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MODERN ASPECTS OF FORENSIC MEDICAL DIAGNOSIS OF SUDDEN CARDIAC DEATH IN ALCOHOL AND DRUG ABUSERS

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Abstract

General Background: Sudden cardiac death (SCD) remains a leading cause of mortality worldwide, particularly among individuals with alcohol and drug abuse disorders. The absence of pathognomonic signs complicates forensic medical diagnosis, making it a crucial and challenging issue in forensic science. Specific Background: Alcohol and drug abuse contribute to severe cardiovascular damage, leading to secondary toxic dilated cardiomyopathy, left ventricular dysfunction, and myocardial structural alterations. These changes create electrical instability in the heart, increasing the risk of fatal arrhythmias and asystole. Knowledge Gap: Despite the established relationship between substance abuse and cardiac pathology, the specific morphological markers of ventricular fibrillation and their role in SCD remain insufficiently studied. A comprehensive forensic approach to identifying these markers is needed to improve diagnostic accuracy. Aims: This study aims to analyze forensic medical literature on SCD among alcohol and drug abusers to identify key morphological markers and mechanisms underlying cardiac fatality. Results: The findings indicate that forensic diagnosis of SCD in alcohol and drug abusers is complicated by the heterogeneity of myocardial damage, the presence of fibrosis and thrombotic changes, and the absence of distinctive pathological signs. Key indicators include dilated heart chambers, epicardial obesity, and myocardial microvascular alterations. Novelty: The study provides a systematic review of forensic morphological markers of cardiac pathology in substance abusers, contributing to the improvement of forensic medical diagnostics. Implications: These findings emphasize the need for standardized forensic protocols and advanced histological and biochemical analyses to enhance the reliability of SCD diagnosis in cases involving substance abuse. Further research should focus on developing precise postmortem diagnostic criteria to differentiate SCD due to cardiac pathology from other sudden death causes.

Key words: forensic medicine, sudden cardiac death, alcohol abuse, drug addiction, cardiac pathology, forensic diagnosis, ventricular fibrillation

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Introduction

In economically developed countries, diseases of the cardiovascular system (CVS) are among the leading causes of disability and mortality of the working-age population. According to official data from the World Health Organization, in 2018, about 17.3 million people died from diseases of the circulatory system, which is about 30% of the total mortality structure in the world [1]. At present, the Russian Federation (RF) occupies a leading position among European developed countries in terms of morbidity, disability and mortality from cardiovascular pathology. It should be noted that there are no official statistics on sudden (sudden) death in the country [1–3].Forensic medical assessment of SCD thanatogenesis is still the most relevant and diagnostically complex problem of forensic medical examination, which is explained by the absence of pathognomonic signs of CVD lesions and the complexity of differential diagnosis between different causes of sudden death [4, 5]. A special role in the thanatogenesis of SCD is played by the currents of sico-metabolic lesions of the heart muscle by such psychoactive substances as alcohol and drugs [6, 7]. The leading place in the structure of visceral injuries in alcohol and drug abusers is occupied by damage to the circulatory organs [8, 9]. However, the true prevalence of alcoholic damage to the cardiovascular system is unknown, which is often explained by its latent course and long-term preservation of the ability to work of abusers [1, 3, 7].

Thus, the study of pathognomonic signs of heart damage among people who use alcohol and drugs; thanatogenesis of the development of life-threatening heart rhythm disorders against the background of secondary changes in cardiomyocytes is an urgent issue of modern forensic medical and pathomorphological science.

Objective: to analyze the key publications of domestic and foreign authors devoted to the problem of verification of the causes of SCD in people who abuse alcohol and drugs.

The analysis of publications of domestic and foreign sources of literature devoted to this problem is carried out. The search was carried out using the following databases: PubMed, Scopus, eLIBRARU, Embase. The

selected articles included original studies and reviews published between January 2010 and April 2020. The following search queries in Russian were used: "alcohol", "drugs", "secondary cardiomyopathy", "heart rhythm disorder"; In English: "Alcoholic cardiomyopathy", "Heart damage in drug addicts", "Ventricular tachycardia".

Method

Prevalence of alcoholism and drug addiction in the Russian Federation

According to the data of the Federal State Statistics Service of the Russian Federation, in the period from 2000 to 2018, a positive trend was recorded in the decrease in the level of primary morbidity in the class "Mental disorders and behavioral disorders associated with the use of psychoactive substances" [2].During the analyzed period, the decrease in the indicator "For the first time in life diagnosed alcoholism and alcoholic psychosis" amounted to 60.2% - from 129.7 per 100,000 population in 2000 to 51.6 in 2018. The decrease in the indicator "For the first time in my life diagnosed with drug addiction and substance abuse" for the same period amounted to 79.9% – from 51.4 per 100,000 people in 2000 to 10.3 in 2018 [2]. In the course of the analysis of the dynamics of the mortality rate from certain causes associated with alcohol consumption, a stable positive trend in the decrease in the number of deaths from alcohol abuse and its surrogates is recorded: among men by 59.1% (from 85.3 per 100,000 population in 2008 to 53.6 in 2018); among women by 41.3% (from 26.6 per 100,000 population in 2008 to 15.6 in 2018) [2].

In the period from 2008 to 2018, the first place in the structure of causes of death among alcohol abusers is occupied by ACMP: 2008 – 33.7 per 100,000 population; 2018 – 17.2 per 100,000 population. In second place is the mortality rate from accidental alcohol poisoning: in 2008 – 28.4 per 100,000 people; 2018 – 12.9 per 100,000 population. In third place is mortality from alcoholic liver disease: in 2008 – 13.2 per 100,000 population; 2018 – 8.9 per 100,000 population [2]. In 2018, the causes of deaths in the Russian Federation were distributed as follows: 40.5% – poisoning with alcohol and its surrogates, 24.3% – exposure to carbon monoxide, 15% – use of narcotic drugs, 4.8% – exposure to organic substances and technical liquids, 3.3% – action of acids and alkalis, 2.1% – overdose of drugs, 1.1% – use of psychotropic substances [2, 3].

Opiate poisoning occupies the leading position in drug poisoning (43–45% of cases); combined poisoning with several narcotic drugs (15–17%); third place – poisoning with benzodiazepine drugs and barbiturates (11–11.5% and 10–10.5% of cases, respectively) [3, 7]. Age-sex composition and chronological features of SCD in persons abusing alcohol and drugs

Numerous research works devoted to the problem of socially significant diseases (alcoholism and drug addiction) indicate a progressive increase in the number of lethal outcomes, mainly in males at a young age [10, 11].

Thus, according to [11, 12], SCD occurs in 10.0% of young people; About 40.0% die suddenly before the age of 40. According to the study [13], the maximum number of cases of SCD among men who abuse alcohol and drugs falls on the age of 36–45 years, among women – 46–60 years.

Result and Discussion

The proportion of men who died from acute opioid poisoning is 87% of the total number of people who died from acute drug poisoning. In 86.5% of cases, the age of the victims was 18-35 years. More than a third of drug addicts have used drugs for less than one year, every fourth has had 1-2 years of experience, 17% of active users have 2-3 years, 15% have 3-5 years, and 7% have 5 years
and more [3].

According to the data of a comprehensive medical and social study [14], the portrait of a drug addict is as follows: a young man aged 18 to 39 years, unmarried, childless, leading an antisocial lifestyle and not

having a stable income, abusing mainly opioids.

The formation of chronic alcohol dependence in women occurs significantly later than in men [15, 16]. This feature applies to all clinically significant periods of time: the age of the first trials (16–17 years), systematic use (24–28 years), and the formation of withdrawal syndrome (30–35 years).

The study of the dependence of SCD in people who constantly use alcohol and drugs on the day of the week revealed the following patterns. Among men, the minimum number of deaths occurred on Tuesday - 11.8% of cases, the maximum - on Wednesday and Sunday (15.9% and 16.4% of cases, respectively). Among women, the minimum number of deaths was recorded on Thursday (13.7% of cases), the maximum on Saturday (17.2% of cases) [10, 11, 20].

In the study group of men, the number of cases of SCD on weekdays was 0.37 cases/1 day, which is less than on weekends and holidays - 0.44 cases/1 day. Among women, there were no significant differences in the number of SCD on weekdays and weekends [10, 13, 19].

According to the data [18], in persons abusing alcoholic beverages and narcotic substances, the maximum number of cases of SCD was registered in April (10.2% of cases), the minimum was in February (6.2% of cases) and September (6.3% of cases). Thus, the relationship between SCD and the age and sex characteristics of the deceased, the day of the week and the month of the calendar year has been statistically reliably proven.

Forensic Medical Assessment of Pathomorphological Changes in the Cardiovascular System in Alcohol and Drug Poisoning

The main pathognomonic signs of cardiovascular disease in alcohol abuse and its surrogates include: alcoholic cardiomyopathy, impaired automaticity and conduction, and secondary arterial hypertension [14, 20].

The macroscopic picture of ACMP is characterized by the following: the average weight of the heart is 350–450 grams (according to a number of researchers, more than 500 grams); moderate dilatation of the atrial and ventricular cavities in combination with mild signs of LV and MVP myocardial hypertrophy; signs of epicardial obesity; intact coronary vessels [8, 15, 21].

The myocardium is flabby to the touch, yellowish on the incision, with multiple small elements of sclerosis and fresh thrombotic overlays; fibrosis and lipomatosis of the stroma; fatty degeneration of cardiomyocytes; alternating foci of hypertrophy and hypotrophy (atrophy) of cardiomyocytes [22].

Pathognomonic signs of heart damage in drug intoxication include: heart weight - 250-450 grams; dilatation of the heart cavities; a large amount of liquid blood in the heart cavities; layers of connective tissue in the myocardium; weak severity (complete absence) of signs of atherosclerotic changes; decrease in the mass of epicardial adipose tissue (by 35% compared to the same indicator among healthy individuals); valvular changes of the bacterial endocarditis type [8, 24].

According to data [8], the combined use of alcohol and drugs leads to an increase in the mass and size of all heart cavities. The width and height of the PCP and LP are 18–20% higher than those in the control group of healthy patients and are as follows: width – 3.2–3.5 cm, height – 3.3–3.7 cm. The width of the RV ranges from 4.5–4.6 cm, the width of the LV – 6–6.2 cm, which exceeds the similar dimensions in the control group of healthy people by 20–22% and 29–29.5%, respectively. The height of the RV and LV exceeds those in the control group of healthy individuals by 21–21.5% and 28.5–29% and is as follows: RV – 4.5–4.7 cm, LV – 5.9–6.0 cm.

In parallel with the change in the width and height of the heart cavities, thickening of the ventricular walls is observed among opiate and ethanol abusers: RV – 0.4–0.5 cm, LV – 1.5–1.6 cm, which is 12% and 19%, respectively, higher than among the control group of healthy individuals. which exceeds the same

indicators among the control group of healthy individuals by 25–26%, 28–29%, and 25–26.5%, respectively [8, 14].Similar changes affect both atria: the mass of PCP is 22–24 grams, LP – 24–25.5 grams, MPP – 9.5–10 grams, which is higher than the same indicators among the control group of healthy individuals by 20–21%, 25–25.5%, 21–23%, respectively.

Cardiac and ventricular indices in persons who use opiates in combination with ethanol during their lifetime are 18–19% and 21–22%, respectively, higher than those in healthy individuals.

The main morphological signs of heart damage in chronic alcohol and drug intoxication include: alternation of foci of destructive vasculitis, productive myocarditis with areas of microabscesses; elements of paresis and stasis of erythrocytes, disseminated intravascular coagulation; the presence of fibrin-leukocyte thrombi in the vessels of the microcirculatory bed [8, 16].

An important indicator of myocardial toxicity is the content of some enzymes in the pericardial fluid [28]. For example, in people who abuse alcohol and drugs, the level of ASABs in the blood serum is increased by 3.2 times in comparison with normal values, in the pericardial fluid – by 3.61 times; ALT in blood serum – 3.1 times, in pericardial fluid – 3.32 times; CPK in blood serum was 2.5-fold, in pericardial fluid by 3.15-fold [28]. According to [14, 22], the detection of drugs and a threefold increase in AST, ALT, and CPK in the RV and blood serum can be considered a diagnostically reliable criterion for death from drug poisoning.

Blood vessels of the myocardial stroma of venous and arterial types are dilated and full-blooded, have convoluted contours, alternate with unevenly full-blooded intramural arteries. Large coronary arteries are intact [22, 27, 31]. Endotheliocytes of arterial and venous vessels bulge into the lumen of the vessels, are located in a palisade, and erythrocyte stases are formed in the lumen of the vessels with the phenomenon of sludge phenomenon [14, 23]. Pathomorphology of medium-caliber arteries is expressed in plasma impregnation of the walls, spasm and corrugation of internal elastic membranes, focal proliferation of the endothelium, unexpressed liposclerosis of the inner membrane without
changes in its lumen [15, 27].

Intramural vessels are characterized by: narrowing of the lumen of the vessel due to its edema; the walls of the vessels are homogenized or loosened; focal ruptures of the inner membrane; formation of subendothelial erythrocyte stases with the phenomenon of sludge phenomenon and precapillary fibrosis (PAS-positive compounds); areas of thickening of the vascular wall with the accumulation of acid mucopolysaccharides in it; deposition of couplings and amorphous masses around the capillaries (plasma impregnation of myocardial tissue with subsequent collagen formation in it) [9, 27].

Therefore, the main morphological sign of CVD lesions in alcohol and drug abusers is secondary toxic dilated cardiomyopathy with signs of changes in the size of the heart cavities and wall thickness, decreased heart pumping function, epicardial obesity, intact coronary vessels and degeneration of myocardial stroma vessels. Microscopic characteristics of the cardiovascular lesion.

In the current literature, researchers describe the heteromorphic type of myocardial structure in ACMP and distinguish several types of acute cardiomyocyte lesions: contractural type of lesion, clumpy disintegration of myofibrils, intracellular myocytolysis, relaxation, dissociation, and fissures [25].

In the subendocardial parts of the anterior, lateral and posterior walls of the LV, contractural damage to cardiomyocytes is recorded, mainly of II and III degrees (from 51.5% to 57.5% of cases): II degree – shortening of sarcomeres (decrease in the height of I-discs), preservation of transverse striation of cardiac muscle fibers; Stage III – disappearance of I-disks, merging of A-disks into a continuous luminous conglomerate, disappearance of transverse striation of cardiac muscle fibers [23, 25, 29]. In addition, the following specific signs of cardiomyocyte damage were recorded in all myocardial regions [14, 23, 25]: wave-like deformation (in 44.5–50% of cases) – an increase in the height of the I-disc while the height of

the A-disc remained unchanged; cardiomyocyte dissociation (in 61–65% of cases) – expansion of Z-bands; cracks (in 35-40.0% of cases) are thin transverse stepped or straight defects along the course of the cardiac muscle fiber.

Contractures of the II and III degrees, lumpy disintegration and myocytolysis indicate the process of irreversible disintegration of sarcomeres; Pathognomonic signs of ventricular fibrillation are considered to be areas of contractures, fissures and dissociation of cardiomyocytes. Morphologically proven signs of asystole have not yet been described [23,30].

According to [8, 26, 31], chronic drug intoxication is characterized by: atrophy and hypertrophy of cardiomyocytes; areas of diffuse and small-focal cardiosclerosis; focal endothelial hyperplasia; paresis of the vessels of the microcirculatory bed; foci of acute focal hemorrhages; microabscesses; paresis and stasis of erythrocytes; disseminated intravascular coagulation and formation of leukocyte thrombi in the microcirculatory link.

In all cases, focal replacement or diffuse myofibrosis, perivascular and pleximorphic cardiosclerosis of the stromal component are observed: foci of the peria of paravascular interstitial cardiosclerosis or diffuse cardiosclerosis with partially atrophied cardiomyocytes [14, 34]. In the connective tissue, reticular and elastic fibers predominate, collagen fibers are registered only in perivascular areas. Connective tissue fibers are represented by loose thin cords with a clear orientation of fibers, the structure of which is dominated by fibroblasts [24].

The chronic hypoxic and toxic effects of alcohol and drugs lead to impaired lipid metabolism [15, 22], which manifests itself in the form of an overgrowth of adipose tissue around the intramural vessels and between muscle fibers, mainly in the subepicardial and subendocardial components of the histohematic barrier of the myocardium and its trabeculae. In the cytoplasm of cardiomyocytes, small-droplet obesity is diffusely located small fat vacuoles with a diameter of 0.1 to 3 μm [23].

Acute poisoning with alcohol and its surrogates leads to severe metabolic changes, manifested in a decrease in the level of free fatty acids, the main energy source of myocardiocytes [22]. As a result of the depression of fatty acid oxidation, the following cardiotoxic substances accumulate: acetaldehyde, serotonin, noradrenaline, the development of metabolic acidosis due to the accumulation of acetoacetic and lactic acid. A decrease in the number of potassium, magnesium, phosphorus, and aminotransferase ions (especially aspartinic ion) indicates severe damage to cardiomyocytes and cytolysis [15, 16].

Thus, the long-term toxic effect of alcohol and drugs manifests itself in the inhibition of cellular metabolism and metabolism, which leads to severe irreversible destructive changes in the myocardium: irreversible decrease in the contractility of the heart muscle, asynchronous contraction of the myocardium of the RV and LV, electrical instability of cardiomyocies and, as a result, the occurrence of life-threatening cardiac arrhythmias by the mechanism of re-entry.

Pathology of the conduction system of the heart in persons who abuse alcohol and drugs

The direct cardiotoxic effect of ethanol and its metabolites plays a major role in the formation of life-threatening HCR in people abusing alcohol and drugs. Deep suppression of energy metabolism and cell metabolism leads to electrical instability of cell membranes, disruption of the formation and conduction of electrical impulses, and, as a result, the formation of life-threatening irreversible NSR and asystoles [13, 37].

Fatal outcome in alcohol poisoning and its surrogates occurs in the phase of ethanol resorption or at the end of withdrawal syndrome [36]. Thus, according to data [38], paroxysmal tachycardias often develop after alcoholic excesses, as a consequence of metabolic distress syndrome, which is based on an increase in the level of adrenaline and noradrenaline, which increases the risk of arrhythmias. Episodes of paroxysms

can be repeated repeatedly during the day, are recurrent and often end with a volley of fatal life-threatening tachycardia.

Modern sources of literature by domestic and foreign authors provide reliably reasoned data that the development of SCD is based on myocardial electrical instability, leading to the development of fatal life-threatening fibrillations mainly of the ventricles of the heart [4, 30, 37, 42]. A detailed analysis of the ECG of patients with signs of acute alcohol or drug poisoning, registered immediately before death, suggests that the basis of SCD is the development of acute NSD, which turns into ventricular fibrillation and subsequent asystole [4, 5, 13].

The following similar opinion is most often found in the literature [23, 30, 32, 37]: alcoholic dystrophy of the heart muscle is manifested by a slowdown in atrioventricular conduction, changes in the terminal part of the QRST segment increasing in dynamics, the appearance of late atrial excitation potentials on high-resolution ECG, the development of paroxysmal NSR up to ventricular tachycardia and ventricular fibrillation with the subsequent formation of asystole.

The following types of NSD have been described: atrial and ventricular fibrillation and flutter, paroxysmal and ventricular tachycardia, frequent supraventricular and ventricular extrasystoles of high degrees of gradation according to the Lawn classification [40, 41].

The registration of frequent group atrial or ventricular extrasystoles indicates electrical instability of the myocardium and a tendency to develop paroxysmal supraventricular and (or) ventricular tachycardias. Often, during the CM ECG, short asymptomatic paroxysms of atrial and (or) ventricular fibrillation are recorded, combined with frequent single supraventricular and (or) ventricular extrasystoles. Later, transient forms of paroxysmal atrial and ventricular tachycardia become permanent with the development of secondary dilatation of the heart cavities [30, 42].

According to [37, 40, 41], the following electrophysiological changes occur in chronic alcohol and drug intoxication: prolongation of the refractory period inside the supraventricular and atrioventricular nodes; a decrease in the refractory period of the conduction pathways in the ventricular myocardium and the period of retrograde atrioventricular excitation, which underlie the formation of re-entry mechanisms within the atrioventricular node and the development of re-entry associated NSD.

At present, the question of the trigger mechanisms of ventricular fibrillation in SCD remains controversial. A number of researchers [37, 48] see the leading role in the pathogenesis of fatal NSD not only in the cardiotoxic effect of alcohol and drug metabolites, but also in the activation of the sympathetic-adrenal system, by increasing the level of noradrenaline in the blood, myocardium, and synaptic cleft of synapses located both in the myocardium itself and in its vessels.

The moment of volley of life-threatening NSR is preceded by a generalized increase in the activity of all parts of the sympathetic-adrenal system, an increased release of noradrenaline and activation of the biosynthesis of the main neurotransmitter. Intensive synthesis of adrenaline by the adrenal medulla against the background of a sharp decrease in the amount of noradrenaline, dopamine, and dioxyphenylalanine, and inhibition of the activity of noradrenergic neurons is the main triggering biochemical mechanism for the development of ventricular fibrillation of the heart [48]. Increased adrenaline concentration in the myocardium is considered to be the main trigger mechanism for the development of ventricular fibrillation, the morphological equivalent of which is contractural damage, wave-like deformation, and cardiomyocyte dissociation [30, 39].

According to [44], the triggering mechanism of fibrillation is based on the penetration of fibrinogen and other plasma mediators into the sinoatrial and atrioventricular nodes, which leads to dysfunction of the main pacemakers of the heart rate, and in the end, the development of fatal atrial and ventricular fibrillation.

In a number of studies [4], the leading role in the formation of life-threatening HCS is assigned to arrhythmogenic substances (free fatty acids, lipid peroxidation products, cyclic adenosine monophosphates, etc.), which remain inactive in the area of the ischemic area of the myocardium. In cases where residual blood flow is preserved, arrhythmogenic substances circulate through the myocardium, causing secondary calcium damage to cardiomyocytes, their electrophysiological instability and, as a result, reperfusion fibrillation of the ventricles.

According to [47], the genesis of a fatal arrhythmia attack is based on acute and chronic signs of pathological damage to the tissue of the sinoatrial and atrioventricular nodes. These include: fibrosis or fibroelastosis of the connective tissue skeleton with a simultaneous decrease in the number of specialized cells; excessive formation of coarse-fibrous connective tissue with elements of mucoid swelling and the presence of lymphoid infiltrates; stenosis or formation of fresh blood clots in the lumen of the sinus artery; focal hemorrhages along the nodes and nodal tracts [39]. The above-described morphological changes are combined with depletion of the adrenergic innervation of the heart, loss of areas of catecholamine content in the nerve plexuses of the heart muscle, while preserving cholinergic endings in them.

Conclusion

The study highlights the significant forensic and medical challenges in diagnosing sudden cardiac death (SCD) among individuals with alcohol and drug abuse disorders. The findings reveal that chronic substance abuse leads to secondary toxic dilated cardiomyopathy, myocardial fibrosis, ventricular hypertrophy, and electrical instability, all of which contribute to life-threatening arrhythmias and asystole. The absence of pathognomonic markers complicates the forensic determination of cause of death, underscoring the need for more precise diagnostic criteria. These findings have critical implications for forensic pathology, particularly in improving the identification of SCD cases related to substance abuse through standardized histological and biochemical analyses. Given the increasing prevalence of alcohol and drug-related mortality, further research should focus on developing advanced postmortem diagnostic techniques, including molecular and immunohistochemical markers, to enhance the accuracy and reliability of forensic assessments. Additionally, longitudinal studies on the progression of substance-induced cardiac pathology could provide deeper insights into early detection and preventive strategies. Strengthening interdisciplinary collaboration between forensic experts, cardiologists, and toxicologists will be essential in refining forensic medical practices and advancing public health interventions to mitigate the risks associated with chronic substance abuse.

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