How might biofilms affect dressing choice for the diabetic foot?

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Chronic wounds, including diabetic foot ulcers, are colonised by micro-organisms. In the person with diabetes – where the immune response is impaired – this poses a major challenge, with control of infection being recognised as an important aspect of wound management.

Biofilms – aggregations of micro-organisms characterised by a protective extracellular matrix – have long represented a significant healthcare issue in areas such as orthopaedic prosthesis and dentistry. It has been estimated that 65% of human infections involve biofilms (Potera, 1999). In recent years, the paradigm of the place of biofilms in chronic wound care has begun to emerge.

Bjarnsholt et al (2008) stated that the reason many chronic ulcers do not heal is due to the presence of biofilms. Biofilms provide bacteria with a complex defence mechanism which allows them to resist the host's immune system, and antibiotic therapy. James et al (2008) demonstrated the presence of biofilm formation on chronic wounds. They also acknowledged that the role of polymicrobial biofilm communities in preventing wound healing remains unclear.

Challenge of early recognition of biofilms by clinicians

Clinicians may not always equate the presence of a biofilm in the wound bed as being a factor in chronic wound healing. Once a biofilm is established it is difficult to penetrate and disrupt. Therefore, in the authors' view, early identification is essential to achieve positive outcomes. It is difficult to distinguish a critically colonised wound from a biofilm using clinical indicators, but clues may include the appearance of a slimy film over the wound bed, presence of slough and lack of response to antibiotic therapy or antimicrobial dressings. Greater guidance on early identification of those individuals at risk would be of value.

The role of dressings and wound cleansing

Integral to the management of the diabetic foot is regular debridement of devitalised tissue. This helps reduce the bacterial load and remove senescent cells, thus returning the wound to an acute state. In their study on acute and chronic wound fluid, James et al (2008) did not detect biofilm formation in acute wounds. Could it be postulated that regular debridement may also help to reduce biofilm formation? Greater research is required in this area.

To remove biofilms on surfaces such as teeth, regular brushing with a toothbrush is recommended. While scrubbing a chronic wound is not advocated, other practical methods should be considered! Irrigation of the wound can remove loose debris and chronic wound fluid, but may be inadequate for removing micro-colonies of bacteria. The role of surfactant irrigation systems in wound cleansing may, therefore, be a route to explore, and worthy of further debate.

Antimicrobial dressings such as those containing silver are commonly used in wound care; however, not all antimicrobial dressings perform in the same way. Potemkina et al (2008) suggest that the action of silver may depend on the physiological state of biofilms and the longevity of treatment. The longer the biofilm exists, the greater the population density of bacteria; therefore, the need for early detection and prevention is an important factor in determining care pathways.

Antimicrobial dressings with a rapid speed-of-kill and sustained release to prevent reforming of the biofilm may provide a balance to achieve this.

Is the presence of biofilms a hidden epidemic in the diabetic foot? If so, how do we provide support to clinicians to identify clinical indicators and sound advice as to which dressing to use and why? ■

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