Original Research Article

Polycystic Ovarian Syndrome: Clinical Correlation with Biochemical Status

Rudra Narayan^{1*}, Ajit Kumar Nayak^{2*}, Puspanjali Khuntia³, Roma Rattan⁴, Abhijit Mohapatra⁵

¹Dept. of Obstetrics & Gynaecology, S.C.B. Medical College & Hospital, Cuttack, Odisha, India

²Associate Professor, Dept. of Obstetrics & Gynaecology, S.C.B. Medical College & Hospital, Cuttack, Odisha,

India

³Associate Professor, Dept. of Obstetrics & Gynaecology, S.C.B. Medical College & Hospital, Cuttack, Odisha,

India

⁴Associate Professor, Dept. of Biochemistry, S.C.B. Medical College & Hospital Cuttack, Odisha, India ⁵Dept. of Obstetrics & Gynaecology, S.C.B. Medical College & Hospital, Cuttack, Odisha, India

Received: 19-08-2021 / Revised: 18-09-2021 / Accepted: 08-11-2021

Abstract

Background: Polycystic ovarian syndrome (PCOS) is the most common endocrine disorder in women of reproductive age group. PCOS consist of chronic anovulation, menstrual disturbances, hyperandrogenism, polycystic ovaries and metabolic syndrome. **Objectives:** to find out clinical, biochemical and hormonal profiles in PCOS cases and correlate with normal individual. **Material and methods**: A prospective case control study of 200 women in the age group of 18-30 years, 100 having PCOS and 100 in the control group. **Results:** Menstrual irregularity seen in 92 % of PCOS, oligomenorrhoea was the most common presentation. 52% had infertility. 53% were hirsute. Mean BMI, waist: hip ratio, mean LH level, LH: FSH ratio, total testosterone, fasting insulin and total cholesterol were high in PCOS compared to control group and the difference in mean values were statistically significant (P value <0.001). Mean LH was 15.44 ± 7.09 in PCOS and 9.22 ± 4.55 in control group. Mean LH: FSH ratio was 2.86 ± 1.44 in PCOS. Mean fasting insulin in PCOS was 21.45 ± 12.49 and 15.58 ± 5.47 in control group. Mean prolactin was 23.85 ± 12.50 in PCOS. Mean fasting insulin in PCOS was 0 for the PCOS were oligomenorrhoic, hirsute with raised BMI and waist-hip ratio. Mean LH, LH: FSH ratio, testosterone and fasting Insulin level were significantly raised in PCOS.

Keywords: Polycystic ovarian syndrome, luteinising hormone, total testosterone, biochemical status, body mass index This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age and most frequent cause of hyperandrogenism and oligoanovulation both of which have substantial psychological, social and economic consequences [1]. In adult women various studies have reported its prevalence of 4-12%.For the first time in 1935 the classic form of PCOS was described by Stein and Leventhal. PCOS consists of chronic anovulation, menstrual disturbance, hyperandrogenism, polycystic ovaries, and metabolic syndrome.

Rotterdam consensus workshop (2003) sponsored by European Society for Human Reproduction and Embryology (ESHRE) and American Society for Reproductive Medicine (ASRM) defines PCOS as presence of any two of the following three criterias: (1) oligo/anovulation (2) Clinical and /or biochemical signs of hyperandrogenism like acne, hirsutism etc; after other causes of hyperandrogenism have been ruled out (3) Ultrasound appearance of polycystic ovary, presence 12 or more follicles with a diameter of 2 to 9 mm in one or both ovaries or ovarian volume > 10 cm³[2].

Dr. Ajit Kumar Nayak

Associate Professor, Dept. of Obstetrics & Gynaecology, S.C.B. Medical College & Hospital, Cuttack, Odisha, India. E-mail: ajitnayak_og@yahoo.co.in Hyperandrogenism is quite common in PCOS which is principally ovarian in origin, clinically commonly manifested by hirsuitism and acne. Women with PCOS have insulin resistance and hyperinsulinemia. Too much insulin combined with high level of LH can lead to excess production of testosterone in the ovaries which in turn lead to disruption in the follicle maturation process, which hampers ovulation [3]. Most of the late complications of PCOS are related to insulin resistance. More than 40% of women with PCOS demonstrate impaired glucose tolerance test and 10% have type 2 diabetes mellitus. In mild Insulin resistances patients will have slightly elevated fasting serum insulin levels whereas in severe forms it may manifest with acanthosis nigricans. Although all women with PCOS have evidence of Insulin resistance it is more pronounced in those with chronic anovulation. PCOS is closely associated with obesity. Obesity worsens hyperandrogenism and menstrual disturbances. Women with PCOS are more likely to carry excess adiposity in central body region. Central or visceral obesity is associated with greater insulin resistance. Treating PCOS in adolescents is important beyond relieving its symptoms because without treatment there is an increased risk of developing infertility, endometrial hyperplasia and carcinoma, type 2 diabetes mellitus, metabolic syndrome, and possibly cardiovascular disease such as myocardial dysfunction, stroke and hypertension. Aim of this study was to find out and correlate various clinical features, hormonal and biochemical changes among PCOS cases and normal control group.

^{*}Correspondence

Materials and Methods

Women in the age group of 18-30 years attending outpatient department of Obstetrics & Gynaecology, S.C.B. Medical College & Hospital, Cuttack, Odisha, India from July 2017 to December 2018 with irregular cycles or oligomenorrhoea / amenorrhoea, Infertility, hirsuitism, and excessive acne were evaluated. PCOS was diagnosed based on Rotterdam criteria. This prospective study included 100 patients of PCOS (50 married and 50 unmarried) and a control group comprising of 100 (50 married and 50 unmarried) women. Institutional Ethics Committee approval was obtained to conduct the study. Women with current or previous use of oral contraceptives, antiandrogens, ovulation glucocorticoids, induction agents. antidiabetic and antiobesity drugs or other hormonal drugs, pregnancy, breastfeeding, Diabetes Mellitus, chronic illness, hyperandrogenism due to other endocrinopathies were excluded from study. Written informed consent was taken from the study participant. Height, weight, waist and hip circumference were measured. BMI and waist: Hip ratios were calculated. Ferriman & Gallway score was used to grade hirsuitism and a score of > 8 was considered significant. Ultrasound of the lower abdomen was done for each patient. For hormonal assessment morning blood sample was taken to analyze serum LH, FSH, total testosterone, fasting insulin, FT3, FT4, TSH and Prolactin level. Samples were taken on day 2 or 3 of menstrual cycle in menstruating women and at random in women with oligomenorrhea. Normal value for FSH, LH and total testosterone were taken as 3 - 4.3 mIU/ml and 4.2 - 6.3 mIU/ml and 8-60 ng/dl respectively. LH: FSH ratio 2:1 was taken significant. Fasting Insulin

normal value < 25 mIU/L was considered as significant. For thyroid profile FT3 (3.1-6.8 pmol/L), FT4 (12-22pmol/L) and TSH (0.27-4.2 μ IU/ml) were taken as normal values. For serum Prolactin, 2-29 ng/ml was taken as normal value. Hormonal assay was done by using enzyme linked immunosorbent assay (ELISA) kits on ELISA reader (Stat fax-2100 technology instruments) in our institution. FBS, 2 hour PPBS, total cholesterol, triglycerides, HDL and LDL were analyzed by using autoanalyser.

Statistical methods

Statistical analysis was performed with use of SPSS version 20. Categorical data was represented in the form of Frequencies and proportions. Continuous data was represented as mean and SD. While evaluating study data, descriptive statistical methods were used. Chi-square was used as test of significance. Student t-test was used for comparing two groups in terms of quantitative data with normal distribution and Mann Whitney U test was used for comparing two groups in terms of data without normal distribution. Significance was evaluated at the levels of p <0.01 and p <0.05.

Results

In our study mean age of participants among PCOS was 22.60 ± 2.937 years compared to 23.37 ± 2.696 years in the control group and this difference was not statistically significant (P value 0.055). 80% of PCOS and 75% of control group were of < 25 years of age and difference was statistically insignificant (P value = 0.397). Married and unmarried participants were equally distributed in both the groups. (Table 1)

Table 1: Comparison of age and marital status a between the PCOS and control group

the recomparison of age and marital status a between the record and control grou				
Variables	PCOS group N (%)	Control Group N (%)	P value	
Age				
< 25 years	80 (80%)	75 (75%)	0.397	
\geq 25 Years	20 (20%)	25 (25%)		
Marital status				
Unmarried	50 (50%)	50 (50%)	1.00	
Married	50 (50%)	50 (50%)		

Menstrual irregularity was seen 92% (92 out of 100) of PCOS compared to 15% (15 out of 100) in the control group and the difference in proportion was statistically significant (P value < 0.001). Most common menstrual irregularity in PCOS was oligomenorrhoea (61%) followed by amenorrhoea in 18% cases. Most of participants in control group had regular menstruation (80%). (Table 2)

Table 2. Comparison of mensu dar cycle pattern between the 1 COS and control group				
Menstrual cycle	PCOS group N (%)	Control Group N (%)	P value	
Regular	7 (7%)	80 (80%)		
Irregular	7 (7%)	0 (0%)		
Menorrhagia	7 (7%)	8 (8%)	< 0.001	
Oligomenorrhoea	61 (61%)	6 (6%)		
Amenorrhoea	18 (18%)	6 (6%)		
estimate of PCOS and 50 memoral error) error contracted for infortility 520/ (26 error of 50				

Table 2: Comparison of menstrual cycle pattern between the PCOS and control group

Married populations (50 patients of PCOS and 50 normal cases) were evaluated for infertility. 52% (26 out of 50) of PCOS had infertility compared to 10% (5 out of 50) in the control and difference of mean was statistically highly significant (P value <0.001).

53% of PCOS had hirsuitism compared to 13% in the control group and the difference of mean was statistically highly significant (P value <0.001). Incidence of acne in the PCOS was 41% compared to 18% in the control group. This difference of mean was statistically significant (P value = 0.001). (Table 3)

Table 3: Comparison of i	ncidence of hirsutism a	and acne between th	ne PCOS and control

Hirsutism	& Acne	PCOS group N (%)	Control Group N (%)	P value
	Present	53 (53%)	13 (13%)	
Hirsutism	Absent	47 (47%)	87 (87%)	< 0.001
	Present	41 (41%)	18 (18%)	
Acne	Absent	59 (59%)	82 (82%)	0.001

Mean body mass index (BMI) was 26.08 ± 2.97 in PCOS subjects compared to 23.44 ± 1.93 in control group. Similarly, waist hip ratio (WHR) was 0.74 ± 0.10 in PCOS compared to 0.63 ± 0.06 in control group. Mean value of these two parameters which measures obesity were statistically significantly higher in PCOS group compared to control group (P value < 0.001). (Table 4)

Table 4: Comparison of BMI and WHR between the PCOS and control group				
Variable	PCOS group Mean ± SD	Control Group Mean ± SD	P value	
BMI	26.08 ± 2.97	23.44 ± 1.93	< 0.001	
WHR	0.74 ± 0.10	0.63 ±0.06	< 0.001	

Table 5: Comparison of hormonal and biochemical profile between both the groups				
Variable	PCOS group Mean ± SD	Control Group Mean ± SD	P value	
LH	15.44 ± 7.09	9.92 ± 4.55	< 0.001	
FSH	5.63 ± 1.42	5.73 ± 1.32	0.626	
LH: FSH ratio	2.86 ± 1.44	1.71 ± 0.71	< 0.001	
Testosterone	70.69 ± 25.67	30.41 ± 14.43	< 0.001	
Fasting Insulin	21.45 ± 12.49	15.58 ± 5.47	< 0.001	
Prolactin	23.85 ± 12.50	19.69 ± 9.00	0.007	
T3	4.07 ± 1.25	4.36 ± 1.28	0.111	
T4	16.72 ± 3.83	16.22 ± 3.61	0.342	
TSH	3.47 ± 2.40	3.42 ± 3.53	0.921	
FBS	95.92 ± 9.57	95.00 ± 9.59	0.498	
PPBS	121.31 ± 7.84	121.84 ± 6.74	0.609	
Total cholesterol	198.0 ± 20.54	178.94 ± 17.93	< 0.001	
HDL	48.47 ± 3.92	48.73 ± 3.81	0.635	
LDL	93.02 ± 9.21	94.13 ± 8.55	0.378	
TG	140.09 ± 16.01	133.56 ± 13.59	0.002	

Mean LH level in PCOS group was 15.44 ± 7.09 mIU/ml compared to 9.92 ± 4.55 mIU/ml in the control group. Mean FSH level was $5.63 \pm$ 1.42 mIU/ml in PCOS group and 5.73 \pm 1.32 mIU/ml in control group. Mean LH: FSH ratio in PCOS group was 2.86 ± 1.44 compared to 1.71 ± 0.71 in Control group. Mean total testosterone level was 70.69 \pm 25.67 ng/dl in PCOS group and 30.41 \pm 14.43 ng/dl in control group. It was raised in 67 % of patients of PCOS but none of the patients in control group had raised testosterone. Mean fasting insulin value in PCOS group was 21.45 ± 12.49 mIU/L compared to 15.58 ± 5.47 mIU/L in the control group. Mean prolactin level was 23.85 ± 12.50 ng/ml in PCOS group compared to 19.69 ± 9.00 ng/ml in control group. Mean T3 level in PCOS group was 4.07 ± 1.25 pmol/L which was lower than the T3 level in control group i.e. $4.36 \pm$ 1.28 pmol/L (P value- 0.111). Mean total cholesterol level in PCOS group was 198.0 \pm 20.54 mg/dl and in control group was 178.94 \pm 17.93 mg/dl. We found significantly higher values of mean serum LH, LH: FSH ratio, total testosterone, fasting insulin, prolactin and total cholesterol in PCOS group in comparison to control group and the difference in mean value of these hormones between the two groups were statistically significant (P value <0.001). We did not find any statistically significant difference in mean value of serum FSH, TSH, T4 level, FBS, PPBS, LDL, HDL and TG between PCOS and control group. (Table 5)

Discussion

We included study participants in the age group of 18-30 years and found mean age for PCOS was 22.60 ± 2.937 years compared to 23.37 ± 2.696 years in the control group. Kumar AN included subjects in the age group of 19 -35 years and observed mean age was 25.6 ± 3.9 in PCOS and 26.7 ± 3.4 in control group[4]. Spandana and Shetty reported mean age of the patient was 27 ± 5.0 years and majority belonged to the age group of 26.30 years (43%) [5].

We found oligomenorrhoea, the most common menstrual irregularity (61% cases) in PCOS compared to 6% in control group. Spandana and Shetty reported 59% of PCOS had oligomenorrhoea [5]. In our study 8% of PCOS had regular period whereas Panda et al reported 14% of PCOS had normal menstruation [6]. In our study 52% of PCOS had infertility compared to 10% in the control group which was statistically highly significant (P value <0.001). Panda et al found 16% PCOS had infertility compared to 4% in control group [6]. Himabindu and Neelima found infertility among 40% PCOS cases [7]. Arain et al found PCOS as the second most common cause of female factor related infertility i.e. 38.5% [8]. In young women hirsutism is the most common clinical presentation of androgen excess. Degrees of hirsutism vary greatly in different ethnic populations and the threshold of abnormality should be measured on a population basis. We found 53% of PCOS had hirsuitism compared to 13% in the control group. Abdulrazzak et al and Pache TD et al found hirsuitism in 64.49% and 63% of PCOS cases respectively[9, 10]. BMI and waist-hip ratio are the two clinical parameters which

measures obesity was significantly higher in PCOS compared to control group. In the current study, mean BMI was higher in PCOS (26.08 ± 2.97) compared to control group (23.44 ± 1.93) and the mean difference was statistically highly significant (P value<0.001). Hahn S et al also reported statistically highly significant mean BMI in the PCOS (31.30 ± 9.80) compared to the control group (22.80 ± 3.0) [11]. Dipankar et al found mean BMI of 28.98 ± 3.11 in PCOS and 21.63 ± 1.18 in normal female individual [12]. Begum et al reported mean BMI of 28.20 ± 4.50 in PCOS compared to 21.05 ± 4.1 in control group [13]. In our study mean waist-hip ratio was higher (0.74 ± 0.10) in PCOS compared to control group (0.63 ± 0.06). Codner E et al reported mean waist-hip ratio of 0.82 ± 0.10 in PCOS compared to 0.77 ± 0.00 in control group [14].

Nasr et al reported 86.7% PCOS cases had elevated LH level [15]. Yousouf et al found raised LH in 63% PCOS patients and 32% controls [16]. In the current study, mean LH was 15.44 ± 7.09 mIU/ml in PCOS compared to 9.92 ± 4.55 mIU/ml in control group and the mean difference is statistically significant (P value <0.001). This raised LH level correlated significantly with oligomenorrhoea. Mohlig et al reported mean LH of 9.01 ± 0.70 in study group and 6.03 \pm 0.61 in control group [17]. Gupta et al observed mean LH in the study group was 10.46 ± 3.02 and 5.89 ± 2.12 in the control group [18].We observed mean LH: FSH ratio was 2.86 ± 1.44 in PCOS and 1.71 ± 0.71 in the Control group which showed statistically significant difference (P value <0.001). Gupta V et al found mean LH: FSH ratio of 1.97 ± 0.83 in PCOS and 1.17 ± 0.37 in the control group [18]. Lakshmi et al reported mean LH: FSH ratio of 1.3 ± 0.6 among PCOS cases [19]. Maheswari et al reported high mean LH: FSH ratio of 1.63 in PCOS compared to 0.71 in control group [20].

A rise in testosterone levels correlates with increase in LH/FSH ratio, antral follicular count and hirsuitism. Excess androgen boosts insulin resistance leading to raised insulin levels, which in turn fuel androgen synthesis. This increases the symptoms of PCOS, making women more susceptible to diabetes, obesity and cardiovascular ailments. We found mean testosterone level of 70.69 ± 25.67 ng/dl in PCOS compared to 30.41 ± 14.43 ng/dl in control group and the difference is statistically significant (P value <0.001). Maheswari et al reported high mean total testosterone in PCOS (71.58) compared to 28.7 in control group [20]. Robinson et al reported mean total testosterone concentration was significantly higher in the PCOS and was the most frequently (70%) abnormal biochemical marker for PCOS [21]. Kar S reported hyperandrogenic phenotype of PCOS was found to be more prone to metabolic complications as compared to phenotypes with normal androgen level [22]. Nahar et al reported mean total testosterone value of 71.40 \pm 27.9 in PCOS and 30% had total testosterone level above the reference range of 30-95 ng/dl [23].In our study mean fasting insulin in PCOS and control were 21.45 ± 12.49 and 15.58 ± 5.47 respectively. The difference of mean value between

two groups was statistically significant (p value<0.001). Nahar et al reported mean fasting insulin of 30.15 ± 12.13 in PCOS subjects [23]. In our study mean serum prolactin was 23.85 ± 12.50 in PCOS compared to 19.69 ± 9.00 in controls and the difference of mean value is statistically significant. Li Yi et al observed mean prolactin was within normal range for both study (15.91 ± 9.56) and control (18.35 ± 7.83) and found no significance [24]. Islam et al reported hyperprolactinemia in 18.6% PCOS cases [25].

Islam et al found hypothyroidism in 11.4% PCOS cases [25]. Sinha et al detected subclinical hypothyroidism in 22.5 % cases compared to 8.75% cases in control group and 2.5 % cases had overt hypothyroidism. PCOS cases had higher mean TSH than control group (4.547 \pm 2.66 and 2.67 \pm 3.11 respectively, p<0.05) [26]. In the present study mean TSH level was 3.47 ± 2.40 in PCOS patients. Timpatanapong and Rojanasakul reported mean TSH of 3.53 ± 3.28 which is similar to our finding [27]. Karakose et al reported mean TSH of 1.90 ± 1.16 in PCOS and 1.76 ± 0.90 in control group [28]. We did not find any significant correlation of TSH level in PCOS and control group (P value 0.921).Mean FBS was 95.92± 9.57 in PCOS and 95.92± 9.57 in control group. Mean PPBS among PCOS was $121.31\pm$ 7.84 and $121.84\pm$ 6.74 in control group. We didn't find any statistically significant difference of mean FBS and PPBS value between PCOS and controls and were in the normal ranges. Similar to our finding Karakose et al reported mean FBS of 85 ± 10 in PCOS and 82 ± 12 in control group and both in the normal range of 60-110 mg% [28]. In our study, mean total cholesterol level in PCOS was 198 \pm 20.54 mg/dl which was significantly higher than that in the control group i.e. 178.94 ± 17.93 (P value < 0.001). Silfen et al and Leustean et al found mean total cholesterol level of 164.0 ± 32.10 mg/dl and 214 ± 40.08 mg/dl in PCOS cases respectively [29, 30]. Mean LDL level in our PCOS cases was 93.02 ± 9.21 mg/dl whereas Christodoulopoulou et al observed mean LDL of 110.7 ± 33.5 mg/dl in their study [31].

Conclusion

Oligomenorrhoea was the most common presenting symptom among PCOS patients. It was correlated with significantly raised LH level and raised LH: FSH ratio in PCOS cases. More than half of PCOS had infertility and hirsuitism. Mean BMI and waist to hip ratio which measures obesity were significantly higher in PCOS in comparison to control group. Total testosterone level which was elevated in 67% PCOS cases seems to be the best hormonal marker. Mean fasting insulin and total cholesterol level in PCOS group were significantly higher than the control group which could be a significant risk factor for future development of type 2 Diabetes Mellitus and Cardiovascular disease.

Acknowledgement: The authors acknowledge the immense cooperation received from study participants. Authors are very much thankful to all the faculties and staffs of Department of Obstetrics & Gynaecology and Biochemistry, S.C.B. Medical College& Hospital Cuttack, Odisha for their whole hearted support and cooperation while conducting this Research study.

References

- Carmina E, Rosato F, Jannì A, Rizzo M, Longo RA. Extensive clinical experience: relative prevalence of different androgen excess disorders in 950 women referred because of clinical hyperandrogenism. J Clin Endocrinol Metab.2006; 91(1):2–6.
- Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Work Shop Group;Revised 2003 Consensus on Diagnostic Criteria and Long Term Health Risk Related to polycystic ovarian syndrome. Fertil Steril.2004; 81(1):19-25.
- Tsilchorozidou T, Overton C, Conway GS. The pathophysiology of polycystic ovary syndrome. Clin Endocrinol. 2004; 60:1-17.
- Kumar AN, Naidu JN, Satyanarayan U, Ramalingam K, Anitha M. Metabolic and Endocrine Characteristic of Indian Women with Polycystic Ovary Syndrome. Int J Fertil Steril.2016; 10(1):22-28

- Spandan JC, Shetty PKK.A study on clinical, biochemical and hormonal profile of Polycystic Ovary Syndrome patients attending tertiary care hospital. Int J Reprod Contracept Obstet Gynecol.2017:6:1986-92
- Panda SR, Durgavati K, Sahu SK. A Study of Clinical Parameters in the Diagnosis of Polycystic Ovarian Syndrome. Journal of Evolution of Medical and Dental Sciences. 2014;3(42):10570-79
- Himabindu Sangabathula, Neelima Varaganti.Clinical profile polycystic ovarian syndrome-100cases.International Journal of Contemporary Medical Research. 2017;4(6):1249-1253
- Arain F, Arif N, Halepota H. Frequency and outcome of treatment in polycystic ovaries related infertility. Pak J Med Sci.2015; 31(3):694–99
- Abdulrazak H Al-Tae'e.Polycystic Ovarian Syndrome: the correlation between the LH/FSH ratio and disease manifestations. Middle East Fertility Society Journal.2007; 12(1):35-40
- Pache TD, de Jong FH, Hop WC, Fauser BC. Association between ovarian changes assessed by transvaginal sonography and clinical and endocrine signs of the polycystic ovary syndrome. Fertil Steril. 1993; 59(3):544-9.
- Hahn S, Tan S, Sack S, Kimmig R, Quadbeck B, Mann K, et al. Prevalence of the Metabolic Syndrome in German Women with Polycystic Ovary Syndrome. Exp Clin Endocrinol Diabetes.2007;115(2):130-5
- B.Dipankar, M.S.Kumar, M.Satinath and P.Mamata.Clinical correlation with biochemical status in polycystic ovarian syndrome. The Journal of Obstetrics & Ganecology of India. 2005; 55(1): 67-71.
- 13. Begum F. Clinical and Hormonal Profile of Polycystic Ovary Syndrome. Journal of SAFOG. 2009;1(2):22-25
- Codner E, Iniguez G, Villarroel C, Lopez P, Soto N, Sir-Petermann T, et al. Hormonal Profile in Women with Polycystic Ovarian Syndrome with or without Type I Diabetes Mellitus. J Clin Endocrinol Metabol.2007;92(12): 4742-46
- 15. A A Nasr, H Hamzah, ZA El Maaty, H Gaber and O Azzam.Transvaginal ultrasound appearance of the ovary in Infertile women with oligomenorrhoea: association with clinical and endocrine Profiles. Middle East Fertility Society Journal.2004;9(2):140-149
- Yousouf R, Khan M, Kounsar Z, Ahangar S, Lone WA. Polycystic Ovarian Syndrome: Clinical Correlation with Biochemical Status.Surg Sci.2012; 3(5):245–248.
- Mohlig M, Spranger J, Osterhoff M, Ristow M, Pfeiffer AFH,Schill T, et al. The polycystic ovarian syndrome per se is not associated with increased chronic inflammation. Eur J Endocrinol.2004;150:525-32
- Gupta V, Sharma S, Raina SK, Bedi GK. Clinical, Ultrasonographic and Biochemical Correlates of Polycystic Ovarian Syndrome: A Case-Control Study from a Tertiary Care Center in North India. J Sci.2018;45(1):8-12
- Lakshmi KS, Jayasutha J, Chandrasekar A. A Study on Prevalence of Polycystic Ovarian Syndrome at a Tertiary Care Hospital. Int J Pharm Sci Res.2015;6(1):383-85
- 20. Maheswari Thanmgavelu, Usha Rani Godla,S. Godi, S.F.D. Paul and R. Maddaly. A Case-controlled Comparative Hospitalbased Study on the Clinical, Biochemical, Hormonal and Gynecological Parameters in Polycystic Ovary Syndrome. Indian J of Pharmaceutical Sciences.2017;79(4):608-616
- Robinson S, Rodin DA, Deacon A, Wheeler MJ, Clayton RN. Which hormone tests for the diagnosis of polycystic ovary syndrome? Br J Obstet Gynaecol.1992; 99(3):232–8.
- Kar S.Anthropometric, clinical and metabolic comparisons of the four Rotterdam PCOS phenotypes: A prospective study of PCOS women, J Hum Reprod Sci.2013;6(3):194-200

- 23. Khairun Nahar, Gazi Mahfuza, Shirin Akhter Begum, Khodeza Khatun, Rafiqul Islam. Clinical, Biochemical and Hormonal Profile of Poly Cystic Ovary Syndrome. Journal of National Institute of Neurosciences Bangladesh.2017;3(2):94-98
- Li Yi, Wei LN, Liang XY. Follicle-stimulating hormone suppressed excessive production of antimullerian hormone caused by abnormally enhanced promoter activity in polycystic ovary syndrome granulose cells. Fertil Steril.2011;95(7):2354-8
- 25. Islam S, Pathan F, Ahmed T.Clinical and Biochemical Characteristics of Polycystic Ovarian Syndrome among women in Bangladesh. Mymensingh Med J.2015;24(2):310-8
- Sinha U, Sinharay K, Saha S, Longkumer TA,Baul SN, Pal SK, et al. Thyroid disorder in polycystic ovarian syndrome subjects: A tertiary hospital based cross-sectional study from Eastern India. Indian J Endocrinol Metab.2013;17(2):304-9
- 27. Timpatanapong P, and Rojanasakul A. Hormonal Profile and Prevalence of Polycystic Ovary Syndrome in Women with Acne. The Journal of Dermatology.1997;24(4):223-9

Conflict of Interest: Nil Source of Support: Nil

- Karakose M, Cakal E, Topaloglu O, Arslan MS, Ginis Z, Sahin M et al. Is there a link between polycystic ovary syndrome and non-thyroidal illness syndrome? Turk Ger Gynecol Assoc.2013;14(4):216-20
- 29. Silfen ME, Denburg MR, Manibo AM, Lobo RA, Jaffe R, Ferin M, et al. Early Endocrine, Metabolic and Sonographic characteristics of Polycystic Ovary Syndrome (PCOS): comparison between nonobese and obese adoloscents. J Clin Endocrinol Metab. 2003;88(10):4682-8
- Leustean L, Preda C, Fica S, Ungureanu M, Cristea C, Ungureanu D, et al. Clinical, hormonal and metabolic profile in overweight and obese women with Polycystic ovary syndrome. Endocrine Abstracts.2012;29:966
- Christodoulopoulou V, Trakakis E, Pergialiotis V, Peppa M, Chrelias C, Kassanos D, et al. Clinical and Biochemical Characteristics in PCOS Women With Menstrual Abnormalities. J Fam Reprod Health.2016;10(4):184-190