

CLINICAL FEATURES OF DIABETIC FOOT LESIONS - REVIEW

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Abstract: Diabetic foot lesions are common lesions that can occur in other diseases, which are manifested mainly in the diabetic patient. These lesions can cause severe changes in a diabetic patient. In this paper, we reviewed the literature, exemplified by our own case study, in which we described each lesion separately, emphasizing its particularities in the case of the diabetic patient. Early recognition of diabetic foot lesions is extremely important for both the diabetologist and the general practitioner, as this can save the patient from amputation. The shorter the time from detection of the lesion to the initiation of treatment, the greater the chances of a cure.

INTRODUCTION

Diabetic foot is one of the most mutilating and severe complications of diabetes, its prevalence gradually increasing over the years. At the same time, diabetes is a very common pathology, taking into account the fact that the lifestyle of the population is becoming more and more problematic, being associated with obesity and sedentary lifestyle. As a result, the complications of this pathology (including the diabetic foot) will become very common over time.

Despite the progress made in recent years, diabetic foot ulcers continue to be a worrying issue. It has been found that as cardiovascular risk decreases, so does the risk of mortality. However, the average healing time of these ulcers without surgery is about 12 weeks, but it is associated with a very high risk of amputation. The latest data, according to the Veterans Health Administration Population, reported that the 1-year survival rate is 81%, 69% up to 2 years and only 29% up to 5 years.(1)

Types of clinical lesions of the diabetic foot

The lower limbs of patients with diabetes are frequently affected by peripheral diabetic neuropathy. Initially, it may be predominant to affect the sensory fibers, with a large diameter, myelinated (with diminished tactile, vibratory and proprioceptive sensitivity) and also to affect the motor fibers. Later, the damage of small diameter fibers, non-myelinated, type C, is added, which leads to a decrease in thermal and pain sensitivity, as well as to autonomous dysfunctions (affecting the sympathetic innervation). Thus, in patients with symptomatic neuropathy, the decrease in thermal sensitivity, with sensitive neuropathy.(2)

Complications associated with neuropathy include calluses, hyperkeratosis, neuropathic edema, neuropathic ulcers, gangrene, Charcot's foot.

Callus (figure no. 1) is a common lesion, presenting as an area of dense hyperkeratosis, which appeared as a physiological response to small and repeated traumas. In patients with peripheral neuropathy, decreased sensitivity, increased frequency of minor trauma, and deformity of the legs with abnormal pressure distribution at the plantar level, contribute to the formation of callus.

Figure no. 1. Callus on the metatarsophalangeal joint I. Personal collection



Calluses can be a consequence of deformities caused by motor neuropathy - fingers "in the claw", with the prominence of the interphalangeal joints, which favours the production of ulcers.(3) Improper footwear can also lead to calluses on the medial and lateral parts of the toes. Callus is formed on weight-resistant surfaces due to dry skin, insensitivity, and repeated pressure on the foot. It can act as a foreign body and cause ulcers in the leg, so it is very important to debridement it.

Also, in the presence of peripheral neuropathy, **plantar hyperkeratosis** may occur (figure no. 2), which may accompany calluses or may occur in their absence. Hyperkeratosis is often accompanied by cracks, which can be an important way for microbial infections to enter. These changes occur mainly in the calcaneal region, but also in the lateral area of the foot plant.

Another consequence of affecting the sympathetic

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innervation is the presence of **dry skin** (figure no. 3) and hair loss in the lower limbs.

Figure no. 2. Lower left limb hyperkeratosis. Personal collection



Dry skin is often accompanied by pruritus, which can cause itchy lesions, which is a contributing factor to ulcers and infections.(4)

Figure no. 3. Dry Foot. Personal collection



Onychodystrophy (figure no. 4) may be present as a sign of neuropathy, and in cases of severe neuropathy, even nail atrophy may be present.

Figure no. 4. Lower left limb onychodystrophy. Personal collection



Subungual hematoma (figure no. 5) is a collection of blood in the space between the nail bed and the nail, dark purple or dark red, caused by various traumas, but is quite common in patients with neuropathy due to the use of inappropriate footwear and producing high pressures at this level.

Figure no. 5. Superinfected subungual and periungual hematoma



Neuropathic ulcer (figure no. 6) is, by definition, an ulcer caused by neuropathy. For example, a patient who suffers a burn because he cannot feel the high temperature will have a neuropathic ulcer, unlike a patient with a neuropathy that burns accidentally.(3)

Figure no. 6. Right lower limb neuropathic ulcer. Personal collection



Neuropathic ulcer is a consequence of neuropathy and peripheral arterial disease, which if treated improperly can lead to amputation. These ulcers most often occur in areas with repetitive trauma.(5) Neuropathy is the cause of callus formation, which over time, causes ulceration. Repeated trauma, which the patient is unable to experience due to sensory impairment, will eventually lead to skin erosion and ulceration. At the same time, another factor involved in the development of ulceration is vascular damage, present in peripheral arterial disease. Due to the fact that the vascularization is deficient in the respective area, the healing will also be a precarious one, leading in the end even to necrosis and gangrene.(6,7)

Perforating ulcer of the foot or malum perforans pedis (figure no. 7.) is the classic neuropathic ulcer; it can lead to suppurations, abscesses, lymphangitis and even gangrene. It is located under the head of the first or fifth metatarsal, except on the pulp or interphalangeal line of the thumb. The medial and lateral parts of the fingers, as well as the interdigital spaces, are other places of choice for the appearance of ulcers in the lower limbs. It can be single or multiple, symmetrical or not, it begins with a process of circumscribed hyperkeratosis, like a weft, on which in the center appears a small round or oval ulceration, with a diameter of up to one centimeter, with straight cut edges, embossed and hyperkeratosis, with an atonic, gray or burgundy bottom.

Figure no. 7. Perforating ulcer of the foot. Personal collection



Neuropathy accompanied by deformity of the legs increases the risk of ulcers by 12 times, compared to the 1.7-fold increase caused by neuropathy.(8)

Charcot's foot (figure no. 8) was first described in 1868 by Jean-Martin Charcot, a French pathologist and neurologist, in patients with tabes dorsalis (myelopathy due to syphilis). The Charcot's foot is characterized by four different stages: inflammation, fragmentation, covalescence, consolidation. The disease is normally limited to a single passage through these different stages of the disease. The active phase is characterized by a leg with a high local temperature, with the presence of erythema and edema (inflammation), often without pain, due to polyneuropathy. The typical appearance of the final stage of the Charcot foot is the deformity of the foot, the so-called 'rocker bottom' malformation.(9)

CLINICAL ASPECTS

Figure no. 8. Charcot foot. Personal collection



Recent studies show that approximately 23% of patients diagnosed with Charcot's foot are at risk of recurrence within an average of approximately 27 months.(10) The location of choice for Charcot-type lesions is at the level of the tarsusmetatarsal and tarsal joints. Metatarsophalangeal joints are also commonly affected. Charcot's arthropathy has been observed many times in the upper limbs.(8,11)

Gangrene is the end result of ulcers and lower limb infections. In diabetic patients, microvasculopathy causes ischemia, which can lead to gangrene. Most cases of gangrene are diagnosed clinically. Gangrene can be dry or wet, depending on whether or not it is associated with arteriopathy.

In **dry gangrene** (figure no. 9), necrosis is present, the skin is black, the tissues shrink, are crumbly and are removed by exfoliation, with a clear delimitation. The concept of self-amputation is widely followed in this type of injury. However, waiting for the affected limb to self-amputate may increase the patient's discomfort, in addition to other health challenges. Many reports have shown that surgical amputation of the limb with dry gangrene improves the condition of patients.(12) This type of gangrene is found especially in patients who have smoking as a risk factor, high cholesterol, arteriosclerosis and diabetes.

Figure no. 9. Dry gangrene. Personal collection.



In wet gangrene (figure no. 10) the tissue is black, there are beetles, areas of skin necrosis or underlying soft tissue, lymphatic and purulent secretions and exudation fluid. The limit on the unaffected skin shows signs of Celsius, the skin being erythematous and extremely sensitive. Diabetic patients are very sensitive to wet gangrene, and are very prone to infections. Because wet gangrene spreads quickly and can be fatal, emergency surgical treatment is preferable. In the case of wet gangrene, surgical amputation is usually performed to prevent the infection from spreading to other tissues.(13)

Another type of gangrene is **gas gangrene**. The skin is reddish-purple with multiple hemorrhagic bubbles. This is an emergency, requiring hospitalization and intravenous treatment with broad-spectrum antibiotics in the organisms involved, such as Clostridium.(3) According to the 2015 International Diabetes Federation's global report, 9.1 to 26.1 million people with diabetes develop foot ulcers, which can lead to gangrene.(14)

Mőnckeberg sclerosis is a form of arteriosclerosis or hardening of the vessels, where calcium deposits are found in the middle muscular layer of the artery walls (middle tunic), which is often associated with vegetative neuropathy. The consequence of this injury is to limit blood flow to the affected limbs, especially in conditions of high need, such as during exercise. There is an increase in oxygen pressure at this level as a result of the opening of the arteriovenous shunts, but this is not accompanied by a corresponding increase in tissue oxygenation.(15) Figure no. 10. Wet gangrene lower left limb. Personal collection



Patients with diabetes and macrovascular damage may experience intermittent claudication, pain at rest, and in addition may have a decreased or even absent pulse in the foot and / or posterior tibial artery. The appearance of an ischemic ulcer (figure no. 11), often present at the fingertips, is often the first sign. This type of ulcer is well defined, it is not surrounded by callus, it is painful, with slightly purplish edges, in some situations it can be covered by a necrotic staircase, very adherent, with well-defined edges, with or without erythematous tint. Their location is usually above the bony protrusions, ankles, areas where there is no intermediate adipose tissue.

Figure no. 11. Ischemic ulcer of the distal phalanx finger lower left limb. Personal collection



The role of microangiopathy is well known in the appearance and evolution of nephropathy and retinopathy, but in terms of its role in microcirculation is not sufficiently elucidated, but there are changes in microcirculation both anatomically and functionally such as thickening of the basement, increased endothelial permeability, endothelial dysfunction with reduced vasodilator response and increased production of vasoconstrictor substances.(16.17)

Diabetic muscle infarction or diabetic myonecrosis is a rare phenomenon, a serious complication observed in patients with poorly controlled, long-term diabetes, which affects the lower limbs and is more common in females, in patients with type 1 diabetes. However, the rarity of this pathology can be correlated with its resemblance to many other muscle diseases, as well as with a poor knowledge of this disease, and in the literature have been reported less than 200 cases since it was first described, now about 45 years old.(18)

Fungal infections are most commonly involved in the pathology of the diabetic foot, representing the main gateways for bacterial infections, the location of choice being at the level of interdigital spaces - Tinea pedis, Tinea unguum - onychomycosis, at the level of the nails. The prevalence of onychomycosis in patients with diabetes is much more common than in the population without diabetes. Candida albicans is the microorganism most commonly involved in the production of Tinea pedis in interdigital spaces.

Bacterial infections in the diabetic patient's foot are most common in patients with diabetes. They are caused by Staphylococcus aureus of which 20% of ulcers are infected with methicillin-resistant strains. Streptococci, enterobacteria and anaerobes can be present in up to about 40% of ulcers.(19) Necrotizing fasciitis is also called streptococcal gangrene and can be caused by group A streptococcus, Clostridium

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perfringens or a mixture of aerobic and anaerobic bacteria. It is a severe infection that affects the superficial or deep fascia of skeletal muscle, soft tissue, progresses along the fascial planes, has a sudden onset, accompanied by fever and cellulite that progresses very rapidly to gangrene, caused by subcutaneous tissue necrosis, thrombosis and compromising the vessels that vascularize the skin.

Osteomyelitis (figure 12) due to diabetes is largely the result of a soft tissue infection that spreads to the bone, referring to inflammation or bone infection. It is a very commonly missed or underdiagnosed condition in the patient with diabetic foot disorders. Possible bone damage should be suspected in all patients with clinical findings of the infection, in chronic wounds or in case of ulcer recurrence. Osteomyelitis is most often due to incurable ulcers and is associated with an increased risk of amputation.(20,21)

Figure no. 12. Osteomyelitis lesions. Personal collection



CONCLUSIONS

Early recognition of diabetic foot injuries is extremely important, both for the diabetologist and for the general practitioner, as this can save the patient from mutation. The shorter the time from detection of the lesion to the initiation of treatment, the greater the chances of a cure. The variety of lesions and the possibility of their occurrence in the non-diabetic patient often leads to confusion and delayed diagnosis.

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