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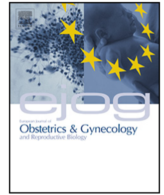

Abdominal obesity can induce both systemic and follicular fluid oxidative stress independent from polycystic ovary syndrome

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ABSTRACT

Objective: The abdominal form of obesity is prevalent in women with polycystic ovary syndrome (PCOS). Visceral fat accumulation seems to play an important role in etiology of PCOS. In this cross-sectional study we evaluated the association of oxidative stress (OS) induced with PCOS and abdominal obesity in serum and follicular fluid (FF) of infertile women.
Study design: A total of 80 women younger than 37 years old undergoing an IVF program were studied in the same period of time from September 2012 to October 2013. Blood serum and FF obtained from 40 women with PCOS (diagnosed by the Rotterdam 2004 criteria) and 40 women without PCOS undergoing IVF were evaluated for two OS markers: lipid peroxide (LPO) and total antioxidant capacity (TAC), after puncture. The patients were divided into 4 groups on the basis of presence of PCOS and waist-to-hip ratio (WHR) or abdominal obesity (OA).
Results: Healthy and PCOS women with abdominal obesity had significantly higher amounts of LPO in the serum and FF as compared with women without abdominal obesity. LPO concentration in FF was significantly lower than in serum and corroborates the hypothesis that the germinal cells have a potent antioxidant mechanism. We also found that LPO concentration in the PCOS group associated with AO had an increasing trend vs. those AO patients without PCOS but this difference was not significant, so the increase in LPO level was approximately independent of PCOS. Based on our results, the association and interaction between PCOS and AO can lead to TAC concentration reduction in patients.
Conclusions: Abdominal obesity can induce local and systemic oxidative stress in PCOS and non-PCOS patients. We suggest that PCOS-induced disorders are likely to be exacerbated in the presence of abdominal obesity.

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Introduction

The polycystic ovary syndrome (PCOS) is a common endocrine disorder [1] with a prevalence of 4–10% in women of reproductive age [2] and it is known to be the main cause of anovulation infertility in these patients [3]. PCOS is characterized by the presence of at least two out of three main features: detection of polycystic ovaries by sonography, hyperandrogenism and chronic oligo/anovulation in patients [4]. It has been defined that the main factors responsible for prevalence of PCOS are genetic and adventitious (environmental) factors and the most important environmental factor is dietary habits [3].

Most women with PCOS inconsistently demonstrate the features of metabolic syndrome, such as insulin resistance (IR) and obesity [5,6]. Approximately 70% of these patients manifest IR and about 50–75% display obesity, especially the abdominal phenotype [3,5,7]. Hyperandrogenism and other clinical features (menstrual abnormalities and anovulation) of PCOS in obese patients are higher than in normal weight patients [4].

It seems that abdominal obesity (AO) may play an important role in the development of PCOS in susceptible individuals, because its consequent insulin resistance can lead to ovarian and in some case, adrenal hyperandrogenism. Resultant androgen excess may

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then also lead to more and more accumulation of fat in the abdominal site [4]. This vicious cycle can result in the development of PCOS complications.

Despite the strong association between AO and PCOS, as well as its important role in PCOS complications, the cause of this correlation is not yet fully clear. It seems, however, that induction of oxidative stress (OS) by visceral accumulation of adipose tissue possibly has a significant effect on PCOS etiology [8]. In this study, to evaluate this hypothesis, the concentration of two OS markers, lipid peroxides (LPO) and total antioxidant capacity (TAC) in blood serum and follicular fluid (as a location reflecting metabolic processes occurring around the mature oocyte before ovulation) of PCOS women with and without AO were measured and compared with healthy women with and without AO.

Materials and methods

Patient selection

A total of 80 women younger than 37 years old undergoing an IVF program were studied in this cross-sectional study. All the participants gave their written informed consent for the collection of blood and follicular fluid samples. Approval for this study was obtained from the Local Ethics Committee of the Royan Institute. The patients were divided into 4 groups on the basis of the presence of PCOS (diagnosed by the Rotterdam 2004 criteria) and waist-to-hip ratio (WHR). Table 1 demonstrates the characteristics of the four groups.

Stimulation protocol

Patients underwent standard controlled ovarian stimulation, which included suppression of pituitary gonadotropin secretion with the GnRH agonist buserelin acetate (Suprefact, Hoechst AG, Germany) by subcutaneous injection (500 mg/d) or by nasal spray (800 mg/d) at the mid luteal phase of the preceding ovarian cycle (day 21). Once ovarian suppression was confirmed, ovarian stimulation was initiated with recombinant FSH (Gonal F; SC injection, 150 IU/d, Serono, Switzerland). When the average diameter of at least three follicles reached at least 18–20 mm, a single dose of hCG (10,000 IU; Pregnyl; Organon, Netherlands) injection was given. Oocytes were retrieved 36 h later using a standard ultrasonically guided follicular puncture.

Sample collection

Blood samples were collected in 10 ml non-heparinized glass tubes just before intravenous injection of an anesthetic for ovarian puncture. FF of each follicle in all patients was aspirated separately within tubes. FFs from follicles smaller than 15 mm, follicles without an egg or with more than 1 oocyte and FF with blood contamination were discarded. The coagulated blood and FF samples were centrifuged at 300 × g for 7 min after transferring to the laboratory, to remove cellular remnants, and clear supernatant was frozen at −176 °C (in liquid nitrogen) and kept for up to 1 month before measurements.

Determination of TAC

A sensitive, easy and rapid assay, known as ferric reducing/antioxidant power (FRAP) [9], was used to measure TAC (an indicator of reactive oxygen species (ROS) activity), both in serum and FF. This assay utilizes the antioxidants as reductants in a redox-linked colorimetric method which absorbance is measured by spectrophotometer (CECIL CE7250. Co: Bio Aquarius, England). A 300 mmol/l acetate buffer (pH 3.6) (3.1 g of sodium acetate $3H_2O$ + 16 ml of glacial acetic acid made up to 11 ml with distilled H_2O), 10 mmol/l 2,4,6-tri-(2-pyridyl)-1,3,5-triazine 98% (Sigma–Aldrich) (3.1 mg/ml in 40 mmol/l HCL) and 20 mmol/l $FeCl_3 \cdot 6H_2O$ in the ratio of 10:1:1 were mixed together as a working FRAP reagent. Then a 50 μl of sample (FF or serum) was added to 1 ml of FRAP reagent in a cuvette. Just 10 min after mixing, absorbance was measured at 593 nm (A_{593}) far from direct sunlight and at room temperature by means of 50 μl water as the reference.

Measurement of LPO

Serum and FF concentrations of malondialdehyde (MDA), as an index of lipid peroxidation, were measured using thiobarbituric acid (TBA) method according to Das et al. [10]. In this method, MDA reacts with TBA and provides a red compound which has an absorbance at 535 nm. Two millilitres of stock reagent (12% w/v tricholoro-acetic acid, 0.375% w/v TBA and 0.25 mol/l warm HCL to dissolve the TBA) was blended with 1 ml of thawed sample and heated in a boiling water bath for 15 min and centrifuged for 10 min at 1000 × g after cooling. Optical density of supernatant fluid was measured against a blank including all the reagents alone. LPO concentration was parented as μM MDA.

Table 1
Comparison of the women's characteristics among four studied groups.

Patients (*n*)	A: PCOS, with AO (*n* = 20)	B: PCOS, without AO (*n* = 20)	C: without PCOS, with AO (*n* = 20)	D: without PCOS and AO (*n* = 20)	*P* value
Age (years)	32.3 ± 3.7	30 ± 2.9	31 ± 4.6	29 ± 3.1	<0.001[a]
WHR	0.87 ± 0.19	0.83 ± 0.11	0.87 ± 0.15	0.82 ± 0.2	<0.001
BMI (kg/m²)	27.1 ± 1.1	26.4 ± 1.3	26.8 ± 0.9	26.2 ± 1.3	<0.001[b]
FSH	5.4 ± 2.7	4.7 ± 2.1	8.1 ± 4.3	7.2 ± 3.5	0.005[c]
LH	0.36 ± 0.57	0.33 ± 0.35	0.30 ± 0.34	0.32 ± 0.34	0.9
Free testosterone	1.7 ± 1.2	0.9 ± 0.5	0.5 ± 0.2	0.5 ± 0.3	<0.001[d]
Serum TAC	0.68 ± 0.1	0.72 ± 0.09	0.68 ± 0.1	0.72 ± 0.09	0.02[e]
FF TAC	0.57 ± 0.09	0.67 ± 0.05	0.67 ± 0.1	0.69 ± 0.07	<0.001[f]
Serum LPO	1.3 ± 0.4	0.95 ± 0.2	1.0 ± 0.2	0.88 ± 0.2	<0.001[g]
FF LPO	1.0 ± 0.3	0.85 ± 0.2	1. 0 ± 0.3	0.79 ± 0.2	<0.001[h]

Data are mean ± SD; AO: abdominal obesity; WHR: waist/hip ratio (WHR > 0.85 = AO); BMI: body mass index.
[a] Group A vs. group B (*P* = 0.01), group A vs. group D (*P* < 0.001), group C vs. group D (*P* = 0.03), differences between others group were not significant.
[b] Group C vs. group D (*P* < 0.001), group A vs. group D (*P* < 0.001), group B vs. group D (*P* < 0.001), differences between others group were not significant.
[c] Group A vs. group C (*P* = 0.05), group B vs. group C (*P* = 0.009), differences between others group were not significant.
[d] Group A vs. group B (*P* = 0.002), group A vs. group C (*P* = 0.001), group A vs. group D (*P* = 0.001), differences between others group were not significant.
[e] Group A vs. group B (*P* = 0.03), group A vs. group D (*P* = 0.06), differences between others group were not significant.
[f] Group A vs. group B (*P* = 0.001), group A vs. group C (*P* = 0.001), group A vs. group D (*P* < 0.001), differences between others group were not significant.
[g] Group A vs. group B (*P* = 0.001), group A vs. group C (*P* = 0.03), group A vs. group D (*P* < 0.001), differences between others group were not significant.
[h] Group A vs. group B (*P* = 0.01), group A vs. group D (*P* = 0.001), group B vs. group C (*P* = 0.01), differences between others group were not significant.

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