Perspective

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Post COVID-19 Cardiovascular Syndrome: An Entity That Cannot Go Unnoticed in the Post-COVID Era

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Introduction

Cardiovascular manifestations and complications of COVID-19 are one of the most important topics in the management of this group of patients, due to the risk of morbidity, mortality and loss of functional capacity [1]. Much of the research on COVID-19 is focused on the acute phase. However, post-COVID 19 syndrome is gaining more and more importance due to the costs and challenges in its management. Post-COVID 19 syndrome is defined as the permanence or genesis of signs and symptoms depending on the organ affected during the acute phase of COVID-19, which may last indefinitely [2]. As this syndrome is characterized according to the organ or organs affected, a large number of phenotypes can occur, the most famous of which is the post-COVID 19 neurological syndrome [3].

Nevertheless, evaluated cohorts report that shortness of breath and fatigue, in both hospitalized and home care patients, and in both those with and without comorbidities, are the most prevalent symptoms over a time span of up to 6 months [4]. However, these sequelae are more intense and last longer in those with a personal history of pulmonary or cardiac disease [4]. Considering that myocardial injury has been reported in the COVID-19 patient, which may be mild and undetectable in the vast majority of cases (since there are asymptomatic patients who may debut with a cardiovascular events), these symptoms may originate from cardiac decompensation.

Conducted a study where they evaluated the level of coronary microvascular injury in patients who had COVID-19 *vs*. control groups through cardiovascular magnetic resonance imaging, showing that patients who had COVID-19 had significantly reduced global myocardial perfusion reserve (2.73[2.10-4.15-11] *vs.* 4.82 [3.70-6.68], p=0.005), significantly increased coronary sinus flow at rest (1.78 ml/min [1.19-2.23 ml/min] *vs*. 1.14 ml/min [0.91-1.32 ml/min], p=0.048), and reduced coronary sinus flow during stress activity (3.33 ml/min [2.76-4.20 ml/min] *vs.* 5.32 ml/min [3.66-5.52 ml/min], p=0.05), compared to controls [5]. These results allowed the authors to conclude that myocardial vascular injury is evident during the post-COVID phase, and probably this pathophysiological microvascular mechanism is responsible for dyspnea and fatigue during the post-COVID-19 syndrome [5].

The use of pharmacological products aimed at improving endothelial function, systemic microcirculation and management of fatigue and dyspnea symptoms during the post-COVID-19 syndrome has also been evaluated, observing a positive response compared to control groups ($p<0.05$) [6]. Given this volume of evidence, we propose for the first time the specific use of the post-COVID-19 cardiovascular syndrome, to target those patients who persist with equivalent cardiovascular symptoms after the acute phase of COVID-19, to evaluate more precisely the coronary endothelial and cardiac microvascular compromise, to prevent and manage cardiovascular complications, reduce costs, control morbidity, mortality and try to recover and maintain the functional capacity of these patients; mainly those with cardiometabolic comorbidities such as hypertension, type II diabetes mellitus, obesity, or cardiac antecedents such as heart failure, who are at higher risk of decompensation and/or major cardiovascular events. Likewise, to propose strategies aimed at cardiovascular rehabilitation with access to specialized health services that provide strict long-term follow-up [7].

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