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Stem Cell Therapy for Blood Cancer

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Introduction

In the last decade, many reports have emphasized the potential of stem/precursor cells as intervention strategy to repair damaged tissue, providing new hope in the scientific and medical community for the treatment of various diseases and conditions considered as incurable before, such as neurodegenerative disorders [1,2], celiac disease [3,4], type 1 diabetes mellitus complications [5,6], muscle damage [7,8] and various blood cancers. Overall, experimental and clinical evidence shows that embryonic stem cells and, in a lesser extent, adult stem cells can generate various tissue types with the potential to replace damaged areas in the body, with a reduced risk of rejection and manageable side effects. These findings have opened new avenues for cell-based cancer therapies, whose majority are at experimental stage. These strategies are providing very encouraging results, particularly in clonal hematopoietic disorders like Myelodysplastic Syndromes (MDS) where the blood-forming cells damaged in the bone marrow are successfully repaired by Hematopoietic Stem Cell (HSC) transplantation with minimal toxicity and improved quality of life [9]. However, due to the complexity of blood cancer pathogenesis and clinical features also shared by MDS, many patients remain non-eligible for HSC transplantation or do not show any improvement following such treatment [10,11], pointing out the need for more research to understand stem cell behavior upon transplantation, particularly the interactions with the diseased hematopoietic niche.

Many laboratories are addressing this issue and are currently studying how stem cell can be used for blood cancer treatment. Interestingly, Song et al. [12] have reported that in the bone marrow of MDS patients, although HSCs and Mesenchymal Stem/Stromal Cells (MSCs) display cytogenetic aberrations, MSCs are particularly affected by chromosomal aberrations with a random loss of chromosomal material. Bone marrow MSCs (BM-MSCs) are multilineage non hematopoietic progenitor cells derived from mesodermal precursors, which play a key role in supporting hematopoiesis and that give rise to different stromal cell lineages [13,14]. The findings of Song et al. [12] suggesting an enhancement of genetic susceptibility of BM-MSCs in MDS patients indicate that genetic alterations in these cells may constitute a key mechanism in the pathogenesis of MDS and of subsequent more aggressive cancers like acute myeloid leukemia. BM-MSCs express various proto-oncogenes which have been reported as crucial for HSC maintenance like Notch ligands and Wnt [15,16]; MSC-derived osteoblasts regulate the HSC niche through Jagged-1/Notch-1 signaling [17]; and BM-MSCs have been reported to play a pivotal role in the survival of Acute Lymphoblastic Leukemia (ALL) and chronic lymphoblastic leukemia B-cells [18,19]. In addition to the genetic aberrations affecting BM-MSCs and to the release or expression of oncogenes, the latter cells would determine the onset of high-risk MDS by displaying poor ability of hematopoietic support and immunosuppressive abilities [20]. These observations may explain at least in part the improvement of the condition of some MDS patients following bone marrow transplantation, i.e. an adjunction of healthy BM-MSCs that probably rescues BM-MSC cell populations and restores hematopoietic support. Beside bone marrow or HSC transplantation, other approaches aiming at restoring the HSC niche are being tested, including the promising peripheral blood stem cell transplantation.

The purpose of this special issue is to communicate and present some of the latest research carried out in this area while reviewing other important recent developments in the field.

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