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# Neuroinflammatory Disease following Severe Acute Respiratory Syndrome Coronavirus 2 Infection in Children

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**Objective** To describe neurologic, radiologic and laboratory features in children with central nervous system (CNS) inflammatory disease complicating severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection.

**Study design** We focused on CNS inflammatory diseases in children referred from 12 hospitals in the Paris area to Necker-Sick Children Reference Centre.

**Results** We identified 19 children who had a history of SARS-CoV-2 infection and manifest a variety of CNS inflammatory diseases: encephalopathy, cerebellar ataxia, acute disseminated encephalomyelitis, neuromyelitis optica spectrum disorder, or optic neuritis. All patients had a history of SARS-CoV-2 exposure, and all tested positive for circulating antibodies against SARS-CoV-2. At the onset of the neurologic disease, SARS-CoV-2 PCR results (nasopharyngeal swabs) were positive in 8 children. Cerebrospinal fluid was abnormal in 58% (11/19) and magnetic resonance imaging was abnormal in 74% (14/19). We identified an autoantibody co-trigger in 4 children (myelin-oligodendrocyte and aquaporin 4 antibodies), representing 21% of the cases. No autoantibody was found in the 6 children whose CNS inflammation was accompanied by a multisystem inflammatory syndrome in children. Overall, 89% of patients (17/19) received anti-inflammatory treatment, primarily high-pulse methylprednisolone. All patients had a complete long-term recovery and, to date, no patient with autoantibodies presented with a relapse.

**Conclusions** SARS2-CoV-2 represents a new trigger of postinfectious CNS inflammatory diseases in children. *(J Pediatr 2022;247:22-8)*.

Coronavirus disease 2019 (COVID-19) owing to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection was declared a world pandemic in March 2020.[1] In adults, infection ranges from asymptomatic infection to severe respiratory failure. Since the beginning of the COVID-19 pandemic, there are increasing reports of neurologic complications, including nonspecific headache, delirium, dizziness, and stroke.[2-5] Severe inflammatory central nervous system (CNS) events, such as encephalitis, acute disseminated encephalomyelitis (ADEM), and optic neuritis are rare, but have also been reported in case reports or small series of cases.[5,6] There are case reports of positive myelin

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| ADEM | Acute disseminated encephalomyelitis |
| AQP4 | Aquaporin 4 |
| CNS | Central nervous system |
| COVID-19 | Coronavirus disease 2019 |
| CSF | Cerebrospinal fluid |
| IL | Interleukin |
| MIS-C | Multisystem inflammatory syndrome in children |
| MRI | Magnetic resonance imaging |
| MOGAD | MOG-associated disorder |
| MOG | Myelin-oligodendrocyte glycoprotein |
| SARS-CoV-2 | Severe acute respiratory syndrome coronavirus 2 |

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oligodendrocyte glycoprotein (MOG) antibodies (9 adults and 3 children with encephalomyelitis or optic neuritis).[7-19]

The disease course in children with SARS-CoV-2 infection generally is less severe.[20,21] Approximately 10% of infected adults develop hypoxemic pneumonia and about 3% develop acute respiratory distress syndrome; however, respiratory symptoms are less frequent in children. In contrast, multisystem inflammatory syndrome in children (MIS-C) is now well-described as a SARS-CoV-2-related condition with estimated prevalence of 1-2 per 10 000 infected children.[22,23] Some patients with MIS-C present with a neurologic inflammatory disease, among other clinical symptoms. However, data focusing on neurologic issues in children with SARS-CoV-2 infection are limited.[5,6] We report a case series of 19 children who presented with neurologic symptoms in association with SARS-CoV-2 infection and had a final diagnosis of neurologic inflammatory disease with or without associated MIS-C.

## Methods

We performed a monocentric retrospective chart review of pediatric patients referred to the Necker-Sick Children Hospital in Paris, France, between January 1, 2020, and July, 1, 2021. Inclusion criteria were age less than 18 years, neurologic signs, and positive testing for SARS-CoV-2 infection by reverse transcription PCR performed less than 6 weeks before onset of neurologic symptoms or a seroconversion following the symptoms with a prior history of SARS-CoV-2 exposure. MIS-C was defined according to the 2020 World Health Organization definition.[24] Data were collected by screening of clinical, laboratory, and radiologic records. For the study and to ensure homogeneity, 1 physician and 1 radiologist reassessed clinical and radiological data retrospectively.

SARS-CoV-2 PCR testing was performed on nasopharyngeal swab specimens using the Abbott Real Time SARS-CoV2 assay (Abbott). Until February 2021, the SARS-CoV-2 immunoglobulin (IgG) II antibody test (Abbott) was used to detect IgG against the nucleocapsid protein. Sera with a ratio of 0.5 or higher were considered as positive according to the manufacturer recommendations. From February 2021, the SARS-CoV-2 IgG II Quant antibody test (Abbott) was used to quantify IgG against the spike protein. Results of 50 UA/mL or greater were considered as positive according to the manufacturer's recommendations. The presence of IgG oligoclonal bands in the cerebrospinal fluid (CSF) was determined using isoelectrofocusing on agarose gel performed on the semi-automatic HYDRASYS system (Sebia). Anti-aquaporin 4 (AQP-4) and anti-MOG antibodies were detected in serum using a cell-based assay by indirect immunofluorescence, following manufacturers' instructions (Euroimmun). The biological activity of alpha interferon in the CSF was measured as the protection conferred by serial CSF dilutions to cultured cells towards vesicular stomatitis virus, as previously described.[25]

Brain magnetic resonance imaging (MRI) and spine MRI were performed in all the patients, including systematically 3-dimensional T1-weighted imaging, T2 and T2 fluid-attenuated inversion recovery weighted imaging, diffusion-weighted imaging, sagittal T2-weighted imaging of the spine, and postcontrast T1-weighted imaging of both brain and spine.

Data management was authorized by the National Commission for Computing and Liberties to the Public Assistance Hospital of Paris (N°1980120). All patients received a written information regarding the use of their clinical data for scientific studies and publications according to national legislation.

Descriptive statistics were used for all study variables and expressed as median and range values, and categorical data were expressed as proportions (%).

## Results

From January 1, 2020, to July 1, 2021, 21 children with a CNS inflammatory encephalopathy in the context of SARS-CoV-2 infection were referred to the Necker-Sick Children Hospital in Paris from 12 pediatric departments in the Paris area. Admission of 2 of these children was related to a severe co-infection with another pathogen (1 patient with varicella zoster virus and *Staphylococcus aureus* infection and 1 patient with *Fusobacterium necrophorum* infection) with systemic features and died with multiple infectious and vascular complications; these cases have been reported previously.[26] We describe here 19 other children who had no co-infection and had a final diagnosis of CNS inflammatory disease related to SARS-CoV-2 infection.

The median age was 8.7 years (range, 1.4-15.3 years); there were 12 girls. None of the children presented with a medical history of severe infection, vaccine complication, or an underlying neurologic abnormality. One child had a history of systemic-onset juvenile idiopathic arthritis and was free from treatment for many years. Another child had sickle cell disease and received bone marrow transplant 2 weeks before the SARS-CoV-2 infection. No child had received a SARS-Cov-2 vaccine (which was not available during this period). Six of the 19 children did not display any SARS-CoV-2 infectious symptoms, and the other 13 presented with fever, abdominal pain, diarrhea, headache, and/or asthenia (including all the patients with MIS-C).

An MIS-C phenotype (according to the World Health Organization definition) was present in 6 children, with fever, laboratory evidence of systemic inflammation, and symptoms of myocardial dysfunction occurring at a median of 3.5 days (range, 3-5 days) after the onset of general symptoms.[24] All 6 children with MIS-C showed elevated cardiac enzymes and myocardial dysfunction; 2 patients required a dobutamine infusion for less than 48 hours. Thirteen children (68%) had neurologic disease absent MIS-C. In children with systemic symptoms, neurologic symptoms occurred at a median of 3 days (range, 1-15 days): 10 children had cerebellar symptoms (ataxia, dizziness) and 9 had impaired cognitive functions or altered consciousness. Other sporadic symptoms were loss of vision, parasthesia or hyperesthesia, sphincter disorder, or facial palsy.

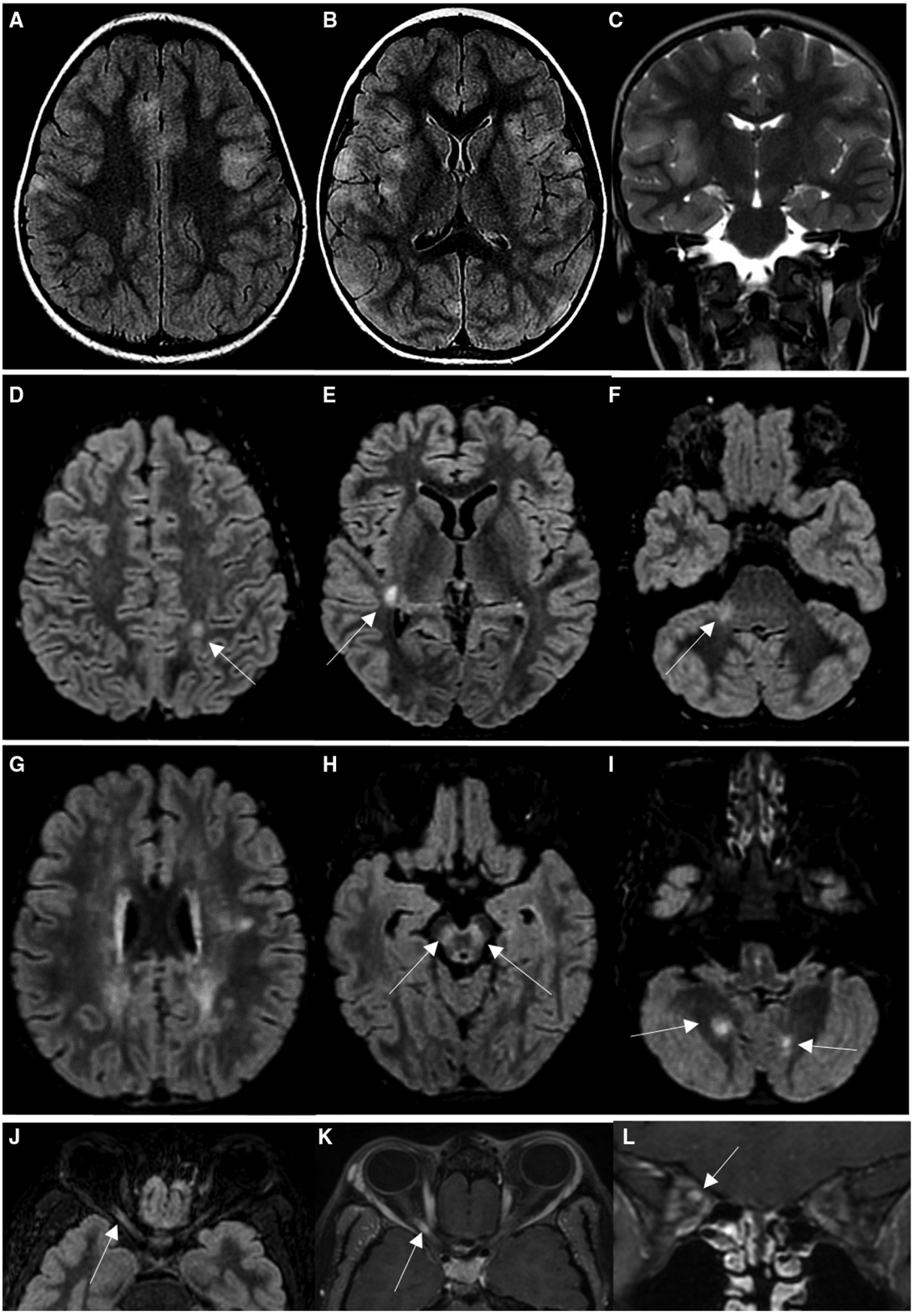


**Figure 1.** Demyelinating disorders in MRI in children associated with SARS-COV-2. MOGAD (top row). **A-C,** Multifocal and asymmetric cortical T2-fluid-attenuated inversion recovery (FLAIR) hyperintensities **A, B,** readily appreciated on coronal T2-weighted images, **C**. MOGAD (middle row). **D-F**, Small multiple white matter lesions, supratentorial and infratentorial on T2-FLAIR weighted images. MOGAD (middle row). **G-I,** Large confluent white matter lesions **G,** with substantia nigra involvement **H,** and small cerebellar lesions on T2-FLAIR-weighted images. AQP4-neuromyelitis optica spectrum disorder—bottom row. **J-L,** Prechiasmatic right optic nerve hyperintensity on axial T2-FLAIR **J,** with focal enhancement on axial **K,** and coronal **L,** contrast-enhanced T1-weighted images, suggesting right optic neuritis.

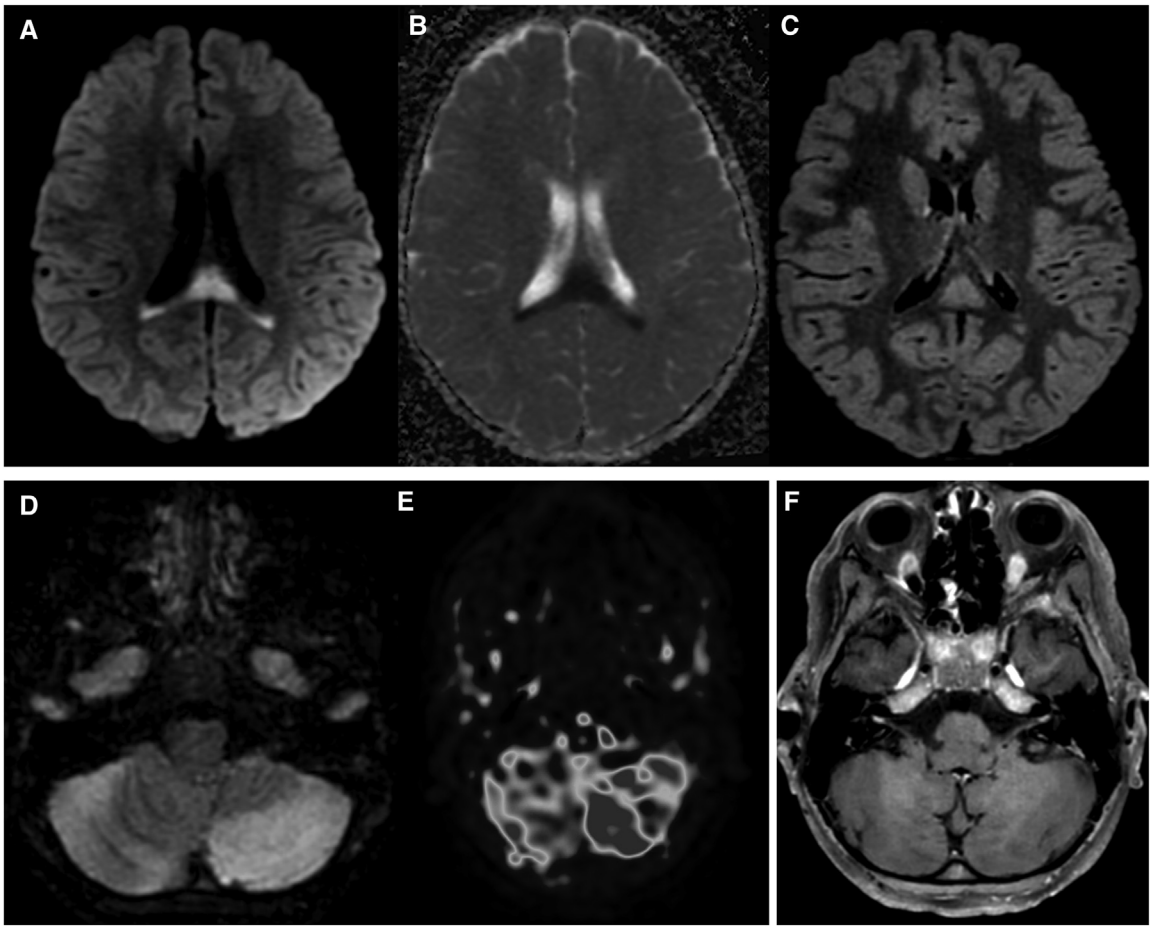


**Figure 2.** Other MRI inflammatory disorders in children associated with SARS-COV-2. Cytotoxic lesions of corpus callosum syndrome (CLOCCs) (top row). **A-C,** Well-limited restricted diffusion in the splenium of corpus callosum with low apparent diffusion coefficient values **A, B,** and T2 fluid-attenuated inversion recovery (FLAIR) hyperintensity **C,** suggestive of CLOCCs after SARS-CoV-2 infection. Cerebellitis (bottom row). **D-E,** Large and asymmetric cortical and white matter T2-FLAIR hyperintensities of the cerebellum **D,** with high blood flow on an arterial spin labeling sequence **E,** suggesting acute cerebellitis. Bilateral facial neuritis (bottom row). **F,** Bilateral enhancement of the meatal segment of facial nerves in internal auditory canal, bilaterally.

All patients had a history of SARS-CoV-2 exposure before the disease, and all tested positive for circulating antibodies against SARS-CoV-2 during and/or after disease. At the onset of symptoms (systemic or neurologic), SARS-CoV-2 PCR (nasopharyngeal swabs) were positive in 8 children (17 tested), including 1 with MIS-C. All children had other negative viral PCR in nasopharyngeal swabs (multiplex testing for parainfluenza, syncytial respiratory virus, metapneumovirus, rhinovirus, enterovirus, and other coronaviruses).

CSF examination was performed for all patients at a median of 8 days (range, 0-17 days) after the onset of the neurologic symptoms. Pleocytosis was present in 11 of 19 children (median, 23 white blood cells/$\mu$L; maximum 300, with a predominance of lymphocytes or neutrophils). PCR for *Listeria monocytogenes*, cytomegalovirus, enterovirus, herpes simplex virus 1 and 2, human herpes virus 6, parechovirus, and varicella zoster virus were negative in the CSF for all children. Only 1 of 19 patients had an elevated protein in the CSF (≥50 mg/dL). CSF oligoclonal bands were identified in 2 patients of 10 tested. When tested in 9 patients, interferon alpha secretion in CSF was always negative. Cytokines (interleukin [IL]1, IL6, IL10, tumor necrosis factor) in CSF were measured in 9 patients and were abnormal in 3 patients: isolated increased IL6 (1118 and 1551 pg/mL, 200-fold normal) in 2 children; increase of all 3 cytokines of 10-fold or more above normal in 1 child.

The MRI was abnormal in 14 of 19 patients. Four MRIs showed ADEM with multifocal brain lesions (1 had associated optic neuritis); 3 showed cytotoxic lesions of corpus callosum with a well-limited restricted diffusion area in the splenium of the corpus callosum and T2-fluid-attenuated inversion recovery hyperintensities, 2 showed cerebellitis; 1 each showed isolated optic neuritis, an isolated facial neuritis and multineuritis (Figure 1). Five of 19 MRI of the spine were abnormal: 5 showed myelitis, of which 3 were associated with ADEM, 1 was isolated, and 1 was associated with contrast enhancement of the nerve roots of

the cauda equina. All cases of myelitis showed involvement of more than 3 vertebral bodies (Figure 1).

Anti-MOG and anti-AQP4 antibodies were positive in serum in 4 of 10 children tested (Table; available at www.jpeds.com). One girl with positive anti-AQP4 antibodies was 14 years old and had no significant past medical history. She had isolated visual symptoms in the right eye. Her neurologic examination was normal. A diagnosis of optic neuritis was made clinically and confirmed by MRI. Her SARS-CoV-2 nasal PCR was positive without general symptoms. An MRI of the brain showed optic neuritis without brain or spine abnormalities (Figure 2). The other 3 children with positive anti-MOG antibodies were 1.5, 4.0, and 10.0 years old and 2 were boys; 2 had no general symptoms of SARS-CoV2 infection. In all 3 cases, a SARS-CoV-2 positive history was found in a close relative 1 month before the child's symptoms. None of the children had MIS-C symptoms. They presented with neurologic symptoms (2 with ataxia and 1 with seizures, loss of consciousness, facial palsy, and hemiparesis). The third patient required hospitalization in the critical care unit. MRI showed ADEM-like patterns, confirming MOG-associated disorder (MOGAD) diagnosis: 1 with multifocal and asymmetric cortical lesions, 1 with multiple supratentorial and infratentorial white matter lesions, and 1 with confluent white matter lesions as well as substantia nigra involvement and small cerebellar lesions (Figure 2).

Two children among the 19 had no anti-inflammatory treatment. Among the others, 14 received high-dose pulse of methylprednisolone (12 children 30 mg/kg/d ×3 days, maximum = 3000 mg; 2 children 10 mg/kg/d ×3 days), 16 were given corticosteroids orally between 1 and 2 mg/kg/d for 1-3 months; 11 received intravenous immunoglobulins (2 g/kg), and a few received other immune-regulatory treatments (1 each was given anti-IL1, remdesivir, anti-IL6, plasma exchange, and rituximab). All the children had favorable outcome with an Expanded Disability Status Scale and a modified Rankin Score of zero at 1 month after the disease with no relapse identified. Three of the 4 children with the anti-MOG and anti-AQP4 autoantibodies received a high-dose pulse of methylprednisolone. All recovered completely at 1 month (Expanded Disability Status Scale score of 0, modified Rankin Score of 0) and no relapse was reported at last follow-up beyond 6 months. However, anti-AQP4 (n = 1/1) and anti-MOG (n = 2/3) autoantibodies remained positive in the serum. A follow-up MRI was performed between 3 and 6 months later if initially abnormal and all had normalized.

## Discussion

We report a large case series of children with nonspecific neurologic symptoms as well as neuroinflammatory disease related to SARS-CoV-2 infection (with or without associated MIS-C), expanding the clinical and radiologic spectrum of manifestations. Eight children had cerebellar symptoms, 2 had specific CNS symptoms (optic neuritis and central facial palsy), and another 5 had more diffuse symptoms of encephalopathy (isolated cognitive or consciousness impairment) or ADEM (motor and cognitive impairment). The MRI was normal in 5 children, but cytotoxic lesions of corpus callosum (n = 3) and ADEM (n = 4) were frequent. Neurologic symptoms occurred a median of 3 days after systemic symptoms (fever) but 31% (6/19) had no fever or prior signs of SARS-CoV-2 infection. Six children had a co-occurrence of a MIS-C, without specificity in their neurologic signs. These results are concordant with previously described cases in adults or children, except we did not find any evidence of acute hemorrhagic leukoencephalitis on MRI in our series.

The effects of SARS-CoV-2 infection include neurologic disorders ranging from nonspecific encephalopathy and stroke, as well as presumed infectious or postinfectious inflammatory disease, such as Guillain-Barre syndrome, vasculitis, ADEM, myelitis, and encephalitis. In a case series of COVID-19 patients from Wuhan, China, neurologic symptoms were observed in 36.4% of patients but neurologic symptoms generally were nonspecific and poorly documented: dizziness, headache, impaired consciousness, acute cerebrovascular event, and ataxia.[27] In a report of 43 patients from London, with confirmed, probable, or possible SARS-CoV-2 infection, 12 had a confirmed inflammatory CNS disease (2 encephalitis, 9 ADEM, 1 myelitis) and none had specific antibodies identified in the serum or CSF.[3] Le et al collected the data of hospitalized adults with SARS-CoV-2 infection from 338 hospitals in 6 countries.[28] The mean increase in the proportion of patients with impairment of consciousness was 5.8% and 8.1% for other disorder of the brain during the pandemic. The increase of encephalitis, myelitis, and encephalomyelitis was not significant. In a systematic review of ADEM and acute hemorrhagic leukoencephalitis with SARS-CoV-2 infection, Manzano et al analyzed 46 patients from 26 case reports or series from 8 countries. The median age was 49.5 years (6 patients were <20 years old).[12] ADEM occurred in 31 cases and acute hemorrhagic leukoencephalitis in 15. Only 1 patient was positive for anti-MOG (a 13-month-old child) and none was positive for anti-AQP4 (only 15 patients were tested for both antibodies). The final modified Rankin score was 4 or more in 64% of, patients including 32% who died. Abdel-Mannan et al reported 4 children with MIS-C and neurologic symptoms (confusion, agitation, dysarthria, and weakness) associated with cytotoxic lesions of corpus callosum syndrome by MRI.[29] Three of the 4 children were tested for *N*-methyl-d-aspartate receptor, MOG, and AQP4 antibodies and were negative. However, the determination of association between viral disease and neuroinflammatory diseases requires a combination of historical control and large patient registries.[30]

MOGAD is a distinct demyelinating disorder with a phenotype and an outcome different from that of multiple sclerosis and neuromyelitis optica spectrum disorder. Twelve cases of MOGAD have been associated with COVID-19 in the literature, mostly in adult males (8/12) with optic neuritis or encephalomyelitis.[7-11,13-19] Our case series confirms that the onset of MOGAD can be triggered by SARS-CoV-2 infection and that 16% (3/19) of children with a neuroinflammatory

disease triggered by SARS-CoV-2 had MOG antibodies. Moreover, considering only children with radiologic criteria of MOGAD, the MOG autoimmune cause represents 3 of 7 patients (43%). For these 3 patients, the follow-up, which lasted from 12 to 18 months, did not show any relapse and MOG antibodies remained positive 1 year after onset in 2 of the children. One girl with AQP4 antibodies had optic neuritis, but was otherwise asymptomatic upon SARS-CoV-2 infection. Considering that 4 of our patients met the criteria of respectively MOGAD (for MOG antibodies) and neuromyelitis optica spectrum disorder (for AQP4 antibodies), we can hypothesize that these autoantibodies likely are the cause of neurologic symptoms and not only the consequence of a hyperinflammatory response. In a report of serial serum analyses in 67 children with MOG antibodies published by Waters et al, 57% were seropositive at onset and later became seronegative (median time to conversion, 1 year).[31] Among all participants who were positive for anti-MOG antibodies at presentation in that study, clinical relapses occurred in 9 of the 24 children (38%) who remained persistently seropositive and in 5 of the 38 children (13%) who converted to seronegative status. However, AQP4 autoimmunity is rarer in children than MOGAD. It is possible that SARS-CoV-2 infection associated with AQP4 antibodies remains rare. One year after optic neuritis, our patient with AQP4 antibodies remained positive, although she had no neurologic relapse.

In a model of virus-induced encephalomyelitis in mice with MOG autoantibodies, Burrer et al showed that the presence of the autoantibodies resulted in increased infiltration of mononuclear cells into the brain.[32] Moreover, an early severe demyelinating process was increased in the brains and spinal cords of infected mice. The authors' hypothesis was that viral infections could lead to more profound immunopathology in the presence of some latent autoimmune condition. In the literature, the association of positive MOG antibodies with a viral infection is rare: only 13 cases have been described in association with the widespread Sars-CoV-2 infection.

Even though SARS-CoV-2 presentation is dominated by pulmonary disease in adults, pediatric clinical forms often are characterized by postinfectious nonpulmonary inflammatory or immune-mediated responses. The rare occurrence of these conditions suggests the possible existence of underlying predisposing factors in the host. More studies are needed to seek genetic variants in individuals susceptible to these conditions during SARS-CoV-2 infection and to better understand the root cause of the aberrant hyperinflammatory response. ■

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**Table. Clinical, radiologic, and laboratory characteristics of the cohort**

| Patients | Age at disease (y) | Sex | Medical history | Systemic and respiratory symptoms of covid | Cardiac symptoms | Neurologic symptoms | SARS-CoV-2 PCR | SARS-CoV-2 serology | Treatment |
|---|---|---|---|---|---|---|---|---|---|
| P1 | 10.6 | Male | 0 | Fever, asthenia | 0 | Ataxia, sphincter dysfunction, pyramidal signs | Positive | Positive | No |
| P2 | 1.6 | Male | 0 | 0 | 0 | Ataxia | Negative | Positive | MP pulse |
| P3 | 4.2 | Female | 0 | 0 | 0 | Seizure, impaired consciousness, facial palsy, hemiparesis | Negative | Positive | MP pulse IVIg |
| P4 | 14.9 | Female | Juvenile arthritis | 0 | 0 | Low visual acuity (right eye) | Positive | Positive | MP pulse |
| P5 | 5.4 | Male | Hyperthermic seizure | Fever, rash | Cardiac dysfunction | Seizure, impaired consciousness | Negative | Positive | MP pulse IVIg anti-IL1 |
| P6 | 5.6 | Female | Sickle cell disease, bone marrow transplantation | Cough then acute respiratory distress syndrome | 0 | Bilateral facial palsy | Positive | N/A | IVIg, remdesivir, anti-IL6 |
| P7 | 7.3 | Female | 0 | Fever, vomiting | 0 | Ataxia | N/A | Positive | MP pulse |
| P8 | 8.7 | Female | 0 | Abdominal pain, diarrhea, headache, fever | Cardiac dysfunction | Ataxia, cognitive impairment, agitation | Positive | Positive | MP pulse, IVIg |
| P9 | 5.0 | Male | 0 | Fever, rash | Cardiac dysfunction | Cognitive impairment, hallucinations | Negative | Positive | MP pulse, IVIg |
| P10 | 7.3 | Male | Thrombocytopenic purpura | Fever, diarrhea | 0 | Seizure, ataxia | Positive | Positive | MP pulse, IVIg |
| P11 | 13.9 | Female | Cimeterre syndrome | Fever, abdominal pain | Cardiac dysfunction | Trismus, diffuse pain, cognitive impairment | Negative | Positive | IVIg, MP pulse |
| P12 | 7.5 | Male | 0 | Abdominal pain, headache | Cardiac dysfunction | Ataxia, cognitive impairment | Negative | Positive | MP pulse, IVIg |
| P13 | 9.2 | Female | 0 | Fever, vomiting | Cardiac dysfunction | Impaired consciousness, agitation, distended bladder | Negative | Positive | MP pulse, IVIg |
| P14 | 13.3 | Female | 0 | Fever, diarrhea, abdominal pain | 0 | Cognitive impairment, unilateral facial palsy | Negative | Positive | 0 |
| P15 | 1.4 | Female | 0 | Fever, diarrhea, rash | 0 | Agitation, trismus, diffuse pain | Negative | Positive | PLEX, RTX, MP pulse, IVIg |
| P16 | 10.7 | Female | 0 | Headache, dizziness | 0 | Ataxia, paresthesia, distended bladder | Positive | N/A | MP pulse |
| P17 | 10.2 | Female | 0 | 0 | 0 | Ataxia, cranial nerve palsy | Positive | Positive | MP pulse, IVIg |
| P18 | 13.0 | Female | 0 | 0 | 0 | Ataxia | Positive | N/A | Oral steroids |
| P19 | 15.3 | Male | 0 | 0 | 0 | Headache, cognitive impairment ataxia | *U* | Positive | MP pulse |

*CLOCC*, cytotoxic lesions of corpus callosum; *IVIg*, intravenous immunoglobulin; *MP*, methylprednisolone; *N/A*, not available, *PLEX*, plasma exchange, *RTX*, rituximab, *TNF*, tumor necrosis factor.

| Day of lumbar puncture after onset of neurologic symptoms | CSF protein (g/L) | CSF leucocytes (cell/mm$^3$) | Oligoclonal bands | Positive autoantibodies | CSF IL1 (pg/mL) | CSF IL6 (pg/mL) | CSF IL10 (pg/mL) | CSF TNF (pg/mL) | CSF IFN (UI/mL) | MRI result |
|---|---|---|---|---|---|---|---|---|---|---|
| 8 | 0.29 | 9 | 0 | MOG | 7.5 | 2.1 | <2 | 41.7 | 0 | ADEM |
| 9 | 0.51 | 300 | N/A | MOG | 8 | 1551 | 7.2 | 7.4 | 0 | ADEM + optic neuritis |
| 2 | 0.2 | 23 | 0 | MOG | N/A | N/A | N/A | N/A | N/A | ADEM |
| 7 | 0.2 | 8 | 0 | AQP4 | 23.8 | <2 | 1.7 | 22.1 | 0 | Optic neuritis |
| 7 | 0.28 | 50 | N/A | N/A | <2 | 1118 | 135 | N/A | N/A | Normal |
| 3 | 0.3 | 66 | N/A | N/A | N/A | N/A | N/A | N/A | N/A | Contrast enhancement of cranial nerves |
| 5 | 0.19 | 15 | Positive | N/A | 125 | 0 | 0 | *U* | 0 | Normal |
| 3 | 0.2 | 4 | N/A | N/A | N/A | N/A | N/A | N/A | N/A | Normal |
| 1 | 0.39 | 21 | N/A | N/A | N/A | N/A | N/A | N/A | N/A | Normal |
| 8 | 0.32 | 2 | 0 | 0 | 7.9 | 0 | 0 | 0 | 0 | CLOCCs |
| 0 | 0.17 | 0 | N/A | N/A | N/A | N/A | N/A | N/A | N/A | Myelitis |
| 0 | 0.15 | 0 | 0 | N/A | N/A | N/A | N/A | N/A | 0 | CLOCCs |
| 0 | 0.26 | 13 | N/A | N/A | 171 | 273 | 721 | 357 | 0 | CLOCCs |
| 0 | 0.18 | 0 | 0 | N/A | N/A | 10 | 0 | 0 | 0 | Contrast enhancement of peripheral and cranial neve roots |
| 3 | 0.27 | | 0 | 0 | 71 | 0 | 0 | N/A | N/A | Myelitis and contrast enhancement of peripheral nerve roots |
| 17 | 0.7 | 165 | N/A | 0 | N/A | N/A | N/A | N/A | N/A | Myelitis and ADEM |
| 2 | 0.3 | 4 | 0 | 0 | N/A | N/A | N/A | N/A | 0 | Cerebellar inflammation |
| 16 | 0.21 | 2 | N/A | N/A | N/A | N/A | N/A | N/A | N/A | Normal |
| 4 | 1.7 | 110 | Positive | N/A | N/A | N/A | N/A | N/A | N/A | Cerebellar inflammation |