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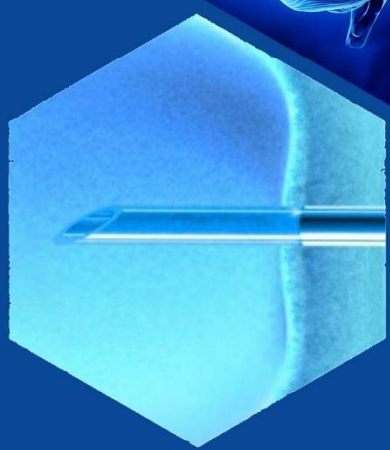
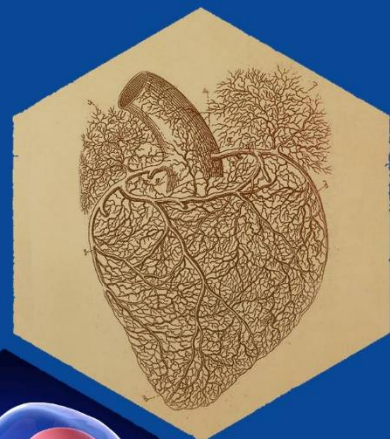
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LABORATORY FEATURES OF COVID-19 ASSOCIATED CARDIOVASCULAR SYNDROME

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ABSTRACT

This article will study the features of laboratory findings in patients with cardiovascular complications associated with COVID-19 infection. A prospective study was carried out. The main group (n=150) consisted of patients with changes in the cardiovascular system related to COVID-19, i.e., newly diagnosed CV pathology, and patients with exacerbation of cardiac pathology against the background of COVID-19. The comparison group (n=154) included patients with COVID-19 without any significant concomitant cardiac pathology. The average age of patients in the main group was 67.7 years (from 47 to 86 years) and in the comparison group - 66.1 years (from 48 to 88 years). The study groups were representative and did not differ statistically, like COVID-19 symptoms and the degree of lung involvement. It was found that at the initial stages and in the dynamics of COVID-19 in combination with CVS pathology, it is characterized by a statistically significant difference in the levels of D-dimer ($p<0.001$), ferritin ($p<0.001$), C-reactive protein ($p<0.05$), sedimentation rate erythrocytes ($p<0.05$), procalcitonin ($p<0.001$), leukocyte indices (neutrophil-lymphocyte index ($p<0.001$), neutrophil nuclear shift index ($p<0.01$) and leukocyte shift ($p<0.001$)). The COVID-19-associated cardiovascular syndrome is characterized by a more pronounced hypercoagulable syndrome, inflammatory tissue damage, and an immune-inflammatory response with a statistically significant difference in specific pro-inflammatory markers and leukocyte indices.

Keywords: COVID-19, SARS-CoV-2, cardiovascular system, laboratory markers, comparative analysis.

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ЛАБОРАТОРНЫЕ ОСОБЕННОСТИ COVID-19 АССОЦИИРОВАННОГО С СЕРДЕЧНО-СОСУДИСТЫМ СИНДРОМОМ

АННОТАЦИЯ

Данная статья посвящена изучению особенности лабораторных находок у пациентов с сердечно-сосудистыми осложнениями на фоне инфицирования COVID-19. Проведено проспективное исследование. Основную группу (n=150) составили пациенты с изменениями сердечно-сосудистой системы (ССС), связанными с COVID-19, т.е. впервые выявленная патология ССС, и пациенты с обострением кардиальной патологии на фоне COVID-19. Группа сравнения (n=154) включала пациентов с COVID-19, однако без какой-либо значимой сопутствующей кардиальной патологии. Средний возраст пациентов в основной группе составил 67,7 лет (от 47 до 86 лет), в группе сравнения – 66,1 лет (от 48 до 88 лет). Группы исследования были репрезентативны и не отличались статистически по характеру симптомов COVID-19 и степени поражения легких. Установлено, что на начальных стадиях и в динамике COVID-19 в сочетании с патологией ССС характеризуется статистически значимым отличием уровней D-димера (p<0.001), ферритина (p<0.001), С-реактивного белка (p<0.05), скорости оседания эритроцитов (p<0.05), прокальцитонина (p<0.001), лейкоцитарных индексов (нейтрофильно-лимфоцитарный индекс (p<0.001), индекс ядерного сдвига нейтрофилов (p<0.01) и лейкоцитарного сдвига (p<0.001)). COVID-19 ассоциированный с сердечно-сосудистым синдромом характеризуется более выраженным гиперкоагуляционным синдромом, воспалительным повреждением тканей и иммунно-воспалительной реакцией со статистически значимым отличием специфических провоспалительных маркеров и лейкоцитарных индексов.

Ключевые слова: COVID-19, SARS-CoV-2, сердечно-сосудистая система, лабораторные маркеры, сравнительный анализ.

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ЮРАК-ҚОН ТОМИР СИНДРОМИ БИЛАН БОҒЛИҚ COVID-19 НИНГ ЛАБОРАТОР ҚОН ТАҲЛИЛИ ХУСУСИЯТЛАРИ

АННОТАЦИЯ

Ушбу мақола COVID-19 инфекцияси фонида юрак-қон томир асоратлари бўлган беморларда лаборатория топилмаларининг хусусиятларини ўрганишга қаратилган. Истикболли тадқиқот ўтказилди. Асосий гуруҳ (n=150) COVID-19 билан боғлиқ юрак-қон томир тизими ўзгаришлари бор беморлардан иборат, яъни, юрак-қон томир патологияси илк маротаба аниқланган, ёки COVID-19 фонида илгари мавжуд булган юрак патологияси кучайгани холат билан беморлар. Таққослаш гуруҳига (n=154) COVID-19 билан оғриган, аммо сезиларли юрак патологияси бўлмаган беморлар киритилган. Асосий гуруҳдаги беморларнинг ўртача ёши 67,7 ёшни (47-дан 86 ёшгача), таққослаш гуруҳида – 66,1 ёшни (48-дан 88 ёшгача) ташкил этди. Тадқиқот гуруҳлари вакили бўлган ва COVID-19 белгиларининг табиати ва ўпканинг шикастланиш даражаси бўйича статистик жиҳатдан фарқ қилмаган. COVID-19 инфекцияси юрак-қон томир тизими патологияси билан бирга кечиши дастлабки

босқичларида ва кейинчалик D-dimer ($p < 0.001$) динамикаси, ферритин ($p < 0.001$), C-реактив оксил ($p < 0.05$), эритроцитлар чўкиши тезлиги ($p < 0.05$), прокальцитонин ($p < 0.001$), лейкоцитлар индекслари (нейтрофил-лейкоцит индекси ($p < 0.001$), нейтрофил ядро силжиш индекси ($p < 0.01$) ва лейкоцитлар силжиши ($p < 0.001$)) динамикаси статистик жиҳатдан сезиларли фарқ килган. Юрак-қон томир синдроми билан боғлиқ COVID-19 инфекцияси гиперкоагуляция синдроми, яллиғланиш ва тўқималарининг шикастланиши, ва ўзига хос иммун-яллиғланиш реакцияси белгилари бўлган лейкоцитар индексларида статистик жиҳатдан сезиларли фарқ билан тавсифланади.

Калит сўзлар: COVID-19, SARS-CoV-2, юрак-қон томир тизими, лаборатория белгилари, қиёсий таҳлил.

Introduction.

The lungs are the main organ affected by COVID-19, but patients infected with SARS-CoV-2 also develop systemic disorders with a wide range of clinical manifestations [1, 2]. One central system this virus affects is the cardiovascular system (CVS) [3, 4].

Pre-existing cardiovascular disease (CVD) increases mortality in COVID-19 patients, and some COVID-19 patients experience cardiovascular complications, including myocarditis, heart rhythm abnormalities, endothelial cell damage, thrombotic events, and myocardial interstitial fibrosis [5, 6, 7]. The underlying pathophysiology of COVID-19-related cardiovascular complications is not fully understood, although direct viral infection of the myocardium has been proposed as a possible mechanism [9, 10].

As a rule, combining COVID-19 with cardiovascular pathology leads to additional difficulties in diagnosis, choice of management tactics, and treatment of patients in emergency conditions [11, 12].

According to many authors, it is necessary to perform diagnostic instrumental and laboratory studies accurately and to distribute patients depending on the degree of severity [4, 5, 7, 13, 14]. Since the primary complaints may be minimal and characterized only by tightness in the chest area and increased heart rate, a prerequisite for hospitalization of COVID-19 and SSc patients is the control not only of symptoms and clinical course of the disease but also of specific blood analysis parameters, namely coagulation function, inflammation indicators, calculation of leukocyte indices, and biochemical indices of liver and kidney function [4, 6, 10, 12].

Therefore, the present study aimed to compare the peculiarities of laboratory findings in patients with cardiovascular complications against the background of COVID-19 infection.

Method and materials.

The study included patients with predominantly baseline moderately severe course of COVID-19. The leading group ($n=150$) consisted of patients with COVID-19-associated SSc changes, i.e., first-diagnosed SSc pathology, and patients with SSc exacerbation on the background of COVID-19. The comparison group ($n=154$) included patients with COVID-19 without any significant concomitant cardiac pathology. The mean age of patients in the main group was 67.7 years (from 47 to 86 years); men were 48, and women - 102. The mean age of patients in the comparison group was 66.1 years (48 to 88 years). There were 50 men and 104 women.

According to the nature of the disease symptoms, the study groups were representative and did not differ statistically in the frequency of occurrence of one or another COVID-19 sign. Typical manifestations of COVID-19 in patients of the study groups were fever noted in 86.2% of cases, dry cough (65.4%), fatigue (43.1%), dyspnea (24.3%), sore throat (15.5%), headache (13.2%), myalgia (13.8%), chills (12.5%), vomiting (7.2%), diarrhea (5.6%), hemoptysis (1.6%), and conjunctival hyperemia (1.6%).

Laboratory blood tests results were studied and analyzed in a comparative aspect, namely coagulation function (D-dimer, ferritin), indicators of inflammatory tissue damage (CRP, SOE, procalcitonin), and indices of general blood analysis with calculation of leukocytic indices (neutrophil-leukocytic, nuclear and leukocytic shift index) were evaluated.

These research materials were subjected to statistical processing using parametric and nonparametric analysis methods. The Student's t-criterion was calculated when comparing mean values in customarily distributed quantitative data populations. Differences in indicators were considered statistically reliable at the significance level of $p < 0.05$.

Results.

Analysis of the dynamics of mean D-dimer values (Fig. 1) showed that in the leading group, both at admission to the hospital and after two weeks of treatment, it was statistically significantly higher. If initially in patients of the leading group in some cases, D-dimer reached 3850 ng/mL, with an average value of $1453,6 \pm 82,3$ ng/mL, then on 14 days of treatment, it was also higher than average values, amounting to $742,3 \pm 33,2$ ng/mL (from 320 to 1250 ng/mL). In the comparison group, the following values were obtained: initially at hospitalization, D-dimer was $1164,7 \pm 63,6$ ng/ml (from 470 to 3040 ng/ml), and by the 14th day of treatment, it decreased to reference values, averaging $428,3 \pm 24,6$ ng/ml (from 180 to 830 ng/ml).

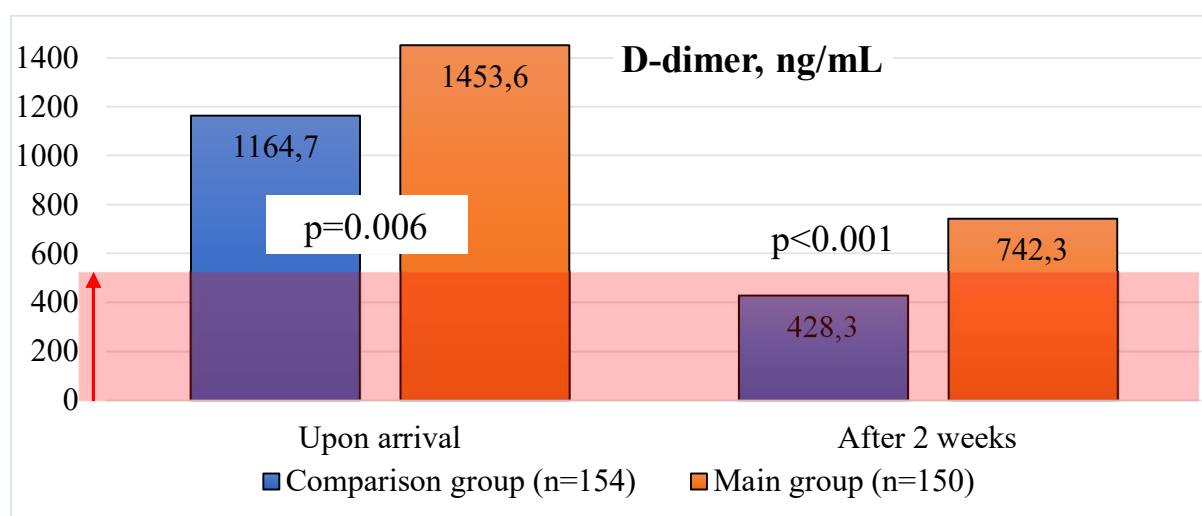


Fig. 1. Dynamics of D-dimer level in COVID-19 patients with CCC pathology (main group) and without CCC pathology (comparison group)

The dynamics of ferritin level (Fig. 2) also confirmed higher coagulation activity in the combination of COVID-19 and cardiovascular pathology.

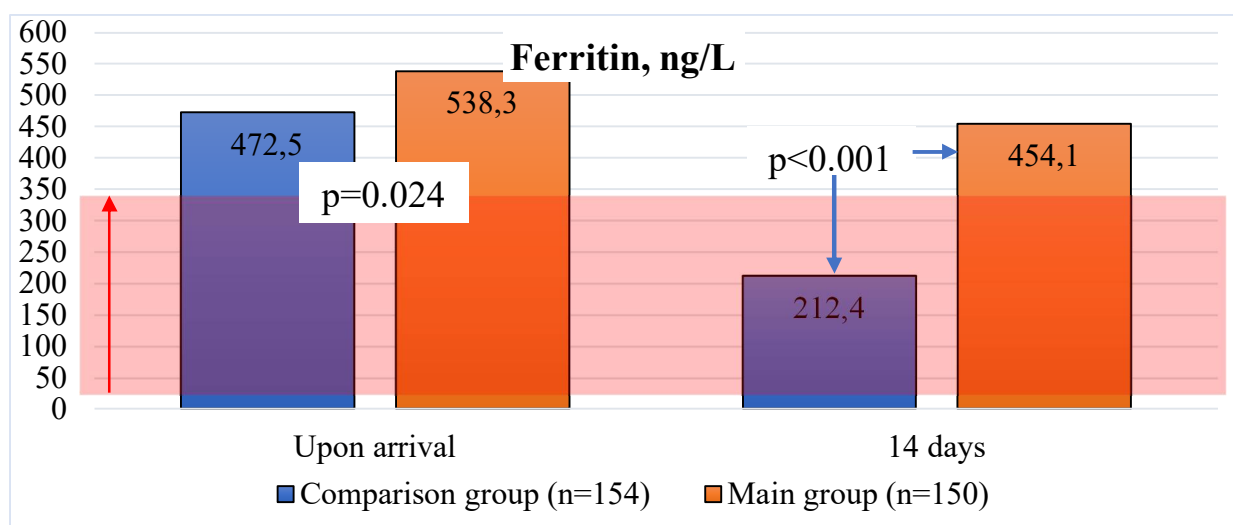


Fig. 2. Ferritin level dynamics in COVID-19 patients with CCC pathology (main group) and without CCC pathology (comparison group)

Thus, if on the day of hospitalization of patients, the mean values of ferritin were equal to 538.3 ± 22.4 ng/ml (from 370 to 1100 ng/ml) and 472.5 ± 18.6 ng/ml (from 310 to 820 ng/ml) in the main and comparison group, respectively, then by day 14 these indices decreased against the treatment background with a significant intragroup difference, however, in the main group the mean ferritin level remained higher than in the comparison group, amounting to 454.1 ± 19.4 ng/mL (from 270 to 710 ng/mL) vs. 212.4 ± 14.3 ng/mL (from 140 to 420 ng/mL) ($t=9.54$; $p<0.001$).

The study of the dynamics of the most frequently determined indicator of inflammatory tissue damage, C-reactive protein (CRP) level (Fig. 3) in the study groups, showed that at the initial stage, more precisely in the acute phase, the combination of COVID-19 with cardiovascular pathology leads to higher CRP figures (51.8 ± 3.4 mg/L; range 14-103) than in uncomplicated COVID-19 or without a comorbid cardiovascular background (42.4 ± 2.6 mg/L; range 12-80), which could be observed in the comparison group, with a statistically significant difference ($t=2.20$; $p=0.03$).

In dynamics, against the background of treatment by 14 days, it is possible to suppress the immuno-inflammatory reaction with a significant decrease in SRB levels in both study groups; it can also be noted that no intergroup difference was found at this stage of the study (Fig. 3).

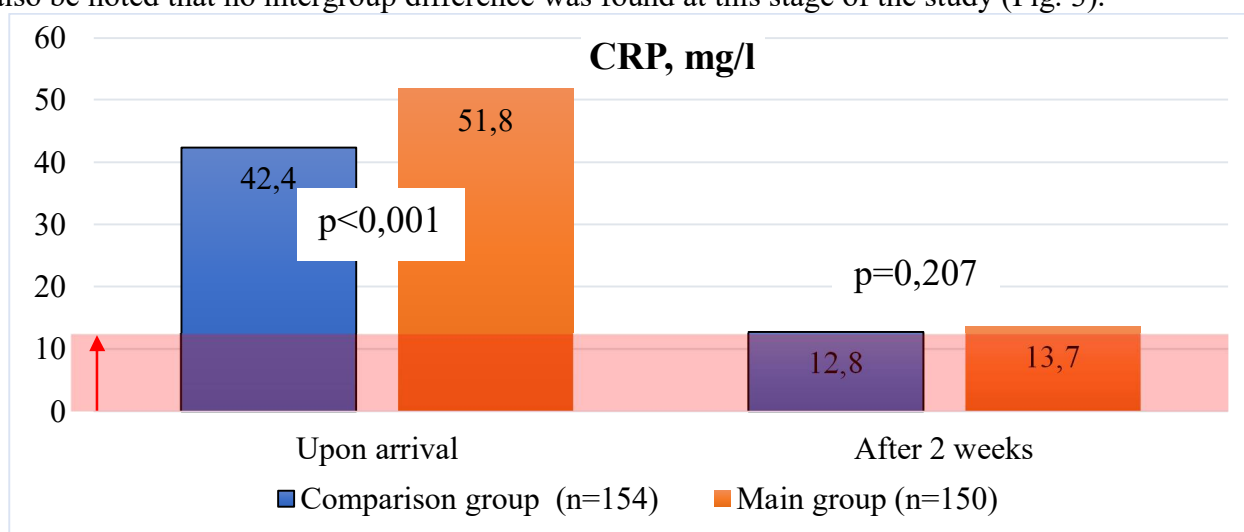


Fig. 3. Dynamics of CRP level in COVID-19 patients with CCC pathology (main group) and without CCC pathology (comparison group)

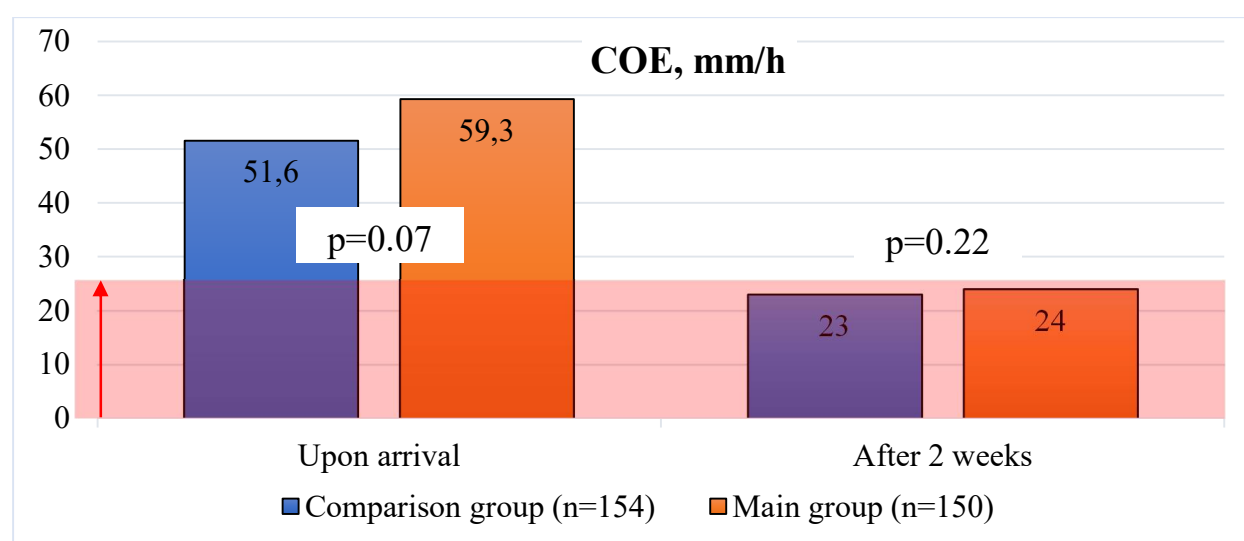


Fig. 4. Dynamics of COE in COVID-19 patients with CCC pathology (main group) and without CCC pathology (comparison group)

Among patients in the main group, in 82.7% (124 out of 150) of cases, the erythrocyte sedimentation rate (ESR) was higher than 40 mm/h with a mean value of 59.3 ± 3.2 mm/h (36-92 mm/h), which indicated the probability of severe inflammation in patients with COVID-19 and cardiovascular pathology (Figure 4). As expected, COE was higher among patients requiring oxygen therapy and intensive care than those without. The comparison group showed lower COE in the first 24 hours at hospital admission with a mean of 51.6 ± 2.9 mm/h (28 to 72 mm/h) but with no significant difference ($t=1.78$; $p=0.07$). At the same time, there were significantly fewer cases with an increase of COE of more than 40 mm/h (72.7%; 112 out of 154; $p=0.025$) than in the main group. In dynamics, it was possible to observe a significant decrease in COE on the background of treatment in both groups. Thus, at the control analysis on the 14th day, the mean values of COE were 23.6 ± 1.4 mm/h (from 12 to 34 mm/h) and 26.4 ± 1.8 mm/h (from 14 to 42 mm/h) in the comparison group and the main group ($t=1.23$; $p=0.22$), respectively (Fig. 4).

It is known that the increase in procalcitonin level indicates the severity of the patient's condition in bacterial infection and sepsis.

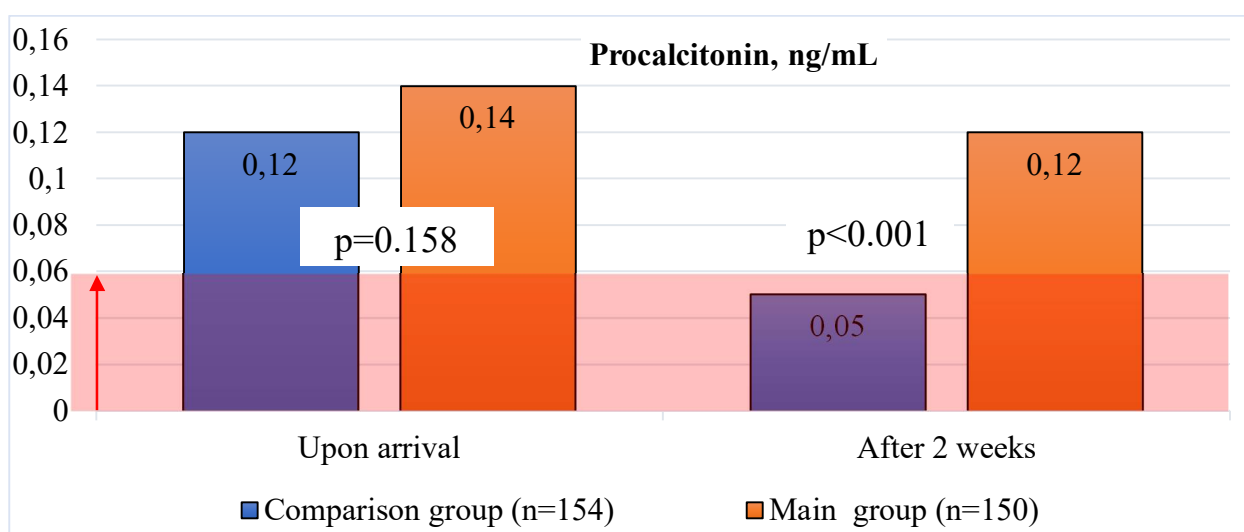


Fig. 5. Dynamics of procalcitonin level in COVID-19 patients with CCC pathology (main group) and without CCC pathology (comparison group)

In our study, the initial procalcitonin values were equal to 0.12 ± 0.01 ng/mL and 0.14 ± 0.01 ng/mL on average in the comparison group and main group, respectively, and ranged from 0.04 to 0.23 ng/mL in the comparison group and from 0.04 to 0.32 ng/mL ($t=1.41$; $p=0.158$), i.e., in all cases, the risk of septic complications development was low (Fig. 5). In dynamics on the 14th day of the disease in the main group of patients with the combination of COVID-19 and cardiovascular complications procalcitonin decreased insignificantly, to 0.12 ± 0.01 ng/mL, range from 0.03 to 0.42 ng/mL. Due to the deterioration of the general condition, 38 main group patients were transferred to ORIT. In the comparison group, procalcitonin decreased with a statistically significant difference both within the group and in contrast to the main group ($t=4.95$; $p<0.001$).

The proportion of patients in the main group with abnormal leukocytic indices was significantly higher than in the comparison group. The patients of the main group had a higher number of leukocytes (mean $6.4 \pm 0.09 \times 10^9/l$) than in the comparison group ($5.8 \pm 0.08 \times 10^9/l$; $t=4.98$; $p<0.001$) and neutrophils (mean $4.6 \times 10^9/l$ vs. $3.40 \times 10^9/l$; $t=11.5$; $p<0.001$). At the same time, lower lymphocyte counts were noted in patients in the main group compared to the comparison group (median $0.7 \pm 0.01 \times 10^9/L$ vs. $1.0 \pm 0.01 \times 10^9/L$; $t=13.4$; $p<0.001$) and platelets ($188.5 \pm 3.4 \times 10^9/L$ vs. $213.0 \pm 4.7 \times 10^9/L$; $t=4.22$; $p<0.001$), respectively (Table 1).

Table 1.

Comparative data on blood cell counts in the study groups at admission

Laboratory parameters	Normal range	Patients group		
		Comparison group (n=154)	Main group (n=150)	t-criteria, p
The number of leukocytes, $\times 10^9/l$	3,5–9,5	5,8 \pm 0,08 (4,1–7,4)	6,4 \pm 0,09 (4,6–8,5)	t=4.98; p<0.001
The number of neutrophils, $10^9/l$	1,8–6,3	3,7 \pm 0,04 (2,5–5,9)	4,6 \pm 0,05 (3,0–7,2)	t=11.5; p<0.001
The number of lymphocytes, $\times 10^9/l$	1,1–3,2	1,0 \pm 0,01 (0,7–1,3)	0,7 \pm 0,01 (0,5–1,1)	t=13.4; p<0.001
The number of thrombocytes, $\times 10^9/l$	180–320	213,0 \pm 4,7 (160–240)	188,5 \pm 3,4 (130–230)	t=4.22; p<0.001

To study the dynamics of the leukocytic formula, the neutrophil nuclear shift index, neutrophil-lymphocyte index, and leukocytic shift index were calculated in the first 24 hours of hospitalization and dynamics on the 14th day of treatment in inpatient settings.

The neutrophil nuclear shift index was calculated as the ratio of the sum of all unsegmented neutrophils (myelocytes, metamyelocytes, and bacilli) to the number of segmented cells.

It is known that COVID-19 infection is accompanied by a nuclear shift of neutrophils to the left, i.e., an increase in the index of more than 0.10 (normal from 0.05 to 0.10).

According to this index, it is possible to assume the severity of the infectious-inflammatory disease. Thus, if it ranges from 0.3 to 1.0 - the patient's condition is of average severity; in cases of more than 1.0 - the disease is severe.

Although clinically and according to CT lung data, the study groups were comparable, and the patient's condition was classified as moderately severe, the average values of the nuclear shift index indicated that a significant part of the patients in the base group were in moderately severe and severe condition, and the comparison group, it was possible to observe cases with a mild degree of infectious-inflammatory process.

In the dynamics on the 14th day, in the main group of the study, according to this laboratory index, the majority of patients had mild and medium severity of both COVID-19 and CCC pathology.

At the same time, patients with COVID-19 clinic progression and worsening heart failure were transferred to ORIT (n=38) and had a nuclear shift index higher than 1.0 at hospitalization and on the 14th day of treatment.

The mean nuclear shift index in the main group was significantly higher than in the comparison group both on day 1 of hospitalization, 0.41 \pm 0.04 (range 0.05 to 1.4) vs. 0.27 \pm 0.02 (vary 0.05 to 1.0) (t=3.13, p=0.002), and on day 14 of treatment, 0.42 \pm 0.04 (range 0.05 to 1.2) vs. 0.28 \pm 0.03 (range 0.03 to 1.2) (t=2.80, p=0.005).

Another predictor of unfavorable outcomes in COVID-19 patients is the estimated neutrophil-lymphocyte index (NLI), which is calculated by dividing the absolute neutrophil count by the total lymphocyte count. Patients with COVID-19 clinical progression and transferred to ORIT had an NLI of 2.0 or more.

Baseline NLI results showed that in the main group of patients, the mean NLI was 1.52 \pm 0.08 (0.5 to 2.80), which was statistically significantly higher (t=5.20, p<0.001) than in the comparison group, where the mean NLI was 1.0 \pm 0.06 (0.5 to 2.2).

The average NLI was decreased in both groups of the study with a statistically significant difference; however, in the main group, it remained higher than in the comparison group due to a more considerable number of patients with progression of pathology against the background of circulatory hypoxia due to concomitant pathology of SSc. The mean values of NLI on the 14th day

of hospitalization were 1.37 ± 0.07 (0.5-2.4) and 1.18 ± 0.06 (0.5-2.4) in the main and comparison groups, respectively ($t=2.06$, $p=0.04$).

The index combining all calculated leuko-formula data, i.e., leukocyte shift index, which is calculated as the ratio of the sum of eosinophils, basophils, and neutrophils to the sum of monocytes and lymphocytes, showed that a relatively significant decrease in lymphocytes and monocytes in COVID-19 associated cardiovascular syndrome, as well as an increase in the number of neutrophils, as expected gave high values of this index in the main group, with a statistical difference from the comparison group.

Mean values of baseline leukocyte shift indices were 1.32 ± 0.07 (0.5-2.6) and 0.86 ± 0.05 (0.5-1.9) in the main and comparison group, respectively ($t=5.35$, $p<0.001$).

In dynamics, this index was reduced in both study groups, and on the 14th day of hospitalization of patients was 1.18 ± 0.06 (0.5-2.1) and 0.73 ± 0.04 (0.4-1.6) in the main and comparison group, respectively ($t=6.24$, $p<0.001$).

Conclusion.

In COVID-19, associated with a cardiovascular syndrome, there is a more pronounced immune-inflammatory response, as evidenced by the levels of leukocyte indices, which were significantly higher in the main group than in the comparison group. At the initial stages and in the dynamics of COVID-19 in conjunction with the pathology of SSc is characterized by a more pronounced hypercoagulable syndrome, inflammatory tissue damage, and immune-inflammatory response with a statistically significant difference in the levels of D-dimer ($p<0.001$), ferritin ($p<0.001$), CRP ($p=0.03$), COE ($p=0.025$), procalcitonin ($p<0.001$), and leukocytic indices: NLI ($p<0.001$), neutrophil nuclear shift index ($p=0.002$) and leukocyte shift ($p<0.001$).

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