Research Article

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Course and Outcomes of Acute Coronary Syndrome in the Presence of New Coronavirus Infection COVID-19

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ABSTRACT We analyzed the clinical condition of patients with COVID-19 of varying severity, changes in instrumental and laboratory parameters, and assessed the impact of the severity of the course of a new coronavirus infection on the outcomes of acute coronary syndrome.

AIM OF STUDY To study the mutual influence of acute coronary syndrome and the new coronavirus infection COVID-19 on the nature of the course and outcomes of the disease.

MATERIAL AND METHODS In March 21, 2020 – May 31, 2021, 3 625 patients were treated for COVID-19, including 131 patients with acute coronary syndrome due to COVID-19 disease. All patients underwent a number of studies: computed tomography of the chest, electrocardiography, echocardiography, monitoring of biomarkers of myocardial damage, diagnostic coronary angiography and, if necessary, intracoronary therapeutic intervention.

RESULTS Data on the distribution of patients with COVID-19 according to the presence or absence of ST segment elevation on the electrocardiogram and the degree of lung tissue damage, as well as information on mortality in these groups, are presented. The role of troponin I in the assessment of myocardial ischemia was analyzed. The direct dependence of its level on the volume of lung damage was found. The inverse relationship was shown between the degree of damage to the lung tissue and the indices of oxygen saturation in the blood. A poor prognostic value of low left ventricular ejection fraction in patients with COVID-19 disease has been described.

CONCLUSIONS The development of acute coronary syndrome in the course of COVID-19 significantly worsens the prognosis of the disease, which requires the development of algorithms for providing medical care to patients in this category, as well as maximum vigilance in their treatment.

Keywords: COVID-19; acute coronary syndrome with ST segment elevation; non-ST elevation acute coronary syndrome; echocardiography; ejection fraction of the left ventricle; saturation of oxygen in the blood; troponin I for COVID-19

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ACS – acute coronary syndrome
ALV – artificial lung ventilation
CA – coronary arteries
CAD – coronary artery disease
CT – computed tomography
EchoCG – echocardiography
ECG – electrocardiography
EF – ejection fraction
LV – left ventricle
NCI – new coronavirus infection

INTRODUCTION

Since the beginning of the COVID-19 pandemic, discussions about the tactics of examining and treating patients with acute coronary syndrome (ACS) in the course of coronavirus infection have not ceased. The most pressing issues were the assessment of the degree of mutual influence of these diseases on the severity of the condition of patients and on clinical outcomes. This study presents an analysis of the clinical characteristics of patients with COVID-19 of varying severity, changes in instrumental and laboratory parameters, and an assessment of the impact of the severity of the course of a new coronavirus infection (NCI) on the outcomes of ACS.

Aim of the study: to study the mutual influence of ACS and NCI COVID-19 on the nature of the course and outcomes of the disease.

MATERIAL AND METHODS

From March 21, 2020 to May 31, 2021, 3 625 patients were treated for COVID-19 infection at the N.V. Sklifosovsky Research Institute for Emergency Medicine, including 131 patients hospitalized for ACS due to COVID-19 disease. All patients underwent a number of studies: computed tomography (CT) of the chest, electrocardiography (ECG), echocardiography (EchoCG), monitoring of biomarkers of myocardial injury, diagnostic coronary angiography and, if necessary, intracoronary therapeutic intervention.

Among the patients there were 71 patients with ST segment elevation, mean age 71.7 years (min 33; max 97), and 60 patients without ST segment elevation, mean age 72.7 years (min 44; max 92) (Fig. 1).

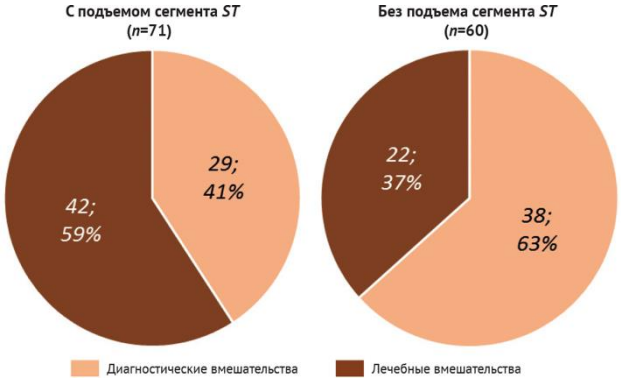

Fig. 1. Coronary angiography and therapeutic coronary interventions (n=131)

During hospitalization and in the following days, according to indications, all patients underwent ECG registration, echocardiography, and biochemical blood tests to determine markers of myocardial injury. All patients underwent CT scan of the chest during hospitalization in order to determine the volume of lung tissue damage.

The determination of the degree of damage to the lung tissue by coronavirus pneumonia was performed on the basis of the Order of the Moscow Department of Health dated Apr 08,2020 No. 373 (as amended on Apr 17, 2020) [1]. The degree of lung tissue lesion was assessed according to the accepted system: CT0 — absence of foci of inflammation and infiltrates; CT1 — the presence of signs of viral pneumonia, involving the volume of up to 25% of the lung tissue; CT2 — the volume of lung damage from 25 to 50%; CT3 — lung tissue damage from 50 to 75%; CT4 — lesion of more than 75% of the lung tissue. All patients hospitalized with clinical and ECG signs of ACS underwent invasive coronary angiography and, if necessary, intracoronary therapeutic intervention.

RESULTS

CHARACTERISTICS OF LUNG TISSUE LESION IN PATIENTS WITH ACUTE CORONARY SYNDROME ACCORDING TO COMPUTED TOMOGRAPHY DATA

Out of 131 patients with COVID-19 admitted to the infectious department with ACS symptoms, 39 had a lesion exceeding 75% of the lung tissue volume (CT4); in 20 — 50–75% (CT3); in 26 — 25–50% (CT2); in 39 — up to 25% (CT1); in 7 patients no foci of inflammation and infiltrates were detected (CT0).

Comparative analysis of groups of patients, distributed according to the presence or absence of ST segment elevation, showed that in patients with ST segment elevation, pronounced changes in the lung tissue predominated. Moderate, severe and extremely severe lesions of the lung tissue (CT2-CT4) were detected in 75% of patients in this group (Fig. 2).

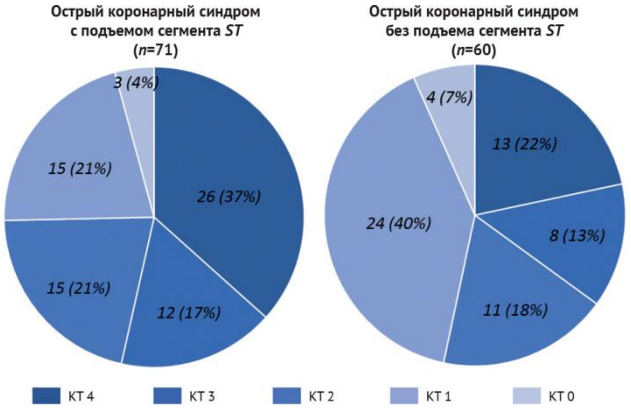

Fig. 2. Distribution of patients depending on the CT pattern of lung lesions

In the group of patients without ST segment elevation, moderate, severe and extremely severe lesions of the lung tissue (CT2–CT4) were detected in 53% of patients (Fig. 2).

Today, in the diagnosis of acute ischemic or other myocardial damage, in addition to the ECG study, much attention is paid to biochemical markers and, first of all, the level of troponin I in blood plasma [2]. An elevated troponin I level is considered a specific cardiac marker and indicates the presence of ischemic or other myocardial injury.

In our study, in the group of patients with clinical signs of myocardial infarction and ST segment elevation (n = 71), according to the results of coronary angiography, 20 patients did not find significant changes in coronary artery requiring therapeutic interventions. In this group of patients, troponin I levels were studied, which averaged 1.47 μg/l (the norm is 0.00–0.023 μg/l). Next, the levels of troponin I were compared within this group of patients (data from 13 patients are available), taking into account the CT picture of lung tissue damage.

Average values of troponin I level in patients with ST segment elevation were distributed as follows: CT4 (n =3) — 5.55 μg/l, CT3 (n =2) — 0.22 μg/l, CT2 (n =3) — 0.14 μg/l. The maximum level of troponin I was obtained in a patient with extremely severe lung disease (TP4) and amounted to 13 μg/l. The mean troponin I in the subgroup of patients with lung involvement in the degree of CT1 (n = 4) was 0.06 μg/l.

However, another patient from the CT1 subgroup, hospitalized with an ACS clinic and ST segment elevation, who also did not show significant changes in CA, had a significant increase in troponin I up to 1.4 μg/l, indicating ischemic or other myocardial lesion. Further examination revealed a number of comorbidities in the patient: acute myopericarditis, arterial hypertension stage III, 3rd degree, paroxysmal atrial fibrillation, chronic pancreatitis, chronic duodenitis. It is to be recalled that the average level of troponin I in the group (CT1) was only 0.06 μg/l.

ST segment elevation (n = 60), during diagnostic studies, hemodynamically insignificant coronary artery lesions were found in 18 patients. In all 18 patients of this group, before coronary angiography, the level of troponin I was determined, which average value was 0.31 μg/l (norm 0.00–0.023 μg/l). In 13 patients out of 18, the level of troponin I was within the normal range. Taking into account the CT picture, the average values of the level of troponin I in patients of this group were distributed as follows: CT4 (n = 3) — 1.05 μg/l; CT3 (n =1) — 0.027 μg/l; CT1 (n =8) — 0.05 μg/l; CT0 (n =3) — 0.08 μg/l.

The maximum level of troponin I was obtained in a patient with extremely severe lung disease (TP4) and amounted to 2.7 μg/l. In the group of patients with moderate lung tissue damage (CT2), troponin I data of 0.01 μg/l were obtained in one patient, and troponin I was significantly higher in two patients of this group with severe concomitant diseases. In one patient, troponin I was 0.52 μg/l, she suffered from acute myocarditis, coronary artery disease (CAD), chronic heart failure stage IIB, chronic kidney disease C3b, arterial hypertension stage III, grade 3, the risk of cardiovascular vascular complications 4, type II diabetes mellitus, permanent atrial fibrillation. In another patient of this group, suffering from coronary artery disease, acute heart failure, chronic kidney disease C2, anemia, an increase in the level of troponin I up to 1.2 μg/l was found.

Thus, the data obtained indicate a direct association of an increase in the level of troponin I, clinical, ECG signs of myocardial ischemia, and the degree of damage to the lung tissue by coronavirus infection without an obvious lesion of the coronary artery.

Moreover, this trend is found both in patients with severe myocardial damage, accompanied by ST elevation, and in patients with ACS, which occurs without ST segment elevation. This suggests the hypoxemic nature of ischemic myocardial damage associated with a violation of the gas exchange function of the lungs and increasing in accordance with an increase in the degree of damage to the lung tissue. The small number of observations does not allow definitive conclusions to be drawn. However, the study of the identified trend should probably be the subject of further, more detailed studies. At the same time, of course, it is necessary to take into account other serious diseases that can accompany COVID-19 and lead to myocardial damage.

ASSESSMENT OF OXYGEN SATURATION IN THE BLOOD

Before admission to the X-ray operating room, out of 71 patients with ST segment elevation on the ECG, 24 patients were on mechanical ventilation, 47 were breathing independently. In patients breathing independently, the average oxygen saturation in the blood was 91.3%; in patients on mechanical ventilation it was 93.6%.

Among 60 patients without ST segment elevation before admission to the X-ray operating room, 9 people were on mechanical ventilation, 51 were breathing independently. In patients on spontaneous breathing, the average saturation of oxygen in the blood was 93%; in patients on mechanical ventilation — 95.1% (Fig. 3).

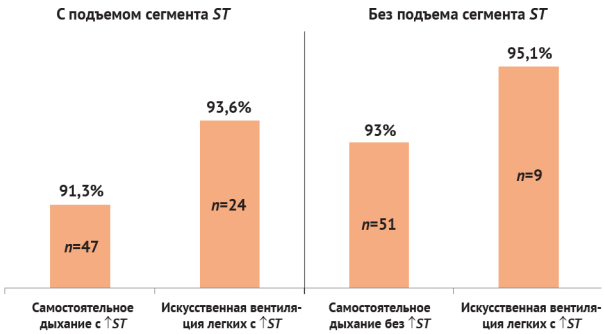

Fig. 3. Blood oxygen saturation (mean values) in all patients

Notes: *- the symbol " ST " means ACS with ST segment elevation on the ECG; the symbol "without ST" means ACS without ST segment elevation on the ECG

The low oxygen saturation level was taken to be less than 95% oxygen saturation. Among 71 patients with ST elevation, initially low saturation was detected in 34 patients (47.9% of the group), 12 of them were on a ventilator.

The comparison of the level of oxygen saturation in the blood with the degree of lung tissue damage and ECG changes in patients with ACS revealed complex relationships between these processes in the context of developing coronavirus pneumonia. A general inverse relationship was found between the level of oxygen saturation in the blood and the volume and severity of lung tissue damage in patients with varying degrees of myocardial ischemia.

Thus, among patients on spontaneous breathing with ST segment elevation and CT4 lung damage, the average oxygen saturation in the blood was 86.1%; in patients with lung involvement CT3 — 91.1%; in patients with lung lesions CT2 — 91.2%; in patients with lung lesions CT1 — 93.1%; in patients with CT0 — 97.3% (Fig. 4).

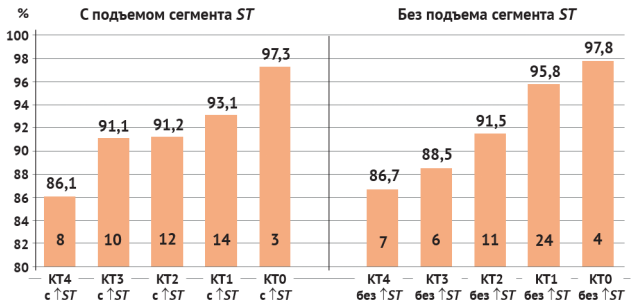

Fig. 4. Saturation of oxygen in the blood in patients on spontaneous breathing, depending on the CT pattern of lung damage

Notes: *- the symbol " ST" means ACS with ST segment elevation on the ECG; the symbol "without ST" means ACS without ST segment elevation on the ECG

Among patients with ACS without ST segment elevation on the ECG, who were breathing independently, with CT4 lung lesion, the average oxygen saturation in the blood was 86.7%; in patients with lung involvement CT3 — 88.5%; in patients with CT2 — 91.5%; in patients with CT1 — 95.8%; in patients without signs of lung tissue damage (CT0), oxygen saturation in the blood was 97.8% (Fig. 4).

The picture of oxygen saturation in the blood in patients with COVID-19 on mechanical ventilation differed significantly from the distribution of the same indicators in patients on spontaneous breathing. Thus, among patients with ST-segment elevation, who were on mechanical ventilation, with extremely severe lung damage (CT4), the average oxygen saturation averaged 93.3%; in patients with CT3 — 97.5%; in patients with CT2 — 93%; with CT1 — 94% (Fig. 5). Patients without signs of lung tissue damage (CT0) did not have mechanical ventilation.

Among patients without ST elevation on mechanical ventilation, mean blood oxygen saturation in patients with CT4 lung disease was 94%; with CT3 — 98.5% (Fig. 5). Patients with lesser lung tissue lesions (CT2–CT0) were not put on a ventilator.

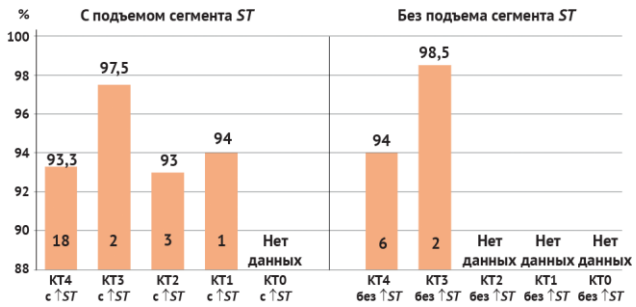

Fig. 5. Oxygen saturation in the blood in patients on mechanical ventilation, depending on the CT pattern of lung damage

Notes: *- the symbol " ST" means ACS with ST segment elevation on the ECG; the symbol "without ST" means ACS without ST segment elevation on the ECG

The comparison of data on oxygen saturation in the blood in patients on mechanical ventilation with varying degrees of lung damage shows a rather mixed picture. Significant and multidirectional differences in the data obtained in groups of patients with ACS with and without ST segment elevation and the apparent lack of dependence of the level of oxygen saturation in the blood, despite the ventilation mode, can be explained, first of all, by a small sample, as well as severe comorbidity, significant differences in age, the presence of excess weight up to various degrees of obesity and significant differences in the size of ischemic myocardial damage.

Unfortunately, all patients with or without ST elevation who were put on a ventilation, regardless of the severity of the coronary lesion and the degree of lung damage, died.

EVALUATION OF THE CONTRACTILE FUNCTION OF THE LEFT VENTRICLE OF THE HEART

The ejection fraction (EF) of the left ventricle (LV) was determined by echocardiography. The mean LV EF in patients with ST elevation before admission to the operating room was 43%. The average LV EF in patients without ST segment elevation before admission to the operating room was 47.2%.

Depending on the degree of the lung tissue lesion in the group of patients with ST segment elevation, the LV ejection fraction in patients with extremely severe lung changes (CT4) was 44.7%; in patients with severe damage to the lung tissue (CT3) — 34%; in patients with moderate changes in the lung tissue (CT2) — 49.3%; in patients with changes in the lungs CT1 — 44.6%, in patients without lung damage (CT0) — 57% (Fig. 6).

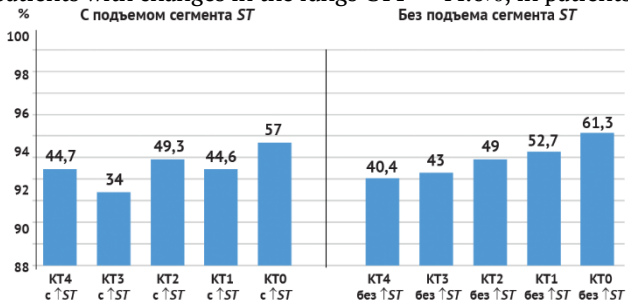

Fig. 6. LV ejection fraction in patients with ACS with and without ST elevation on the ECG with varying degrees of lung tissue damage

Notes: *- the symbol " ST" means ACS with ST segment elevation on the ECG; the symbol "without ST" means ACS without ST segment elevation on the ECG

In the group of patients without ST segment elevation who were referred for coronary angiography and having signs of extremely severe lung damage: LV CT4 EF was, on average, 40.4%, in patients with lung lesions CT3 — 43%, in patients with lung tissue damage to the level CT2 — 49%, in patients with CT1 — 52.7%, in patients without signs of viral pneumonia — CT0, the LV ejection fraction averaged 61.3% (Fig. 6).

The comparison of LV EF in the presented groups of patients demonstrates the multidirectional changes in this indicator in patients with ACS and ST segment elevation, that is, with critical myocardial ischemia. At the same time, there is no relationship between the level of lung tissue damage and the volume of LV EF. This is probably associated with differences in the volume and depth of myocardial injury in these patients. At the same time, ischemic myocardial damage most likely prevails over the influence of systemic hypoxemia, which is associated with the degree of lung damage. In contrast, in patients with non-ST elevation ACS, there is a clear inverse relationship between the depth of lung injury and the preservation of LV myocardial contractility.

ANALYSIS OF MORTALITY IN PATIENTS WITH ACUTE CORONARY SYNDROME THAT DEVELOPED IN THE COURSE OF CORONAVIRUS PNEUMONIA

Among patients with ST elevation (n = 71), 26 had extremely severe lung disease (CT4). All 26 patients of this subgroup died, which was 100%. Of the 12 patients with severe lung lesions 50-75% (CT3), 8 patients (66.7%) died. In 15 patients, lesions of the lungs of moderate severity (CT2) were found; 9 patients in this subgroup died (60%). Out of 15 patients with mild lung injury (up to 25%) (CT1), 7 patients (46.7%) died. In 3 patients no foci of inflammation and infiltrates were detected at the time of hospitalization or coronary angiography (CT0). However, 2 patients in this subgroup died (Fig. 7).

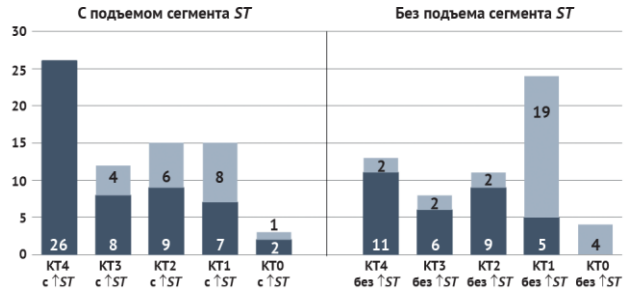

Fig. 7. CT pattern and mortality information in all patients with ACS + COVID-19 (n=131)

Notes: * — the symbol "ST" means ACS with ST segment elevation on the ECG; the symbol "without ST" means ACS without ST segment elevation on the ECG

As mentioned above, in 13 patients in the group of patients with ST-segment elevation ACS without significant changes in the coronary artery, the level of troponin I was studied at the time of arrival at the X-ray operating room. In this subgroup, myocardial infarction was successfully transferred against the background of COVID-19 infection and 5 patients survived. The average level of troponin I upon hospitalization or coronary angiography in these 5 patients was 0.34 μg/l (min 0.01, max 1.4). In 8 patients of this subgroup who died at different stages of COVID-19 disease, the average level of troponin I was 2.2 μg/l upon hospitalization or

coronary angiography (min 0.015, max 13). In 5 out of 8 patients who died, severe and extremely severe course of the disease with massive damage to the lung tissue (CT3–CT4) was noted.

Of 60 patients without ST segment elevation, 13 had lung lesions greater than 75% (CT4); 11 patients from this subgroup died (84.6%). These were 3 patients without coronary lesions, 7 patients with multiple and severe coronary lesions who did not undergo intracoronary therapeutic intervention, and 3 patients with hemodynamically significant lesions who underwent therapeutic intracoronary intervention.

Eight patients had severe lung lesions (CT3); 6 patients in this subgroup died (75%). In 11 patients, 25–50% lung lesions were detected (CT2); 9 patients died (81.8%). In 24 patients, lung lesions up to 25% (CT1) were detected; 5 patients in this subgroup died (20.8%). In 4 patients no foci of inflammation and infiltrates were detected (CT0). All patients from the CT0 group were successfully treated and discharged for outpatient follow-up (Fig. 7).

Four patients with ST elevation and severe lung injury (CT2–CT4) were transferred to the X-ray operating room on the first day after hospitalization and died after coronary angiography, which accounted for 3.1% of all diagnostic and therapeutic intracoronary interventions. In 2 patients, death occurred during X-ray surgery. Another 2 patients died in the early postoperative period.

In the group of patients with ACS without ST segment elevation (n = 60), diagnostic coronary angiography in 18 patients, as already mentioned, revealed hemodynamically insignificant coronary artery lesions. However, among these patients (n = 18), 4 patients died during the further development of the disease. In 2 of them, according to the CT scan, an extremely severe lesion of the lung tissue was noted. In 13 patients out of 18, the level of troponin I was within the normal range. In 14 surviving patients, the mean troponin I level was 0.09 μg/L (min 0.01; max 0.52). In 4 patients who died, the average level of troponin I was 1.1 μg/l (min 0.11; max 2.7). The maximum level of troponin I in this group was obtained in a patient with CT4 and amounted to 2.7 μg/l.

DISCUSSION

Since the start of the pandemic, it has become clear that COVID-19 disease has a pathological effect on many organs, including the cardiovascular system. Mechanisms of myocardial injury include direct viral damage, mismatch between tissue oxygen delivery and consumption. This contributes to a hyperinflammatory state, the development of stress and inflammatory cardiomyopathy [3]. The possible occurrence against this background of atherosclerotic plaque rupture or thrombosis of the coronary arteries in situ further aggravates the course of the disease, leading to the development of ACS up to myocardial infarction.

Non-coronary myocardial injury in COVID-19, often associated with an increase in troponin I, can create significant difficulties in diagnosing ACS caused by coronary artery occlusion. In this regard, it is necessary to focus not only on specific markers of myocardial damage, but also on the presence of typical clinical symptoms, ECG and EchoCG data, and the presence of complications such as acute heart failure and cardiac arrhythmias. The data obtained in our study are confirmed by Russian and foreign publications [4, 5].

A high level of troponin I is one of the most important indicators for assessing myocardial trauma. However, many factors can influence the increase in troponin I levels: severe respiratory infections and sepsis, acute respiratory distress syndrome in adults, hypoxia, arterial hypotension, pulmonary hypertension, and renal dysfunction. All of these pathological conditions can lead to false positive results in patients with COVID-19.

An increase in troponin I or troponin T in patients with non-cardiac pathology is also described by other authors. The data on the role and incidence of myocardial injury in the noncardiac surgical population were published after a large international prospective cohort study VISION (vascular events in noncardiac surgery patients cohort evaluation), covering more than 15,000 patients over 45 years of age who underwent surgery with a duration of general anesthesia of at least one hour and hospitalized for at least one day after surgery [6].

In all patients in the postoperative period, the level of troponin T was determined 6–12 hours after surgery, as well as on the 1st, 2nd, and 3rd postoperative days. The statistical analysis was aimed primarily at identifying the correlation of postoperative troponin levels with 30-day mortality and the incidence of various postoperative complications. In this study, it was found that troponin values indicating the presence of damage (necrosis) of the myocardium were achieved in 8.3% of all patients, indicating a widespread perioperative myocardial injury in the general surgical population. However, only a subset of these patients could be diagnosed with myocardial infarction according to the current universal international definition. According to F. Botto et al., even among patients with peak troponin T ⩾0.04 ng/ml, only 15.8% had symptoms associated with myocardial ischemia, and changes in the ischemic nature of the ECG were found only in 34.9 % of cases [7].

In the postoperative period, 65% of myocardial injuries identified by an increase in troponin concentration were absolutely asymptomatic. Regardless of the presence or absence of clinical signs of myocardial ischemia, an increase in the level of troponin in the postoperative period correlated with 30-day mortality, being its most reliable predictor. Such a high predictive value of postoperative "troponinemia" for clinical outcome after noncardiac surgery led researchers to introduce a new term to define all clinical cases of postoperative troponin elevation, regardless of the presence of concomitant clinical signs of myocardial ischemia: MINS (myocardial injury after noncardiac surgery).

In our observations, an increase in the level of troponin I in patients with COVID-19 was an unfavorable prognostic sign even in the absence of ischemic myocardial damage. An increase in this marker accompanied severe lung damage or myocarditis. Similar data were published by domestic and foreign authors [8–10]. However, the differential diagnosis of acute myocarditis in the context of COVID-19 causes many difficulties [11]. We would like to emphasize that high levels of troponin I predominated in patients with massive lesions of the lung tissue, which indicated myocardial ischemia associated with hypoxia, even in patients without severe coronary pathology.

In patients with ACS accompanied by ST elevation, signs of massive lung disease (CT4) prevailed, while in the group of patients without ST elevation there were more patients with mild pneumonia (CT1). Among patients with ST-segment elevation and massive lung disease (CT4), death occurred in all patients (100%), regardless of the degree of coronary disease. But still, attention should be paid to the fact that this group was dominated by patients with multivessel lesions of the coronary arteries.

In the group of patients with ST segment elevation and CT3 lung lesions, a high mortality rate is also noted – 66.7%. Regardless of the therapeutic intervention or the need for it, patients died both with and without hemodynamically significant lesions of the coronary arteries.

Among patients with ST elevation and CT2 lung involvement, death occurred in 60%, regardless of the degree of coronary disease.

In the group without ST segment elevation, but with massive lung disease (CT4), death occurred in 84.6%, regardless of the degree of coronary disease. In this subgroup, only 2 patients survived, which was 15.4%. One of the surviving patients did not have significant coronary lesions, the second had a local coronary lesion. Transluminal balloon angioplasty and coronary artery stenting were performed.

In the group without ST segment elevation on the ECG with CT3 lung involvement, death occurred in 75% of patients. These patients died regardless of whether they underwent intracoronary medical intervention or not.

In the group without ST segment elevation group with CT2 lung involvement, death occurred in 82% of patients, regardless of the degree of coronary disease.

Among other groups of patients with lung disease (CT1 and CT0) and ACS accompanied by ST-segment elevation, there is no significant relationship between mortality/survival and the presence of coronary disease.

There were no deaths in the group of patients without ST elevation and CT0.

Our study showed that low LV EF is an unfavorable prognostic sign in patients with COVID-19 disease. According to published data, the rate of hospitalization and/or death in patients with COVID-19 and reduced LV EF is significantly higher than in patients with preserved LV contractility [12].

Patients with reduced EF may have less "reserve" to cope with multiple organ failure that develops in the context of COVID-19. Like other acute illnesses, COVID-19 infection can be accompanied by endothelial dysfunction, electrolyte imbalance, hyperinflammatory response, and hypercoagulability. The cytokine storm observed in severe COVID-19 can lead to further decompensation of an already weakened myocardium [13].

In a number of patients, COVID-19 infection, if severe, is accompanied by acute respiratory damage, which can cause inflammatory and ischemic damage to the myocardium, the development of oxidative stress in the presence of severe hypoxia and anemia. Since the SARS-CoV-2 virus is able to enter erythrocytes and displace hemoglobin, the resulting respiratory failure is exacerbated by a decrease in hemoglobin levels and oxidative damage [14, 15].

In our study, in both groups of patients with lung damage CT4–CT1, who breath independently, there was an inverse relationship between the degree of damage to the lung tissue and oxygen saturation in the blood – the lower the degree of lung damage, the higher the saturation. In the group of patients with ST segment elevation, the effect of mechanical ventilation distorted this pattern, and therefore the results of the analyzes showed multidirectional changes.

CONCLUSION

COVID-19 disease has a pathological effect on many organs and systems, including the cardiovascular system. The mechanisms of myocardial injury are both direct viral damage and the development of a mismatch between tissue oxygen delivery and consumption.

However, myocardial damage in COVID-19, often associated with an increase in troponin I levels, can create significant difficulties for the diagnosis of acute coronary syndrome. In this regard, it is necessary to focus not only on specific markers of myocardial damage and the results of coronary angiography, but also on the presence of typical clinical symptoms, electrocardiography and echocardiography data, and the presence of complications such as acute heart failure and cardiac arrhythmias.

The development of acute coronary syndrome against the background of COVID-19 as the underlying disease is an aggravating factor and significantly worsens the prognosis. First of all, this applies to severe and extremely severe degrees of lung damage – CT3 and CT4.

Cardiac function under conditions of acute coronary syndrome in patients with COVID-19 experiences significant depression, manifested by a sharp decrease in the left ventricular ejection fraction in patients with any degree of lung damage. We can talk about the negative synergy of ischemic damage to the myocardium and lung tissue, which further exacerbates the damage to the heart muscle.

Further accumulation of clinical experience in endovascular treatment of acute coronary syndrome and the development of algorithms for providing medical care to patients with acute coronary syndrome in the context of COVID-19 disease are required.

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