THE NEUROPSYCHIATRIC FACE OF COVID-19: A BRIEF PSYCHOTIC ATTACK CASE REPORT

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INTRODUCTION

COVID-19 is a disease that causes an acute severe respiratory syndrome, the agent is SARS-COV-2 (World Health Organization 2020). Although many effects of COVID-19 on health and social life have been observed, it has effects on many diseases and systems waiting to be discovered. Initially, psychiatric research focused on the emotional effects of illness on the general population and psychiatric illnesses (Rehman & Lela 2020). Further studies show that COVID-19 can cause many psychiatric diseases, including depression, panic disorder, and post-traumatic stress disorder (Anjum et al. 2020). Moreover, as Troyer et al. did, studies examining its direct role in the development of neuropsychiatric symptoms related to Sars-Cov-2 have also started (Troyer et al. 2020). The findings of COVID-19 that can be associated with central nervous system (CNS) involvement can be listed as new-onset loss of taste and smell, cerebrovascular diseases, encephalopathy, encephalitis. Recent studies reveal cases of COVID-19 with reactive psychosis and neurological symptoms (Mao et al. 2020). Here, we present a case of psychosis that could be associated with symptomatic COVID-19 in a patient with no personal history of psychotic illness.

CASE REPORT

A 41-year-old female patient diagnosed with COVID-19 was referred to our psychiatry clinic with complaints of aggression, mystical delusions, inappropriate affection, disorganized behavior, refusal to eat, and not sleeping for the last 4 days. The total score on the Positive and Negative Syndrome Scale (PANSS) at admission was 132, and aripiprazole was added at 5 mg/day and gradually increased to 15 mg/day within 10 days. She was taking Leflunomide 20 mg/day, Feno-fibrate 267 mg/day, Metformin 1000 mg/day, Piperacillin-tazobactam 18 g/day in the treatment. There is no history of smoking, alcohol, or substance abuse. She had depression, diabetes mellitus, psoriasis, previous tuberculosis disease, and hypertriglyceridemia. There was no family history of psychiatric illness.

In her first admission, the patient had poor self-care, dull, disoriented, and distracted, the speed and amount of speech decreased, the associations were relaxed, the thought content was limited, she had delusions, without hallucinations, there was no insight.

On physical examination, there were no pathological reflex, lateralizing signs, extrapyramidal system findings, nuchal stiffness, and bilaterally motor forces were normal, there was a chronic plaque lesion compatible with psoriasis on the right tibia anterior aspect.

In the examinations at admission, CRP was 178, Creatine Kinase was 1064, LDH was 491, Ferritin was 1685, and was higher than the reference ranges; Procalcitonin, D-Dimer, Hemogram, Electrolytes, Liver, Kidney, and Thyroid function tests were within the normal range.

Cranial Magnetic Resonance Imaging was normal, and Electroencephalography wasn’t characteristic. The patient applied to the external center 2 weeks ago with fever, cough, and headache complaints. The clinicians performed the thorax computer tomography (CT) and the saliva test of SARS-CoV-2 RT-PCR. The patient was admitted to the hospital showing bilateral ground-glass opacities on CT (shown in Figure 1) and positive for RT-PCR test. Pneumonia symptoms did not improve despite being treated with Favipiravir 600 mg and levofloxacin 750 mg all 5 days, the CRP value was found to be 122 on the fifth day of treatment, and extensive consolidation areas in favor of bacterial pneumonia was observed in the repeated thorax CT (shown in Figure 2). Piperacillin-tazobactam 18 g/day was added due to secondary bacterial pneumonia and stopped favipiravir, levofloxacin. In the second week of her hospitalization, she was referred to our psychiatry clinic from an external center due to worsening personal care functionality, negativism, non-goal orientation, aggressive behavior, persecutive delusions, and disorientation. With Piperacillin-tazobactam 18 g/day and aripiprazole 15 mg/day application, after 2 weeks of follow-up, the values decreased to CRP 50, Creatine Kinase 180, LDH 340, D-Dimer, Quantitative 0.24, Ferritin 390, respectively; her orientation and attention were normal, affect was slightly suppressed, speech speed and the amount increased, thought content was limited, he was goal-oriented, delusion and hallucinations were regressed. PANSS score fell 48 and reapplied SARS-CoV2 RT-PCR was negative. The patient was thought to have a short psychotic attack due to COVID-19 and was discharged with 15 mg/day oral aripiprazole. She was followed for 6 months. The dose of aripiprazole was gradually tapered and discontinued. The patient who has no residual symptoms is being followed up without medication.
DISCUSSIONS

In our case, a patient with COVID-19 was reported with a psychotic attack characterized by new-onset delusional and disorganized behavior. No findings were indicating central nervous system infection on nor neurologic examination neither laboratories. The mental state of the patient did not fluctuate during the day and considering the sudden onset of the complaints after infection, the patient was evaluated as a short psychotic attack due to COVID-19. Lumbar puncture wasn't performed because infectious diseases specialists didn't see it necessary for the patient had no encephalitis/meningitis symptoms.

Coronavirus infection can directly damage cells; also indirectly, such as hypoxic damage, immune damage, increase in hyperinflammatory response can cause disorders in systems in other ways (Troyer et al. 2020). SARS Cov-2 induces a more extensive immune response involving proinflammatory chemokines and cytokines after a series of reactions to be involved dendritic cells following infection of respiratory cells. So, the CNS may be the first organ affected due to a risky blood-brain barrier (BBB), which is closely related to increased circulating cytokine levels. If the tight junctions of the BBB become loose or break, the barrier becomes more permeable and cytokines can leak inside, and activate microglia and as a result of the reactions that occur. Later all these reactions can cause CNS dysfunction and encephalopathy (Meneses et al. 2019). In our case, as stated in the Serrano-Castro study, we reported cases of CRP and Ferritin elevation, and we thought that the hyperinflammatory response in CNS may also be related to the pathogenesis of primary
psychotic disorders (Mehta et al. 2020, Balcioglu et al. 2020, Serrano-Castro et al. 2020). Iatrogenic factors should also be considered. Psychotic findings are rare side effects that can occur with the use of levofloxacin or favipiravir (Chen et al. 2020). Favipiravir and levofloxacin have no dual drug interaction. Besides that, as in the case of Kiangkitiwan et al., the drug-related side effects decrease in a very short time after the drug is discontinued, and the side effects of the drug cause damage to other organs such as liver, kidney, and heart; but the absence of such situations in our case has kept us away from drug-related psychotic attacks. We think that medicines are facilitators and grounds for this process.

In addition to our thoughts on the effects of COVID-19 in the short term, Sinanović states in his study how much damage the CNS involvement will cause to the brain and that inflammation seems to trigger neurological and neurodegenerative diseases in the long term (Sinanović et al. 2020). It seems that this situation will keep us very busy in the coming years.

In our case, delusions were a psychiatric symptom seen in COVID-19 patients. In most cases, psychotic symptoms may be the result of various pathological mechanisms, including the direct effect of the virus on the CNS, indirect effects (inflammatory reactions, metabolic disorders, hypoxia, etc.), and the negative effects of pharmacological treatments used against the virus. Larger studies should be designed to continue exploring this question.

Acknowledgements: None.

Conflict of interest: None to declare.

Contribution of individual authors:
Hasan Gökçay: data collection, the first draft. Irem Yazici Karabulut: study design. Hasan Belli: approval of the final version.

References

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