

Endothelial Progenitor Cells Is a Determinant of Vascular Function and Atherosclerosis in Ankylosing Spondylitis

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Abstract : Objective: Endothelial progenitor cells (EPCs) have reparative potential in overcoming the endothelial dysfunction and reducing cardiovascular risk. EPC depletion has been demonstrated in the setting of established atherosclerotic diseases. With this background, we evaluated whether reduced EPCs population are associated with endothelial dysfunction, subclinical atherosclerosis and inflammatory markers in ankylosing spondylitis (AS) patients without any known traditional cardiovascular risk factor in AS patients. Methods: Levels of circulating EPCs (CD34+/CD133+), brachial artery flow-mediated dilatation, carotid intima-media thickness (CIMT) and inflammatory markers i.e erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), tissue necrosis factor (TNF)- α , interleukin (IL)-6, IL-1 were assessed in 30 AS patients (mean age 33.41 ± 10.25 ; 11 female and 19 male) who fulfilled the modified New York diagnostic criteria with 25 healthy volunteers (mean age 29.36 ± 8.64 ; 9 female and 16 male) matched for age and sex. Results: EPCs (CD34+/CD133+) cells were significantly ($0.020 \pm 0.001\%$ versus $0.040 \pm 0.010\%$, $p < 0.001$) reduced in patients with AS compared to healthy controls. Endothelial function (7.35 ± 2.54 versus 10.27 ± 1.73 , $p = 0.002$), CIMT (0.63 ± 0.01 versus 0.35 ± 0.02 , $p < 0.001$) and inflammatory markers were also significantly ($p < 0.01$) altered as compared to healthy controls. Specifically, CD34+CD133+ cells were inversely multivariate correlated with CRP and TNF- α and endothelial dysfunction was positively correlated with reduced number of EPC. Conclusion: Depletion of EPCs population is an independent predictor of endothelial dysfunction and early atherosclerosis in AS patients and may provide additional information beyond conventional risk factors and inflammatory markers.

Keywords : endothelial progenitor cells, atherosclerosis, ankylosing spondylitis, cardiovascular

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