Introduction. Hyperglycemia is a condition in which an excessive amount of glucose circulates in the blood plasma and is a common adverse reaction of anabolic steroids therapy, affecting 20% to 50% of patients without a history of diabetes. In addition, glucose levels are often elevated among patients with prediabetes and previously well-controlled diabetes during steroid therapy. Anabolics stimulate glucose production by the liver and inhibit peripheral glucose uptake, resulting in insulin resistance allowing blood glucose levels to rise and remain higher.

Aim of the study. To determine the manifestations of hyperglycemia after utilization of anabolics.

Materials and methods. It was made the bibliographic and personal investigations of hyperglycemic state due to anabolics. Twenty-five healthy male power athletes were followed up during their self-regimen of substance abuse.

Results. In our investigation, there is determined that more than half of the men receiving high-dose steroids develop hyperglycemia, with an incidence of 86% of at least one episode of hyperglycemia and 41% of athletes presenting a mean blood glucose ≥ 140 mg/dL Hyperglycemia incidence in men without a prior history of diabetes mellitus (DM) to steroid use varies from 34.3% to 56% for athletes with 1-3 years of anabolic utilization. The manifestations of hyperglycemia were: polyuria (36%), polydipsia (29%), polyphagia (41%), dizziness (18%), shakiness (19%), irritability or moodiness (37%), anxiety or nervousness (26%), trouble concentration (15%). The development of hyperglycemia was observed on 41% athletes, 29 - 41 years old, who reported a consumption of AAS for 1-3 years. They self-administered high doses of oral stanozolol, oxymetholone, methandrostenolone and ibutamoren. For management of hyperglycemia, if diet and physical exercise do not reduce the glucose levels adequately, it is recommended to prescribe antidiabetic drugs, such as metformin, DPP-4 inhibitors or sulfonylureas that are effective and work by increasing insulin release from the pancreas but they may cause hypoglycemia.

Conclusions. Complications associated with steroid-induced hyperglycemia are often underestimated despite hyperglycemia being a well-known adverse effect of anabolic therapy. Appropriate management of hyperglycemia due to anabolics is oral antidiabetic agent, such as a DPP-4 inhibitors, metformin, or by using the weight-based NPH insulin may reduce the risk of adverse outcomes, including symptomatic hyperglycemia and new-onset diabetes.

Key words: Steroid, Anabolic, Hyperglycemia, Treatment.

DEPARTMENT OF MOLECULAR BIOLOGY AND HUMAN GENETICS

315. CLINICAL AND GENETIC STUDY OF THROMBOPHILIA IN PREGNANCY

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Introduction. Thrombophilia is defined as an abnormal coagulation state of blood that increases the risk of thrombosis. Pregnancy represents a physiological hypercoagulation state. But, women with acquired and hereditary thrombophilia are at increased risk of developing

venous thromboembolism and other associated gestational vascular complications like Recurrent Pregnancy Loss (RPL), preeclampsia, intrauterine growth restriction, and placental abruption during pregnancy. These complications are a major cause of maternal and fetal morbidity and mortality.

Aim of the study. This study focuses on the women who reported RPL, without any positive pregnancy and the identification of genetic factors that lead to the formation of thrombosis (F2 G20210A, F5 G1691A, MTHFR C677T, MTHFR A1289C, MTR A2756G, MTRR A66G), involved in fibrinolysis (PAI-1 4G/5G) and their association with primary female infertility.

Materials and methods. Research design was constructed as case-control type. The case group was represented by 44 patients with RPL, without any positive pregnancy, with normal karyotype, and lack of other causes (intrauterine infections, uterine pathology) responsible for the RPL. The control group included 57 patients with 2 positive pregnancies who did not receive anticoagulant treatment. The Odds Ratio (OR) was calculated for the case group and control group, at a 95% confidence interval, and p values <0,005 were considered statistically significant. OR>1 demonstrate a strong association between mutation and RPL, OR<1 show a weak association.

Results. We found that G1691A mutation in F5 gene encoding factor V (Leiden) (for heterozygous genotype OR=8,84; 95% CI; 1,02-76,42; p<0,05) and mutation G20210A in gene F2 encoding factor II (prothrombin), (for heterozygous genotype OR=7,18; 95% CI; 0,81-63,87; p<0,05), are major risk factors for RPL and primary female infertility. Carriers of the homozygous genotype after mutant allele were not determined in either group. The 4G/5G polymorphism of the PAI-1 gene, in this study was not associated with RPL and primary female infertility. Analysis of genes involved in folate cycle as MTHFR C677T mutation (OR=3,33; 95% CI; 1,37-8,09; p<0,05 for the heterozygous genotype and OR=3,73; 95% CI; 0,99-14,05; p<0,05 for the homozygous genotype after the mutant allele), MTR mutation A2756G (for the heterozygous genotype OR=2,91; 95%CI; 1,19-7,08; p<0,05 and for the homozygous genotype after the mutant allele OR=6,30; 95%CI; 1,17-34,03; p<0,05), MTRR mutation A66G (for the heterozygous genotype OR=2,40; 95%CI; 1,02-5,62; p<0,05 and for the homozygous genotype after the mutant allele OR=5,77; 95%CI; 0,99-33,68; p<0,05),demonstrated that these polymorphisms are major risk factors of RPL and primary female infertility. A1289C mutation of the MTHFR gene was not associated with RPL and primary female infertility.

Conclusion. According to the results of the study, it is recommended the genetic diagnosis of all patients with RPL, without organic or infectious causes, for detection of the genetic factors involved in hereditary thrombophilia.

Key words: hereditary thrombophilia, recurrent pregnancy loss, primary female infertility

316. CLINICAL AND CYTOGENETIC VARIATIONS IN MALE INFERTILITY CAUSED BY KLINEFELTER SYNDROME

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Introduction. Klinefelter's syndrome (KS) is the most common genetic cause of human male infertility characterized by gynecomastia, hypogonadism and azoospermia. About 80–90% of patients with Klinefelter's syndrome have an homogenous 47,XXY karyotype, the classic form of Klinefelter's syndrome. The prevalence of Klinefelter's syndrome is 1 in 700 men. Many patients with Klinefelter syndrome remain undiagnosed due to clinical variations.