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AMYLOIDOSIS IN THE CARDIOVASCULAR SYSTEM

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In the most industrialized countries with a high degree of urbanization (including Ukraine), the leading cause of morbidity and mortality is occupied by diseases of the cardiovascular system. Detection of amyloidosis is prognostically the most serious complication for patients with various diseases of the cardiovascular system, causes the development of functional organ failure and patient's death.

The aim of the work is a detailed study of amyloidosis problems, especially its etiology and pathogenesis.

Today we know at least about 29 different proteins, which are the causative agents of amyloid diseases, and the specificity of the protein amyloid fibrils allows selecting AL-, AA-, AF-, and ASC-amyloidosis. Causes of diversity of organs and tissues that are affected by amyloidosis are not precisely known, but heart is the most frequently affected organ. Hearts affecting amyloid proteins include such types: AL-amyloidosis (amyloid light chain), familial amyloidosis, senile systemic amyloidosis (SSA), isolated atrial amyloidosis (IAA) and the secondary (AA) amyloidosis. Systemic AL amyloidosis is the most diagnosed form of the clinical amyloid disease. AL fibrils are formed from the monoclonal immunoglobulin light chains and account for most of the variable domains (VL). Hereditary systemic amyloidosis is caused by deposition of amyloid fibrils derived from genetic variants of the transthyretin (TTR), apolipoprotein A-I, lysozyme or alpha chains fibrinogen and other species. Rare manifestations of a familial non-TTR amyloidosis are mutations in genes encoding fibrinogen, gelsolin, lysozyme and apolipoprotein A1 and A2. Senile systemic amyloidosis is caused by the deposition of amyloid fibrils derived from the "wild" type of normal transthyretin and it is always presented by a slowly progressive, infiltrative amyloid cardiomyopathy. The precursor protein for an isolated atrial amyloidosis is atrial natriuretic peptide (ANP), which forms the amyloid deposits only in the atrium. This disease is a true representative of the localized forms of amyloidosis which is not damaging other organs.

The conclusion. Recently amyloidosis of the heart went out of the discharge of rare diseases. This was made possible by the different methods of research, including the possibility of studying the heterogeneity of the protein composition of amyloid fibril formation. Further detailed study of the protein composition of amyloid fibrils allow to understand the etiological features of amyloidosis, to study in detail amyloidogenic and find new ways of early detection in the organism, and the effective treatment of amyloid diseases, which subsequently will lead to a reduction in mortality among people with diseases of the cardiovascular system.

STUDY OF FIBRONECTIN IN PSORIATIC PLAQUE

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Fibronectin (FN) is a family of structurally and immunologically similar glycoproteins that are contained in blood plasma and on the surface of certain cells. One of the most important properties of FN is ability to maintain cell morphology, as well as participation in the processes of cell differentiation and proliferation. Metabolism of soluble and insoluble FN in patients with psoriasis has been studied by some researchers, but the results have been conflicting.

Objective - to study the content of fibronectin in the skin of psoriasis patients.

Materials and methods. The 33 biopsy of psoriatic papules and plaques were taken from 33 patients with psoriasis in the age range 23-50 years have been studied. The immuno morphological study of FN was performed by immunoassay using polyclonal monospecific antibodies to FN.

Results. The immuno morphological study have revealed that FN visualized in the epidermis, where it is normally absent, in all studied specimen. FN was located in epidermis mainly in the granular layer. Glycoprotein was not found in the basement membrane, although just dermo epidermal connection is a typical place of FN accumulation in the normal skin. The large number of FN have been visualized as part of perivascular infiltrates in the dermis.

Conclusions: Thus, pathological dislocation FN was detected in all of the studied skin biopsies. This glycoprotein is deposited in the epidermis, according to psoriatic hyperproliferation. Lack of FN in the basal layer probably connected with its deep penetration into the epidermis and the presence of glycoprotein in the granular layer - with hyperplastic reacting of these cells.

SUBACUTE INFLUENCE OF HEAVY METAL SALTS AND CORRECTION OF THEIR EFFECTS BY VITAMIN E IN THE URINARY BLADDER

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Introduction: One of the dangerous factors for our planet ecosystem is the technogenic environment pollution which have a negatively affect for living organisms. Among the large-scale environment pollution factors of air, water and soil the specific role given to heavy metal salts (HMS). Increasing of the salts content of iron, manganese, chromium, copper, cobalt, nickel, plumbum and zinc around us is a very dangerous, due to their toxicity and spreading. Microelements play the role of metabolic processes catalysts, but their toxicity, excess the maximum allowable limits and excessive influence into the body leads to their cumulation.

Aim: our aim was to research the histological changes in the urinary bladder of rats after the subacute influence of heavy metal salts and vitamin E.

Material and methods: the research was conducted on mature male rats, which were divided into three groups – control, experimental with consumption of HMS, experimental with consumption of HMS and vitamin E. Histological specimens were stained with hematoxylin and eosin.

Results: Compared with control rats, in the experimental group which consumed the HMS were determined morphological changes and edema of all layers of the urinary bladder wall, destruction and desquamation of epithelium, local venous plethora and stasis of blood vessels, signs of local mixed-cells and leukocyte infiltration. The research of experimental group of rats which consumed HMS in combination with vitamin E were revealed the reduction of edema of urinary bladder wall, insignificant mixed-cells infiltration, lack of expressed dystrophic and destructive changes.

Conclusions: Thus, the subacute intoxicated by HMS leads to morphological changes in the wall of urinary bladder. The vitamin E using in combination with HMS leads to compensatative and optimizative of morphological indicators of urinary bladder.