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Study of Level of various Infertility hormones like FSH, LH, Testosterone, Thyroid hormone and Prolactin in obese hyperglycemic and non-obese normoglycemic women of polycystic ovarian syndrome (PCOD) in southern Rajasthan, India

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Abstract: The polycystic ovary syndrome (PCOS) is a mostly hyper androgenic disorder and is possibly the most common endocrinopathy of premenopausal women. The primary defect in polycystic ovary syndrome (PCOS) appears to be an exaggerated androgen synthesis (testosterone) (secretion by the ovaries and the adrenal glands) and hyperglycemia or insulin resistance. The objective of the study is to study the level of various Infertility hormones like FSH, LH, Progesterone, Estradiol, Testosterone, Insulin, HOMA-IR, Thyroid hormone (T3,T4,TSH)& Prolactin and in obese hyperglycemic and non-obese normoglycemic women of Polycystic ovarian syndrome(PCOD). This study includes total 500 female participants of age Group between 18-40 year of age**.** They were divided in to two group. Group 1(n=300) includes women having PCOD and Group 2(n=200) is control Group. Fasting Blood samples were obtained from all participants to measure Blood sugar, Lipid Profile, Testosterone, Progesterone, Estradiol FSH, LH, Insulin, HOMA-IR, Thyroid hormone(T3,T4,TSH) and Prolactin. History of PCOD women also had taken with Age, Menstrual cycle, hirsutism, acne, BMI, Waiste Hip ratio & Veg or Occasional Non-veg food diets. The Mean level of Fasting Blood sugar, S. cholesterol, S.Triglyceride, S. Insulin, S. Testosterone, S. Estradiol ,S.FSH ,and S.LH is found to be higher in both obese hyperglycemic & non-obese normoglycemic PCOD group (Except: S. Progesterone) as compared to control group and difference among them found to be statically significant. In PCOD women were also finds symptoms of oligomenorrhoea or anovulatory menstrual cycle, histustim with different F-G score, obesity in different ratio. From our study I would like to conclude that Obesity is a common finding in PCOS and aggravates many of its reproductive and metabolic features. The relationship between PCOS and obesity is complex, not well understood, and most likely involves interaction of genetic and environmental factors. Insulin resistance and weight gain are two contributing factors to PCOS. Insulin resistance typically causes the body to produce more insulin than normal (hyperinsulinemia). Higher levels of insulin can then cause ovaries to produce too much testosterone which can impair normal ovulation from occurring. Hyperandrogenism caused other secondary characteristics like Virilization, hirsutism (hairs on body), acne, obesity etc. These symptoms differ according to age of PCOD women, early age hyperandrogenism which further leads to metabolic syndrome with insulin resistance in later age.
Keywords: PCOD, Testosterone, Insuline, FSH, LH ,Prolactin, Progesterone, Estradiol, Thyroid hormone

INTRODUCTION

In polycystic ovary Syndrome (PCOS), increased androgen production results in high levels of luteinizing hormone (LH) and low levels of follicle-stimulating hormone (FSH), so that follicles are prevented from producing a mature egg. Without egg

production, the follicles swell with fluid and form into cysts. Every time an egg is trapped within the follicle, another cyst forms and the ovary swells, sometimes reaching the size of a grapefruit. Without ovulation, progesterone is no longer produced, whereas estrogen levels remain normal [1].

Factors that cause PCOS are insulin resistance & obesity, Genetic tendency, bad dietary habbits, weakened immune system, accumulation of toxins etc. In PCOS while diabetes mellitus and impaired glucose tolerance are easily diagnosed, the diagnosis of and concern for insulin resistance as a precursor disorder is underappreciated [2, 3].

Polycystic ovarian syndrome (PCOS) is a highly prevalent hormonal and metabolic disorder among reproductive aged women worldwide. Women with polycystic ovarian syndrome (PCOS) have widely varying phenotypes and seek medical care for differing reasons. In addition to concern for menstrual cycle function, ovulation, hirsutism and acne, many polycystic ovarian syndrome (PCOS) women have abnormal glucose metabolism.

MATERIAL & METHOD

This prospective study was conducted at Department of Biochemistry and Department of Obstetrics & Gynaecology, RNT Medical college and associated group of hospital, Udaipur, Rajasthan, India from June 2012-Dec 2013.

A total of 500 subjects of age group between 18-40 years belonging to both normal & polycystic ovary syndrome will be classified as:

Group-1:300 women with PCOD (Cases) of polycystic ovary disease will be taken.
Group-2:200 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, and menstrual history, age of menarche, education level and family history of PCOD. All women were gone through gynaecological ultrasonography to determine their uterus and ovary condition.

Inclusion criteria

Women with PCOD are attending outdoor OPD of the hospital, first time diagnosed PCOD ,Diagnosed polycystic ovarian syndrome, age ranging from 18-40 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**,** hyperProlactinemia 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. A Uniq ID number was given to each sample to hidden the identity of participants. All samples were centrifugated at 3000 RPM at clinical biochemistry laboratory for a period of 10 minutes to obtain a Plasma and serum.

Blood Glucose (FBS) measured by GOD POD method and lipid profile (S.Cholesterol, Triglyceride, HDL, VLDL, LDL) measured by enzymatic colorimetric method from all samples.

Various Endocrinal Hormones like, Testosterone, LH, FSH, Estradiol, and Progesterone was measured by enzyme linked immune assay (ELISA) method based on electrochemilumnescence from all samples. Thyroid hormone and Prolactin was done only for case group (exlusion criteria).

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 independent student's t test. By using' values now P value is less than 0.05 (P value < 0.05), it is significant. Comparison of the categorical variables (among category comparison) was done by using Chi-Square test.

RESULTS & DISCUSSION

The present study has been conducted on 300 PCOD women attending in OPD of R.N.T. medical college & associated group of hospitals, Udaipur and Geetanjali medical college & hospital, Udaipur. The results have been compared with 200 age and matched health women. All subjects were taken in the age group of fertile age 18-40 years.

Clinical data of all groups were collected via a questionnaire supplemented with an interview & were subjected to:

1. Verbal consent has been taken from patient after explaining the aim of the study.
2. **History**
   - Marital status or single
   - Education - awareness about PCOD & its consequences.
   - Socioeconomic status- life style & diet (veg/non-veg/fastfood/junk food) behaviour of subjects
   - Menstrual history- to determine the period of cycle relate with PCOD symptoms
   - Past history-to know the no of children and any type of history of Diabetes, Hypertension
   - Family history- family history of poly cystic ovary disease.
   - Hirsutism- to examine for excessive male hair pattern in females.

3. **General Examination**
   - Blood pressure (systolic and Diastolic) – measured in semi recumbent position. Two blood pressure recording 4 hours apart have been obtained.
   - Body mass index (BMI) – equals to weight in kilograms devided by th square of the height in meters.

     BMI = (Weight in KG) / (Height in meters)^2

   - Abdominal examination – to determine any metabolic syndrome and inflammation.
   - Hirsuism examination (F-G score) - When evaluating a woman with hirsutism, the Ferriman–Gallwey (FG) score is a simple and commonly used method to quantify hair growth. This method evaluates nine androgen sensitive sites and grades them from 0 to 4. Scores between 8 and 15 are usually considered to be mild hirsutism, whereas scores greater than 25 indicate severe hirsutism. Some limitations of this scoring system include: (a) the variation in hair growth between different ethnic groups; (b) failure to account for regional hirsutism; and (c) the fact that many women may have treated their excessive hair growth with cosmetic measures, such as chemical depilatories, electrolysis, laser therapy, etc.

Ferriman-Gallway (F-G) score

This is the established and most prevalent system of measuring hirsutism. It was introduced in 1961. The scorecard of every body part analyzed began from 0 (zero terminal hairs) to 4 (massive terminal hair growth or frankly hirsute) and the numbers are added up to a maximum count of 36. The survey also suggested that hair production over the forearm and lower leg were less androgen receptive. This was an important finding since androgen is the main hormone that determines terminal hair production and its dysfunction is one of the main causes of hirsutism. Excluding the other nine body parts, 4.3% of patients evaluated registered a count of greater than 7. Hence, the experts conducting the study concluded that a score of 8 or more suggests hirsutism. Later there have been various modifications of the method. Some have been based on the analysis of hair production preferentially in the sideburn region, lower jaw, and upper neck, or perineal region and this has led to new measurement processes. Based on this score pattern and other clinical tests, hirsutism can be evaluated as mild, moderate or severe. DM Ferriman, JD Gallwey[4].

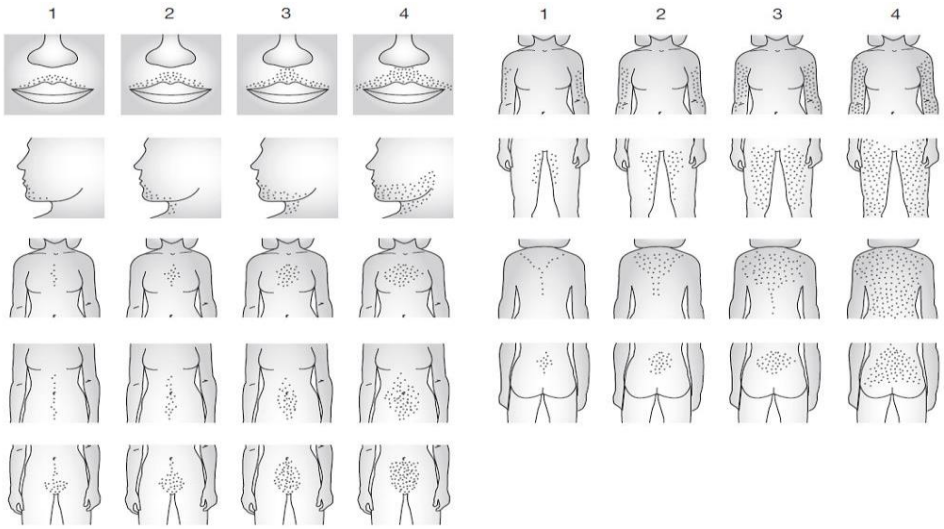


Figure 2 The modified Ferriman–Gallwey scoring system for hirsutism. Each of the nine body areas is rated from 0 (absence of terminal hairs) to 4 (extensive terminal hair growth) and the numbers in each area are added to obtain the total score. A score ≥6–8 generally defines hirsutism. Permission obtained from Humana Press © Azziz R *et al.* (2006) *Androgen Excess Disorders in Women: Polycystic Ovary Syndrome and Other Disorders*, edn 2. Totowa, NJ: Human Press.

Fig: The Modified Ferriman- Gallway scoring system [4]

Gynecological Ultrasonography

To determine condition of uterus and ovary whether normal or immature follicles as a form of poly cyst, their numbers, size and volume, specifically looking for small ovarian follicles. These are believed to be the result of disturbed ovarian function with failed ovulation, reflected by the infrequent or absent menstruation that is typical of the condition. In a normal menstrual cycle, one egg is released from a dominant follicle - essentially a cyst that bursts to release the egg. After ovulation the follicle remnant is transformed into a progesterone-producing corpus luteum, which shrinks and disappears after approximately 12–14 days.

Table–1: Age wise distribution of participants

			GROUP		Total
			Control	Cases	
AGE (in years)	<20	Count	25	38	63
		% within GROUP	12.5%	12.7%	12.6%
	21-30	Count	158	185	343
		% within GROUP	79.0%	61.7%	68.6%
	31-40	Count	17	77	94
		% within GROUP	8.5%	25.7%	18.8%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

Table–2: Comparison of weight between case and control group

			GROUP		Total
			Control	Cases	
WEIGHT	<55 KG	Count	189	130	319
		% within GROUP	94.5%	43.3%	63.8%
	56-65 KG	Count	11	121	132
		% within GROUP	5.5%	40.3%	26.4%
	66-86 KG	Count	0	49	49
		% within GROUP	0.0%	16.3%	9.8%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

Table−3: Comparison of waste hip(W/H) ratio between case and control group

			GROUP		Total
			Control	Cases	
W/H RATIO	<0.85	Count	191	94	285
		% within GROUP	95.5%	31.3%	57.0%
	0.86-0.95	Count	9	172	181
		% within GROUP	4.5%	57.3%	36.2%
	>0.95	Count	0	34	34
		% within GROUP	0.0%	11.3%	6.8%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

Table−4: Comparison of BMI between case and control group

			GROUP		**Total**
			Control	**Cases**	
BMI	<25	Count	200	244	444
		% within GROUP	100.0%	81.3%	88.8%
	26-30	Count	0	41	41
		% within GROUP	0.0%	13.7%	8.2%
	>30	Count	0	15	15
		% within GROUP	0.0%	5.0%	3.0%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

Table−4A: Distribution of obese participants based on BMI

			GROUP		**Total**
			Control	Cases	
OBESE	CLI. OB.	Count	0	16	16
		% within GROUP	0.0%	5.3%	3.2%
	MOR.OB.	Count	0	3	3
		% within GROUP	0.0%	1.0%	0.6%
	NO	Count	200	240	440
		% within GROUP	100.0%	80.0%	88.0%
	OBESE	Count	0	41	41
		% within GROUP	0.0%	13.7%	8.2%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

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

			GROUP		**Total**
			Control	Cases	
M.STATUS	M	Count	57	122	179
		% within GROUP	28.5%	40.7%	35.8%
	M*	Count	43	65	108
		% within GROUP	21.5%	21.7%	21.6%
	M**	Count	16	10	26
		% within GROUP	8.0%	3.3%	5.2%
	M***	Count	2	3	5
		% within GROUP	1.0%	1.0%	1.0%
	S	Count	82	100	182
		% within GROUP	41.0%	33.3%	36.4%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

(M: Married,M*: Married having one child,M**: Married having two child,M***: Married having three child)

Table−6: Comparison based on menstrual cycle history between case and control group

			GROUP		Total
			Control	Cases	
M.H./CYCLE	<5	Count	0	22	22
		% within GROUP	0.0%	7.3%	4.4%
	5-9	Count	0	277	277
		% within GROUP	0.0%	92.3%	55.4%
	>=10	Count	200	1	201
		% within GROUP	100.0%	0.3%	40.2%
Total		Count	200	300	500
		% within GROUP	100.0%	100.0%	100.0%

Table−7: Showing Hirsutism status of Case group

TOTAL COUNTS	HIRSUTISM		NON HIRSUTISM	
	Counts	valid %	Counts	valid %
Cases (300)	160	53%	140	47%

Table−8: Comparison of various biochemical parameters between case and control group

parameter	Group	N	Mean ± SD	p-value
FBS(mg/dl)	Case	300	106.7 ± 19.4	<0.001
	Control	200	96.12± 17.03	
S.choletsterol(mg/dl)	Case	300	189.1± 45.47	<0.001
	Control	200	157.49± 23.80	
S.Triglyceride(mg/dl)	Case	300	160.69 ± 36.98	0.025
	Control	200	154.62 ± 23.42	
S.HDL(mg/dl)	Case	300	40.24 ± 6.30	0.006
	Control	200	38.66± 6.25	
S.LDL(mg/dl)	Case	300	116.95± 42	<0.001
	Control	200	87.98 ± 22.27	
S.VLDL(mg/dl)	Case	300	32.0 ± 7.32	0.032
	Control	200	30.84 ± 4.72	

Table−9: Comparison of level of various endocrinal hormonal status between case and control group

parameter	Group	N	Mean SD	p-value
S.LH(µIU/ml)	Case	300	147.12 ± 39.13	<0.001
	Control	200	90.86 ± 43.62	
S.FSH(µIU/ml)	Case	300	76.42 ± 45.67	<0.001
	Control	200	22.22 ± 17.11	
S. Testosterone(ng/ml)	Case	300	13.82 ± 6.38	<0.001
	Control	200	2.67 ± 1.48	
S.Estradiol(pg/ml)	Case	300	235.3±90.29	<0.001
	Control	200	60.9±19.69	
S. Progesteron(ng/ml)	Case	300	2.58±3.13	0.001
	Control	200	1.72±2.26	
S. Insulin(U/ML)	Case	300	15.52 ± 6.29	<0.001
	Control	200	7.44± 2.04	
HOMA IR	Case	300	75.45± 41.15	<0.001
	Control	200	31.83± 10.69	

Table−10: Results of thyroid hormone and prolactin in case group

Parameter	Number(n)	Result(Mean±SD)
S.TSH(µIU/ml)	300	1.22±6.8
S.T3(nmol/l)	300	2.18±0.57
S.T4(nmol/l)	300	134.06±30.53
S.Prolactin(µIU/Ml)	300	228.08±11.5

Table−11: Comparison between 'vegeterian diets with fastfood(FF) eaters & nonvegetarian diets with fast food eaters' in hyperglycemic cases (Total: 74 / 300 Cases)

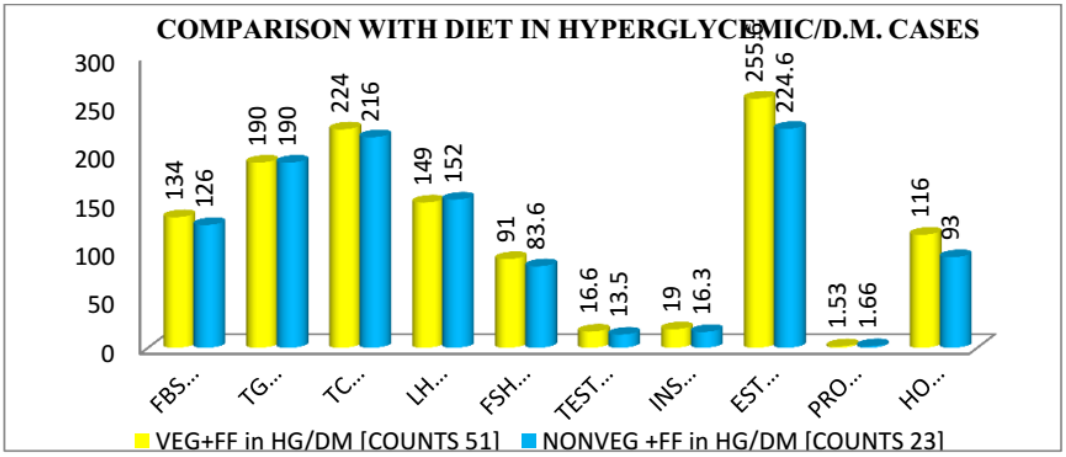
S.NO.	COUNTS	51	23
	PARAMETERS	Veg + FF in HG/DM Mean	Nonveg + FF in HG/DM Mean
1.	Fasting Blood sugar (mg/dl)	134	126
2.	UREA (mg/dl)	25	24.7
3.	CREATININE (mg/dl)	0.6	0.57
4.	TG (mg/dl)	190	190
5.	TC (mg/dl)	224	216
6.	HDL (mg/dl)	39	38
7.	LDL (mg/dl)	148	140
8.	VLDL (mg/dl)	38	38
9.	LH (µIU/ml)	149	152
10.	FSH (µIU/ml)	91	83.6
11.	TESTOSTERONE(ng/ml)	16.6	13.5
12.	INSULIN(U/ml)	19	16.3
13.	ESTROGEN (pg/ml)	255.6	224.6
14.	PROGESTERONE(ng/ml)	1.53	1.66
15.	HOMA-IR	116	93

Table−12: Hyperglycemic /Diabetes mellitus case with obesity and without obesity (Total: 74 / 300 Cases)

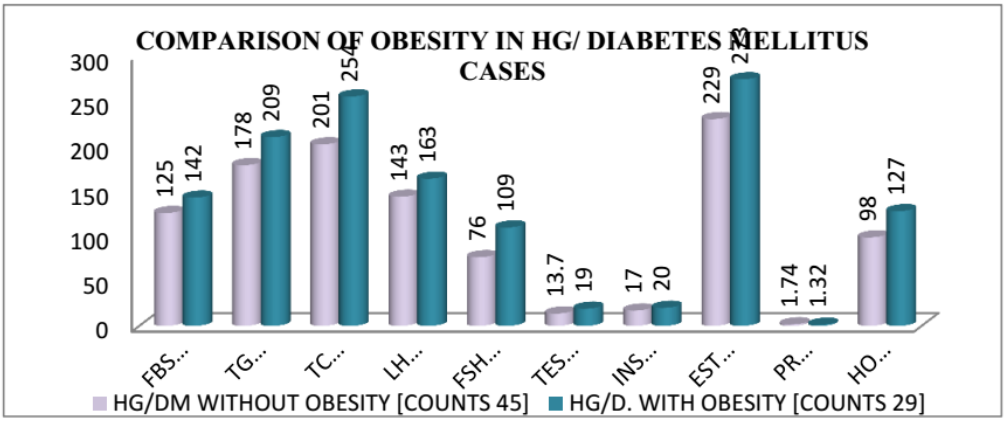
S.NO.	COUNTS	45	29
	PARAMETERS	HG/DM WITHOUT OBESITY Mean	HG/DM WITH OBESITY Mean
1.	Fasting Blood sugar (mg/dl)	125	142
2.	UREA(mg/dl)	24	26
3.	CREATININE(mg/dl)	0.58	0.62
4.	TG (mg/dl)	178	209
5.	TC (mg/dl)	201	254
6.	HDL (mg/dl)	41	37
7.	LDL (mg/dl)	126	176
8.	VLDL (mg/dl)	35	42
9.	LH (µIU/ml)	143	163
10.	FSH (µIU/ml)	76	109
11.	TESTOSTERONE(ng/ml)	13.7	19
12.	INSULIN (U/ml)	17	20
13.	ESTROGEN (pg/ml)	229	273
14.	PROGESTERONE(ng/ml)	1.74	1.32
15.	HOMA-IR	98	127

Table−13: Comparison of different groups of 'oligomennrohea (o/m), o/m with & without Hirsutism' among case (TOTAL: 268/300 CASES)

S.NO.	COUNTS	268	148	120
	PARAMETERS	OLIGOMENNROHEA (OM) Mean	OM WITH HIRSUTISM Mean	OM WITHOUT HIRSUTISM Mean
1.	Fasting Blood sugar(mg/dl)	107	109	105
2.	UREA (mg/dl)	25	25	25
3.	CREATININE(mg/dl)	0.58	0.57	0.59
4.	TG (mg/dl)	161	165	155
5.	TC (mg/dl)	190	198	181
6.	HDL (mg/dl)	40	40	41
7.	LDL (mg/dl)	118	125	109
8.	VLDL (mg/dl)	32	33	31
9.	LH (µIU/ml)	147	151	143
10.	FSH (µIU/ml)	77	81	73
11.	TESTOSTERONE(ng/ml)	14	15	13.5
12.	INSULIN (U/ml)	16.2	17	15
13.	ESTROGEN (pg/ml)	237	240	232
14.	PROGESTERONE (ng/ml)	1.73	1.64	1.9
15.	HOMA-IR	79	84	72.6

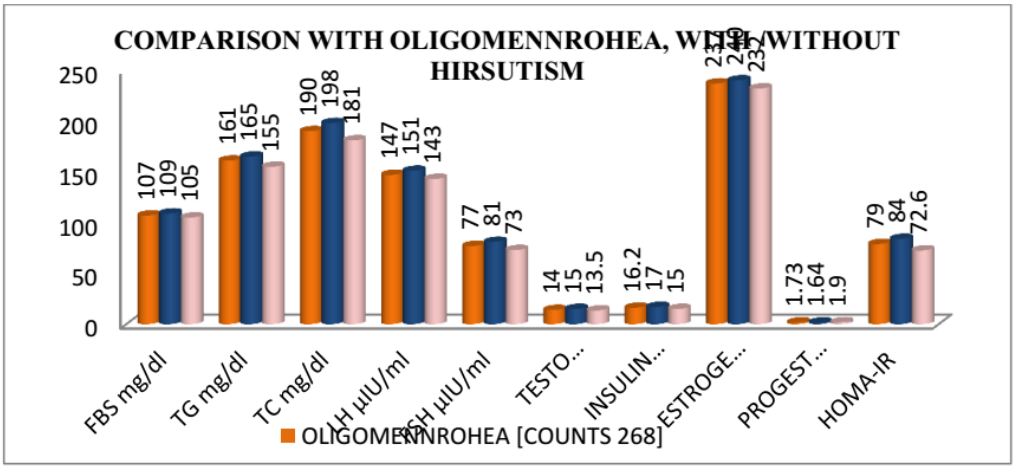


Graph-1: Graphical presentation of Comparison between 'vegeterian diets with fastfood(FF) eaters & nonvegetarian diets with fast food eaters' in hyperglycemic cases (table 11)



Graph-2: Graphical presentation of Hyperglycemic /Diabetes mellitus case with obesity and without obesity.

Table-12



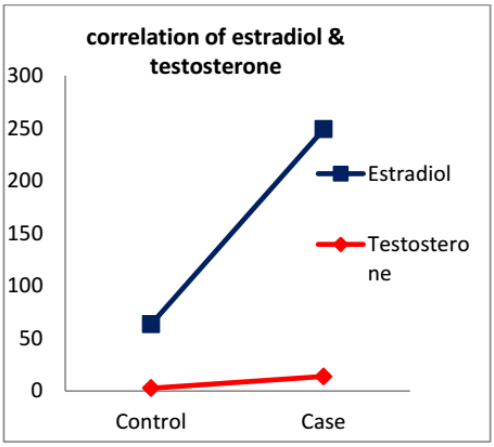
Graph-3: Comparison of different groups of 'oligomennrohea (o/m), o/m with & without Hirsutism' among case (TOTAL: 268/300 CASES) (Table-13)

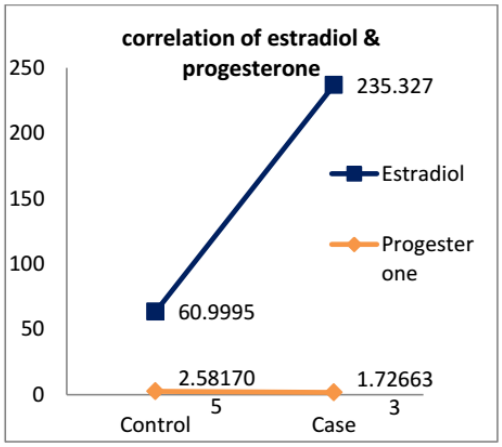
Table−14: Comparison of different type of 'obesity' among cases (one way anova)

S.NO.	COUNTS	240		41		16		3		Df2 (welch) / Anova	P VALUE
	PARAMETERS	Non Obese Mean	SD	Obese Mean	SD	Clinical Ob. Mean	SD	Morbid Ob. Mean	SD		
1.	FBS(mg/dl)	103.3	15.10	115.1	23.52	134.5	35.35	108.3	10.40	8.87	**0.014**
2.	UREA (mg/dl)	24.8	5.56	25.5	6.16	26.4	7.0	26.3	0.57	0.557	0.644
3.	CREATININE (mg/dl)	0.58	0.21	0.55	0.25	0.61	0.33	0.5	0.05	0.14	0.932
4.	TG (mg/dl)	153.3	29.96	177.7	40.34	214.5	49.0	225	70.0	8.58	**0.002**
5.	TC (mg/dl)	176.9	36.64	222.6	36.98	270.6	47.22	270	28.93	49.88	**<0.001**
6.	HDL(mg/dl)	40.77	6.15	38.31	6.49	37.1	5.11	40.6	14.15	8.70	0.075
7.	LDL(mg/dl)	105.7	34.27	148.4	33.3	190.9	45.68	184.3	18.34	47.50	**<0.001**
8.	VLDL (mg/dl)	30.6	5.98	34.9	7.74	42.5	10.29	45	14.0	8.58	**0.004**
9.	LH(µIU/ml)	145.6	38.0	147.9	47.9	165.1	25.88	158.3	41.63	1.33	0.264
10.	FSH(µIU/ml)	74.1	42.67	79	56.98	95.9	49.10	120.6	67.0	8.65	0.332
11.	INSULIN (U/ml)	15.9	6.0	15.18	5.86	20	8.74	17.03	7.95	3.56	**0.015**
12.	TESTOSTERONE (ng/ml)	13.6	6.0	13.55	6.17	18.23	10.0	16.96	7.46	8.68	0.304
13.	ESTROGEN(pg/ml)	234.5	87.62	224.2	89.46	298	104.8	151	118.0	3.77	**0.011**
14.	PROGESTERONE (ng/ml)	1.63	2.11	1.77	2.11	1.81	3.0	6.3	6.13	8.61	**0.006**

- Comparison of the fasting basal sugar (FBS) between the two groups shows that FBS is higher (mean value = 106.7 ± 19.49) in Cases group than Controls (mean value = 96.1 ± 17.0.(Table 8)
- Comparison of the Triglyceride (TG) between two groups shows that TG is higher (mean value = 160.6 ± 36.98) than Controls (mean value = 154.6 ± 23.42). Comparison of Total Cholesterol (TC) between two groups shows that TC is higher (mean value = 189.1 ± 45.47) in Cases than Controls.(Table 8)
- Comparison of the luteinizing hormone (LH) between two groups shows that LH is higher (mean value 147 ± 39) in Cases than Controls (mean value = 90.8 ± 43.6). (Table 9)
- Comparison of the follicular stimulating hormone (FSH) between two groups shows that FSH is higher (mean value 76.4 ± 45.6) in Cases than Controls (mean value = 22.2 ± 17.1). (Table 9)
- Testosterone is higher (mean value 13 ± 6.3) in Cases than Controls (mean value = 2.67 ± 1.4). (Table 9)

- Insulin hormone is higher (mean value 15.5 ± 6.2) in Cases than Controls (mean value = 7.4 ± 2.0). (Table 9)
- Estradiol is higher (mean value 235 ± 90.2) in Cases than Controls (mean value = 60.9 ± 19.6). (Table 9)
- Progesterone is higher (mean value 2.58 ± 3.1) in Controls than Cases (mean value = 1.72 ± 2.2). (Table 9)





Graph−4 & 5: correlation of estradiol & testosterone; correlation of Estradiol & progesterone

Comparison of various parameters in different group's categories among hyperglycemic/ diabetes mellitus cases

- In study there is comparison between vegetarian diets with fast food eaters and Nonvegetarian diets with fast food eaters in hyperglycaemic 74 cases out of 300 PCOD women cases. All parameters are higher in veg with FF eaters. Progesterone ng/ml mean values are 1.53 & 1.66 (lower in veg eaters) in hyperglyemic/DM women.(Table 11 & graph 1)
- In study there is comparison between Without Obesity &With Obesity in hyperglycaemic/DM 74 cases out of 300 PCOD women cases. All parameters are higher in Hyperglyemic /DM with obesity group. Progesterone ng/ml mean values are 1.74 & 1.32 (lower in Hyperglyemic /DM with obesity group) in women. (Table 12 & graph 2)**.**
- In study there is comparison among different groups of Oligomennrohea (O/M), O/M with Hirsutism & O/M without Hirsutism in 268 cases out of 300 PCOD women cases. All parameters are higher in O/M with Hirsutism group compare to O/M without Hirsutism group. Progesterone ng/ml mean values are 1.73, 1.64 & 1.9 (lower in O/M with Hirsutism group) in women (Table 13 & graph 3)**.**

Dunaif *et al*. said that in teenagers, abnormalities in glucose metabolism manifest prior to dyslipidemia, suggesting that assessment of glucose metabolism is even more important in younger women. DM is diagnosed by an 8 h fasting plasma glucose ≥ 126 mg/dL, 2 h glucose value ≥ 200 mg/dL after oral glucose tolerance test (OGTT) or random glucose ≥ 200 mg/dL with symptoms of DM confirmed by either fasting plasma glucose or OGTT. The prevalence of IGT in obese adolescents is surprisingly as high as 15%. It's interesting that despite all the research into PCOS, the exact relationship between the condition and weight gain (or loss) is unclear. But being overweight, and especially increased abdominal fat, seems to be a strong predictor of having other hormonal problems – such as raised male hormones and tendencies to having diabetes [5, 6].

Dahlgren E. *et al.* study, women with PCOS present a more atherogenic lipid profile with elevations of serum triglycerides (TG) and reductions of serum high density lipoprotein (HDL) cholesterol concentrations and are at a greater risk of cardiovascular diseases compared with age-matched control subjects. These clustering risk factors associated with PCOS emphasize the importance of studies to distinguish between metabolic and hormonal alterations which are related to PCOS or to obesity [7, 8].

Mckenna *et al.* Moghetti *et al.* & Hammami *et al*., Most studies have shown hypersecretion of adrenal hormones in PCOS subjects compared with healthy women, but the mechanism of this hypersecretion is not well understood. Possible explanations include hypersensitivity of the adrenal gland to adrenocorticotrophic hormone (ACTH) stimulation, conjugated hypersecretion from the ovary and the adrenals, dysregulation of steroid synthesis enzymes

such as 11β-hydroxysteroid dehydrogenase (11β-HSD) and hyperstimulation of cytochrome *P*450c-17α activity in the adrenal glands as a result of hyperinsulinaemia in these patients[9-14,15].

Acien P *et al.* & Jayagopal V *et al.* studies show that obese (compared to lean) PCOS women tend to have a higher degree of IR. Correlation between hyperandrogenism and IR is significant in many studies but not as significant as the link between insulin abnormalities and obesity. PCOS women demonstrate greater variation in insulin parameters compared to controls, independent of weight. Some human data shows a high degree of correlation between hyperandrogenism and IR. The relationship between hyperandrogenism and IR seem to differ between PCOS and non-PCOS women [16-19].

Unfer *et al.* & Gluck *et al.* studies said that the ratio of LH (Luteinizing hormone) to FSH (Follicle stimulating hormone) is greater than 1:1 (sometimes more than 3:1), as tested on Day 3 of the menstrual cycle. The pattern is not very specific and was present in less than 50% in one study [20,21].

Knochenhaur ES *et al.* Asuncion M *et al.* studies show women presenting with menstrual dysfunction and hirsutism, 86% (and 68% of women with hirsutism but apparent eumenorrhea) had PCOS. Alternatively, in this populational study only 8% of patients with menstrual dysfunction alone (i.e. without hirsutism) had PCOS, raising questions regarding the high proportion of PCOS suggested affecting women with oligoovulatory infertility. Prevalence rate of reported menstrual dysfunction in our population was 22.8%. The incidence of oligomenorrhea (14.6%; 28 of 192) in the studied population may be overestimated because of the definition we used, i.e. less than 8 cycle/yr; this definition was also used in the recent study by Knochenhauer *et al*. H/A was confirmed in 6.77% of the examined women with oligomenorrhea or oligomenorrhea and hirsutism [21].

Hull MG *et al.* population, 74% of women with hirsutism were estimated to suffer from PCOS, and 76% (20.1 of 26.5) of women diagnosed with PCOS demonstrated hirsutism, consistent with our findings in studies of women seeking care for hirsutism [22].

The study demonstrates that there is a relationship between the degree of hormonal abnormality and the menstrual irregularities and hirsutism in women with PCOS suggesting that there may be a progressive nature to the syndrome.

One of the important etiologic factors in acne is an increase in sebaceous gland activity, which is androgen dependent. Acne is a common manifestation of hyperandrogenemia. Therefore, acne may not only cause cosmetic concern but may also be a sign of underlying disease. In females, the most common cause of hyperandrogenemia is polycystic ovary syndrome (PCOS).

CONCLUSION

Obesity is a common finding in PCOS and aggravates many of its reproductive and metabolic features. The relationship between PCOS and obesity is complex, not well understood, and most likely involves interaction of genetic and environmental factors. Insulin resistance and weight gain are two contributing factors to PCOS. Insulin resistance typically causes the body to produce more insulin than normal (hyperinsulinemia). Higher levels of insulin can then cause ovaries to produce too much testosterone which can impair normal ovulation from occurring and further leads to metabolic syndrome in PCOD women with symptoms of Hirsutism, Oligomennrohea and acne.

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