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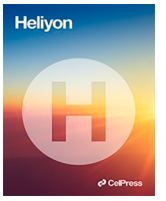

Research article

# The distinction of area postrema syndrome between MOGAD and NMOSD

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ARTICLE INFO

*Glossary:*
AP
Area postrema
APS
Area postrema syndrome
AQP4
Aquaporin-4
CBA
Cell-based assays
NMOSD
Neuromyelitis optica spectrum disorder
IgG
Immunoglobulin G
MOG
Myelin oligodendrocyte glycoprotein
MRI
Magnetic resonance imaging
NVH
Nausea
Vomiting
Hiccups
ON
Optic neuritis

ABSTRACT

*Background and objectives:* Both myelin oligodendrocyte glycoprotein-IgG associated disorders (MOGAD) and neuromyelitis optica spectrum disorder (NMOSD) are demyelinating diseases of the central nervous system. They present similar clinical manifestations such as optica neuritis, myelitis and area postrema syndrome (APS). The distinctions of optica neuritis (ON) and myelitis between them have been elaborated to great length while their differences in APS remain to be elucidated. We aim to report the frequency of APS in patients with MOGAD as well as NNOSD patients, and to compare the characteristics of APS between patients with MOGAD and those with NMOSD.

*Methods:* Seven MOG-IgG positive APS patients were retrospectively identified between 2017 and 2022. APS phenotypes have been previously described. The similarities and differences between MOGAD and NMOSD patients with APS was compared, including the frequency and duration of APS between the two diseases, and their incidences of accompanied subtentorial lesions have also been described and compared.

*Results:* We reviewed a cohort of 218 MOG-IgG-positive patients, and 396 patients with NMOSD. 200 MOGAD patients and 332 NMOSD patients were included in this study. In the cohort, seven patients with MOG-IgG-positive antibody presented with APS were analyzed, four of whom had disease onset with APS. Of the 332 patients with NMOSD, 47 had APS attacks while 31 had APS at disease onset. In patients with MOGAD, 2 had nausea, 3 had vomiting, 5 had hiccups, and 1 patient presented with all three symptoms above. In patients with NMOSD, 70.2 % had nausea, vomiting and hiccups at the same time during APS attacks. Apart from the medulla oblongata, other subtentorial regions were also affected in 6/7 MOGAD patients while 14/47 NMOSD patients had other subtentorial regions involved. During an APS attack, the incidence of concomitant lesions in the brainstem and other regions was significantly greater in MOGAD than in the NMOSD cohort ($P = 0.008$*).

*Conclusion:* APS is a rare, but not isolated clinical manifestation of MOGAD. APS happened more frequently with other supratentorial and subtentorial lesions in MOGAD. The symptoms of NVH (nausea, vomiting, hiccups) tended to happen respectively in MOGAD compared with NMOSD. The phenotype or mechanism of APS in MOGAD may differ from that in NMOSD.

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## 1. Introduction

MOGAD is an inflammatory demyelinating disease of the central nervous system characterized by MOG-IgG seropositivity. Cohort studies suggest differences in clinical and paraclinical presentation, treatment response and prognosis. MOGAD is now considered to be a disease entity in its own right, distinct from AQP4-IgG positive NMOSD [1–3].

Patients with MOG-IgG present with isolated optic neuritis or transverse myelitis, acute disseminated encephalomyelitis (ADEM), brainstem or cerebellar features, or cerebral cortical encephalitis [4]. Brainstem lesions in the midbrain, pons and medulla oblongata can all be observed in MOGAD. However, the appearances of brainstem lesions were not reliably distinguished between patients with MOG-IgG and AQP4-IgG seropositive NMOSD [5,6].

Area postrema syndrome (APS) is defined as an episode of otherwise unexplained nausea, vomiting, and hiccups(NVH) [7,8]. APS is a core clinical manifestation of AQP4-IgG-seropositive NMOSD. Recently, several case reports and studies have indicated that APS

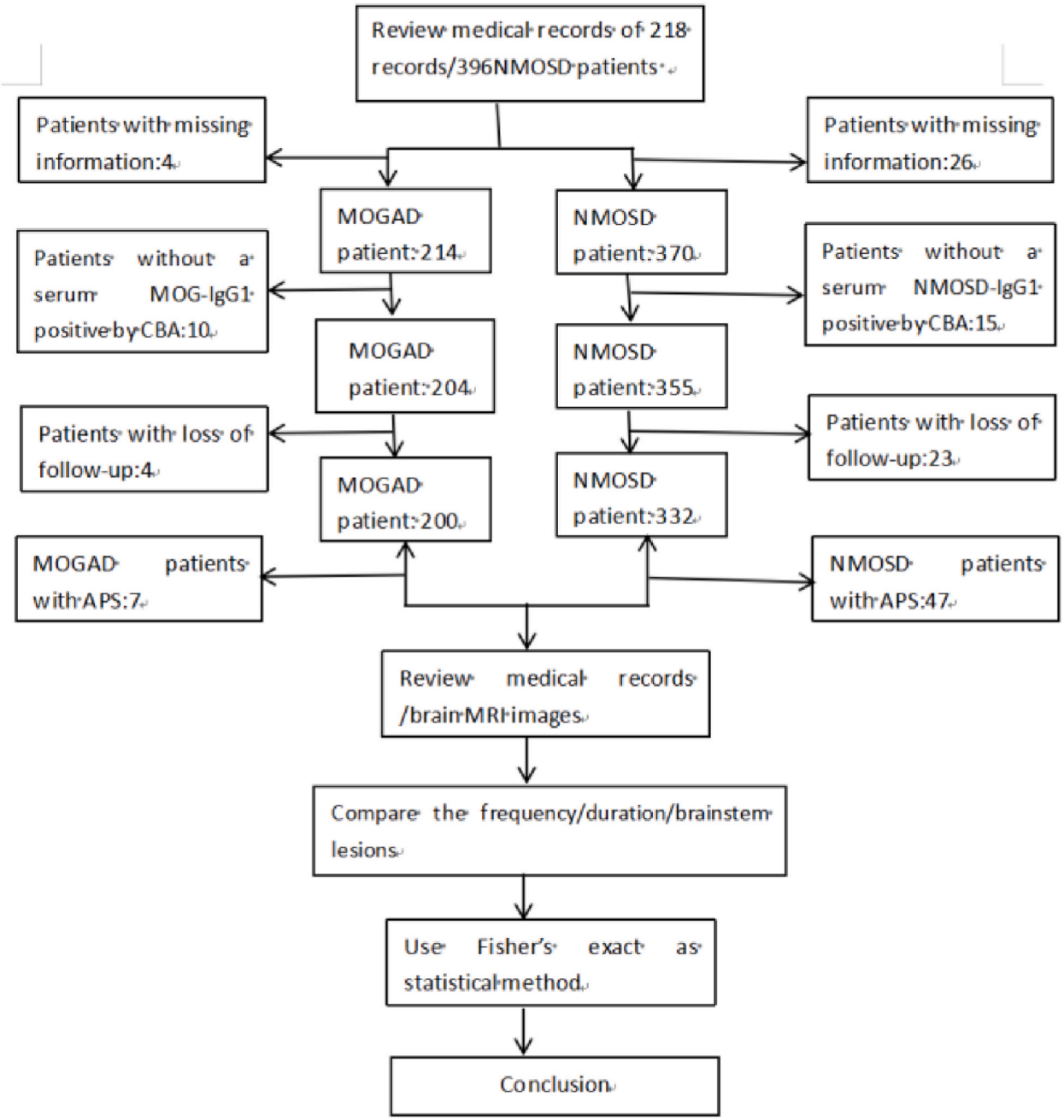


**Fig. 1.** Flow chart of our study design.

might also appear in MOGAD. In 2020, a 24-year-old woman experienced APS with associated headaches and weight loss for more than one year. In the case report, the final diagnosis was MOGAD [9]. Abhay et al. reported a case of APS as the initial presentation of double-seropositive aquaporin-4 and myelin oligodendrocyte glycoprotein (MOG) antibodies [10]. However, owing to its low frequency in MOGAD, data on APS involvement in MOGAD are limited to subsets of case series, and only a few studies have focused on APS in MOGAD [11,12]. However, the characteristics of APS in this demyelinating disorder have not been explored. Herein, we report the largest series of MOG-IgG positive patients with APS. The characteristics of APS, that is., frequency, onset, duration and immunotherapy, are described. Furthermore, we compared MOGAD and NMOSD with respect to APS features.

## 2. Methods

### 2.1. Participants

This study included 332 participants with AQP4 antibodies and 200 with MOG antibodies from the First Affiliated Hospital of Wannan Medical College and Huashan Hospital of Fudan University between 2017 and 2022. Finally, the frequency and characteristics of APS were evaluated in participants with AQP4 and MOG antibodies, through a retrospective review of the medical records based on physicians' active questioning of APS. APS was defined according to the following recently proposed criteria: (1) acute or subacute, single or combined, episodic or constant nausea, vomiting, or hiccups; (2) persistent for at least 48 h; and(3) unknown etiology. The exclusion criteria were as follows: (1) patients with an APS course with >1 month; (2) patients with double positive antibodies against both AQP4 and MOG, or (3) patients with incomplete medical records. After a detailed review of the medical records, we found seven MOGAD and 31 NMOSD patients with APS. We further analyzed the clinical characteristics and imaging features of the patients.

### 2.2. MOG-IgG and NMOSD-IgG detections

MOG-IgG was tested for clinical purposes in all patients and was detected using a commercial fixed-cell-based assay (CBA) (Euroimmun, Germany), which employs recombinant human full-length MOG as an antigenic substrate. AQP4-IgG was tested using a commercial CBA (Euroimmun, Germany) employing recombinant human full-length AQP4 (Fig. 1).

### 2.3. MRI review

All available magnetic resonance imaging (MRI) sequences were reviewed within 6 weeks of attacks, either at the First Affiliated Hospital of Wannan Medical College or Huashan Hospital or at other qualified centers. Sagittal and axial T2-weighted, and T2 fluid-attenuated inversion recovery (FLAIR) sequences were analyzed. Medical records were assessed for accompanying clinical manifestations attributable to dysfunction of the area postrema (AP), spinal cord, and other subtentorial including midbrain, pons, and cerebellum (Figs. 4 and 5). Two neuroradiologists blinded to the antibody seropositivity and clinical information then reviewed the AP, subtentorial, and spinal cord MRI findings in the MOGAD and NMOSD cohorts.

### 2.4. Study design

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### 2.5. Case series presentation

The seven patients with APS in MOGAD included six males and one female. Their clinical features were shown in Table 1.

### 2.6. Statistical analysis

Statistical analyses were performed with SPSS, version 26.0. The characteristics are reported as the mean ± standard deviation (SD) or median (range) for continuous variables and as frequencies and percentages for categorical variables. Chi-square, and Fisher's exact

**Table 1**
Clinical features of MOGAD in our reported cases.

| Case | Onset age | Sex | relapses | Attacks of APS | Duration of nausea(days) | Duration of vomiting(days) | Duration of hiccups(days) | Other subtentorial lesions besides AP | myelitis |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 24 | male | 13 | 1 | 0 | 8 | 0 | midbrain; pons | / |
| 2 | 32 | male | 6 | 1 | 0 | 0 | 10 | pons | C4-5 |
| 3 | 27 | male | 4 | 1 | 0 | 20 | 3 | / | C1–C4; T6-T8 |
| 4 | 33 | female | 1 | 1 | 0 | 0 | 8 | cerebellum | / |
| 5 | 38 | male | 0 | 1 | 0 | 0 | 7 | midbrain; pons | C2–C5 |
| 6 | 31 | male | 0 | 1 | 3 | 3 | 7 | / | / |
| 7 | 49 | male | 4 | 1 | 7 | 0 | 0 | midbrain | / |

tests were used, as appropriate. A 2-tailed p values less than 0.05 were considered significant.

### 2.7. Standard protocol approvals, registrations, and patient consents

This study was approved by the Institutional Review Board of the First Affiliated Hospital of Wannan Medical College and Huashan Hospital, Fudan University. Written informed consent was obtained from all the participants.

## 3. Results

### 3.1. Incidence and phenotypes of APS in MOGAD and NMOSD patients

We identified seven patients (one woman, six men; mean age: 33.4 ± 8.2 years) with APS among 200 MOG-IgG positive patients (six had available MRI figures, Fig. 4). Serum AQP4-IgG was negative in all patients. Moreover, 47 patients (38 females, nine males; 31.1 ± 13.2 years) with APS were identified among the 332 NMOSD patients (Fig. 5).

APS occurred as inaugural symptom in four patients in the MOGAD cohort and 31 patients in the NMOSD cohort. The mean duration of APS episodes was 9.5 ± 4.7 days in MOGAD patients, and 18.1 ± 13.7 days in NMOSD. In the MOGAD cohort, two patients had nausea, three patients had vomiting and five presented with hiccups. Only one patient experienced NVH simultaneously during an episode. None of the patients had isolated APS attacks because they presented with symptoms of optic neuritis or myelitis during the clinical course. In the NMOSD cohort, 36 patients experienced nausea (MOG vs NMOSD, 28.6 % vs 76.6 %, P = 0.019*), 41 patients experienced vomiting (MOG vs NMOSD,42.9 % vs 87.2 %, P = 0.017*) and 42 patients presented with hiccups (MOG vs. NMOSD,71.4 % vs 89.4 %, P = 0.220). 33 patients experienced NVH during an APS attack (MOG vs NMOSD 14.3 % vs 70.2 %, P = 0.008**). A comparison of APS phenotypes and clinical features is presented in Table 2 and Table 3. The frequencies of ON, myelitis and subtentorial lesions in addition to AP during the entire disease course in MOGAD and NMOSD patients were 57.1 %, 29.0 %, 85.7 % and 66.0 %, 87.2 %, 29.8 %, respectively (P = 0.687, P = 0.017*, P = 0.008**). During an APS attack, the percentage of concurrent lesions in the optic nerve ( 14.3%vs10.6 %, P = 0.584 ) and spinal cord(42.9 % vs 27.7 %, P = 0.410) were not significantly different while percentage in brainstem (57.1 % vs 12.8 %, P = 0.017*)and other brain regions(57.1 % vs 2.1 %, P = 0.001**) were prominently different. (Fig. 2). The incidence of APS onset in patients with MOGAD was not significantly different from that in patients with NMOSD (MOG vs NMOSD,57.1 % vs 66.0 %, P = 0.474). APS tended to occur in the early stages of both demyelinating diseases, particularly MOGAD (Fig. 3). The percentages of patients using immunosuppressant during the entire disease course in the MOGAD and NMOSD cohorts with APS were 14.3 % and 87.2 %, respectively (P = 0.002**). During recent follow-up, 28.6 % (2/7) of the MOGAD cohort continued receiving immunosuppressant drugs whereas 87.1 %(27/31) of the patients in the NMOSD cohort continued using immunosuppressors.

## 4. Discussion

The distinction between ON and myelitis in MOGAD and NMOSD has been well established [13–15], whereas little attention has been given to APS, another core symptom in NMOSD. The current study delineated a comprehensive clinical picture of APS in MOGAD and compared APS in NMOSD. Women were more frequently affected in NMOSD patients than in MOGAD patients (88.86 % vs 58.00 %, P < 0.0001**) in our cohort (Table 4).

The average age of onset in the MOGAD (33.42 ± 8.2 years old) group was similar to that in the NMOSD group (33.1 ± 13.2 years old). In a Korean study [12],1.9 % (2/107) adults with MOG antibodies developed APS. APS with isolated INVH and a discrete lesion in the AP were found in only one patient, representing 0.6 % of the MOGAD cohort [11]. In our study, the incidence of APS was 3.2 % (7/208) in the MOGAD cohort. Only one patient had concurrent APS with diplopia but without any other clinical phenotypes. Patients with at least one episode of APS during the course of the study accounted for 11.9 % (47/396) of the patients in the NMOSD cohort. The frequencies of initial onset with APS of MOGAD and NMOSD were 57.1 % (4/7) and 66.0 % (31/47), respectively, with no significant difference. However, APS in the MOGAD group seldom occurred in isolation while more than half of the patients with NMOSD

**Table 2**
Comparison of MOGAD and NMOSD patients with APS.

| Cohort | MOGAD(n = 7) | NMOSD(N = 47) | P |
|---|---|---|---|
| Onset age (years old) | 33.42 ± 8.2 | 33.1 ± 13.2 | – |
| Sex ratio (female%) | 14.3 % | 80.9 % | - |
| ON | 57.1 % | 66.0 % | 0.687 |
| Myelitis | 29.0 % | 87.2 % | **0.017**[a] |
| Frequency of APS in the whole cohort | 3.5 % | 14.16 % | **<0.001**** |
| Other brainstem lesions besides AP | 85.7 % | 29.8 % | **0.008**** |
| Time before diagnosis ( months ) | 5.0 | 5.1 | – |
| Immunosuppressors usage | 14.3 % | 87.2 % | **0.002**** |

Data in bold represent values with P values less than 0.05.
[a] Immunosuppressors include prednisone, azathioprine, mofetil, tacrolimus, and rituximab.
[b] No P value of time before diagnosis was obtained since there was no *t*-test between a cohort of seven MOGAD and 47 NMOSD.

**Table 3**
Comparisons between MOGAD and NMOSD with APS.

| Cohort | MOGAD(n = 7) | NMOSD(N = 47) | P |
|---|---|---|---|
| Initial onset of APS | 57.1 % | 66.0 % | 0.474 |
| Isolated APS attack | 0 | 51.1 % | **<0.001**** |
| Mean attacks of APS | 1 | 1.3 | – |
| Patients with nausea | 28.6 % | 76.6 % | **0.019**[a] |
| Patients with vomiting | 42.9 % | 87.2 % | **0.017**[a] |
| Patients with hiccups | 71.4 % | 89.4 % | 0.220 |
| Patients with NVH | 14.3 % | 70.2 % | **0.008**** |
| APS duration(days) | 9.5 ± 4.7 | 18.1 ± 13.7 | – |

[a] APS duration is the duration from the onset attack of APS to when it diminished.
[b] No P value of mean attacks of APS was obtained since there was no *t*-test between a cohort of seven MOGAD and 47 NMOSD.

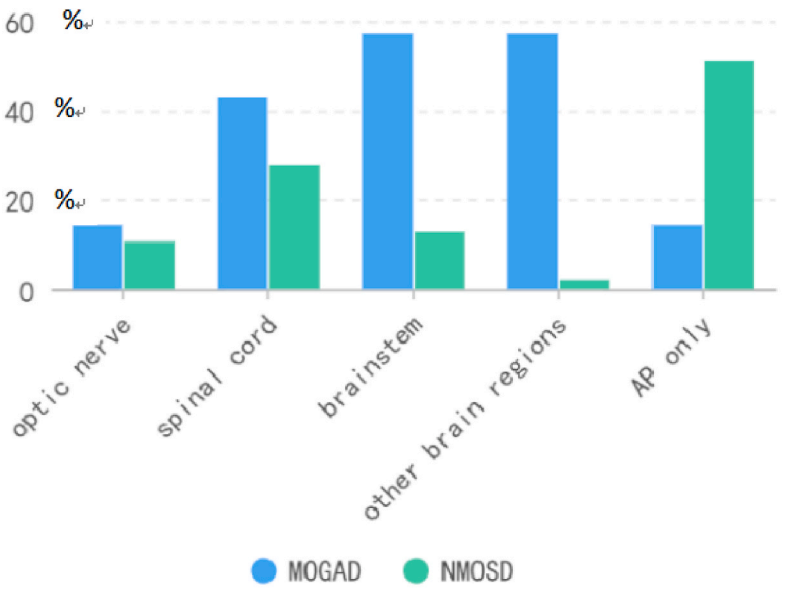


**Fig. 2.** The incidences of different regions (optic nerve/spinal cord/brainstem/other brain regions/AP) involved during an APS attack in MOGAD and NMOSD. Fisher's exact test was used to compare incidences. Significances were found in frequencies of involvement of regions of brainstem (57.1 % vs 12.8 %, P = 0.017*)and other brain regions(57.1 % vs 2.1 %, P = 0.001**) with APS attacks between MOGAD and NMOSD patients.

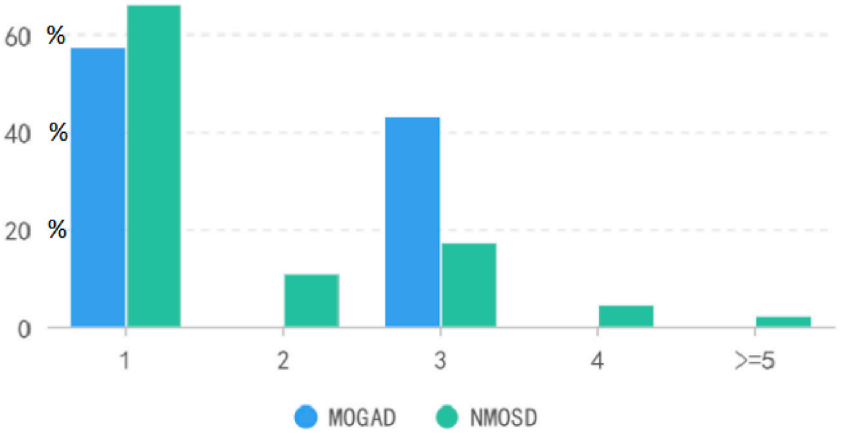


**Fig. 3.** The onset of APS mostly occurs at the early stage of the disease course in both MOGAD and NMOSD patients. More than half of the APS episodes occurred during the first attack in both disorders (57.1 % vs 66.0 %, P = 0.474). Fisher's exact test was used to compare the incidences.

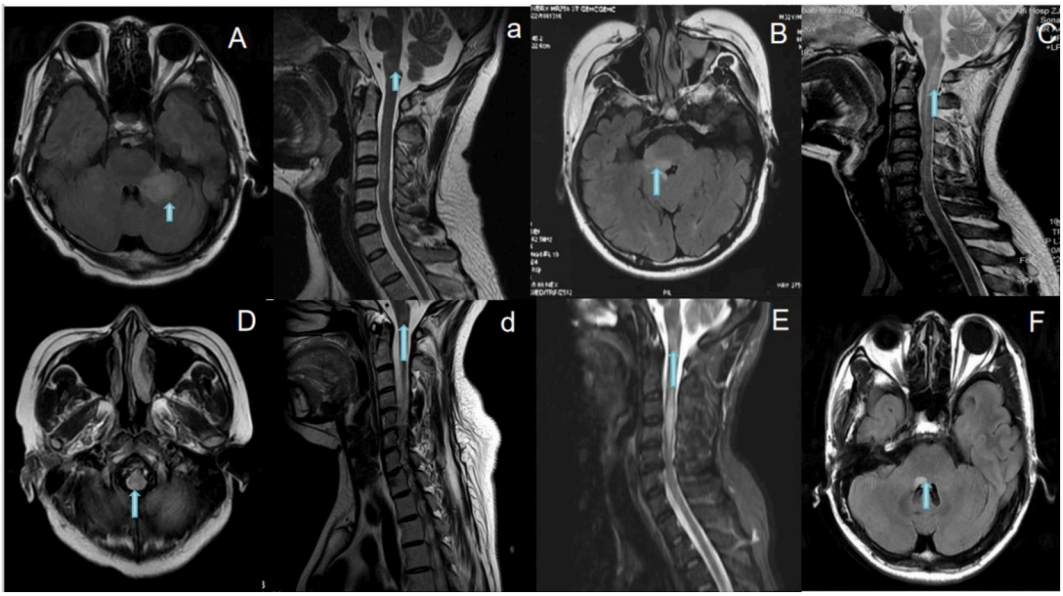


**Fig. 4.** T2-weighted fluid-attenuated inversion recovery and Sagittal T2-weighted scan show lesions in the area postrema(AP) and multifocal lesions in the medulla and cervical spinal cord.
A-a. Patient 1. A male first experienced APS in 2015. He had 14 attacks in total and the remaining relapses were ON and myelitis. Figure A showed the AP foci with the left pedunculus involved. Panel a showed the dorsal medulla and cervical spinal cord lesions. The midbrain was also involved (not shown in the figure)
B. Patient 2. A male patient was diagnosed with MOG-IgG-associated IDDs following an APS attack in 2016. The area around the fourth ventricle and pons were affected during the course.
C. Patient 3. A male patient experienced nausea and hiccups in 2016 when MOG-IgG was detected in his serum in 2017. He experienced five attacks through his course. The medulla, cervical spinal cord and pons have been targeted in patients with APS.
D. Patient 4. A-33-year-old female experienced nausea, vomiting, hiccupping (NVH), lower limb weakness, and slurred speech in 2021. She was diagnosed with MOGAD soon after MOG-IgG antibodies were detected. Subsequently, the patient experienced another attack. Cortical and cerebellar lesions were observed via magnetic resonance imaging (not shown in Fig. 2).
E. Patient 5. A 38-year-old male presented with headaches, memory loss, double vision, and hiccups during the first episode in 2020. The patient had not experienced a second episode during the last follow-up visit. There were no lesions in his pons, medulla and cervical spinal cord.
F. Patient 6. A 31-year-old male visited our hospital with double vision and NVH. He never experienced a second episode during follow-up. The lesion was confined to the AP on brain MRI.

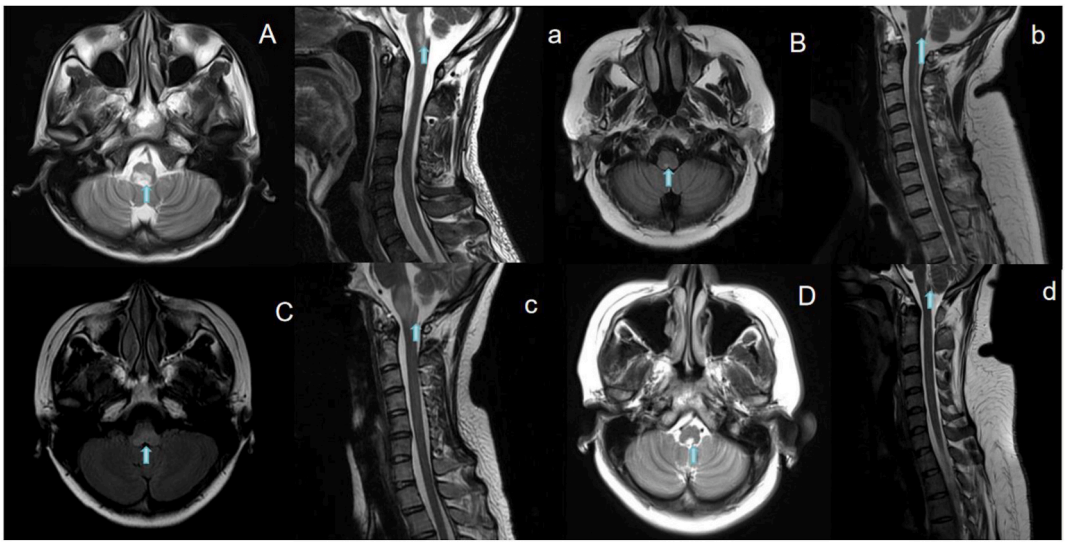


**Fig. 5.** The T2-weighted fluid-attenuated inversion recovery and Sagittal T2-weighted scan showing lesions in the area postrema and multifocal lesions in medulla and cervical spinal cord in NMOSD patients.

**Table 4**
Characteristics of MOGAD and NMOSD in our cohort.

| Cohort | MOGAD (n = 200) | NMOSD (n = 332) | P |
|---|---|---|---|
| Onset age (years old) | 28.16 ± 10.5 | 38.72 ± 11.0 | **<0.0001**** |
| Sex (female%) | 116/200 ( 58.00 % ) | 295/332 ( 88.86 % ) | **<0.0001**** |
| Disease duration (months) | 38 ( 4–1479 ) | 44 ( 5–466 ) | – |
| Onset phenotype | | | |
| ON | 98 | 126 | **0.0144*** |
| Myelitis | 32 | 92 | **0.0021**** |
| Brainstem + Cerebral syndrome | 50 | 49 | **0.0040**** |
| Mixed | 20 | 65 | **0.0033**** |
| Baseline MOG/AQP4-ab titre ( serum ) | | | |
| | 1:100(1:10–1:1000) | 1:100 ( 1:10–1:10000 ) | – |

experienced APS without concurrent clinical symptoms. APS tended to occur independently in patients with NMOSD but not in patients with MOGAD. According to our data, 72.3 % (34/47) patients with APS in the NMOSD group initially presented to gastroenterologists and underwent extensive workup including upper gastrointestinal endoscopy, transit studies, and abdominal computed tomography. No gastrointestinal disease was identified by the gastroenterologist for any patients. APS at onset especially when when it occurs independently might delay the diagnosis of NMOSD and cause further neurological deficits in the form of ON, transverse myelitis, or brainstem syndrome [10].

A comparison of the incidences and duration of NVH between the two cohorts revealed that patients with NMOSD suffer more from this syndrome. The incidences of nausea, vomiting, and hiccups were 28.6 %, 42.9 %, and 71.4 % in the MOGAD group, whereas in NMOSD group, the percentages were 76.6 %,87.2 %, 89.4 %, respectively($P = 0.019$, $P = 0.017$,$P = 0.220$). Nausea, vomiting, and hiccups tended to occur concurrently in patients with NMOSD with an incidence of 70.2 %. In the MOGAD group, only one patient experienced NVH at the same time during an APS episode. Moreover, the duration of NVH in NMOSD patients was longer than that in MOGAD patients(18.1 ± 13.7days vs 9.5 ± 4.7 days). Netravathi et al. concluded that exclusive emesis without hiccups appeared to be more common in MOGAD than in NMOSD [16]. Although hiccups without nausea and vomiting seemed more common in MOGAD in our research. Nausea, vomiting and hiccups usually don't occur together in an APS attack in this disorder.

We also observed that APS occurred more frequently in conjunction with other subtentorial lesions, such as those in the midbrain, pons or cerebellum, in MOGAD. Other brain regions such as the cortex, and temporal lobes are also affected during APS attacks. Meanwhile, myelitis is more commonly observed in NMOSD patients with APS. Kunchok et al. concluded that most patients with MOGAD with INVH did not have discrete AP lesions, but had patchy, poorly demarcated lower brainstem lesions (most commonly in the context of ADEM) [11]. AP is highly abundant in the AQP4 water channel; therefore, it is usually founded in AQP4 antibody–-positive individuals. Only a few cases of MOG-positive/AQP4-negative patients with APS have been reported in the literature [12]. APS in MOGAD may be caused by the entrance of pathogenic MOG-IgG and MOG-specific lymphocytes due to the lack of blood-brain barrier (BBB) at the AP and disruption of the emesis circuit. The lack of BBB may also explain the preference of AP involvement in individuals with combined occurrence of supratentorial and subtentorial lesions in MOG-positive individuals in our research.

In conclusion, APS was a rare clinical feature in southern Chinese people with MOG antibodies. Isolated APS tended to occur more frequently in patients with NMOSD than in those with MOGAD. While NMOSD patients usually had concurrent NVH and a longer duration of one episode, MOGAD patients tended to have only one episode of the NVH symptoms. NMOSD is more likely to affect the spinal cord, whereas MOGAD is more likely to involve other subtentorial areas throughout the disease course. During an APS attack, the brainstem and other regions of the brain are more frequently involved simultaneously in MOGAD than in NMOSD. The phenotypes of APS may differ between these two disorders. These findings are convenient for the differential diagnosis between the two disorders when antibodies are temporarily negative or unavailable. Patients with isolated APS have a greater chance of being diagnosed with NMOSD, whereas those with concomitant brainstem lesions tend to be diagnosed with MOGAD. For those already diagnosed, NMOSD patients with APS might have more severe clinical symptoms and a longer course of nausea, vomiting, or hiccups than MOGAD patients. Future studies are warranted to identify the underlying mechanisms of such a distinction in APS between the two entities.

This study had several limitations. Firstly, it was limited by its retrospective nature and potential referral bias with data from only two centers. We followed-up all the patients with their latest attacks and current immunosuppressants, and will continue to conduct follow-up if prospective studies are warranted. Secondly, due to the low incidence of MOGAD and a rare appearance of APS in this disease, we only included seven MOGAD patients in this cohort. That may influence the accuracy of this research to some extent. Thirdly, some of the patients we included didn't have a long follow-up which might bias the conclusion of this study. However, to the best of our knowledge, this study included the largest case series of MOGAD patients with APS, and a direct comparison of the APS phenotypes between these two demyelinating disorders was lacking in the previous research. For the first time, we attempted to distinguish the features between the two diseases. This may offer convenience in clinical practice in distinguishing the two, especially when the detection of the antibodies was unavailable. The treatment strategy for NMOSD differs from that for MOGAD, since they have different mechanisms. Early differentiation between them could lead to timely and effective therapy, which reduces the degrees of disability. In the future, multicenter studies with larger cohorts are needed to verify our conclusions further. The mechanisms underlying.

APS under these two conditions remain to be elucidated. In the future, animal researches and vitro experiments are warranted to illustrate the underlying mechanisms of APS in MOGAD and NMOSD.

## Ethics statement

This study involved human participants and was approved by the medical ethics committees of Huashan Hospital (HIRB-2021073, 2021.02.24). Written informed consent to participate and publish was acquired from each patient.

## Data availability statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request from a qualified researcher.

## CRediT authorship contribution statement

**Ying Chen:** Writing – original draft, Investigation, Formal analysis, Data curation, Conceptualization. **Jingzi Zhangbao:** Writing – review & editing, Investigation, Formal analysis, Data curation, Conceptualization. **Junfeng Xu:** Investigation, Data curation. **Lei Zhou:** Resources, Formal analysis, Data curation. **Zhiming Zhou:** Writing – review & editing, Supervision, Conceptualization. **Chao Quan:** Writing – review & editing, Supervision, Project administration, Methodology, Formal analysis, Data curation.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgment

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## References

[1] S.S. Zamvil, A.J. Slavin, Does MOG Ig-positive AQP4-seronegative opticospinal inflammatory disease justify a diagnosis of NMO spectrum disorder? Neurol Neuroimmunol Neuroinflamm 2 (2015) e62. 13.
[2] R. Hohlfeld, K. Dornmair, E. Meinl, H. Wekerle, The search for the target antigens of multiple sclerosis, part 2: CD8+ T cells, B cells, and antibodies in the focus of reverse-translational research, Lancet Neurol. 15 (2016) 317–323.
[3] A. Sebastian López-Chiriboga, Association of MOG-IgG serostatus with relapse after acute disseminated encephalomyelitis and proposed diagnostic criteria for MOG-IgG–associated disorders, JAMA Neurol. 75 (11) (2018 Nov) 1355–1363.
[4] Brenda Banwell, et al., Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: international MOGAD Panel proposed criteria, Lancet Neurol. 22 (2023) 268–282.
[5] M. Jurynczyk, R. Geraldes, F. Probert, et al., Distinct brain imaging characteristics of autoantibody-mediated CNS conditions and multiple sclerosis, Brain 140 (2017) 617–627, 49.
[6] S.A. Banks, P.P. Morris, J.J. Chen, et al., Brainstem and cerebellar involvement in MOG-IgG-associated disorder versus aquaporin-4- IgG and MS, J. Neurol. Neurosurg. Psychiatry (2020), https://doi.org/10.1136/jnnp-2020-325121 published online Dec28,2020.
[7] D.M. Wingerchuk, B. Banwell, J.L. Bennett, et al., International Panel for NMO Diagnosis, International consensus diagnostic criteria for neuromyelitis optica spectrum disorders, Neurology 85 (2) (2015) 177–189.
[8] E. Shosha, D. Dubey, J. Palace, et al., Area postrema syndrome: frequency, criteria, and severity in AQP4-IgG-positive NMOSD, Neurology 91 (17) (2018) e1642–e1651.
[9] Block DO. Allison, Anti-myelin oligodendrocyte glycoprotein (MOG) associated disease masquerading as prolonged intractable nausea and vomiting, Multiple Sclerosis and Related Disorders (2020), https://doi.org/10.1016/j.msard.2020.102308.
[10] Abhay Ranjan, et al., Area postrema syndrome an initial presentation of double-seropositive AQP4 and MOG antibodies, Neurology: Clinical Practice August 12 (4) (2022), https://doi.org/10.1212/CPJ.0000000000200065 e82-e84.
[11] Amy Kunchok, et al., Does area postrema syndrome occur in myelin oligodendrocyte glycoprotein-IgG-associated disorders (MOGAD)? Neurology® 94 (2020) 1–4, https://doi.org/10.1212/WNL.0000000000008786.
[12] Jae-Won Hyun, et al., Value of area postrema syndrome in differentiating adults with AQP4 vs. MOG antibodies, Frontiers in Neurology June 11 (2020).
[13] Ram Bhupal Reddy Nagireddy et al,Clinicoradiological comparative study of Aquaporin-4-IgG seropositive neuromyelitis optica spectrum disorder (NMOSD) and MOG antibody associated disease (MOGAD): A prospective observational study and review of literature,J Neuroimmunol,volume 361,577742.
[14] Giulia Fadda, Myelitis features and outcomes in CNS demyelinating disorders: comparison between multiple sclerosis, MOGAD, and AQP4-IgG-positive NMOSD, Front. Neurol. (November 07 2022).
[15] Elia Sechi, et al., Comparison of MRI lesion evolution in different central nervous system demyelinating disorders, Neurology® 97 (2021) e1097–e1109, https://doi.org/10.1212/WNL.0000000000012467.
[16] M. Netravathi, Prodromal emesis in MOG-antibody associated disorder, Mult Scler Relat Disord 58 (2022 Feb) 103463, https://doi.org/10.1016/j.msard.2021.103463.