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Case Study

CASE STUDY: Personal Experience as a Physician and Anti-MOG Positive Neuromyelitis Optica Patient (MOGAD)

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Abstract

Neuromyelitis Optica (NMO) is a rare demyelinating disease, recently segregated from other demyelinating diseases due to its distinctive features that set it apart from multiple sclerosis (MS) and other central nervous system (CNS) disorders. Particularly, the presence of autoantibodies against myelin oligodendrocyte glycoprotein (MOG) has emerged as a specific subtype of NMO, known as MOGAD. This article presents a unique case in which a physician, with a specialization in neuroimmunology, personally experiences the challenges of delayed diagnosis and clinical management of this disease.

***Keywords:** Neuromyelitis Optica, MOGAD, Delayed diagnosis, Symptomatic treatment, Medical awareness, Clinical protocol modification, Empathy, Pain, Anti-MOG.*

Objectives

1. To promote awareness about the importance of early diagnosis of MOGAD: Investigate and discuss the urgent need for early diagnosis of MOGAD to improve clinical outcomes and patient quality of life, highlighting the benefits of timely treatment in disease management.
2. To differentiate MOGAD from Neuromyelitis Optica (NMO) in terms of symptomatology and prognosis: Analyse the differences between MOGAD and NMO, emphasizing the importance ofrecognizing MOGAD as a unique medical entity and not simply a variant of NMO, which will allowfor a more specific and effective treatment approach.
3. To advocate for the inclusion of Anti-MOG antibody analysis in neurology protocols: Evaluatethe clinical utility and accessibility of Anti-MOG antibody screening as a fundamental diagnostic tool, emphasizing its simplicity, low cost, and minimal invasiveness compared to other more invasive diagnostic tests.
4. To highlight the importance of not dismissing MOGAD due to its low diagnostic rate: Examine current barriers to MOGAD diagnosis, such as lack of disease awareness among healthcare professionals and the absence of MOGAD inclusion in standard diagnostic protocols, and advocate for a more proactive approach to detection and treatment of this underdiagnosed disease.
5. To foster empathy and compassion from the medical community towards MOGAD patients: Explore the psychological and emotional impact of chronic pain on the daily lives of patients, emphasizing the need for patient-centered medical care that recognizes and appropriately addresses the physical and emotional suffering associated with MOGAD.
6. To raise awareness about the impact of chronic pain on patient's lives: Investigate and analysethe devastating impact of high-intensity chronic pain on daily functionality, quality of life, and emotional well-being of MOGAD patients, and advocate for a comprehensive treatment approach that prioritizes effective pain management as a fundamental aspect of patient care.

7. To promote a change in diagnostic evaluation of patients with neurological symptoms consistent with Multiple Sclerosis (MS), and other more common neurological pathologies, encouraging systematic consideration of Anti-MOG antibody disease (MOGAD) as a relevant differential diagnostic entity. This objective aims to redefine conventional diagnostic criteria, raising awareness about MOGAD and addressing the possibility that a significant percentage of patients diagnosed with MS may actually have MOGAD. Thus, proposing a radical shift in the perception of the incidence of this disease, so that it is recognized as a potentially underdiagnosed condition due to lack of systematic consideration in clinical evaluation. This objective aspires to a more thorough and sensitive approach in medical practice, allowing for early and accurate identification of MOGAD in patients with neurological symptoms, which could have a transformative impact on disease management and prognosis.

8. To understand comorbidities between MOGAD and other autoimmune diseases, as well as neurological diseases, such as Horton's syndrome, or systemic diseases, such as antiphospholipid syndrome, lipedema, etc., in order to improve early identification, clinical management, and treatment outcomes in affected patients. By better understanding the associated comorbidities with MOGAD, it is hoped to improve diagnostic accuracy, optimize therapeutic management, and improve clinical outcomes and quality of life for affected patients.

9. To raise awareness within the medical community about the importance of addressing complex cases with multiple symptoms in a comprehensive and holistic manner, avoiding feelings of laziness or disinterest due to the case's difficulty, and promoting empathetic and patient-centered medical care. This article seeks to promote a medical culture that values and respects the patient's experience, avoiding apathy or disinterest in the face of case complexity, and providing compassionate and patient-centered care following medical ethics guidelines and the concept of the patient as a whole.

Introduction

Neuromyelitis Optica (NMO) is an autoimmune disease of the central nervous system characterised by recurrent attacks primarily affecting the spinal cord and optic nerves, leading to significant disabilities in patients from the first onset.

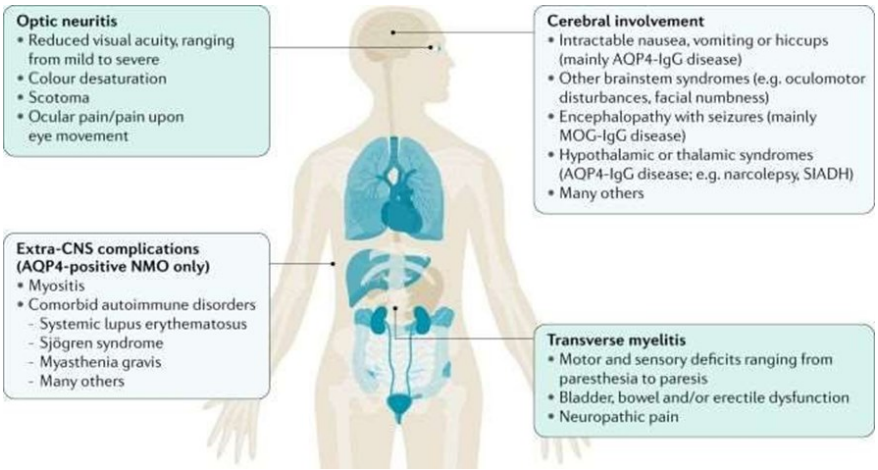


Image 1. *Typical Involvement of MOGAD at the Systemic Level.*

Anti-Myelin Oligodendrocyte Glycoprotein (MOG) Positive Neuromyelitis Optica Spectrum Disorder (NMOSD) represents a distinct subgroup of patients within the NMOSD spectrum, characterized by the presence of autoantibodies directed against oligodendrocyte myelin (MOG). Although it shares clinical similarities with traditional NMOSD, MOGAD exhibits unique features in terms of pathology, treatment response, and prognosis.

Below, I present a comparative table between Neuromyelitis Optica (NMO) and Anti-MOG Autoimmune Encephalomyelitis (MOGAD) based on some of their clinical and diagnostic characteristics:

Table 1. Differences between NMO and MOGAD according to recent studies. It is important to note that this comparative table is based on the provided information and generalities observedin the medical literature up to my last update in January 2022.

Characteristic	Neuromyelitis Optica (NMO)	Myelin Oligodendrocyte Glycoprotein Antibody Disease (MOGAD)
Pain	Present only during relapse, with intermittent episodes	Chronic, persistent intense pain
Spasticity	Lower	Higher
Incidence in adults	Higher	Lower
Treatment response	Good	Variable, even refractory
Prognosis	Good with treatment, moderate risk of permanent disability	Severe, risk of death and permanent disability
Relapses	Fewer	Frequent in the early years post-diagnosis
Disability after first episode	Not necessarily, variable	Can be very severe
Other significant differences	Possibility of optic nerve and spinal cord involvement	Clinical presentation variability, possible brainstem and cerebellar involvement

It is important to highlight that, while there are some distinctive differences between NMO andMOGAD, there may also be overlaps and variations in clinical presentation and therapeutic management in individual cases. Therefore, a comprehensive evaluation by a specialised physician is essential for accurate diagnosis and appropriate treatment planning.

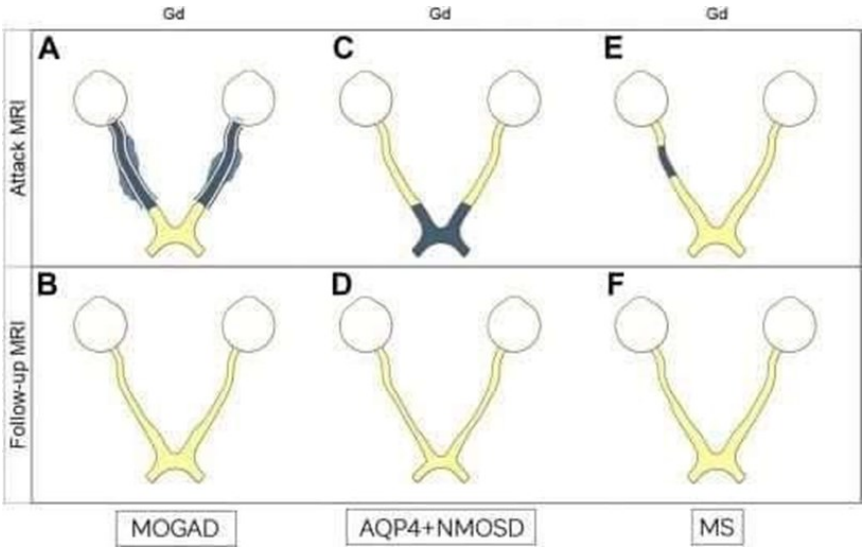


Image 2. Schematic Representation of Differences between MOGAD, NMO, and MS in the Presentation of Optic Neuritis in an Attack and its Visibility on Imaging during and after the Attack.

Patients' refractory to treatment in the context of MOGAD present a significant clinical challenge due to the lack of response to standard therapies and the persistence of disabling symptoms. The identification and management of these patients requires a thorough evaluation and individualised therapeutic approach. Some important considerations for addressing treatment-refractory patients in MOGAD include:'

The presence of comorbidities, such as other autoimmune diseases or concurrent neurological disorders, may influence treatment response and should be investigated and appropriately treated.

Consideration should be given to adjusting or changing immunosuppressive treatment regimens to address persistent inflammatory activity and reduce disease progression. This may include the use of second-line immunosuppressive agents or targeted biological therapies.

In addition to treating the underlying disease, it is important to address neurological symptoms and sequelae comprehensively through rehabilitation therapies, pain management, and psychological support.

Participation in clinical trials and research studies may offer innovative therapeutic options and access to experimental treatments for patients refractory to conventional therapies.

In summary, the management of treatment-refractory patients in MOGAD requires a multidisciplinary and personalised approach that addresses both the underlying disease and the individual needs of the patient. Early identification and aggressive management of these cases are crucial to improving clinical outcomes and quality of life for affected patients.

Despite the growing recognition of MOGAD, its diagnosis remains challenging due to its low prevalence and lack of awareness among medical professionals. Early identification of this disease is crucial to ensure appropriate treatment and improve clinical outcomes for affected patients.

In this article, we present the unique case of a female patient, a physician with a background in neuroimmunology, who has been diagnosed with MOGAD. It is my own personal experience that is described in the article below. Through my personal experience, I seek to explore the challenges associated with delayed diagnosis, clinical management, and treatment implications of this poorly understood disease. Furthermore, the importance of increasing medical awareness of MOGAD, as well as the urgent needs for research and clinical care to address the needs of affected patients, will be discussed.

Case Presentation

I present my own case, a female patient, professionally a doctor. I began experiencing neurological symptoms in 2018 at the age of 33. The initial symptoms I reported were paresthesias, dysesthesias, myoclonus, sharp pains, burning sensations, and hypersensitivity in various regions of the upper and lower limbs, accompanied by severe headaches, experiencing seizures on two occasions. During an episode, I also developed dysphagia and mild dysarthria, along with analytical signs of blood hypercoagulability and central nocturnal apneas.

Personal and Social Context

My personal and social history is that of a 33-year-old woman (currently 38) reflecting a successful trajectory filled with passion for her work and aspirations. As a doctor and head of a medical service in a prestigious company, I had achieved significant professional and financial recognition. Additionally, my venture in the Hospitality and Events sector with my partner showcased my versatility and ability to carry out multiple projects successfully.

My dream then was to specialise beyond the knowledge I had in my medical practice, by undertaking courses and with the desire to study a specialty in neurophysiology, which reveals my constant pursuit of professional growth and knowledge in my field. This ambitious goal demonstrates my dedication and commitment to excellence in my career, as it has always beenmy vocation to help others and my aim to provide the best possible care to all patients.

On a personal level, my passion for sports, travel, and family time stood out as I tried to find balance between work and enjoying life. My energy, good humour, and zest for life depict a multifaceted and passionate woman in all aspects.

However, the sudden onset of an illness that forces me to leave my job and also close my company with my husband represents an unexpected and challenging turn in life's path. The need to distance myself from my professional dreams and close a business that I had built with effort and dedication implies a painful process of adaptation and a significant readjustment in my entire life. The difficult decision to close the company and disengage from my medical practice not only affects my financial stability but also my own identity and sense of personal fulfilment. This situation poses enormous emotional and logistical challenges, as I am suddenly confronted with the need to rebuild my professional and personal life in a new direction.

In summary, this story is the story of many other patients who suffer from disabling illnesses, but I can only speak through my own voice, and that is why I try to make them see that my story exemplifies the complexity and resilience required to face unexpected changes and adapt to new circumstances. My determination, passion, and ability to face challenges have been and continue to be crucial in this transition process towards a new stage in my life, where emotional support and professional reinvention have played a fundamental role in my emotional stability and personal fulfilment.

Disease Course

The first symptoms manifested in 2018, with a presentation of dizziness and nocturnal apneas diagnosed through a sleep study as sustained nocturnal hypoventilation. The BIPAP therapy along with nocturnal oxygen revealed the presence of central apneas, in addition to cervical pain and syncope. There was also an episode of dysarthria and seizures on two occasions with loss of consciousness, tonic-clonic movements, and subsequent fatigue. I visited the nearest hospital emergency department several times without much success.

Now, imagine being in the shoes of a patient with worrying neurological symptoms, seeking answers and support from your trusted doctor, who is also a professional colleague. As a patientovernight, and without prior notice, you experience unexplained muscle tremors, loss of balance, and blurry vision, symptoms that have profoundly impacted your daily life. However, instead of being met with understanding and attention, you encounter skeptical looks and conjectures questioning the validity of your symptoms. You face the distressing reality of being misunderstood by your own doctor, who, instead of seeking a medical explanation for your symptoms, being so florid, hints at the possibility of mental pathology or even substance use. You feel misunderstood, devalued, and invisible, while struggling to convey to the doctor the severity of your situation. The emotional impact of this experience is overwhelming. You are overcome with a sense of helplessness and despair as you find yourself trapped in a maze of medical doubts and prejudices. You wonder if you will ever find relief for your symptoms or if you will be condemned to live in a state of uncertainty and suffering indefinitely.

This situation somehow visually illustrates the profound impact that the lack of understanding and empathy from doctors can have on the lives of patients with neurological symptoms. Beyond physical pain, the emotional suffering caused by the sense of misunderstanding and neglect can be devastating. It is crucial for doctors to recognise and address the importance of listening to and validating their patients' experiences, rather than prematurely dismissing themas manifestations of psychological or behavioural problems.

It was not until the second seizure crisis that the medical team decided to admit me for further examination, at that time, as I mentioned earlier, I was 33 years old. Initial diagnostic tests revealed significant findings on a magnetic resonance imaging (MRI), showing myelopathy fromC5 to C7, approximately 10 x 1 mm in size, along with findings on T2-weighted sequences showing punctate hyperintensities in bilateral frontoparietal subcortical white matter, without expression in T1, diffusion restriction, or contrast enhancement. Additionally, I experienced decreased vision in the right eye, with relevant findings on contrast-enhanced computed tomography demonstrating dilation of both ophthalmic veins and altered campimetry. Furthermore, D-dimer, CRP, and leukocytes were markedly elevated. During hospitalisation, I experienced fainting and sustained hypotension, as well as paresthesias and pains in various regions of the body. A lumbar puncture was performed but yielded no significant results.

Initially, a diagnosis of cluster headache was considered, and treatment with manidon, topiramate, and home oxygen was implemented. The lesions observed in the MRI, as well as the dilation of the ophthalmic veins, were overlooked, and treatment was reduced to mere symptomatic management without providing any diagnosis. Despite therapeutic efforts, there was no improvement, especially in cervical pains and severe headaches, and I underwent several sphenopalatine ganglion blocks, without success. Finally, due to severe pains, the anaesthesia team decided on intravenous lidocaine infusion of 220MG IV as a pain treatment, which slightlyimproved the symptoms.

Additionally, I was prescribed Palexia from the pain unit, a potent relaxant that, although did not fulfil its primary function of relieving nocturnal pain predominantly, caused alterations in memory and daytime sensory perception of my own life. Therefore, in consensus with the pain unit team, I decided to discontinue the treatment with this drug, as seeing the effects it had on me and its ineffectiveness for the intended purpose, I did not want to lose my own identity and decided to relinquish the only palliative treatment that could be offered to me at the time. I was informed about the possibility of neurostimulator implantation, but the limited scientific evidence of improvement for my case, as well as the conflicting opinions among several experts on the topic, led me to decide against undergoing the implant due to the risk-benefit ratio.

In 2020, I experienced what I believe to be a pseudo-relapse or undiagnosed relapse with predominantly cervical symptoms, paresthesias, great difficulty in extending the legs, and sensations of electric shocks in the spine that were not investigated due to the global pandemicsituation, which was overwhelming the healthcare system. I had to endure all the pain and neurological symptoms without any treatment other than paracetamol as an analgesic, which had long ceased to be effective.

In 2021, I returned to the neurology service due to intense paresthesias in both hands, arms, and the cervical area. Due to my previous profession and continuous computer use, they considered the most common diagnosis, nerve compression due to carpal tunnel syndrome, andI underwent surgery on both hands after performing some somatosensory potentials of the hand that resulted positive for median nerve injury.

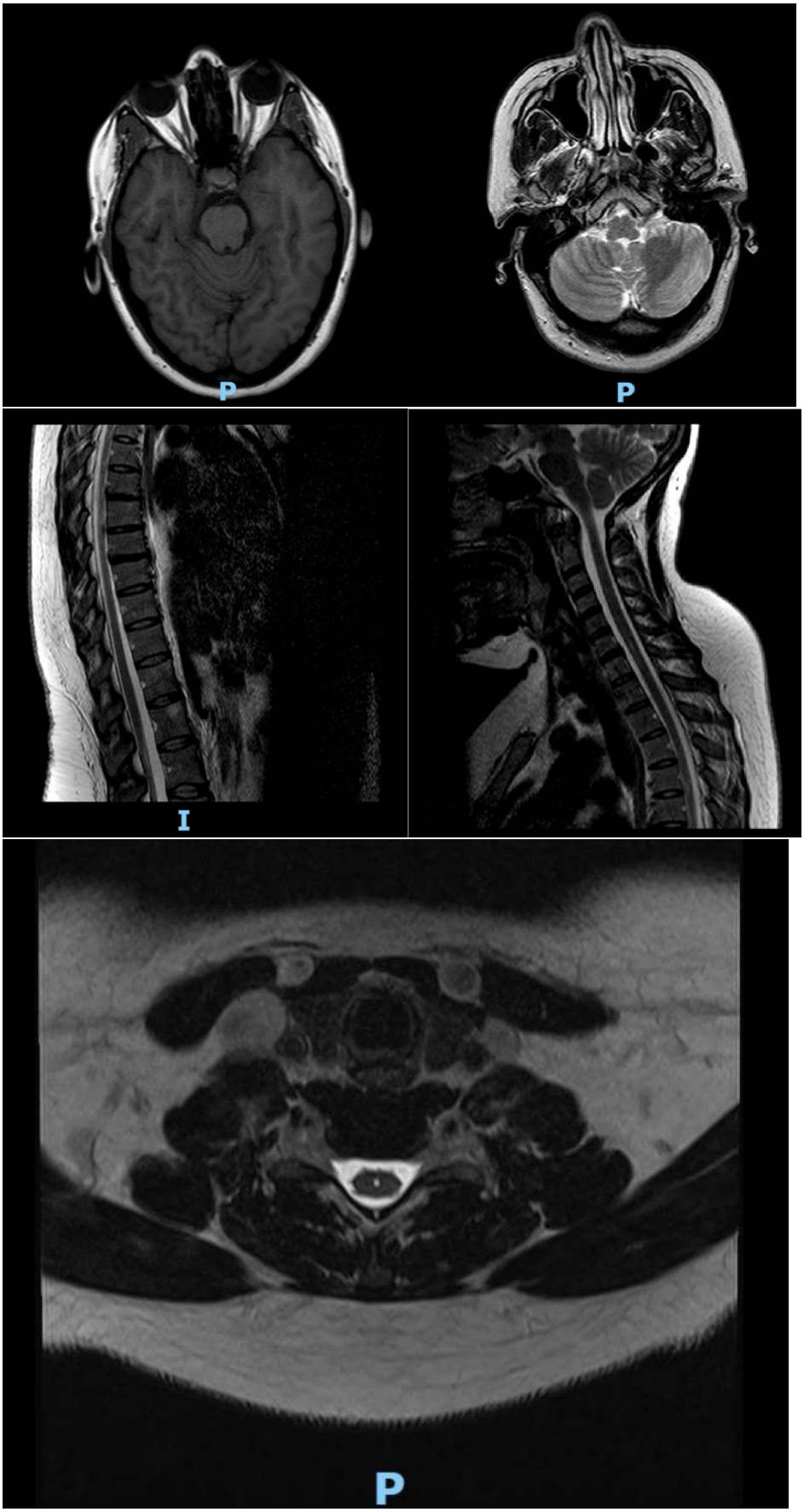
Due to the previous symptoms and the diagnosis of multiple sclerosis in a close relative, the medical team studying my case began to consider a possible atypical MS, but it was not thoroughly investigated due to the ongoing global situation with COVID-19, which was still overwhelming healthcare facilities and delaying tests and treatments beyond the much-neededcare for pandemic-affected patients.

In 2022, I experienced what I believe to be a third/fourth relapse, (although the diagnosis was not made until this moment, so for the medical team, it was considered the first one), characterised by intense muscle pain, spasticity, clonus, hyperreflexia, vertigo, and loss of strength, mainly in the lower limbs, urinary incontinence, projectile vomiting, and nausea. Magnetic resonance imaging confirmed the persistence of cervical myelopathy, and incidentallyidentified a tubular image with contrast enhancement, classified as a venous hemangioma in the right cerebellum, which was not considered relevant at that time.

Due to the symptoms I was experiencing at that time, visual evoked potentials were performed,which showed elongation of the signal on the right side and borderline on the left side.Neurophysiological tests (somatosensory evoked potentials) were performed, which concludedthat there is demyelinating involvement of the somatosensory pathways in the upper limbs, as well as electromyography, which showed neurogenic traces corresponding to territories between C5-C8 on the left with moderate denervation and neurogenic traces between L2-S1 with mild-moderate bilateral denervation, more accentuated on the left side. A lumbar puncture was performed again, with an objective result of 4 erythrocytes, 3 leukocytes, 62 glucose, 21.7 proteins, and no oligoclonal bands were detected.

Analytical test results revealed the presence of positive anti-MOG antibodies at a titre of 1:10, while anti-Aquaporin 4 antibodies were negative. No diagnostic criteria for multiple sclerosis were observed, so it was ruled out. Based on these analytical findings, the diagnosis of Demyelinating Disease in the Spectrum of Anti-MOG Positive Neuromyelitis Optica (MOGAD) was established, i.e., it took more than 4 years to reach a conclusion of demyelinating disease solely due to its low prevalence, with symptoms and objective tests compatible with the disease. From this very point, it is worth highlighting the importance of comprehensive evaluation and consideration of differential diagnoses that include less common diseases in patients with atypical presentations of demyelinating neurological diseases.

In summary, I experienced progressive symptoms for over four years before receiving a definitive diagnosis of MOGAD. The initial symptoms were erroneously attributed to various causes by several medical specialists, leading to fragmented diagnoses by specialties andsubspecialties, but never a diagnosis that encompassed all the presented symptoms. Despite thepresence of objective tests, such as magnetic resonance imaging and cerebrospinal fluid analysis, the appropriate diagnosis was significantly delayed. This led to the development of sequelae from the relapse(s), as one may perceive it. These sequelae currently result in a recognized disability degree of 77%, requiring a third person for performing basic activities of daily living, inability to work, and special needs due to reduced mobility. In addition to this impactful burden, difficult to bear for someone of my age (currently 38 years old), residual sequelae symptoms add up, including spasticity, pain, chronic fatigue, loss of strength, memoryloss, and a sense of frustration seeing symptoms becoming chronic.



Own MRI Images

Now, pause for a moment from receiving clinical information and engage in an exercise of empathy. Imagine yourselves at this moment waking up each morning to the same routine: thesound of the alarm clock, the aroma of freshly brewed coffee, and the gentle light of dawn filtering through the window. But this time, as you try to get out of bed, you realize something has changed. Your legs feel heavy, as if anchored to the mattress, and it's difficult to fully extendthem due to pain. With great effort, you manage to sit on the edge of the bed, but the simple act of standing up exhausts you more than usual.

With determination, you head to the bathroom, but as you look at yourself in the mirror, you realize something has changed in you. Your hands lack the same strength when trying to hold the toothbrush, and the simple task of brushing your hair becomes a titanic challenge. The clothes you once put on with ease now seem like an insurmountable barrier, and the idea of facing the outside world suddenly becomes daunting because even the most minimal task feelsdifficult.

At that moment, reality hits you hard: you are no longer the independent and self-sufficient person you used to be. The mere idea of not being able to dress or shower without assistance becomes a dark and terrifying abyss threatening to engulf you entirely. You face the harsh truth that the disease has stolen your autonomy and dignity, leaving you vulnerable and dependent on others.

This is what I felt the first time. It's a vivid example that I believe can be significant for you to understand the impact of your life taking a 180-degree turn overnight. And furthermore, imagine that not only does this repeat every day, but also you see your capabilities dwindling tothe point of being unable to walk without assistance.

Current Status

Currently, I am in a phase with clinical symptoms suggestive of a relapse or pseudo-relapse withdaily intense pains in legs, especially the right, and constant tingling in the cervico-thoracic region radiating towards the left arm, with loss of strength and pain, dysesthesias, paresthesias,and increased spasticity. In the MRI, punctiform hyperintensities are again visualised in the bilateral frontoparietal subcortical white matter on T2-weighted sequences, and at the moment, the positivity of Anti-MOG antibodies is not evident, leading my medical team to think that treatment is not necessary as I am not apparently in an active relapse. It seems important to highlight at this point that the current guidelines according to their patient management manual(as of late 2023) of the Spanish Society of Neurology establish that, even in the absence of detection of these antibodies, preventive treatment should be considered and maintained to avoid recurrence of relapses in patients diagnosed with MOGAD who have had more than one relapse or recurrent symptoms, as well as in treatment-refractory patients. This therapeutic approach includes the use of specific biological medications designed to modulate the immuneresponse (biological drugs) and thereby reduce the inflammatory activity associated with MOGAD.

Discussion on Emotional and Psychological Impact

Resilience is a crucial aspect in the battle against diseases like MOGAD. I know it's a much-usedterm, especially lately, but it's a daily struggle that most of the time even the medical team themselves are unable to fully appreciate. In this context, my experience as a patient facing thechallenges of this disease aims to underscore the importance of emotional strength and support from close surroundings. It's crucial to have support from a multidisciplinary team, which I believe is essential in treating a chronic patient, as in the case of MOGAD.

As a patient, I have demonstrated a remarkable ability to adapt and resist the obstacles posed by the disease. Throughout my journey, I have fortunately had the invaluable support of my husband primarily and other loved ones, who have been a fundamental pillar in my coping process with the disease.

Moreover, being an exceptional case where I am both a doctor and a patient, I have found valuable support in the international scientific community by sharing my story and experienceswith various expert colleagues in MOGAD around the world. This interaction has allowed my personal experience to become a significant contribution to understanding and addressing the disease from a more comprehensive and humane perspective.

As part of my ongoing commitment to research and improving healthcare, I embarked without hesitation on a specialist course in neuroimmunology. This decision reflects my total dedication to thoroughly understanding my illness and actively collaborating with the neurology team that oversees my case, as they had not encountered any patient similar to me. However, it should benoted that studying a medical specialty remotely while dealing with a disabling disease presents significant challenges. The inherent difficulties of this dual task, both physically and emotionally, highlight the determination and perseverance I have in seeking knowledge and improvement.

My commitment to personal growth, improving relationships with close people, and contributing to scientific advancement in the field of immune diseases is hoped to be seen as an inspiring example of how resilience and seeking support can turn challenges into opportunities for growth and empowerment.

The loss of the ability to perform basic activities of daily living not only affects physically but alsohas a profound emotional and psychological impact on the patient's life. It is a painful reminderof human fragility.

Disabling neurological diseases can trigger a cascade of complex emotions, including fear and uncertainty about the future. These are common and understandable responses to the loss of physical and cognitive function. The prospect of facing a life marked by limitations and dependency generates profound distress, at least initially in the course of the disease.

Furthermore, it not only affects the patient in this case but also their personal and social relationships. Patients may experience feelings of isolation and loneliness as their ability to engage in social activities diminishes. Imagine, if it is already complicated to have the empathy of the healthcare professional, who may understand the etiopathogenesis of the disease, its symptoms, and its evolution, it is much more difficult to make the patient's closest circle understand what a disease like MOGAD entails and its consequences and impact on daily life, since many of the symptoms are "silent" and imperceptible to another individual. Consequently,close relationships, such as family and friends, may struggle to adapt to the change in the patient's health status, leading to tensions and conflicts, thereby exacerbating the difficulty in managing the patient's emotions, even generating low self-esteem and a greater sense of loneliness and incomprehension.

In this context, it is essential for the medical team to understand and acknowledge the emotional trauma experienced by patients with disabling neurological diseases. Empathy and sensitivity from healthcare professionals can make a significant difference in the patient's quality of life. It is crucial for doctors not only to focus on the clinical aspect of the disease but also to consider the psychological and emotional impact it has on the patient's life.

In conclusion, disabling neurological diseases, and in this case MOGAD, represent a challenge not only from a physical standpoint but also emotional and social. Fear and uncertainty about the future, as well as changes in personal and social relationships, can have a profound impact on patients' lives. It is essential for the medical team to recognise and address these emotional dimensions of the disease to provide comprehensive and empathetic care that improves the quality of life of patients in this difficult situation.

From the perspective of a caregiver of a patient with MOGAD, one can describe the complexity and emotional challenges they face. As a caregiver, my husband is in a unique position to witnessthe daily struggle against debilitating symptoms, as well as to provide physical and emotional support. He is an objective witness to the fasciculations, myoclonus, fatigue, and many other symptoms only evaluable by those closest to the patient and who live with them daily. It is very tough to see the person you love gradually losing abilities, and it is complicated for the caregiverto manage the feeling of being able to cope not only with the disease and its implications but also to feel up to the circumstances and be a pillar for the improvement of the person they care for. The uncertainty about the course of the disease and the effectiveness of treatment also generates anxiety and stress in the caregiver, while the need to adapt to the new demands of care and manage interpersonal relationships with the patient and other family members adds an additional burden. In this sense, it is crucial for caregivers to receive adequate support, both in terms of medical resources and emotional support, to be able to play their role effectively and provide an optimal care environment for the patient with MOGAD.

In conclusion, this is my story, a personal struggle as a fundamental pillar in facing complex diseases. My testimony highlights the importance of emotional and scientific support, as well as the determination to acquire specialized knowledge to collaborate in the prevention andtreatment of my illness.

Intervention and Management

The current management approach to MOGAD focuses on controlling acute flares and preventing long-term recurrences. Immunomodulatory treatments, such as corticosteroids and immunosuppressive agents like rituximab, azathioprine, or mycophenolate mofetil, are commonly used to control inflammation and reduce autoimmune activity in the central nervoussystem. However, response to these treatments may vary among patients, highlighting the needfor individualized assessment and ongoing monitoring to adjust therapy as needed.

In addition to pharmacological treatment, comprehensive management of MOGAD may also include rehabilitation therapies, psychological support, and patient education. Physical and occupational rehabilitation can help improve functionality and quality of life, while psychological support can mitigate the emotional impact of the disease on the patient's well-being and their environment.

Managing MOGAD should involve a multidisciplinary and personalized approach to address symptoms, prevent relapses, and improve patients' quality of life.

It is important to note that MOGAD presents a complex network of comorbidities with other autoimmune and systemic diseases, which adds an additional challenge to the diagnosis and clinical management of affected patients. Identifying these comorbidities is crucial for understanding the varied clinical presentation and potential complications associated with the disease.

One of the most relevant comorbidities is the association of MOGAD with other autoimmune diseases of the central nervous system. The overlap of neurological symptoms between these diseases can hinder diagnostic differentiation and lead to errors in therapeutic management.

Moreover, an association has been observed between MOGAD and a wide range of systemic autoimmune diseases, such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), Crohn's disease, and autoimmune thyroiditis, among others. This coexistence of autoimmune diseases can complicate diagnosis and clinical management, as symptoms may overlap and be erroneously attributed to a single condition.

Another important comorbidity to consider is the predisposition of MOGAD patients to develop thromboembolic disorders, especially in the context of the presence of antiphospholipid antibodies and other abnormalities in blood coagulation. These thrombotic events can have serious consequences and require careful monitoring and management to prevent additional complications.

In summary, the comorbidity between MOGAD and other autoimmune and systemic diseases represents a significant challenge in clinical practice. Early identification and appropriate management of these comorbidities are essential to improve clinical outcomes and the quality of life of affected patients. A comprehensive and multidisciplinary approach, taking into accountthe complexity of these interactions, is essential to provide optimal care for patients with MOGAD and their associated comorbidities.

Next, I will provide a brief overview of the management that has been in place in my specific case against the disease.

Treatment and Clinical Management

The treatment, in my case, has focused on addressing the relapses symptomatically, including the use of corticosteroids during acute relapses, intravenous immunoglobulins, and rituximab to modulate the immune response. However, the lack of a specific therapeutic approach for MOGAD has limited the effectiveness of treatment, and no preventive treatment for relapses has been administered.

After the relapse in 2022, in addition to corticosteroid and immunoglobulin infusions, I also underwent plasma exchange, the efficacy of which could not be evaluated due to complicationsarising from my blood hypercoagulability, which had not been previously considered relevant (even though I had previously experienced bilateral pulmonary embolism in 2019). This hypercoagulability led to venous thrombosis at the site of the plasma exchange catheter, resulting in the immediate suspension of treatment without being able to assess the response. Following this episode, a more in-depth hematological study was conducted, revealing the presence of antiphospholipid syndrome, which is currently being treated with Warfarin.

As a patient, I continue to experience significant chronic pain, dependence on a third party for basic daily activities, decreased vision, spasticity, clonus, paresthesias, and mobility difficulties, all of which have negatively impacted my quality of life.

Currently, I am only receiving treatment for acute symptoms, such as baclofen, clonazepam, gabapentin, and Sativex for spasticity, and paracetamol for pain, which is no longer effective. I have been undergoing physiotherapy and hydrotherapy independently, which help me cope with daily life, as well as engaging in light passive and active exercises to maintain muscle tone and improve posture and mobility as much as possible.

However, it is important to note that every day I wake up in pain, my sleep quality is very poor due to either light sleep or frequent awakenings caused by continuous fasciculations, involuntary muscle contractions, sudden intense pain, difficulty falling asleep due to pain, as well as problems with recent memory retention, sometimes worsened chronic fatigue, and intense headaches.

I am not currently prescribed any biological treatments, and I have not been able to eliminate the pain at any point in these almost 6 years, which greatly limits me, not only due to the disability caused by the sequelae but also due to the social difficulties posed by living a life withchronic intense pain. These challenges can impact interpersonal relationships, leading to a senseof dependence. Additionally, there is the fear of an uncertain future and the frustration of feeling unable to halt the progression of a disease that increasingly restricts me.

Table 2. As an example, a pain intensity measurement table is provided, in which it can be observed, as previously mentioned, that the pain I experience daily starts in the morning at an intensity scale of 7 and can reach 10 at various times throughout the day.

Intensity	Description
0	No pain: No pain experienced, completely pain-free.
1-3	Mild or intermittent pain: Pain is mild or comes and goes, easily tolerable.
4-6	Moderate pain: Pain is moderate and manageable with medication.
7	Intense pain: Pain is intense and frequently interferes significantly with daily activities, requiring medication for relief but not fully eliminated.
8.5-9	Severe pain: Significant increase in pain intensity, with extreme intensity limiting activity and movement.
10	Maximum pain: Moments of maximum pain intensity, characterized by involuntary and sudden muscle contractions, extremely painful such as diaphragmatic spasticity.

Table 3. Comparison of pain intensity in MOGAD versus other neurological conditions.

Neurological Disorder	Pain Scale (usual)	Description
MOGAD (Myelin Oligodendrocyte Glycoprotein Antibody Disease)	7-10	Pain in MOGAD can range from a 7 in the morning to a 10 at night due to sudden involuntary muscle contractions, such as diaphragm spasticity. The intensity of the pain can be debilitating and significantly affect the patient's quality of life.
Multiple Sclerosis	0-10	Pain in multiple sclerosis can range from a burning sensation to stabbing pains. It can manifest in various parts of the body and be chronic or intermittent.
ALS (Amyotrophic Lateral Sclerosis)	0-10	Pain in ALS is variable but typically not a prominent symptom. It may be related to musculoskeletal complications or secondary symptoms but is not usually chronic or constant in most cases.
Peripheral Neuropathies	0-10	Peripheral neuropathies can cause chronic pain, tingling, burning sensation, or other symptoms in the limbs. The intensity of the pain can vary depending on the underlying cause and disease progression.
NMO (Neuromyelitis Optica)	0-10	Pain in NMO may be present during acute bouts of central nervous system inflammation but is not necessarily chronic to the same extent as in MOGAD. It can vary in intensity and duration during periods of disease exacerbation.

Table 4. This table reflects the variation in pain intensity throughout the day in a patient with MOGAD, from a usual intensity of 7 upon awakening to a significant increase to 8.5-9 by the end of the day. Additionally, it highlights the presence of moments of extreme intensity (10) due to sudden muscle contractions, such as diaphragmatic spasticity.

Time of Day	Pain Intensity
Upon Awakening	7
Noon	8
Afternoon	8.5 - 9
Evening	8.5 - 9
Moments of Diaphragmatic Spasticity	10

Discussion

Neurological Demyelinating Diseases, particularly MOGAD, pose unique challenges both in diagnosis and clinical management for professionals. Despite their clinical significance, research in this field has been notably limited compared to more common neurological conditions like Multiple Sclerosis (MS), largely due to its low incidence. This lack of research has contributed to widespread ignorance about demyelinating diseases, especially MOGAD, among the medical and scientific community.

Early diagnosis of MOGAD is crucial to prevent disease progression and minimize long-term sequelae. However, lack of awareness and understanding about this disease among neurology teams has led to delayed or incorrect diagnoses in many cases. Failure to recognize atypical symptoms and resistance to considering uncommon diagnoses have contributed to underestimating the prevalence of MOGAD and unnecessary suffering of patients.

Moreover, biases towards MOGAD patients are observed even within the scientific and medical realm. These biases may manifest in neglecting patients' reported symptoms and disregarding their experiences and perspectives, as evidenced by the case presented. Failure to acknowledge patients' voices, even when they are fellow professionals, is deeply concerning and underscores the need for more patient-centered care and closer collaboration between doctors and patients in the diagnostic and treatment process.

Regarding MOGAD treatment, there's a need for unified criteria and therapeutic approaches to optimize clinical outcomes. This includes not only treating acute relapses but also managing daily symptoms and implementing preventive strategies to prevent recurrence. Research into new therapies specifically targeting MOGAD is essential to improving patient prognosis and addressing unmet medical needs in this field.

Currently, there's a lack of systematic consideration regarding the possibility of pseudo-relapses in the context of demyelinating diseases like MOGAD, unlike in diseases such as MS. This omission is based on the premise that only a new relapse is considered if there's a detection of anti-MOG antibodies positivity in specific serological tests. However, from a clinical perspective, this restriction may be considered risky and limited since not all patients present the same alterations and are more heterogeneous than in other types of demyelinating diseases.

As a physician and patient with experience in this field, I argue that this approach may expose the patient to a greater risk of developing a new disabling relapse, potentially resulting in more severe sequelae and disabilities. Furthermore, the presence of recurrent symptoms or clinical exacerbations, even in the absence of anti-MOG antibodies positivity, may indicate the occurrence of pseudo-relapses requiring appropriate therapeutic intervention to prevent further harm.

It's essential to recognize that evaluating new symptoms or clinical exacerbations in patients with MOGAD can be challenging due to variability in clinical presentation and symptom overlap with other neurological disorders. Medical teams may face difficulties in assessing the importance of new symptomatology and differentiating it from pre-existing symptoms or other pathological processes, leading to underestimation of the severity of the situation and delay in appropriate therapeutic intervention.

In this regard, I advocate for a more holistic and sensitive evaluation of patients with MOGAD, considering both positivity of anti-MOG antibodies and the presence of recurrent clinical symptoms or exacerbations not attributable to other known causes. This would allow timely and appropriate therapeutic intervention to prevent disease progression and improve long-term clinical and functional outcomes.

In summary, it is imperative to increase awareness and research into demyelinating diseases, especially MOGAD, within the medical and scientific community. This includes the need to recognize and address existing biases towards MOGAD patients, as well as promoting closer collaboration between doctors and patients in the diagnostic and treatment process. Patients' voices and experiences must be valued and considered in all aspects of medical care, and active efforts should be made to improve the quality of life of those affected by demyelinating diseases such as MOGAD.

This case highlights the unique challenges associated with the diagnosis and management of anti-MOG positive NMO, particularly in a context where the disease is poorly recognized by physicians. Variability in clinical presentation and lack of awareness about this disease can lead to misdiagnoses and treatment delays. The importance of considering NMO in patients with atypical neurological symptoms, along with the need for specific diagnostic tests, such as autoantibody analysis, is underscored in this case.

Reflexive Discussion

MOGAD is a rare autoimmune neurological disorder that affects the central nervous system. Scientific literature has progressed in understanding this disorder, but often it is in the personal experience of patients where nuances and details that greatly enrich our understanding can be found.

By examining the personal experience of a patient with MOGAD and comparing it with relevant literature, it is possible to identify unique aspects that may not have been highlighted in scientific studies. For example, how symptoms affect daily quality of life, the psychological impact of the disease, or even the individual response to treatment could be crucial elements that a patient can contribute to clinical research. This comparison between personal experience and relevant literature serves to contextualize the disease in a more comprehensive framework. Patients' personal stories can provide unique perspectives that add to the body of knowledge established through objective evidence.

Reflecting on how an individual case of MOGAD can inform and enrich scientific and clinical understanding of the disease involves considering not only the medical and biological aspects but also the human and emotional aspects involved. By integrating these diverse perspectives, a more complete and empathetic understanding of the disease can be obtained, which in turn can improve the care and treatment provided to patients.

In summary, the personal experience of a patient with MOGAD can provide valuable insights that complement and enrich the existing scientific literature, thereby contributing to a greater understanding of this relatively "new" pathological entity.

As a patient with MOGAD, I find myself immersed in a whirlwind of complex and challenging emotions. Uncertainty about my health status and fear that new pains will persist become constant companions in my daily life. Over the four years prior to diagnosis, my life has been marked by a frustrating quest for answers, facing the lack of something that could shed light on my condition and guide the way towards appropriate treatment. The two years following the diagnosis have also been marked by frustration, not only due to the progressive loss of abilities but also due to the ncertainty of an uncertain future, the search for treatments, and the relentless quest for answers. This constant struggle is exacerbated by the lack of empathy and understanding from the medical staff who have treated me, who seem not to grasp the magnitude of the debilitating pain I experience. The feeling of being ignored or minimized in my experiences of pain increases my sense of despair and isolation. Furthermore, the lack of clarity about the prognosis of my disease and the possibilities of improvement adds an additional layer to my situation. In this context, pain becomes an omnipresent enemy that dictates the limits of my daily life, while the lack of medical attention focused on pain management and prevention of a new flare reinforces the sense of helplessness and hopelessness. In this journey marked by illness, I long not only for relief from physical pain but also for genuine recognition and support from medical staff, who recognize the crucial importance of addressing my experience as a patient with MOGAD.

Conclusions

In conclusion, Neuromyelitis Optica Spectrum Disorder (NMOSD) Anti-MOG Positive Disease (MOGAD) remains a **largely unexplored field** in medical research, with limited understanding ofits pathophysiology and specific treatment options. Lack of knowledge and awareness among medical teams about this disease has resulted in delayed or incorrect diagnoses, exacerbating frustration and significant sequelae experienced by affected patients.

It is important to highlight the variability of symptoms from one patient to another in MOGAD, underscoring the need to address patients holistically rather than segmenting them by organs or medical specialties. **Late diagnosis can have devastating consequences**, with debilitating sequelae from the first flare and a considerable risk of serious complications, including death.

The implementation of **effective preventive treatment** is crucial to prevent flare recurrence andminimize the risk of long -term sequelae. However, the lack of universal criteria for MOGAD treatment has resulted in a diversity of therapeutic approaches and a lack of consensus in the medical community.

Proper assessment of the existence of **flares and pseudo-flares** in MOGAD is of paramount importance to ensure comprehensive and effective medical care for affected patients. Early identification and distinction between real flares and pseudo-flares can have significant implications for clinical management and prognosis of the disease. Failure to consider pseudo- flares and the restriction to anti-MOG antibody seropositivity as the sole criterion for defining a new flare may lead to an underestimation of the severity of the disease and a delay inappropriate therapeutic intervention. This may expose patients to a higher risk of developing permanent disabilities and debilitating sequelae, as well as worsening quality of life.

Therefore, it is essential to adopt a more complex and sensitive approach in the evaluation of patients with MOGAD, which includes **consideration of recurrent clinical symptoms** or exacerbations not attributable to other known causes. This will allow timely and appropriate therapeutic intervention to prevent disease progression and improve long -term clinical and functional outcomes. Ultimately, awareness and medical education about the importance of assessing flares and pseudo-flares in MOGAD are crucial to improving the care and managementof this poorly understood disease. Only through a comprehensive and collaborative approach between doctors and patients can we hope to optimize outcomes and quality of life for those affected by MOGAD.

From the patient's perspective, it is crucial that the **voices and experiences** of those affected byMOGAD are heard and considered by healthcare professionals. This can help increase awarenessof the disease, improve diagnosis and treatment times, and promote more patient-centered care.

It is important to note that MOGAD is an entity distinct from multiple sclerosis (MS), and as such, requires a **unique approach** in terms of diagnosis, treatment, and clinical management. Given the short time since its recognition as a disease in itself, there is a significant shortage of medical literature on its evolution, prognosis, and usual outcomes of diagnostic tests. The same happens if we encompass it as NMOSD since they have differential characteristics both in imaging tests, as in analytics, as well as in the prognosis and course of the disease.

In Europe and in Spain, particularly, it is crucial to advocate for greater awareness and education among medical professionals about MOGAD, as well as the importance of preventive treatment for affected patients. Only through increased research, interdisciplinary collaboration, and advocacy for patient rights can we hope to improve outcomes and quality of life for those affected by this devastating disease.

The lack of **empathy from doctors** towards the pain experienced by patients with MOGAD is a prominent concern and, unfortunately, a persistent reality in many cases. Pain, being one of the most bothersome and disabling symptoms associated with this disease, deserves special attention from the medical community. It is essential for doctors to recognize and understand the magnitude of the suffering that pain imposes on the daily lives of patients with MOGAD. Moreover, they should adopt a holistic perspective when treating these patients, considering not only the physical aspects of the disease but also its emotional and social implications.

The lack of a definitive cure for MOGAD underscores the importance of addressing and alleviating present symptoms, especially **pain**, as a priority in patient management. When interacting with patients, doctors should strive to cultivate a relationship based on trust and compassion, demonstrating genuine concern for the patient's well-being and showing empathytowards their experience of pain. This empathetic attitude not only strengthens the doctor- patient relationship but also significantly contributes to improving the overall patient experiencewith medical care.

My personal experience as a patient, combined with my medical background, highlights the need for greater awareness and education among the medical community about NMOSD anti- MOG positive. Early diagnosis and appropriate treatment are crucial to improving clinicaloutcomes and quality of life for affected patients. Greater research is required into specific therapies for MOGAD, as well as a comprehensive approach to addressing daily symptoms and needs of diagnosed patients.

In conclusion, the **inclusion of MOGAD in medical diagnostic protocols is crucial** to ensure early and accurate identification of this disease in patients with compatible symptoms. The implementation of simple and inexpensive tests, such as Anti-MOG antibody analysis, in combination with comprehensive clinical evaluation, will allow for more efficient detection andappropriate medical care for affected patients. This action can lead to faster diagnosis, timely treatment, and significant improvement in clinical outcomes and quality of life for patients withMOGAD, in addition to being a much less expensive screening test than other objective tests such as MRI or CSF analysis.

Finally, it is imperative to recognize that the size of lesions on magnetic resonance imaging or the negativization of Anti-MOG antibodies after treatment does not rule out the presence of MOGAD in patients with recurrent symptoms, as it is a chronic disease with a higher recurrenceof flares in the first 5 to 6 years after diagnosis. The need for a preventive approach in treating this disease, even after apparent remission in objective tests, with the need to avoid flare reactivation and prevent long-term clinical deterioration is evident. This perspective emphasizes the importance of continuous monitoring and **proactive medical care** to optimize long-term outcomes in patients with MOGAD.

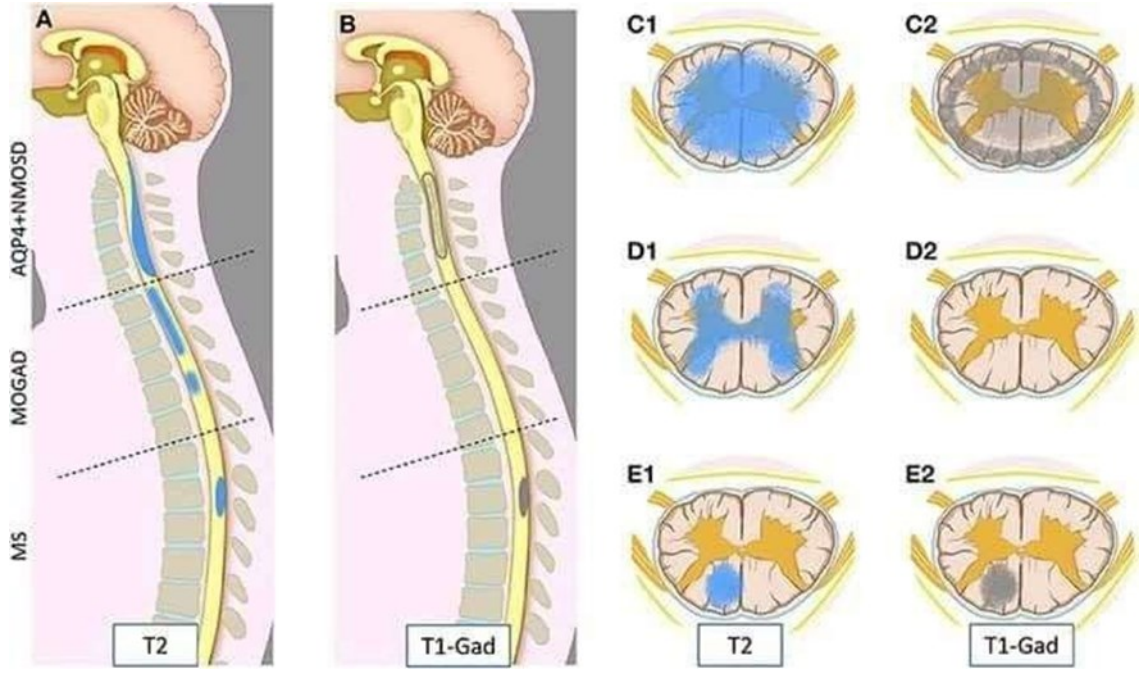


Image 3. *Representation of how lesions may not be visible on the MRI of MOGAD compared to NMO and MS.*

Informed consent

I, as the author of the article and patient, hereby give my consent for the use and publication ofmy own personal medical data in the scientific article entitled "CASE STUDY: Personal Experience as a Physician and Patient of Anti-MOG Positive Neuromyelitis Optica (MOGAD)" to be presented and/or published in the SVOA Neurology (SVOA-NE) journal ISSN: 2753-9180. I understand thatthese data will include information related to my medical history, diagnosis, treatment, and clinical outcomes associated with my health condition.

I declare that I have been duly informed about the purpose and scope of the scientific article, aswell as the importance of sharing my personal experience in relation to the discussed disease therein. I acknowledge that the disclosure of my personal medical data is voluntary and that I have the right to withdraw my consent at any time prior to the publication of the article.

I understand that all reasonable efforts will be made to protect my privacy and confidentiality during the process of drafting, reviewing, and publishing the article. I agree that my name may be used in connection with the disclosure of my personal medical data, provided that it is doneappropriately and respectfully.

Furthermore, I understand that the scientific article may be reviewed and cited by other researchers, physicians, and healthcare professionals in the academic and clinical fields. Iconsent to my personal medical data being used for research and educational purposes, provided that applicable ethical and legal principles are respected.

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Conflict of Interest

None.

References

Abdelhak, A. (2022). Blood GFAP as an emerging biomarker in brain and spinal cord disorders.Nat Rev Neurol, 18(3), 158-172.

Ambrosius, W. (2020). Myelin oligodendrocyte glycoprotein antibody-associated disease:Current insights into patho-physiology, diagnosis, and treatment. Int J Mol Sci, 22(1).

Arru, G. (2020). HERV-W antibody response in patients with MOG-IgG-associated disorders,multiple sclerosis, and NMOSD. J Neuroinflammation, 338.

Asseyer, S. (2020). Prodromal headache in MOG-IgG-positive optic neuritis. Mult Scler RelatDisord, 40.

Bancos, S. A. (2021). Brainstem and cerebellar involvement in MOG-IgG-associated disorderversus aquaporin-4-IgG and MS. J Neurol Neurosurg Psychiatry, 92(4), 384-390.

Banwell, B. (2023). Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: Proposed criteria by the International MOGAD Panel. Lancet Neurol, 22(3), 268-282.

Brain and spinal cord atrophy in NMOSD and MOGAD: Current evidence and future perspectives L. Lorefice a, R. Cortese b a Department of Medical Sciences and Public Health.(n.d.). Multiple Sclerosis Center.

Bruijstens, A. L. (2020). EU paediatric MOG consortium: Part 1: Classification of clinical phenotypes of paediatric myelin oligodendrocyte glycoprotein antibody-associated disorders.Eur J Paediatr Neurol, 29, 2-13.

Bülbül, A. (n.d.). Understanding multiple Sclerosis and MOGAD. Alperbulbul.Co. Retrieved April5, 2024, from https://www.alperbulbul.co/blog/genetics_148

Cacciaguerra, L. (2019). Brain and spinal cord imaging features in neuromyelitis opticaspectrum disorders. Ann Neurol, 85(3), 371-384.

Carta, S. (2021). MOG antibodies alone in CSF: Pathological findings support diagnostic value.Acta Neuropathol, 141(5), 801-804.

Carta, Sara et al. “Significance of Myelin Oligodendrocyte Glycoprotein Antibodies in CSF: A Retrospective Multicenter Study.” *Neurology* vol. 100,11 (2023): e1095-e1108. doi:10.1212/WNL.0000000000201662

Corbali, O., & Chitnis, T. (2023). Pathophysiology of myelin oligodendrocyte glycoprotein antibody disease. Frontiers in Neurology, 14. https://doi.org/10.3389/fneur.2023.1137998

Correale, J. (2019). Mechanisms of neurodegeneration and axonal dysfunction in progressivemultiple sclerosis. Biomedicines, 7(1).

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): Currentknowledge and challenges. (2023). J Neurol, 270(8), 4132-4150.

Fadda, G. (2022). Characteristics and outcomes of myelitis in central nervous system demyelinating disorders: Comparison among multiple sclerosis, MOGAD, and AQP4-IgG-positive NMOSD. Front Neurol, 13.

Flanagan, E. P., & Waters, P. J. (2023). Serum MOG-IgG titres should be routinely performed inMOGAD diagnosis and follow-up: No. Mult Scler, 29(8), 928-929

Geraldes, R. (2018). The current role of magnetic resonance imaging in differentiating multiplesclerosis from its imaging mimics. Nat Rev Neurol, 14(4), 199-213.

Ayroza Galvão Ribeiro Gomes, Ana Beatriz et al. "Immunoglobulin A Antibodies Against Myelin Oligodendrocyte Glycoprotein in a Subgroup of Patients With Central Nervous System Demyelination." *JAMA neurology* vol. 80,9 (2023): 989-995. doi:10.1001/jamaneurol.2023.2523

Höftberger, R. (2020). Pathology of myelin oligodendrocyte glycoprotein inflammatory demyelinating disease of the central nervous system accompanying autoantibody. ActaNeuropathol, 139(5), 875-892.

Hor, J. Y., & Fujihara, K. (2023). Epidemiology of myelin oligodendrocyte glycoprotein antibody-associated disease: A review of worldwide prevalence and incidence. Neurol Front, 14.

Jarius, S. (2016). MOG-IgG in NMO and related disorders: a multicentre study of 50 patients.Part 3: Brainstem involvement: frequency, presentation and outcome. J Neuroinflammation,13(1).

Jarius, Sven, in cooperation with the Neuromyelitis Optica Study Group (NEMOS), Ruprecht, K.,Kleiter, I., Borisow, N., Asgari, N., Pitarokoili, K., Pache, F., Stich, O., Beume, L.-A., Hümmert, M.W., Ringelstein, M., Trebst, C., Winkelmann, A., Schwarz, A., Buttmann, M., Zimmermann, H., Kuchling, J., Franciotta, D., … Wildemann, B. (2016). MOG-IgG in NMO and related disorders: a multicentre study of 50 patients. Part 2: Epidemiology, clinical presentation, radiological and laboratory features, treatment responses, and long-term outcome. Journal of Neuroinflammation, 13(1). https://doi.org/10.1186/s12974-016-0718-0

Jeong, I. H. (2016). Comparative analysis of treatment outcomes in patients with neuromyelitisoptica spectrum disorder using multifaceted assessment criteria. Mult Scler, 22(3), 329-339.

Jurynczyk, M. (2017). Clinical presentation and prognosis of MOG antibody disease: a UK study.Brain, 140(12), 3128-3138.

Jurynczyk, Maciej, Messina, S., Woodhall, M. R., Raza, N., Everett, R., Roca-Fernandez, A., Tackley, G., Hamid, S., Sheard, A., Reynolds, G., Chandratre, S., Hemingway, C., Jacob, A., Vincent, A., Leite, M. I., Waters, P., & Palace, J. (2017). Clinical presentation and prognosis inMOG-antibody disease: a UK study. Brain: A Journal of Neurology, 140(12), 3128-3138. https://doi.org/10.1093/brain/awx276

Kaneko, K. (2018). CSF cytokine profile in MOG-IgG+ neurological disease is similar to NMOSDAQP4-IgG+ but distinct from MS: a cross-sectional study and potential therapeutic implications. J Neurol Neurosurg Psychiatry, 89(9), 927-936.

Keller, C. W. (2021). Complement activation is a prominent feature of MOGAD. Ann Neurol,90(6), 976-982.

Kwon, Y. N. (2021). Myelin oligodendrocyte glycoprotein-immunoglobulin G in CSF: clinical implications of testing and association with disability. Neurol Neuroimmunol Neuroinflamm,9(1).

Lin, L. (2022). Plasma complement 3 and complement 4 are promising biomarkers to distinguish NMOSD from MOGAD and are associated with blood-brain barrier disruption inNMOSD. Front Immunol, 13.

Association of MOG-IgG serostatus with relapse after acute disseminated encephalomyelitis and proposed diagnostic criteria for MOG-IgG–associated disorders. JAMA Neurology, 75(11),1355. https://doi.org/10.1001/jamaneurol.2018.1814

Distribution criteria of brain lesions distinguish MS from NMOSD by AQP4 antibodies and fromMOG antibodies disease. J Neurol Neurosurg Psychiatry Res, 88(2), 132-136.

Mader, S. (2023). Dissection of pathogenic mechanisms mediated by Fc receptor and complement of autoantibodies against myelin oligodendrocyte glycoprotein. Proc Natl Acad SciUSA, 120(13).

Maillart, E., Deiva, K., & Marignier, R. (2024). Clinical characteristics of patients with myelinoligodendrocyte glycoprotein antibodies. Current Opinion in Neurology, 10.1097/WCO.0000000000001265. https://doi.org/10.1097/wco.0000000000001265

Marignier, R. (2021). Disease associated with antibodies against myelin-oligodendrocyteglycoprotein. Lancet Neurol, 20 (9), 762-772.

Mariotto, S. (2019). Relevance of myelin oligodendrocyte glycoprotein antibodies incerebrospinal fluid of seronegative cases. Neurology, 93(20), e1867-e1872.

Mason, K. (2024, marzo 19). New diagnostic criteria for MOGAD distinguishes the disease fromMS and NMOSD. Cuanschutz.edu. https://news.cuanschutz.edu/ophthalmology/new- diagnostic-criteria-for-mogad-distinguishes-the-disease-from-ms-and-nmosd

Montalvo, M. (2022). Acute symptomatic seizures secondary to myelin oligodendrocyteglycoprotein antibody-associated disease. Epilepsy, 63(12), 3180-3191.

neurology [@mohamud_neuropsy]. (2024, March 23). MOGAD; Myelin oligodendrocyte glycoprotein antibody associated disease. Youtube. https://www.youtube.com/watch?v=-78PEWfUc1Y

Reindl, M., & Rostasy, K. (2023). Serum MOG IgG titres should be routinely assessed in thediagnosis and follow-up of MOGAD: Yes. Mult Scler, 29(8), 926-927.

Reindl, M., & Waters, P. (2019). Antibodies against myelin oligodendrocyte glycoprotein inneurological diseases. Nat Rev Neurol, 15(2), 89-102.

Rode, J. (2023). Time to steroid treatment affects visual outcome in optic neuritis in MOGAD. JNeurol Neurosurg Psychiatry, 94(4), 309-313.

Stiebel-Kalish, H. (2019). Is time equal to vision in acute treatment of AQP4 and MOG opticneuritis cohort? Neurol Neuroimmunol Neuroinflamm, 6(4).

Watanabe, M. (2019). Serum GFAP and neurofilament light as biomarkers of disease activityand disability in NMOSD. Neurology, 93(13), e1299-e1311.

Whittam, D. H. (2020). Treatment of MOG-IgG-associated disorder with rituximab: aninternational study of 121 patients. Multiple Sclerosis Journal, 44.

Wingerchuk, D. M., Banwell, B., Bennett, J. L., Cabre, P., Carroll, W., Chitnis, T., de Seze, J., Fujihara, K., Greenberg, B., Jacob, A., Jarius, S., Lana-Peixoto, M., Levy, M., Simon, J. H., Tenembaum, S., Traboulsee, A. L., Waters, P., Wellik, K. E., Weinshenker, B. G., & International

Panel for NMO Diagnosis. (2015). International consensus diagnostic criteria for neuromyelitisoptica spectrum disorders. Neurology, 85(2), 177-189. https://doi.org/10.1212/WNL.0000000000001729

Yang, M. (Ed.). (n.d.). Neuronal antibodies as diagnostic biomarkers of autoimmune encephalitis and demyelinating inflammatory disease Neuronal antibodies are diagnosticbiomarkers of autoimmune encephalitis.

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