The Internist’s Approach to the Non Healing Wound

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Objectives

- Learn how to do a basic wound assessment
- Learn to recognize the presentations of the most common types of wounds
- Identify common barriers to wound healing
- Be aware of different types of therapies used in wound healing
  - Dressings, hyperbaric oxygen, debridement, negative pressure therapy, skin substitutes
Chronic Wounds

- Affect 5.7 million patients annually
- Account for $20-25 billion in healthcare costs annually
- Patients often have other significant comorbidities
- Quality of life issues
  - Appearance
  - Odor
  - Social stigma
  - Financial costs
  - Emotional and physical stress/pain
  - Disability for patient and lost productivity for patients and caregivers
The Wound Care/Hyperbaric Medicine subspecialty

- Wound Healing and Hyperbaric Medicine has evolved into a subspecialty
- “Wet to Dry” dressings are practically a thing of the past
- New modalities have transformed the care
  - Hyperbaric oxygen
  - Skin substitutes
  - Negative Pressure therapy
  - Enzymatic debridement
  - Effective dressings tailored to address needs of the wound
Wound Care/Hyperbaric Medicine

- 900 HBO facilities nationwide and ?? Wound clinics
- Multidisciplinary team of physicians and clinicians
- “CWS” certification
- Streamlined patient flow to promote
  - Evaluation
  - Treatment
  - “One stop shop”
Wound Healing
A Quick Review of Wound Healing

**Inflammatory Phase**
- 0 to 3 days
- Phagocystosis (WBC like macrophages, neutrophils ingest bacteria, dead tissue)
- Vasodilation (WBC release inflammatory markers to cause swelling, redness)

**Proliferative Phase**
- 3 to 21 days
- Fibroblasts produce collagen, new blood vessel growth, rich vascular network in collagen matrix
- Contraction and epithelialization, wound edges move toward the center to close the wound

**Maturation/Remodeling**
- Scar develops tensile strength
- 80% strong as original skin
Compromised Wound Healing

- Arterial Blood supply occlusion
- Tissue ischemia
- Hypoxia and CO2 retention
- Microcirculation changes
- Loss of vascular membrane integrity
- Leakage of intravascular fluids
- Edema
Assessment of Chronic Wounds
Types of Chronic Wounds

- Vascular ulcers
  - Arterial
  - Venous
  - Lymphedema
- Neuropathic/diabetic foot ulcers
- Pressure or decubitus
- Traumatic
- Surgical

- Atypical
  - Bullous pemphigoid
  - Vasculitides
  - Vasculopathy
  - Malignancy
- “Internal” wounds
  - Radiation injury
How do you Assess a Wound?

- **Appearance**
  - Irregular or uniform?
  - Draining heavily or dry?
  - Eschar
  - Granulation tissue
- **Size**
  - Area, Depth, Undermining
  - Sinus tracts, fistulae
- **Location**
  - Leg, ankle, toes, trunk
- **Drainage**
  - Degree, color, consistency
  - “Bioburden” and necrotic tissue
- **Pain**
- **Temperature, erythema**
- **Surrounding skin**
  - Maceration, eczematous, induration
Barriers to Wound Healing

- Vascular dysfunction (arterial or venous)
- Bioburden and infection
- Scarring and fibrosis
- Edema
- Pressure
- Necrotic tissue
- Host factors
  - Nutrition, comorbidities, social history
  - Medications: steroids, antirheumatic agents, chemotherapy
How are those barriers treated?

- Vascular correction – angioplasty or venous intervention
- Bioburden and infection – routine debridement, antibiotics, drainage control
- Scarring and fibrosis – medications, surgery, negative pressure therapy
- Edema – compression therapy
- Pressure - offloading
- Necrotic tissue – routine debridement, bedside or OR
- Host factors
  - Nutrition – dietary evaluation, lean body mass monitoring
  - Comorbidities – control
  - Social history – smoking cessation, assistance at home
  - Medications – drug holidays if possible
Vascular Ulcers

- Healthy Vein Valves & Correct Blood Flow
- Damaged Vein Valves & Incorrect Blood Flow

- Normal artery
- Artery narrowed by atherosclerosis
- Plaque

Blood flow
Venous Ulcers

**Etiology**
- Typically from long standing venous hypertension
- Distension damages vein walls leading to exudation of fluid
- Valvular incompetence
- Chronic inflammation and ischemia from high interstitial fluid pressure

**Location**
- “Gaiter” (medial aspect of leg from calf to below ankle) distribution of both legs

**Ulcer Appearance**
- Shallow with irregular borders
- Increased drainage
- Fibrotic scar tissue
- Bioburden
Venous Ulcers

- **Skin exam**
  - Edematous
  - Stasis changes, pitting, lipodermatosclerosis, atrophie blanche
  - Hemosiderin staining
  - Palpable pulses

- **Symptoms**
  - Painful, especially when debrided
  - Recurrent cellulitis

- **Medical History**
  - Family History of “vein problems”, personal history of DVTs/phlebitis
  - “Standing” Occupations
  - Multigravid females
Venous Ulcers

- **Diagnosis**
  - Presentation
  - Confirm by Venous Studies, test for insufficiency/reflux

- **Treatment**
  - Venous intervention (eg. closure)
  - Compression therapy
    - Multilayer bandaging systems
  - Local wound care: debridement, dressing selection
  - Pain control
  - Medications, eg. pentoxifylline
  - Negative pressure
  - Skin substitutes and/or grafting
Compression Therapy

- Application of “4 layer” compression wraps
Lymphedema Ulcers

- **Etiology**
  - Develop from lymphedema and “phlebolymphedema”
  - Excessive accumulation of interstitial fluid and poor lymph flow
  - Any disruption of lymph channels
    - Primary (congenital) or Secondary classification
    - Secondary lymphedema often from obstructive or inflammatory processes
      - Tumor, surgery, infectious, venous insufficiency

- **Location**
  - Usually in the extremities, can move into trunk

- **Ulcer Appearance**
  - Ulcers are irregular, heavy drainage
  - No specific predilection to location
Lymphedema Ulcers

- **Skin Exam**
  - Stasis changes
  - Edematous and fibrotic
  - Papillomatosis, dimpling causing smooth but bumpy skin
  - “Stemmer’s sign”

- **Symptoms**
  - May or may not be painful
  - Recurrent cellulitis

- **Medical History**
  - Multiple comorbidities
  - Morbid obesity is common
Lymphedema Ulcers

- **Diagnosis**
  - Clinical
  - Important to rule out other causes of edema, eg. CHF, RI

- **Treatment**
  - Compression therapy
    - Manual lymphatic decongestive therapy
    - Lymphedema pumps
  - Avoid diuretics!
Arterial Ulcers

- **Etiology**
  - Pressure – shoewear, braces
  - Trauma
  - Embolus – acute event
  - Ischemia – progressive PAD
  - Infection
  - Poor healing due to compromised arterial blood flow

- **Location**
  - Usually lower extremities, often shin, feet or toes

- **Ulcer Appearance**
  - Punched out, dry appearing
  - Little drainage unless infected
  - Yellow slough or exudate, maybe eschar
  - Wet or dry gangrene
Arterial Ulcers

- **Skin exam**
  - Nonpalpable or diminished DP and PT pulses
  - Cool skin, loss of hair on toes
  - Periwound skin is “blue”, taut, shiny or show reactive hyperemia
  - Legs may be skinny, nails dystrophic
  - “Monophasic” pulse sounds by hand held doppler

- **Symptoms**
  - Rest pain or night pain
  - Claudication, dependency improves

- **Medical History**
  - Multiple comorbidities: DM2, CAD, CVA
  - “ABCDEs”: A1C, BP, Cholesterol, Diet/Obesity, Exercise, Smoking
Arterial ulcers

- **Diagnosis**
  - Arterial evaluation
    - ABI studies
    - Arterial dopplers
    - MRA
    - Vascular referral

- **Treatment**
  - Vascular evaluation and intervention imperative
  - Conservative approach until intervention
  - After intervention: debridement, moisture balance, skin substitutes
Diabetic Foot Ulcers
Diabetic Foot Ulcers

- **Etiology**
  - Diabetic neuropathy and its sequelae
  - Arterial and “small vessel” disease
  - Pressure
  - Deformity/Charcot foot

- **Location**
  - By definition, anywhere on dorsal or plantar aspect of foot

- **Ulcer Appearance**
  - Often full thickness, may probe to bone or tendon
  - Sometimes communicate from surface to surface
  - Drainage can be heavy
  - Initial appearance is often necrotic
  - If on plantar aspect, callus is very common
Diabetic Foot Ulcers

- **Skin exam**
  - Neuropathy
  - Concurrent arterial disease common
    - Rubor
    - Eczematous

- **Symptoms**
  - Neuropathy makes wound insensate

- **Medical History**
  - DM2, PAD, HTN, hyperlipidemia are common
Diabetic Foot Ulcers

- **Diagnosis**
  - Clinical history

- **Treatment**
  - Arterial assessment (pulses, ABIs with TBIs)
  - Topical agents (eg. PDGF)
  - DM2 control
  - Offloading
  - Serial debridements
  - Appropriate dressings
  - Hyperbaric oxygen
Pressure Ulcers
Pressure Ulcers

- **Etiology**
  - Localized ischemia from prolonged pressure over a bony prominence
  - Damage at superficial and deeper layers

- **Location**
  - Mid body: Sacrum, buttocks, coccyx, ischium
  - Foot: Heel, lateral and medial ankles, toes
  - Upper body: Elbows, scapula, shoulder
  - Head: Occiput, ears

- **Ulcer Appearance**
  - Staged from I to IV according to NPUAP
  - I – Intact skin with non blanching redness
  - II – Partial thickness wound or blister, red wound bed
  - III – Full thickness with slough, no bone/muscle/tendon
  - IV – Full thickness with exposed bone, muscle or tendon
  - Unstageable – full thickness tissue loss with base covered by eschar/slough
Pressure Ulcers

- **Skin exam**
  - Poor turgor, inelastic
  - May be wet from incontinence
  - Localized skin dermatoses

- **Symptoms**
  - Painless to painful

- **Medical History**
  - Neurological injury, neuropathy
  - Cachexia, malnutrition
  - Immobility
Pressure Ulcers

- **Diagnosis**
  - Clinical

- **Treatment**
  - Offloading!
    - Severity of ulcer will dictate offloading surface
    - Offloading devices and practices need to be routinely evaluated
  - Minimize moisture, friction, shearing forces
  - Nutritional assessment and intervention
  - Local wound care including serial debridements
  - Reconstructive surgery in severe cases
  - Prevention and Braden Scale
    - Recommend familiarizing oneself with the Braden scale for predicting pressure ulcer risk
Hyperbaric Oxygen
Hyperbaric Oxygen

- Treatment where patient breathes 100% oxygen at pressure greater than atmospheric.
- Oxygen dissolves into plasma and generates very high oxygen partial pressure gradients:
  - Up to 2000 mm Hg, or 20 times PO2 from room air.
- Multiple different mechanisms through which hyperbaric oxygen effective:
  - Significantly increases activity of fibroblasts and neutrophils.
  - Drives angiogenesis.
  - Reduces local edema.
  - Reduces reperfusion injury.
- Wounds/injury must be ischemic for therapy to be effective:
  - Arterial disease, edematous conditions, infectious, radiation injury.
- Be aware of unusual symptoms with radiation history:
  - Pelvic pain, discharge, hematuria, hematochezia.
Indications for HBO

- Acutely compromised or failed skin flap or graft
- Preparation for skin graft
- Severe diabetic foot ulcers
- Osteo and soft tissue radionecrosis
- Chronic Refractory Osteomyelitis
- Clostridial Myonecrosis (Gas Gangrene)
- Crush injury, Compartment syndrome
- Acute arterial insufficiency
- Necrotizing Fasciitis
- Carbon monoxide poisoning
- Decompression illness
- Arterial Gas embolism
- Brain abscess
- Thermal Burns
- Exceptional Blood Loss Anemia
Atypical Ulcers
Atypical Ulcers

- Pyoderma Gangrenosum
- Livedoid Vasculopathy
- Bullous Pemphigoid
- Porphyria
- Necrobiosis Lipoidica Diabeticorum
- Leukocytoclastic Vasculitis
- Mycobacterium marinum ulcer
- Atrophie Blanche
- Malignant melanoma
- Basal cell carcinoma
- Squamous cell carcinoma
- Mycosis fungoides
- Scleroderma
- Antiphospholipid syndrome
- Factitious dermatitis

• Biopsies are often needed to make the diagnosis
Dressings and Wound Care
Advanced Wound Dressings

- 4 Basic Functions of Advanced Wound dressings
  - Protect the wound from contamination or infection
  - Promote wound cleansing and debridement of unhealthy tissues
  - Absorb excess tissue fluid and wound exudates/debris
  - Maintain a moist environment for healing
- Hydrating - hydrogels
- Absorptive – foams, alginates
- Collagen
- Antimicrobial
- Skin Substitutes
  - Extracellular matrix products
  - Living cell therapy products
    - Growth factors and living keratinocytes
How do I start to workup the chronic wound?

- Do a good wound assessment
  - Size, appearance, drainage, pain
  - Choose a dressing to address immediate needs of wound
- Assess the patient’s vascular status
  - Palpable pulses, Arterial studies
  - Venous studies (order “reflux” evaluation)
  - Edema control (be cautious if arterial compromise)
- Surgical (vascular or plastics) referral
- Based on history and appearance, classify the wound type
How do I start to workup the chronic wound?

- Evaluate for presence of infection
  - Avoid swab cultures or antibiotics if not needed
  - Imaging studies
- If needed, determine patient’s offloading requirements
  - Appliances and surfaces
  - Assess risk for ulcer development and healing
- Examine the patient’s host factors
  - Nutrition, comorbidities, social history, medications
  - Anemia, renal insufficiency, liver dysfunction
  - Discuss likelihood of wound healing with your patient
How do I start to workup the chronic wound?

- Wound Center referral
- When patient comes back to you
  - Explain conditions that may result in wounds or delay wound healing (e.g., edema)
  - Educate on therapy to reinforce compliance with recommendations
  - Routinely discuss skin care and ulcer prevention
Summary

- Wound Care and Hyperbaric Medicine has really become its own subspecialty
- Wounds can be a result of multifactorial etiologies
- Most chronic wounds fall into 6 categories but atypical wounds should always be in the differential
- Multidisciplinary approach is often needed to treat the chronic or complex wound/limb salvage
- Primary care providers can initiate a good workup
- Patient education can help tremendously to heal and keep healed