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

INTRODUCTION

Rheumatoid arthritis (RA) is the most common chronic inflammatory systemic autoimmune disease with multifactorial etiology.[1] It characterized by pain, swelling, stiffness, and synovial joint inflammation due to immune-mediated response.[2] Its prevalence of about 1% of the general population in western countries depends on age. RA is more prevalent among women,[3] but the difference is great during the reproductive years.[4] Interestingly, the highest incidence among women has been reported between 55 and 64 years of age, during the pre- or post-menopausal stages.[5] These gender differences seem to be highest before menopause,[6] which have led to the hypothesis that female hormonal factors play a role in RA disease development.[7] Menopause is a turning point in every woman's life. The onset of menopause is associated with a hormone deficiency, which is a contributory factor for the increased incidence of RA [8]. Alpizar-Rodriguez , suggesting that the acute decline in ovarian function might contribute to the development of autoimmunity associated with RA.[9]

Hormonal changes have assumed that they may act as a trigger for the evolution of certain diseases that occur around menopause.[10] 17β-estradiol is the primary female sex

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Received: 01-01-2019
Revised: 22-01-2019
Accepted: 29-01-2019