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ORIGINAL ARTICLE

The effects of adipocytokines on the endocrino-metabolic features and obstetric outcome in pregnant obese women with polycystic ovary syndrome

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KEYWORDS

Polycystic ovary syndrome;
Pregnancy outcome;
Android obesity;
Gynoid obesity;
Adipocytokines

Abstract *Objective:* To estimate the effects of adipocytokines on the metabolic and endocrine features, and the obstetric outcome in pregnant women with polycystic ovary syndrome (PCOS).

Design: Prospective cross sectional study.

Main outcome measures: plasma concentration of adipocytokines, insulin resistance/hyperinsulinism (IR/HI), lipid profile, androgens and obstetric outcome.

Materials and methods: The study included hundred pregnant PCOS (PPCOS) women with android obesity (group 1), 100 pregnant non-PCOS women with android obesity (group 2), 100 PPCOS women with gynoid obesity (group 3), and 100 pregnant non-PCOS women with gynoid obesity (group 4). All patients in the four groups were primigravidae and women with PCOS (groups 1 and 3) became pregnant after treatment with clomiphene citrate and/or gonadotropins.

Plasma concentrations of fasting glucose, fasting serum insulin, insulin sensitivity by quantitative insulin sensitivity check index (QUICKI), serum concentrations of triglycerides (TGs), total cholesterol (TC), high density lipoprotein cholesterol, (HDL-c), low density lipoprotein cholesterol (LDL-c); plasma adipocytokines: interleukin (IL)-10, adiponectin (both are insulin sensitizers and anti-inflammatory), pro-inflammatory cytokines: IL-6, prothrombin activator inhibitor-1(PAI-1), high sensitivity C-reactive protein (hsCRP); serum total testosterone (TT), sex-hormone binding globulin (SHBG), free androgen index (FAI) were estimated for the four groups between

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22 and 24 weeks' gestation Glucose loading test was done at 22–24 weeks' gestation to check for gestational diabetes mellitus and if normal it was repeated at 30–34 weeks. Gestational hypertension (GH), preeclampsia (PE) and preterm labor (PTL) (delivery < 37 weeks' gestation) were recorded.

Results: Groups 1 and 2 with android obesity had IR/HI, (QUICKI $< 0.331 \pm 0.010$). Groups 3 and 4 with gynoid obesity had normal insulin sensitivity (NIS), (QUICKI $> 0.331 \pm 0.010$). Serum concentration of TGs, LDL-c, and plasma concentration of IL-6, PAI-1, hsCRP were significantly higher in groups 1 and 2 than groups 3 and 4. Serum HDL-c, plasma IL-10 and adiponectin were significantly higher in groups 3 and 4 than groups 1 and 2. Serum TT and FAI were significantly higher in groups 1 and 3 (cases of PPCOS) than their controls. There was no significant difference in the serum concentration of TC between the four groups.

Incidence of spontaneous early miscarriage (SM) in groups 1, 2, 3, and 4 was 36%, 12%, 33%, and 11% respectively. Incidence of SM was significantly higher in PPCOS than non-PCOS pregnancy irrespective of the type of obesity.

Rate of late pregnancy complications, GDM, GH, PE and PTL was significantly higher in groups 1 and 2 with android obesity than groups 3 and 4 with gynoid obesity. There was no significant difference in the rates of cesarean section (CS) between the 4 groups. The rates of neonatal complications and perinatal mortality were significantly higher in groups 1 and 2 (android obesity) than groups 3 and 4 (gynoid obesity).

Conclusion:

1. TT and FAI were significantly higher in PPCOS with android obesity than PPCOS with gynoid obesity.
2. Incidence of early SM (9–12 weeks) was significantly higher in PPCOS than in non-PCOS pregnancy irrespective of the type of obesity.
3. Incidence of late-onset pregnancy complications, GDM, GH, PE and PTL was significantly higher in patients with android obesity than patients with gynoid obesity. PCOS *per se* seemed to be not related to the incidence of late-onset pregnancy complications.Pregnant patients with android obesity, (both PCOS and non-PCOS) with diminished serum concentration of anti-inflammatory cytokines, and increased serum concentration of pro-inflammatory cytokines had IR/HI and dyslipidemia. Pregnant patients with gynoid obesity, (both PCOS and non-PCOS) with normal serum concentration of anti- and pro-inflammatory cytokines had NIS and normal lipid profile.
4. In pregnancy with android obesity patients had reduced plasma concentration of IL-10 and increased concentration of IL-6 which may impair the development of the placenta with increased risk of PTL.
5. Neonatal complications and perinatal mortality were significantly higher in PPCOS with android obesity than PPCOS with gynoid obesity.

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1. Introduction

The prevalence of obesity has increased dramatically over the past three decades. Polycystic ovary syndrome (PCOS) is a common endocrine disorder in women of reproductive age. The estimated prevalence of this disease is 5–10% as reported in population based studies. Approximately 80% of patients with PCOS are obese. This makes PCOS coincides with the metabolic syndrome. Obese pregnant PCOS (PPCOS) women may have the cumulative metabolic effects of pregnancy and PCOS [1].

The impacts of these metabolic disorders on the outcome of pregnancy are controversial. Mikola et al. [2] found a lack of association between PPCOS and pregnancy complications especially preeclampsia (PE). Kashyap and Claman [3] found that many pregnancy complications increased significantly in PPCOS versus non-PCOS controls.

Boomsma et al. [4] performed a meta-analysis of 15 valid studies including 720 women presenting with PPCOS and 4505 pregnant controls. They found a significantly increased incidence of pregnancy complications as gestational diabetes mellitus (GDM), gestational hypertension (GH), PE and preterm labor (PTL) versus controls. They stated that in their analysis, a higher multiple pregnancy rate, parity, age, socioeconomic class, and BMI in women with PCOS were important potential confounders. They believed that there is a need for studies avoiding these confounders.

The aim of this study was to estimate the effects of blood adipocytokines on both endocrino-metabolic features and obstetric outcome of obese PPCOS women avoiding the mentioned confounders.

2. Materials and methods

Between March 2007 and March 2013, 200 obese patients (body mass index (BMI) $\geqslant 30$) with PCOS, attending Infertility Clinic, Tanta University Hospitals, became pregnant after treatment with clomiphene citrate and/or gonadotropins, 100 patients with android obesity (group 1) and 100 patients with gynoid obesity (group 3). Hundred non-PCOS pregnant

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