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A SEVERE CASE OF COVID-19 THAT DEVELOPED ENCEPHALITIS AND TREATED SUCCESFULLY WITH IV IMMUNOGLOBULINS

Medicine

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ABSTRACT

Neurologic manifestations of COVID-19 have attracted widespread attention. Though the respiratory system is the primary target of SARS-CoV-2, it is now increasingly recognised as a neuropathogen. Clinical features range from mild lethargy to deep coma and evidence of focal or diffuse neurologic signs and symptoms. The majority of patients with encephalitis are in critical condition. We report here a case of COVID-19-related encephalitis who developed persistent drowsiness 11 days after admission. The patient recovered well upon treatment with intravenous immunoglobulins (IVIg).

KEYWORDS

COVID-19, SARS-CoV-2, Encephalitis, IVIg, CECT.

INTRODUCTION

Coronavirus disease (COVID-19) was first recognised in December 2019 in China. Since then, it has spread rapidly to the rest of the world. The World Health Organization declared the outbreak as a Public Health Emergency of International Concern on 30th January 2020 and a pandemic on 11th March 2020. A typical COVID-19 case presentation can range from asymptomatic or mild to severe respiratory illness (acute respiratory distress syndrome).[1,2] As the pandemic progressed, a broad spectrum of clinicopathological entities are being reported, including and not limited to metabolic syndromes, electrolyte abnormalities, acute tubular necrosis, neurological syndromes, thromboembolic and cardiac events, including myocarditis and arrhythmias.[3,4,5] The first case of COVID-19 associated encephalopathy was reported in March 2020. With the evolution of pandemic, numerous neurological manifestations are observed by researchers and clinicians globally.

CASE PRESENTATION

A 63-year-old female with a medical history of hypertension presented to the Emergency Room of Medicover Hospital, Health City Visakhapatnam, on the 7th day of symptoms with fever and body aches for one week and shortness of breath for one day. She followed up with her primary care provider for the past week and received antipyretics and multivitamin supplementation until dyspnea developed. Her initial vital signs were: pulse rate 88 beats per minute, respiratory rate 25 breaths per minute, temperature 98.6°F, oxygen saturation 87% on room air, and blood pressure 126/70 mmHg. Examination of the respiratory system revealed bilateral diffuse coarse crackles. Cardiovascular system examination revealed normal first and second sounds with no murmurs or clicks. The abdomen was soft without any evidence of tenderness or organomegaly, and bowel sounds were present.

Investigations were done (**Table 1**). **HRCT Report:** Multiple diffuse ground-glass opacities. CT Severity Score 19/25 (Severe lung involvement).

The patient was admitted to the Critical Care Unit. Primary care was initiated, including oxygen supplementation via face mask according to her requirements as well as intravenous (IV) canula for fluids and IV drugs.

The following treatment plan was decided on and immediately started, IV methylprednisolone, IV remdesivir, and other supportive medications.

In the following days, there was a gradual increase in the patients' oxygen requirement, and she received High-Flow Nasal Oxygen.

On the 18th day of symptom onset, the patient became drowsy. There were no signs of meningeal irritation. CSF analysis (**Table 2**) was done.NCCT (**Figure1,2**) and CECT Brain (**Figure 3**) shows oedema in the bilateral occipital regions. Grey white matter differentiation is maintained. Cortical sulci appear effaced, involving bilateral occipital and temporal areas on post-contrast sequences; there is a mild heterogeneous enhancement of the sulcal spaces in the corresponding regions—features suggestive of FOCAL encephalitis.

We put the patient on IV immunoglobulins (IVIg) at a 2gm/kg body weight dose over five days, and improvement in the mental state and oxygen saturations was noticed over the next week. The patient discharged after 28 days of symptom onset.

Table 1. Laboratory investigations performed on day of admission

COMPLETE BLOOD COUNT	Haemoglobin 11.1 g/dL RBC 3.93 million cells/cumm WBC 8,700 cells/cumm Platelets 243,000/cumm Packed Cell Volume 34%
BLOOD GROUPING RH TYPING	O POSITIVE
CREATININE UREA	0.9 mg/dL 43 mg/dL
ELECTROLYTES	Sodium 137 mmol/L Potassium 4.3 mmol/L Chloride 104 mmol/L
RANDOM BLOOD SUGAR	296 mg/dL
LIVER FUNCTION TESTS	TOTAL BILIRUBIN 0.3 mg/dL DIRECT BILIRUBIN 0.1 mg/dL SGOT (AST) 55 U/L SGPT (ALT) 41 U/L ALKALINE PHOSPHATASE

	66 U/L TOTAL PROTEINS 6.3 g/dL ALBUMIN 3.0 g/dL GLOBULIN 3.3 g/dL A/G RATIO 0.9
NASOPHARYNGEAL SWAB	Rapid Antigen Test positive
C REACTIVE PROTEIN	176 mg/L
D-DIMER	583 ng/mL
LDH	496 IU/L
FERRITIN	925 ng/mL
HIV 1 & 2 (RAPID TEST)	NON REACTIVE
HBsAg	NON REACTIVE
HCV (RAPID TEST)	NON REACTIVE
PT-INR	1.1

Table 2. Cerebrospinal Fluid Analysis.

SPINAL FLUID	LAB VALUES
COLOUR	Colourless
WBC	4 cells/cumm
RBC	150 cells/cumm
GLUCOSE	124 mg/dL
PROTEIN	50 mg/dL
OPENING PRESSURE	18cm cmH_2O
GRAM STAIN	No organism is seen
ZN STAIN	No organism is seen
CULTURE	No growth

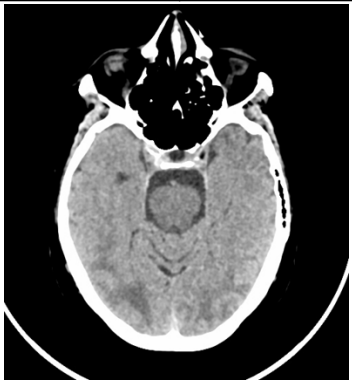

Figure 1.

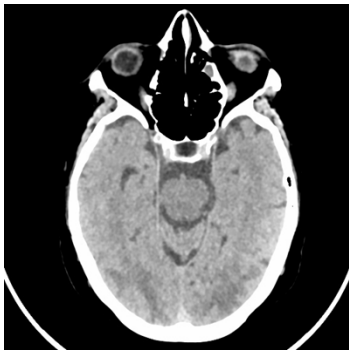

Figure 2.

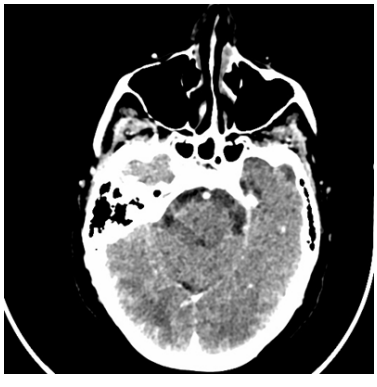

Figure 3.

DISCUSSION

Recently, the number of reported cases of COVID-19-associated encephalitis is increasing. Since SARS-CoV-2 affects the elderly and those with preexisting conditions more, patients with prior neurological disorders and severe acute respiratory symptoms are at an increased risk of encephalopathy on initial presentation. The neuroinvasive properties of SARS-CoV-2 are not well-understood due to a lack of experimental data. Proposed mechanisms through which the virus enters the CNS are:

- Retrograde dissemination via the olfactory epithelium and cribriform plate.
- Transfer from peripheral nerve terminals to the CNS via synapse connected routes.
- Damaged blood-brain barrier during the phase of viremia.[6]
- Neurological damage can occur via
- Neuro-inflammation caused by the cytokine storm.
- Hypoxia caused by severe pneumonia and acute respiratory distress syndrome (ARDS).[7]

Diagnosis of COVID-19 encephalitis can be highly challenging and requires a high index of suspicion. SARS-CoV-2 dissemination is transient, and its CSF titer may be extremely low.[8,9] EEG and MRI showed nonspecific findings.[9,10,11]Treatment is mainly supportive. Various treatment modalities, including IVIg, high-dose IV steroids, and immunomodulators, have been tried in multiple cases, with mixed outcomes.[9,12] In the present case, early recognition of mental deterioration with prompt initiation of IVIg could be the reason for improvement. However, a larger-scale study may be needed to verify this claim. The absence of CSF PCR analysis is a limitation in this case report.

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