ORIGINAL ARTICLE

# BACTERIOLOGICAL PROFILE AND ANTIBIOTIC SUSCEPTIBILITY PATTERNS IN DIABETIC FOOT INFECTIONS, AT LADY READING HOSPITAL, PESHAWAR

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**Background:** Diabetic foot ulcer is one of the common complications of diabetes and is also the major cause of hospitalization across the world. To treat it properly, bacteriological profile is important to institute appropriate treatment. This study is done with the objective to determine the microbiological profile and antibiotic susceptibility patterns of organisms isolated from diabetic foot ulcers in Lady Reading Hospital, Peshawar Pakistan. **Methods:** This cross-sectional study was conducted from January to June 2019. Swab samples were collected from 114 patients with diabetic foot infections and inoculated on appropriate media. Antibiotic susceptibility tests were done by Kirby Bauer disk diffusion method. **Results:** *E. coli* were predominately isolated in the study, with ESBL in 41.6% of the cases. Strains of Pseudomonas with MDR and XDR were isolated in 21.8% and 6.25% of the patients respectively. Majority of Gram-positive isolates were *Staphylococcus aureus*, those were MRSA in 76.6% of samples. The commonly involved sites of DFU were the toes and forefoot, and the main causes were blister formation or trauma. Most of the patients were identified to have risk factors such as peripheral neuropathy, peripheral arterial disease, over weight and poorly controlled diabetes. **Conclusion:** In our study, Gram negative aerobes were predominantly isolated in the diabetic foot infections. A significant number of MDR isolates were also observed. Lack of awareness about DFU and inappropriate use of broad-spectrum antibiotics may be the main cause of increase in the frequency of MDR isolates.

**Keywords:** Diabetic foot ulcer; Diabetic foot infections; Antibiotic susceptibility

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## INTRODUCTION

Diabetic foot ulcer (DFU) is one of the common complications of diabetes and is also the major cause of hospitalization across the world.[1,2] The prevalence of DFU in different communities has been identified to vary from 3–18%.[2,3] Diabetic foot ulcer has a wide spectrum of presentation, from a mild to highly severe condition that may culminate in amputation. DFU may lead to psychological disorders, socioeconomic stresses due to high cost and poor productivity, low quality of life both due to emotional and physical disability and even death.[2,4] In Pakistan about 14–20% of patients with DFU undergo amputation, which is an alarming figure.[5]

The International Working Group on the Diabetic Foot (IWGDF), has described a DFU as a full-thickness wound penetrating through the dermis (the deep vascular and collagenous inner layer of the skin) located below the ankle in a diabetic patient.[6] The predisposing factors for DFU are many and include; poorly controlled diabetes over a course of time, foot traumas, mechanical pressures particularly due to excessive weight of patient, tobacco smoking, prolonged duration of diabetes with advancing age.[2,3]

Diabetic peripheral neuropathy and peripheral vascular disease are the two main risk factors for DFU and subsequently infections. Both neuropathy and ischemia cause dryness, cracks, fissures, callosities and repeated trauma to insensate feet, which augments chances of DFU. The ischemic feet have compromised supply of phagocytic cells and micro nutrients to the affected site, so DFUs once infected have a protracted course to heal.[3,4]

Diabetic foot infections (DFI) may be monomicrobial or polymicrobial. Commonly isolated aerobic bacteria are *Staphylococcus aureus, Streptococcus pyogenes, Escherichia coli, Pseudomonas aeruginosa, Proteus, Klebsiella* species. In about 25% of patients' anaerobic bacteria are isolated. Unfortunately, many bacteria develop the ability to adapt and

overcome the antibiotics efficacy in wound milieu and hence develop resistance to antibiotics. Antibiotic resistance in DFI has become a noteworthy concern. MRSA (Methicillin-resistant *Staphylococcus aureus*) has prevalence of 10–22% and resistant strains of *E coli* are found in about 30% of DFU. In hospitalized patients' prevalence of MRSA ranges from 15–30% depending on the standards of health care. Consequent to antimicrobial resistance there is further burdening of the health care system with increase in morbidity, mortality and treatment cost. The appropriate management of DFI warrants in-depth knowledge of its features and susceptibility patterns of microorganisms. For this reason, culture and sensitivity testing of DFU is an essential early investigation that would facilitate judicious use of antibiotics, that may lead to risk reduction in the morbidity and amputation rate of the affected patients.[1,5]

The treatment of DFI is initiated empirically according to local standards of practice. Most health systems have documented Gram positive organisms as main causative organisms while others have isolated Gram negative as main causative organisms. The antibiotic susceptibility and resistance patterns of these organisms usually change over the course of time, that depends on many factors like previously used antibiotics and its duration of treatment, frequency of hospitalization for the same DFI, duration of hospital stay(s), neuropathy, size of ulcer and underlying osteomyelitis.[6–9] In Pakistan, *Staph aureus* and *E coli* are found to be the most commonly isolated organisms from DFI, however both microbes have shown considerable MDR.[5,7]

Several studies claim that deep tissue biopsy is the gold standard for culturing technique in diabetic foot infections. But many studies results have shown that swab cultures are reliable in diabetic soft tissue infections and while those with osteomyelitis require deep tissue biopsy.[10]

Undoubtedly DFU and DFI are a global public health concern with a 5-year survival of 50–60% after the occurrence of first DFU that is actually worse than many malignant conditions. The key focus should be shifted to the prevention of DFU. Education and enhancing awareness of the health care professionals (physicians, nurses, paramedics, podiatrists etc.), patients and their caregivers, self-management through diet, lifestyle, medication, blood glucose testing, foot care and check-ups including foot wear counselling and periodic screening for diabetic complications including foot assessment are the key components of prevention.[11–13]

The present study was designed to determine the frequency of microorganisms (bacteria) involved in diabetic foot infections and the antibiotic susceptibility patterns in patients presenting to The Department of Diabetes & Endocrinology, MTI, Lady Reading Hospital Peshawar. Besides that, the underlying risk factors (i.e., peripheral arterial disease, sensory peripheral neuropathy, poor glycaemic control) were also observed in those patients. The study was first of its kind in our local population. This study would help to formulate our local and institutional guidelines for prescribing appropriate antibiotics in DFI and to avoid irrational use of broad-spectrum antibiotics, hence preventing development of multi drug resistance (MDR). Furthermore, the study would also give us insight into developing preventive strategies to avoid DFU and consequent DFI.

## MATERIAL AND METHODS

It was a cross-sectional study, conducted from 1st January to 31st June 2019 at The Department of Diabetes and Endocrinology, MTI Lady Reading Hospital Peshawar. The ethical approval was sought from Hospital Ethical Review Board. Sample size was determined through WHO sample size calculator with 95% confidence level, 5.5% absolute precision and 10% reported frequency of MRSA in DFU.[1] All patients with diagnosed Type 2 Diabetes Mellitus who presented to our Unit with DFI were included in the study through non-probability consecutive sampling. DFI was defined clinically and biochemically as foot ulcer in patients with diabetes with 3 or more of following: signs of inflammation (redness, warmth, tenderness), oedema, purulent discharge, TLC (total leukocyte count) >10,000 or C-reactive protein >6 mg/L. Patients with cellulitis (intact skin) who didn't require incision and drainage or those with no growth on cultures were excluded. Besides that, patients with pregnancy or acute medical conditions like myocardial infarction, stroke, and major surgery in the past 6 weeks or end-stage chronic kidney disease (CKD), chronic liver disease (CLD) or underlying malignancies were excluded.

Written informed consent was taken from each patient after explaining the study protocol. Baseline demographic information (age, gender, address etc.) of each patient was recorded on a *pro forma*. Duration of diabetes, duration of ulcer, preceding event that led to DFU (trauma,

blister, or other causes) was noted. Patients were assessed clinically for underlying sensory peripheral neuropathy and peripheral vascular disease (PAD). PAD was diagnosed as ankle brachial index (ABI) <0.9 in either leg using a bidirectional handheld Doppler ultrasound instrument. Peripheral neuropathy was defined as reduced vibration (by 128 Hz tuning fork) or light touch perception evaluated using a 10 g Semmes–Weinstein monofilament.

Deep tissue swabs were collected from patient's wounds after scrubbing the site with normal saline and debridement if required. Culture and sensitivity were done in hospital's pathology laboratory by standard microbiological methods and antibiotic susceptibility was performed according to the guidelines of Clinical and Laboratory Standards Institute (CLSI). The culture media used for aerobic organisms were Maconkey agar, Blood agar & Chocolate agar plates. Antibiotic susceptibility tests were done by Kirby Bauer disk diffusion method on Mueller Hinton agar plates. MDR (multi-drug resistance) was defined as resistance to 3 or more drugs of the following classes: Fluoroquinolones (Ciprofloxacin), Aminoglycosides (Amikacin, Gentamicin), Beta-lactams (Tazobactam), and Carbapenems (Imipenem, Meropenem). XDR (Extended drug resistance) was defined as resistance to all antimicrobial categories except one or two (Polymyxin, Colistin). ESBLs organisms were defined as having Beta-lactamases capable of conferring bacterial resistance to the Penicillins, Cephalosporins (first, second, and third-generation) and Aztreonam (but not to Carbapenems) by hydrolysis of these antibiotics, and which are inhibited by Beta-lactamase inhibitors such as Clavulanic acid.

Data was analysed using SPSS 22. Mean and standard deviations were calculated for quantitative variables (i.e., age, duration of diabetes and duration of ulcer). Frequencies and percentages were calculated for categorical variables (i.e., gender, glycaemic control, sensory neuropathy, peripheral arterial disease, organisms isolated and their drug sensitivity pattern). The results were presented in the form of graph and tables.

## RESULTS

A Total of 114 cases were recorded. Tables-1 and 2. Outline basic data of sample. Amongst the bacterial isolates *E coli* was the most common with frequency of 36 (31.57%), followed by *Pseudomonas* species in 32 (28.07%) cases. (Table 3) MDR (multi-drug resistance) was found in 7 (21.9%) of Pseudomonas isolates. XDR (Extended drug resistant) was found in 2 (6.25%) of Pseudomonas isolates. (Table-3). Most of the bacterial isolates were sensitive to Cefoperazone/Sulbactam, Piperacillin/ Tazobactam, Carbapenems, Polymixin-B and Colistin. (Table-4) Most of bacterial isolates were resistant to Co=Amoxiclav, Cephalosporins & Quinolones. (Table-5)

**Table-1: Frequencies and percentages of basic data**

| | Frequency | Percentage |
|---|---|---|
| **Gender distribution of the patients** | | |
| Male | 66 | 57.9 |
| Female | 48 | 42.1 |
| **Location** | | |
| Toes and fore-foot | 63 | 55.26 |
| Mid-foot | 25 | 21.92 |
| Heel | 20 | 17.5 |
| Whole foot | 6 | 5.3 |
| **Cause of diabetic foot** | | |
| Post-blister | 49 | 43.0 |
| Trauma | 40 | 35.1 |
| Unknown | 25 | 21.9 |
| **HbA1c (%)** | | |
| (between 6–7) | 3 | (2.6 |
| (between 7–8) | 7 | 6.14% |
| (between 8–16) | 104 | 91.2% |
| **Foot Complications of Diabetes** | | |
| Peripheral Neuropathy | 106 | 93.0 |
| Peripheral Arterial Disease | 46 | 40.4 |

**Table-2: Means and Standard Deviation of the Quantitative variables**

| | Mean | SD |
|---|---|---|
| **Age** | 56.38 years | 10.37 |
| **Duration of Diabetes** | 12.1 years | 5.90 |
| **Duration of hospital stay (days)** | 12.39 days | 8.06 |
| **Duration of ulcer (weeks)** | 4.96 | 3.94 |
| **HbA1C** | 10.67 | 1.64 |

**Table-3: Bacterial Isolates of study group of patients**

| Bacteria | n (%) | ESBL | MRSA | MDR | XDR |
|---|---|---|---|---|---|
| *Staphylococcus* | 30 (26.31%) | - | 23 | - | - |
| *E. coli* | 36 (31.57%) | 15 | - | - | - |
| *Pseudomonas* | 32 (28.07%) | - | - | 7 (21.8%) | 2 (6.25%) |
| *Proteus* | 9 (7.9%) | 2 | - | - | - |
| *Klebsiella* | 6 (5.2%) | - | - | - | - |
| Others | 1 (0.87) | - | - | - | - |
| Total | 114 | 17 | 23 | 7 | 2 |

ESBL: Extended spectrum beta-lactamase, MRSA: Methicillin-resistant Staphylococcus aureus
MDR: Multi-drug resistant, XDR: Extended-drug resistant

**Table-4: Microorganisms & their antibiotics sensitivity**

| Antibiotics | *Staphylo-coccus Aureus* (n=30) MRSA=23 | *E.coli* (n=36) ESBL=15 | *Pseudomonas* (n=32) MDR=7 XDR=2 | *Klebsiella* (n=6) | *Proteus species* (n=9) ESBL=2 | Enterobacter (n=1) | *Strepto-coccus* (n=0) | Others species (n=0) |
|---|---|---|---|---|---|---|---|---|
| Co-Amoxiclav | 7 (23.33%) | 8 (22.2%) | 3 (9.37%) | 2 (33.3%) | 3 (33.34%) | 0 (0%) | - | - |
| Moxifloxacin | 5 (16.7%) | 9 (25%) | 12 (37.5%) | 4 (66.6%) | 7 (77.7%) | 1 (100%) | - | - |
| Quinolones (Ciprofloxacin, Levofloxacin) | 3 (10%) | 8 (22.2%) | 12 (37.5%) | 3 (50%) | 6 (66.6%) | 1 (100%) | - | - |
| Cefoperazone/ Sulbactam | 26 (86.7%) | 35 (97.22%) | 21 (65.6%) | 5 (83.3%) | 8 (88.9%) | 1 (100%) | - | - |
| Cephalosporins (Ceftriaxone, Cefotaxime) | 7 (23.3%) | 15 (41.66%) | 20 (62.5%) | 4 (66.7%) | 7 (77.8%) | 0 (0%) | - | - |
| Carbapenems (imipenem, Meropenem) | - | 36 (100%) | 22 (68.7%) | 6 (100%) | 9 (100%) | 1 (100%) | - | - |
| Piperacillin/ Tazobactam | - | 36 (100%) | 29 (90.6%) | 6 (100%) | 9 (100%) | 1 (100%) | - | - |
| Aminoglycosides | 16 (53.3%) | 28 (77.8%) | 23(71.8%) | 5 (83.3) | 7 (77.78%) | - | - | - |
| Clindamycin | 23 (76.6%) | - | - | - | - | - | - | - |
| Vancomycin | 23 (100%) | - | - | - | - | - | - | - |
| Linezolid | 23 (100%) | - | - | - | - | - | - | - |
| Teicoplanin | 23 (100%) | - | - | - | - | - | - | - |
| Fusidic Acid | 20 (66.67%) | - | - | - | - | - | - | - |
| Polymixin-B | - | 35 (97.2%) | 32 (100%) | 6 (100%) | 0 (0%) | 1 (100%) | - | - |
| Colistin | - | 35 (97.2%) | 32 (100%) | 6 (100%) | 0 (0%) | 1 (100%) | - | - |
| Doxycycline | 15 (50%) | - | - | - | - | - | - | - |
| Co-trimaxazole | 14 (46.67%) | - | - | - | - | - | - | - |

**Table-5: Microorganisms & their antibiotics resistance**

| Antibiotics | *Staphylo - coccus Aureus* (n=30) MRSA=23 | *E-coli* (n=36) ESBL=15 | *Pseudomonas* (n=32) MDR=7 XDR=2 | *Klebsiella* (n=6) | Proteus species (n=9) ESBL=2 | *Entero bacter* (n=1) | *Strepto-coccus* (n=0) | Others (n=0) |
|---|---|---|---|---|---|---|---|---|
| Co-Amoxiclav | 23 (76.66%) | 28 (77.8%) | 29 (90.6%) | 4 (66.6%) | 6 (66.67%) | 1 (100%) | - | - |
| Moxifloxacin | 25 (83.3%) | 27 (75%) | 20 (62.5%) | 2 (33.3%) | 2(22.2%) | 0 (0%) | - | - |
| Quinolones (ciprofloxacin, Levofloxacin) | 27 (90%) | 28 77.7%) | 20 (62.5%) | 3 (50%) | 3(33.3%) | 0 (0%) | - | - |
| Cefoperazone /Sulbactam | 4 (13.3%) | 1 (2.7%) | 11 (34.5%) | 1(16.6%) | 1(11.1%) | 0 (0%) | - | - |
| Cephalosporins (Ceftriaxone, Cefotaxime) | 23 (76.66%) | 21(58.33%) | 12 (37.5%) | 2(33.3%) | 2(22.2%) | 1 (100%) | - | - |
| Carbapenems (imipenem, Meropenem) | - | 0 (0%) | 10 (31.25%) | 0(0%) | 0(00%) | 0 (0%) | - | - |
| Piperacillin/Tazobactam | - | 0 (00%) | 3 (9.37%) | 0(0%) | 0(00%) | 0 (0%) | - | - |
| Aminoglycosides | 14 (46.66%) | 8 (22.22%) | 9 (28.1%) | 1(16.6%) | 2 (22.2%) | - | - | - |
| Clindamycin | 7(76.66%) | - | - | - | - | - | - | - |
| Vancomycin | 0 (00%) | - | - | - | - | - | - | - |
| Linezolid | 0 (00%) | - | - | - | - | - | - | - |
| Teicoplanin | 0 (00%) | - | - | - | - | - | - | - |
| Fusidic Acid | 10 (33.33%) | - | - | - | - | - | - | - |
| Polymixin-B | - | 1 (2.7%) | 0 (0%) | 0(0%) | 9 (100%) | 0(00%) | - | - |
| Colistin | - | 1 (2.7%) | 0 (0%) | 0(0%) | 9 (100%) | 0(00%) | - | - |
| Doxycycline | 15 (50%) | - | - | - | - | - | - | - |
| Co-trimaxazole | 16 (53.33%) | - | - | - | - | - | - | - |

## DISCUSSION

Diabetes over the last two decades has emerged as a global health problem with significant hospitalisation due to its associated complications. One of the most common complications yet a substantially ignored one is the diabetic foot with different categories of ulceration and infections. It has been stated in a European trial that half of such ulcers are associated with ischemia (49%) or infection (58%), or the combination of both in one third of cases (31%).[14] This aggravates the condition, resulting in amputations and mortality of those patients.

In our study the baseline characteristics of 114 diabetic foot ulcer patients showed 57.9% were males and 42.1% were females. There was not a very wide-range difference in gender but increased male prevalence has been reported in many other studies.[9,15–17] This may be due to outdoor physical activity among males compared to females or perhaps poor attention to foot care in our setting, particularly when people tend to walk bare footed and neglect the relevance of using proper foot wear.

The mean age of patients included in our study was in mid-fifties, that matched the findings of another group [17], but the age group observed in other studies was much lower in comparison to ours.[5,9,16] Our findings reflect that perhaps diabetic foot infections are likely affiliated with advancing age of the patient. Another possible reason maybe the duration of diabetes, it may correlate positively with the occurrence of diabetic foot problems. Some of the studies had the mean duration of diabetes less than 10 years[16,18], although the ones by Zahid et al and Anvarinejad *et al* was similar to ours with diabetes for over 13–15 years.[5,19]

There was a very noticeable observation that majority, 92% of our patients had poor glycaemic control, which was also noted in other studies albeit not such substantial number of patients. In two Iranian studies mean blood sugar levels instead of HbA1c was utilized as a predictor of Diabetes control.[5,19–22] The conjectures for poorly controlled Diabetes could be that in our study most of the patients were from a socially deprived economic strata and possibly their compliance and insight into self-management maybe far from desirable.

The patients presented to our hospital within an average time frame of five weeks after the occurrence of Diabetic foot afflictions. That correlated with the time of patient presentation at a few other centres[16,17], although in one center the patient presented from as early as one week to a maximum duration of one year after developing diabetic foot[18]. The early management of this condition would obviously be associated with a better prognosis.

There are many predisposing causes of DFU with subsequent infection. Our observations were that around one quarter patients have no precipitating cause for developing this condition, which matched the findings of a local study [20]. We further found that a greater number of patients in our study had some definitive predisposing condition like trauma, injury, blistering or callus formation that led to DFI.

Peripheral neuropathy is a well-recognized entity in patients with diabetes and has got a positive correlation in all such cases of DFU. Our observations were that almost all the patients had peripheral sensory neuropathy at the time of presentation, which matched the findings of study by Hena *et al.*[23] The reason for the greater percent of neuropathy may be the longer duration of diabetes and poorer glycaemic control, as reflected by high HbA1c levels. The studies in which the duration of diabetes was less with a fair glycaemic control had lesser extent of neuropathy as detected in their patients with Diabetic foot infections.[17,24]

Peripheral arterial disease was noted in a substantial number of our patients, with resultant ischemic and neuro-ischemic ulcers. There were similar observations by Ali *et al*[20], who had detected peripheral arterial disease as a very valid underlying feature. In another study 30% patients with peripheral arterial disease were initially diagnosed as ischemic ulcer on the day of hospital admission. The peripheral arterial disease was further confirmed by CT angiography in that study.[19]

The most common site of ulcer was noted at the fore foot particularly the lateral and big toes, followed by the mid foot. Perim *et al* have reported that the right forefoot to be predominantly affected with diabetic foot ulcers.[25] That was consistent with the findings by two other studies.[15,21]

Gram negative micro-organisms were predominantly seen in our study, same were the observations made by various other centres[15, 22,26] who reported to isolate gram negatives in majority of their patients. However, we had greater trend for infections with *E coli*, *Pseudomonas, Proteus* and *Klebsiella*, which was also observed by Zahid *et al* even though their study predominantly isolated Gram positives organisms.[5,22] In comparison, two other group noted *Pseudomonas* and *Klebsilella* isolates as the most commonly detected organisms.[15,26]

Gram positive micro-organisms were predominantly isolated as the most common strain by many workers from various centres across the world.[1,5,16,27] The most commonly isolated in our study were *Staphylococcus aureus*, and there were similar findings in studies from China, Iran, Egypt, Kuwait, India, Brazil and USA.[15,16,18,23–25,27–29]

However in a recent Iranian study growth of *Enterocci* has been predominantly detected.[22]

Over the last two decades MRSA has been recognized, as a common pathogen in DFU patients isolated in 10-40% of those patients.[24] In fact, the first two isolates of Vancomycin resistant MRSA strains were detected from diabetic foot patients.[27] Lately the community-acquired MRSA has been noted with alarmingly trend for insurgence.[27] In our study MRSA was isolated in 23% of the total strains, and accounted for 88% of *Staphylococcus* isolates. Anvarnijad also noted such trend in his study and documented MRSA as 78% of total *Staphylococcus* growth[19] whereas Umadevi reported 65.5% growth of MRSA from total Staphylococcal yield[26]. On the other hand the rate of MRSA growth was detected much lower in a Pakistani, Indian and Iranian studies.[5,15,24] The probabilities for high yield of MRSA in our set up may be due to injudicious use of antibiotics, improper strategies for infection control both in the hospital and community and with florid spread of community MRSA.

Besides that, there is a higher incidence of MDR pathogens in patients with DFUs that further impedes treatment of DFI, both medically as well as surgically and may culminate in amputations of lower limbs at different levels. Such MDR infections could possibly lead to increased length of stay in hospital, with resultant higher cost of management, morbidity and mortality of the patients.[5,9] We noted 41.6% and 22.2% of ESBL producing isolates of *E coli* and *Proteus* respectively in our patients which matched the findings of another study group in terms of isolation patterns, however besides *E coli* and *Proteus*, the other study had also isolated *Klebsilla* and *Enterobacteriaceae*, although there yield was 62.5–56%.[26] In Iranian study 53% of Gram negative bacteria were ESBL positive.[19] In an Indian study the ESBL producing strains of *E coli*, *Pseudomonas* and *Proteus* were found in equal distribution.[30]

In our study MDR *Pseudomonas* was detected in 21.9% of the isolates and the XDR *Pseudomonas* isolates were found in 6.25% of total *Pseudomonas* strains. Our MDR growth was comparatively low in contrast to the study by Zahid, Sekhar and Gadepali, and Ramakent who had alarmingly high growth of MDR even up to 80%.[5,9,15,17] Of note these studies had MDR isolates both for gram negatives as well as gram positive organisms. The likely assumptions were considered to be previous courses of antibiotics by other health care practices prior to consultation at the study centre or a protracted course of DFU. Interestingly several factors were found to be associated with MDR infections and those included the presence of neuropathy, ulcer size greater than 4 cm, underlying osteomyelitis, the requirement for deep surgical intervention and poor glycaemic control.[9]

With regards to the spectrum of antibiotic sensitivity, we noted that the *E coli* was highly sensitive to Carbapenems (Imipenem, Meropenem), Piperacillin/Tazobactam and Cefoperazone/Sulbactam. The antibiotic resistance for *E coli* was detected against Quinolones, Co-amoxiclav and Cephalosporins which was consistent with the findings of other studies across the globe.[5,9,16,23,30]

We also observed that the other Gram-negative organisms displayed a similar trend of antibiotic sensitivity but with regards to antibiotic resistance, *Klebsiella* and *Proteus* exhibited lesser resistance against Quinolones.

The MRSA strains that were isolated in our study had 100% sensitivity to Linezolid, Vancomycin and Teicoplanin, which has also been documented in another local study.[5] However, the studies by Citron, Sekhar, Gadepalli and Ahmadshooli have reported the sensitivity of MRSA to doxycycline and trimethoprim-sulfamethoxazole, which were much more cost effective.[9,15,22,27] Moreover, there we also recorded a very high resistance of MRSA against Quinolines in about 90% of the isolates, that was consistent with the findings in other studies as well.[5,16]

## CONCLUSION

DFU is a common complication encountered by diabetics due to a variety of causative factors. The superadded infection worsens the magnitude of morbidity and mortality and poses a strain on economy. Therefore, it is important to understand the causes of DFU and the patterns of microbiological infections to implement efficacious management strategies. We should also encourage the health care teams to employ preventive strategies for Diabetic foot problem, by generating awareness through public health strategies.

## AUTHORS' CONTRIBUTION

IU: Conception of study design, acquisition of data, data compilation and analysis, final approval of manuscript. SSA: Conception of study design, analysis and interpretation of data, drafting the work and revising and final approval of manuscript. IA: Final approval of manuscript. MNK: Final approval of manuscript. MRS: Analysis and interpretation of data, drafting the results. MUR: Acquisition of data, final approval of manuscript. SAM: Acquisition of data, final approval of manuscript

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