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PROLIFERATIVE STAGE OF INFLAMMATION CHARACTERISTICS

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Abstract
The proliferative phase of inflammation is the main stage of tissue repair and is characterized by the activation of cell proliferation, angiogenesis and the formation of extracellular matrix. The work discussed the mechanisms of the proliferative stage of inflammation, its connection with various diseases, and studied the development of new therapeutic approaches aimed at improving regenerative processes.

Keywords: Proliferation, inflammation, angiogenesis, fibroplasia, tissue regeneration, cytokines, growth factors, hypoxia, chronic inflammation, diabetes wounds, atherosclerosis.

Introduction

Inflammation of tissues injury eliminate reach and of tissues integrity to restore directed main physiological is a process [1]. He is one how many from stages consists of: exudation , proliferation and regeneration [2]. Proliferative phase damage deliverer factors eliminate from after tissues in recovery solution doer role plays [3-6]. This of stage main duties of tissues integrity recovery, angiogenesis and regeneration encouraging of cells increase own into takes [7-8].

Inflammation proliferative of the phase Mechanisms: Inflammation proliferative stage one how many main processes own into takes, their each one to himself special biological important have.

Angiogenesis - present from veins new blood veins harvest to be [8]. This is the process damaged to tissues blood supply recovery, oxygen and food substances delivered to give and metabolites release for is necessary [6]. Angiogenesis VEGF (blood vein endothelial growth factor) and Like FGF (fibroblast growth factor) growth factors with activates. This molecules endothelial cells moved pass and increase encourages this capillary of the network formation take will come

Cells proliferation. Regeneration in the process main role TGF - β and PDGF (platelet growth factor) under the influence active multiplying fibroblasts plays [7]. This cells damage to the place moved passes and there collagen and from the cell except of the matrix another structural parts synthesis do it starts Collagen restored to tissues mechanic strength gives [10].

From the cell except of the matrix formation ECM cells supports and of tissues structure defines [9]. Proliferative in phase fibronectin and hyaluronic acid from acid organize found temporary matrix is synthesized later he is mature collagen and with elastin replaced [8]. This is the process fibroblasts and of macrophages coordinated work demand does [7]. Epithelization . Keratinocytes wound surface across moved , epithelium defects fills Theirs activity cytokines and growth

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factors , that's it including EGF (epidermal growth factor) with in order is inserted [5]. Epithelization the wound closes and of the skin or mucus of the floor protection barrier restores [10].

Proliferative to the phase of factors effect Proliferative phase of signals complicated network , including cytokines , growth factors and from the cell except matrix components with in order is inserted [1].

1. Cytokines. Interleukins (IL -1, IL -6) and tumor necrosis factor (TNF - a) fibroblasts and of macrophages in activation main role plays [7]. This molecules not only cell proliferation strengthens, perhaps angiogenesis and tissues again to build regulate the processes as well puts [8].

2. Growth factors. VEGF and FGF is new of veins formation stimulates, and TGF -β two bilaterally role plays: ECM synthesis in order puts and excess inflammation suppresses [6,7]. PDGF of fibroblasts migration and to division help gives and EGF epithelization stimulates [5]

3. Oxygen and metabolism. Damaged of tissues hypoxia which enhances the production of VEGF hypoxia instigator factors (HIFs) activate [8] . Oxygen deficiency conditions cell metabolism anaerobic to glycolysis passes and cell division for necessary energy provides [6].

Proliferative phase and diseases between dependency. Inflammation proliferative stage violation chronic wounds, fibrosis or excess scars such as one row pathological to the circumstances take coming can

1. Sugary diabetes wounds Sugary diabetes with hurt in patients the wound treatment process significant level it breaks of inflammation proliferative stage pathological changes with depends, of angiogenesis decrease: Hyperglycemia new blood veins formation for necessary such as VEGF, which is main growth factors work release suppresses Enough angiogenesis if not, tissues hypoxic being it remains recovery process inhibition does [4].

Fibroblast dysfunction: Sugary in diabetes fibroblasts tissues in recovery important role who plays collagen increase and synthesis to do ability loses [6].

Chronic inflammation: Sugary diabetes in wounds constant activated macrophages excess inflammation produce cytokines (IL-1, TNF-α). will issue , this of inflammation recovery to the stage to enter hindrance does [1]. Epithelization enough that it is not and from the cell except of the matrix delay because of chronic become will come this while of the patient life quality significant level worsens [10].

2. Atherosclerosis. Inflammation proliferative stage main process blood vein of the wall smooth muscle of cells activation is considered This mechanism atherosclerotic of plaques in development important role plays [11]:

Smooth muscle hyperplasia: Blood vein of endothelium damage in response smooth muscle cells to intimacy passes and under the influence of PDGF and TGF-β active respectively a lot starts. This is blood veins of the walls to thicken and their of elasticity to decrease take comes [2].

From the cell except of the matrix excess synthesis : Fibroblasts and smooth muscle cells excess amount collagen and another matrix proteins work will issue , this while plaque fibrous layer formation help gives [7].

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Angiogenesis violation: abnormal capillaries atherosclerotic in plaques harvest will be and easy cracks, microhemorrhages cause emits and inflammation strengthens [8]. These processes to vasoconstriction, blood of flow to the violation and myocardium infarction or stroke risk increases.

3. Rheumatoid arthritis. Rheumatoid in arthritis of inflammation proliferative stage pathological to the feature have being , this binder of tissue no to be done take comes [7]:

Panus formation: Panus is it multiplying synovial cells , fibroblasts and inflammation from the cells organize found abnormal level enlarged synovium. It's aggressive layer uncle and the bone no does [8, 2].

Cytokine imbalance: IL-1, IL-6 and TNF-α osteoclast activity encourages and bone degradation increases. Same that's it cytokines chronic inflammation supports and fibroblasts activates [1].

Regeneration enough that it is not: Like VEGF growth factors in process participation but it is new of veins formation and tissues recovery joint of structures no to be done to cover for enough not [6, 5]. Rheumatoid in arthritis proliferative stage inflammation enhancer and in the joints non-refundable to changes take coming pathological of the process one to the part becomes [10].

4. Lungs fibrosis. Lungs tissues pathological recovery with from the cell except of the matrix excess synthesis appear to be it is possible while to fibrosis take comes [6]:

Fibroblast activation: TGF-β stimulates fibroblasts a lot amount collagen work emits to myofibroblasts rotation encourages . It is interalveolar of septa to thicken and lungs tissues elasticity to decrease take comes [7].

Chronic angiogenesis : blood vein formation violation hypoxia strengthen it is possible while of tissues next again structure encourages [5].

Permanent inflammation : Inflammation of cells constant activity alveolar to the normal regeneration of the epithelium hindrance makes , pathological circle concludes [1]. Lungs fibrosis breath get function significant level breaks and breath deficiency such as heavy clinical to appearances take comes [8].

5. Keloid scars . Keloids are proliferative in phase too much except increase as a result surface coming pathological expanded scars [6]:

of fibroblasts too much except activity : This is the normal regeneration of cells from the end and then collagen synthesis in doing continue , as a result a scar thickens [7].

Damaged ECM remodeling : Usually type III collagen type I collagen with replaced , but in keloids this process is broken [8].

Stable hypoxia : blood of supply enough not HIF activation support and of tissues to growth help to give possible [5].Keloid scars only cosmetic in terms of dysfunction without being , maybe binder changes in the tissue cause emits [10].

Summary:

Inflammation proliferative stage damaged of tissues recovery which provides complicated and a lot edged is a process . However , this stage disorders serious to pathologies take coming can of proliferation molecular mechanisms and cells and from the cell except matrix between mutually

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effects to understand new treatment methods work exit for very important [1]. This studies complications prevention to get and different diseases for forecast to improve help gives

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