1. **Introduction**
   - Diabetes is the single most important risk factor for lower limb amputation
     - Responsible for 40-60% of all lower limb amputations in the USA
     - Relative risk for lower limb amputation is 40 times greater in diabetics
     - Increased incidence for contra-lateral limb amputation in 30-50% of patients within 3 years
   - Foot ulceration is one of the most common and important complications of diabetes
     - Affects 15-25% of all diabetics during their lifetime
     - 20% Of all hospital and other costs in diabetic patients are due to foot ulcers and/or amputations
   - RSA has one of the highest incidences of diabetes mellitus worldwide
     - ± 7% Of the general population
     - > 7% Of the urban black population and 14% of the asian population
   - High socio-economic impact
     - Direct costs of medical therapy
     - Loss of personal income
     - Loss of productivity
     - Loss of quality of life

2. **Pathogenesis**
   Three mechanisms are involved in the pathology of the diabetic foot:
   1. Neuropathy
   2. Ischaemia
   3. Infection

2.1. **Neuropathy:**
   Peripheral neuropathy is a common complication and affects up to 60% of all diabetics and is present in up to 80% of patients with diabetic foot lesions. It is usually a polyneuropathy, diffuse and symmetrical.
   - **Sensory neuropathy.**
     - Results in:
       - Decreased sensation of pain, pressure and proprioception
   - **Motor neuropathy.**
     - Results in:
       - Atrophy of the intrinsic foot muscles with flexion deformity of the toes
       - Imbalance between the long flexor and extensor muscles
       - Typical deformities of claw toes, hammer toes, pes cavus and equinus deformity
All of the above lead to abnormal distribution of the weight bearing surface of the foot with increased pressure on the metatarsal heads. 90% Of all ulcers are situated over pressure points.

- **Autonomic neuropathy:**
  - Results in:
    - Loss of sympathetic tone
    - Increase in AV shunting with defective distribution of nutrients
    - Anhidrosis with dry skin, fissures in the skin and callous formation
    - Increased flow in bone with osteopenia

**Charcot’s foot/joint:**
This is the end result of the sensory, motor and autonomic neuropathy namely an unstable foot and ankle with destruction of bone and joints.

### 2.2. Ischaemia:
Two types of vascular involvement is seen in diabetics namely:

#### 2.2.1. Microangiopathy:
Typically affects the capillaries and arterioles in the eye (retinopathy), kidneys (nephropathy) and peripheral nerves (neuropathy). In the diabetic foot there is not a micro-vascular occlusive disease, but a **functional impairment** of the micro-vascular system. This is characterized by:
- Endothelial dysfunction
- Thickening of the capillary basement membrane with impaired
  - Leucocyte migration
  - Distribution of nutrients
  - Inflammatory hyperaemic response on trauma
- Impaired neurogenic mediated vasodilatation
- Maldistribution of blood flow with decreased capillary flow to the skin resulting in ischaemia of the skin

#### 2.2.2. Macroangiopathy:
This is the typical atherosclerotic lesions of the coronary and peripheral arteries as seen in non-diabetic patients. It however differs from the distribution of atherosclerotic lesions in non-diabetics in that it is more common, affects younger individuals without sex difference and is multisegmental. The most striking difference is a pattern of multisegmental stenoses and occlusions of the **infragenicular arteries** (Arteria tibialis anterior, posterior and fibularis) with often-intact pedal arteries.

### 2.3 Infection:
The diabetic foot is more susceptible to infection due to:
- Structural changes in the foot secondary to underlying neuropathy
- Ischaemia caused by diabetic atherosclerotic disease and functional impairment of the micro-vascular system
- Metabolic changes associated with diabetes
Infection may be caused by a simple puncture wound, fissure of the skin, lesion of the nail plate, interdigital web space or a neuropathic ulcer.

Infection usually involves the plantar space of the foot, which consists of three compartments, viz. the medial, central and lateral plantar compartments. The thick plantar fascia forms the floor of these compartments whilst the roof is formed by the metatarsal bones and interosseous membrane. There are two thick intermuscular septae which run from the calcaneous to the heads of the first and fifth metatarsals respectively, and which divide the plantar space into the medial, central and lateral compartments.

The intrinsic muscles of each digit are confined within the respective plantar compartments so that untreated distal phalangeal infection may progress to a plantar abscess. Infection in these anatomic compartments cause increase in intracompartmental pressure with decreased capillary blood flow and progressive tissue ischaemia and necrosis. Because the roof of each compartment consists of bone and fascia, deep space infections may show little abnormality on the dorsum of the foot.
PATHOGENESIS OF THE DIABETIC FOOT

Neuropathy

Motor

Foot deformity

Abn pressure points

Callous

Sensory

↓ Sensation

Dry skin

↓ Sensation

Fissures

Autonomic

Impaired microcirculation

Peripheral vascular insufficiency

Micro

Macro

Ischaemia

Ulcer

Infection

Gangrene

Diabetes Mellitus

3. Clinical evaluation

3.1. History:

The following aspects are important in the history:

- Development of ulcer/infection – often follows on a seemingly insignificant event eg. minor trauma with cutting of a toe nail, superficial burn due to a hot water bottle or heater, fissure of the skin, blister due to inappropriate footwear, etc
- Duration of the ulcer:
  - Longstanding chronic ulcer is indicative of underlying ischaemia
• Recurrent or relapsing ulcers are indicative of an underlying structural abnormality in the foot with pressure points or untreated deep seated infection? osteomyelitis
• Symptoms of peripheral vascular disease – typical claudication and rest pain as seen in non-diabetics may be absent due to underlying neuropathy
• The classical symptoms of infection, eg. fever, rigors, etc may be absent
• Bloodglucose control – poor control or increased insulin requirements may be indicative of underlying infection
• Presence of other risk factors – smoking, dyslipidaemia
• Concomitant comorbidities – impaired cardiac and renal function
• Functional status – previous mobility. Bed ridden or wheelchair patients may be served better by primary amputation rather than extensive bypass surgery

3.2. Physical examination:
The following aspects are important:
• Infection
  1. Look for a port of entrance for the infection. Thorough examination of the toes, web spaces, plantar surface, etc
  2. Remove hyperkeratotic skin, callous and escar for possible underlying infection
  3. Other signs of infection eg. purulent discharge, crepitus, tenderness, cellulitis, erythema, sinus. Fever and tachycardia may be absent in diabetics
  4. If bone is visible in the depths of an ulcer or sinus it may be indicative of underlying osteomyelitis

• Arterial supply:
  1. Pulse state of the limb: absent pedal pulses are indicative of ischaemia
  2. Other signs of ischaemia include:
      a. Distal ulcers (tip of the toe)
      b. Ulcers not associated with pressure points (weight bearing surfaces or exostoses)
      c. Gangrene
      d. Absent granulation or bleeding in the ulcer
      e. Fissures, especially on the heel
      f. Decreased hair growth
      g. Pallor, especially on elevation
  3. ABI is often inaccurate due to hardening of the arteries (medial calcinosis). Toe pressures may be of more value.

• Neuropathy:
  A common complication in diabetics and important cause for ulcers.
  1. Protective sensation can be examined with the Semmes-Weinstein monofilament. Inability to feel the monofilament when it is pressed against the skin, correlates with an increased risk of ulcers
2. Typical deformities of sensori-motor neuropathy, eg. claw toes, Charcot joint, etc

4. **Special investigations**
   - MCS – pus swabs should be taken of all discharges, ulcers and sinuses before commencing antibiotic therapy. Culture should be taken from the depth of the wound or sinus to prevent only culturing superficial colonizing organisms.
   - X-rays for underlying osteitis, joint deformity and fractures
   - Arterial Duplex Doppler:
     Provides anatomical and physiological information regarding plaque, stenoses, occlusions, flow velocities, etc
   - Regional transcutaneous oxygen tension (TcPO2) is of value in predicting wound healing. Normal ± 60mmHg, critical limb ischaemia usually < 40mmHg and impaired wound healing < 20mmHg. Diabetics may develop ulceration at higher TcPO2 levels than non-diabetics due to AV shunting and micro-vascular dysfunction.
   - Arteriography:
     Absent pedal pulses is an indication for an arteriogram. Remember: the distribution pattern in diabetics differs from the non-diabetic population in that the infragenicular arteries (tibial arteries) are more commonly involved with often patent pedal arteries. The arteriogram should therefore show run-off into the foot.

5. **Treatment**
   Effective treatment of infection is the highest priority. Spectrum varies from limited superficial infection to fulminant sepsis with extensive necrosis and destruction of bone.

5.1. **Limited superficial infection:**
   a. No evidence of deep plantar space involvement or systemic infection
   b. Outpatient treatment
   c. Oral antibiotics according to MCS
   d. Mostly staphylococcal or streptococcal infection; therefore Penicillin or first generation Cephalosporin
   e. Appropriate wound care
   f. Non weight bearing (“off loading”)

5.2. **Ulceration and/or gangrene with deep plantar space infection:**
   a. Hospitalisation
   b. Bedrest with elevation of the involved limb
   c. Intravenous antibiotics. Start with broad-spectrum antibiotic and adjust according to MCS results. Usually polymicrobial infection and initial empirical choice of antibiotic may include the following:
      i. Clindamycin with a fluoroquinolone
      ii. Clindamycin with a third or fourth generation cephalosporin
      iii. Antipseudomonal penicillin.
   d. Antibiotics are prescribed for 10-14 days in the absence of osteomyelitis; in the presence of osteomyelitis a prolonged course of 4-6 weeks is given
e. Abscess or deep plantar space infection requires immediate surgical drainage with debridement of all necrotic tissue. Incisions are placed according to the anatomic compartments in the foot.

f. In the case of a non-functional foot or life threatening sepsis with haemodynamic instability an emergency guillotine amputation is indicated to remove the source of sepsis. The amputation is revised to a final level as soon as the sepsis is under control, metabolic abnormalities have been corrected and the arterial supply is adequate.

5.3. Ischaemia:
Revascularisation is indicated in cases of ischaemia (absent pedal pulses). This may require bypass surgery and/or endovascular procedures (percutaneous transluminal angioplasty ± stenting). Due to the distribution of the disease it may be required to do a distal bypass to one of the pedal arteries (dorsalis pedis, tibialis posterior). Autogenous vein is the conduit of choice.

5.4. General measures:
- Optimal glucose control
- Correct all metabolic and haematological abnormalities
- Optimal nutritional status
- Optimal foot care with regards to:
  - Local ulcer treatment and wound care
  - Non weight bearing treatment of the foot (“off loading”)

There are therefore three clinical scenarios:

1. The septic foot without ischaemia:
   Effective treatment of the infection with antibiotics, debridement and drainage as required.

2. The septic foot with ischaemia:
   Effective treatment of the sepsis as above followed by revascularisation.

3. Tissue loss without infection:
   Patients may present with ischaemia and/or gangrene without infection. Arterial revascularisation required.

6. Long term treatment and prevention of diabetic foot complications:
1. Optimal diabetic control
2. Optimal foot care including hygiene, care of toenails, prevention of mechanical or thermal trauma, proper footware (podiatrist)
3. Prevention of neuropathic ulcers:
   - Special footware to redistribute the weight bearing surfaces away from the metatarsal heads and other pressure points (podiatrist).
4. Timeous and active treatment of any infection, trauma and ulcers
5. Corrective orthopaedic surgery of foot abnormalities e.g. claw toes, hammer toes etc

Multi-disciplinary diabetic foot clinics are important in the longterm follow up of the diabetic patient and prevention of these complications.
TREATMENT OF THE DIABETIC FOOT

History and examination

IS THERE INFECTION?

Yes

Correct metabolic abnormalities

Patient septic, unstable, non salvageable foot

Guillotine amputation

Deep infection

IV Antibiotics

Drain sepsis

Debride necrotic tissue

Superficial infection

Oral Antibiotics

Wound care

Foot care

No

IS THERE ISCHAEMIA?

Yes

Salvageable, functional limb

Continued wound care, debridement, A/B

No

No

Formal amputation

Yes

Arteriogram

Revascularize

IS CORRECTIVE FOOT ∅ NEEDED?

Yes

∅

No

LONG TERM PREVENTATIVE TREATMENT