Case Report

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TANAFFOS

A Severe Case of Bilateral COVID-19 Pneumonia with Concurrent Ischemic Stroke and Myocardial Infarction

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Looking at the recent data provided in literature, we can see an association between cardiovascular and cerebrovascular accidents in COVID-19 thought to be related to severe inflammation and prothrombotic environment caused by the virus. This article reports a patient presenting with typical signs and symptoms of SARS-CoV-2 infection including flu like symptoms and respiratory distress. Initially a chest CT was performed that showed characteristic findings of atypical pneumonia caused by SARS-CoV-2 virus which was later confirmed with a nasopharyngeal PCR positive for COVID-19. During the course of admission patient developed unstable angina. Further testing confirmed an acute ST elevation myocardial infarction. While on anticoagulant treatment, patient showed signs of cerebrovascular accident. An emergency brain CT was ordered which did not yield any significant changes supporting our clinical diagnosis. Further diagnostic workup using magnetic resonance imaging disclosed evidence of cerebral ischemia in medial cerebral artery territory. Our study suggests that prophylactic anticoagulant regiment is not reassuring in COVID-19 patients and close observation and vigilance, can help clinicians to act timely and can improve patient survival.

Key words: COVID-19; Acute Myocardial Infarction; Stroke; Multiple Infarcts

INTRODUCTION

Nowadays our entire world is struggling with an unprecedented challenge: Corona virus disease. As of October 2020 over one million deaths caused by COVID-19 are registered by World Health Organization (1). Symptoms include cough, fever, headache, fatigue, myalgia and dyspnea and may range from asymptomatic to severe multi-organ failure leading to death (2). With growing numbers of infected cases, increasing evidence of many non-respiratory manifestations are emerging (3). Studies suggest that thrombotic complications seem to be higher in patients with COVID-19. This may be due to cytokine storm and severe inflammation, hypoxia, endothelial dysfunction and immobilization (4, 5). In this category, acute coronary syndrome (6-10) and cerebrovascular accidents (11-14) are amongst the most serious and life threatening compilations of the disease. Interestingly enough, our report discusses a case of simultaneous myocardial infarction and ischemic stroke in a PCR confirmed SARS-CoV-2 pneumonia patient.

CASE SUMMARIES

A 54-year-old male with a prior history of hypertension and dyslipidemia presented to the emergency department (ED) complaining of fever and dyspnea. Reviewing the systems showed headache, myalgia and loss of appetite. Patient had close contact with a confirmed COVID-19 case 10 days earlier. Drug history included Atorvastatin 40 mg

daily and Amlodipine 10 mg daily. Therefore with a strong suspicion for the novel virus infection, patient was admitted in respiratory ward. Upon initial assessment in the ED, patient's vital signs were: hypertensive with blood pressure of 140/90 mmHg, fever of 38.5°C, tachycardia with HR of 112 beats per minute, tachypnea with respiratory rate of 30 per minute, and a saturation of 80% without assistive oxygen showing severe hypoxemia. Physical examination was unremarkable except for bilateral rales in lungs. According to the typical clinical picture, a chest CT was performed (Figure 1). The imaging revealed bilateral parenchymal ground glass opacities along with peripherally distributed consolidations which characterizes COVID-19 pneumonia. An electrocardiogram was performed showing no abnormal changes. CBC showed absolute lymphocyte count of 927 cells/mcl. Additional assessments revealed elevated CRP (80 mg/dl) and ESR (48 mm/h), high LDH (1148 units/l) and otherwise normal findings. PCR on nasopharyngeal swap specimen detected COVID-19.

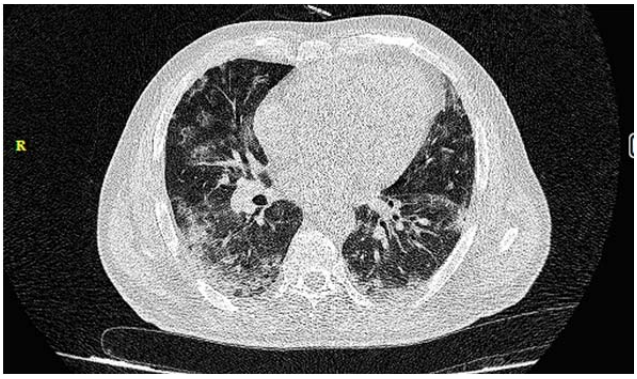

Figure 1. Chest CT showing peripheral dominant consolidations and parenchymal ground glass opacities consistent with COVID-19 pneumonia

We started treatment with the following regiment for COVID-19 including interferon 1-b 4.4 mcg subcutaneously, Remdesivir 200 mg on the first day followed by 100 mg daily for the rest of the treatment course, dexamethasone 8 mg daily, and enoxaparin 60 mg daily subcutaneously.

On the second day of admission patient developed a typical chest pain lasting 2 hours and reported cold sweats. An EKG was performed immediately. Significant changes including ST segment elevation from V1 to V4 and T wave inversion from V1 to V3 leads were found (Figure 2). Initial and follow-up Troponin I assessment were 5.4 pg/ml and 418 pg/ml respectively. Bedside echocardiography showed hypokinesia in anterolateral cardiac wall with an estimated LVEF of 40-45%. We started treatment for myocardial ischemia: Aspirin 300 mg stat and followed by 80 mg daily, Clopidogrel 300 mg stat and then 75 mg daily, Atorvastatin 80 mg every 12 hours and Heparin infusion 1000 u/h. Pethidine 25 mg and Nitroglycerin infusion was used to manage pain. PTT was checked 6 hours into the treatment, confirming a therapeutic range. Follow-up control of PTT levels indicated a range of 50-80 seconds throughout the treatment. Patient was closely observed using cardiac monitoring and pulse oximetry. In the upcoming days patient was hemodynamically stable, no arrhythmia occurred and chest pain was under control. Because of certain institutional limitations during the pandemic and respecting the patient's autonomy in choosing the treatment course from available options, reperfusion therapy was not performed.

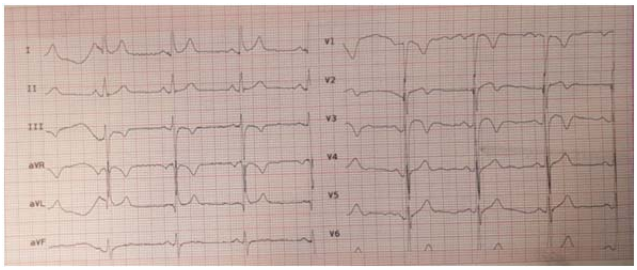

Figure 2. EKG showing ST segment changes with T wave inversion in precordial lead suggestive of an acute myocardial infarction

On the third day, the already complicated patient and the medical team faced a new challenging complication. Sudden dysarthria was the first sign of this process. In the next hour, patient developed right facial hemiparesis and severe weakness on the right side of the body. Upon examination, right sided facial hemiparesis and right sided hemiplegia affecting both upper and lower extremities were found. Babinski sign was positive on the right side. Sensory functions were normal and deep tendon reflexes showed no abnormality. Heparin infusion was

discontinued immediately and a brain CT was ordered. The imaging did not present any significant changes. According to patient's clinical picture, a brain MRI was the best choice for further investigation (Figure 3). MRI showed a high signal area in the right centrum semiovale in T2-flair sequences with restriction and ADC correlation concluded to be situated at the right MCA artery territory suggesting infarction. Taking into account that no evidence of hemorrhage was found in imaging studies, treatment with Heparin infusion, Aspirin and Clopidogrel was resumed. Although hemifacial paresis and dysarthria persisted, right sided motor dysfunction resolved after a few days.

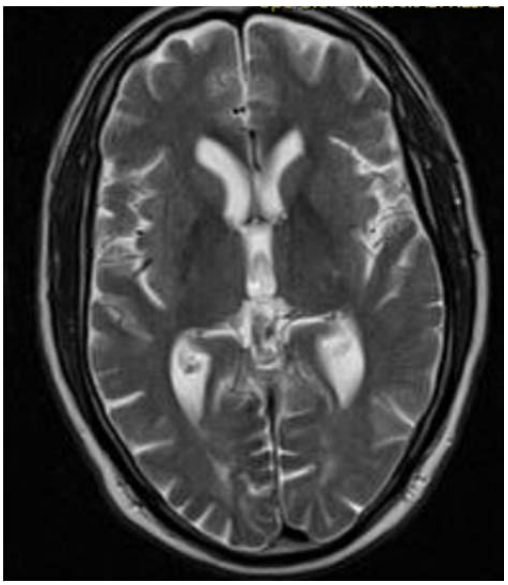

Figure 3. Brain MRI demonstrating ischemic changes in MCA territory

Patient's clinical condition improved after anti-COVID treatment with interferon, remdesivir, prophylactic antibodies and corticosteroids. O_2 saturation increased from 80% to 90%. Follow-up CT scan of the lungs showed partial resolution of parenchymal lesions (Figure 4). Patient did not experience any other episodes of cardiac or cerebral ischemic attacks, and no arrhythmia occurred during the time of admission. Considering his stable condition, we continued outpatient treatment for myocardial ischemia and the patient was referred to do an elective angiography after complete remission from COVID-19.

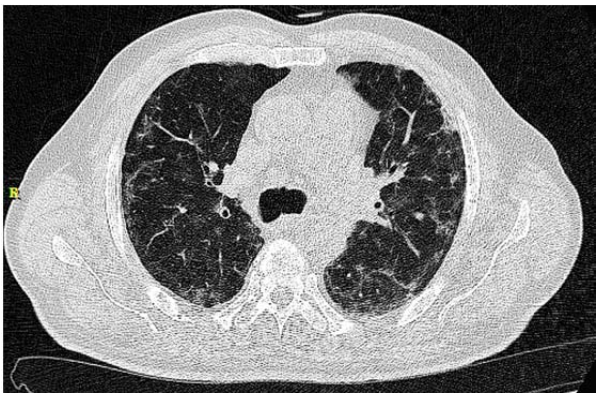

Figure 4. Follow-up chest CT showing partial resolution of lesions in both sides

DISCUSSION

SARS-CoV-2 virus can seemingly create a prothrombotic environment in the body predisposing COVID-19 patients to thromboembolic accidents (15). Concurrent cardiovascular conditions, and irregularities in blood count such as lymphopenia can predict a cerebrovascular event (both of which were present in our patient) (14), and in the setting of hypercoagulable state such as COVID-19 infection, lead to catastrophic outcomes. Although many cases of such patients are reported in literature (4-7, 9-11, 14-17), this report discuses an almost unique presentation of simultaneous ischemic events in two of the most vital organs. As for our patient, although prophylactic measures to prevent thrombosis were taken after MI, another life threatening thrombotic accident happened. Bringing this matter to light can be both very fascinating, and frightening but it seems that prophylactic anticoagulant use is not enough of a safety measure in COVID-19 patients. Of course this is just a hypothesis made by our observations and similar reports (14). Surely further investigations can be of much help to clarify the matter.

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