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COVID-19 Sequelae: Long-Term Impairments in Physical Performance

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Abstract

Introduction: Five years ago, the world faced the COVID-19 pandemic. The rapidly spreading disease necessitated extensive research by scientists and physicians on symptom progression, therapeutic options, and, most importantly, complications associated with the infection. Some of the most common post-COVID-19 complications affect the ability to return to physical activity and subsequent physical performance. Although the acute phase of infection tends to present with mild symptoms in physically active individuals, the resulting complications can be far more disruptive, significantly impairing the recovery process and the ability to resume training.

Aim of the study: The aim of this study is to review the available literature to analyze potential COVID-19 complications and their impact on physical performance and return to play (RTP).

Materials and methods: A comprehensive review of the literature available across reputable databases including PubMed, Scopus, Google Scholar, Embase and Cochrane Library. The study was conducted using a systematic search of keywords, including "COVID-19", “post-

COVID”, “return to play”, "myocarditis", "athletes," "long COVID-19", "physical performance", “Fatigue” and “exercise tolerance”.

Results and conclusion: Physically active individuals tend to experience a milder acute phase of infection; however, they are also affected by complications resulting from the disease. Cardiovascular and respiratory complications, along with chronic long-COVID syndrome, reduce physical performance and delay the return to peak fitness. It is crucial to reintroduce training gradually and conduct thorough monitoring of the patient's condition, despite the desire for a rapid return to exercise.

Keywords: COVID-19, myocarditis, athletes, long COVID-19, physical performance, Fatigue, exercise tolerance.

I. Introduction

The SARS-CoV-2 virus, first detected in Wuhan, China in 2019, profoundly impacted the world and mobilized scientists and medical professionals to conduct extensive research aimed at mitigating the negative consequences of the pandemic. As a result, there is now a vast body of literature dedicated to the etiopathogenesis of the disease, its clinical course, complications, and treatment.

In this study, we aim to focus on the complications that COVID-19 may cause in a specific population- athletes. These individuals rely heavily on their physical fitness and endurance, and any impairment in these areas can have severe consequences for their professional careers. Research indicates that maintaining physical activity predisposes individuals to a milder course of the disease and reduces the risk of post-COVID-19 complications [1]. Given this, one might expect that athletes are not particularly vulnerable to adverse outcomes following infection. Indeed, studies have shown that athletes generally experience mild and self-limiting symptoms of COVID-19 [2] and had one of the lowest hospitalization rates, with only 0.2% requiring specialized medical care [3].

However, the complications associated with the disease often persist long after the acute phase, significantly hindering athletes' ability to return to their pre-illness level of performance. In this article, we will analyze the available literature on the most commonly observed post-COVID-19 complications in athletes, their health-related and professional consequences, and the key insights that can be drawn five years after the pandemic.

II. COVID-19 Overview

The SARS-CoV-2 virus belongs to the coronavirus family, with a strong affinity for the respiratory system. It causes an acute respiratory infection, with primary symptoms including fever, cough, and shortness of breath. Infected individuals also frequently report fatigue, myalgia, anosmia, ageusia, and rhinorrhea. Symptom severity varies widely, ranging from mild to severe cases that may result in death [4].

The highest risk of severe COVID-19 progression is observed in individuals over 65 years of age, obese patients, pregnant women, and those with persistent conditions such as diabetes, asthma, chronic kidney disease, or immunodeficiency. The risk of thromboembolic complications is also significantly increased.

Treatment depends on symptom severity, the patient's risk factors for severe disease, and overall clinical condition. In high-risk cases, antiviral therapy is initiated within five days of symptom onset. Antiviral agents used in COVID-19 treatment include molnupiravir (for patients not requiring oxygen therapy) and remdesivir, which is approved for those needing supplemental oxygen. Other therapeutic options include nirmatrelvir and human recombinant monoclonal antibodies.

Additionally, immunomodulatory therapy may be employed, including corticosteroid regimens, anti-cytokine agents, and kinase inhibitors. In patients with a low risk of severe disease progression, symptomatic treatment with over-the-counter anti-inflammatory medications is recommended [5].

Long COVID, defined as a syndrome of symptoms persisting for more than three months after the initial infection, affects between 30% and 80% of patients. This condition encompasses a broad spectrum of symptoms, including fatigue, chest pain, dyspnea, and exercise intolerance. Additional complications of SARS-CoV-2 infection include myocarditis and neurological disorders [5].

Currently, multiple generations of SARS-CoV-2 vaccines are available. According to WHO recommendations, patients at high risk of severe or complicated disease should receive two vaccine doses, administered 6 to 12 months apart. In young, healthy individuals, routine

administration of a second vaccine dose is not recommended [5]. While the vaccine does not prevent infection, it significantly reduces the severity of symptoms [6].

III. Cardiac complications

At the beginning of the COVID-19 pandemic, cardiovascular complications raised significant concern among physicians. The SARS-CoV-2 virus induces a strong inflammatory response that disrupts the functioning of the respiratory and circulatory systems [7], leading to hypoxia and impaired blood flow [8], which indirectly causes myocardial injury. A similar mechanism is observed in sepsis and ARDS [9][10]. Additionally, studies [11] have identified the impact of SARS-CoV-2 on the ACE-2 protein located in cardiomyocytes. This raised concerns that the virus might target the heart muscle. However, later research [12] refuted this theory. Tissue samples from the heart were collected, RNA was isolated, and the presence of viral material in the tissues was traced using in situ hybridization. The findings revealed that the source of infection was not within the cardiomyocytes but in the interstitium and infiltrating macrophages.

There are two mechanisms leading to myocardial ischemia: Type 1, in which the primary factor is plaque rupture/erosion [13], and Type 2, also known as demand ischemia, where the issue is insufficient oxygen supply due to increased demand and/or transport difficulties [14]. Such conditions occur in hypoxia, hypoperfusion, and tachycardia. Type 2 ischemia is the characteristic mechanism for COVID-19, although both types of ischemia have been confirmed in COVID-19 patients [15].

Among hospitalized patients, elevated troponin levels were observed in 20-30% of cases [16]. These levels correlated directly with the severity of the patient's condition [17] and were associated with the levels of inflammatory markers, such as CRP, D-dimers, ferritin, and fibrinogen [18].

Among athletes infected with COVID-19, the most concerning complication was myocarditis. This condition accounts for 3-10% of cases of sudden cardiac death in young competitors [19]. Athletes are a particularly at-risk group for developing myocarditis due to repeated physical exhaustion during training sessions [20]. Intense physical exercise can

impair immune responses; within 3 to 72 hours post-exercise, an individual is more susceptible to infection [21].

Myocarditis is a disease with symptoms that vary in severity, ranging from mild, self-limiting, nonspecific cardiac symptoms to the onset of fulminant heart failure [22]. The most common pathogens responsible for myocarditis are viruses, with pathogens such as adenoviruses, enteroviruses, parvovirus B19, and human herpesvirus 6 (HHV-6) detected during endomyocardial biopsies. Other causative agents include bacteria, fungi, and autoimmune diseases [23]. Typical symptoms of myocarditis include angina, dyspnea, palpitations, or syncope. Occasionally, only symptoms of a viral illness are present. Athletes, being more attuned to the complaints of their bodies, may report nonspecific feelings of fatigue, weakness, exercise intolerance, and muscle aches [24]. In severe cases, dyspnea, chest pain, malignant arrhythmias, heart block, and cardiogenic shock may occur [25]. Notably, according to recent studies [26], among young individuals (mean age 19.3 ± 6.2 years), only 47% reported any symptoms before death.

In the diagnosis of myocarditis, high sensitivity troponin should be used in conjunction with CRP [27]. However, care must be taken to avoid sample collection within 24-48 hours of training [28], as recent physical activity may accelerate troponin release and skew the results [29]. Caution is also needed when interpreting ECG findings, as many athletes exhibit ST-segment changes and T-wave abnormalities [30], which may mimic pathological changes but are actually physiological for the athlete's heart muscle. In contrast, ECG changes in athletes after COVID-19 infection were not common, even among patients who had been diagnosed with myocarditis through cardiac magnetic resonance imaging [31]. As seen, the diagnosis of myocarditis is complex and fraught with pitfalls. Imaging studies such as echocardiography can assist in diagnosis, revealing left ventricular wall thickening, increased myocardial echogenicity, localized myocardial hypokinesis, and potential dysfunctions of both the right and left ventricles [32]. Another helpful diagnostic tool is cardiac magnetic resonance imaging. It is recommended for athletes presenting with clinical symptoms of acute myocarditis, elevated troponin levels, and ECG abnormalities. The examination should be conducted within 2 to 3 weeks from the onset of symptoms or detection of abnormalities in other tests [33]. The gold standard for diagnosing myocarditis is endomyocardial biopsy; however, due to the invasive nature of the procedure and the associated high risks, it remains reserved for difficult cases [34].

Although early in the pandemic, imaging changes in the heart muscle were observed in half of young athletes [35] and one-quarter of elite competitors [36], subsequent studies [37] have reassessed these hypotheses and determined the incidence of myocarditis in athletes post-COVID-19 infection to be below <2%. However, a precise assessment of the potential risk will only be made following analysis based on athlete registry data [38].

There is considerable uncertainty regarding the recovery of athletes after COVID-19 infection. Athletes diagnosed with myocarditis should refrain from training for at least 3 to 6 months. This time frame has been shown to correspond to the resolution of inflammatory changes in cardiac magnetic resonance imaging and the disappearance of clinical symptoms, although the duration of rest should depend on the severity of symptoms and the assessment of cardiovascular risk [39]. After this period, a detailed evaluation of cardiac enzyme levels and ventricular systolic function should be conducted, followed by exercise testing. If all parameters are normal, athletes may gradually resume training [40]. During active COVID-19 infection, particularly when clinical symptoms are present, physical activity should be avoided [41]. Despite the availability of advanced laboratory tests and imaging methods, in some cases, it may be difficult to determine the risk of myocarditis. Certainly, certain groups of athletes, depending on the severity of their symptoms, should remain under cardiological supervision, and those at high risk of myocarditis should undergo thorough testing before being cleared for further training. We must remain vigilant and ensure thorough follow-up for athletes recovering from COVID-19.

IV. Respiratory complications

The SARS-CoV-2 virus is characterized by a particular affinity for the respiratory system. Numerous cases have been described in which COVID-19 infection left lasting effects on the respiratory capacity of affected individuals.

A common complication of SARS-CoV-2 infection is limited physical exertion capacity and hyperventilation during exercise [42], as well as decreased respiratory muscle conditioning [43], ventilatory inefficiency during exercise with reduced forced vital capacity (FVC) at rest [44], and dysfunctional breathing, defined by the presence of hyperventilation and variable breathing frequencies [45].

Research results regarding impaired respiratory function in athletes are conflicting and somewhat unclear. Several studies have shown no impact of COVID-19 infection on pulmonary function [46][47]. For example, a spirometry study on 13 professional football players found no deterioration in lung function after COVID-19 infection compared to pre-infection results [48]. Another study revealed a decline in mid-flow values of forced expiratory flow 75-25 (FEF75-25) rates in ¼ of athletes, but other spirometric parameters remained within normal limits [49]. On the other hand, a different study [50] demonstrated that over 30% of athletes exhibited abnormal FEV1% predicted and FEV1/FVC ratios between 3 to 6 months after COVID-19 infection, indicating airway obstruction. Other researchers [51] showed a 5.4% decrease in FVC, a 6.3% decrease in FEV1, and a 20.4% reduction in peak expiratory flow (PEF) compared to pre-infection values. In many cases, disturbances were only detected after more advanced testing. For instance, spiroergometry tests (CPET) revealed several abnormalities. Among a study of 13 elite cross-country skiers, it was shown that athletes post-COVID-19 had an average $\dot{V}O_2$peak 27% lower. Parameters such as minute ventilation, oxygen pulse, and breathing reserve also decreased [52]. Another group of researchers also published results clearly showing that parameters like $\dot{V}O_2$peak and minute ventilation decreased by 7% and 14%, respectively. These changes were accompanied by a 4% higher peak heart rate [53].

Furthermore, in a study involving a group of 18 athletes presenting symptoms of increased exertion during exercise post-COVID-19 infection, three of them exhibited abnormal oxygen uptake, while another three reached peak oxygen uptake between 80-90% of expected values. Some athletes also showed low breathing reserve at the end of physical exertion. A follow-up conducted after 5 months showed improvement in resting heart rate and an increase in peak oxygen consumption. Clinical symptoms reported by athletes during exertion, such as dyspnea, chest pain, and cough, also improved [54]. This evidence suggests that COVID-19 infection did indeed impact physical performance, but over a longer recovery period, respiratory function improved.

A limitation of many of the analyses is the small sample sizes. To draw appropriate conclusions about whether SARS-CoV-2 infection impacts respiratory function, large-scale studies are needed. Although the findings are conflicting, the theory that COVID-19 may impact physical performance and respiratory function in some individuals seems plausible. A method for managing the dysfunctions caused by the virus includes an appropriately long

recovery period, gradual reintroduction of training to regain baseline pulmonary function, and respiratory training.

V. Long COVID

During studies on the course and progression of COVID-19, persistent symptoms of infection that lasted for several weeks were often observed. Based on these observations, two disorders were identified: post-acute COVID syndrome (PACS), in which manifestations perserve for 3 weeks after the onset of the disease, and chronic COVID, also known as long COVID, in which symptoms last or new reactions appear 12 weeks after the onset of the disease. For a diagnosis of long COVID to be made, symptoms must persist for at least 2 months [55].

Symptoms of long COVID can present with a wide spectrum of manifestations and can involve clinical features from all body systems. The most commonly reported symptoms include cough, chest pain, dyspnea, abdominal pain, nausea, chronic fatigue, muscle pain, sleep disturbances, and cognitive dysfunctions [56]. More nonspecific symptoms, such as skin rashes or ocular symptoms like dry eyes and increased light sensitivity, can also occur [57]. It is also important to note that 20-50% of patients may develop psychiatric disorders, including depressive and anxiety disorders [58].

According to research [59], approximately 10-35% of outpatients experienced persistent symptoms of long COVID. In hospitalized patients, this percentage increases to as high as 85%. This represents a significant number of individuals for whom the battle with the disease does not end after leaving the hospital.

When it comes to the occurrence of long COVID symptoms in athletes, studies provide contradictory data. One analysis [60] found that only 1.2% of athletes reported experiencing any manifestations. The most common symptoms were loss of smell and taste (63%), dyspnea (20%), and cough (15%). Interestingly, 3.8% of athletes reported signs during physical activity after returning to exercise, with dyspnea (58%), chest pain (36%), and exercise intolerance (23%) being the most frequently observed symptoms. Another study [61], involving Olympic and Paralympic athletes, showed that 14% of participants experienced persistent manifestations, and as many as 27% were unable to return to training at the intensity they had before the illness. Athletes most commonly reported chronic fatigue,

dry cough, and headache (71%, 57%, and 52%, respectively). It was also found that competitors who reported chills, chest pain, breathing difficulties, and loss of taste had a lower chance of returning to their normal physical activity within 40 days after infection compared to those who did not exhibit these symptoms [62]. SARS-CoV-2 infection may also predispose individuals to co-infections with other viruses, such as Epstein-Barr virus (EBV). Some of the long-term post-COVID manifestations and complications could be related to co-infection with this virus [63].

While most athletes experience SARS-CoV-2 infection with mild signs, persistent symptoms resulting from long COVID can be more severe and troublesome for them. Most disorders associated with long COVID are managed symptomatically. To improve physical fitness and reduce symptoms related to the respiratory and cardiovascular systems, gradual reintroduction of physical exercise is recommended, with cessation if symptoms like dyspnea arise [64]. For conditions such as chronic fatigue and depression or anxiety disorders, cognitive-behavioral therapy (CBT) has shown effectiveness [65]. Medications such as selective serotonin reuptake inhibitors (SSRIs), serotonin and norepinephrine reuptake inhibitors (SNRIs), and melatonin may also be helpful. Proper nutrition, sleep hygiene, and stress management are essential, as neglecting these aspects can exacerbate the inflammatory state [66]. There is data [67] suggesting that vaccination in individuals who were previously unvaccinated against COVID-19 reduces symptom severity. While the mechanisms behind this improvement are not well understood, it is hypothesized that the vaccine helps eliminate the remaining virus in the body, reduces the immune response, and resets the immune system. Improvement has been observed in about 30-40% of patients who received the vaccine.

VI-. Summary

COVID-19, specialists remain vigilant. During the pandemic, the sports industry was one of the first to return to normalcy. Thus, oversight and the creation of recommendations for a safe return to sports were of paramount importance. However, comprehensive and detailed analyses on the impact of COVID-19 on various bodily functions are still lacking. The studies conducted so far indicate that athletes are, to varying degrees, at risk of complications arising from the infection. The basis for the actions of medical professionals caring for athletes is a thorough evaluation of symptoms and heightened awareness of complaints

reported by athletes during the period of returning to normal physical activity. Return to play (RTP) should be gradually implemented, with any concerning symptoms reported by athletes closely monitored. In cases of severe symptoms or a diagnosis of myocarditis, the most important approach is extended rest from training—typically 3-6 months. In other complications, such as chronic fatigue, sleep disturbances, and depressive or anxiety disorders, pharmacological interventions, such as selective serotonin and norepinephrine reuptake inhibitors (SSRIs/SNRIs) and melatonin, may be beneficial. Cognitive-behavioral therapy (CBT) should also be considered. Additionally, vaccination against COVID-19 may have a positive effect on persistent and troublesome symptoms in patients who were previously unvaccinated.

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