UC San Diego

UC San Diego Previously Published Works

Title

Clinical Reasoning: A 33-Year-Old Man With Rapidly Progressive Ataxia.

Permalink

https://escholarship.org/uc/item/84r5n0b4

Journal

Neurology, 107(11)

ISSN

0028-3878

Authors

Chandrasekhar, Pallavi

Pham, Vy X

Timple, Liz

et al.

Publication Date

2026-12-08

DOI

10.1212/wnl.0000000000218740

Copyright Information

This work is made available under the terms of a Creative Commons Attribution License, available at https://creativecommons.org/licenses/by/4.0/

Peer reviewed

eScholarship.org Powered by the California Digital Library
University of California

RESIDENT & FELLOW SECTION

Clinical Reasoning: A 33-Year-Old Man With Rapidly Progressive Ataxia

Pallavi Chandrasekhar,[1] Vy X. Pham,[1] Liz Timple,[1] Robert Deiss,[2] Ronald J. Ellis,[3] Anastasie Dunn-Pirio,[3] and Katherine Longardner[3]

Correspondence
Dr. Chandrasekhar
pallavichandrasekhar99@gmail.com

Neurology® 2026;107:e218740. doi:10.1212/WNL.0000000000218740

Abstract

Immunocompromised patients presenting with subacute neurologic deficits pose a diagnostic challenge given the broad differential diagnosis including infections and autoimmune disorders. We describe a 33-year-old man with AIDS who presented with subacute onset of progressive confusion, blurry vision, diplopia, and generalized cerebellar ataxia following hospitalizations for opportunistic infections. This case emphasizes the clinical and diagnostic approach used to refine the differential diagnosis of rhombencephalitis in an immunocompromised patient. Readers will gain an understanding of the various infectious, immunologic, and neoplastic etiologies to consider as well as the interplay between these pathologies.

Section 1

A 33-year-old man with AIDS and recent opportunistic infections (OI) presented with subacute neurologic deficits. He was diagnosed with HIV at age 26 but self-discontinued antiretroviral treatment (ART) at age 30. Six weeks before presentation, he was hospitalized for acute respiratory failure due to *Pneumocystis jirovecii* pneumonia in the setting of advanced immunosuppression (HIV viral load: 530,576 copies/mL; CD4 count: 35 cells/μL). He was restarted on bictegravir/emtricitabine/tenofovir alafenamide with reported adherence. However, 4 weeks before presentation, he was readmitted with fever and headache. Brain MRI at that time was unremarkable. CSF showed normal cell count (4 cells/mm^3) with elevated protein (52 mg/dL, normal: 15–45 mg/dL). Plasma cytomegalovirus (CMV) DNA was detected, and valganciclovir was initiated. On discharge, fever and headache resolved, but mentation was slightly sluggish. One week before presentation, he developed progressive dysarthria, confusion, incoordination, imbalance, and blurred vision over several days. He initially presented to an outside hospital where serum autoimmune and infectious studies were unrevealing. Lumbar puncture showed elevated cell count (7 cells/ mm^3) and protein (75 mg/dL). Brain MRI was completed (findings discussed below).

Two days after discharge, his symptoms worsened, prompting presentation to our hospital. He was alert and awake but confused, with poor recall. Cranial nerve examination was remarkable for bilateral abducens palsies, slowed horizontal saccades, and ataxic dysarthria. Motor and coordination examinations revealed generalized cerebellar ataxia with dysmetria on finger-to-nose and heel-to-shin testing, and dysdiadochokinesia with rapid alternating movements, with more right-sided impairment, without weakness. He exhibited truncal titubation and a wide-based, unsteady gait. Deep tendon reflexes were diffusely brisk, with mute plantar responses. Fundoscopic examination was not possible due to confusion and titubation.

Questions for Consideration:

1. Where can the lesion be localized?
2. What etiologies should be considered?

GO TO SECTION 2

[1]School of Medicine, University of California San Diego; [2]Department of Medicine, University of California San Diego; and [3]Department of Neurosciences, University of California San Diego.

Copyright © 2026 American Academy of Neurology

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Section 2

Truncal ataxia and titubation localize to the cerebellar vermis, while appendicular incoordination implicates the bilateral cerebellar hemispheres. Similar findings may also result from disruption of cerebellar afferent or efferent pathways, including the cerebellar peduncles, dentato-rubro-thalamic tracts, or vestibulocerebellar circuits.

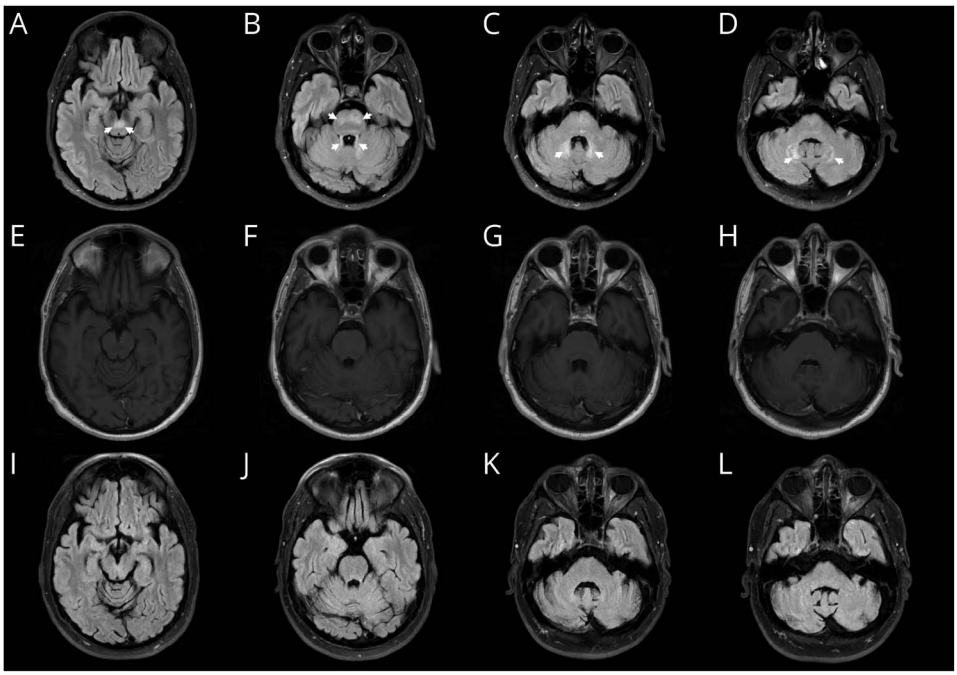
Bilateral abducens palsies localize to the pons or sixth cranial nerves. Dysarthria may arise from dysfunction of cranial nerves VII, IX, X, XII, or upper motor neuron (UMN) or cerebellar pathways. However, prominent cerebellar signs and scanning speech without bulbar or facial deficits supported ataxic dysarthria. Hyperreflexia localizes to the UMN tracts in the contralateral brain or brainstem, or ipsilateral cervical spinal cord.

Consistent with these clinical findings, brain MRI obtained at symptom onset demonstrated T2-hyperintense, nonenhancing lesions involving the bilateral cerebellar dentate nuclei and superior cerebellar peduncles, extending into the midbrain and pons (Figure, A-H). Possible diffusion restriction was noted within the T2 hyperintense lesions, although this finding was not clearly visualized. Right-predominant cerebellar lesions corresponded to more severe ipsilateral symptoms. Superior cerebellar peduncle and dentate nuclei involvement explained postural instability. Abducens nerve palsies explained the diplopia, although assessment of blurred vision was limited. Combined brainstem and cerebellar lesions were consistent with rhombencephalitis.

In this immunocompromised patient with subacute progression, leading etiologies included infectious, inflammatory, neoplastic, and autoimmune/demyelinating processes. OI associated with rhombencephalitis include toxoplasmosis, tuberculosis, listeria, mycoplasma, herpes simplex virus, CMV, Epstein-Barr virus, neurosyphilis, John Cunningham (JC) virus leading to progressive multifocal leukoencephalopathy (PML), and fungal infections.

ART reinitiation raised concern for immune reconstitution inflammatory syndrome (IRIS), which can "unmask" residual infections, trigger autoimmune pathology, or intensify inflammation surrounding preexisting demyelinated lesions. PML-immune reconstitution inflammatory syndrome is a rare condition in which immune recovery triggers an exaggerated inflammatory response to JC virus, causing neurologic deterioration.[1] Given the recent OI, acute disseminated encephalomyelitis (ADEM) characterized by postinfectious inflammatory brain and spinal cord demyelination[2] was also considered. Other possible autoimmune considerations included neuromyelitis optica spectrum disorder (NMOSD)—although this typically presents with optic neuritis, longitudinal transverse myelitis, and/or area postrema syndrome[3]; myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease (MOGAD)—which may manifest with optic neuritis, transverse myelitis, brainstem encephalitis, or ADEM-like features[4];

Figure Initial and 5-Month Follow-Up MRI Brain With and Without Contrast



Axial T2-weighted fluid-attenuated inversion recovery (FLAIR) images at 4 levels demonstrate extensive bilateral hyperintense lesions involving the midbrain tegmentum, pons, superior cerebellar peduncles, and cerebellar dentate nuclei (A–D, arrows), without gadolinium enhancement on T1-weighted postcontrast images (E–H). Five-month follow-up T2-weighted FLAIR images show near-resolution of the lesions (I–L).

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

neurosarcoidosis; and Bickerstaff encephalitis. Primary CNS lymphoma was less likely given the rapid progression and absence of mass-like lesions on MRI without enhancement.

Question for Consideration:

1. What diagnostic tests and additional imaging are most appropriate to refine the differential diagnosis?

GO TO SECTION 3

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

Section 3

To narrow this differential, we performed an extensive evaluation for infectious, autoimmune, and metabolic etiologies (Table). An ultrasensitive JC virus polymerase chain reaction returned negative. Autoimmune testing, including aquaporin-4 and MOG antibodies, was unrevealing. CSF showed elevations in nucleated cells (7 cells/mm^3), Immunoglobulin G (IgG) index (0.68, normal: 0.28–0.66), and myelin basic protein (MBP) (15.3 ng/mL, normal: 0–5.5 ng/mL), with otherwise unremarkable findings. Serum infectious studies were negative aside from elevated HIV viral load (16,100 copies/mL), reduced from prior; CD4 count: 74 cells/μL. Owing to the largely negative laboratory findings and lack of clinical improvement, repeat imaging was performed to assess for lesion progression and appearance of new lesions. Repeat MRI of the brain, orbits, cervical spine, and thoracic spine with and without contrast demonstrated stable-appearing lesions involving the brainstem and cerebellum, without spinal cord involvement.

Questions for Consideration:

1. What is the updated differential diagnosis?
2. Should he receive empiric therapy?

GO TO SECTION 4

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

Table Diagnostic Workup

Test	Results			Reference range
Serum studies				
CD4 count, absolute	6 wk before admission: 35 cells/μL (L)	On admission: 74 cells/μL (L)	HD22: 14 cells/μL (L)	250–1,200 cells/μL
HIV-1 quantitative viral load assay, plasma	1 wk before admission: 347,322 copies/mL	On admission: 16,100 copies/mL	HD14: 149 copies/mL	Not detected
HIV-1 drug resistance by NGS	No resistance to integrase strand transfer inhibitor drug classes, protease inhibitor drug class, or nonnucleoside reverse transcriptase inhibitor drug class. Resistance to nucleoside reverse transcriptase inhibitor drug class agents—emtricitabine and lamivudine			
Toxoplasma gondii PCR	Not detected			Not detected
Cytomegalovirus (CMV) quantitative viral load assay	Not detected			Not detected
Epstein-Barr virus quantitative viral load assay	<35 [IU]/mL			Not detected
Epstein-Barr virus IgM antibody	Negative			Negative
Epstein-Barr virus IgG antibody	Positive			Negative
Cryptococcal antigen screen	Negative			Negative
JC virus antibody	Positive			Negative
BK virus quantitative viral load assay	Not detected			Not detected
Borrelia Burgdorferi total antibodies	0.42			0.00–0.89
HTLV I/II antibodies	Negative			Negative
West Nile virus Ab IgM	0.04 IV			≤0.89 IV
Aquaporin-4 receptor antibody, IgG	<1:10			<1:10
MOG FACS titer	1 wk before admission: Negative, titer: 0	HD13: Positive, titer: 1:100		Negative, <1:20 titer
Ganglioside antibodies with Gq1b	Negative			Negative
ANA	Negative			Negative
Antineutrophil cytoplasmic antibodies	Negative			Negative
SSA antibodies	<0.5 U/mL			<7.0 U/mL
SSB antibodies	<0.6 U/mL			<7.0 U/mL
Anti-MPO antibodies	<0.3 U/mL			<3.5 U/mL
Anti-PR3 antibodies	<1.0 U/mL			<2.0 U/mL
C3	102 mg/dL			90–180 mg/dL
C4	11 mg/dL			10–40 mg/dL
Anti–double-stranded DNA	5 [IU]/mL			<10 [IU]/mL
Vitamin B1	188 nmol/L (H)			70–180 nmol/L
Vitamin B12	310 pg/mL			232–1,245 pg/mL
Mycobacterium tuberculosis antigen	Negative			Negative
Syphilis screen	Positive			Negative
RPR quantitative	Nonreactive			Nonreactive

Continued

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

Table Diagnostic Workup *(continued)*

Test	Results			Reference range
CSF studies				
Total nucleated cells	4 wk before admission: 4 cells/mm^3	1 wk before admission: 7 cells/mm^3 (H)	HD1: 6 cells/mm^3 (H)	0–5 cells/mm^3
RBC	90 cells/mm^3 (H)			0 cells/mm^3
Neutrophils %	2			NA
Lymphocytes %	76			NA
Macrophages %	22			NA
Number of cells counted	100			NA
Total protein	4 wk before admission: 52 mg/dL (H)	1 wk before admission: 75 mg/dL (H)	HD1: 47 mg/dL (H)	15–45 mg/dL
Glucose	4 wk before admission: 48 mg/dL	1 wk before admission: 44 mg/dL	HD1: 53 mg/dL	40–70 mg/dL
Culture	No growth			No growth
Gram stain	No organisms seen			No organisms seen
IgG index	0.68 (H)			0.28–0.66 ratio
Oligoclonal bands	Negative			Negative
Myelin basic protein	15.3 ng/mL (H)			0.00–5.50 ng/mL
Meningitis/encephalitis nucleic acid detection test	Not detected			Not detected
JC virus DNA, ultrasensitive, quantitative, real-time PCR	Negative			Negative
Borrelia species by PCR	Not detected			Not detected
Epstein-Barr virus PCR	Not detected			Not detected
HIV-1 viral DNA	Not detected			Not detected
HIV-2 viral DNA	Not detected			Not detected
RPR quantitative	Nonreactive			Nonreactive
Cryptococcal antigen screen	Negative			Negative
Coccidioides antibody by complement fixation	<1:2			<1:2
VDRL	Nonreactive			Nonreactive
Mycobacterium tuberculosis PCR	Not detected			Not detected
Mayo Clinic Encephalitis Panel (ENC2)	Negative			Negative

Abbreviations: Ab = antibody; ANA = antinuclear antibody; BK = human polyomavirus 1; ENC2 = Encephalopathy, Autoimmune/Paraneoplastic Evaluation, Cerebrospinal Fluid; HD = hospital day; HTLV = human T-cell lymphotropic virus; Ig = immunoglobulin; IgG = Immunoglobulin G; IgM = Immunoglobulin M; JC virus = John Cunningham virus; MOG FACS = myelin oligodendrocyte glycoprotein fluorescence-activated cell sorting assay; MPO = myeloperoxidase; NGS = next-generation sequencing; PCR = polymerase chain reaction; PR3 = proteinase 3; RBC = red blood cell; RPR = rapid plasma reagin; SSA = Sjögren syndrome antigen A (anti-Ro); SSB = Sjögren syndrome antigen B (anti-La); VDRL = Venereal Disease Research Laboratory.
Unless otherwise specified, the results listed were sent during the admission to our hospital.

Section 4

PML was unlikely given the negative JC virus test and because neuroimaging was atypical for PML; the dentate nuclei are usually spared and the middle, rather than the superior, cerebellar peduncles are involved.[5] Unrevealing serum and CSF studies made infectious conditions less likely. Therefore, the differential shifted toward autoimmune/inflammatory etiologies, particularly IRIS or ADEM. IRIS was supported by ART reinitiation, high HIV viral load, and recent OI. Demyelinating disease was supported by elevated CSF MBP and MRI findings. Although brain biopsy was considered due to

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

diagnostic uncertainty, it was deferred given the lesion locations and clinical plateau. As neuroimaging findings, particularly T2-hyperintense brainstem and cerebellar lesions, favored an autoimmune/inflammatory etiology, serum antibody testing was repeated, including MOG and aquaporin-4. MOG antibody returned positive (1:100) 2 weeks after admission. Titers at this level have an estimated 82% positive predictive value for MOGAD.[6] Low-titer false positives (typically 1:20–1:40) have been reported with other inflammatory or demyelinating disorders.[7]

Given the negative infectious evaluation, he was empirically treated for inflammatory etiologies with a 5-day IV methylprednisolone course followed by oral prednisone taper. He demonstrated mild early clinical improvement, but without significant change on the Scale for Assessment and Rating of Ataxia.[8] Owing to limited response to corticosteroids, he received 5 sessions of plasma exchange followed by 5 days of IV immunoglobulin, with transition to monthly infusions. He remained hospitalized for 10 weeks to complete immunotherapy. One week after discharge, ophthalmologic evaluation revealed bilateral optic nerve atrophy and impaired visual acuity, consistent with sequelae of optic neuritis. In the following months, dysarthria and ataxia gradually improved but persisted, and vision remained poor. Repeat brain MRI 5 months later showed marked improvement in T2-hyperintense brainstem and cerebellar lesions (Figure, I-L).

Discussion

MOGAD is an autoimmune disorder causing inflammation and demyelination of the CNS.[9] Current diagnostic criteria include a demyelinating event (e.g., ADEM, optic neuritis, or transverse myelitis), positive MOG-IgG testing, supportive clinical or neuroimaging findings, and exclusion of alternative diagnoses.[4] MOGAD was deemed the most likely diagnosis, likely triggered by IRIS following ART reinitiation.

This case illustrates the importance of timing in MOG-IgG testing. Although data on initially false-negative results are limited, particularly in adults and individuals with HIV/AIDS, one study described a median interval of 38.5 days from symptom onset to seropositivity.[10] Therefore, when clinical suspicion remains high despite initial negative testing, repeat MOG-IgG testing should be considered, particularly near an active clinical event. MOG-IgG titers may decline over time, with one pediatric cohort reporting median seronegative conversion after approximately 1 year.[11]

Our patient's neuroimaging findings were typical for MOGAD, which frequently involves the brainstem and cerebellum.[12,13] However, demyelinating diseases may present differently in people with HIV due to immune dysregulation, particularly dysfunctional T-cell responses.[14] This may explain more aggressive phenotypes and poorer outcomes reported in HIV-associated MOGAD,[9] although scarce literature exists regarding IRIS/MOGAD overlap in HIV. CNS autoimmune manifestations of IRIS are uncommon, typically emerging within 3–6 months of ART initiation; they may present as focal or multifocal demyelination resembling multiple sclerosis or severe diffuse leukoencephalopathy, although the underlying pathophysiologic mechanisms remain unclear.[15]

Neuroimaging is important in the evaluation of rhombencephalitis but must be interpreted cautiously. Classic patterns can help distinguish infectious etiologies from inflammatory or demyelinating lesions, which more often appear as multifocal or poorly demarcated lesions.[16] In practice, however, imaging findings frequently overlap, reinforcing that diagnosis cannot rely on imaging alone. When imaging is indeterminate, serial imaging assessing the lesion evolution, diffusion restriction, and gadolinium enhancement pattern may narrow the differential.

Our case highlights the diagnostic challenges of evaluating rhombencephalitis in immunosuppressed individuals, where both OI and immune-mediated processes must be considered. CSF studies and neuroimaging were critical in narrowing the differential. Overall, this case was consistent with MOGAD occurring in the setting of IRIS. Clinicians should consider MOG-IgG testing when demyelinating features are present, and standard infectious and autoimmune evaluations are unrevealing. Clinicians should remain vigilant for IRIS following ART initiation and consider overlapping pathologies in immunocompromised patients. When clinical suspicion remains high, repeating targeted studies may be necessary, as early results may be falsely negative. Ultimately, the diagnosis is clinical, shaped by integrating the entire presentation, laboratory, and neuroimaging results rather than reliance on any individual diagnostic test.

Acknowledgment

The authors thank Dr. Sophia Raefsky and Dr. Angelica Means for their contribution to the patient's care and to writing the initial draft.

Author Contributions

P. Chandrasekhar: drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data. V.X. Pham: drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data. L. Timple: drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data. R. Deiss: drafting/revision of the manuscript for content, including medical writing for content. R.J. Ellis: drafting/revision of the manuscript for content, including medical writing for content. A. Dunn-Pirio: drafting/revision of the manuscript for content, including medical writing for content. K. Longardner: drafting/revision of the manuscript for content, including medical writing for content; analysis or interpretation of data.

Study Funding

The authors report no targeted funding.

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.

Disclosure

P. Chandrasekhar, V.X. Pham, L. Timple, R. Deiss, R.J. Ellis, and A. Dunn-Pirio report no disclosures relevant to the manuscript; K. Longardner served as a journal editor for *Frontiers in Neurology*, served as a consultant for Lundbeck and Prime Market Access, and received research funding from the NIH, Mission MSA, and Acadia Pharmaceuticals. Go to Neurology.org/N for full disclosures.

Publication History

Received by *Neurology*® March 20, 2026. Accepted in final form August 21, 2026. Submitted and externally peer reviewed. The handling editor was Resident & Fellow Section Deputy Editor Ariel Lyons-Warren, MD, PhD.

References

1. Kannaditharayil D. Pml IRIS: a difficult diagnosis (P02.271). *Neurology.* 2012;78(-Meeting Abstracts 1):P02.271. doi:10.1212/wnl.78.1_meetingabstracts.p02.271
2. Anilkumar AC, Foris LA, Tadi P. *Acute Disseminated Encephalomyelitis.* StatPearls. StatPearls Publishing; 2026. Accessed April 17, 2026. ncbi.nlm.nih.gov/books/NBK430934/
3. Shumway CL, Patel BC, Tripathy K, De Jesus O. *Neuromyelitis Optica Spectrum Disorder (NMOSD).* StatPearls. StatPearls Publishing; 2026. Accessed April 17, 2026. ncbi.nlm.nih.gov/books/NBK572108/
4. Banwell B, Bennett JL, Marignier R, et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD panel proposed criteria. *Lancet Neurol.* 2023;22(3):268-282. doi:10.1016/S1474-4422(22)00431-8
5. Adra N, Goodheart AE, Rapalino O, et al. MRI shrimp sign in cerebellar progressive multifocal leukoencephalopathy: description and validation of a novel observation. *AJNR Am J Neuroradiol.* 2021;42(6):1073-1079. doi:10.3174/ajnr.A7145
6. Sechi E, Buciuc M, Pittock SJ, et al. Positive predictive value of myelin oligodendrocyte glycoprotein autoantibody testing. *JAMA Neurol.* 2021;78(6):741-746. doi:10.1001/jamaneurol.2021.0912
7. Manzano GS, Salky R, Mateen FJ, et al. Positive predictive value of MOG-IgG for clinically defined MOG-AD within a real-world cohort. *Front Neurol.* 2022;13:947630. doi:10.3389/fneur.2022.947630
8. Schmitz-Hübsch T, du Montcel ST, Baliko L, et al. Scale for the assessment and rating of ataxia: development of a new clinical scale. *Neurology.* 2006;66(11):1717-1720. doi:10.1212/01.wnl.0000219042.60538.92
9. Gadama Y, Du Preez M, Carr J, et al. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) and human immunodeficiency virus infection: dilemmas in diagnosis and management: a case series. *J Med Case Rep.* 2023;17(1):457. doi:10.1186/s13256-023-04191-7
10. Forcadela M, Rocchi C, San Martin D, et al. Timing of MOG-IgG testing is key to 2023 MOGAD diagnostic criteria. *Neurol Neuroimmunol Neuroinflamm.* 2024;11(1):e200183. doi:10.1212/NXI.0000000000200183
11. Fadda G, Armangue T, Hacohen Y, Chitnis T, Banwell B. Paediatric multiple sclerosis and antibody-associated demyelination: clinical, imaging, and biological considerations for diagnosis and care. *Lancet Neurol.* 2021;20(2):136-149. doi:10.1016/S1474-4422(20)30432-4
12. Uzawa A, Oertel FC, Mori M, Paul F, Kuwabara S. NMOSD and MOGAD: an evolving disease spectrum. *Nat Rev Neurol.* 2024;20(10):602-619. doi:10.1038/s41582-024-01014-1
13. Banks SA, Morris PP, Chen JJ, et al. Brainstem and cerebellar involvement in MOG-IgG-associated disorder versus aquaporin-4-IgG and MS. *J Neurol Neurosurg Psychiatry.* 2020;28:jnnp-2020-325121. doi:10.1136/jnnp-2020-325121
14. Naidoo A, Paruk H, Bhagwan B, Moodley A. Atypical presentations of acute disseminated encephalomyelitis (ADEM) in HIV infection. *J Neurovirol.* 2017;23(1):160-170. doi:10.1007/s13365-016-0481-0
15. Johnson T, Nath A. Neurological complications of immune reconstitution in HIV-infected populations. *Ann N Y Acad Sci.* 2010;1184:106-120. doi:10.1111/j.1749-6632.2009.05111.x
16. Tillema JM, Pirko I. Neuroradiological evaluation of demyelinating disease. *Ther Adv Neurol Disord.* 2013;6(4):249-268. doi:10.1177/1756285613478870

Downloaded from https://www.neurology.org by 2603:8001:5800:3c97:94d6:16ac:13db:eef0 on 15 November 2026

Copyright © 2026 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.