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SOME ASPECTS OF ATRIAL MYOCARDIAL PATOMORPHOGENESIS IN SUDDEN CORONARY DEATH

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Abstract: Sudden cardiac death (SCD) occupies an important place in the structure of mortality from cardiovascular diseases. Every year, several million people suddenly die around the globe. SCD accounts for 15-20% of all non-violent deaths among residents of industrialized countries. According to recent data, in approximately 60% of cases, IHD is clinically manifested by acute coronary syndrome, in 24% by stable angina, and in the remaining 16% of cases by SCD. Old age is a condition in which the likelihood of sudden death increases. The most common cause of sudden death in the elderly is ischemic heart disease, which accounts for more than 80% of cases of SCD.

Keywords: Sudden death, method, circulatory failure, old age.

INTRODUCTION

The World Health Organization's definition of sudden cardiac death is that it occurs unexpectedly in individuals considered healthy or in good condition within six hours of the onset of a heart attack [1].

The most important thanatogenetic risk factors for SCD include the triad: electrical instability of the myocardium (manifested clinically by ventricular arrhythmias), decreased contractile function of the left ventricle (decreased cardiac output) and myocardial ischemia (angina). It has been established that the most universal mechanism of cardiac arrest in SCD is ventricular fibrillation; asystole is less common [2]. The key to the problem of SCD is to identify the trigger mechanism that induces ventricular fibrillation.

MATERIALS AND METHODS

In case of sudden coronary death, both chronic and acute manifestations of coronary heart disease are found in the myocardium of the deceased, in which cardiosclerotic changes and hypertrophy of the heart muscle develop against the background of severe atherosclerosis.

Most researchers attribute increased tone of the sympathetic system and reperfusion of ischemic myocardium as triggering mechanisms for SCD [3]. However, along with this information, there are opposing or clarifying opinions, indicating the role of adrenaline in the occurrence of cardiac fibrillation.

Along with data on structural changes in the ventricles of the heart during sudden cardiac death, there are only a few studies on the morphofunctional state of the atria and their features in this pathology in elderly and senile people.

RESULTS AND DISCUSSION

In this regard, the purpose of this study was to study the characteristics of the morphofunctional state of the atria in elderly and senile people with coronary artery disease during sudden death.

To achieve this goal, autopsy material (heart) of 20 suddenly deceased people with coronary artery disease in old and senile age was used.

The material for histological examination was taken an hour after death, fixed in a 15% formaldehyde solution, and after soaking in alcohols of increasing strength, it was embedded in paraffin. Sections 7-8 microns thick were deparaffinized, stained with hematoxylin and eosin, according to van Gieson, according to Nissl, and impregnated with silver. Histological sections were studied using a Standard 25 microscope.

For electron microscopy, the material was fixed in a 2.5% glutaraldehyde solution in phosphate buffer for 2 hours and further fixed in 1% OsO4 in the same buffer with sucrose. The material was dehydrated in alcohols of increasing concentrations and acetone, and poured into Araldite according to the generally accepted method. Sections were examined on JEM 100B and EMV-100AK electron microscopes.

The analysis of structural changes in the myocardium of the ventricles of the heart and the identified changes coincided with the available data on those in sudden cardiac death against the background of pronounced and characteristic age-related features [2]. They manifested themselves in atherosclerotic arteries, the presence of spasm and complete closure of their lumen due to plaques and signs of cardiosclerosis, stromal edema, and hypertrophy of preserved cardiomyocytes. Sometimes there were scars from former necrosis and fragmentation of muscle fibers along the intercalary discs. In some areas, there was vacuolar degeneration of cardiomyocytes, pyknosis, and vice versa, hypertrophy of nuclei and cells. Destructively changed areas alternated with hypertrophied or slightly changed fibers.

Due to less research, we examined the atria in sudden cardiac death.

Structural changes in the left atrium during sudden cardiac death often involved all tissue components. Atherosclerotic plaques were found in the arteries. Elements of the microvasculature were sharply expanded with formed blood elements in some areas and emptying of the lumen and a small number of capillaries in others. Blood clots occur in veins and atherosclerotic arteries. The intervening spaces are often expanded due to stromal edema or the development of adipose tissue.

Electron microscopy in cardiomyocytes reveals swelling of the sarcoplasm, disappearance of glycogen lumps, swelling of mitochondria with destruction of crypts, and stripes of myofibril overcontraction, which is associated with hypokinesia and contracture type of myocardial damage.

When assessing the condition of the atrial myocardium, the degree of severity of changes in the intramural ganglia and pathways is important. In many ganglia, only single neurons are found and sclerotic changes in the stroma are present. In some neurons in the ganglia, vacuolar degeneration of the cytoplasm, deformation and death of nuclei, neuronal atrophy and stromal sclerosis occurred. Nissl staining reveals a basophilic substance in some of them, but signs of edema and atrophy of some neurons remain.

CONCLUSION

The data obtained indicate profound changes in the atrial myocardium during sudden cardiac death in elderly and senile people. Along with signs of ischemic vascular damage due to atherosclerosis, changes that are attributed directly to fibrillation are of interest [2]. To these we include pronounced fragmentation of myocardial fibers and destructive signs in the neurons of the intramural ganglia, which are postganglionic motor cells of the parasympathetic system, transmitting vagal influence, which contributes to the predominance of the influence of

catecholamines of sympathetic innervation. One of the causes of SCD, namely arrhythmias, asystole, blockades, should be called edema of the interstitial stroma and compression of the pathways. Due to the possibility of foci of necrosis (infarction) in the atria, the phenomenon of reperfusion of ischemic myocardium with the release of arrhythmogenic substances leading to ventricular fibrillation and sudden death is also possible.

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