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Journal of Clinical Cardiology and Cardiovascular Interventions
Felicia Hasta *

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Case Report

A Patient Developing Both Myocardial Infarction and Stroke after COVID-19 Pneumonia

Felicia Hasta*, Mukul Anand, Laura Strong, Sarabjeet Singh-Chhabra
Central Cardiology Medical Clinic, Bakersfield, CA
***Corresponding Author:** Felicia Hasta, Central Cardiology Medical Clinic, Bakersfield, CA

Received Date: May 06, 2021; Accepted Date: May 17, 2021; Published Date: May 24, 2021
Citation: Felicia Hasta., Mukul Anand., Laura Strong., Sarabjeet Singh-Chhabra., (2021) A Patient Developing Both Myocardial Infarction and Stroke After COVID-19 Pneumonia. *J. Clinical Cardiology and Cardiovascular Interventions*, 4(11); **Doi:10.31579/2641-0419/181**

Abstract

We herein report an unusual case of a patient developing both cardiovascular and cerebrovascular sequelae to COVID-19 pneumonia. While COVID patients have been reported to experience one or the other, there has been little discussion of the presentation and pathophysiology of a patient presenting with injury to both organ systems. This article will consider the pathophysiology common to cardiovascular and cerebrovascular injury in the setting of COVID as well mechanisms that affect each system separately. It also discusses useful investigations which may assist in diagnosis and treatment of patients presenting as such.

Key words: covid; stroke; myocardial infarction; COVID-19; cardiovascular

Introduction

We herein report an unusual case of a patient developing both cardiovascular and cerebrovascular sequelae to COVID-19 pneumonia. While COVID patients have been reported to experience one or the other, there has been little discussion of the presentation and pathophysiology of a patient presenting with injury to both organ systems. This article will consider the pathophysiology common to cardiovascular and cerebrovascular injury in the setting of COVID as well mechanisms that affect each system separately. It also discusses useful investigations which may assist in diagnosis and treatment of patients presenting as such.

Case Presentation

The case presented is of a 67-year-old male who suffered a myocardial infarction (MI) and left-sided stroke following COVID-19 pneumonia. The patient has a history of lymphoma, hypertension (well-controlled by furosemide), and hyperlipidemia (controlled by diet and atorvastatin) but no prior history of coronary artery disease (CAD).

The patient was initially admitted from the emergency department (ED) due to fever, chills, and dyspnea upon exertion. He tested positive for COVID-19 and was treated with 1 unit convalescent COVID-19 plasma 2 days after admission. Lethargy and confusion increased progressively during this time. It became evident that the patient had an altered mental status secondary to acute metabolic encephalopathy. The patient required endotracheal intubation and mechanical ventilation due to worsening shortness of breath 5 days prior to admission. A chest computed tomography (CT) angiogram with contrast was performed which concluded in findings consistent with bilateral pneumonia. He was treated with intravenous (IV) remdesivir, antibiotics, dexamethasone, and acute coronary syndrome (ACS) protocol for troponin elevation.

The patient's chest pain and elevated troponins (1.87, normal is <0.03) required further investigation with transthoracic echocardiogram (ECHO). The procedure revealed an ejection fraction (EF) of 45-50% along with mild left ventricular (LV) systolic dysfunction and wall motion abnormality. While there was no other stenosis or regurgitation, findings of hemodynamic instability indicated a 2D ECHO on the 11th day of hospitalization. The procedure revealed LV diastolic dysfunction, tricuspid valve regurgitation, and mitral valve regurgitation. These findings were supported by an electrocardiogram (EKG) which indicated left atrial (LA) enlargement and left axis deviation. The patient later suffered complications of MI in the left anterior descending artery (LAD).

The patient also had imaging studies consistent with left-sided cerebrovascular accident (CVA).

The patient was discharged to home after a two month period of hospitalization. Follow up chest x-ray 35 days after discharge demonstrated bilateral interstitial scarring but no signs of pneumonia, pericardial effusion, or pleural effusion. Due to elevated troponins, outpatient workup was recommended to consider occlusive CAD as a differential once he achieved hemodynamic stability.

Discussion

While COVID-19 pneumonia has been linked to both neurological and cardiovascular complications individually, this patient notably experienced ischemic injury to both systems.

The association between COVID-19 and cerebrovascular events has been well documented.

A study of COVID-19 infected patients in New York reported that 0.9% of hospitalized COVID patients suffered ischemic stroke, with

cryptogenic strokes occurring twice as often in these patients than in non-COVID patients. [1] Further studies found that the COVID patients experiencing strokes and large vessel occlusion tended to be younger and with less cardiac comorbidities than non-COVID patients with the same neurovascular conditions. [1] The patient described in this study is a notable exception as he is of the average age of stroke patients and did suffer a cardiac comorbidity.

However, an analysis of data collected from December 2019-April 2020 found that 1.3% of COVID positive patients developed acute ischemic stroke, with many being older and with cardiac comorbidities as in this patient. Specifically, it reported that the risk of MI was higher in patients with COVID-related stroke than in COVID patients who did not suffer a stroke (10.7% vs. 4.6%, P=0.003). (2) This analysis found that COVID patients who developed acute ischemic stroke tended to be older, Black, and have been previously diagnosed with hypertension, diabetes, hyperlipidemia, atrial fibrillation, or congestive heart failure. [2] The patient reported here is 67 years old, Caucasian, and suffers from hyperlipidemia and hypertension, fitting many of the criteria put forth by this study. The same analyses also found that COVID patients who developed acute ischemic stroke also tended to suffer cardiovascular events during the same hospitalization. However, unlike this subject patient, the analyses showed a tendency toward multisystem involvement including acute kidney injury and hepatic failure. [2] This analysis concluded that while COVID-19 was a predisposing factor, the risk of acute ischemic stroke was mainly found in patients who were already at risk for stroke due to other medical risk factors.

Many mechanisms have been proposed to explain COVID-related cerebrovascular events, including hypercoagulability due to cytokine storm and direct viral-induced endotheliopathy. ACE2, a protein found on lung epithelial cells which breaks down pro-inflammatory factor angiotensin II, has been proposed as the entry site for the SARS-CoV-2 viral particle. [1] ACE2 is also expressed in cardiomyocytes and astrocytes, allowing for direct viral invasion of the heart and brain respectively. [3] The inhibition of ACE2 by viral particles may thus allow systemic inflammation and cytokine storm, resulting in dysfunction in multiple organ systems and blood vessels. These inflammatory changes to blood vessels result in activation of the coagulation cascade and subsequent elevation of inflammatory markers. One such unifying factor in affected patients is elevation of D-dimers, which was reported in more than 90% of COVID-associated stroke patients in New York. [1] Another such finding is elevations in inflammatory markers IL-6 and C-reactive protein (CRP) as a result of cytokine storm, a proposed mechanism of COVID-associated thrombosis associated with both MI and stroke in patients without a prior history. [1] This patient had elevated inflammatory markers, coagulation factors, and troponins which are summarized in the table below.

	Upon Admission	Nearing Discharge
Troponins (<0.03)	1.87	N/A
ESR (<13)	42	73
CRP (<1.00)	8.10	4.80
D-Dimers (0-230)	481	2,545
PT (9.5-12.6)	16.8	15.8
PTT (23.3-35.8)	31.6	24.6
INR (<4.00)	1.51	1.40
LDH (90-200)	596	143
Ferritin (11.0-307.0)	1,675.9	1,658.2

Table 1: *Lab results of the subject patient upon admission to and nearing discharge from the hospital*

Normal values are indicated in the parentheses next to each parameter. Troponins were elevated upon admission reflecting ongoing MI. Many inflammatory (ESR, CRP) and coagulation (D-Dimers, PT) markers were elevated upon admission and remained high at discharge, illustrating ongoing inflammatory consequences of COVID-19. Two coagulation markers, PTT and INR remained normal. Other markers of ongoing infection (LDH, ferritin) were also elevated upon admission.

There have been multiple reported manifestations of cardiac complications due to COVID-19, including myocarditis, MI, heart failure, arrhythmias, and thromboembolic events. Because such cardiac manifestations are common, measurement of cardiac enzymes such as troponins is recommended for all patients hospitalized with COVID-19 by the Chinese Clinical Guidance for COVID-19 Pneumonia Diagnosis and Treatment as well the World Health Organization (WHO). [4] Other organizations, such as the American College of Cardiology recommend only measuring these enzymes only if clinically indicated. The patient discussed here did in fact have elevated troponins in the ED, requiring follow up investigation with ECHO. [4]

Many COVID patients who raised strong clinical suspicion for MI (based on symptoms, elevated serum troponins, and ECG findings) were later

found to have acute myocarditis. ECHO allowed distinction between these two conditions, with findings of focal wall motion abnormalities in acute coronary syndromes (ACS) and either normal findings or global dysfunction in myocarditis. The ECHO performed on the patient in this study was consistent with ACS/MI as it demonstrated left ventricular wall and atrioventricular valve dysfunction. [4]

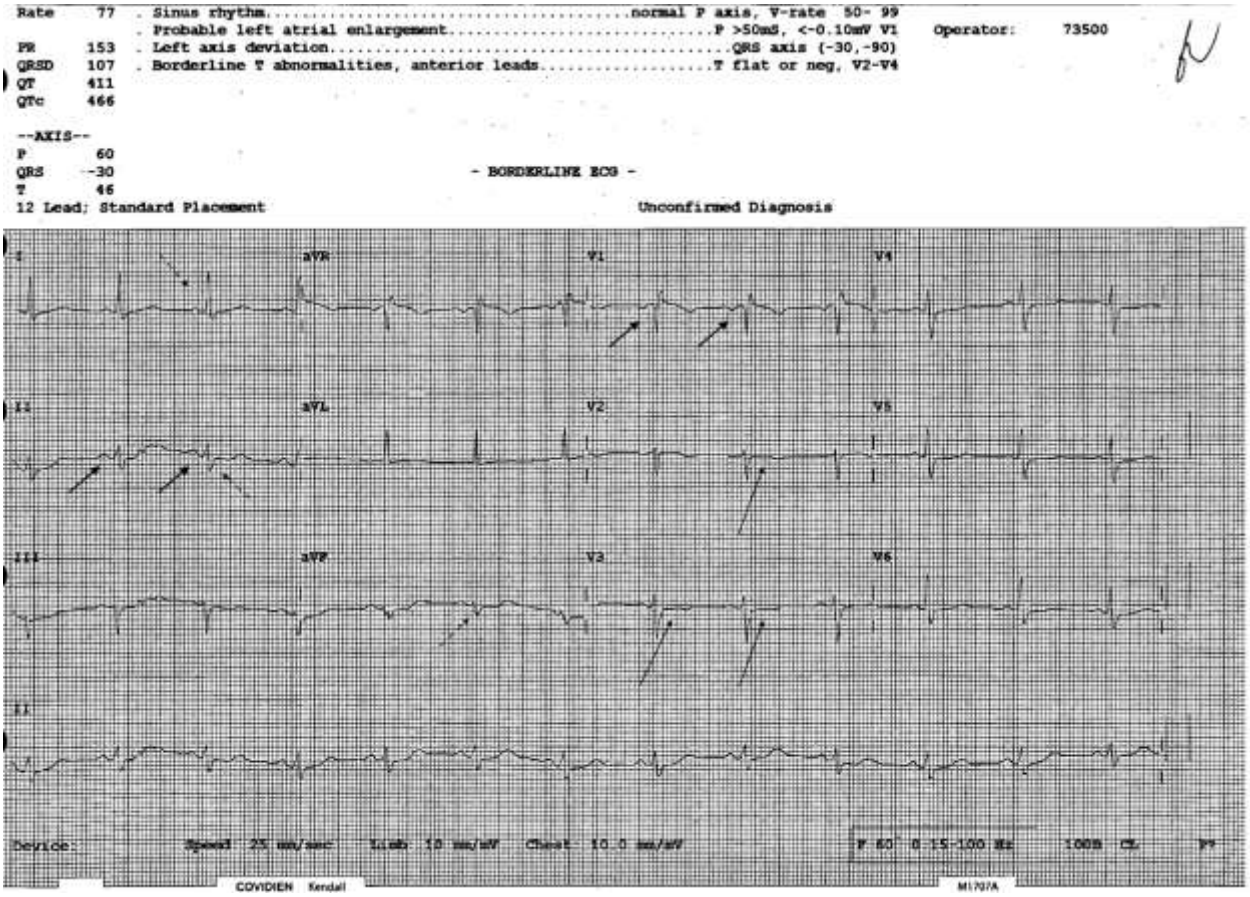

Figure 1: *The patient's EKG at presentation to the ED*

Interpretation is significant for probable LA enlargement (enlargement of P waves in leads II and V1, denoted by bold arrows) and left axis deviation (positive QRS in lead I and negative in leads II and avF, denoted by dashed arrows), which supports ECHO findings of mitral regurgitation and LV wall motion abnormality. Borderline T wave flattening/depression in the anterior leads (long arrows in leads V2 and V3) reflects ischemia in the areas supplied by the LAD.

The proposed mechanisms of cardiovascular injury due to COVID-19 are similar to those for COVID-related stroke: a combination of immunogenic hypercoagulability and direct viral-induced damage. However, more recent research also emphasized the likelihood of direct viral damage to cardiac tissue due to viral binding of surface ACE2 protein, as mentioned above. Autopsy samples from a Toronto study showed SARS-CoV viral RNA in 35% of the hearts of expired COVID patients. In the analysis that followed, it was found that every sample where viral RNA was detected showed signs of myocardial inflammation, suggesting the presence of the virus resulted in cardiomyopathy every time. [5]

These pathophysiologies, either individually or in combination, most commonly result in Type I or Type 2 MI as defined by the Fourth Universal Definition of MI. [7] A type 1, or spontaneous MI is "due to acute coronary atherothrombotic myocardial injury with either plaque rupture or erosion and, often, associated thrombosis."[8]. Plaque rupture or erosion in the setting of COVID can be achieved through either direct viral-induced endothelial dysfunction as defined above or indirectly through inflammation-induced hypercoagulability. [4] Type 2 MI, defined as an infarction resulting from a mismatch in oxygen supply and demand is also possible through a number of mechanisms. These include decreased perfusion from atherosclerosis, endothelial dysfunction and vasoconstriction from increased angiotensin II (a result of ACE inhibition), and hypoxemia from acute respiratory distress syndrome (ARDS) in COVID pneumonia. [7]

As discussed, three mechanisms, or "pathways" are common to cardiovascular and cerebrovascular injury in COVID patients. The first, described as the "hypoxia pathway" suggests that ARDS in COVID patients results in hypoxia in both myocardial and neuronal cells, resulting in ischemic injury to both organs. The second "RAAS (renin angiotensin aldosterone system) pathway" is a result of viral ACE2 inhibition, resulting in increased systemic angiotensin II and subsequent endothelial dysfunction in both the heart and brain. The third, or "immune pathway"

describes the effect of pneumonia-induced systemic inflammatory response (SIRS), resulting in proinflammatory cytokine storm which cause plaque instability and rupture in the heart and brain. [3]

Conclusion

Because of the common mechanisms in COVID injury to the heart and brain, it makes sense that a small number of patients, such as this one, experienced injury to both systems while hospitalized with COVID-19 pneumonia. Unfortunately, there is limited data from such cases readily available, and the few patients that have been reported ultimately died of their injuries [9]. This study is consequently limited as it cannot be compared to surviving patients with the same presentation. However, thorough review of the events and findings in the case presented here may have prognostic value in future cases of patients who present similarly. To assist in recognizing potential patients, it may be helpful to obtain an ECHO (to assess for wall motion abnormality and other acute findings), CT head (to assess for ischemic brain injury), and levels of troponins, inflammatory and coagulation markers. Another limitation is loss to follow up, as the patient did not complete scheduled outpatient appointments after hospital discharge. However, it was recommended the patient receive follow up CT head and ECHO to assess any long-term changes in the heart and brain.

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DOI:10.31579/2641-0419/181