МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»

М А Т Е Р І А Л И

105-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького персоналу
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Матеріали підсумкової 105-ї науково-практичної конференції з міжнародною участю професорсько-викладацького персоналу Буковинського державного медичного університету, присвяченої 80-річчю БДМУ (м. Чернівці, 05, 07, 12 лютого 2024 р.) – Чернівці: Медуніверситет, 2024. – 477 с. іл.

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У збірнику представлені матеріали 105-ї підсумкової науково-практичної конференції з міжнародною участю професорсько-викладацького персоналу Буковинського державного медичного університету, присвяченої 80-річчю БДМУ (м. Чернівці, 05, 07, 12 лютого 2024 р.) із стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

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hyperproduction of androgens in the ovaries, which are also transformed into estrone in peripheral tissues, which stimulates the excretion of LH. The vicious circle closes and hormonal disorders develop, which accompany PCOS, i.e. hyperandrogenism. Violations at any level of the hormonal homeostasis regulation system lead to one result – absolute or relative hyperestrogenization of a woman's body. And hyperestrogenization is the main factor that causes dyshormonal hyperplasia of the mammary glands. In addition, estrogens have a prolactin-stimulating effect, and prolactin increases the number of estradiol receptors in the tissue of the mammary glands, which contributes to the development of proliferative processes, the formation of dyshormonal proliferates and carcinomas in the mammary glands.

Conclusions. Thus, reducing body weight is the first and the most important stage of treatment for this comorbid pathology. It should be alsonoted that a decrease in body weight has a positive effect on metabolic associated fatty liver disease, which in turn reduces hyperestrogenemia, since estrogens are metabolized in the liver and the protein-synthesizing function of the liver is restored, that is, the concentration of sex globulin binding hormones increases and the amount of active androgens and estrogens decreases.

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CHARACTERISTICS OF URINARY SODIUM EXCRETION OF RATS IN THE DYNAMICS OF EXPERIMENTAL DIABETES MELLITUS DEVELOPMENT

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Introduction. Tubulopathies with electrolyte disbalance require a detailed study in the dynamics of diabetic renopathy progression as they may lead to the changes in local hemodynamics in the kidneys, deterioration of nephron function and, subsequently, to structural changes in the kidney tissue, progressive impairment of kidney function.

The aim of the research was to study the peculiarities of urinary sodium excretion in the dynamics of alloxan-induced experimental diabetes mellitus.

Material and methods. The experiments were carried out on 63 white non-linear mature male rats, 53 with experimental diabetes mellitus (EDM) of varying duration induced by intraperitoneal administration of alloxan in a dose of 160 mg/kg of body weight, 10 intact rats served as the control group. 10, 20, 25, 30, 40 and 45 days after its administration, the animals were withdrawn from the experiment. Under condition of water induced 2-hour diuresis, sodium content in the urine, its excretion, absolute and relative reabsorption, proximal and distal tubular transport (including standardized by glomerular filtrate (GF) volume), were determined.

Results. On the 11^{th} day of the experiment, the maximal urine loss of sodium was observed for the entire duration of the experiment: the urinary concentration of sodium ions in animals of this group exceeded the control index by 5,9 times, sodium excretion increased by 6,5 times, including standardized by GF (by 4,7 times).

Sodium excretion including that standardized by the volume of GF, by the 21st day of the experiment 4,8 and 3 times decreased respectively, accompanied by a significant reduction of sodium urine level (by 2,2 times). However, the latter was found to be 2,7 times higher than that of the control level, and sodium excretion – absolute and standardized one – was 1,3 and 1,5 times higher, respectively.

On the 26^{th} day of the experiment the absolute sodium urine excretion 1,3 times decreased as compared to that index on the 21^{st} day of diabetes, practically reaching the control level. Cation excretion standardized by GF volume almost six-fold decreased in comparison to the index of 21-day diabetes and by 3,8 times – as compared to the control.

On 31^{st} and 41^{st} day of the experiment absolute sodium excretion raised by 3,1 and 2,5 times in comparison with the corresponding values on the 26^{th} day of the experiment and standardized sodium excretion – by 2,9 and 3,1 times correspondingly. It exceeded the level of electrolyte excretion in control rats by 3,3 and 2,7 times respectively. However, remaining 24,6% and 19,7% less than the level of control as for the standardized by the volume of GF sodium excretion. The

urinary sodium concentration increased markedly – by 4,1 times on the 31st day of the experiment and by 3,7 times in its 41-day duration.

Sodium urine loss on the 46th day of alloxan-induced EDM was found to be markedly reduced as compared to the level of 41-day alloxan diabetes, still slightly exceeding the control level. Both absolute and standardized sodium excretion continued to reduce, remaining 74,6% and 45,9% higher than the values of animals of the control group respectively.

Conclusions. Ionoregulatory function of the kidneys in rats with alloxan-induced experimental diabetes is characterized by the intensification of natriuresis at all stages of the experiment. The increase in urinary sodium loss in the early stages of alloxan-induced experimental diabetes is primarily stipulated by glomerular hyperfiltration, followed by an enhancement of filtration sodium load to the nephron. The loss of proportionality between the filtered amount of sodium and its proximal reabsorption causes a decrease in the total reabsorption potential of the tubular segment of the nephron in the dynamics of alloxan-induced experimental diabetes. It is reflected primarily on the proximal tubules, and subsequently induces a functional weakening of the tubule-tubular connection and relative dysfunction of the distal segment of the nephron with subsequent inhibition of aldosterone-dependent regulatory mechanisms.

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THE ROLE OF VITAMIN D DEFICIENCY IN THE PATHOGENESIS OF ENDOCRINE AUTOIMMUNE DISEASES

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Introduction. In recent years, a large amount of new data has emerged on the role of vitamin D not only in the regulation of phosphorus-calcium metabolism, but also in the development of autoimmune diseases. One of the most common combinations of autoimmune diseases in the practice of an endocrinologist is the combination of type 1 diabetes mellitus with autoimmune thyroiditis.

The aim of the study. In this study, we evaluated 25-hydroxyvitamin D (25(OH)D) levels in patients with type 1 diabetes mellitus without concomitant autoimmune pathology and in patients with type 1 diabetes mellitus with autoimmune thyroiditis.

Material and methods. 28 patients with type 1 diabetes mellitus (18 women and 10 men), mean age 32 + 2.4 years, were registered at the regional endocrinology center. All patients were tested for 25(OH)D, calcium, thyroid-stimulating hormone (TSH), anti-thyroid peroxidase (anti-TPO), anti-thyroglobulin (anti-Tg), and pancreatic antibodies, namely glutamate decarboxylase (GAD), to confirm the autoimmune genesis of diabetes. The control group consisted of 20 healthy individuals without established autoimmune diseases.

Results. In the group of patients with type 1 diabetes mellitus, the value of 25(OH)D was below 14.6 + 1.8 mg/l compared to the control group (30.2 + 3.4 mg/l). In 10 out of 28 patients with type 1 diabetes mellitus, the titer of antibodies to anti-TPO and anti-Tg was elevated. 5 of them were treated with euthyrox at the time of the examination and two were diagnosed with subclinical hypothyroidism. Patients with type 1 diabetes mellitus with elevated titers of anti-TPO and anti-Tg had lower 25(OH)D values compared to patients with normal titers of anti-TPO and anti-Tg 13.6 (6.8 + 22.1 mg/mL) vs. 20.2 (11.1 + 26.7 mg/mL), respectively.

Conclusions. The study data showed a more significant decrease in 25(OH)D levels in patients with type 1 diabetes mellitus in combination with a positive titer of thyroid antibodies. These results may indicate the impact of vitamin D deficiency on the development of nonspecific autoimmune aggression directed against several organs and systems.