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.

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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.

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DIAGNOSIS OF HYPOTHYROIDISM IN PATIENTS WITH HYPERPROLACTINEMIA

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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.

The aim of the study. Diagnosis of hypothyroidism in patients with hyperprolactinemia.

Material and methods. Fifteen men aged 40 to 49 years were examined. The following were determined: thyroid-stimulating hormone (TSH), free thyroxine (T4 free), prolactin, total and free testosterone. MRI of the head was performed.

Results. The study was conducted in the presence of complaints of decreased potency and libido and infertility. Thyroid-stimulating hormone (TSH) - 5.8 ± 1.2 μIU/ml (N 0.27 - 4.2). Thyroxine free (T4 free) - 0.52 ± 0.11 mg/dL (N 0.70 - 1.48). Prolactin - 480 ± 0, 42 μIU/ml (N 86 - 324). Testosterone total - 1.80 ± 0.14 mg/ml (N 20 - 49 years 2.49 - 8.36). Testosterone free - 0.74 ± 0.12 pg/ml (N 19 - 55 years 1. 0 - 28.28). Due to elevated prolactin levels, MRI of the head was performed to exclude prolactinoma.

All patients had no changes on head MRI. Patients were prescribed Euthyrox 50 mcg, 1 tablet in the morning, on an empty stomach. Two months later. Thyroid-stimulating hormone (TSH) - 3.2 ± 0.4 μIU/ml (N 0.27 - 4.2). Thyroxine free (T4 free) - 0.82 ± 0.24 mg/dL (N 0.70 - 1.48). Prolactin - 280 ± 0, 86 μIU/ml (N 86 - 324). Testosterone total - 2.92 ± 0.22 mg/ml (N 20 - 49 years 2.49 - 8.36). Testosterone free - 1.46 ± 0.11 pg/ml (N 19 - 55 years 1. 0 - 28.28)

Patients' potency and libido normalized.

Conclusions. Hypogonadism in the setting of hypothyroidism can be caused by both hyperprolactinemia and direct gonadal dysfunction, as thyroid hormones affect the level of sex hormones and the male reproductive system in general. The importance of the earliest possible diagnosis of hypothyroidism is due to the fact that the administration of thyroid replacement therapy to patients will contribute to the positive dynamics of the pathological process in almost any organ or organ system.

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THE ROLE OF DYSLIPIDEMIA IN THE DEVELOPMENT OF COGNITIVE DISORDERS IN LATENT AUTOIMMUNE DIABETES IN ADULTS

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Introduction. LADA is a heterogeneous type of diabetes (DM) that combines features of type 1 diabetes mellitus (T1DM) and type 2 diabetes (T2DM) and according to the modern classification belongs to T1DM group. Diabetic dyslipidemia significantly increases the risk of the development and progression of cardiovascular and neurological pathology. Patients with DM and dyslipidemia have a higher incidence of vascular dementia and cognitive impairment, early detection of which is important for DM management.

The purpose of the study. To determine the characteristics of lipid metabolism in patients with LADA and its phenotypes depending on the degree of cognitive impairment.

Research material and methods. 34 patients with LADA (18 – LADA1 and 16 – LADA2) and 20 practically healthy individuals were examined. All patients underwent a general clinical examination, determination of indicators of carbohydrate metabolism. Psychodiagnostic testing included the Montreal Cognitive Assessment Test (MoCA Test). To assess the degree of dyslipidemia, lipidogram data (total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), atherogenity index (AI)) were analyzed.

Research results. Normal cognitive function was found in 56% of patients with LADA, moderate cognitive impairment – in 35% and dementia in 9% of patients. In LADA1 phenotype 61% of patients had normal indicators, 33% – moderate impairment and 6% - dementia, while in LADA2 group in 50% of patients there were not any cognitive problems detected, 37% had moderate cognitive impairment and 13% – dementia.

In the LADA group, TC increased by 84.8% relative to the control group ($p<0.001$). Analyzing the fractions of lipoproteins, it was found that in the LADA group, the concentration of LDL-C increased by 2.3 times compared to the control ($p=0.000$). The level of HDL-C in the LADA group was 43.3% lower than in the control group ($p=0.000$). The TG level significantly