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# A study of various endocrinal hormones and insulin resistance in women of polycystic ovarian syndrome (PCOD) in southern Rajasthan, India

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**Abstract**
**Background:** Polycystic ovary Syndrome (PCOS) is one of the most common female endocrine disorders. PCOD produces symptoms in approximately 5% to 10% of women of reproductive age (12–45 years old). It is thought to be one of the leading causes of female subfertility.
**Objectives:** The objective of the study is to establish correlation among insulin, testosterone, estrogen & progesterone hormones, lipid profile among the women with polycystic ovary syndrome (PCOS), in order to evaluate their diagnostic and prognostic significance
**Methodology:** 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 insulin, HOMA-IR, Testosterone, FSH,LH and Prolactin**.**
**Result:** The Mean level of Fasting Blood sugar, S.cholesterol, S.triglyceride S.insulin, S.testosterone, S.FSH, S.LH, S.prolactin 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:** From our study I would like to conclude that as PCOS is a very complicated endocrine disorder. Blood tests to measure hormone levels, an ultrasound to look at your reproductive organs and thorough personal and family histories should be completed before a PCOS diagnosis is confirmed. Depending on your symptoms, your physician will determine exactly which tests are necessary. Assessing hormone levels serves two major purposes. First of all, it helps to rule out any other problems that might be causing the symptoms. Secondly, together with an ultrasound and personal and family histories, it helps your doctor confirm that you do have PCOS.

**Keywords:** PCOD, insulin, HOMA-IR, testosterone, FSH, LH, prolactin

## 1. Introduction
Polycystic ovary Syndrome (PCOS) is a condition that affects women of menstruating age. Although it is not life-threatening, it can be uncomfortable and can cause fertility challenges for some women. Women with polycystic ovary Syndrome (PCOS) can get pregnant, but their risk of pregnancy complications is higher than average [1]. The polycystic ovary syndrome (PCOS) is a mostly hyper androgenic disorder and is possibly the most common endocrinopathy of premenopausal women [2]. The primary defect in polycystic ovary syndrome (PCOS) appears to be an exaggerated androgen synthesis (Testosterone) and secretion by the ovaries and the adrenal glands (according to "NIH criteria", 1990) [2, 3]. In a substantial proportion of polycystic ovary syndrome (PCOS) patients, the primary defect in androgen secretion is triggered by factors such as the hyperinsulinism resulting from insulin resistance and/or the secretion of metabolically active substances by visceral adipose tissue, because these factors may facilitate androgen synthesis at the ovaries and the adrenals of predisposed women [2].
Therefore, the present investigations will be carried out to assess estrogen, progesterone, estrogen-dominance, Testosterone, LH, FSH & insulin hormones level, HOMA-IR level. Subsequently regular assessing of sugar glucose, lipid profile and differential diagnosis of prolactin (PRL)in clinical biochemistry laboratory is important to monitor & study the effect of these parameters among normal and polycystic ovary Syndrome (PCOS) women & its adverse consequences.

## 2. Material & Method
This prospective study was conducted at Department of Biochemistry and Department of Obstetrics & Gynaecology, Geetanjali Medical College & 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, 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 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, 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 GOD POD method and lipid profile (S.cholesterol, Triglyceride, HDL,VLDL,LDL) measured by enzymatic colorimetric method from all samples.

-Fasting Insulin level estimation was done by enzyme linked immune assay (ELISA) method based electrochemilumnescence and HOMA-IR will be estimated by calculation (fasting sugar×fasting insulin/22.5).

-Various Endocrinal Hormones like Estrogen(E2), progesterone, estrogen-dominance, Testosterone, LH, FSH, Insulin and Prolactin was Measured by enzyme linked immune assay (ELISA) method based on electrochemilumnescence 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 independent student's t test. By using't' 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.

## 3. Results & Discussion

Infertility, hirsutism, and oligomenorrhea were more common among the subjects with PCOS, but there was a considerable spontaneous restitution of cyclic regularity with time. Women with PCOS were more often hysterectomized and entered menopause later compared with referents. The hormone data show a typical profile for PCOS. Compared with referents women with PCOS showed marked increase in prevalence of central obesity, higher basal serum insulin concentrations, and a higher prevalence of diabetes mellitus and hypertension [6, 5].

**Table 1:** Age wise distribution of participants

| Group | Number(n) | Mean Age (Yr) |
|---|---|---|
| Group 1(PCOD ) | 300 | 27 ± 5.4 |
| Group 2(Control) | 200 | 24.98 ± 4.13 |

**Table 2:** Location wise distribution of participants

| Location | Group 1(PCOD ) | Group 2(Control) |
|---|---|---|
| Rural | 114(38%) | 72(36%) |
| Urban | 186(62%) | 128(64%) |
| Total | 300(100%) | 200(100%) |

**Table 3:** Comparison of weight between case and control group

| Group | Number(n) | Mean wt (kg) |
|---|---|---|
| Group 1(PCOD ) | 300 | 56.91 ± 8.24 |
| Group 2(Control) | 200 | 45.36 ± 5.8 |

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

| Group | Number(n) | Mean W/H ratio |
|---|---|---|
| Group 1(PCOD ) | 300 | 0.89 ± 0.13 |
| Group 2(Control) | 200 | 0.79 ± 0.0.3 |

**Table 5:** Comparison of BMI between case and control group

| Group | Number(n) | Mean BMI |
|---|---|---|
| Group 1(PCOD ) | 300 | 22.70 ± 3.73 |
| Group 2(Control) | 200 | 17.72 ± 2.41 |

**Table 6:** Comparison of Marital status between case and control group

| Group | Number(n) | Married | Single |
|---|---|---|---|
| Group 1(PCOD ) | 300 | 200(66.7%) | 100(33.3%) |
| Group 2(Control) | 200 | 118 (59%) | 82(41%) |

**Table 7:** Comparison of systolic Blood pressure (SBP) between case and control group

| | | | Group | | Total |
|---|---|---|---|---|---|
| | | | Control | Cases | |
| SBP | 100-130 | Count | 176 | 135 | 311 |
| | | % within Group | 88.0% | 45.0% | 62.2% |
| | 131-150 | Count | 24 | 128 | 152 |
| | | % within Group | 12.0% | 42.7% | 30.4% |
| | >150 | Count | 0 | 37 | 37 |
| | | % within Group | 0.0% | 12.3% | 7.4% |
| Total | | Count | 200 | 300 | 500 |
| | | % within Group | 100.0% | 100.0% | 100.0% |

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

| | | | Group | | Total |
|---|---|---|---|---|---|
| | | | Control | Cases | |
| DBP | <80 | Count | 161 | 148 | 309 |
| | | % within GROUP | 80.5% | 49.3% | 61.8% |
| | 81-100 | Count | 39 | 147 | 186 |
| | | % within GROUP | 19.5% | 49.0% | 37.2% |
| | >100 | Count | 0 | 5 | 5 |
| | | % within GROUP | 0.0% | 1.7% | 1.0% |
| Total | | Count | 200 | 300 | 500 |
| | | % within GROUP | 100.0% | 100.0% | 100.0% |

**Table 9:** 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 10:** Showing valid Hirsutism status of Case group

| Total Counts | Hirsutism | | Non Hirsutism | |
|---|---|---|---|---|
| | Counts | valid % | Counts | valid % |
| Cases (300) | 160 | 53% | 140 | 47% |

**Table 11:** Comparison of various biochemical parameter 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. cholesterol (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 12:** 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. Insulin(U/ML) | Case | 300 | 15.52± 6.29 | <0.001 |
| | Control | 200 | 7.44 ± 2.04 | |
| S. Estradiol(pg/ml) | Case | 300 | 235.32 ± 90.29 | <0.001 |
| | Control | 200 | 60.99± 19.69 | |
| S. Progesteron(ng/ml) | Case | 300 | 1.72 ± 2.26 | <0.001 |
| | Control | 200 | 2.58 ± 3.13 | |
| HOMA-IR | Case | 300 | 75.45 ± 41.15 | <0.001 |
| | Control | 200 | 31.83± 10.69 | |

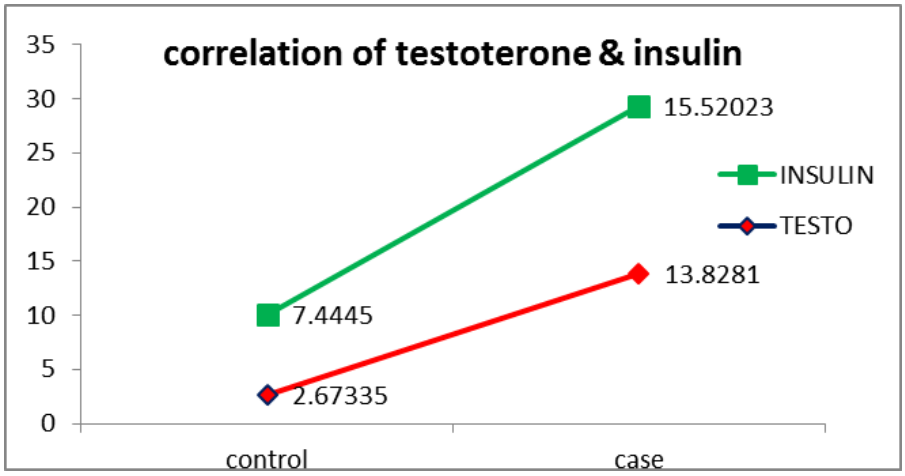


**Fig 1:** Showing correlation of testoterone & insulin between case and control group

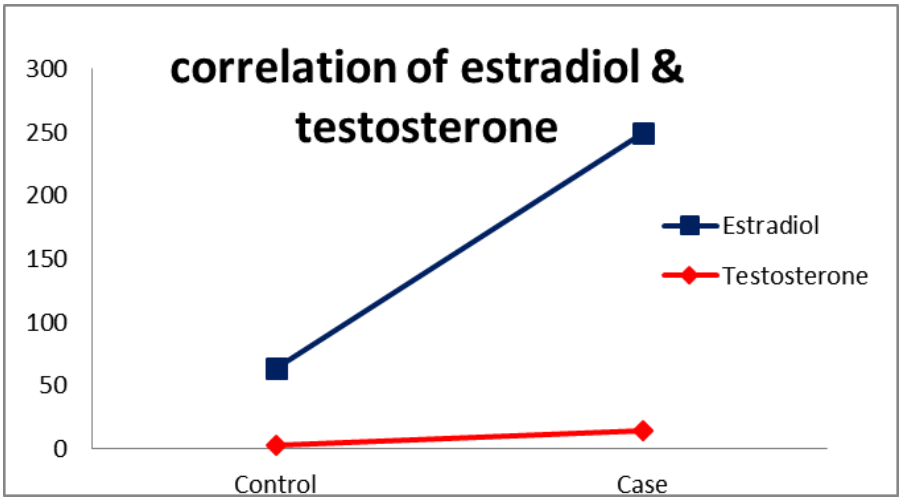


**Fig 2:** Showing correlation of estradiol & Testosterone between case and control group

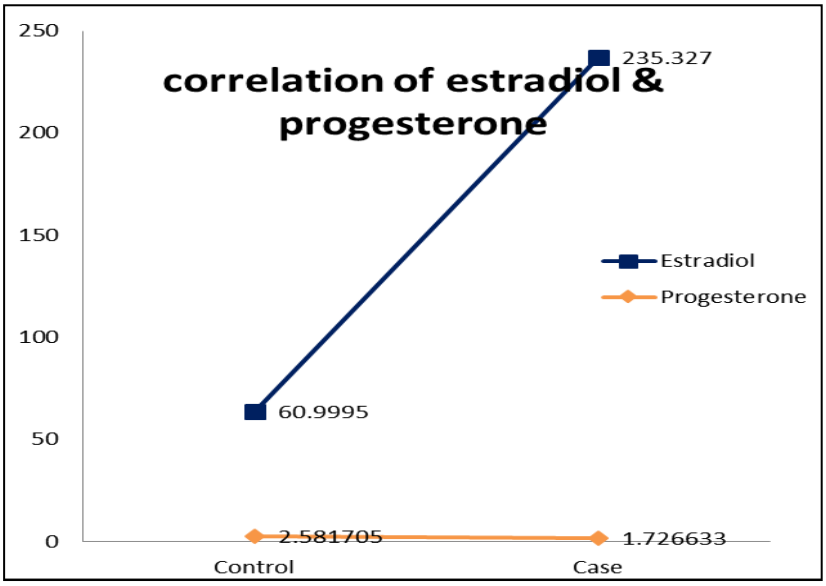


**Fig 3:** Showing correlation of estradiol & progesterone between case and control group

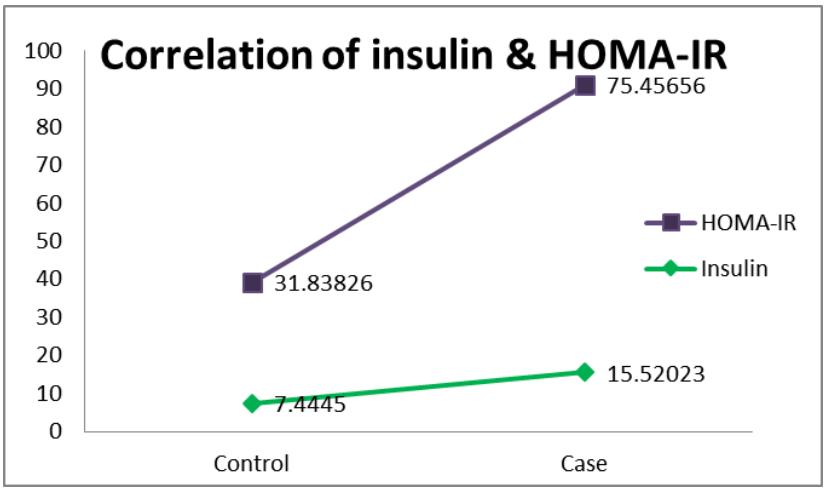


**Fig 4:** Showing Correlation of insulin & HOMA-IR between case and control group

- 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 11)
- 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 11)
- 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 12)
- 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 12)
- Testosterone is higher (mean value 13 ± 6.3) in Cases than Controls (mean value = 2.67 ± 1.4). (Table 12)
- Insulin hormone is higher (mean value 15.5 ± 6.2) in Cases than Controls (mean value = 7.4 ± 2.0). (Table 12)
- Estradiol is higher (mean value 235 ± 90.2) in Cases than Controls (mean value = 60.9 ± 19.6). (Table 12)
- Progesterone is higher (mean value 2.58 ± 3.1) in Controls than Cases (mean value = 1.72 ± 2.2). (Table 12)
- HOMA-IR is higher (mean value 75 ± 41.1) in Cases than Controls (mean value = 31.8 ± 10.6). (Table 11)

Hardiman P. *et al*., Ricardo Azziz *et al*. study show that polycystic ovary syndrome (PCOS) is a genetically complex endocrine disorder of women of uncertain etiology and is a common cause of anovulatory infertility, menstrual dysfunction, and hirsutism. PCOS appears to be associated with an increased risk of metabolic aberrations, including insulin resistance and hyperinsulinism, type 2 diabetes mellitus, dyslipidemia, cardiovascular disease, and endometrial carcinoma [4-10].

Cahill D. *et al*. study shows overweight and obesity: a common finding in women with PCOS because their body cells are resistant to the sugar-control hormone insulin. This insulin resistance prevents cells using sugar in the blood normally and the sugar is stored as fat instead [11, 12, 13, 14].

By Hull M study, A high LH: FSH ratio was the most frequently found abnormality (raised in 68.4% of patients) followed by LH (65.8%), free Testosterone (FT, calculated from total Testosterone (T). Each of these four estimations was above the normal ranges in 25% of patients [15].

By Hardiman P. *et al*., disorders of lipid metabolism (dislipidemia), cholesterol and triglycerides, PCOS patients show decreased removal of atherosclerosis-inducing remnants, seemingly independent of insulin resistance/Type II diabetes. Cardiovascular disease, with a meta-analysis estimation, a 2-fold risk of arterial disease for women with PCOS relative to women without PCOS, independent of BMI [4, 16, 17].

Dunaif *et al*., Hyperinsulinaemia and insulin resistance are a well-known feature in polycystic ovarian syndrome (PCOS). Whether hyperinsulinaemia in PCOS is primarily due to a defect in insulin action to increased insulin secretion, to decreased hepatic clearance of insulin, or to an interaction between all these disorders is, however, not clear [18, 19, 20].

Legro RS *et al*. The homeostatic model assessment (HOMA), a more complex fasting calculation, has been compared to clamp techniques with good results. HOMA is the product of fasting glucose (mg/dL) and insulin (μU/mL) divided by a constant[21-24]. One major limitation of HOMA rests on the previous reflection that many young PCOS women display stimulated but not fasting metabolic abnormalities [22, 23, 24].

## 4. Conclusion

From our study I would like to conclude that as PCOS is a very complicated endocrine disorder. Blood tests to measure hormone levels, an ultrasound to look at your reproductive organs and thorough personal and family histories should be completed before a PCOS diagnosis is confirmed. Depending on your symptoms, your physician will determine exactly which tests are necessary. Assessing hormone levels serves two major purposes. First of all, it helps to rule out any other problems that might be causing the symptoms. Secondly, together with an ultrasound and personal and family histories, it helps your doctor confirm that you do have PCOS.

## 5. References


1. Garruti G, Depalo R, Vita MG, Lorusso F, Giampetruzzi F, Damato AB *et al*. Adipose tissue, metabolic syndrome and polycystic ovary syndrome: from pathophysiology to treatment. Reprod biomed. Online. 2009; 19:552-563.
2. Martínez-Bermejo E, Luque-Ramírez M, Escobar-Morreale HF. Obesity and the polycystic ovary syndrome.
3. Artini PG, Di Berardino OM, Simi G, Papini F, Ruggiero M, Monteleone P. Best methods for identification and treatment of PCOS. Minerva Ginecol. 2010; 62(1):33-48.
4. Hardiman P, Pillay OC, Atiomo W. Polycystic ovary syndrome and endometrial carcinoma.". Lancet. 2003; 361(9371):1810-2.
5. Ricardo Azziz, Keslie Woods S, Rosario Reyna, Timothy Key J, Eric Knochenhauer S *et al*. The Prevalence and Features of the Polycystic Ovary Syndrome in an Unselected Population. 2004, 89(6).
6. Hull MG. Epidemiology of infertility and polycystic ovarian disease: endocrinological and demographic studies. Gynecol Endocrinol. 1987; 1:235-245. [Cross Ref] [Medline]
7. Azziz R. The evaluation and management of hirsutism. Obstet Gynecol. 2003; 101:995-1007. [Cross Ref] [Medline]
8. Ovalle F, Azziz R. Insulin resistance, polycystic ovary syndrome, and type 2 diabetes mellitus. Fertil Steril. 2002; 77:1095-1105 [CrossRef] [Medline]
9. Wild RA. Long-term health consequences of PCOS. Hum Reprod Update. 2002; 8:231-241.[CrossRef] [Medline]
10. Legro RS. Polycystic ovary syndrome and cardiovascular disease: a premature association? Endocr Rev. 2003; 24:302-312. [Abstract] [Medline]
11. Cahill D. PCOS. Clin Evid (Online). 2009; 2:1408.
12. Wild S, Pierpoint T *et al*. Long-term consequences of polycystic ovary syndrome: results of a 31 year follow-up study. Human Fertility. 2000; 3:101-05.
13. Winters SJ, Talbott E *et al*. Serum Testosterone levels decrease in middle age in women with the polycystic ovary syndrome. Fertil Steril. 2000; 73:724-29.
14. Atiomo W, Khalid S, Parameshweran S, Houda M, Layfield R. Proteomic biomarkers for the diagnosis and risk stratification of polycystic ovary syndrome: a systematic review. BJOG. 2009; 116:137-143.
15. Hull MGR, Savage PE, Bromham DR. Anovulatory and ovulatory infertility: results with simplified management. Br. Med. J. 1982; 284:1681-85.
16. Hardiman P, Pillay OC, Atiomo W. Polycystic ovary syndrome and endometrial carcinoma. Lancet. 2003; 361(9371):1810-2.
17. New MI. Nonclassical congenital adrenal hyperplasia and the polycystic ovarian syndrome. Annals of the New York Academy of Sciences 687 (1 Intraovarian). 1993, 193-205.
18. Dunaif A, Segal KR, Futterweit W, Dobrjansky A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. Diabetes. 1989; 38:1165-1174.
19. 271-Ciampelli M, Fulghesu AM, Cucinelli F *et al*. Heterogeneity in beta cell activity, hepatic insulin clearance and peripheral insulin sensitivity in women with polycystic ovary syndrome. Hum. Reprod. 1997; 12:1897-1901.
20. 272-Holte J, Bergh T, Berne C *et al*. Enhanced early insulin response to glucose in relation to insulin resistance in women with polycystic ovary syndrome and normal glucose tolerance. J. Clin. Endocrinol. Metab. 1994a; 78:1052-1058.
21. Pedersen SD, Brar S, Faris P, Corenblum B. Polycystic ovary syndrome: validated questionnaire for use in diagnosis. Canadian Family Physician. 2007; 53(6):1042-7, 1041
22. Bhattacharya SM, Ghosh M. Insulin resistance and adolescent girls with polycystic ovary syndrome. J Pediatr Adolesc Gynecol. 2010; 23:158-161.
23. 81-Polson DW, Adams J, Wadsworth J, Franks S. Polycystic ovaries: a common finding in normal women. Lancet. 1988; 1(8590):870-872.
24. 298-Vrbíková J, Cibula D, Dvoráková K, Stanická S, Sindelka G, Hill M *et al*. Insulin sensitivity in women with polycystic ovary syndrome. J Clin Endocrinol Metab. 2004; 89:2942-2945. [PubMed]