On March 2020, WHO has declared the coronavirus disease (COVID-19), caused by the virus severe acute respiratory syndrome coronavirus 2 outbreak, a pandemic. Young people can be affected by the disease, and some of them require hospital admission and intensive care.

The clinical manifestations of COVID-19 include cardiac involvement and complications, among which are myocarditis (including fulminating cases), arrhythmias and rapid-onset heart failure. Since the first cases reported in Wuhan, China, increased levels of serum myocardial biomarkers were found in the sickest patients and associated with worst outcomes. In a series of 41 cases, 12% patients had elevated levels of troponin, indicating myocardial injury. Another study showed acute arrhythmias in 17% and acute myocardial injury in 7% of patients with COVID-19. In a meta-analysis including six studies with a total of 1527 patients with COVID-19, 8.0% suffered acute cardiac injury with an incidence about 13-fold higher in critically ill patients admitted in intensive care units. Increased levels of natriuretic peptides has also been reported in these patients and associated with worst prognosis.

In some cases, cardiac involvement occurred even in patients without symptoms and signs of interstitial pneumonia, reinforcing the importance of subclinical cardiological investigation and measurement of cardiac biomarkers. Moreover, postmortem analysis suggests myocardial infiltration with mononuclear inflammatory cells. The mechanisms responsible for acute myocardial injury in patients with COVID-19 are not fully understood, but inflammatory reaction with cytokine storm, immunological factors, ACE2-related signalling pathways, hypoxia and direct myocardial damage by viral invasion may be involved.

Despite the many aspects and doubts that remain to clarify regarding COVID-19 disease implications in the various medical and scientific societies, there is high pressure to resume sports competitions, mainly in football, due to economic and competitive factors. However, from the clinical point of view, it seems to us premature to restart exercise before a clear control of this infection. A better clarification of the disease evolution and the potential long-term cardiorespiratory complications is also needed. Additionally, it is important to underline that, as described in other topics of sports cardiology, exercise training may contribute to the development of some cardiovascular conditions, even in apparently healthy individuals.

We opine that in athletes clinically recovered from a proved infection (even those with mild disease, without cardiac symptoms or hospital admission), a subclinical myocardial injury may be present. We recommend a medical evaluation before the athlete resume trainings, eventually with exams as transthoracic echocardiogram, maximal exercise testing and 24-hour Holter monitoring to exclude subclinical disease.

However, due to the risk of transmission, these exams should be performed with caution and following all the recommended protective measures, specifically regarding exercise testing, in which it may be difficult to find facilities willing to perform these tests. In order to identify a very low-risk group, who can be cleared without the need for supervised exercise testing, the evidence of recovery and risk stratification should be based in another information, such as: clear exclusion of symptoms suggestive of myocarditis or myopericarditis; deep knowledge of the infection “status” (repetition of viral tests when appropriate or immunological tests if available); exclusion of ongoing myocardial injury (normal troponin measurements); exclusion of significant arrhythmias (normal 24/48-hour Holter monitoring). If the absence of positive findings the athletes can resume exercise-training.

In the presence of any positive and in those with serious COVID-19 infection, the management should be similar to other cases of myocarditis, with a further work-up (eg cardiac magnetic resonance, implantable looping recorders, among others). If the diagnosis of myocarditis or myopericarditis is established, a period of disqualification (3–6 months) is needed, according to the clinical severity and duration of the illness. After this period, it is reasonable to resume training and competition if left ventricular systolic function has returned to the normal range, serum biomarkers of myocardial injury have normalised and clinically significant arrhythmias such as frequent or complex repetitive forms of ventricular or supraventricular arrhythmias are absent on 24-hour Holter monitoring and exercise test. We highlight that myocarditis is a cause of malignant arrhythmias, left ventricular dysfunction and sudden cardiac death in athletes.

In this context of uncertainty, it is essential to assure equity and safe conditions for the protection of all stakeholders to resume sports activities—a single athlete can be a vector of transmission. For this purpose, physicians should be involved in the decision and contribute to establish specific protocols to evaluate athletes affected by COVID-19. Cardiac evaluation is of utmost importance due to the direct complications of the disease and the potential adverse effects of some medications used for the treatment of COVID-19 (eg, steroids; antibiotics, and antimalaric, antiviral, anti-inflammatory or immunosuppressant drugs), with implications for exercise training.
REFERENCES


