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REVIEW ARTICLE

Treating diabetic foot infection in the emergency department: fundamental considerations

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Diabetic foot infection is one of the most serious complications the patient with diabetes mellitus can develop and it is a frequent reason for hospital admission. Such infections are also the most frequent cause of amputation unrelated to trauma in the West, and this condition can be life-threatening if not managed properly. To reduce the high rates of morbidity and mortality related to diabetic foot infection, early detection and rapid start of treatment are essential. A multidisciplinary approach to management is advised. The emergency department physician plays a key role in providing an early diagnosis, prescribing appropriate antibiotic therapy, and ordering that samples be sent to the microbiology department for processing. This review is an update of fundamental considerations the emergency physician must bear in mind in order to assure the best outcome for these patients. [Emergencias 2012;24:211-218]

Key words: Diabetic foot. Infection. Emergency health services.

Introduction

Diabetes mellitus is a highly prevalent health problem affecting 3.5 million people in Spain, and this figure increases by 5% per year. Diabetic foot is defined as any type of inframalleolar lesion presenting in a diabetic patient. The risk of ulcers in diabetic patients, the primary lesion of diabetic foot, increases in the presence of the following factors: diabetes mellitus of more than 10 years, poor glycemic control or other diabetes-related comorbidities (cardiovascular, renal or diabetic retinopathy)[1]. Diabetic foot is a major health problem for diabetic patients, accounting for 25% of reasons for hospital admission in these patients[2]. Furthermore, it severely affects quality of life.

Diabetic patients are liable to develop ulcers, either ischemic or neuropathic, which can develop serious infections. Fifteen percent of diabetic patients will suffer diabetic foot infection at some time, which means an annual incidence of 1-4%[3].

These infections not only threaten the integrity of the affected foot, but also the patient's life.

The frequent use of antibiotics in these patients may lead to the development of resistance and this is an important factor to consider in the choice of empirical antibiotic treatment. The emergence of multiresistant microorganisms, especially methicillin-resistant *Staphylococcus aureus* (MRSA) and extended spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae, worsen the prognosis and increase the risk of amputation. Despite current therapies, 15% of patients with diabetic foot infection undergo surgical amputation, and this is the most frequent non-traumatic cause of amputation.

The management of diabetic foot infection is complex; it must be approached in a multidisciplinary way, and always considering that poor management or delay in diagnosis and appropriate therapeutic measures can lead to amputation or death[4].

Pathogenesis

Ischemia, neuropathy and infection are the 3 pathogenic mechanisms underlying diabetic foot complications. Diabetic neuropathy is a key factor for the onset of complications and present in 85-90% of cases. Sustained hyperglycemia is the main risk factor involved. Sensorial alterations make the patient insensitive to changes in temperature or micro-trauma, while motor neuropathy produces muscle atrophy and weakness, leading to loss of joint stabilization. Autonomic neuropathy causes anhidrosis, causing dryness with cracks appearing in the skin. An opening occurs in arteriovenous communications, which causes an increase in blood flow, but also a decrease in capillary network perfusion and the appearance of edema.

Diabetic macroangiopathy is another key factor, present in 40-50% of cases, generally associated with the presence of neuropathy. Diabetics are more prone to atherosclerosis, affecting medium and large caliber arteries, and this develops early and rapidly[5]. Microangiopathy, also present in these patients, has not been correlated with decreased blood flow or the appearance of ulcers.

Alterations in leukocyte function, secondary to hyperglycemia, such as changes in diapadesis and leukocyte adhesion, decreasing chemotactic, phagocytic and cytotoxic capacity, can lead to less effective polymorphonuclear leukocytes with impaired response to pathogenic stimuli, which increases the risk of infection. While some studies have found a negative correlation between glycosylated hemoglobin concentration and polymorphonuclear bactericidal activity, others have shown that good glycemic control normalizes some of these deficits in cellular immunity[6].

Vasculopathy, neuropathy, trauma, poor control of blood glucose, immune system alterations and poor hygiene determine the development of diabetic foot infections. However, the most important risk factor for diabetic foot infection is loss of the skin barrier, as a result of peripheral neuropathy, favoring colonization by microorganisms and subsequent infection.

Thus, in the pathophysiology of diabetic foot there are a number of high risk predisposing factors (neuropathy, macro and microangiopathy): some factors act as triggers (usually mechanical trauma), producing ulcers or necrosis, and some are aggravating factors, which determine the prognosis of the limb, such as infection which causes extensive tissue damage, ischemia which hinders healing, and neuropathy which prevents awareness of lesions and triggers[7].

With regard to etiology, the microorganisms involved in the infection are determined by the type of infection and patient situation (antibiotic treatment, manipulation or previous hospitalization). Gram positive cocci are frequently involved, so adequate antibiotic coverage against them should be provided. There is a correlation between the microorganisms involved and type of injury - superficial or deep (Table 1). Superficial infections are usually mono-microbial gram-positive cocci. Deep infections or those of long-standing ulcers are usually poly-microbial, and in addition to gram-positive cocci, gram-negative and anaerobic bacteria are often isolated.

When presenting in association with other microorganisms, the pathogenic role of negative coagulase *Staphylococcus, Corynebacterium* spp., *Candida* spp. and *Enterococcus* spp. is under debate. Their importance is clearly greater when predominant or single agents are isolated in a deep sample. The bacteria isolated can be multiresistant; and the following risk factors have been described: previous antibiotic therapy, long-duration antimicrobial treatment, frequency of hospital admission for the same wound, length of hospital stays, presence of osteomyelitis and / or neuropathy and ulcer size[7]. In patients who have received antibiotic treatment before or who have been hospitalized recently, resistant bacteria such as MRSA, ESBL-producing enterobacteriaceae or non-fermenting gram-negative bacilli (*Pseudomonas*

Table 1. Etiology of diabetic foot infections

Infection	Micro-organism
Celulitis	*S. aureus*
Erysipelas	Beta hemolytic streptococcus
Ulcer not treated with antibiotics	*S. aureus*
	Beta hemolytic streptococcus
Ulcer treated with antibiotics or with prolonged evolution (usually polymicrobial)	*S. aureus* (both sensitive or resistant to methicillin)
	Streptococcus spp.
	Enterococcus spp.
	Enterobacteriaceae
	P. aeruginosa
	Other non-fermenting gram-negative bacilii
	Corynebacterium spp.
	Candida spp.
Necrotizing fasciitis or myonecrosis (usually polymicrobial)	Aerobic gram-positive cocci
	Enterobacteriaceae
	Non-fermenting gram-negative bacilli
	Anaerobes

spp. and *Acinetobacter baumannii*) can be isolated[7].

Clinical and microbiological diagnosis

The diagnosis of diabetic foot infection is clinical; the picture may vary from mild cellulitis to deep infection of the soft tissue with bone involvement. However, clinical diagnosis is not simple. The classical signs of infection such as heat, redness, pain or wound purulence may not be present due to neuropathic or vascular alterations in these patients, or because the infection is deeply embedded. Sometimes, evidence of systemic infection may even be absent in diabetic patients. Hyperglycemia may be the only manifest clinical sign, so in the absence of another clear reason for this, we must always suspect a possible infectious origin. Similarly, the laboratory test alterations typical of infectious processes, such as leukocytosis, may occasionally be absent. It may be helpful to have C-reactive protein or procalcitonin values to evaluate the process.

What is essential is a thorough examination of the ulcer, looking for fluctuating or abscessed tissue and determining depth. If bone tissue can be seen, osteomyelitis can be assumed, but not seeing the bone does not imply the absence of osteomyelitis.

There are several classification systems to predict evolution of diabetic foot, such as the Texas[8] or Wagner[9] systems, based on ulcer depth or the presence of ischemic alterations and / or infection. The clinical utility of such classification is to determine the risk of acute process to decide on the optimal therapeutic management and the final assignment of the patient. IDSA[10] classification, based on the clinical situation and the visual appearance of the lesion, is simple and useful for predicting the evolution of the affected foot, the risk of amputation and the probability of survival (Table 2).

Once the clinical diagnosis of infection has been made, the next task is approximate etiologic diagnosis. The characteristics of the ulcer, essentially depth, can help identify a suspected causal mechanism. However, other data should be considered, such as duration of this lesion or previous antibiotic treatment (Table 1).

During ED assessment, plain x-ray is required if there is any doubt about the presence of osteomyelitis, although the result may be normal in early stages, so the absence of alterations does not exclude the diagnosis. The attending physician should look for thickening of the periosteum and bone destruction. Any focus of bone destruction near an ulcer should be considered as osteomyelitis.

Finally, we must determine the presence or absence of associated ischemia in order to establish the prognosis and assess the need for revascularization treatment. During the process of taking the medical history, the patient should be questioned about the main characteristic symptoms of chronic lower limb ischemia, in particular intermittent claudication and pain at rest. Physical examination should include palpation of pulses (femoral, popliteal, tibial and pedal), and auscultation for murmurs at the femoral level[7].

Although treatment in the ED must be established empirically, it is essential to take biological samples for cultivation, from the base of the ulcer and after debridement, but avoiding necrotic areas. If the patient shows clinical manifestations of systemic disease, blood samples should be obtained. The methods for tissue sample collection include: biopsy, scraping, percutaneous aspiration and swabs. Biopsy is taken from the base of the ulcer, after surgical debridement. Scraping is performed similarly, with a sterile curette to collect a sample of tissue (1-1,5 mm^3) from the base of the ulcer. Percutaneous aspiration with a fine needle can be used in cases of cellulitis and pus collection. Sampling by swab is the most common method for its simplicity and availability; however, this method offers worse sensitivity and specificity. The sample is taken from the base, rotating the swab with some pressure to squeeze the tissues. Tissue samples are preferable to fluid in principle since more microorganisms are collected.

Table 2. IDSA classification of diabetic foot infection severity (adapted from the Spanish Society of Angiology and Vascular Surgery classification)

Severity of infection	Clinical signs of infection
No infection	No signs of inflammation or suppuration.
Mild infection	No signs of systemic infection. Evidence of pus or 2 or more signs of inflammation.
Mild to moderate infection	No systemic signs of infection. Cellulitis <2 cm. Deep tissue infection (through subcutaneous tissue, no signs of abscess, lymphangitis, arthritis, osteomyelitis, myositis or critical ischemia).
Moderate to severe infection	No signs of systemic infection. Cellulitis >2 cm. Deep tissue infection (through subcutaneous tissue, with abscess, lymphangitis, arthritis, osteomyelitis, myositis or critical ischemia).
Severe infection	Any infection that is accompanied by systemic toxicity (fever, chills, vomiting, confusion, metabolic instability, shock).

Treatment

Antibiotic treatment plays a fundamental role in patient outcome. However, debridement of devitalized tissue is a prerequisite, followed by drainage of deep abscesses and ongoing local treatment. Early aggressive debridement helps prevent amputation and preserve foot function in many cases. It is essential to assess the need for early revascularization. In patients with poor outcome despite these measures, partial or total amputation should be considered, and localized amputation should be prioritized insofar as possible. Treatment of diabetic foot infection should be multidisciplinary; urgent assessment by a general or vascular surgeon is essential in patients with moderate or severe infection.

We should also initiate medical treatment for normalizing glycemic values and metabolic alterations. Patients with severe infection may present acidosis, hyperosmolality or azotemia on arrival at the ED, or severe sepsis or septic shock, and in this context the appropriate therapeutic measures should be applied early.

There are other novel treatments such as the use of growth factors or hyperbaric oxygen. Their use is not yet standardized so the recommendation for use should be individualized and, anyway, only when the clinical picture has stabilized.

Antibiotic treatment

There are no data to support antibiotic treatment of chronic ulcers, even when tests are positive. Antimicrobial treatment is indicated if there are clinical criteria for systemic or local infection, either the presence of pus or two or more signs of inflammation (erythema, induration, pain, tenderness, warmth)[10]. Laboratory data are not particularly useful for diagnosing infection, except in the case of osteomyelitis[14].

The antimicrobial treatment of diabetic foot infection is conditioned by ischemia which hinders the supply of antibiotics to the septic focus, impairs leukocyte function and may induce renal failure[15]. Ischemia and leukocyte alterations worsen the response to treatment and the patient's condition may deteriorate rapidly, in a matter of days or even hours[15,16]. Due to functional neutrophil defects in diabetics, it is advisable to use bactericidal antibiotics over long periods; the ischemia conditions the use of high doses and the presence of renal dysfunction implies avoidance of nephrotoxic drugs, such as aminoglycosides[10,15-18]. The severity of infection and probable etiologic agents involved condition the choice of empirical antimicrobial treatment, as well as the site and route of administration. Gram-positive cocci are the predominant pathogens, so the antibiotics chosen should always be active against them. In chronic ulcers previously treated with antibiotics or surgically managed, and inpatients, we may find MRSA, ESBL-producing enterobacteriaceae, *P. aeruginosa,* other non-fermenting gram-negative bacilli, coagulase-negative *Staphylococcus* and *enterococcus*. Patients with evidence of ischemia or gangrene require antibiotic coverage against anaerobic pathogens. There are certain risk factors associated with infection by MRSA and ESBL-producing enterobacteriaceae, which should be considered on establishing empirical antibiotic treatment (Tables 3 and 4)[7].

In cases of mild infection, where the goal is to deal with gram-positive cocci, treatment can be established with amoxicillin-clavulanate during 7-14 days on an outpatient basis. However, the patient should be subject to close monitoring to assess the evolution of infection. In patients allergic to betalactams we can use levofloxacin, moxifloxacin or clindamycin, but if MRSA is suspected then cotrimoxazole and linezolid are recommended (Table 5)[19,20]. Moderate or severe infections are usually polymicrobial and can be life-threatening or lead to amputation of the foot. For these reasons, treatment should be intravenous wide spectrum antibiotics, so the patient will require hospi-

Table 3. Risk factors associated with infection by methicillin-resistant *Staphylococcus aureus* (MRSA)

Colonization or previous infection by MRSA.
Local MRSA prevalence > 10%.
Two or more of the following:
– Hospital admission in the last year.
– Proceeding from a health center with endemic MRSA.
– Treatment with a quinolone in the last 6 months.
– Patient > 65 years.
– Patient in a dialysis program.

MRSA: Methicillin-resistant *Staphylococcus aureus.*

Tabla 4. Risk factors associated with infection by ESBL-producing enterobacteria

Age> 65 years.
Female sex.
Hospitalization in the last year.
Recurrent urinary tract infection.
Previous use of quinolones.
Diabetes.

ESBL: extended spectrum beta-lactamases.

talization[17-19]. In this context there are different treatment options: ertapenem[20,21], third generation cephalosporin plus metronidazole[22], amoxicillin-clavulanate[23] or piperacillin-tazobactam[24] (mainly when *P. aeruginosa* is suspected). Ertapenem offers certain advantages in terms of dosage, spectrum, good tissue penetration, bactericidal potency and limited inoculum effect but has no selective pressure on *P. aeruginosa* and does not act on this microorganism.

In cases with risk of infection by MRSA, the following must be added: daptomycin, linezolid, or vancomycin[25,26]. The benefits of daptomycin include rapid bactericidal action, activity against biofilms of chronic ulcers and vegetative phase bacteria, and it is safe for patients with renal insufficiency. The advantage of linezolid is its oral and intravenous availability, and good oral bioavailability, but it is bacteriostatic and can induce thrombocytopenia especially in prolonged treatment and in patients with renal insufficiency. Vancomycin has problems of bioavailability at the focus of infection and renal toxicity[7].

In cases of suspected infection by ESBL-producing enterobacteriaceae, the treatment of choice is a carbapenem, and the best option is ertapenem, provided there is no risk of co-infection with *P. aeruginosa*. Risk factors for infection by *P. aeruginosa* are: chronic ulcers, exuding ulcers or those treated with wet dressings or hydrotherapy, warm weather inducing foot sweating, especially in people who wear inadequate footwear without socks, and having received antibiotic treatment in the previous month[7].

In seriously ill patients recently treated with antibiotics or unresponsive to initial therapy, we must consider the implication of less common microorganisms and extend coverage with an antipseudomonal lactam. Moreover, in these serious cases where treatment failure may be fatal, we must provide initial coverage against MRSA. In cases with β-lactam allergy, it is useful to include tigecycline in combination with a fluoroquinolone (levofloxacin or ciprofloxacin) or amikacin to act against *P. aeruginosa*[27]. The administration of continuous infusion piperacillin-tazobactam or carbapenem offers pharmacodynamic and pharmacokinetic advantages which have been shown to reduce mortality and hospital stay in patients with serious infection due to *P. aeruginosa*[7].

Failure of appropriate antibiotic treatment may be due to resistance, over-infection or extension to bone tissue. Patients previously hospitalized

Table 5. Empirical antimicrobial treatment of diabetic foot

Infection	First choice	Alternatives
Mild	Amoxicillin – clavulanate acid 875/125 mg every 8 h oral	Levofloxacin (500 mg/12-24 h oral) or moxifloxacin (400 mg/24 h oral) Clindamycin (300 mg/8 h oral) Cotrimoxazole (800/160 mg every 8-12 h oral)
Moderate	Ertapenem (1 g/24 h iv) ± linezolid (600 mg/12 h iv/v) o daptomycin (4-6 mg/kg/24 h iv) or glycopeptide iv	Piperacillin-tazobactam (4/0, 5 g/6-8 h iv) or amoxicillin-clavulanate acid (2/0, 2 g/6-8 h iv) or 3rd generation cephalosporin iv, or fluoroquinolone1 iv / oral + metronidazole (500 mg / 8 h iv / oral or clindamycin (600 mg/6-8 h iv / oral) ± linezolid (600 mg/12 h iv / oral) or daptomycin (4-6 mg/kg/24 h iv) or glycopeptide iv
Severe	Imipenem (0,5-1 g/6-8 h iv) or meropenem (0,5-1 g/6-8 h iv) or piperacillin-tazobactam (4/0,5 g/6-8 h iv) + linezolid (600 mg/12 h iv) or daptomycin (4-6 mg/kg/24 h iv) or glycopeptide iv	Tigecycline (50 mg/12 h iv)[2] + fluoroquinolone[1] or amikacin (15-20 mg/kg/24 h iv)

[1]Ciprofloxacin 400 mg/8-12 h or levofloxacin 500 mg/12-24 h. [2]The first dose of tigecycline should be 100 mg i.v.

and receiving broad spectrum treatment for long periods are usually infected with bacteria such as MRSA or enterococci resistant to glycopeptides. Although MRSA used to be exclusively hospital-acquired, it is now increasingly community-acquired. Finally, it should be noted that empirical antibiotic treatment should be adjusted according to culture results and sensitivity pattern.

Referral to the emergency department and criteria for hospital admission

A patient with diabetic foot infection should be evaluated in the ED when there is: suspicion or evidence of ischemia or osteomyelitis, mild infection that does not improve after 7 days of appropriate treatment, or in cases of moderate or severe infection, or with evidence of systemic involvement.

Mild infections can be managed on an outpatient basis, but with appropriate outpatient follow-up to monitor the course of the infection. Moderate infections create the most difficult decision-making problems since the spectrum of clinical presentation can vary considerably, from almost mild to limb threatening. Obviously, patients with severe infection should be hospitalized and treated there.

In general, hospital admission is indicated for patients with systemic signs of serious infection, those with deep infection or rapidly progressing processes, extensive necrosis, presence of critical ischemia, need for surgical diagnosis and / or therapeutic treatment, and those without adequate social support.

Osteomyelitis

Osteomyelitis occurs in 22-66% of diabetic patients with ulcers[28]. Osteomyelitis is present in 10-20% of infections in diabetic foot soft tissue classified as mild, but in moderate or severe cases this association increases to 50-60%[29]. The pathogens most frequently involved are *S. aureus* and hemolytic *Streptococcus.* For chronic ulcers, gram-negative anaerobes should be considered.

The importance of the diagnosis of osteomyelitis is that it substantially determines the prognosis and therapeutic management. Its presence conditions a higher rate of antibiotic treatment failure and amputations. Furthermore, diagnosis in clinical practice is complicated. It is established by the presence of necrosis and osseous inflammation, and isolation of the microorganism responsible in bone tissue culture. However, early diagnosis in the ED is critical, based on clinical suspicion and imaging studies.

The diagnosis should be suspected in patients with refractory infection, antibiotic treatment failure, or if the bone is visible or palpable. A recent work[30] has proposed that the diagnosis be classified as definite, probable, possible or unlikely based on the combination of clinical, microbiological and radiological criteria (Table 6). The decision to treat would be based on this classification.

In case of clinical suspicion, radiography in two projections should be performed for the typical changes that occur in osteomyelitis: areas of increased density that reflect necrosis, osteolysis, cortical thickening, increased bone diameter, periosteal or hyperplastic reaction, intraosseous cavities (abscesses), isolated bone segments (abduction) and bone deformities. However, radiographic changes are only observed late, at least two weeks after onset, and only when bone destruction is 30-50%[31]. The imaging test with highest sensitivity and specificity is undoubtedly magnetic resonance, although computed tomography (CT) is also superior to radiography, and shows good sensitivity but not as good specificity. CT can identify early alterations and demonstrate bone abscesses or abduction in bone. A complication of establishing the diagnosis of osteomyelitis is difficult differential diagnosis with Charcot neuro-osteoarthropathy, very frequent in the diabetic foot, as this can cause non-infectious bone changes difficult to distinguish from those attributable to osteomyelitis.

Bone biopsy is recommended when the diagnosis of osteomyelitis remains doubtful after imaging tests, and if osteomyelitis is shown but the causal agent and / or its sensitivity to antibiotics is unknown.

There is no consensus on the best management of diabetic patients with osteomyelitis given the absence of appropriate clinical trials. The choice is between aggressive surgery or conservative surgical treatment associated with antibiotic therapy for several weeks. Although the trend is to employ intravenous treatment, no studies have demonstrated that prolonged intravenous therapy is more effective than oral treatment. If surgery is performed with resection of bone fragments or amputation, 3-6 weeks of antibiotic treatment may be sufficient. However, if surgery is not aggressive, then antibiotic treatment may be more prolonged, up to 6 months, and must always be

Table 6. Diagnostic criteria for osteomyelitis

Definite:
– Histology and positive bone culture
– Pus in the bone on surgical examination
– Atraumatic detachment of bone fragments removed from an ulcer.
– Intraosseous abscesses on MRI

Probable:
– Spongy bone visible in an ulcer.
– MRI scan bone edema with other signs of osteomyelitis
– Positive bone marrow culture but negative or absent histology
– Positive bone histology but culture negative or absent

Possible:
– Plain x-ray: cortical destruction
– MRI scan: bone edema or pus
– Positive bone probe
– Viable cortical bone
– ESR > 70 mm without other plausible causes
– Ulcer that does not heal within 6 weeks or persistent ulcer (>2 weeks) with clinical evidence of infection

Criteria	Probability	Management
1 definite 2 probable	> 90%	Treat
1 probable + 2 possible 4 possible	50-90%	Consider treating but perform more studies.
2 possible	10-50%	Do not treat. Perform more studies

VSG: velocidad de sedimentación globular.

guided by microbiological test results.

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Aspectos fundamentales a tener en cuenta en la atención a la infección del pie diabético en urgencias

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La infección del pie diabético es uno de los cuadros clínicos más importantes que puede padecer el paciente con diabetes mellitus a lo largo de su vida, y constituye un motivo frecuente de ingreso hospitalario. En el mundo occidental es la primera causa de amputación no traumática y en caso de no recibir un manejo adecuado puede provocar la muerte del paciente. Para disminuir la elevada morbimortalidad del proceso son fundamentales la detección precoz del proceso y la instauración inmediata del tratamiento. El abordaje de la infección del pie diabético debe ser multidisciplinar, y es fundamental el papel del *urgenciólogo* en el diagnóstico precoz del proceso, la adecuación del tratamiento antibiótico y la toma de las muestras microbiológicas oportunas. El objetivo de esta revisión es actualizar los conocimientos en aquellos aspectos que el *urgenciólogo* debe conocer para mejorar el pronóstico de estos pacientes. [Emergencias 2012;24:211-218]

Palabras clave: Pie diabético. Infección. Servicio de urgencias.