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Case Report

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A Neuroleptic malignant syndrome patient associated with COVİD-19: Case report

Abstract

Introduction: Various neurological manifestations associated with COVID-19 have been detected, and its incidence has been increasing. Neurological symptoms in COVID-19 patients have been determined by 36.4% and were common with severe infection. In literature, neuroleptic malignant syndrome relevant to patients with COVID-19 has been reported very few.

Case: A 72-year-old man was admitted to our clinic with an altered mental status, speech disorder, occasionally urinary incontinence. His tremors, diaphoresis, tachycardia, and fever had been developed 3 days before admission. In the neurologic examination, the patient was disoriented and confused, and he had a lead pipe posture. The cerebral magnetic resonance imaging (MRI) and diffusion-weighted MRI was normal. The creatinine kinase (CK) was increased. The diagnosis of the neuroleptic malignant syndrome (NMS) has been confirmed based on DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th edition), Nierenberg, and Levenson criteria. Non-enhanced chest computed tomography (CT) has shown the COVID-19 infiltration. COVID-19 test by PCR (Polymerase Chain Reaction) was positive in the nasopharyngeal swab. After bromocriptine and intravenous hydration treatment, his CK began to downtrend on day 3, and rigidity also improved.

Conclusion: In our case first reported in Turkey, the neuroleptic malignant syndromer are in patients with COVID-19 has been determined. It aimed to raise awareness on this subject.

Keywords: neuroleptic malignant syndrome, COVID-19, pandemic

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Introduction

Neurological manifestations associated with COVID-19 (Corona Virus Disease 2019) have been determined, and studies indicate various central nervous system clinical presentations had.[1,2] Neurological symptoms in COVID-19 patients have been determined by 36.4% and were common with severe infection.[1] There are 3 patients in the literature who have developed neuroleptic malignant syndrome (NMS) with COVID-19.[3,4] We aimed to report an NMS patient associated with COVID-19, therefore, draw attention to this issue.

Case

A 72-year-old man was admitted to theNeurology Department of Cankiri State Hospital with an altered mental status. He had bipolar disorder for 40 years and has been followed regularly for 10 years by a psychiatrist. He had been taking lithium 600 mg and olanzapine 5 mg daily. He had also been taking warfarin due to pulmonary embolism for 8 years. Four days before admission, his altered mental status, speech disorder, and occasionally urinary incontinence have initiated. He had tremors, diaphoresis, tachycardia, and fever for 3 days before admission. In the patient's vital signs, his body temperature was 40.1°C, blood pressure was 140/90 mmHg, and his pulse was 115/min. His oxygen saturation was 95%. In a neurologic examination, the patient was disoriented and confused. Meningeal irritation finding was not determined. He had a rigid posture (leadpipe).

In laboratory examinations, kidney function tests (creatinine: 1.49 mg/dl, blood urea nitrogen: 51 mg/dl), liver function tests (AST:55 U/L, ALT:62 U/L), C-reactive protein (81.9 mg/L), creatinine kinase (7032 U/L) and D-dimer (805µg/L) were evaluated increased. Thyroid function tests were normal. Although albumin level (3.5 g/dl) in the blood was normal, calcium level decreased (8 mg/dl). The lactate dehydrogenase was evaluated increased (477 U/L). Complete blood count (CBC) was normal. The partial thromboplastin time (PTT) (29.3sec), international normalized ratio (2.28), and activated PTT (38.9 sec) were increased. In a complete urinalysis test, there was proteinuria. The lithium levels in the blood were normal (0.6mmol/L). COVID-19 test by PCR (Polymerase Chain Reaction) was positive (nasopharyngeal swab). In cerebrospinal fluid (CSF) examination, protein (38 mg/dl), glucose (78 mg/dl), sodium (138 mEq/L) were evaluated normal. No cells were observed in the direct CSF examination. There was no growth of microorganisms in the CSF culture test. COVID-19 test by PCR in CSF was negative.

In brain computed tomography (CT), cerebral magnetic resonance imaging (MRI), and diffusion-weighted MRI was normal. Non-enhanced chest computed tomography (CT) showed peripheral ground-glass opacities in both lungs, inferior lobes basalis, and upper lobes parenchyma (Figure 1). EEG showed generalized slowing.

The patient was admitted to the intensive care unit (ICU). Favipiravir and intravenous hydration treatment were started. Neuroleptic malignant syndrome was confirmed. Bromocriptine 2.5 mg was started three times daily via a nasogastric tube. Bacterial pneumonia developed in the patient, and moxifloxacin started. His CK began to downtrend on day 3, and rigidity also improved. On day 5, non-invasive mechanical ventilation was started. Then, acute respiratory distress syndrome developed in the patient. He was intubated. None of the sedative drugs were given to the patient. On day 12, the patient died.

Discussion

The diagnosis of the neuroleptic malignant syndrome (NMS)

has been confirmed based on DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th edition), Nierenberg, and Levenson criteria.[5–7] It is suggested that the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) uses angiotensin-converting enzyme 2 (ACE2) receptors. ACE2 has been determined in the substantia nigra, ventricles, middle temporal gyrus, the posterior cingulate cortex, and the olfactory bulb. Therefore, the SARS-CoV-2 virus causes neurological symptoms.[2]

The literature indicated that favipiravir could cause rhabdomyolysis because CK levels were determined higher in patients with influenza that took favipiravir.[4] Higher CK levels are not associated with favipiravir in our case because the patient did not take favipiravir when the NMS diagnosis was determined. Jin et al. have suggested that COVID-19 infection could cause rhabdomyolysis as a late complication.[8] When the NMS diagnosis was confirmed in our patient, he was in the early period of COVID-19. Kajani et al. have reported a case that NMS during the acute infectious phase of COVID-19.[3] Moreover, it is known that olanzapine has a low potential for extrapyramidal adverse effects.[9] Consequently, it may suggest that although olanzapine is a risk factor for NMS in our patient, COVID-19 infection may make the patient develop NMS more suspectable. Further studies are needed for the relation between NMS and COVID-19.

Conflicts of interest

The author declares no conflict of interest.

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