

PARTICULARITIES OF CURATION OF ATHLETES WITH PROTRACTED COURSE OF COVID-19

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This paper reviews publications covering the delayed clinical and functional manifestations of SARS-CoV2 among athletes competing at national and international levels; we describe the prevalence of multiorgan failure associated with protracted COVID as registered in sportsmen and people not going in for sports. The review reports the results of a retrospective analysis of data yielded from clinical, instrumental and laboratory tests undertaken by the Russian national team athletes that had COVID-19. We highlight the most informative indicators that reflect the condition of sportsmen with protracted coronavirus infection course, define the approaches making resumption of active training safe and compile the list of the most significant criteria supporting admission to such training and competitions. Lastly, the paper presents the parameters subject to inclusion in the prognostic model (binary logistic regression) describing the dynamics of residual multiorgan failure in athletes, including minors, who have had COVID-19 or viral pneumonia of a different etiology.

Keywords: elite sports, elite athletes, SARS-CoV2 coronavirus infection, post-Covid syndrome, cardiovascular system, myocarditis in sports, multisystem inflammatory syndrome, binary logistic regression

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ОСОБЕННОСТИ КУРАЦИИ СПОРТСМЕНОВ ПРИ ЗАТЯЖНЫХ ВАРИАНТАХ ТЕЧЕНИЯ COVID-19

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Представлен обзор публикаций, посвященных отсроченным клиническим и функциональным проявлениям коронавирусной инфекции SARS-CoV2 в контингенте спортсменов национального и международного уровней; дана характеристика распространенности полиорганных поражений при затяжном течении коронавирусной инфекции среди спортсменов, а также лиц, не занимающихся спортом. Описаны результаты ретроспективного анализа данных клинико-инструментального и лабораторного тестирования спортсменов сборной команды России, перенесших COVID-19. Выделены наиболее информативные показатели, отражающие состояние спортсменов с пролонгированным течением коронавирусной инфекции; определены подходы к безопасному возобновлению спортивной деятельности, сформирован перечень наиболее значимых критериев допуска к тренировочному и соревновательному процессу. Определены параметры, подлежащие включению в прогностическую модель (бинарную логистическую регрессию) динамики резидуальных полиорганных нарушений у спортсменов, в том числе несовершеннолетних, перенесших коронавирусную инфекцию или вирусную пневмонию иной этиологии.

Ключевые слова: спорт высших достижений, элитные спортсмены, коронавирусная инфекция SARS-CoV2, постковидный синдром, сердечно-сосудистая система, миокардит в спорте, мультисистемный воспалительный синдром, бинарная логистическая регрессия

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The effect of COVID-19 reverberates through all aspects of our lives, including sports, a socially significant area. In addition to the risks associated with the severity of acute symptoms and the possibility of complications, SARS-CoV2 often entails long-term consequences that may hinder professional activities of recovering patients, including athletes competing at national and international levels, or elite and sub-elite athletes, in the terminology of the IOC. Mitigation of these risks requires complex (multidirectional) measures taken not only during the acute course of the disease [1], but also generally when there is a risk of an epidemic and when the patient is followed-up after recovery and resumes training. Currently, seeking to develop an effective tool preventing severe delayed effects of SARS-CoV2, researchers continue searching for early markers of both latent and persistent damage to various functional systems. These efforts are especially significant in the field of sports and, above all, for young athletes. First of all, it is not fully understood how high-intensity physical exertion affects the course of coronavirus and other viral infections, which disallows identification of the risk group in the cohort of seemingly healthy individuals with high functional state characteristics and effective adaptive and compensatory mechanisms. In addition, there are no well-described concrete options of prognosis of COVID course and outcomes in athletes, and there is no specific data on the most probable risks of complications depending on age, gender and type of sports activity, on the severity and nature of the clinical course of the disease. All these aspects add urgency to the investigation of coronavirus infection's effect on the health of athletes and search for predictors of complications (including their delayed varieties) manifesting, first of all, in the cardiovascular system.

COVID-19 course variants

The WHO defines COVID-19 as a disease manifesting as a severe acute respiratory syndrome caused by the SARS-CoV2 coronavirus. The infection caused by the new virus was first reported on December 31, 2019, and on March 11, 2020 the WHO announced the pandemic [2].

Initially, COVID-19 was considered an acute infection with full resolution of manifestations (mildly and moderately severe) within 2-3 weeks. However, over time, there appeared a growing body of evidence confirming symptoms persisting for a longer time, up to 6 months or even longer.

On October 30, 2020, National Institute for Health and Care Excellence (NICE) suggested differentiating between following forms of COVID-19:

- 1) acute COVID-19, with subjective and objective manifestations lasting up to 4 weeks;
- 2) ongoing symptomatic COVID-19, lasting from 4 to 12 weeks;
- 3) post-COVID syndrome [3].

Definition of post-COVID syndrome

Initially, the term "post COVID-19" was introduced to describe the condition that typically develops in people with suspected or confirmed SARS-CoV-2 3 months after the onset of the disease, with symptoms that cannot be attributed to another disease lasting at least 2 months [3]. The ICD-10 code U09.9 for post COVID-19 condition was legitimized in September 2020; in ICD-11, it is RA02, currently used to describe only unspecified conditions. The respective complex of symptoms complex may manifest after acute form of COVID-19 or persist post recovery. The most common symptoms are fatigue, shortness of breath, cognitive dysfunction etc.; as a

rule, they complicate daily activities and may change over time or recur [4].

Prevalence of multiple organ symptoms of the post COVID-19 syndrome

According to a statistical review and meta-analysis of 60 studies that jointly included 257,348 people, male patients tend to suffer the post COVID-19 syndrome more often than female, and the symptoms may recur in patterns [5]. Table 1 presents the predominant attributes of the syndrome.

Cardiac manifestations of SARS-CoV-2 infection and post COVID-19 syndrome

The current and expected state of the cardiovascular system (CVS) largely conditions achievements in sports, cyclic and competitive in particular; CVS is one of the most significant, but at the same time vulnerable systems ensuring general and special physical performance, which means that effectiveness of the CVS is one of the most important factors shaping athletic performance [6], when the former is reduced, the latter is limited, with ensuing prospect of further professional growth, including the risks of premature termination of sports career or development of catastrophic cardiac events, i.e. sudden cardiac death, or SCD. A past coronavirus infection in the background may complicate differential diagnosis of adaptive remodeling, identification of signs of stress cardiopathy and post-inflammatory myocardial damage [7, 8].

Our preliminary observations (data from the Ogarev University Medical Institute) show that hearts of almost 40% of athletes who had COVID-19 have undergone persistent changes, both morphometric (heart size) and functional (left ventricular myocardial contractility), as registered with echocardiography (Figure 1).

In the contingent of athletes we observed, the signs of SVR maladaptation registered in the controlled exertion tests (decreased tolerance to controlled physical load, hemodynamic shifts, electrical instability of the myocardium) were virtually consistent but significantly less stable.

The laboratory indicators that are diagnostically significant in the cases of myocardium inflammation and stress-induced SVR changes also varied significantly in their intensity and stability, as shown by the biochemical markers of myocardial damage. Compared to their less trained fellows, professional athletes with signs of maladaptive SVR remodeling more often had higher levels of cardiac fraction of creatine phosphokinase and troponin I: 6.3% vs 27%, respectively ($p < 0.05$). In addition, 65% of the observed athletes had the cortisol levels going up during onset of COVID-19, with their subsequent return to the reference values in 55% of them. In 80% and 58% of the athletes, we registered pronounced COVID-19-induced changes in the levels of CPK and LDH (recovered subsequently in 65% and 60% of them).

The mentioned markers can reflect not only the load on the mechanisms supporting the current state of myocardial function, but also predict the vector of subsequent changes with a certain degree of probability (factoring in the dynamics recorded in a month or more after verification of the disease).

Manifestations of the current long-term variants of COVID-19 and post COVID-19 syndrome vary (intra-group differences) and often have a wavy pattern, which translates into a wide range of manifestations and their associations, as well as heterogeneity of dynamics. It should be clarified that the data on the prevalence of prolonged Covid-19 is currently

Table 1. The frequency of occurrence of signs of post COVID-19 syndrome (according to Alkodaymi et al.)

Observation period	3–6 months	6–9 months
Sign		
General and pulmonary manifestations		
Fatigue	32% (<i>n</i> = 7268, 25 studies)	36% (<i>n</i> = 8191, 19 studies)
Shortness of breath	25% (<i>n</i> = 8132, 28 studies)	–
Cough	15 % (<i>n</i> = 7539, 22 studies)	–
Alopecia	9% (<i>n</i> = 478, 4 studies)	10% (<i>n</i> = 4276, 5 studies)
Neuropsychic manifestations		
Sleep disorders	24% (<i>n</i> = 4369, 8 studies)	29% (<i>n</i> = 242000, 12 studies)
Anxiety	21% (<i>n</i> = 4324, 7 studies)	23% (<i>n</i> = 240756, 7 studies)
Depression	14% (<i>n</i> = 4099, 5 studies)	23 % (<i>n</i> = 4377, 6 studies)
Clouded sensorium	22% (<i>n</i> = 466, 5 studies)	22 % (<i>n</i> = 854, 4 studies)
Cognitive disorders	14% (<i>n</i> = 670, 6 studies)	15% (<i>n</i> = 1987, 5 studies)
Headache	12% (<i>n</i> = 5699, 12 studies)	14% (<i>n</i> = 7170, 13 studies)
Anosmia	9% (<i>n</i> = 5400, 16 studies)	15 % (<i>n</i> = 6596, 17 studies)
Ageusia	8% (<i>n</i> = 5127, 13 studies)	13% (<i>n</i> = 6505, 16 studies)
Cardiovascular manifestations		
Exercise intolerance	19% (<i>n</i> = 5203, 6 studies)	45% (<i>n</i> = 850, 5 studies)
Rapid heartbeat	14 % (<i>n</i> = 5401, 8 studies)	14% (<i>n</i> = 4735, 7 studies)
Chest pain	11 % (<i>n</i> = 5758, 15 studies)	12% (<i>n</i> = 4318, 10 studies)
Musculoskeletal system disorders		
Joint pain	14% (<i>n</i> = 4829, 8 studies)	23% (<i>n</i> = 5288, 8 studies)
Myalgia	12% (<i>n</i> = 5453, 10 studies)	19 % (<i>n</i> = 3490, 9 studies)
Gastrointestinal tract disorders		
Diarrhea	10% (<i>n</i> = 4908, 7 studies)	5% (<i>n</i> = 3318, 8 studies)
Nausea	8% (<i>n</i> = 480, 3 studies)	4% (<i>n</i> = 3419, 8 studies)

insufficient, but, judging by a number of studies, more than 144 million people live with this condition in the world, and most of them suffer from symptoms that generally affect the quality of life and functional status negatively [9].

The echocardiographic evidence from our study correlates, to a certain extent, with the previously registered COVID-19 consequences, including autonomic dysregulation associated with aortic rigidity, as well as ventriculo-aortic insufficiency and left ventricular dysfunction, all seen even 6 months after discharge from the hospital. These disorders may be caused by prolongation of the infection [10].

The published data also suggest that vaccination of people with post COVID-19 syndrome can prevent progression of persistent symptoms. In some cases, specific immunoprophylaxis can even completely eliminate the symptoms, with the probable explanation therefor being the cumulative protective effect of multiple vaccinations in the context of prevention of recurring viral infections. In UK case-control study that involved over 4,000 patients, two doses of the vaccine reduced the prevalence and severity of persistent cardiac manifestations of COVID-19 (compared with the unvaccinated) and often made the course asymptomatic [11].

The TriNetX retrospective cohort study undertaken in the US gave similar but more significant results. The researchers examined over 81 million electronic medical records seeking to evaluate the delayed complications of COVID-19, i.e. those identified 6 months after confirmed SARS-CoV-2 infection, and, in particular, uncover the probable effect the number of vaccinations (1 vs 2) has thereon. A single dose of vaccine was associated with a lower subsequent risk of admission to the intensive care unit, intubation or lung ventilation, hypoxemia, oxygen demand, respiratory failure, hypercoagulopathy,

venous thromboembolism, seizures, psychotic disorders and hair loss; for most of such outcomes, the risk was even lower for the revaccinated, who, possibly, have the advantage of a less severe course and smaller chance of re-infection [12]. Another study was conducted in France; it involved 910 patients already diagnosed with long COVID-19 and found that vaccination translated into a milder post COVID-19 syndrome and improved quality of life 2 months after administration of the vaccine. In effect, vaccination doubled the frequency of remission of the persisting symptoms [13]. However, such studies need to be continued.

There is also a certain shortage of publications documenting aggravation of cardiovascular dysfunction in patients with post COVID-19 syndrome that developed against the background of chronic diseases of the CVS. To a large extent, the reason behind this situation lies in the insufficiency of understanding of how SARS-CoV-2 itself and the acute pathological processes it initiates affect functioning of the CVS and the progression of its chronic disorders.

In case of COVID-19, most medical interventions are trials designed to compensate and prevent aggravation of acute clinical manifestations and to prove the efficacy of drug therapy. In the field of cardiology, research efforts were mainly aimed at finding cellular mechanisms driving the said manifestations and, accordingly, at developing sound diagnostic and therapeutic strategies. The respective studies relied on the activities of multicenter/national research groups (e.g., the European Society of Cardiology), which prepared a scientific statement on COVID-19-related cardiovascular complications, covering myocardium and pericardium diseases. Then, the focus shifted to research projects dedicated to promotion of active lifestyle, which

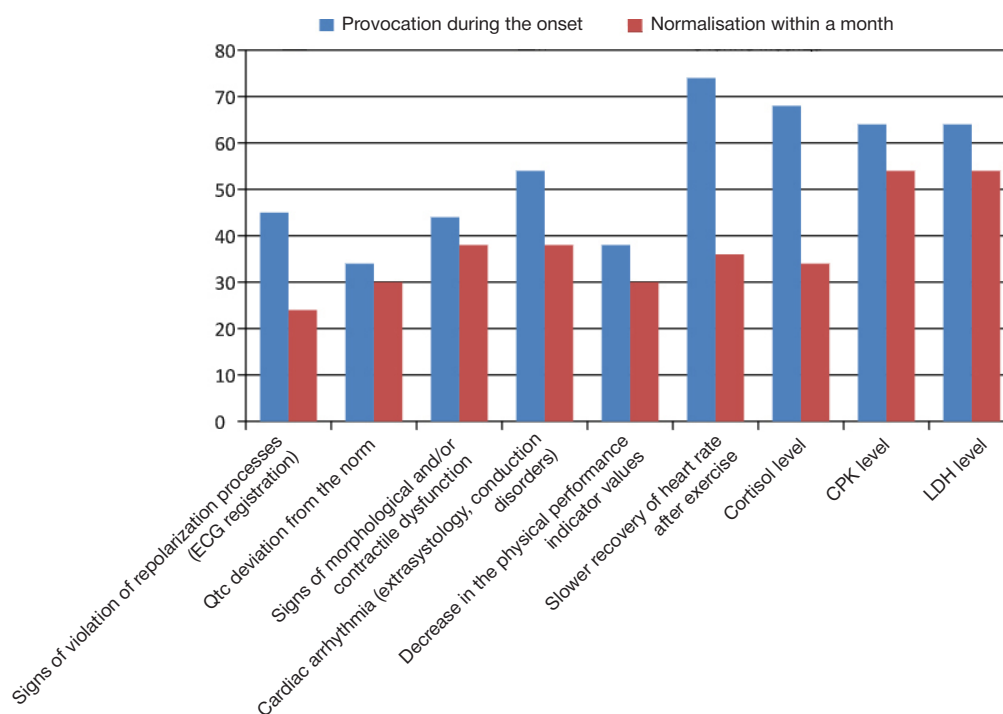


Fig. Dynamics of the CVS clinical and laboratory markers in athletes who had coronavirus infection

allows realization of the preventive aspects of measures taken to counter chronic pathology. In view of the duration of the COVID-19 pandemic and the probabilistic risks of other global shocks caused by viral invasion, it is necessary to widely implement national protocols aimed not only at elimination of the immediate threats, but also at creation of long-term behavioral determinants that reduce the prevalence of CVD [14]. In our opinion, predictive models based on the mathematical analysis of valid indicators reflecting the state of essential systems of the body can play a significant role in increasing the effectiveness of this activity of the world community.

Manifestations (reflected in instrumental and laboratory tests) confirming a SARS-CoV-2 and post COVID-19 syndrome diagnosis

In some COVID-19 patients, cardiac symptoms (e.g., chest pain, shortness of breath, fatigue and palpitations) persist for several months after the disease, but there is also another scenario: gradual, prolonged manifestation of signs of damage to the organs, including myocardium, that were detected only with instruments and laboratory testing [15–19].

One of the main problems is the development of myocarditis, since its course can be asymptomatic or subclinical but it still remains arrhythmogenic, which translates into a pronounced risk of sudden cardiac death [20, 21]. Moreover, physical exertion during the acute phase of myocarditis can aggravate myocardial damage and trigger progression to fatal arrhythmias [20–23]. In this connection, there have been developed the Return to Play recommendations that suggest the routine of resuming sports activities after coronavirus infection [24, 25].

In addition, it is acknowledged necessary to differentially diagnose inflammatory myocardial damage and its sports remodeling [20, 22].

A prospective multicenter observational study that involved over 19000 US athletes included a cohort of SARS-CoV-2-positive patients (*n* = 3018) that had their CVS examined. ECG revealed changes in 0.7% (21/2999) of them, EchoCG — in 0.9% (24/2556) [26].

A systematic review analyzed data from 16 studies, which involved 890 COVID-19 convalescents; 14% of them were diagnosed with myocarditis, and late gadolinium enhancement (LGE), a contrast technique for MRI, allowed detecting myocardial fibrosis in 20.5% of the examined [22]. Among the sample, 35.5% were athletes, and 17.1% had documented MRI-registered changes, including myocarditis in 2.5% of cases, while among people not going in for sports the results were 62.5% and 23.9%, respectively [27].

Another, more representative review presents an analysis of articles that cover data on 3131 athletes who had COVID-19. In the studies, from 0 to 15% of the athletes were diagnosed with myocarditis, pericardial effusion was the diagnosis in 0–58% of cases and MRI later contrast enhancement allowed detecting changes in 0–46% of the participants [28]. Obviously, the significant differences result from the different inclusion criteria (e.g., timing of examinations considering the time of confirmation of the infection status), lack of a control group and various results assessment methods, although MRI is the most sensitive and specific non-invasive method for diagnosing myocarditis in any category of persons, including those who had COVID-19. The diagnostic value of the method is believed to grow 1–3 months after the disease [29].

Table 2. The degree of risk of COVID-19-induced CVS damage when the changes are detected with instruments and laboratory tests

Sign risk	Reduced performance	High CPK level	Rhythm and conduction disturbances	Increased cortisol level	Heart rate recovery time extension against load
Relative, cu	3.75	2.34	3.77	2.42	3.86
Absolute, cu	0.6	0.22	0.38	0.35	0.58

MRI-detected changes — focal myocardial fibrosis, both post-myocarditis and post-ischemic, — are quite common among elite athletes (24–38% of cases); these manifestations are not associated with coronary pathology, and it is necessary to monitor the dynamics as they can trigger life-threatening arrhythmias [30–32].

A small study [33] that involved 26 athletes presents, as we believe, gives data closest to the real values. The athletes had COVID-19 and underwent cardiac MRI thereafter: 4 (15.4%) of them showed signs of myocarditis, 8 (30.8%) had late contrast enhancement without increased T2, which indicates previous damage/fibrosis of the myocardium. Overall, among the recovering athletes, MRI signs of myocarditis in combination with other criteria allowing establishing the respective diagnosis, were registered in 0 — 2.3–7.6% of cases, with other signs — pericardial effusion, myocardial contractility disorder, abnormal movements of walls or septum — detected more often [34–38]. It was established that myocarditis is more common in male athletes and in persons under 21 years of age with COVID-19-associated multisystem inflammatory syndrome (MSIS) [26, 35, 36, 39–43].

Daily monitoring with a Holter monitor uncovered supraventricular and ventricular extrasystoles in athletes ($n = 90$) who had asymptomatic or mild COVID-19 (53.3% and 52.2% of cases, respectively) [44].

The level of troponin, one of the biochemical markers of myocardial damage, exceeded the upper limit of the reference range in 0.9% (24/2719) of athletes, but a clear connection between biomarker levels and changes detected with EchoCG and MRI was not reported. There is a number of other studies [26, 28, 33, 34, 45] that also mention no such connection, which once again confirms the problematic nature of differential diagnosis of post COVID-19 changes of the myocardium and its transformation caused by sports loads.

Thus, there are very diverse data on myocardial changes in athletes after COVID-19. Young athletes, in general, tend to run a lower risk of cardiovascular complications; some athletes may have no clinical symptoms of involvement of heart in the pathological process [27, 28]. Most authors agree that subjective manifestations (complaints of weakness, fatigue, poor performance) are not typical for recovering athletes. Patients with increased troponin levels, ECG abnormalities that suggest myocarditis (diffuse inversion of the T wave, ST segment elevation without reciprocal depression, expansion of the QRS complex), and/or echocardiographic anomalies typical of myocarditis (ventricular wall movement anomalies, decreased myocardial contractility, pericardial effusion, ventricular dilation, abnormal ventricular tension) are referred to a cardiologist for consultation and, with respective indications, MRI scanning [34]. In the context of detection of myocardial electrical instability and arrhythmias, Holter monitor is more significant diagnostically than standard ECG; EchoCG in combination with biomarkers (troponin I/T) provides the maximum amount of information relevant to the diagnosis of myocardial damage in athletes that had COVID-19.

Thromboembolic complications are registered in 27–31% of COVID-19 patients [46]. Factoring in the risk of coagulopathies, some cases require determining the parameters of clotting (D-dimer, international normalized ratio (INR), activated partial thromboplastin time (APTT), prothrombin time) [24, 45]. Before the COVID-19 pandemic, D-dimer level above 400 ng/ml was registered in 7.9% of rugby players, and the median was 231 ng/ml (215; 270); coronavirus infection doubled the number of such cases: D-dimer above 400 ng/ml is seen in 17.3% of cases, and the median has grown to 270 ng/ml (215; 318) [47].

At the same time, it should be remembered that sports injuries inherent in rugby can also drive the D-dimer level up [47].

A variety of psychological and autonomic disorders, the symptoms of asthenia, are also typical manifestations of the post COVID-19 syndrome [48, 49].

Athletes may experience post-exercise weakness resembling chronic fatigue or fibromyalgia [50]. Some sportsmen complain of shortness of breath and palpitations, including when upright, which requires checking for the postural tachycardia syndrome [51].

It should be noted that organizational approaches to diagnostic search in the sample of athletes with cardiac symptoms differ from the those practiced generally, which are sufficient for verification of the post COVID-19 syndrome. The objective characterization of the type and severity of clinical manifestations is the decisive component of the diagnosing, and patients with suspected cardiovascular system damage mandatorily undergo the following tests:

- 1) basic laboratory testing (full blood count, basic metabolic panel, troponin T, C-reactive protein tests);
- 2) ECG;
- 3) EchoCG;
- 4) Holter monitoring;
- 5) comprehensive assessment of the state of respiratory system; checks for orthostatic hypotension and postural tachycardia are also recommended [34].

A consultation with a cardiologist is recommended to patients (including recovering patients) that:

- 1) have abnormal results of cardiological testing;
- 2) have a documented pathology of the CVS with newly emerged or worsening symptoms;
- 3) have confirmed COVID-19-induced cardiac complications;
- 4) have cardiac and/or respiratory symptoms that cannot be explained in any other way [34].

Methodological aspects of safe return to professional sports

Currently, there are various clinical recommendations regulating resumption of physical activity after a coronavirus infection. All the adopted protocols stratify athletes in accordance with the course of the disease. For example, for asymptomatic COVID-19 the recommendation is to refrain from physical exertion for at least 2 weeks, the monitoring method of choice is ECG at rest, values beyond the normal range therein is a reason for EchoCG and exercise tests as a minimum [52, 53].

A similar, that is, rather conservative, but at the same time more multimodal approach to assessment of the possibility of resumption of training activities is being developed by the American College of Cardiology [34, 52, 54]. This approach includes the so-called "triad of tests": 12-lead ECG, EchoCG, cardiac troponin level measurement (by a highly sensitive method). However, almost in all cases it is the exercise testing that is crucial for the decision to resume or refrain from training activity; this is the position of the cardiological expert communities of the West as consolidated in 2015 [56].

The efficacy of protocols that include MRI requires a separate discussion: it enables verification of damage to the myocardium and pericardium associated with COVID-19 [33, 36, 37, 39], but the analysis of data from the American Professional cohort ($n = 789$) and the multicenter register of cardiac outcomes in athletes ($n = 3018$) demonstrated low detection of myocarditis in athletes (0.6–0.7%) [26, 40]. One of the subsequent studies aimed at summarizing the data on the diagnostic significance of MRI screening among sportsmen

showed that myocarditis occurred in 2.3% of cases [35]. Experts of the European Association of Preventive Cardiology (EAPC) recognized troponin level growth as an indication for a heart MRI with contrast [23]; for athletes, admission to a hospital or documented heart damage should call for expansion of the diagnostic methods with primarily biochemical tests (brain natriuretic peptide and its terminal fragment) [56].

In other words, professional communities have agreed on the need for triad screening regardless of the nature of symptoms. In addition, they deemed expedient to monitor athletes under the age of 21 after a coronavirus infection; observation lasting up to 8 weeks can enable timely diagnosis of MSIS [55].

In general, in case of myocarditis it is necessary to follow the previously adopted recommendations, since currently there is no evidence that myocarditis associated with COVID-19 clinically and pathophysiologically differs significantly from its other forms [47]. Resumption of training is recommended after 3-6 months, with the function of the left ventricular and levels of cardiac biomarkers back to normal, no clinical manifestations, Holter monitoring and ECG-registered disorders in the context of exercise tests [23, 56].

The approaches to building a predictive mathematical model based on the valid predictors of multi-organ lesions peculiar to long COVID-19

Forming the approaches to predicting the levels of risks associated with coronavirus infection, we used methods of multidimensional statistical analysis, binary logistic regression in particular. Logistic regression is a type of multiple regression enabling analysis of the relationship between several independent variables (also called regressors or predictors) and a dependent variable. Binary logistic regression is used when the dependent variable is binary (that is, it can take only two values).

The necessary calculations were carried out as part of the retrospective analysis of medical records of 59 National team athletes that had COVID-19; they underwent in-depth medical examinations seeking readmission to sports activities six months after resumption of the training process. The

characteristics meeting the requirements for inclusion in the mathematical model and thus most promising were: exercise tests aimed at registration of performance deterioration, heart rate recovery time extension, episodes of increased levels of CPK and cortisol (Table 2).

Such factors as reduced physical performance and extended post-exercise recovery period corresponded to a higher level of absolute risk. The possible explanation for the slower dynamics of restoration of performance during the recovery period lies in the resistance of disorders of recovery processes associated with the viral invasion (figure 1).

CONCLUSION

The published papers report a wide range of frequency of occurrence of COVID-19-induced CVS damage in the recovering athletes, with those under the age of 25 running a lower risk of cardiac symptoms associated with coronavirus than their older counterparts.

Most athletes that recovered from asymptomatic or mild COVID-19 had the biomarkers of myocardial damage, as well as indicators of ECG and EchoCG within the reference values range. If the athlete has complaints of cardiac profile and/or suffered the diseases in moderate and severe forms, the decision on resuming sports activities can be made only based on the data of cardiological examination, including, as a minimum, ECG, EchoCG and troponin level measurement.

The following diagnostic criteria applied to gauge the post-COVID-19 damage to the myocardium are the most informative: diffuse inversion of the T wave, ST segment elevation without its reciprocal depression, expansion of the QRS complex, new non-physiological ECG patterns; among the indicators registered with EchoCG — anomalies of the left ventricular wall movement, decreased myocardial contractility, ventricular dilation, abnormal ventricular tension, pericardial effusion, new disorders and aggravation of the existing ones.

The inclusion of these indicators in a mathematical model — logistic regression — opens up prospects for predicting the risks of post COVID-19 syndrome and multi-organ lesions.

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