ORIGINAL ARTICLE

17β -estradiol Hormone and Interleukin 1-beta Change Related to Menopause in the Women with Rheumatoid Arthritis

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Abstract

The depletion of 17β-estradiol-2 (17β-E2) is one of the factors that cause the risk of rheumatoid arthritis (RA) in females in the case of menopause. The aim of this study is to investigate whether the change in 17β-E2 levels and interleukin 1-beta (IL-1β) is associated with menopause in RA women and whether there is a relationship between them. 96 RA women were divided into three groups as follows: Group 1 (women of reproductive age) – 30, Group 2 (premenopausal women) – 32 (menstrual or normal menstrual period without menstruation for a period of not >6 months, and Group 3 (postmenopausal women) – 34 women in menopause (menopause for at least 12 months). Serum levels of 17β-E2, IL-1β, and anti-cyclic citrullinated peptide were evaluated by enzyme-linked immunosorbent assay. The results showed that a change in concentration of 17β-E2 resulted in excessive production of IL-1β in women during reproductive age, premenopausal, and postmenopausal compared to female control. Furthermore, there is a highly inverse correlation between IL-1β and 17β-E2 in the serum of pre- and post-menopausal RA women. On the other hand, the study showed a positive correlation between IL-1β and sex hormones 17β-E2 in women of reproductive age who suffer from RA. Moreover, the study confirmed that the most risk factor is 17β-E2. The study showed that a lack of 17β-2 concentration after menopause causes an increased concentration of IL-1β and this, in turn, stimulates the development of RA disease during menopause. Menopause-associated 17β-E2 deficiency plays the major role in the pathogenesis of RA.

Key words: 17β-estradiol, interleukin 1β, menopause, rheumatoid arthritis