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POLYCYSTIC OVARIAN SYNDROME : UNDERSTANDING THE ENDOCRINOLOGICAL PARADIGM

KEY WORDS: PCOS, Infertility, LH/FSH, Testosterone.

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ABSTRACT

Endocrine and metabolic dysfunctional pathways of androgen synthesis have all been demonstrated to play a role in the pathophysiology of PCOS. So we did comparative study of endocrinological pattern between PCOS infertile case and non PCOS infertile control. Approximately 50% of all women with PCOS are obese but irrespective of the presence of obesity, these women are frequently insulin resistant. Many hormonal pattern are altered in this metabolic condition responsible for primary infertility and is corroborated with hyper androgenic state as we found in our study. Identification of this clinical state could lead to better clinical outcome regarding infertility and prevention of long term Metabolic and cardiovascular complication.

Conclusion: Polycystic Ovarian syndrome should be interpreted as metabolic syndrome showing the tip of iceberg in context to immediate gynecological and long term metabolic complication.

INTRODUCTION

Polycystic ovary syndrome is a most common endocrinological disorder of women in the reproductive age group; affecting 5-10% of population. It is characterized by menstrual irregularities, biochemical and clinical hyperandrogenism (hirsutism, seborrhea and acne). PCOS is a wonderful state that how different pathways meets to maintain a self perpetuating cycle of endocrine and metabolic dysfunction. This Disordered pathways of androgen synthesis .have all been demonstrated to play a role in the patho physiology of PCOS. Approximately 50% of all women with PCOS are obese but irrespective of the presence of obesity, these women are frequently insulin resistant. Studies indicated that hyperinsulinemia stimulates the ovarian cytochrome P_{450} c17α activity in women with PCOS which play a critical role in the development of hyperandrogenism. [1] Long term complications of PCOS include the development of endometrial cancer, type II diabetes, vascular atherosclerosis and many cardiovascular anomalies. So we aimed to study the endocrinological abnormality in patients of PCOS compare to non PCOS subjects in this part of country.

MATERIAL AND METHODS

The study was carried out in the Department of Obstetrics and Gynecology in collaboration with the Department of Pathology and Department of Medicine, Chhatrapati Shahuji Maharaj Medical University, Lucknow (a tertiary care hospital). A total of 50 diagnosed cases of polycystic ovary syndrome based, according to Rotterdam criteria.[2] Control were 40 non PCOS women seeking infertility treatment due to other causes with no USG and clinical signs suggestive of polycystic ovary syndrome .Hormonal assessment was made during the early follicular phase of menstrual cycle (D2-D5).

Serum luteinizing hormone (LH) ≥10.0 U/L.[3,4]Serum follicular stimulating hormone(FSH) levels, LH/FSH ratio ≥ 2.5, Serum testosterone levels >80 ng/dl was taken as a parameter for the diagnosis of PCOS and hyperandrogenemia .[5,6] After the subjects were selected, 5 ml of blood sample was drawn from the antecubital vein and collected in vacutainer blood collecting tube. Tube was inverted 3 to 4 times and blood allowed to clot. All the specimen were transported to the laboratory within 30 min of collection .Statistics were calculated with the aid SPSS for Windows, Version 16 to find the differences between two groups the independents t-test has been used. For the purpose of this study 95% confidence level has been chosen and corresponding 'p' value <0.05 has been taken as significant.

Observation:

Mean age in cases was 26.10±4.08 and was comparable without statistical difference from controls. (Table-1)

Table-1 Mean age.

Group	Mean Age in yrs	S.D
Case	26.10	4.08
Control	27.01	4.28

Table 2- Pattern of infertility

Infertility	Case		Control		P value
	No	%	No	%	
Primary	38	84.4	31	77.5	0.08
secondary	7	15.6	9	22.5	0.08
Total	45		40		

** Five unmarried subjects in case group .

X_c=0.604, df=1, p= 0.08.(non significant)

Primary infertility in cases was (84.4%) than control (77.5%) .On Pearson Chi test the difference is not significant (p<0.08). (Table-2)

Table3a- Mean value of Hormonal parameters and comparison between case and control..

Biochemical Parameters	Case(n=50) (Mean ± SD)	Control (n=40) (Mean ± SD)	Independent sample test sig(p value)
S.LH(IU/L)	9.78±4.15	5.58±3.16	.000
S.FSH(IU/L)	4.43±1.41	6.19±2.47	.000
S.LH:S.FSH	2.29±.96	0.98±0.60	.000
S.TSH(IU/L)	3.21±1.97	3.50± 2.23	.524
S. Testosterone(ng/dL)	58.53±24.00	33.92±14.15	.000

Serum LH mean value in cases was significantly high (9.78±4.15 Il/L) compared to (5.58±3.16) control group. Serum FSH was significantly low in cases (4.43±1.41) compared to control group (6.19±2.47). S.LH:S.FSH ratio was high in cases of PCOS compared to non PCOS infertile woman. There was no significant difference observed in TSH. Total serum testosterone was significantly high in Cases (58.53±24.00) compared to control (33.92±14.15)..(Table-3a)

Table 3b.: Distribution of subjects according to Hormonal Parameter in PCOS.

Hormonal Parameter		Case No	%	Control No	%	Total No	%	p Value
Serum LH	<10	25	50	36	90	61	67.8	X_c=16.28 , df=1, p<0.001
	≥10	25	50	4	10	29	32.2	
LH/FSH Ratio	<2.5	24	48	39	97.5	63	70	X_c=25.92 , df=1, p<0.001
	≥2.5	26	52	1	2.5	27	30	
Serum Testosterone	<80	29	58	40	100	69	76.7	X_c=21.91 , df=1, p<0.001

Hormonal parameter comparison showed significant result between case and control. High serum LH(≥10) was found in 50% of subjects in case group vs. 10% in control group (p<0.001).High serum LH/FSH was present in 52% cases vs. 2.5% in control group(p<0.001), similarly total serum testosterone was more than 80 ng/dl in 42% of case compared to none in control group(p,0.001).(Table 3b)

DISCUSSION:

Polycystic ovarian syndrome is a heterogeneous disorder without a simple definition as a clinical triad of hyperandrogenism, anovulation and obesity in women with enlarged polycystic ovaries. It is one of the most important causes of hyper androgenic chronic anovulation and infertility. It accounts for 75% cases of anovulatory infertility, occurring in 30-40% of women with secondary amenorrhea and 85-90% with oligomenorrhea , most of subjects are hyper androgenic.[3] The long term squeal of PCOS includes the development of endometrial carcinoma, type II diabetes and cardiovascular anomalies. Recently the role of intraovarian growth factors, neuropeptides and cytokines as autocrine and paracrine regulators in the development of polycystic ovary is much in talk[7] , This high Chronic LH stimulation in PCOS induces sustained hypersecretion of androgens of theca compartment leading to hyperinsulinemia ,it contribute to the development of abdominal obesity .[9] Mean age in cases was 26.10+_4.08yrs and was comparable without statistical difference from controls. In this study the primary infertility in cases were (84.4%) and control (77.5%) was not statistically significant.Abnormal menstrual pattern was found in considerably higher number of PCOS's patients (80%), hirsutism was present in (56%) of cases and significantly higher than control (p<0.001).The hormonal panel abnormalities like elevated serum LH, LH/FSH ratio, and testosterone were consistent with polycystic ovarian syndrome and our findings are consistent with previous studies.[3,5,9] All parameter were significantly raised in case group when compared to control group comparable to earlier study. [5,9] Weight loss is associated with an impressive rate of resumption of ovulation and pregnancy. [10] Low dose oral contraceptive pills can be used safely in anovulatory, hyper androgenic, hyperinsulinemic patients.[11] Patients resistant to oral contraceptive treatment may require suppression with a GnRH agonist. Few studies indicate that both obese and non obese PCOS with hyperinsulinemia respond to metformin treatment. [1]

Conclusion:

Infertility associated with PCOS has been attributed to many factors including anovulation, abnormal gonadotrophin secretion, elevated serum androgens, and dysfunction of ovarian growth factors and their binding proteins. We probe to compare hormonal pattern between patient of PCOS and non PCOS subject coming to infertility clinic. It may be recommended that when PCOS is diagnosed, other metabolic complications should be investigated. Gynecologists should aim to identify short-term problems of PCOS, i.e. reproductive failure, without neglecting the serious, long term complications, i.e. cardiovascular and metabolic derangements. Further studies on the effect of insulin sensitizers and their therapeutic implications are required.

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