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Research Article

OSTEOARTHRITIS IN POSTMENOPAUSAL WOMEN

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ABSTRACT

Osteoarthritis is the most common disease of women after menopause. There are many factors to develop the disease. Hormones play important role to in this context. The objective of the present study is to determine whether the levels of thyroid and sex hormones are associated with osteoarthritis (OA) in postmenopausal women. Forty three patients suffering from OA and twenty control subjects were included in this study. Thyroid and sex hormones were measured in the serum by enzyme linked immunosorbent assay technique. In OA patients serum estrogen levels were low as compared to control subjects($p<0.001$), but these patients did not show any significant change in thyroid hormones and progesterone hormone levels when compared with control subjects. The findings suggest that estrogen deficiency after menopause may contribute to develop OA in postmenopausal women.

Key Words: Hormones, Osteoarthritis, Postmenopausal women

INTRODUCTION

Osteoarthritis, is the most common chronic joint condition in the old age.It is a degenerative disorder of synovial joint, characterized by loss of articular cartilage, with reactive change in subchondral & marginal bone, synovium & para articular structure. Obesity is a risk factor for development of knee arthritis[1]. Age is a major risk factor for the occurrence of osteoarthritis, but the mechanism by which the age is involved in the etiology of osteoarthritis is largely unknown[2,3].

A role for sex hormone in development of osteoarthritis has been suggested based on the increasing rate of osteoarthritis during or soon after menopause [4]. Estrogen lower serum calcium & phosphate and inhibit bone resorption. Hurley *et al* [5] found low concentratin of calcitonin in patients who were on estrogen replacement therapy. Normal human osteoblast cells express progesterone receptors [6]. Chaissan et al[7] has found an association between thyroid status & chondrocalcinosis or osteoarthritis. Human auricular chondrocytes produce IL-6. IL-6 production by stromal osteoblastic cells is inhibited by 17B-estradiol at transcriptional levels through receptor mediated mechanisms. Estrogen deficiency as well as its effect on IL-6 production may also make osteoclast precursors sensitive to IL-6.

MATERIALS AND METHODS

The osteoarthritis patients having age 50 years or more were selected from civil hospital, Karachi. Duration of study was about one year that is from December 2012 to August 2013.The study was performed in accordance with ethical standards, permission was given by the Civil Hospital Karachi, Pakistan. The history was recorded by questionnaire. Patients were selected, having symptoms of

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osteoarthritis, such as pain, stiffness, soreness, aching, discomfort, swelling & tenderness. Radiographs were taken for assessment of severity of osteoarthritis. Patients taking any hormone replacement therapy (HRT), non steroidal anti-inflammatory drugs (NSAID), having metabolic disease, rheumatoid arthritis (RA), joint, systemic lupus erythromatosis (SLE) were excluded from the study.

Control Postmenopausal women were selected having no sign of osteoarthritis clinically. BMI (Body mass index) was calculated of all patients and control subjects. Blood samples from osteoarthritis female patients and control subjects were collected. About 10ml blood was collected in centrifuge tubes. Serum was separated by centrifugation and was kept at -70ºC till the further analysis of biochemical parameters. Estradiol, progesterone, calcitonin were analyzed by ELISA.

RESULTS AND DISCUSSION

A total of 43 female postmenopausal osteoarthritis patients and 20 control subjects participated in the study.

Table 1 shows the age and BMI of controls and osteoarthritic subjects, but no significant differences were found between BMI of patients and control subjects.

Table 2 shows the status of thyroid, calcitonin, estrogen and progesterone hormones in patients and control groups. Estrogen hormone was statistically decreased ($p<0.001$) in patients as compared with control subjects. Osteoarthritis is a chronic painful, disabling condition affecting synovial joints. It is a common age related disorder, which is present in more then 10% of the persons older than 65 years of age, it results in substantial disability and economic cost in elderly person[8] .Osteoarthritis is a disease of cartilage, early cartilage abnormalities in patients with this disease are followed by changes in periarticular trabecular bone & thickning of the subchondral plate [9,10]. The women experience more severe arthritis in the knee. Genetics, anatomy and prior knee injury are risk factors for developing osteoarthritis[11].

There are several risk factors of osteoarthritis obesity may be an important risk factor. Body weight & body mass index was associated with an increased risk of osteoarthritis at all joints [12]. In healthy peoples, cartilage defects increase with increasing age and body mass index.

The high incidence of osteoarthritis in women just after menopause has suggested that estrogen deficiency play a role in causing disease. The studies have shown that women taking estrogen have a decreased prevalence & incidence of radiographic osteoarthritis[13]. In the present study there was a significant decrease in the level of estrogen in post menopausal women patients as compared to post menopausal control women. Thyroid hormones are essential for the development, growth & metabolism of tissue, including bone. It is known that the action of thyroid hormones on bone tissue can be direct [14,15].

Osteoarthritis is a disease in which there is no balance between synthesis and degradation of collagen & evidences tell us that thyroid hormones fail to inhibit collagen synthesis. Present study do not show any change in the level of thyroid hormone in the osteoarthritis patients as compared to control subjects (Table-2), chaisson et al found no change in thyroid hormone levels. Evidences tell us that thyroid hormones fail to inhibit collagen synthesis [16].

CONCLUSIONS

Osteoarthritis is a disease of old age and mainly affected by several factors. Our findings suggest that hormones, like estrogen deficiency after menopause may develop the disease of OA in postmenopausal women.

Table 1: Age and BMI of control and osteoarthritis postmenopausal female patients.

Characteristic	Control (n=20)	Patients (n=43)
Age (years)	56 ± 2.02 (49-63)	57 ± 1.9 (48-65)
BMI (Kg/m^2)	20.9 ± 0.73	21.6 ± 0.92

Values are the mean ± S.E.M.

Table 2 : Hormones in control and osteoarthritis subjects

Group	T3 ng/dl	T4 ug/dl	Calcitonin pg/ml	Estrogen pg/ml	Progesterone ng/ml
Control	129.27+1.44	6.15+0.28	21.17+0.12	51.02 +5.38 (20)	3.65 +0.71 (20)
Patients	130.09 +1.91 (43)	5.97 +0.23 (43)	20.87 +0.11 (43)	* 27.55 +3.46 (43)	2.89 +0.45 (43)

Values are the mean ± S.E.M. * $p < 0.001$ as compared to control subjects

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