Огляд

Research

НИРКИ KIDNEYS

DOI: 10.65327/kidneys.v14i4.571

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Polymicrobial Infections and Rising Resistance in Diabetic Foot Ulcers: Clinical Implications for Antibiotic Management

For citation: Kidneys. 2025;14(4):01-08. Acceptance- 30/10/2025 Received- 15/10/2025
Doi: 10.65327/kidneys.v14i4.571

Abstract

Diabetes mellitus (DM) is a chronic metabolic disorder with significant complications including diabetic foot ulcers (DFUs). These ulcers often infected by a diverse range of bacterial pathogens represent a major challenge due to the rising prevalence of antimicrobial resistance (AMR). Foot infection in diabetic patients with chronic kidney disease (CKD) or diabetic kidney disease (DKD) is worse owing to uremic immune dysfunction, retarded wound healing, poor microvascular circulation, as well as changed antibiotic metabolism. Patients with dialysis-dependent issues and those of kidney-transplant are even at higher risk due to poor immunity and frequent contact with healthcare. It is important to the knowledge of the pathogen behavior and resistance patterns in this population to be informative in nephrology and renal-care management. This study investigated the antibiogram of bacterial pathogens isolated from male diabetic patients to understand resistance patterns and guide effective therapeutic strategies. Over two years, 102 male patients with DFUs from multispecialty hospitals in and around Madurai, Tamil Nadu, India, were examined. Deep swabs were collected and processed for bacterial isolation. Antibiotic susceptibility testing was conducted on the isolated bacteria. A total of 21 bacterial isolates were identified including 47.6% gram positive and 52.4% gram negative bacteria. Predominant isolates included *Staphylococcus aureus*, *Pseudomonas* sp. and *Klebsiella* sp. Antibiotic susceptibility testing revealed varied responses with cloxacillin and nitrofurantoin showing notable efficacy against gram positive bacteria while doxycycline and ciprofloxacin demonstrated the highest sensitivity against gram negative isolates. However, multidrug resistance was prevalent particularly in *Staphylococcus* sp., *Acinetobacter* sp. and *Escherichia coli*. The study highlights a significant prevalence of advanced stage ulcers delays in care seeking and polymicrobial infections complicating treatment. The findings underscore the necessity for targeted antibiotic therapy informed by routine antibiogram surveillance and the urgent implementation of antibiotic stewardship programs. Understanding pathogen resistance trends is crucial for optimizing treatment outcomes and mitigating the burden of antimicrobial resistance in diabetic populations. These findings have direct implications for kidney disease management, where antimicrobial resistance limits renal-safe antibiotic options for CKD, dialysis, and post-transplant patients.

Keywords: Antimicrobial resistance, Antibiotic susceptibility, Bacterial Pathogens, Diabetic Foot Ulcers, Polymicrobial infections, Chronic Kidney Disease, Diabetic Kidney Disease, renal Impairment, Hemodialysis, Kidney Transplantation.

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder marked by persistent hyperglycaemia resulting from impaired insulin secretion or action [1]. It affects millions worldwide and contributes substantially to morbidity and mortality. Among its major

complications, diabetic foot ulcers (DFUs) are particularly debilitating, affecting about 15% of patients during their lifetime and remaining a leading cause of non-traumatic lower-limb amputations [2]. Their management is often complicated by bacterial infections that can progress to osteomyelitis, sepsis, or limb loss if untreated. Therefore, understanding the antibiogram of DFU-associated pathogens is critical for effective therapy and preventing severe complications [3]. Diabetic kidney disease (DKD) is one of the most common and serious complications of long-standing diabetes. Patients with CKD experience impaired immune responses, reduced microvascular supply, and systemic inflammation, all of which contribute to increased susceptibility to foot infections. The uremic environment further delays wound healing, making DFUs more severe and harder to treat in this population [4].

The microbial profile of diabetic foot ulcers (DFUs) is highly diverse, involving both gram-positive and gram-negative bacteria, often in polymicrobial combinations [5]. Common pathogens include *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae* and *Escherichia coli [6]*. The rise of methicillin-resistant *S. aureus* (MRSA) and multidrug-resistant gram-negative organisms has made empirical antibiotic therapy increasingly unreliable, reinforcing the need for targeted treatment based on susceptibility testing [7]. Periodic antibiogram surveillance is therefore essential for guiding clinical decision-making. DFU infections are further influenced by peripheral neuropathy, ischemia and diabetes-related immune dysfunction, which promote bacterial colonization and complicate treatment. Resistance patterns vary among pathogens—for example, *S. aureus* frequently resists β-lactams, while *P. aeruginosa* often shows resistance to aminoglycosides and fluoroquinolones—highlighting the importance of culture-based therapy in managing DFUs effectively[8]. In addition to diabetes-related immunosuppression, reduced renal function introduces additional challenges. CKD and dialysis patients frequently encounter multidrug-resistant organisms because of repeated hospital exposure, chronic inflammation, and accumulation of uremic toxins. These factors make DFU infections in CKD patients more complex, with higher risks of systemic spread, prolonged hospitalization, and limb amputation [9].

Several studies have highlighted the unique challenges associated with DFU infections in men with diabetes. Men are reported to have a higher prevalence of diabetic foot complications than women potentially due to differences in foot biomechanics, footwear habits and hormonal factors. Additionally, delayed presentation and poor glycaemic control in male patients further contribute to the severity of infections and their resistance profiles [10]. Studies such as those by Lipsky et al.,[11] and Nwankwo et al.,[12] have emphasized the critical role of culture based diagnostics and antibiograms in improving the management of DFUs in male patients.

Emerging research shows a rapid increase in multidrug-resistant pathogens in DFUs, reflecting the global escalation of antimicrobial resistance (AMR) [13]. The World Health Organization attributes this trend largely to the overuse and misuse of antibiotics [14]. In diabetic foot infections, prolonged empirical use of broad-spectrum agents without culture-based guidance worsens resistance, leading to treatment failures and higher amputation rates [15]. Addressing this challenge requires early microbiological diagnosis, strict antibiotic stewardship and improved therapeutic strategies [16]. This study therefore examines the antibiogram of bacterial pathogens isolated from DFUs in male DM patients to identify resistance patterns and guide rational antibiotic selection, contributing to efforts to combat AMR in diabetic foot infections [17]. For nephrologists, understanding DFU infection patterns is clinically important, as CKD patients often require renal-adjusted antibiotic dosing and face higher risks of nephrotoxic drug injury. Antibiotic selection becomes more restricted in CKD stages 3–5, dialysis populations, and kidney-transplant recipients due to immunosuppression and impaired drug clearance. Therefore, antibiogram insights are essential to guide safe and effective treatment strategies in renal-care settings [18].

Materials and Methods

Selection of diabetic mellitus (DM) patients and study duration

Patients with diabetic foot ulcers (DFUs) across various Wagner grades who presented to multispecialty diabetes hospitals in and around Madurai were enrolled as inpatients or outpatients. The study was conducted from January 2021 to September 2023. Only patients who had received antibiotics for more than 72 hours were included, with each participant enrolled once. Demographic data were recorded, and specimen collection procedures were designed to avoid superficial contamination by sampling only from clinically infected deep tissue.

Renal Function Assessment

Renal function parameters including serum creatinine, blood urea nitrogen (BUN), and estimated glomerular filtration rate (eGFR) were recorded from available hospital records. CKD staging (G1–G5) was done based on KDIGO 2022 guidelines. Dialysis status (hemodialysis or peritoneal dialysis) and kidney-transplant history were also documented where applicable. These data were used to interpret infection severity and antibiotic susceptibility in relation to renal impairment.

Collection and processing of swabs from DFU male patients

Deep-tissue samples from DFUs were collected using sterile swabs pre-soaked in glucose broth and immediately streaked onto blood agar, Mannitol salt agar and MacConkey agar plates. The plates were incubated at 37 °C for 24 hours and examined for

bacterial growth. Individual colonies were then subcultured onto fresh media to obtain pure isolates.

Identification and biochemical analysis of bacterial isolates

The isolated bacterial colonies were identified based on their physical and biochemical characteristics using the guidelines provided in Bergey's Manual of Determinative Bacteriology.

Antibiogram of bacterial pathogens in DFU

Antimicrobial susceptibility was determined using the disk diffusion method according to CLSI guidelines. Bacterial isolates were standardized to a 0.5 McFarland turbidity and inoculated onto Mueller–Hinton agar using sterile swabs. Antibiotic discs were applied, and the plates were incubated at 37 °C for 18–20 hours. Zones of inhibition were then measured and interpreted. Gram-positive isolates were tested against clindamycin, nitrofurantoin, gentamicin, cloxacillin, ampicillin, chloramphenicol, amoxiclav and erythromycin. Gram-negative isolates were evaluated using doxycycline, ofloxacin, cefotaxime, ciprofloxacin, co-trimoxazole, tobramycin, ceftazidime and streptomycin. All media, antibiotic discs and reagents were obtained from HiMedia Laboratories, Thane.

Results and Discussion

Figure 1 shows the gender distribution of the 171 patients, with males accounting for 58% (n = 99) and females 42% (n = 72). This indicates a higher proportion of male patients presenting with diabetic foot complications, a trend reported in previous studies. Men often experience greater prevalence and severity of DFUs, which may be related to differences in foot care practices, delayed healthcare seeking and poorer glycaemic control.

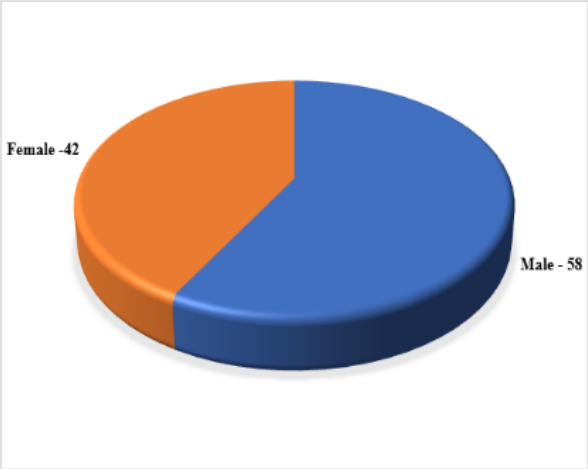


Figure 1. Percentage of DM patients based on gender

Figure 2 shows the age distribution of DM patients, with prevalence increasing with age and peaking in the 51–60 year group (35%), followed by 61–70 years (24.5%) and 41–50 years (20.5%). Younger adults (20–40 years) accounted for only a small proportion of cases. A decline was observed in the 71–80 year group (10%), with no cases reported above 81 years. This pattern reflects the well-established rise in diabetes risk with advancing age.

The higher prevalence of DM in middle-aged and older adults is consistent with evidence that diabetes risk increases with age due to insulin resistance, lifestyle changes and comorbidities such as hypertension and obesity. The reduced numbers in the oldest age groups may reflect survivorship bias, as patients with long-standing poorly controlled diabetes have higher mortality before reaching advanced age. Prevention efforts in these age groups typically emphasize lifestyle modification and regular screening, particularly for middle-aged adults who show the highest prevalence.

Figure 2. Percentage of DM male patients based on age group

Figure 3 shows that most DM patients belonged to low-income (50%) or lower-middle-income groups (41%), while only a small proportion were from upper-middle (4%) or high-income groups (5%). This pattern reflects the higher diabetes burden among individuals with limited financial resources, likely due to reduced access to healthcare, unhealthy living conditions and challenges in managing risk factors. These findings highlight the need for targeted interventions addressing socioeconomic disparities in diabetes prevention and care.

This pattern is consistent with findings [19] highlighted that individuals with lower socioeconomic status (SES) are more likely to experience poor glycaemic control and increased diabetes related complications due to limited access to healthcare, lower health literacy and inadequate resources for disease management. The findings observed that low-income populations face significant barriers to preventive care, such as limited access to regular health screenings and early interventions. These barriers often contribute to the higher prevalence of diabetes and related complications in these groups. Additionally, the study emphasized that environmental factors such as limited access to healthy food options and safe spaces for physical activity, further exacerbate the risk of diabetes in low income communities. In contrast, the data showed that the upper middle and high income groups accounted for only 4% and 5% of DM cases, respectively. The study noted that higher income individuals typically have better access to healthcare services including specialized diabetes care and are more likely to engage in health promoting behaviours such as regular physical activity and adherence to a balanced diet. Furthermore, the study found that socioeconomic disparities also influence access to advanced diabetes management technologies such as continuous glucose monitoring (CGM) and insulin pumps which significantly improve glycaemic control but are often unaffordable for lower income individuals. The study [20] emphasized that these disparities in access to care and technology contribute to better health outcomes in higher income groups further widening the health gap between socioeconomic classes. Addressing these disparities requires targeted public health interventions. The findings advocated for

policies to increase health literacy, improve access to preventive care and provide financial assistance for diabetes management in low income communities. The study [21] also suggested that integrating social determinants of health into diabetes care frameworks and expanding community based interventions could help bridge the gap in health outcomes between different income groups.

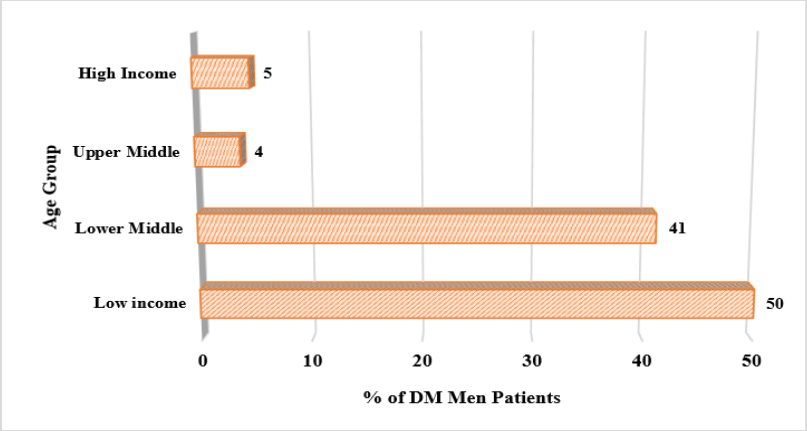


Figure 3. Percentage of DM male patients based on income group

The distribution of diabetic foot ulcers (DFUs) based on Wagner's classification was shown in Fig. 4. Grade I ulcers were identified in 5% of patients, Grade II in 10.8%, the majority (66.6%) in Grade III, followed by 11.7% with Grade IV and 5.9% with Grade V ulcers characterized by extensive gangrene. The predominance of Grades III and IV underscores the severity of DFU presentations at diagnosis. Similar trends have been reported previously found only 5% of patients with Grade I ulcers reflecting delays in early healthcare seeking, while emphasized Grade II as a critical stage for intervention and linked inadequate early management to progression toward advanced grades.

The study also identified gangrenous complications consistent with the findings associated localized gangrene in diabetic populations with peripheral arterial disease and delayed clinical care. Grade V ulcers characterized by extensive gangrene and often serving as a precursor to limb amputation were observed in 5.9% of patients. Importantly, the literature highlighted that such outcomes are largely preventable through timely multidisciplinary intervention and strict glycemic control [22].

Overall, these findings emphasize the need for proactive DFU management. Community-based screening, improved access to care, patient education and routine foot assessments are essential to prevent ulcer progression. The high proportion of advanced-stage ulcers in this study underscores the importance of timely multidisciplinary intervention to reduce amputation risk and improve outcomes.

The distribution of diabetes types among 102 patients was presented in Fig 5. The majority of cases accounting for 90.2% (92 patients) were diagnosed with Type II diabetes while Type I diabetes was identified in 6.9% of the total cases. Additionally, a small proportion 2.9% was classified as recent-onset diabetes indicating newly diagnosed cases. These results align with findings that Type II diabetes accounts for over 85% of diabetes cases globally, largely driven by lifestyle factors such as obesity, physical inactivity and aging populations [23].

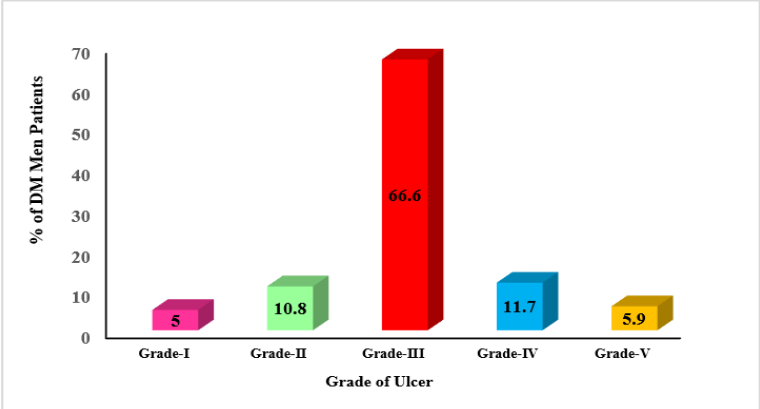


Figure 4. Percentage of DM male patients based on Wagner's grade of ulcer

The study observed a lower prevalence of Type I diabetes often linked to autoimmune mechanisms and predominantly affecting younger individuals while emphasized that early stage diabetes is often underdiagnosed due to mild or asymptomatic presentations which delays appropriate medical

intervention. The predominance of Type II diabetes in this population underscores the urgent need for public health strategies aimed at prevention and early detection emphasized that tailored interventions such as insulin therapy for Type I and lifestyle modifications for Type II are crucial for improving clinical outcomes and preventing complications. This comprehensive approach to diabetes management could significantly reduce the healthcare burden and improve the quality of life for affected individuals [24].

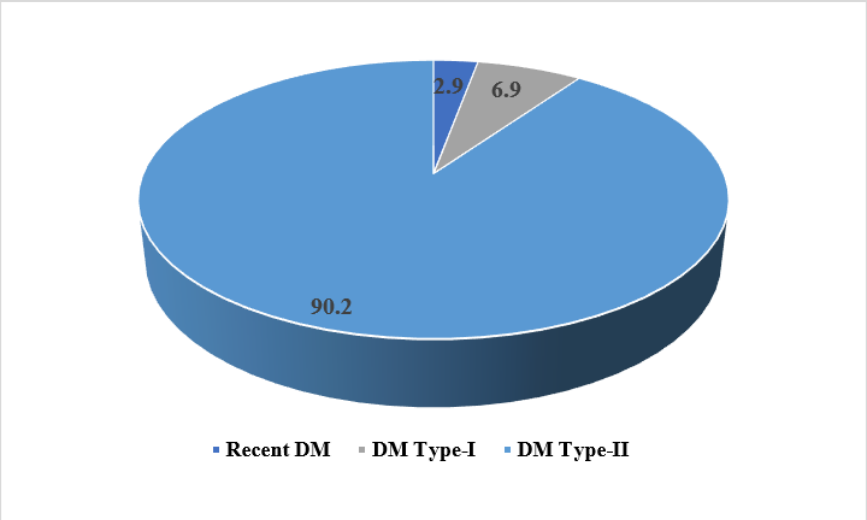


Figure 5. Percentage of DM male patients based on type

The distribution of patients by diabetes duration was shown in Fig. 6. Among 102 participants, half (50%, n = 51) had been living with diabetes for 9-12 years while 20.6% (n = 21) reported 3-5 years, 14.7% (n = 15) had 6-8 years and 10.8% (n = 11) had 0.5-2 years. Only 3.9% (n = 4) reported a disease history exceeding 12 years. This pattern revealed a substantial burden of long standing diabetes within the cohort with the majority experiencing the disease for nearly a decade. Such chronicity underscores the need for durable management strategies to address the cumulative impact of prolonged hyperglycaemia. These findings aligned with previous studies. The individuals with ≥10 years of diabetes are at markedly greater risk of neuropathy, retinopathy and cardiovascular disease, that intermediate durations (3-8 years) encompassing 35.3% of the present cohort, are strongly associated with microvascular complications [25]. Conversely, the smaller proportion with shorter disease duration (0.5-2 years; 10.8%) reflects the underdiagnosis and inadequate early management. The very low proportion of patients surviving beyond 12 years (3.9%) reflects the findings linked this to excess morbidity and mortality from advanced complications. Comprehensive care that includes lifestyle adjustment, organized monitoring, and prompt detection of problems is essential to improve outcomes and prolonging survival.

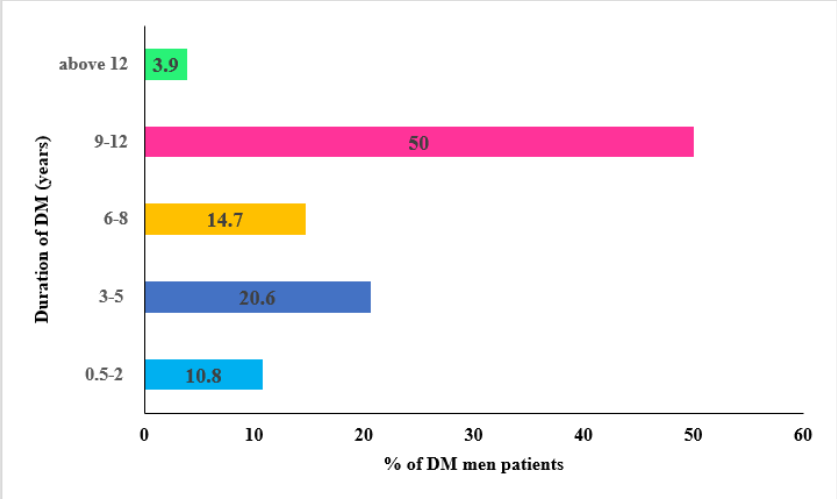


Figure 6. Percentage of DM male patients based on duration

A considerable proportion of the diabetic male cohort presented with reduced renal function consistent with diabetic kidney disease. Patients with lower eGFR tended to exhibit more advanced Wagner grades, suggesting that renal impairment may complicate the progression and healing of diabetic foot ulcers. This

study identified 21 bacterial isolates representing both gram-positive and gram-negative species (Tables 1 and 2). Gram-positive isolates included *Staphylococcus* spp., *Staphylococcus aureus* (M15, M33, M38), *Staphylococcus epidermidis* (M8) and *Streptococcus* sp. (M49). Gram-negative isolates comprised *Pseudomonas*, *Klebsiella* and *Proteus* spp., along with *Acinetobacter* sp. (M7) and *Escherichia coli* (M19). All these organisms are well-known contributors to diabetic foot infections.

Figure 7 shows that 47.6% of isolates were gram-positive and 52.4% were gram-negative, indicating a slightly higher prevalence of gram-negative bacteria. As shown in Figure 8, *Staphylococcus* spp. were most common (24%), followed by *Pseudomonas* spp. (19%). *Klebsiella* spp. and *Staphylococcus aureus* each contributed 14.3%, while *Proteus*, *Acinetobacter*, *E. coli*, *Staphylococcus haemolyticus* and *S. epidermidis* were less frequent (4.8–9.5%).

These findings show a predominance of gram-positive *Staphylococcus* spp., along with significant gram-negative pathogens such as *Pseudomonas* and *Klebsiella* spp. (Fig. 9). This distribution underscores the need for targeted diagnostic and therapeutic approaches in managing DFU infections. The predominance of Pseudomonas, Klebsiella, and Acinetobacter is notable, as these organisms are commonly associated with infections in CKD and dialysis patients. Frequent healthcare exposure, impaired immunity, and prolonged antibiotic therapy in renal patients may facilitate colonization by these multidrug-resistant species.

Similar studies have reported a high prevalence of *Pseudomonas* and *Klebsiella* species in DFUs, confirming their clinical relevance. These results align with the present study, which also found these gram-negative pathogens alongside dominant gram-positive *Staphylococcus* spp.

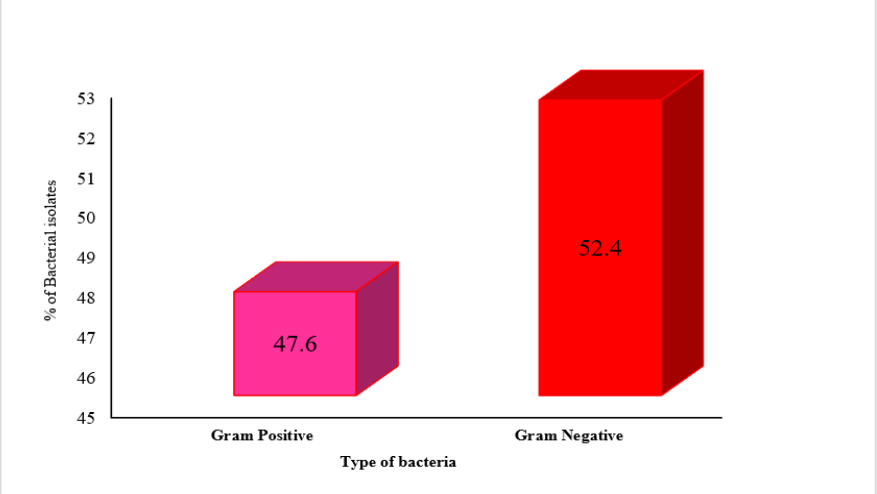


Figure 7. Percentage of bacterial isolates from DM male patients' foot ulcer

Antibiotic susceptibility testing of gram-positive isolates (Table 3, Fig. 10) showed varied responses. Cloxacillin showed the highest activity, particularly against *Staphylococcus epidermidis* (M8), followed by nitrofurantoin and erythromycin. Clindamycin and chloramphenicol showed moderate activity, while ampicillin was largely ineffective. The most resistant isolates were *Staphylococcus haemolyticus* (M2), *Staphylococcus* sp. (M12) and *Streptococcus* sp. (M49). These findings align with reported clindamycin and erythromycin sensitivity and further support nitrofurantoin's usefulness in diabetic patients.

Kidney-Adjusted Antibiotic Considerations

The antibiotic susceptibility patterns identified in this study must be interpreted carefully in the context of CKD. Several antibiotics commonly used for DFU infections—including aminoglycosides, vancomycin, and certain fluoroquinolones—pose significant nephrotoxic risk or require strict renal dose adjustment. Even beta-lactams such as cefotaxime and ceftazidime require modification of dosing intervals in moderate to advanced CKD. As multidrug-resistant organisms increase, nephrologists must balance antimicrobial potency with renal safety to avoid treatment-induced decline in kidney function. Culture-guided therapy is therefore indispensable for CKD and dialysis patients.

In kidney transplant recipients, immunosuppressive therapy further increases susceptibility to polymicrobial infections. Multidrug-resistant organisms such as Pseudomonas and Klebsiella have been associated with graft-threatening systemic infections, emphasizing the importance of precise microbial identification and resistance profiling.

The high level of antimicrobial resistance observed in this study is particularly concerning for nephrology care, where therapeutic options are already limited by drug-related nephrotoxicity. Fluoroquinolones, aminoglycosides, and certain β-lactams require dosage modification or avoidance in advanced CKD, making culture-guided therapy indispensable.

CKD imposes substantial immunologic and metabolic constraints that increase the severity of diabetic foot infections. Uremic toxins impair leukocyte function, while chronic inflammation and malnutrition hinder

tissue repair. As a result, CKD patients—especially those on dialysis—present with more complex infection profiles and higher amputation risks.

Among the gram-negative isolates (Table 4, Fig. 11), doxycycline, ciprofloxacin and ceftazidime showed the highest activity, particularly against *Pseudomonas* sp. (M40) and *Klebsiella* sp. (M42), with inhibition zones up to 33 mm. Ofloxacin, co-trimoxazole and tobramycin showed moderate efficacy, while pronounced resistance occurred in *Acinetobacter* sp. (M7), *E. coli* (M19) and *Pseudomonas* sp. (M43). These findings reflect the growing multidrug resistance among gram-negative pathogens and confirm doxycycline's strong activity against *Klebsiella*. Overall, the results highlight the importance of routine susceptibility testing, careful antibiotic selection and strong stewardship to manage DFU infections and limit resistance.

The findings from this study highlight the need for integrated foot-screening and infection-monitoring protocols in CKD, dialysis, and diabetic kidney disease clinics. Routine assessment of skin integrity, early detection of ulceration, and prompt culture-guided therapy can significantly reduce systemic infection risk in renal patients. Collaboration between nephrologists, infectious disease specialists, and wound-care teams is essential to optimize antibiotic selection and prevent nephrotoxic drug exposure. Incorporating DFU risk assessment into kidney-disease management pathways may reduce hospitalization, improve patient quality of life, and protect long-term renal outcomes.

Subanalysis: Relevance of Findings in CKD and Diabetic Kidney Disease

Although the present study did not include direct measurement of kidney function, the clinical relevance of these findings is substantial for patients with chronic kidney disease (CKD) and diabetic kidney disease (DKD). Individuals with impaired renal function typically exhibit delayed wound healing, impaired leukocyte activity, and reduced microcirculation, all of which amplify infection severity. The microbial trends observed in this study mirror bacterial patterns commonly reported in CKD populations, especially the increased presence of gram-negative bacilli such as Pseudomonas and Klebsiella. These organisms often colonize dialysis patients due to immunosuppression, vascular access exposure, and frequent antibiotic use. The observed antimicrobial resistance profiles therefore have important implications for infection control and therapeutic choices in CKD/DKD patients.

For nephrologists, integrating routine foot examinations into CKD and dialysis clinics, early detection of infections, and reliance on antibiogram-driven therapy are essential steps to prevent systemic complications and protect renal function. These results highlight the therapeutic import

Many antibiotics demonstrating susceptibility in vitro, such as aminoglycosides and some cephalosporins, pose nephrotoxicity risks or require dosage adjustments in CKD. Thus, nephrologists managing DFU infections in CKD patients must balance microbial sensitivity with renal safety. The resistance of Acinetobacter and E. coli further complicates treatment in patients with compromised kidney function.

Table 1. Biochemical characteristics of bacteria isolated from DM male patients

S. No	Physical and Biochemical Test	Inference																				
		M2	M8	M11	M12	M15	M21	M29	M33	M38	M49	M5	M7	M19	M24	M32	M34	M40	M42	M43	M44	M46
Physical Characters																						
1.	Gram staining	G +ve	G +ve	G +ve	G +ve	G +ve	G +ve	G +ve	G +ve	G +ve	G +ve	G -ve	G -ve	G -ve	G -ve	G -ve	G -ve	G -ve	G -ve	G -ve	G -ve	G -ve
2.	Morphology	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Cluster	Cocci in Chain	Rod	Cocco-bacilli	Rod	Rod	Rod	Rod	Rod	Rod	Rod	Rod	Rod
3.	Motility	Non-motile	Non-motile	Non-motile	Non-motile	Non-motile	Non-motile	Non-motile	Non-motile	Non-motile	Non-motile	Motile	Non-motile	Motile	Non-motile	Non-motile	Motile	Motile	Non-motile	Motile	Motile	Motile
Biochemical Characters																						
4.	Indole Test	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	+
5.	Methyl red test	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	+	+
6.	Voges-Proskauer test	+	+	+	+	+	+	+	+	+	+	-	+	-	+	+	-	-	+	-	_-	_-
7.	Citrate utilization	+	-	-	-	-	-	-	-	-	-	+	+	-	+	+	+	+	+	+	+	+
8.	Glucose	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
9.	Fructose	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	-	+	-	+	+
10.	Galactose	+	+	+	+	+	+	+	+	+	+	-	-	+	+	+	-	+	+	-	+	+
11.	Lactose	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	-	-	-
12.	Maltose	+	+	+	+	+	+	+	+	+	+	-	+	+	+	-	-	+	-	-	+	+
13.	Sucrose	+	+	+	+	+	+	+	+	+	+	+	+	-	+	+	-	+	+	-	+	+
14.	Rhamnose	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	+	-	-	-

15.	Mannitol	-	+	+	+	+	+	+	+	+	+	+	-	+	+	+	-	+	+	-	+	+
16.	Oxidase test	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	-
17.	Catalase	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
18.	Coagulase	-	-	+	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-
19.	Starch hydrolysis	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
20.	Urease test	+	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	+	+
21.	Gelatin utilization	+	-	+	+	+	+	+	+	+	+	+	-	-	-	-	+	+	-	+	+	+
22.	Nitrate reduction	-	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

M2- *Staphylococcus haemolyticus*	M12- *Staphylococcus* sp	M29- *Staphylococcus* sp	M49- *Streptococcus* sp	M19- *E.coli*	M34- *Pseudomonas* sp	M43- *Pseudomonas* sp	+- Positive
M8- *Staphylococcus epidermidis*	M15- *Staphylococcus aureus*	M33- *Staphylococcus aureus*	M5- *Pseudomonas* sp	M24- *Klebsiella* sp	M40- *Pseudomonas* sp	M44- *Proteus* sp	- - Negative
M11- *Staphylococcus* sp	M21- *Staphylococcus* sp	M38- *Staphylococcus aureus*	M7- *Acinetobacter* sp	M32- *Klebsiella* sp	M42- *Klebsiella* sp	M46- *Proteus* sp	

S. No	**Organism**	**Isolate Number**
Gram Positive Bacteria		
1.	*Staphylococcus haemolyticus*	M2
2.	*Staphylococcus epidermidis*	M8
3.	*Staphylococcus* sp.	M11
4.	*Staphylococcus* sp.	M12
5.	*Staphylococcus aureus*	M15
6.	*Staphylococcus* sp.	M21
7.	*Staphylococcus* sp.	M29
8.	*Staphylococcus aureus*	M33
9.	*Staphylococcus aureus*	M38
10.	*Streptococcus* sp.	M49
Gram Negative Bacteria		
11.	*Pseudomonas* sp.	M5
12.	*Acinetobacter* sp.	M7
13.	*E. coli*	M19
14.	*Klebsiella* sp.	M24
15.	*Klebsiella* sp.	M32
16.	*Pseudomonas* sp.	M34
17.	*Pseudomonas* sp.	M40
18.	*Klebsiella* sp.	M42
19.	*Pseudomonas* sp.	M43
20.	*Proteus* sp.	M44
21.	*Proteus* sp.	M46

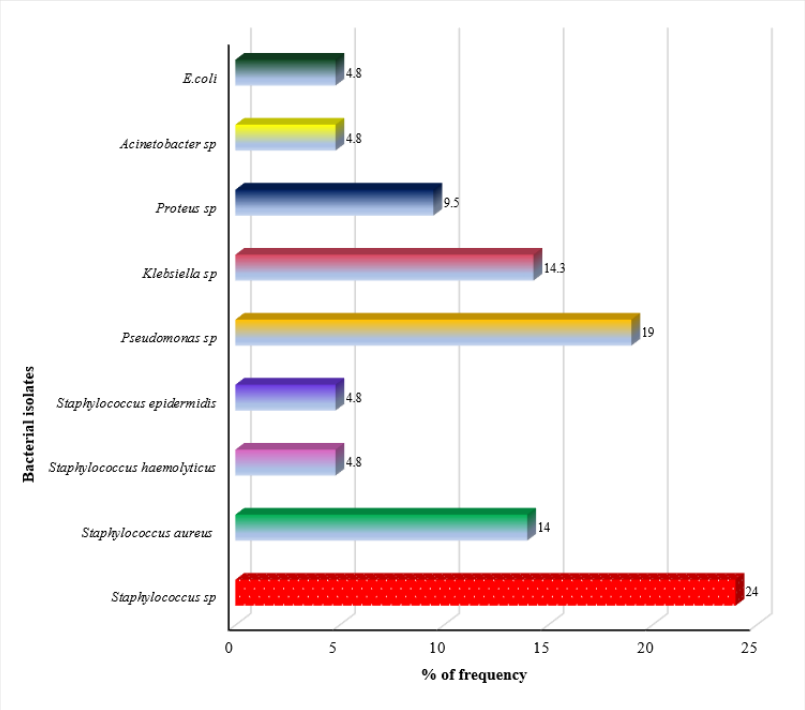


Table 2. Isolation of bacteria from DM male patients' foot ulcer

Figure 8. Percentage of frequency of different bacterial isolates from DM male patients' foot ulcer

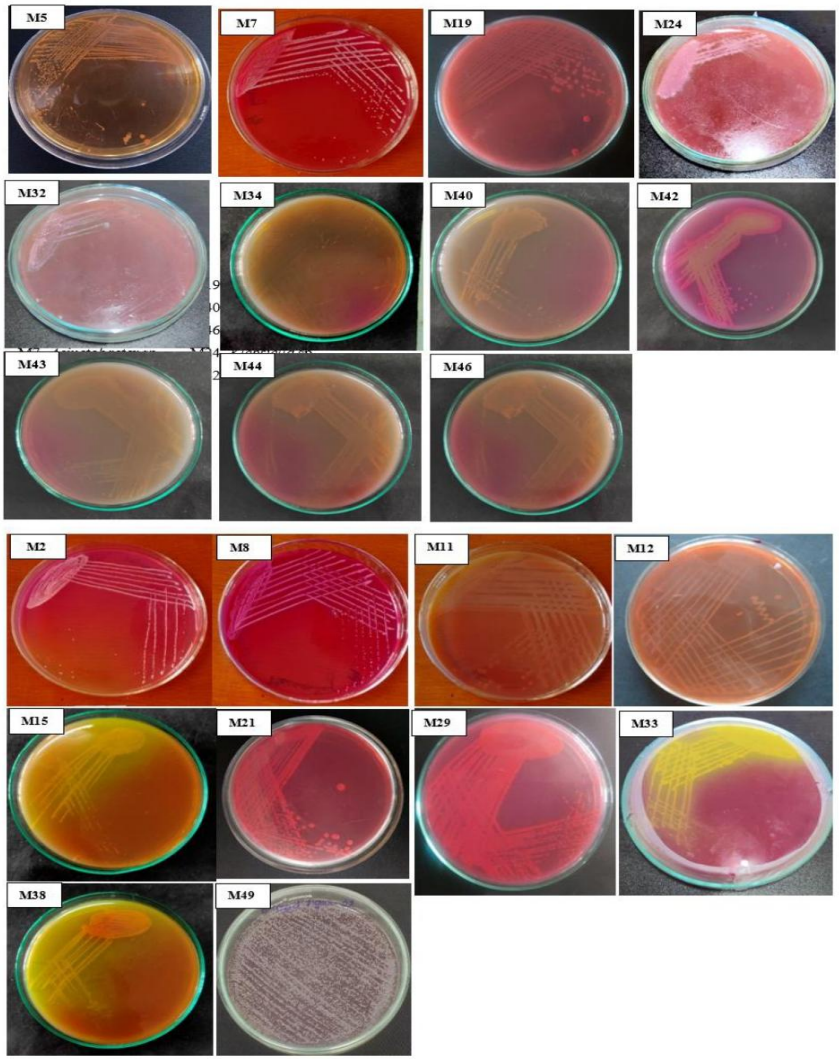


Figure 9. Gram positive and Gram negative bacteria isolated from DM male patients' foot ulcer

M2- *Staphylococcus haemolyticus*
M5- *Pseudomonas* sp.
M7- *Acinetobacter* sp.
M8- *Staphylococcus epidermidis*
M11- *Staphylococcus* sp.
M12- *Staphylococcus* sp.
M15- *Staphylococcus aureus*
M19- *E. coli*
M21- *Staphylococcus* sp.
M24 - *Klebsiella* sp.
M29- *Staphylococcus* sp.
M32 - *Klebsiella* sp.
M34- *Pseudomonas* sp.
M33- *Staphylococcus aureus*
M38- *Staphylococcus aureus*
M49- *Streptococcus* sp.
M40- *Pseudomonas* sp.
M42- *Klebsiella* sp.
M43 - *Pseudomonas* sp.
M44- *Proteus* sp.
M46- *Proteus* sp.

Table 3. Antibiotic sensitivity test of Gram-positive bacteria isolated from DM male patients

S.NO	ANTIBIOTIC DISCS	CON. OF ANTIBIOTICS (µg)	ZONE OF INHIBITION mm									
			M2	M8	M11	M12	M15	M21	M29	M33	M38	M49
1	Clindamycin (CD)	2	0 (R)	0 (R)	19 (I)	0 (R)	0 (R)	18 (I)	0 (R)	0 (R)	0 (R)	0 (R)
2	Nitrofurantoin (NIT)	300	0 (R)	22 (S)	23(S)	8 (R)	19 (S)	8(R)	0 (R)	12 (I)	0 (R)	16 (I)
3	Gentamicin (GEN)	10	0 (R)	15 (S)	23 (S)	7 (R)	0 (R)	13 (I)	8 (R)	0 (R)	18 (S)	14 (I)
4	Cloxacillin (COX)	30	0 (R)	34 (S)	27 (S)	0 (R)	0 (R)	0 (R)	17 (R)	0 (R)	0 (R)	0 (R)
5	Ampicillin (AMP)	10	0 (R)	13 (R)	20 (R)	0 (R)	0 (R)	0 (R)	0 (R)	10	0 (R)	0 (R)
6	Chloramphenicol (C)	30	0 (R)	22 (S)	24 (S)	8 (R)	0 (R)	15 (I)	0 (R)	0 (R)	0 (R)	15 (I)
7	Amoxyclav (AMC)	30	14 (I)	16 (I)	0 (R)	0 (R)	0 (R)	0 (R)	0 (R)	14 (I)	19 (S)	0 (R)
8	Erythromycin (E)	15	0 (R)	22 (S)	26 (S)	0 (R)	0 (R)	0 (R)	0 (R)	0 (R)	11 (R)	0 (R)

M2- *Staphylococcus haemolyticus*
M8- *Staphylococcus epidermidis*
M11- *Staphylococcus* sp
M12- *Staphylococcus* sp
M15- *Staphylococcus aureus*
M21- *Staphylococcus* sp
M29- *Staphylococcus* sp
M33- *Staphylococcus aureus*
M38- *Staphylococcus aureus*
M49- *Streptococcus* sp

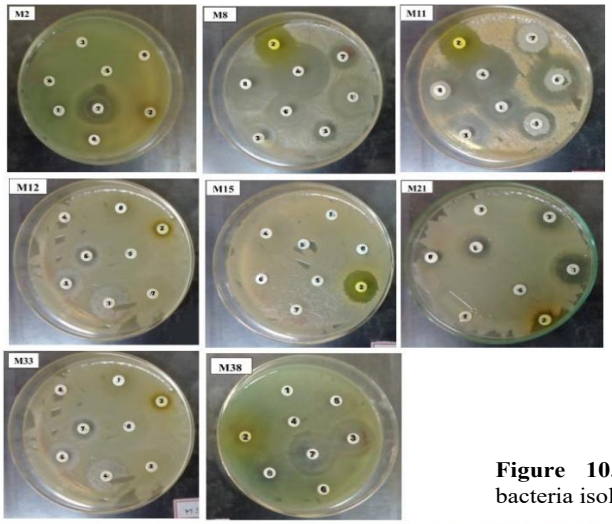
S- Sensitive
I- Intermediate
R-Resistant

S. NO	ANTIBIOTIC DISCS	CON. OF ANTIBIOTICS (µg)	ZONE OF INHIBITION (mm)										
			M5	M7	M19	M24	M32	M34	M40	M42	M43	M44	M46
1	Doxycycline (DO)	30	16 (S)	0 (R)	0 (R)	14 (S)	22 (S)	16 (S)	31 (S)	33 (S)	0 (R)	7 (R)	12 (S)
2	Ofloxacin (OF)	5	20 (S)	19 (S)	12 (R)	10 (R)	12 (R)	16 (S)	28 (S)	22 (S)	12 (R)	11 (R)	0 (R)
3	Cefotaxime (CTX)	30	12 (R)	22 (I)	10 (R)	24 (R)	22 (I)	18 (R)	30 (S)	20 (I)	0 (R)	0 (R)	25 (S)
4	Ciprofloxacin (CIP)	5	18 (I)	12 (R)	16	7 (R)	21 (S)	16 (I)	31 (S)	29 (S)	19(I)	18(I)	0 (R)
5	Co-Trimoxazole (COT)	25	0 (R)	15 (I)	0 (R)	14 (I)	16 (S)	0 (R)	28 (S)	28 (S)	0 (R)	0 (R)	0 (R)
6	Tobramycin (TOB)	10	18 (S)	0 (R)	15 (S)	0 (R)	14 (I)	0 (R)	27 (S)	25 (S)	0 (R)	10 (R)	0 (R)
7	Ceftazidime (CAZ)	30	17 (I)	23 (S)	19 (S)	12 (R)	21 (S)	15 (I)	31 (S)	20 (S)	17 (I)	16 (I)	0 (R)
8	Streptomycin (S)	10	10 (R)	10 (R)	13 (I)	14 (I)	12 (I)	17 (S)	30 (S)	23 (S)	15 (S)	19 (S)	10 (R)

Table 4: Antibiotic sensitivity test of Gram-Negative bacteria isolated from DM male patients

M5- *Pseudomonas* sp.
M32- *Klebsiella* sp.
M43- *Pseudomonas* sp.
M7- *Acinetobacter* sp.
M34- *Pseudomonas* sp.
M44- *Proteus* sp.
M19- *E. coli*
M40- *Pseudomonas* sp.
M46- *Proteus* sp.
M24- *Klebsiella* sp.
M42- *Klebsiella* sp.

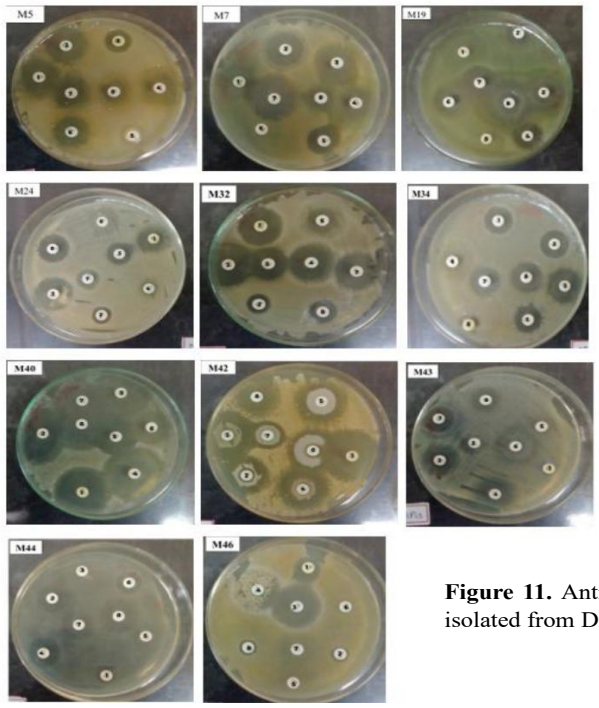
S- Sensitive
I- Intermediate
R-Resistant



M2- *Staphylococcus haemolyticus*
M8- *Staphylococcus epidermidis*
M11- *Staphylococcus* sp.
M12- *Staphylococcus* sp.
M15- *Staphylococcus aureus*
M21- *Staphylococcus* sp.
M29- *Staphylococcus* sp.
M33- *Staphylococcus aureus*
M38- *Staphylococcus aureus*
M49- *Streptococcus* sp.

1- Clindamycin (CD)
2- Nitrofurantoin (NIT)
3- Gentamicin (GEN)
4- Cloxacillin (COX)
5- Ampicillin (AMP)
6- Chloramphenicol (C)
7- Amoxiclav (AMC)
8- Erythromycin (E)

Figure 10. Antibiogram profile of Gram-Positive bacteria isolated from DM male patients' foot ulcer



M5- *Pseudomonas* sp.
M7- *Acinetobacter* sp.
M19- *E. coli*
M24- *Klebsiella* sp.
M32- *Klebsiella* sp.
M34- *Pseudomonas* sp.
M40- *Pseudomonas* sp.
M42- *Klebsiella* sp.
M43- *Pseudomonas* sp.
M44- *Proteus* sp.
M46- *Proteus* sp.

1- Doxycycline (DO)
2- Ofloxacin (OF)
3- Cefotaxime (CTX)
4- Ciprofloxacin (CIP)
5- Co-trimoxazole (COT)
6- Tobramycin (TOB)
7- Ceftazidime (CAZ)
8- Streptomycin (S)

Figure 11. Antibiogram profile of Gram-Negative bacteria isolated from DM male patients' foot ulcer.

Conclusion

This study underscores the critical role of antibiogram analysis in managing DFU infections among male diabetic patients. The prevalence of multidrug-resistant bacteria calls for early microbiological diagnostics and tailored therapies. Effective implementation of antibiotic stewardship programs, combined with patient education on foot care and timely medical interventions, is essential for reducing AMR and improving patient outcomes.

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Conflict of interest

The author should declare that there is no conflict of interest.

Acknowledgements

Nil

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