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Acute psychosis associated with recent SARS-CoV-2 infection: A case report

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ABSTRACT

The COVID-19 pandemic has created overwhelming circumstances not only in the medical field, but in other walks of life. SARS-CoV-2, the causative virus of COVID-19 [1], primarily affects the respiratory system leading to respiratory illnesses of varying severity ranging from mild flu-like symptoms to acute respiratory distress syndrome [2]. However, the clinical manifestations of COVID-19 are not limited to the respiratory system [3]. There is a growing body of literature showing the incidence of a varying clinical spectrum of neuropsychiatric manifestations in a significant proportion of COVID-19 patients [4]. With the variability in neuropsychiatric presentation of COVID- 19, multiple mechanisms have been proposed to explain the pathophysiology of these presentations [5]. In this case report, we present a 20-year-old female with no significant respiratory symptoms or previous history of psychotic episodes who manifested with acute psychosis as a significant complication of COVID-19.

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following rapidly progressing acute psychosis. She had been diagnosed with COVID-19 infection by polymerase chain reaction

(PCR) testing one month prior after exposure to a roommate who

tested positive after developing respiratory symptoms. Her only

symptom at time of diagnosis was fatigue which resolved by

hospital presentation. At baseline, the patient was a high

functioning college student with no history of mental illness.

Approximately 2 weeks after her COVID-19 diagnosis, she

developed insomnia and panic attacks, which progressed over a

span of two weeks to acute psychosis manifested by anxiety,

agitation, forgetfulness, disorganized thoughts, paranoia, flight of

ideas, insomnia and visual hallucinations of her recently decreased

grandmother. She was initially prescribed quetiapine, but due to

progression of psychosis, she was admitted for further diagnostic

COVID-19, a nasopharyngeal swab was obtained and tested by PCR,

and was noted to be positive. Computed tomography of the head without contrast and magnetic resonance imaging of brain did not demonstrate any abnormalities. Continuous electroencephalo-

gram was normal, and both urine drug screen and blood alcohol

test were negative. Acetaminophen, salicylate and heavy metals

toxicities were excluded. Cerebrospinal fluid (CSF) protein, glucose and cellular counts were normal. A meningitis/encephalitis panel (Biofire), which included numerous bacterial and viral targets, was negative. Additional workup including serum human immunode-

ficiency virus (HIV) antigen/antibody and treponema pallidum

Based on the hospital policy regarding admission testing for

Introduction

The COVID-19 pandemic has created overwhelming circumstances not only in the medical field, but in other walks of life. SARS-CoV-2, the causative virus of COVID-19 [1], primarily affects the respiratory system leading to respiratory illnesses of varying severity ranging from mild flu-like symptoms to acute respiratory distress syndrome [2]. However, the clinical manifestations of COVID-19 are not limited to the respiratory system [3]. There is a growing body of literature showing the incidence of a varying clinical spectrum of neuropsychiatric manifestations in a significant proportion of COVID-19 patients [4]. With the variability in neuropsychiatric presentation of COVID-19, multiple mechanisms have been proposed to explain the pathophysiology of these presentations [5]. In this case report, we present a 20-year-old female with no significant respiratory symptoms or previous history of psychotic episodes who manifested with acute psychosis as a significant complication of otherwise mild episode of COVID-19 infection.

Case report

A 20-year-old female with a prior history of mild intermittent asthma and atopic dermatitis was admitted to the hospital

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workup.



Case report





antibody was also negative. Routine laboratory tests including complete blood count (CBC), erythrocyte sedimentation rate, C-reactive protein, creatinine kinase, thyroid stimulating hormone, folic acid and vitamin B12 were within normal ranges. CSF encephalopathy panels were completed, and the results did not suggest autoimmune encephalopathy as an underlying cause for this patient's acute psychosis.

After discussion with the microbiology lab, COVID-19 PCR testing was performed on the patient's CSF sample. Though this test is non-FDA approved, multiple reports in the literature showed that the COVID-19 PCR testing on CSF samples has been performed and was positive in some cases [6,7]. Our patient's CSF sample was negative for COVID-19.

The psychiatry team were consulted, and after assessing the patient's past medical history, clinical manifestations and laboratory findings, the patient was diagnosed with hyperactive catatonia. She was started on high-dose lorazepam, with the intention to taper off the dose in the outpatient settings.

Discussion

We describe a young female, with recent COVID-19 infection and no prior psychiatric history, who developed acute psychosis. Despite extensive workup, no etiology of her symptoms was identified. The rapid onset with no prior psychiatric symptoms was considered to be a highly unusual presentation. Therefore, COVID-19-induced psychosis was thought to be the most likely etiology of her symptoms.

A number of studies have reported the incidence of neuropsychiatric complications in COVID-19 patients [5,8]. More specifically, acute psychosis was described as a complication of COVID-19 in a few studies and case reports [8–13]. However, the pathophysiology of how COVID-19 causes non-respiratory illnesses, including neuropsychiatric complications, is not yet well understood.

Other coronaviruses such as SARS-CoV-1, have shown the ability to gain access to the central nervous system (CNS) via retrograde axonal transport from the peripheral nerves, primarily the olfactory nerve [14]. Based on this, there is speculation that SARS-CoV-2 may have a similar neuro-invasive potential [5,15], which could lead to encephalitis and/or neuropsychiatric complications. Another suggested mechanism of the CNS involvement is through increased permeability of the blood-brain barrier due to significant systemic inflammation allowing SARS-CoV-2 infected lymphocytes to cross into the CNS [7]. Overall, despite the uncertainty regarding the mechanism of SARS-CoV-2 neuro-invasion, there are a reports of CSF PCR tests were positive for SARS-CoV-2 [6,7,16].

CNS side effects may be present in COVID-19 infection for other reasons. Systemic corticosteroids such as dexamethasone have shown benefit in severe COVID-19 and these agents have previously been linked to neuropsychiatric manifestations, including psychosis [17]. As this patient was never systemically ill enough to meet criteria for dexamethasone therapy, steroidinduced psychosis was excluded as a potential etiology in her case.

Mast cells hyperactivation has also been suggested as a cause of COVID-19-induced psychosis. COVID-19 has been found to be associated with overstimulation of both peripheral and central mast cells [18], and mast cell activation has been associated with increased risk of psychiatric symptoms and diseases [19], including paranoid psychosis. Furthermore, psychological stress related to the COVID-19 pandemic or being diagnosed with COVID-19 infection has been suggested as another contributing factor to development of psychiatric illness [18]. An extensive medical and psychiatric history from the family of the patient suggested that this was less likely for this patient.

Clearly there is uncertainty about the exact mechanism by which a COVID-19 illness could trigger psychiatric manifestations and mental stress may have been a contributing factor given the equivocal laboratory and imaging results.

Further research is needed to understand the neuropsychiatric manifestations of COVID-19, and the potential pathophysiologies that might lead to such complications. Physicians treating known COVID-19 patients or in settings with high community spread should be alert and maintain a high level of suspicion for neuropsychiatric symptoms as a possible presentation and or a complication of COVID-19 infection.

Author statement

Laura Selby: writing original draft, resources.

Mohamed Elfil: writing original draft.

Trevor C. Van Schooneveld: Supervision, writing, reviewing, and editing.

Nada Fadul: Supervision, writing, reviewing, and editing.

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Ethical approval

None needed.

Consent

Consent for publication was obtained from patient's medical decision maker per hospital policy by written consent.

Author contribution

Selby- literature review, writing of manuscript. Elfil- writing of manuscript, literature review. Van Schooneveld and Fadul- writing of manuscript.

Declaration of Competing Interest

No conflicts of interest to disclose for any authors.

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