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CASE REPORT

Immune-mediated cerebellitis following SARS-CoV-2 infection—a case report and review of the literature

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Abstract

The coronavirus disease 2019 (COVID-19) can be associated with a wide variety of neurological manifestations. Some of these manifestations might result from the ongoing systemic inflammatory state, but the pathophysiology of specific neurologic involvement is still unclear. In this article, we report a patient who developed an isolated cerebellar syndrome 9 weeks after an episode of COVID-19. The reverse-transcriptase polymerase chain reaction (RT-PCR) for SARS-CoV-2 was positive on cerebrospinal fluid (CSF). A post-infectious–autoimmune-cerebellitis following COVID-19 was suspected, and the patient was treated with corticosteroids, leading to a complete recovery within a few weeks. We review the other cases of COVID-19-associated cerebellar syndrome reported so far and discuss the potential pathophysiological mechanisms underlying this neurologic manifestation.

Keywords Coronavirus · COVID-19 · SARS-CoV-2 · Neurological manifestation · Cerebellitis · Post-infectious

Introduction

Since its description from Wuhan (China) in December 2019, the coronavirus disease 2019 (COVID-19) has spread around the world, causing over six million deaths so far. COVID-19 can be associated with a wide variety of neurological manifestations, ranging from non-specific headache, dizziness, confusion, ageusia, or anosmia to more severe and specific involvement of either central or peripheral nervous system, including myelitis, encephalitis, or demyelinating inflammatory polyneuropathy (Bougakov et al. 2021; Harapan and Yoo 2021; Niazkar et al. 2020). While it is postulated that non-specific manifestations result from the ongoing systemic inflammatory state, the pathophysiology of specific neurologic involvement is still unclear (Jha et al. 2021; Xu et al. 2021). In this paper, we report a case of COVID-19-associated cerebellitis, and we review the other cases reported so far in the literature and discuss the potential pathophysiological mechanisms underlying this neurologic manifestation.

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Case presentation

In December 2020, a 55-year-old woman developed headache and asthenia for 7 days then cerebellar ataxia and dysarthria for 3 days before being admitted in this hospital. She had suffered from a mild episode of reverse-transcriptase polymerase chain reaction (RT-PCR)-proven COVID-19 9 weeks before. At patient's admission, RT-PCR for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on nasopharyngeal swab was negative, and she did not report any respiratory symptom. General blood analyses, including measure of C-reactive protein, were unremarkable (see Table 1). Cerebrospinal fluid (CSF) analysis revealed an increased white blood cell count with predominant lymphocytic pattern (85 cells per microliter, 99% lymphocytes) but normal glucose and protein levels (73 mg/dL and 331 mg/L, respectively). PCR multiplex assay for Herpes simplex viruses 1 and 2, *Cytomegalovirus*, Varicella-zoster virus, Human herpes virus 6, Epstein–Barr virus, *Enterovirus*, and Human parechovirus on CSF were negative as were direct examination and bacterial, mycobacterial, and fungal cultures. By contrast, RT-PCR for SARS-CoV-2 was positive on CSF. Antibodies directed at

Table 1 Laboratory results and cerebrospinal fluid findings

Laboratory results		
Test	**Result**	**Normal range**
Nasopharyngeal RT-PCR for SARS-CoV-2	Positive 10 weeks before admission Negative on days 0, 4, 8, 11, 16, 18, 25, 32, and 36 after admission	Negative
Hemoglobin (g/dL)	14.5	11.7–14.9
White blood cell count (10^3/mm^3)	7.33 36% lymphocytes	3.90–10.20 20–44%
Thrombocyte count (10^3/mm^3)	347	150–370
D-dimers (µg/L)	< 190	< 500
C-reactive protein (mg/L)	< 0.5	0.0–5.0
LDH (U/L)	225	125–220
Coxsackies and Echovirus serology	Negative	Negative
Cytomegalovirus serology	IgM negative IgG 136 UI/mL	Negative Positive > = 14
Epstein—Barr virus serology	IgM negative IgG > 750 UA/mL	Negative Positive > = 20
Herpes simplex virus serology	IgM negative IgG > 30.0 UA/mL	Negative Positive > = 1.0
Borrelia burgdorferi serology	IgM negative IgG < 10.0 UA/mL	Negative Positive > = 15
Mycoplasma pneumoniae serology	IgM negative IgG < 9 UA/mL	Negative Positive > = 11
Hepatitis B virus serology	Positive HBs, negative HBc Compatible with vaccination	
Hepatitis C virus serology	Negative	Negative
Human immunodeficiency virus 1 and 2 serology	Negative	Negative
Brucella serology	Negative	Negative
Treponema pallidum VDRL and TPHA	Negative	Negative
West Nile virus serology	IgM negative IgG negative	Negative
Tick-borne encephalitis virus serology	IgM negative IgG negative	Negative
Measle virus serology	IgM negative IgG > 300.0 UI/mL	Negative Positive > = 16.5
Rubella virus serology	IgM negative IgG 11 UI/mL	Negative Positive > = 10
Toxoplasma gondii serology	IgM negative IgG 9.4 UI/mL	Negative Positive > = 8.8
Mumps serology	IgM negative IgG 151 UA/mL	Negative Positive > = 11
SARS-CoV-2 serology	IgG 34 (local unit)	Positive > = 15
Antinuclear antibodies	Negative	Negative
Anti-neutrophil antibodies	Negative	Negative
Antibodies against neuronal cell surface antigens (anti-NMDA receptor, – LGI1, – CASPR2, – GABAb receptor, – AMPA1 receptor, – AMPA2 receptor, – DPPX)	Negative	Negative
Antibodies against neuronal intracellular antigens (– Hu, – Ri, – Yo, – Ma2/Ta, – CV2 (CRMP5), – amphiphysin, – Recoverine, – SOX1, – titine, – ZIC4, – Tr)	Negative	Negative
Anti-GAD65 and GAD67 antibodies	Negative	Negative
Anti-ganglioside antibodies (Anti-GM1, -GM2, -GM3, -GD1A, -GD1B, -GD3, -GQ1B, -GT1A, -GT1B, -sulfatide)	Negative	Negative
Anti-MOG and anti-AQP4 antibodies	Negative	Negative

Table 1 (continued)

Laboratory results		
Test	**Result**	**Normal range**
Anti-gliadin antibodies	IgG < 10.0 U/mL	Positive > = 10
Anti-transglutaminase antibodies	IgA < 10 U/mL	Positive > = 10
Vitamin E	9.81 mg/L	8.60–19.24
CSF findings		
4 days after the admission (before treatment)		
Test	**Result**	**Normal range**
CSF white blood cell count (cells/mm^3)	85 99% lymphocytes	0–5
Glucose (mg/dL)	73	45–75
Protein count (mg/L)	331	150–450
Multiplex PCR assay for *Escherichia coli* K1, *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, *Streptococcus agalactiae*, *Streptococcus pneumoniae*, *Cytomegalovirus*, *Enterovirus*, Herpes simplex virus 1 and 2, Varicella-zoster virus, Human herpes virus 6, Human parechovirus, *Cryptococcus neoformans*/gattii	Negative	Negative
RT-PCR for SARS-CoV-2 in CSF	Positive 36.2 cycles	
12 days after the admission (before treatment)		
Test	**Result**	**Normal range**
White blood cell count (cells/mm^3)	50 97% lymphocytes	0–5
Glucose (mg/dL)	72	45–75
Protein level (mg/L)	224	150–450
Isoelectric focusing of proteins	Presence of many IgG oligoclonal bands in CSF	
32 days after the admission (after treatment)		
Test	**Result**	**Normal range**
CSF cell number (/mm^3)	34 82% lymphocytes, 18% monocytes	0–5
Glucose (mg/dL)	94	45–75
Protein level (mg/L)	235	150–450
Isoelectric focusing of proteins	No oligoclonal bands in CSF	
3 months later		
Test	**Result**	**Normal range**
White blood cell count (cells/mm^3)	< 5	0–5
Glucose (mg/dL)	68	45–75
Protein level (mg/L)	257	150–450
Isoelectric focusing of proteins	No oligoclonal bands in CSF	

neuronal cell surface antigens were negative in both serum and CSF. Brain imaging with both computed tomography and magnetic resonance imaging (MRI) were strictly normal. Whole-body [^{18}F]-fluorodeoxyglucose positron emission tomography (^{18}FDG-PET) did not find any lesion suspected to be malignant.

In the following week, the ataxia severely worsened. A second CSF sample revealed a persistently increased white blood cell count (50 cells/mm^3, 97% lymphocytes) and oligoclonal bands (OCBs). RT-PCR for SARS-CoV-2 was no longer positive in the CSF and remained negative on nasopharyngeal swab (performed every week for all hospitalized patients according to the sanitary measures recommended in the institution at that time).

A post-infectious cerebellitis following COVID-19 was suspected. The patient was treated with intravenous pulses of methylprednisolone (1 g per day for 5 days), followed by an oral taper over 6 weeks. No antiviral treatment was administered since none of these had been proven safe and effective for COVID-19 at that time. Three days after corticosteroid initiation, the patient started to improve clinically. She was discharged to a rehabilitation center where she gradually

improved. CSF analysis was controlled 2 months later and was strictly normal (normal white blood cell count, disappearance of OCBs).

One year later, the patient had fully recovered. She has been fully vaccinated against SARS-CoV-2 without any notable side effect. Neurologic symptoms never recurred.

Discussion

To the best of our knowledge, this is the 20th case of COVID-19-related isolated cerebellar syndrome reported so far, the second one with a RT-PCR for SARS-CoV-2 positive in the CSF (see Table 2). According to these reports, COVID-19-related cerebellar syndrome usually progresses over a few days and can affect patients of any age. The CSF can be normal or show signs of inflammation (increased WBC count or protein level). Brain MRI is usually normal or reveals T2-weighted hyperintense lesions in the cerebellum. Most patients were treated with intravenous corticosteroids, sometimes associated with intravenous immunoglobulin or antiviral therapy (see Table 2). One patient was treated with antiviral therapy alone, and another one did not receive any treatment (Povlow and Auerbach 2021). All of them showed clinical improvement within days to weeks, and, as for our patient, the prognosis is usually excellent. In the presented case, the delay between COVID-19 symptoms and neurologic manifestations was particularly long (9 weeks; from a few days to 5 weeks in reported cases).

SARS-CoV-2 infection was repeatedly associated with a wide range of neurologic manifestations. Besides non-specific signs reflecting encephalopathy, SARS-CoV-2 may also be responsible for specific damage to both central and peripheral nervous systems, and the pathophysiologic mechanisms of which are still debated. Two main processes have been hypothesized: direct damage by viral invasion of the nervous system, or an immune-mediated mechanism (Jha et al. 2021; Koralnik and Tyler 2020; Xu et al. 2021). Concerning viral invasion, it is currently accepted that the virus is able to enter the central nervous system (CNS) either by penetrating through the cribriform plate of the ethmoid bone, or by crossing the blood brain barrier (BBB), made permeable by the inflammatory state or the migration of infected activated immune cells into the CNS (Abboud et al. 2020; Jafari Khaljiri et al. 2021; Jha et al. 2021; Xu et al. 2021). The virus can subsequently infect glial cells and neurons after binding to ACE2 receptors expressed on their surface (Harapan and Yoo 2021; Xu et al. 2021).

The other proposed mechanism hypothesized to underlie SARS-CoV-2-related neurologic manifestations is an immune-mediated phenomenon triggered by molecular mimicry leading to a cross-reactivity towards viral and host antigens with similar shared epitopes. Supporting this hypothesis, various anti-neuronal antibodies were found in the serum or CSF of patients hospitalized with neurologic manifestations following SARS-CoV-2, including encephalitis or acute inflammatory demyelinating polyneuropathy (Payus et al. 2022). Our report suggests that the two hypotheses, direct invasion and immune-mediated, are not necessarily exclusive. Indeed, while a positive SARS-CoV-2 RT-PCR in the CSF suggests a viral invasion of the CNS, the temporal course of the disease as well as its spectacular response to corticosteroids is typical of an immune-mediated mechanism. Therefore, a third possibility could be that the virus first infected brain cells causing mild subclinical cellular damages which resulted in the release of neuronal antigens into the extracellular space and systemic circulation, thereby facilitating their presentation to lymphocytes. This phenomenon has already been proposed as the trigger of several autoimmune diseases such as systemic lupus erythematosus (Quaglia et al. 2021). Finally, a fourth mechanism proposed in the literature is a massive systemic release of cytokines, called "cytokine storm," which alters the BBB and let cytokines and inflammatory cells enter the central nervous system. This hypothesis, which was suggested following the discovery of high levels of proinflammatory cytokines in the serum and CSF of patients suffering from SARS-CoV-2-related neurologic manifestations, probably accounts for encephalitis or other non-specific neurological manifestations such as confusion, headaches, and altered consciousness (Payus et al. 2022).

Although we believe that our hypothesis is of interest, we must acknowledge the intrinsic limitations of single-case reporting, including the need for further studies to support our hypotheses and demonstrate a link between CNS viral invasion, neuronal antigen release, and the development of an immune-mediated phenomenon.

In conclusion, the presented case provides new perspectives on the potential pathophysiological mechanism underlying CNS damage induced by SARS-CoV-2 infection.

Table 2 Characteristics of the reported COVID-19-related cerebellar syndromes (references: see supplementary Appendix 1)

Authors	Age (years), sex	Temporal progression of the cerebellar syndrome	Concomitant signs/ symptoms (in addition to the cerebellar syndrome)	Lesions visualized through brain MRI	CSF analysis abnormalities	RT-PCR assay for SARS-CoV-2 on NP swab and CSF	Lungs involvement	Presence of antibodies directed against neuronal surface or intracellular antigens	Treatment	Outcome
Plumacker et al. (present case)	55, F[a]	Over 3 days	Headache, asthenia	Normal	Increased WBC[a] count (85/mm^3) with lymphocytic pattern	Positive on NP[a] swab 8 weeks before admission but negative at admission. Positive on CSF[a]	Unknown	No	Intravenous methylprednisolone 1 g daily for 5 days then oral taper	Complete recovery after 1 year
Fadakar et al	47, M[a]	Over 7 days	Headache	T2/FLAIR[a] hyperintense, gadolinium-enhancing cerebellar lesions involving both hemispheres and the vermis	Increased opening pressure (25 cmH2) Increased WBC count (10/mm^3) with predominant lymphocytic pattern (80%) Elevated protein level (58 mg/dL)	Positive on both NP swab and CSF	No	No	Lopinavir/ritonavir 400/100 mg twice daily for 14 days	Major improvement; recovery almost complete after 1 month
Povlow and Auerbach	30, M	Over 2 days		Normal	Increased WBC count (7/mm^3) with lymphocytic pattern	Positive on NP swab Not performed on CSF	Yes, concomitant and asymptomatic (found with chest CT[a])	No	No pharmaceutical treatment Physical therapy	Incomplete improvement after 10 days
Moreno-Escobar et al	24, M	Over 9 days	Headache, photophobia, myalgias, fever, mild encephalopathy	T2/FLAIR hyperintense cerebellar lesions, nodular leptomeningeal enhancement and restricted diffusion along the bilateral cerebellar folia	Increased WBC count (8/mm^3), with predominant lymphocytic pattern	Positive on NP swab 21 days before admission. Negative on CSF	No	No	Intravenous methylprednisolone 1 g daily for 5 days	Complete resolution over a few days

Table 2 (continued)

Authors	Age (years), sex	Temporal progression of the cerebellar syndrome	Concomitant signs/symptoms (in addition to the cerebellar syndrome)	Lesions visualized through brain MRI	CSF analysis abnormalities	RT-PCR assay for SARS-CoV-2 on NP swab and CSF	Lungs involvement	Presence of antibodies directed against neuronal surface or intracellular antigens	Treatment	Outcome
Malayala et al	63, M	Over a few days	Low-grade fever and confusion	T2/FLAIR hyperintense bilateral cerebellar white matter lesions	Normal	Positive on NP swab 5 days before admission Not performed on CSF	Yes, concomitant and symptomatic	Not performed	Systemic corticosteroids and intravenous remdesivir (unknown duration and dosing)	Improvement after 4 days of treatment
Emekli et al	54, M	Over 1 week	Myalgias	T2/FLAIR hyperintense bilateral cerebellar cortical lesions and mild pial contrast enhancement	Increased WBC count (20/mm^3), with predominant lymphocytic pattern	Positive on NP swab 4 weeks before admission. Negative on CSF	Yes, concomitant and asymptomatic (found with chest CT)	Anti-GAD[a] antibodies	Intravenous methylprednisolone 1 g daily for 10 days then oral taper Intravenous immunoglobulin 0.4 g/kg/day during 5 days, monthly for 3 months	Rapid improvement over a few days; recovery almost complete after 3 months
Asan et al	44, M	Over a few days		Normal	Normal	Positive on NP swab at admission. Negative on CSF	No	Anti-GFAP[a] antibodies	Intravenous methylprednisolone 1 g daily for 5 days	Rapid improvement; complete recovery after 3 months
Grimaldi et al	72, M	Over a few days	Fever	Normal	Elevated total protein level (49 mg/dL)	Positive on NP swab at admission. Negative on CSF	Yes, a few days before and asymptomatic (found with chest CT)	Autoantibodies directed against Purkinje cells' nuclei, striatal neurons, and hippocampal neurons	Intravenous immunoglobulin 0.4 g/kg/day during 5 days then intravenous methylprednisolone 1 g daily for 5 days	No improvement after intravenous immunoglobulins Improvement after corticosteroids

Table 2 (continued)

Authors	Age (years), sex	Temporal progression of the cerebellar syndrome	Concomitant signs/symptoms (in addition to the cerebellar syndrome)	Lesions visualized through brain MRI	CSF analysis abnormalities	RT-PCR assay for SARS-CoV-2 on NP swab and CSF	Lungs involvement	Presence of antibodies directed against neuronal surface or intracellular antigens	Treatment	Outcome
Werner et al	62, M	Over several days	Fever	Slight generalized brain atrophy with accentuation of atrophy in the cerebellum	Normal	Positive on NP swab 2 days after admission. Negative on CSF	Yes, several days before and symptomatic	No	Intravenous methylprednisolone 500 mg daily for 5 days	Slow improvement; recovery almost complete after 4 months
Dar et al	65, M	Over 2 days		Not performed	Normal	Positive on NP swab a few weeks before admission. Not performed on CSF	Yes, severe COVID-pneumonia a few weeks before	No	Intravenous methylprednisolone 1 g daily for 5 days	Improvement
Chattopadhyay et al	70, M	Over 2 days		Multiple old lacunar infarcts without any other abnormal signal changes	Elevated total protein level (60 mg/dL)	Positive on NP swab 5 weeks before admission but negative at admission. Not performed on CSF	Unknown	No	Intravenous methylprednisolone 1 g daily for 5 days	Rapid improvement
Mudabbir et al	41, M	Over 3 days		Mild cerebellar atrophy	Normal	Positive on NP swab 40 days before admission but negative at admission. Negative on CSF	Yes, 40 days before admission	Not performed	Intravenous methylprednisolone 1 g daily for 5 days then oral taper	Complete recovery after 1 week

Table 2 (continued)

Authors	Age (years), sex	Temporal progression of the cerebellar syndrome	Concomitant signs/symptoms (in addition to the cerebellar syndrome)	Lesions visualized through brain MRI	CSF analysis abnormalities	RT-PCR assay for SARS-CoV-2 on NP swab and CSF	Lungs involvement	Presence of antibodies directed against neuronal surface or intracellular antigens	Treatment	Outcome
Oosthuizen et al	52, M	Over 6 days	Signs of encephalitis 7 days after admission	T2/FLAIR hyperintense non-enhancing lesions involving the central midbrain and dorsal pons	Increase in WBC count (49/mm^3), with predominant lymphocytic pattern	Negative on NP swab at admission but positive 17 days after admission. Positive on CSF	Yes, concomitant and asymptomatic (found with chest CT)	Anti-amphiphysin antibodies	Oral prednisone 1 mg per kilogram per day	Improvement but incomplete recovery after 6 months
O'Neill et al	5, M	Over 11 days	Low-grade fever	Normal	Increased opening pressure (28 cmH2O) Increased WBC count (8/mm^3) with predominant lymphocytic pattern (86%)	Positive on NP swab at admission. Negative on CSF	No	Not performed	No pharmaceutical treatment Physical therapy	Complete recovery after 2 months
Akçay et al	3, M	Over several days	Fever, altered mental status, meningeal irritation signs	T2/FLAIR symmetrical pathological signal with restricted diffusion in both cerebral hemispheres	Normal	Negative on NP swab and CSF	Yes, concomitant and symptomatic	Not performed	Intravenous immunoglobulin (1 g/kg/day for 2 days) and intravenous methylprednisolone 30 mg/kg/day for 5 days then oral taper	Complete recovery after 1 month

Table 2 (continued)

Authors	Age (years), sex	Temporal progression of the cerebellar syndrome	Concomitant signs/ symptoms (in addition to the cerebellar syndrome)	Lesions visualized through brain MRI	CSF analysis abnormalities	RT-PCR assay for SARS-CoV-2 on NP swab and CSF	Lungs involvement	Presence of antibodies directed against neuronal surface or intracellular antigens	Treatment	Outcome
Sharma et al. (patient 1)	12, M	Over several days	Headache, altered mental status, fever	T2/FLAIR confluent asymmetric hyperintensities involving both cerebellar hemispheres with faint folial enhancement	Normal	Positive on NP swab. Negative on CSF	Unknown	Not performed	Temporary external ventricular drain in the right lateral ventricle. Corticosteroids and acyclovir for 14 days (unknown dosing)	Complete recovery after 6 weeks
Sharma et al. (patient 2)	10, M	Over 2 days	Headache, drowsiness	T2/FLAIR cerebellar hyperintense lesions	Normal	Positive on NP swab. Negative on CSF	Unknown	Not performed	Temporary external ventricular drain during 3 days. Corticosteroids and acyclovir for 14 days (unknown dosing)	Complete recovery after 3 months
Tomar et al	13, M	Over 10 days	Headache, fever	Normal	Normal	Positive on NP swab 10 days before the admission, negative at admission Negative on CSF	Yes, a few days before and mildly symptomatic	No	Intravenous methylprednisolone 500 mg/day for 5 days	Rapid improvement

Table 2 (continued)

Authors	Age (years), sex	Temporal progression of the cerebellar syndrome	Concomitant signs/ symptoms (in addition to the cerebellar syndrome)	Lesions visualized through brain MRI	CSF analysis abnormalities	RT-PCR assay for SARS-CoV-2 on NP swab and CSF	Lungs involvement	Presence of antibodies directed against neuronal surface or intracellular antigens	Treatment	Outcome
Khan et al	11, M	Over 3 days	Generalized tonic–clonic seizures, headache, delirium, meningeal irritation signs, and shock	Diffuse cerebellar swelling with T2/ FLAIR hyper intensity, restricted diffusion and gadolinium enhancement	Not performed	Unknown	Unknown	Not performed	Intravenous methylprednisolone 30 mg/kg/ day for 3 days then intravenous dexamethasone (0. 6 mg/kg/ day) and mannitol for intracranial hypertension Intravenous immunoglobulin (2 g per kilogram) and oral prednisolone (2 mg/ kg/day) then tapper	Complete recovery after 4 months
Sotgiu et al	5, M	Over a few days	Headache	Diffuse T2-FLAIR Hyperintense cerebellar cortical lesions	Not performed	Positive on NP swab 4 weeks before the admission, negative at admission. Not performed on CSF	No	No	No pharmacological treatment	Complete recovery after 5 days

[a]CSF states for cerebrospinal fluid, CT for computed tomography, F for female, FLAIR for fluid-attenuated inversion recovery, GAD for glutamic acid decarboxylase, GFAP for glial fibrillary acidic protein, M for male, NP for nasopharyngeal, and WBC for white blood cell count

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Data availability Further information about the patient can be obtained directly from the authors.

Declarations

Ethics approval and consent to participate Informed consent for patient's clinical course and images to be published was provided by the patient's surrogate.

Competing interests The authors declare no competing interests.

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