كليـــــــة الـطـب
والصيدلة - مراكش
FACULTÉ DE MÉDECINE
ET DE PHARMACIE - MARRAKECH
جامعة القاضي عياض
UNIVERSITÉ CADI AYYAD

Year 2025 Thesis N°:190

Myelin Oligodendrocyte Glycoprotein Antibody Associated Disease (MOGAD):

Experience of the Neurology Department at Mohammed VI University Hospital Center in Marrakech

THESIS

PRESENTED AND DEFENDED PUBLICLY ON 18/06/2025

BY

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Born on 20/06/1996 in Youssoufia

TO OBTAIN THE DEGREE OF DOCTOR OF MEDECINE

KEYWORDS

MOGAD – Clinical presentation – MRI features – Anti MOG –Treatment

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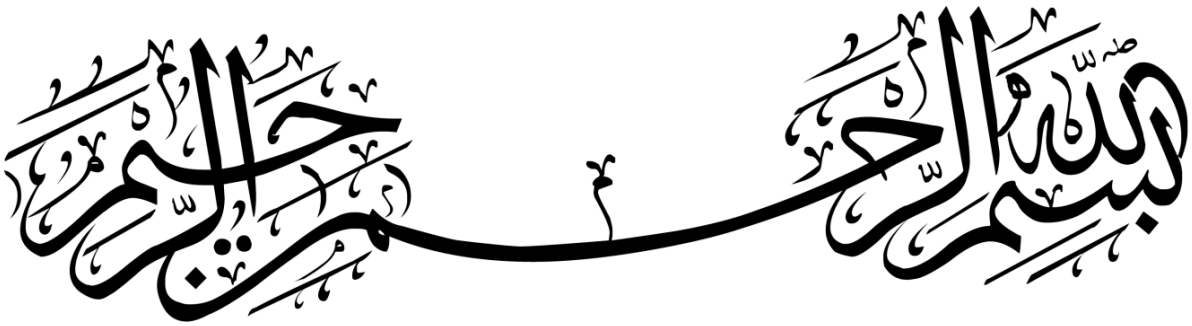

سورة آل عمران

بسم الله الرحمن الرحيم

إِنَّ فِي خَلْقِ ٱلسَّمَٰوَٰتِ وَٱلْأَرْضِ وَٱخْتِلَٰفِ ٱلَّيْلِ وَٱلنَّهَارِ
لَءَايَٰتٍ لِّأُو۟لِى ٱلْأَلْبَٰبِ ﴿١٩٠﴾

Hippocratic Oath

Upon being admitted to the medical profession, I pledge my life to the service of humanity.
I will treat my teachers with the respect and gratitude they deserve.
I will practice my profession with conscience and dignity.
The health of my patients will be my first goal.
I will not betray the secrets entrusted to me.
I will maintain by all means in my power the honor and noble traditions of the medical profession.
The physicians will be my brothers.
No consideration of religion, nationality, race, political and social considerations will come between my duty and my patient.
I will maintain strict respect for human life from the moment of conception.
Even under threat, I will not use my medical knowledge in a manner contrary to the laws of humanity.
I pledge this freely and on my honor.
Declaration of Geneva, 1948.

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314	TITOU Hicham	MC	Dermatologie
315	EL GHOUL Naoufal	MC	Traumato-orthopédie
316	BAHI Mohammed	MC	Anesthésie-réanimation
317	RAITEB Mohammed	MC	Maladies infectieuses
318	DREF Maria	MC	Anatomie pathologique
319	ENNACIRI Zainab	MC	Psychiatrie
320	BOUSSAIDANE Mohammed	MC	Traumato-orthopédie
321	JENDOUZI Omar	MC	Urologie
322	MANSOURI Maria	MC	Génétique
323	ERRIFAIY Hayate	MC	Anesthésie-réanimation
324	BOUKOUB Naila	MC	Anesthésie-réanimation
325	OUACHAOU Jamal	MC	Anesthésie-réanimation
326	EL FARGANI Rania	MC	Maladies infectieuses
327	IJIM Mohamed	MC	Pneumo-phtisiologie
328	AKANOUR Adil	MC	Psychiatrie
329	ELHANAFI Fatima Ezzohra	MC	Pédiatrie
330	MERBOUH Manal	MC	Anesthésie-réanimation
331	BOUROUMANE Mohamed Rida	MC	Anatomie
332	IJDDA Sara	MC	Endocrinologie et maladies métaboliques
333	GHARBI Khalid	MC	Gastro-entérologie
334	ATBIB Yassine	MC	Pharmacie clinique
335	EL GUAZZAR Ahmed (Militaire)	MC	Chirurgie générale
336	HENDY Iliass	MC	Cardiologie

337	MOURAFIQ Omar	MC	Traumato-orthopédie
338	ZAIZI Abderrahim	MC	Traumato-orthopédie
339	HATTAB Mohamed Salah Koussay	MC	Stomatologie et chirurgie maxillo faciale
340	DEBBAGH Fayrouz	MC	Microbiologie-virologie
341	OUASSIL Sara	MC	Radiologie
342	KOUYED Aicha	MC	Pédopsychiatrie
343	DRIOUICH Aicha	MC	Anesthésie-réanimation
344	TOURAIF Mariem	MC	Chirurgie pédiatrique
345	BENNAOUI Yassine	MC	Stomatologie et chirurgie maxillo faciale
346	SABIR Es-said	MC	Chimie bio organique clinique
347	IBBA Mouhsin	MC	Chirurgie thoracique
348	LAATITIOUI Sana	MC	Radiothérapie
349	SAADOUNE Mohamed	MC	Radiothérapie
350	TLEMCANI Younes	MC	Ophtalmologie
351	SOLEH Abdelwahed	MC	Traumato-orthopédie
352	OUALHADJ Hamza	MC	Immunologie
353	BERGHALOUT Mohamed	MC	Psychiatrie
354	EL BARAKA Soumaya	MC	Chimie analytique-bromatologie
355	KARROUMI Saadia	MC	Psychiatrie
356	ZOUITEN Othmane	MC	Oncologie médicale
357	EL-OUAKHOUMI Amal	MC	Médecine interne
358	AJMANI Fatima	MC	Médecine légale
359	MENJEL Imane	MC	Pédiatrie
360	BOUCHKARA Wafae	MC	Gynécologie-obstétrique
361	ASSEM Oualid	MC	Pédiatrie
362	ELHANAFI Asma	MC	Médecine physique et réadaptation fonctionnelle
363	ABDELKHALKI Mohamed Hicham	MC	Gynécologie-obstétrique
364	ELKASSEH Mostapha	MC	Traumato-orthopédie
365	EL OUAZZANI Meryem	MC	Anatomie pathologique
366	HABBAB Mohamed	MC	Traumato-orthopédie
367	KHAMLIJ Aimad Ahmed	MC	Anesthésie-réanimation
368	EL KHADRAOUI Halima	MC	Histologie-embryologie-cyto-génétique
369	ELKHETTAB Fatimazahra	MC	Anesthésie-réanimation
370	SIDAYNE Mohammed	MC	Anesthésie-réanimation

371	ZAKARIA Yasmina	MC	Neurologie
372	BOUKAIDI Yassine	MC	Chirurgie Cardio-vasculaire

LISTE ARRETEE LE 03/02/2025

DEDICATIONS

To God the Almighty, for His endless blessings and guidance.

To My Beloved Parents Abdelghani BENSAIH and Leila RACHDI:

Dear Father and Mother,

Words cannot fully express my gratitude for everything you have done for our family and for me. Thank you for always being there for me, supporting me through every step, and believing in me unconditionally. Your sacrifices, love, and guidance have been the driving force behind everything I have accomplished.

This thesis is dedicated to you as a small token of my appreciation. None of this would have been possible without your unwavering support and encouragement.

May Allah bless you with good health, endless joy, and a long, fulfilling life.

To my lovely sister Abir BENSAIH:

Dear Abir,

You have been my constant companion through every twist and turn in life—celebrating the joyous moments and offering unwavering support during the toughest times. I deeply admire the strong, kind, and thoughtful woman you have become. Your generosity, humor, and genuine care for others inspire me every day. Your kindness and presence bring so much light to our family.

May Allah bless you with happiness, success, and all the goodness this world has to offer. I am forever grateful to witness your journey and see you grow into the remarkable person you are.

To my wonderful brother Mohamed-Idriss BENSAIH:

Dear Mohamed-Idriss,

Having you as my brother is truly a blessing. Your optimism, sense of humor, and your ability to bring happiness to everyone around you are qualities I truly cherish.

Thank you for being such an amazing brother, I am incredibly proud of you, not only for your brilliant grades in school, but also for the kind, intelligent, and talented person you are becoming.

May Allah bless you with endless success and happiness. I look forward to seeing you grow, thrive, and accomplish your dreams.

To All My Friends,

Thank you for your love, support, and the countless moments we've shared. From the laughter that brightened our days to the challenges we faced together.

Your friendship has been a source of strength and joy, and I am truly grateful for each and every one of you.

Through the ups and downs, your presence has made this journey even more meaningful.

ACKNOWLEDGMENTS

TO THE PRESIDENT OF THE JURY: Professor Mona ZAHLANE:

We are deeply honored by your acceptance to chair the jury for this thesis. Throughout our studies, we have benefited from your excellent teaching, which has always been clear, helpful, and encouraging.

It is with deep gratitude that we acknowledge your role as the chair of our thesis jury. Your guidance and expertise have been invaluable.

We sincerely appreciate the time and dedication you have given to evaluating our work.

With respect and gratitude, we thank you.

TO THE SUPERVISOR AND DIRECTOR OF THIS THESIS: Professor Nissrine LOUHAB:

I am profoundly grateful to have had you as a mentor throughout this journey. Your kindness, humility, and visionary approach to research have made this experience truly rewarding.

You have consistently guided me with thoughtful advice and provided invaluable insights that have shaped this work. Your patience and encouragement have had a profound impact, shaping both this research and my approach to scientific inquiry. I would like to thank you for introducing me to this subject and trusting me with this work. I will always be grateful for your availability and patience, especially considering your busy schedule and professional obligations.

This thesis would not have been possible without your dedication and support. I am honored to have learned from your expertise and wisdom. Please accept my deepest gratitude and respect.

TO THE JUDGES

Professor Brahim ADMOU:

It is a great honor to have you as part of this distinguished jury.

Your expertise in immunology, along with your kindness and experience, is deeply valued. We are profoundly grateful for the time and effort you have devoted to reviewing our work, and we have great respect for your judgment and insight.

Professor Mohamed CHRAA:

We are honored to have you as part of this jury. Your expertise and professionalism have always been an inspiration. I deeply appreciate your involvement and the time you've dedicated to evaluating my work. Please accept my sincere gratitude and respect.

Professor Dounia BASRAOUI:

It is a great honor for us to have you as part of this distinguished jury. We would like to express our deep gratitude for your time and attention in reviewing our work. We deeply appreciate your availability and the time you've dedicated to evaluating my work. Please accept our sincere expression of respect and deep appreciation.

Professor Sara BELGHMAIDI:

We are honored to have you as a member of the jury for our thesis. Your expertise and insights are invaluable, and your contribution will significantly enhance the quality of this academic endeavor. We are truly grateful for your commitment to supporting and guiding our research.

To the medical teams of the following departments: Neurology, Internal Medicine, Immunology, Radiology and Ophthalmology.

I would like to express my sincere gratitude to all the individuals who contributed to the successful completion of this thesis.

ABREVIATIONS

LIST OF ABBREVIATIONS

MOG	: Myelin Oligodendrocyte Glycoprotein
AQP4	: Aquaporin-4 (related to Neuromyelitis Optica)
NMO	: Neuromyelitis Optica
MS	: Multiple Sclerosis
MRI	: Magnetic Resonance Imaging
CSF	: Cerebrospinal Fluid
IVIG	: Intravenous Immunoglobulin
EDSS	: Expanded Disability Status Scale
FLAIR	:Fluid Attenuated Inversion Recovery (MRI sequence)
IgG	: Immunoglobulin G
IgM	: Immunoglobulin M
BBB	: Blood-Brain Barrier
T2WI	: T2-Weighted Imaging (MRI technique)
T1WI	: T1-Weighted Imaging (MRI technique)
ON	: Optic Neuritis
OCT	: Optical Coherence Tomography
VEP	: Visual Evoked Potentials
ADEM	: Acute Disseminated Encephalomyelitis
PCR	: Polymerase Chain Reaction
LP	: Lumbar Puncture
PE	: Papilledema
IIT	: Idiopathic Intracranial Hypertension
VA	: Visual Acuity

OUTLINE

INTRODUCTION

Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) is a central nervous system demyelinating disorder recently recognized as an antibody-mediated autoimmune disease (1,2). Although it shares clinical and radiological similarities with NMOSD and MS, MOGAD is now established as a distinct clinical entity [1,2]. It refers to a demyelinating syndrome characterized by the presence of IgG autoantibodies targeting myelin oligodendrocyte glycoprotein (MOG), a transmembrane surface protein found on the outermost layer of CNS myelin and oligodendrocyte [3,4].

The worldwide incidence of MOGAD is approximately 1.6–4.8 per million people, with a prevalence of 1.3–2.5 per 100,000 people. MOGAD's incidence is known for a biphasic behavior, the first peak in children (5–10 years) and the second one in adults (20–30 years) [5–7].

Neurological phenotypes are heterogeneous and dominated by Optic neuritis, followed by ADEM, Myelitis and brainstem involvement [8].

The updated diagnostic criteria published in 2023 have significantly enhanced the ability to distinguish MOGAD from other CNS demyelinating disorders, leading to an increase in the number of diagnosed cases.

Not enough studies have been published about MOGAD in Morocco. Our study aims to describe our experience diagnosing and managing MOGAD cases at the Mohammed VI University Hospital, with a particular focus on the clinical, paraclinical, therapeutic, and prognosis aspects.

PATIENTS AND METHODS

This retrospective study analyzed MOGAD patients hospitalized in the Neurology department at Mohamed VI University Hospital of Marrakech between 2016 and 2024.

Data collection included detailed clinical, paraclinical, therapeutic, and evolutionary information, extracted from a pre-established clinical record sheet (see the annex).

I. Objectives of this study :

This study aimed to review the epidemiological, clinical, and paraclinical characteristics of MOGAD, treatment management and how to prevent relapses of this disease.

II. Inclusion criteria:

Patients were included in this study if they fulfilled all of the following criteria:

1. Clinical presentation compatible with MOGAD, such as:
    - Optic neuritis (unilateral or bilateral)
    - Transverse myelitis (particularly longitudinally extensive)
    - Acute disseminated encephalomyelitis (ADEM)
    - Brainstem or cerebral syndromes suggestive of demyelination

2. Diagnosis made at the Neurology Department of the Mohammed VI University Hospital Center Marrakech between 2016 to 2024.

3. Clinical records available, including:
    - Demographic data
    - Details of clinical presentation
    - MRI imaging
    - Biological findings
    - Treatment received

III. Exclusion criteria:

- Patients with other demyelinating diseases such as MS, NMO, and other auto-immune conditions were excluded from this study.

- Incomplete files.

To rule out infectious causes, all patients underwent systematic serological testing.

IV. Methods :

In this study, we followed these steps :

- **Step 1:**

We created a medical summary sheet (see appendix I), which contains different parameters including Identity, past medical history, clinical and paraclinical features, management, and evolution of the patients.

- **Step 2 :**

We searched and collected the essential data from the medical records.

- **Step 3 :**

We entered the data into Microsoft Excel software, organized and analyzed them.

RESULTS

I. Frequency:

Twelve patients met the 2023 MOGAD diagnostic criteria and were included in this study, accounting for 1.5% of all inflammatory CNS disease cases diagnosed in our department.

II. Age:

The median age at disease onset was 27.8 years old (range: 10–48 years old). The majority of cases occurred in adults (83.3%).

III. Sex:

A slight male predominance was observed, with 66.6% of patients being male (sex ratio 2/1).

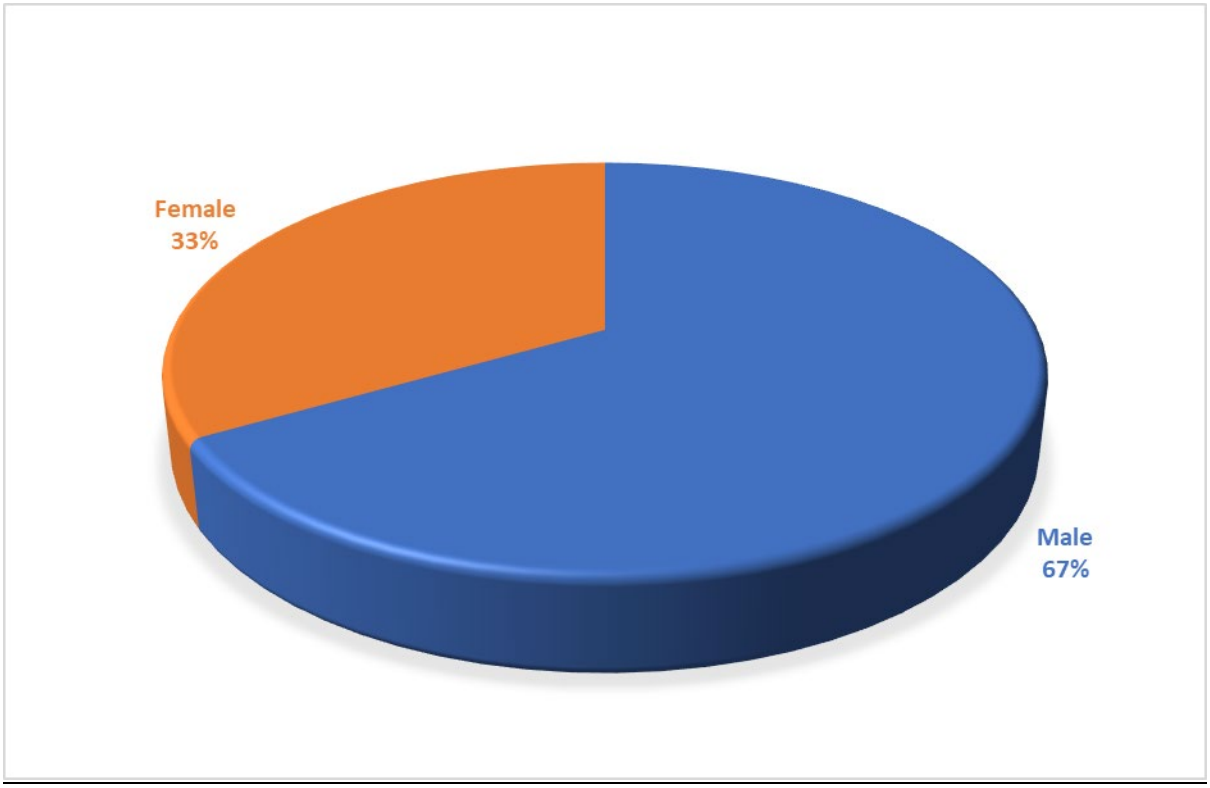


Figure 1: Sex distribution among Mogad patients in our study.

IV. Clinical History:

Three patients had a relevant medical history, one had four prior episodes of optic neuritis, one patient with recurrent oral aphtosis, and one experienced optic neuritis associated with bilateral diplopia.

V. Clinical manifestations:

A severe decrease in visual acuity was the most common symptom at onset in patients presenting with optic neuritis (7 patients), among these, one patient presented with homonymous hemianopsia.

Lower limb weakness with paresthesia were observed in three patients with Myelitis.

Gait difficulties, ataxia, and diplopia were noted in two patients with brainstem and cerebellum involvement.

VI. Clinical examination:

1. Neurological exam:

Patients with optic neuritis showed no additional neurological deficits outside of visual impairment. Patients with myelitis presented with paraparesis or tetrapyramidal syndrome. Concerning the patient presenting with brainstem involvement, the examination revealed cerebellar ataxia, dysarthria, horizontal nystagmus, and gait instability.

2. Ophthalmic exam:

All seven patients with ON exhibited a sudden decline in visual acuity, ranging from counting fingers to 7/10.

Fundoscopic examination, performed in all ON cases, identified papilledema in six ON patients (85.7%).

Visual field testing was conducted on four patients.

VII. Brain and Spinal cord MRI:

Brain and spinal cord MRI were performed in all patients of this study, confirming 7 cases of Optic Neuritis, 2 cases of transverse myelitis as onset clinical presentation, and one case of myelitis as relapse form of ON. One case of brainstem and cerebellar syndrome and one case of cortical and subcortical syndrome. (Details of abnormalities are described in the table below)

Table 1: MRI findings in the patients of our study:

Cases	Brain MRI and spinal cord MRI
17-year-old male with Longitudinally Extensive Myelitis	T2 hyperintensity in the cervical and thoracic spinal cord (C6-T3) with associated spinal cord swelling.
A 43-year-old male with Longitudinally extensive myelitis	Diffuse T2 hyperintensity throughout the cervical spinal cord (C2-C7).
32 year-old male with ON	FLAIR hyperintensity with a globular and thickened morphology of the right optic nerve in its intra-orbital segment (7 mm

	diameter). Moderate hyperintensity is noted on diffusion-weighted imaging, with homogeneous post-gadolinium enhancement. No associated brain lesions.
10-year-old female with ON	Diffuse contrast enhancement of the left optic nerve (6 mm diameter), indistinct margins, and an associated nodular lesion in the temporoparietal region.
48 year-old female with ON	Bilateral T2 hyperintensity of the optic nerves, with additional hyperintensity in the posterior pons.
19 ear-old male with ON	Diffuse T2 hyperintensity of the left optic nerve.
31 female with ON associated with Idiopathic Intracranial Hypertension	- Angiographic sequence of the brain MRI showed evidence of Idiopathic Intracranial Hypertension associated with a slight enhancement in the left optic nerve. - T2 prominent subarachnoid space around the optic nerve. - Enhancement in the left optic nerve. - Transverse sinus Hypoplasia.
26-year-old male with MOGAD-associated encephalopathy	Cortical and subcortical T1 hypointensity associated with T2/FLAIR hyperintensity, without contrast enhancement. The optic nerves are unaffected.
27 year-old male with Cerebellar syndrome associated with brainstem syndrome	Diffuse subtentorial T2 hyperintensity is located in the peri-aqueductal region, pons, and cerebellum. Diffuse Subtentorial T2 hyperintensity located in the peri-aqueduct region, pons and cerebellum.
19-year-old male with ON + Myelitis	Diffuse T2 hyperintensity of the left optic nerve.
45 year-old male with Myelitis	Diffuse longitudinally extensive hyperintensity interesting cervical spine: C2-C5.
26 year-old male with ON	Right optic nerve hyperintensity with contrast enhancement.
48 year-old female with ON	Left optic nerve hyperintensity with contrast enhancement. EPV: left axonal neuropathy.

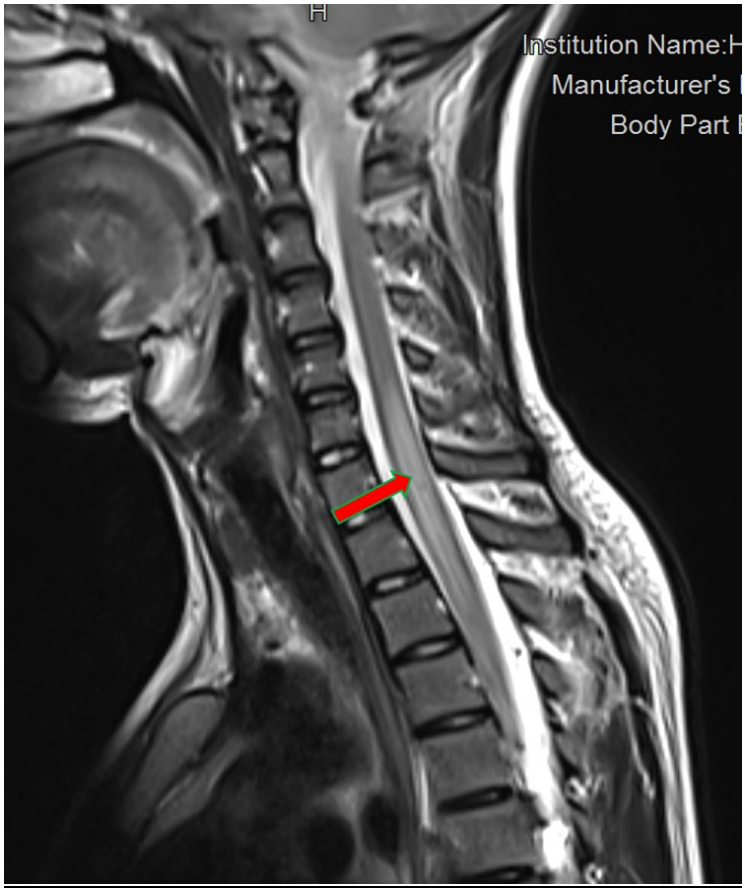


Figure 2: Spinal cord MRI of a 17-year-old MOGAD patient presenting with longitudinally extensive myelitis, showed T2 hyperintensity in the cervical and thoracic spinal cord (C6 to T3).

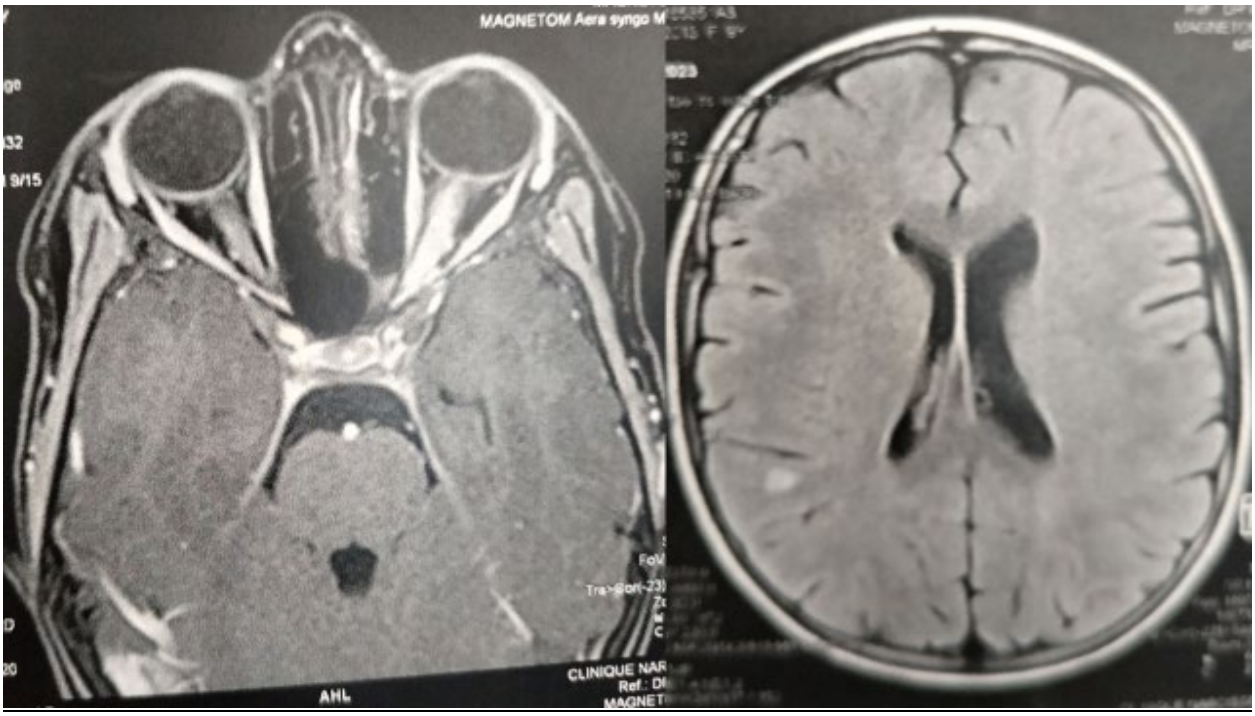


Figure 3: Brain MRI of a 10-year-old female patient presenting with severe optic neuritis. A: Diffuse contrast enhancement of the left optic nerve with Perineural edema; B: juxta-cortical Flair nodular hyperintensity

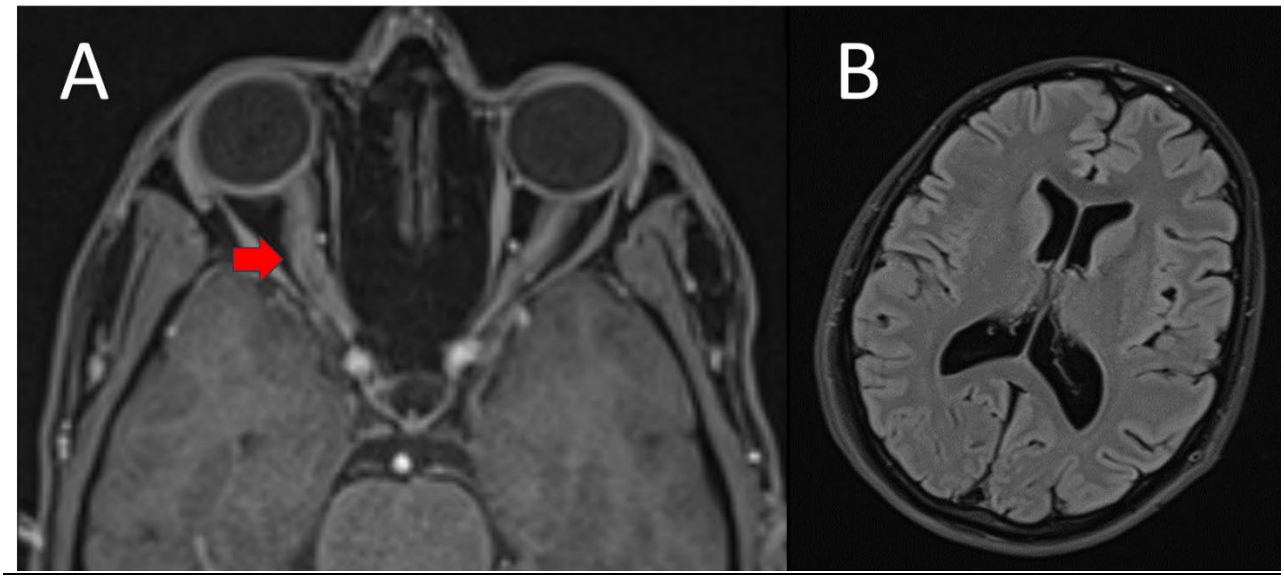


Figure 4: Flair Hyperintensity with Globular and thickened morphology of the right optic nerve in its intra-orbital segment, with a maximum diameter of 7mm, moderate diffusion-weighted

hyperintensity. Additional to a Homogeneous enhancement after gadolinium injection B : Cerebral MRI shows no abnormalities.

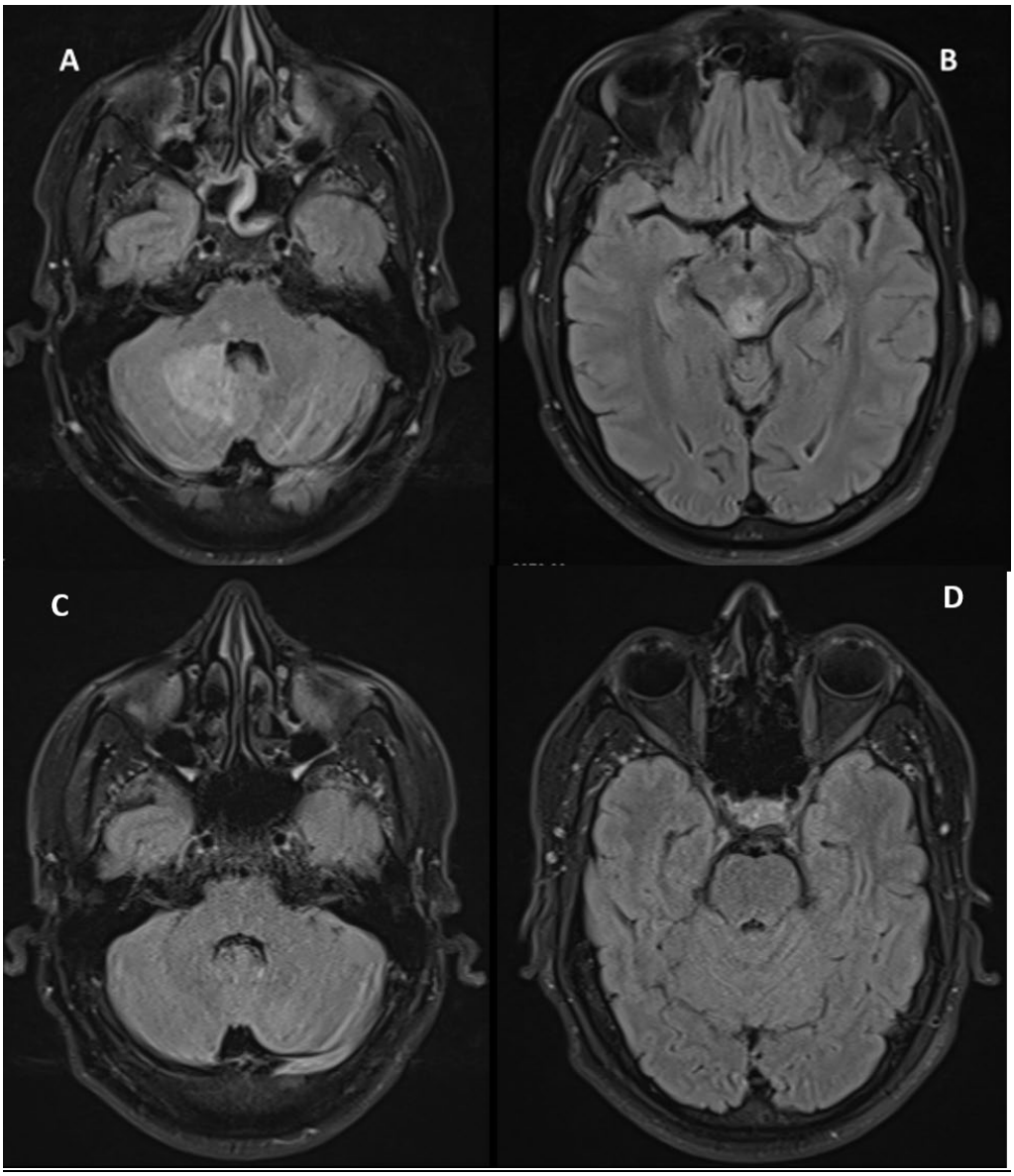


Figure 5: MRI findings of a 17-year-old patient with recurrent diplopia. (A, B) Initial brain MRI reveals diffuse hyperintensity in the peri-aqueductal region, pons, and cerebellum. (C, D) Follow-up MRI demonstrates complete resolution of these lesions after treatment.

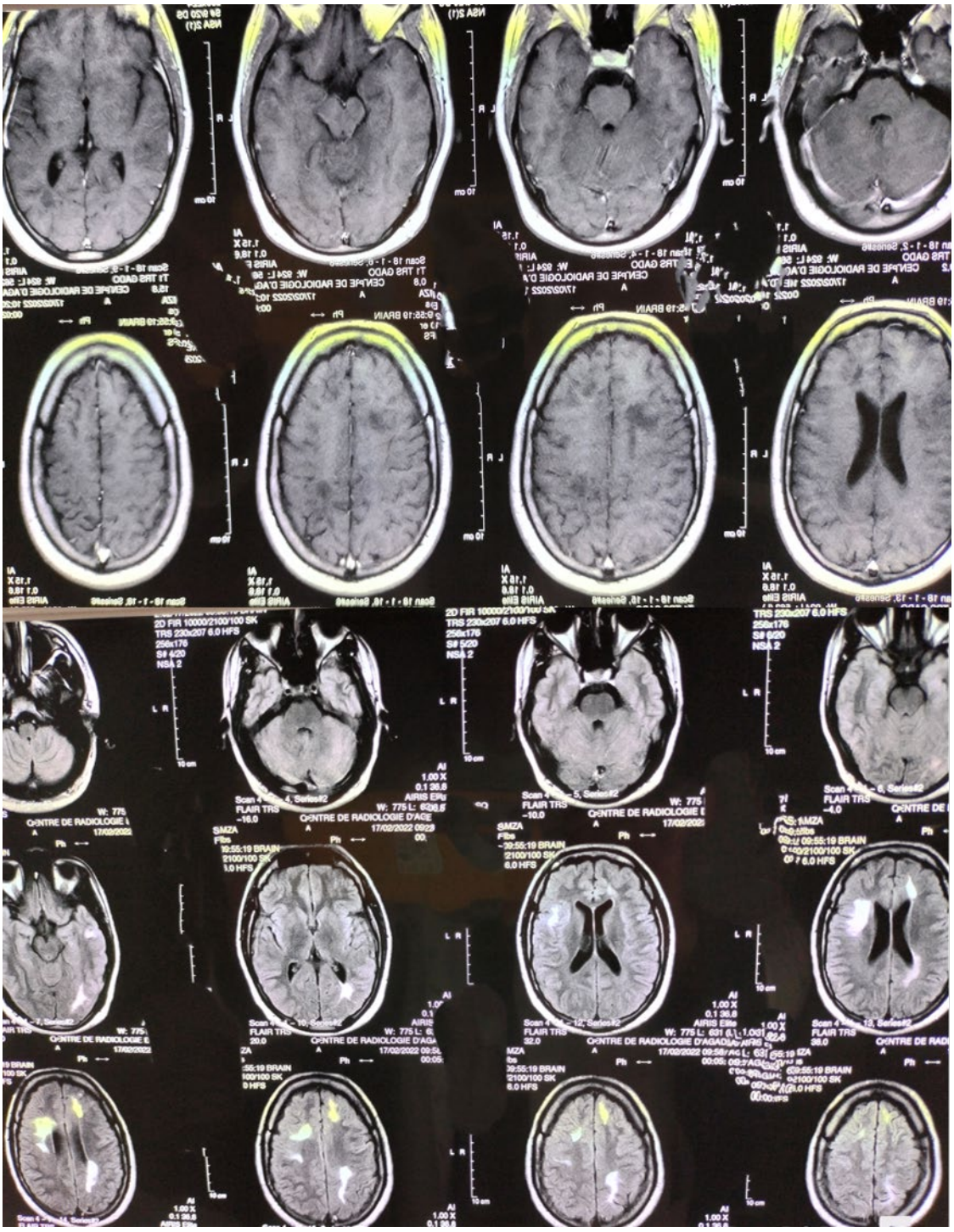


Figure 6: Brain MRI of a 21-year-old MOGAD patient, showing cortical and subcortical T1 hypointensity associated with T2/FLAIR hyperintensity, with no contrast enhancement. The optic nerves are unaffected.

VIII. CSF Testing:

Cerebrospinal fluid (CSF) analysis in all patients showed no abnormalities, except in one patient with concomitant MOGAD and intracranial hypertension. This patient demonstrated an opening pressure of 27 cmH_2O, mildly elevated protein levels (61 mg/dL), normal glucose levels (19 mg/dL), normal cellularity, and sterile cultures. Serum and CSF immunoelectrophoresis revealed no oligoclonal bands

IX. Immunology Analysis:

Anti-MOG and anti-AQP4 testing were performed in all patients usin cell bases assays. Anti-AQP4 was negative in all patients; Anti-MOG was positive in 6 patients and negative in 6 patients.

X. Additional Laboratory findings:

Routine blood tests, including inflammatory markers and metabolic panels, revealed no abnormalities in all patients.

XI. Clinical forms of MOGAD:

Among the 12 patients in this study, the most common clinical presentation was optic neuritis (n=7; 58.3%), followed by myelitis (n=3; 25%), cortical-subcortical involvement (n=1; 8.3%), and brainstem-cerebellar syndrome (n=1; 8.3%).

Table 2: Anti-MOG testing results' distribution for each clinical presentation.

Clinical forms	Anti-MOG Positive	Anti-MOG Negative
ON	3	4
Myelitis	1	1
MOGAD-associated encephalopathy	-	1
MOGAD-associated encephalopathy	1	-

XII. treatment and evolution:

- **Acute phase treatment:**

All patients received intravenous methylprednisolone (IVMP) for five days. One patient with severe myelitis required plasmapheresis (five sessions, every other day).

- **Maintenance Therapy:**

Immunosuppressive therapy was initiated in 6 cases, consisting of rituximab for 5 patients and Azathioprine for 2 patients; Azathioprine was substituted with Rituximab in one patient after showing signs of side effects.

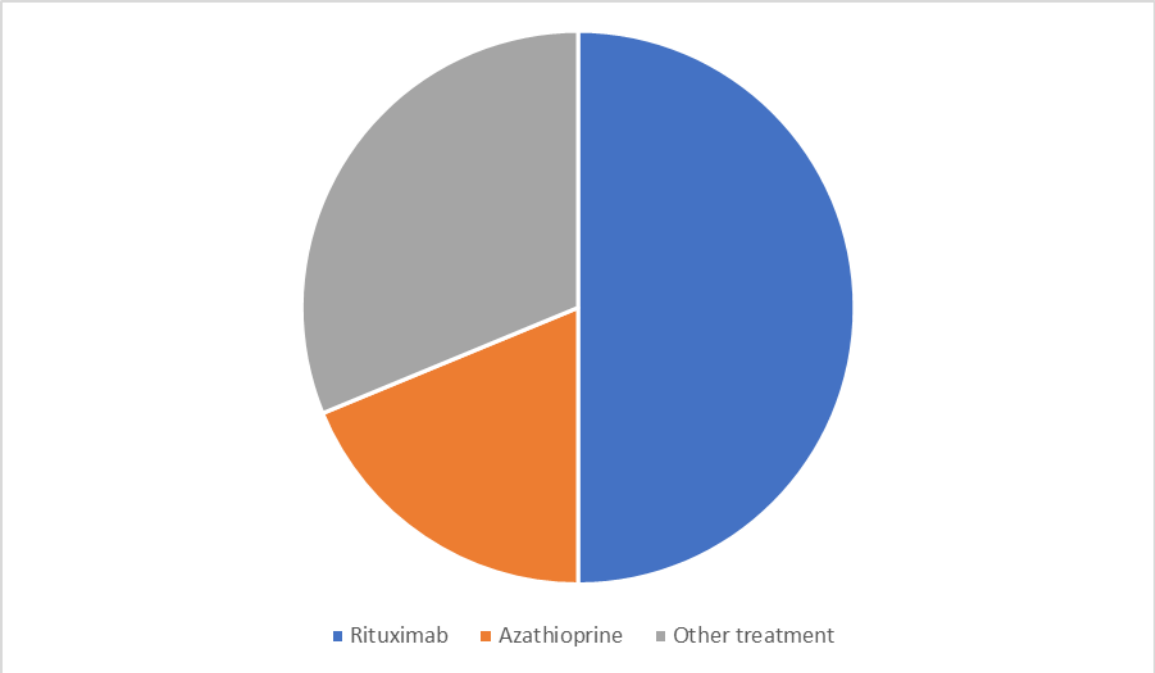


Figure 7: Distribution of treatment received by patients in our study

Table 3: Distribution of monophasic episodes and relapsing episodes for each clinical presentation

Clinical form	Monophasic	Relapsing
Optic Neuritis (ON)	5	2
Myelitis	2	0
Brainstem and cerebellar syndrome	1	0
MOGAD-associated Encephalopathy	1	0

SUMMARY TABLE OF MOGAD CASES OF OUR STUDY

	Age/Sex	Medical History	Clinical History / Onset Episode	Clinical Examination	Cerebral MRI	Spinal Cord MRI	CSF Testing	Serology (Anti-MOG / Anti-AQP4)	Biology	Treatment	Follow-up 1	Follow-up : Ophthalmic exam	Follow-up 2	Follow-up 3	Follow-up 4	Other Tests (VEP)
1	17 years old Male	No significant history	motor deficit 5 days before admission associated with acute urinary retention and constipation	-Difficulty standing (difficult upright position). -Difficult gait. -Does not hold mangazzini position in lower limbs. -Muscle strenght : lower limbs : 3/5 ; Upper limbs : 5/5 -Lower limbs Tonus : spastic hypotonia ; upper limbs : conserved -Tendon reflexes : upper limbs : brisk non diffuse monokinetic reflex ; lower limbs : diffuse brisk polykinetic reflex -Bilateral babinski sign Sensitivity :upper limbs dysesthesia seems conserved In total : spastic para pyramidal syndrome	T2 Hyperintense lesion of the spinal cord in cervical and thoracic levels (C6-D3) associated with spinal cord swelling at the corresponding site.	longitudinal T2 Hyperintense lesion regarding the dorsal portion of the spinal cord	Normal	Anti-MOG+ Anti-AQP4-	Normal	IV steroids (Methylprednisolone) 1g/day for 5 days.						
2	32 yeas rold Male	Oral aphtosis (4 episodes a year)	Sudden decrease in visual acuity 15 days prior to admission	Decreased visual acuity in the right eye (VA of the Right Eye: 1/10) neurological examination: normal. Pupillary light reflex: absent consensual pupillary light	Flair Hyperintense lesion with Globular and thickened morphology of the right optic nerve in it's intra-orbital segment, with a maximum diameter of 7mm, moderate diffusion-weighted hyperintensity.	Normal	Normal	Anti-MOG+ Anti-AQP4-	Normal	IV steroids (Methylprednisolone 1g/day during 5 days.)						

				reflex: present Grade 1 papillary Edema. clinical	Additional to a Homogeneous enhacement after godolinium injection. No brain lesion identified.											
3	10 years old Female	No significant history	Sudden decreased visual acuity in left eye 3 days before admission. tryearsient and episodic lower limb weakness	Sudden decrease in visual acuity of the left eye, blurry vision painful movement of the eyes with no other associated signs. Ophtalmic exam : Right eye: counts fingers , Left eye : 10/10. Fundoscopic exam : Grade 2 Papillary Edema	Intense and diffuse contrast enhancement in the left optic nerve, measuring s 6 mm in diameter with indistinct margins, respecting the optic chiasm, and ocular globe. Additionaly to a nodular temporoparietal lesion.	Normal	Normal	Anti-MOG+ Anti-AQO4-	Normal	IV Steroids (750 mg of Methylprednisolonel/day during 7 days)	Complete recovery	Left Eye : 10/10 ; Right Eye : 10/10 ; Papilledema : none				
4	48 years old Female	No significant history	Bilateral decreased visual acuity 15 days before admission. Treated before admission with Methyl prednisone	Bilateral decreased visual acuity 15 days before admission; Right Eye: 7/10; Left Eye: 6/10; no Papillary Edema. Day 3 after admission, Dizziness and vomiting, headache and blurry vision followed by conscience troubles and 7th nerve paralysis	Bilateral T2 hyperintensity of the optic nerves, accompanied by hyperintensity in the posterior protuberance.	Initial MRI normal	White cells count : 210 lymphocytes . Proteine: 1,8 g/L. Glucorachia : 0.82	Anti MOG - Anti AQP4 -	Normal	1g of Methylprednisolone daily for 5 days. Acute attack treatment : Rituximab 1g	very good					
5	27 years old Male	4 episodes of binocular diplopia resolutive in 3 days. Dysarthria and walking problem		Cerebellar Syndrome (ataxia, negative Romberg, hypermetria finger-nose, dysarthria). Horizontal	Diffuse subtentorial hyperintensity located in the peri-aqueductal region, pons, and cerebellum.	Normal	Proteins : 0,57 g/L . Glucose : 0, 66 g/L	Anti-MOG- Anti AQP4 -	Normal	Azathioprine 150 mg/ day	Very good					

				nystagmus												
6	26 years old Male	The patient's medical history dates back to the age of 10 years – 2006 right optic neuritis with temporary visual impairment resolved with corticosteroids – 2011 right hemibody weakness, fully resolved – 2020 diplopia, resolved over seven months – 2021 bi-temporal visual field loss.		Tetra pyramidal irritation. Bilateral Hemianopsy. VA 9/10 right eye; 10/10 left eye. Fundoscopy : normal	Cortical and subcortical T1 hypointensity associated with T2/FLAIR hyperintensity, without contrast enhancement. The optic nerves are unaffected.	Normal	Normal	Anti MOG positive Anti AQP4 négative	Normal	Rituximab 1 g at day 1 and day 15 then every 6 months	Good					
7	19 years old male	No significant history	Rapid onset of symptoms. Decreasing in visual acuity in the left eye, Blurry vision and left eye pain	Severe decrease in left visual acuity; counts fingers at 2m. Grade two papillary Edema at fundoscopy. Negative left Marcus Gunn sign. The rest of the exam is normal	Diffuse T2 hyper intensity of the left optic nerve.	Normal	Normal	Anti MOG negative Anti AQP4 negative	Normal	IV steroids 1g/day for 5 days.	Good evolution. Visual acuity back to 9/10		2 months after being discharged, the patient presented with paresthesia in the 4 limbs. Clinical exam: brisk deep tendon reflexes, diffuse and polykinetic. Follow-up MRI; Longitudinally extensive Myelitis (longitudinal cervical Hyper intensity the length of 3 vertebraes.) Treatment:	4 months later, 2nd relapse episode, this time marqued with dizziness and balance issues. Rapidily evolving. Clinical exam: Nystagmus. Diffuse polykinetic reflexes in the 4 limbs. MRI: T2 hyperintensities, round, located in the medium left cerebellum peduncle.	3rd relapse episodes, Grade 1 optic neuritis with right visual acuity 6/10. 5 days of steroids, full recovery. Rituximab was then prescribed.	

													Steroids 5 days, paresthesia resolved. Additional treatment : Azathioprine			
8	31 years old female	No significant history	11 days before admission, the patient experienced a rapidly progressive decrease in visual acuity in the right eye follow 2 days later by a decrease in visual acuity of the left eye. With eye pain especially during ocular mobilisation. Associated 3 days later with a frontal headache ,vomiting and dyschromatopsia without phonophotophobia	The clinical examination revealed optic neuropathy characterized by severely reduced visual acuity (counting fingers at 1 foot in both eyes), bilateral papilledema, dyschromatopsia, and limited abduction of the left eye, with no observed visual field abnormalities.	Brain MRI with angiographic sequence showed evidence of Idiopathic Intracranial Hypertension associated with a slight enhancement in the left optic nerve. A : T2 prominent subarachnoid space around the optic nerve. B: Enhancement in the left optic nerve . C: Tryearsverse sinus Hypoplasia	Normal	The CSF study showed an opining pressure of 27 cmh2o, mildly elevated p protein (61 mg/dl), normal glucose (19mg/dl) , with a normal cellularity and sterile culture. Serum and CSF proteins' immunoelectropherosis detected No oligoclonal bands.	Mog positive AQP4 negative	Normal	Acétazolamide 1.5 grams associated with Potassium supplementation and 1g of methylprednisolone during 5 days.	Patient discharged with oral corticosteroids and acetazolamide. Follow-up : improvement in the headache and the visual acuity .	resolution of the optic disc Edema, color vision and normal visual acuity.				Visual evoked potential revealed a conduction delay in both eyes.
9	43 years old male	No significant history	Lower limbs paresthesia with ataxia	Medullar syndrome brisk reflexes in 4 limbs associated with para-paresia , with deep sensitivity	Diffuse T2 hyperintensity observed throughout the cervical spinal cord from levels C2 to C7.	Normal	Normal	Anti-MOG negative Anti-AQP4 negative	Normal	Treated with Steroids + Plasmapheresis + Rituximab	Full recovery at follow up					

				abnormalities												
10	45 years old male (isolated myelitis)	No significant history	Paresthesies membre inferieur , faiblesse , ,ataxia , troubles de sensibilité profonde	paraparesia 4/5 , tbles de sensibilité profonde	diffuse Hyper intensity cervical longitudinaly C2-C5	Normal		Anti-MOG negative Anti-AQP4 negative		steroids no improvement	follow up rituximab	Full recovery				
11	48 years old female	No significant history	installation a 7 jours avec baisse de l'acuité visuelle aigue et douleurs oculaire gauche,	Visual acuity: 5/10 in the left eye, 10/10 in the right eye. Stage 2 papilledema in the left eye. Positive Marcus Gunn sign. Visual field in the left eye shows a significant defect with scotomas.	Hyperintensity of the left optic nerve with contrast enhancement. Papilledema: left axonal neuropathy.	Normal		Anti-MOG negative Anti-AQP4 negative	nor mal	steroids 1g	Good recovery , AV : 10/10. Regression of left eye papilledema from stage 2 to stage 1. Complete recovery of the visual field.	2 months later: similar episode, LVA: 6/10 with stage 2 papilledem a.		Treatment adjustments: Azathioprine was substituted with Rituximab after showing signs of side effects.		
12	26 years old male	No significant history	Headache and eye pain with decreased visual acuity of the right eye	The clinical examination revealed optic neuropathy characterized by severely reduced visual acuity (counting fingers at 1 foot in both eyes), bilateral papilledema, dyschromatopsia, and limited abduction of the left eye, with no observed visual field abnormalities.	Right optic nerve hyperintensity with contrast enhacement. Papilledema			Anti-MOG negative Anti-AQP4 negative	Nor mal		Monophasic MOGAD, IV steroids 5 days followed by oral steroids.	full recovery				

DISCUSSION

I. History of MOG and MOG-antibody:

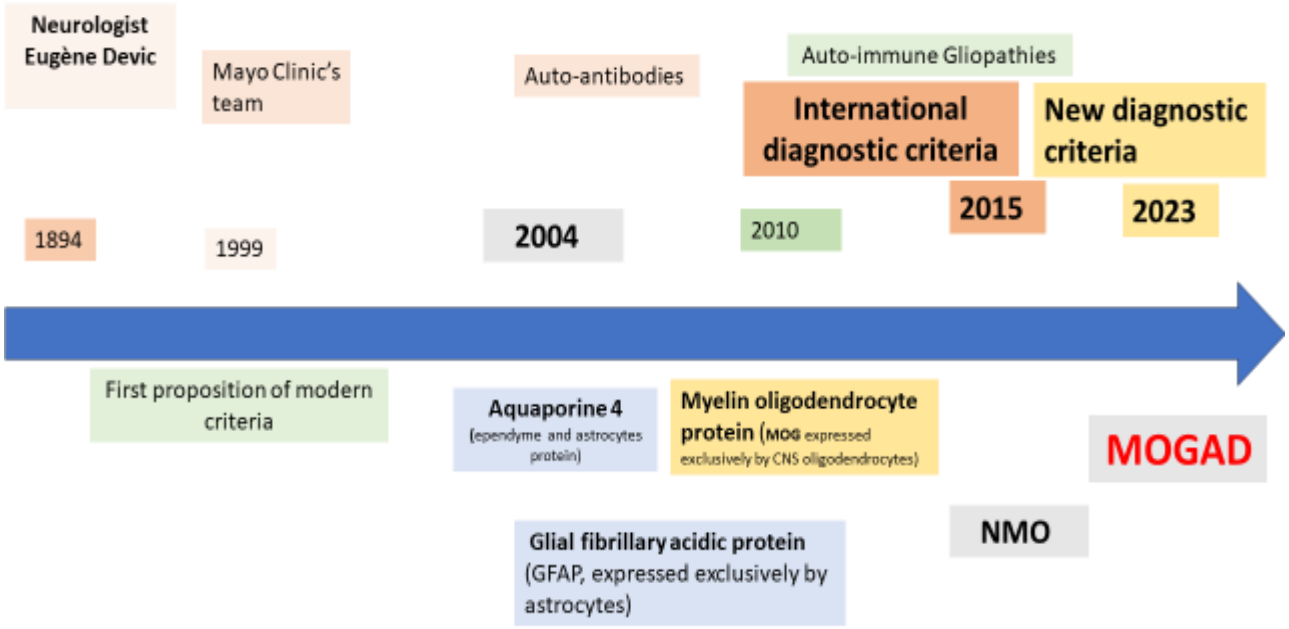


Figure 8: Chronological events leading to the establishment of MOGAD as a distinct neurological entity.

In the 1980s, MOG protein was identified as a key potential target for autoantibody in CNS myelin in experimental autoimmune encephalomyelitis models. In 2003, based on Western Blot detection, MOG-IgM was proposed as a biomarker to predict the transition from clinically isolated syndromes to multiple sclerosis (MS). However, further studies using similar assays such as ELISA, demonstrated that Anti-MOG lacked specificity to a disease, as it was found with similar frequencies in patients with MS, other demyelinating CNS conditions, and healthy controls. Afterward, a pivotal 2007 study conducted by O'Connor et al. revealed that assays detecting MOG in its three-dimensional conformation, rather than linear epitopes on denatured proteins, identified Anti-MOG recognizing conformational epitopes in patients with acute disseminated encephalomyelitis (ADEM) and optic neuritis. In this context, "tridimensional conformational form" emphasizes that the assay detects MOG protein in its natural 3D folded state, rather than in a denatured or linear (unfolded) form. This distinction is important because

antibodies often recognize proteins based on their specific 3D structure, and detecting the native form can provide more accurate diagnostic results [9,10].

Later research using cell-based assays expressing full-length human MOG on mammalian cells confirmed Anti-MOG in patients with non-MS demyelinating CNS disorders, including 30-70% of seronegative NMOSD cases. Anti-MOG -associated clinical and MRI features extend beyond the NMOSD phenotype, leading to the classification of MOGAD as a distinct entity [9].

II. Epidemiology:

The worldwide incidence of MOGAD is approximately 1.6–4.8 per million people, with a prevalence of 1.3–2.5 per 100,000 people [5–7]. MOGAD's incidence is known for a biphasic behavior, the first peak in children (5–10 years) and the second one in adults (20–30 years). So far, no high-risk ethnicities or sex preferences have been identified in MOGAD [11–13].

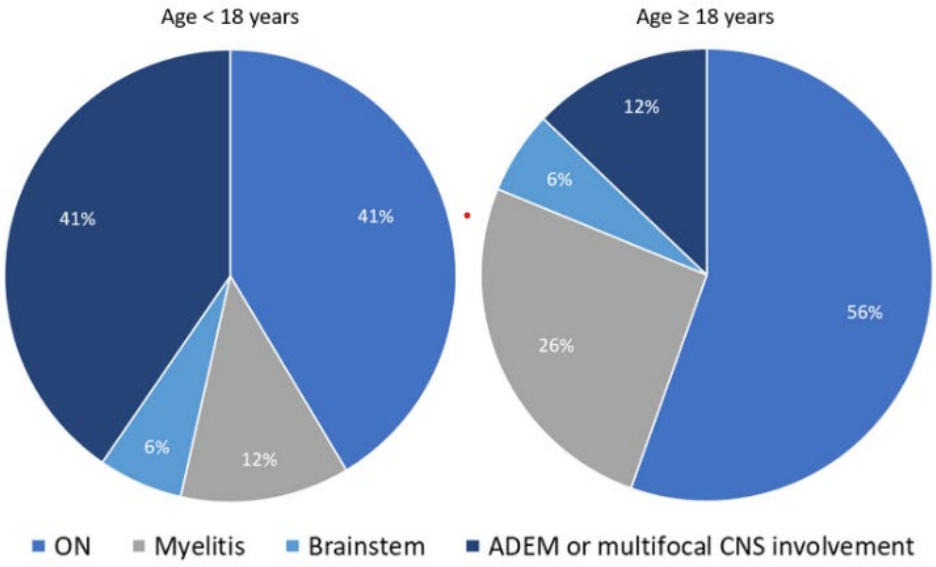


Figure 9: Distribution of disease phenotypes of MOGAD by age: The most common presentation is ON (optic neuritis, unilateral or bilateral), followed by myelitis, brainstem syndromes, ADEM), and multifocal central nervous system involvement without encephalopathy [14].

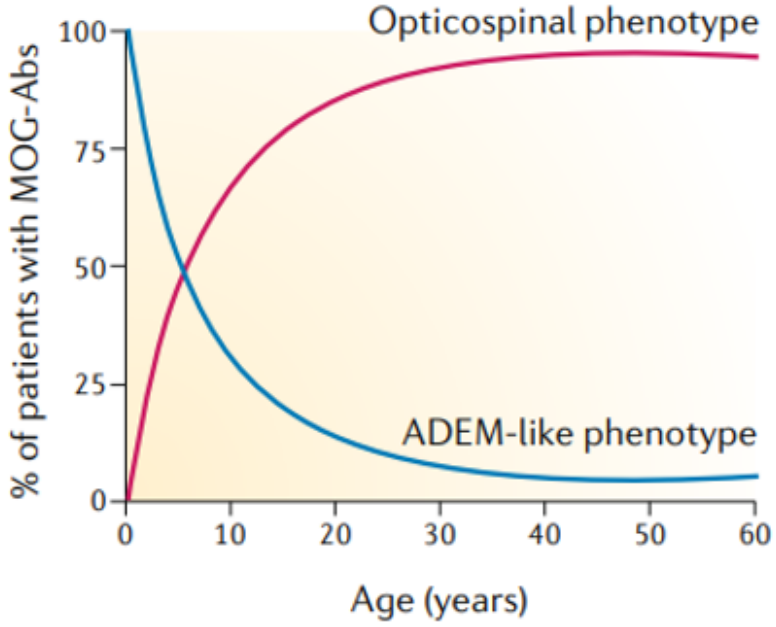


Figure 10: Graph illustrating the correlation between age and two distinct neurological phenotypes in MOGAD: the ADEM-like phenotype and the optico-spinal phenotype. The incidence of the ADEM-like phenotype significantly declines after the age of 10, while the optico-spinal phenotype peaks around the age of 40.

III. Physiopathology:

The role of MOG-IgG in MOGAD pathophysiology is not fully elucidated to this day. However, located at the outermost layer of myelin, this protein is exposed to MOG antibodies. It is worth mentioning that MOG protein represents approximately 0.05% of the total proteins in the CNS [14,15].

The current hypothesis suggests that MOG-IgG binds to myelin's oligodendrocytes glycoproteins triggering the production of IL-6 and B-cell activating factor (BAFF), with the recruitment of CD4+ T cells and macrophages, leading to oligodendrocytes destruction and neuronal damage [16].

In preclinical models, when C57BL/6 mice were immunized with the MOG peptide, they developed progressive paralysis, referred to as EAE, where MOG-specific CD4+ T cells were key contributors [17]. Similarly, LEW.1AV1 rats immunized with MOG develop EAE, with optic nerve and spinal cord involvement resembling phenotypes found in NMOSD (Sakuma et al., 2004) [10].

MOG autoantibodies have been found to activate the complement cascade. Moreover, MOG autoantibodies have been found to activate the complement cascade (Mader et al., 2011) [18]. These studies suggest that MOG is a potential antigen in human demyelinating diseases. And clinical evidence supports the pathogenic nature of this autoantibody [10].

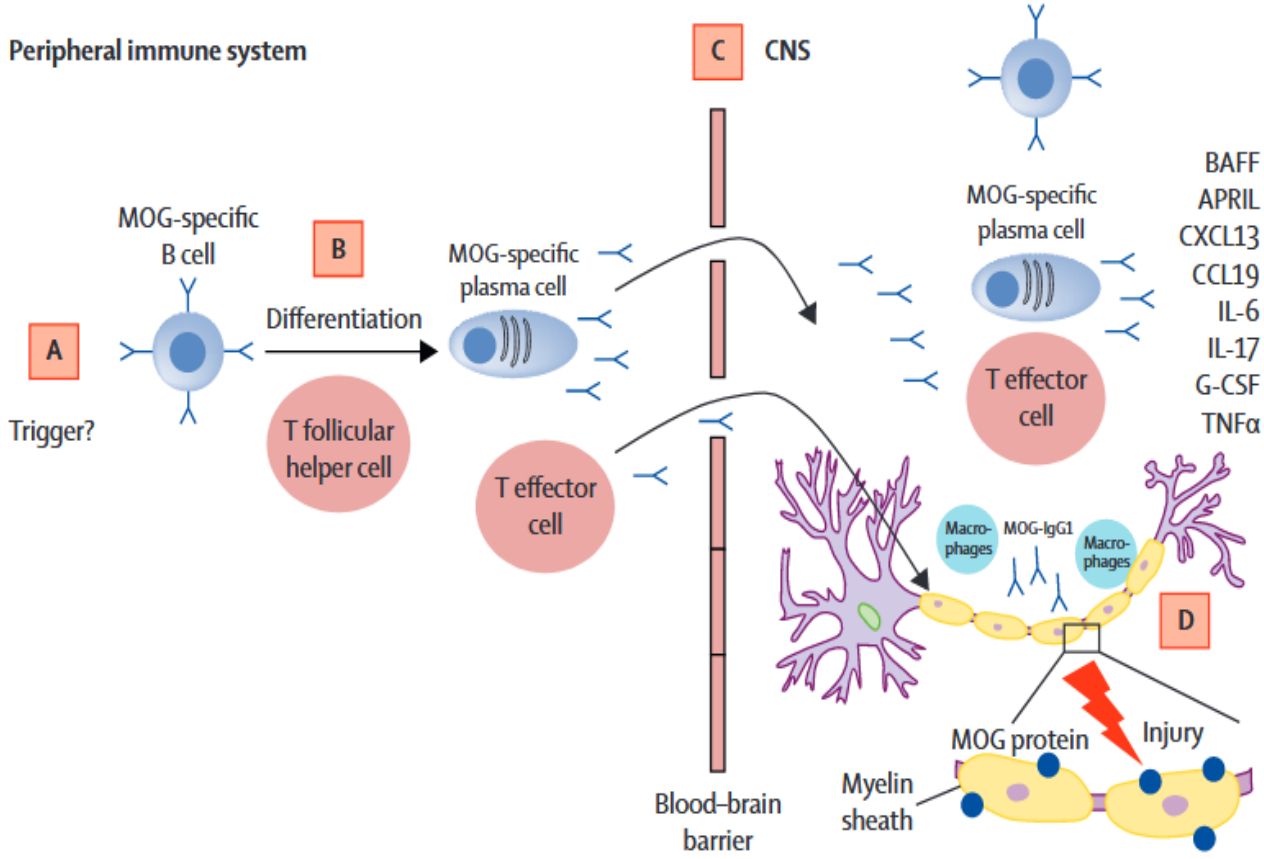


Figure 11: Physiopathology of Oligodendrocytes' myelin sheath damage in MOGAD, by Cabo et al. [19].

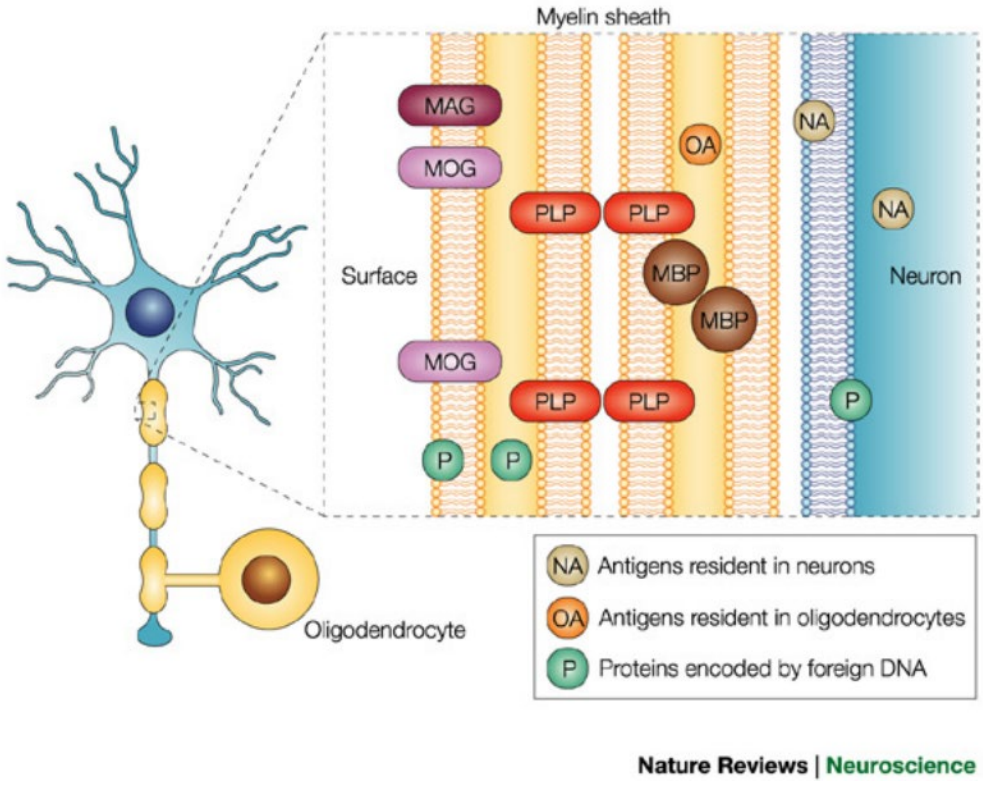


Figure 12: structure of the axon and myelin sheath.

In Kohyama and al.'s study, two in vitro assays were performed to determine the cytotoxicity and viability of MOG-expressing cells when exposed to MOG-IgG. The first assay aims to quantify the proportion of damaged or dead cells (PI-positive cells) after exposure to MOG autoantibodies. When MOG-GFP cells were incubated in anti-MOG-positive sera (rabbit and human) in the presence of complement; Flow cytometry showed significant cell damage (PI-positive) in MOG-GFP cells compared to empty cells. The second assay aims to observe cell viability after exposure to anti-MOG-positive sera, showing that MOG-GFP cells significantly decreased, whereas empty cells were intact. These findings confirm that anti-MOG-positive sera are cytotoxic to MOG-GFP cells in the presence of complement, with no significant differences in cytotoxicity among various disease groups [10].

IV. Clinical manifestations:

In adults with MOGAD, Optic Neuritis is the most common clinical presentation either at the onset or during the disease course (up to 80%) [16]. It can be isolated or associated with other clinical presentations such as ADEM and Myelitis [11]. It is often associated with vision loss, dyschromatopsia, and eye pain worsened by eye movement [11]. Additional features such as optic perineuritis, bilateral involvement, eye pain (mistaken often for headache) preceding visual loss, and optic disc Edema at fundoscopy, which is often moderate to severe and sometimes associated with optic hemorrhages are all signs suggesting the diagnosis of MOGAD [12,14,20]. At nadir Visual loss is usually severe to moderate with a median visual acuity between counting fingers and moving hands [21,22]. Recovery is usually complete or almost complete in MOG-ON, and residual permanent blindness in at least one eye is rare (6-12%) [11,21,22].

Myelitis is the second most common presentation of MOGAD (26%). It can be isolated or associated with ADEM or optic neuritis [23]. The symptoms suggest a spinal cord lesion and manifest as acute or subacute sensory, motor, and autonomic disturbances. Additional signs include tetraparesis, paraparesis, or paralysis, Lhermitte's sign, bladder urgency or retention, and sexual dysfunction. Similar to optic neuritis, MOGAD attacks tend to be moderate to severe at the nadir, with a median Expanded Disability Status Scale (EDSS) score of 5.5. Over 30% of patients are wheelchair dependent at nadir. However, acute flaccid weakness accompanied by areflexia may suggest the diagnosis of MOGAD, indicating potential anterior gray matter involvement. In the long term, only 6% to 7% of MOGAD patients, compared to 37% to 44% of those with AQP4-positive NMOSD, will require a gait aid. Persistent sphincter dysfunction affects more than 50% of MOGAD patients with a history of myelitis, and many need ongoing intermittent catheterization. A pattern that may mimic acute flaccid myelitis seen after enterovirus infections [11].

The third clinical manifestation of MOGAD is brainstem and cerebellar syndromes. Signs of infratentorial lesions can be observed in MOGAD as well as NMOSD. In NMOSD, infratentorial involvement is manifested with Area Postrema syndrome accounting for up to 60% of brainstem/cerebellar NMOSD patients, and sometimes representing an extension of cervical

spinal cord lesion [8,24]. The typical presentation includes hiccups and vomiting. In MOGAD, on the other hand, brainstem and cerebellar phenotypes are typically exhibited by diplopia (26%) and ataxia (45%). It usually occurs in the context of polyfocal cerebral syndromes or ADEM [25]. However, episodes of isolated trigeminal neuralgia, facial numbness, and diplopia are much more common in MS [11].

Regarding cerebral manifestation, ADEM is defined by polyfocal CNS symptoms, unexplained encephalopathy, and large lesions in the grey and white matter visible on MRI. In case of severe encephalopathy, Mechanical ventilation could be necessary [11,26]. Additionally, ADEM is the most common presentation in pediatric patients younger than 12 years (20%–60%) [8,16]. Although the acute phase in ADEM is potentially severe, the prognosis is generally good [11].

Finally, cerebral cortical encephalitis has recently been described in MOGAD, accounting for 7% of MOGAD patients, among which the majority are adult patients. Clinical signs consist of headache (79%), seizures (68%), encephalopathy (63%), and fever (42%). Typical MRI findings are T2-flair cortical hyperintensity with cortical and leptomeningeal enhancement after contrast injection. These lesions usually resolve after an acute attack [11,27,28].

	Optic neuritis	Transverse myelitis	Acute disseminated encephalomyelitis
Clinical features	Up to 80% of patients, either at onset or during the disease course; simultaneous bilateral involvement in up to 40%; average high contrast visual acuity at nadir counting figures; optic nerve head swelling (papillitis); might have peripapillary haemorrhage; more steroid responsive than in AQP4-NMOSD and multiple sclerosis	Spinal cord involvement in 30% of episodes at onset and up to 50% during the disease course; motor disability might be similar to AQP4-NMOSD; urinary, bowel, and erectile dysfunction are common; more steroid responsive than AQP4-NMOSD and multiple sclerosis	Most frequent presentation in children (<18 years); only in about 5% of adult presentations; seizures at onset observed in up to 40% of children with acute disseminated encephalomyelitis; MOG antibody associated ADEM at higher risk of higher risk of post-acute disseminated encephalomyelitis epilepsy
Imaging	Extensive T2-weighted and gadolinium enhancing lesion in the optic nerve or chiasm, more evident on orbit MRI; predominates in the anterior parts of nerve but might extend to optic chiasm; perineural gadolinium enhancement; peripapillary retinal nerve fibre layer thinning frequent on OCT but clinical-radiological paradox (despite severe atrophy of retinal nerve fibre layer, visual acuity is preserved); attack related retinal nerve fibre layer thinning with temporal predominance; microcystic macular in 24%	Initially described as longitudinally extensive transverse myelitis but short myelitis in up to 40%; involvement of the conus medullaris (more frequent than in MS and AQP4NMOSD); abnormalities confined to grey matter (sagittal line and axial H sign) and nerve roots; less frequent gadolinium enhancement than AQP4-NMOSD and multiple sclerosis; initial spinal cord MRI negative in 10% of patients; frequent complete resolution at follow-up scan	Large, hazy, and poorly demarcated asymmetrical bilateral lesions; deep grey matter involvement, most commonly affecting the thalamus; lesions might be highly enhancing; corpus callosum, brainstem and cerebellum involved; frequently associated to spinal cord involvement; frequent complete resolution at follow-up scan
CSF	Rare oligoclonal bands (<10%); presence of frequent mild lymphocytic pleocytosis	Rare oligoclonal bands (<10%); presence of frequent mild lymphocytic pleocytosis	Rare oligoclonal bands (<10%); presence of frequent mild lymphocytic pleocytosis
Risk of relapse and outcome	Patients aged <45 years at higher risk of relapse than older ones (>18 years); permanent visual impairment (visual acuity <20/100) rare at 2 years; reversible visual dysfunction from first episode in up to 75%; progressive thinning of peripapillary retinal nerve fibre layer (but not of the combined ganglion cell and inner plexiform layer) might be observed in absence of new clinical attacks	Good or full recovery from the onset attack in 60% younger patients (<18 years); around 20% of patients had permanent motor disability at 2 years (disability status scale >3·0); irreversible motor disability at last follow-up was explained by disability at onset attack in 68·4% patients who reached DSS 3.0 and 87·5% who reached disability status scale EDSS 6.0; permanent bowel, bladder, and erectile dysfunction are frequent despite good motor recovery	Up to 50% of children (<18 years) will relapse after acute disseminated encephalomyelitis; phenotype at relapse might be multiple disseminated encephalomyelitis, or acute disseminated encephalomyelitis-optic neuritis; a small proportion of children (<18 years) will have a single relapse within 3 months; behavioural and cognitive problems might occur after acute disseminated encephalomyelitis and are more common in relapsing group (up to 50%); up to 10% (predominantly very young children [younger than 7 years]) can develop a leukodystrophy-like phenotype with large confluent highly enhancing lesions and significant brain atrophy over time

Other, less common, phenotypes have also been reported in patienst with MOG-Ab; (1) isolated brainstem involvement in about 7% of adults and 30% of children (younger than 18 years; postrema syndrome is rare); (2) cortical (unilateral or bilateral) encephalitis with or without white matter involvement; (3) cranial neuropathies or mixed central and peripheral syndromes; (4) features of chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids; (5) pseudotumour cerebri-like, associating bilateral papillitis to elevated CSF opening pressure. For references and information, please see appendix (pp 2–5). MOG=myelin-oligodendrocyte glycoprotein. AQP4-NMOSD=anti-aquaporin-4 antibody-associated neuromyelitis optica spectrum disorder. OCT=optical coherence tomography. EDSS=expanded disability status scale. ADEM=acute disseminated encephalomyelitis

Figure 13: Summary table of clinical phenotypes in MOGAD, by Marignier and al. [16].

V. Diagnostic criteria:

A 2023 expert panel published a consensus paper on MOGAD's diagnostic criteria. The criteria require at least one major clinical demyelinating episode from the following: optic neuritis, myelitis, ADEM, cerebral monofocal or polyfocal deficits, brainstem or cerebellar deficits, and cerebral cortical encephalitis, usually with seizures. The second criterion is MOG-IgG antibody testing, performed using cell-based assay serum tests. If the test is clearly positive, no additional supporting criteria are needed. However, if it is low positive, lacks a reported titer, or is negative in serum but positive in CSF, additional criteria must be met: AQP4 antibody seronegative and at least one supporting clinical or MRI feature. CSF MOG testing is not routinely recommended. However, if the clinical phenotype strongly suggests MOGAD but serum MOG-IgG is negative, CSF testing should be considered. The third criterion is the exclusion of other CNS demyelinating diseases, including patients meeting McDonald's 2017 diagnostic criteria for MS. MRI features and clinical syndromes supporting MOGAD should be carefully considered alongside these criteria.

Typical MOGAD optic neuritis findings include bilateral optic nerve involvement with longitudinal demyelination affecting at least 50% of the nerve's length, perineural optic sheath enhancement, and optic disc edema. Similarly, myelitis in MOGAD is characterized by longitudinally extensive lesions, central cord involvement with the typical 'H' sign, and conus medullaris involvement. Finally, MOGAD-related brain, brainstem, and cerebral syndromes typically present on MRI with polyfocal hyperintense T2 lesions in supratentorial and often infratentorial white matter, deep gray matter involvement, poorly defined T2 hyperintensity in the pons, medulla, and middle cerebellar peduncles, as well as cortical lesions with or without meningeal enhancement (8).

<table>
<tr><th colspan="5">Diagnosis of MOGAD (requires fulfilment of A, B, and C)</th></tr>
<tr><td>(A) Core clinical demyelinating event</td><td colspan="4">Optic neuritis*
Myelitis†
ADEM‡
Cerebral monofocal or polyfocal deficits§
Brainstem or cerebellar deficits¶
Cerebral cortical encephalitis often with seizures||</td></tr>
<tr><td rowspan="4">(B) Positive MOG-IgG test</td><td rowspan="4">Cell-based assay: serum**</td><td>Clear positive††</td><td colspan="2">No additional supporting features required</td></tr>
<tr><td>Low positive‡‡</td><td colspan="2" rowspan="3">• AQP4-IgG seronegative AND
• ≥1 supporting clinical or MRI feature</td></tr>
<tr><td>Positive without reported titre</td></tr>
<tr><td>Negative but CSF positive§§</td></tr>
<tr><td rowspan="3">Supporting clinical or MRI features</td><td colspan="2">Optic neuritis</td><td colspan="2">• Bilateral simultaneous clinical involvement
• Longitudinal optic nerve involvement (> 50% length of the optic nerve)
• Perineural optic sheath enhancement
• Optic disc oedema</td></tr>
<tr><td colspan="2">Myelitis</td><td colspan="2">• Longitudinally extensive myelitis
• Central cord lesion or H-sign
• Conus lesion</td></tr>
<tr><td colspan="2">Brain, brainstem, or cerebral syndrome</td><td colspan="2">• Multiple ill-defined T2 hyperintense lesions in supratentorial and often infratentorial white matter
• Deep grey matter involvement
• Ill-defined T2-hyperintensity involving pons, middle cerebellar peduncle, or medulla
• Cortical lesion with or without lesional and overlying meningeal enhancement</td></tr>
<tr><td colspan="5">(C) Exclusion of better diagnoses including multiple sclerosis¶¶</td></tr>
</table>

Figure 14: Diagnostic criteria by Banwell et al. International Panel of Experts describing the consensus to establish the diagnosis of MOGAD [8].

VI. MRI features:

MOG-ON. Generally, MOGAD is characterized by severe acute attacks visible on MRI by extensive T-2 lesions affecting the brain, spine, and orbit. These lesions are usually described as "fluffy" due to undefined margins, contrary to MS in which lesions are well-circumscribed on MRI [13,29].

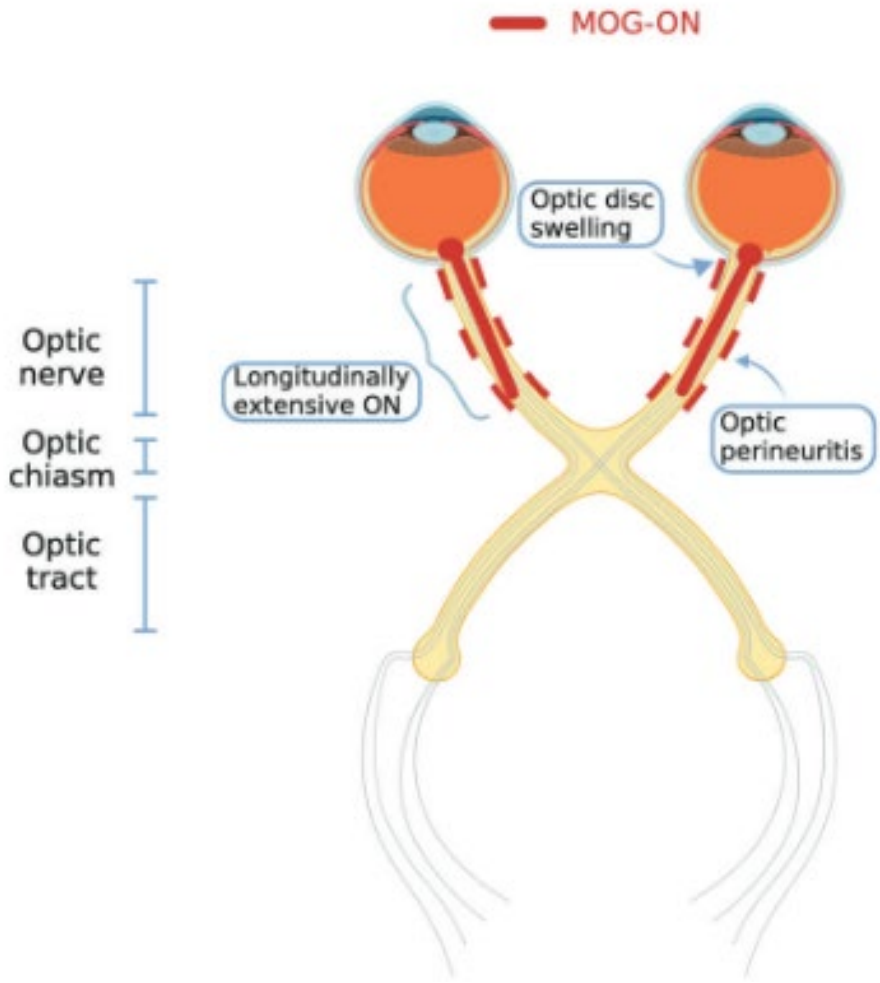


Figure 15: Localization of visual pathway lesions in MOG-ON : [20]

Bilateral longitudinally extensive ON, with optic disc swelling, involvement of the optic nerve sheath or perineuritis of the optic nerve, and the retrobulbar segment of the optic nerve.

Regarding ON, typical radiological features include bilateral longitudinal Optic nerve T-2 hyperintensity, usually affecting the anterior segment at onset (more than 50% of the pre-chiasmal segment) associated with peri neural optic sheath enhancement involving orbital fat. [8,20] To note that bilateral longitudinally extensive enhancement of the optic nerve not involving the chiasm is suggestive of MOGAD [30]. Optic disc edema is more reported on OCT and Fundoscopy but can also be visible on MRI [20].

Regarding myelitis, approximately 70% of MOGAD patients present with longitudinally extensive transverse myelitis (LETM) lesions affecting at least three vertebral segments on sagittal T2-weighted MRI (11). Some T2 lesions may be subtle and correlate with the severity of the clinical presentation. Multifocal spinal cord abnormalities can occur, and spinal cord necrosis is rare.

A valuable radiological clue is the presence of MRI abnormalities in the conus medullaris. Lesions in this spinal cord segment are significantly more common in MOGAD (26%) compared to other demyelinating disorders such as MS and NMOSD (23).

Abnormalities on axial T-2 images are often central involving grey and white matter. T-2 hyperintensity is limited to grey matter exhibiting a H-shaped image (H-sign). T-2 hyperintensity can be observed in the central canal and usually resolves in follow-up [11].

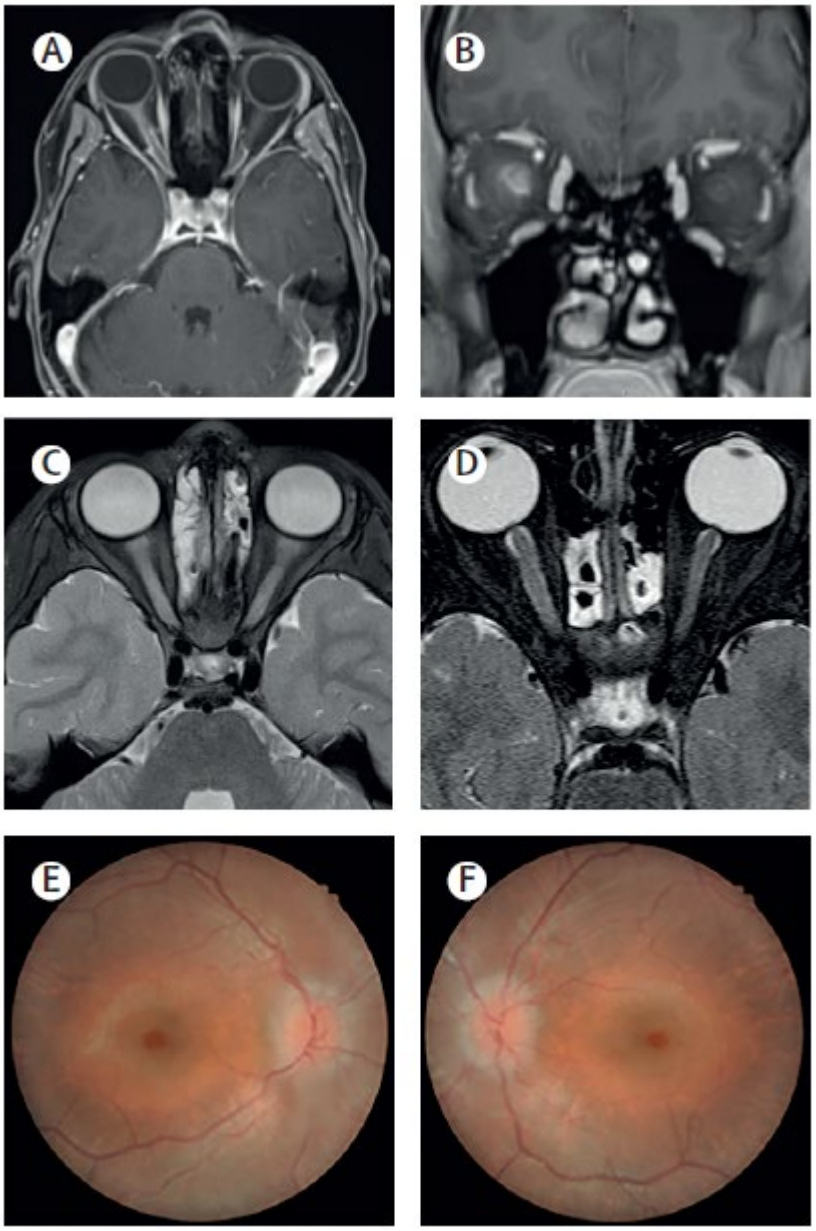


Figure 16: MRI and fundoscopic lesions in MOGAD: [8]

A: Peri neural optic sheath enhancement; B: Coronal view, right optic nerve swelling, and enhancement; C: Bilateral longitudinally extensive optic nerve T2 hyperintensity; D: Optic disc swelling; E: Optic disc Edema on fundoscopy; F: Optic disc edema on fundoscopy

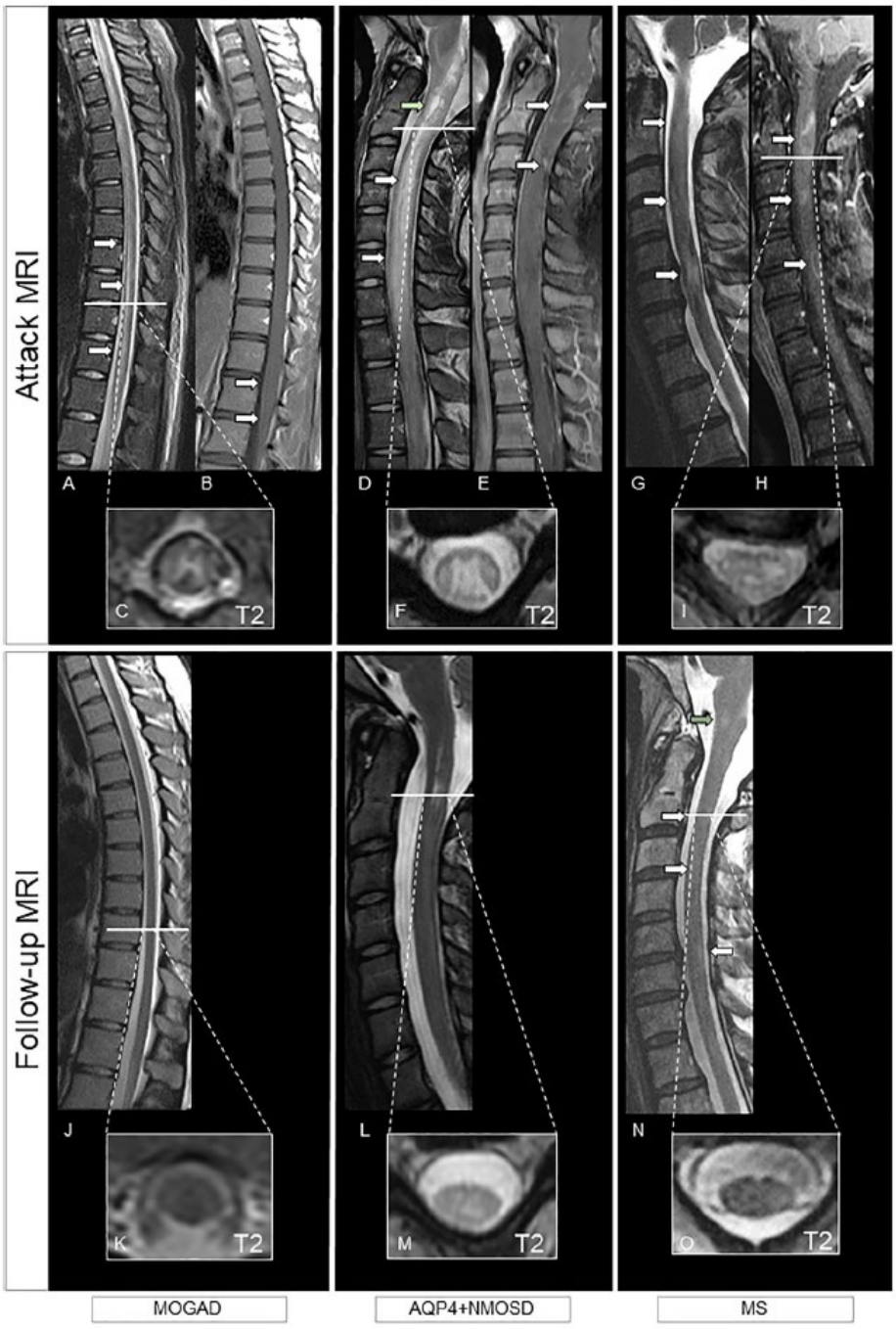


Figure 17: Comparison of spinal cord MRI findings in MOGAD, NMOSD, and MS [11].

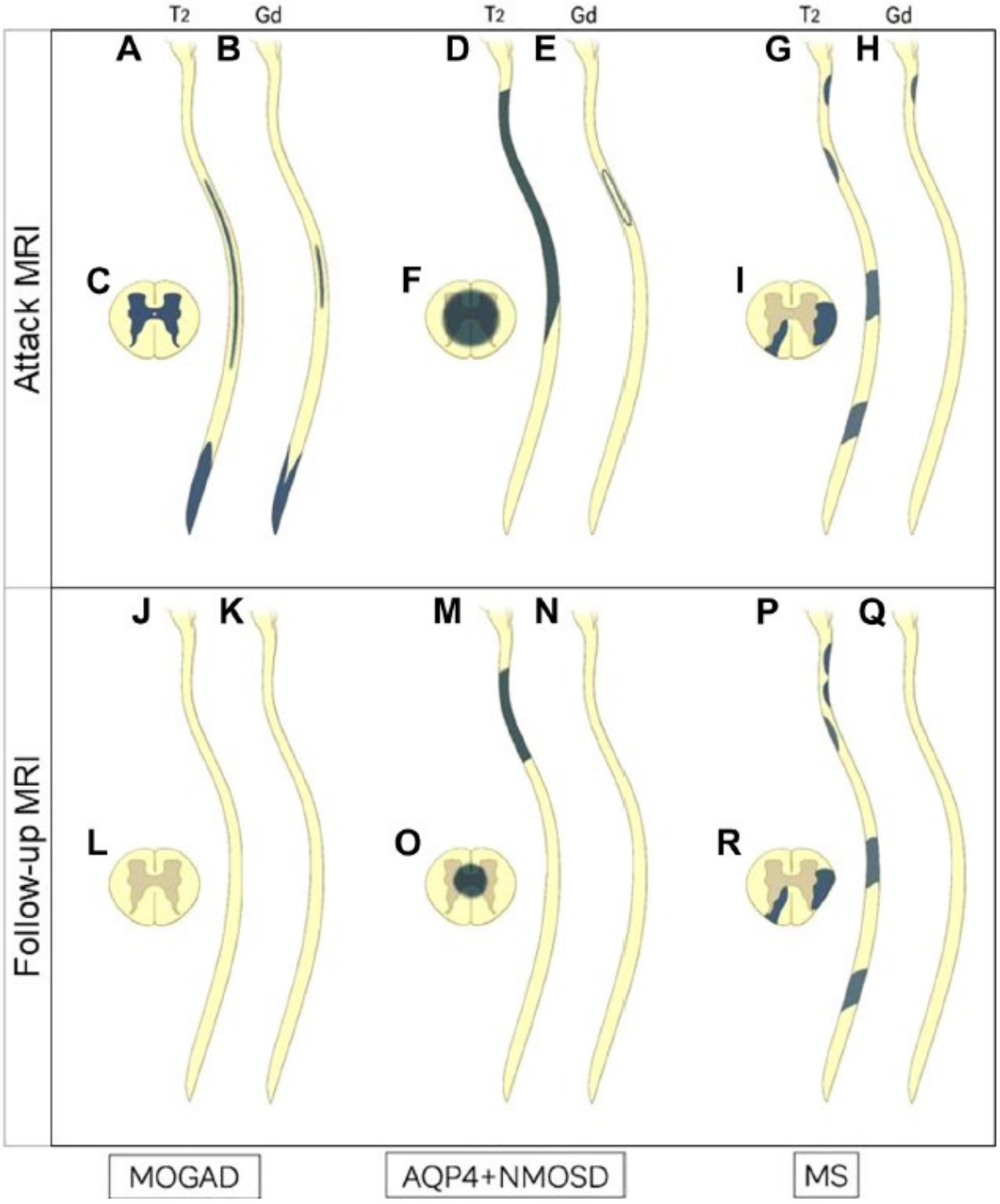
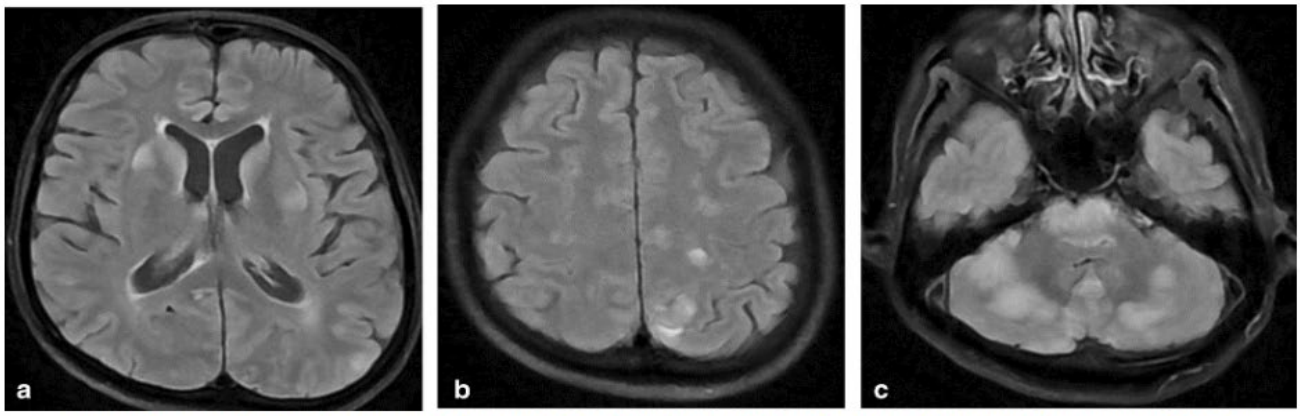


Figure 18: Illustration of typical findings in spinal cord MRI and evolution of lesions at follow-up in MOGAD, NMOSD, and MS. [11]

Brain lesions in MOGAD are found in 43% to 53% of patients, they include deep grey matter, brainstem lesions, cortical lesions, subcortical or juxtacortical lesions, brainstem lesions, cerebellum, large hemispheric lesions and Leukodystrophy like images.

The most characteristic MRI findings in MOGAD brain imaging are lesions of the deep grey matter and large abnormalities in cerebellar peduncles. Additionally, cortical lesions are present in MOGAD. Cerebral cortical encephalitis is visible on FLAIR sequences and involves large cortical areas [11,19,25].



The case illustrates MOGAD-related encephalitis in a 48-year-old male who presented with generalized tonic–clonic seizures. The brain lesions show typical characteristics of MOGAD—bilateral, asymmetrical, T2 hyperintense, and poorly demarcated with a fluffy appearance. **a** Axial T2 FLAIR MR image shows hyperintense signal in bilateral basal ganglia, cortex, and subcortical and deep white matter in parieto-temporal lobes. **b** Axial T2 FLAIR MR image shows hyperintense signal in bilateral cortex and subcortical and deep white matter in fronto-parietal lobes (more on left side). **c** Axial T2 FLAIR MR image shows hyperintense signal in pons and cerebellum

Figure 19: Brain MRI of a 48 year-old male with MOGAD-related encephalitis [11].

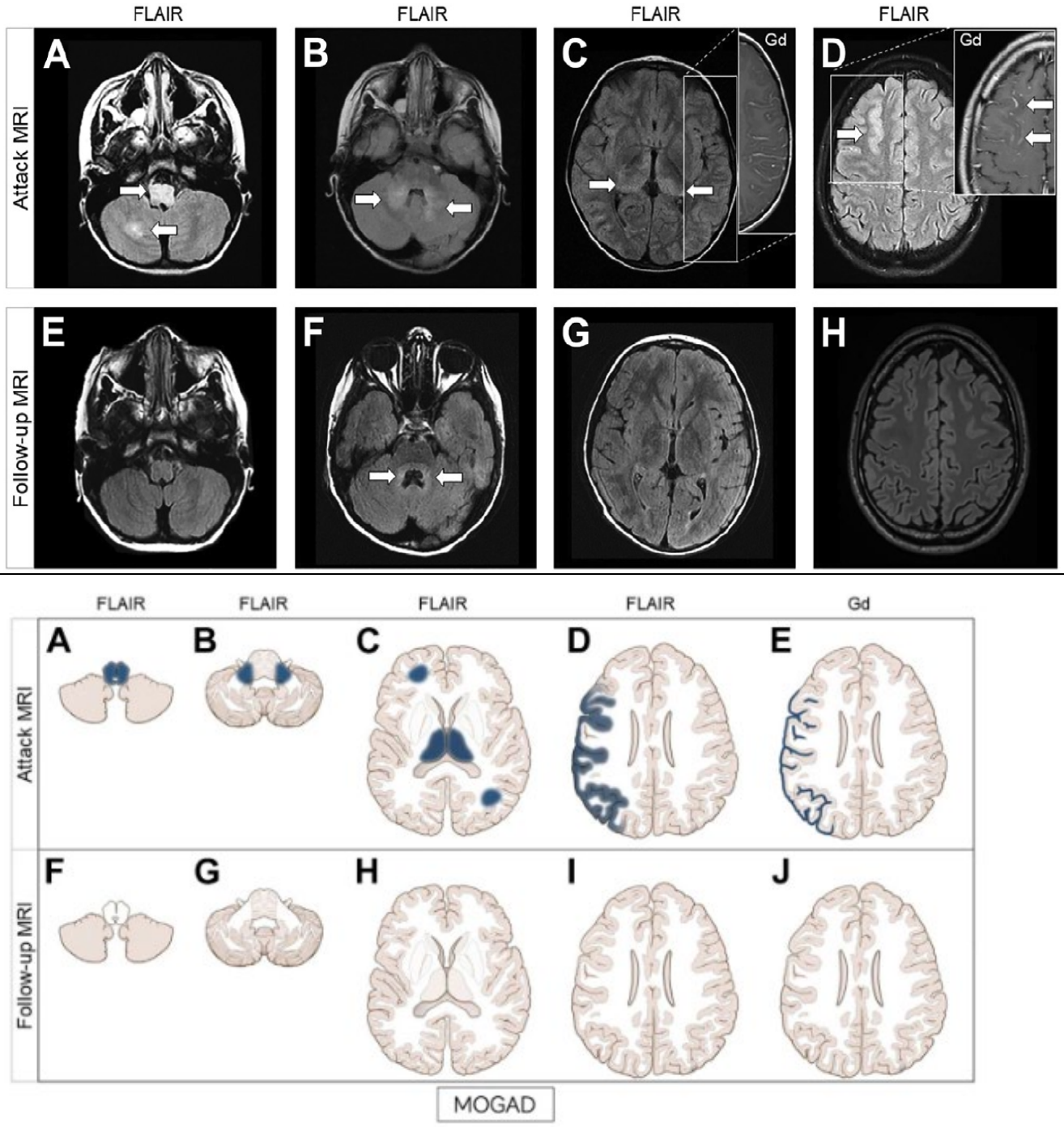


Figure 20 Brain lesions in MOGAD patients are shown with acute-phase findings in the top row and follow-up imaging in the bottom row (axial view). A T2 lesion in the medulla (A) and bilateral fluffy T2 lesions in the middle cerebellar peduncles (B), thalami, and white matter (C) resolve by follow-up (F, G, H). Extensive cortical T2 lesion (D) with leptomeningeal enhancement (E) also resolves (I, J) [31].

VII. CSF Analysis:

1. MOG testing

The preferential specimen for MOG antibody testing is serum. However, CSF testing is more promising and requires further studies. It's recommended that MOG testing be realized on serum using human full-length MOG cell-based assays. If live cell-based assays are unavailable, fixed cell-based assays are an alternative even though the latter has less specificity and sensitivity compared to live cell-based assays. ELISA has low specificity and sensitivity, which makes it not recommended for MOG-IgG testing.

According to the MOGAD 2023 Diagnostic criteria panel, serum MOG-IgG should include qualitative results (negative, low positive, or clear positive) and semi-quantitative results (titers, flow cytometry ratio, and visual scores). In fixed cell-based assays, titers of 1:100 or higher are considered clear positives. Results from fixed or live assays are regarded as low positives if they fall within the low range of the individual live assay or if titers range from 1:10 to less than 1:100 in fixed assays [8].

The probability of detecting MOG-IgG seropositivity is highest when patients are tested during the onset of acute attack. For optimal results, testing should ideally be performed before starting corticosteroids, immunoglobulins, or apheresis, as these treatments diminish serum MOG-IgG levels [32].

Initial serum MOG-IgG titers have limited prognostic value for relapse prediction. However, ongoing MOG-IgG seropositivity, especially with persistently high titers, is linked to a 2-10-fold increased risk of relapse. Current data are insufficient to establish optimal timing and interpretation of serial MOG-IgG testing. A standardized definition of persistence and further prospective studies are needed to clarify the prognostic importance of persistent seropositivity [8].

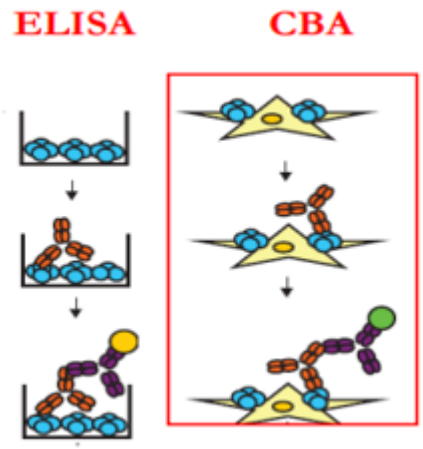


Figure 21: difference between ELISA and CBA testing by Marignier and al.

2. CSF TESTING: (general testing)

CSF pleocytosis, defined by white cell counts above 5 cells/µL, is observed in over 50% of patients experiencing a first demyelinating attack with MOG-IgG positivity. Up to 12% of these patients present with white cell counts exceeding 100 cells per high-power field. CSF pleocytosis is more common during active disease episodes than in remission and occurs more frequently in patients with ADEM or transverse myelitis compared to those with optic neuritis. CSF protein levels are elevated in 30% of patients with a first demyelinating attack and MOG-IgG, though this finding does not differentiate MOG-IgG-associated demyelination from other neuro-inflammatory conditions [8,33-36].

DISCUSSION OF OUR MOGAD SERIES

I. Epidemiological features:

In Xiaoying Lei and al. study the median age of patients was 40 years old at disease onset (range 20-67). 70% of patients were women [37].

Dauby and al.'s study noted a 1/1 sex ratio with the median age of clinical onset equal to 27.7 (range 9.8-39.5).

Halen and al.'s study included 42 MOG antibody-positive patients (27 female, 64.2%) with a median age of disease onset at 29 years (range 3–62; 9 pediatric cases), 47% were non-Caucasian (20 patients: 9 Asian, 7 South Asian, 4 other.

In Messias and al.'s study the mean age of the cohort was 35.8 years, and the mean age at disease onset was 31.0 years (range 6-64). Twenty-four patients (58.8%) were Caucasians. A modest majority were females (n ¼ 23; 56.1%), with a strong female predominance in the pediatric cohort (80%).

In our study, the mean age at onset was 27.8 years, with a male predominance of 66.6% (sex ratio: 2:1).

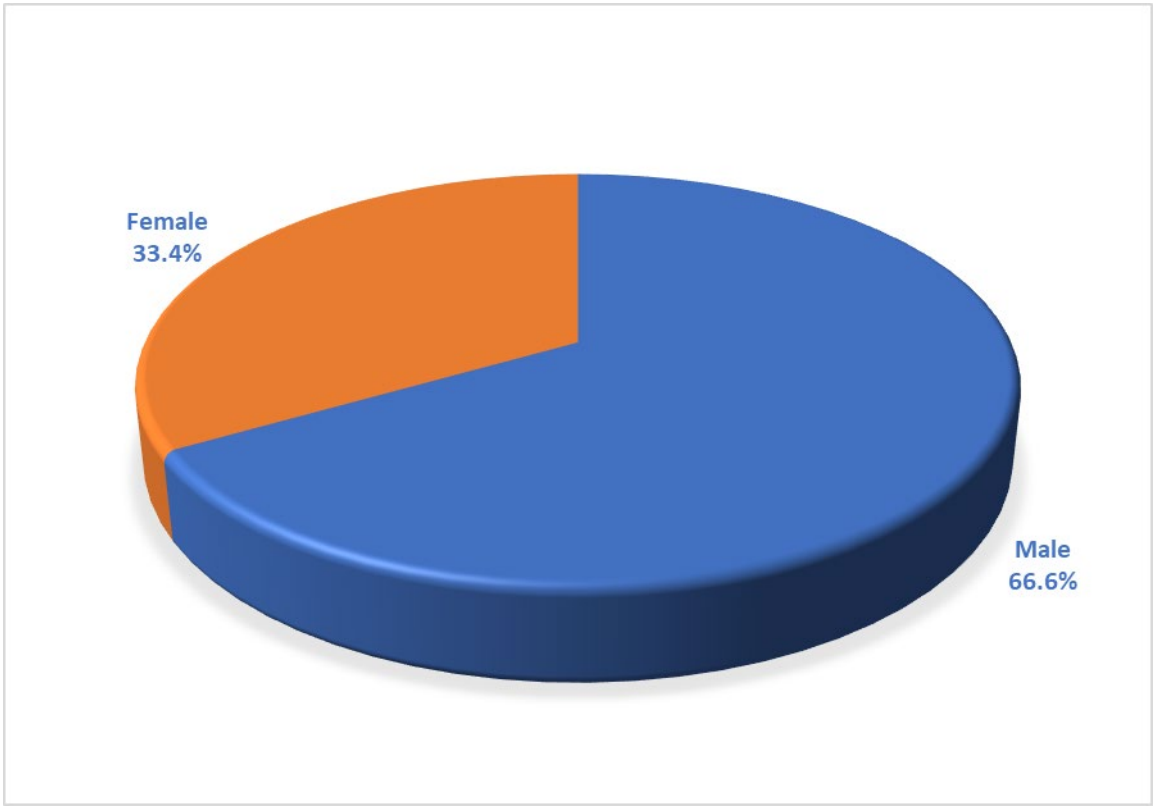


Figure 22: sex distribution within our MOGAD cohort.

II. Clinical features:

In Xiaoying Lei and al.'s study, demyelinating episodes in the central nervous system (CNS) were noted, with optic neuritis (ON) being the most prevalent presentation among the 25 patients (53%), of which 15 (60%) were unilateral. Followed by Rhombencephalitis (n=8, 17%), Limbic Encephalitis (n= 4, 9%), 4 cases of concurrent Myelitis and ON (n=4, 9%), ADEM-like form (n=3, 6%). As for relapse, 20 patients experienced a relapse attack (74%). Among these, 10 episodes were ON accounting for 50%, and 70% were unilateral ON. RE, LE and MY accounted for 25, 20 and 5% respectively. One patient presenting with RE also met the diagnostic criteria of area postrema syndrome (APS). Another patient with RE presented with imaging characteristics of chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) [37].

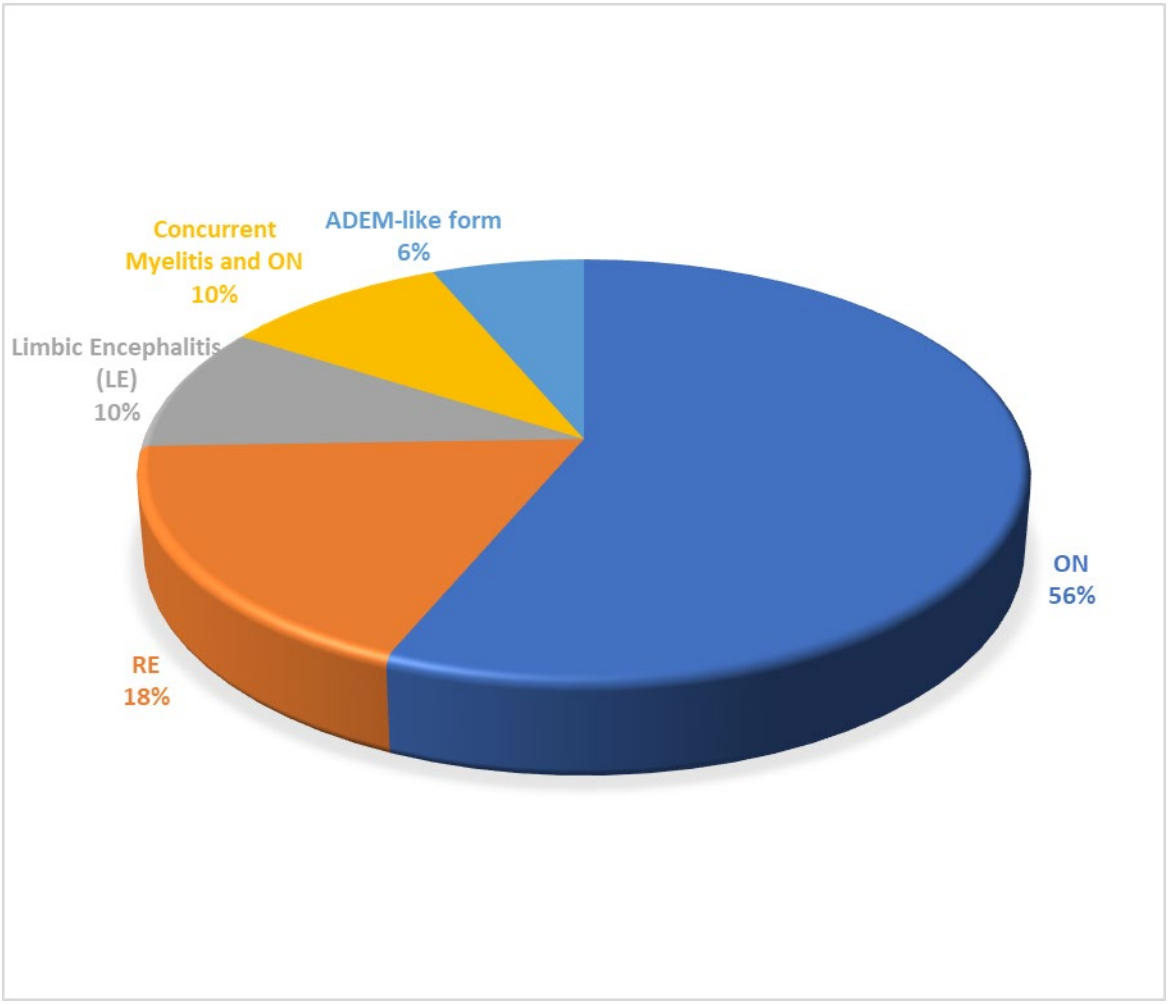


Figure 23: Distribution of MOGAD patients in Lei and al.'s study by clinical manifestations.

Messias and al.'s study describes the clinical data of 41 patients with MOGAD meeting the 2023 diagnostic criteria. The clinical manifestations were dominated by ON representing 68.3% of the cohort, among which 32.1% were bilateral. The study noted that 17 fundoscopy exams were performed and 58.8% revealed Optic Disc Edema. Also, headache was present among 75% of the patients with ON. The second and third most common presentations were ADEM (12.5%) then Transverse Myelitis (9.8%). 2 patients presented with Myelitis and FLAMES/cortical encephalitis accounting for 4.9% of the cohort. The median disease duration was 59.6 months, with 21 patients (51.8%) experiencing a relapsing episode, ON was the most common form of relapse. The median relapses per patient was 2.5 (ranging from 1 to 13), with 14 patients having

3 or more relapses. The study showed that, with all relapses taken into account, 32 patients (82.9%) presented with ON at some point.

The median EDSS was 4.4 at nadir during first relapse and 2.1 during the final follow-up visit.

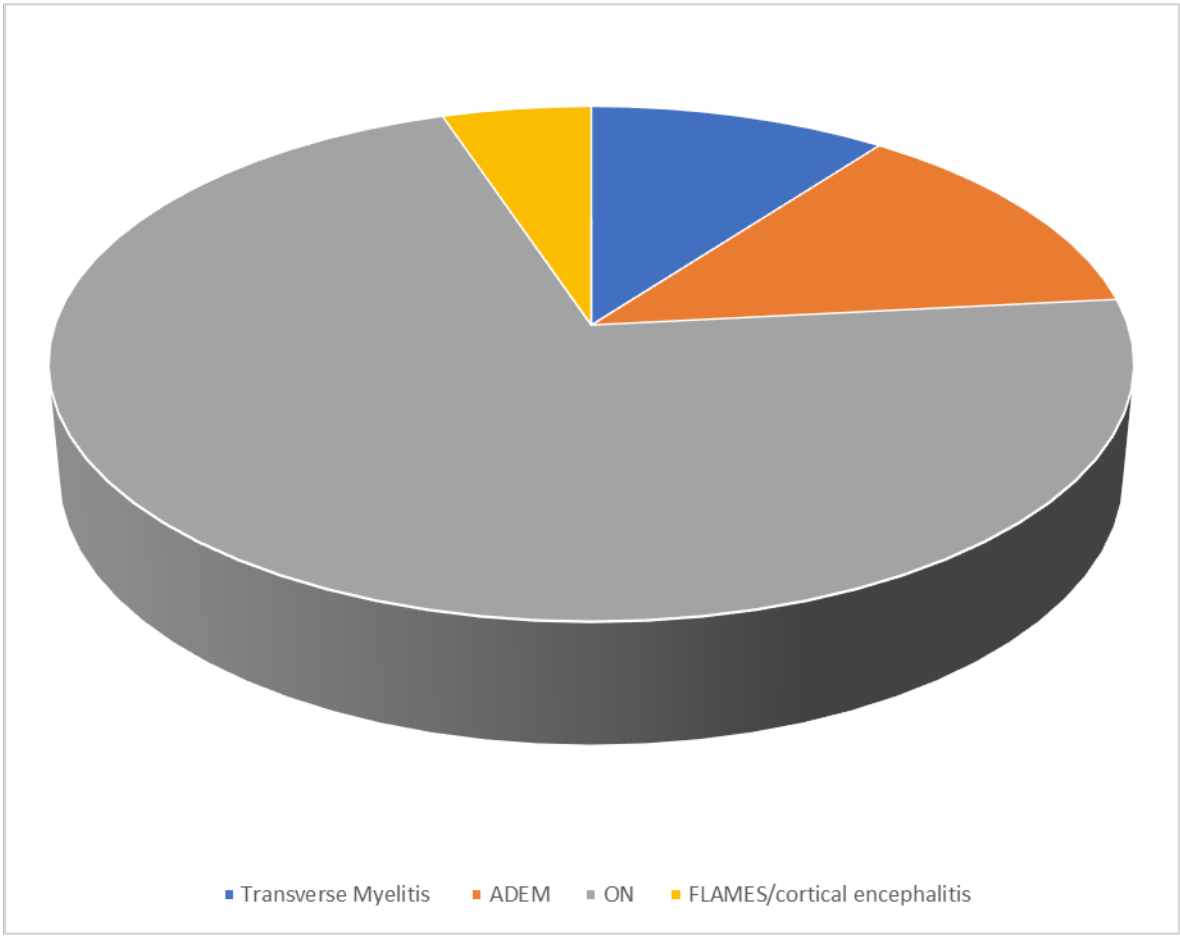


Figure 24: Distribution of clinical phenotypes in Messias and al's cohort study

As for Dauby and al., Among the 8 MOGAD patients studied, the most common onset clinical episode was ON (n=7; 87.5%), of which 57.1% were bilateral. Followed by 1 patient presenting with Isolated Myelitis (12.5%). The MOGAD transverse myelitis affected the upper cervical cord expanding to the lower medulla, as opposed to NMOSD's transverse myelitis which affected the lower cervical cord expanding to thoracic levels.

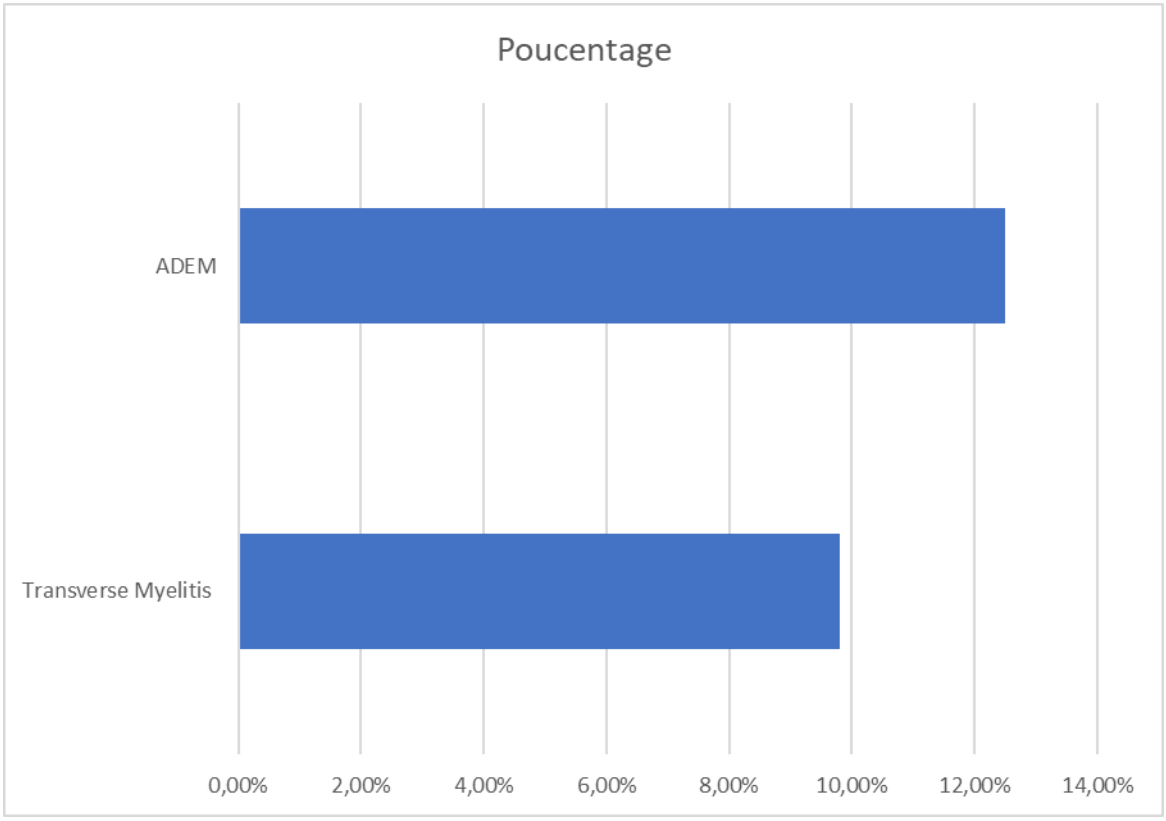


Figure 25: comparison between frequency of ADEM and Transverse myelitis in Dauby and al.'s MOGAD series.

Halen and al.'s study showed that the most common onset presentation was isolated optic neuritis, occurring in 55% (23 patients), with bilateral optic neuritis in 8 cases. Myelitis was recorded as the second most common presentation, accounting for 21% of patients. Among these 6 patients presented with longitudinally extensive transverse myelitis (LETM). Other onset episodes included brainstem syndromes, optic neuritis associated with myelitis, and cerebral syndromes such as tumefactive lesions, seizures, and ADEM. Although, the incidence of ADEM at onset was low, as most of the cohort was adult.

Our study identified optic neuritis as the most common clinical phenotype (n=7; 58.33%), followed by myelitis (n=3; 25%). MOGAD-associated Encephalopathy and cerebellar syndrome associated with brainstem involvement were less frequent, observed in one patient each (8.33%).

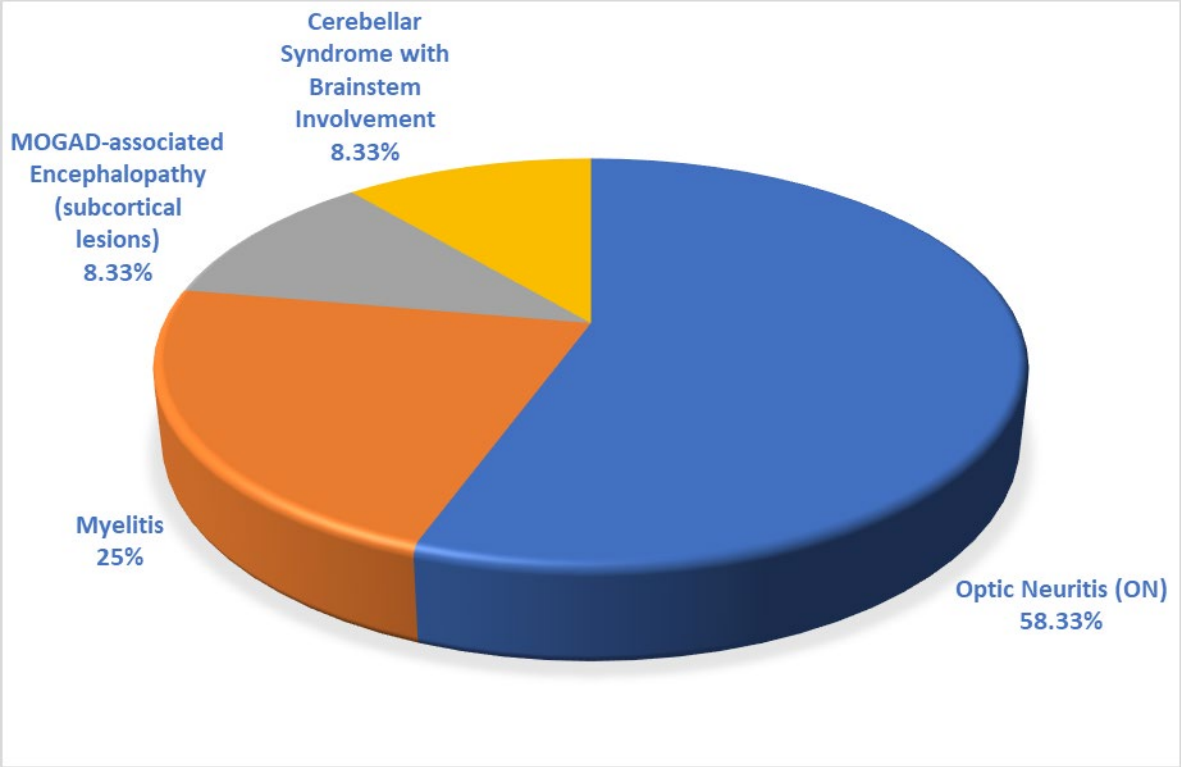


Figure 26: Distribution of the clinical presentation of our MOGAD series.

Regarding the clinical examination in our series, severe visual acuity loss was the most common symptom, observed in all patients with ON (n=7; 58.33%), followed by papilledema, which was also present in ON patients (n=6; 50%). Eye pain was reported in three patients (n=3; 25%), while limb weakness and sensory abnormalities were recorded in three patients (n=3; 25%). Headache was noted in two patients (n=2; 16.6%), and gait difficulties were observed in two patients (n=2; 16.6%). Urinary retention accompanied by constipation was reported in one patient (n=1; 8.33%), while cerebellar syndrome associated with brainstem involvement (ataxia, dysarthria, negative Romberg sign, etc.) was recorded in one patient (n=1; 8.33%).

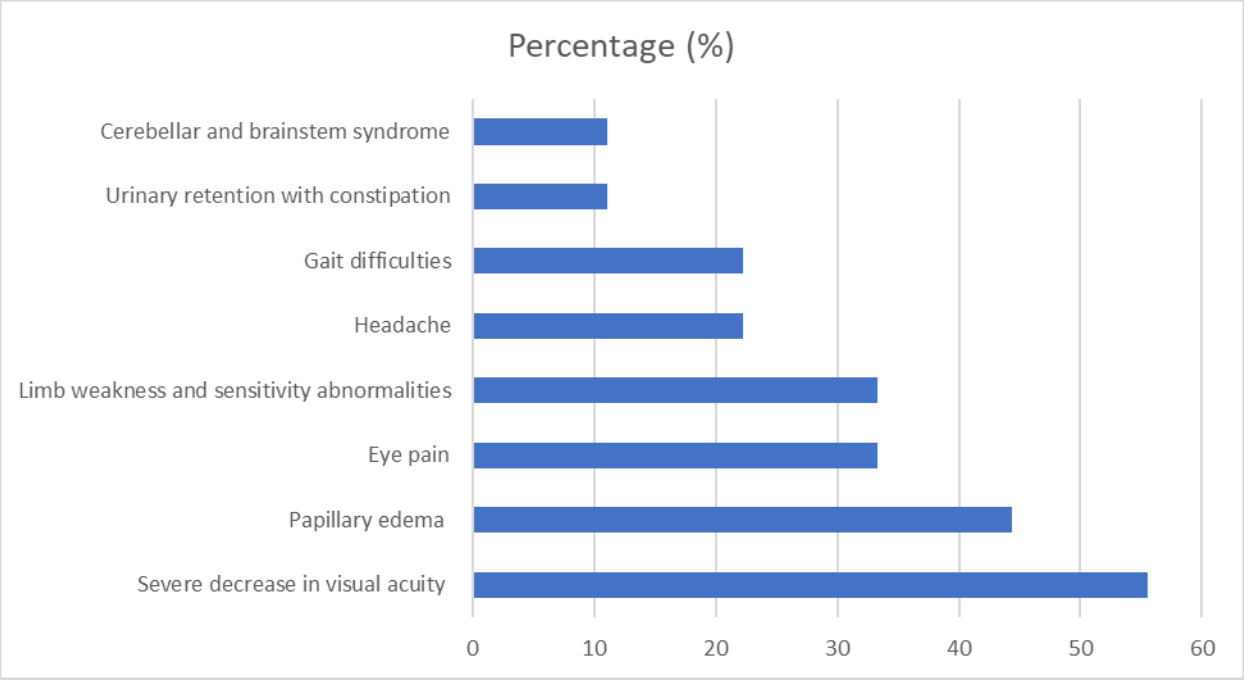


Figure 27: Distribution of the clinical exam findings in our MOGAD case series.

III. CSF and Serological Analysis:

Lei and al. performed 29 lumbar punctures. Elevated protein level was recorded in 10 samples (34%), and pleocytosis in 4 (14%). Also, 19 intrathecal oligoclonal bands tests were performed, and only 3 came back positive (19%).

In Messias and al. study Mog Testing was performed on 26 patients during acute phase. The median relapses before MOG-antibody testing was 2.3. As for CSF tests, 43.9% of the cohort presented with white blood cells superior to 5 cells/mm3. Concerning protein concentration, 26.8% of the cohort presented with CSF protein levels superior to 45mg/dl, and 17.2% had elevated protein levels. Oligoclonal bands tests were performed on 29 patients, only 5 came back positive [38].

Dauby and al. observed Lymphocytic Pleocytosis in 2 patients (25%), and 2 patients showed signs of auto-immunity either antinuclear uncharacterized antibody (n=1) or anti-GM1 IgM antibodies, both asymptomatic. No oligoclonal bands were found.

In our study CSF testing and serological testing revealed no abnormalities. In MOG-negative patients, we were not able to perform CSF anti-MOG testing.

IV. Treatment:

Lei et al. administered IVMP for 40 out of 47 attacks (85%). Combination therapy with IVMP and IVIG was given for three attacks (6%) due to severe encephalopathy. No treatment was administered for the remaining four attacks. During the remission stage, six patients (67%) were treated with Mycophenolate Mofetil (MMF), while three patients received monotherapy with prednisone, rituximab, or teriflunomide, respectively (37).

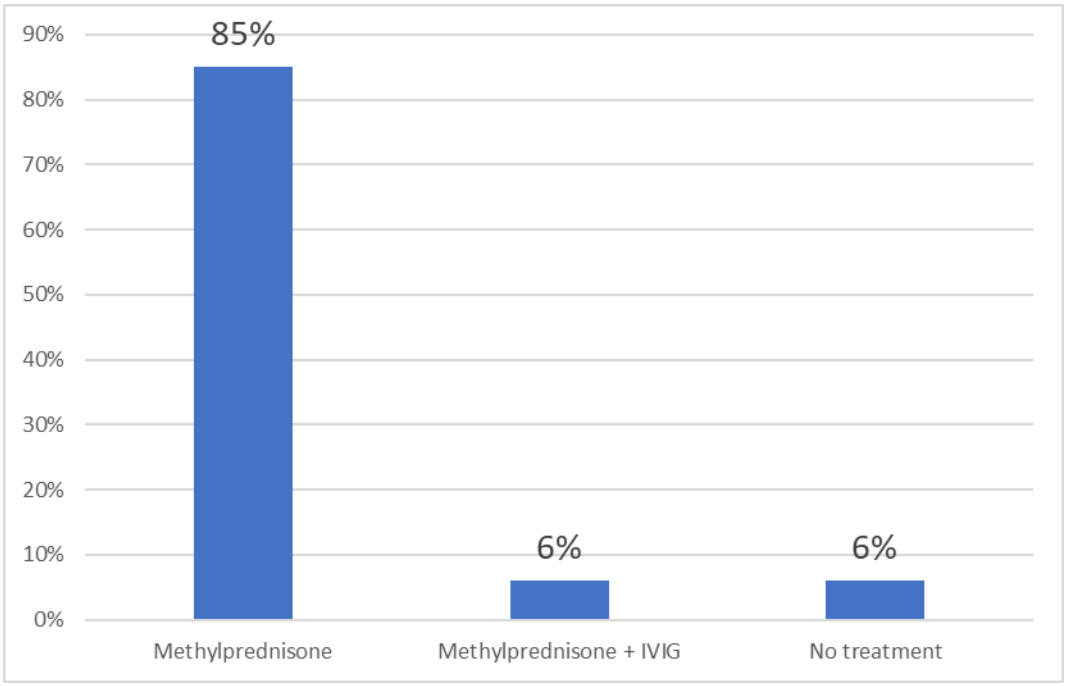


Figure 28: Distribution of treatment received by patients in the acute phase in Lei and al.'s study

Among the 41 patients in Messias and al.'s study, 33 received treatment during the acute phase accounting for 80.5%. Among these 33 patients, 45.4% received 1g of intravenous methylprednisolone (IVMP) for 3 to 5 days, and 12 patients (36.4%) received IVMP in association with plasma exchange. Three patients (9.1%) received oral and IM steroids and three patients received intravenous immunoglobulin (IVIG) [38].

Immunosuppressive treatment was prescribed to 34 patients (82.9%) with 20 patients (58.8%) receiving Azathioprine, 11 patients receiving Rituximab (32.4%), and 11 patients receiving other treatment (32.4%), the rest of this group (3 patients) received 3 other medication that weren't mentioned in the study [38].

Treatment alterations among thirteen patients (38.2%) were required due to the absence of improvement of symptoms or adverse effects. Rituximab was the most commonly prescribed as second-line treatment (n=6; 46.2%), followed by azathioprine and immunoglobulin (n=2, each; 23.1%) [38].

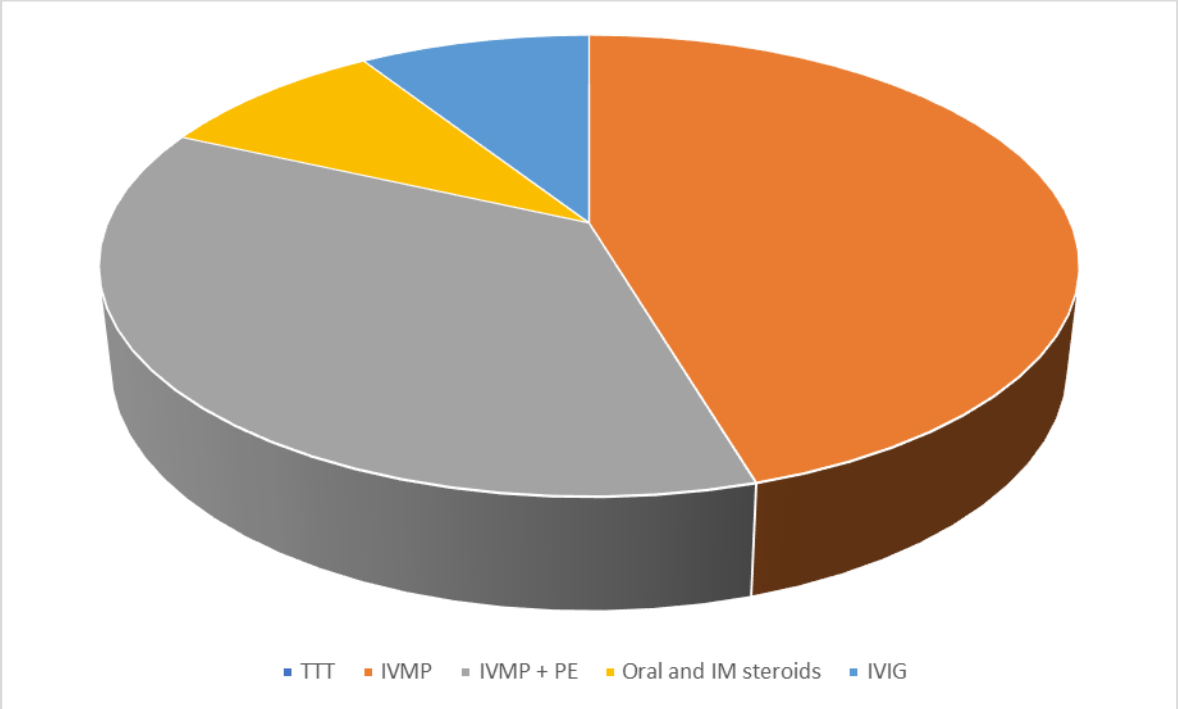


Figure 29: Distribution of treatment prescribed among the patients during the acute phase

In Halen and al's study Disease severity at onset was moderate to severe in 64% of the patients. However, the majority (74%) responded well to initial high-dose steroid therapy, with 21% receiving additional acute treatments such as plasma exchange, Mitoxantrone, or IVIG.

In our study, all patients received IV 1g of Methyl Prednisolone for 5 days during the acute phase. One patient with myelitis did not respond to corticosteroids, 5 sessions were then administered every other day. As for maintenance therapy, Rituximab was prescribed for 8 patients (66.6%) and 3 patient received Azathioprine (25%) and no maintenance treatment was prescribed for the rest of the patients (n=5; 41.6%)

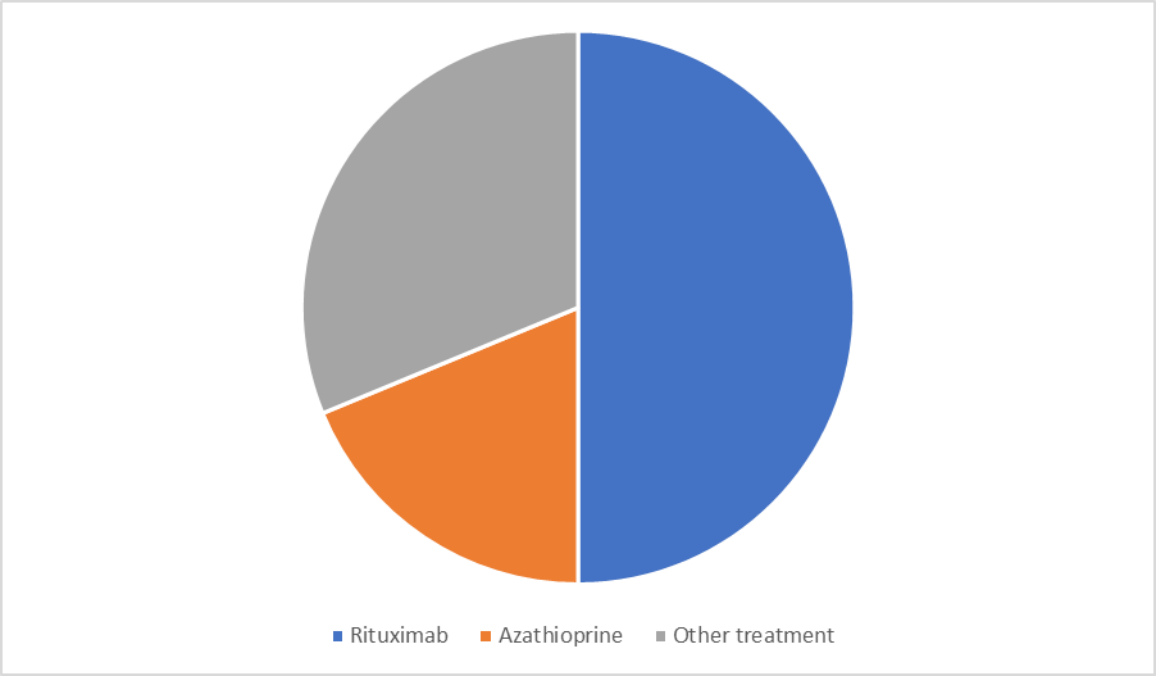


Figure 30: Distribution of therapy administered in our case series.

V. Prognosis:

In Messias and al.'s study the median follow-up period was 20 months (range 6–127). At follow-up, twelve (44%) patients experienced a relapsing course, and the median time to the first relapse was 9.5 months (range 2–120). The median Expanded Disability Status Scale score at the nadir was 3.5 (range 2–8) and was 0 (range 0–3) at the last follow-up [37].

Our study noted a favorable evolution of the disease after treatment, we recorded full-recovery at follow-up in 8 patients (66.6%). However, one patient experienced a disease course marked by relapsing episodes. The onset presentation of this patient was left ON, initial VA of the left eye was severely decreased: counts fingers at 2 meters. After managing the acute phase in our neurology department, the patient was discharged with a prescription of oral corticosteroids. During the first follow-up, the patient displayed a significant improvement, with an almost complete recovery of visual acuity (LVA: 9/10). During the second follow-up (2 months after being discharged), the patient presented with paresthesia of the upper and lower limb, clinical exam revealed brisk, diffuse and polykinetic tendon reflexes. Follow-up MRI was performed and revealed a longitudinally extensive myelitis (Longitudinal T2 hyperintensity in the cervical spinal cord spanning three vertebral levels. The patient was hospitalized and received 1g of IVMP for 5 days additionally to Azathioprine as maintenance treatment. A good evolution was recorded and the symptoms resolved. 4 months later, the patient presented with a second relapse episode, marked by cerebellar symptoms such as dizziness and balance problems. The clinical exam identified brisk nystagmus reflexes and polykinetic reflexes, brain MRI revealed round T2 hyperintensity lesions located in the medium left cerebellum peduncle. The episode was managed with IVMP and Azathioprine was maintained, the patient's symptoms resolved and was then discharged. The same patient had a 3rd relapse episode with decreased visual acuity in the right eye (6/10) and grade 1 right papilledema. IVMS was prescribed for 5 days, and Azathioprine was substituted with Rituximab, a full recovery was noted.

VI. MRI features:

A study conducted by J. Tom Mishael and al. on an Indian cohort evaluated brain MRI during the course of disease in MOGAD patients.

Among the 30 patients studied, 10 patients had normal MRIs and 20 showed abnormalities, of which 15 were symptomatic.

Most of the brain lesions are T2 hyper-intense, bilateral, asymmetrical poorly demarcated. In children, the common lesions are bilateral, and large, involving deep grey nuclei, cerebellar peduncles, and brainstem.

In this study, brainstem lesions were observed in 50% of patients (n=10), with 60% of these cases involving the pons. Midbrain and medullary lesions were each recorded in 20% of patients (n=4). The most common locations of MRI abnormalities were the supratentorial deep white matter (45%; n=9), followed by the juxtacortical/subcortical white matter (40%; n=8). Basal ganglia lesions were detected in 35% of patients (n=7).

Other affected brain regions included the corpus callosum and thalamus (25% each), cerebellum (20%), hippocampus (20%), cerebral peduncles (15%), cerebellar peduncles (15%), and internal capsule (15%). Periventricular white matter and hypothalamic involvement were noted in two patients (39).

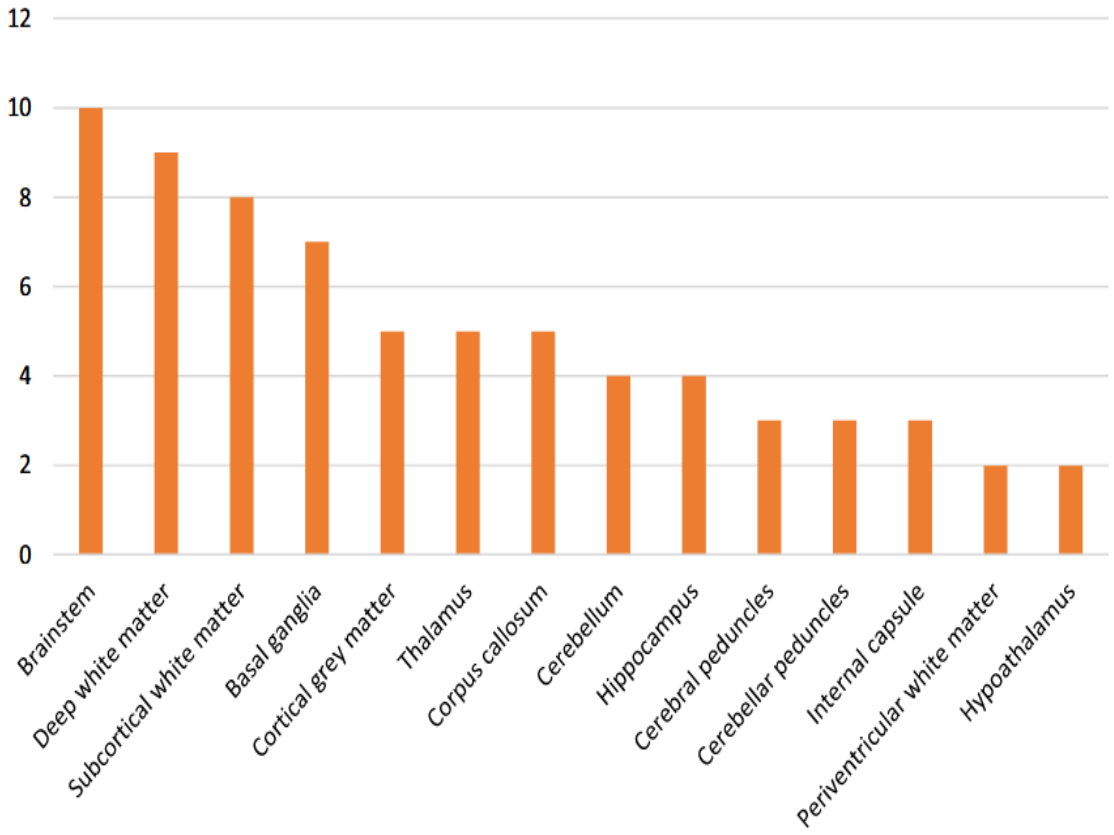


Figure 31: Distribution of Brain MRI lesions in J. Tom Mishael and al. study [39].

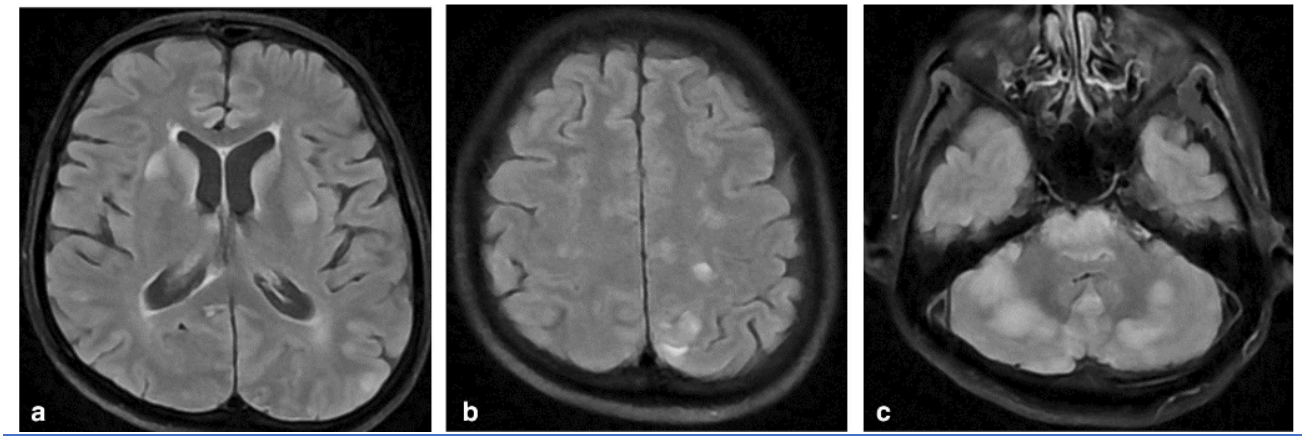


Figure 32: Brain MRI of a 48-year-old male with MOGAD-related encephalitis who presented with generalized tonic-clonic seizures. The lesions exhibit typical MOGAD characteristics—bilateral, asymmetrical, T2 hyperintense, and poorly demarcated with a "fluffy" appearance.

- (A) T2 hyperintense signal in the bilateral basal ganglia, cortex, and subcortical and deep white matter of the parietal and temporal lobes.
- (B) T2 hyperintense signal in the bilateral cortex and subcortical and deep white matter of the frontoparietal lobes.
- (C) T2 hyperintense signal in the pons and cerebellum

In the same study, 21 spinal cord MRIs were performed. 9 patients had T2 hyper-intense.

MRI findings in our cohort revealed a diverse range of radiological presentations, emphasizing the heterogeneity of MOGAD. Longitudinally extensive transverse myelitis (LETM) was observed in 25% of cases, with T2 hyperintensity spanning cervical and thoracic levels (C2-T7). Spinal cord swelling was present in one-third of these cases.

Optic neuritis (ON) accounted for 66.6% of presentations. Among these, unilateral ON with T2 hyperintensity and gadolinium enhancement occurred in 85%, while 14.2% showed bilateral involvement with posterior protuberance hyperintensity. Additionally, nodular temporoparietal lesions were identified in 8.33% of the cohort.

Idiopathic intracranial hypertension (IIH) was noted in 16.6%, associated with optic nerve enhancement. Transverse sinus hypoplasia, and an enlarged subarachnoid space found in one case (8.33%). Cortical and subcortical lesions were present in 8.33% of cases, manifesting as T1 hypointensity and T2/FLAIR hyperintensity without contrast enhancement. Similarly, subtentorial hyperintensity involving the pons, cerebellum, and peri-aqueductal region was noted in 8.33%.

These findings underscore the variability of MOGAD-associated MRI abnormalities and highlight the importance of integrating imaging and clinical data for precise phenotyping.

Conclusion

Our MOGAD series noted the following particularities:

- Male predominance.
- Atypical phenotypes like Idiopathic Intracranial Hypertension associated with optic neuritis.
- Brainstem lesions.
- Cortical and subcortical lesions.
- Our series recorded numerous relapsing MOGAD cases.

Although MOGAD shares similarities with other CNS diseases, it stands out as a distinct neurological entity due to its heterogeneous clinical, biological, and radiological features. Current evidence suggests that its long-term prognosis is generally benign. However, the underlying mechanisms and disease course remain incompletely understood.

Further studies are needed to identify relapse risk factors and establish optimal treatment protocols for both the acute phase and recurrent episodes, adapted to the varied presentations and phenotypes of this condition.

ABSTRACTS

Abstract

Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) is a recently recognized inflammatory demyelinating disease of the central nervous system (CNS). It can present as a monophasic or relapsing disorder.

This thesis aims to describe the frequency, clinical, paraclinical, immunological, therapeutic, and prognostic characteristics of MOGAD cases diagnosed at the Neurology Department of Mohammed VI University Hospital in Marrakech between 2016 and 2024.

Patients were diagnosed according to the 2023 International Diagnostic Criteria for MOGAD. Serum anti-MOG and anti-AQP4 antibody testing was performed using the Fixed Cell-Based Assay.

A total of 12 patients were included in the study (male-to-female ratio: 2:1), with a mean age of 27.8 years.

- Clinical Features: Optic neuritis was the most frequent presentation, occurring in five patients as an isolated manifestation. Other presentations included two cases of myelitis, two cases of optic neuritis associated with idiopathic intracranial hypertension (IIH), one case of optic neuritis with concurrent myelitis, one case of brainstem involvement, and one case of cortical encephalitis.
- Immunological Findings: Serum anti-MOG antibodies were positive in six patients, while all patients tested negative for anti-AQP4 antibodies.
- Treatment & Evolution: All patients received intravenous methylprednisolone during acute attacks. Rituximab was prescribed in five cases, while azathioprine was used in two cases. All patients showed clinical improvement.

MOGAD predominantly affected adults, with monophasic forms being the most common in our cohort. A particular finding was the association of optic neuritis with Idiopathic Intracranial Hypertension in two patients.

Despite its similarities with other CNS demyelinating disorders, MOGAD remains a distinct entity with variable clinical, immunological, and radiological features. Its long-term prognosis is generally favorable, but disease evolution remains incompletely understood. Further studies are required to identify relapse risk factors and optimize treatment strategies.

Résumé

La maladie à anticorps anti-MOG (MOGAD) est une entité récemment décrite appartenant aux maladies inflammatoires démyélinisantes du système nerveux central. Elle peut se présenter

sous forme monophasique ou à rechute.

Notre thèse a pour objectif de décrire la fréquence, le profil clinique, paraclinique, thérapeutique et pronostique des cas de MOGAD suivis au service de neurologie au centre hospitalier universitaire Mohammed VI de Marrakech durant la période allant de 2016 à 2024.

Le diagnostic a été retenu selon les nouveaux critères internationaux de diagnostic de la MOGAD publiés en 2023. Le dosage des Anticorps Anti-AQP4 et anti-MOG au sérum (Fixed Cell Based Assay) a été réalisé chez tous les patients.

12 patients ont été inclus dans cette étude. Sexe ratio H/F : 2/1. L'âge moyen était de 27.8 ans.

Sur le plan clinique, la Névrite optique était la manifestation clinique la plus fréquente, présente chez 5 patients dans sa forme isolée. Suivie de deux cas myélite, 2 cas de névrite optique associée à une hypertension intracrânienne idiopathique, un cas de névrite optique associée à une myélite, un cas d'atteinte du tronc cérébral et un cas d'atteinte encéphalique.

Sur le plan biologique, les anticorps sériques anti-MOG étaient positifs chez 6 patients.

En ce qui concerne le protocole thérapeutique, le bolus de méthylprednisolone a été instauré chez tous les patients. Le Rituximab a été prescris chez 5 patients et l'Azathioprine chez 2 patients. L'évolution était favorable chez tous les patients.

La plupart des cas de MOGAD étaient des adultes, les formes monophasiques étaient les plus fréquentes.

Notre étude a noté une forme assez particulière de MOGAD faite de Névrite Optique associée à une hypertension intracrânienne idiopathique (HII) chez deux patients.

En conclusion, la MOGAD, bien que similaire à d'autres maladies du SNC, est une entité distincte par sa variabilité clinique, biologique et radiologique. Son pronostic à long terme est généralement favorable, mais son évolution reste mal comprise. Des études supplémentaires s'avèrent donc nécessaires afin d'identifier les facteurs de rechute entre autres et ainsi optimiser la démarche thérapeutique.

ملخص

يُعدّ مرض الأجسام المضادة للبروتين الدبقي قليل التغصن المرتبط بإزالة الغلاف العازل للألياف العصبية من الأمراض الالتهابية التي تم التعرف عليها حديثًا، والتي تصيب الجهاز العصبي المركزي. يمكن أن يظهر هذا المرض على شكل نوبة واحدة أو نوبات متكررة.

يهدف هذا البحث إلى وصف نسبة انتشار المرض، والخصائص السريرية، والاختبارات المصاحبة، والفحوصات المناعية، وخيارات العلاج، ومسار المرض لدى المرضى الذين تم تشخيصهم في قسم الأمراض العصبية بالمركز الاستشفائي الجامعي محمد السادس بمراكش خلال الفترة من ألفين وستة عشر إلى ألفين وأربعة وعشرين.

تم تشخيص جميع المرضى وفقًا للمعايير الدولية المعتمدة في السنة الثالثة بعد الألفين . المضادة للبروتين الدبقي قليل التغصن كما أجريت تحاليل للكشف عن الأجسام المضادة في الدم باستخدام تقنية الفحص الخلوي الثابت.

شملت الدراسة اثني عشر مريضًا، وكانت النسبة الأعلى للذكور بنسبة ثلثي الحالات، وبلغ متوسط العمر سبعة وعشرين عامًا وثمانية أشهر.

كان التهاب العصب البصري أكثر الحالات شيوعًا، حيث ظهر لدى خمسة مرضى كعرض منفرد. كما تم تسجيل حالتين مصابتين بالتهاب النخاع الشوكي، وحالتين بالتهاب العصب البصري مصحوبًا بارتفاع الضغط داخل الجمجمة، وحالة واحدة لالتهاب العصب البصري مع التهاب النخاع الشوكي، وحالة واحدة لالتهاب جذع الدماغ، وحالة واحدة لالتهاب الدماغ.

الفحوصات المناعية :أظهرت التحاليل أن نصف المرضى يحملون أجسامًا مضادة للبروتين الدبقي قليل التغصن، في حين كانت نتائج التحاليل سلبية لجميع المرضى فيما يخص الأجسام المضادة.

العلاج والتطور المرضي :تلقى جميع المرضى علاجًا بالكورتيزون خلال المرحلة الحادة، وتم وصف علاج مناعي متخصص لخمسة مرضى، بينما تم استخدام علاج آخر مُثبط للمناعة لدى مريضين. أظهرت جميع الحالات تحسنًا سريريًا واضحًا.

كان أغلب المصابين من البالغين، وشكلت النوبات المنفردة النسبة الأكبر من الحالات

.لالتهاب العصب البصري المصحوب بارتفاع الضغط داخل الجمجمة لدى مريضين كما لوحظ وجود حالة غير معتادة.

ورغم تشابه هذا المرض مع بعض الاضطرابات العصبية الأخرى، إلا أنه يتميز بتنوع الأعراض السريرية، والاختلافات المناعية، .ويبدو أن المآل العام للمرض جيد، إلا أن تطوره على المدى الطويل ما زال غير مفهوم بشكل كامل، مما يستدعي إجراء المزيد من الدراسات لتحديد عوامل تكرار النوبات وتحسين أساليب العلاج.

APPENDICES

Exploitation Sheet – MOGAD Patient

Patient Information

- **Patient Name**: _____
- **Patient ID**: _____
- **Date of Birth**: _____
- **Gender**: ☐ Male ☐ Female
- **Diagnosis Date**: _____
- **Medical History**:
  - ☐ Multiple sclerosis
  - ☐ Neuromyelitis optica
  - ☐ Other (please specify): _____

Clinical Examination

- **Symptoms at Diagnosis**:
  - ☐ Vision loss
  - ☐ Headache/Eye pain
  - ☐ Muscle weakness
  - ☐ Ataxia
  - ☐ Urinary incontinence
  - ☐ Neurological pain
  - ☐ Gait difficulties
  - ☐ Paresthesia
  - ☐ Other (please specify): _____

- **Neurological Assessment (Neurological function score)**: _____

MRI Imaging

- **MRI Performed**: ☐ Yes ☐ No
- **Type of Imaging**:
  - ☐ FLAIR
  - ☐ T2
  - ☐ Post-contrast T1
- **Lesion Locations**:
  - ☐ Optic nerve
  - ☐ Medulla
  - ☐ Middle cerebellar peduncles
  - ☐ White matter
  - ☐ Cortical/Subcortical
- **Lesion Description**:
  - ☐ Fluffy
  - ☐ Hypointense
  - ☐ Hyperintense
  - ☐ Other (please specify): _____

Laboratory Tests

- **Anti-MOG Antibody Results**:
  - ☐ Positive
  - ☐ Negative
  - Test Date: _____

Follow-up (3, 6, 12-month follow-up)

- Lesions Resolved:
  - ☐ Yes
  - ☐ No
- Lesion Evolution:
  - ☐ Reduction
  - ☐ Stability
  - ☐ Increase
- Clinical Symptoms:
  - ☐ Improvement
  - ☐ No change
  - ☐ Worsening

Treatment Administered

- Type of Treatment:
  - ☐ Corticotherapy
  - ☐ Plasmapheresis
  - ☐ Immunosuppressants
  - ☐ Other (please specify): _____
- Response to Treatment:
  - ☐ Good response
  - ☐ Partial response
  - ☐ No response

Additional Notes

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قسم الطبيب

أقْسِم بِالله العَظِيم

أن أراقبَ الله في مِهْنَتِي.

وأن أصُونَ حياة الإنسان في كآفّةِ أطوَارهَا في كل الظروف والأحَوال باذلا وسْعِي في انقاذها مِن الهَلاكِ والمرَضِ والألَم والقَلَق.

وأن أحفَظَ لِلنَاسِ كرَامَتهُم، وأسْتر عَوْرَتهُم، وأكتمَ سِرَّهُمْ.

وأن أكونَ عَلى الدوَام من وسائِل رحمة الله، باذلا رعايتي الطبية للقريب والبعيد، للصالح والطالح، والصديق والعدو.

وأن أثابر على طلب العلم، وأسَخِّرَه لِنَفْعِ الإِنْسَان لا لأذَاه.

وأن أُوَقّرَ مَن عَلَّمَني، وأُعَلّمَ مَن يَصْغرَني، وأكون أخا لِكُلّ زَميلٍ في المِهنَةِ الطِّبّيَة مُتعَاونِينَ عَلى البرّ والتقوى.

وأن تكون حياتي مِصْدَاق إيمَاني في سِرّي وَعَلانيَتي، نَقِيَّة مِمّا يُشينهَا تجَاهَ الله وَرَسُولِهِ وَالمؤمِنين.

والله على ما أقول شهيد

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السنة 2025 الاطروحة رقم : 190

مرض الأجسام المضادة لبروتين الميالين قليل التغصن المرتبط بالخلايا الدبقية:(MOGAD)
تجربة قسم طب الأعصاب في المركز الاستشفائي الجامعي محمد السادس بمراكش

الأطروحة

قدمت ونوقشت علانية يوم 2025/06/18

من طرف

حسام الدين بنسايح

المزداد في 20/06/1996 ب اليوسفية

لنيل شهادة الدكتوراه في الطب

الكلمات المفتاحية

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