Hormonal Disturbance in Normal and Diabetic Postmenopausal Women

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Abstract

Postmenopausal women are at high risk for diabetes, and previous studies suggest that endogenous sex hormones (as opposed to hormone replacement therapy) may play a role. LH, FSH, Prolactin, Testosterone, Progesterone, Estrogen, Glucose were measured for 63 individuals, 28 of these individuals were normal and 35 cases were diabetic patients from January 2010 to June 2011. When diabetes mellitus or insulin resistance occur these lead to decrease of insulin level and decrease of estrogen level. LH, FSH, Prolactin and Estrogen were decreased in diabetic postmenopausal women rather than control, while Testosterone, Progesterone and Glucose were increased.

Introduction

Hormones are substances that serve as vehicles for intracellular and extracellular communication. They have been defined as chemical substances that are produced by a gland in one part of the body, secreted into the bloodstream, and act on a target organ elsewhere. Feedback control mechanisms play a major role in the regulation of hormone levels and are an important feature of the endocrine system (1).

Important reproductive hormones include glycoprotein hormones made by the pituitary gland, such as luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Serum sex hormones may be related to the risk of several diseases in postmenopausal women. E1 (estrone) is secreted by the ovary, but most is converted from androstenedione in peripheral tissues. Estrone is a more potent estrogen than estriol but is less potent than estradiol. Estrone is the major circulating estrogen after menopause (2).

After the onset of menopause, the average women in developed countries live for nearly 40 years in an estrogen-deficient state. Both estrogen and progesterone affect cells response to insulin. This response, in turn, affects the level of blood glucose. Unfortunately, less than 20% of these women receive any form of treatment (3).

Obesity, fat distribution, and body composition also alter with age. Generally, fat mass increases until about the age of 65 and then it begins to decrease. Lean body mass decreases steadily from the fifth or sixth decade onward. In women, adiposity tends to
concentrate in the abdomen (central obesity) (4).

Diabetes mellitus (DM) is found to be associated with an increase in uterine size in postmenopausal women (5). In addition, the relative risk of endometrial cancer in diabetic women is four-folds higher than in nondiabetic women (4, 6). The risk of endometrial cancer also increases with the use of unopposed estrogen in non-hysterectomized women and is reduced with the use of cyclical or continuous progestins (7–9).

**Materials and Methods**

(Five-Ten) ml of venous blood was drawn from diabetic and control subjects after (12-14) hour fasting and allowed it to clot in plain tube at room temperature. The serum was aspirated after centrifugation at (3000 rpm) for 30 minutes, then divided into aliquots in plastic tubes and stored at (−20 °C) until the time of estimation. Serum samples were collected from 63 individuals, 28 of these individuals were normal and 35 cases were diabetic patients admitted to Tikrit Teaching Hospital in Tikrit city from January 2010 to June 2011. Postmenopausal diabetes women with age ranges from (48-70) year with patients of Type II D.M.

Patients and controls had hormonal assay of LH, FSH, PRL, Estrogen, Progesterone, Testosterone and Glucose. All these control individuals are non-diabetic and non-smoker with no familial history of diabetes or personal history of hypertensive, thyroid or renal diseases. Serum (LH, FSH and PRL) were determined by using AccuBind ELISA Microwells (sandwich enzyme immunoassay kit) (Monobind Inc., USA). Serum (Testosterone, Progesterone and Estrogen) were determined by using AccuBind ELISA Microwells (competitive enzyme immunoassay kit) (Monobind Inc., USA). Fasting blood glucose was measured by enzymatic oxidation method in the presence of glucose oxidase. Results were analyzed statistically using (t) test by using the statistical program Minitab. Averages were compared to calculations of the characteristics of the application Duncan's Multiple Range Test by probability level P ≤ 0.05.

**Results**

The Mean±SD of LH for female control and diabetic patients were (25.54±11.37) and (22.24 ±11.74) respectively. These results showed that there was no significant difference between female control and diabetic patients. FSH for female control and diabetic patients were (45.45±12.81) and (38.62±10.64) respectively. These results showed that there was significant difference (p≤ 0.05) between female control and diabetic patients. Prolactin for female control and diabetic patients were (10.04±5.46) and (7.1±2.94) respectively. These results showed that there was
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Significant difference (p≤ 0.05) between female control and diabetic patients. Testosterone for female control and diabetic patients were (0.86±0.29) and (1.04±0.42) respectively. These results showed that there was no significant difference between female control and diabetic patients. Progesterone for female control and diabetic patients were (1.09±0.58) and (1.73±0.75) respectively. These results showed that there was highly significant difference (p≤ 0.01) between female control and diabetic patients. Estrogen for female control and diabetic patients were (11.14±6.13) and (7.05 ±3.75) respectively. These results showed that there was significant difference (p≤ 0.05) between female control and diabetic patients. Glucose for female control and diabetic patients were (115.8±17.64) and (267.7±62.96) respectively. These results showed that there was highly significant difference (p≤ 0.01) between female control and diabetic patients.

Discussion

In this study, estrogen was decreased normally in postmenopausal women; therefore, LH and FSH was increased by feedback inhibition lead to increased testosterone and progesterone in this age but when diabetes mellitus or insulin resistance occur these lead to decrease of insulin level and decrease of estrogen level. Obesity itself increases insulin resistance, and the emerging dyslipidemia and disturbances in the coagulation system (10). These results were in agreement with the results found by (Maneesh et al (11), Abdullah & Bakry (12), Weher et al (13) and Kim (14)).

Postmenopausal women are at high risk for diabetes, and previous studies suggest that endogenous sex hormones (as opposed to hormone replacement therapy) may play a role. However, previous studies have not examined whether changes in sex hormones are associated with changes in glucose tolerance, and whether such changes might be explained by concurrent changes in adiposity and insulin. Previous studies have not examined the influence of diabetes prevention interventions upon sex hormones (14).

The variations in circulating FSH levels with increasing age are most probably due to changes in ovarian physiology affecting the secretory pattern of the gonadotrope, and the ovary becomes increasingly resistant to stimulation by gonadotropins, probably due to the decreased number of follicles, which leads to a decline in the production of both estrogens and inhibins. A number of studies (15-20) have shown low circulating concentrations of inhibin B in older persons.
reproductive-age women. Whether the rise in FSH is in part the result of a primary neuroendocrine change or is explicable entirely by a response to falling feedback signals from the aging ovary is controversial. The present study aimed to make a detailed longitudinal investigation of the hormonal characteristics of women during postmenopausal in normal and diabetic cases.

**References**

Table: - The Mean±SD of all parameters in control and diabetic Postmenopausal women.

<table>
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<tr>
<th>Tests</th>
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<tr>
<td>Number of subject</td>
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<td>35</td>
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<tr>
<td>LH (mIU/ml)</td>
<td>25.54±11.37</td>
<td>22.24±11.74</td>
</tr>
<tr>
<td>FSH (mIU/ml)</td>
<td>45.45±12.81</td>
<td>38.62±10.64</td>
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<td>7.1±2.94</td>
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<td>Testosterone (ng/ml)</td>
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<td>1.04±0.42</td>
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<td>Progesterone (ng/ml)</td>
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<td>Estrogen (pg/ml)</td>
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<td>Glucose (mg/dl)</td>
<td>115.8±17.64</td>
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