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АКТУАЛЬНІ ПИТАННЯ ТЕОРЕТИЧНОЇ ТА ПРАКТИЧНОЇ МЕДИЦИНИ

Topical Issues of Clinical and Theoretical
Medicine

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AMYLOIDOSIS IN THE CARDIOVASCULAR SYSTEM*Getmans'ka Valeriia, student**Scientific supervisor – PhD, associate prof. Moskalenko R.**Sumy State University, Department of Pathology*

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*Kuksa A., 6 year, 2-nd medical faculty.**Kharkiv national medical university, Kharkiv, Ukraine**Ph.D., Assoc. Prof. Tkachenko S.**Dermatology, venereology and medical cosmetology department*

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.