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Case Report

Acute ischemic stroke in COVID-19: a case report

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ABSTRACT

Corona virus disease (COVID-19) is an infectious disease caused by SARS-CoV-2 virus. It predominantly affects respiratory system causing fever, cough, and breathlessness. But it can also affect central nervous system. Clinicians must adopt methodical approach in investigating a patient with neurological manifestation. Early detection and treatment is required for neurological disease of COVID-19 to reduce post COVID neurological sequel.

Keywords: Stroke, COVID-19, D-dimer, CRP

INTRODUCTION

According to world health organization, a case definition for a suspected COVID-19 patient is the presence of fever, cough and shortness of breath as typical clinical symptoms. Neurological manifestations like headache, dizziness, anosmia, taste impairment, cerebrovascular disease, encephalopathy, myositis have been reported in COVID-19 patients along with the classical symptoms of fever and respiratory involvement.[1] A review by a panel of the World Stroke Organization reported that the risk of ischemic stroke during COVID-19 is around 5%.[2] Patients with COVID-19 who had strokes were more likely to be older and have hypertension and higher levels of D-dimer.[2] The median time from diagnosis to ischemic stroke in one small single-center study was 10 days.[3]Similarly, among 50 patients with ischemic stroke admitted in Wuhan, China, there was more comorbidity, lower platelet counts, lower leukocyte counts, and higher levels of D-dimers, cardiac troponin I, NT probrain natriuretic peptide, and interleukin-6.[4] Acute ischemic stroke (AIS) in brain is also emerging as an important neurovascular or neurological complication of COVID-19, associated with extreme immune responses leading to dysregulated coagulation system and generalized thrombo-embolic status and increased risk of acute ischemic stroke.[5] It is currently unclear to what extent macrovascular complications contribute to the mortality and morbidity of COVID-19.[6]

CASE REPORT

65 years old female presented to us with chief complaint of right sided weakness of upper limb and lower limb since 4 days, which was sudden in onset, progressive in nature and attained maximum weakness on the first day. She also has history of loss of speech since 4 days. Patient was able to comprehend and was not able to speak. No history suggestive of cranial nerve involvement. No history suggestive of sensory involvement and cerebellar involvement. No history of bowel and bladder disturbances. Not a known case of hypertensive, diabetic disease or cardiac disease. No past history of transient ischemic attack or stroke. Patient also gives history of fever since 3 days, intermittent in nature, high grade associated with chills and rigors, subsided by taking medication. Dry cough since 2 days. No history of breathlessness and chest pain. On examination, patient

was conscious, obeying to commands. Pulse rate was 110 beats per minute, blood pressure was 130/80mm Hg, respiratory rate was 28 breaths per minute. Temperature was 100°F. Oxygen saturation was 92 percent room air. CNS examination, patient had motor aphasia, hypotonia of right upper and lower limb. According to medical research council, power of right upper limb and lower limb was 2/5. Superficial reflexes were absent. Right plantar reflex was extensor. Left plantar reflex was flexor. Sensory system was normal. Cerebellar system and gait couldn't asses. Skull and spine was normal. No signs of meningeal irritation. Other systemic examination was normal.

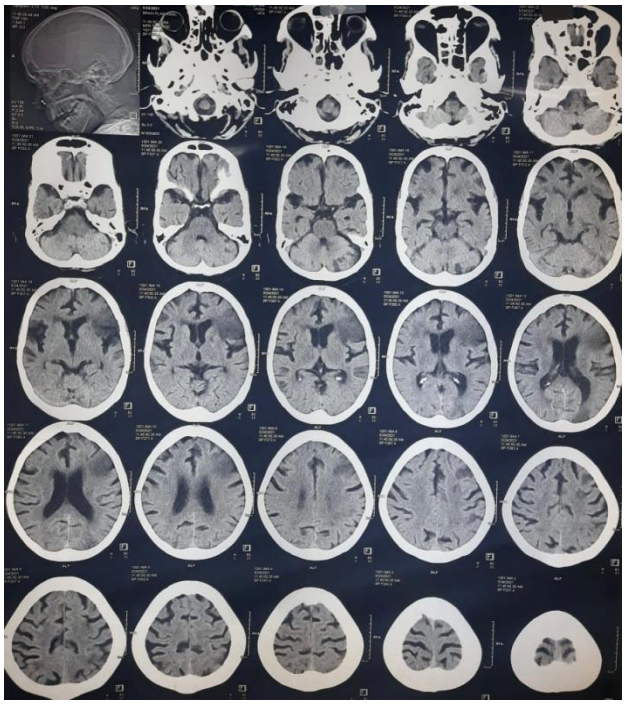

Figure 1: Plain CT brain showing Left infero-frontal and parieto-occipital infarct.

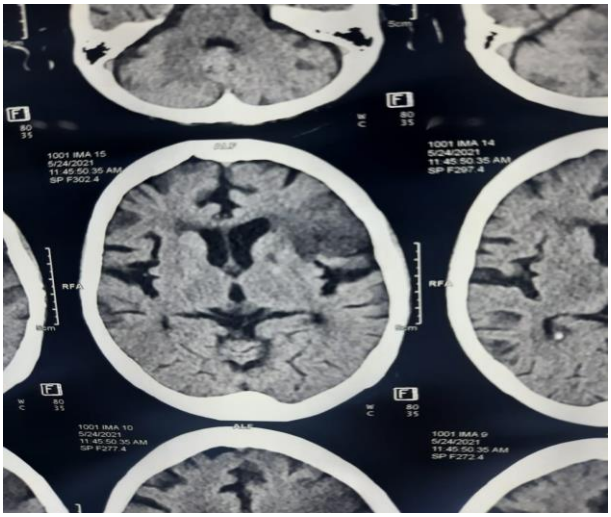

Figure 2: Plain CT brain showing left fronto-parietal infarct.

Arterial blood gas analysis was suggestive of type 1 respiratory failure. Blood investigations revealed haemoglobin 12.6 gm%, total count was 8060, neutrophils 79% lymphocytes 12%, NLR was 1.01, renal function test and liver function test was normal. Thyroid function test and lipid profile was normal. Glycosylated haemoglobin was 6.1%. Random blood sugar was 156 mg/dl. Serum LDH 127IU/l, d-dimer was 4.8, serum CRP was 27 mg/dl, serum ferritin was 39.7 µg/l, interleukin-6 was 0.18 pg/ml. ECG showed sinus tachycardia. Chest X-ray was suggestive of bilateral lower and mid zone infilterates, predominantly peripheries (Figure 3). RT-PCR for SARS-CoV-2 was positive. CT brain plain revealed left inferofrontal and parieto-occipital infarct (Figure 1-2).

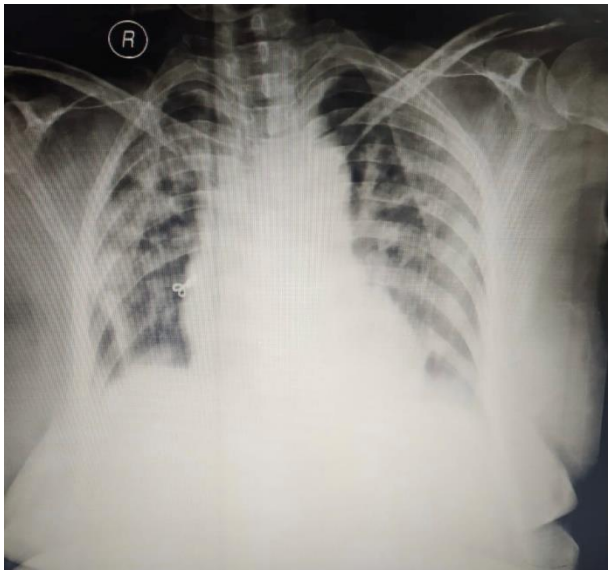

Figure 3: Chest X-ray PA view showing bilateral mid and lower zone infilterates predominantly involving peripheral fields.

Patient was diagnosed with right sided hemiparesis with left sided ischemic stroke with COVID-19 pneumonia. Patient was started on oxygen 4 litres per minute, injection mannitol 100 ml three times daily, injection ceftriaxone 1 gm i.v. two times a day, injection dexona 6 mg i.v. once a day, injection pantop 40 mg i.v. twice a day, injection enoxaparin 40 mg subcutaneous twice a day, tab ecospirin 150 mg once a day, tab atorvastatin 40mg once a day. Power of right sided upper and lower limb gradually improved over period of 10 days, room air saturation improved to 95%. Patient was discharged with clinical improvement.

DISCUSSION

COVID-19 is a current worldwide pandemic with multiple complications. Studies has shown multiple neurological manifestations including cerebrovascular accidents in patients with severe infection.[7] The current understanding on COVID-19 pathogenesis includes inflammation, endothelial dysfunction, and coagulopathy

appear to play critical roles in COVID-19-associated cerebrovascular disease (CVD).[8] Inflammatory processes of SARS-CoV-2 have fundamental roles in stroke in either the aetiology and pathophysiology of cerebral ischemia[9], the presence of COVID-19 infection could be a factor in the genesis or worsening of stroke in addition to the potential risk of cardio-embolic stroke due to ACE-2 expression in the heart and subsequent cardiac dysfunction.[10] Numerous studies have reported that recent respiratory infections are known to increase the short-term risk of ischemic stroke.[11-16] Higher rates of strokes have been observed in patients with coronavirus disease 2019 (COVID-19) and large-vessel stroke has been reported as a presenting feature of COVID-19 in the young.[17,18]

Even though respiratory symptoms predominantly seen in COVID-19 patients, our patient presented with weakness of right upper limb and lower limb as chief complaint. Her CRP and d-dimer was elevated. This indicates that SARS-CoV-2 has increased affinity towards the ACE2 receptors which are expressed in endothelium and arterial smooth muscle cells in the brain, resulting in inflame-matory process, endothelial dysfunction and prothrombotic state causing acute ischemic stroke in our patient. Final diagnosis in current investigation was acute ischemic stroke with COVID-19 pneumonia.

CONCLUSION

COVID-19 infection can be the independent risk factor for developing acute ischemic stroke due to its pro-thrombotic state. Those patients with neurological manifestations should be evaluated early and diagnostic work up and timely treatment should be done to prevent morbidity and mortality.

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