Gonadotropin-Releasing and Antimullerian Hormones Levels and Other Hematological Parameters among Females with Polycystic Ovaries at Al-Najaf City

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Summary

Objectives: To evaluate the hormonal parameters efficacy as markers for polycystic Ovaries in women.

Methodology: This is a case-control study containing fourteen samples of female polycystic ovarian patients. According to age, it is separated into four groups. Their ages ranged from 15 to 55 years old. Between October 2021 and February 2022, samples were obtained from patients at Al-Sadder Hospital center / Najaf Ashraf. A total of fourteen volunteers were used as a control group in this investigation. The study is explained to both the patients and controls group.

Results: The age difference between the cases and the controls was not significant. Residence, or education. Between women with PCOS and controls, none of the hematological markers showed any significant changes. (e.g., HGB, RBC, WBC, and platelet). The mean LH, PRL, FSH and AMH, DHEA was significantly higher in those with polycystic ovarian syndrome Patients in Comparison with Control ($P < .001$).

Conclusion: The study concluded that AMH and DHEA hormones play vital roles in polycystic Ovaries, An overabundance of androgens damages the endocrine and immunological systems, as well as causing metabolic changes.

Keywords: Gonadotropin-Releasing Hormone, Antimullerian Hormone, Polycystic ovary.

1. Introduction

Hyper-androgenism (is indication of an excess male hormones or androgen influences; for instance, practically, for instance hirsutism, and/or chemically, such as hyperandrogen aemia or high androgen levels.), ovulatory problems (as well as menstrual problems), and polycystic ovarian morphology) are all symptoms of polycystic ovary syndrome (PCOS) (PCOM; ovaries with an abnormally high number of prenatal follicles). Clinical manifestations vary and can be divided into a There are a number of phenotypes based on the presence or absence of specific features [1].

The old PCOS diagnostic criteria were not widely recognized; in fact, there were significant differences in opinion, finding it tough to compare and comprehend results from various centers [2]. Until 1990, Polycystic ovarian syndrome was thought to affect women who had regular periods and clinical or biochemical hyperandrogenism, as well as individuals who had fixed cycles and PCOS. The previously accepted diagnostic criteria for PCOS were established by the 1990 NIH/NICHHD consensus conference for PCOS diagnosis. Oligo-menorrhea or amenorrhea, as well as medical or biochemical indications of hyper-androgenemia, were used to define this condition. As a result, in nations that used this categorization, PCOS was diagnosed in the lack of an ultrasound appearance of PCOS [3].

On the other hand, PCOS as it is defined in Europe focuses on the presence of PCOS on ultrasonography in combination with one or both of the next features Obesity, oligo-/amenorrhea, or amenorrhea, and a higher luteinizing hormone (LH) level are all symptoms of clinical and/or bio-chemical hyperandrogenism (acne, hirsutism, or alopecia). Despite the fact that polycystic ovaries frequently coexisted with many symptoms, signs, or biochemical disturbances, and According to this description, PCOS is a dynamic condition in which an individual's traits might vary over time., The discovery of a polycystic ovary on ultrasound, as well as obesity, was enough to confirm the diagnosis., Despite the fact that it was understood that policy generally coexisted with more than one symptom, indication, or physiological problem. The existence of 12 or even more follicle with a diameter of 2–9 mm and/or an ovarian volume more than 10 cm are currently accepted ultrasonography criteria for polycystic ovaries.

The much more likely cause of PCOS is, which is prevalent in 90%

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of patients, is functionally ovary hyper-androgenism, that is triggered by dysregulation of stero-idogenesis, which makes ovarian androgen production more sensitive to LH [4]. Insulin resistances (IR) and compensating hyperinsulinemia are important in the pathophysiology of the pituitaryovarian axis [5]. As the rate of Gn-RH pulses increases and shorter pulses stimulates LH, resulting in a drop in FSH, The pathogenesis of PCOS appears to be influenced by neuroendocrine abnormalities [6]. In a mouse model of PCOS, Caldwell et al. [7] revealed that androgen receptor signing was a prominent extra-ovarian mediator in the development of PCOS symptoms. In PCOS, the pulsatility of GnRH and LH is enhanced. Appears to stem as the "cumulative effect of altered GnRH stimulatory and inhibitory neurotransmitters in the hypothalamic-pituitary region," according to Chaudhari et al. [6]. The brain is implicated in the development and pathogenesis of PCOS, according to new research. Obesity, which has become a global epidemic, Insulin resistance is caused by a variety of factors, the most prevalent of which is diabetes [8]. Encourages the diagnosis and detection of PCOS. Obesity promotes insulin resistance and suppresses gonadotropin production while increasing testosterone production from circulating androstenedione. Anovulation of PCOS is linked to obesity-related compensatory hyperinsulinemia as a reaction to insulin resistance [4]. In PCOS, sufficient weight reduction increases ovulation by lowering insulin sensitivity [9]. Inflammation is triggered by abdominal adipocyte hypertrophy, Hyperandrogenism exacerbates this problem in PCOS [10]. stimulating adipose tissue mononuclear cells to produce proinflammatory cytokines in response to glucose and saturated fat consumption [11]. Obesity has been associated to a reduction in ovulation and an increase in LH levels [12]. Obesity can explain for increased peripheral testosterone formulation in the absence of PCOS [13]. PCOS increases the risk of cardiovascular diseases (CVD), which is connected to insulin resistances, T2DM, and metabolic disorders in both obese and non-obese women. In a Danish registration system study discovered a total Cardiovascular event rate of 22.6 per 1000 patient years in PCOS vs. 13.2 per 1000 patients yrs in controls (P 0.001) [14].

2. Methodology

This is a control study containing fourteen sample of female polycystic ovarian patients. According to age, it is separated into four categories. Their ages ranged from 15 to 55 yrs old. Between October 2021 and February 2022, patient samples were obtained at Al-Sadder Hospital Center / AL-Najaf AL-Ashraf. A total of fourteen subjects were used as a comparison group in this investigation. The study is explained to both the participants and the control subjects. Serum AMH, LH, FSH, DHEAS and Prolactin levels were measured as part of the biochemical tests. Specialist physicians made diagnoses based on a few unambiguous clinical characteristics, which were then verified by laboratory testing. In this investigation, the cobas e 411 was employed, which is a completely automated analyzer that employs a proprietary ElectroChemiLuminescence (ECL) technology for immunoassay analysis.

3. Statistically Analysis

For the statistical analysis, SPSS version 18.0 was employed, and the one-way ANOVA test was used for serum CA125 and CA15-3, while the chi-square test was used for immunohistochemical alterations. Statistically significances was defined as a P-values with less than 0.05.

4. Results and Discussion

The females mean lifespan was 28.83.9 years. There was no notable age difference between the patients and the controls. Residence, or education. Those with PCOS had a significantly greater BMI than women without the condition. (P0.001, Table 1). Between females with PCOS and controls, there were no significant changes in any of the hematological indicators. (e.g., RBCs, HGB, platelets and WBCs) (Table 2).

Table (1): Assessment of the sociodemographic features of females with PCOS with control.

Variable	PCOS (N = 40)	Controls (N=40)	P value
Age,years	28.7 ± 3.9	31.2± 4.8	0.54
BMI, kg/m2	30.5±7.8	27.8±5.8	0.378
Rural residence, n (%)	16 (40)	12 (20.0)	0.388
Education level secondary level, n (%)	18 (45)	30 (75)	0.500
Housewives, n (%)	22 (55)	10 (25)	0.711

Table (2): Hematological parameters in PCOS patients and controls.

Variables	Polycystic ovarian syndrome (n = 40)	Controls (n =40)	P value
WBC x.10/L	6400(5300–7892)	6375(5312–7475)	0.987
Neutrophils x.109/L	2645(1907–4165)	3100(1977–4110)	0.671
Lymphocyte	2770(2290–3107)	2425(1932–2205)	0.333
Monocytes x.109/L	470(367–652)	455(375–600)	0.532
Eosinophils x.109/L	145(70–222)	100(70–1850)	0.186
Basophils x.109/L	20(10–30)	20(10–30)	0.989
RBC x.106/mm3	4800(4555–5155)	4730(4527–5010)	0.411
Hemoglobin g/.dl	11.3 (11.18–15.7)	12.7(11.18–15.7)	0.858
Hematocrit,%	38.20(36.45–39.93)	38.15(36.10–40.58)	0.669
MCV., fL	80,65(73,100–84,650)	80,550(74,450–85,850)	0.467
MCH., pg	26,30(23,375–28,125)	26,500(24,375–27,850)	0.971
MCHC., g/L	328(318–333)	324(313–330)	0.184
RBC distribution width. %	13,500(12,850–15,20)	13,650(12,775–14,825)	0.758
Plateletcount,103/mL	312(2678–368)	324(291–371)	0.423
Meanplatelet volumes.,fL	10,700(9900–11,400)	10,500(9800–11,200)	0.351
Platelet-distribution width., %	15.4(15.1–15.7)	15.4(15.1–15.7)	0.861

In the current study, there were no important differences in hematologic parameters among women with PCOS and controls. This finding agrees with Ucakturk et al. [15], who reported no major differences in HGB, RBC, platelets count, or WBC between PCOS and non-PCOS females. Between PCOS patients and controls, there was no statistical difference in blood iron levels. Hematological indicators such HGB, WBC count, MPV, RDW, basophil counts, and PCOS, have all yielded various

outcomes in previous research. As a result, more study into blood parameters and PCOS is required [16].

In comparison to controls, the mean LH/FSH was considerably greater in patients with polycystic ovarian syndrome. (P =.0001). In this investigation, the mean testosterone level in the patients was considerably greater than in the controls groups, which is in line with earlier studies. LH causes the adrenal gland to make androstenedione, a weaker androgen, which leads to higher testosterone levels. Increased LH levels imply that the hypophyseal-adrenal axis has a role in POS development. For establishing hyperandrogenemia, the American Association of Clinical Endocrinologists (AACE) recommends measuring free testosterone using equilibrium dialysis. PCOS is thought to be the result of a variety of pathophysiological processes, including Hyper-insulinemia, ovarian anomalies, and elevated LH levels, all of which contribute to increased ovarian androgen output [17]. LH levels in patients are substantially greater than in controls, which is consistent with previous findings.\ despite the fact that the amount of LH is a significant criterion in PCOS, it is not included in guidelines since it changes with the stages of the menstrual cycle.

In females with PCOS, a high level of LH is a difficult trait that occurs in nearly half of the patients. The optimal period to determine LH level is between 2 weeks after the start of the menstrual cycle and 3 weeks before the next cycle, when LH is minimally repressed. This stage is known as the "specific oligomenorrhoeic phase" (SOP), and it occurs exclusively after a duration of 35 days v

Table (3): luteinizing hormone (LH), follicles stimulating hormones (FSH) and testosterones in polycystic ovarian syndrome patients according to gender in comparison with control.

Parameters	Age group	Group		P-value
		Patient N=40 Mean.±SD	Control. N=40 Mean.±SD	
Luteinizing Hormone (LH)	20-30 ys	5.83±0.60	4.85±0.41	NS
	31-40 ys	4.86 ±0.52	4.85±0.41	
	41-50 ys	5.23±0.61	4.85±0.41	
	51-60 ys	4.72±0.54	4.85±0.41	
Follicle - Stimulating Hormone (FSH)	20-30 ys	5.65±0.84	4.23±0.49	NS
	31-40 ys	5.21±0.56	4.23±0.49	
	41-50 ys	6.12±0.63	4.23±0.49	
	51-60 ys	5.58±0.64	4.23±0.49	
Total Testosterone	20-30 ys	3.4 ± 0.83	3.25±0.69	NS
	31-40 ys	3.12±0.84	3.25±0.69	
	41-50 ys	2.94±0.74	3.25±0.69	
	51-60 ys	4.14±0.59	3.25±0.69	

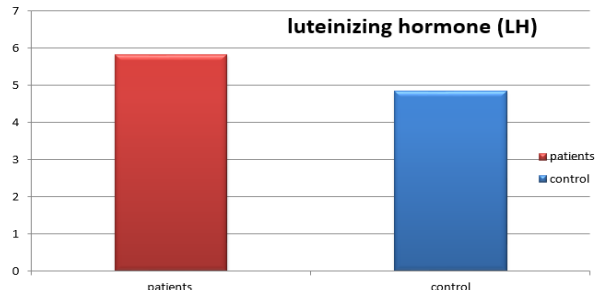

Figure (1): The total concentration of LH among patient and control.

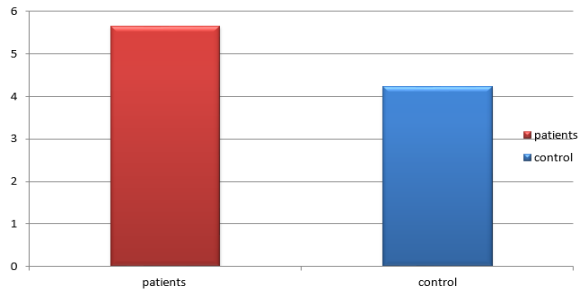

Figure (2): The total concentration of follicle - stimulating hormone (FSH) among patient and control.

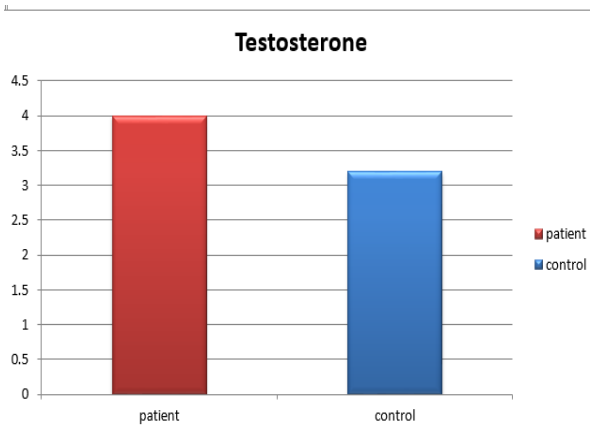

Figure (3): The total concentration Total testosterone among patient and control.

Between PCOS patients (3.74 0.61 ng/ml) and the control group P0.05 (1.740.39) ng/ml, AMH levels revealed a significant statistical rise. As shown in table, the mean concentration of AMH among patients of different ages was not statistically different (3) [18]. The clinical significance of AMH has been studied, and it has been proven AMH levels in women with PCOS are two to three

times greater than in individuals with normal ovarian function Cook et al. [19] found that ovulatory patients had considerably in their study, those with greater amounts of AMH than controls had higher levels of AMH. When compared to controls, women with PCOS have higher levels of AMH concentration, regardless of whether they are lean or obese, as shown in various studies including samples from a population of thin and overweight women. Women with polycystic ovarian syndrome have slightly higher DHEAS levels in their blood (PCOS). DHEAS levels are high in around 20% to 30% of women with PCOS.

Table (4): antimullerian(AMH) and dehydroepiandrosterone (DHEA) in polycystic ovarian syndrome patients according to gender in comparison with control.

Parameters	Age group	Group		P.values
		Patient N=40 Mean.±SD	Control N=40 Mean.±SD	
Antimullerian(AMH) ng/ml	20-30 ys	3.74±0.61	1.74±0.39	<0.05
	31-40 ys	3.52 ± 0.64	1.74±0.39	
	41-50 ys	2.94±0.56	1.74±0.39	
	51-60 ys	3.12±0.62	1.74±0.39	
Dehydroepiandrosterone (DHEA) mcg/dL	20-30 ys	124.5 ± 23.8	57.23 ± 5.48	
	31.-40 ys	172.33 ± 21	64.2 ± 4.12	
	41.-50 ys	122.4 ± 18.3	82.36 ± 12.4	
	51.-60 ys	156.7 ± 14.9	71.8 ± 8.7	

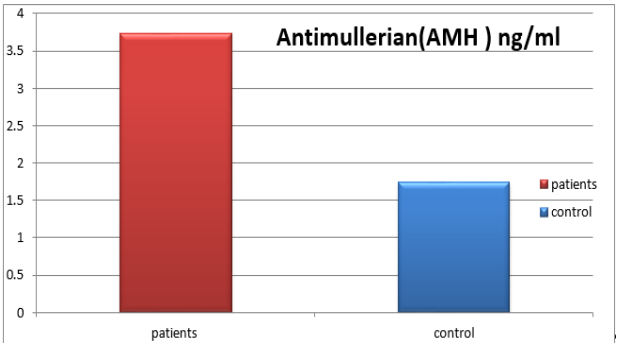

Figure (4): The total concentration antimullerian(AMH) among patient and control.

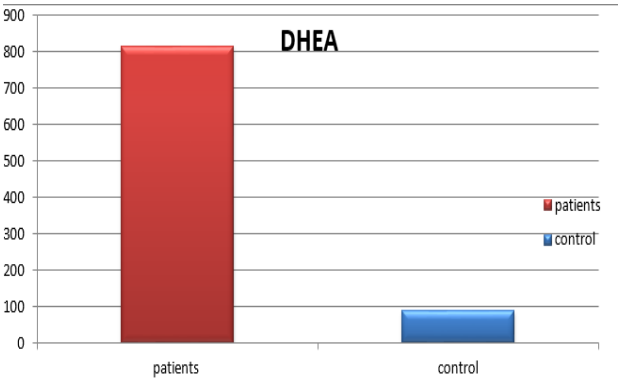

Figure (5): The total concentration dehydroepiandrosterone (DHEA) among patient and control.

5. Conclusion

We concluded that AMH and DHEA hormones play an important role in polycystic ovaries, where excess androgens impair the endocrine and immune system, leadind to metabolic changes.

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