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A new view to
DIABETIC FOOT ULCER

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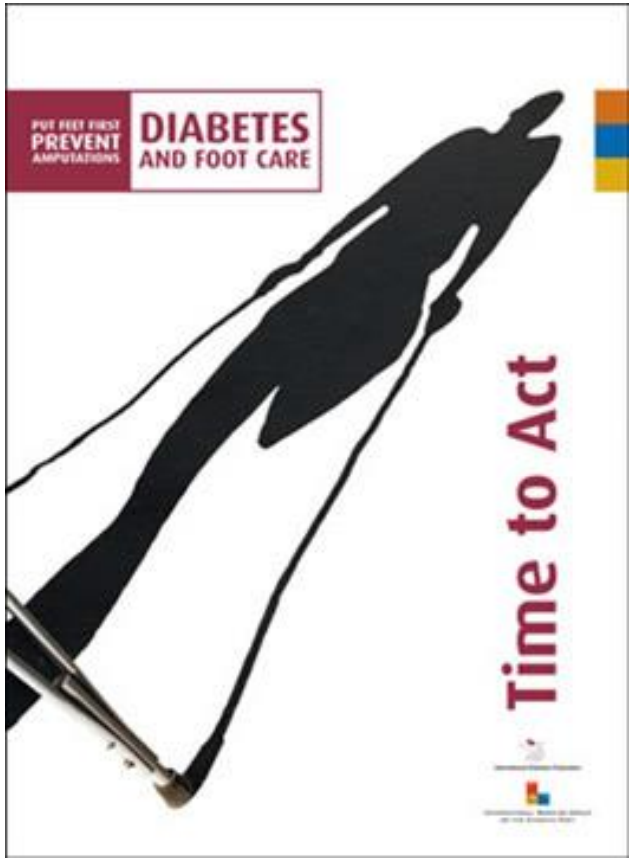
Every twenty seconds a lower limb is lost to diabetes. In most cases, the amputation could have been avoided

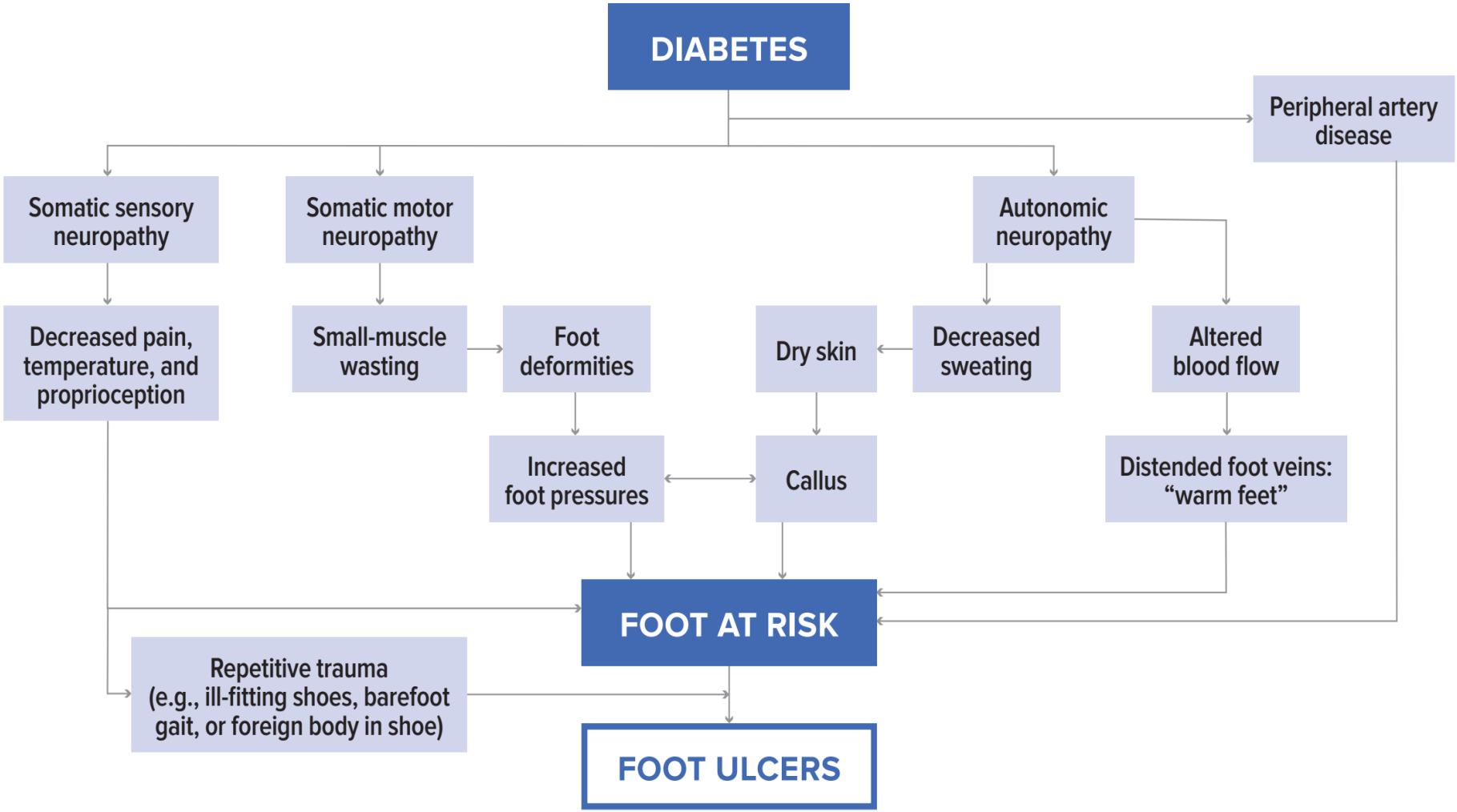


- Among patients with diabetes, 15% develop a foot ulcer, and 12-24% of individuals with a foot ulcer require amputation.
- Indeed, diabetes is the leading cause of nontraumatic lower extremity amputations in the United States.
- In fact, every year approximately 5% of diabetics develop foot ulcers and 1% require amputation.

Why DFUs?

- Neuropathy
- Arterial insufficiency
- Prone to infection
- Prone to trauma
- Chronic diabetic complications
- Poor hygiene
- Improper habits and beliefs

Why DFUs?


DIABETES
Peripheral artery disease
Somatic sensory neuropathy
Somatic motor neuropathy
Autonomic neuropathy
Decreased pain, temperature, and proprioception
Small-muscle wasting
Foot deformities
Dry skin
Decreased sweating
Altered blood flow
Increased foot pressures
Callus
Distended foot veins: "warm feet"
FOOT AT RISK
Repetitive trauma (e.g., ill-fitting shoes, barefoot gait, or foreign body in shoe)
FOOT ULCERS

PATHOGENESIS/PATHPHYSIOLOGY

- Every type of neuropathy may lead to DFUs:
  - Sensory
    - Superficial
    - Proprioceptive
  - Motor/
    - Weakness/strength imbalance
    - Deformity
  - Autonomic
    - Dry skin
    - Impaired Pressure-Induced Vasodilation

Foot deformities

Foot deformities

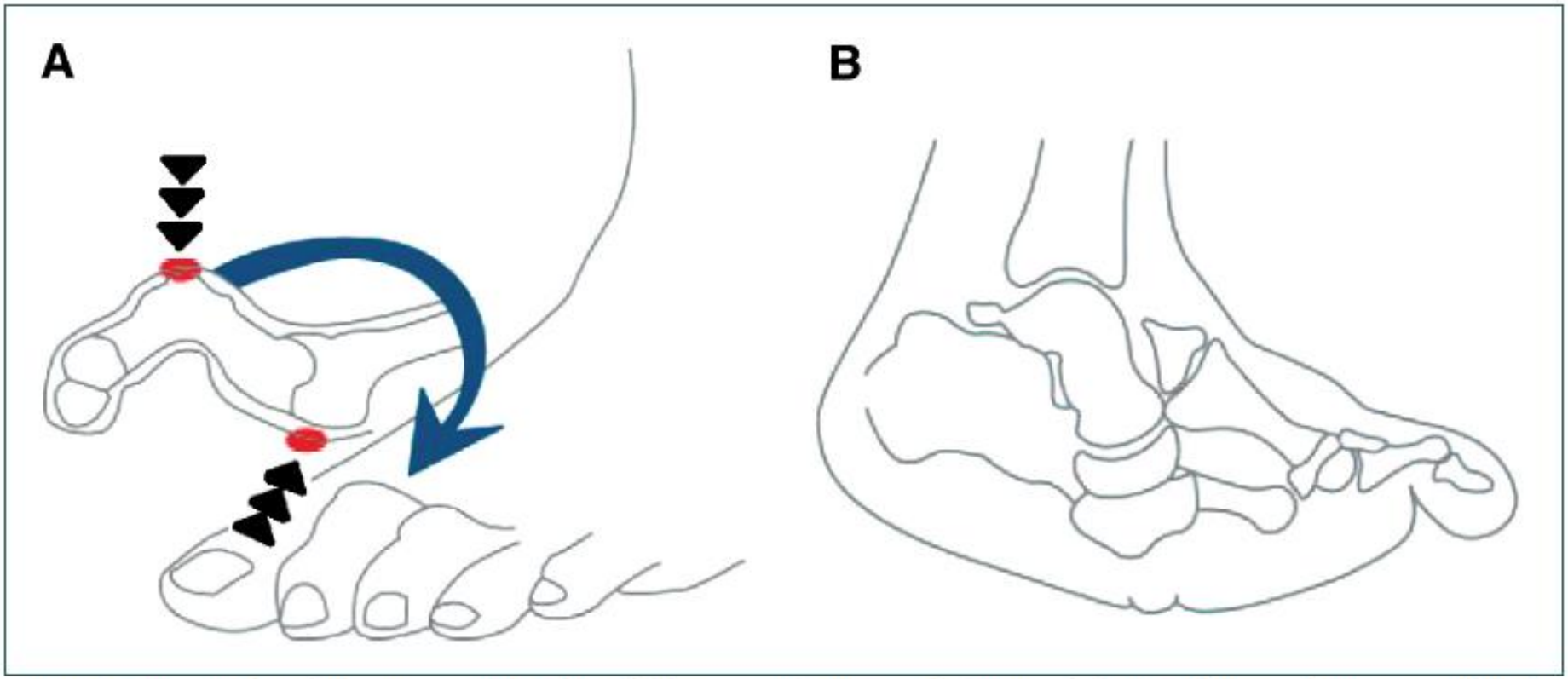


Figure 1. Common foot deformities resulting from diabetes complications: A) claw toe deformity (increased pressure is placed on the dorsal and plantar aspects of the deformity as indicated by the triple arrows); and B) Charcot arthropathy (the rocker-bottom deformity leads to increased pressure on the plantar midfoot). Adapted from Ref. 13.

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Foot deformities

Foot deformities

Foot deformities

PATHOGENESIS/PATHPHYSIOLOGY

PATHOGENESIS/PATHPHYSIOLOGY

- Another mechanism of DFU is arterial insufficiency.
- Peripheral vascular disease, usually in conjunction with minor trauma, may result in a painful, purely ischemic foot ulcer.

Atherosclerosis

Ischemic ulcer

Ischemic ulcer

PATHOGENESIS/PATHPHYSIOLOGY

- Diabetic patients are also prone to infection(fungal or bacterial), and once infection occurs, the healing is impaired.
- Infective ulcers have different characteristics(deep, malodor, purulent, destructive)

Deep osteomyelitis

classification

- Diabetic foot ulcers can be classified based on the underlying mechanisms or ulcer features.
  - Based on the underlying mechanisms
    - Neuropathic
    - Ischemic
    - Infectious
    - Traumatic?
    - Atypical
  - Based on the ulcer features
    - Wagner classification
    - University of Texas classification

The SINBAD system for classifying and scoring foot ulcers

Category	Definition	SINBAD score
Site	Forefoot	0
	Midfoot and hindfoot	1
Ischemia	Pedal blood flow intact: at least one pulse palpable	0
	Clinical evidence of reduced pedal blood flow	1
Neuropathy	Protective sensation intact	0
	Protective sensation lost	1
Bacterial infection	None	0
	Present	1
Area	Ulcer <1cm^2	0
	Ulcer ≥1cm^2	1
Depth	Ulcer confined to skin and subcutaneous tissue	0
	Ulcer reaching muscle, tendon or deeper	1
Total possible score		6

Based on the underlying mechanisms

- **Chronic pressure (neuropathic)**
  - Painless/callus/ pulse+/pressure site/pink
- **Ischemic**
  - Painful/Bulla/Pulseless/Tip of fingers/red
- **Infectious**
  - below or between toes/purulent/deep ulcers
- **Traumatic**
  - Every location and shape
- **Atypical**
  - Unexpected location, presentation or behavior

Neuropathic ulcer

Neuropathic ulcer

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Neuropathic ulcer

Neuropathic ulcer

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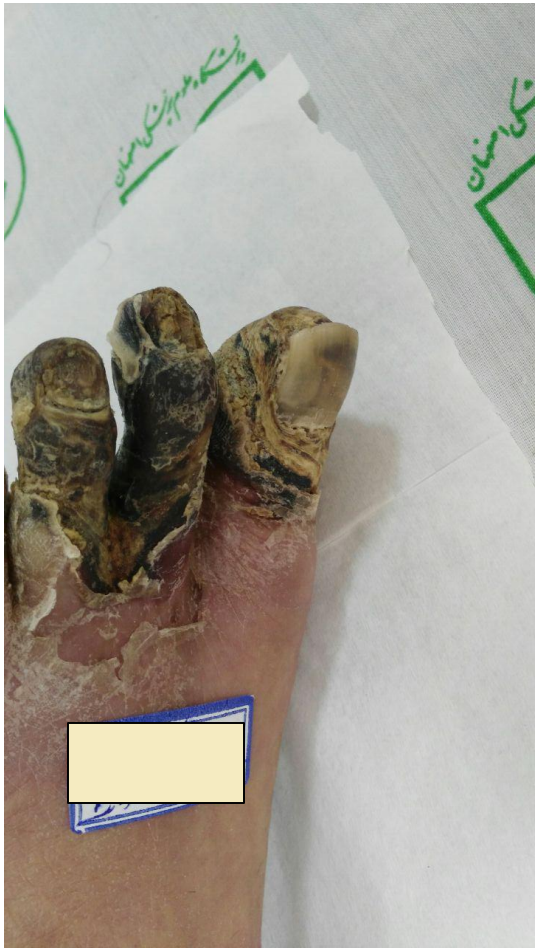
Neuropathic ulcer on charcot foot

Ischemic ulcer

Ischemic ulcer



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Ischemic ulcer

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INFECTIOUS ULCERS

INFECTIOUS ULCERS

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Infectious ulcers

Infectious ulcers

Infectious ulcers

Infectious ulcers

Primary infectious ulcer

Atypical ulcers

Atypical ulcers

Traumatic DFU

- **Traumatic DFUs are very prevalent and have different characteristics.**
- **They may mimic other types of DFUs.**
- **They can be diagnosed by history and clinical examination.**

- Mechanical
- Heat
- Cold
- Chemical
- Others

Traumatic ulcer

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Traumatic DFU

Traumatic DFU

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Traumatic DFU

Traumatic DFU

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13:14:38

Traumatic / infectious

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Traumatic / infectious

Traumatic / infectious

Charcot foot

Charcot foot

Charcot foot

Charcot foot

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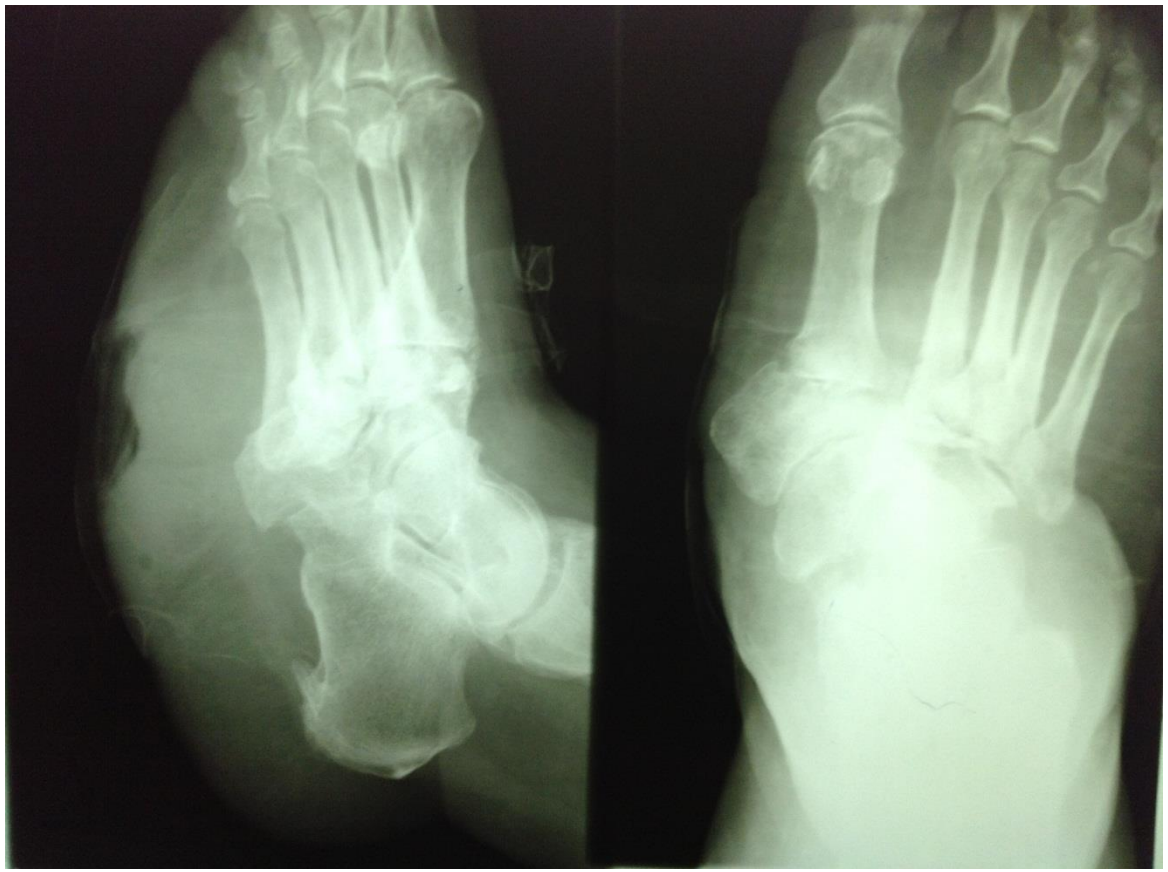
Charcot foot



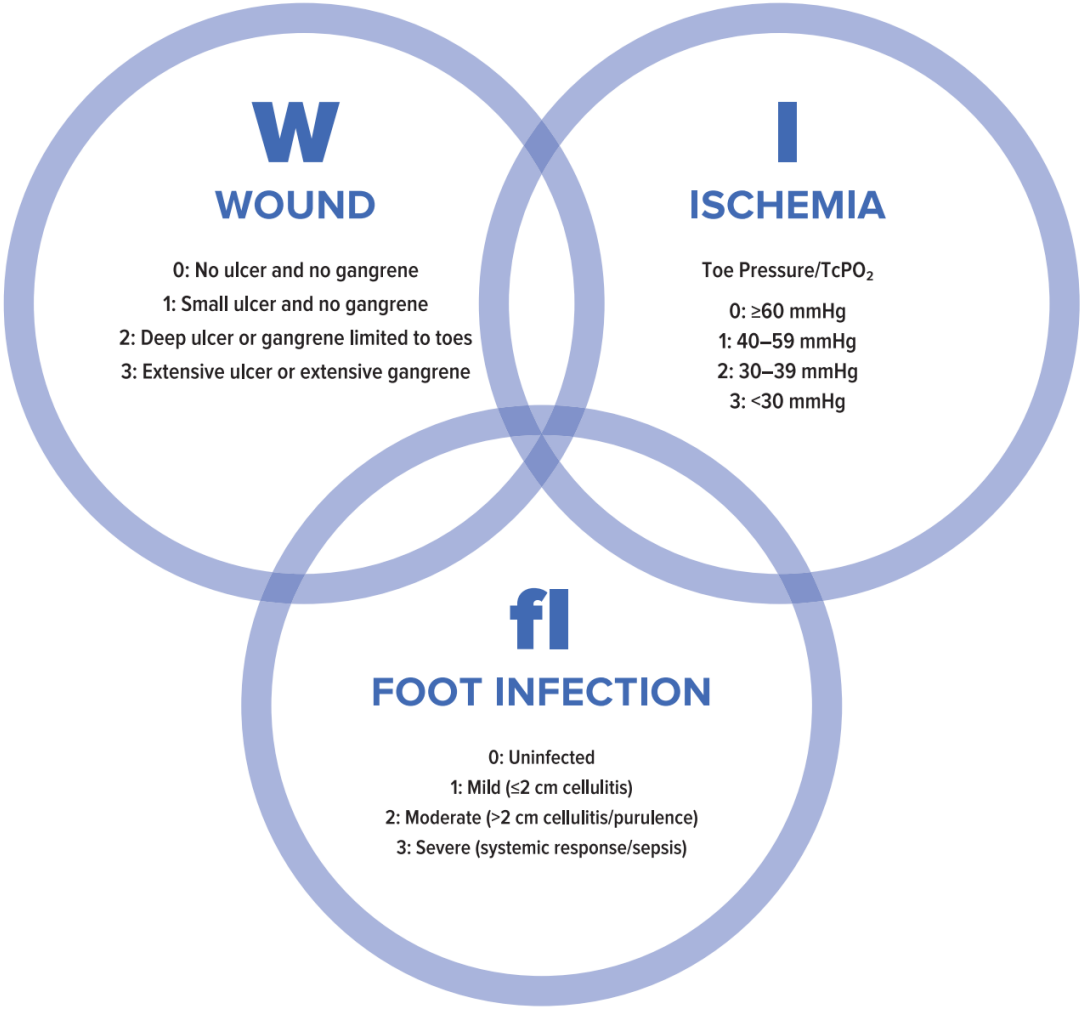
WAGNER DIABETIC FOOT ULCER CLASSIFICATION SYSTEM

Grade	Description
0	No ulcer, but high-risk foot (e.g., deformity, callus, insensitivity)
1	Superficial full-thickness ulcer
2	Deeper ulcer, penetrating tendons, no bone involvement
3	Deeper ulcer with bone involvement, osteitis
4	Partial gangrene (e.g., toes, forefoot)
5	Gangrene of whole foot

UNIVERSITY OF TEXAS WOUND CLASSIFICATION SYSTEM

Stage	Grade 0	Grade 1	Grade 2	Grade 3
A	Preulcer or postulcer lesion No skin break	Superficial ulcer	Deep ulcer to tendon or capsule	Wound penetrating bone or joint
B	+ Infection	+ Infection	+ Infection	+ Infection
C	+ Ischemia	+ Ischemia	+ Ischemia	+ Ischemia
D	+ Infection and ischemia	+ Infection and ischemia	+ Infection and ischemia	+ Infection and ischemia

SVS WIfI classification system



Transcutaneous oxygen pressure measurement

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SVS Threatened Limb Classification System, With Clinical Stages 1–4 Based on Severity of Wound, Ischemia, and foot Infection (WIfI)

	ISCHEMIA: 0				ISCHEMIA: 1				ISCHEMIA: 2				ISCHEMIA: 3			
WOUND: 0	1	1	2	3	1	2	3	4	2	2	3	4	2	3	3	4
WOUND: 1	1	1	2	3	1	2	3	4	2	3	4	4	3	3	4	4
WOUND: 2	2	2	3	4	3	3	4	4	3	4	4	4	4	4	4	4
WOUND: 3	3	3	4	4	4	4	4	4	4	4	4	4	4	4	4	4
	fI: 0	fI: 1	fI: 2	fI: 3	fI: 0	fI: 1	fI: 2	fI: 3	fI: 0	fI: 1	fI: 2	fI: 3	fI: 0	fI: 1	fI: 2	fI: 3

Each of the three WIfI components is graded from 0 to 3. Based on Delphi consensus, the 64 possible combinations were placed into one of four clinical stages based on the estimated baseline risk of amputation. For example, a limb scoring Wound: 1, Ischemia: 3, and foot Infection (fI): 2 would be at high risk for amputation, or clinical Stage 4. Adapted from ref. 7.

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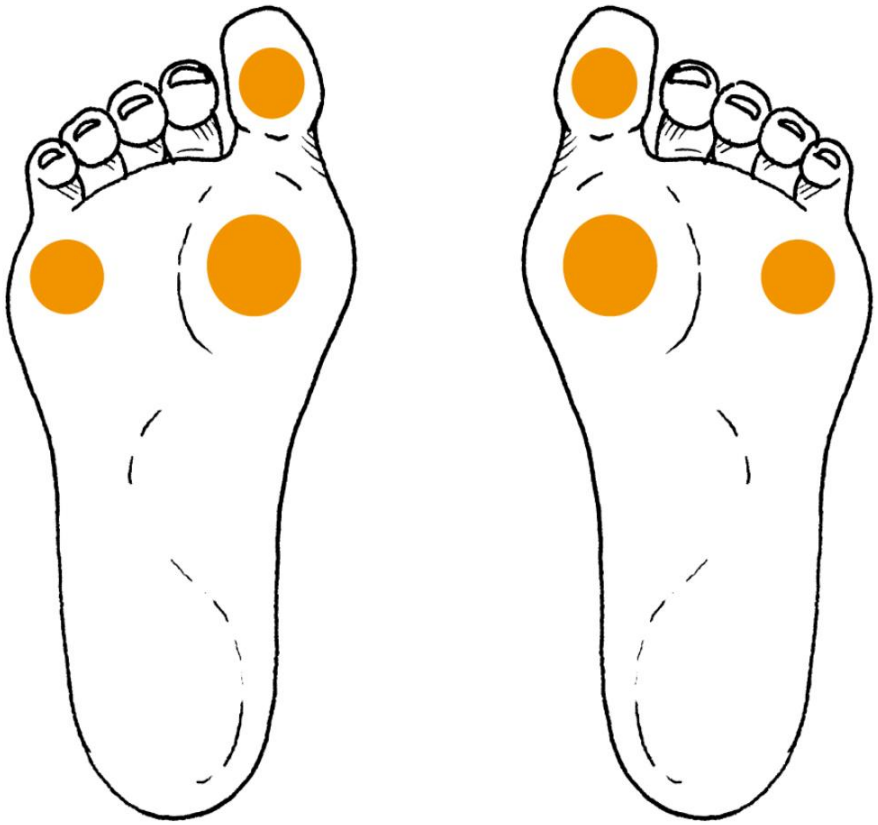
Approach to the patients, limb preservation

- The term “limb preservation” refers to the salvage of a limb that would have otherwise required surgical amputation.
- Limb preservation requires a series of steps including re-establishing adequate perfusion, serial debridement, appropriate wound coverage, aggressive infection management, and correction of underlying biomechanical abnormalities.

Approach to the patients

- The role of team/ multidisciplinary approach
- History and physical examination
- Neurologic evaluation
- Vascular evaluation
- Bacteriologic investigation
- Imaging
- Others

Sites that should be tested for loss of protective sensation with the 10g Semmes-Weinstein monofilament



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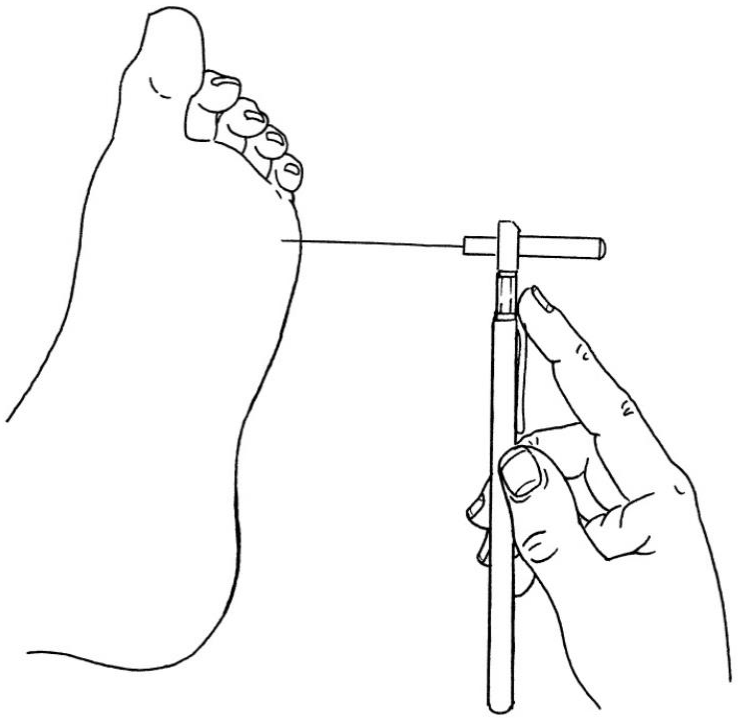
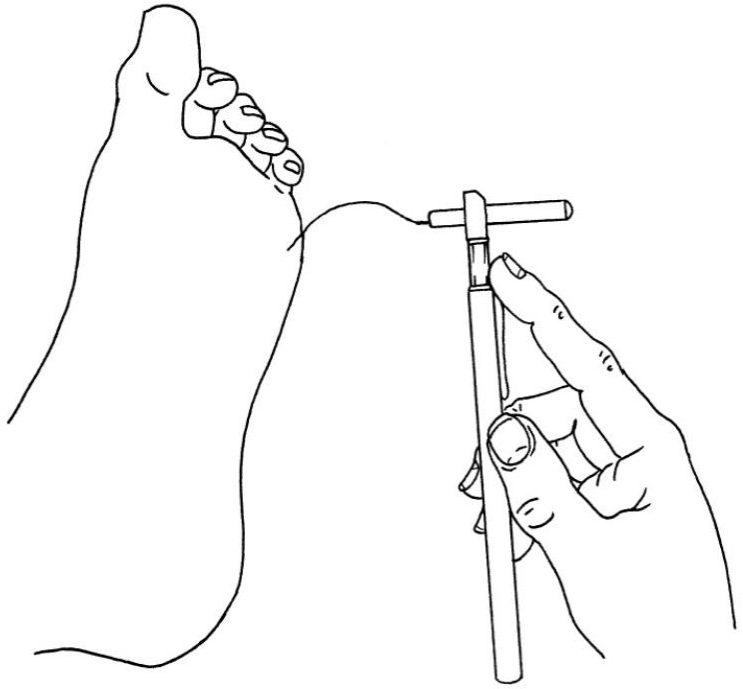
Proper method of using the 10g Semmes-Weinstein monofilament





Foot screening sheet for clinical examination

Presence of a full thickness ulcer Risk factors for foot ulceration	Yes / No
Peripheral neuropathy (one or more of the following tests)	
- Protective sensation (monofilament) undetectable	Yes / No
- Vibration (128 Hz tuning fork) undetectable	Yes / No
- Light touch (Ipswich touch test) undetectable	Yes / No
Foot pulses	
- Posterior tibial artery absent	Yes / No
- Dorsal pedal artery absent	Yes / No
Other	
- Foot deformity or excessive bony prominences	Yes / No
- Limited joint mobility	Yes / No
- Signs of abnormal pressure, such as callus	Yes / No
- Ruddy discoloration on dependency	Yes / No
- Poor foot hygiene	Yes / No
- Inappropriate footwear	Yes / No
- Previous ulcer	Yes / No
- Lower extremity amputation	Yes / No

Imaging

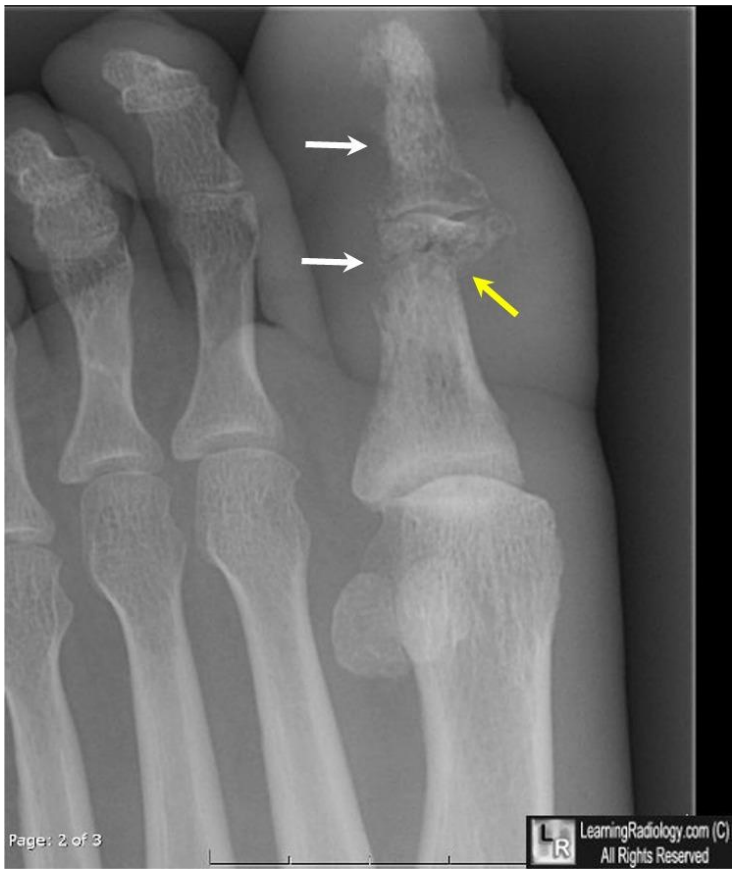
- Plain X rays
- Ultrasound
- Doppler Ultrasonography
- MRI
- CT scan
- Radioisotope scan/WBC scan

Features characteristic of diabetic foot osteomyelitis on plain X-rays

- New or evolving radiographic features* on serial radiographs**, including:
  - Loss of bone cortex, with bony erosion or demineralization
  - Focal loss of trabecular pattern or marrow radiolucency (demineralization)
  - Periosteal reaction or elevation
  - Bone sclerosis, with or without erosion
- Abnormal soft tissue density in the subcutaneous fat, or gas density, extending from skin towards underlying bone, suggesting a deep ulcer or sinus tract.
- Presence of sequestrum: devitalized bone with radiodense appearance separated from normal bone
- Presence of involucrum*: layer of new bone growth outside previously existing bone resulting and originating from stripping off the periosteum.
- Presence of cloacae*: opening in the involucrum or cortex through which sequestrum or granulation tissue may discharge.

Osteomyelitis

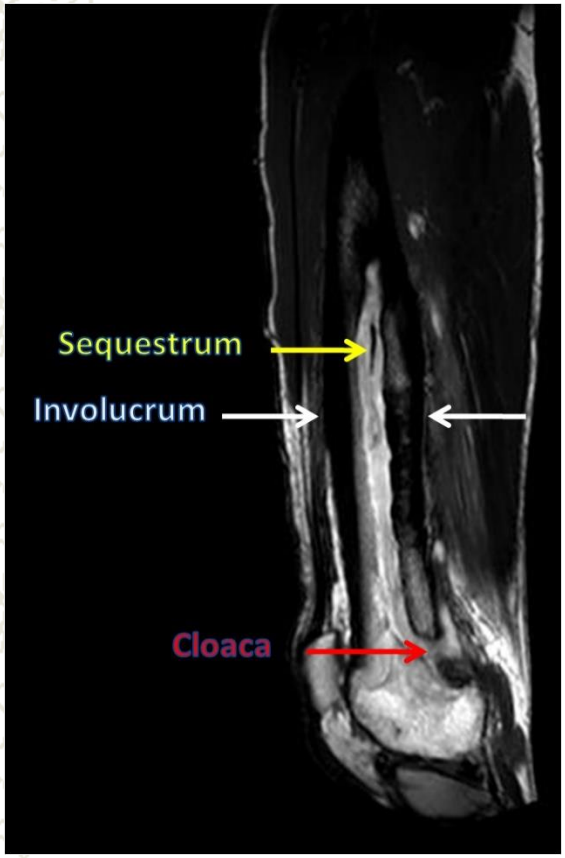
- Bone destruction of proximal and terminal phalanges of great toe with destruction of the cortex characteristic of osteomyelitis (white arrows).
- There is also a pathologic fracture through the proximal phalanx (yellow arrow)

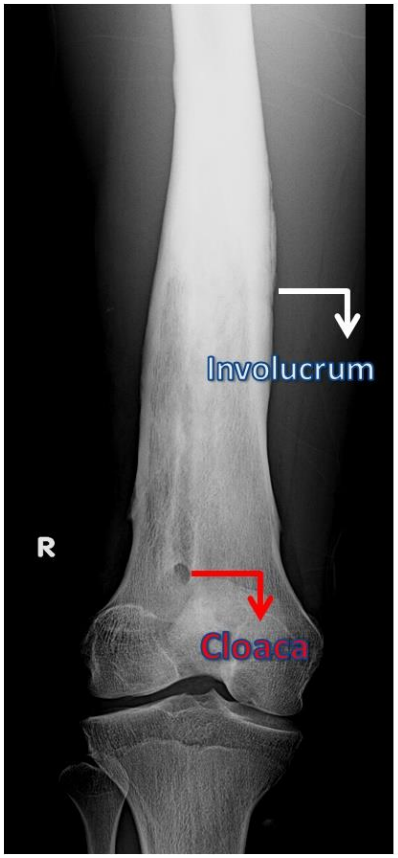


Osteomyelitis

- Bone destruction of head of 2nd metatarsal with periosteal new bone formation characteristic of osteomyelitis

Osteomyelitis





Modified ADA Diabetic Foot Risk Classification

Priority	Indications	Timeline	Suggested Follow-up
URGENT (active pathology)	▸ Open wound or ulcerative area, with or without signs of infection ▸ New neuropathic pain or pain at rest ▸ Signs of active Charcot deformity (red, hot, swollen midfoot or ankle) ▸ Vascular compromise (sudden absent DP/PT pulses or gangrene)	Immediate referral/ consultation	As determined by specialist
HIGH (ADA risk category 3: the diabetic foot in remission)	▸ Presence of diabetes with a previous history of ulcer or lower-extremity amputation ▸ Chronic venous insufficiency (skin color change or temperature difference)	Immediate or "next available" outpatient referral	Every 1–2 months
MODERATE (ADA risk category 2)	▸ PAD ± LOPS ▸ DP/PT pulses diminished ▸ Presence of swelling or edema	Referral within 1–3 weeks (if not already receiving regular care)	Every 2–3 months
LOW (ADA risk category 1)	▸ LOPS ± longstanding, nonchanging deformity ▸ Patient requires prescriptive or accommodative footwear	Referral within 1 month	Every 4–6 months
VERY LOW (ADA risk category 0)	▸ No LOPS or PAD ▸ Patient seeks education on topics such as routine foot care, athletic training, appropriate footwear, or injury prevention	Referral within 1–3 months	At least annually for all people with diabetes

DP, dorsalis pedis; LOPS, loss of protective sensation; PT, posterior tibial. Modified from Diabetes Care 2008;31:1679–1685 (ref. 6), with permission from the American Diabetes Association, ©2008.

Approach to the patients indications of urgent admission

- Sepsis or moderate to severe infection
- Sever ischemia or gangrene
- Exposed bone or joint
- Bad clinical background
- Unstable situation

role of The team/ multidisciplinary approach

Principles of ulcer treatment

- *Stop ongoing trauma(Relief of pressure and protection of the ulcer)*
- *Restoration of skin perfusion*
- *Treatment of infection*
- *Local wound care*
- *Metabolic control and treatment of comorbidity*
- *Education for patient and relatives*

Relief of pressure and protection of the ulcer

- **Stop ongoing trauma(heat, cold, pressure, etc.)**
- Mechanical off-loading
- Total contact casting or other casting techniques
- Temporary footwear
- Individually moulded insoles and fitted shoes
- Non-weight bearing
  - limitation of standing and walking
  - crutches, etc

Off loading

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TCC application

Off loading shoes, ortho wedge, boot

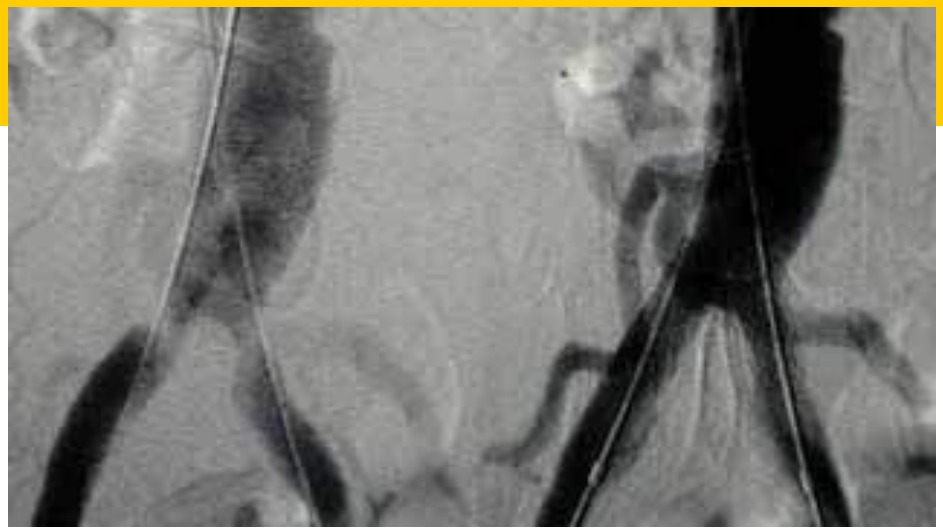
Restoration of skin perfusion

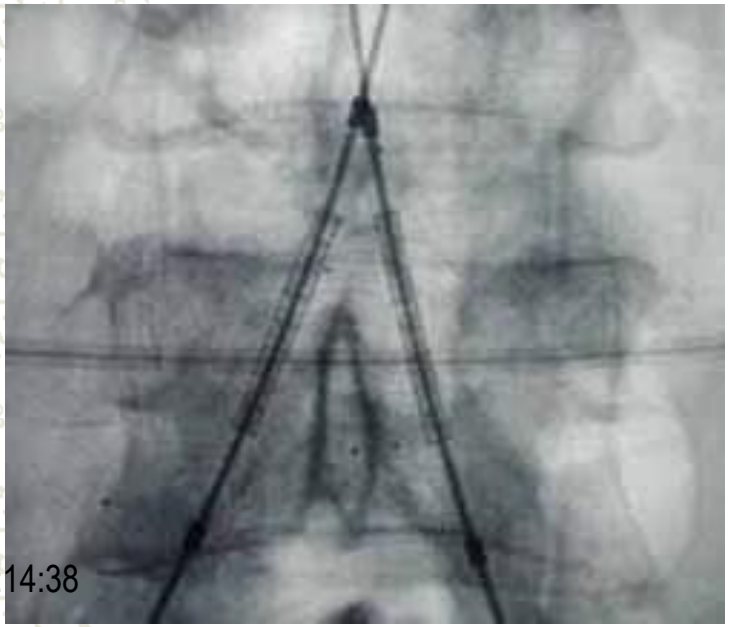
- Arterial revascularization procedures: results do not differ from people without diabetes, but distal revascularization procedures (angioplasty or bypass-surgery) are needed more frequently.
- The benefits of pharmacological treatment to improve perfusion have not been established.
- Emphasis should be placed on cardiovascular risk reduction (cessation of smoking, treatment of hypertension and dyslipidaemia, use of aspirin).

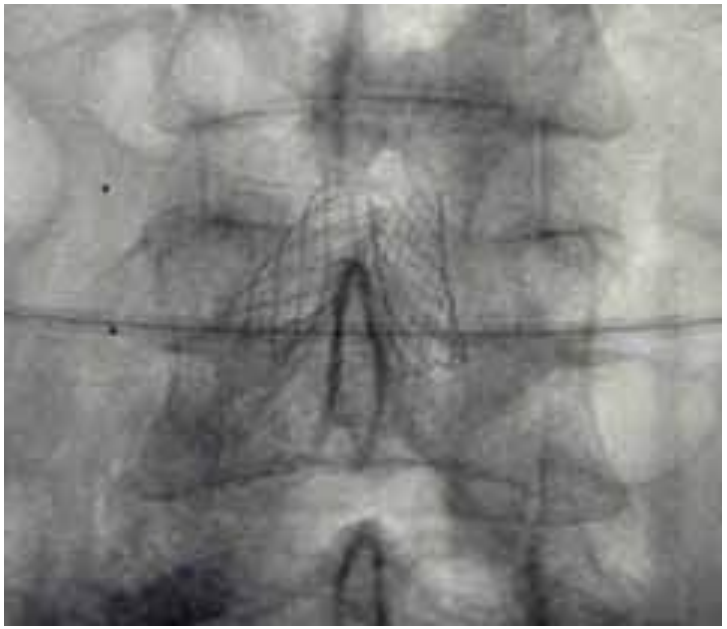
Diagnostic Interpretation of Ankle- Brachial Index

Resting ABI	Severity
0.91-1.30	Normal
0.70-0.90	Mild obstruction
0.40-0.69	Moderate obstruction
< 0.40	Severe obstruction
> 1.3	Poorly compressible

Stenting







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Treatment of infection

Clinical description	IDSA	IWGDF
Wound without purulence or any manifestations of inflammation	Uninfected	1
≥2 manifestations of inflammation (purulence or erythema, pain, tenderness, warmth, or induration); any cellulitis or erythema extends ≤2 cm around ulcer, and infection is limited to skin or superficial subcutaneous tissues; no local complications or systemic illness	Mild	2
Infection in a patient who is systemically well and metabolically stable but has ≥2 cm; lymphangitis; spread beneath fascia; deep tissue abscess; gangrene; muscle, tendon, joint, or bone involvement	Moderate	3
Patient who demonstrates signs of systemic inflammatory response syndrome manifested by ≥ of the following: Temperature >38°C or <36°C Heart rate >90 bpm Respiratory rate >20 breaths/minute or $PaCO_2$ <32 mmHG White blood cell count >12,000 or <4,000 cells/microliter or ≥10% immature cells (bands)	Severe	4
Systemic toxicity may also present with anorexia, chills, hypotension, confusion, vomiting, acidosis, hyperglycemia, and/or azotemia. The presence of critical limb ischemia may increase the level of severity.		

Diabetic foot infection classification system according to the IDSA and International Working Group on the Diabetic Foot (IWGDF). The two classifications are virtually identical. Adapted from Lipsky et al. (11).

Treatment of infection

- Superficial ulcer with skin infection
  - debridement with removal of all necrotic tissue and oral antibiotics targeted at Staphylococcus aureus and streptococci
- Deep (limb-threatening) infection
  - surgical drainage as soon as possible (emergency referral) with removal of necrotic or poorly vascularized tissue, including infected bone
  - revascularization if necessary
  - broad-spectrum antibiotics intravenously, aimed at Gram-positive and negative micro-organisms, including anaerobes

Local wound care

- Management of ulcer base
- Management of ulcer edge/debridement
- Role of alternative tissues
- Role of negative pressure wound healing (VAC)
- Role of hyperbaric oxygen
- Ozone therapy

Wound dressing

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Local wound care debridement

- Wound bed preparation provides clinicians with a comprehensive approach for removing the barriers to healing, thereby stimulating the growth of new tissue and wound closure.
- The first step is aggressive debridement through the use of surgical, autolytic, mechanical, or biological methods to remove all necrotic tissue, slough, and firm eschar, since each of these impede healing.

Local wound care debridement

- Debridement may also promote healing by creating a clean wound surface free of senescent cells and biofilms, which shield bacterial colonies and may make them more resistant to infection management.

Methods of wound debridement

Surgical and sharp using scalpel and scissors. Highly selective with rapid results. Should only be undertaken by a skilled practitioner.

Mechanical such as hydrotherapy and wound irrigation. Rehydration can ease removal of the surface eschar and removes surface debris. However, these are relatively slow techniques and there is little evidence to support their use. Potential for cross infection needs to be considered if using hydrotherapy. There is also a theoretical risk of fluid embolism and promotion of infection if irrigation is too vigorous.

Autolytic using hydrocolloids and hydrogels. Rehydration of necrotic tissue through the use of a hydrogel or by keeping the wound moist, and removal of devitalised tissue using the body's own enzymes. This method is in common use but prolongs the time needed for debridement.

Enzymatic using preparations such as streptokinase or streptodornase or bacterial-derived collagenases. Streptokinase and streptodornase aim to break down and rehydrate necrotic tissue, but despite being available for more than 30 years, there is little evidence to support their use over alternative methods. Also, the need to score the eschar before application may increase the risk of damage. Bacterial-derived collagenases show great potential and may promote healing.

Biological such as maggot therapy. The larvae of Lucilia sericata (greenbottle fly) digest necrotic tissue and pathogens. This technique is rapid and selective, although much of the evidence to support its use is derived from anecdotal reports.

Chemical such as hypochlorite. No longer widely used as application can be painful and underlying tissue is damaged.

Local wound care debridement

Debridement Maggot Therapy

Larvae of *Phaenicia (Lucilia) sericata*

Live maggots

Our experiments

- تجربه عملی ما در لارو درمانی از سال 1391 شروع شد.
- در آن زمان مهمترین چالش ما پیدا کردن منبع قابل اعتماد لارو مگس فنیشیا سریکاتا بود.
- بررسی ها نشان داد یکی از اساتید حشره شناسی به نام دکتر میراب زاده موفق به تولید آزمایشگاهی لارو این حشره شده اند.
- با همکاری تیم ایشان ما موفق به شروع عملی لارو درمانی در اصفهان شدیم.
- با پیوستن دو نفر از دانشجویان پزشکی در قالب پایان نامه دانشجوئی، این روند تسریع شد.
- حتی وقتی از جنس و گونه لارو اطمینان داشتیم نیز گاهی تا چند هفته هیچ گونه لاروی به دست ما نمی رسید.
- تلاش اولیه ما برای تولید لارو این حشره در اصفهان با شکست روبرو شد.
- با قطع همکاری تیم دکتر میراب زاده مجبور به جایگزینی همکارانی از دانشگاه تهران شدیم ولی متاسفانه لارو

Our experiments

Our experiments

Our experiments

Our experiments

Our experiments














Maggot therapy

- تاکنون بیش از دویست بیمار تحت لارو درمانی قرارگرفته اند.
- از این بیماران 42 نفر تحت معیارهای زخمهای آتیپیک وارد مطالعه پایان نامه شده اند و نتایج سایر بیماران نیز ثبت شده است.

V.A.C THERAPY

Local wound care

Common goals of negative pressure wound therapy

- • Promote rapid reduction in wound volume
- • Promote growth of granulation tissue and contraction of wound edges
- • Manage exudate
- • Prepare the wound bed for transition to another treatment modality such as MWH, surgical closure, or a flap or graft
- • Reduce bioburden

LOCAL WOUND CARE

COMMON GOALS OF NEGATIVE PRESSURE WOUND THERAPY

- • Decrease hospital stay length
- • Decrease morbidity and mortality
- • Decrease frequency of dressing change
- • Prevent deterioration of the wound
- • Minimize contamination and wound odor by providing a temporary barrier
- • Improve quality of life

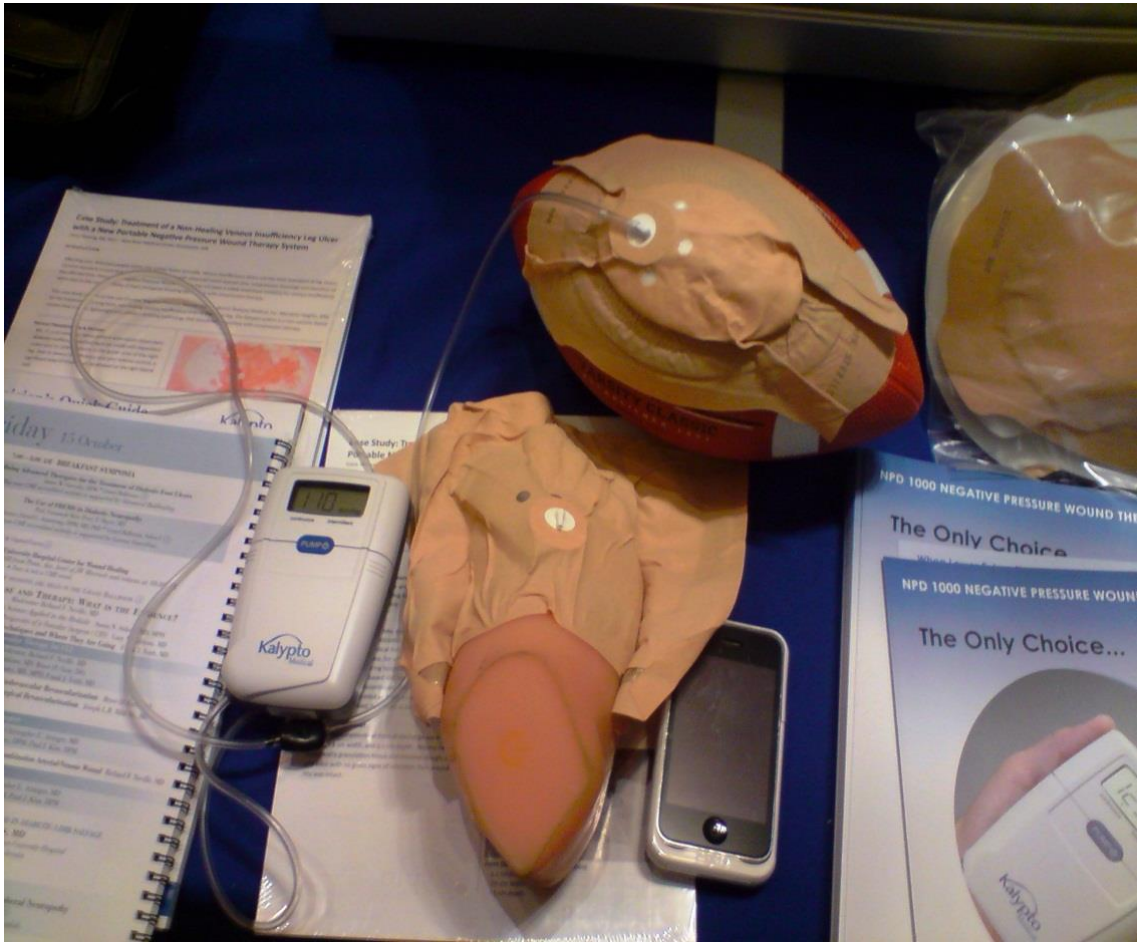
Local wound care, NPWT Contraindications

- Do not use NPWT in wounds where there is evidence of the following:
- • Exposed vital organs
- • Inadequate debridement of the wound
- • Untreated osteomyelitis or sepsis within the vicinity of the wound
- • Untreated coagulopathy
- • Necrotic tissue with eschar
- • Malignancy in the wound
- • Allergy to any component required for the procedure

Local wound care, NPWT Precautions

- Reasons to use NPWT with caution:
- • Active bleeding or a risk of bleeding (eg, there is difficultly achieving wound hemostasis, patient is taking anticoagulants)
- • An exposed blood vessel close to the wound
- • Difficulty maintaining a seal
- • Uncontrolled pain
- • Evidence of previous patient noncompliance with or intolerance to the procedure

Foundational VAC therapy



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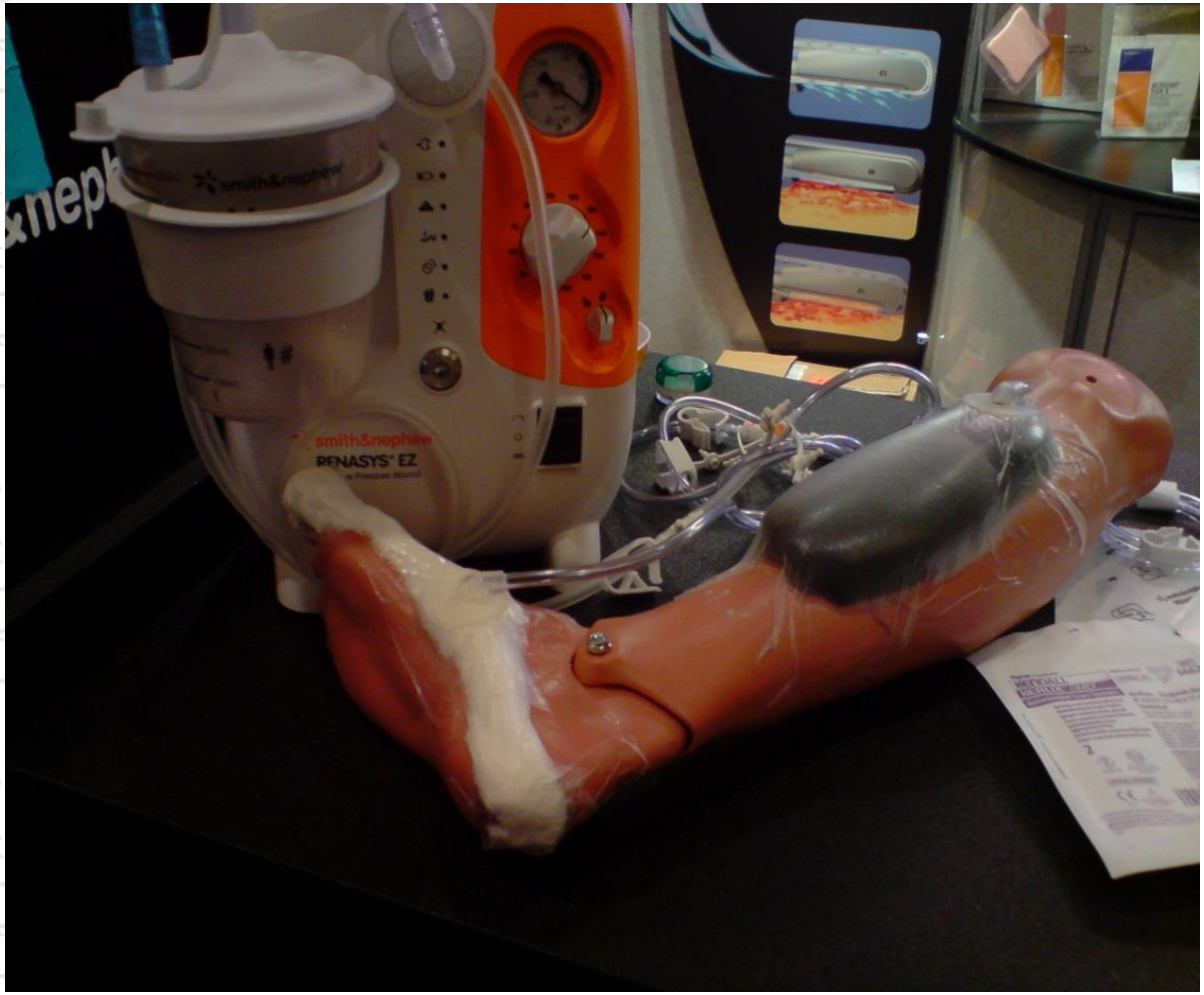
Foundational VAC therapy



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Vacuum therapy

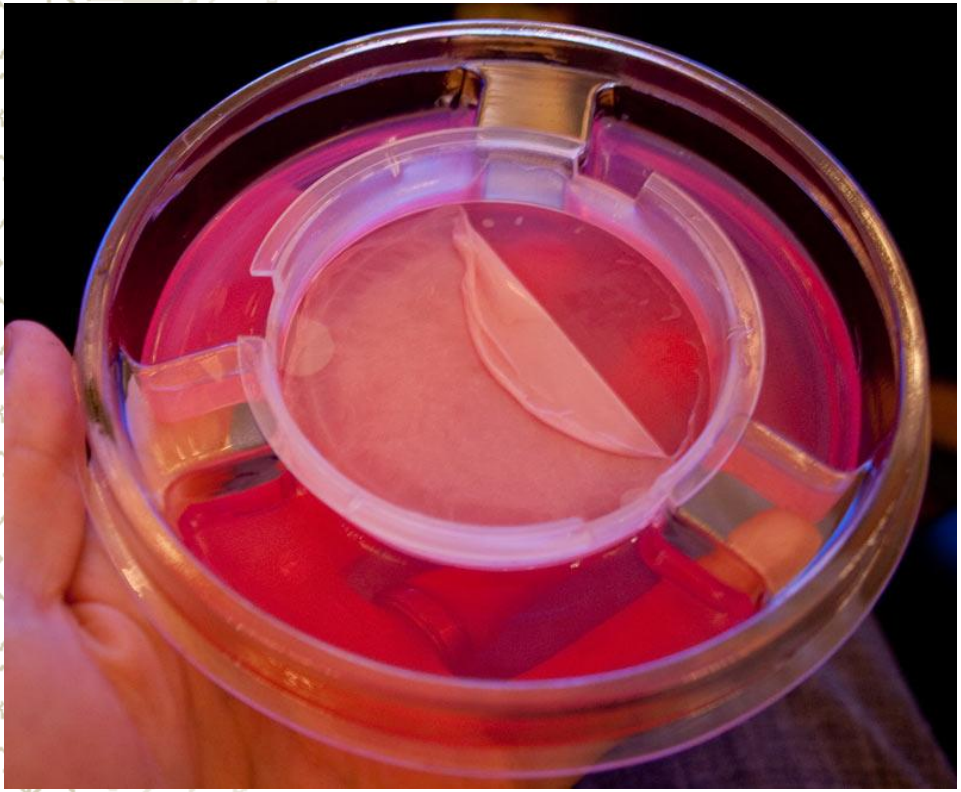
Metabolic control and treatment of comorbidity

- Glucose management
- Cardiovascular management
  - **Lipid management**
  - **Blood pressure management**
- Kidney management
- Others

Converting a Complex Wound to a Simple Wound: The Role of Extracellular Matrix

- Growth factors / cytokine topicals
  - VGEF
  - regranex
- Bio active wound adjuncts
  - Integra
  - Gamma graft
- Living tissues
  - Apligraft
  - dermagraft

Bioengineered alternatives Tissue



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Cryopreserved amniotic membrane

Hyperbaric Oxygen Therapy

- Patients undergoing HBO therapy enter a chamber filled with 100 percent oxygen atmosphere pressurized to 2.0 to 2.5 ATA (atmospheric pressure absolute), which is equivalent to the pressure 33 to 48 feet below sea level.
- Hyperbaric oxygen therapy treatment “force feeds” oxygen through the lungs to the rest of the body.
- A 90-minute treatment in this hyperbaric environment induces “hyperoxygenation” with oxygen levels over 10 times the normal amount in the bloodstream.

Interestingly, oxygen under hyperbaric conditions "behaves as drugs" and hyper-oxygenation causes:

- • a decrease in leg edema and excessive inflammation
- • an increase in the growth factors and receptors (VEGF and PDGF)
- • doubled flexibility of red blood cells
- • increase in bactericidal capacity
- • mobilization of the stem cell within the bone marrow to increase the circulating progenitor cells within the blood stream eightfold

Hyperbaric oxygen chamber

Guidelines For HBO In Diabetic Foot Wounds

- • Presence of diabetes and lower extremity wounds
- • Thirty days of standard wound care have shown no improvement
- • Wounds must be Wagner Grade III (deep wound with abscess, osteomyelitis or tendonitis extending to those structures), Wagner Grade IV (gangrenous toes and forefoot) or V (gangrenous foot).

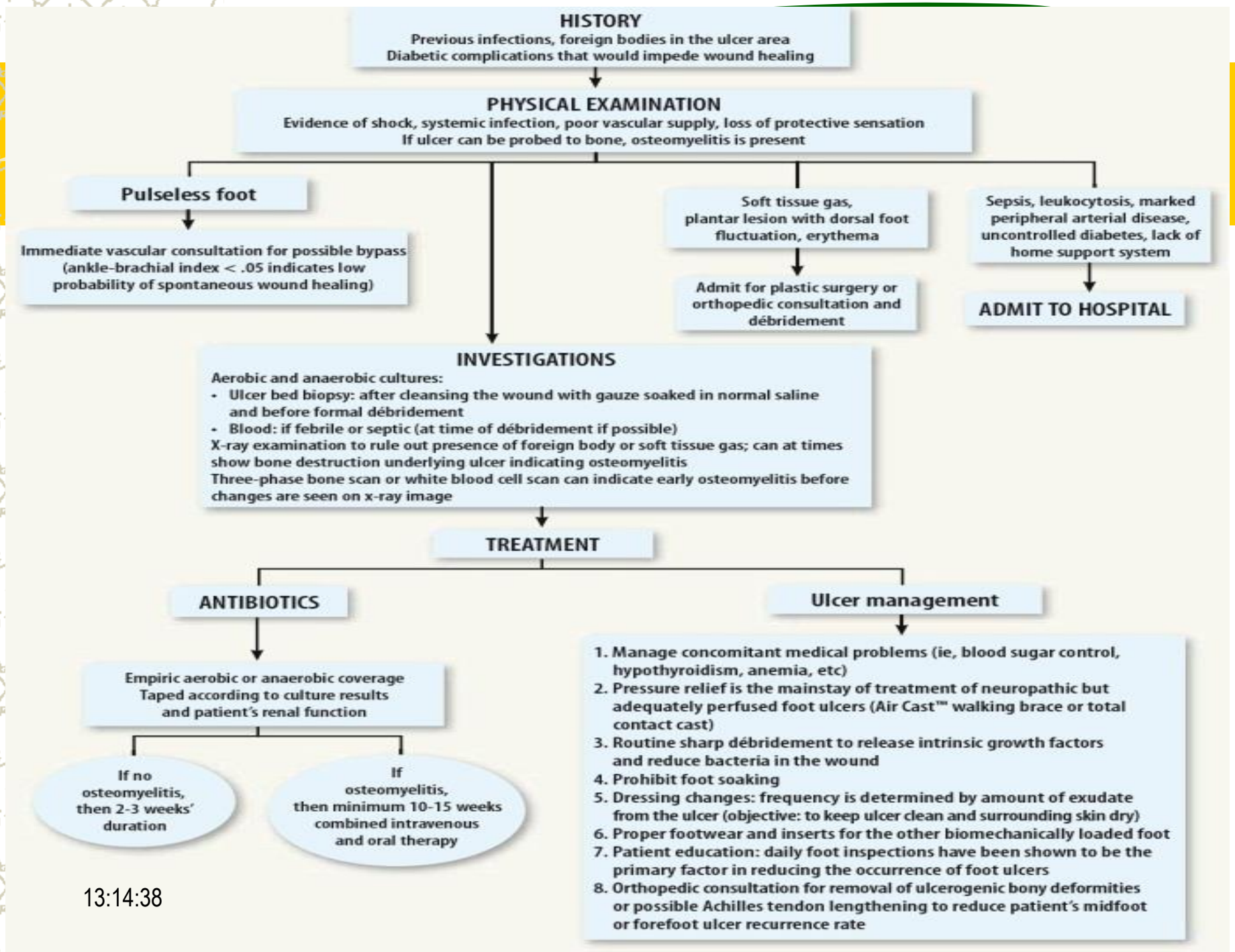
complications

- The risk versus benefit ratio of HBO is remarkably favorable.
- The most common side effect of HBO is otic barotrauma
- Claustrophobia
- No fire accidents or fatalities have been reported in the United States with medical HBO chambers.

Conclusion

Questions to ask

- Is there any infection?
- Is there ischemia?
- Is there excess necrotic tissue?
- Is there deep tissue loss?
- Is there pressure ulcer?
- Is there deformity?
- Is there atypical wound behaviour?


HISTORY
Previous infections, foreign bodies in the ulcer area
Diabetic complications that would impede wound healing
PHYSICAL EXAMINATION
Evidence of shock, systemic infection, poor vascular supply, loss of protective sensation
If ulcer can be probed to bone, osteomyelitis is present
Pulseless foot
Immediate vascular consultation for possible bypass (ankle-brachial index < .05 indicates low probability of spontaneous wound healing)
Soft tissue gas, plantar lesion with dorsal foot fluctuation, erythema
Admit for plastic surgery or orthopedic consultation and débridement
Sepsis, leukocytosis, marked peripheral arterial disease, uncontrolled diabetes, lack of home support system
ADMIT TO HOSPITAL
INVESTIGATIONS
Aerobic and anaerobic cultures:
- Ulcer bed biopsy: after cleansing the wound with gauze soaked in normal saline and before formal débridement
- Blood: if febrile or septic (at time of débridement if possible)
X-ray examination to rule out presence of foreign body or soft tissue gas; can at times show bone destruction underlying ulcer indicating osteomyelitis
Three-phase bone scan or white blood cell scan can indicate early osteomyelitis before changes are seen on x-ray image
TREATMENT
ANTIBIOTICS
Empiric aerobic or anaerobic coverage
Taped according to culture results and patient's renal function
If no osteomyelitis, then 2-3 weeks' duration
If osteomyelitis, then minimum 10-15 weeks combined intravenous and oral therapy
Ulcer management
1. Manage concomitant medical problems (ie, blood sugar control, hypothyroidism, anemia, etc)
2. Pressure relief is the mainstay of treatment of neuropathic but adequately perfused foot ulcers (Air Cast™ walking brace or total contact cast)
3. Routine sharp débridement to release intrinsic growth factors and reduce bacteria in the wound
4. Prohibit foot soaking
5. Dressing changes: frequency is determined by amount of exudate from the ulcer (objective: to keep ulcer clean and surrounding skin dry)
6. Proper footwear and inserts for the other biomechanically loaded foot
7. Patient education: daily foot inspections have been shown to be the primary factor in reducing the occurrence of foot ulcers
8. Orthopedic consultation for removal of ulcerogenic bony deformities or possible Achilles tendon lengthening to reduce patient's midfoot or forefoot ulcer recurrence rate

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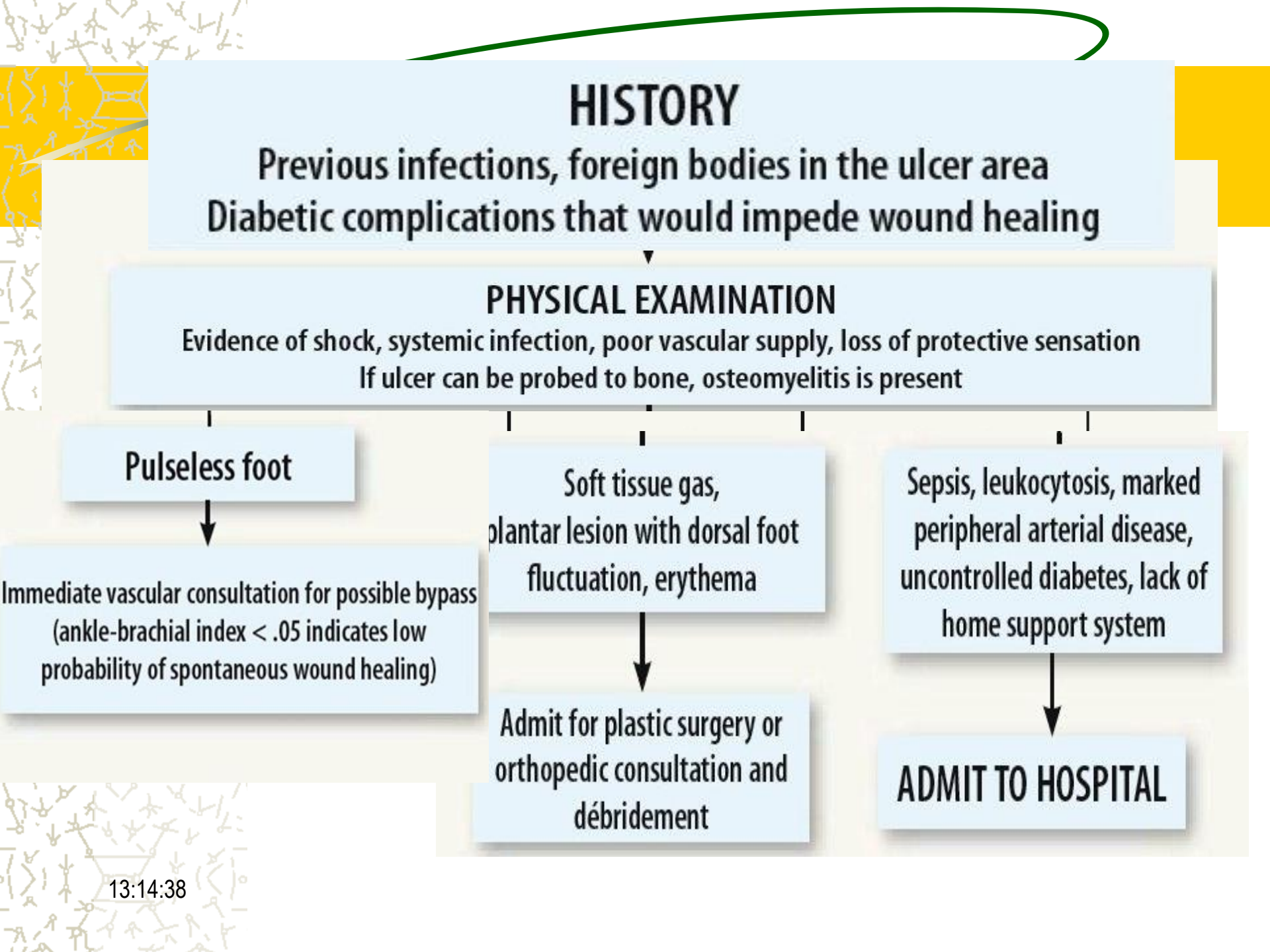
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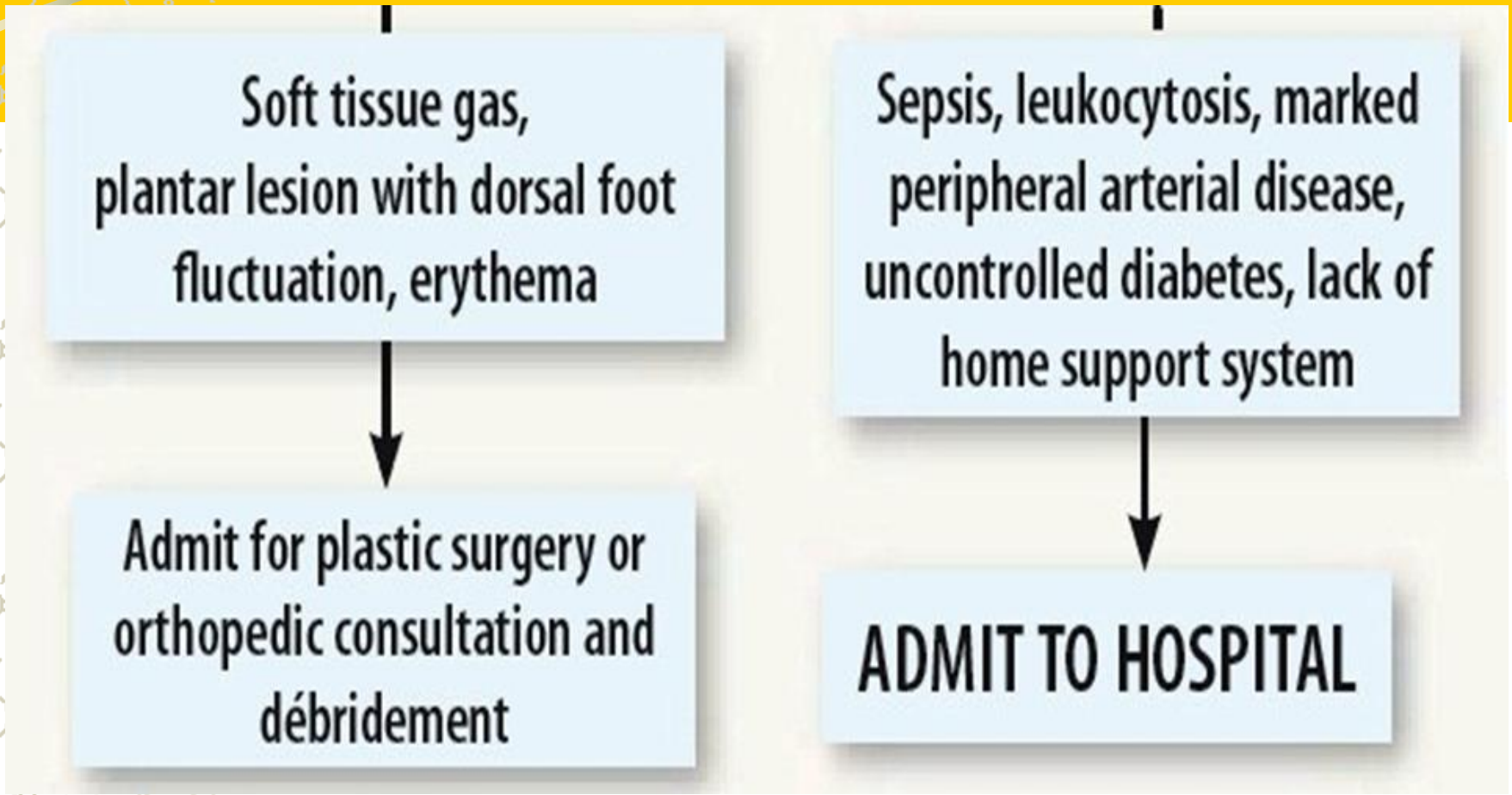
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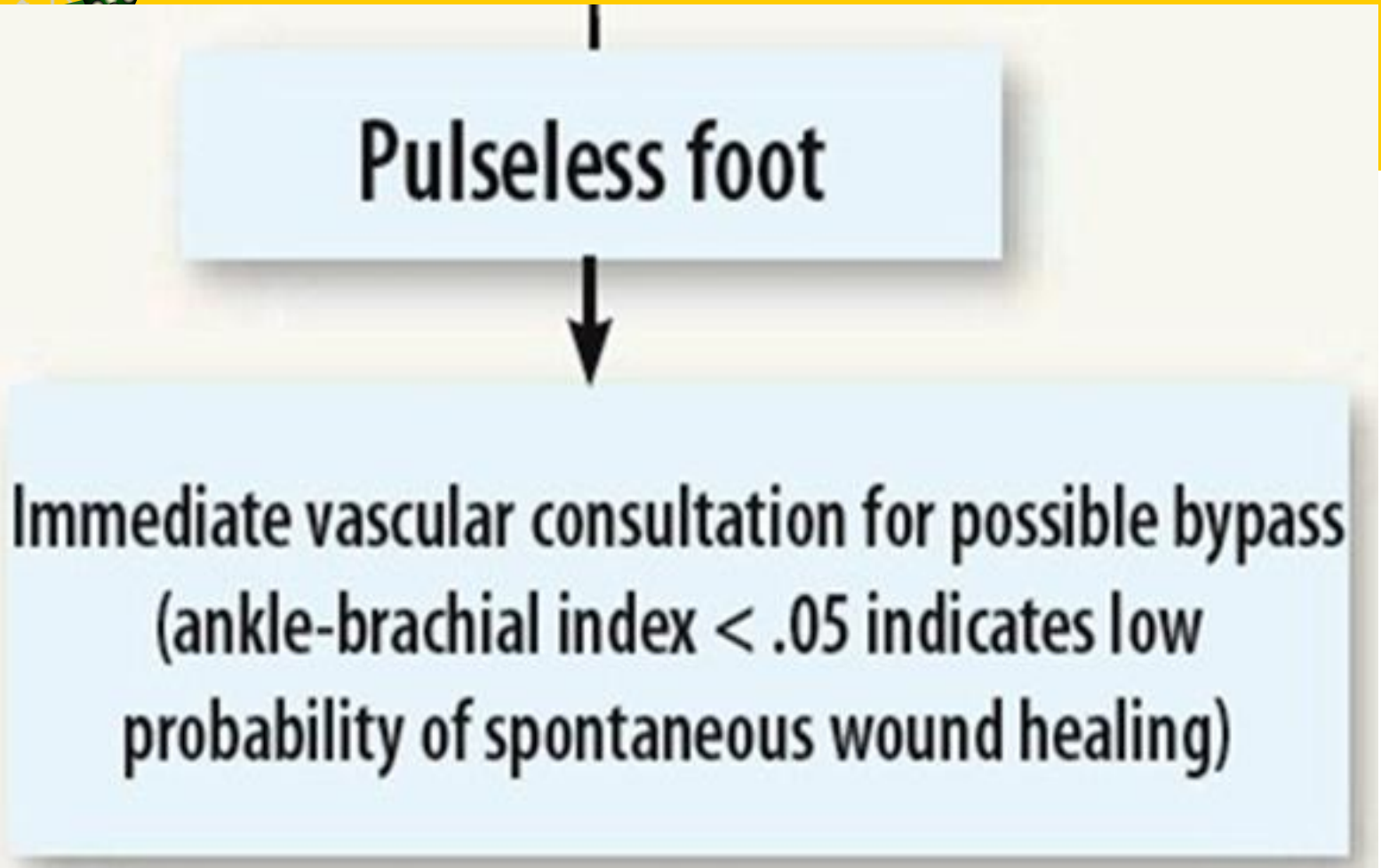
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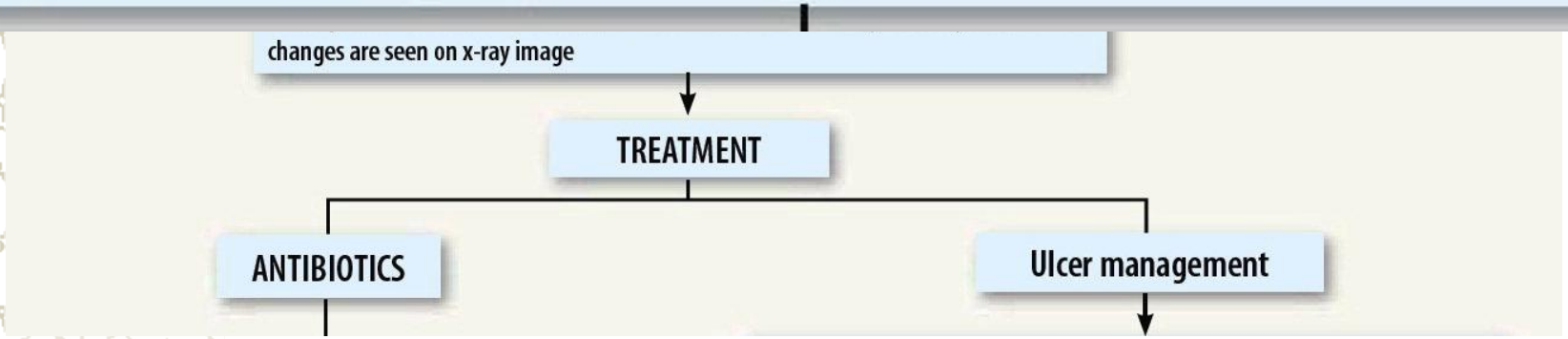

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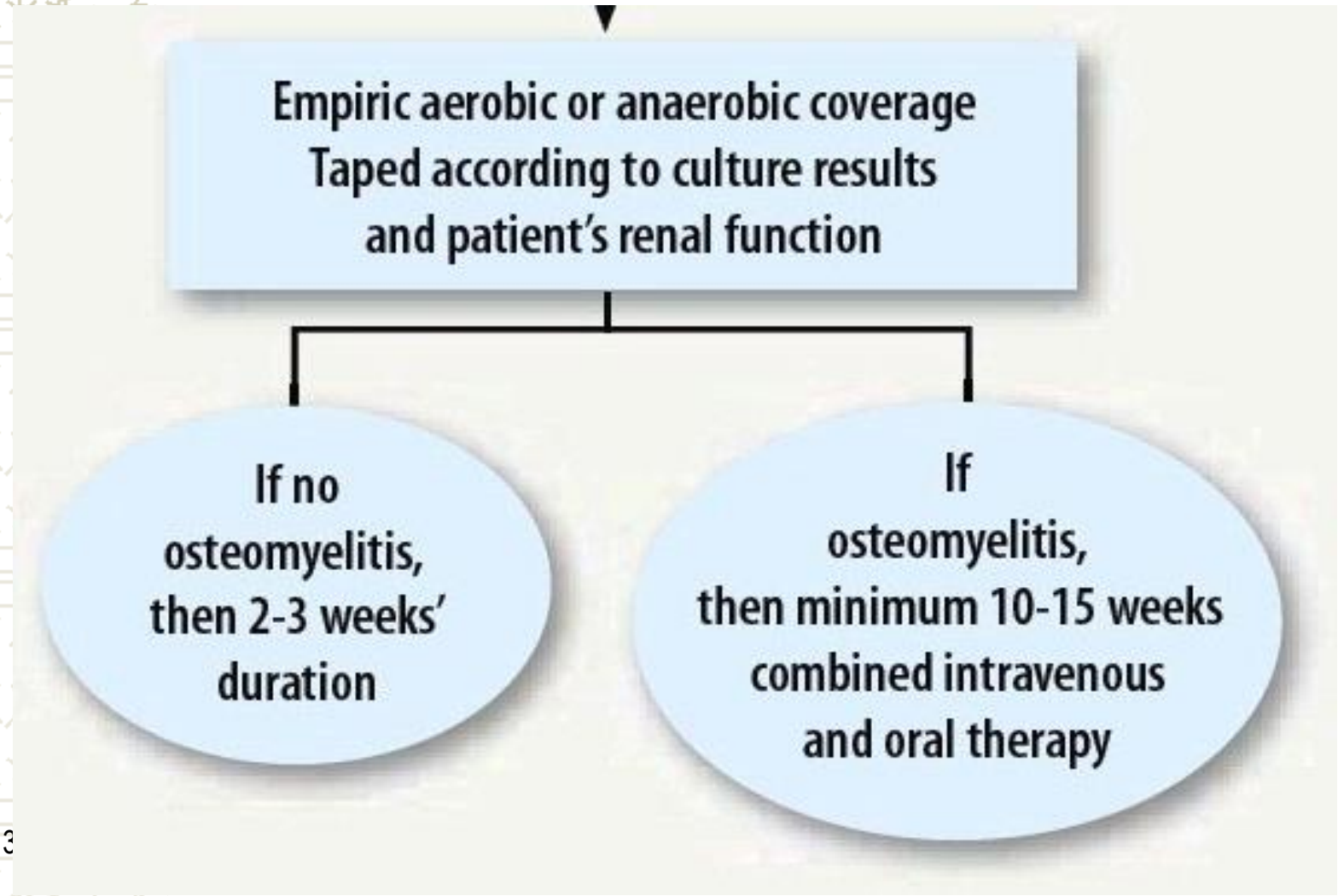
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Some cases

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My recommendation

- Always think to the underlying mechanism of ulcer:
- Ischemic?
  - Pulses present or absent?
- Neuropathic?
  - Usual site or atypical position?
- Infective?
  - Extent of necrosis
  - Collection?
- Traumatic?

In refractory ulcers or unusual presentations:

- Detect uncovered situation:
  - Off loading
  - Vascular supply
  - Infection
  - Debridement
- Consider adjuvant therapy
- Detect atypical ulcers

Thank you and hope for a good rain