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FORENSIC-MEDICAL FEATURES OF THE HEART IN CASES OF ACUTE CORONARY INSUFFICIENCY WITH SUDDEN CARDIAC DEATH

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Summary. The article provides an assessment of changes in the endocardium of the atria and ventricles of the heart in cases of sudden cardiac death due to acute coronary insufficiency. In cases of sudden death due to acute coronary insufficiency, distinct focal pathological changes were observed in the connective tissue of the endocardium of the left atrium and ventricle, manifested by loss of fibrillar structures and fragmentation of its non-cellular elements. This corresponded to local contractile changes in the cardiomyocytes, localised in the subendocardial space of the affected parts of the heart, leading to local fragmentation and an increase in the thickness of the endocardial folds. In addition, the loss of their characteristic waviness and the acquisition of linearity were observed. These changes are considered to be manifestations of morpho-functional disorders of the endocardium of the specified heart segments.

The aim of the study was to determine the characteristics of endocardial changes in the atria and ventricles in cases of sudden death due to acute ischaemic heart disease.

Materials and Methods. The study material included 12 hearts from young and middle-aged individuals without cardiovascular disease (control group) and 12 hearts from working-age individuals who died suddenly of acute ischaemic heart disease.

Conclusions: At the time of death from acute ischaemic heart disease, there were pronounced focal pathological changes in cellular and non-cellular components in the connective tissue of the endocardium of the left atrium and ventricle. These changes caused focal contractile changes in cardiomyocytes localised in the subendocardial layer of the myocardium of the left ventricle. The identified changes in the subendocardial layer of the myocardium caused focal thickening, fragmentation, and increased thickness of the endocardial folds, their undulation, which can be considered as manifestations of morpho-functional disorders of the endocardium.

Keywords: atria, ventricles, endocardium, sudden death, acute ischaemic heart disease.

Introduction. The papillary-trabecular apparatus (PTA), consisting of papillary (ventricular) muscles, muscular trabeculae and chordae tendineae, is a component of the heart responsible for its contractile function [1]. The literature provides information on the weight indicators of the ventricles and their papillary-trabecular apparatus under normal conditions, in relation to age, and in certain pathological conditions [2-4], particularly in cases of sudden death in adolescents and young people attributed to connective tissue dysplasia [6]. The endocardium, which covers the PTA, may not at first sight attract much attention as an object of pathological changes in the heart. According to Miao Q. [5], the subendocardial space is of more interest. However, the available information is contradictory and not systematised [7, 9]. For example, in cases of heart failure, there are widespread changes in the relief of the endocardium and the formation of structural arrangements of individual muscle fibres above the level of its surface, which is due to widespread contractures of the muscle fibres of the subendocardial layers of the myocardium. According to the author, in cases of sudden death due to ischaemic heart disease, the pathomorphology of the endocardium is characterised by focal changes in the relief of its surface, also caused by limited contractions of the muscle fibres of the subendocardial layer of the myocardium.

The aim of the study was to determine the characteristics of endocardial changes in the atria and ventricles in cases of sudden death due to acute ischaemic heart disease.

Materials and Methods. The study material consisted of 12 hearts from young and middle-aged individuals who had no history of cardiovascular disease and who had died of mechanical trauma (control group). The characteristics of pathomorphological changes in the endocardium of the atria and ventricles were studied in 12 hearts from individuals of working age who died suddenly of acute ischaemic heart disease (AIHD). Macroscopic examination of the endocardial surface relief was performed on heart specimens after dissection and on serial histotopographic «crosshatch» sections of the atria and ventricles. Injection of a 0.25 % solution of silver nitrate through the coronary arteries was used to identify the characteristics of the endocardial endolethium. Scanning electron microscopy of native specimens (SEMNP) was performed after passing the «critical point» according to Elbaum M recommendations [2]. Scanning electron microscopy of dehydrated myocardial specimens, gold-sputtered and examined under a scanning electron microscope (SEM) was performed to obtain scanograms. The results obtained were analysed using variational statistics.

Results of the study and discussion. The relief structure was found to be quite complex, influenced by the architectural features of the atrial PTA. The PTA is predominantly composed of trabeculae of varying thickness, branching or interconnected by transverse partitions. Spaces in the form of round or oval slits are present between the PTA fragments.

Examination of the endocardial surface by scanning electron microscopy revealed that the endocardial surface of the ventricles is characterised by undulating folds in the form of roller-like elevations and depressions between them, and by tree-like branching of smaller folds from larger «main» folds.

The morphological features of the endocardium contribute to the «roughness» of its relief, which is designed to create turbulence in the blood flow within the heart chambers.

In control subjects and those who died of AIHD, the length of the folds in the atria did not differ significantly in size, averaging 20.0-40.0 μm, with distances between them ranging from 7.0-15.0 μm. The length of the folds in the left ventricle in the control group and in the cases of death due to AIHD averaged 20.0-40.0 μm, with distances between them ranging from 7.0-15.0 μm.

However, in cases of death from AIHD, focal changes in the relief of the endocardium of the left atrium and ventricle were characteristic, including thickening, fragmentation and increased thickness of the folds, together with pronounced undulation.

At magnifications greater than x1000 in scanning electron microscopy, the endocardial surface was observed to be covered by a continuous monolayer of endothelial cells, which were flattened and expanded. Some of these cells showed marginal processes, perinuclear processes and folds. Perinuclear processes were localised in the central part of the endothelial cells, while marginal folds were unevenly distributed, mainly at the edges. Both the endocardial folds and the marginal protrusions, perinuclear elevations and endothelial cell folds can be considered as manifestations of the anatomical-functional features of the endocardium, which are designed to ensure turbulence of blood flow within the cardiac chambers.

Examination of 0.25 % silver nitrate impregnated histological sections of left ventricular myocardium with the endocardial surface showed that in control subjects they were predominantly hexagonal in shape. On average, each endothelial cell was in contact with 6 neighbouring endothelial cells. In cases of sudden death due to AIHD, alcohol and narcotic poisoning, these cells showed polymorphism and the degree of their connectivity with neighbouring cells varied, ranging from 3 to 8.

Using histological methods, it was found that in the subendocardial layer of the myocardium (subendocardial layer), behind the layer of endothelial cells, there was a large amount of elastic fibres, which can be considered as compensation for significant haemodynamic loads on the atria.

Conclusions: At death from AIHD, there were pronounced focal pathological changes in cellular and non-cellular components in the connective tissue of the endocardium of the left atrium and ventricle. Endocardial endothelial cells showed polymorphism, and the degree of their connectivity with neighbouring cells varied from 3 to 8. Pathological changes in non-cellular components included thickening, loss of fibrillarity and evidence of fragmentation. These changes caused focal contractile

changes in cardiomyocytes localised in the subendocardial layer of the left ventricular myocardium. The identified changes in the subendocardial layer of the myocardium caused focal thickening, fragmentation and increased thickness of the endocardial folds, their undulation, which can be considered as manifestations of morpho-functional disorders of the endocardium.

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СУДОВО-МЕДИЧИНІ ОСОБЛИВОСТІ СЕРЦЯ ВНАСЛІДОК ГОСТРОЇ КОРОНАРНОЇ НЕДОСТАТНОСТІ В РАЗІ РАПТОВОЇ СЕРЦЕВОЇ СМЕРТІ

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Резюме. У випадках раптової смерті від гострої коронарної недостатності в сполучній тканині ендокардію лівого передсердя та шлуночків спостерігалися виразні вогнищеві патологічні зміни, що супроводжувалися втратою фібрилярних структур та фрагментацією її не-клітинних елементів. Це спричинили місцеві контрактурні зміни каріоміоцитів, які локалізувалися в субендокардіальному просторі вказаних відділів серця, що призводило до місцевої фрагментарності та збільшення товщини складок ендокардію. Крім того, спостерігалася втрата їх характерної хвилястості та придбання лінійності. Ці зміни є проявами морфофункціональних порушень ендокардію зазначених відділів серця.

Мета дослідження: визначити особливості ендокардіальних змін передсердь і шлуночків у випадках раптової смерті внаслідок гострої ішемічної хвороби серця.

Матеріали та методи. Матеріал дослідження охоплював 12 сердець осіб молодого та середнього віку, які не страждали серцево-судинними захворюваннями (контрольна група) та 12 сердець осіб працездатного віку, які раптово померли від гострої ішемічної хвороби серця.

Висновки. Під час смерті від гострої ішемічної хвороби серця в сполучній тканині ендокарда лівого передсердя та шлуночка спостерігалися виражені вогнищеві патологічні зміни клітинного та неклітинного компонентів. Ці зміни викликали вогнищеві скоротливі зміни кардіоміоцитів, локалізованих у субендокардіальному шарі міокарда лівих відділів серця. Виявлені зміни в субендокардіальному шарі міокарда спричинили вогнищеве потовщення, фрагментацію та збільшення товщини складок ендокарда, їх хвилястість, що можна розглядати як прояви морфофункціональних порушень ендокарда.

Ключові слова: передсердя, шлуночки, ендокард, раптова смерть, гостра ішемічна хвороба серця.

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