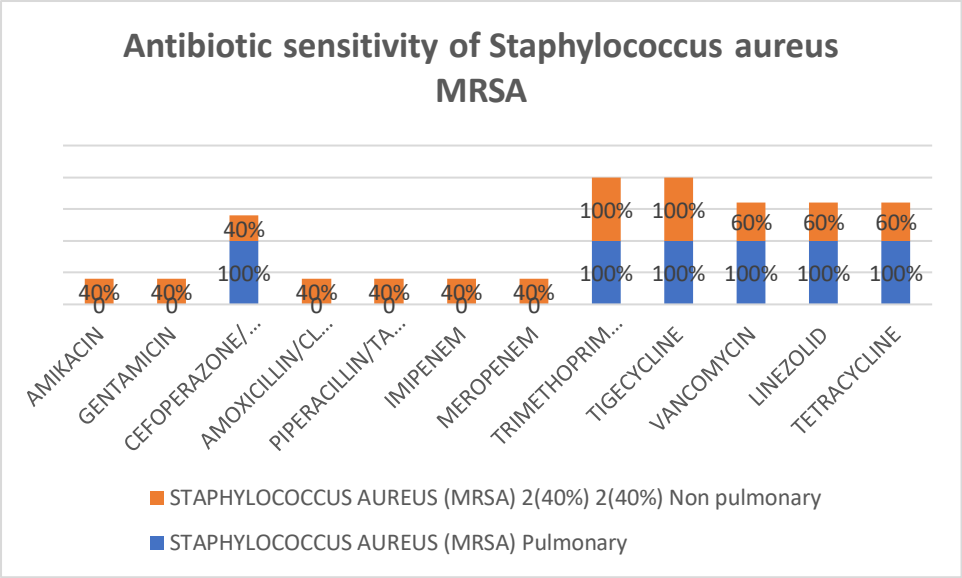


TABLE 31: DISTRIBUTION OF AND ITS STREPTOCOCCUS PNEUMONIAE ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

STREPTOCOCCUS PNEUMONIAE	PULMONARY	NON PULMONARY	TOTAL			chisquare test p value
	3	0	3			
	ANTIBIOTIC RESISTANCE		**ANTIBIOTIC SENSITIVITY**			
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	0	0	NA	3(100%)	0	NA
CEFOPERAZONE/SULBACTAM	0			3(100%)		
CEFTRIAXONE	3(100%)			0		
CIPROFLOXACIN	0			3(100%)		
CEFEPIME	0			3(100%)		
CEFUROXIME	3(100%)			0		
CEFUROXIME AXETIL	3(100%)			0		
AMOXICILLIN/CLAVULINIC ACID	0			3(100%)		
PIPERACILLIN/TAZOBACTAM	0			3(100%)		
MEROPENEM	3(100%)			0		
TRIMETHOPRIM/SULFAMETHOXAZOLE	0			3(100%)		
ERTAPENEM	0			3(100%)		

GRAPH 32: DISTRIBUTION OF AND ITS STREPTOCOCCUS PNEUMONIAE ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY INDICATIONS OF VAP

Streptococcus pneumonia is only isolated in 3 pulmonary cases while no cases are isolated in

Non pulmonary group.

These isolates showed 100% resistance to Ceftriaxone, Cefuroxime, Cefuroxime axetil and Meropenem. Amikacin, Cefoperazone/Sulbactum, Ciprofloxacin, Cefepime, beta lactams, trimethoprim/Sulfamethoxazole and Ertapenem showed 100% sensitivity.

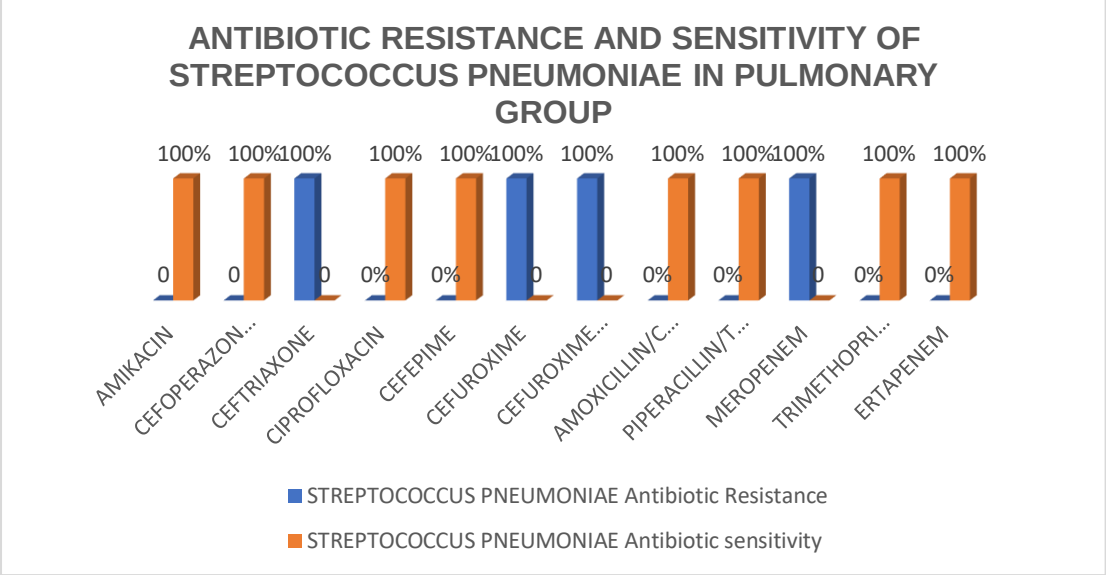


TABLE 32: DISTRIBUTION OF AND ITS ESCHERICHIA COLI (CRE) ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

ESCHERICHIA COLI (CRE)	Pulmonary	Non pulmonary	Total			Chi square test p value
	0	1	1			
	ANTIBIOTIC RESISTANCE		ANTIBIOTIC SENSITIVITY			
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary	
AMIKACIN	0	1(100%)	NA	0	0	NA
CEFTRIAXONE		1(100%)			0	
CIPROFLOXACIN		1(100%)			0	
LEVOFLOXACIN		1(100%)			0	
IMIPENEM		1(100%)			0	
PIPERACILLIN/TAZOBACTAM		1(100%)			0	
MEROPENEM		1(100%)			0	
TRIMETHOPRIM/SULFAMETHOXAZOLE		1(100%)			0	
TIGECYCLINE		0			1(100%)	

GRAPH 33: DISTRIBUTION OF AND ITS ESCHERICHIA COLI (CRE) ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS NON-PULMONARY INDICATIONS OF VAP

Escherichia Coli (CRE) is only seen in Non pulmonary group in 1 case (1.58%)

Tigecycline is the only sensitive antibiotic (100%) where as Amikacin, Ceftriaxone, Ciprofloxacin, Levofloxacin, Imipenem, Piperacillin/Tazobactam, Meropenem, Trimethoprim/Sulfamethoxazole are 100% resistant.

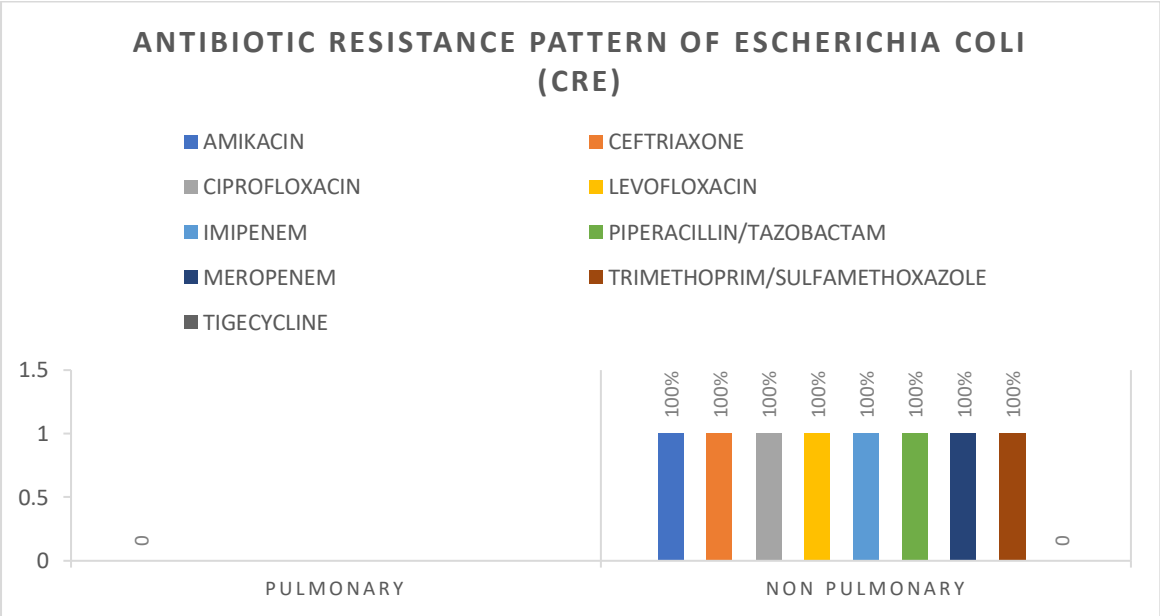


TABLE 33: DISTRIBUTION OF AND ITS CITROBACTER FREUNDII ANTIBIOTIC RESISTANCE AND SENSITIVITY PATTERNS IN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

Citrobacter Freundii is only seen in Non-pulmonary group in 1 case (1.58%)

CITROBACTER FREUNDII	Pulmonary	Non pulmonary	Total		P value
	0	1	1		
	ANTIBIOTIC RESISTANCE		ANTIBIOTIC SENSITIVITY		
	Pulmonary	Non pulmonary		Pulmonary	Non pulmonary
AMIKACIN	0	1(100%)	NA	0	0	NA
GENTAMICIN		1(100%)			0	
CEFTRIAXONE		1(100%)			0	
CIPROFLOXACIN		1(100%)			0	
LEVOFLOXACIN		1(100%)			0	
PIPERACILLIN/TAZOBACTAM		1(100%)			0	
MEROPENEM		1(100%)			0	

TRIMETHOPRIM/SULFAMETHOXAZOLE		1(100%)			0	
AMOXYCILLIN/CLAVULINIC ACID		0			1(100%)	

In Non-pulmonary group, Amoxicillin clavulanic acid is the only sensitive antibiotic (100%) for Citrobacter freundii.

GRAPH 34: DISTRIBUTION OF AND ITS CITROBACTER FREUNDII ANTIBIOTIC RESISTANCE PATTERN IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

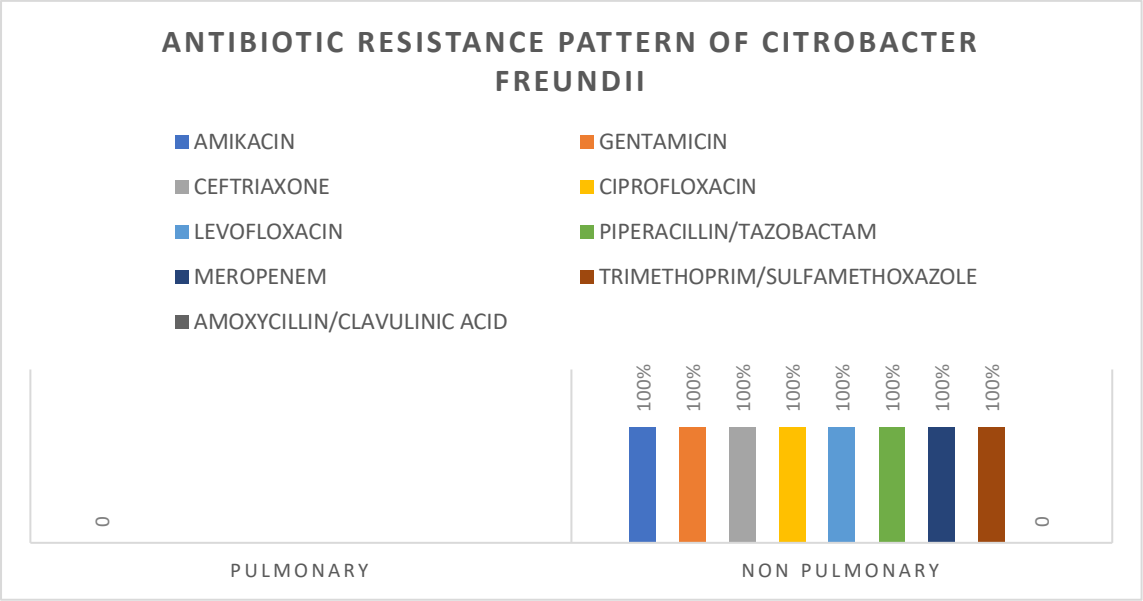


TABLE 34: COMPARISON OF OUTCOME OF VAP IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

OUTCOME	Pulmonary	Non pulmonary	Total	Chi square test	P value
DAMA DUE TO FAMILY ISSUES	0	10(15.9%)	10(7.9%)	9.2466	P=0.0024*
DAMA DUE TO FINANCIAL ISSUES	6(9.5%)	14(22.2%)	20(15.9%)	3.7736	P=0.05*
DEATH DUE TO CP ARREST	15(23.8%)	2(3.2%)	17(13.5%)	11.6548	P=0.0002*

DEATH DUE TO SEPSIS	4(6.3%)	11(17.5%)	15(11.9%)	3.6787	P=0.05*
IMPROVED	35(55.6%)	24(38.1%)	59(46.8%)	3.8262	P=0.05*
REFERRED TO HIGHER CENTRE	3(4.8%)	2(3.2%)	5(4%)	0.2066	P=0.6494
TOTAL	63(100%)	63(100%)	126(100%)		

TABLE 35: DISTRIBUTION OF MORTALITY OF VAP AMONG PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

	Pulmonary n=63 %	**Non pulmonary n=63 %**	**Total n %**	**Odds Ratio (OR)**
Death	19(30.1%)	13(20.6%)	32(25.3%)	
Improved	35(55.5%)	24(38%)	59(46.8%)	1.002

GRAPH 35: DISTRIBUTION OF MORTALITY OF VAP AMONG PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

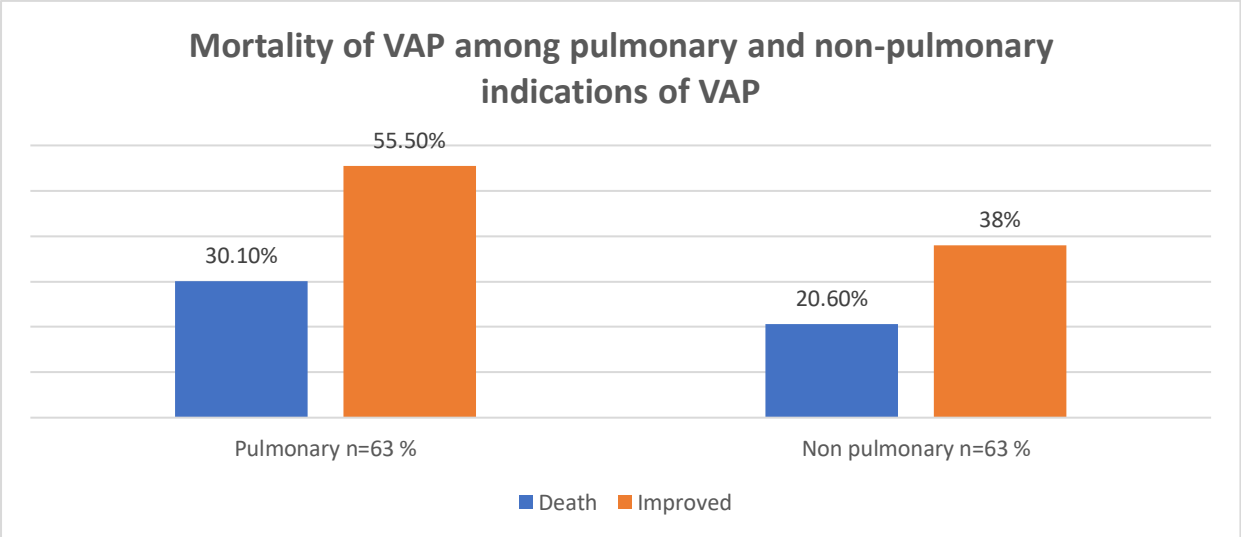
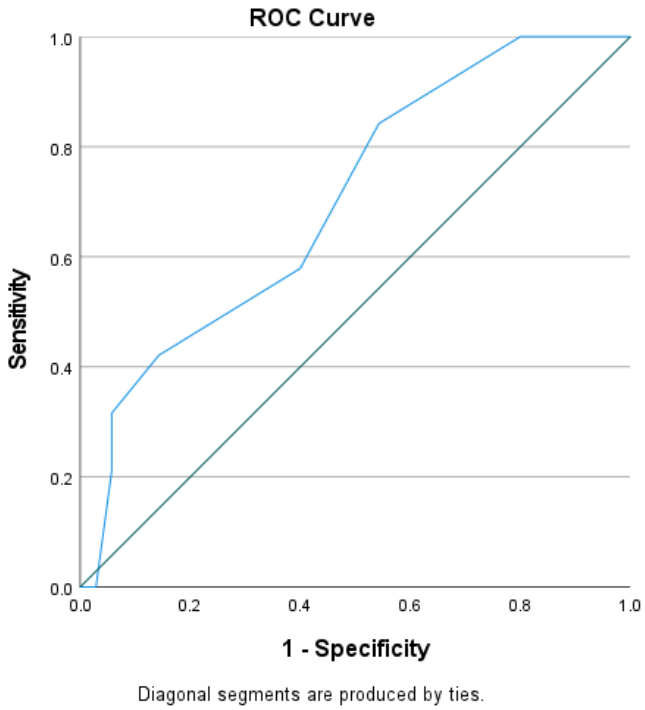


FIGURE 6: PREDICTIVE OUTCOME OF MODIFIED CPIS SCORE AMONG VAP IN PULMONARY INDICATIONS OF VAP



When analyzing the ROC curve, we found an AUC of 0.702. We observed that a CPIS above 4 is associated with a sensitivity of 84% and a specificity of 46% in predicting the outcome of VAP in pulmonary cases.

AREA UNDER THE CURVE				
TEST RESULT VARIABLES: SCORES				
Area	**Std. Error [a]**	**Asymptotic Sig.[b]**	**Asymptotic 95% Confidence Interval**	
			Upper bond	Lower bond
0.702	**0.073**	**0.015**	**0.559**	**0.844**
a. Under the nonparametric assumption				
b. Null hypothesis: true area = 0.5				

- Sensitivity: 84% (0.84)
- Specificity: 46% (0.46)

At the point of highest sensitivity and specificity, cutoff value was taken as 4 for modified CPIS score in this study.

TABLE 36: DISTRIBUTION OF DIAGNOSIS OF VAP AND PREDICTIVE OUTCOME OF MODIFIED CPIS SCORE IN PULMONARY INDICATIONS OF VAP

MODIFIED CPIS SCORE IN PULMPONARY GROUP	PREDICTIVE OUTCOME		
	Death n %	**Improved n %**	**Total**
≤4	3 (15.8%)	16 (45.7%)	19 (35.2%)
>4	16 (84.2%)	19 (54.3%)	35 (64.8%)
TOTAL	19 (100%)	35(100%)	54(100%)

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	4.836[a]	1	0.028

GRAPH 36: DISTRIBUTION OF PREDICTIVE OUTCOME OF MODIFIED CPIS SCORE IN PULMONARY INDICATIONS OF INDICATIONS OF VAP

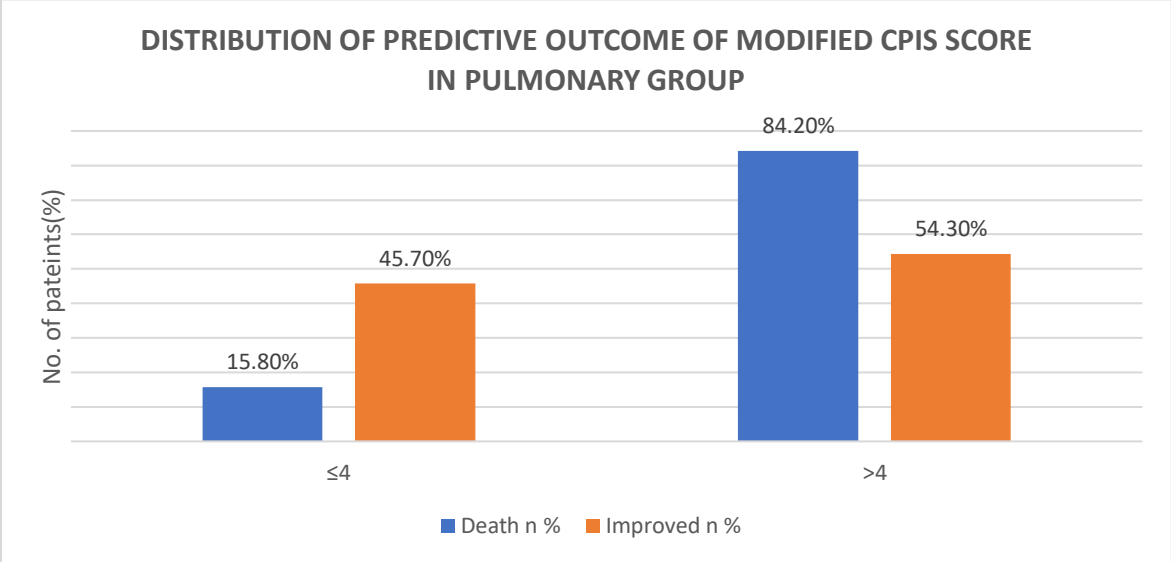
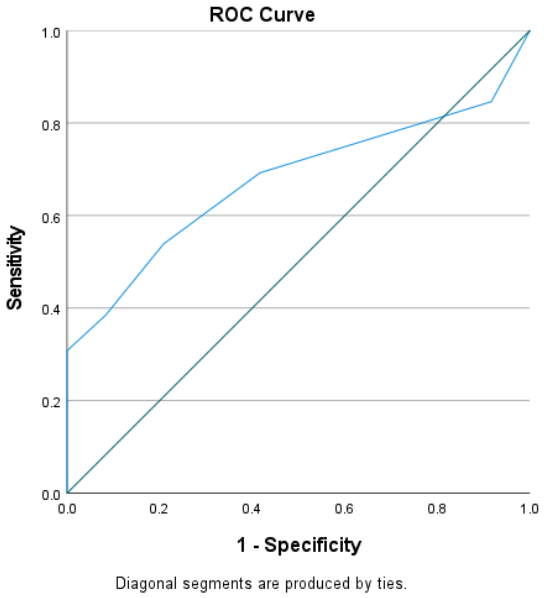


FIGURE 7: PREDICTIVE OUTCOME OF MODIFIED CPIS SCORE AMONG VAP IN NON-PULMONARY INDICATIONS OF VAP



When analyzing the ROC curve, we found an AUC of 0.676. We observed that a CPIS above 6 is associated with a sensitivity of 84% and a specificity of 46% in predicting the outcome of VAP in pulmonary cases.

AREA UNDER THE CURVE				
TEST RESULT VARIABLES: SCORES				
Area	**Std. Error[a]**	**Asymptotic Sig.[b]**	**Asymptotic 95% Confidence Interval**	
			Upper bond	Lower bond
0.676	0.105	0.08	0.47	0.883
a. Under the nonparametric assumption				
b. Null hypothesis: true area = 0.5				

- Sensitivity: 54% (0.54)
- Specificity: 79% (0.79)

At the point of highest sensitivity and specificity, cutoff value was taken as 6 for modified CPIS score in this study.

TABLE 37: DISTRIBUTION OF DIAGNOSIS OF VAP AND PREDICTIVE OUTCOME OF MODIFIED CPIS SCORE IN NON-PULMONARY GROUP

MODIFIED CPIS SCORE IN NON-PULMONARY GROUP	No. of VAP cases n %	PREDICTIVE OUTCOME	

		Death n %	Improved n %	TOTAL
≤6	25 (67.6%)	6 (46.2%)	19 (79.2%)	25 (67.6%)
>6	12 (32.4%)	7 (53.8%)	5 (20.8%)	12 (32.4%)
TOTAL	37 (100%)	13 (100%)	24(100%)	37(100%)

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	4.194[a]	1	.041

GRAPH 37: DISTRIBUTION OF PREDICTIVE OUTCOME OF MODIFIED CPIS SCORE IN NON-PULMONARY INDICATIONS OF VAP

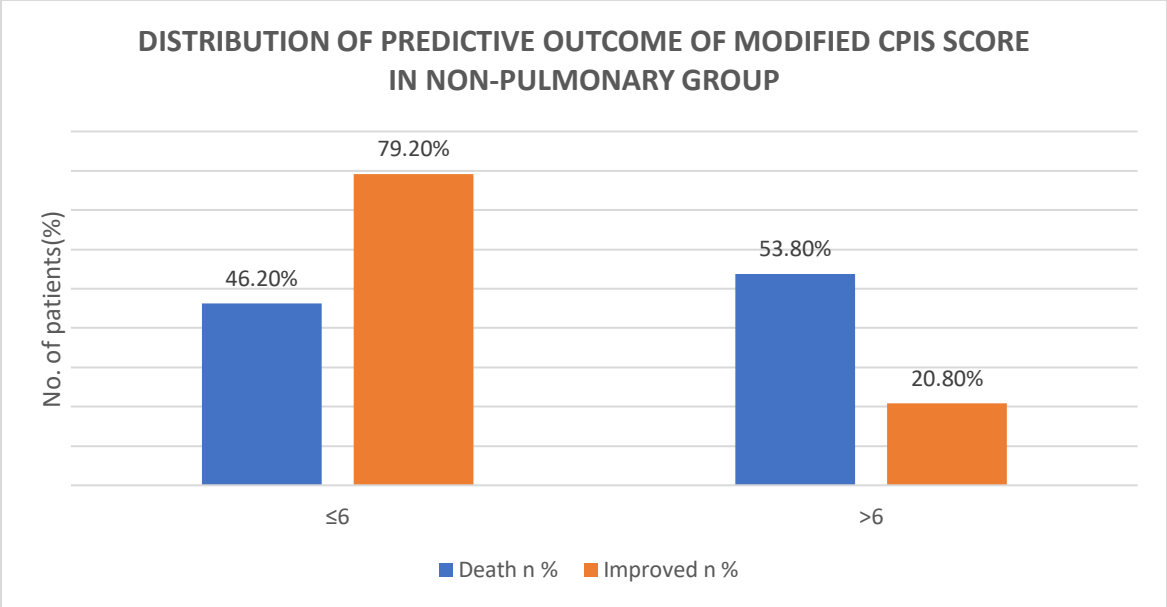
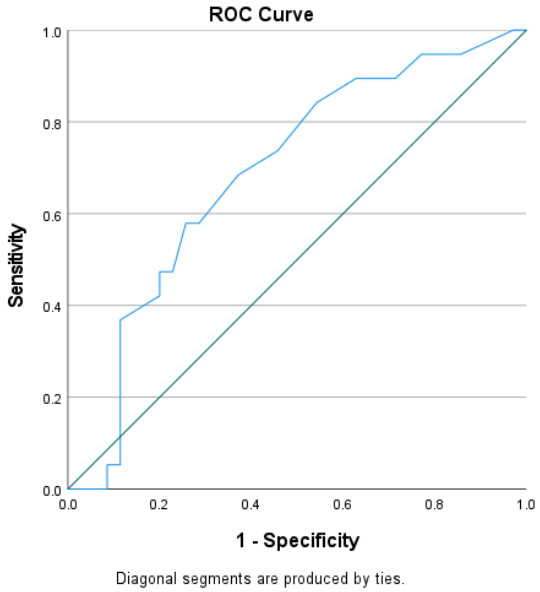


FIGURE 8: PREDICTIVE OUTCOME OF APACHE 2 SCORE AMONG VAP IN PULMONARY INDICATIONS OF VAP



- Sensitivity: 58% (0.58)
- Specificity**:** 74% (0.74)

At the point of highest sensitivity and specificity, cutoff value was taken as 23 for modified APACHE 2 score in this study.

AREA UNDER THE CURVE				
TEST RESULT VARIABLES: SCORES				
Area	**Std. Error[a]**	**Asymptotic Sig.[b]**	**Asymptotic 95% Confidence Interval**	
			Upper bond	Lower bond
0.688	0.075	0.024	0.541	0.834
a. Under the nonparametric assumption				
b. Null hypothesis: true area = 0.5				

When analyzing the ROC curve, we found an AUC of 0.676. We observed that a CPIS above 6 is associated with a sensitivity of 84% and a specificity of 46% in predicting the outcome of VAP in pulmonary cases.

TABLE 38: DISTRIBUTION OF DIAGNOSIS OF VAP AND PREDICTIVE OUTCOME OF APACHE 2 SCORE IN PULMONARY INDICATIONS OF VAP

APACHE 2 SCORE IN PULMONARY GROUP	PREDICTIVE OUTCOME		
	Death n %	Improved n %	Total
≤23	8(42.1%)	26 (74.3%)	34 (63%)
>23	11 (57.9%)	9 (25.7%)	20 (37%)
TOTAL	19 (100%)	35 (100%)	54 (100%)

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	5.469[a]	1	.019

GRAPH 38: DISTRIBUTION OF PREDICTIVE OUTCOME OF APACHE 2 SCORE IN PULMONARY INDICATIONS OF VAP

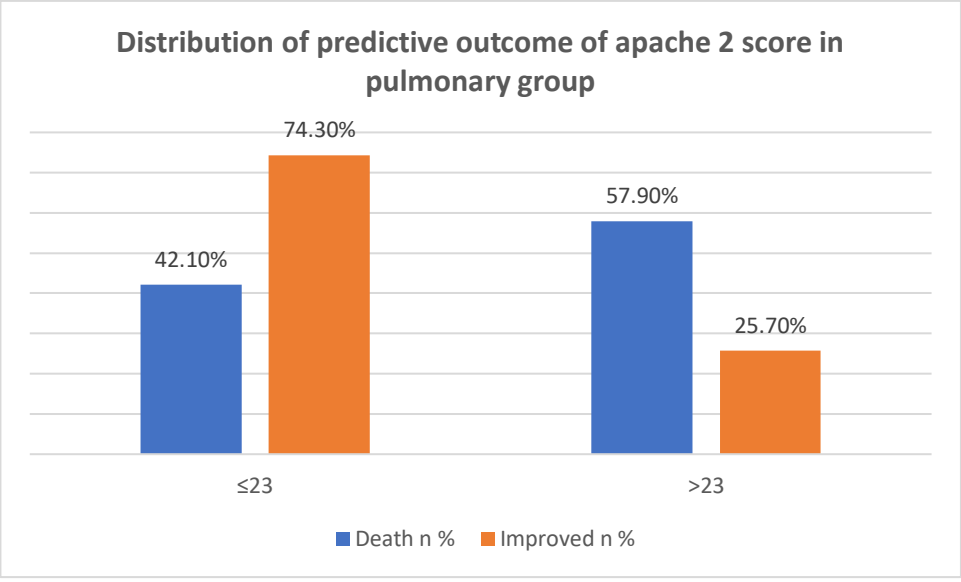
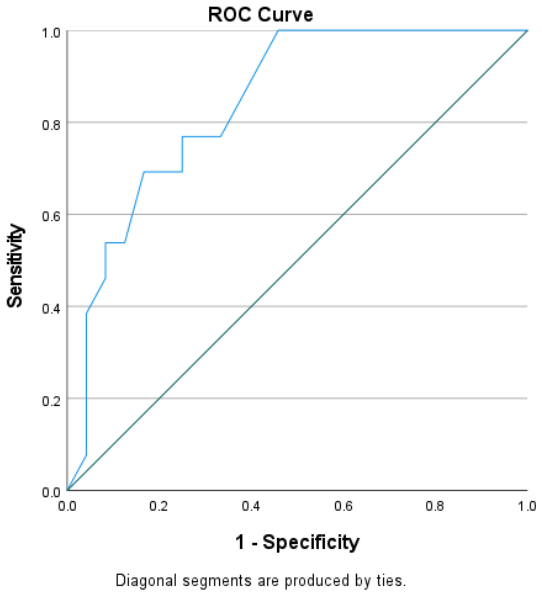


FIGURE 9: PREDICTIVE OUTCOME OF APACHE 2 SCORE AMONG VAP IN NON-PULMONARY INDICATIONS OF VAP



- Sensitivity: 100% (0.01)
- Specificity: 54% (0.54)

At the point of highest sensitivity and specificity, cutoff value was taken as 18 for modified APACHE 2 score in this study.

AREA UNDER THE CURVE				
TEST RESULT VARIABLES: SCORES				
Area	**Std. Error[a]**	**Asymptotic Sig.[b]**	**Asymptotic 95% Confidence Interval**	
			Upper bond	Lower bond
.841	0.064	0.001	0.715	0.965
a. Under the nonparametric assumption				
b. Null hypothesis: true area = 0.5				

TABLE 39: DISTRIBUTION OF DIAGNOSIS OF VAP AND PREDICTIVE OUTCOME OF APACHE 2 SCORE IN PULMONARY INDICATIONS OF VAP

APACHE 2 SCORE IN NON-PULMONARY GROUP	**PREDICTIVE OUTCOME**	

	Death n %	Improved n %	Total
≤18	0	13 (54.2%)	13 (35.1%)
>18	13 (100%)	11 (45.8%)	24 (64.9%)
TOTAL	13 (100%)	24 (100%)	37 (100%)

GRAPH 39: DISTRIBUTION OF DIAGNOSIS OF VAP AND PREDICTIVE OUTCOME OF APACHE 2 SCORE IN NON-PULMONARY INDICATIONS OF VAP

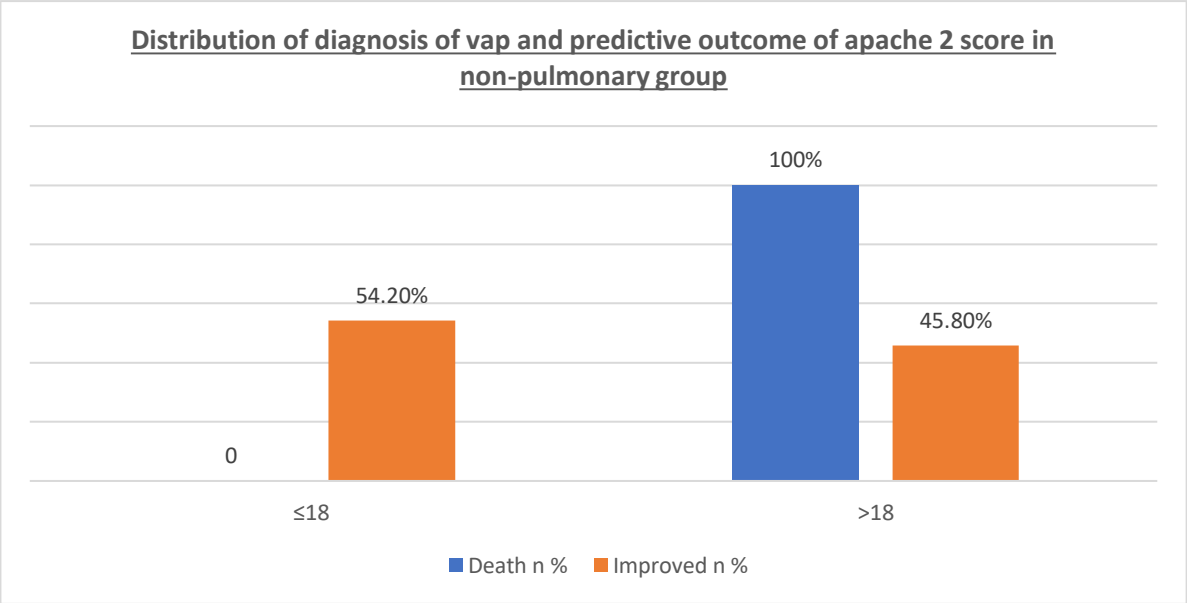
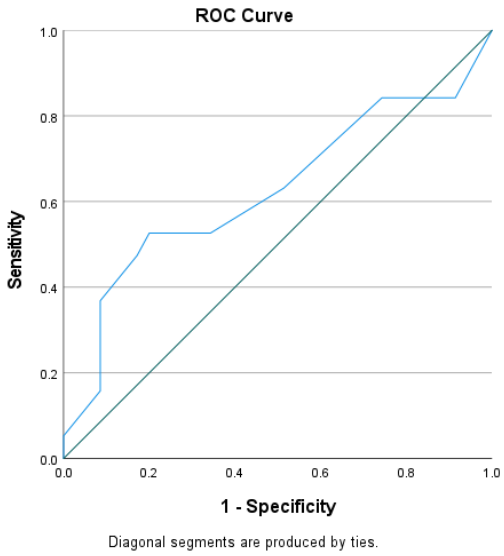


FIGURE 10: PREDICTIVE OUTCOME OF SOFA SCORE AMONG VAP IN PULMONARY GROUP



AREA UNDER THE CURVE				
TEST RESULT VARIABLES: SCORES				
Area	**Std. Error[a]**	**Asymptotic Sig.[b]**	**Asymptotic 95% Confidence Interval**	
			Upper bond	Lower bond
0.626	0.086	0.13	0.456	0.795
a. Under the nonparametric assumption				
b. Null hypothesis: true area = 0.5				

- Sensitivity: 53% (0.53)
- Specificity: 80% (0.80)

At the point of highest sensitivity and specificity, cutoff value was taken as 8 for modified SOFA score in this study.

TABLE 40: DISTRIBUTION OF PREDICTIVE OUTCOME OF SOFA SCORE IN PULMONARY INDICATIONS OF VAP

SOFA SCORE IN PULMONARY GROUP	PREDICTIVE OUTCOME		
	Death n %	**Improved n %**	**Total**
≤8	9 (47.4%)	28 (80%)	37 (68.5%)
>8	10 (52.6%)	7 (20%)	17 (31.5%)
TOTAL	19 (100%)	35 (100%)	54 (100%)

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	6.079[a]	1	.014

GRAPH 40: DISTRIBUTION OF DIAGNOSIS OF VAP AND PREDICTIVE OUTCOME OF SOFA SCORE IN PULMONARY INDICATIONS OF VAP

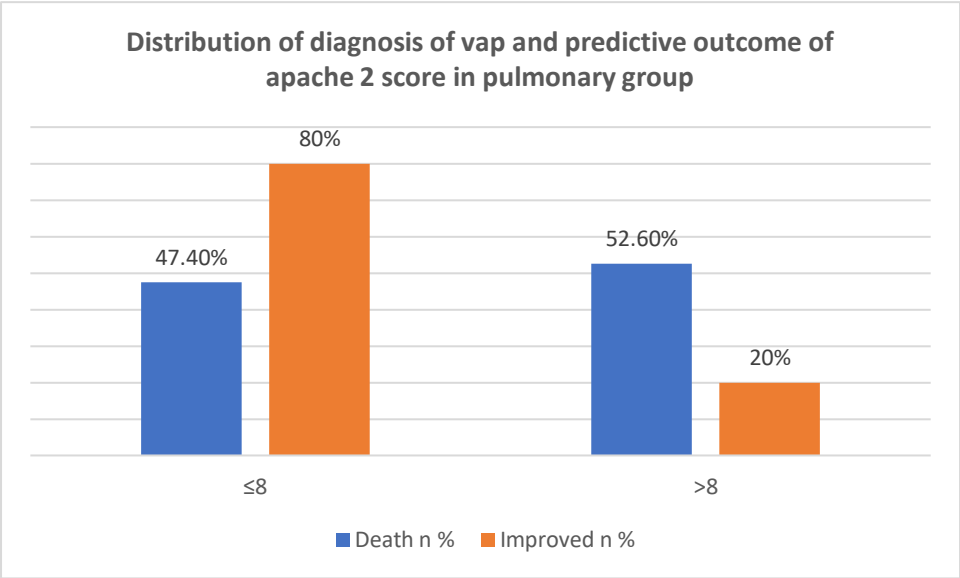
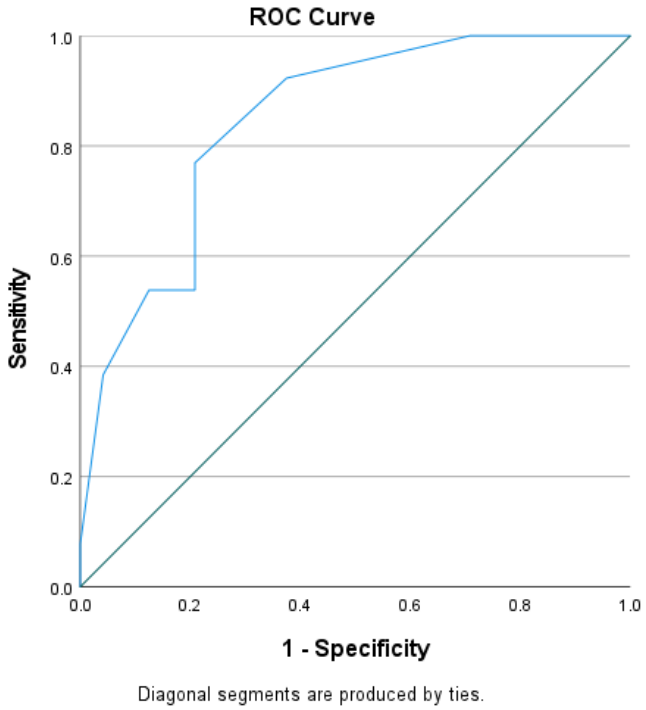


FIGURE 11: PREDICTIVE OUTCOME OF SOFA SCORE AMONG VAP IN NON-PULMONARY INDICATIONS OF VAP



AREA UNDER THE CURVE				
TEST RESULT VARIABLES: SCORES				
Area	**Std. Error[a]**	**Asymptotic Sig.[b]**	**Asymptotic 95% Confidence Interval**	
			Upper bond	Lower bond
.846	.064	.001	.721	.971
a. Under the nonparametric assumption				
b. Null hypothesis: true area = 0.5				

- Sensitivity: 77% (0.69)
- Specificity: 79% (0.71)

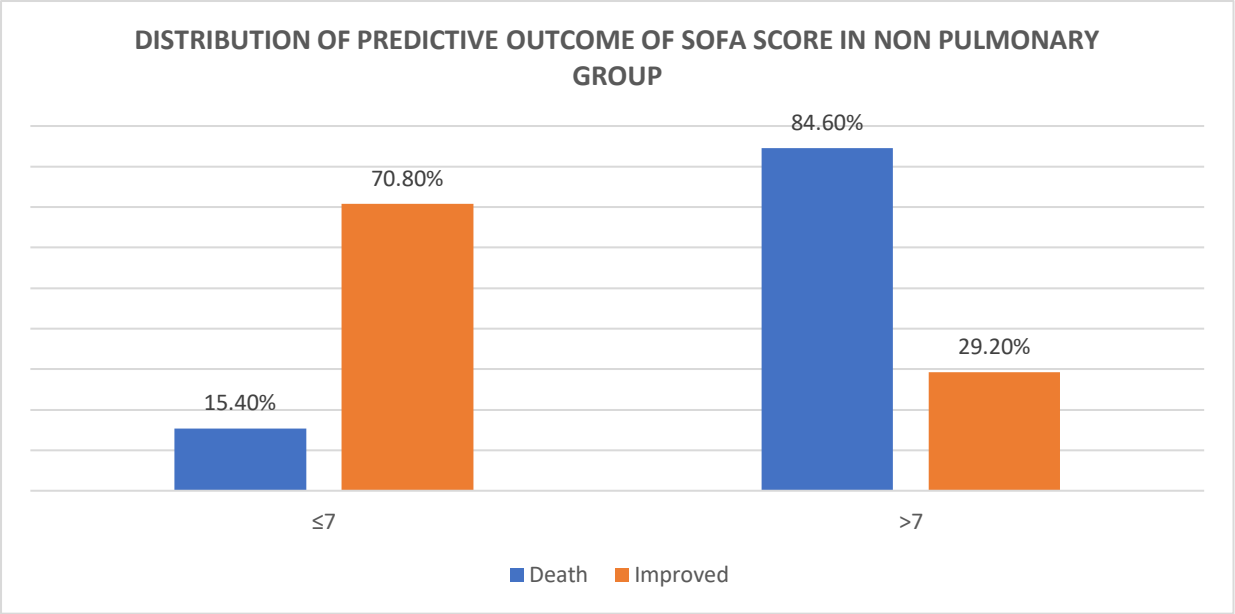
At the point of highest sensitivity and specificity, cutoff value was taken as 7 for modified SOFA score in this study.

TABLE 41: DISTRIBUTION OF PREDICTIVE OUTCOME OF SOFA SCORE IN NON-PULMONARY INDICATIONS OF VAP

SOFA SCORE IN NON-PULMONARY GROUP	PREDICTIVE OUTCOME		
	Death n%	**Improved n%**	**Total**
≤7	2 (15.4%)	17 (70.8%)	19 (51.4%)
>7	11 (84.6%)	7 (29.2%)	18 (48.6%)
TOTAL	13 (100%)	24 (100%)	37 (100%)

	Value		Asymptotic Significance (2-sided)
Pearson Chi-Square	10.378[a]		.001

GRAPH 41: DISTRIBUTION OF PREDICTIVE OUTCOME OF SOFA SCORE IN NON-PULMONARY INDICATIONS OF VAP


DISTRIBUTION OF PREDICTIVE OUTCOME OF SOFA SCORE IN NON PULMONARY GROUP
84.60%
70.80%
29.20%
15.40%
≤7
>7
Death
Improved

DISCUSSION

Ventilator-associated pneumonia (VAP) is a most significant healthcare-associated infection that occurs in patients on mechanical ventilation. It is a major concern in intensive care units (ICUs) due to its impact on patient outcomes, prolonged hospital stays, and increased healthcare costs. This study explores the Incidence, organisms causing VAP, and outcome for the prevention and treatment of VAP. In this study 126 VAP patients were randomized and allocated into two groups of Pulmonary and Non pulmonary based on the diagnosis on admission.

AGE AND GENDER DISTRIBUTION

AUTHOR AND YEAR	**Mean +SD of age in Pulmonary indications of VAP**	**Gender in Pulmonary indications of VAP**
Hassan Mumtaz et al. 2023[69]	53.5 years	Male: 64.88% Female:35.12%
Gopi C Khilnan et. al, 2022[70]	62.45 ± 8.32 years	Male: 58.8% Female:47%
But. A et.al. 2017[71]	69.9 ± 15.9 years	Male: 79% Female: 21%
Vijay Hadda et.al, 2014[72]	61 ± 11.3 years	Male: 58.7% Female:41.3%

Evans R Fernández-Pérez, M.D. et.al, 2014 (73)	71 (57 to 78 years)	Male: 52% Female:48%
OUR STUDY	58 ± 16.4 years	Male: 71.4% Female: 28.5

The age of the patients in the Pulmonary and Non pulmonary groups being studied ranged from 18 years to 85 years & the mean age was 58 ± 16.4 years and 49 ± 18.03

AUTHOR AND YEAR	**Mean +SD of age in Non-pulmonary indications of VAP**	**Gender in Non-pulmonary indications of VAP**
Battaglini D et.al, 2023 (74)	54 (36-65 years)	Male: 45.6% Female: 37.6%
Watson K et. al, 2022 (75)	58.2 ± 14.2 years	Male: 64% Female:36%
Suljevic I et.al, 2020 (76)	60.4 ± 16.8 years	Male: 51.6% Female: 48.4%
Robba C et.al, 2020 (77)	39.5 (25-55 years)	Male: 83.6% Female: 16.3%

Roxanne Buterakos DNP et.al, 2015 [78]	43.9 ±17.9 years	Male: 80.9% Female: 19.1%
OUR STUDY	49 ± 18.03	Male: 76.1%% Female: 26.1%

INCIDENCE OF VAP:

In this study, out of 254 Mechanically ventilated patients from different ICU's the incidence of VAP in pulmonary group is 47% and non-pulmonary group is 52.5%.

Author and year	**n**	**Patients with VAP**	**Incidence of VAP in Pulmonary group**
Dr. Satakshi Manwan et.al, 2024 [79]	100	30	30%
Luis Filipe Reyes et.al, 2023 [80]	50.5%		
Cihan semet et.al, 2023 [81]	366	83	22.9%
Chernet Manaye Belay et.al, 2022 [82]	312	87	27.9%
Laurent Papazian et.al, 2020 [83]	5%– 40%		
	153	35	22.8%
OUR STUDY	**134**	**63**	**47%**

Author and year	**n**	**Patients with VAP**	**Incidence of VAP in non-pulmonary group**
Patil et.al, 2025 [84]	96	53	54%
Diego Enrique Prieto-	223	131	58.7%

Alvarado et.al, 2024 (85)			
Sina Chen et.al, 2023 (86)	2301	970	42%
Zhang et.al, 2019 (87)	78	27	35%
Pierre Esnault et.al, 2017 (88)	175	106	57.4%
OUR STUDY	**120**	**63**	**52.5%**

ET/TRACHEOSTOMY SECRETIONS CULTURE ORGANISM AMONG VAP CASES IN PULMONARY AND NON-PULMONARY INDICATIONS OF VAP

Among the pulmonary cases (n=63), the three most frequently isolated pathogens were the Gram-negative organisms which are *Acinetobacter baumannii Complex* (16 cases, 25.39%), *Klebsiella pneumoniae* (14 cases, 22.2%), and *Pseudomonas aeruginosa* (8 cases, 12.69%). Ghopi C Khilnani et.al[67], in 2022 in a study found that *Acinetobacter baumannii* was the most frequent organism (n = 8, 47%), followed by *Klebsiella pneumoniae* (n = 5, 29%), *Pseudomonas aeruginosa* (n = 1, 6%), in Pulmonary group which is similar to our study. Other notable organisms included *Serratia marcescens* (5 cases, 7.93%), *Staphylococcus aureus* and *Escherichia coli* (4 cases each, 6.34%).

Most common gram-positive organism isolated is *Staphylococcus aureus* (6.34%) in our study. Additionally, Klebsiella pneumoniae (MDRO) and Streptococcus pneumoniae were detected in 3 cases (4.76% each). Less common organisms such as Klebsiella oxytoca (2 cases, 3.17%) and Klebsiella aerogenes, Enterobacter cloacae complex, Enterobacter aerogenes, and MRSA Staphylococcus aureus (each with 1 case, 1.58%) were also identified. In a similar study by Dr. Satakshi Manwani et. al, (79), in 2024 provided an overview of pathogens identified in VAP patients in which the most common pathogens isolated were

Pseudomonas aeruginosa (33%), *Staphylococcus aureus* (including MRSA, 27%), *Klebsiella pneumoniae* (20%), *Acinetobacter baumanni* (13%), and *Escherichia Coli* (7%) in respiratory failure patients who developed VAP which is nearly similar to our study.

Akshaya N. Shetti [(89)] et. al, in 2022 in their study with sample size of 240 patients with 21 positive VAP cases 20 were affected by gram-negative organisms and 1 patient was affected by gram-positive organisms. Most commonly isolated bacteria in their study were *Acinetobacter species* in 9 (38.09%) patients and *Pseudomonas aeruginosa* in 9(38.09%) patients each and E. coli in 2 (9.52%) patients and *Klebsiella species* in 2(9.52%) patients each and *Staphylococcus aureus* in 1(4.76%) patient.

Sona Hinkova[(90)] et.al, in 2025 in their study showed that the common pathogens causing VAP were *Pseudomonas aeruginosa* (28.1%), *Klebsiella pneumoniae* (26%), *Acinetobacter* spp. (22%), and *Serratia marcescens* (6.0%) which is correlated to our study.

In the non-pulmonary group (n=63), *Acinetobacter baumannii Complex* was again the most prevalent pathogen, accounting for 15 cases (23.8%), followed closely by *Klebsiella pneumoniae* with 14 cases (22.2%), *Klebsiella pneumoniae* (MDRO) which are the most common gram-negative organisms and which is similar to a study conducted by Abdul Rehman Azam[(91)] et.al, in 2025 where *Acinetobacter baumanni* and *Klebsiella pneumoniae* are the most common gram-negative organisms.

Flavia Eniko Pinto[(92)] et. al, in 2024 in their study with 1166 VAP cases found that the predominant organisms are *Acinetobacter baumanni, Staphylococcus aureus, Pseudomonas, and Klebsiella*, accounted for 70%-80% of cases.

Roxanne Buterakos DNP[(78)] et.al, in 2022 in their study found that *Staphylococcus aureus* was the most prevalent gram-positive organism in traumatic brain injuries and blunt injuries in SICU which is correlated with our study.

There is positive correlation between Serratia marcescens between two groups with more number of organisms isolated in pulmonary group than Non pulmonary group and is

statistically significant with p value of 0.0230 from chi square test in our study which is correlated with Andria Barrios[93] et.al, in 2025 in their study in intensive care found that the majority of serratia marcescens strains in intensive care are isolated from respiratory samples (81.5%).

ET/TRACHEOSTOMY CULTURE SECRETIONS ORGANISM AND ITS RESISTANCE AND SENSITIVITY PATTERNS

1) ACINETOBACTER BAUMANNI AND ITS RESISTANCE AND SENSITIVITY PATTERN:

In our study, amongst 31 cases with *Acinetobacter baumanni* infection we identified 16 cases (25.39%) in the pulmonary group and 15 cases (23.8%) in the non-pulmonary group.

Antibiotic resistance and sensitivity patterns:

Acinetobacter baumanni shows resistance to Amikacin and gentamicin in 11 pulmonary cases (68.75%) and 13 non-pulmonary cases (86.66%). Resistance to Cefoperazone/Sulbactam was higher in the non-pulmonary group (46.66%) compared to pulmonary cases (18.75%).

Both Imipenem and Meropenem showed high resistance rates in pulmonary (93.75%; 100%) and Non pulmonary groups (86.6% ; 93.3%) which is correlated with Yuting Li et.al[94] in 2024 where *Acinetobacter baumanni* complex showed partial resistance to carbapenems and penicillins.

In a similar study by Khalil KA et.al[95], in 2025 found that *Acinetobacter baumanni* is highly resistant to carbapenems (Imipenem, Meropenem), Fluoroquinolones, Cefoperazone/Sulbactam and Ceftriaxone which is in contrary to our study

Our study revealed Levofloxacin, Amoxicillin/Clavulanic Acid resistance was significantly higher in the non-pulmonary group with p = 0.0023*, p = 0.0020* and p = 0.0018* which is similar to a study by Edhem Unver et al[96], in 2019 where most of the *Acinetobacter* isolates were resistant to ciprofloxacin, Levofloxacin, Amoxicillin/Clavulinic acid and carbapenems.

Sensitivity to Trimethoprim/Sulfamethoxazole was observed in 31.25% of pulmonary cases, while no non-pulmonary cases were sensitive to this drug. Sensitivity to Tigecycline was noted in 62.5% of pulmonary cases and 100% of non-pulmonary cases with p value 0.0018*. Edhem Unver et. al[96], in 2019 found that the most sensitive antibiotics against *Acinetobacter spp.* were tigecycline (95%), trimethoprim sulfamethoxazole (49.1%) which is similar to our study. In a study conducted by Patil et.al[84], in 2025 found that all gram-negative bacteria most commonly *Acinetobacter baumanni* are highly resistant to all antibiotics except Tigecycline and Colistin.

2) ACINETOBACTER BAUMANNI MDR AND ITS RESISTANCE AND SENSITIVITY PATTERN

In our study of 126 cases with VAP, 2 cases (6.45%) were identified as *Acinetobacter baumanni* MDR strains, both of which were from the **non-pulmonary group**.

Antibiotic resistance and sensitivity patterns:

Our study revealed that both MDR cases were 100% resistant to Amikacin, Ceftriaxone, Piperacillin/Tazobactam and Meropenem. Whereas 1 case (50%) was resistant to Cefoperazone/Sulbactam, Ciprofloxacin, Levofloxacin and Trimithoprim/Sulfamethoxazole which is nearly similar to a study done by Vishal B Shete et.al[97] where VAP due to MDR *Acinetobacter* develops fast resistance to different groups of antibiotics including aminoglycosides, fluoroquinolones, and carbapenems.

We found that 1 case (50%) was sensitive to Levofloxacin, Trimethoprim/sulfamethoxazole and Tigecycline which is aligned with a study conducted in a single centre by Patil et.al[84], 2025 where most patients who were admitted to the ICU for neurological indication showed MDR pathogens (23 patients; 95.8%), than patients who were admitted to the ICU for respiratory indication and most of the gram negative bacteria are resistant to Ceftriaxone (100%), Piperacillin/Tazobactam (95%) and Meropenem (100%) and there is no resistance to colistin (100%) and tigecycline which are the effective drugs for *Acinetobacter baumanni*

MDR pathogens.

3) KLEBSIELLA PNEUMONIAE AND ITS RESISTANCE AND SENSITIVITY PATTERN

Our study showed 28 cases of *Klebsiella pneumoniae* infections, with 14 cases in each group.

Antibiotic Resistance and sensitivity patterns:

Our study found that Amikacin & Gentamicin are resistant in both pulmonary and non-pulmonary cases. Ceftriaxone shows 92.85% resistance in both pulmonary and non-pulmonary cases. Ciprofloxacin & Levofloxacin shows higher resistance in non-pulmonary cases. Cefuroxime & Cefuroxime Axetil were 57.14% resistance in pulmonary cases. Amoxicillin/Clavulanic Acid showed 71.42% resistance in non-pulmonary cases vs. 21.4% in pulmonary cases p=0.009*. Piperacillin/Tazobactam showed 78.5% resistance in pulmonary cases vs. 7.14% in non-pulmonary cases with p=0.0002*. Imipenem showed 50% resistance in non-pulmonary cases vs. 7.14% in pulmonary cases (p=0.01*).

Non-pulmonary cases had higher resistance to Imipenem and Amoxicillin/Clavulanic Acid. Pulmonary cases had higher resistance to Piperacillin/Tazobactam and Cefuroxime derivatives.

Amikacin and Gentamicin sensitivity was observed in 21.4% of pulmonary cases and 42.8% of non-pulmonary cases and Tigecycline sensitivity was observed in 64.28% of pulmonary cases and 92.85% of non-pulmonary cases, Trimethoprim/Sulfamethoxazole sensitivity was higher in non-pulmonary cases (50%) compared to pulmonary cases (35.7%). which is similar to a study Flávia Eniko Pinto et.al[92], 2024 where the majority of cases showed sensitivity to Amikacin, Tigecycline and 35% for Trimethoprim/Sulfamethoxazole amongst Klebsiella pneumoniae isolates. In a similar study conducted by Edham Unver et.al[96], in 2019 found that tigecycline (85.7%) and Amikacin (85.7%) are the most effective antibiotics in Klebsiella pneumoniae infection.

Resistance to ceftriaxone and meropenem was notably high in both pulmonary and non-

pulmonary cases which is correlated with a study by Khalil A et.al, [95] where they found klebsiella pneumonia shows high resistance to fluoroquinolones, piperacillin/tazobactam, amoxicillin/clavulanic acid.

4) KLEBSIELLA OXYTOCA AND ITS RESISTANCE AND SENSITIVITY PATTERN

Out of 5 cases of *Klebsiella oxytoca* infections in our study, 2 were pulmonary and 3 were non-pulmonary cases in our study.

Antibiotic resistance and sensitivity patterns:

Klebsiella oxytoca showed 100% resistance to Amikacin, Cefoperazone/Sulbactum, Ceftriaxone and fosfomycin in both pulmonary and non-pulmonary cases. Ciprofloxacin, Cefuroxime and Cefuroxime Axetil pulmonary cases. Piperacillin/Tazobactam Resistance was significantly higher in non-pulmonary cases with p=0.045*. Carbapenem Resistance was higher in non-pulmonary cases (100%) compared to 50% in pulmonary cases.
Amikacin sensitivity was observed in 100% of pulmonary cases, but no sensitivity was noted in non-pulmonary cases (p=0.045*). Tigecycline sensitivity was observed in 66.6% of non-pulmonary cases.

Statistically significant resistance differences were found for piperacillin/tazobactam resistance (higher in non-pulmonary cases, p=0.045)* and amikacin sensitivity (higher in pulmonary cases, p=0.045)*.

Jing Yang et.al[98], in 2021 in his study in klebsiella oxytoca antimicrobial resistance proven that many isolates of the complex have acquired genes mediating resistance to a variety of antimicrobial agents, including β-lactams (e.g., penicillins, cephalosporins, and carbapenems), aminoglycosides, quinolones, and colistin which is correlated with our study.

5) KLEBSIELLA SPP PNEUMONIAE MDRO AND ITS RESISTANCE AND

SENSITIVITY PATTERN

This study highlights widespread multidrug resistance in *Klebsiella spp. pneumoniae (MDRO)* infections, with extensive resistance to cephalosporins, fluoroquinolones, and aminoglycosides.

Our study revealed 100% resistance to ceftriaxone and cefoperazone/sulbactam in non-pulmonary cases (p=0.033*) suggests the presence of extended-spectrum beta-lactamase (ESBL) or AmpC beta-lactamase-producing *K. pneumoniae*. Additionally, piperacillin/tazobactam resistance was significantly higher in non-pulmonary cases (p=0.033*).

Amikacin showed significantly better sensitivity in pulmonary cases (100%) compared to non-pulmonary cases (16.6%, p=0.02*). Resistance to ciprofloxacin and levofloxacin was 100% in non-pulmonary cases and 66.6% in pulmonary cases (p=0.033*), indicating severe fluoroquinolone resistance. In a similar study conducted by Ghazal Bayatinejad et.al, [(99)], in 2023 had proven that combinations of colistin-meropenem and amoxicillin/clavulanate in combination with meropenem, colistin, or amikacin showed synergism against 60–70% MDR Klebsiella pneumoniae isolates.

6) KLEBSIELLA AEROGENES AND ITS RESISTANCE AND SENSITIVITY PATTERN

Our study revealed 2 cases of *Klebsiella aerogenes* with 1 case in each group.

Antibiotic Resistance and Sensitivity

The antibiotic susceptibility profile showed 100% sensitivity to all tested antibiotics including Aminoglycosides (Amikacin, Gentamicin), Cephalosporins (Cefoperazone/Sulbactam), Ceftriaxone, Cefepime, Fluoroquinolones (Ciprofloxacin, Levofloxacin), Beta-lactams (Piperacillin/Tazobactam), Carbapenems (Imipenem, Meropenem), Fosfomycin and Tigecycline without any resistance in both group which is correlated with a similar study done by Adel Malek et.al[(100)], of next generation sequencing of Klebsiella aerogenes isolates

showed high phenotypic susceptibility to all antibiotics including Aminoglycosides, Cephalosporins, Ceftriaxone.

7) PSEUDOMONAS AERUGINOSA AND ITS RESISTANCE AND SENSITIVITY PATTERN

A total of 12 cases of *Pseudomonas aeruginosa* infections were identified, with 8 pulmonary and 4 non-pulmonary cases in our study.

Antibiotic Resistance and Sensitivity Patterns

In our study, high resistance was observed against certain beta-lactams, including Cefuroxime (62.5%) and Cefuroxime Axetil (62.5%), which were only resistant in pulmonary cases. Levofloxacin and Trimethoprim/Sulfamethoxazole resistance was higher in non-pulmonary cases (p=0.03*). Ceftriaxone resistance (50%) was observed only in pulmonary cases. Imipenem and Meropenem resistance was similar in both groups (~37.5% in pulmonary and ~25% in non-pulmonary cases). Tigecycline showed moderate sensitivity (25-50%) in both groups.

In our study, Pulmonary isolates showed higher resistance to cephalosporins (Cefuroxime, Cefuroxime Axetil, Ceftriaxone). Non-pulmonary isolates had higher resistance to fluoroquinolones (Levofloxacin, Ciprofloxacin) and Trimethoprim/Sulfamethoxazole. Carbapenem resistance (Imipenem and Meropenem) was moderate and similar between both groups which is aligned to a study conducted by Flavia Eniko Pinto et.al[92], revealed most of the *Pseudomonas aeruginosa* isolates showed high sensitivity to Aminoglycosides (>90%) and low sensitivity to Cephalosporins.

Edhem Unver et.al [96], in their study revealed that Acinetobacter, Pseudomonas aeruginosa have high carbapenem resistance in recent years. Antibiotic susceptibilities of Pseudomonas were found to be colistin (94.1%), ceftazidime (57.8%), gentamicin (55.5%), ciprofloxacin (50%), amikacin (50%), and piperacillin/tazobactam (42.1%) which is nearly in contrary with our study.

8) PSEUDOMONAS AERUGINOSA MDR AND ITS RESISTANCE AND SENSITIVITY PATTERN

Our study showed 2 cases of MDR *Pseudomonas aeruginosa* infections, both from non-pulmonary group.

Antibiotic Resistance and Sensitivity Patterns:

In our study, high resistance (100%) was observed against fluoroquinolones (Ciprofloxacin, Levofloxacin) and Piperacillin/Tazobactam, indicating severe multidrug resistance. 50% resistance was seen against Amikacin, Gentamicin, Cefoperazone/Sulbactam, Ceftriaxone, Imipenem, Meropenem, and Trimethoprim/Sulfamethoxazole. Sensitivity was limited, with only one case (50%) showing susceptibility to Amikacin, Cefoperazone/Sulbactam, Imipenem, and Meropenem.

In a single centre retrospective case control study conducted in 2020 by Ann fan yang et.al[(101)], found that MDR Pseudomonas Aeruginosa Resistance was most common to aztreonam (39.9%), followed by cefepime (26%), gentamicin (25.6%), piperacillin/tazobactam (24.4%), levofloxacin (21.7%), ciprofloxacin (19%), meropenem (15.1%), amikacin (8.1%), and tobramycin (2.3%) which is in contrary to our study.

9) ENTEROBACTER CLOACAE COMPLEX AND ITS RESISTANCE AND SENSITIVITY PATTERN

A single case of *Enterobacter cloacae complex* infection was identified in a pulmonary sample in our study.

Antibiotic Resistance and Sensitivity Patterns

Complete resistance (100%) was observed against Cefuroxime, Cefuroxime Axetil, Colistin, and Fosfomycin, limiting treatment options. The isolate was sensitive 100% to multiple antibiotics including Amikacin, Gentamicin, Cefoperazone/Sulbactam, Ceftriaxone, Ciprofloxacin, Piperacillin/Tazobactam, and Carbapenems (Imipenem, Meropenem) which is similar to a study by Medini K Annavajhala et.al, [(102)], in their study found that ECC is more

resistant to penicillins and 1st and 2nd generation cephalosporins due to low expression of chromosomal *ampC* genes encoding cephalosporinase.

10) ESCHERICHIA COLI AND ITS RESISTANCE AND SENSITIVITY PATTERN

A total of seven cases of *Escherichia coli* infections were identified, with four pulmonary and three non-pulmonary isolates in our study.

Antibiotic resistance and sensitivity patterns:

In our study, *E. coli* showed high Resistance to Ceftriaxone, Ciprofloxacin, and Amoxicillin/Clavulanic Acid exhibited resistance in both pulmonary and non-pulmonary isolates, with higher resistance in non-pulmonary infections.

E. coli isolates were 100% susceptible to Meropenem and Tigecycline, suggesting these as reliable treatment options. Amikacin and Gentamicin showed strong effectiveness, with 75–100% susceptibility rates across pulmonary and non-pulmonary infections which is in contrary to a study by Edham Unver et al[(96)], in 2019 where Tigecycline and Amikacin are 100% sensitive to E. coli and Gentamicin, Meropenem shows partial 66.6% sensitivity.

In a similar study by Edhem Unver et.al[(96)], E. coli was 100% sensitive to Tigecycline, Colistin, Amikacin and partial sensitivity to Gentamicin, Piperacillin/Tazobactam.

11) SERRATIA MARCESCENS AND ITS RESISTANCE AND SENSITIVITY PATTERN

In our study, a total of five cases of *Serratia marcescens* infections were identified, all from pulmonary group.

Antibiotic Resistance and Sensitivity Patterns

This study showed all 5 cases are 100% resistant to Cefuroxime, Cefuroxime Axetil, and Amoxicillin/Clavulanic Acid, indicating intrinsic beta-lactam resistance. 80% resistance to Cefoperazone/Sulbactam, Ceftriaxone, Cefepime, and Carbapenems (Imipenem, Meropenem, Ertapenem), suggesting extensive drug resistance (XDR).

Trimethoprim/Sulfamethoxazole and Tigecycline showed 100% sensitivity, making them

potential treatment options. Amikacin was effective in 80% of cases which is similar to a study conducted by Edham Unver et.al[96], in 2019 where Tigecycline, Amikacin, Trimethoprim/Sulfamethoxazole, Fluoroquinolones showed 100% sensitivity to serratia marcescens and is highly sensitive to Tigecycline, Trimethoprim/Sulfamethoxazole, Amikacin, Gentamicin, fluoroquinolones and piperacillin/tazobactam making them effective against these isolates.

12) STAPHYLOCOCCUS AUREUS AND ITS RESISTANCE AND SENSITIVITY PATTERN

A total of 10 *Staphylococcus aureus* isolates were identified in this study, with 4 pulmonary and 6 non-pulmonary cases.

Antibiotic Resistance and Sensitivity Patterns

There was 100% resistance to Cefuroxime Axetil, Amoxicillin/Clavulanic Acid, Meropenem, and Trimethoprim/Sulfamethoxazole P=0.0027* and 50% resistance to Ciprofloxacin and Piperacillin/Tazobactam in pulmonary cases.100% resistance to Erythromycin in non-pulmonary cases P=0.0027* and Benzyl Penicillin and Nitrofurantoin showed 50% resistance in non-pulmonary isolates.

Levofloxacin was 100% effective in non-pulmonary isolates with P=0.0027*. Clindamycin was 100% effective in non-pulmonary cases and 50% effective in pulmonary cases. Cefoperazone/Sulbactam was 66.6% effective in non-pulmonary cases P=0.0455*.

In a similar study done by Fluvea Eniko Pinto et.al[92], Staphylococcus aureus showed 100% resistance to teicoplanin, tetracycline, tigecycline and vancomycin.

In another study conducted by Mojtaba Ahmadinejab et.al, [103], showed that *Staphylococcus aureus* showed highest resistance to trimethoprim/Sulfamethoxazole, cephalosporins (75.6%) and cloxacillin and susceptibility to vancomycin and linezolid (100%) which is nearly similar to our study.

13) STAPHYLOCOCCUS AUREUS (MRSA) AND ITS RESISTANCE AND

SENSITIVITY PATTERN

A total of 6 Methicillin-Resistant *Staphylococcus aureus* (MRSA) isolates were identified, with 1 pulmonary and 5 non-pulmonary cases in our study.

Antibiotic Resistance and Sensitivity Patterns

In our study, there was 100% resistance to Ciprofloxacin and Levofloxacin in both pulmonary and non-pulmonary isolates P=0.0463*, indicating fluoroquinolone resistance. Erythromycin resistance was detected in 3 non-pulmonary isolates, suggesting possible inducible macrolide resistance.

There was 100% sensitivity to Tigecycline and Trimethoprim/Sulfamethoxazole in all isolates P=0.0463*, making these viable treatment options.

Vancomycin, Linezolid, and Tetracycline showed good efficacy 60–100% sensitivity, suggesting their role in MRSA therapy which is aligned with a study done by Khalil A et.al[(95)], revealed that there was 100% resistance to oxacillin and amoxicillin clavulanate and all cases with MRSA 100% sensitive for tetracycline, vancomycin, linezolid, tigecycline, and trimethoprim-sulfamethoxazole.

14) STREPTOCOCCUS PNEUMONIAE AND ITS RESISTANCE AND SENSITIVITY PATTERN

A total of 3 pulmonary *Streptococcus pneumoniae* isolates were identified in our study.

Antibiotic Resistance and Sensitivity Patterns:

There was 100% resistance to Ceftriaxone, Meropenem, Cefuroxime, and Cefuroxime Axetil, indicating significant beta-lactam resistance and 100% sensitivity to Amikacin, Cefoperazone/Sulbactam, Ciprofloxacin, Cefepime, Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Trimethoprim/Sulfamethoxazole, and Ertapenem. This suggests fluoroquinolones and combination beta-lactam inhibitors remain effective treatment options.

Li Yang et.al [(104)], in their study proven that *Streptococcus pneumoniae* revealed high resistance rates to penicillin (45%), erythromycin (60%), and clindamycin (40%), and

maintaining low resistance to ceftriaxone (10%) and levofloxacin (5%).

15) ESCHERICHIA COLI (CRE) AND ITS RESISTANCE AND SENSITIVITY PATTERN

Only 1 non-pulmonary case of carbapenem-resistant *E. coli* (CRE) was identified.

Antibiotic resistance and sensitivity patterns:

There was 100% resistance to carbapenems (Imipenem, Meropenem), Piperacillin/Tazobactam, fluoroquinolones, Ceftriaxone and Amikacin. In a study of Hasan Ejas et.al [(105)], showed about 90% of the CRE patients showed resistance to fluoroquinolones and carbapenems. The frequency of amikacin resistance was 29% and that of fosfomycin resistance was 33% and Tigecycline was 100% sensitive to CRE.

16) CITROBACTER FREUNDII AND ITS RESISTANCE AND SENSITIVITY PATTERN

In our study, only one non-pulmonary case of *Citrobacter freundii* was identified.

Antibiotic resistance and sensitivity patterns:

Citrobacter freundii isolates showed 100% resistance to aminoglycosides, Ceftriaxone, fluoroquinolones, β-lactamase inhibitor (Piperacillin/Tazobactam), Meropenem and Trimethoprim/Sulfamethoxazole. Only Amoxicillin/Clavulanic Acid showed 100% sensitivity. In a study from a teritiary hospital, Ruben S Maghembe et.al [(106)], identified the strain exhibited phenotypic resistance to trimethoprim/sulfamethoxazole, with indeterminate phenotypes for ciprofloxacin, levofloxacin, and intermediate sensitivity to amoxicillin/ clavulanic acid. Recently from Western Uganda, unsequenced C. freundii isolates are identified from sepsis with resistance to cotrimoxazole and carbapenems.

OUTCOME OF VENTILATOR ASSOCIATED PNEUMONIA IN PULMONARY AND NON-PULMONARY INDICATIONS FOR MV

	Pulmonary n=63 %	Non pulmonary n=63 %	Total n %	Odds Ratio (OR)
Death	19(30.1%)	13(20.6%)	32(25.3%)	
Improved	35(55.5%)	24(38%)	59(46.8%)	1.002

A total of 35 patients are excluded from our study due to various reasons like Discharge against medical advice (DAMA) due to financial issues, family issues and referral to higher centres. Out of 91 patients, overall mortality rate in our study in VAP patients is 32 (25.3%) and improvement is seen in 59 (46.8%) patients. In a prospective observational study by Neelima Ranjan et.al([76]), (a study of VAP) VAP has been associated with overall mortality rates of 47.3% and another study conducted by Vijay Hadda et.al, [(72)], showed 51% mortality. Our study shows high mortality in pulmonary group 19 (30.1%) compared to Non pulmonary group 13 (20.6%). Death due to Cardio-Pulmonary Arrest (CP Arrest) was significantly more frequent in pulmonary cases (23.8%, p=0.0002*), suggesting a greater impact of respiratory compromise on mortality. Death due to Sepsis was more common in non-pulmonary cases (17.5%) with p=0.05*, emphasizing the increased risk of systemic infections in Non-pulmonary patients.

Improvement Rates were significantly higher in pulmonary cases 35 (55.6%) compared to non-pulmonary cases 24 (38.1%) which is statistically significant with p=0.05*, possibly reflecting better treatment response for respiratory infections. An Odds Ratio of **1.002** indicates that the likelihood of mortality is almost the same for both pulmonary and non-pulmonary infections

In a similar study conducted by Rinuado M et. al[(107)], demonstrated that COPD is associated with higher ICU mortality in patients with VAP and a broad range has been reported on ICU mortality of 38% to 60% in VAP patients with COPD. In another prospective cohort study conducted by Dr. Satakshi Manwani et. al, [(79)], in 2024 revealed higher ICU mortality rates (50%) in VAP patients with severe pneumonia and respiratory failure.

Caiden Taowei Lu et.al [108], in their study showed that the ICU mortality rate in the patients with COPD is 31% and VAP patients without COPD is 35%.

PREDICTIVE OUTCOME OF DIFFERENT SCORES IN VAP AMONG PULMONARY AND NON-PULMONARY INDICATIONS FOR MV

1) PREDICTIVE VALUE OF MODIFIED CPIS SCORE IN PULMONARY AND NON PULMONARY INDICATIONS OF VAP

In our study we observed area under the curve of 0.702 **(95% CI: 0.559–0.844, p = 0.015)**, We observed that a CPIS >4 is associated with a sensitivity of 84% and a specificity of 46% in picturing the mortality in VAP. This high sensitivity implies that the modified CPIS is effective in identifying patients at higher risk of mortality, though its lower specificity indicates that a proportion of survivors may still be misclassified as high risk which is similar to a study conducted by Mircea Stoian et.al [109], in 2024. Our study found a significant difference in mortality rates based on CPIS scores. Among patients with a CPIS score of ≤4, the mortality rate was 15.8%, whereas for those with a CPIS score of >4, the mortality rate increased to 84.2%. Previous studies shows that a higher CPIS (typically >6) has been associated with **increased mortality** in patients with VAP.

When analyzing the ROC curve, we found an AUC of **0.676 (95% CI: 0.47–0.883, p = 0.08)**. We observed that a CPIS above 6 is associated with sensitivity of 54% and a specificity of 79% **in forecasting the mortality** in VAP. The moderate sensitivity suggests that the modified CPIS may not detect all high-risk patients, but its higher specificity indicates better accuracy in identifying survivors. The study found a significant difference in mortality rates based on CPIS scores. Among patients with a CPIS score of **<6**, the mortality rate was **46.2%**, whereas for those with a CPIS score of >6, the mortality rate was 53.8% among Non-pulmonary cases.

Xiao-Yu Zhou et. al[110], conducted a single centre study and analyzed that the CPIS does not have good discrimination power for predicting mortality in neurological and surgical patients. CPIS may be a useful for predicting the attributable mortality of VAP.

Demosthenes Makris et.al[111], in a study of impact of COPD with VAP, CPIS were significantly higher in COPD patients compared to patients without COPD

PREDICTIVE VALUE OF APACHE 2 SCORE IN PULMONARY AND NON - PULMONARY INDICATIONS FOR MV

Our study identified a modified APACHE II score of 23 as the cutoff for predicting mortality, with a sensitivity of 58% and a specificity of 74% which is nearly similar to a study conducted by Tian et. al[112], in their study of APACHE II's predictive accuracy for critically ill patient mortality, the test with a cut-off value of 17 is the most effective for predicting ICU patient outcomes which is nearly correlated with our study.

The area under the ROC curve (AUC) was 0.688 (95% CI: 0.541–0.834, p = 0.024), indicating a moderate discriminatory ability of the APACHE II score in distinguishing survivors from non-survivors. Patients with APACHE II ≤23 had a mortality rate of 42.1%. Patients with APACHE II >23 had a mortality rate of 57.9%. In contrast, patients with APACHE II >23 had lower mortality, which could be attributed to a different illness trajectory or better response to interventions.

In their research study, Hosseini et. al[113], revealed that APACHE II score had strong predictive accuracy for predicting outcomes in surgical and medical ICUs.

The modified APACHE II score demonstrated a strong ability to discriminate between survivors and non-survivors, as evidenced by an area under the ROC curve (AUC) of 0.841 (p = 0.001). This high AUC, with a 95% confidence interval ranging from 0.715 to 0.965, suggests that the score is a reliable tool in predicting mortality risk among non-pulmonary patients. The nonparametric assumption for the ROC analysis confirms that the model

performs significantly better than chance (with the null hypothesis set at an AUC of 0.5). The study identified a cutoff value of 18 with a sensitivity of 100%, meaning all patients who eventually succumbed to their illness were correctly identified as high risk and a specificity of 54%, indicating that nearly half of those predicted to be at high risk may not actually experience mortality. A higher sensitivity ensures that no high-risk patients are missed, which is crucial in clinical settings where early intervention can significantly impact patient outcomes. Patients with an APACHE II score ≤18 had no mortality where >18 had a mortality rate of 100%.

Artrien Adhiputri et.al[114], in their study proved that compared to SOFA and SAPS II scores, APACHE II was the most dominant predictor for mortality.

This substantial difference in mortality rates highlights the APACHE II score's clinical utility in identifying high-risk patients. The findings suggest that patients with scores above 18 require closer monitoring, aggressive intervention, and possibly a higher level of care to improve survival outcomes.

Xiao-Yu Zhou et. Al[109], in their study data suggests that APACHE II is strongly helpful for predicting mortality in patients with VAP

Naved et. al[115], and Gupta et.al[116], took APACHE II score to evaluate the condition of patient at admission and they found that patients with high scores had higher mortality rate thus supporting our study

PREDICTIVE VALUE OF SOFA SCORE IN PULMONARY AND NON-PULMONARY INDICATIONS FOR MV

The AUROC was **0.626** (95% CI: 0.456–0.795, p = 0.13). This suggests that the modified SOFA score has moderate discriminative ability in predicting mortality. However, the lack of statistical significance (p > 0.05) suggests that the model may not be a strong predictor on its own. At the established cutoff of **≥8**, the sensitivity and specificity were **53%** and **80%**,

respectively. Among patients with a **SOFA score ≤8**, 47.4% (9/19) of those who died and 80% (28/35) of those who improved fell into this category, suggesting that a lower SOFA score was more commonly associated with survival

The present study evaluates the predictive ability of the modified Sequential Organ Failure Assessment (SOFA) score in determining mortality among non-pulmonary patients. The findings suggest that the SOFA score is a valuable prognostic tool, with a high discriminatory ability, as reflected by an area under the curve (AUC) of 0.846. A critical cutoff score of **≥7** was identified for mortality prediction. At this threshold, the SOFA score demonstrated a sensitivity of **77%** and specificity of **79%**. This AUC value indicates strong predictive performance, significantly higher than the null hypothesis value of 0.5 ($p = 0.001$). Patients with a SOFA score of **≤7** had a markedly lower mortality rate (15.4%), whereas those with a score of **>7** exhibited a significantly higher mortality rate (84.6%). The association between SOFA scores and mortality was further confirmed through a Pearson Chi-Square test, which yielded a statistically significant value of $\chi^2 = 10.378$, $p = 0.001$. A cutoff of **≥7** is indicative of a high risk of mortality, reinforcing the importance of early identification and intervention in critically ill patients.

Indriasari et.al[117] , in a single centre study analyzed that a SOFA score of 10-12 has a mortality rate of 88.5%, while a SOFA score of 15-24 shows a mortality rate of 100%. The analysis results show that the higher the SOFA score, the higher the mortality rate.

In their research, Hosseini et. al[113], demonstrated that although both the SOFA and APACHE II scores had strong predictive accuracy for outcomes in surgical and medical ICUs, the SOFA is the preferred option due to its ease of use.

LIMITATIONS

The study included a relatively small sample size of 126 patients, which may limit the generalizability of the findings. A larger data could provide more robust conclusions and reduce the impact of outliers or anomalies.

Many patients were excluded from the study due to financial issues which further caused reduced sample size to predict the outcome

All comorbidities have not been known or recorded when patients are assessed on admission using scores, so there may be data on acute diseases which develops after admission or immune disorders that have not been taken into account in the assessment.

Despite the statistical insignificance of few compared data monitoring these changes over time may be crucial for assessing respiratory health and treatment efficacy in these groups.

CONCLUSION

In this study, the mean age in pulmonary group is 58 ± 16.4 and in Non-pulmonary group is 49.5 ± 18.03 respectively and male predominance was dominated in our study.

Out of 254 Mechanically ventilated patients, the incidence of Ventilator associated pneumonia in pulmonary group is 47% in pulmonary group and 52.5% in non-pulmonary group.

Patients who didn't develop VAP were not included in the study. VAP is diagnosed based on new infiltrates on Chest X-ray after 48 hours of mechanical ventilation and positive ET/Tracheostomy secretion culture. Among 126 VAP patients (63 in each group) COPD patients in pulmonary group and traumatic brain injury (TBI) patients in non-pulmonary group patients have a higher rate of VAP development.

Among the pulmonary cases, the three most frequently isolated pathogens were the gram-negative organisms which are *Acinetobacter baumannii Complex, Klebsiella pneumoniae,* and *Pseudomonas aeruginosa* and the most common gram-positive organism is *Staphylococcus aureus.*

Other notable organisms included *Serratia marcescens, Staphylococcus aureus and Escherichia coli, Klebsiella pneumoniae* (MDRO) and *Streptococcus pneumoniae* were detected. Less common organisms such as *Klebsiella oxytoca* and *Klebsiella aerogenes, Enterobacter cloacae complex, Enterobacter aerogenes,* and *MRSA Staphylococcus aureus* were also identified.

In the non-pulmonary group, *Acinetobacter baumannii Complex* was again the most prevalent pathogen, followed closely by *Klebsiella pneumoniae* and *Klebsiella pneumoniae* (MDRO) which are the most common gram-negative organisms. and most common gram-positive organism is again the *Staphylococcus aureus.*

Other less common organisms included *Pseudomonas aeruginosa* (MDR) and *Acinetobacter*

spp. as well as *Enterobacter aerogenes*, *Escherichia coli* (CRE), and *Citrobacter freundii*. Serratia marcescens is only seen in the pulmonary group in our study which is statistically significant and correlated with other studies.

Most of the organisms in pulmonary group are resistant to Carbapenems > Ceftriaxone > Cefuroxime > Piperacillin/Tazobactam > Ciprofloxacin = Amikacin > Cefoperazone/Sulbactam.

Most of the organisms in Non-pulmonary group are resistant to Fluoroquinolones > Piperacillin/ Tazobactam > Ceftriaxone > Carbapenems (Meropenem > Imipenem) > Amoxicillin/Clavulunic acid > Aminoglycosides > Cefoperazone/Sulbactam

Most of the organisms are sensitive to Tigecycline followed by Trimethoprim/Sulfamethoxazole, Cefoperozone/sulbactam and Aminoglycosides in both the groups.

A total of 35 patients are excluded from our study due to various reasons like Discharge against medical advice (DAMA) due to financial issues, family issues and referral to higher centres. Out of 91 patients, overall mortality rate in our study in VAP patients is 25.3% and improvement is seen in 46.8% patients. Our study shows high mortality in pulmonary group 30.1% compared to Non pulmonary group. Improvement Rates were significantly higher in pulmonary cases 55.6% compared to non-pulmonary cases 38.1% which is statistically significant.

Among patients with a Modified CPIS score of **≤4**, the mortality rate was **15.8%**, whereas for those with a modified CPIS score of **>4**, the mortality rate increased to **84.2%**. This highlights the strong association between higher modified CPIS scores and adverse patient outcomes in pulmonary group. Among non-pulmonary group

Modified CPIS has moderate predictive ability for mortality in VAP.

APACHE 2 score is a moderate predictor for mortality in pulmonary cases with moderate sensitivity and specificity. Among non-pulmonary group, APACHE II score demonstrated a strong ability to discriminate between survivors and non-survivors. The study identified a cutoff value of 18 with a sensitivity of 100%, meaning all patients who eventually succumbed to their illness were correctly identified as high risk. A higher sensitivity ensures that no high-risk patients are missed, which is crucial in clinical settings where early intervention can significantly impact patient outcomes.

SOFA score is a moderate predictor for mortality in pulmonary cases with moderate sensitivity and specificity. Among non-pulmonary group, the findings in our study suggest that the SOFA score is a valuable prognostic tool, with a high discriminatory ability. A cutoff of **≥7** is indicative of a high risk of mortality, reinforcing the importance of early identification and intervention in critically ill patients.

RECCOMMENDATIONS

There are a number of measures that can help prevent ventilator-associated pneumonia and to reduce mortality and morbidity. Semiupright positioning reduces risk of aspiration and is the most effective method. NIV using continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BPAP) eliminates the need for intubation in few patients, and associated with a reduced incidence of VAP. Hand washing,

SUMMARY

In this study 126 VAP patients were randomized and allocated into two groups of Pulmonary and Non pulmonary based on the diagnosis on admission. Baseline Chest X-ray will be done immediately after Intubation or Tracheostomy and Chest X-ray after 48 hours will be repeated and compared. Any new pulmonary lesion will be considered as VAP according to the ATS/IDSA Guidelines. Course in the hospital of all patients developing VAP followed up till the discharge of the patients. ET tube secretions, Tracheostomy tube secretions will be sent for Culture and Sensitivity for isolation of organism and resistance pattern. Predictors of severity like Apache-II score, SOFA score, and CPIS will be calculated and outcomes will be analyzed and compared.

1) The mean age is 58 ± 16.4 and 49.5 ± 18.03 in pulmonary and non-pulmonary group respectively.
2) There was male predominance dominant in our study.
3) The incidence of Ventilator associated pneumonia in pulmonary group is 47% in pulmonary group and 52.5% in non-pulmonary group.
4) Among 126 VAP patients (63 in each group) COPD patients in pulmonary group and traumatic brain injury (TBI) patients in non-pulmonary group patients have a higher rate of VAP development.
5) Among the pulmonary cases, the three most frequently isolated pathogens were the gram-negative organisms which are *Acinetobacter baumannii Complex, Klebsiella*

pneumoniae, and *Pseudomonas aeruginosa* and the most common gram-positive organism is *Staphylococcus aureus.*

6) In the non-pulmonary group, *Acinetobacter baumannii Complex* was again the most prevalent pathogen, followed closely by *Klebsiella pneumoniae* and *Klebsiella pneumoniae* (MDRO) which are the most common gram-negative organisms. and most common gram-positive organism is again the *Staphylococcus aureus.*

7) Most of the organisms in pulmonary group are resistant to Carbapenems > Ceftriaxone > Cefuroxime > Piperacillin/Tazobactam > Ciprofloxacin = Amikacin > Cefoperazone/Sulbactam.

8) Most of the organisms in Non-pulmonary group are resistant to Fluoroquinolones > Piperacillin/ Tazobactam > Ceftriaxone > Carbapenems (Meropenem > Imipenem) > Amoxicillin/Clavulunic acid > Aminoglycosides > Cefoperazone/Sulbactam

9) Most of the organisms are sensitive to Tigecycline followed by Trimethoprim/Sulfamethoxazole, Cefoperozone/sulbactam and Aminoglycosides in both the groups.

10) Among patients with a Modified CPIS score of **≤4**, the mortality rate was **15.8%**, whereas for those with a modified CPIS score of **>4**, the mortality rate increased to **84.2%**. This highlights the strong association between higher modified CPIS scores and adverse patient outcomes in pulmonary group. Among non-pulmonary group Modified CPIS has moderate predictive ability for mortality in VAP.

11) APACHE 2 score is a moderate predictor for mortality in pulmonary cases with moderate sensitivity and specificity. Among non-pulmonary group, APACHE II score demonstrated a strong ability to discriminate between survivors and non-survivors. The study identified a cutoff value of 18 with a sensitivity of 100%, meaning all patients who eventually succumbed to their illness were correctly identified as high risk. A higher sensitivity ensures that no high-risk patients are missed, which is crucial

in clinical settings where early intervention can significantly impact patient outcomes.

12) SOFA score is a moderate predictor for mortality in pulmonary cases with moderate sensitivity and specificity. Among non-pulmonary group, the findings in our study suggest that the SOFA score is a valuable prognostic tool, with a high discriminatory ability. A cutoff of **≥7** is indicative of a high risk of mortality, reinforcing the importance of early identification and intervention in critically ill patients.

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ANNEXURE I

ETHICAL COMMITTEE APPROVAL LETTER

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/ 871/2022-23 1/4/2023

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: "COMPARISON OF VENTILATOR ASSOCIATED PNEUMONIA BETWEEN PULMONARY AND NON-PULMONARY INDICATIONS FOR MECHANICAL VENTILATION".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: Dr POTHIREDDY MANISHA REDDY

NAME OF THE GUIDE: Dr. KEERTIVARDHAN D KULKARNI, ASSOCIATE PROFESSOR,DEPT. OF RESPIRATORY 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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College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE II

II: PATIENT CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) S.H.R.I. B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA – 586103, KARNATAKA

Principal Investigator: Dr. Pothireddy Manisha Reddy (Department of Respiratory Medicine)

P.G. Guide: Dr. Keertivardhan D Kulkarni (Professor and HOD of Respiratory Medicine)

Co Guide: Dr. Sanjeev kumar N. Bentoor (Professor and HOD of Medicine)

B.L.D.E. (Deemed to be University)

Shri B. M . Patil Medical College, Hospital and

Research Centre, Sholapur Road,

Vijayapura- 586103

PURPOSE OF RESEARCH

I have been informed that the purpose of this study is to assess - Comparison of VAP between pulmonary and non-pulmonary indications for mechanical ventilation".

I have been explained the reason for conducting this study and selecting me/my ward as a subject for this study. I have also been given the free choice for either being included or not in the study

PROCEDURE:

I understand that I will undergo a detailed history and clinical examination and investigations.

RISKS AND DISCOMFORTS:

I understand that I/my ward may experience discomfort while doing the procedure, and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that/my wards participation in this study will help in finding out

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. 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 a code number. The code key connecting the name to numbers will be kept in a separate secure location.

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

REQUEST FOR MORE INFORMATION:

I understand that I may request more questions about the study at any time.
Dr. POTHIREDDY MANISHA REDDY is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during this study, which might influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

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

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will 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 this research, the procedures required and the possible risks andbenefits, to the best of my ability in the patient's own language.

Date:

Dr Keertivardhan D Kulkarni

(Guide)

Dr Pothireddy Manisha Reddy

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. POTHIREDDY MANISHA REDDY has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore, I agree to give my consent to participate as a subject in this research project

_____ _____

(Participant) Date

_____ _____

(Witness to sign above) Date

ANNEXURE III

PROFORMA

Name of the patient:

Age/Sex:

Address:

IP no/OP no:

DOA:

DOD:

Occupation:

Presenting Complaints:

History of Present Illness:

Past history:

Personal history:

1. Tobacco chewing:

2. Smoking:

3. Alcoholism:

Family history:

GENERAL PHYSICAL EXAMINATION:

Built Nourishment Pallor Icterus Clubbing Cyanosis Lymphadenopathy Edema

Vital parameters:

a. GCS:

b. Pulse:

c. BP:

d.spo2:

e. Respiratory rate:

f. Temperature:

SYSTEMIC EXAMINATION:

RESPIRATORY SYSTEM

ABDOMEN EXAMINATION

CARDIOVASCULAR SYSTEM

CENTRAL NERVOUS SYSTEM

DIAGNOSIS:

INDICATION FOR MECHANICAL VENTILATION:

INVESTIGATIONS:

Complete blood count:

Total Count	
Neutrophils %	
Lymphocytes %	
Monocytes %	
Eosinophils %	
Basophils %	
Hemoglobin (gm/dl)	
Platelet count (per cu.mm)	

Chest X-ray on day of intubation/tracheostomy:

Chest X-ray after 48 hours of mechanical ventilation:

Culture sensitivity of Endotracheal and tracheostomy secretions and resistance pattern:

PROGNOSTIC FACTORS:

APACHE II

TEMPERATURE	
MEAN ARTERIAL PRESSURE	
HEART RATE	
OXYGENATION	
a)If FiO2 >0.5 use A-a gradient	
b)If FiO2<0.5 use PaO2	
RESPIRATORY RATE	
ARTERIAL PH	
HC03	
K+	
Na+	
SERUM CREATININE	
HAEMATOCRIT	
TLC	
GCS (SCORE=15-GCS)	
A=Total Acute Physiology Score	
B=AGE POINTS	
CHRONIC HEALTH EVALUATION C	
TOTAL SCORE(A+B+C)	

MODIFIED CLINICAL PULMONARY INFECTION SCORE

TRACHEAL SCRETIONS	
CHEST X RAY INFILTRATES	
TEMPERATURE	
TLC	
PA02/FI02 RATIO	

SOFA SCORE

PA02/FI02 RATIO	
PLATELETS	
BILIRUBIN	
MEAN ARTERIAL PRESSURE	
GCS	
CREATININE	

DURATION OF MECHANICAL VENTILATION :

DURATION OF HOSPITAL STAY :

RE-INTUBATION:

NEED FOR TRACHEOSTOMY:

FINAL OUTCOME:

SIGNATURE:

ANNEXURE IV

THESIS MASTER CHART

Name	Age	Sex	Diagnosis at the time of admission	TOTAL WBC	CHEST XRAY ON THE DAY OF INTUBATION	CHEST XRAY AFTER 48 HOURS OF INTUBATION	ET/TRACHEOSTOMY CULTURE	Antibiotic sensitive	Antibiotic resistance	APACHE 2	APACHE 4	MODIFIED CPIS	SOFA	Complications	Outcome
LAXMAN B BIRADAR	83	M	CARCINOMA LUNG	16.28	left lower zone homogenous opacification	left lower zone homogenous opacification with left middle zone heterogenous opacities 5	Klebsiella oxytoca			23	50	6	10	NO COMPLICATIONS	referred to higher centre
hanamanth ogeppa seli	75	M	ACUTE EXACERBATION OF COPD	14.21	hyperluscent lung fields	hyperluscent lung fields with perihilar infiltrates	Acinetobacter baumannii complex	Tigecycline, Minocycline	Piperacillin/Tazobactam, Gentamicin, Amikacin, meropenem, ciprofloxacin, levofloxacin	56	55	3	3	SEPTIC SHOCK	improved
mallayya g mathpati	50	M	POST TB SEQUELAE	12.23	left upper and midzone fibrosis	left upper and midzone fibrosis	Klebsiella pneumoniae	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	21	60	7	7	NO COMPLICATIONS	death due to cp arrest
ashok mahadevappa jiti	55	M	PULMONARY TB	19.81	b/l diffuse multiple cavitations	b/l diffuse multiple cavitations with right lower lobe patchy infiltrates	Klebsiella pneumoniae	Amikacin , Gentamicin, Tigecycline	Amoxiclav, Piperacillin/Tazobactam, ceftriaxone, Meropenem, Trimethoprim/sulfamethoxazole	37	49	4	3	SEPTIC SHOCK	death due to sepsis
basangouda hachireddy	78	M	ACUTE EXACERBATION OF COPD	8.21	hyperluscent lung fields	hyperluscent lung fields with right upper zone infiltrates	Escherichia Coli	Cefoperazone/Sulbactum, Trimethoprim/sulfamethoxazole, imipenem, meropenem	Amoxiclav, Piperacillin/Tazobactam, ceftriaxone,	44	46	5	4	SEPTIC SHOCK	improved
esubai sidaray honamore	78	F	ACUTE EXACERBATION OF COPD	13.12	hyperluscent lung fields	hyperluscent lung fields with left lower zone homogenous infiltrates	Acinetobacter baumannii complex	Tigecycline, trimethoprim/sulfamethoxazole	Meropenem, Amikacin, Gentamicin, ceftriaxone, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	32	64	6	10	ARDS	damia due to financial issues
pandit zalaki	42	M	POST TB SEQUELAE	11.14	right upper zone fibrosis	right upper zone fibrosis with heterogenous infiltrates	Klebsiella pneumoniae	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	19	49	5	2	NO COMPLICATIONS	improved
sakkubai ramu jadhav	64	F	COMMUNITY ACQUIRED PNEUMONIA	13.28	multiple patchy infiltrates in b/l lower zones	multiple diffuse infiltrates in b/l lung fields	Acinetobacter baumannii complex	Cefoperazone/Sulbactum, Trimethoprim/sulfamethoxazole	Meropenem, Amikacin, Gentamicin, ceftriaxone, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Amoxiclav, Imepenem	26	65	7	8	SEPTIC SHOCK	death due to sepsis
keshav	64	M	INTERSTITIAL LUNG DISEASE	18	b/l lower zone dense reticular shadows	b/l lower zone dense reticular shadows	Serratia marcescens	trimethoprim/sulfamethoxazole, Tigecycline, amikacin	amoxicillin/clavulinic acid, cefuroxime,cefuroxime Axetil, ceftriaxone,cefoperazone/sulbactum, cefipime,ertapenem, imipenem, meropenem	35	105	9	10	ARDS	death due to cp arrest
TASLEEM	70	F	POST TB SEQUELAE	12	left upper lobefibrocavitatory lesion	left upper lobefibrocavitatory lesion with left lower zone non homogenous infiltrates	Staphylococcus aureus	ciprofloxacin, Trimethoprim/sulfamethoxazole, Cefoperazone/Sulbactum	amoxicillin/clavulinic acid, cefuroxime, cefipime,ertapenem, imipenem, meropenem	12	44	4	4	NO COMPLICATIONS	improved
BASAVARAJ SHIVAKANTAPPA MADAR	50	M	ACUTE EXACERBATION OF COPD	10	hyperluscent lung fields with left midzone infiltrates	hyperluscent lung fields with left midzone infiltrates	Escherichia Coli	Cefoperazone/sulbactum, ertapenem, imipenem, meropenem, amikacin, gentamicin, tigecycline,colistin, trimethoprim/sulfamethoxazole	amoxicillin, piperacillin/tazobactam, cefuroxime,cefuroxime Axetil, cefipimeceftriaxone	39	55	6	7	ARDS	improved
ARJUN NIDONI	85	M	ACUTE EXACERBATION OF COPD	15.18	hyperluscent lung fields with tubular heart	hyperluscent lung fields with right upper zone infiltrates	Serratia marcescens	Amikacin , Gentamicin, Tigecycline, trimethoprim/sulfamethoxazole	amoxicillin/clavulinic acid, cefuroxime,cefuroxime Axetil, ceftriaxone,cefoperazone/sulbactum, cefipime,ertapenem, imipenem, meropenem	22	62	4	4	B/L PLEURAL EFFUSION	improved
LALESAB KADARASAB NASHI	65	M	ACUTE EXACERBATION OF COPD	27.01	hyperluscent lung fields with tubular heart	B/L lower zone heterogenous infiltrates with hyperluscent lung fields with tubular heart	Escherichia Coli			18	44	5	6	SEPTIC SHOCK	death due to sepsis
13782	CHANNAPPA MANAPPA PATTAR	74	M	COMMUNITY ACQUIRED PNEUMONIA	20.87	heterogenous alveolar infiltrates	heterogenous alveolar opacifications	Staphylococcus aureus	ciprofloxacin, Trimethoprim/sulfamethoxazole, Cefoperazone/Sulbactum	amoxicillin/clavulinic acid, cefuroxime, cefipime,ertapenem, imipenem, meropenem	18	50	6	5	B/L PLEURAL EFFUSION	improved
1927	BHAGYASHREE BADIGER	25	F	COMMUNITY ACQUIRED PNEUMONIA	9.1	left lower zone illdefined homogenous opacity with air bronchogram	left upper middle and lower zone heterogenous opacifications	Serratia marcescens	trimethoprim/sulfamethoxazole, Tigecycline, amikacin	amoxicillin/clavulinic acid, cefuroxime,cefuroxime Axetil, ceftriaxone,cefoperazone/sulbactum, cefipime,ertapenem, imipenem, meropenem	20	40	7	5	NO COMPLICATIONS	improved
220201	HAJMALANG DASTAGIRISAB YARANAL	42	M	POST TB SEQUELAE	22.1	b/l upper zone fibrocavitatory lesions	b/l upper zone fibrocavitatory lesions and left lower lobe heterogenous infiltrates	Acinetobacter baumannii complex	ciprofloxacin, tigecyclin	amikacin, gentamicin, cefepime, imipenem, meropenem, piperacillin/tazobactam, ceftriaxone, trimethoprim/sulfamethoxazole	14	43	4	5	ARDS	improved
168415	ABDUL RAJA M JAMADAR	75	M	CARCINOMA LUNG	20.69	right lower zonehomogenous opacification	right lower zone homogenous opacification	Klebsiella pneumoniae	fosfomycin,tigecycline,trimethoprim/sulfamethoxazole	amoxicillin/clavulanic acid, piperacillin/tazobactam, cefuroxime, cefuroxime axetil, ceftriaxone, cefoperazone/sulbactum, cefipime, ertapenem, imipenem, meropenem, amikacin, gentamicin	15	55	3	3	ARDS	referred to higher centre
150723	GURULINGAPPA	77	M	ACUTE EXACERBATION OF ASTHMA	10.2	hyperluscent lung fields with prominent bronchovascular markings	hyperluscent lung fields with prominent bronchovascular markings and left lower lobe heterogenous infiltrates	Klebsiella pneumoniae	tigecylin	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefuroxime, Cefuroxime Axetil, Ceftriaxone, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	20	64	6	9	PULMONARY EDEMA	improved
167006	VENKAPPA NARASAGOND	60	M	INTERSTITIAL LUNG DISEASE	19.33	b/l lower zone reticular opacities	b/l lower zone reticular opacities	Klebsiella pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem,meropenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone	24	57	5	10	ARDS	death due to cp arrest
83685	TRIMURTHI MOHAN TELLE	26	M	PULMONARY TB	21.16	multiple calcifications over b/l lung fields	multiple calcifications over b/l lung fields with left lower zone patchy heterogenous infiltrates	Streptococcus pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone, imipenem, meropenem	18	60	6	5	NO COMPLICATIONS	improved
152886	NALINI G KULKARNI	79	F	COMMUNITY ACQUIRED PNEUMONIA	17.94	right upper zone homogenous infiltrate	right upper zone and midzone homogenous infiltrate	Klebsiella pneumoniae	imipenem, meropenem	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Ceftriaxone, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin	30	82	6	6	ARDS	death due to cp arrest
6641	SHANTHABAI BIRADAR	65	F	ACUTE EXACERBATION OF COPD	12.86	hyperluscent lung fields with tubular heart	hyperluscent lung fields with tubular heart and right midzone	Streptococcus pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone, imipenem, meropenem	14	84	4	5	B/L PLEURAL EFFUSION	death due to cp arrest
193930	SHIVARAY AMBANNA PUJARI	45	M	PULMONARY TB	11	right lower zone homogenous infiltrate with left upper,mid and lower zone fibrocavitatory lesions	right lower zone homogenous infiltrate with left upper,mid and lower zone fibrocavitatory lesions	Klebsiella pneumoniae	Amoxicillin/Clavulanic Acid, Cefoperazone/Sulbactam, Cefepime, amikacin, gentamicin, ciprofloxacin, tigecyclin, fosfomycin,trimethoprim/sulfamethoxazole	Piperacillin/Tazobactam, Ceftriaxone, Cefuroxime, cefuroxime axetil	19	106	2	5	PULMONARY EDEMA	improved
					left upper zone homogenous	left upper zone and mid									

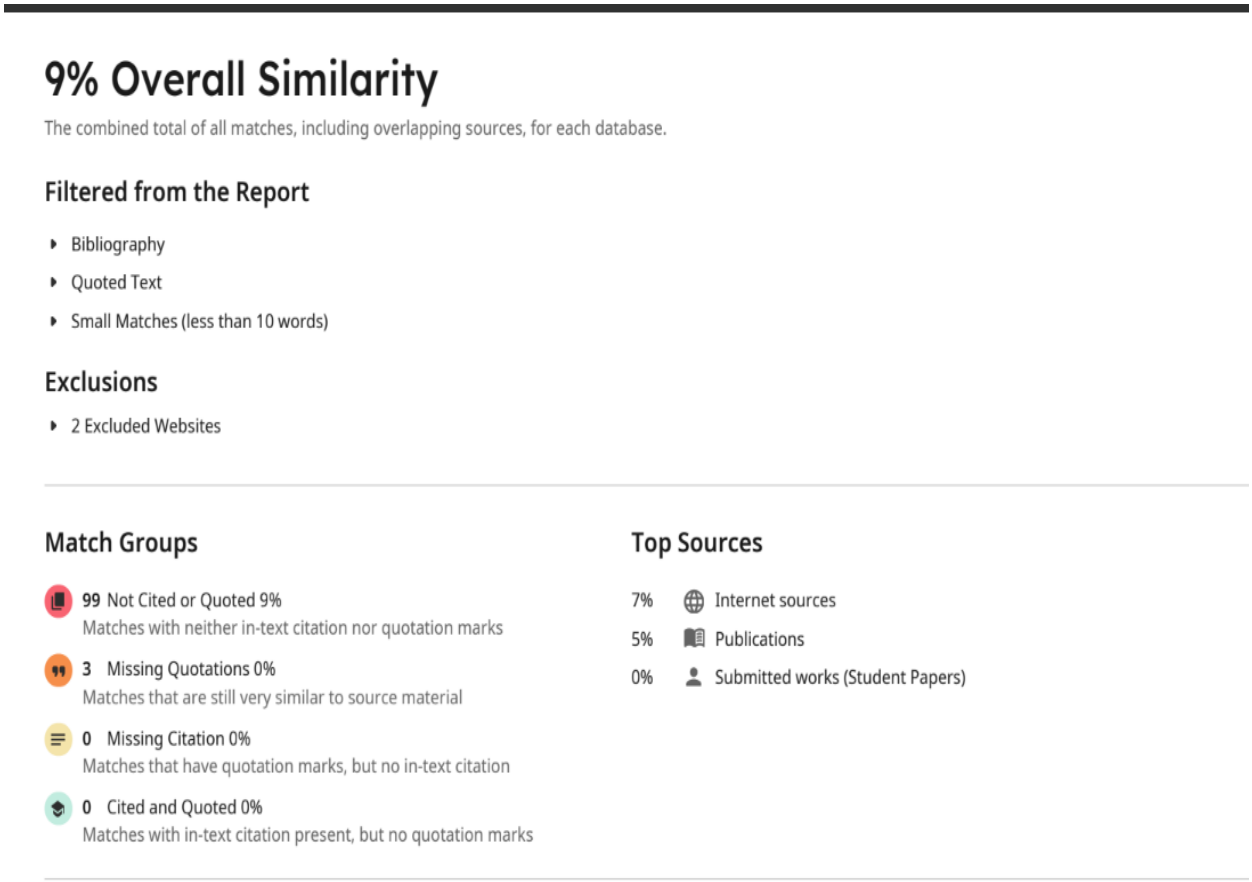
1378 2	CHANNAPPA MANAPPA PATTAR	74	M	COMMUNITY ACQUIRED PNEUMONIA	20.87	heterogenous alveolar infiltrates	heterogenous alveolar opacifications	Staphylococcus aureus	ciprofloxacin, Trimethoprim/sulfamethoxazole, Cefoperazone/Sulbactum	amoxicillin/clavulinic acid, cefuroxime, cefipime,ertapenem, imipenem, meropenem	18	50	6	5	B/L PLEURAL EFFUSION	improved
1927	BHAGYASHREE BADIGER	25	F	COMMUNITY ACQUIRED PNEUMONIA	9.1	left lower zone illdefined homogenous opacity with air bronchogram	left upper middle and lower zone heterogenous opacifications	Serratia marcescens	trimethoprim/sulfamethoxazole, Tigecycline, amikacin	amoxicillin/clavulinic acid, cefuroxime,cefuroxime Axetil, ceftriaxone,cefoperazone/sulbactum, cefipime,ertapenem, imipenem, meropenem	20	40	7	5	NO COMPLICATIONS	improved
2202 01	HAJMALANG DASTAGIRISAB YARANAL	42	M	POST TB SEQUELAE	22.1	b/l upper zone fibrocavitatory lesions	b/l upper zone fibrocavitatory lesions and left lower lobe heterogenous infiltrates	Acinetobacter baumannii complex	ciprofloxacin, tigecyclin	amikacin, gentamicin, cefepime, imipenem, meropenem, piperacillin/tazobactam, ceftriaxone, trimethoprim/sulfamethoxazole	14	42	4	5	ARDS	improved
1684 15	ABDUL RAJA M JAMADAR	75	M	CARCINOMA LUNG	20.69	right lower zonehomogenous opacification	right lower zone homogenous opacification	Klebsiella pneumoniae	fosfomycin,tigecycline,trimethoprim/sulfamethoxazole	amoxicillin/clavulanic acid, piperacillin/tazobactam, cefuroxime, cefuroxime axetil, ceftriaxone, cefoperazone/sulbactum, cefipime, ertapenem, imipenem, meropenem, amikacin, gentamicin	15	55	3	3	ARDS	referred to higher centre
1507 23	GURULINGAPPA	77	M	ACUTE EXACERBATION OF ASTHMA	10.2	hyperluscent lung fields with prominent bronchovascular markings	hyperluscent lung fields with prominent bronchovascular markings and left lower lobe heterogenous infiltrates	Klebsiella pneumoniae	tigecyclin	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefuroxime, Cefuroxime Axetil, Ceftriaxone, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	20	64	6	9	PULMONARY EDEMA	improved
1670 06	VENKAPPA NARASAGOND	60	M	INTERSTITIAL LUNG DISEASE	19.33	b/l lower zone reticular opacities	b/l lower zone reticular opacities	Klebsiella pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem,meropenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone	24	57	5	10	ARDS	death due to cp arrest
8368 5	TRIMURTHI MOHAN TELLE	26	M	PULMONARY TB	21.16	multiple calcifications over b/l lung fields	multiple calcifications over b/l lung fields with left lower zone patchy heterogenous infiltrates	Streptococcus pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone, imipenem, meropenem	18	40	6	5	NO COMPLICATIONS	improved
1528 86	NALINI G KULKARNI	79	F	COMMUNITY ACQUIRED PNEUMONIA	17.94	right upper zone homogenous infiltrate	right upper zone and midzone homogenous infiltrate	Klebsiella pneumoniae	imipenem, meropenem	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Ceftriaxone, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin	30	82	6	6	ARDS	death due to cp arrest
664	SHANTHABAI BIRADAR	65	F	ACUTE EXACERBATION OF COPD	12.86	hyperluscent lung fields with tubular heart	hyperluscent lung fields with tubular heart and right midzone	Streptococcus pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone, imipenem, meropenem	14	84	4	5	B/L PLEURAL EFFUSION	death due to cp arrest
1939 30	SHIVARAY AMBANNA PUJARI	45	M	PULMONARY TB	11	right lower zone homogenous infiltrate with left upper,mid and lower zone fibrocavitatory lesions	right lower zone homogenous infiltrate with left upper,mid and lower zone fibrocavitatory lesions	Klebsiella pneumoniae	Amoxicillin/Clavulanic Acid, Cefoperazone/Sulbactam, Cefepime, amikacin, gentamicin, ciprofloxacin, tigecyclin, fosfomycin,trimethoprim/sulfamethoxazole	Piperacillin/Tazobactam, Ceftriaxone, Cefuroxime, cefuroxime axetil	19	106	2	5	PULMONARY EDEMA	improved
						left upper zone homogenous	left upper zone and mid										
1378 2	CHANNAPPA MANAPPA PATTAR	74	M	COMMUNITY ACQUIRED PNEUMONIA	20.87	heterogenous alveolar infiltrates	heterogenous alveolar opacifications	Staphylococcus aureus	ciprofloxacin, Trimethoprim/sulfamethoxazole, Cefoperazone/Sulbactum	amoxicillin/clavulinic acid, cefuroxime, cefipime,ertapenem, imipenem, meropenem	18	50	6	5	B/L PLEURAL EFFUSION	improved
1927	BHAGYASHREE BADIGER	25	F	COMMUNITY ACQUIRED PNEUMONIA	9.1	left lower zone illdefined homogenous opacity with air bronchogram	left upper middle and lower zone heterogenous opacifications	Serratia marcescens	trimethoprim/sulfamethoxazole, Tigecycline, amikacin	amoxicillin/clavulinic acid, cefuroxime,cefuroxime Axetil, ceftriaxone,cefoperazone/sulbactum, cefipime,ertapenem, imipenem, meropenem	20	40	7	5	NO COMPLICATIONS	improved
2202 01	HAJMALANG DASTAGIRISAB YARANAL	42	M	POST TB SEQUELAE	22.1	b/l upper zone fibrocavitatory lesions	b/l upper zone fibrocavitatory lesions and left lower lobe heterogenous infiltrates	Acinetobacter baumannii complex	ciprofloxacin, tigecyclin	amikacin, gentamicin, cefepime, imipenem, meropenem, piperacillin/tazobactam, ceftriaxone, trimethoprim/sulfamethoxazole	14	42	4	5	ARDS	improved
1684 15	ABDUL RAJA M JAMADAR	75	M	CARCINOMA LUNG	20.69	right lower zonehomogenous opacification	right lower zone homogenous opacification	Klebsiella pneumoniae	fosfomycin,tigecycline,trimethoprim/sulfamethoxazole	amoxicillin/clavulanic acid, piperacillin/tazobactam, cefuroxime, cefuroxime axetil, ceftriaxone, cefoperazone/sulbactum, cefipime, ertapenem, imipenem, meropenem, amikacin, gentamicin	15	55	3	3	ARDS	referred to higher centre
1507 23	GURULINGAPPA	77	M	ACUTE EXACERBATION OF ASTHMA	10.2	hyperluscent lung fields with prominent bronchovascular markings	hyperluscent lung fields with prominent bronchovascular markings and left lower lobe heterogenous infiltrates	Klebsiella pneumoniae	tigecyclin	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefuroxime, Cefuroxime Axetil, Ceftriaxone, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	20	64	6	9	PULMONARY EDEMA	improved
1670 06	VENKAPPA NARASAGOND	60	M	INTERSTITIAL LUNG DISEASE	19.33	b/l lower zone reticular opacities	b/l lower zone reticular opacities	Klebsiella pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem,meropenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone	24	57	5	10	ARDS	death due to cp arrest
8368 5	TRIMURTHI MOHAN TELLE	26	M	PULMONARY TB	21.16	multiple calcifications over b/l lung fields	multiple calcifications over b/l lung fields with left lower zone patchy heterogenous infiltrates	Streptococcus pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone, imipenem, meropenem	18	40	6	5	NO COMPLICATIONS	improved
1528 86	NALINI G KULKARNI	79	F	COMMUNITY ACQUIRED PNEUMONIA	17.94	right upper zone homogenous infiltrate	right upper zone and midzone homogenous infiltrate	Klebsiella pneumoniae	imipenem, meropenem	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Ceftriaxone, Cefoperazone/Sulbactam, Cefepime, ertapenem, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin	30	82	6	6	ARDS	death due to cp arrest
	SHANTHABAI BIRADAR	65	F	ACUTE EXACERBATION OF COPD	12.86	hyperluscent lung fields with tubular heart	hyperluscent lung fields with tubular heart and right midzone	Streptococcus pneumoniae	Amoxicillin/Clavulanic Acid, Piperacillin/Tazobactam, Cefoperazone/Sulbactam, Cefepime, ertapenem, amikacin, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole	Cefuroxime, Cefuroxime Axetil, Ceftriaxone, imipenem, meropenem	14	84	4	5	B/L PLEURAL EFFUSION	death due to cp arrest
1939 30	SHIVARAY AMBANNA PUJARI	45	M	PULMONARY TB	11	right lower zone homogenous infiltrate with left upper,mid and lower zone fibrocavitatory lesions	right lower zone homogenous infiltrate with left upper,mid and lower zone fibrocavitatory lesions	Klebsiella pneumoniae	Amoxicillin/Clavulanic Acid, Cefoperazone/Sulbactam, Cefepime, amikacin, gentamicin, ciprofloxacin, tigecyclin, fosfomycin,trimethoprim/sulfamethoxazole	Piperacillin/Tazobactam, Ceftriaxone, Cefuroxime, cefuroxime axetil	19	106	2	5	PULMONARY EDEMA	improved
						left upper zone homogenous	left upper zone and mid										

1436 24	raju k nadaf	55	M	SEPSIS	type 1 respiratory failure	b/l diffuse patchy infiltrates	right lower zone heterogenous infiltrates	Klebsiella pneumoniae	Tigecycline, Gentamicin, trimethoprim/sulfamethoxazole	Piperacillin/Tazobactam, Amikacin, meropenem, ciprofloxacin, levofloxacin, Amoxiclav, Ceftriaxone	35	80	5	5	SEPTIC SHOCK	death due to sepsis
1492 40	umar faruk adavani	42	M	STROKE	low gcs	normal	b/l perihilar infiltrates	Klebsiella pneumoniae	Tigecycline, Cefoperazone/Sulbactum	Piperacillin/Tazobactam, Amikacin, meropenem, ciprofloxacin, levofloxacin, Amoxiclav, Ceftriaxone	22	50	7	4	SEVERE METABOLIC ACIDOSIS	damma due to financial issues
1530 28	prashant chandrakanth dalawai	30	M	CLD	airway protection	left pleural effusion	left gross pleural effusion with mid zone infiltrates	Acinetobacter baumannii (MDR)	Tigecycline, Cefoperazone/Sulbactum	Piperacillin/Tazobactam, Amikacin, meropenem, ciprofloxacin, levofloxacin, Ceftriaxone, Trimethoprim/sulfamethoxazole	18	34	6	6	B/L PLEURAL EFFUSION	improved
1555 57	basavaraj shivakantappa madar	51	M	SEPSIS	type 1 respiratory failure	b/l moderate pleural effusion	right moderate with left mild pleural effusion	Escherichia coli	Tigecycline, Meropenem, Amikacin, Imepenem, Trimethoprim/sulfamethoxazole, Gentamicin, cefoperazone/sulbactum	Piperacillin/Tazobactam, Amoxiclav, cetriaxone, ciprofloxacin	26	110	7	12	SEPTIC SHOCK	death due to sepsis
1543 43	jetteppa d kachakanur	36	M	OP POISONING	type 1 respiratory failure	normal	left lower zone infiltrates	Acinetobacter baumannii (MDR)	Trimethoprim/sulfamethoxazole	Piperacillin/Tazobactam, Ceftriaxone, Cefoperazone/sulbactum, Meropenem, Amikacin	21	56	5	5		improved
1543 47	prakash bhimanna badiger	27	M	IHD	type 1 respiratory failure	b/l perihilar infiltrates	b/l perihilar infiltrates	Pseudomonas aeruginosa	Piperacillin/Tazobactam, Meropenem, imipenum, Cefoperazone/Sulbactum,		17	34	3	3	ALTERED SENSORIUM	damma due to family issues
1673 74	sadappa basappa badiger	55	M	TRAUMATIC BRAIN INJURY	airway protection	hyperinflated lung fields	hyperinflated lung fields with right upper zone infiltrates	Klebsiella pneumoniae	Amikacin , Gentamicin, Tigecycline	Amoxiclav, Piperacillin/Tazobactam, ceftriaxone, Meropenem, Trimethoprim/sulfamethoxazole	16	48	6	5	SEPTIC SHOCK	damma due to financial issues
1742 90	prabhugouda sharanagouda biradar	28	M	BURNS	low gcs	normal	right lower zone heterogenous infiltrates	Pseudomonas aeruginosa			21	50	4	4	B/L PLEURAL EFFUSION	improved
1755 52	bhimappa basappa chigari	50	M	NEUROTOXIC SNAKE BITE	airway protection	normal	left lower zone infiltrates	Staphylococcus aureus (MRSA)			13	46	3	3	B/L PLEURAL EFFUSION	improved
2174 35	SANGANABASAVA GUJJAR	70	M	TRAUMATIC BRAIN INJURY	airway protection	normal	right upper zone infiltrates	Klebsiella pneumoniae	Tigecycline, Gentamicin, trimethoprim/sulfamethoxazole	Piperacillin/Tazobactam, Amikacin, meropenem, ciprofloxacin, levofloxacin, Amoxiclav, Ceftriaxone	32	87	7	12	SEPTIC SHOCK	damma due to family issues
3195 07	PRAMILA	50	F	CKD	airway protection	normal	right upper zone infiltrates	Staphylococcus aureus	Trimethoprim/sulfamethoxazole, Tigecycline, ciprofloxacin, levofloxacin	Clindamycin, Benzyl penicillin, Erythromycin, Nitrofurantoin	22	64	9	14	SEPTIC SHOCK	damma due to family issues
1939 30	shivaray ambanna pujari	45	M	NEUROTOXIC SNAKE BITE	airway protection	normal	left midzone infiltrates	Klebsiella pneumoniae	Amikacin, Gentamicin, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Tigecycline, Cefoperazone/Sulbactum, Amoxiclav	Piperacillin/Tazobactam, Meropenem, ceftriaxone, Imepenem	18	57	7	7	ALTERED SENSORIUM	improved
1165 34	AISHWARYA ANAND BADIGER	19	F	BURNS	airway protection	normal	b/l perihilar infiltrates	Escherichia coli (CRE)	Tigecycline	Piperacillin/Tazobactam, Amikacin, meropenem, ciprofloxacin, levofloxacin, Ceftriaxone, Trimethoprim/sulfamethoxazole, Imepenem	24	77	3	6	SEPTIC SHOCK	death due to sepsis
1967 03	shankreppa irasangappa reshami	85	M	TRAUMATIC BRAIN INJURY	airway protection	hyperinflated lung fields	hyperinflated lung fields with left lower zone infiltrates	Acinetobacter baumannii complex	tigecycline	Meropenem, clindamycin, Erythromycin, meropenem, Imepenem, Nitrofurantoin	27	80	6	8	B/L PLEURAL EFFUSION	damma due to financial issues
1978 92	dilip shivappa haladakeri	43	M	CLD	airway protection	normal	b/l moderate pleural effusion with right mid zone infiltrates	Staphylococcus aureus (MRSA)	Trimethoprim/sulfamethoxazole, Tigecycline, Tetracyclin, Vancomycin, Linezolid	levofloxacin, Ciprofloxacin, Erythromycin	22	89	8	12	SEPTIC SHOCK	death due to sepsis

1978 92	dilip shivappa haladakeri	43	M	CLD	airway protection	normal	b/l moderate pleural effusion with right mid zone infiltrates	Staphylococcus aureus (MRSA)	Trimethoprim/sulfamethoxazole, Tigecycline, Tetracyclin, Vancomycin, Linezolid	levofloxacin, Ciprofloxacin, Erythromycin	22	89	8	12	SEPTIC SHOCK	death due to sepsis
3252 55	mallu gurappa nagaral	30	M	SEPSIS	airway protection	normal	left lower zone infiltrates	Klebsiella pneumoniae	Amikacin, Gentamicin, Trimethoprim/sulfamethoxazole, Tigecycline	Meropenem, ceftriaxone, ciprofloxacin, levofloxacin , Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	32	102	6	10		referred to higher centre
3478 5	irappa gurappa belavadaggi	22	M	OP POISONING	airway protection	normal	left moderate pleural effusion	Pseudomonas aeruginosa	Ciprofloxacin, Amikacin	Meropenem, levofloxacin, Cefoperazone/Sulbactum	19	52	4	4		improved
8368 5	trimurti mohan tele	26	M	TRAUMATIC BRAIN INJURY	airway protection	normal	right upper zone infiltrates	Klebsiella pneumoniae ssp pneumoniae (MDRO)	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	14	56	6	6		improved
8804 5	musthafa m mujawar	60	M	CKD	airway protection	normal	left midzone infiltrates	Klebsiella pneumoniae	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	30	69	6	8	SEPTIC SHOCK	death due to sepsis
1122 34	laxmibai ganapati durve	65	F	TRAUMATIC BRAIN INJURY	type 2 respiratory failure	normal	right mid zone perihilar infiltrates	Acinetobacter baumannii complex	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	12	46	4	4	B/L PLEURAL EFFUSION	dama due to family issues
1321 61	salman raza	33	M	SEPSIS	airway protection	normal	right lower zone heterogenous infiltrates	Klebsiella oxytoca	tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	17	54	5	5		improved
1321 71	harish s teggelli	38	M	TRAUMATIC BRAIN INJURY	airway protection	normal	left midzone infiltrates	Acinetobacter baumannii complex	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	20	59	5	10	DEMENTIA	improved
1344 89	basanna ningayya guruvin	63	M	SEPSIS	type 1 respiratory failure	normal	left upper zone homogenous infiltrate	Acinetobacter baumannii complex	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	15	67	7	14	SEPTIC SHOCK	dama due to financial issues
1488 87	asma m antaraganji	19	F	SEPSIS	type 1 respiratory failure	normal	right mid zone perihilar infiltrates	Staphylococcus aureus (MRSA)	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem, Tigecycline	ciprofloxacin, levofloxacin	17	59	4	4	SEPTIC SHOCK	dama due to financial issues
1507 23	gurulingappa s biradar	77	M	MENINGOENCEPHALITIS	airway protection	normal	right mid zone perihilar infiltrates	Klebsiella pneumoniae ssp pneumoniae (MDRO)	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	23	76	8	12	SEPTIC SHOCK	dama due to family issues
1684 15	abdul rajak m jamadar	75	M	PARKINSON DISEASE	airway protection	left moderate pleural effusion	left moderate pleural effusion	Klebsiella pneumoniae	Tigecycline, Amikacin, Gentamicin, cefoperazone/sulbactum	Meropenem, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Amoxiclav, Imepenem	12	54	5	5	B/L PLEURAL EFFUSION	improved
1670 06	venkappa narasagond	60	M	BURNS	airway protection	normal	right lower zone heterogenous infiltrates	Klebsiella pneumoniae	Meropenem, Amikacin, Gentamicin, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem, Tigecycline	Ceftriaxone	19	60	9	12	B/L PLEURAL EFFUSION	dama due to family issues
1783 73	gurappa sangappa metri	42	M	TRAUMATIC BRAIN INJURY	airway protection	left sided blunting of costophrenic angle with 4,5,6 rib fractures with icd insitu	left sided blunting of costophrenic angle with 4,5,6 rib fractures	Klebsiella oxytoca	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	13	54	5	5	PULMONARY EDEMA	improved
1869 00	ravi mahadevappa kolar	25	M	TRAUMATIC BRAIN INJURY	airway protection	normal	right lower zone heterogenous infiltrates	Klebsiella pneumoniae ssp pneumoniae (MDRO)	Amikacin, Trimethoprim/sulfamethoxazole, Meropenem, Tigecycline	Gentamicin, ceftriaxone, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	18	55	9	6	PULMONARY EDEMA	dama due to financial issues
1905 91	basavaraj h kunjoti	40	M	TRAUMATIC BRAIN INJURY	airway protection	normal	right mid zone perihilar infiltrates	Klebsiella pneumoniae ssp pneumoniae (MDRO)		Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem, Tigecycline	24	44	6	5		improved
1949 70	mahadevi madar	40	F	STROKE	airway protection	normal	left lower lobe infiltrates	Pseudomonas aeruginosa	Imepenem, meropenem, Amikacin, Cefsperazone/Sulbactum	Piperacillin/Tazobactam, Ciprofloxacin, Levofloxacin	32	68	3	7	SEVERE METABOLIC ACIDOSIS	death due to sepsis
2128 87	sangappa ramappa lalawar	40	M	TRAUMATIC BRAIN INJURY	airway protection	normal	right lower zone heterogenous infiltrates	Enterobacter aerogenes	Tigecycline, trimethoprim/sulfamethoxazole	Meropenem, Amikacin, Gentamicin, ceftriaxone, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Amoxiclav, Imepenem,Cefoperazone/Sulbactum	25	51	7	4	PULMONARY EDEMA	improved
2143 65	adappa amrappa ganganagoudar	73	M	STROKE	airway protection	hyperinflated lungs	right lower zone heterogenous infiltrates	Acinetobacter baumannii complex	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	16	46	4	4		improved
2135 16	gurappa ramappa chalawadi	66	M	STROKE	airway protection	normal	right mid zone perihilar infiltrates	Acinetobacter baumannii complex	Tigecycline	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem	19	79	6	8	ARDS	death due to sepsis
1083 61	iranagouda sahebgouda patil	54	M	TRAUMATIC BRAIN INJURY	airway protection	prominent bronchovascular markings	prominent bronchovascular markings perihilar infiltrates	Staphylococcus aureus (MRSA)	Meropenem, Amikacin, Gentamicin, ceftriaxone, Trimethoprim/sulfamethoxazole, Piperacillin/Tazobactam, Cefoperazone/Sulbactum, Amoxiclav, Imepenem, Tigecycline	ciprofloxacin, levofloxacin	17	57	5	5	SEPTIC SHOCK	improved

ANNEXURE V

PLAGIARISM REPORT



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