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PHYSICAL ACTIVITY IN PATIENTS WITH POLYCYSTIC OVARY SYNDROME AND OBESITY

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Introduction. Polycystic ovary syndrome and obesity are probably the most frequent endocrine disorders among women of fertile age, affecting approximately 20% of the female population. Polycystic ovary syndrome is characterized by elevated ovarian production of androgens, disturbed ovulation and ultrasound findings of polycystic ovaries. Although the aetiology of polycystic ovary syndrome remains largely unknown, there are strong indications of a genetic predisposition.

Hyperandrogenism and insulin resistance, the characteristic endocrine features of the pathogenesis of polycystic ovary syndrome, explain the various associated symptoms. The primary abnormality appears to be increased production of androgens by the ovaries, augmented by disrupted feedback control of pulsatile gonadotropin releasing hormone secretion, resulting in elevated secretion of luteinizing hormone and relative follicle-stimulating hormone deficiency. The clinical consequences are the characteristic polycystic ovarian morphology and anovulation leading to menstrual disorders and reduced fertility, as well as hirsutism and acne. In addition, women with polycystic ovary syndrome are more insulin resistant, independent of obesity, leading to secondary hypersecretion of insulin, which directly stimulates androgen production by the ovarian theca cells. Furthermore, insulin inhibits hepatic synthesis of sex hormone-binding globulin, thereby elevating levels of free and bioavailable testosterone. Moreover, insulin resistance may lead to metabolic changes including abdominal obesity.

This condition is usually managed by treating the symptoms, including menstrual disorders, infertility, hirsutism and overweight/obesity, although specific treatment is not always necessary. However, a healthy lifestyle, including regular physical activity, is clearly beneficial and should therefore be recommended first.

The aim of the study. The effective method of polycystic ovary syndrome treatment is lifestyle modification. The aim of this study was to determine the effect of a twelve-week aerobic exercise on ovarian androgens and body composition of women with polycystic ovary syndrome.

Material and methods. In this clinical study, 20 obese patients (body mass index > 25 kg/m^2) and 20 lean patients (body mass index < 20 kg/m^2) with polycystic ovary syndrome were selected. Lean and obese patients were randomly divided into intervention and control groups. The intervention group participated in a 12 week, 3 sessions per week or at least 150 minutes per week with intensity 65-80% of maximal heart rate for 25-30 minutes of aerobic exercise, while this intervention was not performed in the control group. Body mass index, waist-hip ratio, luteinizing hormone, follicle-stimulating hormone, prolactin and testosterone hormones and free androgen index were measured before and after the intervention. The data were analyzed using the paired and independent T-tests.

Results. Body mass index decreased significantly after exercise in obese group ($P<0,0001$). The reduction of prolactin was not significant after exercise in both lean and obese groups ($P>0,05$). Luteinizing hormone/follicle-stimulating hormone ratio significantly decreased after exercise in the lean group ($P=0,049$).The testosterone concentration was significantly decreased after exercise in both lean and obese groups ($P<0,05$). But our observations indicate that mild forms of hyperandrogenism by polycystic ovary syndrome may improve physical activity and performance. Serum levels of testosterone correlated positively with increased muscle strength in the polycystic ovary syndrome group. Androgens enhance physical performance through their effects on muscle tissue, bone mass, erythropoietin, the immune system and behavioral patterns.

Conclusions. A period of exercise with body weight loss and reduction of body mass index can contribute to the decline of testosterone concentration, decrease of luteinizing hormone/follicle-stimulating hormone ratio, improvement of metabolic condition. It prevents from the increasing of prolactin and hyperandrogenism side effects.

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PECULIARITIES OF RENAL MECHANISMS OF CARBOHYDRATE STATUS REGULATION IN THE EARLY PERIOD OF EXPERIMENTAL DIABETES WITH UNDERLYING PHARMACOLOGICAL BLOCKADE OF RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM

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Introduction. Based on the pathophysiology of renin-angiotensin-aldosterone system (RAAS), its multifactorial influence on the renal functions obviously requires a detailed study at different stages of nephropathy progression in order to identify pathological factors that are directly promoted by RAAS, and the processes that are hemodynamic consequences of its influence. Specific attention is attracted to the investigation of RAAS involvement in the regulation of renal mechanisms of glucose transport in case of diabetic kidney.

The aim of the study. To explore peculiarities of renal mechanisms of carbohydrate status regulation in the early period of alloxan-induced experimental diabetes mellitus (EDM) with underlying pharmacological blockade of RAAS.

Material and methods. The experiments were carried out on 16 white non-linear mature male rats with alloxan-induced EDM; 10 intact animals served as a control group.On 11^{th} day after the induction of EDM kaptopril was administered intraperitoneally to 8 diabetic rats for the pharmacological blockade of RAAS, all the rats were withdrawn from the experiment. The concentration of glucose in the blood plasma and urine, its excretion, filtration load, reabsorption, clearance, tubular transport were studied under conditions of water 2-hour diuresis.

Results. On the 11^{th} day of the experiment, after pharmacological blockade of the intrarenal RAAS, typical diabetic changes in the carbohydrate status persisted in the experimental animals: alloxan-induced hyperglycemia reached the level 2,9 times higher than the control one, 1,4 time succeeding the rate of rats with 11-day EDM without captopril administration. The level of glucosuria after kaptopril administration was found to be significantly higher as well (by 1,7 times) than the corresponding parameter of diabetic rats without pharmacological blockade of RAAS, and the excretion of glucose raised by 2,5 times, including the one standardized in volume of glomerular filtrate (1,7 times). The filtration charge of glucose in alloxan-diabetic rats under kaptopril action exceeded the control level by 6,3 times and was found to be twice higher than the corresponding index of alloxan-diabetic rats without RAAS blockade, consequently causing an intensification of the absolute proximal tubular transport of glucose (by 5,9 times as compared to the group of intact animals and by 2 times – to the group of diabetic rats without RAAS blockade). A significant decline in the relative glucose reabsorption (by 6,8% in comparison with the level control group, as well as by 1,9% – with the corresponding index of diabetic rats without RAAS blockade) was probably caused by the overload of the proximal tubule by filtrated glucose. Glucose clearance under the condition of pharmacological blockade of RAAS was 64 times higher than that of the control and by 1,9 times exceeded its level in alloxan-diabetic rats without RAAS blockade. The concentration index of glucose in alloxan-diabetic rats after kaptopril administration was 36,1 times higher than the corresponding parameter of intact animals and 1,3 times higher than that of alloxan-diabetic rats without RAAS blockade.

Conclusions. The intensification of renal glucose transport during 11-day experimental diabetes mellitus with underlying pharmacological blockade of renin-angiotensin-aldosterone system is stipulated not only by hyperglycemia and «above-threshold» glucose in the ultrafiltrate, but also by an intensification of the hyperfiltration phenomenon and, as a result, the functional inability of the proximal tubules to adequate glucose reabsorbtion. The further influence of