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Modern Forensic Approaches to the Diagnosis of Sudden Cardiac Death

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Abstract This article analyzes the role of modern imaging techniques and the significance of morphological findings in the forensic investigation of sudden cardiac death (SCD). SCD is characterized by a high frequency of fatal outcomes occurring within a short period following the onset of initial symptoms, or in their absence. A significant challenge in forensic practice arises when macroscopic autopsy findings are inconclusive. Contemporary approaches to the postmortem diagnosis of SCD integrate morphological, histological, and immunohistochemical analyses. The identification of signs of acute myocardial ischemia, cardiomyopathy, channelopathies, and substrates for ventricular fibrillation is of particular importance. The development and implementation of standardized postmortem diagnostic algorithms can improve the accuracy of cause-of-death determination, especially in young individuals.

Keywords Sudden cardiac death, Forensic examination, Myocardial infarction, Cardiomyopathy, Morphology

1. Introduction

Sudden cardiac death (SCD) remains a leading cause of mortality worldwide, particularly among young adults. Modern research emphasizes the molecular and genetic underpinnings of SCD, offering new perspectives for forensic diagnostics. In economically developed countries, the annual incidence of SCD ranges from 18.6 to 128 cases per 100,000 inhabitants [1,2]. Epidemiological data indicate that ischemic heart disease (IHD) is the predominant cause of SCD overall. In individuals under 45 years of age, SCD is most frequently associated with various forms of cardiomyopathy. In an analysis of deaths in persons under 35, Andersson, Vasan, et al. found that IHD and cardiomyopathy accounted for a significant and comparable percentage of SCD cases [3,4,5]. Postmortem genetic analyses have revealed that in up to 40% of SCD cases attributed to cardiomyopathy, mutations were identified in genes responsible for cardiac structural proteins, while in 60%, mutations were found in genes related to ion channel function and electrolyte metabolism [6,7]. It is also noted that impaired cardiac conduction can result from mutations in genes encoding desmosomal proteins [8,9].

According to World Health Organization statistics, cardiovascular diseases (CVDs) accounted for 30% of all global deaths in 2008, amounting to 17.3 million lives lost; of these, 7.3 million deaths were due to IHD and 6.2 million to stroke. Projections suggest that by 2030, approximately 23.6 million people will die from CVDs annually, with heart disease and stroke likely remaining the leading causes [WHO].

In forensic practice, the primary criteria for determining the cause of sudden death are the macroscopic and microscopic findings from autopsy, aimed at identifying significant morphological alterations in organs and tissues [10,11,12]. However, a comprehensive understanding of the clinical, morphological, and molecular biological characteristics of various SCD etiologies is required to elucidate its mechanisms fully. Such understanding is crucial for developing effective diagnostic, preventive, and therapeutic strategies. Although modern forensic examination increasingly incorporates advanced laboratory and instrumental techniques, numerous challenges persist, necessitating further research.

The purpose: To study the pathomorphological mechanisms of sudden cardiac death and their significance for determining thanatogenesis in forensic medical examination, to improve diagnostic accuracy.

2. Materials and Methods

A clinical, anamnestic, morphological, and statistical study was conducted. The study material comprised 126 autopsy cases from the Republican Scientific and Practical Center for Forensic Medical Examination (Tashkent), collected between 2021 and 2025. The diagnosis of SCD was confirmed based on clinical history and morphological data. During autopsy, tissue samples from all major internal

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organs, including the heart, were collected for histological examination. Tissue sections were processed using standard methods, embedded in paraffin, and stained with hematoxylin and eosin. All histological slides were examined in detail using light microscopy at 10×, 20×, and 40× magnifications. The most informative and significant pathomorphological changes identified in the heart, lungs, kidneys, and vasculature were documented photographically.

3. Results and Discussion

Histopathological analysis of myocardial tissue revealed the following characteristic changes (Figure 1): Cardiomyocyte Dystrophy: Pronounced dystrophic changes were observed, including granular and vacuolar degeneration. There was a loss of transverse striation, myolysis, and cytoskeletal disintegration within the cytoplasm. These alterations, which develop in the context of ischemia, hypoxia, and metabolic stress, lead to a complete loss of cardiomyocyte function. Cell Death: Signs of myocytolysis and fragmentation were detected in individual cells, indicating terminal cardiomyocyte injury. Cellular Disproportion: Pathological hypertrophy of some cardiomyocytes contrasted with atrophy in others, suggesting a focal hemodynamic imbalance within the myocardium. Interstitial Edema: Edema of the cardiac interstitium, associated with hypoxic injury and venous congestion, was noted. This leads to stromal loosening, destabilization of tissue architecture, and disruption of intercellular signaling. Microcirculatory Disturbances: Impairments manifested as passive dilation of capillaries and venules, alongside erythrocyte stasis, indicating a severe reduction or cessation of blood flow. These findings are characteristic of acute cardiac decompensation leading to death without resuscitative intervention. The spectrum of morphological abnormalities described was most frequently associated with underlying cardiomyopathies and neurogenic cardiac injury in our series.

Table 1. Occurrence of sudden death due to pathogenetic causes (in 126 studied cases)

Age group (amount)	SD occurrence	Description
18-29 years (n=9)	Relatively low	Channelopathies, hypertrophic cardiomyopathy, and myocarditis resulting from hereditary and arrhythmogenic causes.
30–39 years (n=18)	Average	Arrhythmias, the initial stage of ischemic disease, (effect of pregnancy, medications)
40–49 years (n=31)	High	Atherosclerosis, ischemic heart disease, hypertension, and diabetes that arose against the background of such risk factors
50-59 years (n=35)	High	Conditions associated with cardiovascular diseases (IHD, fibrillation, valve pathology)
60 and older (n=33)	High	Arising as a result of concomitant somatic diseases

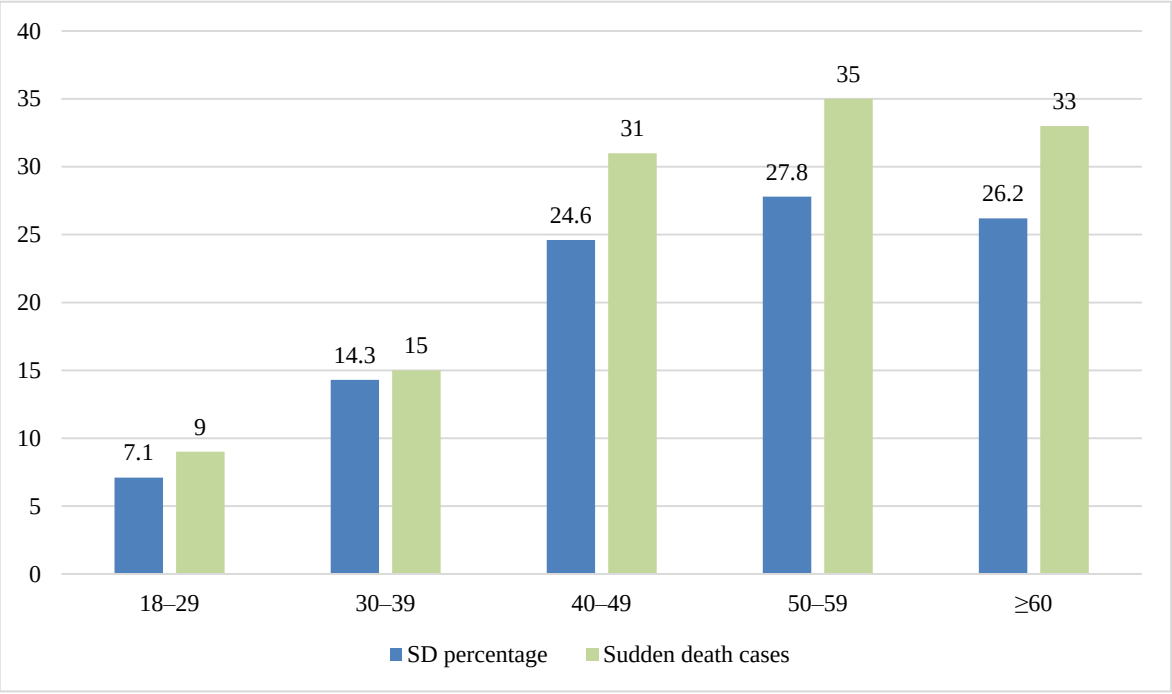

Diagram 1. Age distribution of sudden death cases and their percentage by age groups

Table 2. Distribution of sudden death by age group (n = 126)

Age group (years)	SD occurrence rate	Percentage of SD
18–29	9	7,1%
30–39	18	14,3%
40–49	31	24,6%
50–59	35	27,8%
≥60	33	26,2%
Total	**126**	**100%**

A review of medical histories, autopsy reports, and ancillary data from the studied cases provided insight into the morphogenetic and pathomorphological changes in parenchymal organs and the cerebral and major systemic vasculature. The analysis confirmed that SCD occurs across all age groups, with varying prevalence and etiological profiles according to age.

Upon analyzing these cases, the highest incidence of sudden death was recorded in the 50–59-year age group (27.8%) and the ≥60-year age group (26.2%). The 40–49-year age group also showed a high percentage, constituting 24.6%, indicating that cardiovascular diseases and their complications become most active and manifest during this age period. A relatively low incidence of SD (7.1%) was observed in the young adult group (18–29), where cases often occurred against a background of genetic or arrhythmogenic pathology. In the 30–39-year age group, 14.3% of cases were recorded; SD in this age group frequently arose due to latent cardiac pathologies, the effects of unsupervised medication use, or inflammatory processes. The causes of sudden death identified included pulmonary artery thromboembolism in 32 cases (%), acute renal failure in 14 cases (%), pulmonary edema in (89.5%), DIC syndrome in 17 cases (30.6%), and cerebral edema in 41 cases (10.3%) (Table 2).

Sudden death cases were categorized into 5 groups based on etiology: cardiogenic, arrhythmogenic, neurogenic, occurring against a background of latent disease, and resulting from exogenous causes.

Arrhythmogenic SD: In our study, among deaths up to 18-30 years of age where hereditary pathology was present, 4 cases observed in the hospital were associated with an SCN5A gene mutation (located on chromosome 3, involved in coding for the biosynthesis of sodium channel proteins in cardiomyocytes) resulting in significant ST-segment elevation on ECG. Neurogenic SD: 4 cases were attributed to cerebral circulatory disturbance due to subarachnoid hemorrhage. Cardiogenic SD: 29 cases were due to myocardial infarction, and 7 cases to myocarditis. Out-of-hospital SD: 32 cases of sudden death without prior clinical signs occurred outside the hospital. Other: 18 cases were due to thromboembolism, 4 cases were due to complications of pregnancy, and 1 case resulted from adverse drug effects.

The analysis revealed that in humans, various pre-existing chronic conditions and comorbid diseases served as a background leading to SD, including hypertension (27.3%), IHD (25.0%), diabetes mellitus (18.1%), obesity (20.4%), HLP (11.3%), and diseases such as rheumatism and cardiomyopathy. Clinical-morphological analysis of those who died from SD showed that fatalities during the acute phase of the disease constituted 61.7%, while cases occurring in conjunction with background diseases accounted for 38.3% (Graph 3).

One of the factors leading to vascular endothelial injury is considered to be the effect of the SD mechanisms mentioned above. As a result of arrhythmogenic destructive influence, severe dystrophic changes intensify in endothelial cells as well. This is due to vacuolization of their cytoplasm, desquamation, and detachment. Endothelial injury is a key local factor for thrombosis, leading to intravascular coagulation of blood and fibrinogen. Studying the pathomorphological changes resulting from these mechanisms at the microscopic level allows for the following conclusions:

1. Cases of sudden death most frequently occur in individuals over 50 years of age, particularly in the 50–59 (27.8%) and ≥60 (26.2%) age groups, which together constitute nearly half of all cases. This is related to the untimely diagnosis and inadequate treatment of cardiovascular diseases, especially ischemic heart disease and hypertensive cardiomyopathy.
2. The high rate of SD (24.6%) observed among individuals aged 40–49 indicates the danger of cardiac pathologies that progress with minimal or no clinical symptoms, or under insufficient diagnostic monitoring, during a period of high work activity (e.g., subclinical atherosclerosis, arrhythmias).

Table 3. Pathogenesis of Sudden Death by Etiological Cause

Groups	Causes	Pathologies leading to SD
Cardiogenic SD	Disturbances in cardiac conduction and mechanical activity	IHD, myocardial infarction, cardiomyopathies, myocarditis
Arrhythmogenic SD	Hereditary syndromes	Brugada syndrome, WPW syndrome
Neurogenic SD	Aortic aneurysm, subarachnoid hemorrhage	Cerebrovascular accident
SD Occurring Against a Background of Latent Disease	Disruption of electrolyte balance and thromboembolism due to narcotics	Often without external clinical signs
SD Resulting from Exogenous Causes	Intoxications, traumas	Drugs, toxic substances, pregnancy complications

3. Although SD is less common in the young adult group (18–29 years, 7.1%), cases in this group are primarily due to genetic, arrhythmogenic, or congenital cardiomyopathies, as well as toxic influences (narcotics, energy drinks, stress), which require thorough investigation. Strengthening preventive and screening measures in this direction is warranted.
4. The fact that nearly 40% of SD cases correspond to the 40–49 and 50–59 age groups underscores the necessity in clinical practice of implementing timely diagnostics, echocardiography, and Holter monitoring more widely for assessing the cardiovascular status of individuals in these age brackets.

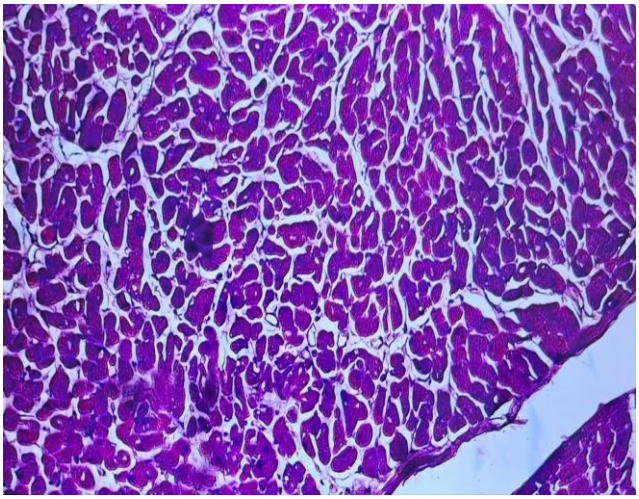

Figure 1. Cardiomyocytes exhibit marked dystrophic changes, fragmentation, and irregular hypertrophy. The cell cytoplasm shows signs of myolysis, and the stroma displays interstitial edema. The myocardium shows uneven blood filling, paretic dilation of capillaries, and erythrocyte stasis. Stained with hematoxylin and eosin. Magnification 10×20

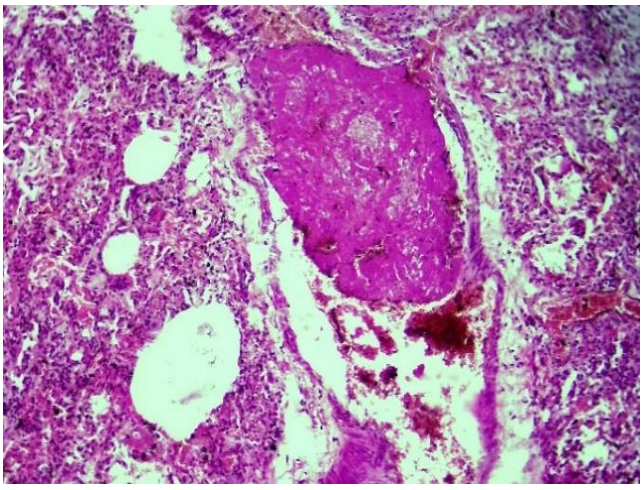

Figure 2. Sudden death occurred during childbirth due to pulmonary thromboembolism (PTE), with a fibrinous thrombus formed in a major branch of the pulmonary artery; thrombi were also detected in the capillaries. Stained with hematoxylin and eosin. Magnification 10×20

Morphological examination of lung tissue (Fig. 2) revealed edema secondary to acute venous congestion in the setting of sudden cardiac death. Pathological changes were also observed in the epithelium of the lower respiratory tract, specifically the bronchioles and respiratory bronchioles. Microscopic analysis identified dystrophic alterations in the cytoplasm and nuclei of epithelial cells, accompanied by interstitial edema. Affected cells exhibited shape distortion, nuclear displacement, detachment from the basement membrane, and, in some instances, desquamation into the bronchiolar lumen. Additional findings included nuclear aggregation, hyperchromasia, flattening, and a transition of the epithelium to a multilayered structure with attenuated, eosinophilic staining. The bronchiolar lumina contained desquamated epithelial cells, erythrocytes, lymphoid cells, macrophages, and tissue debris. Pathological alterations in the bronchiolar wall comprised randomly distributed edematous fibrous structures within the basement membrane, pronounced interstitial edema with myxomatous features, and infiltration by activated macrophages, lymphocytes, erythrocytes, and necrotic material.

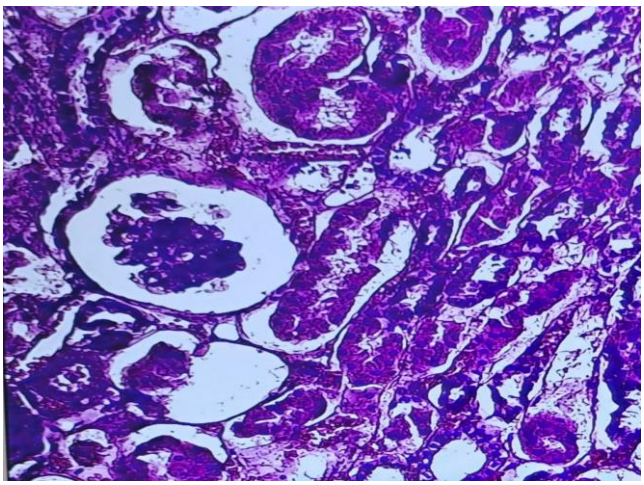

Figure 3. Sudden death was associated with necrosis of the renal convoluted tubules (a) and atrophy of the glomerular capillary network. (Deceased: male, 34 years old). Stained with hematoxylin and eosin. Magnification 10×20

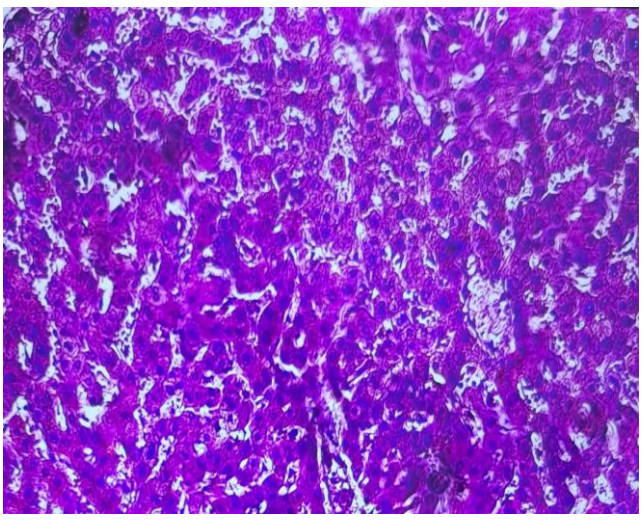

Figure 4. In a case of sudden death, hepatocytes displayed hydropic and vacuolar degeneration, which developed in the context of acute venous congestion. Stained with hematoxylin and eosin. Magnification 10×20

Histological analysis of renal tissue (Fig. 3) demonstrated significant degenerative changes in the tubular epithelium, most prominently in the proximal tubules. Findings included vacuolar degeneration of the cytoplasm, pyknosis of nuclei, and signs of nephrosis indicated by homogeneous cellular staining. Desquamation of epithelium into the tubular lumen, cellular debris, and abundant cytoplasmic material were observed in some tubules, which were often dilated and occasionally filled with cellular aggregates. While glomeruli were largely preserved, the Shumlyansky-Bowman capsule exhibited a weak filtration reaction, indicative of a passive process. The interstitial stroma showed marked edema without evidence of inflammatory leukocytic infiltration. Vascular changes included signs of passive hyperemia, erythrocyte stasis, and capillary dilation. Arteriolar walls were thinned, displaying alterations characteristic of ischemia. These findings are consistent with acute ischemic tubular necrosis, typically arising in the context of sudden death, hemodynamic shock, adrenal insufficiency, or ischemic hypoxia.

Examination of liver tissue (Fig. 4) revealed hepatocytes with signs of hydropic and vacuolar degeneration, developing in the context of acute venous congestion. Bile canaliculi exhibited features of mechanical cholestasis due to bile stasis.

4. Conclusions

Sudden cardiac death (SCD) remains a leading cause of mortality in the working-age population and poses a significant diagnostic challenge in forensic medicine. Determining the cause of death in such cases necessitates a comprehensive approach, mandating the use of histological and immunohistochemical methods. In this study, the highest incidence of SCD was identified in the 50–59 (27.8%) and ≥60 (26.2%) age groups. The application of modern technologies, such as molecular autopsy and subcellular imaging, significantly enhances pathogenetic analysis and enables the detection of latent hereditary cardiopathologies and channelopathies not apparent on routine macroscopic examination. The standardization of forensic diagnostic algorithms for SCD—incorporating detailed history-taking, clinico-pathological correlation, and integration of advanced laboratory methods—is of paramount importance. In young individuals and in cases without significant atherosclerosis, primary diagnostic focus should be directed toward cardiomyopathies and primary electrical heart disorders. This necessitates close collaboration among forensic experts, geneticists, and pathologists.

In summary, the refinement of forensic diagnostic methodologies for SCD, grounded in evidence-based medicine and a multidisciplinary approach, not only clarifies the immediate cause of death but also contributes to preventive strategies for families with a hereditary predisposition.

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