Review Article

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Stem cell therapy in avoiding pathogenesis of Rheumatoid Arthritis

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Introduction

Rheumatoid Arthritis is an inflammatory disease of joints. It is a chronic autoimmune disease I.e.; it's means when a person's own immune system mistakenly attacks own body's tissue. Rheumatoid Arthritis can not only damage joints of a person's body but also can damage a wide range of body's system like eyes, skin, heart, lungs blood vessels. Rheumatoid Arthritis serves inflammation in joints (generally small joints like the one joining figures to hands) which can ultimately lead to bone erosion or physical disabilities. To treat this autoimmune inflammation disease – modifying antirheumatic drugs (DMARDS) are used. However only 70% of the patients dealing with RA get satisfactory results. Mesenchymal stomal/ stem cells (MScs) are cells which have tissue repairing and immuno-modulatory properties. They are being used for managing RA. MScs are adult stem cells and multipotent i.e., present in many tissues. These cells can renew on their own and can also differentiate into numerous tissues like fat cells, muscles.

The Human MSCs has capability to differentiate into mesodermal linages such as adipocytes, chondrocytes, ectoderm(neurocytes)and endoderm linages (hepatocytes). It expresses cells surface markers like clusters as CD29, CD44, CD73, CD90, CD105 and lacks CD14, CD34, CD45 expression and HLA (Human Leukocyte Antigen) - DR. Human MSCs were first reported in bone marrow and till now have been isolated from variety of other tissue like adipose tissue amniotic fluid, dental tissue, umbilical cord. MSCs uses both innate & adaptive immunity for regulation of inflammation.

Pathogenesis

The main tissue in RA is synovium. It is a membrane that lines the cavity of synovial joints. This membrane lubricates the joint surfaces and provides nutrients to cartilage synovium consists of a lining of macrophage like synoviocytes [1] , which show significant differences in their respective genomes and have completely different pa thoph y siology [2]. The primar y genetic risk factors of RA include alleles encoding the HLA-DR region [1, 3-10]. Other critical its specific targeting of the joints remain unclear, further research is required to fully understand this process [1]. The fulminant stage of RA involves hyperplastic synovium, cartilage damage, and bone erosion [1].

Along with bone loss, both inflammation and autoimmune responses are potential causes of RA progression [1]. This cascade of reactions is activated when fibroblast-like synoviocytes (FLSs) interact with immune cells of the innate and adaptive immune systems [10]. Some of the immune cells responsible for inflammation are monocytes, macrophages, T lymphocytes, and B cells [1,5,6]. The synovial membrane and cartilage undergo significant inflammation, causing hyperplastic synovium and cartilage destruction that eventually led to bone erosion [11]. Hyperplastic synovium is a critical characteristic of RA, and there are two hypotheses regarding its cause. The first is that the abnormal proliferation of FLSs ultimately leads to the production of inflammatory cytokines and mediators that continue joint destruction [1,7]. The second is that the resistance to apoptosis due to defects in tumor protein p53 triggers the hyperplastic synovium [1,8]. Here, the shortage of chondrocytes caused by apoptosis would result in cartilage degeneration and joint-space narrowing via directed and invasion [1,9,4].

How S-MSC can be an effective tool for therapy of RA; Conclusions and future aspects

Within the normal synovial tissue, two types of non-hematopoietic stromal cells: FLS and S-MSC appear to play an important role in controlling the inflammation and immune hemostasis. In normal conditions, closely related FLS and S-MSC can act as immunomodulatory cells controlling the magnitude of immune responses. Both stromal lineage cells retain some level of immunosuppressive capability during pathological conditions such as RA, which can be detected in vitro. However, due to various factors within RA milieu and as a result of a direct contact with inflammatory cells and cytokines, the immunomodulatory function of S-MSC and FLS seem to be disturbed. The proliferation of RA FLS which acquire aggressive pro –inflammatory phenotype within the synovium takes the upper hand.

Moreover, the function of S-MSC seems to be shifted towards immune stimulation. Based on these considerations, the use of S-MSC as cell therapy for autoimmune disorder such as RA needs to be complemented with targeting the inflammatory factors within the synovium. Essentially, further investigation into the interaction between S-MSC and RA FLS and with immune cells is still required to improve the therapy of RA. Some pre-clinical studies have been shown that targeting of signaling molecules in active pro-inflammatory cells including-RA FLS could be of value in treatment of RA. Therefore, combination of MSC therapy together with targeting RA FLS

must be considered. Furthermore, joint MSC harvesting sites, doses as well as routes and schedules of delivery remain underexplored and merit further investigation before this type of therapy could become a clinical reality.

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