



THE DIABETIC FOOT

H Gill

DEFINITION

Defined as a group of syndromes in which neuropathy, ischemia and infection lead to tissue breakdown resulting in morbidity and amputation.

EPIDEMIOLOGY

The lifetime risk that a diabetic patient will acquire foot lesions (ulcers/gangrene) has been estimated at 15% to 25%, with an annual incidence of 1.0% to 4.1%. The incidence of these lesions appears similar in type 1 vs. type 2 diabetic patients, although type 2 diabetic patients comprise approximately 90% of the total diabetic population. Diabetics are 15X more likely to have an amputation than non-diabetics.

In >15% of these patients, ulcers will ultimately lead to amputation.

The risk for an initial foot ulcer is increased in patients who have had diabetes for >10 years, are male, have poor glycemic control, and already have other cardiovascular, renal, or retinal co morbidities

AETIOLOGY/PATHOPHYSIOLOGY

The aetiology and pathophysiology of diabetic foot ulcer usually has many components. Multicenter trial attributed 63% of diabetic foot ulcers to the critical triad of peripheral sensory neuropathy, trauma and deformity. Other risk factors include ischaemia; callus and oedema. The risk factors for ulceration are also predisposing factors for amputation.

NEUROPATHY

There are two theories

1. Vascular-decreased blood supply to the nerves leads to hypoxia of the nerve.
2. Metabolic-nerve tissue is unique as it does not require insulin for glucose

to be absorbed into the cells. Consequently the blood glucose levels within nerve axons will be directly proportional to that of blood. Glucose is then transformed into sorbitol and fructose which eventually leads to numerous biochemical and functional abnormalities. As the rate of polyol pathway is directly dependent on the level of glucose, persistent hyperglycemia feeds the process.

SENSORY NEUROPATHY

There is a symmetrical sensory loss in glove and stocking distribution. Patients can present with numbness, paresthesia or burning under the feet. Ulcers commonly occur with minor wounds that the patient is unaware of e.g. improper trimming of nails, thermal injuries (hot water bottles) and foreign bodies entering the foot.

MOTOR NEUROPATHY

With diabetics there is weakness and wasting of the intrinsic muscles of the foot, which in turn leads to imbalance between the long flexor tendons and the extensor tendons resulting in the flexors dominating. Ultimately the changes seen in the feet that of pes cavus (high arched foot) and clawing of the toes (hammer toes). With the clawing of the toes there is drawing of the fat pad normally under the metatarsal heads forward, which reduces the cushioning effect under the metatarsal head as a result there is increased pressure leading to calluses which in turn act as foreign bodies and resulting in ulceration with the clawing of the toes the toes become non weight bearing which further increases the pressure under the metatarsal heads and heels predisposing to ulceration.

AUTONOMIC NEUROPATHY

This leads to loss of sweating and changes in the normal microcirculation auto regulation. Loss of sweating leads to a dry foot that may cause fissures and cracks which predisposes to ulceration. With the loss of microcirculation auto regulation there is increased peripheral blood flow which leads to arterio-venous shunting which leads to ischaemia of the deeper tissues.

The shunts are about 20-70um in diameter and are under sympathetic control, with loss of tone the shunts open and blood bypasses the capillary bed.

ISCHAEMIA

The incidence of PVD in diabetics is 4X more than non-diabetics. In diabetics the occlusive disease involves primarily the tibial and peroneal vessels between the ankle and the knee. The distal vessels are usually spared.

Arterial disease in diabetics is due to atherosclerosis and medial sclerosis. Atherosclerosis is 20x more common than in non-diabetics. Diabetics have decreased ability to develop collateral circulation and as a result of neuropathy, claudication or rest pain is absent due to the neuropathy. The critically ischaemic foot may be relatively warm with little discoloration. Calcification of the tunica media produces a rigid tube as a result can falsely elevate arterial blood pressure. Medial sclerosis does not cause ischaemia.

OEDEMA

Is common in diabetics and is usually related to cardiac, renal or venous disease. It impairs cutaneous circulation and impairs healing. It predisposes to ulcer formation.

INFECTION

There is an increased risk of infection due to an impaired immune system.
Impaired humoral immunity
Impaired PMN function
Loss of protective skin barrier

CONNECTIVE TISSUE CHANGES

Due to enzymatic glycosylation of collagen, it leads to stiffening of the connective tissue surrounding the joint. As a result there is limited joint mobility which leads to

- Stiffness of the joints of the foot
- Abnormal gait

With continued hyperglycemia there is continued catabolism with a negative nitrogen balance. Synthesis of proteins such as fibroblasts and collagen is impaired resulting in poor wound healing.

DIABETIC CHARCOT'S OSTEO-ARTHROPATHY

It is a rare but serious complication. It was first described by Dr Jean Charcot in 1868.

It affects <1% of diabetics. It is usually unilateral, but 20% are bilateral.

It involves the following:

- Midtarsal joint 60%
- Metatarsophalangeal joint 30%
- Ankle joint 10%

PATHOPHYSIOLOGY

The exact aetiology of neuro-arthropathy remains ill-defined.

The two major theories are:

- Neurotraumatic: proposes decreased protective sensation allows cumulative mechanical trauma to result in fractures and joint destruction.
- In contrast the neurovascular theory proposes that a neurally initiated vascular reflex of autotomy leads to increased bone blood flow and active bone resorption by osteoclasts.

Four factors are essential for arthropathy to develop

- Peripheral neuropathy
- Unrecognized injury
- Increased local blood flow.
- Repetitive stress in injured structures

The exact interplay between these factors has not been determined but it is believed that the process starts with ligamentous failure and proceeds to subluxation or dislocation, with fractures or results in fixed deformity.

Usually presents with painless deformity of the foot which is hot swollen and erythematous. The differential to consider is gout, cellulitis and osteomyelitis.

The Charcot foot in the diabetic is progressive condition that is not confined to the bones but also affects all the tissues in the lower extremity. It is often confused with infection and osteomyelitis of the foot. Early identification and management to prevent amputation is essential.

RISK FACTORS FOR ULCERATION

General or systemic contributions:

- Uncontrolled hyperglycemia
- Duration of diabetes
- Peripheral vascular disease
- Blindness or visual loss
- Chronic renal disease
- Older age

Local issues

- Peripheral neuropathy
- Structural foot deformity
- Trauma and improper fitted shoes
- Callus
- History of prior ulcer or amputation
- Prolonged elevated pressure
- Limited joint mobility
- Previous ulcer and or amputation

ASSESSMENT OF THE DIABETIC FOOT

1. General assessment
2. Assess comorbid condition
 - CVS
 - Renal
 - Neurological
 - Eyes
 - Metabolic, water and electrolytes
3. Assessment of the foot.
 - Neuropathy
 - Ischaemia
 - Deformity
 - Callus
 - Swelling
 - Skin breakdown and ulcer evaluation
 - Infection
 - Necrosis

ASSESSMENT OF NEUROPATHY

Up to 35% of patients have asymptomatic neuropathy. The classical neuropathic foot is warm, dry, insensitive, dilated veins, good pulses, pes cavus, clawed toes and hyperkeratosis under the forefoot and heel.

1. Assess pain and touch with a pin and cotton wool
2. Ankle reflexes
3. Vibration sense with a tuning fork at 128HZ. Three readings are taken at the medial malleolus and pulp of the great toe. Inability to feel vibration at that frequency indicates peripheral neuropathy.
4. Nylon monofilament test
 - 10 point monofilament test used. Inability to feel more than 4 out of 10 points on the foot indicates presence of sensory neuropathy.

VASCULAR ASSESSMENT

The purely ischaemic foot will feel cool have absent pulses and trophic changes. If any of the peripheral pulses are felt significant ischaemia is

unlikely. If the pulses are absent or asymmetrical then further tests are required. Approximately 60% of non-healing ulcers are due to PAD.

NON INVASIVE VASCULAR TESTS

1. Measurement of ankle brachial index

- Normal is 0.9-1.1
- <0.45 is limb threatening
- Medial sclerosis can falsely elevate the result
- A dampened waveform with a normal ABI suggests calcified vessels and a falsely elevated ABI

2. Arterial Duplex: structural location of stenosis, occlusions and velocity and waveform of the blood travelling.

3. Toe pressure measurements

- This reflects blood flow more accurately in patients with diabetes.
- An absolute systolic measure <45mmHg is abnormal

4. Transcutaneous oxygen measurements

- A normal TcPO₂ is >55mmHg
- A TcPO₂ <30mmHg suggests that wound healing is less likely.
- It is an independent predictor of ulceration and wound healing.

INVASIVE VASCULAR TESTS

A carefully performed arteriogram must show the appropriate inflow source and outflow target artery and must incorporate the complete infrapopliteal circulation, including foot vessels.

FOOT DEFORMITY

- Skin cracks, fissures, calluses
- Claw toes. Hammer toes

- Rocker bottom feet
- Pes cavus
- Hallux valgus and hallux rigidis
- Charcot's foot
- Fixed flexion deformity of pip joints
- Flexion deformity of pip joints

Deformity usually leads to a bony prominence which results in high pressure and ulceration.

ASSESSMENT OF PLANTAR PRESSURE

Normally the metatarsal heads and heels are the greatest weight bearing area. In diabetics there is loss of protection of fat pads and reduced ankle joint dorsiflexion. This causes early lifting of the heels resulting in premature loading of the forefoot and increased duration of pressure.

These and other structural defects causes increase in pressure of forefoot during the static and contact phases of the gait. Peak pressures maybe several folds higher than non-diabetics. Foot pressure can be measured in the following ways:

1. Podotrack

- Footprint mat that quantifies plantar pressure by visual comparison between greyness of the foot print and a calibration card
- It is shown to identify 90% of high risk areas correctly

2. Optical pedograph

- This is a computerized pressure measurement device
- The system consists of a glass plate and is illuminated at its edge by strip lights and covered with a thin sheet of opaque reflective plastic
- >12.3kg/cm² are at risk of ulceration

INFECTIONS

Look for bacterial and fungal infections. Fissures or cracked skin

between the toes and heels can act as portal of entry for infection. Ulcers should also be probed to detect sinus tracts, dissection of the ulcer into tendon sheaths and bone or joints. A positive probe to bone has a high predictive value for osteomyelitis. The existence of odour and exudates should be noted and if exudates are present, they should be sent for MCS. Generally limb threatening infections can be defined by cellulitis extending beyond 2cm from the ulcer perimeter as well as a deep abscess, osteomyelitis or critical ischaemia. Polymicrobial infections usually predominate i.e.:

- gram positive cocci (Staphylococcus, Streptococcus, Enterococcus)
- gram negative (E.Coli, Pseudomonas, Klebsiella, Proteus)
- anaerobes. (Bacteroides, Clostridium perfringens, Peptostreptococcus)

The typical inflammatory signs of infection, including erythema, rubor, cellulitis, or tenderness, may be absent or diminished. Also frequently absent are the usual systemic manifestations of infection, including fever, tachycardia, or elevated white blood cell count. Unexplained hyperglycemia should prompt an aggressive search for a source of infection because the patient's elevated glucose level may be the only sign of impending problems.

Careful palpation of the foot for areas of tenderness or fluctuance is important to detect undrained abscesses in deeper tissue planes. All ulcers must be carefully inspected and probed and superficial eschars unroofed, to look for potential deep space abscesses

RADIOLOGICAL EVALUATION

Should be done to:

- Monitor bone and joint changes

- Detect subcutaneous air (non-clostridial)
- Detect foreign bodies
- Detect osteomyelitis

XR are not sensitive for acute infections of bone

- latent period of 10-14 days
- needs 50% of bones loss

If the XR is negative but there is strong suspicion of osteomyelitis then other modalities available to make the diagnosis.

- Bone scan-falsely positive due to hyperemia
- CT-scan can localize infection but cannot define proximal extent of sepsis

MANAGEMENT

A multidisciplinary approach must be taken for patients with diabetic feet due to multifaceted nature of the comorbidities that can occur in these patients. This approach has demonstrated significant improvement in outcomes including reduction in incidence of major amputations.

Treatment based on the Wagner classification

Grade 0: foot at risk, no ulceration

Grade 1: superficial diabetic ulcer, no infection

Grade 2: extension of ulcer to deeper tissue i.e. tendons, joints or fascia.

Grade 3: deep ulcer with abscess or osteomyelitis involving joints, bone, etc

Grade 4: localized gangrene (wet/dry) toe/foot

Grade 5: extensive gangrene

University of Texas Wound Classification System of Diabetic Foot Ulcers

Grade I-A: non-infected, non-ischemic superficial ulceration

Grade I-B: infected, non-ischemic superficial ulceration

Grade I-C: ischemic, non-infected superficial ulceration

Grade I-D: ischemic and infected superficial ulceration

Foot at risk:

Identify patients at risk i.e. look at risk factors. Patients with foot at risk are classed into the following categories

Category 0: no sensory neuropathy-once a year follow up

Category 1: sensory neuropathy – twice a year follow up

Category 2: signs of neuropathy and PAD and or foot deformities-once every 3 months

Category 3: previous ulcers-once a month

ESTABLISHED ULCER

The primary goal in treating ulcers is to obtain wound closure. Management of the foot is determined by

- Severity(grade)
- Vascularity
- Infection

Rest and elevation should be instituted at the first presentation.

Relief of pressure by “off-loading”:

- bed rest
- crutches
- wheelchair maybe recommended to totally off load pressure from the foot
- TCC (total contact cast) is considered the optimal management of neuropathic ulcers. Problem-must be reapplied weekly and requires considerable experience to avoid iatrogenic lesions.
- Acceptable alternatives are removable braces and half shoes

Mainstay of treatment is debridement of all necrotic tissue, callus and fibrous tissue

Unhealthy tissue must be debrided back to bleeding tissue to allow full visualization of the extent of the ulcer

and detect underlying abscesses or sinuses

NEUROPATHIC ULCER

The first step in the treatment of any neuropathic ulcer is restriction of weight bearing of the involved extremity. Patients with limb-threatening foot infections and noncompliant patients will require hospitalization and bed rest, followed by evaluation and management of arterial ischemia.

Uncomplicated neuropathic ulcers will often heal with topical therapy and non-weight bearing, and a trial of outpatient care is warranted. Topical dressings should be aimed at maintaining a moist environment with saline impregnated gauze, topical antibiotic ointments, or other similar agents. The ulcer should be protected from excessive pressure by placing of an accommodative pad around the lesion to distribute pressure to surrounding tissues.

Heavy callus around the edges of the lesion should be trimmed away to reduce peak plantar pressure, and shoes should be replaced with a stiff-soled “healing sandal.”

Custom-molded orthotics and extra-depth shoes, running shoes, or custom-molded shoes in the case of severe foot deformity, are also prescribed to prevent future recurrence.

Hyperbaric oxygen therapy facilitates wound healing for diabetic foot ulcers and thus lower amputation rates by promoting wound healing through anti-edema, antibacterial, and neovascularization effects.

INFECTIONS

In the absence of deep infection or necrosis, minor infections or ulcers may be managed conservatively with local wound care, antibiotics, or both.

In patients with salvageable ischemic foot lesions and concomitant active infection, the infection needs to be

controlled before vascular surgical intervention.

In addition to instituting broad-spectrum antibiotics, options include open debridement and drainage or partial foot amputation. A short delay (usually <5 days) before revascularization to control active infection is justified; however, longer waits to “sterilize wounds” is inappropriate and may result in further necrosis and a lost opportunity to save the foot.

Patients with limb-threatening infections (Abscess, Cellulitis, Osteomyelitis, Gangrene) require immediate hospitalization, immobilization, and intravenous antibiotics. Cultures from the depths of the ulcer should be sent; wound swabs are unreliable and should not be performed.

Empiric broad-spectrum antibiotic therapy should be initiated to cover the poly-microbial infections usually seen in diabetic patients. Empiric antibiotic regimens are dictated by institutional preferences, local resistance patterns, availability, and cost.

- Penicillin + Gentamycin + Metronidazole.
- Augmentin
- Clindamycin+ Ciprofloxacin

Patients with abscess formation or necrotizing fasciitis must undergo prompt incision, drainage, and debridement, including partial open toe, ray, or forefoot amputation.

Wounds should be packed open with saline-moistened gauze, and dressings should be changed two to three times a day. Wounds should be examined daily, and additional bedside or operative debridement should be repeated as needed.

Adequate dependent drainage is crucial, and limited incisions with closed-suction or Penrose drains should be avoided.

Underlying osteomyelitis requires

- bony resection
- antibiotics (culture driven)

- amputation

VASCULARITY

Each operation must be individualized according to the patient’s available venous conduit and arterial anatomy. Options include open surgical bypass or endovascular intervention. Treatment of underlying ischaemia is critical to a successful outcome regardless of topical therapies. Vascular consultation should be sought when the patient presents with

- ischaemic wound
- non healing ulcer despite appropriate management

A major component of limb salvage surgery in these patients is to restore pulsatile flow to the foot.

MEDICAL TREATMENTS

Primary prevention involves aggressive glycemic control (goal hemoglobin A1C <6.5% to 7.0%); management of associated risk factors such as smoking, hypertension, hyperlipidemia, and obesity; periodic physical examinations, including a vascular examination; and probably most important, proper foot care and hygiene strategies.

- Zero smoking
- Physical activity of 180 minutes/ week
- BP < 140/90 mm Hg
- Cholesterol: Total < 5.0mmol/l
LDL

cholesterol < 2.2 mmol / l

HDL

cholesterol >1.0mmol/l fasting TG < 2.0 mmol/l

- HbA1c < 6.5% in diabetics
- BMI < 25

THE MANAGEMENT GOALS OF CHARCOT FOOT

Aim is to offload the affected extremity, prevent further collapse and deformity, and protect the opposite foot. The first step of treatment is an extended period of non-weight bearing

and castor splint immobilization to promote eventual healing of the joint. The use of accommodative footwear is essential to long-term management. Surgery is rarely indicated, and a stabilizing procedure is done most safely after the disease has reached a quiescent stage. Amputation is reserved for those rare patients with severe uncorrectable deformities, those with chronic ulcers plagued by such extensive osteomyelitis that the foot is unsalvageable, or after failed open reconstructions.

AMPUTATIONS

Certain patients may not be appropriate candidates for arterial reconstruction because of their overall health status.

Elderly patients with severe dementia who are non-ambulatory or bedridden, or who have severe flexion contractures of the knee or hip, have no prospect of rehabilitation and are inappropriate candidates for traditional vascular procedures and will require primary amputation.

Patients with terminal cancer with a very short life expectancy or similar lethal co-morbidities do poorly with open revascularization and are probably better served by endovascular intervention or primary amputation.

Patients with an unsalvageable foot due to extensive necrosis from ischemia or infection also require primary amputation.

Natural history of amputations:

30 day post op mortality:

- AKA 13-16%, BKA 6-9 %
- Up to 50%

Survival (3 years):

- AKA 39% BKA 57%

Rehabilitation

- BKA : 60%, AKA : 40%

OFF LOADING

A final aspect of managing diabetic foot ulcers is offloading to decrease pressure on the extremity. Offloading strategies involve combinations of bed rest, crutches or wheelchairs, casting, foams or padding, and healing shoes or walking boots.

Only after wound healing has been achieved should weight bearing be reinstated back to baseline levels, and consultation with a physical therapist should be obtained when necessary.

The last alternative remains amputation. Closed minor toe or transmetatarsal amputations are practical after infection control and revascularization and typically leave the patient with a functional foot for walking.

In situations involving extensive tissue loss precluding a functional foot, when there are non-healing wounds in the setting of patent grafts and for control of sepsis, amputation below the knee is necessary.

PREVENTION

Foot examination should be performed in diabetic patients at least once a year and more frequently in those patients at high risk of foot ulceration. Identification of patients at risk is the most important aspect of amputation prevention. Education is an integral part of prevention, should be simple and repetitive. Education should be targeted at both healthcare providers and patients. Several studies have shown that foot care programs including education, regular examinations and risk categorization can reduce the occurrence of foot lesions in up to 50% of patients.

PATIENT EDUCATION

DO's:

- wash feet daily, dry well, inspect
- check hidden areas carefully
- anti-fungal powder

- careful nail hygiene
- early treatment of wounds
- wear comfortable, well fitted shoes
- natural fibre socks are best

DON'T's:

- walk barefoot EVER
- wear new shoes without "breaking in"
- leave wounds untreated
- burn your feet
- cut nails too short
- ignore discomfort

INDICATIONS FOR REFERRAL:

ACUTE:

- Callus formation
- Ulceration
- Ischaemic change
- Acute local sepsis
- Non-healing trauma

CHRONIC:

- recurrent callus / ulceration
- worsening deformity
- worsening neuropathy
- deteriorating sugar control
- onset of ischaemic symptoms



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