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Biochemical predictor for polycystic ovary syndrome

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Polycystic ovary syndrome (PCOS), one of the frequent endocrine metabolic conditions that affect women of reproductive age, is illustrated by hyperandrogenemia, sparse or amenorrhoeic period, and unproductiveness, in addition to metabolic abnormalities such as obesity, insulin resistance, anomalous glucose tolerance, and dyslipidemia, with enduring vulnerability to diabetes, cardiovascular ailments, tumors etc. In this study, 185 female PCOS patients were recruited following 2003 "Rotterdam criteria". Their hematological, biochemical and endocrine characteristics were compared against age-matched 36 healthy female subjects. Majority of the PCOS patients were suffering from oligomenorrhea. BMI and waist-to-hip ratio altered among PCOS patients in comparison to healthy subjects in this study. Significant increase in leukocyte count among PCOS patients in comparison to healthy subjects may discharge inflammatory mediators causing prooxidant microenvironment, and significantly increased neutrophil to lymphocyte ratio among PCOS patients. Significant alteration in fasting blood sugar (FBS) and serum insulin level, development of insulin resistance and alteration in insulin sensitivity amplified PCOS symptoms causing ovarian dysfunction. Dyslipidemia, including increased LDL-cholesterol, cholesterol, triglyceride levels in PCOS patients in our study is a familiar pattern. Except unbound T_4 (thyroxine), no change was observed among studied remaining reproductive hormones such as thyroid stimulating hormone (TSH), leutenizing hormone (LH), follicle stimulating hormone (FSH) and prolactin levels. The area under the curve of uric acid to creatinine ratio was used to discriminate between cases and controls and observed an of AUC=0.938 indicating excellent diagnostic ability at a cut-off > 4.68 with 87.5% sensitivity and specificity of 88.9% in diagnosing PCOS. Correlation analysis also revealed that uric acid to creatinine ratio was significantly associated with platelet count, neutrophil to lymphocyte ratio, fasting blood sugar, LDL-cholesterol and FSH.

Keywords: Dyslipidemia, Fasting blood sugar, Follicle stimulating hormone (FSH), Leukocyte, Leutenizing hormone (LH), Polycystic ovary syndrome (PCOS), Uric acid by creatinine ratio

Polycystic ovary syndrome (PCOS) also acknowledged as Stein and Leventhal syndrome, named after Irving F. Stein, Sr. and Michael L. Leventhal who first detailed in 1935[1], is a familiar endocrine malfunction affecting 5%-10% of child-bearing age women globally, related with several metabolic morbidities[2]. Women with PCOS may have irregular menstruation, acne, and excessive amounts of androgenic hormones[3]. It is recognized by the presence of distended ovaries with several tiny cysts and androgen secreting stroma which is hypervascularized. This syndrome also described in infertility, obesity and excess hair growth[4]. In addition, hirsutism (excessive hair growth), androgenetic alopecia (male or female hair pattern loss), seborrhea (excessive oil production), acanthosis nigricans (dark velvety patches of skin) and skin tags (small, soft, flesh-colored benign growth) are familiar skin appearance[5]. Over a long period, PCOS may enhance the threat of endometrial cancer and metabolic syndromes such as diabetes mellitus, high blood pressure, altered lipid profile in blood, cardiovascular diseases *etc*[3].

Most symptoms first appear in adolescence around the start of menstruation. However, in some women symptoms develop in early-mid 20`s. Anovulation in PCOS patients is a common issue. In PCOS, the hormonal imbalance interferes with the normal ovulation process, often resulting in the absence of ovulation that can lead to infertility. This includes elevated levels of male hormones (androgens) and possibly abnormal content of luteinizing hormone (LH)[6], and a resultant distorted ratio involving LH and the follicle stimulating hormone (FSH)[7]. Low serum prolactin (PRL) may be metabolic threat in infertile PCOS patients[8]. The onset of PCOS has been linked to numerous genetic and environmental aspects[9].

However, polycystic ovaries with non-specific findings were noted in women with no endocrine or

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metabolic abnormalities[10]. Though, presence of polycystic ovaries using ultrasonography was suggestive of PCOS, but not necessarily diagnostic. The European Society of Human Reproduction (ESHRE) and the American Society for Reproductive Medicine (ASRM), both have expanded the phenotypic appearance of PCOS to comprise any two out of the three important features of PCOS: (1) medical or biochemical hyperandrogenism, (2) proof of oligo-anovulation, (3) polycystic featuring-ovarian features on ultrasound, prohibiting other relevant disorders; which is normally known as the "Rotterdam Criteria"[11].

Estimation of blood androgen level in women is difficult. Commonly accessible investigations for the estimation of androgens are mostly unreliable. In addition, the content of testosterone fluctuates during the entirety of a day, and other similar steroids may interfere in assay[12]. Moreover, biochemical hyperandrogenism defined by increased total or free testosterone is measured by premium assays, e.g. liquid chromatography mass spectrometry or extraction/ chromatography immunoassay[13].

The clinical appearance of elevated androgen levels in women including abnormal degree of hair growth is commonly computed using the Modified Ferriman–Gallwey (MFG) counting system by trained personnel. Unfortunately, there remains interobserver variability[13]. Therefore, oligo-amenorrhea (cycles at more than 35 days apart or less than 8 cycles per year) may be considered as indicator for ovulatory impairmnent to establish PCOS[13].

The 2003 Rotterdam criteria recommended PCOM as either 12 or more follicles estimating 2–9 mm in diameter or an ovarian dimension of more than 10 cm^3 for any ovary. Later on, a little lowered follicle number threshold was recommended by the 2018 International Evidence Based Guidelines for the Assessment and Management of PCOS, at ≥20 follicles per ovary and/or an ovarian dimension of ≥10 cm^3 (Ref. 14).

There is no clearly defined biochemical criterion based on metabolic and hormonal factors to identify contributors to Polycystic Ovary Syndrome (PCOS). Therefore, the aim of our study is to identify biochemical predictors from altered metabolic and hormonal profiles in PCOS patients to enable more effective management.

Materials & Methods

Subject selection

The study was designed following the ethical guidelines of the 1975 Declaration of Helsinki and all recruited subjects gave written informed consent to the study. Patients were chosen among those who had visited Obstetrics and Gynaecology Department, College of Medicine & JNM Hospital, WBUHS. Patients satisfied "Rotterdam criteria"[14] were included. Exclusion criteria were: i. pregnant women, ii. usage of drugs to treat Diabetes, Thyroid disorder and Autoimmune diseases (including congenital adrenal hyperplasia, Cushing's syndrome, tumors secreting androgen, and that affect endocrinal parameters). A total of 185 female PCOS patients were recruited. Among them 112 subjects had oligomenorrhea (infrequent periods), 37 subjects had amenorrhea (absence of periods) and 36 subjects from dysmenorrhe (moderate to severe pain caused by menstrual periods). Age-matched 36 healthy female subjects who were found no ovarian cysts by ultrasonography of abdomen or having no clinical history of PCOS were considered as Control. Ethics Committee of the Institution approved the procedures (Ref No. F-24/ PR/COMJNMH/IEC/23/2116; dated 6/3/2024).

Methods

Blood samples were collected from the study subjects for complete blood picture, biochemical and hormonal analysis. Routine diagnostic kits from Transasia Biomedicals Pvt Ltd, were used for the biochemical analysis and chemiluminescence immunoassays (Siemens-healthineers) were performed for hormonal analysis. All other analytical grade chemicals were purchased from E. Merck or SRL, India.

Statistical analysis

Results are expressed as mean ± SE (standard error). Groups were compared by One-way analysis of variance (ANOVA). Statistical software IBM SPPS Version 22 and Graph Pad Prism 5 were used for the data analysis.

Results

Body mass index (BMI) and waist-to-hip ratio of PCOS patients were found significantly higher in comparison to the age matched healthy subjects (Table 1). As shown in Table 2, absolute cell count is significantly changed in PCOS patients in comparison to healthy subjects. Though, no change was observed in Hemoglobin content among studied groups; yet, 12 oligomennorrhea patients, 3 amenorrhea patients and

4 dysmenorrhea patients had anemia. Neutrophil to lymphocyte ratio (NLR) is considered as marker of inflammation and most of the studies indicated >2 showed prognostic values, and we had also observed significant change in NLR among PCOS patients compared to the healthy subjects (Table 2). Moreover, WBC count was found significantly correlated with platelet count, neutrophil to lymphocyte ratio among hematological parameters.

Fasting blood sugar (FBS) level significantly elevated in PCOS patients in comparison to healthy subjects (Table 3); and 28 oligomennorrhea patients, 13 amenorrhea patients and 4 dysmenorrhea patients had hyperglycemia at the time of diagnosis. Moreover, insulin level among PCOS patients was also significantly higher than healthy subjects in our study (Table 3). Therefore, to understand β-cell dysfunction, Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was estimated[15] using the formula: HOMA-IR= fasting insulin (μIU/L) x fasting glucose (nmol/L)/22.5, or HOMA-IR= fasting insulin (μIU/L) x fasting glucose (mg/dL)/ 405.

Table 1 — Anthropometric profile: age, BMI and waist-to-hip ratio of polycystic ovarian syndrome (PCOS) patients and healthy subjects

Parameters	Healthy subjects (n=36)	PCOS patients (n=185)
Age (yrs)	23.08± 0.43	23.81 ± 0.44
BMI (kg/ m^2)	23.22 ± 0.4	25.88 ± 0.26*
Waist to hip ratio	0.83 ± 0.01	0.91 ±0.01*

Values are mean ± SE of number of observations (n)

P values: *< 0.001 compared to normal healthy subjects

Table 2 — Hematological profile of polycystic ovarian syndrome (PCOS) patients and healthy subjects

Parameters	Healthy subjects (n=36)	PCOS patients (n=185)
Hb (g %)	12.04 ± 0.24	12.32 ± 0.11
WBC (× 10^9/L)	3.78 ± 0.17	7.22 ± 0.16*
Neutrophils	2050.94 ± 99.02	4178.45 ± 134.86*
Lymphocytes	1528.5 ± 74.94	2598.07 ± 59.95*
Monocytes	126.66 ± 9.18	272.72 ± 12.37*
Eosinophils	74.44 ± 8.03	182.08 ± 11.14*
Platelet Count (K/μL)	185.7 ± 7.39	227.26 ± 5.96#
NLR	1.36 ± 0.03	1.79 ± 0.08#

Values are mean ± SE of number of observations (n). Values within bracket show normal range. NLR: Neutrophil to lymphocyte ratio

P values: *< 0.001, #< 0.05 compared to normal healthy subjects

Interestingly, PCOS patients showed significantly elevated HOMA-IR level in comparison to healthy subjects in our study (Table 3).

Further, to diagnose insulin resistance in our study, as a reliable and accurate index of insulin sensitivity the Quantitative Insulin Sensitivity Check Index (QUICKI) was calculated using fasting blood glucose and plasma insulin concentrations, as: QUICKI = 1/[log (fasting insulin, U/mL) + log (fasting glucose, mg/dL)][16]. We also observed significant change in QUICKI level among PCOS patients in comparison to healthy subjects (Table 3).

No significant change was observed in renal function tests (urea and creatinine levels) among studied groups (Table 4). However, serum uric acid level as well as uric acid to creatinine ratio in PCOS patients showed significantly higher value than healthy subjects (Table 4).

Table 3— Fasting blood sugar, serum insulin, HOMA-IR and QUICKI index among of polycystic ovarian syndrome (PCOS) patients and healthy subjects

Parameters	Healthy subjects (n=36)	PCOS patients (n=185)
Fasting Blood Glucose (mg/dl) (70-110)	81.44 ± 1.2	108..06 ± 2.33*
Insulin (μIU/L) (<25)	5.37 ± 0.13	12.5 ± 0.67*
HOMA-IR (<2.5)	1.08 ± 0.03	3.91 ± 0.29*
QUICKI (<0.4)	0.38 ± 0.01	0.34 ± 0.01*

Values are mean ± SE of number of observations (n). Values within bracket show normal range

P values: *< 0.001compared to normal healthy subjects

Table 4 — Renal function tests: urea, creatinine, uric acid and uric acid-to-creatinine ratio in serum of polycystic ovarian syndrome (PCOS) patients and normal healthy persons

Parameters	Healthy subjects (n=36)	PCOS patients (n=185)
Urea (mg/dL) (15-42 mg/ dL)	23.8 ± 1.65	23.75 ± 0.82
Creatinine (mg/dl) (0.6-1.2 mg/dL)	0.87 ± 0.03	0.85 ± 0.01
Uric acid (mg/dl) (3.5-6 mg/ dL)	3.51 ± 0.17	5.19 ± 0.07*
Uric acid / Creatinine	4 ± 0.11	6.28 ± 0.12*

Values are mean ± SE of number of observations (n). Values within bracket show normal range

P values: *< 0.001 compared to normal healthy subjects

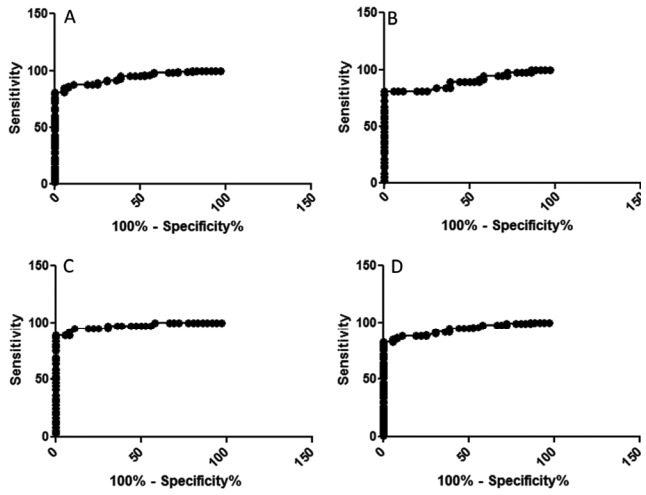

Fig. 1 — AUC curve for Uric acid to creatinine ratio among PCOS patients: (A) ROC of Healthy *vs* oligomenorrhea; (B) ROC of Healthy *vs* amenorrhea; (C) ROC of Healthy *vs* dysmenorrhea; and (D) ROC of Healthy *vs* Total PCOS

Receiver operating characteristic (ROC) curves were plotted with 1-specificity on the X-axis and sensitivity on the Y-axis. Area under the curve of uric acid to creatinine ratio was used to assess the prediction utility. The area under the curve was used to discriminate between cases and controls. The AUC=0.938 showed excellent diagnostic ability at a cut-off > 4.68 with 87.5% sensitivity and specificity of 88.9% in diagnosing PCOS (Fig. 1). Further, we determined the cut-off value as >4.725 that identified 97 (%) oligomennorrhea patients, 30 (%) amenorrhea patients and 33 (%) dysmenorrhea patients.

Lipid profile (particularly, cholesterol, triglyceride and LDL-cholesterol) among PCOS patients significantly altered compared to healthy subjects (Table 5). Among PCOS patients, serum cholesterol level exceeded normal level in 26 patients, serum triglyceride level exceeded normal level in 75 patients; and LDL-cholesterol level exceeded normal level in 65 patients; while 45 patients showed less than normal level of HDL-cholesterol.

Except free thyroxine (FT_4), no significant change was observed in remaining studied hormone profile, viz., thyroid stimulating hormone (TSH), leutenizing hormone (LH), follicle stimulating hormone (FSH) and prolactin levels among studied groups (Table 6). However, 44 PCOS patients showed subclinical hypothyroidism (Normal FT_4, and elevated TSH) and 7 patients showed hyperthyroidism (elevated FT_4, and normal TSH). Interestingly, 54 patients showed LH/ FSH ratio above 2.

Table 5 — Lipid profile (in mg/ dL) of polycystic ovarian syndrome (PCOS) patients and normal healthy persons

Parameters	Healthy subjects (n=36)	PCOS patients (n=185)
Total cholesterol	117.39 ± 4.96	166.36 ± 2.46*
Triglycerides	103.44 ± 3.7	148.33 ± 4.28*
Low Density Lipoprotein cholesterol (LDL-C)	61.61 ± 3.75	92.49 ± 1.8*
High Density ipoproteins cholesterol (HDL-C)	43.64 ± 1.28	45.67 ± 0.69
Very Low Density Lipoproteins cholesterol (VLDL-C)	24.92± 1.87	24.26 ± 0.66

Values are mean ± SE of number of observations (n)

P values: *< 0.001 compared to normal healthy subjects

Table 6 — Hormone status in polycystic ovarian syndrome (PCOS) patients and normal healthy persons

Parameters	Healthy subjects (n=36)	PCOS patients (n=185)
Free Thyroxine (ng/dl) (0.58-1.64)	1.1 ± 0.02	1.20 ± 0.19#
Thyroid Stimulating Hormone (TSH) (μIU/ml) (0.34-5.6)	2.07 ± 0.14	5.21 ± 1.12
Luteinizing Hormone (LH) (mIU/L) (2.12-10.9)	7.06 ± 0.18	12.6 ± 1.54
Follicle Stimulating Hormone (FSH) (mIU/L) (3.85-8.78)	7.68 ± 0.31	7.5 ± 0.89
LH/FSH Ratio (<2)	0.96 ± 0.03	2.0 ± 0.24
Prolactin (ng/L) (3.34-26.72)	9.03 ± 0.86	12.71 ± 1.36

Values are mean ± SE of number of observations (n). Values within bracket show normal range

P values: #< 0.05 compared to normal healthy subjects

Discussion

Etiologic heterogeneity of PCOS is illustrated by oligomenorrhea or amenorrhea and features of hyperandrogenism[17]. In our study, majority of the patients had oligomenorrhea, which is in agreement with other study [18]. PCOS is demonstrated by hyperandrogenemia, sparse or amenorrhoeic period, and unproductiveness, in addition to metabolic abnormalities such as obesity, insulin resistance, anomalous glucose tolerance, and dyslipidemia, with enduring vulnerability to diabetes, cardiovascular ailments, tumors *etc*.[19]

Obesity exaggerates metabolic and possibly hormonal disarrays in patients with PCOS[20], anthropometric properties and body composition analyzers are gradually used to evaluate obesity and

adiposity in clinical and population research. Though, body mass index (BMI) has been extensively used to characterize obesity; however, it cannot precisely predict body adiposity[21]. Waist circumference (WC) and the waist-to-hip ratio (WHR) that imitates an enhancement in visceral fat have been extensively used to calculate abdominal obesity in patients with PCOS[22]. In this scenario, we had observed significant alterations in BMI, and WHR in PCOS patients in comparison to healthy subjects in this study.

The relation between hematological parameters and PCOS is a part of dynamic research. Several studies related to hematological parameters and PCOS have shown different results[4]. It was documented that leukocytosis is a prospective analyst of low-grade inflammation in PCOS[23]. Significant increase in WBC count among PCOS in comparison to healthy subjects in our study is in agreement with other study[24]. Elevated and triggered WBCs discharge inflammatory mediators, *e.g*. neutrophilic myeloperoxidase and NADPH oxidase, causing in the addition of oxygen-free radicals. This prooxidant microenvironment begins the flow towards atherosclerosis, hypertension, and metabolic syndrome[25]. Though, significant increase in neutrophil to lymphocyte ratio among PCOS in comparison to healthy subjects in our study is in agreement as reported elsewhere[26].

Significant alteration in fasting blood sugar (FBS) level among studied groups in our study suggested that the women with PCOS are at enhanced risk for gestational diabetes mellitus (GDM)[27], and the risk of type 2 diabetes mellitus (T2DM)[28]. In effect, inflammation, ROS generation and hyperandrogenemia amplify PCOS symptoms and are also linked to ovarian dysfunction[29]. Appearance of impaired insulin secretion in our study is a major determinant of hyperglycemia and, consequently higher T2DM risk among this population[30]. Deranged insulin level among PCOS patients in our study was further extended to calculate insulin resistance. The detection of IR is superior while using the calculated indices HOMA and QUICKI[31]. HOMA-IR was developed based on feedback dependence between fasting serum glucose and insulin concentration that correlated well with isoglycemic glucose clamp; while QUICKI index was developed for assessment of insulin sensitivity[32]. Interestingly, the mean values of HOMA-IR and QUICKI indexes were significantly higher among PCOS cases when compared with controls in some study[10]. However, significantly higher HOMA-IR index and significantly lower QUICKI index in our study is in agreement with other study for classic phenotypes[33].

PCOS is generally related to the progression of metabolic abnormalities, including dyslipidemia, hypertension, and T2DM[34], which are the major causes of kidney disease[35], and creatinine is reported to be a marker of kidney damage[34]. Our study did not show any change in urea or creatinine level among PCOS compared to healthy subjects. Serum uric acid (UA), the ultimate product of purine catabolism can be used as a predictor of pregnancy conceptions and undesirable foetal outcomes[36]. A growing number of epidemiological studies including this study have demonstrated increased serum UA level in abnormal pregnant patients having PCOS, endometriosis, *etc*.[35-36]. It is hypothesized that androgens may raise serum UA level by persuading hepatic purine nucleotides metabolism[37], which is also a causative factor for kidney disease[38]. Positively correlated uric acid and creatinine levels in our study are also connected with the majority metabolic dysfunctions associated to PCOS such as obesity, dyslipidemia and hypertension in other studies[39,40].

An association between serum creatinine and hyperuricemia in both genders has been previously established[39]. The relevance of the UA/Cr ratio lessens the interference due to gender and renal function[41]. In fact, serum UA/Cr ratio is intricately linked to metabolic disorders than uric acid and is intimately interrelated to metabolic disorders[41]. Therefore, we have estimated UA/Cr ratio in this study. Our subjects showed significant higher UA/Cr ratio in PCOS patients than healthy women. The ROC curve analysis demonstrated the Area under curve of uric acid/ creatinine ratio of 0.938 with 87.5% sensitivity and 88.9% specificity at a cut off value of > 4.68l for the diagnosing PCOS. The UA/Cr may be a valuable predictor in the pathogenesis and prognosis of metabolic syndrome (MS)[42].

Dyslipidemia, including increased LDL-cholesterol, cholesterol, triglyceride levels and decreased HDL-cholesterol are familiar parameters in PCOS patients [43], however, these abnormalities are not uniform in all populations. Androgens play a significant role in hyperlipidemia[44]. Hormone dysbalance and environmental factors in PCOS women may influence lipid metabolism[45]. Studies suggest that hyperandrogenism is an important cause of lipid abnormalities [44]. In our study, triglyceride, total cholesterol and LDL-cholesterol level are significantly elevated in PCOS in comparison to healthy subjects.

Due to the multifactorial nature of the disease, normal distribution in hormonal profile was not found among studied patients. The thyroid gland has multifaceted regulatory functions on metabolism, reproduction, and emotion, as well on hormones production that affect on the majority of the human cells[46]. The clinical features of PCOS are narrowly connected to a few thyroid diseases, such as autoimmune thyroiditis (AIT), and subclinical hypothyroidism (SCH)[46], and their coexistence may categorize patients with advanced reproductive and metabolic hazard[47]. Although data on thyroid function/ dysfunction in women with PCOS are sparse, and confusing, growing evidence suggests a potential link between these diseases[48]. For instance, subclinical hypothyroidism (SCH) is allied with polycystic ovary syndrome (PCOS)[49]. PCOS patients showed higher prevalence of subclinical hypothyroidism than healthy subjects in our study is in agreement with other study[50]. We also found at least 25% of our patients were suffering from SCH. In patients with PCOS, SCH is coupled with comorbidities like dyslipidemia and IR[51]. Though mean fT4 level was within normal limit, yet oligomenorrhea patients showed higher mean level. Likewise, mean level of thyroid stimulant hormone (TSH) level in oligomenorrhea patients showed higher level compared to healthy subjects. Though hypothyroid disorders and enhanced TSH level are commonly reported in PCOS[52]; no significant change among studied groups was observed in our study.

Two hormones namely luteinizing hormone (LH) and follicle-stimulating hormone (FSH) are secreted by the pituitary gland during the menstrual cycle promote folliculogenesis, and are considered monitors of ovulation. However, whether one or both of them participate in essential ovulation-regulatory role is yet to establish[53]. Additionally, LH assists in controlling the length and order of the menstrual cycle in females by participating in ovulation and implantation of an egg in the uterus[54]. Since LH secretion is regulated by the hypothalamic-pituitary-gonadal axis, excess production of LH during the follicular phase suggests harmful effects on the fertility process[55]. PCOS is a regular condition in women coupled with an increased level of LH and a decreased level of FSH as observed in this study is also in agreement with other study[3]. Both these parameters are significantly correlated. While an insignificant increase in LH to FSH (LH/FSH) ratio in PCOS compared to healthy group was observed in our study, a significant change was reported in other studies[56,57]. This discrepancy between LH and FSH results in inappropriate generation of testosterone, disrupting the regulation of the menstrual cycle and reduced fertility[3].

Another pituitary gland secreted hormone, prolactin (PRL) is primarily involved in stimulating the proliferation and differentiation of mammary cells required for lactation[58]. Serum PRL level is directly associated with the metabolism in the pancreas, liver, hypothalamus, and adipose tissue[59]. Though several studies indicated changes in serum PRL level was linked to PCOS development[56], yet no significant change in PRL level in our study, indicating the association between PCOS and the influence of various content of PRL on metabolic homeostasis are inadequate and inconsistent[59].

The limitation of the study is that the study is from single centre and the other limitation is sample size. Secondly, heterogeneity among study subjects limits its uniformity.

Conclusion

Our study found that an elevated serum uric acid to creatinine (UA/Cr) ratio is a promising predictor of polycystic ovary syndrome (PCOS). However, further multicenter studies with larger patient populations are needed to validate these findings.

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Conflict of interest

All authors declare no conflict of interest.

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