МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ

М А Т Е Р І А Л И

106-ї підсумкової науково-практичної конференції
з міжнародною участю
професорсько-викладацького колективу
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Матеріали підсумкової 106-ї науково-практичної конференції з міжнародною участю професорсько-викладацького колективу Буковинського державного медичного університету (м. Чернівці, 03, 05, 10 лютого 2025 р.) – Чернівці: Медуніверситет, 2025. – 450 с. іл.

У збірнику представлені матеріали 106-ї науково-практичної конференції з міжнародною участю професорсько-викладацького колективу Буковинського державного медичного університету (м. Чернівці, 03, 05, 10 лютого 2025 р.) зі стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

Загальна редакція: професор Геруш І.В., професорка Годованець О.І., професор Безрук В.В.

Наукові рецензенти:
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професорка Хухліна О.С.
професор Чорноус В.О.

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the activity of the kidneys as the main efferent link of the regulation of water-salt metabolism. As a target organ and the site of degradation of most calcitropic factors, the kidneys significantly affect calcium homeostasis and vitamin D metabolism, and renal disorders are undoubtedly considered a risk factor for the development of secondary osteoporosis. Since diabetic nephropathy is one of the leading causes of renal failure among the numerous chronic complications that constantly develop against the background of DM, regardless of its type and duration, it is reasonable to clarify the role of renal dysfunction in the development of mineral metabolism disorders in case of DM.

The aim of the study. The research is aimed at exploration of the peculiarities of calcium and phosphates excretion in the dynamics of experimental diabetes mellitus development.

Material and methods. The experiments were carried out on 54 white non-linear mature male rats – 24 animals with 11-, 26- and 46-day long alloxan-induced experimental diabetes mellitus (EDM), induced by single intraperitoneal administration of alloxan in the dose of 160 mg/kg of body weight, and 30 intact animals of the control. After assessment of water-induced 2-hour diuresis (in ml/100 g of body weight for 2 hours), urine and plasma creatinine concentration were determined, GFR was calculated based on endogenous creatinine clearance. The calcium urine content was detected by the intensity of coloration in the presence of o-cresolphthalein complexone, the level of phosphates in urine – by photometry of the phosphoromolybdate complex. The calculation of electrolyte excretion was carried out. The data obtained were statistically processed with determination of the mean value and standard errors, the non-parametric Mann-Whitney rank test was used to assess the probability of difference between the studied groups.

Results. It was shown, that urinary calcium and phosphorus concentration did not undergo significant changes at the initial stage of diabetic renopathy development in comparison with the corresponding indices of intact rats, demonstrating a tendency to decrease – by 8,7 ($p>0,3$) and 6,5% ($p>0,6$) as to urinary calcium and phosphorus concentration respectively. The excretion of calcium and phosphorus increased unreliably – by 7,3% ($p>0,6$) and 10,7% ($p>0,5$), respectively – on 11th day of EDM.

On the 26th day after administration of the diabetogenic substance, the calciuric response of the kidneys of rats reached statistically reliable values (the calcium content in the urine of animals of this group exceeded that of intact animals by 12,0% ($p=0,05$), and its excretion – by 24,6% ($p<0,01$)) and was accompanied by a significant intensification of phosphates excretion (the urine concentration of phosphorus of animals with 26-day EDM exceeded the control level by 88,0% ($p<0,001$), phosphates excretion – by 2,1 times ($p<0,001$)).

The trends established on the 26th day of alloxan-induced EDM persisted hereinafter – calcium and phosphorus ions were detected in the urine of alloxan-diabetic rats on the 46th day of the experiment in the amounts significantly exceeding the control values, in particular, by 1,2 ($p=0,05$) and 2,1 times ($p<0,001$), respectively. The excretory fractions of these ions increased significantly as well: calcium excretion exceeded the corresponding index in the group of intact animals by 27,2% ($p<0,05$), phosphate excretion – by 2,3 times ($p<0,001$).

Conclusions. The development of calcium-phosphorus homeostasis disturbances in the dynamics of experimental diabetes is a consequence not only of an imbalance of calcitropic factors, but kidney dysfunction as well in response to metabolic processes induced by hyperglycemia. Transtubular transport of calcium and phosphates in case of alloxan-induced experimental diabetes mellitus is characterized by changes of the intensity of tubule-specific reabsorption of cations and depends on the duration of the experiment.

Pashkovska N.V.

PROSPECTS FOR THE TREATMENT OF AUTOIMMUNE DIABETES

Department of Clinical Immunology, Allergology and Endocrinology
Bukovinian State Medical University

Introduction. Autoimmune Type 1 Diabetes (T1D) is a chronic endocrine disorder that arises due to autoimmune destruction of pancreatic beta cells. With the global rise in incidence and

challenges in disease management, the relevance of innovative treatment methods for T1D is continually increasing.

The aim of the study. To analyze current trends and prospects in the treatment of autoimmune diabetes, including immunomodulatory therapy, cellular technologies, artificial insulin regulation systems, gene therapy, and individualized treatment approaches, based on a comprehensive review of literature data.

Material and Methods. Current data on prospects for autoimmune diabetes treatment were analyzed by conducting searches on Google Scholar, Scopus, Web of Science, PubMed Medline, and Embase for relevant scientific research.

Results. The analysis revealed a range of promising prospective treatment methods for autoimmune diabetes, whose efficacy and safety require further investigation.

Immunomodulatory Therapy. The primary pathogenic mechanism of T1D is related to an autoimmune response targeting pancreatic beta cells. Modern immunomodulatory drugs can modify the autoimmune response by reducing the activity of autoaggressive T-cells. Immunomodulators, such as anti-CD3 antibodies, immunosuppressive agents, or antigen-specific vaccines, offer selective inhibition of the pathological immune response without suppressing overall immunity.

Stem Cell Therapy. Stem cell therapy is one of the most promising approaches for restoring the beta-cell population. Using pluripotent stem cells to induce differentiation into beta cells offers the possibility of creating an endogenous source of insulin-producing cells. However, this method requires further refinement, particularly in the areas of cell differentiation control and preventing recurrent autoimmune attacks.

Islet Transplantation. New technologies, such as islet cell encapsulation, aim to protect transplanted cells from the immune system, minimizing rejection risks without aggressive immunosuppression.

Artificial Pancreas and Advanced Insulin Pumps. Systems that consist of insulin pumps, glucose sensors, and specialized software can adaptively regulate insulin levels in real-time. Such systems significantly reduce the risks of hypo- and hyperglycemia, improve glycemic control, and lower the frequency of complications.

Gene Therapy and Genetic Research. Genetic approaches to T1D treatment include genetic modification of immune cells or pancreatic cells, allowing the blocking of autoimmune responses. Gene therapy, especially CRISPR-Cas9 gene editing, aims to reduce immune system aggression against beta cells.

Preventive Vaccines. This approach involves introducing beta-cell antigens to induce immune tolerance toward the body's own pancreatic cells. Although still in the experimental phase, creating a vaccine could be a significant step in preventing T1D among individuals with a high genetic risk.

Personalized Medicine. Genetic analysis and autoantibody monitoring enable the development of individualized therapeutic approaches, enhancing treatment effectiveness.

Conclusions. Modern research reveals new possibilities for improving T1D control, including immunomodulation, cellular and genetic technologies, artificial insulin systems, and personalized medicine. However, these methods require further investigation to ensure maximum efficacy and safety.

Pavlovych L.B.

DIAGNOSIS OF HYPOTHYROIDISM IN PATIENTS WITH HYPERPROLACTINEMIA

Department of Clinical Immunology, Allergology and Endocrinology
Bukovinian State Medical University

Introduction. Diagnosis of hypothyroidism is difficult due to the variety of symptoms, and the severity of thyroid insufficiency can vary from patient to patient. In 30% of patients with hypothyroidism, hyperprolactinemia develops, which is associated with increased secretion of thyrotropin-releasing hormone by the hypothalamus, which in turn stimulates the production of TSH and prolactin in the adenohypophysis.