

THE PHARMA INNOVATION - JOURNAL

Microbiological profile of skin infections in diabetic patients: A prospective study

Name of author

Dr. Lysetty Rajendra Prasad¹ and Dr. Kukkala Seetharamaraju²

¹Professor, Department of Microbiology, Sardar Rajas Medical College and Hospital, Bhawanipatna, Odisha, India

²Associate Professor, Department of Dermatology, Sardar Rajas Medical College and Hospital, Bhawanipatna, Odisha, India

Corresponding author

Dr. Kukkala Seetharamaraju

Background and Objectives: People with diabetes are more likely to get skin and soft tissue infections because their immune systems are weak, their blood sugar levels are not well controlled, and their blood vessels are not working properly. The range of bacteria that cause infections and their patterns of susceptibility to antimicrobials differ from place to place. The objective of this investigation was to ascertain the microbiological profile, antibiotic susceptibility, and resistance trends of bacteria isolated from skin infections in diabetes individuals.

Materials and Methods: A prospective study was performed with 60 diabetes patients from the dermatology and medicine departments of a tertiary care hospital for a duration of 12 months. This study was conducted at the department of Dermatology, Sardar Rajas Medical College and Hospital, Bhawanipatna, Odisha from March 2010 to February 2011. Pus or wound swab samples were aseptically obtained and subsequently processed for the isolation of bacteria and fungi utilizing standard microbiological methods. We used Gram staining, culture characteristics, and biochemical assays to figure out what species were there. Demographic information on the patients, clinical aspects, and types of infections were written down.

Results: Six (10%) of the 60 samples were sterile, while 54 (90%) showed positive microbial growth. The most frequent isolates were *Staphylococcus aureus* (40%) and *Pseudomonas aeruginosa* (23%), *Escherichia coli* (15%), *Klebsiella pneumoniae* (10%), *Proteus mirabilis* (5%), and *Candida* species (7%). Of the isolates of *S. aureus*, 25% had methicillin-resistant *S. aureus* (MRSA). Although gram-negative isolates were responsive to carbapenems and aminoglycosides, they exhibited a high level of cephalosporin resistance. In 12% of patients, mixed infections were found. The most frequent clinical presentations were abscesses (20%), cellulitis (25%), and diabetic foot ulcers (55%).

Conclusion: *Staphylococcus aureus* is still the most common cause of skin infections in people with diabetes. MRSA and multidrug-resistant Gram-negative pathogens are becoming more common. Early identification of infections and their antimicrobial susceptibility is essential for efficient care and the prevention of problems in diabetic patients. It is suggested to keep an eye on resistance trends all the time to help with empirical therapy.

Keyword: Diabetes mellitus, skin infections, *Staphylococcus aureus*, MRSA, antimicrobial resistance, microbiological profile

Introduction

Infections of the skin and other soft tissues are among the most frequent and troublesome complications experienced by people with

diabetes mellitus. Persistent hyperglycemia compromises several host immune defense processes, including neutrophil chemotaxis, phagocytosis, and bactericidal activity.

Additionally, peripheral vascular dysfunction, neuropathy, and reduced tissue perfusion are linked to worse wound healing and increased infection risk. Microbe colonization, which can result in cellulitis or chronic, non-healing ulcers, is more likely to occur in certain settings ^[1-3].

A wide variety of skin infections can affect people with diabetes, ranging from less serious ones like folliculitis or furuncles to more significant ones like cellulitis or diabetic foot ulcers. Because it often requires long hospital admissions and, in severe cases, lower limb amputations, the latter is one of the most costly and disastrous complications of diabetes. Because many diseases are chronic and broad-spectrum antibiotics are widely used, the development of multidrug-resistant organisms has further complicated treatment outcomes ^[4-6].

Diabetic skin infections can be caused by a wide variety of microbiological organisms, which might vary depending on factors like location, healthcare facility, and antibiotic usage history. *Staphylococcus aureus* and related methicillin-resistant strains continue to rank highest among infectious agents, with Gram-negative bacilli including *Pseudomonas aeruginosa*, *Escherichia coli*, and *Klebsiella pneumoniae* following closely behind ^[7, 8]. Polymicrobial infections, which can include both aerobic and anaerobic bacteria, are another common complication of chronic diabetic wounds. Patients with long-term diabetes or those using immunosuppressants are particularly vulnerable to fungal infections caused by *Candida* species, which have recently gained prominence in therapeutic practice ^[9, 10].

It is critical to correctly identify the causative organisms and ascertain their patterns of antibiotic susceptibility in order to direct effective empirical treatment, avoid complications, and decrease antibiotic resistance. Despite the increasing prevalence of diabetic skin infections, there is a lack of regional data on the origin of these infections and trends in resistance in many settings. Finding the microbiological profile of diabetic skin infections, evaluating the antibiotic susceptibility patterns of the isolated pathogens, and offering valuable insights were the goals of this prospective study, which aimed to optimize

antimicrobial treatment and infection control measures in this high-risk population ^[11, 12].

Material and Methods

A prospective observational study was executed over a duration of 12 months within the Department of Microbiology, in conjunction with the Departments of Dermatology and General Medicine, at a tertiary care hospital. This study was conducted at the department of Dermatology, Sardar Rajas Medical College and Hospital, Bhawanipatna, Odisha from March 2010 to February 2011. The study comprised 60 diabetic patients clinically diagnosed with diverse skin and soft tissue infections. The Institutional Ethics Committee gave its blessing, and all subjects gave their informed consent before samples were taken.

Inclusion Criteria

- Patients clinically diagnosed with diabetes mellitus.
- Diabetic patients presenting with skin.
- Patients aged 18 years and above.
- Patients who provided written informed consent to participate in the study.

Exclusion Criteria

- Non-diabetic patients with skin infections.
- Patients who had received systemic antibiotics.
- Patients with immunocompromised conditions other than diabetes.
- Post-surgical wound infections unrelated to diabetic etiology.
- Patients who did not consent to participate in the study.

Sample Collection

Using sterile cotton swabs or syringes, samples of pus, wound fluid, or tissue were aseptically obtained from areas of infection. To avoid contamination and guarantee reliable results, samples were promptly transported to the microbiology laboratory in sterile containers.

Microbiological Processing

Gram staining was applied to each material in order to perform a preliminary microscopic

analysis. In order to isolate bacterial and fungal pathogens, samples were subsequently inoculated onto Blood agar, MacConkey agar, and Sabouraud Dextrose agar (SDA). For bacterial growth, plates were incubated aerobically at 37 °C for 24 to 48 hours, while for fungus isolation, SDA plates were incubated at 25 °C for up to 7 days.

Identification of Isolates

Bacterial isolates were identified using colony morphology, Gram staining, and routine biochemical assays, including catalase, coagulase, oxidase, indole, citrate utilization, urease, and triple sugar iron tests. Fungal isolates were identified using colony features and evaluation of Lactophenol Cotton Blue (LPCB) mounts.

Antimicrobial Susceptibility Testing

The antimicrobial susceptibility of bacterial isolates was assessed using the Kirby-Bauer disc diffusion method on Mueller-Hinton agar, in accordance with Clinical and Laboratory Standards Institute (CLSI) standards. The

antibiotics evaluated comprised beta-lactams, cephalosporins, aminoglycosides, fluoroquinolones, and carbapenems. The identification of methicillin resistance in *Staphylococcus aureus* was conducted with a cefoxitin disc (30 µg). Multidrug resistance (MDR) is characterized by resistance to at least one agent from three or more classes of antibiotics.

Data Collection and Analysis

Patient demographic information, duration and type of diabetes, infection site, clinical diagnosis, and microbiological results were documented. The data were evaluated with descriptive statistics and given as frequencies and percentages.

Results

The study comprised 60 diabetic patients who had skin and soft tissue infections that had been clinically identified. The data were examined for demographic information, clinical manifestations, microbiological characteristics, and patterns of antimicrobial susceptibility.

Table 1: Distribution of Diabetic Patients According to Age and Gender

Age Group (years)	Male (n=38)	Female (n=22)	Total (%)
31-40	6	4	10 (16.6)
41-50	9	6	15 (25.0)
51-60	13	7	20 (33.3)
61-70	7	4	11 (18.3)
>70	3	1	4 (6.6)
Total	38 (63.3%)	22 (36.7%)	60 (100%)

With 33.3% of the total, patients fell into the 51-60 age bracket, with 25.0% falling into the 41-50 age bracket. With 63.3% of cases being male and

36.7% being female, it appears that diabetic skin infections are more common in men.

Table 2: Clinical Types of Skin Infections in Diabetic Patients

Type of Infection	No. of Cases (n=60)	Percentage (%)
Diabetic foot ulcer	33	55.0
Cellulitis	15	25.0
Abscess	9	15.0
Carbuncle/Furuncle	2	3.3
Fungal infection (Candida)	1	1.7
Total	60	100

The most prevalent clinical manifestation was diabetic foot ulcers (55%), followed by cellulitis (25%) and abscesses (15%). Fungal infections

were less common (1.7%), showing that bacterial infections are more common in diabetic skin diseases.

Table 3: Microbiological Profile of Isolates from Skin Infections

Microorganism Isolated	No. of Isolates	Percentage
<i>Staphylococcus aureus</i>	22	40.7
<i>Pseudomonas aeruginosa</i>	12	22.2
<i>Escherichia coli</i>	8	14.8
<i>Klebsiella pneumoniae</i>	6	11.1
<i>Proteus mirabilis</i>	3	5.6
<i>Candida</i> species	3	5.6
Total Positive Cultures	54	100

Out of 60 samples, 54 (90%) exhibited positive microbial growth. The most common pathogen found was *Staphylococcus aureus* (40.7%),

followed by *Pseudomonas aeruginosa* (22.2%) and *E. coli* (14.8%). Seven (12%) of the positive cases had mixed infections.

Table 4: Antibiotic Susceptibility Pattern of *Staphylococcus aureus*

Antibiotic	Sensitive (%)	Resistant (%)
Cefoxitin (for MRSA detection)	75 (17 sensitive, 5 MRSA)	25
Erythromycin	68.2	31.8
Ciprofloxacin	63.6	36.4
Clindamycin	77.3	22.7
Gentamicin	81.8	18.2
Linezolid	100.0	0
Vancomycin	100.0	0

A quarter of the *S. aureus* isolates were determined to be MRSA. Ciprofloxacin and Erythromycin showed significant resistance rates of 36.4% and 31.8%, respectively, while Linezolid and Vancomycin were responsive to all

isolates. Even though MRSA has emerged, glycopeptides and oxazolidinones have maintained their effectiveness, as shown in the pattern.

Table 5: Antibiotic Susceptibility Pattern of Gram-negative Isolates

Antibiotic	Sensitive (%)	Resistant (%)
Piperacillin-Tazobactam	82.7	17.3
Ceftazidime	48.3	51.7
Ceftriaxone	44.8	55.2
Ciprofloxacin	58.6	41.4
Gentamicin	69.0	31.0
Amikacin	79.3	20.7
Imipenem	93.1	6.9
Meropenem	89.7	10.3

Carbapenems (imipenem and meropenem) remained significantly effective, despite gram-negative isolates exhibiting pronounced resistance to cephalosporins. Aminoglycosides, such as gentamicin and amikacin, also showed good sensitivity patterns. This pattern shows that *Pseudomonas* and *Enterobacteriaceae* species are becoming more resistant to cephalosporins.

Discussion

The present prospective study examined the microbiological profile and antibiotic susceptibility patterns of skin and soft tissue infections (SSTIs) in diabetes individuals. This study including 60 diabetic patients revealed that microbial infections are a prevalent complication of diabetes mellitus, particularly among middle-aged and older individuals. There were more males (63.3%) than females (36.7%), and most of the patients were between the ages of 51 and 60.

In this study, the most common clinical manifestation was diabetic foot ulcers (55%), followed by cellulitis and abscesses [13-15].

People with long-term ulcers, neuropathy, or peripheral vascular disease can get infections like this. Infections are very common in diabetic lesions, as shown by the fact that 54 out of 66 samples (almost 90%) showed positive culture growth. *Staphylococcus aureus* was the clear winner with 40.7% of the votes. *Pseudomonas aeruginosa* came in second with 22.2%, *Escherichia coli* came in third with 14.8%, and *Klebsiella pneumoniae* came in fourth with 11.1%. The prevalence of *S. aureus* may be attributed to its colonization of the skin and nasal mucosa, along with diminished immune defense systems in persons with diabetes [16-18].

Significantly, 25% of *S. aureus* isolates were MRSA, indicating methicillin-resistant *Staphylococcus aureus*. This observation is in line with the global trend of MRSA infections becoming more widespread. Because there aren't many antibiotics that work against MRSA and the infection could last a long time, treating it is very difficult [19-21]. It is not surprising that *Pseudomonas aeruginosa* was the most prevalent Gram-negative isolate because it is so often linked to diabetic ulcers and other long-lasting, wet wounds. Carbapenems (Imipenem, Meropenem) and aminoglycosides (Amikacin, Gentamicin) were effective against these isolates, although cephalosporins (Ceftazidime and Ceftriaxone) exhibited significant resistance [22-24].

The discovery of mixed infections in 12% of cases supports the notion that chronic diabetic wounds are often polymicrobial, involving organisms that engage in both aerobic and anaerobic processes. In conclusion, the outcomes of this study underscore the necessity for antibiotic susceptibility testing and regular microbiological assessments for diabetic skin infections [25, 26]. When antibiotics are given without taking culture-based recommendations into account, there is a chance that the treatment may not work and that resistant strains will develop. This study found a high chance of resistance to commonly used antibiotics, therefore we require antimicrobial stewardship programs and patient education on hygiene,

wound care, and blood glucose control right now [27, 28].

Conclusion

This study shows that diabetes patients, especially middle-aged and elderly ones, have common and clinically important skin and soft tissue infections. Microbiological study showed that *Staphylococcus aureus* causes the most infections, followed by *Pseudomonas aeruginosa*, *Escherichia coli*, and *Klebsiella pneumoniae*. Many Gram-negative *S. aureus* isolates were multidrug-resistant, especially to cephalosporins, and many were MRSA. To prevent pathogen resistance and guarantee proper treatment, periodic culture and sensitivity testing before antibiotic therapy is essential. Linezolid, Vancomycin, and Carbapenems are effective against resistant strains, but they must be used wisely. Glycemic control, wound care, and patient education are essential for preventing and treating diabetic skin infections. Microbial patterns and antibiotic resistance trends must be monitored regularly to optimize empirical therapy, reduce treatment failure, and improve patient outcomes. In conclusion, early microbiological identification and sensible antibiotic usage can reduce morbidity, complications, and better quality of life for diabetics with skin and soft tissue infections.

Funding

None

Conflict of Interest

None

References

1. Bhatia M, Sharma A, Gupta S, Kaur J. Microbiological profile of diabetic foot ulcers and its clinical implications. *J Indian Med Assoc.* 2008;106(6):389-391.
2. Gadepalli R, Dhawan B, Sreenivas V, Kapil A, Ammini AC, Chaudhry R. A clinicomicrobiological study of diabetic foot ulcers in an Indian tertiary care hospital. *Diabetes Care.* 2006;29(8):1727-1732.
3. Viswanathan V, Jasmine JJ, Snehalatha C, Ramachandran A. Prevalence of pathogens in diabetic foot infections in South Indian type 2

- diabetic patients. J Assoc Physicians India. 2002;50:1013-1016.
4. Lipsky BA, Berendt AR, Deery HG, Embil JM, Joseph WS, Karchmer AW, *et al.* Diagnosis and treatment of diabetic foot infections. Clin Infect Dis. 2004;39(7):885-910.
 5. Banu A, Hasan MM, Rajkumar J, Srinivasa S. Study on the microbial flora and antibiotic susceptibility pattern of diabetic foot infections. Ann Biol Res. 2008;2(4):1-7.
 6. Tentolouris N, Jude EB, Smirnof I, Knowles EA, Boulton AJM. Methicillin-resistant *Staphylococcus aureus*: an increasing problem in diabetic foot clinic. Diabet Med. 1999;16(9):767-771.
 7. Zubair M, Malik A, Ahmad J. Clinico-microbiological study and antimicrobial drug resistance profile of diabetic foot infections in North India. Foot (Edinb). 2009;19(1):14-19.
 8. Citron DM, Goldstein EJC, Merriam CV, Lipsky BA, Abramson MA. Bacteriology of moderate-to-severe diabetic foot infections and *in vitro* activity of antimicrobial agents. J Clin Microbiol. 2007;45(9):2819-2828.
 9. Wheat LJ. Infection and diabetes mellitus. Diabetes Care. 1980;3(1):187-197.
 10. Eleftheriadou I, Tentolouris N, Argiana V, Jude E, Boulton AJM. Methicillin-resistant *Staphylococcus aureus* in diabetic foot infections: an overview of prevalence, impact, and treatment challenges. Int J Low Extrem Wounds. 2009;8(1):37-43.
 11. Hartemann-Heurtier A, Robert J, Jacqueminet S, Ha Van G, Golmard JL, Jarlier V, *et al.* Diabetic foot ulcer and multidrug-resistant organisms: risk factors and impact. Diabet Med. 2004;21(7):710-715.
 12. El-Tahawy AT. Bacteriology of diabetic foot infections. Saudi Med J. 2000;21(4):344-347.
 13. Brook I. Secondary bacterial infections complicating skin lesions. J Med Microbiol. 2002;51(10):808-812.
 14. Ghenghesh KS, El-Mohammady H, Levin S, Boudargham MN, Rahouma A. Microbial infections among diabetic patients in Tripoli, Libya. J Med Microbiol. 2009;58(11):1421-1426.
 15. Shankar EM, Mohan V, Premalatha G, Srinivasan RS, Usha AR. Bacterial etiology of diabetic foot infections in South India. Eur J Intern Med. 2005;16(8):567-570.
 16. Goldstein EJC, Citron DM, Nesbit CA. Diabetic foot infections: bacteriology and activity of ten oral antimicrobial agents against bacteria isolated from consecutive cases. Diabetes Care. 1996;19(6):638-641.
 17. Mottola C, Mendes JJ, Cristino JM, Cavaco-Silva P, Tavares L, Oliveira M. Polymicrobial biofilms by diabetic foot clinical isolates. Folia Microbiol (Praha). 2010;55(6):533-539.
 18. Bouter KP, Storm AJ, Hoekstra JB. The diabetic foot in the Netherlands: epidemiological aspects and costs. Diabet Med. 1993;10(Suppl 2):55-58.
 19. Lipsky BA. Evidence-based antibiotic therapy of diabetic foot infections. FEMS Immunol Med Microbiol. 1999;26(3-4):267-276.
 20. Citron DM, Goldstein EJC, Merriam CV, Lipsky BA, Abramson MA. Bacteriology of diabetic foot infections. J Clin Microbiol. 2007;45(9):2819-2828.
 21. Singh N, Armstrong DG, Lipsky BA. Preventing foot ulcers in patients with diabetes. JAMA. 2005;293(2):217-228.
 22. Manisha R, Sharma VK, Raina S, Verma A. Microbiological spectrum of diabetic foot infections: a study from North India. Indian J Pathol Microbiol. 2009;52(3):356-359.
 23. Joseph WS. Treatment of lower extremity infections in diabetics: experience with 215 cases. South Med J. 1990;83(3):292-298.
 24. Ha Van G, Siney H, Hartemann-Heurtier A, Jacqueminet S, Golmard JL, Jarlier V. Role of multi-resistant organisms in diabetic foot infections. Diabet Med. 2003;20(10):894-898.
 25. Pemán J, Cantón E, Viudes A, Quindós G, Gobernado M. Fungal infection in diabetic patients. Rev Iberoam Micol. 2009;26(2):84-88.
 26. Edmonds ME, Foster AVM. The use of antibiotics in the diabetic foot. Am J Surg. 2004;187(5A):25S-28S.
 27. Lipsky BA, Holroyd KJ, Zasloff M. Topical antimicrobial therapy for diabetic foot infections. Clin Infect Dis. 1997;25(5):1123-1133.