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Coronavirus Pandemic

Age and chest computed tomography severity score are predictors of long-COVID

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Abstract

Introduction: About one-third of acute coronavirus disease 2019 (COVID-19) survivors have suffered from persisting symptoms called long-COVID. Clinical factors such as age and intensity (moderate or acute) of COVID-19 have been found to be associated with long-COVID. Many tissues might be damaged functionally or structurally during acute COVID-19 which can be detected by blood assays and chest computed tomography (CT). We aimed to evaluate the relationship between long-COVID and the initial findings of blood assays and chest CT as possible predictors.

Methodology: The study included patients with acute COVID-19. Laboratory tests and chest CT were obtained from each patient at the time of admission to the hospital. Chest CT was evaluated for pneumonic involvement and severity score. Multivariable regression model was created to find the factors that were independently associated with long-COVID.

Results: There were 60 (38.2%) patients with long-COVID and 97 (61.8%) without. Baseline demographic, laboratory and chest CT parameters were similar in both groups, except for age, chronic lung disease and chest CT severity score (46.9 ± 15.1 years vs 52.6 ± 15.9 years, $p = 0.03$; 11.7% vs 3.1%, $p = 0.03$ and 10.3 ± 9.6 vs 6.5 ± 7.6, $p = 0.02$, respectively). In multivariable model, chest CT severity score (OR: 1.059, 95% CI: 1.002-1.119, $p = 0.04$) and age (OR: 0.953, 95% CI: 0.928-0.979, $p < 0.001$) were independently associated with long-COVID.

Conclusions: Chest CT severity score and age were independently associated with long-COVID and may be used to predict the future risk of long-COVID.

Key words: long-COVID; post-COVID; chest CT; pneumonia chest CT severity score.

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Introduction

The coronavirus disease 2019 (COVID-19) pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has led to devastating effects on humanity, with millions of deaths and morbidity. Fortunately, its mortality rate has decreased after the development of vaccines. However, more than one-third of COVID-19 survivors have experienced longer-term symptoms such as dyspnea, cough, fatigue, sleep disturbances, anxiety, or depression [1,2]. The presence of these symptoms without complete recovery after four weeks of acute COVID-19 is now called 'long-COVID' and has emerged as a critical health issue because of its adverse effects on the quality of life and personal productivity [3].

Reducing long-COVID induced morbidity has been an essential target in clinical practice and detecting factors associated with the development of long-COVID symptoms is the first step in this way. Therefore, some clinical parameters have recently been evaluated in several studies and found to be associated with long-COVID [2,4-6]. However, it is clear that many functional and structural impairments in tissues and organs have also happened during the acute phase of COVID-19. In the clinical management of these patients, some blood tests and imaging techniques such as blood oxygen saturation (SpO_2), D-dimer, and chest computed tomography (CT) have been used widely to assess tissue or organ damage and clinical status. Therefore, we proposed that these parameters might be associated with the development of long-COVID. Thus, in this study, we aimed to evaluate the relationship between long-COVID and initial findings of diagnostic work-up, including biochemical tests and chest CT, besides clinical parameters as possible predictors.

Methodology

Study population

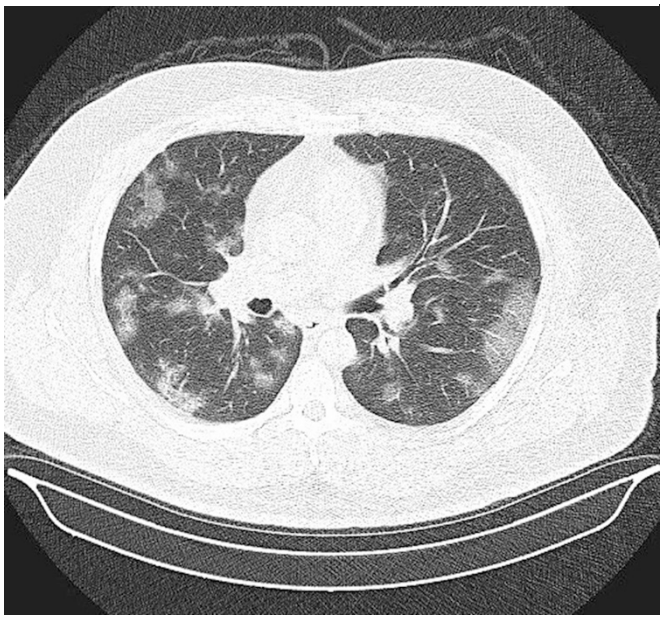
The study population consisted of consecutive adult patients (> 18 years of age) with COVID-19 diagnosed by real-time reverse transcription polymerase chain

reaction (RT-PCR) test in the prevaccination era of the pandemic. Only patients who had chest CT and laboratory assays obtained during the initial diagnostic work-up were included in the study. Hypertension (HT), diabetes mellitus (DM), cardiovascular disease (CVD, including coronary heart disease, heart failure or ischemic stroke) and chronic lung disease (chronic obstructive pulmonary disease (COPD) and active asthma) were assessed by the presence of previous medical history and/or drug use. Exclusion criteria were the absence of chest CT imaging or laboratory assays, being a critical patient (individuals who had respiratory failure, septic shock and/or multiple organ dysfunction), no follow-up data, and mortality. The study was conducted in accordance with the guidelines in the Declaration of Helsinki. The University of Health Sciences Turkey, Bursa High Specialization Training and Research Hospital Clinical Research Ethics Committee approved the study (protocol number 2011-KAEK-25 2023/02-14).

Assessment of the patients with acute COVID-19

After the diagnosis of COVID-19, all patients underwent a standard work-up including blood assays and SpO_2 along with detailed clinical assessment. In addition, according to our national management algorithm used during the first period of the COVID-19 pandemic where there were no vaccines, the patients with suspected lung involvement also underwent a chest CT imaging to assess possible involvement and determine its severity if present.

Figure 1. 39-year-old male patient with a positive reverse transcriptase polymerase chain reaction (RT-PCR) test for COVID-19. Chest computed tomography (CT) shows focal ground glass opacities in both lungs. Chest CT severity score was calculated as 18.



Severity of COVID-19 was evaluated by using COVID-19 treatment guidelines published by National Institutes of Health [7]. According to this guideline, the patients were cathegorized into mild, moderate, severe and critical illness.

1. Mild cases: mild or minimal clinical symptoms, no sign of pneumonia on chest imaging

2. Moderate cases: fever and respiratory symptoms, pneumonia on chest imaging and $SpO_2 \geq 94\%$ in room air.

3. Severe cases: severe respiratory distress and/or increased respiratory rate > 30 breaths/min and/or decreased SpO_2 in room air with < 94% and/or arterial partial pressure of oxygen (PaO_2)/fraction of inspired oxygen (FiO_2) < 300 mmHg and/or lung infiltrates > 50%

4. Critical cases: respiratory failure requiring mechanical ventilation and/or septic shock and/or other organ failure requiring intensive care unit (ICU) admission.

Patients with impaired clinical status and/or work-up results were hospitalized and admitted to the ICU by the attending physician. All patients with critical illness were excluded from the study because high mortality and morbidity were expected.

Assessment of chest computed tomography imaging

Chest CT imaging was performed by a 64 slice multi-detector CT scanner (Somatom Sensation, Siemens, Erlangen, Germany) during inital examination of the patients with acute COVID-19. All chest CT images were evaluated for the presence of pneumonic involvement, number of involved lung lobes and severity score. Chest CT severity score was calculated by using a predefined method [8]. According to this method, the two lungs were divided into 20 regions and were evaluated for ground glass opacities. Each region was scored as 0 point for no parenchymal involvement, 1 point for ≤ 50% opacification and 2 points for > 50% opacification. Finally, the chest CT severity score was obtained by adding the points from each region (Figure 1).

Assessment of long-COVID and follow-up

Long-COVID was diagnosed by using the COVID-19 rapid guideline published by the National Institute for Health and Care Excellence (NICE) [3]. According to this guideline, the presence of signs and/or symptoms related to COVID-19 that continued or developed 4 weeks after acute infection were accepted as long-

COVID. Fatigue, dyspnea, arthralgia or myalgia, sleep disturbance, mental confusion, cough, palpitation, mood change, change in smell and taste, chest pain, pins and needles, headache, dizziness and irregular menstrual period were accepted as the syptoms of long-COVID. The study population was retrospectively followed up for two years. During this time, follow-up data were obtained from either hospital visits or telephone interviews. After the follow-up period, the patients without follow-up data were excluded from the study.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation and categorical variables were expressed as numbers and percentages. The normal distribution of continuous variables was evaluated by using the Kolmogorov-Smirnov test and histogram. Continuous variables were analyzed by independent-sample t-test or Mann-Whitney U test according to normal distribution. Chi-square and Fisher's exact tests were used for categorical variables. The relationship between long-COVID and other factors was initially evaluated by univariate analysis. Subsequently, a multivariable logistic regression model was created to detect independently related factors to long-COVID. A p value of less than 0.05 was considered statistically significant. All statistical analyses were carried out by the SPSS 21 statistical software (SPSS Inc., Chicago, Illinois, USA).

Results

One hundred and fifty-seven patients were included in this study. 60 (38.2%) patients had long-COVID symptoms (group 1) and 97 patients had complete recovery without any additional symptoms (group 2). Baseline demographic and clinical parameters of both the groups are presented in Table 1. Age was lower and chronic lung disease was more prevelant in group 1 than group 2 (46.9 ± 15.1 years vs 52.6 ± 15.9 years, $p = 0.03$ and 11.7% vs 3.1%, $p = 0.03$, respectively). Other demographic parameters including gender, HT, DM, CVD and body mass index (BMI) were similar in both groups. Initial clinical properties of acute COVID-19 infection were also similar in both groups. Initial laboratory and imaging features were similar in both groups, except for chest CT severity score (10.3 ± 9.6 vs 6.5 ± 7.6, $p = 0.02$) (Table 2).

The results of univariate and multivariate analysis were shown in Table 3. In univariate analysis, age,

Table 1. Demographic and clinical properties of study population.

	Long-COVID (n = 60)	No long-COVID (n = 97)	*p*
Demographic properties			
Age, years	46.9 ± 15.1	52.6 ± 15.9	0.03
Gender (male/female)	24/36	48/49	0.25
Hypertension, n (%)	14 (23.3)	25 (25.8)	0.73
Diabetes mellitus, n (%)	10 (16.7)	19 (19.6)	0.65
Chronic lung disease, n (%)	7 (11.7)	3 (3.1)	0.03
Cardiovascular disease, n (%)	6 (10.2)	6 (6.3)	0.4
BMI (kg/m^2)	27.1 ± 5.4	27.5 ± 4.8	0.4
Clinical properties			
COVID-19 disease severity, n (%)			
Mild	15 (25)	36 (37.1)	
Moderate	36 (60)	54 (55.7)	0.14
Severe	9 (15)	7 (7.2)	
Hospitalization, n (%)	25 (41.7)	27 (27.8)	0.07
Duration of hospitalization, day	4.2 ± 7.6	2.2 ± 4.0	0.09
ICU hospitalization, n (%)	3 (5)	0	0.05
Long COVID symptoms			
Fatigue, n (%)	45 (75)		
Dyspnea, n (%)	34 (56.7)		
Arthralgia or myalgia, n (%)	13 (21.7)		
Sleep disturbance, n (%)	13 (21.7)		
Mental confusion, n (%)	12 (20)		
Cough, n (%)	10 (16.7)		
Palpitation, n (%)	9 (15)		
Mood change, n (%)	9 (15)		
Change in smell and taste, n (%)	9 (15)		
Chest pain, n (%)	5 (8.3)		
Headache, n (%)	4 (6.7)		
Dizziness, n (%)	4 (6.7)		
Pins and needles, n (%)	3 (5)		
Irregular menstrual period, n (%)	1 (1.7)		

COVID-19: coronavirus disease 2019; BMI: body mass index; ICU: intensive care unit.

Table 2. Laboratory and imaging features of study population.

	Long-COVID (n = 60)	No long-COVID (n = 97)	*p*
Laboratory findings			
SpO_2	95.8 ± 3.5	96.8 ± 2.1	0.18
CRP, mg/L	31.5 ± 43.2	25.1 ± 37.7	0.45
WBC, K/µL	6.9 ± 2.6	6.2 ± 2.2	0.1
Lymphocyte, K/µL	1.9 ± 0.9	1.6 ± 0.6	0.25
Ferritin, ng/mL	178 ± 184.2	212.2 ± 295.9	0.46
D-dimer, µg/mL	0.7 ± 0.8	0.6 ± 0.8	0.23
Fibrinogen, mg/dL	415.1 ± 143.4	403 ± 122.5	0.73
Creatinine, mg/dL	0.8 ± 0.3	0.9 ± 0.7	0.1
Chest CT findings			
Pneumonic involvement, n (%)	45 (75)	61 (62.9)	0.11
Number of involved lung lobe	3.0 ± 2.1	2.3 ± 2.1	0.06
Chest CT severity score	10.3 ± 9.6	6.5 ± 7.6	0.015

SpO_2: Arterial blood partial oxygen pressure; CRP: C-reactive protein; WBC: White blood cell count; CT: Computed tomography.

SpO_2 and chest CT severity score were associated with long-COVID. However, only chest CT severity score (OR: 1.059, 95% CI: 1.002-1.119, $p = 0.04$) and age (OR: 0.953, 95% CI: 0.928-0.979, $p < 0.001$) were independently associated with long-COVID in multivariable analysis.

Discussion

In this study, the relationship between long-COVID and initial patient characteristics including clinical, laboratory and imaging findings was evaluated. We found that age and chest CT severity score were related to long-COVID. This is the first study that identified an independent relationship between chest CT severity score and long-COVID.

COVID-19 induced mortality has fortunately been controlled after effective vaccination. However, some COVID-19 survivors did not recover completely and presented persisting symptoms after acute infection. It was realized that these symptoms are caused by longer term effects of acute COVID-19 infection and is now called long-COVID [3]. This has been an important research area because of its limiting effects on the quality of life and personal productivity. Until today, several studies investigated possible clinical factors associated with long-COVID [2, 4-6]. Age, female gender, moderate or severe acute COVID-19, COPD, multiple symptoms at baseline and tobacco consumption were identified as risk factors in these studies. However, they did not take into account the possible effect of non-clinical parameters such as basic blood assays and chest CT findings. Some patients can have severe deterioration in these non-clinical parameters that might play a role in the development of long-COVID. For example, some unfortunate patients could have lung involvement with more severe damage or thromboembolic events during acute disease than others. Therefore, it is expected that blood assays and chest CT findings might be associated with long-COVID.

Chest CT was abundantly used during the COVID-19 outbreak because it was a quick way to get data about the effect on lungs. Furthermore, this data could be quantified by using some scoring systems such as chest CT severity score that allowed a more objective assessment of the lung. The chest CT severity score was one of the most useful parameters that has been reported to be independently associated with ICU admission, need for intubation and mortality during acute COVID-19 [9-12]. In addition, Steinbeis *et al* recently reported that the severity of chest CT in acute COVID-19 correlated with pulmonary function and respiratory symptoms over one year [13]. However, the possible effect of chest CT severity score on the development of long-COVID was unknown.

This study is the first to suggest the relationship between chest CT severity score and long-COVID. Some possible mechanisms might explain this relationship. Fibrosis can be proposed as the first explanatory mechanism because interstitial thickening and fibrosis as well as diffuse alveolar damage including alveolar epithelial cell destruction and hyaline membrane formation in lung tissue were suggested in patients with acute COVID-19 [14]. In

Table 3. Predictors of long-COVID symptoms.

	Univariate analysis			Multivariable analysis		
	OR	95% CI	*p*	OR	95% CI	*p*
Age	0.977	0.956-0.998	0.031	0.953	0.928-0.979	< 0.001
SpO_2	0.878	0.777-0.992	0.037	0.865	0.717-1.043	0.13
Chest CT severity score	1.053	1.013-1-095	0.008	1.059	1.002-1.119	0.04

COVID: coronavirus disease; SpO_2: arterial blood partial oxygen pressure; CT: computed tomography; CI: confidence interval; OR: odds ratio.

addition, fibrosis can develop in many other tissues and organs such as kidney, heart and liver [15-17]. Fibrosis is an irreversible structural change that can reflect longer term effect of COVID-19 on tissues and organs.

Vasculopathy might be second explanatory mechanism for this relationship. During acute COVID-19, the endothelium has a pivotal role in the immune response to SARS-CoV-2 that can cause endothelial dysfunction and damage presenting thrombosis and ischemia. This vasculopathy affects nearly any organ including the lung, myocardium, kidney, brain, skin and deep veins and may persist long after the initial infection has been eliminated [18-21]. In addition, there is a relationship between chest CT severity score and vasculopathy in patients with COVID-19 pneumonia [22]. Thus, we propose that higher chest CT severity score during acute COVID-19 might reflect an increased risk for fibrosis and vasculopathy during follow-up.

On the other hand, our findings have clinical importance in practice. The initial chest CT severity score may be used to predict the risk of patients for long-COVID.

This study has some limitations. First, our study population had a relatively small number of patients. However, we created a homogenous study population by using carefully selected exclusion criteria. Thus, our study results are reliable and valuable. Second, possible improvements or worsening of initial chest CT findings during the follow-up period was not taken into account because no chest CT imaging was made after hospital discharge. However, the main hypothesis of this study was to evaluate the effect of initial chest CT severity score on long-COVID. Third, we did not have a chance to assess the presence of fibrosis or vasculopathy by an objective laboratory method. Therefore, our results should be confirmed by further studies.

Conclusions

There was an independent relationship between initial chest CT severity score and long-COVID. This may be used to predict the patients who have a risk of developing long-COVID symptoms.

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