



## Evaluation of the Relationship Between Dehydroepiandrosterone Sulfate-Total Testosterone Ratio and Metabolic Parameters in Patients With Polycystic Ovary Syndrome

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### ABSTRACT

**Background** In this study we aimed to evaluate the correlations between dehydroepiandrosterone sulfate-total testosterone (DHEAS/TT) ratio and insulin resistance, glycemic and lipid parameters.

**Material and Methods** A total of 35 patients with polycystic ovary syndrome (PCOS) and 34 healthy volunteers were included in the study. Anthropometric, clinical, biochemical, lipid and glycemic measurements were performed according to routine standards. Patients' demographic, clinical, anthropometric, biochemical, glycemic, lipid and hormonal parameters were measured and recorded. DHEAS/TT ratio was calculated in all patients. DHEAS/TT ratio and metabolic parameters were compared between the PCOS and control groups.

**Results** There were significant differences between the PCOS and control groups in terms of fasting blood glucose, total cholesterol, high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c), triglycerides, insulin and homeostatic model assessment-insulin resistance (HOMA-IR) values (for all,  $p < 0.05$ ). Androstenedione and DHEAS/TT values were statistically significantly higher in the PCOS group. Pearson's correlation analysis revealed no statistically significant correlation between DHEAS/TT ratio and body mass index (BMI), HOMA-IR and lipid profile.

**Conclusions** In PCOS patients, glycemic and lipid parameters, HOMA-IR and DHEAS levels were significantly higher compared to the control subjects. In addition, the DHEAS/TT ratio was also significantly higher at limit in the PCOS group. However, no correlations were observed between DHEAS/TT ratio, BMI, glycemic and lipid parameters, suggesting that DHEAS/TT ratio is not an appropriate method to be used in prediction of metabolic status in patients with PCOS.

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## Introduction

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine conditions, characterized by hyperandrogenic signs and symptoms, polycystic ovaries and ovulatory dysfunction. PCOS affects 10% to 20% of women of reproductive age with an increased risk of developing insulin resistance, dyslipidemia, obesity, diabetes, and cardiovascular disease.<sup>1</sup> Although different definitions of PCOS have been used based on the inclusion criteria until 2012, a consensus was reached at that time on the use of Rotterdam 2003 definition as it captures the broadest population of PCOS subjects (NIH). According to the Rotterdam 2003 diagnostic criteria, PCOS could be diagnosed after ruling out the related disorder by the following three diagnostic criteria: 1) oligo-anovulation, 2) clinical and/or biochemical signs of hyperandrogenism, and 3) polycystic ovaries.<sup>2</sup> The prevalence of PCOS among unselective reproductive-aged women has been reported between 15 and 20% based on the Rotterdam definition.<sup>3-5</sup>

PCOS is correlated with adiposity and insulin resistance that lead to hyperinsulinemia, which triggers increased androgen secretion.<sup>6</sup> The androgen excess is considered the major driving force in the development of signs and symptoms of PCOS. Excessive androgen production by ovaries and adrenals contributes to hyperandrogenism, which is among the diagnostic criteria of PCOS. Increased levels of testosterone is one of the most common manifestations of hyperandrogenism in women.<sup>7</sup> Total testosterone level has been reported to be a strong predictor of the presence and degree of hyperandrogenism.<sup>8</sup>

In women with PCOS, not only increased ovarian androgen, but in up to 50% of these patients increased dehydroepiandrosterone sulfate (DHEAS) levels are also observed. DHEAS is a pre-hormone produced by the adrenal cortex and can be converted into DHEA, which is considered as an active hormone with conversion into testosterone.<sup>9</sup> PCOS patients with higher DHEAS levels and excessive androgen have lower insulin resistance than those with lower DHEAS levels.<sup>10</sup> Although 40-70% of women with PCOS have increased levels of DHEAS, the exact mechanism that triggers production of androgens from the adrenal gland is yet to be clarified.<sup>11</sup>

Correlations between DHEAS, total testosterone, insulin resistance, dyslipidemia, obesity and diabetes in PCOS patients are complex and have not been fully understood. Some studies have claimed that investigation of adrenal versus ovarian androgen ratio may reflect associations between PCOS and metabolic parameters more clearly.<sup>12</sup> Recent studies have focused on the evaluating metabolic risks of women with PCOS using dehydroepiandrosterone sulfate-testosterone ratio.<sup>13,14</sup> Based on this information, in this study we aimed to evaluate the correlations between dehydroepiandrosterone sulfate-total testosterone (DHEAS/TT) ratio and insulin resistance, glycemic and lipid parameters.

## Material and Methods

The study protocol was approved by our Institutional Review Board and was conducted in accordance with the Declaration of Helsinki. A total of 35 female patients diagnosed with PCOS according to the revised Criteria and followed-up in the Department of Endocrinology and Metabolism of our hospital and 34 healthy volunteer women as the control group were included in this prospective study. The control group consisted of age matched women who had no menstrual irregularity and clinical or biochemical signs and symptoms of hyperandrogenism, and who accepted participating in the study voluntarily. Patients with systemic diseases such as hypertension, diabetes mellitus, cardiovascular disease, renal failure, hepatic failure, gastrointestinal malabsorption diseases and malignancy, other androgen excess disorders and pregnant women were excluded from the study. Anthropometric, clinical, biochemical, lipid and glycemic measurements were performed according to routine standards.

Patients' demographic (age), clinical (Ferriman-Gallwey score [FGS], systolic blood pressure, diastolic blood pressure pulse), anthropometric (height, weight, body mass index [BMI], waist circumference, hip circumference, fat percentage), biochemical (urea, creatinine, aspartate transaminase [AST], alanine transaminase [ALT]), glycemic (fasting blood glucose, postprandial blood glucose, insulin, HOMA-IR), lipid (total cholesterol, HDL-c, LDL-c, triglycerides) and

hormonal parameters (total testosterone [TT], free testosterone [FT], dehydroepiandrosterone sulfate [DHEAS], androstenedione, follicle-stimulating hormone [FSH], luteinizing hormone [LH] and estradiol [E2]) were measured and recorded. DHEAS/TT ratio was calculated in all patients. DHEAS/TT ratio and metabolic parameters were compared between the PCOS and control groups.

The diagnosis of PCOS was established according to the 2003 Rotterdam ESHRE/ASRM PCOS Consensus Workshop Group Criteria.<sup>15</sup> Accordingly, patients with two of the following criteria were considered to have PCOS: oligomenorrhea-amenorrhea (menstrual dysfunction of >35 days and more than six cycles), clinical or biochemical signs and symptoms of hyperandrogenism, presence of polycystic ovaries detected on ultrasonography.

Standard anthropometric measurements were made in all participants. Waist circumference was measured in the standing position, at midpoint between the lower costal margin and the iliac crest. Hip circumference was measured in the standing position as the greatest circumference over the buttocks. BMI value was calculated by dividing weight in kilograms by square of height in meters.

The blood samples were collected following an overnight fasting. Hormonal measures included TT, FT, FSH, LH, E<sub>2</sub>, androstenedione and DHEAS. Hormonal analysis was made before the diagnosis of PCOS to rule out other causes of excess androgen. All hormonal parameters were analyzed with enzymatic chemiluminescence (Immulite 2000 Immunoassay System; Siemens Healthcare Diagnostics, Biemann, Germany). The other parameters measured in the fasting blood samples included lipid profile, fasting blood glucose, urea, creatinine, AST, ALT, and HOMA-IR.

#### *Statistical Analysis*

Analysis of the data obtained in this study was performed using SPSS version 23.0 (SPSS, Statistical Package for Social Sciences, IBM Inc., Armonk, NY, USA) software. Normal distribution of the variables was evaluated with the Kolmogorov-Smirnov test. Comparison of the continuous variables between the groups

was made with the Mann-Whitney U test as the variables were skewed, while Chi-square test was used for comparison of the categorical variables. Continuous variables are expressed as mean  $\pm$  standard deviation and categorical variables as number and percentage. Pearson's correlation analysis was used for the evaluation of correlations between the variables.  $p < 0.05$  values were considered statistically significant.

## **Results**

A total of 69 subjects with 35 being PCOS patients and 34 control subjects were included in the study. The mean age was  $26.48 \pm 4.22$  years in the PCOS group and  $27.58 \pm 3.50$  years in the healthy control group. No statistically significant difference was found between both groups ( $p = 0.133$ ). Among the anthropometric parameters, the mean values of weight, BMI, waist circumference, hip circumference, and fat percentage were statistically significantly higher in the PCOS group compared to the control group (for all,  $p < 0.05$ ). The amount of hirsutism as measured by FGS was statistically significantly higher in the PCOS patients ( $11.55 \pm 4.99$  vs  $3.68 \pm 1.46$ ;  $p < 0.001$ ). Anthropometric and clinical features and biochemical parameters of the subjects are given in Table 1.

Looking at the lipid and glycemic results of the subjects; there were significant differences between the PCOS and control groups in terms of fasting blood glucose, total cholesterol, HDL-c, LDL-c, triglycerides, insulin and HOMA-IR values (for all,  $p < 0.05$ ). Lipid and glycemic parameters of the groups are presented in Table 2.

In the hormonal analysis, androstenedione and DHEAS/TT values were statistically significantly higher in the PCOS group compared to the control subjects (both,  $p < 0.05$ ), while the mean estradiol value was significantly lower in the PCOS group. Hormonal results of the groups are given in Table 3. Pearson's correlation analysis revealed no statistically significant correlation between DHEAS/TT ratio and BMI, HOMA-IR and lipid profile. The correlations of DHEAS/TT ratio with BMI, FGS, glycemic and lipid parameters are shown in Table 4.

**Table 1.** Anthropometric, clinical and biochemical measurements of the groups.

	PCOS (mean±SD)	Control (mean±SD)	p value
Age (years)	26.48±4.22	27.58±3.50	0.133
Weight (kg)	74.79±17.75	61.31±12.76	<0.001*
Body mass index (kg/m <sup>2</sup> )	28.27±6.76	22.77±5.04	<0.001*
Waist circumference (cm)	88.21±17.10	74.73±10.58	<0.001*
Hip circumference (cm)	105.13±10.87	99.45±7.94	0.011*
Fat percentage (%)	31.91±9.07	25.25±7.70	0.001*
FGS	11.55±4.99	3.68±1.46	<0.001*
SBP (mmHg)	109.00±7.44	112.50±6.70	0.037*
DBP (mmHg)	68.25±7.12	67.45±11.75	0.785
Urea (mg/dL)	20.95±5.93	22.18±5.38	0.232
Creatinine (mg/dL)	0.73±0.10	0.67±0.06	0.003*
AST (U/L)	17.95±6.38	15.98±3.60	0.159
ALT (U/L)	19.00±13.50	13.28±7.26	0.025*

PCOS: polycystic ovary syndrome, FGS: Ferriman-Gallwey score, SBP: systolic blood pressure, DBP: diastolic blood pressure, AST: aspartate transaminase, ALT: alanine transaminase.

**Table 2.** Lipid and glycemic values of the groups.

	PCOS (mean±SD)	Control (mean±SD)	p value
Fasting blood glucose (mg/dL)	88.61±8.85	81.48±7.88	0.001*
Total cholesterol (mg/dL)	186.71±35.39	166.38±36.69	0.049*
HDL-c (mg/dL)	46.32±8.00	54.30±10.91	0.002*
LDL-c (mg/dL)	116.94±32.46	97.75±24.49	0.014*
Triglycerides (mg/dL)	117.44±64.36	83.83±37.56	0.011*
Insulin (IU/mL)	14.07±7.11	7.89±4.74	<0.001*
HOMA-IR	3.25±1.84	1.60±0.99	<0.001*

PCOS: polycystic ovary syndrome, HDL-c: high density lipoprotein cholesterol, LDL-c: low density lipoprotein cholesterol, HOMA: homeostasis model assessment-insulin resistance.

**Table 3.** Hormonal analysis results of the groups

	PCOS (mean±SD)	Control (mean±SD)	p value
TT (ng/mL)	1.17±0.38	1.16±0.28	0.778
FT (pg/mL)	1.77±0.63	1.93±1.04	0.614
DHEAS (µg/dL)	335.42±131.45	280.24±97.22	0.079
DHEAS/TT	282.35±91.92	245.90±70.58	0.049
Androstenedione (ng/mL)	4.57±1.54	3.31±1.30	<0.001*
FSH (mIU/mL)	4.66±1.15	5.17±2.39	0.921
LH (mIU/mL)	8.02±5.68	7.70±7.27	0.254
E2 (pg/mL)	63.18±70.57	105.18±75.31	0.002*

PCOS: polycystic ovary syndrome, TT: total testosterone, FT: free testosterone, DHEAS: dehydroepiandrosterone sulfate, DHEAS/TT: dehydroepiandrosterone sulfate-total testosterone, FSH: follicle-stimulating hormone, LH: luteinizing hormone, E2: estradiol.

**Table 4.** Correlation between DHEAS/TT and various parameters studied.

	DHEAS/TT	
	r	P
FGS	0.132	0.279
BMI	-0.064	0.6
Weight	-0.051	0.676
Fasting blood glucose	0.151	0.222
Total cholesterol	0.079	0.537
HDL-c	-0.015	0.905
LDL-c	-0.002	0.95
Triglycerides	0.116	0.365
HOMA-IR	-0.037	0.775

DHEAS/TT: dehydroepiandrosterone sulfate-total testosterone, FGS: Ferriman–Gallwey score, BMI: body mass index, HDL-c: high density lipoprotein cholesterol, LDL-c: low density lipoprotein cholesterol, HOMA-IR: homeostasis model assessment-insulin resistance.

## Discussion

In the present study, we investigated DHEAS/TT ratio in patients with PCOS compared to healthy volunteer subjects. PCOS is a multifaceted disorder in relation with metabolic status as determined by obesity, lipid and glycemic parameters. Numerous studies have been conducted on PCOS since it was described first by Stein and Leventhal in 1935.<sup>16</sup> PCOS is one of the most common endocrine disorders in women of reproductive age. As expected, in the present study anthropometric measurements other than height were significantly higher in the PCOS group compared to the controls. Overweight and obesity affects approximately 60% to 80% of PCOS patients.<sup>2</sup> The main pathophysiological components of PCOS are gonadotropic dysfunction and insulin resistance, both of which are related to BMI. In our study, the mean BMI value was significantly higher in PCOS patients compared to the control subjects (28.77 vs 22.77,  $p < 0.001$ ). Bizon et al.<sup>17</sup> found BMI as 24.00 kg/m<sup>2</sup> in PCOS patients, while Neubronner et al.<sup>18</sup> reported this index as 25.14 kg/m<sup>2</sup>. In this context, our findings are close to those of the other studies. In the present study, we investigated the correlation of DHEAS/TT ratio with BMI value, but could not find any association. Guducu et al.<sup>14</sup> reported a significant correlation between DHEAS/FT in PCOS patients. Kosus et al.<sup>13</sup> grouped their PCOS patients in patients with a DHEAS/TT ratio lower than 4.40 and those with a DHEAS/TT ratio higher than 4.40. They found a significant difference between the groups in terms of BMI with higher levels observed in the patients with a DHEAS/TT < 4.40. Since we did not group the patients in this way, we could not compare our results exactly. In that study, only PCOS patients with different DHEAS/TT were included without a control group. We believe that including a healthy group as in our study would give more comparable results. It has been proposed that HOMA-IR is a better marker for evaluating insulin resistance in women with PCOS (19). In our study, the mean HOMA-IR was significantly higher in PCOS patients (3.25 vs 1.60,  $p < 0.001$ ). Lerchbaum et al.<sup>12</sup> found the median HOMA-IR value as 1.2 in PCOS patients with elevated FT. This is higher than our findings, but in our PCOS

group, FT was not elevated. Alebic et al.<sup>20</sup> found the median HOMA-IR values as 2.3, a closer value to our result of 3.25. The authors defined a cut-off value of 3.15 for HOMA-IR in prediction of IR.

Presence and pathogenesis of lipid abnormalities in PCOS remains controversial with some studies showing lipid disturbances in PCOS patients.<sup>21</sup> Ibrahim et al.<sup>22</sup> demonstrated that women with PCOS have atherogenic lipid profile characterized by increased cholesterol, LDL and triglycerides, especially in obese PCOS patients. Manjunatha et al.<sup>23</sup> reported dyslipidemia as the most common abnormality in PCOS with elevated total cholesterol, LDL-c and triglycerides and low levels of HDL-c. Similarly in the present study total cholesterol, LDL-c and triglycerides were increased and HDL-c was decreased in the PCOS patients compared to the control group.

In our study, DHEAS/TT ratio was statistically significantly higher in the PCOS group ( $p = 0.049$ ), indicating increased DHEAS values in PCOS. However, we could not find any correlation between DHEAS/TT ratio and BMI, HOMA-IR and lipid parameters in the patient group. This result indicates the need for further comprehensive studies with a larger series of patients to clarify this issue.

The major limitation of this study is the relatively small number of subjects. In addition to DHEAS/TT, DHEAS/FT could also be analyzed. However, this issue has been studied before and might complicate the study results. The strengths of our study include the detailed metabolic characterization. Further comprehensive multi-center studies are needed to enlighten relationships between PCOS and metabolic status indicators. New parameters with cut-off values calculated could be integrated to the existing criteria to refine the diagnosis of PCOS.

## Conclusions

In PCOS patients, glycemic and lipid parameters, HOMA-IR and DHEAS levels were significantly higher compared to the control subjects. In addition, DHEAS/TT ratio was also significantly higher at the limit in the PCOS group. However, no correlations were observed between DHEAS/TT ratio, BMI, glycemic and lipid

parameters, suggesting that DHEAS/TT ratio is not an appropriate method to be used in prediction of metabolic status in patients with PCOS.

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This study has been presented in 18<sup>th</sup> Uludag Internal Medicine National Winter Congress, 7<sup>th</sup> Bursa Family Medicine Association National Congress, 12<sup>th</sup> Uludag Internal Medicine Nursing Congress, 3-6 March 2022, Bursa, Turkey.

### **Conflict of interest**

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

### **Authors' Contribution**

Study Conception: OOG; Study Design: OOG, SC; Supervision: OOG, SC; Materials: OOG, SC; Data Collection and/or Processing: OOG, SC; Statistical Analysis and/or Data Interpretation: OOG, SC; Literature Review: OOG, SC; Manuscript Preparation: OOG, SC; Critical Review: OOG, SC.

## **References**

1. Goodarzi MO, Carmina E, Azziz R. DHEA, DHEAS and PCOS. *J Steroid Biochem Mol Biol.* 2015 Jan;145:213-25. doi: 10.1016/j.jsbmb.2014.06.003.
2. Azziz R. Controversy in clinical endocrinology: diagnosis of polycystic ovarian syndrome: the Rotterdam criteria are premature. *J Clin Endocrinol Metab.* 2006 Mar;91(3):781-5. doi: 10.1210/jc.2005-2153.
3. Mehrabian F, Khani B, Kelishadi R, Ghanbari E. The prevalence of polycystic ovary syndrome in Iranian women based on different diagnostic criteria. *Endokrynol Pol.* 2011;62(3):238-42.
4. Yildiz BO, Bozdag G, Yapici Z, Esinler I, Yarali H. Prevalence, phenotype and cardiometabolic risk of polycystic ovary syndrome under different diagnostic criteria. *Hum Reprod.* 2012 Oct;27(10):3067-73. doi: 10.1093/humrep/des232.
5. Rashidi H, Ramezani Tehrani F, Bahri Khomami M, Tohidi M, Azizi F. To what extent does the use of the Rotterdam criteria affect the prevalence of polycystic ovary syndrome? A community-based study from the Southwest of Iran. *Eur J Obstet Gynecol Reprod Biol.* 2014 Mar;174:100-5. doi: 10.1016/j.ejogrb.2013.12.018.
6. Christodoulaki C, Trakakis E, Pergialiotis V, Panagopoulos P, Chrelias C, Kassinis D, Sioutis D, Papantoniou N, Xirofotis D. Dehydroepiandrosterone-sulfate, insulin resistance and ovarian volume estimation in patients with polycystic ovarian syndrome. *J Family Reprod Health.* 2017 Mar;11(1):24-9.
7. Rosenfield RL, Ehrmann DA. The Pathogenesis of polycystic ovary syndrome (PCOS): The hypothesis of PCOS as functional ovarian hyperandrogenism revisited. *Endocr Rev.* 2016;37(5):467-520. doi:10.1210/er.2015-1104.
8. Yang Y, Ouyang N, Ye Y, Hu Q, Du T, Di N, Xu W, Azziz R, Yang D, Zhao X. The predictive value of total testosterone alone for clinical hyperandrogenism in polycystic ovary syndrome. *Reprod Biomed Online.* 2020 Oct;41(4):734-42. doi: 10.1016/j.rbmo.2020.07.013.
9. Khan SH, Rizvi SA, Shahid R, Manzoor R. Dehydroepiandrosterone sulfate (DHEAS) levels in polycystic ovarian syndrome (PCOS). *J Coll Physicians Surg Pak.* 2021 Mar;31(3):253-7. doi: 10.29271/jcpsp.2021.03.253.
10. Abbott DH, Zhou R, Bird IM, Dumesic DA, Conley AJ. Fetal programming of adrenal androgen excess: Lessons from a nonhuman primate model of polycystic ovary syndrome. *Endocr Dev* 2008;13:145-58. doi: 10.1159/000134831
11. Ersoy AO, Tokmak A, Ozler S, Oztas E, Ersoy E, Celik HT, Erdamar H, Yilmaz N. Are progrenulin levels associated with polycystic ovary syndrome and its possible metabolic effects in adolescents and young women? *Arch Gynecol Obstet* 2016;294:403-9.
12. Lerchbaum E, Schwetz V, Giuliani A, Pieber TR, Obermayer-Pietsch B. Opposing effects of dehydroepiandrosterone sulfate and free testosterone on metabolic phenotype in women with polycystic ovary syndrome. *Fertil Steril* 2012;98:1318-25.
13. Köşüş N, Köşüş A, Kamalak Z, Hızlı D, Turhan NÖ. Impact of adrenal versus ovarian androgen ratio on signs and symptoms of polycystic ovarian syndrome. *Gynecol Endocrinol.* 2012 Aug;28(8):611-4. doi: 10.3109/09513590.2011.650770.
14. Gündüçü N, Kutay SS, Görmüş U, Kavak ZN, Dündür İ. High DHEAS/free testosterone ratio is related to better metabolic parameters in women with PCOS. *Gynecol Endocrinol.* 2015 Jun;31(6):495-500. doi: 10.3109/09513590.2015.1022862.

15. The Rotterdam ESHRE/ASRM-Sponsored PCOS consensus working group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod* 2004;19:41-7.
16. Azziz R, Adashi EY. Stein and Leventhal: 80 years on. *Am J Obstet Gynecol.* 2016 Feb;214(2):247.e1-247.e11. doi: 10.1016/j.ajog.2015.12.013.
17. Bizoń A, Płaczkowska S, Niepsuj J, Czwojdzńska M, Leśniewski M, Nowak A, Pluta D, Madej P, Piwowar A, Franik G. Body Composition and Its Impact on the Hormonal Disturbances in Women with Polycystic Ovary Syndrome. *Nutrients.* 2021 Nov 24;13(12):4217. doi: 10.3390/nu13124217.
18. Neubronner SA, Indran IR, Chan YH, Thu AWP, Yong EL. Effect of body mass index (BMI) on phenotypic features of polycystic ovary syndrome (PCOS) in Singapore women: a prospective cross-sectional study. *BMC Womens Health.* 2021 Apr 1;21(1):135. doi: 10.1186/s12905-021-01277-6.
19. Majid H, Masood Q, Khan AH. Homeostatic Model Assessment for Insulin Resistance (HOMA-IR): A Better Marker for Evaluating Insulin Resistance Than Fasting Insulin in Women with Polycystic Ovarian Syndrome. *J Coll Physicians Surg Pak.* 2017 Mar;27(3):123-6.
20. Alebić MŠ, Bulum T, Stojanović N, Duvnjak L. Definition of insulin resistance using the homeostasis model assessment (HOMA-IR) in IVF patients diagnosed with polycystic ovary syndrome (PCOS) according to the Rotterdam criteria. *Endocrine.* 2014 Nov;47(2):625-30. doi: 10.1007/s12020-014-0182-5.
21. Swetha N, Vyshnavi R, Modagan P, Rajagopalan B. A correlative study of biochemical parameters in polycystic ovarian syndrome. *Int J Biol Med Res* 2013;4:3148-54.
22. Ibrahim TAE, Ali AE, Radwan ME. Lipid Profile in Women with Polycystic Ovary Syndrome. *EJHM* 2020;78(2):272-7.
23. Manjunatha S, Bennal AS, Hiremath S, Veena HC. Effect of PCOS on lipid profile. *Sch J App Med Sci.* 2014;2(3D):1153-5.

