Perspective on Kleidogenesis and Immune System

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Running Title
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BACKGROUND

One of the most disfiguring skin conditions, "Keloids," is easily diagnosed. From the time it was first described as a clinical entity in Egyptian Smith papyrus in 1700 BC and named by Jean-Louis-Marc Alibert, a French dermatologist, as a defined clinical condition in 1806, to medical science, the triggering factor/s and pathogenesis of Keloids so far have remained an enigma. Multiple triggering factors have been suggested, and Interventions have yet to demonstrate a satisfactory resolution. The deficit in our understanding of the etiological factor/s and pathogenesis is the main stumbling block to therapeutic success.

In recent years, the discovery of high levels of inflammatory cytokines in patients with keloids has led to the belief that Keloids are an "autoimmune" condition; hence, a systemic immune intervention needs to be explored.

METHODS

Reviewed available literature and personal discussion with the experts with understanding and opinion, analyzing current pathways of wound healing and keloid formation.

In humans: Wound healing in Fetuses, Keloids in Albino people, patients with Skid, Severe immunocompromised, Iatrogenic Immuno-deficiency, Trans-auto-implanted Keloids.

Patients with common autoimmune conditions, i.e., Rheumatoid arthritis, Crohn's disease, Lupus, Multiple Sclerosis, Type 1 diabetes, Psoriasis, Celiac disease, Vitiligo, and Graves' disease, were studied for the propensity of Keloids.

Experimentally: wound healing in Athymic Rats, Immunodeficient animals, Total-body-irradiation (TBI)-induced Marrow ablation have been investigated

The role of "Keratinocytes" in Skin as a Unique cell engaged in wound healing with its paracrine competence, independent of systemic immune function, and its role in Kleidogenesis has been explored.

The Psycho- neuro-hormonal- immune effects on Keratinocytes were intimately involved in the biology of keratinocytes and the Skin in general. The possible impact of these complex exogenous factors on Keratinocyte biology and wound healing has been scrutinized.

RESULTS

kleidogenesis is a highly complex biological, genetic, intrinsic molecular, and micro-environmental-interactivities. Micro-environmental factors exert a profound effect on Kleidogenesis. However, the genetically predisposed patients with a mutation of RSTS1, CREBBP genes in chromosome 16p13.3 are known to have a proven impact on affected Fibroblasts to initiate Kleidogenesis.

Without macro or micro-injury to the Skin, including the breach of the stratum corneum, the body has no reason to activate the healing process; hence there is no fibroblast recruitment, proliferation, and activities. Thus, there is no kleidogenesis. The natural home of the dermal fibroblast is the "Dermis "layer, whereas that of the Keratinocytes is in the "Epidermis" layer.

The process of "reconstitution" heals superficial wounds without the help of adaptive immunity, which is essential for deeper wound healing, involving regeneration, reconstruction, and remodeling with fibroblast-derived scar tissues, which may lead to the pathway to aberrant healing, including Keloid formation.

No document suggests that patients with compromised Immunity are "more" or "less" prone to developing keloids.

Evolutionarily Human innate immunity is the primary function of the immune system. Adaptive immunity is a consequential adoption to assist innate immunity to destroy invading pathogens and chemicals.

The role of Keratinocytes in "Kleido-genesis" or "Kleido-Lysis" is less trodden and merits concerted attention for identifying new therapeutic pathways.

CONCLUSION

No evidence exists that an auto-immune activity is at work for Kleidogenesis.However, further research is needed to understand higher inflammatory cytokines in keloid patients. Role of "Keratinocytes" in wound Healing; thus, by extension, pathobiology of Kleidogenesis merits attention for exploration of new therapeutic avenues.