A Guide to Wound Care

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Wound Care Program

Patient History

- Current wound / skin regimen
- Previous history of wounds, and wound therapies/interventions
- Co-morbidities (Diabetes, CHF, PAD, Autoimmune dx/SLS)
- H/O current wound: duration, changes in size

Physical Examination

- **Site**
  - Side, anatomic location
  - PULSE EXAM of extremity, Doppler in absence of palpable pulse
- **Size**
  - Length x width x depth; presence of tunneling, undermining
  - Wound base characteristics: Granulation, fibrous tissue, exposed structures, necrosis
- **Signs of infection & severity**
  - Purulence, odor, erythema, induration, crepitus

Diagnostic Studies

- Ordered per disease state

Treatment

- **Consultations**
  - Plastic surgery, Podiatry, Vascular surgery, General surgery, Orthopedics, Internal Medicine, Infectious Diseases, Rheumatology, Dermatology, Nutrition
- **Manage Infection**
  - Debridement of necrotic / non-viable tissue, control of bio-burden
  - Antibiotics only as warranted / Infectious Diseases team

Wound Care

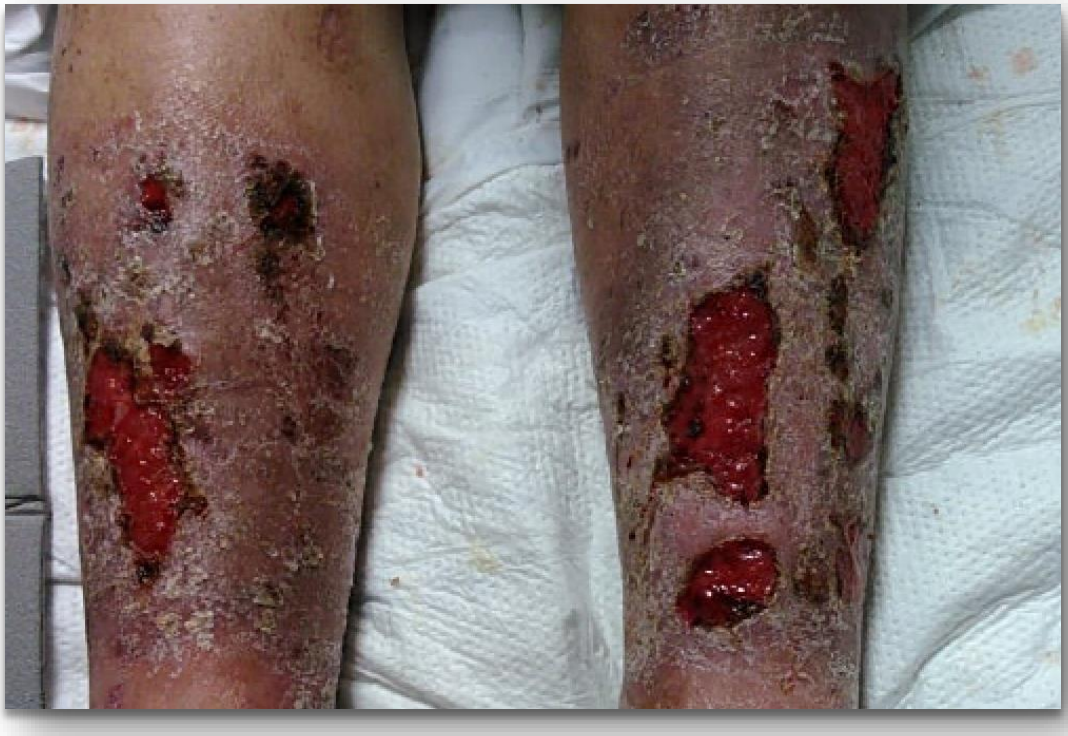
- **Desiccants:** gentian violet (Hydrofera blue); iodine (Iodosorb, Iodoplex); alginates (Maxsorb); hydrofiber (Aquacel)
- **Debriders:** enzymatic (Santyl/collagenase); mechanical (wet/dry); autolytic (Medihoney); surfactant (Plurogel)
- **Foam** (Mepilex, Allevyn)
- **Collagen** (Puracol, Endoform)
- **Skin substitutes/ growth factors:** Apligraf; Dermagraft; EpiFix; Restrata, Puraply

(Multiple agents have antimicrobial properties: Prontosan; Plurogel; iodine; silver; Acetic Acid, Medihoney)

Surgical Care

- Debridement, revascularization, skin grafting, level-sparing amputation, flap coverage

1. Venous Stasis Wounds



1.1 Description

- Fibrogranular tissue and granulation along wound base, crusting along margins, with periwound brawny discoloration, extensive superficial scaling
  - — Common findings: cramping pain, palpable pulses, hemosiderin staining at ankle
  - — Frequently found with surrounding induration and significant edema
  - — Patient history may report significant drainage, edema, and limited pain
- Consistent with Venous Stasis Disease

1.2 History

Etiology: Due to valvular dysfunction or calf muscle dysfunction

- Risk Factors: Obesity, IVDU in lower limbs, thrombosis/trauma, thrombophlebitis, varicose veins, pregnancy, sedentary lifestyle, smoking, conditions that inhibit mobility

1.3 Physical Exam

- Location, dimension (length x width x depth)
- Wound base presence of granulation, exposure of tendon, bone, hardware
- Signs of bacterial overgrowth: strong odor, large amounts of drainage, erythema
- Vascular exam: palpable pulses of the PT and DP, Doppler pulse exam
  - — Differentiate dependent rubor from cellulitis
  - — Common findings: Hemosiderin staining at ankle, ulcer irregular with fibrogranular base that varies in slough and depth, usually highly exudative. Surrounding tissue often shows evidence of venous dermatitis, lipodermatosclerosis (waxy, hard fibrotic tissue), malleolar flare (sunburst capillaries inferior and distal to medial malleolus), and atrophie blanche lesions (atrophic porcelain scars that appear after healing, highly susceptible to deterioration)

1.4 Pertinent Imaging / Diagnostic Studies

- Basic labs if acute / ED presentation: CBC, CMP, ESR/CRP, HgA1C
- Imaging: Plain radiographs, evaluate for periosteal erosion or reaction
  - — Duplex with US: Gold standard to assess for venous reflux
  - — Dx criteria includes:
    - 500 ms of retrograde flow for superficial/perforator veins
    - 1000 ms of retrograde flow for deep veins
  - — For patients with equivocal or normal doppler US findings, can use multi-detector computer venography and MR venography
- Arterial component / mixed etiology ABI / PVRs

1.5 Treatment

- Education for patient/family: Compression, elevation of legs at rest, PT for dorsiflexion, weight control, skin care
- Compression therapy

  (Do not use compression if ABI is < 0.5, uncontrolled CHF, active infection)

  - — Varicosities Class 1: 20 - 30 mmHg
  - — Venous ulcers/prevention Class 2: 30 - 40 mmHg
  - — Refractory ulcers/lymphedema Class 3: 40 - 50 mmHg
  - — Lymphedema Class 4: 50 - 60 mmHg

  Class 2 – 4 compression (30 – 60 mmHg) if ABI > 0.8

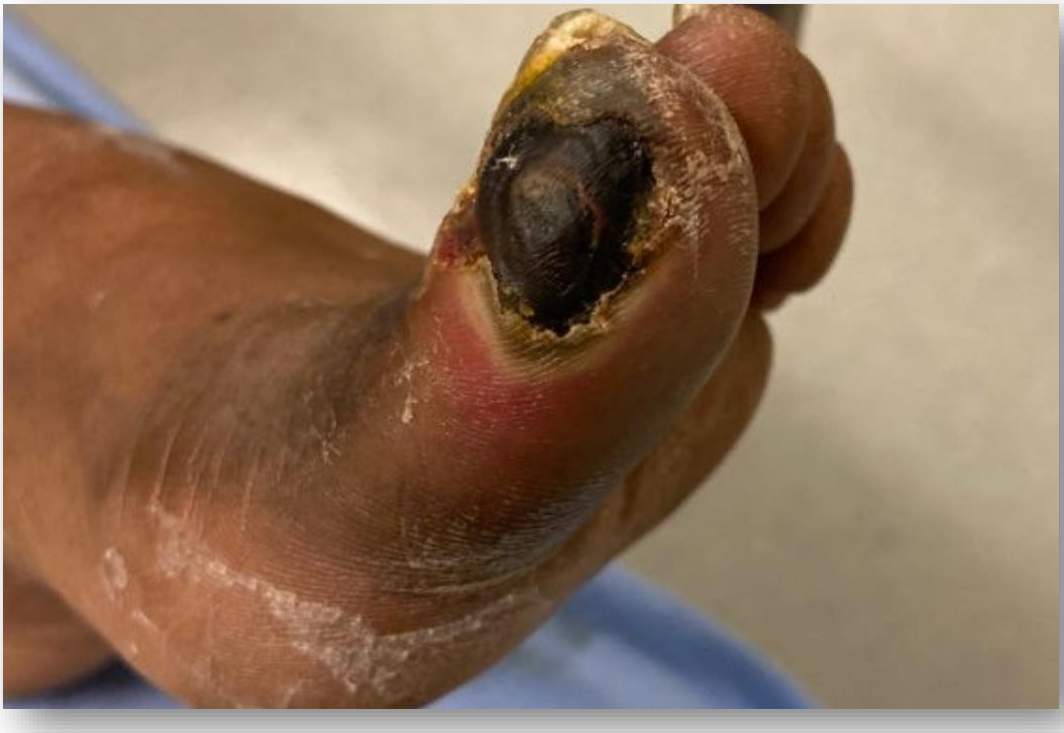
1.6 Wound Care

- Control exudate: alginates/hydrofiber (Maxsorb, Aquacel, Opticel) or foams
- Layered compression wrap (2-4 layers) combination of both elastic and non-elastic layers
- Single layer
    - SurePress: Used early in treatment, can be reused
- Non-elastic
    - Unna boot: Zinc paste wrap, only for ambulatory patients
    - Short-stretch: ambulatory patients with borderline ABI: > 0.5 - < 0.8
- IPC (intermittent pneumatic compression) therapy:
    - If static compression is ineffective or patient is immobile
    - Treatments occur 1-2 times/day for 1-2 hours/session

1.7 Surgical Management

- Endovenous laser ablation (EVLA): Percutaneous technique to ablate incompetent superficial veins.
- Target veins: great saphenous, small saphenous, and accessory saphenous
    - Low risk for infection, ulcers do not need to be healed
    - Contraindications: Acute DVT
        - Relative contraindications: chronic/recurrent phlebitis, severe tortuosity, veins less than 1 cm deep, Aneurysmal GSV segments
- Surgical Options: Debridement, staged skin grafting once compression and vascular therapy optimized

2. Ischemic Gangrene



2.1 Description

- Dry, necrotic ulceration, minimal edema or skin turgor, painful lesions (as compared to Venous stasis)
- Extremity wounds with minimal induration, absence of granulation tissue, and a fibrous tissue base
- Digital wounds may have full thickness desiccation and necrosis of the tissue
- Consistent with Arterial Disease / Ischemic Wound

2.2 History

Etiology: atherosclerosis (most common), vasculitis, thrombus

- Risk Factors: Smoking, older age, male sex, HTN, lack of exercise, DM (d/t increased plaque formation, increased blood viscosity, and hypertrophy of vasculature), hyperlipidemia (particularly LDL)

2.3 Physical Exam

- Location, dimension (length x width x depth), periwound skin integrity/quality, pulses, temperature, cap refill, assessment of pulses, and neurologic exam (motor and sensory)
- Evidence of infection
- Common findings: Pain that ranges from intermittent claudication to constant, associated with pale, cool, shiny skin with hair loss on digits and feet. Dependent rubor and elevational pallor. Non-healing ulcers are deep and circular with pale or necrotic bases with minimal marginal epithelialization. Ulcers are typically less exudative and infection is common.
    - Claudication: pain induced by exercise, relieved by rest. Varies with degree of stenosis, collateral vascular channels, and intensity of exercise
    - Ischemic Rest Pain: Due to severe lack of perfusion, typically localized to forefoot and toes, not usually controlled with analgesics
    - Severe Ischemic Pain: Numbness, paralysis of limb associated with coolness, loss of pulses, and paresthesia
    - Dry gangrene is most common in PAD: eschar formation, desiccation, hard, leathery
    - Wet gangrene: crepitus, blistering, odor, erythema, edema: <u>surgical emergency</u>

2.4 Pertinent Imaging / Diagnostic Studies

- Basic labs: routine CBC, CMP, lipid profile; can consider homocysteine, lipoprotein A, and CRP
- ABIs: Ratio of ankle SBP divided by brachial SBP
    - Normal: 1.00 - 1.09
    - Abnormal: 0.80 - 0.89
    - Markedly Reduced: 0.40 - 0.49 (pain at rest, limb threatening)
    - Critical ischemia: < 0.25
        - If ABI is > 1.3: not valid and indicates calcification of the vessel, TBI warranted
        - TBI: great or second toe, divide toe SBP by the brachial SBP
          Normal is 0.7-0.8; an absolute pressure > 30 is favorable for healing, > 45-55 may be required for healing in diabetics

- Imaging
    - — Duplex US: 1st imaging choice, useful to assess anatomic location and degree of obstruction, provides information on hemodynamics proximal and distal to obstruction
    - — Contrast Arteriography: Gold standard for the evaluation of a threatened limb and for all patients expecting to undergo revascularization

2.5 Treatment

- Lifestyle Modifications: Cessation of smoking, adequate hydration, pain control, graduated walking programs, avoid vasoconstriction, skin inspection, properly fitted shoes, foot and nail care, avoid hot water bottle / heating pads, report injuries ASAP
- Pharmacologic treatment: Antiplatelet agents, vasodilators, antilipemics, hemorrheologics, anticoagulants, thrombolytics, and analgesics

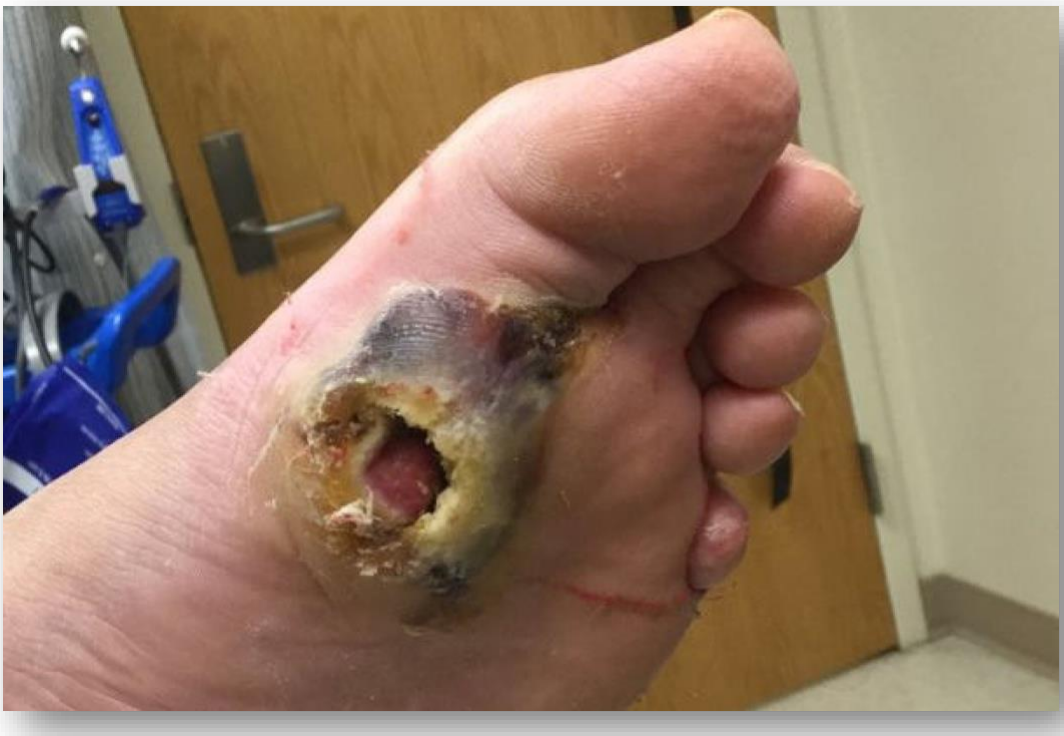
2.6 Wound Care

- Avoid debridement until perfusion has been re-established
- Topical agents that have antimicrobial properties: Prontosan, Plurogel, Silvasorb, Medihoney
- Painting with iodine is often used on non-surgical candidates to promote desiccation and prevent conversion to wet gangrene
    - — Monitor wound closely for deterioration following revascularization for risk of wet gangrene conversion (First week following procedure, upwards of 4-6w from intervention)

2.7 Surgical Management

- Surgical Indications
    - — Poor healing with ABI < 0.50
    - — Failure to respond to conservative treatment with an ABI > 0.50
    - — Intolerable pain
    - — Limb threatening ischemia
- Surgical Interventions
    - — Bypass grafts
    - — Angioplasty
    - — Amputation

3. Neuropathic Wound (Wound in the setting of peripheral neuropathy or severe DM)



3.1 Description:

- Ulcer at the level of the metatarsal head with deep undermining, exposure of muscle, patchy necrosis, hyperkeratosis/ callous formation, and hemorrhages within callus
    - — Common findings: wound with various stages of healing and active granulation; pin-point hemorrhage in callus indicate repetitive trauma; foreign material and infection
    - — Frequently found with Achilles equinus gait resulting in metatarsal head ulcer
    - — Patient history may report the absence of pain
- Consistent with Neuropathic Ulcer

3.2 History

Etiology: Combination of loss of glycemic control, peripheral neuropathy, peripheral vascular disease and immunosuppression

- Risk Factors: Neuropathy (sensory loss results in repetitive trauma, loss of autonomic moisture regulation results in dry, fissured skin), peripheral vascular disease, poor DM control (elevated HgA1C), smoking

3.3 Physical Exam

- Location, dimension (length x width x depth), periwound skin integrity/quality, pulses, temperature
- Charcot Arthropathy
    - — Progressive bony deformity which weakens and changes bone architecture, allowing for frequent/easy fractures. Lack of sensation leads to continued walking on the fracture with additional damage and deformity
    - — Full etiology unclear though combination of change in vascular tone d/t neuropathy and increased bony resorption
    - — Joint dislocation eventually causes collapse of the arch creating the rocker bottom deformity of Charcot
- Polyneuropathy assessment for loss of protective sensation
    - — Semmes-Weinstein 5.07 gauge Monofilament (10 grams of force to buckle)
    - — 128 Hz tuning fork:
        - o Placed at IP joint of hallux to test for vibratory sensation
    - — Ipswich Touch Test:
        - o Light touch at distal tufts of 1, 3, and 5 digits for 1-2 secs
- Evidence of infection (common in neuropathic ulcers)
- Common findings: Usually non painful due to loss of sensation. Ulcers are circular, well defined with thick callous formation usually located on the plantar foot, metatarsals, toes, or side of foot. Ulcers usually present as dark red tissue bases, with varying degrees of exudate and odor.

3.4 Pertinent Imaging / Diagnostic Studies

- Labs: CBC, CMP, ESR, CRP. Recently some studies have suggested procalcitonin, clinical utility has yet to be fully investigated
- Imaging: Plain films initially. MRI if osteomyelitis (OM) is suspected but not obvious on films and/or evaluation of soft tissue abnormalities is needed
- ABIs / PVRs

- Suspected Osteomyelitis:
    - Probing/exposure of bone should prompt immediate evaluation for OM
    - If wound over a bony prominence fails to heal despite adequate offloading, OM should be suspected
    - Plain films: 28-75% sensitive, 64% specific: cortical erosion, periosteal reaction, lucency and sclerosis
    - MRI: 77%-100% sensitive, 40-100% specific: cortical destruction, bone marrow edema, and soft tissue inflammation
    - Confirmation of dx must be done via bone biopsy (histology)

3.5 Treatment

- Education for patient/family
- Low risk patients should be educated on proper foot care, footwear recommendations/ orthotist evaluation, monitoring and maintenance of glycemic control
    - HgA1C < 7% reduces the risk for microvascular disease
- High risk patients may need surgical intervention
- Offloading
    - Total contact cast (TCC): Walking cast that equalizes pressure of the plantar surface
    - Removable Cast Walker
    - Half shoes (Orthowedge)/ post-op shoes

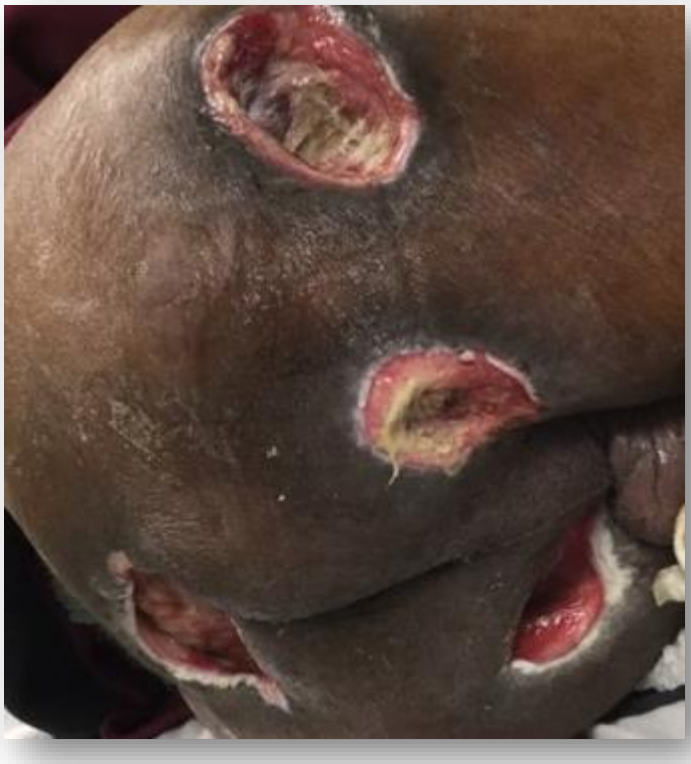
3.6 Wound Care

- Control exudate: alginates, hydrofiber or foams
- Chemical Debridement: Santyl, clostridial collagenase
- Other topical choices include silver impregnated dressing, cadexomer iodine gel
- Skin substitutes (human or bioengineered: Dermagraft, Apligraf, EpiFix, Restrata)

3.7 Surgical Management

- <u>EMG to determine status of polyneuropathy verses tarsal tunnel</u>
- <u>Forefoot wounds frequently associated with Achilles glycosylation/shortening and equinus gait</u>
    - <u>Consider Achilles lengthening</u>
- Surgical or end-vascular revascularization
    - Patients with moderate to severe ischemia and deep wounds benefit from revascularization
- Correction of foot deformities/foot reconstruction
- Amputation

4. Sacral and Ischial Pressure Wounds



4.1 Description:

- Wound secondary to tissue ischemia and pressure necrosis. Based upon the stage, ulcers typically with a moderate amount of serosanguinous drainage and odor
  - — Common findings: tunneling, maceration, eschar formation, exposed bone
  - — Stool soilage may be an issue with sacral and ischial wounds
- Wounds to the sacrum, ischium, and trochanter consistent with Pressure Injury (Stage 4)

4.2 History

Etiology: Unrelieved pressure resulting in ischemia to tissues, necrosis, and breakdown of the tissue.

- Risk Factors: Age, decreased mobility, sensory impairment, co-morbidities (renal disease, DM, thyroid disease, vascular disease), corticosteroid use, impaired circulation, cognitive impairment, fecal/urinary incontinence, malnutrition/dehydration, spinal cord injury (SCI), muscle spasms

- At risk populations for pressure injuries:
  - Advanced age: 3.5% - 69%
  - SCI: Up to 85%
  - Hospitalized children
  - NICU: 3.9 - 16%
  - PICU: 0.8 - 27%
  - Surgical Patients: 4 - 45% depending upon age, nutrition, co-morbidities, length of surgery

4.3 Physical Exam

- Location, dimension (length x width x depth), periwound skin integrity/quality, pulses, temperature, cap refill, evidence of infection
- Presence of spasms, incontinence, moisture, mobility, sensory loss
- Pressure Injury Staging:
  - **Stage 1**: non-blanching erythema over a bony prominence, skin remains intact, may note a temperature difference.
  - **Stage 2**: Shallow crater or an intact fluid filled (serous in color) blister over a bony prominence. Represents partial thickness tissue loss.
  - **Stage 3**: Full thickness tissue loss with involvement of the subcutaneous tissue layer. May see undermining.
  - **Stage 4**: Full thickness tissue loss with exposed tendon, muscle, and/or bone, often accompanied by tunneling and undermining. OM is likely.
  - **Unstageable**: Thick eschar/slough (black, brown, grey, yellow) obscuring true depth of wound bed. The base of the wound will not be visible.
  - **DTI** (Deep tissue injury): Localized purple/maroon tissue over a bony prominence. Boggy to palpation, can be difficult to identify in darker skin tones.

4.4 Pertinent Imaging / Diagnostic Studies

- Labs: Nutritional Status: transferrin, serum albumin and prealbumin, CBC, CMP, HgA1C, CRP/ESR
  - ESR: Sensitive to OM
- Imaging:
  - Bone involvement: plain radiographs and MRI to evaluate underlying OM
  - If possible arterial component/mixed etiology: ABI / PVRs

4.5 Treatment

- Education for patient/family: Critical to impart that prevention is key
- Optimization of medical status
    - Nutrition: supplements as needed
    - Control/eliminate muscle spams
    - Contracture prevention: PT/OT, tenotomy
    - If chronic stool soilage, may benefit from ostomy training and colonic diversion

- Elimination of Pressure
    - Q2 hr turns, 10 second lifts every 10 minutes
    - Prevention of shear during repositioning: lift sheets, avoidance of dragging patient over linens/support surfaces
    - Low air loss mattress/overlay, appropriate cushions for wheelchairs
    - Seat mapping for identification of local pressure areas

4.6 Wound Care

- Control of bacterial burden: silver impregnated dressings, Medihoney, cadexomer iodine, sodium hypochlorite (Dakin's solution), acetic acid, gentian violet, methylene blue
- Control exudate: alginates, hydrofiber or foams
- NPWT

4.7 Surgical Management

- Stage 1 and 2 pressure injuries: Usually medical management including offloading, nutrition, and control of incontinence.
- Stage 3 and 4 pressure injuries: May require surgical intervention, ensure medical/nutritional optimization prior to intervention to prevent recurrence
    - Debridement of eschar and devitalized tissue