Convatec Sponsored Learning:

Recognizing Granulitis And Addressing Biofilm In Hard-to-Heal Wounds.

Wound Hygiene: The Journey So Far

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Terminology Affects Possibility: Chronic Vs. Hard-To-Heal Wounds

Chronic wounds are common and costly to treat. The word "chronic" implies that the wound is long-standing. It also instills a sense of hopelessness. The term "hard-to-heal" is a more appropriate term to describe wounds that are slow to heal for various reasons, including complex medical history and biofilm. The wound is a battlefield. It is not a garden. Wounds that are hard-to-heal require proactive management. Such management includes wound hygiene (including biofilm management).

What Is Biofilm?

Biofilm is the principal cause of hard-to-heal wounds.[1] Biofilm is the natural and preferred mode of bacterial life. Aggregates of bacteria first attach to a living or non-living surface. Then they secrete a sugary, slimy matrix, known as the extracellular polymeric substance (EPS) around themselves. This matrix protects them from extreme conditions, including heat, dryness, host immune response and antimicrobial agents. Lastly, bacteria from the biofilm can disperse and become planktonic (free-floating) again, causing a secondary infection and further tissue damage.

Biofilm Is:

- The predominant and preferred form of bacterial life
- Stubborn (i.e., hard to remove physically)
- Persistent and promotes host inflammatory response
- Associated with chronic infections
- The principal cause of hard-to-heal wounds

Biofilm And Wound Healing

Biofilm acts like a parasite inducing prolonged tissue inflammation (Granulitis) and requires ongoing management to reduce its presence. Thus wound hygiene directly targets the root cause of the hostile environment which stalls healing in the majority of hard-to-heal wounds. Biofilm are difficult to remove physically and can impair epithelial cell migration and granulation tissue formation.[2] The EPS protects the biofilm from harsh external environments, including host immune response and antimicrobials. During the initial phase of normal wound healing, neutrophils, a type of

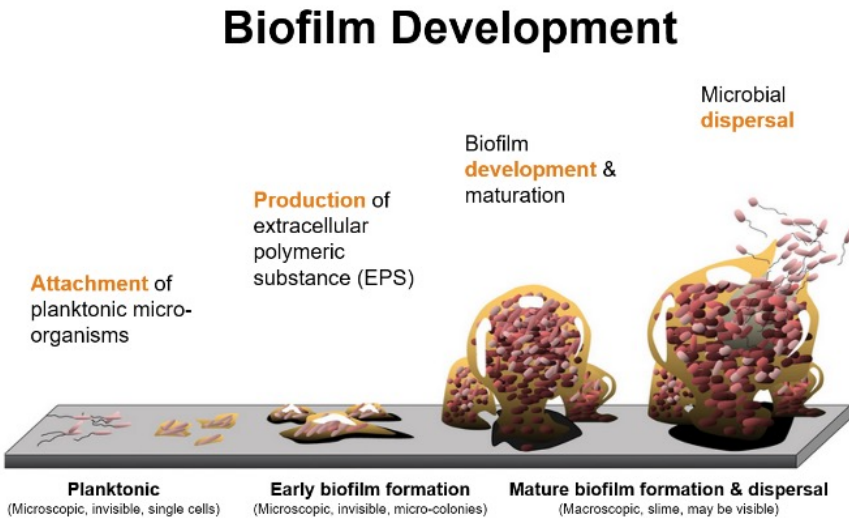


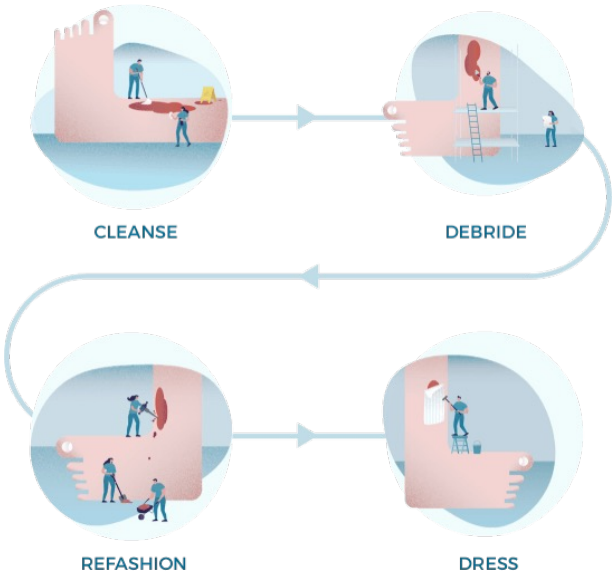
Table 1. Inflammation and infection in acute and hard-to-heal wounds

	Acute wound infection	Chronic wound infection
Causative agent	• Metabolically active planktonic microbial cells	• Metabolically passive and sessile biofilm microbial cells
Infectious process	• Invasion of host viable tissue via virulence expression (e.g., microbial enzymes, toxins)	• Biofilm (parasitic) persistence on host tissue[32] • Persistent inflammation, continuous oxidative damage, fibroblast senescence, degradation of growth factors, sustained NET release[28,33]
Inflammatory response	• Host-controlled response • Neutrophil recruitment to tissue site • Increase in intracellular oxidative burst and microbial killing • NET activation and release (NETosis)[28]	• Microbe-controlled response • Low-grade inflammatory response (IL-1ß and TNF-α expression) compared with acute wounds[24] • Neutrophil aggregation around biofilm, ineffective action leading to host cell senescence and oxidative damage[33] • Persistent NETosis[28]
Clinical manifestation	• Erythema • Heat • Pain/tenderness • Oedema	• Delayed wound healing[17, 33–36] • Wound breakdown[35] • Dull/dark red granular or discoloured tissue[34,35] • Increased exudate[32,34,35] • Friable, unhealthy granulation tissue/bleeding[35,36] • Increased exudate/purulence[32,34,35] • Increased pain[34–36] • Increased malodour[35,36] • Hypergranulation[36] • Epithelial bridging and pocketing in granulation tissue[36]

NET—neutrophil extracellular trap; IL-1ß—interleukin 1ß; TNF-α—tumour necrosis factor-α

white blood cell, are the first line of defense against bacteria. They induce host-controlled inflammation to eliminate the bacteria and initiate healing. In the presence of biofilm, the EPS prevents the neutrophils from accessing the bacteria.

This leads to neutrophils becoming 'frustrated', releasing enzymes and toxins that ultimately damage host tissue while the bacteria remain protected within the EPS. This process is known as microbe-controlled inflammation. It delays wound healing and causes chronic wound infections. The differences between host-controlled and microbe controlled inflammation and their ultimate clinical manifestations are shown in Table 1.



Recognizing Granulitis: Biofilm And Wound Hygiene

Granulitis is a biofilm-induced, prolonged inflammatory condition.[6] It is similar to gingivitis – gum inflammation caused by biofilm. Granulitis is, in essence, inflammation of the granulation tissue in the wound. It can lead to the formation of unhealthy granulation tissue (i.e., discoloured tissue, friable tissue, hypergranulation tissue, epithelial bridging/pocketing). Management of granulitis requires early and frequent local wound management and wound hygiene.

Wound hygiene involves four steps – cleanse, debride, refashion and dress.[7] Table 3 provides guidance for wound hygiene and the management of biofilm.

AQUACEL® Ag+ Extra™: Use As Part Of Your Anti-Biofilm Strategy

As previously mentioned, the EPS of the biofilm prevents antimicrobials and host defense mechanisms from accessing the bacteria. In order to address biofilm, the EPS needs to be degraded or dispersed. Surfactants (i.e., detergents) and metal chelators (e.g., EDTA) have been

Table 2. Guidance For Performing Wound Hygiene Tasks[8]

Tissue Type	Recommended cleansing intensity	Recommended debridement methods	Recommended refashioning intensity
Necrotic	Vigorous (using physical force)	Intensive: • Surigcal • Sharp selective (curette, scalpel, scissors, forceps) • Larval* • Mechanical debridement (including soft debridement pad, gauze or wipes)	Agitate the wound surface to pinpoint bleeding
Sloughy	Vigorous	Intensive: • Surigcal • Sharp selective (curette, scalpel, scissors, forceps) • Larval • Mechanical debridement (including soft debridement pad, gauze or wipes)	Agitate the wound surface to pinpoint bleeding
Unhealthy granulation	Vigorous	Intensive: • Surigcal • Sharp selective (curette, scalpel, scissors, forceps) • Larval • Ultrasonic debridement • Mechanical debridement (including soft debridement pad, gauze or wipes)	Agitate the wound surface to pinpoint bleeding
Healthy granulation	Moderate or gentle, depending on confidence and competence	Gentle: • Mechanical cleansing/debridement (including soft debridement pad, gauze or wipes)	Selectively rub in circular motion over wound bed and periwound skin, as needed
Epithelialisation	Gentle	Not required	Not required

found to be useful in dissolving the EPS of biofilm. The AQUACEL® Ag+ Extra™ dressing contains three components:

- Exudate management: Hydrofiber® technology
- Biofilm management: EDTA (metal chelator) and benzethonium chloride (surfactant)
- Infection management: Ionic silver

AQUACEL® Ag+ Extra™ can be used as step 4 of the Wound Hygiene protocol. It is able to manage biofilm, infection, and exudate, and has been shown scientifically and clinically to facilitate healing in hard-to-heal wounds.

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