Diabetic Foot

Lower-extremity disorders such as ulceration, gangrene, and amputation are significant complications of diabetes mellitus and are leading causes for hospitalization in these patients.

Persons with diabetes have a 15% lifetime risk of developing a foot ulcer and have a 15- to 40-fold higher risk of lower-extremity amputation compared with their nondiabetic counterparts

Survival is poor in diabetic patients after an amputation, with higher levels of amputation generally having lower survival rates, 5-year survival rate after major amputation in this population is less than 50%.

- Diabetes causes more than 70% lower limb amputations

- Diabetes causes more amputations than land mines even in former war zones

- Foot ulceration, sepsis, and amputation are feared complication of diabetes.

Times online December 8,2008

Definition

- Any infection involving the foot in a person with diabetes originating in a chronic or acute injury to the soft tissues of the foot, with evidence of pre-existing neuropathy and/or ischemia

- Diabetic foot is quiet dread of disability because
  - Long stretches of hospitalization
  - Mounting impossible expenses
  - Ever dangling end result of amputation

International Consensus on Diagnosing and Treating the Infect

History

Since the discovery of the association between gangrene of the foot and diabetes 160 years ago, many achievements have been made in treating diabetic foot ulcers.

Ulcer is as a complication of diabetes.

Patients with chronic diabetic ulcers have to deal with pain, infection hospital stays, and amputations.

For patients and their families, this results in a poor quality of life

Through the 19th century the problem of diabetic foot ulcers was described for the first time.

It was ***Marchal de Calvi*** in 1852 and ***Thomas Hodgkin*** in 1854 who realized that there was an association between diabetes and gangrene of the foot.

At that point in time, it was common to treat ulcers by prolonged bed rest, although it was noticed that the wounds would return once the patient started to mobilize again.

Frederick Treves (1853–1923) was an extremely talented surgeon, famous for performing the first appendectomy in 1888.

He suggested a different approach for the treatment of ulcers, using sharp debridement of callus after application of linseed poultices to soften the callus.

After debridement, an antiseptic cream would be applied to the thin fresh pink epidermis. Once the patient started mobilizing again he instructed the patient to wear a thick pad of felt plaster over the healed ulcer to reduce pressure and prevent recurrence of the wound.

At the end of the 19th century therefore, Treves had established three important principles in the treatment of ulceration of the foot: sharp debridement, off-loading pressure, and education about foot care and footwear.

One of discoveries of the 20th century was the isolation of secretion of the pancreas at the University of Toronto by ***Frederick Banting***.

Before insulin therapy, gangrene followed diabetic coma as a cause of death in patients with diabetes.

Insulin, although decreased dying from diabetic coma, the proportion of patients dying from foot disease increased significantly.

Insulin caused patients to live longer, but they experienced complications that had not been seen before.

Elliot Joslin, MD (1869–1962), established the first hospital foot clinic at the New England Deaconess Hospital (Boston, MA) in 1928.

He was also the founder of the multidisciplinary team approach in diabetic foot ulcers, he assigned one graduate nurse and two pupil nurses to educate patients.

Figure 2.12
Charles Best and Frederick Banting in Toronto in 1922 (the dog is thought to have been called Marjorie)

The discovery of penicillin by Scottish scientist

Alexander Fleming in 1928 led to a more conservative approach to surgery. For example, at Deaconess Hospital, the mortality for major amputations fell from 11.6% in to 6.6% in 1944–1949.

Another important achievement of the 20th century was revascularization and limb salvage.

Frank Wheelock (1919–2006) was the first surgeon responsible for the end-to side femoral popliteal bypass graft.

Angioplasty was initially described by an radiologist ***Charles Dotter*** in 1964. he successfully dilated a superficial femoral artery in an 82-year-old patient.

The 21st century: developed a clinical practice guideline for diabetic foot infections.

It was published for the first time in 2004.

The guideline recommends treatment of diabetic foot ulcers with surgical debridement, pressure off the wound, and moist dressings that control excess exudation.

The vascular state of a patient with a diabetic ulcer should always be investigated.

PAD is present in up to 50% of diabetic foot ulcer.

Early detection and treatment of PAD is important in limb salvage.

However less than 50% of the patients with diabetes and an ankle brachial pressure index < 0.5 underwent adequate vascular evaluation.

Patients who undergo major amputation, half of whom are dead within 3 years.

Classification & Staging

Classification & Staging

- A standard classification of diabetic foot is useful to
  - assess the etiology
  - find prognosis
  - facilitate appropriate treatment
  - monitor progress
  - serve as a form of communication

- No universally accepted classification

Classification & Staging

- The diabetic foot is classified on the basis of etiology into

➢ Neuropathic foot (neuropathy is dominant)
  - a. with infection
  - b. without infection

➢ Neuroischemic foot (vascular disease is dominant)
  - a. with infection
  - b. without infection

Chronic pressure (neuropathic) ulcers

Painless/callus/ pulse+/pressure site/pink

Ischemic ulcers

Painful/Bulla/Pulseless/Tip of fingers/red

Primary Infectious ulcers

below or between toes/purulent/deep ulcers

Traumatic ulcers **Every location and shape**

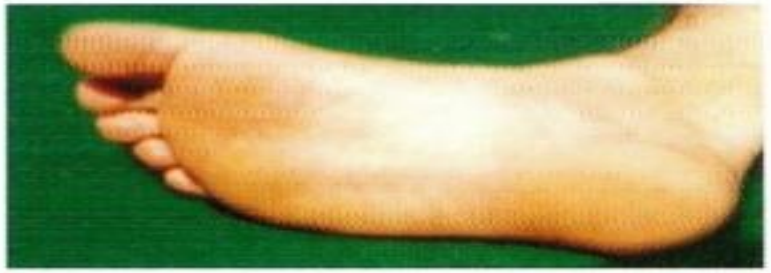
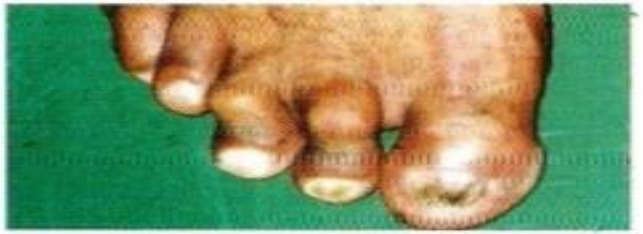
Atypical ulcers **Unexpected location, presentation or behavior**

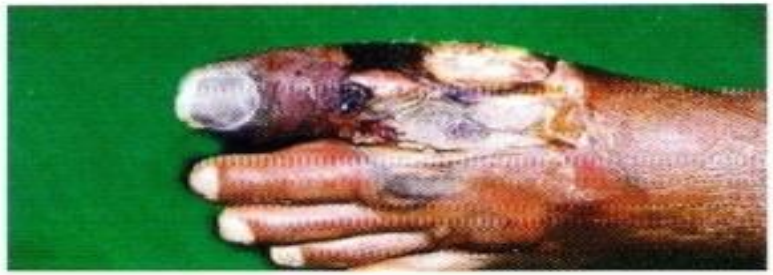
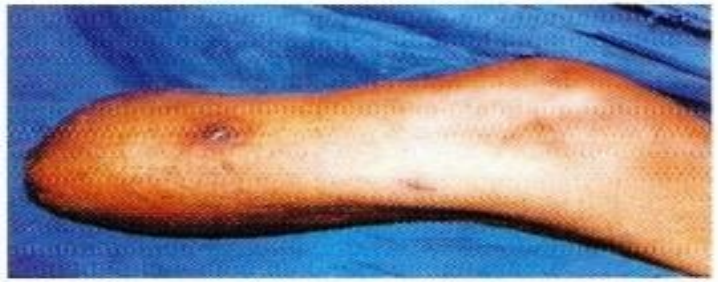
Classification & Staging

- According to natural history (ME Edmond & AV Foster).

Stage	Clinical condition
1.	Normal
2.	High risk
3.	Ulcerated
4.	Cellulitic
5.	Necrotic
6.	Major amputation

Classification & Staging

Stage	Clinical Condition
1.	 Fig. 7.9: Normal
2.	 Fig. 7.10: High-risk
3.	 Fig. 7.11: Ulcerated

Stage	Clinical Condition
4.	 Fig. 7.12: Cellulitic
5.	 Fig. 7.13: Necrotic
6.	 Fig. 7.14: Major amputation

Classification & Staging

- Wagner's Classification of diabetic ulcer
  - It is the most widely coated classification
  - It grades the foot depending on **severity**

Grade	Clinical finding
0	Intact skin (impending ulcer)
1	Superficial
2	Deep to tendon bone or ligament
3	Osteomyelitis
4	Gangrene of toes or forefoot
5	Gangrene of entire foot

Calhoun JH, Cantrell J. Foot Ankle. 1988 Dec;9(3):101-6.

Wound Classification

Texas System and the ***SAD*** System.

Both of these classifications include

Depth

Infection

Ischemia

which are considered important predictors for outcome and treatment.

the Meggit-Wagner System is still the most widely known and used system throughout the world.

Texas Staging

- *A Preulcer Or Postulcer Complete Epithelialised*
- *B Infection*
- *C Ischemia*
- *D Both Infection&Ischemia*

Classification & Staging

Wagner Classification

Grade 0 :

Fig. 7.3: No ulceration in a high-risk foot

Grade 1 :

Fig. 7.4: Superficial ulceration

Grade 2 :

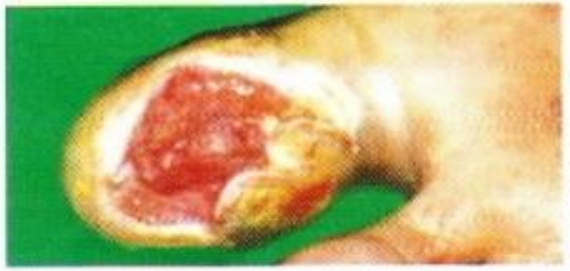


Fig. 7.5: Deep ulceration that penetrates to the tendon, bone or joint

Grade 3 :

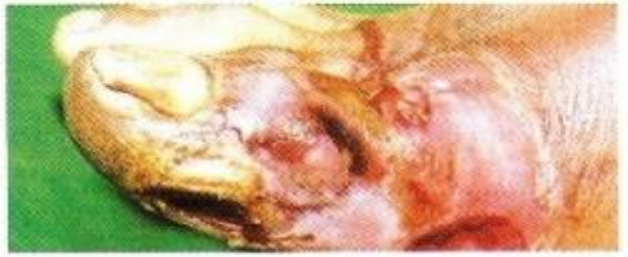


Fig. 7.6: Osteomyelitis or a deep abscess

Grade 4 :

Fig. 7.7: Localized gangrene

Grade 5 :

Fig. 7.8: Extensive gangrene requiring a major amputation

Pathogenesis

- Multi-factorial & Complex :
  1. Neuropathy
  2. Vasculopathy
  3. Immune dysfunction

- Prolonged Hyperglycemia contributes to all the above factors through different mechanisms

Neuropathy

- **Sensory Neuropathy**

Loss of pain sensation

↓

Unnoticed trauma (thermal, chemical, mechanical)

↓

Progression of lesion unchecked

↓

Callous formation

↓

Tissue necrosis & damage beneath callus

↓

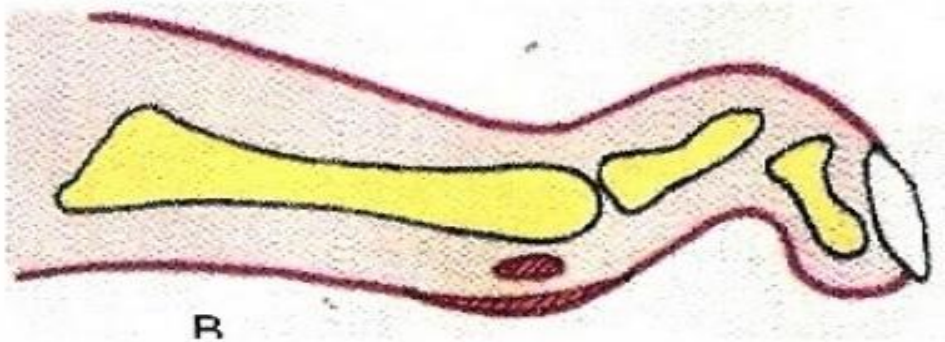
Development of cavities filled with serous fluid

↓

Erupt into surface

↓

Results in ulcer formation



Ulbrecht JS, Cavanagh PR. Clin Infect Dis. 2004 Aug 1;39 Suppl 2:S73-82.

Neuropathy

- **Motor neuropathy**

Weakness and wasting of intrinsic foot muscles

↓

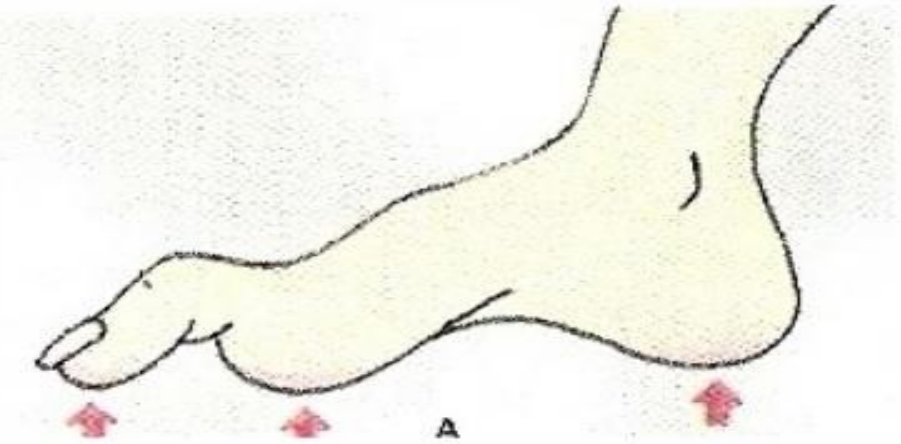
Foot deformities

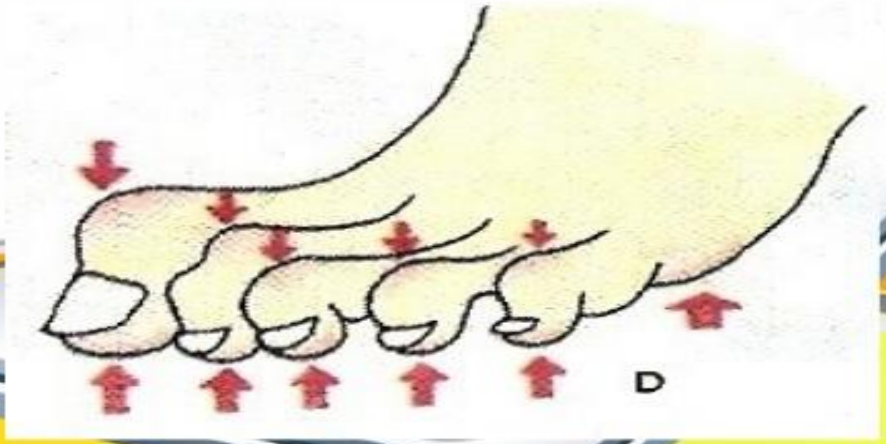
↓

Abnormal gait

↓

Ulceration





Neuropathy

Foot deformities predisposing to ulceration

- Clawed toes
- Bunions
- Nail deformities
- Deformities from previous trauma or surgery
- Ankle equinus
- Pes cavus
- Pes Planus
- Charcot foot
- Hallux rigidus
- Hallux Varus / Hallux Valgus
- Hammer toe

Neuropathy

- **Autonomic neuropathy**

Decreased sweating

↓

Dry & brittle skin

↓

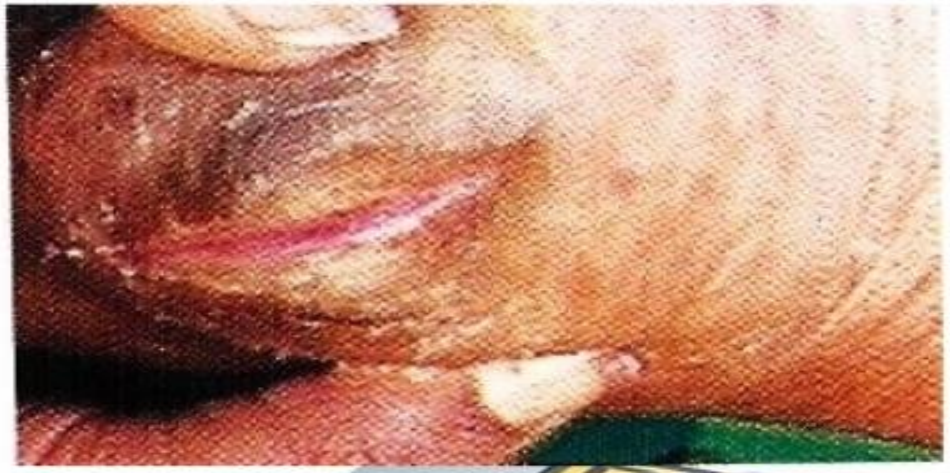
Fissures / Cracks

↓

Secondary infection

↓

Ulceration



Factors contributing to foot ulceration

Intrinsic factors	Extrinsic factors
• Bony prominences	• Walking barefoot
• Limited joint mobility	• Inappropriate footwear
• Deformities	• Falls and accidents
• Callus formation	• Objects inside shoes
• Previous foot ulcer	• Thermal trauma
• Neuroarthopathy (charcot)	• Activity level

Risk factors

Peripheral neuropathy in the main etiology of diabetic foot ulcers.

Peripheral vascular disease

biomechanical dysfunction and deformities

trauma

high plantar pressures

limited joint mobility

duration of diabetes

and elevated glycohemoglobin levels

Infection

Gangrene

Critical triad of ***neuropathy, trauma, and foot deformity*** was present in **63%** of ulcerated patients evaluated.

85% of amputations have an ulceration in their causal sequence

Studies have shown that within 6 years, ***20%*** of the combined diabetes group developed a new foot ***ulcer***, ***11.2%*** had an ***amputation***, and **18.3% died**.

Several measures of both small- and large-fiber neuropathy were predictive of both ulceration and amputation, including

pressure,

temperature,

vibratory perception thresholds

motor nerve conduction velocity (MNCV

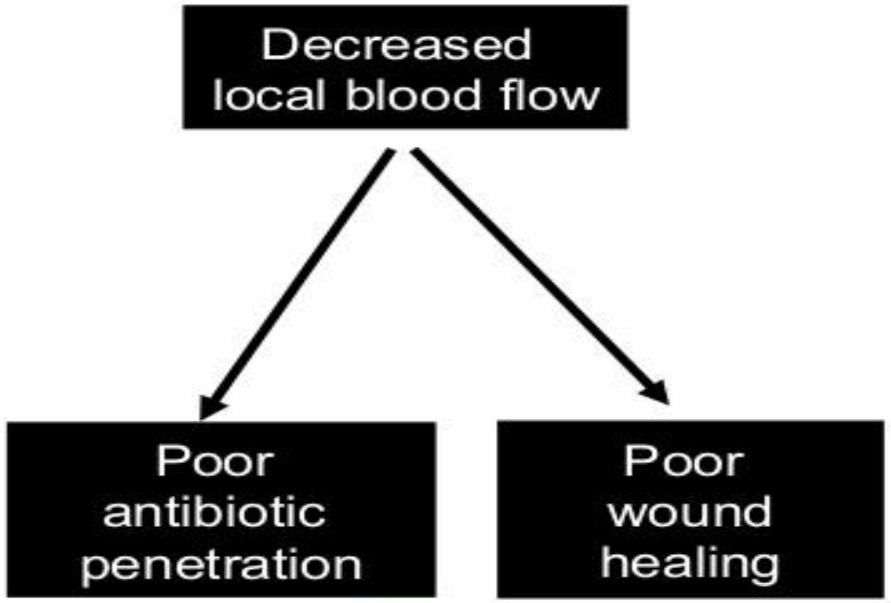
previous ulcer

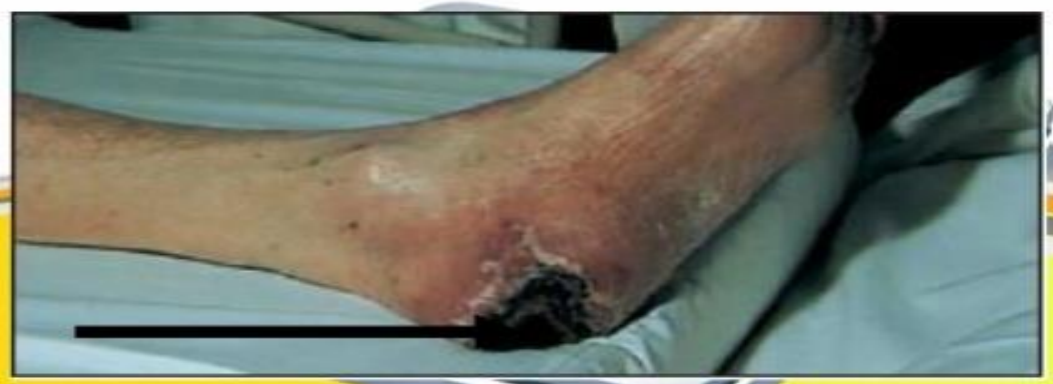
Vasculopathy

In diabetes there is macro and microangiopathy

1. Macroangiopathy
   - Atherosclerosis of large arteries

2. Microangiopathy
   - Increased thickness of basement membrane & endothelial proliferation
   - Capillary damage





Watkins PJ. BMJ. 2003 May 3;326(7396):977-9

Microvascular Physiology in Diabetes

There has been much interest recently in ***nitric oxide (NO)*** and its role in the etiology of metabolic perturbations associated with diabetes mellitus.

Using intradermal NO sensors and laser Doppler flowmetry, have presented that contrasted the physiologic effects of heating on NO production and skin perfusion

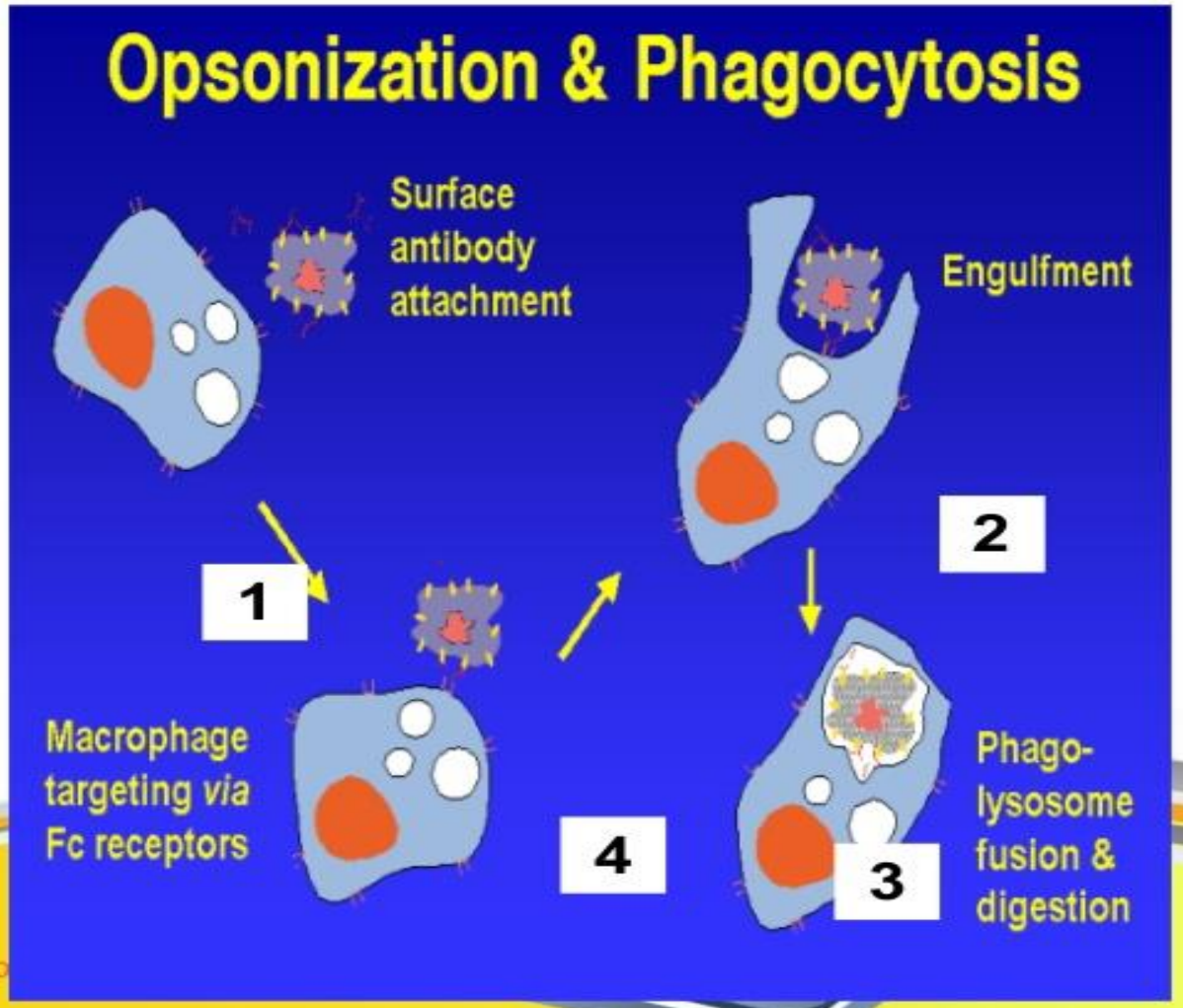
Whereas NO levels increased equally with heat in all groups, maximum perfusion and NO sensitivity (perfusion/NO) was impaired in diabets group.

suggest that NO production is not impaired in dabetes, but that *sensitivity* to NO is reduced and is the hallmark of the ***metabolic syndrome***.

Immune dysfunction

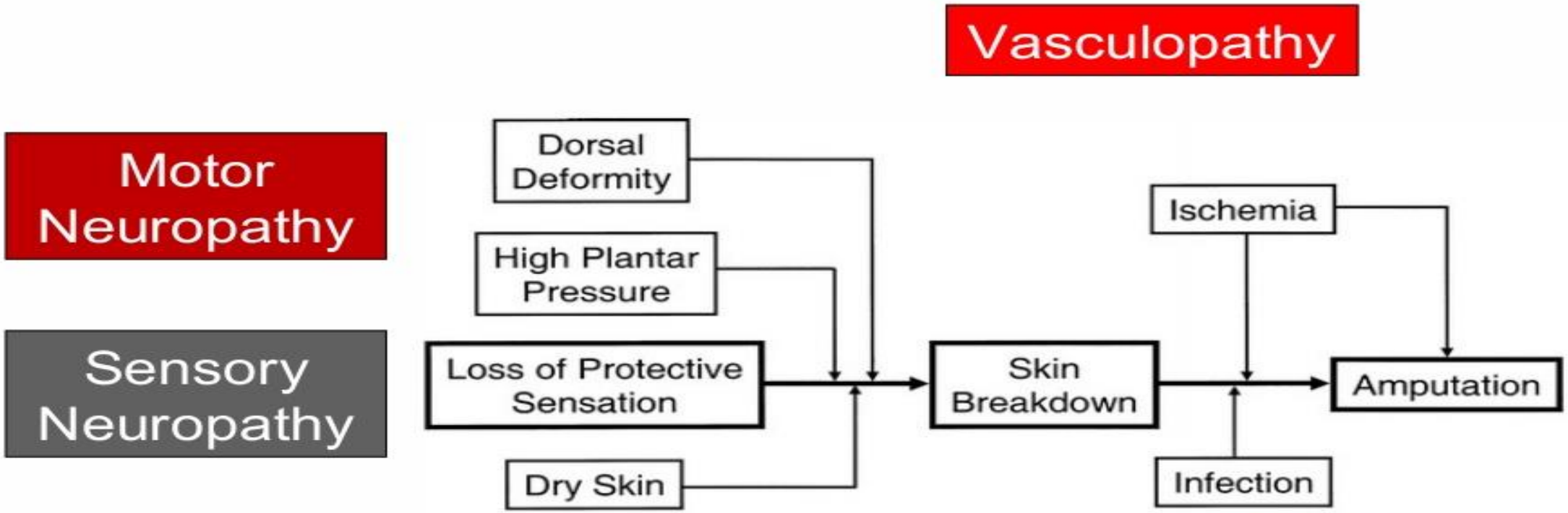
- Impaired defenses against infection

1. ↓ Polymorphonuclear leukocyte migration
2. ↓ Phagocytosis
3. ↓ Intracellular killing
4. ↓ Chemotaxis



Geerlings SE, Hoepelman AI. FEMS Immunol Med Microbio
1999 Dec; 26(3-4):259-65

Complex interplay of factors



Ulbrecht JS, Cavanagh PR. Clin Infect Dis. 2004 Aug 1;39 Suppl 2:S73-82

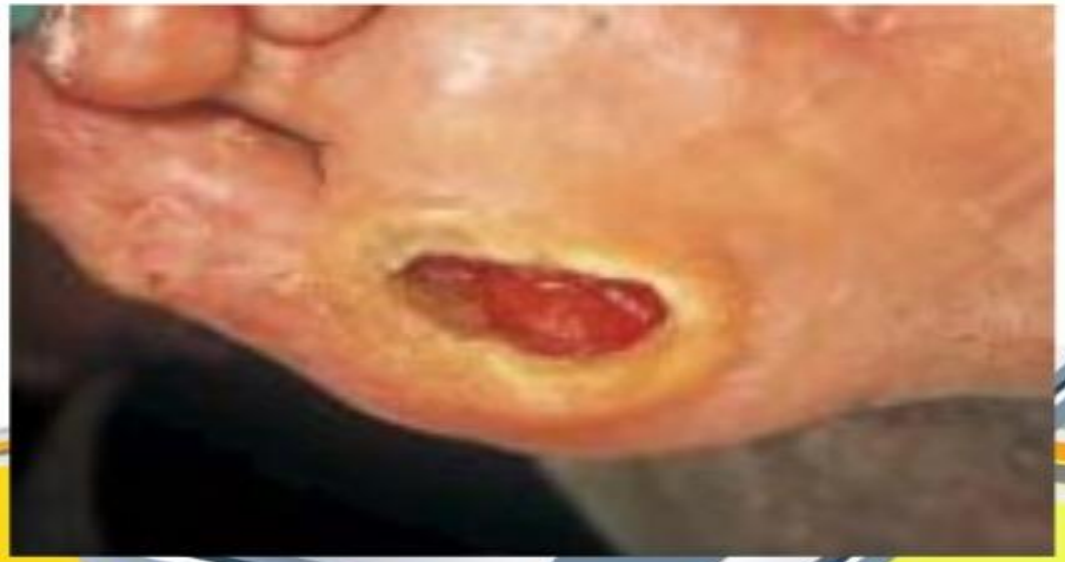
Clinical features

The neuropathic foot

- The foot has diminished sensation
- It invariably warm, with intact, often bounding pulses.
- Ulcers

Pressure points on planter surface

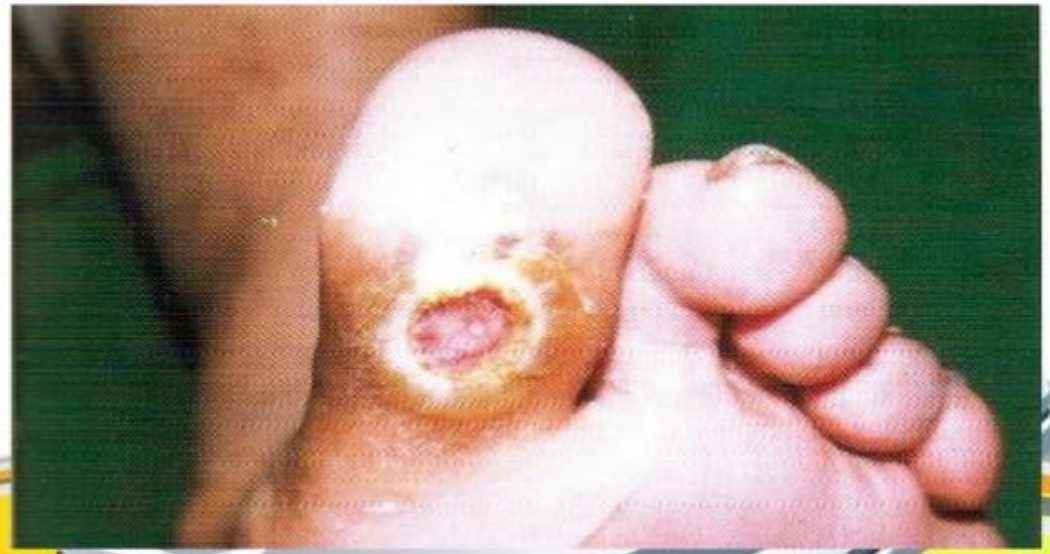
Stress areas on dorsal surface



Neuropathic ulcer

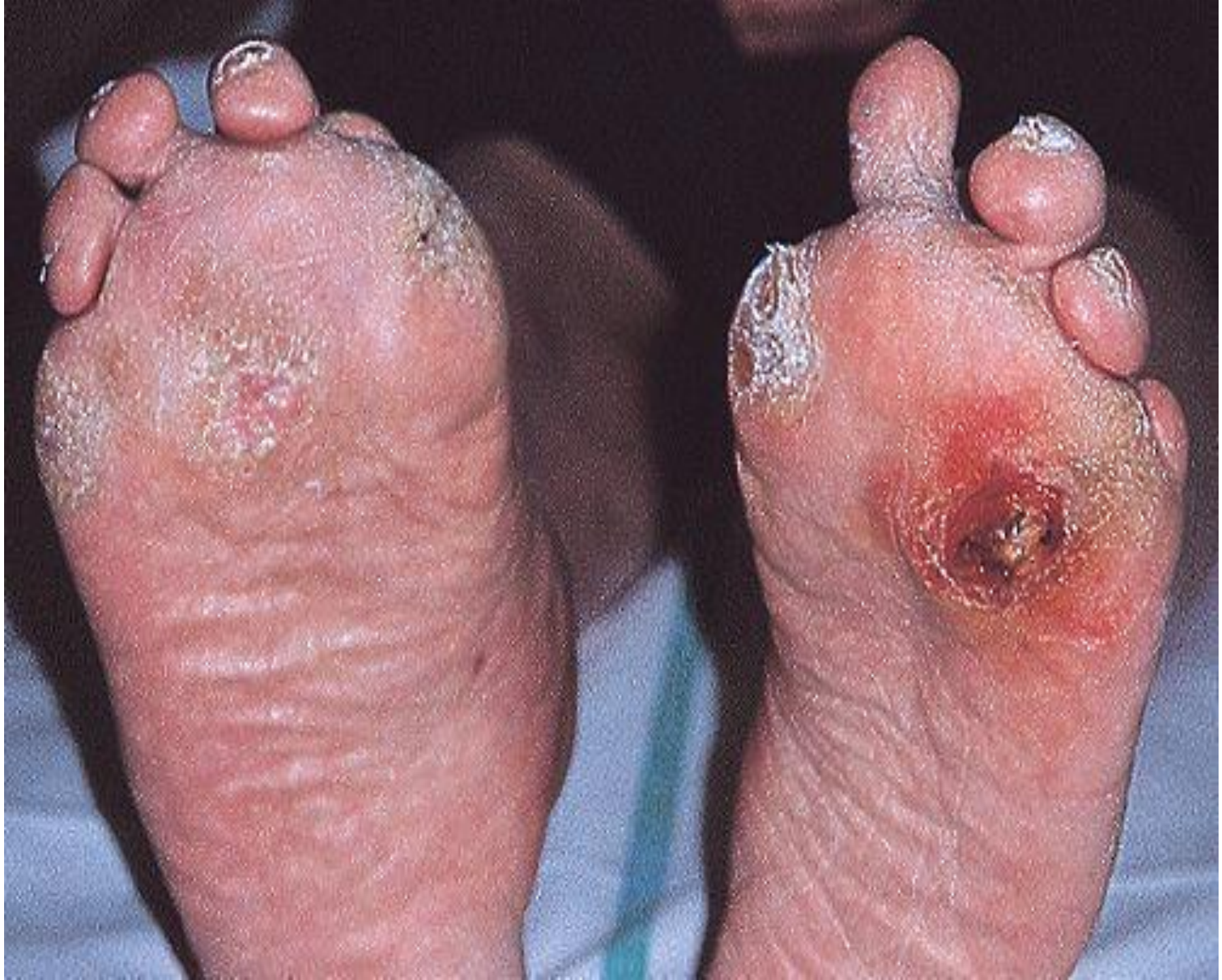
The neuropathic foot

- Ulcer often preceded by callus formation.
- Ulcers can be secondarily infected.
- Quickly lead to cellulitis, abscess formation, and osteomyelitis.
- Sepsis may complicate, resulting in gangrene.



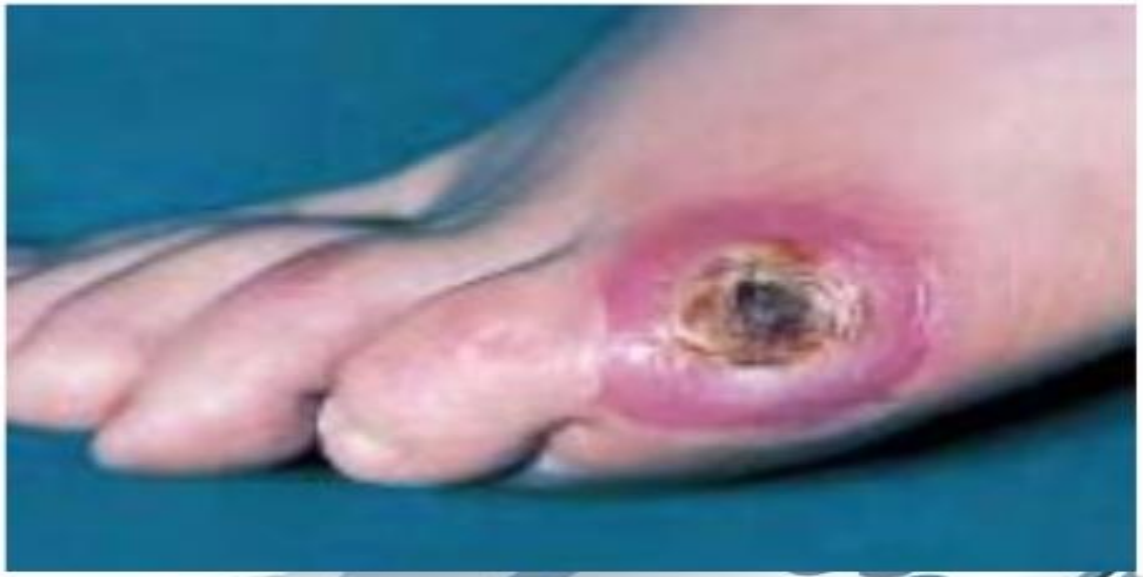
Neuropathic ulcer

Charcot foot



The ischaemic (neuro-ischaemic foot)

- Foot pulses are absent indicating ischaemia .
- Foot is not warm
- Lesions on the margins of the foot & tip of the toes.
- Absence of callus is characteristic features.

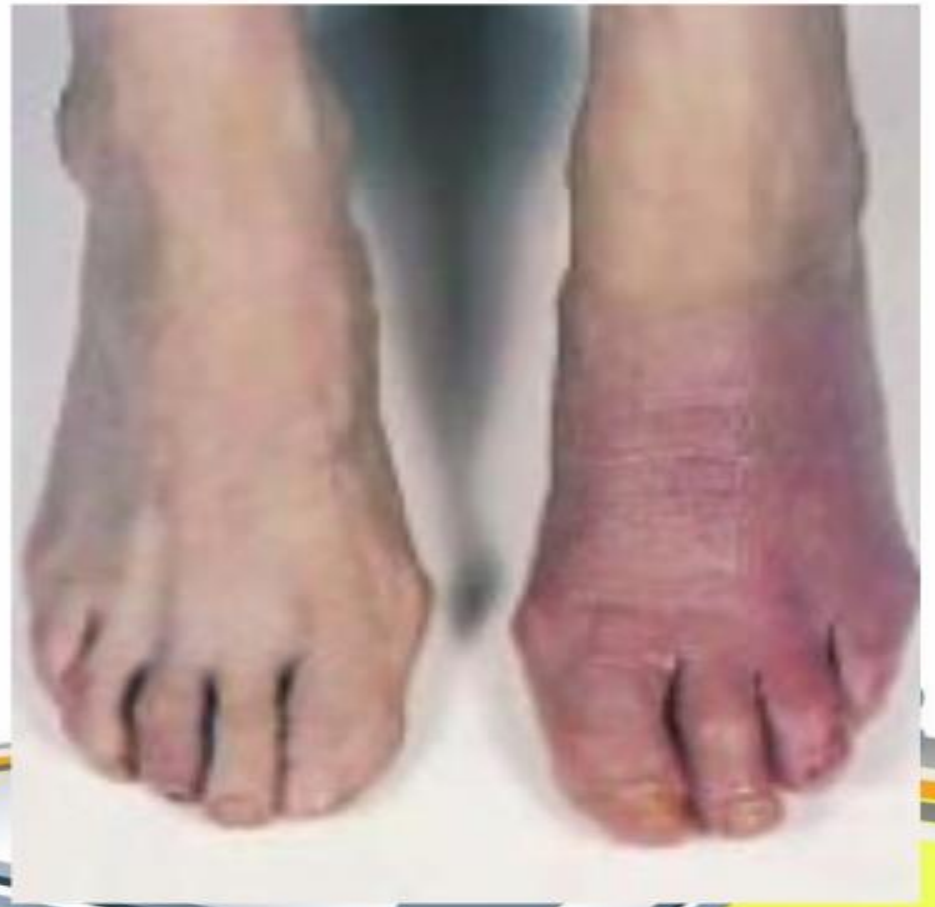


Ischaemic ulcer

The ischaemic (neuro-ischaemic foot)

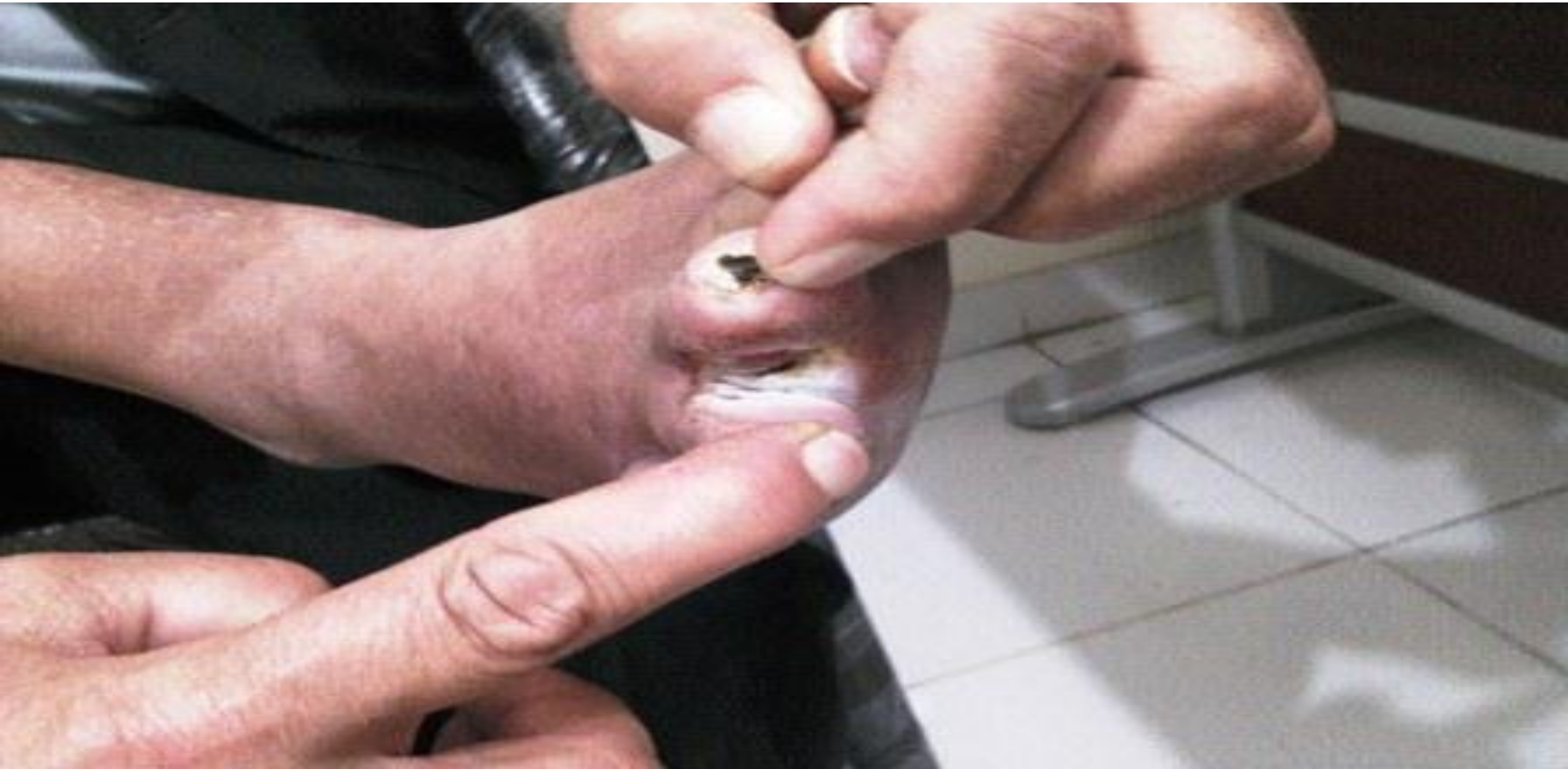
- Gangrene may be present.

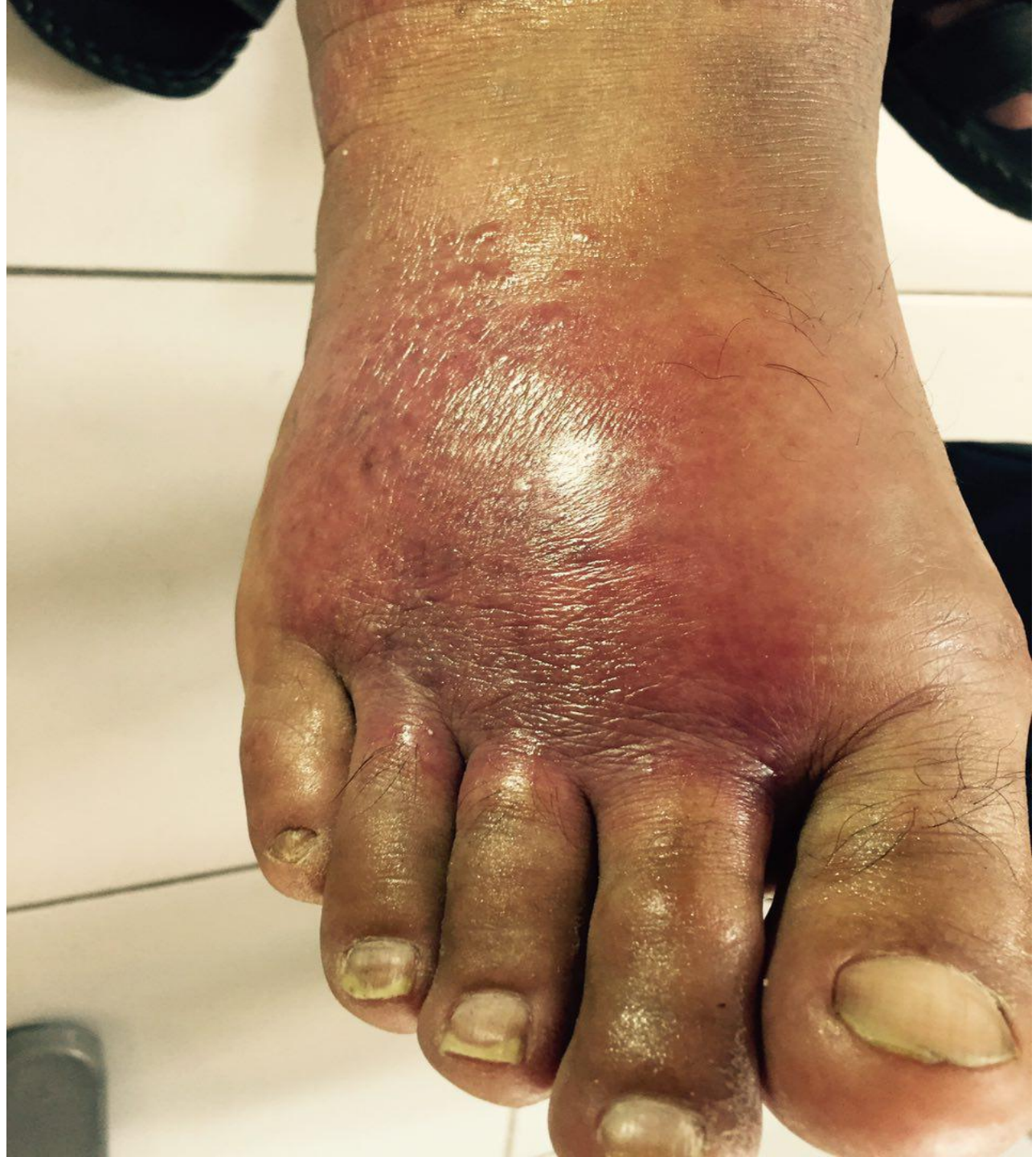
- It is essential to identify critical ischaemia
  - characteristic pink
  - painful
  - pulseless
  - cold foot



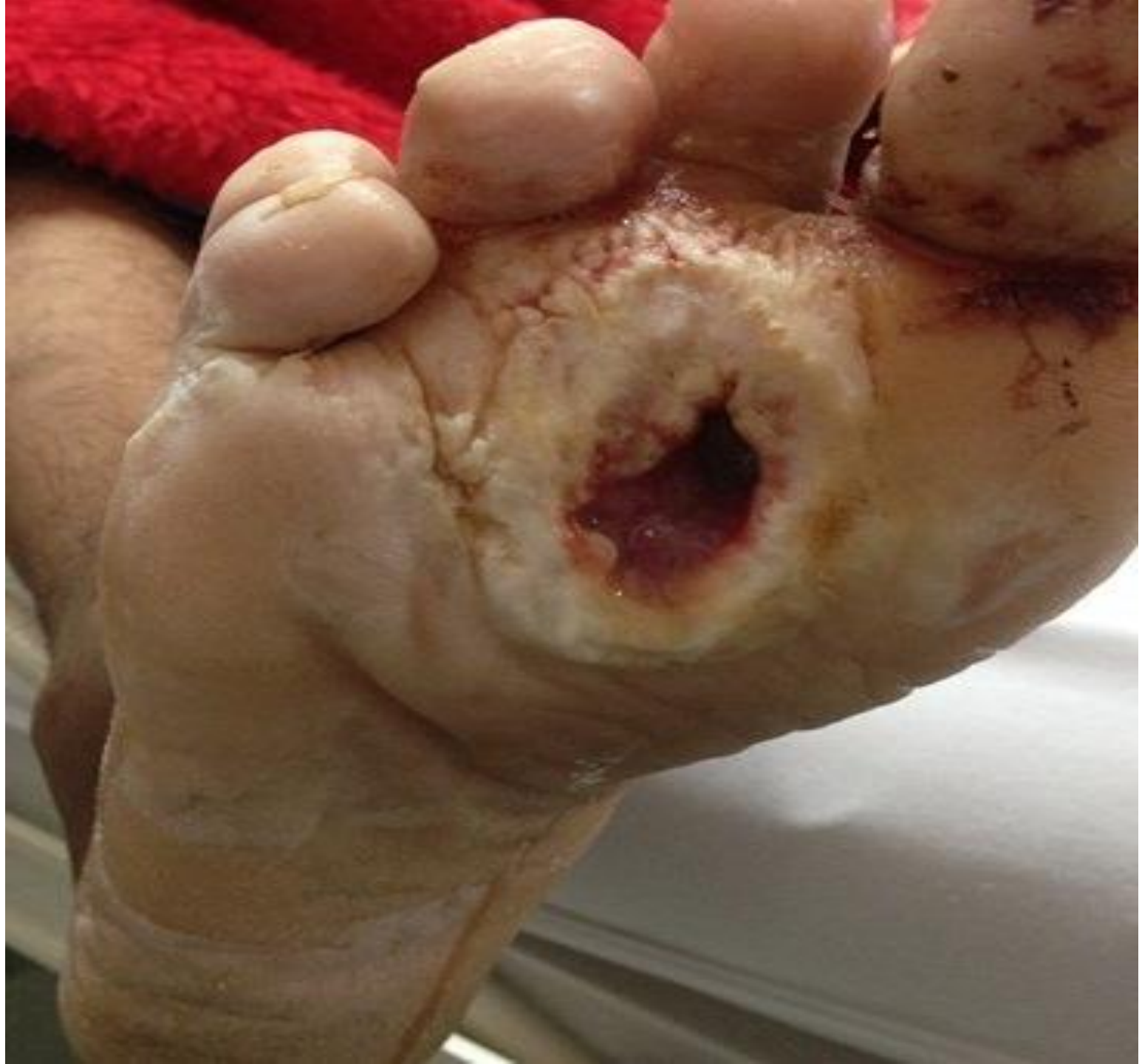
Characteristics	Neuropathic foot	Ischaemic (neuroischemic foot
• Skin temperature	• Warm	• Cold
• Pain	• Painless	• Painful
• Skin colour	• Not altered	• Dependent rubor
• Callus	• Thick at pressure point	• Usually not present
• Ulcer	• Usually on tips of toes & plantar surfaces under metatarsal heads	• Often on margins of foot, tips of toes, heels)
• Peripheral pulses	• Bounding	• Feeble / absent
• ABI	• more than 0.9	• less than 0.9
• Complication	• Charcot Joints	• Critical ischaemia

Primary infectious ulcer

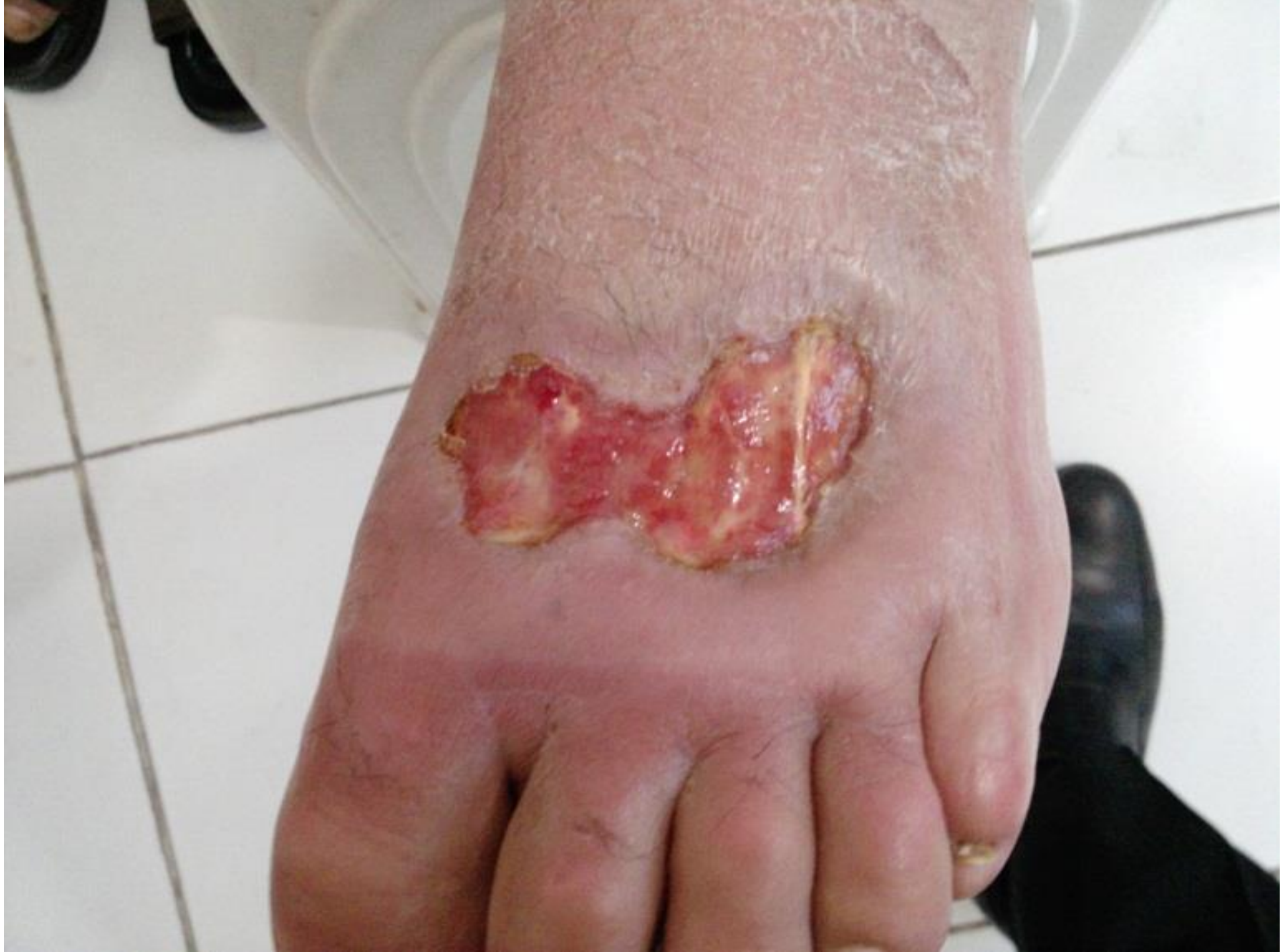


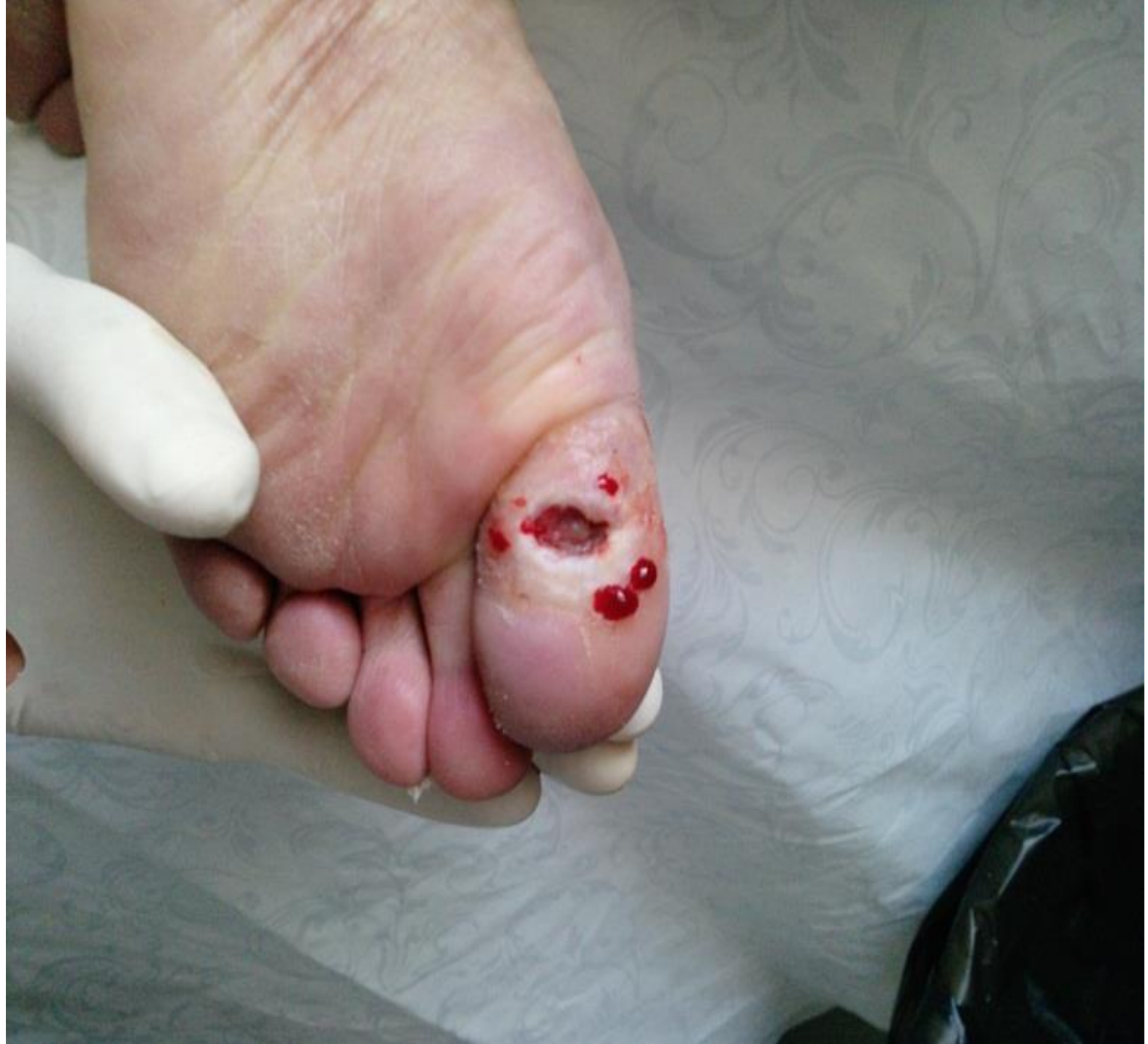


Deep osteomyelitis

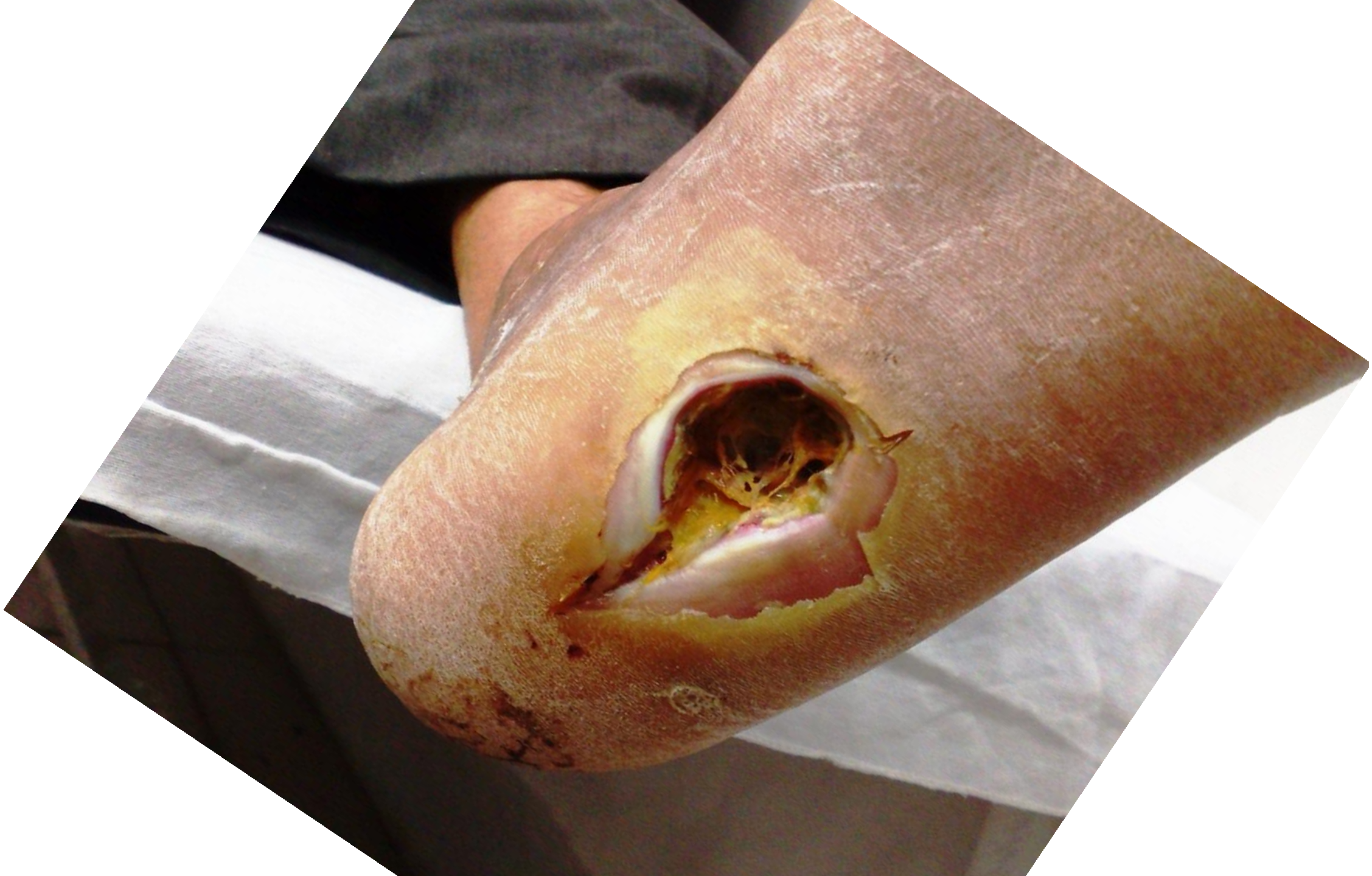


Atypical ulcers





Traumatic ulcer



Amputation were ***10-fold*** higher among diabetic patients

Furthermore, there was high regional variation

After a 26-month period, following a population screening, risk stratification, and treatment intervention,

there was a ***70%*** reduction in amputation

An study in Italy shows, there was nearly a ***30-fold*** incidence in diabetes amputations

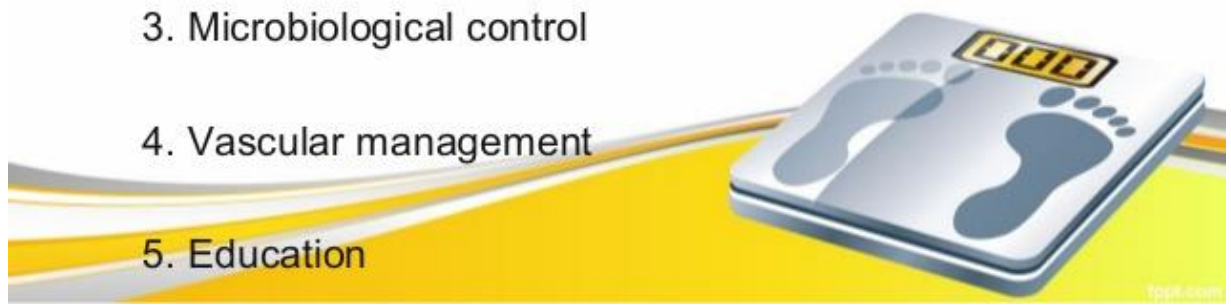
were higher rates in ***older*** persons

Management

Management

Five aspects of patient treatment

1. Mechanical control
2. Metabolic control
3. Microbiological control
4. Vascular management
5. Education

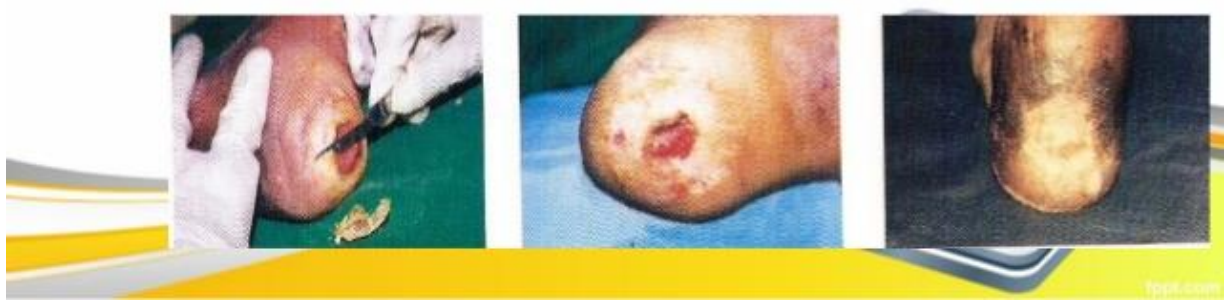


The Neuropathic foot

- Management of the ulcer falls into three parts:
  - removal of callus
  - eradication of infection
  - reduction of weight bearing forces, often requiring bed rest with the foot elevated.

The Neuropathic foot

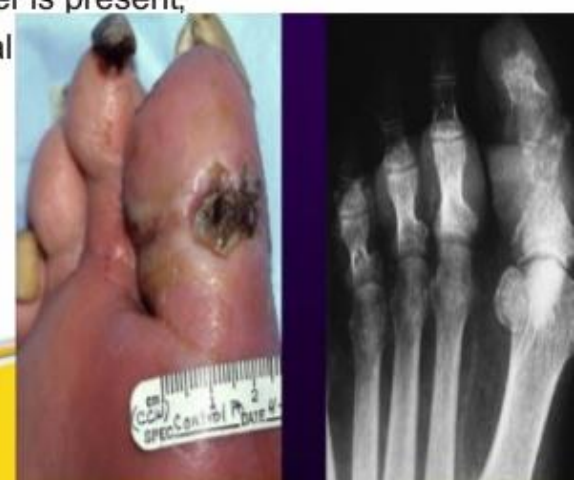
- Excess keratin should be pared away with a scalpel blade to expose the floor of the ulcer and allow efficient drainage of the lesion.



The Neuropathic foot

- Radiograph – to assess the possibility of osteomyelitis

  - A deep penetrating ulcer is present,
  - when lesions fail to heal
  - Continue to recur.

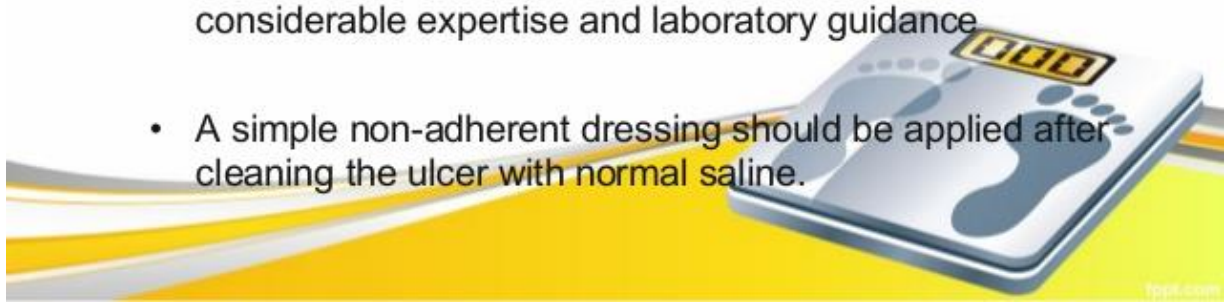


The Neuropathic foot

- A bacterial swab should be taken from the floor of the ulcer
- Culture of excised tissue
- A superficial ulcer may be treated on OPD basis
- Oral antibiotics are prescribed according to the organisms isolated on culture
- The most likely organisms to infect a superficial ulcer are
  - staphylococci
  - streptococci
  - anaerobes

The Neuropathic foot

- Treatment should be started with amoxycillin, flucloxacillin, and metronidazole,
- Antibiotics should be adjusted when results of bacteriological culture are available.
- Choice and duration of antibiotic administration require considerable expertise and laboratory guidance
- A simple non-adherent dressing should be applied after cleaning the ulcer with normal saline.

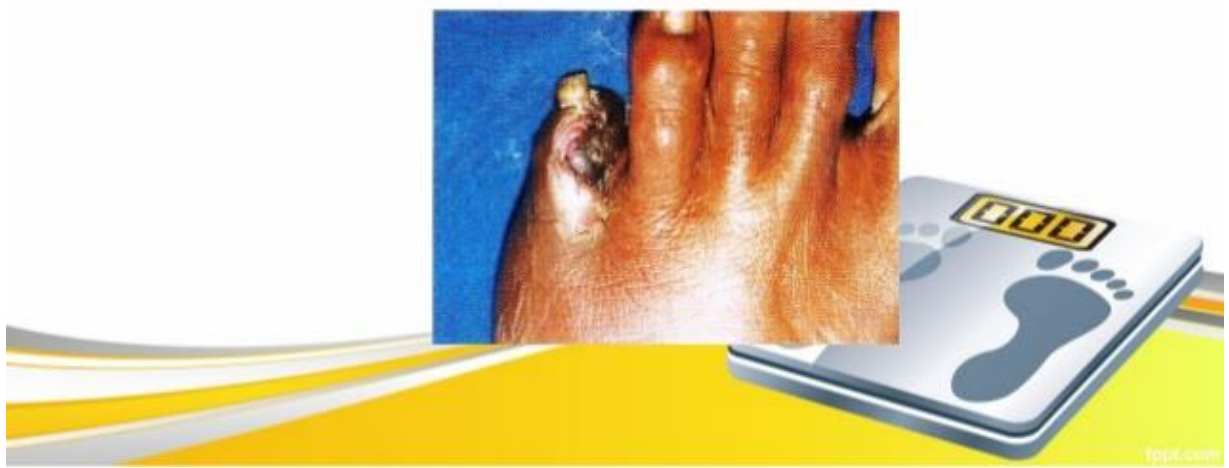


The Neuropathic foot

- Deep indolent ulcer requires off loading, with a total contact plaster cast.
- It should conform to the contours of the foot, thereby reducing shear forces on the plantar surface.
- Any foot lesion which has not healed in one month requires further investigation and a different approach.

The ischaemic foot

- In ischemic foot ulcer does not respond to medical treatment & vascular investigation is required.



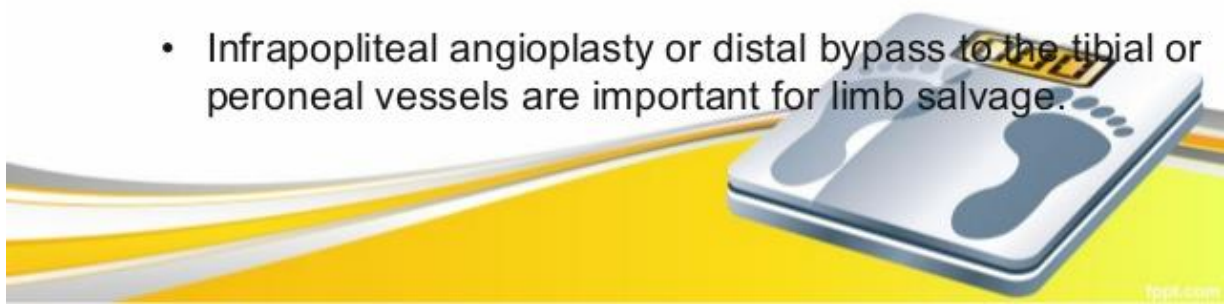
The ischaemic foot

- Doppler studies to measure the pressure index (the ankle/brachial ratio of systolic blood pressure):
  - pressure index1·2 - indicates rigid or calcified vessels or both
  - pressure index1 - normal (or calcified)
  - pressure index0·9 - indicates ischaemia present
  - pressure index0·6 - indicates severe ischaemia.

The ischaemic foot

- Arterial imaging by techniques include
  - duplex scanning,
  - magnetic resonance angiography,
  - conventional arteriography

- Infrapopliteal angioplasty or distal bypass to the tibial or peroneal vessels are important for limb salvage.



The ischaemic foot

- Amputation of the toe is usually unsuccessful in the neuroischaemic foot unless the foot is revascularised.
- If this is not possible, then a dry necrotic toe should be allowed to autoamputate.
- After attempts to control infection, below knee amputation is indicated in those with extensive tissue destruction.

The ischaemic foot

Rest pain in neuroischemic foot

- It can be relieved by successful revascularisation
- Paravertebral lumbar block
- If that fails, opiates have to be given for pain relief
- Below knee amputation may be the last resort.

Urgent treatment

Danger signs: urgent treatment needed

- Redness and swelling of a foot
- Cellulitis, discolouration, and crepitus (gas in soft tissues)
- A pink, painful, pulseless foot even without gangrene indicates critical ischaemia

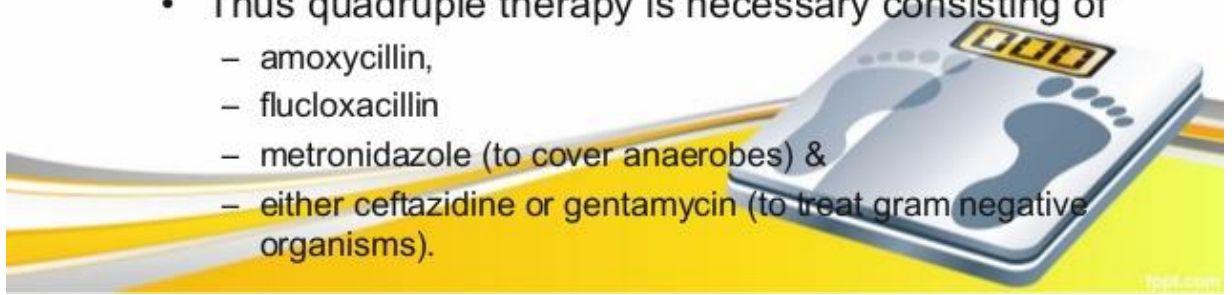
Urgent treatment

1. Bed rest.

2. Intravenous antibiotics.

- It is necessary to provide a wide spectrum of antibiotic cover.

- Thus quadruple therapy is necessary consisting of
  - amoxycillin,
  - flucloxacillin
  - metronidazole (to cover anaerobes) &
  - either ceftazidine or gentamycin (to treat gram negative organisms).



Urgent treatment

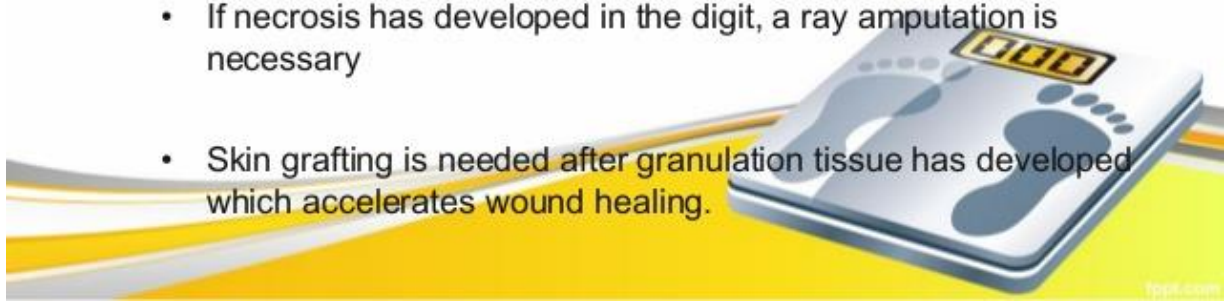
- This treatment can be adapted when results of bacteriological culture are available.
- The emergence of multiple resistant *Staphylococcus aureus* [illegible] *is presenting* a very serious problem,
- Available treatments include intravenous clindamycin , vancomycin and intramuscular teicoplainin.

Urgent treatment

3. An intravenous insulin pump may be necessary to control the blood glucose.

4. Surgical debridement
   - to drain pus and abscess cavities
   - to remove all necrotic and infected tissue
   - to remove devitalised and infected bone resulting from osteomyelitis

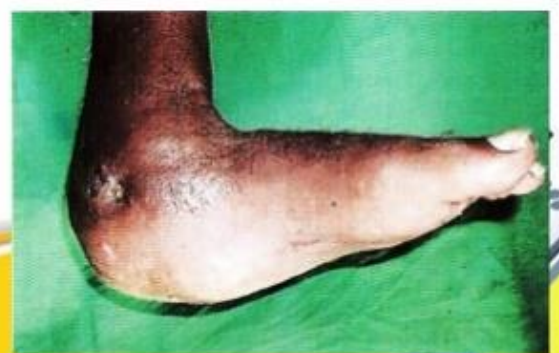
- If necrosis has developed in the digit, a ray amputation is necessary
- Skin grafting is needed after granulation tissue has developed which accelerates wound healing.



The neuropathic joint (Charcot's joint)

- Charcot's foot have loss of pain sensation & rarefaction of the bones.

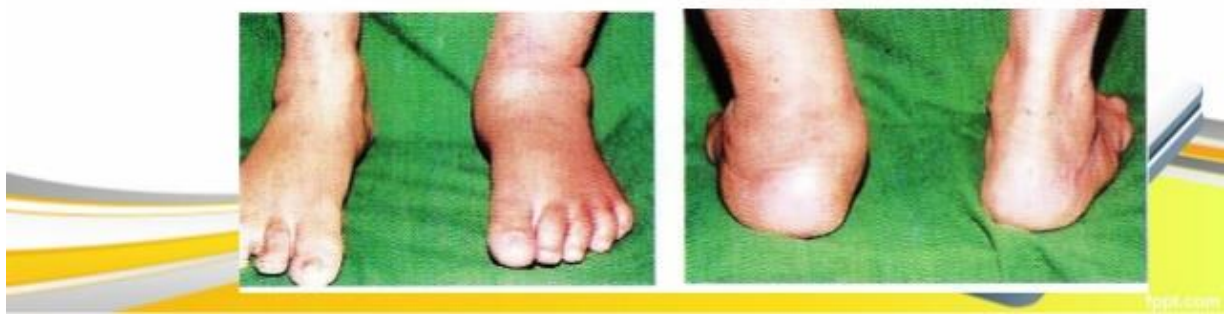
- Abnormal mechanical stresses (usually prevented by pain) damages the susceptible bones by relatively minor trauma.



The neuropathic joint (Charcot's joint)

- Patients present with a hot swollen foot, sometimes aching, this appearances are often mistaken for infection.

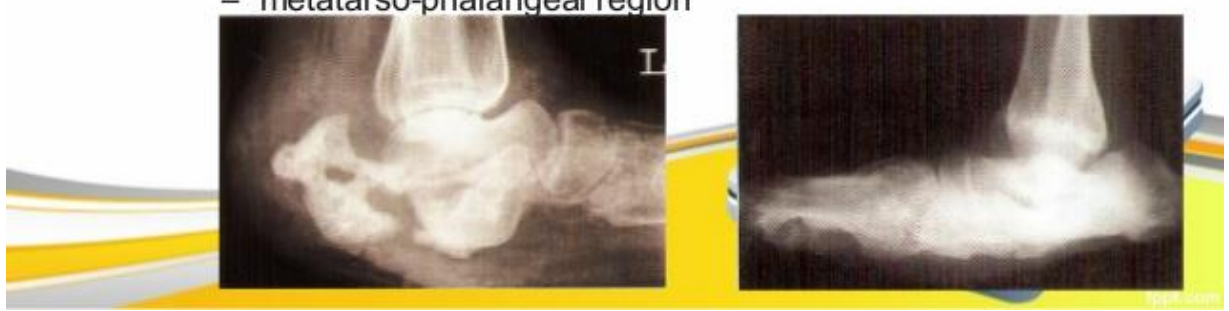
- Injury may have occurred days or weeks earlier, or may not even have been noticed.



The neuropathic joint (Charcot's joint)

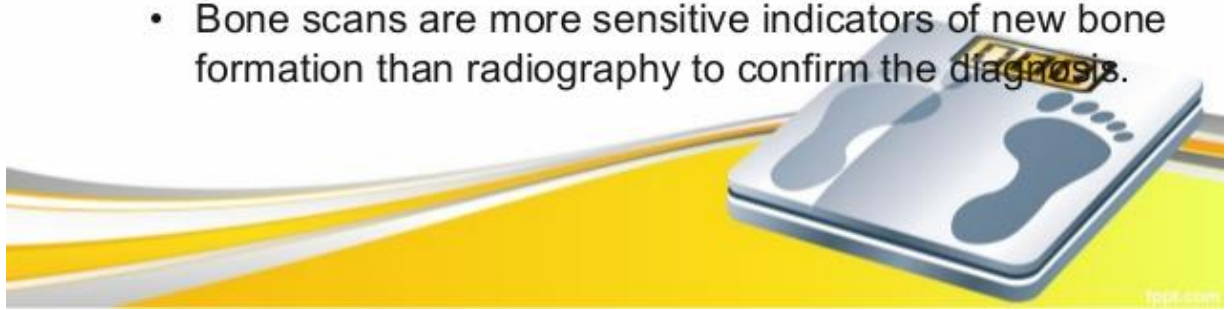
- The destructive process does not continue indefinitely but stops after weeks or months.

- Bony changes are most often seen at the
  - ankle joint
  - tarsal-metatarsal region of the foot or
  - metatarso-phalangeal region



The neuropathic joint (Charcot's joint)

- Early diagnosis is essential.
- The initial presentation of unilateral warmth and swelling in a neuropathic foot is suggestive of a developing Charcot joint.
- Bone scans are more sensitive indicators of new bone formation than radiography to confirm the diagnosis.



The neuropathic joint (Charcot's joint)

- It is essential to exclude infection
- When the diagnosis is difficult
  - gallium white cell scan and
  - magnetic resonance imaging scan

The neuropathic joint (Charcot's joint)

Management

Initial

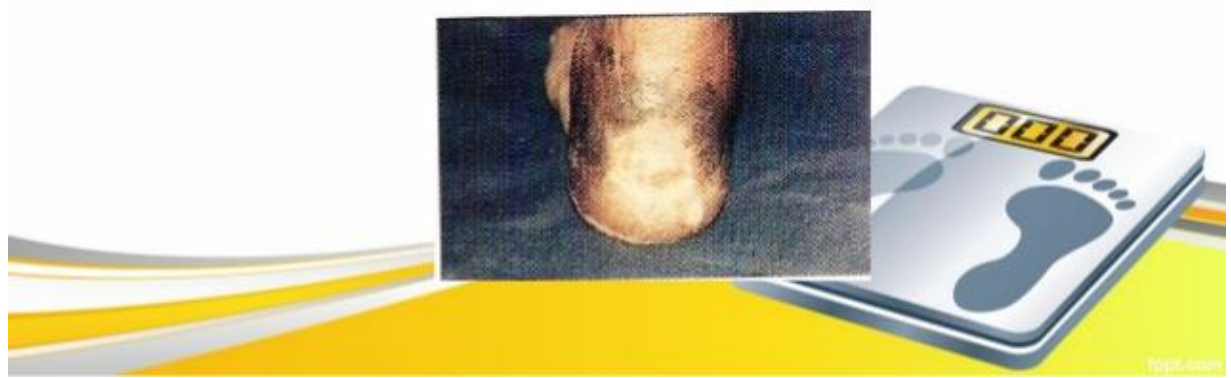
- Rest, ideally bed rest or use of non-weight bearing crutches (until the oedema and local warmth have resolved)
- Alternatively, the foot can be immobilised in a well moulded total contact plaster which is initially non-weight bearing.
- Immobilisation is continued until bony repair is complete, usually in two to three months.
- The use of bisphosphonates in preventing bone damage in Charcot foot is promising.

The neuropathic joint (Charcot's joint)

Long-term management

- Special shoes and insoles should be fitted to accommodate deformity and prevent ulceration (major hazard of the Charcot foot).

Long-term care of wound



Long-term care of wound

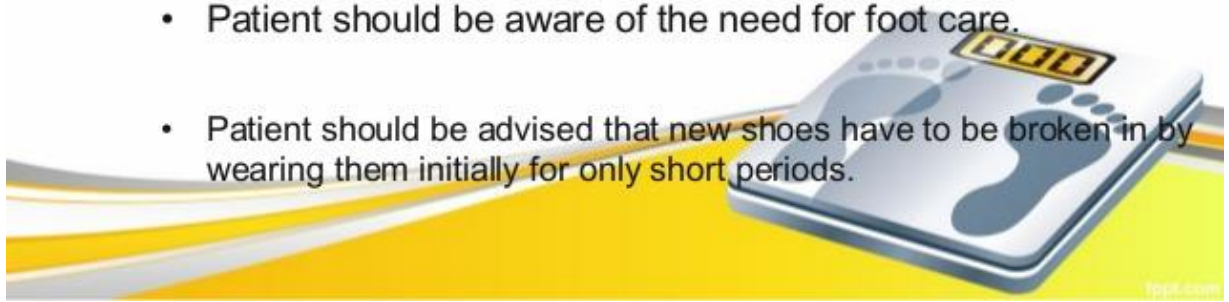
Footwear

- They are required for redistribution of weight bearing forces from vulnerable parts of the foot .
- Moulded insoles made from substances with energy absorbing properties such as plastozote and microcellular rubber are suitable on long-term basis.
- Failure to wear appropriate shoes is common a cause of recurrence in treated patients.

Long-term care of wound

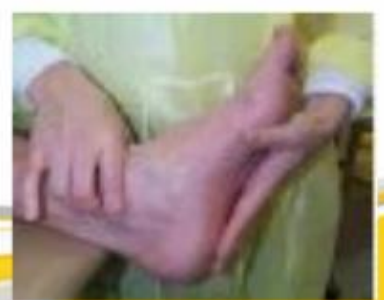
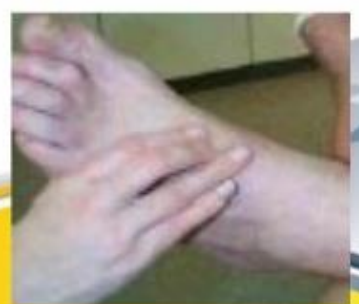
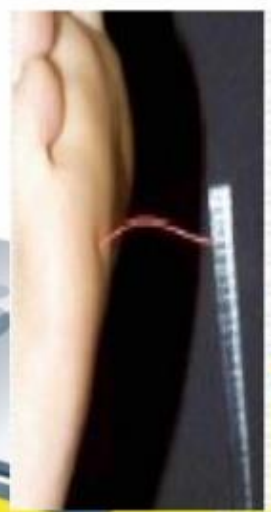
Screening and Prevention:

- The foot must be examined routinely at the onset of diabetes and annual thereafter.
- Identifying the critically ischaemic foot is important.
- Patient should be aware of the need for foot care.
- Patient should be advised that new shoes have to be broken in by wearing them initially for only short periods.



Long-term care of wound

- A simple sensory test should be performed (inability to detect 10 g or more indicates risk of foot ulceration).
- Examine the pulses (dorsalis pedis and posterior tibial).

Long-term care of wound

- Active lesions should be sought and treated immediately (for example, hidden lesions between the toes) .
- Deformities, callus, skin cracks, and discoloration need to be detected and managed.
- Advice and education must follow the examination

Prevention Guidelines

Prevention Guidelines

Low risk foot

- These are foot with normal sensation, palpable pulses
- Individual foot care education.

At risk foot

- neuropathy, absent pulses, or other risk factor described above
- Enhance foot care education.
- Inspect feet every six months.

Prevention Guidelines

High risk foot

- *These are foot with Ischaemia, deformity, skin changes, or previous ulcer*
- Three monthly follow up
- Intensified foot care education
- Special arrangements for people with disabilities or immobility.

Conclusions

- Many foot problems can be prevented, and all diabetic patients should be aware of the potential problem of foot damage.
- Every patient should be issued with information containing straightforward safety instructions.
- A good podiatrist must be available for diabetic patients.

Conclusions

- Close coordination between the podiatrist, orthotist, nurse, physician, and surgeon is vital in the care of the diabetic foot.

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