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Coronavirus Pandemic

A rare clinical presentation of COVID 19: opsoclonus-myoclonus ataxia syndrome

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Abstract

Introduction: Coronavirus disease 2019 (COVID-19) can have symptoms like many neurological diseases, and one of the rare forms of these presentations is opsoclonus-myoclonus ataxia syndrome (OMAS). The pathogenesis of OMAS in adults has not been clearly elucidated and OMAS can be fatal.

Case presentation: We present a 71-year-old male patient who was admitted to the emergency department with complaints of involuntary tremor-like movements in his hands, feet and mouth, and speech impediment for three days, and was followed up with COVID-19. The patient was diagnosed with OMAS and clonazepam treatment was started. He died three days later due to respiratory arrest. Our case is the first case diagnosed with COVID-19-associated OMAS in Turkey.

Discussion: OMAS has no definitive treatment. Early diagnosis and initiation of corticosteroids and intravenous immunoglobulin (IVIG) therapy, if necessary, can be life-saving. In COVID-19 patients with unexplained clinical findings, awareness of different and rare diseases and a multidisciplinary approach has vital importance.

Key words: COVID-19; opsoclonus-myoclonus ataxia syndrome; corticosteroids; intravenous immunoglobulin; IVIG.

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Introduction

The coronavirus disease 2019 (COVID-19) caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), was first reported in Wuhan at the end of 2019, and spread to all parts of the world within a short time. Although it affects the respiratory tract in the foreground, it is known to cause multisystemic involvement. One of these systems is the nervous system. It may have different clinical forms of presentations in the nervous system such as encephalitis, Guillain-Barré syndrome, and acute cerebrovascular disease [1].

Opsoclonus is an ocular motility disorder characterized by spontaneous, arrhythmic, conjugated, rapid twitches occurring in all directions of gaze without gaps. Myoclonus is involuntary and short-term movements that occur in one or more muscles and is caused by the central nervous system. Ataxia is the irregular movement that occurs as a result of deterioration in muscle groups involving coordination, balance, and speech disorders. Opsoclonus-myoclonus ataxia syndrome (OMAS) in adults, can be idiopathic or paraneoplastic. Viral infections are one of the paraneoplastic causes [2,3]. Rare cases have also been reported in COVID-19 patients [4].

In this report, we present a case diagnosed with COVID-19-associated OMAS along with a systematic review of literature on OMAS.

Methodology

Ethical Statement

Written informed consent was obtained from the patient for publication of this case report. The patient's age and gender, SARS-CoV-2 test result, course of the COVID-19 disease, myoclonus and ataxia characteristics, other neurological symptoms and findings, research results, treatments for myoclonus and ataxia, and clinical evaluations were used.

Systematic literature review

A systematic review was performed by using the keywords 'COVID, coronavirus, SARS-CoV-2' and 'adult, myoclonus, ataxia, opsoclonus, and opsoclonus-myoclonus ataxia syndrome' in PubMed, Google

Scholar, Cochrane and Scopus databases between 31 December 2019 and 1 January 2022. The search was limited to English language publications in peer-reviewed journals. Case reports and case series were included in the study if they included the description of patients with SARS-CoV-2 infection and the description of myoclonus or ataxia. Other types of publications were reviewed for data including new cases. Publications and cases attributable to a non-infectious etiology of myoclonus or ataxia were excluded. All the cases are summarized in Table 1 [4-15].

Case report

A 71-year-old male patient was admitted to the emergency service with involuntary tremor-like movements in his hands, feet, and mouth for three days, preventing him from speaking. The patient had no known comorbidities except hypertension and had a history of levofloxacin use for pneumonia in the previous week. It was learned that the patient's wife died 40 days ago due to COVID-19 infection. The COVID-19 reverse transcriptase polymerase chain reaction (RT-PCR) test of the patient was negative twice in his previous hospital admissions. The COVID-

Table 1. Demographic and epidemiological features, diagnosis, treatment, and outcomes of opsoclonus-myoclonus ataxia syndrome (OMAS) related to Coronavirus disease 2019 (COVID-19) patients reported in the literature.

	Case	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7
Reference		[4]	[4]	[4]	[4]	[4]	[4]	[4]
Age/gender	71/ male	51/male	54/male	52/male	42/female	44/male	52/male	39/male
Symptoms of COVİD-19	Dyspnea	Sore throat Back pain Anorexia and mild dyspnea	Malaise Fever Myalgia Coughs Dyspnea	Dry cough Fever Headaches	Fever Myalgia Coughs	Fever Chills 3 days later Seizure	Fever Myalgia Cough	Fever cough Myalgia Nausea Vomiting 10 days later seizure
Neurological symptoms	Myoclonus Opsoclonus Ataxia Hyperekplexy	Myoclonus Opsoclonus Ataxia Voice tremor	Myoclonus Ataxia	Myoclonus Ataxia Voice tremor	Myoclonus Ataxia Voice tremor	Myoclonus Opsoclonus Ataxia Voice tremor	Myoclonus Ataxia Voice tremor	Myoclonus Opsoclonus Ataxia Voice tremor
Neurological symptom onset interval since initial COVID-19 symptoms	7 days	14 days	4 days	16 days	10 days	3 days	21 days	10 days
Laboratory finding		No abnormalities	WBC = 11,500/µL, lymphocyte = 2300/µL, ESR = 45 mm/h and CRP = 2+ Normal serum electrolyte	WBC = 6600/µL Lymph = 1700/µL ESR = 30 mm/h and CRP = 2+ Normal serum electrolytes	No abnormalities	No data	No data	AST = 61 U/L ALT = 69 U/L ESR = 58 mm/h CRP = 75.4 mg/L
CSF analysis	Soidum: 156,7 mEq/L Chlorine: 133 mEq/L CSF COVİD PCR (-) Viral and bacterial pathogens pcr (-) Autoimmune encephalitis panel in serum and CSF (-)	No data	CSF analysis normal	No data	No data	CSF analysis normal	Normal CSF analysis. CSF COVİD PCR (-) Viral and bacterial pathogens pcr (-) Autoimmune encephalitis panel inserum and CSF (-) Oligoclonal bands (-)	No data
COVID-19 laboratory Tests PCR/Antibody IgG-IgM	(+)/none	(-)/none	(-)/(+)	(+)/no data	No data	(+)/no data	(+)/no data	(+)/no data
Brain imaging findings CT/MR	Normal CT Normal MRI	Normal CT	Normal MRI	Normal MRI	No data	Normal MRI	Normal MRI	Normal CT
Lung imaging findings		Few peripheral patchy ground-glass opacities	Patchy peripheral ground glass opacities and consolidations	Patchy peripheral ground glass opacities and consolidations	No data	Patchy peripheral opacities	Patchy peripheral opacities	Patchy predominantly peripheral ground glass opacities and consolidations
Treatments	Clonazepam	Clonazepam Levetiracetam IVIG	Levetiracetam sodium Valproate IVIG	Sodium valproate clonazepam	Sodium valproate clonazepam	Sodium valproate clonazepam IVIG	Clonazepam IVIG	Levetiracetam Sodium valproate Clonazepam IVIG Dexamethasone
Follow up of patients	Exitus in 11 days	Complete recovery after 4 weeks	Partial recovery after one week	Partial recovery after 2 months	No data	Complete recovery after 2 months	Significant improvement after 4 weeks	No data

19 RT-PCR test of the patient was positive in our hospital and the thorax tomography of the patient was found to be compatible with COVID-19 infection. His body temperature, blood pressure, and respiratory rate were normal but he had shortness of breath and needed 5 L/min oxygen. During neurological examination, his consciousness, cooperation, and orientation were normal. The verbal exit of the patient was in the form of single words. Bilateral pupils were isochoric. There were bilateral jerking movements in his eyes that were in all directions. He had no nystagmus. There was no facial asymmetry. There were prominent myoclonic movements in the facial muscles and upper extremities. There was a normal response in bilateral deep tendon reflexes. There was no Babinski sign. His four extremities were spontaneously active. There was no muscle weakness. There were no other pathological physical examination findings. No signs of acute infarction or hemorrhage were found on magnetic resonance imaging (MRI) and brain computed tomography (CT) of the patient. The laboratory findings are summarized in Table 2. It was thought that the patient's findings might be associated with metabolic and electrolyte disorders.

The patient was hospitalized and he was given favipiravir prednisolone 80 gm/day, enoxaparin 6000 units twice a day, and cefepime 2 gm twice a day treatment. No bacterial growth was detected in his blood culture and urine culture. There was no need for oxygen support on the 5th day of his treatment.

Table 1 (continued). Demographic and epidemiological features, diagnosis, treatment, and outcomes of opsoclonus-myoclonus ataxia syndrome (OMAS) related to Coronavirus disease 2019 **(**COVID-19) patients reported in the literature.

	Patient 8	Patient 9	Patient 10	Patient 11	Patient 12	Patient 13	Patient 14
Reference	[5]	[6]	[7]	[8]	[9]	[10]	[11]
Age/gender	Middle age/male	32/male	58/ female	57/female	50/male	57/male	63/male
Symptoms of COVİD	Recovery from COVID-19 lung infection no symptoms	Cough Fever Weakness Loss of appetite	Fever	No symptoms	Fever Tachypne hypoxaemi	nausea, fever, diarrhea, myalgias	fever, diarrhea, cough, malaise, sore throat
Neurological symptoms	Myoclonus Opsoclonus Ataxia	Myoclonus Opsoclonus Ataxia	Myoclonus Opsoclonus Ataxia Dysarthria	Speech changes persistent jerking movements non-rhythmic truncal titubation hyperekplexia, pronounced limb and truncal ataxia, intention tremor dysdiadochokinesis	Abnormal eye movements and jerks of the non-paretic left upper and lower limbs confused distressed and unable to speak or follow commands Opsoclonus limb myoclonus	Tremors myoclonus spontaneous horizontal and vertical oscillations that did not seem to have intersaccadic interval, consistent with opsoclonus.	Opsoclonus Myoclonus Truncal ataxi
Neurological symptom onset interval since initial COVID-19 symptoms	21 days	12 days	No data	About 2 weeks	7 days	10 days	23 days
Laboratory finding	No abnormalities	No Data	Mild leukocytosis CRP = 48 mg/L	the lactic acid 3.8, CRP 0.75 (normal < 0.5 mg/dL).	CRP 230 mg/L lymphocyte 0.7 × 10^9/L.	No data	No abnormalities
CSF analysis	CSF analysis normal Autoimmune encephalitis panel in serum and CSF (-)	No data	CSF analysis normal Autoimmune encephalitis panel in serum and CSF (-)	CSF neuron-specific enolase 34 ng/dL (normal <15 ng/dL) infectious and autoimmune encephalitis panel in serum and CSF (-)	cerebrospinal fluid (CSF) was acellular, with protein = 0.566 g/L (0.15–0.45 serum LGI-1 antibody (+) CSF LGI-1 and other autoantibodies (-)	No data	CSF analysis normal Autoimmune encephalitis panel in serum and CSF (-)
COVID-19 laboratory Tests PCR/Antibody IgG-IgM	(-)/no data	(-)/(+)	(-)/(+)	(-)/(+) (husband and son PCR +)	(+)/no data	(+)/no data	(+)/(+)
Brain imaging findings CT/MR	Normal MRI	Normal MRI	Normal MRI	MRI w/wo contrast, modest caudate nuclei hyperintensities but without restricted diffusion	Brain MRI showed changes in keeping with prior MCA infarction, with Wallerian degeneration	Normal MRI	Normal MRI
Lung imaging findings	No data	Chest x-ray consistent with viral pneumonia	Ground glass opacities	Chest x-ray patchy opacities	CT pulmonary angiogram showed bilateral consolidation and no pulmonary embolism	CT bilateral ground glass opacities in his lungs characteristic of COVID-19	Chest x-ray normal
Treatments	Methylprednisolone Sodium valproate Levetiracetam Clonazepam	Methylprednisolone Sodium valproate Clonazepam	Clonazepam Levetiracetam IVIG Corticosteroids	Corticosteroids Clonazepam Rituximab	levetiracetam clonazepam methylprednisolone	Clonazepam IVIG Corticosteroids	IVIG Corticosteroids
Follow up of patients	Recovery after 1 week	Significant improvement after 24 days	No Data	4 weeks	8 weeks	No data	4 weeks

Table 1 (continued). Demographic and epidemiological features, diagnosis, treatment, and outcomes of opsoclonus-myoclonus ataxia syndrome (OMAS) related to Coronavirus disease 2019 **(**COVID-19) patients reported in the literature.

	Patient 15	Patient 16	Patient 17	Patient 18	Patient 19	Patient 20
Reference	[12]	[12]	[13]	[14]	[15]	[15]
Age/gender	49/male	62/male	78/female	83/male	50/male	80/male
Symptoms of COVİD-19	Fever sweating	Mild symptoms	Weakness Upper respiratory tract symptoms	No data	Asymptomatic	Fever Cough dsypnea
Neurological symptoms	Myoclonus Ataxia Voice tremor	Myoclonus Opsoclonus Ataxia	Myoclonus Opsoclonus Ataxia	Opsoclonus Myoclonus of the four limbs, trunk Ataxic dysarthria Confusion	Myoclonus Opsoclonus Ataxia	Myoclonus Opsoclonus Ataxia
Neurological symptoms at interval since initial COVID-19symptoms	11 days	11 days	14 days	10 days	10 days	10 days
Laboratory finding	No data	No data	No abnormalities	Blood tests excluded other infectious, metabolic or auto-immune diseases	Mild leukocytosis, thrombocytosis, İncreased LDH, CK, CRP	Mild leukocytosis, thrombocytopenia, increased CRP
CSF analysis	CSF analysis normal Autoimmune and infectious encephalitis panel in serum and CSF (-)	CSF analysis normal Autoimmune and infectious encephalitis panel in serum and CSF (-)	Only CSF Total protein 55 mg/dl (15-45 mg/dl) Infectious and Autoimmune encephalitis panel in serum and CSF (-)	CSF analysis normal Autoimmune and infectious encephalitis panel in serum and CSF (-)	No data Autoimmune and infectious encephalitis panel in serum (-)	No data
COVID-19 laboratory Tests PCR/Antibody IgG-IgM	(-)/(+)	(+)/no data	(+)/no data (+)	No data/(+)	(+)/no data	(+)/no data
Brain imaging findings CT/MR	Normal MRI/CT	Normal MRI	Normal CT/MRI	Normal MRI	No data	CT unremarkable
Lung imaging findings	No data	CT lung normal	CT Unremarkable	Thoracic CT minimal form of COVID-19 pneumonitis	X-ray bilateral lung reticular interstitial opacification	CT bilateral ground-glass opacities compatible with interstitial lung disease
Treatments	Corticosteroids Clonazepam biperiden levetiracetam	IVIG Corticosteroids	Levetiracetam IVIG Corticosteroids	IVIG Corticosteroids	Corticosteroids	Corticosteroids
Follow up of patients	5 weeks	4 weeks	10 days	Few days	1 week	1 week

OMAS: opsoclonus-myoclonus ataxia syndrome; WBC: white blood count; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; AST: aspartate transaminase; ALT: alanine transaminase; CSF: cerebrospinal fluid; PCR: polymerase chain reaction; CT: computed tomography; MR: magnetic resonance imagining; IVIG: intravenous immunoglobulin; LDH: lactate dehydrogenase; CK: creatine kinase.

Table 2. Laboratory findings.

Laboratory tests / unit / normal ranges	Day 1	Day 3	Day 5	Day 8	Day 11
Urea (mg/dL) / (19-49)	71	129	111	122	141
Creatine (mg/dL) / (0.7-1.3)	1.33	2.27	1.26	1.44	2.56
AST (U/L) / (< 35)	134	664	215	89	80
ALT (U/L) / (< 50)	255	282	394	272	209
LDH (U/L) / (120-246)	325	1329	573	604	668
Sodium (mEq/L) / (132-146)	152	155	145	148	150
Potassium (mEq/L) / (3.5-5.5)	4.7	4.1	4.4	3.8	4.5
Leukocyte count (x109/L) / (3.6-10.5)	9.54	13.98	19.48	15.01	19.75
Lymphocyte count (x109/L) / (1.1-4)	0.79	0.5	0.91	0.76	0.79
Neutrophil count (x109/L) / (1.5-7.7)	8.08	12.96	17.48	13.19	17.91
Hemoglobin (g/dL) / (12.5-17.5)	15.6	14.0	14.9	14.2	11.4
Platelet count (x109/L) / (160-400)	202	269	302	200	287
CRP (g/L) / (0-0.005)	0.0467	0.0421	0.0024	0.0013	-
Procalcitonin (µg/L) / (< 0.16)	0.14	11.0	1.10	0.3	-
D-dimer (mg/dL) / (< 0.55)	20.8	4.3	5.3	4.2	5.3
Ferritin (µg/L) / (22-232)	1089	1178	969	1063	1056

AST: aspartate transaminase; ALT: alanine transaminase; LDH: lactate dehydrogenase; CK: creatine kinase; CRP: C-reactive protein.

Electrolyte and metabolic disorders were corrected in accordance with the recommendations of the internal medicine department. Improvement was observed in his laboratory findings. Favipiravir treatment was discontinued on the 5thday. Cefepim treatment was completed on the 7thday. Prednol treatment was reduced to 40 gm/day on the 5th day.

Although there was no need for oxygen and metabolic and electrolyte disorders were corrected on the 5th day of his treatment, tremors, myoclonus in the lips, lower and upper extremities, opsoclonus in the eyes, and hyperlexia continued. A lumbar puncture was performed on the patient. Cerebrospinal fluid (CSF) pressure was normal and appearance was clear. The patient's CSF laboratory findings are summarized in Table 3. COVID-19 RT-PCR result was found to be negative. The FTD (Fast-Tract Diagnostics, Siemens Healthcare, Erlangen, Germany) Viral Meningitis was used in the Rotor-GeneQ Real-Time PCR (Qiagen, Hilden, Germany) system. The pathogens investigated in CSF (herpes simplex virus 1-2, mumps virus, varicella-zoster virus, enterovirus, human parechovirus) were detected negative. No growth was observed in the CSF culture. Anti-neuronal antibodies and limbic encephalitis antibodies were examined in the cerebrospinal fluid and blood was negative. Central nervous system infection was not considered in the CSF findings of the patient. Clonazepam treatment was initiated for the patient based on the recommendations of the neurology consultant. Three days later, the patient was admitted to the intensive care unit with a sudden decrease in saturation during his follow-up. He was intubated at the 4th hour of his hospitalization in the intensive care unit, and the patient died of respiratory arrest.

Discussion

Our case is the first case diagnosed with COVID-19-associated OMAS in Turkey. OMAS in adults can be idiopathic, secondary to viral infections, or mostly paraneoplastic. A study on its pathogenesis in children revealed that antibodies, that formed against antigens in the central nervous system target Purkinje cells in the cerebellum [16]. In the pathogenesis of OMAS in adults, antibodies such as anti-RI antibodies, anti-Hu antibodies, N-methyl-D-aspartate receptor (NMDA-R), gamma-aminobutyric acid type A (GABA-A)-type B (GABA-B) receptors, dipeptidyl-peptidase-like protein-6 (DPPX), glutamic acid decarboxylase (GAD), human natural killer 1 (HNK-1), and myelin-associated glycoprotein (MAG) have been identified; however, none are specific or special to this disease [17-19]. Peripheral nervous system diseases such as ischemic stroke, cerebral hemorrhage, encephalopathy, seizures, Guillain Barre syndrome, and many neurological system involvements such as depression and personality changes related to COVID-19 have been reported, but only a few COVID-19-related OMAS cases have been reported globally [20]. Among the cases reported in the literature, 16 were male and 4 were female. Our case is also male, compatible with the literature. When we reviewed the literature, the mean age was 40 years in patients with idiopathic OMAS and 55 years in patients with paraneoplastic OMAS [17,21]. The mean age of patients with OMAS related to COVID-19, as shown in Table 1, is 55 (minimum 32 years, maximum 83 years). Our case was 71 years old and relatively old.

As in our case, in 17 patients, COVID 19 infection was confirmed by RT-PCR/antibody IgG/IgM tests. In addition, 16 patients had symptoms of COVID-19, similar to our patient. Neurological findings of reported patients started, on an average, after 11.5 days (minimum 3 days, maximum 23 days). In our patient, the findings started on the 7thday, similar to the patients in the literature. Common symptoms were opsoclonus, myoclonus, ataxia, and some patients had tremor, which also played an important role in diagnosing the disease. There are no laboratory or other tests that confirm OMAS, and it is a clinical diagnosis. The diagnosis of OMAS is made by excluding other diseases with similar neurological findings. The patient should have MRI with gadolinium, electroencephalography (EEG), and some laboratory results to exclude toxic-metabolic encephalopathy and, especially, hyperosmolar coma, liver disease, and poisonings. In addition, the

Table 3. Cerebrospinal fluid (CSF) findings.

CSF	Results	Unit	Reference Range
Potassium	3.1	mEq/L	< 70% serum level
Sodium	**156.7**	**mEq/L**	**132-146**
Chlorine	**133**	**mEq/L**	**118-132**
Urea	48	mg/dL	
Creatinine	< 3.0	mg/dL	
Glucose	62	mg/dL	40-70
Lactate dehydrogenase	29	U/L	
Total protein	326.26	mg/L	150-400

medications taken by the patient should be reviewed. If these are negative, the patient should be tested for viral infections, including HIV infection. If central nervous system (CNS) infection is suspected, CSF analysis should be performed. CSF findings are typically normal in cases of OMAS, but rarely lymphocytic pleocytosis can be detected. Brain MRI and EEG are also usually normal [17,22]. While the MRI results of 16 patients in the literature (Table 1) were normal, no specific finding was detected in the MRI findings of the other patients. Brain MRI and CT results of our patient were reported as normal in accordance with the literature. In addition, when we examined the cases in the literature, CSF findings were normal in 9 patients, the protein was increased in 2 patients and enolase was high in 1 patient. While there were no findings of infectious and autoimmune encephalitis in serum and CSF findings in 10 patients, only in 1 patient serum LGI-1 antibodies were high, and LGI-1 antibodies and other antibodies in CSF were negative. In our patient, RT-PCR to detect COVID-19, viral and bacterial pathogens in CSF were negative. The autoimmune encephalitis panel in serum and CSF were negative, supporting literature.

Some research has been done on treatment in children; however, an accepted standard treatment regimen has not been reported. Nevertheless, glucocorticoids and intravenous immunoglobulin (IVIG) administration are widely used in first-line therapy. In a study on 53 children with neuroblastoma-associated OMAS who received chemotherapy and prednisolone depending on their risk, it was shown that a better clinical response was obtained in the group with IVIG added to the treatment for 12 months [23]. On the other hand, in adults with paraneoplastic OMAS, neurological symptoms can be expected to resolve with the treatment of the underlying neoplasm [21]. Response to immunotherapy may be less likely in adults, compared to children. However, responses have been reported with corticosteroids, cyclophosphamide, and IVIG in observational studies. In two cases, after failing to respond to immunotherapy, treatment response was obtained with high doses of clonazepam (8 to 12 mg per day) [24]. Based on all these data, as shown in Table 1, antiepileptic drugs were prescribed in 15 patients who developed OMAS due to COVID-19, steroids were given to 14 patients, IVIG was given to 11 patients, and rituximab was given to 1 patient. Our patient was initiated with corticosteroid treatment from the first day and clonazepam treatment from the fifth day of hospitalization. In the literature, patients recovered after an average of 26 days (minimum 4 days, maximum 56 days). Unfortunately, our patient died on the 11th day, while there was no death reported in the literature.

Conclusions

There is no definitive treatment for OMAS. Early diagnosis and initiation of corticosteroids and IVIG therapy, if necessary, can be life-saving. Awareness of different and rare diseases and a multidisciplinary approach has vital importance in the case of COVID-19 patients with unexplained clinical findings.

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