

The Study of Relationship between the circulatory numbers of cells expressing VEGFR1 with Multiple Sclerosis severity

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Background: Previous studies have shown that angiogenesis is involved in progression of chronic inflammatory diseases such as multiple sclerosis (MS). Also, it has demonstrated that vascular endothelial growth factor (VEGF) can trigger angiogenesis as an angiogenic factor. It acts through binding to its membranous receptor VEGFR2 expressed by endothelial cells. Recently, studies have demonstrated that there is a soluble form of VEGFR1 (sVEGFR1) in plasma that can prevent the binding of VEGF to its functional receptor on endothelial cells as a decoy and prevents the continuation of angiogenesis. we can use it to decrease the angiogenesis as a key method for treatment and decreasing the symptom and disability of some inflammatory disease like MS.

Methods and Materials: This case-control study was done on 102 cases of MS lacking any other inflammatory or underlying condition and 75 healthy volunteer subjects selected through systematic randomization. We used a combination of Expanded Disability Status Scale (EDSS), VEGF and sVEGFR1 measured by Elisa technique to examine the correlation between the level of VEGF and sVEGFR1 and severity of MS in patients and its role for decreasing of symptoms. We also investigated the influence of sex, age, treatment duration, and number of frequencies on such variables.

Results: We found an increased level of VEGF and sVEGFR1 in MS patients compared to healthy controls. We also showed that the greater severity of MS, the higher serum level of VEGF and sVEGFR. There was significant effect of age, treatment duration, and number of frequencies on serum level of VEGF and sVEGFR1.

Conclusion: VEGF level is higher in MS patients and sVEGFR1 increases in MS patients as a physiological response to exacerbations of the symptoms to decrease the angiogenesis as well as inflammation. Such condition may improve the symptoms and prevent more disability.

Keywords: Multiple Sclerosis, Vascular Endothelial Growth Factor, Soluble VEGFR-1, Angiogenesis