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FORENSIC MEDICAL ASPECTS OF PATHOLOGICAL CONDITIONS IN THE THANATOGENESIS OF SUDDEN DEATH IN ISCHEMIC HEART DISEASE

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ABSTRACT: A review of literature data on the role of cardiac conduction system pathology in the thanatogenesis of sudden death in alcoholic cardiomyopathy and coronary heart disease from a forensic medical perspective is provided. Modern data on the main pathological changes in the heart's conduction system in alcoholic cardiomyopathy and coronary heart disease are presented. Modern and historical aspects of pathology in the conducting system of the heart are highlighted. Further ways to develop a more detailed study of the thanatogenesis of sudden death in ACMP and coronary artery disease have been identified.

Keywords: cardiac conduction system, sudden death, alcoholic cardiomyopathy, ischemic heart disease.

Alcohol consumption is an integral element of the lifestyle, culture, and everyday life of the majority of the population in many countries worldwide, and is perceived by the public as a socially acceptable phenomenon. However, unreasonable and chronic alcohol consumption causes numerous negative social and medical consequences, leading to physical and moral degradation of a person, which "leaks out" as a chronic alcohol disease or chronic alcohol intoxication. Prolonged alcohol abuse in more than 50% of cases leads to heart damage. However, alcohol affects not only the heart muscle; in chronic alcohol intoxication, pathology also occurs in other internal target organs: the liver, brain, pancreas, etc.

Alcoholic cardiomyopathy (ACMP) is one of the manifestations of chronic alcohol intoxication, which is known to be characterized by multiple organ pathologies. It is one of the relatively common forms of sudden cardiac death and ranks second in frequency after sudden coronary death. Occurs the next day after alcohol exacerbation, and more often after several days of alcohol abuse, at the patient's "emergence" from intoxication. The appearance of pain is not related to physical exertion. The pain lacks the characteristic seizure-like nature typical of angina, i.e., the clarity of its appearance and disappearance, almost never occurs behind the sternum, and is not compressive or pressing in nature. Unlike angina, nitroglycerin in ACMP does not relieve pain, which can last for hours or days. ACMP can manifest as acute rhythm disturbances, atrial flutter paroxysms, or tachycardia. Arrhythmia paroxysms always develop after alcoholic exacerbations, while attacks of rhythm disturbances are often repeated multiple times. The connection between arrhythmia and alcohol abuse is usually clearly observed by the patients themselves. In the genesis of paroxysmal rhythm disturbances in ACMP, in addition to the toxic effect of ethanol on the myocardium, it is necessary to consider the sympathetic-tonic effect of alcohol. Myocardial dystrophy and cardiosclerosis, which develop in ACMP and form its morphological basis, can be accompanied by a disruption of the myocardial contractility and the appearance of heart failure symptoms.

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The mechanism of ethanol's damaging effect on tissues and organs is related to the cytotoxic effect of its metabolite - acetaldehyde. These include both direct toxic effects on cell membranes and indirect effects through other metabolic pathways. In particular, it has been established that ethanol suppresses oxidation-reduction processes by separating the respiration and oxidative phosphorylation processes in mitochondria, activates the lysosomal system, stimulates the lipid peroxidation process, and activates phospholipase A. This can result in damage to cell membranes, increasing their permeability. Death in ACMP, like sudden coronary death, occurs due to fatal heart rhythm disorders resulting in ventricular myocardial fibrillation.

Much remains unclear regarding the triggering mechanisms of ventricular fibrillation in sudden cardiac death. In our time, a significant place in the genesis of this process has begun to be given to arrhythmogenic substations (lysofosphoglycerides, cyclic adenosine monophosphates, free fatty acids, lipid peroxidation products, some forms of prostaglandins) formed in areas of myocardial hypoxia. Under conditions of complete cessation of blood supply to the ischemic section of the myocardium, the arrhythmogenic substances remain inactive in the zone of coagulation necrosis. When residual blood flow or reperfusion persists, these substances spread through the myocardium with blood flow, causing its fibrillation and often subsequent sudden death. This phenomenon has been termed ventricular reperfusion fibrillation, possibly primarily due to reperfusion secondary calcium-induced damage to cardiomyocytes. Purposeful studies of arrhythmogenic substances have shown that in sudden death from coronary heart disease, the content of phospholipids decreases in the myocardium of the ventricles, interventricular septum, and atrium, while the concentration of free fatty acids increases sharply. Such shifts in the biochemical composition of the myocardium during sudden death, according to researchers, indicate the destruction of myocardiocyte membrane phospholipids with the formation and accumulation of free fatty acid lizoforms of phospholipids (lysofosphhatidylcholine and lysofosphhatidylethanolamine), which have a cardiotoxic effect while simultaneously reducing calcium content in the mitochondria of myocardiocytes. The accumulation of lizoforms of phospholipids in mitochondria in coronary artery disease has a thanatogenetic significance, as it causes electro-physiological changes in the myocardium with a decrease in the maximum diastolic potential, amplitude and duration of action potentials, its fractionation, which in combination gives an arrhythmogenic effect. According to epidemiological studies conducted in recent years, sudden death outside of medical institutions is the most frequent variant of death from coronary artery disease, with about 70% of people dying suddenly, and in 2/3 of cases, the duration of a fatal attack does not exceed 1 hour.

Among the many factors contributing to cardiac electrical instability, myocardial hypertrophy, autonomic disorders, hyperthyroidism, intoxication with ethanol, sympathomimetics, and monoamine oxidase inhibitors, etc., are indicated. According to some data, the morphological manifestations of cardiac electrical instability are characterized by: myocardial ischemia foci; hemorrhages and damaged areas along the nodular tracts of the sinus node; hemorrhages or damaged areas involving most of the specific muscle fibers of one of the central nodes of the PSS or a narrow part of the bundle branch. These changes, as a rule, are combined with signs of focal exhaustion of the adrenergic innervation of the heart with the loss of significant areas of

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catecholamines in the nerve plexuses of the myocardium, with relative preservation in these zones of the cholinergic nerve plexuses. According to other authors who hold somewhat different opinions, acute changes in the cardiac conduction system of sudden death in the form of necrosis and hemorrhages are rare. Among this group, necrotic foci in the myocardium were found in only 13-40% of cases in those who died within the first 1-6 hours. Such phenomena are explained by L.V. Kaktursky by the fact that the structure of the PSS is extremely resistant to ischemia, and even in the infarct zone, the PSS fibers die later than the contractile fibers. This is evidenced by the fact that in the early stages of ischemia, in cases of sudden cardiac death, structural damage to the PSS cannot be detected. In a significant number of cases of sudden death in patients with coronary heart disease at the preclinical and early clinical stages, acute changes in the heart muscle are detected in the form of focal damage to cardiomyocytes, leading to electrical instability and cardiac arrest. According to published data, in sudden coronary death, focal myocardial damage of the contracture type occurs among acute changes in the heart muscle in all observations, while foreign authors find contracture injuries in sudden death only in 72-88% of observations. Some authors believe that focal myocardial damage of the contracture type is characteristic of stress reactions and is caused by the effects of catecholamines, therefore it is also found in the control group of persons who died from violent death. The only difference is that the density of these injuries in sudden death is 14 times greater.

If we delve deeper into history, we can note that cardiomorphologists hypothesized the existence of the sinoatrial node of the heart even before describing its morphological substrate. Later, as a result of a thorough histological examination, a sinus node, which plays the role of a heart rhythm controller, was discovered, which was confirmed by elegant comparison of anatomical and electrophysiological data. The PSS consists of specialized cells that initiate heartbeat and coordinate the contraction of heart chambers. The sinoatrial node is a small group of specialized heart muscle fibers located in the wall of the right atrium. It is located to the right of the point where the superior vena cava enters and normally produces an electrical impulse for contraction. The atrioventricular node is located under the endocardium in the lower posterior part of the interatrial septum. A Giss bundle branches from the atrioventricular node, passing through the interventricular septum anteriorly and posteriorly. Inside the partition, the Giss bundle is divided into a wide network of fibers that pass through the left part of the partition - the left leg of the Giss bundle, and into a compact part in the form of a wire running along the right side - the right leg of the Giss bundle. The rhythmic contraction of the heart is ensured by the sequential passage of an electrical impulse through the PSS. The sign of electrical stimulation is the excitation potential, which is formed due to ion currents through the special channels of the sarcolemma. Heart cells responsible for electrical excitation are divided into three types according to their electrophysiological properties, studied through intracellular introduction of microelectrodes:

- Pacemaker cells - rhythm drivers (for example, in the sinoatrial and atrioventricular nodes);

- specialized, fast-conducting fabric (e.g., Purkinje fibers);

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- muscle cells of the ventricles and atria.

As noted above, in normal conditions, the heart rhythm is maintained by the function of the sinoatrial node, in the cells of which the generation of sinus impulses occurs. If we proceed from the generally accepted notions that the occurrence of fibrillation is associated with impairments in the excitability, conductivity, and automaticity of the heart, then the study of the PSS, which carries out the formation and propagation of impulses of cardiac excitation, becomes particularly relevant. This issue is practically not covered in the literature, and small morphological studies of PSS pathology in cases of sudden death were usually limited to the study of its lower sections. The sinus-atrial node is located constantly above the right ear at the point where the superior vena cava enters the right atrium, on the lateral side under the epicardium, and is separated from the endocardium only by a thin layer of connective and muscular tissue. The nodule is not recognized by the naked eye, as it merges with the surrounding tissue in color. The node has the shape of a flat ellipse or crescent located horizontally, its length is approximately 10-15 mm, its height is 5 mm, and its thickness is 1.5 mm. A characteristic feature of the sinus-atrial node is the presence of a disproportionately large artery running in the center of the node along its longitudinal axis. This artery in 55% of cases is one of the first branches of the right coronary artery that branches directly from the aorta; in the remaining 45% of cases, it branches from the initial part of the left coronary artery's circumferential branch. This makes the node's position superior in terms of its function as a control apparatus for central aortic pressure and pulsation. The structural basis of the node is the connective tissue framework, consisting of collagen, elastic, and reticular fibers, in which bundles of fine specialized muscle fibers are located. The characteristic features of these fibers are their random arrangement, lack of transverse stripes, and significantly smaller diameter compared to the fibers of the atrial contractile myocardium. At the periphery of the node are located nerve ganglia, single ganglion cells, and nerve fibers, which are also abundant in the node's tissue.

Detailed analysis of electrocardiograms recorded immediately before death shows that in most cases, the basis of any heart disease that led to sudden death lies in acute disruptions in the rhythm of cardiac activity, which transition into ventricular fibrillation or asystole.

Studies have shown that it is not always possible to identify specific morphological signs explaining the genesis of the fatal outcome of an arrhythmia attack, and in a number of cases, both chronic and acute pathological changes in the node tissue are detected, indicating a possible weakening of its function. These include: a significant increase in the proportion of the connective tissue trunk (fibrosis or fibroelastosis of the node) with a simultaneous decrease in the number of specialized fibers; lymphoid infiltrates in the node tissue; stenosis, and in some cases, fresh thrombosis of the sinus artery; eosinophilia, homogenization, and swelling of the myocyte sarcoplasm, myocytosis of individual specialized fibers; micro-hemorrhages around the node, partially penetrating the node tissue and surrounding the nerve trunks and ganglia; changes in the nerve ganglia located near the node - hyperchromatosis, vacuolization, eccentric displacement of the nuclei, their pycnosis, and caryolysis.

Thus, to develop a more detailed study of the thanatogenesis of sudden death in ACMP and coronary artery disease, the following tasks can be addressed in the future:

- screening of morphological changes in the PSS in death from ACMP;

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-study of the degree of fibrosis and lipomatosis of the PSS in patients with coronary artery disease and ACMP in a comparative aspect;
- study of tissue levels of acetaldehyde in death from ACMP;
- Immunohistochemical analysis of plasma protein transudation products and PSS cell cytoskeleton elements during death from coronary artery disease and ACMP in a comparative aspect.

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