retinopathy I). 8 patients had an initial cataract. The systolic blood pressure level was on average 137 mm Hg. Art., DBP-83 mm Hg. Art. The level of glycemia in all patients on an empty stomach during the last year average 8.85 mmol/1. 12 patients do not have another vascular complications.

Conclusion: thus, it can be concluded that, to some extent, diabetic microangiopathy occurs in patients with type 2 diabetes in the stage of decompensation, with the duration of the course of diabetes from 1 year to 10 years. However, timely ophthalmologic examination allows to reveal the development of DR at an early stage. A stable compensation of carbohydrate metabolism and hypertension prevents aggravation of DR.

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THE ROLE OF CYANOTOXIN B-N-METHYLAMINO-L-ALANINE (BMAA) IN HUMAN NEURODEGENERATIVE DISEASES

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Research relevance: β-N-methylamino-L-alanine (BMAA) is a non-protein amino acid, wich is produsedby cyanobacteria (blue-green algae). BMAA is supposed to play a significant role in serious neurological human diseases, notably the amyotrophic lateral sclerosis/Parkinson-dementia complex (ALS/PDC).

Objectives: to review literature focusing on the role of naturally occurring cyanobacterial toxin β-N-methylamino-L-alanine in human neurodegenerative diseases.

Materials and methods: I have studied literature, which is focused on research of BMAA and cases of neurodegenerative diseases, which were potentially caused by this amino acid, and made a review.

Results: BMAA was first discovered on the island of Guam in seeds of the cycad tree Cycas micronesica, which were used as a food by local population. As BMAA was shown to be neurotoxic and contains in those seeds, it was supposed to be the main cause of the high incidence of ALS/Parkinsonism-dementia complex (ALS/PDC). Later reports about the presence of BMAA in the brain of the deceased patients, who suffered from ALS, PDC, or Alzheimer 's disease in USA, support this hypothesis. BMAA is structurally similar to glutamate and binds to glutamate receptors. It is toxic to neurons at concentrations as low as 10–30 nM. BMAA is produced by cyanobacteria that are present in all aquatic and terrestrial ecosystems and may be accumulated in living tissues in free and protein-bound forms. The possibility of a global presence of BMAA, and its presentation in human bodies lead to the hypotesis that BMAA might be related to the global presence of neurodegenerative diseases.

Conclusions: the current state of knowledge recognizes the neurotoxicity of BMAA on cellular and in vivo level. BMAA may biomagnify in food chains, enter the human diet, and potentially trigger neurodegenerative disease. No other natural toxin has been shown to be the causative factor of a neurodegenerative disorder to date.

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DEVELOPMENT AND AGE-RELATED FEATURES OF MALE REPRODUCTIVE SYSTEM

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Research relevance: disorders of male reproductive system being widely spread in modern global world require good knowledge of its development sources and mechanism algorithm. Genital swelling that occurs on 3–4 w. of gestation is the source of male reproductive system development. Primary reproductive cells appear earlier.

Objective: to analyze the period and age-related features of male reproductive system differentiation paying special attention to its characteristic organs.

Materials and Methods: the literature data give the information that genital cords in which there are reproductive cells grow into mesenchymal stroma of primary kidney from genital swelling. Paramesonephral sulcus is parallel to mesonephral sulcus and chips off. This period is the most crucial and meaningful.

Results: the indifferent stage of reproductive system development observed in both genders is over on the 6 week of embryogenesis. Since this period the differentiation of males and females starts to develop. The male reproductive system development is characterized by a return development of paramesonephral channel. Genital cords are transformed into wavy seminiferous tubules of the testis, while distal ends of genital cords are connected with primary kidney tubules that form appendage tubules. The upper part of mesonephral channel forms epididymis, and the seminiferous channel is formed by the bottom part. Prostate and seminal vesicles develop as parts of urinogenital sine. Seminiferous tubules are not wavy in newborns and look like continuous genital cords.

Conclusion: at the age of 7–8 the tubules start to have a lumen, genocytes produce spermatogenetic epithelium. Seminiferous tubules become wavier at 10–15, and spermatocytes of 1–2 t. appear in their lumina. Supporting cells reach a complete maturity by 12–16, there is an increase of glandulocytes, sperm and testosterone start to be formed. The number of smooth muscle cells increases at 20–35, this is the period of testis functional activity. The gradual atrophy of ending sections in connective tissue begins after 35. Age involution is observed at 50–80 being characterized by spermatogenetic epithelium reduction, spermatogenesis decrease and growth of connective tissue coat that leads to lumina closing.

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