Endokrynologia Polska
Tom/Volume 64; Numer/Number 2/2013
ISSN 0423–104X

Body composition, glucose metabolism markers and serum androgens — association in women with polycystic ovary syndrome

Skład ciała, wskaźniki metabolizmu węglowodanów i androgeny w surowicy — zależności u kobiet z zespołem policystycznych jajników

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Abstract

Introduction: To evaluate the association between abdominal and gynoid fat, glucose and lipid metabolism markers, and serum androgens in women with polycystic ovary syndrome (PCOS).

Material and methods: Anthropometric measurements were performed in 40 women with PCOS aged 19–49 years with body mass index (BMI) 18.7–53.8 kg/m^2. Fasting serum glucose, lipids, insulin, leptin, LH, FSH, oestradiol, androgens, SHGB and TSH were estimated. Body composition was measured by DEXA scan.

Results: Four women (10%) were overweight, and 23 (57.5%) were obese. All subjects were hyperandrogenic (in 33 serum androgen levels were increased), and 16 of them were insulin resistant. All of the obese subjects had the abdominal type of obesity. Body weight, BMI, fat mass, fat mass of the trunk, abdominal and gynoidal fat mass correlated with serum triglyceride, glucose and insulin levels, and with HOMA index and blood pressure. Free androgen index (FAI) correlated with body weight ($r = 0.43$, $p < 0.01$), and BMI ($r = 0.46$, $p < 0.05$).

Conclusions: Using the DEXA method, we demonstrated abdominal type of obesity in all our obese subjects. There were positive significant correlations between fatness, lipids and glucose metabolism indices and blood pressure. Direct positive correlations between free androgen index, body weight and BMI were found. **(Endokrynol Pol 2013; 64 (2): 94–100)**

Key words: *polycystic ovary syndrome, body composition, obesity, hyperandrogenism, insulin resistance*

Streszczenie

Wstęp. Ocena zależności między masą tłuszczu adipoidalnego i gynoidalnego a wskaźnikami metabolizmu węglowodanów i lipidów oraz androgenami u kobiet z zespołem policystycznych jajników (PCOS).

Materiał i metody: U 40 kobiet z PCOS w wieku 19–49 lat, z BMI 18,7–53,8 kg/m^2 dokonano pomiarów antropometrycznych oraz określono na czczo stężenie glukozy, lipidów, insuliny, leptyny, LH, FSH, estradiolu, androgenów, SHBG i TSH. Skład ciała oceniono metodą DEXA.

Wyniki: Cztery kobiety (10%) miały nadwagę, dwadzieścia trzy (57,5%) były otyłe. U wszystkich badanych stwierdzono hiperandrogenizm (u 33 stężenie androgenów w surowicy było podwyższone), a u 16 oporność insulinową. Wszystkie osoby otyłe miały otyłość brzuszną. Wykazano korelację między masą ciała, BMI, masą tłuszczu całkowitego i tułowia, a także tłuszczu adipoidalnego i gynoidalnego a stężeniem triglicerydów, glukozy, insuliny, HOMA i wartościami ciśnienia tętniczego. Wskaźnik wolnych androgenów (FAI) wykazywał korelację z masą ciała ($r = 0,43$, $p < 0,01$) i BMI ($r = 0,46$, $p < 0,05$).

Wnioski: Za pomocą metody DEXA wykazano brzuszny typ otyłości u wszystkich badanych otyłych kobiet z PCOS. Stwierdzono dodatnią korelację między wykładnikami otyłości a badanymi wskaźnikami metabolizmu lipidów i węglowodanów oraz wartościami ciśnienia tętniczego krwi. Wykazano bezpośrednią korelację między wskaźnikiem wolnych androgenów a masą ciała i BMI. **(Endokrynol Pol 2013; 64 (2): 94–100)**

Słowa kluczowe: *zespół policystycznych jajników, skład ciała, otyłość, hiperandrogenizm, insulinooporność*

This study was supported by a grant from the Medical Centre of Postgraduate Education, Warsaw, Poland; No: 501-2-1-07-22/09.

Introduction

Polycystic ovary syndrome (PCOS) is considered to be the most frequent endocrine disorder in women of reproductive age and is present in 5–10% of them [1]. The criteria for diagnosing this syndrome were determined by the international consensus conference in Rotterdam in 2003 [2]. The pathogenesis of PCOS is still far from clear, and it most likely results from a complex of genetic and environmental factors. Women with this syndrome present with marked clinical heterogeneity, although hyperpulsatile gonadotrophin secretion, disturbed ovarian and adrenal steroidogenesis, menstrual irregularity, reduced

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levels of SHBG, and insulin resistance are common features [3–5]. The arrest of antral follicle growth that is observed in PCOS is associated with an abnormal endocrine environment throughout the menstrual circle: hypersecretion of luteinising hormone (LH), and perhaps androgens, and suppression of follicle-stimulating hormone (FSH). Moreover, the intrinsic abnormalities of folliculogenesis in PCOS that affects the very earliest, gonadotrophin independent, stages of follicle development (abnormal granulosa cell proliferation and disparate growth of oocyte and surrounding granulosa cells) perhaps contribute to inhibition of maturation of follicles in the cohort. Also abnormal signalling of local regulators: anti-Müllerian hormone, insulin-like growth factors and sex steroids may play a part in disordered folliculogenesis in PCOS. Since eggs are rarely or never released from their follicles — multiple ovarian cysts develop over time.

Approximately half of the patients with PCOS are overweight or obese [6]. Abdominal obesity that seems to predominate in these cases is a major underlying factor in insulin resistance. However, decreased insulin sensitivity with subsequent hyperinsulinaemia was found also in lean women with PCOS [7]. Excessive adipose tissue, especially abdominal fat, secretes several metabolic factors, with proinflammatory cytokines among them. In recent studies, elevated inflammatory markers, perturbations in fibrinolytic pathways and endothelial dysfunction — a sign of subclinical cardiovascular damage, have been shown in women with PCOS [8-9]. Foltyn et al. demonstrated elevated levels of E-selectin, endothelin-1 and von Willebrand Factor antigen — markers of endothelial dysfunction in women, with this syndrome [10]. Also, androgen excess and fertility disorders contribute to vascular damage in PCOS [11].

A number of different methods of investigating body composition have been devised. Noninvasive, anthropometric measures are widely used because of their simplicity and convenience. Techniques of directly measuring adiposity including computed tomography, total body water or total body potassium estimations have important limitations: exposure to ionising radiations, relatively high cost or methodological complexity. In our study, we used dual-energy X-ray absorptiometry (DEXA).This allows accurate and simple measurement, both of total and regional fat, with marginal exposure to radiation.

The aim of our study was to evaluate the association between abdominal and gynoid fat, glucose and lipid metabolism markers, blood pressure and serum androgen levels in women with polycystic ovary syndrome.

Material and methods

We evaluated 40 women with PCOS aged 19-49 years, mean 28.6 ± 7.6 (x ± SD) with BMI 18.7 – 53.8 kg/m^2, mean 32.27 ± 9.3 The diagnosis of PCOS was based on the Rotterdam consensus criteria: the presence of at least two of the following features: 1) oligomenorrhoea or amenorrhoea; 2) clinical and/or biochemical evidence of hyperandrogenaemia; and 3) polycystic ovaries in ultrasound imaging. Oligomenorrhoea was defined as menstrual periods that occur at intervals of greater than 35 days, with only four to nine periods in a year, and amenorrhoea as the complete absence of menstruation.

Clinical hyperandrogenaemia was defined as the presence of hirsutism or acne. Biochemical hyperandrogenaemia was defined as serum testosterone levels greater than 0.9 ng/mL, dehydroepiandrosterone-sulphate levels greater than 2,000–4,100 ng/mL depending on age, or free androgen index (FAI) > 5.

Ovaries in USG were defined as polycystic when they included either ten or more follicles measuring 2–9 mm in diameter or their volume was greater than 10 cm^3.

The exclusion criteria included hypothyroidism, hyperprolactinaemia (defined as serum prolactin levels greater than 25 ng/mL), Cushing's syndrome, nonclassical congenital adrenal hyperplasia (defined as serum 17-hydroxyprogesterone levels greater than 1.2 and 5.2 ng/mL in the follicular and luteal phase, respectively) and current or previous (within the last three months) use of oral contraceptives and other hormonal, antidiabetic and antiobesity drugs.

A screening session included full physical examination, laboratory tests and imaging. Subjects were studied after an overnight fast. They underwent assessment of body height and weight, and then body mass index (BMI) was calculated. Blood was collected at about 8.00am for glucose, lipids (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol and triglycerides), insulin, leptin, LH, FSH, oestradiol, testosterone, dehydroepiandrosterone-sulphate, 17-hydroxyprogesterone, sex hormone-binding globulin (SHBG), prolactin, free thyroxine, and thyroid-stimulated hormone (TSH) through an iv catheter placed in the forearm. FAI was calculated as testosterone (nmol/L)/SHBG (nmol/L) levels. To estimate insulin resistance, the HOMA index was calculated by the formula: fasting plasma insulin (microinternational units per millilitre) x fasting plasma glucose (millimoles per litre)/22.4. Subjects were considered as insulin resistant when HOMA index was > 2.5. Hypercholesterolaemia was defined as total cholesterol level above 5.2 mmol/L and hypertriglycerydaemia when triglycerides levels were higher than 1.81 mmol/L.

During the same stay in hospital, but usually on another day, all of the subjects underwent transvaginal ultrasonography (TV-USG) and USG of abdomen (to exclude adrenal pathology). At the end of the investigation, body composition by DEXA was determined. The same two operators performed all TV-USG and DEXA measurements, respectively.

The local ethics committee approved this study, and informed consent was obtained from all the participants.

Assays

Glucose was measured with glucose hexokinase reagent set with sensitivity 2.16 mg/dL. An enzymatic colorimetric method was used to measure total cholesterol in the presence of cholesterol oxidase and esterase. The sensitivity was 0.116 mg/dL. HDL-cholesterol was measured with the enzymatic colorimetric method; sensitivity was 3 mg/dL. Triglycerides were also measured with the enzymatic colorimetric method with sensitivity 0.85 mg/dL. All mentioned biochemical measurements were performed using a Roche Cobas Integra 400 chemistry analyser (Roche Diagnostics). Insulin was measured by the immunoradiometric method (Insulin IRMA — Immunotech SA, France); sensitivity was 2.0 mIU/mL. Leptin was measured by RIA (Linco Res. Inc, USA), using rabbit antibodies against human leptin. The sensitivity for this assay was 0.5 ng/mL.

LH, FSH and TSH were measured by the immunochemiluminescence method with IMMULITE 2000 (Siemens Healthcare Diagnostics, Inc). Oestradiol was measured with the same IMMULITE 2000 analyser; sensitivity was 15 pg/mL. Total testosterone was measured by the RIA-CT method (Immunotech SA, France); sensitivity of this method was 0.025 ng/mL. Dehydroepiandrosterone-sulphate was measured by the RIA-CT method (Spectria, Orion Diagnostica, Finland); sensitivity of this method was 10 ng/mL. 17-hydroxyprogesterone was measured by 17OH-RIA-CT Kit (DIAsource ImmunoAssays SA, Belgium); detection limit: 0.02 ng/mL. SHBG was measured by IRMA method (Orion Diagnostics Oy, Finland); detection limit 1.3 nmol/L. Prolactin was measured by IMMULITE 2000 (Siemens Healthcare Diagnostics, Inc); sensitivity of this method was 0.01 μg/mL.

Body mass index was calculated as body weight [kg]/height [m^2]. Subjects with BMI between 25 and 30 kg/m^2 were considered to be overweight, whereas subjects with BMI between 30 and 40 kg/m^2 were considered to be obese, and with BMI above 40 kg/m^2 to be morbidly obese.

To perform measurements of fat mass, we used a region of interest (ROI) programme. In this method, abdominal fat is estimated in the region between the upper part of the pelvis with the upper margin 96 mm superior to the lower part of this region. The lateral part of this region is defined by the lateral part of the thorax. The upper part of the gynoid fat region is defined by the superior part of the trochanter major, with the lower margin 96 mm inferior to the upper part of the trochanter major. The lateral part of this region is defined by the subcutaneous tissue on the hip, which can be visualised using the Image Values option (Fig. 1). We used Lunar Prodigy (GE Lunar, Madison, WI, USA) equipment, which was calibrated each day with a standardised phantom and serviced regularly. The coefficient of variation for measurements of body composition with this method is about 2%.

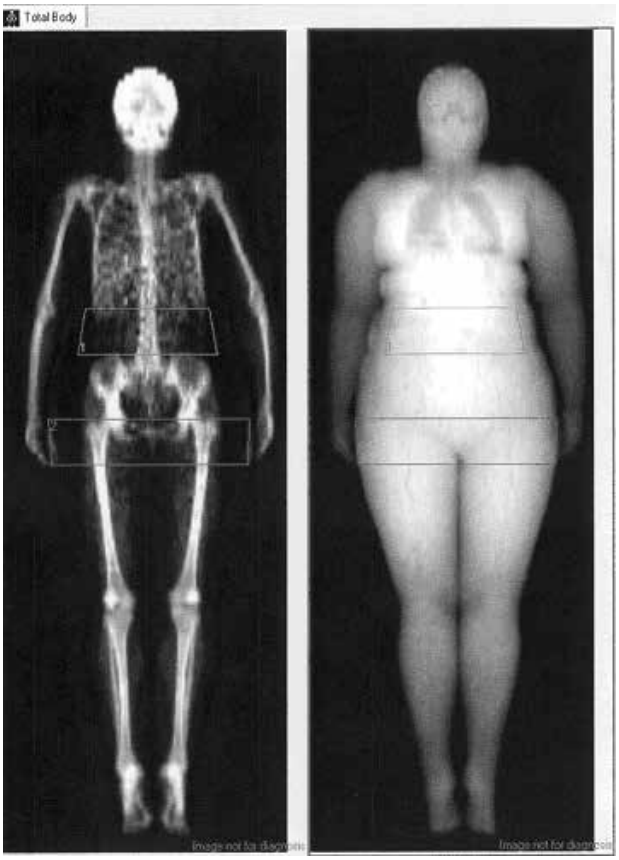

Figure 1. *Example of the regions of interest (ROI) delimiting abdominal (**1**) and gynoid (**2**) fat in one of our studied obese women with polycystic ovary syndrome*

Rycina 1. *Przykład regionów (ROI) wyznaczających obszary tłuszczu brzusznego (**1**) i gynoidalnego (**2**) u badanych kobiet z zespołem policystycznych jajników*

Statistical analysis

All data is presented as the mean ± SD. The normality of the distribution of variables was verified with Kolmogorov-Smirnov and Lilieforse tests. To examine bivariate relationships between data, Pearson correlation or Spearman rank analyses were used. Compari-

Table I. *Anthropometric characteristics, body composition, blood pressure and biochemical results in women with polycystic ovary syndrome*

Tabela I. *Wskaźniki antropometryczne, skład ciała, ciśnienie tętnicze i wyniki badań biochemicznych u kobiet z zespołem policystycznych jajników*

No		N	Mean ± SD	Range
1	Age (years)	40	28.62 ± 7.6	19.0–49.0
2	Height [m]	40	1.6 ± 0.06	1.51–1.85
3	Weight [kg]	40	88.3 ± 27.1	48.0–163.0
4	BMI [kg/m^2]	40	32.26 ± 96.3	18.7–53.8
5	Total FM [kg]	36	41.4 ± 18.9	9.6–82.4
6	FMt [kg]	36	20.8 ± 9.1	4.5–36.9
7	Abdominal FM [kg]	36	3.24 ± 1.2	1.37–6.40
8	Gynoid FM [kg]	36	4.62 ± 2.3	0.34–8.3
9	Glucose [mmol/L]	40	5.02 ± 0.8	4.0–4.45
10	Total cholesterol [mmol/L]	40	6.4 ± 8.9	3.4–61.4
11	LDL cholesterol [mmol/L]	40	2.9 ± 0.87	1.42–5.51
12	HDL cholesterol [mmol/L]	40	1.42 ± 0.4	0.77–2.45
13	Triglycerides [mmol/L]	40	1.42 ± 0.86	0.44–3,8
14	Systolic BP [mm Hg]	40	123.4 ± 12	100–150
15	Diastolic BP [mm Hg]	40	77.7 ± 11.8	60–100

BMI — body mass index; FM — fat mass; FMt — fat mass of the trunk; BP — blood pressure; LDL — low density lipoprotein cholesterol; HDL — high density lipoprotein cholesterol

sons between groups with normal distribution of the data were performed by unpaired Student's t-test, in other cases comparisons were performed by Kolmogorov-Smirnov test for two samples. For all analysis, a two-tailed $p \leq 0.05$ was considered to indicate statistic significance.

Results

Forty women participated in the study. Their mean age was 28.62 ± 7.6 years. Table I shows anthropometric data, body composition, blood pressure and biochemical estimations of the studied subjects. This cohort represented a relatively broad range of age. Four patients (10%) were overweight, 23 were obese (57.5%), and nine were considered to be morbidly obese. All of the obese subjects had increased abdominal fat. Three women were hypertensive, with increased diastolic blood pressure. They had not taken any hypotensive drugs before their recent diagnosis. In 14 women, hypercholesterolaemia and in 11 hypertriglyceridaemia were found.

Table II sets out the hormonal results of the studied women. Thirty-three subjects were hyperandrogenic and all except one were euthyrotic (one woman met the hormonal criteria of subclinical hyperthyroidism).

Table II. *Hormonal results of studied women with polycystic ovary syndrome*

Tabela II. *Wyniki badań hormonalnych u kobiet z zespołem policystycznych jajników*

No	Hormone	N	Mean ± SD	Range
1	Insulin [μIU/mL]	39	11.5 ± 9.6	1.0–33.0
2	HOMA	39	2.68 ± 2.4	0.19–9.86
3	Leptin [μg/mL]	36	34.4 ± 26.0	2.5–86.0
4	Testosterone [ng/mL]	40	0.82 ± 0.44	0.2–2.1
5	DHEA-S [ng/mL]	40	2862.7 ± 1286	518–6740
6	SHBG [nmol/L]	38	38.3 ± 23.6	9.8–115.0
7	LH/FSH	39	1.81 ± 1.8	0.97–11.1
8	Oestradiol [pg/mL]	39	58.2 ± 37.2	15–238
9	Prolactin [μg/mL]	39	15.3 ± 8.5	4.0–41.0
10	TSH [μIU/mL]	40	1.29 ± 0.7	0.012–3.31

HOMA — homeostatic model assessment; DHEA-S — dehydroepiandrosterone-sulphate; SHBG — sex hormone-binding globulin; LH — luteinising hormone; FSH — follicle stimulating hormone; TSH — thyroid stimulating hormone

Twelve had elevated fasting serum insulin levels, and 16 were considered as insulin resistant according to HOMA index.

Correlation between blood lipids, glucose metabolism markers, blood pressure and estimates of fatness in study subjects are presented in Table III. All indices of adiposity significantly positively correlated with all except total cholesterol metabolic parameters. In Figure 2, a significant positive correlation between fat mass of the trunk (kg) and HOMA index is shown.

Table IV presents correlation between estimates of adiposity and serum hormones. We found a significant positive correlation between fat estimates and leptin levels. FAI positively correlated with body weight and BMI. No other correlation between fat mass indices and sex hormones was found. Figure 3 presents a significant positive correlation between FAI and body mass index.

Discussion

In our study, we separated and measured abdominal and gynoid fat in patients with PCOS using the DEXA method. It turned out that in all obese subjects, abdominal fat was increased. Our data revealed a significant correlation between fatness and serum fasting glucose and insulin levels, as well as HOMA index of insulin resistance. We also observed a positive correlation between estimates of obesity and serum triglycerides and between obesity and blood pressure. In the end, we demonstrated direct positive correlations between free androgen index, body weight and BMI.

Table III. *Correlation between serum lipids, systolic and diastolic blood pressure, serum glucose and insulin levels and different estimates of fatness in our studied women with polycystic ovary syndrome. In the table, Pearson's correlation coefficients (rxy) are shown*

Tabela III. *Korelacja między lipidami krwi, skurczowym i rozkurczowym ciśnieniem tętniczym oraz wskaźnikami otyłości u badanych kobiet z zespołem policystycznych jajników. W tabeli przedstawiono wskaźniki korelacji Pearsona (x, y)*

	Body weight	BMI	Total fat	FMt	Abdominal fat	Gynoid fat
Total cholesterol	0.20	0.21	0.20	0.26	0.16	0.03
Triglycerides	0.62[c]	0.66[c]	0.59[c]	0.65[c]	0.40[a]	0.56[c]
Glucose	0.45[b]	0.48[b]	0.51[b]	0.40[a]	0.48[b]	0.40[a]
Insulin	0.69[c]	0.69[c]	0.65[c]	0.69[c]	0.35[a]	0.68[c]
HOMA	0.68[c]	0.68[c]	0.64[c]	0.68[c]	0.37[a]	0.65[c]
SBP	0.40[a]	0.40[a]	0.47[b]	0.48[b]	0.41[a]	0.52[b]
DBP	0.44[b]	0.43[b]	0.56[c]	0.57[c]	0.36[a]	0.65[c]

BMI — body mass index; FMt — fat mass of the trunk; HOMA — homeostatic model assessment; SBP — systolic blood pressure; DBP — diastolic blood pressure; [a]p < 0.05; [b]p < 0.01; [c]p < 0.001

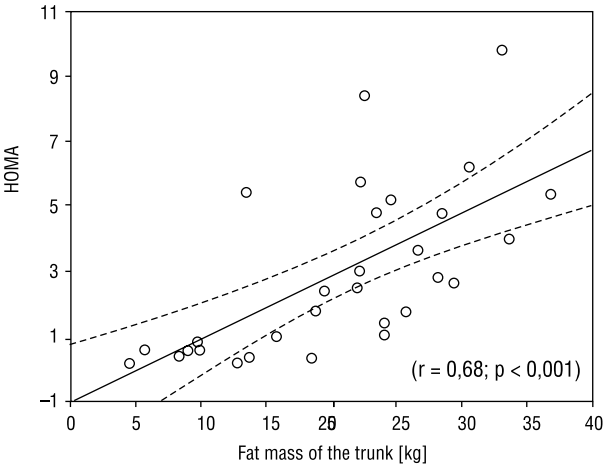

Figure 2. *Significant positive correlation between fat mass in trunk (kg) and HOMA index in studied women with polycystic ovary syndrome (r = 0.68; p < 0.001)*

Rycina 2. *Znamienna dodatnia korelacja między masą tłuszczu tułowia (kg) i wskaźnikiem HOMA u badanych kobiet z zespołem policystycznych jajników (r = 0,68; p < 0,001)*

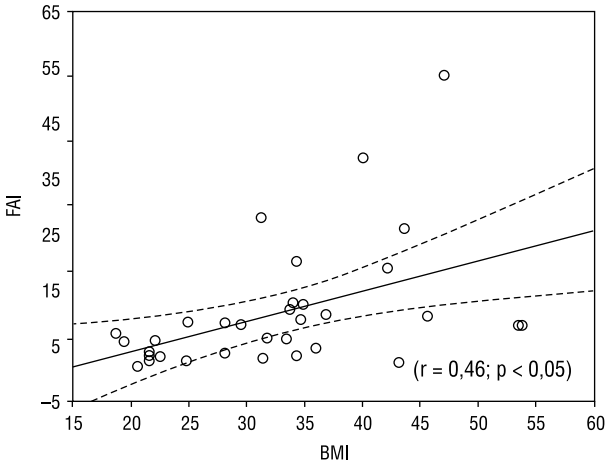

Figure 3. *Significant positive correlation between FAI and body mass index in women with polycystic ovary syndrome (r = 0.46; p < 0.05)*

Rycina 3. *Znamienna dodatnia korelacja między FAI a wskaźnikiem masy ciała u kobiet z zespołem policystycznych jajników (r = 0,46; p < 0,05)*

Table IV. *Correlation between serum hormones and different estimates of fatness in studied women with polycystic ovary syndrome. Table shows correlation coefficients (rxy)*

Tabela IV. *Korelacja między stężeniem hormonów w surowicy i wskaźnikami otyłości u badanych kobiet z zespołem policystycznych jajników. W tabeli przedstawiono wskaźniki korelacji (x, y)*

	Body weight	BMI	FM	FMt	Abdominal fat	Gynoid fat
Leptin	0.88[c]	0.87[c]	0.89[c]	0.80[c]	0.65[b]	0.89[c]
Testosterone	0.21	0.27	0.23	0.12	–0.06	0,28
DHEA-S	–0.18	–0.18	–0.12	0.02	–0.16	–0.12
FAI	0.43[b]	0.46[a]	0.33	0.39	0.13	0.39
LH/FSH	–0.30	–0.25	–0.21	–0.23	–0.35[a]	–0.21
Oestradiol	0.13	0.19	0.11	–0.22	–0.18	0.27

BMI — body mass index; FMt — fat mass of the trunk; DHEA-S — dehydroepiandrosterone sulphate; LH — luteinising hormone; FSH — follicle stimulating hormone; TSH — thyroid stimulating hormone; [a]p < 0.05; [b]p < 0.01; [c]p < 0.001

Diagnosing PCOS according to the Rotterdam criteria, we measured serum androgen levels. However, it has been demonstrated that also measuring salivary androgens may also be useful in making a diagnosis of this syndrome [12]. Moreover, Lewandowski et al. proved recently that gonadotrophin releasing hormone (GnRH) stimulated increase in LH/FSH ratio may be potentially useful as an additional tool in identifying patients with PCOS [13].

It has been previously demonstrated that approximately 40–60% of the women with PCOS are overweight or obese. In our study, this proportion was higher (67.5%). This may be the result of pre-recruitment to the study of the group of women with obesity and PCOS from our out-patients clinic.

Some authors consider that overweight women with PCOS have the central type of obesity. However, estimations of body composition have given contrasting results [14–16]. In our study, we used the dual energy X-ray absorptiometry method to accurately estimate abdominal and gynoid fat. Compared to anthropological measurements, DEXA is able to assess both total and regional fat mass in conditions of minimal exposure to radiation. We applied the technique used by Rissanen et al. [17] and Carmina et al. [18], who determined two regions of interest (ROI) including abdominal and gynoid fat respectively to obtain appropriate measurements. We found increase in abdominal fat in all of our overweight and obese patients. Moreover, highly significant correlations between all indices of obesity, triglycerides, glucose, insulin and blood pressure were observed in our subjects. This is an important observation because metabolic implications of abdominal fat excess are very well known. For example, it was recently demonstrated, that the 'hypertriglyceridaemic waist' phenotype (hypertriglyceridaemia and increased waist circumference) and metabolic syndrome in patients with type 2 diabetes, significantly increases cardiovascular risk [19]. On the other hand, only three of our patients were slightly hypertensive, 14 had hypercholesterolaemia and/ /or hypertriglycerydaemia, and 16 of them were insulin resistant, according to HOMA index. It could be argued that the young age of our subjects explains at least in part this relatively positive data. The question as to whether the presence of risk factors in patients with PCOS contributes to an early increase of cardiovascular events is still open, since follow-up studies have brought divergent results. In one long-term follow up, no increase in the risk of cardiovascular-related deaths during 30 years was observed, whereas in another trial, an excess of non-fatal cerebrovascular events in women with PCOS was demonstrated [20, 21]. Also data regarding the incidence of hypertension in women with PCOS is controversial. In one study, the authors showed that young women with PCOS generally do not manifest increased blood pressure [22]. However, another long-term follow up indicates that in such patients hypertension may develop later in life [23].

Our results suggest that increase in fat mass, particularly in the region of the abdomen, is related to and possibly responsible for insulin resistance and subsequent hyperinsulinaemia. One may speculate that not only abdominal obesity contributes to a decrease in insulin sensitivity in women with PCOS. It has been demonstrated in one of the recent studies that patients with this syndrome had higher insulin levels and reduced insulin sensitivity compared to controls without PCOS, regardless of the degree of fatness [24].

We found a significant correlation between free androgen index (FAI) and body weight and BMI. On the other hand, we couldn't demonstrate any direct correlation between serum androgens (total testosterone, androstendione (data unpublished) and dehydroepiandrosterone-sulphate) and obesity. It is difficult to explain this discrepancy, although the role of sex hormone binding proteins probably should be taken into account. We found relatively low SHBG levels. In this, our data confirms previous results that SHBG suppressed secondary to hyperandrogenaemia is an early finding in obese women with PCOS [25]. Holte et al. [26] similarly demonstrated a positive correlation between obesity and free androgen index. However, they also observed a correlation between obesity and testosterone and DHEA-S in premenopausal women with PCOS [27]. Carmina et al. [18] did not find any correlation between fat parameters and serum testosterone levels in PCOS patients. The apparent divergence between our findings and data from other authors may be explained at least in part by differences in age, obesity advance and androgens levels in the studied groups. However, our data corroborates the relationship between the degree of obesity and androgens levels. Moreover, there is growing evidence that androgen excess may be causally involved in abdominal fat accumulation in women [28, 29]. Hence, pharmacological or surgical amelioration of hyperandrogenaemia might improve the abdominal adiposity characteristic of PCOS and associated metabolic disorders [30, 31]. But our study was not designed to address this interesting problem.

There are some limitations to our study. Firstly, it suffers from the lack of an adequate control group of healthy women. Secondly, it includes a relatively small number of studied patients. Thirdly, it was not possible to differentiate visceral from subcutaneous fat analysing ROI with the DEXA method. In order to do that, computed tomography (CT) should be used, but the limitation of CT is a great exposure to ionising radiation. On the other hand, it has been proved that also subcutaneous

abdominal fat, especially its profound component, is metabolically active and contributes to insulin resistance and its subsequent metabolic and clinical consequences [32], so it seems that DEXA can be considered as a reliable method to identify and appreciate the risk associated with abdominal obesity.

Conclusions

We demonstrated using the DEXA method that overweight and obese women with PCOS have the abdominal type of obesity. We found high positive correlations between fatness and serum fasting glucose and insulin levels as well as with HOMA index. Estimates of obesity positively correlated with triglycerides and blood pressure. We also demonstrated direct positive correlations between free androgen index and body weight and BMI.

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