Clinical Care of Pressure Sores: Biofilm

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Pressure Ulcers

• Are defined by the NPUAP as “localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear.”

• Incidence rates vary significantly by clinical setting:
  – 0.4%-38% in acute care
  – 2.2% to 23.9% in long-term care
  – 0-17% in home care

• An estimated 2.5 million pressure ulcers are treated in the US each year

• A huge economic burden

• A medicolegal burden
  – As many as 87% of cases result in settlements favoring the plaintiffs
Risk factors for pressure ulcers

- Older age
- African-American race
- Lower body weight
- Physical or cognitive impairment
- Poor nutritional status
- Incontinence
- Medical comorbidities including diabetes and peripheral vascular disease

In addition, the presence of pressure ulcers themselves are associated with a poorer overall prognosis and may also contribute to mortality risk.
End-stage pressure ulcers
I usually am called to provide surgical reconstruction of stage III-IV pressure ulcers

- For chronic pressure ulcers that have progressed to an advanced stage and are unable to reliably heal with conservative measures, surgical debridement and vascularized soft tissue reconstruction may serve to provide definitive closure.
Graft vs. Flap

**Graft**
Does not maintain original blood supply.

**Flap**
Maintains original blood supply.
I close most pressure ulcers in suitable candidates with local tissue flaps
Surgical Reconstructions are fraught with frustratingly high early and late rates of failure.

- Failures include:
  - Wound dehiscence
  - Wound infection
  - Bleeding
  - Flap failure
- Recidivism rates due to pressure ulcer recurrence or flap failure is currently unacceptably high with overall rates as high as 25 percent.
- These rates are even higher in spinal cord injured patients with a recurrence rate of 70 percent in one study.
• Given these high rates of recurrence and the significant financial burden on the health care system associated with pressure ulcers, emphasis must be placed on the proper selection of patients for surgical reconstruction and the need to develop improved prevention strategies.

Guidelines for care are essential
Fundamentals: Focus on Biofilm
Timeline of Normal Wound Repair

**Phase**

**Vascular Response**
- Clot formation
- Hemostasis
- Growth factor elaboration
- Collagen deposition
- Collagen cross linking

**Cellular Response**
- Neutrophils
- Macrophages
- Lymphocytes
- Fibroblasts

**Vascular Response**
- Vasoconstriction
- Vasodilation

**Phase**
- Inflammatory
- Proliferation
- Remodeling

**Major Event**
- Injury
- 3 days
- 7 days
- 3 weeks
- 1 year

**Injury**
- 3 days
- 7 days
- 3 weeks
- 1 year

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Basics of Care for all Wounds

- Optimize systemic parameters
- Optimize blood flow/perfusion
- Reduce edema
- Utilize dressings appropriately; consider specific aims and cost effectiveness
- Use pharmacologic therapy when necessary
- Close wounds surgically with grafts or flaps as indicated.
- Debride nonviable tissue
- **Reduce the wound bioburden**
Bioburden

- Bioburden represents the metabolic demands placed on the healing wound by bacteria.
- It is the sum of the by-products produced by bacteria, the competitive exhaustion of nutrients and oxygen by bacteria, as well as the toxic proteases and reactive oxygen species generated by the inflammatory cells of the host.

\[
\text{INFECTION} = \frac{\text{Microbial Count} \times \text{Virulence}}{\text{Host Resistance}}
\]
What do these wounds have in common?

• Bacteria are ubiquitous in all wounds
• What is different in a chronic wound that allows biofilm to persist?
• Do all chronic wounds have biofilm?
• How do we address this as surgeons?
• What is the evidence that biofilm are truly important?
Almost any wound in the body will heal on its own in a healthy environment.

There are many reasons why a wound will not heal – as a matter of fact, hundreds...
Inflammatory Disorders
- Pyoderma gangrenosum
- Granuloma annulare
- Necrobiosis lipoidica
- Sweet's disease (acute neutrophilic dermatosis)

Malignancy
- Marjolin's ulcer
- Metastases
- Primary skin malignancies
- Ulcers associated with hematologic or internal malignancies

Pressure ulcers

Infectious disorders
- Bacterial (Pseudomonas, Staphylococcal scalded skin syndrome, streptococcal necrotizing fasciitis)
- Fungal (blastomycosis, chromomycosis, Madura foot)
- Parasitic (Chagas disease, leishmaniasis)
- Viral (herpes simplex, herpes zoster)

Trauma (including postsurgical trauma)

Burns

Radiation ulcers

Factitious ulcers

Animal bites (dogs, snake, spider)

Medication-related ulcers
- Drug reactions leading blistering and large-scale wounds (erythema multiforme minor, Stevens-Johnson syndrome (erythema multiforme major), toxic epidermal necrolysis)
- Ulcer-causing medications used to treat malignancies (doxorubicin, hydroxyurea)
- Extravasation injuries, particularly with cytotoxic and vasoconstrictive medications

Autoimmune disorders
- Blistering (epidermolysis bullosa, pemphigoid, pemphigus)
- Nonblistering (dermatomyositis, lupus, rheumatoid arthritis, scleroderma)

Atherosclerotic arterial ischemic ulcers

Nonatherosclerotic ischemic ulcers
- Hypertensive ulcers (arteriolar, small vessel ulcers)
  - Sickle cell disease
  - Thrombangiitis obliterans

Vasculitis
- Churg-Strauss syndrome
  - Henoch-Schönlein purpura
  - Leukocystoclastic vasculitis
  - Microscopic polyangiitis
  - Polyarteritis nodosa
  - Urticarial vasculitis
  - Wegener's granulomatosis

Vasculopathy
- Cryoagglutination (cryoglobulins, cryofibrinogens)
- Embolic (cholesterol emboli, hyperoxaluria, cardiac clots, subacute bacterial endocarditis)
- Thrombotic
  - Antiphospholipid antibody syndrome
    - Coumadin necrosis
    - Dego's disease
  - Disseminated intravascular coagulation, purpura fulminans
    - Factor V Leiden deficiency
    - Heparin necrosis
    - Homocysteinuria
    - Livedoid vasculitis
    - Polycythemia vera
    - Protein C/S deficiency
  - Thrombotic thrombocytopenic purpura
  - Calciophylaxis (uremia with hypercalcemia and hyperphosphatemia)
    - Vasospastic/Raynaud's disease
    - Venous stasis ulcers
    - Lymphedema
    - Congenital
    - Acquired

Neuropathic disorders
- Diabetes
- Leprosy (Hansen's disease)
More logical way to treat wounds is to be a lumper, not a splitter

Unifying hypothesis first proposed by Mustoe

Logical: If you address these factors, you can manage most wounds, regardless of etiology
Hypothesis

Bacteria have evolved mechanisms to co-exist with their host to create a specialized niche that protects them from the immune system and from human interventions, in a manner that will not actively kill the host.
Biofilm Impact All Cell Types Involved in Wound Healing

(Lavigne, et al., 2015)
“Take-Home” Messages That I have Learned about Biofilm

• Biofilm is most problematic in unhealthy patients.
• Biofilm is an evolved state that hijacks the body’s wound healing programs to enable persistence.
• Biofilms are multispecies communities that can often cooperate with each other.
• Biofilm infections behave very differently from an acute infection.
• Biofilms form quickly, and reconstitute effectively.
• Debridement is our single best tool to minimize biofilm and allow healing, but other adjuncts are needed.
• Biofilm functions to minimize antimicrobial efficacy.
• We are only beginning to understand how the innate immune system interacts with biofilm. They manipulate the body’s immune response to their advantage.
• Any setting with an altered immune response will enhance the potential for biofilm formation and persistence.
Biofilm is most problematic in unhealthy patients

- In order to heal effectively, cannot myopically focus on just the wound but need to optimize regional and systemic factors that will improve healing.
  - Glucose management
  - Nutrition
  - Inflammation
  - Ischemia
  - Necrotic materials
  - Wound bed preparation = patient preparation
My Protocol

• Identify appropriate candidate for surgical closure
• Have reasonable nutritional parameters
  • Pressure sore patients almost never have normal lab values
• First stage of surgery – excision of pressure sore with ostectomy
  • This is slightly different from debridement
  • Send bone for path and for culture at end of case
• Patient discharged
  • Treat wound with Dakin’s dressings
  • Wet to dry
  • Would like to utilize NPWT with installation
• One week later
  • Close wound with flap if bone biopsy is negative for osteomyelitis and/or wound cultures are negative for virulent bacteria
This is why good wound care is important after debridement.

- **6 h**: Bacteria sparsely covering wound surface.
- **12 h**: Bacteria spreading more evenly over wound surface.
- **18 h**: Bacteria covering wound, assuming biofilm production and architecture.
- **24 h**: Mature biofilm, bacteria contained within matrix.
Wound Bacteria are Different from Skin Microbiota

(Lavigne, et al., 2015)
The wound microbiome changes in response to interventions, such as antibiotics (Lavigne, et al., 2015)
Biofilm defines a chronic wound
The Fundamentals of Pressure Ulcer Reconstruction

- Offloading
- Infection Management
- Control of Bacteria
- Moisture Management
Clinical Examples
Ischial pressure ulcer, stage IV
Giant Stage IV pressure ulcer requiring mobilization of most of buttocks with bilateral V-Y flaps
Free tissue transfer for heel and Achilles tendon reconstruction
Postop-care of flaps

- Pressure relief and minimizing motion for 2 weeks.
- AFT for pressure sores and flaps over bony prominences
- Transition to low air-loss mattress at 2 weeks
- Transfer to rehabilitation center at 2 weeks – perform PT for 4 weeks
- Discharge to home with education and social work support 6 weeks after surgery
- Any evidence of suture line dehiscence treat aggressively
  - NPWT
  - Re-closure
  - Packing
  - Consider return to OR.
Thank you