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Original Research Article

Epidemiological, clinical, biological, therapeutic and evolutionary profiles of infected diabetic foot hospitalized in the endocrinology department of Joseph Raseta Befelatanana University Hospital in Antananarivo, Madagascar

Rija E. Raherison[1]*, Sitraka A. Raharinavalona[2], Thierry Razanamparany[1], Tantely G. L. Andriamitantsoa[3], Radonirina L. Andrianasolo[1], Andrianirina D. P. Rakotomalala[1]

[1]Department of Endocrinology, Joseph Raseta Befelatanana University Hospital (JRBUH), Antananarivo, University of Antananarivo, Madagascar
[2]Departments of Cardiovascular-Diseases and Internal Medicine, Soavinandriana Hospital Center (CENHOSOA), Antananarivo, University of Antananarivo, Madagascar
[3]Organisation Sanitaire Tananarivienne Inter Entreprises (OSTIE), Antananarivo Madagascar

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***Correspondence:**
Dr. Rija E. Raherison,
E-mail: rijaherison@yahoo.fr

ABSTRACT

Background: Diabetic foot infection is a serious risk of amputation and death. Our study aims to describe the epidemiological, clinical, biological, therapeutic and evolutionary profiles of patients hospitalized for infected diabetic foot.
Methods: This is a descriptive cross-sectional study conducted in the Endocrinology department of the Joseph Raseta Befelatanana University Hospital, Antananarivo over a period of 4 years. The diagnosis of infected diabetic foot was made according to the criteria of the French-Speaking Society of Infectious Pathology. We included all patients who had undergone bacteriological examination with antibiogram of their diabetic foot lesion.
Results: Fifty patients were retained, giving a hospital prevalence of 2.84%. Their mean age was 55.50±9.63 years, the sex ratio was 0.72 and mean duration of their diabetes was 6.87±6.13 years. The majority of the lesions were post-traumatic (28%). The mean hospital length stay was 42.82±35.26 days. Infections were classified as stage 4 for 72% of the patients. The main isolated bacteria were Staphylococcus aureus (54.69%), of which 83% were sensitive to Amoxicillin-Clavulanic Acid. Seventy percent of the patients were infected with multidrug-resistant bacteria. The amputation rate was 30% and the mortality rate was 2%.
Conclusions: Therapeutic education on podiatric care must be optimized. Multi-disciplinarity is essential for management. Amoxicillin-clavulanic acid should be preferred as probabilistic antibiotic therapy for diabetic foot infection in our country where bacteriological examination is often lacking.

Keywords: Amputation, Diabetic foot, Infection, Probabilistic, *Staphylococcus aureus*

INTRODUCTION

Diabetic foot infection corresponds to an invasion with multiplication of microorganisms, generally bacteria, leading to tissue damage with or without a systemic inflammatory response. It affects the anatomical structures located under the malleolus of a diabetic subject and often follows a skin ulceration.[1] Twelve to 25% of diabetics will suffer from foot ulceration during their life and 40 to 80% of these ulcerations will become infected.[2,3] Diabetic foot

infection multiplies the risk of amputation by a factor of 10 and can be life-threatening for the patient.1 Although the diagnosis of diabetic foot infections is clinical, knowledge of the involved bacteria is necessary in order to adapt antibiotic therapy and thus reduce the associated morbidity and mortality.[4]

In the European and American continents, Staphylococcus aureus is generally the main isolated pathogenic bacteria.[5] In Africa, the isolated bacteria are variable. In Cameroon, for example, Gram-negative bacteria (GNB) including Escherichia coli were in the majority.[6] On the other hand, in Burkina Faso, Gram-positive cocci (GPC), mainly Staphylococcus aureus, were the most frequently isolated.[7]

In Madagascar, we have few studies on diabetic foot infections, particularly on their bacteriological profile.

Thus, the aims of the present study are to describe the epidemiological, clinical profiles of patients hospitalized for infected diabetic foot, to identify the main responsible bacteria, to describe their sensitivity profile to common antibiotics and to report their therapeutic and evolutionary profiles at the hospital.

METHODS

Study design and patients

This was a descriptive cross-sectional study carried out in the endocrinology department of the Joseph Raseta Befelatanana University Hospital (JRBUH) from January 1, 2017 to December 31, 2020 (4 years), involving patients hospitalized for infected diabetic foot.

We included all patients with known or newly diagnosed diabetes, aged over 18 years, hospitalized for infected diabetic foot and who had undergone bacteriological examination with antibiogram of their lesion.

The diagnosis of infected diabetic foot was made according to the criteria of the French-Speaking Society of Infectious Pathology (SPILF), namely, the presence of at least two of the following signs: redness, warmth, swelling, pain, ulcer discharge or wet gangrene.[3]

Patients with incomplete medical records and patients whose bacteriological examination did not find any bacteria were excluded.

During the study period, two thousand two hundred and thirty-nine diabetic patients were hospitalized at the study site. Sixty-four were hospitalized for infected diabetic foot. Fourteen were excluded because either medical records were incomplete or the bacteriological examination of their lesion did not find any bacteria. In the end, 50 patients were retained for the study.

Variables studied

The epidemiological characteristics studied were age, gender, type of diabetes, duration of diabetes and cardiovascular risk factors associated with diabetes. Clinical characteristics included the triggering factor of the infection, the notion of systemic antibiotic therapy in the pre-hospital period, the time between the onset of the infection and hospitalization, the type of lesion and the severity of the infection. Biological characteristics included the blood glucose level on admission, the glycated haemoglobin (HbA1c) level, the number of leukocytes and neutrophils in the first complete blood count, the first C-reactive protein (CRP) level, the isolated pathogenic bacteria from the local sample for bacteriological analysis of the lesion, the result of the antibiogram and the antibiotic resistance profile of the isolated bacteria. Therapeutic characteristics provided information on whether or not amputation was used. The evolutionary characteristics reported the hospital length stay and its issue.

The positive diagnosis of newly diagnosed diabetes was made according to the American Diabetes Association (ADA) criteria.[8] Lesions were classified as superficial infected ulceration, phlegmon/abscess, osteitis/osteoarthritis and wet gangrene. Bone and/or joint involvement was suspected clinically based on bone exposure, an erythematous, edematous “sausage” appearance of a toe or abnormal mobility of a toe, bone palpable with a sterile blunt metal probe inserted in the depth of the ulcer.[3] The International Consensus on the Diabetic Foot classification with its 4 grades was used to define the severity of the infection.[3]

A single local bacteriological sample of the lesion was taken for each patient. All bacteriological samples were taken by swabbing the lesion after cleaning with physiological serum. Biological examinations were carried out in various laboratories according to their availability and the convenience of the patients. The bacteriological examination consisted of a microscopic examination before and after Gram staining then in a culture in an aerobic environment. The definition of a multi-resistant Bacteria (MRB) is retained by its non-sensitivity to at least one antibiotic in three or more categories of antibiotics.[9]

Amputation is surgery involving the removal of a limb or a segment of the limb.[10] It is "minor" if it involves the toes, rays and metatarsals and "major" if it involves the leg or thigh.[3]

Data processing and analysis

Data were collected from patients' medical records and processed by Epi Info ® software version 7. Statistical analysis consisted of a description of the distribution of patients according to the variables studied. Frequencies and percentages were chosen for qualitative variables and

continuous variables were presented as mean±standard deviation.

RESULTS

Hospital prevalence of infected diabetic foot found in our study was 2.85%.

The mean age of the patients was 55.50±9.63 years (30-70 years). Patients aged between 51 and 60 years and those between 61 and 70 years were the most representative (34 and 30% respectively). The sex ratio was 0.72. These demographic data were summarized in Table 1. All our patients were type 2 diabetics. Diabetes was discovered during hospitalization for 17 patients. The mean duration of diabetes for the 33 diabetics already known before hospitalization was 6.87±6.13 years (0.66-30 years). High blood pressure (HBP), age over 50 years for a man and 60 years for a woman as well as smoking were the main cardiovascular risk factors associated with diabetes with a respective frequency of 52%, 42% and 32%.

Clinically, direct trauma was the main triggering factor of diabetic foot infection (28%), followed by spontaneously occurring blisters (20%) and trauma caused by walking barefoot (12%). Twenty-six percent of patients had already received systemic antibiotic therapy before hospitalization. The mean time between the onset of infection and the admission of patients to the hospital was 42.80±35.26 days (7-365 days). Regarding the distribution of the type of lesion, twenty-six patients (52%), ten (20%), nine (18%) and 5 (10%) had respectively presented a phlegmon/abscess, a superficial infected ulceration, an osteitis and a wet gangrene. Seventy-two percent of the infections were classified as grade 4, eight percent as grade 3 and 20% as grade 2 according to the International Consensus on the Diabetic Foot.

Biologically, the mean blood glucose level on admission was 295±149 mg/dl (127-600 mg/dl). Forty-five patients underwent glycated haemoglobin

(HbA1c) testing, the mean value of which was 10±1.84% (7.4%-15.1%). Regarding the inflammatory assessment, the mean leukocyte count was 15.61±8.15 G/l (4-46 G/l) and that of polymorphonuclear neutrophils (PNN) was 12.670±8.24 G/l (0.1-43 G/l). The mean level of C-Reactive protein (CRP) was 101.25±83.50 mg/l (6-359 mg/l). On bacteriological examination, sixty-four different pathogenic bacteria were isolated, giving an average of 1.26 bacteria per lesion and per patient. The infection was mono-bacterial, bi-bacterial and tri-bacterial for 36 (72%), 12 (24%) and 2 (4%) patients respectively. These bacteria were dominated by GPC including Staphylococcus aureus (54.69%) and Enterococcus spp. (7.81%). Gram negative bacteria were less frequent, mainly Klebsiella pneumoniae (6.25%), Klebsiella oxytoca (4.69%) and Morganella morganii (4.69%) (Table 2). The distribution of the antibiogram results for the two main GPC isolated, namely Staphylococcus aureus and Enterococcus spp. was summarized in table 3. For Staphylococcus aureus, its susceptibility frequency was 83% for Amoxicillin-Clavulanic Acid, 80% each for Oxacillin and Gentamicin and 74% for Ciprofloxacin. For Enterococcus spp., its susceptibility frequency was 60% for Amoxicillin-Clavulanic Acid and 40% each for Amoxicillin and Gentamicin.

The distribution of the antibiogram results for the three mains isolated GNB, namely: *Klebsiella pneumoniae*, *Klebsiella oxytoca* and *Morganella morganii* was summarized in Table 4. All three enterobacteria were consistently susceptible to Amikacin and Imipenem. Seventy-five percent of *Klebsiella pneumoniae* were susceptible to Ciprofloxacin and 50% to Amoxicillin-Clavulanic Acid and Ceftriaxone respectively.

Table 1: Demographic data of patients (n=50).

Data	Number (n=50)	Proportion (%)
Mean age (in years)	55.5±9.63	
31-40	3	6
41-50	11	22
51-60	17	34
61-70	15	30
≥71	4	8
Male	21	42
Sex ratio	0.72	

Klebsiella oxytoca was one time in three susceptible to Amoxicillin-Clavulanic Acid, Ciprofloxacin and Gentamicin. *Morganella morganii* was two times out of three susceptible to Gentamicin and one time in three to Ciprofloxacin. However, it was consistently resistant to Ceftriaxone. Thirty-five patients or 70% were infected with MDR bacteria. Enterococcus spp. was the most frequently MDR GPC (60%) followed by *Staphylococcus aureus* (48.57%) including 5.71% of Methicillin-resistant *Staphylococcus aureus* (MRSA). Among the GNB, Moraganella morganii, *Escherichia coli*, Acinetobacter spp., *Proteus mirabilis*, *Citrobacter farmeri*, *Seratia*

odorifera were all consistently enterobacteria secreting extended-spectrum β-lactamase (ESBL). Klebsiella pneumoniae and Klebsiella oxytoca were MDR bacteria in 75% and 66.67% of cases respectively.

Fifteen patients (30%) had undergone an amputation, including 10 minor and 5 majors. The mean length of hospitalization was 15.98±12.26 days (3-60 days). One patient had died (2%).

Table 2: Isolated Pathogenic bacteria from bacteriological examinations of infected diabetic foot lesions (n=64).

Isolated pathogenic bacteria	Classification	Number (n=64)	Proportion (%)
Staphylococcus aureus	GPC	35	54.69
Enterococcus spp.	GPC	5	7.81
Streptococcus spp.	GPC	3	4.69
Staphylococcus pyogenes	GPC	2	3.13
Staphylococcus agalactiae	GPC	1	1.56
Klebsiella pneumoniae	GNB	4	6.25
Klebsiella oxytoca	GNB	3	4.69
Morganella morganii	GNB	3	4.69
Escherichia coli	GNB	2	3.13
Acinetobacter species	GNB	1	1.56
Proteus spp.	GNB	1	1.56
Miraculous Proteus	GNB	1	1.56
Citrobacter farmeri	GNB	1	1.56
Citrobacter amalonaticus	GNB	1	1.56
Serratia odorifera	GNB	1	1.56

GNB: Gram-Negative Bacteria **GPC:** Gram-Positive Cocci.

Table 3: Results of antibiograms for gram-positive cocci (n=40).

Gram positive Cocci	*Staphylococcus aureus* (n1 =35)		Enterococcus spp. (n2 = 5)	
Antibiotic	**S (%)**	**R (%)**	**S (%)**	**R (%)**
Amoxicillin-Clavulanic acid	83	17	60	40
Oxacillin	80	20	-	-
Gentamicin	80	20	40	60
Ciprofloxacin	74	26	-	-
Amoxicillin	60	40	40	60
Ceftriaxone	40	60	-	-

S: Sensitive **R:** Resistant

Table 4: Results of antibiograms for gram-negative bacteria (n=10).

Gram negative bacteria	*Klebsiella pneumoniae* (n1=4)		*Klebsiella oxytoca* (n2=3)		*Morganella morganii* (n3=3)	
Antibiotic	**S (%)**	**R (%)**	S (%)	R (%)	S (%)	R (%)
Amikacin	100	0	100	0	100	0
Imipenem	100	0	100	0	100	0
Ciprofloxacin	75	25	33.33	66.67	33.33	66.67
Amoxicillin-Acidly Clavulanic	50	50	33.33	66.67	-	-
Ceftriaxone	50	50	33.33	66.67	0	100
Gentamicin	25	75	33.33	66.67	66.67	33.33

S: Sensitive **R:** Resistant**.**

DISCUSSION

To our knowledge, this study represents one of the few local studies providing information on the bacteriological profile and antibiotic sensitivity of infected diabetic foot wounds. The prevalence of 2.85% found in our study was much lower than that of other studies. Indeed, in India, the prevalence of diabetic foot infections was estimated at 6-11%, while in Burkina Faso, it was 18.9%.[7,11] These differences could be explained, in part, by the fact that our study was single-center, only involving patients hospitalized in the Endocrinology department of the JRBUH while theirs were multicenter. Furthermore, our study setting is essentially a Medicine department. Some patients would probably be hospitalized immediately in surgical settings due to the severity of their lesions. The

mean age of our patients (55.50 years) was similar to those reported in other African studies such as those conducted in Burkina Faso (53 years) and Congo (54.6 years).[7,12] Indeed, diabetic foot infection occurred mainly in relatively elderly subjects in our study with predilection ages between 51 and 60 years and between 61 and 70 years. The decline in immunity with age or "immunosenescence" increases the risk of infection.[13] On the other hand, the female predominance of our patients was in disagreement with the results of other studies.[13] We therefore put forward as a hypothesis the deleterious roles of wearing pointed-toe shoes in women promoting deformations and ulcers that are the breeding ground for infections. Similarly, as in the DIABASIS study, women would take the disease more seriously and participate more in self-management of diabetes, while men would be more lax with regard to their state of health and would more rarely accept hospitalization.[14] They could also have presented more serious lesions requiring emergency amputation before any bacteriological sampling. However, these hypotheses remain to be verified.

The mean duration of diabetes progression of 6.87 years found in our study was close to that reported by African authors such as Monabeka et al, (8 years) but was much lower than those reported by other authors from the American continent such as Wukich et al, (14.6 years) and European authors such as Richard et al, (17.5 years).[5,12,15] Indeed, in Madagascar as in Africa, infectious complications of diabetic foot occur a little earlier than elsewhere due to the lack of hygiene, the still frequent walking barefoot, the hot and humid climate and the mistreated and poorly controlled diabetes favouring complications.[16]

In our study, HBP was the cardiovascular risk factor most frequently associated with diabetes, in agreement with literature data.[16,17] Indeed, HBP increases and accelerates the onset of vascular complications and multiplies the risk of diabetic foot by 3.[17]

The lesions of our patients were most often post-traumatic or appeared spontaneously and then became infected, corroborating the data in the literature.[7] This fact raises the inadequacy of the therapeutic education of our patients and the probable role of the neurological complications of diabetes making the lesions painless and thus delaying the discovery of the wounds, leaving time for the infection to set in.[18] Unfortunately, our data did not provide information on the neuropathic and/or arteriopathic nature of the diabetic foot.

More than one in four patients (26%) had already received systemic antibiotic therapy before their hospital admission to us. However, we do not know whether these antibiotics were prescribed by health professionals and followed the recommendations on the management of infected diabetic foot or not. If not, the risk is the emergence of MDR bacteria.

The average hospitalization time of our patients (42.8 days) was close to that reported by Akossou et al, (43.2 days).[16] These results assumed that our patients would tend to neglect their wounds and consult late. This worsens the prognosis. This results not only from the lack of education received by patients but also from the fear of having their feet amputated and especially the very high frequency of self-medication and consultations with traditional practitioners.[16] Thus, it is not surprising that 72% of infections were already grade 4, therefore with systemic signs. In the same study site, in 2015, we have already seen that 43.75% of diabetic foot infections were grade 4 and 43.75% grade 3.[19] Therapeutic education must then be supported so that patients consult early and at the slightest doubt to limit the burdens of the diabetic foot. The prevalence of osteitis in our patients was agreement with the reported data by Hammami et al, in Tunisia (18%).[20] However, it may be biased since its diagnosis was, for most patients, made during clinical examination without performing imaging tests. Furthermore, we did not take any bone sample. Access to these examinations must then be facilitated in our case since bone involvement conditions, among other things, the duration of antibiotic therapy and the possible surgical procedure.[3]

Biologically, the mean blood glucose level of our patients on admission was high at 295 mg/d, shalring the same trend with that of the study by Guira et al, (378 g/dl).[7] Indeed, hyperglycemia can be both the consequence and the contributing factor in the occurrence of an infection.[21] This would be verified by the fact that the mean HbA1c level of our patients was 10%. The hyperleukocytosis, neutrophilic polynucleosis and the high CRP level of our patients reflect the severity of the infection. Indeed, the number of white blood cells, particularly that of neutrophils, is a biological marker recognized by many authors for its reliability in diabetic foot infection.[22] The same is true of the CRP value.[23]

Concerning the bacteriological profile of the wounds, the majority of the isolated bacteria were GPC (71.86%) including Staphylococcus aureus (54.69%). But contrary to certain observations, the infection of the diabetic foot was most often mono-microbial in our case.[24,25] The predominance of *Staphylococcus aureus* is consistent with those reported by several authors such as Guira et al, in Burkina Faso (32.4%), Unachukwu et al, in Nigeria (56.1%), Syeda et al, in Pakistan (46%) and Kelly et al, in America (34%).[7,25-27] Indeed, in diabetics, the skin colonizing flora, generally consisting of aerobic and anaerobic bacteria of low virulence, can be modified, becoming more polymorphic with the appearance of more virulent GPC such as *Staphylococcus aureus* or Streptococcus pyogenes . These bacteria can easily infect diabetic foot wounds despite the diversity of pathogens associated with infections.3 GNB were less present in our study, contrary to the results reported by other teams such as Lokrou et al, (60.65%) and Kow et al (73.4%).[28,29]

The disparity of results from one study to another in terms of isolated bacteria would result from several factors including the hygiene of the patients, their geographical origin, the extent of the ulcers, their chronic evolution, the taking of antibiotics in pre-hospital without forgetting the non-uniformity of the sampling protocols.[3,30] In our case, all the samples were taken exclusively by swabbing. Thus, one of the concerns was to distinguish between the true pathogens and the bacteria of contamination by the commensal flora of the skin. On the other hand, the search for strict anaerobic germs was technically impossible in our conditions. This would make our results non-exhaustive.

The two main GPC isolated in our study were Staphylococcus aureus and Enterococcus spp. The results of the antibiograms indicated that *Staphylococcus aureus* was 83% sensitive to Amoxicillin-Clavulanic Acid, 80% to Oxacillin and Gentamicin, 74% to Ciprofloxacin and 40% to Ceftriaxone. This was similar to the results of the study by Guira's team. In fact, for theirs, the frequency of sensitivity of *Staphylococcus aureus* was 100% to oxacillin, 90% to ciprofloxacin, 75% to amoxicillin-clavulanic acid and 66% to gentamicin.[7]

For Enterococcus spp, its sensitivity frequency was 80% for Amoxicillin-Clavulanic Acid and 40% each for Amoxicillin and Gentamicin in our study.

Since *Staphylococcus aureus* is the most common germ, the use of simple, accessible antibiotics included in international recommendations such as Amoxicillin-Clavulanic Acid and Oxacillin still seems legitimate to identify the majority of pathogenic aerobic flora. Quinolones including Ciprofloxacin, still hold a good place. But given the alarming incidence of bacterial resistance to this class in recent times, probably due to their abusive use, these molecules known for their good bone penetration should be preferred in cases of osteitis.[3]

The three main GNB isolated in our study, namely: Klebsiella pneumoniae, *Klebsiella oxytoca* and *Morganella morganii* were all sensitive in 100% of cases to Amikacin and Imipenem. This result is in agreement with the observation of Hayyat et al, having found a sensitivity close to 100% to Amikacin and Imipenem for all GNB.[31] For the other more classic antibiotics and more accessible in our country such as Amoxicillin-Clavulanic Acid, Gentamicin, Ciprofloxacin and Ceftriaxone, their efficacy seems to be mixed with respect to these main GNB. However, it is difficult to extrapolate this result given the limited number of GNB isolated in our study.

MDR bacteria carriage is high in our patients (70%). This finding confirms Alavi's observation. Indeed, antibiotic resistance has become a major health problem with a rate exceeding 65% among patients suffering from infected diabetic foot.[32] The blind use of broad-spectrum antibiotics is the main factor leading to selective pressure and the emergence of these MDR bacteria.[33]

Other factors incriminated in the literature are the lack of hygiene, insufficient health education of the population, the high frequency of self-medication, the existence of illicit sale of drugs, the existence of fake drugs and drugs of substandard or expired quality circulating, the frequent use of broad-spectrum antibiotics for prophylaxis and the lack of laboratory tests for the identification of pathogens.[34,35] The role of nosocomial infections cannot be ruled out either. In fact, some patients were only able to benefit from bacteriological sampling after a few days of hospitalization for various reasons. Although the isolation of MDR bacteria is not necessarily synonymous with increased virulence, these pose a major challenge in low-income countries like ours where drugs are the responsibility of patients and are often unaffordable.[36] Hence the interest in respecting the rational and codified use of antibiotics in a probabilistic manner following international recommendations, depending on the duration of the lesion's evolution, its severity and prior antibiotic therapy, before adaptation according to the results of the antibiogram.[1,3]

Therapeutically, thirty percent of our staff underwent an amputation, in accordance with data from the African literature. Indeed, the frequency of amputations of diabetic feet in Sub-Saharan Africa varies from 6.3% to 41% since revascularization techniques are rarely practiced there.[16,37] Amputation often remains the only solution to save the patient's life given the serious state of the lesions. This will have the consequences of extending the length of hospitalization and increasing the related expenses. In addition, problems of acquiring equipment (cost, availability, etc.) will be added, surely compromising the quality of life of the majority of patients.

Compared to patients from wealthier countries such as the Americas where the average length of hospital stay was around 8 days and in Israel, 10 days, our patients stayed longer in hospital.[14,38,39] In our country, few health centers perform diabetic foot dressings. Patients are forced to stay in hospital for wound dressings to avoid travel and related expenses. However, our mortality rate was comparable to that in the literature.[39]

Our study was limited by the small sample size, the superficial nature of the local bacteriological samples and the non-uniformity of the antibiogram disks used by the different laboratories where the bacteriological examinations were carried out.

In the future, prospective, multi-center, multidisciplinary studies with uniform protocols are needed to improve our knowledge of diabetic foot infections in Madagascar.

CONCLUSION

Diabetic foot infections often follow trauma in us. The main responsible bacteria are *Staphylococcus aureus*. Often serious, diabetic foot infections have required amputation in 3 out of 10 patients.

All this requires the optimization of therapeutic education on podiatric care, the rational use of probabilistic antibiotic therapy and the multidisciplinary management of diabetic feet. Amoxicillin-Clavulanic Acid can be preferred as probabilistic antibiotic therapy for diabetic foot infection in our country where bacteriological examination is often lacking.

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REFERENCES

1. Lipsky BA, Aragón-Sánchez J, Diggle M, Embil J, Kono S, Lavery L, et al. IWGDF guidance on the diagnosis and management of foot infections in persons with diabetes. Diabetes Metab Res Rev. 2016;32:45-74.
2. Richard JL, Schuldiner S. Epidémiologie du pied diabétique. Rev Med Interne 2008;29:S222-S230.
3. Société de Pathologie Infectieuse de Langue Française. Recommandations pour la pratique clinique Prise en charge du pied diabétique infecté Texte long. Med Mal Infect. 2007;37:26-50.
4. Bowker JH, Pfeifer MA. The Diabetic Foot. 6th ed. St Louis: Mosby. 2001.
5. Richard JL, Lavigne JP, Got I, Hartemann A, Malgrange D, Tsirtsikolou D, et al. Management of patients hospitalized for diabetic foot infection: results of the French OPIDIA study. Diabetes Metab. 2011;37(3):208-15.
6. Nankam JK, Yefou MD, Sobngwi E, Mbanya J. CA-086: Le pied diabétique infecté en Afrique Subsaharienne: choix de l'antibiothérapie. Diabetes Metab. 2016;42:58.
7. Guira O, Tiéno H, Traoré S, Diallo I, Ouangré E, Sagna Y, et al. Écologie bactérienne et facteurs déterminant le profil bactériologique du Pied diabétique infecté à Ouagadougou (Burkina Faso). Bull Soc Pathol Exot. 2015;108:307-11.
8. American Diabetes Association. Classification and diagnosis of diabetes. Diabetes Care. 2020;43(1):14-31.
9. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect. 2012;18(3):268-81.
10. Dorland. Dorland's Illustrated Medical Dictionary. Philadelphia: Elsevier Health Sciences. 1999.
11. Vishwanathan V, Thomas N, Tandon N, Asirvatham A, Rajasekar S, Ramachandran A, et al. Profile of diabetic foot complications and its associated complications-a multicentric study from India. JAPI. 2005;53(10):933-6.
12. Monabeka HG, Nsakala-Kibangou N. Aspects épidémiologiques et cliniques du pied diabétique au CHU de Brazzaville. Bull Soc Pathol Exot. 2001;94(3):246-8.
13. Berthé A, Gueye AD, Marone Z, Ndiaye Y, Faye FA, Diop MM, et al. Causes infectieuses de décompensation du diabète: « un prédateur infectieux se cache chez tout diabétique en Afrique. RAFMI. 2022;7:36-42.
14. Mosnier-Pudar H, Hochberg G, Eschwege E, Virally ML, Halimi S, Guillausseau PJ, et al. How do patients with type 2 diabetes perceive their disease? Insights from the French DIABASIS survey. Diabetes Metab. 2009;35(3):220-7.
15. Wukich DK, Hobizal KB, Brooks MM. Severity of Diabetic Foot Infection and Rate of Limb Salvage. Foot Ankle Int 2013;34(3):351-8.
16. Akossou SY, James YE, Tetekpor S, Tsogbevi S, Amedegnato MD, James KD. La prise en charge du pied diabétique au CHU de Lomé Tokoin. Louvain Medical. 2003;122(6):272-9.
17. Djibril AM, Mossi EK, Djagadou AK, Balaka A, Tchamdja T, Moukaila R. Pied diabétique: aspects épidémiologique, diagnostique, thérapeutique et évolutif à la Clinique Médico-chirurgicale du CHU Sylvanus Olympio de Lomé. Pan Afr Med J. 2018;30:4.
18. Lipsky BA, Berendt AR, Embil J, De Lalla F. Diagnosing and treating diabetic foot infections. Diabetes Metab Res Rev. 2004;20(1):56-64.
19. Raherison RE, Raharinavalona SA, Randriamboavonjy RL, Rakotoarivelo RA, Randria MJD, Rakotomalala DP. Infections communautaires chez les diabétiques Malgaches hospitalisés à de l'Hôpital Joseph Raseta de Befelatanana, Antananarivo Madagascar. Rev Méd Madag. 2015;5(2):577-81.
20. Hammami M, Lahiani D, Guemri B, Maalej M, Elleuch E, Hammami B, et al. Les infections du pied diabétique: Étude de 136 cas. Annales d'Endocrinologie. 2015;76(4):552552.
21. Bartelink ML, Hoek L, Freriks JP, Rutten G. Infections in patients with type 2 diabetes in general practice. Diabetes Res Clin Pract 1998;40(1):15-9.
22. Zemmouri A, Tarchouli M, Benbouha A, Lamkinsi T, Bensghir M, Elouennass M, et al. Profil bactériologique du pied diabétique et son impact sur le choix des antibiotiques. Pan Afr Med J 2015;20:148.
23. Boukhris J, Benyass Y, Boussaidane M, Mekkaoui J, Chafry B, Benchebba D, et al. Infection du pied diabétique: du diagnostic a la prise en charge (revue de la littérature). Revue Marocaine de Chirurgie Orthopédique et Traumatologique. 2018;75:9-15.
24. Lipsky BA, Berendt AR, Cornia PB, Pile JC, Peters EJ, Armstrong DG, et al. 2012 Infectious Diseases Society of America clinical practice guideline for the diagnosis and treatment of diabetic foot infections. Clin Infect Dis. 2012;54(12):132-73.
25. Unachukwu CN, Obunge OK, Odia OJ. The bacteriology of diabetic foot ulcers in Port Harcourt, Nigerian. Niger J Med. 2005;14(2):173-6.

26. Amjad SS, Zafar J, Shams N. Bacteriology of diabetic foot in tertiary care hospital; frequency, antibiotic susceptibility and risk factors. J Ayub Med Coll Abbottabad. 2017;29(2):234-40.
27. 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):2819-28.
28. Lokrou A, Memel AT, Dago KP. Bacteriology of diabetic foot lesions in Ivory Coast. MmM. 2013;7:477-81.
29. Kow RY, Low CL, Ruben JK, Zaharul Azri WMZ, Mor Japar Khan ESK. Microbiology of diabetic foot infections in three district hospital in Malaysia and comparison with South East Asian Countries. Med J Malaysia. 2019;74(5):394-9.
30. Kale DS, Karande GS, Datkhile KD. Diabetic foot ulcer in India: aetiological trends and bacterial diversity. Indian J Endocrinol Metab. 2023;27(2):107-14.
31. Hayat AS, Khan AH, Naila Masood NM, Naila Shaikh NS. Study for microbiological pattern and in vitro antibiotic susceptibility in patients having diabetic foot infections at tertiary care hospital in Abbottabad. World Appl Sci J. 2011; 12(2):123–31.
32. Alavi SM, Khosravi AD, Sarami A, Dashtebozorg A, Montazeri EA. Bacteriologic study of diabetic foot ulcer. Pak J Med Sci. 2007; 23(5):681–4.
33. Sannathimmappa MB, Nambiar V, Aravindakshan R. A cross-sectional study to evaluate the knowledge and attitude of medical students concerning antibiotic usage and antimicrobial resistance. Int J Acad Med. 2021;7(2):113-9.
34. Yenehun Worku G, Belete Alamneh Y, Erku Abegaz W. Prevalence of bacterial urinary tract infection and antimicrobial susceptibility patterns among diabetes mellitus patients attending Zewditu Memorial Hospital, Addis Ababa, Ethiopia. IDR. 2021;14:1441-54.
35. Cheesbrough M. District Laboratory Practice in Tropical Countries, Part 2. New York: Cambridge University Press. 2006.
36. Edmonds M, Foster A. The use of antibiotics in the diabetic foot. Am J Surg. 2004;187(5):25-8.
37. Nouedoui C, Teyang A, Djoumessi S. Profils épidémiologiques et traitement du pied diabétique au Centre National de Diabétique de Yaoundé-Cameroun. Tunis Med. 2003;81(1):20-5.
38. Tan TW, Shih CD, Concha-Moore KC, Diri MM, Hu B, Marrero D, et al. Disparities in outcomes of patients admitted with diabetic foot infections. PLoS One. 2019;14(2):211481.
39. Katz DE, Friedman ND; Ostrovski E, Ravid D, Amrami N, Avivi D, et al. Diabetic foot infection in hospitalized adults. J Infect Chemother. 2016;22(3):167-73.

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