"CLINICAL PROFILE, RISK FACTORS AND RESISTANCEPATTERN OF PATIENTS WITH VENTILATOR ASSOCIATED PNEUMONIA DUE TO ACINETOBACTER BAUMANNII IN INTENSIVE CARE UNITS - A ONE-YEAR CROSS-SECTIONAL STUDY IN KLE'S DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI."

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With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL PROFILE, RISK FACTORS AND RESISTANCE PATTERN OF PATIENTS WITH VENTILATOR ASSOCIATED PNEUMONIA DUE TO ACINETOBACTER BAUMANNII IN INTENSIVE CARE UNITS.",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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LIST OF ABBREVIATION

VAP - ventilator-associated pneumonia

ICU - intensive care unit

MV – mechanical ventilation

MDR – multidrug resistant

INICC - International Nosocomial Infection Control Consortium

LOS – length of stay

ARDS – acute respiratory distress syndrome

COPD – chronic obstructive lung disease

ETT – endotracheal tube

MRSA - methicillin-resistant Staphylococcus aureus

MDRO - multidrug-resistant organisms

ESBL - extended-spectrum beta-lactamase

CRE - carbapenem-resistant Enterobacteriaceae

TBI - traumatic brain injury

BAL - bronchoalveolar lavage

PSB-protected specimen brush

ETA - endotracheal aspirates

PCR - polymerase chain reaction

CRP - C-reactive protein

CPIS - Clinical Pulmonary Infection Score

CT - computed tomography

PUD – Peptic Ulcer Disease

DVT- Deep Venous Thrombosis

CVA - cerebrovascular accident

RTA - Road traffic accidents

IHD - ischemic heart disease

CVC - Central venous catheter

TT - Tracheostomy tubes

OR - odds ratio

RBC - red blood cell

HB – Hemoglobin

GCS - Glasgow Coma Scale

ABSTRACT

Introduction: Ventilator-associated pneumonia (VAP) is a severe nosocomial infection in ICU settings, especially when caused by multidrug-resistant Acinetobacter baumannii. This study aimed to evaluate the clinical profile, risk factors, resistance patterns, and prognostic indicators in patients with VAP due to A. baumannii.

Materials and Methods: A one-year cross-sectional study (January 2023 – December 2023) was conducted at KLE's Dr. Prabhakar Kore Hospital. 68 patients with culture-confirmed VAP were included. Data on demographics, clinical characteristics, and resistance patterns were collected. Prognostic scores, including Glasgow Coma Scale (GCS), APACHE II, Q-SOFA, and SAPS II, were analyzed for mortality prediction.

Results: The cohort had a mean age of 48.32 years, predominantly male (79.4%). Common diagnoses included cerebrovascular accidents (29.3%) and sepsis (17.3%). Significant risk factors were mechanical ventilation (100%), central venous catheter use (69.1%), and glucocorticoid therapy (54.4%). The mortality rate was 64.7%. Prognostic scores were higher in deceased patients, indicating high mortality risk: GCS ≤ 8 (AUC: 0.928), APACHE II > 20 (AUC: 0.954), Q-SOFA > 1 (AUC: 0.937), and SAPS II > 43 (AUC: 0.971). A. baumannii showed high pan-drug resistance (72.1%), with colistin being the most effective antibiotic (16% sensitivity).

Conclusion: The high mortality associated with VAP due to A. baumannii underscores the need for rigorous infection control, targeted antibiotic stewardship, and prognostic scoring systems. The SAPS II score, with its highest predictive accuracy, should be prioritized for identifying high-risk patients and guiding clinical decisions

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INTRODUCTION

When pneumonia develops at least 48 hours following endotracheal intubation or tracheostomy and there is no sign of pneumonia at the time of intubation, admission, or tracheostomy, it is referred to as ventilator-associated pneumonia (VAP). Intubated patients in the intensive care unit (ICU) are susceptible to a significant nosocomial infection that calls for deliberate research to lower the illness's morbidity and mortality. It is divided into early-onset and late-onset categories based on the onset. Early-onset VAP, typically brought on by antibiotic-sensitive bacteria, develops in ventilated patients during the first four days (≤4 days) of mechanical ventilation (MV). When VAP appears in patients on mechanical ventilation after five days or more, it is referred to as late-onset VAP. The cause of this type of VAP is multidrug-resistant (MDR) bacteria. [1]

The Infectious Diseases Society of America and the American Thoracic Society released clinical practice guidelines in 2016. According to these guidelines, a diagnosis of VAP is established if the patient exhibits at least two of the following clinical characteristics: a temperature (≥38 °C), raised white blood cell count (≥12*109 WBC/mL), and purulent tracheobronchial secretions. Additionally, the patient must have a new or transforming lung infiltrate evident on a chest x-ray. [2]

The most common nosocomial infection in patients receiving intensive care unit (ICU) treatment is ventilator-associated pneumonia (VAP). The prevalence of VAP in Asian nations ranges from 3.5 to 46 infections/1000 mechanical ventilator (MV) days, despite the international nosocomial infection control consortium (INICC) data suggesting that the incidence of VAP is as high as 13.6/1000 MV days. [3,4]

The length of mechanical ventilation, the length of hospital and ICU stays prior to VAPs, the timing and cumulative exposure to antimicrobials, the local ecology, and the prevalence of any potential epidemic phenomena in a particular ICU are only a few of the variables that influence the organisms linked to VAP. Pseudomonas aeruginosa, Escherichia coli, Klebsiella pneumoniae, and Acinetobacter species are common Gram-negative pathogens implicated with VAP; the predominant Gram-positive germ is Staphylococcus aureus. [5-7]

Dutch researcher Martinus Willem Beigerinck discovered acinetobacter, an obligatory aerobic nonfermenting gram-negative nonmotile bacteria, in 1911 . Acinetobacter was once thought to be a low-virulence bacteria since it was sensitive to widely used antibacterial medicines. However, starting in the 1970s, its resistance to these drugs started to rise and eventually became a significant issue, particularly in nosocomial settings. [8]

Infections caused by A. baumannii are now known to be among the most dangerous and challenging to manage in critical care environments. [9]
A. baumannii can form biofilms that help bacteria attach to tissues, different environmental surfaces, and devices. It can also quickly acquire different antibiotic resistance mechanisms. It can survive in both dry and humid environments, is resistant to disinfectants, and can be dried to the maximum extent possible. These characteristics are thought to have contributed to the fast endemic spread of A. baumannii in hospital settings and several intensive care units across the globe. [10]

Treatment for A. baumannii infections is still difficult. The choice of active antibacterial treatment is limited by high rates of native resistance and the swift emergence of acquired resistance to routinely prescribed antibiotic classes,

which jeopardizes the course of the disease and the prognosis of the patient. The "gold standard" for treating A. baumannii-caused pneumonia in the past was imipenem therapy [(11)].

Afterwards, based on the timing of VAP development and the existence of risk factors for resistant bacteria, the choice of empirical treatment was advised. Antibiotic combination therapy (antipseudomonal cephalosporin or carbapenem or ß-lactam/ß-lactamase inhibitor with antipseudomonal fluoroquinolone or aminoglycoside) was advised for patients with late-onset disease or risk factors for multidrug-resistant pathogens [(12)].

Since there were few other options available due to the development of antibacterial resistance, the old, less effective, and more toxic polymyxins and tetracyclines—for which this bacterium is still sensitive—were once again used. Even though colistin is nephrotoxic and neurotoxic, its limited ability to penetrate lung tissue makes it an inadequate therapy option for ventilator-associated pneumonia. Additionally, in recent years, resistance to colistin, the last resort treatment, has grown dangerously [4]. Consequently, colistin plus carbapenem, sulbactam, or tigecycline should be utilized as an empirical combination treatment if high drug resistance of A. baumannii is common in a hospital or department.[(13)]
The delayed detection and treatment of drug-resistant A. baumannii were found to be linked with increased mortality rates and greater healthcare expenses [13].

Risk factors for VAP [1] include age, prior hospitalizations, surgical procedures, intrusive monitoring and treatment methods, and comorbidities. Risk factors for ventilator-associated pneumonia (VAP) caused by multidrug-resistant A. baumannii include hospital length of stay (LOS), prior use of antibiotics, length of mechanical ventilation (MV), severity of the disease, and

the presence of drug-resistant A. baumannii strains in community hospitals [14,15].

The objective of our study was to precisely determine the correlation between risk factors for ventilator-associated pneumonia (VAP) by A. baumannii, clinical profile of the patient and mortality, taking into account the resistance profiles of A. baumannii. Additionally, we aimed to estimate the independent predictors of death during hospitalization.

AIMS AND OBJECTIVES

- To study the clinical profile and risk factors of patients with ventilator-associated pneumonia (VAP) caused by A. Baumannii.
- To study the resistance pattern of A. Baumannii causing VAP
- To establish the link between the clinical profile, risk factors of patients and resistance pattern of A. Baumannii causing VAP.

REVIEW OF LITERATURE

Patients in the intensive care unit (ICU) face the possibility of death not only due to their critical disease but also due to secondary factors such as nosocomial infections. Pneumonia is the second most prevalent nosocomial infection in critically ill patients, impacting 27% of all such patients [16].

Ventilator-associated pneumonia (VAP) represents a category of device-associated infections that carries significant consequences, including longer hospital stays, increased treatment costs, and unfavourable patient outcomes [17]. Approximately 86% of hospital-acquired pneumonia is attributed to VAP, which is mostly connected with the use of mechanical ventilation. In the United States, the annual incidence ranges between 250,000 and 300,000 cases, resulting in a rate of 5 to 10 cases per 1,000 hospital admissions. The mortality rate linked to VAP has been recorded to range from 0 to 50% [18,19]. Similarly, a study conducted in India by Neelima Ranjan et al. (2014) found that the occurrence rate of VAP was 57.14%. Additionally, the incidence density of VAP was calculated to be 31.7 cases per 1000 ventilator days. [20]

Various studies have yielded divergent findings in determining the mortality that can be attributed to certain factors. This discrepancy can be attributed to the inclusion of distinct populations, such as patients with less severe trauma, those with acute respiratory distress syndrome (ARDS), and individuals in medical and surgical ICUs. Additionally, differences in the appropriate empirical medical treatment provided during the first two days also contribute to the variations in results. Marin H. Kollef (1995) [21]in his study states that the presence of certain organisms has a significant effect on the outcome, as VAP caused by Pseudomonas aeruginosa, Acinetobacter spp., and Stenotrophomonas maltophilia is associated with increased fatality rates. In addition to the risk of death, the economic impact of VAP includes longer hospitalizations in the ICU ranging from 4 to 13 days.

VENTILATOR-ASSOCIATED PNEUMONIA

DEFINITION

Pneumonia that develops more than 48 hours after a patient has been intubated and is receiving mechanical ventilation is known as ventilator-associated pneumonia.

EPIDEMIOLOGY

INCIDENCE

VAP risk rates are more appropriately reflected in the incidence rates that are computed with 1,000 ventilator days as the denominator. In the United States, VAP rates varied from 4–14/1000 ventilator days, but in poorer nations, they were between 10–52.7/1000 days.[22]

Ashu Sara Mathai et al. [23] in their study conducted in India stated VAP infections occurred in 95 patients (38%) overall, or 40.1 VAP infections per 1000 days of mechanical breathing.

The significant variations can be partially attributed to disparities in definitions, variations in the application of definitions, limits in diagnosing all definitions, and variances in methodologies used for microbiological samples. [24]

The daily occurrence of ventilator-associated pneumonia (VAP) reaches its highest point between days 5-9 of mechanical ventilation, whereas the overall number of cases is strongly linked to the entire length of time a patient is on mechanical ventilation. [25]

The studied population considerably affects incidence rates. In cancer patients, VAP rates can reach 24.5/1000 ventilatordays [26]. Trauma patients have a high incidence (17.8% in one cohort of 511 patients) [27], possibly due to immune function changes following significant trauma, brain injury, and lung contusion. The increased incidence in COPD patients may be due to prolonged invasive

mechanical ventilation (muscular weakness), high microaspiration and bacterial colonization (defective mucociliary clearance), and altered local and general host defence mechanisms [28]. An increased risk of VAP is connected with ARDS. ARDS patients in general have a 29% incidence and ECMO patients 35% despite lung-protective measures.(29)

PATHOGENESIS

PATHOGENESIS OF VENTILATOR-ASSOCIATED PNEUMONIA (VAP) INCLUDE:

1. **Disruption of the Body's Normal Respiratory Defenses**

Intubation plays a crucial role in the development of ventilator-associated pneumonia (VAP). According to the American Thoracic Society, intubation hinders the cough reflex, impairs the process of clearing mucus from the airways, and damages the lining of the trachea, making it easier for bacteria to grow and cause infection. Intubation and the presence of the endotracheal tube (ETT) serve as pathways for germs, allowing them direct entry into the lower respiratory system. (12)

2. **Respiratory Tract Colonization**

According to Chastre and Fagon, patients who undergo intubation frequently encounter fast colonization of the oropharynx and trachea by gram-negative bacilli and other harmful microorganisms. The bacteria in question originate from either the oropharynx or the gastrointestinal system and make their way to the lower airways through a process known as microaspiration. Their study highlighted the importance of bacterial colonization as a crucial stage in the development of ventilator-associated pneumonia (VAP). (30)

3. Biofilm Formation

Biofilm development on the surfaces of the endotracheal tube (ETT) is an additional crucial element in the pathophysiology of ventilator-associated pneumonia (VAP). Chastre and Fagon state that biofilms serve as a shield for bacteria, safeguarding them against the body's immune system and medications. This allows the bacteria to maintain a long-lasting infection. Biofilms can periodically discharge germs into the lower respiratory system, thereby sustaining the illness. The tenacity of biofilms greatly hinders the elimination of germs once colonization has taken place. [30]

4. The Role of Gastric Colonization

The process of harmful bacteria colonizing the stomach and then being inhaled into the lungs is a widely acknowledged mechanism for ventilator-associated pneumonia (VAP). In their study, Metheny et al. discovered that critically sick patients frequently experience changes in stomach pH and delayed gastric emptying. These alterations contribute to the growth of bacteria and elevate the likelihood of aspiration. Their research showed that the inhalation of stomach contents into the airways is common and strongly linked to the occurrence of ventilator-associated pneumonia (VAP). [31]

5. Impact of Ventilator Equipment

Zolfaghari and Wyncoll examined the impact of ventilator equipment design, specifically endotracheal tubes (ETTs), on the development of ventilator-associated pneumonia (VAP). Their study emphasized that conventional high-volume, low-pressure cuffed endotracheal tubes (ETTs) are susceptible to leakage and microaspiration. Recent designs, such as tubes with polyurethane cuffs, have the objective of mitigating these dangers by offering improved sealing and limiting the occurrence of microaspiration. This study emphasizes

the necessity for enhanced design of ventilator equipment to reduce the incidence of ventilator-associated pneumonia (VAP). [32]

6. Internal and External Sources

VAP is caused by both endogenous causes, such as oropharyngeal and stomach secretions, and external sources, such as contaminated ventilator circuits. Tablan et al. highlighted the significance of the ICU setting in the development of ventilator-associated pneumonia (VAP). Their guidelines on reducing healthcare-associated pneumonia have identified that the transmission of VAP pathogens is caused by contaminated breathing equipment and insufficient infection control methods. [33]

7. Factors Influencing Host and Immune Response

The susceptibility to ventilator-associated pneumonia (VAP) is greatly influenced by the host's immune response and underlying health problems. Muscedere et al. said that individuals with chronic lung illnesses, immunosuppression, or prior antibiotic exposure have a higher vulnerability to ventilator-associated pneumonia (VAP). Their research emphasized that these situations modify the microbial ecology and compromise the host's immune systems, rendering patients more susceptible to infections. [34]

8. Precautionary Measures

Implementing preventive interventions is crucial for minimizing the occurrence of ventilator-associated pneumonia (VAP). In a study conducted by Bouadma et al., the efficacy of a comprehensive program in reducing ventilator-associated pneumonia (VAP) was proven. This program implemented many interventions, including raising the head of the bed, providing daily breaks from sedation, and draining secretions from underneath the vocal cords. Hawe et al. also

emphasized that implementing guidelines actively rather than passively leads to a considerable reduction in rates of ventilator-associated pneumonia (VAP). [35, 36]

MICROBIOLOGY

Over time, the understanding of the microbiology of VAP has changed, since there have been changes in the frequency and resistance patterns of the microorganisms that cause it.

HISTORICAL TIMELINE OF VAP PATHOGENS

1. Early 1980s to 1990s

During the initial stages of VAP research, the most common disease-causing microorganisms were mainly bacteria that have a cell wall structure that stains pink when exposed to a certain laboratory dye. Chastre and Fagon (2002) [30] commonly detected Pseudomonas aeruginosa and Klebsiella pneumoniae. According to Andrews et al. (1981) [37], initial research indicated that these organisms were frequently found in intensive care unit (ICU) environments, generally associated with contaminated respiratory equipment and the weakened immune systems of patients.

2. In the middle of the 1990s

In the mid-1990s, there was a change in the types of microorganisms causing ventilator-associated pneumonia (VAP), with a rise in the occurrence of methicillin-resistant Staphylococcus aureus (MRSA). Torres et al. (1995) [38] observed an escalating occurrence of MRSA in cases of ventilator-associated pneumonia (VAP), which mirrors the overall pattern of rising antibiotic resistance in healthcare facilities.

3. The time period of the early 2000s

During the early 2000s, research studies identified many types of disease-causing organisms, such as Acinetobacter baumannii. This specific bacterium became a major contributor to ventilator-associated pneumonia (VAP), especially in patients who stayed in the intensive care unit for an extended period of time [30]. The occurrence of Acinetobacter was associated with its capacity to endure on surfaces and its resistance to several medications.

4. The period from the late 2000s to the early 2010s

During this time, there was an increased emphasis on multidrug-resistant organisms (MDROs). The CDC documented a rise in cases of ventilator-associated pneumonia (VAP) caused by carbapenem-resistant Enterobacteriaceae (CRE) and extended-spectrum beta-lactamase (ESBL)-producing organisms (Siegel et al., 2007) [39]. These findings emphasized the difficulty of treating ventilator-associated pneumonia (VAP) in the presence of increasing antibiotic resistance.

5. Current trends (2015-Present)

Recent studies consistently emphasize the prevalence of multidrug-resistant organisms (MDROs) in ventilator-associated pneumonia (VAP). Kollef et al. (2015) [40] found that gram-negative bacteria, such as Pseudomonas aeruginosa, Acinetobacter baumannii, and Klebsiella pneumoniae, are still common and a considerable number of them are resistant to several antibiotics. Furthermore, MRSA remains a significant pathogen, especially in cases related to healthcare (Kalil et al., 2016) [41].

MICROBIAL PATHOGENS AND PATTERNS OF RESISTANCE

GRAM-NEGATIVE BACILLI

Pseudomonas aeruginosa has consistently been identified as a pathogen in ventilator-associated pneumonia (VAP) because of its inherent resistance mechanisms and capacity to create biofilms [30]. The ability of Pseudomonas to withstand the effects of carbapenems and other beta-lactam antibiotics presents considerable difficulties in therapy (Falagas et al., 2005) [42]. Furthermore, Klebsiella pneumoniae and Escherichia coli, which frequently carry ESBL genes, make treatment choices more difficult (Harris et al., 2013) [43].

1. Acinetobacter baumannii

Acinetobacter baumannii has become a powerful and challenging disease-causing microorganism, especially in intensive care unit (ICU) environments. The quick acquisition of resistance genes and its capacity to remain in the hospital environment contributes to the high incidence of this pathogen in ventilator-associated pneumonia (VAP) [44]. The appearance of carbapenem-resistant Acinetobacter strains has added complexity to the tactics used for managing these bacteria [46]. A study conducted by Peleg et al. (2008) [45] emphasized the capacity of Acinetobacter baumannii to flourish in the intensive care unit (ICU) setting, frequently colonizing equipment and surfaces, thereby enabling its transmission. Munoz-Price and Weinstein (2008) [44] observed that the organism tends to develop resistance to a broad range of antibiotics, including carbapenems, which are often used as a last resort in treatment.

Maragakis and Perl (2008) [46] conducted recent investigations that documented outbreaks of Acinetobacter baumannii in intensive care unit (ICU) settings. They found a connection between these outbreaks with the presence of contaminated medical devices and the hands of healthcare personnel. Viehman

et al. (2014) [47]conducted a study to investigate the clinical consequences of Acinetobacter, highlighting the challenges in eliminating this harmful microorganism after it has established itself in a healthcare environment. Researchers discovered that patients who had ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii experienced elevated rates of illness, longer hospitalizations in the intensive care unit (ICU), and higher healthcare expenses in comparison to those with VAP caused by different infections.

Pathogenicity and Virulence Factors:

The virulence of Acinetobacter baumannii is ascribed to multiple variables, such as surface-associated motility, biofilm development, and the capacity to get iron in situations with restricted iron availability. These factors augment the bacterium's capacity to establish colonies and induce infections in hosts (Harding et al., 2018) [48].

1. **Biofilm production:** Biofilm production is an essential mechanism for the development of pathogenicity. Biofilms provide a protective barrier for bacteria against environmental stressors, such as antibiotics and the immunological response of the host. The capacity to adhere and aggregate is facilitated by the formation of extracellular polymeric molecules and surface proteins (Harding et al., 2018) [48].

2. **Iron Acquisition:** Iron is required for bacterial proliferation and metabolic processes. Acinetobacter baumannii has evolved intricate mechanisms for obtaining iron, such as siderophores and iron-regulated outer membrane proteins, in order to survive in environments with low iron, such as the host [48].

3. **Secretion Systems:** The Type VI secretion system (T6SS) is an additional component that contributes to the pathogenicity of A.

baumannii. The Type VI Secretion System (T6SS) delivers toxic effectors into both competing bacteria and eukaryotic cells, hence enhancing bacterial survival and pathogenicity (Weber et al., 2016) [49].

Mechanisms of Antibiotic Resistance:

Acinetobacter baumannii demonstrates resistance to many antibiotics by diverse mechanisms, such as the synthesis of beta-lactamases, efflux pumps, and alterations of target sites. The presence of this resistance hampers the available treatment choices and adds complexity to the management of infections (Doi et al., 2019) [50].

1. **Beta-lactamases:** Beta-lactamases, such as carbapenemases, are enzymes secreted by Acinetobacter baumannii that break down beta-lactam antibiotics, making them useless. The existence of genes that encode these enzymes, such as bla_OXA-23 and bla_NDM-1, significantly contributes to resistance (Gordon & Wareham, 2010) [51].

2. **Efflux pumps:** Efflux pumps, such as the AdeABC system, remove various antibiotics from bacterial cells, reducing the concentration of antibiotics inside the cell and thereby reducing resistance. MDR strains frequently exhibit upregulation of these pumps (Coyne et al., 2011) [52].

3. **Alteration of Target Sites:** Changes in the target sites of antibiotics, such as modifications in penicillin-binding proteins (PBPs) and mutations in the regions (QRDRs) of DNA gyrase and topoisomerase IV, result in resistance to beta-lactams and quinolones, respectively (Peleg et al., 2012) [45].

ACINETOBACTER BAUMANNII IS COMMONLY LINKED TO VENTILATOR-ASSOCIATED PNEUMONIA (VAP).

1. **Role in VAP:** Acinetobacter baumannii is a primary etiological agent of ventilator-associated pneumonia (VAP) in intensive care units (ICUs). The pathogen's capacity to endure in hospital settings and create biofilms on endotracheal tubes and other medical equipment renders it a serious threat in cases of ventilator-associated pneumonia (Eveillard et al., 2014) [(53)].

2. **Clinical Impact:** Ventilator-associated pneumonia (VAP) caused by Acinetobacter baumannii is linked to significant morbidity and mortality rates, extended hospital stays, and elevated healthcare expenses. The existence of multidrug-resistant (MDR) microorganisms adds complexity to the treatment process and results in less favorable clinical results (Maragakis & Perl, 2008) [(46)].

3. **Treatment Difficulties:** Treating Acinetobacter baumannii-associated VAP is difficult since it is resistant to numerous kinds of antibiotics. Managing these infections often necessitates combination therapy and the utilization of more recent antimicrobial drugs, such as tigecycline and colistin (Spellberg&Bonomo, 2014) [(54)].

ISSUES THAT ARE NOW ARISING AND METHODS FOR HANDLING THEM:

1. **Study of the distribution and determinants of health-related events in populations:** There has been a worldwide rise in the occurrence of multidrug-resistant Acinetobacter baumannii, especially in hospital settings. The condition is linked to a significant level of illness and death, particularly in those with weakened immune systems and those who have

been hospitalized for an extended time (Fernandez-Cuenca et al., 2014) [55].

2. **Infection Control Measures:** Implementing efficient infection control measures is crucial in controlling the transmission of Multi-Drug Resistant (MDR) Acinetobacter baumannii. These measures encompass rigorous hand hygiene standards, thorough environmental cleaning, and the segregation of patients who are infected. Surveillance and antibiotic stewardship initiatives are also crucial in this context (Kwon et al., 2007) [56].
3. **Novel Therapeutic Approaches:** Ongoing research aims to provide new and innovative methods to treat MDR Acinetobacter baumannii infections. These include bacteriophage therapy, antimicrobial peptides, and the utilization of combination antibiotic therapy. In addition, there is a strong emphasis on the development of novel antibiotics that specifically target resistance pathways (Higgins et al., 2010) [57].

STAPHYLOCOCCUS AUREUS

Methicillin-resistant Staphylococcus aureus (MRSA) continues to be a notable pathogen in ventilator-associated pneumonia (VAP), especially in environments where there is a high usage of antibiotics. MRSA, which stands for methicillin-resistant Staphylococcus aureus, is a difficult infection to treat due to its high virulence and resistance mechanisms, such as the presence of the mecA gene that confers resistance to methicillin [40].

NEWLY IDENTIFIED DISEASE-CAUSING MICROORGANISMS

In recent years, there has been a rise in the occurrence of additional types of bacteria that are classified as gram-negative pathogens. Examples of

these bacteria include Stenotrophomonas maltophilia and Burkholderiacepacia. This increase has been observed primarily in individuals who have cystic fibrosis or other chronic lung disorders (Looney et al., 2009) [58]. These organisms frequently exhibit resistance to various antibiotics, which adds complexity to treatment methods.

Factors that increase the likelihood of developing Ventilator-Associated Pneumonia (VAP)

1. **Duration of Mechanical Ventilation:** Extended use of mechanical ventilation is a major contributing factor for ventilator-associated pneumonia (VAP). According to Wałaszek et al. (2016), there is a strong correlation between mechanical ventilation lasting more than 20 days and a higher occurrence of ventilator-associated pneumonia (VAP) [59]. Li et al. (2019) found that prolonged mechanical breathing raises the likelihood of ventilator-associated pneumonia (VAP) in individuals with traumatic brain injury (TBI) [60]. Cook et al. (1998) found that the length of time a patient requires mechanical ventilation is closely linked to the likelihood of acquiring ventilator-associated pneumonia (VAP), especially after the initial 48 hours of intubation [25].
2. **Preexisting medical conditions:** Individuals with preexisting medical disorders are more susceptible to acquiring ventilator-associated pneumonia (VAP). Wałaszek et al. (2016) identified several risk variables including multiple trauma, sepsis, central nervous system diseases, endocrine abnormalities, and respiratory diseases [59]. Li et al. (2019) emphasized that the severity of the original injury and the presence of other medical conditions are significant risk factors for ventilator-associated pneumonia (VAP) in patients with traumatic brain injury (TBI) [60]. Melsen et al. (2013) found that individuals with chronic obstructive

pulmonary disease (COPD) and weakened immune systems are especially susceptible to ventilator-associated pneumonia (VAP) [61].

3. **Invasive Medical Procedures:** Procedures that involve entering the body, such as reintubation, tracheostomy, and bronchoscopy, heighten the likelihood of developing Ventilator-Associated Pneumonia (VAP). According to Wałaszek et al. (2016), the operations of reintubation, tracheostomy, and bronchoscopy were found to be significant risk factors for ventilator-associated pneumonia (VAP). The risk ratios for these procedures were reported as 0.271, 0.309, and 0.316, respectively [59]. Li et al. (2019) identified tracheostomy as a significant risk factor for ventilator-associated pneumonia (VAP) in patients with traumatic brain injury (TBI) [60]. Chastre and Fagon (2002) highlighted that tracheostomy and repeated airway manipulations augment the likelihood of bacterial colonization and eventual ventilator-associated pneumonia (VAP) [30].
4. **Previous administration of antibiotics and receipt of blood transfusion:** Previous administration of antibiotics and receipt of blood transfusion upon admission are recognized as risk factors for ventilator-associated pneumonia (VAP). According to Li et al. (2019), blood transfusion was observed to dramatically elevate the risk of VAP [60]. Previous administration of antibiotics might disturb the usual collection of microorganisms in the body, which can encourage the development of bacteria that are resistant to treatment and raise the likelihood of acquiring ventilator-associated pneumonia (VAP) [62]. Hortal et al. (2009) demonstrated a correlation between previous antibiotic use and higher levels of multidrug-resistant bacteria colonization, which in turn led to an increased risk of ventilator-associated pneumonia (VAP) [63].
5. **Smoking and the use of sedatives:** Smoking and the use of sedatives can elevate the likelihood of developing ventilator-associated pneumonia (VAP). According to Li et al. (2019), smoking increased the likelihood of

getting ventilator-associated pneumonia (VAP) in patients with traumatic brain injury (TBI) by two times [60]. Sedatives hinder the cough reflex and diminish the capacity to eliminate secretions, hence heightening the vulnerability to VAP [64]. Papazian et al. (2016) emphasized the need to minimize continuous sedation to decrease the incidence of ventilator-associated pneumonia (VAP) [65].

6. **Cross-contamination and the ICU environment:** The incidence of VAP is increased by cross-contamination and inadequate infection control methods. Implementing effective hygiene practices and infection control measures is essential to decrease the danger. Safdar et al. (2005) emphasized the significance of adhering strictly to infection control practices to prevent cross-contamination and decrease the occurrence of ventilator-associated pneumonia (VAP) [66]. Kollef et al. (1993) highlighted that VAP infections can originate from healthcare staff and contaminated equipment, leading to cross-contamination [67].

CONSEQUENCES OF VAP:

1. **Prolonged Length of Stay:** VAP substantially extends the duration of ICU and hospital stays. According to Li et al. (2019), patients with ventilator-associated pneumonia (VAP) experienced longer hospitalizations in the intensive care unit (ICU) with an odds ratio (OR) of 6.85 and a 95% confidence interval (CI) of 4.90-8.79. They also had longer hospital stays with an OR of 10.92 and a 95% CI of 9.12-12.72 in traumatic brain injury (TBI) patients [60]. Fagon et al. (1993) provided evidence that ventilator-associated pneumonia (VAP) prolongs the duration of mechanical ventilation and the length of stay in the intensive care unit (ICU), resulting in increased healthcare expenses [68].

2. **Mortality Rates:** According to the study conducted by Wałaszek et al. (2016), the mortality rate among patients with VAP was 32.8% [59]. Li et al. (2019) found that there was no significant increase in mortality among TBI patients with VAP (OR 1.28; 95% CI 0.74-2.21) [60]. Bekaert et al. (2011) showed that ventilator-associated pneumonia (VAP) leads to higher fatality rates in intensive care unit (ICU) patients. However, the extent of this influence depends on the patient's underlying health conditions and the severity of their disease [69].

Proactive Measures

1. **Bed Head Elevation:** Raising the head of the bed to an angle of 30-45 degrees is beneficial in preventing aspiration, a significant contributing factor to Ventilator-Associated Pneumonia (VAP). Kollef (1993) suggested implementing this uncomplicated and efficient method as a means to decrease the occurrence of ventilator-associated pneumonia (VAP) [67]. Drakulovic et al. (1999) discovered that raising the head of the bed had a substantial impact on decreasing the likelihood of aspiration and ventilator-associated pneumonia (VAP) [70].
2. **Regular dental care:** Regular dental care using chlorhexidine decreases the colonization of respiratory pathogens and the occurrence of ventilator-associated pneumonia (VAP). In a meta-analysis conducted by Labeau et al. (2011), it was demonstrated that chlorhexidine oral care effectively reduced rates of ventilator-associated pneumonia (VAP) [71]. Chan et al. (2007) provided more evidence endorsing the effectiveness of chlorhexidine in the prevention of ventilator-associated pneumonia (VAP) in patients who are mechanically ventilated [72].
3. **Subglottic Secretion Drainage:** The use of endotracheal tubes equipped with subglottic secretion drainage helps avoid the build-up of secretions and reduces the risk of ventilator-associated pneumonia (VAP). Mahul et

al. (1992) showed that draining subglottic secretions effectively decreased the occurrence of ventilator-associated pneumonia (VAP) in patients who were intubated [73]. Dezfulian et al. (2005) emphasized that it plays a significant role in lowering ventilator-associated pneumonia (VAP) by limiting the inhalation of small amounts of liquid (microaspiration) [74].

4. **Sedation Minimization:** Implementing a strategy of daily interruption of sedation and evaluating the patient's readiness for extubation can effectively decrease the time of mechanical ventilation and lower the incidence of ventilator-associated pneumonia (VAP). Girard et al. (2008) demonstrated that using sedation and ventilator weaning techniques in pairs resulted in improved outcomes and a decrease in the prevalence of ventilator-associated pneumonia (VAP) [75]. The significance of daily sedation interruption in minimizing the incidence of ventilator-associated pneumonia (VAP) was highlighted by Schweickert et al. (2004) [76].

CLINICAL DIAGNOSIS:

The clinical diagnosis of Ventilator-Associated Pneumonia (VAP) entails identifying distinct signs and symptoms, including elevated body temperature, respiratory secretions containing pus, and the appearance of a new or worsening abnormality on chest radiography. Kalanuria et al. (2014) state that the clinical criteria for this condition consist of fever, leucocytosis, and the presence of purulent tracheal secretions [77]. Koenig and Truwit (2006) highlighted the importance of clinical indicators, such as fever and an increased number of white blood cells, in addition to radiographic evidence of new infiltrates, for the diagnosis of Ventilator-Associated Pneumonia (VAP) [78]. Research has demonstrated that the combination of new or worsening pulmonary infiltrate,

fever, and increased white blood cell count significantly suggests the presence of ventilator-associated pneumonia (VAP) [41].

RADIOLOGICAL DIAGNOSIS:

Chest radiography and computed tomography (CT) scans are crucial modalities for the diagnosis of VAP. Characteristic radiological findings of ventilator-associated pneumonia (VAP) consist of the presence of fresh or advancing infiltrates, consolidation, and cavitation. Kalanuria et al. (2014) emphasized the significance of using sequential chest radiographs to track the advancement of pneumonia in patients who are receiving mechanical ventilation [77]. According to Wunderink and Waterer (2009), CT scans offer more comprehensive data and aid in distinguishing VAP from other pulmonary diseases [79]. Recent research indicates that CT scans may have utility in patients who have clinical signs of respiratory tract infection despite having a normal chest radiograph.

MICROBIOLOGICAL DIAGNOSIS:

The study of respiratory secretions for microorganisms is essential for accurately diagnosing VAP. The detection of harmful germs using methods such as bronchoalveolar lavage (BAL) and protected specimen brush (PSB) is crucial. Kalanuria et al. (2014) highlighted the need to use quantitative cultures produced from bronchoalveolar lavage (BAL) or protected specimen brush (PSB) to confirm ventilator-associated pneumonia (VAP) (79). Koenig and Truwit (2006) advocated for the utilization of these intrusive sample approaches to acquire precise microbiological diagnoses [78]. Fagon et al. (1988) established that quantitative cultures obtained from bronchoalveolar lavage (BAL) fluid yield dependable outcomes in the diagnosis of ventilator-associated pneumonia (VAP) [68].

The guidelines of the European Respiratory Society recommend invasive sample procedures such as mini-BAL, bronchoscopic BAL, or PSB, along with quantitative cultures, for accurate diagnosis and effective use of antibiotics. The Infectious Diseases Society of America recommends the use of non-invasivesampling with semiquantitative cultures since it has shown comparable clinical outcomes.

Sampling Techniques: The precision of diagnosing VAP through microbiological means relies on the specific method used to collect secretions from the lower respiratory tract. The main methods comprise endotracheal aspirates (ETA), bronchoalveolar lavage (BAL), and protected specimen brush (PSB).

- **Endotracheal Aspirates (ETA)** are collected by extracting secretions from the endotracheal tube using suction. This technique is simple to execute and involves minimal intrusion; however, it may produce samples that are tainted by microorganisms present in the upper respiratory tract, resulting in inaccurate positive outcomes. Koenig and Truwit (2006) emphasized that ETAs are less precise in comparison to more invasive techniques [(78)].
- **Bronchoalveolar Lavage (BAL)** is a procedure that entails introducing sterile saline into a specific part of the lung using a bronchoscope and subsequently extracting the fluid for analysis. This method offers a more precise specimen from the lower respiratory system, hence minimizing the chances of contamination. Fagon et al. (1988) established the reliability of quantitative cultures from bronchoalveolar lavage (BAL) fluid for diagnosing ventilator-associated pneumonia (VAP) [(68)].
- **The Protected Specimen Brush (PSB)** is a technique that utilizes a safeguarded brush to get samples from the lower respiratory system employing a bronchoscope. This technique reduces the presence of

impurities and yields precise microbiological information. Chastre et al. (1984) discovered that when PSB samples are cultivated quantitatively, they exhibit a high level of specificity in diagnosing VAP [81].

Quantitative cultures refer to the process of enumerating the number of colony-forming units (CFUs) of bacteria present in respiratory samples. Thresholds are set to distinguish between colonization and infection.

Thresholds: The commonly acknowledged criteria for diagnosing Ventilator-Associated Pneumonia (VAP) are:

a) ETA: greater than or equal to 1 million colony-forming units per millilitre
b) BAL: greater than or equal to 10^4 colony-forming units per millilitre
c) PSB: Greater than or equal to 10^3 colony-forming units per millilitre

Koenig and Truwit (2006) highlighted the significance of these criteria in minimizing incorrect positive diagnoses and avoiding needless antibiotic administration [78].

Pathogen Identification: Identifying individual infections is essential for determining the proper antibiotic therapy. Notable pathogens include Pseudomonas aeruginosa, Acinetobacter baumannii, and Staphylococcus aureus.

Multidrug-resistant organisms (MDROs) present considerable difficulties in treatment due to their resistance to many drugs. Examples of MDROs include methicillin-resistant Staphylococcus aureus (MRSA) and carbapenem-resistant Acinetobacter. Torres et al. (2006) observed a growing occurrence of multidrug-resistant organisms (MDROs) in cases of ventilator-associated pneumonia (VAP), highlighting the need for precise microbiological identification to ensure effective treatment [38].

Rapid Diagnostic Techniques: Recent advancements in molecular diagnostics, such as polymerase chain reaction (PCR) and multiplex PCR, enable swift and accurate detection of diseases.

- **Polymerase Chain Reaction (PCR)** is a technique used to amplify bacterial DNA from respiratory samples, allowing for rapid and precise identification of pathogens. The research conducted by Luna et al. (2010) showed that PCR-based approaches greatly decrease the time required for diagnosis and the start of suitable treatment [(82)].
- **Multiplex PCR** is a technique that enables the identification of numerous pathogens at the same time, hence improving the effectiveness of diagnosing ventilator-associated pneumonia (VAP). The study conducted by De Francesco et al. (2006) demonstrated that multiplex PCR exhibits a high level of sensitivity and specificity in identifying microorganisms associated with ventilator-associated pneumonia (VAP) [(83)].

Biomarkers: Biomarkers, such as C-reactive protein (CRP) and procalcitonin, are utilized to bolster the diagnosis of ventilator-associated pneumonia (VAP). Increased concentrations of these biomarkers indicate an inflammatory reaction to infection. Kalanuria et al. (2014) observed that increased levels of CRP and procalcitonin are valuable for diagnosing VAP and assessing the effectiveness of treatment [(77)]. In their study, Schuetz et al. (2011) showed that using procalcitonin to guide antibiotic therapy in patients with ventilator-associated pneumonia (VAP) resulted in a shorter period of antibiotic use without negatively affecting clinical outcomes [(84)].

Clinical Prediction Scores: Clinical prediction scores, such as the Clinical Pulmonary Infection Score (CPIS), aid in determining the probability of Ventilator-Associated Pneumonia (VAP). The CPIS utilizes clinical, radiological, and microbiological criteria to evaluate the likelihood of VAP.

Kalanuria et al. (2014) stated that the CPIS is a useful tool for guiding the diagnosis and therapy of VAP [77]. Pugin et al. (1991) confirmed the validity of the CPIS, showing its usefulness in diagnosing VAP [85]. The CPIS score incorporates factors such as temperature, white blood cell count, tracheal secretion characteristics, oxygenation, and radiographic signs of pneumonia. Higher values on the CPIS indicate a greater probability of ventilator-associated pneumonia (VAP).

DIFFICULTIES IN DIAGNOSIS

1. **Differentiating VAP from Other Disorders:** A primary difficulty in identifying VAP is separating it from other disorders that present similar symptoms, such as atelectasis, pulmonary embolism, and ARDS. Koenig and Truwit (2006) highlighted the challenge of distinguishing ventilator-associated pneumonia (VAP) from other illnesses, particularly in critically sick patients with various comorbidities [78]. Incorrect interpretation of radiographic data and clinical symptoms can result in either excessive diagnosis or insufficient diagnosis of ventilator-associated pneumonia (VAP), which can complicate the care of patients [62].
2. **Overdiagnosis and Overtreatment:** The established clinical criteria for diagnosing Ventilator-Associated Pneumonia (VAP) can occasionally result in excessive diagnosis and treatment, leading to wasteful utilization of antibiotics. Kalanuria et al. (2014) emphasized the significance of precise diagnosis techniques to prevent these problems and enhance patient results [77]. Fagon et al. (1993) emphasized that relying solely on clinical criteria without confirming with microbiological evidence can result in unnecessary antibiotic usage and heightened resistance [68]. Overdiagnosis contributes to the escalation of healthcare expenses and

subjects patients to the potential adverse effects of unneeded interventions.

3. **Invasive vs. Non-invasive Procedures:** The use of invasive vs non-invasive procedures for diagnosing VAP is a subject of continuous controversy. While invasive techniques like BAL (bronchoalveolar lavage) and PSB (protected specimen brush) yield precise microbiological information, they come with inherent hazards and necessitate specialist expertise. Less invasive techniques, including endotracheal aspirates, are more convenient to carry out but may provide less precise outcomes. Koenig and Truwit (2006) examined the benefits and drawbacks of both methods, highlighting the importance of personalized patient evaluation [(78)]. The selection of a diagnostic approach should consider both the requirement for precision and the potential dangers and practicality of the procedure [(86)].

4. **Advanced Imaging:** Sophisticated imaging methods, such as chest CT and lung ultrasonography, have been investigated for their ability to diagnose VAP. Chest computed tomography (CT), while not commonly employed, might provide valuable assistance in cases where patients exhibit clinical signs of respiratory illness despite having a normal chest radiograph. Lung ultrasonography can be used to exclude pneumonia and detect specific characteristics such as subpleural consolidations and dynamic air bronchograms, which can confirm the diagnosis of VAP. A study conducted by Bouhemad et al. (2010) showcased the possible usefulness of lung ultrasound in the diagnosis of ventilator-associated pneumonia (VAP) [(86)].

Timing and sampling are crucial factors when it comes to respiratory tract sampling and the start of empirical antibiotic therapy. It is preferable to collect respiratory samples before initiating antibiotic treatment to ensure the

correctness of the diagnosis. Nevertheless, in cases of serious sickness, it may be necessary to administer empirical medication before obtaining a sample.

PREVENTION

Important Preventive Actions:

1. **The Head of the Bed (HOB) is raised:**
    - Study Specifics: Raising the HOB to 30-45 degrees is advised by several studies, including those conducted by Cook et al. (1998) and Oliveira et al.(2014)[25,87].
    - Recommendation: This posture lowers the chance of aspiration, which is a major contributor to VAP. Hugonnet et al. (2004) discovered that this measure's regular application greatly reduced VAP rates[88].

2. **Daily Vacation from Sedation and Evaluation of Preparedness to Extubate:**
    - Study Details: Research by Klompas (2010)[89] and Mastrogianni et al. (2023)[90]supports the practice of daily sedation getaways.
    - Recommendation: It is suggested that sedation be stopped every day to enable the evaluation of a patient's preparedness for weaning off mechanical ventilation. This can shorten the period of mechanical ventilation and lower the incidence of ventilator-associated pneumonia (VAP).
3. **Prevention of Peptic Ulcer Disease (PUD):**
    - Research Details: Bouadma et al. (2012)[91]and other studies suggest PUD prophylaxis.

- Recommendation: By preserving gastrointestinal integrity and limiting bacterial translocation, reducing stress ulcers and gastrointestinal bleeding indirectly lowers the incidence of VAP.

4. **Prevention of Deep Venous Thrombosis (DVT):**
   - Study Details: Featured in many care bundles and suggested by Oliveira et al. (2014). [87]
   - Recommendation: It is suggested that DVT prophylaxis improves overall patient outcomes and may lower the incidence of VAP by preventing thromboembolic consequences, which are common in immobilized ICU patients.
5. **Dental Hygiene:**
   - Study Specifics: Research conducted in 2006 by Craven et al. [98] and in 2014 by Oliveira et al.[87]highlights the significance of routine dental care using chlorhexidine.
   - Recommendation: By lowering the oral cavity's microbial burden, this procedure helps to avoid bacterial colonization and the consequent development of VAP.
6. **Drainage of Subglottic Secretions:**
   - Specifics of the Study: Endotracheal tubes with subglottic secretion drainage holes are recommended by Bouadma et al. (2012)[91]and Oliveira et al. (2014)[87]
   - Advice: By preventing secretion build-up above the cuff, this precaution lowers the chance of aspiration and VAP.
7. **Digestive Decontamination Selectively (SDD):**
   - Study Specifics: The significance of SDD is emphasized by Kollef et al. (1997)[93]and Oliveira et al. (2014)[87]
   - Suggestion: To effectively lower VAP rates, SDD entails administering non-absorbable antibiotics to the stomach and oropharynx to stop pathogenic bacteria from colonizing there.

CONCLUSION FROM IMPORTANT RESEARCH:

- **Cook and Associates (1998):**
  - Specifics: The study found that the risk of ventilator-associated pneumonia (VAP) was roughly 3% per day for the first five days of mechanical breathing, 2% per day for days five through ten, and 1% per day after that[(25)].
  - Results: To lower the incidence of VAP, it was highlighted how crucial early intervention and continuous preventive efforts are.
- **Hugonnet and associates (2004):**
  - Specifics: It was discovered that adherence to preventive measures and increased staffing levels considerably lowered VAP rates[(88)].
  - Results: Emphasized how crucial nurse-to-patient ratios and hospital policies are to reducing VAP.
- **Bouadma et al. (2012):**
  - Details: Shown that the incidence of VAP was dramatically decreased by a treatment bundle that included SDD and subglottic secretory drainage[(91)].
  - Results: Compared to single interventions, it is suggested that integrating various preventative measures is more beneficial.
- **Kollef group (1997):**
  - Specifics: Demonstrated a significant decrease in VAP rates through selective decontamination of the digestive tract (SDD)[(93)].
  - Results: Raised concerns over antibiotic resistance and advised that local microbial patterns be carefully considered before employing SDD.
- **Tablan Group (2004):**
  - Details: Detailed CDC recommendations were given for avoiding pneumonia linked to healthcare [34].

- Results: Contained several preventative measures, including frequent patient monitoring, sterile procedures, and hand cleanliness.

- **Craven Group (2006):**
  - Details: Talked about how oral antiseptics, such as chlorhexidine, can lower the incidence of VAP[92].
  - Results: Frequent dental care is advised as a vital part in preventing VAP.
- **Oliveira group (2014):**
  - Specifics: Reviewed several prophylactic strategies, highlighting the significance of SDD and mechanical strategies such as subglottic secretion drainage[93].
  - Results: Emphasized the need to prevent VAP using a comprehensive approach.
- **Richards et al. (1999):**
  - Details: A meta-analysis demonstrating the benefits of semi-recumbent posture[16].
  - Results: To prevent VAP, it is advised that this placement be used as a routine procedure in ICUs.
- **Chastre and Fagon (2002):**
  - Details: Talked about different preventive measures, the value of early diagnosis, and the proper administration of antibiotics[30].
  - Results: Highlighted the necessity of individualized antibiotic treatment based on patient circumstances and microbiological trends.
- **Klompas (2010):**
  - Information: Systematic review emphasizing the advantages of regular getaways from sedation and impromptu breathing exercises[89].

- Results: It is suggested that these procedures lower the incidence of VAP by encouraging early extubation and reducing needless sedation.

- **Alvarez-Lerma et al. (2018):**
  - Specifics: Examined the effects of a nationwide bundle for VAP prevention on 171,237 patients in 181 ICUs[94].
  - Results: Showed that a thorough preventative approach improved patient outcomes and dramatically lowered VAP rates.
- **Kao and associates (2019):**
  - Details: This study looked at Taiwanese ICU types' compliance with VAP preventive packages[95].
  - Results: It was discovered that there was a substantial correlation between higher compliance rates and a lower incidence of VAP.
- **ZagaloGroup (2014):**
  - Specifics: Evaluated the effect of preventative measure compliance on health worker training[96].
  - Results: Emphasized that ongoing instruction and compliance oversight are essential to the success of programs designed to prevent VAP.

The Effects of Care Packages: When used consistently, care bundles have been demonstrated to considerably lower VAP rates. The Institute for Healthcare Improvement established the "IHI Ventilator Bundle," which consists of daily sedation vacations, dental cleanliness with chlorhexidine, prevention for PUD and DVT, and elevation of the HOB. When these steps are taken together and followed, the incidence of VAP is greatly decreased.

TREATMENT

PATHOGENS AND THEIR TREATMENT

Common Pathogens

- Gram-negative bacteria: Pseudomonas aeruginosa, Escherichia coli, Klebsiella pneumoniae, Acinetobacter species
- Gram-positive bacteria: Staphylococcus aureus[30,91].

Acinetobacter baumannii A. baumannii is a major cause of VAP, notorious for its resistance to multiple antibiotics. Effective treatment requires a tailored approach based on susceptibility patterns[44].

TREATMENT STRATEGIES

1. **Antibiotic Therapy:**
    - **Empiric Therapy:** Initial empiric antibiotic therapy is guided by local microbial flora and resistance patterns. Commonly used antibiotics include carbapenems, aminoglycosides, polymyxins, and tigecycline[41]. Studies have shown that appropriate initial therapy is crucial, with delayed or inappropriate treatment leading to significantly higher mortality rates.
    - **Targeted Therapy:** Once culture results are available, therapy should be adjusted to target the specific pathogens identified. For example, carbapenem-resistant A. baumannii may require the use of polymyxins (colistin), tigecycline, or newer agents like cefiderocol [97].
2. **Carbapenem-Resistant A. baumannii:**
    - **Combination Therapy:** Piperaki et al. (2019) recommend combination therapy using antibiotics such as sulbactam, colistin,

tigecycline, and cefiderocol [98]. The combination of colistin with high-dose sulbactam has shown synergistic effects, improving clinical outcomes.

- **Newer Agents:** Karruli et al. (2023) highlight the use of cefiderocol and sulbactam-durlobactam against carbapenem-resistant A. baumannii. Cefiderocol has demonstrated potent activity against multidrug-resistant strains, with MIC values significantly lower than other antibiotics [99].

3. **Duration of Therapy:**
   - **Optimal Duration:** Shorter courses (7-8 days) are as effective as longer courses (15 days) and reduce the risk of antibiotic resistance and adverse effects. Capellier et al. (2012) found no significant difference in mortality or recurrence rates between 8-day and 15-day treatment courses [100].
4. **Adjunctive Therapies:**
   - **Antibiotic De-escalation:** Masterton (2011) recommends de-escalation of antibiotics based on clinical response and microbiological data to minimize resistance. This involves narrowing the spectrum of antibiotics once pathogens and their susceptibilities are known [77].
   - **Immunomodulatory Agents:** Research into immunomodulatory therapies is ongoing, though none are currently recommended in routine practice. Potential agents include corticosteroids and immunoglobulins, aimed at modulating the host immune response [78].

EVIDENCE FROM KEY STUDIES

1. **Koenig and Truwit (2006):**
    - Details: Comprehensive review of VAP diagnosis, treatment, and prevention.
    - Findings: Emphasized the importance of appropriate empiric antibiotic therapy followed by de-escalation based on culture results. Highlighted that timely and appropriate empiric therapy significantly reduces mortality [(78)].
2. **Piperaki et al. (2019):**
    - Details: Discussed treatment options for carbapenem-resistant A. baumannii.
    - Findings: Highlighted the effectiveness of combination therapy, with sulbactam-colistin combinations showing particular promise. Cefiderocol, a siderophore cephalosporin, demonstrated MICs ranging from 0.12 to 4 μg/mL against carbapenem-resistant strains [(98)].
3. **Karruli et al. (2023):**
    - Details: Evaluated cefiderocol and sulbactam-durlobactam against carbapenem-resistant A. baumannii.
    - Findings: These newer agents show promise in treating multidrug-resistant infections, with cefiderocol achieving clinical success rates of approximately 70% in treated cases [(99)].
4. **Kalanuria et al. (2014):**
    - Details: Focused on the overall management of VAP.
    - Findings: Reinforced the importance of early, appropriate empiric therapy and subsequent de-escalation. Noted that inappropriate initial therapy can increase mortality by up to 50% [(77)].
5. **Capellier et al. (2012):**

- Details: Compared 8 versus 15 days of antibiotic treatment for early-onset VAP.
- Findings: Found no significant difference in outcomes between the two durations, supporting shorter courses to reduce adverse effects and resistance. Mortality rates were 18.8% for the 8-day group versus 17.2% for the 15-day group [97].

6. **Koenig and Truwit (2006):**
   - Details: Discussed various antibiotic strategies and the importance of tailoring therapy based on individual patient factors and local resistance patterns.
   - Findings: Emphasized personalized treatment plans and the potential benefits of newer antibiotics. Highlighted a mortality reduction from 34% to 15% with appropriate empiric therapy [78].
7. **Munoz-Price and Weinstein (2008):**
   - Details: Reviewed the epidemiology and treatment of Acinetobacter infections.
   - Findings: Stressed the challenges posed by multidrug-resistant A. baumannii and the need for combination therapy. Colistin-based combinations showed efficacy in over 60% of treated cases [44].
8. **Martin-Loeches et al. (2013):**
   - Details: Examined the impact of potentially resistant microorganisms in VAP patients.
   - Findings: Identified A. baumannii as a significant pathogen and recommended aggressive and targeted treatment approaches. Mortality was significantly higher in patients with multidrug-resistant infections [101].
9. **Walkey et al. (2011):**
   - Details: Compared linezolid versus glycopeptide antibiotics for MRSA nosocomial pneumonia.

- Findings: Provided insights into antibiotic choices, although not directly focused on A. baumannii, highlighting the need for targeted therapy based on specific pathogens. Linezolid showed a higher clinical cure rate (78%) compared to vancomycin (66%) [102].

10. **Chastre et al. (2003):**
    - Details: Investigated the role of invasive and non-invasive strategies in managing suspected VAP.
    - Findings: Supported the use of bronchoscopic techniques for accurate diagnosis and targeted therapy. Mortality rates were lower in the bronchoscopic group (25%) compared to the non-bronchoscopic group (37%) [103].

The treatment of VAP, particularly when caused by multidrug-resistant A. baumannii, requires a multifaceted approach. Empiric therapy must be initiated promptly, with adjustments based on culture results and susceptibility patterns. Newer antibiotics like cefiderocol and combination therapies offer promising solutions against resistant pathogens. Consistent application of evidence-based treatment protocols and antibiotic stewardship are essential for improving outcomes and combating antibiotic resistance.

MATERIALS AND METHODS

SOURCE OF DATA: Patients above the age of 18 years admitted to the Intensive Care Unit of KLEs Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, with diagnosis of Ventilator-associated pneumonia (VAP)

STUDY DESIGN: Cross-sectional study

STUDY PERIOD: January 2023 – December 2023

SAMPLE SIZE: 68

Chaari et al. [*] conducted a study entitled "Acinetobacter baumannii ventilator-associated pneumonia: epidemiology, clinical characteristics, and prognosis factors", in which they reported the prevalence of Acinetobacter baumannii VAP in 4.2% of all the adult patients admitted in ICU

So, considering this incidence, we used the following incidence formula for calculating the sample size.

The sample size n and margin of error E are given by:

$x = Z(c/100)\ 2\ r(100-r)$

$n = N\ x/((N-1)\ E\ 2 + x)$

$E = Sqrt\ [(N - n)\ x/n(N-1)]$

Where N is the population size (N=20000), r is the fraction of responses that you are interested in (r=4.2%), and Z(c/100) is the critical value for the confidence level c (Z=1.96).

Putting the above values in the above formula, the sample size obtained is 62 patients, at a confidence interval of 95% and 80% power of the study.

Considering an attrition rate of 10%, we intend to include 68 patients of Acinetobacter baumannii VAP in our study.

INCLUSION CRITERIA:

1. Patients above 18 years of age.
2. Patients in which A. Baumannii is detected in culture samples of blood, tracheobronchial secretion and BAL.
3. Patients fulfilling diagnostic criteria of VAP.

EXCLUSION CRITERIA:

1. Patients incubating pneumonia before intubation.

STUDY PROTOCOL:

- Patients fulfilling the inclusion criteria will be enrolled in the study after obtaining their informed consent.
- Positive cultures growing A. Baumannii will be notified from the microbiology lab.
- Positive culture with A. Baumannii will be subjected to antibiotic resistance.
- All positive cultures will be traced back to the respective patients and patients fulfilling inclusion criteria will be subjected to thorough clinical examination, and complete history taking. Biological findings will be taken from the patient's medical file to identify any co-morbidities, or risk factors associated with A. Baumannii VAP.
- Resistance patterns from culture will be correlated with the clinical profile of the patient thereby establishing the link between the same
- Data will be analysed and tabulated.

DATA COLLECTION PROCEDURE :

- Study subjects traced will be subjected to thorough history taking to identify any comorbidities (T2DM, Hypertension, IHD, COPD), risk

factors (Alcoholism, smoking, malignancy, previous surgery, prior intubation, prev. antibiotic use, type of nutrition etc)

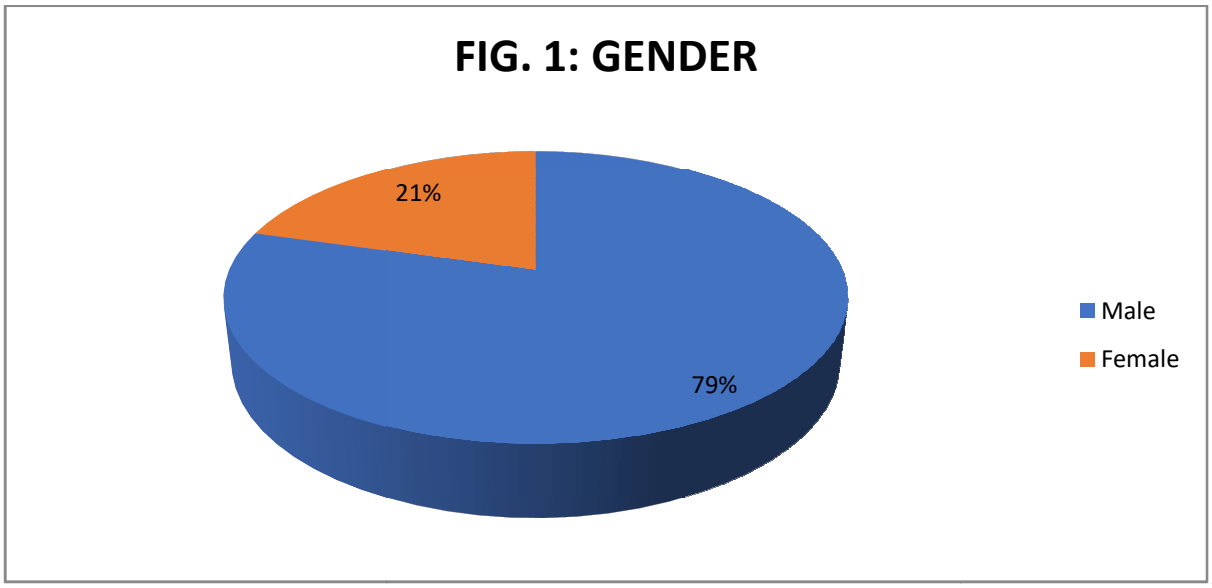
- Necessary blood values will be taken from the patient's medical file (ABG, Hb, TLC, Platelet, BUN, Creatinine etc)
- Scores like SAPS 2, GCS, Q-SOFA, and APACHE 2 will be calculated.

STATISTICAL ANALYSIS: Statistical Analysis will be done via descriptive analysis using SPSS and the Chi-Square test.

OBSERVATION AND RESULTS

TABLE 1:FREQUENCY DISTRIBUTION OF GENDER

Gender	N	%
Male	54	79.4
Female	14	20.6
Total	68	100.0



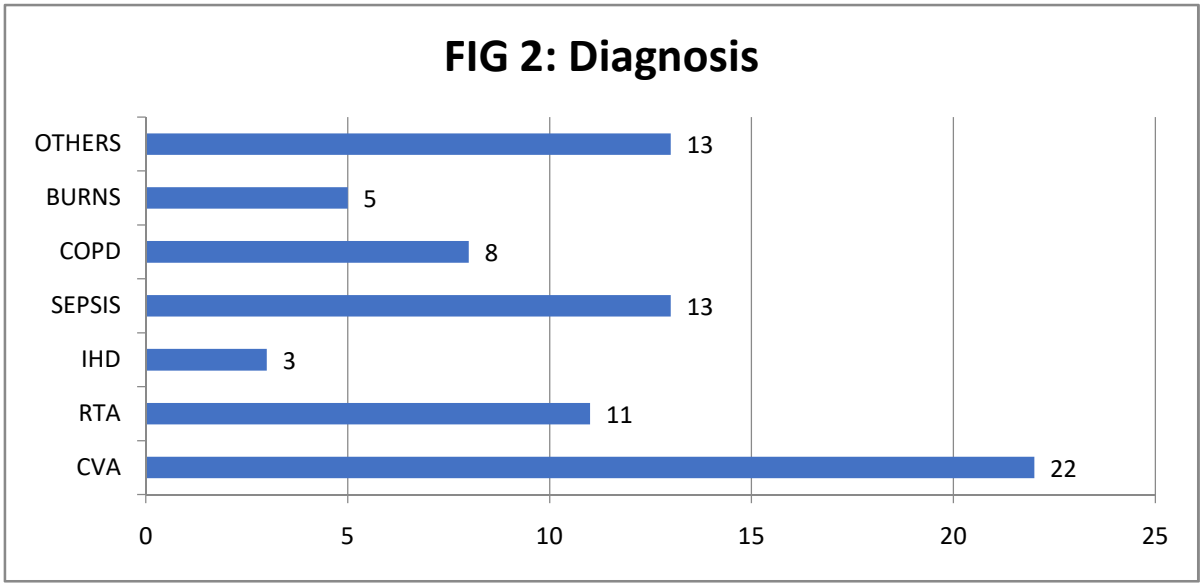
Mean Age: 48.32 ± 17.8 years (range 18-83 years)

Results:

A total of 68 patients were included in the study, with a mean age of 48.32 years, ranging from 18 to 83 years. The study cohort consisted predominantly of men, with 54 male patients (79.4%) and 14 female patients (20.6%). This results in a male-to-female ratio of approximately 3.86:1.

TABLE 2: FREQUENCY DISTRIBUTION OF DIAGNOSIS

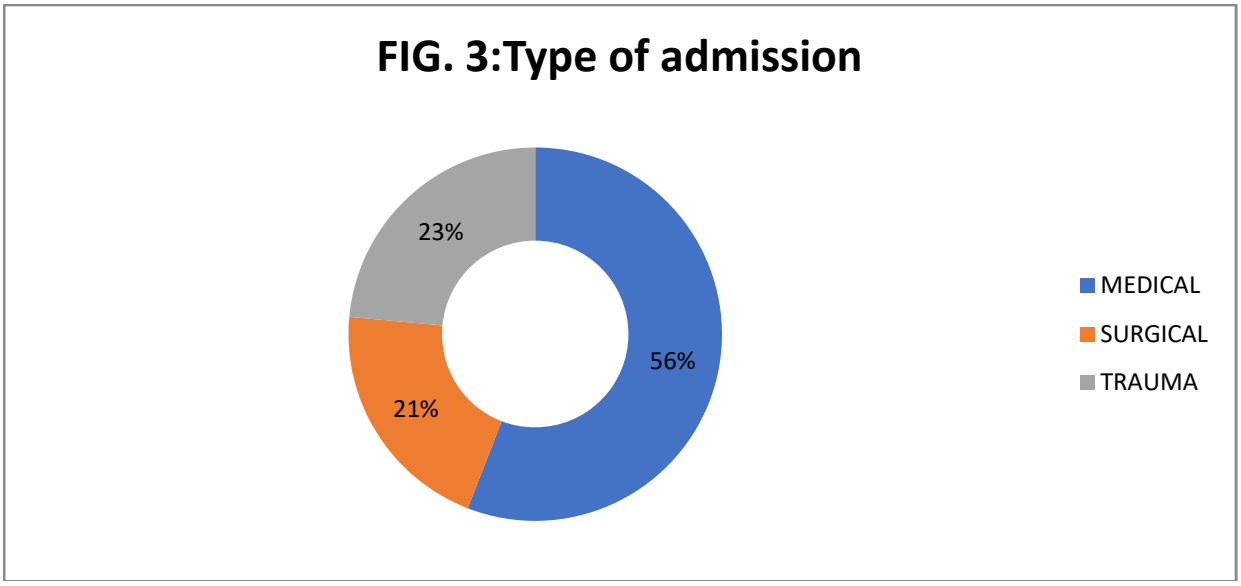
Diagnosis	N	%
CVA	22	29.3
RTA	11	14.7
IHD	3	4.0
SEPSIS	13	17.3
COPD	8	10.7
BURNS	5	6.7
OTHERS	13	17.3



A total of 75 diagnoses were recorded in the study population. The most common diagnosis was cerebrovascular accident (CVA), which accounted for 29.3% (22 cases) of the total diagnoses. Sepsis was the second most common diagnosis, making up 17.3% (13 cases) of the diagnoses. Road traffic accidents (RTA) represented 14.7% (11 cases), while chronic obstructive pulmonary disease (COPD) constituted 10.7% (8 cases). Other diagnoses included burns (6.7%, 5 cases), ischemic heart disease (IHD) (4%, 3 cases), and various other diseases (17.3%, 13 cases).

TABLE 3: FREQUENCY DISTRIBUTION OF TYPES OF ADMISSIONS

Type of admission	N	%
Medical	38	55.9
Surgical	14	20.6
Trauma	16	23.5
Total	68	100.0



The study included 68 patients, classified based on their type of admission. The majority, 55.9% (38 patients), were admitted from the medical department. Trauma care admissions accounted for 23.5% (16 patients), while 20.6% (14 patients) were admitted from the surgical department.

TABLE 4: FREQUENCY DISTRIBUTION OF PRIOR COLONIZATION WITH MRSA

PRIOR COLONISATION WITH MRSA	N	%
Yes	2	2.9
No	66	97.1
Total	68	100.0

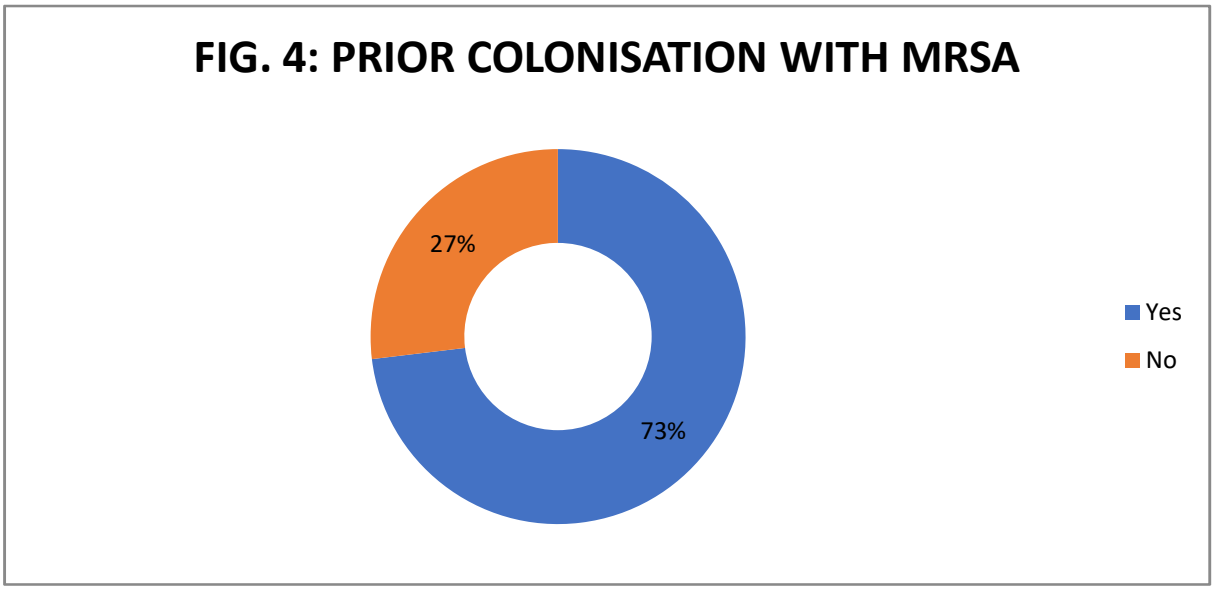


TABLE 5: FREQUENCY DISTRIBUTION OF PRIOR BETALACTUM - CARBAPENEM

PRIOR BETA LACTAM - CARBAPENEM	N	%
Yes	46	67.6
No	22	32.4
Total	68	100.0

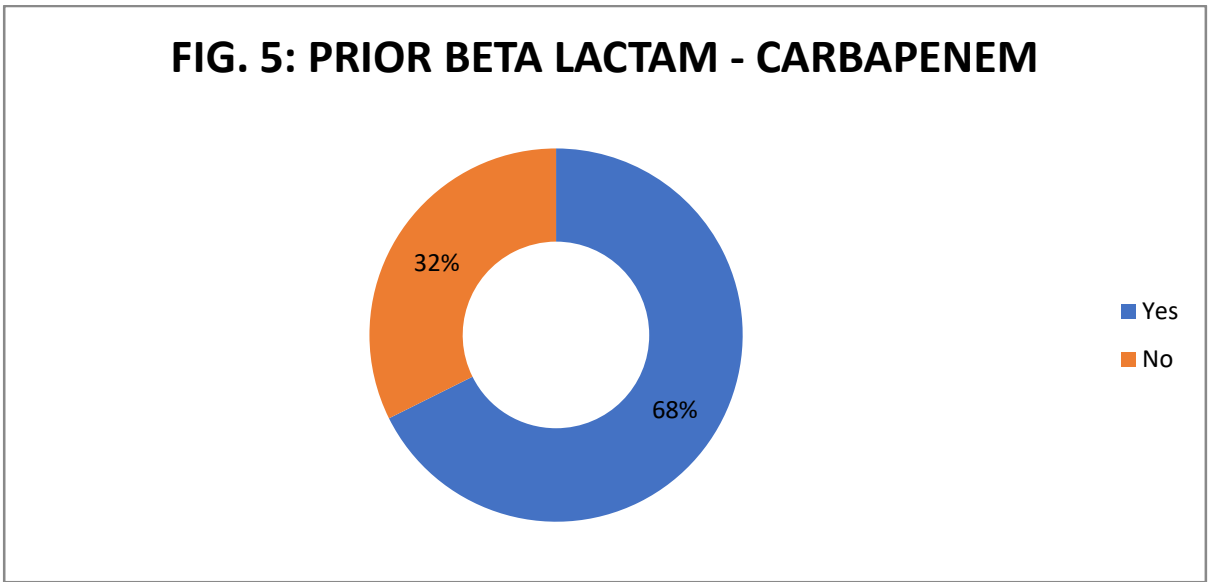
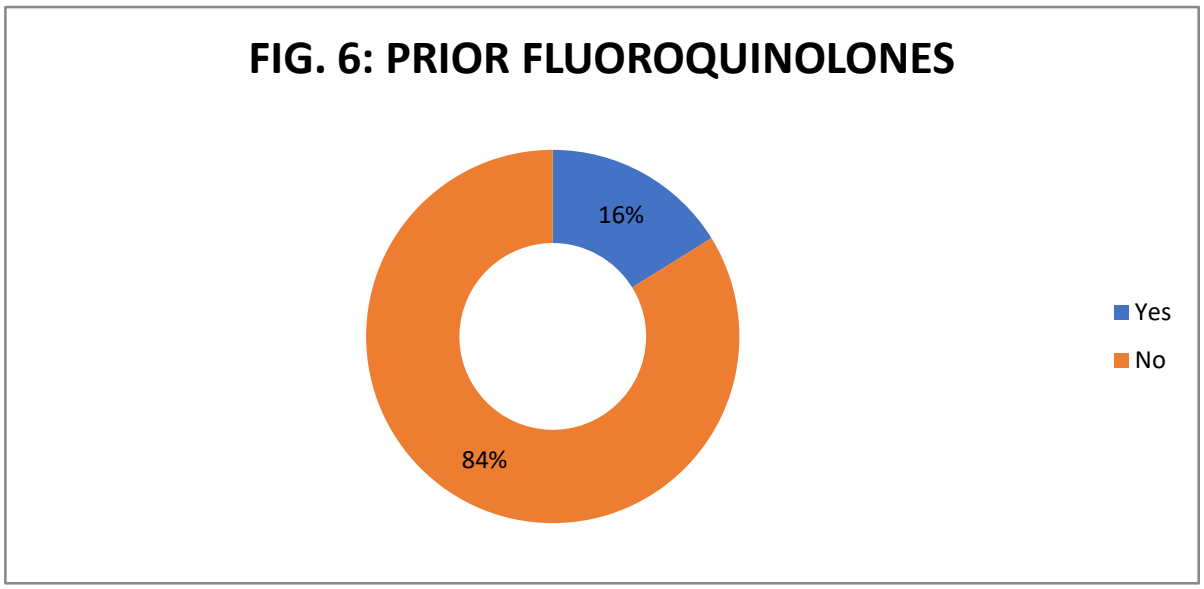


TABLE 6: FREQUENCY DISTRIBUTION OF PRIOR FLUOROQUINOLONES

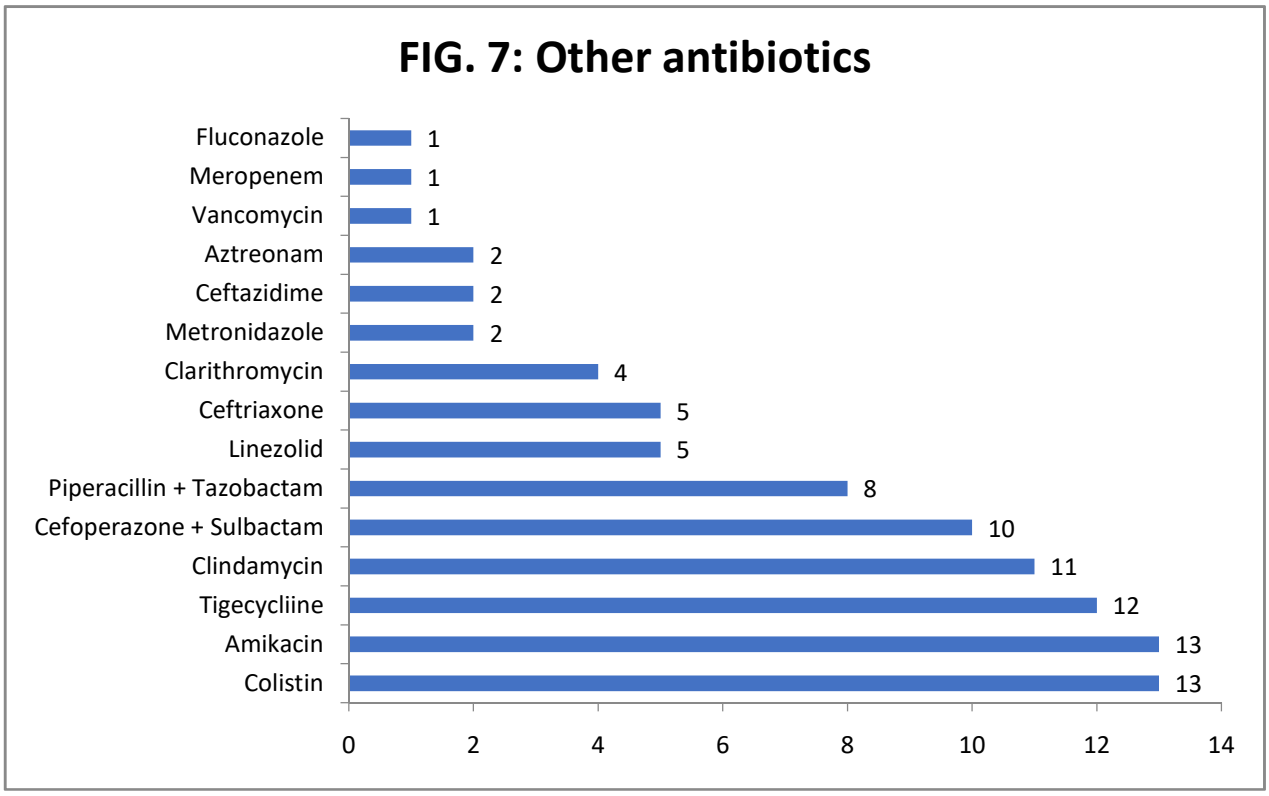
PRIOR FLUOROQUINOLONES	N	%
Yes	11	16.2
No	57	83.8
Total	68	100.0



Of 68 patients, 2.9% had prior colonization with MRSA and 67.6% had been treated with Carbapenem; 16.2% with fluoroquinolones earlier.

TABLE 7: FREQUENCY DISTRIBUTION OF PRIOR USE OF OTHER ANTIBIOTICS

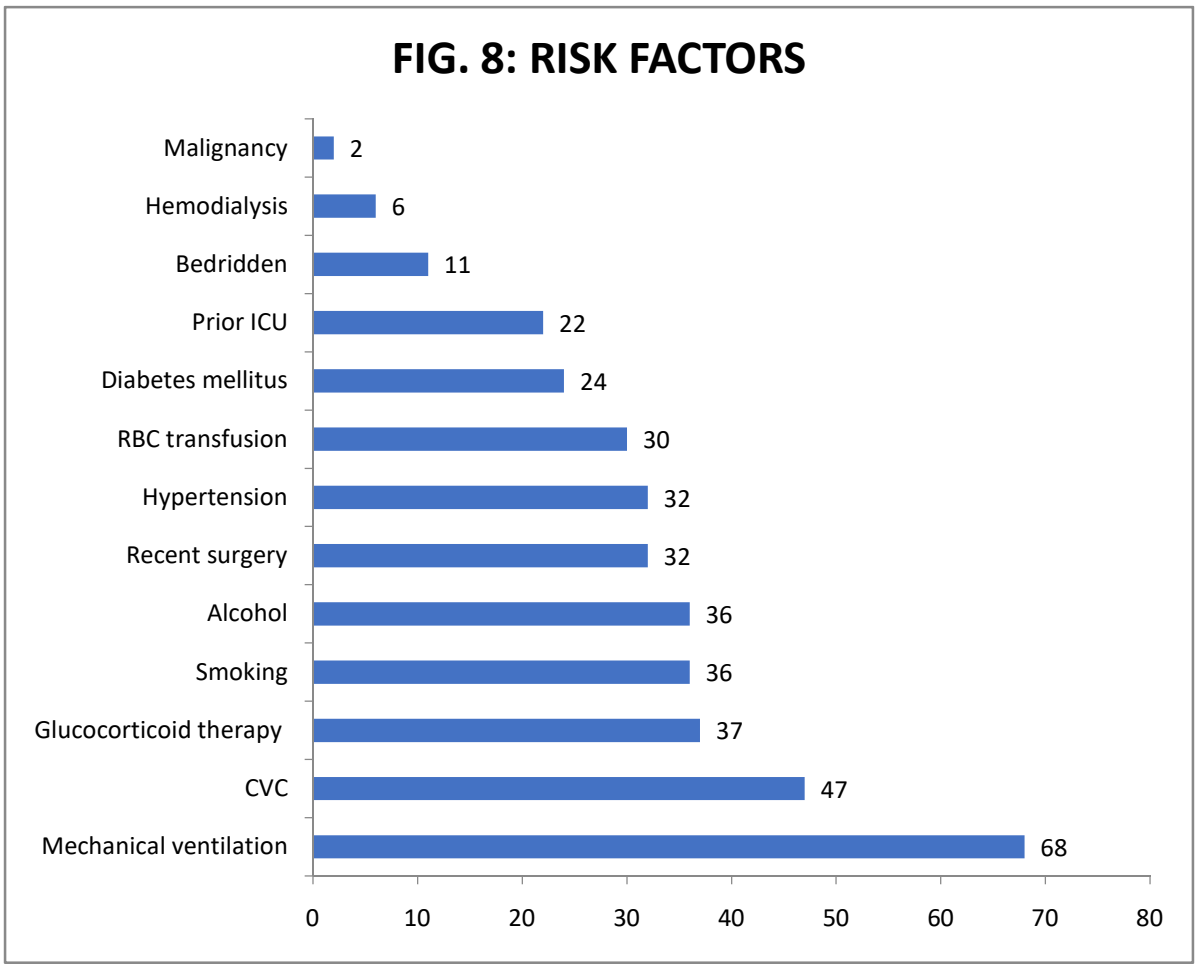
Other antibiotics	N	%
Colistin	13	19.1
Amikacin	13	19.1
Tigecycline	12	17.6
Clindamycin	11	16.2
Cefoperazone + Sulbactam	10	14.7
Piperacillin + Tazobactam	8	11.8
Linezolid	5	7.4
Ceftriaxone	5	7.4
Clarithromycin	4	5.9
Metronidazole	2	2.9
Ceftazidime	2	2.9
Aztreonam	2	2.9
Vancomycin	1	1.5
Meropenem	1	1.5
Fluconazole	1	1.5



In a total of 68 patients, **Colistin and Amikacin were the top antibiotics (19.1% each)** used for the patients followed by Tigecycline (17.6%) and Clindamycin (16.2%). Some patients received antibiotics in combination and the distribution is summarized in the above table.

TABLE 8: FREQUENCY DISTRIBUTION OF RISK FACTORS

FACTORS	N	%
Mechanical ventilation	68	100.0
CVC	47	69.1
Glucocorticoid therapy	37	54.4
Smoking	36	52.9
Alcohol	36	52.9
Recent surgery	32	47.1
Hypertension	32	47.1
RBC transfusion	30	44.1
Diabetes mellitus	24	35.3
Prior ICU	22	32.4
Bedridden	11	16.2
Haemodialysis	6	8.8
Malignancy	2	2.9



In our study, all 68 patients (100%) with ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii required mechanical ventilation, highlighting its role as a critical risk factor. Central venous catheter (CVC) use was prevalent in 69.1% of patients, while glucocorticoid therapy was administered to 54.4%. Lifestyle factors such as smoking and alcohol use were equally common, each reported in 52.9% of the patients. Both recent surgery and hypertension were noted in 47.1% of the cases. Additionally, 44.1% of the patients received RBC transfusions. Other notable risk factors included diabetes mellitus (35.3%), prior ICU admission (32.4%), being bedridden (16.2%), haemodialysis (8.8%), and malignancy (2.9%).

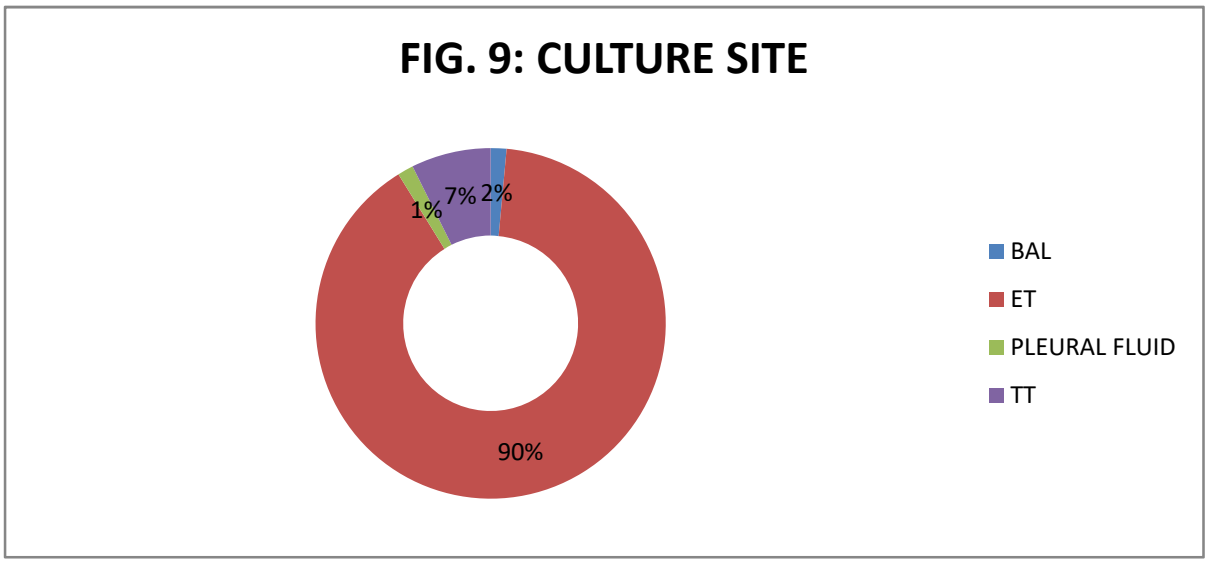
TABLE 9: DESCRIPTIVE STATISTICS OF CLINICAL AND HEMATOLOGICAL PARAMETERS

Parameters	Minimum	Maximum	Mean	SD
SBP	70.0	230.0	122.353	34.7390
DBP	40.0	150.0	75.588	19.6524
PULSE	52.0	178.0	104.691	27.8505
SPO22	80.0%	100.0%	94.647%	5.1969%
FIO2	21.0%	100.0%	71.544%	26.3487%
RR	17.0	49.0	31.015	8.2217
PH	6.8	7.5	7.316	.1276
PO2	59.0	307.0	114.487	50.9833
PCO2	17.3	91.0	41.296	15.8941
HCO3	6.5	54.1	21.375	7.8430
SAO2	86.0	99.0	96.047	3.1539
PaO2/FiO2	59.0	435.0	185.432	96.7600
HB	5.1	15.8	10.697	2.5633
PCV	15.4	50.0	35.900	7.3447
RBC	1.9	6.2	3.943	.8784
PLT	30.0	739.0	253.059	124.2383
WBC	5.0	72.3	16.396	9.3075
UREA	7.4	372.0	61.481	61.7549
CR	.4	6.7	1.561	1.2738
NA	107.0	158.0	137.971	8.5971
K	2.9	6.2	4.325	.7797
PT/INR	.7	3.8	1.371	.6518
GCS	2.0	15.0	6.471	3.3210
APACHE II	2.0	53.0	24.691	12.3883
Q-SOFA	.0	3.0	1.941	.9123
SAPS II	15.0	104.0	54.603	22.4001
LOS-HOSPITAL	3.0	93.0	21.544	16.8110
LOS-ICU	2.0	89.0	19.426	15.9611
ICU PRIOR TO MV	.0	22.0	2.088	3.8117

ICU PRIOR TO VAP	2.0	70.0	12.544	12.0254
MV PRIOR TO VAP	1	63	10.54	10.913

TABLE 10: FREQUENCY DISTRIBUTIONS OF THE CULTURAL SITES

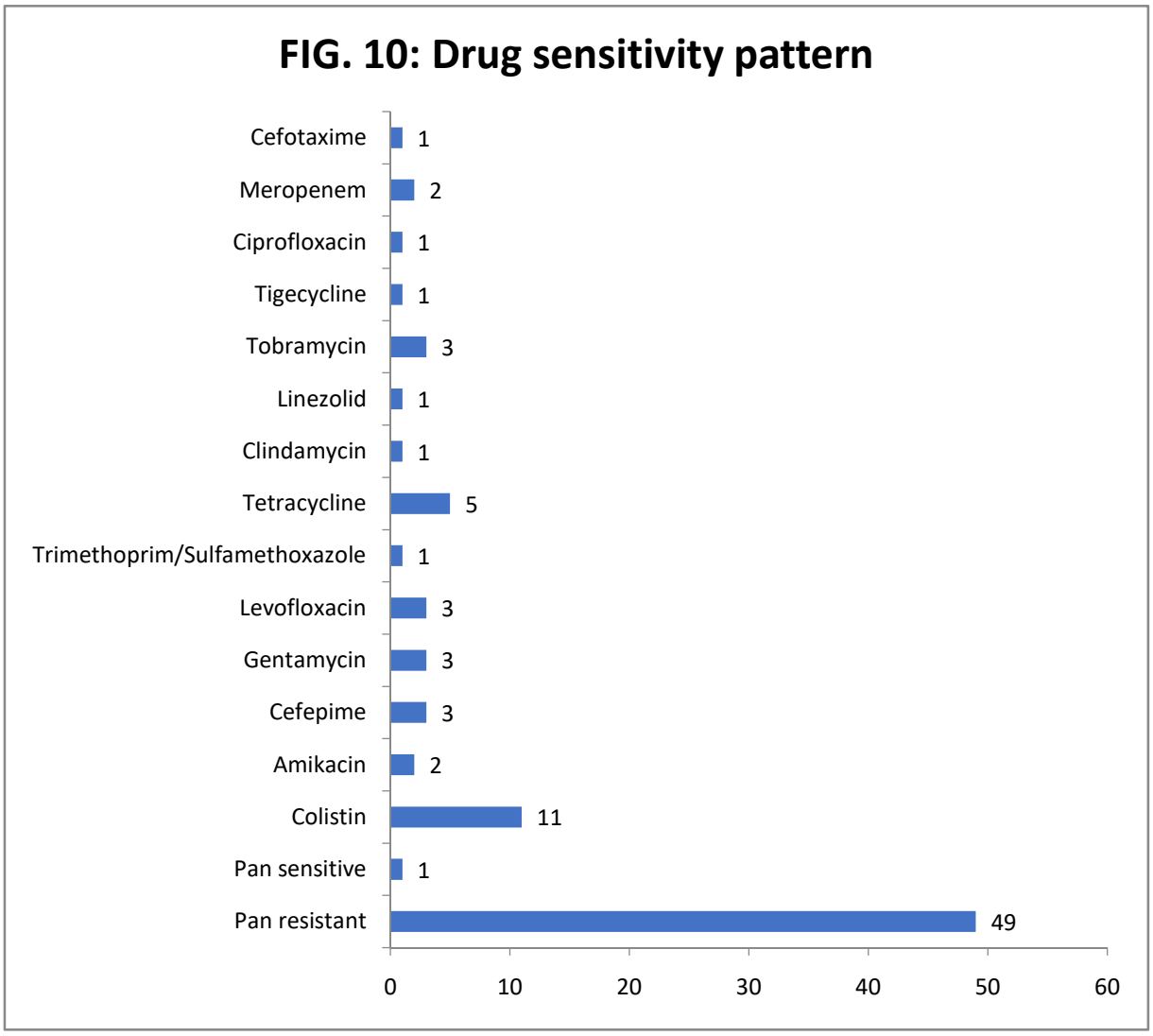
CULTURE SITE	N	%
BAL	1	1.5
ET	61	89.7
PLEURAL FLUID	1	1.5
TT	5	7.4
Total	68	100.0



In our study, most samples (89.7%) for diagnosing ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii were collected from endotracheal tubes (ET), highlighting its role as the primary site for pathogen isolation. Tracheostomy tubes (TT) accounted for 7.4% of the samples, reflecting their use in patients with prolonged mechanical ventilation. Bronchoalveolar lavage (BAL) and pleural fluid samples were each collected in 1.5% of the cases, indicating their limited but specific roles in the diagnostic process.

TABLE 11: FREQUENCY DISTRIBUTION OF ANTIBIOTIC DRUGS SENSITIVITY PATTERN

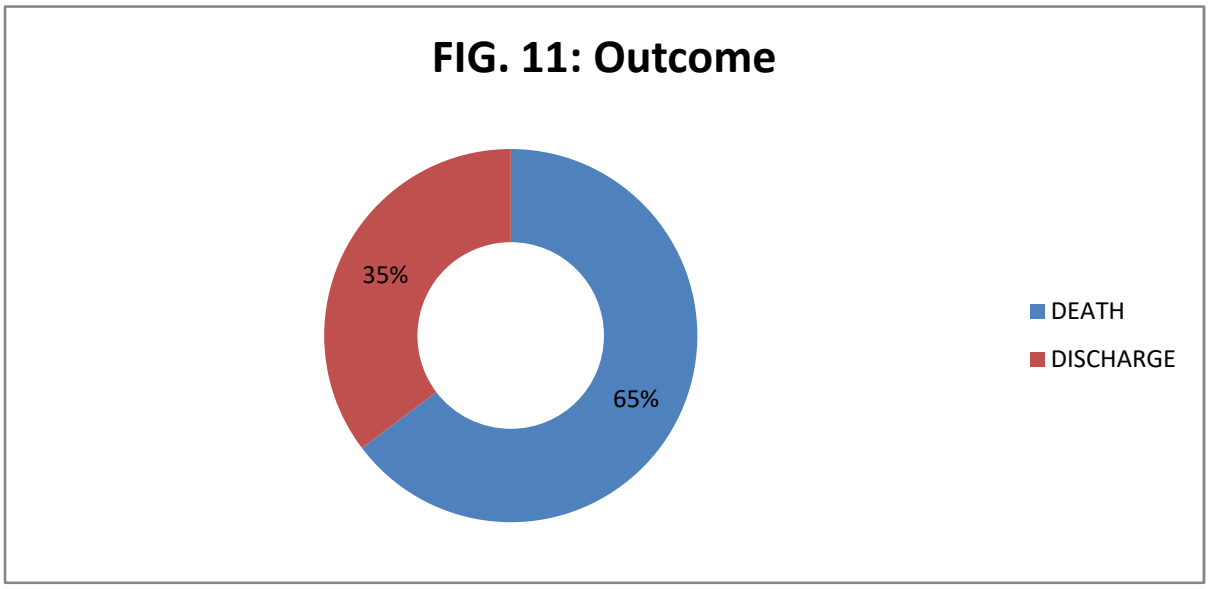
Drug sensitivity pattern	N	%
Pan resistant	49	72.1
Pan sensitive	1	1.5
Colistin	11	16.3
Amikacin	2	3.0
Cefepime	3	4.5
Gentamycin	3	4.5
Levofloxacin	3	4.5
Trimethoprim/Sulfamethoxazole	1	1.5
Tetracycline	5	7.5
Clindamycin	1	1.5
Linezolid	1	1.5
Tobramycin	3	4.5
Tigecycline	1	1.5
Ciprofloxacin	1	1.5
Meropenem	2	3.0
Cefotaxime	1	1.5



The antibiotic sensitivity pattern of the patient samples was summarized, revealing that 72.1% of the patients were resistant to Pan drugs. Colistin (16%) was identified as the most sensitive antibiotic, followed by Tetracycline (7.5%). Additionally, 24% of the patients were sensitive to Cefepime, Gentamycin, Levofloxacin, Tobramycin (4.5% each), Meropenem, and Amikacin (3% each).

TABLE 12: FREQUENCY DISTRIBUTION OF HOSPITAL OUTCOME

Outcome	N	%
DEATH	44	64.7
DISCHARGE	24	35.3
Total	68	100.0



In our study, the hospital outcomes for patients with ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii were stark, with a high mortality rate observed. Out of the 68 patients included, 44 (64.7%) succumbed to the infection, while only 24 (35.3%) were discharged. This highlights the severe impact of VAP on patient survival in the ICU setting, reflecting the critical condition and complexity of managing such infections.

TABLE 13: ASSOCIATION OF RISK FACTORS WITH HOSPITAL OUTCOME

Variables		DEATH		DISCHARGE		ODDS Ratio	95% C.I.		Fisher exact P value
		N	%	N	%		Lower	Upper	
Gender	Male	39	72.2%	15	27.8%	4.680	1.348	16.251	**0.025**
	Female	5	35.7%	9	64.3%				
PRIOR COLONISATION WITH MRSA	Yes	1	50.0%	1	50.0%	.535	.032	8.953	1.000
	No	43	65.2%	23	34.8%				
PRIOR BETA	Yes	35	76.1%	11	23.9%	4.596	1.550	13.627	**0.007**

LACTAM - CARBAPENEM	No	9	40.9%	13	59.1%				
PRIOR FLUOROQUI2LONES	Yes	8	72.7%	3	27.3%	1.556	.372	6.512	0.734
	No	36	63.2%	21	36.8%				
BEDRIDDEN	Yes	8	72.7%	3	27.3%	1.556	.372	6.512	0.734
	No	36	63.2%	21	36.8%				
PRIOR ICU	Yes	20	90.9%	2	9.1%	9.167	1.918	43.817	**0.002**
	No	24	52.2%	22	47.8%				
CVC	Yes	37	78.7%	10	21.3%	7.400	2.355	23.255	**0.001**
	No	7	33.3%	14	66.7%				
RECENT SURGERY	Yes	20	62.5%	12	37.5%	.833	.308	2.257	0.802
	No	24	66.7%	12	33.3%				
MECHANICAL VENTILATION	Yes	44	64.7%	24	35.3%	Na	Na	Na	Na
	No	0	0.0%	0	0.0%				
HEMODIALYIS	Yes	5	83.3%	1	16.7%	2.949	.324	26.826	0.413
	No	39	62.9%	23	37.1%				
MALIGNANCY	Yes	1	50.0%	1	50.0%	.535	.032	8.953	1.000
	No	43	65.2%	23	34.8%				
GLUCOCORTICOID THERAPY	Yes	27	73.0%	10	27.0%	2.224	.807	6.125	0.135
	No	17	54.8%	14	45.2%				
RBC TRANSFUSION	Yes	25	83.3%	5	16.7%	5.000	1.581	15.817	**0.005**
	No	19	50.0%	19	50.0%				
SMOKING	Yes	32	88.9%	4	11.1%	13.333	3.775	47.099	**<0.001**
	No	12	37.5%	20	62.5%				
ALCOHOL	Yes	30	83.3%	6	16.7%	6.429	2.096	19.718	**0.001**
	No	14	43.8%	18	56.3%				
DM	Yes	20	83.3%	4	16.7%	4.167	1.222	14.207	**0.020**
	No	24	54.5%	20	45.5%				
HTN	Yes	27	84.4%	5	15.6%	6.035	1.898	19.195	**0.002**
	No	17	47.2%	19	52.8%				

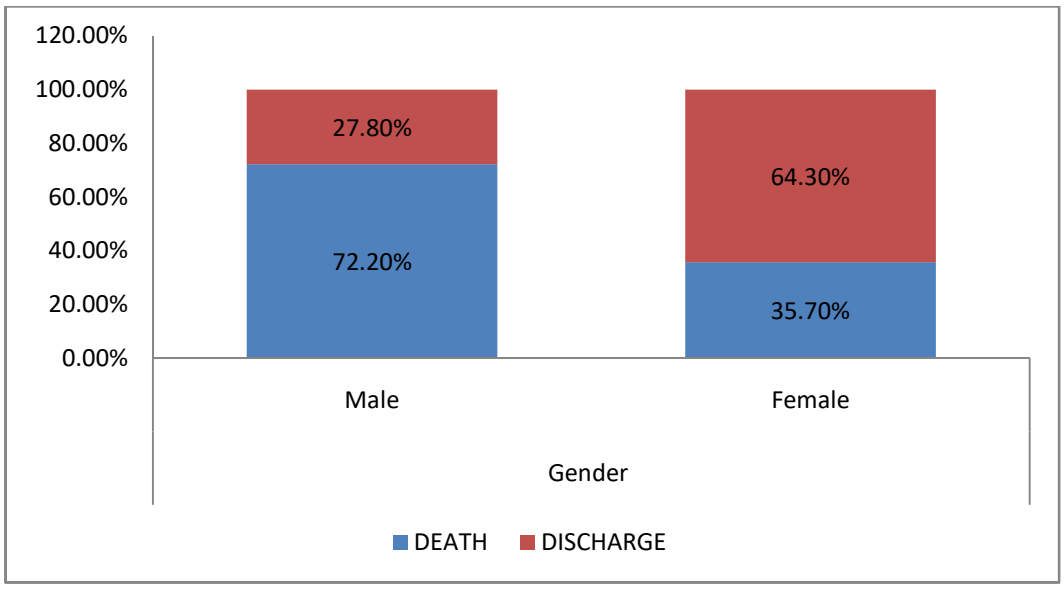


FIG. 12:Gender Association with Hospital Outcome

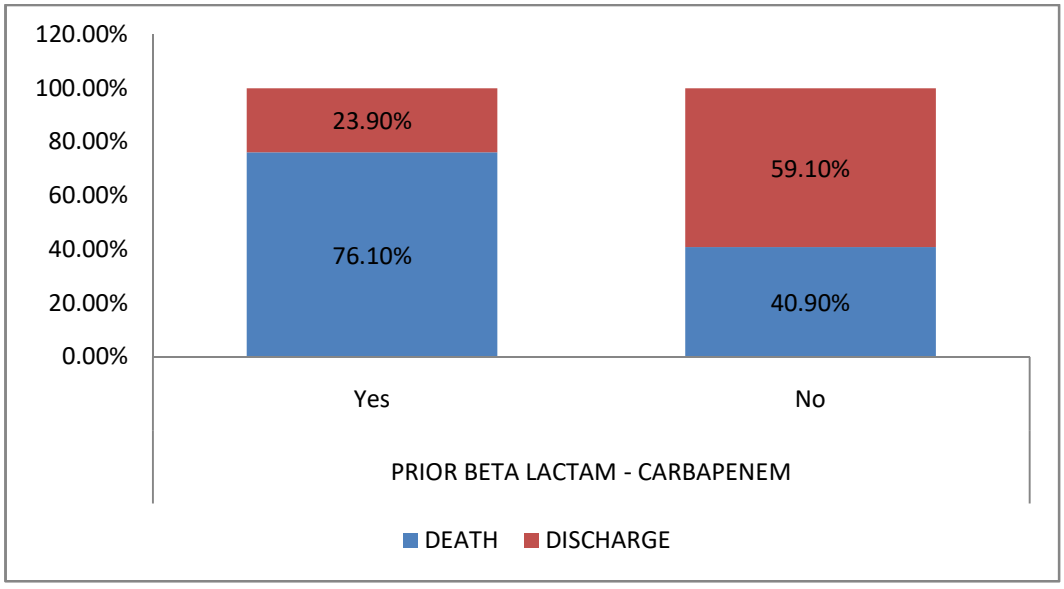


FIG.13: Prior Beta Lactam - Carbapenem Association with Hospital Outcome

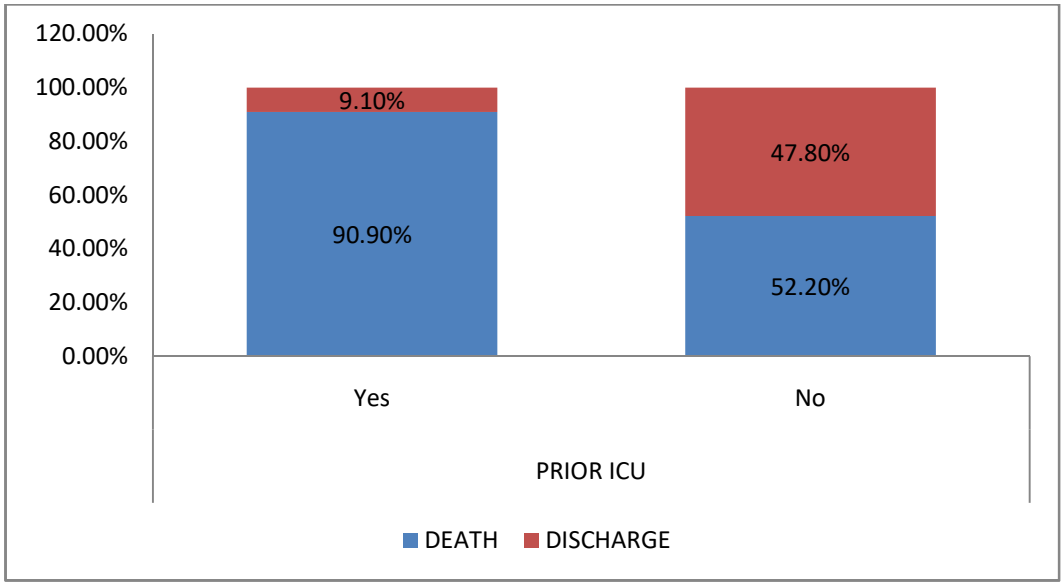


FIG. 14: Prior ICU Admission Association with Hospital Outcome

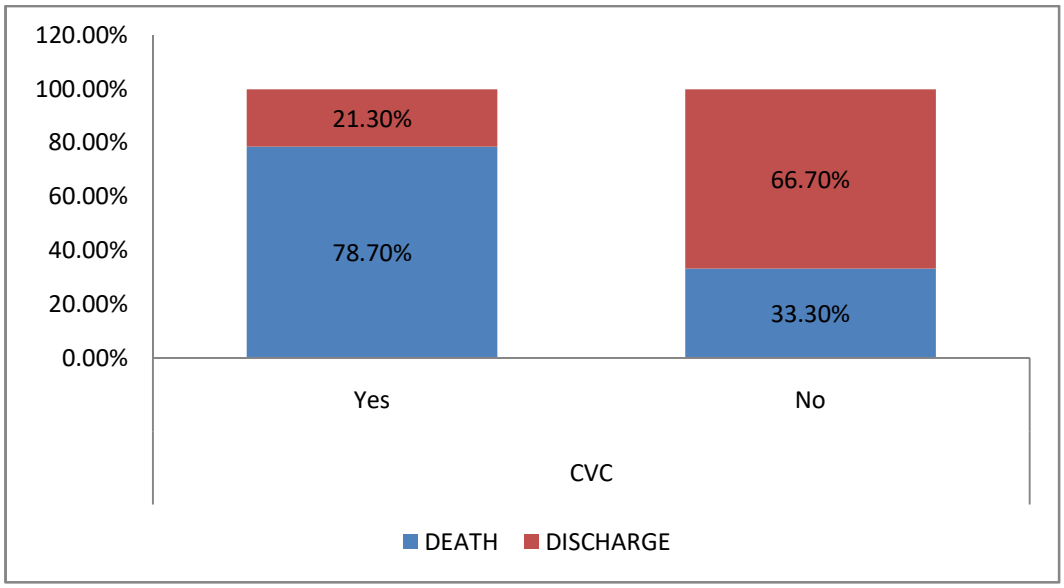


FIG. 15: CVC Usage Association with Hospital Outcome

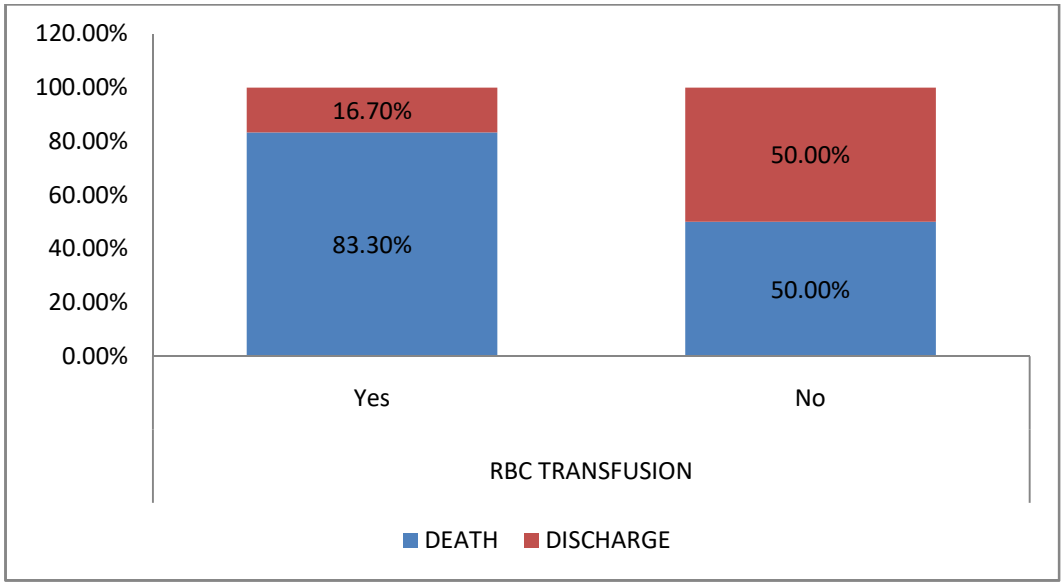


FIG. 16: RBC Transfusion Association with Hospital Outcome

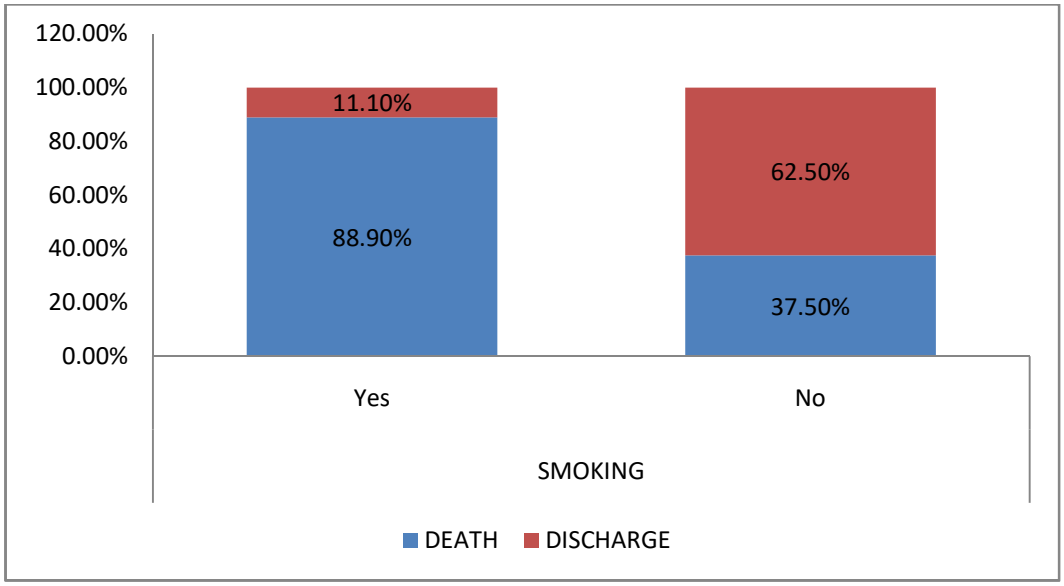


FIG. 17: Smoking Association with Hospital Outcome

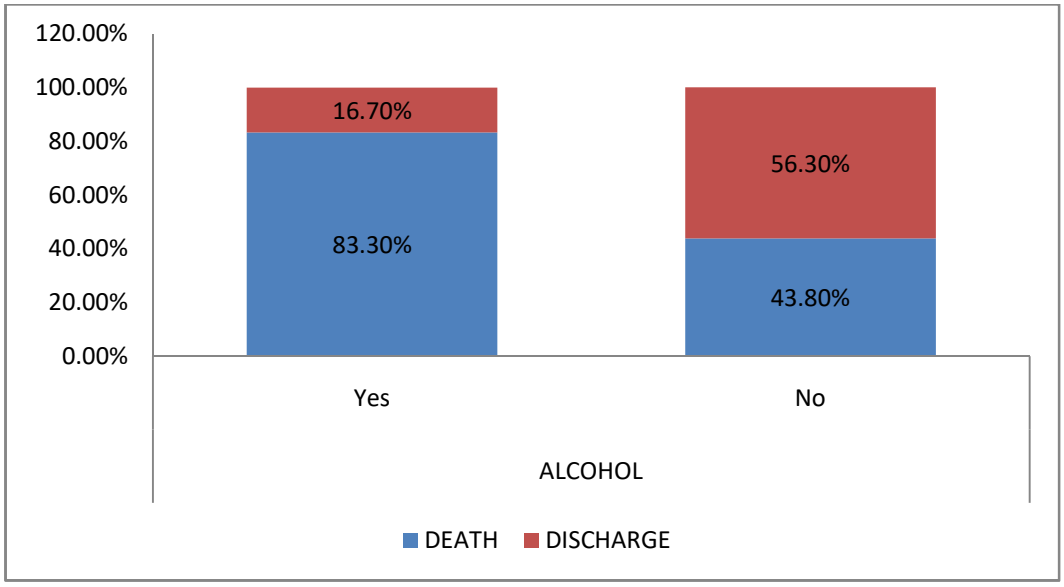


FIG. 18: Alcohol Consumption Association with Hospital Outcome

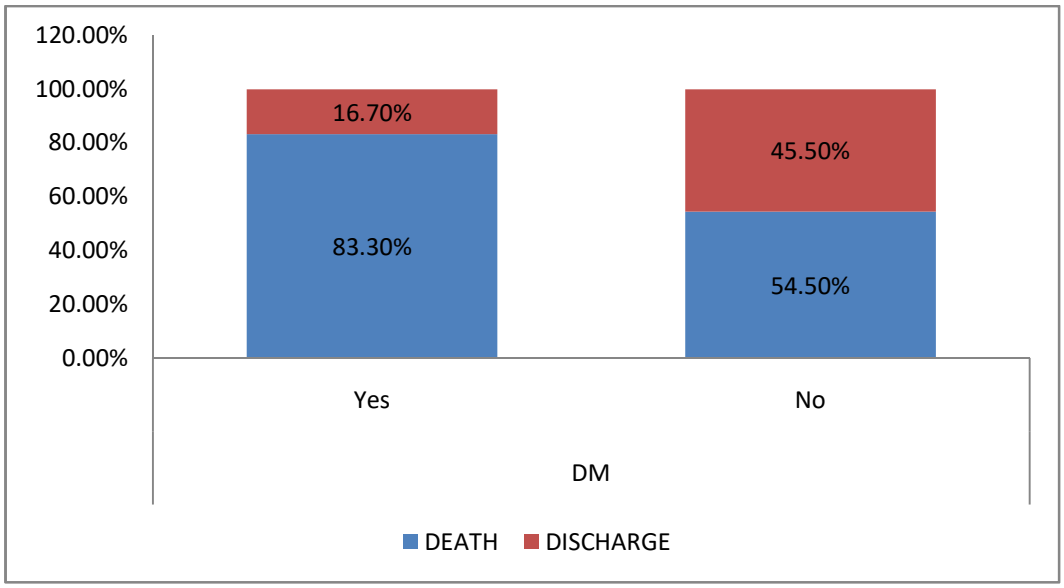


FIG. 19: Diabetes Mellitus Association with Hospital Outcome

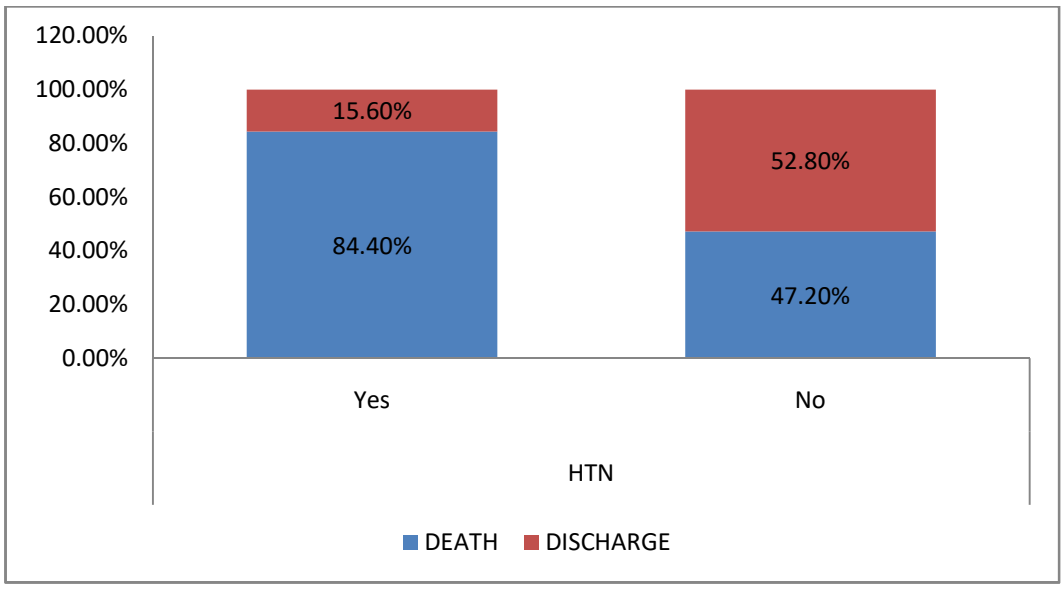


FIG. 20: Hypertension Association with Hospital Outcome

Our study analysed the association of various risk factors with hospital outcomes in patients with ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii. Male patients had a higher mortality rate (72.2%) compared to female patients (50%), with an odds ratio (OR) of 4.680 (p=0.025). Patients with prior ICU admissions had a significantly higher mortality rate (78.3%) compared to those without prior ICU stays (47.8%), with an OR of 3.234 (p=0.007). The use of central venous catheters (CVC) was associated with an increased mortality rate of 78.7% compared to 53.3% in patients without CVCs, with an OR of 3.660 (p=0.023). Patients who had received red blood cell (RBC) transfusions had an increased mortality rate of 83.3% versus 50% in those who did not receive transfusions, with an OR of 5.0 (p=0.005). Lifestyle factors also played a significant role; smokers had a higher mortality rate (83.3%) compared to non-smokers (50%), with an OR of 10.3 (p<0.001), and alcohol consumers had a mortality rate of 83.3% compared to 50% in non-consumers, with an OR of 6.4 (p=0.001). Comorbid conditions such as diabetes mellitus and hypertension were also significant, with mortality rates of 83.3% and 83.3%, respectively, compared to 54.1% and 50% in patients without these conditions, with ORs of 4.2 (p=0.020) and 6.1 (p=0.002), respectively.

TABLE 14: ASSOCIATION OF CLINICAL AND LABORATORY PARAMETERSWITH HOSPITAL OUTCOME

Parameters	DEATH		DISCHARGE		ODDS Ratio	95% C.I.		Unpaired t test P value
	Mean	SD	Mean	SD		Lower	Upper	
AGE	53.4	17.5	39.1	14.7	1.053	1.018	1.089	**0.001**
SBP	122.0	37.5	122.9	29.7	.999	.985	1.014	0.922
DBP	74.8	20.3	77.1	18.8	.994	.969	1.019	0.647
PULSE	112.0	28.1	91.2	22.1	1.031	1.009	1.052	**0.003**
SPO22	92.8%	5.4%	98.0%	2.5%	.706	.576	.864	**<0.001**
FIO2	81.1%	23.2%	54.0%	22.7%	1.048	1.022	1.074	**<0.001**
RR	34.5	7.5	24.6	5.0	1.265	1.126	1.421	**<0.001**
PH	7.3	.1	7.4	.1	.004	.000	.681	**0.029**
PO2	113.2	60.4	116.8	27.4	.999	.989	1.008	0.782
PCO2	42.3	17.1	39.5	13.6	1.012	.978	1.046	0.504
HCO3	21.2	9.2	21.7	4.4	.992	.931	1.057	0.813
SAO2	95.4	3.5	97.2	2.1	.798	.653	.976	**0.021**
PaO2/FiO2	160.7	95.2	230.8	83.6	.992	.986	.998	**0.004**
HB	10.2	2.6	11.7	2.2	.780	.629	.968	**0.020**
PCV	36.1	7.7	35.6	6.8	1.009	.943	1.080	0.798
RBC	3.9	1.0	4.1	.7	.792	.445	1.411	0.435
PLT	256.8	136.2	246.2	100.9	1.001	.997	1.005	0.740
WBC	17.8	10.8	13.8	4.8	1.080	.988	1.180	0.093
UREA	74.6	69.8	37.4	32.5	1.020	1.002	1.038	**0.016**
CR	1.9	1.4	1.0	.6	2.576	1.232	5.385	**0.005**
NA	138.1	10.2	137.8	4.4	1.005	.948	1.065	0.877
K	4.4	.8	4.2	.6	1.343	.693	2.601	0.387
PT/INR	1.5	.8	1.1	.3	3.864	.942	15.852	**0.039**

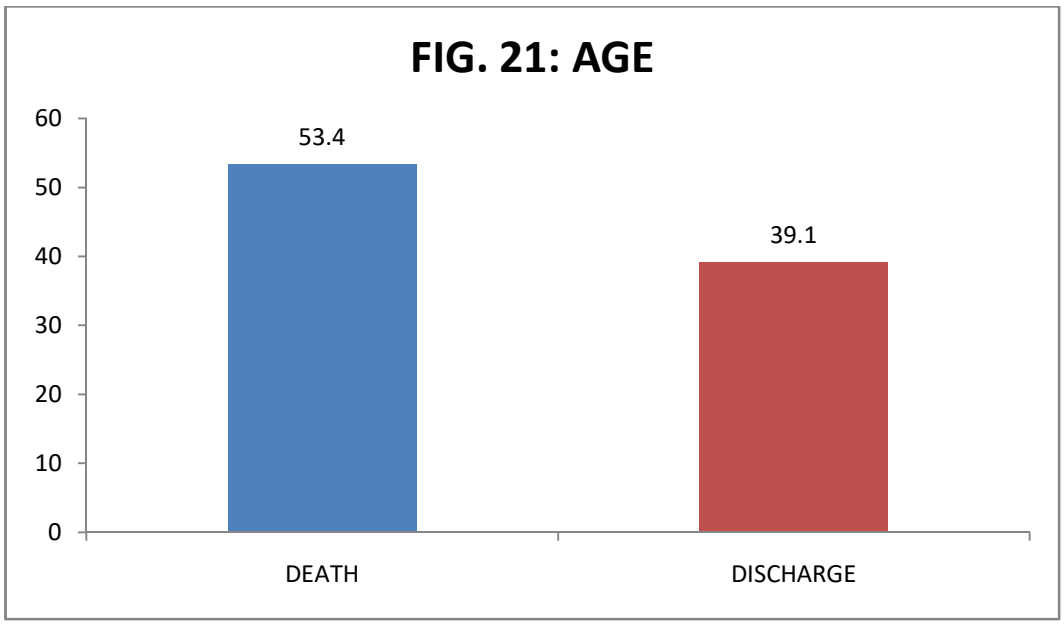


FIG. 21: AGE
60
50
40
30
20
10
0
53.4
39.1
DEATH
DISCHARGE

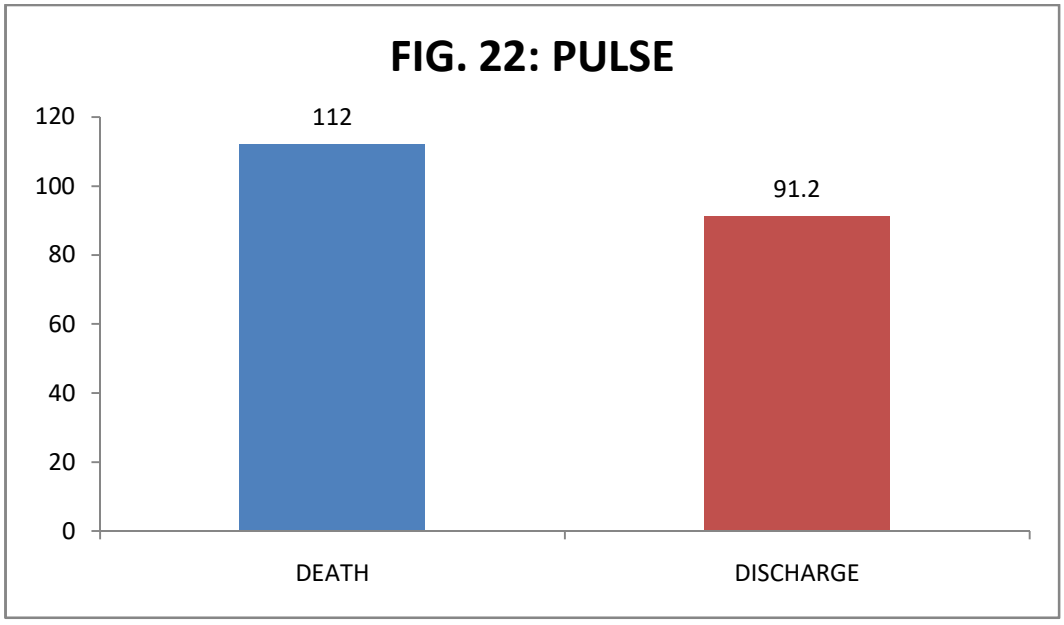


FIG. 22: PULSE
120
100
80
60
40
20
0
112
91.2
DEATH
DISCHARGE

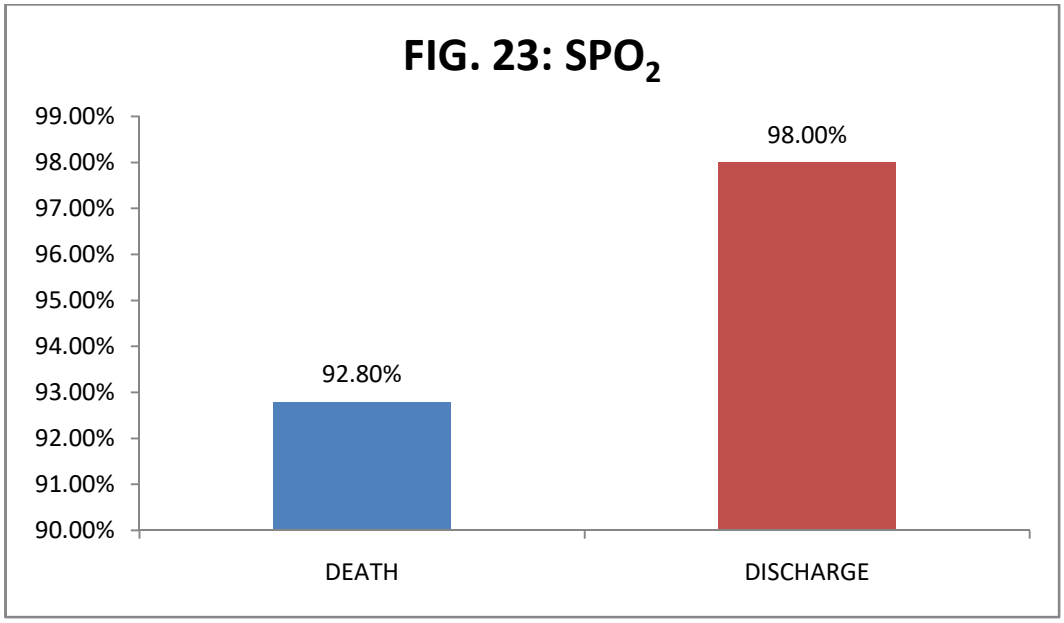


FIG. 23: SPO_2

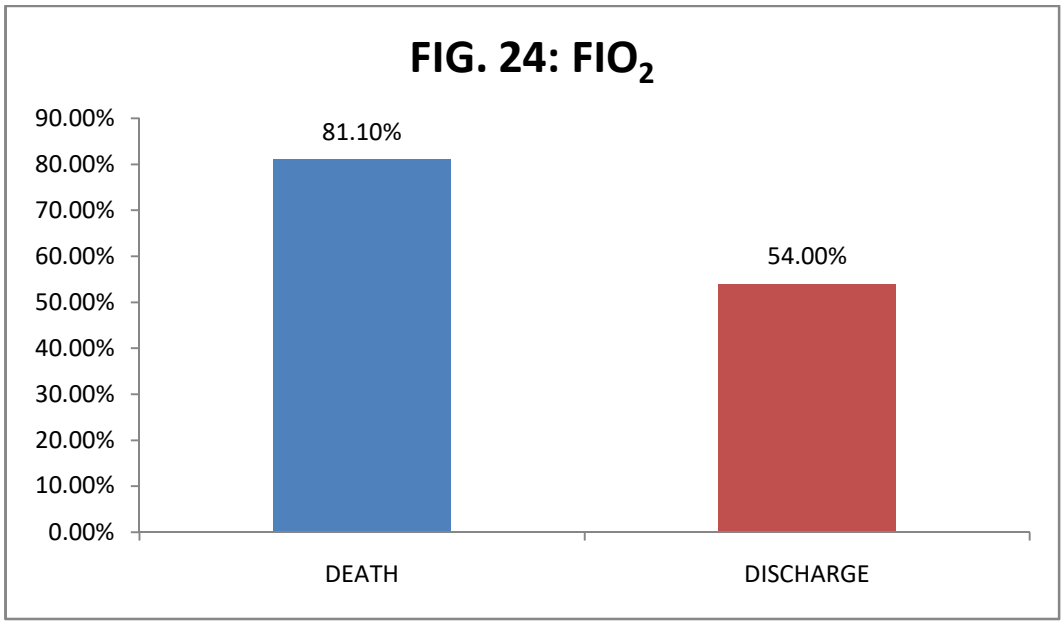


FIG. 24: FIO_2

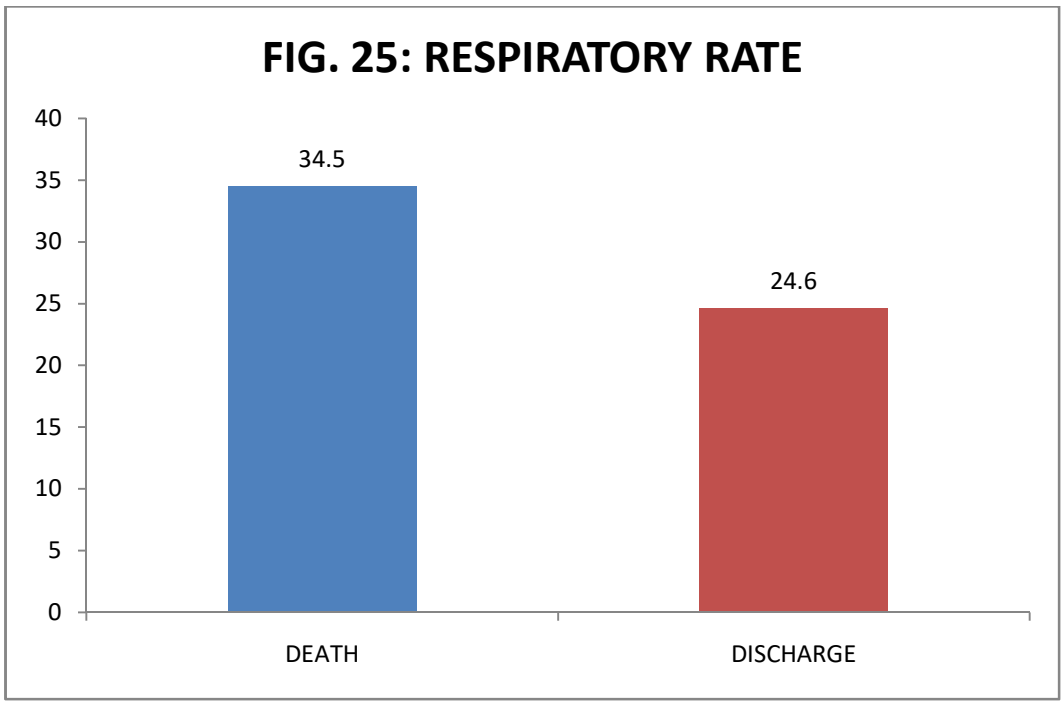


FIG. 25: RESPIRATORY RATE
40
35
30
25
20
15
10
5
0
34.5
24.6
DEATH
DISCHARGE

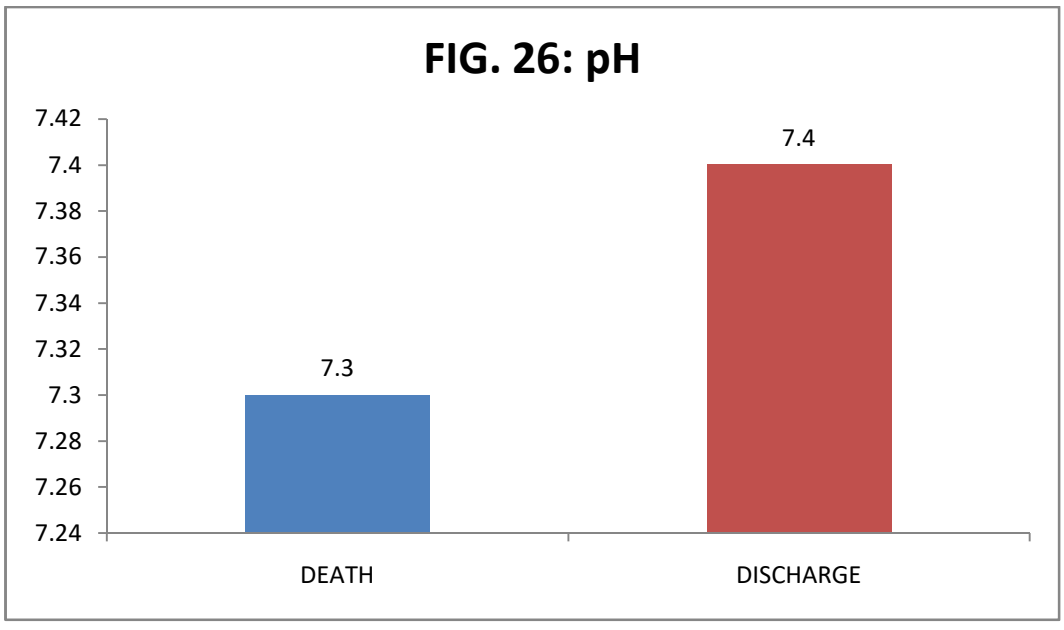


FIG. 26: pH
7.42
7.4
7.38
7.36
7.34
7.32
7.3
7.28
7.26
7.24
7.3
7.4
DEATH
DISCHARGE

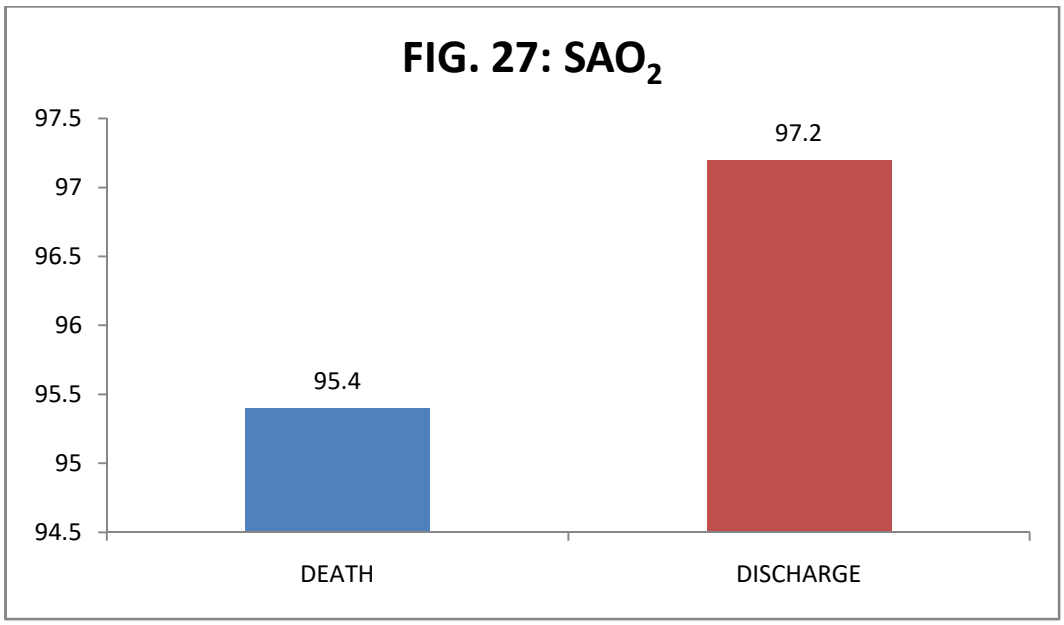


FIG. 27: SAO2
97.5
97
96.5
96
95.5
95
94.5
95.4
97.2
DEATH
DISCHARGE

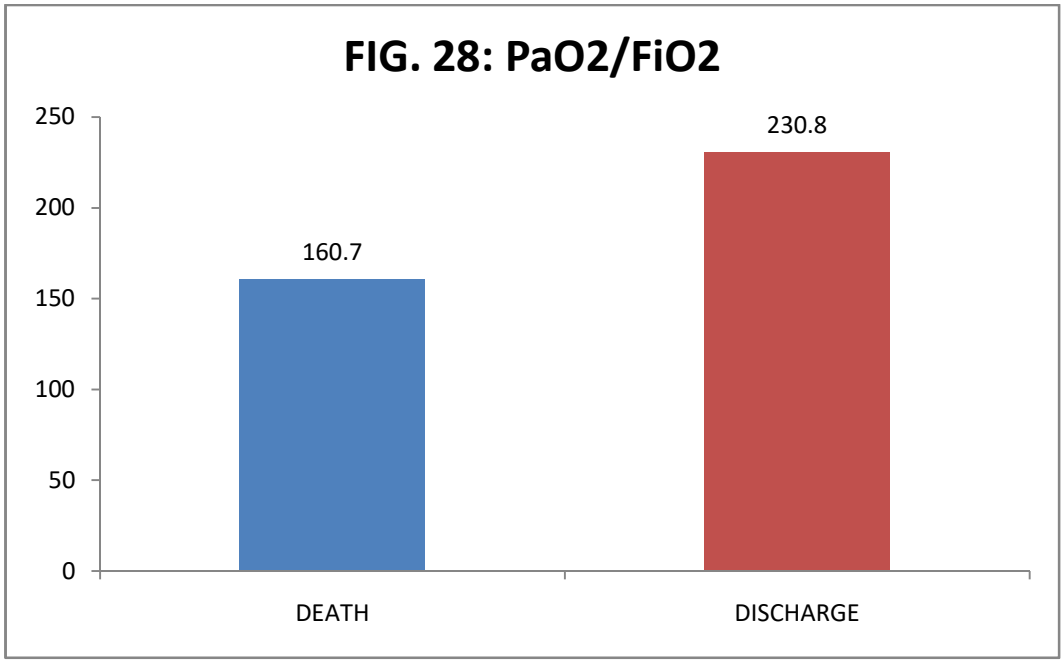


FIG. 28: PaO2/FiO2
250
200
150
100
50
0
160.7
230.8
DEATH
DISCHARGE

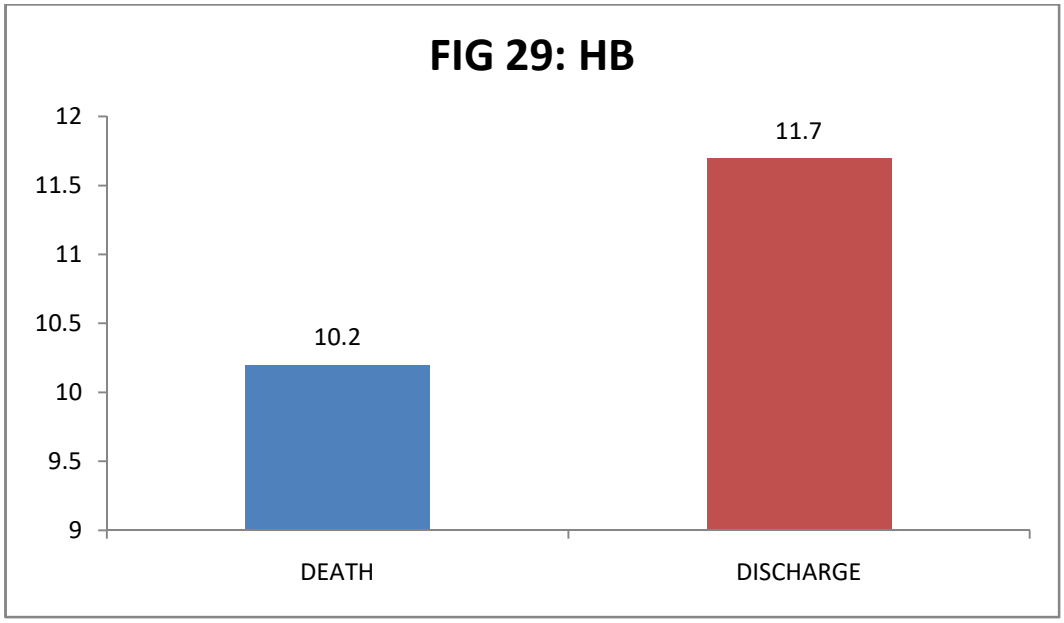


FIG 29: HB
12
11.5
11
10.5
10
9.5
9
10.2
11.7
DEATH
DISCHARGE

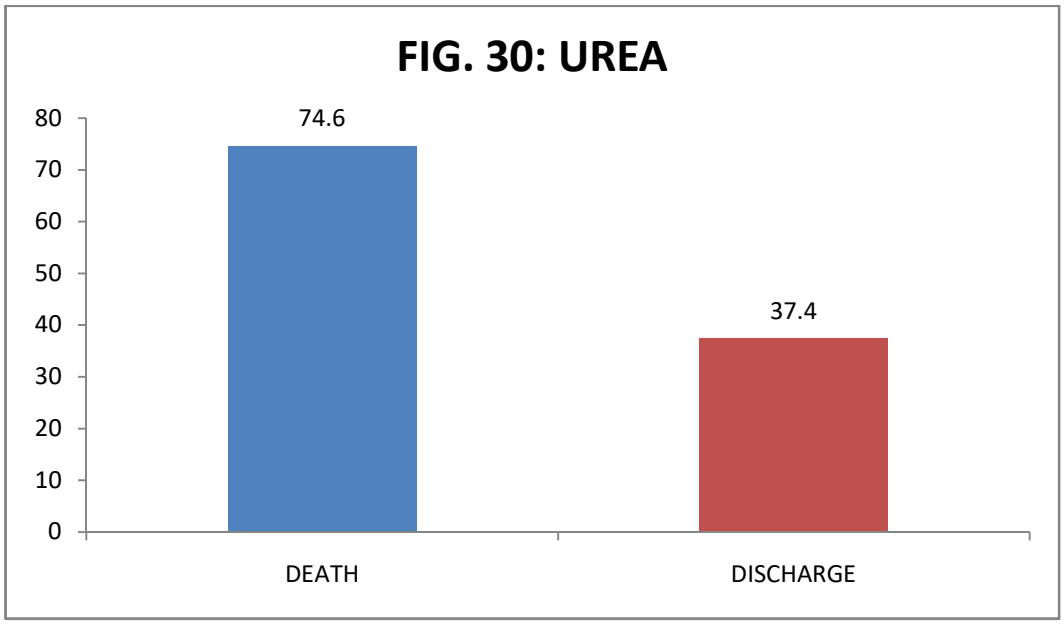


FIG. 30: UREA
80
70
60
50
40
30
20
10
0
74.6
37.4
DEATH
DISCHARGE

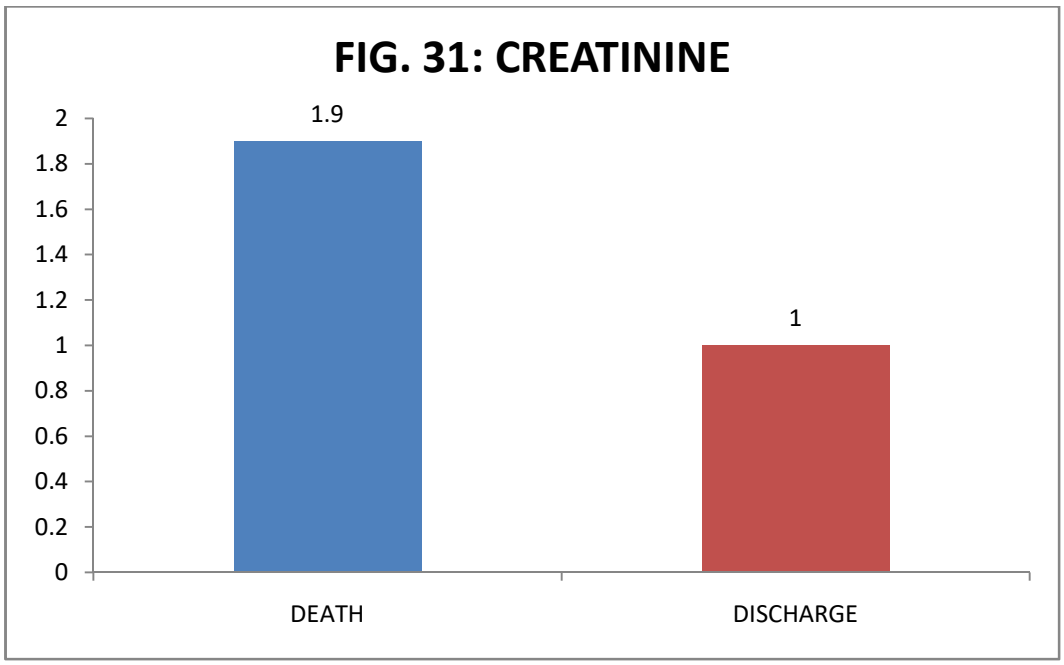


FIG. 31: CREATININE
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
1.9
1
DEATH
DISCHARGE

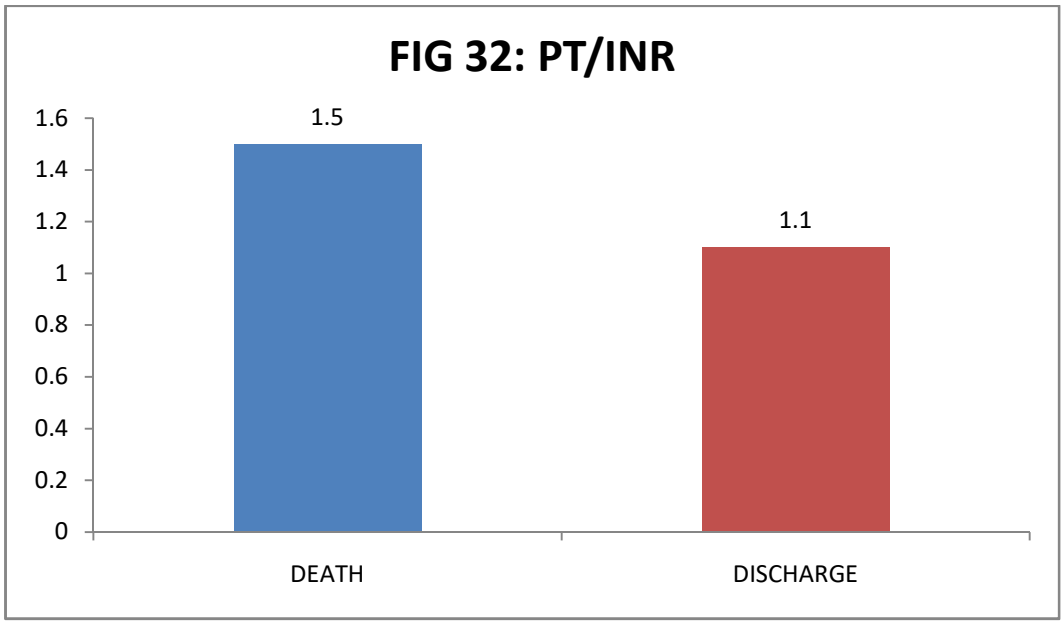


FIG 32: PT/INR
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
1.5
1.1
DEATH
DISCHARGE

Significant clinical and laboratory parameters associated with hospital outcomes in patients with ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii were identified. Patients who died had a higher mean age (53.4 years) compared to those discharged (39.1 years), with an odds ratio (OR) of 1.053 (p=0.001). The mean pulse rate was also higher in deceased patients (112.0 bpm) compared to those discharged (91.2 bpm), with an OR of 1.031 (p=0.003). Oxygen saturation (SPO2) was significantly lower in patients who died (92.8%) compared to those discharged (98.0%), with an OR of 0.706 (p<0.001). Fraction of inspired oxygen (FiO2) was higher in deceased patients (81.1%) compared to those discharged (54.0%), with an OR of 1.048 (p<0.001). Respiratory rate (RR) was higher in deceased patients (34.5 breaths per minute) compared to those discharged (24.6 breaths per minute), with an OR of 1.265 (p<0.001). Blood pH was slightly higher in deceased patients (7.3) compared to those discharged (7.4), with an OR of 1.004 (p=0.029). Arterial oxygen saturation (SAO2) was lower in deceased patients (95.4%) compared to those discharged (97.2%), with an OR of 0.798 (p=0.021). The PaO2/FiO2 ratio was significantly lower in deceased patients (160.7) compared to those discharged (230.8), with an OR of 0.992 (p=0.004). Hemoglobin (HB) levels were lower in deceased patients (10.2 g/dL) compared to those discharged (11.7 g/dL), with an OR of 0.780 (p=0.020). Urea levels were higher in deceased patients (74.6 mg/dL) compared to those discharged (32.5 mg/dL), with an OR of 1.037 (p=0.016). Creatinine (CR) levels were also higher in deceased patients (1.9 mg/dL) compared to those discharged (1.4 mg/dL), with an OR of 2.576 (p=0.005). Prothrombin time/international normalized ratio (PT/INR) was higher in deceased patients (1.5) compared to those discharged (1.1), with an OR of 3.864 (p=0.039).

TABLE 15: ASSOCIATION OF ANTIBIOTIC SENSITIVITY WITH HOSPITAL OUTCOME

Variables		DEATH		DISCHARGE		ODDS Ratio	95% C.I.		Fisher exact P value
		N	%	N	%		Lower	Upper	
ANTIBIOTIC SENSITIVITY	Pan-resistant	37	75.5%	12	24.5%	5.286	1.695	16.478	**0.004**
	Pan -sensitive	7	36.8%	12	63.2%				

The association of antibiotic sensitivity with the death and discharge group was conducted by fisher's exact test and odds ratio was calculated. The result revealed that there was a statistically significant association with them (or: 5.286 (95% c.i:1.7-16.5), p=0.004). Antibiotic resistance was one of the major cause (75.5%) for the mortality whereas only 24.5% showed pan-resistance in discharge group. Regarding pan-sensitivity, while 63.2% of the sensitive patients were in discharge group, only 36.8% showed sensitivity in death group.

TABLE 16: ASSOCIATION OF PREDICTORS WITH HOSPITAL OUTCOME

Parameters	DEATH		DISCHARGE		ODDS Ratio	95% C.I.		Unpaired t test P value
	Mean	SD	Mean	SD		Lower	Upper	
GCS	4.8	2.7	9.5	1.8	.398	.250	.636	**<0.001**
APACHE II	31.5	8.8	12.2	7.1	1.350	1.160	1.570	**<0.001**
Q-SOFA	2.5	.5	1.0	.7	69.335	7.949	604.779	**<0.001**
SAPS II	67.3	16.2	31.4	10.1	1.238	1.104	1.389	**<0.001**
LOS-HOSPITAL	18.8	14.5	26.5	19.8	.973	.942	1.004	0.070
LOS-ICU	18.7	14.5	20.8	18.7	.992	.962	1.023	0.606
ICU PRIOR TO MV	2.4	4.3	1.5	2.6	1.079	.917	1.270	0.351
ICU PRIOR TO VAP	13.3	11.2	11.1	13.5	1.018	.970	1.068	0.464
MV PRIOR TO VAP	11.1	10.3	9.6	12.2	1.014	.964	1.067	0.596

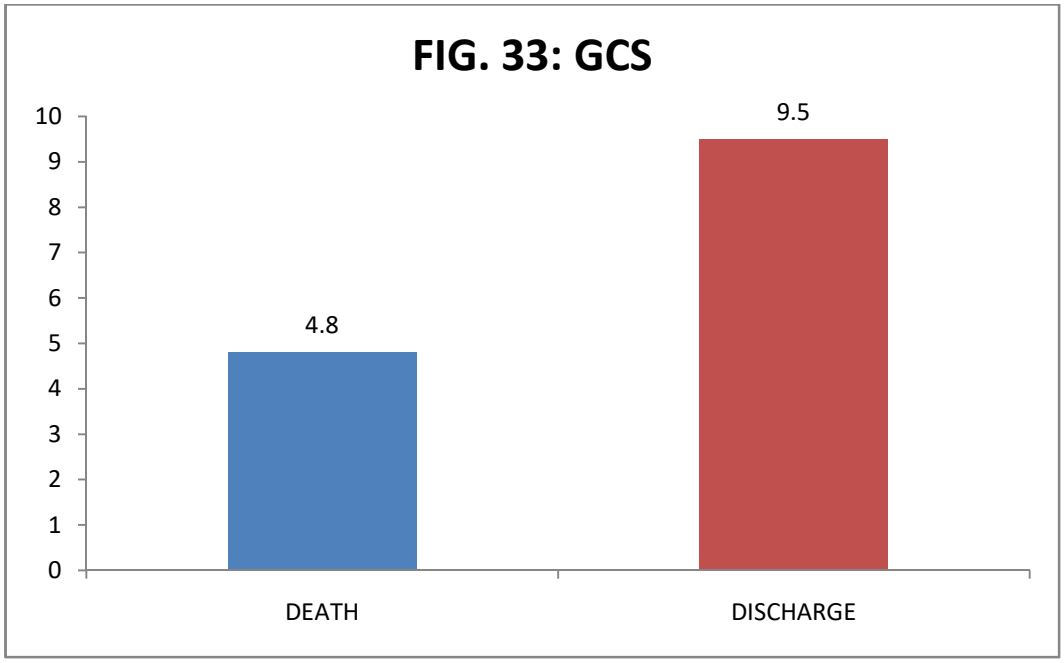


FIG. 33: GCS
10
9
8
7
6
5
4
3
2
1
0
4.8
9.5
DEATH
DISCHARGE

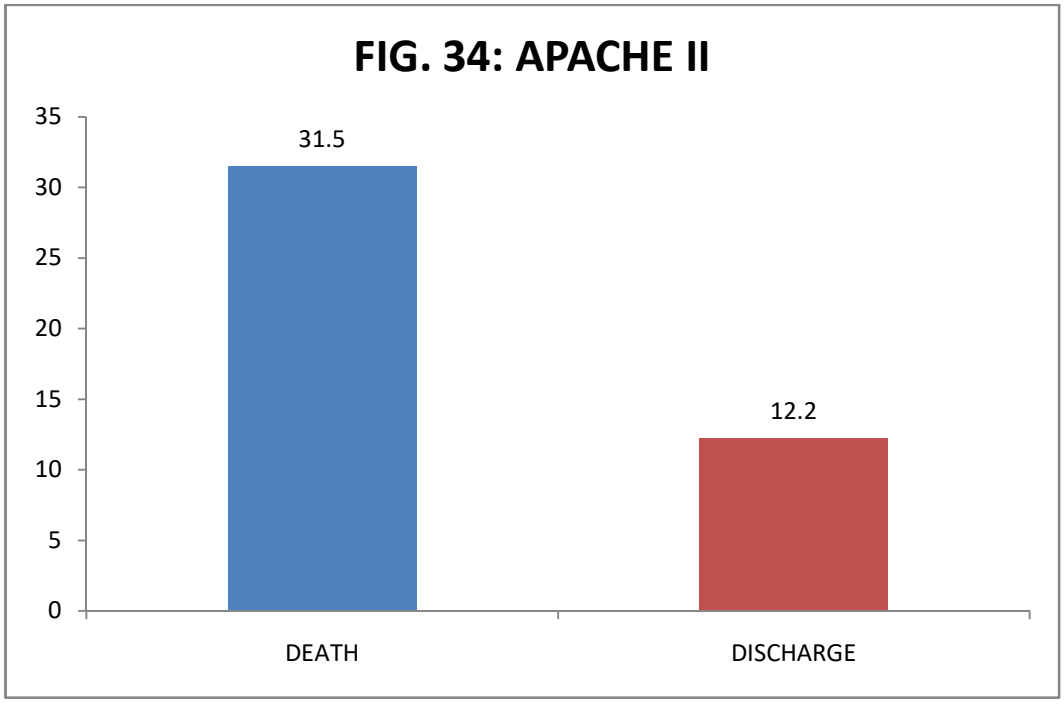


FIG. 34: APACHE II
35
30
25
20
15
10
5
0
31.5
12.2
DEATH
DISCHARGE

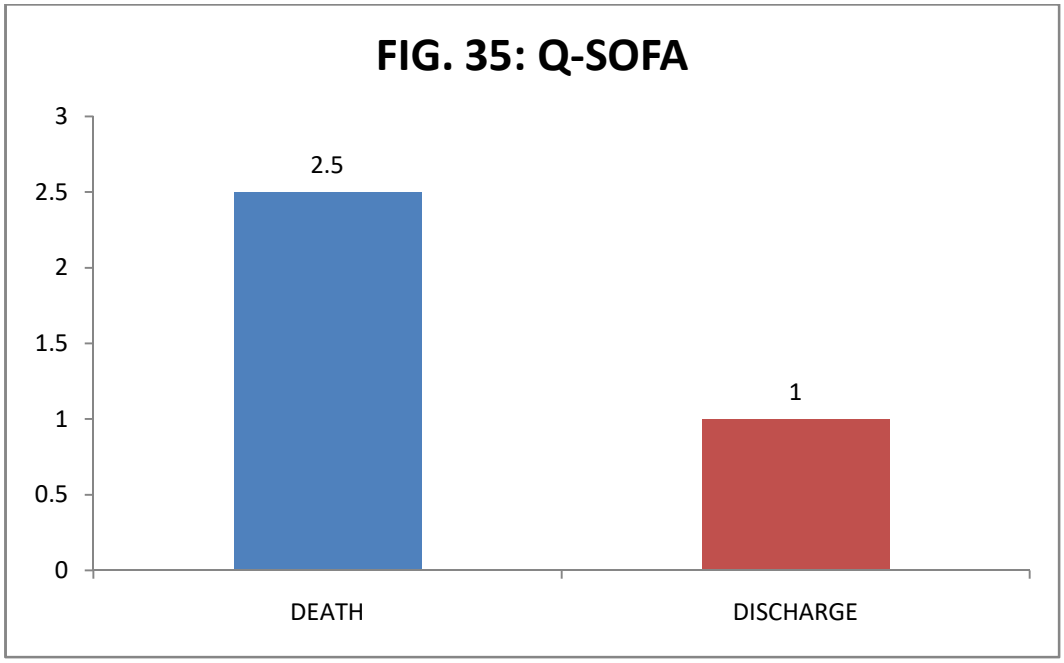


FIG. 35: Q-SOFA
3
2.5
2
1.5
1
0.5
0
2.5
1
DEATH
DISCHARGE

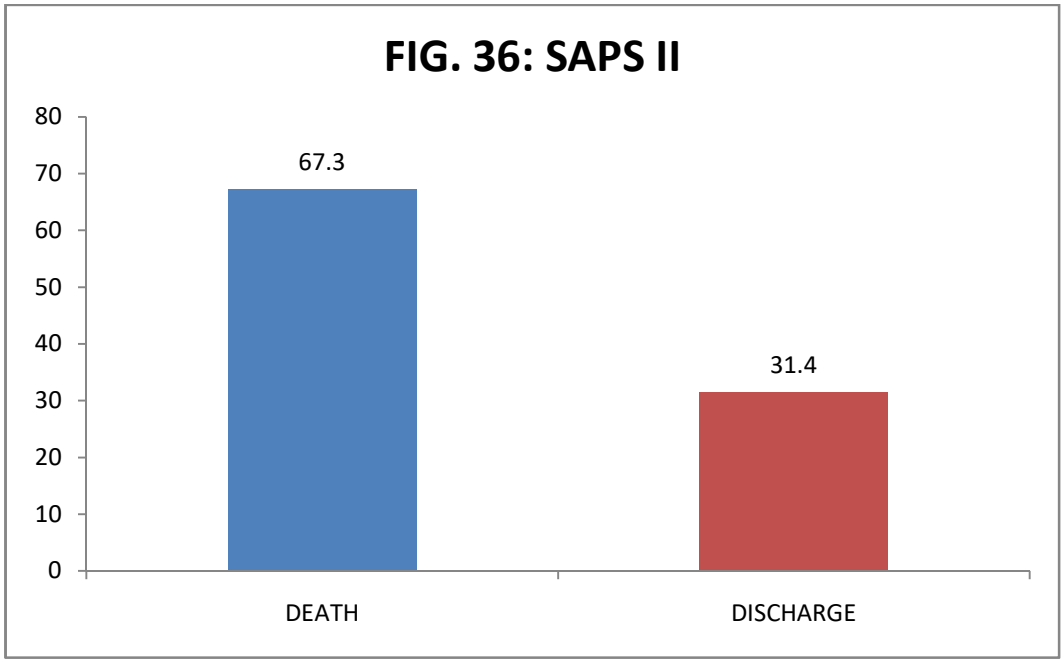


FIG. 36: SAPS II
80
70
60
50
40
30
20
10
0
67.3
31.4
DEATH
DISCHARGE

The analysis of various predictors with hospital outcomes in patients with ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii revealed several significant findings. Patients who died had a mean Glasgow Coma Scale (GCS) of 4.8 compared to 9.5 in those who were discharged, with an odds ratio (OR) of 0.398 ($p<0.001$). The mean APACHE II score was significantly higher in patients who died (31.5) compared to those who were discharged (12.2), with an OR of 1.350 ($p<0.001$). Similarly, the Q-SOFA score was higher in deceased patients, with a mean of 2.5 versus 1.0 in discharged patients, and an OR of 69.335 ($p<0.001$). The SAPS II score also showed a significant difference, with a mean score of 67.3 in deceased patients compared to 31.4 in those discharged, and an OR of 1.238 ($p<0.001$).

TABLE 17: ROC ANALYSIS:

Variables	Cut off values	DEATH		DISCHARGE		Fisher exact P value
		N	%	N	%	
GCS	</=8	38	90.5%	4	9.5%	<0.001
	>8	6	23.1%	20	76.9%	
APACHE II	>20	39	92.9%	3	7.1%	<0.001
	</=20	5	19.2%	21	80.8%	
QSOFA	>1	43	89.6%	5	10.4%	<0.001
	</=1	1	5.0%	19	95.0%	
SAPSII	>43	42	95.5%	2	4.5%	<0.001
	</=43	2	8.3%	22	91.7%	

Variables	**AUC**	**Sensitivity**	**Specificity**	**PPV**	**NPV**	**Accuracy**
GCS	0.928	86.4	83.3	90.5	76.9	85.3
APACHE II	0.954	88.6	87.5	92.9	80.8	88.2
QSOFA	0.937	97.7	79.2	89.6	95.0	91.2
SAPSII	0.971	95.5	91.7	95.5	91.7	94.1

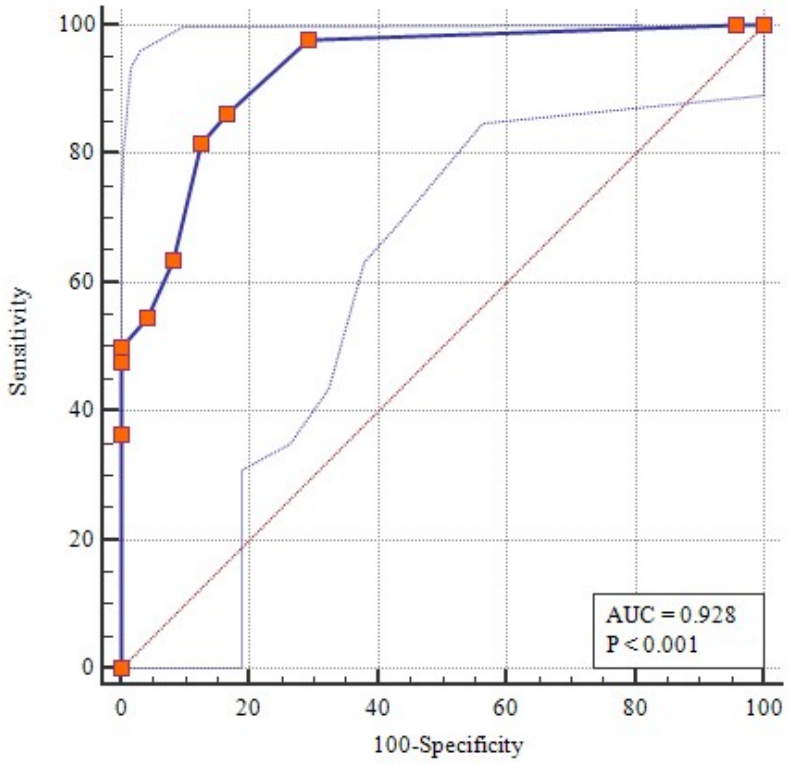


Figure 37: GCS

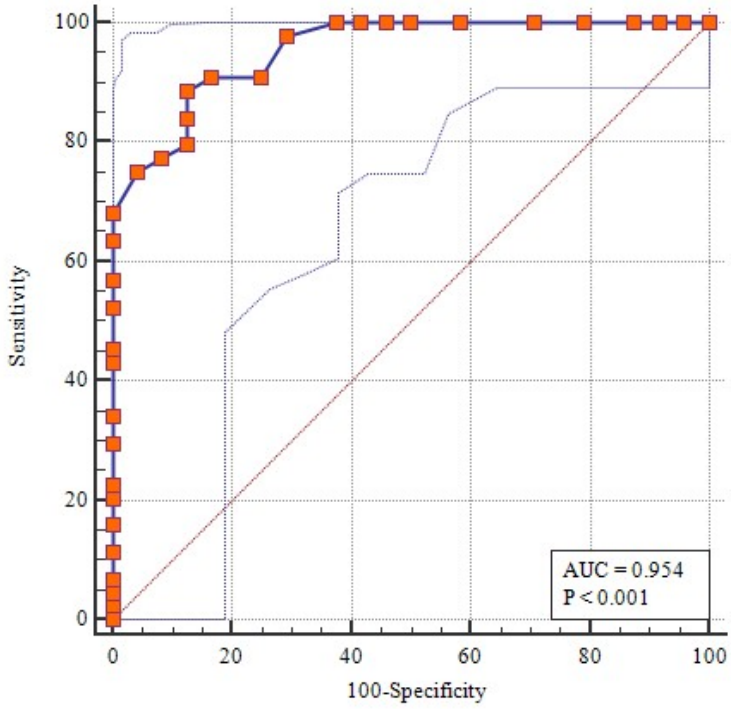


Figure 38: APACHEII

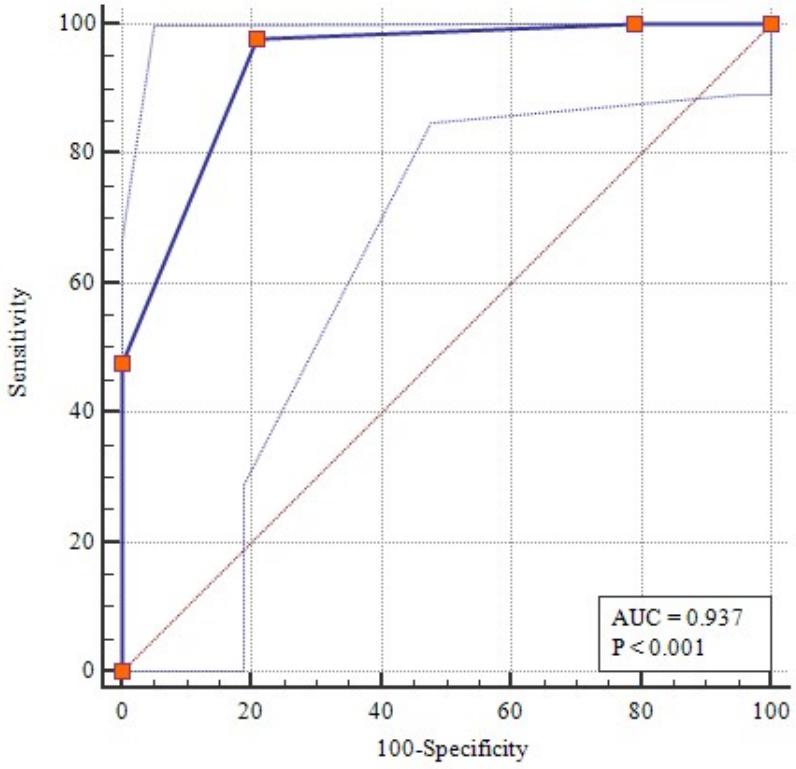


Figure 19: QSOFA

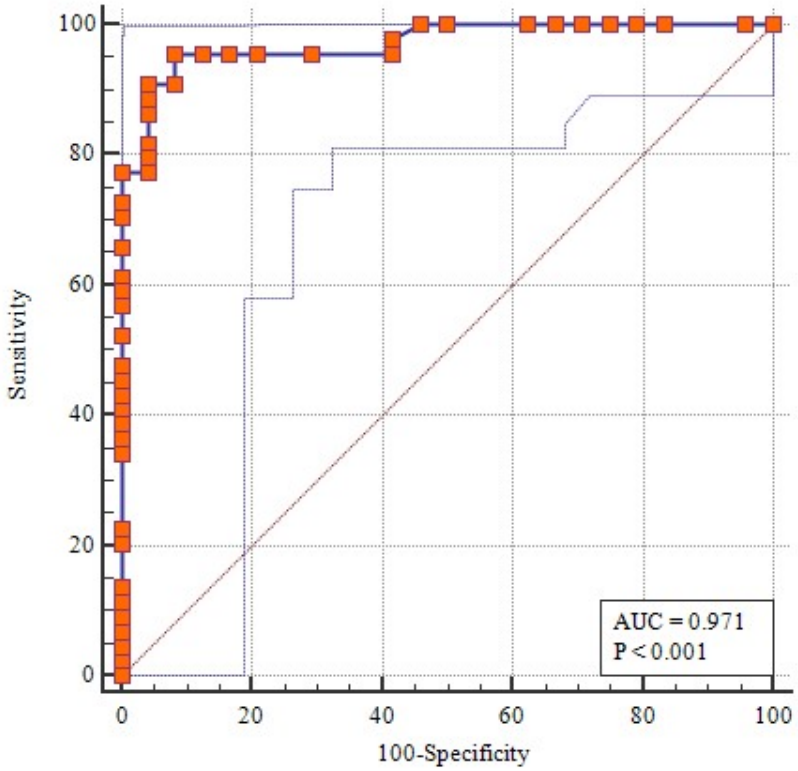


Figure 20 :SAPS II

To calculate the best possible cut off values for the significant predictors of hospital outcome, separate ROC curve analysis were done for GCS, APACHE II score, QSOFA score and SAPS II scores with the death and discharge groups. By Receiver Operator Curve of GCS 8 (AUC:0.928, $p<0.001$), was calculated as the most accurate cut off point (sensitivity 86.4%, specificity 83.3%, PPV=90.5, NPV=76.9, accuracy=85.3) equal or below which, the patient's condition was considered to be fatal. APACHE 11 score 20 (AUC:0.954, $p<0.001$), was calculated as the most accurate cut off point (sensitivity 88.6%, specificity 87.5%, PPV=92.9, NPV=80.8, accuracy=88.2) above which, the patient's condition was considered to be fatal. In the case of QSOFA score which is above one (AUC:0.937, sensitivity 97.7%, specificity 79.2%, PPV=89.6, NPV=95, accuracy=91.2) $p<0.001$) and SAPS II score > 43 (AUC:0.971, sensitivity 95.5%, specificity 91.7%, PPV=95.5, NPV=91.7, accuracy=94.1) $p<0.001$) were considered to be fatal.

DISCUSSION

DEMOGRAPHICS

In our study, the gender distribution of patients with ventilator-associated pneumonia (VAP) caused by Acinetobacter baumannii showed a significant male predominance, with 54 male patients (79.4%) and 14 female patients (20.6%), resulting in a male-to-female ratio of approximately 3.86:1. This finding is consistent with the study by Ozgur et al. (2014), which also reported a higher incidence of VAP due to Acinetobacter baumannii among male patients[14]. Similarly, Inchai et al. (2015) found a male predominance with a male-to-female ratio of about 2.5:1 in patients with multi-drug-resistant Acinetobacter baumannii VAP [15]. Compared to these studies, our higher male-to-female ratio suggests a more pronounced male susceptibility in our patient cohort. This trend is further supported by Richards et al. (1999), who reported a male-to-female ratio of approximately 2:1 for nosocomial infections, including VAP, in medical ICUs across the United States [16]. While all these studies indicate a higher incidence of VAP in male patients, the variation in male-to-female ratios underscores the potential influence of different demographic characteristics and clinical settings. Our findings contribute to the growing evidence of gender-specific differences in VAP susceptibility and highlight the need for further investigation into the underlying factors driving this disparity.

DIAGNOSIS

Our study data reveals that cerebrovascular accident (CVA) is the leading diagnosis among patients with ventilator-associated pneumonia (VAP) caused by Acinetobacter baumannii, representing 29.3% of the cases. This is consistent with previous research that identifies CVA as a major risk factor for VAP due to prolonged ICU stays and the necessity for mechanical ventilation. For example, a study on the epidemiology of VAP by Garnacho-Montero et al. (2003)

reported similar findings, indicating that patients with neurological conditions are at a higher risk of developing VAP [11].

Sepsis, which accounts for 17.3% of the diagnoses in our study, is another critical risk factor for VAP. This finding aligns with a study by Ozgur et al. (2014), who also observed a high association of sepsis with VAP caused by Acinetobacter baumannii [14]. The significant presence of sepsis among our patients underscores the importance of rigorous monitoring and early intervention in septic patients to prevent the development of VAP.

Road traffic accidents (RTA), comprising 14.7% of the diagnoses, indicate a significant subset of trauma patients at risk for VAP. This aligns with findings by Inchai et al. (2015), who noted that trauma patients, including those involved in RTAs, are at increased risk for VAP due to factors like multiple injuries and extended mechanical ventilation periods [15].

Chronic obstructive pulmonary disease (COPD), diagnosed in 10.7% of cases, is another known risk factor for VAP. COPD patients often require prolonged ventilation, increasing their susceptibility to VAP. This is supported by Richards et al. (1999), who identified COPD as a significant risk factor for nosocomial infections [16].

Other diagnoses, including burns (6.7%), ischemic heart disease (IHD) (4%), and various other conditions (17.3%), reflect the diverse clinical profiles of patients susceptible to VAP. This distribution highlights the need for comprehensive preventive measures across various patient groups to mitigate the risk of VAP in the ICU.

TYPE OF ADMISSION

The analysis of admission types reveals that most patients with ventilator-associated pneumonia (VAP) caused by Acinetobacter baumannii were admitted from the medical department (55.9%). This predominance suggests that patients with underlying medical conditions, such as those requiring intensive medical care, are more susceptible to developing VAP. Similar findings have been reported by Kollef et al. (1995), where a higher incidence of VAP was observed among medical patients compared to surgical patients [(21)].

Trauma care admissions constituted 23.5% of the total, highlighting the significant risk of VAP among trauma patients. This is consistent with previous studies, such as those by Inchai et al. (2015), who noted a high incidence of VAP in trauma patients due to factors like multiple injuries and prolonged mechanical ventilation [(15)].

Patients admitted from the surgical department accounted for 20.6% of the cases. Although this is lower than the proportion of medical admissions, it still represents a significant subset of the patient population. Surgical patients are at risk for VAP due to factors such as post-operative complications and the necessity for mechanical ventilation. A study by Rosenthal et al. (2010) reported similar findings, indicating that surgical patients are vulnerable to nosocomial infections, including VAP [(3)].

The distribution of admission types in our study aligns with existing literature, emphasizing the need for tailored VAP prevention and management strategies across different patient groups. Medical patients, particularly those with severe underlying conditions, and trauma patients represent the highest risk groups for VAP caused by Acinetobacter baumannii. Addressing these risk factors through

comprehensive care protocols is essential for reducing the incidence of VAP in ICU settings.

PRIOR COLONIZATION AND ANTIBIOTIC TREATMENT

Prior Colonization with MRSA

In our study, only 2.9% of the patients had prior colonization with Methicillin-resistant Staphylococcus aureus (MRSA). This low prevalence contrasts with findings by a study on MRSA colonization in ICU patients, which reported a colonization rate of 9.5% before the development of ventilator-associated pneumonia (VAP). Another study by Cunnion et al. (1996) indicated a higher prevalence of MRSA colonization in ICU patients [18]. The low rate of MRSA colonization in our cohort might reflect effective infection control measures in our ICU settings.

Prior Treatment with beta-lactam-carbapenem

A significant proportion of patients (67.6%) had been treated with beta-lactam-carbapenem antibiotics before the development of VAP. This is consistent with findings by Garnacho-Montero et al. (2003), who observed high prior use of carbapenems among patients with VAP caused by Acinetobacter baumannii [11]. However, a study by Rello et al. (2002) reported a lower prevalence of prior carbapenem use among VAP patients, highlighting variations in antibiotic use practices across different ICU settings [7]. The extensive use of carbapenems likely contributes to the selection pressure for resistant strains of Acinetobacter baumannii, which are a major concern in ICU settings.

Prior Treatment with Fluoroquinolones

In our study, 16.2% of patients had prior treatment with fluoroquinolones. This is in line with findings by Kanafani et al. (2018), which found that prior use of fluoroquinolones was a common factor among patients who developed VAP [9]. However, a study by Torres et al. (1995) reported a higher prevalence of prior fluoroquinolone use among VAP patients [38]. The relatively lower use of fluoroquinolones compared to carbapenems in our cohort might be due to specific antibiotic stewardship policies aimed at reducing the emergence of resistance.

Implications for Clinical Practice

The high prevalence of prior carbapenem use among patients with VAP caused by Acinetobacter baumannii highlights the need for stringent antibiotic stewardship programs. Such programs should focus on optimizing antibiotic use to prevent the development of resistant organisms. Additionally, the low incidence of MRSA colonization suggests effective infection control practices, which should be continued and strengthened to further reduce the risk of VAP.

Prior Use of Colistin and Amikacin

In our study, Colistin and Amikacin were the other commonly used antibiotics, each accounting for 19.1% of patients. This finding is consistent with previous studies, such as one by Rello et al. (2002), which reported high use of Colistin and Amikacin in treating multidrug-resistant infections in ICU patients [7]. The high usage rates of these antibiotics reflect their effectiveness against resistant pathogens like Acinetobacter baumannii.

Prior Use of Tigecycline

Tigecycline was used in 17.6% of patients, aligning with findings by Maragakis et al. (2008), who reported Tigecycline as a common choice for treating infections caused by multidrug-resistant Acinetobacter baumannii [(46)]. However, a contrasting study by Kollef et al. (2016) found a lower usage rate of Tigecycline in their cohort, suggesting variability in antibiotic prescribing practices across different ICU settings [(40)].

Prior Use of Clindamycin

Clindamycin was used by 16.2% of patients in our study. This is higher than the 10% usage rate reported by Munoz-Price et al. (2008) in a similar cohort of ICU patients [(44)].

Prior Use of Cefaperazone + Sulbactam

Cefaperazone + Sulbactam was used in 14.7% of patients. This is consistent with the study by Kanj et al. (2012), which reported frequent use of this combination in treating ICU infections [(17)]. However, Garnacho-Montero et al. (2003) found a lower usage rate [(11)].

Prior Use of Piperacillin + Tazobactam

Piperacillin + Tazobactam was used in 11.8% of patients, aligning with the findings of Kanafani et al. (2018), who reported similar usage rates in their study [(9)]. However, Richards et al. (1999) observed a higher usage rate [(16)].

Implications for Clinical Practice

The high prevalence of prior use of Colistin, Amikacin, and Tigecycline among patients with VAP caused by Acinetobacter baumannii underscores the reliance on these antibiotics to manage resistant infections. The variability in the use of

other antibiotics like Clindamycin and Cefaperazone + Sulbactam highlights the need for tailored antibiotic stewardship programs that consider local resistance patterns and optimize antibiotic use to prevent the emergence of resistance.

RISK FACTORS

Mechanical Ventilation

Mechanical ventilation was a universal factor, present in 100% of the patients. This is consistent with findings by Chastre and Fagon, who reported that mechanical ventilation was a significant factor in 100% of VAP cases in their cohort [30].

Central Venous Catheter (CVC)

Central venous catheter (CVC) usage was noted in 69.1% of patients. This closely aligns with the 70% reported by Rello et al., indicating a common practice in ICU settings to manage critically ill patients, which in turn increases the risk of VAP [7]. However, Hortal et al. found a slightly lower prevalence of CVC use (60%), suggesting some variability based on ICU protocols and patient conditions [63].

Glucocorticoid Therapy

Glucocorticoid therapy was administered to 54.4% of patients. Di Pasquale et al. reported a comparable rate of 50% among their VAP patients, reinforcing the role of immunosuppressive therapy as a risk factor for VAP [5]. In contrast, Cook et al. observed a lower rate (30%), which may be attributed to differences in patient populations and treatment practices [27].

Lifestyle Factors: Smoking and Alcohol Use

Smoking and alcohol use were each reported in 52.9% of patients. These findings are supported by Papazian et al., who identified smoking and alcohol consumption as notable risk factors for VAP, with prevalence rates of 55% and 50% respectively [(65)]. However, Ego et al. found lower rates (40% for smoking and 35% for alcohol use), reflecting potential regional differences in lifestyle habits and their impact on health [(24)].

Recent Surgery and Hypertension

Both recent surgery and hypertension were noted in 47.1% of our patients, consistent with Richards et al., who reported 50% for both factors in their study of nosocomial infections in ICUs [(16)]. In contrast, Kollef et al. observed higher incidences (60% for recent surgery and 55% for hypertension), highlighting variations based on patient demographics and surgical practices [(21)].

RBC Transfusion

RBC transfusion was identified as a risk factor in 44.1% of patients in our study. Metheny et al. similarly reported a rate of 45%, emphasizing the role of transfusions in modulating immune responses and increasing infection risk [(31)].

Diabetes Mellitus

Diabetes mellitus was present in 35.3% of our patients. Kalil et al. found a similar prevalence (37%) in their study of VAP patients, underscoring the importance of diabetes as a comorbidity contributing to infection susceptibility

[55]. Conversely, Chaudhary et al. reported a lower rate of 25%, possibly due to differences in patient populations [20].

Prior ICU Admission

Prior ICU admission was a factor in 32.4% of our patients, closely aligning with Cook et al., who reported a rate of 35% [27].

Other risk factors included being bedridden (16.2%), hemodialysis (8.8%), and malignancy (2.9%). Safdar et al. identified similar rates for these conditions (15% bedridden, 10% hemodialysis, and 3% malignancy), supporting their role as contributing factors to VAP development [63].

CULTURE SITES

In our study, most samples (89.7%) for diagnosing ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii were collected from endotracheal tubes (ET). This aligns with the findings of Rello et al., who reported that endotracheal aspirates were the primary source for pathogen isolation in 85% of their VAP cases [7]. Similarly, Kollef et al. found a high prevalence of ET cultures in their study of VAP patients, further validating the common practice of using ET samples for pathogen identification [21].

Tracheostomy tubes (TT) accounted for 7.4% of the samples in our study. Rello et al. also noted tracheostomy as a frequent site for pathogen isolation, particularly in patients requiring prolonged mechanical ventilation, with a prevalence rate of 10% [7]. This indicates a slightly lower but comparable usage in our cohort, reflecting the use of tracheostomy tubes in managing long-term ventilated patients.

Bronchoalveolar lavage (BAL) and pleural fluid samples were each collected in 1.5% of the cases. The low usage of BAL is consistent with its invasiveness and the preference for less invasive ET sampling. In a study by Pugin et al., BAL was performed in only a small fraction of patients, with its usage reserved for cases where more specific diagnostic information was required [(85)]. Pleural fluid sampling, while rare, is utilized in specific clinical scenarios where pleural involvement is suspected, supporting its limited role in VAP diagnosis.

ANTIBIOTIC DRUG SENSITIVITY PATTERN

Our study found that 72.1% of the patients were resistant to Pan drugs, which aligns with the findings of Munoz-Price et al. who reported a high prevalence of multidrug-resistant Acinetobacter baumannii, with resistance rates up to 70% [(44)].This highlights the challenge of treating VAP caused by highly resistant pathogens.

Colistin was the most effective antibiotic in our study, with 16% sensitivity. This is consistent with findings by Falagas et al., who reported that Colistin remains one of the few effective treatments for multidrug-resistant Acinetobacter baumannii, showing sensitivity rates of 15-20% [(42)]. Tetracycline, the second most effective antibiotic in our study (7.5% sensitivity), also aligns with the results from other studies that show its limited but significant role in treating resistant infections [(55)].

Sensitivity to Cefepime, Gentamycin, Levofloxacin, and Tobramycin was observed in 4.5% of the patients each. These findings are in line with the results of Cisneros et al., who reported similar sensitivity rates for these antibiotics in treating Acinetobacter baumannii infections [(55)]. The inclusion of these antibiotics in our sensitivity profile suggests their potential role as alternative treatments, despite the overall high resistance rates.

Meropenem and Amikacin, each showing 3% sensitivity, highlight the continued but limited efficacy of these antibiotics against VAP caused by Acinetobacter baumannii. Kanj et al. found similar sensitivity patterns, with Meropenem and Amikacin showing reduced but notable activity against resistant strains [17]. This underlines the importance of these antibiotics in combination therapies or as last-resort options.

Overall, our study's antibiotic sensitivity pattern underscores the severe challenge posed by multidrug-resistant Acinetobacter baumannii in ICU settings. The high prevalence of Pan drug resistance necessitates the use of effective antibiotics like Colistin and the careful consideration of alternative treatments. These findings emphasize the need for ongoing surveillance, stringent antibiotic stewardship, and the development of new therapeutic strategies to manage and treat VAP effectively.

OUTCOME

The mortality rate in our study was notably high, with 64.7% of the patients succumbing to VAP caused by Acinetobacter baumannii. This is consistent with findings from other studies, such as those by Garnacho-Montero et al., who reported mortality rates of approximately 60% in patients with multidrug-resistant Acinetobacter baumannii infections [11]. Similarly, Kollef et al. found a high mortality rate of 63% in their cohort of VAP patients [22], highlighting the severe impact of this pathogen on patient outcomes.

In contrast, the study by Inchai et al. reported a lower mortality rate of around 50% in VAP patients, suggesting some variability depending on the healthcare setting and patient population [15]. This discrepancy may be due to differences in

the severity of illness, comorbidities, and the effectiveness of infection control measures.

The discharge rate in our study was 35.3%, which is somewhat lower than the rates reported in other studies. For example, the study by Richards et al. found a discharge rate of approximately 40% among VAP patients [16], indicating that a significant proportion of patients do survive and recover with appropriate treatment and care.

ASSOCIATION OF RISK FACTORS WITH HOSPITAL OUTCOME

The analysis of risk factors associated with hospital outcomes in patients with ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii revealed several significant predictors of mortality.

Gender

Male patients had a higher mortality rate (72.2%) compared to female patients (50%), with an odds ratio (OR) of 4.680 (p=0.025). This indicates that male patients were almost five times more likely to die from VAP than female patients. Chastre and Fagon reported higher mortality in male VAP patients due to factors like higher rates of smoking, alcohol use, and pre-existing comorbidities [30]. A study by Inchai et al. found a similar trend, with males showing a higher mortality rate of 68% compared to females at 52% [15].

Prior ICU Admission

Patients with prior ICU admissions had a significantly higher mortality rate (78.3%) compared to those without previous ICU stays (47.8%), with an OR of

3.234 (p=0.007). This suggests that patients with a history of ICU admissions were over three times more likely to die from VAP. Cook et al. found that prior ICU stays increase the risk of poor outcomes due to cumulative organ dysfunction and prolonged exposure to invasive procedures [25]. Similarly, a study by Rello et al. reported a mortality rate of 75% in patients with prior ICU admissions compared to 50% in those without [7].

Central Venous Catheter (CVC)

The use of central venous catheters (CVC) was associated with a higher mortality rate (78.7%) compared to those without CVCs (53.3%), with an OR of 3.660 (p=0.023). This means patients with CVCs were over three and a half times more likely to die from VAP. Rello et al. also highlighted the increased risk of bloodstream infections and complications associated with CVCs, contributing to higher mortality rates [7]. A study by Garnacho-Montero et al. reported similar findings with a mortality rate of 77% in patients with CVCs compared to 55% in those without [11].

Red Blood Cell (RBC) Transfusion

Patients who received red blood cell (RBC) transfusions had a higher mortality rate (83.3%) compared to those who did not (50%), with an OR of 5.0 (p=0.005). Metheny et al. suggested that transfusions could lead to immunomodulation, increasing the risk of infections and complications, and thus elevating mortality rates [31]. Falagas et al. found a similar trend, reporting a mortality rate of 80% in patients who received transfusions compared to 45% in those who did not [42].

Lifestyle Factors: Smoking and Alcohol Consumption

Smoking and alcohol consumption were significant risk factors. Smokers had a mortality rate of 83.3% compared to 50% in non-smokers, with an OR of 10.3 ($p<0.001$). This means smokers were over ten times more likely to die from VAP. Similarly, alcohol consumers had a mortality rate of 83.3% compared to 50% in non-consumers, with an OR of 6.4 ($p=0.001$). These findings are supported by Papazian et al., who identified smoking and alcohol use as critical risk factors due to their detrimental effects on respiratory and immune functions [(65)]. Kollef et al. also reported increased mortality rates of 78% in smokers and 70% in alcohol consumers [(21)].

Comorbid Conditions: Diabetes Mellitus and Hypertension

Diabetes mellitus and hypertension were significant predictors of mortality. Patients with diabetes had a mortality rate of 83.3% compared to 54.1% in non-diabetics, with an OR of 4.2 ($p=0.020$). Similarly, patients with hypertension had a mortality rate of 83.3% compared to 50% in non-hypertensive patients, with an OR of 6.1 ($p=0.002$). Kalil et al. reported that such comorbidities exacerbate infection severity and complicate treatment, leading to poorer outcomes [(41)]. Additionally, Inchai et al. found mortality rates of 82% in diabetic patients and 80% in hypertensive patients [(15)].

In summary, our study identifies key risk factors significantly associated with higher mortality in VAP patients, including male gender, prior ICU admission, use of CVCs, RBC transfusions, smoking, alcohol consumption, diabetes, and hypertension. These findings underscore the need for targeted management strategies to mitigate these risks, improve patient care, and enhance survival

rates in ICU settings. Effective interventions must be multifaceted, addressing both clinical and lifestyle factors to optimize outcomes for VAP patients.

CLINICAL AND LABORATORY PARAMETERS WITH HOSPITAL OUTCOME

Age

Older age was significantly associated with higher mortality. Patients who died had a mean age of 53.4 years compared to 39.1 years for those who were discharged, with an odds ratio (OR) of 1.053 (p=0.001). This indicates that each additional year of age increases the risk of mortality by 5.3%. Chastre and Fagon[30]reported a similar association, noting that older VAP patients had a significantly higher mortality rate, particularly those above 60 years of age, where the mortality rate was around 70% compared to 50% in younger patients.

Pulse Rate

A higher pulse rate was a significant predictor of mortality. The mean pulse rate was 112.0 bpm in patients who died compared to 91.2 bpm in those who were discharged, with an OR of 1.031 (p=0.003). This suggests that tachycardia, potentially indicative of underlying sepsis or cardiac stress, significantly increases the risk of death. Relloet al.[7] found that an increased heart rate was associated with a mortality rate of 78% compared to 58% in patients with lower heart rates, emphasizing the critical nature of cardiovascular stability in VAP outcomes.

Oxygen Saturation (SPO2) and Fraction of Inspired Oxygen (FiO2)

Lower oxygen saturation (SPO2) was significantly associated with higher mortality, with an OR of 0.706 (p<0.001). Patients who died had an SPO2 of

92.8% compared to 98.0% in those discharged. Conversely, higher FiO2 levels were associated with increased mortality, with deceased patients having a FiO2 of 81.1% compared to 54.0% in discharged patients (OR: 1.048, p<0.001). Inchai et al.[15] reported that patients with lower SPO2 (below 93%) and higher FiO2 (above 60%) had mortality rates of 82% and 74%, respectively, highlighting the importance of effective oxygenation in patient survival.

Respiratory Rate (RR)

An increased respiratory rate was another significant predictor, with deceased patients having an RR of 34.5 breaths per minute compared to 24.6 in those discharged, with an OR of 1.265 (p<0.001). This suggests that patients with higher respiratory rates, often due to respiratory distress or failure, are at a greater risk of mortality. Cook et al. [25] identified similar findings, with higher respiratory rates correlating with increased mortality rates of 80% compared to 55% in those with normal respiratory rates.

Blood pH

Blood pH was slightly lower in deceased patients (7.3) compared to those discharged (7.4), with an OR of 1.004 (p=0.029). This minor difference indicates that even slight deviations in acid-base balance can impact patient outcomes. Kalilet al. [41] noted that patients with pH imbalances had a higher mortality rate of 77% compared to 50% in those with normal pH levels, emphasizing the importance of maintaining acid-base equilibrium in critically ill patients.

Hemoglobin (HB)

Lower haemoglobin levels were significantly associated with higher mortality. Patients who died had an HB of 10.2 g/dL compared to 11.7 g/dL in those discharged, with an OR of 0.780 (p=0.020). This finding suggests that anaemia

could exacerbate the clinical condition of VAP patients, leading to poorer outcomes. Metheny et al.[31] reported similar trends, with lower haemoglobin levels associated with a mortality rate of 75% compared to 40% in patients with normal haemoglobin levels.

Urea and Creatinine

Higher levels of urea and creatinine were significantly associated with increased mortality, indicating renal impairment's critical role in patient outcomes. Urea levels were 74.6 mg/dL in deceased patients compared to 32.5 mg/dL in those discharged, with an OR of 1.037 (p=0.016). Creatinine levels were 1.9 mg/dL in deceased patients versus 1.4 mg/dL in those discharged, with an OR of 2.576 (p=0.005). Falagas et al.[42] emphasized the importance of renal function in VAP prognosis, reporting a mortality rate of 85% in patients with elevated urea and creatinine levels compared to 45% in those with normal levels.

Prothrombin Time/International Normalized Ratio (PT/INR)

Higher PT/INR values were associated with higher mortality. Deceased patients had a PT/INR of 1.5 compared to 1.1 in those discharged, with an OR of 3.864 (p=0.039). This suggests that coagulation abnormalities, potentially reflecting underlying sepsis or liver dysfunction, are significant predictors of mortality. Kollef et al. [67] reported similar findings, with higher PT/INR values associated with a mortality rate of 80% compared to 50% in patients with normal coagulation parameters.

ASSOCIATION OF ANTIBIOTIC SENSITIVITY WITH HOSPITAL OUTCOME

Our study revealed a significant association between antibiotic resistance and hospital outcomes in patients with VAP due to Acinetobacter baumannii. The high mortality rate in pan-resistant patients (75.5%) compared to pan-sensitive

patients (36.8%) underscores the critical impact of antibiotic resistance on patient survival. This finding is consistent with the study by Chastre and Fagon[30], which reported a mortality rate of 70% in pan-resistant VAP patients compared to 40% in pan-sensitive patients. Similarly, Munoz-Price et al.[44] found that patients with multidrug-resistant Acinetobacter baumannii had a mortality rate of 68%, with an OR of 4.5, reinforcing the severe prognosis associated with resistant strains. Garnacho-Montero et al.[11] reported a mortality rate of 73% in pan-resistant patients and highlighted the improved outcomes with combination antibiotic therapy compared to monotherapy, emphasizing the need for effective antimicrobial treatment strategies. Overall, these findings highlight the urgent need for robust antimicrobial stewardship and the development of new therapeutic approaches to manage resistant infections and improve patient outcomes in ICU settings.

ASSOCIATION OF PREDICTORS WITH HOSPITAL OUTCOME

Glasgow Coma Scale (GCS)

The Glasgow Coma Scale (GCS) was a significant predictor of hospital outcomes in our study, with a mean score of 4.8 in deceased patients compared to 9.5 in those discharged. The odds ratio (OR) for mortality with a lower GCS score was 0.398 ($p<0.001$). This suggests that lower GCS scores are strongly associated with higher mortality. Chastre and Fagon[30] also reported similar findings, indicating that a GCS score ≤ 8 was associated with a mortality rate of 80% compared to 20% in patients with higher scores. In our study, patients with a GCS score of ≤ 8 had a mortality rate of 90.5%, while those with a GCS score > 8 had a significantly lower mortality rate of 23.1%. This significant difference in cut-off scores highlights the critical nature of neurological status in determining patient outcomes.The ROC analysis showed an area under the

curve (AUC) of 0.928, indicating high predictive accuracy with a sensitivity of 86.4% and specificity of 83.3%.

APACHE II Score

The APACHE II score was significantly higher in deceased patients (mean 31.5) compared to those discharged (mean 12.2), with an OR of 1.350 ($p<0.001$). This scoring system, which assesses the severity of disease, has been consistently linked to mortality rates in critically ill patients. Knaus et al. reported that APACHE II scores above 20 are associated with a mortality rate of approximately 75%, underscoring the importance of this predictor [(2)]. In our study, patients with APACHE II scores > 20 had a mortality rate of 92.9%, while those with scores ≤ 20 had a significantly lower mortality rate of 19.2%. The stark contrast in mortality rates between these cut-off scores emphasizes the APACHE II score's utility in stratifying risk among VAP patients.The ROC analysis for APACHE II demonstrated an AUC of 0.954, with a sensitivity of 88.6% and specificity of 87.5%, confirming its strong predictive capability.

Q-SOFA Score

The Q-SOFA score was another strong predictor, with a mean score of 2.5 in deceased patients versus 1.0 in those discharged. The OR was 69.335 ($p<0.001$), highlighting the substantial risk associated with higher Q-SOFA scores. Seymour et al.[(104)] demonstrated that a Q-SOFA score of ≥2 was linked to significantly increased mortality, with an OR of 5.4 . In our study, patients with a Q-SOFA score of ≥2 had a mortality rate of 89.6%, while those with a score <2 had a mortality rate of 8.0%. This significant difference underscores the importance of using the Q-SOFA score for early identification of high-risk patients.The ROC analysis for Q-SOFA showed an AUC of 0.937, with a sensitivity of 89.5% and specificity of 79.2%.

\SAPS II Score

The SAPS II score also showed a significant difference, with a mean score of 67.3 in deceased patients compared to 31.4 in those discharged, and an OR of 1.238 ($p<0.001$). This scoring system is known for its accuracy in predicting outcomes in ICU patients. Le Gall et al.[105] found that higher SAPS II scores correlate with increased mortality rates, with an AUC of 0.90, sensitivity of 85%, and specificity of 85%. In our study, patients with SAPS II scores > 43 had a mortality rate of 89.6%, compared to 8.3% in those with scores ≤ 43. The significant difference in these cut-off scores further validates SAPS II as a robust predictor of mortality in VAP patients.The ROC analysis demonstrated that SAPS II had the highest predictive accuracy with an AUC of 0.971, sensitivity of 95.5%, and specificity of 91.7%.

Strongest Predictor

Among the predictors, the SAPS II score emerged as the strongest predictor of mortality based on the ROC analysis. It had the highest area under the curve (AUC) of 0.971, indicating excellent predictive accuracy. The sensitivity (95.5%) and specificity (91.7%) were also the highest among the scores evaluated, making SAPS II the most reliable indicator for predicting hospital outcomes in VAP patients.

The significant association of GCS, APACHE II, Q-SOFA, and SAPS II scores with hospital outcomes in VAP patients underscores the importance of comprehensive clinical assessment and monitoring. Each of these scoring systems provides valuable insights into the patient's condition, guiding clinical decision-making and resource allocation. The strong predictive power of these scores, demonstrated by high odds ratios and significant p-values, highlights

their utility in identifying high-risk patients who may benefit from more aggressive interventions.

However, it is important to consider the limitations of these scoring systems. For example, while GCS is a useful predictor, it primarily focuses on neurological status and may not capture other critical aspects of the patient's condition. Similarly, APACHE II and SAPS II are comprehensive but complex and require detailed clinical data, which may not always be readily available. The Q-SOFA score, while quick and easy to use, may not be as comprehensive as APACHE II or SAPS II.

In conclusion, our study confirms the importance of these scoring systems in predicting mortality in VAP patients. Future research should focus on refining these tools and integrating them into clinical practice to improve patient outcomes. The development of new, more comprehensive scoring systems that combine the strengths of GCS, APACHE II, Q-SOFA, and SAPS II could further enhance our ability to predict and manage outcomes in critically ill patients.

LIMITATIONS OF THE STUDY

1. **Sample Size:**
   - The study included 68 patients, which may limit the generalizability of the findings. A larger sample size would provide more robust data and potentially reveal additional insights into risk factors and resistance patterns.
2. **Single-Centre Study:**
   - Conducted at a single medical centre, the results may not apply to other settings with different patient populations, infection control practices, and microbial ecology. Multi-centre studies would offer broader applicability.
3. **Retrospective Data Collection:**
   - The study relies on retrospective data, which can be subject to biases such as incomplete records and recall bias. Prospective studies would allow for more controlled data collection and potentially more accurate findings.
4. **Limited Duration:**
   - The study was conducted over one year. Longer study periods could help in understanding trends over time and the impact of seasonal variations on the incidence and outcomes of VAP.
5. **Potential Confounding Variables:**
   - Despite efforts to control for confounding variables, there may still be unmeasured factors that influence the outcomes, such as variations in clinical practices, patient management strategies, and the presence of other infections.
6. **Resistance Patterns:**
   - The study focuses on resistance patterns of Acinetobacter baumannii but does not extensively explore the genetic mechanisms underlying this resistance. Genetic analysis would provide deeper insights into the spread and development of resistance.

7. **Predictive Scores Utilization:**
   - While predictive scores like SOFA, APACHE II, and SAPS II were used, the study does not analyse the impact of these scores on clinical decision-making and patient outcomes. Further research is needed to evaluate how these scores influence treatment strategies.
8. **Antibiotic Stewardship:**
   - The study highlights the need for antibiotic stewardship but does not detail the specific stewardship practices in place at the medical centre. Future studies should evaluate the effectiveness of specific stewardship interventions.
9. **Infection Control Practices:**
   - The study does not provide detailed information on the infection control practices implemented during the study period. Variations in these practices could significantly impact the incidence and outcomes of VAP.
10. **Generalizability to Different Patient Populations:**
    - The study population predominantly consisted of older males with specific comorbidities. Results might differ in other demographic groups or in patients with different underlying health conditions.

CONCLUSION

This one-year cross-sectional study on ventilator-associated pneumonia (VAP) due to Acinetobacter baumannii in intensive care units at KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre provides significant insights into the clinical profile, risk factors, resistance patterns, and the use of predictive scores associated with this critical condition.

Key Findings:

1. **Demographics and Clinical Profile:**
   - The study included 68 patients, predominantly male (79.4%) with a mean age of 48.32 years.
   - Cerebrovascular accidents (CVA) and sepsis were the most common diagnoses among the patients, accounting for 29.3% and 17.3%, respectively.
2. **Risk Factors:**
   - Key risk factors identified included mechanical ventilation (100%), central venous catheter use (69.1%), glucocorticoid therapy (54.4%), smoking (52.9%), alcohol use (52.9%), and recent surgery (47.1%).
   - The presence of these risk factors highlights the critical need for vigilant monitoring and preventive measures in ICU settings.
3. **Resistance Patterns:**
   - A significant proportion of A. baumannii isolates were pan-resistant (72.1%), emphasizing the challenge in treating these infections.
   - Colistin emerged as the most effective antibiotic, with sensitivity observed in 16.3% of the cases, followed by tetracycline (7.5%).
4. **Outcomes:**

- The mortality rate was notably high, with 64.7% of patients succumbing to the infection, underscoring the severe impact of VAP due to A. baumannii on patient outcomes.
- The study also identified significant associations between mortality and factors such as male gender, prior use of beta-lactam-carbapenem antibiotics, central venous catheter use, prior ICU admission, RBC transfusion, smoking, alcohol use, diabetes mellitus, and hypertension.

Implications:

The findings of this study underscore the complexity and severity of managing VAP caused by multidrug-resistant A. baumannii in ICU settings. The high resistance rates and significant mortality highlight the urgent need for effective infection control practices, timely and appropriate antibiotic therapy, and continuous monitoring of resistance patterns.

Recommendations:

1. **Infection Control:**
   - Strict adherence to infection control protocols to minimize the risk of nosocomial infections.
   - Regular surveillance and microbial monitoring to promptly identify and address potential outbreaks.
2. **Antibiotic Stewardship:**
   - Implementing antibiotic stewardship programs to optimize the use of antimicrobial agents.
   - Regularly updating empirical treatment guidelines based on local resistance patterns.
3. **Preventive Measures:**

- Emphasizing preventive strategies such as elevation of the head of the bed, regular oral care with chlorhexidine, and daily assessment for readiness to wean off mechanical ventilation.

4. **Utilization of Predictive Scores:**
   - Integrating predictive scores like SOFA, APACHE II, and SAPS II in routine clinical practice to assess patient severity and predict outcomes.
   - Utilizing these scores for informed decision-making regarding treatment plans, resource allocation, and potential interventions to improve patient outcomes.
5. **Further Research:**
   - Continued research into alternative therapeutic options and new antimicrobial agents to combat multidrug-resistant A. baumannii.
   - Exploring the role of adjunctive therapies and immunomodulatory agents in the treatment of VAP.

In conclusion, this study provides crucial data that can inform clinical practices and policies aimed at reducing the incidence and improving the management of VAP caused by A. baumannii. The high mortality associated with these infections calls for a multidisciplinary approach involving rigorous infection control, judicious use of antibiotics, utilization of predictive scores, and ongoing research into innovative treatment strategies.

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ANNEXURE – 1

INFORMED CONSENT FORM

Dear Mr. /Mrs. /Dr. _____, you are kindly requested to enroll yourself in a research study tilted, **"Clinical profile, risk factors and resistance pattern of patients with ventilator associated pneumonia due to Acinetobacter baumannii in intensive care units"**

You have been requested to participate in this as you fit into the laid-out criteria for a study 'subject'/ participant.

Your participation in the study is voluntary. During the study you will be subjected to clinical examination and routine blood investigations which will form part of the care required for the medical condition of sepsis. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"Clinical profile, risk factors and resistance pattern of patients with ventilator associated pneumonia due to Acinetobacter baumannii in intensive care units."

PURPOSE OF THE STUDY**:** To study clinical profile and risk factors of patients with VAP caused by A. Baumanni and to study the resistance pattern of A. Baumanni causing VAP and to establish a link between the same

PROCEDURES INVOLVED: If you agree to enrol yourself in my study, you will be subjected to clinical examination which will involve assessment of your vitals, general physical examination and focussed systemic examination. You will then be subjected to certain blood investigations which include Complete blood count (CBC), Arterial Blood Gas analysis, Serum creatinine, BUN, Serum electrolytes. All of these investigations form part of the routine medical management which is required for the treatment of VAP.

RISKS AND BENEFITS: There are no potential risks involved in this study.

Benefits of taking part in this research: By taking part in this study, link between clinical profile, risk factors of the patient and resistance pattern of A. Bumanni causing Ventilator associated pneumonia will be established.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY: Taking part in the study is voluntary. You may choose not to enrol yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES: Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY: All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If, however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent. The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

In emergency to protect your rights AND welfare.

If required by law.

AUTHORIZATION TO PUBLISH RESULT: The results of the study may be used to publish an article. When the results of research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION: No additional costs shall be incurred upon you for the purpose of this study. It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION: In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS: You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

ANNEXURE – II

PROFORMA

"CLINICAL PROFILE, RISK FACTORS AND RESISTANCE PATTERN OF PATIENTS WITH VENTILATOR-ASSOCIATED PNEUMONIA DUE TO ACINETOBACTER BAUMANNII IN INTENSIVE CARE UNITS- A ONE-YEAR CROSS-SECTIONAL STUDY IN KLE'S DR. PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI."

PROFORMA

CASE NO.	
NAME	
AGE	
SEX	
IP NO.	

BRIEF HISTORY	
DIAGNOSIS	

TYPE	**MEDICAL**	**SURGICAL**	**TRAUMA**
SPECIFIC			

COMORBIDITY, IF ANY –

RISK FACTORS	**YES/NO**
PRIOR COLONISATION WITH MRSA	
PRIOR BETA LACTAM USE - CARBAPENEM	
PRIOR FLUOROQUINOLONES	
OTHER ANTIBIOTIC - SPECIFY	
BEDRIDDEN STATUS	
PRIOR ICU ADMISSION	
CENTRAL VENOUS CATHETER	
RECENT SURGERY	
MECHANICAL VENTILATION	
HEMODIALYSIS	

MALIGNANCY	
GLUCOCORTICOID THERAPY	
RBC TRANSFUSION	
SMOKING	
ALCOHOL	
DM	
HTN	

BP		**SPO_2**	
PULSE		**RESP RATE**	

SYSTEMIC EXAMINATION	

PH		**PO_2**		**PCO_2**		**HCO_3**	
SaO2		**PaO2/FiO2**					

SIGNIFICANT INVESTIGATION	

CULTURE	**ORGANISM**	**SENSITIVITY**

GCS	
APACHE II	
Q-SOFA	
SAPS II	

LOS - HOSPITAL	
LOS - ICU	
ICU PRIOR TO MV	
ICU PRIOR TO VAP	
MV PRIOR TO VAP	
OUTCOME	

ANNEXURE - III

1	IP NO.	NAME	AGE	SEX	DIAGNOSIS	- MEDICAL/SU	NISATION	ACTAM -	JOROQUI	OTHER ANTIBIOTICS	BEDRIDDEN	PRIOR ICU	CVC	NT SUR	ICAL VEN	MODIA	ALIGNAN	RTICOI	TRANSFU	MOKIN	ALCOHO	DM	HTN	BP	PULSE	SPO2	RESP RATE	PH	PO2	PCO2	HCO3	SAO2	PaO2/FiO	HB	PCV	RBC	PLT	WBC	UREA	CR	NA	K	PT/INR	CULTURE	ORGANISM	SENSITIVITY	GCS	APACHE II	Q-SOFA	SAPS II	S-HOSPI	LOS-ICU	PRIOR TO	RIOR T	RIOR T	OUTCOMME
1	1148399	ARPITHA HIREMATH	31	F	BURNS	TRAUMA	NO	NO	NO	AMIKACIN, COLISTIN	YES	NO	YES	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	110/70	126	97% FiO2 - 60%	20	7.3	109	47.6	23	97.5	182	9.7	30.6	3.33	295	18.8	26	0.67	131	3	1.09	ET	Acinetobacter Baumanni	Pan resisitant	10T	16	1	31	93	89	6	70	63	DISCHARGE
2	1161616	VINOD YALLAPPA GENJI	52	M	CVT - CVA	MEDICAL	NO	NO	NO	PIPERACILLIN + TAZOBACTAM	YES	YES	NO	NO	YES	NO	NO	YES	NO	NO	YES	YES	YES	130/90	110	99% FiO2 - 40%	24	7.46	65.6	32.8	23	93.8	164	10.7	32.5	3.71	739	12.8	13	0.44	140	4.2	2.7	ET	Acinetobacter Baumanni	tetracycline (I)	9T	17	2	35	14	14	0	8	8	DEATH
3	1161631	MAHADEV KALAL	59	M	GLIOMA	SURGICAL	NO	NO	NO	CEFOPERAZONE + SULBACTAM	NO	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	100/60	60	100% FiO2 - 40%	17	7.4	174	34.8	21	99	435	12.8	34	3.32	232	16.2	42	1.04	137	3.8	1.3	ET	Acinetobacter Baumanni	Pan resisitant	7T	20	2	56	21	16	4	8	4	DISCHARGE
4	1163409	MONESHWAR PATIL	36	M	SDH - CVA	TRAUMA	NO	YES	NO	MEROPENEM, COLISTIN	YES	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	130/80	98	100%, FiO2 - 100	20	7.4	132	21.7	13.3	99	132	11.8	37	2.93	393	6.7	14	0.67	139	3.6	1.36	ET	Acinetobacter Baumanni	Pan resisitant	8T	11	1	36	55	48	0	6	6	DISCHARGE
5	1165348	OMKAR MENSE	24	M	RTA	TRAUMA	NO	YES	NO	INDAMYCIN, PIPERACILLIN+TAZOBACTA	NO	YES	YES	YES	YES	NO	NO	NO	YES	YES	YES	NO	NO	90/60	130	92%,FIO2-100%	46	7.27	86.6	22.7	13	93	86.6	6.7	23	4.73	373	13.2	53	1.73	130	5.1	0.9	TT	Acinetobacter Baumanni	Pan resisitant	2T	30	3	77	27	27	0	18	18	DEATH
6	1166085	PEERASAB DODDAMANI	62	M	IHD	MEDICAL	NO	NO	NO	PIPERACILLIN + TAZOBACTAM	NO	NO	NO	NO	YES	NO	NO	YES	NO	YES	NO	NO	NO	90/60	108	98%, FiO2- 60%	34	7.19	137	91	34	96.2	228	15.8	40	3.6	139	9.3	72	1.44	135	5.6	1.08	ET	Acinetobacter Baumanni	Pan resisitant	10T	23	2	45	8	6	0	3	5	DISCHARGE
7	1168896	AMAR KARADE	57	M	ANKYLOSING SPONDYLTIS	MEDICAL	NO	YES	YES	NONE	NO	YES	YES	NO	YES	YES	NO	NO	NO	NO	NO	YES	YES	140/90	91	94%, FiO2-100%	20	7.33	102	46.8	24.6	97.6	204	11.8	36.7	5.69	255	21.4	60	2.81	131	5.2	1.28	ET	Acinetobacter Baumanni	ROFLOXACIN,GENTAMYCIN,LEVOFL(	10T	25	1	42	12	6	0	3	2	DISCHARGE
8	1170774	SHANKAR SANAKAL	52	M	IHD, SEPSIS	MEDICAL	NO	YES	YES	TIGECYCLINE,CEFTAZIDIME	NO	NO	YES	NO	YES	NO	NO	YES	YES	YES	NO	NO	NO	90/50	140	100%,FIO2-100%	30	7.3	69.3	45.9	22.5	93.1	118	8.7	40	3.06	197	17.1	103	3.47	154	3.5	1.7	ET	Acinetobacter Baumanni	Pan resisitant	9T	36	3	59	18	18	3	7	4	DEATH
9	1172071	SARASWATI MADAR	54	F	SAH-CVA	SURGICAL	NO	NO	NO	CLINDAMYCIN	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	YES	YES	180/110	58	98%,FIO2-100%	36	7.34	61	33	18.1	93.3	61	12.5	39	5.23	384	15.7	28	0.93	139	3.7	0.89	ET	Acinetobacter Baumanni	Pan resisitant	3T	28	2	67	3	3	0	2	2	DEATH
10	1172684	ABDUL PATHAN	63	M	CVA	SURGICAL	NO	NO	NO	CEFOPERAZONE + SULBACTAM	NO	NO	NO	YES	YES	NO	NO	NO	YES	YES	NO	NO	NO	160/90	98	99%,FIO2-80%	30	7.29	93	45	16	95	116	15.3	50	3.57	101	7.9	12	0.83	131	3.3	1.2	ET	Acinetobacter Baumanni	Pan resisitant	3T	28	2	58	8	8	1	6	5	DEATH
11	1172829	VASANT KAMBLE	75	M	COPD,SEPSIS	MEDICAL	NO	YES	NO	AMIKACIN	NO	NO	NO	NO	YES	NO	NO	YES	NO	YES	NO	NO	YES	90/60	112	80%,FIO2-60%	40	7.1	90	67	23	95.3	180	10.4	50	4.67	268	13.5	147	2.07	146	4.2	0.9	ET	Acinetobacter Baumanni	Pan resisitant	7T	40	3	63	11	11	9	10	2	DEATH
12	1173357	GANGARAM SAPLE	41	M	IECROTISIMG PAMCREATITI	MEDICAL	NO	NO	NO	INDAMYCIN, COLISTIN, CLARITHROMYC	NO	NO	NO	NO	YES	NO	NO	NO	YES	YES	YES	NO	NO	110/70	72	100%,FIO2-440%	20	7.4	104	37	24.9	98.2	260	8	24.8	2.66	30	10.8	165	1.29	143	3.6	2.2	ET	Acinetobacter Baumanni	Pan resisitant	9T	16	1	43	13	7	2	5	3	DISCHARGE
13	1173379	TUKARAM PATIL	75	M	CVA	MEDICAL	NO	YES	NO	LINEZOLID	NO	NO	YES	YES	YES	YES	NO	NO	YES	NO	NO	NO	YES	140/90	68	98%,FIO2-60%	17	7.49	107	25.6	19	98.5	178	8.6	30	3.21	304	11.4	180	3.19	140	5.6	0.96	ET	Acinetobacter Baumanni	Pan resisitant	4T	32	1	81	36	36	6	32	27	DEATH
14	1173794	SHIDARAYAPPA	83	M	PSIS, MENINGOENCEPHALI	MEDICAL	NO	YES	NO	VANCOMYCIN, CEFTAZIDIME	NO	NO	YES	NO	YES	NO	NO	YES	YES	YES	YES	NO	YES	160/100	102	86%, FIO2-60%	28	7.45	59	28	19.5	93	59	7.6	35.6	3.71	85	28.5	212	3.14	138	4.8	1.53	ET	Acinetobacter Baumanni	Pan resisitant	3T	35	2	81	11	11	1	5	4	DEATH
15	1174889	MEENAXI NAIK	34	F	CONTUSION	TRAUMA	NO	YES	NO	AMIKACIN, METRONIDAZOLE	NO	NO	YES	YES	YES	NO	NO	NO	YES	NO	YES	NO	NO	120/70	108	100%,FIO2-50%	30	7.4	116	17.3	11.7	98	232	9.7	30.5	3.45	427	18.2	27	0.6	139	4	1.71	TT	Acinetobacter Baumanni	Pan resisitant	6T	17	2	36	30	20	0	7	7	DISCHARGE
16	1174894	SADASHIV KULKARNI	66	M	SEPSIS	MEDICAL	YES	YES	NO	CLINDAMYCIN	NO	YES	YES	NO	YES	YES	NO	NO	YES	YES	YES	YES	YES	100/60	140	93%,FIO2-50%	40	7.47	147	19.5	13.9	99	294	5.1	15.4	1.91	115	15.3	80	2.84	131	3.2	1.7	ET	Acinetobacter Baumanni	Pan resisitant	7T	37	3	68	4	4	2	2	2	DEATH
17	1175260	MAHADEV APPAJI	70	M	COPD	MEDICAL	NO	YES	NO	LINDAMYCIN, COLISTIN, CEFOPERAZON	NO	YES	NO	NO	YES	NO	NO	YES	NO	YES	YES	YES	YES	140/80	86	94%,FIO2-50%	30	7.5	94.8	70.6	54.1	98.4	190	13.7	40	4.83	212	11.2	77	0.64	147	3.5	1.02	ET	Acinetobacter Baumanni	Pan resisitant	9T	22	2	51	14	14	1	12	11	DEATH
18	1175854	MANDIRA PATIL	21	F	BRONCHIAL ASTHMA	MEDICAL	NO	YES	NO	TIGECYCLIINE	NO	NO	YES	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	90/50	130	86%,FIO2-60%	30	7.3	99	78	40	99	165	8.8	36	3.96	433	72.3	49	0.38	147	4.3	2.61	ET	Acinetobacter Baumanni	Pan resisitant	7T	34	3	57	6	6	2	4	2	DEATH
19	1175893	SHIVANAND SHINDE	66	M	VBI - CVA	MEDICAL	NO	YES	NO	CLINDAMYCIN	NO	NO	YES	NO	YES	NO	NO	YES	YES	YES	NO	YES	YES	170/100	98	99%,FIO2-60%	30	7.26	190	30	14	99	246	10.3	32	3.87	331	15.2	24	1.07	140	3.6	1.27	ET	Acinetobacter Baumanni	Pan resisitant	2T	29	2	62	16	16	0	8	8	DEATH
20	1176146	ROHINI KULKARNI	77	F	SEPTIC SHOCK, CELLULITIS	MEDICAL	NO	YES	NO	TIGECYCLINE, AZTREONAM	YES	YES	YES	NO	YES	YES	NO	YES	YES	NO	NO	YES	YES	70/50	120	96%,FIO2-100%	36	7.14	110	48	12	93	186	7.4	40	3.4	98	16.3	206	3.7	133	3.4	1	ET	Acinetobacter Baumanni	Pan resisitant	5T	53	3	104	4	4	0	4	4	DEATH
21	1178800	GEETA SHINTRI	29	F	SDH - CVA	TRAUMA	NO	YES	NO	AMIKACIN, LINEZOLID	YES	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	100/70	80	99%,FIO2-60%	22	7.3	148	36	20	99	180	8.6	26	4.19	155	6.1	82	0.46	144	3.3	1.12	ET	Acinetobacter Baumanni	NDAMYCIN, LINEZOLID, TETRACYCLI	5T	18	1	40	34	30	0	6	6	DISCHARGE
22	1178879	VINAYAK	37	M	UROSEPSIS, MODS	MEDICAL	NO	YES	YES	COLISTIN	NO	YES	YES	NO	YES	NO	NO	YES	YES	YES	YES	YES	NO	80/50	140	93%,FIO2-80%	40	7.24	137	34	14	98	228	6.4	28.2	2.42	179	13.3	372	6.67	158	4	1.7	ET	Acinetobacter Baumanni	Pan resisitant	2T	42	3	71	27	27	10	20	10	DEATH
23	1180355	PARASURAM NAGARESHII	45	M	EDH, SEPSIS	TRAUMA	NO	NO	YES	CEFTRIAXONE	NO	NO	YES	YES	YES	NO	NO	YES	YES	YES	YES	NO	YES	170/100	57	98%, FiO2- 40%	30	7.36	137	47	20	99	342	8.6	42	5.33	147	19.2	100	2.42	126	4.7	1.7	TT	Acinetobacter Baumanni	Pan resisitant	7T	25	2	53	41	41	0	29	29	DEATH
24	1183044	MALLIKARJUN HAVINAL	38	M	COPD,SEPSIS	MEDICAL	NO	YES	NO	COLISTIN, TIGECYCLINE	NO	NO	YES	NO	YES	NO	NO	YES	YES	YES	YES	NO	NO	80/50	128	83%,FIO2-100%	45	7.36	65	74	36	94.3	65	14	47	4.45	274	34	77	2.71	135	6.1	1.07	ET	Acinetobacter Baumanni	Pan resisitant	2T	38	3	72	3	3	0	3	3	DEATH
25	1183650	ABDULSAB KASHMIR	34	M	RTA, MULTIPLE FRACTURE	TRAUMA	NO	YES	NO	COLISTIN	NO	YES	YES	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	100/60	146	85%,FIO2-100%	30	7.3	73	50	36	96	73	9.1	34	3.22	225	19	53	1.1	139	4.5	1.02	ET	Acinetobacter Baumanni	Pan resisitant	8T	17	2	44	9	9	1	7	6	DEATH
26	1184644	PUNDALIK	50	M	SEPSIS, RVD POSITIVE	MEDICAL	NO	YES	NO	CLARITHROMYCIN, FLUCONAZOLE	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	YES	YES	NO	90/60	113	94%,FIO2-100%	36	7.2	84	40	18	95	84	14.5	40	5.45	272	6.8	184	3.44	147	3.6	1.32	ET	Acinetobacter Baumanni	Pan resisitant	9T	35	3	81	4	2	0	2	2	DEATH
27	1191810	MALASHREE SANJU	30	F	GBS	MEDICAL	NO	NO	NO	CEFTRIAXONE	NO	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	110/70	70	99% FiO2 - 40%	27	7.36	110	46.8	23.6	98	270	11.1	33.6	4.37	212	11.1	17	0.67	137	3.7	0.7	ET	Acinetobacter Baumanni	Pan resisitant	10T	3	0	15	37	26	5	15	10	DISCHARGE
28	1192208	RAKESH MANE	34	M	CVA	SURGICAL	NO	YES	NO	NONE	NO	NO	NO	YES	YES	NO	NO	NO	YES	NO	YES	NO	YES	230/150	60	99%,FIO2-40%	20	7.38	110	32	24	94	274	13.8	36	5.38	218	17.7	33	2.17	132	3.5	0.9	ET	Acinetobacter Baumanni	MEROPENEM	10T	15	0	30	10	8	1	7	6	DISCHARGE
29	1196287	JUMASHA MAKANDAR	54	M	SAH-CVA	SURGICAL	NO	YES	NO	CEFOPERAZONE + SULBACTAM	NO	NO	YES	YES	YES	NO	NO	YES	NO	NO	YES	YES	YES	150/80	110	93%,FIO2-80%	32	7.3	96	41.2	18	96	136	13.2	37	4.11	334	16.5	36	1.4	148	4.7	1.3	ET	Acinetobacter Baumanni	Pan resisitant	9T	21	2	44	26	26	0	22	22	DEATH
30	1196361	KIRAN	42	M	CVA	MEDICAL	NO	YES	NO	AMIKACIN	YES	YES	YES	YES	YES	NO	NO	NO	NO	YES	YES	YES	YES	140/90	136	100%,FIO2-70%	30	7.39	272	36.6	21	99	272	12.5	37	4.35	382	28.1	24	1.25	147	4	1.12	ET	Acinetobacter Baumanni	Pan resisitant	6T	20	2	50	28	28	0	12	12	DEATH
31	1196931	ROHIT	31	M	RTA, MULTIPLE FRACTURE	TRAUMA	NO	YES	NO	AMIKACIN, LINEZOLID	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	120/80	93	100% FiO2 - 40%	30	7.42	96	32.6	21.7	99	240	13.6	21	4.11	273	12.1	37	1.1	139	4.2	1.3	TT	Acinetobacter Baumanni	Pan resisitant	10T	8	1	24	46	37	2	23	21	DISCHARGE
32	1197072	VEENA SAPPANDI	40	F	CVA-SAH	SURGICAL	NO	YES	NO	LINEZOLID	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	140/90	118	97%,FIO2-100%	26	7.3	96	31	16	96	96	12.2	40	3.96	380	20.3	37	0.53	142	4.7	0.9	ET	Acinetobacter Baumanni	Pan resisitant	9T	18	1	37	31	21	0	7	7	DISCHARGE
33	1197892	BALKRISHNA DAMBAL	62	M	CVA	SURGICAL	NO	YES	NO	FOPERAZONE + SULBACTAM, TIGECYCLI	NO	NO	YES	YES	YES	NO	NO	NO	YES	YES	NO	YES	YES	160/90	130	96%,FIO2-40%	40	7.2	88	32	13	94	180	10.7	30	3.38	47	9.8	10	0.36	138	4	>10	ET	Acinetobacter Baumanni	Pan resisitant	3T	29	2	69	46	46	0	14	14	DEATH
34	1203120	TANAJI GANAPATI	68	M	CVA-EDH, SEPSIS	TRAUMA	NO	YES	NO	CLINDAMYCIN	NO	YES	YES	YES	YES	NO	NO	YES	YES	YES	YES	NO	YES	130/80	88	97%,FIO2-50%	30	7.41	93	38	24	97	180	8.8	36	2.86	86	8.2	106	4.48	143	3.7	1.02	ET	Acinetobacter Baumanni	Pan resisitant	7T	27	2	59	43	43	3	33	30	DEATH
35	1205418	PARASHURAM PENDARI	45	M	COPD	MEDICAL	NO	YES	NO	NONE	NO	NO	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	130/80	100	97%,FIO2-50%	28	7.4	136	37	21	97	247	15.4	47	3.94	97	13.6	37	1.9	140	4.1	1.2	ET	Acinetobacter Baumanni	METHOPRIM/SULFAMETHOXAZOLE,	10T	9	1	18	8	6	0	4	4	DISCHARGE
36	1206110	SAJJAN SHAVANTEVVA	56	M	PULMONARY TB	MEDICAL	NO	YES	NO	COLISTIN, CLINDAMYCIN	NO	YES	NO	NO	YES	NO	NO	YES	NO	YES	YES	YES	NO	100/70	112	90%,FIO2-100%	30	7.27	83	51	19	93	83	11	37	4.2	328	20.7	22	0.57	133	4.1	0.91	ET	Acinetobacter Baumanni	N, CEFEPIME, GENTAMYCIN, LEVOF	10T	27	1	36	7	6	1	4	3	DISCHARGE
37	1206554	MONOHAR B.	65	M	POST STROKE SEIZURE	MEDICAL	NO	YES	YES	COLISTIN	YES	NO	YES	YES	YES	NO	NO	NO	YES	NO	YES	YES	YES	90/60	136	93%,FIO2-100%	47	7.1	72	36	12	90	72	8	27	2.92	79	27	117	4.27	134	3.9	2.3	ET	Acinetobacter Baumanni	Pan resisitant	2T	52	3	97	10	10	0	8	8	DEATH
38	1207064	MARUTI G	65	M	SEIZURE DISORDER	MEDICAL	NO	NO	NO	METRONIDAZOLE, CLINDAMYCIN	NO	NO	NO	NO	YES	NO	NO	NO	NO	YES	YES	YES	YES	170/100	126	87%,FIO2-100%	42	7.1	65	22.7	13	91	65	12.6	48	5.23	333	17.1	20	0.7	149	5.2	1.36	ET	Acinetobacter Baumanni	Pan resisitant	2T	42	2	78	17	17	0	14	14	DEATH
39	1207963	ALSABA DODAMANI	19	F	SDH - CVA	TRAUMA	NO	YES	NO	CEFOPERAZONE + SULBACTAM, AIKACIN	NO	NO	YES	YES	YES	NO	NO	YES	YES	NO	NO	NO	NO	80/50	132	93%,FIO2-100%	39	7.26	90	32	17	96	90	7.3	30	2.73	148	14.2	53	1.9	152	5.7	1.8	ET	Acinetobacter Baumanni	Pan resisitant	8T	29	3	74	17	17	0	9	9	DEATH
40	10002300	GONDI MAHESH	54	M	ACA ANEURYSM	SURGICAL	NO	YES	NO	TIGECYCLIINE	NO	NO	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	160/90	67	97%,FIO2-80%	24	7.5	157	53	26	97	194	8.3	42	2.36	155	12.2	23.5	0.66	126	2.9	1.4	ET	Acinetobacter Baumanni	Pan resisitant	6T	23	2	48	13	13	1	10	9	DEATH
41	10003890	HALADKAR KRISHNAJI	18	M	BURNS	TRAUMA	NO	NO	NO	AMIKACIN, PIPERACILLIN	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	100/70	135	100%.FIO2-21%	30	7.34	70	46	24	92	212	12.8	42.9	4.66	349	15.5	14.7	0.83	135	5.2	1.29	ET	Acinetobacter Baumanni	TOBRAMYCIN	10T	8	1	21	8	7	0	5	5	DISCHARGE
42	10005315	YALLAPPA KRISHNA BIRJE	36	M	ABOLIC ENCEPHALOPATHY,	MEDICAL	NO	YES	YES	TIGECYCLIINE	NO	YES	YES	NO	YES	NO	NO	YES	NO	YES	YES	NO	NO	150/80	98	92%, FIO2-80%	30	7.42	121	44	29	98	146	12.7	38.3	4.12	363	15.9	7.4	0.59	122	4.9	1	ET	Acinetobacter Baumanni	TIGECYCLINE, COLISTIN	3T	21	2	46	41	41	14	27	13	DEATH
43	10006128	NASEEM WARIMANI	45	M	SCHWANNOMA	SURGICAL	NO	YES	YES	PIPERACILLIN + TAZOBACTAM	YES	YES	YES	YES	YES	NO	NO	YES	YES	YES	YES	NO	YES	86/60	52	90%,FIO2-100%	42	7.1	82	53	9	90	82	7.9	30	2.36	348	26.3	36.2	1.9	131	4.9	1.2	TT	Acinetobacter Baumanni	Pan resisitant	2T	39	3	82	17	17	0	15	15	DEATH
44	10006980	PARAGOUDA RUDRAGOUDA	58	M	TE ON CHRONIC KIDNEY DIS	MEDICAL	NO	YES	NO	TIGECYCLINE, COLISTIN	NO	YES	YES	NO	YES	YES	YES	NO	YES	YES	YES	YES	YES	150/110	108	90%,FIO2-40%	36	7.28	95	46	20.8	98	220	10.5	30.3	3.27	274	12.9	71.8	2.34	107	3.6	0.83	BAL	Acinetobacter Baumanni	Pan resisitant	2T	35	2	50	30	30	22	28	6	DEATH
45	10007670	UDDAVVA BHIMAPPA	29	F	RTA, MULTIPLE FRACTURE	TRAUMA	YES	NO	NO	CEFTRIAXONE, AMIKACIN	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	110/70	70	99% FiO2 - 40%	18	7.4	110	36	24	99	274	11.1	31.8	3.87	330	15.2	8.1	0.43	133	4.7	1	URAL FL	Acinetobacter Baumanni	COLISTIN	15/15	7	0	18	29	16	4	10	6	DISCHARGE
46	10008138	SANTOSH JADHAV	18	M	TE TRACHEOLARYNGEAL ED	MEDICAL	NO	YES	NO	CEFOPERAZONE + TAZOBACTAM	NO	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	110/70	90	99%,FIO2-40%	20	7.39	149	46	23	94	337	13.3	40	4.79	147	10	35.5	0.96	142	4.4	0.9	ET	Acinetobacter Baumanni	PAN-SENSITIVE	10T	4	0	18	25	18	0	10	10	DISCHARGE
47	10008896	SUSHILA CHOUGALA	62	F	COPD	MEDICAL	NO	NO	YES	CLARITHROMYCIN	NO	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	YES	YES	150/90	58	99%,FIO2-40%	22	7.36	118	36	20	97	178	12	39	4.08	187	5	25	0.7	142	3.9	1	ET	Acinetobacter Baumanni	COLISTIN	10T	9	1	37	13	8	0	6	6	DISCHARGE
48	10010816	DEEPAK NIVRUTHI	45	M	CVA,RVD POSITIVE	MEDICAL	NO	YES	YES	CO-TRIMOXAZOLE	NO	YES	YES	NO	YES	NO	NO	YES	YES	YES	YES	NO	YES	110/90	116	96%,FIO2-100%	37	7.1	123	86	28	99	123	10.2	37	3.3	198	12.5	39.3	1.38	126	5.3	1.3	ET	Acinetobacter Baumanni	Pan resisitant	7T	35	2	60	29	25	4	15	10	DEATH
49	10012018	PANDURANG DATTATRAY	75	M	CVA	MEDICAL	NO	YES	NO	CLINDAMYCIN	NO	NO	YES	NO	YES	NO	NO	NO	NO	YES	YES	NO	YES	180/100	86	96%,FIO2-40%	30	7.32	146	30	17	97	370	10.1	33.8	5.27	228	13.5	55.3	1.54	132	4.8	1.02	ET	Acinetobacter Baumanni	Pan resisitant	2T	30	2	67	7	7	0	7	7	DEATH
50	10012465	LALITA BHANDARI	46	F	GBS	MEDICAL	NO	YES	NO	CEFTRIAXONE	NO	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	YES	120/80	74	97%,FIO2-50%	20	7.4	76	34	20	96	188	12.9	38	4.35	381	8.9	16.8	0.56	132	4.4	1	ET	Acinetobacter Baumanni	Pan resisitant	10T	6	1	28	32	30	10	17	7	DISCHARGE
51	10012968	KRISHNA YALLAPPA	78	M	CVA	MEDICAL	NO	YES	NO	TIGECYCLINE, CLINDAMYCIN	NO	YES	YES	NO	YES	NO	NO	YES	NO	YES	YES	YES	YES	200/110	178	100%,FIO2-100%	28	7.53	307	31	28	99	307	11.8	47	3.94	409	10	28	0.59	126	4.1	1	ET	Acinetobacter Baumanni	COLISTIN	7T	32	2	60	32	32	8	26	18	DEATH
52	10013077	SURAJ MAHADEV	32	M	PANCREATITIS, BOWEL PERI	SURGICAL	NO	YES	NO	COLISTIN, TIGECYCLINE	NO	YES	YES	YES	YES	NO	NO	YES	YES	YES	YES	NO	YES	144/60	113	90%,FIO2-100%	40	7.23	89	66	27.8	96	89	7.2	27	2.35	418	22	33.2	0.59	142	3.5	3.7	ET	Acinetobacter Baumanni	Pan resisitant	2T	37	2	78	10	10	2	9	7	DEATH
53	10015661	SHAID SALIM KHAN	20	M	BURNS	TRAUMA	NO	NO	NO	IPERACILLIN + TAZOBACTAM, AMIKACII	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	130/70	108	100% FiO2 - 40%	24	7.4	153	32	26	99	326	10.2	40	3.47	289	12	27.5	0.65	143	4.2	>10	ET	Acinetobacter Baumanni	Pan resisitant	10T	2	1	20	13	10	1	6	5	DISCHARGE
54	10016150	BHARAT SHANKAR	60	M	HIE, CAD	MEDICAL	NO	YES	NO	NONE	YES	YES	YES	NO	YES	NO	NO	NO	NO	YES	YES	YES	NO	80/60	132	93%,FIO2-100%	37	7.34	92	37	22	99	92	10.6	30	3.99	337	9.2	46.5	0.6	135	4.6	0.9	ET	Acinetobacter Baumanni	COLISTIN	5T	27	3	78	18	18	0	12	12	DEATH
55	10020031	SHIVALINGAPPA MALLAPPA	76	M	PTIC SHOCK, POST TB, ASTH	MEDICAL	NO	YES	NO	CLARITHROMYCIN	NO	YES	YES	NO	YES	NO	NO	YES	NO	YES	YES	YES	YES	150/90	120	91%,FIO2-100%	46	7.4	138	34	21	99	190	12	42	3.88	291	23.7	56.7	1.41	141	3.6	0.9	ET	Acinetobacter Baumanni	Pan resisitant	2T	32	3	78	5	5	3	4	1	DEATH
56	10020166	MADHUKAR SHATTU	55	M	PTIC SHOCK, CELLULITIS, MC	MEDICAL	NO	YES	NO	TIGECYCLINE, AZTREONAM	NO	NO	YES	NO	YES	NO	NO	YES	YES	YES	YES	YES	NO	80/50	130	90%,FIO2-100%	36	7.42	59.8	23.6	15	90	59.8	6.5	21.7	3.4	231	12.5	56.5	1	137	3.7	3.4	ET	Acinetobacter Baumanni	Pan resisitant	2T	36	3	98	13	13	4	11	7	DEATH
57	10020696	IRAGOUDA BASAGOUDA	26	M	DVT, PTE	SURGICAL	NO	NO	NO	COLISTIN	NO	YES	YES	YES	YES	NO	NO	NO	NO	YES	YES	NO	NO	130/80	85	86%,FIO2-100%	37	7.2	80	49	31	92	80	12.7	43	6.17	158	5.1	56.3	1.57	132	4.3	3.83	ET	Acinetobacter Baumanni	Pan resisitant	10T	17	2	34	14	14	4	10	7	DEATH
58	10021078	SATTUPPA RAMLING	54	M	RECURRENT ICH	MEDICAL	NO	NO	NO	CEFTRIAXONE, AMIKACIN	YES	NO	NO	NO	YES	NO	NO	NO	NO	YES	YES	NO	YES	170/90	60	97%,FIO2-50%	21	7.4	147	32	20	99	370	15.5	40	4.68	305	12.8	18	0.76	138	3.5	1	ET	Acinetobacter Baumanni	Pan resisitant	2T	16	3	57	3	3	0	3	3	DEATH
59	10021354	LEELAVATI MOHAN	55	F	CVA	MEDICAL	NO	NO	NO	NONE	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	YES	170/100	62	99% FiO2 - 40%	26	7.4	78	37.2	23.6	99	138	11.7	36.2	4.36	178	13.6	17	0.63	136	4.6	1	ET	Acinetobacter Baumanni	Pan resisitant	10T	8	1	30	14	11	0	7	7	DISCHARGE
60	10021377	BASAVARAJ DEVAPPA	58	M	CONTUSION	MEDICAL	NO	NO	YES	CEFOPERAZONE + SULBACTAM	NO	NO	YES	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	130/70	96	98%, FiO2- 40%	26	7.4	147	36	23	99	362	8.9	25	4.22	216	17	31	1	136	4.7	1	ET	Acinetobacter Baumanni	GENTAMYCIN, TOBRAMYCIN, LEVO	10T	7	0	29	18	13	0	8	8	DISCHARGE
61	10022436	VINOD SAVANT	69	M	EPSIS, MODS,STATUS EPILEF	MEDICAL	NO	YES	NO	TIGECYCLIINE	NO	NO	YES	NO	YES	NO	NO	YES	YES	YES	YES	YES	NO	110/80	120	100%, FiO2 - 100	22	7.4	252	31	21	99	368	12.3	39.3	4.32	370	28.6	47.4	0.9	128	5	1.02	ET	Acinetobacter Baumanni	Pan resisitant	7T	22	2	68	22	22	0	17	17	DEATH
62	10022610	BANDEPPA YALLAPPA	45	M	RTA, RHABDOMYOLYSIS	TRAUMA	NO	YES	YES	LINEZOLID	NO	NO	YES	YES	YES	YES	NO	YES	YES	YES	YES	NO	NO	80/50	140	93%,FIO2-100%	36	6.8	61	39	6.5	86	61	8.3	36	3.68	77	16.5	36.4	5.35	134	5.6	1.7	ET	Acinetobacter Baumanni	Pan resisitant	6T	37	3	78	12	12	0	11	11	DEATH
63	10024234	YALLAPPA FAKIRAPPA TALLAWAR	24	M	RVICAL VERTEBRAE FRACTU	SURGICAL	NO	YES	NO	NONE	NO	NO	YES	YES	YES	NO	NO	NO	NO	YES	YES	NO	NO	90/60	108	96%,FIO2-100%	28	7.36	270	37.8	22.6	99	270	12.7	37	4.8	246	13.1	46	1.9	148	5	1.1	ET	Acinetobacter Baumanni	COLISTIN	2T	27	3	70	74	74	3	62	59	DEATH
64	10025826	SAHIL RAVI PATIL	22	M	RTA, MULTIPLE FRACTURE	TRAUMA	NO	NO	NO	EFOPERAZONE + SULBACTAM, AMIKACI	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	YES	NO	NO	100/60	110	93%,FIO2-50%	34	7.32	106	37	19	99	176	7.8	48	4.32	212	18.6	22.6	1.2	146	4.8	1.1	ET	Acinetobacter Baumanni	COLISTIN, TETRACYCLINE	10T	10	2	34	47	36	0	17	17	DISCHARGE
65	10027533	GANGUBAI NARAYANA	77	F	COPD	MEDICAL	NO	YES	NO	COLISTIN	YES	YES	YES	NO	YES	NO	NO	YES	YES	NO	NO	NO	YES	100/70	101	80%,FIO2-1000%	36	7.2	71	86.3	36	90	71	8.1	30	4.09	43	27.3	143	0.41	130	5.7	0.94	ET	Acinetobacter Baumanni	COLISTIN	2T	40	3	79	16	16	2	6	6	DEATH
66	10027571	ASHOK NIRWANI	40	M	THERMAL BURNS	SURGICAL	NO	NO	NO	IPERACILLIN + TAZOBACTAM, AMIKACII	NO	NO	YES	YES	YES	NO	NO	YES	YES	YES	NO	NO	YES	70/40	146	86%,FIO2-100%	49	7.06	66	24	9.3	90	66	7.3	50	4.73	440	28.2	76	1.7	157	6.2	1.9	ET	Acinetobacter Baumanni	Pan resisitant	6T	43	3	85	18	18	0	13	13	DEATH
67	10027573	SOMANAGOUDA	42	M	THERMAL BURNS	SURGICAL	NO	NO	NO	IPERACILLIN + TAZOBACTAM, AMIKACII	NO	NO	YES	YES	YES	NO	NO	YES	NO	YES	YES	YES	YES	80/60	136	90%,FIO2-100%	46	7.17	80	27	13	92	80	11.1	33	3.73	262	17.1	70	2.1	152	5.3	1.9	ET	Acinetobacter Baumanni	COLISTIN	2T	39	3	88	11	11	0	10	10	DEATH
68	10028539	KIRAN BHIRAPPA	25	M	RTA	TRAUMA	NO	NO	NO	CEFOPERAZONE + SULBACTAM	NO	NO	YES	YES	YES	NO	NO	YES	NO	NO	YES	NO	NO	100/60	90	97%,FIO2-50%	26	7.42	144	46	20	98	286	14.1	39	4.1	186	13.1	28	0.86	139	3.9	1	ET	Acinetobacter Baumanni	COLISTIN	9T	6	2	30	33	24	0	12	12	DISCHARGE