

Original Article

STUDY OF PREVALENCE OF POLYCYSTIC OVARIAN SYNDROME AMONG ADOLESCENT AND YOUNG ADULT STUDENTS OF NAVODAYA GROUP OF INSTITUTIONS

KOLAN PALLAVI^{1*}, HARSHITHA K.²

¹Department of Obstetrics, Dr. Patnam Mahender Reddy Institute of Medical Sciences, Chevella, Ranga Reddy District, Telangana, India.

²ESIC Hospital, Sanath Nagar, Hyderabad, India

*Corresponding author: Kolan Pallavi; *Email: kpallavi.patnam@gmail.com

Received: 15 Apr 2024, Revised and Accepted: 04 Jun 2024

ABSTRACT

Objective: Polycystic ovary syndrome (PCOS) is considered to be the most common endocrine disorder in women of reproductive age. The objective of this study was to calculate the prevalence of PCOS in Navodaya group of institutions based on Rotterdam criteria.

Methods: A prospective study of 500 girls of Navodaya group of institutions (school, medical, dental, and nursing, pharmacy, physiotherapy and engineering students) aged 13 to 25 y who underwent clinical examination. Out of which, 68 girls with oligomenorrhea and/or hirsutism were invited for biochemical, hormonal, and ultrasonographic evaluation for diagnosis of PCOS by Rotterdam criteria.

Results: Out of 500 girl students, 68 girls satisfied Rotterdam's criteria for PCOS. Under the Rotterdam criteria, the estimated prevalence of PCOS in adolescent and young adult students was 13.6%.

Conclusion: Prevalence of PCOS in adolescent and young adult students of Navodaya Group of Institutions was 13.6%. This draws attention to the issue of early diagnosis in adolescent girls.

Keywords: Endocrine disorder, Polycystic ovary syndrome, Hirsutism, Rotterdam criteria

© 2024 The Authors. Published by Innovare Academic Sciences Pvt Ltd. This is an open access article under the CC BY license (<https://creativecommons.org/licenses/by/4.0/>) DOI: <https://dx.doi.org/10.22159/ijcpr.2024v16i4.5003> Journal homepage: <https://innovareacademics.in/journals/index.php/ijcpr>

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a heterogeneous disorder. As one of the leading cause of ovulatory infertility, it is believed that 5-10% of the reproductive-age female population is living with polycystic ovary syndrome [1]. First recognized in 1935, PCOS is characterized by the presence of polycystic ovaries, menstrual irregularities, and clinical or biochemical hyperandrogenism [2]. The development of PCOS has been linked to hereditary and environmental factors, including genetics, insulin resistance, obesity and birth weight. The presence of PCOS is associated with an increased prevalence of adverse health conditions such as the metabolic syndrome, cardiovascular disease and type II diabetes mellitus. Insulin resistance is believed to play a key role in the development of PCOS and in the development of related conditions. In the past few y, research has been done to better understand the mechanisms behind the development of polycystic ovary syndrome and the impact it has on the female body, particularly in relationship to insulin resistance. Features of hyperandrogenism such as acne, hirsutism, seborrhoea, alopecia characterize PCOS clinically. It is also characterized by reproductive abnormalities such as menstrual disturbances, anovulation, infertility, miscarriage, gestational diabetes and pre-eclampsia. In recent y it has been widely recognized that most women with PCOS have some degree of insulin resistance and early onset of type 2 diabetes. Women with PCOS have the constellation of symptoms such as insulin resistance, obesity, hypertension and dyslipidemia, defining so-called syndrome X and thus an increased incidence of cardiovascular risk factors [3]. No single diagnostic criterion is sufficient for clinical diagnosis. Hence, PCOS remains a diagnosis of exclusion. There have been three separate and distinct efforts to refine the diagnostic criteria for PCOS. In 1990, the National Institute of Child Health and Human Development (NICHD) concluded that major criteria were: Hyperandrogenism and/or Hyperandrogenemia; Menstrual dysfunction; and exclusion of other known disorders having similar Clinical presentations. In 2003, the European Society for Human Reproduction and Embryology (ESHRE) and the American Society for Reproductive Medicine (ASRM) convened in Rotterdam criteria

(2 out of 3) which include Oligomenorrhoea or amenorrhoea; Clinical or biochemical signs of hyperandrogenism; Polycystic ovaries on ultrasound, also excluding other androgen excess disorders. In 2006, Androgen excess and PCOS society (AE-PCOS) concluding the diagnosis requires Hyperandrogenism (hirsutism and/or hyperandrogenemia); Ovarian dysfunction (oligo/anovulation and/or polycystic ovaries); Exclusion of other androgen excess or related disorders [4]. The etiology and pathogenesis of PCOS is still obscure and controversial and an array of plausible pathophysiologies has emerged over the last several decades of study. Inappropriate gonadotropin secretion with elevated LH and relatively low FSH secretion is typical [5]. In women with PCOS, 65-75% have a high LH to FSH ratio due to more increased levels of LH than low levels of FSH [6]. It is reported an association between obesity, insulin resistance and impaired fertility in PCOS women. Although insulin resistance is not a criterion for the PCOS diagnosis, it is the central feature of the syndrome. Insulin resistance may be defined as increased insulin demand to maintain blood glucose levels normal. Among the most frequently used methods to measure insulin resistance in large-scale studies are: fasting insulin, insulin c-peptide, insulin levels during an oral glucose tolerance test (OGTT), and homeostasis assessment model (HOMA) and hyperinsulinemic clamp. Insulin stimulates androgen synthesis in both the ovaries and adrenals. Ovarian stroma, theca cells and the adrenals are not resistant to the effect of insulin on androgen synthesis. Further, insulin acts synergistically with LH and augments steroid synthesis in the ovaries, resulting in follicle arrest and anovulation. Insulin also inhibits the synthesis of sex hormone-binding globulin (SHBG) in the liver, resulting in higher levels of free testosterone [7]. Modern treatment of PCOS is directed towards establishing fertility, including normal and ovulatory cycles and improving hirsutism. One important aim in the treatment of PCOS women is to reduce the incidence of the long-term consequences of metabolic sequelae of PCOS. The present study was aimed to investigate the prevalence of PCOS in Adolescents and young adult students in the age group of 13-25 y in Navodaya group of institutions, Raichur and to prevent the long-term consequences like obesity, infertility, hyperinsulinism, cardiovascular risks etc. The

study also aimed to create awareness and long term complications of this disease and Advise lifestyle modifications.

MATERIALS AND METHODS

Institutional ethics clearance

The study was approved by the Ethical Committee of Navodaya medical college and was conducted according to the guidelines of the institute's Ethical Committee.

Study group and age

The study was conducted on adolescent and young adult students of navodaya group of institutions (school, medical, dental, and nursing, pharmacy, physiotherapy and engineering female students) in the age group of 13-25 y. Adolescent and young adult students meeting the Revised 2003 consensus on diagnostic Rotterdam criteria related to polycystic ovary syndrome which is the modified version of National Institute of Child Health and Human Development 1990 were informed of the study and those who gave consent were included in the study.

Diagnostic criteria

Any two of the three features: oligo/amenorrhea, clinical and/or biochemical hyperandrogenism, and polycystic ovaries, were included in diagnosis. Oligo/amenorrhea: Oligomenorrhea (<9 cycles/yr or menstrual interval>35days) or secondary amenorrhea (absence of menses in last 6 or more months) Clinical hyperandrogenism: Hirsutism, Male type baldness, Acne Hirsutism scoring: Modified Ferriman and Gallway (mFG) score. Scores include<8: Mild; 8-15: Moderate;>15: Severe. Biochemical hyperandrogenism: Serum testosterone level of>76 ng/dl in the absence of other causes of hyperandrogenism. Polycystic ovaries: presence of>10 cysts, 2-8 mm in diameter, usually combined with increased ovarian volume of>10 cm³, and an echo-dense stroma in pelvic ultrasound scan.

Data collection

All students (13-25) who consented for the study were asked questions on the pattern of menstrual cycle, hirsutism, acne, alopecia, and Acanthosis nigricans, and information about past diagnosis or treatment of PCOS or any other illness. Physical examination is done to look for external features of PCOS and also to

exclude other conditions that could mimic PCOS such as Cushing syndrome, adrenal hyperplasia or androgen producing neoplasm. Questions were asked about the use of oral contraceptive pills or any other hormones that could affect the length of the menstrual cycle. Self-reported degree of hirsutism was assessed using modified Ferriman-Gallwey (mF-G) scoring method. The girls were asked to compare the amount of body hair they had with a chart of pictures displaying the degree of hair growth in nine regions (i. e., upper lip, chin, chest, upper and lower abdomen, upper and lower back, upper arms, and medial side of thighs). Hirsutism scores recorded by the girls were checked for accuracy during clinical examination and corrected with the consent of the participant when deemed necessary. Also girls were asked about the presence of acne or hair fall from the scalp although it was not quantified. All girls with oligomenorrhea and/or hirsutism (as per the above said definitions) was asked to come for pelvic ultrasound and biochemical investigations. Ultrasound scanning of the abdomen and pelvis with special attention on ovaries was carried out. Hormone assays were done by RIA (Testosterone) and immunoradiometric assay (TSH, PRL, LH, FSH).

RESULTS

This prospective study is conducted in Navodaya Group of Institutions, Raichur. The study comprises of 500 girl students in the age group between 13-25. Among the 500 screened individuals, 13.6% have PCOS and 86.4% have no PCOS (table 1 and fig. 1) and majority of the students were in the age group of 18-20 (table 2). More cases of PCOS was reported in the age group 21-23 y, followed by 18-20 y; however, the results were insignificant (table 3). Majority of the PCOS patients were reported with Oligomenorrhea (table 4) nearly 60 cases out of 64 cases. It was observed that mean age of PCOS cases was 19.85 y and normal subjects were 19.95. There was no significant difference in the mean age between PCOS and normal subjects. mean height of PCOS cases was 160.18 cms and normal subjects was 159.49 cms. There was no significant difference in the mean height among PCOS and normal subjects mean weight of PCOS cases was 61.94 and Normal subjects was 56.91. There was significant difference in the mean weight among PCOS and Normal subjects. I. e. PCOS cases weight was high than Normal subjects. Similarly, mean BMI, mean Waist circumference and WHR was higher in PCOS cases than normal subjects. This difference was statistically significant (table 5 and table 6).

Table 1: Prevalence of PCOS among adolescents and young adults

		Frequency	Percent
Prevalence	PCOS	68	13.6%
	Normal	432	86.4%
	Total	500	100.0

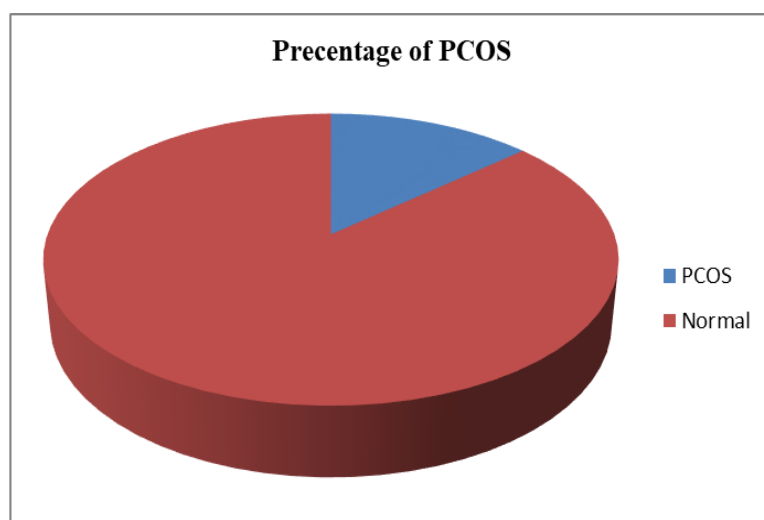


Fig. 1: Percentage of PCOS among study population

Table 2: Age-wise distribution

		Frequency	Percent
Agegroup in y	15-17	56	11.2
	18-20	215	43.0
	21-23	214	42.8
	>23	15	3.0
	Total	500	100.0

Table 3: Association between age and PCOS

Duration		PCOS		Total	Chi-square	Df	p	Inference
		Present	Absent					
Age	15-17	9	47	56	2.787	3	0.42 (>0.05)	Not significant
	18-20	23	192	215				
	21-23	34	180	214				
	>23	2	13	15				
	Total	68	432	500				

Table 4: Association between oligomenorrhea and PCOS

Association		PCOS		Total	Chi-square	df	p	Inference
		Present	Absent					
Oligomenorrhea	Present	60	5	65	423.142	1	0.0001 (<0.001)	Highly significant
	Absent	4	431	435				
	Total	64	436	500				

Table 5: General characteristics of PCOS adolescent and young adult students

Duration	Number	Mean	Std. deviation	Std. error	Range	Minimum	Maximum
AGE (y)	68	19.85	2.21	.27	9	15	24
Menarche age	68	12.57	.74	.09	3	11	14
F-G score	68	10.43	6.69	.81	21	0	21
Height (cms)	68	160.18	4.33	.53	22	150	172
Weight (kgs)	68	61.94	7.17	.87	31	44	75
BMI	68	24.20	2.82	.34	11.20	17.60	28.80
Waist (cms)	68	91.28	6.51	.79	26	76	102
HIP (cms)	68	104.78	6.41	.78	25	90	115
WHR	68	.87	.04	.00	.18	.75	.93
FBS (gm/dl)	67	86.06	8.74	1.07	38	68	106
LH (IU/l)	68	14.57	2.34	.28	11.40	10.60	22.00
FSH (IU/l)	68	8.98	2.20	.27	8.70	4.30	13.00
LH: FSH	68	1.70	.49	.06	1.90	1.19	3.09
Prolactin (mcg/l)	68	6.16	2.37	.29	12.40	1.20	13.60
Testosterone (ng/dl)	68	56.65	30.18	3.66	87.0	12.0	99.0
TSH (mU/l)	68	2.60	1.07	.13	5.12	.38	5.50

Table 6: General characteristics of PCOS and normal adolescent and young adults

Duration	Group	Number	Mean	Std. deviation	t	Df	p	Inference
Age (y)	PCOS	68	19.85	2.214	-.305	498	.761 (>0.05)	Not significant
	Normal	432	19.95	1.923				
Menarche age (y)	PCOS	68	12.57	.739	-.884	498	.377 (>0.05)	Not significant
	Normal	432	12.67	.817				
Height (cms)	PCOS	68	160.18	4.329	1.121	498	.263 (>0.05)	Not significant
	Normal	432	159.49	4.741				
Weight (kgs)	PCOS	68	61.94	7.169	5.474	498	.0001 (<0.001)	Highly significant
	Normal	432	56.91	7.025				
BMI	PCOS	68	24.1976	2.82083	5.713	498	.0001 (<0.001)	Highly significant
	Normal	432	22.3882	2.36070				
Waist (cms)	PCOS	68	91.28	6.513	5.843	498	.0001 (<0.001)	Highly significant
	Normal	432	85.66	7.497				
HIP (cms)	PCOS	68	104.78	6.406	2.988	498	.003 (<0.05)	Significant
	Normal	432	101.90	7.532				
WHR	PCOS	68	.8650	.03919	6.931	498	.0001 (<0.001)	Highly significant
	Normal	432	.8345	.03278				

DISCUSSION

Polycystic ovary syndrome is one of the most common endocrine disorders in women of reproductive age group. This disorder is a

significant public health concern in society, which therefore indicates a need to accurately identify the proportion of women affected. Despite PCOS being considered the most common endocrine disorder in women of reproductive age, prevalence

estimates are highly variable; ranging from 2.2% to as high as 26% [8]. This variability is due to several factors. Firstly, diagnosing the disorder is logistically difficult and secondly, heterogeneity in the presentation of symptoms has contributed to lack of agreement over the diagnostic criteria. This study has shown 13.6% prevalence of PCOS according to Rotterdam criteria in girls between 13 to 25 y of age. Out of those, 94.1% had oligomenorrhea, 95.58% had polycystic ovaries on ultrasound. This higher prevalence in India as compared to other Asian countries could be expected because we know the strong etiological link between PCOS and diabetes, and India has the higher prevalence of diabetes. Ethnic differences must be taken into consideration when analyzing studies on PCOS. In a Chinese article, no cases of hirsutism were found among 915 women. Other investigators have already reported a low prevalence of hirsutism in Asian women. On the other hand, a high proportion of hirsutism has been found in Greek women. Ethnic variations in the occurrence of hirsutism reflect one of the difficulties in standardizing diagnostic criteria for PCOS [8]. Nidhi R *et al.* (2011) [9] conducted a prospective study on 460 girls aged 15 to 18 y to find out the prevalence of PCOS who underwent clinical examination. Out of which 72 girls with oligomenorrhoea and/or hirsutism were invited for biochemical, hormonal and ultrasonographic evaluation for diagnosis of PCOS by Rotterdam criteria. Out of 460 girls, one (0.22%) had oligo/amenorrhoea with clinical hyperandrogenism, 29 (6.30%) had oligomenorrhoea with polycystic ovaries, 1(0.22%) had polycystic ovaries with clinical hyperandrogenism and 11(2.39%) had oligomenorrhoea with polycystic ovaries in presence of clinical hyperandrogenism. Thus 42(9.13%) girls satisfied Rotterdam's criteria for PCOS, which increased to 50.46(10.97%) when imputed data were included. Prevalence of PCOS in Indian Adolescents is 9.13%. Wendy March *et al.* [10] conducted a retrospective birth cohort study was carried out in which 728 women born during 1973–1975 in a single maternity hospital were traced and interviewed in adulthood (age ¼ 27–34 year; n ¼ 728). Symptoms of PCOS (hyperandrogenism, menstrual dysfunction and polycystic ovaries) were identified by examination and the presence of polycystic ovaries in those that did not consent to the ultrasound was imputed. The estimated prevalence of PCOS in this birth cohort using the NIH criteria was 8.7+2.0%. Under the Rotterdam criteria, the prevalence was 11.9+2.4% which increased to 17.8+2.8% when imputed data were included. Under the AES recommendations, PCOS prevalence was 10.2+2.2%, and 12.0+2.4% with the imputed data. Of the women with PCOS, 68–69% did not have a pre-existing diagnosis. It is important that the girls with the symptoms should be referred for ultrasound and blood tests to facilitate early identification and to prevent long-term complications. CVD is well recognized as part of the spectrum of insulin resistance syndrome, leading to endothelial dysfunction, microalbuminuria, proatherosclerotic and inflammatory factors, diabetes, dyslipidemia, and a number of other abnormalities. Reproductive abnormality is a major clinical component of PCOS, with increased gestational diabetes in both obese and nonobese women, pregnancy-induced hypertension, and preeclampsia rates. There is also increased endometrial cancer risk, suggesting the need for cyclic progesterone administration. The risk of ovarian cancer is increased 2.5-fold, particularly among women who had never used oral contraceptives. Lifestyle modifications include diet, exercise and weight loss. The weight loss is recommended as the first line of treatment for women with PCOS. In women with PCOS and excess weight, a reduction of as little as 5% of total body weight has been shown to reduce insulin resistance and testosterone levels as well as improving body composition and cardiovascular diseases [12, 13].

CONCLUSION

PCOS is one of the most prevalent disease with endocrine and metabolic implications. It usually presents with menstrual irregularities and signs of androgen excess, such as hirsutism and acne. The main endocrine abnormality associated with PCOS is insulin resistance, which in the long term, may lead to diabetes and cardiovascular morbidity. Early recognition and prompt treatment in adolescents is essential for preventing long-term sequelae. Implications of PCOS are lifetime risks of endometrial cancer, ovarian cancer, type 2 DM, etc. Thus, early and timely diagnosis is

the key for its treatment. Awareness among young girls is required as they are the primary target. Awareness can be spread through healthcare providers by conducting presentations, workshops, seminars, etc at various places to educate the female population about this chronic syndrome. Weight reduction and lifestyle modification is essential and should always accompany any treatment modality, especially in the crucial life period of adolescence. Metformin is the most widely used insulin-reducing agent. With efficacy and few side effects reported, OCs remain a good therapeutic option, especially in patients with clinical evidence of hyperandrogenemia, while cyclical progestins are used when cycle control is the main therapeutic target.

FUNDING

Nil

AUTHORS CONTRIBUTIONS

All the authors have contributed equally

CONFLICTS OF INTERESTS

Declared none

REFERENCES

1. Ardabili HR, Gargari BP, Farzadi L. Vitamin D supplementation has no effect on insulin resistance assessment in women with polycystic ovary syndrome and vitamin D deficiency. *Nutr Res.* 2012;32(3):195-201. doi: 10.1016/j.nutres.2012.02.001, PMID 22464806.
2. Stein IF, Leventhal ML. Amenorrhea associated with bilateral polycystic ovaries. *Am J Obstet Gynecol.* 1935;29(2):181-91. doi: 10.1016/S0002-9378(15)30642-6.
3. De Leo V, La Marca A, Petraglia F. Insulin-lowering agents in the management of polycystic ovary syndrome. *Endocr Rev.* 2003;24(5):633-67. doi: 10.1210/er.2002-0015, PMID 14570747.
4. Speroff I, Glass RH, Kase NG, editors. Chapter 6. Anovulation. In: *Clinical gynecologic Endocrinology and Infertility*, (7th-Edition). Baltimore: Williams and Wilkins; 1989. p. 213-31.
5. Zawadzki JK, Dunaif A. Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. In: Dunaif A, Givens JR, Haseltine FP, Merriam GR, editors. *Polycystic ovary syndrome*. Polycystic ovary syndrome; 1992. p. 377-84.
6. Kalro BN, Loucks TL, Berga SL. Neuromodulation in polycystic ovary syndrome. *Obstet Gynecol Clin North Am.* 2001;28(1):35-62. doi: 10.1016/S0889-8545(05)70184-4, PMID 11293003.
7. Dunaif A. Insulin action in the polycystic ovary syndrome. *Endocrinol Metab Clin North Am.* 1999;28(2):341-59. doi: 10.1016/S0889-8529(05)70073-6, PMID 10352922.
8. Gabrielli L, Aquino EM. Polycystic ovary syndrome in Salvador, Brazil: a prevalence study in primary healthcare. *Reprod Biol Endocrinol.* 2012 Nov 22;10:96. doi: 10.1186/1477-7827-10-96, PMID 23173761, PMCID PMC3560118.
9. Nidhi R, Padmalatha V, Nagarathna R, Amritanshu R. Prevalence of polycystic ovarian syndrome in Indian adolescents. *J Pediatr Adolesc Gynecol.* 2011 Aug;24(4):223-7. doi: 10.1016/j.jpag.2011.03.002, PMID 21600812.
10. March WA, Moore VM, Willson KJ, Phillips DI, Norman RJ, Davies MJ. The prevalence of polycystic ovary syndrome in a community sample assessed under contrasting diagnostic criteria. *Hum Reprod.* 2010 Feb;25(2):544-51. doi: 10.1093/humrep/dep399, PMID 19910321.
11. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab.* 2004;89(6):2745-9. doi: 10.1210/jc.2003-032046, PMID 15181052.
12. Moran LJ, Noakes M, Clifton PM, Wittert GA, Belobrajdic DP, Norman RJ. C-reactive protein before and after weight loss in overweight women with and without polycystic ovary syndrome. *J Clin Endocrinol Metab.* 2007;92(8):2944-51. doi: 10.1210/jc.2006-2336, PMID 17504892.
13. Deswal R, Narwal V, Dang A, Pundir CS. The prevalence of polycystic ovary syndrome: a brief systematic review. *J Hum Reprod Sci.* 2020 Oct-Dec;13(4):261-71. doi: 10.4103/jhrs.JHRS_95_18, PMID 33627974, PMCID PMC7879843.