Pathomorphology of Myocardial Muscle Fibers in Covid-19

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ABSTRACT

This article covers the morphological features of pathological processes that develop in the myocardium muscle fibers during COVID-19 disease. As a Material, 36 deaths from COVID-19 were examined in autopsy of the cardiac myocardium macro - and microscopic. The results show that it has been confirmed that various pathomorphological deviations may develop as a result of damage to the heart with the virus. As a result of the direct influence of the SARS-CoV-2 virus on the myocardium, the development of all common pathological processes in the heart tissue, the procoaguyant and prothrombogenic effect of inflammation developed under the influence of the virus, myocardial ischemia from insufficient oxygenation caused by microtomyr dysfunction, inflammation of hypersensitivity as a result of an attack. . In the material we studied, vacuolar and fatty dystrophy of cardiomyocytes in myocardial muscle fibers under the influence of the SARS-CoV-2 virus, chaos hypertrophy characteristic of cardiomyopathy of muscle fibers, deformity and contracture of muscle fibers in some cases, fragmentalization, breakdown in other cases, and ultimately necrobiosis and local necrosis of cardiomyocytes were observed

Keywords: COVID - 19, virus-SARS-CoV-2, Heart, myocardium, muscle fibers, cardiomyositis, dystrophy, destruction, fragmentation, hypertrophy, necrobiosis, necrosis.

Relevance of the problem.

In COVID-19, heart myocardial damage is diagnosed based on the amount of troponin in the blood and the result of an ECG or ExoKG. According to data from various authors, myocardial damage occurs from 7% to 27.8%. As a result of the examination of Chinese scientists, COVID-19 causes bad consequences in the cardiovascular system (3, 4). At the same time, the mechanism of myocardial damage in COVID-19 disease remains unknown. In COVID-19, diseases of the cardiovascular system are often manifested by myocarditis, Takosubo cardiomyopathy, acute coronary syndrome, arrhythmia, accidental death from the heart, venous thromboembolism and acute heart failure. The direct effect of SARS-CoV-2 on the myocardium, damage through an angiotensin-converting enzyme in the heart tissue, procoaguyant and prothrombogenic effect of inflammation developed under the influence of the virus, myocardial ischemia from insufficient oxygenation caused by microtomyr dysfunction, inflammation of extreme sensitivity as a result of an attack by cytokines are being studied (2, 7). In a study by some scientists, virus particles were found in myocardial interstitial tissue (6). But, when histological examination of the myocardium,

lymphsitar infiltration characteristic of viral myocarditis was not found (5,6).

The degree of expression of apf2 varies in different tissues and cells. With more lung damage, APF2 expression is superior in myocardium. Including the expression of the enzyme APF2 is more pronounced in the pericitarian cells of blood vessels. Damage to periscites provokes deorganization in the capillary wall, including the endothelium, leading to acute damage and necrosis of cardiomyocytes (1, 3).

Material and methods.

During the 2021 pandemic, the dead from COVID-19 at the Republican Center for pathological anatomy of the SSV of the USSR, during an autopsy examination, fragments were taken from the heart myocardium and hardened for 48 hours in a 10% solution of formalin dissolved in a phosphate buffer. It was washed for 3-4 hours in running water, and then dehydrated in alcohols with increased concentration, and paraffin with the addition of vosk was poured and bricks were made. Histological incisions 5-6 μm thick were obtained from paraffin bricks and painted in hematoxylin and eosin after deparaphinization. Histological preparations light microscopio stida 10, 20, 40 were examined and studied in lenses, pictures were taken from the desired places and transferred to a computer.

When the heart was studied macroscopically, in most cases, a state of cardiomegaly was detected, the wall of the right ventricle of the heart was dilated, in some cases a very strong dilation was observed, and the right ventricular space expanded by 3.6 cm compared to the left ventricle. It was found that there is liquid blood in the cavity of the coronary arteries of the heart, the artery cavity is sharply narrowed in the thin part, that is, in the area of the arteriola braid. In two cases, the presence of microthrombi consisting of a whitish-colored fibrin mass was found in small branches of the crown arteries.

Under microscopic control, the rocket launcher represents a highly specialized, dysregenerative, deformational and necrotic musculoskeletal ability. 36 cases of COVID-19 Dan wilganlar heart myocardium histological school, collectively pathomorphological group.

Table 1.

The degree of occurrence of dystrophic, dysregenerator, deformation and necrobiotic changes in myocardial muscle fibers, %

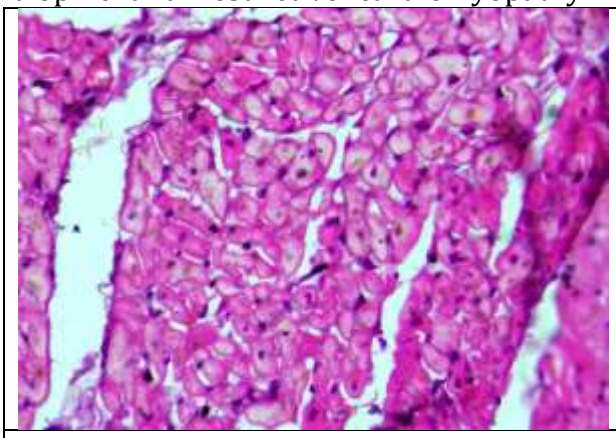
№	Type of pathomorphological changes	Percentage , %
1	vacuolar dystrophy of muscle fibers	24,6
2	fatty dystrophy of muscle fibers	14,8
3	hypertrophy of muscle fibers to varying degrees	16,7
4	deformation and contracture of muscle fibers	35,6
5	fragmenting and breaking of muscle fibers	27,3
6	myolysis and necrobiosis of muscle fibers	41,5

From the pathomorphological changes that the heart myocardium develops in muscle fibers under the influence of COVID-19, hydropical, that is, vacuolar dystrophy of the cardiomyocytes sarcoplasm has been detected relatively much. In this case, it is found that the myocardial intermediate tissue is strongly swollen, as a result of which muscle fibers are flipped. The area around the core of muscle fibers, that is, the sarcoplasm, is swollen, vacuolated, discolored and pale in appearance (fig. The nuclei are set aside and deformed to reveal a condition of cariopychnosis and cariolysis. It is observed that in the sarcoplasm of cardiomyocytes of certain muscle fibers, a pigment of lipofuscin of brown color accumulates.

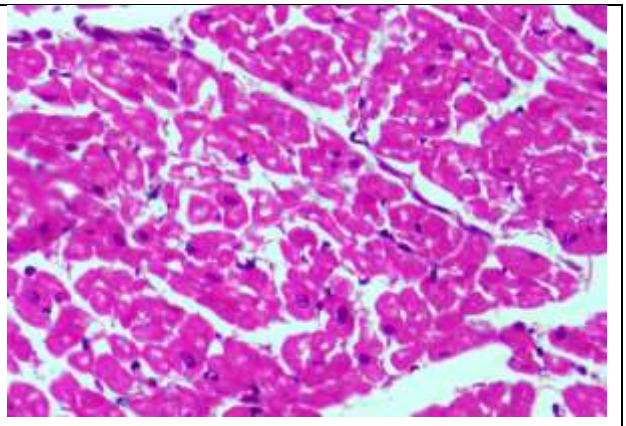
Due to a violation of the metabolism of all substances in the myocardial tissue under the influence of the SARS-CoV-2 virus, the development of fine vascular dyscirculation and hypoxia, lipoprotein membranes in the sarcoplasm of cardiomyocyte cells undergo peroxide oxidation under the influence of

active radicals of oxygen, decompose, and the fat content accumulates separately, causing the development of parenchymatosis fatty dystrophy (fig. As a result, morphologically, in the sarcoplasm of cardiomyocytes, the appearance of initially small, then large-droplet oviduct is observed. This process was determined in our material in 14.8% of cases. It is determined that the sarcoplasm of the muscle fibers from which the fattomers appeared is discolored, the sarcomeres are thickened, deformed and darkly stained with eosin.

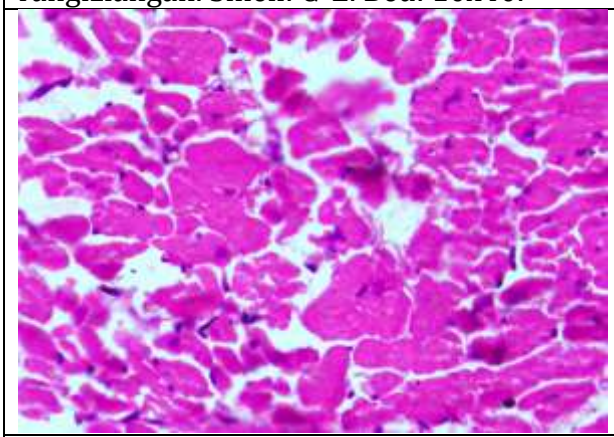
According to the Agency for Disease Control and Prevention, COVID-19 viral liver disease muscle myocardium tolalarida both hypertrophic and restrictive cardiomyopathy typical uzlanganliga and bu condition materializda 16.7% da wchraganligialliglanlanlanadi exactly. Widely known histological myocardium, myofibrillary muscle, keskinskaya musculature, tolstolarga is comparatively 3-4 times larger, myofibrillary sarcomellar, coarse-acid and long-lasting. Cardiomyocytlar muscle core tolasing chetiga surilganligi and karyopycnosis holatga kyrgyzanligi tochlanadi. Bundai kalinlashib, sahta holda hypertrophy langan musculature tolalari joylashgan sahada interstitial long as kuchli stekga uchraganligi topilada. Myocardning sihalarid tolalari rocket is relatively small and the belgilarin histotopograph belgilaring is exactly the carpet.



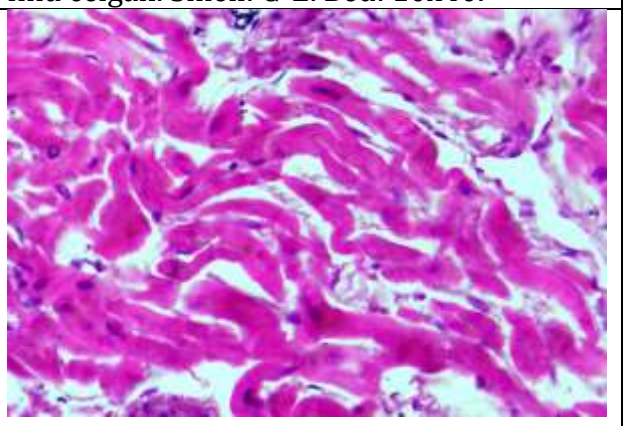
1-picture. Myocardium musculature tolalari sarcoplasmas vacuollashib, rangizlangan. Smell: G-E. Bed: 10x40.



2-picture. Myocardium musculature tolalari sarcoplasmacid small NEEDLE tomchilari find celgan. Smell: G-E. Bed: 10x40.



Picture-3. Some of the myocardial muscle fibers became sharply thickened, hypertrophied and entered the sign of cardiomyopathy. Paint: G-E. Ass: 10x40.

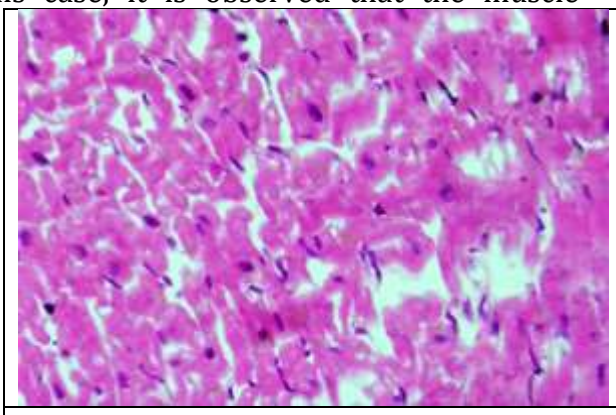


4-Picture. Interstitial craniocerebral myocardial deformity. Binet Eaton: Mr. Letter: 10x40.

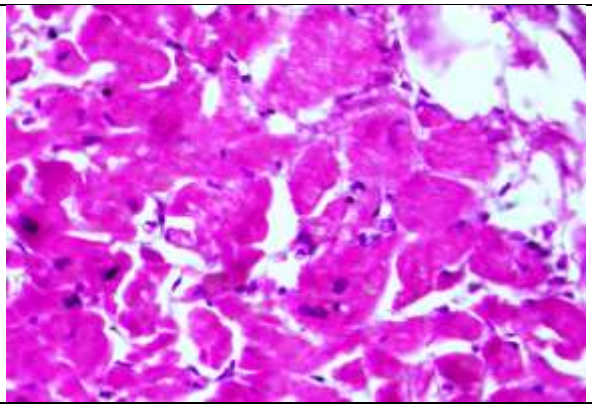
When dyscirculation and edema prevail in some patients due to the toxic effects of the SARS-CoV-2 virus on the myocardium, in others parenchymatosis is vacuolar, in others parenchymatosis fatty dystrophy, when changes characteristic of cardiomyopathy have

developed, in most cases, that is, at 35.7%, the deformation and contracture of muscle fibers predominates (fig. In this case, microscopically, there is a strong swelling in the intermediate tissue, deformation of muscle fibers, changes in thickness to varying degrees. In the myofibrils of deformed muscle fibers, sarcomeres are not detected, it is determined that the myofibrils homogenize and lose their histotopographic properties. It is found that the ends of the muscle fibers are blunt and undergo myolysis. It is determined that the nuclei also changed in different degrees and manifestations, that is, some became longer and stretched, others rounded and hyperchromasia.

Another group of sick myocardium in muscle fibers was found to decompose, fragmentalize. In this case, it is observed that the muscle fibers are destructed and broken down into various small fragments, the cardiomyocytes sarcoplasm is separate, the sarcomere is separate, the part where the core is located is again divided into separate fragments. It is determined that the sarcoplasm and sarcomere of cardiomyocytes disintegrate from the development of strong dystrophy and destructural processes and enter a structureless structure. In most places, myofibrils are observed to swell and myolysize. The nuclei of cardiomyocytes are also of different sizes, the vast majority being enlarged, deformed and subjected to necrobiotic changes. Others are found colorless, light-colored with hematoxylin, with a vacuolated state of carioplasm.



Picture-5. Breakdown and fragmentalization of myocardial muscle fibers. Paint: G-E. Ass: 10x40.



Picture-6. Necrobiosis and necrosis of myocardial muscle fibers. Paint: G-E. Ass: 10x40.

It was found that as a result of the above-mentioned viral poisoning and disorders of metabolism in the myocardium, changes characteristic of necrobiosis and necrosis in the myocardial muscle fibers caused by such pathological processes as advanced dyscirculatory, edema, dystrophy, destruction, dysregeneration, fragmentation. It has been found that myolysis of muscle fibers has developed in two different ways. In the first, it was found that the sarcoplasm of muscle fibers, including cardiomycyte, undergoes intracellular edema, vacuolates, myofibrils break down and undergo aqueous necrobiosis. In the latter case, it was found that the myofibrils of muscle fibers homogenized and entered a rough structure without structure, while the nuclei in cardiomyocytes were lost and necrozed (fig. In necrosis-induced cardiomyocytes, nuclei were observed to initially swell, increase in size, and then lysate chromatin, not bucking, and disappear through the processes of cariolysis and cariopychnosis.

Conclusion

It is known that in COVID-19, the disease of the cardiovascular system is most often manifested by myocarditis, Takosubo cardiomyopathy, acute coronary syndrome, arrhythmia, accidental death from the heart, venous thromboembolism and acute heart failure. It is very likely that polymorphological deviations may develop in the myocardium in most of the indicated heart diseases. As a result of the direct influence of the SARS-CoV-2 virus on the myocardium, the development of all common

pathological processes in the heart tissue, the procoaguyant and prothrombogenic effect of inflammation developed under the influence of the virus, myocardial ischemia from insufficient oxygenation caused by microtomyr dysfunction, inflammation of hypersensitivity as a result of an attack. In the material we studied, vacuolar and fatty dystrophy of cardiomyocytes in myocardial muscle fibers under the influence of the SARS-CoV-2 virus, chaos hypertrophy characteristic of cardiomyopathy of muscle fibers, deformity and contracture of muscle fibers in some cases, fragmentalization, breakdown in other cases, and ultimately necrobiosis and local necrosis of cardiomyocytes were observed.

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