



Student's Journal of Health Research Africa

e-ISSN: 2709-9997, p-ISSN: 3006-1059

Vol.6 No. 12 (2025): December 2025 Issue

<https://doi.org/10.51168/sjhrafrica.v6i12.2465>

Original Article

Changing antibiogram profile of *Acinetobacter baumannii* in diabetic and non-diabetic foot ulcer infections: A comparative observational study.

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Abstract

Background:

Diabetic foot infections represent a major cause of hospitalization, limb loss, and antimicrobial exposure worldwide. Their microbiological landscape is increasingly dominated by multidrug-resistant gram-negative organisms. *Acinetobacter baumannii* has emerged as a clinically significant pathogen in chronic wounds and healthcare-associated infections, frequently exhibiting extensive resistance and narrowing therapeutic choices.

Objectives:

To compare the antimicrobial susceptibility profile and multidrug-resistant (MDR) burden of *Acinetobacter baumannii* isolated from infected foot ulcers in diabetic and non-diabetic patients.

Methods:

This comparative observational study was conducted in the Department of Microbiology, NIMRA Institute of Medical Sciences, Jupudi, Vijayawada, Andhra Pradesh, India, from May to October 2025. A total of 100 patients with clinically infected foot ulcers were enrolled, including 60 diabetics and 40 non-diabetics. Wound specimens were processed using standard culture techniques, and isolates were identified by conventional microbiological methods. Antimicrobial susceptibility testing was performed according to standard guidelines. MDR was defined as non-susceptibility to at least one agent in three or more antimicrobial classes.

Results:

The mean age of participants was 56.2 ± 10.8 years, and 68% were male. Diabetic patients had longer ulcer duration (median 18 vs 9 days). Resistance to ceftazidime and ceftriaxone was higher in diabetic than non-diabetic isolates (88.3% vs 70.0% and 83.3% vs 65.0%, respectively). Carbapenem resistance was 41.7% vs 25.0%, and amikacin resistance was 55.0% vs 37.5%. Colistin and tigecycline retained susceptibility above 90% in both groups. MDR prevalence was higher in diabetics (29/60, 48.3%) than in non-diabetics (11/40, 27.5%; Fisher's exact $p = 0.041$).

Conclusion:

Acinetobacter baumannii from diabetic foot ulcers exhibited a substantially higher resistance burden and MDR frequency. Colistin and tigecycline remained the most effective therapeutic options.

Recommendations:

Strengthen antimicrobial stewardship, implement routine local antibiogram surveillance, optimize wound care, and enforce strict infection control practices.

Keywords: *Acinetobacter baumannii*; diabetic foot infection; foot ulcer; antibiogram; multidrug resistance; colistin; tigecycline.

Submitted: November 01, 2025 **Accepted:** December 01, 2025 **Published:** December 30, 2025

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Introduction

Diabetes mellitus is a major driver of chronic wound burden, and foot ulceration remains one of its most disabling complications. Once an ulcer becomes infected, the risk of hospitalisation, limb-threatening tissue loss, and amputation rises sharply, and timely microbiology-guided therapy becomes central to care [1-3]. Guidelines emphasise careful clinical diagnosis, adequate debridement, and selection of empiric antimicrobials guided by local pathogen prevalence and resistance profiles, followed by targeted therapy after culture results are available [1-3].

In many regions, the bacteriology of infected foot ulcers has shifted towards Gram-negative organisms, particularly in hospital-exposed or chronic wounds. Indian data and other regional reports describe substantial Gram-negative predominance and considerable between-centre variability in susceptibility patterns, highlighting the importance of institution-specific antibiograms [4-6]. The challenge is compounded by frequent prior antibiotic exposure, delayed presentation, and repeated healthcare contact, all of which enrich resistant flora and complicate empiric choices [1-3].

Acinetobacter baumannii is an opportunistic pathogen increasingly recognised in wound and healthcare-associated infections. Its success is linked to environmental persistence, biofilm formation, and an exceptional capacity to acquire resistance determinants [7-9]. Carbapenem resistance in *A. baumannii* is of particular concern because it narrows effective options and is often mediated by acquired carbapenemases and other mechanisms, including oxacillinases and metallo-beta-lactamases [10,11]. Standardised terminology for multidrug resistance supports inter-study comparability and helps translate laboratory surveillance into clinical decision-making [12].

Therapeutic options for multidrug-resistant *A. baumannii* are limited, and in many settings, polymyxins (colistin) and tigecycline are used as key components of salvage or combination therapy, despite ongoing concerns regarding optimal dosing, toxicity, and the variability of clinical evidence across infection syndromes [13,14]. From a stewardship perspective, understanding which agents remain active against local *A. baumannii* isolates in specific clinical niches, such as foot ulcer infections, can reduce unnecessary broad-spectrum exposure and preserve last-line drugs.

Diabetic ulcers differ biologically from non-diabetic traumatic or pressure-related ulcers, with neuropathy, vasculopathy, and impaired innate immunity influencing microbial colonisation and progression to infection. These

factors, together with longer ulcer duration and repeated interventions, can favour the selection of resistant organisms and contribute to heterogeneous antibiograms across patient groups [1-3,7-9]. Comparing diabetic and non-diabetic foot ulcer infections within the same laboratory setting, therefore, offers a pragmatic approach to identify risk-stratified resistance patterns that can inform empiric therapy and local infection-control priorities.

Objectives: This study aimed to characterise and compare the antibiotic resistance patterns of *A. baumannii* isolated from infected foot ulcers among diabetic and non-diabetic patients at a tertiary care teaching hospital, and to quantify the proportion of multidrug-resistant isolates in each group.

Materials and Methods

Study design and setting

This was a hospital-based prospective comparative cross-sectional observational study conducted in the Department of Microbiology, NIMRA Institute of Medical Sciences, Jupudi, Vijayawada, Andhra Pradesh, India, over six months from May 2025 to October 2025. The study included consecutive adult patients presenting with clinically infected foot ulcers, and the antibiotic susceptibility profiles of *Acinetobacter baumannii* isolates were compared between diabetic and non-diabetic patients.

Study participants

Consecutive patients aged 18 years and above with clinical evidence of foot ulcer infection (purulent discharge and/or local inflammatory signs) were screened. Patients were assigned to diabetic or non-diabetic groups based on documented diabetes mellitus status in the medical record, consistent with contemporary approaches to diabetes-related foot infection assessment [1-3]. To ensure uniform interpretation of susceptibility patterns, the analysis included only those cases in which *Acinetobacter baumannii* was isolated as the sole pathogen from the wound culture. Patients with polymicrobial growth, contaminated samples, repeat cultures from the same lesion during the same episode, or incomplete clinical records were excluded.

Specimen collection and microbiological processing

After cleansing the ulcer with sterile normal saline and removing surface contaminants, deep tissue or pus was collected aseptically, preferably after gentle debridement. Specimens were transported promptly to the microbiology



laboratory in sterile containers. Samples were inoculated onto routine bacteriological media and incubated aerobically. Growth was evaluated by colony morphology and Gram staining. Identification of *Acinetobacter baumannii* was confirmed by standard biochemical tests and routine laboratory identification algorithms. Culture and susceptibility findings were reported to the treating teams as part of routine clinical care.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed using standardised laboratory methods and interpreted using contemporaneous breakpoints. The testing panel reflected commonly prescribed agents for Gram-negative wound infections and reserve agents used for multidrug-resistant organisms. It included third-generation cephalosporins (ceftazidime and ceftriaxone), carbapenems (imipenem or meropenem), an aminoglycoside (amikacin), a fluoroquinolone (ciprofloxacin or levofloxacin), and the reserve agents colistin and tigecycline. Internal quality control procedures were followed in accordance with routine laboratory practice.

Definitions

Multidrug resistance was defined using the international interim standard definition: non-susceptibility to at least one agent in three or more antimicrobial categories [12]. Resistance proportions were calculated separately for diabetic and non-diabetic isolates for each antimicrobial agent.

Data management and statistical analysis

Demographic, clinical, and laboratory variables were recorded in a structured proforma. Categorical variables were summarised as numbers (percentage), and continuous variables were described as mean \pm standard deviation or median, as appropriate. Antibiogram results were presented as resistance proportions by group, and the frequency of MDR isolates was summarised to support local empiric therapy decisions and stewardship activities.

Ethical considerations

Institutional permissions were obtained before initiation. Patient confidentiality was maintained by de-identifying data and restricting access to study records to investigators. Participation did not alter clinical management beyond routine specimen processing and reporting.

Results

Study population

A total of 100 patients with clinically infected foot ulcers were included. Sixty patients had diabetes mellitus, and 40 were non-diabetic. *Acinetobacter baumannii* was isolated as the single pathogen in all included cases.

Demographic and clinical profile

The demographic and clinical characteristics are summarised in Table 1. The mean age of the study population was 56.2 ± 10.8 years, and males constituted 68% of participants. The median duration of ulceration showed a longer course in the diabetic group (18 days) than in the non-diabetic group (9 days).

Table 1. Demographic and Clinical Characteristics of the Study Population (N = 100)

Variable	Total (N = 100)	Diabetic (n = 60)	Non-diabetic (n = 40)
Age (years), mean \pm SD	56.2 \pm 10.8	57.9 \pm 9.6	53.6 \pm 11.9
Male sex, n (%)	68 (68.0)	42 (70.0)	26 (65.0)
Female sex, n (%)	32 (32.0)	18 (30.0)	14 (35.0)
Duration of ulcer (days), median	14	18	9

Distribution of isolates

Of the 100 *Acinetobacter baumannii* isolates, 60 (60.0%) were obtained from diabetic foot ulcers and 40 (40.0%) from non-diabetic infected foot ulcers (Table 2).



Table 2. Distribution of *Acinetobacter baumannii* Isolates by Ulcer Type

Ulcer category	Number of isolates (n)	Percentage (%)
Diabetic foot ulcers	60	60.0
Non-diabetic foot ulcers	40	40.0
Total	100	100.0

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Antibiogram profile

Overall resistance was high across multiple antimicrobial classes (Table 3). Resistance to third-generation cephalosporins was higher among diabetic isolates (ceftazidime 88.3% and ceftriaxone 83.3%) than non-diabetic isolates (70.0% and 65.0%, respectively). Carbapenem resistance was observed in 41.7% of diabetic

isolates compared with 25.0% of non-diabetic isolates. Aminoglycoside activity was moderate, with amikacin resistance of 55.0% in diabetics and 37.5% in non-diabetics. Fluoroquinolone resistance (ciprofloxacin/levofloxacin) remained high in both groups and exceeded 70% among diabetics. Colistin and tigecycline demonstrated the highest activity, with resistance below 11% in both groups.

Table 3. Antibiotic Resistance Profile of *Acinetobacter baumannii* Isolates

Antimicrobial agent	Resistance in diabetics (n = 60), n (%)	Resistance in non-diabetics (n = 40), n (%)
Ceftazidime	53 (88.3)	28 (70.0)
Ceftriaxone	50 (83.3)	26 (65.0)
Imipenem / Meropenem	25 (41.7)	10 (25.0)
Amikacin	33 (55.0)	15 (37.5)
Ciprofloxacin / Levofloxacin	43 (71.7)	24 (60.0)
Colistin	4 (6.7)	3 (7.5)
Tigecycline	5 (8.3)	4 (10.0)

Multidrug resistance pattern

Using international standard definitions [12], multidrug-resistant isolates were more common among diabetic patients (48.3%) than non-diabetic patients (27.5%) (Table 4).

Table 4. Multidrug Resistance Pattern Among *Acinetobacter baumannii* Isolates

Resistance pattern	Diabetic (n = 60), n (%)	Non-diabetic (n = 40), n (%)	Total (N = 100), n (%)
MDR isolates	29 (48.3)	11 (27.5)	40 (40.0)
Non-MDR isolates	31 (51.7)	29 (72.5)	60 (60.0)

Discussion

This study demonstrated a substantial antimicrobial resistance burden in *Acinetobacter baumannii* isolated from infected foot ulcers and showed consistently higher resistance proportions and a greater multidrug-resistant (MDR) frequency among diabetic patients. Of the 100 isolates analysed, 60 were from diabetic foot ulcers, and 40 were from non-diabetic ulcers. Resistance to third-generation cephalosporins was markedly higher in diabetic isolates than in non-diabetic isolates, with ceftazidime resistance of 88.3% versus 70.0% and ceftriaxone resistance

of 83.3% versus 65.0%. Fluoroquinolone resistance was also high in both groups and remained greater among diabetics [71.7% vs 60.0%]. MDR isolates were more frequent in diabetic ulcers than in non-diabetic ulcers [48.3% vs 27.5%], supporting the finding that diabetic foot infections carried a heavier resistance burden in this setting [1-3].

Indian and other regional studies have shown that Gram-negative organisms commonly predominate in chronic diabetic ulcers and that susceptibility patterns vary across centres [4-6]. Within this context, *A. baumannii* has emerged as an important pathogen in wound and healthcare-



associated infections because of its environmental persistence, biofilm formation, and marked ability to acquire resistance determinants [7-9]. The high resistance observed in this study to cephalosporins, fluoroquinolones, and aminoglycosides, including amikacin resistance of 55.0% in diabetics and 37.5% in non-diabetics, is therefore in agreement with the broader resistance profile reported for this organism [7-9].

Several factors may explain the greater resistance burden in diabetic ulcers, including longer ulcer duration, repeated wound care exposure, recurrent debridement, and prior antimicrobial use, all of which favour selection of resistant flora and healthcare-associated acquisition [1-3,7-9]. In this study, diabetic patients had a longer median ulcer duration than non-diabetic patients [18 days vs 9 days], alongside a higher MDR proportion [48.3% vs 27.5%]. These findings suggest greater cumulative antimicrobial selection pressure in the diabetic group. Use of international MDR definitions improves comparability across studies and strengthens the clinical relevance of local surveillance data [12]. In settings where *A. baumannii* is prevalent, early specimen collection before antibiotic initiation, and rapid reporting of susceptibility results remain essential for reducing unnecessary broad-spectrum exposure and enabling de-escalation.

Carbapenem resistance deserves particular attention because carbapenems have long been considered key agents for severe Gram-negative infections. In *A. baumannii*, carbapenem resistance is mediated by several mechanisms, especially acquired carbapenemases, along with other alterations that reduce intracellular drug concentrations [10,11]. In this study, carbapenem resistance was considerably higher among diabetic isolates than among non-diabetic isolates [41.7% vs 25.0%]. This finding suggests that routine empiric carbapenem use in chronic or previously treated diabetic ulcers may not always provide adequate early coverage and further reinforces the need for culture-guided therapy.

In contrast, colistin and tigecycline retained the greatest *in vitro* activity in both groups. Resistance to colistin was low in diabetic and non-diabetic isolates [6.7% vs 7.5%], and tigecycline resistance was similarly low [8.3% vs 10.0%], indicating susceptibility above 90% for both agents. Earlier evidence supports their role as reserve drugs against MDR Gram-negative and *Acinetobacter* infections, although their use requires careful stewardship because toxicity profiles and clinical effectiveness vary by infection syndrome [13,14]. Overall, the findings of this study support continued local antibiogram surveillance and risk-stratified empiric

therapy pathways that recognise the higher resistance burden in diabetic ulcers. Strengthening antimicrobial stewardship, environmental cleaning, and hand hygiene is especially relevant for *A. baumannii* because of its capacity to persist on surfaces and spread within healthcare settings [7-9].

Generalizability

Findings are most applicable to tertiary care hospitals in similar regional settings with comparable antimicrobial prescribing patterns and healthcare exposure. Variability in local resistance ecology, laboratory practices, and patient referral profiles may limit extrapolation to primary care centres or geographically distinct populations.

Conclusion

Infected foot ulcers due to *Acinetobacter baumannii* showed extensive resistance to commonly used antimicrobial classes in this tertiary care setting. Isolates from diabetic ulcers carried a higher resistance burden than those from non-diabetic ulcers, including greater resistance to third-generation cephalosporins, fluoroquinolones, aminoglycosides, and carbapenems, and a higher proportion of multidrug-resistant strains. Colistin and tigecycline remained the most active agents *in vitro* across both patient groups. Routine unit-level antibiogram monitoring, early culture sampling, and stewardship-guided selection and de-escalation of antibiotics are essential to improve the probability of early appropriate therapy and to preserve reserve agents for clearly indicated cases. These measures also strengthen local infection control.

Limitations

This single-centre study reflects the microbiology and prescribing context of one tertiary care teaching hospital and therefore limits generalisability. Only monomicrobial *Acinetobacter baumannii* infections were analysed, so polymicrobial ulcers were not represented. Resistance mechanisms were not characterised by molecular methods, and minimum inhibitory concentration distributions were not recorded for all agents. Clinical outcomes such as healing time, amputation, and mortality were not evaluated.

Recommendations

Routine, unit-specific antibiogram surveillance should guide empiric therapy for infected foot ulcers, particularly among diabetic patients with prolonged ulcer duration or prior antibiotic exposure. Early deep-tissue sampling before



initiation of antimicrobials is essential to improve diagnostic yield and enable timely de-escalation. Empiric carbapenem use should be carefully evaluated in high-resistance settings, reserving last-line agents such as colistin and tigecycline for culture-confirmed multidrug-resistant isolates. Structured antimicrobial stewardship programmes, periodic resistance audits, and clinician feedback can reduce unnecessary broad-spectrum exposure. Strengthening wound care protocols, glycaemic control, environmental cleaning, and hand hygiene practices will further limit transmission of resistant *Acinetobacter* strains.

Acknowledgements

The authors acknowledge the technical staff of the Department of Microbiology for their assistance in specimen processing and susceptibility testing. We thank the clinicians and nursing personnel for coordinated patient care and documentation. Institutional administrative support in facilitating study conduct is gratefully appreciated.

Abbreviations

A. baumannii – *Acinetobacter baumannii*
MDR – Multidrug-resistant
DFI – Diabetic foot infection
AST – Antimicrobial susceptibility testing
IDSA – Infectious Diseases Society of America
IWGDF – International Working Group on the Diabetic Foot
MIC – Minimum inhibitory concentration
SD – Standard deviation

Source of funding

The study had no funding.

Conflict of interest

The authors declare no conflict of interest.

Author Contributions

SK-Concept and design of the study, results interpretation, review of literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript. **KM**- Design of the study, results interpretation, review of literature, preparation of the first draft of the manuscript, and revision of the manuscript. **JB**- Review of literature and preparing the first draft of the manuscript. Statistical analysis and interpretation. **BMK**-preparing first draft of manuscript. Statistical analysis and interpretation.

Data availability:

Data is available

Author Biography

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Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.6 No. 12 (2025): December 2025 Issue
<https://doi.org/10.51168/sjhrafrica.v6i12.2465>
Original Article

PUBLISHER DETAILS:

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Student's Journal of Health Research (SJHR)

(ISSN 2709-9997) Online

(ISSN 3006-1059) Print

Category: Non-Governmental & Non-profit Organization

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Location: Scholar's Summit Nakigalala, P. O. Box 701432,
Entebbe Uganda, East Africa

