Wound Healing: From Scare To Scar

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80% of wounds present to primary care offices

Buckle up....
Approach

• Understand what is supposed to happen (med school 1st year)
• Understand what can happen (med school 2nd year)
  – Wound classification
  – Wound etiology
• Identify and address the most basic issues (edema, offloading, arterial evaluation, sugar control, patient environment, nutrition)
• If this doesn’t work, go back to basics, then consider “atypical” etiology

What is normal healing?
Healthy Healing

- Localize injury (HEMOSTASIS)
- Clear out the bad stuff (INFLAMMATION)
  - Pathogens
  - Dead tissue
  - “used inflammatory cells”
- Build up the good stuff (PROLIFERATION)
  - New Extracellular Matrix
  - New blood vessels
  - New epithelium
- Form Scar (EPITHELIALIZATION)
- Strengthen (REMODELING)

Hemostasis

- Immediately
- Clotting activated by Extracellular Matrix components (Type I Collagen)
- Capillary contraction
- Platelet Activation
  - Platelets are the main cells that regulate this phase
  - Direct both inflammation and coagulation
- Localize injury
Inflammatory Phase

- 0-2 weeks
- Neutrophils are predominant
- Macrophages direct both inflammation and coagulation in this phase
- Goals
  - Wall off injured area
  - Remove pathogens and devitalized tissue
  - Release cytokines, chemokines and MMPs
    - Lyse dead tissue
    - Attract more proinflammatory cells to fight infection and clear necrotic tissue
  - Development of a provisional matrix
    - Stimulate angiogenic factors
    - Promote apoptosis in the inflammatory cells noted above
Proliferative Phase - Granulation

- 2 d to 2 weeks
- *Resolution of Inflammatory phase*
- Monocyte/macrophages predominate
  - Direct inflammation and coagulation responses
  - Promote apoptosis in neutrophils
  - Release multiple growth factors and chemokines
- Fibroblast migration
  - PDGF, TGF beta and bFGF
  - Migrate along fibrils in ECM

Proliferation: Granulation

- Transitional replacement
- High density
  - Blood vessels
  - Fibroblasts and macrophages
- Random collagen fibers
- Elevated Metabolic Rate
Epithelialization

• Wound contracts
  – Move tissue to center of wound
• Marginal epithelium proliferate and migrate on to wound
  – Dissolution of desmosomes to release cells
  – MMP secretion to lyse scab or eschar
• Remodeling occurs when confluent sheet completed
• Remember, once you penetrate the dermis, you are forming scar, not new skin
Remodeling

• Up to 2 years
• Scar formation
  – Reduce capillary number
  – Reduce cell density and metabolic activity
• Increased tissue tensile strength
  – Collagen I becomes dominant
  – Collagen more organized
• Maximum strength about 80%
Abnormal healing

No need to be scared…

How to start: What is going on around, to and from the wound?

• History
  – Trauma
    • Acute trauma
    • Repetitive trauma
  – Decrease in activity
    • Where do they spend most of their time?
  – Decrease in nutritional intake
    • When they say they eat, drill down…
  – State of comorbidities
    • Immunosuppression
    • Fluid status (cardiopulmonary/renal)

• Exam
  – Edema
  – Vascular status
  – Contractures, bone and joint changes, foot deformity
  – Neuropathy

• Location of wounds
  – Distal extremities (vascular issues)
  – Bony prominences
How to start: what stage are we in

- **Inflammatory**
  - Diabetic wounds
  - Infected wounds and wounds from primary infection
  - Hematomas (infected or uninfected)
  - Many pressure ulcers
  - Ischemic ulcers
- **Proliferative**
  - Venous stasis ulcers
  - Some infected wounds
- **Mixed**
  - pyoderma
The Diabetic Foot Ulcer: what you need to know

- Mixture of many ulcer types
  - Pressure
  - Arterial (often small inoperable vascular disease)
  - Infection
- Late diagnosis is common, Early diagnosis is key
- Calluses are dangerous
- Not all ulcers in diabetics are diabetic foot ulcers
- DFU= NEUROPATHIC FOOT ULCER

Wagner Classification

- System set up to predict risk for amputation
- Wagner 0: preulcerative callus
- Wagner 1: Soft tissue ulceration (even the deep ones)
- Wagner 2: Ulceration down to bone, but not associated with abscess
- Wagner 3: Ulceration associated with osteomyelitis or abscess
- Wagner 4: Ulceration associated with local gangrene
- Wagner 5: Ulceration associated with foot threatening gangrene
What do you DO?  
Prioritize

- Central issue: Pressure
- Contributing issue: Arterial disease
- Secondary issues: infection, edema
- Underlying issue: Glucose control

First Priority  
PRESSURE- OFFLOAD

- Casting
  - PROS: Cannot remove, customized
  - CONS: Requires specialty clinic, Often heavy and disrupt balance, can result in ulcers on contralateral foot
- Offloading shoes (patients can remove these)
  - PROS: Do not need specialty techs, readily available through vendors
  - CONS: can remove, not customized
Secondary factors

• Arterial disease: revascularization
  – ABIs on every patient with a foot wound
  – Arterial duplex if <0.8 or >1.3

• Infection: tough diagnosis
  – Deep cultures helpful with sterile instruments
  – MRI to establish possible bone involvement
  – If wound heals with sinus tract
    • Inadequately treated infection
    • Dead bone despite adequately, but late, treated infection

Glucose control

• Neutrophil function is impaired with obesity and hyperglycemia
• Chemotaxis, oxidative burst
• Pdgf production impaired
• Keratinocyte signaling pathways (early research) are also impaired.
• Atherosclerotic plaque resolution impaired in diabetes

• Glucose control: several classes of diabetes medications have been associated with a reduction in systemic inflammation, particularly with sulfonylureas, thiazolidinediones, DPP-4i, and insulin

• Work is preliminary but not surprisingly, the gist is that good glucose control is, well, good.
Calluses

- Body’s protection of area of repeated trauma
- Result from a combination of arthropathy (foot deformity from neuropathy) and apocrine dysfunction (moisture imbalance)
- One way valve for moisture
  - Gets in, but can’t get out
  - Breeding ground for bacteria
  - Softens underlying tissue so “rock in shoe” phenomenon causes erosion of tissue to bone

Calluses must be pared

- Remove the “rock in shoe”
- Expose ulcers beneath
- Earlier identification of underlying problems
- Prevention of ulceration
Custom shoes

• Type needed based on types of deformity
• No deformity: comfortable shoes with gel inserts ok. Check for calluses
• Hammer toes: extra depth shoes with custom inserts
• Subluxation: casted shoes with custom inserts
• Good for prevention.
• Do not put patients back in shoes in which they got the callus or ulcer without troubleshooting

Venous insufficiency: The next big one

• Central issue: increased venous pressure
  – Primary- incompetent valves
  – Secondary- R sided heart failure
• Secondary issue: Edema
  – Not all patients with VSUs have edema
  – Edema may also be a marker of other underlying problems
• Complicating factors: arterial disease, infection
Venous insufficiency: management (the trees)

• Check for arterial disease:
  – ABI* (we’ll get back to this)
  – Arterial duplex ABI <0.8 >1.3 or monophasic waveforms at pedal pulses, warrant further work up

• Compression:
  – Wraps
    • PROS: Customized, Variety, effective
    • Cons: Expensive. Requires some specialty training
  – Stockings:
    • PROS: no need for trained personnel, more effective for prevention
    • CONS: not as effective for serious ulcers, lose compression, can be tough to get on
  – Specialty wraps

Venous insufficiency: management (the forest)

• Underlying issues
  – Cardiac: CHF?
  – Pulmonary: primary lung disease
  – Renal
  – Primary venous insufficiency

• You won’t “miss” these but:
  – Make sure you connect what is going on above the waist with what is going on below.
  – Compression may require diuretic to help mitigate increased preload, for example
    • Not an absolute contraindication.
Pressure ulcers (other than neuropathic pressure ulcers)

- Inpatient and outpatient
- Huge initiative to prevent hospital acquired
- Often reflective of social situation
- ASK about
  - Mattress
  - Sleeping habits (bed or chair?)
  - If has wheelchair, was it customized for patient?
  - How old is cushion?
Pressure ulcer staging

- Stage 1: injury to epidermis only. Presents as blanching red area over pressure point.
  - Turn patient
  - Predictive of deeper injury if not addressed
- Stage 2: injury to dermis
  - Differentiate from friction and shearing
- Stage 3: injury to subq tissue
  - Presence of exudate defines stage 3. Debridement.
  - Specialty bed- group 2
- Stage 4: injury to deep structures (muscle, fascia, bone)
  - Specialty bed- group 2 or 3
  - Debridement
  - Flap closure
Pressure Ulcers: Management

• Central issue: OFFLOADING
  – Mattress (wound center can help)
  – Wheelchair and cushion (wound center/seating clinic)

• Contributing Factors:
  – Arterial disease (especially lower ext)
  – Protein malnutrition (albumin, hemoglobin, prealbumin)
  – Obesity (limited mobility and hypovascular adipose)
  – Secondary issue: infection

Many other ulcer types

• Arterial ulcers
• Ulcers from primary infection
• Autoimmune
• Malignancy
• Drug related

• Would love to discuss this but NO TIME!
A few other quick points

Case Studies

Case study 1

• 54yo patient with myasthenia gravis. Draining wounds on his right leg for 3 years, managed at an ID and surgical clinic first here (not in wound center) and then in Atlanta.

• Samples taken only from tracks, growing trichophyton (skin flora).

• Presents to me (for the first time) on long term voriconazole, but with the following:
Presentation outpatient clinic

Deeper problem exposed
Healed and still healed today

Take home lesson

• Sinus tracts are not “wound” or “skin defect” issues
• Sinus tracts are reflective of a necrotizing process beneath.
  – Usually infection
  – Sometimes malignancy
• Usually require ID and surgery
Case Study 2

• 56yo diabetic
• Intermittently compliant with insulin, decided to “take a break” from her medications
• Presented with painful swelling to her RLE
• Admitted, started on “VANCNZOSYN”
• Got worse after 24h, wbc 20K, crp 32, tmax 101, bp stable
• We were called

First contact: 7/19/15 inpatient

FSBG with goal <200
Vanc/clindamycin
Cultures grew Group A strep from wound
Blood cultures negative
WBC improved to 15K, crp 26, afebrile, bp stable
7/13/18 Day 5 active antibiotics
Hemorrhage has extended
Necrosis full thickness in some areas
WBC now 12k, crp 18, afebrile, VSS
Platelets have remained stable 200s
Decision made to wrap 20-30mmhg compression
Continued on ancef

7/16/18 Day 8 antibiotics
Felt much better
Wbc, crp, temp normal
Promised to take insulin
Decision made to discharge home with
Close follow up
30-40mmhg wrap biw at wound center
Keflex po tid to complete 6 more days
First outpatient visit: off antibiotics, 2 weeks post d/c 7/29/15

5 weeks post d/c 8/20/15
Take home lesson

- Reduced length of stay
  - Resource utilization
- Reduced antibiotic duration:
  - Many times extended abx because of ongoing discoloration
  - Often not needed for more than 2 weeks at most in these cases
- Improved quality of life
- Reduced chance for hospital acquired complications
  - No PICC
  - No prolonged bedrest
  - Reduced exposure to MDR bacteria
  - Reduced risk for c difficile associated diarrhea (CDAD)

Final pearls: A few parting thoughts for the PCP office

- Scabs
  - The body’s dressing in normal healing
  - Shroud over dead tissue in abnormal healing
- Calluses
  - Normal response to repeated trauma
  - One way valve for moisture in diabetic
  - Rock in the shoe for a patient with neuropathy
- Sinus tracts
  - The body’s effort to get rid of something.
  - Need to expose infection/necrosis before any healing will take place
Conclusion

• Remember that the wound is part of a patient
• If a wound is not healing, think about the patient and the underlying process
• Moisture, edema, pressure, infection, blood flow
• Think logically and reverse what you can
  – Pressure….offloading
  – Edema….compression
  – Poor blood flow…….revascularization
  – Immunosuppression….stop it if you can do so safely
  – Not healing despite these? Rethink DX.

References

References