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WOUND HEALING ACTIVITY

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ABSTRACT

Tissue regeneration and repair are comprised of a series of molecular and cellular activities that follow the commencement of a tissue lesion in order to restore the damaged tissue. The exsudative, proliferative, and extracellular matrix remodeling phases are sequential events that result from the integration of dynamic processes involving soluble mediators, blood cells, and parenchymal cells. Exsudative phenomena that arise following injury play a role in the development of tissue edema. The proliferative stage contracts myofibroblasts and causes fibroplasia in an effort to lessen the region of tissue injury. At this stage, angiogenesis and re-epithelialization processes can still be seen. Endothelial cells can differentiate into mesenchymal components, and this difference appears to be orchestrated by a set of signaling proteins that have been studied in the literature, known as the Hedgehog pathway. The purpose of this review is to describe the various cellular and molecular aspects involved in the skin healing process.

Keywords: Edema, Myofibroblasts, Proliferation, Angiogenesis, Hedgehog Pathway.

I. INTRODUCTION

In both humans and animals, the process of healing wounds is a significant yet intricate one that is controlled by a series of overlapping phases, such as the phases of hemostasis and inflammation, proliferation, and remodeling. Following a skin injury, the exposed sub-endothelium, collagen, and tissue factor will trigger platelet aggregation, which in turn will cause degranulation and release of growth factors (GFs) and chemotactic factors (chemokines) to form the clot (1). Successful hemostasis will be achieved through all of the aforementioned procedures. The first cells that arrive at the site of damage are neutrophils, which remove bacteria and debris to create an environment that is conducive to wound healing. Here, macrophages gather, aid in the phagocytosis of microorganisms, and cause tissue damage. The inflammatory and hemostasis phases typically take 72 hours to complete (2). The next proliferative phase is typified by a proliferative buildup of connective tissue and many cells. Fibroblasts, keratinocytes, and endothelial cells are all present in the wound. To replace the initial clot formation, extracellular matrix (ECM), which includes proteoglycans, hyaluronic acid, collagen, and elastin, forms a granulation tissue. Numerous cytokines and growth factors (GFs) are involved in this phase, including the interleukin (IL) family, angiogenesis factors (i.e., vascular epidermal growth factor), and the transforming growth factor-β family (TGF-β, including TGF-β1, TGF-β2, and TGF-β3). This stage lasts for several days or weeks (3).

II. TYPE OF WOUND HEALING

The body goes through a complicated process called wound healing to replace injured tissue. Wound healing can take many forms, however it generally falls into three stages:

1. Inflammatory Phase:

This is the body's first reaction to damage to tissue. In order to stop infection and get rid of waste, it involves blood coagulation and the activation of different immune cells.

Acute inflammation is a transient reaction that usually passes after a few days or up to a week.

2. Proliferative Phase:

The body attempts to replace and repair the damaged tissue during this stage.

In this stage, there are two primary forms of wound healing:

a. Primary Intention Healing: This kind of healing happens when the margins of the wound are near to one another and easily sewn or brought into close contact. Scar development is minimal as a result.

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b. Secondary Intention Healing: This happens when a wound is purposefully left open, or when there is a greater space between its edges. It frequently leads in more scar tissue and takes longer to heal.

3. Remodeling Phase:

This stage, which can extend from a few weeks to years, is when the body keeps strengthening and modifying the newly produced tissue. Over time, the scar tissue may progressively reorganize and take on a different appearance (4).

III. NORMAL PROCESS OF WOUND HEALING

The steps involved in a typical wound's healing process have been extensively researched and documented in writing .The biological process of wound healing is intricate and occurs in every tissue and organ in the body (5). This process involves a variety of cell types, such as endothelial cells, fibroblasts, neutrophils, macrophages, lymphocytes, and keratinocytes. Scavenger cells either eliminate the necrotic tissue or use the process of phagocytosis to separate it from the viable tissue (6).

The four stages of wound healing include remodeling, proliferation, inflammation, and hemostasis (7).

IV. CONCLUSION

The preservation of the human body's physiological balance is significantly dependent on the integrity of healthy skin. Our comprehension of wound pathophysiology will be greatly advanced by combining these strategies with cutting-edge tissue, cell, and molecular "omics" technology. In fact, there is a lot of promise for the creation of novel, cutting-edge therapeutic approaches for enhanced wound care in the future.

V. REFERENCES

[1] Landén NX, Li D, Ståhle M. Transition from inflammation to proliferation: a critical step during wound healing. Cell Mol Life Sci. 2016;73(20):3861-3885. doi:10.1007/s00018-016-2268-0

[2] Ellis S, Lin EJ, Tartar D. Immunology of Wound Healing. Curr Dermatol Rep. 2018;7(4):350-358. doi:10.1007/s13671-018-0234-9

[3] Potekaev NN, Borzykh OB, Medvedev GV, et al. The Role of Extracellular Matrix in Skin Wound Healing. J Clin Med. 2021;10(24):5947. Published 2021 Dec 18. doi:10.3390/jcm10245947

[4] Chhabra S, Chhabra N, Kaur A, Gupta N. Wound Healing Concepts in Clinical Practice of OMFS. J Maxillofac Oral Surg. 2017;16(4):403-423. doi:10.1007/s12663-016-0880-z

[5] Guo S, Dipietro LA. Factors affecting wound healing. J Dent Res. 2010;89(3):219-229. doi:10.1177/0022034509359125

[6] Raziyeva K, Kim Y, Zharkinbekov Z, Kassymbek K, Jimi S, Saparov A. Immunology of Acute and Chronic Wound Healing. Biomolecules. 2021;11(5):700. Published 2021 May 8. doi:10.3390/biom11050700

[7] Schultz GS, Chin GA, Moldawer L, et al. Principles of Wound Healing. In: Fitridge R, Thompson M, editors. Mechanisms of Vascular Disease: A Reference Book for Vascular Specialists [Internet]. Adelaide (AU): University of Adelaide Press; 2011. 23.