IN THE NAME OF GOD
THE DIABETIC FOOT

- Ali akbar Beigi M.D.
  General and Vascular
  Surgeon  Taleghani
  Hospital Shahid Beheshti
  U.M.S
Diabetic Foot

Definition:
Infection, ulceration or destruction of deep tissues associated with neurological abnormalities & various degrees of peripheral vascular diseases in the lower limb

(based on WHO definition)
Epidemiology

- 15% is the prevalence of foot ulcer in diabetics in their lifetime.
- 40% - 60% of all non-traumatic amputation in lower limb.
- 85% of diabetic-related foot amputation are preceded by foot ulcer.
- 4 out of 5 ulcers in diabetics are precipitated by trauma.
Epidemiology & Preventive care

Shoe related Trauma 36%
Accidental cut or Puncture 8%
Thermal injury 8%
Decubitus ulcer 8%
Pathophysiology of Foot Ulceration

- Neuropathic
- Ischemic
- Neuro–ischemic
- Immunologic Defects
### Factors associated with foot ulcer

<table>
<thead>
<tr>
<th>Factor</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous ulcer/amputation</td>
<td></td>
</tr>
<tr>
<td>Neuropathy</td>
<td>Sensorimotor</td>
</tr>
<tr>
<td>Trauma</td>
<td>Poor footwear</td>
</tr>
<tr>
<td></td>
<td>Walking barefoot</td>
</tr>
<tr>
<td></td>
<td>Falls/accidents</td>
</tr>
<tr>
<td></td>
<td>Objects inside shoes</td>
</tr>
<tr>
<td>Biomechanics</td>
<td>Limited joint mobility</td>
</tr>
<tr>
<td></td>
<td>Bony prominences</td>
</tr>
<tr>
<td></td>
<td>Foot deformity/osteoarthropathy</td>
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<td></td>
<td>Callus</td>
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<tr>
<td>Peripheral vascular disease</td>
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<td>Socio-economic status</td>
<td>Low social position</td>
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<td>Poor access to healthcare</td>
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<td>Non-compliance/neglect</td>
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<td>Poor education</td>
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</table>
Hammer toe

Claw toe
Biomechanics of foot wear AREAS AT RISK OF ULCERATION
STAGES OF ULCER DEVELOPMENT

1. Callus formation

2. Subcutaneous hemorrhage

Fig 1. Illustration of ulcer due to repetitive stress
STAGES OF ULCER DEVELOPMENT

3. Breakdown of skin

4. Deep foot infection with osteomyelitis
Diabetic Neuropathy

- History & careful foot examination are mandatory to diagnose neuropathy
- Up to 50% of type 2 diabetic patients have significant neuropathy & at risk of foot ulcer
- Sensorimotor & peripheral sympathetic neuropathy are major risk factors for ulcer
FOOTWEAR
Neuropathic Ulcer
Decubitus Ulcer
Periphral vascular disease & diabetic PVD

- **PVD** is the most important factors related to outcome of diabetic foot ulcer
- PVD is diagnosed by simple clinical examination
- Symptoms of ischemia may be masked by neuropathy
- Microangiopathy shouldn't be accepted as primary cause of ulcer
- Non invasive vascular test determines probability of healing Ankle Brachial index & Trans cutaneous oxigenometry
- Outcome of revascularization is similar to that in non-diabetic
Pathways to diabetic foot ulceration
Examination

- Neurological examination
  - Vibration perception – tuning fork at 128 Hz
  - Light pressure - Simmes – Weinstein 10 gram monofilament
  - Light touch
  - Two point discrimination
  - Pain
  - Temperature perception
  - Deep tendon reflexes
  - Clonus
  - Babinski test
  - Romberg test

- Vascular Examination
  - Palpation of pulses
  - Skin/limb colour changes
  - Presence of edema
  - Temperature gradient
  - Skin changes
    - Abnormal wrinkling
    - Absence of hair
    - Onychodystrophy
  - Venous filling time
Clinical features of the ischaemic foot

- Cold
- Pale colour
- Glass like skin
- Little callous
- Pulse less
- Dependent rubor
- Claudication
- Rest pain
- Ulcers on edges

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Ischemic Foot
Preventive care

Biomechanics of foot wear

- Biomechanical abnormalities are consequence of neuropathy, they lead to abnormal foot pressure.
- Foot deformity & neuropathy increase the risk of ulcer.
- Pressure relief is essential for ulcer healing and/or prevention.
- Frequent inspection of shoes & insoles is mandatory.
- Appropriate foot wear significantly reduce ulcer recurrence.
Preventive care

<table>
<thead>
<tr>
<th>Patient educating self examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family Involvement in serial Exam.</td>
</tr>
<tr>
<td>High risk group Detection</td>
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</tbody>
</table>
Infection in diabetic foot is limb threatening

- Signs of infection may be absent in diabetic pt. with foot ulcer
- Superficial infection is usually caused by gram +ve cocci, deep infection is poly microbial
Neuro-osteoarthropathy (sharcot joint)

- Non-infective pathology
- Should be suspected in any swollen hot erythematous foot
- Differentiation from infection is important to prevent misdiagnosis & possible amputation
- Treatment should aim at preventing severe deformity
Charcout Joint
NEURO-OSTEOARTHRITIS (CHARCOT FOOT)
NEURO-OSTEOARTHRTHROPATHY (CHARCOT FOOT) Neuro-osteoarthropathy
Clinical assessment

- Initial Traumatic Event
- Duration of ulcer
- Method of treatment
- Clinical progressing of wound & symptom & sign of toxicity
- Vascular insufficiency history
Clinical assessment

- Physical Exam.
- Probing to bone stands 90% diag. for osteomyelitis
- Vascular Exam. Ankle & toe pressure ltranscutaneous oxigenometrylpulse volum recordung
- X ray imaging
- Bone scan & MRI
Plain X-rays
- Osteomyelitis,
- fractures
- Soft tissue gas
- Dislocations in neuropathic arthropathy
Imaging

Technetium & Tlum bone scans – (osteomyelitis)

MRI - 9osteomyelitis)
Concept of ABI

The systolic blood pressure in the leg should be approximately the same as the systolic blood pressure in the arm.

Therefore, the ratio of systolic blood pressure in the leg vs the arm should be approximately 1 or slightly higher.

ABI has been found to be 95% sensitive and 99% specific for angiographically diagnosed PAD.

Measuring the Ankle-Brachial Index (ABI)
Step 1: Gather Equipment Needed

Equipment needed:

1. Blood Pressure Cuff
2. Hand-held 5-10 MHz Doppler probe
3. Ultrasound Gel

Stabilizing hand
Calculating the ABI

Example Calculation

Right Leg ABI

\[
\frac{60 \text{ mm Hg}}{120 \text{ mm Hg}} = 0.50
\]

Left Leg ABI

\[
\frac{66 \text{ mm Hg}}{120 \text{ mm Hg}} = 0.55
\]

ABI Interpretation

\[\leq 0.90\text{ is diagnostic of peripheral arterial disease}\]

An Auto ABI Device

Systolic Pressures and ABI

PVR Waveforms
Toe Pressures

Doppler or Photoplethysmography (PPG)

- Toe/brachial pressure > 0.7 = normal
- Rest pain usually present in patients with index < 0.15
- Absolute pressure in the toes of 20-30mmHg is usually associated with rest pain
Assessment of Diabetic Foot

- Neuropathy
- Ischemia
- Deformity
- Callus
- Swelling
- Skin breakdown
- Infection
- Necrosis
# Staging of Diabetic Foot

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>High risk</td>
</tr>
<tr>
<td>3</td>
<td>Ulcerated</td>
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<tr>
<td>4</td>
<td>Cellulitic</td>
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<tr>
<td>5</td>
<td>Necrotic</td>
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<tr>
<td>6</td>
<td>Major amputation</td>
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</tbody>
</table>
Diabetic Foot Ulcer Treatment

Modalities

- Microbiological control
- Wound control
- Vascular control
- Mechanical control
- Metabolic control
- Educational control
Diabetic Foot Ulcer Treatment

- Multidisciplinary approach
- Staging dictate the treatment option
- Continuity of care & life long observation
Diabetic Foot Ulcer Treatment

- Rigid shoeing System for repeated local trauma ulcer I total contact Cast 90% healing in non vascular ulcer
- Off loading of ulcer/Half shoes/felted foam plantar dressing
- Non weight bearing ambulation/bed rest
FOOT WEAR
Diabetic Foot Ulcer Treatment

- Debridement (chemical/enzymatic/surgical)
- Dressing (Novin/vacuum therapy)
- Herbal and oriental medicine
- Minor Amputation and Wound closure
Amputation in diabetic PatientD

- Minor/major amputation increased the no. of deformed feet

- Minor amputation is needed:
  * Gangrene as part of debridement

- Minor amputation doesn’t significantly compromise walking ability
Diabetic Foot Infection

- **Surgical debridment is essential in acute deep infection**
- **Osteomyelitis** (diagnoses & treatment)
OSTEOMYELITIS
Major Amputation

- Risk of loss walking ability
- Mortality
- Risk of contra-lateral amputation
- Strict indication
- Careful choice of the level
Revascularization

- Surgical by pass (Vein/prosthetic graft)
- Angioplasty
Angioplasty
Revascularization in PERIPHERAL VASCULAR DISEASE
Adjuvant therapy

- Bioengineered skin substitutes/
- Biologic graft (Amnion membrane)
- Platelet derivated growth factor
- Hyper baric oxygen therapy
- Vacuum therapy
- Ozone therapy
How To Prevent Foot Problems

5 corner stones

- Regular inspection & examination of foot & foot wear
- Identification of high risk patient
- Education of patient, family & health care providers
- Appropriate foot wear
- Treatment of non ulcerative pathology
Diabetic Neuropathy
Diabetic foot

Introduction

- Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.
- The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels.
- Diabetic foot is defined as any foot pathology that results directly from diabetes or its long term complications
- Two types of diabetes: type I and type II diabetes
Epidemiology

- Lesions of the feet affect approx 15% of diabetics in their life with an amputation rate 15 fold higher than non diabetics
- Foot ulcerations are the commonest cause of hospital admission in diabetics
- Atherosclerosis rarely seen in type I diabetics < 40 yrs while it may be present even before diagnosis in type II
- A study conducted showed that diabetes related gangrene was the indication in 17.5% of lower limb amputations while PVD accounted for 55.3%
Epidemiology – risk factors

- Male sex
- DM > 10 years duration
- Peripheral neuropathy
- Abnormal foot structure
- Peripheral arterial disease
- Smoking
- H/O previous ulceration / amputation
- Poor glycemic control (HbA1c > 7%)
Pathophysiology

Factors leading to development of diabetic foot:

- Diabetic macroangiopathy – peripheral arterial occlusive disease
- Diabetic microangiopathy – thickening of basement membranes
- Diabetic polyneuropathy
- Diabetic osteoarthropathy – abnormal foot biomechanics
- Reduced resistance to infection
- Delayed wound healing
- Reduced rate of collateral vessel formation
Diabetic angiopathy

- Diabetic macroangiopathy is histologically similar to non diabetic atherosclerosis but distributed in the distal segments of the lower extremities (calf and foot arteries)
- Arterial calcification readily detectable on plain x ray with constriction noted on angiography. This compromises oxygen supply to the periphery
- Gas exchange is compromised by marked thickening of the capillary basement membrane – a feature of diabetic microangiopathy
Diabetic neuropathy

- This affects the sensory, motor, and autonomic fibers
- Sensory neuropathy - deep sensory perception is reduced resulting in loss of protective reflexes against physical injury. Typically, manifests in a sock-like distribution.
- Motor neuropathy – denervation and atrophy of small foot muscles leading to malum perforans, transverse foot arch instability with clawing and splay foot
- Autonomic neuropathy – vasodilation and absent sweating thus foot is warm, dry, scaly which predisposes to fissure formation
Hallux valgus, hammer toes, erythema over pressure points
Callus formation at pressure points and dry skin are substrate for ulceration.
Pes cavus resulting in callus formation over the pressure points
Diabetic neuropathic osteoarthropathy (DNOAP)

- Destruction of peripheral and autonomic nerves leads to vasodilation and subsequent demineralization and destabilization of foot skeleton
- Sander’s classification based on the location of the lesions in the foot
  - DNOAP I – necrosis of metatarsophalangeal joints with eventual malum perforans, osteolysis and candystick deformities
  - DNOAP II – necrosis of the tarsometatarsal joints (Lisfranc’s joint) resulting in a destabilized backfoot. Subluxation of the navicular leads to a clubfoot with abduction of the forefoot and rocking foot deformity. Exposure of the cuneiform-naviculare joint may lead to ulceration at this location
Central malum perforans – MT I and IV
Osteolysis MT II
Candystick deformity MT III
DNOAP II

Ulceration over the navicular-cuneiform joint
Destruction of lisfranc articulations
Fallen medial arch with navicular subluxation
DNOAP

- DNOAP III – necrosis of Chopart’s joint: talo-navicular articulation. Leads to rocking foot deformity where the middle of the sole becomes exposed to pressure. Ulceration occurs directly beneath the verticalized talus. There is as well broadening of the backfoot, abduction of the forefoot and talonavicular subluxation.

- DNOAP IV – necrosis of the tibiotalar joint.

- DNOAP V – necrosis of the talocalcaneal resulting in a clump backfoot with the bayonet type of deformity.
DNOAP III

Ulceration over the navicular
Verticalisation of the talus
Navicular subluxation
Destruction of talonavicular articulation
DNOAP IV

Destruction of tibiotalar joint

Lateral malleolar prominence at risk of ulceration
Bayonet deformity
Lateral skin ulceration risk
Destruction of talo-calcaneal joint
Increased infection rate

- Skin fissurations predisposes to penetration of infectious microbes
- Polymorphonuclear granulocyte chemotaxis and phagocytosis is impaired
- Polyneuropathy predisposes to deep seated infections due to impaired pain sensation
- Both anaerobe and aerobe infections are implicated in diabetic foot infections
# Patient evaluation

<table>
<thead>
<tr>
<th>Global History</th>
<th>Foot Specific History</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes - duration</td>
<td>General</td>
</tr>
<tr>
<td>Glycemic management/control</td>
<td>Daily activities, including work</td>
</tr>
<tr>
<td>Cardiovascular, renal and ophthalmic evaluations</td>
<td>Footwear</td>
</tr>
<tr>
<td>Other comorbidities</td>
<td>Chemical exposures</td>
</tr>
<tr>
<td>Treating physicians</td>
<td>Callus formation</td>
</tr>
<tr>
<td>Nutritional status</td>
<td>Foot deformities</td>
</tr>
<tr>
<td>Social habits: alcohol, tobacco, drugs</td>
<td>Previous foot infections, surgery</td>
</tr>
<tr>
<td>Current medications</td>
<td>Neuropathic symptoms</td>
</tr>
<tr>
<td>Allergies</td>
<td>Claudication or rest pain</td>
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<tr>
<td>Previous hospitalizations/surgery</td>
<td>Wound / Ulcer History</td>
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<tr>
<td></td>
<td>Location</td>
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<td></td>
<td>Duration</td>
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<td></td>
<td>Inciting event or trauma</td>
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<td></td>
<td>Recurrence</td>
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<tr>
<td></td>
<td>Infection</td>
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<tr>
<td></td>
<td>Hospitalization</td>
</tr>
<tr>
<td></td>
<td>Wound care</td>
</tr>
<tr>
<td></td>
<td>Off-loading techniques</td>
</tr>
<tr>
<td></td>
<td>Wound response</td>
</tr>
<tr>
<td></td>
<td>Patient compliance</td>
</tr>
<tr>
<td></td>
<td>Interference with wound care (Family or social problems for patient)</td>
</tr>
<tr>
<td></td>
<td>Previous foot trauma or surgery</td>
</tr>
<tr>
<td></td>
<td>Presence of edema - unilateral vs bilateral</td>
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<tr>
<td></td>
<td>Charcot foot - previous or active</td>
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<tr>
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<td>Charcot treatment</td>
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</tbody>
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Examination

- Dermatological
  - Skin appearance
  - Calluses
  - Fissures
  - Nail appearance
  - Hair growth
  - Ulceration/infection/gangrene
  - Interdigital lesions
  - Tinea pedis
  - Markers of diabetes

- Musculoskeletal
  - Biomechanical abnormalities
  - Structural deformities
  - Prior amputation
  - Restricted joint mobility
  - Tendo Achilles contractures
  - Gait evaluation
  - Muscle group strength testing
  - Plantar pressure assessment
Classification - Wagner

- Grade 0 - Skin intact, no foot deformity
- Grade 1 - Superficial ulcer
- Grade 2 - Deep ulcer
- Grade 3 - Deep ulcer with infection
- Grade 4 - Limited necrosis
- Grade 5 - Necrosis of the entire foot
Wagner grade 0
Wagner grade 1
Wagner grade 2
Wagner grade 3
Wagner grade 4
Wagner grade 5
University of Texas grading

- Based on wound ulcer depth and vascular status

  **Horizontal component:**
  - Stage A – clean wounds
  - Stage B – non-ischemic infected
  - Stage C – ischemic non-infected
  - Stage D – ischemic, infected

  **Vertical component:**
  - Grade 0 – pre- or postulcerative site that has healed
  - Grade 1 – superficial wound not involving tendon, capsule or bone
  - Grade 2 – wound penetrating to tendon or capsule
  - Grade 3 – wound penetrating bone or joint
Laboratory evaluation

- FBS/RBS
- Glycosylated hemoglobin (HbA1C)
- FHG + ESR
- Wound and Blood cultures
- Serum Chemistry: CRP
- Urinalysis
Diabetic foot infection

- Divided into uncomplicated non limb threatening infection - superficial cellulitis of limited extension that can be treated on an outpatient basis
- Complicated limb threatening infections are more extended and penetrate to deeper tissues, such as tendons, joint capsules, bone or articulations. They require inpatient treatment with surgical debridement and intravenous antibiotics
- Osteomyelitis has therapeutic implications such as prolonged antibiotic courses and need for resections
Diabetic foot infection

- Superficial swabs overestimate the number of likely microorganisms therefore a deep tissue specimen is preferred as it is more representative.
- Aerobic gram +ve cocci most common infecting organisms: *S. aureus* and *β-hemolytic streptococci* (especially group B).
- Chronic wounds have more complex flora: enterococci, enterobacteriaciae, obligate anaerobes, *P. aeuroginosa* and other non-fermentative gram negative rods.
Management

- Preventative foot care
- Diabetic foot ulcer (DFU) care
- Ischemia management
- Neuropathy management
- Surgery
Preventative foot care

- Podiatry - Regular inspection of the foot, appropriate nail care, warm (32°C) soaks, moisturizing creams, early detection of new lesions
- Optimally fitted footwear – well cushioned sneakers, custom molded shoes
- Pressure reduction – cushioned insoles, custom orthoses
- Patient education — need for daily inspection and necessity for early intervention, avoidance of barefoot walking
- Physician education — significance of foot lesions, importance of regular foot examination, and current concepts of diabetic foot management
DFU care

- Debridement – of callus and necrotic tissue using sharp debridement till bleeding tissue, lavage and dressings
- Offloading of the ulcer site to reduce ischaemia via total contact cast, non weight bearing (crutches, bedrest, wheelchair)
- Wound management – maintenance of a moist wound with regular cleaning and dressing
- Infections treated with broad spectrum antibiotics based on culture results. Clindamycin/fluoroquinolone/metronidazole suitable empiric therapy
Ischemia/neuropathy

- Angiography evaluates for chance of catheter intervention or vascular surgery
- Vascular bypass surgery successful if occlusion is supramalleolar but less so in inframalleolar PAOD
- Aspirin is useful for primary and secondary prevention
- Neuropathy treated pharmacologically with agents such as carbamazepine, gabapentin and pregabalin and prevention of minor trauma that will go undetected due to insensate foot
Surgery

- Sharp debridement
- Local procedures to remove areas of chronically elevated pressure (deformities) causing non healing ulcers
- Sequestrectomies
- Amputation
- Correct structural deformities — hammer toes, bunions, Charcot
Indications for amputation

- Uncontrollable infection or sepsis
- Inability to obtain a plantar grade, dry foot that can tolerate weight bearing
- Non ambulatory patient
Other peripheral vascular diseases

- Peripheral arterial occlusive disease (PAOD)
- Post thrombotic syndrome
- Chronic venous insufficiency
PAOD

- Most common cause is atheroclerosis which narrows the lumen of peripheral arteries
- Buerger’s disease is a potentially preventable cause due to its association with smoking
- Symptoms include:
  - Intermittent claudication
  - Ischaemic rest pain
- Signs include:
  - Calf muscles atrophy
  - Loss of hair growth over the dorsum of the toes
  - Thickening of the toenails
  - Atrophy of the skin
  - Delayed capillary refill
  - Ischaemic ulcers
Ischemic ulcers are painful with a ‘punched out’ appearance.

They are commonly located distally over the dorsum of the foot or toes.

The ulcer base usually consists of poorly developed, grayish granulation tissue.

Critical limb ischaemia is defined as persistent ischemic rest pain lasting for more than 2 weeks and/or ulceration of the leg, associated with an ankle systolic pressure < 50 mm Hg and/or a toe systolic pressure of < 30 mm Hg and/or an ABPI < 0.9.
PAOD - Investigations

- Ankle systolic pressure measurement – 12cm cuff used and doppler probe over the dorsalis pedis or posterior tibial artery. <50mmHg implies critical limb ischemia and aggressive revascularisation needed
- Toe systolic pressure – 25mm cuff over proximal phalanx of hallux. Critical limb ischemia at <30mmHg
- Transcutaneous oxygen pressure – electronic probe used. Normal range 30 – 50 mmHg. <30 mmHg implies critical ischaemia
- Doppler ultrasound – operator dependent. More accurate for assessment of femoropopliteal vessels than tibioperoneal arteries
- Arteriography – gold standard, invasive, contrast used. Useful where vascular procedures are being planned
Management of PAOD

- Secondary prevention – statins, aspirin, DM and HTN control, smoking cessation
- Walking exercises 1h/day– reduced intermittent claudication by encouraging collateral vessel formation
- Footwear fitted to reduce pressure and increased warmth. May need to be customised. Minimise exposure to cold and moisture
- Surgical debridement of ulcers with appropriate dressing thereafter, infection control
- Interventional vascular procedures such as percutaneous transluminal angioplasty, bypass procedures indicated in critical limb ischaemia
- Amputation may eventually be necessary
Post thrombotic syndrome

- Symptoms and signs that typically follow DVT
- Caused by post thrombotic recanalisation and valve destruction that leads to chronic ambulatory venous hypertension
- Not limb threatening but adversely affects quality of life
- Symptoms include chronic leg discomfort
- Signs include:
  - Edema
  - Skin changes – pigmentation, dermatitis, liposclerosis
  - Ulcers – typically supramalleolar medial, painless, irregular edges
Management

- Primary prevention by preventing DVT in at risk patients
- Early management of DVT and continued antithrombotic therapy to prevent recurrence of DVT
- Fibrinolysis and thrombectomy – controversial due to minimal benefit and increased risk
- Compression therapy – effective primary prevention of DVT and secondary prevention of PTS after DVT. Layered compression stockings now the mainstay of treatment
- Vascular surgery – not as useful as in varicose veins because PTS is a disease of the deep system
Charcot Foot

- **Neurotraumatic**
  - Decreased sensation + repetitive trauma = joint and bone collapse

- **Neurovascular**
  - Increased blood flow $\rightarrow$ increased osteoclast activity $\rightarrow$ osteopenia $\rightarrow$ Bony collapse
  - Glycolization of ligaments $\rightarrow$ brittle and fail $\rightarrow$ Joint collapse
Classification

- Eichenholtz
  - 1 – acute inflammatory process
    - Often mistaken for infection
  - 2 – coalescing phase
  - 3 - consolidation
Classification

- Location
  - Forefoot, midfoot (most common), hindfoot
- Atrophic or hypertrophic
  - Radiographic finding
  - Little treatment implication
X-ray, physical examination

- Ulceration
  - Pressure relief of ulcer with total-contact cast
  - Exostectomy, debridement, IV antibiotics

- Intact soft tissue
  - Ulceration healed
  - Stage of Eichenholtz at time of Rx

Stage I (Acute inflammation, disintegration)
- Fracture and dislocation
  - Selected cases
  - Serial total-contact casts
  - Reduction and arthrodesis
    - Amputation and prosthesis
    - Severe, uncontrollable deformity

Stage II (Bony coalescence)
- Ankle-foot orthosis (AFO) (removable brace)
  - Progressive weight bearing
  - Decreased swelling, warmth, early new bone formation

Stage III (Bony healing)
- Accommodative footwear and molded insoles and some require indefinite use of AFO
  - Gradual reduction in swelling with minimal fluctuation
Case 1
Case 1
Case 2
Case 3
Case 3
Case 4
Charcot Foot

- Neurotraumatic
  - Decreased sensation + repetitive trauma = joint and bone collapse

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Pressure relief of ulcer with total-contact cast

Ulceration

Exostectomy, debridement, IV antibiotics

Intact soft tissue

Ulceration healed

Stage of Eichenholtz at time of Rx

Stage I
(Acute inflammation, disintegration)

Fracture and dislocation

Severe, uncontrollable deformity

Selected cases

Reduction and arthrodesis

Serial total-contact casts

Progressive weight bearing

Amputation and prosthesis

Decreased swelling, warmth, early new bone formation

Stage II
(Bony coalescence)

Ankle-foot orthosis (AFO) (removable brace)

Gradual reduction in swelling with minimal fluctuation

Stage III
(Bony healing)

Accommodative footwear and molded insoles and some require indefinite use of AFO
Case 1
Case 1
Case 1
Case 2
Case 3
Case 3
Case 4
Case 4
Indications for Amputation

- Uncontrollable infection or sepsis
- Inability to obtain a plantar grade, dry foot that can tolerate weight bearing
- Non-ambulatory patient
- Decision not always straightforward
DIFFERENTIATION OF THE FOOT

HEALTHY FOOT

• **Nerves** let you feel pain, vibration, pressure, heat, and cold.

• **Blood Vessels** Carry nutrients and oxygen to your feet to nourish them and help them heal from injuries.

• **Bones** give your foot shape and help distribute the pressure from your body's weight.

• **Joints** are the connections between your bones. They help absorb pressure and allow your foot to move. Your *arch* is a group of joints that provides stability for your entire foot.

DIABETIC FOOT

• **Damaged Nerves** → difficult to feel pain, pressure, heat, and cold.

• **Blocked Blood Vessels** bring fewer nutrients and oxygen to feet → sores may not be able to heal.

• **Weakened Bones** may slowly shift, causing foot to become deformed and changing the way distributes pressure.

• **Collapsed Joints**, especially a collapsed arch, can no longer absorb pressure or provide stability. The surrounding skin may begin to break down.
DIABETIC FOOT CARE

DIABETES REDUCES SENSATION WHICH CAN LEAD TO INJURIES

- **Blisters or Calluses** start as red or warm spots. They are often caused by unrelieved skin pressure.

- **Ulcers (sores)** may result if blisters or calluses reach the skin's inner layers. Ulcers may become infected.

- **Bone Infection** may occur if infected ulcers spread. Untreated bone infections may lead to loss of foot.
DIABETIC FOOT CARE AND EDUCATION

- CHECK YOUR FEET EVERY DAY
- DO YOU SEE RED SPOTS?
- DO YOU HAVE BLISTERS OR CALLUSES?
CARE AND EDUCATION

IRRITATIONS, SKIN LESIONS

BLISTER

CUTS BETWEEN YOUR TOES
• DO YOU FEEL TINGLING?
• ARE YOUR FEET COLD?
• ARE YOUR FEET NAILS INGROWN?
• HAS YOUR ARCH DECREASED?
CARE AND EDUCATION

- TEST THE TEMPERATURE OF THE WATER BEFORE PUTTING YOUR FEET

- WASH YOUR FEET WITH LUKEWARM WATER AND MILD SOAP
CARE AND EDUCATION

- KEEP SKIN SUPPLE & MOISTURISED

- CUT YOUR NAIL CORRECTLY
  Do not cut the corner of your toe nails
CARE AND EDUCATION

- DO NOT WALK BARE FOOT
EXAMINE YOUR FEET DAILY

- DRY YOUR FEET PROPERLY

- DO NOT SOAK MORE THAN 5 MINUTES
DIABETIC SHOES
How To Select The Right Shoes?

Sock with padding absorbs pressure.

Heel fits without slipping.

Instep conforms to your foot and causes no irritation.

Toe box allows all toes to wiggle.

Shoes should be comfortable as soon as you put them on at the store. You shouldn’t have to “break them in.”
GOOD DIABETIC SHOES

- Both feet measures
- Deep and wide toes box
- Flexible rubber soles
- Cushioned insole, 0.5-1 cm thick and softness
GOOD DIABETIC SHOES.....

- Deep & wide enough to accommodate the foot
- A firm heel counter/Back strap
- Adjustable by laces/velcro fasteners to keep the shoe on the foot securely
- Acceptable to the patient in appearance, cost & function
TYPE OF FOOTWEAR

Custom Molded Shoes With Insoles
TYPE OF FOOTWEAR

Molded Sandal
WARNING SIGNS AND SYMPTOMS OF DIABETIC FOOT PROBLEMS

- **Vascular**
  - Cold feet
  - Calf pain at rest
  - Absent femoral or pedal pulses

- **Neurologic**
  - Burning, tingling or pain sensation
  - Vibration and temperature
  - Hypersensitivity
  - Muscle weakness
  - Tendon reflex
  - Abnormal sweating

- **Dermatologic**
  - Dry skin
  - Skin color changes
  - Recurrent infection
  - Baldness
  - Scaling
  - Itching
  - Painless/Painful wounds
  - Ulceration
  - Ingrown toe nails

- **Musculoskeletal**
  - Change in shape
  - Swelling w/o trauma
  - Claw toes
  - Drop foot

Refer to SPECIALIST
REMEMBER......

• EXAMINE YOUR SHOES BEFORE PUTTING THEM ON

• DON’T ATTEMPT SELF TREATMENT

• SEEK IMMEDIATE MEDICAL ATTENTION
THANK YOU

HATUR NUHUN
Type of Footwear

Molded Insole

1. Increasing wt.bearing area
2. Assist the foot in normal function
Metatarsal bar
Thickening of the skin

Avenue for Infections

Supports
Wrong

Right

C

D
Kategori resiko 3. Edukasi ditekankan pada kontrol penyakit. Alas kaki yang pas, pemeriksaan sendiri, perawatan kulit/kuku/kalus dan pelaporan awal jika terjadi cedera kaki. Pemakaian alas kaki yang dalam, molded ortosa; alas kaki yang dimodifikasi, AFO sesuai yang dibutuhkan. Kontrol ulang 1-12 minggu untuk evaluasi kaki/aktivitas/alas kaki dan perawatan kulit/kuku.

Kategori resiko 2. Edukasi ditekankan pada kontrol penyakit, disain sepatu dan ukuran yang pas, pemeriksaan sendiri, perawatan kulit/kuku/kalus, melapor segera jika terjadi cedera kaki. Pemakaian alas kaki yang dalam, molded ortosa, modifikasi sepatu jika perlu. Kontrol rutin tiap 1-3 bulan untuk evaluasi kaki/aktivitas/alas kaki dan perawatan kulit/kuku.

Kategori resiko 1. Edukasi ditekankan pada kontrol penyakit, disain sepatu dan ukuran yang pas, pemeriksaan sendiri tiap hari, perawatan kulit/kuku, melapor segera jika terjadi cedera kaki. Alas kaki yang pas dengan soft insert/soles. Kontrol rutin 3-6 bulan untuk pemeriksaan kaki dan sepatu dan perawatan kuku.
PATOGENESIS

Diagram showing the patogenesis of diabetes mellitus, including peripheral vascular disease, autonomic neuropathy, and peripheral neuropathy, leading to complications such as impaired wound healing, increased bone reabsorption, joint collapse, deformed foot (Charcot), new pressure points, infection, ulceration, gangrene, and amputation.
DIABETIC FOOT LESION GRADING SYSTEM
- WAGNER
MANAGEMENT

Rehabilitasi:

Multiple factors contributing to the cause of foot problems in the diabetic patient:

1. Agent
2. Host
3. Environment

Interaction but remaining in equilibrium (normal)

NATURAL HISTORY OF FOOT DISORDERS IN THE DIABETIC PATIENT

Prepathogenesis Period

<table>
<thead>
<tr>
<th>Specific Protection</th>
<th>Health Promotion</th>
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</thead>
<tbody>
<tr>
<td>1. Protection from trauma</td>
<td></td>
</tr>
<tr>
<td>2. Eliminate pre-disposing biomechanical factors</td>
<td></td>
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<tr>
<td>3. Use of emollients or drying agents for skin</td>
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<tr>
<td>4. Periodic foot examination</td>
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<td>5. Personal hygiene</td>
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<td>6. Foot health education</td>
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</table>

Period of Pathogenesis

Early Diagnosis Prompt Treatment
1. Case finding measures (e.g., screening)
2. Cure or decelerate disease process
3. Provide facilities for care
4. Provide necessary foot care including orthotics, medical, and surgical podiatric services

Disability Limitation
1. Shorten or avoid disability
2. Prevent or reduce complications
3. Surgical, orthopaedic and/or prophylactic foot care

Rehabilitation
1. Provide hospital and community facilities
2. Provide rehabilitative services
3. Prolonged therapy including orthotic/prosthetic care
4. Surgical rehabilitative procedures
5. Achieve or maintain normal blood sugar

Primary Prevention

Secondary Prevention

Tertiary Prevention

Time

Late
Death
Limb loss
Partial limb loss
Chronic state
Disability
Recovery

Early
Clinical

Pre-Clinical (not detectable)

Early pathogenesis

Latent period

Interaction but resulting in diseases or disorders

Amputation
Necrosis
Osteomyelitis
Ulcer/infection
Hyperkeratosis
Vesicle, fissure
Pain, anesthesia, pruritus
Erythema
Slightly discernible

PREPARED BY: [Name]

DATE: [Date]
Conclusion

- Multi-disciplinary approach needed
- Going to be an increasing problem
- High morbidity and cost
- Solution is probably in prevention
- Most feet can be spared...at least for a while