Effect of hyperlipidemia in the atherosclerotic patients on sex hormones

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Abstract

The current study was designed to explain the relationship of testosterone and estradiol levels with the serum lipid profile alteration. It has included the measurement of serum testosterone and estradiol levels in healthy subjects (40 males and 43 post-menopausal females), atherosclerotics male (40) and post-menopausal (41) atherosclerotic females. Serum triglyceride, cholesterol, HDL-cholesterol, LDL-cholesterol and VLDL-cholesterol were also estimated in those individuals. The results revealed significant (p < 0.05) elevation of serum estradiol level in atherosclerotic males when compared with those of the control group. Estradiol also was found decreased significantly (p < 0.05) when testosterone levels indicated a significant (p < 0.05) in atherosclerotic post-menopausal female when compared with control group. The results identified the details of serum lipid profile changes in conditions of different levels of sex steroids.

Introduction

Initiatives seek to understand the relevance of sex steroids with the abnormalities of lipids and lipoprotines in details, this has been always the hope of biochemists. Hyperlipidemia is a serious medical problem worldwide. The frequent type of such abnormality is hypercholesterolemia. It is the leading cause for development of ischemic heart diseases (1). Hyper triglyceridemia is less common, but it is also a
risky pathological condition. The consequences of this abnormality is pancreatitis and steatosis (2).

Sex steroids are a subset of sex hormones that produce sex differences or support reproduction. They include estrogens, progesterones, and androgens. The major male androgen is testosterone (3, 4).

In observational studies postmenopausal women using estrogen have been found to have a reduced risk of major clinical coronary disease and reduced cardiovascular disease mortality (5). However, a large randomized clinical trial of estrogen replacement therapy for secondary prevention of coronary heart disease in postmenopausal women failed to demonstrate a reduced rate of coronary heart disease events (6). Finally, two studies examining the relation of estrogen replacement therapy to carotid atherosclerosis have yielded conflicting results (7, 8).

**Materials and methods**

- Patients and control group

Eighty one atherosclerotic patient who attended the Al-Hakeem Hospital in the Najaf were enrolled in this study.

They were 40 males (55 ± 10 y), and 41 postmenopausal females (60 ± 5 y).

The control groups consisted of age matched 83 individuals. They were, 40 males (44 ± 7.2 y), and 43 postmenopausal females (60 ± 5.2 y). Blood samples were taken from the fasting patients and control group at 9 AM.

Atherosclerosis cases and controls were chosen on the basis of their average carotid artery ultrasound measurements. A woman was considered postmenopausal if she had not menstruated in the last two years.
Postmenopausal women were further classified as having undergone surgical menopause if they had a bilateral oophorectomy. Natural menopause also described nonmenstruating women 55 years of age or older who had a hysterectomy and had at least one intact ovary.

- **Blood samples**

  Disposable syringes and needles were used for blood collection. Blood samples were obtained from patients and the control group by vein puncture. Sample were allowed to clot at 37°C, and then centrifuged at 3000xg for 10 minutes. Serum were stored at -20°C until analysis of the hormone.

- **Determination of serum estradiol and testosterons levels in patients and control groups**

  The principle assay combines and enzyme immunoassay competition method with a final detection (ELFA).

  Total cholesterol, triglyceride, and HDL-cholesterol was determined using commercially available kit (Bio Merieux-France). LDL-cholesterol, and VLDL-cholesterol was calculated using the following formula that developed previously (10), in which: TC = (HDL-cholesterol) + (VLDL-cholesterol) + (LDL-cholesterol).

  VLDL-triglycerides concentration was determined by dividing triglycerides value obtained on 2.2 (11) when triglycerides concentrations are given in mmol/L.

**Biostatistical analysis**

The results were expressed as mean ± SD. Students t-test was used for assessment of the results. Significant variation was considered when the P value was than 0.05.
Results and discussion

The estimation of serum estradiol, and testosterone concentrations revealed significant (p < 0.05) elevations of estradiol in atherosclerotic males when compared with those of control group. Serum testosterone didn't show significant variation during a comparable elevation (table 1).

In postmenopausal females estradiol was found to decrease significantly (p < 0.05) in the atherosclerotic females when compared with those of control group, while testosterone level was demonstrate to increase significantly (p < 0.05) to those of control group (Table 2).

In atherosclerotic patients, males and postmenopausal females, cholesterol, triglycerides, LDL, and VLDL were elevated significantly (p < 0.001) for triglyceride and VLDL and (p < 0.01) for cholesterol and LDL-cholesterol) in comparison with those of the control group. HDL-cholesterol didn't demonstrate significant change during a comparable evaluation (Table 3, 4).

Several studies have shown beneficial cardiovascular effects of exogenous estrogen, including lower body weight (12-14), increased high density lipoprotein (HDL) cholesterol and decreased low density lipoprotein (LDL) cholesterol (12, 14-16) levels, lower fasting insulin and glucose levels (10, 14), improved brachial artery blood flow (15) and regression of atherosclerotic plaques (16).

Prior to menopause, women have a mush lower risk of cardiovascular disease compared with men of the same age (17); However, menopause initiates a phase of increased risk (18). At the time of menopause compared with the premenopausal state, a woman's endogenous hormonal milieu changes; there is a relative estrogen deficiency and relative increase in testosterone levels.

A weaker form of estrogen, estrone, continues to be synthesized as a result of peripheral conversion from adrenal androstendione in the fat.
Given what is known about the relation between estrogens, androgens and cardiovascular disease in women receiving estrogen replacement therapy, postmenopausal women with significant atherosclerosis and cardiovascular disease would be expected to have lower endogenous estrogen levels and higher endogenous androgen levels than those without significant atherosclerosis.

Studies examining the relation between endogenous postmenopausal hormone levels and cardiovascular disease have yielded conflicting results. Several studies have found no association between sex hormone levels and risk of death from cardiovascular disease (20-22). However, two studies suggest a protective effect of higher androgen levels on cardiovascular disease in postmenopausal women (23, 24).

An interesting point is that all serum lipid profile parameters are significantly changes in the stated population accept HDL-cholesterol concentration. The reason seems to be unclear and not fully understated it needs further clarification. Several reports have stated alterations in the HDL-cholesterol subclasses but the total function still remained constant (25).

Table (1). Serum estradiol and testosterone levels in control group and atherosclerotic males.

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Patients</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estradiol</td>
<td>29.42 ± 19.37</td>
<td>39.17 ± 24.15</td>
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<tr>
<td>Testosterone</td>
<td>3.76 ± 1.89</td>
<td>3.70 ± 2.74</td>
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</table>
Table (2). Serum estradiol and testosterone levels in control group and atherosclerotic postmenopausal females.

<table>
<thead>
<tr>
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<th>Control group</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Estradiol</td>
<td>25.91 ± 23.23</td>
<td>32.05 ± 30.72</td>
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<tr>
<td>Testosterone</td>
<td>0.55 ± 0.11</td>
<td>0.61 ± 0.60</td>
<td>0.05</td>
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</table>

Table 3. Serum lipid profile in control group and atherosclerotic males

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
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</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>2.28 ± 1.14</td>
<td>4.82 ± 2.12</td>
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<tr>
<td>Cholesterol</td>
<td>5.22 ± 2.20</td>
<td>6.95 ± 2.28</td>
<td>0.01</td>
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<tr>
<td>HDL-cholesterol</td>
<td>0.97 ± 0.30</td>
<td>0.76 ± 0.64</td>
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<tr>
<td>LDL-cholesterol</td>
<td>3.63 ± 1.80</td>
<td>4.42 ± 2.22</td>
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<tr>
<td>VLDL-cholesterol</td>
<td>1.02 ± 0.46</td>
<td>2.05 ± 1.33</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Table 4. Serum lipid profile in control group and atherosclerotic postmenopausal females

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Patients</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>1.55 ± 0.61</td>
<td>3.99 ± 1.89</td>
<td>0.001</td>
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<tr>
<td>Cholesterol</td>
<td>4.73 ± 1.78</td>
<td>7.74 ± 3.60</td>
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<tr>
<td>HDL-cholesterol</td>
<td>0.94 ± 0.35</td>
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<tr>
<td>LDL-cholesterol</td>
<td>3.29 ± 1.70</td>
<td>4.86 ± 2.75</td>
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<td>VLDL-cholesterol</td>
<td>0.71 ± 0.20</td>
<td>0.85 ± 0.80</td>
<td>0.001</td>
</tr>
</tbody>
</table>

References


14- Kritz-Silverstein D, Barrett-Connor E. Long-term postmenopausal hormone use, obesity, and fat distribution in older women. JAMA; 275: 469.


تأثير فرط الشحوم لدى الأشخاص المصابين بتصلب الشرايين على البروتينات الجنسية

الخلاصة :

تم تصميم الدراسة الحالية لتوضيح من العلاقة بين مستويات كل من التستوستيرون والأندروسترون مع تغيرات الدهون المصلية . تضمن الدراسة تقدير مستويات التستوستيرون والأندروسترون في مجموعة من الأشخاص (40 رجل و 43 امرأة) مصابين بتصلب الشرايين. 

وذكرت الدراسة تقييم مستويات الدهون المصلية لدى المصابين بتصلب الشرايين عند مقاانتهم مع مجموعة السيطرة. ظهرت دراسة تقييم مستويات الدهون المختلطة لدى المصابين بتصلب الشرايين عند مقاانتهم مع مجموعة السيطرة. 

قد تبينت نتائج تقييم مستويات الدهون المختلطة في مستويات مختلفة من البروتينات الجنسية.