## CASE REPORT

WILEY

# Acute psychosis post-COVID-19 pneumonia

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#### **Abstract**

COVID-19 disease can be associated with several health-related consequences that are directly or indirectly related to infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Acute psychiatric illnesses in the setting of COVID-19 infection are one of the reported consequences. In this case report, we discuss acute onset of psychosis in a young patient that we believe was related to post-COVID-19 infection. Some findings in the EEG in this patient, we believe, were related to use of antipsychotic medications and that caused challenges in the diagnosis. It is important to be aware of post-COVID-19 psychosis and challenges that may be encountered in the workup.

#### **KEYWORDS**

acute psychosis, antipsychotic medication, COVID-19 disease, epilepsy, pneumonia

## **CASE PRESENTATION**

An 18-year-old female with history of mild cognitive disability and mild depression and anxiety was initially admitted for respiratory distress secondary to COVID-19 pneumonia. She was treated with remdesivir and daily IV dexamethasone for 10 days with gradual improvement of respiratory symptoms. At the beginning of 3rd week of admission, the patient suddenly developed severe anxiety, insomnia, and intermittent episodes of agitation, delusions, auditory hallucinations, and suicidal ideation. After 3 days, her psychiatric symptoms and mental status worsened.

On exam, she had poor eye contact and minimal coherent speech and was noted to have repetitive tapping of fingers in bilateral hands, as well as purposeless waving of arms, and agitation. The workup was notable for mildly elevated WBC of 14.4 x10<sup>3</sup>/μl, elevated erythrocyte sedimentation rate (ESR) of 51 mm/h, C-reactive protein (CRP) of 14 mg/L, and slightly elevated Alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Cerebrospinal fluid (CSF) was unremarkable (red blood cells <2 cells/µl, white blood cells <3 cells/μl, protein 15 mg/dl, glucose 63 mg/dl, and negative pathogen panel, gram stain, and culture), and serum and CSF autoimmune encephalopathy panels were negative (see supplemental data). MRI brain was unremarkable.

After start of her psychiatric symptoms, she was initially treated with hydroxyzine, risperidone, and olanzapine. Five days after start of risperidone and olanzapine, a routine electroencephalogram (EEG) revealed suspicious low-amplitude spikes embedded in intermittent generalized rhythmic slow discharges (Figure 1) which raised some concerns about seizures as etiology of her psychiatric symptoms. However, a subsequent continuous video-EEG monitoring for 2 days did not show any correlation with patient's episodes of agitation, hallucination, or emotional lability, and it was speculated that the rare EEG findings were related to the effect of antipsychotic medications. Risperidone was gradually increased to 1.25 mg twice daily (BID) with improvement in her symptoms. Patient continued to have anxiety and delusions. Two weeks after onset of psychotic symptoms, patient was discharged home on risperidone 0.5 mg BID, and olanzapine was discontinued with plans to follow up with outpatient psychiatry clinic.

# **DISCUSSION**

Development of acute psychosis because of SARS-CoV-2 infection has been reported in several cases since 2020. 1-3 However, virus-related psychosis is not unique to SARS-CoA-2 infection; it

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FIGURE 1 EEG (electroencephalogram): slow discharges with suspicious embedded low-amplitude spikes, speculated to be due to the effect of antipsychotic medications

is estimated that 0.9-4% of patients infected with H1N1 variant of influenza A, Ebola, SARS-associated coronavirus, or Middle East Respiratory Coronavirus (MERS-CoV) develop new-onset psychosis versus incidence of 0.015% in the general population. Development of psychosis in COVID-19 patients can be either during the active course of the disease or in the post-recovery phase as was in our presented case. Reported symptoms of psychosis secondary to SARS-CoV-2 infection include anxiety, mania, suicidal and homicidal ideations, decreased speech, paranoid delusions, agitation, auditory hallucinations, and psychogenic polydipsia.<sup>4-8</sup> On workup, many patients have had only elevated ESR, CRP, and elevated liver enzymes (AST and ALT), and no acute findings in CT head, MRI brain, and CSF studies similar to our patient. 5-8 Treatment of psychosis in these patients included use of benzodiazepines, antipsychotic medications such as haloperidol, olanzapine, and quetiapine, and antidepressants such as fluoxetine, sertraline, and trazodone. 5-8

The proposed mechanisms of virus-related psychosis are direct viral infiltration of the central nervous system (CNS), either by hematogenous dissemination or by retrograde axonal transport from respiratory tract to CNS via the olfactory nerve, or presence of proinflammatory cytokines leading to neuroinflammation.<sup>8</sup> However, similar to many of the reported cases in literature, we did not find any clear objective evidence to support presence of neuroinflammation.

There are other differential diagnoses besides post-COVID-19 acute psychosis that we should consider in our case. One is steroid-induced psychosis which can occur within 3 days to 3 weeks of start of steroid therapy. Our reported patient received dexamethasone for 10 days and was discontinued before start of the psychosis. Another differential diagnosis to consider is hospitalization-induced

delirium or psychosis; however, this condition is usually seen in higher age groups with an average age of 65 in most reports. Moreover, although certain underlying psychiatric disorders such as schizophrenia and bipolar disorder have been found to have higher association with hospitalization-induced delirium, we were not able to find such association with conditions such as depression and anxiety. Therefore, we do not think that our case had an increased risk of this condition due to her underlying mild depression and anxiety.

Another interesting point in our case was finding of some EEG abnormalities that first raised suspicion about seizures being the underlying cause of psychiatric symptoms which was later ruled out. We speculate that the EEG abnormalities were likely due to the known effect of antipsychotic medications on EEG. However, no follow-up EEG was performed to confirm this speculation.

## **CONFLICT OF INTERESTS**

The authors have no existing conflict of interest.

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# SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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