

## ASSESSMENT OF RISK FACTORS ASSOCIATED WITH PCOS IN PREMENOPAUSAL WOMEN IN A TERTIARY CARE CENTRE

<sup>1</sup>GNANAPRIYA JEEVANANDANAM, <sup>2</sup>AYSHVARYA RAMALINGAM, <sup>3</sup>YOGAPRIYA V,  
<sup>4</sup>BALASUBRAMANIAN ARUMUGAM, <sup>5</sup>ARCHANA MUKUNDA

<sup>1</sup>Assistant Professor, Department of Biochemistry, Sri Venkateshwara Medical College Hospital and Research Centre, Puducherry.

<sup>2</sup>Assistant Professor, Department of General Anaesthesia, Government Omandurar Medical College, Chennai.

<sup>3</sup>Associate Professor, Department of Biochemistry, Madha Medical College and Research Institute, Thandalam, Kovur, Chennai.

<sup>4</sup>Assistant Professor, Department of Biochemistry, Government Kilpauk Medical College, Kilpauk, Chennai.

<sup>5</sup>Professor, Department of Oral Pathology and Microbiology, Royal Dental College, Palakkad, Kerala.

### ABSTRACT

**Introduction:** PCOS is a heterogeneous endocrine disorder seen in women of reproductive age. It causes abnormality like polycystic ovaries, hyperandrogenism and irregular menstrual cycle. PCOS is thought to be associated with risk factors like Type 2 Diabetes mellitus. It is also associated with risk factors which can be life threatening.

**Aim:** The aim of the current study was to analyze and correlate the biochemical parameters and hormonal parameters of women with PCOS and to identify the risk factors associated with PCOS

**Material & Methods:** Group I comprising of 70 women with PCOS; Group II with 70 women without PCOS were included in the present study. Blood samples from participants of both groups were assessed for biochemical parameters like FBS, Lipid profile, uric acid and hormonal parameters like TSH, prolactin, insulin & HOMA-IR. Premenopausal women in the age range of 18-40yrs diagnosed with PCOS Patients with diabetes mellitus, hypertension, thyroid disorders, cardiovascular disease, Cushing's syndrome, pregnant or lactating women and women on medication for oral contraception, hypoglycemic, lipid lowering drugs, hormonal medications within previous 6 weeks were excluded from the present study

**Results:** The participants of the study were 100% females and in the age group of 18-40years. There was a positive correlation of FBS and Lipid profile among Group I and Group II and the p value was <0.05. Serum uric acid was found to be in normal range in both patients with p value of 0.606. Serum TSH level, Prolactin level, serum insulin level and HOMA-IR were significantly increased in women with PCOS than in healthy controls and the p value was <0.05.

**Conclusion:** The use of simple and cost-effective biochemical parameters might prove to be biomarkers in early detection of these metabolic changes and may help to identify women with PCOS and the associated risk factors.

**Key words:** HOMA-IR, Polycystic Ovarian Syndrome, Prolactin, Thyroid Stimulating Hormone.

### INTRODUCTION

Polycystic Ovarian Syndrome (PCOS) is the most common female endocrine disorder affecting about 7-10 % of women of reproductive age. PCOS results from a functional derangement rather than a specific central or local defect. It is a heterogeneous endocrine disorder which is associated with anovulation and may result from wide variety of causes. [1]

PCOS is characterized by increased ovarian and adrenal androgen secretion, hyperandrogenic symptoms such as hirsutism, acne and/or alopecia, menstrual irregularity and polycystic ovaries. One of the most prominent metabolic symptoms of PCOS is insulin resistance which includes two conditions- Hyperinsulinemia and Impaired Glucose Tolerance. Based on Rotterdam 2003 criteria PCOS is diagnosed if two out of three criteria are present namely

oligo or anovulation, hyperandrogenism and polycystic ovaries. [2]

With the advent of newer laboratory facilities, diagnostic modalities like sonography and routine laparoscopic evaluation incidence of PCOS has increased drastically.[3] Women with PCOS are at higher risk of developing diabetes, hypertension, infertility, cardiovascular disease and malignancies related to hyperestrogen.[4] Thus the aim of the current study was to analyze and correlate the biochemical parameters and hormonal parameters of women with PCOS and to identify the risk factors associated with PCOS.

### Materials and methods

This prospective comparative study was conducted in the Department of Biochemistry, SCB Medical College and

Hospital Cuttack. The current study was conducted as a thesis research for partial fulfillment of requirement for the degree of MD affiliated to the UTKAL University Odisha. The study was conducted over a period of one year on a sample size of 140 participants divided into 2 Groups.

Group I comprised of 70 premenopausal women in the age range of 18-40yrs diagnosed with PCOS visiting the OPD or indoor in the Department Obstetrics and Gynecology, S.C.B. Medical College and Hospital, Cuttack were included in the study. Patients were selected based on Rotterdam criteria (2003) for diagnosis of PCOS along with their history, physical examination, biochemical investigation and ultrasound of ovaries.

Group 2 comprised of 70 healthy female volunteers with regular menstrual cycle and no clinical or biochemical features of hyperandrogenism, thus excluding the diagnosis of PCOS. Participants with diabetes mellitus, hypertension, thyroid disorders, cardiovascular disease, Cushing's syndrome, pregnant or lactating women and women on medication for oral contraception, hypoglycemic, lipid lowering drugs, hormonal

medications within previous 6 weeks were excluded from the present study.

All the participants of the study were given a questionnaire containing details of age, medical history, menstrual history, family history of Diabetes Mellitus or PCOS. The study was conducted after obtaining ethical approval from the Institutional Ethical Committee. The procedure was explained and Informed consent was obtained from all the participants.

#### Sample collection and testing

5ml of venous blood sample was collected from participants of both Group A and Group B after overnight fasting. 1 ml of blood was stored in a tube containing sodium fluoride as anticoagulant and the plasma was analyzed for Fasting Blood Sugar (FBS). 4ml of sample was stored in a plain tube. The serum was used to assess Insulin, TSH, Prolactin and Lipid Profile using commercial available kits. All biochemical parameters except hormonal assays were analyzed using automated Biochemical Analyzer (Toshiba 120 FR). Hormonal assay was done using Chemiluminescence Immunoassay [CLIA SIEMENS CENTAUR XP. The biochemical assays done based on the principles are mentioned in Table 1

	Biochemical Assay	Principle
1	Fasting Blood Glucose	GOD-POD method
2	Lipid Profile	
	Serum Total Cholesterol	CHOD-POD method
	Serum Triglycerides	GOD-POD method
	Serum HDL (High Density Lipoprotein)	Immunoturbidimetry Assay Method
	Serum LDL (Low Density Lipoprotein)	Freidwald's Formula
	Serum VLDL (very Low Density Lipoprotein)	Freidwald's Formula
3	Hormones	
	Serum TSH	Chemiluminescence Immunoassay [CLIA]
	Serum Prolactin	Chemiluminescence Immunoassay [CLIA]
	Serum Insulin	Chemiluminescence Immunoassay [CLIA]

Table 1: Showing the various biochemical assay done and their principle in the present study

The values obtained were entered into excel sheets and stored. The values were expressed as mean± Standard deviation. Deviation and the findings were analyzed by student t test. Pearson's correlation coefficients were calculated to assess the correlation between the biochemical parameters in the study Group I and Group II. A p value of < 0.05 was considered statistically significant.

#### Results

In the present study 70 patients with confirmed diagnosis of PCOS (Group I) and 70 healthy controls without PCOS (Group II) were selected based on inclusion and exclusion criteria. The blood samples collected from all participants

were analyzed for FBS, lipid profile, Uric acid and hormones like TSH, Prolactin and Insulin.

#### Anthropometric parameters

The mean age and its standard deviation in Group I was 27.35±5.3 and I Group II was 29.4±4.37. The correlation of age in Group I and Group II the p value obtained was 0.715 (not significant). The mean systolic BP noted in Group I was 121.2±2.3 and in Group II was 108.4 ± 1.0. The mean diastolic BP noted in Group I was 79.8 ± 2.7 and in Group II was 72.5 ± 1.4. The correlation of systolic and diastolic BP among Group I and Group II showed a p value of <0.0001 (highly statistically significant). (Table 2)

PARAMETER	Group I [n=70] Mean ± S.D	Group II [n=70] Mean ± S.D	p Value
Age [Years]	27.35±5.3	29.4±4.37	0.715
Systolic BP	121.2±2.3	108.4 ± 1.0	<0.0001
Diastolic BP	79.8 ± 2.7	72.5 ± 1.4	<0.0001
<b>&lt;0.001&amp;&lt;0.0001 - highly statistically significant</b>			

Table 2: Showing mean, standard deviation & P value of Age and BMI in Group I and Group II

### Biochemical parameters

The comparison of biochemical parameters among participants of Group I and Group II results were as follows. The mean, standard deviation of Group I and

Group II were compared and the p value noted was <0.001 for glucose, triglyceride, HDL and VLDL; <0.0001 for total cholesterol and LDL. (Table 3)

PARAMETERS	Group I [n=70] Mean ± S.D	Group II [n=70] Mean ± S.D	p- Value
Glucose (mg/dl)	98±15	85.7±9	<0.001*
T. Cholesterol (mg/dl)	189±25.3	164±9.1	<0.0001**
TG (mg/dl)	130±15	115±24	<0.001*
HDL (mg/dl)	41±5.5	46±4.9	<0.001*
LDL (mg/dl)	118±20.9	80.3±11.8	<0.0001**
VLDL (mg/dl)	25.5±3	22.8±4	<0.001*
<b>&lt;0.001 &amp;&lt;0.0001- highly statistically significant</b>			

Table 3: Showing mean, standard deviation and p value of biochemical parameters in Group I and Group II

### Hormonal parameters

The comparison of hormonal parameters among Group I and Group II results were as follows. The mean, standard

deviation of Group I and Group II were compared and the p value obtained for TSH was 0.02, for prolactin <0.001, for Insulin and HOMA IR <0.01. (Table 4)

PARAMETERS	Group I [n=70] Mean ± S.D	Group II [n=70] Mean ± S.D	p -Value
TSH (mIU/L)	3.7 ± 2.4	2.17 ± 1.09	<b>0.02</b>
Prolactin (ng/ml)	18.3 ± 14.7	9.5 ± 3.7	<b>&lt;0.001</b>
Insulin (μU/ml)	12.1 ± 8.7	6.6 ± 2.5	<b>&lt;0.01</b>
HOMA-IR	2.7 ± 1.3	1.5 ± 0.9	<b>&lt;0.01</b>
<b>0.02, &lt;0.01- statistically significant &lt;0.001- highly statistically significant</b>			

Table 4: Showing mean, standard deviation and p value of hormonal parameters in Group I and Group II

In the present study we correlated Glucose with other biochemical parameters and found that the p value of total cholesterol, triglyceride, LDL and VLDL were <0.05 whereas HDL showed a p value of >0.05. Total cholesterol

when correlated with other biochemical parameters showed that glucose, triglyceride, HDL, LDL, VLDL had a p value of <0.05. (Table 5)

PARAMETERS	Glucose (mg/dl)		T. Cholesterol (mg/dl)	
	r- value	p- value	r- value	p- value
Glucose (mg/dl)	-	-	<b>0.240</b>	<b>0.004**</b>
T. Cholesterol (mg/dl)	0.240	<b>0.004*</b>	-	-
TG (mg/dl)	0.203	<b>0.016*</b>	0.267	<b>&lt;0.001*</b>
HDL (mg/dl)	-0.114	0.179	-0.176	<b>0.037**</b>
LDL (mg/dl)	0.359	<b>&lt;0.0001**</b>	0.813	<b>&lt;0.0001**</b>
VLDL (mg/dl)	0.209	<b>0.013*</b>	0.260	<b>0.002*</b>

Table 5: Showing correlation coefficient and p values of glucose and total cholesterol with biochemical parameters in patients with PCOS

We correlated the hormonal parameters with FBS, Total cholesterol and prolactin and found that TSH showed a p value of <0.05 with only prolactin and with FBS the p value was >0.05; prolactin showed a p value of >0.05 with

FBS and T. cholesterol. Insulin showed a p value of <0.05 with FBS, total cholesterol and prolactin. (Table 6) (Figure 1)

PARAMETER	FBS		T. Cholesterol		Prolactin	
	r- value	p- value	r- value	p- value	r- value	p- value
TSH (mIU/L)	0.076	0.373	-0.011	0.899	0.201	<b>0.019*</b>
Prolactin (ng/ml)	0.119	0.09	0.112	0.187	-	-
Insulin ( $\mu$ U/ml)	0.205	<b>0.015*</b>	0.249	<b>0.003*</b>	0.365	<b>&lt;0.01*</b>

Table 6: Showing correlation coefficient and p values of FBS, total cholesterol and prolactin with hormonal parameters in patients with PCOS

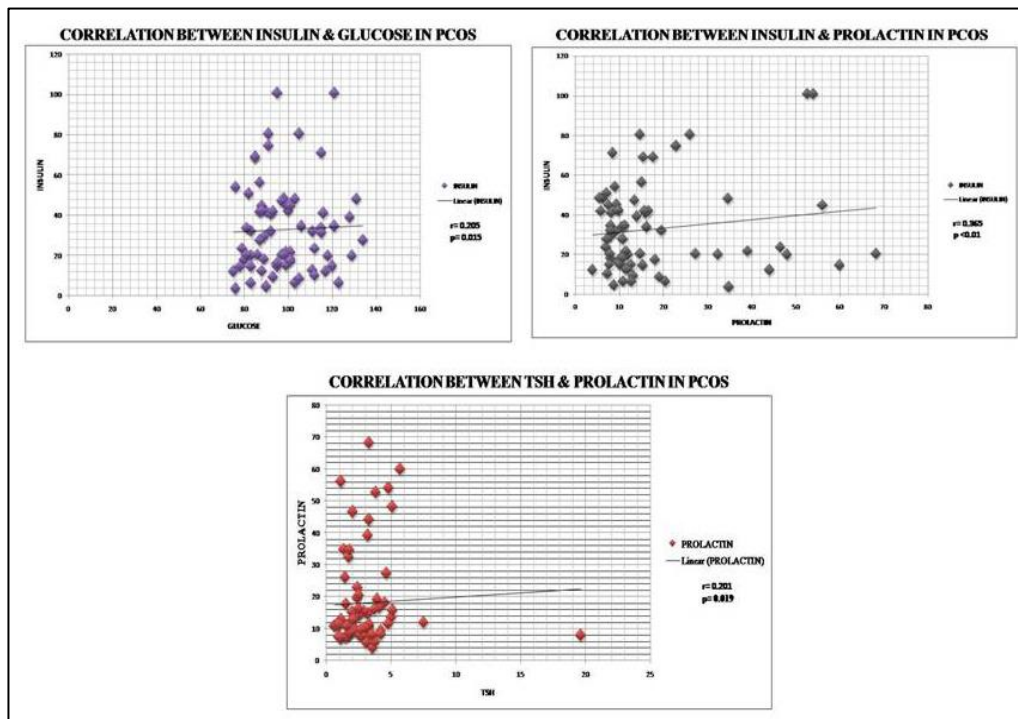


Figure 1: Showing plot graph depicting positive correlation between Insulin and Glucose; Insulin with Prolactin; TSH with Prolactin

### Discussion

The most common cause of menstrual disturbance during reproductive age in females is Polycystic Ovarian Cyst (PCOS) and it is also the most prevalent endocrine disorder. In mid 18<sup>th</sup> century multicystic or sclerocystic ovaries were recognized as pelvic pain or menorrhagia. In the early 20<sup>th</sup> century, prevailing hypotheses viewed them as resulting from inflammation due to infection, congestion due to pressure or partial torsion that disrupted

normal blood flow to the ovary, or from dystrophy due to abnormalities in ovarian nutrition. [5] Irving F Stein and Michael L. Leventhal were the 1<sup>st</sup> to describe a symptom complex associated with anovulation. [6]

Women with PCOS are at higher risk of developing hypertension, psychiatric disorders, lipid disorders, diabetes, cancer and osteoporosis. [3] Thus this study was aimed to analyze and correlate the biochemical parameters

and hormonal parameters of women with PCOS and to identify the risk factors associated with PCOS.

This prospective study was done on 140 subjects; 70 patients with diagnosed PCOS in Group I and 70 healthy controls without PCOS in Group II. 5ml of venous blood sample was drawn and analyzed for biochemical parameters like FBS, total cholesterol and hormonal parameters like TSH, prolactin and Insulin. The values obtained in Group I was compared with Group II. The results of comparison were correlated with Pearson correlation and p value of <0.05 was considered as statistically significant.

PCOS is traditionally diagnosed based on Rotterdam diagnostic criteria 2003. It is not enough to only diagnose PCOS. Clinical, biochemical assay help in early detection and thus aid in intervention and early management of PCOS. [7]

The participants of this study in Group I and Group II were females (100%). The mean age noted in Group I was 27.35 years and in Group II was 29.4 years. However, their comparison was not statistically significant. In the present study correlation of systolic and diastolic BP was highly statistically significant in patients with PCOS than in healthy controls. The finding of our study is similar to the observations of EO Talbott et al 2004. [8]

In the present study FBS was used as an indirect method for detection of insulin resistance which is the most prominent metabolic symptom of PCOS. In the present study the levels of plasma FBS was highly statistically significant in PCOS patients than healthy controls. This finding of ours was similar to the studies of Polak AM et al 2020. However, the findings of our study contradict to findings of VM Vinodhini et al 2004 who found no statistically significant association of FBS in PCOS patients and healthy subjects. Legro et al 2005 and Talbott et al 2007 showed that the conversion rates of normal glucose tolerance to impaired glucose tolerance was higher but statistically insignificant in women with PCOS compared to healthy controls. Women with PCOS are known to show high prevalence of impaired glucose tolerance and Type 2 diabetes. The findings of our study show that PCOS is associated with glucose intolerance resulting from defects in insulin action and  $\beta$ -cell dysfunction. [9-11]

In the present study the serum lipid profile estimated among PCOS patients' healthy controls. It was found that total cholesterol, triglycerides, HDL, LDL and VLDL were highly significantly higher in PCOS patients than healthy controls. The results of the study were consistent with findings of Olivier Valkenburg et al 2008. [12] Abnormal lipid metabolism is one of the main metabolic

characteristics of PCOS patients. Dyslipidemia is a very common metabolic abnormality in women with polycystic ovary syndrome (PCOS) and obesity can alter lipoprotein lipid profiles and glucose metabolism in PCOS. Decrease in HDL and increase in TG levels are well known lipid profile characteristics in women with PCOS. [13]

Hypothyroidism with increased TSH level may lead to failure in regular ovulation in reproductive women. Raised TSH levels decrease energy expenditure, promote adiposity, impair glucose and lipid metabolism. [14] In the present study the TSH level was increased in PCOS patients as against healthy controls and was statistically significant. There was a prevalence of hypothyroidism in 12% PCOS women and 2.8% healthy controls. The finding of the study was consistent with findings of Dahiya et al 2012, Garber et al 2012, and Janssen et al 2004. [15-17]

Uric acid is a metabolic end product of purine metabolism. Serum uric acid is inversely proportional to the insulin sensitivity in patients with metabolic syndrome. Elevated level of serum uric acid is considered to be a simple marker of insulin resistance. [18] In the present study though the serum uric acid level was slightly elevated in women with PCOS when compared to healthy controls but was not statistically significant. The finding of this study was consistent with studies of Anttila et al 1996 and Manuel Luque-Ramirez et al 2008. [19,20]

Prolactin is one of the pituitary hormones which causes irregular menstrual cycle, obesity and infertility. It plays a role as a PCOS interventional hormone and has a potent lipogenic and diabetogenic factor. Increased prolactin inhibits 3- $\beta$ -hydroxysteroid dehydrogenase activity leading to intraovarian hyperandrogenemia. [21] High prolactin level is associated with high level of serum TSH and thus increased prolactin level is associated in 5-30% of patients with PCOS. [22,23] Increased prolactin level decreases glucose tolerance via an increase in insulin resistance. [24] In the present study serum prolactin level was increased in patients with PCOS than in comparison to healthy controls and was found to be highly statistically significant. Our results were consistent with the findings of Roy et al 2014 and Cristianne et al 2014 who found a statistically significant level of prolactin in PCOS women compared to controls. [25,26] Even studies by Ansam et al 2006, Soodabehet al 2010 and Sunita et al 2012 found similar findings in their studies but the results were not statistically significant. [27-29]

Insulin also regulates glucose metabolism, inhibits lipolysis and stimulates amino acid transport. Insulin resistance is a metabolic disorder caused by the impairment of insulin function in inducing glucose uptake and utilization. [30] In the present study serum insulin

level in PCOS women was compared to that of healthy controls and found that serum insulin level was significantly higher in PCOS women. The findings of our study correlated with the findings of Roy et al. Dunaif et al 1987 observed that PCOS related insulin resistance contributed to approximately 10% of cases of glucose intolerance in premenopausal women. [31]

HOMA-IR is a sensitive method for evaluation of IR in PCOS and was used to identify IR in the present study. It was observed that women with PCOS had higher values of HOMA-IR compared to healthy controls. Among 70 healthy controls elevated TSH levels was seen in 4.2%; hyperinsulinemia in 22.8% and hypothyroidism in 10%. This shows that thyroid changes may aggravate IR in PCOS. In the present study it was observed that the level of HOMA-IR was found to be significantly higher in women with PCOS when compared to healthy controls. The findings were consistent with studies of Shou-Kul et al 2012 and Puder et al 2005. [32,33] Sunita et al concluded in their study that insulin resistance is common in Indian PCOS women and was independent of obesity. [34]

## References

1. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovarian syndrome: etiology, pathogenesis and diagnosis. *Nat Rev Endocrinol* 2011;7:219-231.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome, *Fertil Steril* 2004;19:41-47.
3. Rajbanshi I, Sharma VK, Tuladhar ET, Bhattarai A, Raut M, Dubey RK. Metabolic and biochemical profile in women with polycystic ovarian syndrome attending tertiary care centre of central NEPAL. *BMC Women's Health* 2023; 23:208.
4. Goudas VT, Dumesic DA. Polycystic ovary syndrome. *Endocrinol Metab Clin North Am.* 1997;26:893-912.
5. Fogue E and Massabuau G. L'ovaire a petits kystes., *Rev GynecolChirurgAbdom* 1910; 14:97-152.
6. Stein IF, Leventhal ML, Amenorrhea associated with bilateral polycystic ovaries, *Am J ObstetGynecol*1935;29:181-191.
7. Ahmed AA, Moselhy SS, Kumosani TA, Huwait EA, AL Ghamdi MA, Al-Madani KA et al. Ultrasonographic and biochemical assessments as early prediction of polycystic ovarian syndrome in obese women. *AF Health Sci* 2020;20:6767-681.
8. E. O. Talbott, J. V. Zborowski, J. R. Rager, M. Y. Boudreaux, D. A. Edmundowicz, And D. S. Guzik, Evidence for an Association between Metabolic Cardiovascular Syndrome and Coronary and Aortic Calcification among Women with Polycystic Ovary Syndrome. *The Journal of Clinical Endocrinology & Metabolism* 2004; 8911:5454-5461.
9. Polak AM, Adamska A, Krentowska A, Lebkowska A, Hryniewicka J, Adamski M et al . Body composition, serum concentrations of androgens and insulin resistance in different polycystic ovarian syndrome phenotypes. *J CLin Med* 2020;9:732.
10. V.M.Vinothini, V. Devisri, W.Ebenezer William, M.Muthulakshmi, Anjalakshichandrasekar and S.Gnanasambandam. High Sensitive C reactive Protein and Apolipoprotein B levels in Polycystic ovary syndrome: *International Journal of Pharma and Bio Sciences* 2012;3:719-724.
11. Legro RS, Castracane VD and Kauffman RP. Detecting insulin resistance in polycystic ovary syndrome: purposes and pitfalls. *ObstetGynecolSurv*2004;59: 141154.
12. Valkenburg O, Steegers-Theunissen RP, Smedts HP, Dallinga-Thie GM, Fauser BC, Westeryeld EH et al A more atherogenic serum lipoprotein profile is present in women with polycystic ovary syndrome: A case-control study. *J Clin Endocrinol Metab* 2008 93:470-476.
13. Legro RS, Kunselman AR, Dunaif A. Prevalence and predictors of dyslipidemia in women with polycystic ovary syndrome. *Am J Med* 2001;111:607-13.
14. Zhang J, Wu H, Ma S, Gao L, Yu C, Jing F et al . TSH promotes adiposity by inhibiting the browning of white fat. *Adipocyte* 2020;9:264-278.
15. Dahiya K, Sachdeva A, Singh V, Dahiya P, Singh R, Dhankhar R, et al. Reproductive Hormone and Thyroid Hormone Profile in Polycystic Ovarian Syndrome. *Webmedcentral Endocrinology* 2012;3: WMC 003455.

16. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, Mechanick JI, et al. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. *Endocrine Practice*. 2012; 18:988-1028.
17. Janssen OE, Mehlmauer N, Hahn S, Offner AH, Gartner R. High prevalence of autoimmune thyroiditis in patients with polycystic ovary syndrome. *Eur J Endocrinol* 2004;150:363-9.
18. Vuorinen-Markkola, H Yki-Jarvinen. Hyperuricemia and insulin resistance. *J Clin Endocrinol Metab*1994;78:25-29.
19. Anttila L, Rouru J, Penttila T, Irjala K. Normal serum uric acid concentrations in women with polycystic ovary syndrome. *Human Reproduction* 1996; 11:2405-2407.
20. Luque-Ramirez M, Alvarez-Blasco F, Rivera MGU, Escobar-Morreale HF. Serum uric acid concentration as non-classic cardiovascular risk factor in women with polycystic ovary syndrome: effect of treatment with ethinyl-estradiol plus cyproterone acetate versus metformin. *Human Reproduction* 2008;23:1594-1601.
21. Roy George K., Malini N. A. The prevalence and etiology of polycystic ovarian syndrome (PCOS) as a cause of female infertility in central Travancore. *The Bioscan* 2014;9:01-06.
22. Serri O, Chik CL, Ur E, Ezzat S. Diagnosis and management of hyperprolactinemia. *Canadian Medical Association Journal*. 2003;169:575-81.
23. Sharma N, Baliarsingh S, Kaushik GG. Biochemical association of hyperprolactinemia with hypothyroidism in infertile women. *Clinical laboratory*. 2012;58:805-10.
24. Tahia H. Saleem, Howaida A. Nafady, Housny A. Hassan. Serum Prolactin and Blood Glucose Levels Before and After an Oral Glucose-load in Patients with Diabetes Mellitus and Liver Cirrhosis. *Asian Journal of Medical Sciences* 2013;5: 09-18.
25. Roy George K., Malini N. A. The prevalence and etiology of polycystic ovarian syndrome (PCOS) as a cause of female infertility in central Travancore. *The Bioscan* 2014;9:01-06.
26. Cristianne Serafim da Silva Feuser, Jacklyne Silva Barbosa, Evelyn Barzotto da Silva, Sebastiao Freitas de Medeiros. Current insights into gonadotropic pituitary function in the polycystic ovary syndrome. *Asian Pacific Journal of Reproduction* 2014;3:64-7.
27. Ansam A. Al-Bayatti. Insulin resistance and upper-body obesity in polycystic ovary syndrome. *Middle East Fertility Society Journal* 2006;11:202-9.
28. Soodabeh Zandi, SaeidehFarajzadeh, Hamideh Safari. Prevalence of polycystic ovary syndrome in women with acne: hormone profiles and clinical findings. *Journal of Pakistan Association of Dermatologists* 2010;20:194-198.
29. Sunita J Ramanand, Balasaheb B Ghongane, Jaiprakash B Ramanand, Milind H Patwardhan, Varsha M Patwardhan, Ravi Ghanghas et al. Hormonal Profile of Polycystic Ovary Syndrome (PCOS) In Indian Women. *Research Journal of Pharmaceutical, Biological and Chemical Sciences* 2012;3:1159-72.
30. Dunaif A, Graf M, Mandeli J, Laumas V, Dobrjansky A. Characterization of groups of hyperandrogenic women with acanthosis nigricans, impaired glucose tolerance and/or hyperinsulinemia. *J Clin Endocrinol Metab*1987;65:499-507.
31. Roy George K., Malini N. A. The prevalence and etiology of polycystic ovarian syndrome (PCOS) as a cause of female infertility in central Travancore. *The Bioscan* 2014;9:01-06.
32. Xiang SK, Hua F, Tang Y, Jiang XH, Zhuang Q, Qian FJ. Relationship between serum lipoprotein ratios and insulin resistance in polycystic ovary syndrome. *International Journal of Endocrinology*. 2012; 2012, 173281:1-4
33. Puder JJ, Varga S, Kraenzlin M, Geyter CD, Keller U, Muller B. Central fat excess in polycystic ovarian syndrome: relation to low-grade inflammation and insulin resistance. *J Clin Endocrinol Metab* 2005; 90:6014-21.
34. Ramanand SJ, Ghongane BB, Ramanand JB, Patwardhan MH, Patwardhan VM, Ghanghas R et al. Hormonal Profile of Polycystic Ovary Syndrome (PCOS) In Indian Women. *Research Journal of Pharmaceutical, Biological and Chemical Sciences* 2012;3:1159-72.

#### LIST OF TABLES

- Table 1: showing the various biochemical assay done and their principle in the present study  
Table 2: showing mean, standard deviation & P value of Age and BMI in Group I and Group II  
Table 3: showing mean, standard deviation and p value of biochemical parameters in Group I and Group II  
Table 4: showing mean, standard deviation and p value of hormonal parameters in Group I and Group II  
Table 5: showing correlation coefficient and p values of glucose and total cholesterol with biochemical parameters in patients with PCOS  
Table 6 showing correlation coefficient and p values of FBS, total cholesterol and prolactin with hormonal parameters in patients with PCOS

#### LIST OF FIGURES

- Graph1: showing plot graph depicting positive correlation between Insulin and Glucose; Insulin with Prolactin; TSH with Prolactin