

# Training module

## Diabetic foot ulcers

epidemiology, diagnosis, local and systemic treatment

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# Diabetic foot ulcers

The neurological and vascular changes caused by diabetes expose the feet of diabetic patients to an increased risk of wounds, which are difficult to heal. These wounds are serious and costly. They lead to amputation in around 20% of cases. The crucial treatment for chronic diabetic foot ulcers is offloading. It is essential that patients receive therapeutic education to ensure effective prevention.

## 1 | Epidemiology

The seriousness of diabetes complications has made them a Public Health priority: retinopathy is the leading cause of acquired blindness, nephropathy is the leading cause of kidney failure, coronary-artery disease causes a third of myocardial infarctions in men and women, peripheral arterial disease and trophic disorders of the feet potentially lead to amputation.

The annual incidence of foot ulcers in diabetics is estimated to be around 2%. **Globally, 15% of diabetics have presented or will present a foot wound in their lifetime.**

Following a first wound, recurrences are common: **70% of patients having presented a wound have a recurrence within 5 years, with a mortality rate multiplied by 2.4 compared to patients without wounds.**

The cost of treating a diabetic foot ulcer is estimated to be between 8,000 and 30,000 euros depending on the severity of the wound and whether or not surgery is required.

The cost of a lower limb amputation is estimated to be 20,000 euros. More than half of all lower limb amputations concern diabetics and **50% of patients having already had one amputation will require another one within the next 5 years.**

Following an amputation, the number of survivors after 5 years does not exceed 60%.

Through their direct and indirect costs, the trophic foot disorders encountered in diabetics therefore constitute a major public health problem.

## 2 | Pathophysiology

The term “diabetic foot ulcer” groups together all the conditions affecting the foot and related to the consequences of diabetes. As the most distal contact with the ground, the foot is subject to the stress forces of weight-bearing and walking, making it a major target in the event of vascular alterations or impaired sensitivity.

Two mechanisms – often intrinsically linked – are involved in the development of foot ulcers in diabetics: **neuropathy** and **peripheral arterial disease**, to which **infection** must be added as an aggravating factor.

The vast majority of diabetic foot ulcers follow an often mild injury that has gone unnoticed due to the reduction (or absence) of sensitivity as a result of neuropathy. In the event of arterial disease, the skin is more fragile and tissue anoxia compromises healing.

Infection of the wound adversely affects the prognosis and is one of the risk factors for amputation.

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## ▶ A | Neuropathy

▶ This plays a major role in the pathogenesis of the lesions since it is involved in the development of 60 to 80% of initial lesions. It affects the sensory and motor peripheral nervous system, as well as the autonomic system.

- **Sensory damage.** Sensory disorders are a predominant feature and primarily concern **sensitivity to temperature and pain**. Consequently, anaesthesia to temperature and pain develops, leading to a loss of protective sensitivity to normally painful sensations. This **gradual loss of sensitivity** means that local micro-traumas and pressure are not felt by the patient and a wound may develop or worsen without any pain.

Pressure sensitivity disorders are detected using the “**monofilament test**” by applying a nylon fibre calibrated to 10 grams perpendicularly to the skin on the fleshy pads of the big toes and the head of the 1st and 5th metatarsals. This test should be performed at least once per year in all diabetics. The graduated tuning fork test is used to explore sensitivity to vibrations. In addition to objective signs of sensitivity, certain symptoms should be suggestive of neuropathy, remembering that they may be absent, however: paresthesia (numbness), night cramps, pins and needles, burning sensations, etc.

- **Motor damage.** This occurs at a later stage and causes atrophy of the intrinsic muscles of the foot and an imbalance between the flexor and extensor muscles leading to **deformities** such as metatarsal head protuberance and claw toes.

These modifications in the static position of the feet predispose the patient to the development of excessive plantar pressure and promote the development of ulceration on areas of excess pressure or friction.

- **The autonomic damage** leads to **loss of perspiration** and dry skin, promoting skin fissures and cracks.

Over an area of high pressure, dry skin promotes the development of pressure-induced hyperkeratosis, further accentuating the localised excessive pressure and creating a potential site for a trophic disorder.



The monofilament test is used to detect sensitivity disorders.



Foot deformities with claw toes and the presence of a hard corn (keratoma) related to excessive pressure.

## ▶ B | Peripheral arterial disease

▶ This is the **main cause of delayed healing** in diabetic foot ulcers and is associated with neuropathy in around 80% of cases. Pure ischaemic forms are rare and a diabetic foot is more frequently a neuro-ischaemic foot. Compared to non-diabetic subjects, arterial disease is more frequent and occurs earlier, with the lesions affecting the small arteries (microangiopathy) in addition to atherosclerotic damage to the tibial and fibular arteries. It is usually **asymptomatic in the event of associated neuropathy**. Thus, the absence of pain on walking and at rest explains the frequent delay in its diagnosis and the fact that is often revealed first of all by a trophic disorder.

Arterial disease makes the foot more vulnerable due to ischaemic damage to the tissues and adversely **affects the healing prognosis of lesions**. Investigation of arterial disease consists, first of all, of rigorous clinical examination of the distal, posterior tibial and pedal pulses; a reduction in or absence of these is a sign of the presence of upstream stenosis. Doppler ultrasound remains the exam of choice to detect obstructive arteriosclerotic lesions and determine the ankle-brachial index (ABI). Transcutaneous oxygen measurement (TcPO<sub>2</sub>) has a prognostic value in terms of the chances of spontaneous ulcer healing. Angiography is only performed with a view to revascularisation and to provide guidance with respect to the method to be selected as a function of the location of the lesions and the condition of the distal circulation.

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## ▶ C | Infection

- ▶ Although it is not one of the causes of trophic disorders in diabetic foot wounds, infection is a **major detrimental factor**, adversely affecting the wound healing prognosis and **responsible for 25 to 50% of amputations**.

The high level of vulnerability of diabetics to infections can be explained by anomalies in the bactericidal properties of neutrophils and local hypoxia of microvascular origin.

Infection is promoted by a poor glucose balance and the absence of offloading in the event of a wound.

It may be superficial, not exceeding the subcutaneous cellular tissue, but the **major risk is linked to its deep extension into the fascia, the tendons, the joints and the bone**.

The usual clinical signs of infection have a poor diagnostic sensitivity and may be reduced in diabetics, particularly in the event of neuropathy. The same is true for systemic signs, including fever and the usual biological markers.

## ▶ D | Development conditions

- ▶ In 80% of cases, diabetic foot ulcers have a **traumatic cause** (therefore avoidable) **which goes unnoticed due to the presence of neuropathy**. The wounds usually occur over areas of excessive pressure due to deformities of the foot or toes, hyperkeratosis, inappropriate footwear or the presence of foreign bodies in the shoe. Inadequate foot hygiene and foot care carried out by patients themselves on calluses and hard skin may promote the development of a wound. The same is true for the use of corn removal products.



Marked inflammation of the big toe related to the presence of a hyperkeratotic wound masking an underlying pus collection.

# 3 | Evaluation of diabetic foot ulcers

## ▶ A | Clinical aspects

- ▶ Although pure neuropathic or ischaemic forms such as those classically described are more rarely encountered today, recognition of the mechanisms underlying the clinical expression of the wound helps to guide management and treatment strategies, as well as the prognosis.

- **A neuropathic wound** typically presents as **perforating foot ulcer** above a metatarso-phalangeal joint at the plantar arch: the wound is **clear-cut, painless, devoid of necrotic sloughy deposits and surrounded by an area of hyperkeratosis**. The foot is dry and warm, with deformity. The pulses are present and there is a loss of sensitivity. As with all other wounds, this type of wound must be **urgently offloaded in order to heal**.



Bilateral malum perforans pedis typical of neuropathic feet (head of the 1st left metatarsal, head of the 5th right metatarsal). Each wound is ringed by a halo of hyperkeratosis, requiring ablation to reduce local pressure.

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- An **ischaemic wound** occurs against a background of distal hypoperfusion, as reflected by the absence of pedal pulses and the presence of **local signs of ischaemia: coldness, pallor, thin, shiny skin with loss of hair**. Sensitivity is maintained in the absence of concomitant neuropathy. The wound is usually located on an area of friction: **side of the foot or at a toe, without forgetting the foot**. It may be **painful**, often inflammatory and without hyperkeratosis.

- In contrast with a pure neuropathic wound, an ischaemic wound has the appearance of **local necrosis, initially dry** but at high risk of secondary infection spreading towards the neighbouring tissues (tendons, bones) and in which a progression to **wet gangrene** worsens the prognosis. A revascularisation procedure is usually necessary to obtain healing. The **risk of amputation** is high. **Offloading remains a priority**.

- A **mixed, neuro-ischaemic ulcer**, as is most often the case, is defined as ulceration with an appearance less typical than for the wounds described above but in a context combining anaesthesia to temperature and pain and arterial insufficiency. In terms of healing prognosis, priority should be given to assessment and **restoration of sufficient vascularisation**, which is a prerequisite to healing of the wound.



Arteriopathic wound of the outer edge of the 5th and 4th toes due to friction against footwear. Necrotic wound complicated by secondary infection with cellulitis of the adjacent tissues, as reflected by oedema and erythema around the wound.

## ► B | Wound assessment

► A precise assessment of the severity of the wound is essential to tailor its management. Wounds are now classified using **the University of Texas classification system**, which takes into account not only the arterial component but also the infectious component, this system having now replaced the traditional Wagner classification system, which is too imprecise (see tables 1 and 2). This determines the stage of the wound, upon which the type of local treatment will depend. It is possible to use a **red/yellow/black colour scale** to quantify granulation tissue, along with the presence of slough and necrosis. The abundance of exudates must also be quantified.

**The wound dimensions** with determination of the large and small axes of the wound, along with the **depth**, must be determined using a graduated ruler and should be recorded regularly in order to monitor the objective course of the wound. The skin around the wound should also be assessed.



Ulceration partially masked by a thick layer of hyperkeratosis requiring gradual trimming depending on the vascular condition of the patient to evaluate the wound as a whole.

### Table 1: Wagner classification

**Grade 0:** No open wound but possible presence of bone deformity or hyperkeratosis  
**Grade 1:** Superficial ulcer  
**Grade 2:** Deep ulcer penetrating down to tendons, bones or joints  
**Grade 3:** Tendinitis, osteomyelitis, abscess or deep cellulitis  
**Grade 4:** Gangrene of a toe or forefoot, usually associated with deep infection  
**Grade 5:** Extensive gangrene of the foot associated with necrotic lesions and soft tissue infection

### Table 2: University of Texas classification

This includes 4 grades depending on depth (Roman numerals) and four stages depending on the presence of infection and/or arterial disease)

**0:** Epithelialised lesion

**I.:** Superficial wound

**II.:** Tendon or capsule exposure

**III.:** Bone or joint exposure

**A:** Non-infected, non-ischaemic

**B:** Infected, non-ischaemic

**C:** Ischaemic, non-infected

**D:** Infected and ischaemic

Example: a wound rated IB is an infected superficial wound without ischaemia

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## ▶ C | Infection and local samples

- ▶ The clinical signs of local infection may be reduced in a diabetic patient and the infection may only be reflected by indirect signs: worsening of the wound, foul-smelling wound, discharge of modified or purulent exudate, sudden glucose imbalance, recurrence of pain. **Investigation of the wound using a dilator** must be performed to detect any **bone contact**, which is highly predictive of underlying osteitis. A local sample to identify the microorganism should only be taken in the presence of clinical signs of infection. There are numerous methods of taking these samples **following wound preparation** (washing of the wound with soap, rinsing, washing with normal saline): superficial swabbing, needle aspiration, deep curettage or biopsy.



Interdigital wound above the proximal inter-phalangeal joint. Investigation with a probe necessary to detect any bone contact.

# 4 | Treatment

The management of wounds by **multidisciplinary teams reduces the amputation rate by 45 to 85%**. Treatment is both local and systemic. Above all, **offloading** the wound is an essential requirement for healing. Consequently, the patient must be educated accordingly depending on the type of offloading chosen.

## ▶ A | Systemic management

- ▶ A strictly **controlled glucose balance** is essential and it may be necessary to begin (or optimise) insulin therapy. In addition, the patient's protein and calorie intake must be sufficient to allow wound healing.  
In the event of arterial disease, a **complete vascular assessment** will make it possible to judge whether a revascularisation procedure is feasible.  
An **infection** in a diabetic foot ulcer requires systemic antibiotic therapy based on an antibiogram. Pain management involves the usual measures. Finally, it must be ensured that the patient's tetanus boosters are up-to-date.

## ▶ B | Local treatment

- ▶ This consists of offloading, desloughing and the use of an appropriate dressing.

- **Offloading**

- Offloading is imperative** in order to prevent any pressure or friction that would obstruct healing, but patient compliance is poor since offloading devices are often not very aesthetic and difficult to use. In addition to complete bed rest in the case of a hospitalisation, a variety of devices are possible depending on the location of the wound: temporary shoes for anterior or posterior offloading for plantar wounds of the forefoot, removable standard or made-to-measure boots, non-removable resin boots, open or otherwise, etc.

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## • Desloughing

This will depend on **the patient's vascular condition**, hence the importance of the wound aetiology.

Mechanical elimination of **necrotic and sloughy debris** and any **hyperkeratosis** is essential for wound healing.

**In the event of hyperkeratosis** around a malum perforans pedis ulcer, its tangential removal using appropriate instruments (scalpel, curette, etc.) eliminates the ring of callus, relieves pressure around the ulceration and promotes peripheral progression of neo-epidermis.

**In wounds with an ischaemic component, desloughing must be performed with care** and may be done following a potential revascularisation procedure. However, dry, non-inflammatory necrosis should be left in place.

In the event of deep ulceration with osteo-arthritis, infected moist necrosis and infectious cellulitis, desloughing should be performed by a surgeon.

## • Wound healing and dressings

As with all wounds, the principle of **moist-environment healing** is recommended for diabetic foot ulcers. Local antibiotics should be avoided, as should eosin antiseptic, which masks the signs of inflammation, long-term antiseptics, plasters and adhesive dressings on fragile skin. The wound should be cleaned after washing with mild liquid soap and normal saline.

Dressings should be changed frequently to enable careful monitoring of the wound and any signs of exacerbation.

**The choice of dressing** depends on the stage of the wound, the abundance of the exudates, the depth of the wound, the presence or otherwise of infection and the condition of the surrounding skin.

Hydrocolloid dressings are not recommended since they are occlusive and adhesive.

The choice should preferentially be made, depending on the stage of the wound between hydrogels in the event of dry necrosis, **but with care for wounds with an ischaemic component**, and alginates and related dressings for exuding, sloughy wounds.

Hydrocellular dressings and protease inhibitor dressings will be useful at the granulation stage, and contact layer dressings during epithelialisation.

Negative pressure therapy can be used in the event of deteriorating wounds to promote granulation before performing skin grafting. The value of a hyperbaric chamber may be discussed in specialised departments.



Mixed wound with sloughy deposits requiring desloughing depending on the vascular condition.



Thanks to their high level of flexibility, lipidocolloid contact layers (TLC range) can fit awkward locations.

## 5 | Prevention

A diabetic foot ulcer prevention programme can reduce the ulceration rate by 50%.

### ▶ A | Identification of feet at risk

- ▶ The feet of diabetics should be examined **at least every year** to identify feet at risk and adapt preventive measures. Diabetic patients should be graded to determine the wound risk.

Risk grade	Definition	Monitoring
0	Absence of neuropathy	once per year
1	Isolated sensory neuropathy	once per 6 months
2	Neuropathy combined with arterial disease and/or deformity	once per 3 months
3	History of wound > 4 weeks and/or amputation	once per month

In the event of deformities, the prescription of made-to-measure orthotics and shoes may be necessary. High-risk diabetic feet (grade 3) should be managed in specialised centres. It is strongly recommended that patients be monitored by a podiatrist.

### ▶ B | Therapeutic education

- ▶ Grade 1, 2 and 3 patients must receive podiatric education, in particular:
  - **Careful daily inspection** of the feet, using a mirror if necessary or with the help of a member of the family.
  - **Daily washing** with warm, soapy water, drying carefully between the toes with a towel. Ban foot baths: risk of maceration and facilitation of deep penetration of microorganisms.
  - **Systematic wear** of socks made of cotton or wool, to be changed every day.
  - **Wearing of appropriate footwear** (no flip-flops, sandals or open-toed shoes) that is wide enough and systematic inspection of the inside of shoes to check for aggressive elements (seams, foreign bodies, etc.).
  - **Never walk barefoot**, wear rubber shoes when swimming in the sea.
  - **Be careful around all heat sources** (fireplace, hot water bottle, etc.).
  - **Do not use metal instruments or corn removing products for foot care**, cut nails straight across.
  - Do not hesitate to have a **professional perform pedicure procedures**.
  - **Consult a doctor** at the slightest sign of ulceration or foot blisters.



Dry, scaly skin, unguis fragility and nail of the big toe at high risk of becoming in-growing. To stress the importance of educating patients and foot care by a specialised podiatrist.

## 6 | Conclusion

As is confirmed by epidemiological data, foot ulcers in diabetic patients have major human and social repercussions due to the ultimate risk of amputation and the impact on the patient's lifespan. Only an active detection and prevention policy led by health professionals among at-risk patients has already demonstrated that it can reduce the development of ulcers in diabetics by half.

It is with this objective that this training programme has been developed with the assistance of professionals in daily contact with at-risk diabetic patients.



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