

Original Research Article

TO STUDY THE MICROORGANISM ASSOCIATED WITH DIABETIC FOOT ULCER AND THEIR ANTIBIOTIC SENSITIVITY IN A RURALLY LOCATED TERTIARY CARE HOSPITAL

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Received : 29/07/2026
Received in revised form : 25/08/2026
Accepted : 08/09/2026

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DOI: 10.70034/ijmedph.2026.3.931

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 6046-6051

ABSTRACT

Background: Diabetic foot ulcers (DFUs) are highly susceptible to infections, which are a leading cause of non-traumatic lower extremity amputations. In rural Indian settings, environmental and occupational factors often shift the microbial etiology away from standard Western profiles. This study aimed to identify the bacteriological profile and antimicrobial susceptibility patterns of DFU infections in a rural tertiary care hospital to guide empirical therapy.

Materials and Methods: A cross-sectional observational study was conducted involving 110 diabetic patients presenting with foot ulcers. Deep tissue pus swabs were collected and processed for aerobic bacterial isolation. The isolated microorganisms were subjected to antimicrobial susceptibility testing by using the Kirby-Bauer disk diffusion method in accordance with Clinical and Laboratory Standards Institute (CLSI) guidelines. Of 110 patients, 68 (61.8%) were male and 42 (38.2%) were female, the mean age of patients was 53.4 years (Range 20-87 years).

Results: Out of 110 samples, 70 (63.6%) yielded positive microbial growth. The infection profile was overwhelmingly dominated by Gram-negative bacilli, accounting for 98.6% (n=69) of all isolates. *Pseudomonas aeruginosa* was the predominant pathogen (37.1%), followed by *Escherichia coli* (27.1%) and *Klebsiella pneumoniae* (25.7%). Inpatient admission was significantly associated with a higher rate of culture positivity compared to outpatient presentation (67.3% vs. 33.3%, $p = 0.041$). Antibigram analysis revealed alarming resistance to Ciprofloxacin across the major Gram-negative isolates, ranging from 46.2% to 63.2%. Conversely, Piperacillin-Tazobactam and Amikacin maintained excellent efficacy, demonstrating greater than 80% sensitivity across these primary pathogens.

Conclusion: Gram-negative organisms, predominantly *Pseudomonas aeruginosa*, govern the microbial landscape of diabetic foot infections in this rural tertiary setting. The high rate of fluoroquinolone resistance compromises standard empirical treatments. Routine pus culture is mandatory, and empirical therapy for severe infections should prioritize parenteral broad-spectrum agents like Piperacillin-Tazobactam to improve clinical outcomes and prevent amputations.

Keywords: Diabetic foot ulcer, *Pseudomonas aeruginosa*, Antimicrobial resistance, Antibigram, Rural tertiary care, Piperacillin-Tazobactam.

INTRODUCTION

Diabetic foot ulcers (DFU) are among the most severe and debilitating complications of diabetes mellitus, significantly impacting patient morbidity and quality of life.^[11] The development of an infection within a DFU represents a critical clinical milestone, as it exponentially increases the risk of hospitalization and non-traumatic lower extremity amputations.^[2,3] Effective management requires a multidisciplinary approach, emphasizing early diagnosis, surgical offloading, and timely administration of appropriate antimicrobial therapy.^[3,12] The microbiological profile of diabetic foot infections is highly complex and exhibits substantial geographical and demographic variations.^[14]

While Western literature frequently reports Gram-positive aerobic cocci, particularly *Staphylococcus aureus*, as the primary colonizing pathogens in DFUs,^[7] the microbiological profile in tropical and developing countries is markedly different. Studies from India have consistently demonstrated a higher prevalence of Gram-negative bacilli such as *Pseudomonas aeruginosa*, *Escherichia coli*, *Klebsiella* species in diabetic foot infections.^[8,16,17] This geographic variation is heavily influenced by factors such as walking barefoot, poor foot hygiene, agricultural occupational hazards common in rural settings and delayed presentation to healthcare facilities.^[4,9,15] Recent Indian literature further emphasizes the high isolation rates of these environmental pathogens in chronic wounds.^[13,18]

Furthermore, the emergence of multidrug-resistant (MDR) strains in DFU infections complicates empirical management and contributes to prolonged hospitalizations.^[5] The bacteriological profile and antimicrobial susceptibility patterns fluctuate significantly not only between countries but also among different hospital settings within the same geographic region,^[19,26,30] relying on generalized guidelines can result in therapeutic failure and disease progression.

Therefore, continuous surveillance of the local bacteriological profile and antibiogram is essential. This study aims to evaluate the microbial etiology and antimicrobial susceptibility patterns of diabetic foot infections in a rural tertiary care hospital, providing data to guide rational empirical antibiotic therapy.

MATERIALS AND METHODS

Study Design and Setting: A hospital-based, cross-sectional observational study was conducted at the Government Villupuram Medical College and Hospital, a rurally located tertiary care center. The study was carried out after obtaining approval from the Institutional Ethics Committee (IEC). A total of 110 patients previously diagnosed with diabetes

mellitus who presented with diabetic foot ulcers were enrolled in the study. Patients were recruited from the Outpatient Department (OP), general surgical inpatient wards, and the Surgical Intensive Care Unit (SICU).

Inclusion and Exclusion Criteria

Diabetic patients of all ages and both genders presenting with clinically infected foot ulcers (evidenced by the presence of purulent discharge, localized swelling, erythema, or warmth) were included in the study. Patients already receiving systemic or topical antibiotics for more than 48 hours prior to presentation, those with non-diabetic foot ulcers (e.g., venous or arterial ulcers), and those who refused to provide informed consent were excluded from the study.

Sample Collection

Wound samples were collected prior to the initiation, or following the temporary cessation, of empirical antibiotic therapy. The ulcer area and its margins were first cleansed thoroughly with sterile normal saline to remove surface colonizers, necrotic tissue, and superficial exudates. Deep tissue pus swabs were then collected from the base of the ulcer or the active margins using two sterile cotton-tipped swabs. The samples were immediately transported to the microbiology laboratory for processing to prevent the loss of fastidious organisms.

Microbiological Processing and Identification

One swab was used for direct microscopic examination using Gram staining to assess the initial presence of pus cells and bacterial morphology. The second swab was directly inoculated onto standard bacteriological media, including 5% Sheep Blood Agar (BA) and MacConkey Agar (MA). The inoculated plates were incubated aerobically at 37°C for 24 to 48 hours. Plates showing no microbial growth after 48 hours of extended incubation were reported as sterile (negative culture). For positive cultures, the isolated colonies were identified based on their colony morphology, Gram-staining characteristics, motility, and standard biochemical reactions (such as catalase, coagulase, oxidase, indole, methyl red, Voges-Proskauer, citrate utilization, and urease tests).

Total patients (110)



Antimicrobial Susceptibility Testing (AST):

Antimicrobial susceptibility testing of the isolated pathogenic strains was performed using the standard Kirby-Bauer disk diffusion method on Mueller-Hinton Agar (MHA), in strict accordance with the Clinical and Laboratory Standards Institute (CLSI)

guidelines.^[6] A bacterial suspension matching the 0.5 McFarland turbidity standard was prepared and swabbed evenly across the MHA plates. Standard antibiotic disks specific to Gram-negative bacilli (e.g., Amikacin, Ciprofloxacin, Piperacillin-Tazobactam, Meropenem, Ceftazidime) and Gram-positive cocci (e.g., Cefoxitin, Erythromycin, Linezolid) were applied. After 18-24 hours of incubation at 37°C, the zones of inhibition were measured and interpreted as Sensitive (S), Intermediate (I), or Resistant (R).

Statistical Analysis

The collected patient demographic data (age, gender, ward location) and microbiological results (culture positivity, isolated organisms, and antibiogram profiles) were entered and organized in Microsoft Excel. Categorical variables were expressed as frequencies and percentages. Inferential statistics were performed to identify correlations between patient characteristics, culture positivity, and antimicrobial resistance patterns. The Chi-square (χ^2) test was utilized for cross-tabulation analysis. A p-value of <0.05 was considered statistically significant.

Source of admission



Culture positivity rate

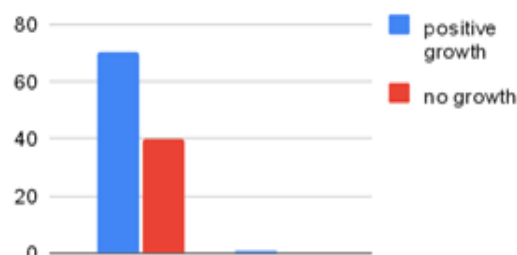


Table 1: prevalence of microorganisms isolated from diabetic foot ulcer

Microorganisms	Numbers of isolated (%)
Gram-Negative	
<i>Pseudomonas aeruginosa</i>	26(37.1)
<i>Escherichia coli</i>	19(27.1)
<i>Klebsiella pneumoniae</i>	18(25.7)
<i>Proteus mirabilis</i>	4(5.7)
<i>Proteus vulgaris</i>	1(1.4)
<i>Acinetobacter species</i>	1(1.4)
Gram-positive	
<i>Staphylococcus aureus</i>	1(1.4)
Variables are presented as frequency (Percentages)	

RESULTS

Patients data: Of a total 110 patients with diabetes, 68 (61.8%) were male and 42 (38.2%) were female. The mean age of the diabetic patients was 53.4 years (Range 20-87 years). The majority of the patients (89.1%, n=98) were admitted as inpatients (Wards/SICU), while 10.9% (n=12) were treated in the Outpatient Department (OP).

Identification of isolated microorganisms

Out of the 110 samples processed, 70 yielded significant bacterial growth, resulting in an overall culture positivity rate of 63.6%. All positive cultures (100%) were monomicrobial. Statistical analysis revealed that hospital admission status significantly correlated with culture positivity; inpatients were significantly more likely to yield a positive culture (67.3%) compared to outpatients (33.3%) ($p = 0.041$). There was no statistically significant difference in culture positivity based on gender ($p = 0.916$) or age group ($p = 0.798$).

Bacteriological Profile of Diabetic Foot Infections

The microbiological landscape was overwhelmingly dominated by Gram-Negative Bacilli (GNB), which

accounted for 98.6% (n=69) of all isolated pathogens. Only a single Gram-Positive Coccus (GPC), *Staphylococcus aureus* (1.4%), was isolated. Among the Gram-negative isolates, *Pseudomonas aeruginosa* was the most frequently isolated organism, accounting for 37.1% (n=26) of the total positive cultures. This was followed by *Escherichia coli* at 27.1% (n=19) and *Klebsiella pneumoniae* at 25.7% (n=18). Other less frequent isolates included *Proteus mirabilis* (5.7%, n=4), *Proteus vulgaris* (1.4%, n=1), and *Acinetobacter species* (1.4%, n=1). Antimicrobial Susceptibility and Resistance Patterns Antimicrobial susceptibility testing (AST) revealed alarmingly high resistance to commonly prescribed oral empirical antibiotics, particularly fluoroquinolones. *Pseudomonas aeruginosa* isolates demonstrated a 46.2% resistance rate to Ciprofloxacin. Similarly, Ciprofloxacin resistance was observed in 57.9% of *E. coli* and 61.1% of *K. pneumoniae* isolates. High resistance was also noted against third-generation cephalosporins; Ceftazidime resistance was 76.9% in *P. aeruginosa*, and Cefotaxime resistance reached 73.7% in *E. coli*. Trimethoprim-Sulfamethoxazole (Cotrimoxazole)

showed the highest overall resistance, failing against 84.6% of *Pseudomonas* and 78.9% of *E. coli* strains. Conversely, parenteral broad-spectrum antibiotics maintained excellent in-vitro efficacy. Piperacillin-Tazobactam emerged as the most effective antibiotic, showing 92.3% sensitivity for *P. aeruginosa*, 94.7% for *E. coli*, and 83.3% for *K. pneumoniae*. Amikacin also demonstrated robust activity, with sensitivity rates of 84.6%, 89.5%, and 94.4% against *Pseudomonas*, *E. coli*, and *Klebsiella*, respectively. Meropenem remained highly effective against *P. aeruginosa* (96.2% sensitive).

The single *Staphylococcus aureus* isolate was identified as Methicillin-Sensitive *Staphylococcus aureus* (MSSA), demonstrating 100% sensitivity to Cefoxitin, Erythromycin, Clindamycin, Linezolid, and Tetracycline, but showing complete resistance to Penicillin.

Multidrug Resistance (MDR) Patterns

A significant clinical observation in this study was the high prevalence of Multidrug-Resistant (MDR) strains among the Gram-negative isolates. Defining MDR as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, a substantial proportion of the major isolates met this criteria. Concurrent resistance to fluoroquinolones (Ciprofloxacin), third-generation cephalosporins (Ceftazidime or Cefotaxime), and folate pathway antagonists (Trimethoprim-Sulfamethoxazole) was frequently observed across *Pseudomonas aeruginosa*, *Escherichia coli*, and *Klebsiella pneumoniae*. Despite this high MDR burden, these isolates largely retained susceptibility to beta-lactam/beta-lactamase inhibitor combinations (Piperacillin-Tazobactam) and aminoglycosides (Amikacin), indicating that while resistance to first-line oral agents is widespread, reserve parenteral classes remain largely uncompromised.

Table 2: Demographic Characteristics of Primary Caregivers

Organisms	Amikacin	Piperacillin Tazobactam	Meropenem	Cotrimoxazole	Ceftazidime	Ciprofloxacin	Cefotaxime
<i>Pseudomonas aeruginosa</i> (n=26)	22(84.6%) S 3(11.5%) I 1(3.8%) R	24(92.3%) S 0(0)	25(96.2%) S 1(3.8%)	0(0)% S 22(84.6%) R	2(7.7%) S 21(80.7%) R 1(3.8%) I	13(50.0%) S 12(46.2%) R 1(3.8%) I	-
<i>Escherichia coli</i> (n=19)	17(89.5%) S 1(5.3%) I 1(5.3%) R	18(94.7%) S 1(5.3%)	2(10.5%) S 0(0)	4(21.1%) S 15(78.9%) R	1(5.3%) S 1(5.3%)	5(26.3%) S 12(63.1%) R 2(10.5%) I	2(10.5%) S 14(73.7%) R 2(10.5%) I
<i>Klebsiella pneumoniae</i> (n=18)	17(94.4%) S 1(5.6%)	15(83.3%) S 2(11.1%) I 1(5.6%) R	5(27.8%) S 2(11.1%) I 1(5.6%) R	5(27.8%) S 13(72.2%) R	1(5.6%) S 5(27.8%)	5(27.8%) S 11(61.1%) R 2(11.1%) I	3(16.7%) S 6(33.3%) R 1(5.6%) I
<i>Proteus mirabilis</i> (n=4)	4(100%) S 0(0)	4(100%) S 0(0)	4(100%) S 0(0)	1(25%) S 2(50%) R 1(25%) I	0(0) S 3(75%) R 1(25%) I	1(25%) S 2(50%) R 1(25%) I	-
<i>Proteus vulgaris</i> (n=1)	1(100%) S 0(0)	1(100%) S 0(0)	1(100%) S 0(0)	0(0) S 1(100%) R	0(0) S 1(100%) R	0(0) S 1(100%) R	-
<i>Acinetobacter species</i> (n=1)	1(100%) S 0(0)	1(100%) S 0(0)	1(100%) S 0(0)	0(0) S 1(100%) R	0(0) S 1(100%) R	0(0) S 1(100%) R	-

Variables are presented as frequency (Percentages) S. Susceptible. R. Resistance. I. Intermediate

Table 2: Clinical Characteristics of Children with Epilepsy

Organisms	Penicillin	Erythromycin	Cefoxitin	Clindamycin	Linezolid	Tetracycline
<i>Staphylococcus aureus</i> (n=1)	0(0) 1(100%)	1(100%) 0(0)	1(100%) 0(0)	1(100%) 0(0)	1(100%) 0(0)	1(100%) 0(0)

Variables are presented as frequency (Percentages) S. Susceptible. R. Resistance. I. Intermediate

DISCUSSION

Diabetic foot ulcers represent a major therapeutic challenge, heavily complicated by the onset of polymicrobial and multidrug-resistant infections. The present study evaluated the clinicomicrobiological profile of 110 diabetic foot ulcer patients in a rural tertiary care setting. The overall culture positivity rate in our study was 63.6%, which aligns with the findings of Sugandhi et al,^[19] and Zubair et al,^[23] who reported similar high infection rates in Indian cohorts. Male patients constituted the majority of the study population (61.8%). This male preponderance is well-documented in literature and is often attributed to higher outdoor occupational exposure like agriculture, the use of inappropriate footwear, and

delayed healthcare-seeking behavior among men in rural population.^[8,10] Furthermore, our statistical analysis demonstrated a significant association between inpatient admission and culture positivity ($p = 0.041$), reflecting that patients requiring hospitalization generally present with advanced, deeply infected Wagner's grade lesions compared to outpatients.

The most profound finding of this study is the near-total dominance of Gram-negative bacilli (98.6%), sharply contrasting with Western literature where Gram-positive aerobes like *Staphylococcus aureus* are classically described as the primary colonizers.^[7,15] In our setting, *Pseudomonas aeruginosa* (37.1%) was the predominant isolate, followed by enteric pathogens such as *Escherichia coli* and *Klebsiella pneumoniae*. This massive shift

toward Gram-negative etiology perfectly corroborates multiple studies from South India, including Viswanathan et al,^[17] and Shanmugam et al,^[5] who emphasize that tropical climates, barefoot walking, and rural agricultural hazards heavily predispose DFU wounds to environmental and water-borne Gram-negative pathogens.^[4,9,16]

The antibiogram profile exposes a critical clinical challenge: the widespread failure of standard empirical oral therapies. Ciprofloxacin, a cornerstone of outpatient DFU management, exhibited resistance rates hovering between 46% to 61% across the major Gram-negative isolates. This high fluoroquinolone resistance, coupled with massive resistance to third-generation cephalosporins and Cotrimoxazole, signals a rapid emergence of multidrug-resistant (MDR) strains in the community.^[24,25] The indiscriminate use of these oral antibiotics in peripheral clinics prior to tertiary hospital referral is a likely driver of this resistance.^[20,27]

Fortunately, our data indicates that parenteral therapies reserve high potency. Piperacillin-Tazobactam and Amikacin demonstrated excellent sensitivity (>80%) across *Pseudomonas*, *E. coli*, and *Klebsiella* species. Based on these findings, and aligning with the Infectious Diseases Society of America (IDSA) guidelines,^[1] empirical therapy for moderate-to-severe diabetic foot infections in our demographic must aggressively target Gram-negative organisms. Initiating empirical treatment with agents like Piperacillin-Tazobactam, rather than relying on Ciprofloxacin or basic cephalosporins, is strongly advocated to prevent treatment failure and limit amputation risks.^[26,30]

Emergence of Multidrug-Resistant (MDR) Pathogens

The prominent multidrug resistance (MDR) pattern observed in this study is a critical public health and clinical concern. A large fraction of the isolated Gram-negative bacilli demonstrated simultaneous resistance to three major antibiotic classes: fluoroquinolones, third-generation cephalosporins, and sulfonamides. This MDR phenotype in diabetic foot ulcers is strongly corroborated by Shanmugam et al,^[5] and Zubair et al,^[10,23] who reported an alarming rise in MDR *Pseudomonas* and *Enterobacteriaceae* in Indian DFU patients. The pathogenesis of DFU—characterized by chronic, open wounds with compromised local vascularity—creates an ideal environment for repeated microbial colonization and biofilm formation. Furthermore, the irrational use of broad-spectrum antibiotics in primary care settings, combined with poor patient compliance to prolonged antibiotic courses in rural demographics, exerts immense selective pressure that drives this MDR proliferation.^[9,25] Consequently, the presence of these MDR strains significantly narrows the therapeutic window, precluding the use of convenient oral regimens and forcing the reliance on intravenous therapies like

Piperacillin-Tazobactam and Carbapenems, thereby increasing the duration and cost of hospitalization.

Limitations

This study has certain limitations. The sample size of 110 patients from a single center may limit the broad generalizability of the statistical correlations. Additionally, the near-absence of Gram-positive organisms (n=1) in this specific cohort restricted our ability to perform comparative statistical analyses between GNB and GPC resistance patterns. Future multi-centric studies incorporating anaerobic culture techniques and molecular resistance typing are warranted.

CONCLUSION

Infections of diabetic foot ulcers in our rural tertiary care setting are overwhelmingly caused by Gram-negative bacilli, predominantly *Pseudomonas aeruginosa*. The alarming resistance to standard oral fluoroquinolones and cephalosporins necessitates a shift in empirical prescribing practices. Prompt wound culturing is mandatory, and early intervention with parenteral broad-spectrum antibiotics, such as Piperacillin-Tazobactam or Amikacin, forms the cornerstone of effective limb-salvage therapy.

REFERENCES

1. Lipsky BA, Berendt AR, Cornia PB, et al. 2012 Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. *Clin Infect Dis*. 2012;54(12):132–173.
2. Boulton AJM, Armstrong DG, Kirsner RS, et al. Diagnosis and management of diabetic foot complications. *American Diabetes Association. Diabetes Care*. 2018;41(8):1839–1845.
3. Singh N, Armstrong DG, Lipsky BA. Preventing foot ulcers in patients with diabetes. *JAMA*. 2005;293(2):217–228.
4. Lavery LA, Armstrong DG, Wunderlich RP, Mohler MJ, Wendel CS, Lipsky BA. Risk factors for foot infections in individuals with diabetes. *Diabetes Care*. 2006;29(6):1288–1293.
5. Shanmugam P, Jeya M, Linda S. The bacteriology of diabetic foot ulcers, with a special reference to multidrug resistant strains. *J Clin Diagn Res*. 2013;7(3):441–445.
6. Clinical and Laboratory Standards Institute (CLSI). Performance Standards for Antimicrobial Susceptibility Testing. 30th ed. CLSI Supplement M100. Wayne, PA: CLSI; 2020.
7. Tentolouris N, Jude EB, Smirnov I, Knowles EA, Boulton AJ. Methicillin-resistant *Staphylococcus aureus*: an increasing problem in a diabetic foot clinic. *Diabet Med*. 1999;16(9):767–771.
8. Gadepalli R, Dhawan B, Kapil A, Mohan V, Gadepalli R. A clinicomicrobiological study of diabetic foot ulcers in an Indian tertiary care hospital. *Diabetes Care*. 2006;29(8):1727–1732.
9. Shahi SK, Kumar A, Kumar S, et al. Prevalence of multidrug-resistant microbes in diabetic foot ulcer and environment of patients. *Asian Journal of Pharmaceutical and Clinical Research*. 2012;5(3):48–50.
10. Zubair M, Malik A, Ahmad J. Clinico-bacteriology and risk factors for the diabetic foot infection with multidrug resistant microorganisms in North India. *Biology and Medicine*. 2010;2(4):22–34.

11. Noor S, Zubair M, Ahmad J. Diabetic foot ulcer—a review of the literature. *Diabetes & Metabolic Syndrome*. 2015;9(3):192–197.
12. International Working Group on the Diabetic Foot (IWGDF). Guidelines on the diagnosis and treatment of foot infection in persons with diabetes (IWGDF 2019 update). *Diabetes Metab Res Rev*. 2020;36(S1):e3280.
13. Vinodkumar C.S., Hiresave S., Kandagal G.B., Bandekar G.N. Metallo- β lactamase producing *Pseudomonas aeruginosa* and its association with diabetic foot. *Indian J Surg*. 2011;73(4):291–294. doi: 10.1007/s12262-011-0287-5.
14. Spichler A, Hurwitz BL, Armstrong DG, Lipsky BA. Microbiology of diabetic foot infections: from Louis Pasteur to ‘crime scene investigation’. *BMC Med*. 2015;13(1):2.
15. Tiwari S., Pratyush D.D., Dwivedi A., Gupta S.K., Rai M., Singh S.K. Microbiological and clinical characteristics of diabetic foot infections in northern India. *J Infect Dev Ctries*. 2012;6(4):329–332. doi: 10.3855/jidc.1827.
16. Ramakant P, Verma AK, Misra R, et al. Changing microbiological profile of pathogenic bacteria in diabetic foot infections: time for a rethink on which empirical therapy to choose? *Diabetologia*. 2011;54(1):58–64.
17. Viswanathan V, Jasmine S, Snehalatha C, Ramachandran A. Prevalence of pathogens in diabetic foot infection in South Indian type 2 diabetic patients. *J Assoc Physicians India*. 2002;50:1013–1016.
18. Mohanasoundaram K.M. The microbial profile of diabetic foot infections. *J Clin Diagn Res*. 2012;6:409–412.
19. Sugandhi P, Prasanth DA. Microbiological profile of bacterial pathogens from diabetic foot infections in tertiary care hospitals, Salem. *Diabetes Metab Syndr*. 2014;8(3):129–132.
20. Tiwari S, Pratyush DD, Dwivedi A, Gupta SK, Rai M, Singh SK. Microbiological and clinical characteristics of diabetic foot infections in northern India. *J Infect Dev Ctries*. 2012;6(4):329–332.
21. Chincholikar DA, Pal RB. Study of fungal and bacterial infections of the diabetic foot. *Indian J Pathol Microbiol*. 2002;45(1):15–22.
22. Gadepalli R., Dhawan B., Kapil A. A clinico-microbiological study of diabetic foot ulcers in an Indian tertiary care hospital. *Diabetes Care*. 2006;29:1727–1732. doi: 10.2337/dc06-0116.
23. Zubair M, Malik A, Ahmad J. Clinico-microbiological study and antimicrobial susceptibility pattern of foot infections in North Indian patients with diabetes mellitus. *J Infect Public Health*. 2011;4(2):71–81.
24. Sivanmaliappan TS, Sevanan M. Antimicrobial susceptibility patterns of *Pseudomonas aeruginosa* from diabetes patients with foot ulcers. *Int J Microbiol*. 2011;2011:605195.
25. Kandemir O, Akbay E, Sahin E, Milcan A, Gen R. Risk factors for infection of the diabetic foot with multi-antibiotic resistant microorganisms. *J Infect*. 2007;54(5):439–445.
26. Banoo S, Puhazhendhi J, Renuka C, et al. Bacteriological profile of diabetic foot ulcer and its antibiotic susceptibility pattern. *Int J Curr Microbiol App Sci*. 2012;1(1):14–23.
27. Citron DM, Goldstein EJ, Merriam CV, Lipsky BA, Abramko MA. Bacteriology of moderate-to-severe diabetic foot infections and in vitro activity of antimicrobial agents. *J Clin Microbiol*. 2007;45(9):2819–2828.
28. Sekhar S.M., Vyas N., Unnikrishnan M.K., Rodrigues G.S., Mukopadhyay C. Antimicrobial susceptibility pattern in diabetic foot ulcer: a pilot study. *Ann Med Health Sci Res*. 2014;4(5):742–745. doi: 10.4103/2141-9248.141541.
29. Banashankari GS, Rudresh HM. Bacteriological profile of diabetic foot ulcer and their antibiotic susceptibility pattern. *Indian Journal of Microbiology Research*. 2016;3(2):167–171.
30. Rao M, Seshadri M. A clinicomicrobiological study of diabetic foot ulcers in a tertiary care hospital. *Int J Med Sci Public Health*. 2014;3(8):937–940.