The conundrum of wound infection

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It is broadly accepted that wound infection poses one of the greatest challenges in wound care. Apart from probably providing the most prevalent cause for delayed healing infection also presents a conundrum that will take some considerable time to unravel. In order to better understand this we need to identify some of the components of this conundrum.

A universally accepted definition of wound infection has yet to be agreed. The objectives of treating infection are to; halt progressive spread, improve quality of life through reduction in distressing symptoms, and to promote healing. Thus, a clear definition that supports these objectives is required (White et al 2013).

The diagnosis of wound infection provides us with an additional challenge. Although acute wound infection is easily recognisable through application of the Celcian signs (redness, swelling, heat and pain) formalized diagnostic criteria of chronic infection remains elusive and interpretation of the subtle signs of infection (Cutting & Harding 1994), are heavily reliant on personal skill. Diagnosis of chronic wound infection is confounded not only by the paucity of evidence but through poor interpretation of existing evidence.

Biofilm encased bacteria generally exist as polymicrobial communities and are often associated with chronic infections. The single celled, free-floating planktonic phenotype tends to be associated with acute infections.

Standard microbiology laboratory operating procedures focus on identifying bacteria through culture and then assessing anti-microbial sensitivity. This approach is linked to the concept that one microbe causes one disease - Koch's postulates (Percival & Dowd 2010). The polymicrobial nature of biofilm communities militates against this approach. However, clinical treatment of wound infection often echoes Koch's precept of 150 years ago that supports the 'one disease – one treatment' approach. This can result in a one-strategy approach to manage infection. When that policy fails, antibiotics are prescribed in sequence. This is, a deficient strategy for treatment of biofilm. Bacteria within a biofilm are essentially mutually dependent. The phenotypic

diversity within the community provides extensive adaptability to any stress (Percival & Dowd 2010). There seems to be little value in focusing on individual virulence genes but more appropriate to understand the biofilm strategies that are able to hijack the host immune response.

Regardless of aetiology all chronic wounds have similar biochemistries where elevated proinflammatory cytokines, matrix metallo proteases, and neutrophils may be found together with degraded protease inhibitors and diminished growth factors (Dielgeman & Evans 2004, Nwomeh et al 1998). This results in an underlying, persistent, yet low-grade inflammatory process that is thought to contribute to chronicity.

It would therefore appear that although inflammatory processes are present in both acute and chronic wounds the form of their manifestation varies by indication. The florid characteristics of acute wound infection together with their longevity of use (circa two millennia) are major contributors to their widespread application and perceived value in supporting accurate diagnoses. Although, wound infection criteria for use in the chronic wound situation have been developed (Cutting & Harding 1994) and validated (Cutting 1998, Gardner et al 2001) their application in clinical practice is inconsistent. It may be worth pondering on the fact that the Celcian signs of infection accepted by all have yet to be formally validated.

The quandary “is the wound infected” is often addressed by “lets take a swab”. Whether this is a qualitative or quantitative approach the result is at best likely to lead to inaccuracies in diagnosis or at worst treatment being applied or withheld in error. It is important to remember that it is the virulence factors expressed by bacteria that result in infection and not mere bacterial presence, which is what a surface swab will capture.

It is generally understood that slough is moist, devitalised fibrinous material that is comprised essentially of protein, and provides an ideal medium for proliferation of bacteria (Hurlow and Bowler 2009). Slough, although a nuisance, is not considered to be inherently pathological. Empirical observation informs us that slough is a frequent target for sharp debridement yet often it is found to promptly re-accumulate. We need to ask ourselves why this should happen. Could slough be more than an infection risk factor? Some suggest that slough is also a thriving microbial community Williams et al (2005), Hurlow and Bowler (2009). Debriding slough from a wound is an essential element of successful

wound care yet there are no published controlled trials that provide supportive evidence that debridement accelerates healing.

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