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COVID-19

Anti-GAD associated post-infectious cerebellitis after COVID-19 infection

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Abstract
The coronavirus disease 2019 (COVID-19), caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), continues to spread rapidly all over the world. Besides severe pneumonia, it causes multisystemic disease, including neurological findings. Here, we present a patient with anti–glutamic acid decarboxylase (anti-GAD) antibody-associated cerebellitis developed after COVID-19 infection. The patient responded well to the immune treatments. Our knowledge about SARS-CoV-2 infection–related neurological disorders is limited. New data are needed to recognize the clinical spectrum of autoimmune neurological disorders that emerges after SARS-CoV-2 infection.

Keywords Ataxia · Anti-GAD · COVID-19 · SARS-CoV-2 · Post-infectious · Cerebellitis

Introduction

The coronavirus disease 2019 (COVID-19), caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), continues to spread rapidly all over the world [1]. Besides severe pneumonia, it causes multisystemic disease, including neurological findings such as anosmia, cranial neuropathies, Guillain–Barre syndrome, and encephalitis. Our current knowledge about post-infectious immune pathologies caused by SARS-CoV-2 is limited. Herein, we present a patient with anti–glutamic acid decarboxylase (anti-GAD) antibody-associated cerebellitis developed after COVID-19 infection.

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Case

A 54-year-old male teacher presented with anosmia and generalized myalgia that started 2 days ago. The patient's past medical history revealed primary hypertension treated with candesartan for 2 years. On admission, the patient did not have any respiratory symptoms, and his vital signs were within normal limits. There was pneumonic infiltration suggestive of asymptomatic pneumonia on his chest computed tomography. The patient's nasopharyngeal real-time reverse transcriptase-polymerase chain reaction (rt-PCR) test for SARS-CoV-2 was positive. He was treated with favipiravir with a loading dosage of 1600 mg and maintenance dosage of 600 mg per day, acetylsalicylic acid 100 mg per day, and paracetamol 1000 mg per day.

After treatment for 5 days, the patient's symptoms resolved. However, 2 weeks later, the patient complained of incoordination during writing due to a slight tremor in his hands. One week later, truncal ataxia was added to the clinical picture causing gait difficulty. On his first neurological examination in the emergency department, the patient was disoriented. He had dysarthria and a convergence spasm in his ophthalmologic examination. Deep tendon reflexes were normoactive, and he had bilateral moderate appendicular and severe truncal ataxia. He could not walk independently with a Scale for Assessment and Rating of Ataxia (SARA) score of 19.5/40.

The patient's brain magnetic resonance imaging (MRI) revealed edematous changes and hyperintensities in the cerebellar cortex in T2-weighted and FLAIR images (Fig. 1). Additionally, mild pial contrast enhancement was also observed in the cerebellum. The patient's cerebrospinal fluid (CSF) examination revealed a normal opening pressure. There were 20 lymphocytes/mm^3 in the CSF. The CSF total protein level was 45 mg/dl (normal range: 15–45 mg/dl); the glucose level was 62 mg/dl with a simultaneous blood glucose level of 97 mg/dl. The CSF culture was sterile.

To investigate COVID-19 encephalitis, the SARS-CoV-2 rt-PCR test was repeated for both CSF and nasopharyngeal specimens with negative results. Additionally, thyroid function tests and serum vitamin B_{12} and folate levels were also within normal limits. However, anti-thyroid peroxidase, anti-thyroglobulin levels, and anti-tissue transglutaminase IgG were slightly higher. VDRL, wright test for Brucella infection, anti-tissue transglutaminase IgA, anti-Hu, anti-Yo, anti-Ri, anti-amphiphysin, anti-Tr, anti-PCA-2, anti-Ma, anti-CV2-1, anti-ANNA-3, anti-NMDA-R, anti-AMPA-R1, anti-AMPA-R2, anti-Caspr2, anti-LGI1, and anti-GABA-R antibodies were negative in the serum samples. Serum anti-GAD antibody level was 114.41 IU/ml (normal range: 0–5 IU/ml). We also performed a chest and abdominal CT scan to investigate further, which did not show any abnormality.

The patient was treated with methylprednisolone 1 gr/day for 10 days and intravenous immunoglobulin 0.4 gr/kg/day for 5 days. One month after the treatment, the patient was able to walk independently without any signs of appendicular and truncal ataxia with a mild tremor in his upper extremities that was successfully treated with propranolol. Monthly intravenous immunoglobulin and oral methylprednisolone treatment were given for 3 months. The patient's SARA score 3 months after his first symptoms was 1/40.

Discussion

Immune-mediated neuronal apoptosis and dysfunction are observed in the autoimmune cerebellar syndromes, including gluten ataxia, opsoclonus-myoclonus syndrome, paraneoplastic cerebellar degeneration, and post-infectious cerebellar syndromes using various mechanisms [2]. One of the well-defined ataxic syndromes, the anti-GAD antibody, may cause an autoimmune cerebellar syndrome by impairing GABAergic transmission via cell-mediated immunity [3].

As far as our knowledge, this is the first case with post-infectious anti-GAD antibody-related cerebellar syndrome after SARS-CoV-2 infection. Reported cases of ataxia associated with SARS-CoV-2 are reviewed in Table 1. Para-/post-infectious ataxia is reported between 7 and 83 years of age. However, the majority of cases were reported in middle-aged male patients as in our case. Besides ataxia, a wide spectrum of clinical findings was observed including opsoclonus, myoclonus, ocular movement disorders, seizures, vertigo, behavioral disorders, involuntary movements, tremor, and dysarthria. Our case adds convergence spasm to these diverse findings. Four out of 31 cases reviewed in Table 1 have abnormal brain imaging including hyperintensities in the brainstem and cerebellum. However, brain FDG-PET abnormalities in the frontal cortex and cerebellum were reported in another three patients. Similar to our case, bilateral cerebellar hemispheres and vermis hyperintensities in FLAIR imaging and cerebellar cortical meningeal contrast enhancement were observed by Fadakar et al. [25]. In contrarily to our case, the presentation of cerebellar ataxia was concomitant with COVID-19 infection, and SARS-CoV-2 rt-PCR test was found positive in CSF [25]. Although autoantibody screening was performed in the majority of cases, anti-amphiphysin, anti-NMDAR antibodies, and autoantibodies directed against the nuclei of

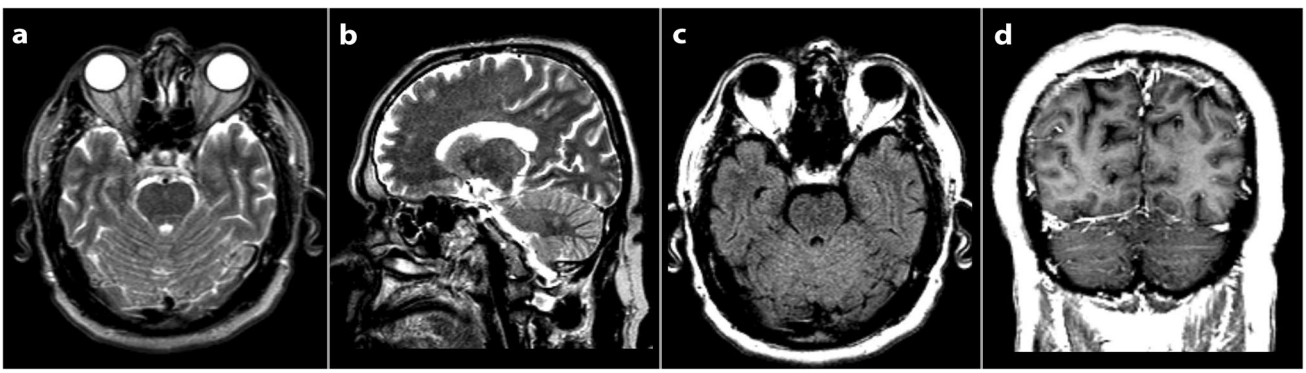

Fig. 1 Brain MRIs show edematous hyperintense changes in T2-weighted and FLAIR images in the cerebellum (**a**, **b**, **c**) and cerebellar pial contrast enhancement (**d**)

Table 1 Literature review of patients with possible immune-mediated post-/para-infectious ataxia related to COVID-19 infection. Publications without enough data and cases with ischemic stroke or peripheral nervous system pathology in proposed etiology are not included

Publication	Age/sex	Clinical findings	Brain imaging	CSF features	Temporal association with COVID-19 infection	SARS-CoV-2 rt-PCR at neurological presentation	Autoantibody screening	Treatment	Outcome
Oosthuizen et al. [4]	52/M	Dysarthria, limb and gait ataxia, nystagmus	Hyperintensities in brainstem	Lymphocytes 49/µL, polymorphonuclear cells: 2/µL Slightly increased IgG index: 0.62 (<0.6)	Presented with neurological symptoms	Nasopharyngeal swab negative at presentation, positive on day 17. Positive in CSF	Anti-amphiphysin positive in serum	Prednisone (1 mg/kg/day)	Dramatical improvement. Independent six months later
Saha et al. [5]	78/F	Opsoclonus, myoclonus, gait ataxia	Normal brain MRI	Elevetaed total protein level (55 mg/dl)	14 days after	N/A	Negative in CSF	Anti-epileptic treatment MP (1 g/day for 5 days)	Responded well to the treatment
Sarigecili et al. [6]	7/M	Gait ataxia, seizure, altered mental status, involuntary movements	Normal brain MRI	Non-inflammatory	Presented with neurological symptoms	Positive in oropharyngeal swab	CSF anti-NMDAR IgG positive	IVIG PLEX MP (30 mg/kg/day for 5 days, 20 mg/kg/day for 2 days)	Partial recovery, ambulating but mildly ataxic
Werner et al. [7]	62/M	Limb and gait ataxia	Generalized brain atrophy with accentuation of atrophy in the cerebellum	OCB Type 4 at presentation, type 1 after therapy	16 days after	Positive in nasopharyngeal swab. Negative in CSF	Negative in CSF and serum	Acyclovir IV high-dose MP	Gradual improvement with acyclovir and more rapidly improvement with MP
Sharma et al. [8]	12/M 10/M	Altered mental status and limb/gait ataxia	Confluent asymmetric (right > left) hyperintensities in both cerebellar hemispheres with faint folial enhancement	Non-inflammatory	2–15 days after	Positive in nasopharyngeal swab. Negative in CSF	N/A	Steroid (dosage N/A) Acyclovir	Recovered without sequelae
Fernandes et al. [9]	58/F	Tremor, severe gait & limb ataxia, dysarthria, action myoclonus	Normal brain MRI	Non-inflammatory	17 days after	Negative in nasopharyngeal swab	Negative in CSF and serum	IVIG Corticosteroid Anti-epileptic treatment	Partial recovery
Sanguinetti et al. [10]	57/M	Myoclonus,gait ataxia, opsoclonus	Normal brain MRI	N/A	5 days after	N/A in CSF	N/A	MP (80 mg/day) IVIG (2 g/kg)	Improvement in ataxia and myoclonus

Table 1 (continued)

Publication	Age/sex	Clinical findings	Brain imaging	CSF features	Temporal association with COVID-19 infection	SARS-CoV-2 rt-PCR at neurological presentation	Autoantibody screening	Treatment	Outcome
Urrea-Mendoza et al. [11]	32/M	Opsoclonus, myoclonus and ataxia	Normal brain MRI	N/A	12 days after	N/A	N/A	Anti-epileptic treatment MP 40 mg/day	Occasional myoclonus with mild ataxia
Chan et al. [12]	44/M	Action myoclonus, dysarthria, limb and gait ataxia	Normal brain MRI	Non-inflammatory	12 days after	Negative in CSF and nasopharyngeal swab	N/A	MP (1 g/day for 5 days)	Complete recovery in 2 months
Foucard et al. [13]	63/M 83/M	Case 1: Confusion, myoclonus, ataxic dysarthria, opsoclonus. Case 2: Action myoclonus with rapidly progressive cerebellar syndrome	Normal brain MRIs	Non-inflammatory	6–10 days after	N/A	Negative in serum and CSF	IV Steroid (1 g/day 5 days) IVIG (0.4 g/kg 5 days)	Rapid improvement
Shah and Desai [14]	Middle-aged/M	Myoclonus, speech, limb and gait ataxia, opsoclonus	Normal brain MRI	Normal	3 weeks after	N/A in CSF	Negative	MP (1 g/day) Anti-epileptic treatment	Recovery in 1 week
Emamikhah et al. [15]	39–54 6 M/1 F	Gait ataxia, myoclonus ± opsoclonus	Normal brain imaging	Non-inflammatory in 3/7. N/A in 4/7	3 days-3 weeks after	5/7 positive, 1/7 negative, 1/7 N/A in nasopharyngeal swab results	1/7 negative in serum and CSF. 6/7 N/A	Anti-epileptic treatment in 7/7 IVIG in 5/7. Dexamethasone in 1/7	Complete recovery in 2/7. Partial recovery in 3/7. N/A in 2/7
Shetty et al. [16]	41/M	Action myoclonus, gait ataxia	Normal brain MRI	Non-inflammatory	10 days after	Negative	Negative in CSF	Anti-epileptic treatment MP (1 g/day for 5 days)	Complete recovery at 6 weeks
Grimaldi et al. [17]	72/M	Myoclonus, limb and gait ataxia, dysartria	Normal brain MRI. FDG-PET: Putamen and cerebellum hypermetabolism, diffuse cortical hypometabolism	Mildly elevated CSF total protein (49 mg/dl)	17 days after	Negative in CSF	Autoantibodies directed against the nuclei of Purkinje cells, striatal and hippocampal neurons in serum and CSF immunostaining	MP (1 g/day for 5 days) IVIG (2 g/kg)	Recovery within 3 weeks

Table 1 (continued)

Publication	Age/sex	Clinical findings	Brain imaging	CSF features	Temporal association with COVID-19 infection	SARS-CoV-2 rt-PCR at neurological presentation	Autoantibody screening	Treatment	Outcome
Povlow and Auerbach [18]	30/M	Limb and gait ataxia, dysarthria, nystagmus	Normal brain MRI	Non-inflammatory	Presented with neurological symptoms	N/A in CSF	Serum ganglioside antibodies and anti-GAD negative	No specific treatment	Partial recovery
Wright et al. [19]	79/M	Gait ataxia, confusion, ocular flutter, opsoclonus	Non-remarkable brain MRI	N/A	8 days after	N/A in CSF	N/A	No specific treatment	Progressive decline leading to death at 43th day
De Marcaida et al. [20]	59/M	Disabling tremor, gait ataxia, left appendicular ataxia, dysarthria, vertigo, confusion	Brain MRI within normal ranges	N/A	2 weeks after	Positive (specimen type N/A)	N/A	Without any intervention	Almost complete recovery
Dijkstra et al. [21]	44/M	Myoclonus, limb and gait ataxia, ocular flutter, behavioral disturbances	Normal brain MRI	Non-inflammatory	2 weeks after	Negative in CSF	Negative in serum and CSF	MP (1 g/day for 5 days) IVIG (1.2 g/kg)	Full recovery within 2 months
Schellekens et al. [22]	48/M	Myoclonus, limb and gait ataxia, hypermetric saccades	Normal brain MRI	Non-inflammatory	13 days after	Negative in CSF	Para-neoplastic antibodies negative in CSF. Anti-VGKC negative in serum	Anti-epileptic treatment	Partial recovery within 2 months

Table 1 (continued)

Publication	Age/sex	Clinical findings	Brain imaging	CSF features	Temporal association with COVID-19 infection	SARS-CoV-2 rt-PCR at neurological presentation	Autoantibody screening	Treatment	Outcome
Delorme et al. [23]	72/M 60/F	Case 1: Myoclonus, ataxia, frontal lobe syndrome Case 2: Limb and gait ataxia, dysartria, frontal lobe syndrome	Case 1: Normal brain MRI. FDG-PET: Bilateral prefrontal and left parietotemporal hypometabolism, cerebellar vermis hypermetabolism. Case 2: Known right mesial scleroris. FDG-PET: Hypometabolism in bilateral orbitofrontal cortices, hypermetabolism in bilateral striatum and cerebellar vermis	Non-inflammatory	Case 1: 15 days after Case 2: Presented with neurological symptoms	Negative in CSF	N/A	Case 1: IVIG (2 g/kg) Case 2: MP (2 mg/kg for 3 days)	Complete recovery
Diezma-Martin et al. [24]	70/M	Voice, limb and gait ataxia, orthostatic tremor	Normal brain MRI	Normal	17 days after	Negative in CSF	N/A	Anti-epileptic treatment	Improvement within a month
Fadakar et al. [25]	47/M	Limb and gait ataxia, dysarthria, vertigo, nystagmus, hypermetric saccades	Brain MRI: FLAIR hyperintensities in bilateral cerebellar hemispheres and vermis, cerebellar cortical meningeal enhancement	Elevated CSF total protein: 58 mg/dl, leukocytes: 10/mm^3	3 days after	Positive in CSF	Negative in CSF and serum	No specific treatment	Marked improvement within a month

OCB oligoclonal bands, *MRI* magnetic resonance imaging, *CSF* cerebrospinal fluid, *M* male, *F* female, *MP* methylprednisolone, *IV* intravenous, *IVIG* intravenous immunoglobulin, *PLEX* plasma exchange, *anti-GAD* anti–glutamic acid decarboxylase, *anti-VGKC* anti–voltage-gated potassium channel, *N/A* non-available

Purkinje cells, striatal and hippocampal neurons in serum, and CSF immunostaining were reported only in three cases [4, 6, 17]. In the reported cases in which patients who had SARS-CoV-2 rt-PCR test were positive either in CSF or nasopharyngeal swabs, it indicates cerebellar syndrome is related to the infectious process. The majority of cases responded well to the immunotherapy, although mortality was reported in one patient without specific treatment.

In our case, the SARS-CoV-2 rt-PCR test was negative in the nasopharyngeal and CSF specimens, whereas anti-GAD antibody was detected with a high titer in the etiological workup of the cerebellar syndrome. It has been reported that the detection of anti-GAD antibodies in high titers suggests autoantibody-specific disease [2]. The dramatic response to immune therapies such as high-dose steroids and intravenous immunoglobulin also suggests the existence of an underlying autoimmune process. Besides, anti-GAD-associated neurological disorders are frequently accompanied by autoimmune disorders such as autoimmune thyroiditis and gluten sensitivity, as in our case [26]. Various side effects are reported with high-dose favipiravir in the treatment of COVID-19 [27]. However, cerebellar ataxia and convergence spasm are not among well-known adverse effects of favipiravir use, and drug toxicity is not a likely cause in our case.

These findings confirm that high titer anti-GAD seropositivity is associated with post-infectious cerebellar syndrome in our case. Besides our findings, anti-amphiphysin, anti-Caspr2, anti-GD1b, and anti-NMDAR antibodies related to neurological disorders after SARS-CoV-2 infection have been reported in the literature, suggesting that SARS-CoV-2 infection might trigger autoimmunity [4, 6, 28–30]. However, it seems complicated to establish a direct pathogenetic relationship between SARS-CoV-2 infection and anti-GAD-associated autoimmune cerebellitis.

Conclusion

Since the first months of its emergence, SARS-CoV-2 infection has been associated with a wide array of neurological and neuropsychiatric findings, including encephalitis, inflammatory central nervous system syndromes, ischemic strokes, and peripheral neurological diseases [31]. Our knowledge about SARS-CoV-2 infection–related neurological disorders is limited. New data are needed to recognize the clinical spectrum of autoimmune neurological disorders that emerges after SARS-CoV-2 infection.

Author contributions ASE and MK designed the study; ASE, AP, and NYG assembled the data. All authors wrote and approved the final article.

Declarations

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent to publish was obtained from the participant. Ethics committee approval was not applicable as the data was analyzed retrospectively and had no effect on treatment.

Conflict of interest The authors declare no competing interests.

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