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Case Report Open Access

Presentation of Cardioembolic Stroke in Covid-19

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Abstract

Corona virus of 2019 was first reported on December 2019, since then so many different manifestations, complications and prognosis have been reported and being studied. Spectrum of Cardiovascular complications is seen in a case of COVID-19, from a mild myocardial injury to a full-blown myocarditis. Severe disease is usually associated with a rise in cardiac biomarkers like B-type natriuretic peptides and cardiac troponin. Most common cardiac cause of mortality in COVID-19 patients is myocarditis resulting in circulatory collapse and death. This article presents a case of COVID-19 complicated with cardio-embolic stroke related myocarditis in a 60 years old male.

Keywords: COVID-1; Cardioembolic stroke; Myocarditis

Introduction

The novel SARS-COV-2 disease pandemic first incidence was recorded in China [1-4] and is still not under control in many countries worldwide. Most severe manifestation of COVID-19 includes acute respiratory distress syndrome, sepsis, cytokine storm, multi-organ failure, thromboembolic events and rarely extra-pulmonary manifestations [5]. Elevated D-dimer, deranged coagulation profile is most commonly present in severe cases. These patients are more prone for pulmonary embolism, deep vein thrombosis and cardio-embolic stroke due to hypercoagulability.

Background

The incidence of COVID-19 cases and the mortality rates are still on the rise. It is important to know and study about the different clinical presentation, progression and complication of COVID-19 cases. Knowledge of prompt and immediate treatment of such complications is important for reducing the mortality rates.

Case Report

A 60-year-old male, who is a known case of type-2 diabetic mellitus for 5 years and dyslipidemia for 5 years, was bought to the casualty with complaints of fever and malaise for 7 days, dry cough for 3 days and complaints of chest pain associated with palpitations. He also gave no history of difficulty in breathing. No history of ischemic heart disease in the past. No significant family and personal history. On examination, bilateral pedal oedema was present. His temperature was 102.2˚F, pulse rate of 130 beats/minute which was irregular, respiratory rate of 20 breaths/minute and blood pressure of 100/60 mmHg. His oxygen saturation was 96% in room air. Respiratory examination was normal, with bilateral air entry and no added sounds. Cardiac examination revealed no murmur. Electrocardiogram showed wide ORS complex, R-R interval was irregular, tachycardia with left bundle branch block was present suggestive of atrial fibrillation with rapid ventricular response or sinus tachycardia with premature atrial contraction. His complete blood count, urea and creatinine were within the normal range, mild electrolyte imbalance was noted, hyponatremia (serum sodium of 130 mEq/L) and hypokalemia (serum potassium of 2.6 mEq/L). Liver enzymes were elevated (SGOT and SGPT). Cardiac biomarkers were also raised, troponin T was positive and BNP was 610pg/ml. Chest radiograph showed consolidation in the lower lobe of the left lung.

Patient was loaded with amiodarone, sodium and potassium correction was done and started on ceftriaxone suspecting community acquired pneumonia. Repeat ECG showed sinus rhythm, but the LBBB and QT prolongation persisted. Respiratory viral panel and blood cultures were negative. Transthoracic echocardiogram showed reduced ejection fraction of 20%, left ventricular dilatation with hypokinesia. Cardiac magnetic resonance imagining was done which revealed left ventricular dilatation with global hypokinesia, cardiac oedema and hyperemia, suggestive of viral myocarditis. COVID-19 RT-PCR was positive and high-resolution computed tomography of chest done was suggestive of CORADS-4 with ground glass patches in the left lung. After 2 days, the patient developed aphasia with no other neurological symptoms. Magnetic resonance imagining and MR-angiography of the brain was done, showed embolic stroke involving the Broca's area affecting the left middle cerebral artery territory. He was treated with tissue plasminogen activator. Following treatment, the patient's conditions improved, vitals stabilized, he was discharged with thromboembolic prophylaxis medications and followed up regularly.

Discussion

The most probable cause of newly diagnosed left ventricular thrombus and patient's stroke was cardio-embolic. Incidence of myocarditis rates in between 1.4% to 7.2% in Covid-19. The gold standard for diagnosing intra-cardiac thrombi is trans-esophageal echocardiography. Recently, Cardiac MRI has been found to be more sensitive and specific in diagnosing intra-cardiac thrombi. Undiagnosed pre-existing atrial fibrillation might also cause increased the risk of patient developing LV thrombus, but return of normal sinus rhythm following electrolyte correction suggests that the atrial fibrillation theory to be unlikely. Presence of LV thrombus with dilated cardiomyopathy and reduced ejection fraction is most likely due to hyper-coagulable state which along with acute myocarditis increases the risk of LV thrombus formation and cardio-embolic stroke.

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Conclusion

This case report shows that, one of a serious complication of COVID-19 is myocarditis which may increase the risk of further myocardial injury like cardioembolic stroke. Cardiac changes in covid-19 are common but myocarditis is very rare. Though full spectrum of cardiac manifestation of COVID-19 is not completely studied, patients who are at a high risk of developing thromboembolism should be started on prophylactic anticoagulation therapy.

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