Acute conditions →

arterial ischemic ulcer
DF ulcer
Venous ulcer
Bed sore
Pressure ulcer

Wound healing and care

at least
على
2 Q

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Process after injury → restore natural integrity

Occurs mostly in all organ – skin عادة نقصدها عن ال

- Wound healing is the normal body reaction of complex and dynamic processes of replacing devitalized and missing cellular structures and tissue layers in response to tissue injury.

- the ultimate outcome of any healing process is repair of a tissue defect.

Regeneration → Repair By Normal cell Bone, liver

Healing By scar tissue 99% of cases.

Goals of wound healing :

1- Hemostases (stop further blood loss) TCP priority / Initial + Most Imp Step
2- restore normal function and structure
3- reform barriers to fluid loss and infection
4- limit further entry of foreign organisms and material,
5- re-establish normal blood and lymphatic flow patterns
6- restore the mechanical integrity of the injured system,

Regeneration is the perfect restoration of the preexisting tissue architecture in the absence of scar formation. Although regeneration is the ideal in wound healing, it is only found in embryonic development, or in certain tissues, such as bone and liver

Hemorrhage

M/C cause → Trauma
But Not only one

- Is the exsanguination of blood from the vasculature into surrounding tissues, a hollow organ or body cavity , or to the outside .

- Is noted by the following terms : -

A. **Hematoma**

- Confined hemorrhage within a tissue or organ .

B. Hemothorax , hemopericardium , hemoperitoneum , and hemarthrosis

- Are hemorrhage into pleural cavity , pericardial sac, peritoneal cavity, or a synovial space , respectively .

C. Petechial hemorrhages, petechiae, or purpura

- Are small punctate (spot) hemorrhages in the skin, mucous membranes, or serosal surfaces.

D. Ecchymosis *Bruises*

- Is diffuse hemorrhage, usually in skin and subcutaneous tissue.

Wound closure types

Imp

Close wound.

- **Primary intention**, closures are those wounds that are immediately sealed with simple suturing, skin graft placement, or flap closure, such as the closure of the wound at the end of a surgical procedure.

Proximation of Wound Edge

open wound.

- **Secondary, or spontaneous intention** involves no active intent to seal the wound. Generally, this type of closure is represented by the highly contaminated wound, which will close by reepithelialization and contraction of the wound.

Normal Body Response

Infected wound → Abscess ulcer

ياخذ وقت اطول

عادة

Inflamatory phase

Proliferative phase → Final Healing By Scar

- **Tertiary intention is also referred to as delayed primary closure**. A contaminated wound is initially treated with repeated débridement and perhaps systemic or topical antibiotics for several days to control infection. Once it is assessed as ready for closure, surgical intervention, such as suturing, skin graft placement, or flap design, is performed.

Between 1st - 2nd

Serial of Debridement / Dressing / NPWT

Normal الساعد ال Body Healing

On Examination → Sign of clean Wound

Clouse "1st"

Larg Infected Wound →

ما التوقع انها تعالج حالها

Categories of surgical wounds

IMP

- Risks of Infection -

Category	Example	antibiotic Recommendation
Clean → No enteric content spillage or Bacterial spillage	thyroid, Hernia, varicose veins, breast Prosthetic surgery: vascular, orthopaedic implants	No Prophylaxis (<3%) except for mesh/prosthesis/implants
Clean Contaminated	Elective cholecystectomy Elective colorectal /prepped bowel (without spillage of content)	Prophylaxis (3-5%)
Contaminated + gunshot + open Trauma	spillage of hollow viscus content (GB,colon) surgeries	Prophylaxis (5-10%)
Dirty	Drainage of abscess Faecal peritonitis ↳ perforated colon	therapeutic (30%)

Most clean surgery : need prophylactic

Preop – 30 Min ← Cardiac surgery
Post op – 2-3 d

او بحالة اذا في prosthesis part

Usual Single Dose

Incisions او

Minimal control Spillage

Phases of wound healing :

- The stages of wound healing are usually overlapping
- Early: (1-4 days)
  - Hemostases (day 0)
  - Inflammatory phase (day1-4)

- Intermediate: (2 days-3 weeks)
  - Granulation/proliferation phase

- Late: (3 weeks to years)
  - Remodelling / maturation phase – Scar formation

Hemostases phase:

- Starts immediately after injury
- Immediate Vasoconstriction
- Initiation of extrinsic and intrinsic coagulation cascade
  ↳ Tissue factor
- platelets aggregation to damaged endothelium and release of adenosine diphosphate (ADP), promoting thrombocyte clumping, which closes the wound.
- Fibrin matrix stimulation. Initial scaffold for wound healing. In later phases of wound healing, the fibrin matrix facilitates cell attachment and serves as a reservoir for cytokines.
- Clot formation / late phase of hemostases

Inflammatory phase:

- Activation of the **complement** and **kinin** cascades.
- **leukocytes chemotaxis** (migration out of the intravascular space and into the wound.)
- **TGF-β and TGF-a, PDGF**
- Polymorphonuclear leukocytes **(PMNs) are the dominant** inflammatory cells in the wound for the first 24 to 48 hours, which phagocytize bacteria and damaged tissue, and also release cytokines such as TNF-alpha and interleukin-1 that further stimulate the inflammatory response and local vasodilation.
- PMN leaves the wound at 72 hours

Chemotaxis
Neutrophils

↑ Efflux of cell.

→ 72H لو مكثت
Release Metalloprotease → Chronic Wound

- سؤال -
(main cell) on this phase
PMNs Neutrophils

لحد 72H → عندما تجي Macrophage

Inflammatory phase:

*Main cell in wound Healing *

- **Migration of monocytes that differentiate into macrophages**

- Macrophages are activated by the locally produced cytokines and are essential for coordination of the healing process. They phagocytize bacteria and damaged tissue, secrete enzymes for the degradation of tissue and extracellular matrix, and release cytokines TGFs, cytokines and interleukin 1 (IL-1), tumor necrosis factor (TNF), and PDGF.for inflammatory cell recruitment and fibroblast proliferation.

Neutrophil ← يليها من

+ fibroblast → collagen تجيبه

Inflammatory phase:

Short on 1ry closure

- Chemotaxis
- Vasodilation
- Phagocytosis

Inflammatory phase:

- The inflammatory phase lasts a well-defined period of time in primarily closed wounds (4 days), but it continues indefinitely to the end point of complete epithelialization in wounds that close by secondary or tertiary intention.

- Foreign material, bacteria, or other imbalances that can change a normal healing wound into one with chronic inflammation and chronic nonhealing wound.

Granulation / Proliferative Phase: 2 days to 3 weeks

- 4 overlapping subphases : fibroplasia, collagen/ matrix deposition, angiogenesis and epithelialization

- **Fibroblast migration** occurs 2 to 5 days after injury . cytokines influence fibroblasts migration into the wound from undamaged tissue. laying down new collagen of the subtypes I and III. (Collagen deposition)

- **angiogenesis** takes place to restore the vasculature by endothelial cells (mediated by FGF and VEGF)

Granulation / Proliferative Phase: 2 days to 3 weeks

- **Epithelialization** restores the barrier between the wound and the external environment.
- Epithelialization of wounds occurs via the migration of epithelial cells from the edges of the wound and from remaining skin appendages.
- Migration of epithelial cells occurs at the rate of 1 mm/day in clean, open wounds.
- Primarily closed wounds have a contiguous epithelial layer at 24 to 48 hours.
- - Cell travel about 3 cm from point of origin in all directions
- EGF plays an important role in epithelization process.

Remodeling/maturation: 3weeks to years

- Includes collagen cross-linkage , wound contraction and scar formation
- Wound contraction frequently referred to in the granulation phase
- Collagen cross-linking .
- synthesized at an accelerated rate for 2 to 4 weeks
- Reaches the peak during the 3rd to 4th week
- Early in normal wound healing, type III collagen predominates but is later replaced by type I collagen.
- Oxygen, vitamin C, alpha-ketoglutarate, and iron are important cofactors for the cross-linkage of collagen fibers.
- d-Penicillamine – inhibits collagen cross-linking

Steroid effect this phase

يوقف Collagen production خلال شهر

يصير قوي خلال

- Cross-linking

- Breaks down ↓↑ formation

بس نفس Amount of Collagen

- Wound contraction:
- is a decrease in the size of the wound without an increase in the number of tissue elements that are present.

- It involves movement of the wound edge toward the center of the wound through the contraction of myofibroblasts. Wound contraction begins 4 to 5 days and continues for 12 to 15 days or longer if the wound remains open.

- Wound contraction occurs to a greater extent with secondary healing than with primary healing.

scar formation and remodeling :

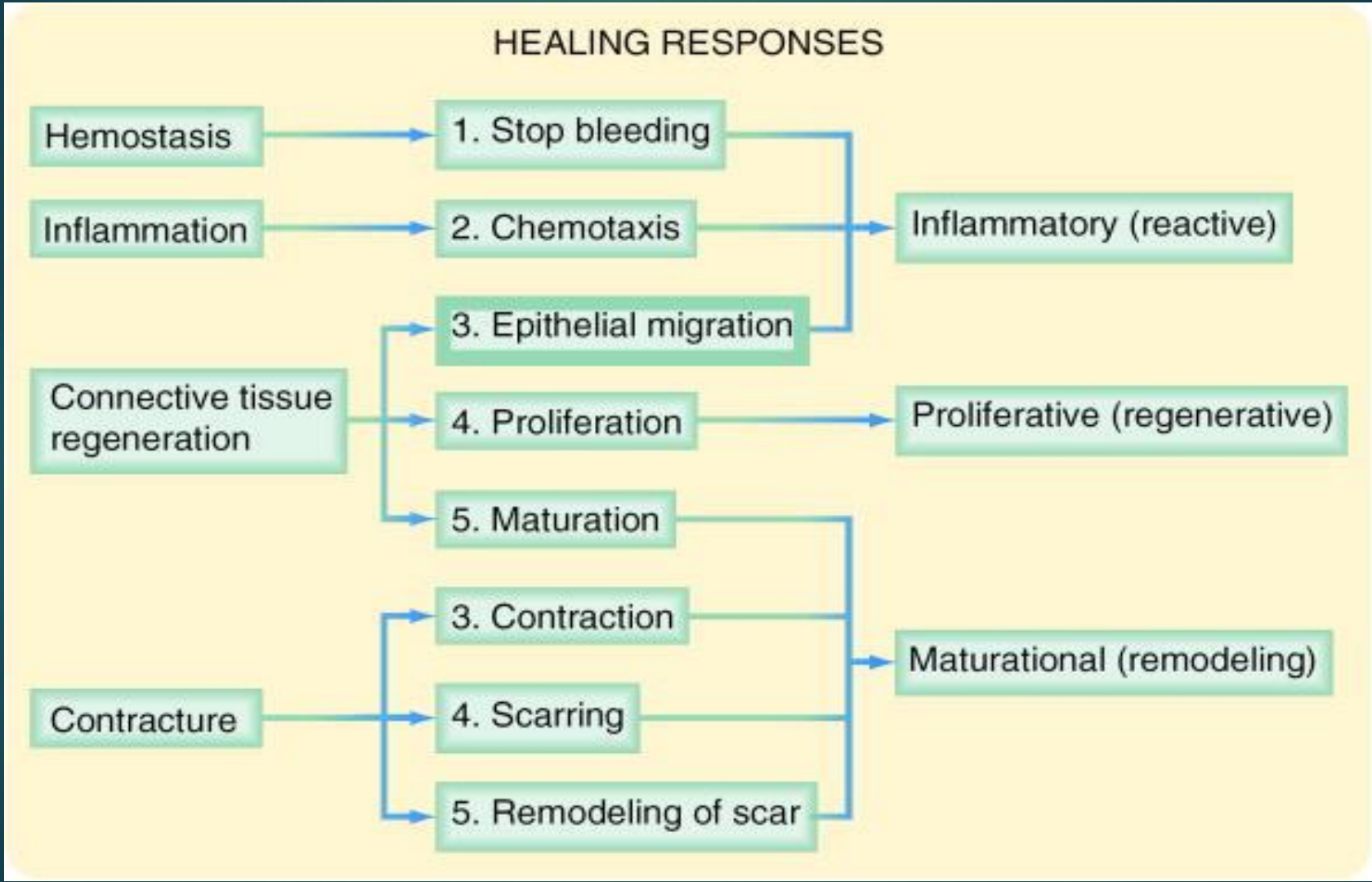
- Starts at 21 days.
- cellularity of the wound decreases.
- Alternating break down and deposition of collagen resulting in no change in the total amount of collagen present in the wound.

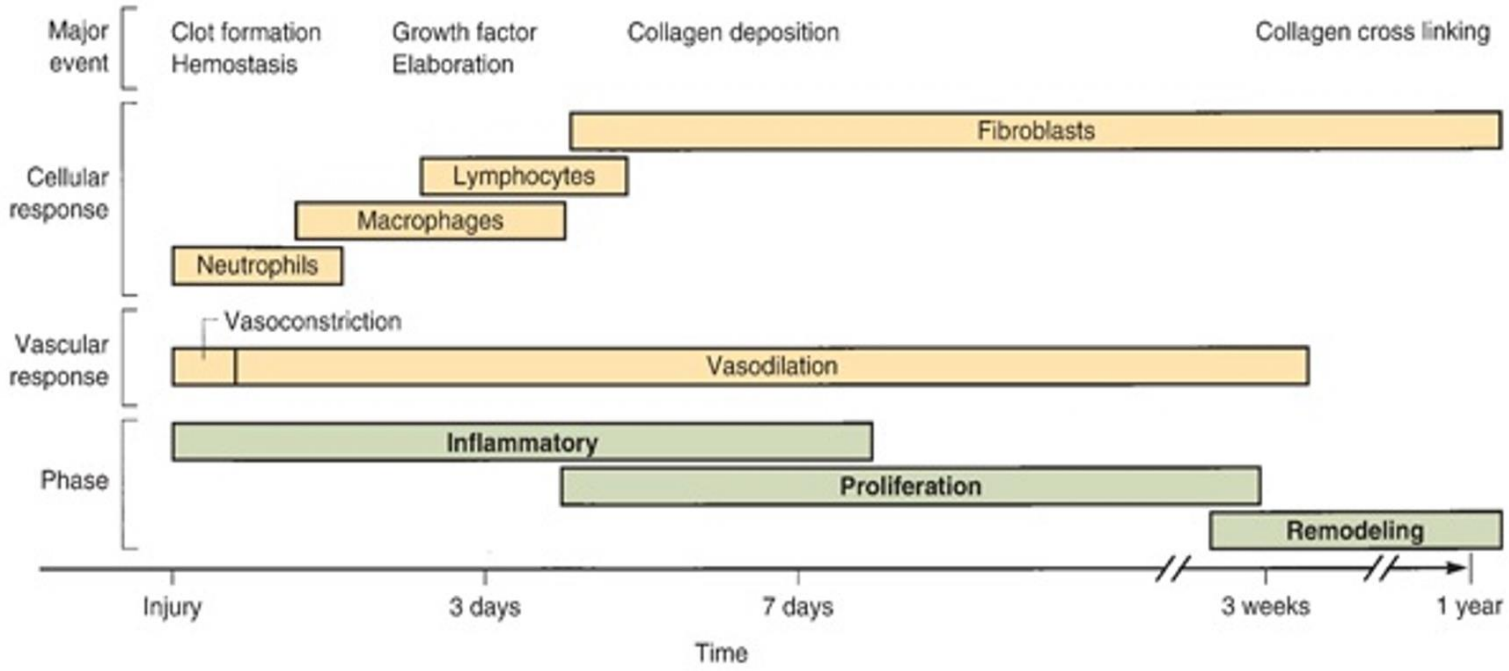
- This process reaches a plateau at 12 to 18 months, but it may last indefinitely.
- Maximal tensile strength of the wound is achieved by 8 weeks, and the ultimate resultant scar has only 80% of the tensile strength of the original skin that it has replaced.
- a well-healed wound never achieves the strength of normal tissue.

Imp

- When Wound Reach MAX. Strength ? 3-6 M , ايثورد الدكتور other Source 9 2M
- " " " Normal Skin " ? Never

Wound healing response





Timeline of phases of wound healing with dominant cell types and major physiologic events.

Collagen

Type	Description
I	Most common type of collagen: **skin**, **bone**, and **tendons** Primary collagen in a **healed wound**
II	**Cartilage**
III	Increased in **healing wound**, also in **blood vessels** and **skin**
IV	**Basement membranes**
V	Widespread, particularly found in the **cornea**

Imp
MCQ

- **Order of cell arrival in wound**
- Platelets
- PMNs
- Macrophages
- Lymphocytes (recent research shows arrival before fibroblasts)
- Fibroblasts

- **Predominant cell type by day**
- Days 0–2 – PMNs
- Days 3–4 – macrophages
- Days 5 and on – fibroblasts

All wounds undergo the same basic steps of repair.

- Acute wound occurs in a normal orderly fashion and often requires minimal practitioner intervention.

- Chronic wound does not follow that orderly progression of healing to restoration of functional integrity, It is stuck in the inflammatory phase owing to a variety of etiologies and does not proceed to closure. and often necessitates a variety of interventions to facilitate complete healing.

Complication ⇒ Marjolin ulcer

- Epithelization / granulation integrity – most important factor in healing open wounds (secondary intention)

- Tensile strength – most important factor in healing closed incisions (primary intention)
- • Depends on collagen deposition and cross-linking of collagen

keloids and hypertrophic scars

IMP

Both keloids and hypertrophic scars are characterized by excessive collagen deposition versus collagen degradation

More Aggressive

Keloids are scars that grow beyond the borders of the original wounds, rarely regress with time, more prevalent among patients with darker pigmented skin, It appears to have a genetic predisposition, tends to occur above the clavicles, on the trunk, in the upper extremities, and on the face.

↑TGF
↑P53

female

✓ Keloids cannot be prevented and are refractory to medical and surgical intervention

Tx: intra-lesion steroid injection; silicone, pressure garments, XRT

Hypertrophic scars are raised scars that remain within the confines of the original wound and frequently regress spontaneously

Can occur anywhere on the body

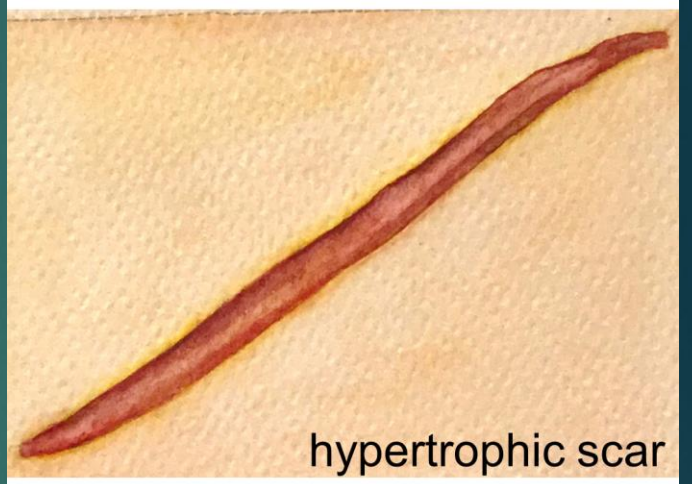
Can be preventable

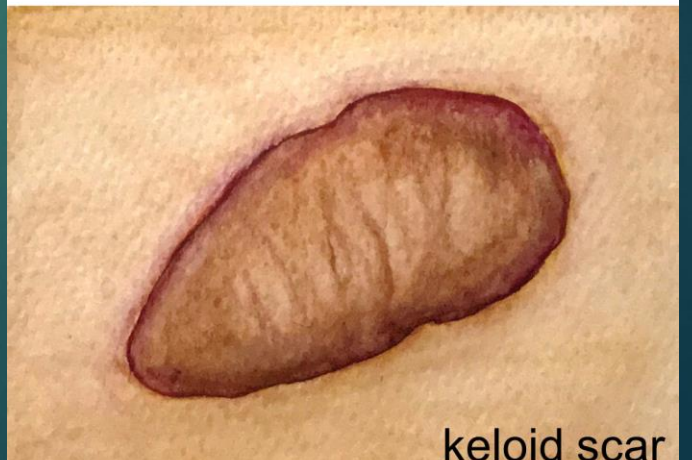
Tx: steroid injection, silicone, pressure garments surgical ???

Better Response

	Hypertrophic	Keloid
Behaviour	Rapid growth then spontaneous resolution over time	Doesn't self-resolve
Acuteness	Acutely	Any time after injury
Demographic	No specific cohort	Pigmented skin with a family history
Symptoms	Itch (mast cells)	Itch, pain, sensitivity
Collagen	Well-organised type III collagen bundles with myofibroblast nodules parallel to the epidermis	Poorly-organised decreased ratio of Type III:Type I collagen, random to the epidermis, upregulated p53.
Area	Limited to original wound	Extend beyond original wound
Recurrence	No	Yes

TNF-a plays an important role





Local and systemic factors that impede wound healing

Local factors

- Inadequate blood supply
- Increased skin tension
- Poor surgical apposition
- Wound dehiscence
- Poor venous drainage
- Presence of foreign body and foreign body reactions
- Continued presence of microorganisms
- Infection
- Excess local mobility,such as over a joint

Systemic factors

- Advancing age and general immobility
- Obesity
- Smoking
- Malnutrition
- Deficiency of vitamins and trace elements
- Systemic malignancy and terminal illness
- Shock of any cause
- Chemotherapy and radiotherapy Immunosuppressant drugs, corticosteroids, anticoagulants
- Inherited neutrophil disorders, such as leucocyte adhesion deficiency
- Impaired macrophage activity (malacoplakia)

- Common factors that Inhibit Wound Healing

- infection / Bacteria > 10^5/cm2 – ↓ oxygen content, collagen lysis, prolonged inflammation

- Devitalized tissue and foreign bodies – retards granulation tissue formation and wound healing

- Cytotoxic drugs – 5FU, methotrexate, cyclosporine, FK-506, etc. can impair wound healing in 1st 14 days after injury
- Diabetes – can contribute to poor wound healing by impeding the early-phase inflammation response (hyperglycemia causes poor leukocyte chemotaxis) + poor blood supply

- Albumin < 3.0 – risk factor for poor wound healing

- Steroids – prevent wound healing by inhibiting macrophages, PMNs, and collagen synthesis by fibroblasts; ↓ wound tensile strength as well

- Vitamin A (25,000 IU) – counteracts effects of steroids on wound healing

- Wound ischemia (hypoxia) – can be caused by fibrosis, pressure (sacral decubitus ulcer, pressure sores), poor arterial inflow (atherosclerosis), poor venous outflow (venous stasis), smoking, radiation, edema, vasculitis

- Ionizing radiation, malnutrition, vit.(C,A) def., (zinc,iron) def., advanced age, malignancy

- **Diseases associated with abnormal wound healing**
- • Osteogenesis imperfecta – type I collagen defect
- • Ehlers–Danlos syndrome – 10 types identified, all collagen disorders
- • Marfan's syndrome – fibrillin defect (connective tissue protein)
- • Epidermolysis bullosa – excessive fibroblasts. Tx: phenytoin
- • Scurvy – Vitamin C deficiency

CHRONIC WOUND HEALING

- chronic wound is a wound that fails to heal in a reasonable amount of time due to a disruption of the normal process of acute wound healing.
- Most chronic wounds are slowed or arrested in the inflammatory or proliferative phases of healing and have increased levels of matrix metalloproteinases, which bind up or degrade the various cytokines and growth factors at the wound surface.
- Treatment of these causes, along with maximal medical management of underlying medical problems, restores more normal healing processes.

1- Intrinsic or local factors

- abnormalities within the wound that prevent normal wound healing.
- (1) foreign body, – Debridement –
- (2) necrotic tissue,
- (3) repetitive trauma,
- (4)hypoxia/ischemia,
- (5) venous insufficiency,
- (6) infection,
- (7) growth factor deficiency,
- (8)excessive matrix protein degradation,
- (9) radiation.

2- Extrinsic or systemic factors

- Optimization of these factors is critical to healing a chronic wound
- (1) Diabetes mellitus,
- (2) steroids , antineoplastic drugs,
- (3) smoking,
- (4) collagen vascular disease,
- (5) malnutrition
- (6)chronic kidney and liver diseases

Chronic Nonhealing Wounds and ulceration

Some complications of chronic wounds

x Sinus formation

x Fistula

x Unrecognised malignancy

x Malignant transformation in the ulcer bed (Marjolin's ulcer)

x Osteomyelitis

x Contractures and deformity in surrounding joints

x Heterotopic ossification (BMP → Fibroblasts → osteoblasts → formation of bone tissue in soft tissue)

x Colonisation by multiple drug resistant pathogens, leading to antibiotic resistance

Assessing wounds

- Size of wound
- Edge of wound

Wound edge characteristics

Edges	Type of ulcer
Sloping	Venous ulcer, healing ulcer
Punched out	Neuropathic ,syphilis ulcer
Rolled	Basal cell carcinoma
Everted	Squamous cell carcinoma
Undermined	Tuberculosis,

- **Site of wound**

Site	**Type of ulcer**
Gaiter area of the leg (5-15 cm Above medial malleolus)	Venous ulcer
Sacrum, greater trochanter, heel	Pressure ulcer
Dorsum of the foot / Tip of toes	Arterial or vasculitic ulcer
Shin	Necrobiosis lipoidica
Lateral malleolus	Venous, arterial, or pressure ulcer
Plantar and lateral aspect of foot and toes	Diabetic ulcer
Sun exposed areas	Basal cell carcinoma; squamous cell carcinoma

- Wound bed (Base)
- Necrotic tissue, slough, and eschar, bone, tendon)
- Depth, base, floor
- Surrounding skin (erythema,necrosis,normal skin)
- Discharge (purulent →Infection)
- Foul smell
- Pain

Base → Palpation VS floor → Inspection

Clinical features of nonhealing wounds

1. Absence of healthy granulation tissue
2. Presence of necrotic and unhealthy tissue in the
3. wound bed
4. Excess exudate and slough — fluid
5. Lack of adequate blood supply — Ischemie
6. Failure of reepithelialisation
7. Cyclical or persistent pain
8. Recurrent breakdown of wound
9. Clinical or subclinical infection

* سؤال امتحان *

Definitive Diagnosis of Infection → Swab culture 10^5 Bacteria

SPECIAL CATEGORIES OF CHRONIC WOUNDS

Diabetic foot ulcers

Mixed Patho

Diabetic foot ulcers can be divided into two groups: those in neuropathic feet (so called **neuropathic ulcers**) and those with ischaemia often associated with neuropathy (so called **neuroischaemic ulcers**).

The crucial difference between the two is the absence or presence of ischaemia. The presence of ischaemia may be confirmed

1. by a ankle brachial index < 0.9 ,
2. claudication

Neuropathic ulcers/ pressure ulcers usually occur on the plantar aspect of the foot under the metatarsal heads or on the plantar aspects of the toes.

Neuroischaemic ulcers are often seen on the margins of the foot, especially on the medial surface of the first metatarsophalangeal joint and over the lateral aspect of the fifth metatarsophalangeal joint.They also develop on the tips of the toes

Med 1st MtP
lat 5th MtP

sensory, motor, proprioception

PIE ⇒ Ulcer – Neuro – Vasculcity – lymphatic

Diabetic Foot Ulcer (DFU)

→ Examination.

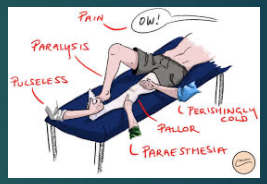
- quality of the peripheral circulation
- Ulcer exam(site,size,shape,edges,base,floor,discharge).
- Web spaces and nails should be examined for evidence of mycotic infection, which may lead to fissuring of the skin and subsequent infection. → fungal infection , Imp Tinea Pedis or Athlete's foot
- degree of sensory loss (pain,temp,vibration,proprioception)
- Semmes Weinstein monofilament test for neuropathy.
- Neuropathic, arthropathic, and vasculopathic ulcers occur on the plantar surface of the metatarsals and extend to the metatarsal head, leaving exposed cartilage.

كيفها كلمة جاية OSCE

- Vascular assessment: 1. general inspection
- Pulses, doppler, capillary refill
- Ankle brachial index
  - Ct Angio
- Skeletal deformities:
- Minor : Hammertoe, claw toe, overriding
- Major : Charcot joint, rocker bottom deformity
- plain x-rays of the foot to evaluate for osteomyelitis.

Shin skin Hair loss Muscle Atrophy → Chronic Ischemia

→ 6P



→ Acute lower limb Ischemia

→ loss of Arch
→ loss of sensation

— Swelling - Bone fragment

Treatment.

Debridement

- Clean wounds are treated with minimal debridement and damp gauze or hydrogel-based dressing changes. Hydrogel dressings may be more effective than damp gauze (Cochrane Rev).

- Exudative wounds may benefit from alginate, hydrocolloid, or Negative Pressure Wound Therapy (NPWT) that removes excess wound exudate

- Vaccum -

Infected wounds

- diagnosed based on clinical signs of infection.

(purulent discharge/foul smell/ edema/ erythema …)

- Plain x-rays may show osteomyelitis or gas in the soft tissues.

- Admission → Poly microbial, Resistent, Need Debridment
- broad-spectrum antibiotic therapy. ***
- thorough exploration with drainage / counterdrainage of all abscess cavities
- debridement of infected, necrotic, or devitalized tissues.
- If became clean then can be managed with local wound care as described.

Antibiotic therapy.

لحد ما تطلع الـ culture

- Initially broad spectrum (Gram-positive and Gram-negative organisms.
- In the acute phase parenteral treatment is indicated.
- Obtain Wound cultures prior to initiation of antibiotics.

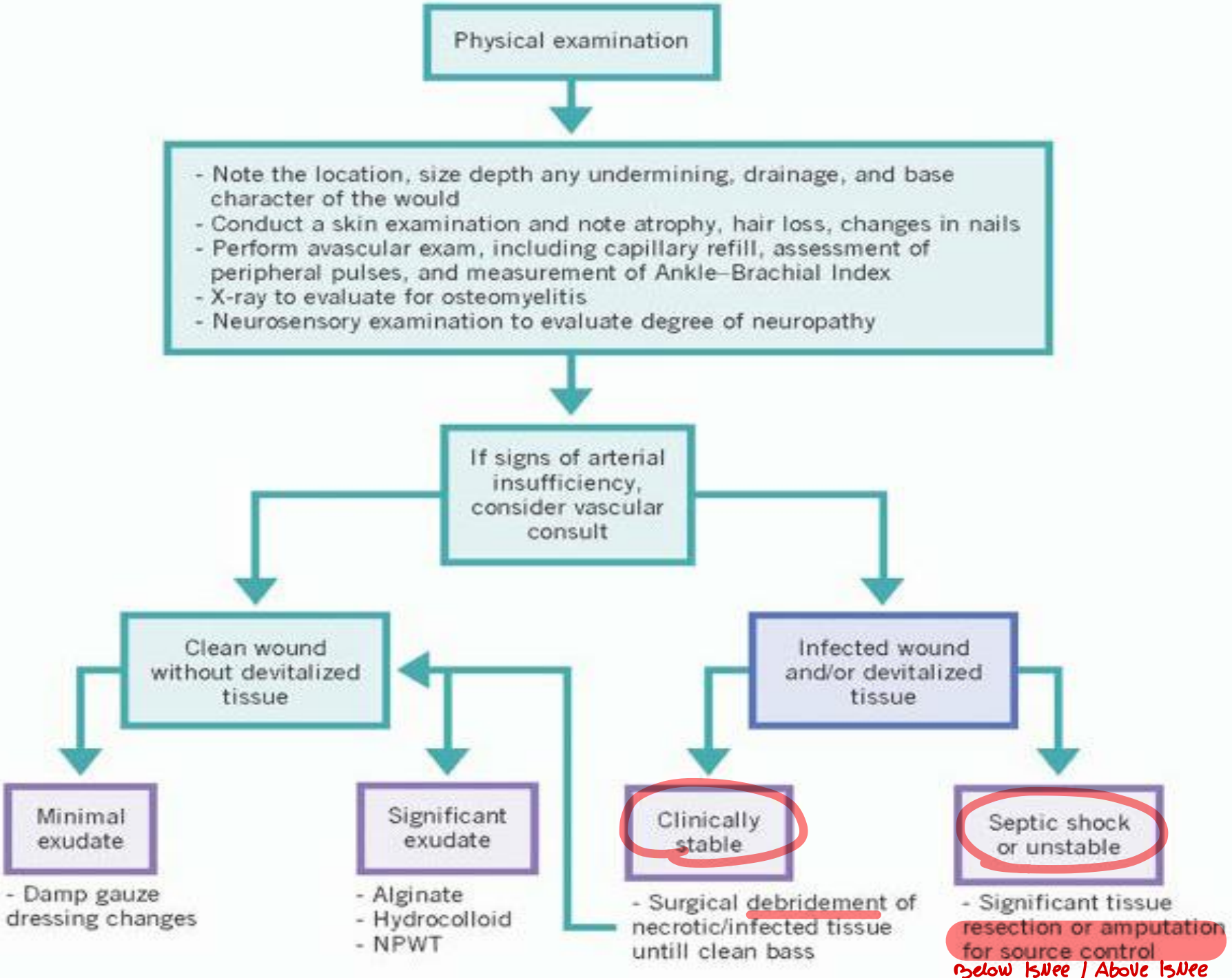
- **Duration** of antibiosis depends on severity of infection.
- mild infections limited to the soft tissue (1-2 weeks),
- moderate or severe infections (2-4 weeks)
- osteomyelitis involving viable bone, (4-6 weeks)
- Consultation with an ID specialist is helpful in guiding therapy

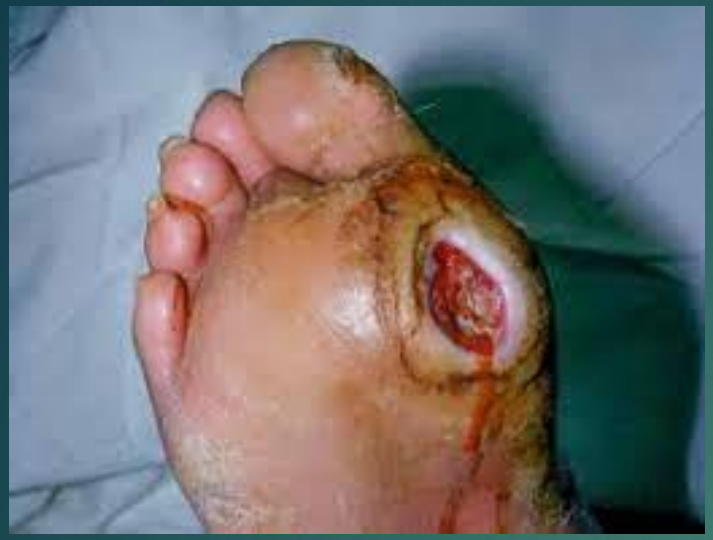
Prevention :

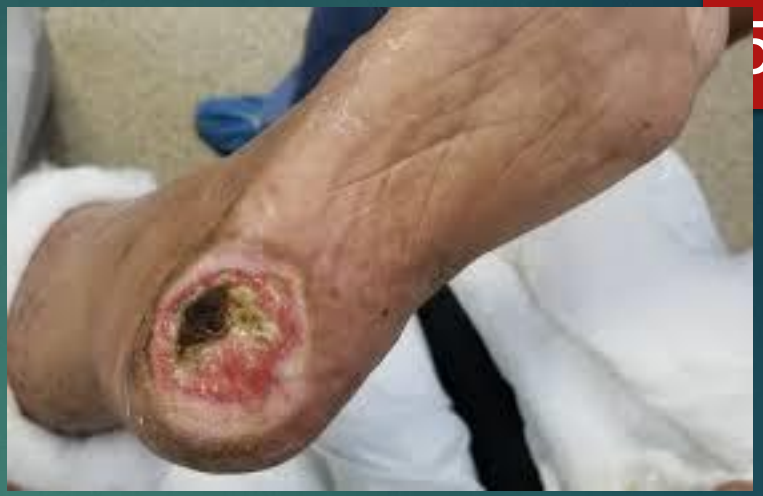
-one of the most important elements in the management of the diabetic foot.

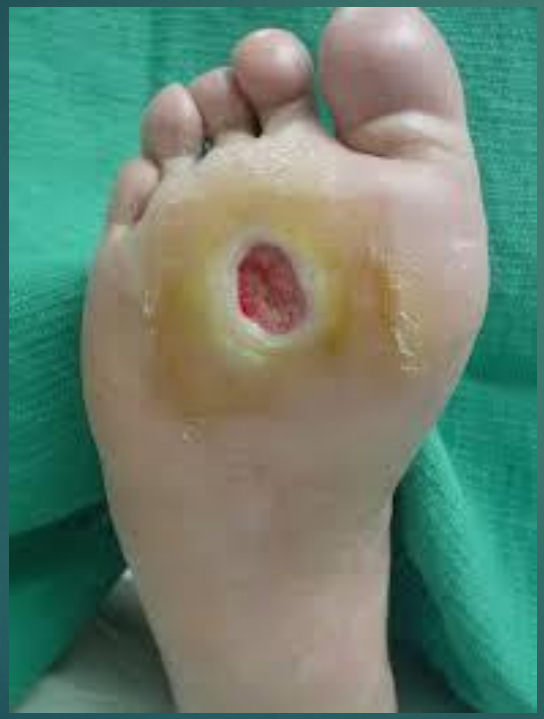
- attention to hygiene and daily inspection for signs of tissue trauma
- Patient education
- custom-made shoes are helpful in relieving pressure on weightbearing areas and should be prescribed for any patient who has had neuropathic ulceration.
- Offloading casts

- Don't forget to Treat the cause (better DM control, revascularization ??)


Physical examination
- Note the location, size depth any undermining, drainage, and base character of the would
- Conduct a skin examination and note atrophy, hair loss, changes in nails
- Perform avascular exam, including capillary refill, assessment of peripheral pulses, and measurement of Ankle–Brachial Index
- X-ray to evaluate for osteomyelitis
- Neurosensory examination to evaluate degree of neuropathy
If signs of arterial insufficiency, consider vascular consult
Clean wound without devitalized tissue
Infected wound and/or devitalized tissue
Minimal exudate
- Damp gauze dressing changes
Significant exudate
- Alginate
- Hydrocolloid
- NPWT
Clinically stable
- Surgical debridement of necrotic/infected tissue untill clean bass
Septic shock or unstable
- Significant tissue resection or amputation for source control
Below ISNee / Above ISNee







↳ Ischemie
ss golds
Pressure point

Arterial insufficiency ulcers

The most common cause is atherosclerotic disease of the medium and large sized arteries. Age, smoking, diabetes, thromboangiitis, vasculitis, thalassaemia, and sickle cell disease.

Chronic

Burger's Disease.
Thromboangiitis Obliterans.

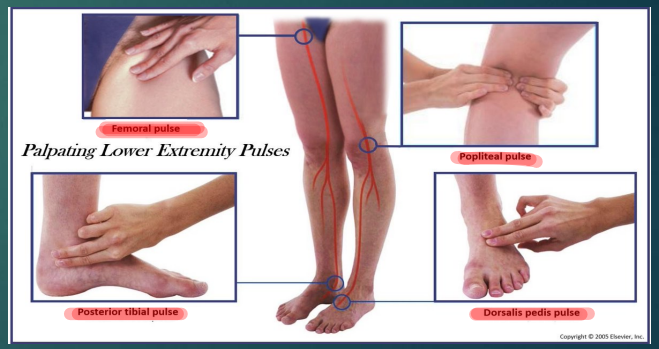
- Embolic events also a cause → afib, showering emboli (blue toe syndrome)

  proximal thrombus rupture with distal embolic propagation

Acute

Arterial insufficiency ulcers

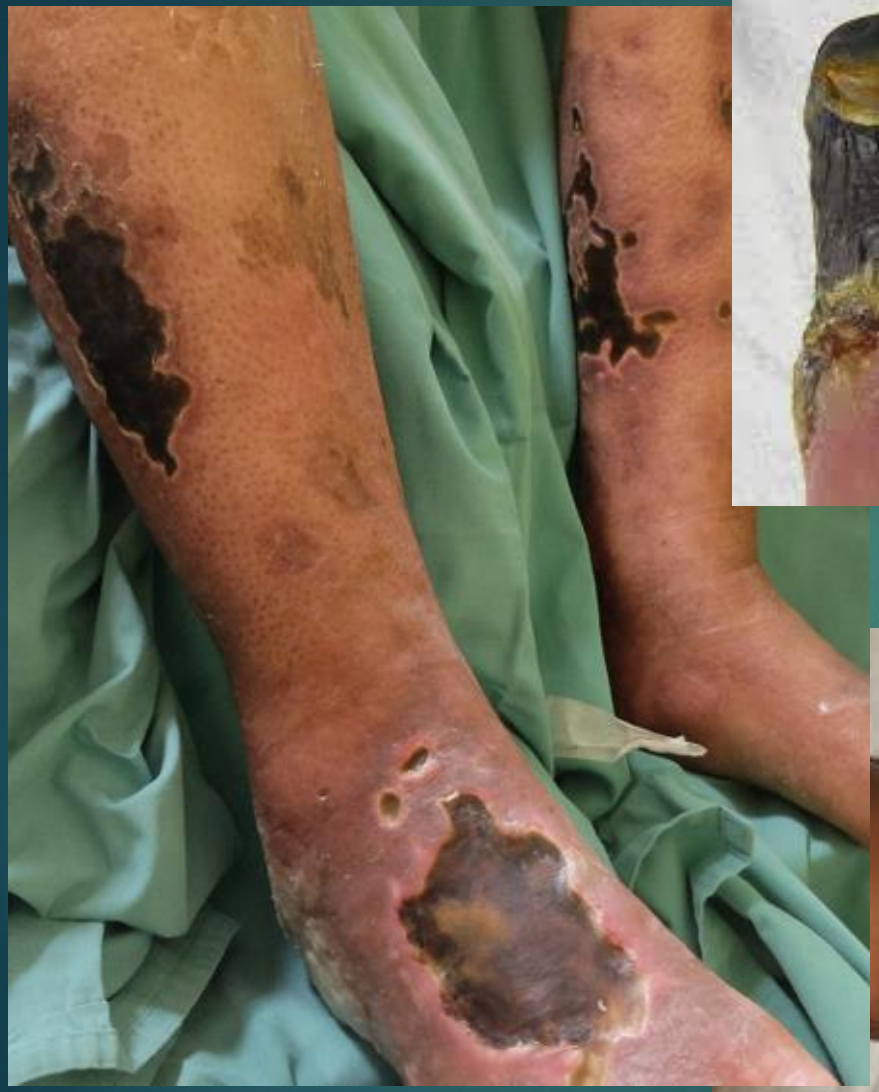
- occur distally on the tips of the toes/ near lateral malleolus.
- The surrounding skin is thin, shiny, and hairless.
- ++ claudication / rest pain;
- some patients with significant neuropathy may lack any pain symptoms
- Peripheral pulses are diminished/ absent.

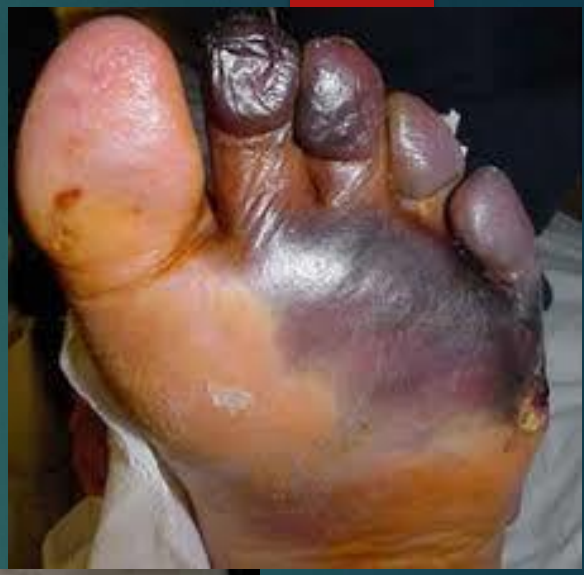


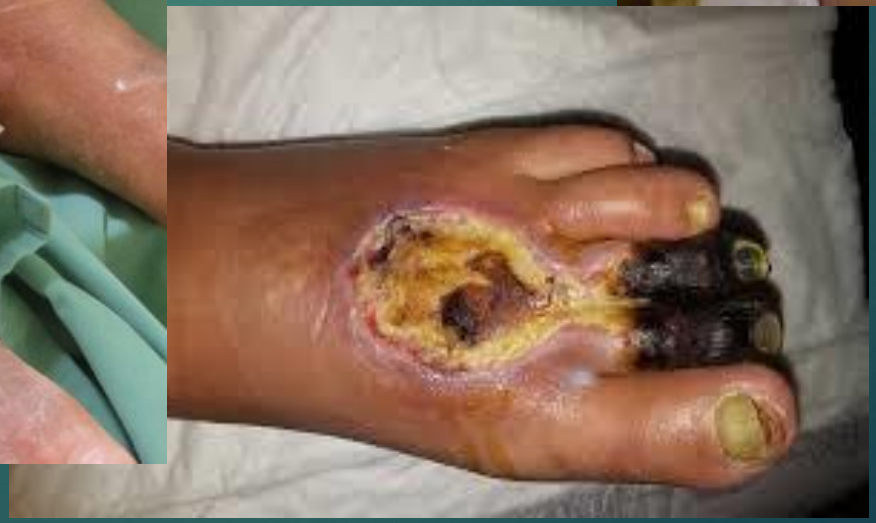
- Thorough vascular evaluation should be obtained, including a peripheral and central pulse examination and segmental Doppler with calculation of ABI.

Arterial insufficiency ulcers

- Neglected chronic arterial insufficiency can result in **dry or wet gangrene.**
- Wet gangrene can lead to an ascending necrotizing infection severe enough to call for amputation while dry gangrene can convert to wet at any time.
- Critical is restoration of arterial inflow (if possible)
- devitalized tissue can be resected to facilitate healing.
- If infection is suspected, obtain wound cultures, debride infected tissue, and institute appropriate antibiotics.







Venous ulceration

Risk factors for venous ulceration

Direct risk factors

- x Varicose veins
- x Deep vein thrombosis
- Chronic venous insufficiency (post phlebitic syndrome)
- x Arteriovenous fistulae
- x Obesity
- x History of leg fracture

Indirect risk factors

- x All risk factors leading to deep vein thrombosis including proteinC, ProteinS, and antithrombin III deficiency
- x Family history of varicose veins
- x A history of minor trauma prior to the development of ulceration may also be identified

Examination

1. 95% in the gaiter area of the leg which may be discrete or circumferential
2. Pitting oedema
3. deposition of haemosiderin within macrophages
4. Lipodermatosclerosis (This is characterised by the dermis and subcutaneous tissue becoming indurated and fibrosed with the lack of pitting oedema; the skin also becomes atrophic, loses sweat glands and hair follicles, and becomes hyperpigmented. Severe lipodermatosclerosis may lead to atrophy blanche—white fibrotic areas with low blood flow.

Venous eczema (erythema, scaling, itching)

Features of venous eczema and cellulitis

Venous eczema

Red, warm, painful, and tender to touch

- Usually chronic

Diffuse and poorly demarcated

Increase in exudate

- Itchy
- Scaly تقشر

Treated with topical steroids

Cellulitis

Red, warm, painful, and tender to touch

- Insidious (usually develops over 24-72 hours)

Usually well demarcated

No increase in exudate

Not itchy

Not scaly

Treated with systemic antibiotics

Treatment :

- Infected Ulcers:

Treat the infection first.

Staphylococcus aureus, Streptococcus pyogenes, and Pseudomonas are most common organisms

Usually treated with local wound care, wet-to-dry dressings, and oral

antibiotics.

- Topical antiseptics initially but should be avoided when clean
- Severe infections require intravenous antibiotics.

Debridement if infected / necrosis present

- Leg elevation
- Compression therapy

Most Imp

is the mainstay of venous ulcer management

Graded compression, with greatest pressure (about 40 mm Hg) at the ankle, tapering off to lower pressure (about 18 mm Hg) below the knee

Mid Metatarsal area

C/I → Arterial Ischemia

- Elastic compression stockings
- Unna boots (zinc oxide + compression)
- Bandage sequential compression

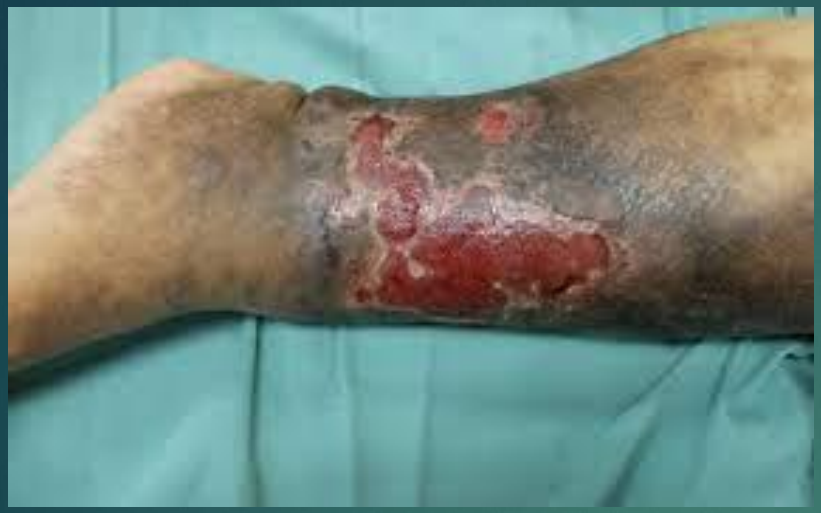
- Dressing with Topical Medications (silver containing ointments, Atrumann AG

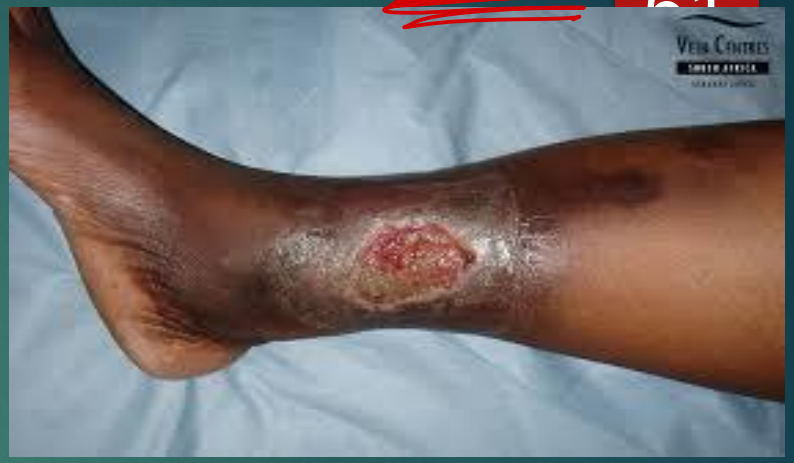
Surgical treatment

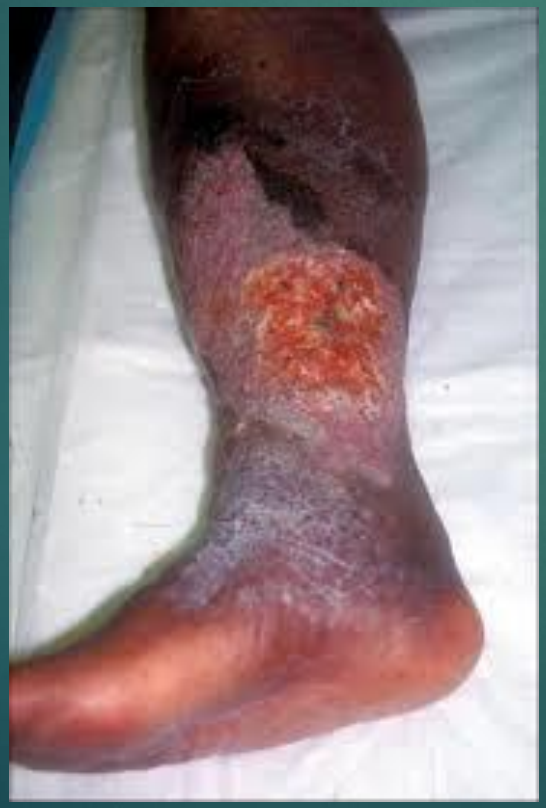
- Only to prevent ulcer in high risk patients with confirmed venous insufficiency (varicose vein, +ve duplex u/s)
- **DVT should be excluded first**
- sclerotherapy,
- saphenous vein stripping,
- endovenous ablation of the saphenous vein,
- Subfascial Endoscopic Perforating Vein Surgery (SEPS), and varicose vein stab avulsion.

Mini OSCE سؤال

61







Features of venous and arterial ulcers

	Venous	Arterial
History	History of varicose veins, deep vein thrombosis, venous insufficiency or venous incompetence	History suggestive of peripheral arterial disease, intermittent claudication, and/or rest pain
Classic site	Over the medial gaiter region of the leg	Usually over the toes, foot, and ankle
Edges	Sloping	Punched out
Wound bed	Often covered with slough	Often covered with varying degrees of slough and necrotic tissue
Exudate level	Usually high	Usually low
Pain	Pain not severe unless associated with excessive oedema or infection	Pain, even without infection
Oedema	Usually associated with limb oedema	Oedema not common
Associated features	Venous eczema, lipodermatosclerosis, atrophie blanche, haemosiderosis	Trophic changes; gangrene may be present
Treatment	Compression is mainstay	Appropriate surgery for arterial insufficiency; drugs of limited value

Interpreting ankle brachial pressure index

Index	Signs and symptoms	Severity of disease	Action
≥ 0.7-1	Mild intermittent claudication, or no symptoms	Mild arterial disease	Reduce risk factors and change lifestyle: stop smoking, maintain weight, exercise regularly, consider antiplatelet agent
0.7-0.5	Varying degrees of intermittent claudication	Mild to moderate arterial disease	As for index ≥ 0.7-1, plus referral to outpatient vascular specialist and possible arterial imaging (duplex scan and/or angiogram)
0.5-0.3	Severe intermittent claudication and rest pain	Severe arterial disease	As for index ≥ 0.7-1, plus urgent referral to vascular specialist and possible arterial imaging (duplex scan and/or angiogram)
≤ 0.3 or ankle systolic pressure < 50 mm Hg	Critical ischaemia (rest pain > 2 weeks) with or without tissue loss (ulcer, gangrene)	Severe arterial disease; risk of losing limb	Urgent referral to vascular emergency on-call team and possible surgical or radiological intervention

ABI Value	Interpretation	Recommendation
Greater than 1.4	Calcification / Vessel Hardening	Refer to vascular specialist
1.0 - 1.4	Normal	None
0.9 - 1.0	Acceptable	
0.8 - 0.9	Some Arterial Disease	Treat risk factors
0.5 - 0.8	Moderate Arterial Disease	Refer to vascular specialist
Less then 0.5	Severe Arterial Disease	Refer to vascular specialist

Less then .3 → Critical Ischemia Rest Pain

Pressure ulcer/ decubitus ulcer/ bedsore

Necrosis

- Pathophysiology: Skin Resistance pressure ↓↓ muscle se tissue ⇒ Sup. ulcer → minimal; Deep ulcer → muscle loss.
- Prolonged pressure to soft tissue over bony prominences in paralyzed or bedridden pts leads to ischemic ulceration and tissue breakdown.
- Muscle tissue is the most susceptible. (undermined tissue loss)
- Common in hospitalized , unconscious, nursing homes or spinal cord injuries patients.
- occiput, sacrum (m/c), greater trochanter, and heels.

pressure ↑ 70 mm – 2H Significant

TABLE 9-1 National Pressure Ulcer Advisory Panel Classification Scheme

سؤال امتحان

Stage	Description
I	Nonblanchable erythema of intact skin; wounds generally reversible at this stage with intervention
II	Partial-thickness skin loss involving epidermis or dermis; may present as an abrasion, blister, or shallow crater
III	Full-thickness skin loss involving damage or necrosis of subcutaneous tissue but not extending through underlying structures or fascia
IV	Full-thickness skin loss with damage to underlying support structures (i.e., fascia, tendon, or joint capsule)

الوديد بلي

IV = MIC Stage .

Eschar

Full thickness
Black skin
Necrotic

سؤال امتحان

- You cannot adequately stage the wound until the eschar is incised and the actual depth is determined.

usual Grad 4

- also look for underlying bony breakdown, osteomyelitis,

Stage 4 Undermined Edge

Prevention :

- Skin care (clean, minimally moisturized)
- Frequent repositioning (every 2 hours)
- Supporting surfaces (e.g. gel pads, air mattresses)
- Adequate nutrition

Prevention > treatment

Treatment :

- Debridement: Until Reach Near Normal tissue.

(excise the eschar and most of the necrotic tissue then autodebridement of the minimal remaining necrotic tissue with wet-to-dry dressing or topical enzymatic collagenase ointments)

- Wound cleaning and dressing:

- If not infected → irrigation with saline and simple dressing

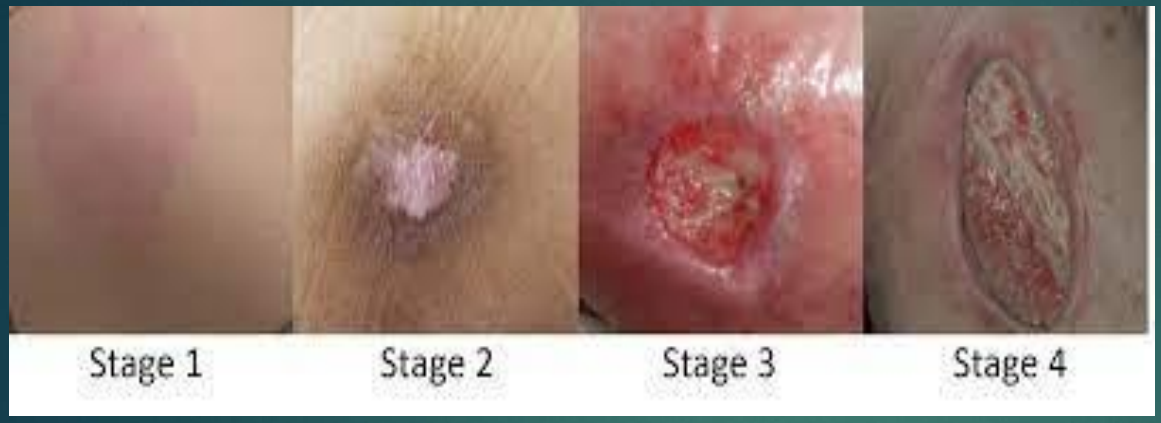
(avoid toxic solutions like povidine , it impairs healing)

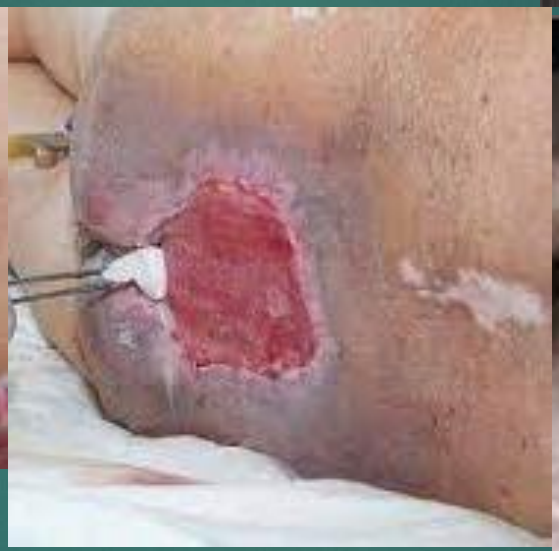
- If bacterial colonization → topical antibacterial
- If infected (pus discharge, foul smelling, erythema, bacteria count >10*5) → adequate debridement , wound cultures, dressing with topical antimicrobials , systemic abx
- When infected wound becomes clean, and all clean wounds are candidates for different dressing materials and VAC negative pressure dressing (vac has faster healing rates

Surgical treatment :

- simple closure
- split-thickness skin grafting
- musculocutaneous flap
- Only in clean wounds , well-motivated patients in whom the cause of immobility had been resolved.

- Bedridden pts with sacral ulcers might benefit from diverting colostomy if pressure cause cannot be addressed


Stage 1
Stage 2
Stage 3
Stage 4



Members of the multidisciplinary team

x Physician

X wound care team ****

x Podiatrist

x Specialist nurse

x Orthotist

x Dietitian

x Radiologist

x Vascular surgeon

x Orthopaedic surgeon

Thank you