Evaluation of Serum Anti-Mullerian Hormone (AMH) and Plasma Metastin Levels in Women with Polycystic Ovary Syndrome (PCOS)

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Abstract

PCOS is one of the most prevalent endocrine disorders in women of reproductive age. The pathophysiological mechanisms leading to anovulation in PCOS are under investigation.

Aim of the Study: The current study was conducted to evaluate serum AMH and plasma metastin levels in PCOS patients and to assess their correlations with the PCOS-related reproductive and metabolic disturbance.

Subjects and Methods: Data from twenty three women with PCOS (ages 21-34 years) were compared with eighteen normal control women with regular menstrual cycles (ages 25-37 years). In each subject biochemical, hormonal, and ultrasonography parameters were studied.

Results: The study showed that serum AMH and plasma metastin concentrations were significantly higher in the PCOS patient group compared to the control group ($p<0.001$). Also, there was a significant positive correlation between the serum levels of AMH and plasma metastin both in the control ($r=0.42$, $p<0.001$) and in the PCOS patient group ($r=0.64$, $p<0.001$).

FSH serum levels were borderline lower in women with PCOS compared with normal healthy controls ($p>0.05$).

Whereas, LH, T and DHEAS serum levels were significantly increased in PCOS patients compared with normal healthy controls ($p<0.001$).

The total number of follicles 2-9mm as well as the main ovarian volume was significantly increased in women with PCOS patients compared with normal healthy controls ($p<0.001$).

AMH and metastin levels were negatively correlated with age ($r=-0.22$ and $r=-0.23$ respectively, $p<0.001$), Body Mass Index (BMI) ($r=-0.34$ and $r=-0.31$ respectively, $p<0.001$), Waist circumference (W) ($r=-0.25$ and $r=-0.26$ respectively, $p<0.001$) and FSH ($r=-0.21$ and $r=-0.23$ respectively, $p<0.05$). Whereas, both are positively correlated with LH ($r=0.48$ and $r=0.43$ respectively, $p<0.001$), T ($r=0.45$ and $r=0.38$ respectively, $p<0.001$), DHEAS ($r=0.32$ and $r=0.33$ respectively, $p<0.001$), total number of small follicles ($r=0.37$ and $r=0.34$ respectively, $p<0.01$), and main ovarian volume ($r=0.16$ and $r=0.19$ respectively, $p<0.05$).

Conclusions: In conclusion, both serum AMH and plasma metastin can be used in the diagnosis and follow-up of patients with PCOS. In addition, the correlation between the two markers implies an interrelated role in the pathogenesis of PCOS.

Key Words: Anti-Mullerian Hormone (AMH) – Plasma Metastin levels – Polycystic Ovary Syndrome (PCOS).

Introduction

POLYCYSTIC Ovary Syndrome (PCOS) is a common endocrine disorder. It affects 5-20% of women reproductive age and is the primary cause of anovulatory infertility [1,2].

Although Polycystic Ovary Syndrome (PCOS) has been recognized for more than 70 years there is no cohesive definition, and the diagnosis still causes debate [3]. It is characterized by anovulation manifested as oligo- or amenorrhea, elevated levels of circulating androgens, and polycystic ovaries as visualized by ultrasound. One of the most common features of PCOS is insulin resistance, represented in 85% of PCOS women [4]. The diagnosis is based on the presence of at least two of the described characteristics, as defined by the Rotterdam Consensus [5].

The pathophysiological mechanisms leading to anovulation in PCOS are under investigation [6]. The defective selection mechanism seems to lead in accumulation of small antral follicles, which contributes significantly to the production of Anti-Mullerian Hormone (AMH) [7].

AMH, a dimeric glycoprotein, is a member of the transforming growth factor-β superfamily. In females, it is synthesized by granulose cells of primary follicles and expressed in pre-antral and small antral follicles [2].

In PCOS, the follicular excess is mainly due to an increase of small antral follicles ranged from 2 to 5mm in size [8]. Therefore, elevated AMH
levels has been demonstrated in PCOS women from several investigators, suggesting that raised AMH levels could represent an indicator of disturbed ovulatory process in PCOS [9]. AMH has an inhibitory effect on FSH sensitivity, explaining in part the mechanism via which AMH interferes with anovulation [10].

Metastin, the KiSS-1 gene product (Kisspeptin), plays a critical role in reproduction by inducing gonadotropin release (LH and FSH) via the G Protein-coupled Receptor 54 (GPR54), which is suggested in mammals to be expressed in hypothalamic GnRH neurons [11,12]. The lack of GPR54 leads to abnormal sexual development in mice and to hypo gonadotropic hypogonadism in humans because of low circulating gonadotropin concentrations [13].

The correlation-ship between plasma metastin and the pathogenesis of women with PCOS was investigated in a limited number of studies, showing that the expression of metastin in PCOS women was significantly higher than that of normal women. The increased levels of metastin might be associated with the pathogenesis of women with PCOS [14,15].

In the present study, the concentration of serum AMH and plasma metastin were measured in PCOS patients and their correlations with the PCOS-related reproductive and metabolic disturbance were assessed.

Subjects and Methods

The study was done in the Aliaa Hospital in Kwait from 2010 – 2012.

Twenty three women with PCOS (ages 21-34 years) and eighteen normal control women with regular menstrual cycles (ages 25-37 years) were recruited for the study. The diagnosis of PCOS was on Rotterdam Consensus criteria [5], in which at least two of the following three criteria were met: Oligomenorrhea (<8 spontaneous menstrual cycles per year for at least 3 years before enrollment) oramenorrhea, clinical and/or biochemical hyperandrogenemia, and polycystic ovariess (>12 follicles in the 2-9mm range and/or an ovarian volume >10 cubic ml per ovary by vaginal ultrasound).

Exclusion criteria for the study included known cardiovascular disease, neoplasms, diabetes mellitus, renal impairment and hypertension. Congenital adrenal hyperplasia was excluded by a serum 17-hydroxyprogesterone (17-OHP) level of less than 3ng/ml. Oral contraceptives or other drugs that could interfere with the hormonal and metabolic studies, if administrated, were discontinued for at least 3 months before the study.

Inclusion criteria for the control group (non PCOS) were a regular menstrual cycle, normal body mass index, and no previous use of medication or oral contraceptives during at least 3 months before the study.

Fully informed written consent was obtained from all of the subjects before entry into the study.

In all women, weight, height, and Waist circumference (W) were measured. Body Mass Index (BMI) (Kg/m²) was calculated by dividing weight by squared height to assess obesity. W was obtained as the smallest circumference at the level of the umbilicus.

Overnight fasting blood samples were collected randomly from PCOS subjects after a spontaneous bleeding episode without hormone-induced withdrawal bleeding and/or between days 3 and 7 of the menstrual cycle for those women who ovulated spontaneously. They were centrifuged immediately; serum and plasma were stored at –20ºC until assayed for serum levels of Luteinizing Hormone (LH), Follicle-Stimulating Hormone (FSH), total Testosterone (T), Dehydroepiandrosterone Sulfate (DHEAS), serum AMH and plasma metastin.

Serum concentrations of LH, FSH and total Testosterone (T) were measured by an automated chemiluminescence system with intra- and inter-assay Coefficients of Variation (CV) of 5.4 and 8.0%, respectively, for LH; 3.0 and 4.6%, respectively, for FSH and less than 3% and 5% respectively, for T (Immulite 200; diagnostic products Corp., Los Angeles, CA, USA) [16].

Dehydroepiandrosterone Sulfate (DHEAS) were measured by RIA with intraassay CV less than 7% (Diagnostic Systems Laboratories, Inc., Webster, TX) [17].

Serum concentrations of AMH were measured by Enzyme-Linked Immunosorbent Assay (ELISA) kit (Diagnostic Systems Laboratories, Inc.) with intra- and interassay CV of 4.12 and 6.19%, respectively, for LH; 4.12 and 6.19%, respectively, for FSH and less than 3% and 5% respectively, for T (Immulite 200; diagnostic products Corp., Los Angeles, CA, USA) [17].

Ultrasound assessment:

Transvaginal ultrasound scans of the ovaries were performed during follicular phase, by experienced sonographers in all the subjects who par-
ticipated in the study (women with PCOS and controls). The presence of polycystic ovaries was diagnosed by the presence of 12 or more follicles in each ovary measuring 2-9 mm in diameter, and/or increased ovarian volume (>10 cm³). If characteristic ultrasound of PCO morphology, according to Rotterdam criteria, was found either on two ovaries or one ovary then the diagnosis of PCO morphology was established. Subjects with an ovarian cyst > 1 mm were excluded from the study.

Statistical analysis:

Statistical analysis was performed using the SPSS 14.0 software package (SPSS Inc., Chicago, IL, USA). Data are presented as means and S.D. The comparison of variables in two different groups was performed using student's t-test. The correlation between variables was analyzed by Spearman's rank correlation analysis. p-value <0.05 was considered statistically significant.

Results

Twenty three women with PCOS with a mean age of 24.4±1.2 (range 21-34 years) and 18 normal control women with regular menstrual cycles with a mean age of 27.15±1.7 (range 25-37 years) were recruited for the study. There was no statistically significant difference in mean age in healthy controls and PCOS patients (p=0.05) (Table 1).

There is no statistically significant difference between PCOS patient group and controls as reading BMI (23.4±1.7 vs. 22.1±1.5 respectively, p>0.05) and W (74.8±6.1 vs. 74.3±6.3 respectively, p>0.05) (Table 1).

FSH serum levels were borderline lower in women with PCOS compared with normal healthy controls (5.70±1.4 vs. 5.98±1.8 respectively, p>0.05). Whereas, LH serum levels were significantly increased in PCOS patients compared with normal healthy controls (8.42±3.2 vs. 4.95±2.3 respectively, p<0.001) (Table 1).

In PCOS patients, serum levels of T was significantly increased compared with normal healthy controls (0.78±0.31 vs. 0.37±0.12 respectively, p<0.001). Also, serum levels of DHEAS were significantly increased in PCOS patients compared with normal healthy controls (284.6±78.2 vs. 190.3±54.1 respectively, p<0.001) (Table 1).

The total number of follicles 2-9 mm on ultrasonography as well as the mean ovarian volume were significantly increased in women with PCO compared with control subjects (22.81±8.2 vs. 12.73±5.1, p<0.001 and 8.38±3.4 vs. 5.16±1.8, p<0.001, respectively) (Table 1).

Serum level of AMH:

Serum AMH concentration was significantly higher in the PCOS patient group compared with the control group. Mean value of serum AMH concentration was 7.52±3.1 ng/ml in the patient group versus 3.1±1.1 ng/ml in control group (p<0.001) (Table 2).

AMH serum levels were negatively correlated with range (r=−0.22, p<0.001), BMI (r=−0.34, p<0.001), W (r=−0.25, p<0.001) and FSH (r=−0.21, p<0.05). Whereas, it is positively correlated with LH (r=0.48, p<0.001), T (r=0.45, p<0.001), DHEAS (r=0.32, p<0.001), total number of small follicles (r=0.37, p<0.01), and mean ovarian volume (r=0.16, p<0.05) (Table 3).

Plasma level of metastin:

Plasma metastin concentration was significantly higher in the PCOS patient group compared with the control group (0.27±0.13 vs. 0.16±0.9 respectively, p<0.001) (Table 2).

A significant negative correlation of metastin and age (r=−0.23, p<0.001), BMI (r=−0.31, p<0.001), W (r=−0.26, p<0.001) and FSH (r=−0.23, p<0.05) were found. Whereas, it is positively correlated with LH (r=0.43, p<0.001) and T (r=0.38, p<0.001), DHEAS (r=0.33, p<0.001), total number of small follicles (r=0.34, p<0.01), and mean ovarian volume (r=0.19, p<0.05) (Table 4).

Based on the data obtained from PCOS patients, Receiver Operating Characteristic (ROC) curves was obtained. The ideal cutoff values of serum AMH and plasma metastin concentrations were recommended to be 7.5 ng/ml and 0.25 pmol/L respectively. Using these cutoff values, the sensitivity and specificity of serum AMH were 89% and 78%, respectively and for plasma metastin the sensitivity and specificity were 74% and 67%, respectively.

Also, there was a significant positive correlation between the serum levels of AMH and plasma metastin both in the control (r=0.42, p<0.001) and in the PCOS patient group (r=0.64, p<0.001).

There was no statistically significant difference in mean age, BMI, W and FSH serum levels in healthy controls and PCOS patients (p=0.05). Whereas, serum levels of LH, T and DHEAS, total number of follicles 2-9 mm and the mean ovarian volume were significantly increased in PCOS patients compared with normal healthy controls (p<0.001).

There was a statistically significant difference in the levels of both serum AMH and plasma meta-
Endocrine parameters:

Plasma metastin (pmol/L)

Whereas, it is positively correlated with serum levels of LH, T, DHEAS, total number of small follicles and mean ovarian volume.

Plasma metastin levels were negatively correlated with age, BMI, W, and serum FSH levels. Whereas, it is positively correlated with serum levels of LH, T, DHEAS, total number of small follicles and mean ovarian volume.

Table (1): Clinical, endocrinal, and ultrasound parameters of women with PCOS and control groups.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (18) (Mean ± SD)</th>
<th>Patient (23) (Mean ± SD)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical parameters:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>27.5±1.7</td>
<td>24.4±1.2</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.1±1.5</td>
<td>23.4±1.7</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>W (cm)</td>
<td>74.3±6.3</td>
<td>74.8±6.1</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td><strong>Endocrine parameters:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FSH (mIU/ml)</td>
<td>5.98±1.8</td>
<td>5.70±1.4</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>LH (mIU/ml)</td>
<td>4.95±2.3</td>
<td>8.42±3.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T (ng/ml)</td>
<td>0.37±0.12</td>
<td>0.78±0.31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DHEA-S (μg/ml)</td>
<td>190.3±54.1</td>
<td>284.6±78.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Ultrasound parameters:</strong></td>
<td></td>
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<td></td>
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<tr>
<td>No. of follicles (2-9mm)</td>
<td>12.73±5.1</td>
<td>22.81±8.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ovarian volume (cm³)</td>
<td>5.16±1.8</td>
<td>8.38±3.4</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*: Student’s t-test.

Table (2): Serum AMH and plasma metastin levels in women with PCOS and control groups.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (18) (Mean ± SD)</th>
<th>Patient (23) (Mean ± SD)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum AMH (ng/ml)</td>
<td>3.1±1.1</td>
<td>7.52±3.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Plasma metastin (pmol/L)</td>
<td>0.16±0.9</td>
<td>0.27±0.13</td>
<td>&lt;0.001</td>
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</tbody>
</table>

*: Student’s t-test.

Table (3): Correlation coefficient between serum levels of AMH and clinical, endocrinal, and ultrasound parameters.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (18) (Mean ± SD)</th>
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<tbody>
<tr>
<td><strong>Clinical parameters:</strong></td>
<td></td>
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</tr>
<tr>
<td>Age (years)</td>
<td>-0.22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>-0.34</td>
<td>&lt;0.001</td>
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<tr>
<td>W (cm)</td>
<td>-0.25</td>
<td>&lt;0.001</td>
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<tr>
<td><strong>Endocrine parameters:</strong></td>
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<tr>
<td>FSH (mIU/ml)</td>
<td>-0.21</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LH (mIU/ml)</td>
<td>0.48</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T (ng/ml)</td>
<td>0.45</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DHEA-S (μg/ml)</td>
<td>0.32</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Ultrasound parameters:</strong></td>
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<tr>
<td>No. of follicles (2-9mm)</td>
<td>0.57</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ovarian volume (cm³)</td>
<td>0.16</td>
<td>&lt;0.05</td>
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</tbody>
</table>

*: Spearman’s rank correlation.

Table (4): Correlation coefficient between plasma levels of metastin and clinical, endocrinal, and ultrasound parameters.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (18) (Mean ± SD)</th>
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<tbody>
<tr>
<td><strong>Clinical parameters:</strong></td>
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</tr>
<tr>
<td>Age (years)</td>
<td>-0.23</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>-0.31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>W (cm)</td>
<td>-0.26</td>
<td>&lt;0.001</td>
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<tr>
<td><strong>Endocrine parameters:</strong></td>
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</tr>
<tr>
<td>FSH (mIU/ml)</td>
<td>-0.23</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LH (mIU/ml)</td>
<td>0.43</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T (ng/ml)</td>
<td>0.38</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DHEA-S (μg/ml)</td>
<td>0.33</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Ultrasound parameters:</strong></td>
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<td></td>
</tr>
<tr>
<td>No. of follicles (2-9mm)</td>
<td>0.34</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ovarian volume (cm³)</td>
<td>0.19</td>
<td>&lt;0.05</td>
</tr>
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</table>

*: Spearman’s rank correlation.

Discussion

PCO is one of the most prevalent disorders in the women of reproductive age. The pathophysiology mechanisms leading to anovulation in PCO are under investigation [2].

The current study was conducted to evaluate serum AMH and plasma metastin level in PCO-related reproductive and metabolic disturbance.

In the present study, it is demonstrated that women with PCO have higher serum AMH, androgen and follicular number compared with normal healthy women. These data support the presence of different pathophysiological mechanisms of PCOS and suggest that additional factors interfere with maturation process in oocyte, a result consistent with that of Carlse, [19] and Park et al., [20]. Durlinger et al., [25] have found that in PCOS there is disturbance in the selection of the dominant follicle resulting in anovulation and accumulation of small antral follicles, which contributes significantly to the production of AMH.

Elevated serum AMH is considered to be linked with anovulation [22,23].

Furthermore, Pellatt et al., [24] have found that AMH production per granulose cell was raised in PCOS in comparison with controls, and also increased expression of AMH and its receptor by granulose cell from PCOS women [25]. In addition Pingy et al., [26] and Laven et al., [27] have reported that serum level of AMH, in women with PCOS are two to three fold higher than in ovulatory women with normal ovaries, an observation consistent with findings of present study.
In current study the PCO patients have serum levels of T and DHEAS that were significantly increased compared by normal healthy controls, a result agreed with those of La Marce et al., [7]. Franks; [28] reported that androgens tend to be higher in anovulatory women with PCOS than their ovulatory counterparts and androgen secretion is higher per theca cell in anovulatory women than ovulatory.

Edar-Geva et al., [29] observed that AMH values were significantly increased in hyperandrogenic PCOS in comparison with normoandrogenic and controls.

Jonard and DeWailly [21] postulated that hyperandrogenemia leads to follicle excess, increasing the intra-ovarian AMH level, which then could exert an inhibiting effect on the selection process, supported by observation that levels were significantly independently associated with circulating AMH. This observation is in support with the finding of this study regarding the statistically significant increased number of follicles, AMH androgen level in PCOS patient compared with normal women. Therefore, they postulated that AMH levels reflect a dynamic interaction between LH secretion and influence, androgens, and endogenous disorders in follicular maturation. Additionally, Grossman et al., [30] suggested that AMH inhibit the aromatase activity leading in turn to increased local androgen production and concentration.

The present study showed that LH serum levels were significantly increased in PCOS patient compared with normal healthy controls. These result agreed with those of Piouka et al., [31]. Willis et al., [32] and Pellatt et al., [24] who postulated that increased serum LH level in PCOS women has a positive effect on granulose cell production of AMH leading to increase serum level. Also they observed higher LH levels in women with sever PCOS who also had the highest AMH concentrations.

The present study demonstrated that the total number of follicles 2-9mm on ultrasonography as well as the mean ovarian volume were significantly increased in women with PCO compared with control subjects, a result consistent with those of Laven et al., [27] and Piouka et al., [31].

The current study showed no statistically significant difference in serum FSH levels between both groups, FSH levels were borderline lower in women with PCOS compared with normal healthy controls. Lower FSH serum levels in PCOS women denuded that AMH is one of the local inhibitors of FSH action in inhibiting granulose cell proliferation. AMH may also exert its action by decreasing granulose cell sensitivity to FSH [10]; Josso et al., [33]; Wachs et al., [17] and Chen et al., [9].

IN the current study, AMH serum levels were negatively correlated with age, BMI, w and FSH in accordance with Kandarakis et al., [2]. Whereas, it is positively correlated with LH, T, DHEAS, total number of small follicles, and mean ovarian volume, so AMH levels correlate with the extent of ovarian dysfunction in PCOS women as documented by Laven et al., [27].

The present study demonstrated that there no statistically significant different in mean age, Body Mass Index (BMI) and Waist circumference (W) in healthy controls and PCOS patient a result consistent with that of Kandarakis et al., [2] and Piouka et al., [31]. This implies that these factors do not account for the observed differences between the two groups.

In the present study, it is of interest that PCOS have statistically significant higher serum levels of metasin compared with normal healthy controls, a result consistent with that of Chen et al., [15]. Also in the current study there was a significant positive correlation between the serum levels of AMH and plasma metasin both in the control and in the PCOS patient group which suggests an interaction of these two molecules. Further straining of a possible link of these two molecules in the anovulatory process of PCOS, comes from the observation of higher expression and production of AMH and its receptor per granulose cell in adolescent PCOS. Piouka et al., [31], with higher expression of metastin in adolescent PCOS women than that of normal adolescent women, which suggest that the increased level of metastin might be associated with pathogenesis of adolescent women with PCOS.

The mechanisms of interaction of AMH and metastin in the oocyte maturation process in PCOS cannot be elucidated in this study. However, the presented data are in support of the role of Insulin Resistant (IR) and hyperinsuleniemia in the pathophysiology of anovulation in PCOS women either through the pathway of hyper androgenemia, by stimulating the development of antral follicles increase sensitivity of granulose cell to FSH, and thus increase the number of follicles and ovarian volume, or through the dysregulation of AMH production from granulose cell, Fulghesu et al., [34] and Chen et al., [9]. Furthermore, Panidis et
plasma metastin and anovulatory markers in the pathogenesis of patients with PCOS. Al-though the mechanism by which it interfere with oocyte maturation has not been clarified yet.

In conclusion, both serum AMH and plasma metastin can be used in the diagnosis and follow-up of patient with PCOS. In addition, the correlation between the two markers implies an interrelated role in the pathogenesis of PCOS. Clearly further studies are required to explore the mechanism of this potentially interactive relationship between plasma metastin and anovulatory markers in the pathogenesis of patients with PCOS.

References


تعتبر متلازمة تعدد الاكياكس المبيضية واحدة من اهم انواع الخلل الصمائيالذي يصيب السيدات في سن الالتباس، وهذا كاعدد من الدراسات التي تهدف لاستقصاء الآليات المختلفة للياب عملية الإصابة المصاحبة لهذه المتلازمة.

الفروض من البحث: في هذه الدراسة تم تقسيم نسبة كل من الهورمون المضاد لقناة مولر والمييستيتيين في مرضا متلازمة تعدد الاكياكس المبيضية ومقارنتها بالسيدات الأصحاء اللاتي يعدين المجموعة الضبطة ودراسة علاقتها بالاضطرابات الأيضية والتناسلية المصاحبة لهذه المتلازمة.

الموضوعي وطريق البحث: أجريت هذه الدراسة على ثلاث وعشرين سيدة من السيدات المصابة بإضطراب متلازمة تعدد الاكياكس المبيضية بالمقارنة مع ثماني عشرة سيدة من الأصحاء اللاتي يشككن المجموعة الضبطة. وتم اختيار كل من المجموعتين في أعمار متشابهة. وقد تم عمل الأتي لكل من المرضى والأصحاء (خذ التأريخ المرضي - فحص إيجابي شامل - فحص الأوراق الفوق صبئية على الباب - قياس نسبة كل من الهورمون المضاد لقناه مولر والمييستيتيين).

النتائج: أظهرت نتائج هذه الدراسة وجود زيادة وتوافق إيجابي ذات دلالة إحصائية في نسبة كل من الهورمون المضاد لقناة مولر والمييستيتيين في مجموعة المرضى عن المجموعة الضبطة. كشفت الدراسة أيضا وجود زيادة ذات دلالة إحصائية في قيم كل الهورمون اللوليوبول، الهورمون المنبه للجربي ومورون الاستروستيرويل. وفي كل جاءت الفحوصات في المريضة الضبطة وحجم المييستيتيين في مجموعة المرضى عن المجموعة الضبطة. وكذلك وجد تواجد إيجابي تو دلالة إحصائية بين كل هذه الهرمونات ونسبة كل من الهورمون المضاد لقناة مولر والمييستيتيين، وجود تواجد سلبي تو دلالة إحصائية بين كل من العمر، ونسب كتلة الجسم وحجم الخصر ونسبة كل من الهورمون المضاد لقناة مولر والمييستيتيين.

الخلاصة: يمكن الاستنتاج بأن الهورمون المضاد لقناة مولر والمييستيتيين ذات أهمية في تشخيص ومتابعة حالات متلازمة تعدد الاكياكس المبيضية. وأن وجود تواجد إيجابي بينهم يدل على أن لها دور في آلية حدوث هذه المتلازمة وهو ما يتطلب العديد من الدراسات لإثباته.