Materials and Methods: 2 groups of patients were examined and taken biomaterial from. In the 1st group of 300 people after surgery aortic tissue biopsy was inspected as well as the main genes that code the essential proteins of aortic tissues. In the 2nd group there were 470 patients in prospective study. Their biomaterial was inspected for proteins and biomarkers to form a biobank.

Results: augmentation index and aortic stiffness in patients with bicuspid aortic valve is very high in comparison to the control group. It is the most common congenital heart defect (0.5–2% of the population). A bicuspid aortic valve can cause the heart aortic valve to narrow. This condition prevents the valve from opening fully, blocks the blood flow and affects the diameter of the aorta. Biological tissue valves degenerate over time because of it. It is usually a nonsyndromic disease and doesn't harm the patient, that's why people may never know about bicuspid aortic valve. The older you are, however, the higher the chance you may get a thoracic aortic aneurysm later in life. The definitive treatment is surgery for the valve and/or aortic root depending on the severity of valve damage and aortic diameter. The significant complications of bicuspid aortic valve in over one-third of affected individuals often lead to high morbidity and mortality. Surgical interventions include aortic valve replacement, or balloon valvuloplasty.

Conclusion: bicuspid aortic valve is the most common cause of thoracic aortic aneurysm according to the ECHO register. Composition of the extracellular matrix (three-dimensional network of macromolecules, such as collagen, enzymes, and glycoproteins) determines the MMP activity. The amount of extracellular matrix establishes the tissue properties (elasticity and stiffness). The decreasing of matrix can therefore lead to aortic dissection and aneurysm. More significant changes may explain the rapid increase in the aorta diameter in patients with BAV. Patients with BAV have changes in arterial wave reflection due to the properties of aortic wall. This leads to abnormal hemodynamics and, in the future, to some more complications, including calcification and thoracic aneurysm itself.

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ON INVOLVMENT OF EYESIGHT IN CASE OF DIABETES MELLITUS

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Research relevance: diabetes mellitus (DM) is a serious medical and social problem in the XXI century. Diabetes is characterized by a complex of negative effects, primarily due to vascular complications. One of these is diabetic retinopathy (DR). Currently, the DR is ranked among the reasons leading to loss of vision in the population of economically developed countries, and has a high level of disability of the population. DR is observed in 90% of patients with diabetes.

Objectives: to analyze the time of appearance of various types of damage to the organ of vision, as a complication of type 2 diabetes in adult patients.

Materials and methods: the study included 17 patients (34 eyes), with type 2 diabetes in the stage of decompensation (10 men and 7 women) aged 56 to 85 years (average age 67 years). The survey was conducted with the help of specialized questionnaires, prepared by us. The following studies were conducted: visometry, biomicroscopy of the anterior segment of the eye and ophthalmoscopy, on the cycloplegic pupil.

Results: according to the survey 7 patients with diabetes diagnosed 5–10 years ago, and 4 patients with diabetes diagnosed 1–4 years ago. An ophthalmologic examination of all these patients revealed diabetic microangiopathy manifested itself in the form of non-proliferative retinopathy (diabetic

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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