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TRANSIENT OLIGOCLONAL BANDS AND COMPLETE BRAIN LESION RESOLUTION IN PEDIATRIC MOGAD INITIALLY DIAGNOSED AS MULTIPLE SCLEROSIS: A 30-MONTH CASE REPORT

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ABSTRACT

Background: Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) can overlap clinically and radiologically with pediatric-onset multiple sclerosis (MS). Transient cerebrospinal fluid (CSF) oligoclonal bands (OCBs), which may substitute for dissemination in time under the 2017 McDonald criteria, can contribute to diagnostic misclassification when interpreted without disease-specific serology.
Case Presentation: A 15-year-old girl presented with binocular diplopia, dysarthria, and balance impairment. Brain MRI showed multiple non-enhancing lesions, and spinal MRI demonstrated multilevel intramedullary lesions. CSF was acellular with CSF-restricted OCBs. The findings were interpreted as fulfilling the 2017 McDonald criteria, and interferon beta-1b was initiated. At month 9, serum MOG-IgG was positive by cell-based assay (titer 1:100), while AQP4-IgG was negative. At month 14, the cerebral lesions had resolved completely, repeat CSF OCB testing was negative, and MOG-IgG remained positive. The diagnosis was reclassified as MOGAD, interferon beta-1b was discontinued, and maintenance intravenous immunoglobulin was initiated at month 18. At month 30, the patient remained clinically stable with an EDSS score of 0; the last documented MOG-IgG result at month 25 remained positive.
Discussion: The longitudinal combination of transient OCB positivity and complete radiological resolution of extensive brain lesions prompted diagnostic reassessment. The case illustrates why formal fulfillment of MS criteria should be integrated with the clinical phenotype, disease-specific antibody testing, and serial MRI findings in pediatric demyelinating disease.
Conclusions: Early serum MOG-IgG testing should be considered in children with a first demyelinating event, particularly when the phenotype or subsequent MRI evolution is atypical for MS. CSF OCB positivity should not be regarded as disease-specific evidence of MS in isolation.

KEYWORDS

MOGAD; Pediatric Demyelination; Misdiagnosis; Multiple Sclerosis; Oligoclonal Bands; Intravenous Immunoglobulin

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Introduction

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) has established itself over the past decade as an immunologically and clinically distinct autoimmune inflammatory demyelinating entity of the central nervous system (CNS) [1, 2]. Pathophysiologically driven by autoantibodies targeting the extracellular domain of MOG expressed on oligodendrocytic surface membranes, MOGAD encompasses a diverse spectrum of clinical manifestations including optic neuritis (ON), transverse myelitis (TM), acute disseminated encephalomyelitis (ADEM), and brainstem or cortical encephalitis [3]. The epidemiological landscape of pediatric inflammatory demyelination differs substantially from adults: MOGAD accounts for up to 40% to 60% of non-MS acquired demyelinating syndromes (ADS) in children, whereas pediatric-onset multiple sclerosis (POMS) represents a less common presentation at initial disease onset [4, 5].

Differentiating MOGAD from pediatric multiple sclerosis (POMS) and neuromyelitis optica spectrum disorder (NMOSD) during the first clinical episode remains a formidable diagnostic challenge [6, 7]. In adolescent patients presenting with polyfocal neurological deficits or simultaneous optic neuritis and spinal cord involvement, clinical features frequently overlap with classic MS syndromes [6]. Furthermore, the diagnostic criteria for multiple sclerosis applied in the present case in their McDonald 2017 version, which was in clinical use at the time of this patient's diagnosis and has since been superseded by the 2024 revision were designed to maximize sensitivity for MS but carry a recognized risk of false-positive classification when applied to non-MS demyelinating conditions such as MOGAD and NMOSD [7, 8, 9]. Both the 2017 and 2024 McDonald criteria permit positive cerebrospinal fluid (CSF) oligoclonal bands (OCBs) or the kappa free light

chain index in the 2024 revision to substitute for dissemination in time (DIT), allowing a single clinical event with space-disseminated lesions and intrathecal humoral activity to fulfill formal diagnostic requirements for definite MS.

Crucially, while intrathecal synthesis of persistent OCBs serves as a cardinal immunological hallmark of MS present in over 85% to 95% of patients lifelong, transient CSF-restricted OCBs can occur in 10% to 15% of patients with MOGAD during acute neuroinflammatory peaks, vanishing upon resolution of the acute attack [10, 11]. When pediatric MOGAD presents transient OCBs, formal fulfillment of McDonald criteria may result in an erroneous diagnosis of MS and subsequent initiation of MS-specific disease-modifying therapies (DMTs) such as interferon-beta or glatiramer acetate. Accumulating evidence indicates that immunomodulatory therapies targeting MS pathways particularly interferon-beta are ineffective in MOGAD and may be associated with paradoxical disease exacerbation [12, 13].

Herein, we report a detailed 30-month longitudinal case of a 15-year-old girl who presented with polyfocal demyelination and transient CSF OCBs, initially leading to a diagnosis of MS and 12 months of interferon beta-1b treatment. Diagnostic reassessment prompted by complete brain lesion resolution on serial MRI identified persistent serum MOG-IgG antibodies (cell-based assay, titer 1:100) and negative repeat OCBs, supporting reclassification as MOGAD according to the 2023 International MOGAD Panel criteria [1]. The patient was transitioned to intravenous immunoglobulin (IVIg) maintenance and remained clinically stable at the 30-month follow-up. This report highlights diagnostic pitfalls, longitudinal radiological evolution, therapeutic implications, and treatment-adherence considerations in pediatric MOGAD.

Case Presentation

Patient information and initial clinical findings

A 15-year-old girl was referred to the Department of Pediatric Neurology at the index presentation (M0) because of subacute neurological symptoms. She reported binocular diplopia of 3 days' duration, together with progressive dysarthria and mild balance impairment evolving over the preceding 6 weeks. Ophthalmoscopy showed bilateral blurring of the optic-disc margins without definite disc swelling. Cranial computed tomography demonstrated cerebellar tonsils extending 3-4 mm below the foramen magnum and was otherwise unremarkable. Her medical history included grass-pollen allergy and brief over-the-counter cannabidiol supplementation that had been discontinued approximately 2 years before symptom onset and was considered unrelated to the presentation by the treating team. A first-degree relative had primary hypothyroidism. Neurological examination showed mild scanning dysarthria, dysmetria on finger-to-nose testing, and subtle instability during tandem gait. No focal motor or sensory deficit was identified. The Expanded Disability Status Scale (EDSS) score was not formally recorded at the initial admission rate.

Initial diagnostic assessment and treatment

At M1, brain magnetic resonance imaging (MRI) with gadolinium demonstrated multiple non-enhancing T2/FLAIR-hyperintense lesions involving the corpus callosum, pons, thalami, subcortical white matter, and a cerebellar hemisphere; the largest lesion measured 13 mm. Cervical and thoracic spinal MRI showed multiple intramedullary lesions at C1/C2 (27 x 6 x 8 mm), C4 (17 x 6 x 8 mm), C5 (10 x 3 x 3 mm), C6 (approximately 10 mm), Th2/3, Th5/6/7, and Th10/11/12. CSF was clear, acellular, and biochemically normal. CSF-restricted OCBs were reported as positive in verified institutional discharge documentation; the original laboratory printout was unavailable for retrospective review. An antinuclear antibody panel was negative, whereas serum complement C3 and C4 concentrations were mildly decreased. Visual evoked potentials obtained at M2 showed bilateral prolongation of P100 latency.

The combination of a first polyfocal demyelinating event, MRI lesions in more than one central nervous system region, and CSF OCB positivity was interpreted as fulfilling the 2017 McDonald criteria, with OCBs substituting for dissemination in time [9]. A diagnosis of relapsing-remitting multiple sclerosis was made. The patient received a weight-adjusted pulse of intravenous methylprednisolone according to the institutional protocol, with resolution of the presenting neurological symptoms. Subcutaneous interferon beta-1b (Betaferon; 250 micrograms every other day) was initiated at M2.

Longitudinal evolution and diagnostic reassessment

At M5, the neurological examination was normal (EDSS 0), although the patient reported subjective memory impairment and mild difficulty concentrating. Brain MRI at M9 showed marked regression of the previously disseminated T2/FLAIR abnormalities. Serum testing performed at a national reference neuroimmunology laboratory detected MOG-IgG by a cell-based assay at a titer of 1:100; serum aquaporin-4 IgG was negative. The available report did not specify whether a live or fixed cell-based platform was used. Interferon beta-1b was continued while the diagnostic reassessment was completed.

At M14, the patient remained neurologically asymptomatic with an EDSS score of 0. Repeat brain MRI showed complete resolution of the previously identified cerebral, brainstem, and cerebellar lesions, without residual gliosis or T1-hypointense lesions. Cervical spinal MRI demonstrated residual, consolidated lesions at C2 and C4. Contrast-enhanced orbital MRI showed bilateral optic-nerve thickening (7.3 mm on the left and 6.9 mm on the right), optic-nerve sheath enhancement, and edema of the surrounding perineural fat. Repeat CSF testing was negative for OCBs. Serum MOG-IgG remained positive at a titer of 1:100 by cell-based assay, and anti-thyroid peroxidase and anti-thyroglobulin antibodies were negative. On the basis of the clinical syndrome, persistent serum MOG-IgG positivity, and supportive longitudinal MRI findings, the diagnosis was reclassified as MOGAD according to the 2023 International MOGAD Panel criteria [1]. Interferon beta-1b was discontinued after approximately 12 months of treatment.

Subsequent treatment and treatment-related events

At M16, a further 3-day pulse of intravenous methylprednisolone was administered. The available documentation did not specify whether this treatment was given for a new clinical relapse, radiological activity, or another indication. During the infusion, the patient developed sinus tachycardia (95-112 beats/min). Electrocardiography showed first-degree atrioventricular block, and Holter monitoring documented a 3.3-second pause. B-type natriuretic peptide was elevated to 1282 pg/mL, whereas troponin was normal. Thyroid testing showed reduced thyroid-stimulating hormones, free triiodothyronine, and free thyroxine concentrations. Cardiology and endocrinology consultations were obtained.

At M18, endocrine assessment resulted in a diagnosis of secondary (central) hypothyroidism, and levothyroxine 62.5 micrograms once daily was started. Increased fasting insulin, vitamin D deficiency, and mixed hyperlipidemia were also documented. Maintenance IVIg was initiated using a weight-adjusted regimen, with infusions scheduled every 6-7 weeks; the dose in g/kg and infusion protocol were not available in the source documentation reviewed for this report.

At M23, the patient developed pain and swelling of the left upper arm after peripheral venous cannulation. Doppler ultrasonography demonstrated thrombosis of the left cephalic vein extending from the antecubital region to its proximal third. Baseline coagulation testing was reported as normal, and an evaluation of prothrombotic risk was undertaken. She was treated with weight-adjusted subcutaneous enoxaparin, followed by topical heparin. Follow-up ultrasonography after 7 days showed venous patency, and the thrombosis subsequently resolved.

During the M25 admission, severe needle-related anxiety caused venous access. The scheduled IVIg infusion was ultimately administered, but the patient declined further cannulation attempts and a proposed oral prednisone regimen. A clinical psychology consultation was arranged to support treatment of adherence. Serum MOG-IgG remained positive by cell-based assay. Cervical spinal MRI showed stable residual lesions at C2 and C4, and orbital MRI demonstrated a reduction in optic-nerve diameter to 6.0 mm.

Follow-up and outcome

At the last documented follow-up (M30), no additional objectively documented neurological attacks had occurred after initiation of maintenance IVIg. The neurological examination remained normal (EDSS 0). Subjective memory difficulties persisted, although formal neuropsychological testing had not been completed. Pituitary MRI showed normal morphology of the gland and stalk without focal abnormalities. The patient was euthyroid while receiving levothyroxine and continued regular IVIg maintenance therapy.

Table 1. Longitudinal clinical, radiological, laboratory, and therapeutic timeline (M0-M30).

Time	Clinical symptoms and events	Neuroimaging findings	Laboratory and serological results	Therapeutic interventions
M0	Binocular diplopia (3 days), progressive dysarthria and balance impairment (6 weeks); blurred optic-disc margins.	CT: cerebellar tonsils 3-4 mm below the foramen magnum; otherwise unremarkable.	Not applicable.	Referral to pediatric neurology.
M1	First polyfocal neurological episode.	Brain MRI: multiple non-enhancing T2/FLAIR lesions in the corpus callosum, pons, thalami, subcortical white matter, and cerebellum. Spine MRI: multilevel lesions from C1/C2 to Th12.	CSF acellular and biochemically normal; CSF-restricted OCBs reported positive.	Weight-adjusted IV methylprednisolone; diagnosis of RRMS.
M2	Clinical recovery.	VEP: bilateral P100 latency prolongation.	ANA negative; mildly decreased C3/C4.	Interferon beta-1b 250 micrograms subcutaneously every other day initiated.
M5	Neurological examination normal (EDSS 0); subjective memory and concentration difficulties.	No new imaging reported.	Mildly reduced hemoglobin; otherwise unremarkable blood tests.	Interferon beta-1b continued.
M9	Assessment of subjective cognitive complaints.	Brain MRI: marked regression of previous T2/FLAIR lesions.	Serum MOG-IgG positive (CBA, 1:100); AQP4-IgG negative.	Interferon beta-1b continued during diagnostic reassessment.
M14	Neurologically asymptomatic (EDSS 0).	Brain MRI: complete resolution of previous brain lesions. Cervical MRI: residual C2/C4 lesions. Orbital MRI: bilateral optic-nerve thickening with sheath enhancement and perineural edema.	Repeat CSF OCBs negative; serum MOG-IgG positive (CBA, 1:100); anti-TPO/anti-TG negative.	Diagnosis reclassified as MOGAD; interferon beta-1b discontinued.
M16	Further IV methylprednisolone pulse; indication not specified. Sinus tachycardia during infusion.	No new neuroimaging reported.	ECG: first-degree AV block; Holter: 3.3-second pause; BNP 1282 pg/mL; troponin normal; low TSH, fT3, and fT4.	Three-day IV methylprednisolone course; cardiology and endocrinology review.
M18	Maintenance treatment initiation.	No new neuroimaging reported.	Secondary hypothyroidism recorded; increased fasting insulin, vitamin D deficiency, and mixed hyperlipidemia.	IVIg initiated every 6-7 weeks; levothyroxine 62.5 micrograms/day.
M22	Routine maintenance admission.	No new imaging was reported.	No new relevant laboratory results reported.	IVIg maintenance infusion.

Time	Clinical symptoms and events	Neuroimaging findings	Laboratory and serological results	Therapeutic interventions
M23	Left upper-arm pain and swelling after peripheral cannulation.	Doppler: thrombosis of the left cephalic vein; follow-up at 7 days showed patency.	Baseline coagulation profile reported as normal; prothrombotic evaluation undertaken.	Weight-adjusted enoxaparin followed by topical heparin; subsequent resolution.
M25	Severe needle-related anxiety; IVIg ultimately administered; psychology consultation.	Cervical MRI: stable C2/C4 lesions. Orbital MRI: optic-nerve diameter reduced to 6.0 mm.	Serum MOG-IgG remained positive by CBA.	IVIg continued; proposed oral prednisone declined.
M30	Clinically stable (EDSS 0); persistent subjective memory complaints.	Pituitary MRI: normal gland and stalk morphology.	Euthyroid on levothyroxine.	Ongoing regular IVIg maintenance.

Abbreviations: AQP4-IgG, aquaporin-4 immunoglobulin G; CBA, cell-based assay; CSF, cerebrospinal fluid; EDSS, Expanded Disability Status Scale; IVIg, intravenous immunoglobulin; MOG-IgG, myelin oligodendrocyte glycoprotein immunoglobulin G; OCBs, oligoclonal bands; RRMS, relapsing-remitting multiple sclerosis; VEP, visual evoked potentials.

Discussion

Learning Point 1: Transient Oligoclonal Bands and Diagnostic Overlap

The presence of CSF-restricted OCBs at the initial presentation was central to the interpretation of the case under the 2017 McDonald criteria. In MS, intrathecal oligoclonal IgG synthesis is detected in most patients and is generally persistent [9]. By contrast, OCBs are reported in a minority of patients with MOGAD and may be transient during acute inflammatory activity [10, 11]. In this patient, OCB positivity at M1 contributed to fulfillment of the dissemination-in-time criterion, whereas repeat testing at M14 was negative. This longitudinal change illustrates that OCB positivity is not disease-specific and should be interpreted alongside the clinical phenotype, MRI characteristics, and disease-specific antibody testing.

The patient's CSF was acellular. Although pleocytosis is common during acute MOGAD attacks, its absence does not exclude the diagnosis. In pediatric patients with a first demyelinating event, a positive OCB result should therefore not be accepted as definitive evidence of MS without consideration of serum MOG-IgG and AQP4-IgG testing.

The subsequently published 2024 revision of the McDonald criteria does not remove the need for careful differential diagnosis [8]. The optic nerve is included as an additional topographic location, and OCBs or the kappa free light chain index may contribute to demonstration of dissemination in time. Neither the central vein sign nor paramagnetic rim lesions was documented in this patient. This case therefore emphasizes that formal criteria should be applied only after consideration of alternative inflammatory demyelinating disorders.

Learning Point 2: Complete Brain Lesion Resolution as a Diagnostic Clue

A key longitudinal observation was the complete radiological resolution of the widespread brain T2/FLAIR lesions between M1 and M14. No formal volumetric segmentation was performed; accordingly, the finding is best described as complete radiological rather than volumetric resolution. Persistent T2 abnormalities and T1-hypointense lesions are more typical of established MS, whereas substantial or complete lesion resolution is well recognized in MOGAD [5, 14, 15].

In this patient, disappearance of the cerebral, brainstem, and cerebellar lesions, together with residual spinal abnormalities and supportive orbital MRI findings, prompted diagnostic reassessment. The case illustrates the value of serial imaging: longitudinal lesion evolution may be more informative than lesion distribution on a single baseline scan and should trigger reconsideration when it is atypical for the working diagnosis.

Learning Point 3: Interferon-Beta Exposure and Therapeutic Implications

Interferon beta-1b was initiated under the initial diagnosis of MS and discontinued after reclassification as MOGAD. This single case cannot establish treatment of ineffectiveness, particularly because no clearly documented clinical relapse occurred during interferon exposure, and the brain lesions regressed. Nevertheless, MS disease-modifying therapies are not established maintenance treatments for MOGAD, and pediatric cohort data support use of MOGAD-directed relapse-prevention strategies in patients with recurrent disease [12, 13].

Maintenance IVIg was initiated at M18, after which clinical stability was documented through M30. Because this is an uncontrolled single-patient observation and the indication for the M16 steroid pulse was not recorded; a causal treatment effect cannot be inferred. Serum MOG-IgG remained positive at M25. Persistent seropositivity has been associated with a relapsing course in cohort studies, but it should be interpreted together with the clinical trajectory and should not alone determine long-term treatment [2, 16].

The therapeutic landscape of MOGAD continues to evolve. Recent observational data have evaluated interleukin-6 receptor blockade as a relapse-prevention strategy [17]. Comparative and randomized evidence remains limited, particularly in children, underscoring the need to individualize maintenance of treatment according to attack history, residual disability, treatment burden, and safety.

Management of Adverse Events and Adolescent Treatment Adherence

At M23, the patient developed thrombosis of the left cephalic vein after peripheral venous cannulation during the period of IVIg treatment. The cephalic vein is part of the superficial venous system; therefore, the event should not be labeled as deep-vein thrombosis unless extension into the deep venous system is documented. IVIg has been associated with thromboembolic events, and local vascular trauma may also have contributed [18]. The temporal association in this case does not establish causality. The thrombosis was resolved following anticoagulant treatment.

During the M16 corticosteroid pulse, sinus tachycardia, first-degree atrioventricular block, a 3.3-second pause on Holter monitoring, elevated BNP, and abnormalities of the thyroid axis were documented. Cardiology and endocrinology assessments were undertaken, and secondary hypothyroidism was subsequently treated with levothyroxine. The available data does not permit a definitive causal attribution to corticosteroids, MOGAD, or another mechanism.

By M25, severe needle-related anxiety had become a major barrier to repeated venous access. Psychological support was incorporated into the treatment plan. This aspect of the case emphasizes that long-term management in adolescents should include prospective assessment of procedural distress, treatment burden, and adherence, in addition to neurological outcomes.

Limitations

Several limitations should be acknowledged. The original printout from the initial OCB analysis was unavailable, and the result was extracted from verified discharge documentation. The reference laboratory report did not specify whether a live or fixed MOG-IgG cell-based assay was used. Exact patient-specific doses and infusion parameters for IV methylprednisolone and IVIg were not available in the source of documentation. The indication for the M16 methylprednisolone pulse was not recorded, preventing reliable classification of that event as a relapse. The available clinical data did not include detailed visual-function measures, optical coherence tomography, or neurological findings sufficient to confirm symptomatic optic neuritis or myelitis independently of imaging. Formal neuropsychological testing was not performed despite subjective memory complaints. Finally, this retrospective single-patient report cannot establish treatment efficacy, prognostic value, or causality of adverse events.

Patient perspective

Due to the patient's transition into adulthood and subsequent transfer to adult healthcare services, her long-term perspective and experiences with the disease and its treatment are no longer available. Consequently, a first-person patient perspective could not be obtained for this report.

Conclusions

This case highlights the diagnostic overlap between pediatric MOGAD and MS and supports early serum MOG-IgG testing in children with a first demyelinating event, particularly when the phenotype or longitudinal MRI evolution is atypical for MS. Transient CSF OCBs should not be regarded as definitive evidence of MS in isolation, especially when repeat testing becomes negative and brain lesions resolve completely. Accurate classification is clinically important because MS-specific disease-modifying therapy is not an established treatment for MOGAD; maintenance therapy and safety monitoring should be individualized.

Informed Consent

Consent was obtained from the patient's legal guardian, and assent was obtained from the patient for publication of this case report and any accompanying clinical data.

Declarations

- **Ethics approval and consent to participate:** Not applicable for a single anonymized case report in accordance with Medical University of Lublin institutional policy; the case report was conducted in accordance with the ethical principles of the Declaration of Helsinki.
- **Consent for publication:** Obtained from the patient's legal guardian and the patient, as above.
- **Availability of data and materials:** All de-identified clinical, laboratory, and radiological data supporting the conclusions of this article are included within the manuscript and its table. Original imaging and laboratory source data are available from the corresponding author upon reasonable request, subject to institutional data protection regulations.
- **Competing interests:** The authors declare that they have no competing interests.

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