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PATHOMORPHOLOGY OF THE CARDIAC TRACT IN ACCIDENTAL MORTALITY OF INFANTS

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Abstract
Morphological study showed that the activity of fibroblasts in cardiac fibrosis in children born to mothers with autoimmune diseases, rheumatism, allergic diseases, resorption of surrounding cardiomyocytes, degeneration of the cardiac pathways, changes as a result of accidental death of children.

Keywords: accidental death, autopsy, respiratory infection, Q-T interval.

Резюме: Морфологик текшириш шуни кўрсатдики, онаси аутоиммун касаллик, ревматизм, аллергик касалликлар билан касалланганлардан туғилган болалар юраги ўтказувчи йўлларида юрак фиброз танасида фибробластларнинг фаоллашганлиги, улар атрофидаги кардиомиоцитларнинг резорбцияга учраганлиги юрак ўтказувчи йўлларининг дегенерацияси, блокланиши оқибатида болаларнинг тасодифий ўлимига хос ўзгаришлар ривожланганлиги кузатилди.

Калит сўзлари: тасодифий ўлим, аутопсия, респиратор инфекция, Q-T интервал.

Резюме: Морфологическое исследование показало, что активность фибробластов при фиброзе сердца у детей, рожденных от матерей с аутоиммунными заболеваниями, ревматизмом, аллергическими заболеваниями, резорбцией окружающих кардиомиоцитов, дегенерацией сердечных путей, изменениями в результате случайной смерти детей.

Ключевые слова: смерть от несчастного случая, вскрытие, респираторная инфекция, интервал Q-T.

The urgency of the problem

For at least two hundred years, the causes of infant deaths have remained a mystery to pediatricians. It is most common in infants up to 6 months of age, and is not clinically evident, or with autopsy changes. Accidental infant mortality was first reported in 1892 by C. Templeman. In 1969, at the II International Conference in Seattle, this terms proposed by J.B. Beckwith (1), accidental death has been termed "accidental death of children whose causes cannot be determined".

In 1979, the syndrome of accidental death of children was included in the International Classification of Diseases with a rubric of 798.0, and in 1995 it was included in the 18th grade as an accidental death of children of breast age R-95. Nowadays, pediatric accidental death syndrome is understood in which the child has no symptoms before death, the child dies in his sleep, often in a tie, and even when examined by autopsy, no specific changes are found (2, 3, 4, 5, 6).

Pediatric accidental death syndrome the incidence rate varies from country to country. In developed countries, where infant mortality is low, i.e., up to 20 deaths per 1,000 infants, pediatric accidental death syndrome is relatively high and ranks first among the causes of infant mortality (7, 8, 9). The world average is 2 per 1,000 babies: 4.4 out of 1,000 babies die in Israel, 1.6 in France, 0.57 in Italy, 1.3 in Germany, 2.3 in the UK and 2.8 in the United States. will be. In the territory of the former USSR, the epidemiology of child accidental death syndrome is poorly understood and it is often replaced by respiratory infection and intestinal infection. Tsinzerling A.V. et al. According to (1989), in recent years in some regions of Russia the incidence of child accidental death syndrome has risen sharply: Saratov region ranks first, 21.8 per 1,000 live births, Ulyanovsk region - 19.8, Astrakhan - 19.1. 18.2 in Volgograd and 17.2 in Tatarstan. To date, several theories on the causes of accidental death syndrome have been proposed in the scientific literature, of which immunological, thymic, infectious, and cardiogenic theories were recognized in the 1960s and 1970s. Pediatric accidental death syndrome The cardiac theory is currently the most well-founded theory, suggesting that sudden death results from slowing ventricular repolarization, ventricular fibrillation due to asystole, and lengthening of the Q-T interval on the ECG. In children under one year of age, the fact that the QT interval on the ECG is long, especially at 2-4 months, and that there is a high risk of death during this period confirms the above idea (10, 11). may also occur due to the slow development of its activity and the activation of the sympathetic nerve that gives the arrhythmia. T.M. James was the first to suggest that children accidental death syndrome has a side effect on the conduction pathways. As James (2001) noted, in the fetal period of the fetus and in the early postnatal period, the tuft of conduction pathways in the area of

the atrioventricular node of the heart is rough and torn, leading to "resorptive degeneration" after birth. That is, the connective tissue around the special conductive tissue of the atrioventricular node ends with the repair of the growing connective tissue. Such changes have been confirmed by other scientists, who have confirmed that such sclerosis of the special tissue of the conduction pathways is a morphological substrate of life-threatening cardiac arrhythmias (12). So far, there is no single-minded theory about the causes of Child Accidental Death Syndrome. Risk factors, developmental mechanisms, direct causes of death, preventive measures have not been developed. In view of the above, the aim of this study was to identify morphological changes in the conduction pathways of children who died prematurely at the age of one year.

Materials and methods

To achieve the purpose of the study, all materials were studied in two groups: 1st group included children who died accidentally in the period under one year of age; Group 2 included children who died of other diseases as a control group. In both groups, children's medical history, illnesses and negative habits experienced by their mothers during pregnancy were recorded. During the autopsy examination, the heart was separated from the body of the deceased child and initially the appearance, structural condition of the compartment and ventricles were studied. The cardiac incision was made taking into account the topography of the conduction pathways, and the areas where the sinus and atrioventricular nodes were located were extensively incised. For microscopic examination, cardiac fragments were solidified in a 10% phosphate buffer solution of formalin for 2 days. After washing for 4 hours in running water, it was dehydrated in concentrated alcohols, then passed through chloroform and poured into paraffin. Histological sections were prepared from them and stained in hematoxylin-eosin for general microscopy, in the Van-Gizon method for the detection of connective fibers, and in the SHIK reaction to show mucopolysaccharides. Histological preparations were studied under a light microscope by Leyka, and data-rich areas were photographed.

Results and their discussion

The results of the study showed that 90% of children who die from pediatric accidental death syndrome occur in the first 6 months, with the peak period of death being 2-4 months. 72% of the children who died were boys. Children accidental death syndrome the risk factors for low birth weight by a child were less than 70%, premature birth, and excessive sweating while the baby was asleep. It was determined by the mother

that under 20 years of age, unmarried, smokers and drinkers during pregnancy, extragenital diseases of the mother, severe forms of geztosis are important. From extragenital diseases it was observed that children accidental death syndrome is caused by autoimmune diseases, infections, blood diseases. Infant mortality in most cases (82.4%) occurs at night, i.e. in the morning, from 4 to 6 p.m. The rate of accidental infant mortality has been observed to increase in the fall and winter seasons. Clinically, pathological examination of children diagnosed with accidental death syndrome revealed congenital defects, meningitis, sepsis, subendocardial fibrosis as the causes of death in 15% of them; in the remaining 85% of cases, the pathomorphological changes were the same, recorded by many scientists, i.e. moderate cyanosis in children with normal appearance, enlargement of the lungs due to swelling and fullness, petechial hemorrhages in the pleura, epicardium, thymus, and meninges. Microscopic examination of the internal organs revealed the presence of only circulatory and superficial dystrophic changes. This means that pathomorphological changes that cause death in the internal organs have not developed. Researchers have found that in some cases there is an inflammatory process in the lungs, in others there is a predominance of circulatory changes, and in other cases there is a viral infection (13). Some scientists have confirmed the idea that tickling the receptors in the mucous membranes of the respiratory tract leads to acute respiratory failure and sudden death (8, 10, 11). Because viral infections have a cardiotropic nature toward the heart, they have been found to result in the development of severe myocarditis and endocarditis in the heart. Microscopically, there was a sharp dilation of blood vessels in the heart tissue, swelling of the endothelium, vacuolation of myocytes and myolysis. The presence of such a large number of foci of necrosis, the appearance of infiltrates from lymphocytes, histiocytes around them, indicates the development of a viral infection (3). The presence of destructive changes in the heart of infants with viral infection, not only damage to the working cardiomyocytes, but also in the cytoplasm of atypical myocytes, which leads to disruption of cardiac function, leading to arrhythmias and even accidental death. Among the study materials, dilated cardiomyopathy was identified in 2 cases, mainly in which the right ventricle of the heart was dilated and dilated, leading to death due to insufficiency. According to the literature, cardiac arrhythmias in dilated cardiomyopathy are fatal. Microscopic examination of the conduction pathways revealed that resorptive degeneration of the atrioventricular node and GIS tumor in the postnatal period occurs in two different ways: associated with embryonic dispersion (11), while in the second completed morphogenesis, partial or complete fibrosis of the myocytes of the AVT and GIS tufts was found, leading to blockade of

cardiac activity. These two types of early morphogenetic anomalies are the leading causes of accidental and accidental death. When the heart of children born with rheumatic diseases and women with systemic lupus erythematosus was pathomorphologically examined, it was found that the central fibrous body fibroblasts are actively proliferating, causing excessive resorption of conductive myocytes. In some cases, embryonic and postnatal fibrosis in AVT and GIS tumors may develop chronically and persistently. It was observed that a number of anomalies appeared in the conduction pathways of the heart. Morphologically conductive pathways have been found in the formation of numerous thick and small tufts of various myocyte cells in the AVT and GIS bundles, and central fibrosis is widespread throughout the body. Sometimes the proximal part of the AVT is connected to the distal end by rings. In other cases, the AVT was found to be disconnected from the GIS bundle, which is functionally manifested by the appearance of a parasystolic rhythm. In another variant, it was found that the tissue of the conducting pathways was connected to the interstitial part by a Gis tuft by bypassing the AVT. In fact, postnatal fibrous degeneration of AVT and GIS tumors is a process that takes place under precise control, and their formation and development can be pathologically disrupted as a result of various pathological effects, culminating in a number of destructive changes. Although central fibrosis controls fibroblasts in the body by receptors, glycoproteins, and other chemical components on these cell membranes, in some cases these controls can become highly aggressive and activate fibroblasts (11, 12). Such uncontrolled fibroplasia can occur at any stage of a person's life, which in turn can lead to accidental death from the heart. Thus, fibroblasts in the central fibrous body are sometimes plyuropotent in nature, developing metaplasia around the cardiac pathways, central fibrosis in the body, interventricular fibrous membrane: fibromatosis, chondromatosis, and even osteomatosis, leading to sudden cardiac death (11, 13).

In Conclusion

It can be said that the heart is the most important organ in the human body. . Morphological changes in the conduction pathways, such as fibroplasia developed for various reasons, can lead to accidental death from the heart, leading to impaired conduction, slow repolarization in the myocardium, the development of asystole and fibrillation in the ventricles. Once the presence of conduction pathways in the heart was identified, it was confirmed that the cause of accidental death syndrome could be the conduction pathways of the heart, once its anatomotopographic, histological, and functional aspects were revealed. Another novelty identified by pathomorphologists

has unintentionally clarified the mechanism of development of death syndrome, i.e., postnatal resorptive degeneration of AVT and GIS tumors is in fact a normative process, resulting in unclear, embryonic AVT and GIS tumors with clearly defined boundaries. become fully developed structures. During the development of this process, for various reasons that have not yet been fully explored, central fibrous body fibroblast cells degenerate on the basis of pathological fibrosis by proliferating towards specific myocytes and causing functionally different levels of blockages, these changes were detected in accidental cardiac deaths. Nowadays, the syndrome of accidental death of children is understood in such a way that before death the child has no symptoms of the disease, the child dies in bed, often in a tie, and even when examined by autopsy, no specific changes are found. Ventricular repolarization, asystole, ventricular fibrillation, which is one of the leading causes of death, are directly related to resorptive degeneration of the conduction pathways and are clinically manifested by prolongation of the Q-T interval on the ECG.

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