Case Report

Myelin oligodendrocyte glycoprotein antibody-associated disease: A central nervous system demyelinating disorder – Case report

Shraddha Mahadev Parab[1], Sanika Sanjay Gawade[1,2], Ankita Raghunath Dharne[1,2], Shruti Dinesh Jadhav[1,2], Sakshi Rajan Chavan[1,2], Kirti Satish Gawade[1,2], Tejas Pravin Oak[1,2], Vijay Arjun Jagtap[1,3]

From [1]Assistant Professor, [2]Final Year B. Pharm Student, Department of Pharmacology, Yashwantrao Bhonsale College of Pharmacy, Sawantwadi, Maharashtra, India, [3]Professor and Principal, Department of Pharmaceutical Chemistry, Yashwantrao Bhonsale College of Pharmacy, Sawantwadi, Maharashtra, India

ABSTRACT

MOGAD, or Myelin Oligodendrocyte Glycoprotein Antibody-associated Disease, is a less-known demyelinating disease that causes inflammation to the CNS (central nervous system) and has been known for a few recent years. It is observed to be in close association with many other neuroinflammatory diseases and is now recognized as a separate condition from Multiple sclerosis (MS) and neuromyelitis optica spectrum disorder (NMOSD). The disease's pathophysiology, treatment, and outlook differ from those for MS or NMOSD, which are concerned with aquaporin-4 antibodies. This study aims to get acquainted with the etiology, clinical manifestations, pathophysiology, epidemiology, diagnosis, and treatment of MOGAD. The study also involves a recent case study of MOGAD. The present study traversed a wide range of literature reviews by referring to articles published in international journals and available on internet sources like Google Scholar and PubMed. The case study refers to a female patient (age: 20 years) with a complaint of right eye vision blurring gradually progressive with mild right eye pain, who was subsequently diagnosed positive for MOGAD. There was no significant medical history, drug history, or medical history related to MOGAD. Treatment of attacks with corticosteroids was the first-line treatment, which was followed by preventive therapy with monoclonal antibodies, Rituximab in biannual cycles. This case study is presented to throw light on a rare disease like MOGAD and encourage further research related to the same.

Key words: MOG Antibody disease, demyelinating, neuroinflammatory, sclerosis, corticosteroids, Rituximab

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), also known as myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease, is a recently identified demyelinating inflammatory disease affecting the central nervous system (CNS). A broad variety of neuroinflammatory diseases, collectively known as MOG-Immunoglobulin G (IgG)-associated disorders (MOGAD), are associated with antibodies against MOG-IgG. The major MOGAD attacks are characterized by transverse myelitis, optic neuritis, and acute disseminated encephalomyelitis. A multiphasic relapsing condition can involve attacks that are singular or that gradually merge [1,2]. Previously thought to be a potential variant of the neuromyelitis optica spectrum disorder (NMOSD), MOGAD is now recognized to be a separate condition from both NMOSD and MS that encompasses a wide range of clinical manifestations [3]. Accurate diagnosis of MOGAD is crucial since the pathophysiology, treatment, and outlook for this disease differ from those for NMOSD or multiple sclerosis (MS) associated with aquaporin-4 antibodies. The associated clinical manifestations have a monophasic course or a relapsing course that can be confused with those of these conditions [4]. There are still difficulties with diagnosis, even with the growing acknowledgment of MOGAD as a separate illness and the development of sensitive and reliable MOG antibody testing techniques [5].

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CASE STUDY

A 20-year-old female patient was admitted with a complaint of blurred vision in the right eye, gradually progressive with mild right eye pain. The patient has a history of a left eye squint, for which surgical correction was performed in 2012. In addition, the patient was

Correspondence to: Shraddha Mahadev Parab, Yashwantrao Bhonsale College of Pharmacy, Maharashtra, Sawantwadi, India. E-mail: shraddhaparab530@gmail.com

diagnosed with typhoid at the age of 2 years and was successfully treated with antibiotics. In terms of drug history, the patient has been administered antibiotics for typhoid, uses minoxidil for hair regrowth, and occasionally consumes over-the-counter medications such as analgesics, antipyretics, and antacids.

Upon examination, the blood pressure was 130/80 mmHg, respiratory rate was 18/min, pulse rate was 78/min, and temperature was 98.4 °F. The patient was conscious, oriented, and obeying. On central venous system examination, S1 and S2 were normal. The chest was clear on respiratory system examination. Per abdomen was soft and non-tender.

Fundus photographs showing the retina, optic disc, and retinal vessels of a patient's eye. The initial fundus photograph shows a swollen optic disc with blurred margins, suggestive of papilledema or optic disc edema. The retinal vessels appear engorged and tortuous, possibly indicating raised intracranial pressure or inflammation, while the retinal background looks hazy, hinting at fluid accumulation. In addition, there are possible blot or flame-shaped hemorrhages near the macula, which itself is obscured, likely due to edema. These features are consistent with optic neuritis, papilledema, or other inflammatory causes of disc swelling, such as demyelinating conditions (Fig. 1a).

Due to the above-mentioned complaints, a cerebrospinal fluid (CSF) study was advised. All the necessary investigations were done and evaluated such as demyelinating lesion fundoscopy. For accurate comparative analysis, it is essential to collect both CSF and serum samples. Intrathecal synthesis within the CNS was indicated by the IgG bands in the immunofixation pattern of CSF that are not in the serum pattern of the same patient. It should be noted that the number of bands in the oligoclonal patterns does not correlate with the diagnosis of the disease, nor with severity and prognosis. Isoelectric focusing of CSF was tested to be positive for antibodies against MOG in serum. Urine routine report and microscopic examinations were normal. Anti-human immunodeficiency virus (HIV) antibodies report was normal, and the venereal disease research laboratory (VDRL) (rapid plasma reagin) report was found to be non-reactive. Electrolytes and urea reports were normal, but the hemogram report (white blood cells count, mean corpuscular hemoglobin [MCH], and MCH concentration) was found to be out of the biological reference interval. The eosinophil count was found to be 0%.

Magnetic resonance imaging (MRI) of the cervical spine reveals oval-shaped, T2 hyper-intensity in the posterior 2/3rd of the cervical cord at the C2 level on T2 axial images, likely representing a demyelinating lesion. Mild posterior protrusions were at C4–5, C5–6, and C6–7 levels. Multiple subretinal and pre-retinal hemorrhages were observed. In view of the presence of optic neuritis on the right and an enhancing T2 hyperintense lesion in the cervical cord, Neuromyelitis Optica/Devic's Disease needs consideration.

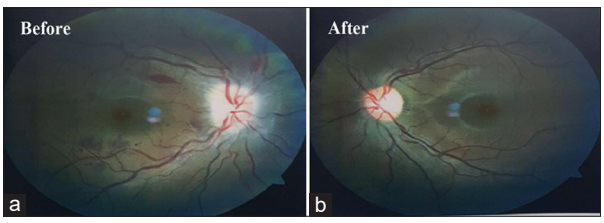

Figure 1: Fundus photographic images of a right eye (a) before and (b) after treatment

Antinuclear Antibody (ANA) blot profile did not detect ANA of IgG class against 18 different antigens associated with various autoimmune diseases such as atopic dermatitis, rheumatic diseases, systemic lupus erythematosus, primary biliary cirrhosis, drug-induced lupus, rheumatoid arthritis, calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia syndrome, polymyositis, interstitial lung fibrosis, overlap syndrome, progressive systemic sclerosis, Sjogren syndrome, disseminated lupus erythematosus, dermatomyositis, mixed connective tissue disease, and sharp syndrome.

The patient was administered injection Avil ½ ampoule (Pheniramine maleate), an antihistaminic used to manage allergic symptoms, and injection Effcorlin 100 mg stat (hydrocortisone sodium succinate), a corticosteroid given to reduce inflammation and swelling. For fever and pain, tablet Crocin (Paracetamol) was given as an analgesic and antipyretic. A planned infusion of injection Rituximab (December 2023) 1000 mg was administered in 500 mL of normal saline, distributed over 6 h in increasing concentrations: 50 mg in 25 mL during the 1st h, 100 mg in 50 mL during the 2nd h, 150 mg in 75 mL during the 3rd h, 200 mg in 100 mL during the 4th h, 250 mg in 125 mL during the 5th h, and 250 mg in 125 mL during the 6th h. In addition, injection Solumedrol 1 g IV (methylprednisolone sodium succinate), a corticosteroid, was given to further manage inflammation.

On discharge, the patient was prescribed tablet Pan 40 mg once daily (Pantoprazole), a proton-pump inhibitor to control gastric acidity; tablet Vibrania-D once daily, a multivitamin supplement; and tablet Dolo 650 mg as needed (paracetamol) for pain and fever relief. The next dose of this cycle of rituximab was administered 15 days after the first dose. Subsequent cycles of rituximab were scheduled in July 2023, January 2024, and August 2024.

Preventive treatment with rituximab aimed at avoiding recurring acute attacks and minimizing right-eye blurring over the course of time. Lifestyle changes such as reducing screen time and adopting a balanced diet were suggested to enhance the probable chances of a speedy recovery. After 4 cycles of rituximab, the patient was relatively stable, with a subsequent decrease in blurring of vision. Headache and dizziness are often encountered but are relieved with sufficient rest. The follow-up image demonstrates a resolved disc edema with clearly defined disc margins, though the optic disc now appears pale, indicating optic atrophy. The retinal vessels have returned to a normal caliber, the macula is clearly visible, and the

retinal background appears clearer with reduced signs of inflammation. This suggests recovery from the acute phase, possibly following treatment, but with residual optic nerve damage, a common outcome of prior optic neuritis or ischemic optic neuropathy (Fig. 1b).

DISCUSSION

This is a case of a 20-year-old female presenting with an acute onset, gradually progressive blurring of vision of the right eye, with mild pain. This raises a reasonable clinical suspicion of optic neuritis, a prototypical feature of demyelinating disorders of the CNS. Because she is not having headaches, vomiting, or weakness in her limbs, it suggests that the process is localized. She requires a thorough neurological examination. The presence of IgG oligoclonal bands in the CSF and absence from serum indicates intrathecal IgG synthesis and indicates some inflammatory process either in the CNS, more likely MS or NMOSD [6]. In the presence of MOG-positive antibodies in the serum, it is highly likely that the diagnosis will be MOG Antibody-Associated Disease (MOGAD)–a diagnosis on the NMOSD spectrum, but with distinct pathophysiology and distinct prognosis [7,8].

The MRI reported a T2 hyperintense lesion in the cervical cord at C2 with evidence of optic nerve involvement, all without significant compression, providing complete support for demyelinating etiology in origin [9]. In addition, a normal Brain MRI and absence of abnormalities in an array of tests, including routine CSF and urinalysis, a chest X-ray, and tests for infectious diseases (HIV, VDRL) will enhance the diagnosis. Rituximab, a CD20-specific monoclonal antibody, is currently the evidence-based treatment for relapsing MOGAD/NMOSD, particularly in individuals with recurrent or severe attacks [10,11]. Corticosteroids (solumedrol, effcorlin) administered with rituximab are used to help suppress the acute inflammatory response associated with active disease during the induction phase of oligodendrocyte specific immune-mediated inflammation. The patient had a good response to the treatment, with visual symptoms approximately 70% improved and reported to be relatively stable for the past few months, indicating stabilization and possible modulation of the disease course. A history of limited and stable visual blurring and intermittent headaches over time suggests ongoing disease with some residual deficits (which is not uncommon in terms of residual deficits in demyelinating diseases). The lifestyle advice to modify screen time and diet provides a holistic approach to recovery and healthy visual function.

In summary, this case illustrates the significance of early recognition and differentiation of demyelinating diseases (MS, NMOSD, MOGAD). Although the disease can be unpredictable in nature, the initiation of appropriate therapy and immunosuppression of the disease is likely to have a large impact on the patient's quality of life and functional status [11].

CONCLUSION

There is sufficient evidence to conclude that MOGAD is a distinct, non-demyelinating CNS disorder that has evolved in unique ways from AQP4IgG+NMOSD and MS. Important clinical MRI characteristics of MOGAD identify a group of patients who are fundamentally different from the aforementioned disorders, and they deserve timely recognition for diagnosis and treatment. MOG-IgG can be viewed as a very specific biomarker for MOGAD disease; however, there are a few caveats, as false-positive results may occur, and we must be meticulous in interpreting lower titers and atypical phenotypes. While prognosis is typically favorable, some individuals living with MOGAD may have very significant residual disability, presenting the compelling importance to prevent repeat attacks for those people living with recurrent disease.

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