Section: General Surgery

Original Research Article

# MICROBIOLOGICAL PROFILE AND ANTIBIOTIC RESISTANCE PATTERNS IN DIABETIC FOOT ULCERS: IMPLICATIONS FOR EMPIRICAL THERAPY

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## Abstract

**Background:** Diabetic foot ulcers (DFUs) are a leading cause of diabetes-related hospitalisation and lower-limb amputation, and their infections are increasingly complicated by multidrug-resistant (MDR) organisms. Knowledge of the local microbiological profile and resistance pattern is essential for rational empirical antibiotic therapy. This study characterised the bacteriology and antibiotic resistance of DFUs at a North Indian tertiary-care centre. **Materials and Methods:** A cross-sectional analysis of 33 patients with infected DFUs was performed. Wound specimens were processed by standard aerobic culture; organisms were identified and antimicrobial susceptibility determined per CLSI criteria. MDR was defined using standard international criteria. Descriptive statistics, chi-square/Fisher and Welch t-tests were applied; $p<0.05$ was significant. **Result:** Mean age was 59.7 ± 10.1 years with a male-to-female ratio of 2.3:1; 76% had poor glycaemic control (HbA1c ≥8%). Cultures were positive in 31/33 (94%); 42% of positive cultures were polymicrobial (mean 1.48 isolates/positive culture). Of 46 isolates, Gram-negative bacteria predominated (61%). The commonest isolates were Staphylococcus aureus (19.6%), Escherichia coli (17.4%) and Pseudomonas aeruginosa (15.2%). MDR organisms accounted for 44% of isolates; 56% of S. aureus were methicillin-resistant and 57% of E. coli/K. pneumoniae were ESBL-type. Gram-negative isolates retained high susceptibility to colistin (100%), amikacin (89%), carbapenems (86%) and piperacillin-tazobactam (75%); all Gram-positive isolates were susceptible to vancomycin, teicoplanin and linezolid. MDR carriage was significantly associated with Wagner grade ≥3 ($p=0.014$). **Conclusion:** DFUs at our centre were predominantly Gram-negative with a high MDR burden. Empirical regimens should pair a beta-lactam/beta-lactamase inhibitor or carbapenem with anti-MRSA cover in severe or high-risk ulcers, then be de-escalated on culture. Periodic local antibiograms are indispensable to guide therapy.

## INTRODUCTION

Diabetes mellitus has reached pandemic proportions, with an estimated 589 million adults aged 20–79 years living with the disease worldwide in 2024, a figure projected to rise to 853 million by 2050.[1] South Asia carries a disproportionate share of this burden, and India alone accounts for approximately 89 million people with diabetes—the second-largest national burden globally.[2] Diabetic foot ulcers (DFUs) are among the most disabling and costly complications of diabetes and are the single most common cause of diabetes-related hospitalisation and non-traumatic lower-limb amputation.[3]

Infection supervenes in a large proportion of DFUs and is the proximate trigger for most major amputations. Effective management depends on prompt, appropriate antibiotic therapy, which is almost always begun empirically before culture results are available.[3] The choice of empirical regimen must therefore be informed by the local microbiological profile and resistance pattern, both of which vary considerably by geography and over time. In the Indian setting, several series have documented a predominance of Gram-negative bacilli and an


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alarmingly high prevalence of multidrug-resistant (MDR) organisms, driven in part by the unregulated use of antibiotics.[4,5] Where empirical therapy is misaligned with the prevailing flora, treatment failure, prolonged hospitalisation and amputation become substantially more likely.[6]

Reported microbiological patterns are not uniform. Some centres describe Gram-negative predominance with high rates of extended-spectrum beta-lactamase (ESBL) production, whereas others report Gram-positive or monomicrobial predominance, underscoring that empirical recommendations cannot be transferred uncritically between institutions.[5,7] Against the background of escalating antimicrobial resistance documented in recent systematic reviews,[7] periodic institution-specific surveillance is essential. The present study was undertaken to define the bacteriological profile and antibiotic resistance pattern of infected DFUs at our tertiary-care centre and to derive evidence-based implications for empirical antibiotic therapy.

## MATERIALS AND METHODS

**Study design and setting:** This was a hospital-based cross-sectional study of 33 consecutive patients presenting with clinically infected diabetic foot ulcers to the Departments of Surgery and Microbiology at a tertiary-care teaching hospital in North India. Adult patients (≥18 years) with established diabetes mellitus and a clinically infected foot ulcer (Wagner grade ≥1 with local or systemic signs of infection) were eligible. Patients who had received a full course of culture-directed antibiotics for the current ulcer episode, and those with non-diabetic ulcers, were excluded.

**Specimen collection and microbiology:** After surface debridement and saline cleansing, deep-tissue or curettage specimens were collected to minimise surface contamination and transported promptly to the microbiology laboratory. Specimens were inoculated onto blood agar, MacConkey agar and chocolate agar and incubated aerobically at 37°C for 24–48 hours. Isolates were identified by colony morphology, Gram staining and standard biochemical reactions.

**Antimicrobial susceptibility testing:** Antimicrobial susceptibility was determined by the Kirby-Bauer disc-diffusion method and interpreted according to Clinical and Laboratory Standards Institute (CLSI) criteria.8 Methicillin resistance in staphylococci was screened using a cefoxitin disc; ESBL production in Enterobacterales was assessed phenotypically. Multidrug resistance (MDR) was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, following the international consensus definition.9

**Statistical analysis:** Data were analysed using Python (SciPy/pandas). Continuous variables are expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Associations between MDR carriage and clinical predictors were tested using the Pearson chi-square test with Yates' correction, or Fisher's exact test where any expected cell count was below five; continuous variables were compared using the Welch t-test. A two-sided p-value < 0.05 was considered statistically significant.

**Data note:** The dataset analysed and reported here is a simulated (synthetic) dataset of 33 patient records and 46 isolates, computer-generated to mirror the distributions of published Indian tertiary-care DFU series for the purpose of demonstrating the full analytical and reporting pipeline. The values are not derived from real patients and must be replaced with an audited institutional dataset before any scientific or clinical inference is drawn or the manuscript is submitted.

## RESULTS

**Demographic and clinical characteristics:** Thirty-three patients with infected DFUs were studied. The mean age was 59.7 ± 10.1 years (range 38-79 years), with a clear male preponderance (male-to-female ratio 2.3:1). The mean duration of diabetes was 10.8 ± 4.5 years and the mean HbA1c was 9.2 ± 1.4%, with 25 patients (76%) showing poor glycaemic control (HbA1c ≥8%). Peripheral neuropathy was present in 76% and peripheral arterial disease in 39% of patients, while 48% had received antibiotics in the preceding four weeks. The demographic and clinical profile is summarised in [Table 1].

**Table 1: Demographic and clinical characteristics of the study cohort (n = 33).**

| Characteristic | Value |
|---|---|
| Number of patients | 33 |
| Age, years (mean ± SD) | 59.7 ± 10.1 |
| Age range, years | 38-79 |
| Male, n (%) | 23 (70%) |
| Female, n (%) | 10 (30%) |
| Male:Female ratio | 2.3:1 |
| Diabetes duration, years (mean ± SD) | 10.8 ± 4.5 |
| HbA1c, % (mean ± SD) | 9.2 ± 1.4 |
| Poor glycaemic control (HbA1c ≥8%), n (%) | 25 (76%) |
| Peripheral neuropathy, n (%) | 25 (76%) |
| Peripheral arterial disease, n (%) | 13 (39%) |
| Prior antibiotic exposure, n (%) | 16 (48%) |
| Wagner grade 1 / 2 / 3 / 4, n | 11 / 9 / 9 / 4 |


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**Culture positivity and microbial profile:** Cultures were positive in 31 of 33 ulcers (94%); 2 specimens showed no growth. Among culture-positive ulcers, 18 (58%) were monomicrobial and 13 (42%) polymicrobial, yielding 46 isolates overall (mean 1.48 isolates per positive culture). Gram-negative bacilli predominated, accounting for 28 of 46 isolates (61%), versus 18 (39%) Gram-positive isolates. The single commonest organism was Staphylococcus aureus, followed by Escherichia coli and Pseudomonas aeruginosa [Table 2]. This Gram-negative predominance is concordant with contemporary Indian and regional DFU series.[4,5,10]

**Table 2: Frequency and Gram classification of the 46 microbial isolates.**

| Organism | n | % of isolates | Gram category |
|---|---|---|---|
| Staphylococcus aureus | 9 | 19.6 | Gram-positive |
| Escherichia coli | 8 | 17.4 | Gram-negative |
| Pseudomonas aeruginosa | 7 | 15.2 | Gram-negative |
| Klebsiella pneumoniae | 6 | 13.0 | Gram-negative |
| Proteus mirabilis | 4 | 8.7 | Gram-negative |
| Enterococcus faecalis | 4 | 8.7 | Gram-positive |
| Coagulase-negative Staphylococcus | 3 | 6.5 | Gram-positive |
| Acinetobacter baumannii | 3 | 6.5 | Gram-negative |
| Streptococcus agalactiae | 2 | 4.3 | Gram-positive |
| Total isolates | 46 | 100.0 | — |

**Multidrug resistance burden:** MDR organisms constituted 20 of 46 isolates (44%), and 17 of 31 culture-positive patients (55%) harboured at least one MDR isolate. Methicillin resistance was detected in 5 of 9 S. aureus isolates (56%), and ESBL-type resistance in 57% of E. coli and K. pneumoniae isolates. These figures are consistent with the high MDR/ESBL/MRSA prevalence repeatedly reported from Indian tertiary centres.[4,9,11]

**Antibiotic resistance patterns:** Among Gram-negative isolates, resistance was high to the agents most often used for unguided empirical therapy—amoxicillin-clavulanate (76%), third-generation cephalosporins such as ceftriaxone (76%) and fluoroquinolones (68%). In contrast, susceptibility remained high to colistin (100% susceptible), amikacin (89%), the carbapenems imipenem and meropenem (86%) and piperacillin-tazobactam (75%) [Table 3]. Among Gram-positive isolates, resistance to penicillin was universal and to erythromycin and fluoroquinolones was common, but all isolates were fully susceptible to vancomycin, teicoplanin and linezolid [Table 4].

**Table 3: Antibiotic resistance pattern of Gram-negative isolates (n = 28).**

| Antibiotic | Resistant / tested | % Resistant | % Susceptible |
|---|---|---|---|
| Amoxicillin-clavulanate | 16/21 | 76.2 | 23.8 |
| Ceftriaxone | 16/21 | 76.2 | 23.8 |
| Cefepime | 12/28 | 42.9 | 57.1 |
| Piperacillin-tazobactam | 7/28 | 25.0 | 75.0 |
| Ciprofloxacin | 19/28 | 67.9 | 32.1 |
| Gentamicin | 14/28 | 50.0 | 50.0 |
| Cotrimoxazole | 13/21 | 61.9 | 38.1 |
| Amikacin | 3/28 | 10.7 | 89.3 |
| Imipenem | 4/28 | 14.3 | 85.7 |
| Meropenem | 4/28 | 14.3 | 85.7 |
| Colistin | 0/24 | 0.0 | 100.0 |

**Table 4: Antibiotic resistance pattern of Gram-positive isolates (n = 18). Cefoxitin and clindamycin not reported for enterococci.**

| Antibiotic | Resistant / tested | % Resistant | % Susceptible |
|---|---|---|---|
| Penicillin | 18/18 | 100.0 | 0.0 |
| Cefoxitin | 6/14 | 42.9 | 57.1 |
| Erythromycin | 11/18 | 61.1 | 38.9 |
| Clindamycin | 6/14 | 42.9 | 57.1 |
| Ciprofloxacin | 10/18 | 55.6 | 44.4 |
| Gentamicin | 8/18 | 44.4 | 55.6 |
| Cotrimoxazole | 8/18 | 44.4 | 55.6 |
| Linezolid | 0/18 | 0.0 | 100.0 |
| Vancomycin | 0/18 | 0.0 | 100.0 |
| Teicoplanin | 0/18 | 0.0 | 100.0 |

**Predictors of multidrug resistance:** On bivariate analysis, MDR carriage was significantly more frequent in patients with Wagner grade ≥3 ulcers ($p = 0.014$, Chi-square). A higher proportion of polymicrobial ulcers harboured MDR organisms, though this did not reach significance in this small sample ($p = 0.083$). Mean HbA1c was numerically higher among MDR-positive patients (9.5 ± 1.5%) than MDR-negative patients (8.8 ± 1.4%), but the difference was not statistically significant ($p =$


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0.196); the categorical association between poor glycaemic control and MDR was likewise non-significant (p = 1.0). Results of the inferential analyses are presented in [Table 5].

**Table 5: Bivariate predictors of multidrug-resistant organism carriage among culture-positive patients (n = 31). p < 0.05 considered significant.**

| Comparison | p-value | Test |
|---|---|---|
| MDR vs Wagner grade (≥3 vs <3) | 0.014 | Chi-square |
| MDR vs culture pattern (poly vs mono) | 0.083 | Chi-square |
| MDR vs glycaemic control (HbA1c ≥8 vs <8) | 1.0 | Fisher exact |
| MDR vs prior antibiotic exposure | 1.0 | Chi-square |
| MDR vs peripheral arterial disease | 0.123 | Chi-square |
| HbA1c by MDR (9.5 ± 1.5 vs 8.8 ± 1.4) | 0.196 | Welch t-test |
| Age by MDR (60.4 ± 10.7 vs 58.3 ± 10.2) | 0.588 | Welch t-test |

## DISCUSSION

This study characterised the microbiology and resistance pattern of infected DFUs and translated the findings into empirical-therapy implications. Three observations stand out: a Gram-negative predominance, a substantial MDR burden, and preserved activity of a small group of reserve agents. Each carries direct consequences for empirical prescribing.

Gram-negative bacilli accounted for roughly three-fifths of isolates, with S. aureus, E. coli and P. aeruginosa the leading pathogens. This pattern mirrors a consistent body of Indian and regional evidence in which Gram-negative organisms outnumber Gram-positive ones in DFUs, in contrast to the historical Western emphasis on staphylococci.[4,5,10] A prospective Indian series similarly reported Gram-negative predominance and high susceptibility of Gram-negative isolates to carbapenems and amikacin,[5] and a large Indian tertiary-care study documented MDR organisms in nearly three-quarters of patients with associations to neuropathy, osteomyelitis and ulcer size.[4] Our polymicrobial rate (42% of positive cultures) is in keeping with regional reports of polymicrobial infection in around 40% of cases.[11]

The MDR burden was considerable: 44% of isolates were MDR, 56% of S. aureus were methicillin-resistant and 57% of key Enterobacterales were ESBL-type. These proportions echo recent multicentre and single-centre data describing rising resistance in diabetic foot infections worldwide,[7,12,13] and reinforce that empirical regimens designed for low-resistance settings are likely to fail locally. Importantly, the agents most commonly chosen for unguided therapy—co-amoxiclav, third-generation cephalosporins and fluoroquinolones—showed resistance exceeding two-thirds among Gram-negative isolates, whereas amikacin, carbapenems, piperacillin-tazobactam and colistin retained high activity.[6,14]

The significant association between MDR carriage and Wagner grade ≥3 is clinically intuitive: deeper, more advanced ulcers are more often chronic, previously treated and colonised by resistant flora, and this gradient has been observed in other cohorts.[4] The non-significant trends for polymicrobial infection and poor glycaemic control are consistent with a true effect that this small sample is underpowered to detect, and merit re-examination in a larger dataset.

Taken together, these data support a stratified empirical strategy. For mild, previously untreated ulcers, narrower Gram-positive–focused cover may suffice, whereas moderate-to-severe or high-risk ulcers warrant broad Gram-negative cover with a beta-lactam/beta-lactamase inhibitor or, where MDR risk is high, a carbapenem, combined with anti-MRSA therapy (vancomycin, teicoplanin or linezolid) pending culture.[3,6,15] Empirical therapy should always be de-escalated once susceptibility results are available, and antimicrobial stewardship reinforced. Because resistance patterns differ markedly between institutions and drift over time, a periodically updated local antibiogram—rather than extrapolation from external guidelines—should anchor empirical choices.[5,14]

**Limitations:** This study has several limitations. The sample of 33 patients is modest, limiting statistical power for subgroup and predictor analyses. Only aerobic culture was performed, so anaerobes and fastidious organisms may be under-represented. The single-centre, cross-sectional design limits generalisability and precludes assessment of clinical outcomes such as amputation or mortality. Finally, and most importantly, the dataset analysed here is synthetic and was generated to demonstrate the analytical pipeline; the quantitative findings must be regarded as illustrative until replaced by audited real-world data.

## CONCLUSION

Infected diabetic foot ulcers at our tertiary-care centre were predominantly Gram-negative and carried a high burden of multidrug resistance, including substantial MRSA and ESBL prevalence. Commonly used first-line empirical agents showed unacceptably high resistance, while amikacin, carbapenems, piperacillin-tazobactam and colistin (for Gram-negatives) and the glycopeptides and linezolid (for Gram-positives) retained reliable activity. Empirical therapy should be stratified by ulcer severity and MDR risk, guided by a current local antibiogram, and promptly de-escalated on culture. Ongoing microbiological surveillance and antimicrobial stewardship are essential to contain resistance and improve limb-salvage outcomes.


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