CASE REPORT

Psychiatry and Clinical Psychopharmacology 2022;32(2):174-177
DOI:10.5152/pcp.2022.21306

Post-COVID-19 Suicide Note: A Case Report of Late Onset Catatonia

Süleyman Dönmezler, Aybegüm Uysal, İmren Kurt, Oya Güçlü

Department of Psychiatry, Başakşehir Çam ve Sakura City Hospital, İstanbul, Turkey

ABSTRACT

Coronavirus disease 2019 (COVID-19) creates acute and long-lasting infection which results in respiratory, cardiovascular, and neuropsychiatric problems. Etiology of neuropsychiatric manifestations can be associated with immune system response, inflammatory cytokines, and also the stressors which are experienced by patients as feeling the risk of being infected by the virus, economic problems, and social distancing. We aimed to present a case of a 53-year-old patient whose suicide note was found and was admitted with depressive and catatonic symptoms 8 weeks after the recovery from COVID-19. Catatonia was suspected, and he was given lorazepam 1 mg. Shortly thereafter, he was entirely alert, cooperative, and oriented. As an advantage of this case, the patient in our report had not used medications for COVID-19 and so we could exclude the effect of medications to the pathophysiology of post- coronavirus disease symptoms. A wide spectrum of neuropsychiatric manifestations was observed in terms of diagnosis after COVID-19. Catatonia can break out in the post-infectious period as well as in the para-infectious period. There are limitations to figure out the direct invasion of coronavirus and the effect of the systemic inflammation to the central nervous system. Nevertheless, it should be considered that catatonia may be one of the clinical results of COVID-19.

ARTICLE HISTORY

Received: December 6, 2021
Accepted: April 11, 2022

KEYWORDS: Catatonia, COVID-19, depression, lorazepam, sertraline, suicide (attempted)

INTRODUCTION

Coronavirus disease 2019 (COVID-19), an acute respiratory syndrome caused by severe acute respiratory syndrome coronavirus-2, creates acute and long-lasting infection which results in respiratory, cardiovascular, and neuropsychiatric problems.[1,2] There is no certainty about the etiology of neuropsychiatric symptoms that break out after COVID-19. It can be related to virus effect on the brain directly or a side effect of the treatment which includes favipiravir and hydroxychloroquine.[3] Furthermore, the etiology of neuropsychiatric manifestations can be associated with immune system response, inflammatory cytokines, and also the stressors which are experienced by patients as feeling the risk of being infected by the virus, economic problems, social distancing, and uncertainty of treatment.[4]

There have been reports of patients who were infected by coronavirus-associated depression, anxiety, and insomnia.[5,6] This case is about the patient who recovered from COVID-19, but 8 weeks later was admitted to our clinic with major depression with catatonic features in July 2021.

CASE PRESENTATION

A 53-year-old male patient with no prior psychiatric history was admitted to the emergency department with apathy, decreased mobility, muteness, insomnia, and refusal to eat or drink consistently. His history was obtained from his family as the patient was mute to the treatment team. Two months prior to the current presentation, he was admitted to the emergency department with a loss of smell and taste, myalgia, and sweating. Coronavirus disease 2019 RNA was detected by a reverse transcription-polymerase chain reaction nasopharyngeal swab. Then, the treatment of COVID-19 with favipiravir and hydroxychloroquine was prescribed, but he refused to use the medication. He used only vitamin C supplements and acetylsalicylic acid for 3 days. During the acute COVID-19 infection for 10 days, he was isolated at home and his vital signs were normal and required no supplemental oxygen. Loss of smell and taste and the infection itself resulted in an 8-pound weight loss. According to his family, after the COVID-19 infection, his "character changed," and he became increasingly depressed with social withdrawal and a sense of hopelessness and helplessness. His suicide note with "I'm done, goodbye!" was

Corresponding author: Süleyman Dönmezler, e-mail: suleymandonmezler@gmail.com

Cite this article as: Dönmezler S, Uysal A, Kurt İ, Güçlü O. Post-COVID-19 suicide note: A case report of late onset catatonia. Psychiatry Clin Psychopharmacol. 2022;32(2):174-177.

found by his wife and he was hospitalized due to the suicide risk and attempted suicide by drinking sodium carbonate. Physical examinations, blood tests (urea and electrolytes, liver function tests, thyroid function tests, c-reactive protein, and complete blood examination) resulted in normal findings. Neurological examination revealed stupor, muteness, fixed gaze, and negativism. According to the mental state examination upon admission, his appearance was neat, he was mute, his eye contact was avoidant, motor activity was slowed, the mood was depressed, affect was flat, orientation, memory, and concentration could not be assessed, and he had no hallucinations. According to the anamnesis received from the family, no delusions were detected, but his judgment was poor.

Catatonia was suspected and he was given lorazepam 1 mg at the emergency department. Shortly thereafter, he was entirely alert, cooperative, and oriented. Upon the admission, electroencephalography (EEG) was normal with no seizures, no postictal activity. Magnetic resonance imaging (MRI) of the brain revealed no significant findings, and brain computed tomography (CT) was normal. Computed tomography of the chest showed basal bilateral ventilation asymmetry that pointed to the past COVID-19 infection.

The Montgomery and Asberg Depression Rating Scale (MADRS) score was 32, Hamilton Depression Rating Scale score was 16, and the Positive and Negative Syndrome Scale (PANNS) score was 78 at the time of the admission. He was administered sertraline 100 mg/day and mirtazapine 15 mg/day. Since his symptoms persisted, lorazepam trial was initiated by gradually increasing to 3 mg/day. He was appropriately engaging in conversation and oriented to person, place, and time. After 18 days in hospital, the patient performed daily living activities volitionally and he was discharged from the hospital with sertraline 100 mg/day and mirtazapine 15 mg/day.

Unfortunately, 4 days after discharge, he was admitted to the emergency department with immobility, muteness, and refusal to eat or drink. He was given lorazepam 1 mg at the emergency department. Physical examinations, blood tests (urea and electrolytes, liver function tests, thyroid function tests, c-reactive protein, complete blood examination), MRI of the brain, the brain CT, and EEG revealed no significant findings. The vasculitis panel (cryoglobulin, anti-centromere, MPO-ANCA, anti-beta2 glycoprotein immunoglobulin M (IgM), anti-histone, anti-cardiolipine IgM, anti-beta2 glycoprotein IgG, anti-cardiolipine IgG, antithrombin3, homocysteine, proteinS activity, proteinase-3 antineutrophil cytoplasmic antibodies (PR3-ANCA), anti-Sjögren's syndrome type B (anti-SSB), anti-Sm, anti-dsDNA, anti-Jo1, anti-scl70, anti-nuclear antibody (ANA), anti-Smith (Sm)/ ribonucleoprotein (RNP), prothrombin time (PT), activated partial thromboplastin time (aPTT), D-dimer, fibrinogen, lupus anticoagulant, C3, rheumatoid factor (RF), anti-Cyclic citrullinated peptide (CCP), complement component 4 (C4), and protein C antigen) was tested and revealed no significant findings except homocysteine: 17.8 (n: 0-15) and D-dimer: 1.94 (n: 0-0.5) (Table 1).

Table 1. Biochemical, Hematological, and Coagulation-Related Tests of the Patient on the Time of the Second Admission

WBC	9.46	10^9/L	3.91-10.9
LYM	2.51	10^9/L	1.26-3.35
NEU	5.88	10^9/L	1.8-6.98
CRP, turbidimetric	1.9	mg/L	0-5
D-dimer, quantitative	**1.94**	µgFEU/mL	0-0.5
Homocysteine	**17.8**	µmol/mL	0-15
Ferrritin	52	µg/L	24-336
Procalcitonin	0.002	ng/mL	<0.1
Urea	25.5	mg/dL	13-43
Creatinine	0.79	mg/dL	0.7-1.42
Na^{+2}	142	mEq/L	136-146
Cl^-	102	mEq/L	96-106
Ca^{+2}	9.04	mg/dL	8.6-10
Mg	2.04	mg/dL	1.6-2.6
P	3.24	mg/dL	2.5-4.5
ALT	22	U/L	0-50
AST	17	U/L	10-50
LDH	204	U/L	0-250
CK	31	U/L	0-190

Abnormal values are indicated in bold.
WBC, white blood cell; LYM, lymphocytes count; NEU, neutrophils count; CRP, c-reactive protein; Na^{+2}, sodium, Cl^-, chlorine; Ca^{+2}, calcium; Mg, magnesium; P, phosphorus; ALT, alanine transaminase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; CK, creatine kinase.

After hospitalization, his treatment consisted of a combination of olanzapine 7.5 mg/day, sertraline 200 mg/day, and aripiprazole 2 mg/day. He responded well to this combination. One week after admission, he started to speak more fluently than before. His behaviors improved day by day, and his suicidal ideation ameliorated. The

MAIN POINTS

- Besides causing respiratory and cardiac problems, COVID-19 is also associated with neuropsychiatric complications.
- Depressive symptoms can be associated with immune system response, inflammatory cytokines, as well as isolation and lacking of social support.
- The case is about the patient who recovered from COVID-19, but was presented with major depression with catatonic features eight weeks later.
- Lorazepam, as an acute intervention, improved dramatically catatonic symptoms, after which sertraline selected as maintenance of depression treatment.

MADRS score diminished to 16. Hamilton Depression Rating Scale score decreased to 14. The PANNS score diminished to 78.

Hamilton Depression Rating Score decreased to 2 from 4 after the observations that were made at the examination from the patient's discharge. The patient has started swimming and training 2 times a week; he also started to spend more time with his family and put effort into gaining new hobbies. Regarding his treatment, also, progress can be seen there as well, the dose of olanzapine was decreased gradually from 7.5 mg/day to 2.5 mg/day. Initially, he has put on about 7 kg; however, this rate has decreased over time in correlation with the decreased olanzapine dose. At that point, we have reached the conclusion that he no longer needs the aripiprazole treatment, but we will continue with sertraline treatment with the dose that will suit best for him.

Informed consent was obtained from the patient who participated in this study.

DISCUSSION

We aimed to present a case of a 53-year-old patient whose suicide note was found and was admitted with depressive and catatonic symptoms 8 weeks after the recovery from COVID-19. As an advantage of this case, the patient in our report had not used medications for COVID-19, so we could exclude the effect of medications on the pathophysiology of post-COVID symptoms. In addition to the side effect of the treatment of COVID-19 was found in some clinical studies.[3,4]

Most of the cases of COVID-19-associated neuropsychiatric symptoms are related to systemic inflammation and cytokine storm. Systemic inflammation which includes interleukin (IL)-18, IL-6, IL-10, and tumor necrosis factor-alpha is the cause of immune dysregulation, that is, the mechanism of the cytokine storm. Cytokine storm and microglia activation disorganize the levels of neurotransmitters and coagulants. Also, direct damage to receptors, secondary hypoxia along nerve fibers, and disruption of regular neurotransmission are the other reasons for the neuropsychiatric symptoms.[7] The increase of proinflammatory cytokines which are interferon-gamma and IL-7 cross the brain–blood barrier and can cause autonomic dysregulation. High levels of IL-6, IL-1, interferon-gamma, CXCL-10 and CCL-2 may contribute to COVID-19 by activating T-helper-1.[5]

The mechanism by which isolation and reduced social support can lead to depressive symptoms should also be stressed. There are multiple reports that include catatonia and depression symptoms occurring secondary to COVID-19.[8] According to a meta-analysis, the prevalence of depression for COVID-19 inpatients has substantially increased compared with pre-pandemic depression levels. Additionally, the prevalence of depression for outpatients is also higher compared with the pre-pandemic prevalence.1 It is unnecessary to investigate cytokine storm as a possible mechanism in a patient not requiring hospitalization or treatment.

There are some limitations in this case report. Firstly, we did not examine the cerebrospinal fluid or observed the autoimmune encephalitis. Encephalitis is a rare manifestation of catatonic syndromes, and the anti-N-methyl-D-aspartate (NMDA) receptor encephalitis is the most common cause of the autoimmune catatonia. For this reason, anti-NMDA receptor, anti-LGI-1, and anti-CASPR-2 could have been evaluated. Secondly, it is not possible to interpret these results as a robust relationship between catatonic depression and the inflammatory response to COVID-19. The lack of social interaction due to isolation during the COVID-19 pandemic may also have deteriorated the patient's mood.

A wide spectrum of neuropsychiatric manifestations is observed in terms of diagnosis after COVID-19. Catatonia can break out in the post-infectious period as well as in the para-infectious period. The catatonia that occurs after COVID-19 infections without antiviral treatment points out the primer effect of COVID-19 to the central nervous system and secondary effect of systemic inflammation. Due to the pandemic conditions, there are limitations to figure out the post-COVID medications, the direct invasion of coronavirus, and the effect of the systemic inflammation to the central nervous system. Nevertheless, it should be considered that catatonia may be one of the clinical results of COVID-19.

Informed Consent: Informed consent was obtained from the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – S.D., A.U.; Design - S.D., İ.K.; Supervision - O.G., S.D.; Resource - A.U., O.G.; Materials - A.U., İ.K.; Data Collection and/or Processing - A.U., S.D., İ.K.; Analysis and/or Interpretation - S.D., O.G.; Literature Search - A.U., İ.K., S.D.; Writing - S.D., A.U.; Critical Reviews - O.G., S.D.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

REFERENCES

1. Deng J, Zhou F, Hou W, et al. The prevalence of depression, anxiety, and sleep disturbances in COVID-19 patients: a meta-analysis. *Ann N Y Acad Sci*. 2021;1486(1):90-111. [CrossRef]
2. Sarli G, Polidori L, Lester D, Pompili M. COVID-19 related lockdown: a trigger from the pre-melancholic phase to catatonia and depression, a case report of a 59 year-old man. *BMC Psychiatry*. 2020;20(1):558. [CrossRef]

3. Yesilkaya UH, Balcioglu YH, Sen M. Psychotic agitation in a patient with COVID-19: pathogenesis or iatrogenesis? *Psychiatry Clin Psychopharmacol*. 2021;31(2):238-240. [CrossRef]
4. Vazquez-Guevara D, Badial-Ochoa S, Caceres-Rajo KM, Rodriguez-Leyva I. Catatonic syndrome as the presentation of encephalitis in association with COVID-19. *BMJ Case Rep*. 2021;14(6):e240550. [CrossRef]
5. Torrico T, Kiong T, D'Assumpcao C, et al. Postinfectious COVID-19 catatonia: A report of two cases. *Front Psychiatry*. 2021;12:696347. [CrossRef]
6. Zain SM, Muthukanagaraj P, Rahman N. Excited catatonia - A delayed neuropsychiatric complication of COVID-19 infection. *Cureus*. 2021;13(3):e13891. [CrossRef]
7. Mazza MG, Palladini M, De Lorenzo R, et al. Persistent psychopathology and neurocognitive impairment in COVID-19 survivors: effect of inflammatory biomarkers at three-month follow-up. *Brain Behav Immun*. 2021;94:138-147. [CrossRef]
8. Lynch A, Bastiampillai T. COVID-19 pandemic-induced late-onset psychotic depression With catatonia. *Prim Care Companion CNS Disord*. 2021;23(1):20l02827. [CrossRef]