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Opinion

Necrosis as a Step of Tissue Repair

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Introduction

Cell death is a crucial step of tissue regeneration, but massive cell death is a cornerstone pathophysiological mechanism of tissue destruction. Let's consider some specific aspects of tissue necrosis and its predecessors.

First of all, tissue destruction always occurs on the background of septic or aseptic inflammation. Any type of inflammation causes the increase of interstitial fluid at least due to a sodium flow from a cell. That is a crucial moment because at some point cells lose their intracellular fluid and this is the exact marker of cell destruction regardless of whether this is a consequence of cell compression or a breakdown of cell stromal components. We can state it because necrosis does not depend on the cell cycle that means that this process is not related to nuclear reactions. Based on this approach we can explain why some tissue can withstand the edema more effectively than others. For example: nerve tissue is extremely sensitive to edema, and we can assume that this is a consequence of a relatively small amount of plasmogel in a nerve cell. It exhausts its possibilities of intracellular fluid loss very quickly. A striking example of this is death of central and peripheral nerves due to edema of nerve fiber regardless of compression. Undeniably, the impact of perineurium and epineurium is incomparable with the capabilities of adipose tissue where the number of organelles is minimal and the cytoplasm is predominantly filled with liquid contents. This specificity is particularly significant for preservation of viability of parenchymal and alveolar organs with variety of pathologies and, of course, for the periphery. In my surgical practice I have often seen complete anatomical recovery of limbs after the treatment of gangrene. I mean bypass operations for people with atherosclerosis and diabetes, frostbite apical necrosis of extremities, who had indications for surgery but they refused amputations and I had to dress wounds for a long time. Wounds, even extensive, that are prone to chronic inflammatory process, are always accompanied by hyper granulation during the period of healing, while acute surgical wounds – almost never. A reasonable question "why" arises. I think that long-term leukocyte phase can form some type of memory that is why autoimmune needs for maintaining inflammation are not updated every moment but are supplied with the usual amount of cytokines protractedly. Thus, if initially cytokines "catalyzed" inflammation, then with the time immunize and, possibly, even form antibodies to TNF family, suppress the pro –inflammatory effects of a number of interleukins. Hence, necrosis clears the way for tissue repair.

Conclusion

a) Disabling surgery must be postponed as long as possible if the toxic complications allow it.

b) Glucocorticosterouds are not the drugs of choice at the stage of purulent inflammatory complications but possible they are preferred in the early septically uncomplicated stages

c) Toxic associated hypoproteinemia and hyperglycemia trigger RAAS and launch kinases cascades.

d) Long-term kinase release in the conditions of chronic inflammation can suppress pro-inflammatory effects of cytokines.

e) Cells with a large number of organelles have the least amount of plasmogel which determines their least ability to resist inflammation

f) Tissues of musculoskeletal function with a large amount of plasmogel have great repair capabilities. But bone tissue due to continuous mineralization process that is associated with activation of growth and mitotic factors in the conditions of excessive and prolonged cytokine activity often leads to the tumor formation.

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