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CASE REPORT

A Case of Myelinating Oligodendrocyte Glycoprotein Antibody-associated Optic Neuritis (MOG-ON)

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Abstract

Myelinating oligodendrocyte glycoprotein antibody-associated optic neuritis (MOG-ON) is a rare demyelinating disease of the optic nerve prone to clinical misdiagnosis and underdiagnosis, the uniqueness of which is that the patient exhibited a sudden onset of severe vision loss and ocular pain. We report a case of MOG-ON, which was confirmed by antibody testing. The patient, a 37-year-old female, presented with visual loss in the right eye and ocular pain of 4 days' duration. Optical coherence tomography (OCT) showed that the right optic nerve head was edematous.

The serum MOG antibody was 1:10. Prednisone treatment was administered, and visual acuity improved significantly after 1 week, and returned to normal at 3 months' follow-up. By reporting this case in detail, we hope to improve clinicians' understanding of MOG-ON and optimize the diagnosis and treatment plan.

Keywords: MOG-ON; Optic nerve; Diagnostic criteria; Treatment.

Introduction

Optic neuritis is commonly diagnosed in both ophthalmology and neurology clinics. With the increasing availability and widespread use of antibody testing, MOG-ON is being identified more frequently. However, diagnosing MOG-ON presents unique challenges, as it can often be misdiagnosed as more common forms of optic neuritis, such as those associated with multiple sclerosis (MS) or neuromyelitis optica spectrum disorders (NMOSD). This report describes a case of acute visual loss in the right eye, detailing the diagnostic process and treatment journey. The case underscores the importance of accurate diagnosis and the critical role of early and consistent hormone therapy in managing this condition.

Case Report/Case Presentation

Patient Wang, a 37-year-old female weighing 50 kg, presented to the hospital on October 14, 2024, complaining of decreased vision and ocular pain in the right eye for four days. She had a cold with a cough and no fever 3 weeks before admission. Four days before hospitalization, the patient experienced a blockage of vision in the right eye accompanied by pain upon eye movement. These symptoms gradually worsened, leading to a progressive decrease in visual acuity and eventual blurred vision. She visited the outpatient ophthalmology clinic, where her visual acuity in the right eye was undetectable, and her left eye had a visual acuity of 0.25 (uncorrected) and -5.00 DS=0.8 (corrected). Funduscopic examination and OCT revealed optic disc edema in the right eye (shown in Fig. 1 and 3a), and the relevant MRI images are also presented (shown in Fig. 4). She was subsequently admitted to the hospital.

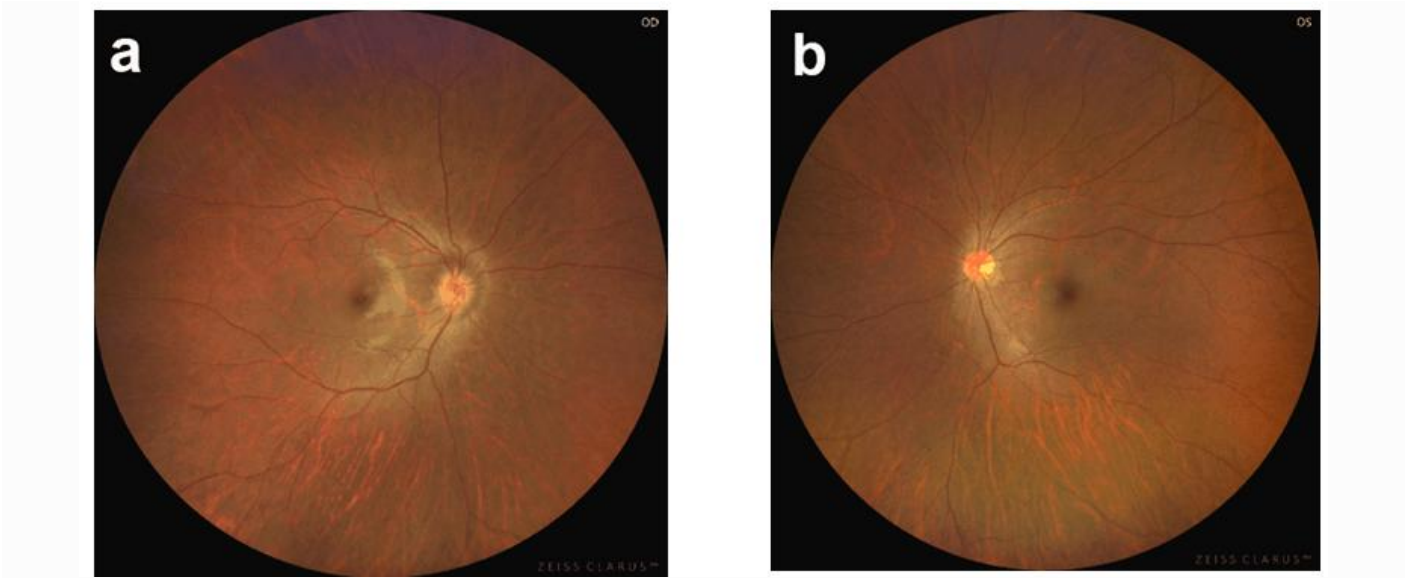

Figure 1 Fundus images of the right eye (a) and the left eye (b) before treatment, suggesting edema of the optic nerve papilla in the right eye

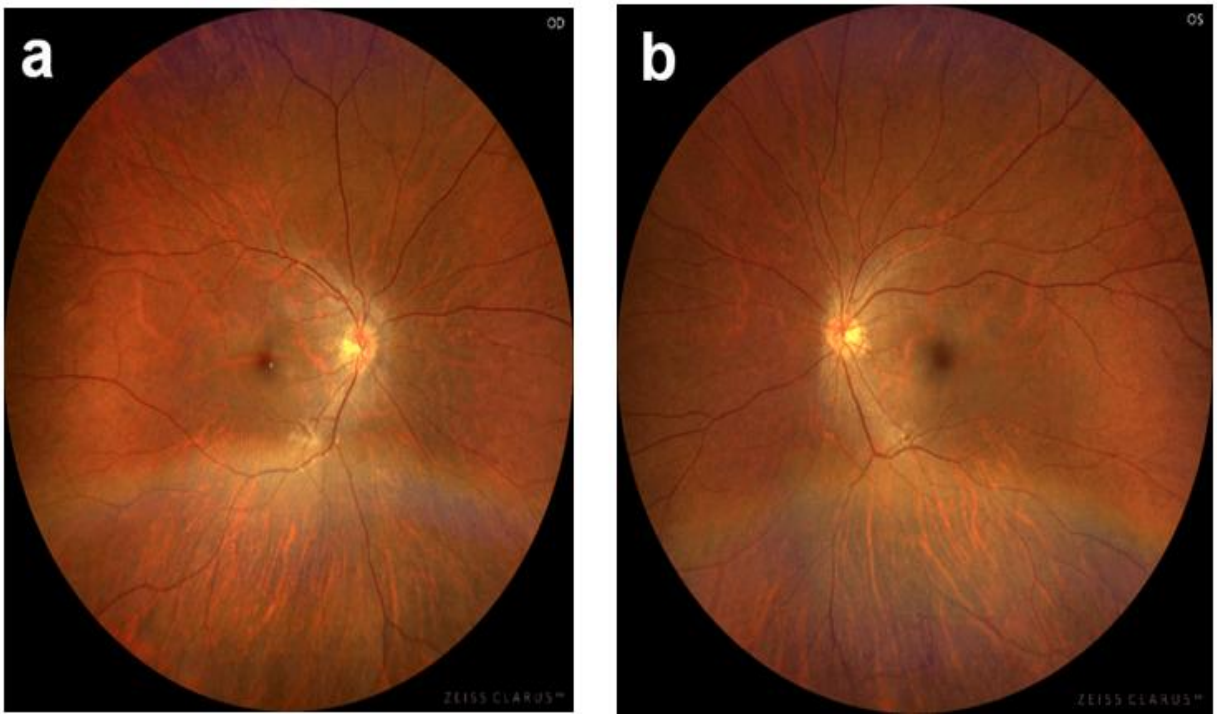

Figure 2 Fundus images of the right eye (a) and the left eye (b) after 1 week of treatment, demonstrating the edema of the optic disc of the right eye has decreased to a greater extent than before

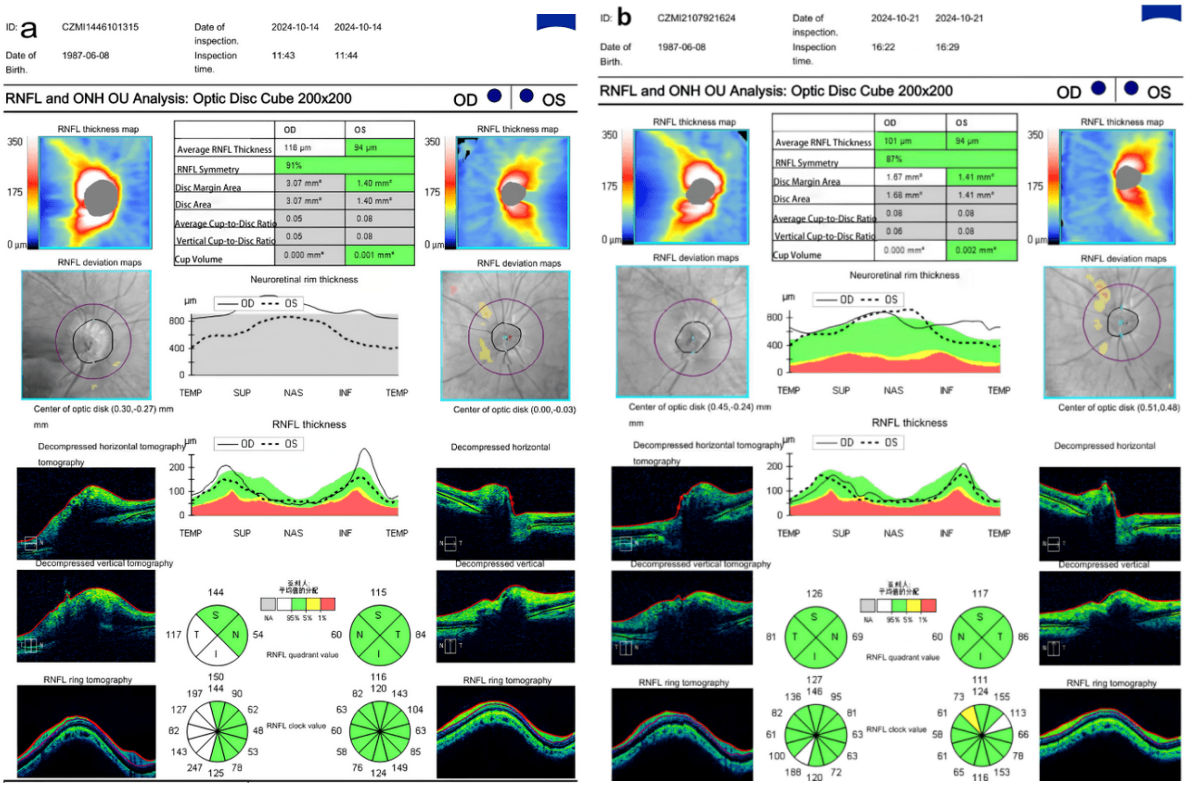

Figure 3 Pre-treatment OCT (a) and 1 week after treatment OCT (b) The patients had an increase in the thickness of the retinal nerve fibre layer, improved symmetry and an increase in Cup-to-Disc Ratio, suggesting that the treatment helped to slow down the progression of optic nerve damage

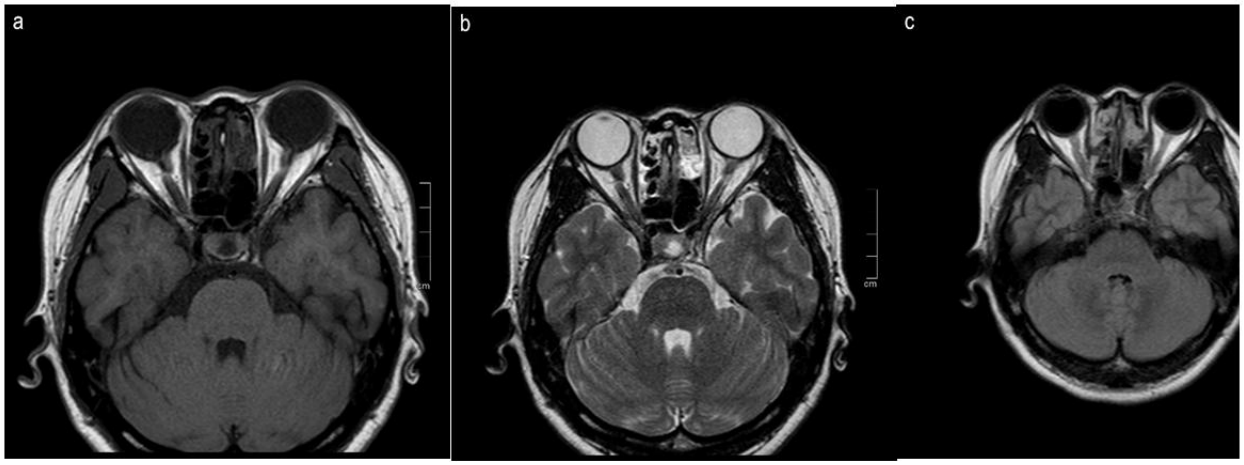

Figure 4 MRI of the optic nerve level. No significant optic nerve edema was observed on T1(a), T2(b), and FLAIR(c) sequences

Her past medical history was unremarkable. On admission, her body temperature was 37.2°C, her breath sounds were slightly coarse in both lungs, and her heart rhythm was regular at 76 beats per minute. Neurological examination revealed clear consciousness, with a pupil diameter of 2.5 mm in both eyes. Light reflexes were present bilaterally. Eye movements were normal, and examination of the remaining cranial nerves revealed no abnormalities. Limb muscle strength and tone were normal, tendon reflexes were symmetrical, and bilateral pathological signs were negative. Sensory and ataxic motor examinations were unremarkable, and meningeal irritation signs were absent. Cerebrospinal fluid (CSF) analysis from lumbar puncture showed a pressure of 205 mmH_2O, a protein level of 467 mg/L, and no leukocytes. Serological testing revealed a positive anti-MOG antibody at a titer of 1:10, while the anti-aquaporin-4 antibody (AQP-4) was negative. Cranial magnetic resonance imaging (MRI) revealed no abnormalities. The VEP test was not

performed in this case due to the patient's significant visual acuity impairment ,which could potentially undermine the validity and reproducibility of the results. Based on the clinical presentation and serological findings, the admission diagnosis was MOG-ON. On the first day of admission, the patient was initiated on intravenous methylprednisolone sodium succinate (80 mg/day) and methylcobalamin neurotrophic therapy. By the second day of treatment, her visual acuity in the right eye showed improvement, and the sensation of occlusion in the right eye was reduced.

After one week of hospitalization, an ophthalmological examination demonstrated that her uncorrected visual acuity was 0.3 in the right eye and 0.25 in the left eye. Corrected visual acuity was -5.00/-0.5*115=0.8 in the right eye and -5.00 DS=0.8 in the left eye. Funduscopic examination and OCT revealed a reduction in optic disc edema in the right eye compared to the initial findings (shown in Fig. 2, 3b, and 4b). The patient was discharged on oral prednisone acetate (40 mg/day). The dosage was tapered to 30 mg/day after two weeks and maintained for two months. At the three-month follow-up, her prednisone acetate dose was further tapered to 10 mg/day for maintenance therapy.

In this case, the patient exhibited a high level of adherence to medical advice, which facilitated the timely initiation of intravenous methylprednisolone sodium succinate therapy and the subsequent transition to oral prednisone acetate as scheduled. During the treatment period, the patient did not report any interruptions to therapy due to adverse drug reactions or personal reasons, thereby demonstrating optimal adherence to treatment. The patient also demonstrated good tolerance to glucocorticoid therapy, without unanticipated events or financial challenges. The long-term prognostic assessment demonstrated a substantial recovery of visual acuity following glucocorticoid therapy, with maintenance of normal levels throughout a three-month follow-up period. This finding suggests a positive response to the treatment regimen.

Discussion

Optic neuritis is an inflammatory disease of the optic nerve with a relatively low overall incidence of 3.7 cases per 100,000 person-years (95% confidence interval [CI]: 3.6 to 3.9) over a 22-year study period. The incidence varies by age group, with 0.77 cases per 100,000 person-years (95% CI: 0.64 to 0.90) in children (1-17 years) and 4.5 cases per 100,000 person-years (95% CI: 4.3 to 4.6) in adults. The etiology of optic neuritis is diverse and can be categorized into four main groups: 1)Demyelinating optic neuritis, including MS, NMOSD, and MOG-ON; 2) Autoimmune-related optic neuritides, such as immune disorders like systemic lupus erythematosus and leukoencephalopathy; 3) Infectious optic neuritis, caused by pathogens such as syphilis, HIV, hepatitis B and C, or intravitreal infections leading to optic nerve inflammation[1]; 4) Unknown etiology, accounting for approximately 30% of cases where the underlying cause remains undetermined[2]. When inflammatory or demyelinating lesions affect the optic nerve, the disease typically presents acutely and progresses rapidly. Common symptoms include reduced visual acuity, visual field defects, loss of color vision, and flashes of light. Studies indicate that optic neuritis often results in monocular vision loss, with most patients experiencing significant vision deterioration within hours to days, followed by gradual improvement over weeks to months (Smith et al, 2023[3]). Visual field defects, including central or peripheral scotomas, are also frequently observed (Johnson & Williams, 2022[4]). Additionally, loss of color vision and photopsia (flashes of light) are hallmark symptoms of the condition.

Optic neuritis significantly impacts patients' quality of life and social functioning. Among the subtypes of demyelinating optic neuritis, MOG-ON is a major entity. Studies suggest that MOG-ON accounts for approximately 1.2% to 6.5% of all demyelinating diseases in adults[5] and up to 40% of acute demyelinating disease manifestations in children[6]. It is responsible for 19.7% of all optic neuritis attacks[7]. The pathogenesis of MOG-ON differs from that of MS and NMOSD, often presenting with bilateral onset, a high incidence of optic disc edema, a recurrence rate of 50-80%, and a tendency toward chronicity.

The diagnosis of MOG-ON requires a comprehensive approach, combining clinical presentation, neuroimaging, serological testing, CSF analysis, and the rigorous exclusion of other

potential diagnoses. MOG-ON presents with a variety of clinical manifestations, including recurrent optic neuritis, marked optic disc edema, and ocular pain exacerbated by eye movement[8]. In some cases, the disease may progress to involve transverse myelitis or acute disseminated encephalomyelitis (ADEM).

Magnetic resonance imaging (MRI) plays a crucial role in the diagnosis of MOG-ON. Characteristic findings include optic nerve thickening and high T2/FLAIR signal intensity, particularly during the acute phase[9, 10]. The detection of MOG-IgG antibodies in both serum and CSF is essential for confirming the diagnosis. The positivity rate of MOG-IgG antibodies ranges from 70% to 80% in patients with MOG-ON, and their presence correlates with disease activity[11].While in this case, the MRI results were normal. This may be explained by the improvement of edema following corticosteroid therapy. Additionally, only a routine non-contrast scan was performed without targeted contrast enhancement, which may have reduced the detection of optic nerve enhancement signals.

Lumbar puncture is often performed to assess intracranial pressure and analyze CSF. While intracranial pressure is typically normal, it may be mildly elevated in some cases, as observed in this patient. CSF analysis may reveal elevated cell counts and protein levels, although these findings are generally nonspecific. Before confirming a diagnosis of MOG-ON, it is critical to rule out other conditions with similar clinical and imaging features, particularly multiple sclerosis (MS) and NMOSD[12].

The primary treatment modalities for MOG-ON include corticosteroid therapy and the use of immunosuppressive agents. Among these, corticosteroid therapy is considered the first-line treatment for acute exacerbations of MOG-ON. It is typically administered in two distinct regimens: High-dose corticosteroid non-pulse therapy (1-3 mg/kg/day), and High-dose corticosteroid pulse therapy (0.5-1 g/day). The majority of patients demonstrate a favorable prognosis with timely and appropriate treatment[2]. The absence of a uniform optimal treatment regimen for MOG-ON is attributable to the paucity of data from treatment studies, which are characterized by small sample sizes and retrospective design. However, studies have demonstrated that high-dose corticosteroid pulse therapy[13] can rapidly improve patients' visual fields and achieve significant recovery of vision and visual fields within the first month.

In a study conducted by Tan Xiao's research team, the effects of corticosteroid pulse therapy and high-dose corticosteroid non-pulse therapy on visual function recovery and retinal nerve fiber layer thickness in patients with first-episode MOG-ON were compared. The findings revealed that patients receiving corticosteroid pulse therapy showed significant improvement in mean deviation (MD) values of the visual field within one month of treatment[14]. In contrast, patients in the high-dose corticosteroid non-pulse therapy group exhibited significant recovery only after six months of treatment. This suggests that corticosteroid pulse therapy facilitates more rapid visual field improvement in patients.

Furthermore, MOG-ON patients are often corticosteroid-dependent, particularly those who exhibit heightened sensitivity to corticosteroid therapy in the early stages. Therefore, it is crucial that corticosteroid tapering is conducted gradually. According to the Chinese expert consensus, adult patients should receive an initial intravenous loading dose of methylprednisolone (1 g/day for 3-5 days), followed by a gradual reduction to oral prednisone (60 mg/day)[9]. Once the dose is reduced to a medium level (30-40 mg/day), patients should transition to a lower maintenance dose of oral prednisone (10-15 mg/day for 1-3 days, followed by 10-15 mg every 1-2 days). This regimen should be maintained for 6 months to 1 year, with subsequent adjustments based on the patient's response.

The tapering process should be carefully monitored to ensure a gradual reduction, typically by decreasing the dose by 5 mg every 2 weeks until reaching a maintenance dose of 10-15 mg/day. This maintenance dose is usually continued for 6 months to 1 year. In pediatric cases, the initial intravenous dose of methylprednisolone is 20-30 mg/(kg·day), with a stepwise reduction following the adult regimen.

In this case, the patient received intravenous methylprednisolone (80 mg) immediately following the initial diagnosis of optic neuritis, with significant symptom improvement observed the next day. A subsequent positive serum MOG antibody test confirmed the diagnosis of MOG-

ON. Given the patient's marked clinical improvement, corticosteroid pulse therapy was not administered. This case underscores the challenges in determining the optimal initial corticosteroid treatment regimen, emphasizing the need for individualized decision-making.

Most patients achieve better visual recovery with corticosteroid therapy; however, recurrences are common in MOG-ON, particularly if the corticosteroid dose is rapidly tapered in the early stages. The frequency and severity of relapses can significantly impact the long-term prognosis of patients, highlighting the need for further research into optimal acute treatment regimens and long-term immunosuppressive therapies to prevent relapses in MOG-ON patients[7].

For patients with recurrent MOG-ON, especially those with incomplete visual function recovery, preventive treatment during the chronic phase is of paramount importance. Commonly used immunosuppressive agents include azathioprine (AZA), mycophenolate mofetil (MMF), and rituximab (RTX). These agents act through diverse mechanisms to inhibit immune system overactivation, reduce inflammation, and prevent nerve damage, thereby decreasing the rate of disease recurrence. For instance, as a purine analog and prodrug, AZA is metabolized intracellularly into 6-mercaptopurine (6-MP), which incorporates into DNA during synthesis, disrupting replication and repair processes. This interference particularly impacts rapidly dividing lymphocytes, ultimately reducing their proliferation and dampening adaptive immune activity[15]. In contrast, MMF is hydrolyzed to its active form, mycophenolic acid (MPA), which selectively inhibits inosine-5'-monophosphate dehydrogenase (IMPDH), a rate-limiting enzyme in the de novo synthesis of guanine nucleotides. Since T and B lymphocytes predominantly rely on this pathway—unlike most cells that utilize salvage pathways—IMPDH blockade depletes guanosine triphosphate (GTP) and deoxyguanosine triphosphate (dGTP), halting DNA and RNA synthesis specifically in lymphocytes[16].

Additionally, intravenous immunoglobulin (IVIG) has shown significant prophylactic efficacy in MOG-ON. The mechanism of IVIG involves modulating the immune system and reducing inflammatory responses through the administration of high doses of immunoglobulins. In some cases, IVIG has been associated with lower recurrence rates, suggesting its potential role in preventing MOG-ON relapses. A retrospective, multicenter, cohort study of adult patients with MOG antibody-associated disease (MOGAD) demonstrated that maintenance IVIG therapy was associated with a reduction in disease recurrence. The study reported that the median annual recurrence rate before IVIG treatment was 1.4 (range: 0-6.1), compared to 0 (range: 0-3) during IVIG treatment[17].

In conclusion, based on solid scientific evidence, including immunology, imaging, clinical response to therapy, and literature review, this report provides a comprehensive clinical description of a rare MOG-ON case, including diagnostic procedures, treatment outcomes, and favorable short-term results, offering valuable insights to enhance clinicians' ability to diagnose and manage this condition. However, the study is limited by its single-case design, absence of a control group, and relatively short follow-up period, which restrict the ability to assess long-term prognosis and the durability of treatment effects. It highlights the importance of accurate diagnosis and timely intervention in MOG-ON. The patient's acute visual loss and ocular pain, coupled with optic disc edema and a positive MOG antibody test, underscored the need for a comprehensive diagnostic approach to differentiate MOG-ON from other demyelinating disorders[18]. Early initiation of high-dose corticosteroid therapy led to significant visual improvement[19], emphasizing the critical role of prompt and appropriate treatment in managing MOG-ON. This report serves as a reminder for clinicians to consider MOG-ON in cases of optic neuritis, particularly when atypical features are present, and to prioritize early and sustained treatment to optimize patient outcomes.

Statements

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.The report does not include personal information that could identify the patient directly or indirectly.

Statement of Ethics

The patient gave oral and written consent to publish the data. All medical interventions have

been carried out according to the latest protocols of therapy. The study protocol was reviewed and approved by Nanjing University of Chinese Medicine and Department of Neurology, and the Department of Ophthalmology, the Affiliated Suzhou Hospital of Nanjing Medical University. The processes involved in this research adhered to the tenets of the Declaration of Helsinki.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

(Yan-Xi Shen, independent author) – Responsible for data compilation and analysis, drafting the manuscript, polishing figures, and final approval of the revised version. Moreover, the author would like to express the gratitude to the Department of Neurology and the Department of Ophthalmology, Affiliated Suzhou Hospital of Nanjing Medical University, for their invaluable support and resources that made this research possible.

Data Availability Statement

The data supporting the findings are not publicly available due to privacy restrictions. The raw data were collected and managed by the Department of Neurology and the Department of Ophthalmology, Affiliated Suzhou Hospital of Nanjing Medical University. Requests for access to the data may be directed to the corresponding author Yan-Xi Shen at 131023103@njucm.edu.cn.

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