

Antimicrobial resistance and genes profiles of *Acinetobacter baumannii* isolated from University Teaching Hospital of Kigali

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ABSTRACT

INTRODUCTION: Antimicrobial resistance is a growing global threat, with carbapenem-resistant *A. baumannii* (CRAB) identified by World Health Organisation (WHO) as a top-priority pathogen. In Rwanda, data on resistance patterns and underlying genetic determinants in *A. baumannii* remains limited. This study aims to assess phenotypic resistance profile and detect key resistance genes to support infection prevention and control (IPC) and antimicrobial stewardship interventions.

METHODS: A cross-sectional study was conducted from February to July 2025. *Acinetobacter baumannii* isolates were identified and tested for antimicrobial susceptibility using the disk diffusion method. Polymerase chain reaction (PCR) was used to detect selected resistance genes. Data on patient demographics, sample types, and hospital wards were collected and analysed.

RESULTS: Of 1746 clinical isolates, 3.2% (n=56) were confirmed *Acinetobacter baumannii*. Most isolates were recovered from male Intensive Care Unit (ICU) patients aged 19–40. All isolates showed 100% resistance to key beta-lactams, high resistance to ciprofloxacin with 87.0%, gentamicin 78.3%, while complete susceptibility to imipenem at 100% was observed. Resistance genes; blaOXA-23 and blaCTX-M were detected in 60.9% and 52.2% of isolates respectively, confirming the genetic basis for carbapenem and ESBL resistance.

CONCLUSION: The study revealed a significant burden of multidrug-resistant *Acinetobacter baumannii* infections, particularly among ICU patients. The high resistance of blaOXA-23 and blaCTX-M highlights the complexity of managing these infections and underscores the need for strengthened IPC measures, routine molecular surveillance, and targeted AMS strategies.

Keywords: Antimicrobial Resistance, *Acinetobacter baumannii*, Resistant Genes, Antibiotics

INTRODUCTION

Antimicrobial resistance (AMR) is a global health threat of the moment. It happens when bacteria,

fungi, viruses and parasites stop being killed or their growth is no longer slowed by drugs that used to work [1]. Carbapenem-resistant *Acinetobacter baumannii* has been classified by the WHO as a

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priority pathogen that requires new therapeutics and threatens to grow across the world [2].

It has been mentioned that ESBL-producing strains cause infections which vary in severity between simple urinary tract infections and life-threatening sepsis [3]. They have genes for resistance to many antimicrobial agents such as aminoglycosides, tetracyclines, chloramphenicol and fluoroquinolone. Thus, very broad antibiotic resistance extending to multiple antibiotic classes is now a frequent characteristic of ESBL-producing bacteria [4].

Studies across different African countries have highlighted the emergence of carbapenem-resistant *A. baumannii* (CRAB) in hospitals, particularly in neonatal and intensive care units (ICUs), where the infection burden is high [2].

In Rwanda, *Acinetobacter baumannii* is an emerging pathogen in healthcare settings, especially in ICUs. A study conducted in 2016 at the University Teaching Hospital of Kigali (CHUK) found a notable prevalence of *A. baumannii* among ICU patients, with several strains exhibiting resistance to commonly used antibiotics, including cephalosporins and aminoglycosides [5].

A 2019 study in Kigali identified an increasing prevalence of carbapenem-resistant *A. baumannii* strains, underscoring the need for enhanced infection control and antimicrobial stewardship [6].

In Africa, AMR cases are currently on the rise because of the following factors; excessive use of antibiotics, lack of proper structures like poor infection control measures and laboratory diagnostic capacity. The current study conducted in Nigeria, South Africa, and Kenya, which demonstrates a very high resistance level in *Acinetobacter* species predominantly carbapenem and extended spectrum cephalosporin [7].

A local study of Executive Summary of National Antibiotic Resistance Profiles carried out in Uganda showed carbapenem resistance rates of 40- 60 % in clinical isolates of *Acinetobacter baumannii* [8]. The issue of concern in the resistance of antimicrobials is well understood in Rwanda, but the data on *Acinetobacter* species are limited. CHUK preliminary findings show that *Acinetobacter* species is prevalent in ICU and is associated with increased length of stay. Yet, their resistance patterns and genetic aspects of their resistance are not well-documented. The present study attempts to address this disparity by looking at the patterns of antimicrobial resistance and

detection of the resistant genes in *Acinetobacter* species in University Teaching Hospital of Kigali (CHUK).

METHODS

Study Area

This study was conducted at CHUK, the largest hospital located in District of Nyarugenge at KN 4 Ave, Kigali City, Rwanda. It is also the biggest referral hospital of the country with a capacity of 483 beds. CHUK provides quality healthcare to the population, training, clinical research and technical support to district hospitals.

Study design

The current study was cross-sectional research in which clinical isolates of the University Teaching Hospital of Kigali (CHUK) were evaluated. This study was conducted from February to July 2025. The research used laboratory techniques to investigate the resistance profile and resistance gene pattern on *A. baumannii*. The study population were patients who had been sampled clinically of which samples such as blood culture, urine culture, sputum, or wound swab, as part of the routine diagnostic work up.

Study population

The study population comprises positive specimen of *Acinetobacter baumannii* on specimen received in the laboratory of any age and any sex of a patient attending Kigali University teaching hospital from February to July 2025. Our study period was used to determine the sample size in which 23 positive samples of *Acinetobacter baumannii* were obtained.

The approval to conduct this study was granted by Ines Ruhengeri Ethical and Research committee dated on 10th February 2025 & Ethic committee of CHUK dated on 28th May 2025 with approval Ref: EC/CHUK/081/2025 and data from patients were kept confidential and used only for this study purpose.

Sample collection and processing

Clinical samples were collected as part of routine diagnostic procedures from both inpatient and outpatient departments, following the standard operating procedures (SOPs) for specimen collection. Specimen types included blood, urine, sputum, and wound swabs. Each sample was appropriately labelled, transported to the

bacteriology laboratory under recommended conditions, and processed immediately. Upon receipt in the laboratory, specimens were evaluated for quality and then processed according to established guidelines for each sample type. Samples were inoculated on relevant culture media such as Blood Agar, MacConkey Agar, and Chocolate Agar, using standard aseptic techniques. The inoculated plates were incubated at 35–37°C for 18–24 hours, after which they were examined for significant bacterial growth. Presumptive bacterial colonies were subjected to further characterization using conventional biochemical tube tests, including oxidase, catalase, motility, citrate utilization, and carbohydrate fermentation tests, to accurately identify *Acinetobacter baumannii*. All isolates confirmed as *A. baumannii* were subjected to antimicrobial susceptibility testing and polymerase chain reaction to determine their resistance patterns. Quality control was conducted to validate the culture and sensitivity findings using *E. coli* ATCC 35218 while positive control (PC) and negative control (NC) were run on PCR to validate the PCR findings. All the control passed were. Statistical software (STATA version 17) was used for data manipulation and analysis.

Antibiotic Susceptibility Profiles

To determine the susceptibility profile of the *Acinetobacter baumannii* isolates, CLSI guidelines M100 version 2025 were used to select the panel of antibiotic disks to be tested. The following antibiotics were selected and tested: Ceftazidime, polymyxin B, Cefotaxime, Ciprofloxacin, Gentamicin, Imipenem, Amoxicillin and clavulanate, Amikacin, Ceftriaxone, Piperacillin-tazobactam, Trimethoprim-sulfamethoxazole. Antibiotic disks were put on muller Hinton agar (MHA) plate with 4 mm distance between each antibiotic, incubated at $\pm 35^{\circ}\text{C}$ for 16- 18 hours. The diameter zone of inhibition was measured using a ruler/ calliper to classify the isolates as sensitive, intermediate or resistance according to CLSI (clinical laboratory institute) guideline interpretive breakpoints.

DNA extraction and amplification

Isolates of *Acinetobacter* were stored in Skim milk medium containing Glycerol at $- 80^{\circ}\text{C}$ for further analysis (Molecular testing) at INES Ruhengeri molecular laboratory. Resistant *Acinetobacter* isolates were amplified and extracted at INES-Ruhengeri using boiling method and Colony PCR. The boiling method relies on high heat to physically

lyse the cells and release the genomic DNA and Colony PCR is a fast and simple method used to check for the presence of a target DNA and boiling method.

The Freshly grown, isolated colonies on a non-selective or selective agar plate were transferred into the tube containing the Sterile Nuclease-Free Water. Swirling the tip vigorously in the liquid and vortex briefly to ensure the bacterial material is fully suspended. The solution should appear slightly cloudy. The tubes were placed into the pre-heated heating block set to 95°C to 100°C . Incubated for 10 to 15 minutes. The high temperature ruptured the bacterial cell wall and membrane (thermal lysis), releasing the genomic DNA into the Sterile Nuclease-Free Water. After boiling, the tubes were transferred to an ice bath for 5 minutes, which aids in the precipitation of cellular debris. Centrifuged the tubes at maximum speed 12000 to 15000 X g for 5 to 10 minutes at room temperature. The clear liquid on top contained the crude bacterial DNA extract. Pipetted the supernatant into a new, clean, labelled microcentrifuge tube.

Detection of resistant genes

PCR was used to identify the presence of specific resistance genes that encode different enzymes, such as beta-lactamase and ESBL, which include blaOXA-23, blaCTX-M. Gene-specific primers for PCR derived from published sequences of the corresponding genes from fishes, and PCR performed using standard protocols.

Statistical analysis

After data collection, they were organized in excel sheet and POWER BI and STATA software version 17 were used for descriptive data analysis and visualization.

RESULTS

A total of 1,746 clinical isolates were processed during the study period, of which 56 (3.2%) were identified as *A. baumannii*. Among these, 23 isolates were randomly selected for molecular analysis.

Most *A. baumannii* isolates were obtained from male patients, with the majority aged between 19 and 40 years. The highest proportion of isolates originated from the Intensive Care Unit (ICU).

A total of 23 clinical samples positive for *Acinetobacter baumannii* were analysed. The majority of patients were male 15 (65.2%), with

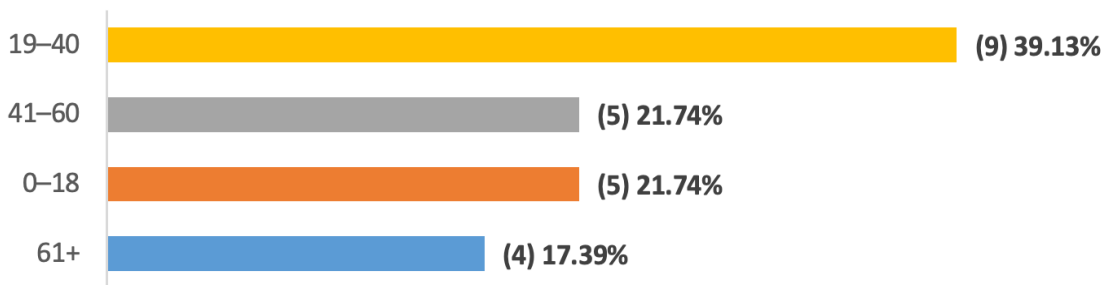


Figure 1: Age distribution of the study participants

females constituting 8 (34.8%). This gender distribution suggests a slightly higher burden or detection among male patients.

The age of patients ranged from 1 to 67 years, with a mean age of 35.7 years, indicating that *A. baumannii* infections affect a wide age range but mainly adults. Most isolates came from adults aged 19–40 (39.1%), followed by the paediatric (0–18) and middle-aged groups. This indicates that young adults are more frequently affected, potentially due to occupational or environmental exposure (Figure 1).

The different samples were collected: 78.26% (n=18) predominantly were expecto, 8.7% (n=2) and 8.7% (n=2) were wound and blood respectively while 4.3% (n=1) was urine (Figure 2).

The distribution of samples across hospital wards showed that the majority originated from the ICU, accounting for (n=18) 78.26% (95% CI: 55.79–91.13). A smaller proportion, (n=3) 13.04% (95% CI: 3.99–35.14), came from the pediatric ward, while only (n=2) 8.70% (95% CI: 2.01–30.65) were from the emergency department. This pattern indicates that samples were predominantly originated from the ICU setting (Figure 3). The antimicrobial

susceptibility testing using antibiotics from Basingstoke company located in United Kingdom for *Acinetobacter baumannii* isolates (N=23) at CHUK revealed a concerning pattern of extensive drug resistance. Interpretation was based on Clinical laboratory institute (CLSI M100 Ed-35) guidelines version 2025. Complete resistance (100%) was observed to several critical beta-lactam antibiotics, including ceftriaxone (CTR, N=23), Trimethoprim-Sulfamethoxazole (STX, N=23) and chloramphenicol (C, N=23), and ceftazidime (CAZ, N=23), indicating the likely widespread presence of β-lactamase enzymes that compromise the efficacy of these commonly used treatments. Similarly, cefotaxime (CTX, N=23) showed high resistance at 65.2%, with 34.8% of isolates not tested, further confirming significant β-lactam resistance.

Resistance to ciprofloxacin (CIP, N=23) was 87%, severely limiting fluoroquinolone treatment options, while gentamicin (GEN, N=23) exhibited 78.3% resistance, highlighting a reduced effectiveness of aminoglycosides. Encouragingly, amikacin (AK, N=23) retained better activity, with only 13% of isolates resistant and 69.6% susceptible, suggesting its potential utility as a therapeutic option (Table 1).

Carbapenem susceptible was notably high for imipenem (IMP, N=23), with all isolates susceptible

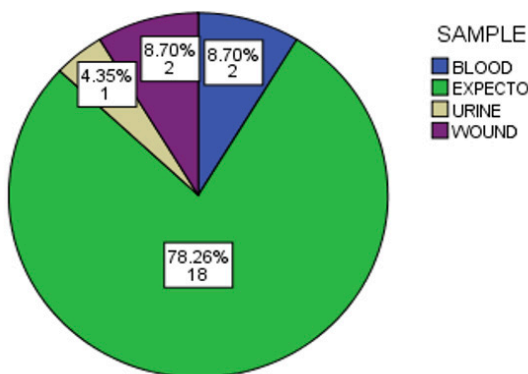


Figure 2: Types of collected patients' samples

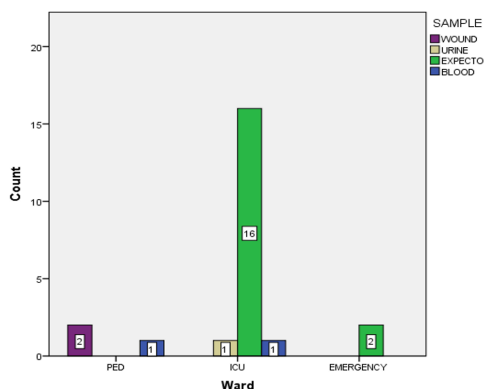


Figure 3: Sample distribution from ward

Table 1: Antibiotic and Resistance genes Rates among A. baumannii Isolates

Antibiotic & Gene	Disk Conc. (µg or IU)	R (Resistance)	S(Sensitive)	NT(Not tested)
AMC (Amoxicillin-Clavulanic Acid)	30 µg	22 (95.7%)	0 (0.0%)	1 (4.3%)
CIP (Ciprofloxacin)	5 µg	20 (87.0%)	3 (13.0%)	0 (0.0%)
CTX (Cefotaxime)	30 µg	15 (65.2%)	0 (0.0%)	8 (34.8%)
AK (Amikacin)	30 µg	3 (13.0%)	16 (69.6%)	4 (17.4%)
C (Chloramphenicol)	30 µg	23 (100.0%)	0 (0.0%)	0 (0.0%)
CTR (Ceftriaxone)	30 µg	23 (100.0%)	0 (0.0%)	0 (0.0%)
PB (Polymyxin B)	300 IU	0 (0.0%)	23 (100.0%)	0 (0.0%)
CAZ (Ceftazidime)	30 µg	23 (100.0%)	0 (0.0%)	0 (0.0%)
GEN (Gentamicin)	10 µg	18 (78.3%)	5 (21.7%)	0 (0.0%)
PTZ (Piperacillin-Tazobactam)	30 µg	0 (0.0%)	23 (100.0%)	0 (0.0%)
IMP (Imipenem)	10 µg	0 (0.0%)	23 (100.0%)	0 (0.0%)
STX (Trimethoprim-Sulfamethoxazole)	25 µg	23 (100.0%)	0 (0.0%)	0 (0.0%)
blaCTX	–	POSITIVE 13 (56.5%)	NEGATIVE 10 (43.5%)	0 (0.0%)
blaOXA23	–	POSITIVE 14 (60.9%)	NEGATIVE 9 (39.1%)	0 (0.0%)

(0% resistant), suggesting preserved efficacy in this small sample and piperacillin-tazobactam (PTZ, N=23) showed complete susceptibility (100%), indicating alternative options in combination therapies. Polymyxin B (PB, N=23) also remained fully effective (100% susceptible), confirming its role as a last-resort agent.

Analysis of resistance genes revealed that blaCTX was detected in 13 (56.5%) of isolates, while blaOXA23 was present in 14 (60.9%), highlighting the genetic basis of β -lactam and carbapenem resistance in this population.

These findings indicate an alarming level of multidrug resistance and presence of resistance genes among A. baumannii isolates at CHUK, with only amikacin and polymyxin B retaining meaningful activity, emphasizing the need for routine susceptibility testing and antimicrobial stewardship to guide effective therapy.

The Acinetobacter baumannii isolates across hospital wards shows a marked concentration in the Intensive Care Unit (ICU), which accounted for 18 of the 23 isolates (78.3%). This dominance reflects the known epidemiological pattern in which A. baumannii is more prevalent in critical care settings. In contrast, only 2 isolates (8.7%)

were detected in the Emergency Department, and 3 isolates (13.0%) were identified in the Paediatric Ward. The low numbers in these wards suggest a comparatively lower burden or transmission likelihood of A. baumannii outside the ICU.

In this study, the prevalence of the resistance genes blaOXA 23 and blaCTX M was found to be 14(60.9%) and 13 (56.5%), respectively, among the tested bacterial isolates. Carbapenem resistance is typically linked to the blaOXA 23 gene and has been found even more frequently (up to 94 to 100 percent) in clinical isolates of Acinetobacter baumannii in other parts of the world. The findings of this study indicate a substantial presence of resistance genes, although slightly lower than those observed in some high-burden clinical settings, underscoring the ongoing need for resistance monitoring and control strategies.

DISCUSSION

In this study, 23 clinical samples confirmed to be positive for Acinetobacter baumannii were examined. Of these, 15 (65.2%) were obtained from male patients, while 8 (34.8%) were from females. This indicates a slightly higher prevalence

or detection rate among men compared to women. Comparable findings have been documented elsewhere; for example, a surveillance study conducted in Saudi Arabia revealed that 67% of cases occurred in males and 33% in females, closely aligning with the results of the present study [9]. The predominance in males may be linked to multiple factors such as occupational exposure, differences in underlying health conditions, and varying risk behaviours between genders. In addition, disparities in healthcare-seeking practices might also play a role in the observed distribution. Nevertheless, since infections were recorded in both sexes, the results highlight that *A. baumannii* remains a pathogen of clinical importance across genders.

In Nigeria, 14 *Acinetobacter* cases were reported, with patient ages ranging from 2 to 95 years and an average age of 38.2 ± 12.4 years. The majority of infections occurred among individuals aged 31–40 years (30%), 51–60 years (30%), and those above 60 years (40%). These results closely mirror the present study, where the mean age was comparable (35.7 vs. 38.2), and adults aged 31–40 were the most affected group, with minimal cases in paediatric patients (<19 years) [10].

Comparable findings were reported from India, where a five-year hospital surveillance of 3,744 *Acinetobacter* isolates (98.5% *A. baumannii*) found a mean age of 34 years (range 1–96). The majority of infections were in adults aged 25–65 years (66%), followed by those 15–24 years (24%), while only 6% occurred in patients younger than 15 years. This supports the current study's observation that young and middle-aged adults (19–40 years) are the most commonly affected group, followed by children and older adults, consistent with the Indian trend of adult predominance [11].

Furthermore, an analysis of a larger dataset (>5,700 isolates) revealed that *A. baumannii* was more frequently isolated from patients younger than 61 years compared to those aged 61–75. Younger men accounted for the highest infection rates, with wound isolates more common among older adults (61–75 years), while blood isolates predominated in younger patients (<61 years). These findings align with the present study, where young adults were most affected, possibly due to environmental or occupational exposures. Data from Saudi Arabia also indicate that age-related trends may vary with specimen type or gender, although younger adults continue to represent the

majority of cases [9,11].

The antimicrobial susceptibility results highlight an alarming pattern of extensive drug resistance among *Acinetobacter baumannii* isolates at CHUK. Complete resistance (100%) to key beta-lactams, including cefotaxime, ceftriaxone, Ceftazidime, and chloramphenicol, strongly suggests widespread β -lactamase activity. Similarly, high resistance to ciprofloxacin (87%) and gentamicin (83.3%) further narrows available therapeutic options. However, amikacin showed relatively preserved activity, with resistance observed in only 15.8% of isolates, making it a potentially useful agent. This finding is consistent with a study from Haikou, China, which also reported high resistance to carbapenems and gentamicin, while amikacin and colistin retained better efficacy [12].

The distribution of samples by ward in this study shows a clear predominance from the Intensive Care Unit (ICU), with 18 out of 23 isolates (78.3%) originating from ICU patients, while only 2 (8.7%) and 3 (13.0%) were collected from the Emergency Department and Paediatric Ward, respectively. This pattern likely reflects the higher clinical acuity, longer hospital stays, and frequent invasive procedures in ICU settings, all of which increase vulnerability to infections and the likelihood of sample collection. Similar findings were reported by Patel [13], where 72% of isolates came from ICU patients, attributed to extended hospitalization and intensive monitoring requirements. Likewise [14] observed that ICU patients contributed the largest share of samples (65%), compared to lower proportions from Emergency (20%) and Paediatric wards (15%), underscoring the ICU as a focal point for nosocomial infection surveillance as also mentioned by . Compared with these studies, the present investigation shows a slightly higher ICU contribution (78.3%), which may indicate a greater emphasis on critical care monitoring or potential sampling bias toward ICU patients. However, the underrepresentation of Emergency and Paediatric cases could limit generalizability, highlighting the need for future research to adopt stratified or proportional sampling across wards to capture a more comprehensive picture of infection dynamics in different hospital units.

This study identified the resistance genes blaOXA-23 and blaCTX-M in 14 (60.9%) and 13 (56.5%) of the isolates, respectively. The blaOXA-23 gene is a well-known marker of carbapenem resistance and has been reported at even higher frequencies,

reaching 94–100% in *Acinetobacter baumannii* isolates from other regions [15]. Likewise, the blaCTX-M gene, responsible for extended-spectrum β -lactamase (ESBL) activity, shows wide global variation, with prevalence ranging from 13.6% in Tanzania to almost 99% in China [16], and between 39.6% and 100% in Gulf countries [17]. Although the prevalence detected in this study is somewhat lower than that documented in certain high-burden clinical settings, it still represents a significant reservoir of resistance genes, reinforcing the necessity for ongoing surveillance and strengthened antimicrobial stewardship efforts.

This study had a few notable limitations. First, it was conducted over a relatively short period, which may not fully reflect seasonal or annual variations in *A. baumannii* resistance patterns. Second, the molecular analysis was restricted to only two resistance genes because the primers available for this project were limited. As a result, other important carbapenemase and ESBL genes could not be evaluated. We therefore recommend larger studies with an expanded molecular panel to better characterize the full spectrum of resistance mechanisms in *A. baumannii*.

CONCLUSION

The present study revealed a significant burden of multidrug-resistant *Acinetobacter baumannii* infections at the University Teaching Hospital of Kigali (CHUK), particularly among patients admitted to the Intensive Care Unit. The isolates demonstrated high resistance rates to multiple antibiotics, including beta-lactams, carbapenems, and fluoroquinolones, which are commonly used in clinical care. Moreover, the molecular detection of resistance genes such as blaOXA-23, blaCTX-M confirms the genetic basis for this resistance and underscores the complexity of treating such infections. These findings not only reflect the local antimicrobial resistance (AMR) situation but also align with global trends, further emphasizing the urgency of strengthening local responses.

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