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EVALUATION OF BIOCHEMICAL PARAMETERS IN CORONARY ARTERY PATIENTS WITH COVID-19 INFECTION

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Keywords

COVID-19, Coronary artery disease, Biochemical parameters

ABSTRACT

As a result of pneumonia brought on by coronavirus disease (COVID-19), acute myocardial infarction, and chronic cardiovascular system damage, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infects host cells via ACE2 receptors. In patients with coronary artery disease (CAD) who are positive for COVID-19, early measurement of cardiac damage through biomarkers and careful monitoring of myocardial damage that may be caused by infection should be performed. The goal of this study was to retrospectively assess the results of biochemical laboratory testing in patients with CAD and COVID-19. Biochemical laboratory results of 70 patients with CAD and COVID-19 and 70 patients only with CAD were examined retrospectively. A significant difference was detected between groups in terms of LDH, CRP, and Troponin I parameters. These biomarkers are important to prevent and rapidly treat COVID-related myocardial damage in patients with CAD.

INTRODUCTION

An atypical viral pneumonia outbreak was caused by the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), which emerged at the end of 2019 in Wuhan, China. This coronavirus infection, also known as COVID-19, has rapidly spread over the world due to its high transmissibility[1]. COVID-19, a multisystemic illness, infection mostly affects the respiratory system. Increased pro-inflammatory cytokine and chemokine production, altered cellular immunological responses, cardiovascular and respiratory failure, abnormal coagulation indices, organ damage, and even death have all been displayed in severe COVID-19 instances[2]. These are all symptoms of SARS-CoV-2 infection of host cells through angiotensin-converting enzyme receptors (ACE2)[2,3]. The cardiovascular and immune systems depend heavily on the membrane-bound aminopeptidase ACE2. ACE2 has been linked to the onset of diabetes mellitus and hypertension in addition to its association with heart health. Furthermore, it has been discovered that ACE2 functions as a receptor for coronaviruses and SARS-CoV-2 infection is triggered by the viral spike protein binding to ACE2, which is abundantly expressed in the lungs and heart. Respiratory symptoms are brought on by SARS-CoV-2, which principally damages alveolar epithelial cells[3].

It is hypothesized that the cardiac injury lead by COVID-19 infection may have an important role in the progression of intense clinical manifestations or negative outcomes in infected people. In patients with COVID-19, myocardial injury is closely correlated with disease severity and even prognosis. Further preventive measures should be taken in patients with pre-existing coronary artery disease (CAD) in relation with the negative effects of COVID-19 on the cardiovascular system[4]. Therefore, it is crucial that patients with COVID-19 be treated promptly

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to reduce mortality once the relevant symptoms appear. Therefore, in a patient with CAD, early measurement of cardiac damage via biomarkers and careful monitoring of any myocardial damage that may be caused by the infection are recommended after hospitalization for COVID-19[5,6].

Especially in the presence of concomitant CAD, COVID-19 infection is severe and the risk of death increases. COVID-19 infections can lead to many clinical pictures ranging from myocardial infection, myocarditis, heart failure, and arrhythmias to venous thromboembolism. Mechanisms such as inflammation, cytokine storm, increased coagulation functions, as well as an imbalance between myocardial oxygen supply and myocardial oxygen demand cause cardiac damage in COVID-19 infections[7]. The goal of this study is to retrospectively assess the results of biochemical laboratory testing in CAD patients who have positive COVID-19 results.

MATERIALS AND METHODS

Study Subjects

This is a retrospective study. The study group included 70 patients with CAD who had a positive PCR test followed up in the Cardiology Service at Mersin University Hospital between September 2020 and December 2021 and the control group included 70 patients with CAD who presented with clinical complaints with suspicion of COVID-19 but whose PCR test was negative. Patients with non-reference values due to malignancy, liver, or kidney disease were excluded from the study. The Mersin University Clinical Research Ethics Committee approved the study (11.05.2022, 2022/330).

Data Collection

The demographic characteristics and laboratory data of all participants were collected from their individual medical records retrospectively. In addition to their age, gender, COVID-19 PCR test results, biochemical data such as urea, Creatine Kinase (CK-MB), C Reactive Protein (CRP), Lactate dehydrogenase (LDH), creatinine, Troponin I, aspartate aminotransferase (AST), and lipid profiles (low-density lipoprotein (LDL), triglyceride (TG), high-density lipoprotein (HDL), total cholesterol), and alanine aminotransferase (ALT) were examined. AST, ALT, LDH, CK-MB, and urea were measured by an enzymatic method, creatinine by the colorimetric Jaffe method, and C Reactive Protein (CRP) by a turbidimetric method in an autoanalyzer (AU680, Beckman Coulter Inc. Japan). The lipid profile was studied via an enzymatic colorimetric method in a Cobas 501 autoanalyzer (Roche Diagnostics, Manheim GmbH, Germany). Troponin I was detected by the chemiluminescence immunoassay method on DXI 800 (Beckman Coulter, Inc.)

Statistical Analyses

It was determined using the SPSS Statistics software, demo version 22 (trial version, IBM Corporation, Somers, NY). The Box-Plot chart method was used to find and eliminate outliers in the continuous data. To detect if the distribution of continuous variables was normal, the Shapiro-Wilk test was applied. For continuous variables, interquartile range (25th–75th percentiles) was used to express mean, median, and SD. Depending on whether the distributions were normal, either Student's t-test or the Mann-Whitney U test was used to determine whether there was a difference between the mean/median values of the patient and control groups for each parameter. Statistics were judged significant for a p value less than 0.05.

FINDINGS

The mean age of the patient group (55 men and 15 women) and controls (48 men and 22 women) was 60.67±10.83 and 58.04± 6.69, respectively. Biochemical parameters are given in Table 1. LDH, CRP, and Troponin I parameters showed a significant difference between the groups. Although CK-MB level was higher in the patient group, it was not statistically significant.

DISCUSSION

In the literature, it was observed that 40% of the patients hospitalized due to COVID-19 had a history of cerebrovascular or cardiovascular disease and 7% had acute cardiac damage at the time of diagnosis and follow-up. Clinical presentation of patients with COVID-19 was considered to have acute cardiac injury. Enzyme elevation (hs-cTn), which indicates cardiac damage, alone is insufficient for the diagnosis of acute cardiac injury, especially acute MI[5,6]. For diagnosis, it is essential to assess symptoms, ECG, and CKMB elevation. In our study, Troponin I and CK-MB parameters were higher in the

patient group to controls, and the elevation in the troponin level was statistically significant. The most likely cause of cardiac enzyme elevation and myocardial damage in the course of COVID-19 is the direct myocardial damage caused by the virus. Due to the intense distribution of ACE2 in the binding site of SARS-CoV-2 in cardiac myocyte cells, the virus has a high affinity for these cells, and therefore, it causes destruction. It is held responsible for the increase in troponin by causing myocarditis [1,5,7,8]. Also, it was reported that there was cardiac damage and there was a significant increase in levels of cardiac troponin I (hs-cTnI) in five of the 41 patients with COVID-19 who received a diagnosis in Wuhan[9]. In another study, there was an increase in the CK-MB along with hs-cTnI in COVID-19 patients, and the reason for this outcome was thought to be complications including acute myocardial damage in patients with severe symptoms. Nevertheless, 58% of patients with severe COVID-19 symptoms were reported to have hypertension, 25% to have heart disease, and 44% to have arrhythmia[10]. Of patients with SARS-CoV-2, 35% had a history of hypertension, and 17% had a history of CAD, according to mortality statistics published in China[10]. Additionally, data show that systemic symptoms are more severe and the incidence of severe pneumonia is higher in patients over 60 years of age and infected with SARS-CoV-2 than in patients aged ≤60 years. It was reported that underlying CAD may aggravate pneumonia and increase the severity of symptoms in patients with SARS-CoV-2 infection[11].

Elevated levels of AST, ALT, and LDH are signs of damaged liver. In COVID-19 illness, immune-mediated injury resulting from a significant inflammatory response, direct cytotoxicity brought on by active viral replication in biliary epithelial cells expressing ACE2, hypoxic hepatitis brought on by anoxia, and drug-induced liver injury are all possible clinical causes[12]. According to our results, there was no difference between the groups in terms of AST and ALT, but LDH was higher in the patient group. Also, no statistical difference was detected between the patient group and controls in terms of lipid profile, urea, and creatinine levels. Most COVID-19 patients (75%–93%) have increased CRP levels[13]. Furthermore, we discovered that CRP, an inflammatory marker, was higher in the patient group compared to controls.

Our study had some limitations because it was retrospective. These limitations were that it was studied in a single center and that the results of D-dimer, Fibrinogen, and Ferritin, which are important in COVID-19 infection and CAD, were not complete in patient files.

CONCLUSION

In conclusion, myocardial damage in patients with COVID-19 is closely related to the severity and even progression of the disease. To reduce mortality, it is important to treat patients with CAD as soon as relevant COVID-19 symptoms appear. Therefore, early assessment of cardiac damage with these biomarkers is important to prevent and rapidly treat COVID-related myocardial damage in patients with CAD.

Table 1. *Biochemical parameters of the patient and control groups.*

Parameter	Patient n:70	Control n:70	p
AST* (U/L)	23.5 (16.5-32.5)	22.05(19-27.1)	0.64
ALT* (U/L)	21.7 (17.1-28.1)	22 (15-28.4)	0.57
LDH* (U/L)	183 (160-214)	161.5 (141-178)	0.004
CK-MB* (U/L)	94.5 (63-257)	80 (50-120)	0.072
CRP * (mg /dL)	5.29 (2.80-12.65)	1.85(0.75-3.35)	0.00001
Troponin I*(ng/mL)	3.11 (2.9-5.4)	0.004(0.002-0.006)	0.002
Urea # (mg /dl)	25.87±8.21	26.45±7.96	0.67
Creatinine# (U/L)	0.817±0.22	0.76±0.17	0.09
Total Cholesterol# (mg/dl)	179.89±42.84	184.3±61.03	0.61
HDL # (mg/dl)	43.6 ±20	41.36±12.7	0.44
LDL # (mg/dl)	104.02±37.09	105.36±41.2	0.87
Triglyceride# (mg/dl)	177.03±82.07	164.5±86.4	0.4

*# Continuous variables are given as mean±standard deviation, * variables are given as median (IQR: interquartile range) p: significance between groups, n: Number of samples.*

Conflict of interest statement

The author declares that they have no conflicts of interests.

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