

## Effect of Polycystic Ovary Syndrome and Hormones Disorder on Enzymes Gammaglutamyl Transferase, Oxaloacetic Transaminase, and Proteins

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### Abstract

Polycystic ovary syndrome (PCOS) is complex metabolic disorder of endocrine gland in women of reproductive age. The present work illustrate the effect of (PCOS) and hormone disturbance on serum enzymes activities, iron status parameters and hormone profile in patient women suffering from, PCOS, acute kidney disease (AKD) with PCOS, acute kidney disease and iron deficiency anemia (IDA). A total of 160 women, include 135 patients and 25 of control apparently healthy group, patients were subdivided into four groups, (G1) consisted of 50 patients of PCOS, (G2) of 30 patients of PCOS with AKD, (G3) of 30 patient of AKD and (G4) involve 25 patients of IDA. The specimens were collected from different hospital in Baghdad city. The patients and control were investigated for enzymes, gammaglutamyltransferase (GGT), glutamic oxaloacetic transaminase (GOT) or aspartate transaminase (AST) & glutamicpyruvic transaminase (GPT) or alanine transaminase (ALT) lactate dehydrogenase (LDH), total serum proteins (TSP), albumin, globulin, total cholesterol (TC), triglyceride (TG), urea, creatinine and uric acid. Also iron status parameters (serum iron, transferrin saturation percent (TS%), transferrin concentration, and total iron binding capacity (TIBC) and ferritin concentration) were estimated. As well as hormone profile (testosterone (TT), Follicle stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PL), estradiol (E), and progesterone (PRG)) were estimated Results reveals highly significant increases for AST or GOT, ALT or GPT, and GGT activities  $P < 0.001$ , and a change in LDH of G1 and G2 patients were highly increased  $P < 0.001$  as compared with normal. Remarkable rising was detected in TSP of (G1) patient while there is significant decrease in proteins level of (G2) patients in comparison with normal. Patients of (G3) show significant difference in GGT, LDH and protein. There is significant change in LDH and albumin in (G4), and no change in AST, ALT, GGT, and proteins as compared with normal. Triglyceride (TG), total cholesterol (TC), creatinine show very highly significant change for patients of (G1, G2, G3&G4). Patients women with PCOS show no remarkable change in uric acid and urea and a change in creatinine concentration. Serum iron parameters were changed significantly in patients of (G1, G2, G3, and G4) in comparison with healthy control groups. Hormones levels were significantly different in PCOS patients as compared with normal individual. In conclusion there were remarkable changes in the activities of AST, ALT, GGT, and LDH. This is the first time for the estimation of a change in LDH activity in women with (PCOS) this could be due to genetic and environmental factor and gene mutation. Hormone levels and iron status parameter changed remarkably may be due to metabolic disturbance and abnormality in the function of hypothalamic neurotransmitter. [DOI: [10.22401/JNUS.20.2.05](https://doi.org/10.22401/JNUS.20.2.05)]

Keyword: Polycystic ovary syndrome, GGT, Iron status, LDH, TG.

### Introduction

Polycystic ovary syndrome (PCOS) is heterogenous complex endocrine disorder characterized by, hyperinsulinemia, hyperandrogenemia, resistance of insulin, and chronic anovulation. It is increasingly recognized for women of reproductive age between 18-40 years, it affects 5%-18% of all women [1]. The primary pathophysiological defect is unknown, and not fully understood,

resistance of insulin, androgen excess and impaired gonadotropin dynamic and metabolic syndrome play a role in the development of this disease [2]. Levels of the sex hormones progesterone and estrogen are out of balance in condition of PCOS, this can cause problems with women's menstrual cycle, fertility, leads to growth of ovarian cysts (benign masses on the ovaries). [3], it has multiple components, metabolic, reproductive, and cardiovascular

function, with health implications across the life span [3, 4]. Resistance of insulin, elevated ratio of LH to FSH, infertility, adiposity of abdominal, abnormalities vascular, dyslipidemia and disturbances metabolism, of carbohydrate including glucose tolerance impaired and are common in PCOS women. Hyperandrogenemia, excess of androgen favoring the deposition of abdominal fat [5-8]. Abnormality of another hormone in PCOS women is excessive production of LH hormone, which is involved in ovarian stimulation to produce hormones and is released from the pituitary gland in the brain [4,9]. In addition, the released level of hormone Latinizing in the brain by the pituitary gland that is involved in ovarian hormone production is elevated. Others factors contributing possibly in PCOS development include decreased level of chronic inflammation in the body and exposure of fetal to male hormones [2,9,10]. In addition, increased levels of several inflammatory biochemical markers and cardiovascular risk and thrombotic are more prevalent in PCOS patients [11]. Evaluation of endocrine may reveal elevated LH levels and androgens; the ovarian follicles poorly develop commonly this disorder forming multiple cysts. Hypertriglyceridemia, increased levels of low-density lipoprotein (LDL) and very-low-density lipoprotein cholesterol (VLDL) with decreased high-density lipoprotein cholesterol (HDL) levels also predispose patients in the PCOS to vascular disease [12]. The abnormal typical pattern of biochemical enzymes liver involves of increasing predominantly serum aminotrasferases, with rising of alanine aminotransferase (ALT), relative to aspartate aminotransferase (AST), accompanied by elevated  $\gamma$ -glutamyltransferase ( $\gamma$ GT levels. [13,14]. Iron is strong pro-oxidant, and dietary micronutrient, caused increasing catalyzed oxidative stress reactions. A vital role of iron in erythrocyte maturation and production of hemoglobin, these are a cause of iron deficiency anemia which result from iron homeostasis abnormality, it is controlled by complex mechanism erythropoietic activity, hypoxia, iron stores, and inflammation [6,10]. Elevated serum body iron concentration put the patients at increased risk for disease of

cardiovascular (CVD) [15]. The content of iron body is regulated tightly by modulating the absorption of iron and from other sources, absence of menstruation, lactation, pregnancy [16]. Ferritin serve as body marker of iron stores in circulation, it is the primary cellular protein for iron storage, and in acute phase reactant, thus may overestimate stores iron in inflammatory state [17]. The transferrin saturation increases in situations when supply iron exceeds demands iron, levels of transferrin are used typically for iron overload diagnosis rather than iron deficiency [18]. The effects of iron sparing of chronic oligomenorrhea might contribute to the increased iron store found in serum individual of PCOS [18]. It was a suggestion that development of glucose tolerance abnormalities caused by iron overload, serum ferritin levels are increased in PCOS, especially when resistance increase [19]. The aim of the present work was to investigate the effect of PCOS and hormone disturbance on the serum enzyme activities AST,ALT GGT,, and LDH, STP, albumin, globulin, TC,TG, urea,, creatinine, uric, hemoglobin, packed cell volume (PCV), and iron status parameters (serum iron, TS%, STIBC, transferrin concentration, and ferritin concentration).Also hormone profile, (TT, PRG, LH, PRL, FSH, and estradiol), all these parameters were evaluated in patients women with PCOS, PCOS with AKD, AKD and patients women with IDA. The estimation of activity of LDH in sera of PCOS patients was done for the first time; there is no data on the effect of this enzyme on PCOS.

### Patients and Methods

A total of 160 women were used in the present study, 135 of whom were patients and 25 of healthy control group they were divided according to pathological case into four groups: The first group (G1) consisted of 50 sera of patients women with PCOS, the age of ( $M \pm SD: 28.4 \pm 4.5$ ) years, they were consecutive patients attending the endocrine gynecological unit and other obtained from different hospital in Baghdad city, central healthy laboratory and Ebn-albalady- hospital, from January 2013 to June 2015. The dysfunction and hyperandrogenism of ovarian patients women

were diagnosed for at least two of the following features; biochemical or clinical sign of androgen excess and assessment of PCOS by ultrasound scan, and blood test for hormone profile levels. Menstrual disturbance, dysmenorrheal, infertility is the chief complaints of patients with PCOS. The subjects in the control group didn't have any systemic diseases, and they didn't use any medications that might affect their reproductive physiology or iron status. The exclusion criteria were: the using of any medication that interfere with hormonal measurement or metabolic during analysis preceding three months, or a history of used drug causing elevation of liver enzymes or hormone or lipids.

A second group (G2) involve 30 patients women with age value of ( $M \pm SD: 33.1 \pm 5.3$ ) years suffering from PCOS with AKD. The third group (G3) consisted of 30 patients women with AKD with age value of ( $M \pm SD: 29.7 \pm 5.3$ ) and the last group (G4) include 25 women with iron deficiency anemia with age value of ( $M \pm SD: 28.5 \pm 3.6$ ) years. All specimens were compared with 25 normal apparently healthy individuals. All patients and normal were collected from different hospitals of Baghdad.

**Sample Collection:** eight ml of venous blood were collected from each normal healthy and patients women after fasting overnight, the specimens were collected in the morning after blood clotting the sera were separated by centrifugation at 3500 rpm for 15 minutes and, the stored sera used for different clinical and biochemical assay.

**Biochemical Assays:** The enzyme activities (AST, ALT, GGT and LDH) were measured by kit method. Other biochemical parameters, protein, albumin, total cholesterol, triglyceride, urea, creatinine and a uric acid, also were measured by using kit method, The concentrations of hemoglobin and packed cell volumes were measured directly by using capillaries heparinized. tubes by kit method randox EDTA-containing tubes was taken by venipuncture from each patient.

**Assay of Iron status:** Serum iron, total iron binding capacity, was measured by kit colorimetric method, Randox,-laboratory limited. Percentage transferrin saturation

(TS%) obtained from the formula [20].  
 $TS\% = (\text{serum iron} / \text{TIBC}) \times 100\%$ .

Transferrin concentration (g / L) serum iron (mol / L) / (TS% $\times$ 3.9). Ferritin was measured by enzyme immunoassay Kit method fluorescent detection (ELFA).

**Assay of hormones:** Serum hormone levels of (TT), (PRG), (LH), (PRL), (FSH) and Estradiol estimated by electrochemiluminescence immunoassay (ECLI) using commercially available kits (Roche & DRG, German).

**Statistical Analysis:** Data analyzed and assess by using (version-10) SPSS program to detect  $\alpha$ -level of significance value. Different means was tested significantly using (ANOVA) analysis of variance of more than two groups and independent studentt-test was used

## Results

Biochemical characteristics of participants patients women and control subject are summarized in tables (1 to 5). The mean value of patients ages with PCOS (G1), and patients women of PCOS with AKD (G2), were different statistically  $P < 0.05$ . Body mass index for G1 and G2 were highly increased significantly in comparison with normal group Table (1). Patients of G3 with acute kidney diseases show no remarkable difference for mean value of age and body mass index, patients of G4 iron deficiency, reveals significant difference for mean value of age and no change in body mass index comparing with control group. Table (2) demonstrates the mean value of serum enzyme activities of AST, ALT, GGT, LDH, for patients and control group.

**Patients with PCOS (G1)** show very highly significant increase in the activity of enzymes **AST, ALT, GGT, LDH and protein concentration  $P < 0.001$**  and no change in concentration of albumin as compared with control group. Patients of G2 demonstrate significantly high elevation in AST, ALT, GGT, and LDH activities. Also in proteins albumin and globulin as compared with normal healthy. Patients of G3 illustrated a highly significant elevation in the activity of enzyme GGT, LDH and protein  $P < 0.001$ , and no change in AST, ALT, and globulin as

comparing with normal healthy group. Patients of G4 have no significant change in the activities of AST, ALT and GGT and statistically significant increased of LDH, albumin and globulin  $P < 0.001$  as compared with normal. Table (3) data of serum TC, TG, creatinine show highly significant increased  $P < 0.001$  for patients of G1 to G4. Patients women of G1 show no remarkable change in uric acid urea and there were increased in creatinine concentration compared with control group. The concentration of uric acid, urea, and creatinine was significantly increased in G3 patients of acute kidney disease. G4 patients of IDA were show no significant change in creatinine concentration and there were increases in uric acid, urea as compared with normal. Table (4) represent iron status parameters in all studied groups and for healthy control group. The concentration of iron increase significantly in patients of G1 and decreased in patients groups of G2, G3 and G4. Transferritin saturation percent (%TS) show significant increase  $P < 0.001$  for patients of G1 and a significant decrease in G2, G3 and G4  $P < 0.001$  in comparison with healthy group. Transferin concentration and total iron binding capacity decreased significantly  $P < 0.001$  in G1 patients, there was highly remarkable rising  $P < 0.001$  for G2 G3 and G4 as compare with normal group. Ferritin concentrations were highly increased significantly for G1 to G4 for patients women. Hemoglobin content and packed cell volume were significantly decreased in all groups when compared with normal healthy group  $P < 0.001$ , The hormone profile levels are summarized in table (5), the concentration of TT, LH, estradiol, were significantly elevated  $P < 0.001$  while PRG, PRL, were decreased significantly  $P < 0.001$  and also a decreased in FSH,  $P < 0.01$ , in comparison with normal healthy group.

### Discussion

Polycystic ovary syndrome is not only a gynecological condition affecting reproductive age women, but also a syndrome comprehensive with a variety of metabolic disorders associated commonly with PCOS [21]. The present result was demonstrate that G1 and G2 patients have significant elevation of BMI, and no significant difference in G3 and

G4 patients as compared with normal healthy group Table (1). The obesity, particularly abdominal phenotype, probably responsible for resistance of insulin and hyperinsulinemia in PCOS women, and possibly high dietary lipid intake, may be mechanism additionally that favors the hyperandrogenism development in PCOS [22, 23]. There was highly significant elevation of enzyme activities of AST, ALT, GGT, in G1 patients and in G2 patients as compared with control group Table (2). Our result show significant rising in LDH activity in sera of G1 and in G2 patients  $P < 0.001$  as compared with normal. The LDH enzymes elevation could be due to rate of enzymes releasing depend on their intracellular location and molecular weight, the lymphatic flow and local blood and due to genetic factor (24), result of increasing ALT in the present study was in agreement with previous work [25], elevation of ALT in PCOS is associated with hyperandrogenemia and it is independent of obesity and GGT levels are usually increased in patients with liver disease and a good predictor for metabolic syndrome and cardiovascular risk [26]. The result reveals that level of GGT, ALT, of patients with PCOS was significantly elevated as compared with control groups. The activities of AST, ALT, GGT enzyme, were elevated above the normal upper limit 1.55, 1.2, 2.87 times for PCOS than normal healthy subject this could be due to severity of insulin resistance and obesity this appear to have an additive effect on liver enzymes elevation [27]. It was observed that the increase in the levels of heart enzyme and cardiovascular AST, LDH, and early subclinical atherosclerosis in PCOS patients, this related to metabolic profile including obesity, the elevation might suggest the possibilities of metabolic derangement in the PCOS group [28]. Patients with acute kidney disease G3 have remarkable increase in the activity GGT, LDH and reduction in protein and albumin concentrations as compared with healthy women this in agreement with previous study [24]. Patients with iron deficiency anemia G4 show significant increased in LDH activities and no changes were estimated in AST, ALT, GGT and protein, while there were reduction in albumin and globulin apparently in comparing with

healthy group. Results of Total cholesterol & triglyceride concentrations were significantly increased in G1, G2, G3 and G4 as compared with normal healthy subject, this was in agreement with previous study [29]. In abdominal fat cells, lipoprotein lipase activity decreased by testosterone and insulin resistance were contribute for atherogenic lipid profile, these abnormalities would be expected to increase the morbidity and mortality, cardiovascular disorders, coronary artery disease in women with the PCOS, [30,31]. Insulin resistance and excess androgen favoring deposition of fat in the abdominal fat cell, further facilitates secretion of androgen by their ovaries and adrenals [32,33]. After adjustment for life style factors like smoking, age, and alcohol use, these could eliminate the differences in terms of TG. It was noted that the changed levels of carbohydrates, amino acids and lipids in PCOS patients specifically note the increased level of triglycerides are reduced high density lipoprotein -cholesterol concentrations in these patients, [34]. It was reported that a major regulator of cholesterol production is insulin, hyperandrogenism and insulin may affect lipoproteins and lipids independently of insulin levels and body weight because dyslipidemia probably secondary to insulin resistance [33,35]. A significant increasing in serum iron concentration of G1 patients and a remarkable decrease in patients of G2, G3, and G4, as compared with normal healthy subject Table(4). Increasing of serum iron in patients, indicate the presence of iron with high availability in different tissues in patients of PCOS and caused by deposition and extraction through hepatic cell [16]. Transferrin saturation percent (%TS) was significantly elevated patients of G1, G2, G3, and G4 as compared with control. The fact that high intake of dietary iron and obesity may facilitate the intestinal absorption caused deposition of iron tissue [21,36]. Transferrin concentration and serum total iron binding capacity were significantly decreased in G1 patients of PCOS and increased in groups G2, G3 and G4 in compared with normal healthy groups, this explained due erythropoietin which has been effected by hyperandrogenemia, that is widely known as a

critical component of PCOS. It was found that excess androgen, abnormal glucose tolerance, is correlated with ferritin levels in premenopausal women [37]. There was significant increased in serum ferritin of groups G1 to G4 in comparison with healthy normal group. The result of increased serum ferritin concentration of G1 patients was in agreement with previous study [16]. In PCOS patients the potential iron overload is contributed to factors that involve the effect of iron sparing of chronic dysfunction of menstrual, decrease in hepcidin hormone, insulin resistance, this leading to increased iron absorption[38]. Increasing of iron store though that it is contributed to hyperinsulinism and deposition of iron in  $\beta$  cell through the reduction of hepatic insulin extraction and metabolism, this indicate that tissue does not become circulated and extracted easily [39]. Oligomenorrhea and less blood loss in PCOS subjects might be the best explanation for their increased highly serum ferritin levels, indicating increased stores iron of body, in obese women with PCOS. Deposition of iron in the cardiac tissues and coronary heart disease (CHD), probably a causes of presence of polycystic ovary syndrome due to elevation of iron store level in patients.[16,21,36]. It was found the main causes of accumulation of iron store and ferritin levels in PCOS patients population were based on hypothesis of reduced blood loss due irregularity to period, whereas others assigned hyperinsulinemia, as serum ferritin observed to be higher compared with regularly menstruating women. Different disorders associated with iron over load and have been reported to affect the function of different endocrine gland. [40, 41, 42]. Result of Table(5) reveals highly significant increases in TT, LH, FSH, estradiol, in PCOS patients, and concentration of progesterone, prolactin was significantly decreased in comparing with normal healthy subjects. Testosterone is a hematopoietic hormone and has a dose-dependent stimulatory effect on erythropoiesis. [29,42,43,]. Increase LH levels, cause observed some abnormality in the function of hypothalamic neurotransmitter. Lutinzing hormone secretion and an increased ratio of serum Lutinzing hormone to follicle stimulating hormone (LH/FSH) during the

menstrual cycle follicular phase has been considered as PCOS marker [39]. In our result the ratio of serum LH/FSH was increased to 2.07 in comparing with normal healthy women 1.07, the patients with PCOS often have change functions of hypothalamopituitary, including increased baseline LH concentrations and elevated LH/FSH ratios, as previously reported [39]. It was reporting that fat disturbance and obesity plays a crucial role in the pathophysiology of hyperandrogenism and abnormalities in metabolism of PCOS and, because of capability of synthesis active androgen in human adipose tissue [44]. In PCOS women hormone concentration affect hemoglobin level, excess of Androgen and resistance of insulin, both of which have strong genetic components. The presence of a relationship was observed between hyperandrogenemia, hyperprolactinemia, hyperinsulinemia and serum iron status parameters were detected in patients of PCOS group [41]. Environmental and genetic factors are likely a cause of PCOS, also the genetics or gene mutations might play role in PCOS development. The associated syndrome with numerous morbidities, including cardiovascular disease infertility; diabetes mellitus, and mood and eating disorders. Oxidative stress may be increased in PCOS women; it increases ferritin synthesis, partly to avoid further oxidative damage, given

that ferritin neutralizes the highly toxic unbound iron [29, 40, 45].

**Table (1)**  
**Mean value of age and BMI for patients women and control group.**

Pathological Cases	No. of Cases	Range (Year)	Age (Year) Mean±SD	BMI (kg/m <sup>2</sup> ) Mean±SD
(PCOS) (G1)	50	18-39	28.4±4.5 ***	32±4.2*
PCOS &AKD(G2)	30	27-35	33.1± 5.3***	29±3.2**
AKD (G3)	30	25-31	29.7±5.3 N.S	27±4.7 N.S
(IDA) (G4)	25	24-30	28.5±3.6***	25± 5.2 N.S
Control	25	26-32	30.5±3	26±3.8

\* Very highly significant difference at  $P < 0.001$

\*\* Highly Significant at  $P < 0.01$

\*\*\* Significant at  $P < 0.05$ , N.S=No significant

Table (2)

Mean Value of serum enzyme activities and protein concentration in patients of (G1) PCOS, (G2) PCOS with AKD, (G3) AKD & (G4) IDA & control group.

Parameter	Mean±SD				
	Normal (n=25)	PCOS (n=50) (G1)	PCOS&AKD (n=30) (G2)	AKD (n=30) (G3)	IDA (n=25) (G4)
AST (U/L)	36.57±2.0	56.52±11.2*	57.5±1.2*	34.9 ±2.4 N. S	35.3±2.4 N.S
ALT (U/L)	24.6±1.5	29.8±2.3*	28±2.1*	24.3±0.98 N.S	25±1.1 N.S
GGT (U/L)	24.1 ±2.9	69.21±21.5*	97.4±3.2*	85.7±5.2*	23.7 ±2.3 N. S
LDH( U/L)	135.3±15	224±13.4*	257±14.8*	173.6±14*	180±12.5*
TSP (g/dl)	8.25±1.38	9.22±0.90**	4.5±0.53*	4.1±0.8*	7.9±0.57 N.S
Albumin (g/dl)	4.88±0.7	5.06±0.68 N.S	2.9±0.3**	2.3±0.6**	4.1±0.43
Globuin (g/dl)	3.37±0.54	4.16±0.58 **	1.6±0.2**	1.8±0.3*	3.8±0.31**

\* Very highly significant difference at  $P < 0.001$

\*\* Highly Significant at  $P < 0.01$ , N.S=No significant

Table (3)

Biochemical characteristics of patients groups and control group. G1 PCOS, G2 PCOS with acute kidney disease, G3 acute kidney disease, G4 IDA and control group.

Parameter	Mean±SD				
	Normal (n=25)	PCOS (n=50) (G1)	PCOS with AKD (n=30) (G2)	AKD (n=30)(G3)	IDA (n=25) (G4)
TC (mg/dl)	178.2±20.7	228±18.9*	270.46±21*	220±19.4*	198.3±31.****
TG (mg/dl)	118±3.9	185±5.3*	150.4±8.2*	170±6.8*	125.4±3.4*
Urea (mg/dl)	33.1±3.4	32.6±2.2 N.S	90.0±5*	104±9****	45.4±3****
Creatinine (mg/dl)	1.5 ±0.4 3	2.9±2.5****	58.2± 6.4 *	49.1±2.4 *	1.7±0.6 N.S
uric acid (mg/dl)	4.2 ±1.52	4.8±1.5N.S	8.2 ±1.2*	10.8 ±0.9*	4.6±0.6****

\* Very highly significant difference at  $P < 0.001$

\*\* Highly Significant at  $P < 0.01$

\*\*\* Significant at  $P < 0.05$

\*\*\*\* Significant at  $P < 0.02$ , N.S=No significant

**Table (4)**  
*Iron status parameters in patients groups, (G1) PCOS, (G2) PCOS with AKD, (G3) AKD and (G4) IDA compared with control groups.*

Parameter of iron status	Normal Group (n=25)	PCOS (n=50) (G1)	PCOS &(AKD) (n=30) (G2)	(AKD) (G3) (n=30)	(IDA) (n=20) (G4)
Serum iron (µmol/L)	14.9±1.03	23.2±3.1*	6.87±0.99*	5.9±2.1*	5.1±3.2*
(TS%) Transferrin saturation	4.89 ±1.72	53.3±2.1*	8.6±2.02*	8.4 ±.5*	5.2±2.7*
Transferrin concentration (mg/L)	150.4±3.3	109.36±3.5*	200.7±4.2*	176.47±4.5*	246.49±5.7*
(STIBC) (µmol/L)	59.86±1.8	43.5±1.4*	79.69±9.10*	70.2±8.4*	98.2±12.8*
Serum Ferritin (ng/ml)	101.0±12	391.2±13.4*	508.42±46.35*	113.2±9.4*	211.9±5.9*
Packed cell volume(PCV)	42±2.4	40±2.1*	36±1.7*	35±3*	34±2.4*
Hemoglobin (g/dl)	13.8±0.5	12.8±0.4*	10.3±0.2*	10.9±0.3*	9.3±1.2*

Values of biochemical parameters in term of (Mean±SD)

\* Highly significant difference at P < 0.001, in comparison of patients with normal group

**Table (5)**  
*The hormone level in women of PCOS and in control group.*

Mean ±SD		
Hormone	Normal (n=25)	PCOS patient (n=50)
TT (ng/ml)	0.9 ±0.17	3.6 ±0.23*
PRG (ng/ml)	17.12±1.9	11.8±1.2*
LH (mIU/ml)	9.8±0.6	16.5±1.43*
PRL (ng/ml)	16.13 ±2.4	12.1±2.5*
FSH (mIU/ml)	8.1±1.1	9.9±0.98 **
Estradiol (Pg/ml)	26.0±3.6	36.8 ±4.2*

\* Very highly significant difference at 0.001

\*\* Highly Significant at 0.01

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