"TO COMPARE CLINICAL PROFILE AND OUTCOMES OF ACINETOBACTER BAUMANII CAUSING INFECTION VS COLONIZATION IN ICU PATIENTS"

BY

REG NO: BG0122010

Dissertation

Submitted to

KAHER, Belagavi, Karnataka

In partial fulfilment
of the requirements for the degree of

M.D.

IN

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE

J. N. MEDICAL COLLEGE
BELAGAVI - 590010. KARNATAKA

SEPTEMBER/ OCTOBER-2025

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Dr. REKHA S PATIL MD
Professor and Head,
Department of General Medicine,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Professor & HOD
Dept. of Medicine
J. N. Medical College, Belagavi

Date : 27/03/2025
Place : Belagavi

Dr.(Mrs.) N.S. MAHANTSHETTI MD
Principal
J. N. Medical College,
Nehru Nagar, Belagavi – 10

PRINCIPAL
Jawaharlal Nehru Medical College
BELAGAVI

Date : 27/03/2025
Place : Belagavi

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Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MoE (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 24-03-2025

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BG0122010
Postgraduate Student,
2022-23 Batch,
Department of General Medicine
J. N. Medical College, Belagavi.

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(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 78 **Date: 18/03/2023**

To.
BG0122010
PG Student in General Medicine
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"TO COMPARE CLINICAL PROFILE AND OUTCOMES OF ACINETOBACTER BAUMANNI CAUSING INFECTION VS COLONIZATION IN ICU PATIENTS"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS

*** ICU - Intensive Care Unit**

*** CDC - Centers for Disease Control and Prevention**

*** WHO - World Health Organization**

*** DNA - Deoxyribonucleic acid**

*** VAP - Ventilator-associated pneumonia**

*** RR - Risk Ratio**

*** AG3 - Acinetobacter genospecies 3**

*** GEIH - Grupo de Estudio de Infección Hospitalaria**

*** OR - Odds Ratio**

*** MDR - Multidrug-resistant**

*** CNS - Central nervous system**

*** TLC - Total leukocyte count**

*** ESR - Erythrocyte sedimentation rate**

*** PCT - Procalcitonin test**

*** RFT - Renal function test**

*** LFT - Liver function test**

*** LAMA - Leave Against Medical Advice**

*** HTN - Hypertension**

*** ST - Sequence types**

*** XDR - extensively drug-resistant**

ABSTRACT

INTRODUCTION:

Acinetobacter baumannii is one of the most significant bacteria causing morbidity and mortality and nosocomial infectons in icu settings and it is also known to cause both infection and sedentary colonization.

Owing to characteristics of patients from whom a.baumannii is isolated,it is often difficult to differntiate colonization from infection. This study aimed to evaluate clinical profile and outcomes of acinetobacter baumanni causing infection vs colonization in icu patients

MATERIALS AND METHODS:

A one year cross sectional study (March 2023-March 2024) was conducted at KLES Dr Prabhakar kore hospital ICU's. A total of 41 patients in which A.Baumanii is detected in blood, urine and sputum culuture are included.

RESULTS:

The mean age of the study participants was 55.05 ± 18.77 years with majority being male (82.9%). The percentage of infection and colonization of A baumannii in ICU patients were 65.9% (n=27) and 34.1% (n=14) respectively. In blood culture among the study participants, 46.4% (n=19), urine culture 12.3% (n=5), and sputum culture 43.9%

(n=18) reported A baumannii. Most common diagnosis among the study participants was respiratory diseases contributing to 39% (n=16). Among the study participants, the mortality rate was 31.7% (n=13), 48.8% (n=20) were discharged and 19.5% (n=8) went LAMA. The discharged rate in infected and colonized groups were 44.4% and 57.1%.The mortality rate in infected and colonized groups were 25.9% and 42.9% respectively and it was statistically significant (p value=0.049).

CONCLUSION:

The Parameters associated with infection by A. baumannii in an endemic situation are the admission at ICU.Most common diagnosis among the study participants was respiratory diseases.Mortality of patients from whom A. baumannii was isolated was influenced by the presence of infection by A. baumannii rather than colonization.

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INTRODUCTION

Acinetobacterr baumanniii emerged as one ofmmost challenging nosocomialo pathogen worldwide, particularly ICU settings. The following Gram-negative coccobacillus has gained significant attention due to remarkable ability of acquiring antimicrobial resistance, persisting in hospital environments causing severe infections in “critically ill patients” .[1] Distinction between true infection and colonization by A. baumannii presents a critical clinical challenge, as it directly influences therapeutic decisions and patient outcomes.

Infection caused by Acinetobacter baumannii defined as apart from isolation of bacteria as a unique microbe of given sample, signs in different studies supported diagnosing infection and no clinical focus could justify the presented features , according to the criteria given by the Centerss for Disease Control and Preventionn (CDC).)iIf the criteria was nottmet as per CDC then that was considered as colonization.

The global burden of A. baumannii infections has been dramatically increased over past three decades and with some regions reporting it as the predominant pathogen in ICU-acquired infections because of it can survive on the both wet andd dry surfaces forr the prolonged periods, coupled withh its natural competence for DNA uptake, has facilitated its emergence as a successful hospital-adapted pathogen.[2] TheWWHO has classifiedd the ccarbapenem-resistantt A. baumanniii ass criticalliimportant pathogen and the urgent need for new therapeuticc approaches and for better understanding of its clinical impact.

The clinical spectrum of A. baumannii ranges from asymptomatic colonization to severe, life-threatening infections. Common manifestations include ventilatorrassociated pneumoniaa, bloodstream infections, wound infections and meningitis. However, the mere isolation of A. baumannii from the clinical specimens does not necessarily indicate infection, as colonization is frequent, particularly in critically ill patients with prolonged ICU stays.[3] This distinction becomes crucial for antimicrobial stewardship, as unnecessary treatment of colonization can contribute to further antimicrobial resistance.

RRisk factors forr acquiring A. baumanniii in the ICU settings can be multifactorial and it includes mechanical ventilation, recent surgical procedure and the presencee of invasivee devices, priorr antimicrobiall therapiess , prolonged hospital stay.[4] Understanding these risk factors is essential for both infection prevention and clinical decision-making. However, the factors that determine whether colonization progresses to infection remain incompletely understood, highlighting the need for better predictive tools and risk stratification methods.

The pathogenesiss of A. baumannii infections involves various factors that contribute to its success as hospital pathogen. These include biofilmm formationn and abilityy to adhere to the epitheliall cellss, iron acquisition systems, an also its outer membrane proteins facilitates survival in hostile environments.[5] Recent research has suggested that certain virulence factors may differ between colonizing and invasive strains, potentially offering new insights into the transition from colonization to infection.

The challenge of differentiating   infection and colonization is particularly pronounced in the critically ill patientss, where traditional markers of infection may be altered by underlying conditions or interventions. Clinical criteria, laboratory parameters, and radiological findings often show considerable overlap between infected and colonized patients.[6] This diagnostic uncertainty can lead to either delayed treatment of true infections or unnecessary antimicrobial therapy for colonization, both of which can adversely affect patient outcomes.

Antimicrobial resistance in A. baumannii has reached alarming levels globally, with many isolates showing resistance to most available antibiotics. The emergence of carbapenem-resistant strains has severely limited therapeutic options, often leaving only polymyxins and tigecycline as effective treatments.[7] This situation emphasizes the importance of accurate diagnosis of true infection to ensure appropriate antimicrobial use while avoiding unnecessary exposure in colonized patients.

The effect off the Acinetobacterr baumanniii onn patient outcomes varies significantly between infection and colonization. While infections are increasingly associated with mortality, prolongedd hospital stay, and the higher healthcare cost and the impact off colonization is less clear.[8] Some studies suggest that colonization may only serving as a reservoir for transmission to other patients and it may predict subsequent infection, while others indicate minimal direct clinical impact of colonization alone.

The development of rapid diagnostic tools and biomarkers to differentiate between infection and colonization has become an active area of research. Traditional clinical criteria and laboratory parameters often lack the specificity needed for accurate distinction .[9] Novel approaches, including molecular diagnostics, proteomics, and machine learning algorithms, are being investigated to improve diagnostic accuracy and guide clinical decision-making.

Prevention and control strategies for A. baumannii in ICU settings must consider both infected and colonized patients as potential sources of transmission. Environmental cleaning, hand hygiene compliance, contact precautions, and active surveillance cultures play crucial roles in controlling spread.[10] However, the resource implications and potential negative impacts of isolation precautions necessitate accurate identification of patients requiring such measures.

AIMS AND OBJECTIVES

Objective:

1. To compare clinical profile and outcomes of acinetobacter baumanni causing infection versus colonization in ICU patients

REVIEW OF LITERATURE

Acinetobacter is gram-negative organism and strictly aerobic, nnon-fermentativee and nonmotile organism andd is oxidase-negative .[11] Acinetobacter includes several species, but A. baumannii has most clinicall significancee.[12] AAcinetobacter lives on both soill andd waterr. The samples can be culturedd fromm urinee, ssaliva, respiratory secretionss, and wounds. This particular organism also known too cause colonization of intravenouss fluids andd the otherr irrigationn solutionss.[13,14]

Difference between Acinetobacter baumannii colonization and infection:[18]

The fundamental difference between A. baumannii colonizationn and infection lies in how bacteria affects the body. During colonization, the bacteria is present on the body but doesn't cause illness, whereas an infection occurs when the bacteria actively causes symptoms and illness. The distinction between these conditions can be observed through specific signs. When iinfected, patients typically presents with the fever and raised white blood cell counts, increased inflammatory markers, and abnormal imaging results.

In contrast, colonization is characterized by an absence of physical findings, and when treated, both clinical and analytical parameters show improvement.

Several risk factors increase the likelihood of both colonization and infection with A. baumannii. These include extended hospital stays, the use of antibiotic therapy, dependence on mechanical ventilation, and the presence of intravascular devices. Additional risk factors encompass advanced age, compromised immune system, recent surgical procedures or invasive interventions, and severe burn injuries. Understanding these risk factors is crucial for healthcare providers to implement appropriate preventive measures.

Regarding treatment approach, medical intervention is only necessary when dealing with active infections. In cases where a patient has an indwelling device and shows no signs of infection, the primary intervention may simply involve replacing the device. This targeted approach helps prevent unnecessary antibiotic use while effectively managing the bacterial presence.

Prevention plays vital role of controlling A.bbaumannii spread, particularly inn healthcare settingsss. Key preventive measures include using sterile water instead of tap water for oral care and consistently using alcohol-based hand antiseptics before and after patient care. Healthcare facilities should also implement comprehensive infection prevention protocols to minimize the risk of bacterial transmission and protect vulnerable patients.

Historicall Perspectives:[19]

The genus Acinetobacter history began in the year 11911 when Dutch microbiologist named Beijerinckk isolated and described the Micrococcuss calco-aceticus from the soil using a calcium-acetateee-containing minimall medium. In the following yearss,ssimilar organisms wereealso discovered anddclassified under various genera and species, with atlleast a count of Fifteen different classifications includinggorganisms such asDDiplococcus mucosuss, Alcaligenes haemolysan’s , Mima polymorphaa, and several others.

Thee namee "Acinetobacter," iss derivedd fromm a Greekk wordd called “akinetos” meaning a nonmotile which was first proposed by Brisouu and Prévot in the year 1954. This designation was intended to distinguish nonmotile microorganisms from motile ones within the Achromobacter genus. However, the name didn't gain widespread acceptance until 1968, when Baumannnandd his colleagues conductedd a comprehensive studyy concluding that previously identified species belongeddto single genus, Acinetobacter.

The genus received official recognition in the year 1971 by a Subcommitteeeon Taxonomy of Moraxellaaand AAllied Bacteria. By 1974, BergeysMManual of the Systematic Bacteriologyylisted genus with single species with name Acinetobacterccalcoaceticus.

Etiologyy:

Even though the Acinetobacter is mostly found as hospital pathogen andd issisolated patients who are admitted in hospitals, caree shouldd bee taken in telling if the isolate is the ccause orr it is only because of colonization. Acinetobacterr is a Wwater-lovingoorganism and has abitlity tto ccolonize thee bbody organs thatt contains fluidd. So in patientss, Acinetobacterr iss foundd in peritoneall fluid, cccerebrospinal fluid, saliva, respiratoryy secretion and urinaryy ttract.[20,21]

Epidemiologyy:

Acinetobacter has gained cclinical importance inn year 11960s with the increasing growth of the ICUs at hhospitals.[22] Thoughh AAcinetobacter is off low virulencee, its ability too survivee and and persist in the environmentt for prolonged durationn of time makess it an easily aquired inn healthcaree settings. The organism hhas llow virulencee butt it iss capablee off causingg infectionn in patientss wwith ffebrile nneutropenia andd organ transplant patients.[23]

Acinetobacter infections are more common in India, especially inn hospitals and iintensive care units (ICUs). Acinetobacter is a Gram-negativee bacteria that colonizes the skinn, wounds, respiratory tracttand GIT. It can also cause iinfections like ppneumonia, meningitiss, andd bloodstream iinfections. The prevalence of the Acinetobacter baumanniii inn India varies depending on study and sample population. Some studies have found that AAcinetobacter baumannii is prevalent inn Indian ICUs, while others have found that it is prevalent in sputum samples.

Prevalence in ICUs:

- Acinetobacter infections in Indian ICUs ranged from 12–41%.[24]
- Another study found that 83% of patients with mechanical ventilation or an endotracheal tube were predisposed to Acinetobacter baumannii.[25]

Prevalence in sputum samples:[26]

- One study found that prevalence of Acinetobacter baumanniii in the sputum samples was highest in age group of 1–10 yyears.
- Another studyy found that prevalence of Acinetobacterr baumannii in the sputum samples was 29.65%.
- Prevalence in other samples[26]
- One study found that prevalence of Acinetobacter baumanniii in samples was 70.2%.

Antibiotic resistance

- Acinetobacter baumannii is naturally multidrug resistant.
- Some studies have found that Acinetobacter baumannii isolates are resistant to cefotaxime, ceftazidimee, cefepime, imipenemm, meropenemm, amikacin, ciprofloxacinn,trimethoprim-sulfamethoxazole,gentamicin,levofloxacinn, piperacillin-tazobactam, ampicillin-sulbactam.

Histopathologyy:[31]

The infection which is ccaused by Acinetobacterr quite same in hhistopathology to anyy otherr gram-negativee bacillii.

Clinicall Significance:[32]

A. baumanniiii causess a variety off infections in both the hospital and community , including skinn andd the softt tissuess, urinary tract infection, meningitiss, and pneumoniaa, with the latter being mostt commonly found infection in bothh the settings. Nosocomial infections are frequently observed in the ccritically ill patientss and risk factors for developing an *A.* baumanniii infection includes extended hospital stayss, immunee ssuppression, advancedd age, presencee of other comorbidd conditions .Due to poor prognosis of the critically ill patients who get A. baumanniii infections, itt is very ddifficult to obtain a definitive mortality rates; however the ccrude morality rrates ranges from 23 to 68%%.

Transmission:[33]

Acinetobacterr species primarily spread in healthcare settings through contaminated environmental surfaces and healthcare workers' hands. However, airborne transmission has also been documented, with notable cases including a healthcare worker contracting severe pneumonia after exposure to aerosolized A. baumannii during patient care. Studies have shown varying rates of air contamination in patient rooms, with some reporting up to a quarter of air samples containing carbapenem-resistant A. baumannii. The potential for airborne transmission suggests that traditional surface cleaning may be insufficient, and new approaches like air disinfection technologies, patient cohorting, and respiratory secretion control may be necessary.

Contrary to common belief, Acinetobacter infections are not primarily linked to immunocompromised status. Instead, the main risk factors include exposuree to broad-spectrum antibiotic, disruption of natural barrierss (such as through catheters or endotracheal tubes), and high colonization pressure. The bacteria commonly affect patients in ICU settings, particularly those who are on mechanical ventilationn, with invasive devices, or recovering from surgery, burns, or trauma. These factors create opportunities for infection primarily through compromised physical defenses and disrupted normal flora rather than immune system deficiencies.

Acinetobacter's remarkable resistance to desiccation contributes to its persistence in healthcare environments. Interestingly, community-acquired infections, particularly pneumonia and bacteremia, occur more frequently in tropical climates with high humidity. There is also a seasonal pattern to infections, with U.S. surveillance data showing a 54% increase in cases between July and October compared to the rest of the year. Environmental factors such as humidifiers and water baths often serve as reservoirs, with high humidity believed to promote bacterial growth.

Diagnosis:

Most patients who are being infected with Acinetobacterr are in the hospital and most commonly involved organ is llung, primarily because of respiratory equipment used for mechanical ventilation and colonization of airways

Pneumoniaa, wound infectionn, catheterr-associatedd bacteremiaa, or nosocomiall meningitiss have all been associated with Acinetobacterr.[34]

Theree aree no ffindings pathognomic to AAcinetobacter infections and often needed to be isolated from other organisms such as eEnterobacter, BBurkholderia, Pseudomonass, and Serratiaa which are gram negative organisms a. Since

AAcinetobacter is mainly causing colonization the treating doctor hass responsibility to show evidence thatt itt iss one that is responsible for pathology in given clinical scenario.

Laboratory Studiess: LLeukocytosis can be seen withh lleft shift.[35] But findings aree nott specific and it always doesn't represent that there is infection Whenn outbreak of Acinetobacter iss observed , the organism is readily isolated andd culturedd from ddifferent fluids from body.

Imaging Studiess: If pneumonia is suspected a chest x-ray is required. Depending on signs and symptoms other imaging tests are obtained

Proceduress: The CSF needs to be analyzed and cultured ,if meningitis is suspected,

HistologiccFindings: There are histopathological features which are specific of Acinetobacterr infection which can be differentiated from other gram-negativee bacillii.

Treatmentt:

Acinetobacterr is multidrugg resistantt and antibiotics such as cephalosporins, penicillins, and macrolides have very littlee orr noo anti-Acinetobacterr activities. When we use these antibiotics it mayy predisposee Acinetobacter infection. If we suspect infection in long-term catheterr or a ppacemaker, it has be removedd.[36]

Colonization usually should not be treated ass itt can lead to moree antibioticc resistancee.

Antibiotics which can be effectivee include meropenemm, sulbactamm, colistinn, polymyxinnB, andd amikacinn. Other alternativess include minocyclinee, rifampinnandd tigecyclinee. Treatment with Monotherapyy is sstandard and combination therapy has shown to be more effective. The duration of therapy can be given from seven to ten days, depending onn the type of patients condition.

REVIEW OF RELATED ARTICLES:

In a study by Uwingabiye J et al,[38] among total of 9644 patients hospitalizedd in Intensive care Units, eight one (i.e:8.4%) developed Acinetobacter infectionss. The Multivariatee logisticc regressionn analysis described the independent risk factors for ICU-acquired Acinetobacter baumannii infections: ICU stays ≥14 days (odds ratio (OR)=6.4), prior use of central venous catheters (OR=18), prior use of mechanical ventilation (OR=9.5), duration of invasive procedures ≥7 days (OR=7.8), previous exposure to antibiotics like imipenem (OR=9.1), previous exposure to amikacin (OR=5.2), previous exposure to antibiotic polytherapy (OR=11.8) and previouss exposuree too corticosteroid therapy (OOR=5). On the other hand, the admission for post-operative care was identified as a protective factor. The crude mortality in patients with *A. baumannii* infection was 74.1%. Multivariate analysis showed that septic shock (OR=19.2) and older age (≥65 years) (OR=4.9) were significantly associated to mortality risk in patients with *A. baumannii* infection.

Sileem AE et al[37] in their study, nosocomial respiratory tract infections were the commonest (79.5%), followed by urinary tract infections (14.1%) in relation to other nosocomial infection. Acinetobacter infection was statistically significant among all the infections ($P < 0.05$). The mortality from acinetobacter infection group (1) was 50% and from colonization group (2) was 13.6% with overall mortality 30%. The antibiotics such as tigecyclin and colistin showed high curability and high sensitivity rate ($p < 0.01$). The mortality rate, comorbidities, ICU stays (days) after acinetobacter, CRP and WBCs were the significant predictors for group (1) patients**.**

Molina J et al[39] in their study, two hundred twenty-one isolates of *Acinetobacter baumannii* and 15 of *Acinetobacter* genospecies 3 (AG3) were consecutively collected in a 30-day period during the nationwide project GEIH-Ab2000. Nosocomial acquisition (*P* = 0.01), intensive care unit admission (*P* = 0.02), and antibiotic pressure (*P* = 0.03) were observed to be lower in the AG3 group. AG3 isolates were more frequently implied in wound infections (*P* = 0.05), while *A. baumannii* tended to be recovered from respiratory samples (*P* = 0.08). To our knowledge, this is the first report analyzing the clinical differences among *Acinetobacter* genospecies, with our findings suggesting that clinical features of AG3 may not be equivalent to those traditionally described for *A. baumannii*.

In a study by Mathai A et al,[24] *Acinetobacter* infection occurred in 94 patients (108 episodes). The most common site of infection was the respiratory tract (83 patients, 76.85%), with medical patients being more susceptible than surgical patients to *Acinetobacter* lung infections (*P* = 0.04), particularly late-onset ventilator-associated pneumonia (VAP) (*P* = 0.04). The majority (63.8%) of infections were acquired in the ICU, and patients with ICU acquired infections were intubated significantly longer than the other patients (*P* = 0.02). Seventy percent of the infections were caused by multidrug-resistant (MDR) strains, and the overall crude mortality rate was over 70%. The most important factors affecting mortality were the duration of intubation (*P* = 0.001) and the inappropriate use of antibiotics (*P* = 0.021) after diagnosis of the infection.

Montero JG et al[40] found that in 2000 and 2010, 103 and 108 patients were included, and the incidence of *A. baumannii* colonization/infection in the ICU decreased in 2010 (1.23 *vs.* 4.35 cases/1000 patient-days; $p<0.0001$). No differences were found in the colonization rates (44.3 *vs.* 38.6%) or infected patients (55.7 *vs.* 61.4%) in both periods. Overall, 30-day mortality was similar in both periods (29.1 *vs.* 27.8%). The rate of pneumonia increased from 46.2 in 2000 to 64.8% in 2010 ($p<0.001$). Performing MSLT, 18 different sequence types (ST) were identified (18 in 2000, 8 in 2010), but ST2 and ST79 were the predominant clones. ST2 isolates in the ICU increased from 53.4% in the year 2000 to 73.8% in 2010 ($p=0.002$). In patients with *A. baumannii* infection, the multivariate analysis identified appropriate antimicrobial therapy and ST79 clonal group as protective factors for mortality.

Guddeti PK et al[25] in their study, a total number of 168 *Acinetobacter* species including 143 *A. baumannii* were isolated from the various clinical specimens, the majority of the isolates were obtained from the respiratory system (66%), followed by urine, pus/wound swab, blood, fluids and other samples. The majority of the patients who had underlying/diagnosed with a disease such as aspiration pneumonia/pneumonia (35%), cerebrovascular accident/haemorrhagic shock (30.7%), respiratory failure (24%), accelerated HTN/HTN(18%), and less common were septicemia (8.4), acute kidney injury/chronic kidney diseases (7.7%) and trauma/burns (5.5%). The antibiotic susceptibility testing showed higher antibiotic resistance to cefotaxime (94%), ceftazidime (93%), cefepime (92%), imipenem (92%), meropenem (90%) and the resistance was low to doxycycline (39%) Polymyxin B (8%). The association between antibiotic resistance and the clinical profile of patients was found significant (p-value < 0.05). In our study, a remarkably high antibiotic resistance pattern was observed in the classes of antibiotics in *A.*

baumannii isolates, mostly MDR and XDR. To address infection caused by antibiotic-resistant *A. baumannii*, appropriate antibiotic administration in a clinical setting is essential. Moreover, local and national surveillance data, stringent infection control, and antimicrobial stewardship are required.

The critical importance of distinguishing between Acinetobacter baumannii infection and colonization in ICU patients, as this distinction significantly impacts patient management and outcomes. Studies consistently demonstrate that while both conditions share common riskk factorss suchh as extended ICU stays, mechanical ventilation, and broad-spectrum antibiotic exposure, their clinical trajectories and mortality rates differ substantially. Infected patients typically require aggressive antimicrobial intervention and show higher mortality rates compared to colonized patients, who often improve without specific antibiotic therapy. This distinction becomes particularly crucial in an era of increasing antimicrobial resistance, where judicious use of antibiotics is paramount. The evidence suggests that implementing robust surveillance protocols, utilizing standardized criteria for differentiating infection from colonization, and adopting preventive measures can significantly improve patient outcomes while promoting antimicrobial stewardship. Future research should focus on developing more rapid and accurate diagnostic tools to differentiate between these conditions, thereby enabling more targeted and effective therapeutic approaches in critical care settings.

MATERIALS AND METHODS

Source of data

Patients admitted to ICU wards of KLE’s Dr Prabhakar Kore Hospital, Belagavi and fulfilling the inclusion and exclusion criteria.

Study period

March 2023 to March 2024

Study design

Cross-sectional study

Sample size: 41

Inclusion criteria

- Patients above 18 years of age
- Patients in which A baumannii was detected in culture samples of blood, urine, sputum samples in ICU patients

Exclusion criteria

- Patients who are not willing to participate in the study.
- Patients with Acinetobacter Baumannii detected in culture samples of blood, urine, sputum and not in ICU

Definition of infection and colonization

Infection by A. baumannii was defined when, aside from the isolation of the bacteria as the unique microbe in a clinical sample, clinical signs or abnormalities in the complementary studies supported the diagnosis of infection and no other clinical focus could justify the clinical manifestations, according to the criteria defined by the Centers for Disease Control and Prevention (CDC). If these criteria were not fulfilled, the case was considered colonization.

Study protocol

- Patients older than 18 years from whom Acinetobacter baumannii was isolated from clinical samples from 2 days after patients who got admitted in KLE's ICU ward during the year 2023 and 2024.
- Culture samples like sputum, urine and blood were collected.
- Cases were divided into infection and colonization as defined earlier and were followed from day 2 admission till the patient stays in hospital
- All the participants fulfilling the inclusion criteria with willingness to participate were included with their consent taken and protocol explained.

RESULTS

A total of 41 study participants were included in the study. In total 41, 65.9% (n=27) were infected and 34.1% (n=14) were colonized with A baumannii.

Table 1: Infected and colonized study participants

CATEGORY	NUMBER	PERCENTAGE
Infected	27	65.9%
Colonized	14	34.1%

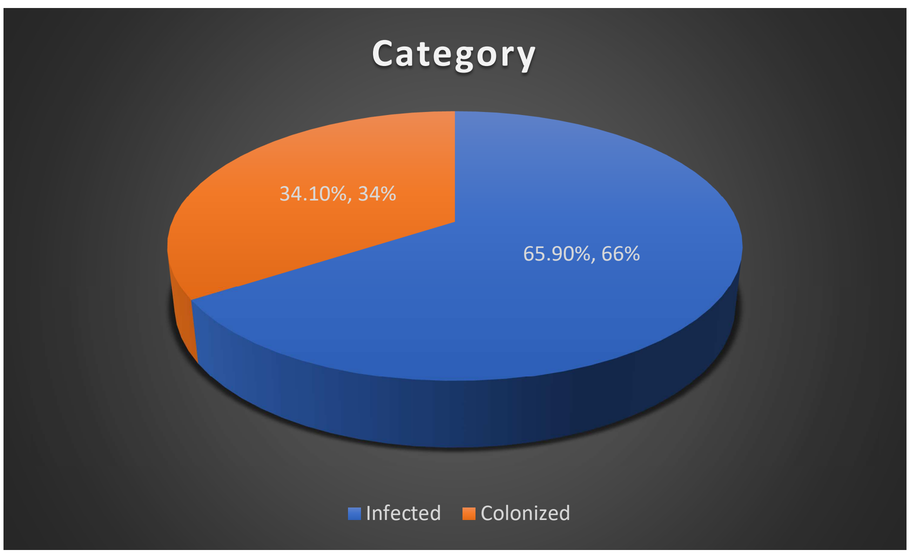

Figure 1: Category of study participants

Age

The mean age of the study participants was 55.05 ± 18.77 years. Distribution of age among study participants was reported in the Table below.

Table 2: Age distribution among study participants

AGE (YRS)	NUMBER	PERCENTAGE
19-40	12	29.3%
41-60	13	31.7%
61-80	12	29.3%
Above 80	4	9.8%

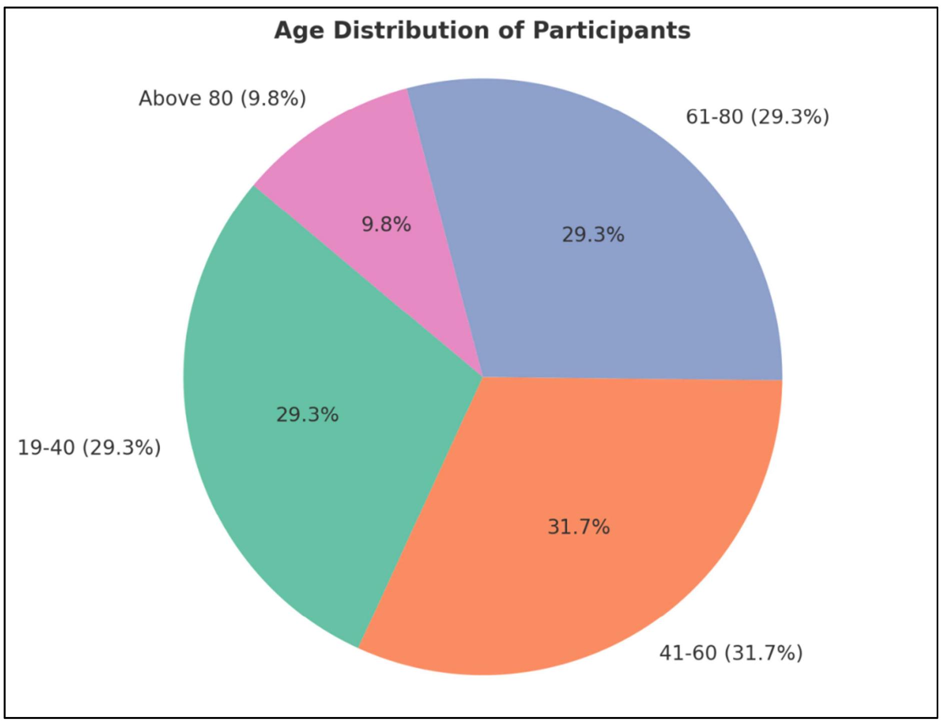

Figure 2: Distribution of age among study participants

The age distribution among the infected and colonized study participants were given in the table below.

Table 3: Age distribution among infected and colonized study participants

AGE	INFECTED	COLONIZED	P VALUE
19-40	8 (29.6%)	4 (28.6%)	0.691
41-60	10 (37%)	3 (21.4%)	
61-80	7 (25.9%)	5 (35.7%)	
Above 80	2 (7.4%)	2 (14.3%)	

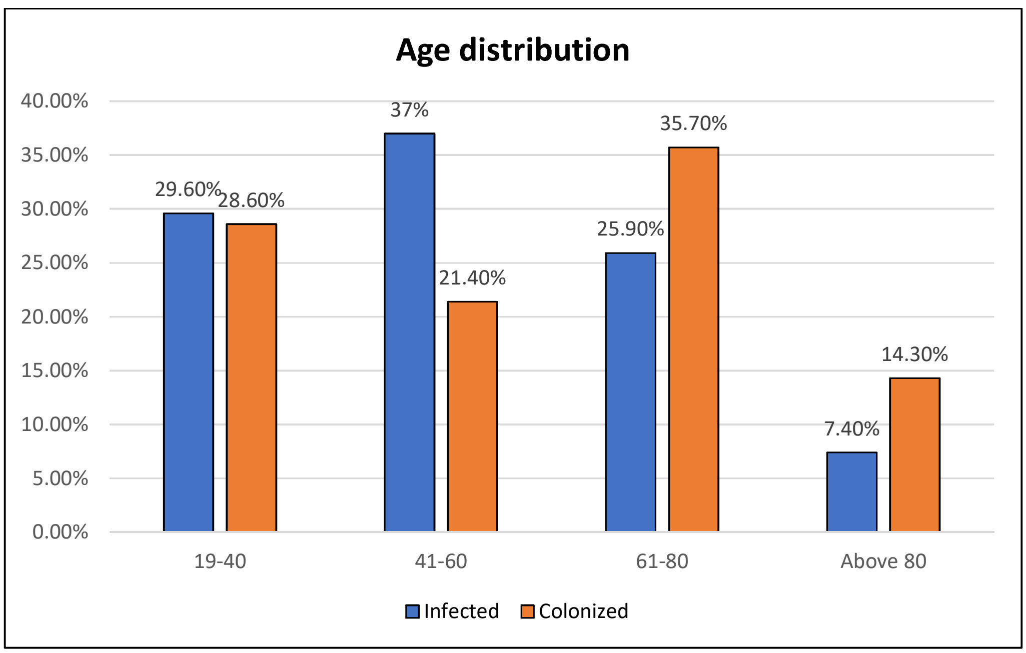

Figure 3: Age distribution among infected and colonized study participants

Gender

Among the study participants, 82.9% (n=34) were males and 17.1% (n=7) were females.

Table 4: Gender distribution among study participants

GENDER	NUMBER	PERCENTAGE
Male	34	82.9%
Female	7	17.1%

The gender distribution among the infected and colonized study participants was given in the table below.

Table 5: Gender distribution among infected and colonized study participants

GENDER	INFECTED	COLONIZED	P VALUE
Male	23 (85.2%)	11 (78.6%)	0.449
Female	4 (14.8%)	3 (21.4%)	

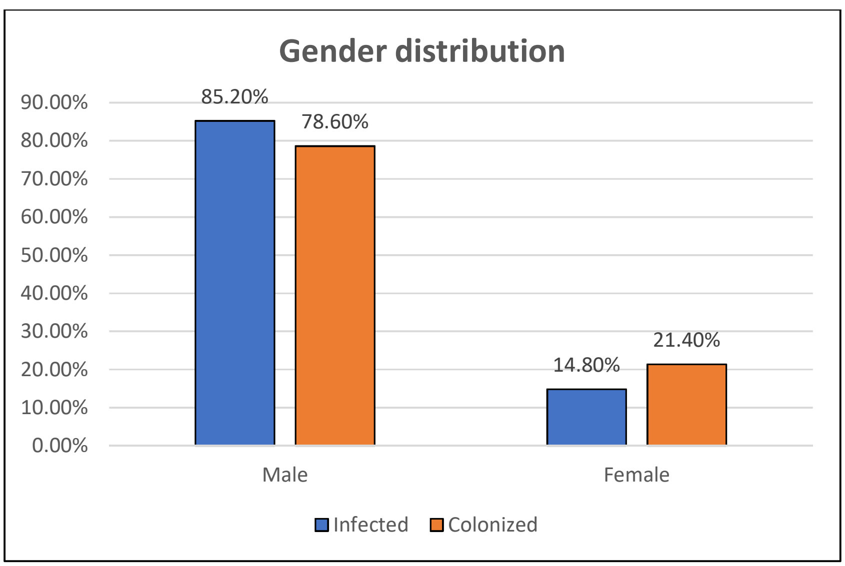

Figure 4: Gender distribution among infected and colonized study participants

Blood culture

In blood culture among the study participants, 46.4% (n=19) reported A baumannii. Other organisms were reported as 7.2% (n=3) and 46.4% (n=19) showed no growth.

Table 6: Organisms in blood culture

BLOOD CULTURE	NUMBER	PERCENTAGE
A baumannii	19	46.4%
Burkholderia cepacia	1	2.4%
Coagulase negative Staph sp.	1	2.4%
K pneumonia	1	2.4%
No growth	19	46.4%

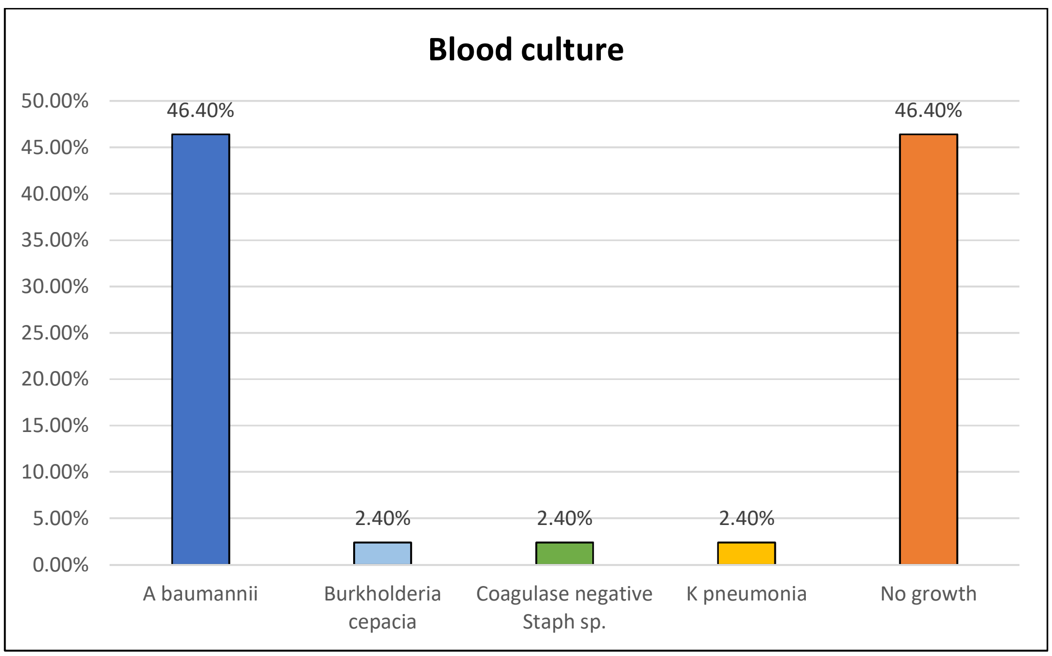

Figure 5: Blood culture report

Urine culture

In the urine culture, A baumannii was reported in 12.3% (n=5). Other organisms were reported as 14.5% (n=6) and no growth was reported in 73.2% (n=30) study participants.

Table 7: Organisms in urine culture

URINE CULTURE	NUMBER	PERCENTAGE
A baumannii	5	12.3%
Candida tropicalis	1	2.4%
Enterobacter cloaca	1	2.4%
Enterococcus feacialis	2	4.9%
K pneumonia	1	2.4%
S. agalagictae	1	2.4%
No growth	30	73.2%

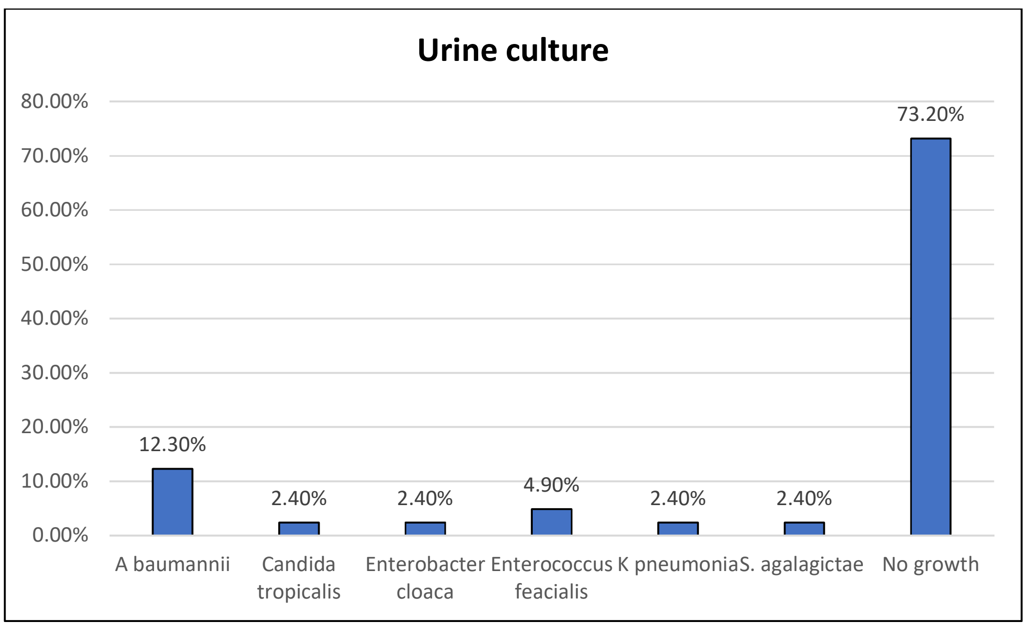

Figure 6: Organisms in urine culture

Sputum culture

In sputum culture, A baumannii was reported in 43.9% (n=18). Other organisms were reported as 12.1% (n=5) and no growth was reported in 46.4% (n=19).

Table 8: Organisms in sputum culture

SPUTUM CULTURE	NUMBER	PERCENTAGE
A baumannii	18	43.9%
E coli	1	2.4%
Pseudomonas aeruginosa	1	2.4%
Staphylococcus hemolyticus	1	2.4%
K pneumonia	2	4.9%
No growth	19	46.4%

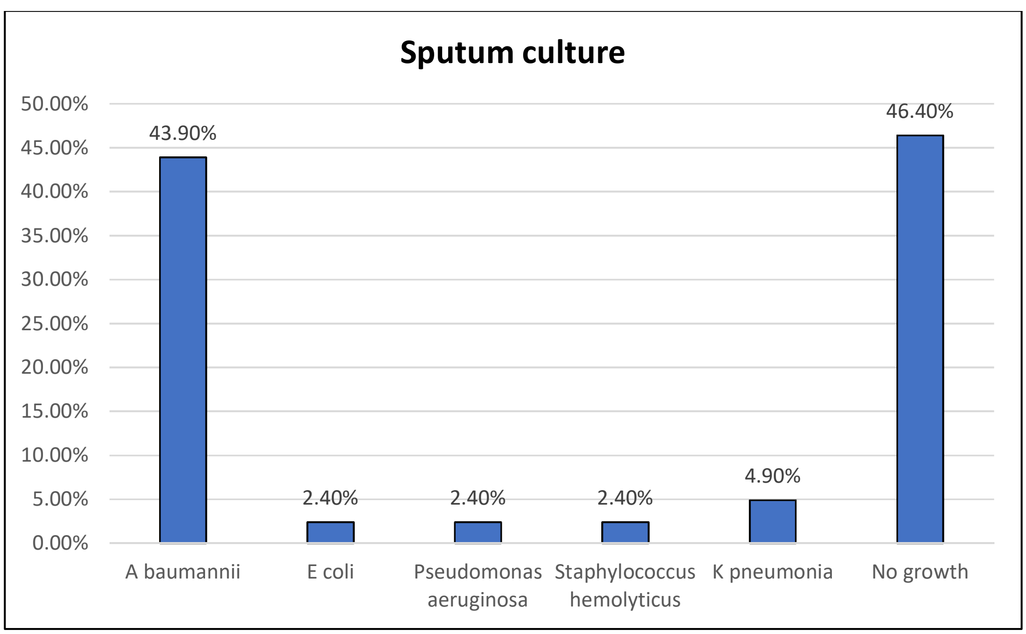

Figure 7: Organisms in sputum culture

Diagnosis

Most common diagnosis among the study participants was respiratory diseases contributing to 39% (n=16), followed by sepsis contributing to 36.6% (n=15). Other diseases include 14.6% (n=6) of central nervous system, 12.2% (n=5 of multiorgan failure, 7.3% (n=3) of liver disease, 4.9% (n=2) of gastrointestinal and pancreatitis each, and 2.4% (n=1) of cardiac, diabetes, organophosphorus poisoning and renal disease each.

Table 9: Diagnosis

DIAGNOSIS	NUMBER	PERCENTAGE
Multiorgan failure	5	12.2%
Cardiac	1	2.4%
Pancreatitis	2	4.9%
Respiratory disease	16	39%
CNS	6	14.6%
Liver disease	3	7.3%
Gastrointestinal	2	4.9%
Diabetic ketoacidosis	1	2.4%
Organophosphorus poisoning	1	2.4%
Sepsis	15	36.6%
Renal	1	2.4%

Distribution of diagnosis in infected and colonized study participants were given in the table below.

Table 10: Distribution of diagnosis among infected and colonized groups

DIAGNOSIS	INFECTED	COLONIZED	P VALUE
Multiorgan failure	3 (11.1%)	2 (14.3%)	0.564
Cardiac	1 (3.7%)	0 (0%)	0.659
Pancreatitis	1 (3.7%)	1 (7.1%)	0.572
Respiratory disease	10 (37%)	6 (42.9%)	0.487
CNS	4 (14.8%)	2 (14.3%)	0.672
Liver disease	3 (11.1%)	0 (0%)	0.274
Gastrointestinal	1 (3.7%)	1 (7.1%)	0.572
Diabetes	1 (3.7%)	0 (0%)	0.659
Organophosphorus poisoning	1 (3.7%)	0 (0%)	0.659
Sepsis	7 (25.9%)	8 (57.1%)	0.053
Renal	1 (3.7%)	0 (0%)	0.659

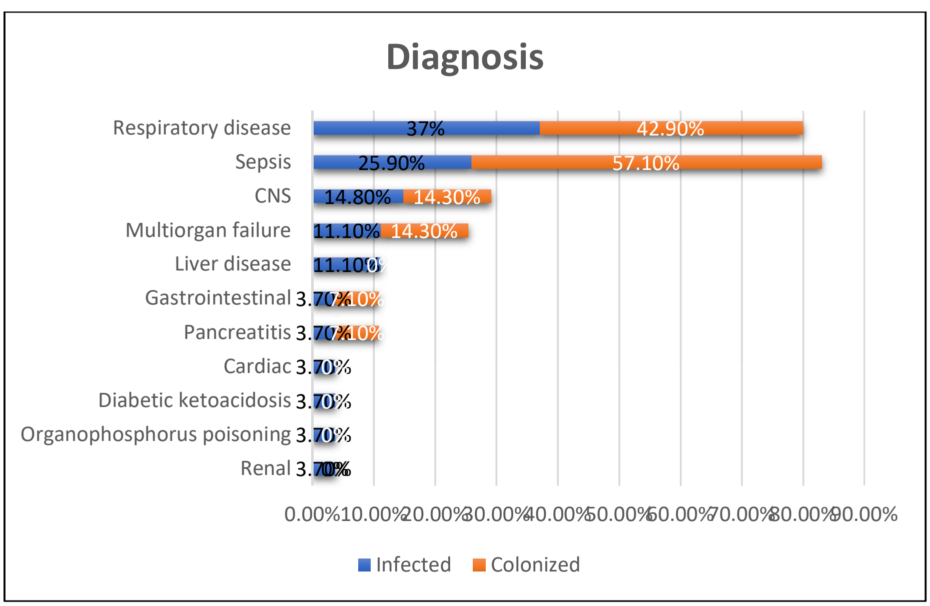

Figure 8: Distribution of diagnosis in infected and colonized groups

Clinical investigations

Hemoglobin

Normal hemoglobin levels (12-17) were reported in 7 (17.9%) of the study participants. Abnormal (<10) and borderline (10-12) hemoglobin levels were reported in 21 (53.8%) and 11 (28.2%) of the study participants.

Table 11: Hemoglobin among infected and colonized groups

HEMOGLOBIN	INFECTED	COLONIZED	TOTAL	P VALUE
Normal	7 (26.9%)	0 (0%)	7 (17.9%)	0.060
Borderline	8 (30.8%)	3 (23.1%)	11 (28.2%)	
Abnormal	11 (42.3%)	10 (76.9%)	21 (53.8%)	

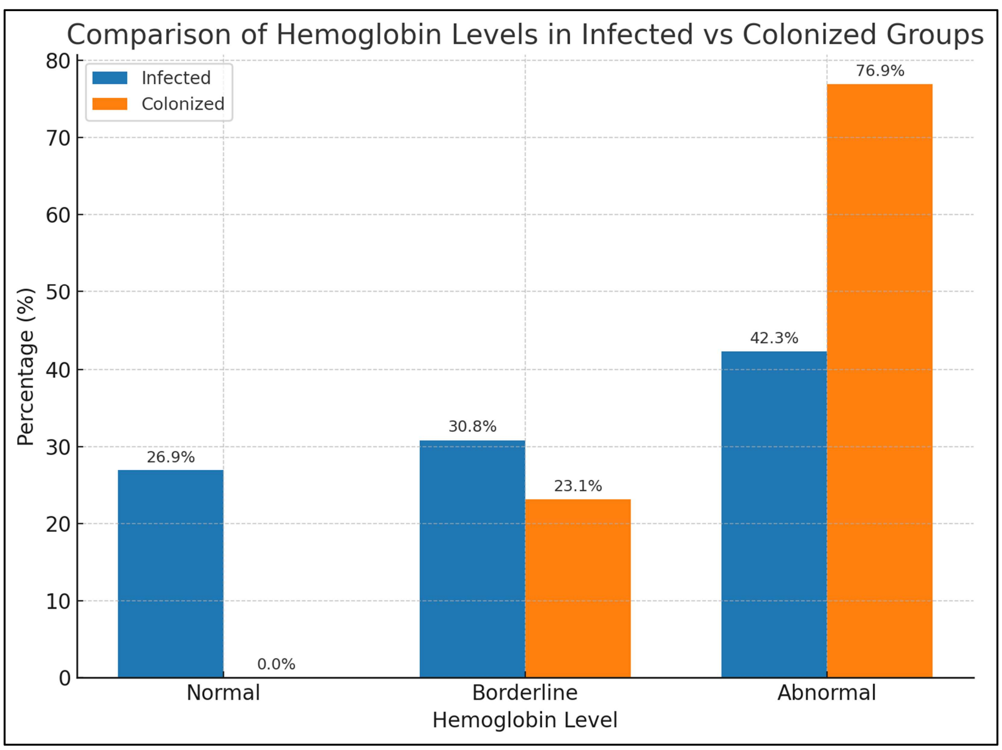

Figure 9: Comparison of hemoglobin levels in infected vs colonized groups

Total leukocyte count

The normal total leukocyte count (TLC) was observed in 8 (19.5%) of the study participants and abnormal total leukocyte levels were observed in 33 (80.5%) of the study participants.

Table 12: TLC levels in infected and colonized groups

TLC	INFECTED	COLONIZED	TOTAL	P VALUE
Normal	4 (14.8%)	4 (28.6%)	8 (19.5%)	0.257
Abnormal	23 (85.2%)	10 (71.4%)	33 (80.5%)	

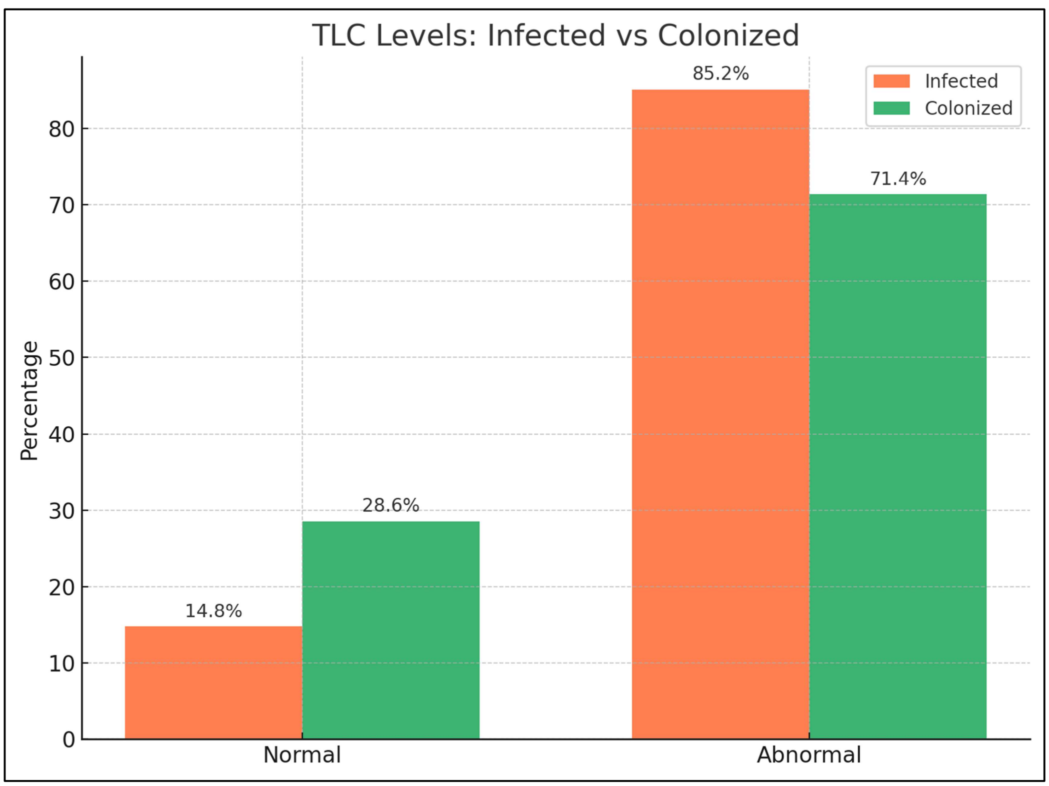

Figure 10: TLC levels in infected and colonized groups

Erythrocyte sedimentation rate

Erythrocyte sedimentation rate (ESR) was normal in 29.3% (n=12), borderline in 58.5% (n=24) and abnormal in 12.2% (n=5).

Table 13: ESR among infected and colonized groups

ESR	INFECTED	COLONIZED	TOTAL	P VALUE
Normal	8 (29.6%)	4 (28.6%)	12 (29.3%)	0.202
Borderline	14 (51.9%)	10 (71.4%)	24 (58.5%)	
Abnormal	5 (18.5%)	0 (0%)	5 (12.2%)	

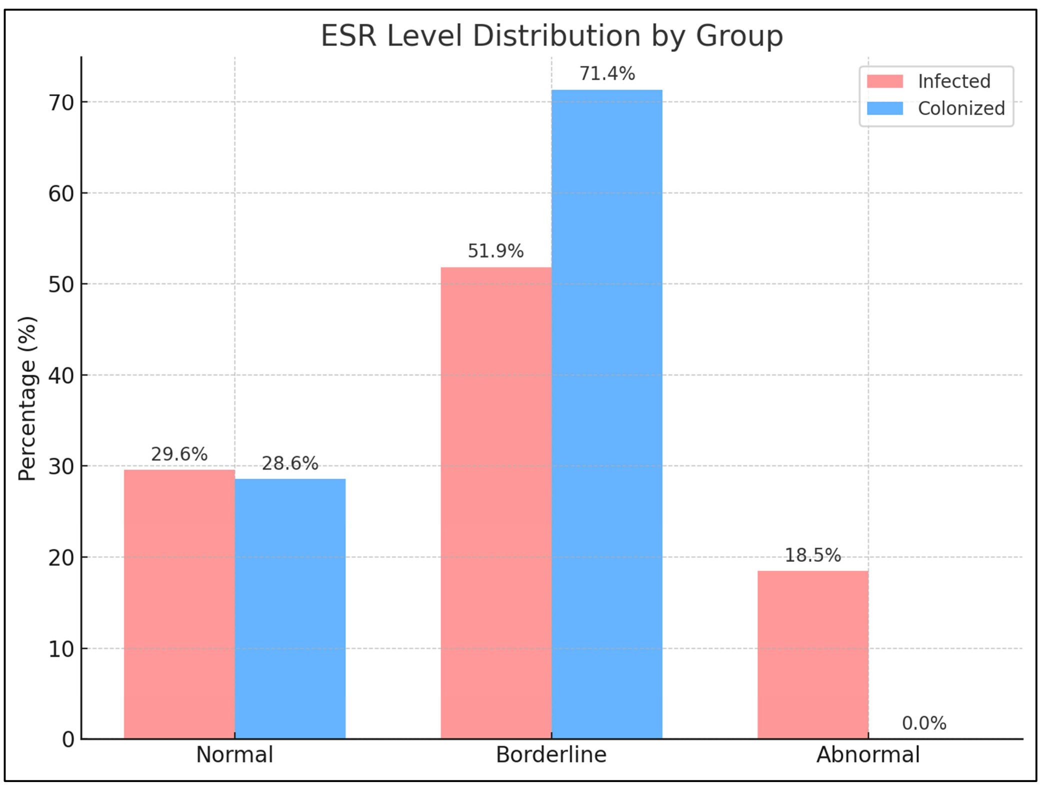

Figure 11: ESR levels in infected and colonized groups

Procalcitonin test

Procalcitonin test (PCT) reported 92.7% (n=41) abnormal levels and 7.3% (n=3) normal levels.

Table 14: PCT among infected and colonized groups

PCT	INFECTED	COLONIZED	TOTAL	P VALUE
Normal	2 (7.4%)	1 (7.1%)	3 (7.3%)	0.735
Abnormal	25 (92.6%)	13 (92.9%)	41 (92.7%)	

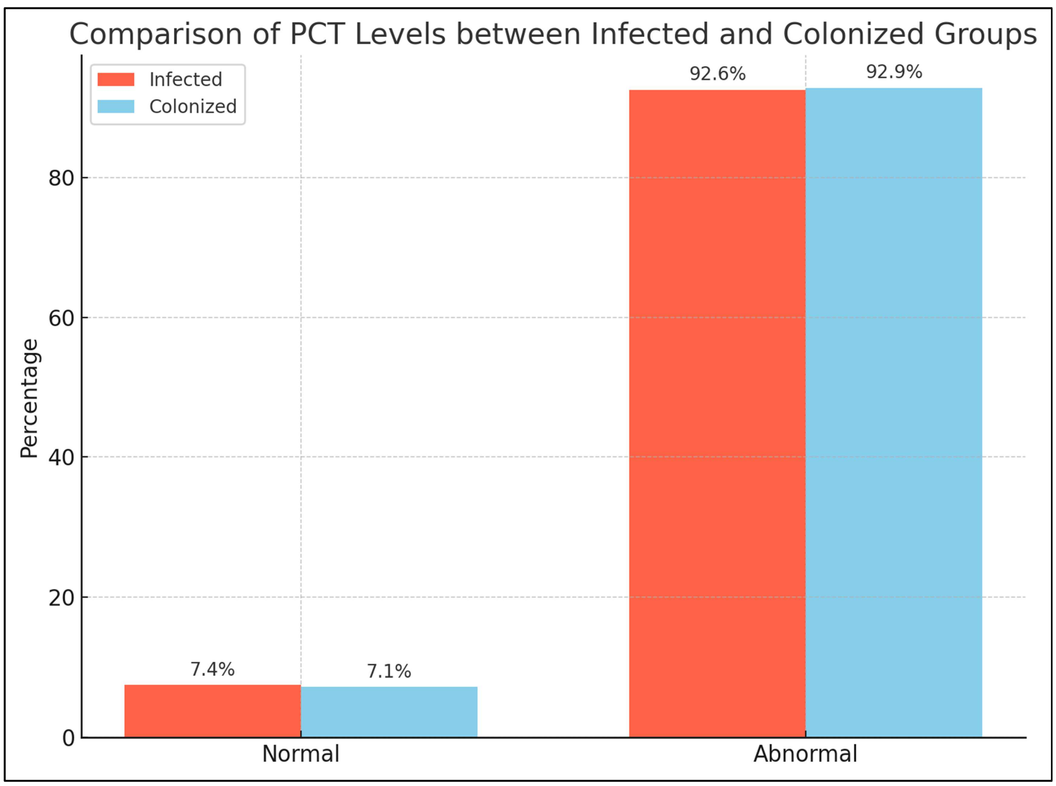

Figure 12: Comparison of PCT levels in infected and colonized groups

Renal function test

Renal function test (RFT) reported normal levels in 51.2% (n=21) of the study participants and abnormal levels in 48.8% (n=20) of the study participants.

Figure 13: RFT among infected and colonized groups

RFT	INFECTED	COLONIZED	TOTAL	P VALUE
Normal	13 (48.1%)	8 (57.1%)	21 (51.2%)	0.415
Abnormal	14 (51.9%)	6 (42.9%)	20 (48.8%)	

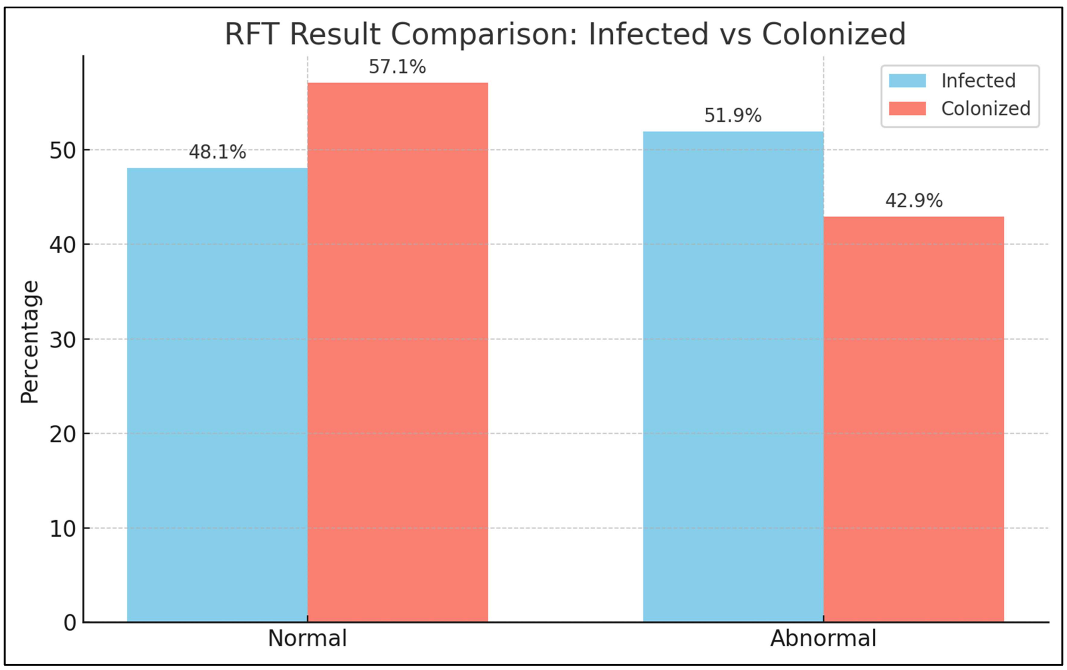

Figure 14: Comparison of RFT among infected and colonized groups

Liver function test

Liver function test (LFT) was reported normal levels in 53.7% (n=22) and abnormal levels in 46.3% (n=19) of the study participants.

Figure 15: LFT among infected and colonized groups

LFT	INFECTED	COLONIZED	TOTAL	P VALUE
Normal	14 (51.9%)	8 (57.1%)	22 (53.7%)	0.504
Abnormal	13 (48.1%)	6 (42.9%)	19 (46.3%)	

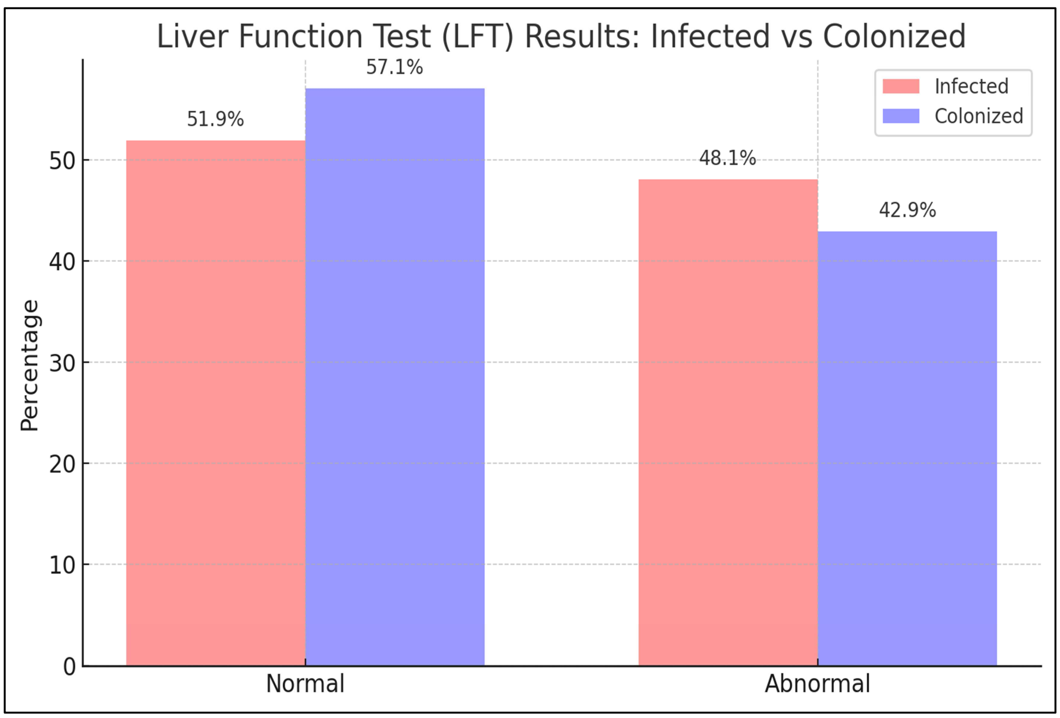

Figure 16: Comparison of LFT among infected and colonized groups

Antibiotics use

Among the study participants, amikacin was used in 19.5% (n=8), colistin was used in 36.6% (n=15), polymyxin-B was used in 17.1% (n=7), Cefoperazone-Sulbactam was used in 39% (n=16), Piperacillin-Tazobactam was used in 31.7% (n=13), meropenem was used in 24.4% (n=10), cefpirome was used in 2.4% (n=1), minocycline was used in 14.6% (n=6), clindamycin was used in 4.9% (n=2) and tigercycline was used in 36.6% (n=15) of the study participants.

Table 15: Antibiotics use among infected and colonized groups

ANTIBIOTICS	INFECTED	COLONIZED	TOTAL	P VALUE
Amikacin	5 (18.5%)	3 (21.4%)	8 (19.5%)	0.565
Colistin	7 (25.9%)	8 (57.1%)	15 (36.6%)	0.053
Polymyxin-B	5 (18.5%)	2 (14.3%)	7 (17.1%)	0.551
Cefoperazone-Sulbactam	9 (33.3%)	7 (50%)	16 (39%)	0.241
Piperacillin-Tazobactam	10 (37%)	3 (21.4%)	13 (31.7%)	0.257
Meropenem	8 (29.6%)	2 (14.3%)	10 (24.4%)	0.246
Cefpirome	1 (3.7%)	0 (0%)	1 (2.4%)	0.659
Minocycline	4 (14.8%)	2 (14.3%)	6 (14.6%)	0.672
Clindamycin	1 (3.7%)	1 (7.1%)	2 (4.9%)	0.572
Tigercycline	11 (40.7%)	4 (28.6%)	15 (36.6%)	0.339

Use of antibiotics in the infected and colonized study participants was given in the figure below.

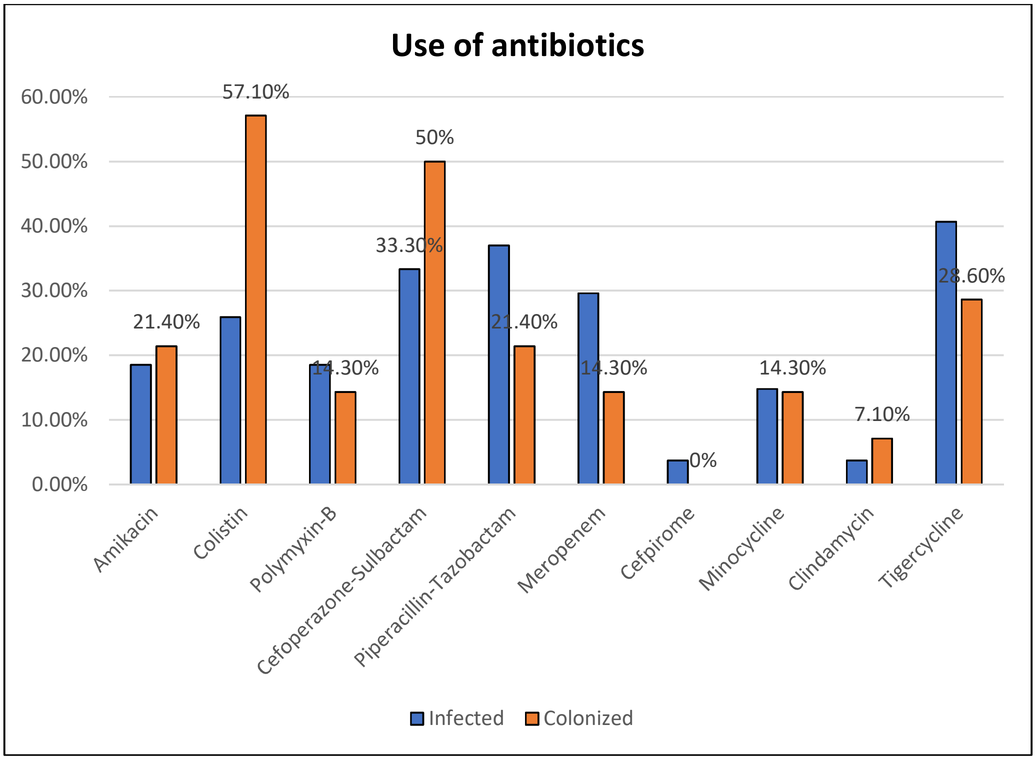

Figure 17: Antibiotics used in infected and colonized groups

Clinical outcomes

Among the study participants, the mortality rate was 31.7% (n=13). Among the study participants, 48.8% (n=20) were discharged and LAMA was 19.5% (n=8).

Table 16: Clinical outcomes

CLINICAL OUTCOMES	NUMBER	PERCENTAGE
Expired	13	31.7%
Discharged	20	48.8%
LAMA	8	19.5%

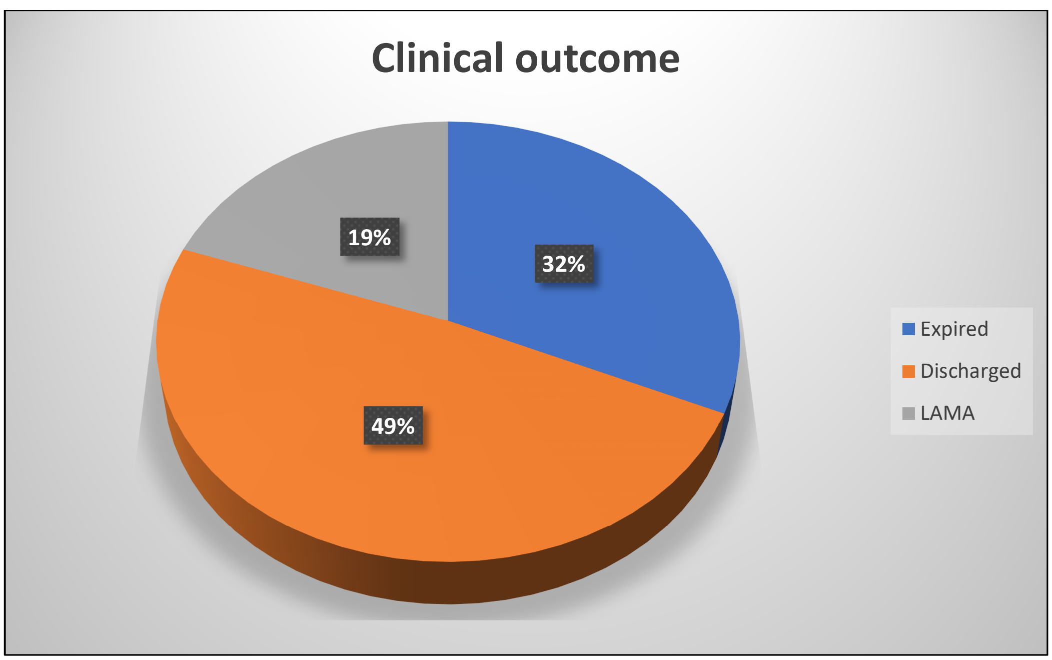

Figure 18: Clinical outcomes

Distribution of clinical outcome in infected and colonized study participants were given in the table below.

Mortality rate among the infected and colonized groups were 25.9% and 42.9% respectively. It was statistically significant (p=0.049).

Table 17: Clinical outcome among infected and colonized groups

OUTCOME	INFECTED	COLONIZED	P VALUE
Death	7 (25.9%)	6 (42.9%)	0.049
Discharged	12 (44.4%)	8 (57.1%)	
LAMA	8 (29.6%)	0 (0%)	

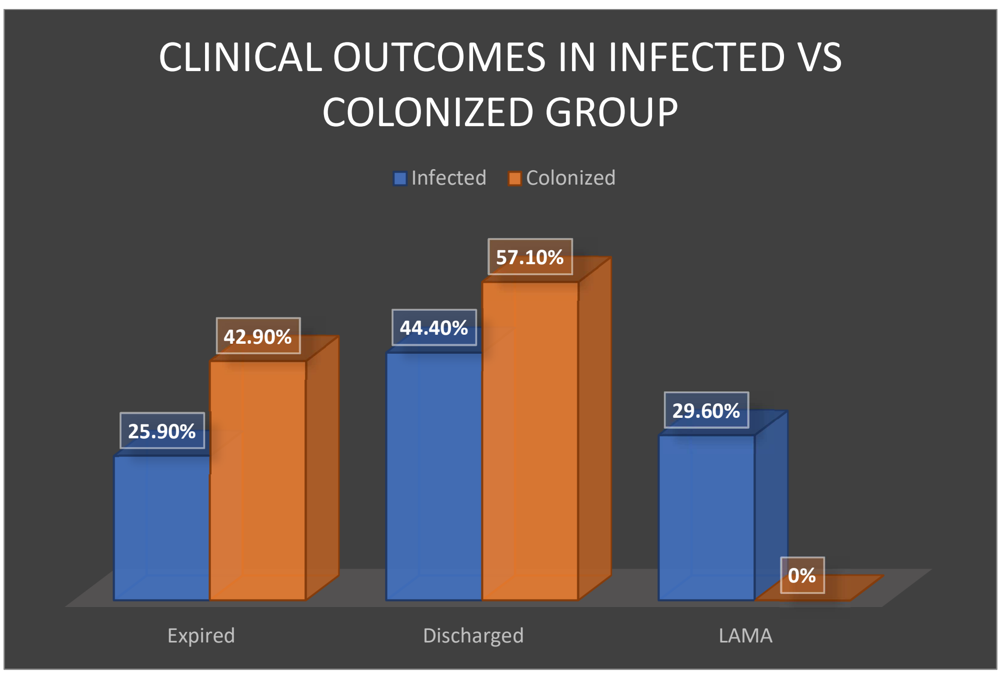

Figure 19: Clinical outcomes among infected and colonized groups

DISCUSSION

A baumannii is a common cause of nosocomial infection. The present study aimed to compare the clinical profile and outcomes of A baumannii in infected and colonized patients in ICU. A total of 41 study participants were included in the study. In total 41, 65.9% (n=27) were infected and 34.1% (n=14) were colonized with A baumannii in the present study.

In the study conducted by Martin-Aspas et al[41] the prevalence of A baumannii infected and colonized were 40% and 60% respectively. In the study conducted by Molina J et al[39], 46.2% showed colonized and 53.8% showed infected A baumannii cases. In the study conducted by Sileem AE et al, 45% were infected and 55% were colonized by A baumannii[37].

Age

In the present study, the mean age of the study participants was 55.05 ± 18.77 years. In the study conducted by Martin-Aspas et al, the mean age of the study participants was 61 ± 17 years. In the study conducted by Guddeti PK et al.[25], the mean age of the study participants was 54.36 ± 16.80 years. Majority of the study participants in the Sileem AE et al study were below 60 years of age (68.2%).

Gender

Among the study participants in the present study, 82.9% (n=34) were males and 17.1% (n=7) were females. Similarly, majority of the study participants were males in studies conducted by Martin-Aspas et al.[41], Guddeti PK et al[25], and Sileem AE et al.[37].

Culture report

In the present study, in the blood culture among the study participants, 46.4% (n=19) reported A baumannii. Other organisms were reported as 7.2% (n=3) and 46.4% (n=19) showed no growth.

In the urine culture, A baumannii was reported in 12.3% (n=5). Other organisms were reported as 14.5% (n=6) and no growth was reported in 73.2% (n=30) study participants.

In sputum culture, A baumannii was reported in 43.9% (n=18). Other organisms were reported as 12.1% (n=5) and no growth was reported in 46.4% (n=19).

In the study conducted by Martin-Aspas et al[41], the isolate sites for A baumannii in infected and colonized patients in sputum were 14% and 8% respectively, in blood were 6% and 0% respectively, and in urine were 0% and 14% respectively.

Diagnosis

Most common diagnosis among the study participants was respiratory diseases contributing to 39% (n=16), followed by sepsis contributing to 36.6% (n=15). Other diseases include 14.6% (n=6) of central nervous system, 12.2% (n=5 of multiorgan failure, 7.3% (n=3) of liver disease, 4.9% (n=2) of gastrointestinal and pancreatitis each, and 2.4% (n=1) of cardiac, diabetes, organophosphorus poisoning and renal disease each.

In the study conducted by Martin-Aspas et al[41], the prevalence of pneumonia, tracheobronchitis, primary bacteremia, and secondary bacteremia were 78%, 19%, 3% and 16% respectively.

In the study conducted by Guddeti PK et al[25], the common diagnosis was aspiration pneumonia or pneumonia in 35% of the patients, followed by, 30.7% in cerebrovascular events or hemorrhagic shock, 24% in respiratory failure, 18% in accelerated hypertension, 8.4% in septicemia,, 7.7% in acute kidney injury and 5.5% in trauma or burns.

In sileem AE et al[37] study, the most common underlying disease were of pulmonary origin (65%).

Antibiotics use

Among the study participants, amikacin was used in 19.5% (n=8), colistin was used in 36.6% (n=15), polymyxin-B was used in 17.1% (n=7), Cefoperazone-Sulbactam was used in 39% (n=16), Piperacillin-Tazobactam was used in 31.7% (n=13), meropenem was used in 24.4% (n=10), cefpirome was used in 2.4% (n=1), minocycline was used in 14.6% (n=6), clindamycin was used in 4.9% (n=2) and tigercycline was used in 36.6% (n=15) of the study participants. Colistin, tigercylin, and piperacillin/ tazobactam were 85%, 97.5% and 20% sensitive cultures in the study conducted by Sileem AE et al[37].

Clinical outcomes

Among the study participants, the mortality rate was 31.7% (n=13). Among the study participants, 48.8% (n=20) were discharged and LAMA was 19.5% (n=8).

In the study conducted by Martin-Aspas et al[41], the mortality rate at 30 days was 24%. In Molina J et al study, the mortality rate was 18.7%.

In our present study, the mortality rate in infected and colonized group were 25.9% and 42.9% respectively. In the study conducted by Sileem AE et al.[37], the mortality rate in infected and colonized groups were 50% and 13.6% respectively.

STRENGTHS OF THE STUDY

1. Focused Clinical Relevance: The study addresses Acinetobacter baumannii, a significant nosocomial pathogen associated with high mortality, particularly in critical care settings, making the findings clinically relevant.
2. Comprehensive Microbiological Sampling: Multiple sampling sites (blood, urine, ET, sputum) were used to assess the distribution and prevalence of A. baumannii, providing a more complete picture of infection patterns.
3. Differentiation Between Colonization and Infection: The study distinguishes between colonization and true infection with A. baumannii, which is clinically important for appropriate management decisions.
4. Multiple Parameter Assessment: The study incorporates various laboratory parameters (RFT, LFT, HB, TLC, ESR, serum PCT) to correlate with infection status, providing insight into potential biomarkers.
5. Outcome Analysis: The study includes clinical outcomes (discharge, LAMA, death), allowing for assessment of the impact of A. baumannii infection on patient prognosis.
6. Antibiotic Usage Documentation: Detailed documentation of antibiotics prescribed provides valuable information on current treatment practices for A. baumannii infections.
7. Statistical Analysis: Appropriate statistical tests were used to assess associations between A. baumannii status and clinical/laboratory parameters, with significant findings in relation to RFT and LFT abnormalities.

LIMITATIONS OF THE STUDY

1. Sample Size: With only 41 patients total and further subdivided into colonization vs. infection groups, the statistical power may be limited, particularly for subgroup analyses.
2. Potential Selection Bias: The method of patient selection is not clearly detailed in the results, raising concerns about potential selection bias that could affect generalizability.
3. Limited Demographic Analysis: While age and gender distribution are provided, there's limited analysis of how these factors might influence susceptibility to A. baumannii infection or outcomes.
4. Heterogeneous Patient Population: The study includes patients with diverse primary diagnosis (from acute pancreatitis to CVA to septic shock), making it difficult to isolate the specific impact of A. baumannii.
5. Incomplete Outcome Assessment: While mortality data is provided, there's no information on length of stay, duration of mechanical ventilation, or long-term outcomes among survivors.
6. Lack of Risk Factor Analysis: The study doesn't appear to analyze factors predisposing patients to A. baumannii acquisition, such as prior antibiotic use, invasive procedures, or ICU stay duration.
7. Unclear Temporal Relationships: It's not apparent whether laboratory abnormalities preceded or followed A. baumannii infection, making causality difficult to establish.
8. Missing Data on Treatment Effectiveness: While antibiotics given are listed, there's no analysis of treatment success rates or factors associated with treatment failure.

9. Limited Statistical Significance: Several of the comparisons (like TLC, ESR, serum PCT) between colonization and infection groups failed to reach statistical significance, possibly due to small sample size or high variability.

CONCLUSION

The percentage of infection and colonization of A baumannii in ICU patients were 65.9% (n=27) and 34.1% (n=14) respectively. The mean age of the study participants was 55.05 ± 18.77 years with majority being male (82.9%). In blood culture among the study participants, 46.4% (n=19), urine culture 12.3% (n=5), and sputum culture 43.9% (n=18) reported A baumannii. Most common diagnosis among the study participants was respiratory diseases contributing to 39% (n=16). Among the study participants, the mortality rate was 31.7% (n=13). Among the study participants, 48.8% (n=20) were discharged and LAMA was 19.5% (n=8). The mortality rate in infected and colonized groups were 25.9% and 42.9% respectively. The discharged rate in infected and colonized groups were 44.4% and 57.1% respectively. Mortality of patients from whom A. baumannii was isolated was influenced by the presence of infection by A. baumannii rather than colonization.

SUMMARY

A cross-sectional study conducted for a period of one year among the patients admitted to ICU wards of KLE's Dr Prabhakar Kore Hospital, Belagavi aimed to compare clinical profile and outcomes of Acinetobacter baumannii causing infection versus colonization.

A total of 41 study participants were included in the study with infection and colonization group of 65.9% and 34.1% respectively. The mean age of the study participants was 55.05 ± 18.77 years. Majority of the study participants were males. In blood culture among the study participants, 46.4% (n=19), urine culture 12.3% (n=5), and sputum culture 43.9% (n=18) reported A baumannii.

Most common diagnosis among the study participants was respiratory diseases contributing to 39% (n=16). Among the study participants, the mortality rate was 31.7% (n=13). Among the study participants, 48.8% (n=20) were discharged and LAMA was 19.5% (n=8). The mortality rate in infected and colonized groups were 25.9% and 42.9% respectively. The discharged rate in infected and colonized groups were 44.4% and 57.1% respectively. Mortality of patients from whom A. baumannii was isolated was influenced by the presence of infection by A. baumannii rather than colonization.

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ANNEXURE – I - INFORMED CONSENT FORM

" TO COMPARE CLINICAL PROFILE AND OUTCOMES OF ACENITOBACTER BAUMANII CAUSING INFECTION VS COLONIZATION IN ICU PATIENTS"

Name of Student/Principal Investigator

Name of Guide/Co Investigators:

Introduction:

-*Acenitobacter baumannii* is the most important in the clinical context since it is the most frequently isolated in nosocomial infections[2] and also the one associated with the highest mortality rate

-Acinetobacter is nonfermenting , gram negative, aerobic coccobacillus

- Acinetobacter baumanni is also known to cause both infection vs sedentary colonization.

-Owing to characteritics of patients from whom A.baumanni is isolated , it is often difficult to differentiate colonization and infection[3-4].

Explanation of procedure:

-Patients older than 18 years from whom A.Baumanni is isolated from clinical samples from two days after patients who got admitted in KLE ICU for the year 2023 and 2024

-Culture samples like sputum, urine ,blood will be included

-Cases will be divided into infection and colonization as defined in the introduction and will be followed from day 2 of admission till the patient stays in hospital

-All the participants fulfilling the inclusion criteria with willingness to participate will be included with their consent taken and protocol explained

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will get benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:,

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study**:**

"TO COMPARE CLINICAL PROFILE AND OUTCOMES OF ACENITOBACTER BAUMANII CAUSING INFECTION VS COLONIZATION IN ICU PATIENTS"

My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

ANNEXURE – II – DATA COLLECTION FORMAT

DATA COLLECTION FORMAT

- Demographic Details

Date:

1. Name of the patient :
2. IP Number:
3. Age:
4. Sex:
5. Address:
6. Occupation:
7. Phone Number:

Chief Complaints:	
Past History	
Personal History	
Treatment History	

- Vitals:

Temperature	
Pulse	
Blood Pressure	
Respiratory Rate	

- Physical Examination:

Index	Yes	No
Pallor		
Icterus		
Cyanosis		
Clubbing		
Lymphadenopathy		

- Systemic Examination:

CVS	
RS	
P/A	
CNS	

8. Please list all medication(s) you take, including dosage :

9. Any history of diabetes /hypertension/thyroid abnormalities
10. Any history of Ischemic Heart Disease:
11. Any history of Stroke:
12. Risk Factors:

Smoking Alcohol Hypertension
Thyroid Disease Stroke Cardiovascular Disease
High Cholesterol/Triglycerides Others: :

- Investigations:

Investigations	Values	Investigations	Values
Hemoglobin		White Total Count	
Platelets count		Liver fuction test	
MCV		Renal function tests	
MCH		Urine culture	
MCHC		Blood culture	
RDW		Sputum culture and sensitivity	
Serum pct			
ESR			

ANNEXURE III

MASTER CHART

AGE	SEX	IPNO	BLOOD CULTURE	URINE CULTURE	SPUTUM CULUTURE	ET CULTURE	HB	TLC	ESR	SERUM PCT	RFT	LFT	Clinical outcome	Antibiotics Given	Primary Diagnosis
40	MALE	10053980	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	7.2	14	42	0.92	WNL	TB:4.26,DB:3.89	Discharged	Meropenem,Clindamycin,Sulbactam	Septic shock secondary to aspiration pneumonia
70	MALE	10058445	NOG	CANDIDA TROPICALIS	NOG	ACINETOBACTER BAUMANNI	8.9	16.4	32	0.46	WNL	WNL	Expired	Colistin, Cefoperazone-Sulbactam	Septic Shock
68	MALE	10053931	COAGULASE -VE STAPTH SPECIES	ACINETOBACTER BAUMANNI	NOG	NOG	10.7	16.6	22	1.26	CREATININE:4	WNL	Expired	Tigecycline, Cefoperazone-Sulbactam, Minocycline	Organophosphorus compound poisining
59	FEMALE	10048288	NOG	ENTEROBACTER CLOACA	NOG	ACINETOBACTER BAUMANNI	10	15	48	3.2	CEATININE:2.1	WNL	Discharged	Colistin, Cefoperazone-Sulbactam	Septic shock secondary to bacterial pneumonia
80	MALE	10054966	NOG	ACINETOBACTER BAUMANNI	NOG	NOG	9.8	13	48	3.2	CREATININE:1.9	WNL	Discharged	Tigecycline, Piperacillin-Tazobactam, Minocycline	Viral pneumonia
61	MALE	10057950	ACINETOBACTER BAUMANNI	ENTEROCOCCUS FEACIALIS	NOG	PSEUDOMONAS AUEROGINOSA	8.8	12	24	0.27	CREATNINE:1.4	ALBUMIN:2.4	LAMA	Colistin, Cefoperazone-Sulbactam	Acute Pancreatitis
60	MALE	10054545	NOG	NOG	KLIEBSIELLA PNEUMONIA	ACINETOBACTER BAUMANNI	9	33.4	28	3.52	CREATININE:3.0	ALBUMIN:1.5	Expired	Meropenem, Cefoperazone-Sulbactam, Minocycline	Sepsis with multiorgan dysfunction
54	MALE	10057864	NOG	ACINETOBACTER BAUMANNI	NOG	NOG	10.1	18	20	2.4	CREATININE:2.4	ALBUMIN:2.3	LAMA	Tigecycline, Piperacillin-Tazobactam, Polymyxin B	Acute respiratory distress syndrome
83	MALE	10058212	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	9.2	13.2	48	1.4	CREATININE:2	ALBUMIN:2.3	Discharged	Colistin, Amikacin, Polymyxin B	Sepsis with multiorgan dysfunction
37	MALE	10005315	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	8.5	12.9	28	2.28	WNL	TB:3.04,DB:2.07	Expired	Tigecycline, Piperacillin-Tazobactam, Polymyxin B	Pulmonary tuberculosis
45	MALE	10008226	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	11.9	37.7	3	4.14	CREATININE:3.40	TB:1.90,DB:3.24,IB:2.66	Discharged	Tigecycline, Cefoperazone-Sulbactam	Decompensated chronic liver disease with hepatic encephalopathy and UGI bleed
48	MALE	10038588	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	14.1	2.8	80	2.4	CREATININE:4.78	TB:5.44,DB:5.27,IB:0.17	Discharged	Colistin, Piperacillin-Tazobactam	Septic shock with multiorgan dysfunction
82	MALE	10038670	BURKHOLDERIA CEPACIA	ENTEROCOCCUS FEACIALIS	NOG	ACINETOBACTER BAUMANNI	10.5	5.7	18	5.52	WNL	WNL	Discharged	Colistin, Cefoperazone-Sulbactam	Septic shock secondary to aspiration pneumonia
35	MALE	10038800	NOG	NOG	ACINETOBACTER BAUMANNI	NOG	14	12	40	3.2	CREATININE:1.5	WNL	Discharged	Colistin, Piperacillin-Tazobactam	Neuroendocrine tumor of liver
29	MALE	10040640	ACINETOBACTER BAUMANNI	WNL	NOG	NOG	13.7	10.7	52	1.32	WNL	WNL	LAMA	Meropenem, Cefoperazone-Sulbactam, Polymyxin B	Right sides hemiparesis with left thalamic bleed
68	FEMALE	10041823	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	12	14	48	3.2	WNL	WNL	Expired	Colistin, Cefoperazone-Sulbactam	Septic sock
73	MALE	10042608	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	8.5	8.3	40	5.81	CREATININE:2.15	WNL	Discharged	Colistin, Piperacillin-Tazobactam	Septic shock with bilateral pneumonia
58	MALE	10059459	NOG	NOG	ACINETOBACTER BAUMANNI	NOG	11	13	80	2.4	CREATININE:1.9	WNL	Discharged	Piperacillin-Tazobactam	Acute CVA -Right mca-pca infarction
32	MALE	10013077	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	8.7	10.8	28	4.67	WNL	TB:1.86,DB:1.55,IB:0.31	Expired	Tigecycline, Piperacillin-Tazobactam	Acute on chronic pancreatitis
33	FEMALE	10020068	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	12	15	20	2.3	WNL	ALBUMIN:3.2	Expired	Meropenem, Piperacillin-Tazobactam	Multi-organ Dysfunction
60	MALE	10020813	NOG	NOG	ACINETOBACTER BAUMANNI	NOG	8.9	15.4	86	0.47	WNL	WNL	Discharged	Meropenem, Amikacin, Polymyxin B	Severe Pneumonia
77	MALE	10023024	NOG	STREPTOCOCCUS AGALAGICTAE	NOG	ACINETOBACTER BAUMANNI	8.8	8.2	18	0.74	WNL	WNL	Discharged	Tigecycline, Amikacin	Acute on chronic subdural hematoma
60	MALE	10024419	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	15.4	11.8	38	2.4	WNL	WNL	LAMA	Meropenem ,cefpirome	Hypertensive enchephalopathy
29	MALE	10026405	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	12.3	17.2	38	1.2	WNL	WNL	Expired	Colistin, Cefoperazone-Sulbactam	Severe Pneumonia
59	FEMALE	10029608	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	8.3	15	28	0.78	WNL	TB:10 ,DB:9.72,IB:8.78	Expired	Tigecycline, Cefoperazone-Sulbactam, Minocycline	Acute Respiratory Failure
63	MALE	10030961	NOG	ACINETOBACTER BAUMANNI	NOG	NOG	8.9	14.8	10	3.2	CREATININE:11	WNL	Discharged	Meropenem, Tigecycline	Urosepsis with acute kidney injury
24	MALE	10030691	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	7.5	2.1	41	2.2	WNL	SGOT:1031 SGPT:827	Expired	Tigecycline, Amikacin, Minocycline	Acute Respiratory distress syndrome
48	MALE	10031537	KLEIBSIELLA PNEUMONIA	ACINETOBACTER BAUMANNI	STAPHYLOCOCCUS HEMOLYTICUS	NOG	11	11.4	10	4.96	WNL	TB:1.46,ALBUMIN:2.4	Expired	Colistin, Cefoperazone-Sulbactam	Septic shock with multiorgan dysfunction
68	MALE	10031651	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	10.4	16.7	24	2.97	WNL	WNL	LAMA	Meropenem, clindamycin	Recurrent stroke-right MCA-PCA infarction
74	MALE	10032151	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	8.1	16.8	20	2.39	POTTASIUM:6.18	WNL	Discharged	Piperacillin-Tazobactam	Acute pulmonary edema
60	MALE	10033443	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	11.2	13.4	42	1.8	CREATININE:2.1	WNL	Discharged	Meropenem, Piperacillin-Tazobactam	Septic Shock
94	MALE	10033572	NOG	NOG	ACINETOBACTER BAUMANNI	NOG	9.7	11.1	48	4.5	WNL	ALBUMIN:2.9	LAMA	Colistin, Piperacillin-Tazobactam, Polymyxin B	Septic shock with Bilateral pneumonia
67	MALE	10035166	ACINETOBACTER BAUMANNI	NOG	ACINETOBACTER BAUMANNI	PSEUDOMONAS AUEROGINOSA	8.4	17.5	22	1.59	WNL	ALBUMIN:2.9	Discharged	Colistin, Amikacin, Polymyxin B	Septic shock
70	MALE	10063721	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	8.6	21.4	16	83.3	CREATININE:6.14	WNL	Discharged	Tigecycline, Cefoperazone-Sulbactam, Minocycline	Acute exaceberation of chronic obstructive pulmonary disease
34	MALE	10067021	ACINETOBACTER BAUMANNI	NOG	NOG	KLEIBSIELLA PNEUMONIA	15.6	27	28	3.4	WNL	WNL	Discharged	Tigecycline, Amikacin	Right subarachanoid hemorrhage
81	MALE	10067631	ACINETOBACTER BAUMANNI	KLEIBSIELLA PNEUMONIA	NOG	NOG	9.4	0.9	16	1.52	CREATININE:4.08	ALBUMIN:3.3	Expired	Tigecycline, Cefoperazone-Sulbactam	Septic shock secondary to aspiration Pneumonia
42	FEMALE	10068129	NOG	NOG	NOG	ACINETOBACTER BAUMANNI	11	12.6	48	3.2	CREATININE:5.87	TB:3.89,DB:1.26,IB:2.63	Expired	Colistin, Amikacin	Small bowel obstruction s/p exploratory laparotomy
38	FEMALE	10033848	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	8.3	39	67	3.3	WNL	ALBUMIN:2.2,SGOT:148,SGPT:102	Discharged	Tigecycline, Cefoperazone-Sulbactam	Diabetic ketoacidosis
42	MALE	10039959	ACINETOBACTER BAUMANNI	NOG	NOG	E.COLI	15.7	18	10	2.4	CREATININE:4.2	WNL	LAMA	Tigecycline, Amikacin	Septic shock
30	FEMALE	10070409	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	11.3	28.7	40	1.54	POTTASIUM:2.98	ALBUMIN:2.1	LAMA	Meropenem, Piperacillin-Tazobactam	Acute Respiratory Failure
22	MALE	10083894	ACINETOBACTER BAUMANNI	NOG	NOG	NOG	8.8	14.4	WNL	4.2	WNL	WNL	Discharged	Colistin,Sulbactam	Subarachanoid hemorrhage