Post-COVID-19 psychosis: Cotard's syndrome and potentially high risk of harm and self-harm in a first-onset acute and transient psychotic disorder after resolution of COVID-19 pneumonia

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Abstract
Aim: This report aims to illustrate the possibility of an acute onset of psychosis after COVID-19 infection in a patient without previous history of psychiatric disorders and to highlight the need for early screening and intervention in such cases.

Methods: Clinical presentation of a case, followed by clinical discussion and literature review of the effect of the new coronavirus SARS-CoV-2 and its impact on mental health in terms of neuropsychiatric conditions.

Results: We present a case of acute and transient psychotic disorder following complete recovery of COVID-19 bilateral pneumonia. The patient has no prior psychiatric history and presents with acute onset, disorganized behaviour, Cotard’s delusion and a potentially high risk of psychotic homicide and suicide.

Conclusion: Early intervention and treatment with antipsychotic medication are of crucial importance for the effective treatment and complete recovery of these patients.

KEYWORDS coronavirus SARS-CoV-2, Cotard’s syndrome, first-onset psychosis, post-COVID-19 psychosis

1 INTRODUCTION

The COVID-19 pandemic caused by the new coronavirus SARS-CoV-2 has a significant impact on mental health globally. On one hand, the prolonged psychological and psychosocial stressors such as fear of disease and/or death, unexpected losses, the imposed isolation and insecurity about the future (Walker & McCabe, 2021) when combined with inadequate coping mechanisms can lead to emergence of reactive psychiatric symptoms (Fischer et al., 2020) with the most common COVID-crisis related mental health problems being insomnia, anxiety, and depression (Majadas et al., 2020). Rarely, cases of COVID-crisis related reactive psychosis have been described in SARS-CoV-2- negative individuals, including healthcare workers (Agostino et al., 2021).

On the other hand, the COVID-19 infection itself is associated with various neuropsychiatric symptoms. An estimated 34% of the patients surviving COVID-19 are diagnosed with a neuropsychiatric disorder in the following 6 months (Taquet et al., 2021). Neuropsychiatric manifestations range from delirium to long-term fatigue, with the most common diagnosis being mood or anxiety disorder (Butler et al., 2020). The estimates for the incidence of psychotic disorder after COVID-19 infection seem relatively low: 1.40% for all psychotic disorders and 0.42% for first-onset psychotic disorders. Yet, there are reports of first-onset psychosis with grossly disorganized (including homicidal and suicidal) behaviour after COVID-19 infection (Ferrando et al., 2020). Therefore, screening and long-term monitoring of individuals at risk for acute psychiatric complications have been recommended (Butler et al., 2020).
This report presents a case of a new onset of acute and transient psychotic disorder following treatment and complete recovery from COVID-19 pneumonia in a patient without previous history of psychiatric disorders. The purpose is to illustrate the possibility of an acute onset of psychosis after COVID-19 infection and highlight the potential risk of harm or self-harm.

1.1 | Case presentation

Mr. A is a 43-year-old male, married, with two children, who runs a family business and has no serious medical conditions in his medical history. He has never been referred for psychiatric consultation or psychiatric treatment before and has no family history of psychiatric disorders.

Initially, Mr. A presented in a medical outpatient setting with subfebrile temperature, cough and weakness lasting several days. He tested positive for SARS-CoV-2 on a PCR test and was treated first in an outpatient setting with antibiotics (Amoxicillin 500 mg BID for 5 days), vitamins (Vitamin C and D) and antiaggregants (Aspirin 300 mg for 5 days). On the seventh day of the positive PCR test, he had a chest X-ray showing development of pulmonary pneumonia and his saturation dropped to 90%, so he was hospitalized in a COVID-19 ward. The treatment continued with non-steroid anti-inflammatory drugs (Paracetamol 500 mg, PRN), antibiotics (Ceftriaxone 2 mg per day for 14 days), mucolytics (Bromhexine 8 mg BID for 10 days), corticosteroids (Dexamethasone 4 mg i.v. BID for 10 days), antiocoagulants and antiaggregants (Enoxaparine sodium 0.6 mg s.c. for 14 days then stopped and switched to Aspirin 300 mg per day) and saline and glucose infusions.

He had no signs of hypoxemia and his saturation in hospital setting was above 94% without him requiring oxygen. He was determined to have a mild to moderate case of COVID-19 pneumonia without hypoxemia. He had no psychiatric symptoms present during the hospital stay and upon his discharge. He was discharged him upon complete resolution of somatic symptoms after 3 weeks of hospital treatment. He had a negative PCR test upon discharge and his recommended treatment included only Aspirin 300 mg per day.

At home, he became anxious and tense; he could not sleep for the first 2 days. Then, he started speaking of having permanent damage and not being cured from the virus. He blamed the doctors for having faked the negative results. On the third day after his discharge, he became highly agitated, and he smashed walls and doors at his home. He had hypochondriac to nihilistic delusions that he was still infected with the virus. He was sure that the damage done to his lungs is irreversible and that there is probably damage to the brain. The delusions even reach Cotard’s syndrome. The patient expressed ideas that he has already died, and his organs are rotten, so this is why before his hospital admission, he wanted to kill his relatives to spare them the same slow suffering.

His treatment started with Haloperidol, Zuclopenthixol, Promethazine, glucose and saline infusions. After 5 days of parenteral therapy, his conditions rapidly improved. The patient became cooperative and began taking Haloperidol 9 mg per day orally and Olanzapine 10 mg per day. Within 2 weeks, the psychotic symptoms resolved completely and Haloperidol was gradually discontinued. The patient was fully compliant and aware of having had a psychotic episode. He was discharged in a stable mood without any psychotic symptoms, his treatment being monotherapy with Olanzapine 10 mg per day and a referral for a follow-up and evaluation in outpatient setting.

The first follow-up was scheduled 30 days after the onset of psychotic symptoms. The patient appeared fully compliant, without any psychotic symptoms, and at no risk of harm or self-harm. He reported, however, mild fatigue, mood instability and anxiety. On second follow-up 3 months after the discharge, the patient was having a complete recovery and was in stable mood without any psychotic symptoms. Monotherapy with Olanzapine 5 mg per day for another 3 months was recommended as a prophylaxis.

2 | DISCUSSION

In summary, this report presents a case of post-COVID first-onset psychosis with potentially high risk for psychotic homicide and suicide in a 43-year-old male without prior psychiatric history. The clinical presentation consists of acute onset of psychotic symptoms and grossly disorganized behaviour (within 2 days after discharge from somatic hospital) with polymorphic clinical presentation: delusional affect, fear, confusion, selective mutism, fluctuations in the conditions with rapid changes from paranoid delusions of persecution, harm and reference to nihilistic delusions up to Cotard’s delusion, delusions of
impoverishment and doom, aggressive behaviour with imminent risk of harm and self-harm. The episode resolved completely in less than 30 days after treatment with high doses of typical neuroleptic. Fatigue and non-specific affective symptoms after the resolution of psychotic symptoms are present for 3 months, followed by a complete recovery on monotherapy with Olanzapine.

Some hypothesis of the onset of neuropsychiatric symptoms in COVID-19 patients include severe pneumonia with hypoxia, autoimmune mechanisms, and inflammation, treatment with corticosteroids, and direct neurotropic effect of the virus (Varatharaj et al., 2020). In the current case, no plausible mechanism could be identified as a direct cause of the psychosis because the psychotic symptoms developed after complete somatic recovery and the patient had negative PCR and negative antigen test for SARS-CoV2. Prior to the psychotic symptoms, the patient did not have severe COVID-19 illness but a moderate pneumonia without hypoxemia and without need for oxygen treatment. He was on short-term treatment with low doses of corticosteroids but they were discontinued 2 weeks before the onset of psychotic symptoms. Antibiotic treatment was present only during the somatic hospital treatment and were then discontinued. Upon the onset of psychosis the patient had no other medication except for antiaggregant given for prophylaxis due to slight elevation of some parameters in the haemocoagulation panel. Upon psychiatric admission, he had no clinical or laboratory signs of inflammation and the C-reactive protein was within the normal range.

Therefore, ruling out potential somatic causes such as hypoxia, direct neurotrophic effect or corticosteroid-induced psychosis, the clinical presentation of the case is similar to cycloid psychosis and should be classified as an episode of Acute and transient psychotic disorder (F23 according to the ICD-10). The differential diagnosis of Organic psychotic disorder (F06) could be considered only if a plausible mechanism can be assumed as causative for the psychosis, but here we could not assume a direct somatic cause. Also, the patient required high doses of potent typical antipsychotic initially while in other COVID-19-induced psychosis case presentations are of a rapid resolution on low doses of antipsychotics (Parra et al., 2020). Therefore, the onset and resolution of psychotic symptoms and their variation in time, as well as the required dosage of antipsychotics drugs in this case are consistent with the independent psychiatric diagnosis.

In literature review, the incidence rate of new-onset psychosis in COVID-19 patients might seem relatively low compared to other psychiatric disorders (0.42% 6-month incidence of first-onset psychosis in COVID-19 survivors) (Taquet et al., 2021). However, the estimated actual annual incidence rate of first-onset psychosis in the general population is 0.086% among those aged 15 to 29 and 0.046% in those aged 30 to 59. (Simon et al., 2017) This might indicate that incidence of newly-onset psychosis after COVID-19 can be much higher than expected. The most common features of new-onset psychoses are reported to be structured paranoid and nihilistic delusions mixed with confusion, similar to our case (Parra et al., 2020).

It appears that COVID-19-related topics like virus contamination and impending doom are tightly involved in the phenomenology of psychosis during the COVID-19 pandemic with the result of the production of nihilistic or even Cotards’ delusions and the potential for increased risk of harm and self-harm. These topics are observed in COVID-induced psychosis as cases describe grossly disorganized (including homicidal and suicidal) behaviour as first manifestations of COVID-19 in otherwise asymptomatic, SARS-CoV-2 positive individuals. (Ferrando et al., 2020). Also, they are present in COVID-crisis related exacerbation of psychosis without COVID-19 illness (Fischer et al., 2020).

This case illustrates that acute psychiatric complications in COVID-19 patients can be a severe concern (Butler et al., 2020) as psychotic disorders are normally associated with substantial premature mortality and morbidity and here we have the addition of the influence of the COVID-19 pandemic on the phenomenology of psychotic symptoms.

3 | CONCLUSION

First-onset psychosis after COVID-19 infection can be observed and have a potentially high risk of harm or self-harm. It is crucial to raise awareness and monitor psychotic symptoms in COVID-19 survivors to prevent potentially hazardous outcomes. Early intervention and proper use of antipsychotic medication are of crucial importance for the effective treatment and complete recovery of these patients.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The authors confirm that the data supporting the findings of this study are available within the article.

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How to cite this article: Ignatova, D., Krasteva, K., Akabalieva, K., & Alexiev, S. (2022). Post-COVID-19 psychosis: Cotard’s syndrome and potentially high risk of harm and self-harm in a first-onset acute and transient psychotic disorder after resolution of COVID-19 pneumonia. Early Intervention in Psychiatry, 16(10), 1159–1162. https://doi.org/10.10111/eip.13254