CARDIOVASCULAR SYSTEM STATUS IN PATIENTS WITH DYSPNEA AFTER COVID-19-ASSOCIATED PNEUMONIA

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Abstract

The aim of this study is to determine the contribution of cardiovascular system impairment to the development of dyspnea in post-acute COVID-19 pneumonia patients.

Materials and methods. A total of 108 patients with dyspnea in the post-acute phase of COVID-19 (at 47 (38; 62) days after the onset of symptoms) were examined. These patients had experienced COVID-19-associated pneumonia between April and November 2021. 21 patients were excluded due to comorbidities that could be a potential cause of dyspnea in the post-COVID-19 period. Thus, a group of 87 patients was formed, with an average age of 56 (49; 65) years, including 42 (48.3%) males. Patients were divided into three subgroups based on the severity of their acute COVID-19 illness: subgroup 1 (35 patients) — moderate, subgroup 2 (37 patients) — severe and subgroup 3 (15 patients) with critical. Investigation methods included clinical examinations, the mMRC dyspnea questionnaire, chest CT scans (both anamnestic and during examination), spirometry, ECG, echo-Doppler, serum NT-proBNP levels.

Results. Patients in subgroups 1 and 2 in the post-acute phase of COVID-19 had less pronounced dyspnea compared to patients in subgroup 3 (1 (1; 2) and 2 (1; 3) vs. 3 (2; 4), respectively) ($p < 0.001$). Residual changes on chest CT scans were more frequently observed in subgroup 3 than in subgroups 1 and 2 (15 (100%) vs. 7 (20.0%) and 20 (54.1%), respectively) ($p < 0.001$). Obstructive ventilation disorders were only observed in subgroups 1 and 2 (in 15 (42.9%) and 6 (16.2%) patients, respectively), while restrictive disorders were found in subgroups 2 and 3 (in 14 (37.8%) and 12 (80.0%) patients, respectively). Clinically significant rhythm or conduction disturbances were not detected on ECG in any patient. According to echo-Doppler, subgroup 1 had diastolic dysfunction of the left ventricle (LV) in 3 (8.5%) patients; subgroup 2 had diastolic dysfunction of both the LV and right ventricle (RV) in 5 (13.5%) and 4 (10.8%) patients, respectively; and subgroup 3 had diastolic dysfunction of both the LV and RV in 4 (26.7%) patients. Mild pulmonary hypertension was observed only in subgroups 2 and 3 (in 13 (35.1%) and 14 (93.3%) patients, respectively). Elevated NT-proBNP levels were found in 22.9% of patients, with no significant differences between subgroups ($p > 0.05$).

Conclusions. The severity of dyspnea in the post-acute phase of COVID-19 is related to the duration of lung involvement and the severity of the acute illness. In patients with moderate-severity acute COVID-19, dyspnea in the post-acute phase is primarily due to residual respiratory system changes (in 20.0% of patients) and mild obstructive ventilation disorders (in 42.9% of patients). Cardiovascular system involvement in these patients is mainly detected by elevated serum NT-proBNP levels (in 22.9% of patients), which may serve as a marker for the heart failure onset, necessitating consultation with a cardiologist. In patients with severe- acute COVID-19, dyspnea in the post-acute phase is due to residual morphological lesions in the respiratory system based on chest CT findings (in 54.1% of patients) and cardiovascular pathology (in 45.9% of patients). Therefore, managing this patient cohort in the post-acute phase requires collaboration between pulmonologists and cardiologists, especially when dyspnea does not correlate with morphological lesions in the respiratory system on chest CT. In patients with critical acute COVID-19, dyspnea in the post-acute phase is consistently caused by both residual morphological lesions in the respiratory system (in 100% of patients) and cardiovascular pathology (in 93.3% of patients). This substantiates the imperative for joint management by pulmonologists and cardiologists.

Key words: coronavirus disease, COVID-19, pneumonia, dyspnea, post-acute period, cardiovascular system, systolic dysfunction, diastolic dysfunction, heart failure, pulmonary hypertension, NT-proBNP.

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