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Associated risk factors of Poly Cystic Ovarian disease in gynecologist refer; King Saud Medical City Riyadh Saudi Arabia

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ABSTRACT

Background: Polycystic ovary (PCO), a condition with a wide range of symptoms, can present in many different ways. Oligo- or anovulation, clinical and/or biochemical signs of hyperandrogenism, and PCO on ultrasound are the minimum two requirements for polycystic ovarian syndrome. Most information about PCO frequency in Saudi Arabia is unclear. *Objective:* In order to determine the clinical, etiologic and biochemical aspects of hirsutism in Saudi females, this study looked at the levels of reproductive hormones in PCOS patients, as well as the effects of age and body mass index (BMI) on the hormonal findings. It also examined ultrasonography results. *Methodology:* Between April 2022 and April 2023, a cross-sectional study was carried out among 213 PCOS patients who had undergone clinical evaluation and hormone testing at the Obstetrics and Gynecology Clinic at the King Saud Medical City in Riyadh, Saudi Arabia. *Conclusion:* Increased levels of testosterone and luteinizing hormone/follicle stimulating hormone (LH/FSH) and lower levels of progesterone, hormone-binding globulin (SHBG), and FSH were predictive with PCOS. This is unrelated to either age or BMI. Future studies with a larger sample size and data on insulin levels are required for a better understanding of the PCOS symptoms in the Saudi population.

Keywords: Polycystic ovaries, polycystic ovarian disease, polycystic ovarian syndrome.

1. INTRODUCTION

The most common endocrinopathy in women of reproductive age is PCOS and PCOD, which has a prevalence of up to 10%. It is a complicated condition that was first discovered to be the root of hirsutism and protracted anovulation in PCOS patients (Azziz and Adashi, 2016). PCOS is a condition with a variety of manifestations, but it is more prevalent among women in Saudi Arabia and around the world. A study on Saudi females in the ages of (18 to 28) revealed an outrageously higher estimate of PCOS prevalence at 53.7% (Shaista, 2013). The Rotterdam criteria, also known as the European Society of Human Reproduction and Embryology/American Society for Reproductive Medicine criteria, now includes a number of phenotypes based on a combination of any two of the three ultrasound findings of menstrual irregularity, polycystic ovaries, and hyperandrogenism (Rotterdam, 2004).

However, polycystic ovary is a common phenomenon in women who suffer from idiopathic hirsutism and oligomenorrhea (Adams et al., 1986). Numerous studies have established that women can have PCOS at ultrasonography without showing any indicators of androgen excess but with indications of ovarian dysfunction (Dewailly et al., 2006). PCOS is diagnosed by exclusion, just as other illnesses, and illnesses with phenotypes like PCOS must be eliminated. Congenital adrenal hyperplasia, Cushing disease, and virilizing cancers are a few examples (Legro et al., 2007). The main signs of PCOS are often irregular menstruation, acne, and an excess of androgenic hormones. Obesity is a common symptom in PCOS patients (Alemzadeh et al., 2010). It is important to note that PCOS is not merely a condition of the reproductive system because it has been associated to type 2 diabetes, metabolic syndrome, and occasionally cardiovascular disease (Setji and Brown, 2007).

According to ultrasound screening of individuals in the study with suspected PCOS, the presence of 12 or more 2-9 mm follicles appears to be more sensitive than ovarian volume or stromal brightness to diagnosis PCOS (Shaista, 2013). The reproductive hormones of PCOS-afflicted women have been compared to those of healthy controls by numerous study teams. Reduced levels of FSH and SHBG were seen in numerous investigations (Franks et al., 1991). Furthermore, PCOS patients exhibited higher mean testosterone concentrations than controls. Additionally, studies have shown that PCOS patients had greater levels of LH and the LH/FSH ratio than did healthy controls (Santbrink et al., 1997).

However, the results varied depending on the day of the cycle on which the hormones were measured. For example, LH levels in PCOS patients were only markedly elevated late in the menstrual cycle and not early (Iwasa et al., 2009). Even though the exact etiology for PCOS is unknown, three main pathophysiologic ideas have been put out to explain the clinical results Taylor et al., (1997), according to a study from 2006. These hypotheses include the ovarian hypothesis, and the LH hypothesis.

2. METHOD

Women who had visited the Obstetrics and Gynecology Clinic at the King Saud Medical City in Riyadh, Saudi Arabia, between April 2022 and April 2023 were the study's population. From all visits we specifically selected women who were diagnosed as PCOS according to Revised 2003 Rotterdam Criteria and finally we included 213 Saudi women with PCOS who in the study. According to the Rotterdam ESHRE/ASRM-sponsored PCOS consensus group, oligo- and/or anovulation, clinical and/or biochemical proof of hyperandrogenism, and polycystic ovaries are necessary in two out of every three instances to make the diagnosis of PCOS. Congenital adrenal hyperplasias, androgen-secreting tumors, and Cushing's disease were all ruled out as potential causes. Ultrasonography markers of PCOS include the existence of 12 or more 2–9 mm ovarian follicles, a peripheral distribution of ovarian follicles, an ovarian volume more than 10 cm3, and a significantly echogenic ovarian stroma (Shaista, 2013).

For the objectives of this investigation, a senior ultrasonographer in collaboration with a consultant gynecologist established all diagnoses. Each participant signed a release of information form. Blood samples were obtained between the first and fifth days of the period. LH, FSH, estradiol (E2), dehydroepiandrosterone sulfate (DHEASO4), SHBG, total testosterone, prolactin, and progesterone were all measured using an immunoassay. According to the definition of regular menstruation, there should be 9–16 cycles every 9–35 days, with no more than a 4-day variation in the duration between cycles. Acne and/or blackheads on the subjects' faces, necks, upper arms, chests, and backs were examined. The modified Ferriman and Gallwey score was used to assess the subjects for hirsutism (Tropeano et al., 1996). Hirsutism was indicated by a Ferriman and Gallwey score greater than 7. Each participant in the study gave written informed consent, and the King Saud Medical City ethics board approved the study's conduct under IRB number (H1R1-27-Sep23-01).

3. RESULTS

Table 1 lists the primary demographics of the cases and controls. There were 213 people in total, with 87 (40.85%) in the ages of (38 to 45), 81 (38.03%) in the ages of (18 to 27), and 45 (21.1%) in the ages of (28 to 37). According to BMI, 0 patients had an underweight BMI, 50 had a normal BMI, 20 had an overweight BMI, 63 had a class I BMI, 72 had a class II BMI, and 8 had a class III BMI. Regarding the clinical presentation (78.4%, 68.08% and 22.5%) of the subjects were had acne, oligomenorrhea, and infertility correspondingly. Additionally, 93.4% of the patient has 12 or more follicles that range in size from 2 to 9 mm.

Table 2 shows the results of multivariate analyses we conducted to eliminate age and BMI as potential confounding variables. LH/FSH and total testosterone did in fact show a positive correlate with disease (regression coefficient = 0.1 and 0.07, respectively, with P = 0.02 and 0.03, respectively). However, there was a negative relationship (regression coefficient = -0.01, -0.003, and -0.1, respectively, with P = 0.04, 0.03, and 0.001, respectively) between the disease and the hormones FSH, SHBG, and progesterone. Table 3 and Figure 1 demonstrate the relation between metabolic syndrome and different age groups in PCOS patients.

Table 1 Demographics statistics

		N	%
Age	18 to 27	81	38.03
	28 to 37	45	21.13
	28 to 37	45	21.13
Body mass index	less than 18.5	0	0
	From 18.5 to 24.9	50	23.47
	From 25 to 29.9	20	9.39
	From 30 to 34.9	63	29.58
	From 30 to 34.9	63	29.58
	From 35 to 39.9	72	33.8
	More than 40	8	3.756
Acne history	Yes	167	78.4
	No	46	21.6
Ferriman–Gallwey score	Less than 7	22	10.33
	More than 7	191	89.67
Oligomenorrhea history	Yes	145	68.08
	No	68	31.92
Infertility	Yes	48	22.54
	No	165	77.46
Ovarian Follicle	≥ 12 follicles or more follicles measuring 2–9 mm	199	93.43
	Ovarian volume > 9 cm	14	6.573

Table 2 Age and body mass index adjusted comparison of reproductive hormones

	Regression coefficient	P value
LH	0.03	0.19
FSH	0.01	0.04
LH//FSH	0.1	0.02
E2	0.002	0.7
DHEA - SO4	0.003	0.94
SHBG	0.003	0.03
TOTAL TESTOSTERONE	0.07	0.03
PROLACTIN	0.002	0.6
PROGESTERONE	0.1	0.001
CORTISOL	0.001	0.9

Table 3 Metabolic syndrome prevalence rates for women with PCOS vary by age group

Age (years)	Metabolic syndrome	Patients with PCOS (n=213); n (%)	p-value
18 to 27 N= 81	Have Metabolic syndrome	21 (9.8)	0.069
	Don't have Metabolic syndrome	60 (28.1)	
28 to 37 N= 45	Have Metabolic syndrome	27 (12.6)	0.002
	Don't have Metabolic syndrome	18 (8.4)	
38 to 45 N= 87	Have Metabolic syndrome	68 (31.9)	0.002*
	Don't have Metabolic syndrome	19 (8.9)	

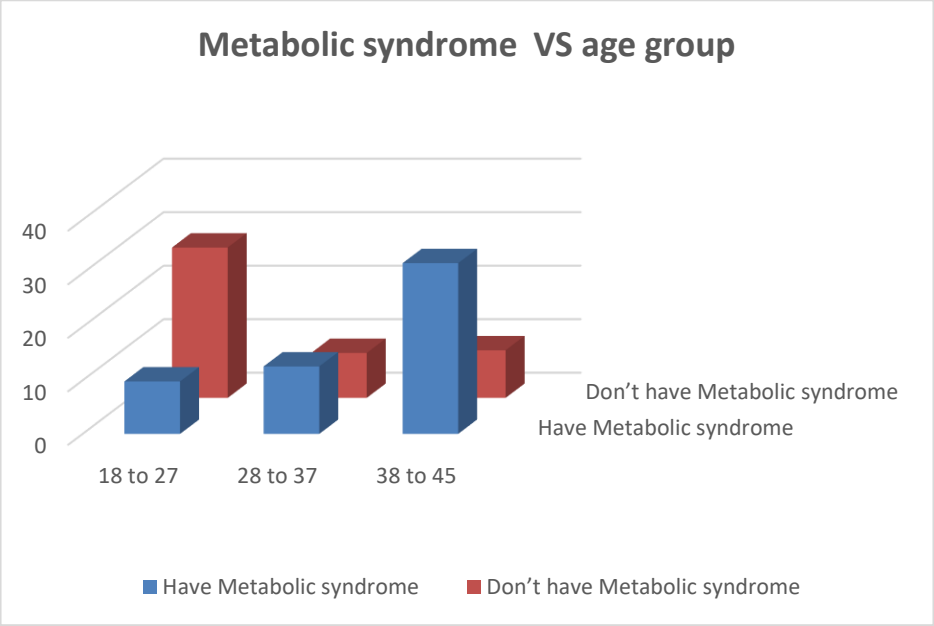

Figure 1 Metabolic syndrome prevalence rates for women with PCOS vary by age group

4. DISCUSSION

Both the clinical presentation and the level of the reproductive hormones in Saudi women with PCOS were assessed in this study using the Rotterdam criteria. Since there aren't many studies that discuss PCOS in the Saudi population, our work is noteworthy. Although PCOS can manifest at any age throughout the reproductive years Yusuf et al., (2023), the majority of patients (40.8%) in our research group were in the ages of (38 to 45). Another study found that children as young as 9 years old can acquire PCOS (Sirmans and Pate, 2013). In addition, 33.8% of them were classified as obese even though obesity is not a diagnostic prerequisite because their BMIs ranged from 35 to 39.9. This reflects the fact that obesity is a common finding in both PCOS and the Saudi population as a whole. The literature on the connection between BMI and levels of reproductive hormones has produced conflicting results.

In contrast, some studies found a correlation between lower LH and higher BMI Bronstein et al., (2011), but other studies found no relationship between BMI and LH (Banaszewska et al., 2003; Toprak et al., 2001). On the other hand, recent research indicates that the clinical symptoms and signs of PCOS, including those related to metabolism, may be affected by ageing (Tropeano et al., 1996). In fact, the results of the multivariate regression analysis showed that cases, independent of age and weight, had higher levels of LH/FSH and total testosterone and lower levels of FSH, SHBG, and progesterone. Even in the adjusted comparison, we failed to find any evidence of an increase in LH levels. We failed to find a noticeable increase in LH in PCOS patients, in contrast to earlier published studies (Santbrink et al., 1997).

One important factor that tends to affect the results is the change in hormone levels that occurs with the menstrual cycle. According to a recent publication Iwasa et al., (2009), elevated LH levels are not especially reproducible throughout the early menstrual cycle, which is when we tested LH in our study. The fact that the majority of our patients had a BMI of 25 or above may

have had a substantial influence on the results because it has been demonstrated that lean PCOS patients had greater levels of LH than obese PCOS patients (Laven et al., 2002). According to multiple studies in the literature Falcetta et al., (2021), not all PCOS patients exhibited elevated LH, and this most likely contributed to the average LH elevation seen in our study not reaching statistical significance. Because there isn't enough information available on insulin levels, there are certain restrictions on how the hormonal data should be interpreted.

5. CONCLUSION

Increased LH/FSH, testosterone, and reduced levels of FSH, SHBG, and progesterone were all indicators of PCOS. This is unrelated to either age or BMI. To completely understand how PCOS develops in the Saudi population, more studies with a larger sample size and information on insulin levels are necessary.

Abbreviations

PCOS: Polycystic ovarian syndrome
PCOD: Polycystic ovarian disease
LH: Luteinizing hormone
FSH: Follicle stimulating hormone
SHBG: Hormone-binding globulin
BMI: Body mass index
DEHA: Dehydroepiandrosterone sulfate

Ethical approval

Each participant in the study gave written informed consent. Research approved by king saud medical city review board under IRB (H1R1-27-Sep23-01)

Author's Contribution

Ibtesam Fawaz Alshammari: Participated in all steps of research from idea to publication; Fawaz Abdullah Ibrahim Alabdan, Rakan Hassan Marghlani, Ibrahim Ismail Hamami, Abdulaziz Abdo Ali Agili: Participated in writing introduction and discussion; Sultan Hamed Alanazy, Abdullah Ahmed Mohamed Ghouth, Saad Khaleel Alonze, Alanazi, Salha Aowad: Participated in writing results method and collecting literature

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Conflict of interest

The authors declare that there is no conflict of interests.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

REFERENCES AND NOTES

1. Adams J, Polson DW, Franks S. Prevalence of polycystic ovaries in women with anovulation and idiopathic hirsutism. Br Med J (Clin Res Ed) 1986; 293(6543):355-9. doi: 10.1136/bmj.293.6543.35
2. Alemzadeh R, Kichler J, Calhoun M. Spectrum of metabolic dysfunction in relationship with hyperandrogenemia in obese adolescent girls with polycystic ovary syndrome. Eur J Endocrinol 2010; 162(6):1093-1099. doi: 10.1530/EJE-10-0205
3. Azziz R, Adashi EY. Stein and Leventhal: 80 years on. Am J Obstet Gynecol 2016; 214(2):247.e1-247.e11. doi: 10.1016/j.ajog.2015.12.013
4. Banaszewska B, Spaczyński RZ, Pelesz M, Pawelczyk L. Incidence of elevated LH/FSH ratio in polycystic ovary syndrome women with normo- and hyperinsulinemia. Rocz Akad Med Bialymst 2003; 48:131-134.
5. Bronstein J, Tawdekar S, Liu Y, Pawelczak M, David R, Shah B. Age of onset of polycystic ovarian syndrome in girls may

be earlier than previously thought. J Pediatr Adolesc Gynecol 2011; 24(1):15-20. doi: 10.1016/j.jpag.2010.06.003
6. Dewailly D, Catteau-Jonard S, Reyss AC, Leroy M, Pigny P. Oligoanovulation with polycystic ovaries but not overt hyperandrogenism. J Clin Endocrinol Metab 2006; 91(10):3922-3927. doi: 10.1210/jc.2006-1054
7. Falcetta P, Benelli E, Molinaro A. Effect of aging on clinical features and metabolic complications of women with polycystic ovary syndrome. J Endocrinol Invest 2021; 44(12):2725-2733. doi: 10.1007/s40618-021-01594-5
8. Franks S, Kiddy D, Sharp P. Obesity and polycystic ovary syndrome. Ann N Y Acad Sci 1991; 626:201-206. doi: 10.1111/j.1749-6632.1991.tb37915.x
9. Iwasa T, Matsuzaki T, Murakami M. Reproducibility of luteinizing hormone hypersecretion in different phases of the menstrual cycle in polycystic ovary syndrome. J Obstet Gynaecol Res 2009; 35(3):514-519. doi: 10.1111/j.1447-0756.2008.00998.x
10. Laven JS, Imani B, Eijkemans MJ, Fauser BC. New approach to polycystic ovary syndrome and other forms of anovulatory infertility. Obstet Gynecol Surv 2002; 57(11):755-767. doi: 10.1097/00006254-200211000-00022
11. Legro RS, Barnhart HX, Schlaff WD, Carr BR, Diamond MP, Carson SA, Steinkampf MP, Coutifaris C, McGovern PG, Cataldo NA, Gosman GG, Nestler JE, Giudice LC, Leppert PC, Myers ER; Cooperative Multicenter Reproductive Medicine Network. Clomiphene, metformin, or both for infertility in the polycystic ovary syndrome. N Engl J Med 2007; 356(6):551-66. doi: 10.1056/NEJMoa063971
12. Rotterdam. ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 2004; 81(1):19-25. doi: 10.1016/j.fertnstert.2003.10.004
13. Setji TL, Brown AJ. Polycystic ovary syndrome: diagnosis and treatment. Am J Med 2007; 120(2):128-132. doi: 10.1016/j.amjmed.2006.06.029
14. Shaista SG. Prevalence and ultrasound features of Polycystic ovaries in young unmarried Saudi females. J Microsc Ultrastruct 2013; 1(1-2):30-34. doi: 10.1016/j.jmau.2013.06.002
15. Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. Clin Epidemiol 2013; 6:1-13. doi: 10.2147/CLEP.S37559
16. Taylor AE, McCourt B, Martin KA, Anderson EJ, Adams JM, Schoenfeld D, Hall JE. Determinants of abnormal gonadotropin secretion in clinically defined women with polycystic ovary syndrome. J Clin Endocrinol Metab 1997; 82(7):2248-2256. doi: 10.1210/jcem.82.7.4105
17. Toprak S, Yönem A, Cakir B, Güler S, Azal O, Ozata M, Corakçi A. Insulin resistance in nonobese patients with polycystic ovary syndrome. Horm Res 2001; 55(2):65-70. doi: 10.1159/000049972
18. Tropeano G, Vuolo IP, Lucisano A, Liberale L, Barini A, Carfagna P, Caroli G, Menini E, dell'Acqua S. Gonadotropin levels in women with polycystic ovary syndrome: their relationship to body weight and insulin levels. J Endocrinol Invest 1996; 19(3):139-145. doi: 10.1007/BF03349856
19. Santbrink EJ, Hop WC, Fauser BC. Classification of normogonadotropic infertility: polycystic ovaries diagnosed by ultrasound versus endocrine characteristics of polycystic ovary syndrome. Fertil Steril 1997; 67(3):452-458. doi: 10.1016/s0015-0282(97)80068-4
20. Yusuf ANM, Amri MF, Ugusman A, Hamid AA, Wahab NA, Mokhtar MH. Hyperandrogenism and Its Possible Effects on Endometrial Receptivity: A Review. Int J Mol Sci 2023; 24(15):12026. doi: 10.3390/ijms241512026