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Case Report

Infective Endocarditis and Multi-Infarct Coexisting With Covid-19

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ABSTRACT

Covid-19 infection is an important public health problem that has turned intoan outbreak. The disease is highly contagious, and its main clinical symptoms are fever, non-productive cough, fatigue, myalgia, and dyspnea. The virus is mainly transmitted by droplets. Death risk due to Covid-19 increases in patients with hypertension and cardiovascular disease. The relationbetween Covid-19 and Infective Endocarditis (IE) has been published in the literature. In this article, we aimed to discuss the relation between Covid-19 and Infective Endocarditis (IE) in the light of literature, upon the detection of Covid-19 in a patient with heart valve operation due to infective endocarditis.

INTRODUCTION

An epidemic of pneumonia, thought to be related with a novel coronavirus, was reported in Wuhan, China, in December 2019. This virus rapidly spread throughout the world and led to a pandemic. In studies, hypertension, cardiovascular diseases, chronic lung disease, cancer, diabetes, and older age have been identified as risk factors for Covid-19-related morbidity and mortality [1]. Covid-19 infection symptoms occur after an incubation period of approximately 5.2 days. The average time from the onset of Covid-19 symptoms to death is known as 14 days, but sometimes it varies between 6 and 41 days [2]. It can lead from clinically asymptomatic to acute respiratory distress syndrome and multiorgan failure. The most common clinical features include fever, cough, sore throat, increased mucus expectoration, headache, myalgia, fatigue and dyspnea. In addition to these, Covid 19 can present with gastrointestinal symptoms (diarrhea, nausea, vomiting) [3].

Laboratory examination results show that most patients have normal or decreased white blood cell count and lymphocytopenia. However, in severe patients, neutrophil count, D-dimer, blood urea nitrogen and creatinine levels increase significantly, lactate dehydrogenase, bilirubin and alanine aminotransferase increase, and hypoalbuminemia may occur. Creatine kinase, ferritin and troponin increase can be seen in patients. In addition, inflammatory factors Interleukin (IL) -6, IL-10, tumor necrosis factor-α increase occurs. Complications include acute respiratory distress syndrome, arrhythmia, shock, acute kidney injury, acute heart damage, liver dysfunction, and secondary infection [4,5].

CASE

A 62-year-old male patient was admitted to our clinic with fever, fatigue symptoms and covid-positive (PCR). In the history of the patient, he was admitted to the hospital with weakness on the left side of his body on 14.04.2020 with a diagnosis of cerebrovascular disease. In laboratory tests, glucose 92mg/dl (80-110), BUN 14mg/dl (7-28), serum creatinine 0.8mg/dl (0.6-1.3), CRP 63mg/dl (0-5), Hb 13,5mg/dl, thrombocyte 228000/mm3, normal thyroid hormones, trace protein in complete urinalysis, d-dimer 0.78 ug / ml (0-0.5), respiratory acidosis in blood gas. In the cerebral tomography, an atrophic pattern was observed in the cerebellar sulci in the posterior fossa, and fibrotic streaks were detected in the apical segment of the upper lobe of the right lung on thorax tomography. Diffusion restriction consistent with early infarction was detected in the right occipital cortex and subcortex in the right parietal silvian sulcus location on diffusion magnetic resonance imaging of the patient. Lower extremity doppler and echocardiography were found to be normal. Antibiotics, proton pump inhibitors, nonsteroidal anti-inflammatory drugs, acetylsalicylic acid and clopidogrel were started to be performed. The patient was discharged with acetylsalicylic acid and clopidogrel. On 06.05.2020, he was re-admitted to a different hospital with the complaint of left-sided weakness. In the laboratory examination, glucose was 113 mg/dl, BUN 16 mg/dl, serum creatinine 0.7 mg/dl, CRP 49 mg/dl, and sedimentation 20 mm/hour. In thoracic tomography, fibrotic recessions were observed in both lung apices, pleuropenchymal band in the left lung lower lobe laterelobasal segment, and a 3mm diameter nodular appearance in the lateral segment of the right lung lower lobe located close to the pleura.In cranial magnetic resonance imaging, atrophy in the cerebellar sulcus, prescentral and postcentral gyrus cortical and subcortical edema, adjacent to the right central sulcus, T2A flair hyperintense signal increase and diffusion restriction area in diffusion-weighted images (acute-subacute infarct), patchy areas of T2A in both cerebral white matter flair hyperintense signal change is observed (ischemic gliotic changes). Later, on 10.06.2020, he applied to the emergency department with abdominal pain, fever, cough,dyspnea and diarrhea. In laboratory tests (10.06.2020) glucose was 123 mg/dl, BUN 10 mg/dl, serum creatinine 0.6 mg / dl, sodium 130 mEq / L (135-145), potassium 4.1 mEq / L (3.5 -5.5), procalcitonin 0.13 ng / ml (0-005), CRP 96 mg / L, leukocyte count 11.9 (4.5-11), Hb 11.2 g / dl (13.5- 17.5), platelet count 244000 / mm3, sedimentation 56 mm / hour (0-20).

Abdominal tomography showed hepatosteatosis, a large non-contrast hypodense area in the central part of the spleen, which is compatible with focal splenic infact, and a 1.5 cm hypodense area compatible with parenchymal non-enhancing focal infact was observed at the middle and upper pole of the left kidney (Figure 1). In brain magnetic resonance imaging, diffusion restriction was observed in the right parietal region extending to the lateral ventricle neighborhood (acute and subacute infact).

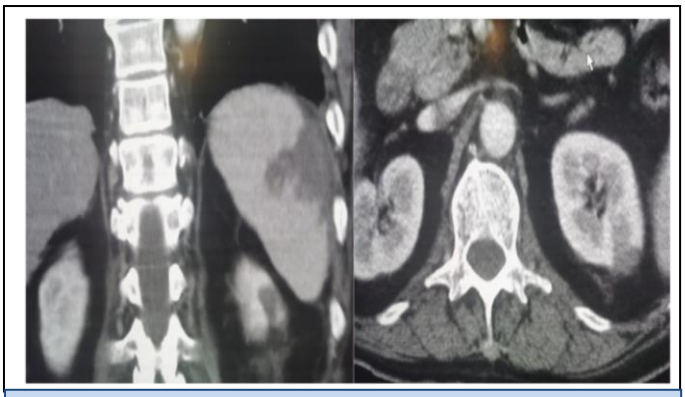

Figure 1: In the middle part of the left kidney, an infarct involving the cortex in the posterior part, and a wide-based wedge-shaped hypodense infarct in the parenchymal and especially peripheral area in the middle part of the spleen were detected.

Thoracic tomography revealed wall thickening in both lungs, locally fibrotic bands in the right lung, posteriorly fibroatelectasis in the bilateral lower zones, and minimal pleural effusion on the right. The patient could not be hospitalized because the hospital was totally loaded and transferred to an other hospital. As a result of the examinations performed there, infective endocarditis was diagnosed, and the patient underwent a mitral valve operation (03.07.2020). As the etiology of infective endocarditis, brucella agglutination test (1/80) was found and treatment was started for it, but no valve culture was performed, and any microorganisms were reproduced in blood cultures. CRP was high, sedimentation was 56 mm / hour, ferritin 456 microg / L (30-300), thyroid hormones and urine samples were normal. Acute kidney injury (serum creatine 1.5 mg / dl) and anemia (Hb 10 g / dl)

developed during follow-up. The patient was transferred to our hospital after Covid-19 PCR test performed on 11.07.2020 was positive. In laboratory examination, glucose 94 mg / dl, BUN 17 mg / dl, creatinine 1.49 mg / dl, CRP 49 mg / dl, sedimentation 69 mm / hour, lactate dehydrogenase 517 IU / L (135-225), troponin 79.9 ng / L (0-14), ferritin 1742 microg / L, D-dimer 3.21 ug / ml, Hb 10 g / dl, platelet 214000 / mm, TSH was detected as 20 (high) and FT4 as 0.5 (low).Brucella treatment was continued and faviripavir, hydroxychloroquine, vitamin C and anticoagulant were started. There was no reproduction in their culture. Levothyroxine was started in the patient with hypothyroidism. Plasma treatment was given to the patient twice. On 27.07.2020, glucose 96 mg / dl, BUN 26 mg / dl, creatinine 1.67 mg / dl, LDH 728 IU / L, CRP 9 mg / dl, D-dimer 3.37 ug / ml, troponin T 44, 8 ng / L, procalcitonin 0.07 ng / ml, Hb 9.9 g / dl, platelet count 331000 / mm3. Upon the improvement of the patient's complaints, he was discharged with recommendations.

DISCUSSION

Infective Endocarditis (IE) is an infection that usually develops in the endocardial surface of the heart with impaired valve structure or anatomical defects. IE, previous endocarditis; with valvular heart disease, congenital heart disease and intracardiac foreign body; intravenous drug user; It is more common in patients undergoing chronic hemodialysis, solid organ and hematopoietic stem cell transplantation compared to the normal population. Although IE is rare, it is still an important infectious disease due to its morbidity and high mortality rate.

Covid-19 affects many organs. Although pulmonary involvement is the most common, cardiovascular system, cerebral and renal involvements are detected. COVID-19 can lead to cardiac involvement and injury through the following possible mechanisms: 1-Indirect injury due to increased cytokines and immune-inflammatory response, 2-Direct invasion of cardiomyocytes by the virus, 3-It develops as a result of respiratory damage from the virus that causes hypoxia that causes oxidative stress and damage to cardiomyocytes. It is associated with multiple direct or indirect cardiovascular complications such as myocarditis, ventricular dysfunction, heart failure, pericardial effusion, infective endocarditis, myocarditis, myocardial injury, arrhythmia, and venous thromboembolism. As can be seen, cardiovascular diseases is an important cause for hospitalization and the cause of death in this patient group [6,7].

Covid 19 and IE association is rarely reported in the literature. Amir et al. Found IE, acute decompensated heart failure and nasopharyngial real time PCR Covid 19 positivity in a 61-year-old male patient who presented with dyspnea, fever and mild non-productive cough. The patient's history included hypertension and smoking [8]. A 69-year-old female patient who presented with fatigue and cough, had fever and dyspnea one week ago. There is aortic valve replacement in her medical history. As a result of the evaluations, it was found that the patient who was found to have staphylococcus aurius in the blood culture, aortic valve infective endocarditis, gastrointestinal and intracerebral hemorrhage was found to be Covid 19 positive [9]. Benmalek et al. Detected Covid 19 and tricuspid valve endocarditis in a 76-year-old female patient (history of DM and HT). In the catheter culture of the patient, coagulase negative staphylococcus and candida albicans grew in the urine culture [10]. Nonbacterial infective endocarditis was detected in a 76-year-old female patient with Covid 19 positive. Non-new cerebral infarction detected. Later, cerebral, spleen, and renal multiple infarcts were detected [11].They detected multiple renal infarcts in a 62-year-old male patient (kidney transplant in his history) on computed tomography scan to exclude pulmonary embolism. In addition, intestinal and renal infarction was detected in a 58-year-old male patient [12]. In our case, infarction was found in the cerebral, renal and spleen. Our patient did not have valvular disease on echocardiography at his previous hospitalizations, and his Covid 19 PCR test was positive after he was operated with a heart valve surgery before coming to our hospital. Although IE was brucella in terms of etiology, the diagnosis of brucella was not definite. We think that Covid 19 positivity may be in the etiology of IE.

Ramos-Martínez et al. Detected hospital-acquired IE in 4 cases during the Covid 19 outbreak. All patients had risk factors for IE. In the blood cultures of these patients; enterococcus fecalis grew in two, candida albicans in one, and methicillin-sensitive staphylococcus aureus in the other. As a result of this study, it was suggested that the risk of hospital-acquired IE increased during the Covid 19 outbreak [13]. In the study in which 1216 patients with Covid 19 positive were included, abnormal

echocardiography findings were detected in 667 patients. Left ventricular abnormalities were found in 479 patients and right ventricular abnormalities in 397 patients, myocardial infarction in 36 patients, myocarditis in 35, cardiomyopathy in 19, tamponade in 11 patients, and endocarditis in 14 patients [14].

Various therapeutic and preventive strategies have been used against previous Coronavirus outbreaks, including vaccines, immunotherapy, and antiviral drugs. These treatments have been attempted for the current Covid 19. Among the effective drugs to manage Covid-19 patients, remdesivir can be used alone with lopinavir / ritonavir or interferon beta, in conjunction with conventional plasma and monoclonal antibodies, but large studies are needed. Chloroquine and hydroxychloroquine are used. Various therapeutic agents have been proposed for the clinical management of Covid-19, such as lopinavir / ritonavir, chloroquine, and hydroxychloroquine. Favipiravir and hydroxychloroquine are effective against Covid 19. When necessary, IL6 inhibitor or IL1 receptor antagonist is given [5,13]. As a result, there is an uncommon relationship between Covid 19 and cardiovascular disease. IE is rarely seen in patients with Covid 19, and covid 19 increases the risk of IE. In our case, we think that the positive PCR of Covid 19 in the patient who was diagnosed with IE and the absence of reproduction in the cultures may have a role in the etiology of IE.

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