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Neuroimaging feature in identifying acute myelopathy etiologies: comparison between neuromyelitis optica spectrum disorder and cervical spondylotic myelopathy

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Abstract

Objective The clinical symptoms of neuromyelitis optica spectrum disorder (NMOSD) and acute cervical spondylotic myelopathy (CSM) may overlap in some cases. This study aimed to investigate the differences in imaging features between NMOSD and CSM in acute myelopathy.

Methods We included 78 patients in this retrospective study, including 28 NMOSD patients and 50 CSM patients. The demographic characteristics and clinical symptoms of the two groups of patients were compared. The T1 signal intensity, length of the spinal cord involved by T2 hyperintensity, degree of intervertebral disc degeneration, proportion of thoracic and lumbar cord involvement, proportion of brain involvement and lesion enhancement rate in magnetic resonance imaging (MRI) were compared between the two groups of patients. The number, length, location on the sagittal image, pattern on the sagittal image, and distribution on the axial image of the lesions in the contrast-enhanced MRI of the two groups were evaluated.

Results There were differences between NMOSD and CSM patients in the proportion of women, the proportion of bowel and bladder symptoms, mRS levels, the length of the spinal cord involved by T2 hyperintensity, degree of intervertebral disc degeneration, the proportion of thoracic and lumbar cord involvement, the proportion of brain involvement, the enhancement rate and number of lesions ($p < 0.05$). Among NMOSD patients, linear, patchy and ring or semi-ring enhancement were present in 8(30.8%) ,14 (53.8%) and 4(15.4%)patients, respectively, and axial gray and white matter were involved in 17 (65.4%) patients. Among patients with CSM, 9(36.0%) patients showed longitudinal oriented flake, 16 (64.0%) patients showed pancake-like enhancement, and 21 (84.0%) patients showed axial white matter involvement only. The differences in enhancement pattern on sagittal images and axial involvement were statistically significant ($p < 0.05$).

Conclusions Early differential diagnosis of NMOSD and CSM in acute myelopathy can be made by analyzing images and the number, length, sagittal enhancement pattern, and axial involvement of gadolinium-enhanced lesions.

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Keywords Acute myelopathy, Neuromyelitis optica spectrum disorder, Cervical spondylotic myelopathy, Magnetic resonance imaging, Gadolinium enhancement

Introduction

Myelopathy refers to any pathological process affecting the spinal cord. Acute myelopathies are spinal cord disorders characterized by a rapidly progressive course reaching nadir within hours to a few weeks that may result in severe disability [1]. Acute myelopathy includes non-inflammatory causes (such as compression or ischemia) and inflammatory causes. Acute myelopathy is considered a diagnostic dilemma due to different pathogenesis and overlap of clinical and radiographic findings among different etiologies.

Cervical spondylotic myelopathy (CSM) is the most common form of spinal cord injury in adults [2]. The natural history of CSM is inconsistent. Many conditions are chronic and progressive [3], 20–62% of symptomatic patients experience neurological deterioration [4]. Neuromyelitis optica spectrum disorder (NMOSD) accounts for one-third or more of central nervous system inflammatory diseases in Asians [5]. The acute neurological deterioration of insidious CSM is similar to that of acute and subacute NMOSD, with overlapping clinical symptoms, which can be easily misdiagnosed clinically. Additionally, patients with NMOSD may have concurrent CSM, making it difficult to distinguish the cause of symptoms [6]. From a neuroimaging perspective, both NMOSD and CSM can detect high-intensity lesions in the cervical spinal cord, and some NMOSD patients have cervical spinal cord compression. NMOSD and CSM can lead to permanent disability or even death if they are not diagnosed early and receive medical or surgical intervention.

Magnetic resonance imaging (MRI) is critical for the diagnosis of NMOSD and CSM. CSM can also present with multilevel spinal cord edema similar to NMOSD, manifesting as T2 hyperintensity across multiple vertebral segments [7]. At this time, enhanced MRI imaging becomes more important in the identification of the two. We studied the neuroimaging characteristics of NMOSD and CSM with the aim of early diagnosis and intervention of NMOSD and CSM in acute myelopathy in clinical work, thereby improving clinical outcomes.

Materials and methods

Experimental subjects

The study included hospitalized patients diagnosed with NMOSD ($n=28$) and cervical spondylosis ($n=50$) at the Third Hospital of Hebei Medical University between January 2021 and February 2024 (Fig. 1). The inclusion criteria for patients with NMOSD are as follows: (1) Patients with onset time less than 21 days [8]; (2) Patients diagnosed with NMOSD according to international consensus diagnostic criteria [9]; (3) Aquaporin 4 (AQP4) antibody serology was positive; (3) Clinical and imaging evidence confirmed involvement of the cervical spinal cord. The inclusion criteria for patients with CSM are as follows: (1) Patients with onset time less than 21 days [8]; (2) Diagnosis of CSM confirmed by clinical and imaging evidence [10]. Patients with incomplete basic clinical data, and imaging data were excluded. Collect patients' clinical data, including gender, age, symptoms, and modified rankin scale (mRS) at admission [11]. This study was approved by the Ethics Committee of the Third Hospital of Hebei Medical University.

Neuroimaging

The MRI examination used a 3.0T magnetic resonance imager produced by Philips of the Netherlands to complete the scan of the head and spinal cord. Scanning sequences included T1-weighted imaging, T2-weighted imaging, T2 fluid attenuated inversion recovery sequences of the head and spinal cord, and gadolinium-enhanced images of the spinal cord. Spinal cord imaging includes sagittal and axial views.

Imaging assessment

Two radiologists specializing in spinal cord disease reviewed all images without knowledge of the final diagnosis. When there is a disagreement between the two parties, consensus is reached through negotiation. If multiple lesions were present, the larger lesion was selected for description of contrast enhancement pattern and assessment of signal intensity. Evaluation of MRI: (1) signal intensity of cervical spinal cord lesions on T1-weighted imaging, (2) the number of involved vertebral segments on sagittal images of cervical spinal cord lesions on T2-weighted imaging, (3) evaluate the extent of intervertebral disc degeneration based on Pfirrmann criteria [12], (4) thoracic and lumbar spinal cord involvement, (5) brain involvement, and (6) enhancement of cervical spinal cord lesions. Evaluation of enhanced MRI features: (1) the number of discrete enhanced lesions, (2) the number of involved vertebral segments on the sagittal images, (3) the pattern of enhancement on the sagittal images, and (4) the distribution of enhancement in white matter and/or gray matter on the axial images.

Statistical analyses

All data were analyzed statistically using SPSS software (version 25.0, IBM, USA). Continuous variables that adhered to normal distribution were expressed as

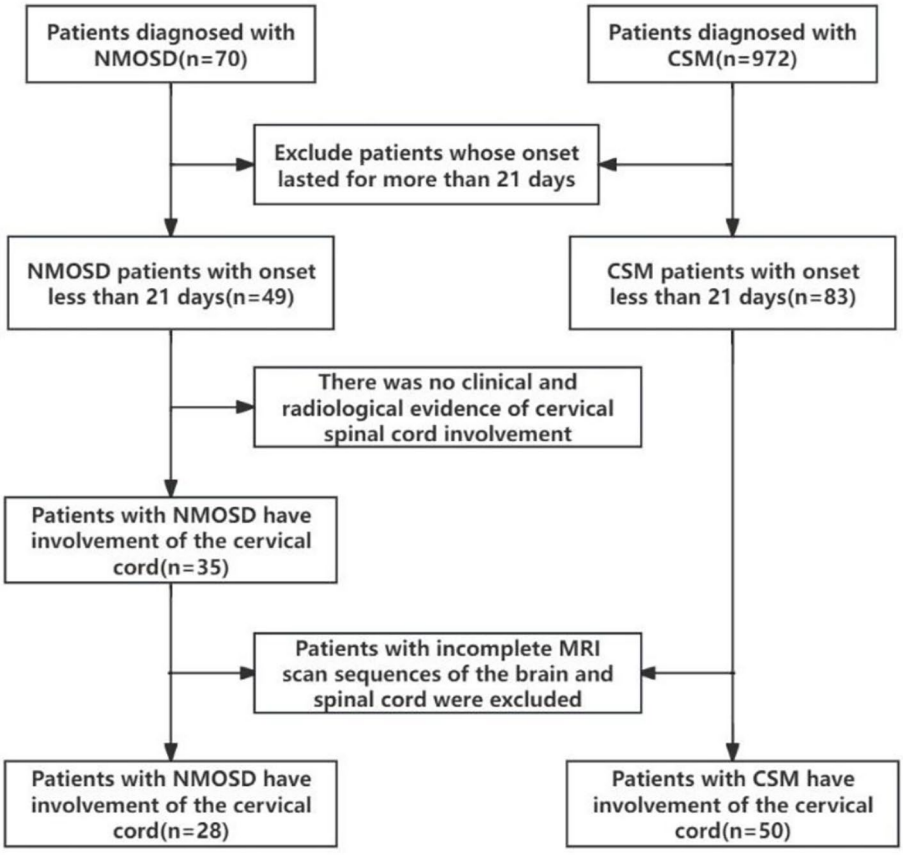

Fig. 1 Flowchart of the enrolled patients
NMOSD, Neuromyelitis Optica Spectrum Disorder; CSM, Cervical Spondylotic Myelopathy

mean±standard deviation (SD), and t-test was used for comparison between two groups. Continuous variables that were not normally distributed were expressed as the median with interquartile range (IQR), and the Mann–Whitney U test was used to compare between two groups. Categorical variables were displayed as counts and percentages, and comparisons between two groups were performed using the Pearson's chi-squared test. p value <0.05 was considered as a statistically significant difference.

Results

Demographic characteristics and clinical symptoms

A total of 28 patients with NMOSD and 50 patients with CSM were included. The age of NMOSD patients was 47.54±16.10 years old, and the age of CSM patients was 53.18±12.23 years old. The difference was not statistically significant ($p=0.086$). Among NMOSD patients, 25 (89.3%) were female, and among CSM patients, 15 (30.0%) were female. The proportion of females in NMOSD patients was significantly higher than that in CSM patients ($p<0.001$). Regarding the clinical symptoms of patients, the proportion of bowel/bladder symptoms($p=0.002$) and mRS levels($p<0.001$) in NMOSD patients were significantly higher than those in CSM patients. However, there was no statistically significant difference in the proportion of numbness, paresthesia, pain, and weakness symptoms between the two groups of patients ($p>0.05$). See Table 1 for details.

General imaging features

In T1-weighted images of patients with NMOSD and CSM, both lesions appeared isointense or hypointense, and there was no statistically significant difference between the groups ($p=0.091$). The mRS level of patients with low-intensity lesions on T1-weighted images in NMOSD patients was higher than that of patients with isointense lesions ($p=0.00$), but there was no relationship between the two in CSM patients, see Table 2. In NMOSD patients, T2-weighted hyperintense lesions affected a median of 4(3-5.75) vertebrae, and in CSM patients, T2-weighted hyperintense lesions affected a median of 2(1–2) vertebral bodies. The T2-weighted hyperintense lesions in NMOSD patients involved longer vertebral bodies than those in CSM patients ($p<0.001$). The Pfirrmann grade of intervertebral discs in NMOSD

Table 1 Demographic characteristics and clinical symptoms of the patients

	Total($n=78$)	NMOSD($n=28$)	CSM($n=50$)	t/χ2/Z	*p*-value
Age, y, mean ± SD	51.15 ± 13.91	47.54 ± 16.10	53.18 ± 12.23	1.742	0.086
Female, *n* (%)	40(51.3)	25(89.3)	15(30.0)	25.251	<0.001
Symptoms, ***n*(%)**					
Numbness	71(91.0)	25(89.3)	46(92.0)	0.000	1.000
Paresthesia	44(56.4)	14(50.0)	30(60.0)	0.730	0.393
Pain	30(38.5)	14(50.0)	16(32.0)	2.457	0.117
Weakness	54(69.2)	23(82.1)	31(62.0)	3.419	0.064
Bowel/bladder	20(25.6)	13(46.4)	7(14.0)	9.900	0.002
mRS, median (IQR)	2.0(1.0–3.0)	2.5(2.0–4.0)	1.0(1.0–3.0)	-3.638	<0.001

Table 2 Relationship between T1-weighted image signal intensity and mRS

	Hypointense	Isointense	Z	*p*-value
Total, median (IQR)	3.5(2.5-4.0)	2.5(2.0–3.0)	-3.036	0.002
NMOSD, median (IQR)	4.0(3.5–5.25)	3.0(2.5-3.0	-3.169	0.002
CSM, median (IQR)	2.5(2.0–4.0)	2.5(2.0–3.0	-0.873	0.383

patients was lower than that in CSM patients ($p<0.001$). Among NMOSD patients, 6 (21.4%) had thoracic or lumbar cord involvement, and 10 (35.7%) NMOSD patients had brain involvement, while no CSM patients had brain and thoracic or lumbar cord involvement. The enhancement rate of lesions in NMOSD patients was significantly higher than that in CSM patients ($p<0.001$), see Table 3; Fig. 2. There was an NMOSD patient with syringomyelia, see Fig. 3B.

Enhance imaging features

Among the included patients, 26 (92.9%) patients with NMOSD and 25 (50.0%) patients with CSM showed lesion enhancement on images. The median number of enhancing lesions was 2(1-3.25) in NMOSD patients and 1(1–1) in CSM patients ($p<0.001$). The median length of enhancement in sagittal images was 2(2–4) segments in patients with NMOSD and 1(1–1) segment in patients with CSM ($p<0.001$). Enhanced lesions in NMOSD patients appear scattered and in varying locations on sagittal images, while enhancement in CSM patients is most common at the C4-5 and C5-6 levels, and is limited to one intervertebral level. On sagittal images, among NMOSD patients, 14 (53.8%) patients showed patchy enhancement (Fig. 1B), 8 (30.8%) patients showed linear enhancement (Fig. 1A), and 4 (15.4%) patients showed ring or semi-ring enhancement (Fig. 2A). Among patients with CSM, 16 (64.0%) patients showed transverse band or pancake-like enhancement (Fig. 1D), and 9 (36.0%) patients showed irregular patchy enhancement along the longitudinal long axis (Fig. 1E). The difference in the enhancement form between the two groups of patients was statistically significant ($p<0.001$). On axial images, enhancement involved only white matter in 9 (34.6%) patients with NMOSD and 21 (84.0%) patients with CSM. Enhancement was seen in both gray and white matter in 17 (65.4%) NMOSD patients and 4 (16.0%) CSM patients ($p<0.001$), see Table 4; Fig. 2.

Discussion

Acute myelopathy has the characteristics of sudden onset and high disability rate, and the highly overlapping clinical symptoms of NMOSD and DCM make diagnosis particularly difficult.

Therefore, clinical interpretation of neuroimaging is crucial and is the key to early diagnosis and treatment. Our study indicated that there were significant differences between NMOSD and CSM in conventional MRI imaging, brain involvement, and lesion enhancement rates. In contrast-enhanced images, there were significant differences in the number, length, sagittal enhancement pattern, and axial involvement of the lesions of the two diseases.

Regarding demographic characteristics, our results showed that patients with NMOSD had a higher

Table 3 General imaging features of the patients

	Total($n=78$)	NMOSD($n=28$)	CSM($n=50$)	χ2/Z	*p*-value
T1-weighted image signal intensity, ***n*(%)**				2.861	0.091
Hypointense	43(55.1)	19(67.9)	24(48.0)		
Isointense	35(44.9)	9(32.1)	26(52.0)		
Number of T2 hyperintense vertebral bodies, median (IQR)	2(1–3)	4(3-5.75)	2(1–2)	-5.824	<0.001
Pfirrmann classification of intervertebral discs, median (IQR)	3.0(2.75-4.0)	2.0(2.0–3.0)	4.0(3.0–4.0)	-5.037	<0.001
Thoracic and lumbar spinal cord involvement, *n* (%)	6(7.7)	6(21.4)	0(0)	8.785	0.003
Brain involvement, *n* (%)	10(12.8)	10(35.7)	0(0)	17.412	<0.001
Lesion enhancement, *n* (%)	51(65.4)	26(92.9)	25(50.0)	14.566	<0.001

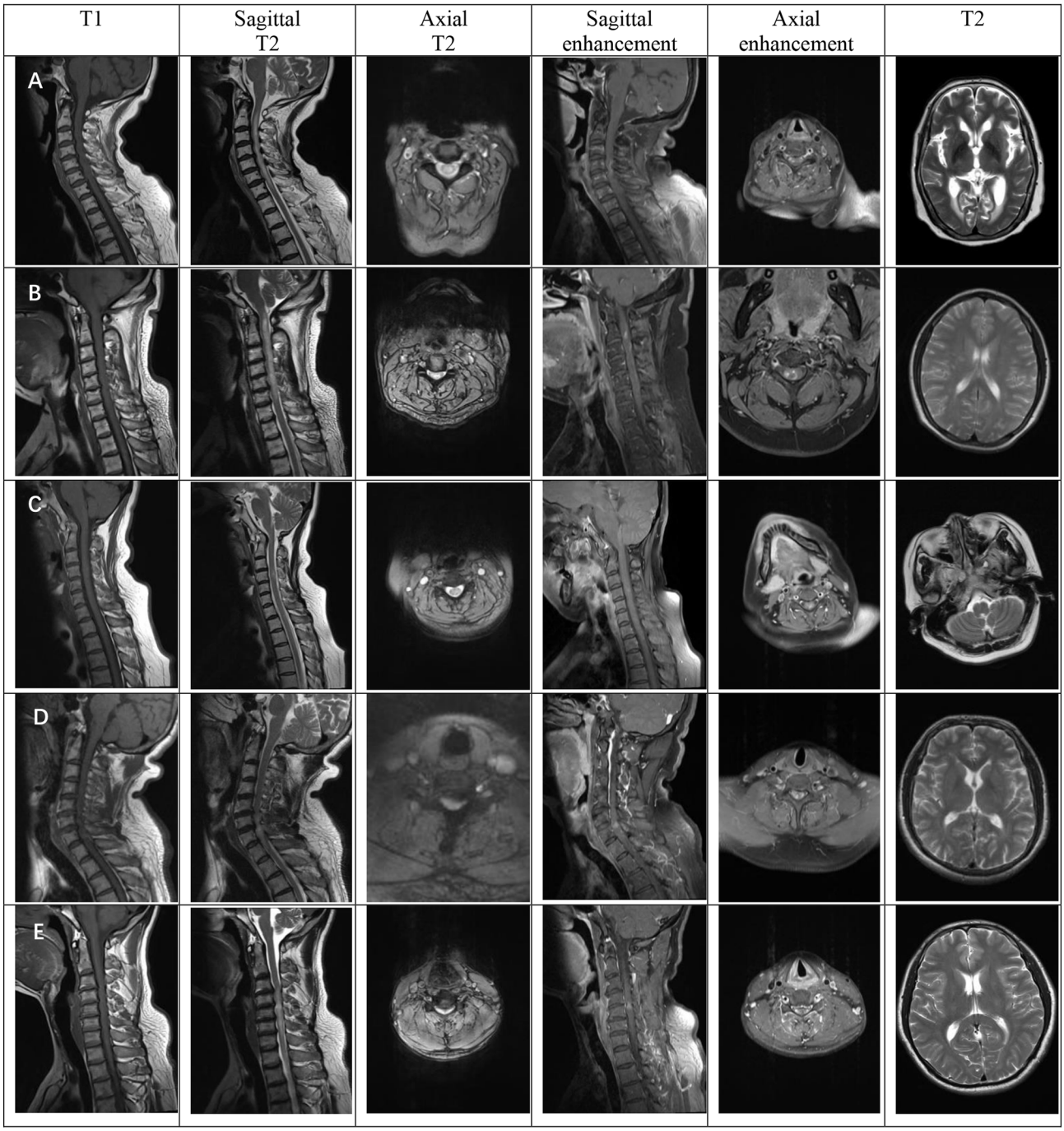

Fig. 2 Examples of typical images of patients with NMOSD and CSM. (**A**) A 76-year-old female patient with NMOSD. The lesions showed long T1 and long T2 signals, linear gadolinium enhancement in sagittal images, and axial involvement of gray and white matter. (**B**) A 37-year-old male patient with NMOSD. The lesion showed long T1 and long T2 signals, patchy enhancement in sagittal images, gray and white matter involvement in the axial position, and involvement of the right basal ganglia area. (**C**) A 43-year-old female patient with NMOSD. The lesion showed long T1 and long T2 signals, patchy enhancement in the sagittal image, gray matter involvement in the axial position, and area postrema in the medulla oblongata. (**D**) A 46-year-old male patient with CSM. The lesion showed long T1 and long T2 signals, pancake-like enhancement in the sagittal image, and only involved white matter in the axial view. (**E**) A 33-year-old male patient with CSM. The lesion showed T1 isointense, long T2 signal, and patchy enhancement in the longitudinal long axis of the sagittal image. Only the white matter was involved in the axial position

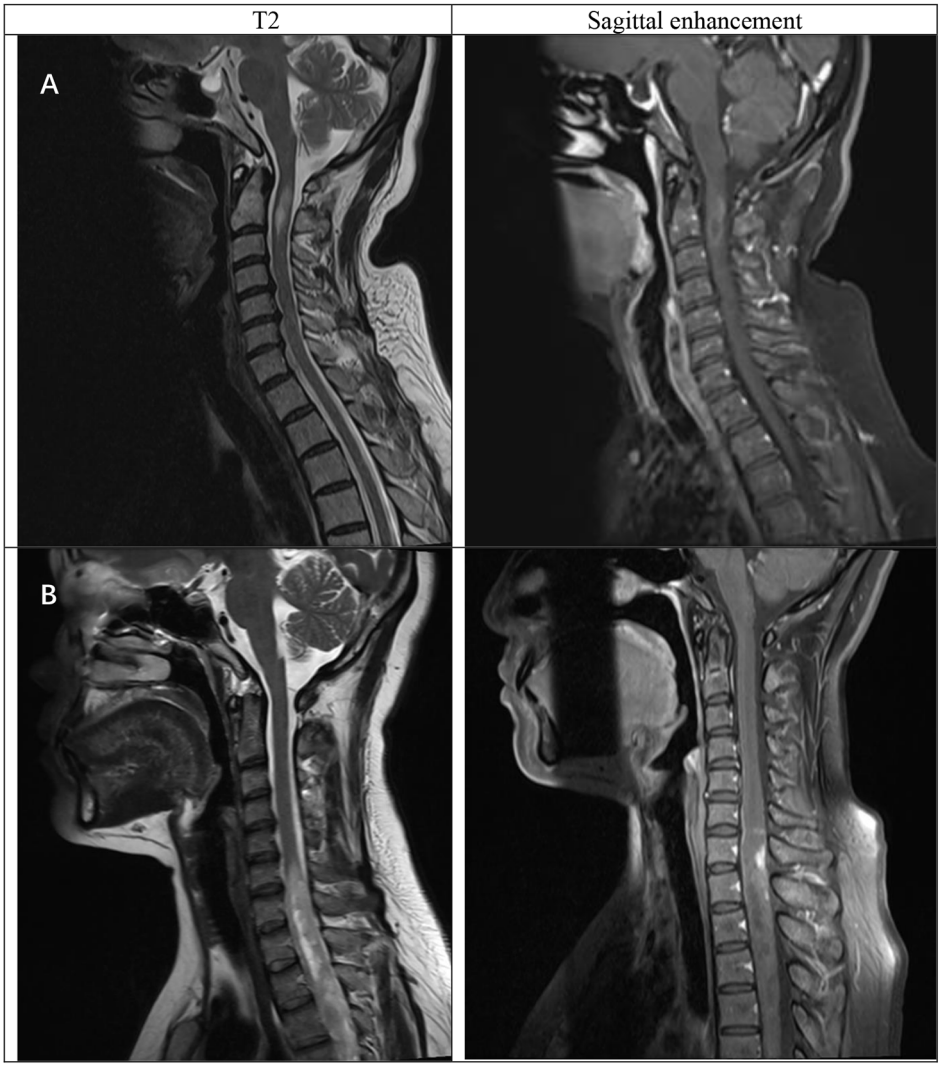

Fig. 3 Rare neuroimaging manifestations of NMOSD. (**A**) Imaging manifestations of ring enhancement in NMOSD. (**B**) A patient with NMOSD 14 days after onset was complicated by syringomyelia

proportion of females, whereas patients with CSM had a higher proportion of males. The results are consistent with previous studies in which nearly 90% of seropositive NMOSD cases were female and two-thirds were male [13, 14]. Our research showed that most clinical symptoms of NMOSD and CSM were similar, such as: numbness, paresthesia, pain, weakness. However, patients with NMOSD had a higher proportion of bowel/bladder disorders compared with CSM.

NMOSD and CSM spinal cord lesions mostly appear as hypointense or isointense on T1-weighted images and hyperintense on T2-weighted images. We found that spinal cord T1 hypointense lesions were more common in patients with NMOSD. Due to pathophysiological reasons, T1 hypointensity means black holes leading to higher EDSS scores and severe disease, especially AQP4-positive disease [15]. T1 hypointense lesions are a sign of permanent demyelination and axonal damage, and it has been hypothesized that they are caused by tissue damage or damage to the blood-brain barrier [16]. Our study found that 35.7% of NMOSD patients had brain involvement, and 3 (10.7%) patients had medulla oblongata involvement. Although NMOSD has traditionally been considered a disease that does not involve the brain, brain abnormalities are not uncommon in patients with neuromyelitis optica-related conditions. Studies have shown that 31% of NMOSD patients have abnormal brain MRI at onset. The most common site of brain involvement in NMOSD is the corticospinal tract (such as the posterior limb of the internal capsule and the cerebral peduncle), accounting for 44% of brain involvement. Involvement of the medulla oblongata is more suggestive of NMOSD [17]. This result is similar to ours. Research in Argentina showed that 5.4% of NMOSD patients present with Area

Table 4 Enhance imaging features of the patients

	Total (*n* = 51)	NMOSD (*n* = 26)	CSM (*n* = 25)	χ^2/Z	*p*-value
Sagittal image enhancement					
Number of enhanced lesions, median, median (IQR)	1(1–2)	2(1-3.25)	1(1–1)	-4.783	<0.001
Number of vertebral segments involved in lesion enhancement, median (IQR)	1(1–2)	2(2–4)	1(1–1)	-5.564	<0.001
Locations of the enhancement, ***n*(%)**					
C2	8(16.7)	8(34.8)	0(0)	8.081	0.004
C3	13(25.5)	10(38.5)	3(12.0)	4.699	0.030
C4	22(43.1)	12(46.2)	10(40.0)	0.197	0.657
C5	24(47.1)	8(30.8)	16(64.0)	5.649	0.017
C6	24(47.1)	10(38.5)	14(56.0)	1.574	0.210
C7	13(25.5)	9(34.6)	4(16.0)	2.325	0.127
Enhancement pattern on sagittal images, ***n*(%)**				70.681	<0.001
Linear	8(15.7)	8(30.8)	0(0)		
Patchy	14(27.5)	14(53.8)	0(0)		
Ring or semi-ring	4(7.8)	4(15.4)	0(0)		
Transverse band (pancake-like)	16(31.4)	0(0)	16(64.0)		
Longitudinal oriented flake	9(17.6)	0(0)	9(36.0)		
Distribution of axial images, ***n*(%)**				12.833	<0.001
White matter	30(58.8)	9(34.6)	21(84.0)		
Gray and white matter	21(41.2)	17(65.4)	4(16.0)		

postrema syndrome, manifesting as intractable hiccups, nausea, and vomiting [18]. Area postrema syndrome is of high diagnostic significance for NMOSD [19]. Since area postrema lesions are easily overlooked, special attention is required. A small number of NMOSD patients may also have ependymal and diencephalon involvement around the ventricular system [20]. Therefore, brain MRI is also an important part of the differential diagnosis of myelopathy. The lesion enhancement rate of NMOSD was as high as 92.9% in this study, and the lesion enhancement rate of CSM was 50.0%. Studies have shown that approximately 7% of cases of CSM have enhancement, often leading to misdiagnosis as tumor or myelitis [21]. A study of CSM patients with enhanced intramedullary lesions found that 37% had a subacute onset (less than 8 weeks) and the other 63% had an insidious onset [22]. The CSM enhancement rate in this study was higher than that in previous studies. This may be because the patients enrolled in this study were acute patients with a course of less than 21 days. Acute patients have more severe edema due to rapid onset of spinal cord disease and no adaptation before onset.

Our observation of spinal cord enhancement on MRI can help guide initial differential diagnosis. Overlapping features between inflammatory and non-inflammatory myelopathies may lead to misdiagnosis. This study provided strong evidence for the clinical value of sagittal images and T1 MRI enhancement patterns. Our study showed that on enhanced sagittal images, enhancing lesions involving multiple locations and long vertebral segments are suggestive of NMOSD. In patients with CSM, enhancement was most common at the C4-5 and C5-6 levels, most of which were located at or immediately below the maximum stenosis, usually as a single enhancement focus and limited to a certain intervertebral level.

The enhancement pattern of the spinal cord in sagittal images and the involvement of axial enhancement play a key role in distinguishing NMOSD from CSM. The enhancement pattern of the spinal cord in NMOSD is variable: patchy and inhomogeneous, with ill-defined margins (cloud-like), and a periependymal linear pattern (pencil-thin enhancement) [23]. In our study, 53.8% of NMOSD patients showed patchy enhancement, 30.8% of patients showed linear enhancement, 15.4% of patients showed ring or semi-ring enhancement, and 65.4% of patients showed central involvement of axial gray and white matter. CSM patients showed pancake-like enhancement (64.0%) and longitudinal patchy enhancement (36.0%), most of which only involved the axial white matter, showing a peripheral type. Some studies have shown that sagittal transverse flattened pancake-like enhancement just below the area of maximal stenosis, with associated periaxial white matter enhancement, is characteristic of CSM [24]. Studies analyzing enhancement results in NMOSD and CSM patients have shown that 50% of CSM patients have pancake-like enhancement features, while there are no cases of pancake-like enhancement in NMOSD, suggesting that the specificity of pancake-like enhancement in diagnosing CSM is 100% and the sensitivity is 50–73% [25]. Studies have shown that ring enhancement accounts for one-third of NMOSD patients, but in this study, ring enhancement accounted for 15.4% and is uncommon [26]. Ring enhancement of

the spinal cord distinguishes NMOSD from longitudinally widespread myelopathy caused by causes other than multiple sclerosis, including CSM, and is therefore suggestive of demyelinating disease [26].

Interestingly, one NMOSD patient in our study was complicated by syringomyelia. Previous studies have also found that syringomyelia may occur in the acute stage of NMOSD myelitis, but will slowly disappear in the later stages [27]. It is unknown whether syringomyelia is the result of demyelination in NMOSD or whether it just occurs in patients with NMOSD. The literature currently available is limited and new case reports are needed to provide research evidence.

Limitations

First, this study is a single-center retrospective study, which has the inherent limitations of a retrospective study. A single-center study will lead to a limited sample size and a limited patient group. NMOSD is a rare disease, which will lead to a small sample size. Secondly, acute myelopathy is a myelopathy in which symptoms progress to the lowest point within up to 21 days after onset. We did not determine the peak of the disease and intervened after admission to the hospital for diagnosis. The enrolled population may have subacute myelopathy, resulting in bias. Additionally, we did not follow up the patient's prognosis and could not analyze the evolution of the enhancement pattern.

Conclusions

MRI and contrast-enhanced MRI have differential value in NMOSD and CSM in acute myelopathy. The typical imaging manifestations of NMOSD are T2 hyperintensity involving more than three vertebral body segments, the number and length of enhanced sagittal images exceeding two, patchy or linear enhancement, axial involvement of gray matter and brain height. The typical manifestation of CSM is T2 hyperintensity involving less than 3 vertebral segments. On the enhanced sagittal image, a pancake-like or longitudinal sheet-like enhanced lesion located at or below the narrowest part of the spinal cord, one lesion, no more than one vertebral body in height, and peripheral enhancement in the axial position only accumulates in the white matter.

Abbreviations

NMOSD	Neuromyelitis optica spectrum disorder
CSM	Cervical spondylotic myelopathy
MRI	Magnetic resonance imaging
AQP4	Aquaporin 4
mRS	Modified rankin scale
SD	Standard deviation
IQR	Interquartile range

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Author contributions

WL, WB, and HR were responsible for the conception and design of the trial. Data collection and management were performed by WL, RS, SC, JL, YS, XL. Data analysis was performed by WL, WB, and RS. WL wrote the first draft of the paper. WB reviewed and revised the manuscript. HR reviewed and edited the manuscript and gave final approval. All authors listed made significant contributions to the research and writing of this article. All authors (WL, WB, RS, SC, JL, YS, XL, HR) read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and was reviewed and approved by the Ethics Committee of the Third Hospital of Hebei Medical University. Due to review given the nature of the study and the anonymity of the data, the ethics committee waived the need for informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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