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Cases of Adolescent Sudden Cardiac Death During Physical Exercise: Autopsy, Histological Findings

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ABSTRACT

According to experts' recommendation unexpected death was defined as sudden cardiac death (SCD) due to cardiac causes that occurs in a short time period within 1 hour of acute symptoms onset in person with known or unknown cardiac vascular disease. It was later suggested that sudden death be defined as a natural death occurring unexpectedly within 24 hours of the onset of acute symptoms. The most common cause of cardiac arrest is coronary artery disease. Less common causes include major blood loss, lack of oxygen, very low potassium, heart failure and intense physical exercise. This report about of two autopsy cases of sudden adolescent cardiac death during physical education classes. As a result of strenuous muscular load and inadequate oxygen supply, myocardial hypoperfusion, electrical instability, and left ventricular dysfunction occurred.

Keywords: Sudden cardiac death, physical exercise, adolescent death, autopsy, histological findings

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INTRODUCTION

Hidden and unrecognized heart disease is the most common cause of sudden death. The term "sudden cardiac death" has been customarily used to denote the case of death of a person previously in a physiologically and psychologically stable state, whose death occurred between 1-2 minutes and 24 hours from the appearance of the first symptoms, due to cardiac arrest amid sudden asystole or ventricular fibrillation, with no other clinical signs to indicate an alternative diagnosis [1]. Sudden cardiac death is a collective concept that integrates several nosological units that cause different forms of heart pathology [2]. At present, the risk of sudden adolescent death in physical activities and sports has become an increasing concern to specialists, including forensic experts. Liberthson (1996) stated that 20% of sudden deaths among young people occur during sporting exercise [3]. The official definition of "sudden death in sports" refers to deaths occurring directly during physical exertion, and those occurring within 1-24 hours of the appearance of the first symptoms [4]. Among all cases of adolescent death, "sudden death" accounts for approximately 13% [4]. In analyzing cases of sudden death during exercise, it is essential to consider any underlying pathology within the cardiovascular system, which is the most common cause of sudden death syndrome in adolescents [5, 6].

Statistics indicate that between 6 and 12 young people die suddenly during physical education classes every year in Russian schools due to sudden cardiac arrest [4]. The equivalent statistical information is not currently available in the Republic of Kazakhstan. Predisposing factors in sudden cardiac death, contributing to the emergence of life-threatening conditions, can be due to either congenital or acquired cardiovascular system pathology. In acquired cardiovascular pathology underlying the development of sudden cardiac death syndrome, consideration needs to be given to the role of the inflammatory processes within the myocardium and the endocardium, with the possible subsequent formation of valve defects and cardiomyopathies. In 20% to 40% of cases, sudden cardiac death in children has been observed against a background of acute myocarditis [7]. Acute myocarditis in 80% of cases may cause ventricular fibrillation, provoked by ventricular tachycardia, and less frequently by bradycardia and asystole [8, 9]. We report the autopsy of two cases of the adolescent's death in physical education classes at school: of 13-year-old teenage girl and a 16-year-old teenage boy.

MATERIALS AND METHODS

The objects of the study were two deceased teenagers (13, 16 years old), whose death occurred during a physical education lesson at school. Death has occurred suddenly after a run. According to the teacher, the children were healthy. We did not have any information about the past medical history of children. The only thing that was known was that a 16-year-old teenager had a Ramadan fast in that period. Autopsies were performed at the Institute of Forensic Examinations, a branch of the Center for Forensic Examinations of the Ministry of Justice of the Republic of Kazakhstan, because the deaths of teenagers were unexpected, inexplicable and were requested by official death investigator. Autopsies were sequential structured examinations. They were specifically address the major causes of extra-cardiac and cardiac death. All aspects of the autopsy procedure were adhered by to the Recommendations on the Harmonisation of Medico-Legal Autopsy Rules produced by the Committee of Ministers of the Council of Europe [10]. During the autopsy non-cardiac causes of death were excluded. After excluding non-cardiac causes sudden death have been considered cardiac in origin. The heart was examined according to Guidelines for autopsy investigation of sudden cardiac death [11]. A histological examination of the heart was also performed. Histological preparations were staining according to Amineva and Mitrofanova's methodological manual [12] and with iron hematoxylin according to Rego [13].

RESULTS AND DISCUSSION

The autopsy of 13-year-old teenage girl showed an increase in the size of the heart, measuring 11 x 9.5 x 5.5 cm (heart size for this age is normally estimated to be 6.5 x 7.5 x 3 cm), moderate hypertrophy of the left ventricular wall, with left ventricular wall thickness of 1.1 cm (normal thickness, 0.8 cm), a right ventricular wall thickness of 0.3 cm (normal thickness, 0.2 cm) in the measured section, and the muscle of the heart demonstrating uneven blood filling. Histological examination of heart preparations revealed focal vascular-stromal myocardial lipomatosis, with the

appearance of adipose tissue around intramural vessels and between muscle fibers. This was considered to be hypoxic-ischemic myocardial damage showing chronic characteristics (Fig. 1a, b).

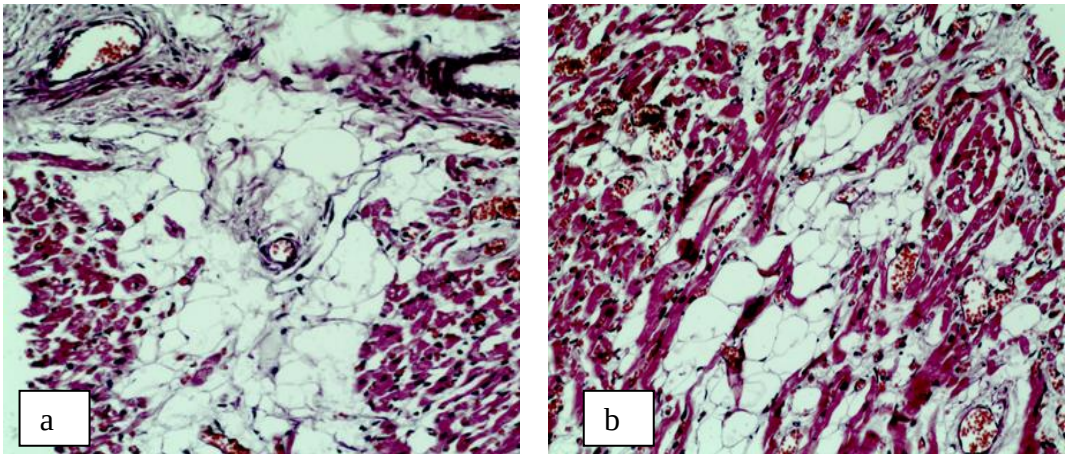

Figure 1 (a, b). Vascular-stromal myocardial lipomatosis, as a manifestation of chronic hypoxic-ischemic changes in the myocardium, stained using hematoxylin and eosin (H & E), x200

Changes in myocardial muscle fibers in the form of separate scattered foci of atrophied thinned muscle fibers, accompanied with diffuse interstitial mesh and perivascular sclerosis, also also appeared to demonstrate the chronic nature of the condition. The myocardial interstitium was diffuse and focally infiltrated with round-celled elements (Figure 2a, b).

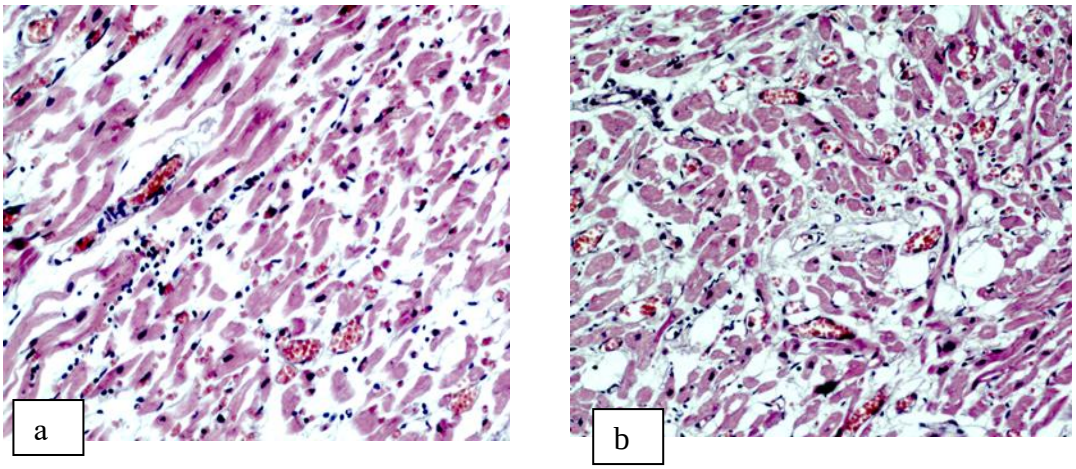

Figure 2 (a, b). Diffuse and focal infiltration of myocardial interstitium by lymphocytes, H & E stain, x200

The most severe changes in the myocardium were manifested in the form of diffuse and focal fragmentation of muscle fibers without preservation of basal membranes, dissociation with preservation of the continuity of the basal membranes, and contractures of muscle fibers of varying severity (Fig. 3a, b).

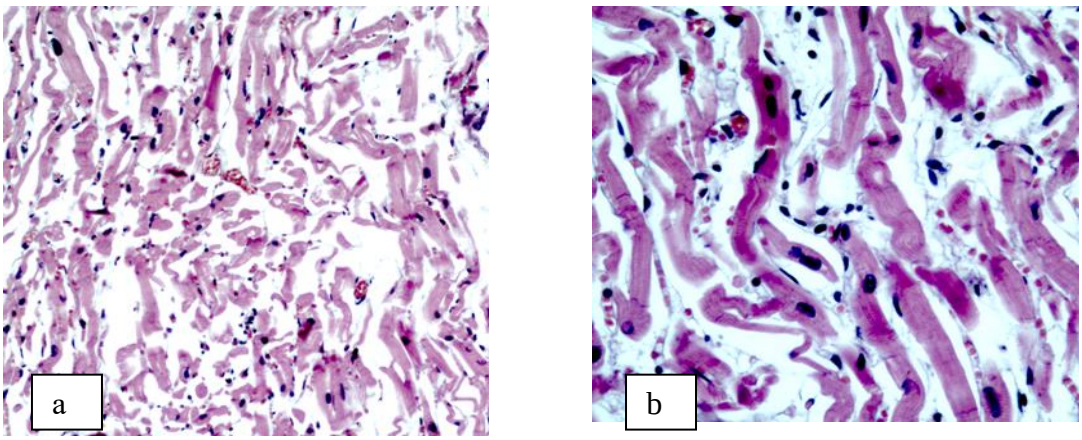

Figure 3 (a, b). Contractures of varying severity, fragmentation and dissociation of the muscle fibers in the myocardium with interstitial myocarditis in the background, H & E stain, a - x200; b - x400

In the study of intramural nerve fibers, areas of inflammatory round-cell infiltration were detected (Figure 4 a, b).

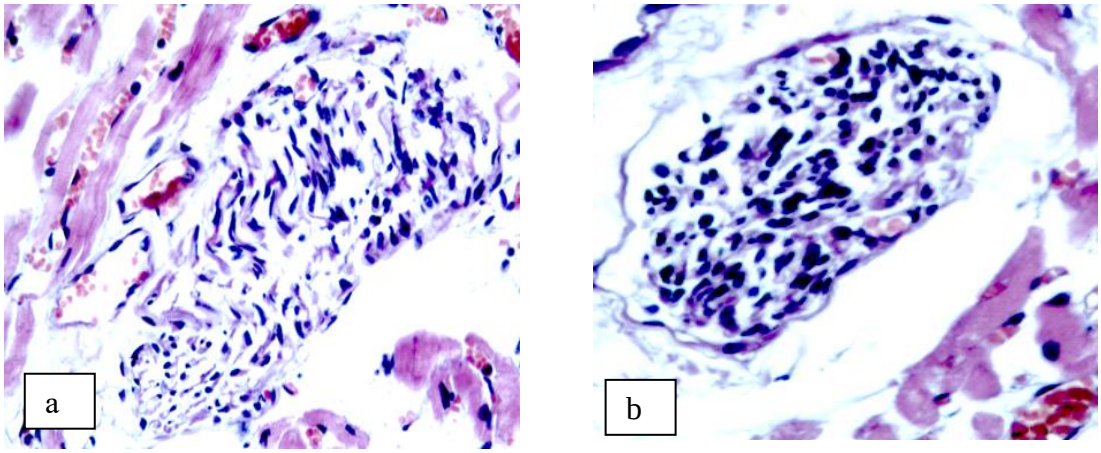

Figure 4 (a, b). Infiltration of the round-cell cells of intramural nerve fibers, H & E stain, x400

Ischemic muscle fibers were of a scattered, disseminated nature when stained with iron hematoxylin according to Rego, and the sarcoplasm of only one part of the cardiomyocyte sarcomeres was stained in black or in a dark gray color (Figure 5 a, b).

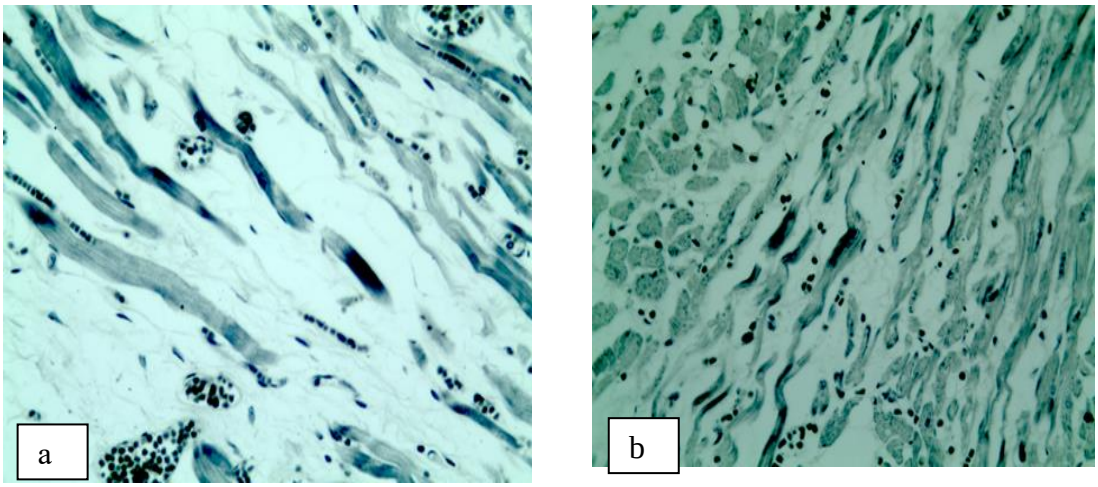

Figure 5 (a, b) - Ischemic muscle fiber, with staining only of individual sarcomeres of cardiomyocytes, staining according to Rego, a - x 200.

In this case, chronic serous myocarditis with diffuse lymphocytic inflammatory infiltration was identified, with initial fibrosis (grade 1) according to the Marburg classification (1997) [14], complicated with cardiac arrhythmia, following total fibrillation of the myocardium, and histologically manifesting as diffuse fragmentation, dissociation and contractures of varying severity, being the immediate cause of death.

The second case: autopsy of 16-year-old teenage boy showed an increase in the size of the heart of 9.5 x 9.0 x 6.5 cm, as compared to measurement values within the normal range for this age of 6.5 x 7.5 x 3 cm, moderate hypertrophy of the left ventricular wall of 1.2 cm (normal thickness, 0.8 cm), the right ventricle measuring 0.3 cm (normal thickness, 0.2 cm), and moderate mainly left-sided hypertrophy of the walls of the coronary arteries. In the section, the autopsy showed the unevenness of the blood filling the myocardium in the region at the base of the anterior wall of the left ventricle of the heart in an oval shaped area of 1.5 x 0.8 cm, and the heart muscle appeared pale brown along the periphery, with small dotted dark-red hemorrhages that engulfed the muscular layer and the endocardium.

In the histological examination of heart preparations, focal sclerosis, more pronounced in subendocardial areas, was regarded as the result of chronic hypoxic ischemic lesions, with replacement of ischemic cardiomyocytes due to proliferation of connective tissue (Fig. 6 a, b). Additionally, there was a diffuse proliferation of the connective tissue both inside the myocardial interstitium and around the vessels. Small branches of the coronary arteries showing unevenly thickened walls due to proliferation of connective tissue under the intima were observed.

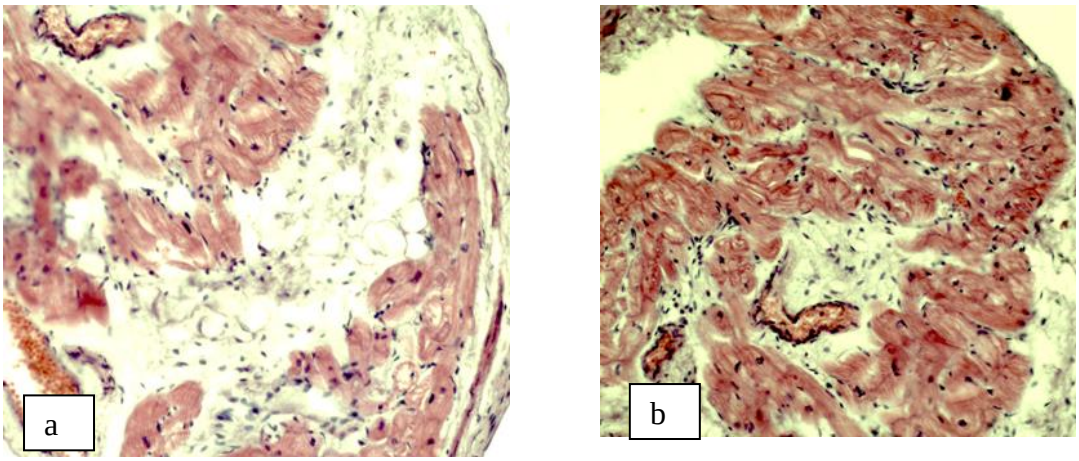

Figure 6 (a, b). Sites of subendocardial focal cardiosclerosis, H & E stain, a-x200

With reference to the acute manifestations, widespread pronounced perimuscular and perivascular myocardial edema was prevalent, regarded as a manifestation of plasmorrhagia, and as a result of a circulatory disorder. In addition, there were violations of rheological properties of blood in the form of erythrostasis and erythrocyte sludge, not only in the tissue but in all other internal organs. Uneven blood filling of the vessels of the microcirculatory bed of the cardiac muscle was noted as one of the manifestations of ischemia (Fig. 7a, b).

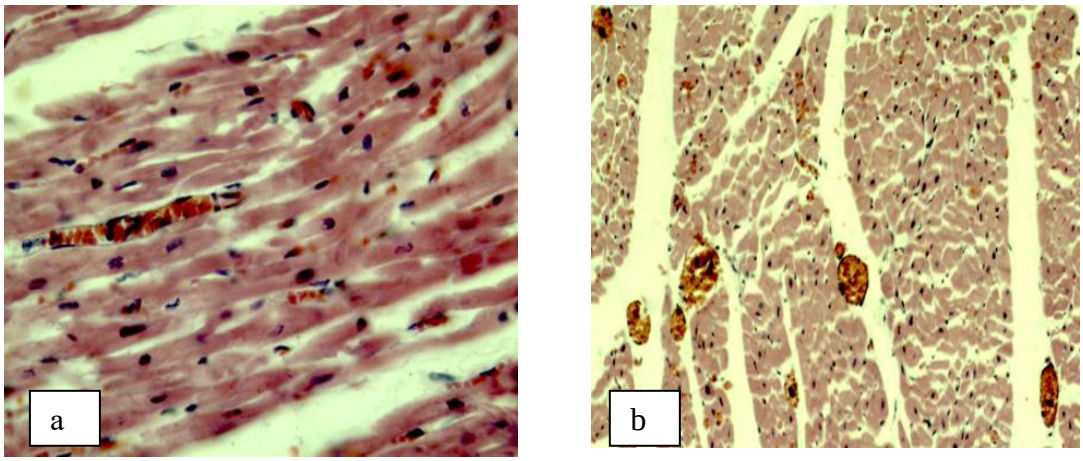

Figure 7 (a, b). a - Erythrostasis in the intramural capillaries, H & E stain x400; b - Erythrocyte sludge in the vessels of the microcirculatory bed, H & E stain, x200

The most severe focal changes to the myocardium, identified as having a dystrophic and necrobiotic character in the staining with hematoxylin and eosin, were more frequent near the endocardium. When viewing the subendocardial zones of the myocardium, cardiomyocytes with uneven coloration of the cytoplasm with alternating dense and light areas, disappearance of the sarcoplasm (lumpy decomposition of myofibrils) in some places, and areas of karyorrhexis and karyolysis (Fig. 8a, b) were observed.

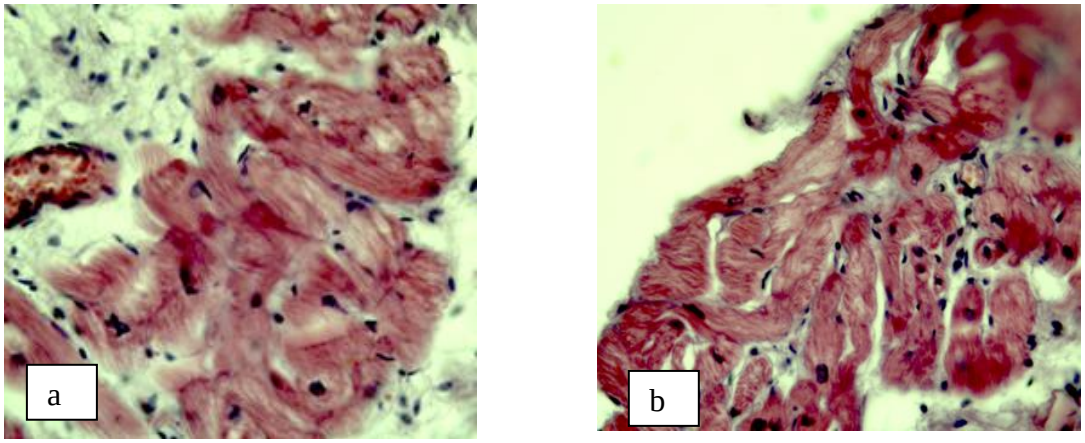

Figure 8 (a, b) - Lumpy decomposition of the cytoplasm, H & E stain, x200

Morphologically, ischemic muscle fibers stained with iron hematoxylin, according to Rego, were small-focal and diffuse, staining the sarcoplasm, most often of the cardiomyocyte, in either black or dark gray. The changes were more pronounced in the subendocardial zone and inside the myocardium Fig. 9 (a, b).

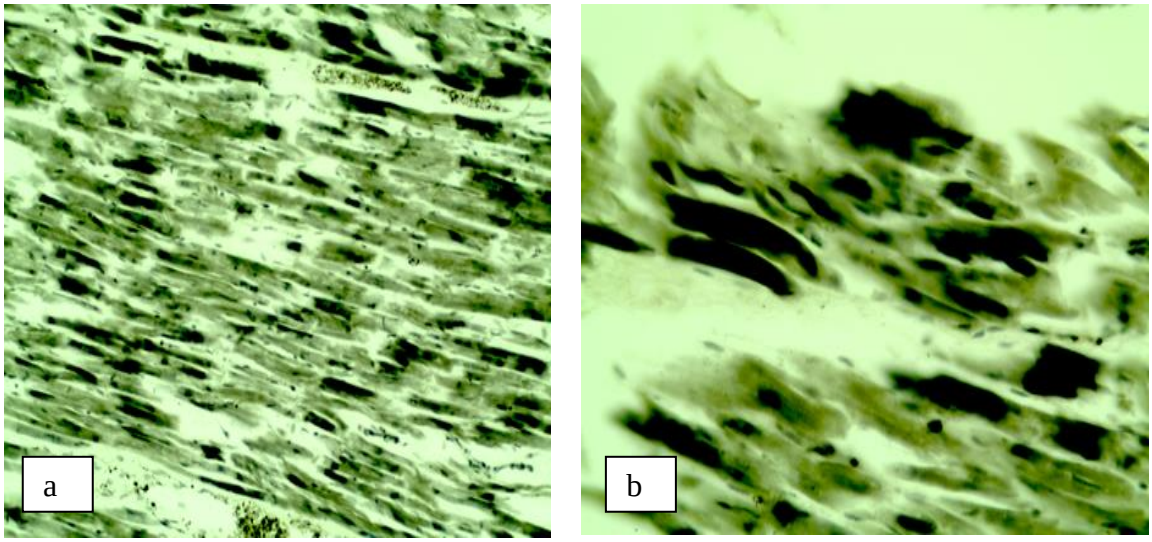

Figure 9 (a, b) - Staining in black or dark gray of the sarcoplasm of the cardiomyocyte entirely, staining according to Rego, x200

There were focal contracture lesions of the myocardium (Fig. 10).

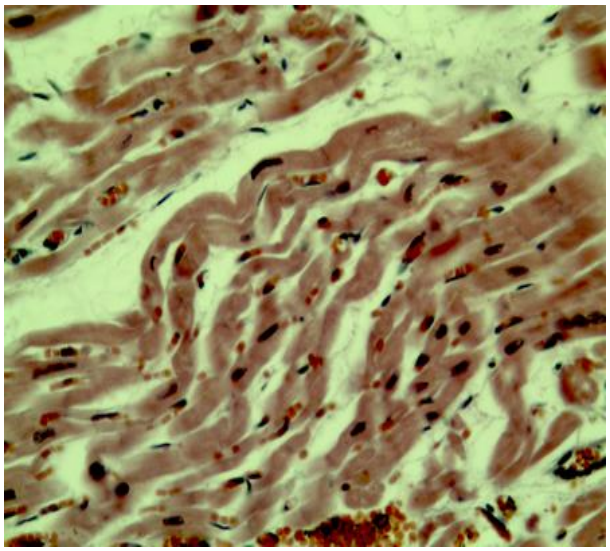

Figure 10. Focal contractures of myocardial muscle fibers, H & E stain, x200

In the study of intramural nerve fibers, demyelination was detected (Fig. 11a), and dystrophic changes in the form of vacuolization of nerve fibers, lumpy decomposition of nuclei and myoplasm, areas of inflammatory round-cell reaction, and necrosis of individual nerve fibers with the phenomena of karyorrhexis and karyolysis of nuclei (Fig. 11b) were observed.

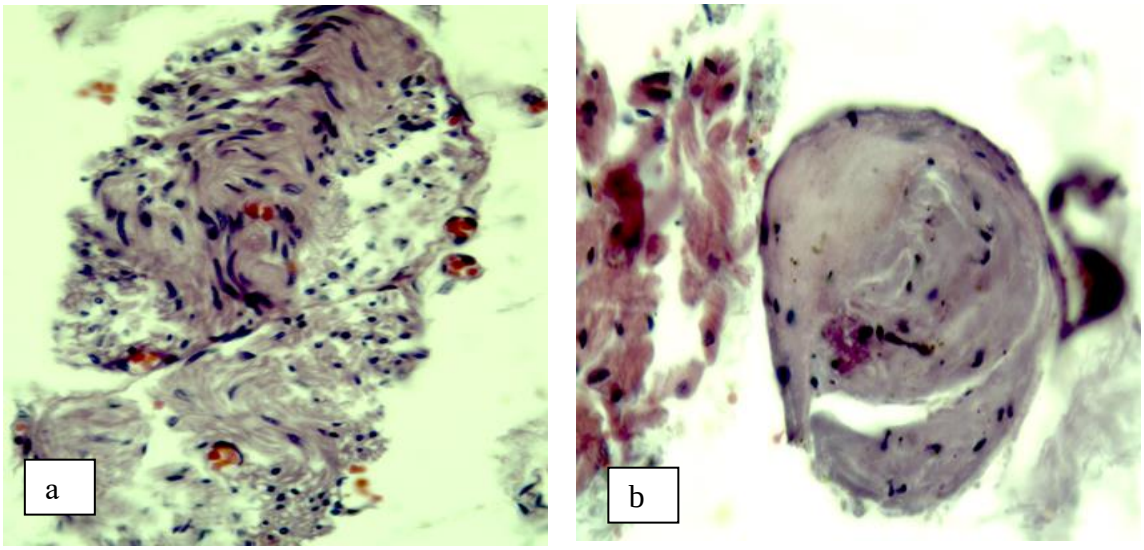

Figure 11 (a, b). a - Cross-section of the nerve trunk with demyelination, signs of dystrophy and inflammatory lymphocytic infiltration, H & E stain, x400; b - Intramural nerve trunk with signs of necrosis, H & E stain, x400

Based on the study results, the cause of death was identified as acute subendocardial myocardial infarction of the anterior wall of the left ventricle of the heart, at a pre-eclampsia stage. Throughout life adolescent suffered from coronary heart disease, namely, stenosing coronary atherosclerosis and diffuse small-focal cardiosclerosis.

CONCLUSION

Ischemic myocardial lesions can be the cause of sudden death syndrome in children due to insufficient oxygen supply to certain parts of the myocardium, electrical instability, and left ventricular dysfunction due to excessive physical exertion.

Chronic hypoxic-ischemic changes in the myocardium in these two autopsy cases acted as provocative, aggravating, and complicating factors.

Because of intense physical muscular load and due to inadequate coronary blood flow, myocardial hypoperfusion arose which, in one case, led to fibrillation and, in another case, led to ischemic myocardial damage.

Objects of forensic medical examination in the form of incoming corpses of adolescent children should be more thoroughly examined for the presence of pathology in the cardiovascular system and be supplemented with anamnestic information.

Even a healthy child or adolescent needs an individualized approach to physical activity training, with physical loads adapted to the child's body, and taking into account the child's state of health and the possibility of hidden pathology.

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