

Research Article



Anti-Neutrophil Cytoplasmic Antibody (ANCA)-Associated Vasculitis Involving the Central Nervous System with MOG-IgG Associated Disease: A Rare Case Report and Review of the Literature

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Abstract:

Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) and Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody (MOG-IgG)-associated disorders (MOGAD) – though both immune disorders – have rarely been reported together. Here we present their first documented co-occurrence and review CNS manifestations in AAV.

Methods We report a rare case of a 76-year-old female patient who developed MOGAD following her diagnosis of AAV. A literature search was conducted in the PubMed to analyze AAV's clinical features in the CNS.

Results The patient was diagnosed with AAV after initial interstitial pneumonia and peripheral nerve injury, followed by intermittent headache, extensive abnormal white matter signals with meningeal enhancement, positive serum MOG antibody. Immunotherapy failed to relieve headache effectively, and the patient died of pneumonia after 14 months. Through a literature search, a total of 358 cases of AAV and CNS involvement were identified. The retrieved studies revealed that 102 patients had meningeal involvement, which is the most commonly affected site in the CNS. Hypertrophic pachymeningitis and ischemic stroke are the main manifestations of AAV in the CNS.

Conclusion Rare AAV complicated by MOGAD case has poor outcomes. Studies indicate AAV frequently affects the CNS via hypertrophic pachymeningitis or ischemic stroke, typically presenting with headache and dural involvement.

Keywords Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV), Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody (MOG-IgG)-associated disorders (MOGAD), eosinophilic granulomatosis with polyangiitis (EGPA).

Introduction

Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) constitutes a spectrum of systemic small-vessel vasculitides, distinguished by the presence of detectable ANCA in serum. The disease predominantly involves small vessels, encompassing arterioles, capillaries, and venules, with occasional involvement of medium-sized arteries. AAV stands as the

prevalent form of systemic small-vessel vasculitis encountered in clinical settings. The classical subtypes of AAV encompass granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and eosinophilic granulomatosis with polyangiitis (EGPA), also recognized as Churg-Strauss syndrome[1, 2].

Myelin oligodendrocyte glycoprotein (MOG) is a glycoprotein uniquely expressed on oligodendrocytes within the central nervous system. Anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibody (MOG-IgG)-associated disorders (MOGAD) represent a recently delineated immune-mediated inflammatory demyelinating disease of the central nervous system, with its pathogenesis potentially implicated with MOG-IgG. The principal clinical manifestations include meningoencephalitis, optic neuritis, myelitis, acute disseminated encephalomyelitis, brainstem encephalitis, and others[3-5].

To our knowledge, this report documents the inaugural case of MOGAD following the onset of AAV. Furthermore, a comprehensive literature review is conducted to elucidate the involvement of the central nervous system in AAV.

Materials and Methods

We searched the PubMed databases from the year of creation until August 2025. The multiple search Medical Subject Headings (MeSH) terms were systemically used alone

or in combination: “Wegener granulomatosis”, “Churg-Strauss syndrome”, “microscopic polyangiitis”, “Anti-Neutrophil Cytoplasmic Antibody-Associated Vasculitis”, and “central nervous system”. Additional references were found by hand search.

Case Report

A 76-year-old female patient presented with unexplained fever starting from June 2015, with a peak body temperature of 40 °C, accompanied by fatigue, sore throat, and headache. She was initially diagnosed with interstitial pneumonia at another hospital. Subsequent tests revealed positive antinuclear antibodies at a titer of 1:100, weakly positive anti-SC170, positive pANCA, positive anti-myeloperoxidase antibodies, and positive PR3 antibodies. She was diagnosed with AAV and interstitial pneumonia and received corresponding pharmacological treatment, after which her body temperature returned to normal.

In February 2016, the patient experienced bilateral foot pain and numbness and was diagnosed with AAV involving the peripheral nervous system. From February 2020, she developed recurrent low-grade fever, with a body temperature around

37.6°C, accompanied by chills and headache but without shivering, nausea, or vomiting. Her fever could resolve after taking medicine, with each episode lasting about one day but recurring every half month. In May 2020, she was hospitalized due to disease recurrence, and her treatment was adjusted (prednisone tablets, 10 mg/day; tacrolimus, 0.5 mg/day). However, the patient failed to consistently take tacrolimus, and the hormone dosage was gradually tapered to 5 mg/day, which was maintained from April to October 2023. During this period, she experienced intermittent fever again, accompanied by headache and left hip pain. On October 20, 2023, she presented to the rheumatology department of our hospital with headache and fever and was later transferred to the neurology department.

Upon admission, her vital signs were as follows: body temperature 36.5 °C, pulse rate 78 beats/min, respiratory rate 20 breaths/min, and blood pressure 115/74 mmHg. She was alert and oriented, with negative Kernig's and Brudzinski's signs. Both pupils were equal in size and round, with normal light reflexes and no horizontal nystagmus (-). Her tongue protruded centrally, and bilateral nasolabial folds were symmetric. Muscle strength was graded as 5- in both upper extremities and 4+ in both lower extremities (due to femoral head necrosis), with normal muscle tone and tendon reflexes in all four limbs. She exhibited hyperalgesia on the left side of her body, while other superficial and deep sensations were symmetrically normal. The left pathological reflex was positive, while the right was negative. Internal medicine examination revealed normal heart and abdomen findings, coarse breath sounds in both lungs, scattered Velcro rales in the lower lobes of both lungs, and no edema in both lower extremities.

Past medical history included a 7-year history of hypertension, which was currently normal without medication. She had a history of osteoporosis and had been taking calcitriol and calcium carbonate for a long time. In July 2022, she was diagnosed with tongue cancer at Yichang Central Hospital and underwent surgical resection followed by postoperative radiotherapy, which was completed on October 23, 2023. MRI in 2022 indicated left femoral head necrosis, while MRI in May 2023 showed bilateral avascular necrosis of the femoral heads.

Cerebrospinal fluid (CSF) pressure was 150 mmH₂O, with a white blood cell count of 6×10^6 /L. CSF biochemistry revealed a protein level of 44.3 mg/dl (reference range: 15-45 mg/dl), with normal glucose and chloride levels. Both CSF and blood tests for autoimmune encephalitis, paraneoplastic syndromes, and demyelinating diseases were negative. Blood MOG antibodies

were positive at a concentration of 1:32, while CSF MOG antibodies were negative. In figure 1, Enhanced magnetic resonance imaging (MRI) showed widespread increased T2 signal intensity in the cerebral white matter of both cerebral hemispheres, extensive thickening of the cranial meninges, and marked meningeal enhancement on contrast-enhanced scans.

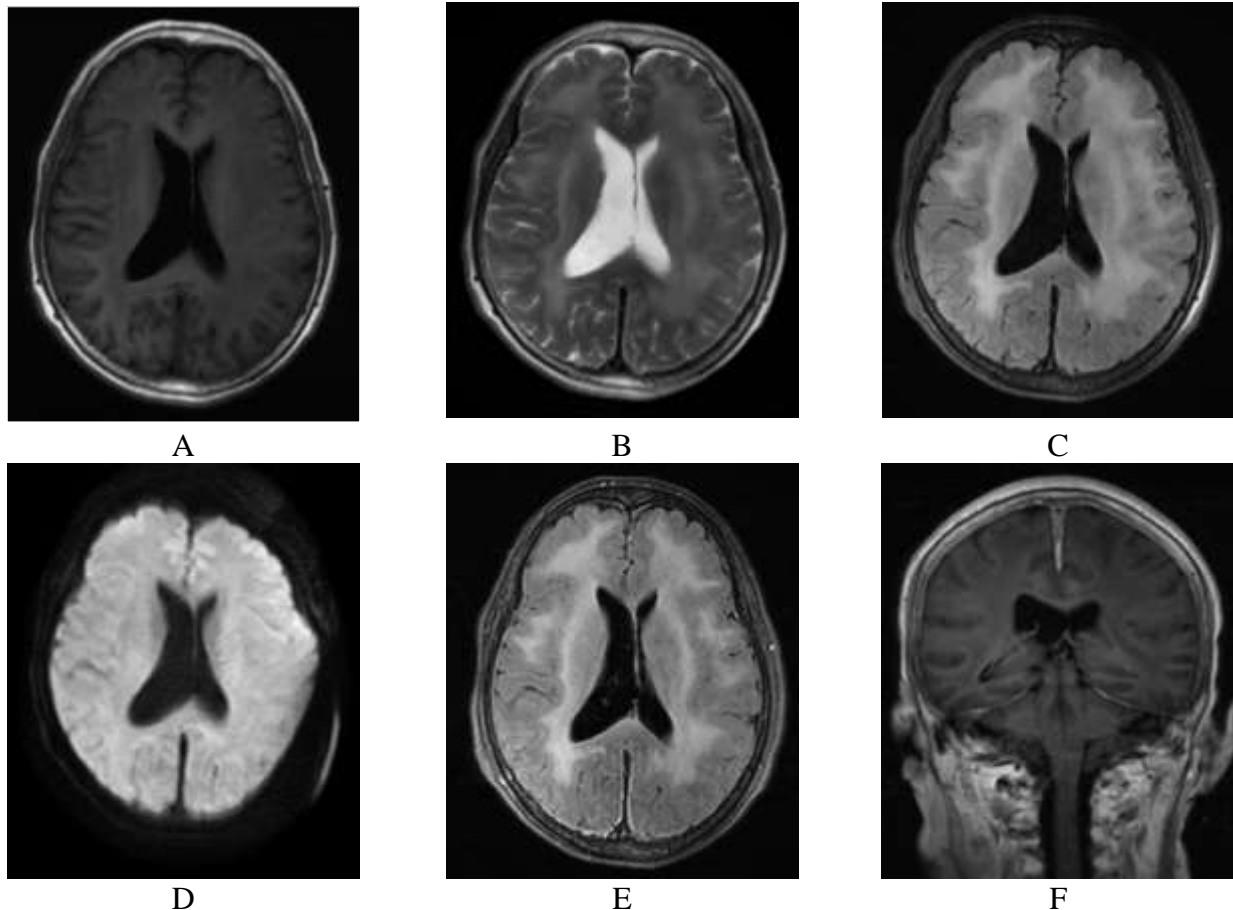


Figure 1. No abnormal signal intensities were identified on the T1-weighted sequence (A) and the DWI sequence (D). Extensive abnormal signal intensities in the cerebral white matter were visualized on the T2-weighted (B) and T2 flair sequences (E). Marked enhancement of extensive regions of the cerebral white matter (E) and meninges was observed on the contrast-enhanced sequences (F).

Treatment: Considering her history of femoral head necrosis and patient preferences, hormone pulse therapy was not administered. On November 9, 2023, she received intravenous immunoglobulin therapy. On November 16, 2023, she was initiated on ofatumumab immunotherapy, with mycophenolate mofetil added concurrently. However, a follow-up enhanced cranial MRI on April 3, 2024, still showed diffuse cerebral white matter edema and degeneration, as well as extensive meningeal thickening and enhancement. The patient continued to experience headaches

during follow-up visits at 6 and 12 months. At the 14-month follow-up, she died of pneumonia.

Results

Through a comprehensive literature search focusing on AAV and the central nervous system, a total of 500 cases were retrieved. However, 142 of these cases lacked detailed documentation regarding central nervous system involvement and were consequently excluded from the analysis. Ultimately, 358 cases were included in the actual analysis.

In table 1, we can clearly observe that the number of cases involving the meninges is considerable, reaching 102 cases and accounting for approximately 28.49% (102/358) of the cases with detailed records. The frontal lobe, an extremely vital functional area of the brain, was involved in 68 cases, representing about 18.99% of the total. The parietal lobe was also not spared, with 58 cases involving this region, accounting for approximately 16.20%. The pituitary gland, a crucial hub for endocrine regulation, was involved in 30 cases, making up about 8.38%. Abnormalities in pituitary function often trigger a series of complex endocrine disorder symptoms, affecting multiple aspects of patients' physical health. The basal ganglia, an important area for motor regulation, were involved in 23 cases, accounting for about 6.42%. Its involvement may lead to issues such as paralysis, severely impacting patients' quality of life. The temporal lobe, which plays a significant role in hearing, language comprehension, and memory, was involved in 32 cases, representing about 8.94%, which may pose challenges to patients' auditory

perception and language communication abilities. The brainstem was involved in 17 cases, accounting for about 4.75%. The spinal cord, an important conduit connecting the brain to the peripheral nerves, was involved in 15 cases, representing about 4.19%. Spinal cord lesions can result in sensory and motor dysfunction in the limbs, causing immense suffering to patients. The occipital lobe was involved in 26 cases, accounting for about 7.26%. The thalamus, a relay station for sensory conduction, was involved in 12 cases, representing about 3.35%.

Additionally, there were relatively fewer cases involving certain other sites. The number of cases involving the following sites was all less than 10: cerebellum (7 cases), centrum semiovale (1 case), middle cerebral artery territory (1 case), corpus callosum (1 case), periventricular region (4 cases), corona radiata (4 cases), insular lobe (3 cases), periorbital and sellar regions (5 cases), subarachnoid space (5 cases), ventricles and choroid plexus (4 cases), venous sinus (2 cases), optic nerve (2 cases), dura mater (8 cases), and spinal subarachnoid space (1 case).

Table1. The table presents detailed information on the involvement of central nervous system regions in ANCA-associated vasculitis.

| Lesion site | Case count |
|----------------------------------|------------|
| meninges | 102 |
| Frontal lobe | 68 |
| Parietal lobe | 58 |
| Occipital lobe | 26 |
| Temporal lobe | 32 |
| Basal ganglia region | 23 |
| Pituitary gland | 30 |
| Brainstem | 17 |
| Thalamus | 12 |
| Cerebellum | 7 |
| Paraventricular | 4 |
| Corona radiata | 4 |
| Insular lobe | 3 |
| Centrum semiovale | 1 |
| Middle cerebral artery territory | 1 |
| Corpus callosum | 1 |
| Periorbital and sellar regions | 5 |
| Subarachnoid space | 5 |
| Ventricles and choroid plexus | 4 |
| Venous sinus | 2 |
| Optic nerve | 2 |
| Spinal cord | 15 |

| | |
|-------------------------------|---|
| Dura mater of the spinal cord | 8 |
| Spinal subarachnoid space | 1 |

In table 2, Headache emerged as a prominent symptom, affecting 129 patients. Regarding consciousness and cognitive symptoms, confusion and language disorders were noted in 45 and 48 patients respectively, with psychiatric disorders seen in 25 cases and cognitive impairment in 2. Among visual-related symptoms, reduced visual acuity was most common, occurring in 59 patients, followed by diplopia (31 cases), optic neuritis (29 cases), visual field defect + hemianopia (10 cases), cortical blindness (4 cases), and central retinal artery occlusion (3 cases), with 2 cases also showing abnormal gaze; these symptoms indicate potential involvement of various segments of the visual pathway, from the eyeball to the optic nerve and visual cortex, leading to diverse visual dysfunctions. Auditory symptoms included hearing loss in 27 patients, suggesting auditory system involvement, while olfactory dysfunction was rare, with only 2 cases.

Motor disorders were significant, affecting 95 patients, including facial paralysis (17 cases), dysphagia (16 cases), tongue movement disorders (3 cases), and gait abnormalities (9 cases). Sensory disorders were present in 65 patients, and ataxia was observed in 10 patients, with 3 cases lacking specific type details; among the clearly defined types, vestibular ataxia accounted for 4 cases, cerebellar ataxia for 2, and proprioceptive ataxia for 1. Neuroendocrine symptoms were seen in 18 patients, indicating possible effects on neuroendocrine regulatory centers such as the hypothalamus-pituitary axis, leading to hormonal imbalances and related symptoms. Autonomic nervous symptoms included urinary and fecal incontinence in 7 patients, reflecting potential autonomic dysfunction. Special signs were rare, with only 1 patient each presenting with Horner's syndrome and trapezius muscle weakness.

Table 2. This table displays the clinical signs observed in 358 cases of ANCA-associated vasculitis.

| clinical signs | Case count |
|----------------------------------|------------|
| Headaches | 129 |
| disturbance of consciousness | 45 |
| Speech disorders | 48 |
| Seizures | 47 |
| Psychiatric disorders | 25 |
| cognitive impairment | 2 |
| Loss of visual acuity | 59 |
| diplopia | 31 |
| optic neuritis | 29 |
| visual field defect | 10 |
| cortical blindness | 4 |
| Central retinal artery occlusion | 3 |
| gaze | 2 |
| ptosis | 9 |
| Vestibular syndrome | 10 |
| olfactory dysfunction | 2 |
| Hearing impairment | 27 |
| Movement disorders | 95 |
| Sensitive deficiency | 65 |
| ataxia | 10 |
| vestibular ataxia | 4 |
| Cerebellar ataxia | 2 |
| proprioceptive ataxia | 1 |
| Gait abnormalities | 9 |
| facial palsy | 17 |

| | |
|-------------------------------|----|
| dysphagia | 16 |
| dysglossia | 3 |
| neuroendocrine dysfunction | 18 |
| bowel and bladder dysfunction | 7 |
| Horner's syndrome | 1 |
| trapezius muscle weakness | 1 |

Table3. The table presents the clinical manifestations of ANCA-associated vasculitis involving the central nervous system.

| Clinical manifestations | Case count |
|--|------------|
| Ischemic cerebrovascular disease | 97 |
| Hypertrophic cranial pachymeningitis | 80 |
| Pituitary lesion | 29 |
| Intracerebral hemorrhage | 31 |
| Subarachnoid hemorrhage | 17 |
| Vasculitis | 16 |
| Spinal cord lesion | 16 |
| Posterior Reversible Encephalopathy Syndrome (PRES) | 16 |
| Tumor-like lesion | 11 |
| Inflammatory mass | 10 |
| Meningitis and/or encephalitis | 10 |
| Demyelinating encephalopathy | 8 |
| Progressive multifocal leukoencephalopathy (PML) | 4 |
| Hemorrhagic cerebral infarction | 4 |
| Venous sinus thrombosis | 4 |
| Epilepsy | 2 |
| Chronic subdural hematoma | 2 |
| Hypertrophic spinal pachymeningitis | 2 |
| Cerebral hernia | 2 |
| Uveitis | 1 |
| Orbital apex syndrome | 1 |
| Subdural abscess | 1 |
| Mass in the fourth ventricle | 1 |
| Reversible Cerebral Vasoconstriction Syndrome (RCVS) | 1 |
| Optic neuritis | 1 |
| Cerebral Amyloid Angiopathy-like appearance | 1 |
| Granuloma | 1 |
| Frontal lobe syndrome | 1 |
| Brain abscess | 1 |

Table 3 delineates the distribution of neurological manifestations in patients with AAV, categorizing diseases based on the frequency of affected cases. Among these manifestations, ischemic cerebrovascular disease emerges as the most prevalent condition, with 97 documented cases, followed by hypertrophic pachymeningitis (80 cases), cerebral hemorrhage (31 cases), and pituitary lesions (29 cases), collectively representing the highest-incidence group.

Diseases with a moderate incidence include subarachnoid hemorrhage (17 cases), posterior reversible encephalopathy syndrome (PRES, 16 cases), spinal cord lesions (16 cases), and tumor-like lesions (11 cases), indicating a notable but less frequent subset of neurological complications.

In contrast, conditions with a relatively low incidence (≤ 10 cases) encompass inflammatory masses (10 cases), meningitis and/or encephalitis (10 cases), demyelinating encephalopathy (8

cases), progressive multifocal leukoencephalopathy (PML, 4 cases), hemorrhagic cerebral infarction (4 cases), venous sinus thrombosis (4 cases), epilepsy (2 cases), chronic subdural hematoma (2 cases), hypertrophic spinal pachymeningitis (2 cases), and cerebral hernia (2 cases). Additionally, isolated cases (n=1) were identified for uveitis, orbital apex syndrome, subdural abscess, mass in the fourth ventricle, optic neuritis, cerebral amyloid angiopathy-like appearance, granuloma, frontal lobe syndrome, and brain abscess, underscoring the heterogeneous and occasionally rare nature of neurological involvement in this vasculitic syndrome.

Discussion

We report the first case of AAV coexisting with MOG antibody disease (MOGAD). Currently, AAV is considered an ANCA-mediated immune disorder, with its primary target antigens being proteinase-3 (PR3) and myeloperoxidase (MPO). MOG-related diseases are mediated by anti-myelin oligodendrocyte glycoprotein immunoglobulin G antibodies (MOG-IgG), which target MOG proteins on the surface of oligodendrocytes, leading to myelin injury and loss. To date, no studies have explored the relationship between PR3 antibodies and MOG antibodies. Three studies utilized MOG-induced experimental autoimmune encephalomyelitis (EAE) models to assess biochemical markers, including MPO[6-8]. However, none of these articles explicitly addressed the direct relationship between MPO and MOG. Thus, there remains a lack of foundational research linking AAV and MOGAD. As MPO functions as an immune effector molecule and MOG acts as a myelin protein, no direct connection between them has been established. Future studies we will explore potential interactions between MOG and MPO in neuroimmune diseases.

In this reported case, the patient initially presented with interstitial pneumonia, later developing peripheral neuropathy without renal involvement. Laboratory findings revealed positive perinuclear ANCA, MPO antibodies, and PR3 antibodies, supporting a diagnosis of AAV—specifically eosinophilic granulomatosis with polyangiitis (EGPA). EGPA frequently involves the nervous

system, predominantly affecting peripheral nerves[9]. André et al. documented 26 new cases of central nervous system (CNS) involvement in EGPA and retrospectively analyzed 62 previously reported cases. Among these, 86% exhibited CNS manifestations at EGPA diagnosis, while 2% developed CNS symptoms prior to EGPA confirmation[10]. Diagnosing EGPA becomes particularly challenging when CNS symptoms precede typical systemic features.

In a comprehensive analysis of the literature review involving 358 cases of AAV affecting the central nervous system, Table 1 reveals notable variations in CNS involvement, with meninges most frequently implicated (102 cases, ~28.49%), followed by frontal (68 cases, ~18.99%) and parietal lobes (58 cases, ~16.20%), while pituitary gland (30 cases, ~8.38%), basal ganglia (23 cases, ~6.42%), temporal lobe (32 cases, ~8.94%), brainstem (17 cases, ~4.75%), spinal cord (15 cases, ~4.19%), occipital lobe (26 cases, ~7.26%), and thalamus (12 cases, ~3.35%) also affected, with other regions like cerebellum showing lower involvement. Table 2 details clinical symptomatology, with headache predominant (129 patients), alongside consciousness/cognitive disturbances (confusion, language disorders, etc.), diverse visual symptoms (reduced acuity, diplopia, etc.), auditory (hearing loss), motor (facial paralysis, dysphagia, etc.), sensory, and ataxic manifestations, as well as neuroendocrine and autonomic dysfunction, with special signs being rare. Table 3 stratifies neurological manifestations by incidence, highlighting ischemic cerebrovascular disease (97 cases), hypertrophic pachymeningitis (80 cases), cerebral hemorrhage (31 cases), and pituitary lesions (29 cases) as high-incidence conditions, with subarachnoid hemorrhage, PRES, spinal cord lesions, and tumor-like lesions showing moderate incidence, and inflammatory masses, meningitis/encephalitis, and others demonstrating low incidence, alongside isolated cases of rare conditions, collectively underscoring the heterogeneous clinical spectrum of AAV and the necessity for tailored diagnostic and therapeutic approaches based on the prevalence and severity of specific neurological manifestations. Consistent with our findings, Wu et al. reported headache and stroke as cardinal features of AAV [11]. Yasuhiro's research highlights headache as the hallmark presentation of ANCA-associated

pachymeningitis, with seizures being another common manifestation[12].

Limitations: Among the 500 retrieved cases of AAV involving the central nervous system, 142 cases were excluded from statistical analysis due to the absence of detailed descriptions of clinical or imaging features specific to the central nervous system. Furthermore, the earliest included studies date back to 1961, raising concerns about potential misdiagnoses and missed diagnoses attributed to the limited diagnostic criteria available at that time.

In summary, this study reports the first case of AAV comorbid with MOGAD and systematically reviews CNS manifestations of AAV. Key features include headache, aphasia, seizures, and altered consciousness. Differential diagnosis is essential for pachymeningitis and stroke. Future research we will investigate potential links between MOG and MPO.

Conclusion

We present a rare case of AAV coexisting with MOG-related disease. Through literature review and analysis of central nervous system manifestations in AAV, we found that hypertrophic pachymeningitis and ischemic stroke were the predominant features. Although AAV is not a common etiology for either hypertrophic pachymeningitis or ischemic stroke, clinicians should consider differentiating AAV during the diagnostic workup of these conditions.

Declarations

Ethical approval This study is a retrospective study. On July 28, 2025, 2023, I proposed the research plan and submitted it to the Medical Ethics Committee of Yichang Central People's Hospital. The Ethics Committee approved the plan on August 1st 2025, with approval number 2025-254-01.

Conflict of interest The authors declare no competing interests.

Funding information

No funding received

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