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Commentary

Myocarditis in athletes after COVID-19 infection: The heart is not the only place to screen

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ABSTRACT

COVID-19 patients are susceptible to hypercoagulability. For the safe return to sports after COVID-19, athletes or individuals wanting to resume physical activity should complete screening for myocardial injury and myocarditis. In addition, patients with COVID-19 are reported at prevalence of 27%–31% for venous thromboembolic events. The probability of deep vein thrombosis and pulmonary embolism prior to intensive exercise after COVID-19 infection should be considered. The prevalence of cardiac injury is reported at 19%, and the prevalence of deep vein thrombosis and pulmonary embolism is higher than that for myocarditis. Thus, the heart is not the only system needing screened. Examination for myocardial injury and myocarditis are mandatory. Also, deep vein thrombosis, and pulmonary thromboembolism must be considered, and when possible, blood troponin values, D-dimer prothrombin time, and activated partial thromboplastin time levels are determined for COVID-19 infection athletes or any individual before returning to sporting practice or intense physical activity or exercise.

The world is suffering from the coronavirus disease (COVID-19) pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The overall virus's course and outcome still remain uncertain. The first human cases of COVID-19, the disease caused by the novel coronavirus causing COVID-19, subsequently named SARS-CoV-2, were first reported by officials in Wuhan City, China, in December 2019.[1]

The pathophysiology of COVID-19 is complex, but some unrecognized complications of the illness include coagulation disorders and cardiovascular system complications follow this illness. COVID-19 has the potential to negatively impact the safe resumption of competitive sports, increasing risk for sudden cardiac death. Coagulation cascades with thrombin generation are activated by proinflammatory cytokines is a process known as immune-thrombosis or thromboinflammation and is known as a complication in COVID-19 infection patients.[2]

The cell entry of SARS-CoV-2 depends on the angiotensin-converting-enzyme 2 (ACE2) receptor, which is widely expressed in the heart, endothelial cells and is linked to inflammatory activation. Autopsy data from three COVID-19 patients showed infection of the endothelial cells in the heart and diffuse endothelial inflammation, but no sign of lymphocytic myocarditis.[3] Because the heart abundantly expresses ACE2, the heart is vulnerable to SARS-CoV-2 infection. Autopsies of patients with severe acute respiratory syndrome (SARS) have revealed that 35% (7/20) patients were positive for the SARS-coronavirus (SARS-CoV) genome in the cardiac tissue, and patients with SARS-CoV cardiac infections had a more aggressive illness and earlier mortality than those without it.[4]

Athletes have the same risk as the general population for COVID-19 with a lower risk of complications or severe illness according to COVID-19 infection reports. Middle-aged athletes have the same risk of infection as the general population, with a lower risk of complications or severe illness associated with COVID-19 infection. However, athletes, as any other individual, are not free of subsequent complications from COVID-19 sequelae, may not be able to avoid subsequent complications from the disease which can trigger an exaggerated inflammatory response that could cause myocardial injury.[5]

Evaluation of COVID-19 positive athletes with mild symptoms who are not hospitalized and individuals with significant symptoms who are hospitalized, the focus is on myocardial injury or myocarditis in COVID-19 infection patients. Myocardial injury, myocarditis, and the risk of sudden cardiac death are the focus for cardiovascular prevention. Proposed screening for discharge cardiac injury with physical examination should include electrocardiogram, echocardiography, stress testing, cardiac magnetic resonance, and rhythm monitoring. The evaluation of troponin levels is important at discharge for myocardial injury. Return to play after myocarditis is predicated on normalization of ventricular function, the absence of inflammation biomarker evidence, and the absence of inducible arrhythmias.[6]

Different proposals regarding the resumption of sports and exercise after COVID-19, such as a position paper by the Dutch Sports Cardiology Section of the Netherlands Society of Cardiology, have recently been published.[7] Based on different categories of disease severity, this point-of-view article provides the sports cardiologists or sports physicians a practical guide regarding pre-participation screening, and clinical management strategies for competitive athletes and highly active individuals >16 years of age after COVID-19 infection. Moreover, a complete analysis of arrhythmias, myocardial, and coronary complications are described with an excellent flowchart to assist clinicians in the stratification of patients. In addition to suggesting pre-participation screening, and diagnostic and therapeutic strategies, this report also provides general sports advice. However, this report does not consider the possibility of venous or pulmonary thromboembolic complications due to COVID-19 which must be evaluated prior to the return of athletes to sporting activities.[7]

The highest mortality rates are observed in patients with underlying cardiovascular disease and elevated cardiac troponin levels. Observed cardiac injuries are 19% of patients hospitalized with COVID-19 and is associated with a higher risk of in-hospital mortality.[2,5]

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Is the heart the only place to screen prior to safe returning to sports or physical activity? This question is most important. When an evaluation is correctly completed, the answer is no. Consideration only given to the heart is not enough. A comprehensive cardiovascular evaluation determining cardiac complications in athletes or any individual after COVID-19 infection is recommended.

During COVID-19 infection, two separate pathologic coagulation processes are responsible for producing clinical manifestations. In the microcirculation of the lungs and potentially other organs such as the myocardium, direct local vascular and endothelial injury occurs leading to microvascular clot formation and angiopathy, which also occurs in the systemic circulation due to hypercoagulability and hyperfibrinogenemia. Also, the possibility of large vessel thrombosis, deep vein thrombosis (DVT), and major thromboembolic events exist. Pulmonary sequelae have been reported in 20%–30% of intensive care unit patients.[8]

Increased D-dimer level, a fibrin degradation product, is used in the initial screening for determining the stage of hypercoagulopathy in COVID-19 infection patients. In most patients suffering mild COVID-19 disease, hypercoagulopathy, a common condition associated with DVT, a high risk of hypercoagulopathy exist, and is a likely COVID-19 complication.[8,9] Thromboembolic complications that lead to pulmonary embolism are reported in the composite incidence of thrombotic events, which is 31%. Venous thromboembolic events are the most common (27%), and the majority of these events are pulmonary embolisms. Predictors of thrombotic events are increased age, evidence of coagulopathy on screening blood tests, higher D-dimer levels, prothrombin time above the upper limit of normal, and an activated partial thromboplastin time above the upper limit of normal.[2,9]

A large Chinese study that included 1099 COVID-19 positive patients from 552 hospitals revealed that D-dimer concentrations above the threshold of 0.5 mg/L in 46.4% of the patients; 60% of these patients developed severe COVID-19 manifestations. In these patients, D-dimer levels (2.12 mcg/mL, 0.77–5.27) were four times higher than non-severely COVID-19 infected patients (0.61 mcg/mL, 0.35–1.29). The use of a D-dimer cut-off value $>$ 1.5 mcg/mL to predict DVT demonstrated a sensitivity of 85% and specificity of 88.5%.[10]

The heart is not the only place to screen. At discharge, determining myocardial injury and myocarditis are mandatory. Also, DVT and pulmonary thromboembolism should be considered at discharge. If possible, include obtaining troponin values, D-dimer prothrombin time, and activated partial thromboplastin time levels are recommended for COVID-19 infection athletes or any individual before returning to sporting practice or intense exercise, or physical activity.

Submission statement

The manuscript has not been published and is not under consideration for publication elsewhere.

Authors' contributions

All authors contributed equally to the publication.

Conflict of interest

The authors have no conflict of interest to report.

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