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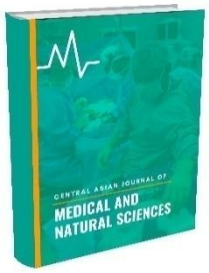

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Adrenal Morphology in Sudden Coronary Death

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Abstract: Modern ideas about the state of adrenal glands in angina pectoris and acute myocardial infarction are based mainly on the data of clinical and laboratory studies and experiments. The issues of functional morphology of adrenal glands in these forms of ischemic heart disease (IHD) are given insufficient attention.

Key words: morphology, pathologic anatomy, adrenal glands, coronary death.

Introduction. As it is known, diseases of cardiovascular system occupy, according to the data of forensic-medical and pathologoanatomical autopsies, the leading place in the structure of morbidity and mortality of able-bodied population. The risk of occurrence, prognosis and outcome of this pathology are largely conditioned by the adequacy of adaptive processes, in connection with which the question of the role of the endocrine system in its development cannot be ignored. Since the late 1970s, attempts to generalize the accumulated material on this issue have been repeatedly made in the specialized literature. Along with the assessment of the contribution of various endocrine organs to the development of the pathology under study, the role of the adrenal glands as one of the leading links of the neuroendocrine system was analyzed to a certain extent in these works [9]. There are no data on the dependence of morphofunctional changes of the gland on the rate of dying, the influence of the underlying disease on the nature of adaptation reactions of the adrenal glands in conditions of acute "cardio-vascular" stress has not been studied, a set of morphological criteria of the functional state of the gland for the purposes of diagnostics of "emergency syndrome of nonspecific adaptation" on sectional material has not been defined. The data available in the literature about hypertrophic, atrophic and dystrophic changes in the adrenal glands in patients with atherosclerosis and hypertension are contradictory and do not lend themselves to a clear systematization that could guide the morphologist in the interpretation of detectable changes, which is due to the great dynamism of gland morphology in extreme states [1, 3].

Acute coronary syndrome is a set of symptoms and clinical manifestations of the period of exacerbation of CHD on the basis of which it is possible to recognize the beginning of NS or MI. The basis of unstable angina and myocardial infarction is coronary artery thrombosis. Thrombus is formed at the site of damage to the integrity of the atherosclerotic plaque [5]. The severity of thrombosis varies. ACS remains one of the leading causes of hospitalization and mortality of patients in industrialized countries. ACS includes early stages of MI onset, in which the highest risk of lethal outcome is observed. In this regard, correct and adequate treatment tactics are of great importance, which determines the outcome of the disease. In this regard, identification of ACS as a group concept

is of great practical importance [8]. In the study of human cadavers after VSS, when there was CHD combined with fresh MI, good preservation of nerve plexuses in the adrenal medulla was noted. At the same time, along with unchanged nerve terminals, numerous reactively changed fibers were found. Signs of these reactive changes were manifested in the focal thickening of nerve fibers, as well as in the hypertrophy of endings detected both on the bodies of chromaffinocytes and on the bodies of microganglion neurons in the adrenal medulla. The histofluorescence study revealed a significant decrease in the luminescence intensity of catecholamines in the adrenal medulla due to the release of hormones into the bloodstream [6]. Gluco- and mineralocorticoids, sex hormones, and catecholamines produced by the adrenal glands significantly affect metabolic processes in the myocardium. Glucocorticoids, contributing to the increase of glucose formation from protein (primarily in muscle tissue), lead to a decrease in the permeability of the vascular wall, provide resistance of the body in stressful situations, induce the synthesis of oxidoreductases, have anti-inflammatory properties, have a positive inotropic effect and, increasing coronary blood flow, favorably affect the characteristics of hemodynamic and ECG-indicators. Therapy with glucocorticoids in patients with acute myocardial infarction contributes to the reduction of the volume of the heart muscle necrosis zone [7]. Mineralocorticoids influence capillary permeability, vascular tone, blood pressure level, besides, they have some properties of glucocorticoids. The influence of sex steroids produced by adrenal glands on a number of functional and metabolic processes that play an important role in the pathogenesis of coronary heart disease (CHD) has also been proved: contractile activity of cardiomyocytes, lipid peroxidation, the state of lysosomal membranes, the level of catecholamines; they have been found to have significant antiarrhythmic activity [4].

These data are of particular interest given the information that the volume of sex steroid hormones produced by the adrenal glands exceeds their total amount secreted by other organs [2]. The dependence of the severity of morphofunctional changes in the adrenal glands on the duration of the atonal period was also noted. If during a short atonal period, up to 24 h, morphologic changes in the adrenal glands reflected the suppression of the cortical substance function, then during a long atonal period they had the opposite direction [5]. It was noted that in myocardial infarction complicated by circulatory insufficiency, there is a sharp increase in the functional activity of the tubular zone, with electrolyte parameters clearly correlating with the structural features of the adrenal cortex. Functional activity of the bundle zone in myocardial infarction is increased throughout the disease [3]. The increase in adrenal glucocorticoid function observed in the early terms of myocardial infarction is replaced by suppression during its organization.

Conclusions: Thus not only quantitative but also qualitative changes in adrenal steroidogenesis in the acute and subacute periods of myocardial infarction have been established [2]. In the acute period there is an increase in the excretion of products with low androgenic activity. The excretion of cortisol and androgenic fractions of corticosteroids in patients with large-focal myocardial infarction directly depended on the duration of pain attack, especially in reflex collapse. With increasing severity, concomitant chronic circulatory failure, glucocorticoid and androgenic functions of the adrenal glands significantly decreased.

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