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CASE REPORT

Cerebro Vascular Accident In Sars-Cov-2 Patient: A Case Report

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Abstract

Severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) is a new strain of corona virus and we are still learning about various ways in which the disease presents, of which to be noted are uprising micro vascular and macro vascular complications which have found to worsen the disease outcome. Here we are presenting a case of SARS-CoV-2 positive patient who had developed Cerebro Vascular Accident (CVA) in the course of hospital which had further worsen the diseased and its outcome. Other than hypoxia induced altered sensorium, clinician must be aware of other possible reason of reduced consciousness in SARS-CoV-2 patients and to rework up diagnoses moreover if the clinical picture does not fully fit in to the patients presenting signs and symptoms. There is an Urgent need to Study the preventive aspect of stroke in SARS-CoV-2 as well as the management of stroke in SARS-CoV-2 patients which will help in preventing and treating CVA in the future.

1 | BACKGROUND

There is much to be learned from the new SARS-CoV-2 and since its novel very little is known about the diseases and various ways in which it presents, based on literature available so far particularly from various Intensive Care Units (ICU) worldwide, we are witnessing micro vascular complications as one of an emerging hypothesis to explain the multi organ failure in deteriorating patients. It is so far unclear that to what extent macro vascular complications contribute to the mortality and morbidity of SARS-CoV-2

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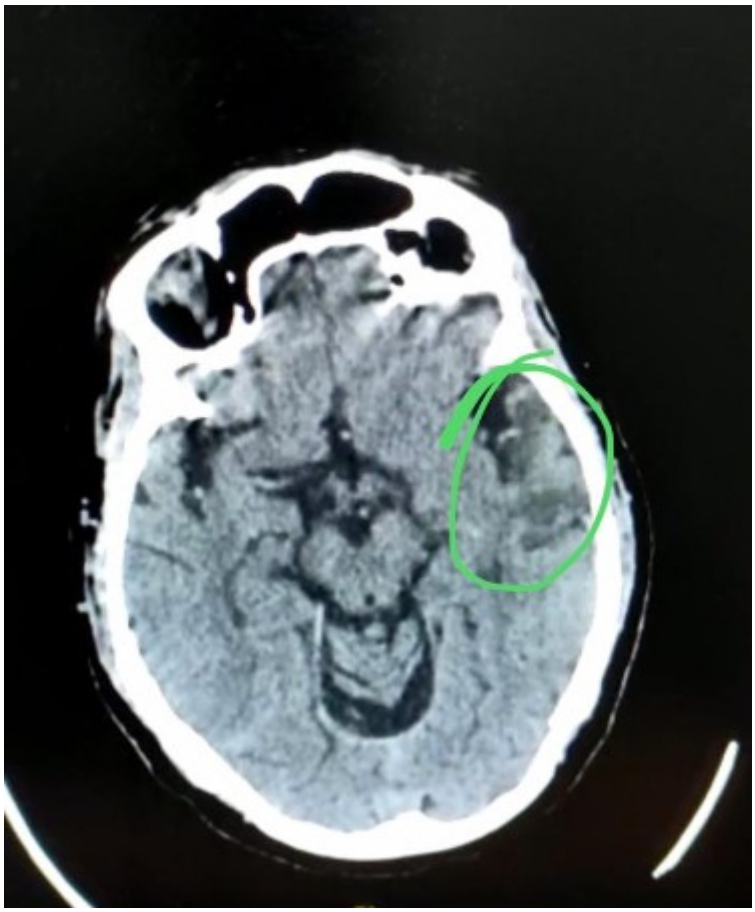

FIGURE 1: A slice of the CT images of the head demonstrating left middle cerebralartery infarction in temporal region

2 | CASE PRESENTATION

A 60 years old male presented to hospital with a three days of cough and shortness of breath. Patient visited health care profession on the second day of his symptom onset, he was advised to undergo test for SARS-CoV-2 due to his signs and symptoms, and patient gave his swab samples for SARS-CoV-2infection, after laboratory-confirmation by Reverse Transcription Polymerase Chain Reaction (RTPCR) patient immediately got admitted in the hospital for further management. Patient had an underlying co morbid condition of HyperTension(HT) for which patient was on regular medication. On examination patient was conscious, oriented, a febrile, patient was not anaemic, there was no icterus, no cyanosis, no lymphadenopathy and no generalised oedema, he was in respiratory distress as patient was tachypneic and had respiratory rate of 30 breaths/min,he was able to maintain only 85-87 % of oxygen saturation(spo_2) on room air. On chest auscultation, there were bilateral crackles but no wheeze. Cardiovascular examination was unremarkable with a regular pulse; his blood pressure reading was 130 on systole and 90 on diastole. Abdominal examination was also unremarkable and neurological examination at this stage was unremarkable .Patient was initially admitted in wards and was requiring 15 liters of oxygen through Non Re Breathing Mask (NRBM) to maintain oxygen saturation of 94% he was treated with antiviral (Remdesivir), steroid, anticoagulant, antihypertensive drugs and supportive measures. His respiratory status was persistently the same and with same oxygen requirement .On the third day of hospital stay his oxygen saturation started to drop from 94% to 90% he was given addition of 10 litres of oxygen through nasal prones with that he was able to maintain oxygen saturation of 94%. Antibiotics were started in view of secondary infection (total count was increased) . During the hospital stay his

B.P maintained in between 120-130 of systole and 70 to 80 of diastole with oral anti hypertensive medications (Amlodipine 2.5 mg once daily), On sixth day of hospital stay suddenly he developed an episode of involuntary movements followed by vomiting and his oxygen saturation started to drop further from 94% to 80% despite of maximum oxygen support by NRBM and nasal prons, he was immediately shifted to Intensive Care Units(ICU) and connected to Non-invasiveVentilation (NIV)support. An Arterial Blood Gas (ABG) analysis taken at that time showed type 1 respiratory failure which gave the impression of hypoactive delirium secondary to SARS-CoV-2 pneumonia. A neurological examination at the time showed that he was responding to pain, had a Glasgow Coma Scale (GCS) of 7 (M5V1E1), with equal tone, equal and reactive pupils and an equivocal right plantar reflex with an extensor left plantar reflex. The consultant considered worsening SARS-CoV-2 infection as a possibility but noted that the deterioration in conscious level was reasonably abrupt and urgent Computerized Tomography (CT) brain was taken which showed left MCA territory infarct with multiple hypo dense area involving the grey white matter interfaces of the left parietal lobe and left temporal lobe

3 | INVESTIGATIONS

His initial investigation involved routine blood investigation which included total count of 13500 / per micro litre, platelet 2,00,000 / per micro litre ,urea 45milligrams per decilitre ,creatine 83milligrams per decilitre ,c- reactive protein- 17.46 milligram per litre ,prolactine - 0.08 ng/ml ,ferritin-793.9 ng/ml and interlukin-6.23 pg/ml,sodium-128 mEq/L,potassium-4.9millimoles per liter,Bicarbonate 23 mEq/L, his total counts increased on third day to 16,846/ per micro litre ,CT Chest showed 25-50% of bilateral lower lobes and 10-25% of the rest of the individual lobes bilaterally involvement .echo showed ejection fraction of 55% with no clots and no aneurysm, CT brain was taken which showed left MCA territory infarct involving infarction multiple hypo dense area involving the grey white matter interfaces of the left parietal lobe and left temporal lobe

4 | DIFFERENTIAL DIAGNOSIS

Our differential diagnosis in the beginning was hypoactive delirium or delirium because of secondary metabolic decomposition caused by COVID-19 infection itself. But, the patient's blood investigation and blood markers were not suggestive of infection and patient had stable renal function, which made us differ from our initial differential diagnosis and made us investigate further in detail to search for any other possible reasons for his symptoms.

Apart from the initial differential diagnosis there were few other differential diagnoses which include non-convulsive status epilepticus, encephalitis due to SARS-CoV-2 infection or any massive intracerebral event. But was not able to come to any conclusion as the patient had reduced consciousness level and further neurological examination was also inconclusive because of patients reduces consciousness level. More over patient did not have any past history of epilepsy neither did patent underwent any imaging to suggest structural abnormality of brain which could have led to seizures. Finally we were able to clinch at the diagnosis only through brain imaging.

5 | TREATMENT

He was already on anti coagulants and later neurostimulants were added, considering patient's poor baseline function and the extent of the infarction of the left parietal lobe and left temporal lobe, it was from the opinion of ICU treating doctors that even if the patient survives there was no potential role for rehabilitation. Considering that, further investigation was not done to probe the aetiology of the stroke as it would not be of much benefit to the patient. He was offered supportive and palliative care.

6 | OUTCOME AND FOLLOW-UP

His GCS fluctuated on third day post CVA, In view of severe respiratory distress and low GCS, he was

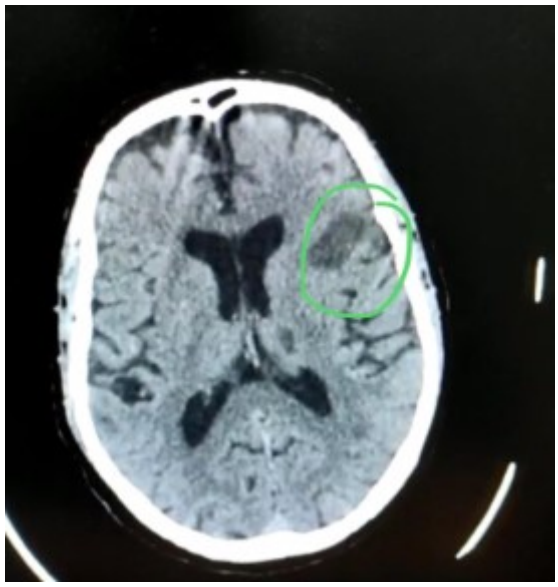

FIGURE 2: A further slice demonstrating infarction in the left parietal region.

incubated and connected to mechanical ventilator subsequently patient become thermodynamically unstable isotropic support was started despite of all supports and care patient developed cardiogenic shock and become asystole, even after Cardiopulmonary resuscitation patients was not able to revive and declared dead .

7 | DISCUSSION

SARS-CoV-2 is a new stain of corona virus which was first reported in Wuhan, China. At present SARS-CoV-2 disease has now spread world-wide and currently infecting more than 2 million people. [1] In the current scenario, we consider this case to be of interesting and may aid some evidence to the pathophysiologyof this novel SARS-CoV-2 disease,

Currently, there is a increasing interest on the various vascular complication caused by SARS-CoV-2 disease (1) .So far in autopsy of SARS-CoV-2 diseased corpse there are evidence of micro vascular injuries and those evidence postulate that micro vascular complications plays a vital role in severity of disease and has a important role in tohe disease outcome (2) . Macro vascular complication are also reported among the SARS-CoV-2 disease patients who become critically ill and need ICU care in hospitals it may be because of systemic inflammatory response of SARS-CoV-2 disease and further restriction of movements by SARS-CoV-2 disease patients in critical care units ,which along with tissue hypoxia has increased thromboembolism incidents and in some cases disseminated intravascular coagulation is also noted . [4] This events can result in both arterial as well as venous thromboembolism, causing increased morbidity in an critical ill and vulnerable SARS-CoV-2 disease patient .It is reported in a study that despite of thromboembolism prophylaxis given to SARS-CoV-2 disease patients admitted in ICU have developed pulmonary embolism, deep vein thrombosis, ischemic stroke, myocardial infarction and systemic arterial embolism. (3–5) (6)

In a another study it is reported that ,all the SARS-CoV-2 disease patients who developed ischemic stroke were found to have pre-existing risk factors for stroke and age >50 years had significant relationships between incidence of stroke among SARS-CoV-2 disease patient and severity of disease . Nevertheless, it is notable that there is significant overlap of risk factors between SARS-CoV-2 and ischemic stroke. [5, 7]In a various studies done in SARS-CoV-2 patients there is positive association found between cerebral vascular accidents and adverse outcome and prognosis among SARS-CoV-2 patients [8]

The disturbances of the normal coagulation pathway in SARS-CoV-2 patient may be linked vascular complication in severely ill SARS-CoV-2 patient

leading to mico vascular and macro vascular complication. (9) further raising the question of whether treatment dose anticoagulation should be considered for all critically ill SARS-CoV-2 patients, with pre-existing risk factors for thrombosis.

The diagnosis of this patient was difficult to arrive based on his clinical features alone, further timely diagnosis of any new vascular event is difficult in SARS-CoV-2 patients who require intubation and ventilation as patients are sedated which may cause hindrance neurological examination .in patients with SARS-CoV-2,it is important to have high suspicion for any intra cerebral event and to get imaging of the brain a early as possible ensuring that Cerebro vascular event is not missed

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