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Original Article

Does low pentraxin-3 levels associate with polycystic ovary syndrome and obesity?

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Abstract: Pentraxin-3 (PTX3) a cytokine-inducible molecule is released from various tissues. Its level increases as a response to different inflammatory conditions. Polycystic Ovary Syndrome (PCOS) is considered as a proinflammatory state. The aim of this study was to investigate the association between PTX3 and various metabolic and hormonal parameters in PCOS patients. This study included 64 new diagnosed PCOS patients who had been never treated previously with PCOS and 46 healthy controls with matched age and body mass index (BMI). PTX3, biochemical and hormonal parameters of both groups were measured. The patients were divided into obese and non-obese subgroups according to BMI (above or lower than 25 kg/m^2). PTX3, HOMA-IR and high sensitive C-reactive protein (hs-CRP) levels of these subgroups were compared. Serum PTX3 (p=0.013), hs-CRP (p=0.015) and HOMA-IR (p=0.023) levels of PCOS patients were significantly higher than the control group. Serum PTX3 has been found to have negative correlations with BMI (r=-0.318, $p < 0.001$), waist circumference (r=-0.306, $p < 0.001$), HOMA-IR (r=-0.324, $p < 0.001$) and hs-CRP (r=-0.206, p=0.031). Subgroup analysis revealed PCOS women with obesity to have significantly higher serum PTX3 level than non-obese PCOS subjects (p=0.012), non-obese controls (p=0.015) and obese controls (p=0.002). Women with new diagnosed PCOS especially obese subjects had significantly lower serum PTX3 than the control group. PTX3 has been found to be negatively correlated with BMI and insulin resistance. Low PTX3 level may have a role in the etiology of PCOS and in the formation of atherosclerotic diseases by stimulation of chronic inflammation.

Keywords: Pentraxin-3, polycystic ovary syndrome, insulin resistance, obesity, body mass index

Introduction

Polycystic ovary syndrome (PCOS) is a frequent female disease which has especially insulin resistance (IR) and chronic inflammation in its etiology [1]. In addition, stimulation of pituitary-adrenal axis, luteinizing hormone (LH) and dehydroepiandrosterone sulfate (DHEAS) have major role in PCOS formation [2]. IR is of great importance in the etiology of PCOS since LH and free testosterone (FT) levels increase with increasing insulin level [3]. C-reactive protein (CRP) is a good marker for systemic inflammation that is found to be high in PCOS patients, at the same time it reflects IR level [4]. However, it is insufficient in most cases to demonstrate the prognosis of a disease so the search for new markers increased especially for PCOS.

Pentraxin-3 (PTX3) a new inflammatory marker is a member of the long pentraxin family which is a component of the humoral immunity of innate immune system [5]. It is released in response to inflammatory stimuli by a many cells such as endothelial cells, monocytes, macrophages and fibroblasts [6]. In the contrary to CRP which is synthesized in the liver in response to local inflammation [7]. PTX3 is released from peripheral tissues in response to proinflammatory signals. Therefore unlike the synthesis of CRP, PTX3 is not affected by drug-induced hepatic protein synthesis [8, 9] which

makes it a useful and probably a better marker of inflammation. PTX3 level has been found to be high in many inflammatory diseases such as sepsis, rheumatoid arthritis, systemic lupus erythematosus and late phase myocardial infarction [9]. PTX3 level was investigated in PCOS patients and was found to be higher, lower or similar to the control group. Moreover there are studies which have shown body mass index (BMI) and IR to be positively or negatively correlated with PTX3 [10-12]. As both obesity and IR have important roles in PCOS etiology. PTX3 also may have an important role in PCOS etiology. In addition, PTX3 deficiency is associated with severe defect fertility [13].

In this study, we aimed to investigate whether PTX3 level of PCOS patients is higher, lower or similar to the control group. Our second aim was to investigate whether there is a difference of PTX3 level between obese and non-obese PCOS subjects and whether PTX3 level have correlations with the etiologic factors of PCOS such as IR, FT, LH and DHEAS.

Methods

Study population

This study included 64 patients with new diagnosed PCOS and 46 healthy controls with matched age and BMI. All the patients gave a written consent. The demographic information of all subjects were gathered and evaluated. They included the complaint at presentation, age (years), age of menarche, last menstrual date, number of gravidity, numbers of parity, number of abortus, number of living children, menstrual cycle regularity (number of days between cycles/number of days of menstrual bleeding/total amount of bleeding in a cycle in pads) and the age of the first and last pregnancy. The days of menstrual cycle were determined according to the anamnesis. Transvaginal and/or transabdominal ultrasonography of all the subjects cases were performed to determine uterus size (mm), myometrial structure, endometrial thickness (mm), size of ovaries (mm), number of follicles (number) and their diameters. The diagnosis of PCOS was made according to the Rotterdam European Society for Human reproduction and Embryology/ American Society for Reproductive Medicine-sponsored PCOS Consensus Workshop Group [14].

The revised criteria for PCOS diagnosis were as follows, with at least two of the following being required: 1) Oligo-ovulation and/or anovulation which were defined by the presence of oligomenorrhea (mean interval between bleeds ≥ 35 and < 182 days) or amenorrhea (mean interval between bleeds ≥ 182 days) confirmed by luteal progesterone and normal serum follicle stimulating hormone (FSH) levels (normal range: 1.0-10.0 IU/l). 2) Clinical hyperandrogenism was defined as the presence of at least one of the following three features: hirsutism, acne and androgenic alopecia. Biochemical hyperandrogenism was defined as a serum testosterone level > 60 ng/dL (> 2.08 nmol/L). 3) At least one ovary examined by ultrasound contained 12 or more follicles measuring 2-9 mm in diameter and/or increased ovarian volume (> 10 mL).

After performing general physical and gynecological examination Ferriman-Gallwey (FG) scores (points), height (cm), weight (kg) and waist circumference (WC) (cm) were measured. Body mass index (BMI) was calculated as body weight (kg)/square height (m^2). Overweight or obesity in adults was defined by World Health Organization (WHO) as BMI > 25 kg m^2 for overweight and BMI > 30 kg/m^2 for obese. According to the previous studies which investigated PTX3 level of two groups divided into BMI lower or above 25 kg/m^2 we considered the subjects with BMI above 25 kg/m^2 to be named obese. FG scoring was used to evaluate hair growth in 11 areas of the body: upper lip, chin, chest, upper back, lower back, upper abdomen, lower abdomen, upper arms, forearms, thighs and legs, a score was established for each area. Absence of terminal hair growth was scored from 0 to a maximal growth as 4+. A total score of 8 or higher was defined as hirsutism.

Exclusion criteria: pregnancy, any endocrine disorder such as Cushing's syndrome, 21-Hydroxylase deficiency, congenital adrenal hyperplasia, thyroid dysfunction, hyperprolactinemia, diabetes and a history of gestational diabetes Subjects with chronic diseases such as cardiovascular, hepatic, hematologic, chronic renal failure, hypertension and cancer were excluded from the study. Users of oral contraceptives, anti-androgenic, glucocorticoids, antihypertensive, antidiabetics and anti-obesity drugs, smoking and alcohol consumption were excluded from the study. All the subjects were non-smokers and had normal physical activity.

Control group consisted of healthy subjects who had hirsutism score < 8, regular menses every 21-35 days and normal androgen levels. None of the women in the control group had polycystic ovary in ultrasound.

Clinical, biochemical and hormonal measurements

Venous blood samples were obtained from all subjects following an 8-12 h overnight fast between 09:00 and 10:00 in the morning between 3 and 5 days of spontaneous or progesterone induced menstrual cycle. The levels of fasting plasma glucose (FPG), fasting plasma insulin (FSI), total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL) and high-sensitive C-reactive protein (hs-CRP) were measured. All the patients underwent a 75 gr oral glucose tolerance test (OGTT). When the test results were determined patients with diabetes who was excluded in the study. An IR score Homeostasis Model Assessment-IR (HOMA-IR) was calculated by the following formula: HOMA-IR=FPG (mmol/L)×FSI (mU/mL) /22. 5. The hexokinase method was used to measure FPG levels and the photometric method (Abbott Architect c16000 autoanalyser) was used to measure the TC, TG, HDL, and LDL levels. The concentration of hs-CRP was measured using immune-tubidimetric method with Abbott Architect C16000 otoanalyzer (Abbott Diagnostic, USA). Serum levels of insulin, FSH, LH, DHEAS, total testosterone (TT) and thyroid stimulating hormone (TSH) were measured using chemiluminescent microparticle enzyme immunoassay (CMIA) method with Abbott Architect i2000 (Abbott Diagnostic, USA). Serum 17-hydroxyprogesterone (17OHP) and FT were measured by radioimmunoassay.

Measurement of PTX3

Tubes containing EDTA with 2 ml were used collecting blood. The blood then was immediately transferred to centrifuge tubes which contain aprotinin (0. 6 TIU/ml of blood) inside. The tubes were gently rocked several times to inhibit the activity of proteinases before being centrifuged. The centrifuge process was carried out at 1600 g rate for 15 min at 4°C condition to obtain the plasma that was stored in -80°C freezer until the time of assay. The concentration of PTX3 was measured using enzyme-linked immunosorbent assay (ELISA) method. We used commercially available human pentraxin-3 ELISA kit (Hycult biotech, Netherlands). The procedure for the ELISA method was carried out according to the instructions provided by the manufacturer. Absorbance was measured at a wavelength of 450 nm using ELISA reader. The correlation coefficient of this assay system between the theoretical values and the actual values was 0.99 and the minimum detection level was about 0.01 ng/ml. The intra-assay standard deviation was always under 10%. The ELISA assay did not cross-react with the short pentraxins and CRP. The levels of PTX3 were presented as ng/ml.

Statistical analysis

Data were analyzed using SPSS Software (Version 18, SPSS, Inc., Chicago, IL, USA). Results were expressed as mean±standard deviation. The Mann Whitney U (non-normal distribution parameters) and Independent Student T test (normal distribution parameters) were used to compare the continuous variables. Pearson correlation test was used for calculation of associations between variables. Subgroup analysis was performed by One-Way ANOVA test and bonferrroni from post hoc tests according to BMI levels. A p value of less than 0.05 was considered to be statistically significant.

Results

The mean age of PCOS group was 22.9±4.3 years, BMI was 30.3±8.7 kg/m^2, WC was 95.9±18.7 cm and FG score was 13.7±3.6. The mean age of the control group was 21.9±4.5 years, BMI was 29.7±5.2 kg/m^2, WC was 92.3±12.2 cm and FG score was 4.2±1.0 (p < 0.001). PTX3 level of PCOS group was obviously lower than the control group (5.5±2.8 ng/ml vs. 6.8±2.7 ng/ml, p < 0.001). However, the levels of hs-CRP (0.5±0.8 mg/dl vs. 0.2±0.3 mg/dl, p=0.015), FT (3.1±1.8 pg/ml vs. 2.4±0.8 pg/ml, p=0.013), FSH (4.5±1.2 mIU/mL vs. 4.1±0.6 mIU/mL, p=0.026), LH (5.8±3.2 mIU/mL vs. 4.5±1.7 mIU/mL, p=0.010), 17-OHP (1.6±1.0 ng/ml vs. 1.3±0.6 ng/ml, p=0.038), FPG (95.5±9.8 mg/dl vs. 88.6±10.5 mg/dl, p < 0.001) and HOMA-IR (2.7±1.5 vs. 2.0±1.3, p=0.023) in PCOS group were significantly higher than the control group. All biochemical and hormonal results are shown in **Tables 1** and **2**.

Table 1. Demographical characteristics and biochemical values of controls and PCOS patients

Parameters	*PCOS*	*Controls*	*p value*
	(n=64)	(n=46)	
Anthropometric measures			
Age (years)	22.9±4.3	21.9±4.5	0.208
BMI (kg/m^2)	30.3±8.7	29.7±5.2	0.604
Waist circumference (cm)	95.9±18.7	92.3±12.2	0.221
FG score	13.7±3.6	4.2±1.0	0.001
Glucose metabolism			
FBG (mg/dL)	95.5±9.8	88.6±10.5	0.001
FSI (μ IU/ml)	11.0±6.6	9.4±6.0	0.182
HOMA-IR	2.7±1.5	2.0±1.3	0.023
Lipid profile			
HDL (mg/dL)	49.9±12.2	47.3±10.4	0.221
LDL (mg/dL)	114.4±32.7	104.9±22.7	0.077
TG (mg/dL)	106.2±53.1	90.6±36.6	0.072
TC (mg/dL)	184.5±37.2	169.8±26.8	0.019

BMI: body mass index, FG: Ferriman-Gallwey, FPG: fasting plasma glucose, FSI: fasting serum insulin, HOMA-IR: homeostasis model assessment insulin resistance index, HDL: high density lipoprotein, LDL: low density lipoprotein, TG: triglyceride, TC: total cholesterol.

Table 2. Hormonal values and inflammation markers of controls and PCOS patients

Parameters	*PCOS*	*Controls*	*p Value*
	(n=64)	(n=46)	
Inflammation markers			
Pentraxin-3 (ng/ml)	5.5±2.8	6.8±2.7	0.013
hs-CRP (mg/dl)	0.5±0.8	0.2±0.3	0.015
Hormonal parameters			
Total testosterone (ng/ml)	1.1±0.4	1.0±0.2	0.130
Free testosterone (pg/mL)	3.1±1.8	2.4±0.8	0.013
DHEAS (μg/dL)	272.0±117.0	265.2±81.1	0.721
E2 (pg/mL)	40.8±17.9	39.6±10.7	0.679
FSH (mIU/mL)	4.5±1.2	4.1±0.6	0.026
LH (mIU/mL)	5.8±3.2	4.5±1.7	0.010
17-OHP (ng/ml)	1.6±1.0	1.3±0.6	0.038
TSH (μIU/ml)	2.2±1.4	2.1±1.4	0.717

Mean±standard deviation. hs-CRP: high-sensitive C- reactive protein, DHEAS: dehydroepiandrosterone sulfat, E2: estradiol, FSH: follicle-stimulating hormone, LH: luteinizing hormone, 17-OHP: 17-hydroxyprogesterone, TSH: thyroid stimulating hormone.

According to Pearson correlation results serum PTX3 level was negatively associated with FG score (r=-0.197, p=0.039), BMI (r=-0.318, p < 0.001), WC (r=-0.306, p < 0.001), HOMA-IR (r=-0.324, p < 0.001), FSI (r=-0.301, p < 0.001) and hs-CRP (r=-0.206, p=0.031). However, HOMA-IR has positive correlation with BMI (r=0.518, p < 0.001), WC (r=0.487, p < 0.001), FPG (r=0.430, p < 0.001), FSI (r=0.953, p < 0.001) and hs-CRP (r=0.520, p < 0.001). All correlation analysis results are shown in **Table 3**.

Subgroup analysis according to BMI revealed that PTX3 level of obese PCOS females (4.8±2.8 ng/ml) was significantly higher than non-obese PCOS (6.7±2.6 ng/ml, p=0.012), non-obese control (6.9±2.4 ng/ml, p=0.015) and obese-control (6.8±2.8 ng/ml, p=0.002). HOMA-IR of obese PCOS patients (3.1±1.6) was significantly higher than non-obese PCOS (1.7±0.6, p < 0.001), non-obese control (1.2±0.9, p < 0.001) and obese-control (2.4±1.3, p=0.018). In addition, hs-CRP of obese PCOS patients (0.6±0.9 mg/dl) was significantly higher than non-obese PCOS (0.1±0.1 mg/dl, p=0.002), non-obese control (0.1±0.1 mg/dl, p=0.006) and obese-control (0.2±0.3 mg/dl, p=0.005). All subgroup analysis results are shown in **Table 4**.

Discussion

In our study, while PTX3 level of young women with newly diagnosed PCOS was significantly lower than the healthy control group, HOMA-IR, hs-CRP, FPG, FT and LH levels were significantly higher than the control group. While PTX3 level was negatively correlated with HOMA-IR, BMI, WC and hs-CRP it had no correlation with LH, FT and TT. When subgroup analysis was performed according to BMI, PTX3 level of obese PCOS subjects has been found to be significantly lower than the other three groups. PTX3 level of non-obese PCOS subjects has been found to be lower than both obese and non-obese healthy controls. However, it was statistically insignificant. PTX3 level of PCOS patients was previously investigated by few studies and interestingly PTX3 level was found to be higher, lower or similar to the healthy controls. Similarly, PTX3 had both positive and negative correlations with BMI, WC, hs-CRP and IR [10-12]. As IR takes a role in the etiopathology of PCOS patients, this is an

Table 3. Pearson correlation coefficients (r) between pentraxin-3 levels and measured parameters in PCOS subjects

	Pentraxin-3		HOMA-IR	
Variable	*r Value*	*p Value*	*r Value*	*p Value*
Age	0.093	0.333	0.149	0.121
BMI	-0.318	0.001	0.518	0.001
Waist circumference	-0.306	0.001	0.487	0.001
FG score	-0.197	0.039	0.148	0.122
FPG	-0.024	0.800	0.430	0.001
FSI	-0.301	0.001	0.953	0.001
HOMA-IR	-0.324	0.001		
HDL	0.107	0.268	-0.337	0.001
LDL	-0.003	0.977	0.163	0.091
TG	-0.143	0.135	0.149	0.119
TC	-0.005	0.957	0.079	0.411
hs-CRP	-0.206	0.031	0.520	0.001
Total testosterone	-0.031	0.750	0.162	0.091
Free testosterone	-0.172	0.072	0.034	0.723
17-OHP	0.010	0.917	0.106	0.270
DHEAS	-0.054	0.575	0.070	0.468
E2	0.128	0.183	0.089	0.355
FSH	0.142	0.138	-0.106	0.270
LH	-0.108	0.261	0.100	0.300
TSH	0.000	0.999	0.112	0.242

BMI: body mass index, FPG: fasting plasma glucose, FSI: fasting serum insulin, HOMA-IR: homeostasis model assessment insulin resistance index, HDL: high density lipoprotein, LDL: low density lipoprotein, TG: triglyceride, TC: total cholesterol, hs-CRP: high-sensitive C-reactive protein, DHEAS: dehydroepiandrosterone sulfate, E2: estradiol, FSH: follicle-stimulating hormone, LH: luteinizing hormone, 17-OHP: 17-hydroxyprogesterone, TSH: thyroid stimulating hormone.

extremely complicated situation. However, PTX3 levels in PCOS still is unclear why that increase or decrease.

Similar to our data, many studies have reported PTX3 level to be diminished in obese subjects and PCOS patients [11, 15-17]. It has been reported that adiponectin level is decreased secondary to elevated oxidative stress in adipose tissue. Probably, raised reactive oxygen species are suppressed to PTX3 leading to lowering PTX3 level [18]. As PTX3 inhibits classical complement pathway, low level of PTX3 presumably leads to chronic inflammation of obese subjects [19]. Also, PTX3 level has been found to be low in early hours of myocardial infarction [20]. As PTX3 inhibits p-selectin released from neutrophils, low level of PTX3 increases thrombocytes aggregation thus atherosclerotic disease may occur [20]. In our study, also, PTX3 was negatively correlated with BMI, WC and IR. Moreover when subgroup analysis according to BMI was performed PTX3 level of obese patients with PCOS was obviously low. Even though, it was not statistically significant PTX3 level of non-obese PCOS female was lower than obese females. As we know, PCOS is highly related to oxidative stress. According to our results, PTX3 level can be suppressed by increased oxidative stress in PCOS. These findings have shown the course of PTX3 to be low especially in early years of PCOS. It has not been clearly understood so far whether PTX3 level is lower in PCOS especially those with obesity so new studies are needed in this subject.

On the other hand, few studies have shown PTX3 level to be elevated in PCOS patients and obese subjects [12, 21]. These studies have reported PTX3 level to be positively correlated with BMI, WC and IR. However, we know that organism is in equilibrium. Probably low or high PTX3 level may lead to a damage in the organism. Unlike few studies, many studies reported that PTX3 level is inversely associated with obesity. PTX3 is a protein that prevents metabolic syndrome. Its low level has been reported to lead to obesity [22]. On the contrary PTX3 mRNA is found in plenty in the visceral adipose tissue (VAT) and the increase in VAT has been reported to increase PTX3 gene expression in spite of low PTX3 level [23]. At the same time as proinflammatory cytokines are released excessively from VAT the decreased release of PTX3 may be accompanied with these cytokines so the atherosclerotic process is accelerated [24]. In our study, PTX3 level of obese subjects has been found to be low. WC ratios of the patients were really higher than the control group. In fact, even though WC reflects VAT it is not always trusted. In Rallidis et al. investigated carotid atherosclerosis which is a good marker for atherosclerosis has reported that direct measurement of VAT to be more valuable than WC [25]. Interestingly, our study and the other studies conducted on PCOS and VAT has not been investigated. Thus, further studies that would investigate VAT and PTX3 levels in PCOS patients may open a new era for the etiopathogenesis of PCOS.

In this study, PTX3 has been found to have negative relation with hs-CRP and IR. Subgroup

Table 4. Subgroup analysis of PCOS and the control group by ANOVA

	PCOS		Control	
Variable	*BMI ≤ 25 kg/m²*	*BMI > 25 kg/m²*	*BMI ≤ 25 kg/m²*	*BMI > 25 kg/m²*
BMI (kg/m²)	21.2±2.0*	35.2±7.0	24.0±2.7*¶	32.1±3.8≠α
WC (cm)	77.7±8.2*	105.8±15.2	79.7±7.8*¶	97.7±9.3¥α
Pentraxin-3	6.7±2.6M	4.8±2.8	6.9±2.4E	6.8±2.8β
HOMA-IR	1.7±0.6*	3.1±1.6	1.2±0.9*U	2.4±1.3R
hs-CRP (mg/dl)	0.1±0.1β	0.6±0.9	0.1±0.1□	0.2±0.3µ
TT (ng/mL)	1.2±0.4	1.0±0.4	0.9±0.3Ø	1.0±0.2
FT (pg/mL)	2.9±1.3	3.2±2.0	2.3±1.0	2.4±1.8W
LH (mIU/mL)	7.0±3.6E	5.2±3.0	4.6±2.0€	4.5±1.5α
DHEAS4	244.5±126.4	285.9±111.9	270.4±77.6	264.3±82.7

BMI: body mass index, WC: waist circumference, hs-CRP: high-sensitive C-reactive protein, TT: total testosterone, FT: free testosterone, LH: luteinizing hormone, DHEAS: dehydroepiandrosterone sulfat. *$p < 0.001$, β$p=0.002$, ¥$p=0.003$, µ$p=0.005$, □$p=0.006$, ≠$p=0.008$, M$p=0.012$, E$p=0.015$, R$p=0.018$, W$p=0.039$ vs. obese PCOS, α$p < 0.001$, €$p=0.010$, Ø$p=0.033$ vs non-obese PCOS, ¶$p < 0.001$, U$p=0.006$ vs obese control.

analysis revealed that beside hs-CRP of obese PCOS females being higher than the other three groups it was at the upper limit of the normal values. IR is known to be in the basis of PCOS. Elevated levels of both IR and hs-CRP demonstrate the acceleration of atherosclerotic process in these patients. Previous studies shown that PTX3 level is inversely associated with serum CRP level and IR [18, 26]. At the same time low PTX3 level as explained in the mechanisms above (chronic inflammation by classic complement system) contributes to the acceleration of this process. In addition, low PTX3 is related to coronary artery disease [17]. Therefore, PTX3 is known to be negative an inflammatory marker. In our study, hs-CRP level was approximately normal even in new diagnosed obese PCOS patients and was higher in the other three groups. The slow course of the inflammatory process might lead to high hs-CRP level thus PTX3 level might be found to be low.

Interestingly, PTX3 had no association with TT, FT, LH and DHEAS. Elevated FT, LH and DHEAS levels contribute to the etiology of PCOS. LH level of non-obese PCOS subjects were higher than other groups which indicates its important role in the etiology of PCOS in non-obese females. PTX3 may be effective only in the inflammatory process and via IR on the etiology of PCOS. Thus may explain the combination of the atherosclerotic disease during PCOS course. In addition, PTX3 level is known to be elevated with age [17, 21]. PTX3 levels of middle or advanced aged patients were investigated in many studies conducted on atherosclerotic process. Studies conducted in PCOS investigated especially the second decade patients. As age increases, the atherosclerotic process and its complications appear. The reason for finding low PTX3 level in our study may be the young and newly diagnosed patients. As the disease progresses PTX3 release may dramatically reduce by secondary to continuous IR, oxidative stress and proinflammatory cytokines. So further studies with long term follow up including middle and advanced aged PCOS patients are needed to be more informed in detail about PTX3.

Conclusions

PTX3 level was found to be low in newly diagnosed PCOS patients. Especially obese PCOS patients had obviously lower PTX3 level. Unlike few studies, many studies reported that PTX3 level is inversely associated with obesity as our study. PTX3 is a protein that prevents metabolic syndrome. Its low level has been reported to lead to obesity and coronary artery disease. Probably, low PTX3 level may cause PCOS. As PTX3 is a new molecule further studies with large populations and long term follow up are needed to be conducted on PCOS patients.

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Disclosure of conflict of interest

None.

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