LETTER

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Post-COVID 19 syndrome: Inquiring about reported phenotypes

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ABSTRACT

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The COVID-19 pandemic has become possibly the largest public health problem of the 21st century. As of August 20, 2021, the Center for COVID-19 Tracking at Johns Hopkins University reports more than 200 million cases and more than 4 million deaths.[1] However, another of the great unknowns are the sequelae left by the acute phase of COVID-19 and the impact they have on health systems through parameters of health costs, morbidity, mortality and disability.[2] The period of time when the sequelae are observed has been termed post-COVID-19 syndrome. Is defined as the persistence or appearance of signs and/or symptoms after the acute phase of the disease, specifically in two time periods, between 4 and 12 weeks and after week 12, and potentially compromising the functional capacity of the individual.[2] This syndrome can occur in young patients as well as in patients with advanced disease, and in those with or without a history of comorbidities.[2] One of the greatest challenges today is the implementation of strategies for early diagnosis and timely management of major complications presented in this period of time, especially in low- and middle-income countries where resources for health systems are scarce and where there is a very high overall disease burden.

Although the evidence does not specify many phenotypes of this syndrome, the results of observational studies suggest the presence of a large number of phenotypes. The first is post-COVID 19 tachycardic syndrome, described by Ståhlberg et al[2], in which palpitations are present in approximately 50% of patients.[2] The causality of this phenotype is unknown, however, it has been observed that these patients also present orthostatic hypotension and findings of myocardial injury through cardiac imaging studies.[2]Urmeneta Ulloa et al[3] carried out a study in which they evaluated 57 patients with post-COVID syndrome vs. control group through cardiac magnetic resonance imaging, evidencing that T2 mapping values (suggestive of oedema) were higher in the study patients than in the controls (50.9 ± 4.3 msvs 48 ± 1.9 ms, $p < 0.01$). But, no between-group differences were observed for native T1 nor for circumferential strain or radial strain values (18.6 ± 3.3% vs 19.2 ± 2.1% ($p = 0.52$) and 32.3 ± 8.1% vs 33.6 ± 7.1% ($p = 0.9$), respectively).[3] A study carried out by Drakos et al[4], who evaluated coronary microvascular disease in COVID-19 patients by 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.[4] Based on the above, the authors concluded that there is cardiac microvascular injury in COVID-19 patients, which may trigger major cardiovascular events in the post-COVID-19 phase.[4] This allows us to affirm that there is indeed a silent lesion in the cardiovascular system, specifically at the myocardial level, which may facilitate the presentation of this phenotype.

Pasiniet al[5] studied the serum blood profile of 75 patients with post-COVID syndrome, finding that all patients had very

high serum concentrations of ferritin and D-Dimer. 87 and 72% of patients had clinically significant low levels of hemoglobin and albumin, respectively. Seventy three percentage had elevations in erythrocyte sedimentation rate and CRP.[5] Twenty seven percentage had elevations in LDH, allowing the authors to conclude that these findings explain a time window of inflammatory and thromboembolic disease risk.[5] Based on the above, it is perceived that there is a phenotype associated with a post-COVID 19 metabolic disorder, which can affect any endocrine-dominant organ.

Post-COVID 19 neurological syndrome[6,7], which can occur even in patients who did not present neurological manifestations, is one of the phenotypes where a slow but persistent increase in new evidence has been observed, which attempts to answer many questions about neuroinflammation and central nervous system involvement during the acute phase of COVID-19. During the process of this phenotype, cerebrovascular disorders, neuroimmune or neurometabolic disorders, derived from the neuroinflammation of the pathophysiology of COVID-19, may occur.[6,7] This could be the highest risk phenotype due to neurological compromise, risk of decompensation and death.[6,7]

In this order of ideas, it can be observed that the follow-up of patients who develop any type of COVID-19 phenotype (mild, moderate or severe) should be multidisciplinary, and the ideal would be the design of centers or departments specialized in post-COVID syndrome,[8] to prioritize those patients with the highest number of risk factors and reduce the risk of developing any other type of complication that may increase the risk of death and loss of functional capacity.

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