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Plasma Growth Factors in Gastroenterology: The Utilty in Intestinal Regeneration and Repair in Necrotizing Enterocolitis

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Background

The use of plasma rich in platelet growth factors, has become a technique increasingly used in various fields of medicine. The power cell tropism for certain tissues, attributed to growth factors, has currently talk of a new medical discipline as regenerative medicine. Not only has experienced an ever increasing boom in various medical specialties, but simultaneously has increased exponentially types and methodology for obtaining application forms even for the same pathology. Although it is known of the existence of some types of platelet growth factors so as plasma, yet know the mechanisms by which produce the phenomena of regeneration, immunomodulation and cell differentiation. Perhaps the mechanisms that explain the cell biology of this type of treatment could be related to the paracrine effect these growth factors through various types of receptors in cell lines capable of modulating apoptosis or antiapoptotic signals to produce phenomena of proliferation, differentiation, maturation or tissue death.

Physiology of Autologous Plasma Growth Factors and Role in Pathogenesis of Neonatal Necrotizing Enterocolitis

They are proteins with great capacity of diffusion through the tissues in the organism that are found both in the platelet alpha granules and lysozyme of leukocytes, especially in mononuclear type and hematopoietic progenitors. But growth factors have also been identified in other organic fluids such as saliba, bile, gastric and intestinal juice, or cerebrospinal fluid.

The major plasma growth factors of the most known function are

PDGF (platelet-derived growth factor origin): Its main function is to indirectly promote angiogenesis via macrophages by a mechanism of chemotaxis. Activates macrophages, has a significant mitogenic activity on mesenchymal cells as well as neurons, microglia cells, promoting the proliferation of oligodendrocytes and remyelination and facilitates the formation of type 1 collagen.

TGF-Beta (transforming growth factor-Beta): Its main mission is to chemotaxis. Induces proliferation and differentiation of mesenchymal cells. It promotes collagen synthesis by osteoclasts. It is pro-angiogenic tissue, inhibits osteoclast formation and proliferation of epithelial cells in the presence of other factors. Induces differentiation of neuronal stem cells

FGF (Fibroblast growth factor): Enables the proliferation and differentiation of osteoclasts, fibroblasts and induction of fibronectin by these neural and epitelial stem cells. Inhibit osteoclast action. It is an important pro-angiogenic action chemotactic activity on epitelial and endothelial cells.

IGF-1 (Insulin-like growth factor 1): It induces the proliferation and differentiation of mesenchymal cells and like coating has a potent mitotic effect on neural progenitor stem cellularity. It facilitates the synthesis of osteocalcin, alkaline phosphatase and type 1 collagen by osteoblasts.

VEGF (Vasculo endotelial growth factor): Enables chemotaxis and differentiation of endothelial cells, it promotes blood vessel permeability.

EGF (Ectodermal growth factor): Great proapoptótotical capacity, chemotaxis and differentiation of epithelial cells, renal, neural, glial and fibroblasts.

HGF (Hepatocyte growth factor): Its main function of cell proliferation and differentiation, chemotaxis, angiogenesis and extracellular matrix synthesis

Necrotizing enterocolitis (NEC) is the most common gastrointestinal emergency encountered in the neonatal

period. The exact pathogenesis of NEC is unknown and likely multifactorial. Prematurity, aberrant bacterial colonization, hypoxia, and intestinal ischemia have all been implicated. Localized intestinal mucosal injury is thought to result in an amplified cycle of bacterial invasion, immune activation, uncontrolled inflammation, and gut barrier failure, leading to necrosis, perforation, sepsis, and shock.

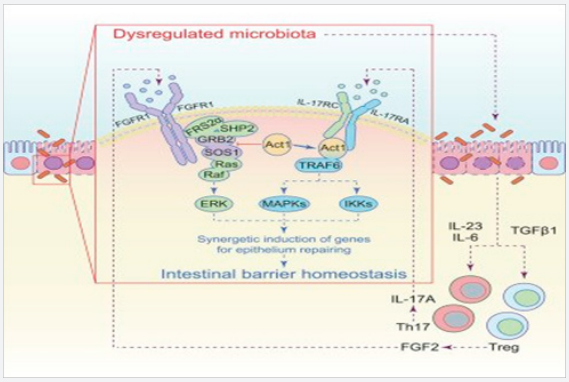

Figure 1: Activation of growth factors to repair lesion of intestinal epithelium

In the dynamic milieu of the developing gut, growth factors play a critical role in intestinal development. Growth factors like Epidermal growth factor (EGF, insulin-like growth factor (IGF), hepatocyte growth factor (HGF), have also been established to be important mediators of gastrointestinal repair, with roles in cellular proliferation, differentiation, migration, and survival (Figure 1). It is logical to consider that absent or reduced levels of these specific factors that are normally expressed during later periods of gestation may contribute to the development of NEC. As such, exogenous replacement of these key factor(s) may be of clinical value in the prevention and treatment of NEC have all been implicated in the pathogenesis and prevention of NEC. Recent evidence suggests that such factors may act synergistically to prevent intestinal injury. After intestinal resection has been shown to increase epithelial proliferation as well as increase protein concentration and mucosal DNA content.

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