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Letter to the Editor

Intralesional administration of human recombinant epidermal growth factor improves healing and reduces amputations in patients with severe diabetic foot ulcers

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Sir,

Diabetic foot ulcer (DFU) is a major and devastating complication, affecting approximately 15% to 25% of all diabetic persons in their lifetime.[1] Of those patients who develop DFU, 45% are not cured by standard therapy.[2]

In DM, failure in the repair process of distal peripheral soft tissues leads to the characteristic appearance of chronic wounds. Hyperglycemia is the trigger. A chronic wound is defined as a wound that has intrinsic impairment in the normal healing process. A biological definition would be documented intrinsic impairment to normal healing that is characterized by a prolonged inflammatory phase, slow forming extracellular matrix and decrease in the rate of epithelialization.

A DFU may be considered a pro-inflammatory tissue that is sustained and/or exacerbated by the pro-inflammatory, pro-oxidant, and pro-degradative phenotypes of the metabolically deregulated host, causing failure in peripheral soft tissue repair. [3] Preclinical studies suggest that diabetic wounds may not only represent a biochemically hostile environment due to the reduced growth factor availability as well as due to changes in growth factor action.[4]

Evidence shows that diabetic patients have decreased concentrations of growth factors in their tissues, notably epidermal growth factor (EGF). The primary effects of growth factors activate and direct every stage of wound healing, and, specifically, epidermal growth factor (EGF) induces mitogenic, motogenic, and cyto-protective actions that are essentials for healing events and could be summarized as follows: (a) stimulation of productive cells to migrate toward the injured area, (b) stimulation of granulation tissue outgrowth – including extracellular matrix accumulation, maturation and de novo angiogenesis, (c) stimulation of wound contraction by myofibroblast activation and proliferation, and (d) stimulation of the damaged area resurfacing by epithelial cell migration and proliferation. EGF is also endowed with angiogenic activity, promoting the growth of a vascular mesh within the wound bed. The mechanisms behind this