ISSN: 2684-4559 Open Access

Biofilm-Associated Infections: Implications for Chronic Wound Management

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Introduction

Chronic wounds, including diabetic foot ulcers, venous leg ulcers and pressure injuries, represent a major clinical challenge worldwide due to their prolonged healing times, high recurrence rates and substantial healthcare costs. One of the key factors contributing to delayed healing and treatment failure in chronic wounds is the presence of microbial biofilms. Biofilms are structured communities of microorganisms embedded in a self-produced extracellular matrix, which confers resistance to antimicrobial agents and host immune defenses. These biofilm-associated infections complicate wound management by promoting persistent inflammation, tissue damage and impaired repair mechanisms. Understanding the role of biofilms in chronic wounds is critical for developing effective diagnostic, preventive and therapeutic strategies. This article explores the formation and characteristics of biofilms, their impact on chronic wound pathophysiology and current approaches to managing biofilm-associated infections [1].

Description

Biofilms form when planktonic (free-floating) bacteria adhere to wound surfaces and produce Extracellular Polymeric Substances (EPS) composed of polysaccharides, proteins and nucleic acids. This matrix not only anchors the bacteria but also creates a protective environment that enhances survival. Within biofilms, microbial cells exhibit altered phenotypes, including reduced metabolic activity and increased expression of genes related to antibiotic resistance and virulence. These adaptations make biofilms notoriously difficult to eradicate with conventional antimicrobial therapies. Chronic wounds provide an ideal niche for biofilm development due to factors such as persistent tissue hypoxia, necrotic debris, moisture and impaired host immune responses. The polymicrobial nature of wound biofilms, often involving pathogens like Staphylococcus aureus, Pseudomonas aeruginosa, Enterococcus species and anaerobes, contributes to their resilience and pathogenicity. These biofilms induce a chronic inflammatory state characterized by the sustained presence of neutrophils and pro-inflammatory cytokines, which paradoxically delays healing and promotes tissue destruction [2].

Clinically, biofilm-associated infections manifest as wounds that fail to progress through normal healing stages despite standard care. Signs may include increased exudate, malodor, friable granulation tissue and persistent pain. However, biofilms are notoriously difficult to detect with routine microbiological cultures due to their sessile nature and the presence of viable but non-culturable bacteria. Advanced diagnostic techniques such as confocal laser scanning microscopy, fluorescence in situ hybridization and molecular methods offer improved detection but are not widely available in clinical

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Received: 28 January, 2025, Manuscript No. jid-25-168962; Editor Assigned: 31 January, 2025, PreQC No. P-168962; Reviewed: 11 February, 2025, QC No. Q-168962; Revised: 18 February, 2025, Manuscript No. R-168962; Published: 25 February, 2025, DOI:10.37421/2684-4281.2025.9.311

settings. Management of biofilm-associated infections requires a multifaceted approach. Mechanical debridement remains the cornerstone, physically disrupting the biofilm matrix and removing necrotic tissue to reduce microbial burden and promote healing. Adjunctive therapies include topical antimicrobials such as silver, iodine and honey, which possess anti-biofilm properties. Systemic antibiotics are generally less effective against biofilms due to limited penetration and altered bacterial susceptibility but may be indicated in cases of invasive infection or systemic involvement. Emerging strategies to combat biofilms focus on targeting biofilm-specific mechanisms. These include the use of quorum sensing inhibitors that disrupt bacterial communication essential for biofilm formation and maintenance, enzymatic degradation of the extracellular matrix and the application of antimicrobial peptides. Novel technologies such as photodynamic therapy and electrical stimulation have shown promise in enhancing biofilm disruption and wound healing. Additionally, the development of biomaterials resistant to biofilm formation is an active area of research. Preventive measures are equally important, particularly in high-risk populations such as diabetics and the elderly. Optimizing glycemic control, improving vascular perfusion, maintaining skin integrity and implementing strict wound care protocols can reduce biofilm establishment and chronic infection risk. Interdisciplinary care teams, including infectious disease specialists, wound care nurses and surgeons, play a vital role in managing these complex cases.

Conclusion

Biofilm-associated infections significantly impair chronic wound healing by creating a protective microbial environment resistant to conventional therapies and host defenses. Their presence leads to persistent inflammation, tissue damage and clinical treatment failures. Effective management requires early recognition, aggressive mechanical debridement and the use of anti-biofilm agents combined with systemic therapy when indicated. Advances in diagnostic modalities and anti-biofilm therapeutics hold promise for improving outcomes. Ultimately, a comprehensive, multidisciplinary approach that includes prevention, timely intervention and innovative treatment strategies is essential to address the challenges posed by biofilms in chronic wound management.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Palmer, Sterling. "Biofilm-Associated Infections: Implications for Chronic Wound Management." *Clin Infect Dis* 9 (2025): 311.