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Abstract

Cytogenetic characteristics of mesenchymal stromal cells in Myelodysplastic Syndromes

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Introduction: Bone marrow (BM) resident mesenchymal stromal cells (MSCs) play a vital role in regulation of hematopoiesis, homeostasis and maintenance of hematopoietic stem cells. It is controversial whether the characteristics of MSCs are altered in Myelodysplastic Syndromes (MDS). In this study we aimed to describe the cytogenetic profiles of culture expanded MSCs isolated from patients with MDS.

Methods: MSCs obtained from the BM of 18 primary MDS patients were culture-expanded by seeding BM mono nuclear cells. Immunophenotype of MSCs was analyzed by flowcytometry. Differentiation studies of MSCs were performed by inducing MSCs towards osteogenic and adipogenic cell lineages. MSCs were karyotyped using G-banding technique. Presence of more than two spreads with same abnormality was considered as a clone. FISH was done on BM cells and MSCs of del(5q) patients.

Results: Culture-expanded MDS-MSCs showed similar morphology to that of control MSCs. Both control and MDS derived MSCs were able to differentiate towards adipogenic and osteogenic tissues. All MSCs were positive for CD73, CD90 and CD105 and were negative for CD34 and CD45. Majority of MDS-MSCs had normal karyotypes (69%). Abnormal karyotypes were found in 31% of MDS-MSCs. Aberrations were present in chromosomes 3, 6 and 7. Their BM karyotypes were normal. Two clones (normal and aberrant) of MSCs were identified in all the patients with abnormal MSC karyotypes. None of the patients with abnormal BM karyotypes had aberrations in their MSC karyotypes.

Discussion: MDS-MSCs showed similar characteristics with control MSCs with respect to cell morphology, presence of MSC cell surface markers and differentiation ability towards adipogenic and osteogenic tissues. Cytogenetic profiles of MDS-MSCs are distinct from that of their hematopoietic counterparts thus the occurrence of genetic abnormality in BM-MSCs in MDS could be an independent event. The exact role of these abnormalities in disease pathogenesis needs to be studied further.