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Apheresis therapies in MOGAD: a retrospective study of 117 therapeutic interventions in 571 attacks

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ABSTRACT

Background Incomplete attack remission is the main cause of disability in myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD). Apheresis therapies such as plasma exchange and immunoadsorption are widely used in neuroimmunology. Data on apheresis outcomes in MOGAD attacks remain limited.

Methods We retrospectively evaluated all apheresis treated attacks occurring in patients with MOGAD between 2008 and 2023 at 18 Neuromyelitis Optica Study Group centres. Treatment response was categorised as complete, partial or no remission. Preattack and follow-up Expanded Disability Status Scale (EDSS) and visual Functional System Scores (FSS) were used to calculate absolute outcomes (ΔEDSS/ΔvisualFSS). Predictors of complete remission were analysed using a generalised linear mixed model.

Results Apheresis was used for 117/571 (20.5%) attacks in 85/209 (40.7%) patients. Attacks with simultaneous optic neuritis and myelitis were treated more often with apheresis (42.4%, n=14) than isolated myelitis (25.2%, n=35), cerebral manifestation (21.0%, n=17) or isolated optic neuritis (17.6%, n=51). Apheresis was initiated as first-line therapy in 12% (4.5 (IQR 0–11) days after attack onset), second-line therapy in 62% (15 (IQR 6.75–31) days) and third-line therapy in 26% (30 (IQR 19–42) days). Complete remission was achieved in 21%, partial remission in 70% and no remission in 9% of patients. First-line apheresis (OR 2.5, p=0.040) and concomitant disease-modifying therapy (OR 1.5, p=0.011) were associated with complete remission. Both parameters were also associated with a favourable ΔEDSS. No differences in outcomes were observed between the apheresis types.

Conclusion Apheresis is frequently used in MOGAD attacks. An early start as first-line therapy and concomitant disease-modifying therapy predict full attack recovery.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Apheresis therapies are effective for treating disease attacks in multiple sclerosis and neuromyelitis optica spectrum disorder, with early intervention being crucial for better outcomes; however, data in myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) are limited.

WHAT THIS STUDY ADDS

⇒ This multicentre study demonstrates that apheresis therapies are commonly used in MOGAD attacks and identified early first-line apheresis therapy and prior disease-modifying treatment as two significant predictors of full attack recovery.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study highlights the importance of early initiation of apheresis therapy and the use of disease-modifying treatments in patients with MOGAD with recurrent disease attacks.

INTRODUCTION

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a rare autoimmune-inflammatory disease of the central nervous system (CNS). Despite a partial overlap in its clinical presentation with multiple sclerosis (MS) and aquaporin-4-IgG positive (AQP4-IgG$^+$) neuromyelitis optica spectrum disorder (NMOSD), there are significant pathophysiological and clinical differences between these entities.[1]

In contrast to MS, the accrual of disability in MOGAD is predominantly linked to disease attacks.[2] Although patients recover significantly

better from attacks than patients with AQP4-IgG$^+$ NMOSD, relevant residual deficits are still common.[3] Persistent visual impairment after optic neuritis (ON) has been reported in 7–24%,[4–6] impaired ambulation in 14–25%[6 7] and bladder dysfunction due to myelitis (MY) in 20–59% of patients with MOGAD.[4 5] Cerebral manifestations are also possible and may result in long-term cognitive impairment, especially in patients with acute disseminated encephalomyelitis.[8 9] Thus, prevention and optimal treatment of MOGAD attacks are crucial for preventing disability. While intravenous methylprednisolone (IVMP) is generally effective, some attacks remain corticosteroid refractory and require apheresis.[10]

Apheresis procedures such as plasma exchange (PLEX) and immunoadsorption (IA) have been reported to be highly effective in NMOSD[11 12] and MS.[13] In NMOSD, the very early use of apheresis (immediately after the onset of the attack) is considered even more critical, as previous studies have shown that early intervention significantly influences the clinical outcome.[11 14] Thus, the American Society for Apheresis strongly recommends PLEX (grade 1B) and IA (grade 1C) for the treatment of NMOSD attacks.[15] Moreover, both treatment methods are included in the treatment guidelines of the Neuromyelitis Optica Study Group (NEMOS) and Guidelines of the German Neurological Society.[16]

Despite several studies on apheresis in MS and NMOSD,[11 17–20] data on clinical outcomes after apheresis in acute MOGAD attacks remain lacking. Therefore, this study aimed (a) to describe clinical characteristics of attacks treated with apheresis in a large multicentre MOGAD cohort and (b) to identify variables predicting favourable post-apheresis outcomes.

MATERIALS AND METHODS

Patients with MOGAD were identified through the NEMOS registry (209 patients with 571 attacks) and all 85 patients who underwent PLEX and/or IA between January 2008 and December 2023 at 18 specialised centres in Germany, Switzerland and Austria were included in this retrospective, multicentre, cross-sectional study (figure 1). MOGAD was diagnosed at the respective centres with MOG-IgG testing by cell-based assays (CBA) at the discretion of each centre using the laboratory's

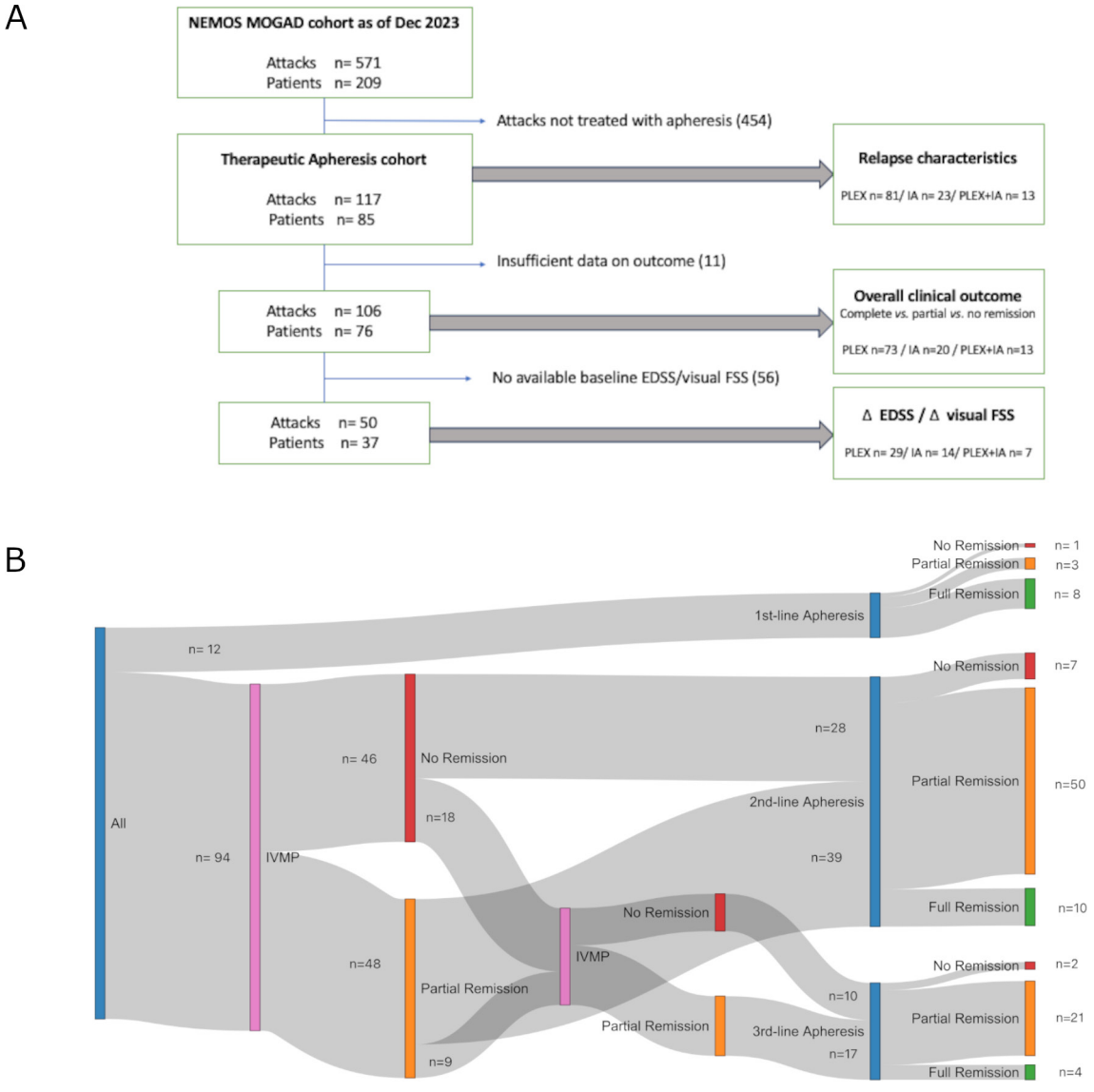

Figure 1 Study flow chart (A). Patient trajectories across attack treatments (n=106) (B). EDSS, Expanded Disability Status Scale; FSS, Functional System Score; IA, immunoadsorption; IVMP, intravenous methylprednisolone; MOGAD, myelin oligodendrocyte glycoprotein antibody-associated disease; NEMOS, Neuromyelitis Optica Study Group; PLEX, plasma exchange.

cut-offs (EUROIMMUN, Lübeck, Germany, fixed CBA, ≥1:10; Medical University of Innsbruck, Innsbruck, Austria, live CBA, ≥1:160; Labor Krone, Bad Salzuflen, Germany, live CBA, ≥1:160; Research Center for Neuroimmunology and Neuroscience, University of Basel, Basel, Switzerland, live CBA, flow cytometry, mean fluorescence intensity ≥3.0).

Demographic, clinical and apheresis-related data were retrieved from the NEMOS registry and supplemented through an experienced physician at the according site via review of medical records between March and October 2023.

This study followed the reporting guideline 'Strengthening the Reporting of Observational Studies in Epidemiology'.

Main outcome and measures

Characteristics of all patients with MOGAD treated with PLEX and/or IA were analysed. Where available, clinical outcomes were further investigated to identify factors associated with a favourable post-apheresis result.

The clinical outcome was categorised into three groups[12]: (1) complete remission (disappearance of attack symptoms compared with baseline), (2) partial remission (gain in function without reaching the baseline) or (3) no remission (no neurological recovery).

If detailed clinical data on functional scores from the last study visit preceding and the first follow-up (FU) visit after the attack were accessible, we calculated the absolute clinical outcome using the following equation: *Δvisual FSS or ΔEDSS=Follow-up score–baseline score*. As the overall Expanded Disability Status Scale (EDSS) might not sufficiently represent visual impairments, we specifically assessed Δvisual Functional System Score (FSS) in the ON subgroup.

Statistical testing

Statistical analyses were performed using SPSS V.29.0 (IBM) and R (V.4.4.0) with a significance threshold set at $p<0.05$. Continuous variables were compared using non-parametric statistical tests (Mann-Whitney U test for two groups, Kruskal-Wallis test for more than two groups). Correlation analysis was conducted using Spearman correlation. The χ^2 test was used to assess the association between two categorical variables.

Predictors of a favourable clinical outcome were identified through multivariate analysis using a generalised linear mixed model (GLMM). This model was employed to accommodate patients who underwent multiple apheresis therapies during their lifetime. Age, gender, line of therapy, type of apheresis, disease duration, disease-modifying therapy (DMT; yes/no), attack phenotype (ON/MY) and dosage of previous IVMP were defined as independent variables. Out of 106 attacks with known outcomes, 100 had complete data for all variables in the GLMM. A complete case analysis was implemented for the GLMM to ensure the robustness of our findings while minimising potential biases.

RESULTS

Cohort characteristics

209 patients of the whole NEMOS MOGAD cohort (female:male=130:79, median age at disease onset 33.5 (IQR 23.0–46.0) years) experienced 571 attacks (ON: n=289, MY: n=139, cerebral manifestation: n=81, simultaneous ON+MY: n=33, other: n=29) during a median disease duration of 5.8 years (IQR 3.2–11.1). Among these, 117 (20.5%) attacks in 85 (40.7%) patients were treated with apheresis (table 1). 80 of 85 patients (94%) fulfilled the recently proposed international MOGAD diagnostic criteria (supplemental table 1).[21]

Table 1 Patient characteristics (n=209)

	Apheresis (n=85)	Non-apheresis (n=124)	P value
Female sex, n (%)	51/85 (60.0)	79/124 (63.7)	0.587*
Autoimmune comorbidities	12/72 (17.0%)	14/119 (11.3%)	0.378*
Ethnicity	**n=80**	**n=110**	0.846*
White	74/80 (93.0%)	104/110 (94.5%)	
Asian	1/80 (1.0%)	2/110 (1.8%)	
Arabic	4/80 (5.0%)	3/110 (2.4%)	
Hispanic (America)	1/80 (1.0%)	1/110 (0.8%)	
Clinical characteristics			
Age at disease onset, year, median (IQR)	34 (22.0–49.0) (n=83)	33 (23.0–43.0) (n=118)	0.254†
Apheresis at first attack, n (%)	50/85 (58.8)	–	
Age at apheresis, year, median (IQR)	38 (26.3–50.0) (n=80)	–	
Disease duration, year, median (IQR)	6.0 (3.7–9.9) (n=84)	5.5 (2.9–12.7) (n=118)	0.747†
Overall number of attacks	297 (n=85)	274 (n=118)	
Number of attacks treated with apheresis	117 (n=85)	–	
ARR until last FU, median (IQR)	0.4 (0.2–0.9) (n=84)	0.3 (0.2–0.6) (n=118)	0.109†
EDSS at last FU	2 (1.4–4.0) (n=82)	1.5 (1.0–2.5) (n=113)	<0.001†

*X^2 test.
†Mann-Whitney U test.
ARR, annual relapse rate; EDSS, Expanded Disability Status Scale; FU, follow-up.

Patient gender, ethnicity, occurrence of autoimmune comorbidities, age at disease onset, disease duration and annual relapse rate (ARR) at last FU did not differ between the non-apheresis and the apheresis cohort. However, the median EDSS at the last FU visit was significantly lower in the non-apheresis than in the apheresis group (1.5 vs 2.0, $p<0.001$) (table 1).

Apheresis was initiated most frequently in multifocal attacks with simultaneous ON+MY (14/33, 42.4%), followed by MY (35/139, 25.2%), cerebral manifestation (17/81, 21.0%) and ON (51/289, 17.6%). 69 patients were treated with apheresis therapy once and further 16 patients for multiple (median 2.5 (IQR 2.0–5.0)) attacks.

81 attacks (69%) were treated with PLEX (median 5.0 (IQR 5.0–7.0) sessions), 23 (20%) with IA (median 6.0 (IQR 5.0–7.0) sessions) and 13 (11%) with a combination of PLEX and IA (median 2.0 (IQR 1.5–5.0) PLEX sessions; median 3.0 (2.5–4.5) IA sessions). Apheresis was administered as first-line therapy in 14 (12%), second-line therapy in 73 (62%) and third-line therapy in 30 (26%) attacks.

Among the 14 attacks receiving apheresis as first-line therapy, only two occurred at disease onset. All other first-line treated attacks occurred in patients already diagnosed with MOGAD; four of whom had a history of apheresis treatment.

The remaining 103 out of 117 attacks were initially treated with IVMP (cumulative median corticosteroid dose 5000 (IQR 5000–10 000) mg). The majority received one IVMP pulse (71% second-line apheresis, median corticosteroid dose 5000 (IQR 5000–5000) mg), while the rest received two IVMP pulses (29%

third-line apheresis, median corticosteroid dose 13 000 (IQR 9000–15 000) mg).

Only two attacks were treated with a combination of IVMP and intravenous immunoglobulins (125 and 145 g) before undergoing apheresis. 77/117 (65.8%) attacks occurred without concomitant DMT use and of these 50 (64.9%) at disease onset. Among patients with DMT use, B-cell depletion therapy was present in 17/117 attacks (14.5%), anti-interleukin-6 receptor therapy in 2 attacks (1.7%), azathioprine in 8 attacks (6.8%), mycophenolate mofetil in 4 attacks (3.4%) and a total of 9 attacks (7.7%) occurred during therapy with other MS-typical DMTs.

Clinical outcomes following apheresis therapy

The overall clinical outcome was available in 106/117 attacks (47 ON, 32 MY, 13 ON+MY, 14 cerebral manifestation) of 76 patients (female:male=45:31, median age at apheresis 38.0 (IQR 27.0–50.0) years) which were treated with 73 PLEX, 20 IA and 13 combined PLEX/IA procedures. Complete remission was achieved in 22 patients (21%), most patients improved partially (70%, n=74) and almost one in 10 patients did not improve at all (9%, n=10).

We could not identify any difference in remission status among the three types of apheresis (p=0.161, n=106), nor when comparing PLEX versus IA separately (p=0.315, n=93). There was no difference in remission status according to attack phenotypes (p=0.360) or focal/multifocal symptom manifestations (p=0.693). However, complete remission occurred significantly more often in patients with concomitant use of DMT (p=0.003) as well as after first-line apheresis (n=8/12, 67%, p=0.001) compared with second-line (n=10/67, 15%) or third-line (n=4/27, 15%) apheresis (figure 2). The delay to apheresis was significantly shorter in first-line (4.5 (IQR 0–11) days) than in second-line (15 (IQR 7–31) days, p<0.001) or third-line (30 (IQR 19–42) days, p<0.001) therapy. Apheresis was revealed to be most effective if started within 2 days of attack onset, with complete remission rates dropping radically afterwards (figure 3).

A multivariate GLMM analysis confirmed an association between the use of apheresis as first-line therapy and prophylactic DMT with a good response to apheresis therapy defined as complete remission (table 2).

Additional detailed responses could be calculated in 47% of attacks (50/106). We found no difference in the △EDSS between PLEX and IA+PLEX (n=29 vs 21; △2 (IQR 0.5–4.0) vs △1.5 (IQR 0–2.3), p=0.243) or focal and multifocal attacks (n=43 vs 7; △1.5 (IQR 0–3.5) vs △1.5 (0.5–5.0), p=0.989). The difference between relapse phenotypes was not significant (n=18 ON △0.75 (IQR 0–2) vs n=7 ON/MY △1.5 (0.5–3.5) vs n=17 MY △2.5 (1.5–4.25) vs n=8 cerebral manifestations △0.75 (−0.375 to 4.25), p=0.059).

However, patients with concomitant DMT had a significant lower △EDSS than those without DMT (n=20 vs 30, △0 (IQR 0–0.9) vs △2.5 (IQR 1.5–4.0), p<0.001). It was also significantly lower in first-line treated patients than in the rest of the cohort (n=7 vs 43, △0 (IQR 1–1.5) vs △2 (IQR 0–4.0), p=0.023). Neither patient age (p=0.719) nor apheresis delay correlated with the △EDSS (p=0.542).

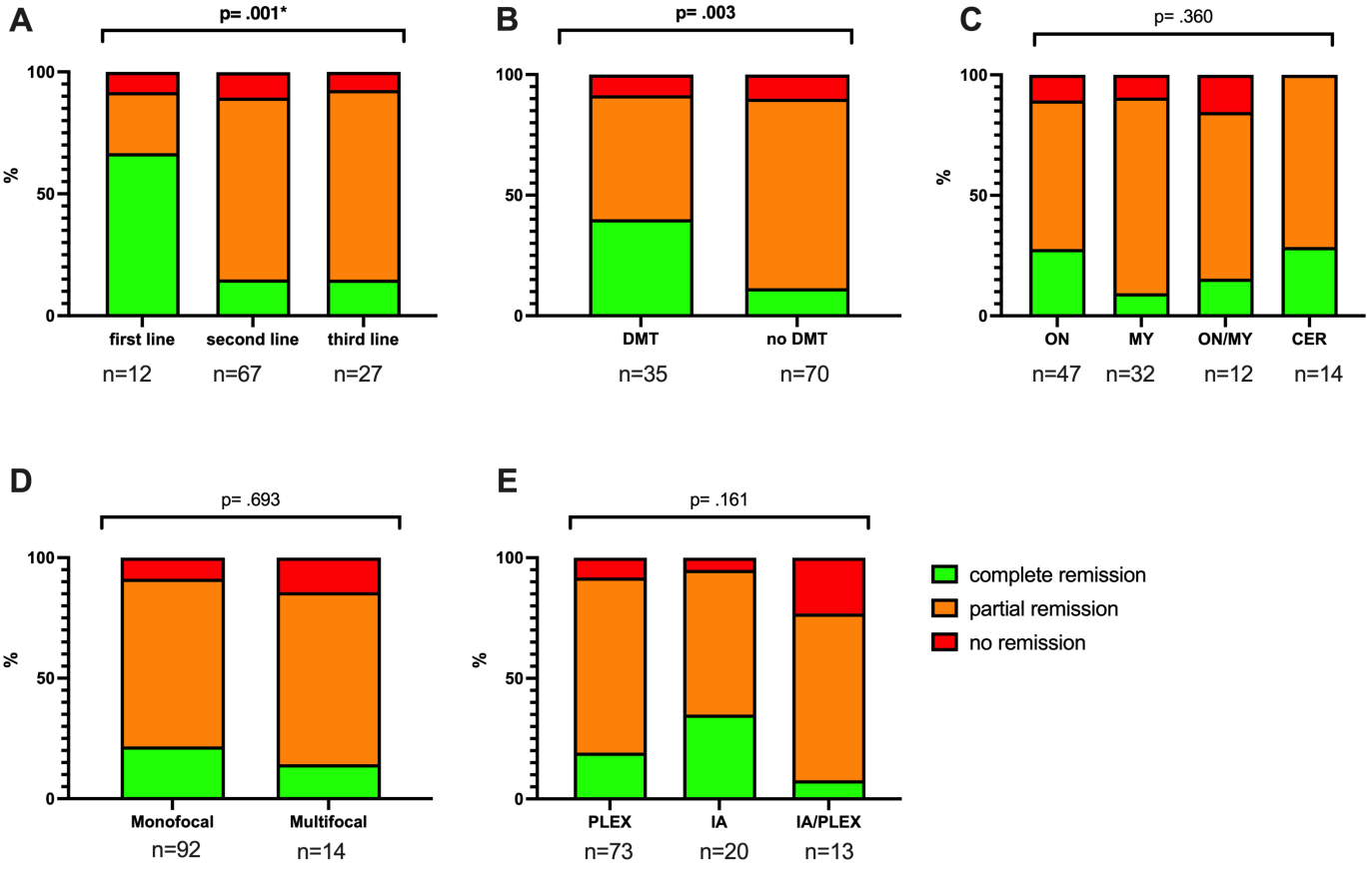

Figure 2 Overall clinical outcome in n=106 myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) attacks according to therapy line (A), DMT use (B), different attack phenotypes (C), the distribution of symptoms (D) and type of apheresis (E). X^2 test. Significant values (p<0.05) are shown in bold. CER, cerebral manifestation; DMT, disease-modifying therapy; IA, immunoadsorption; MY, myelitis; ON, optic neuritis; PLEX, plasma exchange.

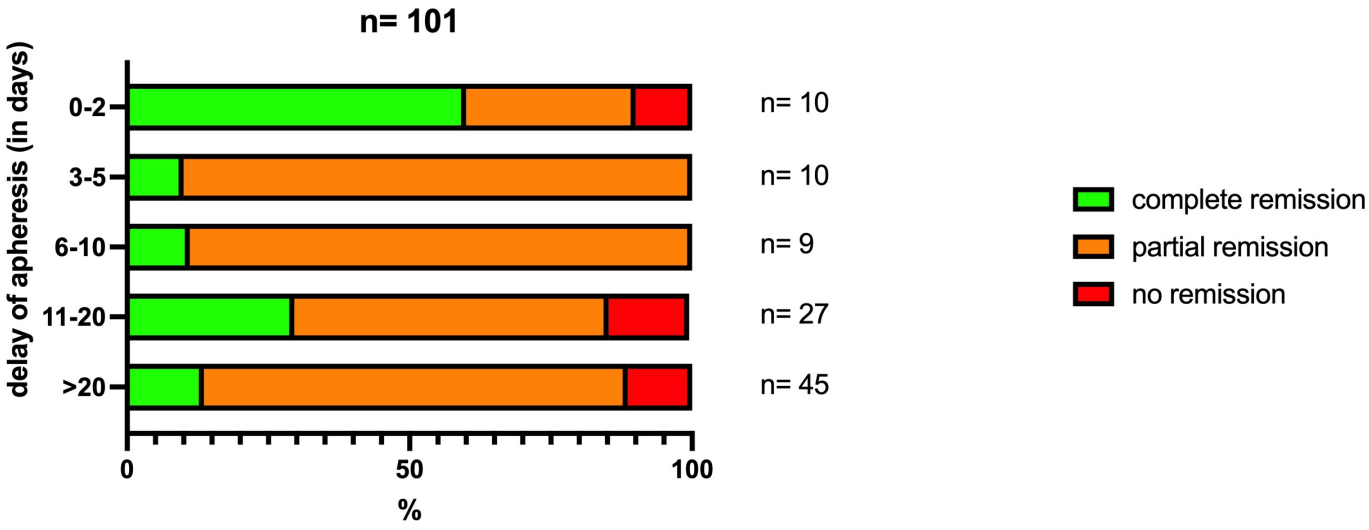

Figure 3 Bar chart showing the percentage of remission status based on apheresis delay (in days).

Considering the possible underestimation of visual deficits in the EDSS score, we additionally evaluated the △visual FSS, available in 22/50 (44%) of ON attacks. We likewise found no difference in the △visual FSS when comparing PLEX and IA+PLEX (n=13 vs 9, △1 (IQR 0–3.5) vs △0 (IQR 0–2.0), p=0.430). The △visual FSS did not differ in patients with and without DMT (n=11 vs 11, △0 (IQR 0–1.0) vs △2 (IQR 0–4.0), p=0.105) or first-line treated patients and the rest of the cohort (5 vs 17, △0 (IQR 0–2.0) vs △1 (IQR 0–2.0), p=0.528). However, the △visual FSS correlated with the patient's age at apheresis (p=0.042, r=0.437), indicating that younger patients benefited more.

Complications

Apheresis-related complications occurred in 8/117 treated attacks (7%), 5 during PLEX, 3 during combined PLEX/IA and none during IA. Four patients developed procedure-related systemic infections (2 PLEX, 2 PLEX/IA), one patient had heparin-induced thrombocytopenia, another patient had thrombocytopenia of unknown cause and one patient experienced transient cardiac arrhythmia (all PLEX). In one case, apheresis had to be stopped due to thrombosis of the internal jugular vein (PLEX/IA).

Table 2 Generalised mixed model analysis: factors associated with complete remission

	P value	OR	95% CI
Female (vs male)	0.217	1.006	−0.601 to 2.612
Age at attack (per year)	0.425	0.02	−0.029 to 0.069
Disease duration (per year)	0.208	−0.100	−0.258 to 0.057
ON (vs non-ON)	0.752	0.267	−1.406 to 1.941
MY (vs non-MY)	0.138	−1.519	−3.535 to 0.497
First line (vs second/third line)	**0.032**	2.579	0.225 to 4.933
DMT use (vs absence)	**0.017**	1.477	0.274 to 2.679
PLEX (vs IA+PLEX)	0.230	0.811	−0.521 to 2.144
IVMP dose (g)	0.627	0.042	−0.130 to 0.215

Generalised mixed model analyses with remission status of attacks (complete remission vs partial remission and no remission) as the dependent variable, n=100 attacks, p=0.040.
Significant values (p<0.05) are shown in bold.
DMT, disease-modifying therapy; IA, immunoadsorption; IVMP, intravenous methylprednisolone; MY, myelitis; ON, optic neuritis; PLEX, plasma exchange.

DISCUSSION

In this study, we systematically investigated the frequency and effectiveness of apheresis therapy in a large multicentre MOGAD cohort. Early start of apheresis, as first-line therapy, and concomitant use of DMT were associated with favourable clinical outcomes. Notably, the type of apheresis had no impact on the remission status.

Although MOGAD attacks are generally considered steroid sensitive,[1 22] the prevalence of steroid refractory relapses remains unknown. Data regarding the effectiveness of apheresis therapy in MOGAD attacks are also limited. Most previous studies on therapeutic apheresis in autoimmune CNS conditions lack adequate representation of patients with MOGAD, likely due to its rarity and only recent recognition as a distinct disease entity. We demonstrate that the use of apheresis in MOGAD is not uncommon in German-speaking countries, with every fifth MOGAD attack being treated with apheresis. Similarly, a study on treatment regimens by the French NOMADMUS study group observed that 28% of the included 67 MOGAD attacks underwent PLEX.[22] Moreover, every fourth patient in a large international study examining PLEX outcomes in ON (n=395) tested positive for MOG-IgG.[23]

In contrast, a prospective single-centre study in China evaluating the efficacy of PLEX in a heterogeneous cohort of patients with ON included only a minority of MOGAD-ON (6/124, 4.8%).[10] It is unclear whether this is due to different genetic backgrounds or different therapeutic approaches.

We suppose that steroid refractory relapses are not uncommon in MOGAD, with 20% of attacks and 41% of patients in our cohort being treated at least once with apheresis. The majority of patients (88%) received apheresis treatment as a second-line (after a median of 5000 mg IVMP) or third-line treatment (after a median of 13 000 mg IVMP). Moreover, the disability level at the last FU was significantly higher in the apheresis group compared with the non-apheresis group, despite similar disease duration and ARR.

Analysing factors associated with a favourable post-apheresis outcome, we identified that early use of apheresis as first-line therapy is beneficial for achieving complete remission from MOGAD attacks. Our observation is in line with multiple previous studies on other demyelinating CNS disorders including MS and NMOSD.[11 12 14 17 24 25] A meta-analysis also confirmed that the timepoint of PLEX initiation significantly influenced the

outcome, as indicated by EDSS reduction.[26] Moreover, in our study, complete remission was most frequently achieved in a very early apheresis initiation, specifically within the first 2 days after attack onset. This finding is also consistent with the results of two previous studies in AQP4-IgG$^+$ NMOSD, which identified a comparable optimal apheresis period of 0–2 days.[11 14]

Early elimination of disease-mediating antibodies can result in rapid clinical improvement. Despite demonstrating less robust complement activation compared with AQP4-IgG due to its bivalent binding pattern,[27] MOG-IgG can nonetheless activate the complement system. Interestingly, neuropathological findings in MOGAD are similar to MS pattern II lesions,[28] which are known to be particularly responsive to apheresis therapy.[13] Early removal of the autoantibodies and probably further inflammation-promoting factors could prevent further damage at the cellular level, similar to the reversible dysfunction as demonstrated in AQP4-IgG$^+$ NMOSD.[17]

Because of a possible monophasic disease course, the use of long-term immunotherapy in MOGAD remains a matter of debate. However, our results indicate that the presence of DMT in patients with severe relapses has a significant impact on post-apheresis clinical outcomes, especially in MY and cerebral manifestations. This finding was not significant in ON attacks, probably due to the smaller size of the ON subgroup. Data from other previous studies are controversial. Abboud *et al* reported comparable effects in NMOSD: preventive DMT resulted in a higher likelihood of reaching baseline EDSS after the attack.[29] Interestingly, their cohort consisted of mostly non-ON (69% MY, brain and brainstem) cases as well. In contrast, two other studies demonstrated no effect of concomitant DMT use on NMOSD attack recovery.[11 30] However, one of these studies investigated patients with ON attacks only.[30] It seems to be plausible that DMT prevents relapses and can reduce severity and neuroaxonal damage in those relapses that occur. Reduced relapse severity under DMT has been demonstrated in previous studies in NMOSD and MS.[31 32] Nevertheless, it remains unclear whether this effect differs between ON and non-ON attacks.

Depending on the relapse phenotype, there seem to be other factors relevant to the attack recovery. In ON attacks, the severity of vision loss at attack nadir and higher age at the attack were two additional risk factors for worse outcomes, as shown by Chen *et al*.[23] Interestingly, in a previous study comparing adult and paediatric patients with MOGAD, a younger age at ON onset demonstrated an even stronger association with visual outcomes compared with the grade of subsequent retinal atrophy involvement. This likely indicates a significant role of age-dependent cortical neuroplasticity in the visual cortex.[33] The patient's age might be a positive influencing factor in MOGAD-ON independent of an attack therapy.

In contrast to PLEX, which removes the entire blood plasma, IA selectively filters antibodies from the patient's blood. IA was shown to be effective in MS[34] and NMOSD.[35–37] Some studies even propose a better long-term outcome after attack treatment with IA in MS,[18] others found no differences between PLEX and IA in MS and NMOSD.[11 38] In line with these findings, clinical outcomes in our MOGAD cohort did not differ among different types of apheresis (PLEX, IA, PLEX/IA).

Apheresis was generally well tolerated, similar to findings from previous studies.[18 39] However, apheresis-related complications occurred in 7% of attacks, and in one case, apheresis had to be stopped due to thrombosis of the internal jugular vein.

Despite several strengths, including the large number of included attacks in this rare disease and the multicentre study design, our study has several limitations. The retrospective nature of the study and the inclusion of patients from university hospitals only could result in a selection bias, potentially skewing the data towards more severe cases. Baseline and FU EDSS data were not available for all patients. We cannot exclude prolonged effects of high-dose corticosteroids in patients receiving second-line and third-line apheresis therapies that might have influenced clinical outcomes. Additionally, we were unable to analyse other clinical and paraclinical findings, such as the grade of retinal atrophy and exact visual acuity in ON, or levels of neurofilament light chain and glial fibrillar acidic protein, due to a lack of biosamples. Although highly suggestive of MOGAD based on (para)clinical characteristics, 5/85 patients (6%) did not fully meet the proposed diagnostic criteria. Further, ideally prospective comparative studies are needed to confirm our findings.

Our study revealed that severe relapses requiring apheresis therapy are not rare, affecting approximately every fifth MOGAD attack in our cohort. All types of apheresis seem to be equally effective for the treatment of attacks in MOGAD. An early, first-line apheresis improved the outcome substantially. DMT was another relevant factor associated with a favourable outcome. This evidence is supporting the recommendation for long-term immunotherapy in patients with MOGAD with severe relapses.

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Supplemental Table 1. Characteristics of MOG-IgG+ patients not meeting the 2023 MOGAD criteria

ID	Gender	Age at onset	MOG-titer	Relapses	Disease duration	OCB	Additional information/ Comment
1	m	34y	Positive w/o titer (fixed CBA)	Isolated ADEM	7y	NA	Episode of severe encephalopathic syndrome, monophasic course, no further attacks (without DMT), suggestive of MOG-IgG+ ADEM (initial MRI not available)
2	m	53y	Low positive 1:50 (fixed CBA)	Isolated Myelitis	8y	neg	Isolated Myelitis, monophasic course, OCB negative, suggestive of MOGAD
3	m	50y	Low positive 1:32 (fixed CBA)	3x ON unilateral	9y	NA	(Recurrent) isolated ON suggestive of MOGAD with early relapse after steroid tapering, no data on papilledema/OCT or MRI in the acute stage available
4	m	42y	Low positive 1:32 (fixed CBA)	6x ON unilateral	2y	neg	
5	f	30y	Low positive 1:32 (fixed CBA)	1x ON unilateral	7y	NA	

Abbr.: ADEM= acute disseminated encephalomyelitis, CBA= cell-based assay, DMT= disease modifying therapy, f= female, m= male, MRI= magnetic resonance imaging, MS= multiple sclerosis, MOGAD= myelin oligodendrocyte glycoprotein antibody-associated disease, NA= not available, OCB= oligoclonal bands, OCT= optical coherence tomography, ON= optic neuritis, w/o= without, y= years.

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