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Editorial Article

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Living With Diabetes and Impaired Wound Healing

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Abstract

Diabetes is a systemic disorder that affects almost all body systems, either directly or indirectly through its complications. Among the acute complications, acute metabolic derangements, urinary tract infections, skin and other infections with side effects of drugs are important. The major chronic complications are retinopathy, nephropathy, neuropathy, ischemic heart disease, cerebro-vascular accidents (CVA), blood circulation disorders and dermal lesions. Between these, blood circulation disorders are the major morbidities. The effects of diabetes on healing are diverse, multi-factorial, complex and inter-related. Wound healing is a major concerned with diabetes, more with type II than compared with type I. In fact, diabetes affects almost all stages of wound healing to some extent. This editorial discusses the underlying mechanisms that causes delayed wound healing, as those have been extensively investigated in the past few decades.

Keywords: Diabetes; wound healing; complications

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The hyperglycaemia associated with diabetes can cause tissue damage in two ways. The first pathway is the intracellular hyperglycaemia caused by increased fluctuation through different metabolic pathways, which can adversely affect cellular functions. This is the underlying mechanism of early diabetic cataracts and peripheral neuropathy. Non-enzymatic glycation of proteins is the second most important pathway for long-term complications in diabetes. In this process, amino acid group of proteins without any enzymatic involvement gets attached with glucose molecule chemically forming a stable invention. These stable harvests then build up in excess and float upon the cell membrane exterior and bind with structural and circulating proteins. They are called 'Amadori products'.

Proteins with a longer half-life, such as collagen, fibrin, albumin and haemoglobin, build up complex glycation end products form slowly from Amadori products through series of further reactions. The extent of these reactions depends on the concentration of glucose, the duration of hyperglycaemia and the half-life of these proteins.

The microtubular protein tubulin forms non-reducible aggregates during non-enzymatic glycation and contributes to the defective axoplasmic transport seen in diabetic neuropathy. Non-enzymatic glycation is also responsible for the hardening of the glomerular

basement membrane seen in diabetic nephropathy (Makita et al, 1991). This results from accumulation of albumin and trapping of immunoglobulin G.

Non-enzymatically glycated collagen binds soluble proteins to form in situ immune complexes characteristic of diabetic nephropathy (Brownlee et al, 1984). Similarly, dense basement membrane with microcirculation leads to reduced blood flow and decreased oxygen perfusion to the tissues, resulting in delayed wound healing with secondary infections (McMurry JF Jr, 1984). The important proteins from a wound healing perspective that are affected by non-enzymatic glycation are collagen, fibrin and keratin.

Fibroblast initially secretes high molecular weight glycoprotein during synthesis of extracellular matrix proteins. This glycoprotein is known as fibronectin. The latter is associated with major functions with immune cells, fibroblasts and endothelial fibronectin acts like a transduction agent following an wound contraction and re- epithelialisation.

The robust capability of fibronectin with collagen, gelatin and heparin with be lowered by non- enzymatic glycation. Di Girolamo et al (1993) were unable to show significant differences in functional activities of fibronectin between people with diabetes and controls.

The employment of glucose to the human cells, tissues and initiation of protein synthesis is accompanied by an anabolic hormone known as insulin. Metabolism of carbohydrates, proteins and fats, are very much essential for cellular activities occurring as a sequence during the process of wound healing. In diabetes, all these three major metabolisms will be at risk affecting the normal activity of cells and tissues (Cooper, 2003).

Proteins are the structural units of healing wounds. Collagen and proteoglycans are the important proteins in the context of wound healing (Deodhar and Rana, 1997). A lack of insulin in diabetic wounds results in more protein degradation uncoupled from protein synthesis. Collagen formation is reduced and the existing collagen lacks tensile strength. This also adversely affects fibroblast and polymorphonuclear (PMN) cell functions.

Deficiency of insulin leads to more proteolysis, glycogenolysis and lipolysis. Fatty acids and triglycerides are mobilised to provide energy. Fatty acids are vital for the genesis of cell membranes and if the fat stores are depleted, this synthesis is slowed down. Ketone bodies are synthesized in the liver and converts free available fatty acids into ketone bodies along with the assistance of counter-regulatory hormones. In a healing wound, the presence of ketone bodies is a sign of inadequate nutrient supply or lack of insulin (Gavin, 1989).

- Fluid and electrolyte imbalances

Hyperglycaemia affects the kidneys by altering the renal threshold levels leading to osmotic diuresis and loss of water and electrolytes. The resulting decreased intravascular volume can lead to decreased perfusion pressure and decreased tissue oxygenation and hence delayed wound healing. Insulin is essential for the entry of potassium into the cell. Regulation of electrolyte balance can be impaired because of diabetes itself or because of an increase in counter-regulatory hormones such as glucagon and corticoids.

- Effects on immune system

Neutrophils and macrophages are the main competitors as they bring down the inflammatory and proliferative stage occurring during the process of wound healing. They perform important functions, including phagocytosis, migration to the wound bed, clearing the debris and producing a wide range of cytokines that orchestrate the healing cascade. Experimental studies have shown that impaired PMN cell function leads to delayed wound healing (McMurry, 1984).

Goodson and Hung (1977) postulated that defective wound healing in diabetes is related to the abnormal tissue responses during the inflammatory phase. The major functions of polymorphonuclear(PMN) cells will be held back with prolong hyperglycemia. The events under acute stages of inflammation like cell migration, chemotaxis, adherence and bactericidal activity of phagocytosis will get sluggish

(C Rask 2013). PMN cells also play a role in angiogenesis by releasing fibroblastic growth factors, vascular endothelial growth factor, tumor necrosis factor-alpha, transforming growth factor- beta.

Diabetes is associated with a substandard immune system. The severity of these defects is directly associated with the longevity of diabetes. In the proliferation and maturation phases of wound healing, the lymphocyte function and production of lymphokines are impaired. Thus, resulting in consequential increased risk of infections and delayed wound healing. In diabetic patients with bacterial infection, the bactericidal activity of PMN cells will be compromised and deteriorated as the disease progressed.

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