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Olgu Sunumu / Case Report

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A RECOVERED PATIENT AFTER MYOCARDIAL INJURY RELATED TO COVID-19; A CASE REPORT

COVİD-19 HASTALIĞINA BAĞLI MİYOKARD HASARINI İYİLEŞEREK ATLATAN BİR HASTA; VAKA SUNUMU

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ABSTRACT

Angiotensin converting enzyme 2 receptors are reported to have an important role in myocardial injury in Covid-19. Direct myocardial toxic effect of SARS-CoV-2 virus, myocarditis, cytokine injury, microangiopathic injury related to infection may cause myocardial injury and eventually troponin levels increase. Mechanisms causing myocardial injury are; unstable coronary artery plaques resulting in myocardial hypoxia, upregulation of angiotensin converting enzyme 2 receptors in coronary arteries and cardiac tissue, direct toxic effect of virus towards myocytes, systemic inflammation, myocardial interstitial fibrosis, excessive immune response to interferon, increased cytokine release from helper T lymphocytes which lead to myocardial injury. In this reported case, a 47 year old male patient, recovered from Covid-19 who had mild clinical course and good outcome was reported. Patient was followed in ward and he did not need intensive care.

Keywords: *Covid-19, myocardial injury, troponin*

ÖZET

Anjiyotensin dönüştürücü enzim 2 reseptörleri Covid-19'a bağlı miyokard hasarında önemli bir role sahiptir. SARS-CoV-2 virüsünün miyokarda direk toksik etkisi ve enfeksiyona bağlı gelişen miyokardit, sitokin hasarı, mikroanjiyopatik hasar neticesinde miyokard hasarı gelişir ve neticede troponin seviyeleri yükselir. Miyokard hasarına neden olan mekanizmalar; miyokardda hipoksiye neden olan anstabil koroner arter plakları, kalp dokusunda ve koroner arterlerde Anjiyotensin dönüştürücü enzim 2 reseptör upregülasyonu, virüsün miyokard hücrelerine direk toksik etkisi, sistemik inflamasyona bağlı doku hasarı, miyokarda interstisyel fibroz, interferon etkisine bağlı abartılı immün cevap, helper T lenfositlerinden sitokin salınmında artış olarak belirtilebilir. Bu bildirilen vaka, Covid-19 hastalığına bağlı gelişen miyokard hasarını iyileşerek atlatmış, klinik seyri ve gidişatı iyi seyretmiş 47 yaşında erkek bir hastadır. Hasta serviste yatırılarak izlemiştir ve takipte yoğun bakım ihtiyacı gelişmemiştir.

Anahtar Kelimeler: *Covid-19, miyokard hasarı, troponin*

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INTRODUCTION

SARS-CoV-2 binds to angiotensin converting enzyme 2 (ACE2) in order to enter host cells. Since ACE2 receptors exist profoundly in cardiovascular system, it can be a target for SARS-CoV-2 infection. ACE2 receptors are reported to have an important role in myocardial injury in Covid-19 (1). Direct myocardial toxic effect of virus, myocarditis, cytokine injury, microangiopathic injury related to infection may cause myocardial injury and eventually troponin levels increase (2). Most of patients having myocardial injury usually complain from high fever, cough, dyspnea and myalgia which are frequent symptoms in Covid-19, but not from symptoms specific to myocardial injury. Differential diagnosis should consider acute coronary syndromes, stress related cardiomyopathy, sepsis related cardiomyopathy and especially in older patients coronary angiography may be necessary (3). A patient who had acute myocardial injury related to Covid-19 will be reported in this paper.

Presentation Of Case

A 47 years old, Caucasian, male patient complaining from high fever, headache and myalgia admitted to outpatient clinic of hospital. He has been hospitalized for Covid-19. He had history of contact with a Covid-19 patient and he did not have any accompanying disease and he was not using any medication.

Physical examination revealed high fever (38.5 Celsius, forehead, infrared thermometer) and on auscultation respiratory sounds were coarse. Other examinations were normal. Laboratory findings are reported in Table I. SARS-CoV-2 PCR test was positive. Thorax CT revealed specific basal peripheral ground glass opacifications bilaterally. According to national Covid-19 treatment guide (4) hydroxychloroquin 2x200 mg and azithromycin 1x500 mg was started. Vitamin C 4x1000 mg was also given as supplemental treatment. On day 4, patient had palpitations, electrocardiogram was normal, he had no chest pain, echocardiograpy performed by cardiologist could not reveal any pathologic finding. But troponin I levels were high as 5.14 ng/ml (normal: up to 0.3 mg/L). Coronary angiography was performed, but did not reveal any coronary artery disease. The patient was then, diagnosed as myocardial injury due to Covid-19. On follow up troponin levels were 3.87 ng/ml (treatment day, 7), 3.83 ng/ml (day 8) and 3.66 ng/ml (day 9). On tenth day, his control PCR for SARS-CoV-2 was found negative and he was discharged.

Discussion

SARS-CoV-2 causes Covid-19 disease which greatly resembles SARS-CoV disease experienced at 2003. Thus, myocardial injury mechanisms are thought to be similar. Those mechanisms are; unstable plaques in coronary arteries resulting in myocardial hypoxia, upregulation of ACE2 receptors in coronary arteries and cardiac tissue, direct toxic effect of virus towards myocytes, systemic inflammation, myocardial interstitial fibrosis, excessive immune response to interferon, increased cytokine release from helper T lymphocytes which lead to myocardial injury (5-7).

Autopsy findings of deceased Covid-19 patients revealed mononuclear cell infiltrations, but SARS-CoV-2 itself could not be demonstrated (8). SARS-CoV-2 causes myocarditis and ischemia, thus results in myocardial injury and increase in troponin levels (9). In a study with 41 Covid-19 patients reported from China, 12% had myocardial injury related to infection (10). Another study reported increased troponin levels in 15% of patients (11). Data from Covid-19 treatment ward in our hospital from where current case is being reported, reveals that 3 (5%) patients out of 65 who had been diagnosed as Covid-19 had troponin levels increased. Only one patient (2%) had myocardial injury (prepublished information). A study covering 138 patients in China, reported 7% had acute myocardial injury and 80% of these was followed in intensive care unit (12). There are many unknown facts about underlying mechanisms and frequency of acute

Table 1. Laboratory findings of patient

Parameter	Value	Normal Range
White blood cell count (x10^9L)	5.31	4-11
Neutrophil count (x10^9L)	3.14	2-7
Hemoglobin (g/dl)	14.80	13.5-18
Creatinin (mg/dl)	1.08	0.7-1.2
PTT-INR[1]	1.16	0.8-1.2
C-reactive protein (mg/L)	4.40	0-5
Procalcitonin (ng/ml)	0.02	0-0.5
Creatinin kinase (U/L)	49	39-308
Lactate dehydrogenase (U/L)	135	135-225
D-dimer (ng/ml)	284	0-500
CK-MB[2] (ng/ml)	1.20	0-5
Troponin I(ng/ml)	5.14	0-0.3

[1]Prothrombin time-International normalized ratio
[2]Creatinin kinase-myocardial band

myocardial injury in Covid-19, yet waiting to be clarified.

In this reported case, clinical course was mild and outcome was good, patient was followed in ward and he did not need intensive care. Eventually, he was discharged with clinical improvement after SARS-CoV-2 infection. If can be emphasized that studies are required to inspect Covid-19 related myocardial injury and explain its mechanisms.

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REFERENCES

1.)Wang Y, Roever L, Tse G, et al. 2019-Novel Coronavirus-Related Acute Cardiac Injury Cannot Be Ignored. Curr Atheroscler Rep. 2020; 22(3): 14. doi: 10.1007/s11883-020-00842-y.

2.)Bennett CE, Anavekar NS, Gulati R, et al. ST-segment Elevation, Myocardial Injury, and Suspected or Confirmed COVID-19 Patients: Diagnostic and Treatment Uncertainties. Mayo Clin Proc. 2020; 95(6): 1107–1111. doi: 10.1016/j.mayocp.2020.04.005

3.)Caforio ALP, Coronavirus disease 2019 (COVID-19): Myocardial injury. Available from: URL: http://www.uptodate.com/contents/coronavirus-disease-2019-covid-19-myocardial-injury, Accessed 28th May, 2020.

4.)Turkish Ministry of Health Covid-19 Information Website. Available from: URL: https://covid19bilgi.saglik.gov.tr/depo/rehberler/COVID-19_Rehberi.pdf, Accessed 28th May 2020.

5.)Inciardi RM, Lupi L, Zaccone G, et al. Cardiac Involvement in a Patient With Coronavirus Disease 2019 (COVID-19). JAMA Cardiol 2020. doi: 10.1001/jamacardio.2020.1096

6.)Chen C, Zhou Y, Wang DW. SARS-CoV-2: a potential novel etiology of fulminant myocarditis. Herz 2020; 45(3): 230-232. doi: 10.1007/s00059-020-04909-z

7.)Babapoor-Farrokhran S, Gill D, Walker J, et al. Myocardial Injury and COVID-19: Possible Mechanisms. Life Sci. 2020; 253: 117723. doi: 10.1016/j.lfs.2020.117723

8.)Xu Z, Shi L, Wang Y, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. Lancet Respir Med. 2020; 8(4): 420–422. doi: 10.1016/s2213-2600(20)30076-x

9.)Madjid M, Safavi-Naeini P, Solomon SD, et al. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. JAMA Cardiol 2020; 5(7): 831. doi: 10.1001/jamacardio.2020.1286

10.)Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020; 395(10223): 497–506. doi: 10.1016/S0140-6736(20)30183-5

11.)Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020; 395(10223): 507–13. doi: 10.1016/s0140-6736(20)30211-7

12.)Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan, China. JAMA. 2020; 323(11): 1061. doi: 10.1001/jama.2020.1585

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