ORIGINAL ARTICLE

BACTERIOLOGY OF DIABETIC FOOT IN TERTIARY CARE HOSPITAL; FREQUENCY, ANTIBIOTIC SUSCEPTIBILITY AND RISK FACTORS

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Background: Diabetic foot being one of the frequent and disabling complications of diabetes**.** In view of widespread regional variation in causative organisms and antimicrobial susceptibility, the current study aimed to determine frequency of causative organisms, their antimicrobial susceptibility and associated risk factors. **Methods**: This descriptive cross-sectional study was conducted in 6 months' duration at dept. of Medicine; PIMS Hospital Islamabad. Type 2 Diabetes mellitus patients with diabetic foot ulcer were enrolled after informed consent. Patients already receiving antibiotics, having no growth on culture and >3 weeks' duration of ulcer were excluded. Sample from wound was sent for culture and sensitivity. Antibiotic susceptibility testing identified the susceptible and resistant strains of organisms. **Results:** Among 114 patients (66.67% males and 33.33% females); mean age was 55.11±11.96 years. *Staphylococcus aureus* was identified in 46%, *E. coli* in 28%, *Pseudomonas* in 6%, *Klebsiella* in 3.5% and other organisms in 17%. 92% of *S. aureus* was sensitive to Vancomycin and 67% to Clindamycin. Amongst *E. coli*, 81% showed sensitivity to Imipenem, 69% to Aminoglycosides and 31% to Quinolones. Glycaemic control was unsatisfactory in 65.8%. Peripheral vascular disease was found in 46% patients and sensory neuropathy in 94%. **Conclusion:** *Staphylococcus aureus* was the most frequent isolate amongst gram positive organisms while *E. coli* amongst gram-negatives. Vancomycin is suggested to be the drug of choice for gram positive and Imipenem for gram negative organisms. Appropriate antimicrobial therapy according to susceptibility patterns would reduce the morbidity and emergence of multidrug resistant organisms in diabetic foot infections.

Keywords: Antibiotic susceptibility; Bacteriology; Diabetic foot; Glycaemic control

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INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder with disturbance in carbohydrate, protein and lipid metabolism caused by absolute or relative deficiency of insulin.[1] Diabetic foot involvement including infections and foot ulcers are one of the frequently seen and disabling complications of diabetes leading to significant morbidity and mortality.[2]

Amongst the diabetic population, about 8–20% experience foot ulcer in life time.[1] It is one of the most common causes of hospital admissions in diabetics.[3] Diabetic foot ulcers have 15–45% higher risk of amputation of limb as compared to foot ulcers secondary to other aetiologies. The common underlying causes are neuropathy, foot deformity, trauma to foot, higher plantar pressures and peripheral vascular disease.[4] The local trauma and pressure, along with lack of sensation secondary to neuropathy and micro-vascular disease in the lower limbs may lead to foot infections that may extend in severity from simple superficial cellulitis to chronic disabling osteomyelitis.[5] The limited mobility of joints, pre-existing deformity of bony prominences and poor foot care further contribute to the risk of foot ulceration.[6,7] Skin and subcutaneous tissue infections are more frequent and severe in diabetic versus non- diabetic population. Also, the diabetic foot related hospitalizations are more than double in diabetic patients.[8] In general, foot infections in diabetics take prolonged time to cure than similar infections in non-diabetics.[9] The gold standard for assessing the diabetic foot remains the deep tissue cultures and sensitivity. The deeper the sample is obtained from tissue, the more reliable is the yield of culture. The underlying reason being superficial swabs may take the sample of colonizing organisms yielding the false positive results.[10]

Most of diabetic foot infections are mono-microbial with predominant involvement of gram positive bacteria.[11] *S. aureus* has been reported to be involved in about 33% of diabetic foot infections followed by *Pseudomonas* (12%), *Enterococci* (9%) and *E. coli* (8%).[3] *Klebsiella* is found in 14% of the patients in a study conducted by Gomez *et al*.[12] Anaerobic organisms are involved in 25% of the patients.[3]

Previous studies have shown that *S. aureus* is sensitive to Imipenem, Levofloxacin and Amikacin, with the sensitivity rates of 100% while it showed some sensitivity to Ceftriaxone (66 %),

Gentamycin (73%), Co-Amoxiclav (46.6%), Ceftazidime (46%) and Ciprofloxacin (63%).[2] Clindamycin and Vancomycin resistance was present in 54% and 63% respectively.[4] All the gram-negative isolates including *E. Coli* and *Enterococci* have sensitivity approaching 100% to Amikacin, Imipenem and Levofloxacin. Ceftriaxone and Ceftazidime show 50% activity against *E. Coli* while its sensitivity is 70% to Gentamycin and 40% to Co-Amoxiclav.[2] This shows high resistance to Clindamycin and Vancomycin.[4] *Pseudomonas aeruginosa* is 100% sensitive to Imipenem, 96% sensitive to Amikacin and Levofloxacin and 50% sensitive to Ceftriaxone while it has been shown to be comparatively less sensitive to Ceftriaxone (56%), Ceftazidime (72%), Gentamycin (48%), Co-Amoxiclav (44%) and Ciprofloxacin (48%) in previous studies.[2] *Klebsiella* has been previously shown to be sensitive to Ciprofloxacin and Gentamycin[1] but has shown resistance to Cloxacillin, Amoxycillin, Clindamycin, Vancomycin and Ceftazidime.[4]

Antibiotic resistance has become a major problem in diabetics, with resistance observed in up to 65% of patients with diabetic foot.[4] The *Methicillin-resistant Staphylococcus aureus i.e., MRSA* has prevalence of 10–22% and resistant *E. coli* accounts for 30% of diabetic foot infections.[5,11] Among the hospitalized patients, prevalence of *MRSA* in diabetic foot infections has been found to be 15–30% depending on the geography.[13]

The timely recognition and appropriate management of superficial diabetic foot ulcers improve the survival rate. Similarly, aggressive management of complicated infections prevents amputations in diabetics. Appropriate antibiotics are critical for the treatment plan of diabetic foot infections.

Decision of empirical antibiotic therapy depends on knowledge of prevalent microbial flora and their susceptibility profile. The purpose of this study is to assess the frequency of various organisms involved in diabetic foot ulcers, their drug sensitivity patterns in our setup and associated risk factors; so that appropriate antibiotics can be initiated earlier while awaiting the culture and sensitivity results which can improve the cure rate and prognosis in diabetics.

MATERIAL AND METHODS

This descriptive cross sectional study was conducted at Dept. of Medicine, PIMS Islamabad. Ethical approval was obtained from institutional review committee. Sample size was calculated by WHO sample size calculator with 95% confidence level, 5% precision level and 8% reported prevalence of diabetic foot ulcer in Pakistan. Patients with Type 2 Diabetes mellitus presenting to diabetic foot clinic and outdoor Medicine dept. with diabetic foot ulcer were included by non-probability consecutive sampling after informed consent. Diabetic foot was defined as foot ulcer, cellulitis or deep abscess in patients with Type 2 Diabetes mellitus. Patients receiving prior antibiotic therapy, patients with Type 1 Diabetes, those having no growth on cultures, having ulcer for more than 3 weeks and non-diabetic patients presenting with foot ulcer were excluded.

Demographic information (i.e., age, gender and contact address) was obtained. Duration of diabetes, duration of ulcer and history of previous hospitalizations due to foot ulcer was documented. Glycaemic profile was reviewed and labelled as satisfactory at HbA1c <7% as per criteria of American Diabetic Association (ADA). Patients were clinically assessed for presence of sensory neuropathy and peripheral vessels were examined to document the peripheral vascular disease.

Pus or discharge from the wound was obtained from each patient and was cultured in hospital's pathology laboratory. For aerobic and anaerobic organisms, culture media used was Mac-Conkey agar, Blood agar, Thioglycollate broth and Robertson's cooked meat media. The conventional biochemical tests were applied to identify the bacterial isolates. Kirby Bauer's disc diffusion technique on Muller Hinton agar was applied for obtaining antimicrobial susceptibility. The frequencies of various organisms isolated and their sensitivities to antibiotics were documented. All this information was documented on a specially designed *pro forma*.

SPSS-17 was used for data analysis. Descriptive statistics applied to calculate the mean and standard deviation for quantitative variables (i.e., age, duration of diabetes and duration of ulcer), and frequencies and percentages for qualitative variables (i.e., gender, glycaemic control, previous history of hospitalization due to ulcer, sensory neuropathy, peripheral vascular disease, organisms isolated and their drug sensitivity pattern). The results were presented in the form of tables and graphs.

RESULTS

Among 114 patients included, there were 76 (66.67%) males and 38 (33.33%) females. Mean age was 55±11.96 years. Glycaemic control was found to be satisfactory in 75 (72%) patients and was not found to be associated with age (p=0.479).

There was history of previous hospitalizations for foot ulcer in 15.8 % patients. 52 (46%) patients had evidence of peripheral vascular disease in the lower limbs and 107 (94%) had sensory neuropathy on clinical examination without any significant association with age (p=0.205; Table-1).

S. aureus was isolated in 52 (45.61%) patients with diabetic foot ulcers. *E. coli* was isolated in 32 (28%), *Pseudomonas* in 7 (6%), *Klebsiella* in 4 (3.5%) and other organisms in 19 (16.7%) patients. There was no association of gender with the bacteriology (p>0.05; figure-1).

Among patients with *S. aureus* infection, 92% showed sensitivity to Vancomycin, 42% were sensitive to Co-amoxiclav, 27% to Quinolones, 67% to Clindamycin, 85% to Imipenem, 54% to Aminoglycosides and 15% to Cephalosporins. Among patients with *Pseudomonas* infection, 86% showed sensitivity to Aminoglycosides, 71% to Imipenem, 43% were sensitive to Cephalosporins, 43% had sensitivity to Vancomycin, 28.5% to Quinolones, 28.5% to Co-amoxiclav and 28.5% were sensitive to Clindamycin (Table-3).

Among *E. coli* infections, 81% showed sensitivity to Imipenem, 69% to Aminoglycosides, 41% to Vancomycin, 31% to Quinolones, 12% to Co-amoxiclav, 31% to Clindamycin, and 34% to Cephalosporins. Among *Klebsiella* infections, 9% were sensitive to Aminoglycosides, 9% to Imipenem, 6% to Vancomycin, 6% to Clindamycin, 3% to Quinolones, 3% to Co-Amoxiclav and 3% to Cephalosporins. Out of 19 patients with other organisms, 68% showed sensitivity to Quinolones, 63% to Co-Amoxiclav, 58% to Vancomycin, 37% to Clindamycin, 63% to Imipenem, 63% to Aminoglycosides and 32% to Cephalosporins. Among *S. aureus* infection, 73% were resistant to Quinolones, 57.6% to Co-amoxiclav, 7.6% to Vancomycin, 32.7% to Clindamycin, 15.4% to Imipenem, 46% to Aminoglycosides and 84.6% to Cephalosporins. Among *Pseudomonas* infection, 28.5% showed resistance to Quinolones, 28.5% to Co-amoxiclav, 42.8% to Vancomycin, 28.5% to Clindamycin, 71% to Imipenem, 86% to Aminoglycosides and 43% to Cephalosporins. Out of 32 patients with *E. coli* infection, 31% showed resistance to Quinolones, 12.5% to Co-amoxiclav, 40.6% to Vancomycin, 31% to Clindamycin, 81% to Imipenem, 69% to Aminoglycosides and 34% to Cephalosporins (Table-3).

Among *Klebsiella* infection, resistance to Quinolones, Co-amoxiclav and Vancomycin was 25% each. 50% of *Klebsiella* was resistant to Clindamycin, 75% to Imipenem, 75% to Aminoglycosides and 25% to Cephalosporins. Among patients with other organisms, 68 % showed resistance to Quinolones, 63% to Co-amoxiclav, 58% to Vancomycin, 36.8% to Clindamycin, 63% to Imipenem, 63% to Aminoglycosides and 31.5% to Cephalosporins.

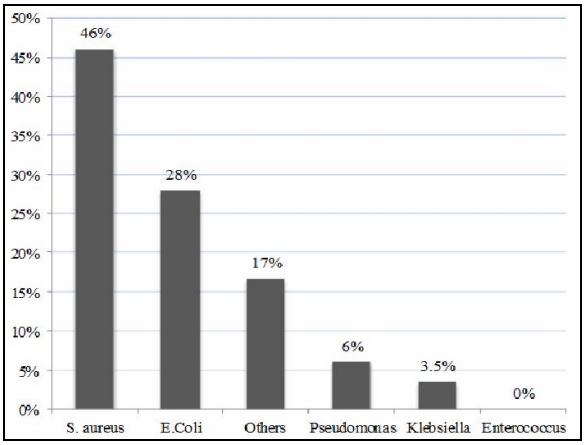

Figure-1: Frequencies of various organisms isolated from the diabetic foot patients (n=114)

Table-1: Demographic details and risk factors in patients with diabetic foot (n=114)

Variable	n (%)
Age (years) Mean+SD (range)	55+11.96 (20–80)
Gender	
• Males	38 (33.33)
• Females	76 (66.67)
Glycaemic control	
• Un-satisfactory	75 (65.8)
• Satisfactory	39 (34)
Sensory neuropathy	107 (93.86)
Peripheral vascular disease	52 (46)
Previous hospitalization	18 (15.79)

Table-2: Organisms identified and their susceptibility to various anti-microbial agents (n = 114)

Organism identified	Quinolones	Co-Amoxiclav	Vancomycin	Clindamycin	Imipinem	Amino-glycoside	Cephalosporins
S. aureus (n = 52)	14 (26.9%)	22 (43.3%)	48 (92.3%)	35 (67.3%)	44 (84.6%)	28 (53.8%)	8 (15.4%)
E. coli (n = 32)	10 (31.3%)	4 (12.5%)	13 (40.6%)	10 (31.3%)	26 (81.3%)	22 (68.8%)	11 (34.4%)
Pseudomonas (n = 7)	2 (28.6%)	2 (28.6%)	3 (42.9%)	2 (28.6%)	5 (71.4%)	6 (85.7%)	3 (42.9%)
Klebsiella Spp. (n = 4)	1 (25%)	1 (25%)	2 (50%)	2 (50%)	3 (75%)	3 (75%)	1 (25%)
Enterococcus Spp. (n = 0)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Others (n = 19)	13 (68.4%)	12 (63.2%)	11 (57.9%)	7 (36.8%)	12 (63.2%)	12 (63.2%)	6 (31.6%)
Total (n = 114)	40 (35.1%)	41 (36%)	77 (67.5%)	56 (49.1%)	90 (78.9%)	71 (62.3%)	29 (25.4%)

Table-3: Resistance of Organisms isolated from diabetic foot to various antimicrobial agents (n=114)

	Quinolones	Co-Amoxclav	Vancomycin	Clindamycin	Imipinem	Aminoglycoside	Cephalosporin
S. aureus **(n=52)**	38 (73.1%)	30 (57.7%)	4 (7.7%)	17 (32.7%)	8 (15.4%)	24 (46.2%)	44 (84.6%)
Pseudomonas **(n=7)**	5 (71.4%)	5 (71.4%)	4 (57.1%)	5 (71.4%)	2 (28.6%)	1 (14.3%)	4 (57.1%)
Enterococcus Spp. **(n=0)**	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
E. coli **(n=32)**	22 (68.8%)	28 (87.5%)	19 (59.4%)	22 (68.8%)	6 (18.8%)	10 (31.3%)	21 (65.6%)
Klebsiella Spp. **(n=4)**	3 (75%)	3 (75%)	2 (50%)	2 (50%)	1 (25%)	1 (25%)	3 (75%)
Others (n=19)	6 (31.6%)	7 (36.8%)	8 (42.1%)	12 (63.2%)	7 (36.8%)	7 (36.8%)	13 (68.4%)
Total (n=114)	74 (64.9%)	73 (64%)	37 (32.5%)	58 (50.9%)	24 (21.1%)	43 (37.7%)	85 (74.6%)

DISCUSSION

Diabetic foot ulcers are more prone to bacterial infections that spread rapidly, leading to irreversible tissue damage due to impaired immune function and blunted macrophage phagocytosis. Complications usually begin with an unrecognized foot ulcer in a patient with an insensitive foot that gets infected, leading to significant morbidity. This may lead to lower extremity amputations if not treated timely and properly.

In diabetic foot infections, the patterns of microbial infection are not consistent. Hence repeated evaluation of causative organisms and their antibiotic susceptibility is required for the selection of appropriate empirical therapy. The progression of infection in diabetic foot occurs as a result of suppressed immune status, delayed diagnosis, underestimation of extent of infection, and suboptimal antimicrobial therapy.[14]

The judicious use of antibiotics in diabetics leads to development of drug resistant organisms, so it is preferred to treat only the clinically infected wounds and use the narrowest-spectrum antimicrobial agent possible. Also, failure to treat diabetics with diabetic foot appropriately can lead to poor outcome in the form of sepsis or limb amputation. The clinician should decide about the empirical therapy on the basis of regional data available showing prevalence of causative organisms and also considering the local antibiotic resistance patterns, in particular considering the possibility of MRSA.[15,16] The current study shows that in general both gram-positive and gram-negative species are isolated from diabetics with moderate to severe diabetic foot infections who have not received the antimicrobial therapy.

The mean age observed in this study was 55.11 years. The mean duration of diabetes was 7.98 years. Nyamu *et al* found the mean age of 56.9 years in an African study.[17] Several studies have been conducted at different diabetic centres having variation in quality of patient care. Hence from the comparable figures of mean age in these studies one may conclude that there are certain contributing factors for diabetic foot that are time dependent. The diabetics from various regions have these factors in common irrespective of the environment. However, the mean age of onset of diabetes varies in different continents all over the world.

Among the patients presenting with diabetic foot, 66.67% were males and 33.33% were females. This shows higher number of males as compared to females presenting with diabetic foot. An international study conducted by Mohan Soundaram *et al* also showed that prevalence of diabetic foot ulceration is 65% in males and 35% in females.[18] The possible reasons could be the improper hygiene, lack of foot care and type of foot wear among males. Also, in our country males have better access to health care facility as compared to females.

Early recognition and management of the contributing factors responsible for the development and poor healing of diabetic foot ulcers is required to reduce the morbidity in these patients. The most important of these risk factors are past history of foot ulceration, peripheral neuropathy (that leads to loss of protective sensation in diabetics), deformity of bony prominences and peripheral arterial disease. The role of these risk factors has been confirmed by the results of a study conducted by Davis *et al*.[19] It was a community-based study that included 1300 Type 2 Diabetics. He found the incidence of limb amputation to be 3.8 per 1000 patient-years. Amputation was found to be associated with foot ulceration, ankle brachial index of less than 0.9, raised HbA1C levels and peripheral neuropathy.

In this study, the prevalence of sensory neuropathy was 93.86 % and it was more common in 40–60 years of age group. A South Indian study conducted by Dhanasekaran *et al* showed that diabetic neuropathy was seen in 63.2% of diabetic foot ulcers.[20] Neuropathy is a micro-vascular complication common in diabetics with poor glycaemic control. The development of neuropathy can be deferred by appropriate glycaemic control. The diabetics with neuropathy have been found to have prolonged mean duration of ulcers, advanced stage of ulcers and poor glycaemic control indicated by raised HbA1c. The significance of good glycaemic control should be considered as an important aspect of primary prevention in the management plan of diabetic foot ulcers. The earlier identification of neuropathy before the development of its complications is the key factor to prevent

diabetic foot infections.[21]

Glycaemic control in our study population was unsatisfactory in 65.8% patients and 34% patients had satisfactory glycaemic control. Study conducted by Nyamu PN *et al* showed generally poor glycaemic control in maximum number of patients, i.e., 18.3% of the patients had HbA1c <7%.[22] Females had poor glycaemic control than males. In this environment, women are still underprivileged and dependent on husbands for financial support and approach to healthcare facility. Patients with diabetes are less tolerant to infections and this is adversely affected by poor glycaemic control. Thus, a repetitive cycle is established with worsening hyperglycaemia that further impairs the response of diabetics to infection.[23]

Microbiologically, diabetic foot infections are generally polymicrobial.[23] The microbiological yield of diabetic foot wounds varies according to the extent of infection and foot involvement. The superficial diabetic foot infections are usually secondary to aerobic *gram-positive cocci*. However, the ulcers which are deep, chronic, or previously managed with antibiotics are more likely to be poly-microbial. Such wounds may bear the *Enterococci*, *Enterobacteriaceae*, *Pseudomonas aeruginosa* and anaerobes in addition to commonly seen organisms in diabetic foot.[24]

In this study, the most common microorganisms obtained from lesions were *S. aureus*, *group-B Streptococci*, *Enterococci*, anaerobic bacteria and enteric gram-negative pathogens. *Staphylococcal* species comprised 45.6% of all isolates recovered from the foot ulcers of diabetic patients. The prevalent *S. aureus* in this study is in concordance with the results and findings of Karchmer *et al* (76%) and Gu GH et al (78%).[25,26] However, our study shows the comparatively lower figure. This difference could be explained by the variation in causative organisms according to extent of ulcer, chronicity of ulcer and host defence factors.

E. coli was second most common isolated organism in 28.07% of samples in our study. Usually the deep and limb threatening infections are poly-microbial. The causative organisms being aerobic gram-positive cocci and gram-negative bacilli (e.g. *Escherichia coli*, *Klebsiella* species and *Proteus*). We further observed that both gram-positive and gram-negative infections were frequent in the studied population. In previous reports, researchers have shown the predominance of gram-positive infections in their regions. Similar observations were reported in a study conducted on a Southern Indian population.[27] However in this study, anaerobes were found to be far less than previously reported. The possible reasons could be delayed transportation of samples to the laboratory or inappropriate sampling technique.

The higher incidence of *E. coli* in diabetic foot infections has been observed. The possible reason could be previous use of antimicrobial agent. The prior exposure of patients to antimicrobials might also explain the higher prevalence of drug resistant *S. aureus*. *E. coli* was the second most common isolated bacteria from the diabetic foot in this study. A study conducted by Varaiyah et al in India showed *E. coli* and *Klebsiella* to be most frequent isolates.[28]

Among the anti-microbial susceptibility, Vancomycin and Imipenem were observed to be most effective against *S. aureus*, *Klebsiella* and gram-negative aerobes. The administration of Vancomycin in diabetic nephropathy may bear side effects in view of its exclusive excretion by kidneys. Hence, there is need to adjust dose accordingly. As an alternate, many other antibiotics can be used for this purpose. These include Gentamicin, Co-amoxiclav, 3rd generation Cephalosporins, Clindamycin, other Aminoglycosides and Quinolones. All these drugs showed different level of sensitivity and resistance to various organisms identified. Third generation cephalosporin is almost ineffective against *S. aureus*. Aminoglycosides have good sensitivity against *Pseudomonas*; *E. coli* and to some extent against *S. aureus*. But aminoglycosides are potentially nephrotoxic particularly in diabetic patients with underlying diabetic nephropathy or renal disease.

Quinolones should not be used as single empirical anti-microbial agent in view of their insufficient activity against *S. aureus, Streptococci* and *Klebsiella.* The clinician should be aware of the common causative organisms in diabetic foot infections and their antimicrobial susceptibility pattern in order to administer appropriate empirical antimicrobial therapy before the individual's own culture and sensitivity report is available.

Hence in current scenario, there is no single antibiotic that can cover all the organisms and thus combination of drugs has to be used keeping in view the multi-drug resistance. The emergence of resistant strains represents a compounding problem standing against the efforts to prevent amputation as infection is the single most common cause leading to amputations. Even if the microorganism is sensitive to one particular antimicrobial agent, the drug is unlikely to attain therapeutic concentration at the site of infection because of virulence factors that include proteases, haemolysins, collagenases and short-chain fatty acids. Thus, leading to inflammation and impaired wound healing. This contributes further to the chronicity of infection. Yao *et al* suggested that

biofilms may be formed in diabetic foot that impairs penetration of anti-microbial agents into the infected area.[29]

Foot infections of diabetic patients are initially managed by empirical therapy against likely causative organisms. The incidence of osteomyelitis and amputation of limb will decrease drastically provided that diabetic foot infections are recognized early and treated vigorously. A patient who has one episode of diabetic foot infection has higher chances to develop another infection. Hence preventive action at an early stage can reduce the further risk. Empirical antibiotics should be guided by the category of foot infection, available regional microbiological data and various host factors (i.e., neuropathy and peripheral arterial disease).

We recommend the use of molecular tools for diagnosis of bacterial infection only in such situations where suspicion of infection is high despite the negative culture. Application of advanced techniques, such as rDNA PCR, ERIC PCR etc., to evaluate the infection status and bacterial diversity of the isolates in diabetic foot wounds has been suggested in the literature. Measurement of inflammatory markers has also been used to differentiate the infected from non-infected foot ulcers in of diabetics. However, positive yield of culture sensitivity will always receive priority over the molecular study results for the selection of antibiotics. If we have knowledge regarding the characteristics of infection, i.e., the type of bacteria commonly found and the laboratory evidence of infection, the selection of anti-microbial agent may be close to appropriate, even if the culture reports are not obtained while initiating the antibiotic therapy.[30]

CONCLUSION

The prevalence of gram-positive infection is higher in diabetic foot patients from our region. *Staphylococcus aureus* was the most frequent isolate among gram-positive organisms while *E. coli* among the gram-negatives. In view of drug resistance, Vancomycin and Imipenem are the drugs of choice for gram positive cocci; and Imipenem and Aminoglycosides for gram negative infections. The selection of the antibiotic treatment should be based on the predominant organisms isolated and antimicrobial susceptibility patterns. This would improve the overall antibiotic efficacy and reduce the emergence of multidrug resistant organisms in diabetics.

AUTHORS' CONTRIBUTION

Each of the authors contributed to data collection; write up, statistics, literature review and referencing.

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