EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Coden USA: EJBPBO
Volume: 13
Issue: 7
Year: 2026

ISOLATION AND ANTIBIOTIC SUSCEPTIBILITY PATTERN OF ACINETOBACTER IN SURGICAL INTENSIVE CARE UNIT AT A TERTIARY CARE HOSPITAL

Miss. Ifshana Shafi[1]*, Miss. Inara Bashir[2], Mr. Peerzada Mohammad Idrees[3], Mr. Shaikh Anaitullah[4], Mr. Shuiab Altaf[5]

*[1,4]Laboratory Technician at Sher-i-Kashmir Institute of Medical Sciences Soura.
[2]Laboratory Technician at Biocare Clinical Laboratory Ganderbal.
[3]Operation Theatre Technician at The SKJ Hospital Anantnag.
[5]Data Entry Operator at Sher-i-Kashmir Institute of Medical Sciences Soura.

***Corresponding Author: Miss. Ifshana Shafi**
Laboratory Technician at Sher-i-Kashmir Institute of Medical Sciences Soura.

DOI: https://doi.org/10.5281/zenodo.21336665

How to cite this Article: Miss. Ifshana Shafi[1]*, Miss. Inara Bashir[2], Mr. Peerzada Mohammad Idrees[3], Mr. Shaikh Anaitullah[4], Mr. Shuiab Altaf[5]. (2026). Isolation And Antibiotic Susceptibility Pattern Of Acinetobacter In Surgical Intensive Care Unit At A Tertiary Care Hospital. European Journal of Biomedical and Pharmaceutical Sciences, 13(7), 509–524.

Article Received on 15/06/2026 **Article Revised on 05/07/2026** **Article Published on 10/07/2026**

ABSTRACT

Introduction: *Acinetobacter* has become a significant concern in hospitals, particularly among critically ill patients in intensive care units. This study aimed to assess the presence of multidrug-resistant (MDR) *Acinetobacter species* in patients admitted to the surgical intensive care unit of our hospital. The finding will help guide timely interventions to prevent the spread of these pathogens, protect patients from ineffective treatments, and reinforce infection control practices. **Material and Methods:** This Prospective study was conducted in the Department of Microbiology and Surgical Intensive Care Unit at Sher I Kashmir Institute of Medical Sciences, Soura, J&K, from February to July 2024. Samples were cultured on 5% sheep blood agar and MacConkey agar, followed by overnight aerobic incubation at 37◦C. After the isolation of typical colonies, identification was carried out using standard microbiological methods. Antibiotic susceptibility testing was performed in accordance with CLSI guidelines. **Results:** a total of 80 unique samples were collected from the surgical ICU. Of which, 50 samples exhibited microbial growth, while 30 were sterile. Of the 50 positive samples, 30 isolates of *Acinetobacter* species were identified. The highest proportion of isolates (23.3%) was found in the 60-70 age group, with the majority (96.6%) obtained from tracheal aspirates. Additionally, 25 patients had extended hospital stays of 7 days or more before *Acinetibacter* was isolated, whereas only 5 patients had been hospitalized for 7 days or less prior to the isolation. Among the patients, 28 were on ventilator support. Resistance was observed in 29 isolates (96.6%) for both Amikacin and Tobramycin. High resistance rates were also noted for tetracycline, Tigecycline, Gentamycin, Levofloxacin, and Cefotaxime, with 93.3% of isolates being resistant to each. Additionally, 27 isolates (90%) were resistant to Cefepime, and 86.6% showed resistance to both Meropenem and Imipenem. However all *Acinetobacter* isolates were sensitive to Polymyxin B, while 19 isolates (63.3%) were sensitive to Peperacillin-Tazobactam. **Conclusion**: Risk factors such as advanced age, ventilator support, and prolonged hospital stay in ICU patients should heighten suspicion of MDR *Acinetobacter* infection. Antibiotic therapy should be initiated only after conducting susceptibility testing.

KEYWORDS: MDR *Acinetobacter* in ICU, Nosocomial Infections, Antibiotic Resistance.

INTRODUCTION

Table 1: Taxonomic classification of *Acinetobacter baumanni.*

Domain	**Bacteria**
Phylum	Pseudomonadota
Class	Gammaproteobacteria
Order	Pseudomonadales
Family	Moraxellaceae
Genus	Acinetobacter
Species	A. baumannii

Acinetobacter, a gram-negative coccobacillus, has emerged as an important nosocomial pathogen. It is non-motile, encapsulated that belongs to *Neisseriaceae* family. Acinetobacter is oxidase-negative, indole-negative, and catalase-positive. It is commonly found in the environment and healthcare settings, isolated from medical personnel and equipment.[1,2,3,4] This strictly aerobic bacterium does not require specialized nutrients and can grow on standard laboratory media such as tryptic soy agar and blood agar.[5] Acinetobacter species form dome-shaped, non-pigmented colonies measuring 1 to 2 mm, with smooth or pitted surfaces. They are non-lactose fermenting and can persist on various surfaces, including in aqueous environments.[6,7] Acinetobacter comprises over 20 species, with *Acinetobacterbaumannii,*[8] previously known as *Acinetobactercalcoaceticus* anitratus, being the most prevalent. It accounts for the majority of ICU infections, representing 80% of clinical isolates.[9,1,2,10,4,11,12]

Identifying *Acinetobacter* species is difficult because standardized methods are not readily available. Traditional methods relied on characteristics like growth temperature, colony shape, and glucose fermentation. These methods could identify the *A. calcoaceticus* - *A. baumannii* complex but not closely related species.

Acinetobacter isolates are divided into three primary categories:

- ***Acinetobacter calcoaceticus-baumannii* complex** (glucose-oxidizing and nonhemolytic) (*A. baumannii* can be identified by OXA-51 typing),
- ***Acinetobacter lwoffii*** (glucose-negative and nonhemolytic), and
- ***Acinetobacter haemolyticus*** (hemolytic).

Genus-level identification of ***Acinetobacter*** can also involve assessing acid production from glucose metabolism and chromosomal DNA transformation assays. Molecular techniques include repetitive extragenic palindromic sequence-based PCR, ribotyping, pulsed-field gel electrophoresis (PFGE), random amplified polymorphic DNA (RAPD), amplified fragment length polymorphism (AFLP), and analysis of tRNA and 16S-23S rRNA intergenic spacers. Among these, PFGE, AFLP, and amplified 16S ribosomal DNA restriction analysis (ARDRA) are frequently used due to their high accuracy. Additionally, newer methods like multilocus sequence typing (MLST) examine genetic relationships by amplifying housekeeping genes.

Acinetobacter species are widely found in soil and water, capable of surviving on both moist and dry surfaces, and are resistant to many disinfectants, which contributes to their persistence in healthcare settings. They can grow across a range of temperatures, allowing them to adapt to diverse environments.

Acinetobacter baumannii is a significant cause of hospital-acquired infections, particularly ventilator-associated pneumonia (VAP). It is also associated with skin and wound infections, bacteremia, and meningitis. A. lwoffii, on the other hand, is more commonly linked to meningitis.

Acinetobacter baumannii frequently causes bacteremia, urinary tract infections (UTIs), secondary meningitis, infective endocarditis, and infections of wounds and burns. It is a leading cause of VAP in ICU patients, with prolonged intubation and aspiration being key risk factors. The pathogen can enter the bloodstream, causing bacteremia with high mortality rates. UTIs are often associated with prolonged catheterization and prior antibiotic use.

Infections of the skin and soft tissues are common in trauma and burn patients, posing treatment challenges. While rare, A. baumannii can cause meningitis, primarily after surgical procedures, as well as neonatal sepsis, which can be fatal. It is also implicated in cases of infective endocarditis, keratitis, peritonitis, and occasionally in severe neonatal infections.

Acinetobacter baumannii is notable for its resistance to various antibiotics, including broad-spectrum cephalosporins, β-lactam antibiotics, aminoglycosides, quinolones, and increasingly, carbapenems. It can survive on human skin and dry surfaces for weeks, and its resistance to many disinfectants contributes to its spread in hospital settings. The antibiotic-resistance genes, typically found on plasmids, can be transferred to other bacteria through horizontal gene transfer. In healthy individuals, Acinetobacter colonies on the skin have been linked to a lower incidence of allergies, suggesting a potential protective role against allergies.

Acinetobacter can be isolated from various human sources, including skin, pharynx, sputum, urine, vaginal secretions, and stool.[13] Beyond human skin, it has been found in soil, water, fish, meat, vegetables, hospital air, tap water, faucets, sink basins, bed mattresses, bedside urinals, and respiratory therapy equipment. Colonization and infection with Acinetobacter are more common during warmer and more humid months.[14,15] A. baumannii has been responsible for an increasing number of outbreaks worldwide, especially in ICUs, where it can persist for extended periods.[16,17]

Epidemiological studies highlight the hospital environment and colonized patients as key reservoirs for the bacteria. ICU infections tend to be more severe, and the pathogens involved may differ from those in other hospital populations. Although ICUs account for a small portion of total hospital beds, infection rates are disproportionately higher due to more frequent and prolonged antibiotic use, given the critical condition of ICU patients, who are more susceptible to nosocomial infections. A. baumannii causes a wide range of hospital-acquired infections in compromised individuals, especially ventilator-associated pneumonia.[18] Its rapid

development of antimicrobial resistance.[19] and ability to persist on various surfaces has made it a significant cause of hospital-acquired infections.[20,21]

The prevalence of multidrug-resistant Acinetobacter baumannii strains has been rising, leading to increasingly fatal hospital infections.[22] This surge in antibiotic resistance is largely due to the overuse of antimicrobial agents.[23] A. baumannii exhibits a broad array of resistance mechanisms, such as acquiring plasmids, transposons, and integrons, as well as producing carbapenemases.[24] Resistance to carbapenems is widespread, significantly limiting treatment options. Numerous carbapenem-hydrolyzing beta-lactamases have been detected in A. baumannii, with class B beta-lactamases exhibiting significant hydrolytic activity against carbapenems.[25] As a result, carbapenems, once the preferred treatment, are no longer commonly used for Acinetobacter infections.[24]

Acinetobacter spp. exhibit remarkable resistance to several key antibiotics. While the exact mechanisms behind multidrug resistance are not fully understood, recent research indicates that MDR Acinetobacter strains can produce a variety of enzymes mediated by both chromosomal and plasmid genes. These bacteria can generate aminoglycoside-modifying enzymes, making them resistant to aminoglycosides.[26] Beta-lactamases confer resistance to penicillins, cephalosporins, and carbapenems.[27,28] They can also reduce antibiotic uptake by modifying outer membrane pores or creating active efflux systems.[29] Additionally, Acinetobacter can alter target proteins to prevent antibiotics from binding effectively. For example, changes in the topoisomerase IV gene contribute to quinolone resistance,[30] while altered penicillin-binding proteins lead to penicillin resistance.[28]

Infections caused by multidrug-resistant Acinetobacter have a high mortality rate, especially in critically ill patients. These infections are often linked to prolonged mechanical ventilation, extended ICU stays, and longer hospitalizations. Over recent decades, Acinetobacter has emerged as a major nosocomial pathogen, responsible for significant morbidity and mortality.[31,32] Its widespread presence in ICU environments, coupled with insufficient infection control practices, has contributed to the growing prevalence of Acinetobacter infections. Despite the global concern, relatively few studies have been published on the topic. Understanding and addressing Acinetobacter infections in ICUs is crucial. This study aims to identify the presence of multidrug-resistant Acinetobacter species in patients admitted to the surgical ICU, enabling timely interventions to control their spread, prevent ineffective treatments, and ensure proper infection control measures.

REVIEW OF LITERATURE

Until 1970, *Acinetobacter* spp. were considered rare causes of nosocomial infections in the intensive care unit (ICU).[33] In recent years, however, the incidence of *Acinetobacter* infections has reached a point of concern and poses a threat to hospitalized populations around the world.[9,34] Outbreaks have been increasingly reported regularly.[1,35,36] Moreover, most of those outbreaks were caused by multidrug-resistant (MDR) strains of this organism.[9,37,33] The initial concern about multidrug-resistant *Acinetobacter* strains began in 1991 when the first hospital-wide outbreak occurred in New York.[34] Now multidrug-resistant *Acinetobacter* strains are observed worldwide.[33,35,38] In Thailand, multidrug-resistant *Acinetobacter* infections have been described repeatedly.[10,39] Their extraordinary ability to develop multiple resistance mechanisms against major antibiotic classes used in the intensive care unit (ICU) including cephalosporin, aminoglycosides, carbapenems, and quinolones leads to the development of multidrug-resistant Acinetobacter strains which then spread to cause increased mortality and morbidity.[4]

Poutanem SL *et al*. in the year 1997. Studied the medical records of 39 patients with *Acinetobacter bacteremia*. In 24 cases (62%) they found that bacteremia was considered to be clinically significant. Most of the infections were nosocomial, and the majority of these were acquired in an intensive care unit (ICU). In most of these cases (85%), Acinetobacter bacteremia was thought to have contributed to death. The following variables were associated with a greater risk of mortality: age >65 years (OR=16; p=0.01); development of septic shock (OR=22;p=0.004); and the presence of coagulopathy (OR=20;p=0.03).[40]

Rotimi OV *et al*. in the year 1998. Studied the prevalence and antibiotic susceptibility pattern of consecutive Gram-negative bacterial isolates in two intensive care units (ICUs) in Saudi Arabia (Jeddah) and Kuwait, 106 and 101 isolates, respectively. They found that the most common bacterial isolates in Jeddah versus Kuwait intensive care units (ICUs) were *Pseudomonas aeruginosa* (26%, 26%, *Escherichia coli* (23%, 3%), *Klebsiella pneumonia* (20%, 17%), inducible *Enterobacteraecae* group (17%, 14%), and *Acinetobacter spp.* (9%, 33%). Dominant features of the study in Jeddah were the *E.coli* and *klebsiella* species demonstrating multi-resistance to monobactams, cephems, and all the three aminoglycosides, and evidence of two classes of resistance to β-lactam antibiotics which were not seen among the Kuwait isolates. The Kuwait *Pseudomonas* species were more sensitive to Imipenem than the Jeddah *Pseudomonas spp*. (100% versus 68%).[41]

Siau H *et al*. in the year 1998, studied the epidemiological characteristics of 18 patients with *Acinetobacter bacteremia*. Patients (mean age, 55.5 years) developed bacteremia after an average of 14.1 days of hospitalization. Fifteen of 16 patients survived bacteremia caused by *Acinetobacter baumannii*. Cultures of blood from the remaining two patients yielded

Acinetobacter lwoffii. Most patients (78%) reside in the general ward, while four patients (22%) are under intensive care. Genotyping by arbitrarily primed polymerase chain reaction (PCR) analysis and the temporal sequence of isolation were more useful than phenotyping by antimicrobial susceptibility in the determination of the source of bacteremia, and the intravascular catheters were the leading infection source (39% of cases).[42]

Urban C *et al*. in the year 2003, studied control of infection due to multidrug-resistant *Acinetobacterbaumannii* (MDR-Acinetobacter) by identifying isolates as clonally related, leading to enhanced infection-control measures, including cohosting, surveillance, contact precautions, initial therapy with Ampicilin/sulbactam and local Polymyxin B, and more recently, therapy with synergistic antibiotic combinations. They also found that class restriction of cephalosporin has been associated with a reduction in cephalosporins-cephamycin-carbapenem resistance among nosocomial *Klebsiella* isolates. This has been supplemented by restriction of carbapenem use after an initial 24-hour period to reduce the selection of porin-deficient, carbapenem-resistant *Acinetobacter baumannii* and *Pseudomonas aeruginosa*. Their research suggested that eradication of MDR-Acinetobacter nosocomial colonization may prevent subsequent infection.[43]

Martro E *et al.* in the year 2003. The disinfection and antisepsis are of primary importance in controlling outbreaks of *Acinetobacter baumannii*, a nosocomial pathogen that frequently shows multiple antibiotic resistances. In their study, they assessed the susceptibility of nine *Acinetobacter baumannii* strains isolated during a sustained intensive care unit (ICU) outbreak, to several antiseptics and disinfectants based on European standards. While the tested strains showed diverse antibiotic resistance patterns, they were equally sensitive to the biocides over time, nor was there a correlation between resistance to antibiotics and a decreased susceptibility to antiseptics or disinfectants observed in this study.[44]

Rungruanghiranya S *et al*. in the year 2005, studies the rising incidence of *Acinetobacter* infection in the intensive care unit (ICU) causing a great concern to all clinicians and intensivists worldwide due to their extraordinary ability to develop resistance to multiple classes of antibiotics. *Acinetobacter* can infect virtually any body site, particularly the lower respiratory tract, the bloodstream, and the urinary tract. Their research shows that infection is mainly related to the inappropriate or previous use of antibiotics and the increasing use of invasive devices in the intensive care unit (ICU). Although they found that carbapenem is currently considered the drug of choice for these pathogens, the occurrence of carbapenem-resistant strains has led to fewer treatment options.[45]

Maragakis LL *et al*. in the year 2008. Multidrug-resistant *Acinetobacter baumannii* is recognized to be among the most difficult antimicrobial-resistant gram-negative bacilli to control and treat. Increasing antimicrobial resistance among *Acinetobacter* isolates has been documented, although definitions of multidrug resistance vary in the literature. *Acinetobacter baumannii* survives for prolonged periods under a wide range of environmental conditions. The organism causes outbreaks of infection and healthcare associated infections including bacteremia, pneumonia, meningitis, urinary tract infection, and wound infection. Antimicrobial resistance greatly limits the therapeutic options for patients who are infected with this organism, especially if isolates are resistant to the carbapenem class of antimicrobial agents. Because therapeutic options are limited for multidrug-resistant *Acinetobacter* infection, the development or discovery of new therapies, well-controlled clinical trials of existing antimicrobial regimens and combinations, and greater emphasis on the prevention of healthcare-associated transmission of multidrug-resistant *Acinetobacter* infection are essential.[46]

Tacconelli E *et al.* in the year 2008 studied the multidrug-resistant (MDR) *Acinetobacter baumannii calcoaceticus* complex (*Acinetobacter*) and found that it has emerged as an important cause of nosocomial infections. They included 514 patients in their study. One hundred and thirty-seven patients were infected and 120 were colonized. They found that a Charlson score >3 and previous methicillin-resistant Staphylococcus aureus isolation and beta-lactam use were independent risk factors for colonization and infection. Bedridden status and previous intensive care unit (ICU) admission were associated with colonization, while the presence of a central venous catheter and surgery were related to infection. The analysis of LRs showed an association between the presence of more than two risk factors and colonization or infection. The highest predicting value was observed for the presence of more than two risk factors and colonization in patients with no history of intensive care unit (ICU) admission.[47]

Enoch DA *et al*. in the year 2008 investigated and managed an outbreak of multidrug Carbapenem-resistant *Acinetobacter baumannii* in Cambridge, UK, and found that it has become endemic in many hospitals in the UK. All patients colonized or infected with MRAB-C were included. Enhanced infection control precautions were introduced in Phase 1 of the outbreak. The adult neurosciences critical care unit (NCCU) was partially closed in Phase 2 and strict patient segregation, barrier nursing, and screening thrice weekly was introduced. When control was achieved, NCCU was reopened (Phase 3) with post-discharge steam cleaning and monthly cleaning of extract and supply vents. There were 19 cases, 16 in the NCCU and three in the general intensive care unit (ICU). The mean age was 52 years, with six cases being female. All patients were mechanically

ventilated and ten had either an extra ventricular drain or intracranial pressure monitoring device in place. Four patients developed a bacteremia, with one further case of ventriculitis. Nine patients had no clinical evidence of infection and four were identified initially on screening. Ten patients were treated; there were eight deaths. Environmental samples showed heavy contamination throughout NCCU. MRAB-C affects critically ill patients and is associated with high mortality. This outbreak was controlled by early involvement of management, patient segregation, screening of patients and the environment, and increased hand hygiene environmental cleaning, and clinical vigilance. A multidisciplinary approach to outbreak control is mandatory.[48]

Saranraj P et al. in the year 2011 studied the nosocomial infections which occur worldwide and affect both developed and resource-poor countries and found that infections acquired in healthcare settings are among the major causes of death and increased morbidity among hospitalized patients. Antibiograms are the aggregate percentages of organisms susceptible to various antibiotics on a hospital formulary and are usually presented on an annual basis. The main purpose of this information was to guide empiric antimicrobial therapy before specific patient culture results are available. The prevalence of nosocomial infection-causing bacteria and their antimicrobial susceptibility pattern were reviewed in their study. Antibiograms are used to track the antibiotic resistance patterns of clinically important microorganisms detected by laboratories. They used hospital antibiograms to provide useful information for the selection of empiric therapy for a presumptive diagnosis as well as detect trends towards antimicrobial resistance. Hospital laboratories usually generate an antibiogram every six to twelve months and the data is then entered into an antibiogram database. Limitations of hospital antibiograms are that they do not sort out community-acquired infections from nosocomial infections and some laboratories may not thoroughly unduplicated their data, thus giving a picture of a larger number of resistant isolates than is the case. Their review assesses the following topics: Impact of nosocomial infection, Nosocomial infection sites, Microorganisms causing nosocomial infections, Methods of acquisition of nosocomial infections, Antimicrobial use and drug resistance in nosocomial infection, Antibiogram of nosocomial infection causing bacteria and Limitation of antibiogram.

Ozdemir *et al.* in the year 2011. They studied the clinical characteristics and outcomes of *Acinetobacter baumannii* infections in pediatric patients in a pediatric intensive care unit (PICU) in Turkey. The ages ranged from 1 month to 16 years with a mean age of 55.5 months, and the male-to-female ratio was 1:1.5. Ventilator-associated pneumonia (10 patients) was the leading diagnosis, followed by catheter-related bloodstream infection (4 patients), and bacteremia and ventilator-associated pneumonia associated with meningitis (1 patient) due to *Acinetobacter baumannii*. Mechanical ventilation (93.3%), central venous catheter (73.3%), urinary catheter (93.3%), and broad-spectrum antibiotic usage (80%) were the frequently seen risk factors. Neuromuscular (40%) and malignant (26.7%) disorders were the most common underlying diseases: Nosocomial *Acinetobacterbaumannii* is commonly multidrug-resistant, prolongs the length of stay in the PICU and increases the mortality rates in pediatric critical care.[50]
Nahar A *et al*. in the year 2012. They studied critically ill patients who acquire an infection during their stay in ICU and the frequency of their infections varies considerably in different populations and clinical settings. The purpose of their study was to know the antimicrobial sensitivity pattern of *Acinetobacter* isolates from tracheal aspirate, blood from the central venous catheter, peripheral blood, urine, and endotracheal tube of patients admitted to ICU at BSMMU (Bangladesh Sheikh Mujib Medical University) hospital, Dhaka, Bangladesh over a one year from January 2010 to December 2010. A total of 95 ICU samples were studied of which 32 (33.7%) *Acinetobacter* species were isolated from endotracheal tube (100%), tracheal aspirate (54.3%), and blood from central venous catheter (36.4%), peripheral blood (13.6%), and urine (12.5%). *Acinetobacter* isolates were 100% resistant to Amoxicillin, Ceftriaxone, Cefuroxime, and Gentamicin. A higher level of resistance was recorded for Amikacin (68.4%) and Imipenem (66.7%). Lower resistance only showed in Colistin (10.5%).[51]

Bhattacharyya S *et al.* in the year 2013. *Acinetobacter* species are gram-negative coccobacilli causing various nosocomial infections. They possess different types of beta-lactamases like SHV, TEM, and others which lead to treatment failure in case of infections due to this pathogen. Studying their species distribution in various infections and drug resistance patterns is hence important. Hence their study was aimed to study the different infections caused by *Acinetobacter* species, in a tertiary care hospital in Eastern India and its antibiotic susceptibility pattern.[52]

Kumar E *et al*. in the year 2014. The species belonging to the genus *Acinetobacter* are currently reported as opportunistic pathogens and show multi-drug resistance in hospitalized patients. Their study aimed to isolate the clinical *Acinetobacter* species from NFGNB (Non-Fermentative Gram-Negative Bacilli) other than *Pseudomonas* and analyze their antibiogram (Antibiotic susceptibility) patterns. Their study helps to know the persistence of antibiotic resistance and decide the optional treatment feasible for *Acinetobacter* infections.[53]

Gayathri P *et al.* in the year 2015. They studied the *Acinetobacter* species and their antimicrobial resistance pattern. The study was done for a time of one year from July 2013 to June 2014. A total number of 452 patients were included in the study. The culture reports of these

patients were analyzed which included ET secretions from patients with end tracheal infections, BAL from pneumonia, and Sputum of lower respiratory tract infections patients. The cultures that were positive for *Acinetobacter* species were identified and their resistance towards antimicrobials was tested. Among 82 strains of *Acinetobacter* species were obtains comprising of 48 (58.53%) from ET samples, 22 (26.83%) from BAL fluid, and 12(14.64%) from Sputum culture of these 12 strains were found to be resistant to Amikacin. *Acinetobacter specie*s was found to be sensitive to Imipenem and Amikacin. Imipenem is found to be very effective in controlling infections caused by *Acinetobacter*.[54]

Ellis *et al.* in the year 2015. *Acinetobacter baumannii* can cause serious healthcare-associated infections (HAIs) and the incidence is increasing, with many strains now resistant to multiple antibiotic classes. They studied the factors associated with healthcare-acquired infections (HAIs) caused by antimicrobial resistance as compared with antimicrobial-susceptible strains of *Acinetobacter baumannii* and investigated trends in the incidence of resistance over time. Electronic data from two U.S.A hospitals in a large urban healthcare system over the years 2006-2012 were used for the analysis.[55]

Lob, *et al*. in the year 2016, a recent global surveillance study spanning 2011 to 2014 highlights the challenge of multidrug-resistant *Acinetobacter baumannii* in intra-abdominal and urinary tract infections. With 2337 isolates from 453 hospitals across 48 countries, MDR rates ranged from 47% in North America to over 93% in Europe and the Middle East. Imipenem susceptibility was highest in North America (64%) and lowest in Europe and the Middle East (≤11%). Amikacin showed the highest overall activity, with 11–38% susceptibility against MDR isolates. Trend analysis revealed increasing MDR rates in the Middle East and decreasing susceptibility to various antimicrobial agents in Africa, Europe, and the Middle East over the study period. These findings underscore the need for targeted antimicrobial therapy and infection control measures.[56]

Castilho*et al*. in the year 2017. They study the concerning rise in antimicrobial resistance, particularly in intensive care unit (ICU) settings, with *Acinetobacterbaumannii* posing a significant threat. Among 1333 ICU patients, 56 developed *Acinetobacter baumannii* infections, with most isolates showing resistance to beta-lactam drugs, including cephalosporins and carbapenems. A striking 91.1% of isolates were multi-drug resistant (MDR), with the OXA-23 gene being prevalent (55.1%), often associated with MDR strains. Biofilm formation was common (76.8%). Polymyxins and tigecycline were among the few effective drugs. Molecular profiling identified 12 distinct clusters, underscoring the need for proactive measures against emerging resistance in healthcare settings.[57]

Tewari *et al*. in the year 2018. This study assessed antibiotic susceptibility among *Acinetobacter* species isolates from clinical specimens. Of 16,452 samples, 67 (0.4%) were positive for *Acinetobacter*, with urine being the primary source (38.8%). Most isolates (80.3%) were resistant to three or more antibiotic classes. Colistin showed 100% susceptibility, while *A. baumannii* had 80% susceptibility to tigecycline and 53.3% to carbapenem. Combination therapy, especially colistin and tigecycline, may be effective against multidrug-resistant *A. baumannii* until new options emerge.[58]

Namiganda *et al.* in the year 2019. Study conducted at BéniMessous University Hospital Center in Algiers, 19 strains of *Acinetobacter baumannii* were isolated from various samples, with predominance in pulmonary samples (36.84%) and from the intensive care unit (57.89%). These strains exhibited high resistance to 11 antibiotics tested, while all remained sensitive to colistin. Moreover, 15 strains were identified as *metallo-β-carbapenemase* producers, 10 as *cephalosporinase,* and three as extended-spectrum β-lactamases (ESBL) producers. Testing 59 actinobacterial strains, 19 (32.20%) showed activity against at least one A. baumannii strain, with the genus Saccharothrix exhibiting the highest activity. Streptomyces sp. WAB9 demonstrated the broadest spectrum of activity. These findings underscore the ongoing need for colistin as an effective antibiotic against *A. baumannii* and highlight the potential of certain actinobacterial strains as alternative sources for new bioactive molecules against multidrug-resistant *A.baumannii* strains.[59]

Mabon*iet al.* in the year 2020. This study addresses the occurrence and antimicrobial resistance of *Acinetobacter sp*. in animals, shedding light on their potential role in multidrug resistance dynamics. It identifies seven *Acinetobacter* species in various animal species, with MDR *A.baumannii* notably found in canine infections, suggesting a possible pathogenic profile and spread in the human environment. The research underscores the need for specific antimicrobial susceptibility guidelines for animal isolates due to varying resistance patterns and the absence of established clinical breakpoints. Overall, this study contributes to understanding the impact of animals on bacterial resistance and informs future efforts in establishing guidelines for testing animal-associated *Acinetobacter* isolates.[60]

Al-Tamimi*et al.* in the year 2022. This study conducted in Jordan over a decade examined 622 *Acinetobacter baumannii* isolates from tertiary hospitals. Most infections occurred in males aged 18–60, predominantly in surgery or critical care units, with infected wounds being common. Multidrug resistance (MDR) was prevalent, particularly in patients over 60, males, critically ill patients, and those with infected wounds. Resistance was highest for cephalosporins, carbapenems, and fluoroquinolones, with colistin and tigecycline showing the lowest resistance rates. OXA-51 and OXA-

23 genes were prevalent, while KPC and IMP genes were rare. This study underscores the importance of early identification, particularly in high-risk patients, and highlights colistin and tigecycline as effective treatment options.[61]

AIMS AND OBJECTIVES

- To isolate ***Acinetobacter*** species from various clinical samples obtained from the surgical intensive care unit (SICU)
- To analyze the antibiotic susceptibility profiles of ***Acinetobacter*** species identified in the SICU
- To examine the risk factors contributing to ***Acinetobacter*** infections in patients admitted to the SICU

MATERIALS AND METHODS

Study Design

This prospective study was conducted over six months, from February 2024 to August 2024, in the Department of Microbiology at Sheri Kashmir Institute of Medical Science, Soura.

Sample Collection

Clinical samples were collected from cases admitted to the surgical intensive care unit (SICU) of Sheri Kashmir Institute of Medical Science (SKIMS) Hospital. Demographic data including name, sanitarium number, age, coitus, instance point and date, former antibiotics taken within 48 hours before instance collection, opinion, and ICU specialty were strictly recorded. The samples from the ICU included tracheal aspirate, blood from central venous catheters, abdominal drain fluid, foam, and crack hearties, collected from cases with catheters.

Isolation and Identification of Acinetobacter

The collected samples were cultured on 5% sheep blood agar and MacConkey agar, then incubated aerobically overnight at 37°C. Characteristic colonies were identified, counted, and subjected to further analysis. *Acinetobacter* was linked through Gram staining, motility, and catalase and oxidase tests, using standard biochemical ways. These tests included indole, methyl red, citrate, urease, triple sugar iron agar (TSI), PPA, and Hugh and Leifson media. Colorful sugars were used for species identification.

Sensitivity Testing

Antibiotic vulnerability for each insulate was determined using the Kirby-Bauer slice prolixity system on Mueller-Hinton Agar, clinging to the 2023 CLSI recommendations, with 0.5 McFarland turbidity norms. The antibiotics evaluated included Amikacin (30µg), Amoxicillin (20/10µg), Ampicillin (10µg), Aztreonam (30µg), Cefoperazone-sulbactam (75/10µg), Cefepime (30µg), Cefotaxime (30µg), Cefoxitin (30µg), Ceftazidime (30µg), Chloramphenicol (30µg), Ciprofloxacin (5µ g), Cotrimoxazole (25µg), Gentamicin (30µg), Imipenem (10µg), Nalidixic acid (30µg), Netilmicin (30µg), Nitrofurantoin (30µg), Piperacillin-tazobactam (100/10µg), Tigecycline (30µg), and others with established antimicrobial susceptibility profiles. The vulnerability results were interpreted by measuring the zone compasses and relating them with CLSI norms.

Figure 1: *Acinetobacter baumannii* grows on MacConkey Agar.

Figure 2: *Acinetobacter baumannii* grows on Blood Agar.

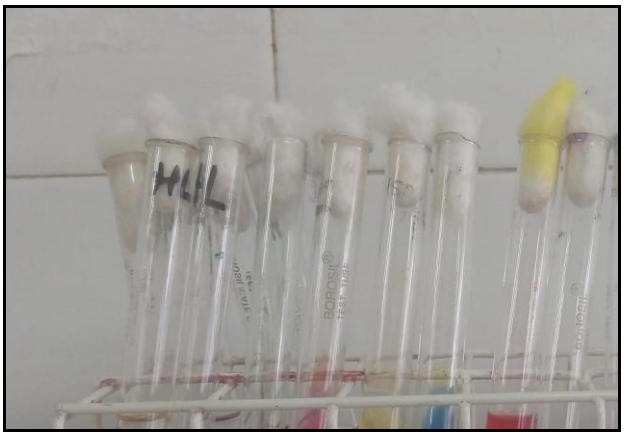

Figure 3: Set of Biochemical used for identification.

1) Hugh and Leifson: Fermented
2) Indole: Negative
3) PPA: Negative
4) Urease: Negative
5) Citrate: Citrate utilization
6) Methyl red: Negative
7) Triple sugar iron agar: Alk/A formation
8) Glucose: Fermented
9) Lactose: NF (non-fermented)
10) Sucrose: NF (non-fermented)
11) Mannitol: NF (non-fermented)

Figure 4: decarboxylases used in biochemical tests of A. baumannii.

Decarboxylases

1) Control: Negative
2) Lysine: Negative
3) Arginine: Negative
4) Ornithine: Negative

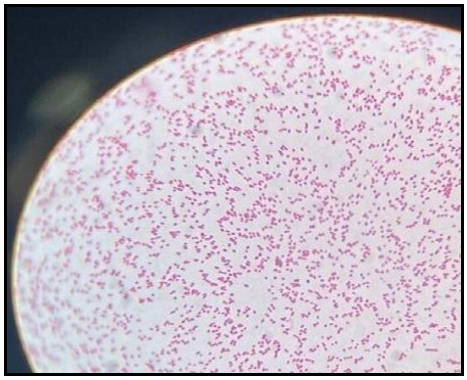

Figure 5: Gram stain of *Acinetobacter* (Gram-negative coccobacilli are seen).

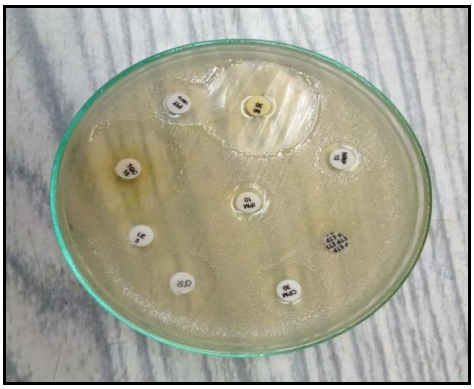

Figure 6: Antibiotic susceptibility on Mueller Hinton agar.

floxacin: Resistant

1) Meropenem: Resistant
2) Ampicillin+sulbactum: Resistant
3) Tigecycline: Sensitive
4) Ertapenem: Resistant
5) Cefpodoxine: Resistant
6) Cefepime: Moderate sensitive
7) Amikacin: Resistant
8) Polymyxin B: Sensitive
9) Ceftriaxone: Resistant
10) Levofloxacin: Resistant

RESULTS

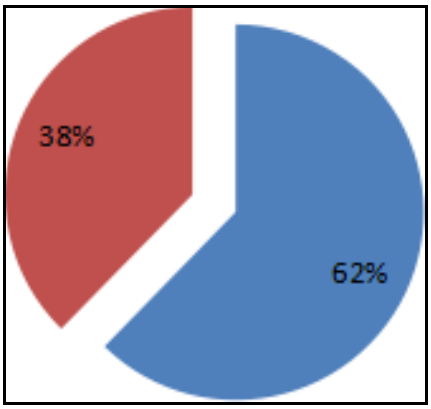

Figure 4: Chart depicting the total number of positive and negative samples (n=80).

During the six months, a total of 80 non- duplicate samples were collected from the surgical ICU at SKIMS and reused in the microbiology department. Of these 80 samples, 50 were set up to be positive, while 30 were sterile (Figure 4). Among the 50 positive samples, the breakdown of isolates was as follows 30 *Acinetobacterspp*. 8 *Klebsiellaspp*. 6 MRSA, 4 *Escherichia* coli, and 2 *Enterococcus* (Figure 5). These isolates were linked and vindicated using various styles in agreement with CLSI guidelines.

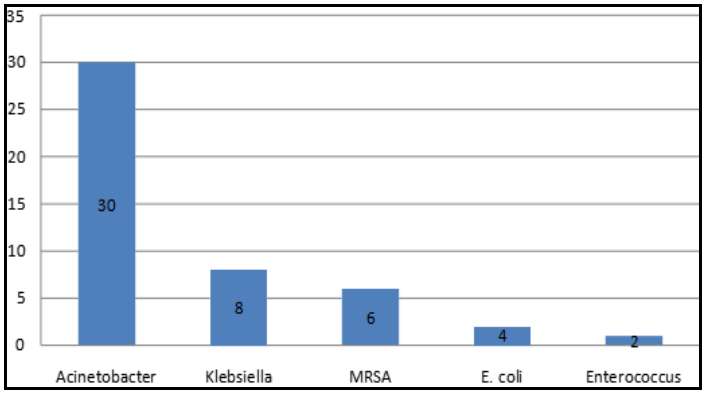

Figure 5: Chart depicting the total no. of isolates from various samples (n=50).

Among the *Acinetobacter sp*. isolates, the topmost chance(23.3) came from cases that progressed 60- 70 times, followed by16.7 from the 20- 40 time age group, 13.3 from the 40- 60 time age group,6.7 from the 10- 20 time age group, and 3.3 from the 0- 10 time age group(Table 1).

Of these isolates, 17(56.7) were from male cases, and 13(43.3) were from female cases (Table 2, Figure 6).

Table 2: Shows the age-wise distribution of *Acinetobacter species* (n=30)

Age	Presence of bacterial Specie	
	Acinetobacter species	
	No. of isolates	%
0-10	1	3.3
10-20	2	6.7
20-30	5	16.7
30-40	5	16.7
40-50	4	13.3
50-60	4	13.3
60-70	7	23.3
70 above	2	6.7
Total	**30**	**100.0**

Table 3: Total no. of males and females.

		The presence of bacterial species	
		Acinetobacter species	
		No.	%
Sex	Female	12	40.0
	Male	18	60.0
Total		30	100.0

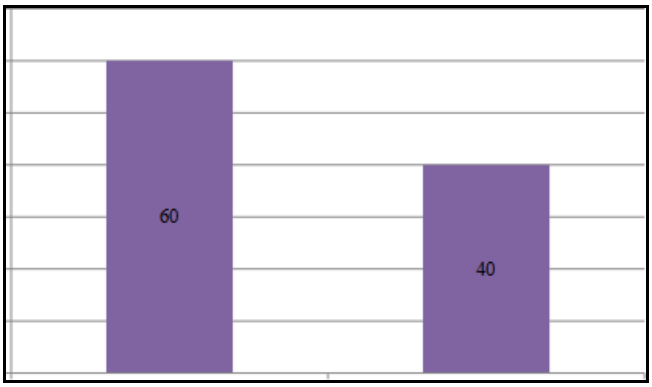

Figure 6: Sex-wise distribution of *Acinetobacter* infection.

The maturity of *Acinetobacter* strains was isolated from tracheal aspirates(96.6), with only 3.3 coming from crack mariners, and no isolates were set up in other sample types (Table 3, Figure7).

Among the cases whose societies were positive for *Acinetobacter*, 25(83.3) had dragged sanitarium stays of further than 7 days, while only 5(16.6) had been rehabilitated for 7 days or lower before *Acinetobacter* was isolated (Table 4).

Of the 30 cases with *Acinetobacter*-positive samples, 28(93.3) were on ventilator support, and 2(6.6) had endured surgery within the last seven days before the sequestration of *Acinetobacter* (Table 5, Figure 8).

Before the sequestration of *Acinetobacter* species, 23 cases had been on antibiotics, including Ceftriaxone (12 cases), Imipenem (1 case), Levofloxacin (5 cases), and Cefaprazone (5 cases), while only 7 cases had no history of antibiotic use(Table 6).

Table 4: Sample-wise distribution of *Acinetobacter* isolates.

		The presence of bacterial species	
		Acinetobacter species	
		No.	**%**
Sample	**Tracheal aspirate**	29	96.6
	Wound swab	1	3.3
Total		**30**	**100.0**

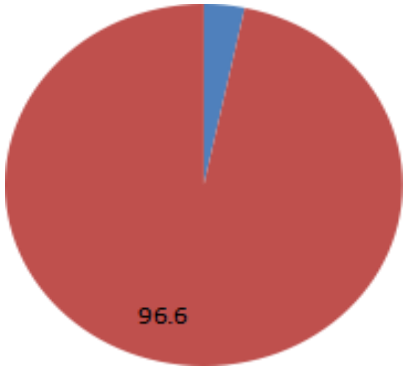

Figure 7: Sample-wise distribution of *Acinetobacter* isolates.

Table 6: Factors contributing to the risk of Acinetobacter species.

Hospital stay	No.	%
≤ 7	5	16.6
≥ 7	25	83.3
Total	**30**	**100.0**

Risk factors			
Presence of bacterial species	*Acinetobacter species*		
_	**Prior Surgery**	**Ventilator support**	**Total**
No.	2	28	**30**
%	6.6	93.3	**100.0**

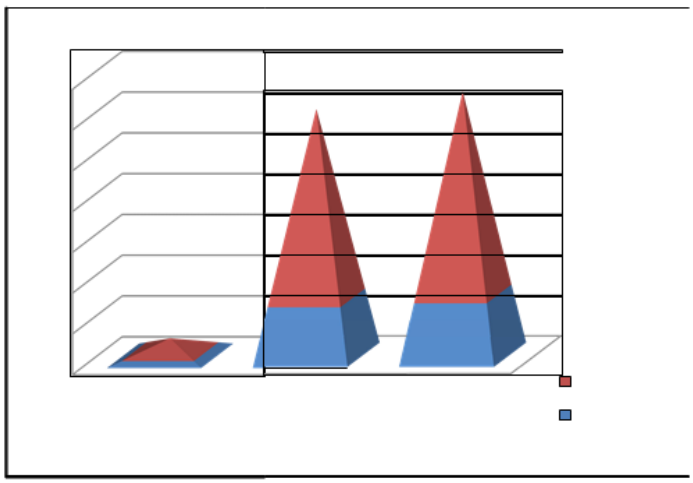

Table 7: Prior history of antibiotics before isolation of *Acinetobacter species.*

H/O antibiotics before isolation	No. of patients
Ceftriaxone	12
Imipenem	1
Levofloxacin	5
Cefaprazone	5
No antibiotic	7
Total	**30**

In this study, all *Acinetobacter sp*. isolates (100) showed resistance to Ampicillin. Twenty- nine isolates (96.6) were resistant to both Amikacin and Tobramycin. High resistance rates were also observed for Tetracycline, Tigecycline, Gentamicin, Ofloxacin, Levofloxacin, Aztreonam, and Cefotaxime (93.3 each). Also, 27 isolates (90) were resistant to Cefepime, 86.6 to Meropenem, Imipenem, and Ertapenem each, and 83.3 to Ciprofloxacin and Norfloxacin each. Ceftriaxone resistance was detected in 80 of the isolates. Resistance to Ampicillin Sulbactum was seen in 53.3 of the isolates, CefaprazoneSulbactum in 50, and PipercillinTazobactum in 36.6 of the isolates.

All 100 *Acinetobacter* isolates exhibited sensitivity to Polymyxin B.Nineteen isolates (63.3) were sensitive to PipercillinTazobactum, and 50 were sensitive to CefaprazoneSulbactum. Ampicillin Sulbactum perceptivity was observed in 46.6 of the isolates. Tigecycline perceptivity was seen in 6.6, Ceftriaxone in 20, and Ciprofloxacin, Norfloxacin, Aztreonam, and Cefotaxime perceptivity in 16.6 each. Perceptivity to Imipenem, Ertapenem, Meropenem, and Ceftazidime was seen in13.3 of the isolates each, Cefepime in 10, and Amikacin and Tobramycin in3.3 of the isolates (Table 7, figures 9 and 10).

Table 8: Antibiotic susceptibility pattern of *Acinetobacter species* isolated.

Antibiotics	Sensitive (%)	Resistant (%)
Polymyxin B	30(100%)	0
Tetracycline	2(6.6%)	28
Tigecycline	2(6.6%)	28
Imipenem	4(13.3%)	26
Amikacin	1(3.3%)	29
Gentamicin	2(6.6%)	28
Ceftriaxone	6(20%)	24
Pipercillin + Tazobactum	19(63.3%)	11
Ampicillin + Sulbactam	14(46.6%)	16
Ciprofloxacin	5(16.6%)	25
Ertapenem	4(13.3%)	26
Meropenem	4(13.3%)	26
Ofloxacin	2(6.6%)	28
Levofloxacin	2(6.6%)	28
Cefepime	3(10%)	27
Cefaprazone + Sulbactum	15(50%)	15
Norfloxacin	5(16.6%)	25
Ceftazidime	4(13.3%)	26
Ampicillin	0(0%)	30
Tobramycin	1(3.3%)	29
Aztreonam	2(6.6%)	28
Cefotaxime	2(6.6%)	28

DISCUSSION

Acinetobacter species are typically harmless but can survive in hospital settings for prolonged durations. Although patient colonization is common, *Acinetobacter* can cause serious infections, particularly in ICU patients or those with underlying conditions. Recently, hospital-acquired infections by this genus have increased. *Acinetobacter baumannii* is the primary cause of such infections, leading to pneumonia, endocarditis, meningitis, and wound and urinary tract infections.

In our study, we recovered 80 bacterial isolates from SKIMS ICU patients over six months, with 30 (38.96%) being *Acinetobacter* species. A similar study by Nahar A *et al.* (2012).[62] reported a prevalence of 33.7%, lower than ours. Bhattacharyya S *et al.* (2013).[63] found a 33.3% prevalence, also lower. Huidrom S et al. (2015).[64] noted a 12.6% prevalence, significantly lower. Sharma P *et al.* (2015).[65] identified *Acinetobacter baumannii* in 19.7% of gram-negative isolates from 2116 samples, lower than our hospital. The higher prevalence in our hospital may be due to broad-spectrum antibiotic

use in the ICU. Kumar et al. (2014).[66] reported a 79.93% prevalence from 294 NFGNB isolates, higher than our study.

Most *Acinetobacter* species in our study were from the 60-70 years age group (23.3%), predominantly older patients. Bhattacharyya S *et al.* (2013).[63] reported a mean patient age of 27 years, lower than our study. Huidrom S *et al.* (2015).[64] observed a wide age range, with most patients (38.7%) in the 60-70 years age group, similar to our findings. Rola N et al. (1999).[67] reported a mean age of 50 (range 21-84), also lower. Rit K *et al.* (2012).[68] noted *Acinetobacter* infections were more common in patients over 40 years old.

Acinetobacter species were more frequently isolated from males (56.7%) than females (43.3%), showing a slight male preponderance. Bhattacharyya S *et al.* (2013).[63] found a higher male-to-female ratio (46:1). Huidrom H *et al.* (2015).[64] reported 66.1% male and 33.9% female isolates. Rit K *et al.* (2012).[68] in India found a male-to-female ratio of 1.5:1, while Özdemir *et al.* (2011).[50] in Turkey reported a ratio of 1:1.5.

Most *Acinetobacter* species were isolated from tracheal aspirates (96.8%), followed by wound swabs (3.3%). Nahar A *et al.* (2012).[65] in Bangladesh reported 100% isolation from endotracheal tubes, followed by other sources. Bhattacharya S *et al.* (2013) in India reported higher isolation from urine (54%), pus (23%), CSF (12%), and other samples (11%). Huidrom S *et al.* (2015) found *Acinetobacter* species mainly from pus/swabs (22.6%).

Our study focused on hospital stay length. Patients were divided into two groups: those with a duration of 7 days or less and those exceeding 7 days. We found 25 patients (83.3%) with >7 days of hospital stay and 5 patients (16.6%) with ≤7 days, indicating clinical significance. Aimsaad L. et al. (2009).[69] reported a median hospital stay of 35 days for *Acinetobacter*. Husni R *et al.* (1999) found a mean ICU stay of 12.8 days before infection. Biendo M *et al.* (1999)[70] noted a mean ICU stay of 16.7 days (range 2-210 days), aligning with our study.

Our study investigated the risk factors associated with hospital-acquired Acinetobacter infections. Patients on ventilators (93.3%) and those who underwent recent surgery (6.6%) were at higher risk. Additionally, 76.6% of patients were on broad-spectrum antibiotics before *Acinetobacter* isolation. Ozdemir *et al.* (2011) reported that 92.3% of patients were mechanically ventilated, and 85% were treated with multiple antibiotics before infection, similar to our findings. A longer hospital stay (>7 days) was a significant predictor of *Acinetobacter* infection.

All *Acinetobacter* species in our study were 100% resistant to Ampicillin and 100% sensitive to Polymyxin B. High resistance was seen for Amikacin, Tobramycin (96.6% each), Tetracycline, Tigecycline, Gentamicin, Ofloxacin, Levofloxacin, Aztreonam, and Cefotaxime (93.3% each). Cefepime resistance was 90%, and resistance to Ceftazidime, Ertapenem, Imipenem, and Meropenem was 86.6% each. Ciprofloxacin and Norfloxacin resistance was 83.3% each, and Ceftriaxone was 80%. Lower resistance was seen for Ampicillin+sulbactum (53.3%) and Cefoperazone-sulbactam (50%), with the least resistance to Piperacillin-tazobactam (36.6%).

Nahar A. *et al.* (2012) found *Acinetobacter* species were 100% resistant to Amoxicillin, Cefuroxime, Ceftriaxone, and Gentamicin, with high resistance to Amikacin (68.4%) and Imipenem (66.7%), and low resistance to Colistin (10.5%). Gayathri P et al. (2015) reported 15% Imipenem resistance, lower than our 96.6%, possibly due to empirical Imipenem use in our settings. Safari M et al. (2013) found high resistance to Aztreonam, Ceftazidime, Ciprofloxacin, Piperacillin, and Cefotaxime (97-98% each), with no sensitive isolates for Aztreonam and Cefotaxime, and minimal Tigecycline resistance. In our study, 6.6% were sensitive to Aztreonam and Cefotaxime, and 93.3% were resistant to Tigecycline. High sensitivity was noted for Piperacillin-tazobactam (63.3%), followed by Cefoperazone-sulbactam(50%), Ampicillin+sulbactum (46.6%), Tigecycline (6.6%), and Ceftriaxone (20%). Lower sensitivity was observed for Ceftazidime (13.3%) and other antibiotics (6.6% each). Least sensitivity was seen for Imipenem, Amikacin, Ertapenem, and Tobramycin (3.3% each). Huidrom S et al. (2015) in India reported high resistance to Ampicillin (83.9%), Amikacin (77.4%), Gentamicin (77.4%), Ceftazidime (85.5%), Ceftriaxone (62.9%), Cefotaxime (77.4%), Imipenem (40.3%), Meropenem (50.0%), and Cotrimoxazole (83.9%), with 90.3% MDR. All isolates were 100% sensitive to Colistin. Our study's findings are in agreement with these results.

CONCLUSION

In conclusion, *Acinetobacter* has become a significant threat in hospital acquired infections, particularly among critically ill patients in intensive care units. Our study found that *Acinetobacter* species exhibit high resistance to most commonly used antibiotics. Notably, lower resistance was observed with Piperacillin-tazobactam (36.6%). The hospital-based epidemiological data revealed a very high incidence of multidrug-resistant (MDR) *Acinetobacter* isolates. However, all isolates were sensitive to Polymyxin B (100%), and a significant proportion was sensitive to Piperacillin-tazobactam (63.3%).

This alarming data underscores the growing problem of MDR *Acinetobacter* infections, especially in ICU settings, presenting a substantial challenge for physicians. The emergence of these resistant strains complicates treatment protocols and necessitates the development of comprehensive antibiotic policies and guidelines at a national level.

To combat this issue effectively, producing a local antibiogram database is essential. Such a database would enhance the understanding of antimicrobial resistance patterns specific to a tertiary care hospital. This improved knowledge would aid in formulating better treatment strategies, ensuring that healthcare providers can respond more effectively to infections caused by MDR *Acinetobacter* species. Implementing these measures is crucial to manage and mitigate the impact of these resistant infections on patient outcomes in ICU settings.

REFERENCES

1. Corbella X. Montera A. Pujol Metal. Emergence and rapid spread of carbapenem resistance during a large and sustained hospital outbreak of multiresitam*Acinetobacter baumannii.* J ClinMicrobiol 2000; 38: 4086-95.
2. Houang ETS, Chu YW, Leung CM *et al.* Epidemiology and infection control implications of *Acinetobacter spp*. in Hong Kong. J ClinMicrobiol., 2001; 39: 228-34.
3. Chastre J. Trouillet JL et al. Problem pathogens (*Pseudomonas aeruginosa* and *Acinetobacter*). Semin Respir Infect, 2000; 15: 287-98.
4. Chastre J *et al* Infections due to *Acinetobacterbaumannii* in the ICU. SeminRespCrit Care Med 2003; 24: 69-78.
5. Mandel AD, Wright K, McKinnon JM, *et al.* Selective medium for selection of Mima and Herellea organisms. J Bacteriol., 1964; 88: 1524-5.
6. Fournier PE, Richet H *et al.* The epidemiology and control of *Acinetobacter baumannii* in health care facilities. Clinical Infections Diseases, 2006; 42: 692-699.
7. WarskowAL, Juni E *et al.* Nutritional requirements of *Acinetobacter* strains isolated from soil, water, and sewage. Journal of Bacteriology, 1972; 112: 1014-1016.
8. Bergogne-Berezin E, Towner KJ *et al. Acinetobacter spp*. As nosocomial pathogens: microbiological, clinical, and epidemiological features. ClinMicrobiol Rev., 1996; 9: 148-65.
9. Afzal-Shah M, Livermore DM, *et al.* Worldwide emergence of carbapenem-resistant *Acinetobacter spp*. 1998. J Antimicrob Chemother, 1998; 41: 576-7.
10. Srifuengfung S, Sangsawang M, Komolpis P *et al.* Bacterial pathogens (non-myco bacterium) from sputum culture and antimicrobial susceptibility. Southeast Asian J Trop Med Public Health, 1998; 29: 96-9.
11. National Nosocomial Infections Surveillance (NNIS) System report, data summary from January 1990- May 1999, issued June 1999. Am J Infect Control, 1999; 27: 520- 32.
12. Yu YS, Xu XW, Kong HS *et al.* Typing and characterization of carbapenem-resistant *Acinetobacter calcoaceticus-baumannii* complex in a Chinese hospital. J Med Microbiol., 2004; 53(Pt 7): 653-6.
13. Agusti C, Pujol M, Argerich MJ *et al.* Short-term effect of the application of selective decontamination of the digestive tract on different body site reservoir ICU patients colonized by multiresistant *Acinetobacter baumami.* J Antimicrob Chemother, 2002; 49: 205-8.
14. Chu YW, Leung CM, Houang ET *et al.* Skin carriage Of *Acinetobacters* in Hong Kong, J Clin Microbiol., 1999; 37: 2962-7.
15. Anstey NM, Currie BJ, Withnall KM, *et al.* Communityacquired*Acinetobacter pneumonia* in the Northern Territory of Australia. Clin Infect Dis., 1992; 14: 83-91.
16. Villers D, Espaze E, Coste-Burel M et al. Nosocomial *Acinetobacter baumannii* infections: microbiological and clinical epidemiology. Ann Intern Med., 1998; 129: 182-189.
17. Strulens MJ, Carlier E, Maes N, *et al*. Nosocomial colonization and infection with multiresistant*Acinetobacter baumannii*: outbreak delineation using DNA macrorestriction analysis and PCR-fingerprinting. J Hosp Infect, 1993; 25: 15-32.
18. Bergogne-Be'rezin E *etal. Acinetobacter spp*. As nosocomial pathogens: microbiological, clinical and epidemiological features Clin Microbial Rev., 1996; 9: 14865.
19. Hangerger H. Garcia Rodriguez JA, Gobernado M *et al*. Antibiotic susceptibility among aerobic gram-negative bacilli in intensive care units in 5 European countries. JAMA, 1999; 281: 67-71.
20. Bergogne-Be're'zin E *et al. Acinetobacter spp*. As nosocomial pathogens: microbiological, clinical and epidemiological features. ClinMicrobiol Rev., 1996; 9: 148-165.
21. Webster C, Towner KJ, Humphreys H, *et al.* Survival of *Acinetobacter* on three clinically related inanimate surfaces. Infect Control HospEpidemiol., 2000; 21: 246.
22. Zheng W, Yuan S, Li L *et al.* Analysis of hospital departmental distribution and antibiotic susceptibility of *Acinetobacter* isolated from sputum samples. Am J Infect Control, 2013; In press.
23. PoirelL.Nordmann P *et al.*Carbapenem resistance in *Acinetobacter baumannii:*
24. mechanisms and epidemiology. ClinMicrobiol Infect., 2006; 12(9): 826-836.
25. Neonakis IK, Spandidos DA, Petinaki E *et al.* Confronting multidrug-resistant *Acinetobacter baumannii*: a review. Int J Antimicrob Agents, 2011; 37(2): 102-109
26. Karalı N. Sundsfjord A. Towner K *et al.* Insights into the global molecular epidemiology of carbapenem non-susceptible clones of *Acinetobacter baumannii*. Drug Resist Updat., 2012; 15(4): 237-247.
27. Miller GH, Sabatelli FJ, Hare RS *et al.* The most frequent aminoglycoside resistance mechanisms change with time and geographic area: a reflection of aminoglycoside usage patterns? Aminoglycoside

Resistance Study Groups. Clin Infect Dis., 1997; 24(Suppl 1): 846-562.
28. Danes C, Navia NM, Ruiz J *et al*. Distribution of B-lactamases in *Acinetobacter baumannii* clinical isolates and the effect of Syn 2190 (Amp C inhibitor) on the MICs of different ß-lactam antibiotics. J Antimicrob Chemother, 2002; 50: 261-4.
29. Quale J, Bratu S, Landman D *et al*. Molecular epidemiology and mechanisms of carbapenem resistance in *Acinetobacter baumannii* endemic in New York City. Clin Infect Dis., 2003; 37: 214-20.
30. Bou G, Cervero G, Dominguez MA *et al*. Characterization of a nosocomial outbreak caused by a multiresistant*Acinetobacter baumannii* strain with carbapenems- hydrolyzing enzyme: high-level carbapenems resistance in *A. baumannii* is not due solely to the presence of beta-lactamases. J ClinMicrobiol., 2000; 38: 3299-305.
31. Vila J, Ruiz J, Goni P *et al*. Quinolone resistance mutations in the topoisomerase IV par gene of *Acinetobacter baumannii*. J Antimicrob Chemother, 1997; 39: 757-62.
32. Tankovic JP, Legrand P, De Gatines G *et al.* Characterization of a hospital outbreak of imipenem-resistant *Acinetobacter baumannii* by phenotypic and genotypic typing methods. J ClinMicrobiol., 1994; 32: 2677-81.
33. Castle, Tenney JH, Weinstein MP M, *et al.* The outbreak of a multiply resistant *Acinetobacter* in a surgical intensive care unit: epidemiology and control. Heart Lung, 1978; 7: 641-4.
34. Villers D, Espaze E, Coste-Burel M *et al.* Nosocomial *Acinetobacter baumannii* infections: microbiological and clinical epidemiology. Ann Intern Med., 1998; 129: 182-9,
35. Kanafani ZA, Kara L. Hayek S *et al*. Ventilator-associated pneumonia at a tertiary care center in a developing country, incidence, microbiology, and susceptibility patterns of isolated microorganisms. Infect Control HospEpidemiol., 2003; 24: 864-9.
36. Fridkin SK *et al*. Increasing prevalence of antimicrobial resistance in intensive care units. Crit Care Med., 2001; 29(Suppl 4): N64-8.
37. Go ES. Urban C. Bums J *et al*. Clinical and molecular epidemiology of *Acinetobacter* infections sensitive only to polymyxin B and sulbactam. Lancet., 1994; 344: 1329-32.
38. Tomasz A et al. Multiple-antibiotic-resistant pathogenic bacteria. N Engl J Med., 1994; 330: 1247-51.
39. Karlowsky JA, Draghi DC, Jones ME *et al.* Surveillance for antimicrobial susceptibility among clinical isolates of *Pseudomonas aeruginosa* and *AcinetobacterBaumanni* from hospitalized patients in the United States, 1998 to 2001. Antimicrobial Agents Chemother, 2003; 47: 1681-8.
40. Thongpiyapoom S, Narong MN, Suwalak N *et al*. Device-associated infections and patterns of antimicrobial resistance in a medical-surgical intensive care unit in a university hospital in Thailand. J Med Assoc Thai., 2004; 87: 819-24.
41. PoutanenS.M, LouieM.Simor AE *et al*. Risk factors, clinical features and outcome of *acinetobacter bacteremia* in adults Eur. J. Clin. Microbiol Infect. Dis., 1997; 16.
42. Vincent RO, Noora A Al-Sweih, "JalilahFeteihet al. The Prevalence and Antibiotic Susceptibility Pattern of Gram-negative Bacterial Isolates in Two ICUs in Saudi Arabia and Kuwait. Diagnostic Microbiology and Infectious Disease, January 1998; 30(1): 53-59.
43. Hong Siau, Kwok-Yung Yuen, Pak-Leung Ho *et al*. *AcinetobacterBacteremia* in Hong Kong: Prospective Study and Review Clinical Infectious Diseases, 1999; 28: 26-30.
44. Carl Urban, Sorana Segal-Maurer, James J. Rahal*et al*. Considerations in Control and Treatment of Nosocomial Infections Due to Multidrug-Resistant *Acinetobacterbaumannii* Clinical Infectious Diseases, 2003; 36: 1268-74.
45. Martro E. A. Hernandeza, J. Arizaet *al*. Assessment of Acinetobacter baumanniisusceptibility to antiseptics and disinfectants Journal of Hospital Infection Rungruanghiranya S, Charurat Somboonwit, Termkiat Kanchanapoom Assessment of *Acinetobacter baumannii* susceptibility to antiseptics and disinfectants J Infect Dis Antimicrob Agents, 2005; 22: 77-92.
46. Lisa L, Maragakis, and Trish Perl *et al.Acinetobacter baumannii:* Epidemiology, Antimicrobial Resistance, and Treatment Options. Clinical Infectious Diseases, 2008; 46: 1254-63.
47. Tacconelli E, Daniela Manno, Teresa Spanu*et al.* Prediction models to identify hospitalized patients at risk of being colonized or infected with multidrug-resistant *Acinetobacter baumannii calcoaceticus complex* Journal of Antimicrobial Chemotherapy, 2008; 62: 1130-1137. doi: 10.1093/jac/dkn289.
48. Enoch DA, C Summers b, NM Brown, *et al*. Investigation and management of an outbreak of multidrug-carbapenem-resistant *Acinetobacter baumannii* in Cambridge, UK Journal of Hospital Infection, 2008; 70: 109-118.
49. Saranraj P,D Stella *et al.* Antibiogram of Nosocomial Infections and Its Antimicrobial Drug Resistance International Journal of Pharmaceutical & Biological Archives, 2011; 2(6): 1598-1610.
50. Özdemir, Halil, Kendirli *et al.* Nosocomial infections due to *Acinetobacter baumannii* in a pediatric intensive care unit in Turkey. Nosocomial infections due to *Acinetobacter*, May 1, 2011.
51. AzizunN, Shaheda Anwar, Ahmad Abu Saleh*et al.* Isolation of *Acinetobacter species* and their antimicrobial resistance pattern in an Intensive care unit (ICU) of a tertiary care hospital in Dhaka Bangladesh J Med Microbial., 2012; 06 (01): 03-06.
52. Bhattacharyya S, Bhattacharyya 1, Rit K *et al*. Antibiogram of *Acinetobacter spp.* Isolated from various clinical specimens in a tertiary care hospital in West Bengal, India. Biomedical Research, 2013;

24(1): 43-46.
53. Kumar E, K Usha, BV Ramana*et al.*Prevalence of *Acinetobacter species* and its antibiogram pattern in a tertiary care hospital, Tirupati, Andhra Pradesh DVR Sui Gopal, UPRBS, 2014; 3(1): 352-358.
54. Gayathri Devi P, V Alice Raniand K Saileela *et al*. Isolation of *acinetobacter species* in ICU.Der Pharmacia Lettre, 2015; 7(7): 49-51.
55. Darcy Ellis, Bevin Cohen, Jianfang Liu *et al*. Risk factors for hospital-acquired antimicrobial-resistant infection caused by *Acinetobacter baumannii* Antimicrobial Resistance and Infection Control, 2015; 4: 40. DOI 10.1186/s13756-015-0083-2.
56. Lob, S.H., Hoban, D. *et al.* 2016. Regional differences and trends in antimicrobial susceptibility of *Acinetobacter baumannii.* International journal of antimicrobial agents, *47*(4): 317-323.
57. Castilho, S.R.A., *et al.* 2017. *Acinetobacter baumannii* strains isolated from patients in intensive care units in Goiânia, Brazil: Molecular and drug susceptibility profiles. *PLoS One*, *12*(5): e0176790.
58. Tewari, R., Chopra D *et al.* 2018. Antimicrobial susceptibility patterns of an emerging multidrug-resistant nosocomial pathogen: *Acinetobacter baumannii. The Malaysian Journal of Medical Sciences: MJMS*, *25*(3): 129.
59. Namiganda, V., Mina, Y., *et al.* 2019. Antibiotic Resistance Pattern of *Acinetobacter baumannii* strains isolated from different clinical specimens and their sensibility against bioactive molecules produced by Actinobacteria. *Arabian Journal for Science and Engineering*, *44:* 6267-6275.
60. Maboni, G., S, M. *et al.* 2020. Antimicrobial resistance patterns of *Acinetobacter spp*. of animal origin reveal a high rate of multidrug resistance. *Veterinary Microbiology*, *245:* p.108702. Maboni, G., Seguel, M., Lorton, A. and Sanchez, S., 2020. Antimicrobial resistance patterns of *Acinetobacter spp*. of animal origin reveal a high rate of multidrug resistance. *Veterinary Microbiology*, *245:*108702.
61. Al-Tamimi, M.A *et al.* 2022. Multidrug-resistant *Acinetobacter baumannii* in Jordan. *Microorganisms*, *10*(5): 849.
62. Nahar N, Anwar S, Saleh A, Ruhul MD. Isolation of Acinetobacter species and their antimicrobial resistance pattern in an Intensive care unit (ICU) of a tertiary care hospital in Dhaka, Bangladesh Bangladesh J Med Microbial., 2012; 06(01): 03-06.
63. Bhattacharyya S, Bhattacharyya 1, Rit K, Mukhopadhyay PK, Dey JB, Ganguly U, Ray R. Antibiogram of Acinetobacter spp. Isolated from various clinical specimens in a tertiary care hospital in West Bengal, India. Biomedical Research, 2013; 24(1): 43-46
64. Huidrom S, Girish N, Rajendran R. Isolation and study of antimicrobial susceptibility and resistance patterns of Acinetobacter spp. from Intensive Care Unit of a tertiary care hospital in Bengaluru International Journal of Advanced Research, 2015; 3(8): 1121-1125.
65. Sharma P, Bashir Y, Kaur S, Kaur P, Aggarwal. Emerging Antimicrobial resistance and clinical relevance of Acinetobacter isolates in a tertiary care hospital of rural area of Punjab, India. Journal of Microbiology and Antimicrobial Agents, 2015; 1(1): 8-12.
66. Kumar E, Usha K, Ramana BV, Chaudhary A, Gopal. Prevalence of Acinetobacter species and its antibiogram pattern in a tertiary care hospital, Tirupati, Andhra Pradesh DVR SaiGopal, IJPRBS, 2014; 3(1): 352-358.
67. Rola N, Lawrence S, Alejandro C. Risk factors for an outbreak of multi-drug-resistant Acinetobacter nosocomial Pneumonia among intubated patients. Chest, 1999; 115(5): 1378-1382. doi: 10.1378/chest.115.5.1378.
68. Rit K, Saha R. Multidrug-resistant acinetobacter infection and their susceptibility patterns in a tertiary care hospital. Niger Med J., 2012 Mar 4; 53: 126-8.
69. Aimsaad L., Diraphat P, UtrarachkijF. Epidemiological Characteristics of Acinetobacter baumannii Infections at Phramongkutklao Hospital.J Med Assoc Thai., 2009; 92(Suppl 7): $164-72.
70. Biendo M. Laurans G, Lefebvre JF, Daoudi F. Epidemiological Study of an Acinetobacter baumannii outbreak by using a combination of antibiotyping and ribotyping Journal of clinical.