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.

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 pneumatic 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/mm³ 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 B₁₂ 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
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 | Elevated 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 | IVIGPLEX 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 |
| Shama et al. [8]            | 12/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 |
|                            | 10/M    |                                                        |               |                                       |                                             |                                               | N/A |                         |                                    |
| 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 |
| 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                      | 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    | Case 1: Confusion, myoclonus, ataxia, 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                              | Negative                                      | MP (1 g/day) Anti-epileptic treatment | Recovery in 1 week                  |                             |
| Emamikhah et al. [15]           | 39–54    | 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 | Antiepileptic 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, dysarthria                                       | Normal brain MRI | 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                  |                             |
| 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 leading to death at 43th day | Progressive decline             |
| 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   | Case 1: Myoclonus, ataxia, frontal lobe syndrome    | Case 1: Normal brain MRI.            | Non-inflammatory | Case 1: 15 days after                       | Negative in CSF                                  | N/A                    | Case 1: IVIG (2 g/kg) | Complete recovery |
|                   | 60/F    | Case 2: Limb and gait ataxia, dysarthria, frontal lobe syndrome | FDG-PET: Bilateral prefrontal and left parietotemporal hypometabolism, cerebellar vermis hypermetabolism. |                   | Case 2: Presented with neurological symptoms |                                                 |                        | Case 2: MP (2 mg/kg for 3 days) |                      |
|                    |         |                                                       | Case 2: Known right mesial scleroris. |                   |                                              |                                                 |                        |                           |                          |
|                    |         |                                                       | FDG-PET: Hypometabolism in bilateral orbitofrontal cortices, hypermetabolism in bilateral striatum and cerebellar vermis |                   |                                              |                                                 |                        |                           |                          |
| 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/mm³ | 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 detected 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 autoimmune-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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