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Clinico-Microbiological Profile and Treatment Outcomes of Patients with Diabetic Foot Ulcers (DFUs) in a Tertiary Care Hospital - A Prospective Study

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ABSTRACT

Background: Diabetic foot ulcer (DFU) remains one of the most serious complications of diabetes mellitus, contributing significantly to morbidity, lower-limb amputation, and mortality. A thorough understanding of the causes and appropriate management of diabetic foot ulceration is crucial to preventing lower-extremity amputations.
Objectives: To assess the clinico-microbiological profile, socio demographic characteristics, and treatment outcomes of patients presenting with DFU in a tertiary care hospital.
Methods: This prospective hospital-based study, involving 50 consecutively enrolled patients with DFU. Socio demographic and clinical data were recorded. Wound samples were collected aseptically and processed for Gram staining, culture, and antibiotic susceptibility testing. Patients were managed as per ulcer severity, conservative care, debridement, disarticulation, or amputation and followed until discharge.
Results: The study included 27 males (54%) and 23 females (46%), with most patients aged 50–64 years. Type 2 diabetes accounted for 96% of cases, and 66% had diabetes for more than eight years. Peripheral neuropathy was present in 56%, hypertension in 42%, and peripheral arterial disease in 28%. Microbial growth was detected in 90% of cultures, with 58% monomicrobial and 32% polymicrobial infections. *Staphylococcus aureus* and *Klebsiella pneumoniae* were the predominant isolates. Overall, 84% of patients achieved healing, while 26% required major or minor amputations.
Conclusion: DFU primarily affect patients with long-standing, poorly controlled diabetes. Early-stage ulcers showed good healing outcomes, whereas advanced grades contributed to a 26% amputation rate. Strengthening early detection, foot-care practices, glycaemic control, and culture-guided therapy is essential to reduce complications and improve patient outcomes.

Keywords: Diabetic foot ulcer; Microbial profile; Antibiotic sensitivity; Wagner grading; Amputation.

INTRODUCTION

India ranks second after China in the global diabetes burden.[1] According to International Diabetes Federation, one out of every seven diabetic adults worldwide reside in India, and one in every third household has diabetic patients. In 2021, India recorded 74.9 million individuals with diabetes, a figure expected to escalate to 124.9 million by 2045.[2]

Diabetic foot is defined as infection, ulceration or destruction of tissues of the foot associated with neuropathy and/ or Peripheral Arterial Disease in the lower extremity of a person with diabetes mellitus.[3] The classic triad of neuropathy, ischemia and infection characterize the diabetic foot. Diabetic foot is mainly classified into three types. Neuropathic foot in which neuropathy dominates, ischemic foot in which occlusive vascular disease dominates and Neuro- ischemic in which both ischemic and neuropathic changes are seen.[4]

Diabetic foot ulcer is a serious and common complication of diabetes mellitus that often result in a diminished quality of life.[5] Life time risk of developing a foot ulcer in diabetics is 15%–25%.[6]

According to Global estimates (2010–2020), incidence of diabetes-related amputations (both minor and major), is increasing.[7] In diabetic people, the risk of amputation is attributable to the combination of peripheral neuropathy and infection stemming from diabetes and the presence of impaired arterial flow due to peripheral artery disease.[8] Higher re-amputation and mortality rates are seen in patients with major amputation, repeat amputation, or ischemic cardiomyopathy.[9] Diabetic foot resulting in limb amputation has major impact on an individual, as it not only causes physical deformity, but also leads to economic dependency and social deprivement.[10]

In a diabetic patient, poor treatment adherence leading to high blood sugar, diminished immunity, lack of knowledge and negligence on foot care increases the risk of diabetic foot ulcers.[11] The majority of foot-care activities—such as washing and drying the feet, applying moisturizer, routinely trimming nails, checking the feet with a mirror for ulcers, looking for ingrown nails, choosing appropriate footwear, avoiding walking barefoot, and regularly consulting a healthcare provider—are very important in the prevention of foot ulcers.[12]

A thorough understanding of the causes and management of diabetic foot ulceration is crucial to preventing lower-extremity amputations. Assessing key factors, patient demographics, duration of diabetes, comorbidities, treatment adherence, foot-care awareness, ulcer grade, and bacteriological profile can significantly improve management and reduce severe complications, including amputation, re-amputation, and mortality.

Objectives of the Study

1. To assess the clinico-microbiological profile of patients with diabetic foot ulcers.
2. To evaluate the socio demographic profile of diabetic patients presenting with diabetic foot ulcers.
3. To examine the treatment outcomes associated with diabetic foot ulcers.

MATERIALS & METHODS

This hospital-based prospective study was conducted in the Department of General Surgery at Hassan Institute of Medical Sciences (HIMS), Teaching Hospital, Hassan, during the year 2025. All consecutive patients admitted with diabetic foot ulcers during the study period were screened for eligibility. The study included both male and female patients presenting with foot ulcers who were either known diabetics or newly diagnosed during admission based on WHO criteria (FBS ≥126 mg/dL and/or 2-hour PPBS ≥200 mg/dL).[13] Patients not willing to participate in the study were excluded from the study. The sample size was estimated using the formula:

$n = Z^2 p q / d^2$, where: Z = 1.96 (standard normal deviate at 95% confidence level). p = 0.73, proportion of bacterial growth reported

in a previous study, [14] q=0.27, d = 18% of p, relative precision, The calculated sample size was 43. After adding a 5% attrition rate, the final sample size was 45and the final sample size was rounded off to 50.

METHODOLOGY

After obtaining approval from the Institutional Ethics Committee and written informed consent from all participants, data were collected using a semi- structured proforma. Wound samples were obtained and sent to the Microbiology Laboratory for culture and sensitivity analysis.

Data Collection

A semi-structured proforma was used to gather socio demographic and clinical data. Information collected included age, sex, occupation, place of residence, family history, type and duration of diabetes, prior treatment history, foot-care practices, smoking and alcohol habits, and comorbidities such as hypertension. In addition, details about ulcer duration, presence of intermittent claudication, and neuropathic symptoms were recorded.

Clinical Examination of Ulcer and assessment of Comorbidities and Diabetes-Related Complications

Ulcers were clinically assessed for site, size, and depth, and graded according to Wagner's Classification.[15] Both feet were examined for callus formation and any associated abnormalities. Local signs such as foul smell, local rise of temperature, discharge, and peri-wound discoloration were noted.

Patients were examined for associated comorbidities and complications of diabetes, including hypertension, peripheral vascular disease, neuropathy, nephropathy, and retinopathy.

Peripheral circulation was assessed using Arterial Doppler of the affected limb.

Peripheral neuropathy was evaluated by testing:

- Pain sensation: using pinprick and recorded as present or absent
- Vibration sense: Using a 128 Hz tuning fork on the medial and lateral malleoli
- Pressure sensation: Using a 10 g monofilament at standard plantar sites
- Ankle (Achilles) reflex: Elicited using a standard percussion hammer

Neuropathy severity was quantified using the Neurological Disability Score (NDS) for each foot.[16]

Sample Collection for Bacteriological Analysis

Samples were collected prior to initiating antibiotic therapy. Specimens such as pus, wound exudate, or tissue biopsy were obtained. The ulcer surface was cleansed with sterile normal saline, and superficial debris was removed using a sterile scalpel. Two sterile swabs were obtained from the deeper portion of the ulcer using firm, rotatory motion:

- One swab for Gram staining
- One swab for culture and sensitivity

Samples were immediately transported to the Microbiology Department for processing.

Routine and specific investigations were carried out for all patients. These included:

- Complete blood count (CBC), blood grouping and Rh typing, and bleeding time/clotting time
- Random blood sugar (RBS), fasting blood sugar (FBS), post-prandial blood sugar (PPBS), and glycated haemoglobin (HbA1c)
- Screening tests for HIV and HBsAg
- Blood urea and serum creatinine
- Serum electrolytes
- Serum albumin and bilirubin
- Urine analysis for albumin, sugar, and microscopy
- Arterial Doppler study of the affected limb to assess peripheral circulation

Treatment and Follow-up

Patients received appropriate treatment based on ulcer severity; conservative management, debridement, debridement with disarticulation, or amputation.

Treatment response was monitored and documented until discharge.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics (29.0.2). Descriptive statistics were used to summarise all study variables. Categorical variables—such as sex distribution, comorbidities, ulcer type, Wagner grade, microbial profile, and antibiotic sensitivity patterns etc, were presented as proportions and percentages. 95% confidence intervals (CI) were computed using the Wilson score formula. Graphical representations such as bar charts and pie charts were used to illustrate the key findings.

RESULT

Table 1: Socio-demographic Characteristics of Study Participants with Diabetic Foot Ulcer (DFU).

Variables	Female (n=23) No. (%)	Male (n=27) No. (%)	Total (n=50) No. (%)
Age (years)			
35–49	4 (17.4)	4 (14.8)	8 (16)
50–64	9 (39.1)	12 (44.4)	21 (42)
65–79	7 (30.4)	8 (29.6)	15 (30)
≥80	3 (13)	3 (11.1)	6 (12)
Smoking			
Yes	0 (0)	10 (37)	10 (20)
No	23 (100)	17 (63)	40 (80)
Occupation			
Homemaker	9 (39.1)	—	9 (18)
Agriculture	4 (17.3)	10 (37)	14 (28)
Other Manual Labourer	2 (8.7)	3 (11.1)	5 (10)
Vendor	3 (13)	3 (11.1)	6 (12)
Sedentary	5 (21.7)	4 (14.8)	9 (18)
Others -Clerical/Teacher/Shop keeper etc.	—	7 (25.9)	7 (14)

The study included 50 patients, 23 (46%) females and 27 (54%) males. The majority were aged 50–64 years (42%), followed by 65–79 years (30%). Smoking was reported in 10 males (37%) and none of the females. Most participants were engaged in agriculture (28%) or homemaking (18%), while others were involved in sedentary work (18%), vendors (12%), manual labourer (10%), or clerical/teaching/shopkeeping roles (14%). (Table 1)

Table 2: Clinical Profile of Study Participants with Diabetic Foot Ulcer (DFU).

Variables	Female (n=23) No. (%)	Male (n=27) No. (%)	Total (n=50) No. (%)
New or Known Case			
Newly diagnosed	4 (17.4)	5 (18.5)	9 (18)
Known case	19 (82.6)	22 (81.5)	41 (82)
Type of DM			
Type 1	1 (4.3)	1 (3.7)	2 (4)
Type 2	22 (95.7)	26 (96.3)	48 (96)
Duration of Diabetes			
Low (1–4 yrs)	2 (8.7)	3 (11.1)	5 (10)
Medium (5–7 yrs)	6 (26.1)	6 (22.2)	12 (24)
High (8–15 yrs)	15 (65.2)	18 (66.7)	33 (66)
Diabetes Treatment			
Oral Hypoglycaemics	16 (69.6)	19 (70.4)	35 (70)
Insulin	5 (21.7)	5 (18.5)	10 (20)
Both	2 (8.7)	3 (11.1)	5 (10)
Treatment Adherence			
Yes	9 (39.1)	11 (40.7)	20 (40)

No	14 (60.9)	16 (59.3)	30 (60)
Duration of Ulcer			
<3 months	11 (47.8)	14 (51.9)	25 (50)
3–6 months	8 (34.8)	9 (33.3)	17 (34)
6 months–1 year	4 (17.4)	4 (14.8)	8 (16)
Type of Ulcer			
Neuropathic	9 (39.1)	10 (37)	19 (38)
Ischaemic	3 (13.0)	7 (25.9)	10(20)
Neuro ischaemic	4 (17.4)	5 (18.5)	9(18)
Not Classified	7 (30.4)	5 (18.5)	12 (24)
HbA1c (%)			
<7 (Good control)	5 (21.7)	6 (22.2)	11 (22)
7–9 (Moderate control)	8 (34.8)	9 (33.3)	17 (34)
>9 (Poor control)	10 (43.5)	12 (44.4)	22 (44)

Most patients were known cases of diabetes (82%) and had type 2 diabetes (96%). The majority had a long duration of diabetes (8–15 years, 66%). Oral hypoglycaemics were the most common treatment (70%), while 40% reported adherence to treatment. Half of the patients had ulcers of less than 3 months' duration. Glycaemic control was suboptimal in many, with 44% having poor control (HbA1c >9%) and 22% achieving good control (HbA1c <7%). (Table 2)

Table 3: Prevalence of Comorbidities in Patients with Diabetic Foot Ulcers (DFU)

Variables	Female (n=23) No. (%)	Male (n=27) No. (%)	Total (n=50) No. (%)
Hypertension (HTN)			
Yes	9 (39.1)	12 (44.4)	21 (42)
No	14 (60.9)	15 (55.6)	29 (58)
Peripheral Arterial Disease (PAD)			
Yes	6 (26.1)	8 (29.6)	14 (28)
No	17 (73.9)	19 (70.4)	36 (72)
Peripheral Neuropathy (PN)			
Yes	13 (56.5)	15 (55.6)	28 (56)
No	10 (43.5)	12 (44.4)	22 (44)
Grading of PN			
No Neuropathy	10 (43.5)	12 (44.4)	22 (44)
Mild Neuropathy	10 (43.5)	9 (33.3)	19 (38)
Moderate Neuropathy	2 (8.7)	4 (14.8)	6 (12)
Severe Neuropathy	1 (4.3)	2 (7.4)	3 (6)

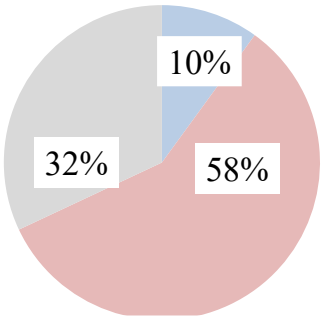
Hypertension was present in 42% of patients, peripheral arterial disease in 28%, and peripheral neuropathy in 56%. The prevalence of these comorbidities was comparable between males and female. (Table 3)

Table 4: Causes of Diabetic Foot Ulcers among Study Participants.

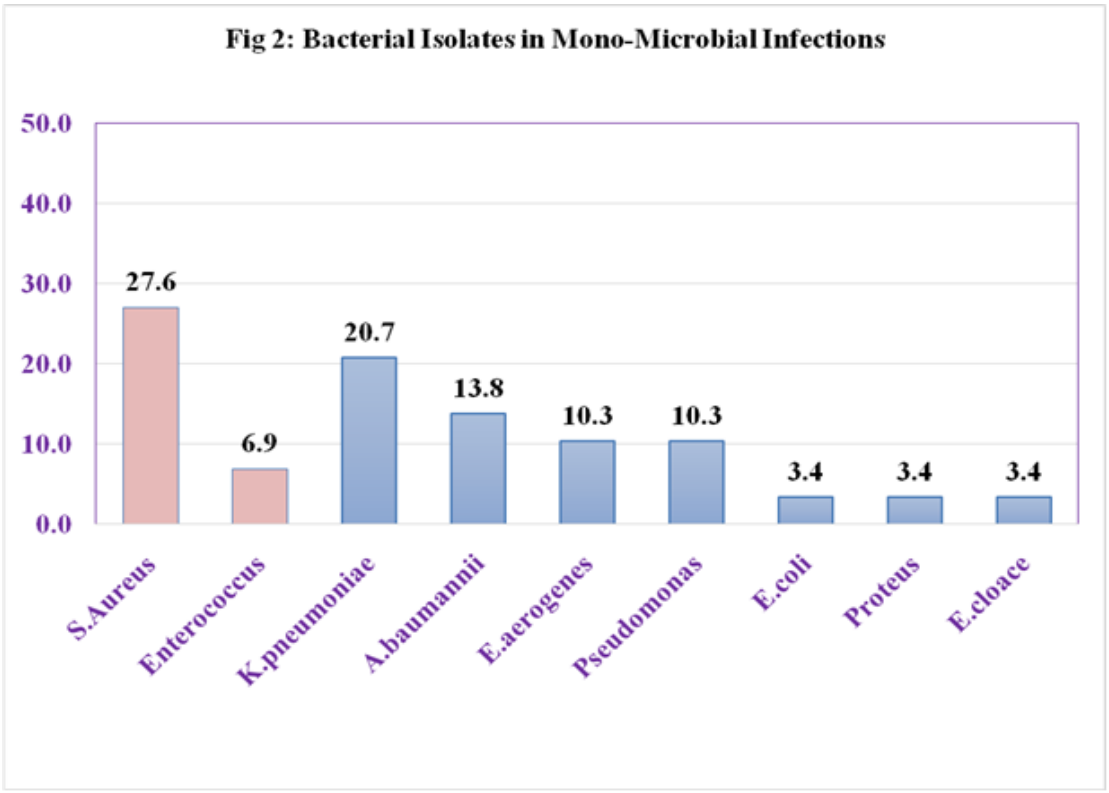
Causes	Female (n=23) No. (%)	Male (n=27) No. (%)	Total(n=50) No. (%)
Unknown	3 (13)	5 (18.5)	8 (16)
Swelling to Ulcer	2 (8.7)	4 (14.8)	6 (12)
Trauma	3 (13)	5 (18.5)	8 (16)
Improper Footwear	5 (21.7)	10 (37)	15 (30)
Boil	1 (4.3)	3 (11.1)	4 (8)
Previous Amputation	2 (8.7)	4 (14.8)	6 (12)
Barefoot	7 (30.4)	5 (18.5)	12 (24)
Thermal	3 (13)	1 (3.7)	4 (8)
In-growing Toenail	1 (4.3)	3 (11.1)	4 (8)
Self-Removal of Callus	0 (0)	4 (14.8)	4 (8)
Total	**23 (100)**	**27 (100)**	**50 (100)**

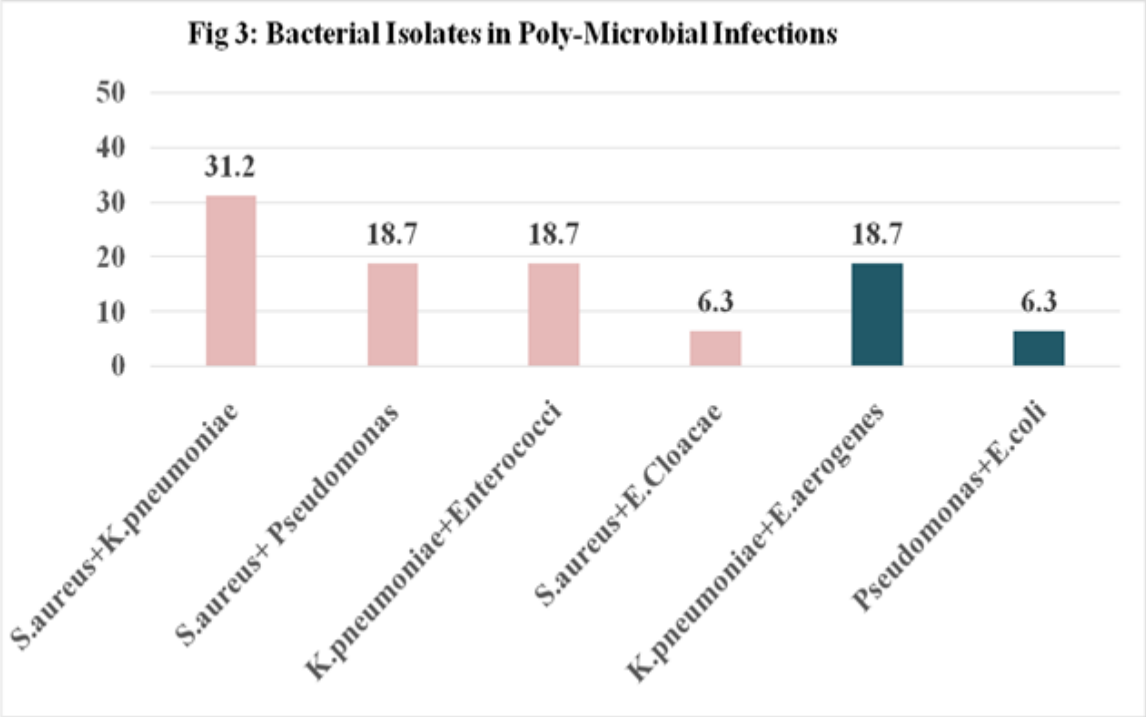
The most common causes of ulcers were improper footwear (30%) and barefoot walking (24%). Other causes included trauma and unknown factors, as well as swelling leading to ulcer, previous amputation, boils, thermal injury, in-growing toenail, and self-removal of callus. Trauma, boils, and self-removal of callus were more frequent in males, whereas barefoot walking and thermal injuries were more common in females. (Table 4)

Fig 1: Distribution of microbial gr patterns in DFUs.



Pie chart showing the distribution of microbial growth in 50 DFU cultures. Of these cultures, 5 (10%; 95% CI: 4.4–21.4%) showed no growth, 29 (58%; 95% CI: 44.2–70.7%) were monomicrobial, and 16 (32%; 95% CI: 20.8–45.8%) were polymicrobial (Fig. 1).





The Simple bar graphs illustrate the distribution of monomicrobial and polymicrobial infections among the culture-positive samples. Of the 45 culture-positive cases (90% of total samples), 29 (64.4%) were monomicrobial, while 16 (35.6%) were polymicrobial infections. (Fig 2 & 3)

In monomicrobial infections, the most frequent isolates were Staphylococcus aureus (27.6%) and Klebsiella pneumoniae (20.7%), followed by Acinetobacter baumannii (13.8%), Enterobacter aerogenes, Pseudomonas spp., and Enterococcus spp., with E. coli, Proteus, and Enterobacter cloacae occurring less often. (Fig 2)

In polymicrobial infections, the most frequent combinations involved S. aureus and K. pneumoniae, often accompanied by other gram-negative organisms. (Fig 3)

Table 5: Antibiotic Sensitivity Profile of Most Frequently Encountered Organisms

Organisms	No (%)	Sensitivity
S. aureus -	17(37.8%))	85.7%: RIF, GEN 71.4%: Clindamycin, DOX, TETRA 57.2%: LZ, CX 28.6%: COT, ERY, LEVO 14.3%: AK, AT, CFM, CAZ, CPM, CTR, CIP, IMP, OX, PIP/TAZ, TOB
Klebsiella pneumoniae	17(37.8%)	100%: GEN, IMP 80%: AT, LEVO, TETRA 60%: AK, CAZ, CPM, CTR, CIP, DOX 40%: CX, COT 20%: PIP/TAZ, TOB
Pseudomonas spp.	7(15.6%)	100%: GEN, TOB 50%: AK, AT, CAZ, CPM, CIP, COT, LEVO, PIP/TAZ.

Note: n = 45 (Culture-positive samples) , AK – Amikacin, AT – Aztreonam, AMP – Ampicillin, AMC – Amoxicillin–Clavulanic acid, CX – Cefoxitin, CFM – Cefixime, CAZ – Ceftazidime, CPM – Cefepime, CTR – Ceftriaxone, COT – Cotrimoxazole, CIP – Ciprofloxacin, CLINd – Clindamycin, ERY – Erythromycin, DOX – Doxycycline, GEN – Gentamicin, IMP – Imipenem, LEVO – Levofloxacin, LZ – Linezolid, OX – Oxacillin, PIP/TAZ – Piperacillin–Tazobactam, RIF – Rifampicin, TETRA – Tetracycline, TOB – Tobramycin, VM – Vancomycin.

S. aureus showed highest (85.7%) susceptibility to rifampicin and gentamicin, followed by moderate sensitivity to clindamycin, doxycycline, and tetracycline, with low response to most β-lactams and fluoroquinolones. K. pneumoniae demonstrated complete (100%) sensitivity to gentamicin and imipenem, with moderate susceptibility to aminoglycosides, cephalosporins, and fluoroquinolones. Pseudomonas spp. showed excellent (100%) susceptibility to gentamicin and tobramycin, and about half of the isolates were sensitive to the remaining tested antibiotics. (Table 5)

Table 6: Antibiotic Sensitivity Profile of Less Frequently Encountered Organisms

Organism	No (%)	Qualitative Description of Sensitivity
Enterococci	5 (11.1)	Showed 100% sensitivity to Ampicillin (AMP), Linezolid (LZ), and Vancomycin (VM).
A. baumannii	4 (8.9)	66.7% sensitivity to AK, CX, IMP, TOB, and TETRA, 33.3% sensitivity to AT, CFM, CAZ, CIP, CPM, CTR, COT, GEN, LEVO, and PIT.
Enterobacter aerogenes	3 (6.7)	66.7% sensitivity to AT, CPM, and COT 33.3% sensitivity to AK, CX, CIP, IMP, LEVO, and PIP/TAZ.
E. coli	2 (4.4)	Both isolates (100%) were sensitive only to AK, AT, AMC, CX, GEN, IMP, TOB.
Enterobacter cloacae	2 (4.4)	Both isolates (100%) were sensitive to AK, AMC, CX, CFM, CAZ, CIP, CTR, GEN, IMP, PIP/TAZ
Proteus spp.	1 (2.2)	The single isolate (100%) was sensitive to almost all tested antibiotics, including AK, AT, AMC, CX, CFM, CAZ, CIP, CPM, CTR, GEN, IMP, LEVO, PIP/TAZ, TOB.

Note: n = 45 (Culture-positive samples)

Enterococci showed complete (100%) susceptibility to ampicillin, linezolid, and vancomycin. Acinetobacter baumannii demonstrated 66.7% sensitivity to amikacin, cefoxitin, imipenem, tobramycin, and tetracycline, while showing only 33.3% susceptibility to the remaining tested agents. Enterobacter aerogenes showed 66.7% sensitivity to aztreonam, cefepime, and cotrimoxazole, and 33.3% sensitivity to other antibiotics including aminoglycosides, fluoroquinolones, carbapenems, and piperacillin–tazobactam.

E. coli isolates exhibited uniform (100%) sensitivity to amikacin, aztreonam, amoxicillin–clavulanate, cefoxitin, gentamicin, imipenem, and tobramycin. Similarly, Enterobacter cloacae isolates were 100% sensitive to amikacin, amoxicillin–clavulanate, cefoxitin, cefixime, ceftazidime, ciprofloxacin, ceftriaxone, gentamicin, imipenem, and piperacillin–tazobactam. (Table 6)

Table 7: Clinical Course of DFU According to Wagner Classification.

Wagner Grade	Treatment	Outcome [No. (%)]		Total
		Healing	Non-Healing	No. (%)
Grade 0: No ulcer / pre-ulcer lesion	Not applicable	0 (0)	0 (0)	0 (0)
Grade 1: Superficial ulcer	Conservative	12 (85.7)	2 (14.3)	14 (28)
Grade 2: Ulcer extension to tendon/bone	Debridement	9 (90)	1 (10)	10 (20)
Grade 3: Deep ulcer with abscess/osteomyelitis	Toe Articulation	7 (70)	3 (30)	10 (20)
Grade 4: Gangrene of forefoot	Forefoot Amputation	2 (66.7)	1 (33.3)	3 (6)
Grade 5: Gangrene of entire foot	Below Knee Amputation	8 (88.9)	1 (11.1)	9 (18)
	Above Knee Amputation	4 (100)	0 (0)	4 (8)
Total		**42 (84)**	**8 (16)**	**50 (100)**

Superficial ulcers (Grade 1) were seen in 28% of cases and treated conservatively, while 20% each had ulcers extending to tendon/bone (Grade 2) and deep ulcers with abscess or osteomyelitis (Grade 3). Forefoot gangrene (Grade 4) occurred in 6%, and gangrene of the entire foot (Grade 5) required amputation, with 18% undergoing below-knee and 8% undergoing above-knee procedures, giving a total amputation rate of 26% (95% CI: 16.1%–39.0%). No patients had pre-ulcer lesions (Grade 0).

Overall, 42 out of 50 patients (84%; 95% CI: 71.5%–92.3%) healed, while 8 (16%; 95% CI: 8.9%–28.5%) did not, demonstrating that Wagner grading effectively guides management and predicts outcomes. These results highlight that early-stage ulcers respond well to conservative or minor surgical interventions, whereas advanced grades often require major amputation but still have favourable healing rates (Table 7)

DISCUSSION

Socio-Demographic and clinical Profile

The present study included 50 patients with diabetic foot ulcers (DFU), comprising 27 (54%) males and 23 (46%) females. The majority were aged 50–64 years (42%), followed by 65–79 years (30%). Smoking was reported in 10 males (37%) and none of the females. Most participants were engaged in agriculture (28%) or homemaking (18%), and 18% were involved in sedentary work.

Most patients had type 2 diabetes (96%), with 66% having diabetes for 8–15 years. Treatment adherence was observed in 40% of patients. Glycaemic control was suboptimal, with 44% having poor control (HbA1c >9%) and 22% achieving good control (<7%).

Comorbidities included hypertension (42%), peripheral arterial disease (28%), and peripheral neuropathy (56%). The most common causes of ulcers were improper footwear (30%) and barefoot walking (24%), while trauma, boils, and self-removal of callus were more frequent in males, and thermal injuries in females.

These findings align with previous reports: Shruthi et al. [17] observed 51.4% aged 55–64 years; Kala Yadhav and Abhirup et al. [14,18] reported older adults with long-standing diabetes and high prevalence of neuropathy; Chapparbandi et al. [19] noted male predominance with neuropathy and peripheral arterial disease; Anoop et al. [20] reported 74.7% males, most aged 58–67 years. Other studies also reported male predominance (64–76%), older age, long-standing diabetes, and suboptimal glycaemic control. [21-25] International studies similarly noted male predominance and older age. [26-28]

Microbial Growth Patterns

In the present study, 90% were culture-positive. Monomicrobial infections accounted for 64.4% (29/45), with Staphylococcus aureus (27.6%) and Klebsiella pneumoniae (20.7%) predominating. Polymicrobial infections (35.6%) commonly involved S. aureus and K. pneumoniae alongside other gram-negative organisms.

Previous studies reported similar findings: Shruthi et al. [17] observed 70.9% monomicrobial infections, mainly K. pneumoniae and S. aureus; Moghaddam et al. [29] reported 63.8% monomicrobial infections with S. aureus predominance; Kala Yadhav and Abhirup et al. [14,18] found 28–38% mixed growth, primarily Pseudomonas, Klebsiella, and S. aureus.

Other studies reported 74.8% monomicrobial infections, with Pseudomonas aeruginosa (29.5%) and S. aureus (16.6%) as most common. Internationally, Pemayun & Naibaho[26] reported P. aeruginosa and S. aureus predominance; Ahmad et al. [30] noted Pseudomonas spp. and MRSA as common pathogens; Anyim et al. [27] observed polymicrobial infections with anaerobes dominating; Hariftyani et al. [28] found E. coli most frequent; Aleem et al. [31] reported MRSA and Pseudomonas predominance; Goh et al. [32] described 53.3% polymicrobial infections; Murshed [33] noted S. aureus and Pseudomonas as major isolates.

These findings highlight significant variability, with both gram-positive and

gram-negative organisms contributing substantially to diabetic foot infections.

Antibiotic Sensitivity

S. aureus showed highest susceptibility to rifampicin and gentamicin (85.7%), moderate sensitivity to clindamycin, doxycycline, and tetracycline, and low susceptibility to β-lactams and fluoroquinolones. K. pneumoniae was fully susceptible to gentamicin and imipenem (100%), with moderate susceptibility to aminoglycosides, cephalosporins, and fluoroquinolones. Pseudomonas spp. was fully susceptible to gentamicin and tobramycin (100%) and partially sensitive to other antibiotics. Less common organisms (Enterococci, Acinetobacter baumannii, Enterobacter aerogenes, E. coli, and Enterobacter cloacae) showed variable sensitivity.

Previously published regional data have shown high levels of antibiotic resistance among isolates, underscoring the need for culture-guided antimicrobial therapy.[14,17] Other Indian and international studies confirmed susceptibility of gram-positive isolates to vancomycin and linezolid, and variable sensitivity of gram-negative isolates to aminoglycosides and carbapenems.[23,28,29] Khan et al. reported 27% MRSA and 100% ESBL strains, with gram-positive isolates fully sensitive to vancomycin and linezolid, and P. aeruginosa fully sensitive to amikacin, tobramycin, piperacillin–tazobactam, and ciprofloxacin.[34]

Treatment Outcome

Management in the present study was guided by Wagner grading, with early-stage ulcers managed conservatively or with limited surgical procedures, and advanced stages requiring major intervention. Conservative management was used for Grade 1 ulcers, while Grades 2 and 3 commonly required debridement or toe articulation. Advanced gangrenous ulcers (Grades 4 and 5) necessitated major amputation, resulting in an overall amputation rate of 26%.

Abhirup et al. documented 63 minor debridements, along with 16 forefoot amputations, one below-knee, and one above-knee amputation.[18] Similarly, Chapparbandi et al. reported a range of surgical interventions, including both minor procedures and major amputations.[19] Anoop et al [20] reported interventions based on ulcer severity without specific amputation rates. Other studies have reported variable amputation rates. Burande et al. observed a combined amputation rate of 50.9%, with 46% undergoing minor and 5% undergoing major amputations (54 out of 106 patients). [21] Similarly, Ramani et al. reported 12% minor and 8% major amputations in their cohort. [35] while international studies reported mild to moderate surgical interventions according to ulcer severity, with amputation rates ranging from 8–14.1%. [26,28]

CONCLUSION

This prospective study provides an in-depth understanding of the clinico-microbiological characteristics and treatment outcomes of diabetic foot ulcers in patients attending a tertiary care hospital. Most participants were older adults with long-standing, poorly controlled type 2 diabetes, and a high burden of neuropathy, peripheral arterial disease, and hypertension—factors that collectively predispose individuals to ulcer formation and impaired wound healing. Improper footwear, barefoot walking, and minor trauma emerged as frequent precipitating factors, highlighting the continued gaps in preventive foot-care practices among diabetic patients.

Microbiological evaluation revealed that 90% of ulcers were culture-positive, with monomicrobial infections being more common than polymicrobial. Staphylococcus aureus and Klebsiella pneumoniae were the predominant isolates, and the antibiotic sensitivity patterns underline the need for updated local antibiograms to guide effective empirical therapy. The observed distribution of organisms and varying levels of antibiotic

resistance reiterate the importance of rational antimicrobial stewardship.

Clinical outcomes were closely linked to ulcer severity at presentation. Early Wagner grades showed favourable healing with conservative or minor surgical interventions, whereas advanced ulcers often required major procedures. The study recorded an overall amputation rate of 26%, including both below-knee and above-knee amputations, underscoring the significant morbidity associated with late presentation and severe ulcer grades. Despite this, the majority of patients (84%) achieved healing by discharge, demonstrating that timely and appropriate management can still lead to positive outcomes.

Overall, these findings emphasize the critical role of early detection, glycaemic control, patient education on foot care, and prompt multidisciplinary management in reducing complications and preventing amputations. Strengthening community awareness, implementing routine foot screening in diabetic clinics, and enhancing adherence to treatment guidelines can substantially mitigate the burden of diabetic foot disease. Further studies with larger samples and analytical designs are recommended to expand on predictors of healing, microbial patterns, and long-term functional outcomes.

Limitations of the Study:

This single-centre study with a small sample size limits the generalizability of the findings to wider populations. Anaerobic and fungal cultures were not performed, which may have resulted in underestimation of the complete microbial spectrum involved in diabetic foot infections.

Declaration by Authors

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