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Original Research Article

## Clinical study of polycystic ovarian syndrome (PCOS) in tertiary care centre

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### ABSTRACT

**Background:** A true definition of polycystic ovarian syndrome involves a blending of the morphological and histological ovarian changes with endocrine abnormalities associated with these changes. Objective of present study was to detect the differences among women with PCOS and without PCOS.

**Methods:** Twenty-five subjects with PCOS were included in the study on first cum first basis and twenty-five non PCOS subjects just next to each PCOS cases were included as control group.

**Results:** Mean age of PCOS and Non PCOS subjects were 42.68 and 43.88 years respectively. Sixteen percentage of PCOS were from higher socio-economic status, which was statistically significant. PCOS subjects had 96% hirsutism, 48% acne, 4% heavy voice and breast atrophy which was also statistically significant.

**Conclusions:** Women with PCOS have a hormonal imbalance and metabolism problems that may affect their health and fertility, we found PCOS subjects are more in urban population with strong family history. Lifestyle modification and dietary modification helps for the long-term health prognosis. women who are at risk of PCOS to be educated and managed for the continuing health risk and planned potential therapeutic strategies. Further research has to look into possibility of genes linked into PCOS.

**Keywords:** Hormonal imbalance, PCOS, Non PCOS

### INTRODUCTION

A true definition of polycystic ovarian syndrome involves a blending of the morphological and histological ovarian changes with endocrine abnormalities associated with these changes. The original description given by Stein and Leventhal included obesity, amenorrhoea, infertility and hirsutism in association with bilateral enlarged cystic ovaries showing a typical histological appearance of thickened capsule, multiple cysts and dense hypertrophied interstitial tissue.<sup>1,2</sup>

#### *Clinical presentation of PCOS*

There is wide variation in the clinical presentation of

polycystic ovarian syndrome and according to Marantides, only 10% to 20% of women with polycystic ovarian syndrome may report symptoms.<sup>3</sup>

Women with PCOS seek health care for 3 major reasons:

- Menstrual irregularities
- Anovulatory Infertility
- Androgen excess

Menstrual irregularity usually dates back to puberty and may take the form of oligomenorrhoea, amenorrhoea, or frequent periods. However, menstrual cycles may be normal. Studies show that oligomenorrhoea is present in 29% to 47% of women with PCOS, amenorrhoea in 19%

to 51% of cases, and frequent periods in 3% of cases. Normal cycles are present in 15% to 30% of cases.<sup>4</sup>

The ovarian hyperandrogenism is the most common abnormality in polycystic ovarian syndrome. It is dependent on gonadotropin secretion, more so with classic polycystic ovarian syndrome. This trait may possibly be related to cytochrome p 450 C17 alpha dysregulation.<sup>1,5</sup> Incidence of infertility in PCOS is 75%. It is mainly due to anovulation.

The androgen excess of PCOS is usually manifested by varying degrees of hirsutism and rarely acne. Such symptoms are usually associated with serum total testosterone concentration more than 60 mg/dl and androstenedione concentration more than 200 mg/dl. More advanced signs of androgen excess such as clitoromegaly and loss of female body contour can be associated with hyperthecosis.<sup>6-8</sup> Hirsutism refers to the development of excessive terminal hair on the face or body in women and is a sensitive marker for increased androgen action. A male type of hair distribution is usually seen.<sup>4,6,9</sup>

Obesity in women with PCOS is usually truncal, and they tend to have a high waist/hip ratio. It is associated with more severe forms of the disease including extreme anovulation, oligomenorrhea, and increased hirsutism, but non-obese women with PCOS may also present with these features.<sup>4,6,9</sup> There is also insulin resistance in PCOS. Several mechanisms have been proposed for the state of insulin resistance. Peripheral target tissue resistance, decreased hepatic clearance or increased triglycerides and decreased HDL cholesterol levels.<sup>6,10</sup>

Peripheral insulin resistance associated with hyperandrogenism can be due to mutation of the insulin receptor gene. "Luprechaunism" is a rare syndrome in young girls with mutation in the receptor gene. It is associated with severe insulin resistance, polycystic ovaries, and hyperandrogenism and acanthosis nigricans. The ratio of fasting glucose to fasting insulin less than 3.0 defines hyperinsulinemia.<sup>6,10</sup>

Thyroid disorders are associated with menstrual dysfunction and also can have serious adverse impact on pregnancy outcomes and child development. The overall high prevalence of thyroid dysfunction in women warrants specific testing to exclude the diagnosis (serum thyroid-stimulating hormone, TSH) in all anovulatory women, including those with hyperandrogenism, but not for diagnosis of PCOS.<sup>8</sup>

Hyperprolactinemia is highly associated with menstrual dysfunction and is one of the most common causes of secondary amenorrhea.

The many causes of hyperprolactinemia are considered at length elsewhere. Hyperprolactinemia is associated with increased adrenal androgen production in vivo and in

vitro but its prevalence among women who present with hyperandrogenism is quite low and generally <3%.

The high prevalence of hyperprolactinemia among women with menstrual dysfunction justifies specific testing to exclude the diagnosis in all anovulatory women but not for diagnosis of PCOS.<sup>8</sup>

**METHODS**

This hospital based study was conducted at the Department of Obstetrics and Gynecology, Gangori Hospital attached to S.M.S. Medical College, Jaipur, from April 2013 to March 2014. Twenty-five women with PCOS attending Gynecology OPD, Gangori Hospital, Jaipur on first come first basis were selected as cases.

After each case, the consequent woman was selected as control for normal group. In this manner twenty-five controls were selected. Before including in the study, diagnosis was confirmed by history, clinical examination and ultrasonography in all the subjects.

Quantitative data were summarised in form of Mean±SD. The difference in both the study and control groups was analysed using the unpaired 'T' test. The level of significance for all statistical analysis was kept at 95%.

**RESULTS**

Data tabulated in Table 1, shows that there was no significant difference observed in age between PCOS subjects and Control subjects.

**Table 1: Distribution of the study subjects according to age.**

Age (in years)	PCOS subjects		Control subjects		P-value
	No. of Subjects (n=25)	%	No. of Subjects (n=25)	%	
15-20	12	48	7	28	0.266
20-30	12	48	15	60	
>30	1	4	3	12	
Total	25	100	25	100	

**Table 2: Distribution of the study subjects according to their area of residence.**

Area of residence	PCOS subjects		Control subjects		P-value
	No. of Subjects (n = 25)	%	No. of Subjects (n = 25)	%	
Rural	7	28	15	60	0.046
Urban	18	72	10	40	
Total	25	100	25	100	

Table 2, shows that 32% of PCOS subjects and 60% of Control subjects were from rural population. 68% of PCOS subjects and 40% of control subjects were from urban population. There was a statistically significant difference observed in relation to area of residence of subjects.

**Table 3: Distribution of the study subjects according to their socio-economic status.**

Socio-economic status	PCOS subjects		Control subjects		P-value
	No. of Subjects (n = 25)	%	No. of Subjects (n = 25)	%	
Lower	9	38	19	76	0.008
Middle	12	48	6	24	
Upper	4	16	0	0	
Total	25	100	25	100	

Data compiled in Table 3, shows the socio-economic status of subjects according to Modified Kuppaswamy Classification. Socio-economic status of PCOS subjects was statistically significantly higher than control subjects.

**Table 4: Distribution of the study subjects according to hyperandrogenic complaints related to PCOS.**

Hyperandrogenic complaints	PCOS subjects		Control subjects		P-value
	No. of subjects (n=25)	%	No. of subjects (n=25)	%	
Hirsutism	24	96	0	0	<0.001
Acne	12	48	2	8	<0.001
Heavy voice	1	4	0	0	0
Breast atrophy	1	4	0	0	0

Data tabulated in Table 4 shows that hyperandrogenic complaints were significantly more in PCOS subjects as compared to control subjects i.e. hirsutism 96%, acne 42%, heavy voice 4% and breast atrophy 4%.

**Table 5: Distribution of the study subjects according to parity.**

Parity	PCOS subjects		Control subjects		P-Value
	No. of Subjects (n = 25)	%	No. of Subjects (n = 25)	%	
P <sub>0</sub>	2	8	0	0	0.008
P <sub>1</sub>	2	8	0	0	
P <sub>2</sub>	12	48	10	40	
P <sub>3</sub>	9	36	6	24	
≥P <sub>4</sub>	0	0	9	36	
Total	25	100	25	100	

Data compiled Table 5, suggested that parity of PCOS subjects was significantly lower than control subjects.

**Table 6: Distribution of the study subjects according to family history.**

Family history of 1 <sup>st</sup> degree relatives	PCOS subjects		Control subjects	
	No. of Subjects (n = 25)	%	No. of Subjects (n = 25)	%
Metabolic syndrome	1	4	0	0
Ovarian malignancy	1	4	0	0
PCOS	8	32	0	0
No	15	60	25	100
Total	25	100	25	100

None of the 1st degree relatives of control group had significant family history of PCOS and related disorders. While statistically significant family history of PCOS and related disorders was found in PCOS subjects as shown in Table 6.

**Table 7: Distribution of the study subjects according to mean BMI.**

Groups	No. of subjects	BMI (kg/m <sup>2</sup> )	P-value
		Mean±SD	
PCOS subjects	25	32.28±3.285	<0.001
Control subjects	25	24.16±3.412	
Total	50	28.22±5.273	

In PCOS subjects mean BMI was 32.28 kg/m<sup>2</sup>, while in Control subjects it was much lower i.e. 24.16 kg/m<sup>2</sup>.

So, there was a significant difference between mean BMI among PCOS and control subjects as shown in Table 7.

**Table 8: Distribution of the study subjects according to mean waist: hip ratio.**

Groups	No. of subjects	Waist/Hip ratio	P-value
		Mean±SD	
PCOS subjects	25	0.80±0.073	<0.001
Control subjects	25	0.70±0.081	
Total	50	0.75±0.091	

Data tabulated in Table 8, depicts the mean waist: hip ratio of the study population.

Mean waist: hip ratio of PCOS subjects was 0.80 and control subjects was 0.70. Mean waist: hip ratio was significantly higher in PCOS subjects which was statistically significant.

Data tabulated in Table 9, depicts that serum biochemical markers were significantly higher among the PCOS subjects as compared to control subjects, which was statistically significant.

**Table 9: Distribution of the study subjects according to mean waist: hip ratio.**

Variables	Groups	No. of Subjects	Mean±SD	P-value
Fasting blood sugar levels (mg/dl)	PCOS subjects	25	118.72±22.09	<0.001
	Control subjects	25	91.00±13.30	
S. insulin Levels (µIU/ml)	PCOS subjects	25	30.68±7.936	<0.001
	Control subjects	25	13.76±5.174	
S. testosterone levels (mg/dl)	PCOS subjects	25	118.32±94.011	<0.001
	Control subjects	25	24.72±9.053	
S. triglycerides levels (mg%)	PCOS subjects	25	276.72±92.761	<0.001
	Control subjects	25	116.64±19.388	
S.HDL levels (mg%)	PCOS subjects	25	33.84±11.575	<0.001
	Control subjects	25	62.24±10.990	

## DISCUSSION

Twenty-five women with PCOS were included as cases in study on first come first basis and women with normal ovaries just next to each PCOS case were included as control group.

Diagnosis of cases was confirmed by history, clinical examination, relevant bio chemical tests and ultrasonography in all the subjects (Rotterdam criteria). Women with malignancy anywhere in the body, diseases which manifest PCOS secondarily i.e. androgen secreting tumours, congenital adrenal hyperplasia and Cushing syndrome were excluded from the study.

### Diagnosis of PCOS

According to the 2003 Rotterdam Consensus Conference, the revised diagnostic criteria includes any two of the following:

- Oligomenorrhoea / anovulation
- Clinical and or biochemical signs of hyperandrogenism
- Polycystic ovaries on USG
- Exclusion of other etiologies such as congenital adrenal hyperplasia, androgen-secreting tumours and Cushing syndrome.<sup>8,11</sup>

### Laboratory findings

Laboratory testing in the evaluation of a woman suspected to have PCOS includes serum assessment of

- Androgens,
- Gonadotropins,
- Glucose metabolism.

Other ominous pathologies such as ovarian androgen producing tumours and extragonadal sources of hyperandrogenemia should be sealed out.

### Imaging assessment

Pelvic USG (USG Criteria - Presence of 12 or more follicles in either ovary measuring 2 to 9 mm in diameter and / or increased ovarian volume (>10 ml). A single ovary meeting these criteria is sufficient to affix the PCOS diagnosis, three-dimensional ultrasound, MRI and colour Doppler mapping.<sup>11,12</sup>

In our study, it was observed that the patients in the control group belonged mostly to rural area while that in the PCOS group came from urban area. It is not possible to provide an explanation for this, because the urban population is expected to be more informed with better medical facilities.

In this study, most of the members in the control group belonged to lower and middle socio-economic stratum group, while most in the PCOS group belonged to middle and upper class. This was expected as most subjects in control group were from rural area. Since subjects of PCOS group, in general belonged to upper socio-economic class, it may be deduced from this observation that high incidence of this syndrome could be related to their life style.

PCOS group had oligomenorrhoea (100%), and other hyper androgenic problems like hirsutism (96%), acne (48%), heavy voice (4%), and breast atrophy (4%), which was not reported in control group. Similar observations were also reported by Adams J et al.<sup>13</sup> They reported oligomenorrhoea (100%), hirsutism (65%). Infertility (85%) as the main complaints of PCOS patients.

## CONCLUSION

Women from urban population, higher socio-economic status were more in PCOS group compared to controls and this difference was statistically significant. Clinical and biochemical features of hyperandrogenism were marked in PCOS subjects as compared to control subjects. Women with PCOS have a hormonal imbalance and metabolism problems that may affect their health and

fertility, we found PCOS subjects are more in urban population with strong family history. Lifestyle modification and dietary modification helps for the longterm health prognosis. women who are at risk of PCOS to be educated and managed for the continuing health risk and planned potential therapeutic strategies. Futher research has to look into possibility of genes linked into PCOS.

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## REFERENCES

- Adam HB, Joop SE, Seang-Lin T, Dewailly D. Ultrasound assessment of the polycystic ovary : international consensus definitions. *Human Reprod Update.* 2003;9:505-14.
- Fulghesu AM, Ciampelli M, Belosi C, Apa R, Pavone V, Lanzone A. A new ultrasound criterion for the diagnosis of polycystic syndrome: the ovarian stroma / total area ratio. *Fertil Steril.* 2001;76:326-31.
- Marantides D. Management of polycystic ovary syndrome. *Nurse Pract.* 1997;22(12):34-38.
- Takahashi K, Eda Y, Abu Musa A, Okada S, Yoshino K, Kitao M. Transvaginal ultrasound imaging, histopathology and endocrinopathy in patients with polycystic ovarian syndrome. *Human Reprod.* 1994;9:1231-6.
- Abdel Gadir A, Khatim MS, Mowafi RS, Alnasser HM, Muharib NS, Shaw RW. Implications of ultrasonically diagnosed polycystic ovaries. Its corrections with basal hormonal profiles. *Human Reprod.* 1992;7(4):453-7.
- Rumack CM, Wilson BR, Charboneau JW. *Diagnostic ultrasound.* 3<sup>rd</sup> ed. Missouri: Elsevier Mosby; 2005:561-2.
- Tsilchorozidou T, Overton C, Conway GS. The pathophysiology of polycystic ovary syndrome. *Clinical Endocrinol.* 2004;60:1-17.
- Fritz MA, Speroff L. *Clinical Gynaecologic Endocrinology and Infertility. Chronic Anovulation and The Polycystic Ovarian Syndrome.* 8<sup>th</sup> ed. Lippincot: William and Wilkins;2012:475-531.
- Brown DL, Henrichsen TL, Clayton AC, Hudson S, Coddington CC, Vella A. Ovarian stromal hyperthecosis. *J Ultrasound Med.* 2009;28:587-93.
- Nardo LG, Buckett WM, White D, Digesu AG, Franks S, Khullar V. Three dimensional assessment of ultrasound features in women with Clomiphene citrate-resistant polycystic ovarian syndrome (PCOS): ovarian stromal volume does not correlate with biochemical indices. *Human Reprod.* 2002;17:1052-5.
- Berek JS, Berek DL. *Berek and Novak's Gynecology. Endocrinological Disorders.* 8th ed. Lippincot: William and Wilkins;2013:1075-94.
- Mukherjee GG, Chakravarty BN. Polycystic ovary syndrome: an update. *Imaging of the Polycstic Ovary.* 1st ed. Jaypee Brothers;2013:30-36.
- Adams J, Polson DW, Abdul Wahid N, Morris DV, Frank S, Mnson HD et al. Multifollicular Ovaries: clinical and endocrine features and response to pulsatile gonadotropin releasing hormone. *Lancet.* 1985;2:1375-9.
- Choi JH, Gilks CB, Auersperg N, Leung PC. Immunolocalization of gonadotropin-releasing hormone (GnRH)-I, GnRH-II, and type I GnRH receptor during follicular development in the human ovary. *J Clin Endocrinol Metabol.* 2006;91(11):4562-70.
- San-Millán JL, Escobar-Morreale HF. The role of genetic variation in peroxisome proliferator-activated receptors in the polycystic ovary syndrome (PCOS): an original case-control study followed by systematic review and meta-analysis of existing evidence. *Clin Endocrinol.* 2010;72(3):383-92.
- Wood JR, Nelson VL, Ho C, Jansen E, Wang CY, Urbanek M, McAllister JM, Mosselman S, Strauss JF. The molecular phenotype of polycystic ovary syndrome (PCOS) theca cells and new candidate PCOS genes defined by microarray analysis. *J Biol Chem.* 2003;278(29):26380-90.
- Marx TL, Mehta AE. Polycystic ovary syndrome: pathogenesis and treatment over the short and long term. *Cleve Clin J Med.* 2003;70(1):31-3, 36-41, 45.
- Dunaif A. Hyperandrogenic anovulation (PCOS): a unique disorder of insulin action associated with an increased risk of non-insulin-dependent diabetes mellitus. *Am J Med.* 1995;98(suppl 1A):33S-39S.
- Garruti G, de Palo R, Rotelli MT, Nocera S, Totaro I, Nardelli C, Panzarino MA et al. Association between follicular fluid leptin and serum insulin levels in nonoverweight women with polycystic ovary syndrome. *BioMed Res Int.* 2014;2014:980429.
- Tock L, Carneiro G, Pereira AZ, Tufik S, Zanella MT. Adrenocortical production is associated with higher levels of luteinizing hormone in nonobese women with polycystic ovary syndrome. *Int J Endocrinol.* 2014;2014:620605.

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