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Case Report

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Case Report-MOG Associated Disease

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ABSTRACT

Purpose: To report a case of neuromyelitis optica spectrum disorder – MOG Associated Disease (MOGAD) presented to tertiary care center at western Vidarbha region in Maharashtra

Case Report: We described a case of neuromyelitis optica a 28 years old female presented to our hospital with complaints of diminution of vision in right eye since 15 days. History of cough, cold, fever, redness & watering of right eye 1 month ago. Diagnosed as case of MOGAD at a tertiary care centre with relevant investigations. She underwent conservative medical management and relieved.

Results: We diagnosed case as MOGAD based on antibody assay (**anti MOG-Myelin Oligodendrocyte Glycoprotein-Positive**).

Conclusion: Neuromyelitis optica is a chronic autoimmune disease. Anti MOG antibodies are produced in 7.4 % of patients. It is now classified as MOGAD.

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Introduction

Neuromyelitis Optica Spectrum Disorders (NMOSD) and anti-Myelin Oligodendrocyte Glycoprotein (anti-MOG) syndromes are immune-mediated inflammatory conditions of the Central Nervous System (CNS) that frequently involve the optic nerves and the spinal cord. Because of their clinical manifestations and habitual relapsing course, they are frequently confounded with Multiple Sclerosis (MS). Early and accurate diagnosis of these distinct conditions is very relevant as they have different therapeutic approaches [1].

Myelin Oligodendrocyte Glycoprotein (MOG) is a myelin protein exclusively expressed at the outermost surface of the myelin sheath and oligodendrocyte membranes in the CNS [2]. MOG antibody had long been considered a potentially pathogenic autoantibody involved in human inflammatory demyelinating diseases, especially multiple sclerosis, but previous results on the detection of MOG antibody by ELISA or western blot were confusing because of the low specificity. However, by using cell-based assays, it has become clear that the MOG antibody is detectable in a proportion of patients with such inflammatory CNS demyelinating diseases as optic neuritis, acute myelitis, Neuromyelitis Optica Spectrum Disorder (NMOSD) without aquaporin-4 (AQP4) antibody [3].

Meanwhile, patients with typical multiple sclerosis are essentially negative for the MOG antibody [4]. MOG antibody associated disease is an independent autoimmune demyelinating disease targeting MOG [5].

The majority of MOG Antibody (MOG-Ab) seropositive patients have Optic Neuritis (ON), encephalitis with brain demyelinating lesions, and/or transverse myelitis [6]. MOGAD may have a monophasic or relapsing course. Attacks usually develop over days and may plateau with variable recovery over weeks to months. Attacks may be preceded by an infectious illness or vaccination [7]. The studies and data regarding MOG syndrome are insufficient which necessitates research and reporting of this case so that it will add to knowledge in clinical presentation, classification and to set treatment protocols of such cases.

Case Report: MOG Associated Disease (MOGAD)

A 28 years old female patient presented with complaints of pain in the both eyes since 5 days and diminution of vision in both eyes more in right eye since 15 days which worsened since 5 days. She had history of viral illness 1 month ago, history of headache, vomiting, giddiness, altered sensorium, head injury, fall, autoimmune disorders, HIV infection was ruled out. Ocular examination showed visual acuity in right eye Hand Movements Close to Face (HMCF) and in left eye 6/60; colour vision was abnormal in both eyes. Near vision N/60. Optic Disc was hyperaemic with blurred margins in both eyes and cup to disc ratio was inappreciable. Foveal Reflexes were dull in both eyes. Neurological examination was normal. Cognitive functions were normal. Laboratory investigations such as test for anti-DNA antibodies, retroviral antibodies (by ELISA method), Thyroid functions, Syphilis, thymoglobulin, and rheumatoid factor were done in addition to standard hematologic investigations. All investigations were normal. Testing for myasthenia gravis-related antibodies was performed (AchRAb) and was negative. Cerebro-Spinal Fluid (CSF) analysis showed absence of oligoclonal bands. NMO-IgG serum antibody test was negative and Anti MOG-positive by HEK293cell-based assay. MRI brain & orbit showed right optic nerve thickening, increased signals on FLAIR & T2W images, and enhancement with contrast study S/O right optic neuritis with altered signals in left posterior ganglio-capsular region.

Based on clinical examination, radiological investigations, CSF analysis, MOG antibody test, clinical diagnosis of MOG associated disease is done. Intravenous methylprednisolone was given (1 gm/day) for 5 days followed by oral steroid 50 mg/day, Tapered over 2 weeks and 8 days intravenous immunoglobulin daily 5 vials.Her vision began to improve by ninth day, and at the end of seventh week it was 6/6 in both eyes. Now, for more than one year, she is leading a normal life and is being followed up regularly for relapses.

Discussion

In recent years, there have been several studies that have provided new insights into anti-Myelin Oligodendrocyte Antibody (anti-MOG) disease. Ramanathan S, Prelog K, Barnes EH, et al. studied Radiological differentiation of optic neuritis with myelin oligodendrocyte glycoprotein antibodies, aquaporin-4 antibodies, and multiple sclerosis and found that optic neuritis in patients with anti-MOG antibodies had a distinct radiological phenotype compared to those with aquaporin-4 antibodies or multiple sclerosis. Patients with anti-MOG antibodies were more likely to have a longitudinally extensive optic neuritis lesion on MRI, and less likely to have brain lesions or spinal cord lesions [8]. Jarius S, Ruprecht K, Kleiter I, et al. Proposed MOG-IgG in NMO and related disorders: a multicenter study of 50 patients. Part 2: Epidemiology, clinical presentation, radiological and laboratory features, treatment responses, and long-term outcome and reported that among 50 patients with Neuromyelitis Optica Spectrum Disorders (NMOSD), 16 (32%) were positive for anti-MOG antibodies. The authors noted that anti-MOG disease may account for a significant proportion of cases of NMOSD [9,10]. identified anti-MOG antibodies in a subgroup of anti-AQP4 antibody-negative NMO patients (about 25%), but not in anti-AQP4 antibody-positive patients similar to our patient, some of the MOG-seropositive patients presented with a pediatric disease onset. Data of this study suggests that MOG-seropositive patients show a diverse clinical phenotype with clinical features resembling NMO (attacks confined to the spinal cord and the optic nerves) [10].

Conclusion

MOG-associated disease presenting as bilateral optic neuritis following a viral infection is rare, causing vision loss with improvement after IV immunoglobulin and steroids. It is a newly recognized autoimmune demyelinating condition with diverse clinical symptoms. Diagnosis requires clinical, radiological, and laboratory correlation. Immunomodulatory therapies are used, but the optimal treatment remains under evaluation, highlighting the importance of this case report.

Declarations

Ethics Approval and Consent to Participate: Approved by the institutional ethics committee.

Consent for Publication: Patient consent obtained; all authors consent to publication.

Availability of Data and Material: Data sourced from institutional case records.

Competing Interests: The authors declare no competing interests.

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Authors' Contributions: Author 1 examined the case and structured the manuscript; Author 2 proposed the case report and analyzed data; Authors 3 and 4 edited and corrected the manuscript. All authors contributed to writing.

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