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MULTIPLE SCLEROSIS: A CASE REPORT

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Abstract

This case report provides a comprehensive overview of multiple sclerosis (MS), focusing on the latest understanding and management strategies for the condition. MS is a complex, disabling disorder that has significant social and economic consequences. It can affect any part of the central nervous system, which results in a highly variable clinical presentation, making diagnosis and treatment challenging. Although the underlying aetiology of MS is still not known, we summarise those with most evidence of association. This review highlights that progressive MS is an area where there is currently a paucity of available disease – modifying treatments and this will be a major focus for future development. The brain and spinal cord imaging during diagnostics in the Department of Neurology at the University Clinical Center of Kosova for Supratentorial lesions, Infratentorial lesions, T1 lesions, Gadolinium detected more than 50 present lesions, bile lesions spread widely in the two hemispheres. Central Pons left. Ped. cereb. dex. Bilateral cerb. Many lesions in T1. GL is not given. Cases like this one predominantly affect young adults, with a higher prevalence in women. Recent advances in the understanding of MS pathophysiology, including insights into immune system dysregulation, neuroinflamation, and the role of the blood – brain barrier, have significantly shaped the evolution of therapeutic approaches.

Keywords: multiple sclerosis, autoimmune condition, progressive multiple sclerosis, diagnostic criteria, case report.

Introduction

Multiple sclerosis (MS) is a chronic, unpredictable neurological condition that affects the central nervous system (CNS), causing wide range of symptoms and disabilities. Characterized by the immune system attacking the protective covering of nerve fibers (myelin), MS disrupts normal nerve signal transmission, leading to both physical and cognitive challenges. The disease manifests in different forms, with patients experiencing periods of relapse and remission, or in some cases, a gradual progression of disability. While the exact cause of MS remains unclear, it is thought to involve a combination of genetic, environmental, and immune system factors. MS predominantly affects young adults, and impact on individuals can be profound, influencing quality of life and posing significant social and economic burdens. Despite considerable advances in understanding its pathophysiology, there is still no cure for MS, but ongoing research and the development of disease – modifying therapies offer hope for better management and improved outcomes for those living with the condition.

Case Report

A 16-year-old female presented with right-sided diplopia and difficulty with eye movements, starting 7 days prior. She had no prior neurological symptoms, and her medical history was unremarkable. She denied smoking, alcohol, or substance use, and there was no family history of autoimmune or neurological diseases.

Clinical Examination

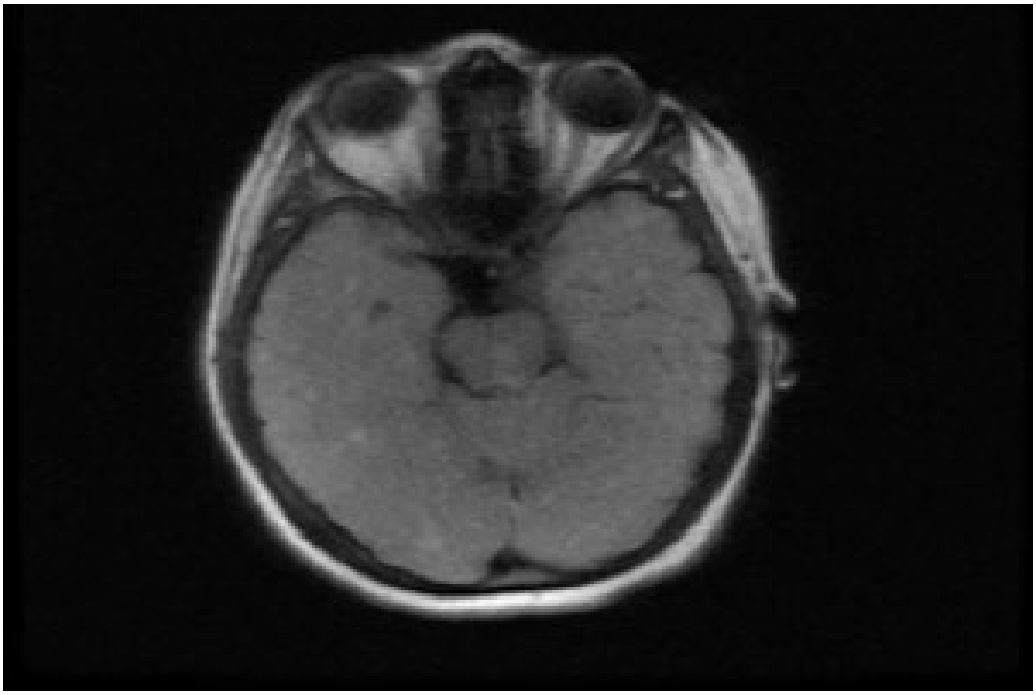

Figure 1. Axial T2-weighted MRI of the brain. The image reveals prominent hyperintense lesions in the periventricular region and corpus callosum, indicative of demyelination. (University Clinical Center of Kosovo – Clinic of Neurology)

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MULTIPLE SCLEROSIS CASE REPORT

Arbëresha Tërpuni

Doctor of Medicine

Neurological examination revealed right internuclear ophthalmoplegia (INO), characterized by horizontal nystagmus in the left eye and impaired adduction of the right eye on horizontal gaze. Mild right-sided weakness and persistent nystagmus, despite initial corticosteroid therapy, indicated ongoing neuroinflammation.

Imaging Findings

MRI of the brain and cervical spine showed over 50 hyperintense lesions on T2-weighted images, involving both supratentorial and infratentorial brain regions, as well as multiple cervical spinal cord levels (C2, C4-5, C6-7, T1-T3). Gadolinium enhancement indicated active inflammation and blood-brain barrier disruption, suggestive of an aggressive course of multiple sclerosis (MS).

Cerebrospinal Fluid (CSF) and Laboratory Findings

CSF analysis revealed oligoclonal bands (OCBs) with two bands, indicating intrathecal antibody synthesis, and a slightly elevated IgG index, consistent with MS. Negative aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein (MOG) antibody tests (AQP4 0.576) ruled out neuromyelitis optica spectrum disorder (NMOSD) and MOG-associated disease.

Treatment and Management

The patient was treated with intravenous methylprednisolone (IVMP) for 5 days, leading to partial improvement in diplopia and right-sided strength. Following IVMP, oral corticosteroids were prescribed for tapering. Given the significant lesion burden and intrathecal inflammation, disease-modifying therapy (DMT) was initiated to reduce relapse risk and prevent neurodegeneration, which is crucial in pediatric MS to mitigate early disability.

Differential Diagnosis

The main conditions to consider in this case were multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), and MOG antibody-associated disease. MS was the most likely diagnosis based on the patient's clinical presentation, MRI findings, and cerebrospinal fluid (CSF) profile. The presence of numerous gadolinium-enhancing lesions, oligoclonal bands, and an elevated IgG index strongly supports MS. The patient's improvement with corticosteroids also aligns with typical MS relapses. NMOSD was ruled out due to the absence of AQP4 antibodies and the lack of characteristic MRI findings. MOG antibody-associated disease was excluded based on negative MOG antibodies and MRI features, further supporting MS.

Imaging and CSF Findings

MRI revealed numerous lesions throughout the brain and spinal cord, with gadolinium enhancement indicating active inflammation and blood-brain barrier disruption, common in relapsing-remitting MS. Pediatric MS often presents with a higher lesion burden than adult-onset MS, possibly due to a more pronounced inflammatory response in younger patients. CSF analysis showed oligoclonal bands and a mildly elevated IgG index, typical of MS, suggesting

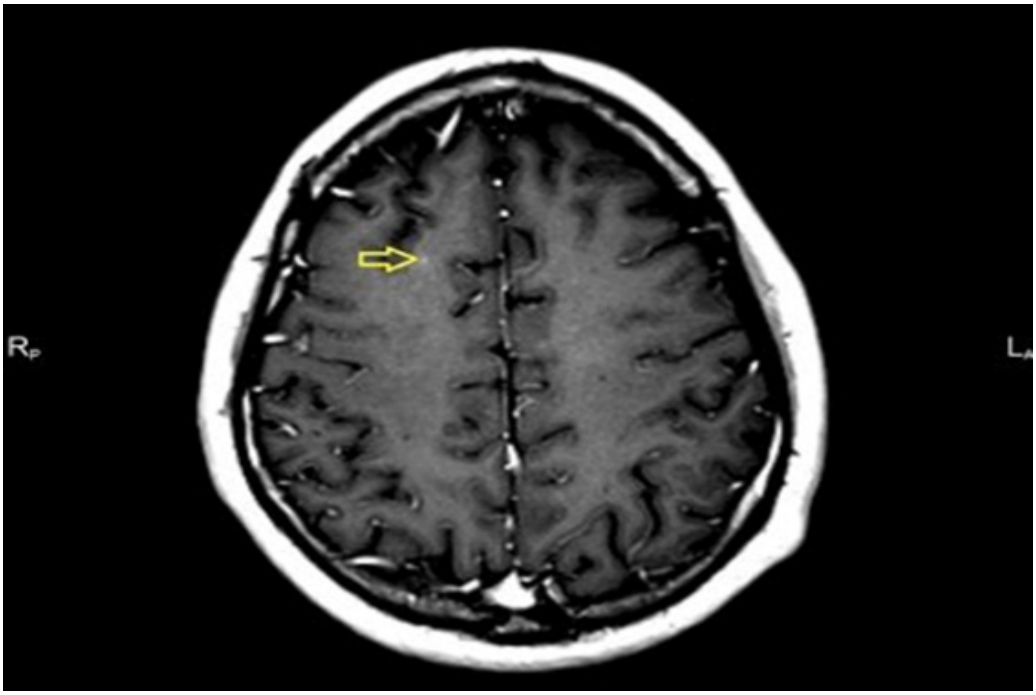

Figure 2. Axial T1-weighted MRI with gadolinium contrast enhancement. The image shows an enhancing lesion (indicated by the yellow arrow) located in the right frontal lobe, adjacent to the central sulcus.

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chronic CNS inflammation. Oligoclonal bands are present in up to 95% of MS cases and, when observed with compatible clinical and imaging findings, are a strong indicator of MS.

Implications for Management

The high lesion burden in this patient suggests an aggressive form of MS, requiring more intensive treatment and closer monitoring than in adult-onset cases. Early initiation of disease-modifying therapy (DMT) is crucial in pediatric MS to reduce relapses and prevent long-term disability. While the patient showed partial improvement with corticosteroids, the high lesion load indicates that long-term DMT is essential to prevent future relapses and further CNS damage. The patient will continue to be monitored with follow-up MRIs and neurological assessments to track disease activity and therapy effectiveness, as pediatric MS often results in a higher cumulative disability over time, warranting aggressive management.

Discussion

This case represents an aggressive presentation of pediatric-onset multiple sclerosis (POMS), characterized by a very high lesion burden, multifocal CNS involvement, and evidence of ongoing inflammatory activity. Although MS is classically considered a disease of young adults, pediatric cases account for up to 10% of all MS diagnoses, with a small subset presenting before adolescence. These younger patients often display a more inflammatory disease phenotype, with frequent relapses and rapid lesion accumulation on MRI, as seen in this case.

The radiologic findings in this patient are striking. More than 50 hyperintense lesions on T2-weighted images involving both supratentorial and infratentorial regions, in addition to extensive spinal cord involvement, indicate widespread demyelination. The presence of multiple gadolinium-enhancing lesions points to active inflammation and blood–brain barrier disruption. In children, such extensive dissemination of lesions is less common but is associated with a more aggressive course and a higher risk of early disability if not treated promptly. Involvement of both the brainstem and cervical spinal cord also correlates with more severe initial neurological symptoms, including diplopia and limb weakness, as seen in this patient.

The cerebrospinal fluid (CSF) findings further support the diagnosis of MS. The detection of oligoclonal bands and an elevated IgG index reflect intrathecal synthesis of immunoglobulins, which is a well-established hallmark of MS. Importantly, the negative aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein (MOG) antibody results helped to exclude neuromyelitis optica spectrum disorder (NMOSD) and MOG antibody-associated disease (MOGAD). These disorders can mimic MS, particularly in pediatric patients, but they differ in their immunopathology, prognosis, and treatment response. NMOSD typically shows longitudinally extensive spinal cord lesions and optic neuritis without disseminated brain lesions, while MOGAD often presents with large, poorly demarcated brain lesions that resolve more completely between attacks. The pattern of multifocal brain and spinal lesions with persistent OCB positivity is much more consistent with MS.

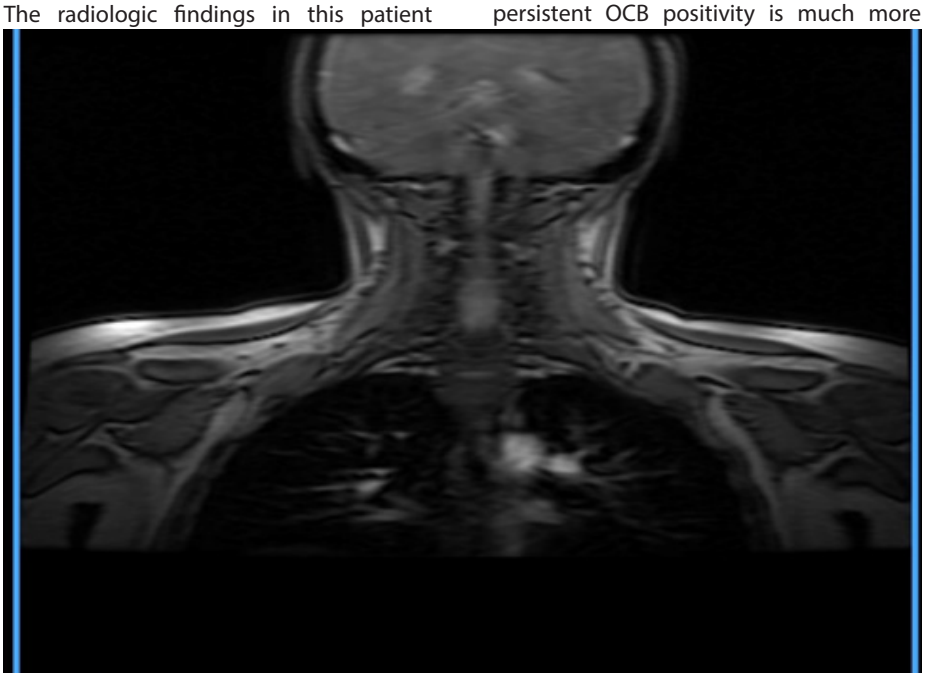

Figure 3. Magnetic resonance imaging done shows lesions in the Cervical part (C spine 3 – pl loc).

Pathophysiologically, pediatric MS involves immune-mediated demyelination driven by autoreactive T and B lymphocytes, leading to inflammation, myelin loss, and axonal injury. The disease appears to have a stronger inflammatory component in children than in adults, which may explain the extensive MRI findings. Although children often recover better from relapses due to greater neuroplasticity, the frequent inflammatory attacks contribute to cumulative neuronal loss and may result in earlier cognitive decline or disability if the disease is not adequately controlled.

The therapeutic response observed after intravenous methylprednisolone (IVMP) supports the diagnosis of MS, as relapses in MS typically show good short-term improvement with corticosteroids. However, the partial rather than complete recovery also highlights the need for early initiation of disease-modifying therapy (DMT). In pediatric MS, early and effective immunomodulation is crucial—not only to reduce relapse frequency but also to protect neurodevelopment and cognitive function. Increasing evidence favors the early use of high-efficacy agents such as fingolimod, natalizumab, or ocrelizumab in children with aggressive disease courses similar to this one. These therapies have demonstrated significant reductions in relapse rates and MRI activity, translating into better long-term outcomes.

The long-term prognosis in pediatric MS is influenced by early disease activity and treatment response. Although children often experience a relapsing–remitting course initially, the interval between relapses may shorten over time, and conversion to a secondary progressive phase can occur earlier than in adults. Cognitive impairment, fatigue, and psychosocial challenges are also common and can significantly impact quality of life and academic performance. Therefore, management should extend beyond pharmacologic therapy to include multidisciplinary care—neurological follow-up, neuropsychological evaluation, physical rehabilitation, and educational support.

From a diagnostic standpoint, this case emphasizes the importance of a comprehensive evaluation in pediatric demyelinating diseases. Overlapping features among MS, NMOSD, and MOGAD can lead to diagnostic uncertainty, especially early in the disease course. The combination of typical MRI findings,

positive OCBs, and the exclusion of other demyelinating antibody-mediated conditions was essential in establishing a definitive diagnosis of MS. Continued monitoring with serial MRI and clinical assessments remains important to track disease activity and guide therapeutic adjustments.

In summary, this case highlights the challenges of diagnosing and managing pediatric-onset MS, particularly when it presents with such an extensive lesion burden and active inflammation. Early recognition and timely initiation of effective DMTs are critical to minimizing irreversible neurological damage and preserving long-term function. This case reinforces the concept that pediatric MS is not simply an early form of adult MS but rather a distinct, highly inflammatory disease that requires vigilant follow-up and an aggressive, individualized treatment approach.

Conclusion

This report describes a 16-year-old female presenting with suspected pediatric-onset multiple sclerosis (MS), characterized by a high lesion burden and significant neurological symptoms, including right-sided internuclear ophthalmoplegia and weakness. The diagnosis of MS was supported by MRI findings showing multiple gadolinium-enhancing lesions throughout the CNS, the presence of oligoclonal bands in the cerebrospinal fluid (CSF), and an elevated IgG index. The negative results for AQP4 and MOG antibodies effectively excluded neuromyelitis optica spectrum disorder (NMOSD) and MOG antibody-associated disease.

This case emphasizes the critical importance of early and accurate diagnosis in pediatric MS cases. Pediatric-onset MS often progresses more aggressively than adult-onset MS, with a higher frequency of relapses and an increased risk of early disability. Early initiation of disease-modifying therapy is crucial to reduce inflammation, prevent further relapses, and slow disease progression. This case underscores the necessity for increased clinical awareness and a thorough, multidisciplinary approach to managing pediatric MS, as it can significantly affect a patient's quality of life and long-term outlook.

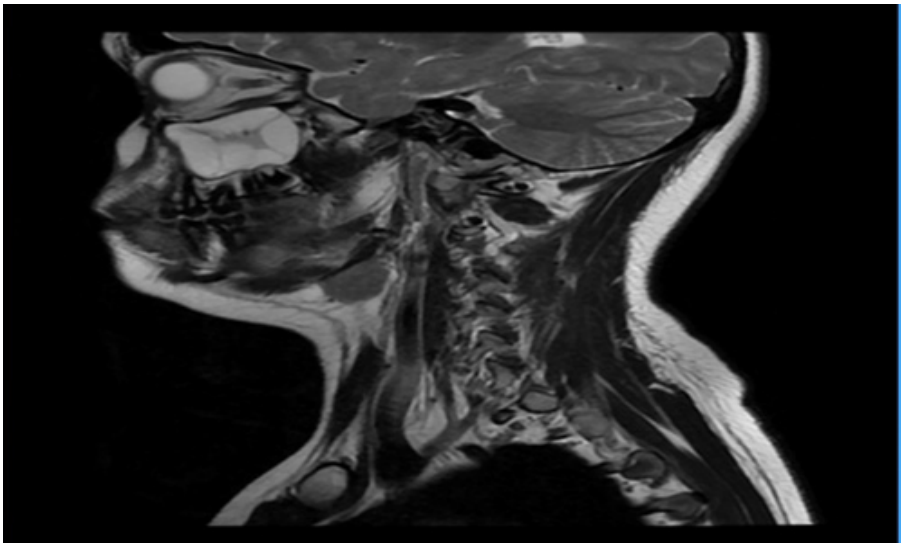

Figure 4. Magnetic resonance imaging (MRI) done shows lesions in the Cervical part (Sag T2 frFSE).

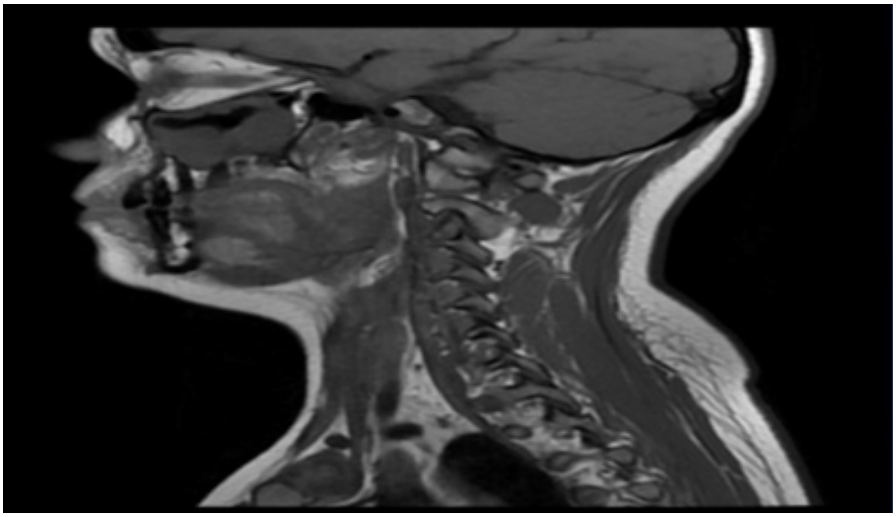

Figure 5. Magnetic resonance imaging (MRI) done shows lesions in the Cervival part (Sag T1).

Referencat:

1.Rovira A, Wattjes MP, Tintoré M, et al. MAGNIMS consensus guidelines on the use of MRI in multiple sclerosis - clinical implementation in the diagnostic process. Nat Rev Neurol. 2015;11(8):471-82.

2.Oh J, Vidal-Jordana A, Montalban X. Multiple sclerosis: clinical aspects. Curr Opin Neurol. 2018;31(6):752-9.

3.Geraldes R, Ciccarelli O, Barkhof F, et al. The current role of MRI in differentiating multiple sclerosis from its imaging mimics. Nat Rev Neurol. 2018;14(4):213-31.

4.Brownlee WJ, Hardy TA, Fazekas F, Miller DH. Diagnosis of multiple sclerosis: progress and challenges. Lancet. 2017;389(10076):1336-46.

5.Krajnak KM, Ko JJ, Zed PJ. The impact of implementing standardized risk stratification for venous thromboembolism prophylaxis in hospitalized medical patients. J Thromb Thrombolysis. 2023;55(2):416-25.

6.Filippi M, Rocca MA, Ciccarelli O, et al. MRI criteria for the diagnosis of multiple sclerosis: MAGNIMS consensus guidelines. Lancet Neurol. 2016;15(3):292-303.

7.Thompson AJ, Baranzini SE, Geurts J, Hemmer B, Ciccarelli O. Multiple sclerosis. Lancet. 2018;391(10130):1622-36.

8.Kaiser CC, Maghzi AH, Levy M, et al. Neuroinflammatory disorders: differential diagnosis in the era of myelin oligodendrocyte glycoprotein antibodies. Front Neurol. 2019;10:877.

9.Arnold DL, You X, Yau B, et al. Brain atrophy and disability worsening in patients with relapsing–remitting multiple sclerosis: a 12-year follow-up of the pivotal interferon beta-1a trial. Mult Scler J. 2021;27(1):77-86.

10.Dobson R, Giovannoni G. Multiple sclerosis - a review. Eur J Neurol. 2019;26(1):27-40.v)

Table 1. Data from the neurological examination.

INO dex	The dex eye does not go to the right and nystagmus appears, at the same time the eye does not go medially
EN (after IVMP)	The dex eye goes completely laterally, the sin eye follows almost medially

Table 2. Data from the ophthalmological visit.

	VOD	VOS
Opthalmological Visit	0.7	0.7