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Evaluation of the Pro-inflammatory Cytokine Tumor Necrosis Factor- α in Adolescents with Polycystic Ovary Syndrome

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Abstract

Background—Patients with Polycystic Ovarian Syndrome (PCOS) often suffer from co-morbidities associated with chronic inflammation characterized by elevations in pro-inflammatory cytokines. There is limited data on markers of chronic inflammation, in particular Tumor Necrosis Factor-alpha (TNF- α), in adolescents with PCOS.

Objectives—To compare serum levels of TNF- α in overweight or obese adolescents with PCOS and obese controls. In the PCOS group, to correlate serum TNF- α levels with body mass index (BMI) z-score, severity of hyperandrogenism, degree of insulin resistance, and ovarian ultrasound (US) characteristics.

Methods—We performed a cross-sectional retrospective analysis of clinical and biochemical findings in 23 overweight or obese adolescent females with PCOS (mean BMI z-score 2, mean age 15.2 yrs) and 12 obese age- and sex-matched controls (mean BMI z-score 2, mean age 14.1 years). All subjects were post-menarcheal. Serum TNF- α levels were compared between groups. In the PCOS group, cytokine levels were correlated with BMI z-score, androgen levels, fasting insulin and glucose levels as well as ovarian US features.

Results—Both groups were comparable in age, BMI z-score, fasting glucose, and fasting insulin. Mean free testosterone was 9.76 ± 5.13 pg/mL in the PCOS group versus 5 ± 2.02 pg/mL in the control group ($p=0.0092$). Serum TNF- α was 7.4 ± 4 pg/mL in the PCOS group versus 4.8 ± 3.16 pg/mL in the control group ($p = 0.0468$). There was no significant correlation between serum TNF- α and BMI z-score, free testosterone, fasting insulin, or fasting glucose. No correlation existed between serum TNF- α and ovarian follicle number, distribution, or volume.

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Conclusions—Serum TNF- α is elevated in overweight/obese adolescents with PCOS. Chronic inflammation in adolescents with PCOS render them at a potential increased risk for the development of atherosclerosis, type 2 diabetes, cancer, infertility, and other co-morbidities. Every effort should be made to identify adolescents with PCOS early and initiate aggressive therapy to prevent future complications.

Keywords

Polycystic Ovary Syndrome; Tumor necrosis factor- α (TNF- α); Adolescents

Introduction

Polycystic Ovary Syndrome (PCOS) is an endocrine disorder that afflicts 5–11% of reproductive aged women and is thought to be the most common endocrinopathy in this population.^{1,2} Recent clinical guidelines suggest that PCOS should be diagnosed in adolescents based on a thorough clinical picture including oligomenorrhea, increased androgen levels, clinical signs and symptoms of androgen excess, and exclusion of other causes of hyperandrogenemia.³ PCOS is known to be associated with a state of low-grade systemic inflammation.^{4–9} Multiple studies in adults with PCOS have demonstrated elevated levels of inflammatory indices such as IL-6, IL-18, TNF- α , and CRP.^{10–11} As many of these factors are produced by adipocytes and PCOS is often associated with obesity, data on whether the increased levels are a result of obesity, PCOS, or both have been conflicting.^{4–10}

A potential link between PCOS and elevations in the proinflammatory cytokine TNF- α is of particular interest, as TNF- α has been shown to impact many aspects of ovarian function, including follicular growth, ovulation, and corpus luteum regression.^{12–14} Ovaries have been shown to both produce TNF- α and contain TNF- α receptors.^{13, 15} In rats, excess testosterone as seen in PCOS has been shown to induce a greater release of TNF- α , thus leading to an increase in androstenedione and a decrease in estradiol production in the rat ovary.¹⁶ It is therefore possible that altered TNF- α levels in PCOS contributes both to the short-term ovarian dysfunction and hyperandrogenic state seen in the disease as well as to long-term effects in the ovaries and in other organ systems.

There is a lack of data examining serum TNF- α in adolescents with PCOS. In this study we compared serum levels of TNF- α in overweight or obese adolescents with PCOS and obese controls. In the PCOS group, we also correlated TNF- α levels with body mass index (BMI) z-score, severity of hyperandrogenism, degree of insulin resistance, and ovarian ultrasound (US) characteristics.

Subjects and Methods

Patient Population

The Institutional Review Board of New York University School of Medicine and Bellevue Hospital approved this cross-sectional retrospective pilot study. Data was reviewed from 2005–2010. All patients were followed by the Pediatric Endocrinology service at Bellevue

Hospital Center or New York University Medical Center, two large urban academic tertiary care centers. We studied 23 females (age 12.3–17.7 years) with PCOS and 12 age, BMI, and sex matched controls (age 11.4–16.5 years). Table 1 shows all subjects baseline demographic and clinical characteristics obtained from a chart review. Exclusion criteria included pregnancy, ovarian or androgen secreting tumor, thyroid dysfunction, hyperprolactinemia, Cushing syndrome, and congenital adrenal hyperplasia. No subject was taking insulin sensitizers or any medication known to influence the menstrual cycle. The diagnosis of PCOS was based on the revised 2003 ESHRE/ASRM Rotterdam criteria (3), which require two out of three following characteristics: (1) oligo/anovulation, (2) clinical/biochemical signs of hyperandrogenism, and/or (3) polycystic ovaries on ultrasound (the presence of 12 or more follicles measuring 2–9 mm in diameter and/or ovarian volume > 10 ml). Hirsutism was defined as a Ferriman-Gallwey grade of 8 or higher. Amenorrhea was defined as cessation of periods for more than 3 months. Oligomenorrhea was defined as less than 6 cycles per year. Only post-menarcheal subjects were selected. Based on data from the National Health and Nutrition Examination Survey, overweight was defined as a BMI between the 85–95th % and obese as a BMI > 95th % for age and sex.

Hormonal Immunoassays

Blood samples from the PCOS group and controls were obtained during routine Pediatric Endocrinology clinic visits. Blood sample collection was not timed with respect to menstrual cycle. Total Testosterone was measured by well validated LC/MS with an analytical sensitivity of 1 ng/dL (Quest Diagnostics). The percent free Testosterone was determined by equilibrium dialysis. The free Testosterone level was calculated based on total and percent free. Biochemical hyperandrogenism was defined as a free Testosterone of 7.5 pg/mL or greater. Fasting serum insulin was determined by radioimmunoassay with a lower limit of detectability of 2.5 µU/mL (Quest Diagnostics). HOMA-IR was calculated by dividing the product of insulin (µU/mL) and glucose (mg/dL) by 405.¹⁷ The HOMA-IR cutoff point for diagnosis of insulin resistance was ≥ 3.16 .¹⁸ Following collection and processing, serum samples for TNF-α were stored at –20° C until hormone analyses were performed. Serum TNF-α was measured in duplicate using human multiplexing bead immunoassays (Biosource, Camarillo, CA) utilizing LuminexRin accordance with the manufacturer's instructions. All serum TNF-α measurements were performed in the same lab using the same assay. The lower limit of detectability for this assay is 0.1 pg/mL.

Ultrasound Data

All PCOS subjects underwent transabdominal pelvic ultrasonography as part of their PCOS diagnostic workup. The images were obtained using a Phillips HDI 5000 ultrasound machine with a curved 5 mHz transducer. No ultrasound data was available for the control group. Ultrasound images were re-reviewed with a Pediatric Radiologist to confirm the reported measurements and to determine ovarian morphology. Ovarian volume was calculated as $0.5233 \times \text{length} \times \text{width} \times \text{thickness}$, derived from the formula for a prolate ellipsoid. An ovarian volume of 10 ml or more was considered enlarged. As transabdominal ultrasound limits follicle visualization, the number of follicles was described as “few” when fewer than 5, “moderate” when between 5 and 10, and “multiple” when greater than 10.

Follicle distribution was described as central, peripheral, or mixed. The presence of cysts greater than 10 mm was noted.

Statistical Methods

A P value of <0.05 was considered significant. Comparisons of two groups on continuous variables were made using the Student t test; for comparisons of more than 2 groups, anova tests were used. The χ^2 tests were performed to assess the correlations between two categorical variables. The correlation between continuous variables was assessed using Pearson's correlation coefficient. All analyses were two-tailed and performed using SAS. Results are reported as mean $\pm$ SD.

Results

Clinical and demographic characteristics (Table 1)

There was no significant difference in mean age between the PCOS group (15.2 ± 1.8 years) and controls (14.1 ± 1.7 years) ($p = 0.08$). BMI Z-score was similar in the PCOS group (2.07 ± 0.48) and controls (2.04 ± 0.25) ($p = 0.84$). The majority of subjects in both groups were Hispanic (78.26% of the PCOS group $n = 18$, 75% of the control group $n = 9$, $p = 0.74$). Of the remaining PCOS subjects, 3 were Asian and 2 were of mixed ethnicity. Of the remaining controls, one was Asian, one was African American, and one was Native American. The average age at menarche was similar between the PCOS group (11.6 ± 1.3 years) and controls (11.4 ± 1.5 years) ($p = 0.6$). Most PCOS subjects were oligo- or amenorrheic (91.3%) while the majority of control subjects had regular menses (91.67%) ($p < 0.0001$). One or more physical signs of hyperandrogenism (acne, alopecia, or male pattern baldness) was present in 86.96% of PCOS subjects compared with 41.67% of controls ($p = 0.01$). There was no significant difference in the presence of acanthosis nigricans between the PCOS group (78.26%) and control group (87.82%) ($p = 0.8$).

Hormonal and biochemical profiles (Table 2)

Serum TNF- α was significantly higher in the PCOS group (7.4 ± 4.08 pg/mL) compared with controls (4.8 ± 3.16 pg/mL) ($p = 0.0468$). The PCOS subjects had significantly higher free Testosterone levels (9.76 ± 5.13 pg/mL) compared to controls (5 ± 2.02 pg/mL) ($p = 0.0092$). Data on fasting insulin and glucose levels were available in 19 out of 23 PCOS subjects and 7 out of 12 control subjects. Of these, there was no significant difference in insulin resistance defined as a HOMA-IR ≥ 3.16 ($p = 0.53$).

Ovarian ultrasound characteristics (Tables 3 and 4)

Transabdominal pelvic ultrasound data was available for all PCOS subjects.

Ovarian Volume—Mean left ovarian volume was 9.75 ± 5.78 mL. Mean right ovarian volume was 9.55 ± 4.07 mL. Overall mean ovarian volume was 9.65 ± 4.58 mL. Ovarian volume was greater than 10 mL in 35% of images. Serum TNF- α did not significantly correlate with ovarian volume (left ovary $r = -0.25$, $p = 0.25$, right ovary $r = 0.01$, $p = 0.95$).

Ovarian Morphology—Peripheral follicular distribution was noted in 76% of ultrasound images ($n = 35$). In the remainder, the follicular distribution was mixed ($n = 11$). No significant correlation existed between serum TNF- α and peripheral follicle distribution (left ovary $p = 0.47$, right ovary $p = 0.16$). In 38/46 ultrasound images, more than 10 follicles were noted. In the remainder, between 5 and 10 follicles were noted (83% of images demonstrated greater than 10 follicles). There was no significant correlation between serum TNF- α and follicle number ($p = 0.69$ on left ovarian images, $p = 0.62$ on right ovarian images).

Discussion

This pilot study demonstrates significantly increased serum TNF- α levels in overweight and obese adolescent females with PCOS compared with BMI- and age-matched controls. Elevated serum TNF- α reflects a state of chronic inflammation with potential negative implications for the future development of cardiovascular disease, metabolic disease, and cancer in this young study population.

We examined obese and overweight adolescent females with and without PCOS ages 11–17 years and found an elevation in serum TNF- α in those with PCOS without correlation to BMI. Although it is difficult to delineate whether raised serum TNF- α contributes to the pathogenesis of PCOS or is an effect of PCOS, these results suggest that a factor intrinsic to PCOS may contribute to the elevated levels. Although TNF- α impacts follicular growth, ovulation, and corpus luteum regression, we did not find a significant correlation between serum TNF- α and ovarian ultrasound characteristics common in PCOS, such as enlarged ovarian volume, increased follicular number, or peripheral follicle distribution.^{12–14} This may also be related to recent suggestion that multifollicular ovaries are a feature of normal puberty that may be difficult to distinguish from PCO morphology.³

There are potential implications of elevated serum TNF- α on multiple organ systems. TNF- α promotes the synthesis of IL-6, which regulates the synthesis of C-reactive protein, an acute phase reactant.⁹ Both IL-6 and C-reactive protein are considered strong risk markers for cardiovascular events.⁹ TNF- α has been implicated in plaque extension in atherosclerosis as well as autoimmune diseases, such as systemic lupus erythematosus.^{19, 20} TNF- α may also help induce osteoporosis by helping to activate osteoclasts.¹⁹ The release of TNF- α may promote viral infections, such as HIV.²¹ Elevated serum TNF- α may play a role in ovarian carcinogenesis and metastasis.²² Studies have shown increased expression of TNF- α in ovarian carcinoma tissue compared to normal ovarian epithelial cells and elevated serum TNF- α levels in patients with ovarian cancer compared to controls.^{12, 23, 24} A positive correlation has been identified between TNF- α and endometrial cancer, the development of which may be more common in women with PCOS.^{25, 26}

Our study demonstrates increased serum TNF- α in overweight/obese adolescents with PCOS. The data reported here represent a pilot study and, therefore, did not benefit from the advantages inherent to a larger sample size with greater power. As this was a retrospective study, it was not possible to follow subjects over time to examine outcome measures such as the possible development of disease states with inflammatory pathogenesis. In addition,

although in this cross-sectional retrospective study, ultrasound characteristics did not correlate with levels of TNF- α , it would have been ideal to have longitudinal ultrasound data as well as control group data for comparison. It was an advantage of the study to have BMI-matched subjects and controls. However, serum TNF- α levels in normal weight adolescents with PCOS would have allowed for further control of the potentially confounding factor of increased visceral fat.

Serum TNF- α levels may be elevated in adolescent patients with PCOS. These high levels may contribute to the potential development of infertility, atherosclerosis, type 2 diabetes, and certain cancers over an extended period of time. Future studies are needed to clarify the effects of high serum TNF- α in PCOS patients and better delineate the long term implications.

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Table 1

Demographic and clinical characteristics of PCOS subjects and obese controls

	PCOS	Controls	p
n	23	12	
Age (years)	15.2 ± 1.84	14.08 ± 1.7	0.08
Ethnicity			0.74
Hispanic	18 (78.3%)	9 (75%)	
Asian	3 (13%)	1 (8.3%)	
Other*	2 (8.7%)	2 (16.7%)	
BMI (kg/m ²) Z Score	2.07 ± 0.48	2.04 ± 0.25	0.84
Age at menarche (years)	11.63 ± 1.31	11.35 ± 1.45	0.6
Menstrual history			0.0001
Regular	2 (8.7%)	11 (91.7%)	
Oligomenorrheic	14 (60.9%)	1 (8.3%)	
Amenorrheic	7 (30.4%)	0	
Acne present	15 (65.2%)	8 (66.7%)	0.18
Hirsute	17 (73.9%)	0	0.0001
Acanthosis nigricans present	18 (78.3%)	10 (83.3%)	0.8

Data are expressed as n (%) and mean ± SD.

* Other = African American, Native American, or mixed ethnicity

Table 2

Hormonal and biochemical profiles of PCOS subjects and controls

	n	PCOS	n	Controls	p value
TNF-α (pg/mL)	23	7.4 ± 4.08	12	4.8 ± 3.16	0.0468
free Testosterone (pg/mL)	23	9.76 ± 5.14	6	5 ± 2.84	0.0092
fasting glucose (mg/dL)	19	88.63 ± 12.82	10	89.2 ± 8.61	0.89
fasting insulin (μU/L)	19	21.12 ± 10.5	7	18.96 ± 7.61	0.58
HOMA-IR	19	4.79 ± 2.45	7	4.55 ± 1.43	0.53

Data expressed as mean ± SD.

Table 3

Pelvic ultrasound characteristics of PCOS group

Ovarian Volume (mL)	
Right ovarian volume	9.55 ± 5.78
Left ovarian volume	9.75 ± 4.07
Mean ovarian volume	9.65 ± 4.58
Follicle Distribution (%)	
Peripheral	76%
Mixed	24%
Follicle Number	
Few	0
Moderate	17
Multiple	83

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Correlation between serum TNF- α and PCOS subjects' pelvic ultrasound indices, degree of obesity, and hormonal data

Table 4

	<i>r</i>	<i>p</i>
Ultrasound indices		
Ovarian volume		
Left ovary	-0.25	0.25
Right ovary	0.01	0.95
Mean ovarian volume	-0.15	0.49
Peripheral follicle distribution		
Left Ovary		0.47
Right Ovary		0.16
Follicle Number		
Left Ovary		0.69
Right Ovary		0.62
Degree of obesity		
BMI Z Score	0.18	0.41
Hormonal data		
Free testosterone (pg/mL)	-0.03	0.91
Fasting insulin (μ U/mL)	-0.07	0.79