

Original Research Article

DIABETIC FOOT DISEASE: CLINICAL PRESENTATION, CULTURE PROFILE AND LINKS TO GLYCAEMIC CONTROL — A SINGLE-CENTER DESCRIPTIVE ANALYSIS

Naila Kunnummal¹, Ranjith Mannarakkal², Yousef Hisham²

¹Assistant Professor, Department of General Surgery, MES Medical College, Perinthalmanna, Kerala, india

²Associate Professor, Department of General Surgery, MES Medical College, Perinthalmanna, Kerala, india

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Corresponding Author:

Dr. Yousef Hisham,

Associate Professor, Department of General Surgery, MES Medical College, Perinthalmanna, Kerala, india.
Email: yousefhishamchakeeri@yahoo.in

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ABSTRACT

Background: Diabetic foot infection is common and still drives preventable lower-limb amputations. The first antibiotic and wound plan should be anchored to local culture and susceptibility data in day-to-day care. Tightening glucose control supports granulation, dampens recurrent infection, and improves the odds of healing. We set out to describe the clinical picture, culture profile, and antimicrobial susceptibility among adults admitted with diabetic foot disease at a tertiary hospital, and to examine whether admission glycemic measures were linked to in-hospital outcomes.

Materials and Methods: We ran a descriptive, hospital-based study over 12 months and enrolled 138 consecutive adults with diabetic foot infection (Wagner grade $\geq$ I). We captured demographics, comorbidities, ulcer features, random blood sugar (RBS), and HbA1c. Bacterial identification and antimicrobial susceptibility testing were performed using the Kirby–Bauer disc diffusion method. Associations between clinical and microbiological variables were analyzed using Fisher’s exact test.

Results: The largest age band was 50–69 years (64.5%); 68.1% were men. Ulcer (47.8%) was the most typical presentation, and Wagner grade II predominated (74.6%). Culture yielded a single organism in 67.4%, polymicrobial growth in 21.0%, and no growth in 11.6%; Gram-negative isolates overall were 55.8%. The top microorganisms were *Staphylococcus aureus* (26.1%), *Klebsiella* spp. (25.4%), *Pseudomonas* spp. (20.3%) and *Escherichia coli* (20.3%). Piperacillin–tazobactam showed notable activity against *E. coli* (57.1%) and *Pseudomonas* (64.3%); amikacin against *Pseudomonas* (71.4%); meropenem against *Pseudomonas* (85.7%); and linezolid against MRSA (100%), while ampicillin was broadly ineffective. Polymicrobial growth correlated with an extended hospital stay ($p < 0.001$); prolonged stay was also associated with *E. coli* ($p = 0.005$), *Pseudomonas* ($p = 0.001$), *Klebsiella* ($p = 0.003$), *S. aureus* ($p = 0.046$), and MRSA ($p = 0.040$). HbA1c and RBS were not significantly associated with ulcer duration or length of stay.

Conclusion: In this cohort, diabetic foot infections were predominantly Gram-negative, with *Staphylococcus aureus* as the leading single isolate. Empiric therapy should provide coverage for *S. aureus* and local Enterobacterales/*Pseudomonas*, followed by early culture-guided de-escalation. Polymicrobial and pathogen-specific profiles, rather than admission glycaemia, were the strongest predictors of prolonged hospitalization.

Keywords: Diabetic foot; microbiology; antimicrobial susceptibility; glycemic control; Wagner grade; India.

INTRODUCTION

Diabetic foot disease (DFD) encompassing ulceration, soft-tissue infection, osteomyelitis, and gangrene on the background of neuropathy and/or ischaemias among the most burdensome complications of diabetes in low- and middle-income settings.^[1] Many patients with diabetic foot disease reach the hospital late, often with deep soft-tissue infection that needs staged procedures or prolonged wound care. These realities impair quality of life and add substantially to care costs.^[2] At the population level, the steady rise in diabetes in India and worldwide means more people are at risk for ulceration and infection, despite better awareness and preventive programs.^[3]

The microbiology of diabetic foot infection shifts with stage. Early or superficial disease is more often monomicrobial; chronic or long-standing ulcers tend to be polymicrobial with a broader mix of pathogens.^[4] Across Indian tertiary-care series, the dominant isolates have included *Staphylococcus aureus*, *Enterobacteriales* (especially *Klebsiella* and *Escherichia coli*), and *Pseudomonas aeruginosa*, with variable contributions from streptococci and enterococci. Importantly, organism distributions and resistance patterns are contextual and dynamic shaped by prior antibiotic exposures, wound chronicity, procedure environments, and local infection-control practices.^[5] Periodic, centre-specific updates therefore remain essential to guide empiric therapy and antimicrobial stewardship.

Glycaemic control is mechanistically linked to infection defence and wound healing through effects on neutrophil function, cytokine signalling, angiogenesis, and extracellular matrix turnover.^[6] Yet, in routine inpatient care, the magnitude of association between admission glycaemic indices (random blood sugar, HbA1c) and short-term outcomes such as length of stay is uncertain.^[7] In many real-world cohorts, organism burden and mix, adequacy and timing of debridement, and the need for vascular or reconstructive procedures appear to exert more proximate effects on hospital trajectory than chronic glycaemic ‘memory’.^[8]

Against this clinical and biological backdrop, we undertook a single-centre descriptive analysis of patients admitted with DFD to a government tertiary hospital in northern Kerala. We aimed to characterise clinical presentation and procedures, delineate culture profile and antimicrobial susceptibility to commonly used agents, and examine links among microbial patterns, admission glycaemic indices, and in-hospital outcomes. By reporting practice-proximal data from our service, we seek to inform initial antimicrobial choices, reinforce the rationale for early source control and de-escalation, and clarify the relative role of glycaemic indices in near-term outcomes.

MATERIALS AND METHODS

Study design and setting: We conducted a hospital-based, descriptive study at Government Medical College, Kannur, Kerala, India, over a continuous 12-month. The general surgery service receives referrals from primary and secondary facilities, creating a real-world DFD case-mix. The institutional ethics committee approved the protocol (223/2018/ACME; 05 February 2018), and written informed consent was obtained from all participants. **Participants and data:** Consecutive adults (≥ 18 years) with type 2 diabetes and foot infection of Wagner grade I or higher were eligible. We excluded non-diabetic varicose ulcers. A structured proforma captured demographics, comorbidities, risk factors, presentation, ulcer site and duration, Wagner grade, random blood sugar (RBS), and glycated haemoglobin (HbA1c). Peri-admission RBS and categorical HbA1c bands (good 6–7%, fair 7.1–8%, poor 8.1–9%, bad $>9\%$) were used. Procedures performed and length of stay were abstracted from operative and discharge records.

Microbiology: After cleansing and debridement, swab or tissue specimens were obtained for Gram stain, culture, and antimicrobial susceptibility testing by Kirby–Bauer disc diffusion per Clinical and Laboratory Standards Institute criteria. The local panel included penicillins/ β -lactam- β -lactamase inhibitors, cephalosporins, fluoroquinolones, aminoglycosides, lincosamides, glycopeptides, oxazolidinones, carbapenems, and polymyxins. Culture outcome was recorded as single-organism growth, polymicrobial (≥ 2 organisms), or no growth.

Outcomes and analysis: Clinical profiles, organism distribution, and susceptibility patterns were primary descriptive outcomes. Secondary outcomes examined associations between glycaemic indices and ulcer duration or length of stay, and between growth pattern or specific organisms and length of stay. Associations were assessed using Fisher’s exact test (two-sided $\alpha=0.05$) with conventional software; multivariable modelling was not pre-specified.

RESULTS

Cohort and presentation: Among 138 admissions, most patients were 50–69 years (89, 64.5%), with 31 (22.5%) aged ≥ 70 and 18 (13.0%) younger than 50; men predominated (94, 68.1%). Ulcer was the most typical presentation (66, 47.8%), followed by abscess (35, 25.4%), gangrene (26, 18.8%), and cellulitis (11, 8.0%). On Wagner grading, grade II lesions were most frequent (103, 74.6%), while grade I accounted for 8 cases (5.8%) and advanced disease ($\geq$ grade III) for 27 cases (19.6%).

Glycemic indices and procedures: Among 138 admissions, RBS at presentation was ≤ 200 mg/dL in 79 (57.2%), 201–300 mg/dL in 35 (25.4%), and ≥ 301 mg/dL in 24 (17.4%). By longer-term control, HbA1c bands were good (6–7%): 28 (20.3%), fair

(7.1–8%): 62 (44.9%), poor (8.1–9%): 22 (15.9%), and bad (>9%): 26 (18.8%). The procedural pathway reflected limb-salvage practice: debridement was most frequent (58, 42.0%), followed by incision & drainage (35, 25.4%), toe disarticulation (22, 15.9%),

dressings only (12, 8.7%), and other procedures like split-skin grafting or below-knee amputation (11, 8.0%). Length of stay was <10 days in 57 (41.3%), 10–20 days in 48 (34.8%), and >20 days in 33 (23.9%).

Table 1: Cohort and presentation

Domain	Item	n (%)
Demographics	Age <50	18 (13.0%)
Demographics	Age 60–69	89 (64.5%)
Demographics	Age ≥70	31 (22.5%)
Demographics	Sex: Male	94 (68.1%)
Demographics	Sex: Female	44 (31.9%)
Presentation	Ulcer	66 (47.8%)
Presentation	Abscess	35 (25.4%)
Presentation	Gangrene	26 (18.8%)
Presentation	Cellulitis	11 (8.0%)
Severity (Wagner)	Grade I	8 (5.8%)
Severity (Wagner)	Grade II	103 (74.6%)
Severity (Wagner)	≥Grade III (III–V)	27 (19.6%)

Table 2: Glycemic indices

Domain	Item	n (%)
Glycaemia (RBS)	≤200 mg/dL	79 (57.2%)
Glycaemia (RBS)	201–300 mg/dL	35 (25.4%)
Glycaemia (RBS)	≥301 mg/dL	24 (17.4%)
Glycaemia (HbA1c)	Good (6–7%)	28 (20.3%)
Glycaemia (HbA1c)	Fair (7.1–8%)	62 (44.9%)
Glycaemia (HbA1c)	Poor (8.1–9%)	22 (15.9%)
Glycaemia (HbA1c)	Bad (>9%)	26 (18.8%)
Procedures	Debridement	58 (42.0%)
Procedures	Incision & drainage	35 (25.4%)
Procedures	Toe disarticulation	22 (15.9%)
Procedures	Dressings only	12 (8.7%)
Procedures	Other (SSG/BKA)	11 (8.0%)
Hospital stays	<10 days	57 (41.3%)
Hospital stays	10–20 days	48 (34.8%)
Hospital stays	>20 days	33 (23.9%)

Culture and susceptibility: Among 138 admissions, cultures yielded growth in 122 (88.4%): single-organism growth in 94 (68.1%), polymicrobial growth in 28 (20.3%), and no growth in 16 (11.6%). The most frequent isolates were *Staphylococcus aureus* (26.1% of the cohort), *Klebsiella* spp. (25.4%), *Pseudomonas* spp. (20.3%), and *Escherichia coli* (20.3%). On susceptibility testing, *E. coli* showed the highest activity with meropenem 75.0%, followed by piperacillin–tazobactam 57.1% and amikacin 53.6%; *Pseudomonas* spp. were most susceptible to meropenem 85.7%, amikacin 71.4%, and piperacillin–tazobactam 64.3%. *Klebsiella* spp. demonstrated modest activity to meropenem 45.7%, amikacin 37.1%, and piperacillin–tazobactam 34.3%.

All MRSA isolates were linezolid-susceptible (100%).

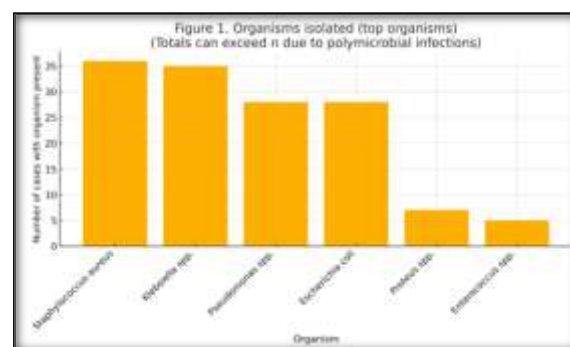


Table 3: Culture and susceptibility

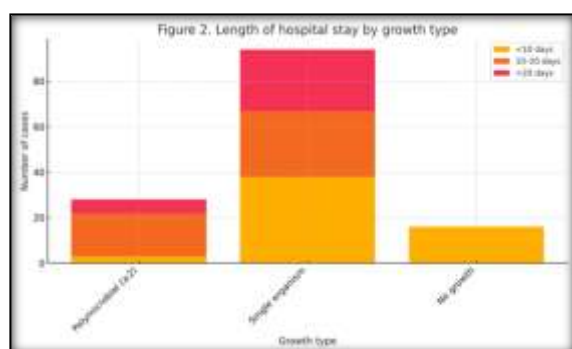
Domain	Item	Value
Growth pattern	Single organism	94 (68.1%)
Growth pattern	Polymicrobial (≥2)	28 (20.3%)
Growth pattern	No growth	16 (11.6%)
Top organisms	<i>Staphylococcus aureus</i>	26.1% of cohort
Top organisms	<i>Klebsiella</i> spp.	25.4% of cohort
Top organisms	<i>Pseudomonas</i> spp.	20.3% of cohort
Top organisms	<i>Escherichia coli</i>	20.3% of cohort
Selected susceptibility	<i>Escherichia coli</i>	Meropenem 75.0%; Piperacillin–tazobactam 57.1%; Amikacin 53.6%
Selected susceptibility	<i>Pseudomonas</i> spp.	Meropenem 85.7%; Amikacin 71.4%; Piperacillin–tazobactam 64.3%
Selected susceptibility	<i>Klebsiella</i> spp.	Meropenem 45.7%; Amikacin 37.1%; Piperacillin–tazobactam 34.3%
Selected susceptibility	MRSA	Linezolid 100.0%

Associations: Polymicrobial growth correlated with a more extended hospital stay ($p<0.001$). Organism-specific associations with prolonged stay included *E. coli* ($p=0.005$), *Pseudomonas* ($p=0.001$), *Klebsiella* ($p=0.003$), *S. aureus* ($p=0.046$), and

MRSA ($p=0.040$). Longer ulcer duration was associated with polymicrobial growth ($p=0.006$). Neither admission RBS nor HbA1c category was significantly associated with ulcer duration or length of stay.

Table 4: Key bivariate associations

Comparison	p-value	Interpretation
Polymicrobial growth vs length of stay	<0.001	Significantly longer stays with polymicrobial
Ulcer duration vs polymicrobial growth	0.006	Significant; longer ulcer ↔ polymicrobial
Gram-negative infection vs length of stay	<0.001	Significant
Presence of <i>E. coli</i> vs length of stay	0.005	Significant
Presence of <i>Pseudomonas</i> vs length of stay	0.001	Significant
Presence of <i>Klebsiella</i> vs length of stay	0.003	Significant
Presence of <i>S. aureus</i> vs length of stay	0.046	Significant
Presence of MRSA vs length of stay	0.040	Significant
RBS/HbA1c vs ulcer duration	0.820 / 0.155	Not significant
RBS/HbA1c vs length of stay	0.560 / 0.706	Not significant



DISCUSSION

In this tertiary-care study, most patients presented with clinically advanced but still potentially salvageable disease, reflected by the predominance of Wagner grade II ulcers and a treatment profile that favored debridement over major amputation. Microbiological cultures showed overall dominance of Gram-negative bacilli, although *Staphylococcus aureus* remained the most frequent single isolate.^[9] Polymicrobial growth and specific organism patterns, particularly *Pseudomonas aeruginosa*, *Klebsiella* species, *Escherichia coli*, and *S. aureus*/MRSA were linked to extended hospital stays. Admission glycemic measures, however, showed no independent association with ulcer chronicity or duration of admission in unadjusted analyses.^[10] The flora profile mirrors patterns reported from Indian tertiary centres, where chronicity, prior antibiotic exposure, and environmental contact favour Gram-negative recovery while *S. aureus* persists as a dominant pathogen.^[11] Polymicrobial growth is linked to more extended stay, which is clinically plausible, and a longer interval to culture-guided narrowing of therapy.^[12] The association between specific organisms and prolonged stay may reflect their intrinsic resistance potential, biofilm propensity, or the complexity of required source control (e.g., tendon or bone involvement). While chronic hyperglycaemia impairs host defences and wound healing biology, the absence of a

significant signal for RBS or HbA1c on length of stay likely denotes the dominance of immediate surgical and microbiological determinants in the inpatient phase.^[13] Aggressive inpatient glucose optimisation, which is standard practice, may also attenuate the influence of baseline indices on short-term outcomes.^[14] These findings emphasise that, for hospital courses, early source control and prompt de-escalation may matter more than admission to the glycemic category, even as long-term optimization remains essential for prevention and healing.

Our data argue for initial empiric coverage of *S. aureus* (with MRSA consideration according to risk) and local Gram-negatives, including *Pseudomonas*, where clinically indicated, with early de-escalation within 48–72 hours once cultures and clinical response allow.^[15] Ampicillin is generally unsuitable for empirical therapy in this context. For moderate to severe infections, piperacillin–tazobactam provides a practical first-line option, with carbapenems reserved for resistant strains or cases complicated by deep-seated sepsis. Linezolid is a valuable option when MRSA or *Enterococcus* is suspected or confirmed. Building in a scheduled “antibiotic time-out” at 48–72 hours help clinicians narrow therapy and shorten duration once cultures return, balancing effectiveness with lower toxicity and resistance pressure.

The predominance of Wagner grade II in our cohort points to missed chances for prevention and earlier referral. Structured education about daily self-inspection, protective footwear, and early review for minor trauma plus ready access to off-loading and wound-care services can curb progression and reduce amputations.^[16] Equally important are coordinated pathways that bring together surgery, endocrinology, microbiology, podiatry and vascular teams.

Consecutive enrolment and organism-level susceptibility data improve our findings' internal validity and real-world usefulness. Even so, some limits apply. Being a single-center study with frequent use of swabs rather than deep-tissue samples, we likely under-detected anaerobes. The absence of routine perfusion imaging and minimal adjustment for confounders (e.g., ulcer duration,

perfusion status, organism mix) restricts causal claims. It may underestimate the role of anaerobes in advanced infections.

Future work should use prospective cohort designs with deep-tissue/aspirate sampling, dedicated anaerobic culture, routine perfusion assessment, and integrated vascular interventions. Risk-adjusted models that account for neuropathy, ischemia, prior antibiotics, and biofilm-forming organisms would clarify outcome drivers. Regularly updated center-specific antibiograms should guide empiric therapy, and stewardship should prioritize early de-escalation and disciplined treatment duration.

CONCLUSION

In our study, Gram-negative organisms accounted for most infections overall, while *Staphylococcus aureus* remained the single most common isolate. Polymicrobial growth and specific pathogens like *Escherichia coli*, *Klebsiella* spp., *Pseudomonas aeruginosa*, and *S. aureus*/MRSA were linked to extended hospital stays. Empiric therapy should cover *S. aureus* plus the predominant local Gram-negatives. Add antipseudomonal coverage when clinically indicated, and step down promptly once cultures are available. Prevention and well-coordinated multidisciplinary care remain central to avoiding complications and amputation.

Ethics approval: The study received approval from the Institutional Ethics and Research Committee, Government Medical College, Kannur (Ref. No. 223/2018/ACME; meeting held on 05 February 2018). Before enrolment, written informed consent was obtained from all participants.

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