Myelin Oligodendrocyte Glycoprotein (MOG) Antibody Disease in a 11-year-old child with COVID 19 infection

Archana Khan (arch.agr@gmail.com)
NH SRCC Children's Hospital, Mumbai, India  https://orcid.org/0000-0003-0842-2198

Hiren Panwala
NH SRCC Children's Hospital, Mumbai, India  https://orcid.org/0000-0003-3718-8708

Divya Ramadoss
NH SRCC Children's Hospital, Mumbai, India  https://orcid.org/0000-0002-0782-6142

Raju Khubchandani
NH SRCC Children's Hospital, Mumbai, India  https://orcid.org/0000-0003-4543-901X

Case Report

Keywords: COVID 19, Myelin Oligodendrocyte Glycoprotein Antibody Disease, Optic Neuritis

DOI: https://doi.org/10.21203/rs.3.rs-104308/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License
Abstract

Optic Neuritis is the most common presentation of MOG Antibody Disease (MOG-AD). We share our experience with a 11-year-old boy who developed MOG associated Optic Neuritis temporally associated with SARS-CoV-2 infection. He responded well to intravenous methylprednisolone therapy followed by oral prednisolone. While various neurological and ophthalmological manifestations of COVID-19 have been described previously, there are few case reports of Optic neuritis associated with COVID-19. Our case further supports the evidence to suggest that SARS CoV-2 is another such virus that triggers MOG-AD.

Introduction

Myelin oligodendrocyte glycoprotein (MOG) is a glycoprotein located on the myelin surface in the central nervous system. Optic Neuritis is the most common presentation of MOG Antibody Disease (MOG-AD) seen in 54–61% of patients and is followed by myelitis. (1). Isolated optic neuritis though rare have higher rate of bilateral presentation in children as compared to adults. Common causes include autoimmune diseases such as systemic lupus erythematosus or MOG-AD, post or para infectious conditions, granulomatous disease, paraneoplastic disorders, and demyelination. (2)

Case Report

We share our experience with a 11-year-old boy who presented with a rapidly progressive loss of vision. Four days after a brief episode of fever, he complained of redness and painful movement of both eyes which resolved with topical treatment prescribed by his family physician. Two weeks later, he presented to us with a 3-day history of rapidly progressive loss of vision in the right eye. There were no other systemic or neurologic complaints. On physical examination he had bilateral conjunctival congestion, painful eye movement and no perception or projection of light in the right eye. Pupils were bilaterally equal and reactive. Rest of the neurological system including cranial nerves was normal.

Detailed ophthalmological examination revealed a vision of <N/36 and disc edema with grade 3 Relative Afferent Pupillary Defect in the right eye. Vision in the left eye was 6/9 and rest of the eye examination was within normal limits.

Blood counts revealed hemoglobin 13 g/dL, white cell count 6.2 x 10^3/µL (neutrophils 35%, lymphocytes 44%, monocytes 9%), platelets 456 x 10^3/µL, ESR 13 mm/hour and CRP was 0. SARS-CoV-2 nasopharyngeal swab was positive by Cartridge-Based Nucleic Acid Amplification Test (CBNAAT). Serum anti-SARS-CoV-2 IgG antibodies were reactive 20.8 (Normal range <1.0). Serum myelin Oligodendrocyte Glycoprotein (MOG) antibody was positive and Aquaporin 4 antibody was not detected. CSF showed proteins 10 mg/dL (normal 15-60 mg/L), glucose 60 mg/ dL (40 - 70 mg/dL). CSF white blood cells were elevated at 55 cells/mL (normal < 5 cells/ mL). CSF Oligoclonal bands, MOG- IgG and bacterial cultures were negative. CSF- SARS- CoV-2 RNA PCR was not done due to lack of availability.
MRI findings were consistent with MOG antibody associated disease (3). There was bilateral asymmetrical optic neuritis, right > left, involving the intra orbital and intracanalicular portion on right side and intra orbital portion on the left side. The swelling and altered signal intensity were long segmental and diffuse in the right intra orbital optic nerve and patchy in the left intra orbital optic nerve. There was associated enhancement of the optic nerve sheath in the right orbit. The chiasmatic, retro-chiasmatic portion as well as optic tracts were normal in signal intensity and appearance on both sides. (Figure 1) No restricted diffusion was noted within the optic nerves. Rest of the brain and spine did not show lesions in the white or grey matter. No abnormal brain parenchymal or meningeal post contrast enhancement was seen. We thus concluded a diagnosis of isolated bilateral optic neuritis associated temporally with SARS CoV-2 infection. There was a drastic positive response to 5 days of intravenous methylprednisolone administered at 30mg/kg followed by oral prednisolone at 2 mg/kg/day. Throughout the hospital stay the child remained stable and there was no evolution of any other features. After 10 days of treatment, right eye vision improved to 6/9 and left eye vision was 6/6. Slit lamp and fundus examination was normal. Prednisolone is being slowly tapered over 3 months.

**Discussion**

SARS-CoV-2 causes a multipronged multiorgan damage through direct and /or immune mediated injury through release of cytokines. While the respiratory system involvement is the most recognized and common life-threatening manifestation, there is increasing evidence of extra-pulmonary involvement of SARS CoV-2, especially in organs with high angiotensin-converting enzyme 2 (ACE 2) receptor expression including the nervous system and eye (4). Multiple studies have found various ophthalmic manifestations such as hyperaemia, chemosis, epiphora, and increased secretion (5), however conjunctivitis, uveitis, retinitis and optic neuritis have been reported in feline and murine models (6). While, neurological manifestations of hyposmia, hypogeusia, headache, dizziness, altered mental status and GBS have been well established (4), there have been three case reports of SARS CoV-2 associated optic neuritis in a 15 year old boy with anti MOG-IgG positive NMOSD, a 26 years old man with bilateral MOG associated optic neuritis and myelitis and a 29 year old woman with Non MOG associated Optic Neuritis (7-9). Anti MOG associated ON is also frequently associated with viral infections (9-10) and our case adds to the body of evidence that SARS CoV-2 is another such virus that triggers MOG-AD.

**Declarations**

**Consent and declaration for publication fee:**

A written informed consent was obtained from the family to publish this manuscript.

**Funding:**

No specific funding was received from any bodies in the public, commercial or not-for-profit sectors to carry out the work described in this article.
Conflict of interest:

Authors declare no conflicts of interest

Contribution of each author:

Dr Archana Khan, Dr Divya Ramadoss and Dr Raju Khubchandani, were involved in collecting patient’s data and in the clinical management of this patient. Dr Archana Khan, Dr Hiren Panwala, Dr Divya Ramadoss and Dr Raju Khubchandani were involved in conducting literature search, drafting the manuscript, and in revision of the scientific content. Dr. Hiren Panwala has conducted and reported on the MRI of brain and spine of this child and drafted parts of the manuscript related to the findings.

Acknowledgement

Dr Hitendra Mehta diagnosed Optic Neuritis in the child and is currently following up for the same.

S Ophthalmology, F.M.R.F (Vitreoretinal Fellowship Shankar Netralaya, Chennai)

Medical director

Vitreoretinal surgeon

Infiniti Eye Hospital, Spencer Building, Forjett Mumbai 400036, Maharashtra, India

Bibliography


