Original Research Article

Comparison of endocrine and metabolic characteristics of lean and overweight/ obese women with PCOS

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Received: 22-08-2020 / Revised: 29-09-2020 / Accepted: 27-10-2020

Abstract

Aims & objective :1. To study clinical features in lean and overweight/obese women with PCOS 2. To compare the hormonal assays between lean and overweight/obese women PCOS 3. To evaluate the metabolic parameters in lean and overweight/obese women with PCOS .**Material & Methods** :This was a prospective observational study conducted in Jehangir Hospital, Pune.from 1st December 2014 to 31st August 2015. All women with a history of oligo/ amenorrhea and clinical signs of hyperandrogenism like acne or hirsutism, were enrolled in the study. Patients fulfilling at least two out of three Rotterdam Consensus revised diagnostic criteria of PCOS were recruited. A detailed history was taken and examination done. Women were stratified into 2 groups lean and overweight/obese on the basis of BMI. All women were subjected to blood hormonal and metablic profile and USG pelvis. Differences in endocrine and metabolic parameters were studied **Results :**There was more marked hyperinsulinemia [4%vs 3.3%], relative hyperglycemia[8.1% vs 2.1%], with deranged lipid profile [25.1%vs 10.3%]in the overweight/obese women as compared to lean women with PCOS.The LH level was higher in the non obese[33.3%] PCOS group as compared to the overweight/obese PCOS [22.5%] .**Conclusion** :Extensive efforts should be made to fully investigate the syndrome in order to make lifestyle changes to delay the serious longterm effects of the disease

Keywords: PCOS, Diabetes Mellitus, BMI, Glucose, Lipid Profile, Insulin, Insulin Resistance, Hirsutism.

Introduction

Polycystic ovary syndrome (PCOS), characterized by both reproductive and metabolic aberrations, is the most common endocrinopathy in younger women, with an estimated prevalence of 5– 10% in the general population[1,2]. It is a frustrating experience for women, often complex for managing clinicians and is a scientific challenge for researchers. Its prevalence among infertile women is 15%–20%[3].PCOS is an enigma as its underlying pathophysiology is not fully understood. It is associated with hyperinsulinemia, glucose intolerance, obesity and altered lipid profile. Insulin resistance is thought to be the uniting pathogenic factor in the associations between hypertension, glucose intolerance, obesity, lipid abnormalities and coronary artery disease, which together constitutes metabolic syndrome or syndrome 'X.' Studies done on Indian population, though limited, have suggested that abnormalities of the insulin receptor are more common in Indian women with PCOS compared to white women with PCOS[4]. Obesity has a significant impact on the clinical presentation and metabolic manifestation of PCOS. Obesity, particularly in women with PCOS, can result

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in many reproductive disorders like menstrual irregularity, infertility and miscarriage. Approximately half of all the women with PCOS are overweight or obese. Obesity has also been shown to be an independent predictor of conversion to impaired glucose tolerance or type 2 diabetes mellitus (DM) in women with PCOS. There are also differences detected in lipid profile in overweight/obese compared with lean women with PCOS, with elevated triglycerides and lower high-density lipoprotein (HDL) cholesterol being the most consistently described. There are endocrine and metabolic differences between lean and overweight/obese women with PCOS such as more marked hyperinsulinemia and insulin resistance, relative hyperglycemia, and lower Sex Hormone Binding Globulins (SHBG) in the overweight/obese women as compared to lean women with PCOS. No treatment is a panacea, because treatments, so far, have been directed at the symptoms but not at the syndrome itself. Extensive efforts should be made to fully investigate the syndrome in order to make therapy more successful and to delay the serious long term effects of the disease on patients' health. Emphasis of management is shifting from symptomatic management to lifetime management with increasing focus on long term effects of PCOS like diabetes mellitus, hypertension, infertility and uterine malignancy. Indian women have higher predisposition to insulin resistance and diabetes mellitus, so we decided to study the effects of PCOS on metabolism in our population and whether we can find any differences and predictors in lean and overweight/ obese PCOS.

Aims and objectives

- To study clinical features in lean and overweight/obese women with polycystic ovarian syndrome.
- To compare the hormonal assays between lean and overweight/obese women with polycystic ovarian syndrome
- To evaluate the metabolic parameters in lean and overweight/obese women with polycystic ovarian syndrome

Material and methods

This study was conducted in the Department of Obstetrics and Gynaecology, Jehangir Hospital, Pune following approval by the Ethics committee.

Type of Study

Prospective Observational Study.

Period of Study

1 year (1st Dec 2014 to 30th Nov 2015)

Study population-All women who fulfilled the inclusion criteriawere enrolled in the study.

Sample size

By considering prevalence of PCOS as 6% in general population, sample size[N] was calculated using following formula

$$N = \frac{4xPxQ}{L2}$$

P-Prevalence [6%]Q-1-P [94%]

L-Experimental error [5%]

$$N = \frac{4x6x94}{25} = 90.2$$

Hence the sample size was 90; we enrolled 94 cases in our study.

Inclusion criteria

- Women diagnosed with PCOS using Rotterdam diagnostic criteria
- Women in age group 15-40 years.
- Women who were at least 2 years postmenarchal.

Exclusion criteria

i. Women with known endocrine disorders such as hypothyroidism, hyperprolactinemia, Diabetes mellitus, adult onset adrenal hyperplasia.
ii. Women with known metabolic disorders such as dyslipidemia.
iii. Premature ovarian failure.
iv. Those who refused to consent.

Methodology

All women with a history of oligo/ amenorrhea and clinical signs of hyperandrogenism like acne or hirsutism, were enrolled in the study from the outpatient clinic. Patients fulfilling at least two out of three Rotterdam Consensus revised diagnostic criteria of PCOS (2003) were recruited after obtaining consent[6,7].These criteria were defined as:

- Oligo- and/or anovulation
- Clinical and/or biochemical signs of hyperandrogenism
- Polycystic ovaries

On ultrasonography morphology of polycystic ovary was considered as presence of 12 or more follicles in each ovary measuring 2-9 mm in diameter arranged peripherally with hyperechoic stroma, and/or increased ovarian volume (>10 ml). A detailed history was taken. Menstrual history was recorded which included age at menarche, date of last menstrual period (LMP) with regularity of menses, duration and amount of bleeding.

1. Oligomenorrhea was defined as when menstrual bleeding occurred at an interval of 35 days to 6 months, with less than 9 menstrual periods per year.

2. Secondary amenorrhea was defined as absence of menstruation for 6 months.

A detailed family history was taken including history of PCOS, Diabetes mellitus, hypertension, dyslipidemia, heart disease and malignancy. History of acne, hirsutism, infertility or inability to lose weight was also recorded. A thorough general, systemic and gynaecological examination was done.

Height, weight, blood pressure, BMI (in kilograms per square meters), Waist circumference (in inches) at the level of anterior superior iliac spine was recorded.

Women were then stratified into two groups on the basis of BMI. Group A included women with BMI < 24.9 (Lean PCOS) and Group B was comprised of women with BMI ≥ 24.9 (Overweight and Obese PCOS) Virilizing signs like excessive male pattern facial and body hair as well as acne, male pattern balding or alopecia, increased muscle mass, deepening voice or clitoromegaly were noted. Hirsutism is excessive facial and body hair caused by excess androgen production. For hirsuitism modified Ferriman- Gallwey score was used. Nine body areas were scored from 0(no hair) to 4 (frank virile) including upper lip, chin, chest, upper and lower abdomen, thigh, upper and lower back and arm. A total score of 8 or more was considered abnormal. Acanthosis nigricans is diffuse velvety thickening and hyperpigmentation of skin. Women were examined for the presence of Acanthosis nigricans in areas such as nape of the neck, axillae, area beneath the breasts, intertriginous areas and exposed areas like elbows and knuckles.[21-28].All recruited women were subjected to the following tests on 2nd day of menses and the reference values will be taken as follows

Table 1:Various tests and their normal ranges

Sr. No	Test	Normal Range
1	LH	2.4-12.6 mIU/ml
2	FSH	3.5-12.5 mIU/ml
3	Serum Prolactin	3.24-29.12 ng/ml
4	DHEAS	35-430 ug/dl
5	Serum testosterone	2.8-8 ng/ml
6	Fasting blood glucose	70-110 mg/dl
7	Fasting serum Insulin	2-25 uU/ml
8	TSH	0.4-4.5 uU/ml
9	Total cholesterol	<200mg/dl
10	Triglycerides	40-250mg/dl
11	LDL	60-130mg/dl
12	HDL	30-70mg/dl

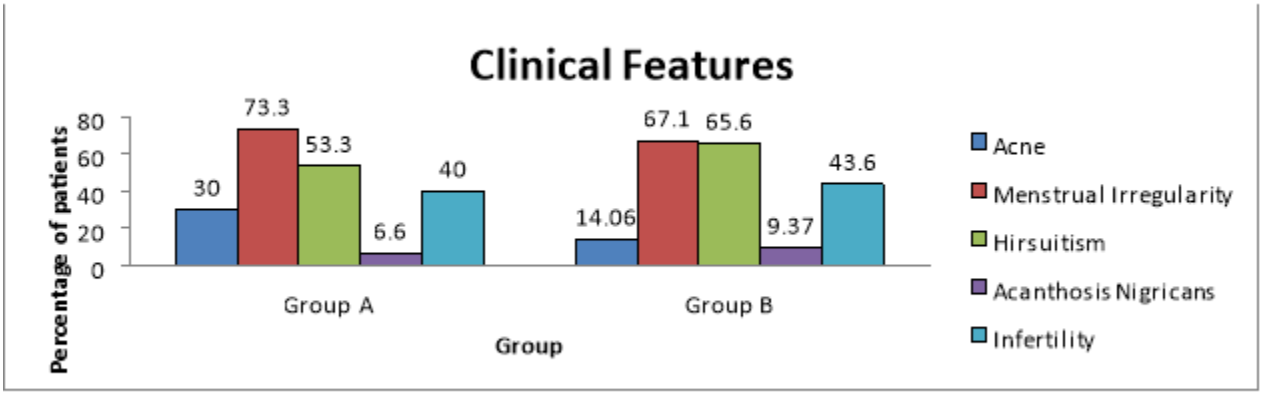
Ultrasonography (USG) pelvis was done. Endocrine and metabolic differences in both groups were then studied. All findings were recorded on a predesigned proforma and the data was analysed by application of appropriate statistical tests. For comparing qualitative data between the two groups, Chi-square test was used, whereas 2 independent sample t-test or Mann – Whitney U test was used to analyze quantitative data. P value < 0.05 was considered significant[29-33].

Observations and results

Clinical features-

Table 2:Clinical features

Clinical features	Group A (n-30)	Group B (n-64)	P value
Acne	9[30%]	9 [14.06%]	0.121
Menstrual irregularity	22 [73.3%]	43[67.1%]	0.717
Hirsuitism	16[53.3%]	42[65.6%]	0.311
Acanthosis nigricans	5 [6.6%]	6[9.37%]	0.495
Infertility,	12 [40%]	28[43.6%]	0.395



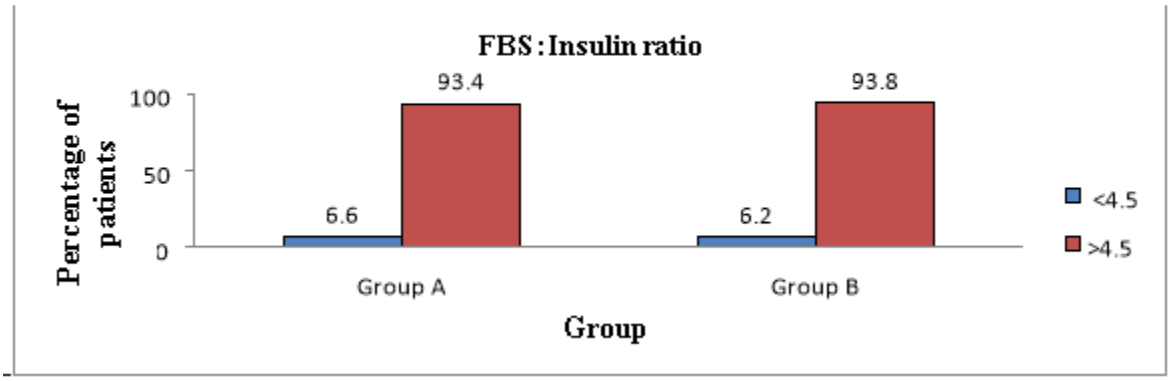
Menstrual irregularity is most common symptom followed by hirsutism and infertility.

Fig 1:Clinical features

Table 3: FBS: Insulin ratio

FBS:Insulin ratio	Group A N-30	Group B N-64
< 4.5	2[6.6%]	4[6.2%]
> 4.5	28[93.4%]	60[93.8%]

P value - 0.707



Difference in FBS :Insulin ratio of lean and obese was found to be statistically insignificant.

Fig 2: FBS: Insulin ratio

Total Cholesterol

Table 4:Total Cholesterol

Groups	Mean Cholesterol (X SD)
Group A (N = 30)	157.23 26.97
Group B (N = 64)	159.86 31.24

P value- 0.677

There was no statistically significant difference between two groups in mean cholesterol level.

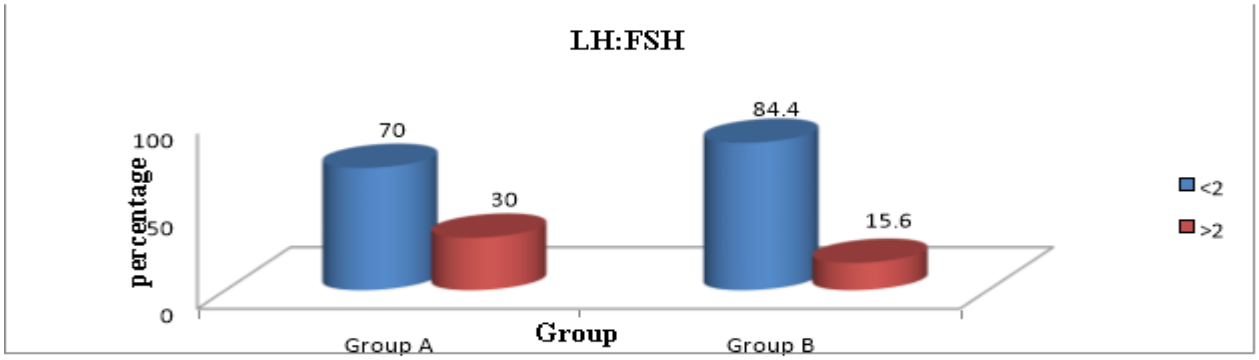
LH:FSH ratio

Table 5: LH:FSH ratio

LH:FSH ratio	**Group A N -30**	**Group B N-64**

< 2	21[70%]	54[84.4 %]
> 2	9 [30%]	10 [15.6%]

P value is 0.179



Lean PCOS had a higher percentage of patients with raised LH :FSH ratio but it was not statistically significant.

Fig 3:LH:FSH ratio

Testosterone

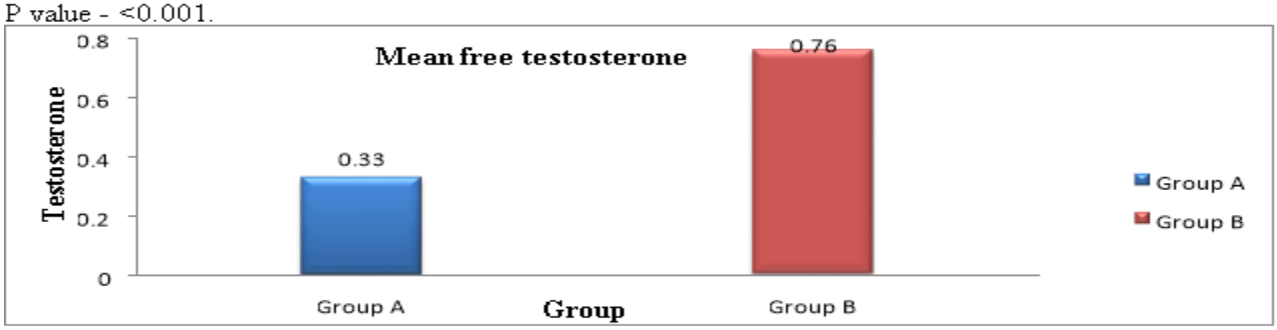
Table 6: Testosterone levels

Groups	Mean Free testosterone (X SD)
Group A (N = 30)	0.33 0.48
Group B (N = 64)	0.76 0.59

Discussion

PCOS (Polycystic ovarian syndrome) is one of the most common endocrine disorders in women of reproductive age, affecting 5-10 % of women worldwide[6].

P value - <0.001.



Mean free testosterone was significantly higher in obese PCOS than lean PCOS

Fig 4:Mean free testosterone

Data on prevalence of PCOS is variable, in part because of lack of international consensus on diagnostic criteria of PCOS. The prevalence of polycystic ovaries (PCO) in the general population has been studied widely but only a few investigators have taken up the challenge to study the prevalence of the syndrome.[2]We studied 94 patients in the age group of 18-40 years fulfilling the criteria of PCOS. Women were stratified into 2 groups on the basis of BMI. Group A included 30 women with BMI <24.9 (Lean PCOS) and Group B comprised of 64 women

with BMI ≥ 24.9 (Overweight and Obese PCOS) [34-38].

Age

In our study on 94 PCOS patients, majority of the patients in group A [70%] and Group B [54.8%] belonged to the age group of 15-25 years, while only 30% patients from group A and 40% patients from group B belong to age group of 26-35years. There was only one patient in age group 36-40 years from group B. Sujatha et al conducted a study on 80 patients with PCOS in age group of females 18-40 years and noted 52.5 % nonobese patients in age group 20-24 years ,while 30% obese in age group 2024 year and 30 % in 25-29years[2].

According to Bronstein et al PCOS may occur at a younger age in girls who develop early pubarche and thelarche. Therefore, the diagnosis and workup should be considered in young girls with risk factors suggestive of PCOS[22].

BMI

In our study 32% patients were lean 15.9 % overweight and 52.1% were obese.

Table 7:BMI Study

Author	Population studied	Overweight (%)	Obese (%)
Aziz et al., 2004	**USA**	**19.4**	**58.2**
Shringi et al., 2003	**India**	**41.1**	**37.8**
Carmina et al	**Italy**	**60.9**	**28.4**

This variance in prevalence may be due to different criteria used for defining overweight and obese in different studies. It may also depend on the prevalence of obesity in that population.

Clinical Features

Themost common symptom in our study was menstrual irregularity. 73.3% of Group A and 67.1% of Group B had irregular menses.

In adolescent and young women, the age of onset of obesity and that of menstrual irregularities are significantly co-related[64-68].

Infertility

In our study, 40% cases in group A and 43.6% in group B had infertility. In a study by Sujatha et al the prevalence of infertility in nonobese PCOS was 42.5% and obese PCOS was 55%[2].According to Hartz et al, presence of anovulatory cycles, oligo-amenorrhoea infertility and hirsutism either separately or in association, were significantly higher in obese than in normal weight women[65].The relationship between excess body fat and reproductive differences appears to be stronger for early onset obesity, although this remains a controversial issue due largely to heterogeneity of overweight or obese pre-adolescent or adolescent population investigated[39-48].

Hirsutism

A slightly higher incidence of hirsutism was seen in overweight/obese women as compared to lean women with PCOS in our study[49-56].53.3 % patient in Group A and 65.6% in group B had hirsutism, however the difference was not statistically significant [P-0.179].This correlates with higher mean testosterone found in obese compared to lean.

As per a study conducted by Kiddy et al, obese women with PCOS had a greater prevalence of hirsutism (lean 56% compared with obese 73%). [25]

While Saxena et al found hirsutism was comparable in both groups [P>0.05]. [27]

Sujatha et al in their study noted a slightly higher prevalence of hirsutism in nonobese [70%] than obese [80%] women with PCOS and further opined that hirsutism in PCOS typically begins in adolescence and progresses with age so simple reassurance is not appropriate for most women with hirsutism[2].

Acne

In the adolescent, clinical evidence of androgen excess, such as severe acne or hirsutism, may prompt evaluation for PCOS[5].In our study 30% patients in group A and 14.06 % patients in Group B had acne The difference was not significant [P>0.05]. Similar to our study Saxena et al found 9.5% lean and 15.5 % of obese had acne which also did not achieve statistical significance. [P>0.05][27], whereas Sujatha et al in their study found that 42.5% nonobese and 47.5 % obese women had acne[2].Slayden et al in their study quoted that acne may be the presenting symptom of underlying hyperandrogenism [57-66]. The study by Saxena et al study also states that PCOS patients are insulin resistant and compensatory hyperinsulinemia promotes formation of androstenedione and testosterone in ovary which is seen clinically as acne and hirsutism.

Acanthosis nigricans

Acanthosis nigricans, when not related to malignancy,is considered to be a dermal manifestation of hyperinsulinaemia[67-69].In our study 9.37 % of obese PCOS and 6.6% of lean PCOS had acanthosis nigricans which is statistically insignificant. 6.6 % of

lean and 4.6 % of obese PCOS had acanthosis with hyperinsulinemia. Sujatha et al have found higher incidence of acanthosis nigricans in obese than lean PCOS [Lean 10 % vs obese 42.5 %] but this was not statistically significant[2].

Family history

In our study in Group A family history of diabetes mellitus was present in 40% while 61% patients in Group B. But this difference was not statistically significant [P-0.94]. Silfen et al noted a family history of diabetes mellitus [Nonobese 55% vs obese 82 %] was high in both groups[71].Whereas Saxena et al quoted that family history of diabetes mellitus was more common in overweight as compared to lean and may indicate a genetic predisposition[27].In our study family history of hypertension was present in 57% from group A and 62.5 % from Group B which was not statistically significant [P-0.96]. Similar to diabetes mellitus, Silfen et al noted that family history of cardiovascular disease. [Nonobese 64%, obese 55%]was high in both groups[71].In our study 10% of patients from Group A and 12.5 % patients from Group B had family history of PCOS which was statistically insignificant [P-0.99]. Familial clustering of PCOS cases suggests that genetic factors play an important part in its etiology. Heterogeneity of clinical and biochemical features in PCOS can be explained by the interaction of a small number of key genes with environmental, particularly nutritional factors[72].

Investigations

Glucose

There was no significant difference in mean fasting blood sugar levels in two groups in our study [P-0.148].Mean fasting blood sugar was 83.43± 9.31 in Group A and 86.46±11.06 in Group B . 6.6 % patients from Group A and 6.2% patients from Group B had FBS:Insulin ratio < 4.5 [P-0.707]. Similar to our study Silfen et al noted similar levels of Fasting glucose [Nonobese 85.9± 9.6,obese 88.4±6.5] and HbA1c in the obese and nonobese group, and none of the subjects had impaired fasting glucose or diabetes. Despite normal fasting glucose levels, 27% of the obese PCOS subjects who underwent testing had impaired glucose tolerance compared with none of their nonobese counterparts, although this difference was not statistically significant most likely due to the small sample size. This corroborates the limited sensitivity of fasting glucose levels in predicting glucose intolerance in PCOS[70-72].Contrarary to our study Majumdar and Singh noted an increased prevalence of IGT (Nonobese 10% vs obese 25%, P= 0.000,) and type two diabetes mellitus in obese group. (Nonobese 6% vs obese11.7%, P= 0.0000)[60].

In a study by Legro, nonobese PCOS patients have a 10.3% prevalence of IGT and 1.5% of type two diabetes mellitus. Obese PCOS patients have a 31% rate of IGT and 7.5% met the criteria for type two diabetes mellitus, which is three times that of the general population. PCOS women are at a significantly increased risk for IGT and type two diabetes mellitus at all weights and at a young age[56].

Lipid Profile

In our study mean cholesterol levels were compared. It was 157±26.92 in group A and 159.86±31.24in group B. The difference was insignificant. (P= 0.677). Mean levels of serum HDL in group A was higher i.e. 44.67±8.26 and in group B was 40.27±6.19 and the difference was statistically significant. (P= 0.024).

There was significant difference in mean LDL levels in two groups. In group A it was 87.50±22.31 which was much less than 102.27±21.44 in group B, and this difference was significant. [P > 0.004] Mean triglyceride levels was 97.7±28.73 in Group A which was less as compared to 117.17±43.74 in Group B and this difference also was statistically significant. (P = 0.012). Obesity is often associated with a more atherogenic lipid profile in women with PCOS. Specifically, elevated triglycerides and lower HDL cholesterol comprise the most consistently reported alterations in obese compared with nonobese subgroups[29]. In a study by Silfen et al, the obese adolescents with PCOS similarly demonstrated a more atherogenic lipid profile, with higher LDL and lower HDL cholesterol, than their nonobese counter parts[71].Holte J et al noted higher free fatty acids, total cholesterol and low density lipoprotein cholesterol in obese PCOS than in nonobese PCOS patients[51].Strowitzki et al demonstrated dyslipidemia in both lean and obese PCOS women [73]. Significantly higher LDL (P=0.018) and lower HDL (P=0.015) levels were found in obese PCOS by Tao Tao et al[74].As per a study by Graf M J there were significant decreases (P less than or equal to 0.01) in highdensity lipoprotein (HDL) levels in the obese PCOS[75].Insulin and higher fat distribution play an important role in regulating lipid levels.

Recently obesity has been reported to be a significant predictor for development of metabolic syndrome within PCOS patients[78]. However Legro et al noted that LDL cholesterol was disproportionately elevated with PCOS when compared to other insulin resistant states[79]. Finally Karla A opined that with ethnic differences in prevalence of PCOS, insulin resistance and lipid parameters one should know status of their population before implementing the measures used in a different race and place[35].Talbott et al noted in a

review that women with PCOS had dyslipidemia, increased blood pressure, plasminogen activator inhibitor and coronary artery calcification. An interesting observation that they made was that abnormal lipid profile difference between PCOS cases and controls was mainly seen in women aged less than 45 years, while carotid artery changes were seen in PCOS women after 45 years. This indicates that dyslipidemia occurring at a younger age translates into atherosclerosis and cardiovascular disease later in life. Although obesity is often associated with metabolic disorders, lean women with PCOS also have been found to have hyperinsulinemia and dyslipidemia[80].

TSH

In our study mean TSH in Group A was 2.30 ±1.21 while in Group B it was 2.27±1.13. This difference did not achieve statistical significance [P=0.921]. Similar to our study, Saxena et al found no significant difference in serum TSH of lean and obese PCOS.[27] Serum TSH (mU/ml) [lean PCOS 2.4 ± 1.8, Obese PCOS 2.3± 1.6, P>0.05] was not significantly different though Sinha et al noted significantly higher prevalence of goiter (27.5% vs. 7.5%) and subclinical hypothyroidism (22.5% vs. 8.75%) in PCOS patients as compared to controls[81].

LH and FSH

In our study mean FSH level in group A was 7.07±4.95 which was comparable with 6.84±2.17 in group B and hence the difference was insignificant. (P = 0.815). Mean LH was also comparable in Group A [9.23 □ 5.61] and in Group B [8.47 □ 5.71] hence the difference was not significant. Saxena et al in their study had a similar hormonal profile in both groups, with high Serum LH[Lean 16.6 ± 0.68 Obese16.8± 10.42] and LH /FSH ratio[Lean2.6±1.1 Obese 2.5 ±0.83] in both groups with low progesterone denoting anovulation in both groups leading to irregular cycles[27].Silfen et al found that the mean LH level was higher in the nonobese PCOS group compared with the obese PCOS group, whereas their mean FSH levels were comparable. In our study 30 % patients in Group A and 15.6 % patients in Group B had LH :FSH ratio > 2.The difference was statistically insignificant [P-0.179]. Most women with PCOS exhibit an increased level of LH and a higher ratio of LH to FSH than non PCOS women[43] which is explained by Saxena et al as being due to hyperinsulinemia probably acting at the level of hypothalamo-pituitory axis and stimulating LH secretion. This leads to anovulation with irregular cycles. Also the increased androgen is converted to estrone and is responsible for causing endometrial hyperplasia and potentiates LH secretion[27].

Insulin

Polycystic ovary syndrome (PCOS) is not only a reproductive endocrinopathy but also a metabolic disorder. PCOS is associated with hyperinsulinemia, glucose intolerance, obesity and altered lipid profile.[82,83]In our study the mean serum fasting insulin level in group A was 8.73±6.17 which was not significantally different to that in Group B [10.56±6.58] . SimiliarlySaxena et al noted comparable levels of fasting insulin levels in both groups P>0.05[27]Contrary to our finding Silfen at al noted higher levels of fasting insulin in obese [Nonobese12.0±3.6 u/ml,obese 30.7± 18.3u/ml, P <0.001][71].Most Obese women with PCOS are insulin resistant [Nonobese 9.4(7.7-14.1) obese 29±20][76].Insulin sensitivity was found to be normal in muscle, liver, and adipose tissue in slim women with PCOS[86].Sujatha et al further recommends treatment of insulin resistance which will reduce ovarian androgen secretion and can cause resumption of ovulatory menstrual cycle. It also improves the clinical and biochemical features of PCOS and decreases the risk of endometrial and breast malignancies, cardiovascular and cerebrovascular disease. This also corresponds with studies by Gadir A et al and Soloman CG et al[87,88].Srezednicka et al drew attention to the role of insulin in lipid abnormalities observed in women with PCOS[89]. In their study, after adjustment for age, BMI and sex steroids, fasting insulinemia was a significant explanatory variable for total triglycerides, suggesting that hyperinsulinemia, independent of obesity, might play a role in the lipid disturbances of PCOS. In a similar study done by Robinson et al, they found that insulin insensitivity contributes significantly beyond BMI to the low HDL cholesterol in women with polycystic ovaries[90].They concluded in their study that polycystic ovary syndrome is associated with biochemical risk factors for premature vascular disease that cannot be explained by obesity alone. But a study done in India by Bhattacharya et al found no correlation between the fasting glucose / insulin ratio and the triglyceride levels[91].

Androgen

In our study, mean free testosterone was 0.33 ±0.48 among group A. This was significantly less than 0.76 ±0.59 in group B. [P <0.001]. 173.17±95.44 was the mean DHEAS in group A and was comparable to 146.58±74.4 in group B and thus the difference was not significant. [P = 0.185] In a study by Silfen et al study the nonobese subjects had higher mean levels of androstenedione and DHEAS (ug/dl) [Nonobese 205.3±76.3,obese146.1± 68.8, P <0.05] than obese but

similar total testosterone (ng/dl) [Nonobese 49.8 ± 18.7 Obese 58.4±20.4] suggestive of increased adrenal hyperandrogenism. In addition,the concentration of dihydrotestosterone was shown to be higher in the nonobese than in the obese PCOS group, possibly indicating increased peripheral conversion [71].A study was conducted by Acien P et al on 137 PCOS patients [100 Nonobese and 37 Obese PCOS]. Nonobese women with PCOS had insulin and metabolic variables similar to those without PCOS, and most obese women with PCOS were more hyperandrogenic [Testosterone Nonobese0.8ng/ml, Obese 1. 04 ±0.5], DHEAS [Nonobese3.1±1.4, obese 2.9 ±1.6][76].

Prolactin

In our study mean prolactin was 13.45± 6.84 in Group A and 12.06 ± 6.23 in Group B which was statistically insignificant [P-0.349]. Similarly, Sujatha et al in their study on 42 lean and 58 overweight PCOS noted comparable levels of serum prolactin[2]. As per Roy Homberg, anovulation caused by hyperprolactinemia is usually associated with prolactin concentration more than twice the upper limit of normal. Mildly elevated serum prolactin which is not associated with anovulation may be seen in about 30 % of women with PCOS and these should not be treated[8].Thus overweight/obese and lean PCOS manifest clinical, metabolic and endocrine differences. This observation may influence future management protocols like lifestyle modification to delay the onset and effects of metabolic syndrome and management of infertility. Although obesity is often associated with metabolic disorders, lean women with PCOS also have hyperinsulinemia and dyslipidemia, both of which are risk factors for cardiovascular diseases. Thus general prevalence of insulin resistance in South Asian women with PCOS is quite high. More research is needed to find out the reason for insulin resistance and dyslipidemia in Indian population.

Results

From this study, after after an extensive study of clinical features, harmonal assays and metabolic parameters in comparison with BMI- overweight/obese women with PCOS against lean women with PCOS - significantly marked hyperinsulinemia[4%vs3.3%], relative hyperglycemia[8.1% vs 2.1%], with deranged lipid profile [25.1%vs 10.3%] was observed with overweight/obese women than lean women with PCOS.In contrast , the LH level was higher in the non-obese [33.3%] PCOS group as compared to the overweight/obese PCOS [22.5%] group.

Conclusion

- PCOS is a common complex condition in women associated with psychological, reproductive and metabolic features.It is a chronic disease with manifestations across the lifespan and represents a major health and economic burden.
- Obese PCOS are associated with more atherogenic lipid profile than lean PCOS. It is important therefore that those caring for these patients understands not only management issues pertinent to their speciality but also appreciate the other potential health risk in these women and counsel accordingly.
- According to my study, the obese group has higher levels of testosterone, since hyperandrogenemia can lead to more atherogenic lipid profile, dietary changes along with exercise should be advised to these patients.
- Longitudinal follow up studies of these women is therefore recommended to evaluate effects on future fertility and onset of metabolic disorders. But cost is constraint.

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Conflict of Interest: Nil
Source of support:Nil