Streszczenie w języku angielskim

Polycystic ovary syndrome (PCOS) is the common endocrinopathy in women of reproductive age. The Rotterdam criteria for PCOS recognize four clinical phenotypes of the syndrome. Phenotype A, which meets all three current criteria for PCOS: clinical/biochemical hyperandrogenism, menstrual dysfunction and polycystic ovarian morphology (PCOM), phenotype B involving clinical/biochemical hyperandrogenism and menstrual dysfunction, phenotype C characterized by clinical/biochemical hyperandrogenism and PCOM, phenotype D in which menstrual dysfunction and PCOM are observed. In PCOS, apart from ovarian dysfunction, we can observe metabolic disorders such as abdominal obesity and insulin resistance. In state of insulin resistance, accompanied by hyperinsulinemia, insulin acts on the ovarian theca cells, leading to increased production of androgens. It has been reported that increased serum testosterone levels in PCOS women are associated with excess of visceral fat amount, which may lead to development of insulin resistance and more frequent occurrence of impaired glucose tolerance. Improper diet balance of patients with PCOS leads to abdominal obesity and insulin resistance, however there are conflicting data on which macronutrient in the diet (proteins, fats, carbohydrates) is connected the most with human obesity. Previous studies have suggested a link between weight gain and imbalance in leptin and ghrelin concentrations in women with PCOS. Leptin reduces appetite and increases energy expanditure, while ghrelin increases appetite.

The aim of the study was to assess the concentrations of androgens, leptin and ghrelin in the blood of patients with polycystic ovary syndrome in correlation with anthropometric parameters and diet.

Material and Methods:

The first study group consisted of 146 women: 89 patients with PCOS divided into four phenotypes according to Rotterdam criteria (phenotype A, B, C, D) and 57 control women. In this group serum concentrations of total testosterone androstenedione and DHEA-S were measured with radioimmunoassay (DIAsource ImmunoAssays S.A., Belgium), whereas serum concentrations of serum sex hormone–binding globulin (SHBG) were assessed with immunoradiometric method (ZenTech, Angleur, Belgium). In all women the insulin resistance index (Homeostasis Model Assessment of Insulin Resistance, HOMA-IR) was calculated, the free androgen index (FAI) was calculated and the dual-energy X-ray absorptiometry (DXA)

analysis was performed, estimating the weight visceral adipose tissue (VAT) and gynoid adipose tissue, and then the android (A) to gynoid (G) ratio (A/G ratio) was calculated.

In the second group of 73 women: 39 subjects with PCOS and 34 healthy women completed a consecutive three-day dietary diary to identify the macronutrient and micronutrient intake. Subsequently, using Diet 5.0 program the content of daily nutrients intake in g/day (proteins, fats, carbohydrates) were calculated and the concentration of leptin in the blood plasma was determined by the immunoenzymatic method (Human Leptin ELISA, BioVendor, Brno, Czech Republic), while the concentration of total ghrelin was determined by the radioimmunometric method (GHRT-89HK, RIA, Millipore, USA).

Results:

In phenotypes A, B and C, we observed higher FAI in comparison to the control group (all $p < 0.01$), as well as in phenotype A in comparison to phenotype D ($p < 0.01$). Only in phenotype A we found higher visceral adipose tissue (VAT) mass and android/gynoid ratio (A/G ratio) in comparison to the control group (all $p < 0.01$). We found a positive correlations of HOMA-IR with VAT and A/G ratio in all phenotypes (all $p < 0.05$), moreover we found relationships between FAI and HOMA-IR in phenotype A ($r = 0.40$, $p = 0.01$), phenotype B ($r = 0.47$, $p = 0.03$) and phenotype C ($r = 0.66$, $p < 0.01$). Accordingly, A/G ratio was related with FAI in all phenotypes (all $p < 0.05$).

In the PCOS group, the plasma leptin concentration correlated positively, and ghrelin plsma concentration correlated negatively with the dietary fat content ($r = 0.36$, $p = 0.02$; $r = -0.37$, $p = 0.02$). Accordingly, in the PCOS group we found a positive association of HOMA-IR with serum leptin levels ($r = 0.5$, $p < 0.01$) and a negative relationship with the serum ghrelin concentration ($r = -0.4$, $p = 0.03$). Moreover, we found a positive relationship between HOMA-IR and total dietary fat ($r = 0.38$, $p = 0.03$).

Conclusions:

1. Due to the greater amount of visceral adipose tissue in the A PCOS phenotype, patients presenting this phenotype have a greater risk of developing metabolic disorders compared to the control group.
2. The amount of visceral adipose tissue is associated with insulin resistance and serum concentration of androgens in both the normoandrogenic and hyperandrogenic phenotypes of PCOS.

3. A high-fat diet in patients with PCOS is associated with the development of insulin resistance, disturbances in the balance between the concentration of leptin and ghrelin in the plasma, leading to the development of obesity.