The Relation between hormonal changes and Polycystic Ovary Syndrome (PCOS)

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Abstract:
Polycystic ovary syndrome (PCOS), whose genetic basis is not completely well understood, is the most common endocrine disorder in women in their reproductive years. Blood samples were collected from two groups. The first group included (65) females with PCOS and the second group included (30) normal females. Blood samples collected from the Babylon Maternity and Pediatrics Teaching Hospital. The age of patients and healthy ranged from (18 to 45) years. Hormonal study of Estradiol (E2), Luteinizing hormone (LH), Follicle stimulating hormone (FSH), Testosterone (T) and Prolactin were done for each patient. The results were as follows: There were significant (P<0.05) lower in E2 and FSH levels in PCOS women, there were no significant (P>0.05) in LH and Prolactin levels in PCOS women, there were significant differences (P<0.05) in Testosterone levels when compared with healthy women.

Key words: PCOS, Luteinizing hormone, Follicle stimulating hormone, E2

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Introduction:
Polycystic ovary syndrome (PCOS) is a heterogeneous disorder characterized by hyperandrogenism and chronic anovulation. Depending on diagnostic criteria, 6% to 20% of reproductive aged women are affected. Symptoms of PCOS arise during the early pubertal years. Both normal female pubertal development and PCOS are characterized by irregular menstrual cycles, anovulation, and acne. Owing to the complicated interwoven pathophysiology, discerning the inciting causes is challenging. Most available clinical data communicate findings and outcomes in adult women[1]. Whereas the Rotterdam criteria are accepted for adult women, different diagnostic criteria for PCOS in adolescent girls have been delineated. Diagnostic features for adolescent girls are menstrual irregularity, clinical hyperandrogenism, and/or hyperandrogenemia[2]. Pelvic ultrasound findings are not needed for the diagnosis of PCOS in adolescent girls. Even before a definitive diagnosis of PCOS, adolescents with clinical signs of androgen excess and oligomenorrhea / amenorrhea, features of PCOS, can be regarded as being “at risk for PCOS.” Management of both those at risk for PCOS and those with a confirmed PCOS diagnosis includes education, healthy lifestyle interventions, and therapeutic interventions targeting their symptoms. Interventions can include metformin, combined oral contraceptive pills, spironolactone, and local treatments for hirsutism and acne. In addition to ascertaining for associated comorbidities, management should also include regular follow-up visits and planned transition to adult care providers. Comprehensive knowledge regarding the pathogenesis of PCOS will enable earlier identification of girls with a high propensity to develop PCOS. Timely implementation of individualized therapeutic interventions will improve the overall management of PCOS during adolescence, prevent associated comorbidities, and improve quality of life[3].

Materials and Methods:
Subjects:
A case-control study was carried out through period between “from July 2019 till January 2020” A total of (65) women aged between (18-45) years were attending from Babylon Maternity a Pediatrics Teaching Hospital. Two study groups have been investigated. Thirty-five out of (65) women were patients diagnosed by their as polycystic ovary syndrome compared with thirty apparently healthy women as control. Patients and control were with analogous age.

Patients group (PCOS group):
This study has included fifty infertile Iraqi women with PCOS. Patients were selected from Babylon Maternity and Pediatrics Teaching Hospital.

**Healthy control group (fertile):**
The healthy control group consists of twenty-five healthy fertile women of different ages (16-45 years). Venous blood samples (5 ml) have been collected from each woman of both PCOS and healthy control.

**Excluded criteria:**
All disorders that can result in menstrual irregularity and hyperandrogenism, including adrenal or ovarian tumors, thyroid dysfunction, congenital adrenal hyperplasia, pituitary diseases, acromegaly, Cushing syndrome, and other causes of infertility besides PCOS.

**Hormonal assay:**
About (5) ml of venous blood was drawn from the subjects and was allowed to clot in plain tubes at room temperature. The serum was obtained by centrifugation of the blood at (5000 rpm for 5 min.), then froze until the hormonal VIDAS, which was performed by using the Mini VIDAS apparatus (Biomerieux company, France, through an enzyme-linked fluorescent assay (ELFA) technique.

**Ethical approval:**
The necessary ethical approval from the ethical committee of the hospital and patients and their followers must be obtained. Moreover, all subjects involved in this work are informed and the agreement required for doing the experiments and publication of this work is obtained for each one prior to the collection of samples. For every female or her followers, the procedure had been informed before samples were collected, making absolutely sure that they understood the procedure that is to be carried out.

**Statistical Analysis**
The Statistical Analysis System-SAS was used to effect of different factors in study parameters. A chi-square test was used to significant compare between percentage and the Least Significant Difference (LSD) test was used to significantly compare between means in this study.

**Results and Discussion**
The comparison of hormonal assay between patients and control was illustrated in Table (1), which shows the non-significant difference of LH and Prolactin while the statistically significant difference was FSH level with (P-value = 0.04), E2 (P-value = 0.001) and Testosterone (P-value = 0.001). The lack of difference in LH and Prolactin could be attributed to the prior medical treatment taken by patients who participated in the study. The results were agreed with Holt et al., [4] who suggested that FSH levels were lower in young women with PCOS than in the early follicular phase of women with normal ovaries. The mechanism behind these low levels, which may partly explain the lack of follicular growth, is probably increased production of inhibiting B from the increased number of natural follicles in polycystic ovaries [5].

Current results were in agreement with the finding by Begawy et al., [6]. The authors explained the reduction level of FSH to the High levels of (inhibin) has been found in the PCOS women and this leads to FSH, reduced [7]. Overexpression of Follistatin leads to an increase in ovarian androgen production [8]. Moreover in PCOS the Estrone level increase due to the conversion of androstenedione in adipose tissue which additionally stimulates LH and inhibits FSH [9]. Nawras, [10], showed that no significant differences (P>0.05) in the level of FSH. Also, the result was different than Mezaal, [11], who reported the same findings in the Iraqi population.

Results obtained revealed that the E2 hormone was significantly lower (p<0.05) in PCOS women (33.88 ± 2.39) pg/ml compared with a healthy control group (53.09 ± 7.02) pg/ml (Table 1). These results agreed with Chang and Katz, [12] who showed the E2 hormone level in PCOS patients may be low to normal. The increase in serum Anti Mullerian hormone (AMH) level in PCOS women results from increased production of this hormone per follicle, this amount led to inhibits aromatase activity, therefore, the follicle doesn’t produce a sufficient amount of E2 hormone [13]. Data in Table (1) showed that the testosterone in PCOS women is higher (1.33 ± 0.28) ng/ml than the healthy control group (0.513 ± 0.07) ng/ml but non-significant, patients with PCOS showed the elevated in testosterone levels than the normal range (0.1-0.9) ng/ml. These results are in agreement with the study of Carmina et al., [14].
There are additional causes of hyperandrogenism \cite{15}. Increased synthesis of testosterone precursors due to a dysregulation of theca cell androgen production. Hyperinsulinemia, which has been proposed as the primary event leading to hyperandrogenism. Increased serine phosphorylation of the insulin receptor, resulting in activation of both ovarian and adrenal P450c17a enzymes and promoting androgen synthesis. Genomic variants in genes related to the regulation of androgen biosynthesis, function, and the availability of androgens to target tissues, insulin resistance, and metabolic syndrome.

**Table (1):** Comparison between patients and control in hormone profile.

<table>
<thead>
<tr>
<th>Group</th>
<th>NO.</th>
<th>Mean ± SD FSH(IU/ml)</th>
<th>Mean ± SD E2(pg/ml)</th>
<th>Mean ± SD LH(IU/ml)</th>
<th>Mean ± SD Prolactin (ng/ml)</th>
<th>Mean ± SD Testosterone (ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>35</td>
<td>6.73 ± 0.23</td>
<td>33.8±2.39</td>
<td>3.48 ± 0.33</td>
<td>17.05 ± 1.21</td>
<td>1.33 ± 0.28</td>
</tr>
<tr>
<td>Control</td>
<td>30</td>
<td>8.01 ± 0.66</td>
<td>53.09±7.02</td>
<td>3.29 ± 0.59</td>
<td>18.58 ± 2.35</td>
<td>0.513 ± 0.07</td>
</tr>
<tr>
<td>P-value</td>
<td>65</td>
<td>0.04</td>
<td>0.001</td>
<td>0.18</td>
<td>0.27</td>
<td>0.001</td>
</tr>
</tbody>
</table>

\* (P<0.05), NS: Non-significant.

In conclusion, PCOS can be considered as a complex and heterogeneous metabolic syndrome manifestation of different phenotypes. The patients showed a decrease in levels of E2 and FSH that lead to inhibition of ovulatory and infertility. Also, the study showed significantly elevated testosterone levels in all patients.

**References:**


