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Pathomorphological Changes in the Cardiovascular System in COVID-19 Disease

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Abstract This study investigates pathomorphological changes in the cardiovascular system in patients with COVID-19 and post-COVID syndrome. The findings demonstrate significant structural and vascular alterations in the myocardium, largely resembling those observed in elderly patients with acute and chronic ischemic heart diseases. The most prominent changes include chronic cardiosclerosis, focal myocarditis, and endothelial dysfunction in the coronary vessels. In addition, pronounced atherosclerotic alterations were identified, characterized by plaque instability, structural destruction, and progression toward decompensation. These processes were frequently associated with enhanced thrombus formation, indicating a high thrombogenic potential in affected patients. The observed pathological changes suggest that SARS-CoV-2 infection contributes not only to acute myocardial injury but also to the progression of pre-existing cardiovascular conditions. Furthermore, the persistence of these alterations in the post-COVID period highlights the long-term impact of the disease on cardiac structure and function. Overall, the results emphasize the critical role of vascular and myocardial damage in the pathogenesis of COVID-19-related cardiovascular complications.

Keywords COVID-19, SARS-CoV-2, ACE2 receptors, Myocardium, Coronary vessels, Cardiovascular system, Myocarditis, Post-COVID syndrome

1. Introduction

Coronavirus disease 2019 (COVID-19), caused by the SARS-CoV-2 virus, remains a major global health challenge due to its multisystem involvement and long-term consequences. Although primarily affecting the respiratory system, increasing evidence demonstrates significant involvement of the cardiovascular system, which substantially contributes to morbidity and mortality rates [1,2]. The virus gains entry into host cells via angiotensin-converting enzyme 2 (ACE2) receptors, which are highly expressed in cardiomyocytes and vascular endothelial cells, thereby facilitating direct myocardial and vascular injury [9,11].

The pathogenesis of cardiovascular complications in COVID-19 is multifactorial and involves direct viral cytotoxic effects, endothelial dysfunction, dysregulation of the renin–angiotensin–aldosterone system, and an excessive inflammatory response known as a cytokine storm [3,4,12]. These mechanisms result in myocardial injury, ischemia, microvascular dysfunction, and activation of prothrombotic pathways, significantly increasing the risk of thromboembolic events [5,13].

In addition, systemic hypoxia, metabolic imbalance, and increased sympathetic activity further aggravate myocardial damage and contribute to the development of cardiac arrhythmias and conduction disturbances [8]. Elevated levels of cardiac biomarkers, particularly troponins, are considered important indicators of myocardial injury and are associated with poor prognosis [2,14].

Clinical and pathological studies indicate that COVID-19 patients frequently develop myocarditis, pericarditis, myocardial infarction, and pulmonary embolism, especially in severe forms of the disease [6,15]. Moreover, persistent cardiovascular complications have been observed in the post-COVID period, including left ventricular dysfunction, myocardial fibrosis, and chronic inflammatory changes, which may lead to long-term cardiac impairment [7,12].

Thus, the study of pathomorphological changes in the cardiovascular system in COVID-19 remains highly relevant for understanding disease mechanisms, improving diagnostic accuracy, and optimizing therapeutic strategies.

Purpose of the study

The purpose of study the pathomorphological changes in the cardiovascular system during the periods of the COVID-19 disease and the post-covid syndrome.

2. Materials and Methods

This study included 60 patients who died from acute

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COVID-19 infection and post-COVID syndrome between 2020 and 2023, as well as 18 control subjects who died from non-COVID-related causes. The subjects were stratified by age into decade-based groups starting from 20 years. Autopsy data were obtained from the Department of Infectious Diseases of the Pathological Anatomy Center of the Ministry of Health of the Republic of Uzbekistan, as well as from hospitals in the Andijan region.

During autopsy, the heart was examined macroscopically. Heart weight, anatomical structure, and morphometric parameters, including dimensions and left ventricular wall thickness, were measured. Particular attention was given to the assessment of atherosclerotic changes in the coronary arteries, as well as the presence of stenosis and thrombosis.

For histological examination, myocardial tissue samples measuring approximately 1×1 cm were collected and fixed in 4% neutral buffered formalin. Tissue sections were obtained from the anterior and posterior walls of the left ventricle (2 cm above the apex), below the fibrous ring of the mitral valve, and from the interventricular septum. After dehydration in graded alcohol and chloroform, the samples were embedded in paraffin blocks.

Paraffin sections with a thickness of 5–8 μm were prepared and stained with hematoxylin and eosin for microscopic evaluation.

3. Results

Morphological examination of the myocardium in approximately one-third of autopsy cases of patients who died from COVID-19 revealed venous congestion and the presence of both small and large sclerotic foci. Microscopic analysis showed venous plethora, perivascular sclerosis, and signs of ischemia. Cardiomyocytes exhibited a hypercontractile state accompanied by structural alterations, including undulation, fuchsinophilia, and fragmentation (Fig. 1). Severe contracture injuries corresponding to grade III were predominantly observed in the ventricular myocardium. Notably, in all examined cases, blood within the cardiac cavities remained in a liquid state (Tables 1 and 2).

Morpho- and planimetric examination of the coronary vessels revealed the presence of lipid deposits and fibrous plaques in the intima and media, with a maximum area of up to 10%. The thickening of the coronary vessel walls and the severity of atherosclerotic lesions increased progressively with age. These indicators were minimal in the 20–29-year-old group and reached their maximum in individuals aged 50–59 years.

Table 1. Macroscopic and morphometric parameters of the heart in covid-19 patients

Age group	Heart weight (g)	Heart dimensions (cm)	Left ventricle wall thickness (cm)
	Control	COVID-19	RKS
20–29	280.2	310.2	330.4
30–39	290.2	320.4	340.5
40–49	270.2	320.6	345.2
50–59	290.1	330.2	360.4
60–69	270.2	330.4	360.8
≥70	265.2	335.1	380.2

Notes: X — mean, G — standard deviation, m — standard error.

Table 2. Microscopic morphometric indicators of the heart in covid-19 patients

Age group	Left ventricle contractile coefficient	Wavy cardiomyocytes coefficient	Symptoms of dysstasia
	Control	COVID-19	RKS
20–29	52.1	54.3	58.2
30–39	52.4	56.2	60.4
40–49	54.5	57.3	62.5
50–59	56.4	58.3	59.9
60–69	56.7	57.1	58.3
≥70	56.9	59.5	62.3

Notes: X — mean, G — standard deviation, m — standard error.

Table 3. Age-related dynamics of atherosclerotic changes in coronary arteries (control group, % of total surface)

Age group	Lower branch of left coronary artery	Branch of left coronary artery	Right coronary artery
20–30	0.7 ±0.1	0.4 ±0.1	0.7 ±0.1
30–40	4.6 ±0.3	2.3 ±0.3	3.6 ±0.5
40–50	10.5 ±0.7	5.2 ±0.4	10.1 ±1.0
50–60	18.5 ±1.2	14.1 ±1.1	17.6 ±1.5
≥60	30.0 ±1.4	21.7 ±1.1	29.5 ±1.5

Notes: Values are presented as mean ±standard deviation.

A clear correlation was observed between the extent of atherosclerotic damage and the surface area of fibrotic plaques. The average size of fibrous plaques relative to the intimal surface of coronary vessels increased with age. Notably, after the age of 50, a significant progression was observed, resulting in stenosis of approximately one-quarter of the vessel lumen (Table 3).

Pathomorphological examination of the hearts and coronary vessels of patients who died from COVID-19 revealed multiple structural alterations. Macroscopically, myocardial ischemia was observed alongside small and large sclerotic foci, atrophy of cardiomyocytes, focal lipid deposition, and thickening of the left ventricular wall. In some cases, myogenic dilatation was also noted.

Microscopically, the myocardium exhibited venous engorgement, perivascular edema, hypercontractile cardiomyocytes, wavy folds, fuchsinophilia, fragmentation, and, in certain instances, necrosis. Coronary vessels showed lipid deposits, fibrous plaques, atheromatous lesions with areas of destruction, perivascular sclerosis, endothelial dysfunction, intimal and medial damage, ischemia, and thrombosis.

These findings indicate that COVID-19 induces both macro- and microstructural damage in the myocardium and coronary vessels, highlighting the multifactorial pathogenesis of cardiac injury in this disease (see Figure 1).

Dystrophic and necrobiotic changes, as well as focal and diffuse interstitial and perivascular sclerosis, were observed in atrophied cardiomyocytes. Fat inclusions and fatty dystrophy were also noted in the cytoplasm of some cardiomyocytes.

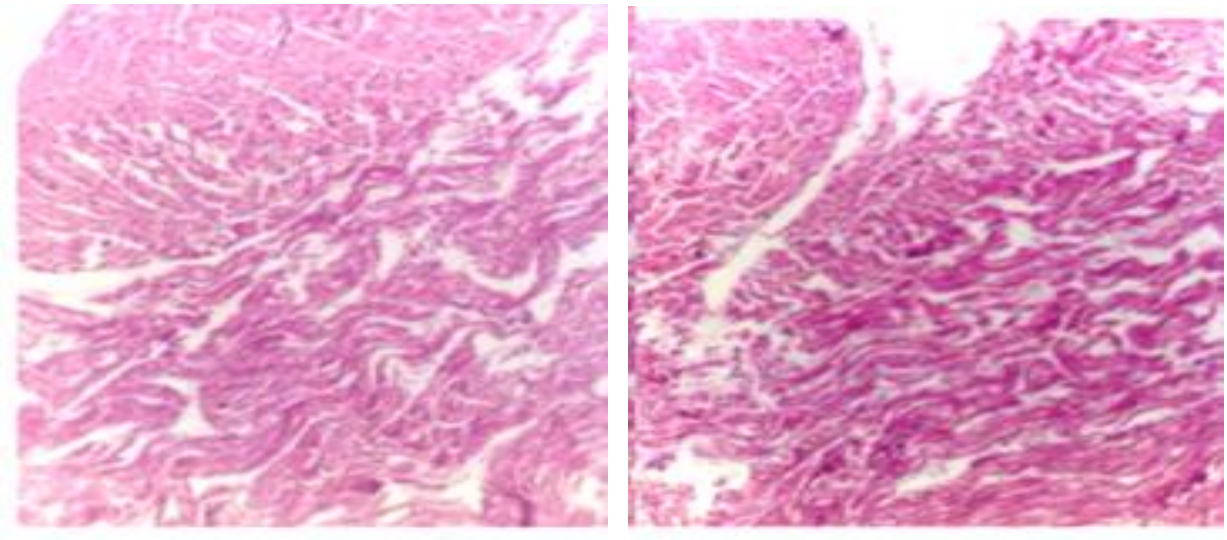

Figure 1. Cardiomyositis contracture damage of the 2nd-3rd degree in the death of the COVID-19 disease. Stained by the hemm.-eosin method. 4x40 magnification

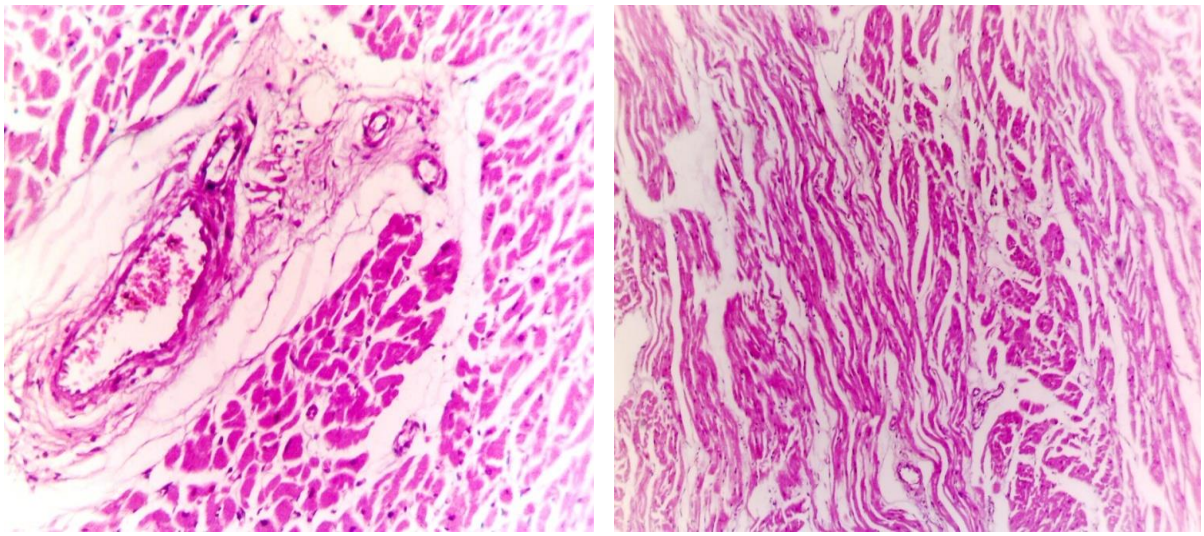

Figure 2. Patient F.F., born in 1995. 1-2. moderate fullness, perivascular sclerosis, swelling, interstitial tissue swelling, focal adipose tissue growth, small sclerosis, foci, undulating course of some groups of cardiomyositis, fragmentastia, a small amount of lymphoid infiltration in the interstitial tissue. Hemm.-eosin staining. Magnification 4*12.5

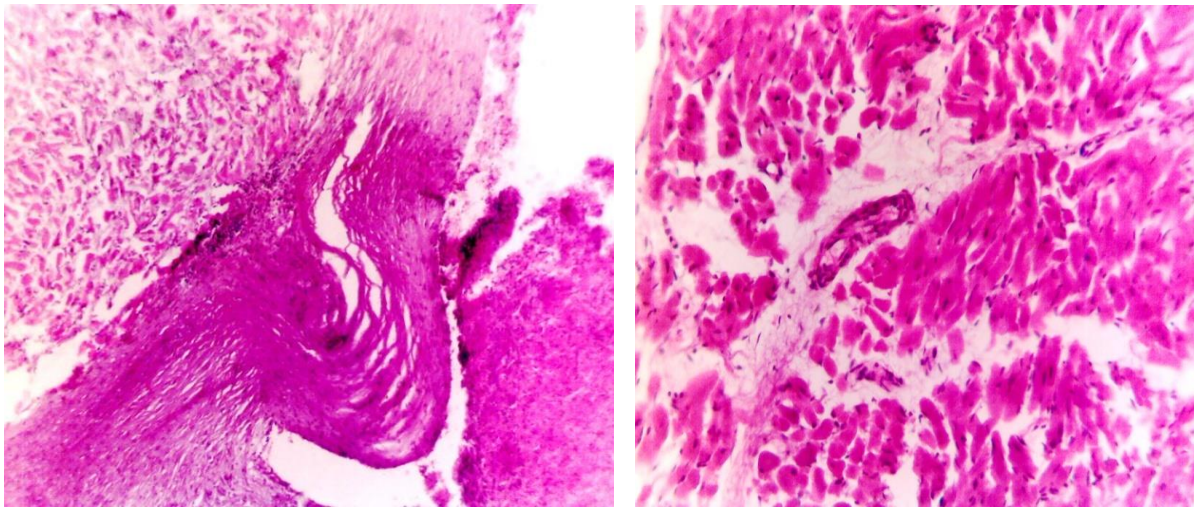

Figure 3. Patient I.X. 1988y.t. 1. Lipoidosis, liposclerosis, atheromatous changes in the intima and media layers of coronary vessels due to sharp narrowing and desquamation of the intima. 2. Patient B.I. 1990 moderate fullness, perivascular sclerosis, edema, small sclerosis foci in the myocardium, a small amount of lymphoid infiltration, the wave-like course of some groups of cardiomyocytes, fragmentation, and atrophic changes. Hemm.-eosin staining. Magnification 1. - 4*12.5. 2.10*12.5

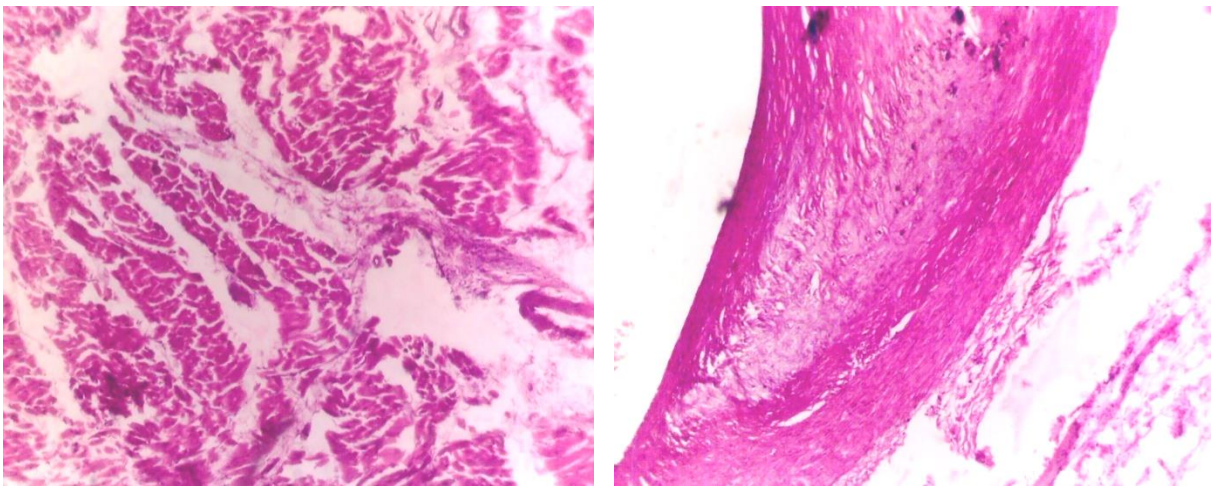

Figure 4. Patient U.S., born in 1982. 1. Perivascular sclerosis, edema of perivascular and interstitial tissue, small sclerosis foci, undulating course of certain groups of cardiomyocytes, fragmentation, necrobiotic changes, 2. lipidosis, lipo sclerosis, atheromatous in the intima and media layers of coronary vessels. Changes, wall thickening, and cavity narrowing to 1/3. Hemm.-eosin staining. Magnification 1. 4*12.5. 2.10*12.5

These dystrophic alterations in the myocardium were accompanied by a reduction in oxidative phosphorylation, slowed protein synthesis, impaired lipid metabolism, disrupted electrolyte balance, and decreased oxygen absorption. Accumulation of acidic glycosaminoglycans in the myocardium resulted in swelling of both the myocardial tissue and the stroma.

Following COVID-19 infection, post-COVID syndromes may develop, with decreased contractile function of the left ventricular myocardium due to sclerotic changes and conduction disorders identified as major risk factors [10]. Metabolic, ischemic, and mechanical factors were also recognized as contributing elements (substrate + trigger factors).

Histological examination of the ventricles demonstrated predominance of contracture damage of the 2nd–3rd degree in cardiomyocytes. Contractures were primarily located in the anterior, lateral, and posterior walls of the left ventricle, predominantly affecting subendocardial and intramural regions (see Figures 2). In patients aged 20–29 years, myocarditis with small sclerosis foci was observed, including three cases of focal myocarditis. In patients aged 30–39 years, small sclerosis foci (100%) and large sclerosis foci (40%) were noted in the myocardium, along with focal myocarditis in four cases (see Figures 2–3).

In 40-49-year-old patients, foci of small sclerosis in the myocardium, stenotic atherosclerotic changes in the coronary vessels in 40% of cases, atheromatous changes, their destruction, and focal myocarditis were observed in 80% of cases.

In patients aged 40–49 years, histological examination revealed small foci of myocardial sclerosis. Stenotic atherosclerotic changes in the coronary vessels were observed in 40% of cases, while atheromatous lesions, their partial destruction, and focal myocarditis were detected in 80% of cases (see Figure 4).

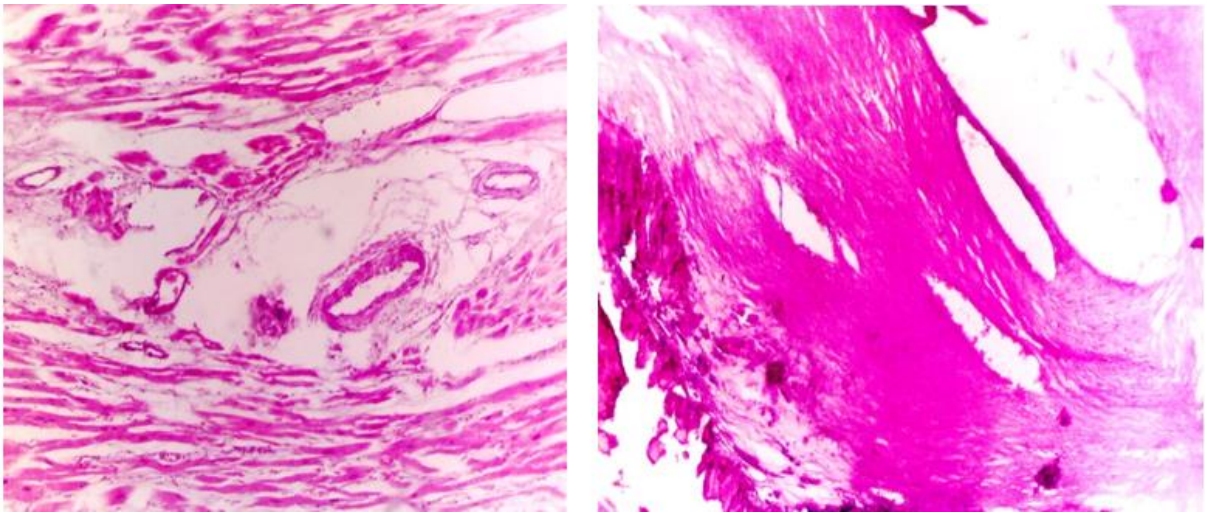

Figure 5. Patient G.K. 1969 1- moderate venous congestion, perivascular sclerosis, edema, interstitial tissue edema, small and large foci of sclerosis, undulating course of certain groups of cardiomyocytes, fragmentation, necrobiotic changes, foci growth of fatty tissue, desquamation of the intima of 2 coronary vessels, atheromatous changes in the intima and media, foci of calcification, destruction, a sharp narrowing of the cavity, decompensation, using the Hemm-eosin method live Magnification 4*12.5

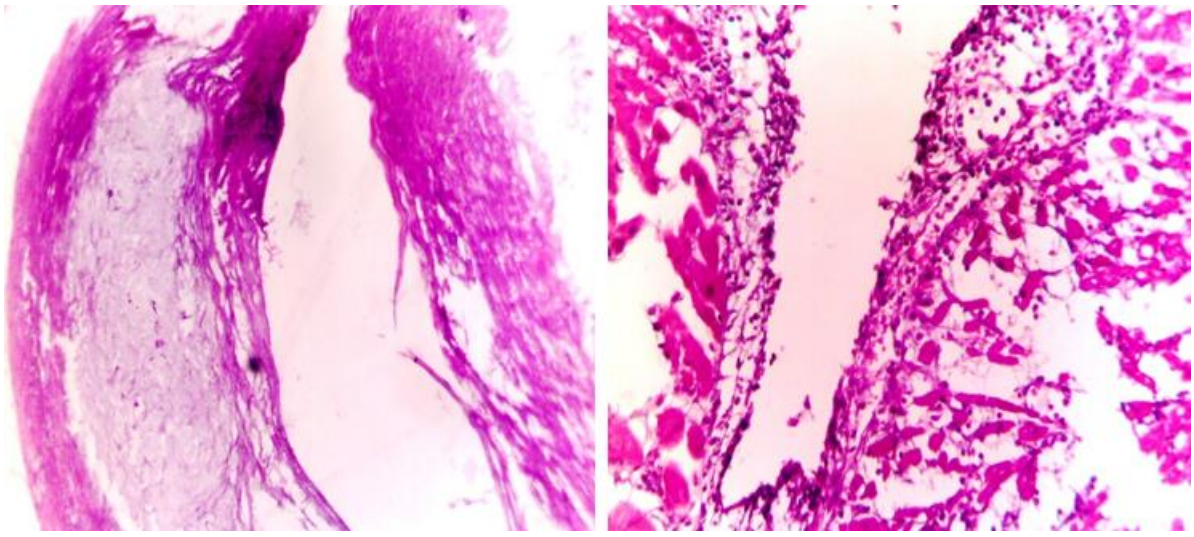

Figure 6. Patient S.M. was born in 1960. 1. atheromatous changes in the coronary vessels, thickening of the intima and media layers, thickening of the wall, narrowing of the cavity to ½ part, destruction, 2. lymphoid infiltration in the intima, perivascular edema, sclerosis, edema in the surrounding myocardium, small sclerosis focus staining by Gemm.-eosin method—magnification 10*12.5 and 4*12.5

Table 4. Age-related dynamics of atherosclerotic changes in coronary vessels in the study group (in % of the total area)

Age group	Lower branch of the left coronary artery	Branch of the left coronary artery	Right coronary artery
20–30	0.9 ± 0.1	0.4 ± 0.1	1.0 ± 0.1
30–40	5.1 ± 0.4	2.8 ± 0.1	4.1 ± 0.1
40–50	12.1 ± 0.9	6.3 ± 0.3	11.5 ± 0.7
50–60	22.1 ± 1.4	15.4 ± 1.4	24.3 ± 1.1
≥60	27.1 ± 1.6	20.1 ± 1.7	29.6 ± 1.6

In patients aged 60–69 years, both acute and chronic ischemic heart diseases were observed in all cases. Small atherosclerotic foci were present in 80% of patients, post-infarction large-focal sclerosis in 20%, and stenotic atherosclerosis with focal myocarditis in 60% of cases. Myocardial infarction was identified in 40% of patients (see Figure 5).

When measuring the thickness and stenosis of the coronary vessel walls, morphometric indicators were minimal in patients aged 20–29 years and maximal in those aged 50–59 years, manifesting as destructive changes (see Figures 2). In the examined groups, the average surface area of fibrotic tissue relative to the intimal surface of the vessels increased with age. A pronounced progression of these indicators was

observed after 50 years, resulting in stenosis of approximately one-third of the vessel lumen. A significant correlation was observed between the extent of atherosclerotic damage and the area of fibrotic plaques (Table-4).

4. Conclusions

Morphological and morphometric examination of the myocardium in patients with COVID-19 and post-COVID syndrome demonstrates that precise histopathological diagnosis can be challenging due to the heterogeneity of myocardial changes. The extent of myocardial damage varies depending on the patient's age, disease severity, and duration of infection. Macroscopic and microscopic analyses consistently show an increase in morphometric parameters, including myocardial wall thickness, contractile dysfunction, and the presence of sclerosis foci. Concurrently, atherosclerotic changes in coronary vessels worsen progressively, with increased wall thickening, formation of fibrotic and lipid plaques, intimal and medial destruction, and thrombosis formation.

The findings also highlight the importance of both cardiac and extracardiac triggering factors in the development of myocardial ischemia. Age-related progression is accompanied by an elevated incidence of acute coronary events, including myocardial infarction, accidental cardiac death, acute coronary syndrome, and post-infarction scarring. Focal and diffuse myocarditis, along with endothelial dysfunction in coronary vessels, contribute to impaired myocardial perfusion and contractile function. Decompensation of atherosclerotic lesions and increased thrombogenic activity further exacerbate cardiac injury.

These observations underscore the multifactorial pathogenesis of COVID-19-related cardiac damage, in which viral effects, inflammatory responses, ischemic insults, and age-dependent structural changes interact to determine the severity and clinical outcomes of myocardial injury. Comprehensive morphometric assessment of both the myocardium and coronary vessels is therefore essential for understanding the cardiac complications associated with COVID-19 and for guiding post-infection clinical management.

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