



22 CME-CPD credits will be granted by EACCME

7th PANHELLENIC INTERDISCIPLINARY CONGRESS

on Childhood, Adolescent, Adult and Aged-adult Multiple Sclerosis



19-21 NOVEMBER 2026

HOTEL **Royal Olympic** ATHENS, GREECE

DIALOGUES OF THE GREEK NEUROSCIENTISTS

Advanced immunotherapeutic approaches in neuroautoimmunity

ORGANIZED BY

Ελληνικό Ινστιτούτο Παιδικής, Εφηβικής και Ενηλίκων Σκλήρυνσης κατά Πλάκας (Ε.Ι.Π.Ε.Ε.Σ.)

UNDER THE AUSPICES OF

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
1837–2027 190 ΧΡΟΝΙΑ

IN COLLABORATION WITH

Ινστιτούτο Βιολογίας και Ιατρικής του Στρες (Ι.Β.Ι.Σ.)

ΕΡΕΥΝΗΤΙΚΟ ΠΑΝΕΠΙΣΤΗΜΙΑΚΟ ΙΝΣΤΙΤΟΥΤΟ ΥΓΕΙΑΣ ΜΗΤΕΡΑΣ ΠΑΙΔΙΟΥ & ΙΑΤΡΙΚΗΣ ΑΚΡΙΒΕΙΑΣ

Ινστιτούτο Αυτοάνοσων Συστηματικών και Νευρολογικών Νοσημάτων (Ι.Α.ΣΥ.Ν.Ν.)

Scientific Program



Welcome Address

Dear colleagues,
Dear students,

It is with great pleasure that we welcome you to the ***7th Panhellenic Interdisciplinary Congress on Pediatric, Adolescent, Adult, and Older Adult Multiple Sclerosis,*** organized by the ***Hellenic Institute of Pediatric, Adolescent and Adult Multiple Sclerosis (EIPEES).***

This year's congress marks two important milestones: 11 years since our first Panhellenic Congress in 2015 and 15 years since our interdisciplinary team organized its first congress devoted exclusively to multiple sclerosis (MS) in 2011. The congress is held under the auspices of the National and Kapodistrian University of Athens Medical School, with the support of scientific organizations and sponsors.

Recent advances in Neuroimmunology and Neuroimaging have profoundly transformed our understanding of MS, the prototypical demyelinating disease of the central nervous system (CNS), as well as its mimics and other autoimmune and demyelinating disorders.

These developments are reflected in updated **diagnostic criteria for MS** and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), together with the emergence of innovative immunotherapeutic strategies. These include chimeric antigen receptor T-cell (CAR-T) therapies, complement C5 inhibitors, and neonatal Fc receptor (FcRn) inhibitors. At different stages of clinical development and implementation, these approaches offer promising opportunities for selected autoimmune disorders affecting the central and peripheral nervous systems.

This year's congress will explore these advanced therapeutic strategies, distinguishing treatments already established for specific clinical indications from those undergoing investigation. Particular emphasis will be placed on **CAR-T cell therapy**, continuing and expanding the scientific dialogue initiated two years ago. Distinguished immunologists and other specialists from Greece and abroad will critically examine the opportunities, limitations, and future applications of these therapies.

Remyelination remains another fundamental objective in MS research. Students, early-career researchers, and experienced scientists will discuss the cellular and molecular mechanisms of myelin repair, alongside emerging technologies such as optogenetics, which offer new opportunities to investigate and understand nervous system function.

Additional highlights include pregnancy in women with MS, the relationship between pregnancy, obesity, and the use of **glucagon-like peptide-1 (GLP-1) receptor agonists**, and a dedicated session on immunopharmacology, a rapidly evolving field with important implications for personalized treatment.

A defining feature of our congress has always been its commitment to the **next generation of scientists**. This year, we will again provide a platform for students from the Greek-language and English-language medical degree programs of the Athens Medical School, students from other universities, doctoral candidates, postdoctoral researchers, and young immunologists. We firmly



WELCOME ADDRESS

believe that scientific progress depends on the creative interaction between experience, intellectual curiosity, innovation, and critical thinking.

Distinguished neurologists, neuroimaging specialists, rheumatologists, and pediatric neurologists will also address essential aspects of everyday clinical practice, differential diagnosis, and contemporary therapeutic management.

Continuing another valued tradition, the congress will feature a special session exploring the **intersection of neuroscience and the arts, focusing this year on *neuroschediastics*.**

The congress brings together physicians, researchers, and healthcare professionals from a broad range of disciplines. Its scientific scope extends from fundamental and translational research to highly specialized immunotherapeutic approaches, emphasizing the essential role of interdisciplinary collaboration.

Beyond presenting new scientific findings, we hope this congress will provide a stimulating forum for exchanging knowledge, sharing experiences, encouraging critical discussion, and fostering productive scientific debate.

We look forward to welcoming you to the ***Royal Olympic Hotel in Athens, November 19–21, 2026***.

With warm collegial regards.

Marinos Dalakas **Maria Anagnostouli** **George Chrousos**





Committees

Organizing Committee

President:
Anagnostouli Maria

Vice Presidents and Honorable Members of the Scientific Committee:
Dalakas Marinos
Chrousos George

Members
Goules Andreas
Vartzelis Georgios

Scientific Committee

Chitnis Tanuja
Gastaldi Matteo
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Anagnostouli Maria
Angeli Maria
Arkoudis Nikolaos-Achilleas
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Bogdanos Dimitrios
Boziki Marina-Kleopatra
Chatzipanagiotou Stylianos
Chrousos George
Dalakas Marinos
Daponte Ariadni
Derdas Stavros
Demetriou Maria
Dinopoulos Argyrios
Efthymiou Evgenia
Evangelopoulou Maria-Eleftheria
Giorgi Melpomeni
Gkougka Dionysia
Gontika Maria
Goules Andreas
Gravanis Achilleas
Ioannidis Anastasios
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Lantzi Myrto
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Markoglou Nikolaos
Mastorodemos Vasileios
Mouskou Stella
Nikolaidou Maria
Nikolaou Chrysoula
Ntoskas Triantafyllos
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Papageorgiou Sokratis
Patas Konstantinos
Patrinos Georgios
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Skarlis Charalambos
Spyridonidis Alexandros
Stathopoulos Panos
Toulas Panagiotis
Tsagkaropoulos Antonios
Tsiamaki Eirini
Tsimourtou Vaia
Tzavellas Elias
Vartzelis Georgios
Velonakis Georgios
Vlachakis Dimitrios
Voudris Konstantinos



Scientific Program

THURSDAY 19 NOVEMBER 2026

10:00 - 10:15 **Objectives of the 7th Panhellenic Interdisciplinary Conference on Pediatric, Adolescent, Adult, Elderly, MS**

Maria Anagnostouli, Marinos Dalakas, George Chrousos, Andreas Goules, George Vartzelis

10:15 - 10:30 **Introduction – Welcome to the Congress**

Greetings from the International Pediatric MS Study Group, IPMSSG

Speaker: **Tanuja Chitnis**

10:30 - 12:30 **Young Scientists – Doctoral Candidates Forum**

Chair: **Maria Anagnostouli, George Vartzelis**

Exosomes in Multiple Sclerosis, as Biomarkers and Therapeutics. A New Era in Precision Medicine — **Myrto Lantzi**

The role of HLA alleles in childhood and adolescent multiple sclerosis. The twenty-five years Hellenic experience — **Evangelia - Iliana Papadaki**

Molecular mimicry in Stiff Person Syndrome — **Konstantinos Barsakis**

Immune Dysregulation in Multiple Sclerosis: From Autoimmune Mechanisms to Neuroinflammation — **Maria Demetriou**

The role of the HLA system in the development of molecular therapies from umbilical cord blood — **Panagiotis Mallis**

Multicenter registry of pediatric-onset multiple sclerosis in greece and longitudinal Study of biomarkers as indicators of disease activity and treatment response — **Maria Angeli**

12:30 - 13:00 **Coffee Break**

13:00 - 15:00 **Session: Rare but highly educational – Presentation of rare cases of demyelinating diseases of the CNS, across the entire age spectrum**

Chair: **Charis Kotsalis, Dionysia Gkouga, Konstantinos Voudris**

Speakers: **Sofia Taximi, Maria Nikolaidou, Aikaterini Adamaki, Danai Spiliopoulou, Anastasia Exadaktilou, Nikoleta-Aikaterini Xixi**



S C I E N T I F I C P R O G R A M

THURSDAY 19 NOVEMBER 2026

15:00 - 16:30 **Current Diagnostic Criteria for Multiple Sclerosis and other demyelinating diseases of the CNS**

Chair: **Anastasios Orologas, Maria - Eleftheria Evangelopoulou**

The laboratory approach	**Konstantinos Patas**
The neurological clinical approach	**Dimitrios Karathanasis**
The neuroimaging approach	**Panagiotis Toulas**
Discussion – Clinical Cases	

16:30 - 17:00 **Coffee Break**

17:00 - 19:00 **Students of Medical Degree English Program and Students of Athens Medical School and Medical School of Nicosia University, Athens: on autoimmunity and new insights on immunopathophysiology of neuroautoimmune diseases**

Chair: **Maria Anagnostouli, George Vartzelis**

Optogenetics in MS therapeutics. Remyelination and more	**Demetrios Fotopoulos**
Autoimmune Encephalitides in children and adults	**Evgenia Evgeniou**
HLA alleles in neuroautoimmune CNS and PNS diseases	**Chrysovalandia Menicou**
Current and future Immunopharmacology	**Anastasia Karapanagou**
Remyelination in MS: an unmet need with increased future therapeutic potential	**Fotini Tourni**

Co - Moderators: **Charalampos Skarlis, Andreas Palantzas**

19:00 - 20:30 **Opening Ceremony**

Official Guest Speakers – Opening Speeches

Chair**: Marinos Dalakas, George Chrousos**

Autoimmunity and Multiple Sclerosis	**Dimitrios Bogdanos**
Neuroschediastics as an innovative field of encounter and union of art and science	**Spyros Poulimenos**

20:30 - 21:00 **Official Greetings - Honorary Awards**



S C I E N T I F I C P R O G R A M

FRIDAY 20 NOVEMBER 2026

09:00 - 11:00 **Gut - brain axis and neuropsychiatric diseases**

Chair: **Chrysoula Nikolaou, Stylianos Chatzipanagiotou**

The microbiological approach — **Anastasios Ioannidis**

The genetic approach — **Stavros Derdas**

The neuropsychiatric approach — **Elias Tzavellas**

11:00 - 11:30 **Coffee Break**

11:30 - 13:30 **Neuroimaging in demyelinating diseases of the CNS across the entire age spectrum**

Chair: **Georgios Velonakis, Panagiotis Toulas**

Neuroimaging in immune - mediated optic neuritis — **Nikolaos-Achilleas Arkoudis**

Neuroimaging in autoimmune encephalitides — **Evgenia Efthymiou**

MOGAD and neuroimaging across the age spectrum. New data — **Vasiliki Barmparousi**

13:30 - 14:00 **Coffee Break**

14:00 - 15:00 **Satellite Symposium** *Sponsored by* SIEMENS Healthineers

Chair: **Stylianos Chatzipanagiotou, Chrysoula Nikolaou**

Neurofilament Light Chain (NfL): A Biomarker Game - Changer in Neuroautoimmune and Neurodegenerative Diseases — **Maria Anagnostouli**

15:00 - 16:00 **Lunch Break**

16:00 - 17:00 **Distinguished Lecture**

Chair: **George Patrinos, Argyrios Dinopoulos, Maria Anagnostouli**

Applications of Artificial Intelligence in Cancer Treatment — **Amanda Psyrri**



S C I E N T I F I C P R O G R A M

FRIDAY 20 NOVEMBER 2026

17:00 - 19:00 **Therapeutic advancements in neuroautoimmune diseases**

Chair: **Marinos Dalakas, Maria Anagnostouli**

MOGAD therapeutics — **Matteo Gastaldi**

Genetically characterized precision medicine in neuropsychiatric diseases — **George Patrinos**

EBV reactivation primes the peripheral immune system in multiple sclerosis relapse — **Tanuja Chitnis**

Anti - Gad neuroautoimmune disorders — **Marinos Dalakas**

19:00 - 19:30 **Coffee Break**

19:00 - 20:30 **Optic neuritis and myelitis (MS, NMOSD, MOGAD, Autoimmune Encephalitides, Systemic Autoimmunity)**

Presentations, Case reports and MEET THE EXPERT SESSION

Chair: **Marinos Dalakas, Maria Anagnostouli, Andreas Goules**

Case one: **Antonios Apostolakis**

Case two: **Melpomeni Giorgi**

Case three: **Stella Mouskou**

Commentators: **Tanuja Chitnis, Matteo Gastaldi**



S C I E N T I F I C P R O G R A M

SATURDAY 21 NOVEMBER 2026

09:00 - 11:00 **New immunotherapies for immune - mediated and neurodegenerative diseases of the CNS and PNS**

Chair: **Anastasios Orologas, Marina-Kleopatra Boziki**

Myasthenia Gravis, in Adults and Children — **Ariadni Daponte**

Neuromyelitis Optica Spectrum Disorders in Adults and Children — **Maria Gontika**

Multiple Sclerosis in Adults and Children — **Nikolaos Markoglou**

Alzheimer's Disease — **Marina-Kleopatra Boziki**

Commentators: **Sokratis Papageorgiou, George Vartzelis, Panos Stathopoulos**

11:00 - 11:30 **Coffee Break**

11:30 - 12:30 **Distinguished Lecture**

Chair: **George Chrousos, Andreas Goules**

Neurotrophins in the Regulation of Demyelination and Remyelination — **Achilleas Gravanis**

12:30 - 13:00 **Satellite Lecture** *Sponsored by* ER·KIM

Chair: **Maria Anagnostouli**

S1P Receptor Modulation in Multiple Sclerosis: What is the Significance of Selectivity? — **Triantafyllos Ntoskas**

13:00 - 14:30 **CAR T cell therapy in neuroautoimmune diseases**

Chair: **Marinos Dalakas, Alexandros Spyridonidis**

Mechanisms of CAR T cell therapies and complications in autoimmune neurology — **Dimitrios Mougiakakos**

Efficacy and complications CAR T therapy in hematologic malignancies — **Alexandros Spyridonidis**

How CAR T therapies are changing the therapeutic algorithm in Autoimmune Neurology: experience from a large series of patients with Stiff Person and Myasthenia — **Marinos Dalakas**

14:30 - 15:30 **Lunch Break**



S C I E N T I F I C P R O G R A M

SATURDAY 21 NOVEMBER 2026

Sponsored by Roche

15:30 - 16:30 **Satellite Lecture**

Planning for the future: Innovation in the Treatment of Multiple Sclerosis

Chair: **Maria Anagnostouli**

Ocrelizumab and innovation in the treatment of Multiple Sclerosis — **Vasileios Mastorodemos**

The puzzle of family planning in MS — **Eirini Tsiamaki**

Discussion

16:30 - 18:00 **Immunopharmacology**

Chair: **Maria Anagnostouli, Charalampos Skarlis**

Introduction to Immunology — **Charis Alexopoulos**

Introduction to Neuroimmunology — **Panos Stathopoulos**

Immunopharmacology and Artificial Intelligence (AI) — **Dimitrios Vlachakis**

18:00 - 18:30 **Coffee Break**

18:30 - 20:00 **Pregnant woman, in the context of neuroautoimmune diseases**

Chair: **George Chrousos, Marinos Dalakas, Maria Anagnostouli**

Family Planning in the Context of Multiple Sclerosis. When and under what conditions — **Antonios Tsagkaropoulos**

Guidelines for immunotherapies for pregnancy, in patients with Multiple Sclerosis and other demyelinating diseases — **Vaia Tsimourtou**

Treatment with GLP - 1 receptor agonists in obese patients with Multiple Sclerosis, with or without pregnancy — **George Chrousos**

20:00 - 20:30 **Final Discussion - Conclusions - Closing of the Congress**

Faculty



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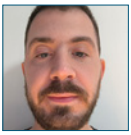
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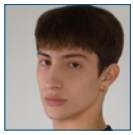


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General Information

Organized by:

Hellenic Institute of Childhood, Adolescent, Adult Multiple Sclerosis (EIPEES)

Under the Auspices:
School of Medicine, NKUA

Co-Organizers:
- Institute of Biology and Medicine of Stress (IBIS)
- Institute of Autoimmune Systemic and Neurological Diseases (IASYNN)
- Research University Institute of Mother Child & Precision Medicine
- UNESCO Chair of Adolescence of Health and Medicine, National and Kapodistrian University of Athens

Dates & Venue:
November 19-21, 2026
Royal Olympic Hotel - KALLIRHOE Hall
28–34 Athanasiou Diakou Street, Athens 117 43
Tel: +30 210 928 8400

Registrations:
Registration and attendance are allowed only to Health Care Professionals and undergraduate/graduate students. Registrations require presentation/proof of professional capacity or status:

Physicians	140€
New Physicians	70€
Residents	50€
Pharmacists	50€
Other healthcare professionals	50€
Nurses	Free
Biologists	Free
Medical Students	Free

The above rates are inclusive of 24% VAT

* **Early-career specialists:** Up to 5 years after obtaining their specialty certification.

** **Undergraduate students**, upon presentation of a valid student ID.



CME – CPD credits:
The event has been accredited with 22 CME – CPE credits, according to EACCME-UEMS criteria.

Certificate of Attendance:
The certificate of attendance will be sent at the end of the event to those delegates who have completed their registration. A necessary condition for receiving the attendance certificate is the completion of at least 60% of the total hours of the scientific program.

Cover artwork:
Spyros Poulimenos

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STUDENTS' ABSTRACTS

(In alphabetical order, due to first author's last name)

AUTOIMMUNE ENCEPHALITIS IN ADULTS AND CHILDREN

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Autoimmune encephalitis (AE) comprises a heterogeneous group of immune-mediated inflammatory disorders of the brain directed against neuronal or other central nervous system antigens. It may present with rapidly evolving cognitive or memory impairment, psychiatric or behavioural change, seizures, movement disorders, speech dysfunction, autonomic instability, sleep disturbance, and altered consciousness. Recognition is challenging because manifestations vary with age and antibody subtype and may resemble infectious, metabolic, seizure-related, psychiatric, neurodegenerative, or paraneoplastic disorders.

This presentation explores the recognition, pathophysiology, diagnosis, and management of AE across adult and paediatric populations. Particular emphasis is placed on the early exclusion of infectious encephalitis, especially herpes simplex virus, which may both mimic AE acutely and later trigger post-infectious autoimmune encephalitis. Pathophysiology is considered through the distinction between cell-surface or synaptic antibody-mediated disease, which often causes potentially reversible neuronal dysfunction, and classical intracellular-antigen syndromes, where cytotoxic T-cell-mediated injury is more prominent.

Antibody-associated phenotypes, tumour associations, and genetic susceptibility are reviewed, including HLA associations involving LGI1, CASPR2, and anti-NMDAR encephalitis. Anti-NMDAR encephalitis is used as a clinical model to illustrate characteristic neuropsychiatric progression, ovarian teratoma association, extreme delta brush on EEG, and the importance of cerebrospinal fluid antibody testing. Diagnostic evaluation integrates MRI, EEG, cerebrospinal fluid analysis, and paired serum–CSF antibody testing, together with the Graus adult and Cellucci paediatric diagnostic frameworks. Particular attention is given to antibody-negative disease, false-positive results, diagnostic mimics, and malignancy screening.

Management includes early first-line immunotherapy, escalation to second-line or rescue treatment when required, and prompt treatment of associated tumours. ICU complications, relapse, rehabilitation, and persistent cognitive and neuropsychiatric sequelae are also addressed, emphasizing that functional independence does not necessarily indicate complete neurological recovery.



OPTOGENETICS IN MS THERAPEUTICS. REMYELINATION AND MORE

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Modern optogenetics is a biological technique which combines genetic engineering and blue light stimulation to control cellular, particularly neuronal, activity through light-sensitive proteins called opsins. The use of optogenetics offers a promising experimental approach to investigating the mechanisms underlying multiple sclerosis (MS) and identifying potential therapeutic targets to produce pioneering health outcomes, especially as current MS therapies and treatment strategies to prevent neurodegeneration and promote functional recovery via remyelination remain limited.

This presentation explores the potential applications of optogenetics in experimental models of MS, with particular emphasis on the cuprizone model, experimental autoimmune encephalomyelitis (EAE), neuronal activity, neuronal stimulation, and myelin repair. The presentation will also question potential MS research applications for optogenetics. These applications may improve our understanding of neuronal signaling pathways, inflammatory processes, and oligodendrocyte differentiation in demyelinating disease.

Although optogenetics is not currently an established treatment for MS, its ability to precisely manipulate neural activity makes it a valuable tool for research and preclinical therapeutic exploration. The presentation will also address the methodological challenges associated with translating optogenetic findings into clinically relevant insights and give special attention to applications for pediatric-onset MS (POMS) patients.

Ultimately, the discoveries concerning light-gated ion channels and optogenetics—which were awarded with the 2026 Nobel Prize in Physiology or Medicine—may provide new insights into the pathophysiology of MS and support the development of strategies aimed at preserving neuronal function, promoting remyelination, and improving neurological outcomes for MS patients of diverse populations.



STUDENTS' ABSTRACTS

CURRENT AND FUTURE IMMUNOPHARMACOLOGY: FOCUS ON DEMYELINATING DISEASES

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Background: Multiple sclerosis (MS), aquaporin-4 antibody-positive neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) are immune-mediated demyelinating disorders of the central nervous system (CNS). Once considered variants of MS, NMOSD and MOGAD are now recognised as distinct entities with divergent immunopathogenesis: T- and B-cell-mediated inflammation in MS, complement-dependent astrocytopathy in NMOSD, and antibody-mediated demyelination in MOGAD. These differences require disease-specific, mechanism-targeted therapies, several of which are approved or in development.

Aim: To summarise the mechanisms of action and clinical effects of current and emerging immunotherapies in each disease.

Methods: PubMed was searched up to October 2026 for reviews, randomised trials and real-world studies of immunotherapies (monoclonal antibodies, Bruton's tyrosine kinase [BTK] inhibitors, chimeric antigen receptor [CAR] T cells) and remyelinating agents in MS, NMOSD and MOGAD, plus regulatory announcements.

Results: First-line immunomodulators and lymphocyte migration blockers reduce relapses in MS but are ineffective in NMOSD and MOGAD, and some may worsen NMOSD. B-cell depletion is effective in MS and NMOSD but less so in MOGAD. Complement C5 inhibitors, interleukin-6 (IL-6) receptor blockade, and anti-CD19 therapy are approved for NMOSD. In a real-world cohort (n=176), C5 inhibitors lowered relapse risk versus rituximab (hazard ratio 0.12). No therapy is yet approved for MOGAD; satralizumab (anti-IL-6 receptor) reduced relapse risk by 68% in a phase III trial and is under regulatory review. Among CNS-penetrant BTK inhibitors, tolebrutinib slowed disability progression in non-relapsing secondary progressive MS and gained EU approval in 2026; fenebrutinib was non-inferior to ocrelizumab in primary progressive MS. Emerging approaches include CAR T-cell therapy, antigen-specific immunotherapy to restore tolerance, and remyelinating agents, which have not yet shown clinical benefit.

Conclusion: Treatment response depends on the underlying disease mechanism, making accurate diagnosis essential. Current therapies control relapses well in MS and NMOSD, but progression and repair remain unmet needs.



STUDENTS' ABSTRACTS

HLA ALLELES IN NEUROAUTOIMMUNE CNS AND PNS DISEASES

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Autoimmune neurological disorders arise when misdirected immune responses breach specialized vascular barriers to target neural, glial, or synaptic antigens. Despite distinct underlying immunopathologies across the central and peripheral nervous systems, these conditions frequently manifest as overlapping clinical phenotypes that complicate diagnosis. Variations within the Human Leukocyte Antigen (HLA) region on chromosome 6p21.3 serve as the essential bridge, translating misleading clinical presentations into their precise underlying immunopathologies.

This bridge operates across three physically linked blocks: Class II acts as the susceptibility engine that presents to CD4+ T cells to license autoreactive B-cell responses; Class I directs tissue targeting and microenvironmental remodeling; and Class III executes physical tissue destruction via complement activation and Membrane Attack Complex assembly. Rather than acting as passive risk markers, HLA molecules function as the decisive "molecular switch": their stereochemical binding grooves provide the precise affinity required to present universal viral triggers like Epstein–Barr virus (EBNA1) in conformations that cross-react against neural self-antigens (GlialCAM).

Integrating landmark genome-wide association studies (IMSGC), functional mimicry benchmarks (Lanz et al.), subtype stratifications (Renton et al.), and Greek cohort analyses from Aeginition Hospital (Anagnostouli et al.), this immunogenetic framework resolves diagnostic boundaries across the central nervous system, the neuromuscular junction, and synaptic encephalitis. Because the nervous system possesses a limited vocabulary to express injury, clinical phenotypes frequently deceive at the bedside. Immunogenetic mapping provides the definitive missing link to resolve this clinical convergence.

By demonstrating that disease subtypes are genetically distinct entities, an individual's HLA architecture guides bedside therapeutic selection, averts catastrophic misdirection, such as treating NMOSD with standard MS disease-modifying therapies, and establishes the foundation for mechanism-targeted precision medicine.



STUDENTS' ABSTRACTS

REMYELINATION IN MULTIPLE SCLEROSIS, AN UNMET NEED WITH INCREASED FUTURE THERAPEUTIC POTENTIAL

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Multiple sclerosis (MS) is a chronic inflammatory disease of the central nervous system characterized by demyelination and axonal damage. Current therapies primarily manage symptoms and slow disease progression but they do not achieve remyelination.

Remyelination represents one of the most accessible strategies for achieving neuroprotection in MS and therefore complements the anti-inflammatory action of the currently used disease-modifying therapies (DMTs). By generating new myelin sheaths around axons, remyelination can restore nerve conduction properties and recover neurological function. While it occurs spontaneously in many lesions in MS , this process becomes increasingly inadequate due to the inflammatory microenvironment and eventually fails in most lesions and patients. There are two theories regarding the mechanism of remyelination. The leading theory supports that remyelination is the result of the recruitment of OPCs (Oligodendrocyte Precursor Cells) to the lesion, whereas the alternative theory emphasizes the re-enlistment of pre-existing mature oligodendrocytes.

Proof-of-concept studies, such as those evaluating clemastine fumarate, successfully demonstrated biological myelin repair and multiple other Phase II trials have offered promising new evidence. However, these positive initial trials are sometimes followed by studies with mixed or negative results. Frequently, trials fail to meet their primary clinical endpoints while still showing positive neuroprotective effects in secondary outcomes or long-term follow-up studies. A possible explanation is that capturing signs of true biological repair in remyelination trials might require highly sensitive outcome measures, longer trial duration and better screening criteria targeting patients with functionally intact axons.

The question remains; are these trials the result of true lack of drug efficacy or is active remyelination being obscured by suboptimal trial design?



RESEARCH PAPERS

MULTICENTER REGISTRY OF PEDIATRIC-ONSET MULTIPLE SCLEROSIS IN GREECE AND LONGITUDINAL STUDY OF BIOMARKERS AS INDICATORS OF DISEASE ACTIVITY AND TREATMENT RESPONSE

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Introduction: The treatment of pediatric-onset multiple sclerosis (POMS) has improved significantly with high-efficacy disease-modifying therapies, reducing relapse rates and MRI disease activity. However, clinical assessment and conventional MRI may not fully capture ongoing subclinical inflammatory activity and tissue injury. Investigating additional biomarkers and long-term disease trajectories requires systematic longitudinal data, particularly in Greece, where a nationwide POMS registry is lacking.

Aim: To establish a nationwide multicenter registry of children and adolescents with POMS followed at pediatric centers across Greece and to investigate imaging and serum biomarkers as practical tools for disease monitoring.

Methods: This multicenter retrospective and prospective study, coordinated by Attikon University Hospital, will include a 10-year retrospective review and 3-year prospective follow-up of previously diagnosed and newly identified patients with POMS meeting current diagnostic criteria. Demographic, clinical, and imaging data will be collected. Serum NfL, GFAP, and CHI3L1 will be measured at baseline, six months after treatment initiation or switching, during relapses, and annually during clinical stability. Clinical assessments, EDSS, and SDMT will be performed every six months, while brain MRI and volumetric analysis will be conducted at baseline and 12, 24, and 36 months.



Results (Expected): The study is expected to characterize the clinical, therapeutic, and imaging features of POMS in Greece. Associations between serum biomarkers and relapses, MRI activity, and subclinical disease progression, assessed through brain volumetry and SDMT, will also be investigated.

Conclusions: Establishing a nationwide registry will improve the systematic documentation and understanding of POMS in Greece. Longitudinal biomarker assessment may facilitate the identification of patients at increased risk of future disease activity, subsequent disability, or subclinical disease progression. These findings may complement clinical and MRI monitoring, supporting more individualized treatment decisions.



R E S E A R C H P A P E R S

SARS-COV-2 INFECTION IS ASSOCIATED WITH ATYPICAL EPSTEIN-BARR VIRUS SEROLOGY AT THE FIRST CLINICAL EPISODE OF MULTIPLE SCLEROSIS

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Introduction: Epstein-Barr virus (EBV) is implicated in multiple sclerosis (MS), but its relationship with other viral infections at clinical onset remains unclear.

Methods: This cross-sectional study included 68 treatment-naive patients at their first MS episode (2021-2024), 69 SARS-CoV-2-infected individuals without MS, and 21 controls. Serology and plasma double-stranded RNA (dsRNA) were measured by ELISA; antiviral cytokines, neutrophil subsets, and neutrophil extracellular traps (NETs) were assessed. Molecular profiling comprised paired total RNA sequencing and J2-based dsRNA immunoprecipitation sequencing (dsRIP-seq; n=10), alongside plasma small extracellular vesicle proteomics (n=19).

Aim: To investigate associations between SARS-CoV-2 serology, atypical EBV serology, and dsRNA-related immune activation at MS onset.

Results: Anti-nucleocapsid (N) IgM was detected in 13/68 patients with MS. Atypical EBV serology, defined by concurrent anti-EBNA1 IgG/IgM positivity, occurred in 11/68. Anti-N IgM positivity was more frequent in this subgroup (7/11 vs. 6/57; p=0.0004). Anti-N and anti-EBNA1 IgM remained correlated after adjustment for age and anti-trinitrophenyl IgM (p=0.012). Double-positive patients had higher dsRNA and anti-dsRNA IgM than double-negative patients (p=0.0055 and p=0.0006). Plasma dsRNA correlated with immature neutrophil proportions (r=0.621). NET release was higher in SARS-CoV-2-seropositive patients with MS than in healthy controls. Interferon-stimulated transcripts inversely correlated with anti-EBNA1 IgM in total RNA-seq; IRF7 showed the strongest inverse correlation within this panel in dsRIP-seq. dsRIP-seq identified 2,088 enriched genes overrepresented in antiviral pathways. Vesicular proteomes differed by SARS-CoV-2 serostatus. In SARS-CoV-2-infected individuals without MS, anti-EBNA1 IgM correlated with dsRNA and anti-dsRNA IgM. Atypical EBV serology was more frequent in SARS-CoV-2-infected patients with MS than in those without MS (7/13 vs. 7/69).

Conclusions: SARS-CoV-2 serological markers were associated with atypical EBV serology and dsRNA-related immune activation at MS onset.



R E S E A R C H P A P E R S

STUDY OF SERUM NEUROFILAMENT LIGHT CHAINS (sNFL) IN MULTIPLE SCLEROSIS, CIS, AND RIS, IN PATIENTS FROM A SINGLE MULTIPLE SCLEROSIS CENTER

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Background: Serum neurofilament light chain (sNfL) is a biomarker of neuroaxonal injury and disease activity in multiple sclerosis (MS). We examined sNfL across diagnostic groups and its associations with MRI, brain atrophy, oligoclonal bands (OCB), sex and age in MS, clinically isolated syndrome (CIS) and radiologically isolated syndrome (RIS).

Methods: sNfL was measured with the Atellica IM NfL assay (Siemens Healthineers) in **215 patients** (MS 102, CIS/RIS 52, other neurological diseases [OND] 61). Groups were compared by Kruskal–Wallis and Dunn's test (Holm). In the 154 patients with MS or CIS/RIS, sNfL (median [Q1–Q3], pg/mL) was compared by brain, cervical and thoracic MRI findings and activity, brain atrophy, OCB type and sex (Mann–Whitney U); the lowest (n = 17) and highest (n = 16) deciles were compared; and ln(sNfL) was regressed on diagnosis, age and sex (robust SE).

Results: sNfL differed across groups (MS 14.95, CIS/RIS 11.40, OND 16.30 pg/mL; p = 0.014) and was lower in CIS/RIS than in MS (p = 0.008) and OND (p = 0.019). In MS and CIS/RIS, sNfL was higher with positive than negative brain MRI (13.9 vs 8.1 pg/mL; p = 0.004), active than inactive brain MRI (17.6 vs 12.3 pg/mL; p = 0.003) and with than without brain atrophy (25.2 vs 12.7 pg/mL; p = 0.005), but did not differ by spinal cord MRI, OCB type or sex (all p ≥ 0.094). Adjusted for age and sex, sNfL was 27.7% higher in MS than CIS/RIS (95% CI 1.2–61.2%; p = 0.040) and rose 1.7% per year of age (p < 0.001). Highest-decile patients were older (44.5 vs 31 years; p = 0.011), older at onset (43.5 vs 31; p = 0.009) and more often had active brain MRI (50.0% vs 0%; p = 0.001).

Conclusions: sNfL is lower in CIS/RIS than in MS and associated with brain MRI lesions, activity and atrophy, but not with spinal cord MRI. As it rises with age independently of diagnosis and sex, sNfL should be interpreted against age.



R E S E A R C H P A P E R S

THE ROLE OF HLA ALLELES IN CHILDHOOD AND ADOLESCENT MULTIPLE SCLEROSIS. THE TWENTY-FIVE YEARS GREEK EXPERIENCE

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Background: *Pediatric-onset multiple sclerosis (POMS)* accounts for approximately 3–5% of all multiple sclerosis cases and is characterized by increased inflammatory activity and a higher annualized relapse rate compared with adult-onset disease. This presentation aims to provide a concise overview of the published Greek research and clinical experience in POMS from our Center, focusing on immunogenetic susceptibility, clinical and neuroimaging characteristics, and therapeutic management.

Methods: A review was conducted of publications by the Greek research group at our Center concerning patients with pediatric-onset multiple sclerosis. Studies investigating HLA-DRB1 and HLA-DPB1 alleles, APOE genotypes and alleles, and the use of disease-modifying therapies were included. The main study characteristics, principal findings, and methodological limitations were also recorded.

Results: Immunogenetic studies consistently demonstrated an association between HLA-DRB1*03 and increased susceptibility to POMS in the Greek population, while additional potentially predisposing or protective associations involving other HLA alleles were explored. The increased frequency of the APOE2 allele among patients with POMS suggests a potentially novel genetic susceptibility factor outside the HLA system. Regarding treatment, first-line therapies were associated with a reduction in relapses, although they did not provide adequate disease control in all patients. Studies of fingolimod and natalizumab provided favourable real-world evidence regarding their effectiveness and safety, particularly among patients with highly active disease. First-line therapies appeared to be more effective in children who initiated treatment earlier.

Discussion: The Greek experience highlights the research value of an immunogenetic approach to POMS and the importance of the early development of an individualized treatment plan. Nevertheless, the findings should be interpreted with caution, as the available evidence is derived predominantly from retrospective, single-center studies without comparison groups.

Conclusion: The Center's longstanding and systematic clinical and research activity has established an important body of knowledge on POMS in Greece, contributing substantially to the understanding of genetic susceptibility and the evidence base supporting therapeutic decision-making. This experience provides a strong foundation for future research and multicenter collaborations aimed at improving prognostic assessment and further individualizing treatment.



R E S E A R C H P A P E R S

REAL-OCRE-ATH: CLINICAL, NEUROIMAGING, AND IMMUNOPHENOTYPIC OUTCOMES OF OCRELIZUMAB TREATMENT IN MULTIPLE SCLEROSIS IN A REAL-WORLD CLINICAL SETTING

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Background: Ocrelizumab, an anti-CD20 monoclonal antibody, depletes B cells and is approved for relapsing and primary progressive multiple sclerosis (MS). We describe radiological, immunophenotypic and clinical outcomes in a real-world cohort treated with ocrelizumab.

Methods: From a total of **660 patients** undergoing treatment with ocrelizumab, a subgroup of **106 patients**—for whom complete clinical and laboratory follow-up data were available—was analyzed. Brain, cervical and thoracic spine MRI findings were classified as unchanged, new lesions/enhancement, or regression; gadolinium (Gd) enhancement was assessed separately. Absolute counts (cells/μL) and proportions of CD3+, CD4+, CD8+ and CD19+ lymphocytes and natural killer (NK) subpopulations, including CD56bright and CD56dim cells, and the Expanded Disability Status Scale (EDSS) were recorded at baseline and at 6, 12 and 36 months. Changes over time were tested with the Friedman test.

Results: Brain MRI remained unchanged in 94/106 patients (88.7%), cervical spine MRI in 96 (90.6%), and thoracic spine MRI in 100 (94.3%); new lesions or enhancement were seen in 10 (9.4%), 9 (8.5%), and 5 (4.7%), respectively. No new Gd-enhancing lesions were recorded in any region. Median CD19+ B-cell counts fell from 179 cells/μL (IQR 126–311) at baseline to 8 (2–38) at 6 months and 2 (0.65–9) at 12 months, and were 7 (0.63–78) at 36 months ($p = 0.010$); CD19+ proportions fell from 3.80% to 0.05% at 12 months ($p = 0.015$). T-cell counts changed little (e.g., CD3+ 1,338 vs 1,197 cells/μL at baseline and 36 months), although differences across time points were significant ($p < 0.001$). CD56bright NK cells decreased from 11 to 3 cells/μL at 36 months ($p = 0.048$), and the CD56bright/dim ratio decreased from 0.339 to 0.056 ($p = 0.064$). Median EDSS was 2.5 (1.5–4.5) at baseline, 2.0 (1.5–4.0) at 12 months, and 2.5 (1.5–3.5) at 36 months ($p < 0.001$).

Conclusions: In this real-world cohort, ocrelizumab was associated with radiological stability in most patients, no new Gd-enhancing lesions, marked B-cell depletion, largely preserved T-cell counts and no worsening of median disability over 36 months. The reduction in CD56bright NK cells warrants further study.



R E S E A R C H P A P E R S

BREAST MILK EXTRACELLULAR VESICLES IN LACTATING WOMEN WITH MULTIPLE SCLEROSIS: DESIGN OF THE MILKEVS-MS MULTIOMICS PROFILING STUDY

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Backround: Milk-derived extracellular vesicles (milkEVs) carry miRNAs, proteins, lipids and metabolites that are implicated in infant development and immune maturation. Interest in milkEVs has grown rapidly, highlighting their roles in health and disease and their potential as natural nanocarriers. However, to the best of our knowledge, the molecular profile of milkEVs from lactating women with Multiple Sclerosis (MS) has not been investigated. Research in this population has focused mainly on breastfeeding safety under disease-modifying therapies (DMTs), showing that several DMTs are compatible with breastfeeding without an increased risk of common adverse infant outcomes.

Milk samples will be collected from lactating women with MS. After defatting and casein removal, EVs will be isolated with an optimized protocol combining a commercial kit and size-exclusion chromatography (SEC). MilkEVs will be characterized by tunable resistive pulse sensing (TRPS; Exoid, Izon), transmission electron microscopy (TEM), and immunoblotting for positive (CD9, CD63, CD81, TSG101) and negative (calnexin) markers. EV cargo will be profiled by small RNA sequencing, LC-MS/MS proteomics and metabolomics, and analysed through an integrative bioinformatics pipeline (differential expression, pathway enrichment, multi-omics integration), stratified by disease activity, EDSS, DMT and postpartum stage.

Our aim is to comprehensively characterize the multi-omic profile of milkEVs in women with MS and to identify cargo molecules associated with immune regulation, inflammatory response and intercellular communication.

MilkEVs-MS will provide the first multi-omics map of breast milk EVs in MS, supporting evidence-based breastfeeding counselling and future personalized interventions for mother and infant.



R E S E A R C H P A P E R S

HLA OVERLAP IN AUTOIMMUNITY AND CANCER: ASSOCIATIONS IN MULTIPLE SCLEROSIS

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Introduction: Human leukocyte antigen (HLA) molecules play a central role in antigen presentation, immune regulation and self-tolerance, influencing susceptibility to autoimmune diseases and malignancies. In multiple sclerosis (MS), HLA class II polymorphisms represent the strongest genetic susceptibility factors, while Epstein–Barr virus (EBV) infection highlights the complex interactions between genetic and environmental factors. HLA molecules are essential to regulating tumor antigen recognition and immune surveillance, however, the extent of overlapping HLA susceptibility between autoimmunity and malignancy remains unclear.

Aim and Scope of the Review: This narrative review explores HLA associations across autoimmune diseases and malignancies, emphasizing MS. It highlights how HLA variations may promote autoimmunity, tumor surveillance or immune escape through altered antigen presentation, T-cell activation and natural killer (NK)-cell regulation. Particular attention is given to shared immunological mechanisms versus allele-specific susceptibility. In MS, emerging evidence suggests cancer-specific genetic overlap, particularly involving Hodgkin lymphoma and brain cancer, although direct evidence remains limited. The review also examines EBV-associated immune responses, malignancy risk and the relevance of HLA diversity to treatment response and immune-mediated adverse events, highlighting its potential in personalized medicine.

Conclusions and Future Perspectives: HLA polymorphisms contribute to a multifactorial susceptibility framework involving environmental, infectious, epigenetic and other genetic influences. Current evidence suggests that overlap between autoimmunity and malignancy is primarily mechanistic rather than allele-specific. Nevertheless, heterogeneous findings limit definitive conclusions regarding shared susceptibility. Future studies integrating high-resolution HLA genotyping, functional investigations and comprehensive characterization of patients with coexisting autoimmune diseases and malignancies are needed to clarify these associations. Improved understanding of HLA-mediated immune regulation may facilitate individualized risk assessment, prediction of therapeutic responses and adverse events, and personalized treatment strategies in MS.



R E S E A R C H P A P E R S

DEVELOPMENT OF A MACHINE LEARNING ALGORITHM FOR THE REFINEMENT OF HLA-TYPINGS, WITH APPLICATION TO POPULATION-BASED BIOMARKER STUDIES.

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Background/Objectives: Human Leukocyte Antigen (HLA) is a system involved in every aspect of physiological immune response. As such, its study has provided valuable insight in therapeutics and in many cases can be used as a biomarker for the diagnosis, prognosis and treatment of autoimmune and immune-related diseases or in pharmacogenomics. However, as HLA typing methods and standards have evolved over time, past patient data are often incomplete or consist of low resolution typings. In this study we apply current advances in bioinformatics and particularly in Machine Learning algorithms in order to impute missing data and/or upgrade low resolution results.

Methods: A locus-specific Extreme Learning Machine (ELM) framework, trained on the Greek national donor registry (n = 117,345), upgrades low-/intermediate-resolution genotypes and imputes untyped loci; output weights are solved analytically. It was validated on two independent Greek cohorts (n = 20,100; 4353) against GRIMM and two expectation maximization baselines, on the same donors.

Results: Accuracy depended on the high-resolution context available (94% for HLA-A with four anchor loci; 19–22% with none) and on the posterior probability threshold (78–91% ORAM, 70–84% GRPT), while call rate fell from 98.1% to ~41%; the two cannot be maximized together. GRIMM called fewer donors (1.8–47.9%) at comparable or higher accuracy. Allele-vocabulary abridgment excluded 14–47% of external-cohort allele calls; stratified by vocabulary membership, ORAM accuracy was 94.2% for in-vocabulary donors and 73.7% for the rest. The training procedure has no convergence guarantee.

Conclusions: ELM upgrading converts legacy HLA data into probabilistic high-resolution calls, with registry-scale training completing in minutes. These calls can increase the usability of older data in biomarkers and big data analysis studies.



CLINICAL PAPERS

2024 MCDONALD CRITERIA FOR MULTIPLE SCLEROSIS: CLINICAL, LABORATORY, AND NEUROIMAGING DATA THAT ARE TRANSFORMING THE DIAGNOSTIC AND THERAPEUTIC APPROACH TO MULTIPLE SCLEROSIS

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Introduction: Multiple Sclerosis (MS) is a multifactorial, neuroinflammatory, and neurodegenerative demyelinating disease of the Central Nervous System (CNS). New McDonald criteria were developed in 2024 and published in 2025.

Results: In summary, the key updates are as follows:

1. Harmonization of the diagnostic approach.

2. CVS (central vein sign), PRLs (paramagnetic rim lesions), and CSF-KFLC (cerebrospinal fluid kappa free light chains) can be used as supportive evidence for diagnosis.

3. In specific cases, Radiologically Isolated Syndrome (RIS) may meet the criteria for MS.

4. Provision of updated guidance for specific population groups—such as children and the elderly—as well as for patients with comorbidities.

Discussion: The revised McDonald criteria expand and redefine the diagnosis of Multiple Sclerosis, while maintaining diagnostic specificity.



C L I N I C A L P A P E R S

CLINICAL TRIALS IN PROGRESSIVE MULTIPLE SCLEROSIS: THERAPEUTIC ADVANCES, TRIAL OUTCOMES, AND UNMET NEEDS

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Introduction: Overlapping inflammatory and neurodegenerative mechanisms complicate treatment and outcome assessment in progressive multiple sclerosis (MS).

Aim: To evaluate therapeutic advances, methodological challenges, and unmet needs.

Methods: A targeted narrative review of five randomised trials in primary and secondary progressive MS assessed eligibility, endpoints, efficacy, and safety.

Results: Ocrelizumab (ORATORIO, primary progressive MS) and siponimod (EXPAND, secondary progressive MS) reduced confirmed disability progression (CDP), with hazard ratios (HRs) of 0.76 (12-week CDP) and 0.79 (3-month CDP), respectively. In HERCULES, tolebrutinib reduced 6-month CDP in nonrelapsing secondary progressive MS (HR 0.69; 95% confidence interval 0.55-0.88). It received EU authorisation in June 2026 for adults without relapses during the preceding two years, with liver monitoring required. Nonrelapsing disease does not necessarily imply absence of MRI activity. ORATORIO-HAND extended ocrelizumab evidence to older and more disabled primary progressive patients, using a composite endpoint of progression on the Expanded Disability Status Scale or Nine-Hole Peg Test, confirmed at 12 weeks (HR 0.70; 95% confidence interval 0.57-0.86); benefit was also demonstrated in the MRI-active subgroup. MS-STAT2 found no significant disability benefit from simvastatin despite earlier favourable brain-atrophy findings. Differences in eligibility, endpoints, and confidence intervals limit cross-trial comparisons.

Conclusions: Therapeutic options have expanded, but unmet needs persist, particularly without overt inflammatory disease activity. Future trials should combine meaningful disability outcomes with validated biomarkers and safety assessment.



C L I N I C A L P A P E R S

TUMEFACTIVE MULTIPLE SCLEROSIS: CLINICAL CHARACTERISTICS, DIAGNOSTIC APPROACH AND THERAPEUTIC MANAGEMENT IN A SERIES OF 11 PATIENTS, OF A SINGLE MULTIPLE SCLEROSIS CENTER

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Introduction: Tumefactive multiple sclerosis (TMS) is a rare manifestation of multiple sclerosis, characterized by considerable heterogeneity in its clinical presentation and imaging features, which may mimic intracranial neoplastic lesions. Differentiating TMS from other pathological conditions represents a diagnostic challenge and is crucial for selecting the appropriate treatment, at the short and long term.

Methods: We evaluated 11 patients with TMS sclerosis who were admitted in the Ward of CNS Demyelinating and Autoimmune Diseases, of the First Department of Neurology, between February 2025 and September 2025. The assessment was based on recorded clinical, laboratory and therapeutic data. Variables examined included age, sex, clinical presentation, imaging findings, cerebrospinal fluid (CSF) biomarkers, Expanded Disability Status Scale (EDSS) scores and therapeutic management.

Objective: To describe the clinical, neuroimaging and laboratory characteristics, diagnostic work-up and treatment of our patients, as well as the diagnostic and therapeutic approaches employed in everyday clinical practice and for the earlier and more comprehensive care of patients.

Results: A total of 11 patients were included, comprising 7 women and 4 men, with a mean age at diagnosis of 38 years (range: 26–57). Clinical manifestations were heterogeneous: 2 patients presented with cognitive impairment, 1 with epileptic seizures and 1 with headache. Among the remaining 7 patients, all presented with sensory symptoms, 5 of whom also had associated gait disturbances. The median EDSS score was 2.0 (range: 0–3.5, n=10). Brain MRI revealed lesions larger than 2 cm in 5 patients, with a maximum diameter of 6.5 cm. Follow-up brain MRI demonstrated radiological improvement in 2 patients, stable imaging findings in 5, the appearance of new lesions in 3, and worsening of the tumefactive lesion with gadolinium enhancement in 1 patient. Spinal cord MRI revealed lesions in 5 patients. Two patients underwent biopsy, with histopathological findings suggestive of demyelinating disease. CSF analysis was performed in 10 patients and revealed pleocytosis (>5 cells/μL) in 6, type 2 oligoclonal bands in 8, an



elevated IgG index in 6 and an elevated kappa index in 4 patients. Anti-MOG and anti-AQP4 antibodies were negative in all 9 patients with documented testing. Autoantibodies were detected in 4 patients (anti-SOX1, ANA/anti-SSA, Pm/Scl-100/AMA-M2 and ANA). During the acute phase, intravenous methylprednisolone was administered to 10 patients, while 2 patients underwent plasma exchange sessions. All patients are currently receiving anti-CD20 therapy (ocrelizumab: 8, ofatumumab: 2, rituximab: 1).

Conclusions: Tumefactive multiple sclerosis represents a diagnostic challenge due to the heterogeneity of its clinical manifestations and imaging characteristics. In our series of 11 patients, the combined assessment of clinical presentation, MRI findings and CSF biomarkers contributed to the diagnostic evaluation, while biopsy was considered necessary in selected cases. The variable radiological course highlights the importance of clinical and neuroimaging follow-up. The administration of anti-CD20 therapy to all patients reflects the therapeutic strategy adopted in this particular cohort. Furthermore, the inpatient administration of treatment offers the additional advantage of closer medical monitoring during drug administration. Despite the limitations associated with the small sample size and retrospective data collection, our findings highlight the importance of an individualized, multifactorial diagnostic approach aimed at the timely recognition of the disease.



C L I N I C A L P A P E R S

SUCCESSFUL OSSEOINTEGRATION OF DENTAL IMPLANTS IN A MOTHER AND DAUGHTER WITH MULTIPLE SCLEROSIS UNDER IMMUNOMODULATORY THERAPY: A REPORT OF TWO CASES

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Introduction: Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the CNS. Long-term immunomodulatory and immunosuppressive treatment, as well as corticosteroid use, raise concerns about wound healing and implant osseointegration. Evidence in this population is limited.

Case presentation: Patient A (49 years) has had relapsing-remitting MS since 2008 (EDSS 2.5) and underwent thyroidectomy for Hashimoto's thyroiditis. In 04/2021, while on natalizumab, she received two ultra-short implants (copaSKY, 5.2 mm) at sites 26 and 27. The implant at site 27 was placed immediately after extraction. The ultra-short implants avoided sinus floor elevation. She later received corticosteroids for approximately one year.

Patient B (73 years), her mother, has had secondary progressive MS since 1997 (EDSS 6.5) and has a history of sarcoidosis treated with corticosteroids. In 02/2022, while on cyclophosphamide, she received two implants (blueSKY) at sites 16 and 15. The implant at site 16 was placed immediately after extraction. In 10/2024, while on rituximab and ten months after her last infusion, she received two immediate post-extraction implants at sites 22 and 23 following periodontal breakdown. Patient B's implants were immediately loaded.

Final restorations were delivered at 3 months in all cases. Neither patient had a clinical relapse in the perioperative period.

Results: All six implants osseointegrated successfully. Over a follow-up of up to 5.5 years, clinical and radiographic findings remain stable, with no complications.

Conclusions: MS and long-term disease-modifying therapy, including potent immunosuppressive agents, do not necessarily contraindicate implant rehabilitation. Careful planning, less invasive options and collaboration with the treating neurologist contribute to predictable outcomes.



RARE CLINICAL CASES

EFGARTIGIMOD ALFA IN SERONEGATIVE JUVENILE GENERALIZED MYASTHENIA GRAVIS: A CASE REPORT

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Background: Juvenile seronegative generalized myasthenia gravis (sn-JMG) is a rare disorder that poses diagnostic and therapeutic challenges. Efgartigimod alfa, a neonatal Fc receptor antagonist, reduces circulating IgG levels. However, evidence regarding its use in pediatric patients remains limited.

Methods: We retrospectively reviewed the diagnostic evaluation, therapeutic management and clinical outcomes of an adolescent with triple-seronegative JMG, along with the relevant literature.

Objective: To describe what is, to our knowledge, the first reported pediatric case of triple-seronegative JMG treated with efgartigimod alfa.

Results: An adolescent girl presented in 2021 with diplopia and esotropia, initially managed as strabismus, with surgical correction in 2024. Recurrent diplopia prompted neurological reassessment, revealing fluctuating ptosis, generalized muscle weakness and fatigability, predominantly affecting the left side. Despite negative myasthenia-related antibodies and normal repetitive nerve stimulation, single-fiber electromyography demonstrated increased jitter, supporting the diagnosis. Intravenous immunoglobulin resulted in temporary improvement, while plasma exchange led to a more substantial response. Given disease severity and corticosteroid-related adverse effects, off-label efgartigimod alfa was initiated in 2025. Following an initially favorable response, mild deterioration, reflected by increasing QMG and MG-ADL scores, prompted a second cycle in 2026. One infusion was delayed due to respiratory infection. One month after cycle completion, the patient experienced severe infectious enteritis, which resolved with conservative management. Subsequent reduction in QMG and MG-ADL scores allowed gradual tapering of concomitant corticosteroid and pyridostigmine therapy. At a later follow-up, the patient reported her best clinical condition since disease onset, particularly regarding diplopia.

Conclusions: This case highlights the potential therapeutic benefit of efgartigimod alfa in sn-JMG. It may represent an additional therapeutic approach for selected pediatric patients, although further evidence is needed.



R A R E C L I N I C A L C A S E S

LONGITUDINALLY EXTENSIVE TRANSVERSE MYELITIS, AS THE INITIAL MANIFESTATION OF NEUROMYELITIS OPTICA SPECTRUM DISORDER (NMOSD) WITH POSITIVE ANTI-AQP4 ANTIBODIES: A CASE REPORT

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Objective: To present a case of longitudinally extensive transverse myelitis (LETM) as the initial manifestation of NMOSD, with strongly positive anti-AQP4 antibodies.

Materials/Methods: A 44-year-old woman with a history of bronchial asthma, arthralgias since adolescence and Raynaud's phenomenon presented to the Emergency Department with progressively worsening gait disturbance over 3 days. Four days earlier, she had experienced acute pain in the anterior chest wall and back and difficulty urinating. Her recent history included an upper respiratory tract infection 10 days earlier. Neurological examination revealed a Brown-Séquard syndrome, with weakness and hypopallesthesia of the right lower limb and hypoesthesia of the left hemitrunk and left lower limb (EDSS = 6). From the first day of hospitalization she deteriorated rapidly, developing paraplegia and becoming bedbound, with a T3 sensory level, severe pain and frequent paroxysmal tonic spasms of the lower limbs (EDSS = 8).

Results: MRI of the cervical and thoracic spine showed extensive transverse myelitis from C2 to T9, with faint peripheral contrast enhancement, while brain MRI showed small nonspecific white matter foci in the cerebral hemispheres. CSF analysis showed 92 cells/mm³ (80% lymphocytes), protein 94 mg/dl, a normal IgG index and kappa index, and type 2 oligoclonal bands. Serum testing revealed a strongly positive anti-AQP4 IgG titer, with negative anti-MOG IgG. Further immunological work-up showed ANA 1:160 and high-titer anti-histone antibodies. Based on these findings, the patient's history of arthralgias and Raynaud's phenomenon, and the mild-to-moderate pericardial effusion on cardiac ultrasound, a rheumatological evaluation was performed, which suggested the probable coexistence of a systemic rheumatic disease. The patient was treated with intravenous corticosteroids and plasma exchange sessions. Only mild clinical improvement followed after the 6th plasma exchange session, together with a significant reduction in the extent of the lesion on follow-up MRI (C6-T2).

Conclusions: This is a patient with acute longitudinally extensive transverse myelitis and positive anti-AQP4 antibodies, who fulfills the 2025 diagnostic criteria for NMOSD, with a coexisting systemic connective tissue disease. After acute-phase treatment with corticosteroids and plasma exchange, a rapid transition to long-term treatment with ravulizumab was made, following the first doses of meningococcal vaccines and the initiation of azithromycin chemoprophylaxis appropriate for complement inhibitors. This case once again underscores the value of shifting the therapeutic use of complement inhibitors from the chronic to the acute phase, as well as the challenges posed by refractory cases, for which novel, innovative options such as autologous stem cell transplantation and CAR-T cell therapies are being investigated.



R A R E C L I N I C A L C A S E S

LONGITUDINALLY EXTENSIVE TRANSVERSE MYELITIS IN AN ELDERLY PATIENT, DOUBLE SERONEGATIVE FOR AQP4 AND MOG AUTOANTIBODIES: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE

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Background: Longitudinally extensive transverse myelitis (LETM) is a potentially disabling spinal cord syndrome associated with a broad differential diagnosis, including inflammatory, autoimmune, infectious, vascular and neoplastic disorders. Establishing the underlying aetiology may be particularly challenging in older patients.

Case Presentation: A 77-year-old man was admitted with a two-month history of progressively worsening paraparesis that led to paraplegia, left-sided truncal neuropathic pain, and urinary and fecal retention. Neurological examination revealed bilateral sensory impairment with an approximate T6 level and paraplegia with reduced and tendon reflexes in lower limb showed crossed adductor reflex. Spinal cord MRI demonstrated a longitudinally extensive gadolinium enhanced lesion (L4-T4) without evidence of spinal vascular malformations. Cerebrospinal fluid analysis showed mildly elevated protein (100 mg/dL), normal glucose (63 mg/dL), and three cells; oligoclonal bands showed a type 4 pattern, with a normal kappa and IgG index. AQP4-IgG and MOG-IgG were negative. Infectious and paraneoplastic investigations, including multiplex PCR, West Nile virus IgM, antibodies associated with paraneoplastic neurological syndromes, chest and abdomen CT scans and PET-CT were unrevealing. Systemic autoimmune testing was largely negative, although anti-PM/Scl100 and anti-histone antibodies were positive. Immunofixation showed IgM monoclonal gammopathy. Serum ACE and electrophoresis were negative. The patient received five days of high-dose intravenous methylprednisolone, followed by tapering and five plasma exchange sessions, with limited response to plasma exchange.

Discussion: Differential diagnosis in this case of LETM is broad. Seronegative neuromyelitis optica spectrum disorder remains a consideration, while the autoimmune serological findings require correlation with the clinical phenotype. Given the patient's age and the diagnostic complexity, neoplastic etiologies such as primary intramedullary spinal cord lymphoma or IgM-monoclinal gammopathy of unknown significance should also be considered.

Conclusion: LETM in older adults requires a systematic and sometimes repeated diagnostic evaluation. Treatment of such cases highlights the therapeutic dilemmas both for the acute treatment and for the choice of the long-term therapeutic strategy.



R A R E C L I N I C A L C A S E S

RECURRENT OPTIC NEURITIS AND PERSISTENT DEMYELINATING LESIONS IN CHILDHOOD: A DIAGNOSTIC CHALLENGE

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Introduction: The differential diagnosis of acquired CNS demyelinating disorders in childhood is often challenging, particularly when clinical, neuroimaging, and immunological findings do not allow definitive disease classification.

Methods: Retrospective description of the clinical course, serial magnetic resonance imaging (MRI) findings, and immunological investigations of a currently 12-year-old child with recurrent optic neuritis, with symptom onset at age 7.

Aim: To highlight the diagnostic and therapeutic dilemmas in a child with recurrent optic neuritis and persistent CNS demyelinating lesions.

Results: A 7-year-old girl presented in March 2022 with acute bilateral optic neuritis following COVID-19 infection, initially normal brain MRI, and type 3 oligoclonal bands (OCBs) in cerebrospinal fluid (CSF). In December 2023, she experienced a second episode of bilateral optic neuritis, again following COVID-19 infection. Brain MRI revealed multiple non-enhancing demyelinating lesions involving the periventricular and subcortical white matter and corpus callosum. Repeat CSF analysis demonstrated type 4 OCBs, while MOG and AQP4 antibodies were negative. Further investigations for infectious, systemic autoimmune, and metabolic disorders were negative. Both episodes were treated with intravenous methylprednisolone, resulting in complete recovery of visual acuity. Serial MRI examinations through September 2025 demonstrated persistent brain lesions, with slight enlargement of two lesions but no new lesions, while spinal MRI was normal. Due to persistent neuroimaging abnormalities, comprehensive reassessment in February 2026 revealed type 1 OCBs, negative CSF kappa free light chains (κ-FLC), and weakly positive serum anti-MOG antibodies. The patient remains relapse-free, with stable MRI findings through September 2026, without long-term immunomodulatory treatment.

Conclusions: The coexistence of recurrent optic neuritis, persistent demyelinating lesions, and evolving immunological biomarkers creates diagnostic uncertainty between multiple sclerosis, MOG antibody-associated disease (MOGAD), and other demyelinating disorders. Longitudinal multimodal reassessment is essential for accurate diagnostic classification and individualized therapeutic decision-making.



R A R E C L I N I C A L C A S E S

ANTI-NMDAR ENCEPHALITIS IN A CHILD: FROM NEUROPSYCHIATRIC MANIFESTATIONS TO SEVERE NEUROLOGICAL DETERIORATION — A CASE REPORT

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Introduction: Anti-N-methylD-aspartate receppor (anti-NMDAR) encephalitis is characterized by diverse neuropsychiatric, seizure-related, and movement manifestations, with potential for severe clinical deterioration requiring intensive immunotherapy.

Methods: Retrospective description of the clinical course, diagnostic investigation, treatment, and outcome of an 8-year-old boy with anti-NMDAR encephalitis.

Aim: To highlight the progressive course of the disease and the clinical response to a combined immunotherapy protocol.

Results: An 8-year-old boy presented with subacute behavioral changes beginning during the summer months, with marked deterioration in September, including intense fears, disorganized speech and severe insomnia, followed by focal seizures. Brain magnetic resonance imaging was normal, whereas elctroencephalography documented left temporal activity. Cerebrospinal fluid (CSF) analysis revealed mild pleocytosis and type 3 oligoclonal bands. Detection of anti-NMDAR antibodies in both serum and CSF confirmed the diagnosis. Investigations for infectious agents, systemic autoimmune diseases, and neoplastic conditions were negative. Despite initial treatment with intravenous immunoglobulin (IVIG) and high-dose methylprednisolone (30 mg/kgr for five days in a row), further deterioration occurred, with orofacial and lingual dyskinesias, hypotonia, and severe gait impairment. Immunotherapy was promptly escalated in the pediatric intensive care unit using a combined PLEX-IVIG-rituximab protocol, comprising six plasma exchange sessions, IVIG administration, and two doses of rituximab administered after the first and final plasma exchange sessions. This was followed by marked improvement in neuropsychiatric symptoms and sleep, along with gradual recovery of motor function, supported by physiotherapy and symptomatic treatment with clonidine, clobazam, quetiapine, and lacosamide. Hospitalization was complicated by central venous catheter-associated deep vein thrombosis of the right lower limb, managed with anticoagulation treatment. Significant neurological and functional improvement was observed at discharge.

Conclusions: Anti-NMDAR encephalitis may progress to severe neurological. Deterioration despite initial immunotherapy. Prompt implementation of a combined immunotherapy protocol, alongside supportive care, may be associated with substantial neurological and functional recovery.



R A R E C L I N I C A L C A S E S

ACUTE AND SEVERE MULTIFOCAL NEUROLOGICAL PRESENTATION IN AN ADOLESCENT WITH DEMYELINATING LESIONS SHOWING IMAGING FEATURES RAISING SUSPICION OF BALÓ-TYPE DEMYELINATION

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Introduction: Acquired inflammatory demyelinating disorders of the central nervous system in children and adolescents exhibit considerable clinical and radiological heterogeneity. Acute, severe multifocal neurological deficits accompanied by extensive white matter lesions pose diagnostic challenges, particularly when some lesions display morphological features raising suspicion of Baló-type demyelination.

Methods: We report the case of a 13-year-old boy presenting with acute multifocal neurological deficits. Clinical findings, initial and follow-up brain magnetic resonance imaging (MRI), cerebrospinal fluid (CSF) analysis, serum and CSF immunological investigations, treatment, and clinical outcome were reviewed.

Aim: To highlight the diagnostic challenges associated with acute, severe multifocal neurological manifestations and atypical MRI findings in paediatric acquired demyelinating syndromes.

Results: The patient presented with dysarthria, right central facial palsy, left upper limb weakness (Medical Research Council grade 3/5), and a mild left-sided spastic-ataxic gait with circumduction. Initial brain MRI revealed multiple bilateral periventricular, deep, and juxtacortical white matter lesions, including involvement of the corpus callosum and callososeptal interface. Some lesions exhibited a radial orientation resembling Dawson's fingers, while other morphological features raised suspicion of Baló-type demyelination. Restricted diffusion was observed in one right periventricular lesion. CSF analysis showed mild pleocytosis, type III oligoclonal bands, and an elevated IgG index of 1.100. Anti-myelin oligodendrocyte glycoprotein (MOG) and anti-aquaporin-4 (AQP4) antibodies were negative. Treatment with intravenous methylprednisolone followed by intravenous immunoglobulin resulted in significant clinical improvement.

Conclusions: The clinical presentation, MRI findings, and CSF profile support an inflammatory demyelinating disorder of the central nervous system. The morphological features of some lesions raise suspicion of Baló-type demyelination, although confirmation requires careful assessment of the original MRI images. Pediatric multiple sclerosis remains an important diagnostic consideration; however, a definitive diagnosis cannot be established from the available findings alone. Clinical and radiological follow-up is essential to clarify the disease course and establish the final diagnosis.



R A R E C L I N I C A L C A S E S

SEVERE PEDIATRIC PAN-NEUROFASCIN ANTIBODY-ASSOCIATED NODOPATHY: DIAGNOSTIC AND THERAPEUTIC CHALLENGES"

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Objectives: Pan-neurofascin antibody-associated nodopathy is a rare autoimmune disorder, particularly in children, characterized by severe peripheral nerve dysfunction and potentially poor response to conventional immunotherapy

Methods: A previously healthy 5-year-old girl presented with progressive lower-limb weakness, frequent falls, Gowers's sign, and areflexia. CSF analysis revealed albuminocytologic dissociation while MRI demonstrated cauda equina nerve root enhancement. Nerve conduction studies showed absent F-waves, suggesting polyradiculopathy. Initially diagnosed with Guillain–Barré syndrome (GBS), she received IVIG (2 g/kg), with transient improvement. Subsequent deterioration prompted a second IVIG course, again with limited benefit.

Results: Extended antibody testing revealed strongly positive anti-NF155, anti-NF186, and anti-NF140 antibodies, (NF155 IgG4 subclass detected), establishing the diagnosis of pan-neurofascin antibody-associated nodopathy. Despite three weekly rituximab doses (375 mg/m²) and complete peripheral B-cell depletion, she continued to deteriorate, losing ambulation and developing upper-limb weakness. Repeat EMG suggested early axonal involvement. Subsequent antibody testing revealed even higher antibody titers, predominantly of the IgG1 subclass. Six plasma exchange (PLEX) cycles over 12 days were administered alongside high-dose methylprednisolone (30 mg/kg/day for 5 days) and IVIG following each cycle. Clinical improvement began after the third PLEX cycle. Neurofascin antibodies became undetectable following completion of treatment. At latest follow-up, she had regained independent ambulation and continued to demonstrate clinical improvement while undergoing corticosteroid tapering.

Conclusion: This case highlights the importance of considering autoimmune nodopathy in children with an atypical or treatment-refractory GBS course. Rituximab, PLEX combined with corticosteroids may represent an effective rescue strategy in severe pan-neurofascin antibody-associated disease.



R A R E C L I N I C A L C A S E S

PERIPHERAL NERVE HYPEREXCITABILITY SYNDROME(PNHS) - MORVAN SYNDROME, IN A PEDIATRIC PATIENT WITH POSITIVE VGKC-COMPLEX ANTIBODIES, CASPR2 AND LGI1.

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Background: PNHs comprise a rare and underdiagnosed entity in children. Clinical manifestations range from isolated neuromyotonia in cramp-fasciculations syndrome, to co-existing autonomic dysfunction in Isaac syndrome, sleep disturbance, agitation and memory impairment in Morvan syndrome and seizures, fluctuations in consciousness and cognitive impairment in limbic encephalitis.

Case report: We present a 13-year-old boy with lower back pain, significant weight loss, hyperhidrosis, hypertension, insomnia and generalized myokymia. Muscle strength and sensory examination were normal. Thyrotoxicosis and underlying neoplasia were excluded following an extensive endocrinological evaluation and whole-body imaging, including brain and spinal MRI, chest and abdomen CT, MIBG and PET-CT. Electromyography demonstrated myotonic discharges, with prominent neuromyotonic discharges. Creatine kinase levels, CSF cell count, and CSF protein levels were normal. Serum antibodies against the voltage-gated potassium channel (VGKC) complex, CASPR2, and LGI1 were positive, whereas all three were negative in the CSF. Oligoclonal bands were classified as type 1. Given the presence of myokymia and autonomic dysfunction in the absence of encephalopathy, Morvan syndrome was considered the most likely diagnosis. Treatment with carbamazepine resulted in complete remission of symptoms within several days. By the time the antibody results became available, the patient was asymptomatic; therefore, immunotherapy was not initiated. At the 6-month follow-up, antibodies against VGKC complex, CASPR2, and LGI1 were undetectable in serum.

Conclusion: This case highlights the diagnostic complexity of PNHs in the pediatric population. Despite the polyautoimmunity the patient achieved complete and sustained clinical remission on a membrane-stabilizing agent without the need of immunotherapy.



R A R E C L I N I C A L C A S E S

ATYPICAL OPTIC NEUROPATHY IN A CHILD: INFLAMMATION OR NEOPLASM? A CASE REPORT

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Introduction: Optic neuritis is an important cause of acute visual impairment in childhood. However, certain optic nerve neoplasms may present with similar clinical and imaging features, posing diagnostic and therapeutic challenges.

Methods: Retrospective description of the clinical course, ophthalmological and imaging findings, and therapeutic management of a 6-year-old girl with atypical optic neuropathy.

Aim: To highlight the importance of longitudinal clinical and imaging reassessment in differentiating inflammatory from neoplastic optic neuropathy.

Results: In March 2026, a 6-year-old girl presented with painless, progressive visual impairment in the right eye (1/10) and a relative afferent pupillary defect (RAPD). Brain and orbital magnetic resonance imaging (MRI) revealed right optic nerve enlargement and contrast enhancement, initially suggestive of optic neuritis. Extensive investigations for demyelinating, autoimmune, and infectious disorders were inconclusive, with negative anti-MOG and anti-AQP4 antibodies. Intravenous methylprednisolone resulted in initial visual improvement (6/10), followed by oral corticosteroids. Following treatment discontinuation, visual acuity deteriorated again (1/10), accompanied by right optic disc edema and pallor. Despite a second course of methylprednisolone and escalation of immunomodulatory treatment with intravenous immunoglobulin and rituximab, severe visual impairment persisted. Serial MRI examinations demonstrated persistent enlargement, optic nerve sheath expansion, increased T2 signal, and marked contrast enhancement of the intraorbital segment of the right optic nerve. The atypical clinical and imaging course raised suspicion of optic nerve glioma, prompting initiation of chemotherapy.

Conclusions: The lack of sustained clinical response to immunotherapy and persistence of imaging abnormalities warrant reconsideration of the initial diagnosis of inflammatory optic neuritis. Early investigation of a possible neoplastic etiology is essential for appropriate therapeutic management.



R A R E C L I N I C A L C A S E S

PRESENTATION OF TWO PATIENTS WITH COEXISTING SARCOIDOSIS AND MULTIPLE SCLEROSIS, WITH A DIFFERENT SEQUENCE OF ONSET. DIFFERENTIAL DIAGNOSTIC AND THERAPEUTIC QUESTIONS.

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Background: The coexistence of sarcoidosis and Multiple Sclerosis (MS) is of particular clinical interest, as it raises questions regarding the diagnostic approach, a possible immunopathogenetic association, and therapeutic management. The present study aims to describe two patients from our Center with a different chronological sequence of diagnosis of the two diseases.

Methods: A retrospective review of the patients' available clinical and imaging data was performed. The chronological sequence of diagnoses, clinical manifestations, treatment course, and disease evolution were recorded, alongside consideration of the relevant literature on comorbidity and HLA associations.

Results: The first patient, a 73-year-old woman, had a history of sarcoidosis with pulmonary and cutaneous manifestations since 1992, for which she received corticosteroids, with no reported need for further specific treatment. In 1997, she was diagnosed with MS, initially presenting with unsteadiness, paresthesias, and eyelid ptosis, subsequently following a secondary progressive course. Following successive therapeutic interventions, she is receiving rituximab, with an EDSS score of 6.5 and no imaging evidence of disease activity at the latest available follow-up. The second patient, a 54-year-old man, was diagnosed with MS in 1999, presenting with blurred vision and diplopia. He sequentially received interferons and fingolimod and has been receiving ocrelizumab since 2022. The incidental detection of mediastinal lymphadenopathy in 2024 prompted further investigation and a diagnosis of sarcoidosis in 2025, currently under observation without specific treatment. At the latest assessment, an EDSS score of 2.5 and a stable neuroimaging lesion burden were recorded.

Discussion: The different chronological sequence of diagnoses highlights the importance of recognizing comorbidity and individualizing monitoring and treatment according to the activity of each disease. In the literature, *HLA-DRB1*15:01* has been associated with increased susceptibility to both diseases, while protective associations have been reported for *DRB1*01* in MS and *DRB1*01:01* in sarcoidosis. Conversely, *DRB1*11:01* has been reported to show a protective association with MS and increased susceptibility to sarcoidosis in specific populations. These associations in both concordant and opposing directions reinforce interest in investigating the immunogenetic basis of this comorbidity.

Conclusion: Our Center's experience highlights the importance of individualized monitoring and reinforces interest in further investigation of the immunogenetic basis of this comorbidity and the investigation of the potential involvement of medications for MS in the development of sarcoidosis.



R A R E C L I N I C A L C A S E S

RARE CASE OF A FEMALE PATIENT WITH ADOLESCENT-ONSET MULTIPLE SCLEROSIS (POMS)AND MULTIPLE ENDOCRINE NEOPLASIA SYNDROME (MEN2B): MULTIPLE IMMUNOPHARMACOLOGICAL CHALLENGES AND DILEMMAS DUE TO THE RARE COMORBIDITY

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Background: The incidence of cancer in MS has been investigated for several years, as not only does it affect ongoing therapeutical decisions, but also, certain disease-modifying treatments (DMTs) may increase the risk of tumorigenesis.

Methods: For the first time, we present a case of a female patient with pediatric onset MS (POMS) and multiple endocrine neoplasia 2B (MEN2B) and analyze the immunological impact of these diseases on the therapeutical choiceç, at the long term.

Results: A. Neurological history: A 28-year-old woman of Hellenic origin was referred to Aeginition University Hospital with dizziness, walking instability and double vision. She had these symptoms from the age of 14 and in 17 years of age, she was diagnosed with MS. After corticosteroid pulse therapy. Two years earlier, she showed symptoms of dysarthria and Bell's palsy and started receiving glatiramer acetate. For the following two years, she had exacerbations with optic neuritis, which subsided after receiving i.v glucocorticoids. Three years later, her treatment was altered to Teriflunomide. Due to repeated relapses involving diplopia and new, gadolinium-enhancing demyelinating lesions, she was ultimately treated with ocrelizumab. B. Endocrinological history: The patient was diagnosed with multiple endocrine neoplasia-2B (MEN2B) at 15 years of age. She received motesanib for 6 years, discontinued following termination of the clinical trial, and vandetanib for 11 years, discontinued because of grade II–III adverse events-AEs. Selpercatinib was initiated at a reduced dose because of a low body mass index. During 18 months of treatment, calcitonin and carcinoembryonic antigen (CEA) levels declined (calcitonin: 11,631 to 2,000 pg/mL; CEA: 463 to 305 ng/mL), accompanied by stabilization of structural disease. Chronic diarrhea, attributed to elevated calcitonin levels and vandetanib-related toxicity, as well as grade II-III skin AEs resolved completely. The patient gained 5 kg, enabling more regular exercise and contributing to improvement in MS.

Discussion: The choice for pharmaceutical agents for both diseases was a real difficult task, with an extensive discussion between neurologist and endocrinologist, since the international literature on the comorbidity and mainly on mutual therapeutic approaches was very limited. Beyond direct RET-kinase inhibition, selpercatinib may modulate the tumour microenvironment by relieving suppression of innate immune responses. Given the role of T-lymphocytes in adaptive immunity, concomitant treatment with anti-CD20 agent ocrelizumab may not directly interfere with this proposed pathway. However, this mechanistic interpretation requires further investigation.

Conclusion: Some rare comorbidities demand a continuous cooperation among various specialties and a profound knowledge of immunopharmacological aspects.



R A R E C L I N I C A L C A S E S

SERONEGATIVE AUTOIMMUNE CEREBELLAR ATAXIA IN CHILDHOOD: A CASE REPORT

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Introduction: Pediatric seronegative immune-mediated cerebellar ataxia (IMCA) remains an ill-defined and frequently unrecognized diagnosis, particularly in young children, whose symptoms are often erroneously attributed to self-limiting post-infectious conditions.

Method: Review of the medical history and relevant literature concerning a girl who, at the age of 2.5 years, presented with acute onset of cerebellar ataxia (mSARA score at diagnosis: 14), with gradual resolution over a two-year period.

Objective: To present a rare case of childhood-onset seronegative IMCA—a condition primarily described in adults—thereby potentially broadening the recognized age range of the disorder, given that very few similar cases have been reported in the literature to date.

Results: Analysis of the cerebrospinal fluid (CSF) revealed pleocytosis and presence of oligoclonal bands, while serial brain imaging and extensive screening for autoantibodies were negative. However, indirect immunofluorescence/immunohistochemistry (TIIF/IHC) of the CSF demonstrated a positive intracellular signal in cerebellar Purkinje cells, a finding that supports a diagnosis of isolated seronegative IMCA. The patient was administered intravenous immunoglobulin (IVIG) and corticosteroids (both intravenously and orally) and showed gradual improvement over a two-year period (mSARA score:1). Clinical improvement and decrease of serum biomarkers NfL and particularly GFAP levels, was associated mainly with corticosteroids, supporting the intracellular origin of the signal.

Conclusions: Isolated seronegative IMCA remains unrecognized and unclassified within the pediatric population. Further research is required to determine whether this condition falls within the spectrum of autoimmune encephalitis or represents a distinct clinical entity, analogous to primary autoimmune cerebellar ataxia in adults. We propose the incorporation of the TIIF/IHC method into the diagnostic workup for cerebellar ataxia syndromes, as it can aid in the diagnosis of seronegative cases and guide personalized therapeutic approaches. Among emerging biomarkers, NfL and GFAP are promising for disease monitoring, underscoring the need for their validation in larger patient cohorts.



R A R E C L I N I C A L C A S E S

SEIZURES OF AUTOIMMUNE ORIGIN: LIMBIC EPILEPSY IN A PEDIATRIC PATIENT WITH APECED

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Introduction: Autoimmune epilepsy is characterized by seizures directly caused by immune-mediated brain inflammation and accounts for 5-10% of unexplained pediatric cases. It should be strongly suspected in patients with pre-existing multisystem autoimmunity.

Objectives: To describe autoimmune limbic epilepsy in a child with genetically confirmed autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED).

Methods: An 8-year-old boy with autoimmune hypoparathyroidism, Hashimoto thyroiditis, and vitiligo, subsequently recognized as manifestations of APECED, presented with recurrent afebrile focal and generalized tonic-clonic seizures, despite levetiracetam treatment. Seizures were preceded by aura-like symptoms, including headache, dizziness, abdominal pain, facial flushing.

Results: Initial EEG, CSF analysis, and cranial CT were unremarkable. However, three serial brain MRI examinations over 10 months demonstrated progressive asymmetric hippocampal abnormalities, with right hippocampal enlargement, FLAIR hyperintensity and increased ASL perfusion, followed by left hippocampal volume loss. These findings, together with the clinical course, raised suspicion of autoimmune limbic encephalitis.

Repeat EEG showed left hemispheric sharp waves. Oligoclonal bands showed a type 4 pattern in serum and CSF. Serum antibodies were positive for Ri/ANNA-2 and anti-GAD, while NMDA-receptor and MOG antibodies were negative. Pancreatic islet autoantibodies were strongly positive, and vaccine-specific antibody titres were markedly reduced. Autoimmune limbic epilepsy was diagnosed.

Intravenous methylprednisolone followed by rituximab for six months resulted in clinical and neuroimaging improvement. Whole-exome sequencing identified a pathogenic AIRE variant, confirming APECED. Type 1 diabetes mellitus developed shortly after discharge. Two years later, seizures recurred without recurrent limbic MRI abnormalities, and oxcarbazepine was added.

Conclusions: Autoimmune neurological manifestations should be considered in children with multisystem autoimmune disease, including APECED, presenting with seizures. Neuronal autoantibodies and evolving hippocampal abnormalities on serial MRI may support an autoimmune mechanism, even when initial investigations are unrevealing. Metabolic disturbances related to hypoparathyroidism or diabetes may coexist and should not preclude consideration of an inflammatory cause.



R A R E C L I N I C A L C A S E S

MYASTHENIA IN THE PEDIATRIC POPULATION: A REPORT OF TWO CASES

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Introduction: Juvenile myasthenia gravis in children is a rare autoimmune disorder characterized by fluctuating symptoms ranging from ocular signs and generalized weakness (mastication, speech, limbs) to severe respiratory distress.

Purpose: To describe two distinct clinical cases that highlight the significant challenges in the diagnosis and long-term therapeutic management of the disease.

Methods / Results: Case 1: A 7-year-old girl presented with sudden-onset strabismus of 10 days' duration following a head injury, with no initial ptosis and an unremarkable history. Brain imaging was negative for orbital fracture or mass. Subsequent testing revealed positive serum acetylcholine receptor antibodies. In the interim, her condition deteriorated, presenting with ophthalmoplegia and fluctuating ptosis with characteristic worsening at the end of the day, but without any generalized fatigue. Due to partial improvement with pyridostigmine, prednisolone (2 mg/kg) was added, yielding clinical improvement without full recovery of ocular motility. Over three years of follow-up, she exhibits exacerbations and remissions confined strictly to the eyes, managed with pyridostigmine adjustments and an alternate-day low-dose prednisolone regimen.

Case 2: A 15.5-year-old female adolescent with obesity, features of anxiety disorder, and otherwise unremarkable medical history reported upper limb numbness and mild generalized weakness of 3 months' duration, worsening over the past month with lower limb involvement. Symptoms showed no diurnal fluctuation, and ptosis was absent. Clinical examination was normal except for reduced muscle strength (4/5). Functional (psychogenic) causes were initially included in the differential diagnosis. On further investigation, the ice pack test was positive despite the absence of ptosis at rest, and electrophysiological testing indicated a neuromuscular junction disorder. Positive acetylcholine receptor antibodies confirmed the diagnosis of Myasthenia Gravis. Thymoma imaging was negative. Prednisolone was added due to inadequate symptom control with pyridostigmine alone.

Conclusions: In conclusion, pediatric myasthenia remains a major clinical challenge due to its rarity and fluctuating symptoms that complicate prompt diagnosis, while treatment requires complex, highly individualized management.



OTHER TOPICS

RESEARCH TO IDENTIFY A QUESTIONNAIRE FOR ASSESSING THE IMPACT OF MUSIC ON HEALTHY INDIVIDUALS AND PATIENTS WITH MULTIPLE SCLEROSIS, IN THE HELLENIC POPULATION

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Introduction: Music is an integral part of our daily lives and has the unique ability to activate multiple brain regions. Music therapy is a non-pharmacological, multifaceted therapeutic intervention that utilizes music as an ameliorative regimen for addressing cognitive, social and psychological requirements. In order to thoroughly investigate the therapeutic effects of music, it is essential to select appropriate, psychometrically validated tools. However, the limited availability of Greek-language music related questionnaires highlights the need for cross-cultural adaptation and psychometric evaluation of instruments originally developed in other languages.

Methods: A literature search was conducted in scientific databases, such as PubMed, and Google Scholar, with keywords related to the effects of music, psychometric evaluation of instruments, Greek-language adaptation, and multiple sclerosis (MS). The relevance of the measured dimensions, the validity and reliability of the instruments, and a Greek translation were carefully examined.

Objective: A review of the beneficial effects of music in both healthy individuals and people with MS revealed a research gap in Greece concerning this clinical population. For this reason, selecting an appropriate questionnaire is necessary for the future assessment of various aspects of music's effects in both healthy participants and individuals with MS, aiming to gain a deeper understanding of the potential therapeutic benefits of music, especially in people with MS.

Results: The Brief Music Experience Questionnaire (Brief MEQ), consisting of 53 items across six subscales, was selected: Commitment to music, Innovative musical aptitude, Social uplift, Affective reactions, Positive psychotropic effects, Reactive musical behavior. A Greek translation is available. however, no published psychometric validation has been identified.

Conclusions: The present study aims to improve the existing Greek translation of the Brief MEQ and subsequently assess its validity and reliability in a sample of 100 participants, including 50 healthy individuals and 50 people with MS. The aim is to provide a foundation for the future use of the questionnaire in this clinical population.



A CASE OF ADOLESCENT-ONSET MULTIPLE SCLEROSIS 32 YEARS AGO

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She was 15 years old—an age when other children played carefree in the street. While others rode bikes or went for walks, she wouldn't go out; instead, she would sit on the sofa watching television. Sometimes she played alone, putting together puzzles, or simply chatted with friends. She felt a heaviness in her eyes; people assumed it was because she was a good student who studied hard and was simply tired. An ophthalmologist's examination revealed no issues, prescribing only natural tears to rest her eyes. Yet, despite studying for school the next day, her academic performance did not always reflect her effort; she would forget things or get them mixed up. It was around the age of sixteen that headaches began to appear. An X-ray revealed nothing serious—just a cyst in the maxillary sinus. She was also plagued by chronic tonsillitis, frequently suffering from fevers and requiring antibiotics. A close friend of hers, Dimitris, had infectious mononucleosis; had she caught it from him, or were her tonsils the root cause? Was it difficult to distinguish the viral infection from purulent tonsillitis? She underwent a tonsillectomy at seventeen. The rest followed at eighteen: severe headaches and vision loss in her left eye. The diagnosis was disclosed only to her parents. I should also mention that she would have outbursts of laughter—to the point of tears—and generally maintained a cheerful disposition. She did not let her spirits sink too low, as she always strove to move forward—a process greatly aided by her love for science and reading. As a university student, she would often study using only her one eye that retained some vision, while the other had no sight at all. Another significant detail dates back to when she took piano lessons at the age of twelve; her teacher would often remark on her beautiful, rounded fingers. It was also observed that the weight of her touch varied depending on the hand—right or left—while she played. Conversely, her primary school teacher used to tell her that her handwriting was poor and difficult to read; she still struggles with her right hand and does not write well. She thought she was writing with rounded letters, but in the end, they were always illegible—I wonder why?





Notes





Notes

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