Bacteriological study of diabetic foot infection in Egypt

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Introduction
Foot infections are one of the major complications of diabetes mellitus and are a significant risk factor for lower extremity amputation. Providing effective antimicrobial therapy is an important component in treating these infections. This study assesses the microbial isolates of patients with diabetic foot infections and their antibiotic susceptibility pattern.

Patients and methods
A prospective study of 75 patients with diabetic foot infections admitted to Al-Azhar university hospitals was undertaken. Bacteriological specimens were obtained and processed using standard hospital procedure for microbiological culture and sensitivity testing.

Results
Overall, 40 (54%) patients had subcutaneous infections, 22 (29%) had infected superficial ulcers, seven (9%) had infected deep ulcers involving muscle tissue, and six (8%) patients had osteomyelitis. A total of 99 pathogens were isolated. Forty percent of patients had polymicrobial infection, 39 (52%) had single organism infections, and six (8%) had no growth. Gram-negative bacteria (67%) were more commonly isolated compared with Gram-positive bacteria (30%). The three most frequently found Gram-positive organisms were *Staphylococcus aureus* (10.2%), *Streptococcus pyogenes* (7.1%) and methicillin-resistant *S. aureus* (7.1%), and the most common Gram-negative organisms were *Pseudomonas aeruginosa* (19.4%), *Klebsiella pneumoniae* (15.3%), and *Acinetobacter* spp. (10.2%). Vancomycin was found to be the most effective against Gram-positive bacteria, whereas imipenem and amikacin were most effective against Gram-negative bacteria on antibiotic testing.

Conclusion
Forty percent of diabetic foot infections were polymicrobial. *S. aureus* and *P. aeruginosa* were the most common Gram-positive and Gram-negative organisms, respectively. This study helps us to choose empirical antibiotics for patients with diabetic foot infections.

Keywords:
bacteriological, diabetic foot, Egypt

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Introduction

Diabetic foot is one of the most feared complications of diabetes and is the leading cause of hospitalization in diabetic patients. Diabetic foot is characterized by several pathological complications such as neuropathy, peripheral vascular disease, foot ulceration, and infection with or without osteomyelitis, leading to the development of gangrene and even necessitating limb amputation [1,2]. Diabetic patients have a lifetime risk as high as 25% for developing foot ulceration [3]. Diabetic ulcers have 15–46 times higher risk of limb amputation when compared with foot ulcers due to other causes [4]. Every year, more than a million diabetic patients require limb amputation [1].

Infected foot ulcer is a common cause of morbidity in diabetic patients, ultimately leading to dreaded complications such as gangrene and amputations. The lifetime risk to a person with diabetes for developing a foot ulcer could be as high as 25% [3]. Infection is most often a consequence of foot ulceration, which typically occurs after trauma to a neuropathic foot [5].

In Egypt, 12% of the adult population (aged 10–79 years) has diabetes. However, because Egypt has a relatively young population, this is corrected to 15% when used to compare with other countries (Fig. 1) [6].

The alarming fact is that Egypt has more diabetic individuals than any other country [7], and the incidence of foot problems and amputations remains very high, accounting for up to 20% of diabetes-related hospital admissions. This can be easily attributed to several practices prevalent in Egypt, such as barefoot walking, inadequate facilities for diabetes care, low socioeconomic status, and illiteracy [8].

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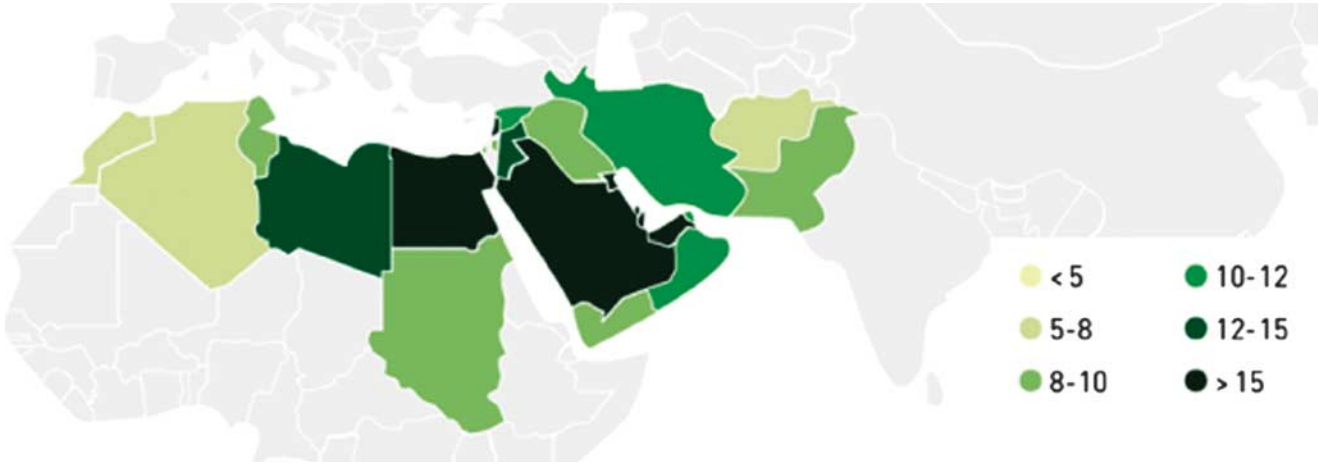
Figure 1



Prevalence (%) estimates of diabetes (20–79 years) in 2011 in the Middle East and North Africa region [4].

The incidence of type 2 diabetes is rising to epidemic proportions in Egypt as well as worldwide [3]. Because of its relatively low case fatality rate, the prevalence of associated chronic complications is expected to increase. The burden of diabetic foot is set to increase further in the future as its contributory factors such as peripheral neuropathy and peripheral vascular disease are present in more than 10% of cases at the time of diagnosis [9].

Infection may be caused by pathogenic bacteria originating from the external environment as well as by bacteria forming physiological microflora of the skin (e.g. *Staphylococcus epidermidis*, *Staphylococcus aureus*, and *Propionibacterium acnes*). Pathogenic microflora is often transferred unconsciously by medical personnel and materials and substances used for treatment. Both chronic venous ulceration and diabetic ulcerations may generally be deemed colonized, although such wounds do not always show clinical signs of infection in every case. The presence of infection depends mainly on the number of microorganisms residing in the wound, whereas the healing process depends on the type of bacterial strains and their pathogenicity [9].

Ulcerations are prone to colonization by nearly every microorganism that can come in contact with their surface. Usually ulcerations contain mixed flora, consisting of several strains of bacteria. Most often these are aerobic bacteria, such as *S. aureus*, *Streptococcus pyogenes*, *Escherichia coli*, *Proteus mirabilis*, *Pseudomonas aeruginosa*, and anaerobic bacteria, for example, *Bacteroides fragilis*, *Clostridium perfringens*, *Peptostreptococcus* spp., and *Prevotella oralis*. The mechanism by which bacteria delay the healing process is not completely understood [10]. It is suggested that the main role is played by the pathogenic virulence factors of bacteria, such as:

(1) Bacterial adhesions, proteins present on the surface of bacterial cells are responsible for their adhesion to host cells, allowing colonization of the ulceration.
(2) Exoenzymes decomposing cell materials such as collagen and fibrinogen, allowing deeper penetration to tissues and modifying bacterial resistance.
(3) Toxins, protein substances released from bacterial cells responsible for clinical signs of infection.

The impaired microvascular circulation in patients with diabetic foot limits the access of phagocytes, favouring development of infection. *E. coli*, *Proteus* spp., *Pseudomonas* spp., *S. aureus*, and *Enterococcus* spp. are the most frequent pathogens contributing to progressive and widespread tissue destruction. Diabetic foot infections are often polymicrobial. Methicillin-resistant *Staphylococcus aureus* (MRSA) can be commonly isolated from 10–40% of diabetic wounds. The increasing association of multidrug-resistant pathogens with diabetic foot ulcers further compounds the challenge faced by the physician or surgeon in treating diabetic ulcers without resorting to amputation. Infection with multidrug-resistant pathogens is also responsible for the increased duration of hospitalization, cost of management, morbidity and mortality of the diabetic patients [11].

In addition, these substances inhibit migration and fibroblast activity. Moreover, infection decreases the amount of oxygen that is available for the process of collagen synthesis. In extreme cases, in which there is lack of proper treatment, significant colonization of the wound may take place, which leads to infection of the ulceration, its enlargement and tissue necrosis. Infected ulcerations may also be a source of generalized infections, such as inflammation of lymph vessels or sepsis, which may eventually require amputation of the limb [11].

From the point of view of the presence of bacteria in the ulceration and clinical signs of inflammation, wounds may be divided into three groups:

(1) Uncolonized ulcerations without clinical signs.
(2) Colonized ulcerations without visible clinical signs.
(3) Infected ulcerations with clinical signs of infection.

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There are numerous articles in the literature on microbiological study of diabetic foot infections along with their susceptibility patterns for antibiotic therapy from different parts of the world. However, no such data are available for Egypt. In view of the above facts, a prospective study was carried out to determine the relative frequency of aerobic microbial isolates cultured from community-acquired diabetic foot infections and to assess their comparative in-vitro susceptibility to the commonly used antibiotics.

Patients and methods

A prospective study was carried out at the Department of General Surgery, Al-Azhar University Hospitals (Egypt). A total number of 75 diabetic foot patients were admitted for surgical management. The patients were admitted to the hospital for the management of infected diabetic foot, ranging from ulcer to osteomyelitis.

The demographics of the participants such as age, sex, diagnosis, and complications of diabetes were collected. Clinical data and details on the examination requested, identity of microbes, and antimicrobial therapy were then extracted from the files of all patients.

Diabetic foot ulcers were classified according to Wagner's Classification and the University of Texas Wound Classification System [12].

Wagner's Classification of Diabetic Foot Ulcers

Grade 0: no ulcer in a high-risk foot.

Grade 1: superficial ulcer involving the full skin thickness but not underlying tissues.

Grade 2: deep ulcer, penetrating down to ligaments and muscles, but no bone involvement or abscess formation.

Grade 3: deep ulcer with cellulitis or abscess formation, often with osteomyelitis.

Grade 4: localized gangrene.

Grade 5: extensive gangrene involving the whole foot.

University of Texas Wound Classification System of Diabetic Foot Ulcers

Grade IA: noninfected, nonischemic superficial ulceration.

Grade IB: infected, nonischemic superficial ulceration.

Grade IC: ischemic, noninfected superficial ulceration.

Grade ID: ischemic, infected superficial ulceration.

Grade IIA: noninfected, nonischemic ulcer that penetrates to capsule or bone.

Grade IIB: infected, nonischemic ulcer that penetrates to capsule or bone.

Grade IIC: ischemic, noninfected ulcer that penetrates to capsule or bone.

Grade IID: ischemic and infected ulcer that penetrates to capsule or bone.

Grade IIIA: noninfected, nonischemic ulcer that penetrates to bone or a deep abscess.

Grade IIIB: infected, nonischemic ulcer that penetrates to bone or a deep abscess.

Grade IIIC: ischemic, noninfected ulcer that penetrates to bone or a deep abscess.

Grade IIID: ischemic and infected ulcer that penetrates to bone or a deep abscess.

Specimens of pus were collected during initial admission to the hospital (provided that no antibiotics were taken within the past 2 days). They were collected by swabbing directly at the base of the infected wound, and similarly, for those who required surgical intervention, pus swabs were taken intraoperatively at the deepest part of the wound. The specimens were obtained using sterile, commercially purchased swabs and transported to the microbiology laboratory immediately. All pus swabs were Gram stained for direct examination. They were cultured on blood agar plates, on MacConkey medium and in a tube of enriched broth culture. The media were incubated at 37° overnight. The broth culture was further subcultured onto the same above-mentioned solid media after overnight incubation, and the plates were incubated aerobically.

The Gram-negative colonies were further identified using the API system (Biomerieux, Paris, France). Staphylococcal isolates were additionally tested for coagulase enzyme production to confirm the presence of *S. aureus*. MRSA was confirmed by the slide latex agglutination test for rapid detection of PBP2 (MRSA screen; Denka Seiken Co., Ltd, Tokyo, Japan). Streptococci isolated were further grouped according to their respective sera (A, B, C, D, and G).

All organisms isolated were subjected to antibiotic sensitivity testing by the Kirby–Bauer disc diffusion method using commercially purchased antibiotic discs and interpreted according to Clinical and Laboratory Standard recommendations. All patients received proper antibiotics according to the culture and sensitivity results as well as metronidazole for associated anaerobic organisms.

Results

The present study included 75 patients, of which 37 were males and 38 were females with the male to female ratio being almost equal. The age ranged from 27 to 72 years, and the mean age was 48 years. Diabetic foot infections were the highest among the age group of 51–60 years, followed by the 41–50 years age group (Table 1).

Diabetic complications were searched for by consulting different specialties (Table 2). The degree and extension of diabetic foot wound were classified in all patients

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Table 1 Demographics of patients

	Age group (years)						Total	
	21–30	31–40	41–50	51–60	61–70	Above 70	Frequency	%
Male	1	4	11	14	6	1	37	49
Female	2	8	8	14	4	2	38	51
Total	3	12	19	28	10	3	75	100

Table 2 Diabetic complications in 75 patients infected with diabetic foot ulcers

Diabetic complication	Value [*n* (%)]
Retinopathy	58 (77.3)
Cardiopathy	53 (70.6)
Nephropathy	48 (64)
Neuropathy	40 (53.3)
Gastropathy	20 (26.6)
Vasculopathy	55 (73.3)
Poor glycemic control[a]	42 (56)

[a]HbA1c ≥ 8.0%.

Table 3 Distribution of patients (%) according to the Wagner and the University of Texas Wound Classification Systems

Classification system	Patients [*n* (%)]
Wagner Classification of Diabetic Foot Ulcers	
Grade 0	0 (0)
Grade 1	10 (13.3)
Grade 2	20 (26.7)
Grade 3	18 (24)
Grade 4	16 (21.3)
Grade 5	11 (14.7)
University of Texas Wound Classification System of Diabetic Foot Ulcers	
Stage A	
Grade I	2 (2.6)
Grade II	6 (8)
Grade III	6 (8)
Stage B	
Grade I	3 (4)
Grade II	7 (9.3)
Grade III	6 (8)
Stage C	
Grade I	4 (5.3)
Grade II	6 (8)
Grade III	8 (10.7)
Stage D	
Grade I	5 (6.7)
Grade II	9 (12)
Grade III	13 (17.4)

according to Wagner and the University of Texas Wound Classification Systems (Table 3).

Overall, 52% ($n = 39$) of the cultures revealed the presence of a single organism, 40% ($n = 30$) had mixed infections, and 8% ($n = 6$) did not show any growth. With regard to clinical severity, 54% ($n = 40$) of the infections involved the subcutaneous level, 29% ($n = 22$) involved superficial ulcers, 9% ($n = 7$) involved deep ulcers, and 8% ($n = 6$) had osteomyelitis (Fig. 2).

The data in Table 4 show the profile of the pathogens isolated. A total of 98 pathogens were identified with an average of 1.31 organisms per patient. Among the aerobic microbes, Gram-negative bacteria ($n = 66$, 67.3%) were seen to be more commonly isolated than Gram-positive bacteria ($n = 29$, 29.6%).

The data in Table 5 show the combination of organisms in mixed infections. The results of the sensitivity patterns of the five commonly detected Gram-positive and Gram-negative pathogens are shown in Tables 6 and 7.

Discussion

This study revealed that 40% of diabetic foot infections were polymicrobial in nature. There were more Gram-negative pathogens isolated when compared with Gram-positive bacteria, with a ratio of about 2 : 1, and they were sensitive mostly to vancomycin and amikacin, respectively.

Our findings showed a relatively fewer number of patients (40%) were infected by two or more pathogens compared with 52% of patients who had a monomicrobial etiology. Raja [13] reported that 42% of patients developed mixed growth. Similarly, Renina *et al.* [14] reported that 58.9% of cases were polymicrobial in nature. Other studies from Jamaica and France document that the prevalence of polymicrobial infection could be as high as 80–87.2% [15,16]. A possible reason for the low incidence of polymicrobial infection in the present study could be clinically mild and superficial subcutaneous infections.

Overall, Gram-negative microbes were the predominant pathogens isolated and this has also been observed in Indian studies by Bansal *et al.* [17], by Shankar *et al.* [8], and by Gadepalli *et al.* [11] (76 vs. 24%, 57.6 vs. 42.3%, and 51.4 vs. 33.3%, respectively). Raja [13] and Renina *et al.* [14] also documented more Gram-negative bacteria than Gram-positive bacteria (52 vs. 45% and 67 vs. 33%, respectively). Thus, it is essential to select antibiotics that are more effective against Gram-negative bacteria in contrast to Gram-positive organisms, which clinicians are inclined to prescribe on observing deep tissue infection or infected gangrene. *P. aeruginosa* (19.4%), *Klebsiella pneumoniae* (15.3%) and *Acinetobacter* spp. (10.2%) were the majority of the causative Gram-negative microorganisms. Among the Gram-positive microorganisms, *S. aureus* (10.2%), *S. pyogenes* (7.1%), and MRSA (7.1%) were more predominantly isolated. These pathogens were believed to have colonised the superficial foot ulcers. These results are comparable with those of Raja [13], of Renina *et al.* [14], and of Bansal *et al.* [17] (Table 8).

With regard to the susceptibility patterns, vancomycin and amikacin appeared to be the best antibiotics for therapy against Gram-positive and Gram-negative organisms, respectively. Vancomycin is usually only indicated for the treatment of MRSA, whereas amikacin is associated with nephrotoxicity, which can deteriorate patients who already have pre-existing diabetic nephropathy.

Based on the results shown in Tables 6 and 7, we could also assume that monotherapy may not be the best management for causal microbes. Thus, the choosing empiric antibiotic therapy for diabetic foot infections can be based on a number of conditions: (a) the severity of

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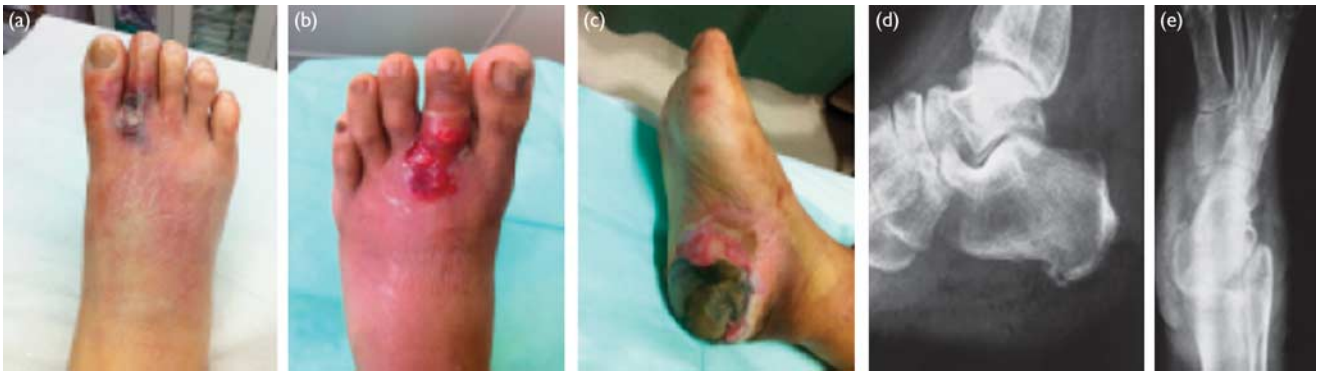
Figure 2



Progression of diabetic foot infections from superficial to subcutaneous infections, osteomyelitis and eventually amputation: (a) infection of the second toe with cellulites, (b) progression to deeper cutaneous infection with exudation of blood-stained serious fluid, (c) deep subcutaneous infection with necrotic slough of the heel and (d) radiographs showing osteomyelitis changes with subcutaneous gas collection (lateral view), and (e) superior inferior view.

Table 4 Types and profiles of organisms isolated in patients with diabetic ulcers

Organisms	Frequency (%)	Organisms	Frequency (%)
Gram-positive aerobes	29 (29.6)	Gram-negative aerobes	66 (67.3)
Staphylococcus aureus	10 (10.2)	*Pseudomonas aeruginosa*	19 (19.4)
Streptococcus pyogenes	7 (7.1)	*Klebsiella pneumoniae*	15 (15.3)
MRSA	7 (7.1)	*Acinetobacter* spp.	10 (10.2)
Group D streptococcus	4 (4.1)	*Proteus mirabilis*	6 (6.1)
Streptococcus pneumoniae	1 (1.0)	*Escherichia coli*	4 (4.1)
Fungus		*Enterobacter cloacae*	3 (3.1)
Candida albicans	1 (1.0)	*Chryseomonas luteola*	3 (3.1)
		Proteus vulgaris	2 (2.0)
		Citrobacter spp.	2 (2.0)
		Alcaligenes faecalis	1 (1.0)
		Pseudomonas cepacia	1 (1.0)

MRSA, methicillin-resistant *Staphylococcus aureus.*

Table 5 Combinations of organisms isolated from patients with mixed infections

Combination of organisms	Frequency (%)
Gram-positive and gram-positive	2 (7)
Gram-positive and gram-negative	12 (40)
Gram-negative and gram-negative	16 (53)
Total	30 (100)

infection, (b) the depth and extent of involvement of infection, and (c) the local pattern of bacterial etiology and their antibiogram.

In the present study, the severity of infection was proportionate with the depth of infection, and the majority of infections were categorized as being superficial or subcutaneous. Mild infection is usually monomicrobial in etiology, and the most common causative organism is *S. aureus* [19], which is 100% sensitive to flucloxacillin (oxacillin) and amoxy/clavulanic acid (Table 6).

If the infection involves deeper tissues, it could be polymicrobial in nature and more likely to be due to Gram-negative microorganisms in different combinations. Hence, the infection can be treated with amoxy/clavulanic acid, ampicillin/sulbactam, and cefuroxime. If the infection is severe and involves deep tissue and bone, ceftazidime, imipenem, meropenem, and levofloxacin are more appropriate, with their sensitivities reaching 98–100%.

There are several limitations in this study that need to be taken into account when interpreting its results. First, the sample size was notably small, with only 75 patients (as the capacity of the surgical department beds is limited), which may limit the power of the study. Second, the method of specimen collection was based on current practice and may not be standardized. All of the specimens evaluated here were collected from pus swabs. However, there are reports that have shown that sampling of bone and soft-tissues is more sensitive compared with sampling from pus swabs alone [20,21]. Another limitation is the prospective nature of the study, which is always a major drawback in the regular follow-up of patients. However, given that the follow-up data are not regular, this study still provides important information and serves as a basis for future studies.

Several studies have also investigated the relationship between the specimen collection method and both numbers and types of organisms recovered from infected wounds. Some studies have reported that tissue specimens are more sensitive and specific, containing fewer

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Table 6 Sensitivity patterns of all isolated Gram-positive organisms

Antibiotics	*Staphylococcus aureus* (*n*=10)	*Streptococcus pyogenes* (*n*=7)	MRSA (*n*=7)	Group D streptococcus (*n*=4)	*Streptococcus pneumonia* (*n*=1)
Vancomycin	100	100	100	100	100
Linzolid	100	–	100	–	–
Penicillin	10	100	0	100	100
Oxacillin	100	100	0	50	100
Erythromycin	70	60	0	50	100
Fusidic acid	80	80	0	50	–
Amoxy/clavulanic acid	100	99	0	100	100
Ampicillin/ Sulbactam	90	100	0	100	100
Gentamicin	90	90	0	100	100
Netilmicin	90	80	0	100	100
Amikacin	100	100	0	100	100
Cephalexin	100	95	0	25	-
Cefuroxime	100	99	0	50	100
Ceftriaxone	90	97	0	100	100
Chloramphenicol	90	100	40	50	100
Imipenem	100	100	20	100	100
Meropenem	80	100	20	100	100
Tetracycline	70	43	10	25	0
Levofloxacin	90	80	90	50	100
Sulfa/trimethoprim	99	70	80	0	100

MRSA, methicillin-resistant *Staphylococcus aureus.*

Table 7 Sensitivity patterns of all isolated Gram-negative organisms

Antibiotics	*Klebsiella* spp. (*n*=19)	*Pseudomonas* aeruginosa (*n*=15)	*Acinetobacter* (*n*=10)	*Proteus* (*n*=8)	*Escherichia coli* (*n*=4)	*Enterobacter* (*n*=3)	*Chryseomonas* spp. (*n*=3)
Ampicillin	100	10	5	65	75	5	5
Ampicillin/ sulbactam	90	30	40	100	75	45	10
Amoxy/ clavulanic acid	100	20	15	100	100	55	20
Cephalexin	90	10	50	90	100	25	10
Cefuroxime	90	20	40	100	100	56	10
Ceftazidime	100	95	50	100	100	83	50
Chloramphenicol	80	30	50	90	100	85	10
Gentamicin	95	100	70	90	100	90	90
Netilmicin	100	99	70	95	100	91	90
Amikacin	100	100	70	97	100	92	95
Tetracycline	50	30	30	50	100	60	5
Imipenem	100	100	55	95	100	97	98
Meropenem	100	100	55	89	100	98	98
Levofloxacin	100	90	50	85	100	90	95
Piperacillin	100	90	20	87	–	70	–
Sulfa/ trimethoprim	80	5	45	93	60	80	50
Polymyxin	–	98	100	–	–	–	–

Table 8 Sensitivity patterns of the all the isolated Gram-negative microorganisms

References	Gram-negative organisms (%)	Gram-positive organisms (%)
Raja [13]	*Proteus* spp. (28) *Pseudomonas aeruginosa* (25) *Klebsiella pneumoniae* (15) *Escherichia* coli (9)	*Staphylococcus aureus* (44) Group B streptococcus (25) *Enterococcus* spp. (9)
Lea *et al.* [18]	*Proteus* spp. (24) *Enterobacter* spp. (21) *Citrobacter* spp. (9) *E. coli* (12)	*S. aureus* (29) *Staphylococcus epidermidis* (3)
Bansal *et al.* [17]	*P. aeruginosa* (22) *K. pneumoniae* (17) *E. coli* (18) *Proteus* spp. (11)	*S. aureus* (19)

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apparent contaminants and more pathogens compared with swab cultures [22,23]. In contrast, others studies have reported that with adequate preliminary debridement, the use of a wound swab is as reliable as the use of a tissue specimen [23,24]. In our study, swab specimens were collected only after thorough cleaning with sterile normal saline, after debridement of the wound and before application of an antiseptic agent. Culture material obtained from deeper tissues only was sent for microbiological study. However, sample collection procedures need to be carefully defined and observed, as skin contaminants may alter the microbial profiles, possibly resulting in misinterpretation of culture reports, with adverse effects on clinical decisions.

The decision on proper management of diabetic foot infection is difficult and is still a matter of debate. Although optimal therapy is yet to be established, most authors agree that the management of these infections requires isolation and identification of the microbial flora; appropriate antibiotic therapy, according to the sensitivity patterns; precise selection and identification of the chronic complications and proper surgical intervention for these complications. Most diabetic foot infections are polymicrobial in nature, and mixed organisms are frequently encountered [25]. However, the spectrum of microorganisms depends mainly on microbial flora of the lower limb, metabolic factors, foot hygiene, and the use of antibiotics [26].

Emergence of resistance among organisms against the commonly used antibiotics has been clearly outlined in various studies as being largely due to their indiscriminate use [27]. There is a direct relationship between the total amount of a certain antibiotic used in a particular hospital during a certain period of time and the number of resistant strains that emerge [28].

Conclusion

Our study has showed that 40% of diabetic foot infections were polymicrobial. *P. aeruginosa* and *S. aureus* were the most commonly identified Gram-negative and Gram-positive microorganisms, respectively. Amikacin and vancomycin were the most effective antimicrobial therapy against Gram-negative and Gram-positive microorganisms, respectively. Levofloxacin and imepenem are also very effective in empiric treatment but are very expensive. Because of the limited suitability of these antibiotics, choosing empiric antibiotic therapy should depend upon the clinical features of the infections and the local pattern of bacterial etiology and its antibiogram.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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