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Original article

Treatment strategies of AQP4-IgG positive neuromyelitis optica spectrum disorder and MOG antibody-associated disorder in Switzerland: a nationwide survey

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ABSTRACT

Introduction: Neuromyelitis optica spectrum disorders (NMOSD) and Myelin Oligodendrocyte Glycoprotein (MOG) antibody-associated disease (MOGAD) are neuroinflammatory conditions characterized by attacks, primarily affecting the spinal cord and the optic nerve. When left untreated, these disorders can result in severe neurological disability. Although recent advancements have improved treatment, many questions remain regarding the optimal management of these rare conditions.

Methods: We conducted a national survey among neurologists in Switzerland experienced in treating NMOSD and MOGAD. The survey comprised 42 questions covering diagnostic methods, acute treatment, maintenance immunotherapy and approaches to long-term strategy.

Results: Twenty-one out of 28 invited neurologists took part in the survey (response rate 75 %). There was high consensus on treating acute attacks with high-dose steroids and with plasmapheresis in severe cases. In NMOSD, 71.4 % recommended oral steroid tapering, compared to 85.7 % in MOGAD. All participants advocated maintenance treatment after the first attack in NMOSD, compared to only 10 % in MOGAD. Indeed, many participants advised starting therapy in MOGAD after the first attack only in cases of severe attacks (38 %), persistent MOG-antibodies (10 %) or both (19 %). Rituximab was the most used first-line maintenance immunotherapy for both diseases. Approaches to treatment strategy for the long-term varied with a tendency to recommend de-escalation or discontinuation in stable patients with MOGAD, but not with NMOSD.

Discussion: This study highlights current treatment approaches to NMOSD and MOGAD across Switzerland. Rituximab remains the most prescribed drug for both conditions. The overall variability in recommendations underscores the need for greater awareness of disease-specific management and for further research to optimize treatment strategies for patients with NMOSD and MOGAD.

1. Introduction

Neuromyelitis optica spectrum disorders (NMOSD) and myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease (MOGAD) are rare inflammatory diseases of the central nervous system (CNS) with a clinical and radiological presentation distinct from

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multiple sclerosis (MS), requiring different treatment strategies (Cacciaguerra and Flanagan, 2024).

Typical clinical presentations of NMOSD are optic neuritis (ON), longitudinal extensive transverse myelitis (LETM) and area postrema syndrome, but other brainstem involvement, diencephalic syndromes and cerebrum involvement can occur (Wingerchuk et al., 2015). Most patients are seropositive for IgG antibodies against the aquaporin-4 (AQP4) water channel on astrocytic foot processes, however, seronegative cases exist (Jarius et al., 2023; Lennon et al., 2005). NMOSD relapses are typically severe and are the main reason for permanent neurological disability (Cacciaguerra and Flanagan, 2024; Kumpfel et al., 2024).

In MOGAD, the autoantibodies are directed against the MOG protein, specifically expressed on the surface of oligodendrocytes (Sechi et al., 2022),[14,15]. Characteristic clinical manifestations include optic neuritis, myelitis, and acute disseminated encephalomyelitis (ADEM) (Banwell et al., 2023; Sechi et al., 2022), which is the most common presentation in pediatric patients with MOGAD (Cacciaguerra and Flanagan, 2024). Recently, it became clear that MOGAD can have an even more heterogeneous presentation than initially thought. It can also present with acute cerebellitis (Ishikura et al., 2022), acute hemorrhagic leukencephalitis (Skarsta et al., 2023) and with feverish anti-MOG-associated cerebral cortical encephalitis often with seizures with typical unilateral cortical FLAIR-hyperintense lesions (Budhram et al., 2019).

High-dose corticosteroids and plasmapheresis are the main therapies for acute attacks in both diseases (Kleiter et al., 2016; Sechi et al., 2022). Oral steroid tapering is generally recommended to prevent early subsequent attacks; however, the optimal duration of oral tapering is unknown (Cacciaguerra and Flanagan, 2024; Kumpfel et al., 2024; Sechi et al., 2022; Takai et al., 2021).

In NMOSD, rituximab, azathioprine and mycophenolate mofetil have been used for a long time as maintenance treatment (Kumpfel et al., 2024). Recently, results from several randomized placebo-controlled trials showed efficacy of monoclonal antibodies targeting the complement system (eculizumab (Pittock et al., 2019), ravulizumab (Pittock et al., 2023)), interleukin 6 (IL6) receptor (satralizumab (Traboulsee et al., 2020; Yamamura et al., 2019)) and CD19-positive B-lymphocytes (inebilizumab (Cree et al., 2019)), leading to substantial changes in the treatment landscape for NMOSD.

In MOGAD, due to a monophasic disease course in 40–50 % of patients and the generally good recovery from relapses, maintenance therapy is often considered only after a second attack (Cacciaguerra and Flanagan, 2024; Sechi et al., 2022). While no approved medication for MOGAD is available, several agents have been used as first-line maintenance therapy, mostly azathioprine, mycophenolate mofetil, rituximab or intravenous immunoglobulins (IVIG) (Chen et al., 2020, 2022) and to a lesser extent tocilizumab (Ringelstein et al., 2022). However, as high-quality evidence from randomized controlled trials is lacking, MOGAD is treated heterogeneously across different regions and countries, highlighting the need for consensus-based guidelines.

The growing number of therapeutic choices for NMOSD/MOGAD ultimately also requires considerations of possible de-escalation scenarios. However, the data on de-escalation experience is sparse.

Considering recent substantial changes in treatment options for NMOSD and the lack of approved treatments for MOGAD, the aim of this study was to explore the current therapeutic strategies of specialized neurologists treating NMOSD and MOGAD in Switzerland, to raise attention to the specific management of these diseases, identify unmet needs and foster scientific exchange about treatment strategies.

2. Methods

We conducted an online survey among adult neurologists in Switzerland experienced in treating neuroimmunological disorders, especially NMOSD and MOGAD. We invited neurologists working at university hospitals, non-university hospitals and in private practice. All language-specific regions (German, French, Italian) were included to achieve a nationwide representation.

The online tool Google Forms (www.google.com/forms) was used for creating the survey. The survey included 43 single- or multiple-choice questions about treatment of attacks, strategies of maintenance treatment, follow-up of patients and strategies for treatment deescalation and discontinuation for MOGAD and NMOSD. The full list of questions from the survey can be found in Supplementary Note 1. Only AQP4-IgG positive NMOSD was considered; for readability reasons, the term NMOSD instead of AQP4-IgG positive NMOSD is used in the results and discussion section. For questions about treatment deescalation and discontinuation in stable patients with NMOSD and MOGAD after two or five years, disease stability was defined as no occurrence of attacks, no silent radiological activity and stable EDSS during this time period. Some questions about the initiation of maintenance treatment explored whether it should be started only after a severe attack, with the definition of severity left to the participant's clinical judgment. Multiple answers were possible for questions about the choice of maintenance treatment and de-escalation strategies. Responses were collected from October to December 2024. 28 expert neurologists were invited to participate via email. Neurologists from neuroimmunology units at Swiss university hospitals (Basel, Bern, Geneva, Lausanne, Zurich), from non-university tertiary referral hospitals and from specialized neuroimmunology private practice were invited to participate.

Microsoft Excel (version of 2016), Python 3.12.4 (Python Software Foundation with the following packages: pandas (McKinney, 2010), plotly and its kaleido engine for interactive visualizations and static image export (PlotlyTechnologiesInc., 2015) and Jupyter Notebooks (Kluyver, 2016)) and Affinity Designer 2 (Serif) were used for data processing and/or the generation of figures. For the Sankey diagram illustrating maintenance treatment strategies, we assigned a weighting factor to each transition based on the number of therapeutic choices (first-, second-, or third-line therapies) made by each participant, to prevent disproportionate influence from physicians whose reponses included multiple treatment options. Participants who did not provide answers for each step were excluded.

The results from the survey were summarized and are presented in a condensed manner for 4 key topics: 1. Acute treatment; 2. Maintenance treatment; 3. Surveillance strategies and 4. De-escalation and discontinuation.

3. Results

3.1. Participants

Twenty-one out of 28 neurologists took part in the survey (response rate 75 %). Fifty-seven percent of the participants indicated to work in a university hospital, 38 % in a non-university hospital and 5 % in private practice. Fifty percent of the participants reported 10 or more years, 20 % five to nine years, 20 % two to four years and 10 % less than two years of experience in treating patients with MOGAD and NMOSD. 24 % of participants treated up to five, 29 % between five and ten, 24 % between ten and 20, 19 % between 20 and 50 and one participant (5 %) treated more than 50 patients with MOGAD and NMOSD. All participants replied to all survey questions.

3.2. Antibody testing

The participants indicated to test for MOG-IgG mainly with live cell-based assays (60 % of participants, with 45 % testing in serum and CSF and 15 % testing only in serum), while a minority used fixed cell-based assays (25 %) or ELISA (5 %), and two participants (10 %) did not know the test method they are using. AQP4-IgG were mainly tested in fixed (45 %) or live (40 %) cell-based assays, 5 % used ELISA and 10 % did not know the test method they are using.

3.3. Treatment of acute attacks

All participants (100 %) indicated to treat acute attacks in MOGAD with high-dose intravenous (i.v.) steroids as first-line (500–1000 mg of methylprednisolone per day for three to five days). In NMOSD, 95 % of participants reported to treat acute attacks with high-dose i.v. steroids, while one participant indicated to use apheresis therapy as a first-line therapy. While in MOGAD most participants (85.7 %) recommended tapering of oral corticosteroids after an acute attack in all or most cases, a substantial part (28.6 %) of participants recommended tapering after acute NMOSD attacks only in severe cases, sometimes or never (Fig. 1). In MOGAD, the recommended duration of oral tapering was on average longer than in NMOSD (MOGAD: 38 % 4–6 months versus NMOSD 5 %; Fig. 1).

As second-line treatment of acute attacks in case of residual symptoms, a majority of participants recommended apheresis therapy in NMOSD (81.1 %) and MOGAD (71.4 %), while a minority recommended repeating high-dose steroid treatment (9.5 % in MOGAD and NMOSD), IVIG (14.3 % in MOGAD and 9.5 % in NMOSD) or ultra-high-dose i.v. steroids alone (4.8 % in MOGAD; Fig. 2).

As third-line attack therapy, 35 % of participants recommended complement inhibitors in NMOSD, whereas only 4.8 % of participants used them for MOGAD. 20 % of participants used cyclophosphamide in NMOSD and only 9.5 % recommended it in MOGAD. The other participants recommended similar treatments in third- as in second-line (Fig. 2).

3.4. Maintenance treatment

All participants (100 %) recommended starting a maintenance treatment after the first attack in NMOSD. In MOGAD, the recommendations were more heterogenous, with 10 % of participants recommending maintenance treatment always and 19 % never after a first attack. The remaining participants considered a maintenance treatment after the first attack in case of a severe attack (38 %), if the MOG-IgG titer remained elevated (10 %) or in case of a severe attack combined with persisting elevated MOG-IgG at follow-up (19 %; Fig. 3).

For NMOSD, rituximab was a first choice drug for the majority of participants (85 %), followed by the recently approved antibody therapies targeting complement (eculizumab/ravulizumab, 40 %), the IL6 receptor (satralizumab, 30 %) or CD19-positive B-cells (inebilizumab, 30 %) as first-line treatment (Fig. 4A). Only a minority indicated to use classical immunosuppressants. As second-line treatment, complement inhibitors, inebilizumab and satralizumab were the preferred choices. In patients refractory to first- and second-line treatment, a combination of several disease-modyfing drugs (DMTs) was the most widely recommended treatment options (Fig. 4A).

For MOGAD, most participants (48 %) recommended rituximab as a

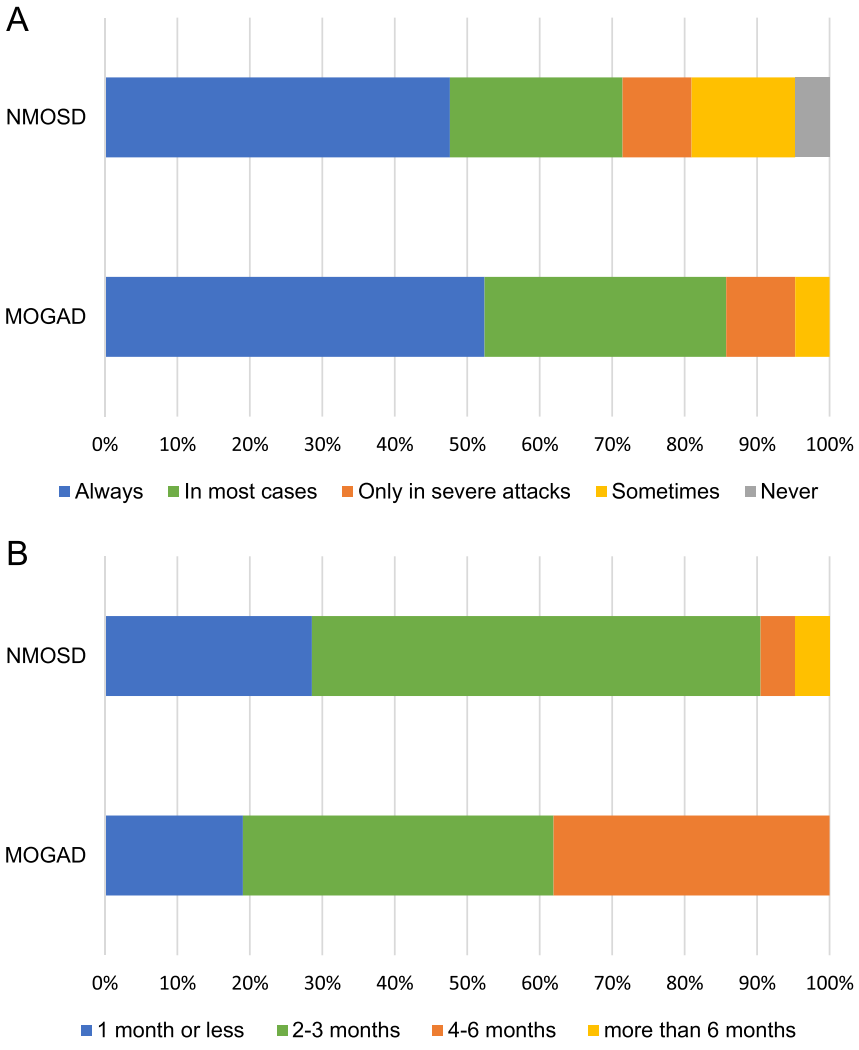

Fig. 1. Recommended use (A) and duration (B) of steroid taper after acute attacks. Stacked bar plots illustrating the percentage of participating neurologists favouring steroid taper in NMOSD (upper panel) and MOGAD (lower panel).

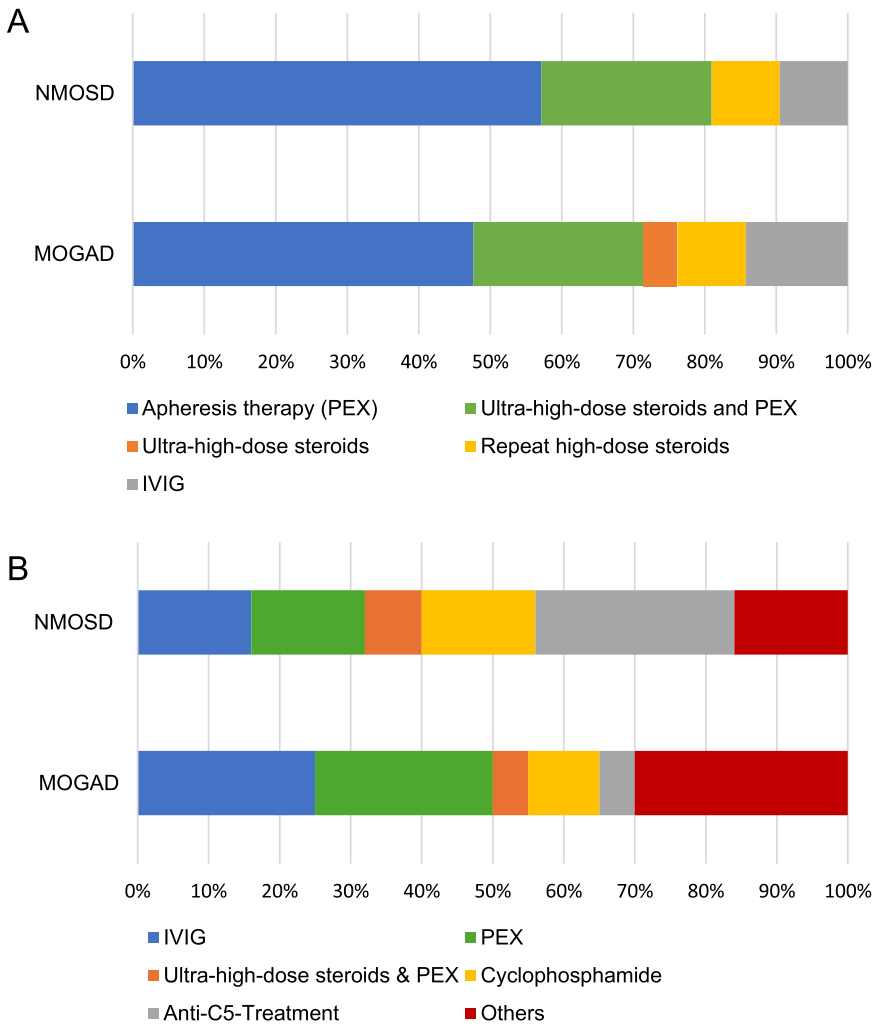

Fig. 2. Recommendations for second-line (A) and third-line (B) treatment of acute attacks. Stacked bar plots illustrating the percentage of participating neurologists in NMOSD (upper panel) and MOGAD (lower panel). IVIG = intravenous immunoglobulins. PEX = plasmapheresis.

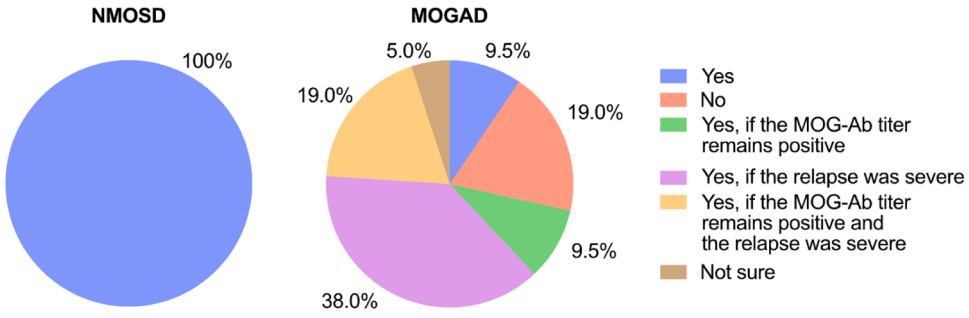

Fig. 3. Maintenance treatment initiation after the first attack. Pie chart demonstrating the percentage of participating neurologists with different recommendations for NMOSD (left panel) and for MOGAD (right panel).

first-line treatment, followed by periodic IVIG (33 %) and azathioprine (Fig. 4B). In second-line, tocilizumab was the preferred option. Third-line treatment recommendations were heterogeneous, with periodic IVIGs being most widely used option, followed by a combination of several immunosuppressants (Fig. 4B).

3.5. *Surveillance strategies during follow-up*

For follow-up of stable patients with NMOSD, 24 % of participants recommended annual cerebral MRI, 48 % recommended annual cerebral and spinal MRI, 14 % recommended no radiological follow-up and 14 % were not sure whether to do routine radiological follow-up or not. One third (33 %) of participants recommended follow-up measurements of AQP4-antibody titers and 38 % recommended regular measurement of serum neurofilament light chain (NfL) or glial fibrillary acidic protein (GFAP).

In stable patients with MOGAD, 43 % of participants recommended annual cerebral MRI, 24 % recommended annual cerebral and spinal MRI, 19 % recommended no routine radiological follow-up and 14 % were not sure whether to do routine radiological follow-up or not.

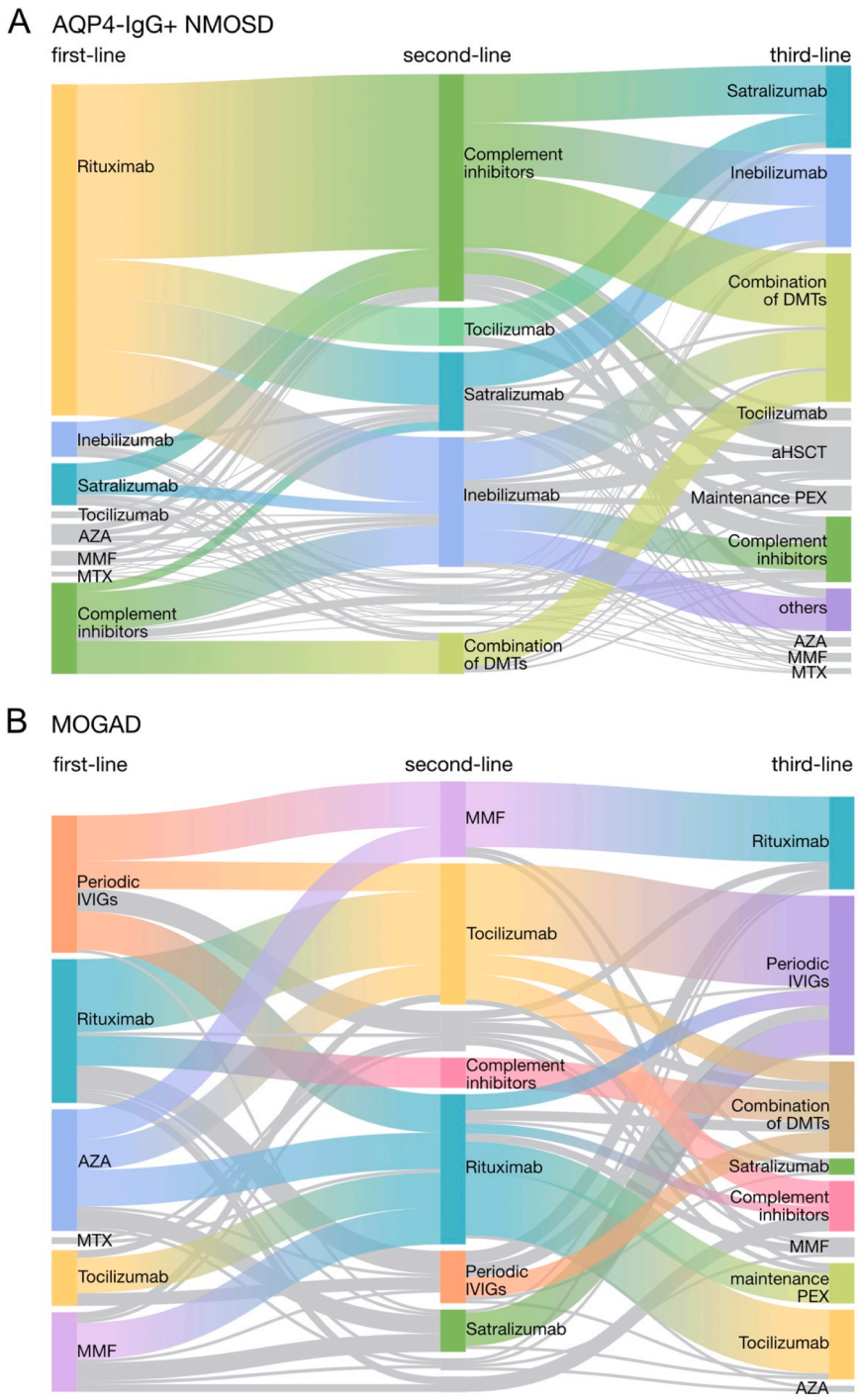

Fig. 4. Escalation strategies for maintenance treatment in NMOSD and MOGAD. Sankey diagram illustrating transitions from first- to second- and third-line therapies as recommended by participating neurologists for (A) AQP4-IgG positive NMOSD and (B) MOGAD. Flows represent treatment transitions, with width proportional to the weighted frequency of each pathway. The top 10 most common transitions at each step are highlighted in color; all remaining transitions are shown in grey. AZA = Azathioprine, MMF = Mycophenolate mofetil, MTX = Methotrexate, DMT = Disease-modifying therapy, aHSCT = Autologous hematopoetic stem cell transplantation, PEX = Plasma exchange.

Seventy-one percent of participants recommended follow-up measurements of MOG antibody titers. Thirty-three percent of participants recommended frequent measurement of NfL or GFAP.

3.6. De-escalation and discontinuation strategies

In stable patients with NMOSD, 35 % of participants considered de-escalating treatment after two years, while 50 % recommended to continue with the treatment. For stable patients with NMOSD under B-cell-depleting treatments, participants recommended extended dose intervals and/or reduced doses guided by monitoring of CD19-positive B-cells (33 %), monitoring of CD19-CD27-positive B-cells (33 %) or with pre-planned extended dose intervals and/or reduced doses irrespective of blood controls (33 %). One participant (5 %) indicated to take regular measurements of NfL and GFAP into account for guidance of dose intervals. 19 % preferred switching to another medication as de-

escalation strategy.

As de-escalation strategies in patients under satralizumab or tocilizumab, 29 % of participants recommended to use extended dose intervals, 24 % recommended switching from intravenous to subcutaneous administration and considered extended dose intervals later and 19 % preferred to switch to another medication (the rest would not recommend de-escelation or does not use anti-IL6-treatment). Only one participant considered using extended dose intervals in stable patients under eculizumab or ravulizumab, while 40 % would switch to another medication for de-escalation (the rest would not recommend de-escelation or does not use anti-C5 treatment).

Seventy-six percent of participants advised against treatment discontinuation in stable patients with NMOSD after five years, regardless of antibody status at follow up. Five percent would consider treatment discontinuation after five years, if AQP4-IgG titer becomes negative. 9.5 % would consider treatment discontinuation if the patient had no history of severe relapses (Fig. 5A).

In patients with MOGAD, 90 % of participants consider treatment de-escalation in stable patients after two years, if the MOG antibody titer becomes negative, while 10 % have no defined strategy yet. If MOG-antibody titer remains positive, only 43 % advise for treatment de-escalation, while another 43 % would not consider de-escalation and 14 % have no defined strategy yet.

In stable patients with MOGAD receiving CD20-depleting treatment, 47.6 % of participants adjust their regimen by extending dose intervals and/or reducing doses based on CD19-positive B-cell monitoring, while 24 % use CD19-CD27-positive B-cell monitoiring. Thirty eight percent use pre-planned extended dose intervals and/or dose reduction. Meanwhile, 28.6 % opt to switch to another medication, and 9.5 % adjust dosing based on GFAP and NfL level monitoring. For de-escalation under anti-IL-6-treatment, 33.3 % first switch to a subcutaneous administration and later consider extended dose intervals, 19 % continue with intravenous adminstration with extended dose intervals, 19 % switch to another medication, and 4.8 % use other strategies. Notably, 28.6 % never de-escalate or do not use anti-IL-6-treatment at all.

A majority of participants (76 %) would consider treatment discontinuation in stable patients with MOGAD after five years if MOG antibody were undetectable regardless of severity of past attacks, while 19 % would only consider this if the patient had no history of severe relapses (Fig. 5B). If the MOG antibody remains positive in stable patients after five years, only 29 % would consider treatment discontinuation for every patient, while 38 % would consider treatment discontinuation if the patient did not have severe relapses in the past, and 19 % would recommend continuing with the treatment (Fig. 5B).

4. Discussion

There is an ongoing debate about the optimal approach to initiating, maintaining, and long-term treatment for NMOSD and MOGAD. The results from this survey offer valuable insights into the current approaches to managing these rare conditions in a real-world setting in Switzerland.

4.1. Acute treatment

High-dose i.v. steroid treatment is usually recommended as first-line treatment for NMOSD attacks (Wingerchuk and Lucchinetti, 2022). For severe attacks, plasmapheresis has proven more effective than steroid treatment, particularly when the spinal cord is involved (Kleiter et al., 2016). It is recommended as a second-line and, in some cases, first-line treatment for acute attacks by national guidelines and NMOSD/MOGAD expert groups from different countries (Hemmer, 2023; Kumpfel et al., 2024). Evidence supporting its use in MOGAD is limited; however, a recent retrospective study found that initiating plasmapheresis early alongside disease-modifying therapy significantly increased the likelihood of full recovery (Schwake et al., 2024). In line with these findings, there was a consensus on treating acute NMOSD and MOGAD attacks with high-dose i.v. steroids among participants of our survey. For patients with residual symptoms, most experts recommended plasma exchange as a second-line treatment, with 81.1 % for NMOSD and 71.4 % for MOGAD. The use of rescue therapies like IVIG, cyclophosphamide or complement inhibition with eculizumab for attacks resistant to steroids and plasmapheresis is highly variable among the participants, reflecting the low-quality evidence for these treatment options (Lin et al., 2021; Lotan et al., 2023; San-Galli et al., 2024; Wang et al., 2020).

The recommendations regarding the use of an oral steroid taper after

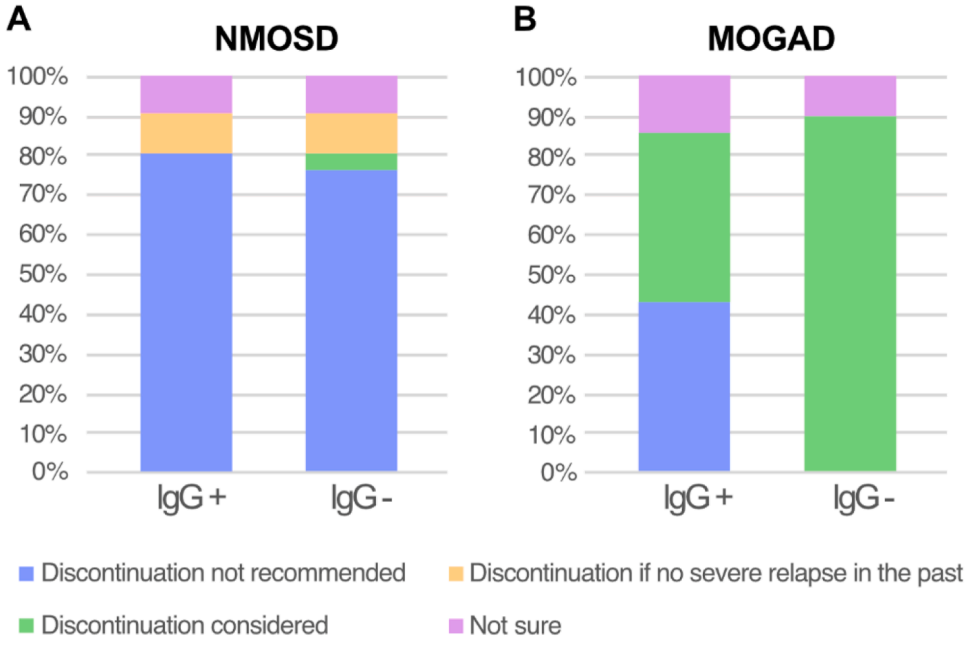

Fig. 5. Discontinuation approach for NMOSD and MOGAD. Stacked bar plots showing the percentage of participating neurologists recommending different discontinuation regimes depending on antibody positivity in NMOSD (A) and MOGAD (B). IgG + = AQP4/MOG-IgG positive at follow-up. IgG - = AQP4/MOG-IgG negative at follow-up.

attacks were also heterogenous. Interestingly, fewer participants recommended oral steroid tapering for NMOSD compared to MOGAD, despite different national guidelines and expert opinions advocating for tapering after NMOSD attacks (Hemmer, 2023; Kumpfel et al., 2024). While guidelines recommend low-dose oral glucocorticoids for three to six months (Kumpfel et al., 2024), 29 % of the participants recommended tapering after NMOSD attacks for a month or less. However, the guideline recommendations are based on expert opinions and high-quality evidence supporting the benefit and optimal duration of steroid tapering in NMOSD remains lacking. For MOGAD, more participants suggested tapering for over three months compared to NMOSD, consistent with previous studies indicating a lower probability of future relapses in patients with MOGAD treated with oral steroids for more than three months, at doses exceeding 12.5 mg of prednisone per day (Kwon et al., 2024; Trewin et al., 2024). Guideline recommendations for the use and duration of oral steroid tapering in MOGAD are not available.

4.2. Maintenance therapy and surveillance strategies

Due to the high risk of a relapsing course, start of a maintenance therapy is recommended after the first attack, especially in AQP4-IgG-positive patients (Wingerchuk and Lucchinetti, 2022). Four therapies, eculizumab, inebilizumab and satraliumab and most recently ravulizumab, have been approved for use in AQP4-IgG-positive NMOSD since 2019. Despite these clinically approved treatment options, off-label rituximab was the preferred first-choice for the majority (85 %) of respondents. Among in-label medications, most participants (40 %) indicated to prefer therapies targeting complement (eculizumab and ravulizumab). Classical immunosuppressants like azathioprine (15 %), mycophenolate mofetil (10 %) or methotrexate (5 %) were used less frequently. This aligns with European guideline recommendations, which suggest continuing classical immunosuppressive therapy in stable patients, but prioritizing antibody treatments for newly diagnosed cases (Hemmer, 2023; Kumpfel et al., 2024). As a second-line option, the newly approved antibody treatments were recommended more often than in first-line. Although participants were not asked to explain their drug choices, factors such as the high cost of newer antibodies, positive experience with rituximab in preventing NMOSD relapses, and limited long-term experience with new treatmets likely influenced these preferences.

The evidence guiding maintenance treatment selection in MOGAD is sparse. Despite recent multicenter retrospective studies questioning the efficacy of rituximab (Durozard et al., 2020; Whittam et al., 2020), it was still the most frequently recommended first-line treatment among participants. This is in line with other recent reports on immunotherapies in NMOSD and MOGAD, where rituximab remains the predominant choice for maintenance treatment in MOGAD (Haussler et al., 2024). Other recommended first-line maintenance treatment options were diverse, including periodic IVIG, azathioprine, mycophenolate mofetil and tocilizumab, each used by 33 % or fewer of the participants. In second- and third-line settings, the recommendations were even more variable, underscoring the urgent need for prospective clinical trials to establish evidence-based maintenance treatment in MOGAD.

Most participants adviced initiating maintenance treatment after a first attack only in case of a severe attack and/or persistent MOG-antibody positivity. This conservative approach appears reasonable, given that approximately 40–50 % of patients with MOGAD may experience a monophasic disease course, and treatment responses during attacks are generally more favorable compared to NMOSD patients (Cacciaguerra and Flanagan, 2024). However, data on a possible association between severe attacks or MOG-IgG persistency and relapse activity remain inconsistent (Cobo-Calvo et al., 2021; Gastaldi et al., 2023; Huda et al., 2021).

The majority of participants recommended annual MRI control with either brain MRI alone or brain and spinal MRI. This is in line with recent recommendations from some national expert groups in Europe, which propose to consider performing brain and spinal MRI not only to monitor disease activity, but also to assess potential adverse effects of treatment (Durand-Dubief et al., 2025; Kumpfel et al., 2024). However, as silent MRI activity in NMOSD and MOGAD is rare compared to individuals with multiple sclerosis (Syc-Mazurek et al., 2022), follow-up MRI in stable NMOSD/MOGAD remains controversial.

4.3. De-escalation strategies and treatment discontinuation in stable patients

Several recent studies demonstrated a significant risk of disease recurrence following immunosuppressive therapy discontinuation in NMOSD (Demuth et al., 2023; Kim et al., 2021). Accordingly, the majority of participants advised against discontinuation of immunosuppressive therapy in stable patients with NMOSD. In contrast, treatment de-escalation in stable patients with NMOSD was supported by one third of participants. These strategies included extending dosing intervals or transitioning from monoclonal antibody therapies to other immunosuppressive medications. Meanwhile, 15 % of participants expressed uncertainty regarding the appropriateness of treatment de-escalation, highlighting the lack of robust evidence on this matter.

In contrast, more participants considered treatment discontinuation or de-escalation in MOGAD. This trend aligns with recently published data suggesting that seroconversion may be associated with a reduced risk of future relapses (Gastaldi et al., 2023; Huda et al., 2021). Consequently, most participants supported treatment discontinuation or de-escalation in seroconverted patients. However, for stable patients who remained seropositive, fewer participants considered treatment de-escalation.

To date, there is a notable absence of studies adressing de-escalation strategies in NMOSD and MOGAD. Two studies investigated the efficacy of low-dose rituximab in NMOSD and reported significant effects on annual relapse rates compared to the pre-treatment period (Xiao et al., 2020; Zhao et al., 2023). A recent study showed a higher relapse risk in patients with NMOSD treated with lower-dose rituximab in extended dose intervals of nine to twelve months compared to standard intervals of six months (Chen et al., 2025). However, these studies were not designed to investigate the feasibility of a de-escalation in stable patients treated with standard dose of rituximab.

4.4. Limitations and strengths

There are several limitations of this study. The number of participants was rather small, coming from one European country. However the diversity in clinical experience, the number of patients treated by each participant and the variety of hospital settings mirror the reality faced by patients with NMOSD and MOGAD in Switzerland. Switzerland`s unique position as a multilingual country centrally located in Europe brings additional contextual influences. Medical opinions in Switzerland are shaped not only by national practices but also by those of neighbouring countries, particularly France, Germany and Italy. This cross-border influence may partially explain the heterogeneity observed in treatment strategies among survey participants, as these strategies might reflect the diverse treatment approaches used in different European language regions. Another limitation of the study origins from the survey methodology itself. The use of multiple-choice questions restricted participants' ability to explain the rationale behind their recommendations. Moreover, the results may primarily reflect the personal opinions of the participants, shaped not only by available evidence but also by their individual experiences in treating patients with NMOSD and MOGAD.

Strengths of the study include the expertise of participants, with half of them having over ten years of clinical experience in treating patients with NMOSD and MOGAD. Furthermore, the study not only addresses treatment initiation recommendations, but also reports strategies for

treatment de-escalation and discontinuation – a research area that certainly remains underexplored. These results may serve as a valuable basis for formulating national recommendations tailored to the Swiss healthcare context in the future.

5. Conclusion

In summary, this study highlighs current approaches to the treatment and surveillance of patients with NMOSD and MOGAD in Switzerland, demonstrating the realities faced by these patients. Despite the availability of new, effective medications, rituximab remains the most commonly prescribed treatment for both conditions. Among in-label treatments for NMOSD, complement inhibitors are the preferred first-line treatment option and are also considered for acute therapy in refractory cases. Moreover, this study provides guidance by considering various de-escalation scenarios in stable patients with NMOSD and MOGAD. The overall heterogeneity in recommendations underscores the need for further research on escalation treatments for acute attacks, the choice of long-term immunotherapy, and strategies for de-escalation and treatment discontinuation in patients with NMOSD and MOGAD.

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Supplementary file legend

Supplementary File 1: The original survey's single- and multiple-choice questions.

CRediT authorship contribution statement

Lukas Steinegger: Writing – review & editing, Writing – original draft, Visualization, Formal analysis, Data curation, Conceptualization. **Veronika Kana:** Writing – review & editing. **Caroline Pot:** Writing – review & editing. **Claudio Gobbi:** Writing – review & editing. **Michael Weller:** Writing – review & editing. **Patrick Roth:** Writing – review & editing. **Marie Théaudin:** Writing – review & editing, Supervision, Formal analysis, Data curation, Conceptualization. **Chiara Zecca:** Writing – review & editing, Supervision, Formal analysis, Data curation, Conceptualization. **Marina Herwerth:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

LS, CG, CP and CZ report no conflicts of interest.

VK has received honoraria for advisory roles and/or lectures for Biogen, Novartis, Merck, Roche, Teva, travel support from Biogen, Merck, Roche and an unrestricted research grant from Roche.

MW has received research grants from Novartis, Quercis and Versameb, and honoraria for lectures or advisory board participation or consulting from Anheart, Bayer, Curevac, Medac, Neurosense, Novartis, Novocure, Orbus, Pfizer, Philogen, Roche and Servier.

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MT has received honoraria for lectures or advisory board participation from Alexion, Merck, Biogen, Roche, Novartis and Sanofi and travel fees from Alexion, Merck, Biogen, Roche and Sanofi.

MH served on scientific advisory boards of Biogen, Merck Serono, Alexion, Roche and Horizon Therapeutics (Amgen), received speaker's honoraria from Biogen and received travel funding from Roche, unrelated to this study. Her institution received a research grant from Roche. She was supported by the Swiss National Science Foundation (SNSF, PZ00'3_216,616/1) and by the Olga-Mayenfisch-Foundation (2024).

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Supplementary materials

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References

Banwell, B., Bennett, J.L., Marignier, R., Kim, H.J., Brilot, F., Flanagan, E.P., Ramanathan, S., Waters, P., Tenembaum, S., Graves, J.S., Chitnis, T., Brandt, A.U., Hemingway, C., Neuteboom, R., Pandit, L., Reindl, M., Saiz, A., Sato, D.K., Rostasy, K., Paul, F., Pittock, S.J., Fujihara, K., Palace, J., 2023. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: international MOGAD Panel proposed criteria. Lancet Neurol. 22 (3), 268–282.

Budhram, A., Mirian, A., Le, C., Hosseini-Moghaddam, S.M., Sharma, M., Nicolle, M.W., 2019. Unilateral cortical FLAIR-hyperintense lesions in Anti-MOG-associated encephalitis with seizures (FLAMES): characterization of a distinct clinico-radiographic syndrome. J. Neurol. 266 (10), 2481–2487.

Cacciaguerra, L., Flanagan, E.P., 2024. Updates in NMOSD and MOGAD diagnosis and treatment: a tale of two Central nervous system autoimmune inflammatory disorders. Neurol. Clin. 42 (1), 77–114.

Chen, J.J., Flanagan, E.P., Bhatti, M.T., Jitprapaikulsan, J., Dubey, D., Lopez Chiriboga, A.S.S., Fryer, J.P., Weinshenker, B.G., McKeon, A., Tillema, J.M., Lennon, V.A., Lucchinetti, C.F., Kunchok, A., McClelland, C.M., Lee, M.S., Bennett, J. L., Pelak, V.S., Van Stavern, G., Adesina, O.O., Eggenberger, E.R., Acierno, M.D., Wingerchuk, D.M., Lam, B.L., Moss, H., Beres, S., Gilbert, A.L., Shah, V., Armstrong, G., Heidary, G., Cestari, D.M., Stiebel-Kalish, H., Pittock, S.J., 2020. Steroid-sparing maintenance immunotherapy for MOG-IgG associated disorder. Neurology 95 (2), e111–e120.

Chen, J.J., Huda, S., Hacohen, Y., Levy, M., Lotan, I., Wilf-Yarkoni, A., Stiebel-Kalish, H., Hellmann, M.A., Sotirchos, E.S., Henderson, A.D., Pittock, S.J., Bhatti, M.T., Eggenberger, E.R., Di Nome, M., Kim, H.J., Kim, S.H., Saiz, A., Paul, F., Dale, R.C., Ramanathan, S., Palace, J., Camera, V., Leite, M.I., Lam, B.L., Bennett, J.L., Mariotto, S., Hodge, D., Audoin, B., Maillart, E., Deschamps, R., Pique, J., Flanagan, E.P., Marignier, R., 2022. Association of maintenance intravenous immunoglobulin with prevention of relapse in adult myelin oligodendrocyte glycoprotein antibody-associated disease. JAMA Neurol. 79 (5), 518–525.

Chen, X., Wang, R., Li, R., Hu, S., Shi, Z., Zhou, H., 2025. A real-world study on the utility of regular rituximab treatment for neuromyelitis optica spectrum disorder. J. Neurol. 272 (3), 194.

Cobo-Calvo, A., Ruiz, A., Rollot, F., Arrambide, G., Deschamps, R., Maillart, E., Papeix, C., Audoin, B., Lepine, A.F., Maurey, H., Zephir, H., Biotti, D., Ciron, J., Durand-Dubief, F., Collongues, N., Ayrignac, X., Labauge, P., Meyer, P., Thouvenot, E., Bourre, B., Montcuquet, A., Cohen, M., Horello, P., Tintore, M., De Seze, J., Vukusic, S., Deiva, K., Marignier, R., Nomadmus, K., groups, O.s., 2021. Clinical features and risk of relapse in children and adults with myelin oligodendrocyte glycoprotein antibody-associated disease. Ann. Neurol. 89 (1), 30–41.

Cree, B.A.C., Bennett, J.L., Kim, H.J., Weinshenker, B.G., Pittock, S.J., Wingerchuk, D.M., Fujihara, K., Paul, F., Cutter, G.R., Marignier, R., Green, A.J., Aktas, O., Hartung, H. P., Lublin, F.D., Drappa, J., Barron, G., Madani, S., Ratchford, J.N., She, D., Cimbora, D., Katz, E., investigators, N.M.s., 2019. Inebilizumab for the treatment of neuromyelitis optica spectrum disorder (N-MOmentum): a double-blind, randomised placebo-controlled phase 2/3 trial. Lancet 394 (10206), 1352–1363.

Demuth, S., Collongues, N., Audoin, B., Ayrignac, X., Bourre, B., Ciron, J., Cohen, M., Deschamps, R., Durand-Dubief, F., Maillart, E., Papeix, C., Ruet, A., Zephir, H., Marignier, R., De Seze, J., Group, N.S., 2023. Rituximab de-escalation in patients with Neuromyelitis Optica spectrum disorder. Neurology 101 (4), e438–e450.

Durand-Dubief, F., Shor, N., Audoin, B., Bourre, B., Cohen, M., Kremer, S., Maillart, E., Papeix, C., Ruet, A., Savatovsky, J., Tourdias, T., Ayrignac, X., Ciron, J., Collongues, N., Laplaud, D., Michel, L., Deschamps, R., Thouvenot, E., Zephir, H., Marignier, R., Cotton, F., Group, N.S., 2025. MRI management of NMOSD and MOGAD: proposals from the French expert Group NOMADMUS. J. Neuroradiol. 52 (1), 101235.

Durozard, P., Rico, A., Boutiere, C., Maarouf, A., Lacroix, R., Cointe, S., Fritz, S., Brunet, C., Pelletier, J., Marignier, R., Audoin, B., 2020. Comparison of the response to Rituximab between myelin oligodendrocyte glycoprotein and aquaporin-4 antibody diseases. Ann. Neurol. 87 (2), 256–266.

Gastaldi, M., Foiadelli, T., Greco, G., Scaranzin, S., Rigoni, E., Masciocchi, S., Ferrari, S., Mancinelli, C., Brambilla, L., Mancardi, M., Giacomini, T., Ferraro, D., Della Corte, M., Gallo, A., Di Filippo, M., Benedetti, L., Novi, G., Versino, M., Banfi, P.,

Iorio, R., Moiola, L., Turco, E., Sartori, S., Nosadini, M., Ruggieri, M., Savasta, S., Colombo, E., Ballante, E., Jarius, S., Mariotto, S., Franciotta, D., group, N.s., 2023. Prognostic relevance of quantitative and longitudinal MOG antibody testing in patients with MOGAD: a multicentre retrospective study. J. Neurol. Neurosurg. Psychiatry 94 (3), 201–210.

Haussler, V., Trebst, C., Engels, D., Pellkofer, H., Havla, J., Duchow, A., Schindler, P., Schwake, C., Pakeerathan, T., Fischer, K., Ringelstein, M., Lindenblatt, G., Hummert, M.W., Tkachenko, D., Butow, F., Giglhuber, K., Flaskamp, M., Schiffmann, I., Korporal-Kuhnke, M., Jarius, S., Dawin, E., Revie, L., Senel, M., Herfurth, M., Walter, A., Pompsch, M., Kleiter, I., Angstwurm, K., Kaste, M., Grothe, M., Wickel, J., Rommer, P.S., Sieb, J.P., Kramer, M., Then Bergh, F., Tumani, H., Klotz, L., Wildemann, B., Aktas, O., Ayzenberg, I., Bellmann-Strobl, J., Paul, F., Kumpfel, T., Friede, T., Berthele, A., Stellmann, J.P., Neuromyelitis optica study, g., 2024. Real-world multicentre cohort study on choices and effectiveness of immunotherapies in NMOSD and MOGAD. J. Neurol. Neurosurg. Psychiatry.

Hemmer, B.e.a., 2023. Diagnose und therapie der multiplen sklerose, neuromyelitis-optica-spektrum-Erkrankungen und MOG-IgG-assoziierten Erkrankungen, S2k-Leitlinie, 2023. Deutsche Gesellschaft für Neurologie (Hrsg.). Leitlinien für Diagnostik und Therapie in der Neurologie. https://dnvp9c1uo2095.cloudfront.net/cms-content/030050_living_Guideline_MS_V7.1_240105_1704444034393.pdf (Accessed 01/12 2025).

Huda, S., Whittam, D., Jackson, R., Karthikeayan, V., Kelly, P., Linaker, S., Mutch, K., Kneen, R., Woodhall, M., Murray, K., Hunt, D., Waters, P., Jacob, A., 2021. Predictors of relapse in MOG antibody associated disease: a cohort study. BMJ Open 11 (11), e055392.

Ishikura, T., Okuno, T., Takahashi, T., Mochizuki, H., 2022. Myelin oligodendrocyte glycoprotein antibody-associated disease with frequent cerebellitis. Intern. Med. 61 (23), 3629–3630.

Jarius, S., Aktas, O., Ayzenberg, I., Bellmann-Strobl, J., Berthele, A., Giglhuber, K., Haussler, V., Havla, J., Hellwig, K., Hummert, M.W., Kleiter, I., Klotz, L., Krumbholz, M., Kumpfel, T., Paul, F., Ringelstein, M., Ruprecht, K., Senel, M., Stellmann, J.P., Bergh, F.T., Tumani, H., Wildemann, B., Trebst, C., Neuromyelitis Optica Study, G., 2023. Update on the diagnosis and treatment of neuromyelits optica spectrum disorders (NMOSD) - revised recommendations of the Neuromyelitis Optica Study Group (NEMOS). Part I: diagnosis and differential diagnosis. J. Neurol. 270 (7), 3341–3368.

Kim, S.H., Jang, H., Park, N.Y., Kim, Y., Kim, S.Y., Lee, M.Y., Hyun, J.W., Kim, H.J., 2021. Discontinuation of immunosuppressive therapy in patients with Neuromyelitis Optica spectrum disorder with aquaporin-4 antibodies. Neurol. Neuroimmunol. Neuroinflamm. 8 (2).

Kleiter, I., Gahlen, A., Borisow, N., Fischer, K., Wernecke, K.D., Wegner, B., Hellwig, K., Pache, F., Ruprecht, K., Havla, J., Krumbholz, M., Kumpfel, T., Aktas, O., Hartung, H.P., Ringelstein, M., Geis, C., Kleinschnitz, C., Berthele, A., Hemmer, B., Angstwurm, K., Stellmann, J.P., Schuster, S., Stangel, M., Lauda, F., Tumani, H., Mayer, C., Zeltner, L., Ziemann, U., Linker, R., Schwab, M., Marziniak, M., Then Bergh, F., Hofstadt-van Oy, U., Neuhaus, O., Winkelmann, A., Marouf, W., Faiss, J., Wildemann, B., Paul, F., Jarius, S., Trebst, C., Neuromyelitis Optica Study, G., 2016. Neuromyelitis optica: evaluation of 871 attacks and 1153 treatment courses. Ann. Neurol. 79 (2), 206–216.

Kluyver, T., Ragan-Kelley, B., et al., 2016. Jupyter Notebooks – A Publishing Format for Reproducible Computational Workflows. Positioning and Power in Academic Publishing: Players, Agents and Agendas, pp. 87–90.

Kumpfel, T., Giglhuber, K., Aktas, O., Ayzenberg, I., Bellmann-Strobl, J., Haussler, V., Havla, J., Hellwig, K., Hummert, M.W., Jarius, S., Kleiter, I., Klotz, L., Krumbholz, M., Paul, F., Ringelstein, M., Ruprecht, K., Senel, M., Stellmann, J.P., Bergh, F.T., Trebst, C., Tumani, H., Warnke, C., Wildemann, B., Berthele, A., Neuromyelitis Optica Study, G., 2024. Update on the diagnosis and treatment of neuromyelitis optica spectrum disorders (NMOSD) - revised recommendations of the Neuromyelitis Optica Study Group (NEMOS). Part II: attack therapy and long-term management. J. Neurol. 271 (1), 141–176.

Kwon, Y.N., Kim, B., Kim, J.S., Park, K.S., Seo, D.Y., Kim, H., Lee, E.J., Lim, Y.M., Ju, H., Chung, Y.H., Min, J.H., Nam, T.S., Kim, S., Sohn, E., Shin, K.J., Seok, J.M., Kim, S., Bae, J.S., Lee, S., Oh, S.I., Jung, Y.J., Park, J., Kim, S.H., Kim, K.H., Kim, H.J., Jung, J.H., Kim, S.J., Kim, S.W., Jang, M.J., Sung, J.J., Waters, P., Shin, H.Y., Kim, S. M., 2024. Time to treat first acute attack of myelin oligodendrocyte glycoprotein antibody-associated disease. JAMA Neurol. 81 (10), 1073–1084.

Lennon, V.A., Kryzer, T.J., Pittock, S.J., Verkman, A.S., Hinson, S.R., 2005. IgG marker of optic-spinal multiple sclerosis binds to the aquaporin-4 water channel. J. Exp. Med. 202 (4), 473–477.

Lin, J., Xue, B., Zhu, R., Pan, J., Li, J., Lin, Y., Li, X., Xia, J., 2021. Intravenous immunoglobulin as the rescue treatment in NMOSD patients. Neurol. Sci. 42 (9), 3857–3863.

Lotan, I., Chen, J.J., Hacohen, Y., Abdel-Mannan, O., Mariotto, S., Huda, S., Gibbons, E., Wilf-Yarkoni, A., Hellmann, M.A., Stiebel-Kalish, H., Pittock, S.J., Flanagan, E.P., Molazadeh, N., Anderson, M., Salky, R., Romanow, G., Schindler, P., Duchow, A.S., Paul, F., Levy, M., 2023. Intravenous immunoglobulin treatment for acute attacks in myelin oligodendrocyte glycoprotein antibody disease. Mult. Scler. 29 (9), 1080–1089.

McKinney, W., 2010. Data structures for statistical computing in Python. In: Proceedings of the 9th Python in Science Conference, pp. 51–56.

Pittock, S.J., Barnett, M., Bennett, J.L., Berthele, A., de Seze, J., Levy, M., Nakashima, I., Oreja-Guevara, C., Palace, J., Paul, F., Pozzilli, C., Yountz, M., Allen, K., Mashhoon, Y., Kim, H.J., 2023. Ravulizumab in Aquaporin-4-positive neuromyelitis optica spectrum disorder. Ann. Neurol. 93 (6), 1053–1068.

Pittock, S.J., Berthele, A., Fujihara, K., Kim, H.J., Levy, M., Palace, J., Nakashima, I., Terzi, M., Totolyan, N., Viswanathan, S., Wang, K.C., Pace, A., Fujita, K.P., Armstrong, R., Wingerchuk, D.M., 2019. Eculizumab in Aquaporin-4-positive Neuromyelitis Optica spectrum disorder. N. Engl. J. Med. 381 (7), 614–625.

PlotlyTechnologiesInc, 2015. Collaborative data Science. https://plot.ly.

Ringelstein, M., Ayzenberg, I., Lindenblatt, G., Fischer, K., Gahlen, A., Novi, G., Hayward-Konnecke, H., Schippling, S., Rommer, P.S., Kornek, B., Zrzavy, T., Biotti, D., Ciron, J., Audoin, B., Berthele, A., Giglhuber, K., Zephir, H., Kumpfel, T., Berger, R., Rother, J., Haussler, V., Stellmann, J.P., Whittam, D., Jacob, A., Kraemer, M., Gueguen, A., Deschamps, R., Bayas, A., Hummert, M.W., Trebst, C., Haarmann, A., Jarius, S., Wildemann, B., Grothe, M., Siebert, N., Ruprecht, K., Paul, F., Collongues, N., Marignier, R., Levy, M., Karenfort, M., Deppe, M., Albrecht, P., Hellwig, K., Gold, R., Hartung, H.P., Meuth, S.G., Kleiter, I., Aktas, O., Neuromyelitis Optica Study, G., 2022. Interleukin-6 receptor blockade in treatment-refractory MOG-IgG-associated disease and neuromyelitis Optica spectrum disorders. Neurol. Neuroimmunol. Neuroinflamm. 9 (1).

San-Galli, A., Chaumont, H., Bourgeois, Q., Roge, J., Lobjois, Q., Cabre, P., 2024. Eculizumab as rescue therapy in a context of dramatic NMOSD attack: report of two cases. Rev. Neurol. 180 (10), 995–997.

Schwake, C., Ladopoulos, T., Haussler, V., Kleiter, I., Ringelstein, M., Aktas, O., Kumpfel, T., Engels, D., Havla, J., Hummert, M.W., Kretschmer, J.R., Tkachenko, D., Trebst, C., Ayroza Galvao Ribeiro Gomes, A.B., Probstel, A.K., Korporal-Kuhnke, M., Wildemann, B., Jarius, S., Pul, R., Pompsch, M., Kramer, M., Then Bergh, F., Godel, C., Schwarz, P., Kowarik, M.C., Rommer, P.S., Vardakas, I., Senel, M., Winkelmann, A., Retzlaff, N., Weber, M.S., Husseini, L., Walter, A., Schindler, P., Bellmann-Strobl, J., Paul, F., Gold, R., Ayzenberg, I., Neuromyelitis Optica Study, G., 2024. Apheresis therapies in MOGAD: a retrospective study of 117 therapeutic interventions in 571 attacks. J. Neurol. Neurosurg. Psychiatry.

Sechi, E., Cacciaguerra, L., Chen, J.J., Mariotto, S., Fadda, G., Dinoto, A., Lopez-Chiriboga, A.S., Pittock, S.J., Flanagan, E.P., 2022. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): a review of clinical and MRI features, diagnosis, and management. Front. Neurol. 13, 885218.

Skarsta, L., Nicoletti, T., Frick, K., Kana, V., De Vere-Tyndall, A., Weller, M., Roth, P., Herwerth, M., 2023. Acute haemorrhagic leucoencephalitis as clinical manifestation of MOG antibody-associated disease. J. Neurol. Neurosurg. Psychiatry 94 (7), 583–585.

Syc-Mazurek, S.B., Chen, J.J., Morris, P., Sechi, E., Mandrekar, J., Tillema, J.M., Lopez-Chiriboga, A.S., Lucchinetti, C.F., Zalewski, N., Cacciaguerra, L., Buciuc, M., Krecke, K.N., Messina, S.A., Bhatti, M.T., Pittock, S.J., Flanagan, E.P., 2022. Frequency of new or enlarging lesions on MRI outside of clinical attacks in patients with MOG-antibody-associated disease. Neurology 99 (18), 795–799.

Takai, Y., Kuroda, H., Misu, T., Akaishi, T., Nakashima, I., Takahashi, T., Nishiyama, S., Fujihara, K., Aoki, M., 2021. Optimal management of neuromyelitis optica spectrum disorder with aquaporin-4 antibody by oral prednisolone maintenance therapy. Mult. Scler. Relat. Disord. 49, 102750.

Traboulsee, A., Greenberg, B.M., Bennett, J.L., Szczechowski, L., Fox, E., Shkrobot, S., Yamamura, T., Terada, Y., Kawata, Y., Wright, P., Gianella-Borradori, A., Garren, H., Weinshenker, B.G., 2020. Safety and efficacy of satralizumab monotherapy in neuromyelitis optica spectrum disorder: a randomised, double-blind, multicentre, placebo-controlled phase 3 trial. Lancet Neurol. 19 (5), 402–412.

Trewin, B.P., Dale, R.C., Qiu, J., Chu, M., Jeyakumar, N., Dela Cruz, F., Andersen, J., Siriratnam, P., Ma, K.K.M., Hardy, T.A., van der Walt, A., Lechner-Scott, J., Butzkueven, H., Broadley, S.A., Barnett, M.H., Reddel, S.W., Brilot, F., Kalincik, T., Ramanathan, S., Australasian, M.S.G., 2024. Oral corticosteroid dosage and taper duration at onset in myelin oligodendrocyte glycoprotein antibody-associated disease influences time to first relapse. J. Neurol. Neurosurg. Psychiatry. 95 (11), 1054–1063.

Wang, L., Liu, K., Tan, X., Zhou, L., Zhang, Y., Liu, X., Fu, Y., Qiu, W., Yang, H., 2020. Remedial effect of intravenous cyclophosphamide in corticosteroid-refractory patients in the acute phase of Neuromyelitis Optica spectrum disorder-related Optic neuritis. Front. Neurol. 11, 612097.

Whittam, D.H., Cobo-Calvo, A., Lopez-Chiriboga, A.S., Pardo, S., Gornall, M., Cicconi, S., Brandt, A., Berek, K., Berger, T., Jelcic, I., Gombolay, G., Oliveira, L.M., Callegaro, D., Kaneko, K., Misu, T., Capobianco, M., Gibbons, E., Karthikeayan, V., Brochet, B., Audoin, B., Mathey, G., Laplaud, D., Thouvenot, E., Cohen, M., Tourbah, A., Maillart, E., Ciron, J., Deschamps, R., Biotti, D., Rostasy, K., Neuteboom, R., Hemingway, C., Forsyth, R., Matiello, M., Webb, S., Hunt, D., Murray, K., Hacohen, Y., Lim, M., Leite, M.I., Palace, J., Solomon, T., Lutterotti, A., Fujihara, K., Nakashima, I., Bennett, J.L., Pandit, L., Chitnis, T., Weinshenker, B.G., Wildemann, B., Sato, D.K., Kim, S.H., Huda, S., Kim, H.J., Reindl, M., Levy, M., Jarius, S., Tenembaum, S., Paul, F., Pittock, S., Marignier, R., Jacob, A., 2020. Treatment of MOG-IgG-associated disorder with rituximab: an international study of 121 patients. Mult. Scler. Relat. Disord. 44, 102251.

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, N.M.O.D., 2015. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology 85 (2), 177–189.

Wingerchuk, D.M., Lucchinetti, C.F., 2022. Neuromyelitis optica spectrum disorder. N. Engl. J. Med. 387 (7), 631–639.

Xiao, H., Zeng, W., Li, L., Li, L., Cui, Y., Wang, J., Ye, J., Yang, Q., 2020. Retrospective observation of low-dose rituximab treatment in Chinese patients with neuromyelitis optica spectrum disorders in a real-world setting. Front. Neurol. 11, 642.
Yamamura, T., Kleiter, I., Fujihara, K., Palace, J., Greenberg, B., Zakrzewska-Pniewska, B., Patti, F., Tsai, C.P., Saiz, A., Yamazaki, H., Kawata, Y., Wright, P., De Seze, J., 2019. Trial of Satralizumab in Neuromyelitis Optica spectrum disorder. N. Engl. J. Med. 381 (22), 2114–2124.
Zhao, D., Ren, K., Lu, J., Liu, Z., Li, Z., Wu, J., Xu, Z., Wu, S., Lei, T., Ma, C., Zhao, S., Bai, M., Li, H., Guo, J., 2023. Rituximab at lower dose for neuromyelitis optica spectrum disorder: a multicenter, open-label, self-controlled, prospective follow-up study. Front. Immunol. 14, 1148632.