

SARS-COV-2 a trigger of myelin oligodendrocyte glycoprotein-associated disorder

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Introduction

There are several causes to neurological symptoms in coronavirus disease 2019 (Covid-19).¹ It has previously been described that MERS-CoV and HcoV-OC43 are able to initiate immunopathogenic responses in the central nervous system (CNS),² and reports suggest that SARS-CoV-2 can trigger host autoantibody disease.^{3–7}

MOGAD (Myelin Oligodendrocyte Glycoprotein-Associated Disorder) is a monophasic or relapsing inflammatory demyelinating condition of the central nervous system (CNS) characterized by the presence of antibodies to MOG (Myelin Oligodendrocyte Glycoprotein).⁸

One study concluded that SARS-CoV-2 may be a trigger of MOGAD as the authors found a higher occurrence of SARS-CoV-2 IgG antibodies in patients with MOGAD, but the difference was non-significant.⁹ However, another study showed that only one out of 15 patients tested positive for anti-MOG in a group of post Covid-19 acute disseminated encephalomyelitis (ADEM) or acute hemorrhagic leukoencephalitis.¹⁰

We report four cases with onset of MOGAD after SARS-CoV-2 infection and reviewed previously published case reports with similar associations suggesting a role of SARS-CoV-2 in the pathogenesis of MOGAD.

Abstract

SARS-COV-2 frequently cause neurological disorders and is sometimes associated with onset of autoimmune diseases affecting the nervous system. Over recent years, a rare but distinct diagnosis designated myelin oligodendrocyte glycoprotein-associated disorder (MOGAD) has been recognized in patients with attacks of optic neuritis, myelitis, or encephalomyelitis and increased levels of anti-MOG antibodies. The cause of MOGAD is unknown. However, there have been reports of single cases of MOGAD in patients with Covid-19 infection. We report a series of SARS-CoV-2 positive patients that developed MOGAD, but a homology search did not support a cross-reactive immune response to SARS-CoV-2 spike-protein and MOG.

Methods

Four patients were diagnosed with MOGAD after Covid-19 infection, three between April 2020 and April 2021 at the department of neurology, Sahlgrenska University Hospital, Västra Götaland County, and one in May 2021 at the department of medicine, Halmstad Hospital, Halland County. We investigated the incidence of MOGAD in the county of Västra Götaland during the observational period by searching for positive anti-MOG index results determined at the two laboratories performing MOG serology in the county (Sahlgrenska Clinical Immunology Laboratory, and Wieslab Diagnostic Services). We also searched for cases by reaching out to neurologists at neurological departments in the county, and from a recent investigation on the prevalence of MOGAD in Västra Götaland until the year of 2020 using medical records and laboratory data, approved by the director of operations manager (unpublished). Population statistics were collected from Statistics Sweden (SCB).¹¹ The analysis of anti-MOG antibodies was performed with indirect immunofluorescence on MOG-transfected cells, semiquantitatively (EU-90; Euroimmun, Lubeck, Germany), with a cutoff level at ≥1:10. Informed consent was obtained from all subjects for publication of their case.

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Case presentations of MOGAD following COVID-19

See also Table 1, section 1.

Case of acute disseminated encephalomyelitis

A 25-year-old woman with fever and headache tested positive for PCR SARS-CoV-2. Two weeks after Covid-19 infection her condition deteriorated rapidly with severe paraparesis, hypesthesia below Th 5, urinary retention, and decreased consciousness. MRI of the brain and spinal cord revealed multiple non-enhancing cerebral parenchymal and spinal cervicothoracic T2 high intensity lesions. Cerebrospinal fluid (CSF) showed 295 \times 10⁶/L lymphocytes, 229 \times 10⁶/L neutrophils, 103 \times 10⁶/L monocytes, and two oligoclonal IgG bands. She was diagnosed with ADEM and started treatment with iv methylprednisolone 1 g for 5 days. Infectious diagnostics workup was negative, including CSF SARS-CoV-2 PCR. Serum IgG AQP4-antibodies was negative, but serum anti-MOG IgG proved positive (1:1000). MRI 2 weeks later showed new noncontrast enhancing high signal lesions on T2/FLAIR in pons, corpus callosum, mesencephalon, and progress of intramedullary lesions in the cervical and thoracic spinal

Table 1. Case 1–4 clinical data (section 1) and previously published case reports (section 2) with Covid-19 and MOGAD association. All had positive serum anti-MOG.

Case author	Syndrome	Covid-19; MOGAD interval (days)	Age (y)	Sex	Covid-19 symptoms	SARS-CoV-2 test	CSF
Casa pr 1 d in this ran	,		5.07				
Case nr 1–4 in this repo	ADEM	14	25	Female	Fever, headache	NP PCR+	Lymphocytes
	ADEINI		23	remale	rever, fleadache		295/qL
	ON and myelitis	56	20	Male	Minor resp. symptoms	NP PCR+	Lymphocytes 159/qL
	Bilateral ON	14	29	Male	Minor resp. symptoms	NP PCR+	Normal
	Unilateral ON	29	60	Female	Fever, dyspnea	NP PCR+	Normal
Previously published cas	se reports (N = 16):						
Zhou et al, 2020 ¹³	Bilateral ON	"a few days"	26	Male	Cough.	NP PCR+*	WBC 55/qL
Sawalha et al, 2020 ¹⁴	Bilateral ON	14	44	Male	Dyspnea, cough.	NP PCR+*	WBC 3/qL
Zoghi et al, 2020 ¹⁵	Encephalomyelitis	14	21	Male	Fever, dyspnea, cough.	IgG positive	Lymphocytes 90/qL
Woodhall et al, 2020 ¹⁶	MOGAD relapse; ON	6	39	Female	Malaise, coryzal symptoms.	NP PCR+ *	NA
Jumah et al, 2021 ¹⁷	Myelitis (HHV6+)	No interval	61	Male	Fever, arthralgia. Hospitalized	NP PCR+*	WBC 279/qL
Khan et al, 2021 ¹⁸	Bilateral ON	14	11	Male	Brief febrile illness.	NP PCR+*	Lymphocytes 55/qL
Pinto et al, 2020 ¹⁹	CNS vasculopathy	7	44	Female	Minor resp. symptoms.	NP PCR+*	WBC 13/qL
Kogure et al, 2021 ²⁰	Bilateral ON	NA	47	Male	None	NP PCR+*	Normal
Žorić et al, 2021 ²¹	ON	28	63	Male	Pneumonia. Hospitalized	IgG positive	NA
Peters et al, 2021 ²²	Encephalitis	35	23	Male	Headache.	NP PCR+	WBC 57/qL
Vraka et al, 2021 ²³	Encephalitis	10	1	Female	Fever	NP PCR+*	WBC 10/qL
Sinha et al, 2021 ²⁴	Encephalomyelitis	10	11	Female	Fever, cough	NP PCR+*	NA
Athanasopoulos et al, 2021 ²⁵	Encephalitis	No interval	1	Female	Fever	NP PCR+*	Normal
Rojas-Correa et al, 2021 ²⁶	Bilateral ON	45	69	Male	Fever, mild cough	IgG positive	Normal
Cay-Martinez et al, 2021 ²⁷	Encephalomyelitis	NA	7	Female	None	IgG positive	WBC 551/qL
Ruijter et al, 2020 ²⁸	Bilateral ON	Weeks	15	Male	Fever, cough	NA	Normal

ON, Optic Neuritis; MOG, Myelin Oligodendrocyte Glycoprotein; MOGAD, Myelin Oligodendrocyte Glycoprotein-Associated Disease; ADEM, Acute Disseminated Encephalomyelitis; SARS-CoV-2, Severe acute respiratory syndrome coronavirus 2; CSF, Cerebrospinal fluid; WBC, White Blood Cell; PCR, polymerase chain reaction; NP, Nasopharyngeal; Resp, respiratory.

*Positive at the time of MOGAD illness.

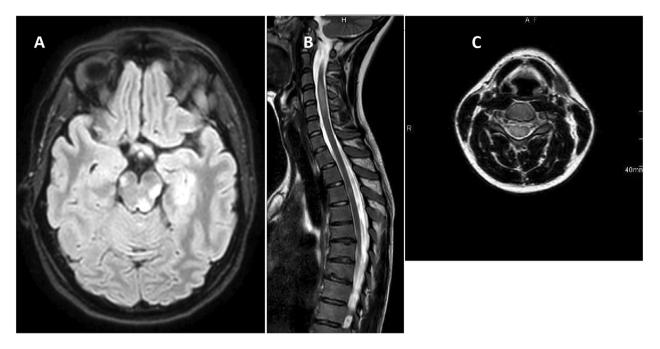


Figure 1. (A-C) MRI brain and spinal cord. Depicted axial cerebral (A) T2-weighted images show high signal lesions bilaterally in pons and left dorsal hippocampus, and (B) sagittal T2-weighted images of the spinal medulla show medullary high signal lesions most pronounced at C4-C6 levels. There was slight patchy contrast enhancement at Th6-Th9 levels in T1 contrast images. (C) Axial T2-weighted image at C4 level.

cord including contrast enhancement from Th6-Th9 (Fig. 1A-C). The patient had a new course of intravenous steroids followed by a 5-day regimen of plasmapheresis, and then methotrexate and slow tapering of oral prednisolone. Four weeks later she had a relapse of optic neuritis with optic disc edema at ophthalmologic examination and confirmed by optical coherence tomography (OCT). MRI revealed a new lesion in the cerebellar peduncle. After e new course of high dose steroids iv, she made a quick recovery and at 4 months follow-up after MOGAD onset MRI revealed virtually complete radiological resolution of the cerebral and spinal lesions. The patient had slight lower extremity dysesthesia and moderate bladder dysfunction, and continued prednisolone 10 mg daily and methotrexate 15 mg weekly.

Case of bilateral optic neuritis and myelitis

A 20-year-old man with a history of personality disorder and polysubstance abuse had SARS-CoV-2 PCR positive Covid-19 infection with minor respiratory symptoms. Eight weeks post infection he presented with gradual onset of headache, photo-and phonophobia, left sided hemianesthesia, severe paraparesis, urinary retention, back pain, and reduced vision. A neuro-ophthalmologic examination including OCT showed bilateral optic disc edema, reduced visual acuity, and color vision consistent with optic neuritis. MRI of the spinal cord revealed pronounced medullary T2 lesions from Th9 to conus medullaris, small focal lesions at Th7 and C6 levels, and slight contrast enhancement at Th9-Th11. A lumbar puncture showed 159 \times 10⁶/L lymphocytes, 39 \times 10⁶/L monocytes, 33×10^6 /L neutrophils, normal IgG index, normal kappa index, and no oligoclonal IgG bands in the CSF. Serum MOG-antibody test was positive at a 1:100 titer. Anti-AQP4 antibody test was negative. Diagnostic workup regarding infectious or rheumatological disease was negative. He was treated with plasmapheresis on five consecutive days followed by iv methylprednisolone 1 g daily for 3 days and then oral prednisolone tapered to 10 mg daily and 15 mg methotrexate weekly. At follow-up after 6 months he displayed residual symptoms of moderate paraparesis.

Case of bilateral optic neuritis

A 29-year-old man had subacute onset of bilateral visual impairment and frontal headache. No significant antecedent health issue was identified apart from a Covid-19 infection 15 days earlier. An ophthalmologist established a diagnosis of papillitis compatible with optic neuritis. OCT showed bilateral optic disc edema. The patient was admitted to the neurology department, but no other neurological deficits were detected. An MRI of the brain

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showed normal results, and lumbar puncture revealed normal opening pressure, normal levels of CSF white blood cells and protein, and oligoclonal IgG bands were negative. Methylprednisolone iv 1 g treatment daily for 3 days was commenced, followed by oral prednisolone tapered to 10 mg daily. No serum AQP4-antibodies were detected, but anti-MOG IgG was positive at a 1:100 titer. Vision returned to normal within 1 month. Anti-MOG serum titer was 1:10 at 6 weeks and negative at 4 months after optic neuritis.

Case of unilateral optic neuritis

A 60-year-old woman, originating from China, presented with Covid-19 with fever, cough, myalgia, and dyspnea. She tested positive for PCR SARS-CoV-2. She was admitted to the intensive care unit due to respiratory insufficiency and received oxygen and oral betamethasone and rivaroxaban. She improved and was discharged from the hospital, but 54 days after the Covid-19 onset she developed a right sided eye pain and an ophthalmologist diagnosed a unilateral optic neuritis with papillitis and reduced visual acuity to 20/100. MRI showed swelling and contrast enhancement in the right optic nerve, but there was no significant pathology in the cerebral parenchyma or the spinal cord. CSF analysis revealed normal cell count, albumin, neurofilament light chain (NfL), and no oligoclonal IgG bands. She was treated with methylprednisolone 1 g daily for 3 days followed by prednisolone with slow tapering. Serology testing for anti-AQP4 was negative but MOG-antibodies were positive with a 1:1000 titer.

Incidence of MOGAD in the county of Västra Götaland

We identified a total of eight adult new incident MOGAD patients in the county of Västra Götaland (population 1 365 245 in 2020¹¹) with a clinical onset between April 2020 and April 2021, corresponding to a MOGAD incidence of 0.59:100 000. These included the three post Covid-19 cases of this report, and none of the other five cases were preceded by a Covid-19 infection. Between April 2019 and April 2020, the year prior the observational period, there was five incident adult cases of MOGAD corresponding to an incidence of 0.37:100 000. An estimation of the incidence of PCR confirmed SARS-CoV-2 infection and MOGAD in the Västra Götaland County between April 2020 and April 2021 was 1.94/ 100.000 (154 388 cases¹²). However, the Covid-19 incidence is probably an underestimation since only few patients with suspected Covid-19 had a confirmatory PCR test during the early phase of the pandemic.

Search for published cases in the literature

A search for previously published cases of MOGAD following a previous PCR confirmed SARS-CoV-2 infection was performed in September 2021 in PubMed and Google Scholar using the keywords COVID OR SARS-CoV-2 AND MOG OR Myelin Oligodendrocyte Glycoprotein OR Neuromyelitis Optica. We found 16 previously published case reports of possible Covid-19 related MOGAD.^{13–28} (Table 1; section 2).

Search for nucleotide and protein homologies between SARS-COV-2 and MOG

Nucleotide homologies between SARS-CoV-2 (NC_045512.2) and MOG (NM_206809.4) were analyzed using the Basic Local Alignment Search Tool (BLAST). No homology was more than 10 base pairs long. Protein homologies between myelin-oligodendrocyte glycoprotein isoform alpha1 precursor (NP_996532.2) and all proteins expressed by SARS-CoV-2 were analyzed using the Constraint-based Multiple Alignment Tool (COBALT). No sequences longer than four amino acids were identified.

Discussion

We report four cases of MOGAD diagnosed shortly after a previous PCR confirmed Covid-19 infection. Our case series support that SARS-CoV-2-associated MOGAD is a post-infectious immune-mediated reaction rather than a parainfectious (direct viral neurotropism) reaction. The mean latency period from onset of Covid-19 symptom to the first manifestations of MOGAD symptoms in our case series were 33 days (14–56), and similar latency (10– 42 days) was seen in patients with SARS-CoV-2 associated with acute transverse myelitis, also believed to be a post-infectious immune-mediated reaction.⁶

In a systematic literature search we found 16 previously published cases of MOGAD associated with Covid-19 infection (Table 1). In line with our four cases, the majority (11 out of 16) had a reported distinct interval (mean 18 days; 6–45) between Covid-19 disease and onset of MOGAD, but in contrast with our cases three had concomitant SARS-CoV-2 symptomatic infection and 10 had concomitant positive SARS-CoV-2 PCR testing.

MOG-IgG-mediated CNS disease including acute disseminated encephalomyelitis (ADEM) have been linked to several infectious triggers especially viral pathogens.^{29–31} There are a number of proposed mechanisms how this infection triggers a post-infectious immune disorder affecting CNS including molecular mimicry, epitope spreading, bystander activation, and polyclonal B-cell activation.³² The propensity for developing autoimmune diseases as an effect of Covid-19 has been previously reported, in prothrombotic autoantibodies,³³ type-1 interferons,³⁴ and the model of molecular mimicry has shown several shared heptapeptides between spike-protein and human genom,³⁵ however none of them corresponded to any sequence in MOG. In this study, we found no linear homology longer than four amino acids between SARS-Cov-2 and MOG. However, molecular mimicry caused by a conformational epitope cannot be ruled out.

A prior Dutch study have reported a mean MOGAD incidence of 0.13:100 000 in the adult population.³⁶ We found a higher incidence of MOGAD in the adult population of Västra Götaland County during the observational period (0.59:100 000), but also in the year before our study period (0.37:100 000). Three out of eight adult patients in the Västra Götaland County had an onset of MOGAD after Covid-19. This seemingly increased risk supports a link between SARS-CoV-2 and MOGAD. However, follow-up studies on the annual MOGAD incidence before, during, and after the Covid-19 pandemic would be of value to better assess a possible relationship.

In conclusion, we showed four cases with MOGAD onset in close relation to a previous Covid-19 infection and a suspected increase of the MOGAD incidence in Västra Götaland County during part of the Covid-19 pandemic. Although both findings suggest an association between SARS-CoV-2 and MOGAD we cannot rule out a coincidental occurrence of MOGAD during the Covid-19 pandemic. Nevertheless, our case series advocate a high vigilance for MOGAD in the occurrence of demyelinating symptoms following Covid-19 infection.

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Author Contributions

Study concept, design, data acquisition, and analysis: M.J, J.L, F.A, C.M, and T.O. Drafting large portions of the manuscript or figures: All authors. All authors critically reviewed and approved of the manuscript.

Conflict of Interest

MJ reports no conflict of interest. JL has received travel support and/or lecture honoraria from Biogen, Novartis, Merck,

Roche, Axelion, Sanofi and BMS; has served on scientific advisory boards for Almirall, Biogen, Novartis, Merck, Roche, BMS and Sanofi; serves on the editorial board of the Acta Neurologica Scandinavica; has received unconditional research grants from Biogen and Novartis. FA reports no conflict of interest. MA has received compensation for lectures and/or advisory board membership from Biogen and Merck. CM reports no conflict of interest. SH reports no conflict of interest. TO reports no conflict of interest.

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