Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Psychiatry Research Case Reports 1 (2022) 100035

Contents lists available at ScienceDirect

Psychiatry Research Case Reports

journal homepage: www.elsevier.com/locate/psycr

New-onset psychosis following COVID-19 infection in a patient with no psychiatric history: A longitudinal case report

Matthew Runyan[a], Jay Fawver[b], Amanda Coupe[c,*], Michelle Drouin[c]

[a] Parkview Health, Parkview Physicians Group – Psychiatry, 1720 Beacon St, Fort Wayne, IN 46805, USA
[b] Parkview Health, Parkview Physicians Group – Mind-Body Medicine, 6920 Pointe Inverness Way, Fort Wayne, IN 46804, USA
[c] Parkview Health, Parkview Mirro Center for Research and Innovation, 10622 Parkview Plaza Dr., Fort Wayne, IN 46845, USA

ARTICLE INFO

Keywords:
SARS-CoV-2
Acute psychosis
Viral inflammation
Lithium
Treatment-refractory psychosis
Outcome metrics

ABSTRACT

Background: Viral infection, including COVID-19, has been implicated as a potential cause of various neurobehavioral issues. An increasing number of case reports suggest that current or recent COVID-19 infection may cause new onset of psychotic symptoms in some individuals, potentially related to viral inflammation or infection of the nervous system.

Case presentation: A 26-year-old woman with no psychiatric history presented with severe psychotic symptoms days after recovery from a mild COVID-19 infection. No other etiologies for psychosis were identified via diagnostic testing, review of medical history, or interviews with family. Her symptoms persisted for approximately two months, requiring three inpatient admissions, various medication trials, and ongoing outpatient follow-up. With continued use of quetiapine and lithium, she returned to living independently and working full-time, and discontinued all medication approximately nine months after symptom onset.

Conclusion: The psychiatric and cognitive effects of COVID-19 infection are not yet fully understood. Given the widespread and ongoing nature of this pandemic, this remains an important focus of further investigation, especially within the context of potential long-term complications.

1. Introduction

The SARS-CoV-2 virus, which causes COVID-19, emerged in December 2019 and has resulted in nearly 550 million infections and over 6.3 million deaths globally as of July 2022 (World Health Organization, 2022). Pandemic-related societal stressors have impacted population mental health; however, evidence also suggests that viral infection increases the risk of various psychiatric complications, including psychosis (Lee et al., 2004; Rogers et al., 2020; Sultana and Ananthapur, 2020; Troyer et al., 2020). Although the etiology of such complications is not fully established, coronaviruses are known to invade CSF and brain tissue (Desforges et al., 2019; Moriguchi et al., 2020) and inflammatory immune response to infection may also cause disruption (Kepinska et al., 2020; Lim et al., 2020; Watson et al., 2021; Smith et al., 2021; Rittmannsberger et al., 2022).

New-onset psychosis concurrent with or immediately following COVID-19 infection has been documented in a growing number of case reports; these etiologies are often posited, though not always supported by diagnostic evidence (e.g., elevated inflammatory markers) (Watson et al., 2021; Smith et al., 2021; Rittmannsberger et al., 2022). Some reports are also confounded by medications used in COVID treatment (e.g., corticosteroids) or prior psychiatric or substance use history, as well as possible delirium (Watson et al., 2021; Smith et al., 2021; Rittmannsberger et al., 2022). Many cases have also been noted to resolve fairly rapidly with treatment, although, given the recency of this pandemic, few provide long-term follow-up details (Smith et al., 2021; Rittmannsberger et al., 2022). We present the first known case in the midwestern United States—a patient with no psychiatric history who required three inpatient admissions over two months and outpatient follow-up for approximately seven months after discharge.

2. Case presentation

2.1. First admission

A 26-year-old white woman presented to the emergency department (ED) with new-onset psychosis and delusions in early 2021. She had been diagnosed approximately two weeks prior with COVID-19, against which she had not yet been fully vaccinated. Her initial infection did not necessitate medical attention, and she recovered from her symptoms

* Corresponding author.
E-mail address: amanda.coupe@parkview.com (A. Coupe).

https://doi.org/10.1016/j.psycr.2022.100035
Received 28 April 2022; Received in revised form 13 July 2022; Accepted 14 July 2022
Available online xxx

(e.g., anosmia, ageusia, mild dyspnea) while isolating at home. Per family, her behavior was generally normal during this time, although her fiancé, with whom she resided, recalled that shortly before her COVID diagnosis, she had been upset by some patient deaths in her work as a neonatal intensive care nurse. He additionally noted that she was not sleeping well in the few days immediately prior to onset of psychosis, during which she had returned to work, but left her second shift early to rest. That night, she called her mother, crying excessively, saying she was dead and her fiancé was going to die. Her mother brought her to the parental home in the morning and to the ED the following day.

Upon initial evaluation, she appeared panicked, paranoid, and disoriented. When a physician asked about a mutual colleague, attempting to establish rapport, she became agitated, stating, "I'm dead, it doesn't matter." She acknowledged her heartbeat and respiration when prompted, but reiterated, "My mind thinks I'm dead." She denied hallucinations but appeared to be responding to internal stimuli. She had no personal or family psychiatric history, no history of substance misuse, and no pertinent medical history besides the recent COVID infection; diagnostic testing (i.e., blood tests, imaging, drug screen—see Table 1) identified no abnormalities or biological causes (e.g., inflammation), and no specific evidence of encephalitis or delirium was noted. She was admitted to the intensive care psychiatric unit with a possible diagnosis of psychotic disorder due to another medical condition, based on consultation with the hospital's infectious disease specialist.

Throughout hospital days 1–5, psychotic symptoms persisted or worsened. She repeatedly acknowledged auditory hallucinations telling her to kill herself and others, at times stating that she had killed her fiancé and a baby, and experienced secondary obsessional guilt. She also expressed delusional content with perception that she had committed suicide or otherwise died, as well as a preoccupation with going to or already being in Hell, although she was otherwise oriented to place, with some periods of disorganization and confusion (e.g., asking if she had been arrested, wandering into other patients' rooms). Initially, risperidone and trazodone were trialed for psychosis and insomnia (see Table 1 for medication details). On multiple occasions, she required haloperidol and lorazepam for agitation, including intramuscular doses on day 4 after she became physically aggressive with multiple staff members and patients and began to bite her fingers, screaming for help, before removing her clothing. Upon recommendation from the infectious disease specialist, dexamethasone was trialed due to lack of improvement on risperidone and the potential role of her preceding viral infection. Additionally, catatonic symptoms were observed, and clonazepam was started. The next day, she appeared delusional, disorganized, and scared, and asked staff to not leave her alone. Dexamethasone was increased to assess benefit, clonazepam was replaced with lorazepam, and further testing (i.e., EEG, lumbar puncture [LP], NMDA antibodies) was planned if no improvement.

On hospital day 6, she began improving, appearing calmer but still disorganized, and reporting no more hallucinations or violent ideation. On day 7, her thoughts seemed more linear and logical, although motor skills were slowed. Risperidone was decreased in concern for overmedication, and she was transferred to the regular psychiatric unit. By day 10, she had no symptoms or side effects and was sleeping well with trazodone; she was discharged the following day.

2.2. Second admission and outpatient follow-up

Medications were adjusted one week later by an outpatient psychiatrist, including tapering lorazepam and dexamethasone. Assessments (i.e., MSE, PHQ-9, GAD-7) indicated improvement in mental status along with some depression and anxiety (see Table 1), but she expressed optimism and gratitude regarding treatment. However, six days later, she returned to the ED, reporting a recurrence of paranoia, delusions, and insomnia starting two days prior, after lorazepam and dexamethasone doses decreased. She acknowledged auditory hallucinations the previous evening and, again, felt she was dead. The inpatient psychiatrist increased lorazepam and dexamethasone doses; she improved rapidly and was discharged three days later.

She was evaluated by the outpatient psychiatrist multiple times over the following four days. Initially, she denied psychosis but complained of poor focus, severe insomnia, and worsening depression and anxiety, reflected in PHQ-9 and GAD-7 scores. Inpatient medications were continued, with trazodone increased. On the third day, she called a helpline multiple times to report suicidal ideation. The next day, she reported worsening anxiety and insomnia, some paranoia, and a preoccupation with being perceived as "faking" her symptoms. Risperidone and gabapentin were increased, and trazodone was replaced with mirtazapine for antianxiety and antidepressant benefits.

2.3. Third admission

The following day, she was brought to the ED after taking 20 trazodone tablets and unknown amounts of lorazepam and mirtazapine. Sedated but conscious and oriented to time and place, she admitted the overdose was a suicide attempt due to distress regarding her cognitive impairment. She stated that she could not see herself living as she felt currently, and then said, "I'm just making this all up. Can I go home?" She was admitted for the next 18 days. Initially, insomnia persisted despite daytime sedation and addition of quetiapine, and her cognitive function fluctuated—sometimes linear and logical, sometimes paranoid and distraught. On day 3 of this admission, she stated that she killed her sister's children, apparently responding to visual and auditory hallucinations, but denied them for the remainder of treatment. Again, diagnostic testing (i.e., LP, MRI, blood tests) early in admission did not identify inflammation, encephalitis, or other physical or systemic abnormalities.

She was transferred to the psychiatric hospital on day 6. She was medically stable but now denied attempting suicide, claiming she was just trying to sleep. However, self-harm injuries were noted on both wrists, to which she admitted, expressing again that she felt frustrated and unable to escape her condition. She additionally admitted to paranoia that people were watching and criticizing her; this continued for several days. On day 7, mirtzapine was discontinued after she reported suicidal ideation. As her condition worsened, lithium 300 mg twice daily was added on day 9 for mood stabilization, neuroprotection, and prevention of suicidality. Electroconvulsive therapy was considered if no improvement; however, the following day, insomnia, paranoia, and cognition improved. Lithium was changed to bedtime only after newly extreme daytime sedation on day 11. Other medications were adjusted, including increasing quetiapine dosage, after some paranoia and insomnia on days 12–13. She continued to improve over days 14–17, stating she had "never felt this much like [herself] since this started." Lithium was increased to 600 mg at bedtime, and she was discharged on day 18.

2.4. Post-discharge outpatient follow-up

For three weeks after discharge, she participated in an intensive outpatient therapy program, attending multiple sessions per week, and additionally met with the outpatient psychiatrist twice, reporting continued improvements in mood, anxiety, sleep, and stress tolerance, with mild sedation but no symptom recurrence. By 26 days post-discharge, she had minimal depression and anxiety per PHQ-9 and GAD-7, and was living independently, driving unaccompanied, and planning to resume part-time work while tapering off risperidone and continuing monthly follow-up. Over the next five months, she remained asymptomatic with no cognitive impairment, depression, or anxiety, resumed full-time work with good performance, and tapered quetiapine due to side effects, discontinuing it 159 days post-discharge. She successfully discontinued lithium on day 207 post-discharge, having been symptom-free for approximately seven months, and has remained off medication without symptom relapse up to the time of writing.

Table 1
Timeline, medication dosing, and clinical evaluations.

First Admission (2.1)			
Days since onset	**Days since admission**	**Medication changes**	**Clinical evaluation notes**
3–4	1–2	Required haloperidol 5 mg and lorazepam 2 mg orally prn for agitation. Started risperidone 1 mg bid, benztropine 1 mg and hydroxyzine 25 mg bid prn, and trazodone 50 mg prn for sleep disturbance.	Oriented to self only. All blood tests WNR: CRP <0.3 mg/L; WBC 8600/μL, RBC 4.56 million/μL, Hgb 13.5 g/dL, HCT 39.7%, MCV 87.1 fL, RDW 11.2%, platelet 225,000/μL; sodium 140 mEq/L, potassium 3.8 mEq/L, chloride 106 mEq/L, CO_2 22 mEq/L, BUN 8 mg/dL, creatinine 0.74 mg/dL, GFR 112 mL/min, glucose 120 mg/dL, calcium 9.5 mg/dL, total protein 7.6 g/dL, albumin 4.3 g/dL, ALT 19 U/L, AST 24 U/L, LDH 208 U/L, alkaline phosphatase 70 U/L, magnesium 2.0 mg/dL, bilirubin 0.9 mg/dL, ferritin 157 μg/L, anion gap 12.0 mEq/L. EtOH 0.0 mg/dL; negative drug screen. Head CT: no acute findings. No physical abnormalities noted. No specific evidence of encephalitis, seizures, or delirium.
6	4	Required intramuscular haloperidol 5 mg and lorazepam 2 mg prn for extreme agitation. Started dexamethasone 2 mg qd and clonazepam 1 mg bid for catatonia.	n/a
7	5	Required haloperidol 5 mg and lorazepam 2 mg orally prn overnight for insomnia. Increased dexamethasone to 2 mg bid. Discontinued clonazepam; started lorazepam 2 mg tid.	Planned to consider LP, EEG, and NMDA antibody test if no changes.
8-9	6–7	Reduced lorazepam to 0.5 mg tid and risperidone to 0.5 mg bid due to side effects (drooling and gagging); started atropine drops.	n/a
10	8	Increased lorazepam to 1 mg tid.	NMDA antibodies: negative.
12	10	Increased trazodone to 100 mg prn.	n/a
13	11	Discharge: Benztropine 1 mg and hydroxyzine 25 mg bid prn, lorazepam 1 mg tid, dexamethasone 2 mg and risperidone 0.5 mg bid, trazodone 100 mg prn.	n/a
Outpatient Follow-Up (2.2)			
Days since onset	**Days since discharge**	**Medication changes**	**Clinical evaluation notes**
20	7	Instructed to taper and then discontinue lorazepam over 8 days; taper dexamethasone over 12 days. Discontinued risperidone; started lurasidone 40 mg qd prn. Started gabapentin 100-200 mg tid prn for anxiety or insomnia.	MSE: All domains normal/appropriate; PHQ-9: 11; GAD-7: 8; WSAS: 16; ISI: 0 PRISE: constipation, nausea/vomiting, throat dystonia, mild fatigue.
Second Admission (2.2)			
Days since onset	**Days since admission**	**Medication changes**	**Clinical evaluation notes**
26	1	Discontinued lurasidone; re-started risperidone 0.25 mg bid. Increased lorazepam to 0.5 mg and dexamethasone to 2 mg tid.	n/a
Outpatient Follow-Up (2.2)			
Days since onset	**Days since discharge**	**Medication changes**	**Clinical evaluation notes**
30	1	Increased trazodone to 50–200 mg qhs prn.	PHQ-9: 21; GAD-7: 21; WSAS: 40; ISI: 28
33	4	Increased risperidone to 0.5 mg bid and gabapentin to 300 mg tid. Discontinued trazodone; started mirtazapine 15 mg qhs.	n/a
Third Admission (2.3)			
Days since onset	**Days since admission**	**Medication changes**	**Clinical evaluation notes**
34–35	1–2	Provided IV hydration for overdose. Started quetiapine 100 mg qhs; re-started benztropine 0.5 mg twice daily; discontinued dexamethasone.	Oriented to self, time, and place. Telemetry monitoring for potential overdose complications.
36	3	n/a	LP and MRI head: no abnormalities. No specific evidence of encephalitis or delirium. EBV: negative. Coagulation and inflammatory panels: WNR. PTT 10.7; INR 1.0; APTT 25.9; fibrinogen 242 mg/dL.
38	5	Discontinued lorazepam; started clonazepam 1 mg bid. Started melatonin 10 mg qhs.	n/a
40–41	7–8	Discontinued mirtazapine due to side effects. Reduced quetiapine to 50 mg qhs. Increased risperidone to 1 mg bid.	n/a
42	9	Started lithium 300 mg CR bid.	ECT considered if no improvement on lithium.
44	11	Reduced lithium to 300 mg CR qhs only due to daytime sedation.	n/a

(*continued on next page*)

Table 1 (*continued*)

45–46	12–13	Changed benztropine to prn due to side effects (constipation and dry mouth). Changed gabapentin to prn. Reduced clonazepam to 0.5 mg bid. Increased quetiapine to 150 mg qhs due to insomnia.	n/a
47–50	14–17	Increased lithium dose to 600 mg CR qhs. Changed clonazepam to 0.5 mg qhs only due to daytime sedation.	Lithium level: 0.4 mEq/L
51	18	Discharge: continue lithium, quetiapine, and risperidone; discontinue clonazepam.	Lithium level: 0.7 mEq/L
Post-Discharge Outpatient Follow-Up (2.4)			
Days since onset	**Days since discharge**	**Medication changes**	**Clinical evaluation notes**
55	4	Increased quetiapine to 200 mg qhs	PHQ-9: 11; GAD-7: 11; WSAS: 32; ISI: 13
62	11	Reduced risperidone to 1 mg qhs only.	PHQ-9: 5; GAD-7: 7; WSAS: 26; ISI: 4
77	26	Instructed to taper risperidone to 0.5 mg and discontinue in two weeks.	PHQ-9: 2; GAD-7: 3; ISI: 1
98	47	n/a	PHQ-9: 1; GAD-7: 0; WSAS: 11; ISI: 0
125	74	Patient requested to reduce quetiapine to 100 mg qhs due to side effects (dry skin and weight gain).	PHQ-9: 0; GAD-7: 0; WSAS: 0; ISI: 0
181	130	Reduced quetiapine to 25–50 mg qhs due to continued side effects.	PHQ-9: 0; GAD-7: 0; WSAS: 0; ISI: 0
210	159	Patient discontinued quetiapine.	n/a
216	165	Instructed to reduce lithium to 450 mg CR qd in two weeks.	PHQ-9: 0; GAD-7: 0; WSAS: 0; ISI: 0
251	200	Reduced lithium to 300 mg CR qd.	n/a
258	207	Discontinued lithium.	n/a
280	229	n/a	PHQ-9: 2; GAD-7: 0; WSAS: 0; ISI: 1

3. Discussion

The patient presented with new-onset psychosis approximately two weeks after diagnosis with COVID-19, consistent with the average timeline in similar cases (Rittmannsberger et al., 2022). However, this case has notable differentiators, including the absence of certain risk factors and length of treatment. Severe symptoms persisted for nearly two months, requiring three separate admissions, various medication trials, and regular outpatient follow-up with continued antipsychotic use for over six months after discharge. While some post-COVID psychosis patients have required treatment over several weeks (e.g., Lim et al., 2020; Correa-Palacio et al., 2020; Parker et al., 2021) many recovered more quickly (Smith et al., 2021; Rittmannsberger et al., 2022; Al-Busaidi et al., 2021; Ferrando et al., 2020; Lu et al., 2020), with one review noting ≤ 2 weeks of symptoms in over 50% of cases and >2 months of symptoms in less than 5% (Rittmannsberger et al., 2022). Most patients are noted to have fully recovered; however, very few existing case reports provide long-term follow-up details with which to compare the full timeline presented here (Rittmannsberger et al., 2022).

Epidemiological research from previous pandemics associates psychosis during or after coronavirus infection with certain risk factors (e.g., steroid treatment, psychiatric history, psychosocial stress) (Lee et al., 2004). Related to COVID-19, specifically, many reports are confounded by the use of medications with known psychiatric side effects to treat the preceding infection and/or inpatient medical treatment, increasing risk of delirium (Watson et al., 2021; Smith et al., 2021; Rittmannsberger et al., 2022). For example, two case reports describe patients with no psychiatric history but whose COVID infections necessitated admission and treatment with glucocorticoids, antibiotics, and/or hydroxychloroquine (Al-Busaidi et al., 2021; Lu et al., 2020). Other cases are additionally complicated by psychiatric history or other risk factors; e.g., Correa-Palacio et al. (2020) describe a patient who both received high doses of methylprednisolone for COVID-19 and had a history of cocaine use. In contrast, our patient had no history of substance use and did not receive treatment for her mild COVID infection; therefore, it is highly unlikely that at least the initial onset of psychosis symptoms was pharmacologically induced. While she was treated with dexamethasone during her psychiatric admission after initial use of risperidone was unsuccessful, she did not develop any new psychotic symptoms after its initiation, and in fact began to improve soon after.

It is also unlikely that onset was entirely triggered by psychosocial stress (e.g., work-related incidents noted by her fiancé), given the lack of particularly novel or severe stressors and pertinent history. Of note, one review of COVID-related psychosis identified psychosomatic differences between cases preceded by infection and those where psychosis was attributed to pandemic stressors (e.g., isolation, anxiety) (Rittmannsberger et al., 2022). Patients with no preceding infection were far more likely to experience symptoms centered around COVID-related themes and to have a history of psychiatric disorders (Rittmannsberger et al., 2022), neither of which apply here. At baseline, the patient had been psychologically healthy, working full-time in a position requiring a high level of mental and emotional function, and living with a partner who did not report any behavioral changes (e.g., depression or mania) preceding onset of psychosis. She did experience new feelings of anxiety, depression, and suicidality after psychosis onset, which she attributed to frustration with this sudden, unexpected, and life-changing health event. Taken together, this presentation is not consistent with other mixed psychotic/mood diagnoses (e.g., psychotic depression, schizoaffective, bipolar). Her symptoms were also not entirely consistent with delirium (e.g., persistent delusions and auditory hallucinations, lack of visual hallucinations and fluctuating consciousness) and all diagnostic testing revealed no underlying brain abnormalities or other potential physical etiology. NMDA receptor encephalitis, a rare but possible cause of similar presentation, was ruled out via antibody test, and repeated evaluation found no additional infectious, traumatic, metabolic, inflammatory, or other potential causes or alternatives.

It is possible, as others have suggested (e.g., Al-Busaidi et al., 2021; Lu et al., 2020) that SARS-CoV-2 affects the brain directly, or indirectly through a compensatory immune response involving the cytokines network and excitatory amino acids (Sultana and Ananthapur, 2020; Lim et al., 2020). No psychiatric cases thus far have documented an active CNS infection, although this has been found with neurological complications (Rittmannsberger et al., 2022). SARS-CoV-2 antibodies, however, have been found in CSF of one patient with psychotic symptoms who had been presumably COVID-positive for 3 weeks (Noone et al., 2020), and one with mania who had recovered from COVID, testing negative one week prior (Lu et al., 2020). Elevated inflammatory markers

have been noted in many cases (e.g., Lim et al., 2020; Ferrando et al., 2020; Chacko et al., 2020), of which one review found that CRP was the most common (present in 59%), but also noted that nearly 25% of patients did not show any signs of inflammation, potentially due to the delay between somatic and psychotic symptom onset, allowing time for inflammation to subside, particularly after a mild infection (Rittmannsberger et al., 2022). This patient was not medically evaluated for over two weeks after COVID diagnosis, by which point she had recovered from her mild illness, and diagnostic testing subsequently continued for a month. Two CRP panels conducted early in treatment were within normal range and initial head CT was unremarkable; later MRI and LP did not indicate encephalitis. CSF was not tested for SARS-CoV-2 RNA or antibodies, and TNF-alpha and IL-6 levels were never evaluated; however, given lithium's documented benefit for TNF-alpha reduction (Nassar and Azab, 2014), the rapid reduction and ultimate resolution of symptoms after lithium was initiated may suggest potential involvement. While it is unlikely to have psychosis secondary to an inflammatory process without evidence of reaction, the possibility of occurrence prior to evaluation cannot be ruled out, and the neurological impact of even low-grade COVID-related systemic inflammation is not yet fully known (Rittmannsberger et al., 2022).

4. Conclusion

This case underscores the current lack of knowledge regarding COVID-19's full impact on the body and brain, and the importance of longitudinal follow-up in contributing to further understanding of a novel virus with potential long-term complications. Other than preceding viral infection, this patient had no notable risk factors for psychosis besides some recent stress, yet developed a severe and life-threatening condition that took longer than expected to resolve. Dexamethasone was helpful after initial use of a benzodiazepine and antipsychotic showed minimal effect; however, lithium had the most significant impact on her recovery. Although inflammatory proteins (i.e., CRP, TNF-alpha, IL-6) are not mainstream evaluations for psychosis, they may be potential markers for individuals with current, recent, or possible COVID infection presenting with psychiatric complications; however, other etiologies (e.g., delirium) should still be investigated (Watson et al., 2021; Smith et al., 2021; Rittmannsberger et al., 2022). The long-term neurological effects of COVID-19 are not yet known, and this remains an important area of investigation to appropriately diagnose and address future complications.

ALT: alanine aminotransferase; AST: aspartate aminotransferase; APTT: activated partial thromboplastin time; bid: twice a day; BUN: blood urea nitrogen; CO_2: carbon dioxide; CR: controlled release; CRP: C-reactive protein; CT: computed tomography; dL: deciliter; EBV: Epstein-Barr virus; ECT: electroconvulsive therapy; EEG: electroencephalogram; EtOH: ethanol (alcohol); fL: femtoliter; g: gram; GAD-7: Generalized Anxiety Disorder scale, 7-item; GFR: glomerular filtration rate; HCT: hematocrit; HgB: hemoglobin; INR: international normalized ratio; ISI: Insomnia Severity Index; L: liter; LDH: lactate dehydrogenase; LP: lumbar puncture; MCV: mean corpuscular volume; mEq: milliequivalent; µg: microgram; µL: microliter; mg: milligram; min: minute; mL: milliliter; MSE: Mental Status Examination; MRI: magnetic resonance imaging; NMDA: N-methyl-D-aspartate; PHQ-9: Patient Health Questionnaire, 9-item (depression); PRISE: Patient-Rated Inventory of Side Effects; prn: as needed; PTT: prothrombin time test; qd: once a day; qhs: once a day at bedtime; RBC: red blood cell; RDW: red blood cell distribution width; tid: three times a day; U: unit; WBC: white blood cell; WNR = within normal range; WSAS: Work and Social Adjustment Scale.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethical considerations

The publication of this case report was approved by the Parkview Health Institutional Review Board, and the patient consented to the dissemination of her case details after recovering.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

The authors would like to thank all emergency department, hospital, and psychiatric staff involved in the treatment of this patient throughout her entire episode of care.

References

Al-Busaidi, S., Al Huseini, S., Al-Shehhi, R., Zishan, A.A., Moghadas, M., Al-Adawi, S., 2021. COVID-19 induced new-onset psychosis: a case report from Oman. Oman Med. J. 36 (5), e303. doi:10.5001/omj.2022.25.

Chacko, M., Job, A., Caston, F., George, P., Yacoub, A., Cáceda, R., 2020. COVID-19-induced psychosis and suicidal behavior: case report. SN Compr. Clin. Med. 2, 2391–2395. doi:10.1007/s42399-020-00530-7.

Correa-Palacio, A.F., Hernandez-Huerta, D., Gómez-Arnau, J., Loeck, C., Caballero, I., 2020. Affective psychosis after COVID-19 infection in a previously healthy patient: a case report. Psychiatry Res. 290, 113115. doi:10.1016/j.psychres.2020.113115.

Desforges, M., Le Coupanec, A., Dubeau, P., Bourgouin, A., Lajoie, L., Dubé, M., Talbot, P.J., 2019. Human coronaviruses and other respiratory viruses: underestimated opportunistic pathogens of the central nervous system? Viruses 12 (1), 14. doi:10.3390/v12010014.

Ferrando, S.J., Klepacz, L., Lynch, S., Tavakkoli, M., Dornbush, R., Baharani, R., Smolin, Y., Bartell, A., 2020. COVID-19 psychosis: a potential new neuropsychiatric condition triggered by novel coronavirus infection and the inflammatory response? Psychosomatics 61 (5), 551–555. doi:10.1016/j.psym.2020.05.012.

Kepinska, A.P., Iyegbe, C.O., Vernon, A.C., Yolken, R., Murray, R.M., Pollak, T.A., 2020. Schizophrenia and influenza at the centenary of the 1918–1919 Spanish influenza pandemic: mechanisms of psychosis risk. Front. Psychiatry 11, 72. doi:10.3389/fpsyt.2020.00072.

Lee, D.T.S., Wing, Y.K., Leung, H.C.M., Sung, J.J.Y., Ng, Y.K., Yiu, G.C., Chen, R.Y.L., Chiu, H.F.K., 2004. Factors associated with psychosis among patients with severe acute respiratory syndrome: a case-control study. Clin. Infect. Dis. 39 (8), 1247–1249. doi:10.1086/424016.

Lim, S.T., Janaway, B., Costello, H., Trip, A., Price, G., 2020. Persistent psychotic symptoms following COVID-19 infection. BJPsych Open 6 (5), e105. doi:10.1192/bjo.2020.76.

Lu, S., Wei, N., Jiang, J., Wu, L., Sheng, J., Zhou, J., Fang, Q., Chen, Y., Zheng, S., Chen, F., Liang, T., Hu, S., 2020. First report of manic-like symptoms in a COVID-19 patient with no previous history of a psychiatric disorder. J. Affect. Disord. 277, 337–340. doi:10.1016/j.jad.2020.08.031.

Moriguchi, T., Harii, N., Goto, J., Harada, D., Sugawara, H., Takamino, J., Ueno, M., Sakata, H., Kondo, K., Myose, N., Nakao, A., Takeda, M., Haro, H., Inoue, O., Suzuki-Inoue, K., Kubokawa, K., Ogihara, S., Sasaki, T., Kinouchi, H., Kojin, H., Ito, M., Onishi, H., Shimizu, T., Sasaki, Y., Enomoto, N., Ishihara, H., Furuya, S., Yamamoto, T., Shimada, S., 2020. A first case of meningitis/encephalitis associated with SARS-coronavirus-2. Int. J. Infect. Dis. 94, 55–58. doi:10.1016/j.ijid.2020.03.062.

Nassar, A., Azab, A.N., 2014. Effects of lithium on inflammation. ACS Chem. Neurosci. 5 (6), 451–458. doi:10.1021/cn500038.f.

Noone, R., Cabassa, J.A., Gardner, L., Schwartz, B., Alpert, J.E., Gabbay, V., 2020. Letter to the editor: new onset psychosis and mania following COVID-19 infection. Psychiatry Res. 130 (1), 177–179. doi:10.1016/j.jpsychires.2020.07.042.

Parker, C., Slan, A., Shalev, D., Critchfield, A., 2021. Abrupt late-onset psychosis as a presentation of coronavirus 2019 disease (COVID-19): a longitudinal case report. J. Psychiatr. Pract. 27 (2), 131–136. doi:10.1097/PRA.0000000000000533.

Rittmannsberger, H., Barth, M., Malik, P., Malsiner-Walli, G., Yazdi, K., 2022. First-episode psychotic disorders in the wake of the COVID-19 pandemic: a descriptive review of case reports. Acta Neuropsychiatr. 31, 1–22. doi:10.1017/neu.2022.11. Epub ahead of print.

Rogers, J.P., Chesney, E., Oliver, D., Pollak, T.A., McGuire, P., Fusar-Poli, P., Zandi, M.S., Lewis, G., David, A.S., 2020. Psychiatric and neuropsychiatric presentations associated with severe coronavirus infections: a systematic review and meta-analysis with comparison to the COVID-19 pandemic. Lancet Psychiatry 7 (7), 611–627. doi:10.1016/S2215-0366(20)30203-0.

Smith, C.M., Gilbert, E.B., Riordan, P.A., Helmke, N., von Isenburg, M., Kincaid, B.R., Shirey, K.G., 2021. COVID-19-associated psychosis: a systematic review of case reports. Gen. Hosp. Psychiatry 73, 84–100. doi:10.1016/j.genhosppsych.2021.10.003.

Sultana, S., Ananthapur, V., 2020. COVID-19 and its impact on neurological manifestations and mental health: the present scenario. Neurol. Sci. 41 (11), 3015–3020. doi:10.1007/s10072-020-04695-w.

Troyer, E.A., Kohn, J.N., Hong, S., 2020. Are we facing a crashing wave of neuropsychiatric sequelae of COVID-19? Neuropsychiatric symptoms and potential immunologic mechanisms. Brain Behav. Immun. 87, 34–39. doi:10.1016/j.bbi.2020.04.027.

Watson, C.J., Thomas, R.H., Solomon, T., Michael, B.D., Nicholson, T.R., Pollak, T.A., 2021. COVID-19 and psychosis risk: real or delusional concern? Neurosci. Lett. 741, 135491. doi:10.1016/j.neulet.2020.135491.

World Health Organization, 2022. WHO coronavirus (COVID-19) dashboard. https://covid19.who.int/ (accessed 7 July 2022).