EFFECTIVENESS OF PAPAYA DRESSING IN DIABETIC FOOT ULCER

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Abstract:

The diabetic foot ulcer continues to be a significant healthcare problem and a major source of morbidity for the diabetic patient. Hyperglycaemia and insulin deficiency are likely to decrease the formation of granulation tissue and collagen and lead to poor deep-wound tensile strength, deficient capillary in-growth and defective immune function, among other deficiencies. Empirical evidences have shown that a systematic approach to the management of leg ulcers by using papaya preparations can reduce both healing time and costs.

Key Words: diabetes, papaya, foot ulcer, insulin,

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Introduction:

The diabetic foot ulcer continues to be a significant healthcare problem and a major source of morbidity for the diabetic patient. "The Diabetic Foot: From Art to Science", highlights that diabetic foot ulceration represents a major medical, social and economic problem all over the world. While more than 15% of diabetic patients have a history of non-healing foot ulceration, during their lifetime despite meticulous glycaemia and dietary control. The triad of neuropathy, ischemia and infection are culprits in the pathogenesis of the non-healing foot ulcer. Hyperglycaemia and insulin deficiency are likely to decrease the formation of granulation tissue and collagen and lead to poor deep-wound tensile strength, deficient capillary in-growth and defective immune function, among other deficiencies. Amelioration of underlying pathologies is also a prerequisite for good wound healing. The aim of effectively curing the diabetic foot ulcer is to mainly reduce the duration of hospital stay.

Wound healing process

In the normal course, the following stages occur in the process of wound healing. First there is the inflammatory phase characterized by cellular migration and phagocytosis, followed by the proliferative phase (fibroblast migration, collagen and extracellular matrix formation). Angiogenesis happens next, which promotes granulation tissue formation. Epithelialization and scar remodelling is the last phase. These various stages are controlled by a complex interaction of various chemical mediators (molecular environment of the wound) like interleukin, cytokines, growth factors, Matrix Metallo Proteinase (MMP) etc. In most cases, it is not possible to apply the principles of acute wound healing to chronic wounds without considering the

biochemical environment. Chronic wounds have a complex inflammatory nature and produce substantial amounts of exudates, which interfere with the healing process and the effectiveness of advanced therapeutic healing products. The normal pattern and time frame of the cellular and biochemical events is disrupted and the wound is prevented from entering the proliferative phase of healing.

There is often a pro-inflammatory stimulus due to necrotic tissue, a heavy bacterial burden and tissue breakdown that causes cellular and biochemical changes in the wound bed such as increased levels of MMPs, which degrade the Extra Cellular Matrix (ECM) and result in impaired cell migration and deposition of connective tissue. MMPs also degrade growth factors and their target cell receptors, preventing healing and perpetuating the chronic inflammatory phase.

In the diabetic foot ulcer, for the normal repair process to resume, the mechanical barrier to healing, identified as the debris in the wound bed, has to be removed by the application of correct wound care techniques. In order to select the most appropriate intervention, wound bed preparation is imperative to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures.

Concept of wound bed preparation

The concept of wound bed preparation involves a systematic approach to treat chronic wounds. Debridement is often the focus of wound bed preparation. The broad goals are to remove necrotic and / or fibrinous tissue from the wound bed, decrease the bacterial burden, and increase the amount of granulation tissue, decrease exudates and oedema and to reduce the number of abnormal or senescent cells within the wound or at the wound edge.

Wound bed preparation in diabetic foot ulcers

Wound bed preparation in a diabetic foot ulcer is achieved through debridement. It removes nonviable tissues from the wound bed which reduces the number of micro-organisms, toxins and other substances that may inhibit wound healing by stimulating inflammation and delaying granulation and epithelialization. Debridement removes senescent cells in the wound bed and non-migratory cells from the ulcer edge, allows for improved availability of growth factors and removes excessive or abnormal bacteria to facilitate the healing process. To achieve this, several types of debridement methods are available to health care providers viz. surgical / sharp, mechanical, autolytic, enzymatic and biologic. An ancient method of debridement, enzymatic, has had resurgence of late. The other four methods are more commonly used in clinical settings.

Diabetic ulcer typically has a thick rim of keratinized tissue surrounding it. Debridement must occur through the callus, so that the necrotic tissues are removed. Enzymatic debridement is an option if sharp debridement is not possible. Diabetic foot ulcers are likely to require serial debridements rather than a single intervention. The underlying pathogenic abnormalities in diabetic wounds cause a continual build-up of necrotic tissue and regular debridements are necessary to reduce the necrotic burden and achieve healthy granulation tissue. Debridement also reduces wound contamination and therefore, assists in reducing tissue destruction.

Enzymatic debridement

Enzymatic debridement removes necrotic tissue by digesting and dissolving the devitalized tissue in the wound bed, resulting in early initiation of anabolic process and overall enhanced wound healing. It can be used in both infected and non-infected wounds with any amount of necrotic tissue and with none to moderate amount of exudate. It is the preferred treatment when surgical debridement is inappropriate or unavailable. At the same time, clinical and laboratory studies have demonstrated that enzyme does not harm the viable tissue surrounding the wound. One of these enzymes is Papain, a plant derived enzyme that has a long history of successful clinical application. Papain is a proteolytic enzyme derived from the fruit of the papaya tree (*Carica papaya*). It digests necrotic tissue by liquefying fibrinous debris. Papain has cysteine residue at the active site that is capable of breaking down a wide variety of necrotic tissue substrates over a wide pH range (3.0 to 12.0). The rapid debridement is associated with a concomitant appearance of granulation tissue, as determined by clinical assessment. This is a safe and effective, selective and easy to use method and does not harm healthy tissues. It is preferred for wounds requiring continuous debridement to keep the wound free of necrotic debris.

Empirical evidences have shown that a systematic approach to the management of leg ulcers by using papaya preparations can reduce both healing time and costs. Treatment with this preparation resulted in the lesions becoming clean and beginning to granulate, generally within 5 to 12 days. The results from bacterial cultures taken prior

to debridement and again when the wound bed appeared to be clean and granulating, showed that papain debridement was not affected in the presence of various wound microbes. No adverse reactions to the medication were observed.

An application of unripe papaya pulp on diabetic foot ulcer under strict aseptic precautions appeared to be effective in desloughing necrotic tissues, preventing infection and promoting granulation tissues. Possible mechanisms of action may be the activity of proteolytic enzymes, as well as, antimicrobial and peripheral vasodilator activity.

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