

Anitmicrobial Susceptibility Pattern of Acinetobacter Baumanni at Tertiary Care Hospital in Lahore Pakistan

Muhammad Shahid Shakoor ¹, Muhammad Zeeshan², Tehmina Tariq ³, Fakhrulislam Bareman ⁴, Bushra Zainab ⁵, Arooj Amjad ⁶

DOI: <https://doi.org/10.63163/jpehss.v3i2.291>

Abstract

Objective: The aim of study was to check the Anitmicrobial Susceptibility Pattern of Acinetobacter baumannii at tertiary care hospital in lahore Pakistan.

Methodology: Data from clinical specimens of patients with Acinetobacter baumannii infection were retrospectively studied. Standard microbiological techniques were used to screen for antibiotic susceptibility, and the results were then examined to identify patterns of resistance.

Results: Out of 50 clinical isolates of Acinetobacter baumannii, the highest infection rate (52%) was observed in patients above 51 years of age, with a slight female predominance (54%). Alarming levels of resistance were recorded against commonly used antibiotics: 92% to ciprofloxacin, 80% to meropenem, 70% to imipenem, and 72% to gentamicin. Only 24%, 8%, 6%, and 16% of isolates, respectively, remained sensitive to these antibiotics. No significant association was found between gender and resistance patterns for any of the tested drugs ($p > 0.05$). These findings indicate a high prevalence of multidrug-resistant A. baumannii strains in the hospital setting, underscoring the urgent need for improved antimicrobial stewardship.

Conclusion: The study spotlighted Acinetobacter baumannii's antibiotic susceptibility pattern at a tertiary care facility in Lahore, Pakistan. In order to address the growing problem of antibiotic resistance, these findings are crucial for directing antimicrobial stewardship initiatives and empirical treatment decisions.

Keywords: Acinetobacter baumannii, antimicrobial susceptibility

Introduction

Acinetobacter baumannii is a Gram-negative bacterium with a distinctive shape short, slightly rounded, and rod-like (often called a coccobacillus). It was named after Paul Baumann, the bacteriologist who identified it. ⁽¹⁾ This pathogen primarily targets people with weakened immune systems, making it a growing concern in hospitals as a common source of nosocomial (hospital-acquired) infections.

While other Acinetobacter species are frequently found in soil leading some to mistakenly assume A. baumannii also thrives there this particular bacterium is rarely seen outside clinical settings. ⁽²⁾ There have been a few reports of its presence in soil and water samples ⁽³⁾, but its true natural habitat remains unknown.

Bacteria from the Acinetobacter genus don't have flagella, but they can still move—either through a twitching motion or swarming behavior, which is probably driven by type IV pili. A. baumannii might also get around by releasing exopolysaccharide, forming a sticky trail of sugar chains that helps push the bacterium forward ⁽⁴⁾. When identifying Acinetobacter in the lab, microbiologists often use an oxidase test to distinguish it from other Moraxellaceae family

members. That's because *Acinetobacter* species are unique in this group for missing cytochrome c oxidases ⁽⁵⁾.

Acinetobacter baumannii belongs to the **ACB complex**, a group that also includes *A. calcoaceticus* and *Acinetobacter* genomic species **13TU**. These species are not only difficult to distinguish from one another but also represent the most clinically significant pathogens within the *Acinetobacter* genus ^(6,7).

Additionally, *A. baumannii* is classified as one of the **ESKAPE pathogens**—a dangerous group of bacteria known for their high antibiotic resistance and role in the majority of hospital-acquired infections. ⁽⁸⁾

Types:

- **Carbapenems** were once the go-to treatment for multidrug-resistant (MDR) *A. baumannii* infections. However, overuse has led to a troubling rise in carbapenem-resistant strains in recent years. ⁽⁹⁾
- **Polymyxins** have now become the primary antibiotics for MDR *A. baumannii*, despite being initially avoided due to their potential toxic side effects, such as kidney damage and neurotoxicity. ^(10,11)
- **Extensively drug-resistant (XDR) *A. baumannii*** is defined as resistance to at least three major antibiotic classes, including:
 - Penicillins and cephalosporins (even with inhibitor combinations)
 - Fluoroquinolones
 - Aminoglycosides
 - (In most cases) Carbapenems
- **Pandrug-resistant (PDR) *A. baumannii*** takes resistance a step further—these are XDR strains that also resist last-resort drugs like polymyxins and tigecycline. The growing threat of XDR and PDR strains has spurred research into new antimicrobials and alternative treatment strategies. ^(12,13)

Mechanisms of Antibiotic Resistance:

Acinetobacter baumannii employs three primary strategies to resist antibiotics:

1. **Blocking Access:** The bacterium either reduces membrane permeability or activates efflux pumps to prevent antibiotics from reaching their targets inside the cell.
2. **Target Modification:** Through genetic mutations or post-translational changes, *A. baumannii* can alter antibiotic targets, making them unrecognizable to the drugs.
3. **Antibiotic Destruction:** The bacteria can directly neutralize antibiotics through hydrolysis or enzymatic modification.

What makes *A. baumannii* particularly formidable is its remarkable genetic flexibility. This allows for:

- Rapid mutations and genetic rearrangements
- Efficient incorporation of resistance genes from mobile genetic elements

Among these adaptive mechanisms, insertion sequences play a crucial role in reshaping the bacterial genome, driving the evolution of increasingly resistant strains.

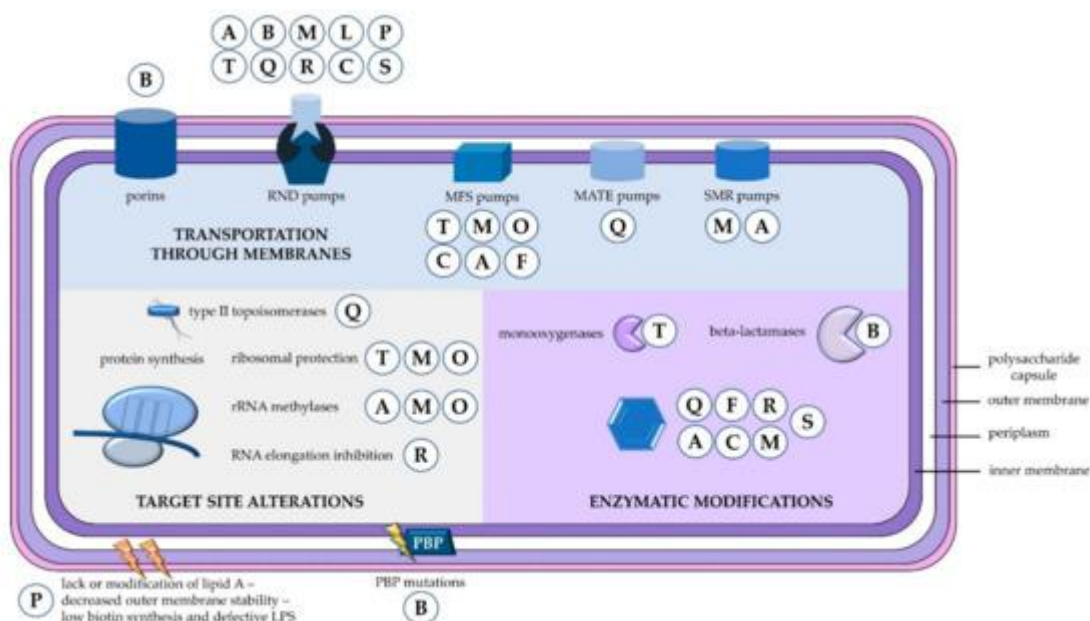


Figure 1. *A. baumannii* employs three primary strategies to resist antibiotics: (1) restricting antibiotic uptake by reducing porin permeability or activating efflux pumps (e.g., RND, MFS, MATE, or SMR families); (2) modifying drug targets (e.g., altered PBPs for β -lactams or mutated DNA gyrase for fluoroquinolones); and (3) enzymatic inactivation (e.g., β -lactamases hydrolyzing beta-lactams [B] or aminoglycoside-modifying enzymes degrading aminoglycosides [A]). These mechanisms are often encoded by mobile genetic elements, enabling rapid spread across strains. The pathogen's arsenal includes resistance to nearly all drug classes: aminoglycosides [A], β -lactams [B], chloramphenicol [C], fosfomycin [F], lincosamides [L], macrolides [M], polymyxins [P], fluoroquinolones [Q], rifamycins [R], sulfonamides/diaminopyrimidines [S], tetracyclines [T], and oxazolidinones [O]. This multidrug resistance, coupled with its environmental persistence, underscores *A. baumannii*'s status as a critical ESKAPE pathogen.

Clinical Significance:

Acinetobacter baumannii has emerged as a formidable multidrug-resistant pathogen capable of causing serious infections across both healthcare and community environments. This versatile bacterium most commonly presents as pneumonia, particularly ventilator-associated cases in ICUs, though increasingly reported as community-acquired pneumonia in tropical regions. It frequently causes bloodstream infections, often secondary to pulmonary or wound infections, with alarmingly high mortality rates. The pathogen also demonstrates significant tropism for wounds, causing surgical site infections, burn wound colonization, and even necrotizing soft tissue infections. In neurosurgical patients, it can lead to devastating meningitis and ventriculitis. While less common, urinary tract infections associated with catheter use do occur. What makes *A. baumannii* particularly concerning is its remarkable ability to develop resistance to nearly all antibiotic classes, earning it designation among the ESKAPE pathogens. Its capacity to persist on environmental surfaces and medical equipment facilitates nosocomial transmission, while emerging reports of community-acquired cases suggest evolving epidemiological patterns that warrant close surveillance. The combination of clinical versatility, environmental resilience, and rapid antimicrobial resistance development makes this organism a critical challenge for modern infection control and antimicrobial stewardship programs.⁽¹⁴⁾

Hospital-Infections:

In healthcare environments, *Acinetobacter baumannii* poses a significant threat to critically ill patients, with infection risk escalating in proportion to the severity of a patient's condition and

exposure to medical interventions. The pathogen demonstrates a predilection for individuals with extended hospitalizations, where prolonged exposure to hospital flora and selective antibiotic pressure create ideal conditions for colonization and subsequent infection. Immunocompromised patients—including those with hematologic malignancies, transplant recipients, or individuals receiving immunosuppressive therapies—face particularly high vulnerability due to impaired host defenses. Advanced age and multiple chronic comorbidities (such as COPD, diabetes, or renal failure) further compound this risk by diminishing physiological reserve. The organism frequently targets trauma and burn victims, exploiting compromised skin barriers and wound environments. Prior antibiotic exposure, especially to broad-spectrum agents, promotes selection of resistant strains, while invasive procedures (including surgery, intubation, or central line placement) provide direct portals of entry. Of particular concern is the pathogen's ability to colonize and persist on indwelling medical devices—including endotracheal tubes, urinary catheters, and vascular access devices—which serve as both reservoirs for infection and conduits for systemic dissemination. This constellation of risk factors underscores the importance of rigorous infection prevention measures in high-acuity care areas, where the convergence of vulnerable patients, invasive technologies, and antibiotic selection pressure creates a perfect storm for *A. baumannii* transmission and infection. ⁽¹⁵⁾

Mortality:

Determining precise mortality rates is challenging because these infections typically occur in patients already facing life-threatening conditions ⁽¹⁶⁾. However, reported crude mortality rates range from a concerning 23% to a staggering 68% ⁽¹⁷⁾, highlighting the significant threat posed by this pathogen. ⁽¹⁸⁾

Mortality rates for community-acquired cases can reach as high as 64%, though it remains unclear whether host factors (e.g., comorbidities) or bacterial virulence traits primarily drive the more aggressive disease presentation compared to hospital-associated infections. ⁽¹⁹⁾

Signs and Symptoms of infection:

As an opportunistic pathogen, *A. baumannii* can cause a spectrum of infections, each with distinct symptomatology:

- **Pneumonia:** Dyspnea, productive cough (often with purulent or bloody sputum), and chest pain.
- **Bloodstream infections (bacteremia):** Fever, chills, and altered mental status, frequently complicating severe pneumonia or wound infections.
- **Meningitis:** Photophobia, neck stiffness, and confusion, particularly in post-neurosurgical or trauma patients.
- **Wound/surgical site infections:** Including necrotizing fasciitis, presenting with severe pain, erythema, and systemic toxicity.
- **Urinary tract infections (UTIs):** Dysuria, urinary urgency, and frequency, though less common than other manifestations.

Systemic symptoms such as fever, nausea, and myalgia may accompany localized infections. Notably, *A. baumannii* can asymptomatically colonize wounds, tracheostomy sites, or the respiratory tract ⁽²¹⁾, complicating differentiation between infection and colonization in clinical settings.

Acinetobacter species are Gram-negative coccobacilli bacteria characterized by their non-motile nature, catalase-positive and oxidase-negative biochemical profile, and tendency to appear in pairs under microscopic examination ⁽²²⁾. While these bacteria commonly colonize human skin and respiratory tract as harmless commensal organisms, they can transform into dangerous opportunistic pathogens in immunocompromised individuals. *Acinetobacter* causes a wide spectrum of infections, with clinical manifestations ranging from pneumonia (particularly hospital-acquired), bloodstream infections (bacteremia and septicemia), and meningitis to wound infections, endocarditis, and urinary tract infections ⁽²³⁾. This remarkable

transition from benign colonizer to aggressive pathogen makes *Acinetobacter* a particularly challenging microorganism in clinical settings, especially among vulnerable patient populations.

Hospitalization, mechanical ventilation, respiratory distress, inadequate treatment, previous infections, antibiotic use, and catheterization are common causes of *Acinetobacter* species infections⁽²⁴⁾. Significant evidence of increased colonization rates in the throat, respiratory, and digestive tracts has been presented by a number of previous outbreaks^(25,26). Many routinely prescribed antibiotic drugs, such as aminoglycosides, fluoroquinolones, and broad-spectrum β -lactams, are no longer effective against *Acinetobacter* species. Most strains are resistant to cephalosporins, and the trend of resistance to carbapenems is increasing⁽²⁷⁾.

Acinetobacter species have been found to vary in their susceptibility to antibiotics; of the strains studied, *Acinetobacter baumannii* has the highest level of resistance^(28,29). *Acinetobacter* is a genus of non-fermentative, nonmotile, gram-negative coccobacilli that includes numerous unnamed tentative species and 27 known species. The most clinically significant of them is *Acinetobacter baumannii* (Ab), which is frequently found to be the main source of infection in hospital environments. Due to its growing importance and prevalence, this opportunistic virus poses a serious threat as it can account for up to 20% of infections in intensive care units globally^(30,31).

Acinetobacter baumannii infections are not limited to hospital settings; reports have described cases that have affected healthy individuals of all ages in community settings, especially in the wake of natural catastrophes and during wars. The pathogen is mostly linked to urinary tract infections, endocarditis, meningitis, burn and surgical wound infections, bloodstream infections, pneumonia linked to ventilators, and infections of the skin and soft tissues^(32, 33). *Acinetobacter baumannii*, which is resistant to multiple drugs, is now considered one of the most dangerous infections linked to healthcare worldwide. Its extended environmental survival and capacity to colonize patients and the hospital setting make it easier for healthcare outbreaks to occur⁽³⁴⁾. Due mostly to genetic mechanisms connected to mobile genetic elements like insertion sequences, plasmids, and antibiotic resistance islands, *Acinetobacter baumannii* has evolved to resist a wide range of common antibiotics, including cephalosporins, aminoglycosides, quinolones, and carbapenems^(35,36).

Additionally, *A. baumannii* uses a variety of resistance methods, such as altering aminoglycosides, generating porin deficiencies in cell wall channels, increasing the expression of multidrug efflux pumps, acquiring β -lactamases to break down antibiotics, and switching target locations. These infections belong to the ESKAPE group, which is distinguished by its ability to elude the effects of conventional antimicrobial drugs. The ESKAPE group includes *Enterobacter* species, *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Staphylococcus aureus*, and *Klebsiella pneumoniae*^(37, 38).

Prominent risk factors for acquiring an *Acinetobacter* infection include prolonged hospital stays, admissions to intensive care units, surgical procedures, bloodstream infections, infections associated with medical devices, and immunosuppressive conditions like renal failure, diabetes, chronic lung diseases, and terminal illnesses like cancer⁽³⁹⁾. Because the pathogen is resistant to antibiotics and is associated with major medical disorders, it is critical to identify the culpable bacteria as soon as possible and determine their susceptibility to antimicrobials in healthcare settings. When broad-spectrum antibiotics are overused and misused, the condition usually gets worse and becomes more treatable diseases⁽⁴⁰⁾. The Infectious Diseases Society of America has classified *Acinetobacter baumannii* as a "red alert" pathogen because of its serious effects on public health. Its annual correlation with about 1.5 million infections highlights its worldwide significance⁽⁴¹⁾.

Material and Methods

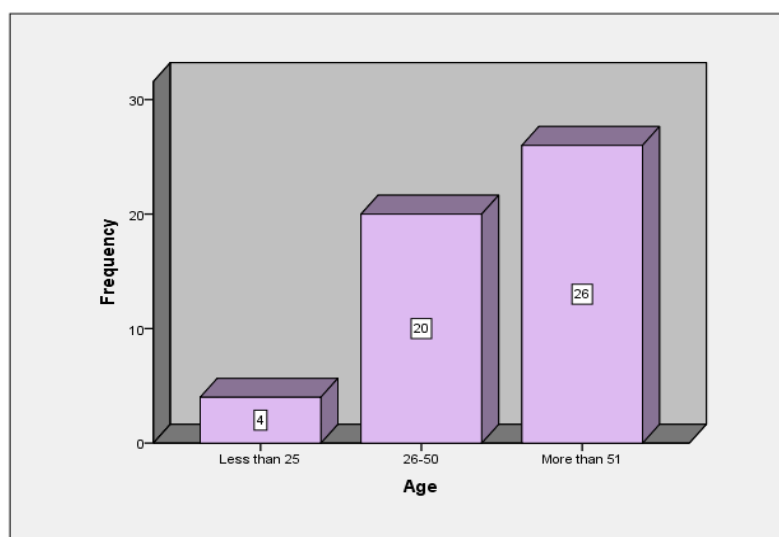
This cross-sectional descriptive study was carried out in a Pakistani tertiary care facility in Lahore over a period of 4 months. The sample was collected from a variety of departments,

including the intensive care units (ICUs), surgical wards, medical wards, of different hospital. Blood, urine, sputum, wound swabs, and other clinical specimens were aseptically taken from patients who were thought to have *Acinetobacter baumannii* infections. Samples were cultured on appropriate media (e.g., blood agar, MacConkey agar). Standard microbiological methods, such as Gram staining, colony morphology, and biochemical testing (such as the oxidase test), were used for the preliminary identification. The Kirby-Bauer disk diffusion method was used to test for antibiotic susceptibility in accordance with Clinical and Laboratory Standards Institute (CLSI) standards. Statistical software (e.g., SPSS) was used to analyze data. We summarized the data using descriptive statistics. Associations between categorical variables were evaluated using the chi-square test.

Results

TABLE 1: FREQUENCY OF AGE DISTRIBUTION

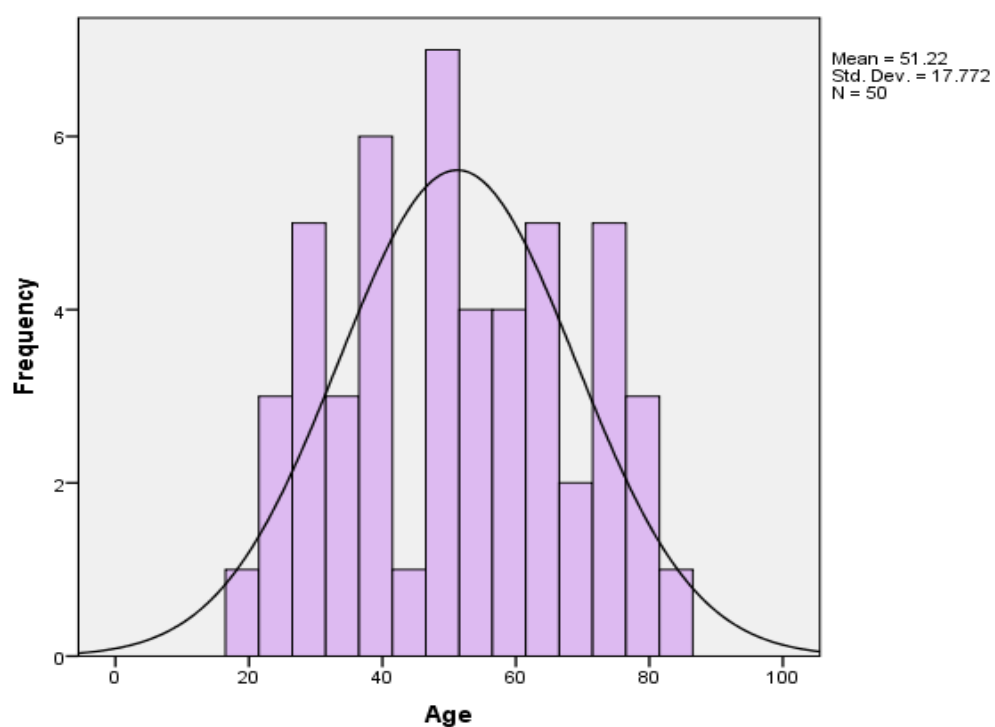
Age Categories	Frequency	Percentage (%)
Less than 25	4	8.0
26-50	20	40.0
More than 51	26	52.0
Total	50	100.0



GRAPH NO 1: FREQUENCY OF AGE CATEGORIES

TABLE 2: STATISTICS OF AGE DISTRIBUTION

Age of patients	N	Minimum	Maximum	Mean	Std. deviation
	50	19	84	51.22	17.772



GRAPH NO 2: AGE DISTRIBUTION OF STUDY POPULATION

TABLE 3: FREQUENCY OF GENDER DISTRIBUTION

Gender	Frequency	Percent
Male	23	46.0
Female	27	54.0
Total	50	100.0



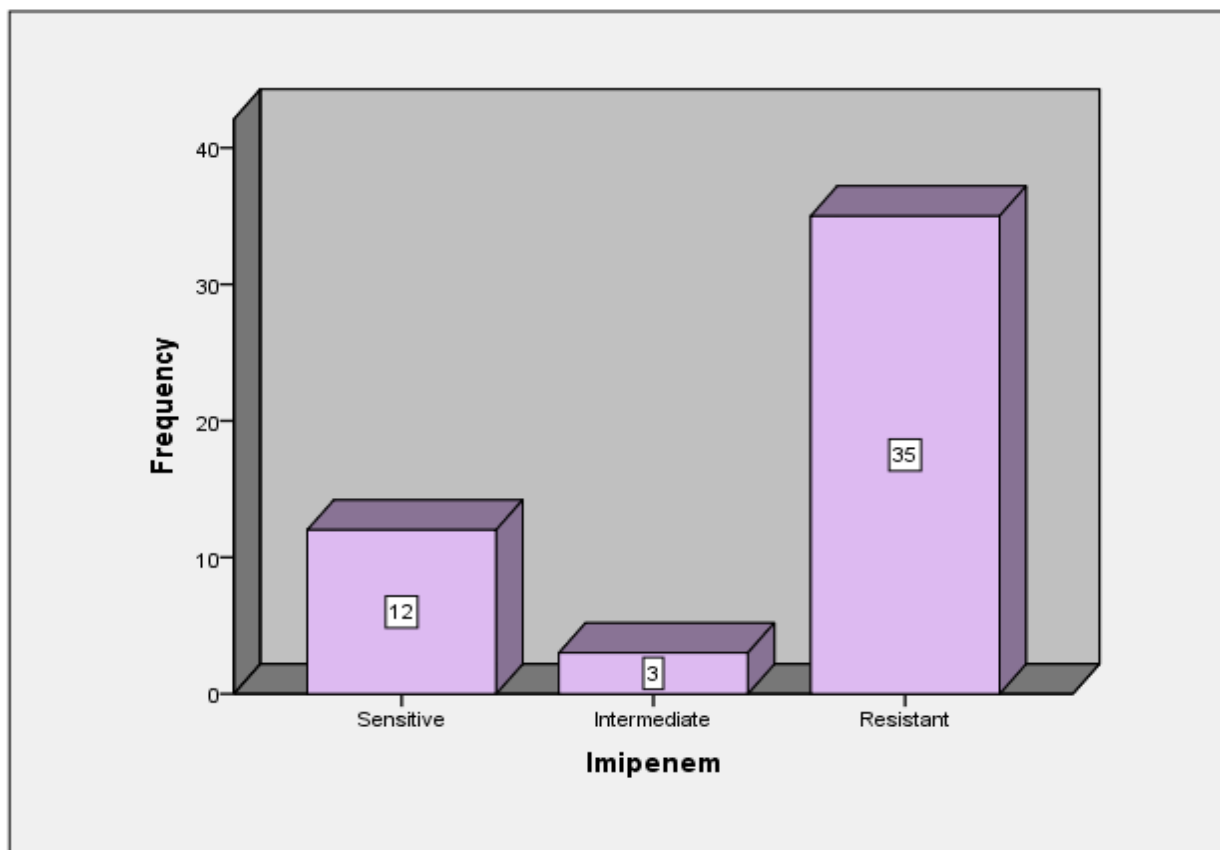
Graph No 3: Frequency of Gender Distribution of Study Population

Frequency Distribution of Imipenem Drug:

High resistance (70%) to imipenem (a carbapenem antibiotic) is alarming, as carbapenems are typically last-resort drugs for *A. baumannii*. Only 24% of isolates were susceptible, highlighting emerging carbapenem resistance.

TABLE 4: FREQUENCY OF IMIPENEM ANTIMICROBIAL SENSITIVITY PATTERN

Imipenem	Frequency	Percent
Sensitive	12	24.0
Intermediate	3	6.0
Resistant	35	70.0
Total	50	100.0



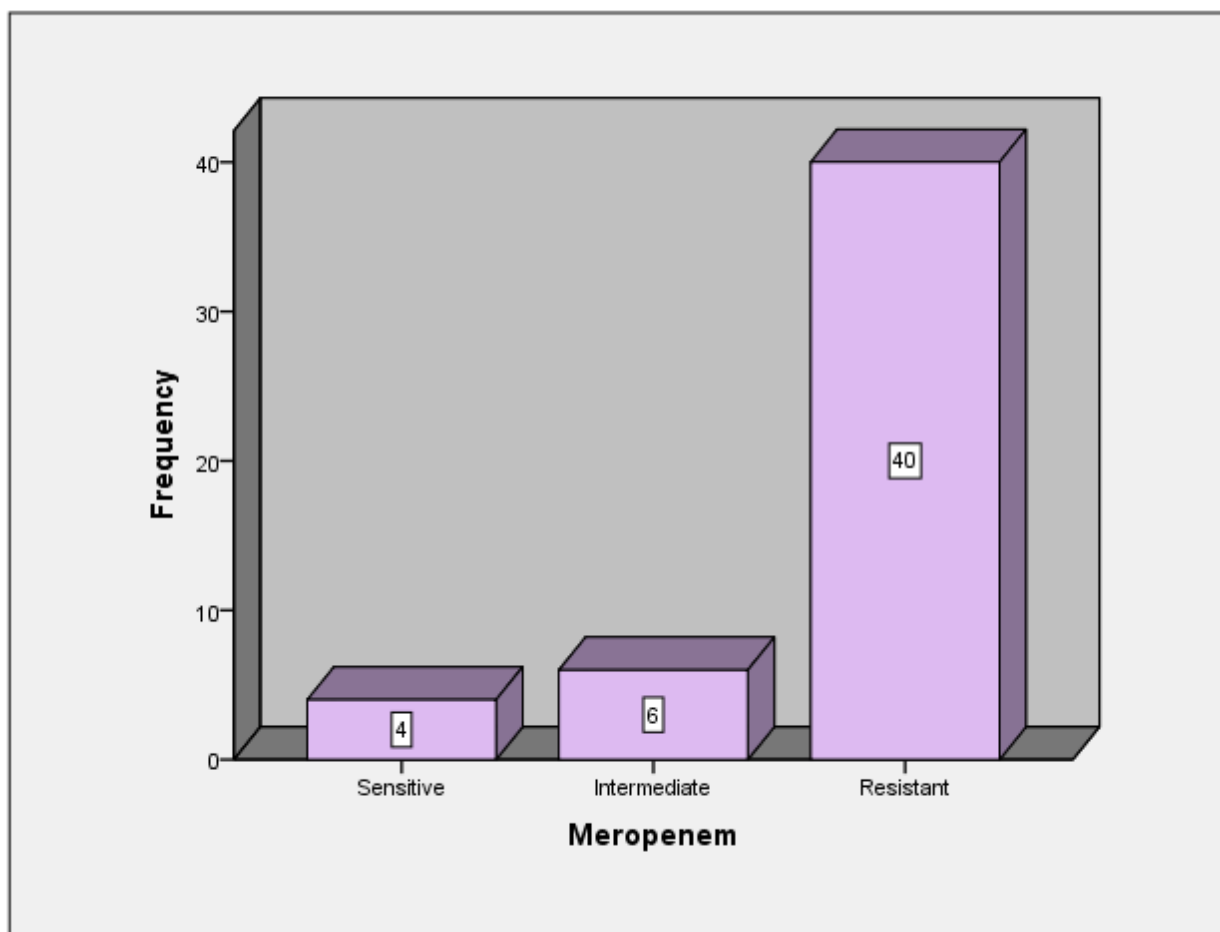
Graph No 4: Frequency of Imipenem Antimicrobial Sensitivity Pattern

Frequency Distribution of Meropenem Drug:

Even higher resistance (80%) to meropenem than imipenem, reinforcing the carbapenem resistance crisis. The low susceptibility (8%) suggests limited treatment options.

Table 5: Frequency of Meropenem Antimicrobial Sensitivity Pattern

Meropenem	Frequency	Percent
Sensitive	4	8.0
Intermediate	6	12.0
Resistant	40	80.0
Total	50	100.0



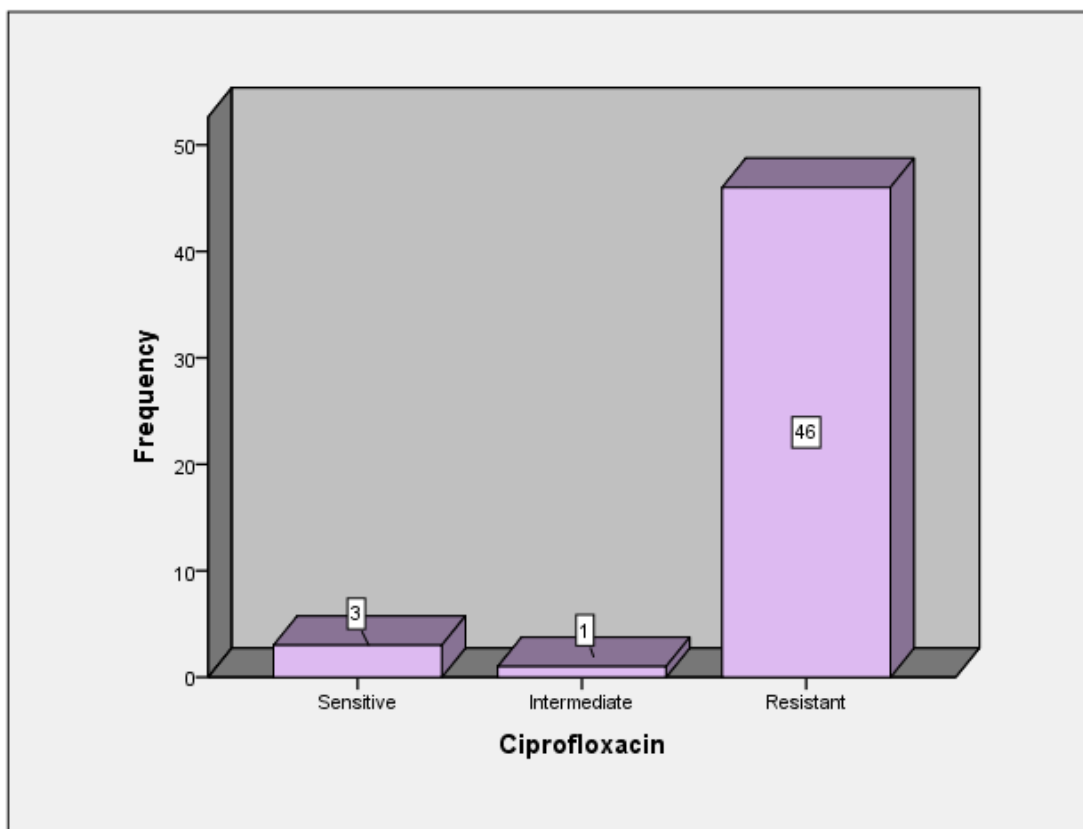
Graph no 5: frequency of meropenem antimicrobial sensitivity pattern

Frequency distribution of ciprofloxacin drug:

Extreme resistance (92%) to ciprofloxacin (a fluoroquinolone) renders this drug ineffective for *A. baumannii* infections in this setting.

Table 6: Frequency of Ciprofloxacin Antimicrobial Sensitivity Pattern

Ciprofloxacin	Frequency	Percent
Sensitive	3	6.0
Intermediate	1	2.0
Resistant	46	92.0
Total	50	100.0



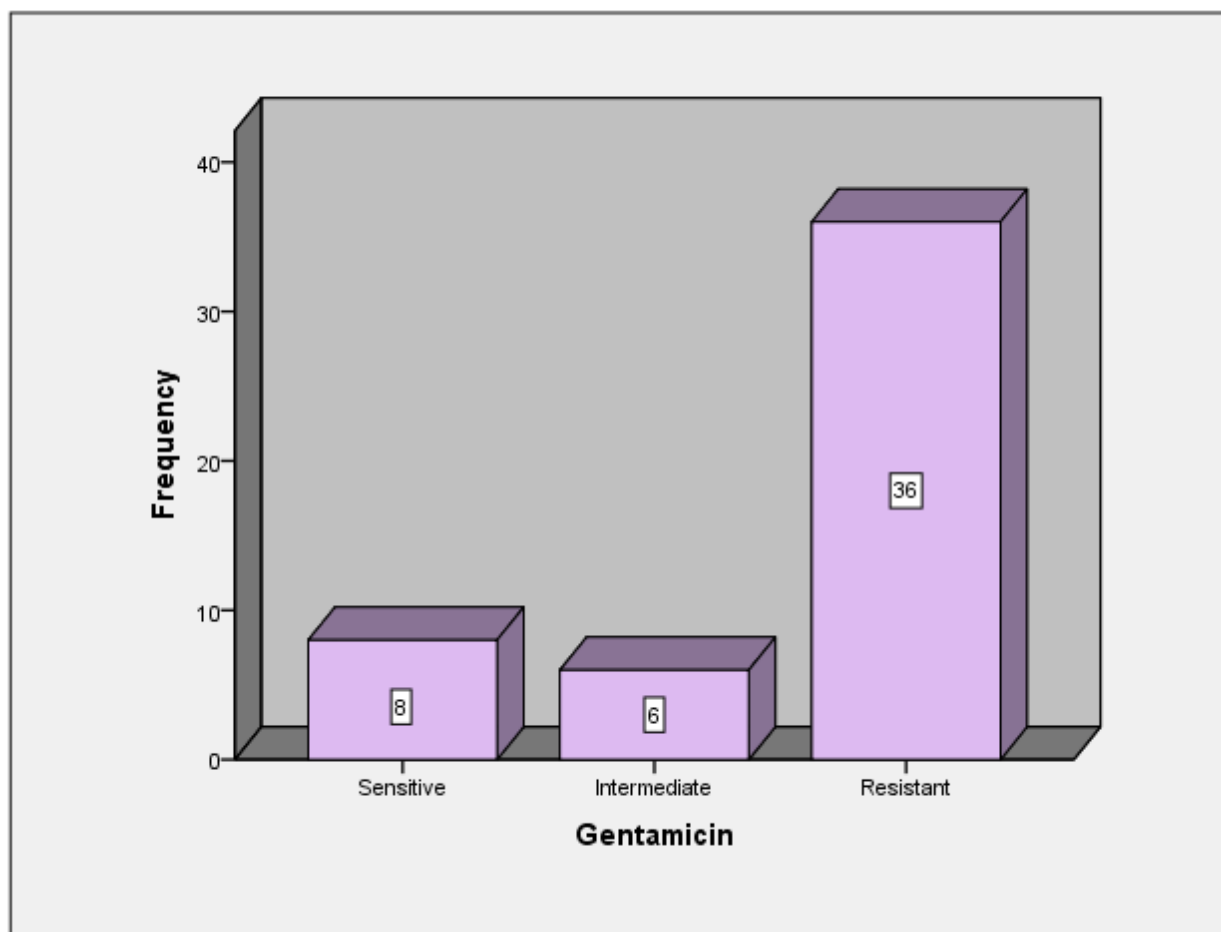
GRAPH NO 6: FREQUENCY OF CIPROFLOXACIN ANTIMICROBIAL SENSITIVITY PATTERN

FREQUENCY DISTRIBUTION OF GENTAMICIN DRUG:

High resistance (72%) to gentamicin (an aminoglycoside) limits its use. The intermediate category (12%) suggests some isolates may respond to higher doses.

TABLE 7: FREQUENCY OF GENTAMICIN ANTIMICROBIAL SENSITIVITY PATTERN

Gentamicin	Frequency	Percent
Sensitive	8	16.0
Intermediate	6	12.0
Resistant	36	72.0
Total	50	100.0



GRAPH NO 7: FREQUENCY OF GENTAMICIN ANTIMICROBIAL SENSITIVITY PATTERN

Discussion:

This study sheds light on the growing challenge of treating *Acinetobacter baumannii* infections, particularly in tertiary care hospitals where critically ill patients are most vulnerable. The results clearly reflect a troubling pattern of resistance to multiple commonly used antibiotics, making this pathogen a major concern in clinical settings.⁽⁴²⁾ A significant portion of the infected patients were over the age of 51, which supports existing knowledge that older individuals are more prone to severe infections. Factors such as weakened immunity, underlying chronic diseases, and prolonged hospital stays may explain why this age group is at greater risk. This emphasizes the importance of paying special attention to elderly patients when it comes to infection control and early treatment.⁽⁴³⁾

Although there was a slightly higher number of female patients in the study, statistical analysis showed no significant difference between males and females in terms of antibiotic resistance. This indicates that the development of resistance in *A. baumannii* is not influenced by gender, and both sexes are equally at risk.⁽⁴⁴⁾

The most striking and concerning finding was the very high resistance rates observed for the antibiotics tested. Ciprofloxacin, often used in clinical practice, showed a resistance rate of 92%, making it almost ineffective against these isolates. Similarly, resistance to gentamicin (72%) limits its usefulness as part of combination therapy.⁽⁴⁵⁾

The situation with carbapenems is particularly worrying. These drugs—imipenem and meropenem—are often reserved as last-resort treatments for serious infections. However,

our findings revealed resistance rates of 70% and 80% respectively. Such high levels of resistance to these critical antibiotics suggest that many cases of *A. baumannii* infection may no longer respond to standard therapies, leaving very few options for effective treatment.⁽⁴⁶⁾

These results are in line with other research done in the region, which has reported a steady rise in multidrug-resistant and even extensively drug-resistant strains of *A. baumannii*. The overuse and misuse of antibiotics, along with inadequate infection control practices, are likely driving these trends. Without urgent intervention, we risk entering an era where common infections become untreatable.⁽⁴⁷⁾

Our study also found that gender did not significantly influence antibiotic resistance, meaning that clinical focus should shift toward more relevant factors like age, prior antibiotic use, ICU admission, and underlying health conditions when designing treatment protocols or preventive strategies.⁽⁴⁸⁾

The data gathered in this research strongly supports the need for robust antimicrobial stewardship programs in hospitals. These programs must promote the responsible use of antibiotics, ensure regular surveillance of resistance trends, and strengthen infection control measures. Furthermore, investment in the development of new antibiotics and alternative therapies is crucial to outpace the evolution of resistant pathogens.⁽⁵⁵⁾

In summary, this study highlights the pressing need for a more strategic and responsible approach to antibiotic use and infection prevention. The rise in drug-resistant *A. baumannii* is not just a local issue—it reflects a broader global health threat that demands immediate action.

Conclusion

This research highlights the serious and growing issue of antibiotic resistance in *Acinetobacter baumannii* infections within a tertiary care hospital in Lahore, Pakistan. The findings paint a concerning picture—this bacterium, once manageable, is now showing high levels of resistance to commonly used antibiotics, including last-resort options like carbapenems.

Among the key observations was the fact that older adults, particularly those over 51, were most affected by these infections. This reinforces the importance of focusing preventive strategies on the elderly and other vulnerable patient groups. Although there was a slightly higher number of female cases, gender did not significantly affect resistance trends, suggesting that resistance is more closely linked to clinical and environmental factors than to demographics.

The resistance rates found in this study—especially the 92% resistance to ciprofloxacin, 80% to meropenem, and 70% to imipenem—are deeply troubling. These numbers reflect a broader pattern of multidrug resistance that is becoming increasingly common in healthcare settings, not just in Pakistan but globally.

These findings stress the urgent need for stronger antibiotic stewardship, improved infection control practices, and continuous monitoring of resistance patterns. Doctors and healthcare providers need accurate, up-to-date data to guide their treatment decisions and avoid unnecessary or ineffective antibiotic use.

In conclusion, *Acinetobacter baumannii* is no longer just a hospital nuisance—it's a serious threat to patient care. If left unchecked, the growing resistance it displays will limit our treatment options and place countless lives at risk. It is now more important than ever to work collectively—healthcare professionals, researchers, policymakers, and the public—to address this challenge with proactive and responsible measures.

Reference:

- Lin, Ming-Feng; Lan, Chung-Yu (2014). "Antimicrobial Resistance in *Acinetobacter baumannii*: From Bench to Bedside". *World Journal of Clinical Cases*. 2 (12): 787–814

- Antunes, Luísa C.S.; Visca, Paolo; Towner, Kevin J. (August 2014). "Acinetobacterbaumannii: evolution of a global pathogen". *Pathogens and Disease*. **71** (3): 292–301.
- Yeom, Jinki; Shin, Ji-Hyun; Yang, Ji-Young; Kim, Jungmin; Hwang, Geum-Sook; Bundy, Jacob Guy (6 March 2013).
- McQueary, Christin N.; Kirkup, Benjamin C.; Si, Yuanzheng; Barlow, Miriam; Actis, Luis A.; Craft, David W.; Zurawski, Daniel V. (30 June 2012). *Journal of Microbiology*. 434–443.
- Garrity, G., ed. (2000). "Pts. A & B: The Proteobacteria". *Bergey's Manual of Systematic Bacteriology*. Vol. 2 (2nd ed.). New York: Springer. p. 454.
- O'Shea, MK (May 2012). "Acinetobacter in modern warfare". *International Journal of Antimicrobial Agents*. **39** (5): 363–75.
- Gerner-Smidt, P (October 1992). *Journal of Clinical Microbiology*. 2680–5.
- Rice, LB (15 April 2008). *The Journal of Infectious Diseases*. 1079–81.
- Nordmann P., Poirel L. Epidemiology and Diagnostics of Carbapenem Resistance in Gram-negative Bacteria. *Clin. Infect. Dis.* 2019;69:S521–S528.
- Piperaki E.T., Tzouvelekis L.S., Miriagou V., Daikos G.L. Carbapenem-resistant *Acinetobacterbaumannii*: In pursuit of an effective treatment. *Clin. Microbiol. Infect.* 2019;25:951–957.
- Garnacho-Montero J., Timsit J.F. Managing *Acinetobacterbaumannii* infections. *Curr. Opin. Infect. Dis.* 2019;32:69–76.
- Karakonstantis S., Kritsotakis E.I., Gikas A. Treatment options for *K. pneumoniae*, *P. aeruginosa* and *A. baumannii* co-resistant to carbapenems, aminoglycosides, polymyxins and tigecycline: An approach based on the mechanisms of to carbapenems. *Infection*. 2020;48:835–851.resistance
- Mulani M.S., Kamble E.E., Kumkar S.N., Tawre M.S., Pardesi K.R. Emerging strategies to combat ESKAPE pathogens in the era of antimicrobial resistance: A review. *Front. Microbiol.* 2019;10:539.
- Dexter C., Murray G. L., Paulsen I. T., Peleg A. Y. (2015).13, 567–573.
- Karageorgopoulos D. E., Falagas M. E. (2008). 8, 751–762
- Freire M. P., De Oliveira Garcia D., Garcia C. P., CampagnariBueno M. F., Camargo C. H., KonoMagri A. S. G., et al. (2016) 22, 352–358.
- Falagas M., Karveli E., Kelesidis I., Kelesidis T. (2007). 26, 857–868.
- Dexter C., Murray G. L., Paulsen I. T., Peleg A. Y. (2015). 13, 567–573.
- Patamatamkul S., Klungboonkrong V., Praisarnti P., Jirakiat K. (2017). 87, 79–86.
- Everyday Health. 18 June 2015. Retrieved 18 April 2017
- Centers for Disease Control and Prevention. 6 November 2019. Retrieved 2023-03-09. Su, CH; Wang, JT; Chien, LJ; et al. (2012).
- W. Guidelines for the Collection, Transport, processing, Analysis, and Reporting of Cultures from Specific Specimens Sources. *Koneman's Color Atlas and Textbook of Diagnostic Microbiology*. 2006:67-110.
- Bergogne-Berezin E, Towner KJ. *Acinetobacter* spp. as nosocomial pathogens: microbiological, clinical, and epidemiological features. *Clinical microbiology reviews*. 1996 Apr;9(2):148-65.
- Husni RN, Goldstein LS, Arroliga AC, Hall GS, Fatica C, Stoller JK, Gordon SM. Risk factors for an outbreak of multi-drug-resistant *Acinetobacter* nosocomial pneumonia among intubated patients. *Chest*. 1999 May 1;115(5):1378-82.
- Van Looveren M, Goossens H, ARPAC Steering Group. Antimicrobial resistance of *Acinetobacter* spp. in Europe. *Clinical microbiology and infection*. 2004 Aug 1;10(8):684-704.
- Sohail M, Rashid A, Aslam B, Waseem M, Shahid M, Akram M, Khurshid M, Rasool MH. Antimicrobial susceptibility of *Acinetobacter* clinical isolates and emerging

- antibiogram trends for nosocomial infection management. *Revista da Sociedade Brasileira de Medicina Tropical*. 2016 May;49:300-4.
- Lyytikäinen O, Kõljalg S, Härmä M, Vuopio-Varkila J. Outbreak caused by two multi-resistant *Acinetobacter baumannii* clones in a burns unit: emergence of resistance to imipenem. *Journal of Hospital Infection*. 1995 Sep 1;31(1):41-54.
- Seifert HA, Baginski RA, Schulze AN, Pulverer GE. Antimicrobial susceptibility of *Acinetobacter* species. *Antimicrobial agents and chemotherapy*. 1993 Apr;37(4):750-3.
- Vila J, Marcos A, Marco F, Abdalla S, Vergara Y, Reig R, Gomez-Lus R, Jimenez de Anta T. In vitro antimicrobial production of beta-lactamases, aminoglycoside-modifying enzymes, and chloramphenicol acetyltransferase by and susceptibility of clinical isolates of *Acinetobacter baumannii*. *Antimicrobial agents and chemotherapy*. 1993 Jan;37(1):138-41.
- Khawaja A, Arshad F, Munir S. *Acinetobacter* species: Prevalence and sensitivity pattern of *Acinetobacter* species among clinical isolates of tertiary care hospital. *The Professional Medical Journal*. 2018 Dec 8;25(12):1949-53.
- Saleem AF, Ahmed I, Mir F, Ali SR, Zaidi AK. Pan-resistant *Acinetobacter* infection in neonates in Karachi, Pakistan. *The Journal of Infection in Developing Countries*. 2010;4(01):030-7.
- Khawaja A, Arshad F, Munir S. *Acinetobacter* species: Prevalence and sensitivity pattern of *Acinetobacter* species among clinical isolates of tertiary care hospital. *The Professional Medical Journal*. 2018 Dec 8;25(12):1949-53.
- Saleem AF, Ahmed I, Mir F, Ali SR, Zaidi AK. Pan-resistant *Acinetobacter* infection in neonates in Karachi, Pakistan. *The Journal of Infection in Developing Countries*. 2010;4(01):030-7.
- Bonnin RA, Rotimi VO, Al Hubail M, Gasiorowski E, Al Sweih N, Nordmann P, Poirel L. Wide dissemination of GES-type carbapenemases in *Acinetobacter baumannii* isolates in Kuwait. *Antimicrobial agents and chemotherapy*. 2013 Jan;57(1):183-8.
- Hood MI, Mortensen BL, Moore JL, Zhang Y, Kehl-Fie TE, Sugitani N, Chazin WJ, Caprioli RM, Skaar EP. Identification of an *Acinetobacter baumannii* zinc acquisition system that facilitates resistance to calprotectin-mediated zinc sequestration. *PLoS pathogens*. 2012 Dec 6;8(12):e1003068.
- Peleg AY, Seifert H, Paterson DL. *Acinetobacter baumannii*: emergence of a successful pathogen. *Clinical microbiology reviews*. 2008 Jul;21(3):538-82.
- Lee CR, Lee JH, Park M, Park KS, Bae IK, Kim YB, Cha CJ, Jeong BC, Lee SH. Biology of *Acinetobacter baumannii*: pathogenesis, antibiotic resistance mechanisms, and prospective treatment options. *Frontiers in cellular and infection microbiology*. 2017 Mar 13;7:55.
- Mulani MS, Kamble EE, Kumkar SN, Tawre MS, Pardesi KR. Emerging strategies to combat ESKAPE pathogens in the era of antimicrobial resistance: a review. *Frontiers in microbiology*. 2019 Apr 1;10:403107.
- Wong D, Nielsen TB, Bonomo RA, Pantapalangkoor P, Luna B, Spellberg B. Clinical and pathophysiological overview of *Acinetobacter* infections: a century of challenges. *Clinical microbiology reviews*. 2017 Jan;30(1):409-47.
- Akova M. Epidemiology of antimicrobial resistance in bloodstream infections. *Virulence*. 2016 Apr 2;7(3):252-66.
- Geisinger E, Mortman NJ, Dai Y, Cokol M, Syal S, Farinha A, Fisher DG, Tang AY, Lazinski DW, Wood S, Anthony J. Antibiotic susceptibility signatures identify potential antimicrobial targets in the *Acinetobacter baumannii* cell envelope. *Nature communications*. 2020 Sep 9;11(1):4522.
- Eliopoulos GM, Maragakis LL, Perl TM. *Acinetobacter baumannii*: epidemiology, antimicrobial resistance, and treatment options. *Clinical infectious diseases*. 2008 Apr 15;46(8):1254-63.

- Bertrand X, Dowzicky MJ. Antimicrobial susceptibility among gram-negative isolates collected from intensive care units in North America, Europe, the Asia-Pacific Rim, Latin America, the Middle East, and Africa between 2004 and 2009 as part of the Tigecycline Evaluation and Surveillance Trial. *Clinical therapeutics*. 2012 Jan 1;34(1):124-37.
- Bertrand X, Dowzicky MJ. Antimicrobial susceptibility among gram-negative isolates collected from intensive care units in North America, Europe, the Asia-Pacific Rim, Latin America, the Middle East, and Africa between 2004 and 2009 as part of the Tigecycline Evaluation and Surveillance Trial. *Clinical therapeutics*. 2012 Jan 1;34(1):124-37.
- Baadani AM, Thawadi SI, El-Khizzi NA, Omrani AS. Prevalence of colistin and tigecycline resistance in *Acinetobacterbaumannii* clinical isolates from 2 hospitals in Riyadh Region over a 2-year period. *Saudi Med J*. 2013 Mar 1;34(3):248-53.
- AssiriAM,Choudhry AJ, Alsaleh SS, Alanazi KH, Alsaleh SS. Evaluation of infection prevention and control programmes (IPC), and assessment tools for IPC-programmes at MOH-health facilities in Saudi Arabia. *Open Journal of Nursing*. 2014 May 23;2014.
- Aly M, Tayeb HT, Al Johani SM, Alyamani EJ, Aldughaisheem F, Alabdulkarim I, Balkhy HH. Genetic diversity of OXA-51-like genes among multidrug-resistant *Acinetobacterbaumannii* in Riyadh, Saudi Arabia. *European journal of clinical microbiology & infectious diseases*. 2014 Jul;33:1223-8.
- Saeed NK, KambalAM, El-Khizzi NA. Antimicrobial-resistant bacteria in a general intensive care unit in Saudi Arabia. *Saudi Med J*. 2010 Dec 1;31(12):1341-9.