ARTIGO ORIGINAL/ORIGINAL ARTICLE

Acute Non-Neoplastic Myelopathies in Pediatric Age: A Decade of Experience from a Pediatric Center

Mielopatias Agudas Não Neoplásicas em Idade Pediátrica: Uma Década de Experiência de um Centro Pediátrico

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Abstract

Introduction: Pediatric myelopathies encompass a broad range of non-traumatic and non-neoplastic etiologies, including inflammatory, vascular, and infectious causes. Despite their low prevalence, the differential diagnosis and management of these conditions remain challenging due to the diversity of clinical manifestations and progression patterns.

Methods: A retrospective observational study was conducted, including children up to 18 years of age diagnosed with acute non-neoplastic myelopathy at our pediatric center between 2010 and 2021. Clinical data, complementary examinations, treatments, and functional outcomes were analyzed from hospital records, with a comparison across different etiologies.

Results: Thirty-six cases were included (50% male), with a mean age at diagnosis of 10.8 years (SD±5.3 years). The identified etiologies were: demyelinating and inflammatory (16 cases), vascular (13), infectious (3), and idiopathic (4). Muscle weakness was the most frequent initial symptom (68.9%), followed by sensory complaints (48.3%). Pain as a presenting symptom was reported in 20% of cases and was associated with idiopathic etiology (p=0.05). Hyperacute onset was characteristic of vascular myelopathies ($p<0.001$), whereas 75% of demyelinating and inflammatory myelopathies presented with subacute onset. Magnetic resonance imaging (MRI) findings revealed that brain lesions were more associated with demyelinating and inflammatory etiologies ($p=0.003$), while anterior spinal cord lesions in axial sections were linked to vascular etiologies ($p=0.018$). A worse functional outcome was observed in cases with hyperacute symptom onset ($p=0.024$).

Conclusion: Pediatric myelopathies exhibit distinct clinical and radiological features depending on the etiology. Symptom onset and imaging findings play a critical role in both etiological identification and functional prognosis. The sample from this study displays characteristics consistent with the existing literature. Understanding the particularities of these conditions, particularly in the pediatric population, is essential for defining an effective clinical approach with the potential to positively impact the quality of life of patients and their families.

Resumo

Introdução: As mielopatias pediátricas apresentam uma ampla variedade de etiologias não traumáticas e não neoplásicas, incluindo causas inflamatórias, vasculares e infeciosas. Apesar da sua baixa prevalência, o diagnóstico diferencial e a abordagem destas situações permanecem desafiantes, devido à diversidade de manifestações clínicas e padrões de evolução.

Métodos: Realizou-se um estudo observacional retrospetivo, envolvendo crianças até aos 18 anos diagnosticadas com mielopatia aguda não neoplásica no nosso centro, entre 2010 e 2021. Dados clínicos, exames complementares, tratamentos e prognóstico funcional foram analisados a partir dos processos hospitalares, com comparação entre diferentes etiologias.

Resultados: Foram incluídos 36 casos (50% do sexo feminino), com idade média ao diagnóstico de 10,8 anos (DP 5,3). As etiologias identificadas foram: desmielinizante e inflamatória (16 casos), vascular (13), infecciosa (3) e idiopática (4). Fraqueza muscular foi o sintoma inicial mais frequente (68,9%), seguida de queixas sensitivas (48,3%). Dor como manifestação inicial foi relatada em 20% dos casos, associando-se à etiologia idiopática (p=0,05). O início hiperagudo foi mais característico das mielopatias vasculares (p<0,001), enquanto 75% das mielopatias desmielinizantes e inflamatórias apresentaram início subagudo. A ressonância magnética revelou que lesões cerebrais estavam mais associadas a etiologias desmielinizantes e inflamatórias (p=0,003), enquanto lesões medulares anteriores em corte axial relacionaram-se com etiologias vasculares (p=0,018). O prognóstico funcional foi pior nos casos com início hiperagudo dos sintomas (p=0,024).

Conclusão: As mielopatias pediátricas apresentam características clínicas e radiológicas específicas, dependendo da etiologia. O início dos sintomas e os achados de imagem desempenham um papel crucial, em termos diagnósticos e também prognósticos. A amostra deste estudo partilha características consistentes com o descrito na literatura. Compreender as particularidades destas condições, especialmente na população pediátrica, é essencial para definir uma abordagem clínica eficaz, com potencial para impactar positivamente a qualidade de vida dos doentes e das suas famílias.

Introduction

Myelopathy encompasses a broad spectrum of etiologies, including inflammatory, infectious, degenerative, vascular, traumatic, and neoplastic causes. In pediatric populations, intramedullary lesions, though rare, can lead to significant morbidity.[1] Acute myelopathy (AM) is characterized by symptoms that reach their peak within 21 days, distinguishing it from subacute or chronic conditions.[2] Diagnostic approaches integrate demographic, clinical, and imaging data. Key clinical signs include corticospinal tract involvement, spinothalamic dysfunction, and sphincter disturbances.[3] Once these signs are identified, magnetic resonance imaging (MRI) becomes pivotal in diagnosing non-compressive etiologies and guiding further testing.[4] Among non-neoplastic AM, inflammatory and vascular causes are the most prevalent.[2]

AM in children arises from diverse etiologies, with acute transverse myelitis (ATM) being a predominant non-compressive cause, accounting for approximately 20% of pediatric cases and affecting an estimated 2 million children annually.[5,6] The causes of ATM include infections (e.g., herpes viruses), vascular events and systemic inflammatory disorders (e.g., systemic lupus erythematosus),[7,8] demyelinating diseases such as multiple sclerosis (MS), acute disseminated encephalomyelitis (ADEM), neuromyelitis optica spectrum disorders (NMOSD)[9] and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD).[10]

Pediatric MS (PMS) accounts for 3%–10% of all MS cases, with an estimated prevalence of 2.5 per 100 000

children.[11] Relapsing-remitting MS (RRMS) is the most common form in pediatric patients, with most diagnoses occurring between the ages of 11 and 13.[12,13] ADEM is a monophasic demyelinating disorder affecting the central nervous system (CNS), typically presenting with encephalopathy and multifocal neurological deficits. It is more common in younger children (mean age 5–8 years) and frequently follows infections or vaccinations.[14,15] Testing for myelin oligodendrocyte glycoprotein (MOG) antibodies, found in 33%–66% of pediatric ADEM cases, supports diagnosis and helps distinguish ADEM from other demyelinating conditions.[16]

Acute myelitis of infectious origin may result from direct neuronal damage (true infectious myelitis) or post-infectious immune-mediated responses. Viral agents such as enteroviruses, coxsackieviruses, and herpesviruses are the most common pathogens involved.[17,18] In addition to motor and sensory disturbances typical of myelopathy, radiculopathy may occur in some cases of infectious myelitis, manifesting as lower motor neuron signs. Advances in imaging techniques and the identification of autoimmune markers have significantly reduced the number of idiopathic inflammatory myelitis diagnoses, enabling more accurate differentiation from infectious causes.[19]

Vascular causes, including spinal cord infarctions, are extremely rare in children and pose significant diagnostic challenges.[20] These events are typically associated with acute motor weakness, sensory deficits, and autonomic dysfunction, depending on the lesion's location.[21] Spinal cord ischemia may occur following minor trauma, arterial disruption, thrombosis, or as a complication of thoracic or abdominal surgery.[20,22] Anterior spinal artery infarction, frequently involving the thoracolumbar spine, is the most characteristic vascular lesion. Early recognition and prompt management are essential to minimizing long-term disability through targeted medical treatment and intensive physiotherapy.

Studies evaluating the clinical, imaging, and laboratory profiles of acute myelopathy in the pediatric population remain limited. This study aims to characterize pediatric patients diagnosed with acute myelopathy and treated at a tertiary care center, through a comparative analysis of clinical presentations, imaging findings, and laboratory data across different etiologies. These results are contextualized within the current literature to enhance understanding and inform future clinical practice.

Methods

A retrospective observational study was conducted at a tertiary pediatric center. Patients diagnosed with AM over an 11-year period, from January 1, 2010, to December 31, 2021, were included. Ethical approval for the study was obtained from the local Ethics Committee.

The study population included children under 18 years of age presenting with AM. Exclusion criteria comprised spinal cord compression, intramedullary neoplasms, and a prior history of malignancy. Patients were eligible if they exhibited acute motor and/or sensory symptoms, with or without sphincter dysfunction, progressing over a maximum period of 21 days. Imaging studies were required to exclude compressive etiologies. Based on the underlying cause, patients were classified into four categories: demyelinating and inflammatory, infectious, vascular and idiopathic.

Demographic information, including age at onset and sex, was collected along with a medical history review encompassing prior infections and vaccinations within the previous 30 days. Clinical symptoms were assessed in terms of motor, sensory, and bladder or bowel dysfunction, with the presence of back pain as an initial symptom recorded in specific cases. The time to maximum neurological deficit was categorized as hyperacute (<6 hours), acute (6-24 hours), or subacute (>24 hours to 21 days). Outcomes were evaluated using the Paine and Byers scale, classifying recovery from "normal" (complete recovery) to "severe" (disabling deficits). A "good" prognosis was defined as complete recovery or the presence of only mild residual symptoms.

Laboratory analysis included cerebrospinal fluid (CSF) testing for pleocytosis, glucose and protein levels, IgG index, and the presence of oligoclonal bands. Serum antibodies, including anti-MOG and anti-AQP4, were assessed in 12 of the 36 patients, reflecting the availability of advanced diagnostic techniques in recent years. Infectious causes of AM were confirmed by serological evidence of recent infection, while idiopathic cases were defined by the exclusion of all other etiologies. Patients with vascular AM secondary to scoliosis surgery were excluded from MRI and laboratory comparisons due to missing initial clinical data.

Spinal and brain MRI findings were evaluated for lesion location (cervical, thoracic, lumbar, or conus medullaris), axial topography (anterior, posterior/lateral, or central), and lesion extent. Lesions extending over more

than three vertebral segments on sagittal T2-weighted images were classified as longitudinally extensive transverse myelitis (LETM). Gadolinium enhancement and the presence of brain lesions were also assessed.

Statistical analyses were performed using IBM SPSS® version 23. Categorical variables were expressed as absolute and relative frequencies, while continuous variables were reported as mean ± standard deviation. Comparative analyses were conducted using the chi-square test, ANOVA, and Kruskal-Wallis test, with statistical significance set defined as $p<0.05$.

This methodology allowed a comprehensive assessment of pediatric AM, emphasizing its diverse etiologies and informing future diagnostic and therapeutic approaches.

Results

1 – Study population

We included 36 cases of AM, with an equal male-to-female distribution (18 females, 1:1 ratio). The mean age at symptom onset was 10.8 ± 5.3 years (range 1-17 years). There were no statistically significant differences in age between the different etiological groups ($p=0.297$).

2 – Clinical characteristics

Among the 29 patients included in the analysis (seven patients with iatrogenic vascular myelopathy related to scoliosis surgery were excluded), the most common presenting symptom was motor impairment, observed in 20 patients (68.9%), followed by sensory disturbances in 14 (48.3%) and sphincter dysfunction in eight (27.6%). Back pain was reported as the initial symptom in four patients (13.8%). Six patients (20.7%) presented with isolated motor involvement at clinical onset, while three (10.3%) had exclusive sensory symptoms. Combined motor and sensory involvement was observed in six patients (20.7%). No cases of isolated bowel or bladder dysfunction were recorded. None of the children in this cohort reported vaccination within one month before the onset of AM, although five (17.2%) had a documented history of infection in the preceding 30 days. Regarding the timing of symptom onset, 16 patients (55.2%) experienced a subacute presentation, seven (24.1%) presented acutely, and six (20.7%) had a hyperacute onset. The main clinical characteristics of the study population are summarized in **Fig. 1**.

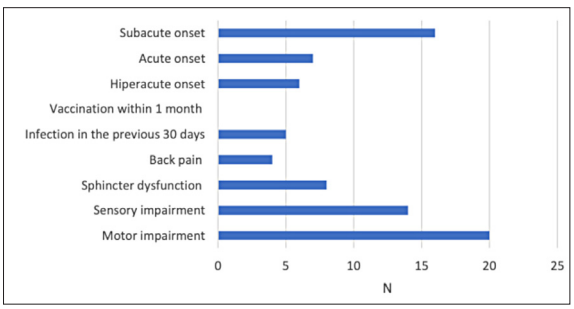

Figure 1. Clinical findings at presentation in AM. Time to reach maximum neurological deficit was classified as hyperacute (< 6 hours), acute (6-24 hours) and subacute (>24 hours to 21 days).

3 – MRI characteristics

Spinal MRIs from 29 patients with initial symptoms of AM (excluding seven with iatrogenic vascular myelopathy following scoliosis surgery, due to uncertain timing of deficit onset) were analyzed. Brain lesions were identified in 18 patients (62.1%). Regarding spinal lesion topography, cervical involvement was observed in 20 patients (68.9%), thoracic (dorsal) in 18 (62.1%), lumbar in one patient (3.4%), and conus/distal cord lesions in six patients (20.7%).

In terms of axial lesion distribution, 10 patients exhibited a central cord pattern, 14 had posterior/lateral lesions, and five showed anterior spinal cord involvement.

4 – Etiologies

Demyelinating and inflammatory myelopathies (n=16; 46%) were the most prevalent conditions in our cohort. The differential diagnosis included MS (n=11), ADEM (n=4) (diagnosis based on the International Paediatric Multiple Sclerosis Study Group – IPMSSG – criteria for pediatric MS and immune-mediated CNS demyelinating disorders [2013 revision], as well as 2017 McDonald criteria), and Fisher's inflammatory myeloradiculitis with anti-GQ1b antibody (n=1).

Thirteen patients (n=13; 37%) were diagnosed with AM of vascular etiology: seven cases were related to scoliosis surgery, two resulted from minor trauma, one was associated with Chiari type I malformation, one was due to renovascular hypertension, and two were considered idiopathic. Infectious AM was identified in three patients (6%), with diagnosis confirmed by positive serology for HSV1, *Mycoplasma pneumoniae*, and *Coxsackievirus*. Finally, four patients (11%) were diagnosed with idiopathic AM after excluding all aforementioned etiologies.

5 – Acute treatment

The medication of choice for the acute treatment of all patients was intravenous methylprednisolone, administered at a dose of 30 mg/kg per day for 3 to 5 days. Antibiotics or antiviral drugs were added in high-risk patients with clear evidence of infection.

6 – Subgroup comparisons

6.1 – Clinical manifestations and etiology

The presence of acute back pain at onset was more commonly associated with idiopathic myelopathy compared to other etiologies ($p=0.05$). While weakness was present in all patients with idiopathic, infectious, and vascular AM, a lower frequency was observed in the inflammatory and demyelinating group (43.8%), which was statistically significant ($p=0.017$). Patients with demyelinating and vascular etiologies (18.8% and 16.7%, respectively) were less likely to have sphincter involvement compared to those with infectious etiology (66.7%) and idiopathic AM (50.0%), although this difference did not reach statistical significance ($p=0.2$). Regarding sensory symptoms, no statistically significant differences were found between the various etiologies ($p=0.7$). All patients with vascular etiology had a hyperacute onset ($p<0.001$), while 75% of patients presenting with subacute onset had demyelinating or inflammatory etiologies ($p<0.001$). Thus, the time course of deficit onset in our cohort differed significantly depending on the underlying etiology.

6.2 – MRI characteristics and etiology

Spinal cord and brain MRI findings were compared across different etiologies. Cranioencephalic lesions were identified in 18 patients, 14 of whom had a demyelinating etiology, particularly ADEM or paediatric MS. None of the patients with vascular myelopathy presented cranioencephalic lesions ($p=0.003$). Lesions were most frequently located in the cervicodorsal region on sagittal imaging across all etiological subgroups. Involvement of the distal cord or conus medullaris was observed in five out of 29 patients: three with a demyelinating etiology, one with infectious myelopathy, and one with idiopathic myelopathy. On axial sections, lesions with anterior topography were significantly more common in vascular etiologies (83.3%) compared to other conditions ($p<0.05$). In children with MS, lesions more frequently involved the posterolateral cord. A central lesion pattern was observed across all etiologies except vascular. Although longitudinally extensive transverse myelitis (LETM) was less frequently seen in children with inflammatory or demyelinating disease (31.0% of cases), this difference did not reach statistical significance when compared with other

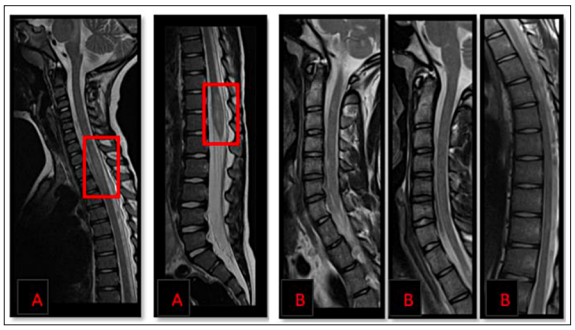

Figure 2. Pediatric myelopathies secondary to demyelinating diseases (sagittal T2-weighted images). (A) ADEM: A 4-year-old patient presenting with encephalopathy and extensive spinal cord signal changes involving the central cord from C7 to D4, as well as the conus medullaris. (B) MS: A 16-year-old patient with motor and sensory deficits. MRI shows typical multiple, small, and short-segment lesions (each <3 vertebral segments) at the cervical level (C5) and dorsal levels (D2-D3, D5-D6, and D9-D10).

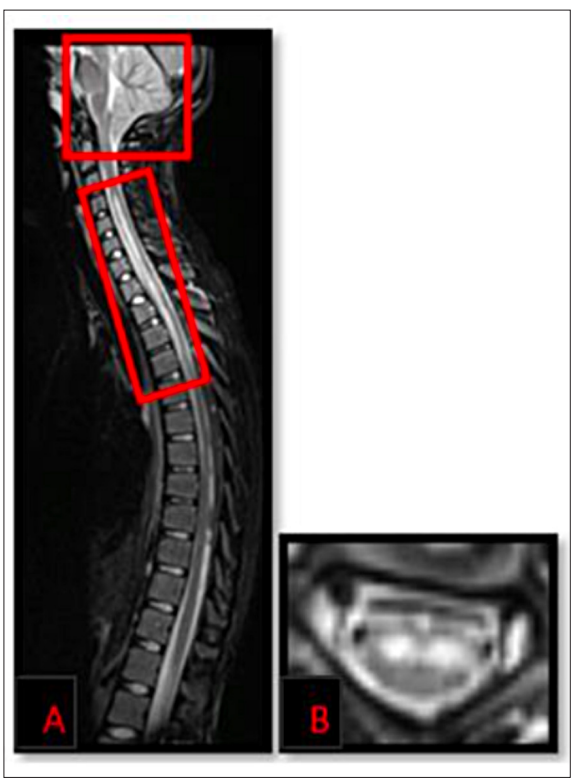

Figure 3. Spinal cord MRI of an 11-year-old patient with Chiari malformation type I. (A) (Sagittal STIR sequence (T2-weighted with fat suppression)) Caudal displacement of the cerebellar tonsils greater than 5mm, with extensive hyperintensity and swelling of the cervical and thoracic spinal cord (C4-D8). (B) (Axial T2-weighted sequence) Lesions predominantly affecting the anterior horn region.

etiologies (p=0.1). Gadolinium-enhancing lesions (Gd+) were found in eight patients, six of whom met the McDonald criteria for paediatric MS. Gd+ lesions were less common among other etiologies. **Figs. 2** and **3** illustrate the varying lesion topographies depending on the underlying diagnosis.

6.3 – CSF profile and etiology

The profile of initial CSF samples obtained after symptom onset was available for 23 patients. Among those with demyelinating conditions (n=16), of the four patients diagnosed with ADEM, one showed elevated CSF protein levels (>40 mg/dL), another had no abnormal findings, and the remaining two presented both pleocytosis and elevated protein levels. Among the 11 patients diagnosed with paediatric MS, eight (72.7%) had oligoclonal bands detected by isoelectric focusing, while fewer than 20% showed pleocytosis and elevated protein levels. The patient with Fisher's myeloradiculitis presented with both pleocytosis and elevated protein levels (>40 mg/dL). In the group with infectious myelopathy (n=3), one patient exhibited both pleocytosis and elevated protein levels, one had an isolated increase in the IgG index, and one showed no CSF abnormalities. Among the four patients with idiopathic myelopathy (n=4), three (75%) had elevated protein levels; two of them also presented oligoclonal bands and pleocytosis. However, when comparing CSF findings across the different etiological groups, no statistically significant differences were observed.

6.4 – Serum antibodies and etiology

Anti-AQP4 and anti-MOG antibodies were tested in 12 patients: one with infectious myelopathy, three with idiopathic etiology and eight with demyelinating conditions. All patients tested negative for anti-AQP4 antibodies. Anti-MOG antibodies were positive in three patients, all of whom were diagnosed with ADEM.

6.5 – Outcome and etiology

The outcome was compared across different etiologies of AM, and the main results are summarized in **Table 1**. Approximately 50% of pediatric patients with AM achieved complete recovery. A good outcome was observed in 14 patients (87.5%) with demyelinating or inflammatory conditions. In contrast, 11 patients (84.6%) with vascular etiology had poor or fair outcomes, a statistically significant difference (p=0.003).

6.6 – Outcome with clinical onset and MRI results

Analysis of outcome groups across clinical and imaging variables revealed no significant differences. However, a statistically significant difference was observed when comparing prognosis based on symptom onset (p=0.024): a subacute presentation was associated with a better outcome (**Table 2**).

Discussion

The study evaluated 36 children with acute myelopathy (AM), analyzing their clinical, neuroimaging, and laboratory features to compare findings with existing

Table 1. Patient outcomes (classified using the modified Paine and Byers scale) according to etiology.

Outcome*	Demyelinating and inflammatory (n=16)	Idiopathic (n=4)	Infectious (n=3)	Vascular (n=13)	Total (n=36)
Good	14/16 (87.5%)	2/4 (50.0%)	2/3 (66.7%)	2/13 (15.3%)	20/36 (55.6%)
Fair	1/16 (6.3%)	1/4 (25.0%)	1/3 (33.3%)	6/13 (46.2%)	9/36 (25.0%)
Poor	1/16 (6.3%)	1/4 (25.0%)	0	5/13 (38.5%)	7/36 (19.4%)

Table 2. Patient outcomes according to the clinical onset of symptoms.

Clinical onset of symptoms	Poor	Fair	Good
Hyperacute	3 (60.0%)	2 (40.0%)	1 (5.3%)
Acute	1 (20.0%)	2 (40.0%)	6 (31.6%)
Subacute	1 (20.0%)	1 (20.0%)	12 (63.2%)

literature. The cohort consisted of 18 boys and 18 girls, consistent with previous reports indicating no significant sex predominance in AM.[23] The mean age at diagnosis was 10.8 ± 5.2 years, supporting findings of a teenage predominance, although a bimodal age distribution has also been described.[24-26] The youngest patient, aged one year, presented with an inflammatory etiology, underscoring the broad age range affected by AM.

Clinical presentations varied according to etiology, with motor weakness being the most common symptom. Back pain was reported in 14% of cases, a lower frequency compared to other studies where rates reached up to 60%.[27,28] In our cohort, back pain was more frequently associated with idiopathic etiologies, contrasting with the established link between back pain and anterior spinal artery syndrome. However, the small sample size limits definitive conclusions regarding pain patterns.

The timing of symptom onset and progression may provide clues to the etiology of AM. Hyperacute presentations are suggestive of vascular causes, whereas subacute symptom progression is more consistent with inflammatory myelopathies, corroborating previous studies.[9,29]

CSF and serum analyses are essential in the evaluation of suspected AM. In this study, 34.8% of patients exhibited normal CSF protein levels and white blood cell counts, aligning with prior reports indicating that 20%–50% of children with AM may have normal CSF findings.[26,28,30] Inflammatory markers such as pleocytosis, elevated protein, increased IgG index, and oligoclonal bands (OCBs) raise suspicion for demyelinating, infectious, or other inflammatory etiologies of AM.

Acute infectious myelitis is typically characterized by pronounced CSF inflammation, including pleocytosis and markedly elevated protein concentrations, often accompanied by systemic infectious symptoms. Diagnostic confirmation usually depends on positive PCR results for specific pathogens in the CSF or on evidence of acute and convalescent serum antibody titers.[26] Oligoclonal band (OCB) positivity was identified in 91.7% of pediatric MS cases in our cohort, reaffirming its diagnostic significance, and was also detected in two idiopathic cases. Antibody testing revealed anti-MOG positivity in 25% of the 12 patients tested, all diagnosed with ADEM. This finding is consistent with previous research linking MOG antibodies to demyelinating syndromes.[31] Despite advancements in diagnostic technologies, antibody screening in this study was inconsistent, reflecting its retrospective design and underscoring the need for standardized protocols for laboratory testing.

Neuroimaging confirmed a predominance of lesions in the cervicothoracic spinal cord, consistent with prior studies.[30,32] In pediatric MS, lesions tended to be shorter, multifocal, and primarily localized to the cervical cord, distinguishing them from ADEM and other etiologies.[33,34] This study corroborates findings by Alper et al, who reported LETM in 66%–85% of children with acute transverse myelitis (ATM).[30] Axial MRI patterns in vascular myelopathies demonstrated characteristic anterior horn involvement, often described as "snake-eyes" or "owl-eyes," which were also observed in our cohort (**Fig. 3**). Gadolinium enhancement, although commonly reported in AM, was present in less than 20% of patients in this cohort, a rate comparable to previous studies reporting 20%–30% prevalence.[30] This lower frequency may be explained by the interval between symptom onset and imaging.[32] Brain MRI revealed asymptomatic lesions in 40% of idiopathic cases, facilitating diagnosis, particularly of demyelinating conditions,[33,34] and serving as a predictor of potential relapses.[35]

Empiric treatment with high-dose corticosteroids remains the standard of care for inflammatory AM. All patients in this study received intravenous methylprednisolone, with adjunctive antibiotics or antiviral agents administered when infectious etiologies were suspected. These treatment approaches align with evidence supporting the efficacy of corticosteroids in managing central nervous system inflammatory disorders.[36,37] Treatment continuation and adjustments were guided by findings from complementary diagnostic evaluations.[38]

Hyperacute presentations were associated with poorer outcomes, consistent with previous studies.[25,37] The most reported sequelae include sensory disturbances and sphincter dysfunction, affecting 15% to 50% of patients. Approximately one-quarter of affected individuals require walking assistance, and 10% to 20% never regain mobility or bladder control.[9] In this cohort, at the time of data collection, 20% of patients exhibited persistent bladder dysfunction, 11% required assistance for ambulation, and 14% presented with severe motor impairments.

This study has several limitations, including its single-center, retrospective design and relatively small sample size, which may introduce selection bias. Furthermore, incomplete data regarding clinical, imaging, and labora-

tory findings, as well as inconsistencies in antibody testing, limit the strength of the conclusions. Future prospective, multicenter studies are needed to provide a more comprehensive understanding of prognostic factors in pediatric AM. Nonetheless, despite these limitations, this study highlights the critical importance of early diagnosis and individualized management in improving outcomes and quality of life for affected patients.

Conclusion

Acute myelopathies of non-neoplastic origin are rare in pediatric populations. However, they can result from a wide range of etiologies, each with distinct clinical, neuroimaging, and laboratory profiles. A thorough understanding of these conditions, particularly when they present early in life, is essential for guiding effective treatment strategies. Timely and appropriate intervention has the potential to significantly improve the quality of life for affected patients and their families over the short, medium, and long term.

As a single-center study, this research offers valuable insights for refining diagnostic and therapeutic approaches within the specific population served by our institution. Nonetheless, broader conclusions require validation through future studies involving larger and more heterogeneous cohorts. Continued research in this field is crucial to advancing the understanding of non-neoplastic acute myelopathies in children and optimizing their clinical management. ■

Prémios e Apresentações Anteriores / Awards and Previous Presentations:

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Contributorship Statement / Declaração de Contribuição

RNR: Research, conception, statistical analysis, organization and elaboration of the manuscript.

MP: Contributed to data collection, preparation and revision of the manuscript.

AC: Organization, elaboration and revision of the manuscript.

RF and FP: Made substantial contributions to the study concept and design, and critically reviewd the manuscript.

All authors approve the final version to be published.

All authors revised the manuscript for important intellectual concepts and gave final approval for the publication of the version.

RNR: Pesquisa, conceção, análise estatística, organização e elaboração do manuscrito.

MP: Contribui para recolha de dados, preparação e revisão do manuscrito.

AC: Organização, elaboração e revisão do manuscrito.

RF e FP: Deram um contributo substancial para o conceito e desenho do estudo, fizeram revisão crítica do manuscrito.

Todos os autores aprovam a versão final a ser publicada.

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