CASE REPORT

Symptomatic COVID-19 Reinfection with Pericardial and Pleural Involvement

Bedriye Koyuncu Sokmen[1], Tugce Hurkal[2]

Abstract

Coronavirus disease 2019 (COVID-19) is a contagious acute respiratory tract infection caused by Severe Acute Respiratory Syndrome CoronaVirus-2 (SARS-CoV-2), a beta coronavirus first discovered in Wuhan, China in late 2019. COVID-19 is spreading rapidly globally and has been officially declared a pandemic by the World Health Organization (WHO) as of March 2020. Most recent studies suggest that immunity can develop after an episode of severe acute respiratory syndrome Infections. There are few cases with severe symptomatic reinfection. Here we present the case of a healthy 46-year-old man with pericardial-pleural and lung involvement in the setting of COVID-19 infection first, and severe symptomatic reinfection thereafter.

Keywords: COVID-19 reinfection, COVID-19 pneumonia, lung CT.

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Introduction

CoronaVirus-2 was first discovered in the sputum of patients with severe acute respiratory distress syndrome in December 2019 (SARS-CoV-2).[1] COVID-19 infection can cause multiple health problems due to the direct and indirect pathophysiological effects.[1,2] Typically, COVID-19 is mainly manifested by respiratory symptoms and lung injury, however, with the multiplied cases, awareness of cardiovascular involvement has increased.[2] Accordingly, several cases of pericardial involvement have been reported during and after symptomatic COVID-19.[2] Most recent studies suggest that immunity can develop after an episode of infection.[3] Symptomatic re-infection with COVID-19 was rare in literature.[4] Here, we present a case of a healthy 46-year-old man with pericardial and pleural involvement in his first infection, and severe symptomatic re-infections in the setting of COVID-19 infection. Informed consent was provided by the patient for publication of the case report.

Case Report

A 46-year-old, otherwise healthy man was diagnosed with COVID-19 on March 31, 2020 with nasopharyngeal RT-PCR (real-time reverse transcriptase-polymerase chain reaction) done twice for confirmation. The case was seen in March 2020 at the Istanbul Florence Nightingale Hospital. There was no significant past medical history. The patient was a non-smoker with no family history of malignancies. The patient's main symptom was shortness of breath. Only C-Reactive Protein (CRP) elevation was present and other blood count was normal including complete blood count, coagulation parameters, and other routine biochemical values. A chest X-ray showed costophrenic sinus blunting. A Computerized Tomography (CT) scan revealed right pleural effusion, pericardial effusion, and mediastinal-hilar lymph nodes, with no pulmonary parenchymal involvement. (Figure-1) Thoracocentesis was performed and pleural fluid (PF) analysis was consistent with an exudative effusion (PF protein/serum protein ratio was 0,6 and PF LDH/serum LDH ratio was 0,7). No organism was detected on routine pleural fluid culture as well as on Acid-Fast Bacilli (AFB) culture and Tuberculosis (TB) PCR was negative. Also, the pleural fluid cytology was negative for malignant cells. It was thought that this was probably secondary to COVID-19 infection, and so he was treated with Hydroxychloroquine and azithromycin. Diuretics were also added to the treatment. Even after treatment with antibacterials (after 1 week), the patient's chest pain and

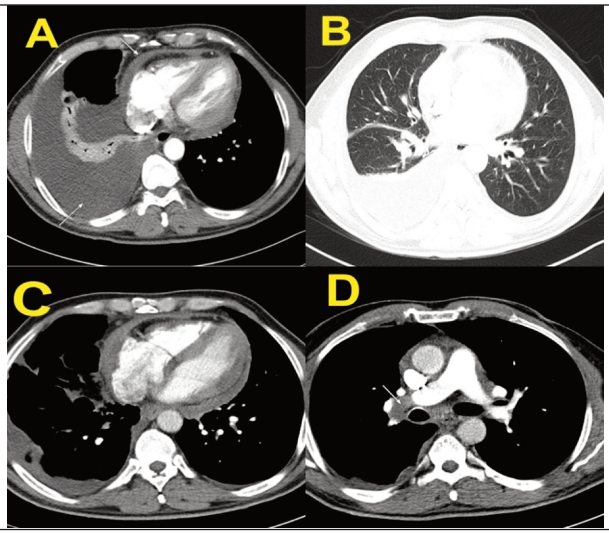

Figure-1: A and B: Pleural effusion and pericardial effusion filling the right hemithorax near total in the axial mediastinum and parenchymal window in lung CT, C and D: Regression of pleural effusion and right hilar lymphadenopathy on lung CT.

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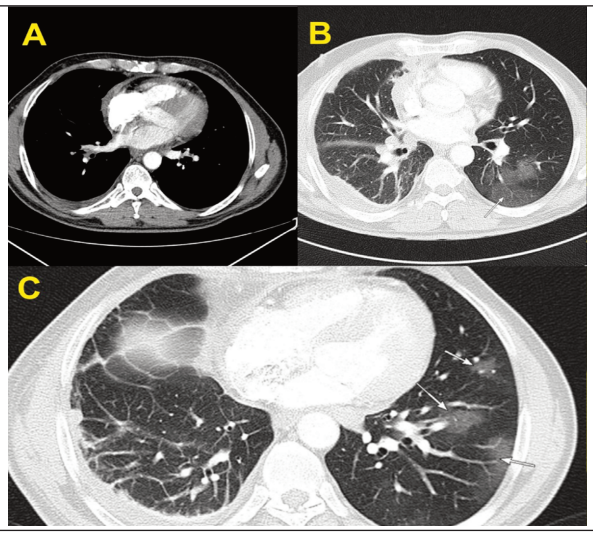

Figure-2: A: Near-total regression in pleural and pericardial effusion on lung CT B and C: Increased right pleural effusion and diffuse ground-glass density areas in both lungs of COVID-19 pneumonia on lung CT during reinfection.

shortness of breath did not improve. His C-Reactive Protein (CRP) and D-Dimer were noted to be 79,4 mg/L respectively,(normal range <10 mg/L), 4000 ng/mL (normal range <550 ng/mL). CT angiography was performed to investigate pulmonary embolism as a potential cause, but was not found. However, due to the high D-Dimer, oral anticoagulants were added to the treatment regime. The RT-PCR test became negative on the 10th day. (Figure-1) A viral panel was studied (RT-PCR) from throat flora and no growth was detected. ANA was significantly positive (1/160) but other rheumatological markers and other possible causes of pericardial effusion (anti-Smith antibody (to rule out Lupus), hepatitis panel (to rule out hepatitis B), QuantiFERON-TB Gold test (to rule out tuberculosis) were negative. Echocardiography revealed an increase in parietal pericardial echogenicity and mild pericardial fluid. This was initially thought to be due to constrictive pericarditis. Methylprednisolone, colchicine, and ibuprofen were given. He received colchicine treatment for 3 months. Echocardiography control was done every 2 weeks. Meanwhile, the patient's clinical condition improved and his laboratory parameters normalized. There was an almost total resolution of pericardial and pleural effusion (Figure-2).

On October 12th, the patient represented with shortness of breath, chest pain, and fever. The patient's SARS-CoV-2 RT-PCR results swab result was positive again. Haematology lab showed WBC: 10*103/ml (normal range: 4.4–11.3 * 103/ml), with a low absolute lymphocyte count of 1.13 * 103/ml (normal range: 1.32–3.57*10^3/ml), lymphocyte percentage of 11.3% (normal range: and 20%–45%) and Hg: 12.1 g/ dl (normal range:14–17.5 g/dl). Coagulation studies revealed an elevated D-Dimer of 2480 ng/ml (normal range <550 ng/mL). CRP value was 68.2 mg/L (normal range <10 mg/L). A Lung CT revealed multifocal ground-glass density areas in both lungs, more prominent in the left lung. An increase in pleural and pericardial effusion was also observed. (Figure-2) Favipiravir, antibiotherapy (teicoplanin and azithromycin), methylprednisolone, and oxygen support were used again. On a repeat CT scan, the patient's COVID-19 pulmonary parenchymal involvement regressed. The clinical condition of the patient also improved.

Discussion

Our case report presents details of the first case of an individual in Turkey to have symptomatic reinfection with SARS-CoV-2. Similar to the observations in the reinfection case in Ecuador, our patient showed an increase in symptom severity in his second infection.[4] The mechanisms that may explain a more serious secondary infection are not known in literature. Firstly, a very high virus load may have caused a second infection and a more severe disease.[5] Secondly, re-infection within these patients can originate in the context of a more virulent or less virulent version of the virus. Thirdly, an antibody-dependent amplification caused by fc-specific immune cells, which were triggered by the virus binding to specific antibodies can be the causative factor. This mechanism has previously been seen with the betacoronavirus causing severe acute respiratory syndrome.[6]

Pericardial effusion within COVID-19 is a rare finding in pneumonia and is considered to be a poor prognostic factor in many publications.[7] The secondary origin of heart disease that may accompany this disease can cause pericardial effusion. However, COVID-19 substituted myopericarditis that can cause myocarditis or pericardial effusion. This involvement has rarely been reported in the literature and the actual incidence is unknown.[2] Also, signs such as mediastinal lymph node enlargement are rare, which are only seen in critical elderly patients and this may be related to underlying comorbidities usually present in the elderly patients.

Panayiotis G Vlachoyiannopoulos et al found that various systemic autoimmune reactivities in almost 70% of patients suggest infectious autoimmune activation after SARS-CoV-2 or para-SARS-CoV-2.[3] They argue that this is an expected result, as the cytokines involved in the cytokine storm, for example, interleukin-6, can trigger autoinflammatory reactions and autoimmunity, possibly through pre-existing native B cell clones or molecular mimics. In our case, only ANA positivity was detected among serological tests.

While the most common etiology of pericardial effusion is tuberculosis in developing countries, in developed countries like the US, the most common cause is idiopathic. In our case report, the presence of COVID-19 in pericardial fluid was determined using PCR. Other causes need to be excluded first, before putting Covid-19 as a cause. In our case, after excluding all rheumatological diseases, malignancy and tuberculosis, we thought that pericardial and pleural effusion, constrictive pericarditis findings could be secondary to COVID-19 infection.

Conclusion

Our case report demonstrates a possible case of COVID-19 reinfection with severe disease and that it is possible, though rare, of reinfection of SARS-CoV-2 and COVID-19 recurrence. Within COVID-19 patients with recurrence of symptoms, this possibility should be further investigated.

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Conflict of Interest: None.

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