INSIGHT ON THE RELATIONSHIP BETWEEN OSTEOPOROSIS AND OSTEOARTHRITIS

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ABSTRACT

In this study, we tried to investigate some of the factors that may have a role in the pathogenesis of osteoarthritis OA and osteoporosis OP, such as BMI, levels of estrogen, IGF-1 and bone mass density. We correlated all these parameters to find the relation between those two pathologic conditions.

Forty postmenopausal females were studied, 20 with OP and 20 with OA. Our results showed a significant elevation of estrogen and IGF levels and BMD in OA group in relation to OP group. The mean BMI showed a significant increase in OA than OP. This inverse relation could be explained by the difference in growth factors and estrogen that stimulate bone formation.

In conclusion: There is an inverse relationship between OA and OP. Thus OA might have a protective or retarding effect on the development of OP and could be a negative risk factor for OP. This could be an important element in determining patients who at risk developing OP and should take preventive therapy for OP at the time of menopause.
INTRODUCTION

OP and OA are generally considered to occur as a general phenomenon, associated with aging and wear and tear. However, the clinical experience and the epidemiological evidence that OP and OA, although both very common in menopausal women are rarely seen together (Dequeker, 1996). Because of the inverse relationship of bone density in OA and OP, it was thought that studies of pathophysiology of OA might provide some insight into the understanding of OP (Dequeker et al., 1993).

This prompted us to investigate changes in the clinical, laboratory and bone mass density in OA and OP patients to find out the possible mechanism accounting for these changes and the possible protective measures.

PATIENTS & METHODS

This study was conducted on forty postmenopausal female patients, 20 patients with primary OP who fulfilled the criteria of Oruber et al. (1984), and 20 OA patients diagnosed according to Kellgren-Moor (1952).

All Patients Were Subjected To:

- A detailed medical history and thorough clinical examination.
- Functional activities of the patients were scored.
- Pain severity was assessed using the visual analogue scale.
- BMI was calculated by this equation.

\[ \text{BMI} = \frac{\text{Weight in Kg}}{(\text{Height}^2 \text{ (cm)})} \]

Laboratory investigations:

- CBC, ESR, RF, renal function tests (serum creatinine), liver function tests (SGOT, SGPT, alkaline phosphatase) to exclude secondary OA or OP.
- Estradiol using EIA gear estradiol.
- Serum insulin-like growth factor-1 (IGF-1) by radioimmunoassay kit (RIA).
Radiological investigation:

Quantitative CT scan (QCT) using CT scanner somatome HIQ (Siemens AG) at lumbar vertebral bodies L1-L2.

RESULTS

This study was conducted on 40 primary OA and OP postmenopausal females. The age of OA patients ranged from 41 to 45 years while that of the OP patients ranged from 42 to 75 years. Most of them were housewives. None of them were alcoholics or cigarette smokers.

Table (1): Clinical, laboratory and radiological data of OP Vs OA subjects.

<table>
<thead>
<tr>
<th></th>
<th>OP (No.=20)</th>
<th>OA (No. =20)</th>
<th>t</th>
<th>p</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>57.1 ± 9.4</td>
<td>55.4 ± 3.69</td>
<td>0.75</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (Kgm/cm²)</td>
<td>30.2 ± 3.8</td>
<td>33.4 ± 4.8</td>
<td>2.3</td>
<td>&lt;0.05</td>
<td>S</td>
</tr>
<tr>
<td>Disease duration (years)</td>
<td>7 ± 2.5</td>
<td>6.3 ± 2.8</td>
<td>0.5</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Parity</td>
<td>6.6 ± 1.5</td>
<td>4.8 ± 2.2</td>
<td>2.5</td>
<td>&lt;0.05</td>
<td>S</td>
</tr>
<tr>
<td>Duration of menopause (years)</td>
<td>10.1 ± 7.5</td>
<td>9.8 ± 4.8</td>
<td>0.91</td>
<td>&gt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Estradiol (picogm/ml)</td>
<td>14.3 ± 7.9</td>
<td>23.5 ± 20.1</td>
<td>6.2</td>
<td>&lt;0.001</td>
<td>HS</td>
</tr>
<tr>
<td>IGF-1 (ng/ml)</td>
<td>73.8 ± 30.1</td>
<td>32.2 ± 25.5</td>
<td>14.6</td>
<td>&lt;0.001</td>
<td>HS</td>
</tr>
<tr>
<td>BMD (mg HA/ml)</td>
<td>-2.3 ± 1.8</td>
<td>+2.5 ± 0.4</td>
<td>10</td>
<td>&lt;0.001</td>
<td>HS</td>
</tr>
</tbody>
</table>

BMI = Body mass index, IGF-1= Insulin like growth factor-1, BMD = Bone mass density.

DISCUSSION

OA and OP are major health problems in the elderly. However, the coexistence of the two disorders in individual patients has been considered to be rare. Hence, an inverse relationship between OA and OP has been proposed (Burger et al., 1996). Our results showed that patients with OP were slender, having lower serum levels of estradiol and IGF-1 than OA patients, with radiological findings suggestive of OP changes and a low bone mineral density than OA patients.
Whereas OA patients were obese, having higher serum levels of estradiol and IGF-1 than OP patients, with higher bone mineral density than OP patients. All these factors may mediate the inverse relationship between OA and OP. This relation was previously supported by many studies. In 1985 Dequeker observed that osteoporotic women were shorter, more slender, and have less fat, muscle girth and strength, while women with OA, although of comparable age and skeletal size were more obese and had more fat muscle mass and strength. These findings support the idea that osteoarthritis and osteoporosis are two different disease entities and not simple phenomena of aging and wear and tear.

Cooper et al. (1991) Proposed that the inverse relationship between OA and OP could be explained based on excess estrogen in OA and its decrease in OP. They explained this proposal by the predominant occurrence of OA in obese perimenopausal women and since obesity in women is a known cause of hyperestrogenism and since loss of estrogen being the major contributory factor in postmenopausal bone loss (OP).

Cumming and Black (1993) explained this relationship by the difference in body weight between people with OP (who tend to be underweight) and people with OA (who tend to be over weight). Another explanation is that physical activity independently increases the risk of OA and reduces the risk of hip fractures. Since high levels of physical activity during youth have been shown to be of key importance in reaching peak bone mass. An alternative interpretation is that they represent variable responses of subchondral bone to mechanical stress. In OP, the osteoblastic response is blunted, whereas in OA, it is accentuated.

It is also possible that there is direct causal relation between the pathological processes of OA and OP. Hart et al. (1994) supported this negative correlation because of increased IGFs and bone mineral density in OA and then decreases in OP patients.

Burger et al. (1996) found that women with OA had significantly more bone and the bone was significantly stiffer. They had higher compressive strength, higher osteocalcin, higher insulin-like growth factor I and II and transforming factor beta content, and a mineralization profile shifting to higher
densities. These quantitative and qualitative differences in bone may explain the inverse relationship between OA and OP.

On the other hand, other studies failed to support this relationship. Price et al. (1987) found a significant difference in bone mineral density when adjusting for age, which disappeared after adjusting for height and weight.

Again, Jones et al. (1995) suggested that individuals with OA, despite higher bone density, are not protected against non-vertebral osteoporotic fracture, apparently due to worsened postural stability and thus an increased tendency to fall.

This discrepancy between results can be explained by the opinion of Burger et al. (1996).

First: This may be related to the fact that most studies included only small numbers of patients, with often poorly matched controls. Second: The methods for assessing both OA and OP differed. Third: not all analysis was carried out with adjustment for age and weight or BMI. Finally: we can conclude like other authors that the findings of an inverse relationship between OA and OP have clinical and theoretic importance.

The knowledge that OA is a negative risk factor for OP can be an important element in determining which patients should begin preventive therapy for OP at the time of menopause i.e. OA could be a good negative indicator for finding patients at risk for OP.

The arthropometric, biologic and biochemical differences between primary OA and OP reflect that systemic and metabolic factors are involved in the pathophysiology of these diseases and that these common crippling diseases are not simple consequences of aging. This fact prompts us to consider prevent and treat these diseases.
REFERENCES


نظرية العلائق بين مرض الفصالي العظمي المفصلي ومرض وهن العظام

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في هذه الدراسة حاولنا فحص بعض العوامل المؤثرة في بانولوجية مرض الالتهاب العظمي المفصلي ومرض وهن العظام ومن هذه العوامل مؤشر الكتلة الجسمية، مستوى الهرمون الأنتيوي ومستوى عامل النمو شبيه الأنسولين-1 والكثافة العظمية وقد تم مقارنة كل هذه العوامل لإيجاد العلاقة بين هذين المرضى.

اشتملت هذه الدراسة على 40 مريضة بعد سن البالغين، 20 يعانين من مرض الفصالي العظمي المفصلي و20 يعانون من مرض وهن العظام.

أوضحنا النتائج ارتفاع نو دالة إحصائية في مستوى الجين ومستوى عامل النمو شبيه الأنسولين-1 و التي يساعد على تكوين العظام و من هذا نستخلص وجود علاقة عكسية بين مرض الالتهاب العظمي المفصلي ومرض وهن العظام.

ومن هذا يتبين أن الالتهاب العظمي المفصلي يمكن أن يكون له تأثير حامي أو مؤخر لحدث وهن العظام و يمكن أن يكون عامل سلبي مضاد لوهن العظام. و من هذا يمكن تحديد المرضى المحتاجين إلى علاج وقائي لوهن العظام.