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EPIDEMIOLOGY OF POLYCYSTIC OVARY SYNDROME IN IRAQI WOMEN

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS), the major endocrineopathy among reproductive-aged women, is not yet perceived as an important health problem in the world. It affects 4 to 21% of women of reproductive age worldwide. The prevalence, diagnosis, etiology, management, clinical practices, psychological issues, and prevention are some of the most confusing aspects associated with PCOS. Aim: investigated the hormonal changes in PCOS female and determination of some factor associated with increased PCOS development. **Materials and Methods:** 250 Iraqi women, their ages were from 20 to 50 years, they were divided into two groups: study group (n=125, Polycystic Ovary Syndrome PCOS) while the other group (n=125 control group mainly normal women), ultrasound were taken in the second menstruate cycle day for each women. **Results:** the mean age of patients was 26.65 ± 10.21, PCSO patients show a statistical higher weight, BMI and waist circumference in compare to control (p-value 0.01, 0.001 and 0.006). Regarding hormonal disturbance, testosterone, progesterone and AMH mean level were significantly higher in patients in compare to control. Family history of DM and obesity is associated with PCOS development. **Conclusion:** The present study summarizes and interprets the results of all pathological samples for the syndrome and highlights the burden of the syndrome, thus supporting the early identification and prevention of PCOS in order to reverse the continuing upward trend of prevalence by ultrasound, clinically, immunological and molecular testing for early identification of the syndrome.

KEYWORD: Polycystic ovary syndrome, PCOS, BMI, metabolic syndrome.

INTRODUCTION

PCOS is a prevalent endocrine condition defined by insulin resistance, an increase in ovarian generated androgen, and biochemically by hyperandrogenism and hyperinsulinemia, all of which lead to poor follicular development and oocyte maturation.[1] PCOS increase morbidity in patients due to increases the risk of metabolic syndrome, type 2 diabetes, cardiovascular disease, endometrial cancer, and obesity.[2]

PCOS affects women of all races and ethnicities who are of reproductive age, and the incidence varies depending on diagnostic criteria. It affects 4 to 21% of females of reproductive age.[3] Primary infertility, early pregnancy loss, and later pregnancy problems were all linked to this disorder. According to a new economic research published in the Journal of Clinical Endocrinology & Metabolism of the Endocrine Society, the cost of diagnosing and treating PCOS is expected to reach $8 billion by 2020.[4]

Pathogenesis of PCOS

The syndrome gain its name from presence of numerous ovarian cysts appeared in ultrasound, those cysts are the immature follicles (developed from primordial follicles) and ceased at the antral stage due to the ineffective ovarian function. The condition involve an interaction defect in hypothalamic–pituitary axis, insulin production, action as well as ovarian dysfunction. Changes in luteinizing hormone (LH) activity, insulin resistance, and a probable propensity to hyperandrogenism have been related to the pathophysiology of PCOS, the fundamental pathophysiologic abnormality in PCOS remains unexplained.[5]

Weight-gain, obesity and central obesity promote insulin resistance by adipokine and fatty acid release and

contribution to low grade chronic inflammation. Losing weight in patients can be effective in improving insulin sensitivity and serum insulin levels.[6]

Etiology

The disease was caused by a complex combination of genetic and environmental variables, which resulted in a diverse, clinical, and biochemical phenotype. The genetic cause of PCOS is unknown, despite the fact that it is common to have a family history of the disease. While the specific gene responsible for the condition's development has not been identified, and the familial links to PCOS are unclear, studies suggest that several genes are involved.[7]

Evidence suggests that having a family history of T2D, increases the chance of the disease progressing in PCOS women. T2D and obesity-related genes and genetic polymorphisms were linked to hyperandrogenism, which has been associated with the PCOS phenotype.[8]

The pathophysiology of IR in PCOS is known to be multifactorial, family histories with IR and obesity appeared to be extremely common in affected women. In addition to the key role played by insulin resistance in the pathophysiology of PCOS.[9] about half of PCOS patients have obesity, primarily central obesity. A higher quantity of central abdominal is associated with insulin resistance. Obesity prevalence in the overall population, varies by age, ethnicity, and geographic region. The chances of IR, obesity, and T2D are increased in first-degree relatives of PCOS-affected women who are both males and women with unknown mode of inheritance.[10]

Alcohol intake, smoking, diets high in fat and poor in fiber, and sedentary lifestyles can all contribute to the development of PCOS. PCOS can be avoided with the support of healthy behaviors including an active way of life and a sufficient diet free of hazardous ingredients. Due to the accumulation of ovarian follicles at different stages of atresia, PCOS women frequently develop polycystic ovaries.

Clinical presentation and diagnosis

PCOS is a hormonal disorder with a wide range of signs and symptoms. However, ovulation irregularities, high androgen levels, and cystic ovaries are three common factors found in PCOS patients. The majority of PCOS patients have menstrual and ovulatory issues, which might include oligomenorrhea, secondary amenorrhea, or hypermenorrhea in some cases.[11]

The diagnosis involve taking a full history and clinical examination as well as laboratory test. The National Institutes of Health (NIH) criteria were established in 1990 in attempt to produce a comprehensive and detailed description for the diagnosis of PCOS.[12]

Rotterdam formulated a new diagnostic criterion named Rotterdam criteria that requires the presence of two conditions out of the three: (1) oligomenorrhea /anovulation, (2) clinical/biochemical hyperandrogenism, and (3) polycystic ovaries (≥12 follicles in each ovary measuring 2–9 mm). This criteria had no clinical or laboratory evidence of hyperandrogenism, and the diagnosis of PCOS is made in women who have ovulatory failure and the appearance of polycystic ovaries on ultrasound.

Hormonal and laboratory investigation is needed, starting with fasting glucose measurement and insulin in case of insulin resistance, Assessment of serum LH and FSH levels. LH/FSH ratio (2:1) is of use in the diagnosis of PCOS, Testosterone, dihydroepiandrosterone sulfate (DHEAS) levels, progesterone, prolactin, anti-mullerian hormone levels (AMH) and Thyroid function test.

MATERIALS AND METHODS

Study design and data setting

The study is a case control study included 250 women using random sample collection of data, the sample is divided into two groups

1- Women who are diagnosed with PCSO.
2- Normal women as control group.

The age of included participant was 20-50 years, the data collected during 3 months from 1st of January 2021 to 1st of April 2021, from patients who visit Baghdad teaching hospital out-patient clinic for follow up or for other purpose.

The anthropometric data

The weight and height were also taken for all participant using a measuring tab for waist circumference of weight measurement and an electronic scale.

Calculation of Body Mass Index (BMI)

Body mass index (BMI) is defined as the weight in kilograms divided by the square of the height:

BMI = weight (kg)/height (m^2)

BMI was categorized into the following

1- BMI less than 24.9 (normal weight).
2- Ranging from 25 to 29.9 (overweight).
3- More than 30 (obesity).

Laboratory evaluation

5ml of venous blood were taken in the second menstruate cycle day for each participant that used for chemical evaluation of fasting blood sugar, Follicular stimulating hormone, lutilizing hormone, progestrone, testosterone, anti-mullerian hormone levels (AMH) using a commercial kit.

RESULTS

The study included 125 previously diagnosed PCOS patients and 125 healthy control, no statistical significant differences in mean of age between the two groups, p-value 0.3, however, PCOS patients had a statistically higher mean of weight, waist circumference and BMI

(73.06 ± 15.80 kg, 100.76 ± 10.56 cm, 28.14 ± 6.51kg/m^2) in compare to control (68.03 ± 15.90 kg, 84.19 ± 5.9 cm, 25.93 ± 6.10 kg/m^2), p-value 0.01, 0.001 and 0.006, as shown in table 3-1.

Table 3-1: participant basic data.

Variables	Group	N.	Mean ± SD	
Age	Patients	125	26.65 ± 10.21	0.06
	Controls	125	24.31 ± 7.54	
Weight	Patients	125	83.06 ± 15.80	0.01*
	Controls	125	68.03 ± 15.90	
Waist circumference	Patients	125	100.76 ± 10.56	0.001*
	Controls	125	84.19 ± 5.9	
BMI	Patients	125	28.14 ± 6.51	0.006*
	Controls	125	25.93 ± 6.10	

*p-value ≤ 0.05

The fasting blood sugar, follicular stimulating hormone, luteinizing hormone, progesterone, testosterone, anti-mullerian hormone were assessed, result revealed that patients had statistically higher FBS (99.76 ± 12.87), progesterone (15.59 ± 4.415), testosterone (42.19 ± 8.04) and AMH (5.69 ± 2.27) compare to control (73.43 ± 6.45, 12.76 ± 3.66, 20.05 ± 4.09, 3.66 ± 1.94), p-value 0.005, 0.006, 0.005 and 0.008, respectively, as shown in table 3-2.

Table 3-2: Mean difference of FBS, FSH, LH, Progesterone, Testosterone and AMH in both groups.

Biomarker		N	Mean ± SD	P value
FBS mg/dl	Patients	125	99.76 ± 12.87	0.005*
	Controls	125	73.43 ± 6.45	
FSH IU/ml	Patients	125	32.86 ± 8.21	0.16
	Controls	125	36.55 ± 7.02	
LH IU/ml	Patients	125	20.06 ± 3.80	0.08
	Controls	125	25.74 ± 7.42	
Progesterone ng/ml	Patients	125	15.59 ± 4.415	0.006*
	Controls	125	12.76 ± 3.66	
Testosterone ng/dl	Patients	125	42.19 ± 8.04	0.005*
	Controls	125	20.05 ± 4.09	
AMH ng/ml	Patients	125	5.69 ± 2.27	0.008*
	Controls	125	3.66 ± 1.94	

*p-value ≤ 0.05

A statistical significant association between presence of family history of DM in PCOS patients, having a BMI > 30 and Waist/ hip ration ≥ 0.8, p-value 0.05, 0.01 and 0.04, as shown in table 3-3

Table 3-3: Association between studied groups and family history of PCOS, DM, high BMI and Waist/ hip ratio.

	Patients	Control	p-value
Family history of PCOS	10 (8%)	2 (1.6%)	0.06
Family history of DM	40 (32%)	30 (24%)	0.05*
BMI ≥ 30	25 (20%)	4 (3.2%)	0.01 *
Waist/ hip ratio ≥ 0.8	36 (28.8%)	18 (14.4%)	0.04*
Total	125	125	

*p-value

DISCUSSION

Polycystic ovarian syndrome is a common disease affecting mainly women at childbearing age, however, the disease significantly lower the fertility ability of affected patients, as well as long-term metabolic and cardiovascular illness. The occurrence of PCOS is influenced by a number of genetic and environmental variables. The effects of this complicated condition extend beyond the reproductive system, affecting affected women's metabolic, cardiovascular, immunological, and psychological health.

The cause of PCOS is unknown, however insulin resistance and obesity have been connected to the condition, and one can contribute to the other in some way. In the current study that included both healthy and

diseased female's patients in reproductive age group, no statistical significant differences in age of patients and control group. The onset of disease is variable, however, most women discover that they have the disease in their 20s and 30s when having trouble getting pregnant.[12] Kałużna M et al.[13] study state that there was no statistical significant difference in mean of age between patients and control group in those with no central obesity, but PCOS patients with central obesity had statistical lower age group.

The weight of PCOS patients was significantly higher that agreed with.[14,15] the BMI of patients also was significantly higher than control group which agreed with.[16] Epidemiological research supports the link between obesity and PCOS, about 38%-88% of women with PCOS are overweight or obese.[17] Women with obesity had an odds ratio of 2.77 for developing PCOS in compare to non-obese.[9]

The waist circumference of included PCOS participant was significantly higher than control group, PCOS female had higher risk of development of central obesity which exacerbates endocrine and metabolic problems.[18] Population with BMI 30 (kg/m^2), the waist circumference may be a reliable indication of metabolic syndrome and insulin resistance. waist circumference appear to aid in identifying individuals in the normal weight, overweight, and class I obese BMI groups who are at heightened risk for health problems.[19]

PCOS has been identified as a significant risk factor for the development of diabetes, and it is advised that women with PCOS be tested for glucose intolerance. In order to diagnose both impaired glucose tolerance and diabetes, a 2-hour oral glucose tolerance test is preferable to a fasting plasma glucose test for the detection of glucose intolerance in PCOS. In the current study a higher mean of FBS had been observed in PCOS patients that agreed with Ansari JN et al.[20] study in which PCOS patients had higher FBS mainly due to insulin resistance, however, measurement of FBS in PCOS is not accurate method to estimate insulin resistance.[21]

Hormonal alteration is a common observation in PCOS patients, current study observe a higher progesterone, testosterone and AMH mean level in PCOS patients that corresponding to previous literatures.[20,13] Despite the higher mean of LH in PCOS, the difference was not approved statistically which could be related to sample size. The increase in progesterone and testosterone regarded a major factor in the development of oligomenorrhea, amenorrhea, infertility, enhanced premenstrual syndrome, and acne.[22]

Higher AMH is a common observation in PCOS patients, AMH is important hormonal that demonstrated to have important role in regulates follicular development and closely related to the severity of PCOS, studies stated that AMH level is changeable according to environmental and hereditary factors. The correlation between AMH and metabolic abnormality has been demonstrate by many studies.[23]

Regarding the factors that associated with PCOS development, current study found an association between positive family history of DM, BMI > 30 and Waist/ hip ratio ≥ 0.8 and PCOS patients.

The finding of Cheng C et al.[24] was agreed with current research, the study state that paternal positive history of DM was associated with increased risk of PCOS development, many evidences point to a significant genetic base for PCOS, which is reflected in a family history of the condition. Mawaddatina T et al.[25] defined that increase in waist circumference, hip circumference arm span and waist-to-hip ratio were a risk factors for PCOS development that is consistent with current finding.

The small sample size and absent of genetic assessment of participant is regarded a limitation point of current study. It's important to include a larger number of patients as well as included those who are at risk of disease or those with positive family history to determine the genetic and environmental factor of Iraqi patients.

CONCLUSION AND FUTURE PERSPECTIVES

The pathogenesis of PCOS remains elusive, with contributions from insulin resistance, adipose tissue dysfunction, abnormal steroidogenesis, and hypothalamic–pituitary–ovarian dysregulation. Genetic variants and epigenetic environmental factors probably contribute to the dysregulation of these varied systems and raise new avenues of research investigation in the rapidly evolving field of PCOS.

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