Seizure after recovery from Covid-19

Sidhartha Chattopadhyay¹, Sagar Basu¹, Kamalesh Majumdar²

Departments of ¹Neuro-Medicine and ²Radiology, KPC Medical College and Hospital, Kolkata, West Bengal, India

ABSTRACT

There are various neurological presentations of covid-19 but here we report a 27-year-old female developed first episode of seizure, 14 days after documented SARS-CoV-2 infection. She had generalized tonic-clonic seizure with multiple injuries. She had no previous or family history of seizure but she had generalized anxiety disorder and was doing well on selective serotonin reuptake inhibitor (SSRI). Electroencephalogram (EEG) showed theta waves background (likely post-ictal discharges). Magnetic resonance imagining (MRI) Brain showed features of focal cerebritis on both the frontal lobes. She was started with anti-epileptic drug. After 6 weeks, repeat MRI Brain and EEG were completely normal.

Keywords: Epilepsy, focal cerebritis, generalized anxiety disorder, SARS-CoV-2, seizure, recovery

Background

The primary and paramount presentation of covid-19 is usually associated with respiratory involvement but reports of neurological manifestations are also increasing.¹ The various neurological features of Covid-19 are viral meningitis, encephalopathy, anosmia, ageusia, optic neuritis, peripheral neuropathy (GBS and other variants), seizure, ataxia, cerebro-vascular diseases, and myositis.² Epilepsy during illness by covid-19 has been recently documented.³⁴ But here we present an unusual and probably, first time in eastern India, case of a 27-year-old unmarried female developed first episode of seizure, 14 days after documented SARS-CoV-2 infection. This type of epileptic seizure after recovery from SARS-CoV-2 infection possibly has not been described in any literature earlier to the best of our knowledge.

Case Presentation

A 27-year-old unmarried female with premorbid history of generalized anxiety disorder (GAD) presented to us with first the episode of generalized tonic-clonic seizure on the previous day. About one week prior to ictus, she was discharged from another hospital with the diagnosis of SARS-CoV-2 infection. According to the eye witness, she had a sudden up-rolling of eyes along with tonic-clonic movements of all limbs followed by lateral tongue bite and fall with injuries of forehead and black eye (left side). The seizure lasted for about 2 minutes. After convulsion, she had post-ictal confusion and lethargy which persisted for about an hour. She was continuing SSRI (Escitalopram 10 mg) for her anxiety disorder. There was no family history of seizure, stress, sleep deprivation, and substance abuse. It was also not associated with fever, headache, neck stiffness, photophobia, nausea, and vomiting. No history of previous traumatic head injury, paresis, visual impairment, speech impairment, and involuntary movements. Her menstrual cycle was normal and regular. She had no additional systemic disease. She received Favipiravir (11600 mg), during her SARS-CoV-2 infection. She was hemodynamically stable with normal sensorium. Neurological and other clinical examinations were normal except blackeye on left side. The cause of the seizure was undetermined at that moment.

Her laboratory analysis includes Hb-10.2 g/dL (n = 12–15.0 g/dL), TLC-8700/cu.mm (n = 4000–11000/cu.mm), ESR-16 (n = <30),

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FBS-106 mg/dL \( (n = 80–120\, \text{mg/dL}) \), Na\(^+\)-138 mEQ/L \( (n = 130–145\, \text{mEQ/L}) \), K\(^-\)-4.2 mEQ/L \( (n = 3.4–5.5\, \text{mEQ/L}) \), Urea-22 mg/dL \( (n = 7–20\, \text{mg/dL}) \), creatinine-0.7 mg/dL \( (n = 0.7–1.3\, \text{mg/dL}) \), Ca\(^++\)-9.2 mg/dL \( (8.5–10.2\, \text{mg/dL}) \), and Mg\(^++\)-1.7 mg/dL \( (n = 1.7–2.2\, \text{mg/dL}) \). Other common infections like malaria and dengue were excluded. A chest X-ray and ECG were normal. MRI Brain showed [Figure 1] bi-frontal FLAIR hyper-intense areas suggesting focal cerebritis. Inter-ictal EEG [Figure 2] showed no epileptiform discharges, but theta background (possibly post-ictal changes) CSF study showed mild elevation of protein 62 mg/dL \( (\text{normal: } 20–40) \) with cell count of 10 (all lymphocytes). CSF viral screening was negative. Serum autoimmune profile, antinuclear antibody, anti-TPO antibody, and para-neoplastic antibody panel were negative.

She was started with Levetiracetam 500 mg twice daily. She was also prescribed Midazolam Nasal spray as SOS basis. SSRI was advised to continue as earlier. She was well counseled about the provoking and risk factors of seizure and also advised not to miss dose.

After 6-week follow-up in OPD, she was clinically stable and had no side effects of Levetiracetam. Repeat MRI Brain and EEG were normal [Figure 3]. No further episode of seizure was occurred. In addition, she was doing her regular day-to-day activities.

Discussion

Seizure was first reported in covid-19 by Moriguchi et al.\(^{[6]}\) Seizure is possibly due to the direct (through olfactory route) or indirect neuroinvasion of SARS-CoV-2 infection in central nervous system and causing focal cerebritis although the exact mechanism is unknown.\(^{[7,8]}\) Nikbakht et al.\(^{[9]}\) described that covid19 infection may break down the integrity of blood brain barrier (BBB) causing relocation of blood cells and proteins which ultimately disrupts the osmotic balance thus leads to seizure. MRI Brain of our patient showed focal cerebritis likely due to neuroinvasion of SARS-CoV-2 infection. Post-stroke epilepsy is another pertinent cause of seizure.\(^{[10]}\) But in our patient, no such evidence of stroke was found. Our patient was diagnosed with SARS-CoV-2 infection, 2 week prior to seizure and had no previous or family history of seizure. Clinical as well as electrophysiological and imagining evidences showed that she had true epileptic seizure. CSF studies showed mild elevation of protein which is usual in any viral infection and moreover the amount of viral load may be not enough for detection.\(^{[11]}\) Herpetic encephalitis seems to be less likely as there was no finding on MRI brain suggestive of such. This is not a case of nonepileptic seizure (NES) because in NES this type of self-injuries with post-ictal phenomenon is uncommon. Although she has GAD for which she is taking SSRI and has continued this drug throughout her hospitalization during covid-19 illness, seizure was not related to SSRI here. Flavipiravir is so far not known to induce seizure. Volkow suggested that patients with substance use disorder (SUD) are mainly susceptible to covid-19 due to their underlying condition.\(^{[12]}\) Many studies showed electrolyte imbalance in covid-19 patients such as hyponatremia, hypocalcemia, and hypomagnesemia.\(^{[13,14]}\) In our patient, the metabolic causes of seizures were excluded. Emami et al.\(^{[15]}\) reported that seizure rate
in covid-19 patients was 0.08%. There is always a diagnostic dilemma between seizure and syncope, but it is clear that our patient had definite seizure due to the nature of clinical manifestation and post-ictal phenomenon. Thus, the patients who are diagnosed with covid-19 infection even after recovery need proper follow-up under primary care physician.

Conclusion

The message through this case report is that neurological complication like epileptic seizure can occur even after recovery from covid-19 illness. Therefore, internists, neurologists, and specially primary care physicians should be aware of such type of complications to diagnose and treat early. We need further observation and studies to access the spectrum of Covid-19 disease. The cause of the seizure in this patient may be due to invasion of SARS-CoV-2 virus to the brain causing focal cerebritis or due to virulent effect of provocative cytokines.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initial will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

Ethical approval

As it is a retrospective case report, ethical approval is not needed.

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Conflicts of interest

There are no conflicts of interest.

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