ROLE OF ESTROGEN REPLACEMENT THERAPY IN THE CONTROL OF IMMUNE SYSTEM IN POSTMENOPAUSAL OSTEOPOROSIS

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INTRODUCTION

Menopause is the main factor responsible for the loss of bone mass in women [1]. It is now clear that the antiresorptive activity of estrogen is the result of genomic and non-genomic effects on bone marrow and bone cells, resulting in a reduction of osteoclasts (OC) formation, in an increase of OC apoptosis and in a decreased ability of mature OC to resorb bone [2]. It is also known that the increase of bone resorption secondary to estrogen deficiency is primarily due to increased OC formation stimulated by several cytokines. The important role of TNF in the molecular mechanisms through which estrogen deficiency causes bone loss has been demonstrated in animal models and also in humans [3,4].

In our recent work we have shown that estrogen deficiency increases osteoclastogenesis through increased production of pro-inflammatory cytokines such as TNF-alpha and RANKL, and that T cells play a key role in postmenopausal bone loss and in osteoclastogenesis in humans [4].

The aim of this study is to investigate the influence of estrogen replacement therapy (HRT) in the control of the immune system and osteoclastogenesis.

METHODS

We enrolled in the study 38 female patients affected by postmenopausal osteoporosis. Patients were randomized to receive HRT plus Calcium and Vitamin D (11), RLX plus Calcium and Vitamin D (12) and Calcium and Vitamin D alone (15).

We evaluated by ELISA technique cytokines levels in sera and in cell cultures supernatants from all patients, at baseline and after 3, 6 and 12 months of therapy.

RESULTS

The authors have no conflict of interest in this study.

REFERENCES

1. Iqbal J. et al. "Follicle-stimulating hormone stimulates TNF production from immune cells to enhance osteoblast and osteoclast formation." PNAS USA (2006); 103:14925-30.

