REVIEW ARTICLE

Diabetic foot: The orthopedic surgery angle

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Abstract  As diabetes takes on pandemic proportions, it is crucial for the orthopedic surgeon to be aware of the issues involved in diabetic foot. Ulceration is related to neuropathy and to arterial disease, a vital prognostic factor for healing; infection plays an aggravating role, increasing the risk of amputation. At-risk feet need to be screened for. Ulcer classification is essential, to set treatment strategy and determine prognosis. Before any treatment is decided on, neuropathy, vascular insufficiency and infection should individually be assessed by clinical examination and appropriate additional work-up. Despite the International Consensus on the Diabetic Foot recommendations, management of diabetic foot in Europe still varies greatly from country to country, very few of which have established reference centers. Management of diabetic foot remains multidisciplinary; but it has been shown that the orthopedic surgeon should play a central role, providing a biomechanical perspective so as to avoid complications recurrence. Strategy notably includes prevention of at-risk foot, revascularization surgery (which should systematically precede orthopedic surgery in case of critical vascular insufficiency), and treatment of ulcers, whether these latter are associated with osteitis or not. Indications for "minor" amputation should be adequate, and meticulously implemented. "Acute foot" is a medical emergency, entailing massive empirically selected I.V. antibiotics to "cool" the lesion. Prophylactic surgery to limit further risks of ulceration is to be indicated with caution and only when clearly justified. France urgently requires accredited specialized multidisciplinary centers to manage severe lesions: deep and infected ulceration, advanced arteriopathy, and Charcot foot arthropathy.

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Introduction

The incidence of diabetes, especially of type 2, is increasing throughout the developed world; the associated complications make this a real challenge for public health. There are presently estimated to be more than 120 million diabetics worldwide, expected to rise to 333 million by 2025. As well as acute metabolic complications, diabetes induces chronic complications related to vascular damage and secondary neuropathy, mainly affecting three locations: eyes, kidneys and feet. Management of diabetic foot lesions has long been neglected.

We will present the classifications of diabetic foot lesions and the means of diagnosis, and attempt to answer the questions facing the orthopedic surgeon:

- how to organize patient management?
- what are the respective roles of orthopedic and vascular surgery?
- how to treat perforating plantar ulcer, with and without associated osteitis?
- is “acute foot” a surgical or a medical emergency?
- what is the place of the prophylactic surgery and the risks of orthopedic surgery in diabetic patients?

The present article is not the place to go into the complex issue of neuro-arthropathy, which requires management in a specialized center.

General considerations

Epidemiology

Studies of diabetic foot and the results reported vary greatly according to the population under study, the diagnostic criteria applied and the degree of specialization of the centers concerned.

Diabetic foot ulcer

Prevalence data are relatively plentiful, but range from 3 to 25% according to the study, country or even region [1–4]. It is presently estimated that 15% of diabetic subjects present ulcer at some point in their life [5].

Amputation

Diabetic foot lesions entail a 15- to 20-fold increase in amputation risk as compared to the general population [6]. Annual incidence varies greatly according to country and region [7]. Some 50% of amputations are in diabetic patients, [8,9] and it is estimated that 5–15% of diabetics will undergo amputation at some point in their life. Age, sex (male), low socioeconomic status and other diabetic complications (notably renal insufficiency, and especially in case of dialysis) specifically increase amputation risk.

Lesion prognosis

Diabetic foot lesions have functional and psychological consequences and severely impact quality of life [10]. Ulcers show frequent recurrence, and reduce life expectancy. Apelqvist et al. [11] demonstrated the negative prognostic implications of amputation in diabetes: a second amputation is necessary within 5 years in 50% of cases, with 58% survivorship.

Physiopathology

Diabetic ulcers show three main distinct but interacting factors: neuropathy and arteriopathy are secondary causes of diabetes, and infection is a decompensation factor.

Neuropathic and arteriopathic complications are seldom isolated but rather associated to a varying degree and leading to neuro-ischemic foot vulnerable to ulcer.

Peripheral neuropathy

The exact prevalence of neuropathy in diabetes is estimated at 20 to 60% depending on the diagnostic procedure, and increases with chronic hyperglycemia, duration of diabetes and patient age. Neuropathy is found in more than 90% of cases of diabetic foot ulcer. It is bilateral, symmetrical and distal, showing ascendant evolution.

Sensory neuropathy. Sensory disorder predominates; signs depend on the type of neural fiber involved. Large fibers are involved in tactile and deep sensitivity, and small fibers in pain and heat sensitivity. Trauma and friction lesions thus become silent.

Motor neuropathy. Motor neuropathy induces weakness and atrophy of the intrinsic muscles of the foot, leading to claw toe. Secondarily, it contributes to loss of joint mobility, which is also due to conjunctive tissue glycosylation inducing fibrosis of the joint, soft tissue and skin.

Vegetative neuropathy. Vegetative neuropathy induces skin dryness with crevasses and fissures providing entry points for infection; it contributes to hyperkeratosis in reaction to hyperpressure. It also opens arteriovenous shunts and induces deregulation of capillary flow: the neuropathic foot is hot, with frequent edema and dilated dorsal veins.

Consequences. Peripheral neuropathy is the main factor in diabetes of atonic ulcer (Fig. 1) with highly hyperkeratotic peripheral halo and neurogenic osteo-arthropathy (Charcot foot).

Arteriopathy

Arteriopathy is usually associated to a varying degree with neuropathy (neuro-ischemic foot), with a low rate of isolated ischemic lesions (around 20%).

Macroangiopathy. Macroangiopathy is not specific to diabetes, but shows specific characteristics. Atheromatous lesions develop earlier and more rapidly, classically showing multi-segment and distal involvement. It mainly affects the distal superficial femoral, popliteal, tibial, peroneal and pedis arteries; aorto-iliac locations are rare.

Microangiopathy. Microcirculatory effects, characterized by thickening of the capillary membrane, induce abnormal exchange and aggravate tissue ischemia. The role of microangiopathy remains controversial.

Consequences. Diabetic arteriopathy progressively induces chronic ischemia, which is an aggravating factor in foot lesions. The foot is cold and the skin becomes thin and shiny.
Ulcers of ischemic origin are often secondary to slight trauma. Unlike neuropathic ulcers, they show an erythematous halo without hyperkeratosis. Associated heel sore, induced by decubitus, is a chronic lesion with poor prognosis. Decompensation of such distal arteritis may lead to ischemia or gangrene in one or more toes by primitive acute distal thrombosis (Fig. 2). Infection is always an aggravating general factor.

**Biomechanics of ulceration**
The association of diabetic neuropathy and plantar hyper-pressure is the cause of most ulceration. Loss of pressure and pain sensitivity leads to repeated local hyperpressure and shear stress in the hyperkeratotic region, under which effusion develops and exteriorizes into an ulcer. Moreover, any mechanical, thermal or chemical wound may also lead to ulceration, diagnosed late due to the absence of associated pain.
Table 1  Plantar hyperpressure and ulceration factors.

<table>
<thead>
<tr>
<th>Plantar hyperpressure and ulceration factors</th>
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<tbody>
<tr>
<td><strong>Intrinsic factors</strong></td>
</tr>
<tr>
<td>Foot morphology (pe cavus, hallux valgus, claw toe, etc.)</td>
</tr>
<tr>
<td>Plantar hyperkeratosis</td>
</tr>
<tr>
<td>Limited joint mobility</td>
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<tr>
<td>Severe foot deformity (Charcot foot)</td>
</tr>
<tr>
<td><strong>Extrinsic factors</strong></td>
</tr>
<tr>
<td>Non-adapted footwear (too tight, projecting seams)</td>
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<td>Foreign body (pebble, nail, etc.)</td>
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<tr>
<td><strong>Behavioral factors</strong></td>
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<tr>
<td>Barefoot walking</td>
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<tr>
<td>Lack of daily foot surveillance</td>
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<tr>
<td>Impossibility of self-care</td>
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<tr>
<td>Poor hygiene (non-treated hyperkeratosis)</td>
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<td><strong>Iatrogenic factors</strong></td>
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<tr>
<td>Maladapted nail care</td>
</tr>
<tr>
<td>Badly performed amputation</td>
</tr>
<tr>
<td>Resection of 1 or more metatarsal heads</td>
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<tr>
<td>Escalating amputation</td>
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</table>

**Plantar pressure threshold.** Most ulcers occur on the small toes or hallux, facing the metatarsal heads. In the literature, it is agreed that there is no predictive threshold for ulceration. The threshold depends on a large number of factors and varies between subjects. The most recent studies focus not only on contact time and degree of pressure, but more particularly on the 3D direction and propagation of pressure in soft tissue. Table 1 shows the factors of plantar hyperpressure and ulceration.

**At-risk feet**

A classification of at-risk feet is essential for drawing up prevention strategies. The International Working Group on the Diabetic Foot (IWGDF) published a 5-group classification according to complication rates (Table 2) [12]. A French multicenter survey performed on a single day for all diabetic patients in or consulting in 16 diabetology centers classified 17.5% as at high risk (groups 2 and 3) [13].

**Ulcer classification**

A classification of diabetic feet is essential for drawing up diagnosis and treatment strategies and to aid prognosis. It further facilitates therapeutic assessment and communication between the teams involved.

Table 2  At-risk foot groups.

<table>
<thead>
<tr>
<th>At-risk groups</th>
<th>Criteria</th>
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<tbody>
<tr>
<td>Group 0</td>
<td>No neuropathy, orthopedic deformity, vascular disorder, foot wounds or history of wound/amputation</td>
</tr>
<tr>
<td>Group 1</td>
<td>Neuropathy</td>
</tr>
<tr>
<td>Group 2a</td>
<td>Neuropathy associated with orthopedic deformity, but adequate joint motion</td>
</tr>
<tr>
<td>Group 2b</td>
<td>Neuropathy and orthopedic deformity associated with joint stiffness</td>
</tr>
<tr>
<td>Group 3</td>
<td>Neuropathy associated with one of the following: Arteriopathy, Charcot foot type deformity (acute or chronic), History of wounds History of major or minor amputation.</td>
</tr>
</tbody>
</table>

Several classifications have been published internationally:

- Wagner’s classification [14];
- the Texas classification, reported by Armstrong in 1996 [15];
- Mike Edmonds’ classification;
- the PEDIS classification [16].

We will describe just Wagner’s classification, the most widely used, and the PEDIS classification, the most recent, based on an international consensus. None of the classifications, however, take account of neurogenic osteoarthropathic foot (Charcot foot).

Wagner (Table 3) classifies lesions in six grades of increasing severity, 0—5. Grades 1 to 3 are basically neuropathic ulcers of increasing severity according to depth and infection, while grades 4 and 5 are vascular lesions. The classification is simple, but fails to take account of the degree of vascular insufficiency that may be associated with grades 1–3.

The PEDIS classification is based on five parameters (Perfusion, Extent, Depth, Infection and Sensitivity) [17] that are important in treating wounds in diabetic subjects (Table 4). Each diabetic wound can be described by five elements, individualizing prognosis. The classification is thus more precise than Wagner’s. Most ulcers are induced by neuropathy, but vascular status determines prognosis. Infection is an extra severity factor for limb prognosis and patient survival.

Table 3  Wagner classification grades.

<table>
<thead>
<tr>
<th>Wagner grade 0</th>
<th>Wagner grade 1</th>
<th>Wagner grade 2</th>
<th>Wagner grade 3</th>
<th>Wagner grade 4</th>
<th>Wagner grade 5</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High-risk foot</strong></td>
<td>Very superficial non-infected ulcer</td>
<td>Very deep infected ulcer, limited cellulitis</td>
<td>Very deep infected ulcer with tendon/fascia and/or bone involvement</td>
<td>Limited gangrene</td>
<td>Extensive gangrene</td>
</tr>
</tbody>
</table>
Table 4  PEDIS classification.

| Perfusion | Grade P1 | No symptoms, no signs of peripheral arteriopathy  
|           | (ABI: 0.9–1.1 or TcPO2 > 60 mmHg) |
| Grade P2 | Symptoms or signs of peripheral arteriopathy, no critical limb ischemia |
| Grade P3 | Critical limb ischemia  
|           | (TcPO2 < 30 mmHg or Systolic ankle pressure < 50 mmHg) |
| Extent   | Wound size (cm²) after debridement |
| Depth    | Grade D1  
|          | Superficial dermal ulcer |
| Grade D2 | Deep ulcer, penetrating below dermis to subcutaneous structures, involving fascias, muscles or tendons |
| Grade D3 | All following layers, inc. bone and/or joint  
|           | (bone contact or ulcer penetrating to bone) |
| Infection| Grade I1  
|          | No symptom or sign of infection |
| Grade I2 | Infection involving skin and subcutaneous tissue  
|          | (at least 2 of the following: local edema or induration, erythema > 0.5–2 cm, pain on pressure, local heat, purulent effusion) |
| Grade I3 | Erythema > 2 cm plus one of the above  
|          | (edema, pain on pressure, heat, effusion)  
|          | or deeper infection (abscess, osteomyelitis, septic arthritis, fasciitis, etc.) |
| Grade I4 | Infection with systemic signs.  
|          | (at least 2 of the following: temperature > 38° or < 36°, heart rate < 90/min, resp. rate > 20/min, PaCO₂ < 32 mmHg, GB > 12000, 10% non-differentiated leukocyte forms) |
| Sensation| Grade S1  
|          | No loss of protective sensation |
| Grade S2 | Loss of protective sensation |

**Neuropathy**

Neuropathy associated with diabetes is progressive but silent. It should therefore be systematically looked for as part of any foot examination in diabetic patients. When diagnosed, even without wound or history of wound, specific preventive education is mandatory, as neuropathy is a factor in foot ulcer. Almost all diabetic patients with ulcer show sensory neuropathy; Charcot foot is another consequence of neuropathy.

**Screening**

Two simple tests should be known. 10 g 5.07 monofilament. Semmens-Weinstein monofilaments are a rapid means of exploring pressure sensitivity. The 5.07 curved nylon monofilament (equivalent to 10 g, corresponding to the sensation level required to avoid foot ulceration) is applied perpendicularly on the skin. Several plantar sites are explored, of which 3 must be sensitive: hallucal, pulpal, and 1st and 5th metatarsal heads. Each site should be tested three times in succession including one sham application in which the monofilament is not applied. This is the most reliable screening method, and it is cheap and within anyone’s capacity! 128 Hz tuning fork. The tuning fork explores vibratory sensitivity on the dorsal side of the 1st metatarsal.

**Other examinations**

Thermo-algesic sensitivity can be assessed on Neurotherm® (hot/cold test) and calibrated Neurotip test. Clinical neurologic examination may if necessary be associated to electrophysiological examination.

**Vascular insufficiency**

Vascular involvement should be systematically investigated as associated neuropathy generally masks the classical symptoms (notably, pain).

Clinical examination may be misleading. Pale cold skin, the classic sign, is often not observed due to associated vegetative neuropathy. Due to mediastinal, dorsalis pedis and posterior tibial pulse does not mean there are no microangiopathic lesions. Table 5 presents the various vascular tests and imaging modalities.

**Infection**

Infection is the aggravating factor in diabetic ulcer, and may show rapid evolution, causing an emergency.

Clinical examination of the foot systematically looks for any entry point. Any general signs are noted: temperature, and heart and respiratory rate elevation. Biological examination analyzes diabetes balance, blood count, erythrocyte sedimentation rate and CRP assay.

**Clinical examination**

Reddening, tumefaction and erythema indicate soft-tissue inflammation. Infection is often deeper than estimated. A “sausage-like” edematous erythematous aspect in a toe suggests osteoarthritis. Perforating ulcers should be
<table>
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<th>Table 5</th>
<th>Vascular tests and imaging.</th>
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<tbody>
<tr>
<td><strong>Vascular tests</strong></td>
<td><strong>Aim</strong></td>
</tr>
<tr>
<td><strong>Ankle/arm index and TcPO2</strong>&lt;br&gt; <strong>Systolic ankle/arm pressure index (AAI)</strong></td>
<td>Demonstrate lower limb arteriopathy and estimate severity by pocket Doppler</td>
</tr>
<tr>
<td><strong>TcPO2</strong> (transcutaneous measurement of partial oxygen pressure)</td>
<td>Assess cutaneous oxygenation</td>
</tr>
<tr>
<td><strong>Radiology</strong></td>
<td><strong>Aim</strong></td>
</tr>
<tr>
<td><strong>Vascular imaging</strong></td>
<td>Screen for diabetic arteriopathy</td>
</tr>
<tr>
<td><strong>Echodoppler</strong></td>
<td>Diagnose lower-limb arterial stenosis</td>
</tr>
</tbody>
</table>

examined using a stylus or sterile forceps: “rough” bone contact indicates osteitis or osteoarthritis unless proved otherwise (Fig. 3).

**Bacteriology**

In the absence of clinical or general signs of infection, it is not recommended to take bacteriological samples, culture of which would show only colonization flora.

In case of superficial or deep infection, on the other hand, bacteriology is essential.

It may comprise:

- **Swabbing.** This requires very strict conditions, to avoid contamination: debridement of necrotic tissue, no antiseptics, washing of the foot in water then of the wound in physiological saline. This should be repeated several times in consultation. To be contributive, several concomitant results are necessary. Deep ulcer sometimes requires deep sampling, or bone sampling by curette.
- **Needle puncture of effusion,** either percutaneous or under ultrasound control.
- **The gold standard remains surgical bone and deep soft-tissue biopsy.**
Superficial infection is usually mono-microbial (Staphylococcus aureus, Streptococcus, etc.) and deep infection multi-microbial (Gram+, Gram− and anaerobic). Bacteriology samples taken in consultation, even under strict conditions, lack specificity, which is to be borne in mind in establishing the appropriate antibiotherapy [18].

Osteo-articular imaging
Infection can be assessed on standard X-ray, CT, MRI and isotopic examination.

Standard X-ray. Signs of osteitis occur later than onset of infection; moreover, ostetic and neuro-arthritic lesions (Charcot foot) can be hard to distinguish. A metaphyseal-diaphyseal lytic aspect, however, is relatively characteristic of osteitis, especially in the forefoot. We recommend systematic standard X-ray in PPU and suspected osteitis, with comparative assessment at 1 and 2 weeks: in case of osteitis, osteolysis, which was initially absent, will be visible at 2 weeks. This is a simple and essential comparative examination to assess osteo-articular infection [19].

CT. CT usefully confirms osteolysis in case of ambiguous X-ray.

Gadolinium-enhanced MRI. The literature [20] recognizes gadolinium-enhanced MRI as a good means of diagnosing osteitis. It differentiates osteoarthritic from neurogenic osteo-arthropathic lesions [21]. We reserve it for “acute foot” with cellulitis. It is the examination of choice for diagnosing deep soft-tissue effusion and extension to tendon sheaths, and serves to guide surgical drainage.

Ultrasound. Ultrasound diagnoses effusion and abscess, and may guide puncture for bacteriology.

Isotopic examinations. In case of uncertain diagnosis on standard X-ray and/or CT, we consider bone technetium scintigraphy using labelled polynuclears to be the examination of choice for diagnosis of osteitis.

Organization of management

International Consensus on the Diabetic Foot

According to the International Consensus on the Diabetic Foot, published by the IWGDF in 1999, prevention and treatment of complications in diabetic foot should be organized at three levels.

Level 1: GPs, nurses and podiatrists
Patient awareness of foot problems and prevention, and of early diagnosis of ulceration.

Level 2: Diabetologists, diabetology nurses, surgeons (general and/or vascular and/or orthopedic)
Management of basic preventive and curative care:

Level 3: Reference centers
Reference centers should be capable of close multidisciplinary teamwork between diabetologist, orthopedic surgeon and vascular surgeon, to manage the most difficult cases: deep infected ulcer, severe arteriopathy, Charcot foot.

Reality is often far from such an ideal. Several studies found that less than 50% of diabetic subjects had annual foot examination, whether by their GP or their diabetologist [22,23], and that home check-ups remained too few, ranging from 20 to 70%.

In 2008, a prospective European study [24] in 14 centers still found treatments that failed to respect international recommendations, with wide variations between countries and centers.
Treatment

Treatment of diabetic foot is usually multidisciplinary, involving different specialties. Vascular and infectious ulcer assessment allows adapted treatment. Basic principles need to be respected: non-weight-bearing, debridement, control of infection, revascularization if necessary, and adapted wound care. Ulcer classification enables the various physicians to use the same tools and particularly to compare results across treatment protocols.

Prevention in at-risk feet

Only preventive measures can limit the incidence of ulcer and amputation and the costs incurred by diabetic foot. They are based on general (optimal glycemic balance, prevention of associated cardiovascular risk, smoking cessation, etc.) and specific measures (podiatry, orthoses, adapted footwear and patient education). Primary prevention begins with screening for risk of ulcer; this involves systematic, at least once-yearly foot examination, notably for neuropathy and foot deformity, and also education in the risk of specific foot complications. Secondary prevention in patients with grade 1–3 risk feet associates education, systematic screening and multidisciplinary follow-up.

Education

Education concerns both the patient and the care workers. The patient. Education is individual and adapted to the patient’s complications and sociocultural level. It comprises several axes: daily self-examination of the foot, podiatry, permanent use of adapted footwear with prohibition of barefoot walking, use of natural fiber seamless socks and stockings, avoidance of aggressive substances and burns, not raising the feet at rest, and early recognition of lesions requiring immediate consultation. The patient’s family is to be involved in education and prevention. Health-care workers. All of the health-care professionals must co-ordinate their actions to avoid contradiction. They should therefore be brought together in a multidisciplinary team, and any center managing diabetic foot should have a nurse specialized in diabetology and education, as well as a podiatrist.

Footwear

Various devices are available to prevent onset or recurrence of foot ulcers. Plantar orthoses. Insoles have a preventive and sometimes curative function. Basically, they distribute pressure, more rarely with corrective elements. Orthoplasties. Orthoplasties are little molded silicone devices that protect areas of conflict with the shoe (notably at the toes). Shoes. Shoes are essential to prevention. They may be adapted mass-produced models, semi-therapeutic or made-to-measure orthopedic shoes.

Role of vascular surgery

Before treating any diabetic ulcer, it is essential to correct vascular insufficiency. Amputation or orthopedic surgery should never be indicated before precise assessment of lower limb vascular status. Anesthesia should bear in mind that coronary involvement is often silent in diabetes. Critical lower limb ischemia in diabetes is seen clinically as an ulcer with necrosis, pain in decubitus or claudication that is intermittent but more frequent than in non-diabetics due to the associated neuropathy. Revascularization, when feasible, is essential to ulcer healing, enabling tissue oxygenation and better diffusion of antibiotics.

In practice, vascular assessment in case of ulcer comprises Doppler and if possible TcPO₂ measurement: if this is < 30 mm Hg, vascular advice is mandatory (P3 on the PEDIS classification). Depending on the complementary examination results, revascularization, using whatever technique, may be envisaged (Fig. 4).

The main revascularization procedures are: distal bridge, endovascular techniques, stenting and percutaneous intentional extra-luminal revascularization. Lumbar sympathectomy is not indicated in lower limb diabetic arteriopathy. Hyperbaric oxygen therapy may be used in case of ulcer with associated non-revascularizable severe arthritis.

Foot ulcers without osteitis

These lesions are usually managed non-surgically, on an ambulatory basis in medical (diabetic podiatry) consultation (D1 and D2 on the PEDIS classification). Treatment comprises non-weight-bearing for the affected foot and wound cleansing and dressing.

Non-weight-bearing

This is the essential and fundamental element of treatment, without which any hope of healing is chimerical. The two basic principles of local offloading and wound debridement are mandatory. Complete bed-rest. Complete bed-rest is the ideal guarantee of non-weight-bearing, but is unrealistic over a long period. It is prescribed during the acute phase of the wound,
associated to use of a wheelchair, or crutches for hygiene care. It is also often an opportunity to take stock of the patient’s situation.

**Postoperative shoes.** We mainly prescribe Barouk™ forefoot or Sanital™ hindfoot pressure-relief shoes. Being removable to allow for dressings and avoid the hyperpressure points induced by cast immobilization, they entail a problem of strict compliance with non-weight-bearing.

**Offloading casts.**

*The Total Contact Cast (TTC).* TTC is the reference treatment for ulcer and acute Charcot foot. The aim is to achieve homogeneous pressure distribution in the plantar arch throughout the step: 30 to 50% of pressure is absorbed by the cast [25]. Its non-removable 24/24 concept is fundamental to success, enabling healing in 70 to 85% of cases. However, it requires great experience in production (Fig. 5). Efficacy in terms of plantar pressure relief and healing is better in the fore- and mid-foot than in the hindfoot [26,27]. It should be changed weekly. The associated complications rate varies from 5 to 30% [28]: friction lesions liable to induce new infected wounds, venous thrombosis, etc.

*Fenestrated [29] and/or removable casts [30].* These enable the wound to be monitored, and reduce the risk of complications. However, while the window around the ulcer makes dressing easier, it can also induce surrounding hyperpressure.

*Commercial removable pneumatic casts (Aircast™).* They are an alternative when qualified cast-makers are not available. They are a little less effective in offloading [31]; removability can be countered by using resin tape [32].

**Efficacy.** Due to compliance issues, non-removable casts give shorter healing times than postoperative shoes and pneumatic casts [33]. The Total Contact Cast is the gold standard according to the IWGDF consensus, but is in fact only used by a few specialized teams [34].

**Wound cleansing**

Wound care begins with disinfection of the wound itself and the surrounding area: careful washing with water, then application of antiseptics; polyiodine solutions are more effective than chlorhexidine and do not affect healing, while preventing the emergence of resistant bacteria (MRSA) [35]. Local antibiotics are not indicated, for the same reasons.

*Any ulcer requires limited debridement in consultation,* using a lancet knife to remove surrounding callus. The wound can then be contoured by curette to remove any yellowish necrotic residue or fibrin. This mechanical cleansing is essential, in order to promote healing. Some ulcers may require removal of necrotic structures and surgical cleansing.

**Dressing**

In the absence of any rigorous comparative studies, there is no consensus as to the type of dressing to use in diabetic foot. Dressings should, however, have a certain number of properties: maintaining a humid microclimate, absorbing exudate, protecting from bacterial contamination, and being replaceable without local trauma. Choice depends on the type and location of the wound.

Biotechnological approaches have been studied in diabetic foot: growth factors (mixtures of PDWHF, PGDF, etc.), platelet gels, live skin substitutes (epidermal, dermal or composite). Physical approaches such as VAC (Vacuum Assisted Closure) and hyperbaric oxygen therapy are sometimes recommended to accelerate neovascularization and healing.

**Role of surgery**

In PEDIS D1 and D2 lesions, orthopedic surgery may be indicated to promote healing or avoid recurrence in resistant forefoot ulcer. There is thus the possibility of percutaneous lengthening of the Achilles tendon, or sectioning the gastrocnemial aponeurotic lamina in case of ankle stiffness without dorsiflexion or even with slight equinus [36,37]. Likewise, metatarsal elevation osteotomy in case of hyperpressure caused by stasis disorder, or percutaneous distal osteotomy of the lateral metatarsals are possible, to relieve hyperpressure on a PPU. The aim of such surgery is to reduce mechanical stress in the forefoot.

In case of significant loss of substance secondary to surgical cleansing, plastic surgery may make a contribution if vascular status is satisfactory (P1, P2 on the PEDIS classification) and factors of hyperpressure can be modified. Grafting may use a small bilobed rotation flap after plantar ulcer resection, or sural (or other) local flaps for heel-sore sequelae.

**Foot ulcers with osteitis**

The association of osteitis and ulcer (PEDIS D3) requires prolonged antibiotherapy, generally beginning with a parenteral course, and managed in coordination with the infectologists. There is, however, no international consensus on infectious lesion management in diabetes [38]. Multidisciplinary management may call on surgeons for revascularization, bone biopsy, bone curettage or minor amputation. It is to be borne in mind that residual osteitis or secondary induction of hyperpressure or of deformity may cause footwear issues and entail a risk of recurrence. Any surgery in case of osteitis should leave a foot that is functional and balanced in terms of tendons. Vascular status and the orthotic options need to be reconsidered after each operation.

**Amputation**

The aim in amputation is to obtain a stump that can easily be fitted, to conserve as great a length as possible while enabling direct closure, and to conserve the patient’s autonomy. Whenever possible, minor amputation fully conserving limb length is to be systematically preferred to full-leg or above-knee amputation.

**Forefoot**

We try to think in terms of function and biomechanics, rather than purely of ulcer and osteitis, in deciding on the level and extent of any amputation [5,18,39]. It is important to avoid progressively slicing away at the toes, which is harmful both generally and psychologically: leaving two or three middle toes on a forefoot is a mechanical absurdity which can only lead to rapid recurrence of ulceration (Fig. 6). In the forefoot, in case of osteitis facing an ulcer, we opt for curative orthopedic surgery, rather than prolonged exclusively medical management guided by bone biopsy or even
diabetic foot: the orthopedic surgery angle

curettage or minimal bone surgery [40] associated to pro-
longed antibiotic therapy. With this “carcinologic” attitude of
minor orthopedic amputation (removing all infected tissue,
extending into the healthy bone) associated to primary clo-
sure and a postoperative antibiotic spectrum of less than
1-month, we have obtained 91% recurrence-free recovery
from osteitis, with a mean cicatrization time of 33 days [19].

According to location, we recommend the following bone
surgery procedures.

Partial toe amputation. We avoid complete amputation,
especially of the 2nd toe, that would induce or increase
hallux valgus. Likewise, 5th toe amputation may induce
5th metatarsal head conflict and hyperpressure, as the lat-
eral side of the foot exerts a braking effect and the hallux

Figure 5  Total Contact Cast. A and B. Protection of forefoot with Velband® separating toes. C. Leg and foot covered by stocking
and then wadding. D. Successive layers of plaster. E. Resin reinforcement. F. Device with absorbent sole to allow walking. G. Example
of ulcer managed with TCC. H. Result at 6 weeks.
a propulsion effect during gait. The objective is interphalangeal amputation with maximal conservation of the proximal phalanx. A ‘‘shark’s mouth’’ incision is preferable, conserving a more richly vascularized pulpar flap.

**Transmetatarsal ray amputation.** We use transmetatarsal ray amputation as an alternative to complete toe amputation, especially for the 2nd and 5th rays, with very satisfactory results, avoiding hyperpressure in the remaining head, which would induce recurrence. Resection of a single lateral ray has little impact on foot width, inducing very little pathologic overload in the adjacent rays (Fig. 7). The 5th metatarsal should be osteotomized obliquely. However, we do our best to avoid hallux or even 1st ray amputation, which would impact the lateral rays, inducing claw toe. In case of hallux lesion, we try to cure the osteitis by antibiotic therapy, possibly associated to Keller arthroplastic resection. When hallux amputation is necessary, 1st metatarsal length should be conserved as much as possible, so as to allow for possible secondary transmetatarsal amputation.

**Isolated metatarsal head amputation.** We occasionally perform isolated amputation of the metatarsal heads. Metatarsal head osteitis is often associated with fixed claw toe or vascular involvement in the toe, which is rather an indication for ray amputation. Moreover, isolated amputation of the metatarsal head is seldom satisfactory in terms of infection control. On the other hand, in certain cases and notably in surgical revision, alignment resection of all the lateral metatarsal heads may be indicated, as in rheumatoid polyarthritis.

**Transmetatarsal amputation.** Transmetatarsal amputation may be considered when it is not possible to conserve at least three metatarsals on the lateral rays or four if the 1st ray is resected. It is a very good procedure if performed electively, with primary closure allowing adequate soft-tissue coverage of the amputation stump. It is associated to plantar extensor tenoplasty to avoid secondary equinus and conserve active motion in dorsiflexion [41]. It is indicated when osteitis lesions involve several rays, and especially when only two or three toes remain following iterative surgery for recurrent ulcer or necrosis (Fig. 3). The level of amputation depends on the septic lesions: skin incision is convex on the dorsal side, and the plantar flap needs to cover the entire resection area, as it constitutes a focus of pressure during walking and shoe-wearing (Fig. 8). Baumgardner’s variant procedure [42] consists in transmetatarsal amputation of one or more metatarsals, conserving the toes.

**Mid- and hind-foot**

Surgical treatment is more difficult in the mid- and hind-foot, as amputation beyond the tarso-metatarsal Lisfranc joint line is less functionally satisfactory. In such locations, therefore, management of diabetic osteitis is medicosurgical, with heavy antibiotic therapy prolonged for several months. Surgery is complementary.

**Lisfranc amputation.** Lisfranc amputation involves considerable loss of foot length, and creates tendon imbalance. It is important to conserve the peroneal tendon insertion (or to reinsert into the cuboid) and anterior tibial tendon. The 2nd metatarsal base, enclosed between the cuneiforms, should be conserved so as to conserve the proximal arc. At end of surgery, or secondarily in case of sepsis, posterior tendon lengthening is often required, in order to avoid equinus.

**Chopart’s midtarsal amputation.** Classically Chopart’s amputation results in secondary varus and equinus decompen-sation. When there is no relative ischemia, anterior tibial and peroneus brevis tenoplasty (by anchors or transosseous reinsertion between the talar head and greater calcaneal apophysis) is associated, with 2–3 cm resection of the Achilles tendon [43], to avoid secondary equinus. Tendon imbalance creates orthotic problems and cutaneous recurrence around the anterior amputation stump, preventing active dorsiflexion (Fig. 9).
Partial or total calcaneectomy. In case of loss of talar substance associated with calcaneal osteitis, partial or often total calcaneal resection [44], by posterior incision, is a salvage strategy. The soft-tissue gain following bone resection often allows primary closure. A talar compensation orthosis is then required.

Other amputations. Syme ankle disarticulation is complex, with a risk of instability of the plantar soft tissues of the distal tibio-fibular stump. Severe infection or ischemia contraindicate this procedure. Pirogoff-Boyd amputation has the advantage of conserving sufficient limb length to avoid the need for orthoses in everyday life.

“Acute” foot

“Acute foot” covers ulcer associated with signs of severe locoregional (PEDIS I3) and/or general infection (PEDIS I4). We usually avoid any emergency surgery, which leads to extensive debridement or amputation without skin closure. Directed cicatrization then often takes several weeks, with fragile stumps and risk of recurrence of ulcer.

Our experience of teamwork between endocrinologists and orthopedic surgeons enables us, almost systematically, including for patients presenting with septicemia and/or diabetic decompensation, to “cool” acute-foot lesions using parenteral empiric broad-spectrum bi- or tri-therapy [18,19,45]. We can recommend [46]: (amoxicilline—clavulanic acid) ± (aminoglycosides [gentamicin or netilmicin] or quinolones) in case of cellulitis; (piperacillin—tazobactam) + (teicoplanin [or vancomycin or linezolid]) + (quinolones) when the limb is threatened; and (imipenem [or ertapenem]) + (teicoplanin or vancomycin or linezolid) + (aminoglycosides) in case of septic shock. After 48—72 hours of this “drug wager”, locoregional and general
infection evolution is reassessed, and indications for abscess debridement or iterative amputation are considered. Emergency gadolinium-enhanced MRI is very useful in acute foot to diagnose deep soft-tissue effusion and extension into tendon sheaths so as to guide surgical drainage.

Such heavy antibiotherapy protocols, drawn up in coordination with infectologists, transform acute into subacute or chronic lesions so as to enable scheduled surgery within 1 or 2 weeks of treatment initiation. Meanwhile, assessment is completed, notably with advanced vascular evaluation by TcPO2 and arterial Doppler and, depending on the results, arteriography and/or angio-MRI. A revascularization procedure ahead of possible orthopedic surgery may be considered. In case of associated osteitis, scheduled orthopedic surgery observes the same principles as in case of ulcer, but with transmetatarsal amputation, as lesions are often more severe and skin necrosis may be associated with the initial cellulitis.

Figure 8 Transmetatarsal amputation. A: Case 1—M3 head osteitis with ulcer. B. Transmetatarsal amputation (post-op aspect and control X-ray). C. Case 2—Hallux gangrene (TcPO2 at 25 mm Hg). D. Amputation with primary closure. E. Case 3—amputation: post-op aspect and X-ray. F. Secondary plantar orthosis compensating forefoot.
Preventive surgery

Diabetes is a risk factor in orthopedic surgery. Diabetic neuropathy and/or arteriopathy were classically contraindications for foot surgery, due to the risk of infection, cicatrization disorder and necrosis. In diabetic neuropathy, however, static disorder and deformity, especially in the forefoot (hallux valgus, claw toe, etc.), increase the risk of ulcers which could lead to secondary amputation. "Preventive" foot surgery is therefore an issue in diabetes.

In practice, two situations are to be distinguished: static disorder of the foot in a diabetic patient without signs of "at-risk foot", and preventive procedures in at-risk diabetic feet with or without history of ulcer.

Elective surgery in diabetes without signs of "at-risk" foot

Surgery for foot stasis disorder requires complete preliminary assessment to rule out neuropathy and arteriopathy.
In the absence of sensorimotor lower limb neuropathy and given a relatively satisfactory vascular status, the risks entailed by foot surgery are not much greater than with non-diabetic patients. The techniques are those classically used with non-diabetic patients.

**Prophylactic surgery to limit ulcer risk**

In contrast, in case of sensorimotor lower limb neuropathy, orthopedic surgery is high-risk and complications may lead to the amputation meant to be prevented. Moreover, the resultant trauma may induce neurogenic osteoarthropathic lesions [47], although their frequency has not been determined. Onset of Charcot foot has also been described following revascularization surgery [48]. Given such Charcot foot complications, which we have found following forefoot surgery in diabetic subjects presenting with neuropathy, we now recommend "preventive" surgery to correct deformity only in case of history of ulcer facing bursitis and shoe-wear conflict. Indications should be well thought out, cautious and justified. This surgery can only be performed as part of a prospective study, with strict assessment of neuropathy and joint decision by a multidisciplinary team. Any history of ulcer increases the risk of infection (14% vs. 3–8%), although improvement in deformity is conserved over the long term [35].

Technically, in the forefoot, surgery consists in arthrodesis rather than conservative procedures, especially in correcting hallux valgus, so as to avoid possible recurrence requiring repeat surgery. Certain simple complementary procedures are available to prevent recurrence of forefoot ulcer: gastrocnemial lamina tenotomy (Strayer) or Achilles lengthening to reduce fixed equinus, flexor tenotomy for reducible claw toe, or interphalangeal arthroplastic resection [40]. Decompensation in abduction and medial Lisfranc flat foot may case mid-foot ulceration, and stabilization by arthodesis and plantar plate osteosynthesis can restore stability and avoid wound recurrence.

**Conclusion**

Diabetes is becoming pandemic. Diabetic foot lesions are a public health issue of growing importance. Management can only be multidisciplinary.

The orthopedic surgeon plays a central role in providing a biomechanical perspective so as, following conservative or surgical treatment, to avoid creating or leaving areas of hyperpressure that would induce recurrence of ulceration.

In case of ulceration, scheduled surgery is preferable to emergency intervention, even in "acute" foot; lesions should always be cooled by antibiotic therapy, even if this has to be empiric. This enables more limited surgery, with first-intention suture to achieve rapid healing and shorter antibiotic therapy courses.

Diabetic feet are high-risk neuropathic and vascular feet. Vascular assessment should always precede indication of orthopedic surgery. If vascular status is insufficient, prior revascularization is mandatory.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

**References**


