## **SUMMARY AND CONCLUSION**

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Rheumatoid arthritis is an inflammatory joint disease with autoimmune basis. The exact mechanism leading to disease development and influencing the disease course are not known. Certain environmental and genetic factors are proven to be important in pathogenesis of rheumatoid arthritis. In particular, the association of HLA-DRB1 alleles that carry the so called shared epitope with rheumatoid arthritis development and/ or severity is, well established (*Rak et al; 2010*).

cells traffic Some are known to from mother into the fetus and from the fetus the mother to during pregnancy.

Small number of these cells persist in their host for decades later, this phenomena is known as microchimerism. The role of microchimeric cells is a matter of great contaverse, some autoimmune disease improved during pregnancy and some got worse. However microchimerism is also found in organs affected by non-immune conditions and detected in peripheral blood of normal individuals, rising the question of whether these cells are of beneficial effect, or play a role in initiation of autoimmune diseases (*Nelson*; 2009).

Rheumatoid arthritis patient who genetically lack of shared epitope can acquire this shared epitope as persistent microchimerism, suggesting that microchimerism could be a risk factor in sever rheumatoid disease (*Rak et al*; 2010).

(B)

Microchimerism, may has a protection role for rheumatoid arthritis if microchimeric shared epitope containing special sequence of amino acids in HLA-DRB1, as DERAA at the position 70-74 (*Nelson*; 2009).

This role study designed to evaluate the of microchimerism in rheumatoid arthritis patients by detecting the CD34+ and CD38+ by using the flowcytometry.40 rheumatoid patients diagnosed according to the criteria of American College Association divided into 3 groups multipara, 10 nulipara, and 10 male, and 60 healthy person as control divided into 3 groups 20 multipara, 20 nulipara, and 20 male.

The result of our study demonstrate insignificant correlation between CD34+,CD38+ in all 3 rheumatoid groups and control.

We also found significant correlation between CD34+,CD38+ and ESR, Hb, morning stiffness, and articular index in multipara group and insignificant correlation between

CD34+,CD38+ and ESR, Hb, morning stiffness, and articular index in the other 2 groups.

Beside the above results ,we found highly significant correlation in considering CD34+, CD38+ in multipara in comparison with the other 2 rheumatoid groups.

In conclusion, our study shows that microchimerism may be responsible for rheumatoid arthritis activity and severity of the disease, we also could not prove the role of microchimerism in rheumatoid pathogenesis.

Finally, detection of microchimerism by using the flowcytometry can give some predilection of the course, severity of rheumatoid arthritis.