Chronic Wound Management in the Inpatient Setting

What the $%@! Is chronic wound management…?

No disclosures

What is chronic wound management, and where does this fit in my practice?

- What makes a wound chronic?
- What's in a name? (Wound vs. ulceration)
- What are the phases of healing?
- What should a healthy wound look like?
- What factors delay healing?
- When is a wound not a wound?
- What do I put on this?
- When (and who) do I call for help?

What is chronic wound management?

- An understanding of disease processes which impair healing
- Ability to recognize stalling/failure to heal
- Understanding normal healing by secondary intention
- Understanding how to modify factors which are preventing healing

What makes a wound chronic?

- 4 generally recognized phases of wound healing (Vascular, inflammatory, proliferative, remodeling)
- Any failure to advance through these phases renders a wound “chronic”
- Disease or external disruption stops normal healing cycle

What's in a name?

- "Wound" generally refers to an acute breach in the skin (surgical wound, traumatic wound) and should advance through the phases of healing normally
- "Ulceration" generally refers to a wound which is stalled due to an underlying disease process and is considered a symptom or manifestation of that disease (venous insufficiency ulceration, diabetic foot ulceration, pressure ulceration)
- In the case of ulcerations, management of that underlying disease is essential to healing.

Cycles of wound healing

- Vascular (Hemostatic phase) – Begins immediately following injury, lasts as long as two days. Platelet adherence begins, growth factor cascade recruits WBCs
- Inflammatory – Lasts up to 6 days in a healing wound, generally the phase in which a wound becomes stalled.
- Proliferative – Lasts roughly 6-21 days in a healing wound. Epithelium begins to migrate over vascular bed, generally begins at the wound margins and spreads across the wound base
- Remodeling – Begins after wound closure, lasts 8 months to 1 year. Increased matrix metalloprotease activity to wound area which reorders deposited collagen. Type 3 fibers become realigned, more like type 1 collagen.

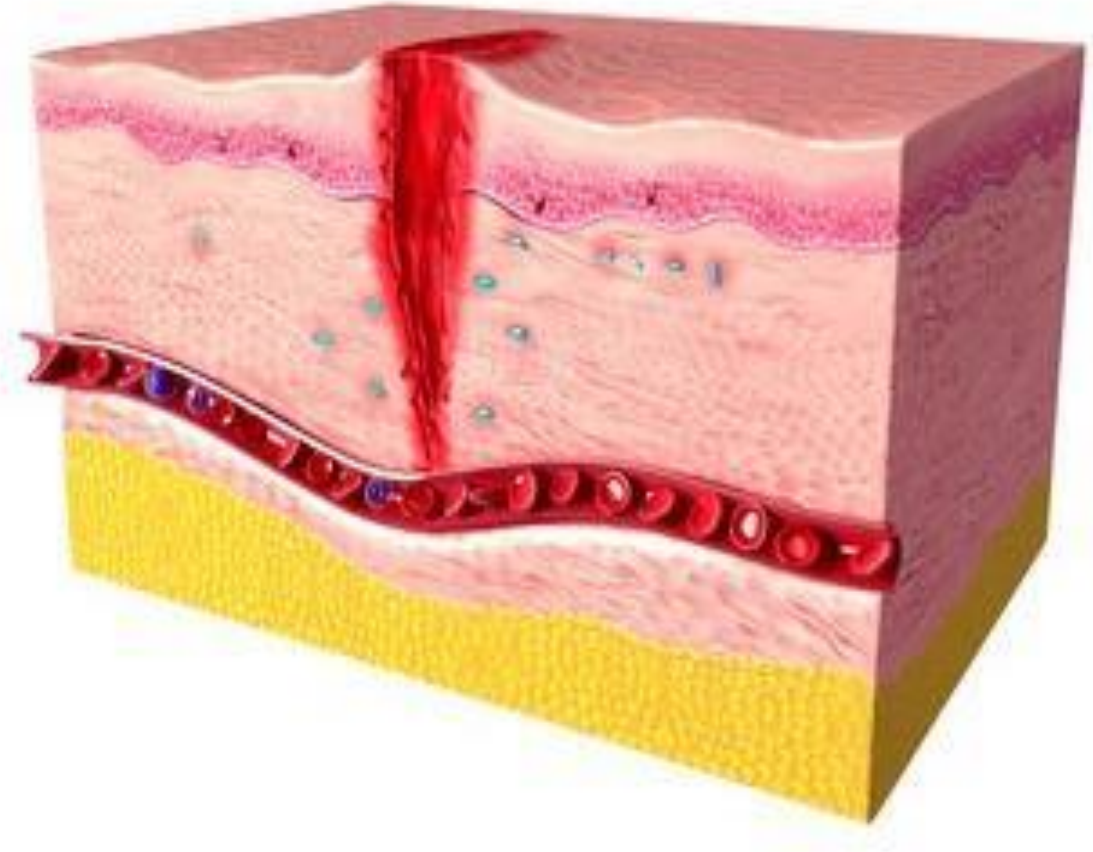
Vascular Phase

Clot formation should take place. This is the only phase of wound healing where “scabbing” is a normal response.

Platelet activity drives wound into next phase of healing.

Debridement essentially “resets” a chronic wound into the vascular phase.

Healthy tissue should bleed slightly upon cleansing or manipulation.



https://www.woundsource.com/blog/four-stages-wound-healing

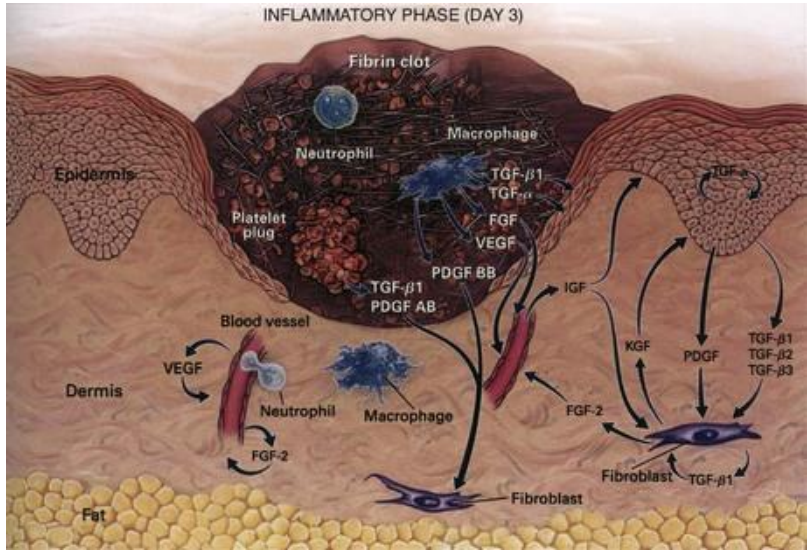
Inflammatory phase

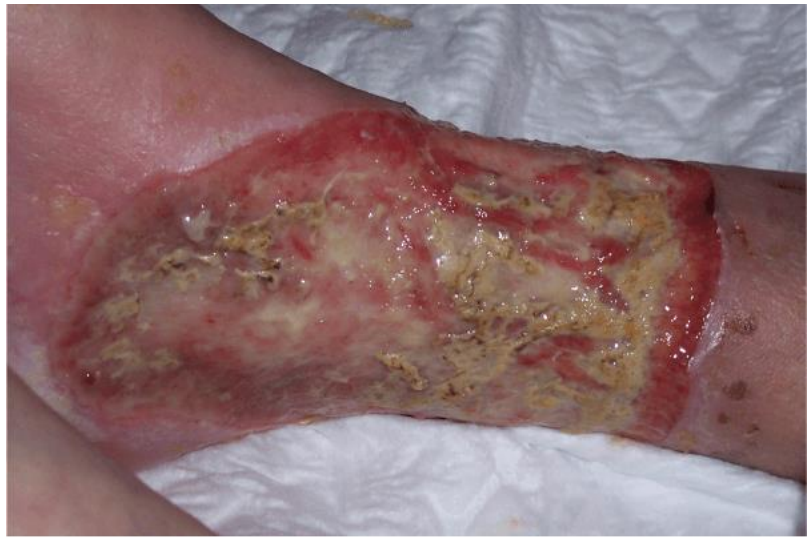
Leukocyte recruitment phase. Neutrophil activity results in expression of PGDF, VEGF.

Collagen deposition occurs; over-accumulation of this can change base from granular to fibrous (fibroslough).

Devitalized leukocytes also often present on wound's surface, frequently mistaken for purulence (leukoslough).

If factors are not appropriate within wound base to advance to proliferation, a wound remains generally inflamed.





https://www.woundsource.com/blog/four-stages-wound-healing

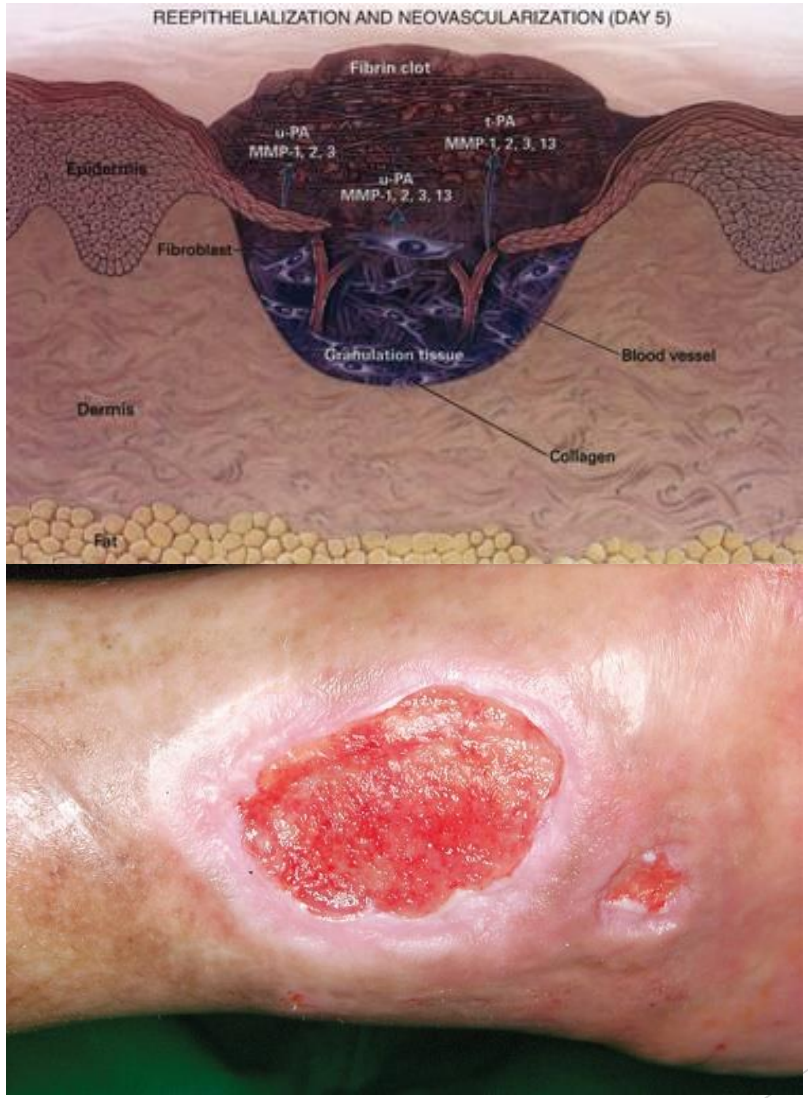
Proliferative phase

Epidermis begins to grow across granular base (or under scab in a non-chronic wound).

Generally advances at a rate of 1 mm every 2 days

This phase continues until the wound is closed or the cycle is interrupted

Type 3 collagen deposited, causes prominence of scar



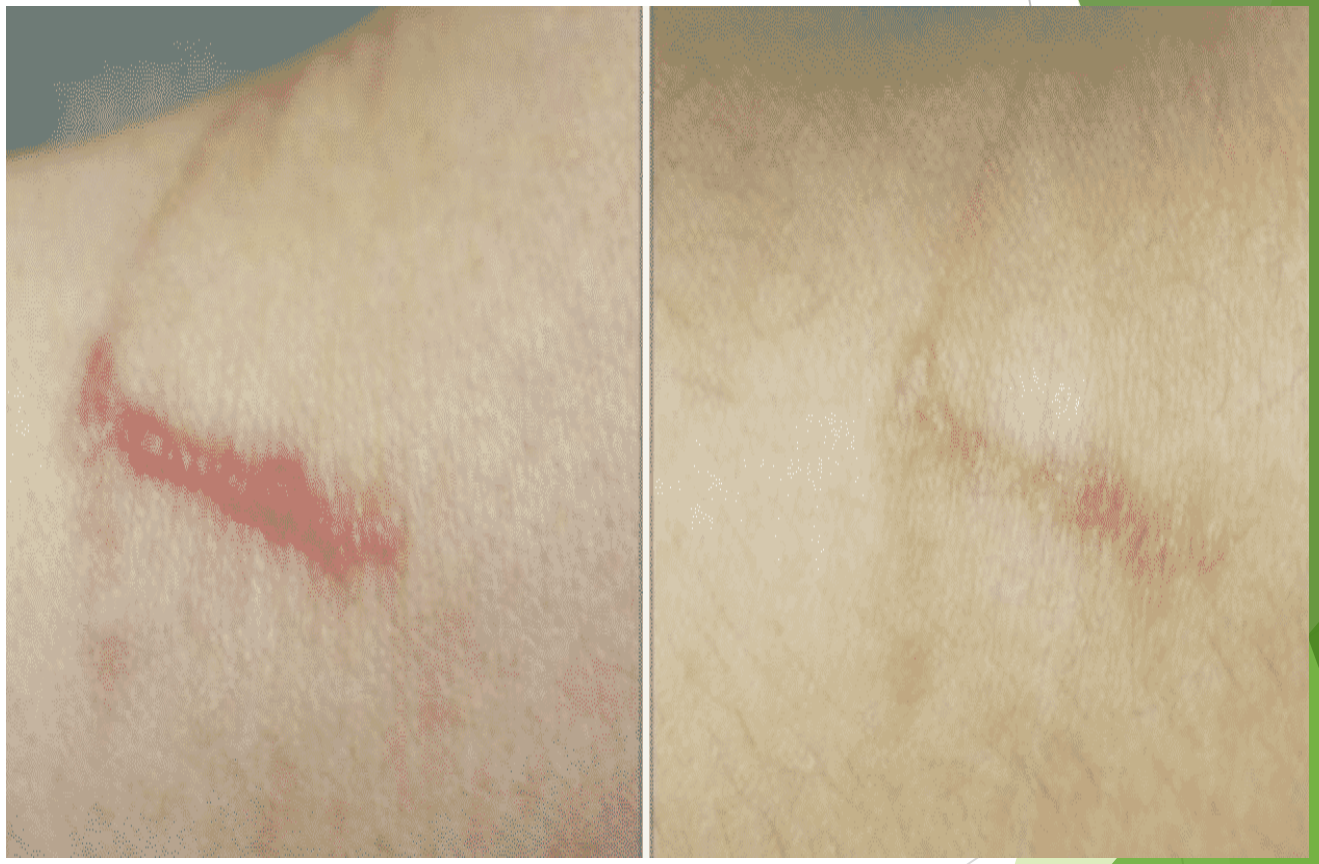
https://www.coloplastprofessional.co.uk/advanced-wound-care/advanced-wound-care-knowledge/wound-healing/

Remodeling (maturation) phase

Collagen reformation takes place(type 3 to type 1)

Scar tissue is thinned, realigned along tension lines, water reabsorbed, fibers move closer together and cross-link

Matrix metalloprotease presence is normal in this phase



https://www.theplasticsfella.com/hypertrophic-vs-keloid-scar/

What should a healthy wound look like?

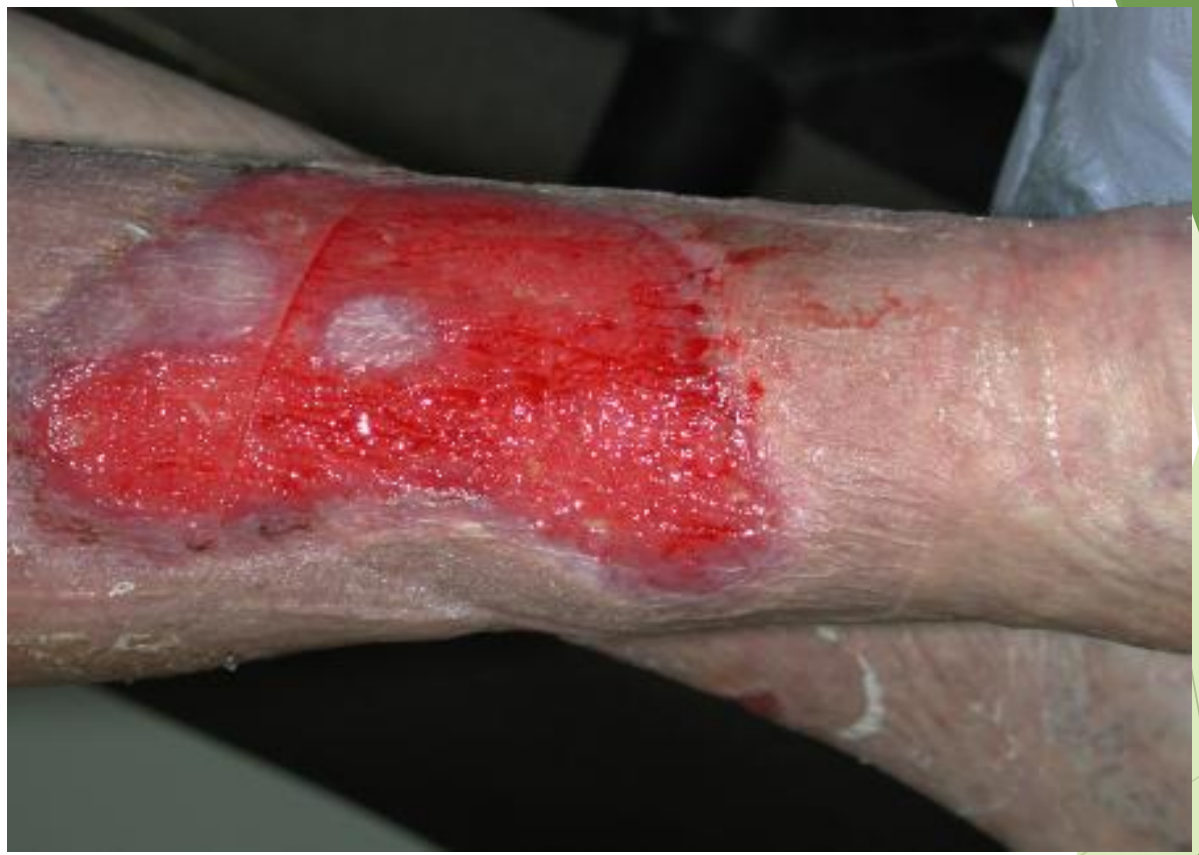
Robust granulation to base

Clearly proceeding epithelium at margins

Pain steadily decreases, very little after initial injury

Drainage is clear yellow (serous), clear pink (serosanguinous) or red (sanguinous)

Leukoslough or fibroslough may be present



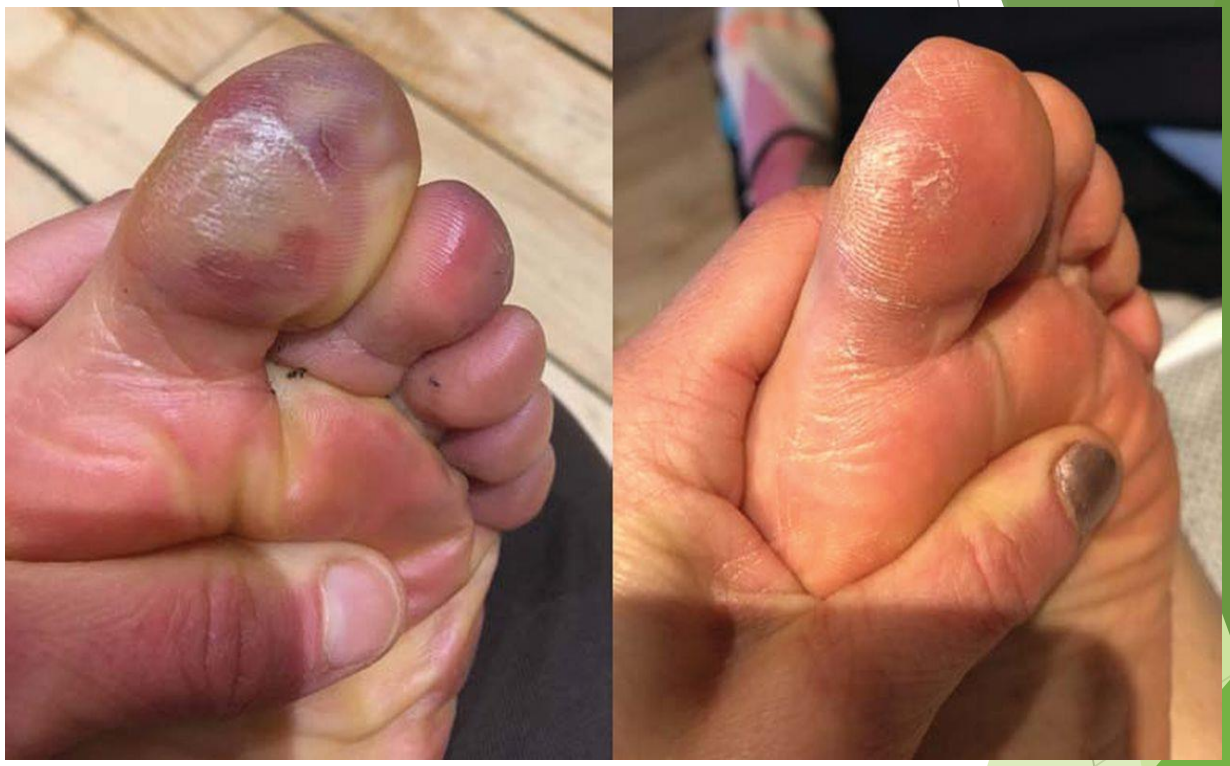
http://www.worldwidewounds.com/2002/april/Vowden/Wound-Bed-Preparation.html

What factors delay healing?

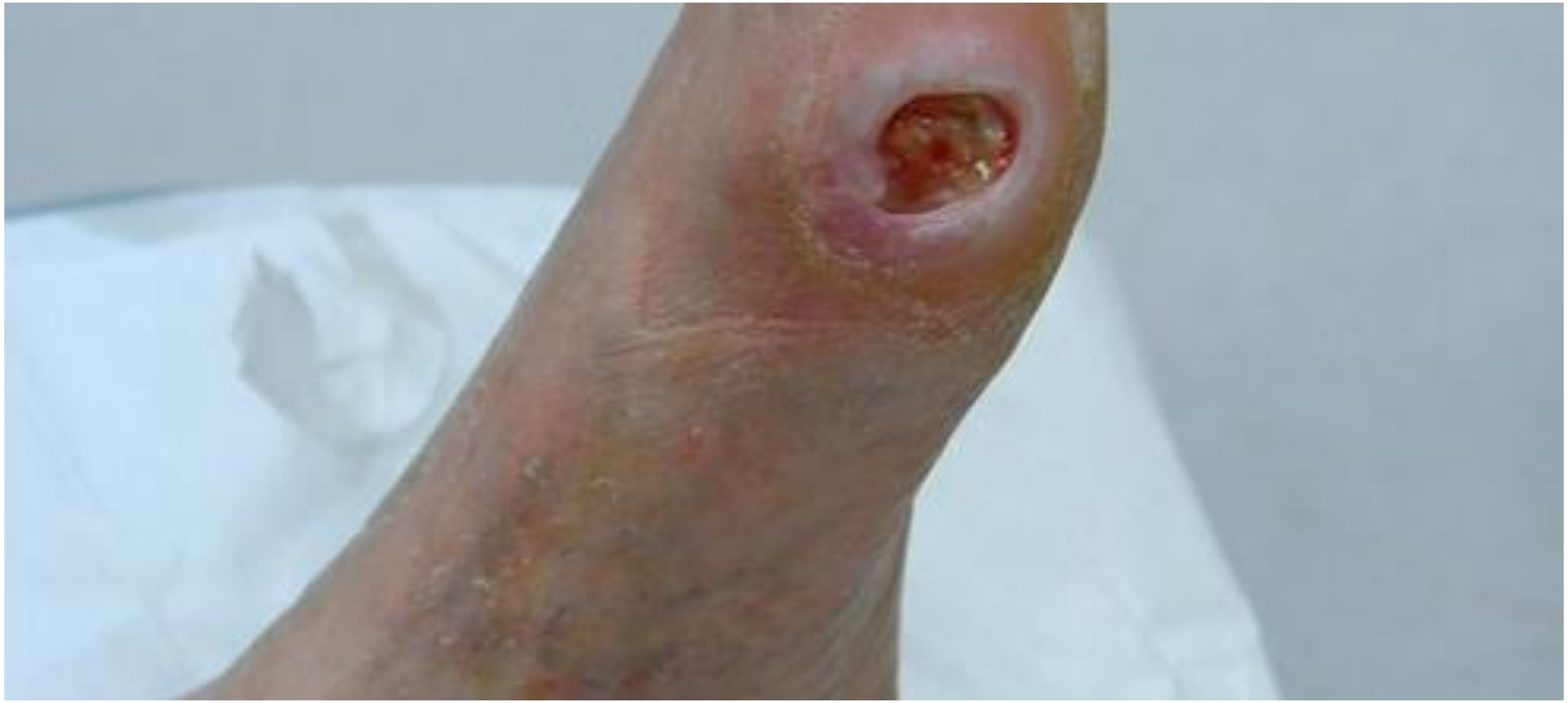
- Impaired perfusion
- Edema
- Presence of non-viable tissue
- Bioburden
- Pressure accumulation
- Microenvironment
- Nutrition/host factors

Perfusion – how do we know if it's adequate?

- If wound is to trunk (non-bony prominence), assess tissue quality (hypoxia, capillary refill, ischemic pain)
- If wound is to limbs, consider ankle/brachial or toe/brachial index (ABI/TBI), transcutaneous oxygen measurement
- Edema impairs perfusion – profound, uncontrolled edema presents obstruction to normal healing because it blocks arrival of nutrients, growth factors, leukocytes. TCOM measurements to edematous limbs reflect critical limb ischemia – if uncontained, wound won't improve



https://www.jrheum.org/content/48/4/616

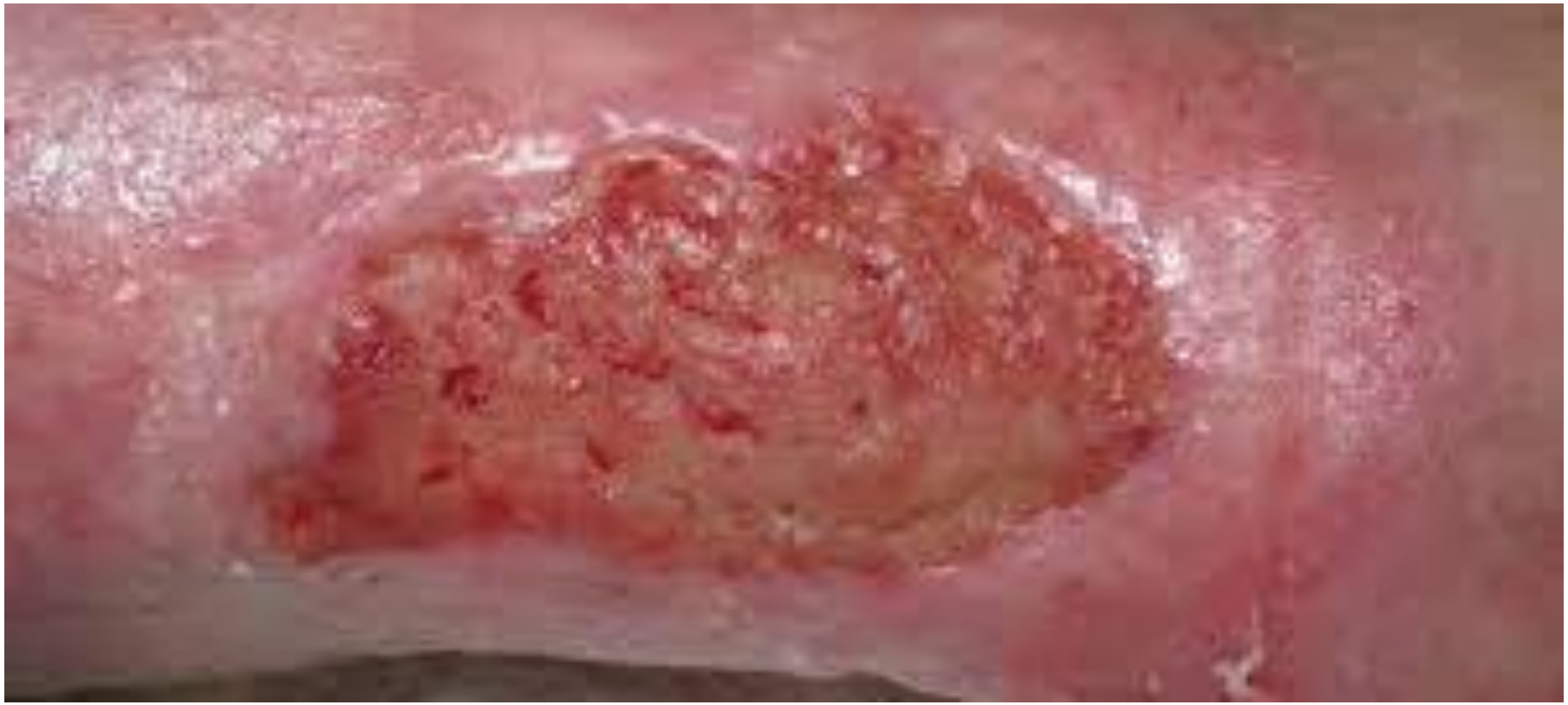


Arterial ulceration – “punched out” appearance

https://www.vascularsociety.org.uk/patients/conditions/12/arterial_ulcer

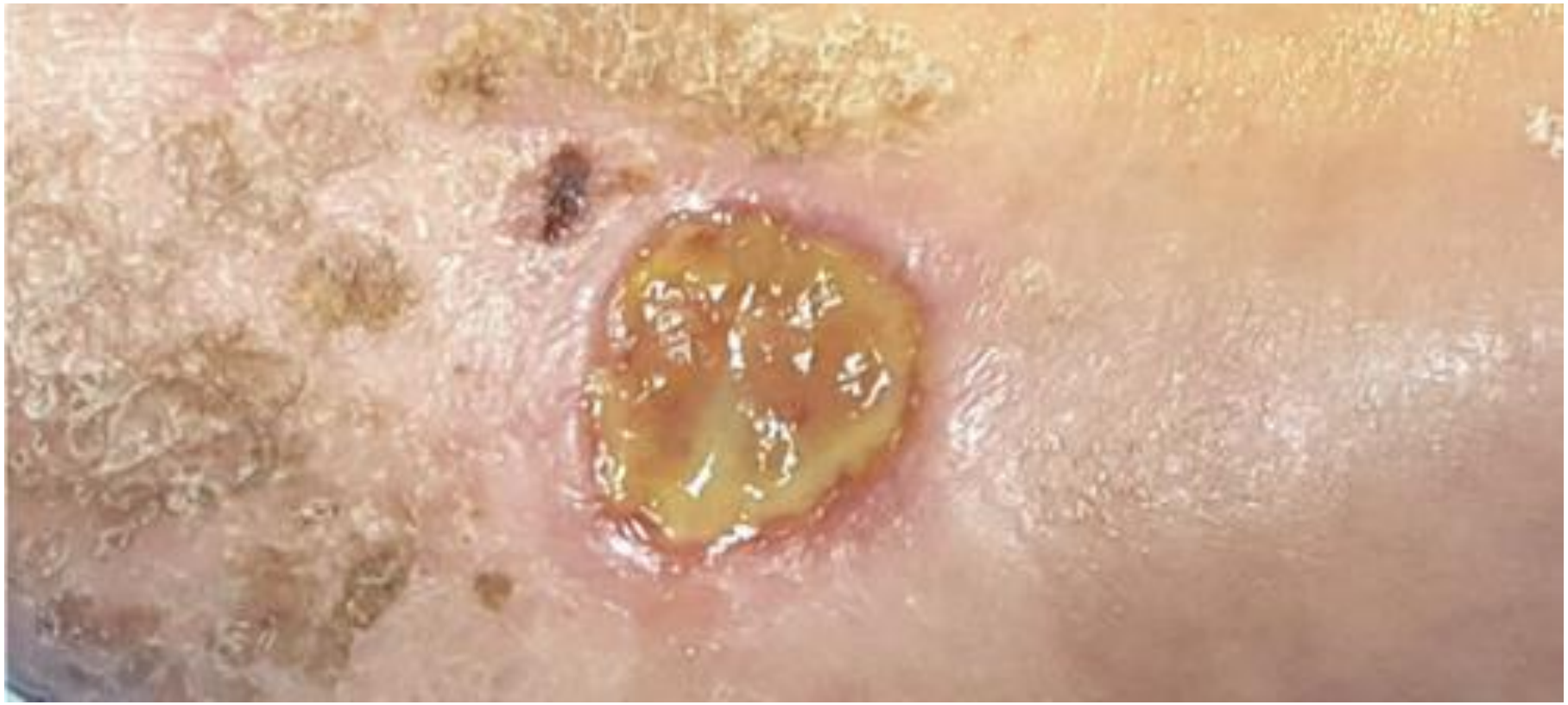
Presence of non-viable tissue

- Fibroslough – Dense, tough collagen layer deposited during inflammation. Difficult to remove, may indicate stalling ulcer
- Leukoslough – Accumulation of devitalized WBCs, protein, exudate. Loose, yellow, “custard-like”, easily removed with gauze. Normal part of wound healing, expected finding in autolytic debridement
- Bioslough – Not technically visible. Biofilm formation/chronic colonization to wound surface
- Necroslough – Eschar, unindentifiable devitalized tissue. Exudate is highly enzymatic, liquefaction necrosis present, malodorous, soft but adherent, should be debrided in stages



Fibroslough

https://www.elenaconde.com/en/fibrinous-tissue-in-venous-ulcers-what-are-we-talking-about/

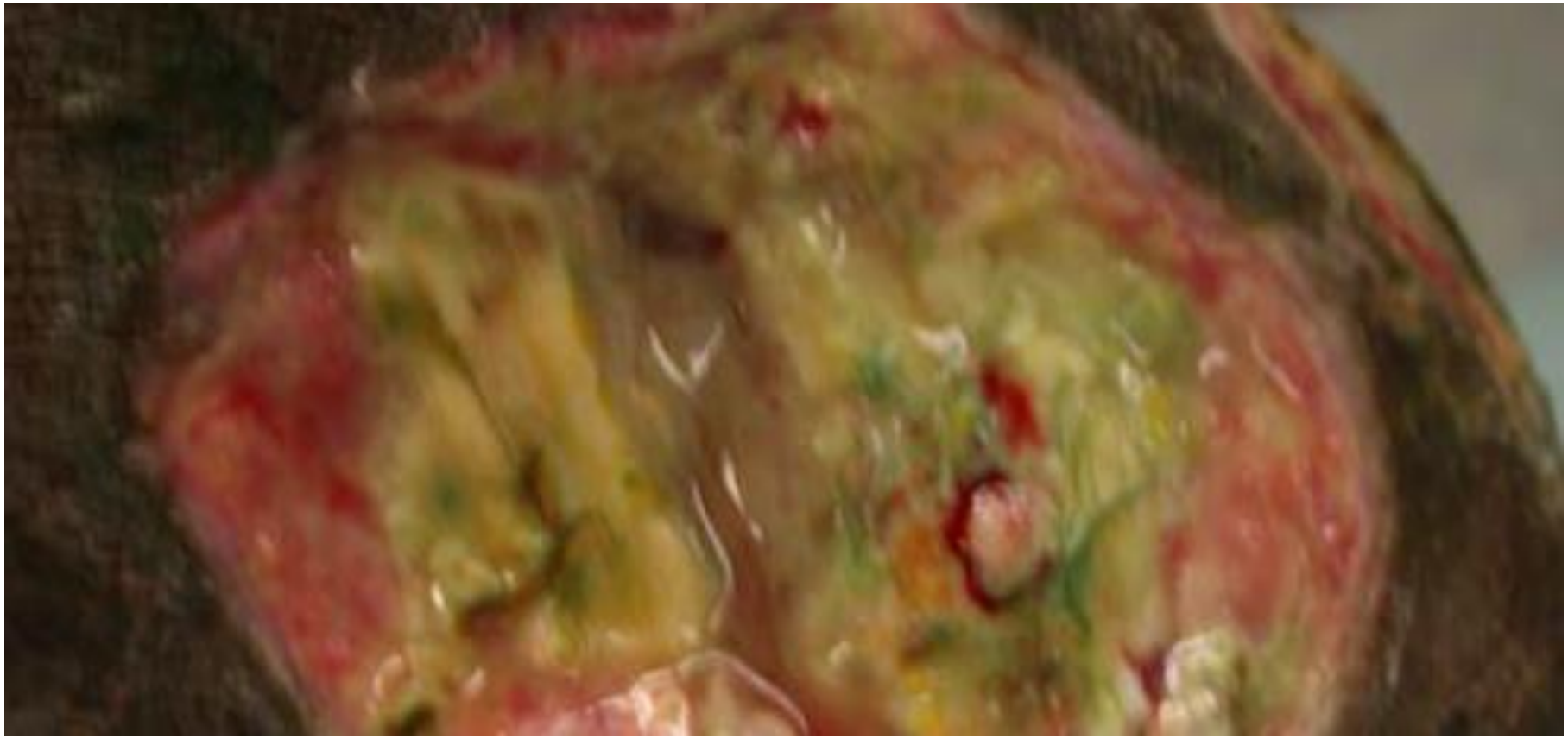


Leukoslough

https://www.magonlinelibrary.com/doi/abs/10.12968/bjon.2019.25.Sup20.S13

Bioslough

https://www.berkshirewestccg.nhs.uk/media/5263/pseudomonas.pdf



Necroslough

Methods of debridement

- Sharp excisional – Scalpel, curette or other surgical instrument used to target specific area of non-viable tissue
- Mechanical – Non-targeted physical removal off non-viable tissue (wet-to-dry bandaging). Destructive to healthy tissue, should be used in very limited circumstances
- Enzymatic – Application of collagenase in the form of ointment which accelerates breakdown of devitalized tissue
- Autolytic – Clean containment of wound site (hydrocolloid dressing) which permits WBC clearance of devitalized tissue
- Biodebridement – Application of living debriding agents (maggot therapy)

Bioburden – When should infection/colonization be suspected?

- Chronic wounds generally do not show cardinal signs of infection
- “Cellulitis” in patients with uncontrolled lower extremity edema may actually be stasis dermatitis or rebound edema. Take careful history in edematous patients
- Most common indications of infection are increased pain, larger wound, abnormal drainage (green, brown, thick yellow)

Bioburden – cont.

- Culture all wounds upon presentation, unless there is clear evidence of improvement
- Tissue culture is gold standard – cleanse area well with saline/gauze, perform debridement, take small piece of tissue from deepest wound aspect
- If performing a swab culture, cleanse area well with saline/gauze, culture *only* the deepest aspect of wound
- Z-swab method is *not* recommended, shows contamination rather than causative organisms
- Look for WBCs on culture. Unless patient is immunocompromised, no WBCs implies colonization, best treated with topical products

Pseudomonas colonization – green, malodorous

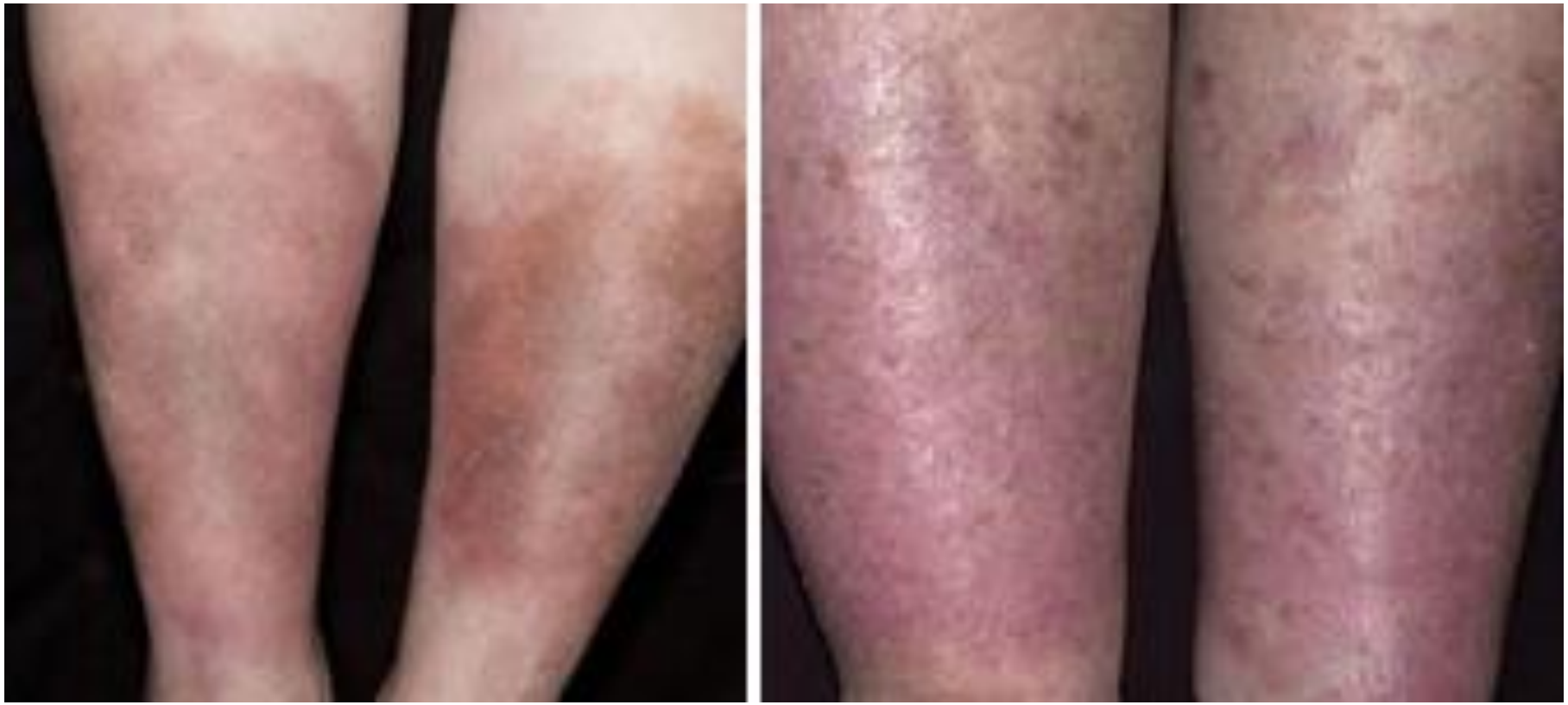
https://www.berkshirewestccg.nhs.uk/media/5263/pseudomonas.pdf

Bioburden, cont.

- Most effective part of wound cleansing is mechanical action – scrubbing with gauze
- Wound cleansers should not be cytotoxic; betadine, isopropyl alcohol, hydrogen peroxide are not appropriate for wound cleansing
- Chlorhexidine is safe for occasional use
- Saline, soap and water, hypochlorous acid solutions are safe for routine use
- Consider Dakin's solution-soaked gauze if colonization is suspected

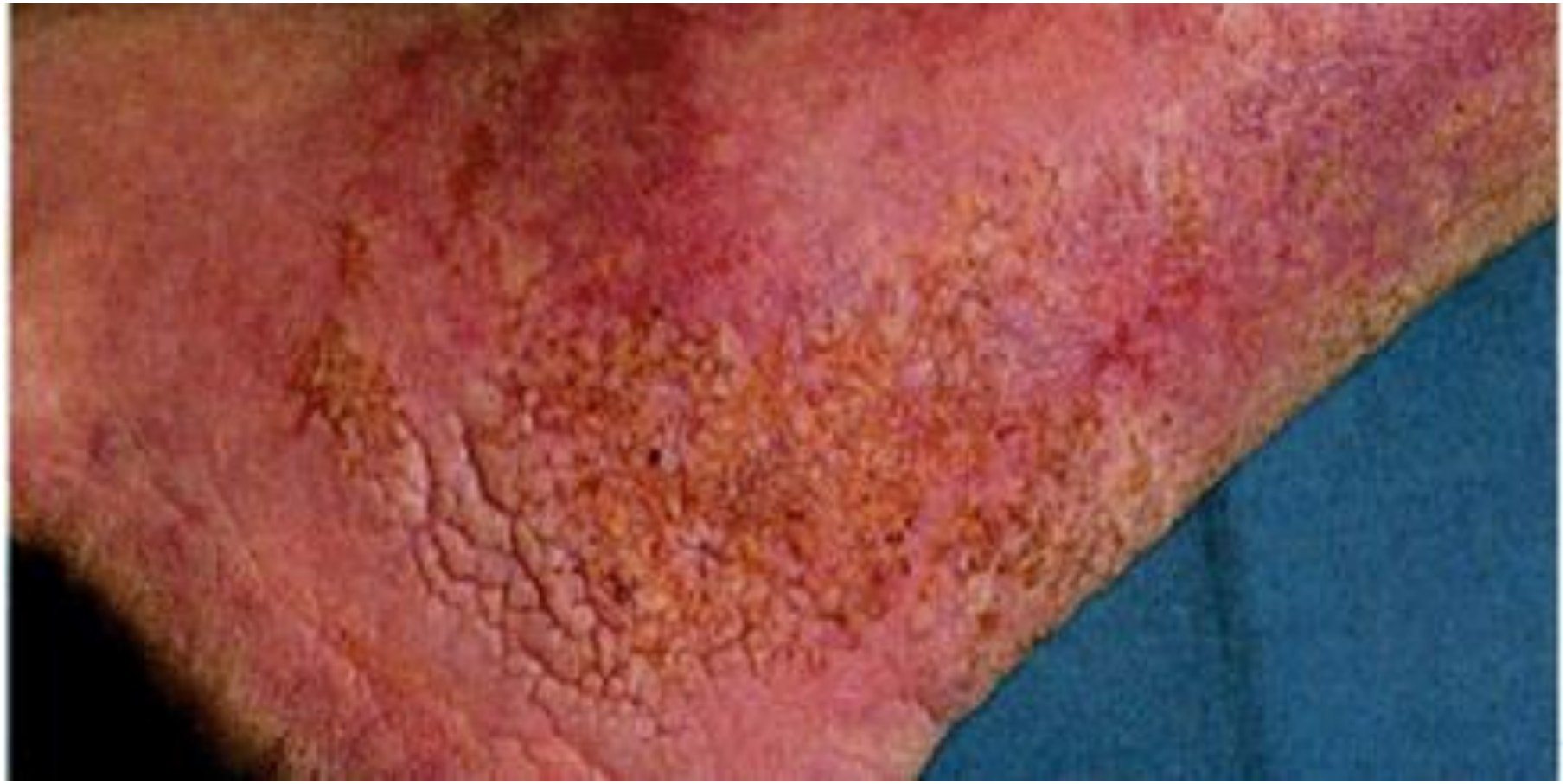
Edema

- Most common cause of lower extremity edema is chronic venous insufficiency – as many as 40% of US population in 2014
- Generally misattributed to CHF, which impacts roughly 2% of US population
- Diuretic use generally ineffective for hydrostatic edema related to vein malfunction
- Compression, therapeutic exercise, leg elevation when sedentary
- Compression bandaging for treatment, compression stockings for prevention
- Worsening lower extremity wounds in the setting of edema should be considered hypoxic – profound edema blocks appropriate perfusion
- Uncontrolled lower extremity hydrostatic edema can become lymphedema (phlebolymphedema), resulting in skin changes such as papillomatosis cutis



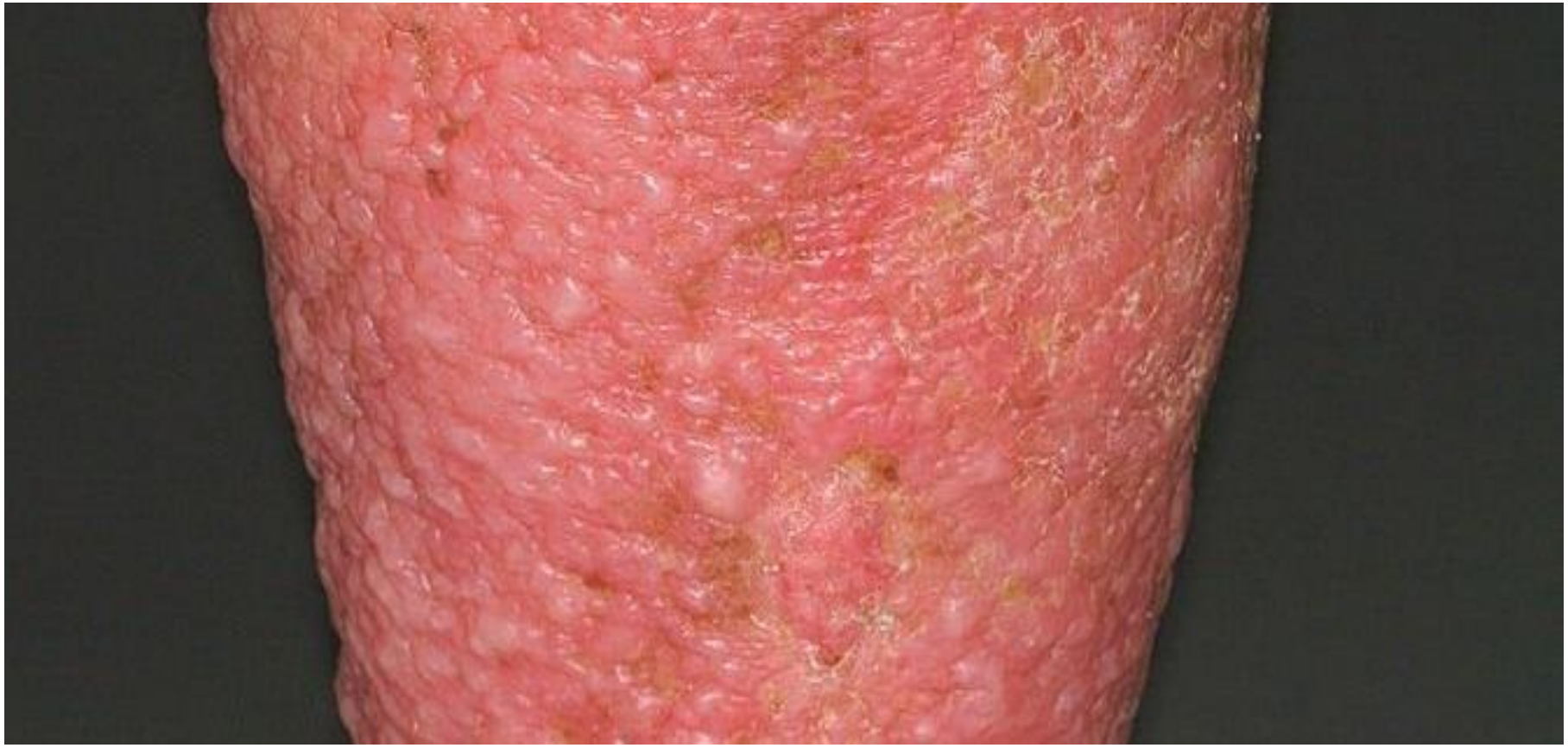
Chronic venous insufficiency

https://www.totallyfeet.net/foot-facts/foot-circulation/venous-insufficiency/



Papillomatosis cutis lymphostatica

https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1524-4725.1993.tb00371.x

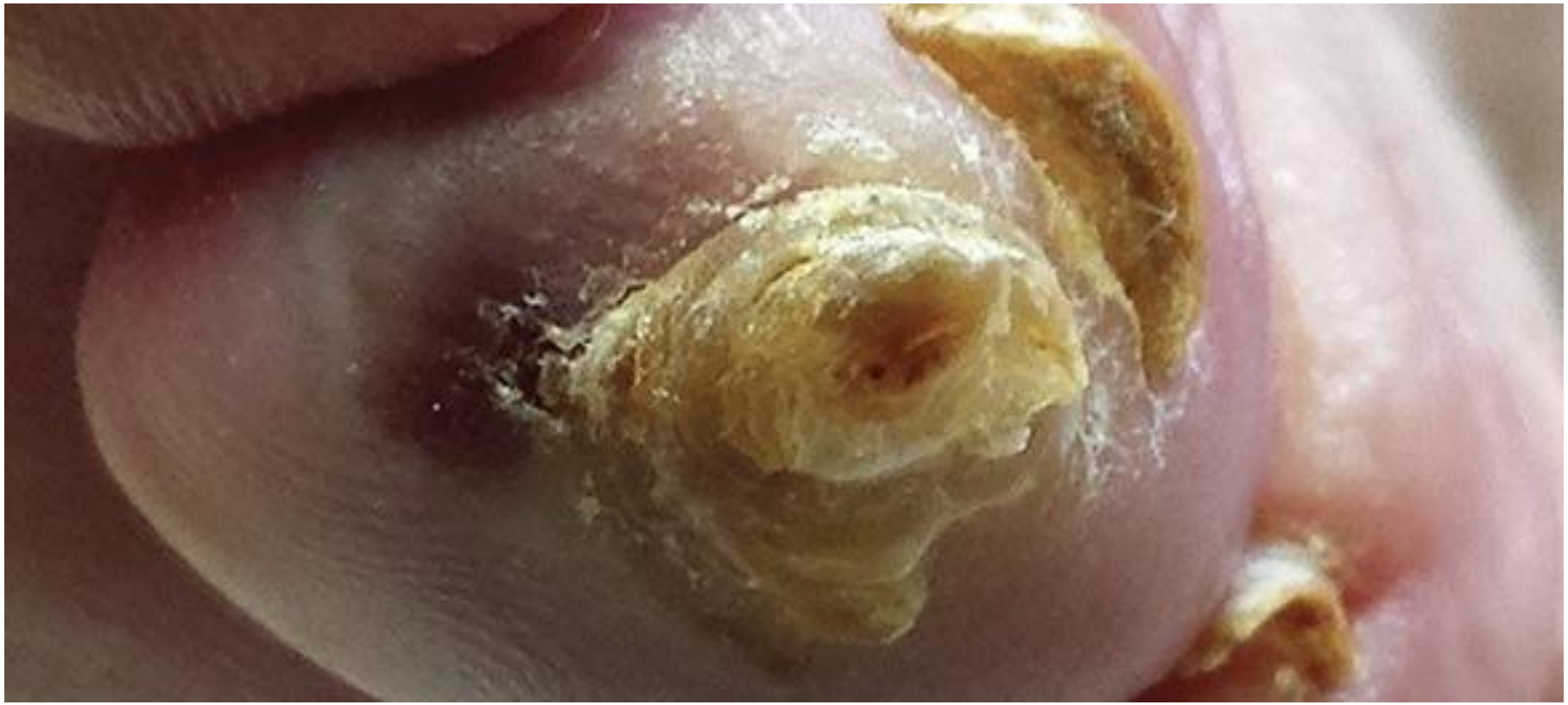


Papillomatosis/phlebolymphedema

https://www.altmeyers.org/en/dermatology/papillomatosis-cutis-lymphostatica-120593

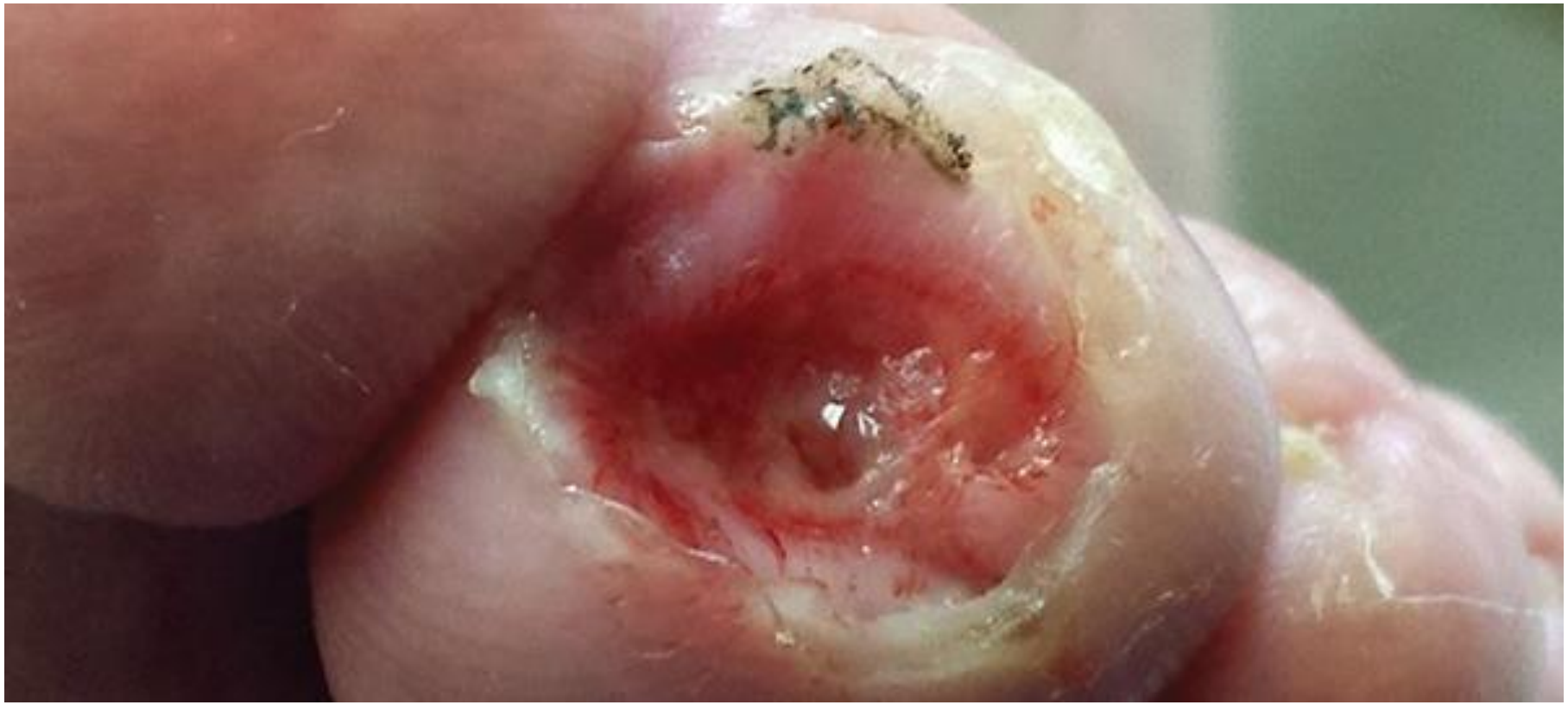
Pressure accumulation

- Pressure between bony prominence and ground/surface creates bloodflow occlusion, resulting in skin breakdown, pressure ulceration.
- Diabetic foot ulcerations take on different classification, but are functionally pressure ulcers due to lack of protective sensation.
- Callus to any ulceration represents inappropriate pressure accumulation – symptom of serious problem.
- Callus removal necessary, but re-accumulation is *guaranteed* if pressure sources are unchanged (total contact cast for treatment, diabetic footwear for daily use once ulcers are resolved)



Pre-ulcerative callus with pressure necrosis

https://lermagazine.com/article/treatment-of-distal-toe-calluses-and-ulcerations



Ulceration uncovered following paring

https://lermagazine.com/article/treatment-of-distal-toe-calluses-and-ulcerations

Nutritional concerns

- Appropriate glycemic control – tissue healing stops when CBGs are routinely higher than 200.
- Protein requirements – 1.5 g/Kg of lean protein daily in chronically wounded patients
- Aim for 3 servings of lean protein roughly the size of a deck of cards daily
- Pre-albumin is poor indicator of actual nutrition in patients with inflammatory conditions or chronic illness; dietary recall/I&O is far more accurate
- Protein intake from whole food is preferred to supplementation, if possible

Host factor considerations

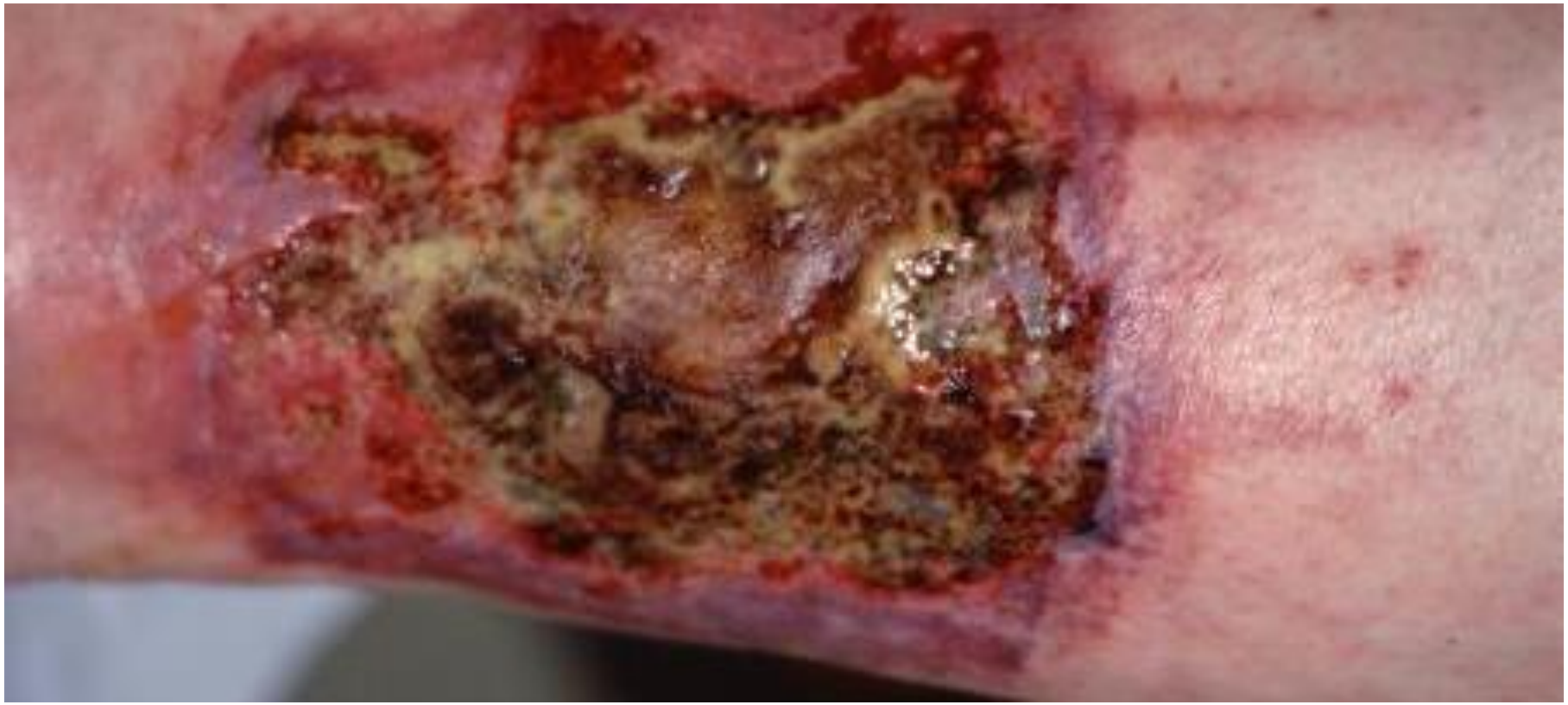
- Smoking history – tobacco is a vasoconstrictor. Even temporary cessation of use aids healing
- Immune compromise
- Blood dyscrasias

Wound microenvironment: But what do I put on it?

- Dressings do not heal wounds – they manage wound conditions
- What does the dressing do?
- What do you want it to do?
- WHY do you want it to do that?

Microenvironment – Is it too dry?

- Overly dry wounds – desiccation
- Is wound remaining uncovered?
- Consider hydrogels, medihoney, petroleum-imbued gauze

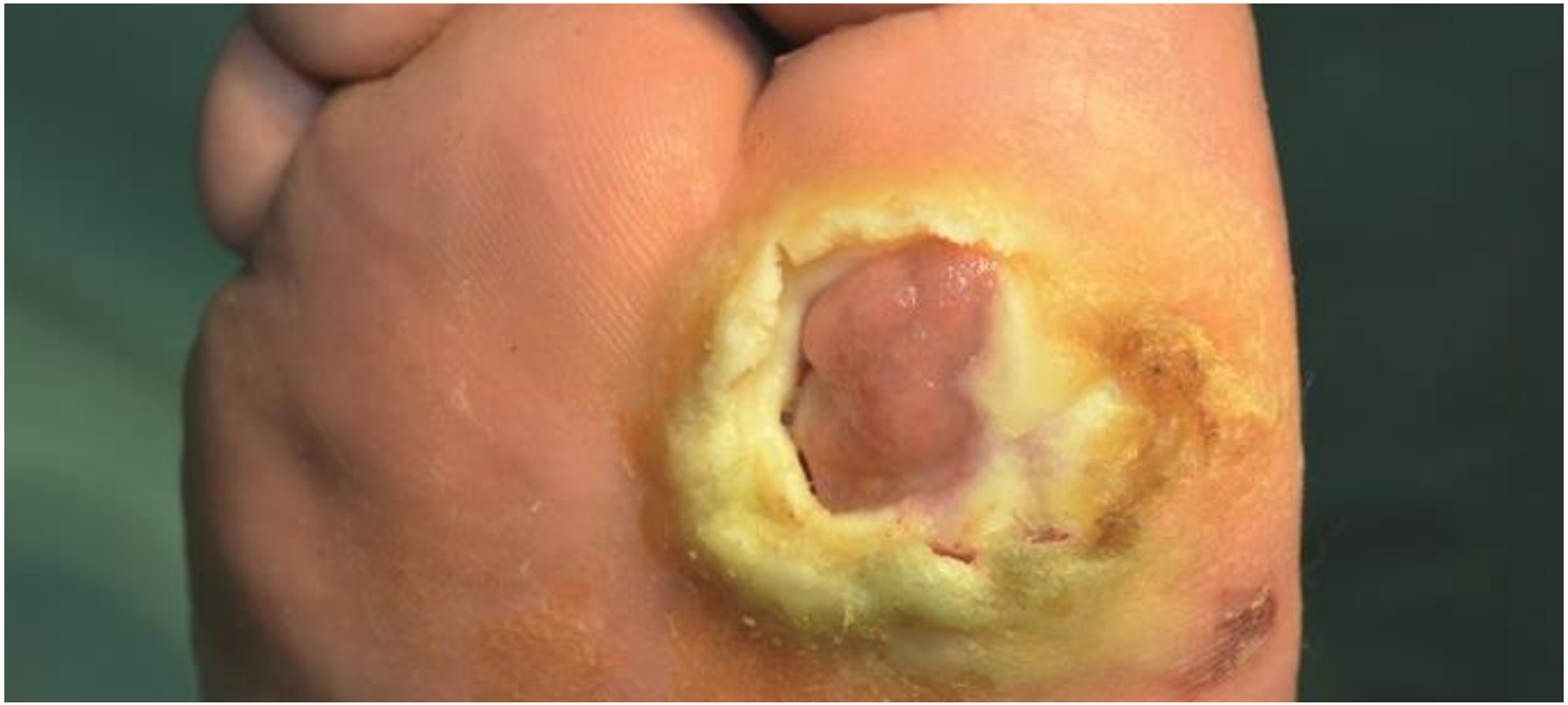


Desiccated wound base

http://www.worldwidewounds.com/2002/april/Vowden/Wound-Bed-Preparation.html

Microenvironment – Is it too wet?

- Overhydration – maceration
- Is edema controlled?
- Has culture been performed?
- Consider hydrofibers, alginates, superabsorbers
- Is compression appropriate?

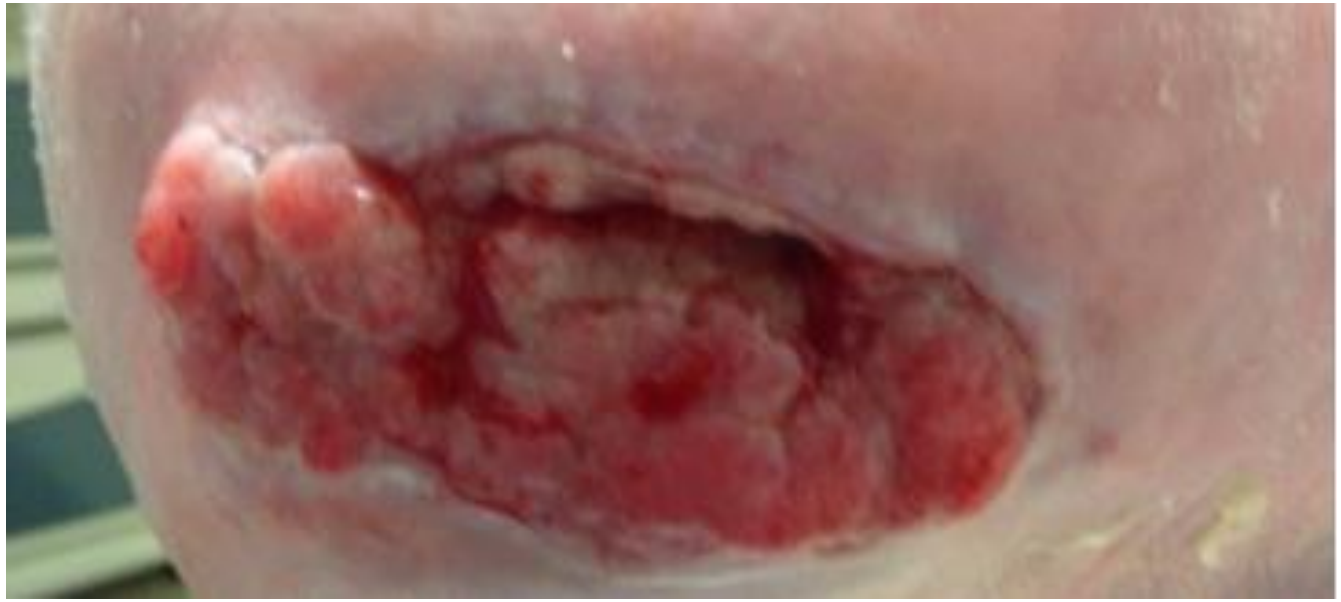


Maceration/overhydration

https://twitter.com/coloplast_wcare/status/950342857684148226?lang=ru

Microenvironment – Is there risk for bioburden?

- Is wound likely to become contaminated?
- Is hypergranulation present?
- Perianal ulcerations, foot ulcerations, lower extremity ulcerations
- Consider bacteriostatic dressings; cadexomer iodine (NOT the same as betadine), silver-imbued dressings, methylene blue/gentian violet dressings
- When in doubt, ask your wound nurses



Hypergranulation

https://sanaramedtech.com/blog/how-to-identify-treat-hypergranulation-tissue/

Summary:

- Appropriate management of chronic wounds requires clear understanding of *why* the wounds are chronic
- Patient education regarding these factors is essential to lasting wound resolution
- Consider your resources as a provider: WOCNs, PT/OT, LLT, podiatry, orthotists
- Refer to outpatient wound management as needed – we're here to help

Questions?

References:

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