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LETTER TO THE EDITOR

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The "post-COVID" syndrome: How deep is the damage?

To the Editor,

We congratulate Halpin et al.[1] on their work to identify residual symptoms in patients with microbiological recovery from COVID-19. The prevalence of residual symptoms in their cohort is much higher than estimates of 35% among out-patient,[2] but comparable with recent cohorts of hospitalized patients (87%).[3] Apart from fatigue, a significant proportion of their patients suffer from persistent dyspnea and neuropsychological symptoms. The reasons for this may be manifold. The study population is older (median age 70.5 years in the ward group) and sicker than the ones previously reported. The majority of patients (67.7% among ward patients and all intensive care unit [ICU] patients) required oxygen supplementation, 32% were admitted to ICUs, and one patient underwent invasive mechanical ventilation. Thus, patients of severe and critical COVID were overrepresented in their cohort when compared with other studies where mild-moderate, severe, and critical COVID comprised 81%, 14%, and 5% of patients, respectively.[4] Previous experience has shown that critically ill patients face prolonged functional impairment after discharge, which may last several years.[5] Older age is a known risk factor for impairment.

The study population includes a large proportion of patients with significant comorbidities, like chronic respiratory diseases (chronic obstructive pulmonary disease and asthma), malignancy, and cardiovascular disease. These diseases carry significant morbidity themselves and may have contributed to the aforementioned symptoms. Thus, analysis with adjustments for baseline health before the illness would provide more meaningful data about the "post-COVID" syndrome.

An important distinction should be made between symptoms due to persistent chronic inflammation (convalescent phase), sequelae of organ damage (acute lung and kidney injury resulting in pulmonary fibrosis and chronic kidney disease, respectively), and nonspecific effects from the hospitalization and social isolation (nutritional anemia, muscle wasting). A subgroup analysis including only mild COVID patients would provide more insight into the postviral syndrome, as this group is unlikely to have chronic organ impairment. Evaluation for the cause of fatigue in this subgroup using simple blood investigations may reveal treatable etiologies, including anemia, vitamin D deficiency, hypothyroidism, cortisol insufficiency, and chronic kidney disease. For example, subclinical thyroid dysfunction is seen in more than half of hospitalized COVID-19 patients as per some reports, although the data of persistence of these lab abnormalities post-discharge is not available.[6]

Pulmonary recovery in COVID-19 lags behind virological clearance.[7] Furthermore, there exists anecdotal evidence of post-COVID fibrosis, which may cause significant dyspnea and cough. Findings on chest imaging and pulmonary function tests in patients with the authors' study population would help delineate the basis of fatigue and dyspnea.

Finally, information on the treatment offered at the post-COVID clinics and the subsequent response would be invaluable to healthcare workers worldwide, who, after facing the first wave of COVID, are now suddenly battling this new "post-COVID" syndrome.

CONFLICT OF INTEREST

The authors declare that there are no conflict of interests.

KEYWORDS

coronavirus, epidemiology, immune responses, inflammation, pandemics, virus classification

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