

ПАТОМОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В СЕРДЦЕ ПРИ ВНЕЗАПНОЙ СЕРДЕЧНОЙ СМЕРТИ

З.К.Хакимов.¹, А.Р.Маматалиев.², М.А.Маматалиева.³

^{1,2,3}Андижанский Государственный медицинский институт.

Для цитирования: © Хакимов З.К., Маматалиев А.Р., Маматалиева М.А.

ПАТОМОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В СЕРДЦЕ ПРИ ВНЕЗАПНОЙ СЕРДЕЧНОЙ СМЕРТИ.ЖКМП.-2024.-Т.1.-№1.-С

Поступила: 16.01.2024

Одобрена: 03.02.2024

Принята к печати: 05.03.2024

Аннотация: В исследовании изучены морфологические и морфометрические изменения в сердце 80 человек в возрасте 20-70 лет и старше перенесших внезапную сердечную смерть. В танатогенезе внезапной сердечной смерти важное значение имеют параллельные возрастные изменения геометрии сердца, атеросклеротические изменения коронарных сосудов, симпатических нервных пучков и ишемия миокарда, фибрилляция желудочков и асистолия, приводящие к электрической нестабильности.

Ключевые слова: морфометрия, внезапную сердечную смерть, танатогенез, гистохимический метод, импрегнация солями серебра.

TO‘SATDAN YURAKDAN O‘LIMIDA - YURAKDAGI PATOMORFOLOGIK O‘ZGARISHLAR

Z.K.Xakimov.¹, A.R.Mamataliyev.², M.A.Mamataliyeva.³

^{1,2,3}Andijon Davlat tibbiyot insituti.

Izoh: © Xakimov Z.Q., Mamataliev A.R., Mamataliyeva M.A.

TO‘SATDAN O‘LIMDA YURAKDAGI PATOMORFOLOGIK O‘ZGARISHLAR. KPTJ.-2024-N.1.-№1-M

Qabul qilindi: 16.01.2024

Ko‘rib chiqildi: 03.02.2024

Nashrga tayyorlandi: 05.03.2024

Annotatsiya: Tadqiqotda to‘satdan yurakdan o‘limi kuzatilgan 20-70 va undan katta yoshdagi 80 ta insonlar yuragini morfologik va morfometrik o‘zgarishlari o‘rganilgan. To‘satdan yurakdan o‘limlarda yurak geometriyasida, toj tomirlarida aterosklerotik o‘zgarishlarda, simpatik nerv tutamlarida va miokard ishemiyasidagi yoshga bog‘liq parallel o‘zgarishlar - qorinchalar fibrilyastiyasi va asistoliyasi, elektr nostabilligiga olib kelishi to‘satdan yurakdan o‘lim tanatogenezida muhim ahamiyat o‘rin tutishi aniqlangan.

Kalit so‘zlar: morfometriya, to‘satdan yurak o‘limi, tanatogenez, gistokimyoviy usul, kumush tuzlari bilan impregnatsiya.

PATHOMORPHOLOGICAL CHANGES IN THE HEART IN SUDDEN CARDIAC DEATH

Khakimov Z.Q.¹, Mamataliev A.R.², Mamataliyeva M.A.³

^{1,2,3}Andijan State Medical Institute.

For situation: © Khakimov Z.Q., Mamataliev A.R., Mamataliyeva M.A.

PATHOMORPHOLOGICAL CHANGES IN THE HEART IN SUDDEN CARDIAC DEATH. JCPM.-2024.P.1.№1-A

Received: 16.01.2024

Revised: 03.02.2024

Accepted: 05.03.2024

Annotation: Morphological and morphometric changes in the hearts of 80 people aged 20-70 and older who had sudden cardiac death were studied in the study. Parallel age-related changes in heart geometry, atherosclerotic changes in coronary vessels, sympathetic nerve bundles, and myocardial ischemia in sudden cardiac deaths - ventricular fibrillation and asystole, leading to electrical instability are important in the teratogenesis of sudden cardiac death. significance was found.

Keywords: morphometry, sudden cardiac death, teratogenesis, histochemical method, impregnation with silver salts.

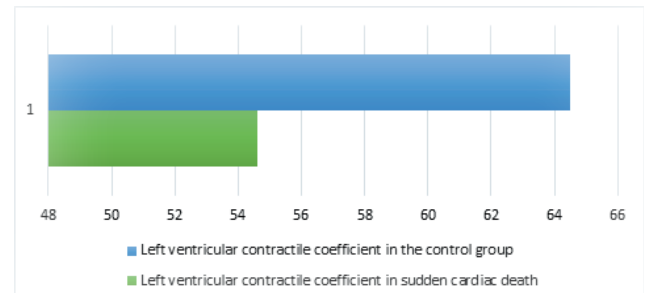
Relevance: Sudden death due to pathological changes in the heart or coronary arteries is usually called sudden cardiac death (SCD). SCD is one of the most common causes of sudden death, accounting for 50% or more of all "cardiac deaths" [1,4]. The mechanism of SCD is associated with ventricular fibrillation or, less often, asystole. It occurs within 1-6 hours from the time the first symptoms of a heart attack appear [2,3,5]. Sudden cardiac death accounts for 0.1-0.3% of all sudden deaths among newborns, 5% in children, 30% in 14-21 year-olds, middle and old age it is 88%. After the age of 40, dystrophic changes in the nerve fibers of the innervation of the heart cause "age-related denervation", a violation of electrical conductivity, resulting in ventricular fibrillation and asystole [6]. The purpose of the study: Study of morphological and morphometric changes in the heart in SCD.

Materials and Methods: in the Study, morphological and morphometric changes of the heart were studied in autopsy and forensic medical histology materials from 20-70 and over 40 subjects with sudden cardiac death in the study and control groups. The dimensions of the heart, its weight, undulating deformity and dissociation of cardiomyocytes, contracture changes, and dystrophic changes in its nerves were studied.

Research results: Structural changes associated with myocardial injury are characteristic of SCD, which are heart geometry– size, cavity shape, muscle mass, and configuration leading to a change in. In the control group, the morphometric examination of the heart revealed the following average parameters: heart volume - 280±3.0g, length 9.5±0.2cm, width 9±0.3cm, thickness 4.8±0.4, left ventricle thickness of the wall - 1.3±0.1cm, thickness of the wall of the right ventricle - 0.4±0.1cm (diagram -1).

In the control group, wavy deformity of cardiomyocytes was equal to 57.5 ± 2.8%, and dysesthesia of cardiomyocytes was equal to 64.4 ± 3.7%. The amount of contractures in the left ventricle was 64.2 ± 5.8%. In the control group, contracture injuries of cardiomyocytes of the 3rd degree were observed in both ventricles.

Diagram - 2
The number of cardiomyocyte waves in the control and research groups (%)



When the amount of undulation of cardiomyocytes (diagram - 2) and the amount of dysesthesia (diagram - 3) were studied about age, it was noted that the indicators were lower in the research group compared to the control group. When the contractile coefficient of left ventricular cardiomyocytes (diagram - 4) was studied about age, it was observed that the indicators in the control group were significantly higher than the indicators in the research group. When the thickness of the wall of the coronary vessels was measured, the morphometric indicators were recorded at the minimum age of 20-29 years and the maximum at the age of 50-59 years. An age-related increase in the dynamics of coronary vessel wall thickening and atherosclerotic damage was observed. It was noted that these changes - lipid spots and fibrotic plaques damage 10-15% of the surface of the vessels and do not affect the permeability and stiffness of the vessels.

Diagram - 3
The number of symptoms of cardiomyocyte dysesthesia in the control and investigated groups (%).

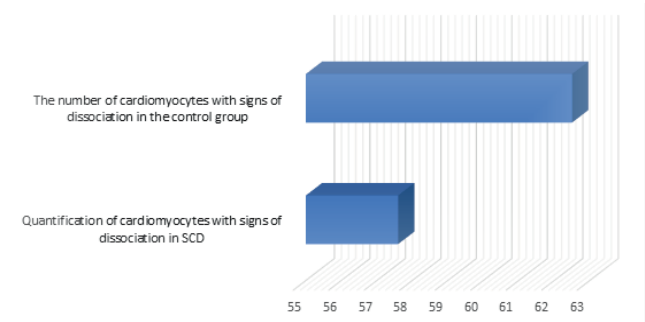
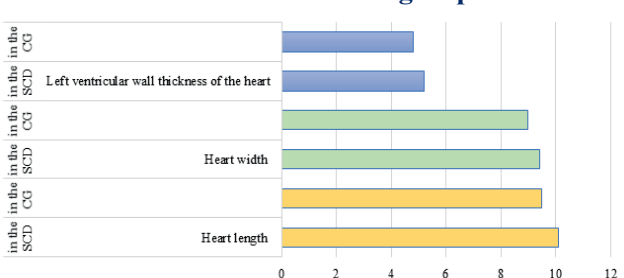


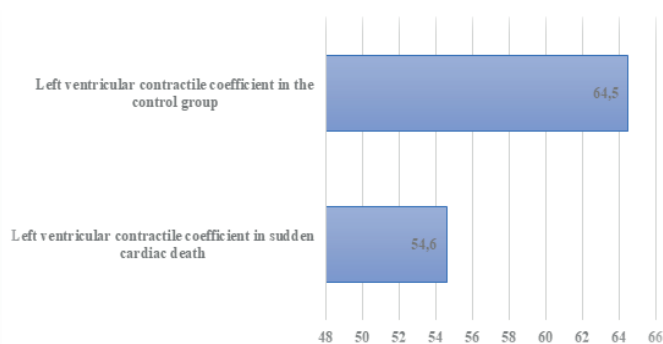
Diagram - 1
Morphometric indicators of the heart (cm) in the control and research groups.



In the examined groups, the average surface size of fibrous plaques relative to the surface of the intima of vessels, depending on age, was $1.0 \pm 0.1\%$ in the lower branch of the left coronary artery in group 1, and $17.2 \pm 1.6\%$ in group 5., and in the branch of the left coronary artery from $0.5 \pm 0.1\%$ in group 1 to $15.2 \pm 1.4\%$ in group 5, and in the right coronary artery from 1.5 ± 0 in group 1, It was $27.1 \pm 2.1\%$ in group 5 from 3% . It was noted that a strong progression of these indicators was observed after 50 years of age, causing stenosis of the vessel space to $1/5-1/4$. Macroscopic myocardium showed small sclerosis foci and atrophy of cardiomyocytes. Dystrophic, necrobiotic changes and focal and diffuse interstitial and perivascular sclerosis were observed in atrophied cardiomyocytes. Deposition and increase of fat inclusions in the cytoplasm of some cardiomyocytes were seen (Pic. 1.1). In sudden cardiac death, macroscopic signs of ischemia in the myocardium, small sclerosis foci, around 10% of lipid spots and fibrous plaques in the intima and media that do not affect the permeability of coronary vessels, microscopic venous to laxity, perivascular sclerosis, ischemia, hypercontractile state of cardiomyocytes, wavy folds, fuchsinophilia, fragmentation were observed.

Diagram - 4

Contractile coefficient (%) of left ventricular cardiomyocytes in control and tested groups.



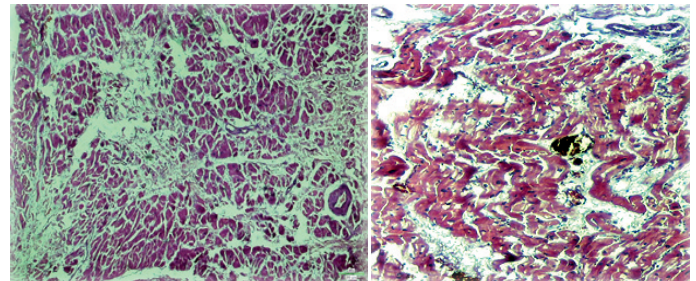
Decreased contractile function of the left ventricular myocardium due to sclerotic changes and conduction disorders was considered the main risk factor of SCD. In the majority of those who died suddenly, no acute changes in the blood vessels of the heart were noted in the morphological examinations. Heart morphometric parameters in the study group: heart weight - $330 \pm 3.3\text{g}$ ($P < 0.001$), length $10.1 \pm 0.2\text{cm}$ ($P < 0.001$), width $9.4 \pm 0.3\text{cm}$ ($P < 0.01$), thickness $5.2 \pm 0.4\text{cm}$ ($P < 0.05$), left ventricular wall thickness - $1.6 \pm 0.1\text{cm}$ ($P < 0.001$), right ventricular wall thickness -

$0.6 \pm 0.1\text{cm}$ ($P < 0.001$). When these indicators were compared, it was higher in sudden cardiac death compared to the control group. During the histological examination of both ventricles, the predominance of 2-3 degree contracture damage of cardiomyocytes was observed (Pic. 1.2).

Picture – 1

Hem.-eosin staining. 4x40 magnification

Hem.-eosin staining. 4x40 magnification



1. Sudden cardiac death (man, 50 years old). There are foci of extensive sclerosis in the tissue of the left ventricle and interventricular septum, swelling of the interstitial tissue, hypertrophy of cardiomyocytes, some of them have a wavy course, fragmentation, perivascular sclerosis, and edema.

2. Contracture injury of cardiomyocytes of the 2nd-3rd degree in sudden cardiac death.

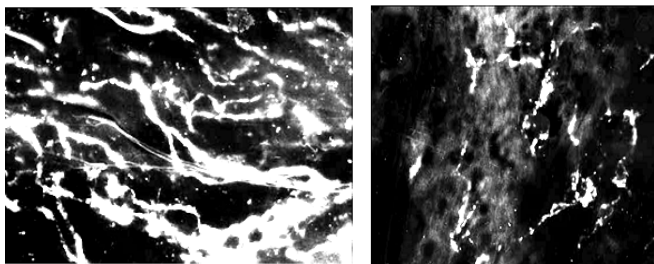
The amount of contractures in the left ventricle was $53.4 \pm 2.0\%$ ($P < 0.001$). The location of contractures was most often found in the anterior, lateral, and posterior walls of the left ventricle, mainly in the subendocardial and intramural sections of the myocardium. In case of sudden cardiac death, wavy deformation in all areas of the myocardium, as well as signs of cardiomyocyte dysesthesia in the area of the placed discs occupying the full field of vision or $2/3$. The number of cardiomyocytes with signs of undulating deformation is $44.2 \pm 5.6\%$ ($P < 0.001$), and the number of cardiomyocytes with signs of dysplasia is $58.7 \pm 2.8\%$ ($P < 0.001$). In the investigated groups, the average surface size of fibrotic plaques relative to the surface of the intima of the vessels was $1.3 \pm 0.1\%$ in the lower branch of the left coronary artery in group 1 and $16.1 \pm 1.2\%$ in group 5., and in the branch of the left coronary artery from $0.7 \pm 0.1\%$ in group 1 to $16.2 \pm 1.1\%$ in group 5, and the right coronary artery from 1.7 ± 1.1 in group 1 % in the 5th group was $32.1 \pm 2.1\%$. A strong progression of these indicators was observed after the age of 50, causing stenosis of $1/5-1/3$ of the vessel cavity.

At autopsies, uneven filling was observed in 1/3 of cases as a sign of macroscopic ischemia in the myocardium. Macroscopic small sclerotic foci and atrophy of cardiomyocytes were manifested. Parallel changes were observed in the myocardium, coronary vessels, and reflexogenic sinocarotid areas. It was noted that the above-mentioned changes "rejuvenate" them when compared with the information in the literature.

In cases of sudden cardiac death, large-scale cardiosclerosis was often observed in the interventricular barrier, which testified to the involvement of the conductive system in the pathological process. As age increased, the importance of involutinal changes in sympathetic innervation increased. It was observed that the initial reactive changes of adrenergic terminals turn into destructive changes. A parallel was noted between their development and the exacerbation of atherosclerotic changes in the vessels (Pic. 2). Reactive and degenerative changes in myelin fibers and receptors in the reflexogenic sinocarotid areas of the heart were observed in the autopsies of people who died suddenly, which culminated in cardiac fibrillation.

Picture – 2. Histochemistry (impregnation with silver salts) x400.

Age-related decrease in the density of adrenergic nerve bundles of the myocardium of the left ventricle.



1. Sympathetic nerve bundles in people aged 30-40.

2. Decrease in the density of sympathetic nerve bundles after 40 years.

In these degenerative changes, the development of heart failure was observed in 8% of people under the age of 40, 24% under the age of 50, 30% under the age of 60, and 38% over the age of 60. Changes in the concentration of cardiac tissue sympathetic nerve bundles and the increase in denervation were seen as the cause of the exacerbation of fibrillations.

Conclusion: Based on the information mentioned above and in the literature, taking material for histological examination from the left ventricle and in-

terventricular septum in autopsy examinations of patients with sudden cardiac death, along with staining with hematoxylin-eosin method, histochemistry to study the condition of sympathetic nerve bundles methods (impregnation with silver salts) are recommended. Increased age-related changes in the nervous system cause neurotrophic disorders of body tissues and, as a result, lead to the development of visceral pathology, and sudden cardiac death.

Changes in the geometry of the heart, atherosclerotic changes in the coronary vessels, changes in sympathetic nerve bundles, and age-related parallel changes in myocardial ischemia - ventricular fibrillation and asystole, and the effect on electrical instability are important in the hematogenesis of sudden cardiac death. is important.

REFERENCES:

1. Яковлева Э.В., Фрейнд Г.Г. Внезапная сердечная смерть: принципы морфологической диагностики // Проблемы экспертизы в медицине. 2012. Т. 12, № 3-4 (47-48). С. 24-26
2. Струков А. И., Серов В. В. Патологическая анатомия: учебник.- М.: ГЭОТАР-МЕДИА, 2019. - 880 с.
3. Фатенков О.В., Рубаненко О.А., Яшин С.С., Авезова Д.Б. Современные аспекты понятия, этиологии, патогенеза и профилактики внезапной сердечной смерти. Кардиология. Наука и инновации в медицине 2(6)/2017. 20-25с.
4. Бокерия О.Л., Биниашвили М.Б. Внезапная сердечная смерть и ишемическая болезнь сердца // Анналы аритмологии. 2013. Т. 10, №2. С. 69-79.
5. Мазур Н. А. Внезапная сердечная смерть // Практик. кардиология. М.: Медпрактика-М, 2009. С. 148–164.
6. Швалев В.Н., Рогоза А.Н., Тарский Н.А. и др. Внезапная сердечная смерть и морфофункциональная диагностика предшествующих возрастных нейродистрофических изменений организма. Pacific Medical Journal, 2017, No. 1, p. 42–51.

Информация об авторах:

- © ХАКИМОВ З.К. – старший преподаватель кафедры Патологической Анатомии и Судебной медицины, Андижанский государственный медицинский институт, г. Андижан. Узбекистан.
- © МАМАТАЛИЕВ А.Р. – кандидат медицинских наук, доцент, заведующий кафедрой Патологической Анатомии Судебной медицины, Андижанский государственный медицинский институт, г. Андижан. Узбекистан.
- © МАМАТАЛИЕВА М.А. – старший преподаватель кафедры Патологической Анатомии и Судебной медицины, Андижанский государственный медицинский институт, г. Андижан. Узбекистан.

Muallif haqida ma'lumot:

- © ХАКИМОВ З.К. – Andijon davlat tibbiyot instituti Patologik anatomiya va sud tibbiyoti kafedrasi katta o'qituvchisi, Andijon sh. O'zbekiston.
- © МАМАТАЛИЕВ А.Р. – tibbiyot fanlari nomzodi, dotsent, Patologik Anatomiya Sud tibbiyoti kafedrasi mudiri, Andijon Davlat tibbiyot instituti, Andijon sh. O'zbekiston.
- © МАМАТАЛИЕВА М.А. – Patologik Anatomiya Sud tibbiyoti kafedrasi katta o'qituvchisi, Andijon Davlat tibbiyot instituti, Andijon sh. O'zbekiston.

Information about the authors:

- © ХАКИМОВ З.К. – senior lecturer of the Department of Pathological Anatomy and Forensic Medicine, Andijan State Medical Institute, Andijan. Uzbekistan.
- © МАМАТАЛИЕВ А.Р. – candidate of medical sciences, associate professor, head of the Department of Pathological Anatomy Forensic Medicine, Andijan State Medical Institute, Andijan. Uzbekistan.
- © МАМАТАЛИЕВА М.А. – senior lecturer of the Department of Pathological anatomy forensic medicine, Andijan State Medical Institute, Andijan. Uzbekistan.