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Stem Cell Augmentation Therapy for Cardiovascular Risk in Ankylosing Spondylitis: STATIN-as Study

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Abstract : Objective: Bone marrow derived stem cells, endothelial progenitor cells (EPCs), protect against atherosclerotic vascular damage. However, EPCs are depleted in AS and contribute to the enhanced cardiovascular risk. Statins have a protective effect in CAD and diabetes by enhancing the proliferation, migration and survival of EPCs. Therapeutic potential of augmenting EPCs to treat the heightened cardiovascular risk of AS has not yet been exploited. We aimed to investigate the effect of rosuvastatin on EPCs population and inflammation in AS. Methods: 30 AS patients were randomized to receive 6 months of treatment with rosuvastatin (10 mg/day, n=15) and placebo (n=15) as an adjunct to existing stable anti-rheumatic drugs. EPCs (CD34+/CD133+) were quantified by Flow Cytometry. Inflammatory measures (BASDAI, BASFI, CRP and ESR), pro-inflammatory cytokines (TNF-α, IL-6 and IL-1) and lipids were measured at baseline and after treatment. Results: At baseline, inflammatory measures and pro-inflammatory cytokines were elevated and EPCs depleted among both groups. EPCs increased significantly ($p < 0.01$) after treatment with rosuvastatin. At 6 months, BASDAI, BASFI, ESR, CRP, TNF-α, and IL-6 improved significantly in rosuvastatin group. Significant negative correlation was observed between EPCs and BASDAI, CRP and IL-6 after rosuvastatin treatment. Conclusion: First study to show that rosuvastatin augments EPCs population in AS. This defines a novel mechanism of rosuvastatin treatment in AS: the augmentation of EPCs with improvement in proinflammatory cytokines and inflammatory disease activity. The augmentation of EPCs by rosuvastatin may provide a novel strategy to prevent cardiovascular events in AS.

Keywords : ankylosing spondylitis, Endothelial Progenitor Cells, inflammation, pro-inflammatory cytokines, rosuvastatin
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