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Original Research Paper

Psychiatry

BIFRONTAL DYSFUNCTION IN COVID-19 INFECTION

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ABSTRACT Acute psychotic disturbances can occur in Covid-19 infection, due to either direct viral infection or inflammatory processes with dysregulation of the immune response. A 34-year-old single male, an auto driver, presented with decreased sleep for 2.5 months, suspiciousness and fearfulness for the past 2 months and a suicide attempt. He had Covid 19 infection 2 weeks prior to behavioral changes. On examination patient was conscious, disoriented to time and place. Talk was decreased, agitated and anxious mood. He had persecutory and nihilistic delusions and olfactory hallucination. He was hypobulic with generalised rigidity. He had bilateral asymmetrical parkinsonian signs and brisk deep tendon reflex. EEG showed generalized slowing activity in frontal regions. CSF study revealed raised protein level. He was managed with IV Methylprednisolone, IV and oral Thiamine, Lorazepam, Haloperidol and Nitrazepam. At discharge, symptoms improved by 50%. At 1 year follow-up, patient reached premorbid level without any medications.

KEYWORDS : Bifrontal dysfunction, COVID 19, Neurotropism

INTRODUCTION

While SARS-CoV-2 is known to cause interstitial pneumonia and acute respiratory distress syndrome (ARDS), there is increasing evidence of diverse neurological manifestations, including encephalopathy, limbic and brainstem encephalitis, stroke, and Guillain–Barré syndrome[1]. Delirium, olfactory disturbances, acute behavioral changes, headache, and cerebrovascular accidents are also common neuro psychiatric complications of SARS CoV-2[2]. These presentations may reflect either direct viral infection or, more likely, inflammatory processes with dysregulation of the immune response[3]. The systemic hyperinflammatory state may be involved in neurologic impairment, as well.

Case Report

34-year-old single male, an auto driver, staying with parents and brother presented to ER with complaints of decreased sleep for 2-1/2 months, suspiciousness, and fearfulness for the past 60 days. 2 weeks after being tested to be covid positive, Patient began expressing apprehension regarding the post covid health issues. He was worried and was not going for work even after completing his isolation period. Patient complained to his parents that his right eye was rotten and the vision on that side was lost. There was history suggestive of increased activities manifested as walking around the house. Sleep was disturbed and appetite was reduced.

Patient tried ISH by hanging on Feb 1st week and was taken to a Govt. Medical College. There he was consulted by the Psychiatry department and advised IP management, which was refused by the patient and family members and agreed for treatment on OP basis. He was advised Tab. Olanzapine 10mg HS, Tab Nitrazepam 10mg HS. Ophthalmology consultation was done in view of diminished vision, and no active intervention was advised. Patient didn't take the drugs prescribed by Psychiatry department.

Three days following this incident, patient gradually developed suspiciousness and fearfulness. Patient had the suspicion that his Auto rickshaw will be seized by the finance company as he was not repaying the loan. Father told the patient that repayment was over 3 months back. But he was not convinced about that, and he tried to hide the auto in backyard of his house and covered it with straw and tarpaulin. Patient had interpersonal issues with his brother who was using alcohol daily. One day before the admission to our hospital, the elder brother threatened to burn the patient after pouring petrol over him. After that patient became very fearful and was crying out loudly, asking for help. According to father patient was behaving in such a way that he was not aware of his surroundings or time. But he could identify his family members. Next day he told his father that he had the smell of petrol over his body as someone had poured it on his body. After some time, he asked his parents to bury him as he was dead and he was irritable towards them, which brought him to ER. No history of any medical or surgical comorbidities.

Family history of psychiatric illness in second degree relative, substance use disorder in 1st and 2nd degree relatives.

On **general examination**, he was moderately built and nourished, BP-130/80mmHg, PR-92/min, RR-18/min, Conjunctival congestion +, GRBS-118mg/dl.

On MSE, patient was conscious, not oriented to time and place.

Talk was decreased, psychomotor activity increased. Mood was anxious.

Patient had persecutory and nihilistic delusion, and olfactory hallucination of smell of petrol over his body.

Course in hospital -

Patient was admitted under psychiatry department and was started on antipsychotic, T. Haloperidol 5mg BD, T. Nitrazepam 5mg HS and T. Trihexyphenidyl 2mg 1-0-0.

On 2nd day patient was disoriented, hypobulic with generalised rigidity, bilateral asymmetrical parkinsonian signs and brisk deep tendon reflex. No meningeal irritations or focal signs were found.

Neuro-medicine consultation was sought. Patient was transferred to Neuro-medicine department in view of suspected **post covid encephalitis/acute disseminated encephalitis** on next day of admission.

CE-MRI Brain demonstrated no significant intracranial abnormality. EEG recording showed a generalized slowing activity, dominant in frontal regions.

CSF study revealed raised protein level. Other CSF studies were negative. He was managed with IV Methylprednisolone, IV Thiamine and IV Lorazepam, followed by low dose Haloperidol and Nitrazepam. Serial EEGs showed gradual

reduction of previous slow activity.

Patient was discharged with 50 % improvement in symptoms. At the time of discharge patient was on T. Quetiapine 50mg 1-0-1, T. Levodopa Plus Carbidopa 125mg ½ 1-1-1 and T. Clonazepam 0.5mg 1-0-1.

1 month follow-up–

Following discharge patient continued to be mute most of the time, spoke only few words like "I am hungry" "wants to sit in Veranda". Patient paced about during the day, stared at everyone, and appeared to be fearful. Patient needed assistance for self-care also. Sleep was adequate. On examination patient had decreased talk and psychomotor activity. Responded to questions in very low voice after much prompting- Hypophonic dysarthria. T. Quetiapine was hiked to 50mg-0-100mg, and rest were continued.

6-month follow-up–

Symptoms were improving. Biological functions were adequate. On examination talk had improved, psychomotor activity was normal. No tremors/rigidity. Self-care was adequate. Patient was advised to go for his work. Dose of drugs was tapered and stopped in 3 months.

1 year follow-up–

Patient reached premorbid level according to family members and was going for his work.

DISCUSSION

Several literature reports have already shown the SARS-CoV-2 neurotropism, although the etiopathology of neuronal damage is not entirely clear[4].

Predominant frontal symptoms with anterior slowing EEG findings and bilateral frontotemporal hypoperfusion have been similarly described in other cases of COVID-19-related encephalopathy[5]. Frontal syndrome may thus be considered the predominant feature of COVID-19-related acute encephalopathy[5]. Moreover, cross-checked serum and CSF cytokines levels may represent a useful biomarker of early detection and progression of the neurologic involvement, as well as a ground for therapeutic recommendations and prognosis predictions.

COVID-19 patients under intensive care showed signs of delirium with confusion (65%), agitations (69%), and altered consciousness (21%), while 33% showed dysexecutive syndrome at discharge[6]. Psychiatric evaluation of patients may be necessary during and beyond hospitalization. Therefore, patients affected by severe COVID-19 need a multidisciplinary team to address it.

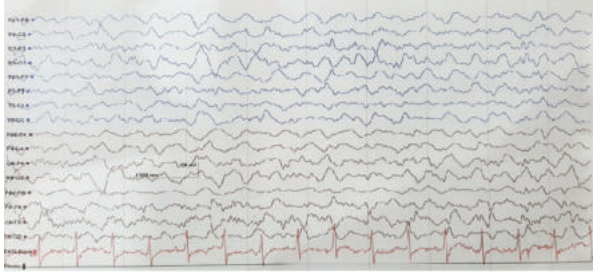

Fig 1: EEG alterations observed at time of first neurologic evaluation with prominent and bilateral frontal slowing.

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