Case Report

HYPOMANIC EPISODE FOLLOWING COVID-19 PNEUMONIA – CASE REPORT

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ABSTRACT

Coronavirus disease-2019 (COVID-19), an acute respiratory infection caused by severe acute respiratory distress syndrome-coronavirus-2 (SARS-CoV-2), was declared a pandemic in early 2020. Although the virus primarily affects respiratory and gastrointestinal systems, it is neurotropic and has neuropsychiatric complications. The most common neuropsychiatric manifestations seen in COVID-19 infection include delirium, depression, anxiety and post-traumatic stress disorder; very few cases of mania have been reported. Here, we report two cases –an elderly male and a middle-aged female – without a past history of psychiatric illness, presenting with hypomanic episodes following COVID-19 infection. Their blood investigations were normal, and neuroimaging revealed chronic and age-related changes only. As they showed a good response to mood stabilizer, further evaluation including the CSF study was deferred. Psychiatric symptoms could have been the outcome of an interaction of the psychosocial stressors associated with the pandemic on the brain, which was made vulnerable by the COVID-19 infection.

Keywords: hypomania, COVID-19, pneumonia

INTRODUCTION

Coronavirus disease-2019 (COVID-19) caused by severe acute respiratory distress syndrome-coronavirus-2 (SARS-CoV-2) was declared as a pandemic by the World Health Organization on March 11, 2020.[1] SARS-CoV-2 attacks respiratory and gastrointestinal systems by attaching to angiotensin-converting enzyme-2 (ACE-2) receptors but has been recognized to be neurotropic and produce neuropsychiatric manifestations.[2] The neuropsychiatric symptoms reported in COVID-19 patients include acute delirium, persistent neurocognitive disorders, mood disorders – like depression, anxiety disorders, obsessive compulsive disorders, post-traumatic stress disorder (PTSD), and rarely psychotic disorders.[3] Very few cases of mania have been reported in the context of COVID-19 infection.[4,5] Here, we report two cases who developed hypomanic symptoms following COVID-19 pneumonia.

Case 1

Mr. R, a 68-year-old, eighth standard educated, married male, presented to Psychiatry OPD with behavioural problems of three weeks duration. Almost one month back, he was investigated for cough with expectoration of one week duration. Rapid Antigen Test (RAT) for COVID-19 was positive, and he was diagnosed with

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COVID-19 pneumonia. As his SpO2 level was 88% without oxygen, he was admitted to the intensive care unit (ICU) at the local hospital and maintained on non-invasive ventilator for four days. He was managed with intravenous antibiotics, favipiravir, steroids (dexamethasone 12 mg for five days, then tapered to 6 mg for two more days and then stopped), low molecular weight heparin and supportive measures. He had elevated total count and hyponatremia at admission, which became normal within the next two days. There were bilateral lower lobe opacities on chest X-ray, and a CT scan of thorax was not done. After ten days, RAT became negative, but he was managed in the ward for five more days for breathlessness before discharge. During hospital stay, no behavioural or cognitive abnormalities were observed. After discharge, he was not on any medicines and was functioning well for about one week.

After this, his relatives observed a gradual change in his behaviour. He was found to wake up early in the morning but was not tired during the daytime and would engage in his self-care activities at a faster pace than usual. Unlike his usual self, he would repeatedly go to the courtyard, face the direction of a nearby temple, chant prayers, and light the lamp in the pooja room more frequently. He would show overfamiliarity, talk loudly to others, and claim that his prayers would bring blessings. No pervasive depressive symptoms or psychotic symptoms were observed at any point of time. He was not on any medications when he presented to Psychiatry OPD. There was no past or family history of any psychiatric illness, including substance use disorder. There were no other medical or surgical comorbidities. His premorbid personality was well-adjusted.

On examination, he was fully conscious and in touch with reality. Psychomotor activity was increased; talk was spontaneous, increased in quantum with decreased reaction time. His mood was elated, and he had inflated self-esteem. No depressive cognition, delusions or perceptual abnormalities were observed. On assessment of cognitive function, he was oriented to time, place and person; his attention and concentration were impaired, and insight was poor. Mini Mental State Examination score was 28. Physical examination findings were within normal limits. MRI Brain showed small vessel ischaemic disease and age-related atrophic changes. EEG was normal. As he had persistently elevated mood, overactivity, inflated self-esteem, and hyper-religiosity, but there were no psychotic symptoms or severe disruption of his social activities, a hypomanic episode was diagnosed. Since there were no psychotic symptoms, he was started on T. Sodium Valproate 200 mg bid, to which he showed a good response. Within two weeks, he reached premorbid levels.

Case 2

Mrs. S, a 53-year-old, third standard educated, married female working as domestic help, presented with recent onset of behavioural changes following her return from the covid treatment centre. She was admitted to a private hospital with symptoms of fever, cough and breathlessness after being tested positive for COVID-19. For five days, she was treated with multivitamin tablets, levocetirizine and montelukast, along with the antihypertensive nebivolol (which she was taking earlier). She had not received any other medications, including steroids. Her blood investigations during covid infection were not available as she was not treated at our hospital during that time. After one week, on testing negative, she was discharged, following which she had gradually worsening behavioural symptoms. She had disturbed sleep and early morning awakenings. She was taking bath during the early hours of the morning, doing her chores earlier than usual and working throughout the day. Unlike her usual self, she was overtalkative, talking loudly and excessively over the phone, often instructing others how to protect themselves from the virus. She also began expressing wishes and plans of buying ornaments. After two weeks of the onset of behavioural symptoms, as her sleep impairment persisted and she was becoming irritable, she was brought to the Psychiatry OPD.

There was no past history of psychiatric illness, but two of her first-degree relatives had a history suggestive of mood disorder, one of whom had committed suicide. Her premorbid personality was well adjusted. On examination, she was conscious and cooperative. Her psychomotor activity was increased, the speech was increased in amount, tone and tempo, with decreased reaction time. She had a euphoric mood and inflated self-esteem. No depressive symptoms or psychotic features were noted. She was oriented to time, place and person; her attention was impaired, but memory intact.

Her Young Mania Rating Scale (YMRS) score was 21. MRI Brain showed chronic infarcts involving the left posterior watershed area, left frontal and parietal lobe, and age-related atrophy. EEG was normal. As there were no psychotic symptoms and her social functioning was not disrupted, a diagnosis of hypomanic episode was made. She was started on Tab. Sodium valproate 200 mg bid and Tab. Lorazepam 2mg HS. CSF study was deferred in the absence of focal neurological deficits and as the patient showed rapid improvement. She was started on Tab. aspirin 75 mg OD by the neurologist. After one week, she was symptomatically better with a YMRS score of 14.

Blood investigations like random blood sugar, liver function tests, renal function tests, sodium, potassium, calcium, phosphorous, thyroid function tests, uric acid, complete blood count, haemoglobin and ESR were normal for both cases.

DISCUSSION

Although the β-coronaviruses, like SARS-CoV-2, primarily invade the respiratory and gastrointestinal systems, it is found to affect the CNS. The route of entry could be through the ACE-2 receptors in the brain, directly through the olfactory bulb. Inflammatory responses, endotheliopathy, breakdown of blood-brain barrier, clotting abnormalities and autoimmunity could contribute to CNS dysfunction and neuropsychiatric complications.[6] Medications like hydroxychloroquine and steroids could also produce such manifestations.[7] Neuropsychiatric sequelae of coronavirus infection have been proposed to be multifactorial in origin – including the direct effect of viral infection on the brain; cerebrovascular disease in the context of a procoagulant state; physiological compromises as with hypoxia or immunological response; the impact of medical management; and psychological factors like social isolation, impact of a potentially fatal illness, fear of infecting others and the stigma associated with the illness.[8]

The first case report of manic-like symptoms in a 51-year-old COVID-19 patient with no past history of psychiatric illness came from China. After clinical improvement, while methylprednisolone was being tapered, the patient developed symptoms of mania. Although the CSF RT-PCR test was negative, the SARS-CoV-2 IgG antibody was positive in CSF. The patient showed rapid improvement with olanzapine.[4] Another case of cannabis-induced mania was reported in a man with no past history of mental illness after self-medication for COVID-19 with homemade remedies containing cannabis.[9] An early-onset first episode of mania was reported in an adolescent boy with no family history of psychiatric illness during the COVID-19 lockdown period – which was perceived as extremely oppressive. All investigations, including neuroimaging, were normal, and he responded well to medications.[10]

Here an elderly male with no past or family history of psychiatric disorders and another middle-aged female with a family history suggestive of mood disorder presented with features suggestive of hypomania in the aftermath of COVID-19 pneumonia. Although organicity was considered in the context of COVID-19 infection, investigation results, including neuroimaging, did not support that. Neurology consultation was done for further evaluation, but CSF study was deferred as both patients improved clinically and did not warrant an invasive procedure. In the second case, although neuroimaging revealed chronic infarcts and age-related atrophic changes, that was not considered in the aetiology of the hypomanic episode as there was no temporal correlation with the onset of mood symptoms. In both these cases, evidence of direct viral infection was not available, but CSF study was deferred. Patients were not on any medications which could cause mood elevation. Psychosocial factors like the social isolation experienced during admission to the hospital or ICU, fear of infecting others and the stigma associated with COVID-19 infection might have contributed to the development of mood symptoms. However, these stressors were not evaluated using a validated questionnaire. These factors might have operated on the brain made vulnerable by COVID-19 infection – especially in the second case with a diathesis for mood disorder – in precipitating the symptoms.

CONCLUSION

We report two cases – one of an older adult and another of a middle-aged woman – without a past history of psychiatric illness, who presented with hypomania in the convalescent phase of COVID-19 pneumonia and responded well to treatment with mood stabilizer. Interaction of psychosocial stressors and a brain vulnerability related to covid-19 infection could have precipitated the mood disorder in both these cases.

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