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A study of hormonal and metabolic imbalance in polycystic ovarian syndrome (PCOD) women of Middle Gujarat, India

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Abstract

Background: Polycystic ovary syndrome (PCOS) is now recognized as an important metabolic as well as reproductive disorder conferring substantially increased risk for type 2 diabetes. Affected women have marked insulin resistance, independent of obesity.

Objectives: The aim is to study hormonal and Metabolic imbalance in polycystic ovarian syndrome (PCOD)women of Middle Gujarat, India

Methodology: This study includes total 200 female participants of age Group between 20-40 year of age**.** They were divided in to two group. Group 1(n=100) includes women having PCOD and Group 2(n=100) is control Group. Fasting Blood samples were obtained from all participants to measure Blood sugar, Lipid Profile insulin, HOMA-IR, Testosterone**,** Estrogens and Progesterone.

Result: The Mean level of Fasting Blood sugar, S.cholesterol, S.triglyceride S.insulin, S. testosterone .S.Estradiol, S.Progesteron and HOMA-IR is found to be higher in PCOD group as compared to control group and difference among them found to be statically significant.

Conclusion: The association between insulin resistance and PCOS has led to the discovery that insulin is an important reproductive hormone and that insulin signaling in the CNS is critical for ovulation. Hyperandrogenism represents a chief attribute of PCOS as elevated androgen levels are the most constant feature, with the majority (−60%) of patients exhibiting hyperandrogenism.

Keywords: HOMA-IR, PCOD ,insulin, testosterone, Estrogen, Progesteron

Introduction

Polycystic ovary syndrome is the most common endocrinopathy among reproductive-aged women in the United States, affecting approximately 7% of female patients.[1] Although the pathophysiology of the syndrome is complex and there is no single defect from

which it is known to result, it is hypothesized that insulin resistance is a key factor. Metabolic syndrome is twice as common in patients with polycystic ovary syndrome compared with the general population, and patients with polycystic ovary syndrome are four times more likely than the general population to develop type 2 diabetes mellitus. [2,3]

There is general agreement that obese women with PCOS are insulin resistant, but some groups of lean affected women may have normal insulin sensitivity. There is a post-binding defect in receptor signaling likely due to increased receptor and insulin receptor substrate-1 serine phosphorylation that selectively affects metabolic but not mitogenic pathways in classic insulin target tissues and in the ovary. Constitutive activation of serine kinases in the MAPK-ERK pathway may contribute to resistance to insulin's metabolic actions in skeletal muscle. [4] Insulin functions as a co-gonadotropin through its cognate receptor to modulate ovarian steroidogenesis. Genetic disruption of insulin signaling in the brain has indicated that this pathway is important for ovulation and body weight regulation. These insights have been directly translated into a novel therapy for PCOS with insulin-sensitizing drugs. [5]

High levels of estrogen are known as estrogen dominance and can occur in women with polycystic ovary syndrome (PCOS). PCOS[3] is a hormone imbalance that can cause irregular periods, unwanted hair growth, and acne. It is characterized by multiple fluid-filled, cyst-like sacs on the ovaries. [6]

PCOS is the most common cause of ovulatory infertility and it's believed that estrogen dominance plays a part.[4] Lack of ovulation results in continuous high levels of estrogen and insufficient progesterone. [7,8]

Unopposed by progesterone, constant estrogen exposure may cause the endometrium to become excessively thickened, which can lead to heavy and/or irregular bleeding (dysfunctional or anovulatory uterine bleeding)

Therefore, the present investigations will be carried out to assess estrogen, progesterone, estrogen-dominance, Testosterone, insulin level and HOMA-IR level. Subsequently regular assessing of sugar glucose, lipid profile is important to monitor & study the effect of these parameters among normal and polycystic ovary Syndrome (PCOS) women & its adverse consequences

Methodology

This prospective study was conducted on 100 patients of PCOS both suspected as well as already diagnosed at department of obstetrics and gynecology kailash cancer hospital and Research center, Goraj,Vadodara,Gujarat from 2012-2013.

A total of 200 subjects of age group between 20-40 years belonging to both normal & polycystic ovary syndrome will be classified as:

Group-1: 100 women with PCOD (Cases) of polycystic ovary disease will be taken.

Group-2: 100 normal women will be taken as control for these parameters.

All PCOD women & controls were underwent a complete history and physical examination. Women with PCOD should be interviewed of their name, address, age, socio-economic status, menstrual history, age of menarche, education level and family history of PCOD. All women were gone through gynaecological ultrasonography to determine their uterus and ovaried condition.

Inclusion criteria

Women with PCOD are attending outdoor OPD of the hospital, first time diagnosed PCOD, Diagnosed

polycystic ovarian syndrome, age ranging from 20-40 years.

Women with PCOD Willing to have physical examinations like Weight, Height, BMI, W/H ratio, Blood Pressure, Hirsutism, Acne, Dark patches, Virilization, Ultra sonography etc.

Polycystic ovary syndrome (PCOS) associated with Diabetes, obesity, Cardiovascular disorders Irregular menstrual disorder and anovulation, Hirsutism & Acne symptoms.

Exclusion criteria

Women with diagnosed adrenal hyperplasia, androgen secreting neoplasm, other pituitary (acromegaly) and adrenal disorders like Cushing syndrome, Virilizing adrenal or ovarian neoplasm, hyper Prolactinemia and other infertility cause**,** Thyroid hormone related infertility, Women having history of smoking, taking alcohol or tobacco chewing**,** Any other type of gynaecologic complications except related with Polycystic ovary syndrome (PCOS) will be excluded from the study.

Fasting 10 ml Venous blood samples were obtained from all participants and collected it in to fluoride and plain vaccutainer. An Uniq ID number was given to each sample to hidden the identity of participants. All samples were centrifugated at 3000 RPM for a period of 10 minutes to obtained a Plasma and serum.

-Blood Glucose (FBS) measured by Glucokinase method and lipid profile (S.cholesterol, Triglyceride, HDL,VLDL, LDL) measured by colorimetric method from all samples at central laboratory of our hospital.

-Estimation of insulin done by chemilumnescence immunoassay(CLIA)method and HOMA-IR will be estimated by calculation (fasting sugar×fasting insulin/22.5).

-Various Endocrinal Hormones like Estrogen(E2), progesterone, estrogen-dominance, Testosterone and Insulin was Measured by chemilumnescence immunoassay(CLIA) method from all samples.

After assessing all the values, Mean, Standard deviation of all subjects & parameters were analysed. Statistical analysis was performed with SPSS software. Comparison between cases and with control is done by using online t-test calculator. p value less than 0.05 (P value < 0.05), is consider as a difference of significant.

Results & Discussion

Study includes total 200 particpants among them n=100 PCOD (Group 1) cases and n=100 control(Group 2). Mean age of participants showing in Table 1.

Table 1: Age wise distribution of participants

Group	Number(n)	Mean Age (Yr)
Group 1(PCOD)	100	26 ±5.4
Group 2(Control	100	27.5 ±4.5

Table 2: Comparison of weight between case and control group

Group	Number(n)	Mean wt (kg)
Group 1(PCOD)	100	55.80± 6.24
Group 2(Control)	100	44.36 ± 5.8

Table 3: Location wise distribution of participants

Location	Group 1(PCOD)	Group 2(Control)
Rural	40(40%)	36(36%)
Urban	60(60%)	64(64%)
Total	100(100%)	100(100%)

Table 4: Comparison of waste hip (W/H) ratio between case and control group

Group	Number(n)	Mean W/H ratio
Group 1(PCOD)	100	0.91 ± 0.13
Group 2(Control)	100	0.81 ± 0.0.3

Table 5: Comparison of Marital status between case and control group

Group	Number(n)	Married	Single
Group 1(PCOD)	100	66(66%)	34(34%)
Group 2(Control)	100	67 (67%)	33(33%)

Table 6: Comparison of BMI between case and control group

Group	Number(n)	Mean BMI
Group 1(PCOD)	100	24.50 ± 3.50
Group 2(Control)	100	19.72 ± 2.41

Table 7: Comparison based on menstrual cycle history between case and control group

			Group		Total
			Case	Control	
M/H cycle (day)	< 5	count	8(8%)	0(0%)	08(4%)
	5-10	count	90(90%)	3(3%)	93(46.5%)
	>10	count	2(2%)	97(97%)	99(49.5%)
Total		count	100(100%)	100(100%)	200(100%)

Table 8: Comparison of systolic Blood pressure (SBP) between case and control group

			Group		Total
			Case	Control	
SBP(mmhg)	100-130	count	47(47%)	90(90%)	137(68.5%)
	131-150	count	43(43%)	8(8%)	51(26%)
	>150	count	10(10%)	2(2%)	12(5.5%)
Total		count	100(100%)	100(100%)	200(100%)

Table 9: Comparison of diastolic Blood pressure (SBP) between case and control group

			Group		Total
			Case	Control	
DBP(mmhg)	<80	count	47(47%)	80(80%)	127(63.5%)
	81-100	count	46(46%)	20(20%)	66(33%)
	>100	count	7(7%)	0(0%)	7(3.5%)
Total		count	100(100%)	100(100%)	200(100%)

Table 10: Showing valid Hirsutism status of Case group

Total count	Hirsutism		Non Hirsutism	
	Counts	Valid%	Counts	Valid%
Cases (100)	82	82%	18	18%

Table 11: Comparison of various biochemical parameters between case and control group

Parameter	Group	Number(N)	Mean ± SD	P value
FBS (mg/dl)	Case	100	105±5	<0.001
	Control	100	99±6	
S. Triglyceride (mg/dl	Case	100	166.5±10.5	0.052
	Control	100	158.6±9.5	
S. cholesterol (mg/dl)	Case	100	192.6±4	<0.001
	Control	100	165±10.5	
S.HDL (mg/dl)	Case	100	48.15±5.3	0.062
	Control	100	41.25±4.5	
S.LDL (mg/dl)	Case	100	111.15±6	<0.001
	Control	100	92.03±5	
S.VLDL (mg/dl)	Case	100	33.3±5.5	0.031
	Control	100	31.72±6	

- Comparison of the fasting basal sugar (FBS) between the two groups shows that FBS is higher (mean value = 105 ± 5) in Cases group than Controls (mean value = 99 ± 6)(Table 11)

- Comparison of the Triglyceride (TG) between two groups shows that TG is higher (mean value = 166.5 ± 10.5) than Controls (mean value = 158.6 ± 9.5). Comparison of Total Cholesterol (TC) between two groups shows that TC is higher (mean value = 192.6 ± 4) in Cases than Controls.(Table 11)

Table 12: Comparison of level of various endocrinal hormonal statuses between case and control group

Parameter	Group	Number(N)	Mean ± SD	P value
S. Insulin(U/ML)	Case	100	15.52 ± 1.5	<0.001
	Control	100	8.50 ±3.5	
HOMA-IR	Case	100	3.95	<0.001
	Control	100	2.06	
S. Testosterone(ng/ml)	Case	300	13.1 ± 2.36	<0.001
	Control	300	3.55 ± 1.50	

S. Estradiol(pg/ml)	Case	300	231.5 ± 50.13	<0.001
	Control	300	74.56 ± 35.5	
S. Progesteron(ng/ml)	Case	300	1.72 ± 2.26	<0.001
	Control	300	2.58 ± 1.13	

- HOMA-IR is higher (mean value 3.95) in Cases than Controls (mean value2.06). (Table 12)

-Testosterone is higher (mean value 13.1 ± 2.36) in Cases than Controls (mean value = 3.55 ± 1.50). (Table 12)

- Insulin hormone is higher (mean value 15.52 ± 2.0) in Cases than Controls (mean value = 8.50 ± 3.5). (Table 12)

- Estradiol is higher (mean value 231.5 ± 50.13) in Cases than Controls (mean value = 74.56 ± 35.5). (Table 12)

- Progesterone is higher (mean value 2.58 ± 1.13) in Controls than Cases (mean value = 1.72 ± 2.26). (Table 12)

Fig 1: Showing Correlation of insulin & HOMA-IR between case and control group

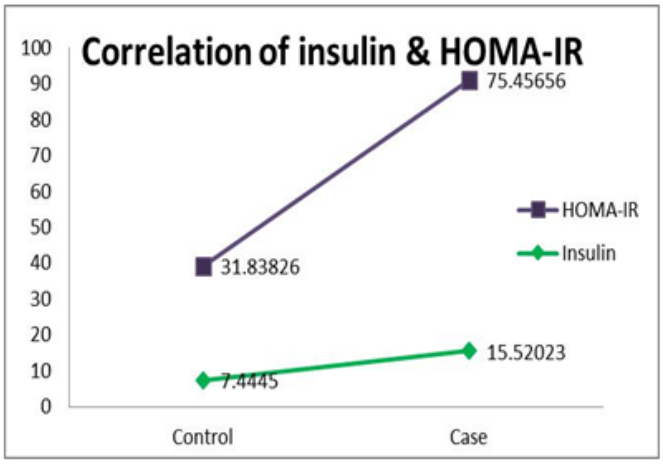


Fig 2: Showing correlation of testoterone & insulin between case and control group

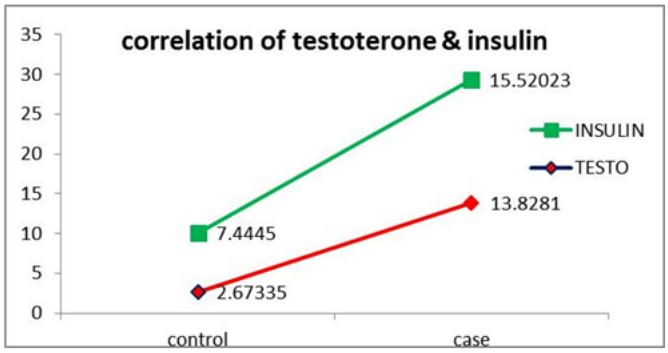


Fig 3: Showing correlation of estradiol & Testosterone between case and control group

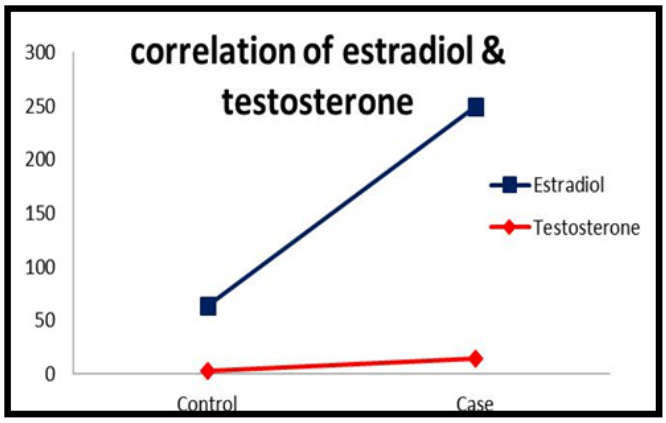
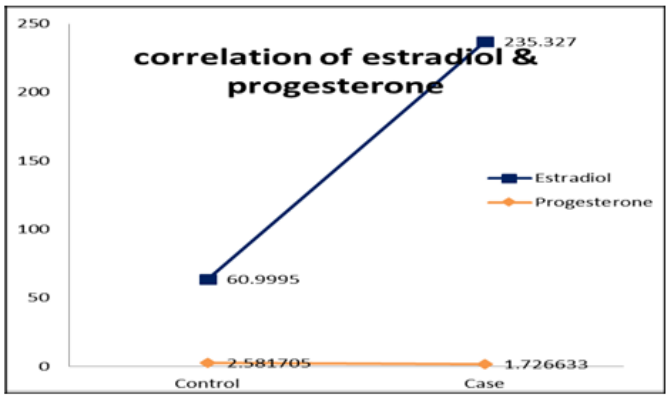


Fig 4: Showing correlation of estradiol & progesterone between case and control group



Women with hyperandrogenic PCOS present with elevated levels of various androgens, including testosterone (T) and the pro-androgens androstenedione (A_4) and dehydroepiandrosterone sulfate (DHEAS), as well as the enzyme required to convert pro-androgens to bioactive androgens, 3β-hydroxysteroid dehydrogenase (3β-HSD) in serum [9,10]. Excess androgens can be

induced by insulin resistance and hyperinsulinemia, as they cause a reduction in sex hormone binding globulin levels, which lead to a subsequent increase in free androgens and unfavourable metabolic profiles[11,12]. The ovarian PCOS morphological traits of enlarged, multi-cystic ovaries and theca interstitial hyperplasia are reported in women who are subjected to high levels of androgens as a result of endogenous adrenal androgen hypersecretion in congenital adrenal hyperplasia[13], or exogenous testosterone treatment in female-to-male transsexuals[14]. Additionally, cultured human theca interna cells removed from PCOS ovaries exhibit higher androgen secretion that continues during long-term culture [15]. These observations corroborate a role for androgens in the acquisition of the PCOS ovarian features.

Conclusion

The association between insulin resistance and PCOS has led to the discovery that insulin is an important reproductive hormone and that insulin signaling in the CNS is critical for ovulation. Hyperandrogenism represents a chief attribute of PCOS as elevated androgen levels are the most constant feature, with the majority (−60%) of patients exhibiting hyperandrogenism.

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